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# MEDICAL DIAGNOSIS

## A MANUAL OF CLINICAL METHODS

" Felix, qui potuit rerum cognoscere causas."

—VIRG. *Georg.* II. 490.

# MEDICAL DIAGNOSIS

## A MANUAL OF CLINICAL METHODS

BY

J. J. GRAHAM BROWN, M.D.

*Fellow of the Royal College of Physicians of Edinburgh*
*Fellow of the Royal Society of Edinburgh*
*Lecturer on the Principles and Practice of Medicine*
*in the School of Medicine of the Royal Colleges, Edinburgh*

FOURTH EDITION

REVISED AND ENLARGED

*WITH ONE HUNDRED AND TWELVE ILLUSTRATIONS*

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# PREFACE TO THE FOURTH EDITION.

As a result of various causes, this book has been for some considerable time out of print. It has now, however, been thoroughly revised, and in great measure re-written. The chapters dealing with the examination of the gastric contents, of the blood, and of the urine, and those treating of the nervous system, have, in particular, been recast and considerably extended.

The addition of many illustrations will, it is hoped, add to the clearness and usefulness of the text.

In view of the amount of work which is now being done on questions relating to metabolic changes, and the large field which this subject presents for investigation by young graduates, it has been thought advisable, in the chapters dealing with the urinary system to retain the references to original authorities, so that the worker may know where to turn for further information on these topics. For the sake of brevity, the references have been almost completely expunged from other portions of the book.

The author gratefully acknowledges the help generously afforded him by many friends.

To Dr John Wyllie he is indebted for permission to make

use of various illustrations, including figs. 86 and 112, to Dr Lovell Gulland for a beautiful preparation of blood from which figure 17 was drawn, to Dr M'Kenzie Johnston for revising the chapter dealing with the nares and larynx, and to Dr John Thomson for much help and encouragement throughout the progress of the work.

J. J. G. B.

3 Chester Street, Edinburgh,
*October*, 1897.

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# INTRODUCTION.

A PHYSICIAN, when consulted by a patient, is naturally enough expected to be an attentive listener to what, to his informed mind, is a strange medley and most confused account of those deviations from health or actual sufferings by which the patient has been driven to seek aid. The more serious symptoms are often lightly touched upon, the more trivial exaggerated, and the whole jumbled together without logical sequence or the slightest attempt at orderly arrangement. To this story, trying as it is to the physician, and all the more trying the more his own mind is duly trained, he ought to listen; for this the patient expects, and perhaps has a right to expect. During the tedious narration, it may give him patience to bear in mind two considerations: first, that from it he must obtain the right end of the clue which is to guide him in the difficult task of ascertaining the nature, extent, and seat of the disease; and second, that by this often most prolix narrative, taken along with his attitude, manner, and expression, the patient, absorbed in his own sufferings, is giving his physician the best opportunity of becoming acquainted with the *ego* with whom he has to deal.

The most critical examination of symptoms, the most careful inquiry into the state of internal organs, the most logical deductions from these as to the morbid changes from which they have originated, will often be erroneous unless the physician is also a student of human nature, and is able to arrive almost intuitively

at some knowledge of the mental characteristics and peculiarities of his patient.

But sooner or later—and more often late than soon—the patient will have arrived at the end of his narration, and then the physician must unravel for himself this tangled web; and, taking the different threads, he must follow them up, and by means of close physical examination, ascertain the condition of the various organs of the body—particularly those which the train of symptoms detailed indicates as implicated in the morbid process. It is only by a methodical examination of the different systems of the body that a satisfactory view of the condition of the patient can be obtained, and the very foundation of rational treatment laid.

In the following pages an attempt will be made to explain the meaning and diagnostic significance of the chief symptoms and physical signs which are met with in disease. These group themselves naturally round the different physiological systems of the body, and under those headings they will be considered. This is not, of course, to be looked upon as a rigidly accurate division, but for practical purposes it suffices, and it has this great advantage—viz., that those who are habituated to follow such an arrangement in the examination of patients, are less apt to neglect minute points which might otherwise escape the memory. Nor is it to be supposed that every patient requires to be subjected to so exhaustive a catechising as this arrangement, if fully carried out, would necessitate. Many trivial complaints call for no such exercise of patience either on the part of the physician or on that of his patient, and in severe or urgent cases the first examination must necessarily be at best rapid and limited. Nor even where close inquiry is desirable, is it necessary to follow accurately the sequence here given; and to some it may seem more suitable to clear up, first of all, the details regarding that system which seems most profoundly implicated, and only thereafter, and more cursorily, to examine into the condition of the others.

It must be carefully borne in mind that in examining a patient we are dealing with a fellow-creature, and that all our

inquiries and all our investigations must be conducted with the utmost courtesy, kindness, and patience.

In the following pages attention will first be directed to certain preliminary inquiries which should be made, and then to the symptoms connected with the various systems of the body.

## CHAPTER I.

# THE GENERAL ASPECT, CONDITION, AND CIRCUMSTANCES OF A PATIENT.

Before entering upon the minute examination of a patient, there are several more general and preliminary enquiries which should be made, and it is needless to say that the care and extent of the investigation required must depend on two factors: first, on its necessity, in view of the special disease present; and second, on the mental and bodily condition of the patient.

After noting the patient's name, age, occupation, residence, &c., it is well to record, in as brief words as possible, and in his own language, his chief complaint. This is not to be in any sense a statement of diagnosis, but simply the patient's own impression concerning his case. Both in cases of phthisis and bronchitis, for example, we might be told that the patient sought advice on account of severe cough, and of this symptom we would make note as the most prominent in his own mind. We further ascertain, as closely as we can, the duration of the present illness, and record it briefly—so many days, months, or years, as the case may be.

Having thus formed in our minds a general idea, however ill-defined, of the case before us, we proceed to consider the

**Family History.**—Inquiry into the general health of the patient's family should be specially directed to ascertain whether any of his near relatives have suffered from those forms of disease which are usually supposed to be hereditary,

such as phthisis, syphilis, rheumatism, gout, heart disease, and various nervous disorders.

**Habits and General Surroundings at Home and at Work.**—Luxurious habits, "fast" living, and excesses of all kinds are frequently the cause of disease, and any evidence of these must be sought for, and among them excessive alcoholic indulgence stands out prominently. To defective or unwholesome diet many ailments may be traced, as well as to long hours of work, and to the bad ventilation or defective drainage of the apartments used. Insufficient or ill-arranged clothing is also a frequent cause of disease.

It is also well known that certain occupations have a special tendency to produce disease. These may be classed as follows, according as they are due to the following causes:—

(*a.*) *Mechanical effects of inhalation of dust*, as in knife-grinders, coal miners, and stone masons.

(*b.*) *Poisonous effects of materials worked in*—such as arsenic, lead, copper, mercury, petroleum, wool containing anthrax, sewer-gas, &c.

(*c.*) *The position and movements required.*—If certain peculiar movements are very frequently repeated, a condition of spastic muscular contraction is apt to arise, which, when it shows itself in clerks, is known as writer's cramp, but which is also met with in connection with such occupations as engraving, sewing, type-setting, harp-playing, &c. There is further the position of the body to be considered, whether sitting, standing, or kneeling; and finally, the amount of muscular exertion which the particular employment demands.

**Previous General Health.**—We should endeavour to ascertain the usual state of health, the date and nature of former ailments, liability to particular morbid conditions, present or previous residences, or other circumstances which may have influenced its production or development, exposure to contagion, &c.; and if the patient be a female, it may be advisable to inquire into the condition of the reproductive functions.

**Origin and Course of the Present Illness.**—It is impossible here to do more than indicate certain general lines on which it is usual to proceed. Having already fixed the date of commencement of the illness, we would next endeavour to gain some accurate idea of the *manner* in which it commenced, with what symptoms, whether it came on suddenly or gradually, to what cause the patient traces his loss of health; and if his statement does not appear to us probable, we must strive by careful, guarded, and unobtrusive cross-examination, to satisfy ourselves on these points. Knowing the usual etiology of such a case as the one we are studying, we possess a guide as to the direction in which our inquiries should be made. The sequence of symptoms may now be ascertained, the date of origin of each, and its severity; and finally, we note to what medical treatment the patient has been subjected, and what was its result in his case.

## Present Condition.

Before proceeding to the examination of each system of the body, it is advisable first to note certain general facts, as follows:

1. Height and weight.
2. Development and muscularity.
3. Condition of the skin as to
   - (*a.*) Colour.
   - (*b.*) Perspiration.
4. Condition of the subcutaneous tissue—
   - (*a.*) Adiposity.
   - (*b.*) Œdema.
   - (*c.*) Emphysema.
5. Expression of the face.
6. Temperament, constitution, or diathesis.
7. Attitude.
8. Evidence of previous injury or disease.
9. Temperature.

1. **Height and Weight.**—In almost all diseases the weight becomes diminished, and in the course of treatment the patient should, when it is practicable, be weighed at regular intervals, when a very valuable indication of the progress of the malady will be in our hands. When, however, we have only the result of one weighing, it is of consequence to know what a man of a given height ought to weigh when in health. For this purpose, Mr Hutchison compiled a table (deduced from the examination of 3000 persons), from which the following figures are taken, which must, however, not be regarded as more than approximate.

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| A man of | 4 | ft. | 6 | in. | to | 5 | ft. | 0 | in. | ought to weigh about 92·26 lbs. |
| ,, | 5 | ,, | 0 | ,, | | 5 | ,, | 1 | ,, | ,, ,, 115·52 ,, |
| ,, | 5 | ,, | 2 | ,, | | 5 | ,, | 3 | ,, | ,, ,, 127·86 ,, |
| ,, | 5 | ,, | 4 | ,, | | 5 | ,, | 5 | ,, | ,, ,, 139·17 ,, |
| ,, | 5 | ,, | 6 | ,, | | 5 | ,, | 7 | ,, | ,, ,, 144·29 ,, |
| ,, | 5 | ,, | 8 | ,, | | 5 | ,, | 9 | ,, | ,, ,, 157·76 ,, |
| ,, | 5 | ,, | 10 | ,, | | 5 | ,, | 11 | ,, | ,, ,, 170·86 ,, |
| ,, | 5 | ,, | 11 | ,, | | 6 | ,, | 0 | ,, | ,, ,, 177·25 ,, |
| ,, | 6 | ,, | 0 | ,, | | ..... | | | ,, | ,, ,, 218·66 ,, |

2. **Development and Muscularity.**—To be typical of perfect health, the various parts of the body should be accurately proportioned one to another. A moderate amount of adiposity is quite consistent with health, provided that the muscular system is correspondingly developed. Generally, as age advances, the tendency to the deposit of fat increases, and this must be borne in mind. At the same time, its rapid accumulation after fifty years of age is not a symptom of health. Spare people are often the longest livers.

3. **Condition of the Skin as to Colour.**

(*a.*) *Pallor* is due to defective filling of the capillaries, to deficiency in the quantity of the blood, or of the hæmoglobin it contains. Pallor, consequently, may arise from any condition which interferes with the formation of the blood (chlorosis, anæmia, &c.), from any disease leading to loss of blood (hæmorrhage) or its nutritive materials (Bright's disease), or,

finally, from any affection of the vascular system interfering with the proper propulsion of the blood (mental emotions, fatty heart, valvular disease, &c.). Paleness of the skin can best be appreciated where the epidermis is thin and the true skin very vascular, as on the ears, cheeks, eyelids, or lips.

(*b.*) *Redness* of skin beyond the natural tint, first and principally shows itself at those points which have just been mentioned in connection with pallor; but it must be borne in mind that those persons who are, by reason of their occupation, exposed to the weather, are usually ruddy in complexion. Apart, however, from this cause, redness occurs either as a result of increase of the amount of blood in the body, or of its hæmoglobin (as is seen in "full blooded" plethoric persons), or is due to dilatation of the capillaries. The latter cause accounts for the blushing produced by mental emotion, as well as that following the inhalation of nitrite of amyl; and in a similar way may be explained the redness of the scalp and face in hemicrania, and the general redness of inflammation and of fever, as well as the redness of face of alcoholics. A very delicate and transparent skin, it should be remembered, may lead to a ruddy look of health in persons who may be in reality anæmic. An examination of the blood will, in such cases, show the true state of matters.

(*c.*) *Cyanosis*, or blueness of the skin, produced by the accumulation of venous blood, varies much in degree. It is first noticeable on the lips, cheeks, conjunctivæ, ears, and point of the nose and fingers, but may become very general. It is due partly to the retardation of the venous flow, so that the blood remains longer in contact with the tissues, and consequently loses more of its oxygen and takes up a larger quantity of carbonic acid, and partly to deficient oxygenation of the blood in the lungs, so that even when it enters the capillaries it is more or less venous. The former cause, chiefly, operates in valvular affections of the heart when compensation is lost, the latter in laryngeal and pulmonary affections where the proper aeration of the blood is interfered with. It is in congenital malformations of the heart, particularly in cases of

constriction of the pulmonary orifice along with perforation of the ventricular septum, that the complete picture of *morbus cæruleus* is formed. The skin then assumes a livid blue colour, particularly on the nose, lips, ears, and fingers, and these tissues become swollen and indurated owing to the stagnation of blood. The clubbed fingers, with their broad, curved nails, due to this cause, are characteristic of congenital heart affections. Cyanosis may also be produced by local vaso-motor changes, as in chilling of the surface, and by any cause which leads to obstruction of a venous trunk.

(*d.*) *Jaundice*, or yellow discoloration of the skin, results from the absorption of bile pigment into the circulation, and the consequent staining of the tissues. It shows itself first under the conjunctivæ.[1] In slight cases there is only a very light tinging, but as the absorption of pigment goes on, the skin becomes citron-yellow, then olive green, and it may, finally, in very severe cases, assume a dark brownish green colour. The question of jaundice will be more fully treated in connection with the urinary system.

(*e.*) *Bronzing* occurs in Addison's disease (disease of the supra-renal capsules). The pigment is deposited in and between the cells of the Rete Malpighi, and is chiefly developed at the points naturally most liable to become darker, and also at the seat of any local irritation. The discoloration commences earliest and becomes deepest on the face, neck, hands, and in the axillæ round the nipples, in the groins, genital regions, and on the abdomen. It also occurs in situations where there has been local irritation, such as that caused by the pressure of garters, or by a recent blister. The mucous membrane of the lips, tongue, and mouth is likewise frequently discolored. The patches of pigmentation are (except those caused by local irritation) arranged symmetrically, and their margins are rarely well defined, but rather shade off imperceptibly. Pigmentation of the skin, closely resembling Addison's disease, is met with in

[1] The observer must not mistake for jaundice the yellow colour of small masses of fat which may be deposited there in advanced life.

chronic phthisis, and in lymphadenoma and leucocythæmia, and the diagnosis of Addison's disease must not be made from the presence of pigmentation alone, unless such pigmentation as has been described is accompanied with the characteristic constitutional symptoms which that disease presents—viz., asthenia, feeble action of the heart, and small, compressible pulse, gastric irritability, gasping, hiccup, sighing, and breathlessness on exertion. Smaller and more sharply defined patches of pigment are sometimes seen in pregnancy, and in disease of the generative organs in the female. In *Pityriasis versicolor* the presence of the parasite *Microsporon furfur* gives rise to a brown discoloration.

(*f.*) *Grey* discoloration of the skin (argyria) occurs after the prolonged use of nitrate of silver. It is most noticeable on those portions of the body which are most exposed to light. From the bluish tinge of cyanosis it can readily be distinguished by the fact that in argyria the discoloration does not disappear on pressure, as is the case in the former condition.

**Perspiration.**—Increased perspiration (hyperidrosis) may be general or local. In some nervous affections, particularly in hemiplegia, it has been seen to be limited to one lateral half of the body. In very many diseases general hyperidrosis may be observed, and furnishes sometimes an indication of considerable importance. Such, for example, is the perspiration which occurs in almost all organic diseases in the stage of collapse, and that which follows the crisis of continued fevers (critical sweat). One of the stages of ague is that of profuse perspiration, and the night sweats of phthisis are among its most distressing symptoms. In acute rheumatism the skin is habitually moist, and usually whenever there occurs considerable dyspnœa, from whatever cause, it is accompanied by perspiration. It must not be forgotten that strong mental emotions (fear) may produce copious sweating.

Local increase of perspiration is usually met with in connection with the axillæ, and the palms of the hands and soles of the feet. The sweat accumulating in these regions sometimes gives rise

to an excessively disagreeable odour (bromidrosis), due to the presence of an organised ferment.

Diminution of perspiration is seen locally in many chronic skin affections, and generally in almost all conditions of pyrexia, in diabetes, and sometimes in the cirrhotic form of Bright's disease. It also occurs in cases of profuse vomiting or profuse diarrhœa where much liquid is leaving the body.

The perspiration may be coloured yellow from the presence of bile pigments in cases of jaundice, and in uræmia it may hold in solution large quantities of urea. In certain rare cases it has been observed to have a blue colour; and still more rarely extravasation of blood on to the surface of the skin has been observed.

**Condition of the Subcutaneous Tissue.**—There are three pathological states to be noted under this head—viz. (*a.*) Adiposity; (*b.*) Œdema; and (*c.*) Emphysema.

### (*a.*) *Adiposity.*

(*a.*) *Adiposity or corpulence*, when it passes the limits of health, is a very important condition. It is characterised by the deposit of fat, chiefly in the subcutaneous, the subserous (omentum and mesentery), and intermuscular connecting tissues, and is usually associated either with a plethoric or an anæmic state of the body. The muscles are weak, the heart weak and dilated, the pulse small, rapid, and irregular. Palpitation and dyspnœa occur on exertion, and attacks of *angina pectoris* are not rare. The signs of bronchial catarrh are often present, and perspiration is usually excessive. In very corpulent persons the sexual functions are weak, and sterility is common. These functions appear to be intimately related to the deposition of fat. Where the sexual functions are absent, as in eunuchs and in women after menstruation has ceased, adiposity is common. Sugar is often present in the urine of very corpulent persons. Corpulence is most usually hereditary, but is sometimes brought on independently of this by

excessive eating, by the immoderate consumption of alcoholic liquors or by deficient exercise.

### (*b.*) *Œdema.*

(*b.*) *Œdema* or anasarca arises from the accumulation of serous fluid in the subcutaneous connective tissue. The swelling usually commences at the ankles—at first appearing only in the evenings, to disappear after the night's rest,—and it may increase to a very great extent, the limbs becoming much distended, pale, smooth, and glossy. The finger pressed upon the skin leaves a deep indentation when removed, which only slowly fills up. In well-marked cases the external genitals become greatly swollen. Occasionally the œdema makes its first appearance in the lower eyelid, then usually arising from the acute inflammatory form of Bright's disease. Œdema may arise—

1. From obstruction to the return of venous blood to the heart. In this way it may occur in almost all diseases of the heart, and in some pulmonary affections, particularly emphysema. Pressure on the inferior vena cava in the abdomen from an enlarged liver or tumour of any kind, or on the veins of the leg, may give rise to œdema; and local œdema is common round abscesses.

2. From alteration in the coats of the vessels, arising from a blood supply defective in amount or in quality. Under this heading fall the so-called hydræmic dropsies seen in diseases of the kidneys, in anæmia, and in many wasting diseases.

Local idiopathic œdema is sometimes seen, the result apparently of angio-neurotic disturbances, and probably analogous to the swellings seen in urticaria.

Inflammatory œdema, an indication of inflammation in the deeper parts, is as a rule of greater interest to the surgeon than to the physician.

In *myxœdema* the subcutaneous tissues are infiltrated with a peculiar mucoid substance, which causes a dropsy-like swelling all over the body. The face is most characteristic. It is swollen, waxy, and expressionless, the swelling differing from ordinary œdema

in two important points—viz., first, in not selecting the more dependent parts of the face (such as the lower in preference to the upper eyelid), but invading every feature alike; and second, in its resiliency, the pressure of the finger leaving no subsequent mark. Along with this peculiar œdema there is marked dryness of the skin, loss of hair, great hebetude of the nervous system, and in late stages insanity is liable to develop.

### (*c.*) *Subcutaneous Emphysema.*

(*c.*) *Subcutaneous Emphysema* of the skin is produced by a collection of gaseous fluid in the subcutaneous cellular tissue. The skin pits very slightly on pressure, and there is a curious and unmistakable feeling of crackling under the finger. Apart from surgical causes, emphysema occurs as the result of loss of continuity in the air passages or at some portion of the alimentary tract. Ulceration or wound of the larynx or trachea may give rise to emphysema, but it more commonly results from rupture of one or more air vesicles in the lung. In the latter case, the air is effused into the interlobular septa, and under the visceral pleura passes back to the root of the lung, thence to the mediastinal cellular tissue, and finally appears in the jugular fossa, and passes under the skin of neck and chest. Perforation of the œsophagus, from whatever cause, is liable to occasion subcutaneous emphysema, and ulceration of stomach or intestines may likewise allow of the escape of air under the skin if these structures have been previously glued to the abdominal walls by adhesions.

**The Expression of the Face** is often characteristic of the disease under which the patient is labouring, and is a very valuable indication, especially in children. It is the general effect upon the observer of a combination of a number of traits, such as the colour, volume, and muscular state of the face, the state of the eyes, &c. To some of these allusion has already been made; a few remarks, however, may be added. In connection with the eyes, an injected condition of the conjunctiva

is common in febrile conditions (particularly in typhus), and in neuralgia of the ophthalmic branch of the fifth nerve. In chlorosis and anæmia generally, the sclerotic has a peculiar white pearl-like lustre. Prominent eyeballs are met with in cases of exophthalmic gôitre, and in connection with tumours of the orbit. Sunken eyeballs are seen in all wasting disorders, due to the atrophy of the fat which lies in the posterior part of the orbit. In regard to the muscles of the face, we may meet with spasmodic contraction in tic-douloureux, tetanus, hysteria, epilepsy, and chorea. In facial paralysis the muscles are paralysed, and the expression on the diseased side is therefore lost. While many diseases have expressions of the face which are more or less characteristic of them, there are one or two facies which stand out more prominently than the rest, and which deserve special attention.

*The Typhoid Facies*, that, namely, which is met with in the typhoid or adynamic state, is characterised by the following symptoms:—The patient lies on his back, dull, expressionless, and somnolent, his eyes half shut, with the pupils dilated. The face is usually emaciated, the lips black at the edges and trembling, and wide enough apart to show the teeth, which are covered with sordes.

*Facies of Heart Disease.*—In mitral disease, after compensation has been lost, the face is swollen and pale, except the lips and cheeks which are blue, the veins of the neck are engorged, and the mouth is usually half open on account of the dyspnœa. In aortic insufficiency, on the other hand, the chief characteristic is extreme pallor.

*Facies of Inspiratory Dyspnœa.*—Eyes wide, head thrown back, nostrils dilated but not working, mouth half open, face pale. Seen in croup, œdema glottidis, paralysis of the posterior crico-arytenoid muscles, &c.

*Facies of Expiratory Dyspnœa.*—The face is swollen, dark reddish blue, nostrils working powerfully, the eyes injected, and the mouth open. The patient is sitting up with the arms fixed to some support so as to allow the extraordinary muscles of respiration to come into play.

*Facies of Facial Paralysis.*—The unopposed muscles on the side not affected draw the features over towards that side, so that the face has a curious one-sided appearance, which is most noticeable when the patient laughs or speaks. On the affected side of the face the cheek hangs loose, and puffs in and out with each respiration, the saliva trickles from the corner of the mouth, the eye is open and watery, and the whole skin of that side becomes smoothed out and loses its wrinkles.

*Facies of Bulbar Paralysis.*—The face is expressionless, does not move in laughter and crying, the mouth is enlarged transversely, and saliva trickles out of the corners. The naso-labial furrows are well defined.

*Facies of Hectic Fever* is chiefly characterised by a circumscribed flush on the malar bones. This flush is also seen in acute pneumonia, and is said to occur chiefly on the same side as the disease.

*Facies of Cholera.*—In the stage of collapse of cholera the face is sunken and wrinkled, the eyeballs retracted, and the countenance livid.

*Facies of Acute Peritonitis.*—The face is haggard, the expression distressed and anxious. The upper lip is drawn upwards so as to expose the teeth.

*Hippocratic Facies* is the name given to the expression of the face immediately preceding death. The face is pale and livid, the eyes sunken and lustreless, the eyelids separated, the nose sharp and pinched, and the lower jaw falling.

**Attitude.**—The attitude of the patient is very frequently determined by the disease from which he suffers. For example, at the height of a severe fever, the patient may be seen lying on the back very flat, with the face turned upwards; and one of the first indications of improvement, is his wish to be turned on to his side. The round-shouldered appearance of the asthmatic and emphysematous is characteristic; and in bed, those who suffer from dyspnœa are usually obliged to be propped up. Again, a patient suffering from acute peritonitis lies with his knees drawn up; and so with all diseased condi-

tions, almost every one of which compels the assumption of some more or less characteristic attitude.

**Evidence of Previous Injury or Disease.**—Apart from the obvious traces of such surgical affections as fractures, &c., it is always of importance to note any indications of previous disease which may meet us in the course of our examination. The pitting of small-pox, the cicatrices over scrofulous glands, the large joints of those who in their childhood have been subject to rickets, old-standing paralysis, the signs of syphilitic lesions, are among these indications.

**Temperature.**—The thermometer used for estimating the temperature of the body ought to be of such a size, and so divided, that tenths of a degree can easily be read off; it ought to have an arrangement for maximum registration; and the purchaser of a clinical thermometer should see that the accuracy of the instrument in question has been tested by comparison with a standard thermometer, which can be done by sending the instrument to the Kew Observatory.[1]

It is most usual to take the temperature by placing the instrument (the index having been first jerked down below the normal point) in the axilla, the skin of which region must be carefully dried, should there have been any perspiration. The length of time during which the thermometer should be allowed to remain in position depends on the make of instrument used. In any case the observer should make sure, by two or three successive readings, that the highest point has been reached. Still more accurate results are to be obtained, and in shorter time, when the instrument is passed into the rectum. In children the temperature is most readily taken in the mouth, the bulb of the instrument lying under the edge of the tongue.

In all cases of importance the temperature ought to be taken

[1] It is well known that a thermometer, originally correct, loses its accuracy after a certain time, owing probably to molecular changes in the glass. Clinical thermometers ought therefore to be compared with the standard from time to time, at intervals say of two years.

twice a-day, morning (9 to 11 A.M.) and evening (5 to 7 P.M.), and in serious cases every two hours. From the data so obtained, charts should be constructed in the usual manner.

While the *mean normal temperature* may be taken to be 98·6° of Fahrenheit's scale, yet variations from 97·5° to 99·5° are met with in healthy persons. When the mercury stands below the former point, however, it usually indicates a state of collapse. Above 99·5° the reading indicates a condition of fever, which, when it surpasses 105°, is spoken of as hyperpyrexia. More important for diagnosis than the actual reading is the manner in which the temperature rises. Four types of temperature curves are to be distinguished.

1. *Continued.*—The temperature rises rapidly and almost continuously to a given height, at which it remains during the time the fever lasts, subject only to such slight differences between morning and evening temperature as may be observed in health, and then it falls to, or even below, the normal point, as suddenly as it rose. This rapid fall, which is coincident with the commencement of convalescence, is denominated *crisis*. This type is met with in typhus, small-pox, measles, &c., but is perhaps best marked in acute lobar pneumonia.

2. *Remittent.*—In this variety the difference between the morning and evening temperatures is considerable, say, 2° F. or more. Hectic fever in all its forms is perhaps the best example, and it is to be noted that occasionally (especially in phthisis) the ordinary state of matters is reversed, the morning temperature being high, and the evening low. During the first few days of typhoid fever the temperature rises in a characteristic manner, each morning remission being considerably under the temperature of the preceding night, but above that of the preceding morning. In typhus fever, as a rule, the rise is more sudden. In well-marked examples of this type of fever temperature, when the disease takes a favourable turn, the re-establishment of the normal temperature takes place very gradually, and the slow fall of temperature is called *lysis*, to distinguish it from the more rapid crisis of which we have already spoken.

3. *Intermittent.*—This type is seen in the different forms of ague. The temperature rises with great rapidity to a very high point, and falls again in a few hours to normal. There then follows a period of intermission, when the condition of the patient is normal, and then again the temperature rises as before, and so on. The intervals vary in duration according to the type of ague present, lasting twenty-four hours in quotidian, forty-eight in tertian, and seventy-two in quartan ague.

4. *Relapsing or Recurrent Type.*—After a rigor the temperature rises to a high point, near which it remains for some days, and then falls to normal (crisis). An interval of immunity from fever of varied duration (5 to 8 days or longer) succeeds this attack, and then follows a relapse, which corresponds very closely to the original attack. These relapses may occur several times before the disease ceases.

## CHAPTER II.

## ALIMENTARY SYSTEM.

The signs and symptoms connected with the alimentary system may be conveniently considered under two sections. The first of these contains the various objective phenomena which present themselves during the examination of the organs of mastication and deglutition, the subjective sensations of digestion, and the phenomena connected with the expulsion of the food from the body, either by vomiting or by defæcation.

The second group includes all these physical signs which the examination of the abdomen affords.

The first of these sub-divisions will be treated of in the present chapter.

**Lips.**—In examining the lips, we have to note—

(*a.*) *Colour.*—Owing to the great transparency of the labial epithelium, any change in the colour of the blood circulating in the minute vessels underlying it can be readily distinguished. When the lips assume a dusky-blue cyanotic colour, we know that the circulation is being carried on imperfectly, which may be due to defective circulation locally or in the lungs, or to interference with the entrance or exit of air to or from the pulmonary alveoli, or to a combination of these causes. The lips may have a pale waxy colour, indicating that the amount of hæmoglobin is either absolutely less than normal, as in cases of loss of blood, or is both absolutely and relatively (in relation to the aqueous constituents of the body) below the normal standard, as, for example, in chlorosis and other forms of anæmia, as well as in many cases of Bright's disease.

(*b.*) *Form.*—Abnormal thickness or thinness of the lips gives an indication of the amount of serum contained in the interstices of the tissues. The thin pinched lips which are seen, for example, in the second stage of cholera, indicate an abnormal diminution of interstitial lymph. Herpetic eruptions upon the lips (herpes labialis) occur in feverish conditions, particularly in pneumonia; and in syphilis deep and painful fissures are often met with at the angles of the mouth. When we find the lips dry, cracked, and coated with sordes, as in most febrile conditions, we know that the patient has been breathing through the mouth, this causing (in combination with the raised temperature) an abnormally rapid evaporation of the saliva which in health keeps the lips moist.[1] The result of this rapid evaporation is, that the solid constituents of the saliva are deposited on the lips in the form of sordes, whilst the cracking is due to the unequal contraction of the epithelial layer in the act of drying. The fact of the mouth being kept constantly open for the purpose of respiration, although frequently due to more or less complete obstruction of the nasal passages, may also result from any cause which interferes with the proper oxygenation of the blood; the natural tendency in such cases being to attempt to increase the quantity of air respired. All dyspnœic patients, therefore, tend to breathe through the mouth, as offering a freer passage for the ingress of air than the nostrils. The fur on the lips occurs under similar conditions as does that upon the tongue, which will be presently described in detail.

(*c.*) *Movements.*—The lips being among the principal organs of expression of the emotions and the will, many affections of the central nervous system lead to trembling of the lips, as for example, delirium tremens and general paralysis of the insane. Abnormal contraction and relaxation of the labial muscles usually mimics and exaggerates some normal expression. The *risus sardonicus* of tetanus and of strychnine poisoning may be taken as examples of this. The opposite condition is seen in the expressionless appearance which occurs in double facial paralysis.

[1] The amount of saliva secreted is also diminished in fever.

**Teeth.**—The following tables show the periods at which the various teeth usually appear. It is important in treating disease in children to bear these dates in mind.

DECIDUOUS OR MILK TEETH.

| | | | |
|---|---|---|---|
| Lower central incisors | appear | about the | 6th-8th month. |
| Four upper incisors, | ,, | ,, | 8th-10th ,, |
| Lower lateral incisors, | ,, | ,, | 12th-15th ,, |
| Upper and lower canines, | ,, | ,, | 18th-24th ,, |
| Four anterior molars, | ,, | ,, | 12th-14th ,, |
| Four posterior molars, | ,, | ,, | 24th-36th ,, |

PERMANENT TEETH.

| | | | |
|---|---|---|---|
| Central incisors, | appear | about the | 7th year. |
| Lateral incisors, | ,, | ,, | 8th-8½ ,, |
| Canines, | ,, | ,, | 12th-14th ,, |
| Anterior bicuspids, | ,, | ,, | 9th-10th ,, |
| Posterior ,, | ,, | ,, | 11th ,, |
| First molars, | ,, | ,, | 5th-6th ,, |
| Second ,, | ,, | ,, | 12th-15th ,, |
| Third ,, | ,, | ,, | 18th-25th ,, |

These periods of dentition are liable to considerable variation even in health. Sometimes the appearance of the milk teeth is greatly delayed in healthy infants. This is very frequently the result of rickets, and in such cases further evidence of that disease should be carefully sought for.

It is important to observe the *shape* of the teeth. In congenital syphilis the *permanent* central incisors of the upper jaw are very often considerably altered, as was originally pointed out by Hutchinson (see Fig. 1). They are shorter and narrower than natural, peg-shaped, with a crescentic notch at the free edge, sometimes grooved down the centre, and set at greater intervals in the gum than is natural.

*Caries* of the teeth must be noted as bearing upon neuralgia, dyspepsia, &c. The continued and excessive use of mercury gives rise to a looseness of the teeth, and to surface markings due to affection of the enamel.

**Gums and Mucous Membrane of the Cheeks.**—Like the lips, the gums show by an anæmic, bloodless appearance the presence of defective circulation, or diminution of hæmoglobin in the blood. In chronic lead-poisoning a *blue line* forms on the gums close to the dental margin, caused by deposit of sulphide of lead, precipitated there by the sulphuretted hydrogen generated by the decomposition of particles of food remaining about the teeth. Swelling and tenderness of the gums, along with the looseness of the teeth already noted, are among the earliest signs of the action of mercury. Spongy gums which readily bleed are met with in cases of scurvy and purpura. Inflammation of the gums is either general (stomatitis), which may be of parasitic origin (muguet or thrush), or local, constituting gum-boil. Bleeding may also result from the sharp

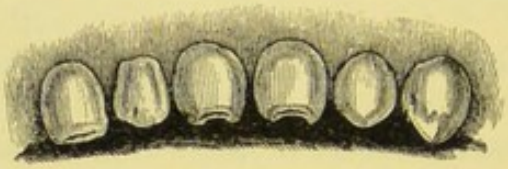


FIG. 1.—Shape of Teeth in Congenital Syphilis.
(After Hutchinson.)

angles of carious teeth, or from the coming away of tartar from the teeth, causing laceration of the neighbouring soft parts. In paralysis of the buccinator muscle the cheek hangs loose, and the food collects between it and the teeth. This condition is seen in facial and bulbar paralysis.

**Tongue.**—In examining the tongue three distinct points have to be considered—(1) its size and shape, (2) its movements, (3) the condition of its surface.

(1.) *Form.*—In health, the tongue varies much in shape, and this without any particular significance. It becomes swollen from various causes, particularly inflammation, the result of small-pox or scarlatina, or the abuse of mercury or other drugs, and from the presence of cancerous or syphilitic new formations. In dyspepsia the tongue has frequently a swollen sodden character, marked at the edges by the teeth against which it has been

pressed, and, even without these causes, when there is great mental hebetude along with defective movement of the organ in the mouth, such œdematous swellings take place. The greatest degree of swelling is however found in cases of acute glossitis, and in macroglossia, often seen in cretins, due to an overgrowth of connective tissue with dilatation of the lymphatics.

(2.) *Movements.*—The tongue receives its motor innervation through the hypoglossal nerve. When the cerebral functions are in more or less abeyance, as in typhus and other severe diseases, the tongue is protruded slowly, with difficulty and with tremor. A tremulous tongue is also met with in drunkards, in cases of general paralysis of the insane, bulbar paralysis, and progressive muscular atrophy.

Paralysis of the tongue is frequent in cerebral affections, (hemiplegia from hæmorrhage, embolism, &c.; bulbar paralysis, general paralysis, the advanced stages of locomotor ataxia, &c.). In unilateral paralysis the tongue when protruded is inclined towards the diseased side. When the paralysis is bilateral, the tongue is relaxed, and wrinkled, and fibrillary contractions may be seen on its surface. The speech then becomes inarticulate and unintelligible.

In chorea, hysteria, and eclampsia, spasm of the lingual muscles is often met with. The peculiar quick spasmodic manner in which the organ is protruded by the choreic patient must be seen to be understood, and once witnessed will never be forgotten.

(3.) *Surface of the Tongue.*—Whenever a patient breathes habitually through the mouth[1] there is a tendency to dryness of the tongue, as well as of the lips (as has been already stated), because of the more rapid evaporation of the saliva and buccal mucus, which usually keep these parts moist. In fever this is still more marked, partly because there is then diminished secretion of these fluids, and partly because the evaporation goes on more quickly on account of the elevation of the tem-

[1] Either on account of obstruction of the nasal passages or from anything which interferes with the proper oxygenation of the blood—dyspnœa in all its forms.

perature. When the tongue becomes very dry, cracks appear on its surface, due to the unequal contraction of the epithelium in the act of drying. A degree of dryness of the tongue is likewise met with in diabetes, and after the administration of certain drugs—atropine, for example.

*Fur on the Tongue.*—In fever when, as has just been said, the saliva evaporates quickly, it deposits its solid constituents upon the lips and tongue. When, however, no abnormal evaporation is taking place, the fur which is found upon the tongue is not to any appreciable extent so formed. It then consists of *debris* of food, of cast-off epithelial cells, and of masses of micro-organisms. These organisms enter the mouth in the air or in the food, are caught on the filiform papillæ, to which they firmly adhere, and there multiply with great rapidity. The fungiform papillæ are too smooth to afford points of attachment, and hence they are usually free from fur, and stand out red and prominent. In scarlet fever these papillæ are more than usually conspicuous, on account of the congestion of the mucous membrane, and they stand out distinctly through the creamy fur which covers the rest of the organ,—the tongue assuming what has been called a "strawberry" appearance. The fur which collects on the lingual surface is being constantly detached by the rubbing of the tongue against the roof of the mouth, gums, and teeth; and as during sleep there is very little movement, the coating is always thickest in the morning. It is also to be remarked that the fur collects most where the tongue is roughest, and where the movement is least in amount—*i.e.*, the centre and

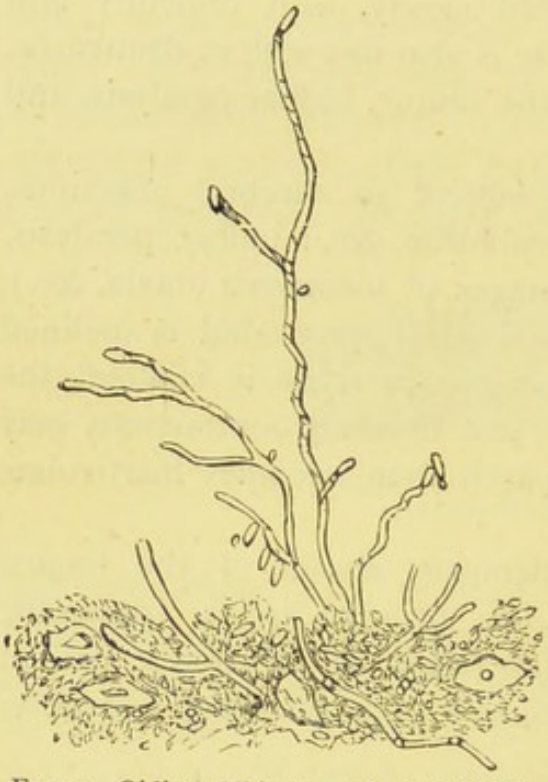


FIG. 2.—Oidium Albicans. (Quain's Dict.)

back part. Increase of fur may result from any condition which diminishes the movements of the tongue on the palate and gums, such as dryness of the mouth, swelling of the tongue, defect of the palate, or disease of the central nervous system (hemiplegia, bulbar paralysis, &c.) paralysing the lingual muscles.

But in addition to the fur which adheres to the tongue, the cavity of the mouth affords a nidus for other forms of vegetable organisms, such as those which give rise to lactic and butyric fermentation of sugar, and acetous of wine. Among these is the parasite which occasions thrush, the *Oidium albicans*. Thrush is usually met with in delicate children, particularly in those who are brought up on cow-milk, and affects the inner surface of the lips, and the mucous membrane of the mouth generally. Small white points first form, which rapidly increase in number, and extend in area, the patches appearing like curdled milk. The reaction of the saliva becomes strongly acid, and thus causes great irritation of the mucous membrane of the mouth, which becomes red, swollen, and painful. When these patches are examined microscopically (see fig. 2) we find, in the midst of a mass of epithelium and salivary corpuscles, the fungus, in the shape of long branching transparent filaments with a double contour, and bearing the spores in a globular receptacle at their extremity. In the meshes of the net-work which these filaments form, there are usually to be seen numerous free spores, which are spherical, and highly refractive.

The colour of the tongue varies considerably under different conditions. In fever it is generally more or less reddened, and it may become blue in cyanosis, or pale where anæmia exists. It must also be borne in mind, that the tongue is liable to be discoloured by particular kinds of food, such as coffee, or milk, and still more markedly by swallowing such medicines as the various preparations of iron.

Finally, the tongue is liable to be the seat of new formations —syphilitic fissures, ulcers, and deposits are frequently met with, and cancerous disease of that organ is not uncommon.

*The sense of taste* will be considered under the nervous system.

**Odour of the Breath** is often an important indication. Offensive breath may be the result of indigestion, but more commonly arises from some disease of the mouth, such as carious teeth, decomposing matter in the crypts of the tonsils, or ulceration of the gums. The peculiarly offensive odour caused by ozæna usually disappears from the breath when the nostrils are compressed and the patient breathes through the mouth alone; of lung affections, those which most commonly cause offensive breath are gangrene and bronchiectasis.

**Saliva.**—The *reaction* of the mixed saliva found in the mouth is normally slightly alkaline, but it becomes acid when retained for long in the buccal cavity, from the formation of organic acids, the result of bacterial action. This is especially the case in dyspepsia, and in diabetes. It is mixed with epithelium and leucocytes, and often contains certain vegetable organisms (*Oidium*). A small quantity of albumen is present in health, and may be much increased in disease. Certain medicines when administered internally appear in the saliva. In particular, the salts of iodine and bromine; urea is also sometimes present in cases of uræmia, but bile and grape sugar are never excreted in the saliva. In disease the saliva may be increased or diminished in quantity—

*Increase of Saliva* (salivation or ptyalism) may arise—

1. From irritation in mouth and throat; as in stomatitis (mercurial or simple), gum-boil, ulcers, dentition, sore throat, &c.

2. From irritation in stomach, pancreas, intestines, uterus; as in cases of dyspepsia, worms, and in pregnancy.

3. From neuralgia, especially of the face.

4. From certain diseases of the brain, medulla, and spinal cord. In insanity, hydrophobia, hysteria, and particularly in bulbar paralysis.

5. From the action of certain drugs, in particular, mercury and jaborandi.

*Diminution of Saliva* is chiefly met with in fevers and in diabetes. It occasionally results from blocking of the salivary

duct with a calculus. It may also be diminished by mental emotions, and by the administration of certain drugs, such as atropin.

**Fauces.**—The examination of the fauces may be conducted in two ways—by inspection and by palpation.

*Inspection.*—The patient should be placed opposite the light, and made to open his mouth widely, while the physician stands in front of him and a little to one side. In most cases it is necessary to depress the tongue by means of a spatula or the handle of a large spoon, and this should be done in such a way that the instrument presses firmly on the horizontal part of the tongue without coming in contact with the soft palate. Sometimes a better view may be obtained by using a reflecting mirror (the laryngoscopic mirror answers admirably), in which case the patient must be placed with his back to the light.

*Palpation.*—Occasionally examination with the finger may be necessary. It is best to stand at the patient's right side, and to introduce the fore-finger of the right hand into the mouth, pressing at the same time with the left hand on the soft parts behind the angle of the jaw. This bimanual method allows the physician to appreciate very accurately the condition of the parts in question.

By a careful use of these two methods we must satisfy ourselves of the condition of the pillars of the fauces, with the tonsils lying between them (in health barely visible); of the soft palate arching up on either side, with the uvula depending from the centre of the posterior wall of the pharynx behind; and of the epiglottis. The mucous membrane of all these parts is moist, and has a red appearance, the colour being particularly deep over the soft palate. On the posterior wall of the pharynx, the vascular ramifications are usually very distinct. In examining the fauces in this way we have to note changes in the mucous membrane in regard to colour, moisture, and smoothness of surface. The presence of abnormal secretions, of ulcers, swellings, tumours, false membranes, and other pathological changes, will thus also become evident. Finally, a careful observation

ought to be made of the movements of the soft palate, and this is best done by making the patient say "ah." We must look for enlargement, inflammation, or ulceration in all these parts, and for changes in the mucous membrane covering them. Apart from mechanical and chemical causes, inflammatory redness of the fauces may occur in many general diseases. It is, however, perhaps most marked in scarlet fever, in which disease throat symptoms occur early. The redness usually beginning in the middle of the soft palate quickly spreads over the whole mucous membrane of the fauces, and becomes very intense, being accompanied with considerable swelling of these parts. The other exanthemata are also frequently accompanied with sore throat, but the redness is less intense. In secondary syphilis an erythematous reddening of the fauces is not uncommon, the eruption being often distributed symmetrically on either side of the soft palate. More chronic inflammatory conditions give rise to a dark-red appearance of the mucous membrane, in which the follicles may be more conspicuously affected; and care must be taken not to mistake the distended mouths of follicles for ulcers. In acute tonsillitis the tonsil on the affected side and the tissues surrounding it become much swollen.

*Ulceration* of the throat may be follicular, or may arise from syphilis, tubercular disease, diphtheria, carcinoma. Septic ulceration occasionally occurs in connection, for example, with erysipelas.

*Diphtheria*, caused by the Klebs-Löffler bacillus, is the most important of all the affections which are met with in this neighbourhood. The tonsils are usually the first part attacked. They become red and swollen, and over the mucous membrane covering them there forms a false membrane, which is at first white, but which gradually assumes a dirty greyish colour. This membrane quickly spreads to the soft palate and uvula, and ultimately to the posterior wall of the pharynx, so that at last the whole fauces may be overspread. When at any point this false membrane is forcibly detached, the mucous membrane below it will be found denuded of its epithelium, raw and bleeding. Over this area the false membrane quickly becomes renewed. The diph-

theritic false membrane must be distinguished from certain other pathological conditions which more or less closely resemble it, such as patches of secretion exuded from the crypts of the tonsils, or layers of purulent secretion on the fauces in chronic catarrh.

This is best done by means of bacteriological examination. A shred torn from the false membrane should be transferred to a sterile test-tube, or, if the membrane cannot be obtained, a swab of cotton wool should be swept over the fauces, and, with the adherent secretion, transferred to the test-tube. Microscopic examination of the membrane or of the section so obtained, is often sufficient for diagnostic purposes, the bacillus showing itself clearly in film preparations, stained for two or three minutes with Löffler's solution. In other cases, however, cultivations have to be made before an accurate diagnosis is possible. On serum-agar the colonies appear when the tubes have been from twenty to twenty-four hours in the incubator. Films prepared from the cultures show the organism clearly.

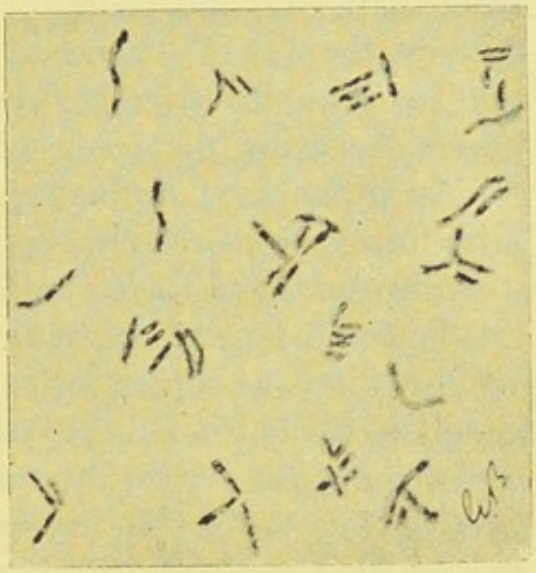


Fig 3.—Bacillus Diphtheriæ, from 24 hours' culture, $\frac{1}{12}$ oil immersion.

The Klebs-Löffler bacillus varies considerably in length. The rods are slender, usually clubbed at one or both ends, more or less curved in shape, and often granular in appearance from the unequal staining of the dye. They are arranged, not in chains, but irregularly. This is not the place to discuss the relations of the pseudo-diphtheritic bacillus, for which special works on bacteriology should be referred to.

Under circumstances where no bacteriological examination is possible, the diagnosis of diphtheria is founded upon the severe general symptoms which present themselves,—the swelling of the cervical glands, the great prostration without high fever, and the

albuminuria. A slight or doubtful case is often shown to have been diphtheria by the subsequent occurrence of post-diphtheritic paralysis, which is usually found in connection with the soft palate (showing itself by the regurgitation of fluid into the nostrils during swallowing), but sometimes affects the limbs.

Tumours in the region of the fauces are usually more of a surgical than of a medical nature. Reference may, however, be made to nasal polypi which sometimes hang down into the throat, to adenoid growths, to tumours of the epiglottis which may show themselves above the root of the tongue, and to the various forms of malignant growths which may be met with in connection with the pharynx. These usually present little difficulty in the way of diagnosis. Bulging forward of the posterior wall of the pharynx may be produced by retro-pharyngeal abscess—usually the result of caries of the cervical vertebræ. Similar prominence in this neighbourhood may, in rare instances, arise from carotid aneurism.

*Movements of the Soft Palate.*—The muscles of the soft palate are innervated partly from the facial nerve by the fibres which pass by the large superficial petrosal nerve to the sphenopalatine ganglion, partly from the spinal accessory nerve and partly from the trigeminus. When the palate is paralysed in cases of facial paralysis, it shows that the lesion is above the geniculate ganglion, as the large superficial petrosal nerve branches off at that point. Occasionally the paralysis of the palate is of central origin, as in hemiplegia, but more frequently it is due to some affection of the nerves in their course, or is peripheral in its origin. Perhaps the most frequent cause is diphtheria. The appearance which the palate presents depends upon the degree of its involvement. When one lateral half of the palate is paralysed, it will be seen that the affected side is further forward, that the raphe is drawn backwards and towards the sound side, and that the arch of the palate has become non-symmetrical, the highest point lying more posterior and nearer the sound side. When the paralysis is bilateral, the palate is lax and immobile, and hangs far forward. Rarely, cases come under observation in which the paralysis is limited to one or two of the palatine

muscles, as, for example, the azygos uvulæ. When that muscle is paralysed the uvula usually becomes bent round to the sound side.

Diphtheritic paralysis of the palate affords the best example of the symptoms which follow such paralysis. When the soft palate is so far paralysed as to prevent the proper closure of the posterior nares, it is found that, during swallowing, the fluid passes into the nose and streams out through the nostrils. The speech also becomes nasal and indistinct. Anæsthesia of the palate often accompanies paralysis. Hyperæsthesia is very common as the result of chronic catarrh, abuse of alcohol or tobacco, and particularly in hysteria.

**Mastication** may be rendered difficult or painful by the presence of inflammatory affections of the lips, gums, cheeks, or tongue, by defective teeth, by cancerous or other ulceration of those parts, or by paralysis or spasm of the muscles employed in the act. The buccinator and the orbicularis oris receive their motor supply from the seventh nerve; and when that nerve is paralysed, the food accumulates between the cheek and the teeth. The motions of the tongue are affected in paralysis of the hypoglossal nerve. The muscles of mastication (masseters, temporals, pterygoids) are innervated by the motor branch of the fifth nerve. Their movement may be interfered with in two ways, either by spasm or by paralysis. *Masticatory spasm* may be tonic or clonic. The tonic form, one of the initial symptoms of tetanus, shows itself by the continuous and rigid contraction of the muscles of mastication, resulting in firm closure of the mouth, the lower jaw being drawn upwards in contact with the upper, and at the same time forced somewhat backwards. Clonic spasm results in the rapid rising and falling of the lower jaw, which produces a sort of chattering of the teeth similar to that seen in ague. Masticatory spasm may be due to such affections as chorea, epilepsy, and hysteria, besides tetanus, as already mentioned. It may also arise reflexly from some peripheral irritation of the fifth nerve. *Masticatory paralysis* shows itself in inability to chew, which is more or less pronounced according

as the condition is bilateral or unilateral. It usually owes its origin to central causes, such as bulbar paralysis, and affections of the cortex cerebri.

**Deglutition** may be rendered difficult and painful by various affections of the mouth and tongue, such as swelling, ulceration, inflammation, &c., but apart from such temporary causes, it may arise from paralysis of various groups of muscles. For purposes of description, the act of deglutition may be divided into three stages: (1.) The gathering up of the food into a bolus and thrusting it through the anterior pillars of the fauces. (2.) Its passage through the upper part of the pharynx until it has passed the orifice of the larynx. (3.) Its descent through the lower part of the pharynx and the œsophagus. The first stage of deglutition is interfered with in paralysis of the hypoglossal nerve, for the tongue cannot then form and force back the bolus into the fauces. Such paralysis is almost invariably of central origin. In the second stage of deglutition, disease of the facial nerve above the geniculate ganglion, by causing paralysis of the soft palate, allows food to pass into the posterior nares, and the same symptom is present in post-diphtheritic paralysis, in syphilitic ulceration of the palate and epiglottis, and in cleft palate. This second stage of swallowing may also be interfered with by paralysis of the muscles of the pharynx. The food sticks at the root of the tongue, and occasions such dyspnœa as to require its removal by means of the finger, and fluids pass readily into the larynx. This paralysis, rare as a peripheral disease, is not uncommon as a result of affections of the pons and medulla, and of diseases of the base of the brain, compressing the cranial nerves. The third stage of deglutition may be interfered with by mechanical obstruction of the œsophagus (impacted foreign bodies, pressure of aneurism or other tumour, simple or cancerous stricture, &c.), or by paralysis of the muscles of the œsophagus, which is very rare as an isolated affection. In the latter case, solids may manage to make their way down the tube by their own weight. Spasm of the œsophageal muscles sometimes prevents deglutition in cases of hysteria.

*Examination of the œsophagus* is practically limited to mediate palpation by means of the œsophageal bougie. The instrument should be warmed, lubricated (preferably with glycerine or white of egg), and, held in the right hand as one holds a pen, passed gently over the back of the patient's tongue, being guided by the left forefinger of the operator over the epiglottis, and then gently and steadily pushed into the stomach. The instrument may catch at the upper end of the œsophagus, but, on the patient being directed to make the movement of swallowing, this difficulty is usually overcome. Of the difficulties and dangers which the operator may meet in the course of this exploration, the following are the most important:—The œsophagus should never be sounded until there is certainty that no aneurismal tumour is pressing upon it, into which the point of the tube might be forced. In ordinary circumstances there is but little danger of the instrument passing through the glottis into the trachea, but whenever paralysis and anæsthesia of the laryngeal structures exists, very great care must be taken. It is hardly necessary to warn the student of the danger of forcing the sound through the œsophageal structures. Such an accident has happened not unfrequently, and it is of course more ready to occur when the walls are softened by cancerous deposit.

The œsophagus proper commences about 6 inches from the incisor teeth, from which the lower end is distant about $15\frac{1}{2}$ inches. It is slightly narrowed at three points; (*a*) at its upper end, (*b*) at a point about $2\frac{3}{4}$ inches lower where the fœtal diverticulum existed, and (*c*) where it pierces the diaphragm.

In sounding the œsophagus we may meet with—

1. *Pain.*—If the pain be felt again and again at the same spot, it indicates a local process, probably of an inflammatory nature. The presence of ulceration may be conjectured if the sound, however carefully it has been introduced, always comes away smeared with blood.

2. *Obstruction.*—This may be caused by the point of the sound finding its way into a diverticulum, and it is characteristic of this condition that the instrument sometimes passes with great ease, and sometimes is absolutely arrested. Strictures of various

kinds also prevent the passage of the sound. The purely spasmodic strictures met with in hysteria may be distinguished from those due to organic disease, by placing the hysterical patient under the influence of chloroform, when an instrument which before was obstinately resisted, now passes freely into the stomach.

In all cases where obstruction is encountered the exact distance from the incisor teeth at which the bougie was arrested should be noted.

*Auscultation of the Œsophagus* was first employed as a means of diagnosis by Hamburger. The stethoscope is to be applied a little to the left of the spinal column in the cervical or dorsal region, and the sound listened to which arises in the œsophagus when the patient swallows water. In health, the act of deglutition is accompanied with a short, clear, gurgling sound. When, however, œsophagal obstruction exists, this sound is prolonged, and altered in character below the seat of the stricture. This method of examination is of little clinical value.

**Appetite.**—Derangements of the appetite may occur both in general diseases and in local affections of the stomach and intestines.

*Anorexia*, or loss of appetite, is present during all acute febrile diseases, and may also result from excessive fatigue of mind or body, or from depressing emotions, such as pain or grief, as well as from the use of narcotics or alcohol. It is also caused by inflammatory affections of the stomach, cancer of the gastric walls, constipation, and other abnormal conditions of the intestines.

*Boulimia*, or excessive appetite, may result simply from the habit of over-eating, or may be due to the presence of worms in the stomach or intestines. It is also present in certain chronic inflammatory conditions of the gastric mucous membrane, is a prominent symptom in the course of diabetes, and occasionally occurs in various nervous disorders (insanity, hydrocephalus, epilepsy, hysteria, and hypochondriasis).

*Pica*, or depraved appetite, in which the patient craves for arious abnormal and even disgusting articles, is sometimes met

with during pregnancy, in patients suffering from chlorosis, in hysteria, in mania, and in the case of idiots.

**Thirst** almost invariably attends all feverish states of the system. It is a marked symptom in diabetes; and in irritative conditions of the stomach thirst commonly appears some hours after eating. Other conditions which give rise to thirst will be alluded to under the head of the Urinary System.

**Sensations during Fasting.**—In the atonic form of chronic dyspepsia there is frequently before meals a feeling of sinking in the epigastrium, along with faintness. Pain, when the stomach is empty, is sometimes due to cancer or gastric ulcer, although in the latter affection the greatest pain is usually felt after food has entered the stomach. Over-secretion of gastric juice may give rise to pain when the stomach is empty, which is then usually relieved by eating.

**Sensations after Eating.**—Painful sensations after food is swallowed, which are referred to the gastric region, vary from the slightest feeling of discomfort or oppression up to the most severe agony. The pain may be due to—

1. *The Presence in the Stomach of Irritating Substances.*—This irritation may be mechanical, when indigestible food has been swallowed; or chemical, due to the presence of corrosive poisons, or, more commonly, of the various abnormal products which are formed in the stomach when digestion is imperfect. In its slighter forms, such irritation gives rise merely to a feeling of weight, discomfort, or distension; in its graver varieties the pain may be very great.

2. *Organic Diseases of the Gastric Wall.*—Of these the most important are cancer and gastric ulcer. In gastric carcinoma the pain may be very severe. It is chiefly referred to the epigastrium and is usually more or less continuous, not being increased by taking food. In cases of gastric ulcer, on the other hand, the pain becomes very severe almost immediately after food has been swallowed, and continues for a considerable time, though, as a rule, the patient is free from pain when the stomach

is empty. The pain is usually sharply localised, and is increased by external pressure.

3. *Nervous Causes.*—Pain in the stomach is occasionally of a purely neuralgic character, due sometimes to hyperæsthesia of the gastric mucous membrane, at other times to changes in the brain or spinal cord. The most marked example of the latter variety of gastralgia is to be found in the "gastric crisis" of locomotor ataxia.

It is often very difficult to distinguish the pain of gastralgia from that of gastric ulcer and of carcinoma. The chief points are that the pain of gastralgia is more diffused than is the case in the other conditions. Nor is it, as a rule, dependent on taking food, indeed it is often relieved by eating and also by pressure over the stomach which in the other two affections increases the pain. The general aspect of the case, the sex of the patient, and, in particular, a neurotic history, will further help to make a correct diagnosis.

The name *heartburn* has been given to a peculiar pain of a hot or scalding character, referred usually to the region of the epigastrium. It is due to abnormal digestion, and is frequently accompanied by acid eructations.

**Flatulence and Eructation.**—The collection of gas in the stomach and bowels may result either from the swallowing of air in excessive quantities, or from fermentative changes in the contents of the stomach and intestines. It usually arises as a result of perversion of the digestive process in some of its various actions. This gaseous distension of the stomach and intestines occurs most readily in cases in which the muscular walls of these viscera have become more or less paralysed—as in hysteria and the more severe forms of fever, particularly in typhoid and puerperal fevers. When it reaches a considerable degree, it is called tympanites or meteorism; and its seat, whether in the stomach or the bowel, may be determined by percussion, as will be afterwards explained. Meteoric distension often interferes with respiration, owing to the upward pressure which it exerts on the diaphragm. This acts in two ways, partly by com-

pressing the lungs and limiting the respiratory movements, and partly by mechanically interfering with the free motion of the heart. This distension is usually relieved by the expulsion of the gas either by the mouth (eructation) or the anus. Eructation usually takes place suddenly and with some force, so that the gas, rushing up the œsophagus into the pharynx, often carries with it some of the liquid or solid contents of the stomach.

**Colic** is a frequent accompaniment of flatulence. Its chief symptom is pain, which is usually situated near the umbilicus, but which diffuses itself in different directions. This pain, which is generally of a sharp, twisting character, comes in distinct paroxysms, which gradually commence, reach a maximum, and as gradually fall away again. This character of the pain, as well as the slowing of the pulse which accompanies it, and the normal temperature, suffice to distinguish colic from peritonitis, rheumatism, &c.

**Methods of directly testing the Digestive Power.**—Much more trustworthy for diagnostic purposes than these subjective sensations is the examination of a specimen of the contents of the stomach, removed for that purpose by means of a stomach tube. In many cases this method will give trustworthy indications as to diagnosis, prognosis and treatment. The contents of the stomach are drawn off by means of a soft rubber tube, following the directions on p. 33.

So as to be able to draw accurate conclusions as to the activity of digestion from the chemical examination of the gastric contents, it is necessary to place the patient under known conditions, *i.e.*, to give him, on an empty stomach, a meal of a simple character, and one with regard to which the behaviour of a healthy stomach is well known. Many such test meals have been employed. The simplest, and probably the best is the test-breakfast of Ewald, which consists of 70 grammes of white bread and two cups (400 c.c.) of weak tea, without milk or sugar. This meal should be taken about eight o'clock, and the stomach contents removed one hour thereafter.

Under normal circumstances the quantity of gastric contents so obtained is about 30 to 50 c.c. If very much more than this is removable from the stomach, say 100 or 200 c.c., then either motor insufficiency or hypersecretion is to be suspected, and the latter all the more if there be much free hydrochloric acid.

The gastric contents so obtained, should be filtered and subjected to the following tests.

### Acidity of Gastric Contents.

The gastric contents, under normal circumstances, give an acid reaction when tasted with litmus paper. This acidity may be found to be present as early as a quarter of an hour after a meal, being then due, not to the presence of free hydrochloric acid, but to lactic acid or to acid salts. Up to this time the hydrochloric acid secreted has all combined with the proteids of the food. Later on, under normal circumstances, the lactic acid disappears and free hydrochloric acid takes its place, reaching its maximum about an hour after eating. Under pathological conditions considerable variations occur, and the acidity of the contents, when withdrawn as described one hour after a test meal, may depend on the presence of hydrochloric acid, free or combined with proteids, on acid salts, particularly acid phosphates, or on organic acids.

Before proceeding to ascertain on which of these substances the acidity depends in any particular case, it is well to determine the

**Total acidity of the Gastric Contents.**—This is readily estimated by titration with a deci-normal solution of caustic soda, ascertaining what quantity of the solution is required to neutralize a given quantity of stomach contents. To 10 c.c. of the filtered gastric contents, carefully measured into a small beaker, 40 c.c. of distilled water are added, and the mixture coloured red with neutral tincture of litmus. From a burette the deci-normal solution of soda is allowed to fall into this mixture until its colour changes to violet, which marks the end of the reaction. After reading off on the burette the number of c.c. of the soda

solution which have been used, the percentage acidity is easily calculated. Thus, if 6·4 c.c. have been required to neutralize 10 c.c. of the gastric contents, it is clear that 64 c.c. would have been required for 100 c.c., and the total acidity is therefore 64 per cent. (If it is desired to calculate this total acidity in terms of hydrochloric acid this is readily accomplished by remembering that 1 c.c. of the deci-normal soda solution corresponds to 0·00365 gramme of hydrochloric acid.)

The total acidity, after Ewald's test-breakfast, is normally from 50 to 60 per cent. The deductions to be drawn from an increased or diminished acidity will be referred to presently.

The determination of the total acidity does not, however, tell us on what that acidity depends. It may be due, as has been said, to free hydrochloric acid, to hydrochloric acid loosely combined with proteids, to acid salts (particularly acid phosphates), or to organic acids, lactic, butyric, acetic. We therefore proceed, first to the detection, and second to the estimation of these substances.

**Detection of free hydrochloric acid.**—Many reagents are employed for this purpose. The best is that of Günzburg, prepared by dissolving 2 grammes of phloroglucin and 1 gramme of vanillin, in 30 grammes of absolute alcohol. A few drops of this solution, mixed with an equal quantity of the filtered gastric contents, placed in a porcelain capsule, and evaporated to dryness over a flame, leave a crimson residue if hydrochloric acid have been present.

**Detection of lactic acid.**—Uffelmann's reagent may be employed for this purpose. This reagent, which should be freshly prepared just before use, consists of a mixture of 20 c.c. of a 1 per cent. solution of carbolic acid, with one drop of tincture of perchloride of iron. It possesses an amethyst blue colour, which changes to lemon yellow on the addition of lactic acid. It is sometimes better, before performing this test, to extract the gastric contents with ether (which takes up the organic acids),

to evaporate off the ether on a water-bath, and to dissolve the residue in water.

**Detection of butyric acid.**—This acid can usually be recognised by its odour, that of rancid butter. It gives a yellowish brown turbidity with Uffelmann's reagent.

**Detection of Acetic Acid.**—It can usually be recognised by its odour. If further proof is required, an etherial extract of the gastric contents should be made, the ether evaporated, and the residue dissolved in water and neutralised with carbonate of soda. If now a drop or two of a dilute solution of perchloride of iron be added, a blood-red colour will be produced if acetic acid be present.

We now pass to the consideration of the quantitative estimation of these substances, of which hydrochloric acid is much the most important from a clinical point of view. So far as concerns clinical investigation, hydrochloric acid, as has been said, is present in two forms—(1) as free hydrochloric acid and (2) as hydrochloric acid in loose combination with proteids. It has been pointed out that the hydrochloric acid, which is secreted from the gastric mucous membrane, enters at first into combination with the proteids of the food, and that it is only after this action has been fully accomplished that the acid appears in a free state in the gastric contents. It is therefore clear that, when one wishes to know how much hydrochloric acid has been secreted —and this is by far the most important point for clinical purposes—it is necessary to estimate the free and the combined hydrochloric acid together. As, however, it is also of interest to know the amount of free acid present, a method of doing this is also given.

**Estimation of Free Hydrochloric Acid.**—The method of Mintz is as follows:—Measure 10 c.c. of the gastric contents into a porcelain capsule, and add slowly deci-normal solution of caustic soda from a burette until a drop of the mixture, removed and tested with the phloroglucin-vanillin solution already mentioned,

fails to give the characteristic reaction, thus showing that all the free hydrochloric acid has been neutralised. From the amount of soda solution used, the calculation is readily made, as before described.

**Estimation of the Free and Combined Hydrochloric Acid.**—This is best performed by means of the method of Sjöquist, which is as follows:—Place 10 c c. of the gastric contents in a platinum capsule, colour with neutral tincture of litmus, and add pure carbonate of barium until the mixture is neutralised. The hydrochloric acid, both free and combined, combines with the barium carbonate to form barium chloride, and, if organic acids are present, they also form barium salts. The mixture is now to be evaporated on a water-bath, and the residue gently charred, whereby the organic barium salts are broken up, leaving insoluble barium carbonate, while the barium chloride remains. Wash the residue with hot water on a filter. The filtrate will contain the barium chloride, which represents all the hydrochloric acid which has been secreted by the gastric mucous membrane.

The estimation of the barium chloride is performed by titrating with a solution of pure bichromate of potassium (8·5 grammes to a litre). The filtrate is mixed with 4 c.c. of a solution containing 10 per cent. of acetic solid and 10 per cent. of sodium acetate, and the bichromate solution run in from a burette until all the barium is precipitated. This point is indicated by the turning distinctly blue of "*tetra*" paper (an abbreviation for *tetramethyl-paraphenyl-diamin*), when dipped in the mixture. The calculation is made readily enough, 1 c.c. of the bichromate solution corresponding to 0·00405 gramme of hydrochloric acid.

Of much less clinical importance is the

**Estimation of Lactic Acid.**—Take 10 c.c. of the gastric contents, and remove the albumin by adding a few drops of dilute sulphuric acid and heating. Evaporate the filtrate on a water-bath to the consistency of syrup. Add a little distilled water, and evaporate to dryness. This removes the fatty acids. Extract the residue with 100 c.c. ether. Evaporate the ether on a

water-bath, and dissolve the residue in distilled water. Titrate with the deci-normal solution of soda, using phenol-phthalein as an indicator. Each cubic centimetre of the soda solution corresponds to 0·09 gramme of lactic acid.

Of the formation of the hydrochloric acid secreted by cells in the fundus of the stomach, it need only be said, without going into details, that it is entirely derived from the chlorides of the blood. Although without its presence peptic digestion cannot go on, to aid digestion is not its only, nor perhaps its chief, rôle. Its presence in the stomach is one of those safeguards which the organism uses to defend itself against disease, for hydrochloric acid is a germicide, and, in presence of the quantity contained in normal gastric juice, many putrefactive and pathological organisms are destroyed. That the cholera spirillum is thus destroyed, Koch showed some years ago. The bacillus of tubercule, and that of anthrax, are more resistant.

The clinical significance of *variations in the quantity of hydrochloric acid* in the gastric contents may be shortly stated as follows :—The normal amount of free and combined hydrochloric acid found in the stomach contents one hour after Ewald's test-breakfast is from 0·1 per cent. to 0·2 per cent. In certain diseases of the stomach, such as in nervous dyspepsia, in simple atony of the muscular walls of the stomach, and occasionally in gastric ulcer, this normal amount of acid may be found.

*Increased amount of hydrochloric acid* (*Hyperchlorhydry*) is seen in cases of hypersecretion, sometimes in the gastric neuroses of the neurasthenic, and very frequently and importantly in gastric ulcer.

*Diminished amount of hydrochloric acid* (*Hypochlorhydry*) is found where the secreting cells of the fundus have suffered, as in chronic gastritis, and in cases of dilatation of the stomach. It is also found in febrile conditions, in chronic wasting diseases, such as phthisis, in chlorosis and other forms of anæmia, and in the earlier stages of gastric carcinoma.

*Absence of hydrochloric acid* (*Anachlorhydry*) is found in many cases of carcinoma of the stomach, and, indeed, is very suggestive of that disease. It also occurs in the advanced stage of

chronic gastritis, in scorbutus, in severe fevers, and occasionally in hysterical and neurasthenic conditions.

*The Ferments of the gastric contents* are two in number, pepsin and milk-curdling ferment or rennet. To test peptic digestion it is usual to place about 20 c.c. of the filtered gastric contents in a small flask, to acidulate, if necessary, with hydrochloric acid, and to add some shreds of fibrin, or small discs of white of egg cut with a cork-borer. If the flask is placed in an incubator at 38° C., about 0·05 gramme of fibrin should, under normal circumstances, be digested in an hour and a half, and the same quantity of egg albumen in about three hours.

Milk-curdling ferment is invariably to be found in the gastric contents, and its presence can be readily demonstrated by adding an equal quantity of the contents of the stomach to 10 c.c. of fresh neutral milk. In an incubator, curdling should take place in about a quarter of an hour.

*The rapidity of absorption possessed by the stomach* may be tested by causing the patient to swallow a capsule containing iodide of potassium, and testing the saliva from minute to minute for the appearance of that substance, using paper moistened with starch solution, to which a drop of nitric acid may be applied. If the stomach is empty the reaction should be obtained in ten to fifteen minutes. In cases of catarrh, dilatation, carcinoma, &c., considerable delay may be noted.

*The motor power of the stomach* may be judged of by its state as to contents six or seven hours after a meal. At that time it should be empty, and if, on the contrary, it is then found to contain any considerable amount of food, a loss of motor power may be inferred.

The motor power may further be tested by means of salol. This substance, unchanged so long as it remains in the stomach, becomes at once broken up into salicylic acid and phenol when

it reaches the intestine and meets the pancreatic juice. The former of these appears rapidly in the urine as salicyluric acid, giving a violet reaction with chloride of iron. Accordingly, if salol be given during a meal, the appearance of salicyluric acid in the urine will approximately mark the time when the substance has passed through the stomach and into the intestine. With 15 grains of salol, the reaction should be found in the urine in sixty to seventy minutes, under normal circumstances, and the last trace should disappear from the urine in about twenty-seven hours. In dilatation of the stomach, and in pyloric obstruction, considerable delay may occur.

**Nausea and Vomiting.**—The expulsion of the contents of the stomach in vomiting is caused by the forcible contraction of the abdominal muscles, diaphragm, &c., the glottis being tightly closed. It is a reflex act, having its centre in the medulla oblongata, close to and intimately associated with the respiratory centre. Vomiting may be produced in many different ways, of which the following are examples: (1) It may be caused by diseased processes involving the vomiting centre in the medulla, such as sclerosis and softening. The vomiting of hysteria, often very persistent, may be due to an increased irritability of this centre; (2) Certain substances, circulating in the blood, have the power of irritating the reflex centre sufficiently to cause vomiting. In this way certain of the emetics act, such as apomorphia, emetin, tartar-emetic, and the vomiting of uraemia is due to a like cause. Perhaps the vomiting seen in the commencement of fevers may also be thus explained, the toxines acting on the centre; (3) The centre may also be irritated by compression, a common result of the increase of intra-cranial pressure produced by tumours of the brain; (4) It has been said that the vomiting centre lies in close association with the respiratory centre, and when the latter is over-excited in efforts to produce cough, as often occurs, for example, in phthisis and in whooping-cough, the stimulation is apt to pass to the vomiting centre and to produce vomiting; (5) Most of the causes of vomiting, however,

act reflexly. From the meninges of the brain, in cases of meningitis, a stimulus passes through the branches of the trigeminus to the reflex centre and causes vomiting, and through unknown paths other cerebral conditions may produce the same result. In this way arises the vomiting which results in some persons from a disgusting odour or a sickening sight. But mainly the reflexes which excite vomiting pass through the branches of the vagus. Tickling of the fauces is well known to cause vomiting. Still more common causes are to be found in connection with the mucous membrane of the stomach. The cause of the irritation is frequently the presence of undigested food, or the products of its decomposition. This is seen in cases of acute and chronic gastritis, and of dilatation of the stomach. Vomiting also occurs in gastric ulcer (usually shortly after food), and in carcinoma. In peritonitis and in many conditions of the intestines, vomiting is apt to occur. For example, in appendicitis and cholera, and particularly in cases of intestinal obstruction it is a very important symptom. The reflex may also be started by the irritation of the passage of a gall-stone, or of a renal calculus, and various uterine conditions, such as pregnancy, may give rise to it. Gastric vomiting is usually preceded by nausea and pain, and bears some relation to the food swallowed; whereas, when this symptom arises from cerebral causes, these are frequently absent.

**Vomited Matter.**—This consists of the contents of the stomach and sometimes of the duodenum. The fluids and solids vomited are more or less acted upon by the gastric juice, and are usually strongly acid. The chemical examination has been described in the previous pages in speaking of the gastric contents.

The condition of the vomited matter may give important indications as to the amount of decomposition going on in the stomach. This may be inferred from the presence of butyric and acetic acids, and, further, if many micro-organisms are present, such as sarcinæ, yeast, bacteria. Much decom-

position depends either on deficiency of hydro-chloric acid, or on defective mobility of the stomach, so that the food remains long there. It is seen in cases of chronic gastritis and dilatation of the stomach.

Of abnormal substances present the most important is *blood*, which may be almost pure, but is generally more or less acted upon by the gastric juice, and thereby coagulated and darkened so as to resemble the grounds of coffee, the hæmoglobin being broken up into globulin and hæmatin. Hæmatemesis (the vomiting of blood) is most frequently met with in connection with gastric ulcer or carcinoma; but it also occurs in blood diseases (yellow fever), in congestion of the veins of the stomach (cirrhosis of the liver, pressure on the inferior vena cava), and sometimes vicariously, when the menstrual flow is arrested. Finally, it may result from wounds of the stomach, or from the bursting of an aneurism into the stomach or œsophagus. There are mainly two conditions which are apt to be mistaken for hæmatemesis — viz., bleeding from the nose, and bleeding from the lungs. The former is only difficult of diagnosis in cases where the blood has been first swallowed and then vomited again, but an examination of the nose and throat will almost always make plain the source of the hæmorrhage. Bleeding from the lungs is more difficult to distinguish from hæmatemesis. The main points of distinction will be enumerated under the head of "Sputum." Bile is frequently found in the vomited matter, rarely pus, and still more rarely fæcal matter. The latter usually points to intestinal obstruction, although it may occur independently of such a condition when the bowel is paralysed as a consequence of peritonitis or typhoid fever.

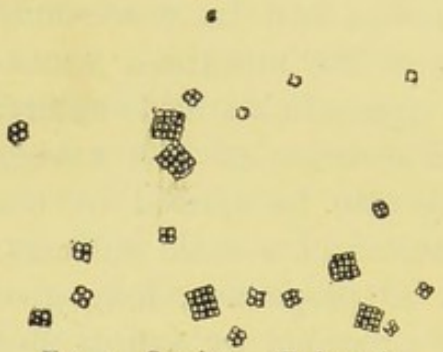


FIG. 4.—Sarcinæ. (Roberts.)

**Defæcation.**—While normally defæcation occurs with regularity once in twenty-four hours, it is not uncommon to find persons

who have two motions in that period, or whose bowels act only once in two days, without the bounds of health being overstepped. In infants, the bowels move frequently—four or five times a day. The following points have to be inquired into :—

1. *The Frequency of the Motions*, and the period at which the bowels act relatively to eating, drinking, exercise, &c.

2. *The Character of the Act of Defæcation.*—Faintness or sickness may precede the act, which may itself be painful and straining, and may be followed by a sensation of the rectum not having been emptied of its contents (tenesmus). In doubtful cases the actual condition of the anus and rectum should be determined, and the presence of piles, fissure, prolapse, ulceration, &c., looked for.

The two conditions of constipation and of diarrhœa demand brief notice.

*Constipation* may result from—

1. Mechanical obstruction ; and this may be caused in various ways, such as from accumulations of various kinds in the bowel, by cicatricial or cancerous stricture, by external compression of the intestines, by strangulation or intussusception, and by spasm or paralysis.

2. Defective peristaltic action.

3. Deficiency of the secretions.

These two last causes may arise from too frequent use of purgatives, from neglect of the regular performance of the act of defæcation, from the abuse of opium, from sedentary and from enervating habits, as well as from derangement of stomach and liver, and from many other causes too numerous to mention.

*Diarrhœa.* — The causes of diarrhœa may be grouped as follows :—

1. While the intestinal canal is normal, diarrhœa may be excited by abnormally strong stimuli, such as improper food, hardened fæces, purgatives, or the presence in the bowels of toxines in course of elimination from the body in such diseases as uræmia and gout. If decomposition of the food has occurred in the stomach or intestines, the toxines thereby produced will act in the same manner.

2. There may be abnormal irritability in the intestinal nervous system to such an extent that the normal stimulus produces so much peristaltic action as to lead to diarrhœa. This condition is found in connection with nervous disease, and in individuals of a nervous temperament. It further occurs when the nerves of the bowel are laid bare by ulcerative processes, and are thus more easily irritated.

3. Pathological conditions of the mucous membrane frequently lead to the pouring out of much secretion into the bowels. This group includes all the grave affections of the intestinal tract, and also many general diseases. In the case of cholera, typhoid fever, dysentery, and tubercule, the diarrhœa is caused by the action of microbes.

**Character of the Fæces.**

1. *Macroscopic Characters.*

(*a.*) *Colour.*—The normal colour of the fæces may be altered by reason of the food eaten: becoming light in colour with milk diet, and dark brown when much meat is taken. Still more marked is the influence of various medicinal agents upon the colour. Iron, charcoal, and bismuth, when taken internally, blacken the motions, while the administration of calomel causes a green, and of logwood a red-brown, colour to appear in the fæces. If all these causes of altered colour be excluded, then the alteration is dependent upon bile or upon blood. The presence of bile gives rise to a yellow or to a green tint, and in its absence the fæces assume a grey or chalky appearance.[1] Where there is profuse diarrhœa the evacuations become very pale in colour, owing to the dilution of the bile. The "rice water" stools of Asiatic cholera probably owe their want of colour in great measure to this cause. In normal fæces, the reaction which is characteristic of bile pigment (see chapter on Urine) cannot be obtained; but when, from whatever cause, the peri-

[1] This chalky appearance is not merely to be traced to the absence of bile pigment, but is likewise due to an abnormal quantity of fat, the result of the disturbance of the digestion of fat, in which process bile plays an important part.

staltic action of the small intestine is increased, this characteristic play of colours will be seen on the addition of nitric acid. Blood in the stools may possess its natural appearance, in which case it has probably come from low down in the intestinal tract, or from hæmorrhoids, and is in consequence not intimately mixed into the substance of the fæces, but merely lies on the surface. If, however, the bleeding point lies higher up, then the blood becomes acted upon by the digestive fluids, and assumes a dark-brown or black appearance in the stools (melæna), with the substance of which it is intimately mixed. If in small quantity, the detection of blood in the fæces may require the tests described in connection with the Urinary system.

(*b.*) *Reaction.*—The reaction of the fæces is variable, but is usually alkaline, almost always so in typhoid fever. In the acute catarrhal enteritis of children the stools are usually acid.

(*c.*) *Form and Consistence.*—The fæces, which are normally consistent and formed, become much harder when constipation exists, their long delay in the intestine affording time for the more complete absorption of the watery constituents. The reverse condition naturally obtains where peristaltic action is increased, and the fæces are rapidly hurried through the intestinal canal. An increased secretion of the intestinal juices adds to this fluidity, and in practice we meet with all gradations of consistency, from an almost stony hardness to nearly perfect fluidity. The presence of a polypus in the rectum may impress a longitudinal groove upon the fæcal masses; and when there is narrowing of the bowel, particularly of the rectum, the fæces are usually thin, long, and narrow.

(*d.*) *The Odour* of normal fæces depends chiefly on the presence of indol and skatol. Where much decomposition has occurred it may be greatly increased.

(*e.*) *Abnormal Substances visible to the naked eye.*—Either on account of their own indigestibility, or from interference with digestion, various articles of food may appear unaltered in the fæces. An excess of starch indicates defective action of the salivary or the pancreatic ferment. Of importance is the detection of gall-stones. When their presence is suspected the fæcal

matters should be carefully washed on a fine sieve. Shreds of mucous membrane, polypi, and other tumours are occasionally met with, and, more frequently still, the various parasites that inhabit the intestinal tract. The round worms include the *Ascaris lumbricoides*, which resembles in shape a common earth-worm; the *Oxyuris vermicularis*, very small and thin, like a piece of cotton thread, about half-an-inch long; and the *Trichocephalus dispar*, which is readily recognised by its anterior end being thin and thread-like, while the posterior is much thicker. It measures from one to two inches in length. The three common varieties of tape worms are *Tænia solium*, *Tænia mediocanellata*, and *Bothriocephalus latus*. The last may be distinguished from the two first by the fact that the sexual openings occur in the middle of the segments, while in the others they are placed at the border. The *Tænia solium* differs from the *Tænia mediocanellata* in many particulars, the most obvious being its possession of from twenty-six to thirty hooklets on the head, which are absent in the *Tænia mediocanellata*. *Echinococcus* cysts are likewise sometimes found in the stools.

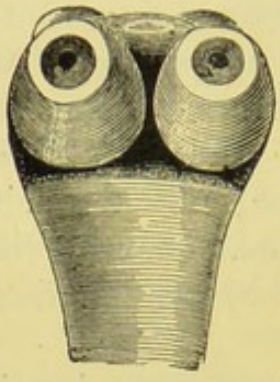


FIG. 5.—Head of *Tænia mediocanellata* × 10 diam. (Quain's Dict.)



FIG. 6.—Head of *Tænia solium*. × 10 diam. (Quain's Dict.)

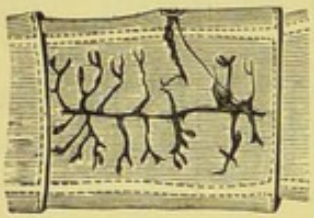


FIG. 7.—Proglottis of *Tænia solium*. × 1½ diam. (Quain's Dict.)

Abnormal quantities of mucus and bile may be seen; and fat in large amount, when not accounted for by diet, usually indicates affection of liver or pancreas. To the presence of blood allusion has already been made.

The evacuation of pure mucus from the bowel without any admixture of fæces points to catarrh of the rectum. When firm fæces are passed completely enveloped in mucus, we may con-

clude that the morbid process affects the lower part of the colon and the rectum. The admixture of mucus with the fæces in abnormal quantity is not always apparent to the naked eye. It often happens that when the fæculent matter is examined microscopically there are found scattered intimately through it small masses of mucus, which are whitish grey, hyaline, and transparent. This peculiar admixture of mucus indicates that the catarrhal affection is limited to the upper portion of the large intestine

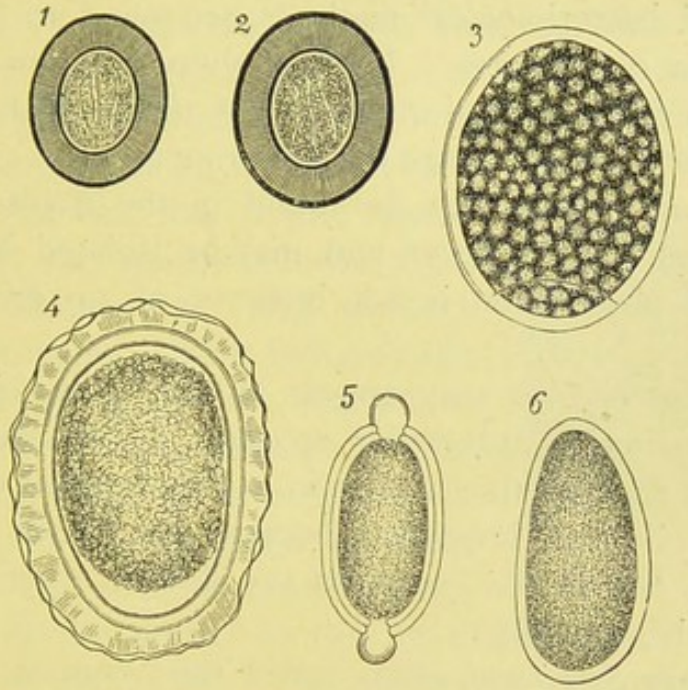



FIG. 8.—EGGS OF THE FOLLOWING FORMS :—

1. Tænia solium.
2. Tænia mediocanellata.
3. Bothriocephalus latus.
4. Ascaris lumbricoides.
5. Trichocephalus dispar.
6. Oxyuris vermicularis.

(and, possibly, the small intestine), while the rectum and descending colon are free from disease. When the stools contain small masses of mucus tinged yellow with bile pigment, we may conclude that the small intestine has become affected.

*Microscopic Examination of the Fæces.* — Fragments of food, including muscular fibre, connective tissue, fat cells and crystals, coagulated albumen, vegetable cells, &c., are readily to be detected in the stools. Besides these, we have to recognise certain elements which are derived from the tissues themselves, as,

for example, epithelial cells, mucus and pus corpuscles, blood corpuscles and crystals of various forms — triple-phosphate, cholesterin, Charcot's crystals, hæmic crystals, and balls of leucin. In the stools may also be found the eggs of the parasitic worms, the adult forms of which have just been described, the general appearance of the more important of which may be gathered from Fig. 8.

**Micro-organisms of the Fæces.**—The fæces, when examined by the usual bacteriological methods, are found to be crowded with bacteria of all sorts. Most of these are non-pathogenic, and have no direct clinical interest. Of those which are pathogenic the more important are the following:—

*The typhoid bacillus* may be found in the stools during the first two weeks of the fever, and may be isolated by plate cultures. This observation is not, however, of any great practical value.

*The tubercle bacillus* may appear in the fæces, either as the result of swallowing tubercular sputum, or because tubercular ulceration of the intestine has set in. This method of investigation is not clinically important, because the bacilli only appear in the stools late in the course of the disease, when a diagnosis has already been made.

*The Cholera Spirillum*, often called the "comma bacillus," is found in enormous numbers (sometimes so as to form a pure culture) in the rice-water stools of Asiatic Cholera. The importance of its recognition is very great, for only by means of its appearance and reactions can an accurate diagnosis be made, and this is especially the case at the commencement of epidemics. These organisms are about 2$\mu$ in length, by about 5$\mu$ in breadth, are curved to the shape of a comma, and most usually lie singly, though some adhere to one another so as to form shapes like the letter S. They are flagellated, and possess active motility. They stain readily with Löffler's methylene-blue solution, or, as Koch recommends, with Ziehl-Neelsen's carbol-fuchsin. To detect and diagnose the organism, film preparations should be made from the flakes floating in the stools, and stained as above-mentioned.

In many cases an almost pure culture may thus be obtained. In hanging-drop preparations the active movements of the organism may be observed. Plate cultures on gelatine and on agar, and stab cultures in gelatine, should now be made. On gelatine plates the colonies appear as circular discs with irregular edges, and, as they grow, the gelatine becomes liquified, forming little cups in which the colonies lie. In stab cultures the track of the needle becomes marked by a whitish line of growth, at the upper end of which the gelatine liquifies and leaves a cavity like an air-bubble. In peptone bouillon the organism grows very rapidly, and in the pellicle which forms on the surface it will be found in great numbers. The *Cholera-red reaction*, described by Bujwid and by Pöhl, may be obtained in this bouillon. It depends on the fact that in the course of its growth the spirillum forms indol from the albuminates, and reduces nitrates to nitrites. Now indol, in the presence of nitrite, gives a red colour on the addition of mineral acids. Consequently, if a few drops of hydrochloric acid or sulphuric acid (each of them quite pure and free from any trace of nitric acid) be added to this bouillon, the Cholera-red reaction will be produced. The reaction is important clinically, because, though many organisms produce indol, very few produce at the same time the nitrite, the presence of which is necessary for the reaction. For other bacteriological details, and, in particular, for the test by intra-peritoneal inoculation in rabbits, special books on the subject should be consulted.

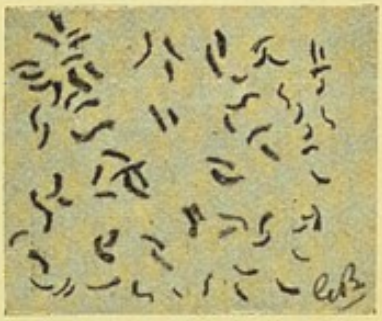


FIG. 9.—Cholera spirillum.

The Finkler-Prior spirillum, which has been found in cases of Cholera Nostras, closely resembles the spirillum of Asiatic Cholera, though rather larger and thicker. It can, however, be readily differentiated by means of cultures. These differences are well shewn in the case of stab-cultures in gelatine, where the organism grows more quickly and liquifies the gelatine much more rapidly than is the case with Koch's spirillum, causing a characteristic funnel-shaped tube to appear in the gelatine.

Another organism, of a class totally different to the preceding, is found in cases of endemic tropical dysentery. In this form of the disease, which is now called "amœbic dysentery," the cause of the affection is an amœba, and as this organism is present in the stools its recognition is of diagnostic value. The organism is rounded or more or less irregular in shape, and shews, during life, slow amœboid movements. It is nucleated, sometimes vacuolated, and often contains red blood corpuscles. As these amœbae rapidly break up and disappear from the stools, the following precautions should, according to Councilman and Lafleur, be taken in attempting to detect them. The stools should be received in a warm bed-pan, and kept at a temperature of 30° to 35° C., until an examination can be made, which should be as soon as possible, and in any case before the stools have become acid. A drop is to be placed on a slide, covered with a cover-glass, and examined, if possible, on a hot stage. A magnifying power of 400 diameters is sufficient.

## CHAPTER III.

# EXAMINATION OF THE ABDOMEN.

### INSPECTION.

The physical examination of the abdomen can only be made with advantage when the patient is in the recumbent posture. It is well to have the shoulders slightly elevated by means of a pillow, so as to relax the abdominal muscles, which can be still further effected by causing the knees to be raised. The examination is conducted by means of inspection, palpation, percussion, and auscultation, each of which will be considered in turn.

The abdominal surface has been arbitrarily divided into certain regions, the position of which is sufficiently indicated in the accompanying diagram.

The form of the abdomen varies greatly within physiological limits. A full dietary and a corpulent habit cause prominence in this region, whilst in old age and after prolonged inanition, the belly sinks in, and its bony walls become unduly prominent.

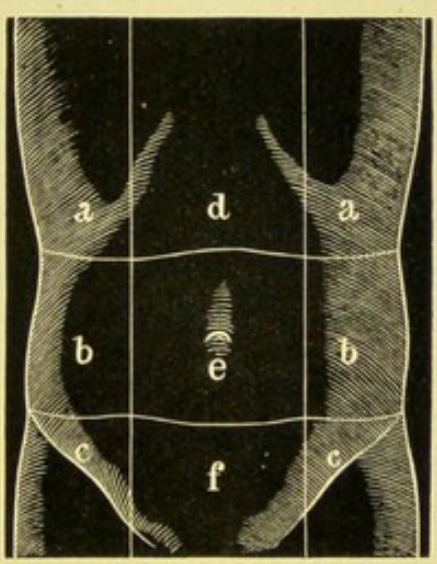



Fig. 10.—Regions of the Abdomen

(*a.*) Hypochondriac regions.
(*b.*) Lumbar regions.
(*c.*) Iliac regions.
(*d.*) Epigastric region.
(*e.*) Umblical region.
(*f.*) Hypogastric region.

The pathological changes which inspection indicates may be considered under three heads—(1) General prominence; (2) General retraction; (3) Local tumefaction.

*General prominence* of the abdomen is found—

1. In *ascites*, where an effusion of fluid has taken place into the peritoneal sac. The fluid gravitates to the most dependent position; and, consequently, when the patient lies on his back, the anterior part of the abdomen is flattened while the sides bulge, whereas, if the erect position be assumed, the prominence is greatest in the hypogastric region.

2. In *meteorism*, or accumulation of gas in the intestines. In this case a change in position does not affect the form of the abdomen, which is more spherical than in the former condition.

When the abdomen is greatly distended, from whatever cause, the diaphragm becomes raised, the ribs pressed outwards, and the position and character of the apex-beat of the heart altered. The abdominal walls become smooth and glistening, the recti muscles are pushed asunder, and in the interval between them the peristaltic motion of the intestines may occasionally be observed. The umbilicus rises, first of all, to a level with the adjoining skin, and subsequently protrudes beyond it. The pressure exerted on the inferior vena cava gives rise to the development of the collateral venous circulation as a delicate blue network over the abdominal parieties, whilst obstruction to the portal circulation occasions distension of the veins at the umbilicus—this varicose condition being the more observable by reason of the prominence of the navel.

Tumidity of the abdomen is an important sign in cases of obstruction of the bowels, especially if that obstruction is chronic, and its importance consists chiefly in the fact that, in many cases (as Wyllie has pointed out), the character of the tumidity indicates the site of the obstruction. When obstruction exists at the lower end of the large intestine,—at the sigmoid flexure or in the rectum,—the whole of the large intestine, and often the small intestine also, presents an appearance of tumidity, shews peristaltic movements, and, in the case of the large intestine specially, offers an elastic resistance to pressure.

In cases of obstruction at the lower end of the small intestine, on the other hand, there is complete absence of swelling and distension of the colon. In this case the tumidity, affecting the

small intestine, appears in the centre of the abdomen, especially between the umbilicus and the pubes. The abdomen then presents what Wyllie calls the "ladder-pattern," as in Fig. 11.

Where the obstruction is at the pylorus the stomach only is distended, and it may shew visible peristaltic movements, its contour standing out in relief during the rigid spasms into which it is thrown from time to time.

*Retraction* of the abdominal walls is met with in cases of inanition from whatever cause (particularly in œsophageal obstruction), and in all wasting diseases. It is also seen in various diseases affecting the nerve centres (as, for example, in tubercular meningitis), and is then attributable to contraction of the intestines, determined by the irritation at the base of the brain.

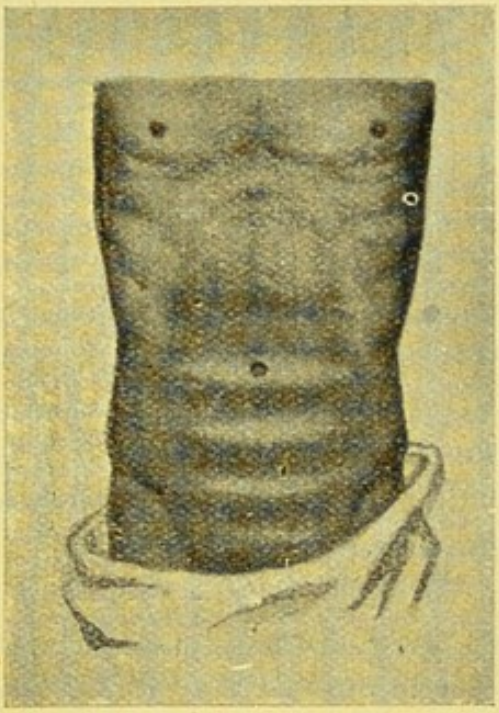


Fig. 11.—Abdomen, "laddr pattern" (after Wyllie).e

In such cases the bony walls become prominent; the vertebral column, with the pulsations of the aorta lying on its left side, may be seen, and the relaxed abdominal walls form pendulous folds.

*Local tumefaction* may occur in connection with various abdominal organs as follows:—

*Stomach.*—Dilatation of this viscus gives rise to an oval swelling occupying the epigastrium, and extending chiefly towards the left. The position of the greater curvature may be indicated by a diagonal furrow running from the right downwards, and to the left.

The outline of the stomach is best appreciated when it is artificially distended, according to the method originally recommended by Frerichs. The patient is made to swallow successively a solution of tartaric acid, and one of bicarbonate of soda, of each salt as much as may be carried on the point of

a table-knife. The result of the mixture of these solutions in the stomach is the development in that viscus of a large quantity of carbonic acid gas, and consequent distension. In a few seconds the position of the greater curvature, and the general outline of the stomach, can be made out with great distinctness. This method is but seldom resorted to in this country, and is not to be recommended.

Tumours of the stomach sometimes cause visible prominence of the abdominal wall. In some cases such tumours do not alter their position with the respiratory movements, and may thus be distinguished from tumours of the liver and spleen, which rise and fall with the respiration; but sometimes this is not so, the gastric tumour mass having contracted such adhesions to neighbouring parts that it moves synchronously with the diaphragm.

*Liver.*—In the healthy adult[1] the liver gives rise to no visible prominence of the abdominal walls. When enlargement takes place, it usually shows itself first under the margin of the ribs, in the right hypochondrium, unless the left lobe be chiefly affected, when the tumefaction appears in the epigastrium. The edge of the liver, or the tumour arising from that organ when visible, may be seen to rise and fall with the respiratory movements—an important diagnostic point.

*Splenic Tumours*, when of large size, cause visible tumefaction of the abdomen generally, particularly on the left side.

Tumours of the *kidneys and omentum* do not, as a rule, cause visible swelling.

Ovarian tumours are usually at first lateral in position, but subsequently they may develop to so great a size as to distend the whole abdomen.

**Abdominal movements** other than those of normal respiration may be seen—(*a*) respiratory, (*b*) pulsatory, (*c*) peristaltic.

[1] In children the liver is, normally, large in proportion to the size of other viscera, and occasions a considerable degree of fulness of the abdomen, extending from the lower border of the ribs on the right side to the level of the umbilicus.

The *respiratory movements* affect the position of all tumours which are attached to the liver or diaphragm. Splenic tumours are also similarly influenced, but to a less degree. Tumours of the stomach and pancreas, etc., are not so affected, unless adhesions have been formed.

*Pulsations* of various kinds are met with in the abdomen, but their nature will be more conveniently considered in connection with the circulatory system.

*Peristaltic motions* of the intestines may be observed in persons in whom the abdominal walls are abnormally thin (as in ascites, *vide antea*), or where such vermicular movements are usually energetic, as in cases of intestinal obstruction.

## CHAPTER IV.

## PALPATION OF THE ABDOMEN.

At no portion of the body is the skilful application of the hand of more essential service to diagnosis than over the abdomen. By carefully pressing with the hand (which should be warmed) at various points with a kind of gentle kneading motion, we obtain by the sense of touch, information regarding—1st, the condition of the abdominal wall; 2nd, the size, form, consistence, and mobility of certain of the abdominal organs, and whether any tumour be present within the cavity of the abdomen; and 3rd, if there be general tumefaction of the abdomen, whether such distension is the result of accumulation of gas in the intestines (tympanites), or is due to the presence of serous or inflammatory exudation; and, in the latter case, whether such exudation be in the peritoneal cavity or be inclosed in a cyst of some kind or another.

The position of the patient is of the greatest importance. He should lie on his back, with head and neck slightly raised, and with the knees flexed and drawn up towards the abdomen. In most cases it is well to engage the patient in conversation while palpating the abdomen, as otherwise the abdominal muscles are usually involuntarily contracted, and the glottis closed. The air in the lungs, retained there by the closure of the glottis, supplies the necessary resistance to this contraction of the abdominal muscles, and if this resistance be removed (as is best done by forcing the patient to open his glottis in speaking) the muscles have nothing to contract upon, and consequently become flaccid. The relaxation of these muscles may be still further aided by diverting the patient's attention. Should there

be necessity for it, the exhibition of chloroform will allow of a very perfect exploration of the abdominal cavity.

**Abdominal Walls.**—The temperature of the skin, the amount of subcutaneous fat, the presence or absence of œdematous or emphysematous swelling of the subcutaneous cellular tissue (see chap. i.) are readily recognised by the palpating hand, and require no special mention here. Localised swellings of the abdominal wall, due to the presence of tumours, of inflammation, or of abscess, may be mistaken for more serious affection of the abdominal organs themselves. The immobility of such swellings, their position being unaltered by the respiratory movements, or by a change in the posture of the patient, will generally suffice to distinguish them. In reality, the physician has seldom any difficulty in satisfying himself of the seat of the swelling, whether in the parieties or in the cavity of the abdomen. In difficult cases, such, for example, as when a deep-seated abscess over the liver simulates a hepatic abscess opening outwards, the history of the case, and the other signs and symptoms, suffice as a general rule to indicate the real seat of the abscess.

The abdominal muscles, and more especially the "recti," present, when contracted, certain inequalities in thickness which are occasionally mistaken by the inexperienced for abdominal tumours.

The various hernial protrusions which are found in the umbilical, femoral, and inguinal regions belong more especially to the domain of surgery.

**Peritoneal Cavity.**—Acute general peritonitis gives rise to great pain and tenderness on pressure over the whole surface of the abdomen. The patient then usually lies on the back, with knees drawn up, partly in order to relax the abdominal muscles, and partly to diminish the pressure of the bed-clothes. The abdominal muscles are usually rigidly contracted. In chronic peritonitis a characteristic doughy resistance is usually to be felt over the affected part, accompanied with some tender-

ness on pressure. Transudation of fluid into the peritoneal cavity (ascites) gives rise to a feeling of fluctuation. This is best appreciated by placing the hand on one side of the abdomen, and giving a smart tap on the surface at a point diametrically opposite. The impulse of the wave so formed can usually be clearly felt when it reaches the opposite wall. If, however, the amount of fluid be small, no such undulation will be observed in the ordinary position. The patient may then be placed on his elbows and knees, when the fluid will gravitate to the anterior part of the sac, and fluctuation can there be obtained.

Friction vibration can also occasionally be felt between two roughened peritoneal surfaces. It may be synchronous with the respiratory movements, and this most frequently if the visceral and parietal layers over the liver and spleen be the seat of the roughness (particularly in carcinoma of the liver). Friction vibration can also be induced in such cases by moving the peritoneal surfaces against one another, and pressure will at all times increase the strength of the friction.

**Liver.**—In the healthy adult, as a rule, only the left lobe of the liver can be felt by the palpating hand, giving rise to a slight feeling of resistance in the epigastric region. On very deep inspiration, however, the edge of the right lobe may sometimes be made to project so far beyond the costal margin as to offer appreciable resistance to the fingers. In children, the liver is of such size as to be readily examined by palpation.

Either as a result of enlargement or of lowered position (due, for example, to the downward pressure of a pleural effusion), the liver may come within reach of the palpating hand, and then we have to examine the condition of its surface, the consistence of the organ, its size, and general shape.

The *surface* of the liver may be smooth or rough. In amyloid and fatty degeneration, and in congestion, the surface of the swollen organ is smooth, a condition readily recognised by palpation. In the case of cirrhosis, the uneven granular surface gives rise to a characteristic feeling of roughness when the abdominal wall is made to glide backwards and forwards

over the surface of the liver. More marked irregularities of surface are found in carcinoma, the distinct nodules of which can be felt, and occasionally the umbilications which these nodules present.

*Tenderness* on pressure is met with in congestion and in all inflammatory affections of the liver, such as hepatic abscess, cirrhosis, catarrh of the bile ducts. In carcinoma it is often a very marked feature, although even in this affection it may be absent. There is usually no tenderness in the case of the waxy and the fatty liver.

The *Consistence* of the liver is somewhat increased in fatty degeneration, still more so in congestion, and to a very marked degree in waxy disease, when the lower edge may assume an almost knife-like sharpness. The presence of fluctuation will usually suffice to distinguish a hydatid tumour or an abscess from a solid growth.[1]

The *size* of the liver varies greatly. In some cases, as in acute yellow atrophy, the organ recedes so far into the concavity of the diaphragm as to be out of reach of palpation. In other instances (congestion, waxy degeneration, etc.), the lower edge may be found as low as the symphysis pubis. It must be carefully borne in mind that the position of the lower border is no safe guide to the size of the liver unless it be taken along with the position of the upper margin as ascertained by percussion.

*Abnormalities in shape.*—The practice of tight-lacing not only forces the liver downwards, but also frequently so compresses the hepatic substance as to give rise to a deep furrow marking off the lower portion of the right lobe. This furrow may often be detected by palpation. Still more obviously is the shape altered by the presence of a large tumour, cancerous or hydatid, growing from some particular part of the organ. It is important to remember that hepatic tumours rise and fall with the respiratory movements, which is not the case with growths in the

[1] When a hydatid tumour lying near the surface of the liver is percussed, a peculiar and very characteristic tremor (hydatid fremitus) may be felt over it, probably due to the reflection, from side to side of the sac, of the undulations into which the fluid has been thrown.

stomach, omentum, pancreas, colon, or kidney, unless they have become adherent to the liver or diaphragm.

Occasionally the gall-bladder may be felt as a small pear-shaped tumour projecting from beneath the lower edge of the liver. Pressure, by emptying it of bile, may cause it to disappear; and in rare cases the presence of gall-stones in the bladder may be ascertained by palpation.

**Spleen.**—In the normal condition the spleen cannot be felt, and this is due partly to its deep-seated position, and partly to the fact that the splenic tissue is too soft to offer resistance to the palpating fingers. When, however, it becomes so enlarged as to reach the extremity of the eleventh rib, or to pass beyond it, then the spleen can be readily recognised. Increase in size of the spleen takes place in numerous diseases, such as leucocythæmia, amyloid disease, recent syphilis, intermittent fever, typhus, enteric, and scarlet fevers, etc.; and in addition, all diseases which produce obstruction to the portal circulation, directly or indirectly (such as cirrhosis of the liver or heart disease), cause splenic congestion, and consequently, enlargement of that organ.

When the spleen is but slightly enlarged, it can be best felt by tilting it upwards from the lumbar region on to the palpating hand. A feeling of increased resistance may thus be appreciated, although the limits of the organ may not be felt with any distinctness.

As the organ increases in size it projects from beneath the margins of the ribs towards the umbilicus, and rises and falls with the respiratory movements. The enlargement is proportionately the same in all diameters, and so the spleen retains its original shape. The splenic notch is readily felt, and is important as a certain indication that the tumour with which we have to deal is splenic. In leucocythæmia, the spleen may attain an enormous size, and fill up the greater part of the abdominal cavity. Except in very rare cases (hydatid disease and carcinoma of the spleen) the surface of the swollen organ is smooth, and there is seldom any tenderness on pressure.

The consistency of the spleen is greatly increased in amyloid disease and in leucocythæmia. In congestive enlargement it is not so resistant, and in acute diseases the tumefied gland is of a very soft consistence. During the exacerbations of intermittent fever the spleen undergoes perceptible enlargement; while, in cases of splenic congestion from portal obstruction, loss of blood from the stomach or intestines causes diminution in its size.

**Pancreas.**—Tumours of the head of the *pancreas* are rarely met with, and are difficult of diagnosis, owing to the way in which the gland is covered by the coils of the intestines, and to some extent by the lower edge of the liver. Hardness, in such cases, can usually be felt to the right of the middle line above the level of the umbilicus. It is deeply seated, not freely movable, and unaffected by the respiratory movements. The disease (which is almost invariably carcinoma) is seldom limited to the pancreas, but attacks the retro-peritoneal lymphatic glands, and other neighbouring parts.

**Stomach and Intestines.**—Tumours of the stomach (usually carcinomatous) are most common at the pylorus. They are then readily felt as irregular nodulated masses in the umbilical region, tender to pressure, freely movable for the most part, and but little affected in position by the rise and fall of the diaphragm.[1] When the greater curvature is the part affected, the tumour mass is found somewhat lower down and to the left. Tumours of the lesser curvature and cardiac end of the stomach are rarely felt during life, as they lie deeply in the concavity of the diaphragm.

When the stomach is dilated palpation usually gives rise to well-marked gurgling or splashing sounds, the *timbre* of which is characteristic.

Pressure over the stomach occasions pain in many diseased conditions of that viscus. It is most circumscribed in cases of gastric ulcer, and is often of great severity.

In the intestines the retention of fæces, chiefly in the large

[1] This last point is often deceptive, for if the tumour have contracted adhesions, it may move with respiration, as has already been said.

intestine, may give rise to localised swelling at various points. These nodular masses are of a doughy consistency, and to a large extent disappear after purgation. Catarrhal and inflammatory conditions of the colon are apt to give rise to inflammation in the neighbouring tissues, resulting in a swelling which is usually ill-defined, doughy, hard, and very tender on pressure. Such inflammatory swelling is met with in the right iliac region in cases of appendicitis. Cancerous masses may occasionally be felt at various parts of the colon; the cæcum and sigmoid flexure being most commonly the seat of the disease. Peristaltic movements of the intestines are occasionally to be felt when the abdominal walls are thin or the movements very energetic, as in stenosis of the bowel (see pages 56 and 59).

An important diagnostic mark in appendicitis is great local tenderness on pressure at what is called "M'Burney's point," which lies at the intersection of a line drawn from the umbilicus to the anterior superior spine, with a second line corresponding to the outer edge of the right rectus muscle.

**Omentum.**—Tumours of the omentum are rare. They are of very various nature: cancerous, tubercular, hydatid, &c., and when developed are readily felt through the abdominal wall. When affected with carcinomatous disease the omentum becomes thickened and retracted, and its lower hardened edge may occasionally be felt crossing the abdominal cavity.

The **Mesenteric Glands** are frequently the seat of tumours. They may be simply enlarged, along with other similar glands throughout the body, or they may be affected with cancerous, tubercular or other deposits. They are smooth, hard, movable tumours of regular form. Occasionally they become fused together, along with other neighbouring structures (loops of small intestines, retro-peritoneal glands, &c.), into masses of considerable size, which, overlying the aorta, may have imparted to them a pulsatile movement.

The **Kidneys** are not, in their normal condition, within the range of palpation; but when they leave their position or

when they increase greatly in size, they may be felt. A floating kidney—that is, one the attachments of which are so loose as to allow of its moving more or less freely through the abdominal cavity—is recognised by its possessing the size and the characteristic renal shape, as well as by its great mobility, and by the presence of the pulsating renal artery, which may sometimes be felt entering at the hilus. The diagnosis is rendered more certain by the aid of percussion, as will be hereafter noticed.

Enlargement of the kidney occurs in hydronephrosis, echinococcus, carcinoma, &c., and the tumour is smooth or nodular according to its nature. It is recognised by its position and relations, its immobility, and its cylindrical shape from above downwards. Inflammatory thickening round the kidney and perinephritic abscess may also be recognised by palpation.

The *urinary bladder*, when distended, forms a pyriform tumour above the pubes, and may reach the level of the umbilicus, or even, in extreme cases, to a still higher point.

*Ovarian tumours* are in most cases cystic, and may be so large as to distend the whole abdomen. When small in size they usually lie on one side only, and gradually cross the middle line to assume an apparently central position. Fluctuation is generally easily made out. The characteristics of such growths will be considered under the head of percussion.

*Uterine tumours*, physiological as well as pathological, may be felt above the pubes. Their consideration belongs to the domain of the gynecologist and obstetrician.

*Aneurism of the abdominal aorta* may affect that vessel in any part of its course. If it lie very deep in the concavity of the diaphragm, it may not be capable of being felt with any distinctness, but, where it can be reached, a pulsating tumour is readily recognised. The pulsation must be distinguished from that produced by a tumour lying on the vessel, which is to be done by noting its true expansile character when compressed laterally between the fingers. Aneurisms of the main branches of the abdominal aorta also occasionally occur.

## CHAPTER V.

## PERCUSSION OF THE ABDOMEN.

THE theory of percussion will be considered hereafter, and need not detain us at this point. Its use in connection with the abdomen is to define the outline of organs which it is not possible to reach by means of palpation. As a rule it is best to use the forefinger of the left hand as a pleximeter, laying it upon the surface of the abdomen, and eliciting a note by striking with one or more fingers of the right hand. The change of note as one passes off such a solid organ as the liver on to air-containing viscera is usually sufficiently obvious.

**Condition of the Peritoneal Sac.**—When transudation of fluid takes place into the peritoneal cavity, it does not, in the first instance, affect the percussion note over the surface of the abdomen, since the small quantity of serum which at first collects, gravitates towards the lowest portion of the sac; and whether this point lie in the pelvis or towards the spinal column (determined by the position of the patient, erect or supine), the collection is too far removed from the surfaces ordinarily subjected to percussion to allow of its influencing the note obtained.

As the quantity increases it gradually makes its presence manifest, causing a dull note to be heard on percussion over the lower parts of the peritoneal cavity. With further increase in the quantity of fluid, the dulness extends its area, until in extreme cases, where the sac is greatly distended with transudation and the bowels compressed, the note over the whole surface of the abdomen becomes absolutely dull. In cases of medium severity, when the patient lies on his back the fluid

gravitates towards the lumbar regions, and the intestines float on its surface, so that the percussion note over the anterior surface of the abdomen is clear and tympanitic, expressing the presence of large air cavities beneath (bowels), while on either side, as we pass towards the lateral and posterior regions, there is dulness corresponding to the position of the ascitic fluid. If the patient lie on his left side, the right side will be the point towards which the air-containing intestines will float, whereas the fluid in the peritoneal cavity will gravitate towards the left. The change in the percussion note thus caused by alteration in the position of the patient is an indication of the presence of free fluid in the peritoneum, and is the more important seeing that it does not occur in the case of pure ovarian dropsy.

Ascites may be the result of increase in the blood pressure within the portal vein, due, for example, to cirrhosis of the liver, or it may be merely a part of the general dropsical condition caused by cardiac or renal disease. It may also arise from local conditions in the peritoneum, as is seen in tubercular peritonitis.

The differential diagnosis of ascites is often very difficult. It is not hard to distinguish it from meteorism, in which there is neither lateral dulness altering with position, nor the undulatory impulse mentioned on page 62, or from an over-distended bladder, in which the dulness is centrally placed over the pubes. The pregnant uterus also is readily distinguished from ascites by the position of the dulness, and, above all, by the auscultation of the fœtal heart. It is often, however, extremely difficult to distinguish ascites from ovarian cystic tumour. The main points of difference are given in the accompanying table.

| Ascites. | Ovarian Cyst. |
|---|---|
| I. *History.*<br>No history of lateral development. | Tumour develops from one iliac fossa. |
| II. *Inspection.*<br>When patient lies on the back there is bulging at the flanks. If the ascites is considerable, the umbilicus is pressed outwards. | The greatest swelling is anterior, not in the flanks. Sometimes one side of the abdomen is more prominent than the other. |
| III. *Percussion.*<br>On percussion there is dulness in the flanks, and a clear note over the centre of the abdomen.<br>Changes of position alter the line of dulness in the manner already described. | The dulness is central, the intestines giving a clear note at the sides.<br>Change of position does not alter the line of dulness. |
| IV. *Aspiration.*[1]<br>Ascitic fluid presents the following characters :—<br>1. Specific gravity 1·010 to 1·015.<br>2. Light straw colour.<br>3. Coagulates spontaneously when exposed to the air.<br>4. Does not contain paralbumen. | Ovarian fluid has the following characteristics :—<br>1. Specific gravity 1·018 to 1·026.<br>2. Amber coloured ; often syrupy.<br>3. Seldom or never coagulates spontaneously.<br>4. Contains paralbumen. |

[1] The microscopic differences between the two fluids cannot be said to be as yet clearly determined.

**Percussion of the Liver.**—A considerable portion of the anterior and upper surface of the liver lies in contact with the anterior wall of the abdomen, and consequently over this area the percussion note is more or less absolutely dull, expressing the presence of a solid organ underneath. This area is spoken of as the *absolute hepatic dulness.*

Above this, the liver recedes from the chest-wall and becomes separated therefrom by a layer of lung of gradually increasing depth. In percussing from above downwards in the right parasternal line, the level at which the percussion note indicates that the subjacent air-space is being encroached upon and rendered shallower, corresponds (in the normal condition) to the highest point to which the liver reaches under the diaphragm, and at this level the deep or *relative hepatic dulness* commences.

The tympanitic resonance of the neighbouring abdominal organs, which contain air, enables us to mark out with considerable ease the lower border of hepatic dulness. The thinness of the lower edge of the liver necessitates that percussion should be made very lightly in order that we may avoid, as much as possible, the transmissions of the vibrations to parts in the vicinity. It is best practised, in this particular instance, by tapping very gently with the forefinger on an ivory pleximeter.

The *lower border of the liver* begins at the left, close to the apex of the heart, and passes diagonally downwards and towards the right, crossing the middle line at a point mid-way between the base of the ensiform cartilage and the umbilicus, and joining the margin of the ribs at an acute angle in the mammary line. From this point backwards to the axillary line the lower border corresponds pretty closely with the margin of the ribs. In some cases careful percussion may detect the presence of the gall-bladder, as a small rounded tumour projecting downwards from the edge of the liver.

*The upper border of absolute hepatic dulness* corresponds to the lower edge of the right lung, except in regard to the left lobe, where it passes imperceptibly into the cardiac dulness. At the right border of the sternum it lies at the level of the sixth rib;

in the mammillary line it corresponds to the upper border of the seventh rib, and in the axillary and scapular lines it reaches respectively the eighth and ninth ribs.

The upper limit of the deep or *relative hepatic dulness* lies about three inches above the absolute dulness. In the mammil-

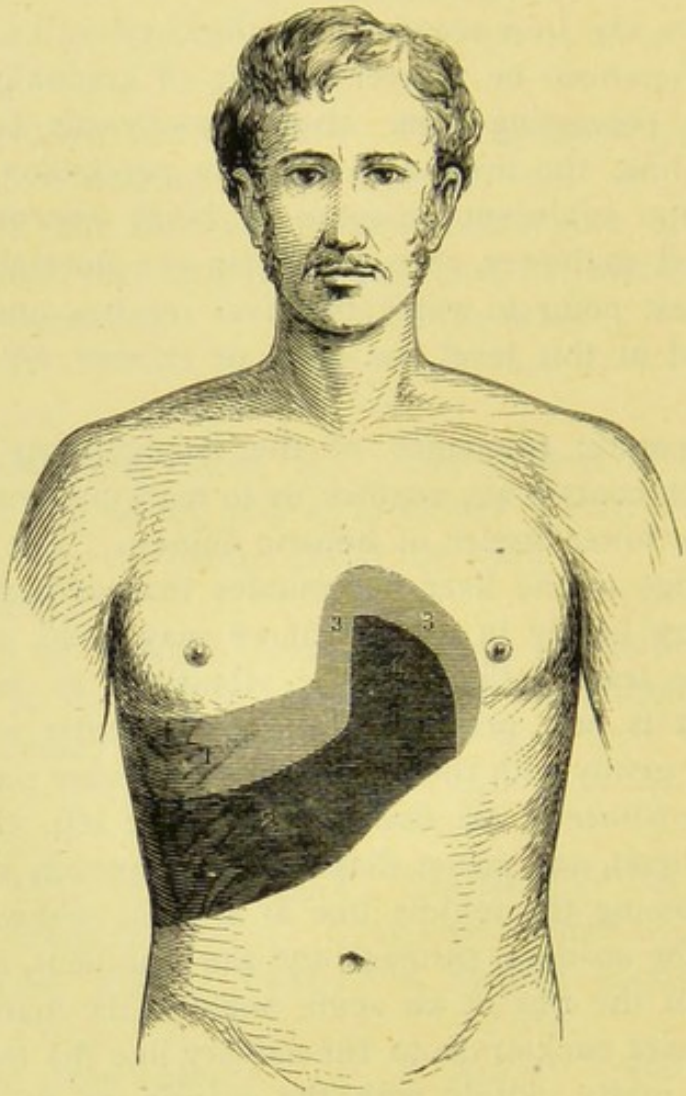



FIG. 12.—Cardiac and Hepatic Dulness.

1. Relative hepatic dulness.
2. Absolute hepatic dulness.
3. Relative cardiac dulness.
4. Absolute cardiac dulness.

lary line the percussion note can usually be noticed to become dull about the fourth intercostal space, or fifth rib.

The position of the hepatic dulness, relative and absolute, is roughly indicated in the accompanying diagram.

The movements of respiration change the position of the liver. Deep inspiration depresses the lower edge considerably, while full expiration permits of a corresponding elevation. But in addition to such alterations in the level at which the lower border of the liver stands, the respiratory movement affects the position of the upper border of the absolute dulness to a much greater extent. This latter alteration in hepatic dulness does not express so much a change in the position of the liver, as the rise and fall of the lower border of the right lung, and the extent of the complementary pleural space occupied by pulmonic tissue.

Changes in the position of the body also cause slight differences in the position of the liver, the organ gravitating towards the most dependent side.

The hepatic dulness may be greatly altered without any actual change in the size of the liver. Thus the colon, distended by air or a coil of the small intestine, may be forced upwards between the surface of the liver and the abdominal parieties, thus preventing the true lower border from being found by percussion, and leading to an apparent diminution in the size of the liver. In pulmonary emphysema the right lung extends lower down than normally, and the upper border of absolute liver dulness is thus depressed. Again, the liver may be elevated abnormally within the concavity of the diaphragm, by reason of increased intra-abdominal pressure ; a greater proportion of the organ will be thus overlapped by lung, and the absolute dulness diminished. If the two last conditions co-exist, all trace of absolute hepatic dulness may fail, and this may occur when the liver is of normal size.

From all this it follows that the extent of the absolute hepatic dulness is no safe guide to the size of this organ, and in all cases it is best to measure from the upper level of the deep or relative dulness to the lower border of the liver. Unfortunately, in some cases such measurements cannot be accurately made (right pleural effusion, pronounced ascites, &c.), but when percussion can determine the deep dulness, the position of the lower border may in such cases usually be ascertained by palpation.

*Displacement of the liver* takes place more frequently down-

wards than upwards. The causes of displacement *downwards* are—(1) Emphysema of the lungs, whereby both hepatic lobes are equally depressed; (2) pleuritic effusion on the right side, which causes depression of the right lobe of the liver, with perhaps slight elevation of the left lobe; (3) right pneumothorax, producing the same conditions. To these may be added, as much rarer causes of downward displacement, various tumours of the mediastinum and of the diaphragm, and encapsuled peritoneal effusions between the diaphragm and the upper surface of the liver. The left lobe of the liver may be slightly depressed by large pericardial effusions, and by effusion of liquid or gas into the left pleural cavity.

*Displacement upwards* occurs less frequently, and to a less extent, than depression. It is occasioned by any condition which produces an increased pressure in the abdominal cavity—ascites, meteorism, ovarian cysts, &c.—and possibly also by cirrhosis of the right lung.

*Hepatic enlargement.*—The increase in size of the liver may be very marked. It may in rare cases rise as high as the second rib (Gerhardt), while its lower edge may reach to a point close to the symphysis pubis. The chief causes of great enlargement of this organ are hydatid tumours, carcinoma, and waxy disease. It is found to a less degree in hepatic congestion (as in mitral disease), occlusion of the bile ducts, fatty degeneration, and in certain cases of cirrhosis. The alteration in the shape of the liver, caused by the practice of "tight-lacing," may simulate actual enlargement.

*Diminution in the size of the liver* occurs in the later stage of cirrhosis, and in acute yellow atrophy of the liver. The organ, as it becomes smaller, leaves the surface of the abdomen and retreats into the concavity of the diaphragm. Its place is occupied by small and large intestine, and, in consequence, all trace of hepatic dulness may disappear. This extreme diminution is met with in the latter disease, whilst in cirrhosis, when the liver is much contracted, the percussion of the lower border is prevented by the almost invariable presence of ascites at that advanced stage of the disease.

**Percussion of the Spleen.**—Placed obliquely in the cavity of the abdomen, the spleen lies with its upper and posterior extremity opposite the tenth dorsal vertebra, in the concavity of the diaphragm, and somewhat overlapped by the left lung. From this point the gland passes downwards and forwards to terminate slightly behind the extremity of the eleventh rib. Its upper and anterior border runs parallel with the ninth rib, while the posterior and lower border follows pretty closely the eleventh rib.

The percussion of the spleen is peculiarly important, because when the organ is but slightly enlarged, it is practically inaccessible to palpation. In consequence of the nearness of large air cavities in the stomach and colon, it is necessary to percuss very lightly in order to obtain the splenic dulness, and not allow the note to become obscured by reason of the tympanitic resonance of these organs.

The spleen is of variable size, and, as age advances, it atrophies so that a small area of dulness may be met with under physiological conditions.

Respiratory movements affect the position of the splenic dulness, deep inspiration depressing it and diminishing its size. When the patient lies on the right side the spleen tends to gravitate towards that direction, and the splenic dulness diminishes or disappears. When the spleen is not greatly enlarged, it is best to percuss it while the patient is in the erect position, with the left arm removed from the side.

The condition of the stomach has an important influence on the percussion of the spleen. If the fundus is greatly distended with food, it occasions a dull note on percussion in the neighbourhood of the spleen, in such a way as altogether to prevent the differentiation of the splenic dulness. On the other hand, if the stomach be much distended with gas, it becomes difficult to determine, with any exactness, the limits of the splenic dulness, because of the tympanitic resonance of the gastric cavity which even a very light stroke can hardly fail to produce.

Conditions similar to those which cause upward displacement of the liver (p. 74) force the spleen in the same direction under

the lower margin of the left lung; and, in this case, as well as when the spleen is overlapped by emphysematous pulmonary tissue, all traces of splenic dulness may disappear.

The presence of ascitic fluid round the spleen will prevent its limits from being determined by percussion, and meteoric distension of the intestine with gas will cause a diminution in the size of the splenic dulness.

In pleuritic effusion and pneumothorax on the left side, as well as in pulmonary emphysema, the spleen is depressed, but in none of these conditions is it possible to define its limits by percussion.

*Enlargement of the spleen* is readily made out by means of percussion; but this method does not give any clue to the cause of the tumefaction, as in such cases the gland enlarges uniformly in all directions. When it passes beyond the borders of the ribs, it is best recognised by means of palpation. The various forms of splenic tumour have been already referred to (page 64), and need not now be recapitulated.

**Percussion of the Kidneys.**—It is only in the rarest of cases that such a method of examination need be resorted to, to aid in forming a diagnosis, and this because (1) the most frequently occurring renal diseases are not accompanied by so great an amount of alteration in the dimensions of these organs as to be appreciable by percussion; and (2) those cases of renal tumours where the tumour mass might be thus recognised are capable of far earlier and much more thorough investigation by palpation. Renal percussion may in fact be regarded as of little importance, except, perhaps, in the single instance of the diagnosis of floating kidney, where the absence of the normal area of renal dulness on one side might confirm the opinion. But as undoubtedly in many cases the percussion of the kidneys can be carried out, the subject must not be entirely ignored here.

The percussion stroke must be very firm, and is best given by means of a hammer and ivory pleximeter.[1]

[1] In persons in whom there is great development of the subcutaneous adipose tissue, the renal dulness cannot be percussed out.

The upper border of the renal dulness cannot be defined, as it passes imperceptibly into that of the liver or spleen, and the internal border lying next the spinal column is also incapable of definition. It is then chiefly the outer border, lying in the lumbar region, parallel to, and at about three finger-breadths distance from the spinal column, that can be marked out, while in some few cases the position of the lower border, close to the crest of the ilium, can also be ascertained.

The patient must be laid prone, the anterior surface of the abdomen being supported on cushions. The lower edges of the hepatic and splenic dulness in the scapular line on either side must first be marked out. Immediately below these levels the tympanitic note of the colon or of the stomach can be heard. If now we percuss inwards on either side towards the vertebral column, the renal dulness will be reached at the distance already indicated. The length of the renal dulness is usually from three to four inches, and occasionally, as I have already said, the lower border may also be defined by percussion.

[The *urinary bladder* does not enter the abdomen unless distended, and then it can readily be percussed out as a pear-shaped tumour lying in the middle line, and giving a dull note.]

**Percussion of the Stomach.**—When the stomach is filled with food, it is impossible to define its boundaries in any way by means of percussion; but when the cavity of the viscus is moderately distended with air, it gives on percussion a tympanitic note of so low a pitch, and of so long duration, that it is readily distinguishable from the tympanitic notes obtained from the neighbouring hollow viscera, and this by reason of the greater size of the air cavity. Should, however, the distension of the stomach with gas increase beyond a certain point, the walls are also thrown into vibration, and a metallic ring results, which renders the definition of the gastric outlines more difficult.[1]

[1] I have already alluded (page 57) to the method of artificially distending the stomach with gas for purposes of diagnosis.

The position of the stomach is such that its border can be satisfactorily defined only in one direction—*i.e.* in the line of the greater curvature; but the ascertaining of the position of this border is sufficient to enable us to say whether the viscus be enlarged or no.

It is best, first of all, to mark out the borders of the various other organs which surround the stomach, namely, the liver and spleen, as well as the position of the diaphragm. It is then found that the deep-pitched note of the stomach extends from the middle line to the left hypochondrium, neither passing to the right of the middle line nor below the level of the umbilicus. If either of those limits be overstepped at any point, the stomach is enlarged.

Dilatation of the stomach may sometimes be so great that the line of the greater curvature may reach almost to the pubes.

Over the *intestines* the percussion note is normally tympanitic, but is higher in pitch than over the stomach.

When the bowels become distended with liquid or solid contents, this note ceases to be heard.

Great distension with gas allows the walls of the intestines to pass also into vibration, thereby producing a metallic note.[1]

**Auscultation of the Abdominal Organs.**—Auscultation of the abdominal organs is rarely of value as an aid to diagnosis, if we except the auscultation of the uterus in pregnancy, which does not concern us here, and aneurisms of the abdominal aorta and its branches, which will be better considered in connection with the circulatory system.

[1] The difference in pitch of the percussion notes of the stomach and of the intestines may be more distinctly recognised by listening with a stethoscope while percussion is being made. Dr R. A. Fleming has recently called attention to the value of this method.

## CHAPTER VI.

# THE EXAMINATION OF THE BLOOD.

The examination of the blood has, of late years, attained great importance as an aid to diagnosis and a guide in the subsequent treatment of many forms of disease. So far as ordinary clinical investigations are concerned the quantity of blood required is very small, and can readily be obtained from a needle-prick. The skin of the patients' finger or the lobe of the ear should first be carefully cleansed with water and with ether, and the prick is best made by means of such a spear-shaped needle as is supplied with Gowers' Hæmocytometer. The blood should be allowed to flow of itself, the tissues not being compressed in any way.

**Specific Gravity.**—This may be tested by means of the method of Roy. He prepares first a series of solutions of glycerine in water of varying strengths, so arranged that they represent a series of specific gravities ranging from 1030 to 1068. The needle of a hypodermic syringe, its point having been rounded off, is used to draw up the drop of blood which has collected on the finger, and from it a very minute drop is allowed to fall into each one of a set of test-tubes containing these solutions of known specific gravity. Beginning with those of low gravity it will be found that the drop of blood sinks, but when that solution is reached which corresponds in specific gravity with the blood, the drop will remain suspended. Hammerschlag's modification of this method is still simpler. A mixture of chloroform and benzol is made in such proportions as to have a specific gravity of about 1059. A drop of blood is allowed to fall into this. If the drop sinks, more chloroform

is to be added; if it rises, more benzol, until the drop remains suspended, neither floating upwards nor sinking downwards, thus shewing that the specific gravity of the liquid is just that of the blood. By means of an ordinary urinometer this specific gravity may then be readily ascertained.

The normal specific gravity appears to vary from 1045 to 1065. In cachectic and anæmic conditions it is much reduced.

### Investigation of Blood-Pigment.

The investigation of the blood-pigment is of great clinical importance. It may be carried out both quantitatively and qualitatively. The quantitative estimation of hæmoglobin is the more important, and should be performed in every serious case, almost as a matter of routine. The qualitative becomes of consequence in diagnosis in special cases.

**Estimation of Hæmoglobin.**—Various instruments have been devised for this purpose. That of Dr Gowers consists of two glass tubes of the same diameter, one of which contains a standard colour-solution[1] (glycerine carefully tinted by means of carmine and picrocarminate of ammonia), while the other, in which the blood to be tested is to be diluted, is graduated so that 100 degrees = two cubic centimetres. There is also a capillary pipette graduated to hold twenty cubic millimetres, a bottle with a pipette-stopper to contain distilled water, and a guarded needle to prick the finger.

The method of using this instrument is as follows. The two tubes (C and D, fig. 13) having been placed upright in the small wooden stand (E) supplied for the purpose, a few drops of distilled water are placed in the bottom of the graduated tube. The blood having been obtained from the finger in the manner already described, twenty cubic millimetres of the blood are measured off by means of the pipette (B), and injected into the distilled water in the graduated tube, which must then be

[1] The tint of this standard solution corresponds exactly to that of a dilution of twenty cubic millimetres of blood with 1980 cubic millimetres of distilled water—*i.e.* a dilution of one in a hundred.

quickly shaken to ensure thorough mixture. More distilled water is now added, drop by drop, until the tint of the diluted blood is the same as that of the standard. The degree of dilution, as indicated by the graduation, expresses the amount of hæmoglobin as compared with that of the standard, and as this is a dilution of one hundred, the degrees of dilution required to obtain the same tint, represent the percentage proportion of the hæmoglobin to that of normal blood.

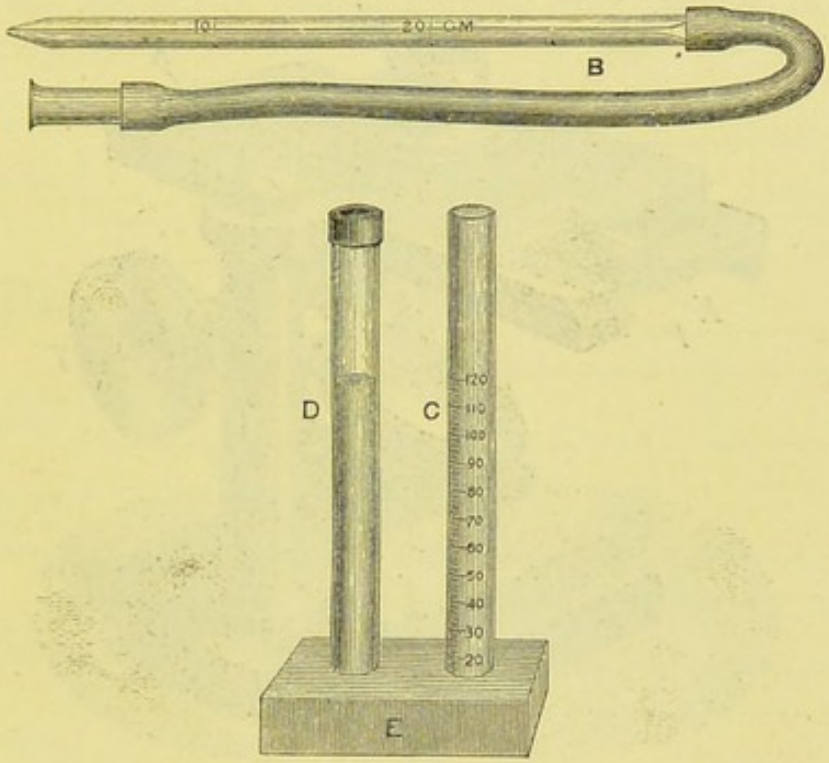



FIG. 13.—Gowers's Apparatus for estimating Hæmoglobin.

Of late the author has employed von Fleischl's Hæmometer (see Fig. 14) in hæmoglobin estimation, with considerable satisfaction. In using this instrument, one side of the metallic cell *a* is filled about one-quarter full of water. A capillary tube supplied with the instrument is then filled with blood from the finger or ear of the patient, and thoroughly and quickly washed out in the water of the compartment *a*. After the blood has been thoroughly mixed with the water, more water is added by means of a pipette to compartment *a*, until it is completely filled. Com-

partment *a'* is filled with water at the same time, and both are covered with a flat disc of glass. Under the compartment *a'*, which contains pure water, lies a wedge of coloured glass, which can be moved to right or to left by means of the screw T, so that a thicker or thinner portion of the wedge may lie below the water. It is thus possible to match the colour of compartment *a'*, containing water, with that of compartment *a*, which contains water and blood. The number on a graduated scale attached

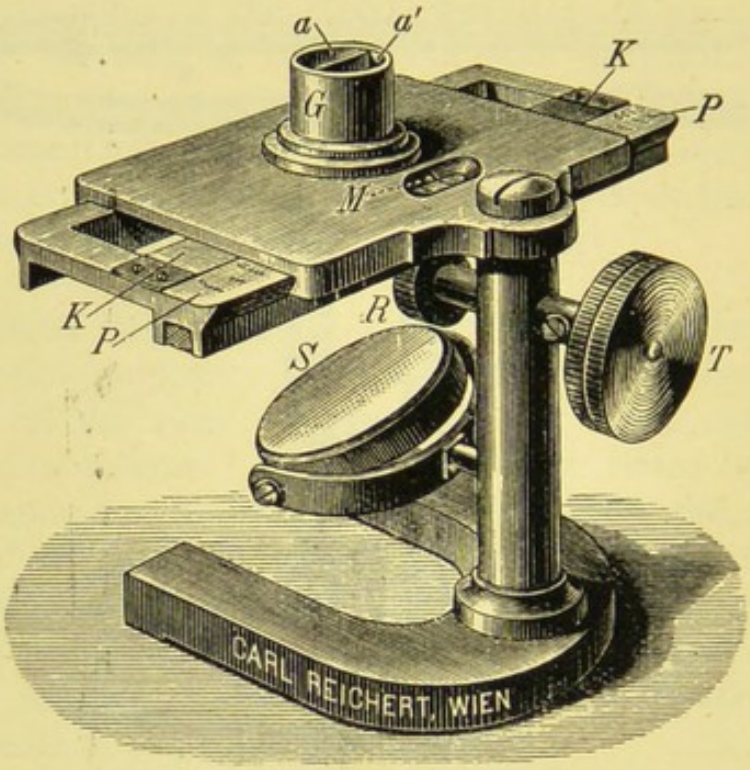



FIG. 14 —Von. Fleischl's Hæmometer.

to the wedge gives the percentage of hæmoglobin in the blood. The observation must be made by gas or lamp-light.

In a recent number of the *Deutsche med. Wochenschrift* (No. 10, 1897), Jolles describes a new instrument (the Ferrometer), for the quantitative estimation of the iron of the blood, for clinical purposes. The author has not, as yet, had an opportunity of testing the capabilities of this instrument.

If the corpuscular richness of the blood is ascertained by means of the hæmocytometer, we are able to compare this with

the amount of hæmoglobin in a very instructive manner. Thus, a fraction, of which the numerator is the percentage of hæmoglobin, and the denominator the percentage of corpuscles, will express the average value of each corpuscle.

Diminution of the quantity of hæmoglobin in the blood, a condition which is sometimes called Oligochromæmia, is met with in the course of all severe wasting diseases, but is most noteworthy in chlorosis, and leucocythæmia, where the percentage may fall very low, to 20% or even lower. In estimating the amount of benefit derived from the use of iron in

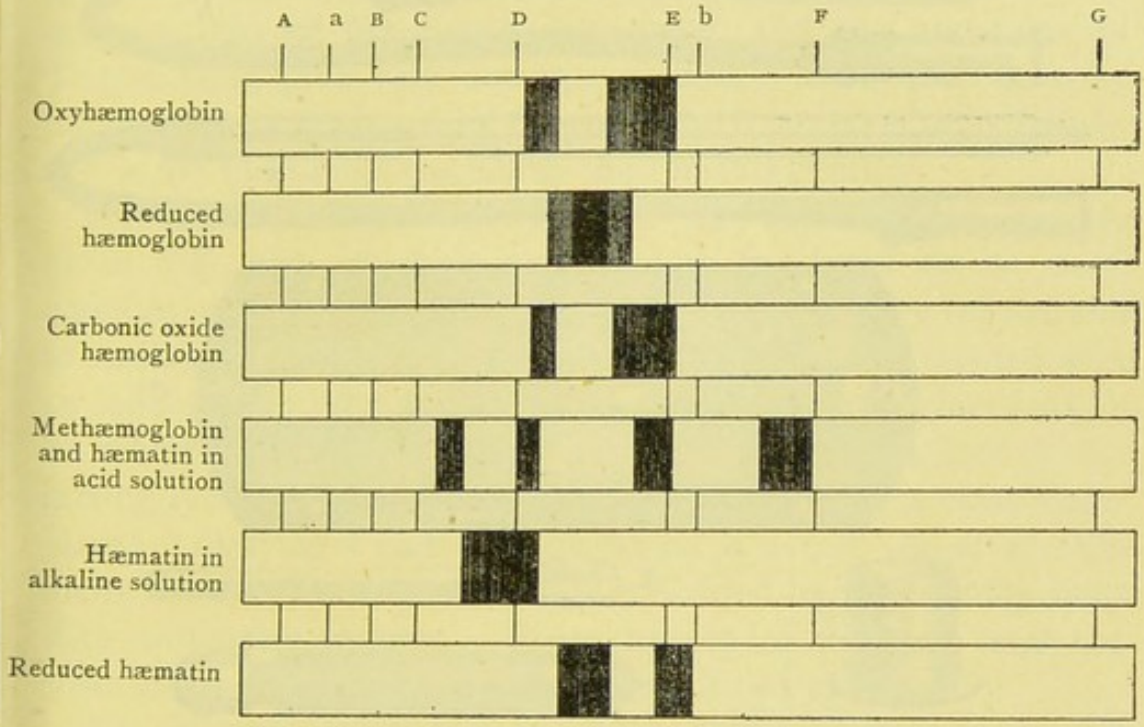



FIG. 15.—Absorption spectra of blood pigment.

any particular case, the hæmoglobinometer is of the utmost service.

**Qualitative Changes in Blood-Pigment.**—These are chiefly investigated by means of the Spectroscope, modifications of which instrument, small in size and moderate in price, are constructed specially for clinical use. The specimen of blood to be investigated, more or less diluted, is placed in a vessel with parallel walls, and held in front of the slit of the spectroscope.

If the blood is normal the spectrum of oxyhæmoglobin, two absorption bands between D and E, will be seen. Under the action of a reducing agent, say ammonium sulphide, the spectrum becomes that of reduced hæmoglobin, one broad band between D and E. On the addition of acids or alkalis, hæmatin is formed, which shows a different spectrum according as it is in an acid or in an alkaline solution. These various spectra are shown in Fig. 15.

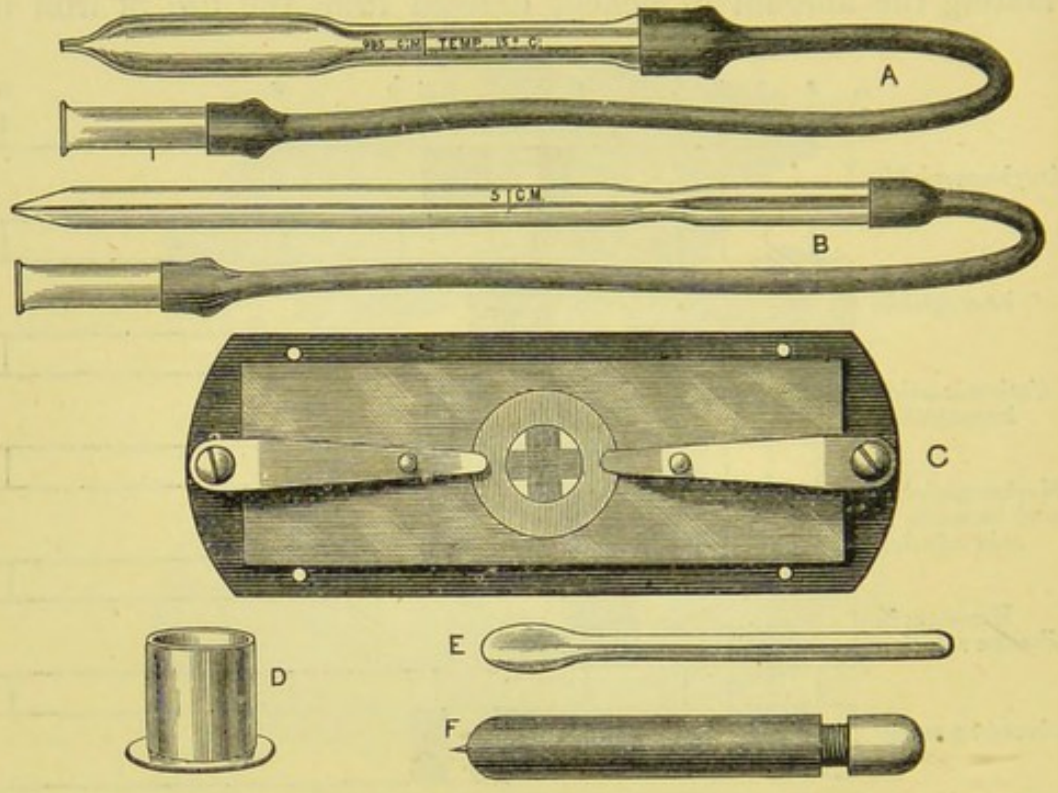



FIG. 16.—Gowers's Apparatus for counting Blood Corpuscles.

A. Pipette for measuring the diluting solution; B. Capillary tube for measuring blood; C. Cell, with divisions in the floor, mounted on a slide to which springs are fixed to secure the cover-glass; D. Vessel in which dilution is made; E. Spud for mixing the blood and solution; F. Guarded needle.

Two changes in the blood-pigment are of special clinical importance. (1.) Under the influence of many drugs, such as chlorate of potassium, nitrite of amyl, permanganate of potassium, phenacetin, and antifebrin, the oxyhæmoglobin of the blood is changed into methæmoglobin, which shews a characteristic spectrum, as in Fig. 15. (2.) In cases of poison-

ing with CO, the blood yields the spectrum of carbonic-oxide-hæmoglobin.

### Microscopic Examination of the Blood.

Under this heading we shall consider; (1) the process for counting the number of the blood-cells; (2) the histological changes which they may exhibit in disease; and (3) the parasites of the blood.

1. *Enumeration of the Corpuscles.*—This method of investigation, which was first attempted by Vierordt, has been followed out by numerous observers, many of whom have described special forms of apparatus for the purpose. While the recent modifications of Malassez's instrument, and the apparatus of Thoma-Zeiss, give very accurate results, I shall here describe the instrument designed by Gowers, which is exceedingly handy, and is the form most frequently used in this country.

Gowers's *Hæmocytometer* consists of—(1) a small pipette (A in Fig. 16), which, when filled to the mark on its stem, contains 995 cubic millimetres; (2) a capillary tube (B) marked to contain five cubic millimetres (each of these is furnished with an india-rubber tube and mouthpiece); (3) a small glass jar (D), in which the blood is to be diluted; (4) a glass stirrer (E) for mixing the blood and the solution in the jar; (5) a brass stage-plate (C) carrying a glass slip, on which is a cell $\frac{1}{5}$th of a millimetre deep. The bottom of this is divided by intersecting lines $\frac{1}{10}$th millimetre squares. Upon the top of the cell rests the cover-glass, kept in position by means of two springs.

The diluting solution may vary. One which answers well consists of a solution of sulphate of soda, 26 grains in one ounce of distilled water, to which is to be added 15 drops of strong acetic acid.

When an observation is to be made, 995 cubic millimetres of the solution are measured by means of the pipette, and placed in the mixing jar. The patient's finger is now to be pierced with the spear-pointed needle supplied along with the instrument, and a drop of blood obtained without any squeezing of the finger, which would alter the proportion of serum and cor-

puscles in the blood. Five cubic millimetres of this blood are drawn into the capillary tube, and then blown into the diluting solution in the jar, any superfluous blood being previously removed from the point of the pipette by means of a soft cloth. The two fluids are well mixed by rotating the stirrer between the finger and thumb, after which a small drop is placed in the centre of the cell, and the cover-glass applied. The drop must lie in the *middle* of the cell, and must not touch its sides. The slip is now placed upon the stage of a microscope, and the lens focussed for the squares, which are marked out by lines drawn on the floor of the cell. In a few minutes the corpuscles sink through the drop, and come to rest on these squares. The number in ten squares is then counted, and this multiplied by 10,000 gives the number in a cubic millimetre of blood.

In counting the white corpuscles, it is well to observe the number of squares in a field and then the number of white corpuscles in a series of fields—raising the focus until they appear like bright points on account of their refractive power.

*The volume relation of corpuscles to plasma* may be determined by means of the hæmatokrit, either the instrument of Hedin or the simpler form of Gaertner. In both cases the blood is drawn into a graduated capillary pipette and centrifugalised, after which the depth occupied on the graduated scale by the layer of corpuscles gives the percentage present.

As the average of the figures of many observers, it may be taken that the number of red corpuscles in each cubic millimetre of blood is in the man, 5,000,000; in the woman, 4,500,000. No doubt, in very perfect health, these figures are exceeded, and an apparent increase in the number of red corpuscles is seen where there has been much loss of water from the blood, as in the severe diarrhœa of cholera.

Diminution in the number of red corpuscles (Oligocythæmia) follows hæmorrhage, but is then of a temporary character. More permanent diminution is seen in all forms of anæmia, being most conspicuous in the pernicious variety.

The leucocytes of the blood vary considerably in number in

different persons. The figures given vary from six to nine thousand in the cubic millimetre. An increase in the number of leucocytes (leucocytosis) is met with very frequently, both under physiological and under pathological conditions.

Physiological leucocytosis is seen in health after a meal, and then depends chiefly on the amount of albuminous food consumed. It also occurs in pregnancy, and in the newly-born infant.

Pathologically, leucocytosis is seen in many infective processes, chiefly where there is exudation into the tissues. Thus it is seen typically in croupous pneumonia, in inflammation (non-tubercular) of the serous surfaces, pleurisy, peritonitis, meningitis, in diphtheria, in acute rheumatism, in scarlet fever, and in various septic processes. Leucocytosis also shows itself as the result of malignant growths. It occurs after copious hæmorrhage, and it is seen shortly before death—the agonal leucocytosis of Litten.

But much more striking increase in the number of the white cells takes place in leucocythæmia, where the enumeration may exceed 500,000 in the cubic millimetre.

**Morphology of the Blood-Corpuscles.**—Blood films are usually prepared by receiving a small drop of blood from the ear or the finger, on a cover-glass, applying another to it, and then separating them by sliding the one cover-glass on the other. These films may be examined fresh, or they may be fixed and stained. Such films are fixed by being kept at a temperature of about 120° C. for ten or twelve hours, or by being placed in a saturated solution of corrosive sublimate, or in a mixture of equal parts of ether and absolute alcohol for a short time. In staining, a great variety of fluids have been used. That of Ehrlich is composed as follows :—

| | | | |
|---|---|---|---|
| Saturated solution of | | Orange G. | 120-135 c.c. |
| Do. | do. | acid Fuchsin | 80-165 c.c. |
| Do. | do. | Methyl Green | 125 c.c. |
| | add, | water | 300 c.c. |
| | | absol. alcohol | 200 c.c. |
| | | glycerine | 100 c.c. |

The cover-glass, after the film has been fixed, is floated for about two minutes on this stain, washed in water, and mounted in canada balsam.

**Red Blood-Corpuscles (Erythrocytes).**—Even in health there is some difference in the size of the red corpuscles, their diameter varying (according to Hayem) from 7·2 to 9$\mu$, but changes very much more marked than this are seen in anæmia, especially in the pernicious form, where small corpuscles (microcytes), measuring about 3$\mu$, and large (macrocytes), measuring from 9 to 12$\mu$, are found.

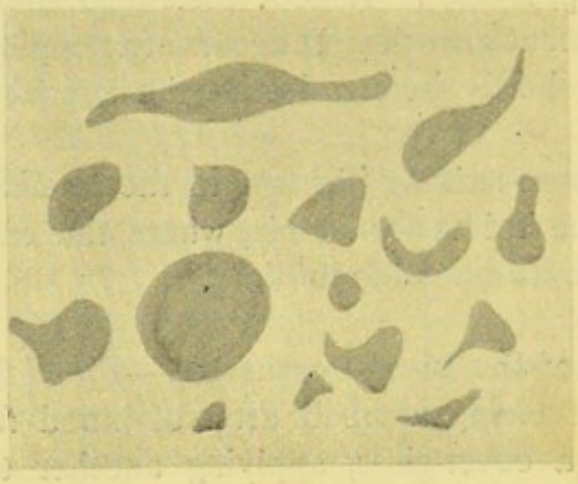


FIG. 17.—Poikilocytosis.

The shape, also, of the corpuscles is apt to be altered in anæmia, and all sorts of irregular forms may be encountered, the corpuscles appearing pear-shaped, kidney-shaped, flask-shaped, &c. This condition of the blood, called *Poikilocytosis*, attains its highest degree in cases of pernicious anæmia.

Other signs of degeneration in the red corpuscles may be met with, such as peculiarities in their reaction to various stains, increase in the size of the central depression, or diminution of the pigment at that part of the disc.

The appearance of nucleated red corpuscles is not uncommon in the blood. These are, according to Ehrlich, of two varieties, (1) Normoblasts, of about the average size of a normal corpuscle,

and containing a nucleus which stains very deeply with basic dyes, while the cell-body takes on acid stains, and (2) Megaloblasts, which are three or four times larger, and are rounder and thicker than the normal cell. They also are nucleated. The first variety is considered to indicate that the blood is undergoing regeneration. The second form occurs in pernicious anæmia.

**White Blood-Corpuscles (Leucocytes).**—There is considerable confusion as to the nomenclature of the different forms of leucocytes met with in the blood. The following list may suffice to indicate their more common designations, and the proportion which each bears, under normal conditions, to the total number of leucocytes in the blood :—

| | |
|---|---|
| Lymphocytes, | 20—30% |
| Hyaline cells, | 2% |
| Oxyphile (neutrophile) cells, | 60—70% |
| Eosinophile cells, | 2—4 % |
| Coarsely granular basophile cells, | $\frac{1}{4}$—$\frac{1}{2}$ % |
| Myelocytes, | Not present normally. |

For a detailed description of these various forms, special works, in particular those of Gulland, of Sherrington, and of Kanthack and Hardy, should be consulted.

Under certain pathological conditions the numerical relation which these cells bear to each other may be disturbed. Very great obscurity surrounds this subject, but the following points may be noted.

*Lymphocytes.*—These are microscopically similar to the small elements of lymphoid tissue. In infancy they are present in large numbers, and in the rickets and syphilis of childhood they tend to become increased. In adults an increase of these cells may take place in chlorosis, pernicious anæmia, and in many forms of debility, but it is seen very specially in the lymphatic form of leucocythæmia.

*The neutrophile*, or, more properly, *oxyphile cells*, vary much in size and in the shape of their nucleus. Usually rather larger

than a red corpuscle, this form of leucocyte possesses a finely granular cell body, and a nucleus which is polymorphous. This class includes most of the phagocytes, forms the bulk of purulent exudations, and is largely increased in most cases of leucocytosis.

*Eosinophile cells.*—The protoplasm of these cells shews coarse, highly refractive granules, which stain deeply with eosin and other acid aniline dyes. The nucleus is large and is shaped like a horse-shoe. These cells are increased in number in many cases of leucocythæmia, and also sometimes in gout, as well as in various diseases of the bones and of the skin. They are diminished in number in acute pneumonia, and in certain other fevers.

*Myelocytes*, commonly met with in bone marrow, are not seen in normal blood. They are of large size, with a nucleus almost filling the cell, and they sometimes stain with eosin. They are seen in the blood in large numbers in cases of pernicious anæmia, and particularly in the splenic-myelogenous form of leucocythæmia. Occasionally a few are met with under other conditions.

## Parasites of the Blood.

Many microbes may from time to time be found in the blood, such as the bacillus of anthrax, of tubercule, of tetanus, staphylococcus, streptococcus, and many others. These are not, however, primarily blood parasites, and they need not be further alluded to here. Of those forms which are specially parasitic in the blood, and the recognition of which has a special diagnostic value, there are few, and to these only attention will be directed in this chapter.



Fig. 18.—Bacillus Anthracis. (After Koch.)

*Spirochæte Obermeierii.*—This spirillum, which is found in the blood of patients suffering from recurrent fever, possesses a very characteristic appearance. On examining a specimen of such blood with the microscope, it will be seen that, moving actively

among the blood corpuscles, are a number of very thin, thread-like organisms. Their length is from three to six times greater than the diameter of a red corpuscle, and in shape they resemble a cork-screw. Their movements are sometimes so vigorous as to move the blood corpuscles with which they may come in contact in their course. They stain, in blood films, with Löffler's solution. When the blood is examined in cases of recurrent fever these spirilla begin to appear shortly before the temperature rises. On the second or third day of the fever they are found in enormous numbers. During the crisis they disappear from the general circulation, and in the intervals they can hardly be found.

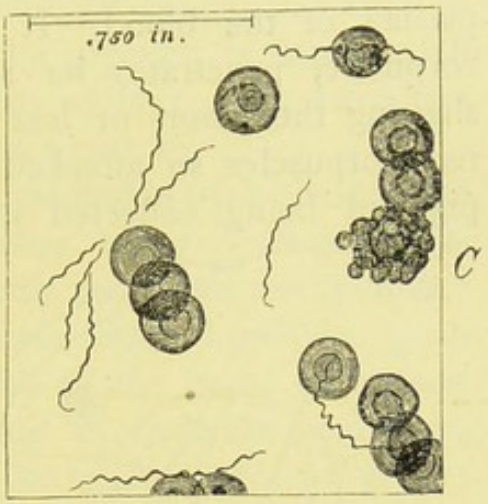



FIG. 19.—Spirochæte Obermeierii. (After Carter.)

*The Malarial parasite*, usually known as the *plasmodium* or *hæmatozoon malariæ*, is a protozoon, and was discovered by

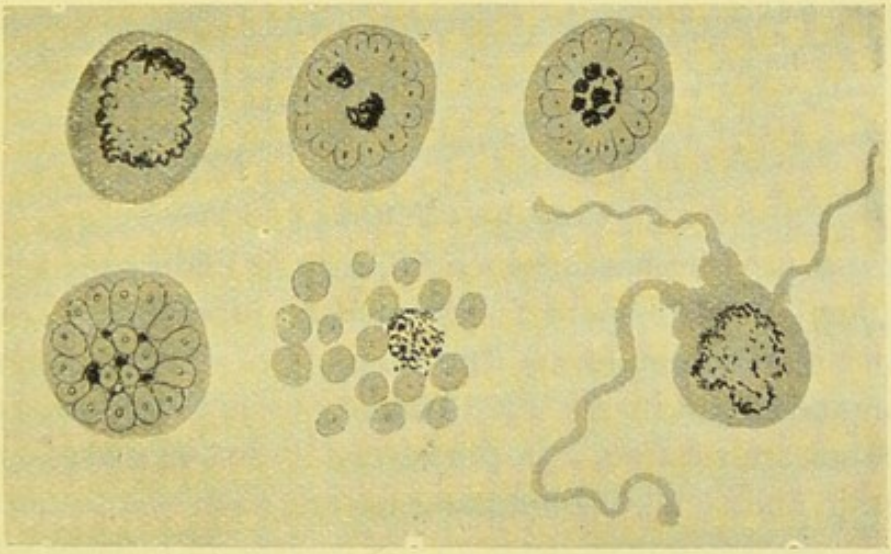


FIG. 20.—Malarial Parasite (after Thayer and Hewetson).

Laveran in 1880. Different varieties of this organism are met with in the tertian, in the quartan, and in the malignant and

acyclic forms of malaria. Each of these varieties passes through a cycle of development, the characteristics of which are pretty well known. At first the organism appears as a small round protoplasmic mass, sometimes flagellated and floating free in the plasma of the blood. It presently attaches itself to a red corpuscle, penetrates its substance, and proceeds to grow, shewing then more or less active amœboid movements. The red corpuscles so attacked usually swell, become pale, their pigment being collected in granules within the plasmodium.

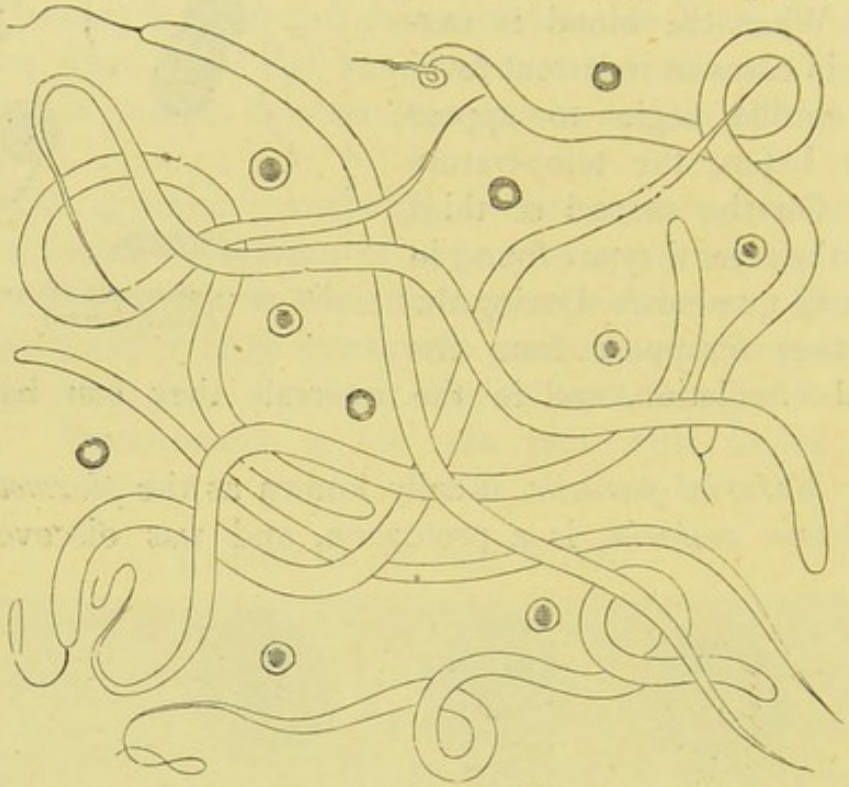


FIG. 21.—Filaria Sanguinis Hominis. (Roberts.)

When the parasite has attained full size, it undergoes a process of segmentation or spore formation (this operation coinciding with the rigor of the fever), and then often presents the rosette appearance shewn in Fig. 20. The corpuscle then breaks up and the spores are set free. In the severer forms of malaria various crescentic and sickle-shaped parasites are seen.

The main differences between the parasite of quartan and that of tertian fever are as follows :—

In quartan fever the duration of the cycle of development of the parasite is 72 hours. The red corpuscles are not decolorised,

nor do they swell up. The pigment granules are coarser and darker than in tertian, the amœboid movements are less active, and the number of spores formed is from 6 to 12.

In tertian fever the cycle is one of 48 hours, the red corpuscles become swollen and pale, the pigment is deposited in fine granules, the amœboid movements of the parasite are active, and the number of spores ranges from 15 to 20.

The plasmodium of malaria is best detected in fresh blood, using, if possible, a hot stage, and examining with a $\frac{1}{12}$ oil-immersion lens. Blood films may be stained with eosin and methyl-blue.

*Filaria sanguinis hominis* is the name which Lewis has given to a nematode worm which is found in the blood of persons suffering from chyluria, certain forms of endemic hæmaturia, elephantiasis, lymphoid affections, &c. These *filariæ* are exceedingly minute, measuring about 0·3 mm. by 0·0075 mm., and are the embryo form of parasites inhabiting other tissues. There are several varieties of these *filariæ*—one which is found in the blood only during the night, one only during the day, and one constantly.

*Distomum hæmatobium* or *Bilharzia hæmatobia* is a parasite inhabiting the portal and mesenteric veins, found in Egypt in cases of hæmaturia. Its presence in the blood is never detected during life.

### Serum-diagnosis of Typhoid Fever.

This very valuable method of diagnosis was first described by Widal in 1896. It depends upon certain properties which the blood of patients suffering from typhoid fever acquires, whereby, when the blood-serum is mixed with a culture of actively moving typhoid bacilli, these bacilli rapidly lose their motility, and become aggregated in clumps.

A drop of the blood to be examined is mixed with nine times its volume of neutral bouillon. Of this mixture, a drop is placed on a microscopic slide and mixed with a drop of a 24-hours' culture of typhoid bacillus, containing rapidly moving bacilli. This mixture is to be covered with a cover-glass and examined with a moderate power.

In a few minutes, if the case is one of typhoid fever, the bacilli become motionless, and run together to form larger or smaller clumps or clusters. If the case from which the blood came be not one of typhoid fever, no such change will be observed.

The phenomenon is a very striking one to witness, and this method of diagnosis is proving of much service in the diagnosis of doubtful cases. During the first week of the fever the reaction is difficult to obtain, but in the succeeding weeks it becomes very distinct, and the blood appears to retain this power for many months after the attack of fever is over.

## CHAPTER VII.

# CIRCULATORY SYSTEM.

### SUBJECTIVE PHENOMENA.

BEFORE addressing ourselves to the physical examination of the heart, there meet us for consideration certain symptoms of a more or less subjective kind.

1. *Pain.*—In anæmic persons, and particularly in women suffering from uterine disease, from chlorosis, or from nervous affections, pain over the region of the heart is frequently complained of, and true cardiac pain may likewise be simulated by neuralgia in the chest wall. In heart disease of any kind, and in particular in fatty degeneration associated with gout, pain may be a more or less prominent symptom. In its most pronounced form—angina pectoris—it comes on in recurring attacks of short duration, but of extreme severity. The first of the attacks usually occurs when the patient is making some exertion. The chest feels as if held in a vice, the pain, which is always severe, and which may be of the most intense character, radiates from the heart to the shoulders, and down the left arm, or down both arms to the wrist, breathing almost ceases, the countenance sometimes becomes livid, and consciousness may be lost. The attack usually passes off as rapidly as it came on, and the patient may be free from its repetition for months or years.

2. *Palpitation.*—The abnormal perception of excited pulsation in the heart or aorta is very frequently due to mental excitement, to dyspepsia, flatulence, anæmia, or nervous debility, or to the action of tobacco, tea or coffee, and is often met with in cases of exophthalmic goître. It also occurs as a result of organic disease of the heart, and in such cases it will be found to be aggravated

by exertion. The palpitation may be objective—*i.e.* the physician may himself recognise the excited action of the heart; or it may be merely subjective—the patient complaining of that sensation without there being the slightest evidence from physical examination of any alteration in the strength of the cardiac pulsations. Derangements of cardiac rhythm will be noticed hereafter.

3. *Fainting* (*syncope*), which is primarily due to failure of the heart's action, is usually ushered in by a train of symptoms, of which the chief are—pallor of the face, chilliness, cold perspiration, a feeling of weakness, of sinking in the epigastrium, and of sickness, pulse small and rapid, or slow and irregular, dimness of vision, ringing in the ears, and gradually increasing unconsciousness. Syncope may be due to organic disease of the heart, to nervous disturbance of the cardiac action (central or reflex), to intense mental emotion (hysteria), to deficiency of the blood supply to the heart muscle, or to want of blood in its cavities. It is of short duration, seldom lasting more than half-an-hour, and can in this way be distinguished from shock. Syncope may be simulated by apoplexy and by epilepsy, but the absence of paralysis and of muscular spasms enables a diagnosis to be readily arrived at. The action of alcohol in large amount, and of certain poisons, may produce a state closely resembling syncope, but the state of the pulse is here a reliable guide, unless, indeed, the poison used act as a cardiac depressant, when the diagnosis may be a matter of very great difficulty.

4. *Dyspnœa.*—The phenomena of dyspnœa, or difficulty of breathing, will be described under the head of the respiratory system. It is, however, necessary to note here that this condition is common is valvular disease of the heart, due to the fact that the pulmonary circulation is interfered with. The dyspnœa may be constantly present, or may only show itself after exertion.

## CHAPTER VIII.

## CIRCULATORY SYSTEM—(*continued*).

### INSPECTION.

The cardiac or *præcordial region* corresponds to the lower part of the anterior mediastinum. It may be said to extend vertically from the second interspace to the sixth cartilage, and transversely from the apex-beat to a point about three-quarters of an inch to the right of the sternum.

The region so marked out overlies the heart and the margins of both lungs which overlap it. More deeply still lie the organs contained in the posterior mediastinum.

In this chapter will be considered—(1) the form and appearance of the præcordia, and (2) the various pulsatory movements which show themselves on the walls of the thorax.

**Præcordia.**—A slight degree of bulging of the thoracic wall in the cardiac region is more readily detected by simple inspection than by means of measurement. It may be the result of curvature of the spinal column anteriorly and to the left, but is more commonly caused by cardiac hypertrophy, pericardial effusion, aneurismal and other tumours adjacent to the heart, or circumscribed pleuritic effusions. When effusion takes place into the pericardial sac, the intercostal spaces widen, they become raised to the level of the ribs, and ultimately may even protrude beyond them. Bulgings caused by aneurisms lie, almost without exception, above the fourth rib.

Depression of the præcordial region, on the other hand, may occur during the absorption of a pericardial effusion, and may remain permanently if extensive adhesions have been formed.

Flattening of the præcordia may only be caused by retraction of the left lung, the result of pleurisy or of fibroid phthisis.

**Pulsations.**

1. *The Cardiac Impulse.*—In health the apex-beat of the heart is found in the fifth left interspace, about an inch and a half or two inches from the left margin of the sternum, and its area does not exceed a square inch in extent. In childhood, however, and in persons who have a short and wide thorax, it may stand somewhat higher, and may be thrown farther to the left; whilst in old age, and in individuals whose thorax is very long and narrow, the cardiac impulse is lower than normal.

While natural breathing does not affect its position, deep inspiration and expiration cause respectively depression and elevation of the apex-beat.

When the patient lies on either side, the apex-beat is deflected in a corresponding direction. This alteration is more marked towards the left.

The cardiac impulse does not always make itself visible on the chest wall. This is usually due to great development of fat or muscle, and in such cases we can by palpation almost always fix the position of the apex-beat.

It may be noted that in certain rare cases there is a congenital transposition of the viscera, the heart lying then on the right side of the thorax.

[Pathological changes in the position of the apex-beat will be considered under the head of Palpation.]

*Systolic indrawing* of the thoracic wall is of two varieties —(1) a recession, which is exactly synchronous with each ventricular systole; and (2) one which immediately succeeds the retirement of the apex of the heart from the chest wall.

The former variety is sometimes met with in healthy persons (particularly children) in whom the chest walls are unusually thin. It occurs in the third and fourth interspaces, and is simply the result of that recession of the base of the heart which is synchronous with the forward movement of the ventricles.

The chest walls are sucked inwards (or rather forced inwards by atmospheric pressure), to prevent the formation of the partial vacuum behind them which would otherwise take place.

The second form is seen at the apex, and is, according to Skoda, pathognomonic of adherent pericardium.[1] If the adhesion be extensive (and particularly if the parietal layer be adherent anteriorly to the wall of the chest and posteriorly to the vertebral column), not merely the intercostal space but even the ribs may be drawn inwards, following the apex of the heart.

(2.) *Pulsation at the root of the neck* may be arterial or venous.

Pulsation in the carotid arteries becomes evident whenever the heart's action is increased in strength (as after great bodily exertion, or from mental excitement), but in its most pronounced form such pulsation is seen in cases of hypertrophy of the left ventricle, along with aortic incompetence. Pulsation in the jugular fossa, when well marked, usually points to simple or aneurismal dilatation of the aorta.

Swelling of the jugular veins is found in cases in which there is some obstruction to the return of blood to the heart, whether that obstacle be situated in the systemic or pulmonary circulation. If from any cause the right ventricle be unable to empty itself completely of blood, it becomes engorged, and, reacting on the right auricle, causes its dilatation; while the auricle so dilated, in its turn retards the flow of blood through the jugular veins, which then exhibit distension. The same effect will, of course, be produced by any obstruction to the return of blood to the heart, whether seated in the lungs themselves, or at the mitral orifice, or the valves which close it.

This distension is necessarily accompanied with more or less pulsation in the vein, the blood being only able to reach the heart during inspiration. This, however, is not the only pulsatory movement which the veins in this region exhibit when they are in a state of distension. The systole of the right ventricle causes a vibration which passes through the tightly-stretched

[1] This sign is not, however, invariably present in such cases, and though adherent pericardium is by far its most common cause, yet it may occur in cases in which the normal movements of the heart are otherwise hindered.

right auriculo-ventricular valve, and the thrill thus communicated to the blood in the dilated auricle is thence transmitted to the jugular veins. In this case the tricuspid valve and the valves at the mouth of the jugular veins are competent, and there is, therefore, no backward flow of blood into auricle or vein; it is simply the impulse which is transmitted. This we can readily satisfy ourselves of by compressing the right jugular vein high up in the neck, and then if the contents of the lower part of the vessel be pressed out, the vein will not fill again from below, since no valvular incompetency exists.

When, however, the tricuspid valve is incompetent, or when the valves in the jugular vein cease to close the lumen of that vessel (either from destruction of its valves, or from extreme dilatation of the vein preventing the valves from doing their duty), the vein when so emptied will be seen to fill from below with regular pulsations corresponding to those of the right ventricle.

Thus is formed the "venous pulse," one of the most important signs of tricuspid incompetence.

Jugular pulsation may occasionally be præsystolic in rhythm, the movement resulting from the transmission of the impulse of the auricular systole into the vein.

Sudden collapse of the jugular veins during the ventricular diastole has been shown by Friedreich to be a sign of pericardial adhesion.

3. *Epigastric Pulsation* may be conveniently divided into two groups—(1) that which is synchronous with the ventricular systole, and (2) that which follows the systole after a slight, but appreciable, delay.

(*a.*) *Synchronous with the Ventricular Systole.* — When the right ventricle is hypertrophied and dilated, it may frequently be felt to pulsate in the epigastrium, and any condition which depresses the diaphragm, or forces the heart towards the right, may give rise to such pulsation.

The liver may also pulsate in the epigastrium, but if the impulse is exactly systolic in rhythm, it can only be occasioned by direct transmission from the adjacent right ventricle.

(*b.*) *Delayed Epigastric Pulsation—i.e.* that which succeeds the ventricular systole after an appreciable interval—may be due to the transmitted impulse of the abdominal aorta. The pulsation is then somewhat to the left of the middle line; it extends downwards towards the umbilicus, and is not diffused laterally. It may be conducted to the parieties by means of tumours, or through the overlying liver. The pulsation may be due to an aneurism on the abdominal aorta or one of its branches, when it will have a distensile character.

The venous pulsation which has been already noticed as occurring in cases of incompetence of the tricuspid valve is not limited to the jugular veins. It also takes place in the inferior vena cava, and the pulsation may in this way be communicated to the liver. If the hepatic veins be likewise affected, the pulsation in the liver becomes not merely heaving but distensile.[1]

In all these conditions the pulsation follows the apex-beat after a slight interval of time. The delay can be best appreciated by fixing with wax, over each pulsating point, a bristle carrying a small flag.

Systolic indrawing of the epigastrium occurs rarely, and is caused by extensive pericardial adhesions.

4. *Arterial Pulsations on the Thoracic Wall.*—Aortic aneurisms frequently give rise to visible pulsation in the upper part of the thorax, above the third rib. Such pulsation is systolic in rhythm, being as nearly as possible synchronous with the ventricular systole. If the ascending portion of the arch be involved, the pulsation usually lies to the right of the sternum; if the transverse portion be the seat of the disease, the pulsation is more in the middle line; and if the aneurism affect chiefly the descending part of the arch, the pulsation lies to the left side of the sternum. Aneurisms of the innominate and subclavian arteries also give rise to visible pulsation in the walls of the thorax. Systolic pulsation at the second left interspace is

[1] To discriminate the various epigastric pulsations mentioned requires the use of palpation as well as inspection, but to preserve the continuity of the subject, they are all grouped together in this chapter.

sometimes communicated to the surface from the subjacent pulmonary artery in cases of retraction of the borders of the lungs.

A diastolic impulse is sometimes to be seen and felt over the seat of the aortic and pulmonary valves in very emaciated persons, especially when the borders of the lungs have become retracted.

*Capillary Pulsation* can occasionally be observed on the cheeks, beneath the nails, or in the line of congestion caused by drawing a sharp point, such as that of a pencil, over the skin, and though sometimes occurring independently of that cause, is usually due to incompetence of the aortic valves, with hypertrophy of the left ventricle.

# CHAPTER IX.

## CIRCULATORY SYSTEM—(*continued*).

### PALPATION.

THE skilled application of the hand to the cardiac region gives important information regarding the heart, which inspection alone is not fitted to communicate, and confirms much that inspection has already indicated. Palpation deals chiefly with the phenomena of the cardiac impulse, and with the various thrills which may occur in connection with the heart's action.

### CARDIAC IMPULSE.

The apex-beat has already been spoken of in the last chapter in its normal condition. We now consider the various changes which it may undergo in disease—alterations in position, in strength, and in extent—remembering that in health the impulse lies between the fifth and sixth ribs, about one and a half or two inches to the left of the sternum; that while ordinary respiration does not affect its position, it is depressed and elevated to a very slight extent by deep inspiration and expiration; and that when the patient lies on the left side it is slightly displaced outwards.

**Alterations in the position of the Apex-beat.**

1. *Vertically.*

The height depends simply on the level of the diaphragm.[1]

Pulmonary emphysema and spasmodic contraction of the diaphragm cause a general depression of the diaphragm, while

[1] With the exception of cases of pericardial effusion, when the diaphragm may be depressed and the apex-beat simultaneously raised.

collections of liquid or of gas in one pleural cavity or in the pericardial sac, an increase of the weight of the heart (particularly from hypertrophy of the left ventricle), or the presence of tumours in the neighbourhood of that organ, produce a local depression in the diaphragm, and each of these conditions finds expression in a lowered position of the apex-beat.

On the other hand, if the diaphragm be raised owing to cirrhosis of one lung, or to contraction following the absorption of a pleural effusion, or by reason of increased abdominal pressure, the result of tumours, ascites, sympanites, &c., the apex-beat will be correspondingly elevated.

2. *Laterally.*

(*a.*) *To the right.*—Hypertrophy and dilatation of the right ventricle displace the apex-beat from its normal position towards the right. In slighter cases the pulsation is greatest in the epigastrium, but in those which are more marked, the apex, or at any rate that portion of the heart-wall which represents the apex, beats under the right edge of the sternum, or even farther to the right. Effusions in the left pleural cavity and tumours of the left lung also push the heart over to the right side; and on the absorption of the exudation, the heart returns again to its normal position, provided that it has not become bound down by adhesions in its abnormal situation. Retraction of the right lung, the result of pleurisy or of cirrhosis, will also cause the apex-beat to move to the right. The apex-beat may be found on the right side of the thorax in cases of congenital transposition of the viscera.

(*b.*) *To the left.*—Hypertrophy and dilatation of the left ventricle not only depress the apex-beat, but also move it considerably to the left. In cirrhosis of the left lung the heart follows the contracting lung towards the left, and effusions into the right pleural cavity, and tumours of the right lung, also produce a movement of the apex-beat in this direction, when they are of considerable amount.

**Strength of the Apex-beat** varies much even in healthy individuals, owing to the varieties in the thickness of the chest-wall,

in the width of the intercostal spaces, and in the extent to which the apex is overlapped by pulmonary tissue. Pathologically, the differences are still more apparent.

Diminished force of the cardiac impulse, even to the extent of being imperceptible to the finger, may be due to—

1. *Intrinsic causes.* — These include abnormal innervation, fatty degeneration of muscular fibre of the heart, myocarditis, and that degeneration of the heart muscle which follows hyperpyrexia of long duration, and lastly, deficiency of proper blood supply.

2. *Extrinsic causes.*—When the visceral and parietal layers of the pericardium become adherent, the movements of the apex of the heart are so interfered with, that all evidence of cardiac impulse may be lost. Effusions of fluid or gas into the pericardium or into the left pleural cavity, as well as intervention of emphysematous lung tissue between the heart and the thoracic wall, all tend to weaken the apex-beat.

*Increase in force* of the cardiac impulse may be of neurotic origin; it may arise from violent exertion or from strong emotions; it may be the result of fever, of endocarditis, of pericarditis, or of any condition which is more than usually favourable to the conduction of the impulse, but by far its most common cause is hypertrophy of the heart. The heaving impulse which results from hypertrophy of the left ventricle is much more easily detected than that which hypertrophy of the right ventricle occasions, as the latter has its point of maximum intensity behind the sternum, and it is in any case never so great as that of the left ventricle.

The *rhythm* of the cardiac pulsations will be noticed in the remarks made on the arterial pulse.

**The Extent of the Impulse.**—Normally, the apex-beat is not perceptible over more than an area of about a square inch, and is limited to the fifth intercostal space. When the pulsation extends much beyond such limits, it is abnormal. In disease the apex-beat not unfrequently becomes diffused over a considerable area, and this may result from increased action of a normal

heart (medication, excitement, &c.), from cardiac hypertrophy, from the application of an unusually extensive area of the heart to the thoracic walls (retraction of the lungs), or merely from great thinness of the chest-wall.

**Double Apex-beat.**—Independent, non-synchronous contraction of the ventricles has been met with in a few rare cases (Charcelay, Leyden, Roy, &c.), where the systole of the right ventricle caused a pulsation in the jugular veins which alternated with that in the carotid arteries.

**Thrills** may be felt by the hand applied over the cardiac region, and these are of two kinds—

1. *Endocardial thrills* are caused by the vibrating eddies which ensue when the blood current is forced through a small opening into a wider space. These conditions are satisfied in cases of stenosis of one of the orifices of the heart, or incompetence of a valve, when at the same time the blood current is sufficiently rapid. The pathological condition which gives rise to the thrill is indicated by the seat of greatest intensity, and the time in relation to the various phenomena of cardiac action.

Thrills in the mitral area (a circle with a radius of one inch round the apex-beat) are systolic or præsystolic, according as they are produced by incompetence of the mitral valve or stenosis of the orifice which that valve covers. Thrills over the second right costal cartilage arise from aortic stenosis or incompetence; in the former case being systolic, and in the latter diastolic in rhythm. Præsystolic and systolic thrills in the tricuspid area indicate respectively stenosis of the orifice and incompetence of the valve. Very rarely a systolic thrill is felt over the pulmonary artery, denoting stenosis of the pulmonary orifice, or a diastolic, indicative of regurgitation.

2. *Pericardial friction fremitus* caused by the rubbing during the heart's action of the two pleural surfaces, which have been rendered rough and uneven by the effusion of lymph, is more readily detected by auscultation than by palpation.

## CHAPTER X.

## CIRCULATORY SYSTEM—(*continued*).

### PERCUSSION.

THE heart, lying in the thoracic cavity, has, in its normal condition, the following relations to the anterior wall of the chest:—

The *right border*, formed almost entirely by the right auricle, stretches in a slightly curved manner from the second right intercostal space, or third costal cartilage, downwards and outwards to the cartilage of the fifth rib on the same side, just at its junction with the sternum. The *left border*, formed by the left ventricle, reaches from the second intercostal space on the left side downwards and outwards to a point about half an inch outside of the apex-beat. The *lower border* corresponds pretty exactly to a line joining the sternal end of the fifth right costal cartilage with the apex-beat. The highest portion of the heart, formed by the appendix of the left auricle, reaches a level which may be indicated by a line joining the lower border of the sternal ends of the second pair of costal cartilages.

To percuss the heart, it is best to have the patient recumbent. No strong percussion is, as a rule, required, and this is specially the case with regard to the absolute dulness, where the tap should be light. In addition to the note elicited, the sense of resistance perceived during percussion is of great value.

The greater portion of the heart is separated from the chest wall by the overlapping lung, and therefore it is only the small uncovered part, consisting of the right ventricle, which yields

on percussion an absolutely dull note. This area of *absolute or superficial cardiac dulness* is readily mapped out. Its right border extends along the mid-sternal line, from the level of the fourth to that of the sixth costal cartilage. The left border is an irregular line stretching from the upper end of the right border to the apex-beat. The lower edge cannot be defined by percussion, because at this point the cardiac merges into the hepatic dulness. Its position can, however, be obtained with approximate accuracy by drawing a line from the apex-beat to the lower end of the right border of the cardiac dulness in mid-sternum. It usually measures from three to four inches. The area thus formed is roughly quadrilateral in shape; and not only do its extent and position vary with each respiration, but they are also affected by the position of the person, and further, by the constant changes of the diaphragm in respiration, necessarily producing corresponding changes in the lie of the heart. The condition of the margins of the lungs (as in emphysema) have also an important influence on the size and position of the area of absolute cardiac dulness. The variations to which this area is thus liable greatly diminish the value of its condition as indicative of disease.

The region of absolute dulness is *increased* in—

(1.) *Hypertrophy and Dilatation of the Left Ventricle*, when the increase takes place chiefly downwards and to the left.

(2.) *Hypertrophy and Dilatation of the Right Ventricle.*—In this case the left margin is little interfered with, while the right is thrown outwards towards the right.

(3.) *Serous Effusion into the Pericardium.*—The dulness here takes a pyramidal form, being limited by the pericardial sac, with the base downwards, resting on the diaphragm.

(4.) Retraction of the borders of the lungs, resulting from cirrhosis or pleuritic adhesions. In such cases a larger area of the heart is exposed, and hence there is an increase of dulness.

(5.) Increase of absolute cardiac dulness may be simulated by various pathological conditions of the neighbouring organs, such as infiltration of the margins of the lungs, pleuritic effusions, &c.

It must be borne in mind, however, that these various diseased conditions of the heart and its investing sac may be present without giving rise to any appreciable changes in the absolute cardiac dulness.

The area of absolute dulness is diminished, or entirely lost, in—

(1.) *Left pneumo-thorax*, where the collection of gas in the left pleural cavity is so great as to force the heart to the right. The area of dulness is usually, in such cases, still to be detected to the right of the sternum; but it is much diminished in size.

(2.) *Emphysema*, when well pronounced, entirely does away with absolute cardiac dulness, the margins of the lungs approaching so near to one another as to overlap the heart completely. In slighter cases the area is only diminished in size. A similar result is produced in these cases in which the margins of the lungs have been fixed over the heart by old pleuritic adhesions.

(3.) In the rare cases in which free gas is found in the pericardium (pneumo-pericardium), percussion elicits a clear note over the whole cardiac area.

(4.) Where the heart is itself of small size, from atrophy, the cardiac dulness will suffer a corresponding diminution.

If, then, as has been said, the indications regarding the heart itself, obtained by examination of the area of absolute cardiac dulness, may be vitiated by various pulmonary conditions, we must seek to obtain information from the percussion of that portion of the heart which is covered by a more or less thick layer of lung.

In percussing the thorax, as we pass towards the cardiac area, the note, which is at first purely pulmonary in character, grows more and more dull, until the limit of absolute cardiac dulness is reached. The reason of this change in the note will be fully discussed when the subject of percussion comes to be treated of as a whole in a subsequent chapter.

It is by noting the point at which this change in the note occurs that the position of the outer margin of the heart can be determined by percussion. This is called the area of *relative*

*or deep cardiac dulness* (see Fig. 22). Now, although it is in most cases not difficult to sketch out the entire area of relative dulness, yet for ordinary clinical purposes it is only necessary to percuss in two directions—vertically, parallel to the left margin of the sternum, and transversely, at the level of the fourth rib.

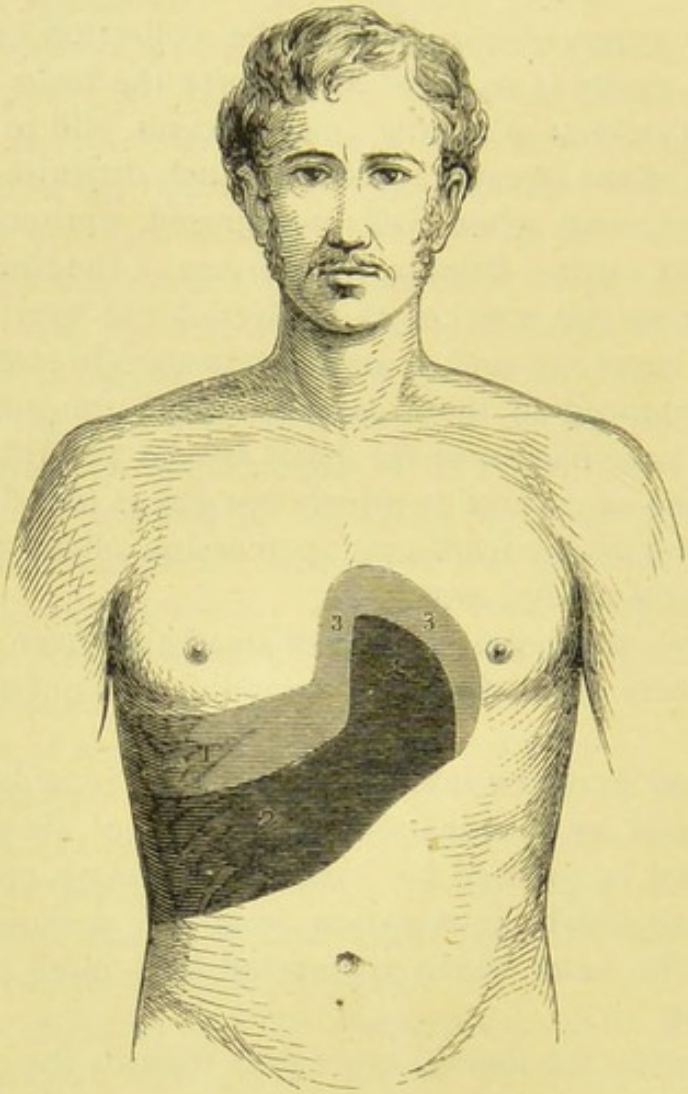



FIG. 22.—Cardiac and Hepatic Dulness.

1. Relative hepatic dulness.
2. Absolute hepatic dulness.
3. Relative cardiac dulness.
4. Absolute cardiac dulness.

(1.) *The vertical line* is thrown about one inch to the left of the sternum in order to avoid the aorta. In this direction the full note of the lung becomes impaired at about the lower edge of the third rib. This is, then, the limit of relative cardiac

dulness in this situation. Prolongation of dulness upwards above the third rib (if it be not caused by dislocation of the heart upwards, by pulmonary consolidation, or by the bulging of an aortic aneurism) arises from pericardial effusion. On the other hand, increase of dulness at the *lower* end of this parasternal line (if the heart be not dislocated downwards) indicates enlargement of the left lobe of the liver.

(2.) *The transverse line* at the level of the fourth rib. The *left border* of the heart is usually marked with sufficient accuracy by the position of the apex-beat, but in cases of uncertainty it may be determined by percussing from without inwards in the direction indicated, when it will be found that the margin of relative dulness lies a short distance (about one inch) to the left of the absolute cardiac dulness. Increase of the dulness to the left usually indicates hypertrophy and dilatation of the left ventricle. The *right margin* of relative dulness at the level of the fourth rib indicates the amount of dilatation of the right auricle. It is usually found about one inch to the right of the margin of the sternum. Increase of dulness in this direction, therefore, takes place when the right auricle is distended, and indicates obstruction to the circulation.

In measuring transversely the extent of the cardiac dulness, it is necessary to bear in mind the curve of the thorax.

The aortic dulness exists in the normal condition simply as a slight rounded projection from the upper part of the relative cardiac dulness. It rises as high as the upper margins of the second costal cartilages.

When dilatation of the aortic arch takes place, the relative dulness becomes better marked, and passes upwards and towards the right, impairing the note over the *manubrium sterni*. In aneurism of the aortic arch, the area of dulness increases correspondingly, and if the tumour approach near to the sternum, there is produced an area of absolute dulness.

When the aneurism comes to press firmly on the breast-bone, the dulness which this gives rise to is not absolutely limited to the site of the tumour, but extends up and down the sternum to a variable distance. This dulness is probably in some measure

caused by the obstruction to the vibration of the sternum which the firm pressure of the tumour presents. A similar alteration in the percussion note may be artificially produced by pressing heavily on the sternum with the hand while percussion is being made at a neighbouring point.

## CHAPTER XI.

## CIRCULATORY SYSTEM—(*continued*).

### AUSCULTATION OF THE HEART.

CARDIAC auscultation is almost invariably practised with the aid of a stethoscope. For ordinary purposes, a simple wooden instrument suffices, the cup of which accurately suits the ear of the auscultator; but in exceptional cases a binaural stethoscope may with advantage be employed.

On listening over the heart, two sounds are to be heard, separated by two pauses of unequal length. The first sound, which is considerably the more prolonged of the two, is followed by a short pause: to it succeeds the short second sound, and finally a long pause. At the apex of the heart the first sound is the louder of the two, the rhythm being there trochaic (– ⏑), while at the base the accent is thrown upon the second sound, as in the iambus (⏑ –).

Associated more or less intimately as the sounds are with the valves of the heart, it is necessary for the observer to have a clear conception of the position which these structures occupy in relation to the anterior thoracic wall.

*The Mitral Valve*, which is situated on a plane considerably posterior to those in which the other valves lie, may be said to correspond to the sternal end of the third left costal cartilage, projecting more or less upwards and downwards into the adjacent intercostal spaces.

*The Tricuspid Valve.*—The attached edge of this valve corresponds to a line drawn slantingly across the sternum from the third left intercostal space to the fifth right costal cartilage.

*The Aortic Valves* lie horizontally, opposite a line joining the

middle of the sternum and the inner end of the third left costal cartilage.

*The Pulmonary Valves* are also placed horizontally, slightly higher and more to the left than the aortic, corresponding to the upper border of the third left costal cartilage, or to the second left interspace.

It will thus be seen that, in relation to the chest-wall, these valves lie very close to one another, a superficial area of half an inch square including a portion of all four (Walshe). The sounds produced in connection with these valves are, however, best heard, not immediately over them, but at that point on the chest-wall at which the cavity into which the vibrating blood is flowing approaches nearest to the surface. Naturally this point where the sound is most intense varies in each case, and hence we have four areas for auscultation.

The *Mitral Area* is a circle about an inch in diameter, surrounding the apex-beat. This is the only point at which the left ventricle comes in contact with the chest-wall.

The *Tricuspid Area* embraces the lower part of the sternum, particularly the left border at the level of the fourth, fifth, and sixth cartilages.

The *Aortic Area.*—The aorta approaches nearest to the chest-wall at the second right costal cartilage, and consequently the aortic area is situated at this point.

The *Pulmonary Area* corresponds to the second left intercostal space (Von Dusch), or to the third left costal cartilage.

The causes of the heart sounds heard in these various areas may be briefly indicated as follows :—

The *first sound* (systolic), synchronous with the ventricular systole, is formed partly in connection with the left ventricle, and partly in connection with the right. In each case its mode of production is similar, and depends (in all probability) upon several factors. First, there is the muscle-sound arising during the ventricular systole, a sound precisely similar to that heard in any muscle during its contraction. In addition to this, there are the vibrations produced in the auriculo-ventricular valves, arising

from the sudden tension caused in them by the ventricular contraction, and the vibrations thereby communicated to the blood in their neighbourhood. Probably the vibrations which arise when the blood-stream is forced into the aorta and pulmonary artery become also audible, as Heynsius suggested, and go to form part of the first sound of the heart.[1]

The *second sound* (diastolic) has its origin in the vibrations produced in the semi-lunar valves at the orifices of each of these vessels during the diastole of the heart, by the blood being forced back upon these valves.

It is evident that we have thus to deal with four sounds during the course of the cardiac revolution, which arise entirely independently of one another.

Two systolic sounds originate at the mitral and tricuspid valves, and in the muscular fibre of the ventricles; and two diastolic sounds are caused by vibration of the semilunar valves at the aortic and pulmonary orifices.

Of these, the first two are synchronous, and are consequently heard as one sound, and, as the last two also take place almost simultaneously, only two sounds, a systolic and a diastolic, are audible over the cardiac area. The systolic sound marks accurately the commencement of the ventricular systole, and the diastolic expresses with equal precision the instant at which the diastole of the heart begins.

The changes in the cardiac sounds which disease produces are of two varieties—(1) Alterations in the normal heart sounds, and (2) murmurs, or adventitious and abnormal sounds.

### Modifications of the Normal Heart Sounds.

The normal sounds may be modified in disease as regards intensity, purity, quality, &c. It will suffice to consider such changes under the three following heads:—

[1] Sir R. Quain, however, in a recent paper (Proceedings of the Royal Society of London, July 1897) maintains that the cause of the first sound consists only in the impact of the blood against the columns of blood in the aorta and pulmonary artery which press upon the semilunar valves.

1. **Variations in Intensity** (intensified or enfeebled)—

(*a*) *Intensified.*

This takes place with regard to all the heart sounds under the influence of mental or bodily excitement, pyrexia, &c., or may be the result of improved conduction, either because the chest walls have become thin, or from condensation of the lung tissue conducting the vibrations more distinctly to the surface.

When the accentuation is limited to one sound, as heard in a particular area, it may result (1) from the better conduction through consolidated lung tissue (particularly in the case of the second sound at the base of the heart), (2) from hypertrophy of the walls of a particular cavity of the heart, or (3) it may arise from increased tension in the column of blood which presses back on the valves.

The first sound is accentuated with a dull, long character in cases of hypertrophy of the left ventricle, as typically seen in chronic interstitial nephritis. It is also intensified in mitral stenosis.

Intensification is most significant when found in connection with the second sound in the aortic or pulmonary areas. In order to determine that either of these sounds is accentuated, it is only necessary to compare it with the other, always bearing in mind, however, that in the normal condition the aortic sound is louder than the pulmonary.

*Accentuation of the aortic second sound* arises from increase of the arterial tension. It is met with, therefore, in cases of general atheroma, and of chronic interstitial nephritis, where the blood tension is raised. It is also seen in dilatation of the aorta, and in aortic aneurism if the valves are still competent.

*Accentuation of the pulmonary second sound* is found wherever there is increased pressure in the pulmonary artery. This leads to a more forceable closure of the pulmonary valves, and consequently to intensification of the sound. It is a sign that some hindrance exists to the circulation in the lungs, and this hindrance may be due either to disease in the lungs themselves, such as emphysema, or to obstruction to the passage of blood to the left ventricle, as occurs in cases of mitral disease, particularly in mitral stenosis.

(*b.*) *The sounds may be enfeebled* by reason of bad conduction through thick chest-walls, emphysematous lungs, pleural effusions, &c., or they may be audible with difficulty on account of loud sounds in the neighbouring lungs. Feebleness of the first sound is seen in all cases where the ventricular contractions are weak, in fatty heart and in other myocarditic conditions. It is a specially important sign in fevers, as it indicates the necessity for stimulation.

2. **Impurity of the Sounds.**—A heart sound is said to be impure when it wants the clearness and definition of normal sounds, or when it consists of, or is accompanied by, irregular vibrations. Such slight changes do not amount to a murmur, but in practice an impure sound is not readily distinguished from one accompanied by a soft murmur.

Impurity in the heart sounds may be caused by thickening of the valves, or by irregular tension and closure of the different cusps of the valve. In acute rheumatism, impurity of the first sound often precedes the appearance of a murmur.

3. **Reduplication of the Heart Sounds.**—Not uncommonly the heart sounds become doubled, each cardiac cycle giving rise to three, or even four, separate sounds. On careful examination, it will be found that one or other sound has become broken up into two. Reduplication frequently occurs in health, and is then intimately associated with respiration and the changes in the intra-thoracic pressure thereby produced. The first sound is reduplicated at the end of expiration and the beginning of inspiration, while the second sound is doubled at the end of inspiration and the beginning of expiration. Such reduplication has no peculiar significance, and only indicates that, in the case of the first sound, the auricular pressure on the right side of the heart is increased, and retards the closure of the tricuspid valve; and in the case of the second sound, that the pressure in the pulmonary artery is increased, and so the closure of the pulmonary valve is accelerated.

In disease, reduplication is more marked and constant. It is

frequently found in connection with the second sound in cases of mitral constriction and lung disease, from the abnormally high tension in the pulmonary circulation thereby produced. It may also arise in cases of aortic stenosis. Reduplication of the first sound may possibly be produced by irregular contraction of papillary muscles, or, in very rare cases, by asynchronous contraction of the ventricles, in addition to its origination from differences of blood tension, as in health.

In the following diagram these various modification of the sounds of the heart are graphically represented.

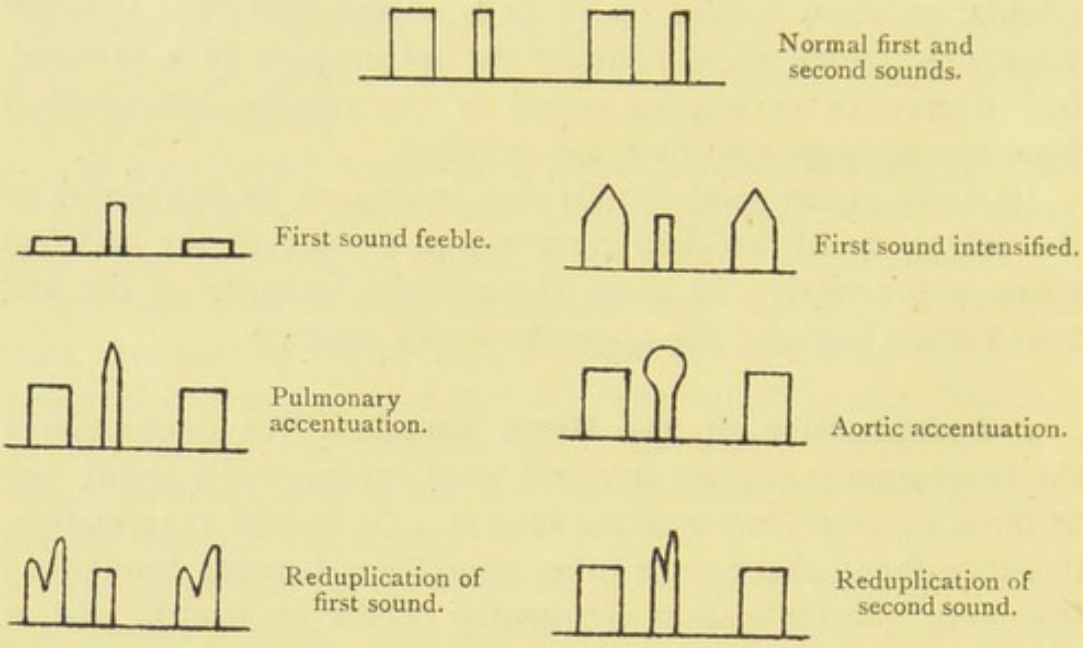



FIG. 23.—Diagram of Modifications of Heart Sounds (after Wyllie).

## MURMURS.

The murmurs which are met with in connection with the heart's action are divided into two groups—(1) *Endocardial*, or those which arise within the heart; and (2) *Exocardial*, or those originating in connection with the outer surface of that organ.

1. **Endocardial Murmurs.**—Murmurs, or abnormal sounds, differ from the natural heart sounds in being more prolonged,

and less sharply defined. Those which are of endocardial origin all arise from oscillations or vibrations in the blood stream as it passes through a narrow opening into a wider space beyond.

To go more particularly into the physical question of the origin of murmurs, we must put away from our minds the idea that such murmurs are ever caused by rubbing of the blood stream upon roughnesses or irregularities on the valves or orifices of the heart. Such a state of matters is physically impossible; for when a fluid streams through a tube, *the walls of which it wets* (as the blood does the endocardium), a thin layer of the fluid becomes attached to the inner wall of the tube by the force of cohesion, and consequently, seeing that the current itself never comes in contact with the tube-wall, no friction between the two is possible (Neumann, Helmholz).

When a fluid is passing along a tube of uniform calibre, no murmur arises, unless the rapidity of flow is very great. The blood current is never rapid enough to give rise to a murmur under these conditions. But when a constriction exists in the tube, and the fluid is thus forced to pass from a narrow into a wider portion, a murmur readily arises; and the greater the difference between the lumen at the two points, the more easily is the murmur produced; or, in other words, the less rapidity of current is required for its production. It is in this way, and under such conditions, that all such cardial murmurs arise. Whenever the blood stream passes with sufficient velocity through a narrow orifice into a wider space beyond, there will be such friction between the fluid particles as to give rise to sonorous vibrations in the fluid, as when, for example, a rent occurs in the aortic or mitral valves, or when the orifices they guard become narrowed by disease.

In the production of murmurs there enters, however, another factor which must not be entirely lost sight of—viz., the condition of the blood. So early as 1837, some observations were made by Williams, which seemed to show that the ease with which murmurs could be produced by driving fluids of different density through veins stood in inverse ratio to the density of the fluid, and this conclusion has been arrived at by many subse-

quent experimenters. It was found, in particular, that when water was added to blood a murmur was more easily produced than when blood alone was used, and this observation has a very important bearing upon anæmic murmurs, and particularly those which arise in veins, as will be hereafter shown. The governing factor is not, however, the density, but the viscosity of the fluid. The lower the viscosity the more readily does the blood pass into eddies and sonorous vibration.

*Endocardial Murmurs* are of two varieties—(1) Those of valvular origin, and (2) those of other than valvular origin.

Of these, the latter class is so rare that it is well invariably to endeavour to associate a murmur with a particular valve or orifice, and only in the event of failure in such an attempt, to consider the possibility of a non-valvular cause.

Having ascertained the presence of a murmur, there are certain points which should be carefully noted—(1) The rhythm of the murmur—*i.e.*, the particular period in the cardiac contraction with which it corresponds; (2) the point of maximum intensity and the direction of propagation; (3) the condition of the normal heart sound at the valve or orifice at which the murmur is supposed to originate; (4) the character of the murmur; (5) the intensity of the murmur.

(1.) *Rhythm.*—To ascertain the rhythm of a murmur it is necessary to lay a finger upon the apex-beat or upon the carotid artery while we auscultate. This gives the time of the ventricular systole, and enables us to say which is the first and which the second sound, and consequently the rhythm of the murmur can be readily ascertained. If, however, the cardiac pulsations exceed 90 per minute, it may be impossible thus to time the murmur, and in such cases we must wait till rest and appropriate medication have reduced the rapidity of the action of the heart.

Murmurs which arise during the ventricular systole are termed *systolic;* those which occur during the ventricular diastole are known as *diastolic;* and, in that the auricular systole precedes

the ventricular, murmurs produced during the contraction of the auricles are known as *presystolic.*

(2.) *Point of Maximum Intensity and Direction of Propagation.*—It has been already stated that the normal heart-sounds are heard with most distinctness in various areas according to the value or orifice at which they arise. These sounds are conducted in the direction of the blood current, and are best heard where the cavity into which the current flows approaches nearest to the surface of the body. The same holds good for murmurs, every endocardial murmur of valvular origin having its points of maximum distinctness in one of these four areas, and being of mitral, tricuspid, aortic, or pulmonary origin, according as it is best heard in the mitral, tricuspid, aortic, or pulmonary area. Two exceptions to this rule, however, exist —viz., (1) a mitral systolic murmur, which is sometimes best heard an inch to the left of the pulmonary area, and (2) an aortic diastolic murmur, which is occasionally most intense at the xiphoid cartilage.

Having satisfied ourselves as to the rhythm, and the point of greatest intensity (and consequently the seat of origin) of the murmur, it is a matter of simple reasoning to discover its mode of production. Thus, for example, a systolic mitral murmur can only be one of regurgitation through incompetence of the valve. A presystolic mitral murmur, on the other hand, must result from stenosis of the mitral orifice, since it occurs at the instant when the blood is being propelled by the auricular systole through the mitral orifice into the ventricle. We shall consider the causation of each particular murmur further on.

The direction of the conduction of murmurs is of use in indicating their origin. Stated generally, it may be said that systolic mitral murmurs are conducted towards the left axilla, and to the angle of the left scapula, while presystolic and diastolic mitral murmurs are localised at the apex and not propagated in any special direction. Tricuspid murmurs are heard over an area corresponding to the right ventricle. Aortic systolic murmurs are propagated up and down the sternum and into the arteries. Aortic diastolic murmurs are propagated down the

sternum and towards the apex. Finally, pulmonary systolic murmurs are usually not audible outside of the pulmonary area; while pulmonary diastolic murmurs may be heard over the right ventricle.

(3.) *The Condition of the Normal Sound at the Orifice at which the Murmur originates.*—The presence of a normal sound, more or less obscured by the accompanying murmur, indicates that the valve is not entirely destroyed. The method of auscultation suggested by Gendrin is of value for the purpose of ascertaining this. He recommends the ear to be slightly raised from the stethoscope, the instrument remaining unmoved, when the sound will become more and the murmur less audible. The real value of the presence of a cardiac sound as an indication of the state of the valve is very questionable. In the case of aortic disease, the auscultation of the arteries gives much more reliable results.

(4.) *The character of the murmur* (soft, blowing, rasping, whistling, &c.) should be noted. As a general rule *direct* murmurs (those which arise in the blood current as it is flowing in its normal direction) are rough; whereas *indirect* murmurs (those which arise from regurgitation) are soft.

(5.) *The intensity of the murmur.*—This depends to some extent on the nature of the lesion, but very importantly on the strength of the cardiac contractions. Thus the loudness of a murmur is an index of the cardiac vigour. It is not unusual to see a murmur disappear when the heart is failing, and reappear when, by appropriate treatment, the cardiac compensation has been restored. Thus, in many cases, the intensity of a murmur is a good sign. This is not the case, however, as regards aortic regurgitation.

Having determined the rhythm and seat of the murmur, it is, as I have already said, no very difficult matter to infer the manner of its causation. This is done by simply bringing to mind what is happening at the valve in question during the particular period at which the murmur is heard. In order to make this plain, we will now consider very briefly the various murmurs met with in connection with each valve and orifice.

### (*a.*) *Mitral Murmurs.*

Mitral murmurs are systolic, diastolic, or presystolic in rhythm, according as they occur during the ventricular systole, the diastole, or immediately before the ventricular systole—*i.e.* during the auricular systole.

*Mitral systolic murmurs.*—These murmurs, originating at the mitral valve during the ventricular systole, indicate that from some cause the valve does not completely cover the orifice, but allows a part of the blood contained in the ventricle to be forced back into the auricle. As a result of this, the blood-pressure in the auricle rises and its cavity becomes dilated, and when this stretching has reached a certain point, the backward pressure is transferred to the pulmonary veins, to the capillaries of the lung, and thence to the pulmonary artery. The increased resistance in that vessel causes the right ventricle to dilate, and subsequently to hypertrophy. The necessary result of this hypertrophy of the right ventricle is that the second sound in the pulmonary area becomes accentuated, for the blood-pressure in the pulmonary artery is raised, and consequently the rebound of the blood column upon the pulmonary valves after the ventricular systole is rendered more forcible. There are thus three chief physical signs to be looked for in cases of mitral incompetence—(1) the systolic murmur, (2) the hypertrophy or dilatation of the right ventricle, and (3) accentuation of the pulmonary second sound. To these there came to be added, subsequently, hypertrophy of the left ventricle, dilatation and hypertrophy of the right auricle, irregularities of rhythm, and other signs.

This incompetence of the mitral valve is the result of one of two processes; either the valve itself has become altered in shape, or the orifice has increased in size, so that the valve which formerly sufficed to occlude it is not now sufficiently large. The former condition arises as a result of endocarditis, in connection with rheumatism, syphilis, scarlatina, &c. The valves become thickened and shrivelled up, and so the incompetence originates.

The second condition (viz., increase in the size of the mitral orifice) is found in all diseases in which relaxation of the cardiac muscle takes place to a marked extent, as in typhus, typhoid, relapsing and rheumatic fevers; in scarlatina, measles, erysipelas, small-pox; and not least importantly, in chorea and in various forms of anæmia, particularly in chlorosis. In all these conditions, the myocardium is so softened and stretched as to allow the mitral orifice to become too large to be covered by the valve. Regurgitation of blood takes place into the auricle; and in passing from a narrow orifice into a cavity, sonorous vibrations arise in the fluid, and in these cases the formation of a murmur is no doubt aided by the watery condition of the blood, in the manner already described.

Mitral systolic murmurs are usually heard with greatest distinctness in the mitral area, and are propagated towards the left axilla and the angle of the left scapula. Occasionally, however, they are most audible a little to the outside of the pulmonary area, especially in the case of anæmic murmurs; and this may arise (as Naunyn suggested) because the sonorous vibrations in the auricle are conducted into the auricular appendix, and become most audible at that point on the chest-wall where the appendix approaches nearest to the surface—*i.e.* about an inch and a half to the left of the pulmonary area.

*Mitral presystolic and diastolic murmurs* arise from the same cause—viz., stenosis (narrowing) of the mitral orifice. Immediately after the ventricles of the heart have contracted they relax and begin to refill with blood, and during the period of time represented by the second, or diastolic sound, and by the long pause, this process of filling goes on. At first the blood follows the retreating walls of the ventricles, propelled partly by gravity and partly by the suction of the dilating ventricle, and so flows slowly through the patent orifices (mitral and tricuspid) into the respective ventricular cavities. But towards the end of the long pause, the auricular contraction takes place, and the remainder of the blood is thus more powerfully forced into the ventricles. In ordinary circumstances these actions take place noiselessly; but when stenosis of the mitral orifices arises, as a result of

endocarditis, the narrowing may be sufficient to throw the fluid into sonorous vibrations. It depends on the rapidity of flow, and the narrowness of the orifice in relation to the size of the ventricular cavity, whether or not a murmur will occur,—and if so, whether it will be diastolic or presystolic in rhythm, or, in other words, whether it will be produced when the blood is flowing into the ventricle immediately after the ventricular systole,

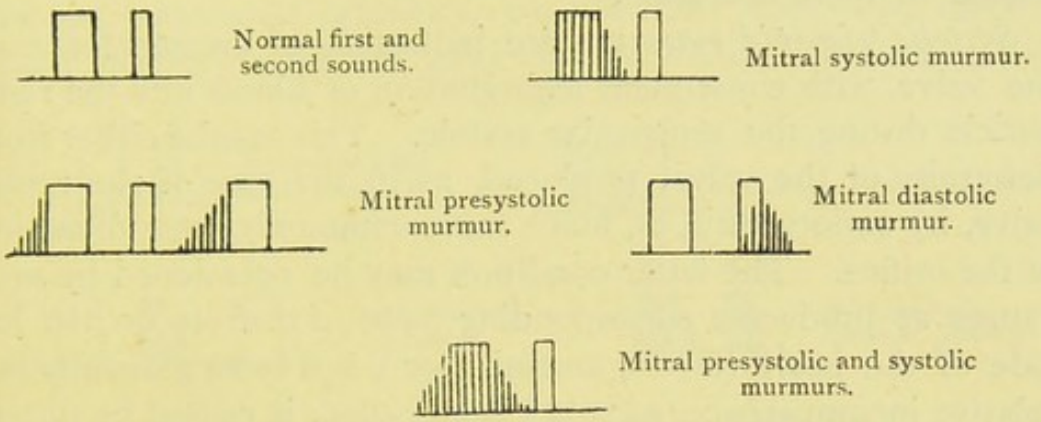



FIG. 24.—Diagram of the various Mitral Murmurs (after Wyllie).

or later on, during the auricular systole. As a rule, the rapidity is greatest during the auricular contraction, and therefore the murmur is presystolic. But, if the auricle be very weak and dilated, the greatest rapidity may be at that time when the suction of the ventricle is most powerful, and, as this occurs in the earlier part of the ventricular diastole, the murmur produced is diastolic.

These murmurs sometimes co-exist, and may either run into one another, and so fill up the whole time occupied by the ventricular diastole, or they may be separated by a very short interval of silence. The diastolic portion is usually soft, whilst presystolic (or auricular-systolic) murmurs are almost invariably rough in character.

Mitral stenosis is followed by much the same physical signs as have been mentioned in connection with mitral incompetence. The right side of the heart hypertrophies and dilates, and the left auricle undergoes similar changes. There is a peculiar

thumping character about the first sound, accentuation and often reduplication of the second pulmonary sound, and usually a well-marked presystolic thrill at the apex. The rhythm of the cardiac pulsations is almost always irregular.

### (*b.*) *Tricuspid Murmurs.*

Tricuspid murmurs resemble those at the mitral valve in regard to their causation.

*Systolic tricuspid murmurs* are indicative of incompetence of the valve, with consequent regurgitation of blood into the right auricle during the ventricular systole. This results either from deformity of the valve, produced, as in the case of the mitral valve, by endocarditis, or, much more commonly, from dilatation of the orifice. The latter condition may be occasioned by such causes as produce a corresponding state of matters on the left side of the heart (fevers, anæmia, &c.), but more generally this relative incompetence, as it has been called, is caused by distension of the right auricle and ventricle, the result of obstruction to the circulation through the lungs, produced most distinctly in cases of stenosis or incompetence of the mitral valve. Tricuspid regurgitation should not be diagnosed, unless the signs in connection with the jugular veins, formerly described, are present.

*Presystolic tricuspid murmurs* are very rarely met with, and never without other valvular complications. They are the result of stenosis of the tricuspid orifice, and the mechanism of their production is similar to that which produces the corresponding mitral murmur.

### (*c.*) *Aortic Murmurs.*

Aortic murmurs are of two varieties—systolic and diastolic. These often co-exist.

*Systolic aortic murmurs* are those produced at the aortic orifice as the blood is propelled into the aorta by the contraction of the left ventricle. Such a murmur arises when the orifice is con-

tracted or roughened as a result of endocarditis. The murmur is usually loud and sawing, occasionally musical, and whilst it is loudest in the aortic area, it can most frequently be heard over the whole front of the heart.

Aortic etenosis leads first to dilatation and second to hypertrophy of the left ventricle, owing to the extra resistance which the ventricle has to overcome in forcing the blood through the aortic orifice. This hypertrophy, and the small hard pulse which will be mentioned presently, are the most important signs to which aortic obstruction gives rise, in addition to the systolic

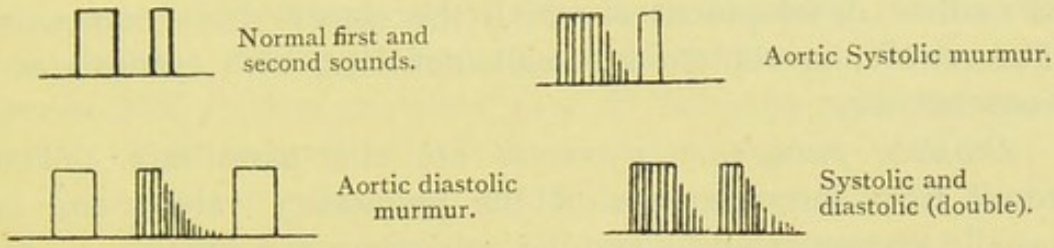



FIG. 25.—Diagram of the Aortic Murmurs (after Wyllie).

murmur, the character and localisation of which have just been described.

*Diastolic aortic murmurs* are the result of incompetence of the aortic valves, the blood regurgitating from the aorta into the left ventricle during the ventricular diastole. The position of maximum intensity of this murmur varies very much. In many cases it is best heard in the aortic area; not uncommonly it is loudest at the ensiform cartilage; rarely, the apex-beat is the situation at which it is most distinct.

Aortic incompetence leads to even more marked hypertrophy of the left ventricle than obstruction does. The very peculiar pulse which is found in such cases (Corrigan's pulse) is of considerable importance in diagnosis, and will be described hereafter.

Most usually these two murmurs are heard together, the so-called double aortic murmur, for the valves are rarely incompetent without presenting some obstruction to the flow of blood over them into the aorta.

### (*d.*) *Pulmonary Murmurs.*

Among pulmonary murmurs we do not include those hæmic murmurs which arise at the mitral valve, and have their seat of greatest intensity an inch or more to the left of the pulmonary area.

True pulmonary murmurs are of very rare occurrence. They are systolic and diastolic in rhythm.

*Systolic pulmonary murmurs* are almost invariably due to congenital constriction of the pulmonary artery. Such cases are rare, and differ much from one another according to the period of cardiac development at which the constriction commenced. The ventricular septum is usually deficient, with cyanosis as a consequence.

*Diastolic pulmonary murmurs* are still more rare. They result from incompetence of the pulmonary valves, and are usually accompanied by systolic pulmonary murmurs.

*Endocardial murmurs of non-valvular origin* may result from—

(1.) Congenital deficiency of some part of the septum which divides the two sides of the heart.

(2.) Changes in the viscosity of the blood in anæmia, chlorosis, &c., may allow of murmurs forming in circumstances under which no such sonorous vibrations would arise in blood of normal composition. It has been already pointed out that many of the hæmic murmurs are mitral in their origin, resulting from incompetence caused by relaxation of the cardiac muscle, and a similar relaxation and relative incompetence occurs at the tricuspid valve, in connection with the right ventricle. A certain porportion of these murmurs may, however, arise in the blood-stream, where no incompetence exists. Such murmurs are soft, invariably systolic, and are usually heard most distinctly over the base of the heart, being formed at the pulmonary or at the aortic orifice.

2. **Exocardial Murmurs.**—These murmurs are caused by the friction of the two pericardial surfaces on one another, when the surfaces have become roughened as a result of pericarditis.

Such friction murmurs are, for the most part, readily distinguished from endocardial murmurs. They are rough and grating, never blowing. They are localised, and are not propagated in the direction of the blood current; and, as they usually arise first towards the middle of the heart, the point of greatest intensity does not generally coincide with any one of the cardiac areas. Further, the rhythm of exocardial murmurs is irregular. They are not confined to any particular phase of the cardiac action, are neither permanently systolic nor diastolic, but vary from minute to minute.

Exocardial murmurs are also sometimes occasioned by friction of two roughened surfaces of the pleura overlying the heart on one another, during the movements of the heart. Such *pleuro-pericardial friction* murmurs vary in intensity with the movements of respiration.

Pericardial friction can hardly be mistaken for the friction of pleurisy, the latter being synchronous with respiration.

# CHAPTER XII.

## CIRCULATORY SYSTEM—(*continued*).

### THE EXAMINATION OF THE ARTERIES, CAPILLARIES AND VEINS.

#### *Arteries.*

THE physical examination of the arteries may be conducted by means of inspection, palpation, percussion, and auscultation.

**Inspection.**—In health, the pulsation of the arteries of the body is but little visible, except under the influence of mental emotion or bodily strain. As the result of disease, however, pulsation may become visible in all the superficial arteries of the body, particularly in the carotid, temporal, and radial vessels. All disturbances of cardiac innervation, such as arise in Graves' Disease, and all feverish conditions, are liable to produce such excited action of the heart as will occasion this visible pulsation. Still more marked is the pulsation when the left ventricle is hypertrophied, and, above all, when the aortic valves have been rendered incompetent. Dilated tortuous, and visibly pulsating temporal or radial arteries are usually found to have undergone atheromatous changes; and finally, inspection may show us the localised pulsation of an aneurism.

**Palpation** of the arterial system is chiefly made use of in connection with the radial artery.

*The radial pulse* is, in health, equal on the two sides; but abnormal distribution, compression, or other pathological con-

dition may so act as to make one pulse weaker than the other. So also, the pulse-wave propagated from the heart outwards towards the periphery may not arrive at the two wrists synchronously. This condition may occur where there is simple or aneurismal dilatation of the aortic arch, and is particularly noticeable if the aneurism be situate between the innominate and the left subclavian. It is to be observed that the interval of time which occurs between the cardiac systole, and the arrival of the blood-wave at the wrist may sometimes be considerably longer than usual. Such delay arises either from stenosis of the aortic orifice rendering the systole slow and difficult, or from aortic incompetence where (as Tripier has pointed out with great probability) the onward wave meets with, and is delayed by, the regurgitating blood.

It may probably conduce to clearness if the conditions of the pulse are considered under four headings—viz., (1) frequency, (2) rhythm, (3) character, (4) condition of the arterial walls.

1. *Frequency of the pulse*, which in the male adult averages about seventy beats per minute (slightly higher in women), varies in healthy individuals according to the age, according to the position of the patient, being faster in standing than in lying, and according to the time of day and the external temperature, and may be greatly influenced by mental emotions, and by the administration of certain drugs. In disease the pulse is sometimes abnormally slow; as for example, in jaundice, in fatty degeneration of the heart, and in some affections of the brain. More frequently, however, the pulse rate is increased in rapidity. The rapid pulse of fever, of collapse, and of various cardiac neuroses is well known. Very generally the pulse is rapid in diseases of the valves of the heart, particularly the mitral.

2. *Rhythm.*—The radial pulsations, which are normally separated by regular intervals of time, and so are rhythmical, may be altered in this relation to each other in a great variety of ways, the normal rhythm being sometimes changed into total irregularity, while at other times the beats, although following

each other in an abnormal manner, still possess a certain rhythm. Amongst the latter may be mentioned the (1) *pulsus bigeminus*, in which each two beats form a group, separated from the two which precede and the two which succeed by longer pauses than the interval which separates each pair; (2) the *pulsus paradoxus*, that variety of pulse, so carefully described by Kussmaul, where with each inspiration the pulse wave becomes smaller, or is completely lost. When it is present in all the arteries of the body, it may be due to one of two causes—either to fibrous adhesions between the aorta and the sternum, or other obstruction which during inspiration prevents the free passage of the blood into the aorta; or it may result from any obstruction to the entrance of air into the lungs, which during inspiration lessens the pressure within the thorax. When the *pulsus paradoxus* occurs only in one radial artery, it is probably due, as Weil has pointed out, to inflammatory adhesion between the pleura and the subclavian artery. In the *pulsus alternans* there is a regular alternation between a small and a large pulsation. When, after a series of regular pulsations, one or more beats are omitted, the pulse is said to be *intermittent*. The intermissions, due either to momentary cessation of the heart's action or to the blood-wave in question being too feeble to reach the wrist, may be regular or irregular, and often occur independently of heart disease. Frequently, however, the intermittent pulse is associated with some affection of the myocardium. Very irregular pulsations, in which no rhythm of any kind can be detected, are commonly (although by no means always) due to affections of the mitral valve, generally to mitral constriction, of which affection an extremely irregular pulse, even in the early stages, is a marked symptom, and one to which considerable diagnostic importance may attach.

3. *The character of the pulse* varies in a great number of ways, giving rise, especially in the works of the older writers, to a very extensive nomenclature. It will be sufficient for ordinary purposes to notice the following points:—

(*a.*) The *expansion* of the pulse. A pulse which reaches its full expansion quickly, and as rapidly collapses again, giving to

the finger the impression of a very quick stroke, is denominated the *pulsus celer*, and this celerity is, as Corrigan first pointed out, most distinct where there is aortic incompetence (hence called Corrigan's pulse). The opposite condition, the *pulsus tardus*, is distinguished by the slow manner in which the artery fills and empties, and this sluggishness may be due to slowness in the contractions of the heart, to a hindrance in the capillary and venous circulation, or to loss of elasticity in the arterial wall itself. It is perhaps most frequently met with as a result of arterial sclerosis.

(*b.*) The *tension* of the pulse, or, in other words, the blood-pressure on the inner surface of the artery, may be approximately estimated by the degree of pressure of the finger required to obliterate the pulse.[1] When the tension is high (as in hypertrophy of the left ventricle, lead colic, peritonitis, &c.), we speak of a *hard*, *tense*, or *incompressible* pulse, and under the reversed circumstances (as in mitral disease), of a soft and compressible pulse. Above all things, however, it must be borne in mind that a simulated impression of tension or hardness may be given to the finger by a rigid condition of the arterial wall, a subject to be presently referred to.

(*c.*) The *volume* of the pulse. A full pulse may be produced by one or more of several factors, powerful ventricular contraction, loss of elasticity of the arterial wall, interference with the blood flow from the arteries into the capillaries, and the actual quantity of blood in the vessels. The opposite conditions may give rise to an empty pulse. The volume of the pulse is best appreciated by noting the amount of blood remaining in the artery between the pulsations. A pulse of considerable volume suggests an increased tonus in the arterioles, and the commencement of chronic interstitial nephritis.

(*d.*) *Condition of the Arterial Wall.*—The radial artery is, normally, soft and yet elastic to the finger. When the arterial coats have become atheromatous, however, the artery feels hard and rigid. This can be best appreciated by pressing on the

[1] The tension may be more accurately estimated by means of the sphygmomanometer of von Basch, which will be hereafter described.

vessel with the finger sufficiently to empty it, and then rolling it under the finger on the bone below.

All these varieties of pulse may be further studied with aid of the sphygmograph.

**Percussion of the Arteries** is almost entirely limited to cases of thoracic aneurism, of which mention has been already made.

**Auscultation of the Arteries.**

1. *In Health.*—As in cardiac auscultation, so also in auscultation of the arteries, we have to distinguish two phenomena—sounds and murmurs. In health, if the stethoscope be placed over the carotid artery as lightly as possible, two sounds are usually to be heard, corresponding respectively to the expansion and contraction of the artery. Of these the latter is simply the second aortic sound conducted into the carotid, and it seems most probable (Weil, Heynsius) that the sound coinciding with the arterial expansion ought also to be regarded as the conducted aortic systolic sound (although it may in part originate in vibrations of the arterial wall). These two sounds can also generally be heard in the subclavian, and occasionally the first can be detected in the abdominal aorta and in the brachial and femoral arteries; but in the more peripheral vessels no auscultatory phenomenon is present in health. If pressure be made with the stethoscope upon an artery, such as the brachial just above the elbow, where normally no sound can be heard, the narrowing of the lumen of the vessel, thereby occasioned, gives rise to vibrations in the blood stream, and to an audible murmur coincident with the arterial expansion. If the pressure be increased, this murmur passes into a sharp sound.

2. *In Disease.*—Sounds or murmurs may be heard in the arteries under three pathological conditions—

(*a.*) *Murmurs conducted from the Heart.*—Aortic murmurs, especially systolic, are propagated into the arteries, and can readily be heard in the carotids.

(*b.*) *Sounds and Murmurs originating in the Arteries in consequence of general Circulatory Disease.*—In aortic incompetence

a sound coinciding with the arterial expansion may be heard, through a lightly-applied stethoscope, in all the accessible arteries of the body, due almost certainly to the rapid transition from extreme relaxation to extreme tension which the arterial coats then undergo. A double sound over the femoral artery is also sometimes to be heard in such cases.

A double murmur may be produced in the femoral artery in cases of aortic incompetence by pressure with the stethoscope, the one murmur being caused by the pulse wave, the other by the returning backward wave, which in such cases flows towards the heart during the arterial collapse. This double murmur may also occasionally be heard in cases of anæmia, typhoid fever, &c.

(*c.*) *Murmurs originating in the Arteries in consequence of Local Changes.*—Such murmurs are to be heard over aneurisms of the peripheral arteries, and over vascular tumours, but more important are the subclavian murmurs. While occasionally occurring in healthy persons, murmurs over the subclavian arteries are much more frequently heard in cases of phthisis, due probably to adhesions between the pleura and the vascular walls, and hence much influenced by the respiratory movements.

The encephalic murmur which Fisher discovered in children has, so far as our present knowledge goes, no diagnostic significance.

## Capillaries.

The state of the capillary vessels need not be specially noticed here, seeing that the more important points have been elsewhere discussed.

## Veins.

Knowledge concerning the condition of the veins may be obtained by inspection and by auscultation.

**Inspection.**—By inspecting the veins we ascertain, firstly, their state as to fulness; and secondly, whether the blood contained in them undulates or pulsates.

*Overfilling* of the veins results either from local obstruction, when the vein becomes tense on the distal side, and such of the collateral branches as are not compressed enlarge so as to carry on the circulation—or from interference with the venous circulation generally. Examples of the variety of engorgement arising from local obstruction are to be found in cases of thrombosis of any of the larger venous trunks, or where the pressure of an aneurism or other mediastinal tumour gives rise to overfilling of the veins of the arm. The distension of the cervical veins which arises where the general circulation is interfered with, has already been described on page 99.

*Undulation of the Veins of the Neck.*—The pulsations in the cervical veins, which correspond to the movements of the heart, have been already described on page 99. It only remains to mention the undulation which the respiratory movement sometimes produces in the jugular veins. When the cervical veins are overfilled, as a result of pulmonary emphysema, of mitral stenosis or other cause, each inspiration diminishes the venous distension, while each expiration increases it, and so the veins show a constant undulation, synchronous with respiration.

**Auscultation.**—Although in cases of tricuspid incompetence systolic sounds are occasionally to be heard over the jugular and femoral veins, the only auscultatory sign which here demands attention is the humming murmur, the so-called *bruit de diable* which is very frequently to be heard in chlorotic females over the bulb or dilatation of the internal jugular vein, and more rarely over the large intrathoracic venous trunks, the superior vena cava, and the innominatic veins. Venous murmurs in the former are best heard at the right border of the sternum, from the first right intercostal space to the third costal cartilage. The murmur in the right innominate vein is usually loudest at the sternal end of the first right costal cartilage, and that in the left over the manubrium sterni. Occasionally a venous hum is to be heard in dilated thyroid veins, and in the subclavians, axillary, brachial, and femoral veins. In venous auscultation, it must be borne in mind that

the slightest unnecessary pressure with the stethoscope may develop an artificial murmur.

The *bruit de diable*, as met with in the jugular vein (generally loudest on the right side), is usually of a continuous soft humming character, and is well marked in cases of anæmia, and particularly of chlorosis.

These venous murmurs appear to depend for their production upon three factors—1st, upon the rapidity of the blood current; 2nd, upon the change in the calibre of the vein at any particular point (such as occurs in a marked manner at the jugular bulb); and 3rd, upon alteration in the quality of the blood, whether this consists in an actual or only a relative increase of the watery elements.

Usually the jugular humming murmurs are continuous, but they very often vary in intensity, and occasionally are actually intermittent. They are influenced in the following ways:—

1. *Changes in the Posture of the Patient.*—When the head is turned to the opposite side, the murmur becomes much intensified, owing to the compression of the vein by the muscles and fascia. Even when no murmur exists when the head is held straight, a faint bruit may be developed when the head is rotated, especially if firm pressure be made with the stethoscope in addition.

Owing to the acceleration of the blood-flow in the veins the murmur is louder when the patient sits or stands than in the recumbent posture.

2. *The Movements of Respiration.*—Sometimes the venous murmur in the jugular is only audible during deep inspiration, and if it be continuous it is almost invariably intensified by that action, in both cases, for this reason—viz., that during inspiration the flow of blood in the vein is accelerated. The same usually holds good with regard to murmurs in the femoral vein, although in rare instances the reverse obtains, and we meet with the remarkable phenomenon of a femoral murmur which is expiratory in rhythm (Friedreich), this probably resulting from the increased abdominal pressure which the descent of the diaphragm occasions, and which retards the blood current in the femoral vein.

3. *The Movements of the Heart.*—The anæmic murmur in the jugular vein is sometimes diastolic in rhythm, as was first pointed out by Chauveau, who ascribed it to the increased blood current in the vein which is the result of the diminution of pressure in the superior vena cava produced during diastole, and which stands closely related to the negative diastolic pressure in the ventricle.

Similar venous murmurs may be heard in the Torcular Herophili by listening over the occiput, and in the orbit by resting the stethoscope lightly over the closed eye.

# CHAPTER XIII.

## CIRCULATORY SYSTEM—(*continued*).

### GRAPHIC CLINICAL METHODS.

When Chauveau and Marey first introduced the sphygmograph and cardiograph to the notice of the profession, it was hoped that a new and more accurate examination of the heart and circulatory system would soon replace the former methods. This hope has not been realised. There is, indeed, little difficulty in obtaining tracings of the pulse wave and heart beat, and these tracings, moreover, are found to vary greatly in different diseases; but the true meaning of these differences is as yet by no means thoroughly understood. The reason for this lies partly in the fact that the meaning of the normal pulse and heart curve has not yet been explained, in all its details, in a fully satisfactory manner. Still, even now, certain trustworthy facts can be obtained by the use of the recording instruments referred to, and the number of these facts will necessarily increase as the characteristics of the normal pulse wave and heart beat, and the modifications which they may undergo in health, become more fully understood. Moreover, the permanence of the records which may be obtained by the use of such instruments, their value in illustrating the history of individual cases, together with the fact that these instruments give results which are more purely objective than those obtainable by other methods, amply justify a somewhat full description of the manner of using the sphygmograph and cardiograph, together with some account of the results obtainable by their help.

**Sphygmograph.**—Although many varieties of sphygmograph have been devised and employed, the original instrument of

Marey, in its latest modification, gives the most satisfactory results. The principle of its construction is to be found in all text-books of physiology, and need not therefore be dealt with here. We may therefore confine ourselves to a consideration of the typical pulse curve, and the modifications which it undergoes in health and in disease.

*The typical healthy curve*, of which the accompanying tracing (fig. 26) is an example, is usually divided into an ascending and descending portion, either or both of which may present certain secondary undulations. In its most typical form (as in fig. 26) the ascending line (*a* to *b*) rises abruptly at first, and afterwards more slowly, till it reaches its highest point. Then descending more obliquely, it usually presents a more or less well-defined notch or indenture (*c*) before it reaches the principal notch or valley (*d*). This latter notch is best known as the dicrotic notch, and is of great importance, corresponding as it does exactly to the closure of the aortic valves. After the dicrotic notch, the curve describes a slight elevation before descending to its lowest level, in the course of which descent a low wave-like eminence (*f*) is not unfrequently to be discovered. Since the point (*a*) corresponds to the opening of the aortic valves, and the point (*d*) to their closure, the artery is, during the time represented by the interval between these two lines, in free communication with the interior of the ventricle, while, during the time of the rest of the curve, the artery is cut off from the heart.[1] The point *d*, therefore, forms the most

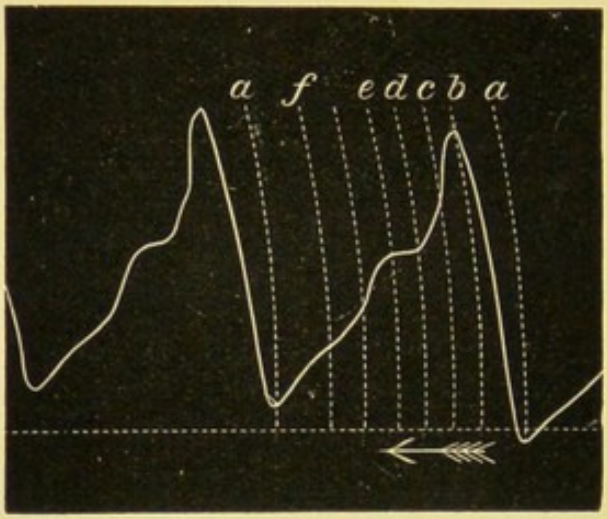



FIG. 26.—Normal Pulse Curve.[1]

[1] For this curve and those which follow, the author is indebted to the kindness of the late Professor C. S. Roy, of Cambridge University.

natural division of the pulse wave into its more fundamental parts, the causes which influence its form during the first half being essentially different from those which modify the second half. Keeping this fact in view, and premising that it is almost always easy to find in any curve, of whatever form, the point which corresponds to *d*, we may now proceed to describe the modifications of the pulse wave which are to be met with.

And first, with regard to changes in the first half of the curve.

*Anacrotic Pulse.*—The typical pulse curve, of which I have given an example above, is sometimes called dicrotic, owing to the fact that it presents a fairly well-marked notch in its descending part, although it is better to restrict the term dicrotism of the pulse wave to cases in which that notch is abnormally well marked. In contradistinction to the dicrotic pulse, it is the custom to call those pulse waves in which a more or less well-marked notch occurs in the ascending line as anacrotic. The tracing (fig. 27) annexed shows a fairly typical example of the anacrotic pulse wave. It can be seen that it differs from the normal pulse wave only in the part which lies between the lines *a* and *d*; in other words, in that part of the pulse wave which corresponds to the time when the aortic valve is open. We would, therefore, expect, *à priori*, that this change in the form of that part of the pulse wave must be due to some difference in the relation between the quantity of blood thrown out of the left ventricle and the elastic resistance offered by the aorta and larger arteries. Let us suppose that the arteries are relatively lax, and that the quantity of blood thrown out of the ventricle is not above normal, then it is not difficult to

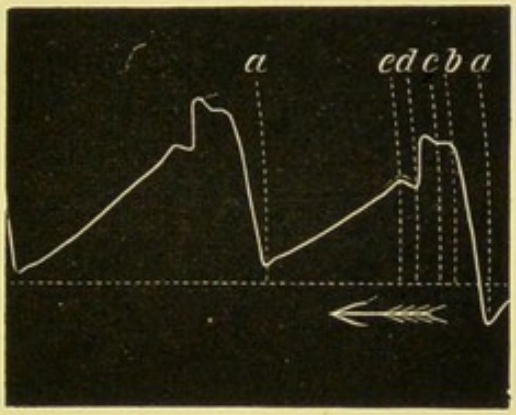



FIG. 27.—Anacrotic Pulse Wave.

[1] The whole pulse wave is delayed in its transmission from the commencement of the aorta to the radial artery, but the delay of the different parts of the curve is usually tolerably equal, so that the relative distance between the up-stroke and the dicrotic notch remains the same.

understand that the ventricle will more readily and more rapidly empty itself than when the vessels are relatively rigid. The result of this is that the point of the pulse wave, where the highest pressure exists, and which corresponds to the highest point of the pulse curve, will occur nearer its commencement than would otherwise be the case. Let us, on the other hand, suppose an extreme case, in which the arteries are very rigid, as in well-marked atheroma or calcification of the larger vessels, these latter, as the contents of the ventricle are forced into them during systole, do not expand to receive the contents of the ventricle, but act more like rigid tubes; the result of which is, that during the cardiac systole the inflow into the vessels, which is always greater than the outflow at that period, produces a continuous rise in arterial pressure during the whole time of systole. The point of highest pressure of pulse wave, or, in other words, the highest part of the pulse tracing, is thus thrown toward the end of the ventricular part of the pulse curve, or, in other words, closer to the dicrotic notch, *d*, which marks the end of the systole. Thus, in cases where the larger arteries are not fitted to contain the quantity of blood in the ventricle, the latter forces the blood at first against a comparatively weak resistance, which, however, goes on increasing very rapidly as the large arteries become gradually more and more tensely filled; and the pressure within these latter necessarily rises from the commencement to the end of the cardiac systole. This, then, is the reason why in such circumstances the highest part of the pulse curve is nearest our line *d*, or the dicrotic notch, which corresponds to the end of the ventricular systole.

Nothing has, as yet, been said of the indenture (*c*) which precedes the dicrotic notch, and which, on that account, is usually described as the pre-dicrotic notch. The exact significance of this notch is still by no means fully understood. It would seem that its appearance results from the fact that, at the moment when the aortic valves are forced open, the column of blood contained in the aortic arch and larger branches receives a sudden impulse towards the periphery, and the inertia of this column of blood, thus set in comparatively rapid motion, produces a

negative wave at the commencement of the aorta, which is propagated towards the periphery in the same manner as the positive wave which preceded it.

Reference has been made to the causes which may, theoretically, produce anacrotism, and also to the probable cause of the pre-dicrotic notch, and we may now consider some of the conditions under which, in practice at the bedside, we find the ventricular part of the pulse wave so modified.

If the glottis be closed, and the pressure within the thorax and abdomen be raised by powerful continuous contraction of the respiratory muscles, we produce a change in the distribution of the blood in the arteries and veins. The intra-abdominal and intrathoracic veins become relatively empty, and an abnormally large quantity of blood accumulates in the systemic arteries. During this state the arterial walls are more or less powerfully distended, and, following known laws regarding arterial elasticity, they are in that condition more rigid than when their calibre is normal. Even in tolerably young subjects, by this means we can easily produce artificially an anacrotic pulse wave, the arteries being rendered relatively rigid in relation to the quantity of blood which is forced into them at each ventricular contraction. This arterial engorgement or high pressure, only temporary in such an experiment, is, however, lasting in certain diseased conditions, the most marked of these being the arterial high pressure which accompanies certain forms of chronic kidney disease, particularly interstitial nepleritis, in such cases indeed the conditions are still more favourable for the production of an anacrotic pulse wave, seeing that not only are the arteries abnormally rigid from the distension, but also that the quantity of blood forced into them with each contraction of the ventricle is relatively and absolutely great, owing to the existence of hypertrophy of the left ventricle, while at the same time the escape of blood from them is hindered by the heightened tonicity of the arterioles. Analogous conditions occur, as already indicated, in cases of atheroma or calcification of the larger arteries, such as occur in old age. The conditions, therefore, which produce the anacrotic pulse wave are in practice either abnormal distension of the larger arteries, accom-

panied or not by hypertrophy of the ventricle, or rigidity of the arterial walls due to atheromatous changes. The more marked these conditions are, the more is the second elevation (*c*) higher than the first (*b* in figs. 26 and 27). In practice, all imaginable intermediate forms between the typical pulse wave of health and the typical anacrotic pulse wave, as in fig. 27, are encountered, and it is usually easy in each individual case to tell from the other phenomena whether the anacrotism is due to simple distension of the arteries from high pressure, to atheromatous changes, or to hypertrophy of the heart. From what has been said, it will be understood that, although the anacrotic pulse wave very often means an abnormally high arterial pressure, this is by no means always the case. Finally, before leaving the changes confined chiefly or entirely to what I have named the ventricular part of the pulse wave, a word may be said regarding the conditions which favour the appearance of a well-marked pre-diastolic notch. The condition fitted to produce this notch in its most marked form, is that in which the part of the systemic arteries nearest the heart is abnormally rigid; for it need scarcely be said that if this latter part of the systemic arterial system is fairly elastic, it will contract behind the suddenly impelled first wave, and prevent more or less completely the formation of a negative wave or tendency towards a vacuum at the commencement of the aorta.

We now turn to consider a different series of changes in the form of the pulse wave, which are due to changes in the arterial circulation of an entirely different kind from those above referred to, and in which the dicrotic notch is abnormally exaggerated.

*Abnormally Dicrotic Pulse Waves.*—It has been already said that, on closing the glottis and contracting powerfully the respiratory muscles, the systemic arteries are at first abnormally filled with blood. This abnormal distension very soon, however, gives place to an abnormal emptiness of these vessels, owing to the fact that the pressure on the intrathoracic veins diminishes the quantity of blood which reaches the ventricle, the result being that the blood accumulates chiefly in the veins of the head and

limbs. The artificial arterial anæmia so produced leads to a characteristic change in the form of the pulse wave, which becomes, as in Fig. 28, smaller in size and more markedly dicrotic than the normal pulse, while all trace of anacrotism completely disappears. It is unnecessary for us to go minutely into the theory of the production of the abnormally dicrotic pulse wave. For practical purposes it will suffice to refer to the conditions which lead to the appearance of this form of curve Roughly speaking, these may be said to consist in abnormal emptiness of the arterial system, such as is produced, for example (*a*) by anæmia after venesection, in which case the absolute quantity of blood in the arteries is diminished, although these latter contain relatively normal amount; (*b*) in cases of unusual expansion of the arterioles and capillaries leading to a relatively rapid outflow from the arteries, as in the condition produced by amyl-nitrite inhalation; or finally, dicrotism may be produced by (*c*) diminution in the quantity of blood which enters the aorta through the ventricle — the most marked examples of which are to be found in cases of uncompensated mitral regurgitation. Such are the conditions which, in practice, are found to produce the dicrotic pulse wave; and it may be noted in passing, that the simple or pure dicrotic pulse wave invariably results from abnormally low arterial pressure, the cause of which, in individual cases, it is rarely difficult to discover.

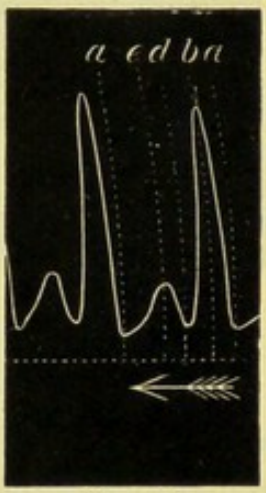



FIG. 28.
Abnormally Dicrotic Pulse Wave.

*Hyperdicrotic* is the term applied to that form of the dicrotic pulse in which the dicrotic notch descends lower than the commencement of the systolic rise. This is due to the fact that each successive cardiac systole follows its predecessor before the pressure within the artery has fallen below that which it presented at the dicrotic notch. This form of curve (Fig. 29), although presenting a notch in its ascending part is due to entirely different conditions from those which produce the true anacrotic pulse wave with which it can never in practice be

confounded, owing to the fact that the rounded smooth eminences of which it is made up show it at a glance to be of the dicrotic type; it is, in fact, an exaggerated dicrotic pulse wave.

The anacrotic and the dicrotic pulse waves are the two principal simple modifications which are met with, but there are various intermediate or combined pulse waves due to combination of the conditions fitted to produce the anacrotic and the dicrotic waves,—for example, in cases of aortic regurgitation with hypertrophied ventricle, the first or ventricular part of the curve usually is of the true anacrotic type, while on cessation of the systole, the reflux into the heart causes a more or less powerful negative wave producing an abnormally deep dicrotic notch. In addition to this the rapid filling of the comparatively empty arteries with each ventricular systole leads to an abnormally steep and high ascending limb of the curve.




FIG. 29.
Hyperdicrotic Pulse Waves.

The conditions which lead to the two principal forms of pulse wave met with in disease have been indicated, but it should be remembered that all possible combinations of these conditions are constantly occurring, leading to some less well-defined type of pulse curve. Into a detailed account of these more complicated pulse curves we cannot enter here. Examples are to be found in the various modifications of pulse curve in prolonged fever cases; in the early stages of which it is often high and bounding with a tendency towards the anacrotic type, but gradually, from day to day the curve becomes more and more dicrotic, and not unfrequently is eventually hyperdicrotic.

**Cardiograph.**—The results which may be obtained by the use of the cardiograph are less satisfactory than those of the sphygmograph. This is in part due to the fact that none of the instruments at present in use for recording the contractions

of the human heart can compare with the sphygmograph in so far as compactness and accuracy are concerned.

*Normal Heart Curve.*—In Fig. 30 is represented a typical normal curve of this kind. The curve, it will be seen, immediately after rising from its lowest point *f*, describes a more or less well marked rounded elevation between *f* and *a*, and from *a* it ascends at first rapidly, afterwards somewhat more slowly, to its highest point *b*, from whence it describes a more or less

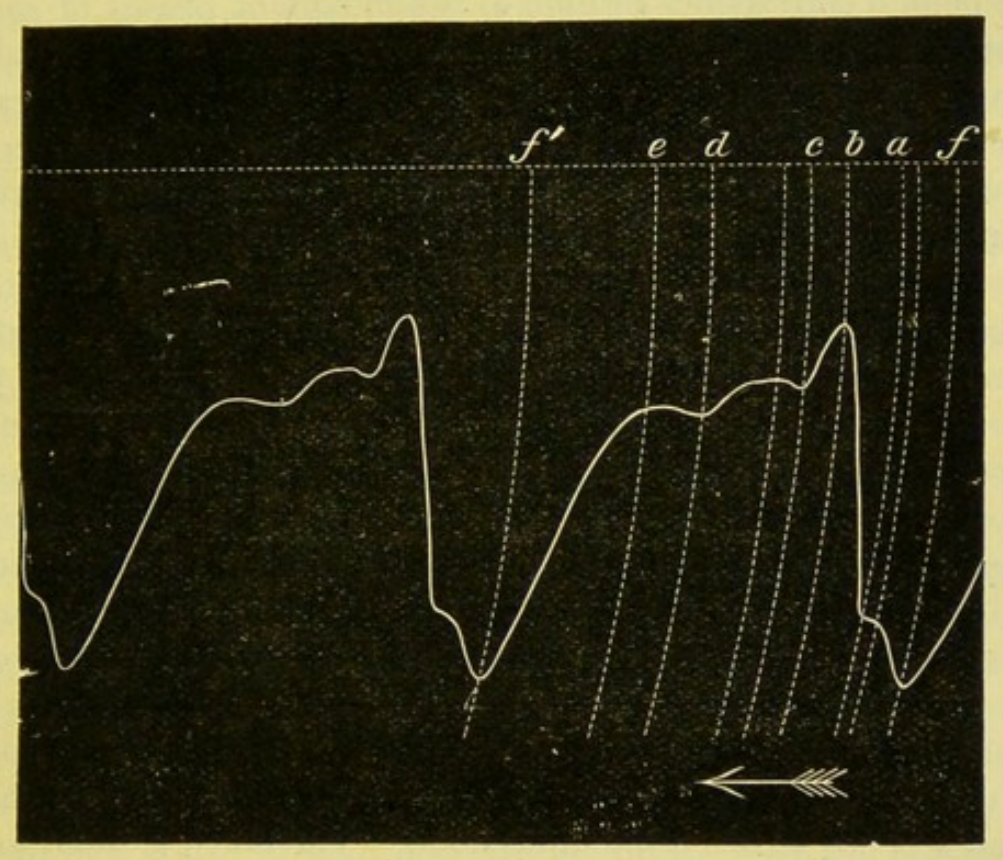



FIG. 30.—Normal Heart Curve.

obliquely descending, usually undulating line to *e*, after which the curve descends, at first slowly, then more rapidly, and finally with increasing slowness, until the point *f'* is reached. That part of the curve lying between *f* and *a* corresponds in time to the contraction of the auricles, and when the curve is taken from the apex, the elevation between *f* and *a* is due to the more or less sudden filling of the ventricles, which results from the auricular contractions. That part of the curve lying between the lines *a* and *e* is produced during the time of con-

traction of the ventricular muscle, while the part from *e* to *f* corresponds with the passive expansion of the ventricular muscle. In so far as the ventricles are concerned, we may divide the whole heart curve into two parts—viz., first, that from *a* to *e*, during which the ventricular muscle is in a state of contraction; and second, that from *e* to *a*, which corresponds to the ventricular diastole. The sudden rise from *a* to *b* is produced by the tightening of the ventricles over their contents, and the point *b* corresponds in time to two important phases of each heart beat—viz., first, the moment of closure of the auriculo-ventricular valves; and secondly, the moment when the heart muscle has fairly grasped its contents. The height of *b* over *e* gives some indication of the difference in antero-posterior diameter of the heart at commencing systole as compared with the end of the systole, for it need scarcely be said that the larger the quantity of blood contained in the ventricle at the commencement of the ventricular systole, the greater will be its antero-posterior diameter, and therefore the more powerful impulse will be given to the chest wall and cardiograph button. As the heart empties itself during systole the antero-posterior diameter of the ventricles diminishes with corresponding rapidity, and the pressure against the chest wall and cardiograph button falls in the same ratio. The result of this is that, *cæteris paribus*, the degree to which the line joining *b* and *e* descends gives a valuable indication regarding the quantity of blood thrown out by the ventricles at each systole. The meaning of the notches *c* and *d* is not satisfactorily understood. The notch *d*, when well marked, probably corresponds to the conclusion of the outflow of blood from the heart, and is therefore the analogue of the dicrotic notch of the pulse wave. It must be added, however, that it is by no means uniformly to be seen. Of greater importance is the position of the last elevation or corner of the curve at *e*, which can in almost all curves clearly be made out. This elevation marks the commencing relaxation of the ventricular muscle, and by measuring the distance between the lines *a* and *e* we are enabled to learn the duration of the ventricular systole in any given case. It may be noted, in passing, that the dura-

tion of the ventricular systole, and the duration of the outflow from these cavities, by no means necessarily or even usually correspond. The ventricular muscle contracts with a certain definite force, and remains contracted for a certain definite time, neither of these being influenced by the quantity of blood contained in the ventricle at the commencement of its contraction. The result of this is, that where a very small quantity of blood is contained in the ventricles at the commencement of their contraction, the outflow from them may have concluded some tenths of a second before the ventricles begin to relax. The distance between the lines *e* and *f* gives some indication of the rapidity with which the heart muscle has relaxed after the conclusion of its contraction. Where the elasticity of the heart muscle is modified, as when the blood contains a largely diminished quantity of oxygen, the ventricular muscle takes a longer time to relax than is normally the case, and the curve from such a beat descends less rapidly than in health.

The cardiographic curve then enables us to measure with very considerable accuracy the absolute and relative duration of the different phases of the cardiac revolution. It also gives us some idea of the force of the ventricular contraction corresponding to the height of the line *a* to *b*, and it further affords valuable information regarding changes in the force and frequency of the heart's action, which make up the different forms of irregularity of the heart.

It is unnecessary to refer more in detail to the normal typical heart curve, and we may now consider those diseased conditions which modify its form ; and first, with regard to the cardiogram in aortic regurgitation. After what has been said regarding the meaning of the various parts of the normal heart curve, it is not difficult to understand in what way these may be modified in a typical case of aortic regurgitation. In the first place, the ventricle, before the contraction of the auricles, is abnormally distended with blood ; and on the auricles propelling their contents into the already filled ventricle, an abnormally great distention of the ventricles occurs. The result of this is that, in cases where there is no failure in the power of the auricular

walls, the elevation between the letters *f* and *a* is abnormally high. On ventricular contraction occurring, the antero-posterior diameter of the heart diminishes very rapidly, corresponding with the abnormally large quantity of blood contained in the ventricle, so that the line joining points corresponding to *b* and *e* is unusually steep, while the regurgitation of blood through the incompetent aortic valves, after the cessation of the systole, causes a dilatation of the relaxing cardiac muscle sufficient to produce, in most cases a very well marked rise after *e*. It is important to note that, in the heart curve of well marked aortic regurgitation, it is often impossible to find the exact point corresponding to *e* in the normal curve at which the systole suddenly ceases. The corner of the curve preceding the descent is usually in the aortic regurgitation cardiogram some fraction of a second later than the time of commencing relaxation. In all, or nearly all, cases of aortic regurgitation, the heart curve presents two well marked peaks, and this may be said to be the distinguishing character of the cardiogram of that disease, and, roughly speaking the more marked this bicornual character is, the greater is the incompetence of the valve. Such is the curve when the ventricular muscle is comparatively unimpaired in contracting power, as, for example, in sudden rupture of one of the cusps of the valve, or when one of these is artificially destroyed in the lower animals; but where the ventricle no longer completely empties its contents at every contraction, the fall of the line from *b* to somewhere about *e* becomes, as we might anticipate, less and less steep, due, it need scarcely be said, to the slighter diminution in the antero-posterior diameter of the ventricles, which occurs when the ventricle no longer empties itself completely at each contraction. In these cases, then, the bicornual character is not so well marked as is otherwise the case in that disease, but still it is usually sufficiently recognisable.

With regard to the curve in cases of mitral incompetence, we would anticipate, where this condition was well marked, that the heart curve would be modified chiefly, if not exclusively, at that part which corresponds in time to the closure of the auriculo-

ventricular valves; in other words, at the point marked *b* in fig. 30; and this to a certain extent is the case. As a rule, we find the ascending line from *a* to *b* less steep than is normally the case, and the peak at *b* rounded off to some extent. This is apparently the only characteristic change in the form of the heart curve which results from simple mitral incompetence; but it is by no means usually well marked, owing to the fact that the large quantity of blood which leaves the ventricle during systole causes a very considerable diminution in the antero-posterior diameter during ventricular systole; so that the line from *b* to *e* is unusually steep, thus tending to cover the rounding off which the peak *e* would otherwise present. It is rare to find that the reflux of blood from the auricle into the ventricle, which follows the conclusion of the systole of the ventricle, distends the latter with sufficient force to cause, as is the case in aortic regurgitation, second elevation after *e*. Finally, where the auricles are hypertrophied, the auricular elevation in the heart curve of mitral incompetence is abnormally well marked. Such are the changes produced by simple mitral incompetence, which has been more or less completely compensated by hypertrophy of the auricle and dilatation and hypertrophy of the ventricle; but in cases where either the auricle or the ventricle begins to fail, there are endless modifications in the cardiogram.

With regard to pure mitral stenosis, we would *à priori* expect that the ascending line from *a* to *b* would be abnormally steep, owing to the abnormal rigidity of the mitral valve, and that the peak *b* would be unusually sharp, corresponding as this does with the thump which is characteristic of the disease in question —and such is certainly sometimes the case; but when it is remembered how commonly stenosis of the mitral valve is complicated with regurgitation, it need cause no wonder to find that the cardiogram in mitral stenosis is by no means characteristic or typical.

**Sphygmomanometer.**—An instrument has been devised and introduced by von Basch, to which he gives the above name, for the purpose of estimating the blood-pressure in the human

subject, and which has undoubtedly considerable clinical value. In principle it closely resembles an apparatus previously described by Prof. C. S. Roy and the author,[1] which was more specially adapted for estimating the blood-pressure in the lower animals. In von Basch's sphygmomanometer, a small cushion of membrane is made to press upon the skin over the radial artery, and the pressure is communicated through water to a column of mercury, or, in the more recent forms of instrument, to a spring manometer, by which its value can be ascertained. The pressure of the cushion upon the radial artery is gradually increased, until all pulsation in the vessel beyond the constricted point ceases; and this point is taken as the maximum arterial pressure.

While open to many sources of error, the readings of this instrument, if taken with sufficient care, appear to give results which are approximately accurate; at any rate, quite sufficiently so for ordinary clinical work. In healthy men between the ages of twenty and sixty the pressure averages about 150 m.m., but may be as low as 135 m.m., or as high as 170 m.m. High pressures much exceeding these, and running as high as 245 m.m., are met with in such affections as chronic Bright's disease with cardiac hypertrophy, while in such diseases as anæmia, phthisis, &c., the blood-pressure is found to be very low.

This instrument seems to be specially suited for watching the progress of individual cases from day to day, and in particular for observing the effects of treatment upon the blood pressure.

[1] *Verhandlung der physiol. Gesellschaft zu Berlin*, 15th Feb. 1878, and in the *Journal of Physiology*, vol. ii. p. 323.

## CHAPTER XIV.

## RESPIRATORY SYSTEM.

THE first symptoms which require notice in connection with the respiratory system are—

**Subjective phenomena**, such as pain, tickling, burning, &c. These are frequently felt over the larynx, trachea, and bronchi, when these structures are the seat of disease, and are usually aggravated by pressure, and by the acts of speaking and coughing. *Pain* may manifest itself in connection with disease of the lung tissue, but it attains its greatest importance in cases of pleurisy, where the pain has a peculiar dragging, shooting character, is increased by pressure, and by any movement of the thorax. Its differential diagnosis is very important.

We must distinguish the pain of pleurisy—

(1.) *From the Pain of Pleurodynia*, or rheumatism of the intercostal muscles. In this condition the pain usually comes on with excessive suddenness, after some abrupt movement, and is unaccompanied by pyrexia, or by friction sound.[1]

(2.) *From the Pain of Intercostal Neuralgia.*—In this affection there are commonly three tender points (*points douloureux* of Valleix) in the course of the affected nerve, one close to the vertebral column, one in the axilla, and a third over the terminal branches near the sternal border. The presence of these points, the neuralgic character of the pain, and the absence of pyrexia and of all pulmonary physical signs, except such alterations of the respiration as the pain occasions, will suffice to distinguish this affection from pleurisy.

[1] It must be remembered, however, that this auscultatory phenomenon may be awanting in the early stage of pleurisy, so that the physician may have to refrain from a positive diagnosis until this symptom has had time to develop.

(3.) *From the Pain of Herpes Zoster.*—This eruption, which follows the course of the intercostal nerves, is often preceded by severe pain, usually of a burning character. The marked cutaneous hyperæsthesia which frequently accompanies this pain, as well as the signs mentioned in the previous paragraph, will suffice to distinguish it from pleurisy.

(4.) *From the Pain of Periostitis and other Surgical Affections of the Thoracic Wall.*—Careful examination of the ribs should make clear the nature of such pain.

**Breathing** will be more conveniently considered hereafter (p. 182).

**Cough.**—The removal of foreign substances from the respiratory passages is effected by means of the acts of sneezing and coughing,—two forms of explosive expiration which are both, as a rule, excited reflexly, and which both consist in a closure of the respiratory passages after a deep inspiration, followed by a sudden, forcible, and noisy opening of the same, the result of a powerful expiratory effort. In the case of sneezing, the closure is effected by the pressure of the soft palate, by means of the tongue, on the posterior wall of the pharynx, while in coughing the closure takes place at the glottis.

Coughing may be excited by irritation of the terminal branches of the superior laryngeal nerve distributed to the mucous membrane of the larynx and trachea. The inhalation of cold air, or of air laden with dust, the passage into the larynx of particles of food, or other foreign bodies, and the collection of secretions, or of such morbid productions of blood, or pus, all tend to excite coughing, which is more liable to occur when, in addition there is hyperæsthesia of the parts, the result of catarrh or inflammation. The terminal branches of the vagus distributed in the bronchi, lung tissue, pleura, or abdominal viscera, or even the small branch to the auditory meatus, may be the starting point of the irritation, while in sensitive individuals, the action on the skin of a draught of cold air is sufficient to set up cough. Anæsthetic conditions of the larynx are occasionally met with in which such local

irritations as those mentioned are not sufficient to excite cough; and depression of the activity of the reflex centre in the medulla, the result, for example, of the accumulation of carbonic acid in the blood, or of the action of opium, may diminish or completely abolish the act of coughing, and thereby cause a dangerous accumulation of secretion in the air passages and alveoli.

In examining cough as a symptom, it is well to note—

1*st. Its Frequency and Rhythm.*—The physician should inquire whether it comes frequently, each individual cough being separated by a tolerably constant interval, or whether there occur paroxysms of coughing with intervals of quietness. The paroxysmal cough is well seen in cases of whooping-cough, where there is a series of short sharp coughs, followed by a long-drawn deep stridulous inspiration. Such paroxysms are often followed by vomiting.

2*nd. Its character.*—This may vary very greatly. The cough may be dry, as in pleurisy, the first stage of phthisis, &c., or moist, as in chronic bronchitis, and in the last stages of phthisis. It may be painful, as in acute pleurisy, and the patient then instinctively tries to suppress the cough which gives him so much suffering; and this short, dry, suppressed cough is frequent at the commencement of acute pneumonia, and also in cases of intercostal neuralgia, pleurodynia, pericarditis, and peritonitis. Very different from this is the loud barking cough of hysteria, which is obviously produced at will, and calculated to attract the utmost amount of attention. In laryngitis, even when the disease is very slight, the cough is hoarse, husky, stridulous, and croupy in character. The hard metallic cough met with in cases of aortic aneurism, where there is pressure upon the trachea, is often of considerable diagnostic value. It is compared by Wyllie to the cry of a gander, and is believed by him to result from vibrations produced in the tracheal air column, originating at the compressed point. A variety of cough, termed *bovine* by Wyllie, and characterised by want of sudden explosive commencement, is sometimes seen in cases of labio-glosso-laryngeal paralysis, and is due to defective closure of the glottis.

3*rd.* Notice whether the cough is obviously brought on by such causes as exertion, change of posture, inhalation of cold air, of dust, or of irritating chemical vapours.

4*th.* Notice if the paroxysm teminates in a fit of vomiting, as so often occurs in whooping cough, phthisis, and chronic bronchitis, or in the prolonged, clear, shrill inspiration which characterises the first of these affections.

**Sputa.**—In almost every affection of the respiratory organs, more or less expectoration follows the act of coughing. Occasionally, however, this is absent; and in the case of young children, even when the cough is accompanied with expectoration, the sputum is swallowed as soon as it reaches the mouth. It must be borne in mind that the material coughed up may not come originally from the respiratory tract: for secretions from the mouth, nose, and pharynx may pass the rima glottidis, and, irritating the mucous membrane of the larynx, be coughed up again. Bleeding from the posterior nares may thus simulate hæmoptysis.

*Chemical Characters.*—As yet the chemical analysis of sputa has not proved of much diagnostic value. Consisting, in the main, of water, sputa have at different times been found to contain serum-albumin, serum-globulin, myosin, nuclein, glycogen, various fatty acids, leucin, tyrosin, &c., in addition to the mucin which is invariably met with even in the healthy state, and which imparts to the expectoration its peculiar viscid character. Albumin is always present when there is inflammation of the air-passages or lung-substance. In cases of diabetes, sugar has been detected in the sputa, and in renal affections urea may sometimes be found.

### *Macroscopic Characters of Sputa.*[1]

For purposes of ready description the various varieties of sputa may be classified as follows, each being named after its principal constituent.

(1.) *Mucous sputum* is transparent, clear, and glassy, and has a viscid and ropy consistence which is best appreciated by

[1] In all cases of laryngeal and pulmonary disease the sputa should be regularly examined, and for this purpose the expectoration for twenty-four hours should be collected in a glass vessel of such shape as to permit of rapid and satisfactory inspection.

pouring it from one vessel into another. It is sometimes present in health, often becoming constant in advanced life, but is most frequently found in the earlier stages of bronchial catarrh. There is a very slight admixture of pus corpuscles.

(2.) *Muco-purulent sputum* may occur in almost every affection of the bronchi and lung. When allowed to stand in a vessel, the pus corpuscles sink to the bottom, leaving the clear mucus floating on the surface. Sometimes, however, a more intimate mixture of these two elements takes place. When cavities are present in the lung, the sputum often takes peculiar forms. Round flattish masses of purulent matter, with well defined margins, are then seen lying at the bottom of the vessel. From the resemblence of these masses to coins this variety of sputum is sometimes called nummular. Very rarely a similar appearance is observed in cases of chronic bronchitis.

(3.) *Purulent sputum* resembles closely ordinary pus as obtained from an abscess. It has the same yellow opaque appearance, and separates into two layers when allowed to stand, the lower being composed of pus corpuscles, the upper of plasma. This variety of sputum is usually derived from suppurating cavities in the lung, or is the result of other collections of pus (for example, empyæma) bursting into a bronchus.

(4.) *Serous sputum* is that form which is met with when copious transudation takes place from the pulmonary circulation, as in œdema of the lungs. It has a characteristic thin, transparent appearance, and is usually copious and frothy. Consisting as it does, in great measure, of transuded serum, this form of sputum contains much albumin.

(5.) *Sanguineous sputum.* — The sputum may be simply streaked with blood (as in the early stages of phthisis, &c.), or the blood may be mixed intimately through the mass. This latter form is that most usually met with in the later stages of phthisis, in cases of hæmorrhagic infarction, and in lobar pneumonia. In the last-named affection the sputum is of a rusty colour, due to chemical alteration of the blood pigment, and this may pass into citron-yellow and green. It has been already said that blood from the throat and posterior nares may trickle into the trachea and be coughed up. The primary source

of the hæmorrhage is then, however, usually clear. It is more difficult to distinguish hæmorrhage from the lungs (hæmoptysis) from that from the stomach (hæmatemesis). The history and physical examination, and the nature of the act by which the blood reached the mouth, will help greatly towards diagnosis; but it must be remembered that the blood coughed up may be swallowed and then vomited. In hæmoptysis the blood is usually bright red, fluid, and frothy, has an alkaline reaction, and when examined microscopically is found to contain more or less of those cellular elements which are peculiar to the respiratory tract. In hæmatemesis the blood is dark and venous, sometimes chocolate-brown, resembling coffee-grounds, often clotted, free from froth, acid in reaction, and when microscopically examined is found to contain fragments of food. This subject has been already considered in reference to hæmatemesis.

*Physical Characters of the Sputa.*

(1.) *Quantity.*—The amount of expectoration may vary very much, and this indication may become of considerable diagnostic value, as, for example, when in the course of some acute affection (bronchitis, pneumonia) the scanty sputum suddenly becomes more abundant and more readily expectorated, showing thereby that the acuteness of the inflammation is subsiding. In bronchiectasis very large quantities of sputum are brought up at one time, and so marked is this symptom that it may suffice in many cases to establish a diagnosis in the absence of other signs.

(2.) *Form and Consistence.* — The more mucus the sputum contains, the firmer will be its consistence, and the more distinct its form. Tenacious sputa are consequently found in the acute stage of bronchitis, pneumonia, phthisis, &c. In the absence of mucus, the sputa lose their individual shapes, and, when collected in a vessel, they coalesce with each other. Such is the case with the purely purulent and the serous sputa. Tough sputa from phthisical cavities preserve their flattened, coin-like (nummular) shape after expectoration—an indication of some diagnostic value.

(3.) *Smell.*—As a rule, sputa are devoid of any very marked odour. When, however, putrefactive organisms are present the

odour of the breath and of the expectoration becomes most overpowering. This occurs to a marked degree in bronchiectasis, putrid bronchitis, and pulmonary gangrene.

(4.) *Colour.*—To the yellow or yellow-green tinge which is imparted to the sputum by pus cells when they are present, allusion has already been made. The red colour of sanguineous sputa has also been described, passing into rust-colour, yellow, saffron, and finally green, as the hæmoglobin becomes more and more highly oxidized. A yellow or a green discoloration frequently appears in the sputa in cases of jaundice, due to the presence of bile pigment; and those who are much exposed to smoke, or who work in coal mines, frequently expectorate the carbonaceous particles which they have inhaled, to such an extent as to blacken the sputum.

### *Microscopic Examination of the Sputa.*

(1.) *Pus, Blood, and Mucus Corpuscles.*—The recognition of these corpuscles is very readily made by means of the microscope. What diagnostic significance attaches to each has been already stated, and does not demand further remark.

(2.) *Epithelial Cells.*—The ordinary pavement epithelial cells of the mouth, pharynx and larynx are almost invariably present in sputa, becoming mixed with the expectoration on its passage through the mouth. They are of large size, polygonal in shape, finely granular, and possess a large, refractive, ovoid nucleus.

The columnar epithelium of the bronchial mucous membrane, both goblet and ciliated cells, are sometimes found in the sputum in the early stage of bronchial catarrh.

Much more important for diagnosis is the occurrence of the epithelium of the pulmonary alveoli. In the sputa, this alveolar epithelium is readily recognised. The cells are spherical or slightly oval, have a diameter two to four times greater than that of a leucocyte (thus distinguished from all other round cells in the sputum), contain granular protoplasmic masses, and possess one or more large oval nuclei with distinct nucleoli. They further differ from all other cells to be found in the sputum in that they readily become pigmented, and undergo fatty and

myelin degeneration,—changes which the other varieties seldom or never show.

Regarding the diagnostic value of the cells, it is important to observe that above the age of thirty to thirty-five years alveolar epithelium is occasionally to be found in the sputa of perfectly healthy persons, but that variety of cell is not found in individuals whose age is below thirty. At all ages, however, alveolar epithelium may be found in the sputa of many affections of the respiratory organs — in œdema, hypostatic congestion, hæmorrhagic infarction, pneumonia, and in all the forms of phthisis. In simple bronchial catarrh of individuals under thirty, no alveolar epithelium is to be found in the sputa, unless the very finest bronchioles be affected, and then these cells appear only in small number. In commencing phthisical catarrh of the apex, however, alveolar epithelium is to be found in considerable quantity long before any physical sign can be detected, and in young individuals in whom all the other causes mentioned can be excluded, the occurrence of alveolar epithelium is very suggestive of commencing phthisis.

In cases of heart failure, with pulmonary congestion, a special variety of these alveolar cells, called *heart-failure cells*, is met with in the sputum. They are recognised by the fact that they contain many brownish-yellow pigment granules, and, when they appear constantly and in considerable numbers, they point to heart failure, usually to mitral disease, provided that the presence of pneumonia and of infarct can be excluded.

(3.) *Debris of Lung Tissue.*—In any disease which involves destruction of lung tissue, we may find in the sputum the elastic fibres which had formed the framework of the broken-down alveolar walls. These fibres may be distinguished under the microscope without difficulty. They usually lie in groups coiled and twisted, sometimes recalling by their arrangement the outline of the alveoli. Their dichotomous branching and well-defined double contour, and still more, their resistance to the action of caustic alkalies, make their recognition a matter of little difficulty. It is well to boil the sputa with caustic soda until the mixture becomes thin and watery. The elastic fibres

will then readily sink to the bottom of a conical glass, and can be secured by means of a pipette.

While the debris of lung tissue occurs by far most frequently in the sputum of phthisis, it may also be found in cases of pulmonary abscess and of gangrene of the lung. In the last-named affection the lung-tissue is only to be found in very fresh sputum. It rapidly disappears, being apparently acted upon and dissolved by some peculiar ferment which is present in the expectoration in such cases. It need hardly be said that where such elastic fibres occur, we have absolute proof of the destruction of lung tissue, hence the great importance of this symptom in case of phthisis where the physical signs are not distinct.

(4.) *Fibrinous Bronchial Casts.*—In pneumonia and in croupous bronchitis, there are sometimes to be found in the expectoration casts in fibrin of the finer bronchioles and their branches. In the sputum they are usually rolled together, and only unroll and spread out when washed with water. The perfect way in which they reproduce the arrangement of the bronchioles makes the

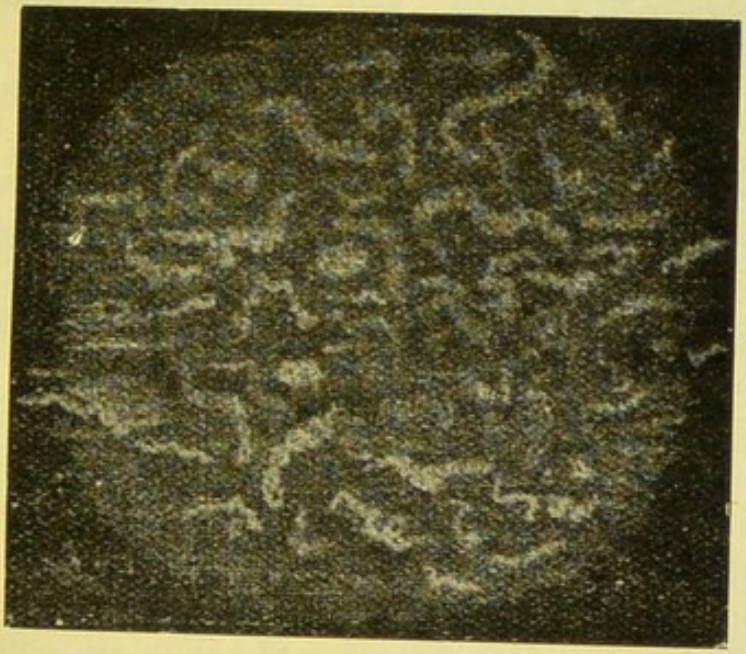


FIG. 31.—Curschmann's Spirals, in Sputum, seen on black ground (after Curschmann).

recognition of these casts easy. In pneumonia they are most numerous in the sputum on the third and fourth day of the affection, and Remak, who devoted much attention to the

subject, believed that the earlier these casts appeared, and the greater their quantity, the more quickly will recovery set in, and the more completely will the affected lung recover from the disease.

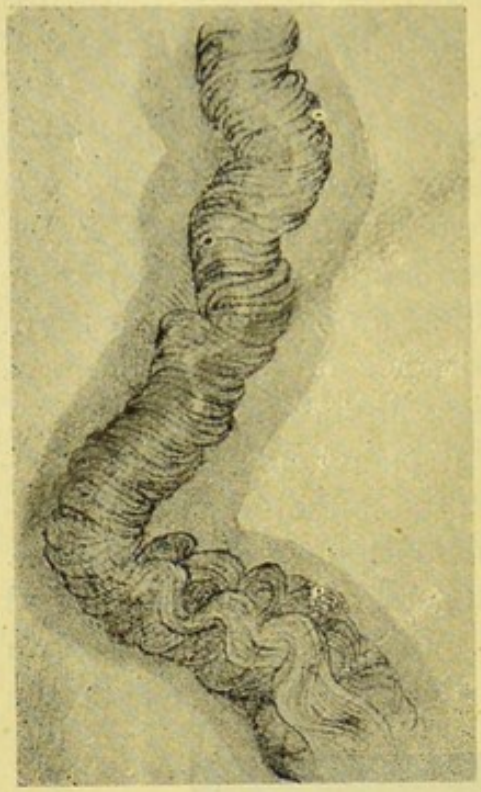


Fig. 32.
Curschmann's Spiral Magnified, (Hartnack 4, Oc. 2) (Curschmann).

Curious bodies, called, after their first describer, *Curschmann's Spirals*, are often found in sputum in cases of bronchial asthma. They consist of threads of mucin, curiously twisted in spiral fashion, and they can often be detected with the naked eye. They are formed in the finest bronchi, and while asthma is the disease in which they are most frequently encountered, they are in no way characteristic of that affection, being often enough met with in bronchial catarrh arising from other causes.

(5). *Crystals* are occasionally met with in sputa, the most common being the long, fine, colourless, needle-shaped crystals of the fatty acids. They have some superficial resemblance to elastic fibres, but are easily distinguished by the fact that they dissolve at once in ether, a reagent which does not affect elastic fibre. These fatty acids are found in cases of putrid bronchitis, bronchiectasis, and pulmonary gangrene.

Another variety of crystal which may be found in the sputum are those usually known as *Charcot-Leyden crystals*, after the name of their discoverers. Their exact nature is a matter of some doubt. In shape they vary somewhat, but are usually long, fine, sharp, and spindle-shaped; they are colourless, are insoluble in alcohol, but are readily dissolved by acid or alkalies. These crystals occur most frequently in asthma, and are, by some, supposed to be the exciting cause of the paroxysm.

Other crystals, such as cholesterin, hæmatoidin, leucin, tyrosin,

oxalic acid, and triple phosphate, occur in the sputum, but do not demand special notice here.

(6.) **Micro-organisms** of various kinds may be found in the

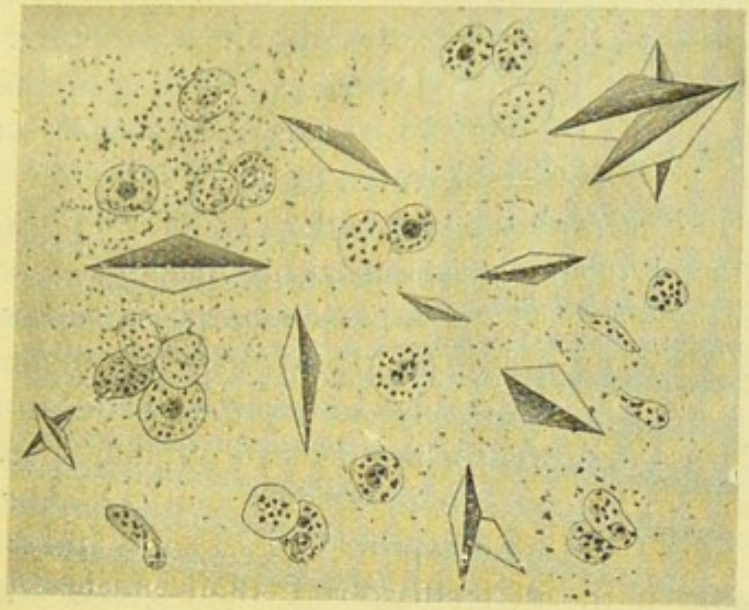


FIG. 33.—Charcot-Leyden Crystals in Sputum.

sputum, such as *leptothrix*, *oidium albicans*, and, rarely, *sarcina*. Bacteria and vibriones are very frequently to be seen in the sputum of gangrene and bronchiectasis.

Of the pathogenic micro-organisms met with in sputum, the most important are the following :—

(1.) *The tubercle-bacillus* of Koch. — These bacilli are delicate, rod-shaped structures, in length usually about 3 $\mu$, and are motionless. They can only be detected after careful staining; and for this purpose several methods may be employed, three of which may be mentioned here.



FIG. 34.—Tubercle Bacilli (after Koch)

(*a*.) Ehrlich's Method.—A thin layer of sputum is spread on

a cover-glass, which is then gently heated over a flame for a few seconds to coagulate the albumen, and placed in a staining solution prepared as follows: Five cubic centimetres of pure anilin are added to 100 cubic centimetres of distilled water, well shaken and filtered, and to the filtrate a saturated alcoholic solution of fuchsin or methyl-violet is added until precipitation commences. The cover-glass is allowed to float on this for half-an-hour. It is then washed in a solution of nitric acid (1 to 2), and afterwards in distilled water. In this way the stain is extracted from everything but the bacilli.

(*b.*) Heneage Gibbes' Method.—This process is to be preferred for clinical purposes, as it is rapid and does not require the use of nitric acid. This staining solution is made as follows:—"Take of rosanilin hydrochloride two grammes, methyl-blue one gramme; rub them up in a glass mortar. Then dissolve anilin oil, 3 c.c., in rectified spirit, 15 c.c.; add the spirit slowly to the stains until all is dissolved, then slowly add distilled water, 15 c.c.; 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 of tubercule are stained red, all other organisms blue.

(*c.*) Ziehl-Neelsen's solution may also be employed. It consists of a mixture of one part of a saturated alcoholic solution of fuchsin with nine parts of a (1 : 20) solution of carbolic acid. If this solution be heated, films will stain in about five minutes. They are then decolorized in nitric acid, washed in water, dehydrated and mounted. Methylene-blue or Bismarck-brown may be used as a contrast stain.

In many cases the detection of the bacilli is made readily enough, in others many films may have to be prepared and examined before they are found. Any cheesy-looking particles in the sputum should be carefully examined.

(2.) *Fraenkel's Pneumo-coccus.*—In the rusty sputum of acute

croupous pneumonia this micro-organism is often to be found. These cocci are small in size, with sharply pointed extremities, and usually are seen in pairs, taking the form of diplo-cocci. They are surrounded by a delicate capsule, and are well stained by Gram's method, or by Ziehl-Neelsen's stain.

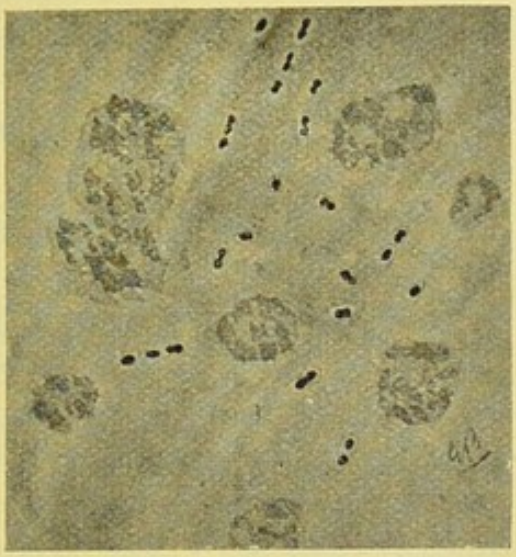


FIG. 35.—Fraenkel's Pneumo-coccus.

These pneumo-cocci are present in most cases of croupous pneumonia, and in many cases of the catarrhal form, and, in all probability, they are then the cause of the inflammation.

(3.) *Friedlander's Pneumo-bacillus.*—In a small percentage of cases of acute pneumonia this organism is to be found in the sputum, either alone or in conjunction with the organism just described. It is not unlike the pneumo-coccus, being also encapsulated, but it is rod-shaped with rounded ends. In the matter of staining there is a distinct difference between the two forms, for, while the pneumo-coccus stains with Gram's method, the pneumo-bacillus becomes decolorized during that process. Whether it stands in any causal relationship to pneumonia is at present uncertain.

(4.) *Pfeifer's influenza bacillus* is to be found in enormous numbers in the bronchial secretion, and consequently in the sputum, in that disease. It appears as very minute, fine, straight, non-motile rods, which stain readily in Loffler's solution or with dilute Ziehl-Neelsen's stain. In all probability this bacillus is the cause of the disease.

Echinococcus-vesicles are in rare cases to be found in the expectoration, having either been previously encysted in the lung, or having bored their way from the liver into a bronchus.

(7.) *Foreign Bodies.* — To the presence of carbonaceous particles in the sputum allusion has already been made. Fragments of food, when present, are easily recognised by means of the microscope.

# CHAPTER XV.[1]

## RESPIRATORY SYSTEM—(*continued*).

### EXAMINATION OF NARES, PHARYNX AND LARYNX.

We now proceed to the physical examination of the organs of respiration, and these will be considered in the order in which they naturally come—the Nares, the Pharynx, the Larynx and Trachea, and the Lungs.

**The Nares.**—The diseases of the Nose appertain more to the domain of Surgery than to that of Medicine; but the methods of examination of this region may be briefly alluded to here. Obstruction of the nasal passages obliges the patient to breathe through his mouth; and the effect of this on the general health is often very marked. The resonance of the nasal cavities is of importance in connection with the voice; and when it is interfered with by obstruction of the nares, the voice acquires a peculiar and characteristic muffled nasal tone. Besides, certain local affections are often found in the nose, to which may be traced such conditions as, asthma, deafness, hay-fever, and many reflex neuroses.

The Nares may be examined by—

(1.) *Inspection.*—"Anterior rhinoscopy" is the term applied to the examination of the nose from the front. The patient and observer should be seated facing each other, and the latter should direct a good light by means of a forehead reflector (as explained more in detail under laryngoscopy) into the interior

[1] This chapter has been revised, and, in part rewritten by Dr M'Kenzie Johnston, Assistant Surgeon to the Ear and Throat Department, Royal Infirmary of Edinburgh.

of the nostrils, which should be dilated, each one in turn, by some form of nasal speculum. By slightly moving about the patient's head, it should then be possible to obtain a view of the interior of the nose. It is sometimes advisable to apply a solution of cocain (5-10 p.c.) to the interior, which has the effect of contracting the mucous membrane, and thus giving a better view of the parts.

"Posterior rhinoscopy" is employed to obtain a view of the nasopharynx and the posterior nares. This procedure is often difficult, sometimes impossible, and always requires patience and skill. The patient should be seated as before, while the observer, gently depressing the tongue with a spatula, introduces a small laryngeal mirror through the mouth, so that its reflecting surface is directed upwards and forwards, behind the soft palate, almost, but not quite, touching the pharyngeal wall. The patient must breathe gently and regularly, so as to relax the palate, otherwise the view is apt to be cut off.

(2.) *Palpation.*—Useful information can be obtained as to the condition of the anterior nares by means of a long silver probe, used while the nose is being thoroughly illumined for inspection. The posterior nasal passages can be quickly and thoroughly examined by the index finger of the right hand passed up behind the palate. Care must be taken to prevent the patient from closing his teeth, otherwise the finger may be badly bitten.

While examining the nose by these methods, the presence of discharge, foreign bodies, adenoids, polypi or other new growths, deviation of the septum, &c., should be carefully noted.

**The Pharynx.**—The pharyngeal passage, forming part of the alimentary, as well as of the respiratory tract, has already been described in detail, but care must be taken not to overlook such things as a retropharyngeal abscess, an abnormal distribution of the pharyngeal vessels, cysts, &c.

**The Larynx.**—The larynx has three principal duties to perform—respiration, phonation, and protection—and is supplied with muscles and nerves to carry out these functions. The

abductor muscles (crico-arytenoidei postici) are chiefly concerned with the respiratory function. The muscles of phonation are the adductors (crico-arytenoidei laterales and arytenoideus), and the tensors (thyro-arytenoideus and crico-thyroideus). The reflex action by which the glottis is closed, serves to protect the larynx against the entrance of foreign substances; this is effected by the adductor muscles.

There are points as to the innervation of the larynx about which differences of opinion still exist, but it is very generally accepted that the spinal accessory is the motor nerve of the larynx. All the muscles of the larynx, except the crico-thyroid and the depressors of the epiglottis, are innervated by the inferior (recurrent) laryngeal nerve—a purely motor nerve. The superior laryngeal nerve supplies motor fibres to the crico-thyroid, and the depressors of the epiglottis, and sensory fibres to the mucous membrane of the larynx. The phonatory or voluntary movements of the laryngeal muscles have a cortical representation, while the abductors, which are the respiratory muscles, are innervated by fibres which take their origin in the bulb.

Semon and Horsley have shown experimentally, that there is in each cerebral hemisphere an area which represents bilateral adduction of the vocal cords. They found that irritation of this cortical area on one side produced bilateral adduction, but that extirpation of one area did not prevent bilateral adduction when the remaining area was stimulated. They therefore conclude that a unilateral cortical lesion cannot produce paralysis of adduction, and further, that a unilateral paralysis of adduction, due to cortical changes, cannot exist.

**Voice.**—As an index of the state of the larynx, the voice is of the utmost importance. More or less huskiness of the voice is associated with almost all the affections of the vocal cords. Aphonia, or loss of voice, may, however, result from other causes, such as paralysis of the muscles of the larynx, or the exhaustion of severe disease; or it may be of a purely functional nature as met with in hysteria. Aphonia must, of course, be distinguished from aphasia (loss of speech), and also from deaf-mutism. This

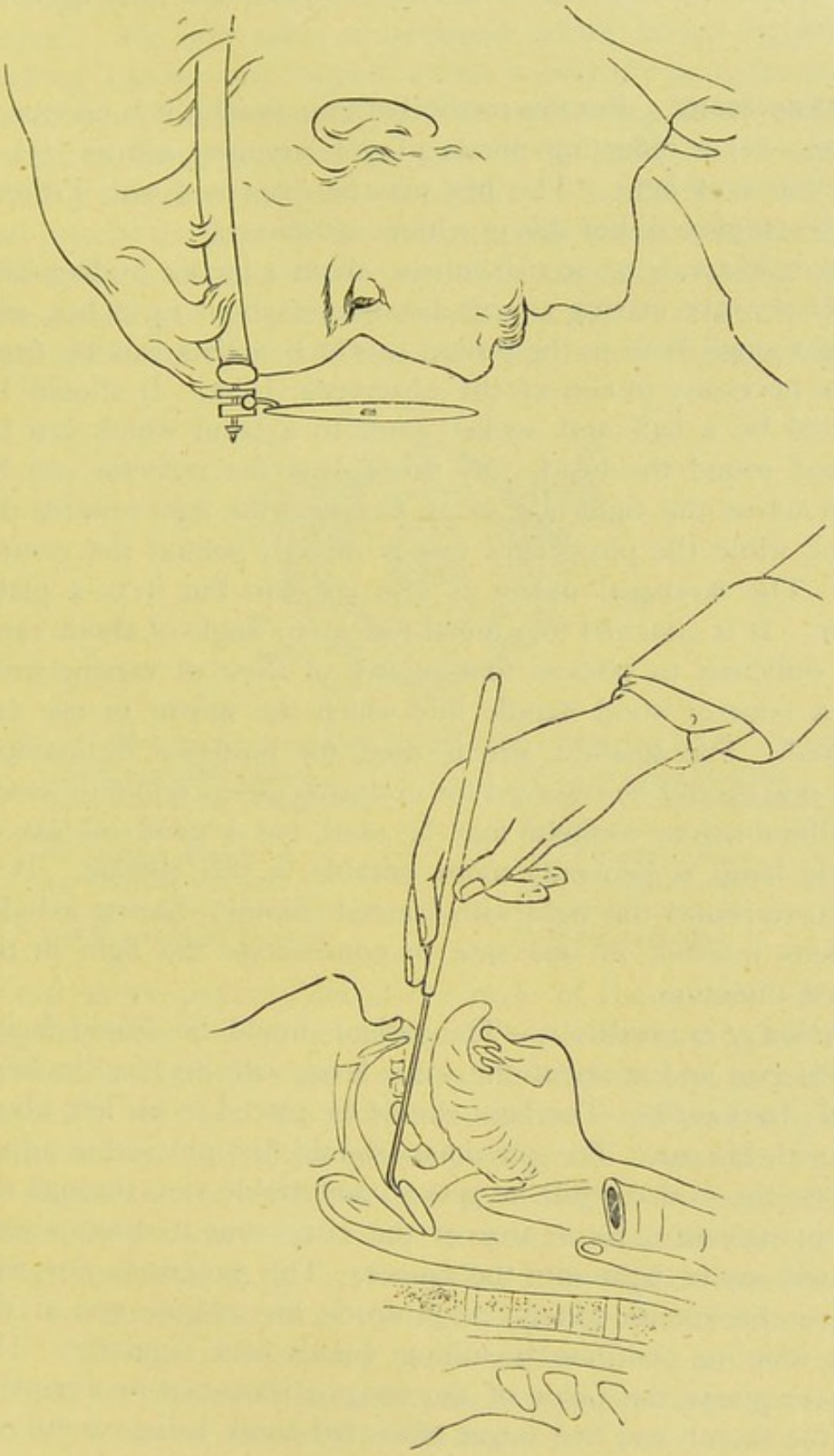


FIG. 36.—Method of using the Laryngoscope. To show the position of the fingers the hand of the observer has been drawn as occupying a position too far removed from the patient's cheek, on which the fingers ought to rest.

subject will be referred to in connection with the nervous system.

**Laryngoscopy**.—For this method of examination it is necessary to have—(1) a reflecting mirror; (2) a laryngeal mirror; (3) a good source of light. The first may be dispensed with if direct light be employed, but this is seldom satisfactory.

The reflector is a circular mirror, about 4 inches in diameter, slightly concave, having a focal distance of about 14 inches, and having a small hole in the centre, to which a lens can be fixed if it be necessary to correct the observer's vision. It should be attached by a ball and socket joint, to a band which can be fastened round the head. By this means the reflector can be drawn across the right eye, so as to reflect the light towards the patient, while the physician's eye is directly behind the central hole. The laryngeal mirror is also circular, but it is a plane mirror. It is attached to a metal rod at an angle of about 120°. It is sufficient to possess four or five of these of varying sizes, with a bone or wood handle into which the mirror in use can be fixed. The smallest size is used for posterior rhinoscopy, while one should be reserved for exclusive use in syphilitic cases. For illumination, sunlight may be used, but a good oil, gas or electric lamp is generally more suitable in this climate. It is best to surround the light with a metal chimney, having a bull's eye lens inserted in one side to concentrate the light in the desired direction.

*Method of examination.*—The patient should be seated facing the observer and at about the same level, with his head inclined slightly backwards. The light should be placed to his left, about level with his ear. The physician should first proceed to adjust his reflector so that while he gets a comfortable view through the hole in its centre, he is able at the same time to direct a well-focussed ray of light into the fauces. The patient is now told to open his mouth widely, to protrude his tongue, and at the same time to continue breathing quietly and regularly. The observer grasps the point of the tongue, protected by a napkin, with the thumb and first finger of his left hand, being careful not

to drag on it but merely to steady it and prevent its being retracted. He now takes a laryngeal mirror in his right hand, holding it as he would a pen, warms it over the lamp (to prevent the moisture of the expired air from condensing on it), and, after testing its temperature on the back of his hand, he introduces it into the mouth. He passes it rapidly to the back of the throat and presses the back of the mirror upwards and backwards, thus raising the uvula out of the way, while carefully avoiding contact with the tongue or pharyngeal wall. It will increase the steadiness of this movement if the third and fourth finger be allowed

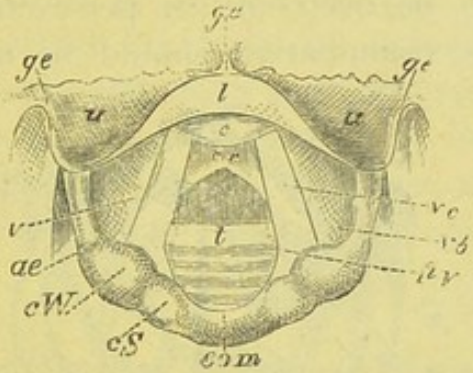



FIG. 37.—Diagram of Laryngoscopic Image in Quiet Inspiration (Morell Mackenzie)

(*ge.*) Glosso-epiglottic folds.
(*u.*) Upper surface of epiglottis.
(*l.*) Lip of epiglottis.
(*c.*) Cushion of epiglottis.
(*v.*) Ventricle of larynx.
(*ae*) Ary-epiglottic fold.
(*cW.*) Cartilage of Wrisberg.
(*cS.*) Capitulum Santorini.
(*com.*) Arytenoid commissure.
(*vc.*) Vocal cord.
(*vb.*) Ventricular band.
(*vp.*) Processus vocalis.
(*cr.*) Cricoid cartilage.
(*t.*) Rings of trachea.

to rest on the patient's jaw at the angle of the mouth. If these steps have been taken quietly, so as not to flurry the patient, the larynx should now be more or less perfectly in view, and if he be desired to say "ah," the movements of the cords can be followed in the mirror. By slight movements of the mirror the whole of the larynx can be explored, and according to its position there will come into view the various structures seen in the laryngeal image, as shown in figs. 37 and 38. It must be borne in mind that the parts reflected in the right side of the mirror (as viewed by the observer) correspond to the patient's left, and *vice versâ ;* and the more anterior structures are seen in the upper part of the mirror, the more posterior in the lower.

It may be well to notice one or two of the difficulties likely to be experienced in performing laryngoscopy. Hyperæsthesia of the pharynx is often met with and is best remedied by the application of some cocain (5-10 p.c.), by means of a brush. An overhanging epiglottis may prevent the cords from being seen, but often the production of a high note by the patient is sufficient to raise it. If this is not successful it may be necessary to elevate the epiglottis by means of a laryngeal probe held in the left hand. Want of care in introducing the mirror, nervousness, and temporary cessation of respiration, dragging the tongue, &c., can generally be overcome by perseverance and practice. The laryngoscopic examination should be made methodically,

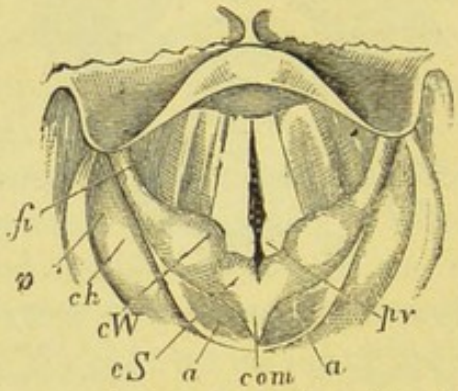



FIG. 38.—Diagram of Laryngoscopic Image in the Act of Vocalisation (Morell Mackenzie).

(*fi*.) Fossa innominata.
(*sp*.) Sinus pyriformis.
(*ch*.) Corner of hyoid bone.
(*cW*.) Cartilage of Wrisberg.
(*cS*.) Capitulum Santorini.
(*a*.) Arytenoid cartilages.
(*com*.) Arytenoid commissure.
(*pv*.) Processus vocalis.

and the information obtained, carefully noted, under such headings as the following:—

(1.) *Colour.*—In the normal larynx the mucous membrane is of a pale coral appearance, and the true cords stand out distinctly, having a pearly-white colour. In anæmic and tubercular conditions the larynx is much paler in colour; whereas, in acute or chronic catarrhal affections, the parts may assume a colour varying from bright red to a deep purple red.

(2.) *Ulceration.*—If ulcers are visible, their position, size, and general features should be noted.

(3.) *Tumifaction.*—Swelling of the parts round the glottis may occur from a variety of causes, and the early recognition of the

nature and the cause of this dangerous condition is of the utmost importance. If new growths are met with, their character, size, and situation should be accurately described.

(4.) *Foreign bodies.*—The laryngoscope may be of great service in discovering foreign bodies, and in aiding their removal.

(5.) *Position and movements of the vocal cords.*—The positions which the cords may assume are—(*a*) In quiet respiration (fig. 37) the cords are separated by twice the distance which separates them after death, or when the recurrent laryngeals are completely paralysed. (*b*) In phonation the cords are in the middle line and almost in contact (fig. 38). (*c*) The cadaveric position is that in which the cords are found after death, and is about midway between adduction and quiet respiration.

These appearances may be modified by the paralysis of the nerves supplying the muscles of the larynx, and the changes so produced are of such importance as to require separate consideration.

**Paralysis of the Superior Laryngeal Nerve** causes anæsthesia of the mucous membrane of the larynx, along with paralysis of the crico-thyroid muscle and of the depressors of the epiglottis. As a result of this the voice is rough and unmodulated, owing to the paralysis of the external tensors (crico-thyroid), and the cord is said to have a wavy outline. Owing to the anæsthesia and the position of the epiglottis, food is apt to enter the larynx and lungs. Exposure to cold, diphtheria, and a direct injury to the nerve, are stated to be the causes of this paralysis.

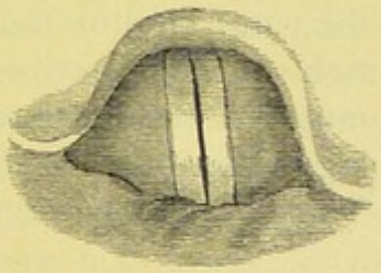


FIG. 39.—Unilateral recurrent Paralysis—phonation (after Ziemssen).

**Paralysis of the Inferior (or Recurrent) Laryngeal Nerve** causes a loss of function in all the intrinsic laryngeal muscles of the side affected, and in consequence the vocal cord is found in the cadaveric position. This paralysis may be due to central changes as in bulbar paralysis, locomotor ataxia, syphilitic and other diseases of the brain. Most fre-

quently the paralysis is caused by pressure on the recurrent laryngeal nerve, or it may be due to a lesion affecting the pneumogastric or spinal accessory.

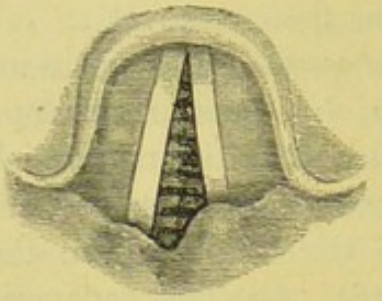


FIG. 40.—Unilateral recurrent Paralysis — inspiration (after Ziemssen).

While the two recurrent nerves are equally liable to suffer from the pressure of such tumours as goitre, cancer of the upper part of the oesophagus, &c., it must be borne in mind that the course of the left nerve round the aorta exposes it specially to injury from the pressure of aortic aneurisms, while the right recurrent is frequently paralysed by being involved in thickening of the right pleura with which it lies in contact—a condition met with in phthisis of the right apex.

**Bilateral Complete Recurrent Paralysis.**—In this rare condition the vocal cords are perfectly immobile, and may be seen to have assumed the cadaveric position; there is absolute loss of voice, and the patient speaks in a whisper, and that with considerable exertion and difficulty, owing to the great expenditure of air on account of the width of the glottis. There is no dyspnœa, but coughing and expectoration become extremely difficult.

**Unilateral Complete Recurrent Paralysis.**—In this condition the vocal cord on the affected side occupies the cadaveric position already described, while the healthy cord has its normal range of movement, and indeed rather exceeds this, even crossing the median line to come in contact with its paralysed fellow. The voice is impure, muffled and high pitched.

### PARALYSIS OF THE INDIVIDUAL MUSCLES SUPPLIED BY THE INFERIOR LARYNGEAL NERVE.

(1.) *Posterior Crico-Arytenoid Muscles.*—These muscles have for their function the opening of the glottis, which is necessary for inspiration. When both are paralysed, a condition ensues,

called double abductor paralysis, which is one of the gravest met with in laryngeal pathology. The two vocal cords are then found to be lying close to each other in the middle line, and from this position they do not move even during inspiration. The consequence is, that there is well marked inspiratory dyspnœa, and this not merely because the cords cannot be drawn asunder by the paralysed muscles, but also because the soft parts of the larynx are forced still closer together, owing to the relatively greater pressure of air above the larynx than in the trachea below, during inspiration. Expiration is, however, comparatively easy, as the internal air pressure is sufficient to slightly separate the cords. Inspiration is, therefore, noisy and laboured, while expiration is comparatively quiet and easy. The voice is usually little affected.

When only one of the posterior crico-arytenoid muscles is paralysed, the vocal cord of the affected side lies in the middle line; the voice is impure, but it is only on exertion that there is any dyspnœa.

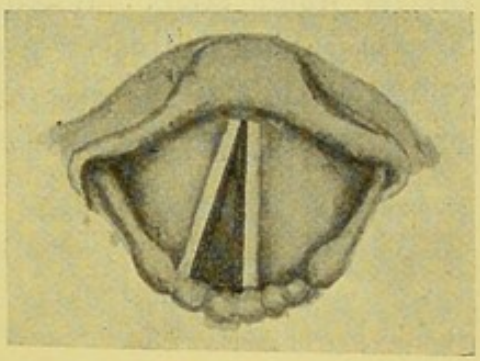


FIG. 41.—Abductor Paralysis on left side —inspiration.

While abductor paralysis has been separately considered as a matter of convenience, it should be clearly understood that, in most cases, it is only the first stage of complete recurrent paralysis. Risien Russell has shown that the abductor and adductor fibres in the recurrent laryngeal nerve preserve a distinct and independent course throughout the whole nerve trunk. Thanks to Semon we know that the abductor fibres are affected sooner than the adductor fibres. It follows then that abductor paralysis is of very considerable diagnostic importance, and indeed it may be the very first symptom to attract attention, and to make the physician suspect the presence of an aneurism or other intrathoracic tumour.

(2.) *Arytenoid muscle.*—This muscle having for its function the closure of the posterior third of the glottis, it will be easily understood that when it is paralysed, both cords lie during

phonation in their normal position for the anterior two-thirds of their length, while at the posterior end of the glottis an open triangle is left through which air escapes unhindered. This little muscle is readily involved in inflammatory mischief of the mucous membrane covering it; and it often succumbs in hysteria.

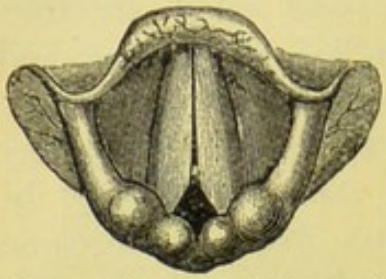


FIG. 42.—Paralysis of Arytenoid Muscle—phonation.

(3.) *Internal Thyro-arytenoid Muscles.*—The action of these muscles is to render the vocal cords tense, and to assist in closing the glottis. When one is paralysed, the cord of the corresponding side on phonation is lax, and slightly concave on its inner edge. When the affection is bi-lateral, this excavation is of course found in both cords, and is often called elliptical paralysis.

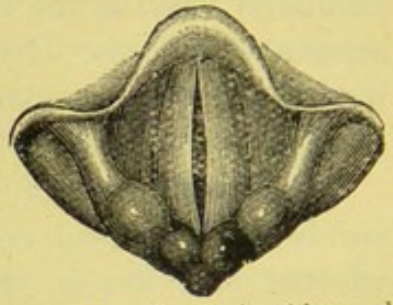


FIG. 43.—Paralysis of internal Thyro - arytenoid Muscles—phonation.

## CHAPTER XVI.

## RESPIRATORY SYSTEM—(*continued*).

### INSPECTION.

In order to determine the position of any particular point on the thoracic wall for the purpose of description or record, the thorax has been divided arbitrarily into certain regions, which may be grouped in the following manner:—

1. *Median or sternal group*, bounded on either side by the sternal border, which comprises—
   - (*a.*) Supra-sternal notch.
   - (*b.*) Superior sternal region.
   - (*c.*) Inferior sternal region. The two last regions are separated by a horizontal line corresponding to the level of the lower border of the third costal cartilage.

2. *Antero-lateral group*, bounded internally by the sternal border, and externally by a line which commences at the first ring of the trachea, runs diagonally outward to the acromion process, and then falls vertically downwards. This group comprises—
   - (*a.*) Supra-clavicular region, lying above the upper edge of the clavicle.
   - (*b.*) Clavicular region, corresponding to the inner half of the clavicle.
   - (*c.*) Infra-clavicular region, from the clavicle to the lower border of the third rib.
   - (*d.*) Mammary region from the third to the sixth rib.
   - (*e.*) Infra-mammary region, from the sixth rib downwards.

3. *The lateral group* corresponds to the axilla, being bounded anteriorly by the vertical acromial line, which limits the antero-lateral group, and posteriorly by the axillary border of scapula. This group comprises—

(*a.*) Axillary region.

(*b.*) Infra-axillary region, which is separated from the former by a horizontal line at the level of the sixth rib.

4. *Posterior group*, bounded externally by the axillary border of the scapula, and internally by the middle line posteriorly. The members of this group are—

(*a.*) Supra-scapular region, lying above the scapula.

(*b.*) Supra-spinous region, corresponding to the supra-spinous fossa.

(*c.*) Infra-spinous region, corresponding to the infra-spinous fossa.

(*d.*) Infra-scapular region, lying below the scapula.

(*e.*) Inter-scapular region, lying between the scapula and the middle line.

## Inspection.

During the inspection of the thorax the patient should be placed in a good light, if possible in a sitting posture, in an unconstrained position, and with the surface of the chest fully exposed. The general outline of the thorax ought to be viewed from the front, from the back, from either side, and from above and behind, looking downwards. Such inspection gives information concerning (1) the form, and (2) the movements of the chest.

1. **The Form of the Chest.**—The typical chest formation, which is, however, but rarely met with, may be said to possess the following characteristics. Conical in form, with the antero-posterior diameter shorter than the transverse, it is symmetrical on both sides, both generally and at each corresponding point. The supra- and infra-clavicular regions are almost on a level

with the clavicles, and from the collar-bones downwards to the fourth rib there is on either side a gentle convexity. The nipple is placed (in the male and virgin female) on the fourth rib or fourth intercostal space, and from this point downwards the chest wall becomes somewhat fattened. In the upper two-thirds of the chest the outlines of the ribs are not well defined, but below this the thinner covering of muscle allows their form

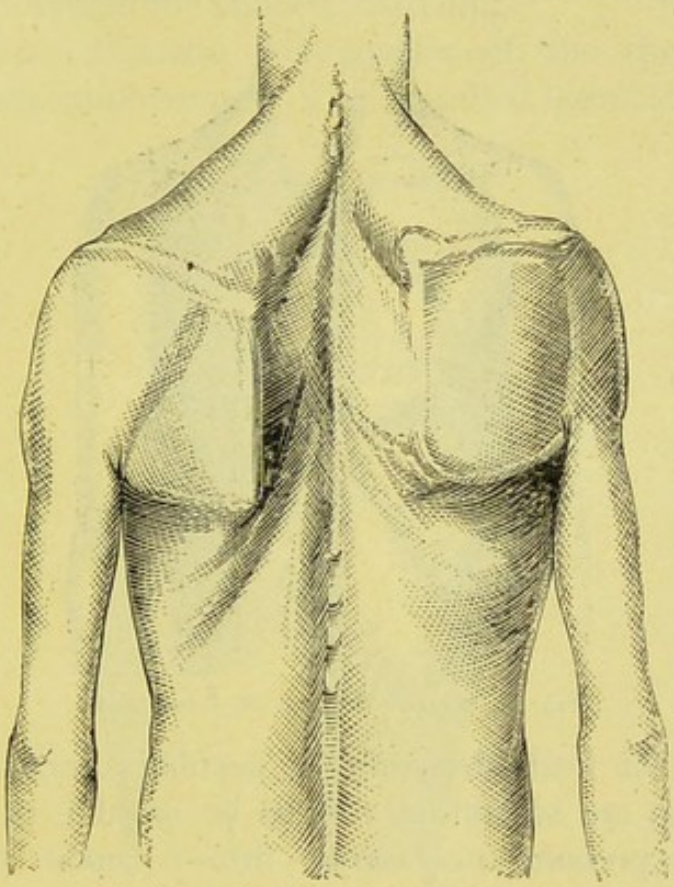


FIG. 44.—Alar Chest. (R. Thompson.)

to become apparent. The spine and sternum occupy an almost exactly median position, and the shoulder-blades are symmetrical.

From this typical form there are many deviations compatible with health (physiological heteromorphisms, as Woillez terms them), of which the principal are those associated with the phthisical and with the rickety constitutions. Many persons who are predisposed to phthisis show a peculiar thoracic conformation which has been called alar, or pterygoid, on account of the wing-like projection of the scapulæ. The chest is long,

narrow, sometimes flattened anteriorly, the ribs oblique, the shoulders sloping, and the throat prominent.

The occurrence of any obstruction to the respiration in childhood, along with rickets, tends to produce the "pigeon-breast," through the yielding of the softened ribs. In this form of thorax the ribs are straightened, and the sternum thrown forwards so that a transverse section of the chest would approach a triangular form. Independently, however, of any pulmonary complication,

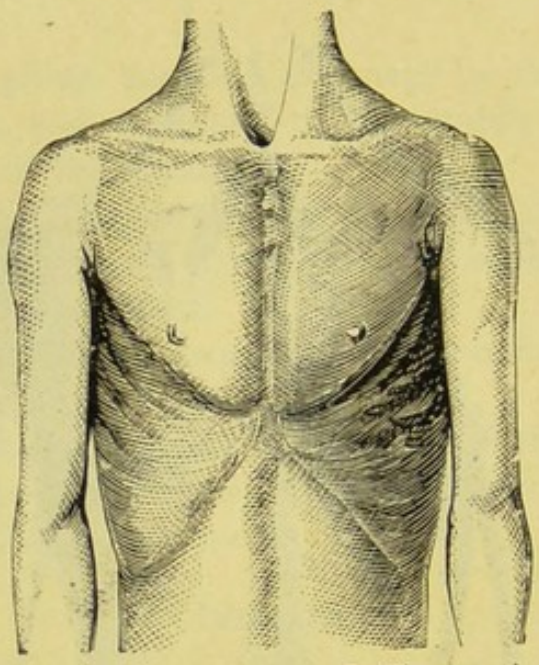


FIG. 45.—Pigeon Breast. (R. Thompson.)

rickets may of itself determine a peculiar thoracic formation, when the ribs are so soft as not to be capable of bearing the atmospheric pressure necessarily thrown upon them during inspiration. A longitudinal groove is thus formed on either side of the sternum.

Irregular formation of the thorax may also be caused by deformities of the spinal column.

*Changes in the Form of the Thorax in Pulmonary Diseases.*

These may be local or general.

(1.) *Local.*—Bulging is met with in encapsuled pleural effusions in empyæma, in pericardial effusions, in cardiac hypertrophy, and over large cavities in the lung. Tumours of the

liver and spleen may also cause bulging, the former at the right side, the latter at the left, and surgical affections of the chest wall may give rise to local swelling. Localised shrinking occurs chiefly in connection with phthisis, when there may be flattening in the supra- and infra-clavicular regions. The rare condition in which there is congenital absence of part of the pectoral muscles must not be mistaken for thoracic flattening.

(2.) *General.*—Bilateral enlargement of the thorax results from pulmonary emphysema. This so-called barrel-shaped chest

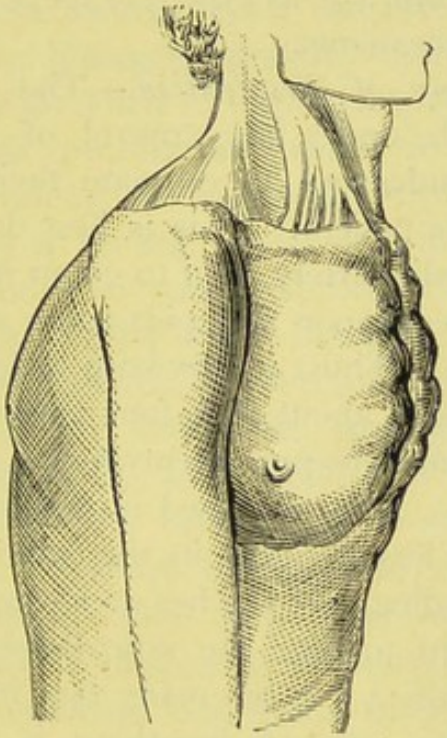


FIG. 46.—Emphysematous Chest. (R. Thompson.)

is enlarged in all its diameters, rounded, and the intercostal spaces wide. The respiratory movements are very slight, and the thorax remains permanently in a condition resembling that of full inspiration. Unilateral enlargement may arise from extensive pneumonia, or from any tumour affecting the greater part of one lung, but it is most evident when effusion of fluid or gas takes place into the pleural cavity. In pleurisy with extensive effusion the diameter of the thorax on the affected side is increased; the intercostal spaces are wide, and rise to the level of the ribs, or even bulge beyond them; the nipple is

moved upwards and outwards, and the heart is pressed over towards the sound side in the manner already described. Unilateral shrinking of the chest may come on as the result of absorption of a pleural effusion when the lung is not in a condition to expand. It is also met with in cases of pulmonary cirrhosis.

2. **Respiratory Movements.**—In connection with the act of breathing we have to note the following points—(1) its frequency, (2) its rhythm, (3) its type, (4) its pain or difficulty, (5) the extent of the movements.

(1.) *The Frequency of Respiration.*—The respiratory movements are so much under the control of the will that the physician should endeavour to estimate their rapidity without the knowledge of the patient. This is best done by holding the fingers upon the radial artery, as if to count its pulsations, while the patient's hand rests upon the epigastrium and rises and falls with the respiration. Whilst in new-born children the breathing is usually at the rate of about forty-four per minute, in the adult male it averages from sixteen to twenty-four, and is slightly more rapid in the female. It is increased in rapidity by exertion, and after meals, and is less frequent in the recumbent posture than when sitting or standing. It reaches its lowest rate during sleep. It is most important to note the ratio between respiration and pulse, which is usually 1 : 4, but many vary from 1 : 1 to 1 : 7.

Pathologically, the act of breathing is rendered slow by stenosis of the larynx or trachea, and by any cerebral disease which interferes with the respiratory centre in the medulla. More common, however, is increase in frequency. This may arise in a variety of ways which will be mentioned in the next chapter, when the subject of dyspnœa is considered.

(2.) *The Rhythm of the Respiratory Movements.*—In health the rhythm of the breathing, when uninfluenced by will, is very regular, expiration following inspiration immediately, and being somewhat shorter in duration, after which there is a short pause. Walshe calculated that if the whole act be taken as equal to 10, then the inspiration may be estimated as 5, the expiration as

4, and the pause as 1. These relations, however, only hold good in health. In disease either the expiration or the inspiration may be altered in duration usually at the expense of the pause. Inspiration is lengthened whenever an obstacle to the entrance of air exists in the larynx or trachea, and this is particularly well marked in cases of paralysis of the posterior crico-arytenoid muscles. Expiration, on the other hand, is prolonged when any obstruction to the exit of air exists in any part of the respiratory tract. The rhythm of the respiratory movements frequently becomes jerking and unequal, particularly in children, where the flexible chest wall yields to the external atmospheric pressure during inspiration, when any obstruction to the free entrance of air exists in the larynx, trachea, or bronchi.

One of the most peculiar alterations in rhythm is seen in Cheyne-Stokes breathing, in which the sequence of the recurring respiratory acts is broken by the occurrence, at intervals of about 1 to 1½ minute, of pauses, during which respiration entirely ceases. These pauses, which last from ¼ to ¾ of a minute, are followed by the gradual resumption of the respirations, which, at first short and superficial, grow gradually deeper up to the point of dyspnœa, after which the breathing becomes again shallower until the next pause is reached, and so on. The exact manner in which this peculiar rhythm is produced is somewhat doubtful. One most important factor seems to be a deficient supply of oxygen to the respiratory centre in the medulla and a consequent depression of its excitability. The Cheyne-Stokes breathing is met with in many cerebral diseases, in uræmia, in fatty degeneration, in valvular disease of the heart, and is usually one of the immediate precursors of a fatal termination.

In meningitis, and other severe cerebral affections, another form of altered rhythm is sometimes seen. It consists in an occasional pause or cessation of respiration, lasting, it may be, for half a minute or longer, and recurring periodically. The difference between this and Cheyne-Stokes breathing consists in the fact that in this variety the breathing commences, after the pause, as an ordinary full breath, and not (as in Cheyne-

Stokes breathing) as a series of shallow respirations gradually growing deeper.

(3.) *The Type of the Respiratory Movements.*—In men the respiratory movements chiefly affect the abdominal walls and the lower ribs (costo-abdominal type), while in women the diaphragm does not take so prominent a part in the act of breathing, and the movement is in great measure confined to the upper part of the thorax (costal type). In disease, however, these conditions may be changed, for anything which interferes with the movements of the diaphragm (such as ascites, peritonitis, and many other affections of the abdomen) will in a man change the type of breathing into the purely costal; whilst the latter type of breathing may be lost in a woman when there is some painful affection of the thoracic organs which obliges the respiration to be chiefly abdominal.

(4.) *Pain and difficulty in Breathing.*—Pain in relation to the organs of respiration has already been mentioned and need not detain us here. When present, it is usually, though not always, aggravated by the respiratory movements. The subject of difficulty of breathing (dyspnœa) will be considered in the next chapter.

(5.) *The Extent of the Movements.*—In the barrel-shaped chest which accompanies vesicular emphysema, there is, as has been already said, diminution of the movements of the chest in all directions. More important, however, for the purposes of diagnosis, are those localised inequalities in the range of the movements which are occasionally met with. When one lung is compressed by reason of pleuritic effusion, or is from any other cause rendered incapable of expansion, the thoracic movements on that side become defective. Phthisical consolidation at the apices gives rise to deficient movement in the upper part of the chest, as compared with the lower; while in cases of stenosis of the larynx and in emphysema, the opposite condition obtains, for then during the expansion of the chest there is depression of the lower intercostal spaces, of the epigastrium, in the supra-clavicular regions, and in the supra-sternal notch.

## CHAPTER XVII.

## RESPIRATORY SYSTEM—(*continued*).

### DYSPNŒA.

**Dyspnœa**, or difficulty of breathing, is one of the commonest and at the same time one of the most important symptoms met with in connection with the act of breathing. It is produced by anything which interferes with the due oxygenation of the blood, and this arises mainly in one or more of three ways: sufficient oxygen may not reach the blood in the capillaries of the lungs; a sufficient flow of blood may not take place in these capillaries; the blood which circulates may be so altered in quality as not to be able to take up enough oxygen for the requirements of the body. Other ways in which the condition arises will be noticed subsequently.

Dyspnœa is, as a rule, both subjective and objective, that is, the patient both feels the need of a more complete oxygenation of the blood and shews the signs of defective aeration. Sometimes, however, we may see a purely subjective dyspnœa, as occasionally occurs in hysteria: at other times the dyspnœa may be purely objective as is seen when it takes place during coma. In the latter case, so long as the patient remains conscious, the dyspnœa shews itself subjectively by his feeling of difficulty of breathing, and objectively by his efforts of increased respiration, and, if these efforts are insufficient, by cyanosis due to the loading of the blood with carbonic acid. But as he becomes unconscious, be it the result of cerebral mischief of another kind, or be it caused by the poisoning with carbonic acid, he loses all subjective feeling of dyspnœa, and one can then only judge of its presence by the laboured breathing and the increasing cyanosis.

The circulation of blood which has not been duly aerated, affects all the tissues. On the respiratory centre it acts as a stimulant and thus produces increased respiratory effort, the breathing either becoming more rapid, or slow and deep. Which of these forms of dyspnœa occurs, depends on the cause. To put the matter shortly, the breathing in any particular case is so altered, as to do the most good with the least expenditure of energy, *i.e.*, to oxygenize the blood to the fullest extent possible without exhausting the nervous and muscular apparatus more than necessary. If by a more powerful effort the obstruction can even for the moment be overcome, then the breathing becomes deep and slow. In other cases, on the contrary, the end is best achieved by rapid and shallow breathing.

The same law governs the relation between inspiration and expiration. In many cases the dyspnœa is inspiratory, *i.e.*, the weight of the effort is thrown on the inspiratory muscles, because the main difficulty is to get the air into the alveoli in sufficient quantity. This is seen in all cases of obstruction of the upper air passages, and in these the long stridor of inspiration may be very striking.[1] In other cases, the main effort is expiratory, because the escape of air from the lungs is in some way interfered with, and hence the expiration is long, laboured and wheezing, as in asthma and in emphysema. In still other cases the dyspnœa is both inspiratory and expiratory. Consequently, the picture of dyspnœa differs somewhat according as one or other, or both, of these conditions is present. In well marked dyspnœa all the accessory muscles of inspiration are called into play, and their contractions form a very striking feature in such cases. These include not merely the dilators of the thorax, such as the sternomastoids, the scaleni, the pectorals, &c., but also those muscles which dilate the nostrils, elevate the soft palate, depress the larynx, and open the glottis. In order to the effective action of these accessory muscles, the patient has to assume the

[1] It should however be borne in mind that there is normally a more rapid and more powerful current of air in the air passages during inspiration, than during expiration, and hence, given the same degree of obstruction, inspiration is always louder than expiration.

sitting posture (orthopnœa) and often grasps the bedstead so as to give the muscles of the chest a fixed point from which to act, and, provided that he is not comatose, the degree of difficulty of breathing which is present may be more or less accurately estimated, according as the position assumed approaches the sitting posture.

Where the dyspnœa is strongly inspiratory and the obstruction great, the action of expanding the thorax tends to produce a vacuum, the air entering with such difficulty and so slowly as not to keep pace with expansion. The result of this is that the atmospheric pressure forces in the more yielding parts of the chest, the lower ribs, the epigastrium, and the tissues above the clavicles. This pressing in of the ribs at the sides of the thorax is specially well seen in children, where these structures are very yielding. Indeed this, as has been already said, (see page 180), is a common cause of certain of the forms of deformation of the chest met with in adult life.

These forced respiratory efforts are of course designed to prevent the blood from becoming venous. In some cases they are successful in doing this, in most, only partially so, with the result that a greater or less degree of cyanosis shews itself.

Many causes, arising from totally different morbid processes, tend to produce dyspnœa, and it should be the aim of the physician, when called to see a case in which this symptom is present, to determine in what particular manner the dyspnœa has been brought about. It will often be found that more than one cause is at work. For example, it is seldom that the cardiac affections producing dyspnœa do not at the same time give rise to pulmonary conditions tending to increase the breathlessness.

The following are the chief ways in which dyspnœa arises :—

(1.) *Dyspnœa from central causes.*—Excitement, as we all know, is liable to cause some acceleration of breathing, and in hysteria and neurasthenia subjective dyspnœa is not uncommon. Various poisons, acting on the respiratory centre, may produce

dyspnœa, as is seen in the uræmic and in the diabetic states, and the heated blood of pyrexia, is of itself a stimulant to the respiratory centre, and gives rise to an increase in the rate of breathing.

(2.) *Dyspnœa from interference with the respiratory movements.*—Anything which curtails these movements may at once bring on dyspnœa. Allusion has already been made to the rapid and shallow breathing which is seen in pleurisy, where, on account of pain, the chest movements are made as limited in their excursions as may be. To compensate for the smaller amount of air which enters with each inspiration, the breathing is, in such cases, correspondingly accelerated.

Much more serious interference with the thoracic movements is seen when the respiratory muscles are paralysed. Affections of the cervical cord, involving the anterior horns, will produce this result, and, if the lesion is bilateral, death is apt to ensue from asphyxia. Tonic spasm of these muscles, such as is seen at one period of the epileptic attack, causes great cyanosis.

Very much the same may be said of the diaphragm. Peritonitis on its surface, or in its neighbourhood, usually causes such pain that the movements are curtailed, and the breathing becomes rapid and shallow. Paralysis of the diaphragm, from central causes or from neuritis of the phrenic nerve, does not, as a rule, cause dyspnœa so long as the patient lies still, the other respiratory muscles sufficing for quiet respiration; the least exertion, however, at once brings on breathlessness. But the diaphragmatic movements may be interfered with by much simpler causes. The upward pressure of abdominal tumours, of ascites, and particularly of tympanites, is often sufficient to limit the respiratory movements and to produce breathlessness.

But, not only may the movements of the thorax be interfered with in the course of disease, but also the excursions of the lungs themselves may be thus limited. This is the case in passive pulmonary congestion, resulting from heart disease, where the great engorgement of the pulmonary capillaries renders the lung tissue, as a whole, stiff, and the consequent limitation of its movements gives rise of itself to dyspnœa.

(3.) *Dyspnœa from obstruction in the air-passages.*—In persons in whom the sensorium is intact, obstruction in the nose such as that caused by catarrh, by polypi, or by adenoid growths, does not materially affect respiration, save in the case of infants in whom, during the act of sucking, the freedom of air play through the nostrils is essential. One of the difficulties in rearing children suffering from that syphilitic affection of the nasal mucous membrane which produces "snuffles," consists in the impossibility of such a child sucking naturally.

In comatose adults, nasal obstruction produced by dried crusts of mucus, may, as Wyllie has shewn to be the case in severe typhus, be of considerable moment. In cases of extreme nervous exhaustion the flapping of the alae nasi, from loss of tone in their muscles, constitutes an impedient to respiration.

In the pharynx, the tongue may fall back on the palate (as in snoring,) and cause stertorous breathing, and in comatose persons, especially in cases of apoplexy, this may produce a considerable degree of respiratory obstruction. The free entrance of air may also be impeded by the presence of enlarged tonsils, adenoid growths, retro-pharyngeal abscess, diphtheritic membrane, and other similar affections.

Much more serious obstruction to respiration occurs in connection with various affections of the larynx. Such obstruction may arise in laryngitis, in oedema of the glottis, in diphtheritic or croupous affections, in spasm of the glottis (as in laryngismus stridulus), from tumours in the larynx itself, or from tumours (particularly aortic aneurism) pressing on the recurrent laryngeal nerve. Perhaps the most serious of all, and one from which fatal asphyxia often arises, is paralysis of the posterior crico-arytenoid muscles. Their function is to keep the glottis open, and when they fail, the chink becomes narrowed in a valvular fashion, the cords being intimately forced together by the air pressure from above. A most serious condition may thus arise.

In connection with the trachea, obstruction to respiration may arise, in some forms of bronchitis, from the accumulation of very abundant mucus. Its lumen may, further, be obstructed by pressure from without. This arises in some cases of medias-

tinal tumour. An aortic aneurism, for example, may press on the trachea near its bifurcation, and it is said that, in cases of heart disease, the distended left auricle may do the same.

In the bronchi, obstruction to the free entrance of air is frequently caused by accumulation of secretion. In the bronchitis of the young, and in that of the aged, this is often a very serious condition. By the pressure of an aortic aneurism, or of the distended left auricle in mitral disease, the left bronchus may be compressed and its lumen seriously narrowed. A very high degree of obstruction is met with in asthma, due, as Roy and the author (Proceedings of the Physiological Society, Journal of Physiology, Vol. XI.) shewed, to spasm of the smooth muscular fibres in the walls of the bronchi. The obstruction in this case is chiefly expiratory.

(4.) *Dyspnœa from diminished alveolar area.*—It is hardly necessary to point out that all forms of exudation into the alveoli of the lungs, whether passive, as in pulmonary oedema, or active, as in pneumonia in its various forms, diminish the air surface used in respiration, and cause more or less dyspnœa. A similar result follows where the cavity of the thorax is encroached on and the lung substance compressed, as occurs in cases of pleuritic effusion and of intra-thoracic tumour. The destruction of many of the alveolar walls, which takes place in vesicular emphysema, produces a like result.

(5.) *Dyspnœa from deficiency of oxygen in the inspired air.*—To some extent the dyspnœa from which mountain climbers suffer, is attributable to the rarity of the atmosphere at high altitudes, larger quantities of air, and consequently deeper breaths being required in order that a normal quantity of oxygen may reach the blood. Similarly, when one breathes a vitiated atmosphere, dyspnœa may arise, though this is in part due to poisoning with carbonic acid.

(6.) *Dyspnœa from defective circulation of the blood.*—Clearly, it may happen that, though a normal quantity of oxygen is reaching the alveoli, the circulation of blood in the pulmonary capillaries may be so slow that oxygen, in quantity sufficient for the needs of the body, is not taken up. Dyspnœa very

frequently arises in this way. All uncompensated valvular lesions, especially those of the left heart, are apt to shew this symptom. But, in many of these cases, not only is there present a more or less constant dyspnœa, but also paroxysms of great breathlessness, occasionally occur, resembling bronchial asthma, a condition usually known as "cardiac asthma." They generally come on during the night, and are, by many, considered to be due to that condition of "lung stiffness" to which allusion has been already made (page 188) as one of the causes of dyspnœa. It seems not unlikely, however, that these attacks may be due to that spasmodic contraction of the bronchi which Roy and the author found (*loc. cit.*) to be produced, to a very marked extent, by defective aeration of the blood.

But, apart from the general disturbance of the pulmonary circulation produced by valvular disease, it should be remembered that a clot lodging in the pulmonary artery, or in one of its main branches, will, by suddenly limiting the flow of blood over the respiratory surface, produce a greater or lesser degree of dyspnœa.

(7.) *Dyspnœa from changes in the quality of the blood.*—Plenty of oxygen may reach the alveoli, and the circulation of blood in the pulmonary capillaries may be unimpaired, and yet dyspnœa may arise from the side of the blood. This occurs when the red corpuscles are either abnormally few in number (as after hæmorrhage, or in diseases involving their destruction), or are so altered as to lead to impairment of their power to take up and carry oxygen. The latter condition is well seen in cases of chlorosis and in leucocythemia, where the dyspnœa may be considerable.

(8.) *Dyspnœa from increased metabolism.*—The last cause of dyspnœa which seems to call for mention here, is that which comes into action when the metabolism of the body is greatly accelerated. More oxygen is then required for the needs of the body, and consequently the respiratory efforts are increased. The breathlessness of exertion is in some measure of this causation, and the acceleration of breathing in conditions of pyrexia is also to a certain extent due to this cause.

## CHAPTER XVIII.

## RESPIRATORY SYSTEM—(*continued*).

### PALPATION.

By laying the hand flat upon the thorax and palpating its walls information may be obtained regarding the form and movements of the chest, the presence or absence of fremitus, of fluctuation, and of certain pulsatory movements other than those already referred to in connection with the circulatory system.

1. **The Form of the Thorax.**—The general form of the chest is best appreciated by means of simple inspection; but localised changes in shape may also be recognised by palpation.

2. **The Movements of the Thorax.**—The information obtained by inspection may be supplemented by laying the hands on the thorax, and estimating the local movements of expansion and elevation at particular parts.

3. **Vocal Fremitus**, or that vibration of the chest wall which may be felt in a healthy person while speaking, is of considerable diagnostic importance. Under the vocal cords in the larynx lies an air column, which extends through the trachea and bronchi to the pulmonary alveoli, and which is set in vibration when the vocal cords vibrate. Through the bronchial walls and lung tissue the thrill so generated is conducted to the thoracic parieties. It is not difficult to understand the conditions under which its intensity becomes increased or diminished. The thickness of the thoracic wall has an important influence, the thrill being more distinct in emaciated

subjects than in those who have much deposit of fat underneath the skin. The intensity of the vocal fremitus also depends upon the loudness of the tone spoken, and upon the depth of its pitch; and finally, it must not be forgotten that it is more distinct in men than in women and children, and that the thrill on the right side is almost invariably greater than that on the left; this being accounted for by the larger calibre of the right bronchus.

In disease the vocal fremitus may be diminished or increased.

(1.) *Diminished.*—Any condition which blocks up the bronchi, such as collection of mucus or pus, or compression by means of tumours, will produce a diminution or a loss of the vocal fremitus over the corresponding part of the chest wall. More marked loss of the thrill is met with where effusion of fluid or gas into the pleural cavity has taken place. If the effusion be extensive, the vocal fremitus may be entirely lost; and should the lung be bound down by extensive adhesions, the fremitus may not be regained even after the entire absorption of the effusion. Thickening of the pleura, from old pleurisy, is a common cause of diminution or loss of vocal fremitus.

(2.) *Increased.*—When infiltration takes place into the air-cells of the lung, the pulmonary parenchyma becomes at once a better conductor of the vocal vibrations, and in consequence the thrill becomes intensified. Such is the case, for example, in lobar pneumonia; and where the lower lobe is affected, the vocal fremitus gives most important aid in distinguishing that affection from pleural effusion. The fremitus is likewise increased where there is phthisical consolidation, particularly if cavities have formed; but should a main bronchial branch leading to the part have become obstructed, either by pressure or by the collection of mucus, pus, or blood, the vocal thrill may be diminished or lost. In the same way, a mediastinal tumour may be so placed as to conduct the bronchial vibrations directly to the chest wall, and thus give rise to an increase of the vocal fremitus.

4. **Pleural, Bronchial, and Cavernous Thrills.** — The pal-

pating hand may also detect the fremitus occasioned by the rubbing together of the roughened pleural surfaces in cases of pleurisy, and large rales in the bronchi or in cavities in the lungs may communicate an appreciable thrill to the walls of the chest. These are, however, of little diagnostic importance, in that they are better appreciated by the aid of auscultation.

5. **Fluctuation.**—When one side of the thorax is distended with fluid, fluctuation may occasionally be detected in it, more particularly and importantly in empyæma. In this latter condition there is often some inflammatory oedema in the chest wall.

# CHAPTER XIX.

## RESPIRATORY SYSTEM—(*continued*).

### MENSURATION.

MENSURATION, which is intended to render precise the information which may be gained by inspection and palpation, and which in some of its devolopments passes much beyond these, is performed by the aid of a variety of instruments which fall to be described in detail.

1. **The Tape-Measure** is used to ascertain the circumference of the chest, which, at the level of the nipples, at the end of expiration, measures in the healthy male adult about thirty-two or thirty-three inches, varying however considerably in different individuals. A full inspiration increases the figure by from two to five inches, while in quiet respiration the inspiration position exceeds the expiration by about half-an-inch. Unfortunately the circumferential measurement of the chest is of comparatively little diagnostic value, as very great variations are met with in health. Much more important is it to learn the relative size of the two sides of the chest. This is most conveniently done by joining two tapes at the commencement of their scales, and fixing this point of junction over the vertebral column. Each side of the chest has thus a tape for itself, and the two measurements can be simultaneously made and compared. In right-handed persons the right half of the chest is about half-an-inch larger in circumference than the left; while in those who are left-handed these conditions are either reversed, or, what is more common, the two sides of the chest are practically identical in size. Unilateral enlargement and shrinking

the result of disease, are very readily detected by means of such measurement.

2. **Callipers.** — Various diameters of the chest may be measured by means of a pair of common steel callipers. Of these the most important is undoubtedly the antero-posterior (sterno-vertebral), which in the phthinoid chest is much diminished—the normal measurement being 9 to 10 inches. It is more difficult to obtain exact measurements of the antero-posterior diameter of either apex. For this purpose, one point of the callipers is to be applied immediately below the centre of the clavicle, and the other on the spine of the scapula at a similar distance from the middle line. If great care be taken, sufficiently reliable results may in this way be obtained, when it will be found almost invariably, that in healthy persons the right measurement very slightly exceeds the left. According to Walshe, an excess of even a fourth of an inch on the right side indicates, however, morbid depression on the left; while, if the left be in excess by that amount, there is still more conclusive evidence of contraction on the right side.

3. **Cyrtometer.**—This instrument, devised by Woillez, consists of a series of small pieces of whale-bone, so articulated together as to form a stiff chain which, when closely applied to the walls of the thorax, retains the curves given to it, and which, when removed and laid upon a large sheet of paper, permits of these curves being marked out on the paper. The instrument may be more simply constructed of two pieces of lead wire, joined together by means of a piece of india-rubber tubing, and in this form it is easily used.

4. **The Perigraph.**—The author has recently (see *The Scottish Medical and Surgical Journal*, July 1897) devised this instrument for the purpose of obtaining tracings of the outline of the thorax, more reliable than the cyrtometer affords, and also tracings taken in planes where the cyrtometer cannot be applied. The instrument depends on the principle of parallel motion, that

which underlies all varieties of pantograph. The ordinary form of the pantograph may be gathered from Fig. 47, where, if F be a fixed point round which the rods may turn, then, when a style fixed in C is moved over a drawing, a pencil in D will delineate a copy, which, when the relative lengths of the rods are as represented in the diagram, would be half the natural size.

It is quite clear, however, that to make this principle of use in delineating the shape of the thorax, some considerable modifica-

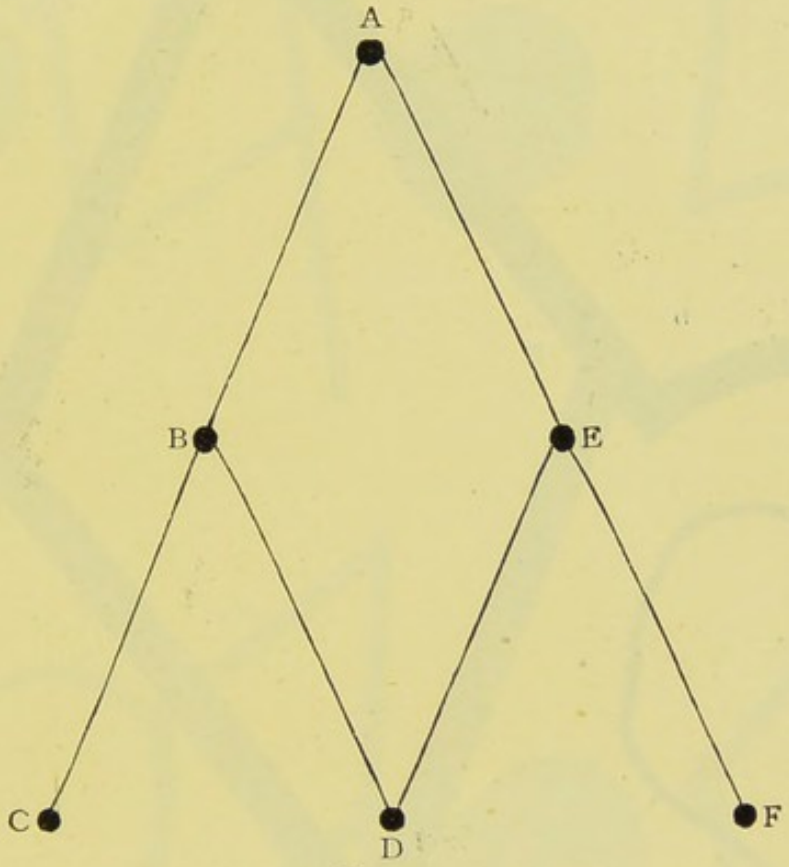



FIG. 47.

tion is necessary in order to enable the point C to move round a more or less cylindrical surface, and this result was attained by substituting for the straight portion B C a sickle-shaped piece of brass, pivoted at B and capable of rotating as will be described.

The instrument, so modified, is shown in Fig. 48. The sickle-shaped portion, constructed of brass, passes from B to C, and is pivoted at b in such a manner that it can be rotated round that centre through an angle of 180°, while the point C continues to lie in the prolongation of the axis of A B. The point F is fixed,

and D bears a pencil which records the motions of the instrument on a sheet of paper placed beneath. Hence, if the point C is made to move round the thorax, the pencil at D will trace the chest form. This instrument, the author has named the Perigraph, or round writer.

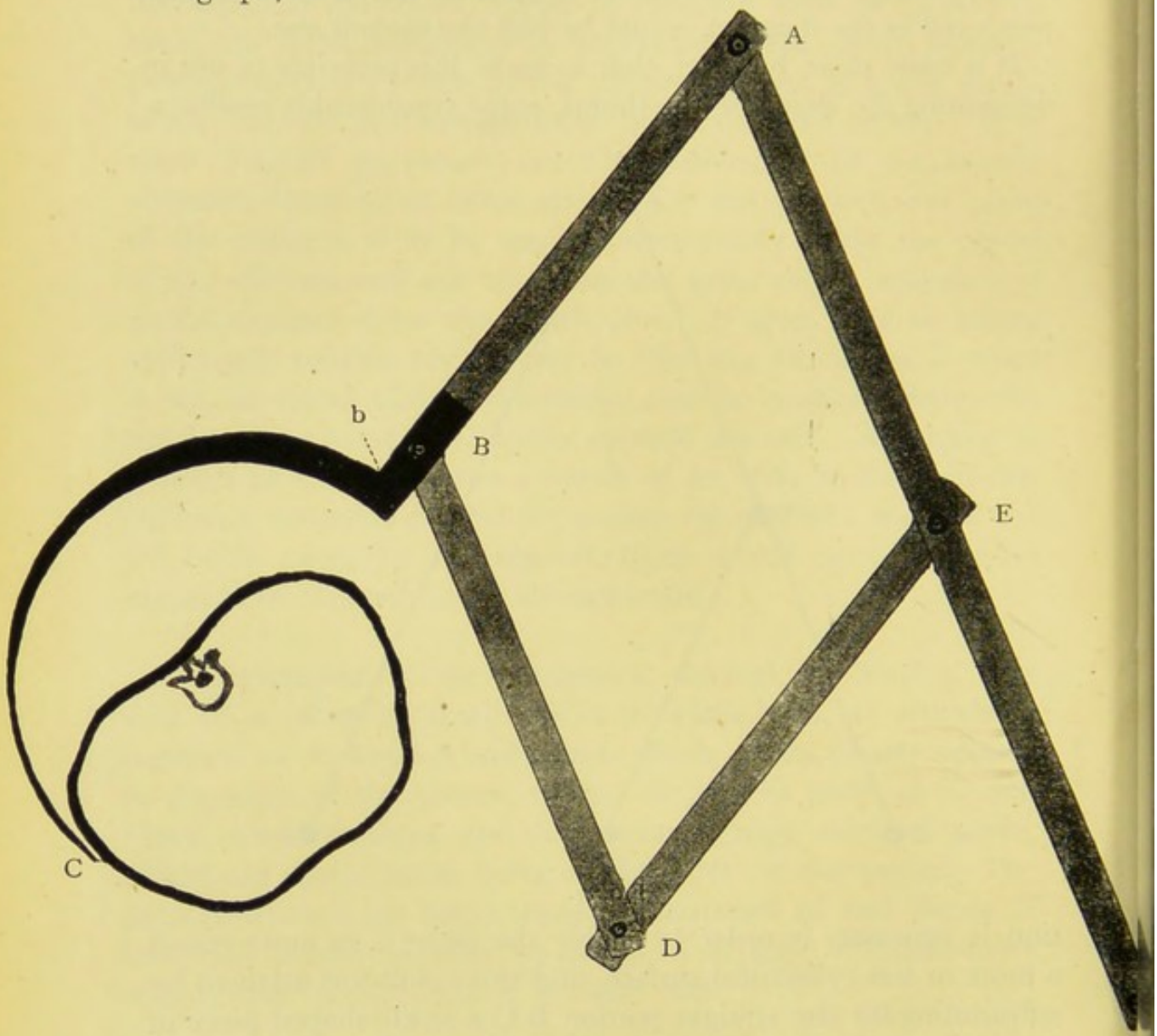



FIG. 48.—The Perigraph.

A glance at Fig. 49 will make clear the motion of the perigraph in its various phases as it passes round the wall of the chest. Beginning in the position marked (1), the point is made to sweep over the skin through the position (2), until it reaches, at (3), the point diagonally opposite that at which it commenced. The

sickle is then rotated through 180° to assume the position indicated by the dotted lines, and the point is moved onwards, through (4), until it completes the circumference of the thorax and arrives at the point from which it started.

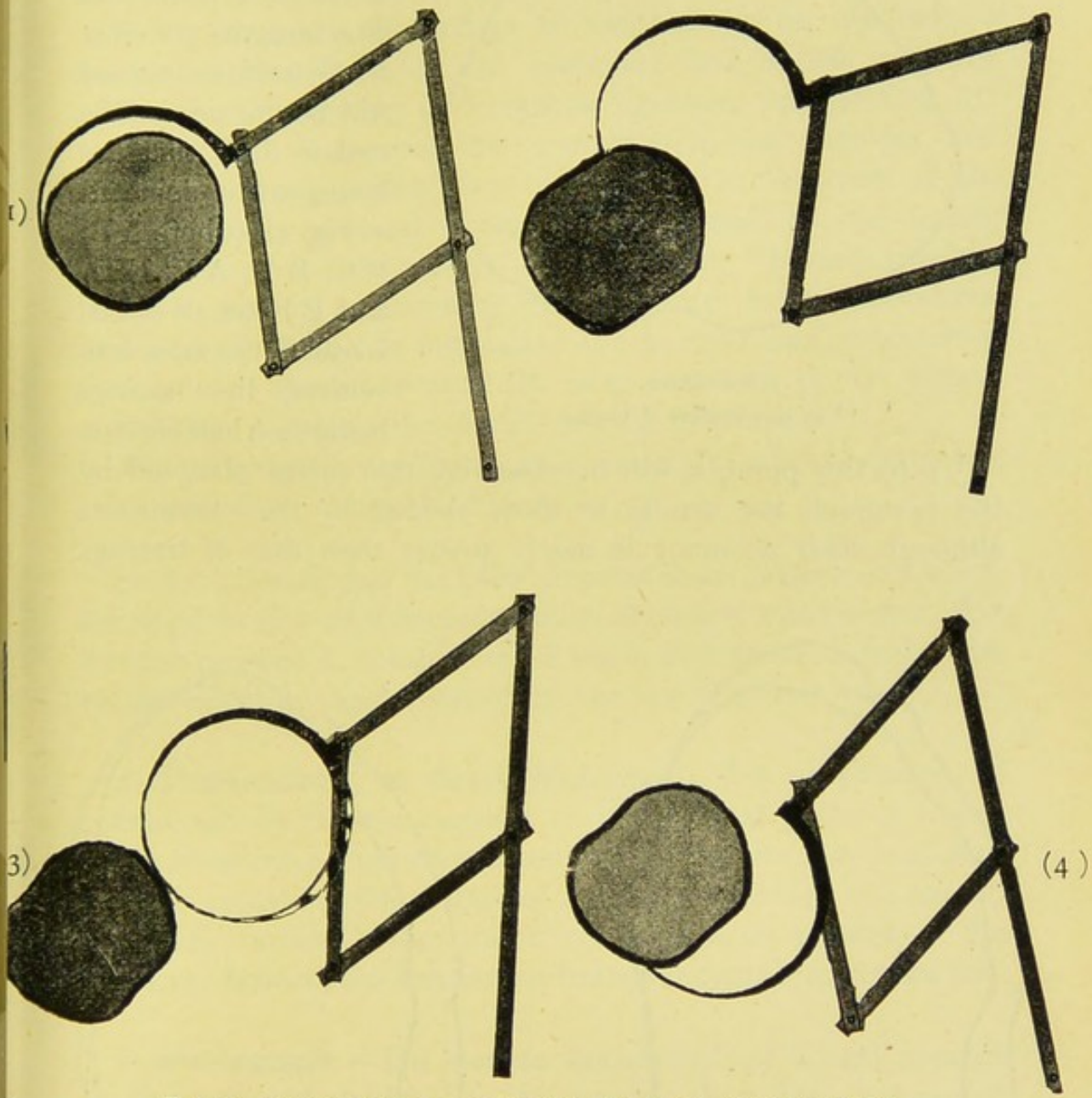


FIG. 49.—Diagram of the movements of the Perigraph, round the Thorax.

A reduced diagram of the shape of the thorax in a very muscular subject, obtained in this way, is shown in Fig. 50.

By varying the relative lengths of the sides of the parallelogram A B D E (in Fig. 48), for which provision is made in the con-

struction of the instrument, the relation of the size of the drawing to the actual size of the thorax may be varied. By making D the fixed point, and attaching the pencil at F, the tracing obtained will be life size. The author finds that the arrangement depicted in Fig. 48 (where A B, B C, B D, A E, E D, and E F are all equal) is usually the most convenient, the tracings being then half life size.

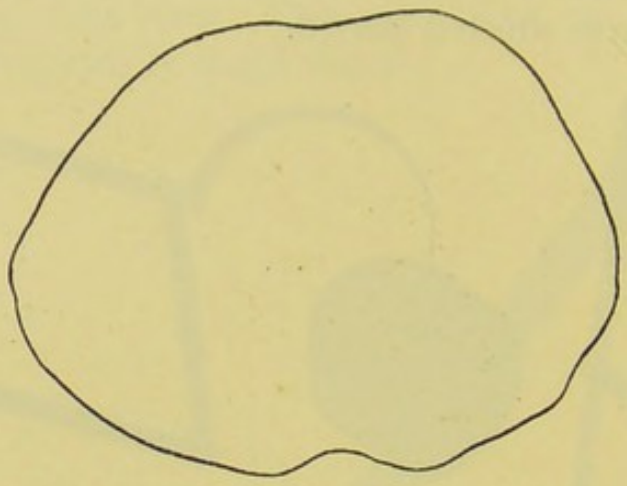



Fig. 50.—Outline of Thorax.

Up to this point, it will be observed, the curves obtained by the perigraph are similar to those yielded by the cyrtometer, although their accuracy is much greater than that of tracings

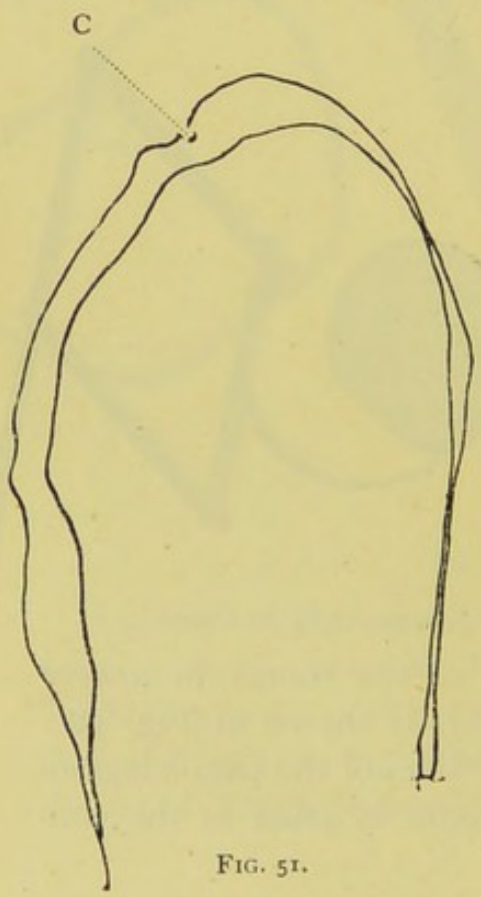



Fig. 51.

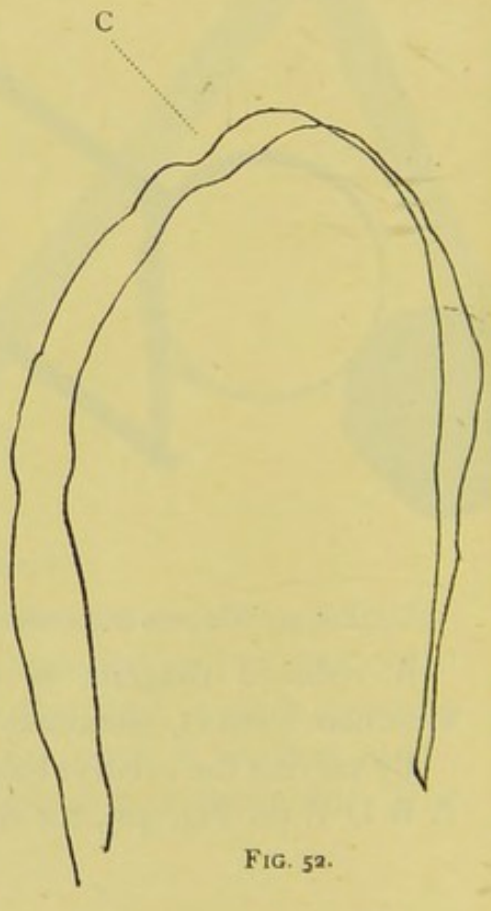



Fig. 52.

obtained by the latter instrument. After the perigraph had been constructed, however, it was found that it was capable of giving outlines of the chest shape, in planes other than the horizontal, at once more interesting and more useful, and such as the cyrtometer could not yield.

If, for example, the patient be made to lie on one side, a tracing of the shape of the abdomen and thorax may be obtained, commencing at Poupart's ligament, and passing upwards in the parasternal line over clavicle and shoulder, and down the corresponding line of the back to the crest of the ilium. Such curves are given in Figs. 51 and 52, the former from a more muscular subject than the latter. In each case the curve is double, representing the difference, in the abdominal and thoracic outline, in inspiration and in expiration respectively. It may be of interest to note the deep insinking of the tissues, at the point C, above the clavicle during inspiration.

The perigraph is applicable to a variety of clinical purposes, other than those above described.

So far, a description has been given of those instruments which are fitted to give us measurements of the chest when at rest, and we now proceed to consider those which have for their design the measurement or registration of the thoracic movements.

5. **Thoracometer, or Chest Measurer.**—This instrument, as constructed by Sibson, consists, in its essential parts, of a dial which measures accurately the vertical movements of a small rod, which is applied to the surface of the chest by means of a spring. Owing to various errors which are necessarily present in the readings obtained, this instrument has never come into general use.

6. **Stethograph.**—The double stethograph of Riegel appears to be more trustworthy in its results than the last-named instrument. Two levers, which are acted upon by the movements of the chest walls at two different points, are arranged so as to record their results on a strip of paper, travelling horizontally by clockwork. The tracings so obtained enable us to

analyse the respiratory movements in a much more exact manner than can be done by means of any other instrument. Pathologically, the most striking changes are those in which there is an impediment to the free entrance or exit of air. For example, where the larynx or trachea is stenosed, while the expiratory curve is normal the inspiratory is much prolonged. The reverse is the case in emphysema, where the expiratory curve is prolonged and irregular.

We now come to the third class of instruments of mensuration, those, namely, which deal with the air passing into and out of the chest.

7. **Spirometer.**—Hutchinson's spirometer consists of a gasometer properly poised and adjusted, into which the patient expires forcibly through an elastic tube, and which is arranged so as to measure the amount of expired air. The "vital capacity" varies with age, stature, and sex, but when allowances have been made, it may be said, as a general rule, that a diminished quantity of air is expired where there is stenosis of larynx, trachea, or bronchi, interference with the free movement of the thoracic walls, or diminution of the respiratory surface of the lungs. Of these diseases, undoubtedly, the most striking in its results is phthisis.

Hutchinson gives the following table of the results he obtained from very numerous observations:—

| Stature. | Capacity of Healthy Males. | Early Stage of Consumption. | Advanced Stage of Consumption. |
|---|---|---|---|
| | Cubic Inches. | Cubic Inches. | Cubic Inches. |
| From 5 feet to 5 feet 1 inch, | 174 | 117 | 82 |
| ,, 5 ,, 1 inch to 5 ,, 2 ,, | 182 | 122 | 86 |
| ,, 5 ,, 2 ,, to 5 ,, 3 ,, | 190 | 127 | 89 |
| ,, 5 ,, 3 ,, to 5 ,, 4 ,, | 198 | 133 | 93 |
| ,, 5 ,, 4 ,, to 5 ,, 5 ,, | 206 | 138 | 97 |
| ,, 5 ,, 5 ,, to 5 ,, 6 ,, | 214 | 143 | 100 |
| ,, 5 ,, 6 ,, to 5 ,, 7 ,, | 222 | 149 | 104 |
| ,, 5 ,, 7 ,, to 5 ,, 8 ,, | 230 | 154 | 108 |
| ,, 5 ,, 8 ,, to 5 ,, 9 ,, | 238 | 159 | 112 |
| ,, 5 ,, 9 ,, to 5 ,, 10 ,, | 246 | 165 | 116 |
| ,, 5 ,, 10 ,, to 5 ,, 11 ,, | 254 | 170 | 119 |
| ,, 5 ,, 11 ,, to 6 ,, | 262 | 176 | 123 |

It must, however, be borne in mind that there are many fallacies in the use of this instrument. Some persons cannot be made to understand how to blow, others by taking great pains attain to higher figures than their average, and finally, by practice, the art of blowing is so readily learned that those accustomed to the instrument can raise the gasometer cylinder to very considerable elevations.

7. **Pneumatometer.**—This instrument, by means of which the force of expiration and inspiration is measured, is most conveniently used in the form devised by Waldenburg, which consists of a simple mercurial manometer connected by means of an elastic tube with a mouthpiece that fits accurately and tightly over the mouth and nose of the patient. When the patient expires through the tube, the column of mercury sinks in the proximal limb of the manometer and rises in the distal, while with inspiration these movements are of course reversed, and in either case the amount of displacement is to be read off on the scale. In a moderately well-developed male the inspiratory pressure is from 60 to 70 mm. of mercury, while the expiratory is from 80 to 90 mm. In the female the pressure in both cases is considerably less. Of more importance than the absolute pressure (which varies much in different individuals) is the difference between the expiratory and the inspiratory pressure, and it must be carefully borne in mind by those who use this instrument, that in healthy persons the power of expiration exceeds that of inspiration by about 20 mm. It is by comparing these two pressures that the most important indications are obtainable. Their relation is altered in disease as follows :—

*Expiratory pressure is increased* in relation to inspiratory, in phthisis (at an early stage), in stenosis of the air passages, in pulmonary congestion, pneumonia, and pleurisy, and in such abdominal affections as impede respiration by pressing the diaphragm upwards.

*Expiratory pressure is diminished*, until it becomes equal to or below the inspiratory, in pulmonary emphysema.

Space does not permit of the description of various other, less important, instruments.

# CHAPTER XX.

## RESPIRATORY SYSTEM—(*continued*).

### THEORY OF PERCUSSION.

WHEN the surface of the chest is percussed in the manner which will be described in the next chapter, a sound is produced which is called the percussion note of the part. This term *note* is, however, apt to mislead, for it is not a simple or pure note, not being composed of a regular series of simple vibrations. Nor is it (as is the case with the sounds produced by musical instruments) made up of one well marked basal or fundamental tone and a series of higher pitched upper partial tones which bear a definite relation to the basal or prime tone. The sound which is heard on percussion of the healthy chest is composed of a large number of tones, bearing no distinct relation to one another, and in it no definite or well marked fundamental note can be distinguished. It is very often assumed that the note produced on percussing the front of the chest—for example, at the level of the second rib—is made up of a distinguishable fundamental or prime tone, corresponding in pitch to the antero-posterior diameter at the particular point in question, just as when one blows across the mouth of a test-tube the prime tone obtained corresponds in pitch to the length of the air column contained in the tube. This view is, however, clearly untenable; for, apart from the inherent improbability of this particular air column alone being set in audible vibration, and not the many others which radiate from the point of percussion to the other limits of the thoracic cavity, there is the fact that in practice it is impossible to find what is the real pitch of this fundamental or prime tone; and, further, it is constantly noticed that the apparent

pitch of the percussion note varies enormously with the variety of pleximeter employed, and still more when the pleximeter and finger are compared, which would not be the case were there a distinguishable prime tone.

The percussion note is made up of vibrations which are derived from three sources.

1. *The Vibrations of the Pleximeter.*—When the finger is employed as a pleximeter, these vibrations are practically inaudible. In the case of an ivory pleximeter, however, they are readily recognised. If the instrument be of the usual form, the vibrations are clear and relatively high in pitch; but provided that the pleximeter be properly damped by being firmly pressed upon the thoracic wall, and be struck with the pulp of the finger alone, or with the india-rubber of a hammer, the tone it gives can be readily discounted by the physician.

2. *The Vibrations of the Thoracic Wall.*—These are of so ill-marked a character (unless the point struck lie over the rib of a very thin subject) and have so little intensity as compared with the intrathoracic note, that in themselves they need hardly be considered, though, as will be presently pointed out, the condition of the chest wall and its vibrations when percussed have a very important influence on the character of the intrathoracic note.

3. *The Vibrations of the Air in the Lungs.*—These vibrations constitute the important part of the percussion note, and must be considered in some detail.

When percussion is made at any point of the chest wall, the air in the lungs is set in vibration, and the point which is struck may be considered as the point of divergence of a series of radiating air columns whose lengths may be represented by lines drawn from the corresponding point on the visceral pleura to the opposite walls of the thorax in all directions. The lengths of these very numerous columns, differ of course, considerably; and since an air column, when set in vibration, produces a note proportionate in pitch to the length of the column, the numerous notes which go to make up the percussion sound vary considerably in pitch. The pulmonic septa also, in all probability, limit

the length of certain of these air columns, and in others they may determine nodal points, and in this way cause still greater differences in pitch. We have thus to consider the intrathoracic percussion sound as composed of a large number of prime or fundamental tones, which vary much in pitch; and as each has an ascending series of upper partial tones, this tends, of course, still further to render the vibrations of the combined percussion note irregular.

In a musical note we have to recognise three distinct characters—viz., intensity, pitch, and quality; and in relation to the percussion sound, these must also be considered.

1. *Intensity.*—The intensity of a musical tone depends upon the amplitude of the individual vibrations of which the tone is composed. In the case of the drum, for example, the intensity of the tone depends upon the vigour with which the drum-head is struck. In the same way, the intensity of the percussion-note depends, to a considerable extent, upon the strength of the stroke. But it must be remembered that the percussion note, as has just been said, is composed of a large number of different tones, so that its intensity in any given case depends also upon the number of these tones which are produced by the blow. Thus, when the greater part of a lung is hepatised, the percussion note over the healthy portion loses much of its intensity, because there are fewer air columns which can be set in vibration. The intensity of the note is, therefore, of considerable diagnostic significance.

2. *Pitch.*—The pitch of a simple tone, such as that of a tuning-fork, depends upon the rate of the vibrations of which it is composed. In the case of a musical note, composed of a prime tone and an ascending series of upper partial tones (as, for example, the note of a stretched cord), the term pitch is understood to mean the pitch of the prime tone. It is thus clear that, in regard to the percussion sound, we cannot, properly speaking, use the term pitch, since it is impossible to detect any fundamental or prime note. It is evidently advisable, however, to retain the term, which is so useful clinically, provided that in using it we carefully keep in mind that we do

not refer to the pitch of a basal note, which, as has been said, does not exist, but to the general pitch of the combination of tones which reach the ear. If we take an illustration from the piano, it will easily be seen what is here meant. Suppose that a number of notes at the treble end of the key-board be struck simultaneously with one or two at the bass end, the general impression will be that of a high pitched sound, and *vice versâ ;* and so also in the case of the percussion sound, if the number of higher tones preponderates greatly over that of the lower tones, the general sound appears to be high in pitch, and *vice versâ.*

It has been said that when the chest wall is struck, the underlying air columns are set in vibration. This is due in great measure to the direct transmission of the impulse, but in some degree, at any rate, these vibrations seem to arise by sympathetic resonance. This demands a few words of explanation.

If a series of tuning-forks, of different pitch, be in turn sounded over the mouth of an empty jar, it will probably be found that the series contains one fork to which the air in the jar, so to speak, answers,—which, when it is sounded, throws the air column into sympathetic vibration, so as to reproduce and strengthen its own note. If the air column be measured, it will now be found that its length is exactly one-fourth the length of the sound-wave produced by the fork in question. This reproduction and reinforcement of the tone is termed resonance. In the same way if a compound tone be sounded in the neighbourhood of such an air column it will be set in sympathetic vibration if the sound-wave of the prime or any of the upper partial tones happens to bear the relation to the length of the column which has just been stated. Now, when the chest wall is percussed it vibrates, as has been already pointed out, and gives rise to a sound which is usually inaudible, and which, in any case, is of little importance. But underlying it there are numerous air columns, certain of which are of a suitable length to be set in sympathetic vibration by certain of the tones of which the sound of the thoracic wall is composed ; so that the quality and general pitch of the intrathoracic note does to a certain extent depend upon the vibrations of the chest wall.

The slight difference in pitch of the percussion sound during expiration and inspiration is thus to be explained. When the chest is in the condition of full inspiration its walls are tenser than during expiration, and, therefore, give a higher pitched note when percussed, which note is reproduced and strengthened by the resonance of the intrathoracic air columns, and thus raises the general pitch of the percussion sound.

The pitch, then, of the percussion sound at any given point depends upon the length of the air columns which are set in vibration, whether that vibration be produced by direct impulse or by sympathetic resonance; and the general pitch of the compound percussion sound depends upon whether the high or low pitched notes are of larger number or of greater intensity. But, as I have already said, the length of some at least of these columns is determined by limiting pulmonic septa. If the lung tissue become relaxed, these septa no longer limit the length of the air columns, which then extend back to the opposite wall of the chest, and consequently give a lower pitched note. Thus, as a whole, the percussion sound depends for its pitch upon three factors—(1) the tenseness of the chest wall; (2) the tenseness of the lung tissue; (3) the length of the underlying air columns.

3. *Quality.*—The quality or *timbre* of a musical note depends upon the number and character of its upper partial tones. Enough has been said in the last pages to explain how this applies to the compound percussion sounds, and the various well-marked qualities which are to be met with clinically will be best discussed and explained in the next chapter, when we come to deal with the practical aspects of percussion.

## CHAPTER XXI.

## RESPIRATORY SYSTEM—(*continued*).

### PERCUSSION OF THE CHEST.

In the preceding chapter an attempt has been made to explain the theory on which the practice of percussion rests. It is now necessary to consider it in its clinical and practical aspects. And first, of the

**Methods of Percussion.**—There are two varieties of percussion, the immediate and the mediate.

*Immediate percussion*, or that in which the chest wall is struck directly with the finger, was the method originally employed. It is now almostly completely discarded, the only exception being the percussion of the clavicles, which may with advantage be struck with the pulp of the forefinger before the percussion of the chest generally is commenced.

*Mediate percussion*, or that variety in which the finger or pleximeter is laid upon the chest wall and receives the stroke, is now almost universally employed.

As a general rule, it is probably best to use one or more of the fingers of the right hand to give the stroke, and to employ the fore or middle finger of the left hand as a pleximeter, applying its palmar surface firmly to that portion of the chest wall which we wish to percuss. For the right hand, a percussion hammer may be substituted, and for a pleximeter we may employ an ivory or vulcanite plate or any of the numerous forms which are to be found in the shops of surgical instrument makers.

Whether the stroke be delivered by means of the bent finger or the hammer, it should be given from the wrist alone, and not

from the shoulder or elbow, and the fingers or hammer should be raised from the pleximeter the moment the blow has been given, so as to allow of the free vibration of the chest. Skilful percussion with the fingers is very difficult to acquire, and requires long practice; but all students should learn it, for although hammer percussion is much easier, the physician may often be in circumstances when he is compelled to percuss without the aid of that instrument. Finger percussion is also much better suited to give the feeling of resistance which, as will be presently shown, is often of considerable importance.

It is of great importance that the chest should be percussed symmetrically, corresponding points on both sides being compared with one another, and it is necessary to see that the patient assumes no position of head, limbs, or trunk which will produce unequal muscular tension on either side.

As a rule, percussion need not be very forcible, though when the chest walls are thickened from the deposition of fat, or are dropsical, a strong blow may be necessary in order to produce a sufficiently audible note.

**The Thoracic Percussion Note.**—In the preceding chapter the author has indicated the theoretical basis on which he believes the practice of percussion may safely be held to rest; but whatever their theoretical beliefs, most physicians will agree that the percussion sound depends mainly upon three factors—viz., (1) the thickness and tension of the chest wall, (2) the tension of the pulmonic tissue, and (3) the amount and disposition of the underlying air; and that it is to physical changes in these three factors that we must look for the cause of variations in the percussion note.

In the following pages we shall consider firstly, the changes in the percussion note which occur, as regards (*a*) intensity, (*b*) pitch, and (*c*) quality (such as the tympanitic note, cracked-pot sounds, &c.); secondly, the feeling of resistance during percussion; and thirdly, the topographical percussion of the lungs.

**The Intensity of the Percussion Sound.**—As has been already

said, the intensity of a simple pendular tone depends upon the amplitude of its vibrations; but in regard to the compound percussion sound, account must also be taken of the number of air columns which are thrown into vibration, and this depends upon the force of the stroke, upon the condition of the chest walls, and upon the volume of underlying air. Remembering that when two parts of the chest are being compared the force of the stroke must in each case be equal, if we are to obtain comparable results, we may limit our attention to the two last factors.

1. *The Condition of the Chest Wall.*—When the thoracic wall is thickened by the deposition of fat, by the transudation of serum into its interstices, by thickening of the pleura, or by any other cause, the subjacent air is thrown with greater difficulty into vibration by the percussion stroke, and the resulting sound is deadened in passing through the thickened chest wall to reach the ear of the physician. The same diminution of the intensity of the sound occurs in health over those portions of the chest which are covered with thick muscle—for example, the scapular regions, and over the pectoralis major; and it should be borne in mind that in labourers in whom, from their occupation, the right pectoralis is considerably more developed than the left, the percussion note is less intense on the right side over that muscle than at a corresponding point on the left. Pleural effusions, also, have the same effect on the intensity of the note, as the layer of fluid prevents the free transmission of the percussion stroke. Such collections of fluid have, of course, a further influence on the note from the compression of the lung tissue which they occasion. The pleural thickening, which remains after an attack of pleurisy, tends to diminish the intensity of the percussion note, partly owing to the increased thickness of the chest wall thereby produced, but also from the manner in which the strong adhesions formed tend to contract, and so bind together the chest wall as seriously to interfere with its free vibration.

2. *The Amount of Air contained in the Chest.*—The intensity of the chest note is diminished whenever, from any cause, there

is a serious diminution of the air contained in the chest. This may result either from compression of the lung tissue, and consequent expulsion of the air, such as takes place in cases of pleural effusion and of tumours pressing upon the lung; or from infiltration into the alveoli, such as occurs in pulmonary œdema, in the exudative stage of acute pneumonia, and in all the forms of chronic phthisis.

An *increase* in the intensity of the percussion sound is met with (1) where the chest walls are thin, in the young, the old, and in emaciated subjects; and (2) where the volume of air is increased, as is seen when the percussion note of full inspiration is compared with that of expiration; and further, in cases of emphysema, where the absolute volume of intrathoracic air is increased, not merely because many of the pulmonary septa have disappeared and their place has been taken by air, but also on account of the permanent position of the thorax in the condition of expansion.

**The Pitch of the Percussion Sound.**—The pitch of a note depends upon the rapidity of the vibrations of which it is composed, and it has been explained in the previous chapter how the term pitch may be more or less correctly applied to such a compound sound as that of percussion. The pitch of the thoracic notes depend on three factors—(1) the tension of the chest wall, (2) the tension of the lung tissue, and (3) the length of the underlying air columns; and it has been already shown how these three conditions tend to modify the pitch of the note.

1. *The State of Tension of the Chest Wall.*—When a full inspiration is made, the tension of the chest wall increases, and consequently the percussion note tends to rise in pitch, although this tendency is to a certain extent counteracted by the increase of the volume of air in the lungs which then takes place. As a whole, however, the pitch during inspiration is higher than during expiration. In the same way, in pulmonary emphysema the percussion note is usually raised in pitch, owing in great measure to the increased tension of the thorax; and though the intensity of the note (as has been already said) is increased in these

cases, the author has sometimes seen emphysema mistaken by the inexperienced for pulmonary consolidation, owing to the heightened pitch.

2. *The State of Tension of the Pulmonic Tissue.*—The increased tension of the lung tissue, during full inspiration, no doubt tends to heighten the pitch of the percussion note, along with the tension of the chest wall above mentioned. The results of relaxation of the lung tissue will be best described when we come to speak of the tympanitic note.

3. *The Length of the underlying Air Columns.*—Whenever the air-containing cavity, lying under the point of percussion, becomes more or less filled up, either as the result of effusion of fluid into the pleural cavity, or of effusion or exudation into the alveoli, such as takes place in œdema, pneumonia, and in the various forms of phthisis, the air column becomes shortened, and the percussion note rises in pitch. The same result follows where, from the deposition of new formations in the lung tissue, the air columns become broken up in their length. Within these limits fall the greater number of pathological conditions which are met with in connection with the lungs. In each special case it is not difficult to see how the note becomes modified as regards its pitch.

In like manner is to be explained the change of note which occurs when we pass from the lungs to such solid organs as the liver and heart, and which enables us to map out their outlines in the manner already described. Take the liver for example: As we percuss downwards in the mamillary line, we reach the upper margin of relative liver dulness—that point, namely, where the sound first becomes modified. It is here that the liver, lying in the hollow of the diaphragm, first begins to encroach upon the air space, filling it up from behind, and thereby shortening the air columns, and diminishing the volume of underlying air. The intensity of the note is thus diminished, and its pitch rises, and these changes in the percussion note become more and more marked until we come to the upper limit of absolute liver dulness, where no lung tissue interposes itself between the liver and the chest wall, and the note, therefore,

becomes absolutely dull. In the same way, the topographical percussion of other solid organs is to be explained.

Passing now to the consideration of certain changes in the quality of the percussion note, we come first, and most importantly to

**The Tympanitic Percussion Note.**—This variety of chest note differs from that of health in that it approaches much more nearly to a pure musical tone—that is, its vibrations become much more regular. The great regularity of these vibrations Gerhardt has shown by means of König's sensitive flame reflected in a rotating mirror. This variety of percussion note is found in perfection over the stomach when that viscus is moderately distended with air. If the stomach be removed from the body, both orifices ligatured, and then moderately distended with air, it will be found to afford a tympanitic note on percussion; but if the distension be continued, a point will be reached when the note loses that peculiar quality and becomes muffled. The reason of this is not far to seek. In the case of moderate distension, the gastric wall is not sufficiently tense to pass into vibration, and thus the sound results simply from the vibrations produced in the contained air; but when the walls become tense from over-distension they also vibrate, and the tones so produced do not harmonise with those of the vibrating air, so that the combined sound is irregular in its vibrations, and therefore no longer tympanitic.

Similarly, if a lung be removed from the body and allowed to collapse, it will, when percussed, give a tympanitic note, which disappears when the lung is again distended with air to a point corresponding to the normal condition. The air in the collapsed lung vibrates as a whole, and the lung tissue is not sufficiently tense to admit either of its passing into vibration, or of the stronger septa breaking up the air columns so as to render the combined note irregular and non-tympanitic, as is the case when the lung is in a state of normal distension. It is to be noted that the pitch of the tympanitic note (which is very readily made out) gives a trustworthy indication of the size of the air cavity,

and this is very important as a means of distinguishing the note of the stomach from that of the neighbouring intestines.

Further illustrations of the tympanitic note in health are to be found when percussion is made on the cheek when the mouth is moderately distended with air, or over the trachea. The latter example is of especial value in that it shows another property of the percussion note—viz., that when the orifice of the cavity is narrowed or closed, the pitch of the note falls. If the trachea be percussed, first with the mouth open, and then with it shut, this lowering of the pitch may be readily detected, and it will be still more obvious if the nostrils be at the same time compressed.

From what has just been said, it will be seen that the tympanitic note may occur in the chest under the following pathological conditions :—

1. Relaxation of lung tissue.
2. The presence of underlying air cavities.
3. Pulmonary consolidation, allowing the broncho-tracheal air column to be set in vibration.

1. *Relaxation of Lung Tissue.*—Just as when the lung is removed from the body, and allowed to collapse, it gives a tympanitic note ; so, when a similar retraction and relaxation of the pulmonic tissue takes place within the thorax, that variety of percussion note may be heard. This is best marked in cases of pleuritic effusion, which, gravitating to the lower portion of the cavity, floats up the lung and causes retraction of the upper portions. When the effusion is small in amount, this tympanitic note can only be detected over that portion of lung which lies immediately above the upper limit of the fluid, but when the effusion is considerable, the whole upper lobe may be tympanitic on percussion.[1] Similarly, effusion into the alveoli (in pneumonia or œdema) may produce a like result. In the first stage of pneumonia the change in the note seems to be produced by relaxation, occasioned by the inflammatory congestion of the lung tissue.

It is particularly to be observed that the pitch of the

[1] When the effusion is excessive, the lung tissue becomes compressed, and the tympanitic quality of the note is consequently lost.

tympanitic note which occurs under the above conditions is not altered by shutting and opening the mouth.

2. *The Presence of underlying Air Cavities.*—When the pleural cavity becomes filled with air (pneumothorax) a typically tympanitic note results from percussion, provided that the distension be not too great. Its pitch is not altered by opening and closing the mouth. When the cavity contains, in addition, serum or pus (hydro- or pyo-pneumothorax), the note changes in pitch with the position of the patient, the fluid gravitating to the most dependant part in each instance, and so altering the lengths of the air columns.

Cavities in the lung tissue, when filled with air, of sufficient size, smooth walled, and near to the thoracic wall, also give a tympanitic note, and as they communicate with a bronchus, the pitch of their note varies when the mouth is opened and closed. The position of the long axis of the cavity may also be ascertained, if it contain fluid as well as air, for the movements of the fluid occasioned by alterations in the position of the patient cause changes in the pitch of the note, just as in hydro-pneumothorax (Gerhardt). This change, when it is observed, is very characteristic of cavities in the lung.

3. *Pulmonary Consolidation, allowing the Broncho-Tracheal Air Column to be set in Vibration.*—I have already alluded to the tracheal sound, which is characteristically tympanitic. In health, however, it is not possible to set in vibration the air column in the bronchi and trachea by percussing over the chest. If, however, the lung tissue be consolidated, the impulse of the percussion stroke may be transmitted to the bronchi, and in this way the tympanitic *tracheal note of Williams* may be produced. This note is almost always found on the left side—rarely on the right, and it is characteristic of it that the pitch is altered by opening and closing the mouth, but *not* by changes in the position of the patient.

Another note peculiar in quality, which must be mentioned, is the

**Cracked-Pot Sound** (*Bruit de pot fêlé*).—The peculiar quality

of this sound is caused by the sudden expulsion of air from a cavity through a small opening in its walls, and it is heard when the hands are pressed together and struck upon the knee, in a manner well known to school-boys, so as to produce a noise closely resembling the rattling of coin. It derives the name (which Laennec first gave it) from the resemblance to the sound produced by striking a cracked jar.

The cracked-pot sound occurs under the following conditions :—

1. *In Health.* — In children, when the glottis is narrowed, either during a fit of crying or when a sustained high-pitched note is being sung, percussion of the chest gives this variety of sound, the air being suddenly forced from the lung through the narrow glottis.[1]

2. *In cases of Relaxation of Lung Tissue*, which I have already described as favouring the production of tympanitic percussion, the cracked-pot sound may sometimes be heard.

3. *In cases of Thoracic Fistulæ*, and pneumothorax, when the percussion stroke expels air through the fistula with a hissing sound.

4. *In cases of Pulmonary Excavation.*—This is by far the most frequent cause of the cracked-pot sound, so much so that when consolidation of the lung tissue exists, the cracked-pot sound may be taken as strong evidence of the presence of a cavity.

The last change in quality which we shall here consider is that which is known as

**Amphoric Resonance**. — This sound is similar to one produced by striking on the side of an empty jar or cask, and it owes its peculiar metallic quality to the high-pitched upper partial tones which it possesses, and which are caused

[1] Also in adults, when the chest is very hairy, and a pleximeter is being used, the cracked-pot sound is apt to be produced. Owing to the instrument not being closely applied to the chest wall, a layer of air intervenes, and a portion is forcibly expelled by the percussion stroke, producing the sound in question. Moistening the hair does away with this difficulty.

by the reflection of the waves of sound from side to side of the closed cavity. These upper partial tones die away slowly.

When speaking of the tympanitic percussion sound, it was pointed out that when the stomach is over-distended with air that quality is lost, the note becoming hard and metallic. Amphoric resonance is then formed, the sound waves being again and again reflected from the tense walls of the viscus.

The conditions necessary for the production of amphoric resonance are that the air-containing cavity should be of considerable size and superficial, that its walls should be smooth and resistant, and that it should either be completely closed or should only communicate with the external air by means of a small opening.

As in the tympanitic note, so also in the amphoric sound, the pitch of the prime tone enables us roughly to estimate the size of the cavity in question.

In thoracic percussion this amphoric echo is met with in two conditions—over pulmonary cavities, and in pneumothorax. In both these cases it is best to combine auscultation with percussion, the physician, listening with a stethoscope in the neighbourhood of the cavity, while an assistant percusses. For percussion it is best to use a plexímeter, and to strike it with some hard substance such as a coin, as the metallic note thus produced brings out by sympathetic resonance the high-pitched upper partial tones of the cavity.

**Feeling of Resistance during Percussion.**—The sound which the percussion of the chest affords is not the only sensation which is perceived by the physician in consequence of the stroke. There is further a sense of the degree to which the chest walls yield to the force of the blow.

This feeling of resistance may be dependent solely upon such changes in the chest wall as tend to increase its solidity (such as deposit of fat, thickening of the ribs, &c.), but if we except these, it gives a trustworthy and sometimes exceedingly valuable indication of the comparative solidity of underlying organs.

Whenever the lung becomes airless, whether from exudation or compression, the resistance is increased; and still more is this the case when effusion of fluid has taken place into the pleura, and most of all over intrathoracic tumours.

Diminution of resistance is met with in cases of pulmonary emphysema, when well marked, and in pneumothorax.

**Topographical and Regional Percussion.**—The limits within which the pulmonary percussion note is heard are of importance, not only in determining the outline of neighbouring solid organs, but as a guide to the physical condition of the lungs themselves.

*The Apices.*—The upper limit of the lung note corresponds to a line which, following at first the clavicular portion of the sternomastoid muscle, curves over to meet the anterior margin of the trapesius, and then passes downwards to the seventh cervical vertebra. This line rises on each side to a point about $1\frac{1}{4}$ to 2 inches (3 to 5 centimetres, according to Leitz) above the clavicle, being perhaps a trifle higher on the right side. In percussing the apices, care must be taken that the patient's head is not turned to either side, and that the direction as well as the force of the stroke is the same on each side. The shrinking of the apices, both vertically and transversely, is one of the first physical signs of incipient phthisis, and is therefore of considerable importance. In pulmonary emphysema, the limits above given may be overstepped to a considerable extent.

*The anterior margins* approach each other at the level of the second cartilage, being separated only by the anterior mediastinum, and continue downwards parallel to each other as far as the fourth rib, where the margin of the left lung curves outwards to follow the line of the absolute cardiac dulness, as described on page 108; while that of the right lung continues vertically downwards as far as the sixth cartilage, where it joins the inferior margin.

*The inferior margins* are much affected by respiration. Their position during quiet respiration may be taken to be as follows:—

The right lung—

| | |
|---|---|
| At sternal border, | 6th rib. |
| Parasternal line, | 6th rib. |
| Mammillary line, | 7th rib, upper border. |
| Axillary line, | 8th rib. |
| Scapular line, | 9th rib. |
| At vertebral column, | 11th rib. |

The left lung—

| | |
|---|---|
| Axillary line, | 8th rib. |
| Scapular line, | 9th rib. |
| At vertebral column, | 11th rib. |

With forced respiration the inferior edges of the lungs rise and fall very considerably,—to such an extent, indeed, that in the axillary line there may be a difference of over 3 inches between full expiration and full inspiration. In cases of emphysema, not only are the lower borders much depressed, but their movement during respiration is greatly interfered with.

The influence of emphysema, and other pathological conditions, on the anterior borders of the lungs, has been already alluded to in connection with the percussion of the heart.

**Regional Percussion.**—The difference of the percussion sound at different parts of the healthy lung depends upon the condition of the chest wall, and upon the number and disposition of the air columns which radiate from the point struck.

*Anteriorly.*—The sound over the apices above the clavicles is clear, but not great in intensity. Below the clavicles the note falls somewhat in pitch, and grows in intensity until we come to the relative dulness of the heart on the left side (lower margin of third rib) and of the liver on the right (fourth interspace, or fifth rib), when in both cases the sound rises in pitch and loses intensity, and does so more and more until the limit of absolute dulness of each solid organ is reached. The right lung is usually slightly duller than the left, owing to the greater development of muscle on the right side. Over the sternum

the sound is clear, deep, and resounding, owing in part to the vibrations of that bone, but chiefly to the fact that the air in both lungs is set in vibration.

*Posteriorly.*—In percussing the thorax posteriorly, the patient should be made to cross his arms in front and bend forward. The note over the scapulæ is less clear than that at the lower portions of the back. The lung note can be heard as low down as the tenth or eleventh rib.

*Laterally.*—In the axillary regions the pulmonary note is intense and clear on both sides, until the dulness of the liver is reached on the right side, and that of the spleen on the left.

# CHAPTER XXII.

## RESPIRATORY SYSTEM—(*continued*).

### AUSCULTATION.

THE auscultation of the lungs may be performed with the aid of a stethoscope, or more simply by applying the ear to the thoracic wall. For obvious reasons, the former method is the pleasanter both to patient and to physician, and it possesses this further advantage, that, by means of the stethoscope, any abnormal auscultatory phenomenon can be more distinctly localised than is possible if the immediate method be employed. The form of instrument is of comparatively little importance, provided that it fits the ear of the physician. The simple wooden stethoscope answers admirably for all ordinary cases, although sometimes the double instrument of Alison may be made use of with advantage.

The position of the patient is of considerable importance. Where there is a choice, probably the sitting posture is the most convenient, but whatever attitude be adopted it must be unconstrained. Of at least equal moment is the posture of the physican, which should be easy and comfortable. The chest of the patient should, if possible, be fully uncovered; but failing this, the intervening clothes must be thin, and all friction between them and the stethoscope sedulously avoided. The instrument should be firmly and accurately applied to the chest, and not till then should the physican apply his ear to the upper end, always remembering that the ear must be moved so as to suit the stethoscope, and not the stethoscope to suit the ear. Attending to these precautions, the whole chest should be carefully examined, corresponding points

on the two sides being compared in the same manner as in percussion.

On auscultating the chest there is to be heard at most points a gentle "breezy" sound—the vesicular murmur—which has been compared to the sighing of wind among leaves, and the special character of which can only be learned by practice. It consists of two murmurs, the one corresponding to inspiration and the other to expiration, of which the first is about three times as long as the second, and is softer and higher in pitch.[1] Not only do pathological changes in the lungs alter the ordinary respiratory murmur, but they often produce totally different sounds, to which in turn our attention must be directed. In ordinary clinical examination, then, the main points to be attended to in regard to auscultation are—

1. The relative duration of the expiratory and inspiratory murmurs.
2. The character or quality of the breathing sounds.
3. The presence or absence of adventitious sounds of various kinds.
4. The character of the vocal resonance (auscultation of the voice).

**Vesicular Murmur.**—In speaking of murmurs arising in the blood current, it has been already pointed out that when a fluid streaming through a tube passes from a narrower into a wider portion, vibrations arise in the fluid owing to the friction of the molecules upon one another, which, if sufficiently rapid, may give rise to an audible murmur. This is equally true with regard to gases. Now, in the air passages there are two points at which such an alteration in calibre is to be found—viz., at the glottis and at the point where the bronchioles enter the alveoli. The rush of air through the narrow glottis sets in vibration the air column contained in the trachea and bronchi, and a blowing murmur results—the tracheal or bronchial mur-

[1] The expiratory murmur is, however, not unfrequently inaudible in healthy persons.

mur—which will be described more fully hereafter; and in a similar manner a murmur arises as the air streams into the air cells. To a combination of these two murmurs, in which the latter predominates, it appears most reasonable to ascribe the formation of the normal vesicular breath sound.[1] Whatever be its origin, however, it may be safely held that, when vesicular breathing is heard, the pulmonary alveoli are fulfilling their function, and when it is absent, that that function is in more or less complete abeyance.

The vesicular murmur is to be heard more or less clearly over the whole pulmonary surface, but it varies in distinctness at different parts according to the thickness of the chest wall and the volume of lung tissue underlying the stethoscope. From various causes vesicular breathing may be absent. Thus it may be replaced by bronchial breathing, or it may be inaudible, owing to the loudness of superadded sounds, or to the interposition of a tumour or of fluid between lung and chest wall; or, finally, it may be absent owing to obstruction of a bronchus or to collapse of lung tissue.

The common modifications of vesicular breathing are as follows:—

1. Harsh or puerile.
2. Jerky.
3. Prolongation of the expiratory murmur.
4. Weak, or even absent.

(1.) *Harsh Vesicular Murmur.* — In children the normal vesicular breath sound is clear, sharp, and loud, and this harsh or puerile breathing appears to depend in part upon the thinness of the chest walls and the greater elasticity of the lung tissue.

In adults harsh or puerile breathing occurs where, part of the respiratory surface having been thrown out of action by disease,

[1] Baas, Gerhardt, and others, however, hold that the vesicular as well as the bronchial murmur arises solely at the glottis, and that the sound is in the former case modified by transmission through the lung tissue. The theory is attractive, but the proof offered seems inadequate, more especially as clinical facts point in the other direction.

the healthy portion is receiving a more than usually liberal supply of air. Over this healthy portion the breath sounds become harsh or puerile. In cases of pneumonia, pleural effusion, and many other lung conditions, this puerile breathing may be heard over the unaffected parts of the lung.

(2.) *Jerky Breathing.*—In nervous persons, and particularly in hysterical women, the inspiratory vesicular murmur is very apt to be broken into three or four distinct parts. This jerky breathing, which is heard over the whole lungs, often disappears when the patient is told to take a deep inspiration, and is of no practical importance. But there is another variety of jerky breathing which differs from that just referred to in two particulars, viz.—(1) it does not disappear with deep inspiration; and (2) it is distinctly localised. This broken respiration is met with in cases of incipient phthisis, and is a sign of considerable importance, depending for its production upon some local obstruction to the entrance of air into the alveoli.

(3.) *Vesicular Murmur with Prolonged Expiration.*—It has been said that in healthy persons the expiratory murmur is frequently inaudible. When it is audible its duration is usually about one-third that of inspiration. When expiration exceeds this length we may conclude that either the lung tissue has lost its elasticity, or that there is some obstruction to the escape of air. One or other of these two conditions is met with in almost every affection of the lungs, so that in pulmonary disease a prolongation of the expiratory murmur is very frequently encountered. It is, however, not uncommon in health to find some degree of harshness with prolonged expiration at the right apex.

(4.) *Weak Vesicular Murmur.*—In cases where the respiratory action is lacking in vigour, the murmur is correspondingly weakened, and a similar result follows where the chest walls conduct sound badly, as results from the deposition of fat, or from œdematous swelling. Thickening of the pleural surfaces, or pleural effusion of limited amount, may also produce weakening of the vesicular murmur.

Any condition which leads to collapse of extensive portions of lung tissue (such as pleural effusion) leads to the abolition

of the breath sounds over the affected part. The obstruction of bronchi with mucus may produce the same result, and until the secretion has been expelled by coughing, the vesicular murmur will remain inaudible.

**Bronchial Respiratory Murmur.**—The second great variety of respiratory murmur is that which is known as laryngeal, tubular, or bronchial. It can be heard in perfection when the stethoscope is placed over the larynx or trachea. Its peculiar character may be imitated by arranging the position of the mouth and tongue to utter the gutteral "Ch" and then

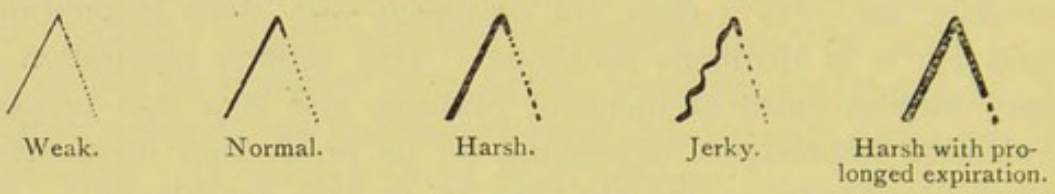



FIG. 53.—Diagram of various types of Vesicular Breathing (after Wyllie).

breathing quietly out and in. The expiratory portion of the murmur is about as long as the inspiratory, and between the two there is a short but quite appreciable break. Bronchial breathing cannot be heard over the lung tissue, under conditions of health.

*Mode of production of the Bronchial Respiratory Murmur.*—The air passing in and out of the chest with the movements of respiration, encounters at the glottis a considerable narrowing of the tube through which it is flowing, and in consequence vibrations arise in the immediate neighbourhood of the narrow point, which are of sufficient rapidity to be audible as a murmur. Underlying this vibrating point there is the air column contained in the trachea and bronchi, which is set in vibration by sympathetic resonance, and thus the glottis murmur is augmented and reinforced. It is in this manner, in all probability, that the bronchial murmur in healthy persons is produced. It can readily be understood how, when the opening of the glottis is narrowed by such a pathological process as croup, the murmur is louder and higher in pitch.

To the vibrations at the glottis, and those of sympathetic resonance in the broncho-tracheal air column, other vibrations may, however, be added in consequence of pathological changes in the air passages. When the lumen of the trachea is narrowed, as the result of the pressure of a tumour, vibrations and a consequent murmur arise at the stenosed point, and are reinforced by the underlying air-column just as the glottis murmur is; and even when the mucous membrane of the trachea and bronchi becomes swollen and roughened by catarrhal processes, the character of the bronchial murmur changes and it becomes harsh—the result, no doubt, of local vibrations.

### *The Bronchial Murmur in Disease.*

As has already been said, the bronchial murmur is only audible in health over the larynx and trachea. It cannot be heard over the chest generally, partly because it is overpowered by the vesicular murmur, and partly because inflated lung tissue is a very bad conductor of sound. This murmur becomes audible, however, under two varieties of pathological conditions as follows:—

(1.) *When the lung tissue becomes condensed*—provided that the condensation is extensive, and lies at or close to the surface of the lung, and contains besides a large and unobstructed bronchus—the vesicular murmur disappears over the condensation, and the bronchial murmur is conducted to the surface and becomes audible. These conditions are fulfilled in the case of acute pneumonia (stage of hepatisation), and in all the varieties of chronic phthisis. Bronchial breathing is therefore heard over hepatised lung, and wherever phthisical consolidation is of sufficient extent. It also occurs when the lung tissue is consolidated as the result of compression and collapse—as, for example, above the level of a pleuritic effusion; but it is not by any means always met with under such conditions, for the pressure of the effusion must be sufficient to cause collapse of the air-cells, and yet not sufficient to obliterate the bronchi.

(2.) *In pulmonary cavities.* Bronchial breathing, of the low pitched variety, may be heard over vomicæ, provided that they are superficial, have smooth walls, are surrounded by condensed tissue, and freely communicate by means of a bronchus with the air in the trachea. In certain cases it may be possible to judge roughly of the size of the cavity by the pitch of the bronchial murmur heard over it, since the air rushing into the cavity excites sympathetic resonance in it—that is, calls forth its special tone, which corresponds to the size of the resonating cavity, and this, if loudly enough heard, gives a guide to its capacity.

It is not desirable to subdivide bronchial breathing into a number of different varieties, as such a course only tends to cause confusion, without apparently promoting any useful purpose. It may, however, be well to mention that many writers recognise a modification of bronchial respiration, which




FIG. 54.—Diagram of Bronchial Breathing (after Wyllie).

Laennec named "cavernous breathing," in which the air appears to the ear of the auscultator to pass into a large hollow space. This variety of bronchial breathing is sometimes heard over pulmonary cavities and over the dilated bronchi of bronchiectasis.

There is, however, one special variety of bronchial breathing to which attention must be directed, viz. :—

### *Amphoric Respiration.*

The peculiar character of this variety of bronchial breathing is perfectly reproduced by blowing into an empty jar or bottle, and its mode of origin is similar to that of the sound so obtained. Amphoric breathing occurs under two pathological conditions—(1) pulmonary excavation, and (2) pneumothorax as follows :—

(1.) *Pulmonary Excavation.*—In order that this peculiar variety of breathing may be produced, the cavity must be of very

considerable size, with smooth firm walls, and must lie superficially. It must contain air, and must be in free communication with a bronchus. In such vomicæ the sonorous waves excited by the respiratory current are reflected again and again from the smooth walls, and so come to have an amphoric character, the prime tone being comparatively low in pitch, and the upper partials high and ringing.

(2.) *Pneumothorax.*—When air escapes into the pleural sac and distends it, the lung tissue becomes compressed; and if this pressure be sufficient not only to drive the air out of the air-cells, but also to cause collapse of the bronchi, no amphoric breathing occurs. But if the fistula by which the air has entered becomes closed before the pressure has becomes sufficient to obstruct the bronchi, the bronchial respiration will be conducted to the immediate neighbourhood of the large air cavity in the pleura, in which, by sympathetic resonance, sonorous vibrations will be excited. These vibrations, owing to the physical conditions met with in pneumothorax (smooth, firm walls, &c.), will have an amphoric character. If some quantity of serum or pus be present, along with air, in the pleural cavity, the pitch of the amphoric sound will vary according to the position of the patient, for the reason already mentioned.




FIG. 55.—Diagram of Amphoric Breathing (after Wyllie).

Thus far, the two great classes of respiratory murmur, the bronchial and the vesicular have been described. Between these two, however, there lies an intermediate variety, which may be called

### *Broncho-vesicular Breathing.*

There may occasionally be heard a respiratory murmur, which even the most practised ear cannot define as being either bronchial or vesicular. This murmur may be audible in healthy

persons when the large bronchi lie near the chest wall, that is opposite the root of the lungs. By placing the stethoscope in the interscapular region, opposite to the third dorsal vertebra this broncho-vesicular murmur may be heard. It is often audible also over the sternum.

This broncho-vesicular murmur is only of diagnostic value when localised in some particular part of the chest, other than those just mentioned, particularly when it is confined to one apex. In the latter case it points to the probability of commencing phthisical change.

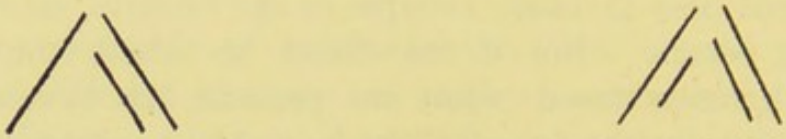


FIG. 56.—Diagram of Broncho-Vesicular or "Indeterminate" Breathing (after Wyllie).

## CHAPTER XXIII.

## RESPIRATORY SYSTEM—(*continued*).

### ADVENTITIOUS SOUNDS ACCOMPANYING RESPIRATION.

In health the respiratory murmur is not accompanied by any other sound, but in the great majority of diseases of the lungs, at some part of their course, there become audible certain abnormal or adventitious sounds which are collectively known under the term râles. Inasmuch as certain of these râles give to the ear the impression of being caused by the bursting of air bubbles in a fluid, while others have a dry snoring or whistling character, they have been divided into two classes—moist and dry râles. Although this division is not scientifically accurate, some of the apparently moist sounds being in reality formed without the presence of fluid, and certain of the dry râles owing their production to the presence of a more or less viscid secretion, yet the division is clinically useful, and ought not to be discarded.

Physicians differ much in regard to the nomenclature of these râles, and as a rule they have been too minutely subdivided. For all practical purposes the following classification will be sufficient :—

1. Moist râles—
   - (*a*) Fine crepitation.
   - (*b*) Medium crepitation.
   - (*c*) Coarse bubbling râles.
2. Dry râles : or rhonchi.
   - (*a*) Sonorous.
   - (*b*) Sibilant.
3. Pleuritic friction.

## Moist Râles.

*Fine Crepitation.*

The peculiar fine moist râle, which Laennec described under this name, has been compared to the sound produced by rubbing a lock of hair between the fingers close to the ear, or to the crepitation of salt when thrown upon the fire; but, as Eichhorst points out, both these sounds are too coarse, and crepitation may be more closely intimated by firmly pressing the moistened thumb against the forefinger, and then suddenly separating the two surfaces, close to the ear.

Although crepitation is probably sometimes due to the bursting of fine bubbles in the very smallest bronchioles, it commonly arises from the sudden separation of the alveolar walls, which have become adherent either to each other or to a mass of viscid secretion in the air cell. It is typically met with in the first stage of pneumonia, of which it is a most important sign. It also occurs occasionally in pulmonary collapse and œdema.

Crepitation occurs almost invariably only during inspiration, and is usually limited to the latter part of it alone. The individual crepitations of which it is composed are characteristically uniform in size, and are unaffected by the act of coughing.

Occasionally in health a momentary crepitation may be heard, usually at the lower posterior border of the lung, but sometimes also at the apex, when a deep inspiration is made, more especially when the patient has been lying on his back, and the respiration has been very quiet for some hours. A knowledge of this fact will prevent any mistake.

*Medium Crepitation.*

*Coarse Bubbling Râles.*

These two varieties of râles being closely associated, they may conveniently be considered together. The difference in the size of the bubbles in each case depends somewhat upon the quantity and quality of the fluid in which they originate, but chiefly upon the size of the space. The finer bubbling râles

arise for the most part in the smaller bronchi, the coarser in the large bronchi, in the trachea, or in pulmonary cavities. In the great majority of cases in which these bubbling râles are heard, they vary in size, and are therefore spoken of as irregular, in contradistinction to the regular fine crepitant râle which has just been described.

Arising in fluid, as these bubbling râles do, we would naturally expect that they would be found most abundantly in the lower portions of the lung—the fluid obeying the law of gravity—and this is generally the case, the base of the lung posteriorly being their most common seat. When, on the contrary, they are heard most abundantly at the apices, and still more when they are exclusively met with there and persist for some time, the condition is one which must be looked upon with considerable gravity, pointing as it does to a local cause, which in the majority of cases is some form of pulmonary phthisis.

The finer bubbling râles in the smaller bronchi occur chiefly at the height of inspiration and the beginning of expiration, while coarse bubbling may be heard both during expiration and inspiration, being then continuous. In both cases a severe fit of coughing may remove the râles for the time. Their amount and intensity depend upon the quantity of fluid, the nearness of the bubbling to the surface, and the strength of the respiration.

In so far as the properties of these bubbling râles, which have as yet been described, go, their presence only informs us that the air current encounters fluid in the respiratory passages, through which it bubbles. We now come to certain qualities in the tone of these râles, which give an indication of the condition of the surrounding pulmonary tissue. If the lung tissue around the point at which the bubbling is taking place is consolidated, the râles assume a clear musical high pitched quality, and are termed *resonant.* Whenever such râles are heard we may conclude, with safety, that consolidation is present (although their absence does not permit of the exclusion of such a condition), and in fact resonant bubbling has a significance exactly similar to that of bronchial breathing. When the râles occur in a large cavity with smooth walls, and near to

the surface of the lung, they assume a peculiarly clear metallic character—the *metallic tinkling* of Laennec. These râles are very musical, and have a high pitch which can readily be determined; and in regard to their physical cause and the conditions under which they occur, they stand in close relation to amphoric breathing and resonance. Similar resonant râles may be heard over large air cavities which lie in close proximity to the lungs, such as a pneumothorax, or even the stomach or intestine when distended with air. In such cases it is not necessary that the râles arise in pulmonary cavities; they may originate simply in the bronchi, and the neighbouring air cavity may act as a resonator, reproducing and intensifying the sound.



FIG. 57.—Symbols of various forms of Accompaniment, for noting on out-line diagrams of the Thorax (after Wyllie).

**Dry Râles or Rhonchi** are produced in the air passages by any pathological process which narrows their lumen, the most common being the accumulation of viscid secretion and the swelling of the mucous membrane. When they arise in the larger bronchi they are low-pitched and snoring (sonorous râles), when in the smaller tubes they have a whistling character (sibilant râles). Both varieties occur chiefly during inspiration, the snoring râles at its commencement, the sibilant not till towards its termination.

Both these varieties of dry râle occur in cases of bronchial catarrh, whether acute or chronic, primary or secondary, and according as they are sonorous or sibilant we may infer that the larger or the smaller bronchial tubes are affected. They are also heard in cases of bronchial asthma, and are then, in some measure, due to the narrowing of the bronchial lumen.

The presence of pulmonary consolidation round the point at which these râles occur imparts to them a ringing musical character, but as their quality is in any case musical, this

change has not anything like the diagnostic value which it possesses in the case of moist râles.

**Pleuritic Friction.**—The gliding of one pleural surface over the other, which occurs normally with each respiration, is accomplished without any sound; but when, as the result of pleurisy, the surfaces become rough and uneven, the sound of friction becomes audible. This sound varies from the lightest rubbing, only perceptible with difficulty, to loud creaking, which can readily be made out on palpation, and which the patient himself both feels and hears. The sound is usually broken up into portions of greater and less intensity, and while it is sometimes audible throughout the whole of both respiratory phases, it is usually limited to the latter portion of inspiration. In cases of pleurisy the friction sound becomes audible whenever the process has advanced sufficiently far to cause considerable roughness of the pleural surfaces, and it of course disappears when those surfaces are separated by effusion, to reappear when absorption of the fluid has taken place. Although the friction sound may sometimes be audible over a great part of the lung, it is usually limited to a small area, and occurs most frequently in the axillary region. When the friction sound is heard at the apex of the lung it points with great probability to phthisis.

With regard to differential diagnosis, the pleuritic friction-sound is sometimes closely simulated by râles in the air passages.

Attention to the following points will usually suffice to distinguish them:—

| *Râles.* | *Friction.* |
|---|---|
| Modified by coughing. | Not modified by coughing. |
| Not affected by pressure of the stethoscope. | Intensified by pressure of the stethoscope. |
| Usually heard over wide area. | Usually localised. |

From pericardial friction the sound of pleuritic friction may

be distinguished by causing the patient to hold his breath, when the latter will disappear and the former continue.

**Auscultation of the Voice.**—In a former chapter the fremitus, or vibration of the chest walls, produced by the act of speaking, has been described. As regards its causation and the various pathological conditions under which it is enfeebled or intensified, the resonance of the voice closely corresponds to the vocal fremitus.

When the stethoscope is applied to the chest while the patient speaks, only a soft indistinct murmur is to be heard, provided that the lung is healthy. On listening over the bronchi, in the interscapular region, this vocal resonance is much intensified, a condition which is termed *bronchophony*. A still greater increase of vocal resonance, resembling that heard when the stethoscope is placed over the larynx as the patient speaks, is known as *pectoriloquy*.

Before speaking of changes in the vocal resonance produced by pathological conditions connected with the lung, it may be as well to repeat what was said in connection with vocal fremitus—viz., that the vibrations of the voice over the thoracic parietes, audible as well as perceptible to palpation, depend for their intensity upon the loudness and depth of pitch of the voice, and upon the thickness of the chest wall; that the vocal resonance (like the corresponding fremitus) is more distinct in men than in women; and that it is almost invariably louder on the right side than on the left, owing to the larger calibre of the right bronchus.

Bearing these points in mind, we may now consider the changes in the vocal resonance which result from pulmonary disease.

*Enfeeblement of the Vocal Resonance.*—The vocal resonance is diminished or entirely lost when the lung is separated from the chest wall by collections of liquid[1] or air in the pleural cavity, and when the bronchi leading to the part of the lung in question have become blocked up with secretion. Thickening of

[1] With the exceptions to be presently mentioned.

the pleura is also a common cause of diminution of vocal resonance.

*Intensification of the Vocal Resonance.*—As has been already said, bronchophony occurs normally over the root of the lung in the interscapular region. When bronchophony occurs at other points of the chest it is pathological, and it then owes its origin to consolidation of lung tissue, and the consequent better conduction of the vocal vibrations to the chest wall. Bronchophony thus arises, along with bronchial respiration, in all diseases which lead to condensation—for example, in acute pneumonia, and in all the forms of phthisis. Pectoriloquy is to be heard noticeably over pulmonary cavities, the resonance of the air in the cavity adding to the intensity of the vocal resonance, and imparting to it in addition a peculiar metallic character.

It has been said that pleural effusions diminish or even suppress the vocal resonance; but this is not always the case. Baccelli pointed out, in 1875, that the resonance of the whispered voice was often heard very clearly over pleural effusion. This whispering pectoriloquy (*pectoriloquie aphonique*) he held to occur only when the fluid was homogeneous (serous effusion), and not when the effusion was heterogeneous (pus). There can be no doubt that this sign is very frequently present in such cases, but later observations have failed to confirm its value in so far as the discrimination between serous and purulent effusions is concerned.

Under certain conditions, the vocal resonance assumes a very peculiar nasal quality, resembling the noise produced by speaking against a comb covered with paper, and which, from its supposed resemblance to the bleating of a goat, Skoda termed.

*Aegophony.*—This variety of bronchophony is most commonly met with in cases of pleuritic effusion, near the upper margin of the fluid, and usually close to the lower angle of the scapula. As to the exact manner of its causation there is some doubt, but most observers are agreed that it depends upon compression and partial obstruction of the bronchi. Its diagnostic value does not materially differ from that of ordinary bronchophony.

**Hippocratic Succussion.** — We must, in conclusion, refer briefly to this sign, which was described by Hippocrates, and which is of considerable interest.

If, in cases of hydro-pneumothorax or of pyo-pneumothorax, the ear be applied to the chest, and the patient shaken, a ringing splashing sound may be heard, which is the sound in question. The splashing noise becomes intensified by the resonating air cavity above the fluid in the manner already described. This succussion sound may also be heard when there is a very large excavation in the lung tissue partially filled with fluid.

## CHAPTER XXIV.

# INTEGUMENTARY SYSTEM.

The study of the affections of the skin is of great importance to the physician, not merely on account of the frequency of their occurrence, the distressing severity of their symptoms, and the deformities which they leave behind them in their course, but also because they are frequently symptomatic of the general condition of the system, mirroring forth with fidelity not a few grave systemic diseases, and still more often the many slighter disorders which it is important to recognise and to check at their outset, and of which the physician may have no other indication. Among the serious diseases of which the condition of the skin gives evidence, it is hardly necessary to mention syphilis, scrofula, as well as all the members of the important group of exanthemata. Then, again, it is well known that errors in diet, and disorders of the digestive functions generally, are apt to cause various forms of skin disease (urticaria, acne, &c.), and without going further into detail in illustration of this point, it may finally be mentioned that many uterine affections, and even pregnancy itself may be accompanied with blotches on the skin (chloasma uterinum), which in certain circumstances may be a symptom of not a little importance.

**Subjective Symptoms** are of comparatively little diagnostic importance in cases of skin disease, although to the patient they are often very distressing. Sensations of heat attend all the inflammatory processes in the skin. Hyperæsthesia and anæsthesia are met with in the various cutaneous neuroses, and shooting pain is a prominent symptom of herpes zoster, frequently preceding the eruption, and persisting for some time

after its disappearance. But of all the subjective symptoms of skin disease, the most distressing is itching. It is very common as a result of the presence of parasites, but frequently occurs independently of such agents.

**Objective Symptoms.**—A patient suffering from skin disease ought to be examined in a well-lighted and warm room, preferably by daylight, and if a male, the whole surface of the body ought to be viewed by the physician if the case is at all important.

The general condition of the skin as to colour and moisture, the deposit of subcutaneous fat, and the presence of œdema and of emphysema are points which have been already considered in Chapter I., and need not be again referred to.

There only remain, therefore, for our consideration, the various eruptions which occur on the skin, and these can only be treated of very briefly here.

## Eruptions.

In considering skin eruptions, there are four main points to which attention should be directed, and under which the facts observed may be satisfactorily classified.

### 1. *The Distribution and Configuration of the Eruption.*

When the whole surface of the body is covered, the eruption is said to be *universal;* when it is irregularly scattered over various parts of the body, it is said to be *diffused.* The configuration of the individual lesions is commonly defined by such terms as *punctate* when of the size of pin-heads, *guttate* when resembling drops of water, *nummular* when of the size of coin, &c.

The distribution of the lesion is often of considerable diagnostic importance. For example, psoriasis is usually only found on the extensor aspect of the limbs, whereas the macular, papular, and squamous syphilides, when they appear on the limbs, are seen on the flexor surfaces. Then, again, certain lesions, as herpes zoster, follow the course of nerves. Lupus

is usually found on the face, erythema nodosum on the leg, seborrhœa sicca on the scalp, acne on the face and back, and the scabies insect selects for its burrows by preference the skin between the fingers.

### 2. *The Elements of the Skin involved.*

Careful inspection of the skin will inform the physician as to the condition of the epidermis, the hair, the orifices of the hair follicles, the sebaceous and the sweat glands. Palpation of the skin will further give information regarding the condition of the true skin, whether it be infiltrated or not. Pressure with the finger on a pigmented spot will show whether the coloration is due to hæmorrhage or to hyperæmia, for in the latter case the colour will disappear on pressure. Further, if the skin be covered with crusts, the removal of these will display the condition of true skin, which in cases of eczema, for example, may be found to be moist, in seborrhœa dry, and in psoriasis bleeding. We have further in the diagnosis of skin cases to rely upon the evidence afforded by the microscopic examination of the hair, crusts, &c., as will be more particularly pointed out when we come to speak of the ætiology of such affections.

### 3. *The Type of Eruption.*

The very numerous forms assumed by skin eruptions are usually defined and described as follows:—

(*a.*) *Macules* (*Maculæ*).—These consist in morbid changes in the colour of the skin, which are circumscribed, and do not involve the whole cutaneous surface, and which are neither elevated above nor depressed below the surface of the skin. Such macules may arise in very various ways. Sometimes, as in erythema fugax, they are occasioned by hyperæmia, and then the colour disappears on pressure; sometimes by hæmorrhage, as in purpura; by increase or decrease of the normal pigment; by exudations into the tissues of the true skin, as in syphilides; and, finally, they sometimes arise from increase in the size and number of the blood-vessels, as in nævi.

(*b.*) *Papules* (*Papulæ*) are small firm elevations above the surface of the skin, varying in colour, and arising in very different ways. The simplest form of papule is seen in the *cutis anserina* or goose-skin, due to the contraction of the muscles of the skin. Pathologically, papules form as the result of hypertrophy of the papillæ (ichthyosis), of cell proliferation in these structures (lupus, syphilis), or of inflammation of and consequent exudation into these papillæ, as in eczema papulosum. Extravasation of blood into the skin may give rise to papules, as is seen in purpura papulosa. Papules may also be formed in connection with the sebaceous glands (milium, comedo, acne), or by accumulation of epidermic cells round the hair follicles, as in lichen pilaris.

(*c.*) *Tubercules* (*tubercula*) are simply exaggerated papules. They are usually occasioned by cell proliferation, and occur as the result of syphilis, carcinoma, leprosy, &c.

(*d.*) *Tumours* (*phymata*) or new growths of any kind. They may be of considerable size, even as large as a child's head. Examples are seen in molluscum, and in the various cystic growths met with in connection with the skin.

(*e.*) *Wheals* (*pomphi*) are flat, irregularly-shaped, firm elevations on the skin, pale in the centre, red at the edges, and which are very fugitive. Wheals are typically seen as the result of the sting of the nettle, and in urticaria. They result from sudden effusion of the serum into the papillæ, and swelling of the cells of the rete malpighii, produced probably by vasomotor changes.

(*f.*) *Vesicles* (*vesiculæ*) are small rounded elevations of the cuticle, varying in size up to that of a split pea, containing serous, sero-purulent, or bloody fluid, and either lying between the mucous and horny layers of the epidermis, or in connection with the hair follicles, or with the sebaceous or sweat glands.

(*g.*) *Blebs* (*bullæ*) only differ from vesicles in point of size, being larger than a split pea.

(*h.*) *Pustules* (*pustulæ*) are elevations of the epidermis, similar in shape to vesicles and blebs, but containing pus. They are

sometimes found in the substance of the true skin (boils), in connection with hair follicles (as in sycosis), or in sebaceous glands (acne), or between the mucous and horny layers of the epidermis, as in small-pox. Pustules usually dry up (with or without bursting) into yellow or brownish crusts, and very often leave permanent cicatrices, if the tissues of the true skin have been involved.

Thus far we have been considering what are called the *primary* lesions, and we now pass to those which are secondary.

(*i.*) *Excoriations* are breaches of the continuity of the skin, often produced by the patient's nails. They give an indication of the amount of itching which is present. When lice are present (phthiriasis), the marks of the scratches are long and straight; in pruritus, they are short and irregular; and in scabies, small and round.

(*j.*) *Scales* (*squamæ*) are portions of epidermis which have become separated by diseased processes in the skin. The deeper and more severe the inflammation, the more marked is the desquamation. The scales may be thrown off as fine bran-like particles (as in prurigo, pityriasis, measles, &c.), or as thin flakes or thick plates (in psoriasis and eczema); or the epidermal layer to be thrown off may, as in scarlatina, separate as a whole, forming a more or less perfect cast of the fingers, or even of the whole hand.

(*k.*) *Crusts* are formed by the drying up of the products of skin disease, serum, pus, blood, &c. When chiefly composed of pus, they have a greenish colour; when mixed with blood, the crusts are brown or black. The firmest and hardest crusts are those met with in syphilitic processes (rupia), when they often assume a form closely resembling that of a limpet shell. The crusts of favus are yellow and cup-shaped.

(*l.*) *Fissures* (*rhagades*) in the skin may involve the epidermis alone, or both the epidermis and the true skin; or they may be seated in mucous membrane. They are usually found where the skin is normally furrowed—as, for example, on the palms of the hands and soles of the feet; at the angles of the mouth; where the upper lip and nose join; at the elbows and knees;

at the anus, and in other similar situations. Fissures are found in cases of chronic eczema and inveterate psoriasis, in syphilis, and in scleroderma.

(*m.*) *Ulcers* are chiefly within the domain of surgery. Their size, depth, shape, situation, and general condition should be noted.

(*n.*) *Cicatrices* follow all diseases or injuries of the skin which involve loss of substance. The character of the scar is not indicative of the preceding disease; but sometimes the number or seat of the cicatrices may afford some indication of their cause.

### 4. *The Etiology of the Eruption.*

Skin eruptions are much influenced by the age and sex of the patient, by the season of the year, and by climate. On these points, and on the heredity of many such diseases, we need not now dwell. Very frequently skin affections are the result of constitutional diseases—as, for example, purpura, scrofula, rickets, all the acute exanthemata, diabetes, &c. We have further to note that diseases of particular organs often give rise to skin eruptions. Disorder of digestion from improper diet (shell-fish, for example), or other cause, is frequently followed by urticaria and acne. In valvular disease of the heart, œdema and small hæmorrhagic extravasations (petechiæ) frequently occur. Bright's disease is often accompanied by pruritus, and sometimes by eczema. Many other instances, too numerous to mention here, will occur to the reader in which diseases of the different internal organs are accompanied by skin eruptions which are more or less characteristic.

A class of eruptions with which it is very important that the physician should be familiar are those which result from internal and external use of certain medicines.

All counter-irritants—such as croton-oil, mustard, cantharides, tartar-emetic, iodine, turpentine, arnica, &c.—give rise to various forms of dermatitis; as do also the various aniline colours with which stockings are sometimes dyed.

The internal administration of medicines is occasionally followed by skin eruptions, a result which is most frequently due to some peculiar idiosyncrasy of the patient. Among these may be mentioned the acne pustules which follow the use of the bromides, the erythema (or even eczema) of the iodides, and the scarlatina-like efflorescence of chloral. Very characteristic of atropia-poisoning are the bright erythematous patches which appear on the chest and neck. Morphia sometimes gives rise to an erythematous eruption resembling that of scarlatina, and the administration of quinine is occasionally followed by a rash of the same description. The eruption of copaiba usually shows itself upon the extremities as a bright papular efflorescence, which is generally very itchy. An erythematous rash, of a measles character, sometimes follows the use of antipyrin. The hypodermic administration of tuberculin and of diphtheria antitoxin may be followed by a scarlatina-like rash.

Among the causes acting locally in the production of skin eruptions may be mentioned (in addition to the external applications just noticed) the following:—Continued exposure to the heat of a strong fire is apt to give rise—in furnacemen and cooks, for example—to eczema. Those who work in acids or alkalis, and especially in aniline dyes, suffer much from eczema. Even the long soaking of the hands and arms in hot water and soap produces in washer-women a hardened, fissured, and even eczematous condition of the skin of these parts. The most important local cause of skin eruptions is, however, undoubtedly to be found in the irritation set up by the various parasites which infest the skin and hair. The diagnosis in such cases is closely bound up with the etiology, and they must be considered together. The parasites which affect the skin belong to both the animal and vegetable kingdoms. The most important of these are the following:—

### *Vegetable Parasites.*

1. *Achorion Schönleinii.*—This parasite gives rise to the disease known as tinea favosa or favus. While it occasionally attacks the nails, it is more usually found upon the scalp, where

it gives rise to the formation of light-yellow, dry, cupped crusts. The hair follicle and hair are first attacked, and then the parasite spreads itself upon the surface of the skin. When a part of one of these crusts is examined with the microscope, after having been soaked in water and treated with acetic acid or an alkali, the parasite (fig. 58) is readily recognised. It consists (1) of spores; (2) of slightly elongated elements, which

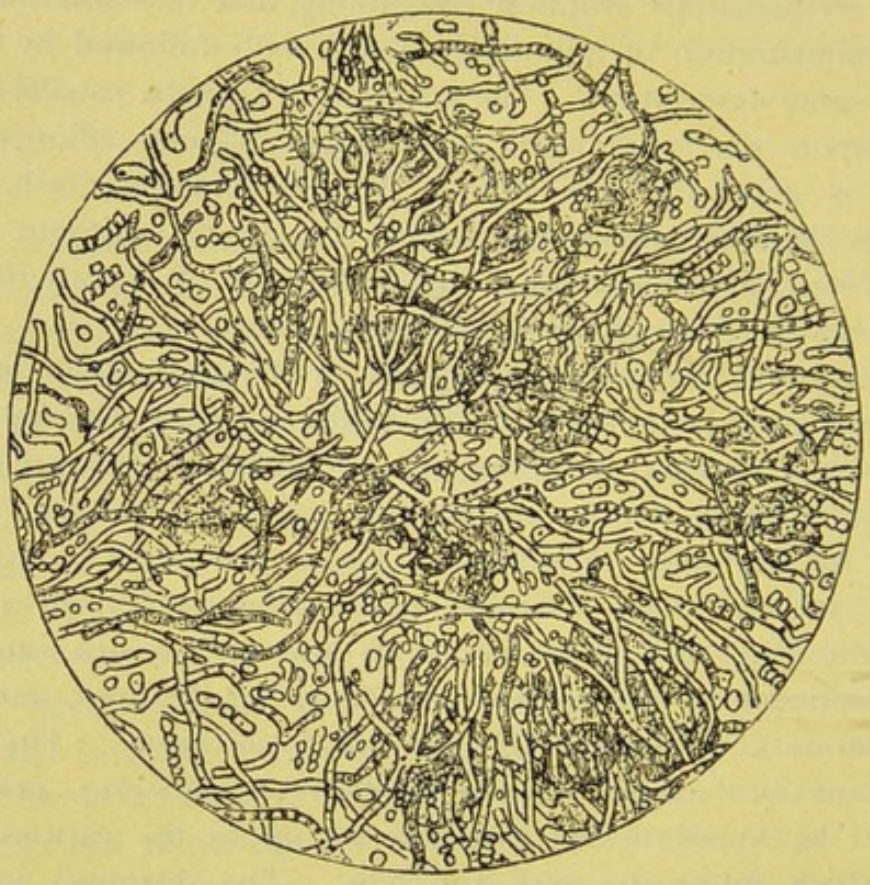


FIG. 58.—Achorion Schönleinii (after Duhring).

are usually united in rows; and (3) of mycelium, which is made up of long, branching, transparent filaments, which may or may not contain spores in their interior. In the favus crust there are always to be found, in addition, numerous micrococci and bacteria.

(2.) *Trichophyton.*—This parasite produces several forms of skin disease, tinea circinata, or ringworm of the body; tinea

tonsurans, or ringworm of the scalp; tinea sycosis, or sycosis parasitica, a similar affection of the beard; tinea cruris, ringworm in the pubic region and thighs, often called eczema marginatum; and onychomycosis, ringworm of the nails. In tinea tonsurans the trichophyton gives rise to considerable irritation of the skin, which results in the formation of circular circumscribed patches of various size, slightly elevated above the level of the skin, of a dull red colour, and usually covered with small branny scales, while round the edges there may be found vesicles, and sometimes even pustules.

On the scalp, ringworm shews itself as one or more circumscribed patches of a greyish or slightly ruddy colour. The hair of the affected parts is short, lustreless, easily drawn out, breaks readily, and the extremities are ragged and uneven. The skin is covered with numerous thin white scales, and occasionally with crusts.

On the beard and upper lip, the parasitic form of sycosis at first exhibits characters closely resembling those of ringworm of the scalp. As the disease advances, however, the skin and deeper parts become inflamed and indurated, and, as a consequence, the affected portions become covered with characteristic tubercular elevations, and pustules occupy the hair follicles.

In all these situations the trichophyton presents somewhat similar microscopic appearances. In the case of tinea circinata, a few of the scales should be scraped off the patch with a penknife, laid on a microscope slide and examined, after the addition of liquor potassæ. In the other forms, a diseased hair should be extracted and examined, after the addition of liquor potassæ or chloroform. Whether in the hair (as in fig. 59) or spread over the surface of the skin, the parasite will be generally found to consist of long, slender jointed filaments (mycelium), together with small, round, highly refractive spores. The latter are most abundant in ringworm of the scalp, infiltrating densely the hair bulb, while the mycelium spreads up the shaft of the hair.

Recent observations, in particular those of Sabouraud, indicate

that there are at least two, if not three or four forms of trichophyton. There is a small-spore variety (Tinea microsporon) met with frequently on the heads of children in obstinate cases. There is also another variety, with large spores (Tinea megalosporon), seen in sycosis and sometimes in tinea circinata, but not so frequently on the scalp.

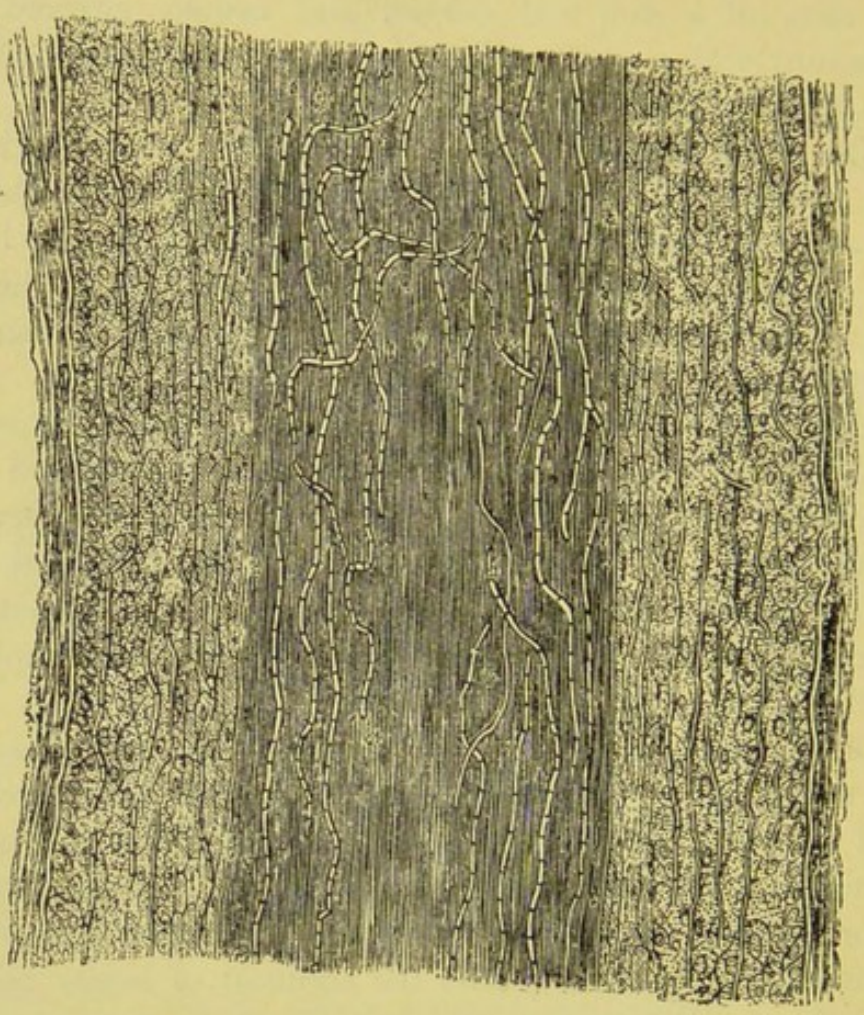


FIG. 59.—Hair affected with Tinea Tonsurans (after Neumann).

(3.) *Microsporon Furfur* is the parasite which gives rise to pityriasis versicolor. This disease is characterised by the presence on the skin (usually of the back and chest) of variously-sized pale yellowish-brown or reddish patches covered with fine powdery scales. It is most frequently met with in those suffering from wasting diseases. The microsporon furfur consists

of spores and mycelium. The spores are small, round or oval, highly refractive bodies, which tend to arrange themselves in groups in a manner which is very characteristic of this parasite. The mycelium consists of fine curved filaments which are usually short and are jointed together, forming a close network. In their interior spores are generally to be seen.

This parasite is very readily detected by means of the microscope. A few of the scales on the surface of the patch should be scraped off with the penknife, placed upon a cover-glass, and treated with liquor potassæ.

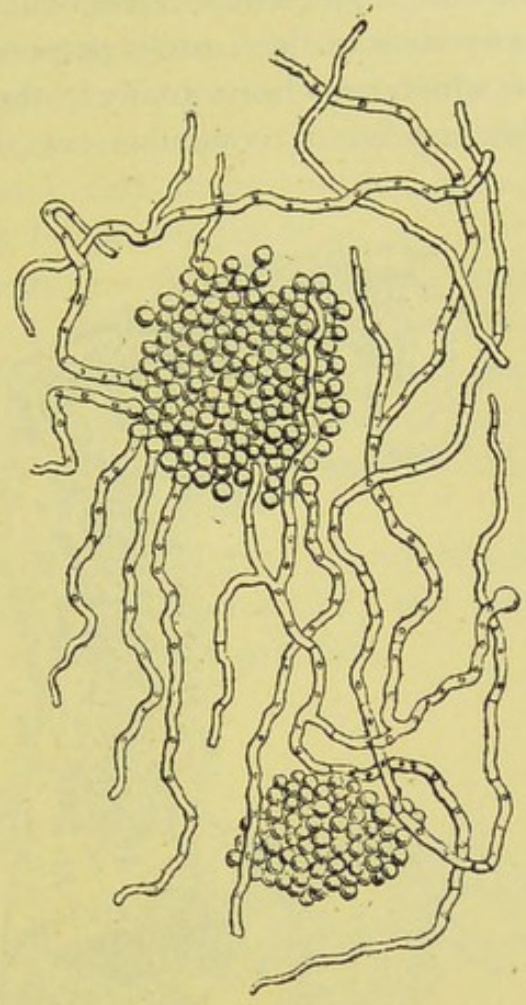


FIG. 60.—Microsporon Furfur (after Neumann).

### *Animal Parasites.*

1. *Sarcoptes Scabiei.*—The skin disease—scabies, or the itch—which is caused by presence of this insect, consists of papules, vesicles, pustules, excoriations, fissures, crusts; in short, an eczema, in the neighbourhood of the burrows in which the insects lie. Scabies is usually found between the fingers, but may spread over the body generally, the insect selecting, however, localities where the skin is soft and thin.

The female insect, which can be seen in fig. 61 lying at the end of its burrow, has an oval body marked with fine undulating lines, a small oval head, and possesses eight legs (the four front legs being furnished with suckers) and numerous bristles. The

male insect does not give rise to the eruption, and is but seldom met with. The female, after being impregnated, bores through the horny layer of the cuticle perpendicularly, and then forms a burrow which runs horizontally in the mucous layer of the epidermis. This burrow, or cuniculus, can often be seen by the naked eye as a whitish elevated line terminating in a minute speck, which is the insect. The cuniculus becomes filled with eggs, of which the insect lays about ten to fifteen, and with small black specks of excrement. The eggs are hatched in about ten days.

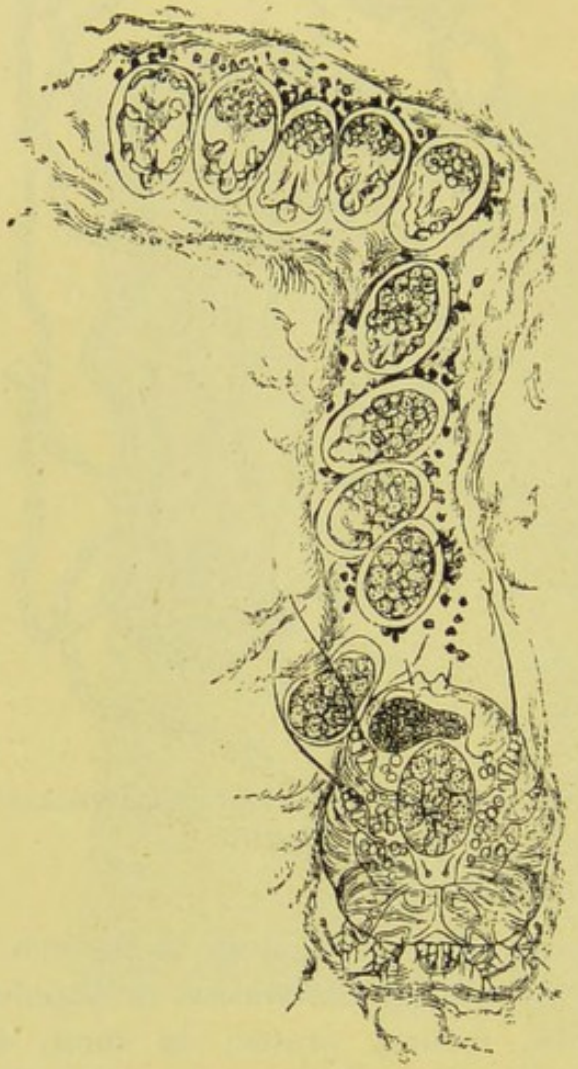


FIG. 61.—Sarcoptes Scabiei (after Neumann).

With a little trouble the itch insect can usually be secured, and the diagnosis thereby rendered certain.

A needle should be introduced into the burrow, which the insect will generally seize hold of, and on the point of which it may be removed.

2. *Pediculus.*—Three varieties of pediculi are met with on the human body, giving rise to the disease termed phthiriasis—the pediculus capitis, pediculus corporis, and pediculus pubis.

The head louse is an elongated ovalish insect of a greyish hue. From its triangularly shaped head spring two antennæ, each consisting of five parts, and to the thorax are articulated

six legs armed with strong claws. The oval whitish eggs are firmly fastened to the hairs of the patient's head by means of a viscid glue-like material. The irritation and itching of the scalp and the consequent scratching give rise to a severe eczematous condition, in which the serous, sanguineous, and purulent exudations mat together the hairs, and almost invariably cause enlargement of the neighbouring lymphatic glands.

The pediculus corporis, or pediculus vestimenti as the insect is sometimes termed, resembles closely the pediculus capitis in structure, but is somewhat larger, and has a more oval head. The cutaneous lesions consist chiefly in long scratch marks, crusts, and papules, along with the minute red points where the insect has bitten.

The pediculus pubis, or crab-louse is smaller and broader than either of the other species. It infests chiefly the pubic region, and usually gives rise to considerable irritation.

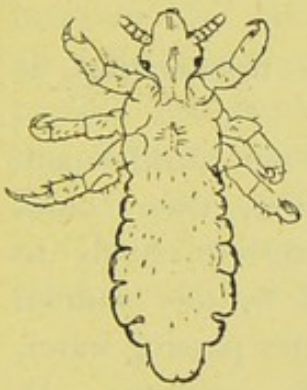


FIG. 62.
Pediculus capitis.

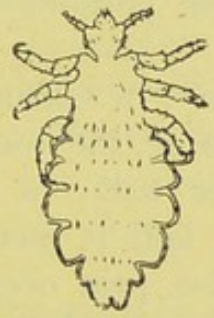


FIG. 63.
Pediculus corporis.
(after Max Braun).

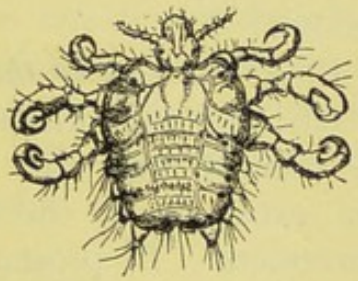


FIG. 64.
Pediculus pubis.

3. *Pulex Irritans*, the common flea, need hardly be mentioned here, were it not that flea bites are occasionally mistaken for purpuric spots. Round the bite, however, there will be seen to be a hyperæmic areola, which is not met with in purpura.

4. *Demodex Folliculorum* is a harmless, worm-like parasite, which inhabits the sebaceous follicles of the skin of the face. If the contents of a prominent follicle be squeezed out and examined with the microscope, the demodex will frequently be found.

## CHAPTER XXV.

# URINARY SYSTEM.[1]

### SUBJECTIVE SYMPTOMS.

BEFORE proceeding to the consideration of the various changes met with in the urine in disease, which must always rank as the most important sign of urinary disorders, it may be well to note certain subjective symptoms which occur in such cases, and which often give valuable indications.

**Pain** may be felt at different portions of the urinary tract, as follows :—

1. *At the end of the Penis.*—In calculus of the bladder pain is felt after micturition, because the rough stone then comes in contact with the bladder wall; it is referred chiefly to the extremity of the penis, and is increased by any sudden movement. In prostatitis, also, pain occurs after passing water, the bladder then contracting on the tender prostate. In women there is often severe pain felt during micturition at the orifice of the urethra, owing to the presence there of a small vascular growth.

2. *In the course of the Urethra.*—When the urethral canal is narrowed by stricture, pain is felt at the constricted point during micturition. In urethritis, also, the pain during the passing of water is referred to the urethra. When the urine is highly

[1] For the reasons given in the preface, it has been thought advisable, in the following chapters, to retain certain of the references to original authorities. These, which are of course far from complete, may serve the purpose of suggesting to the reader the sources from which he may obtain more detailed information on the subjects dealt with in these pages.

acid, concentrated, or contains gravel, urethral pain may occur during micturition.

3. *Over the Bladder in the Supra-Pubic Region.*—This is the common seat of the pain of cystitis, which, it is to be observed, occurs before micturition, and is relieved by that act. In acute cases pain may also be felt deep in the perineum.

4. *In the Loins.*—In cases of pyelitis and of renal calculus there is usually dull aching pain over the loins, which is increased on pressure, and which in the latter disease occasionally passes into violent paroxysms, the pain shooting down the ureters to the testicle and inside of the thigh.

The nervous mechanism of micturition will be considered in a future chapter.

**Frequency of Micturition.**—Whenever the urine is large in quantity, as in diabetes and the waxy form of Bright's disease, for example, there is frequency in micturition. This symptom, however, also occurs in very many other urinary disorders. In all inflammatory conditions of the prostate and bladder, in pyelitis and nephritis, in calculus of the bladder or kidneys, the urine is frequently voided. It is particularly to be noticed that in the cirrhotic or contracting form of Bright's disease, and in hypertrophy of the prostate, the calls to micturate are frequent and occur chiefly during the night.

### *The Examination of the Urine.*

In such a work as this it is of course quite impossible to give an exhaustive account of the many changes which take place in the urine in health and disease, or of the various methods of analysis which have been applied to that secretion. All that can be attempted is to enumerate the more ordinary and clinically significant changes which occur, and the simpler methods of analysis, such as may be carried out by the physician, excluding those which require the more complicated apparatus of a chemical laboratory.

In the present chapter we shall consider the general condition of the urine as to (1) quantity, (2) colour and transparency, (3) odour, (4) specific gravity, and (5) reaction.

**Quantity of the Urine.**—While varying according to the quantity of fluid drunk, the amount of the pulmonary and cutaneous transpiration, and of the alvine discharge, the average quantity of urine voided in twenty-four hours may be taken to be in the adult from 35 to 60 ounces.

The quantity is diminished in all febrile diseases, in heart affections when compensation is lost, in cases of collapse, and generally in all those conditions in which much fluid is passing out of the blood, such, for example, as profuse diarrhœa or perspiration, the rapid accumulation of serum in the pleuræ or peritoneum, &c. Further, there is scanty urine in the inflammatory form of Bright's disease whether there be inflammation of the tubules or of the glomeruli. The quantity may also be diminished in cases of hysteria, and under the action of such drugs as morphia, or cantharides. The urinary flow may be completely suppressed in cases where the ureters are occluded by the impaction of calculi, or by the pressure of morbid growths. Suppression of urine is sometimes seen in hysteria, in acute fevers such as cholera and septicæmia, in collapse from any cause, and in severe cases of acute nephritis.

The urinary flow is *increased* by the administration of diuretics. It is greatly augmented in cases of diabetes insipidus and mellitus, chiefly owing to the large quantities of water which such patients drink. In the cirrhotic, or contracting form of Bright's disease, the quantity of urine secreted is augmented in the later stages, when the heart has become hypertrophied, and the vascular tension increased. On the other hand, in the waxy form polyuria is an early symptom, often occurring even before the presence of albumen can be detected. An increase in the quantity of urine is sometimes met with in nervous affections, particularly in hysteria.

**Colour of the Urine.**—The urine owes its colour to the quantity of pigment it contains, and to the amount of its concentration—very dilute urine being pale, very concentrated having a dark brownish-red colour. For convenience of com-

parison, Vogel's standard scale of colours is usually adopted. The various tints are grouped as follows:—

| | |
|---|---|
| Yellow Urines, | Pale Yellow,<br>Bright Yellow,<br>Yellow. |
| Red Urines, | Reddish yellow,<br>Yellowish-red,<br>Red. |
| Dark Urines, | Brownish-red,<br>Reddish-brown,<br>Brownish-black. |

In order to obtain uniform results as to colour, the urine should be examined in a glass, the diameter of which is about four inches; and if not absolutely clear, the urine should be filtered before its colour is noted.

Very pale urines are met with in healthy persons after copious draughts of water, and further, in cases of diabetes and of anæmia, and after hysterical paroxysms. Highly-coloured urines occur in the febrile state, and under other pathological conditions, which will be mentioned more particularly hereafter.

There is still a great deal of obscurity regarding the various pigments of the urine, indeed it is not accurately ascertained what are the pigments which give to normal urine its peculiar yellow colour. Something may however be said about certain pigments which are known to appear,—chiefly pathologically—in the urine.

*Urobilin.*—This is a reddish pigment, closely allied to bilirubin and to blood pigment, which occasionally appears in very small quantity in the urine under normal conditions, but more often can only be detected after the urine has stood for some little time. In this latter case it is supposed that the urine when passed contains a substance, "urobilinogen," which on being oxidized passes into Urobilin. A product of the oxidation of bile-pigment, Urobilin resembles Hydrobilirubin very closely, both in its reactions and in its spectrum, but it is believed that the two substances are not identical.

Urobilin appears in the urine in considerable quantities under various pathological conditions. It is found in cases where blood has been extravasated into the tissues and its being absorbed, and also in such diseases as lead to destruction of blood corpuscles. Thus Urobilin is found in the urine in cases of fever, of pernicious anæmia, and the like, and as the result of various poisons. But it is in connection with jaundice that Urobilin is most prominently seen. Sometimes, in cases of obstruction of the bile duct, Urobilin is found in large quantity in the urine, to the exclusion, it may be, of bile pigment proper. This condition has been termed "Urobilin Icterus."[1] In it, not only is the urine rendered dark in colour by the presence of Urobilin, but that substance stains the skin a dirty yellow, differing somewhat from the yellowish-green or the lemon-yellow of ordinary jaundice.

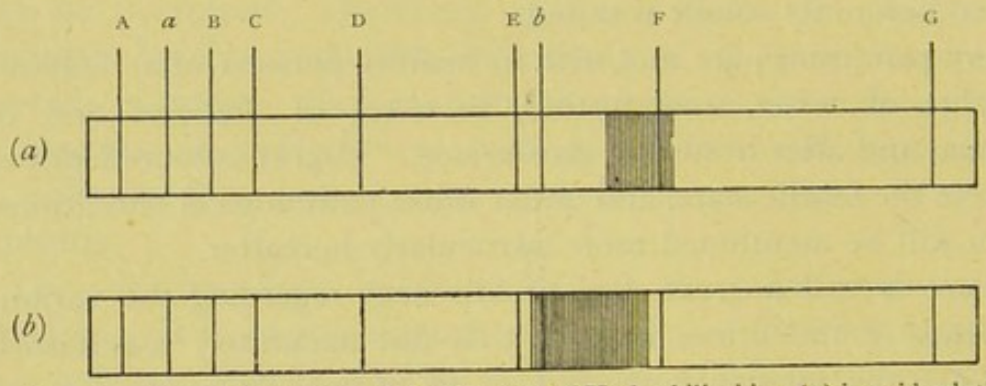



FIG. 65.—Absorption bands of Urobilin and of Hydro-bilirubin; (*a*) in acid solution, (*b*) in ammoniacal solution after the addition of chloride of zinc.

The *detection of uro-bilin* is usually easy, and depends chiefly upon the three following points:—(1) Examined with the spectroscope, most urines which contain uro-bilin show an absorption band between Frauenhofer's lines *b* and F, which is not very well defined, and which shades away towards F. Sometimes, however, the spectrum cannot be made out in the urine itself. In such cases, if the urine be shaken up with ether, the etherial solution of the pigment will show the spectrum clearly. (2) When a small quantity of chloride of zinc is added to an

[1] Compare Grimm "*Ueber Urobilin im Harn*," Virchow's *Archiv.*, 1893, p. 246, and Katz, "*Die klinische Bedeutung der Urobilinuria*," *Wiener med. Woch.*, 1891, Nos. 28-32.

alkaline solution of the pigment, or to a urine containing it, which has been rendered alkaline by the addition of ammonia, a green fluorescence appears.

The view of Macmunn [1] that normal and pathological urobilius are different substances, is not confirmed by recent work.[2]

*Uro-erythrin* is a pinkish-red pigment which often appears in the urines of fever, and of cirrhosis of the liver, and which attaches itself to precipitates of urates and of uric acid, giving the sediment a brick-dust colour. This deposit, however, often occurs in otherwise healthy persons, from errors in diet and other slight causes.

*Indican* is present in small quantity in normal urine, and is somewhat increased when much animal food is taken. In the putrefactive decomposition of albumin there is formed a substance called indol, which, by oxidation in the body, passes into indoxyl and appears in the urine as indoxyl-sulphate or indican. Thus, as might be expected, whenever such albuminous putrefaction is taking place, indican appears, in more or less considerable quantities, in the urine. Now, there are mainly two sets of conditions under which this may occur. There are, first of all, those causes which lead to increased putrefaction in the contents of the intestine, such as constipation, obstruction, typhoid fever, &c.; and secondly, there are the conditions under which collections of pus, either in abscess cavities or in one of the serous sacs, have become septic. In such cases, therefore, indican appears in the urine.

The best test for the presence of indican is that of Jaffé which consists in mixing an equal quantity of concentrated hydrochloric acid with the urine to be tested. When a few drops of a solution of chloride of lime are added, indigo blue is formed; and when this mixture is shaken up with chloroform it will be found that when the chloroform separates it has dissolved the pigment and has assumed a beautiful blue colour. It is important to notice that if any excess of chloride of lime

[1] *Journal of Physiology*, vol. x. (1889) p. 71.

[2] Garrod and Hopkins "On Urobilin," *Journal of Physiology*, vol. xx. (1896) p. 131.

be added it causes a further oxidation of the indigo which prevents the reaction. To obviate this the modification suggested by Obermayer[1] may be made use of. In this process a solution of acetate of lead is added to the urine so long as any precipitate forms. To the filtrate is added an equal volume of a solution made by mixing 0·5 c.c. of a concentrated solution of ferric chloride with 200 c.c. of hydrochloric acid. When chloroform is added to this it dissolves out the indican.

Indigo itself is very rarely seen in the urine, but occasionally when the urine has been allowed to stand and has decomposed, it is formed, and then appears on the edge of the glass and on the surface of the urine as a glistening dark blue film.

Nearly allied to indol is Skatol which appears in the urine in the form of Skatoxyl-sulphate. It also is a product of the putrefactive decomposition of albumin, and while it occurs in normal urine in very small quantity, it is occasionally much increased, especially in the case of disease of the large intestine.

*Hæmato-porphyrin*, a derivative of hæmatin, but containing no iron, and isomeric with bilirubin, is said to occur frequently in healthy urine though in very minute amount. This pigment when present in some quantity, imparts to the urine a dark, almost black colour. When thin layers are examined, the tinge is found to vary from yellowish-red to violet. Hæmato-porphyrin has been found in the urine of a considerable variety of diseases, particularly in typhoid fever, and after the administration of sulphonal, trional and tetronal. According to recent observations of Stokvis[2] the pigment appears in the urine in cases where blood, which has been poured out into the digestive tract, is being absorbed.

The detection of hæmato-porphyrin appears to be best carried

[1] Obermayer, *Ueber eine Modifikation der Jaffé'schen Indicanprobe.* Wiener klin. Wochenschr. 1890, p. 176.

[2] Stokvis, "Zur Pathogenese der Hæmatoporphyrinurie," *Zeit. f. kl. Med.*, vol. xxviii. (1895) p. 1. This article contains references to the chief papers on the subject.

out by means of the method described by Garrod.[1] The urine is precipitated with potassium or sodium hydrate, and the precipitate extracted with acidulated alcohol. The spectrum is characteristic. In acid solution, hæmato-porphyrin gives two bands, one between C and D, close to D, and a broader and darker band between D and E, nearer to D. In alkaline solution the pigment shews four bands; one between C and D, near D; a second and third between D and E, one near D and the other near E; and a fourth band, which is broad and very dark, between b and F. Finally, it may be noted that hæmato-porphyrin contains no albumin, and may in this way be distinguished from hæmoglobin.

*Melanin*, the black pigment which is found in the urine in cases of melanotic cancer, may at times possess some diagnostic significance.

The administration of certain drugs is followed by alteration in the colour of the urine. Thus, after the absorption of carbolic acid, the urine becomes of a dark greenish-brown colour, due to the presence of an oxidation product of hydrochinon. Rhubarb and senna (chrysophanic acid) colour the urine a deep brownish-yellow, which changes to bright red on the addition of an alkali. Logwood imparts a red tinge, and santonin a bright yellow which changes to orange when ammonia is added.

The presence of blood and bile pigments in the urine will be considered hereafter.

**Transparency.**—Normal urine, when freshly passed, is almost invariably transparent; but when allowed to stand, clouds of mucus form in it, which at the end of twelve hours, will be found to have sunk to the bottom of the vessel. In highly-concentrated urine, and especially in that of the various feverish processes, a dense cloud of urates forms after cooling has taken

[1] Garrod, A. "On the occurrence and detection of Hæmatoporphyrin in the urine." *Journal of Physiology*, vol. xiii. (1892), p. 598. Other papers on this subject by Garrod appeared in the same Journal during the years 1893 and 1894, and in the *Journal of Pathology and Bact.*, 1892.

place, which, as well as other urinary sediments, such as phosphates, pus, bacteria, &c., which render urine cloudy, will be considered farther on in these pages.

**Odour.**—Freshly-passed normal urine has a faint odour peculiar to itself, which gradually disappears. When it becomes alkaline, and carbonate of ammonia is formed from the decomposition of urea, an ammoniacal odour develops. When blood or pus becomes added, the urine has a peculiarly offensive odour from its rapid decomposition.

Turpentine, when inhaled or taken internally, imparts an odour of sweet violets to the urine. Copaiba, cubebs, tolu, and asparagus, also communicate a characteristic smell. Finally, in diabetes mellitus, the urine has a faint, sweetish odour, which, if acetonæmia develops itself, comes to resemble that of chloroform.

**Specific Gravity.**—The specific gravity is usually and most conveniently estimated by means of a urinometer. The instrument is dipped into the urine and allowed to float, the point at which the level of the surface of the urine cuts the graduated stem being read off, and thus the specific gravity is ascertained. One or two precautions must, however, be taken. The urinometer must be carefully dried before use, as drops of water adhering to the upper part of the stem tend unduly to depress it. It must also float completely clear of the side of the vessel. As urinometers are graduated for a temperature corresponding to that of an ordinary room (60° F.), observations made on urines before they have cooled down to that point are inaccurate.

The urinometer scale commences at 1000, the specific gravity of distilled water, and usually goes up to 1050. The average specific gravity of normal urine may be taken to be from 1015 to 1025; but readings both above and below these limits are quite consistent with perfect health. The specific gravity of any urine expresses, of course, the quantity of solids which that urine contains in solution. Thus, if we find it in any

particular instance to be, let us say, 1025, we know that there are present solids in such quantity as to suffice to raise the weight of a litre of distilled water from 1000 grammes to 1025.

From the specific gravity so obtained, it is possible roughly to calculate the quantity of solids present in the urine. This may be done by means of the very simple formula given by Trapp, which consists in multiplying the two right hand figures by 2, the result being the amount of the solids in grammes in 1000 c.c. of the urine.

The total quantity of solids present may be accurately ascertained by weighing. A measured quantity of urine is placed in a platinum capsule (the weight of which has been already ascertained), and evaporated over a water-bath. When this operation is complete, the capsule with its contents is weighed, and when, from the figure so obtained the weight of the capsule itself is subtracted, the figure which remains represents the weight of the solids contained in the quantity of urine used. From what has been said, it is clear that as the specific gravity of the urine depends upon the proportion of solids to fluid, it will be affected by changes in the quantity of either. Thus, after copious imbibition of water, the urine of healthy persons may have a specific gravity as low as 1002 ; and, on the contrary, after profuse perspiration, it may rise to 1030 or more. We must thus take into account the *quantity* of the urine passed in twenty-four hours before we allow ourselves to judge what importance is to be attached to the specific gravity. When the quantity is large, we find, if the urine be normal, a low specific gravity ; whereas, when the flow is scanty, the specific gravity is high. If, however, we meet with a urine which, while large in quantity, possesses a high specific gravity, or one which, while small in amount, is low in gravity, then the fact may in each case be noted as distinctly pathological.

Pathological urines may be classified as follows :—

*High Specific Gravity* is found after copious perspiration, vomiting or purging, owing to the consequent concentration of the urine. At the commencement of all acute feverish diseases, the specific gravity of the urine is high, running up

even to 1035, and this, owing, in part, to diminished watery excretion, but also, in great measure, to the increased elimination of urea, sulphates, and phosphates which then takes place. Much more marked and important, however, is the increase of specific gravity met with in the urine of diabetes mellitus. In this disease we find large quantities of urine being passed, the specific gravity of which varies from 1030 to 1060, due to the presence of grape sugar.

*Low Specific Gravity*, when not due to great dilution of the urine, is commonly the result, either of some disturbance of the secreting apparatus of the kidney (Bright's disease, circulatory disease, &c.), or of general interference with nutrition (anæmia, cachexia, &c.), in both cases arising directly from the defective elimination of the urinary salts, particularly urea and its compounds.

**Reaction.**—The reaction of the urine may be tested by means of blue and red litmus paper. Normal urine is acid when fresh, the acidity being due to the presence of acid salts, particularly acid phosphate of soda. During the process of digestion, on account of the outpouring of hydrochloric acid into the stomach,[1] the urine loses in acidity, sometimes becoming neutral, or even alkaline; but it very rapidly regains its former character. The consumption of much vegetable food tends to make the urine alkaline, while animal food has an opposite effect. The administration of alkalis renders the urine alkaline, while that of the mineral acids produces an acid reaction. Such acids as these belonging to the fatty group, which break up and are excreted as alkaline carbonates, render the urine alkaline. Alkalinity of the freshly-passed urine may either be due to a fixed alkali,[2] or to

[1] The hydrochloric acid is formed in the gastric epithelium from chloride of sodium, and, as a result, basic carbonate of soda remains. Were this allowed to circulate in the blood, that fluid would become too alkaline. It is therefore excreted by the kidneys and renders the urine alkaline. See an interesting paper by Frau Prof. Schoumow-Simanowsky, *Arch. f. exp. Path. und Pharm.*, vol. xxxiii. (1895) p. 336.

[2] If the alkalinity be due to the presence of ammonia, the red litmus paper, which has been turned to blue by dipping in the urine, will regain its red tint after drying; but if the alkali be a fixed one, the blue tint will be permanent.

the presence of ammonia, resulting from the breaking up of urea under the influence of micro-organisms. The latter form which is the more common, points to some local disease in the bladder or urethra. Ammoniacal urine is frequently met with in cases of long-standing urethral stricture, chronic cystitis, spinal affection, &c., or may be due to the use of a catheter which has not been rendered thoroughly aseptic.

Alkaline urine, due to the presence of fixed alkali, may result from a variety of causes. Wherever, either from persistent vomiting or from washing out of the stomach, there is removal of acid from the body, the urine loses acidity or becomes alkaline. On the other hand, the alkalinity which is met with in chlorosis is believed to be due to deficiency in the formation of acid. As a result of the rapid absorption of transudations, or of blood which has been poured out into the intestine, the alkaline salts they contain may be thrown in quantity into the urine, and so produce alkalinity. And it is to be remembered that the urine in passing through the urinary tract, may have alkaline secretions added to it, as happens in the case of cystitis.

Increased acidity of the urine is met with in fevers, especially in acute rheumatism, and generally where there is increased destruction of the albumin of the tissues.

The amphoteric reaction, that in which the urine turns red litmus blue, at the same time, turns blue litmus red, is due to the simultaneous presence of the acid phosphate of soda, and of the alkaline basic phosphate.

Normal urine undergoes, when kept too long, fermentative changes which, as they are liable to cause mistakes, must be carefully noted.

1. *Acid Fermentation.*—If the urine be allowed to stand exposed to the air, in a cool place, it will be found that its reaction increases in acidity steadily from day to day. This is accompanied by the precipitation of a yellowish-brown sediment, consisting of uric acid and urates, and frequently of oxalate of lime, along with clouds of mucus.

2. *Alkaline Fermentation.*—After the acid reaction has fully developed itself, it gradually disappears, and the urine becomes

alkaline. This change does not usually set in, when the urine is kept cool, until some ten days have passed, but if there be much pus or mucus present, the alkaline reaction may be detected much sooner; and if any admixture of old, decomposed urine be allowed to take place (as from the glass not having been thoroughly cleaned), it may come on in a few hours. The urine now becomes lighter in colour, opaque, and ammoniacal in odour, the urea having become changed by the action of various forms of micro-organism, into carbonate of ammonia. A white sediment separates, consisting of urate of ammonia, triple phosphate, amorphous phosphates, and carbonate of lime.

**Quantitative Test for Acidity.**—A measured quantity of the mixed urine for twenty-four hours, say 100 c.c., is placed in a porcelain capsule, and a few drops of an alcoholic solution of phenol-phthalein is added. This is then titrated with a deci-normal soda solution. Just as the point of neutralization is passed, the phenol-phthalein, which is colourless in acid solutions, turns suddenly red. The urine may, with advantage, be previously decolorised by shaking it up with animal charcoal. According to Lieblein [1] the accuracy of this method is interfered with by the presence of acid phosphates, which prevent the colour change occurring correctly. The method which Neumeister [2] recommends is as follows. To 50 c.c. of urine are added 25 c.c. of normal soda solution, and the strongly alkaline mixture is heated to boiling, and 25 c.c. of a solution of barium chloride, strong enough to cause complete precipitation of all the phosphoric acid, is added. After filtration, 50 c.c. of the mixture (corresponding to 25 c.c. urine) is taken, a few drops of an alcoholic solution of phenol-phthalein is added, and it is titrated with deci-normal sulphuric acid, until the indicator shews neutrality. The smaller the quantity of sulphuric acid used, the more acid was the original urine.

[1] Lieblein, "Ueber die Bestimmung der Acidität des Harns," *Zeit. f. phys. Chemie*, vol. xx. (1895) p. 81. Also vol. xxi. (1896) p. 97.

[2] Neumeister, *Lehrbuch*, vol. ii. (1895) p. 225.

# CHAPTER XXVI.

## URINARY SYSTEM—(*continued*).

### UREA AND OTHER PRODUCTS OF NITROGENOUS METABOLISM.

NITROGEN is eliminated in the urine in several forms. Somewhere about 89 per cent. is excreted in the form of urea, and the remaining 11 per cent. as uric acid, xanthin, creatinin, and the like. As is well known, these substances are not formed in the kidney, repeated observations having shewn that when both kidneys are removed in animals, urea accumulates in the blood rapidly, and to considerable amount. The products of nitrogenous metamorphosis leave the tissues chiefly in the form of lactate of ammonia, and this, passing to the liver, is there oxidised to carbonate of ammonia, from which, by synthesis in the liver cells, urea is formed. The accuracy of this supposition rests on both experimental and clinical observation. It has been known for some time[1] that in the goose, where the operation is comparatively easy, when the liver is cut out of the circulation, nitrogen is excreted in the form of ammonium lactate instead of uric acid as is usual in the bird. Lately it has been found possible to perform a similar operation on the dog, and in a paper by Hahn, Massen, Nencki and Pawlow,[2] its results are described. They found that the urea decreased, while there was a corresponding increase in the excretion of the ammonia salts. It may also be mentioned that, as von Schroder[3] shewed in 1882, when serum

[1] Minkowski, "Beiträge zur Pathologie der Leber und des Icterus: Ueber den Einfluss der Leberextirpation," &c., *Arch. f. exp. Path. und Pharm.*, vol. xxi., p. 41.

[2] "Die Eck'sche Fistel," &c., *Arch. f. exp. Path. und. Pharm.*, vol. xxxii. (1893) p. 161.

[3] Schroder, *Arch. f. exp. Path. und Pharm.*, vol. xv., p. 364.

containing ammonium lactate is circulated through the liver of the dog after its removal from the body, urea is produced.

There is, further, clinical evidence in support of this view, for, in conditions in which the liver is deeply involved, such as acute yellow atrophy and phosphorus poisoning, there is a decrease of urea in the urine and an increase of ammonia.

As has been said, urea represents about 89 per cent. of the nitrogen excreted in the urine. The remaining 11 per cent. consists of uric acid, creatinin, and the xanthin bases, mainly the first of these. Uric acid is not formed, as in the case of urea, by synthesis in the liver. It takes its origin in all the tissues of the body, in the process of breaking up of old cells, and particularly of the nuclei of these cells, from the nuclein bases of which uric acid is formed. Hence, the richer any organ is in lymphatic tissue, (the spleen, for example), the more will it serve in this way. These nuclein-bases, which include the following, xanthin, guanin, hypoxanthin, paraxanthin, adenin, heteroxanthin, &c., are in part excreted as such in the urine, in part oxidised to form uric acid. What further happens depends on whether the organ in which the cell destruction is occurring, lies on the portal system, or in the general circulation. In the former case the uric acid, carried to the liver, is oxidised to carbonate of ammonia, and, by synthesis, urea is formed. In the latter case the uric acid, not traversing the liver, does not undergo these changes, but appears in the urine as uric acid or its salts. Hence that which appears in the urine represents only a small part of the total uric acid which is formed in the tissues by the breaking up of cells, because those organs in which this process occurs in largest measure, lie within the portal circulation.

The total nitrogenous elimination in the urine is not appreciably affected by exercise, but is increased when much nitrogenous food is taken. It is markedly increased in diseases, such as fevers and diabetes, which lead to a heightened metabolism. It is, on the other hand, diminished in cases where the excretory functions of the kidneys are affected, as in the cirrhotic form of

Bright's disease. As a rule (to which however acute yellow atrophy is a marked exception), the total nitrogenous elimination lies fairly parallel to that of urea.

The best method of estimating the total nitrogen in the urine is that of Kjeldahl, which is conducted as follows.

The urine is filtered, and 5 c.c. carefully measured and placed in a small flask of hard glass. To this is added a little (·3 grm.) yellow oxide of mercury, which hastens the reaction, and 10 c.c. of a mixture of two parts of pure sulphuric acid with one part of Nordhausen sulphuric acid. The mixture is then gently boiled until it becomes colourless, which usually requires about half an hour, and is then allowed to cool. The contents of the flask are washed out into a large flask of 750 c.c. capacity, using as little distilled water as may be for the purpose of washing; and arrangements are made to connect this flask with a suitable apparatus for distilling.

It will be understood that, so far as the process has yet been described, what has happened is this, that, by boiling with sulphuric acid all the nitrogenous substances in the urine have been oxidised and broken up, and thus the total nitrogen in the fluid is now in the form of ammonium sulphate. The further process consists in over saturating the acid mixture with caustic soda, to which a little sulphate of lime has been added, and thus setting free the ammonia. By connecting the flask with a suitable condensing apparatus this ammonia is distilled over and received in a small flask containing a measured quantity of $\frac{1}{5}$ normal sulphuric acid. The distillation requires to be continued for some time, until the fluid which passes over is found to be neutral when tested with litmus paper. The ammonia has now passed completely over, and has combined with a certain quantity of the sulphuric acid. It is only necessary to ascertain how much of the sulphuric acid has thus combined, in order to know how much nitrogen the original specimen of urine contained. This is done by titration with $\frac{1}{5}$ normal soda solution, using cochineal as an indicator, 1 c.c. of the $\frac{1}{5}$ normal sulphuric acid combines with the quantity of ammonia corresponding to 0·0028 grammes of nitrogen.

There are various modifications of Kjeldahl's process. In the details just given I have followed Neumeister.[1]

**Urea.**—Urea is, then, by far the chief product of the nitrogenous tissue change which is taking place in the body, and its increase or decrease is an index of the amount of such change.

*The estimation of urea*, for ordinary clinical purposes, is usually performed by means of the hypobromite method, in one or other of its forms. This method depends upon the fact that urea when treated with hypobromite of soda breaks up into nitrogen, water, and carbonic acid, the last of which is absorbed in the alkaline solution, while the nitrogen comes off as free gas. Of the many forms of apparatus which have been described and are used for this analysis, perhaps the most simple is that of Graham Steele.[2] It consists of an ordinary burette inverted in a tall glass cylinder containing water, and connected by means of an indiarubber tube, with a small conical glass vessel containing a short test-tube. After removing the test-tube, the conical vessel is filled to the depth of about an inch with the solution of hypobromite of soda (the method of preparing which will be presently described), and into this the test-tube is carefully slipped, after 5 c.c. of the urine to be tested have been placed in it. The cork of the conical vessel is then replaced, and the vessel dipped in water. The burette, which is now in communication with the conical vessel, is next raised or lowered as may be required, until the level of the water inside and outside is the same, and this point is read off. The conical vessel is now tilted over so as to allow the urine in the test-tube to flow out and to become mixed with the hypobromite solution. This mixture is followed by a rapid giving off of gas, and after all effervescence has ceased, and the nitrogen which has collected in the burette has had time to cool down to the temperature of the room, the burette is again moved so as to bring the water-level inside to the same height as that outside, and this point read off. The difference of the two readings gives the quantity

[1] *Lehrbuch der physiologischen Chemie*, vol. ii. (1895) p. 237.

[2] *Edin. Med. Journal*, 1874.

of nitrogen which has been given off. Since we know that under ordinary circumstances 1 gramme of urea gives off 354·3 c.c. of nitrogen,[1] the calculation is simple. This method, from the ease with which it can be carried out, is very convenient, but it is not extremely accurate, for not only urea, but also uric acid and creatinin, give off nitrogen when treated with hypobromite of soda. The error is, however, small. The preparation of the solution of hypobromite of soda is made as follows:—100 grammes of caustic soda are dissolved in 250 c.c. of water, making a 40 per cent. solution. To this 25 c.c. of bromine are to be added in a glass tube in which it is supplied and which is to be broken under the soda solution, and the whole shaken vigorously. This solution must be kept in a stoppered bottle and in the dark. It decomposes rapidly, and can only be used when freshly prepared. It is therefore better, in many cases, to prepare smaller quantities at a time. Tubes are sold containing 2 c.c. of bromine, one of which broken in 23 c.c. of the soda solution is sufficient for one urea determination.

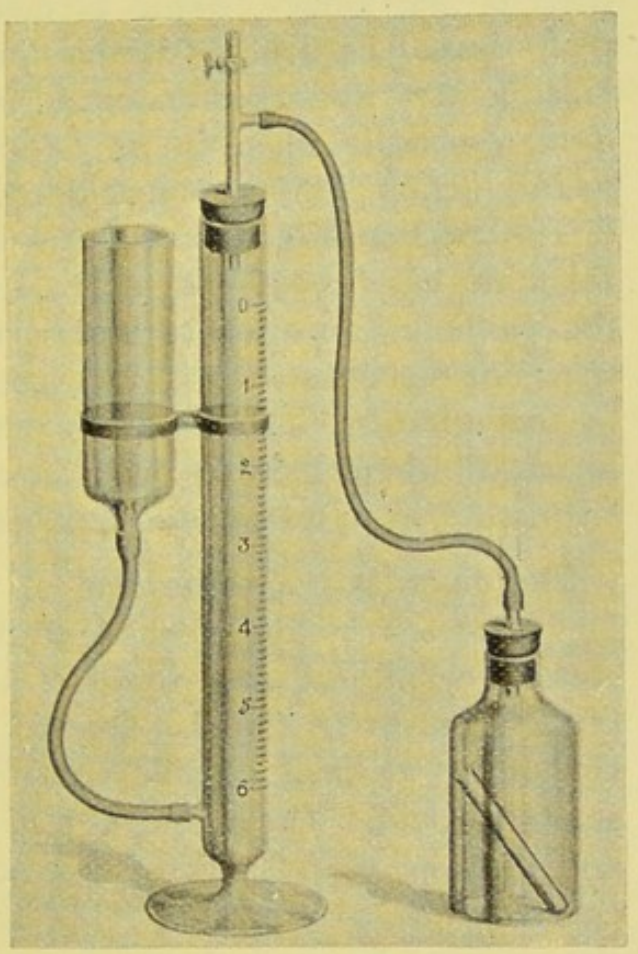


FIG. 66.—Gerrard's Apparatus for estimating Urea.

Another very convenient apparatus for the purpose is that of Gerrard (see fig. 66).

The quantity of urea excreted in twenty-four hours in healthy men on ordinary mixed diet, averages about 32 grammes, say

[1] In theory it should give somewhat more.

500 grains. It is increased after a full meal of animal food. In almost all diseases attended with elevation of temperature, the urea elimination is increased. Thus in typhus, pneumonia, pleurisy, and acute rheumatism, the amount of urea excreted is usually much above normal. In diabetes mellitus, the daily quantity may be very high. This is no doubt in part due to the large quantity of animal food consumed in this disease. On the other hand, the urea is diminished in almost all affections of the kidney, owing to defective eliminating power of that organ. Particularly is this the case with regard to acute inflammatory Bright's disease, and to the cirrhotic or contracting form, especially when in its later stages a degree of inflammatory action becomes superadded.

The relation which urea bears to the salts of ammonia in the urine has been already referred to. In many liver disorders the synthetic formation of urea is interfered with, and in consequence that substance is, to a greater or less extent, replaced in the urine by the salts of ammonia. These salts are also increased where there is much breaking up of the albumins of the tissues, as in fevers and in diabetes.

**The quantitative estimation of the salts of ammonia** may be performed by the method of Schlösing, as follows. In a shallow cylindrical glass vessel of small size are placed 25 c.c. of the urine to be tested. On this vessel rests a glass triangle, supporting a small capsule containing 25 c.c. of $\frac{1}{5}$ normal-sulphuric acid. The whole is placed on a slab of plate-glass, and, after 20 c.c. of milk of lime has been added to the urine, covered with a bell-glass. This latter should be as small in size as may be, and should be well larded round the edge. The ammonia which the urine contains is set free and is absorbed by the sulphuric acid. After three days the process is complete. The bell-glass should then be removed, and the sulphuric acid taken and titrated with $\frac{1}{5}$ normal soda solution, cochineal being used as an indicator.[1]

[1] On this subject see further, Nencki and Zaleski, "Ueber die Bestimmung des Ammoniaks in thierischen Flüssigkeiten und Geweben," *Arch. f. exp. Path. und Phar.*, vol. xxxvi. (1895) p. 385.

**Uric Acid** exists in normal urine in combination with potassium, sodium, ammonium, calcium, or magnesium; and, as all these salts of uric acid (which according to Roberts[1] are quadri-urates), are very much more soluble in hot than in cold urine, they tend to separate out as the urine cools. The cloud of urates which thus so often appears soon after the urine is passed may be readily recognised by warming a small quantity of the urine containing the sediment in a test-tube, when it rapidly becomes clear. Uric acid is readily separated from urine by adding hydrochloric acid. It then deposits itself in crystalline form, the character of which will be described when we come to speak of urinary sediments.

*Detection of Uric Acid.*—It is often important to be able to detect the presence of uric acid in concretions and sediments. Very frequently this may be done by means of the microscope, but this is not always possible. Uric acid can, however, always be detected by means of the *murexid test*, which is applied as follows:—A small quantity of the sediment is dissolved in a porcelain dish with a few drops of nitric acid, and the solution so obtained is evaporated. To the reddish residue one or two drops of dilute ammonia are added, when the beautiful reddish-purple colour of murexid develops itself, which, on the further addition of a few drops of caustic potash, passes into bluish-purple.

The *quantitative estimation* of uric acid may be thus carried out. A large quantity of urine (100-200 c.c.) is taken, and to it is added 10 or 20 c.c. of pure hydrochloric acid. The mixture is allowed to stand for forty-eight hours, and the precipitate of uric acid is then collected in a filter, washed with a little cold water, and weighed. Salkowski has pointed out[2] that by no means all the uric acid which the urine contains is thus removed by precipitation, and that the proportion which remains varies much in different urines. He recommends that the

[1] "The Chemistry and Therapeutics of Uric Acid, Gravel and Gout," London, 1892.

[2] Virchow's *Archiv.*, vol. lii.

filtrate should be further treated with ammonia and magnesia, and after the precipitate of phosphates has been removed by rapid filtration, that the filtrate should be treated with an ammoniacal silver solution. The precipitate which is thus formed, after careful washing, is then decomposed with sulphuretted hydrogen, the filtrate acidulated with hydrochloric acid, and the uric acid which then separates out collected and weighed.

An easier and better method of estimating uric acid in the urine is that of Hopkins.[1] The longer process which he describes is not adapted for clinical use. The shorter method is as follows. Of the urine to be examined 20 c.c. are carefully measured off, and saturated with ammonium chloride. The mixture is allowed to stand for about two hours, and is then filtered through a funnel, the neck of which has been plugged with glass-wool. After this the funnel is washed out with a saturated solution of ammonium sulphate, which removes the chlorides. The plug with the precipitate is then transferred to a small flask, and boiled with a little carbonate of soda solution until it dissolves. The solution so obtained is mixed with 4 c.c. pure sulphuric acid, and titrated with $\frac{1}{50}$-normal permanganate of potash solution, of which 1 c.c. corresponds to 0·0015 gramme of uric acid. At first the colour of the permanganate at once disappears as it falls into the solution, and the end of the reaction is marked by the colour of the drop remaining permanent for an appreciable time.

The average quantity of uric acid excreted in twenty-four hours is about 0·5 to 1 gramme. It is much increased by an animal diet. In most feverish conditions the excretion of uric acid is augmented especially in pneumonia after the critical fall of temperature. During an attack of gout the excretion is diminished, but after the paroxysm is over, it undergoes some increase. It is increased in many hepatic affections, in acute rheumatism, and sometimes in cases of simple indigestion. From what has been said as to the formation of uric acid from the nuclein bases (see p. 266) it will be readily understood that in

[1] F. Gowland Hopkins, "On the Estimation of Uric Acid in Urine," *Journal of Path. and Bact.*, vol. i. (1893) p. 451.

Leucocythæmia, where there is an enormous breaking up of leucocytes, uric acid is found in increased quantity in the urine.

Two products of the oxidation of uric acid in the body may receive very brief mention here. The first of these is :—

*Allantoin.*—It seems probable that this substance is frequently present in normal urine, though in very small quantity. In the urine of the newly-born child, however, its quantity is greater. In dogs poisoned by diamid, according to Borissow,[1] allantoin appears in some quantity in the urine, probably owing to interference with the liver functions. The second oxidation product of uric acid is

*Oxaluric Acid.*—The minute traces of this substance in the form of an ammonium salt, which are present in normal urine, possess at present no practical importance.

## Nuclein Bases.

These have been repeatedly referred to, and it has been pointed out that they are derived from the cell nuclei, and that they are in part oxidized to form uric acid. In part, however, they do not undergo this oxidation, but appear in the urine unchanged. They are constantly present in normal urine, to the amount, according to Krüger and Wulff,[2] of 0·1325 grm. *per diem.* Among the nuclein bases are included the following :—xanthin, hypoxanthin, guanin, adenin, as well as paraxanthin and heteroxanthin. As may be understood from what has been said before, these bases are much increased in quantity in the urine in cases of leucocythemia.

According to Krüger and Wulff, the estimation of these bases may be performed as follows: 100 c.c. of urine is heated to boiling and 10 c.c. of a solution of bisulphite of soda (50 grm. in 100 c.c.) are added, and immediately afterwards 10 c.c. of a 13 per cent.

[1] Borissow, "Ueber die giftige Wirkung des Diamids, des Dibenzoyldiamids, und uber das Vorkommen des Allantoins im Harn," *Zeit. f. phys. Chemie*, vol. xix. (1894) p. 497.

[2] Krüger and Wulff, "Ueber eine Methode zur quantitativen Bestimmung der sog. Xanthinkörper im Harn." *Zeit. f. phys. Chemie*, vol. xx. (1895) p. 176.

solution of sulphate of copper. A precipitate falls, which is at first white, but as the urine is again boiled turns brown. To assist the precipitation, 5 c.c. of a 10 per cent. solution of barium chloride is lastly added, and the whole filtered. The precipitate so obtained represents the uric acid and the nuclein bases, which the urine contained. Its total nitrogen is to be determined by the Kjeldahl process. A separate determination must be made of the uric acid the urine contains. The difference between these two results gives the quantity of the nuclein bases present.

**Creatinin**, a derivative of the creatin of muscle, and also derived from meat taken as food, is a normal constituent of urine, and is present in somewhat larger quantity than uric acid. Its presence is readily detected by adding to a small quantity of urine a few drops of a very dilute solution of nitro-prusside of sodium, when, on the further addition of dilute caustic soda, a beautiful ruby red colour develops itself, which soon passes into deep straw yellow.

*Quantitative Analysis of Creatinin.*—300 c.c. of urine are taken, rendered alkaline by the addition of milk of lime, and then decomposed with chloride of lime until no more precipitation takes place. The filtrate is rapidly evaporated to the consistence of syrup, and mixed with 50 c.c. of alcohol (95 per cent.) The mixture is allowed to stand until all the chloride of sodium has separated out, is then filtered, and the filtrate evaporated down and treated with an alcoholic solution of chloride of zinc. After standing for three days, the zinc-creatinin chloride will have become fully separated, and may then be collected on a filter and weighed. Of this 100 grammes correspond to 62·42 grammes of creatinin.

Probably a better method is that of G. S. Johnson,[1] which is carried out by adding to the urine a twentieth of its volume of a saturated solution of sodium acetate, and subsequently, one-fourth of its volume of a saturated solution of mercuric chloride. The urates, sulphates and phosphates, are thereby precipitated, and the filtrate, on being allowed to stand for twenty-four hours, shews a precipitate of the mercury salt of creatinin.

[1] *Proc. of the Royal Society*, vol. xliii. (1887) p. 493.

This may be collected, dried and weighed. Of this weight, 20·19 per cent. represents creatinin.

The quantity of creatinin excreted, which is normally about one gramme *per diem*, is increased on meat diet, in diabetes, and in fevers such as typhus and pneumonia, and diminished on vegetable diet, and in anæmia, chlorosis, and tuberculosis.

## CHAPTER XXVII.

## URINARY SYSTEM—(*continued*).

### AROMATIC SUBSTANCES OF THE URINE.

THE chief aromatic substances found in the urine are, hippuric acid, the phenols, the oxy-acids, and the substances met with in alcaptonuria. The indol and skatol group has already been considered.

**Hippuric Acid** is always present in the urine, though in small quantity, even in starving animals. It is formed in the body by synthesis, from benzoic acid and glycocoll. This benzoic acid is in part derived from the aromatic substances taken in vegetable food. Hence it is that hippuric acid is present in such large quantity in the urine of herbivora. But it is also formed from the putrefactive decomposition of albumin in the intestines. During such decomposition phenyl-propionic acid is formed, which, being absorbed, becomes oxidized to benzoic acid in the tissues. In the case of the dog, the synthetic formation of hippuric acid takes place only in the kidney:[1] in the rabbit, however, this does not appear to be the case.[2]

In a healthy man, on a mixed diet, the quantity of hippuric acid excreted daily is, on an average, about 0·7 gramme, but this amount is much exceeded if fruit be taken or if benzoic acid be administered. It is also increased in acute febrile conditions, in diabetes, and in certain liver disorders.

[1] Bunge and Schmiedeberg, "Ueber die Bildung der Hippursäure," *Arch. f. exp. Path.*, etc., vol. vi. (1877) p. 233.

[2] Salomon, "Ueber den Ort der Hippursäurebildung," *Zeit. f. phys. Chemie*, vol. iii. (1879) p. 365.

The quantitative estimation of hippuric acid is performed according to Bunge and Schmiedeberg,[1] as follows. The urine is rendered alkaline by means of soda, evaporated nearly to dryness, and the residue extracted with alcohol. The alcohol is evaporated, the residue dissolved in water, acidified with sulphuric acid, and repeatedly shaken up with acetic ether. The latter is separated, evaporated to dryness, and the residue repeatedly extracted with petroleum ether, which removes other organic substances, but does not dissolve hippuric acid. What remains is dissolved in warm water and evaporated. The crystals which separate are weighed.

**Phenols.**—These substances, formed in the intestine by the putrefactive decomposition of albumin, appear very commonly in the urine. The most prominent member of the group is Paracresol, which is always present in the urine, and from which by oxidation is formed Phenol. From this again, by further oxidation, arise two other members of the group, Pyrocatechin and Hydrochinon. All these phenols when they appear in the urine, do so as salts of ether-sulphuric acid. In this way the poisonous phenols are rendered harmless and their excretion provided for.

These phenols are found in somewhat increased quantity in the urine when much vegetable food is taken, on account of the aromatic substances which such diet contains. But more important are the pathological conditions under which an increase takes place. These consist, in the first place, of all diseases which tend to increase the amount of putrefaction in the intestines, such as ileus, strangulated hernia, and peritonitis; and, in the second place, of certain infective processes such as diphtheria, erysipelas and pyæmia, where putrefaction has taken place in abscess cavities or on absorbing surfaces.[2]

The analysis and separation of these substances depends on the fact that boiling the urine with dilute mineral acid causes

[1] Bunge and Schmiedeberg, "Ueber die Bildung der Hippursäure," *Arch. f. exp. Path.*, etc., vol. vi. (1877) p. 233.

[2] See J. S. Haldane, "The Elimination of Aromatic Bodies in Fever," *Journal of Physiology*, vol. ix. (1888) p. 213.

breaking up of the salts of ether-sulphuric acid. Paracresol and phenol, being volatile, can then be distilled off, precipitated with bromine water and estimated as tribromphenol, while pyro-catechin and hydrochinon are non-volatile, and consequently remain in solution. For ordinary purposes, however, the estimation of ether-sulphuric acid itself is sufficient. This will be described in a future chapter.

Hydrochinon is not present in normal urine, but appears there in cases of carbolic acid poisoning, and it is said that it is to this substance that the dark colour of "carbolic urine" is due.

**Aromatic oxy-acids.**—In the process of putrefaction of albumin in the intestines, tyrosin is formed, and this being carried to the liver, becomes oxidized to form these acids. The most important of these oxy-acids are oxy-phenyl-propionic acid (hydro-para-cumaric acid), and para-oxy-phenyl-acetic acid. They appear in very minute traces in normal urine, but are much increased in acute yellow atrophy and in phosphorus poisoning, where much tyrosin is formed. In such cases a third substance belonging to this group, para-oxy-phenyl-glycolic acid, may make its appearance in the urine.

The oxy-acids may be detected, and even approximately estimated, by the following process:—Acidify strongly 100 c.c. of the urine with hydrochloric acid, and distil it until the distillate shows, when tested with Millon's reagent, that the phenols have been driven off. What remains is allowed to cool, and is then repeatedly extracted with ether. The ether extract is then shaken up with a weak soda solution which takes up the oxy-acids. The soda solution is separated, acidified with sulphuric acid, and shaken up with ether which dissolves out the oxy-acids. Finally, the ether being evaporated on a water-bath, leaves these acids as a residue, which gives the Millon's reaction readily.

**Alkaptonuria.**—This peculiar condition of urine was first described by Boedeker in 1859. It possesses two characteristics, first, that when it is rendered alkaline and allowed to stand so that it can absorb oxygen from the air, it gradually

becomes brown and ultimately almost black; and second, that it reduces Fehling's solution, but does not rotate a beam of polarized light. Alkaptonuria may occur in persons apparently healthy. The best work on the subject has been done by Baumann.[1] There appear to be at least two substances which impart these peculiar characters to the urine. The first of these is homogentisic acid. This, according to Wolkow and Baumann, may be found in such urine in very considerable amount,—4 grammes in twenty-four hours,—and is much increased by a meat diet, and particularly by the administration of tyrosin. It is indeed to some peculiar decomposition of tyrosin, probably by a micro-organism, that alkaptonuria is believed to be due. The method of quantitative analysis recommended by Wolkow and Baumann rests upon the reduction of an ammoniacal solution of silver by homogentisic acid.

The other substance found in alkaptonuria is uroleucic acid which was first discovered by Kirk.[2] It differs from homogentisic acid in that it reduces not only Fehling's solution, but also alkaline bismuth solutions.

[1] See, in particular, a paper by Wolkow and Baumann, "Ueber das Wesen der Alkaptonurie," *Zeitschr. f. physiol. Chemie*, vol. xv. (1891) p. 228.

[2] *Journal of Anatomy and Physiology*, 1889, p. 69.

# CHAPTER XXVIII.

## URINARY SYSTEM—(*continued*).

### THE INORGANIC CONSTITUENTS OF THE URINE.

MANY inorganic salts appear in the urine. Those which are of clinical interest are the salts of hydrochloric, sulphuric, phosphoric, and oxalic acids.

**Chlorides.**—The chlorides which appear in the urine,—almost entirely as chloride of sodium,—are, under ordinary circumstances, derived directly from the food. The kidneys, however, do not act simply as filters in this excretion. There is a very interesting automatic mechanism whereby the percentage of salt in the blood is maintained at a certain level. It is known that the tissue-cells cannot functionate unless they are bathed in juices which contain about 0·5 per cent. of chloride of sodium, and it is the duty of the renal epithelium to maintain the salt in the blood at this level. Within reasonable limits this mechanism is perfect. If more salt than is needed reaches the blood, the excess is excreted in the urine. On the contrary, if the food taken is deficient in salt, the quantity of chlorides in the urine diminishes, in order that the blood may retain its due proportion.

It is clear, however, that the blood may become poor in chlorides in other ways than by the mere deficiency of salt in the food. In the case, for example, of the sudden formation of a large exudation,—as in acute pneumonia, or in pleurisy with effusion,—a considerable quantity of salt leaves the blood to pass into the exudation. Hence the blood, being deficient in salt, will retain as much as possible from the food until the right concentration is attained. Therefore, in such cases, the quantity of chlorides in the urine diminishes, and may even

almost disappear, while the exudation is taking place, to return to normal when the equilibrium in the blood is reached. And hence it follows that during the absorption of such an exudation, much salt is taken up by the blood, and, being excreted, raises the amount of chlorides in the urine. A similar train of reasoning shews how the chlorides in the urine come to be diminished after hæmorrhage, and after diarrhœa. In acute fevers there is a diminution of chlorides, even when no exudation is taking place. The cause of this is not wholly clear, but it probably depends chiefly on the small quantity of food taken under such circumstances.

The presence of chlorides in the urine may readily be detected by adding to a small quantity of urine in a test-tube a little nitric acid, and then a few drops of a solution of nitrate of silver. A white flocculent precipitate at once falls, consisting mainly of chloride of silver, but also containing combinations of silver with uric acid, creatinin, xanthin, and urinary pigments. The quantity of chlorides present may, according to Hammersten, be roughly estimated by this simple method. The urine, after being strongly acidified with nitric acid, has added to it drop by drop a concentrated silver-nitrate solution (1 : 8). If the quantity of chlorides be normal the drop sinks to the bottom as a compact cheesy mass. If the chlorides are diminished the precipitate formed is less coherent, and if the quantity present is very small, only an opalescence forms.

*Estimation of the Chlorides.*—Mohr's method depends upon the fact that when to a neutral urine containing chloride and phosphate of sodium and a neutral salt of chromic acid, a solution of nitrate of silver is added, there first occurs a precipitation of chloride of silver; and when the point is reached when all the chlorine contained in the chloride of sodium is so precipitated, there then begins the precipitation of the red chromate of silver. For this analysis we therefore require—

1. *A solution of nitrate of silver*, of which 1 c.c. corresponds to 10 milligrammes of chloride of sodium or to 6·065 milligrammes of chlorine.

2. *A cold saturated solution of neutral chromate of potassium.*
3. *Pure nitrate of potassium.*
4. *Pure carbonate of lime.*

Since the presence of urinary pigments interferes with the accuracy of this method, the silver combining with them, they must first be got rid of in the following manner :—

10 c.c. of urine are mixed with 2 grammes of nitrate of potassium in a platinum capsule, evaporated to dryness, and finally heated in a naked flame until the carbon is completely oxidised. The residue is then dissolved in water in a beaker, acidulated with a dilute solution of pure nitric acid, and then neutralised with a little carbonate of lime. To the fluid so obtained, which need not be filtered, four or five drops of the chromate solution are added, and then the silver solution is gradually dropped into it from a burette, the mixture being constantly stirred. Reddish spots appear where the solution falls, but they disappear on stirring, so long as any chloride of sodium is present. So soon, however, as the whole of that salt is decomposed, the next drop of the silver solution gives rise to a permanent red, which marks the conclusion of the operation. The amount of the silver solution which has been used is now read off, and as we know that each cubic centimetre corresponds to 6·065 milligrammes of chlorine, the calculation is easy.

The average quantity of chlorine excreted in the urine, in the healthy state, in twenty-four hours may be taken to be about 10 or 12 grammes.

**Sulphates.**—The sulphates which are found in the urine are derived from the breaking up of proteid substances, and to a lesser degree from the food. Sulphuric acid exists in the urine in two forms—first, in combination with the alkalies ; and, second, as Baumann has shown, in the form of aromatic ethereal sulphates — chiefly phenyl-sulphate and indoxyl-sulphate, and other aromatic bodies already considered. These aromatic bodies when heated with hydrochloric acid break up into phenol or indigo and sulphuric acid. Acetic acid does not cause this decomposition.

*Estimation of the Sulphates.*—Acidulate strongly with acetic acid, and on the addition of chloride of barium a white precipitate of sulphate of barium will fall, representing the sulphuric acid which was combined with the alkalies. If now the mixture be filtered and heated with hydrochloric acid, a further precipitate of sulphate of barium will fall, representing in this case the ether-sulphates. If these two precipitates be weighed, the total amount of the sulphates, as well as that of each form, may be calculated.

The total quantity of sulphuric acid excreted in the urine in twenty-four hours is about 2 grammes. It is increased and diminished according as more or less albumin is broken up, and therefore it corresponds with the quantity of urea and uric acid.

Of this amount, about ·1 to ·2 gramme is in the form of salts of ether-sulphuric acid. These, as has been already said when speaking of the aromatic compounds, result from these aromatic substances, which are formed chiefly during the putrefaction of the intestinal contents. The quantity of ether-sulphuric acid in the urine is therefore a measure of the amount of putrefaction going on at the time.

Sulphur is also excreted in the form of what have been called by Salkowski the "neutral-sulphur" compounds, taurin, cystin, sulphocyanic acid, and, occasionally, sulphuretted hydrogen. If it is desired to determine the quantity of the neutral sulphur, it is necessary in the first place to estimate the total sulphur present. This is done by treating a measured quantity of urine with fuming nitric acid,[1] and subsequently estimating the sulphur as sulphate of barium. A separate estimation of the sulphates present must be made in the manner already described. The difference between the figures so obtained represents the quantity of neutral sulphur present.

**Phosphates.**—In normal urine phosphoric acid is met with in the form of the phosphates of the alkalies, sodium and potassium

[1] See paper by P. Mohr, "Ueber Schwefelbestimmung im Harn," *Zeitschr. f. physiol. Chemie*, vol. xx. (1895) p. 556.

(alkaline phosphates), and of calcium and magnesium (earthy phosphates). It may also appear in the form of glycerin-phosphoric acid and lecithin. It is derived chiefly from the food, but also in part from the breaking down of tissues of the body which contain phosphorus, principally the osseous and the nervous structures.

When the urine loses its carbonic acid, as it does when heated, the earthy phosphates separate out as a white flocculent precipitate, which becomes redissolved on the addition of acid. The addition of ammonia to urine causes an amorphous precipitate of phosphate of lime, while the phosphate of magnesium unites with the ammonia to form ammonio-magnesian phosphate (triple-phosphate), which appears in a crystalline form. The microscopic appearance of all the various forms of phosphate will be described when we come to speak of urinary sediments.

*Estimation of Phosphoric Acid.* — The principle of Neubauer's method is the following:—When a hot solution of the phosphates in question is acidulated with acetic acid, it gives, with a solution of acetate of uranium, a precipitate of uranium phosphate. The point at which this reaction ends is, from the nature of the precipitate, difficult to determine, and it is consequently necessary to test the mixture from time to time with a solution of ferro-cyanide of potassium, which gives, when there is present the slightest excess of the uranium solution, a dark reddish-brown coloration. The solutions required are:—

1. A solution of uranic nitrate, of which 1 c.c. is equivalent to 0·005 gramme of phosphoric acid, $P_2O_5$.
2. A solution of acetate of soda prepared by dissolving 100 grammes of that salt in 900 c.c. of distilled water, and adding 100 c.c. of pure acetic acid.
3. A solution of ferro-cyanide of potassium not too concentrated.

To 50 c.c. of the urine are added 5 c.c. of soda solution, and the mixture is placed in a beaker, and warmed in a sand-bath. From a burette the uranium solution is gradually added to the

urine, until no further precipitation appears to take place. A drop is now removed, placed on a porcelain slab, and mixed with a drop of the solution of ferro-cyanide of potassium. If there be any excess of uranium—*i.e.*, if the analysis be at an end—a reddish-brown precipitate will appear where the drops come in contact. If this reaction does not take place, more uranium solution must be added to the urine. Each cubic centimetre of the uranium solution used corresponds to 0·005 gramme of phosphoric acid, so that the calculation is easy.

If it is wished to estimate separately the earthy phosphates, these must be precipitated by the addition of ammonia, the precipitate carefully separated by filtration, dissolved in water with the addition of a little acetic acid, and the solution treated in the manner just described.

The average quantity of phosphoric acid which is excreted in the urine in twenty-four hours is in the adult about three grammes, two-thirds of which may be taken to consist of the phosphates of the alkalies, and one-third of earthy phosphates. The quantity depends to a large extent upon the food—animal diet giving rise to more excretion than vegetable—and upon the condition of the alvine secretion, the earthy phosphates in particular being increased in quantity when there is much constipation. Tissue change also influences the phosphatic elimination to some extent, chiefly that which takes place in the nervous structures.

In the feverish state the phosphates are at first diminished, but when convalescence sets in their amount in the urine is increased to a point above normal. In chronic nervous diseases the phosphates are usually present to an excessive amount in the urine, and in osteomalacia the earthy phosphates are increased to such a degree that they may be found to be in excess of the phosphates of the alkalies. A form of phosphaturia, analogous to glycosuria, has been described.

**Oxalic Acid** in the form of oxalate of lime, occurs normally in the urine in small quantities which may be much increased by such vegetables as contain this substance; indeed Craufurd

Dunlop has recently asserted, in a very able paper,[1] that the whole of the oxalic acid of the urine is to be thus accounted for. Oxalic acid is, however, usually regarded as a product of the incomplete oxidation of uric acid, and is said to be found in increased quantity in conditions leading to interference with respiration and circulation.

As regards the symptoms of oxaluria, there is much difference of opinion, but it appears certain that in many cases at all events the appearance of oxalate of lime in quantity in the urine is accompanied with a well-marked train of symptoms. The patient is usually emaciated, very nervous, extremely hypochondriacal, and irritable. There is pain across the small of the back, a degree of irritability of the bladder, and general muscular weakness. It must be remembered, however, that, on the one hand, such a train of symptoms may be present in full development without any oxaluria, and, on the other, that crystals of oxalate of lime may be very abundant without producing any such symptoms.

The best method of estimating oxalic acid appears to be that of Dunlop, which is founded upon those of Reoch and Neubauer. The analysis is tedious and difficult, and hence is not suitable for ordinary clinical work. For its details the original paper[1] should be consulted.

[1] J. Craufurd Dunlop, "The Excretion of Oxalic Acid in Urine," &c., *Journal of Path. and Bact.*, 1896, p. 389.

# CHAPTER XXIX.

## URINARY SYSTEM—(*continued*).

### THE PROTEIDS OF THE URINE.

VARIOUS proteid substances may make their appearance in the urine, under pathological conditions. Those most usually encountered are serum-albumin, serum-globulin (paraglobulin), peptone, albumose, fibrin, and the proteids of the blood.

**Albuminuria.**—That albumin is present in healthy urine, has been maintained by many observers, such as Posner[1] and Senator,[2] and has perhaps been too readily accepted. The investigations of von Noorden,[3] of Leube,[4] of Winternitz,[5] and of Spiegler,[6] go strongly to disprove this view. But while albumin cannot therefore be looked upon, even in very minute traces, as a normal constituent of the urine, yet it is probable that in the form of nucleo-albumin, it not infrequently does occur, resulting then from the solution of the proteids of the cell-elements which the urine contains. This is, however, a chemical and not a clinical question, for such exceedingly minute traces cannot be

[1] Posner, "Ueber physiologische Albuminurie," *Berl. kl. Wochenschrift*, 1885, p. 654.

[2] Senator, "Die Albuminurie, &c.," Berlin, 1890.

[3] C. von Noorden, "Ueber Albuminurie bei gesunden Menschen," *Deutsches Archiv., f. kl. Med.*, vol. xxxviii. (1886) p. 205.

[4] Leube, "Ueber physiologische Albuminurie," *Zeit. f. kl. Med.*, vol. xiii. (1887) p. 1.

[5] Winternitz, "Ueber Elweiss in normalen Harn," *Zeitschr. f. phys. Chemie*, vol. xv. (1891) p. 189.

[6] Spiegler, "Ueber die sogenannte physiologische Albuminurie," Wiener Blätter, 1894, No. 38. I have only been able to see an abstract of this paper in Virchow and Hirsch's Jahresbericht for that year.

detected by the most delicate clinical tests, but require previous precipitation by means of alcohol or ammonius sulphate, from large quantities of urine.

Albuminuria, in the clinical sense, is taken to mean the presence of albumin in the urine in sufficient quantities to be detected by the clinical tests which will be presently described. There are two great factors in its production, and most of the pathological conditions under which albumin occurs in the urine may be referred to one or other of these.

The first and more important cause consists in definite pathological changes in the renal tissues. This includes all the different forms of Bright's disease, where the renal epithelium has become inflamed or degenerated in such a way as to allow the porteids of the blood-plasma to pass into the urine. To this class also belongs the albuminuria which occurs in many fevers, in which without there being any permanent damage, the renal epithelium is so acted upon by toxines in the blood, (or perhaps by the micro-organism itself), as to lose its power of preventing the passage of albumin.

The second cause of albuminuria consists in changes in the circulation through the kidneys, and the essence of such processes, so far as albuminuria is concerned, lies in the fact that, whether from deficiency in quantity or in quality of blood, the vitality of the renal epithelium is interfered with, with the result that albumin is allowed to pass. Such circulatory disturbances may be of very short duration, as the temporary albuminuria which sometimes follows a cold bath,[1] or an epileptic fit, or that which Schreiber[2] was able to induce by compression of the thorax for a minute or more. But, much greater importance attaches to the albuminuria which is the result of heart and lung disease. The backward pressure which is thereby produced, acting along with the defective oxygenation of the blood, lowers the vitality of the renal epithelium. In anæmia and

[1] Geo. Johnson, "On the Etiology of Albuminuria," *Brit. Med. Journal*, 1873, vol. ii., p. 112.

[2] Schreiber, "Ueber experimentell am Menschen zu erzeugende Albuminurie," *Arch. f. exp. Path.*, etc., vol. xix. (1885) p. 237.

other wasting diseases, and in cachectic processes, the morbid quality of the blood, produces a like result. Probably to this category may also be referred the albuminuria which occurs in exophthamic goitre,—a symptom first pointed out by Warburton Begbie.[1]

Mention should also be made of the transitory or cyclic albuminuria, the cause of which is obscure. It usually occurs in young men, and it is remarkable in that at some periods of the day the urine may be quite free from albumin, while at others, usually after meals, the quantity present may be very considerable. The changes which go to produce this condition are unknown, but that they are not of great gravity is shewn by the fact that such patients may exhibit this symptom for a long series of years without developing other renal or circulatory symptoms, and may even ultimately make a complete recovery. This form of albuminuria is often associated with a neurotic or dyspeptic habit.

Contrasting with this, it is to be noted that, while in the inflammatory forms of Bright's disease the albumin present is always considerable in quantity, in the cirrhotic or contracting form the quantity is, in the early stages at least, very small, and that, out of several specimens examined, albumin may only be found in some. The changes which occur in such cases in the circulatory organs (hypertrophy of the left ventricle, with tonicity of the arterioles and a pulse of high tension), do not shew themselves in the cyclic albuminuria of which we have been speaking. In amyloid or waxy degeneration of the kidney, also, the quantity of albumin present is usually small.

It must, finally, be remembered that albumin may find its way into the urine from some affection of the urinary passages. This can usually be distinguished with readiness from renal albuminuria by means of the formed elements the urine contains, such as epithelium or pus, and by the general symptoms of the case.

*Varieties of Albuminous bodies met with.*—In albuminuria

[1] *Edinburgh Medical Journal*, 1874.

the two chief proteids in the urine are, serum-albumin, and serum-globulin, and what has been said as to the clinical aspects of albuminuria applies to these. Serum-albumin is usually present in much larger quantities than serum-globulin, although the reverse may occasionally be the case. The clinical significance of serum-globulin is not known. It does not appear to occur alone, or at any rate very rarely so.

But, besides these two, other bodies may be encountered in the urine. The *albumoses* may make their appearance under certain not well understood conditions, such as in disease of bone, especially in osteo-malacia, as was originally shewn by Bence Jones, and in ulceration of the intestine.

Krehl and Matthes[1] have lately found albumoses in the urine of fever, indeed they believe that this is a very common occurrence, the albumoses disappearing as the temperature falls to normal. Deuteroalbumose and Histon were the varieties found, the first the result of the breaking up of albumin by bacterial action, the second a product of the decomposition of Nucleo-histon, a proteid contained in the leucocytes of the blood and lymphatic glands.

The presence of *nucleo-albumin* may not infrequently be detected, especially where, from catarrhal affections of the urinary passages, there is much mucus present. It has also been found in quantity in cases of leucocythemia, and, according to Pichler and Vogt,[2] nucleo-albumin appears in the urine of dogs for some days after the intra-venous injection of a solution of casein, and also after temporary compression of the renal artery.

Where serum-globulin is present in the urine and at the same time an exudation of fibrinogenic substance takes place in the kidney or in some other portion of the genito-urinary tract, *fibrin* is formed and appears in the urine. The fibrin may be first formed after the urine has been passed, when it appears as a

[1] Krehl and Matthes, "Ueber febrile Albumosurie," *Deutsches Arch.*, vol. liv. (1895) p. 501.

[2] Pichler and Vogt, "Zur Lehre von der Nucleo-albuminurie," *Centralbl. f. inn. Med.*, 1894.

firm clot, or it may be formed in the bladder and make its appearance in the urine as shreds. This condition is seen in chyluria and hæmaturia, and occasionally in diphtheria, and in tubercular disease of the urinary organs.

**Peptonuria.**— A great deal of work has been done on this subject in recent years, and some considerable clinical importance attaches to it. Unfortunately the methods hitherto employed for the detection and isolation of peptones in the urine, have, in most cases, been insufficient. That true peptone (Kühne's) ever appears in the urine is exceedingly doubtful. Pathological urines of varying kind have been analysed by recent observers, using the best methods, without any trace of this substance being found. Nor can true peptone be detected in sputum or in pus. It is probable that the proteid substance which has been found in so-called "peptonuria," is a mixture of albumoses. The best work on the subject known to me is that of Stadelmann,[1] where will be found references to the chief literature on Peptonuria.

But, while chemically this condition is not a true peptonuria, the term may still be used clinically, at any rate until it has been fully ascertained what the substances are which give the reaction with the tests to be presently described.

Albumoses and peptone are the results of the breaking up of albumin under the influence of the digestive ferments. They are not, however, absorbed as such, but undergo further changes into which it is not necessary now to enter. In the case of gastric ulcer, of carcinoma of the stomach, and of intestinal ulceration, certain of these proteids may enter the blood stream and be excreted in the urine. This is what has been called *enterogenic peptonuria.* Another form is known as *pyogenic,* being associated with pus formation in any part of the body. It is in that, in the absence of enterogenic causes, the occurrence of peptonuria points to pus formation, that it is useful clinically in obscure cases. It has been supposed that by bacterial action on the albumins of pus, albumoses and peptones

[1] Stadelmann, "Untersuchungen ueber die Peptonurie," Wiesbaden, 1894.

are formed, and passing into the circulation, are excreted in the urine. The diseases in which this pyogenic peptonuria occurs are chiefly the following: abscesses anywhere in the body, pyelo-nephritis, acute articular rheumatism, epidemic cerebro-spinal meningitis, croupous pneumonia (especially during the absorption of the exudation), phthisis, empyæma, and acute yellow atrophy of the liver.

Peptonuria may also occur in certain blood-diseases, such as scorbutus, and in phosphorus poisoning.

The subject of hæmaturia will be treated of subsequently.

Proceeding to describe the tests for albuminuria, it will probably be most convenient to mention first the general tests, and subsequently the means at command for separating these various proteid bodies. Finally the processes for quantitative analysis will be described.

**Detection of Albumin** (serum-albumin and serum-globulin). —Before testing for albumin, the urine in question must, if not already clear, be rendered so by careful filtration. Of the many methods employed, the following are the most important:—

(1.) *Boiling Test.*—If a small quantity of urine be placed in a test-tube, and heated in the flame of a spirit-lamp or Bunsen burner, it will be found that when the temperature has risen to near the boiling-point the albumin, if present, separates out as a white cloud, which on standing, collects at the bottom of the tube in fine flakes. If the urine contains much earthy phosphates, these are apt to separate when the tube is heated, and the cloud so formed may be mistaken for albumin. It will, however, dissolve on the addition of a few drops of acid, while the cloud of albumin will thereby be rendered denser. If the urine be alkaline to begin with, the albumin may not be separated out on boiling. It is therefore necessary to acidulate with a few drops of acetic acid; but inasmuch as there is some risk of adding too much of this acid, and so preventing the albumin reaction from taking place, it is best to proceed in all cases as follows:—

5-10 c.c. of urine are placed in a test-tube and acidulated with acetic acid, and 1-6 of its volume of concentrated solution of sulphate of magnesia is added. If albumin be present, there will now appear on heating a more or less distinct cloudiness. By this test serum-albumin and serum-globulin are precipitated but not the albumoses or the peptones.

(2.) *Heller's Nitric Acid Test.*—A test-tube, or, better, a small conical glass is taken and filled about one-third full of urine. Down its side, while it is held inclined, are poured slowly a few drops of strong nitric acid, in such a way that, when the glass is again held in an upright position, the acid forms a distinct layer at the bottom. If albumin be present in the urine, a cloud will form at the line of junction of the two fluids.

If the urine contain a large quantity of urates, the addition of nitric acid may cause the separation of the acid urates in the form of a cloud. This cloud lies near the upper surface of the urine, and is therefore not readily mistaken for albumin; but in cases of doubt it is only necessary to warm the glass, and so cause solution of the cloud, or to dilute the urine previously with twice or thrice its volume of water, after which no such cloud will form. A crystalline cloud of nitrate of urea may also form, but its appearance and its solution on beating will suffice to distinguish it.

When copaiba or other resin has been administered, a whitish cloud may appear at the line of junction of the two fluids, which however is soluble on beating.

Heller's test coagulates serum-albumin, serum-globulin, albumoses, and nucleo-albumin, but not peptones.

(3.) *The Ferro-cyanide Test.*—To the urine contained in a test-tube a few drops of acetic acid are to be added, and then a small quantity of a solution of ferro-cyanide of potassium. If albumin be present, a white flocculent precipitate will separate out in the cold. This cloud or precipitate forms at once, and is to be distinguished from that which shews only after the test-tube has been allowed to stand. The latter is due to nucleo-albumin, and indeed if this substance be present in any quantity, a cloud may form on the mere addition of acetic acid.

This test coagulates all the proteids except peptone.

Other tests, such as those in which picric, carbolic, tannic, and metaphosphoric acids are employed, appear to be unnecessary for ordinary clinical purposes.

**Separation of these Proteid Bodies.**—In ordinary clinical work the term "albumin" is understood as meaning the mixture of serum-albumin and serum-globulin which is precipitated by the boiling test. It is sometimes of interest to ascertain whether one or both of these bodies is present in a specimen of albuminous urine. For this purpose it is best to neutralize the urine and then to saturate it with magnesium sulphate, or to add to it an equal volume of a saturated solution of ammonium sulphate. The globulin separates out as a more or less dense cloud, which can be collected on a filter, the filtrate containing whatever serum-albumin was present in the urine.

If, in the case of a particular urine, it is found that while the boiling test gives no reaction, there is a distinct cloud when Heller's test is used, the substance present is either nucleo-albumin or albumose. The ferro-cyanide test should then be tried. If nucleo-albumin be present, it will probably be found that the mere addition of acetic acid is sufficient to cause a cloud to form, while, in the case of albumose, precipitation will not take place until the ferro-cyanide solution has been added.

But cases of this description (in which the boiling test is negative while Heller's test gives a cloud), may occur in which it is advisable to make a much more searching examination of the urine in order to determine exactly what albuminous bodies are present. The following is the method recommended by Neumeister.[1] The urine for twenty-four hours is collected, neutralized, and concentrated on a water-bath at a temperature of 60° C. to 70° C., to about a litre. It is then to be filtered, saturated with ammonium sulphate, and again filtered. The

[1] Neumeister, *Lehrbuch der physiologischen Chemie*, Part I., 1895, page 363. See also this author, "Ueber die Reaktionen der Albumosen," *Zeitschr. f. Biologie*, N.F., vol. viii. (1890) p. 335.

filtrate is to be set aside to be subsequently tested for peptone. The precipitate which has been caught on the filter may be first tested with the Biuret-reaction to ascertain if it is of an albuminous nature and then dissolved in a little water. The solution so obtained is divided into two portions.

The first portion is to be tested for nucleo-albumin. It is to be dialysed against water until the sulphate has completely disappeared. When this has occurred, hetero-albumose, if it was present, will have been precipitated, and can now be removed by filtration. The filtrate, if nucleo-albumin is present, will give the two following reactions. On the careful addition of acetic acid a precipitate will form, which is readily redissolved by mineral acids. Further, if the precipitate formed by acetic acid be collected on a filter, washed with dilute acetic acid and dried, it will yield phosphoric acid when heated with caustic potash and saltpetre.

The second portion is to be tested for albumoses. To a small quantity of this liquid add an equal volume of a concentrated solution of common salt, and then acetic or nitric acid so long as the precipitate which may form increases in quantity. The mixture is then boiled, and if the precipitate passes entirely or partially into solution, so that when filtered at boiling temperature the filtrate, at first clear becomes cloudy as it cools, then it may be concluded that albumoses are present.

To determine what variety of albumose is present it is best to take the dialysed fluid, add acetic acid so as to precipitate the nucleo-albumin, and filter. To the filtrate add pieces of rock-salt to saturation. If the fluid remains clear the primary albumoses are absent. Then add acetic acid which has been saturated with chloride of sodium. This precipitates deutero-albumose.

The filtrate which, in the course of these manipulations was put aside for the purpose of testing for peptone, is to be treated thus. An equal volume of water is to be added, and then a few drops of freshly prepared solution of tannic acid. If no precipitation follows after a little time, peptone is not present. If peptone be present the precipitation should occur at once,

and the precipitate should be redissolved on the addition of excess of tannic acid.[1]

**Estimation of Albumin.**—It is often of great importance to the physician to know the quantity of albumin which is being excreted in the urine from day to day. For ordinary purposes the method of Esbach is sufficient. It is usual in this case to estimate the serum-albumin and serum-globulin together.

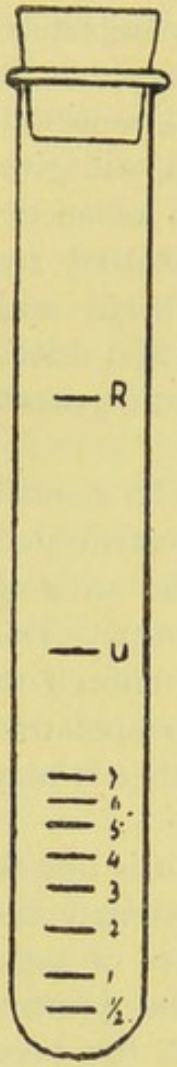



FIG. 67. Esbach's Albuminometer.

The apparatus consists of an albuminometer-tube, which is to be filled up to the mark U with the urine to be examined, which must be acid in reaction, to which is to be added enough of Esbach's reagent to bring the level of the mixture to the mark R on the tube. This reagent is made by dissolving 10 grammes of picric acid and 20 grammes of citric acid in a litre of water. The urine and the reagent should be thoroughly mixed by closing the tube with a rubber cork, and inverting it two or three times. The albumin and globulin are coagulated and precipitated, and after the tube has been allowed to stand for twenty-four hours, the height of the precipitate is read off on an empirical scale, marked on the tube, which is so arranged as to give the amount of albumin in grammes per litre of urine. It is to be noted that, to give accurate results, the urine must not have a specific gravity of more than 1008, nor albumin in an amount greater than 4 per cent. In such cases the urine should be diluted sufficiently to bring it between these limits. In comparing results, it is also important to remember that the temperature of the room in which the tube stands should be approximately the same on each occasion.

In this way also, the relative quantities of albumin and

[1] See Stadelmann "Untersuchungen ueber die Peptonurie," p. 62, Wiesbaden, 1894.

globulin present may be estimated, by first estimating them together as above, and then salting out the globulin by saturating with sulphate of magnesia, and filtering. The filtrate contains the serum-albumin, which may be estimated by Esbach's method as above. Owing however to the high specific gravity of the filtrate, the precipitate will require some days to settle fully.

A more exact method is that by weighing, which is performed as follows:—

The urine is carefully filtered—10-15 c.c. of the filtrate placed in a porcelain dish, acidulated with acetic acid and evaporated to dryness on a water-bath. The residue is extracted, first with hot water, and then with alcohol, placed upon a weighed filter, dried at 100° C., and finally weighed. From the result so obtained must be subtracted the quantity of earthy phosphates and colouring matter which the residue contains, and this is done by burning the filter and the coagulum in a platinum capsule and deducting the weight of the ash so obtained.

Still more simply and quickly may the estimation of the albumin and globulin be carried out by taking the coagulum, after washing as above, and estimating its nitrogen by Kjeldahl's method (see page 267). Knowing that these substances contain 15·8 per cent. of nitrogen, it is clear that the amount of nitrogen given by Kjeldahl's process requires to be multiplied by 6·3 to give the amount of albumin present in the urine. In the same way the amount of globulin present may be exactly ascertained by separating it out by saturating with sulphate of magnesia, and estimating the nitrogen it contains. The difference between these two results would represent the amount of serum-albumin present.

**Ferments in the Urine.**—Of these ferments, pepsin and ptyalin are frequently found in the urine. That their presence is not due to absorption after they have been secreted in the alimentary tract, is shewn by various facts. First, as Hildebrandt[1] has pointed out, these ferments, when injected even

[1] H. Hildebrandt, "Zur Kenntniss der physiologischen Wirkung der hydrolytischen Fermente," *Virchow's Archiv.*, vol. cxxi. (1890) p. 1; also vol. cxii., p. 375.

in very small doses into the blood-stream, are exceedingly poisonous, animals dying with fever symptoms, from the destruction of the red blood corpuscles. Secondly, the quantity of these ferments present in the urine is least during digestion and most in the morning urine when there is a condition of hunger. They can be still further reduced by the administration of pilocarpine which greatly increases the outpouring of these ferments into the digestive tract. Finally, it is known that after ligature of the pancreatic duct, ptyalin and trypsin appear in quantity in the urine.

From these and other similar facts it is believed that it is not the ferments themselves which are absorbed, but the Zymogenes from which the ferments are prepared. These, passing into the circulation, reach the kidneys, where, owing to the acid reaction there encountered, they pass into the condition of fully formed ferments, and so appear in the urine.

Pepsin has been found to be absent from the urine in cases of typhoid fever, and of carcinoma of the stomach, and in certain forms of nephritis.

The presence of pepsin may be detected in the urine in the following way:—Shreds of pure fibrin are added to the urine and allowed to remain in it for some hours so as to absorb the pepsin, a process which will be assisted by frequent agitation. The fibrin is then removed, washed and covered with a 0·2 per cent. solution of hydrochloric acid, which is kept for a little time at a temperature of 38° C. Saturate the solution so obtained with sulphate of magnesia to precipitate the albumin and albumoses, and test the filtrate for peptones with the Biuret-reaction.

To test for ptyalin, stir a large quantity of urine with lime-water, which causes a precipitate of calcium phosphate, along with which the ferment is also brought down. Collect this precipitate on a filter, and suspend it in water to which a little pure starch solution has been added. This mixture is to be kept at a temperature of 38° C. for half an hour, and then filtered. If ptyalin was present in the urine, the filtrate will give the reactions of sugar when submitted to the appropriate tests.

The milk-curdling ferment may also sometimes be found in the urine. For this purpose 5 c.c. of fresh milk, 1 c.c. of a 0·6 per cent. solution of hydrochloric acid, and 5 c.c. of urine are mixed together. In a few minutes when kept at a temperature of 38° C., the milk becomes curdled. This may usefully be compared with a control experiment where the urine has been previously boiled, in which case no curdling takes place.

**Mucus.**—A small quantity of mucus is present in normal

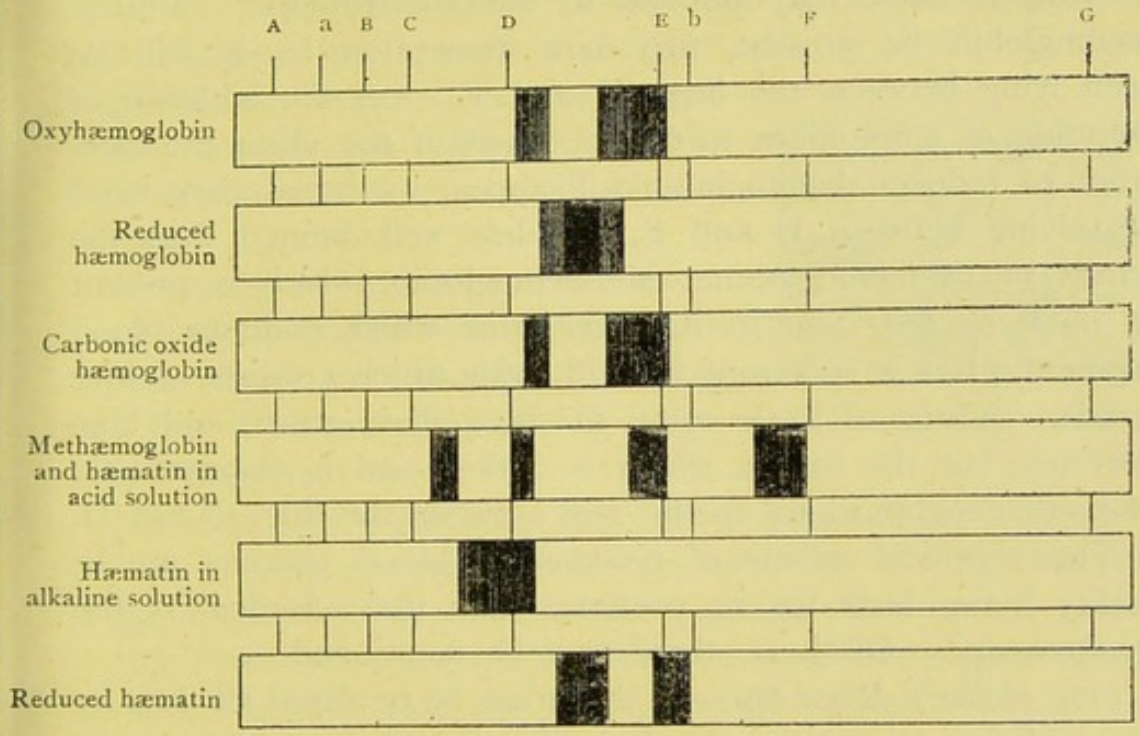



FIG. 68.—Absorption spectra of blood pigment.

urine; but in such affections as catarrh of the bladder or urethra it may be much increased. It is sometimes of importance to be able to distinguish mucus from pus in the urine. This is readily done by filtration, when if the pus be present, the filtrate will give the reactions of albumin; but if it contain mucus it will give that which is characteristic of mucin—*i.e.*, when acidulated with acetic acid a precipitate of mucin separates out in the cold.

**Blood** may be found in the urine as such (hematuria), or only blood pigment may be present (hæmoglobinuria); and these two conditions are readily distinguished by the fact that in the former case blood corpuscles are found on microscopic examination, while in the latter they are absent. The admixture of even a very small quantity of blood gives the urine a peculiar smoky appearance. When it is present in larger amount the urine becomes bright-red or dark-brown. Small quantities of blood are best detected by means of the microscope, but when no corpuscles or crystals of hæmatin are present, recourse may be had to the spectroscope. If oxyhæmoglobin be present, two dark absorption bands will be seen lying between the lines D and E. On the addition of sulphide of ammonium to the specimen of the urine, the spectrum of reduced hæmoglobin will appear—a broad dark band also lying between D and E, and less well defined than the bands of oxyhæmoglobin. Methæmoglobin, which is present in more or less quantity in every urine which contains blood pigment, gives a spectrum in acid urine which consists of four bands. Three of these occur in the yellow, green, and blue portions, but the fourth, which is darker, and is characteristic of methæmoglobin, lies in the red, between the lines C and D.

The chemical means of recognising blood pigment are in reality better tests for its presence than that afforded by the spectroscope. Of these, three may be mentioned.

(1.) *Heller's Blood-test.*—If the urine be rendered alkaline by the addition of caustic potash and then boiled, a precipitate of the earthy phosphates takes place. Under ordinary circumstances this precipitate is white in colour, but if blood pigment be present, it assumes a more or less deep red appearance. This test is a very delicate one. After the administration of Rhubarb, Senna and Chrysarobin, however, a similar colour may be produced.

(2.) *Guaiacum test.* To a small quantity of urine in a test-tube, a couple of drops of tincture of guaiacum, which must be freshly prepared, are added. After mixing, a little ozonic ether is poured into the test-tube, and the whole shaken. If blood is

present, the ozonic ether, on rising to the surface, will shew a distinct blue coloration. Saliva and pus also give this reaction.

(3.) *Teichmann's test.* This is perhaps the most delicate of all. Either the precipitate of Heller's test may be taken, or, better, the urine may be rendered alkaline with ammonia and precipitated with tannic acid. In either case the precipitate is to be collected on a filter and dried. A very small quantity is then placed on a microscope slide, along with a crystal of common salt, and a drop of glacial acetic acid added. Over the whole a cover-glass should be placed, and the slide heated over a flame. On subsequent examination with the microscope the characteristic crystals of hæmin will be seen, if blood pigment was present in the urine. These crystals are oblique rhombic prisms, and have a reddish brown colour.



FIG. 69.—Hæmin Crystals.

In cases of hæmaturia it is important to ascertain from what point in the urinary tract the blood comes, and this is not usually difficult. The hæmorrhage may come :—

(1.) *From the Urethra.*—The blood is mixed with the first portion of urine passed, often being expelled as a long clot, and it may continue to flow in the intervals of micturition.

(2.) *From the Neck of the Bladder*, or prostatic part of the urethra.—In this case the blood usually appears only at the very end of micturition, when the sphincter vesicæ begins to contract.

(3.) *From the Bladder.*—The blood is usually coagulated, and is passed in clots as large as the calibre of the urethra will allow to escape.

(4.) *From the Ureters.*—In this case the blood often appears in the form of long worm-like clots, which are casts of the ureters.

(5.) *From the Kidneys.*—When the blood comes from the kidneys it is uniformly diffused through the urine, is almost never in very large quantity, and when the urinary sediment is

examined, there are found tube-casts, usually containing blood corpuscles.

The morbid renal conditions which tend to produce hæmaturia, are chiefly the following:—acute nephritis, the result, usually, of infective processes, such as scarlet fever, septicæmia, malaria; embolism of the renal vessels, in cases of endocarditis; cancerous or tubercular affections of the kidney; calculus in the pelvis of the kidney; filaria, and other blood parasites.

*Hæmoglobinuria* appears in such diseases as purpura, scurvy, pyæmia, severe typhus, small-pox, jaundice, malaria, and extensive burns, and results from a breaking down of the red blood corpuscles in the blood stream, and the consequent liberation of the hæmoglobin they contain, which then escapes into the urine. After transfusion of blood a similar solution of the red blood corpuscles may take place, and also as the result of poisoning with carbolic, pyrogallic, hydrochloric and sulphuric acids, chlorate of potash and other substances. Hæmaturia also occurs in a paroxysmal form, and is then usually the result of a chill, or of over-exertion, in persons who are liable to this affection.

**Bile in Urine.**—Bile appears in the urine in cases of jaundice, and is a common symptom in disease. It is usual to divide jaundice into two classes.

(*a.*) *Hepatogenous Jaundice.*—This arises from some obstruction to the outflow of bile. The ducts, small or large, may be obstructed by such conditions as catarrh, the presence of foreign bodies, stricture, or the pressure of tumours. In fact, anything which raises the pressure in the biliary passages above normal, leads to the absorption of the bile into the lymphatics, whence it passes into the blood and finally appears in the urine.

(*b.*) *Hæmatogenous Jaundice.*—Under this heading have been grouped cases in which the function of the liver-cells has been interfered with, as in acute yellow atrophy of the liver; and also those in which so extensive a destruction of blood-corpuscles is taking place that the liver is no longer able to deal with the pigment set free. To this latter class belongs the jaundice

caused by the poisons of such diseases as malaria, pyæmia, yellow fever, typhoid fever, &c., and by snake poison, phosphorus, and certain other drugs.

Although this classification is given here, it being the usual one when the subject of jaundice is considered, yet it must be added that it is very doubtful if a true hæmatogenous jaundice ever exists. Recent experimental results go to show that the transformation of blood pigment into bile pigment cannot take place outside the liver.

Bile may appear in the urine either as bile pigment (which is the commoner), or as bile acids. These will be considered separately.

**Bile Pigments in Urine.**—Bile pigment is formed from the hæmoglobin of the red blood corpuscles. When these corpuscles break up, the hæmoglobin is carried to the liver, where it is decomposed into albumin and hæmatin. The latter, taking up water and losing its iron, is changed to bilirubin, which is the pigment which appears in the urine of jaundice when freshly passed. When the urine is allowed to stand exposed to air, biliverdin is formed by oxidation, and when decomposition sets in in the urine, bilifuscin and other derivatives are formed.

The urine of jaundice, therefore, is of a colour varying from saffron yellow to dark brown, and the foam which readily forms when the urine is shaken, is notably tinged.

*Gmelin's Test for Bile Pigment* depends on the play of colours which ensues when urine containing the pigment comes in contact with nitric acid. If a little urine containing bile pigment is placed in a conical glass, and a few drops of nitric acid (which has been allowed to stand exposed to light for some time, and is therefore mixed with nitrous acid) are allowed to run down the edge of the glass and collect at the bottom of the vessel, a series of coloured rings will form in the following order, from above downwards: yellow, red, violet, blue, and green. A still better method is to filter the urine in quantity several times through the same filter, and then to allow a drop of nitric acid to fall on the surface of the filter. The play of colours ending

in green will then be very distinctly seen. In cases in which the pigment is present only in very small quantity, it may be necessary to precipitate the urine with milk of lime. The precipitate, collected on a filter, will show the play of colours when a drop of nitric acid is allowed to fall on it.

**Bile Acids in Urine.**—The salts of these acids may be found in the urine in considerable quantity. The best test of their presence is that of Pettenkoffer. When to a solution of these acids a little cane sugar is added, and then a drop or two of sulphuric acid, a beautiful purple colour develops itself.[1] On this reaction depends their detection in urine. In a small quantity of urine a little sugar is dissolved, a strip of filter-paper is dipped in, and then allowed to dry. If now a drop of sulphuric acid be allowed to fall upon the paper, a purple ring will appear round it if bile acids are present.

The substance to which is due this purple colour, gives a characteristic spectrum, consisting of two bands, one close to F, the other between D and E, close to E.

Unfortunately, other substances which may be present in the urine also give a similar reaction with Pettenkoffer's test, although the spectrum of the coloured substance does not appear to be the same as that afforded by the salts of bile acids. These substances include the phenols, pyro-catechin, stearin, and cholesterin.

Therefore, where exactness is required, it is best to isolate the salts of the bile acids by Plattner's method before using Pettenkoffer's test. For this purpose, after removing any albumin which may be present, the urine should be concentrated, and over-saturated with alcohol, which precipitates the salts. The filtrate, after being mixed with water, is precipitated with ammonia and acetate of lead. This throws down the lead salts of the bile acids. After washing, these salts should be dissolved in boiling alcohol and filtered hot. The filtrate, to which a little soda is to be added, is evaporated, and the

[1] This reaction really depends on the presence of furfurol, a trace of which is formed when sugar is acted on by strong sulphuric acid.

residue extracted with alcohol. From this extract the sodium salts of the bile acids may be precipitated by means of ether.

Besides Pettenkoffer's test, and the spectrum, a physiological test for bile acids may be employed, when they have been isolated in the manner just described. It is well known that the presence of these acids in the blood causes considerable slowing of the pulse in cases of jaundice. This action may be used as a test. To do this the heart of a frog is carefully exposed, touched with a solution of atropine to remove vagus inhibition, and a drop or two of a solution of bile salts painted on. A very marked slowing will take place.

## CHAPTER XXX.

## URINARY SYSTEM—(*continued*).

### SUGAR AND ALLIED BODIES IN URINE.

**Sugar.**—Even in normal urine a small quantity of grape sugar is present; but its quantity is so minute as not to give evidence of its presence with the ordinary tests which are about to be described. When, therefore, sugar is detected by their means, it is present in abnormal amount and constitutes the pathological condition termed Glycosuria.

In some persons, who appear to have a peculiar predisposition in this direction, the mere consumption of an excess of sugar is sufficient to cause glycosuria. This is probably to be explained on the supposition (which has certain experimental facts to support it), that when a large quantity of sugar is suddenly thrown into the intestine, the blood-capillaries are incapable of taking up all the sugar which is presented for absorption. A certain proportion of the sugar therefore, finds its way into the lymphatics, and so, escaping the liver, passes into the general circulation, and is excreted in the urine. In such persons, however, it is only excess of sugar, not excess of starchy food, which causes the glycosuria. Therein such cases differ from true diabetes.

Apart from this dietetic form, a permanent glycosuria indicates some one of the forms of

*Diabetes mellitus.*—This is a symptom of many very different morbid conditions which only agree in this, that, for one reason or another, there is an excess of sugar in the blood, which the kidneys get rid of, so producing glycosuria.[1]

[1] Possibly glycosuria in phlorizin poisoning may be an exception to this statement. See paper by P. A. Levene, "Studies in Phlorizin Glycosuria," *Journal of Physiology*, vol. xvii. (1895) p. 259.

There are two great clinical varieties of diabetes, the light form and the severe form. These differ in almost every particular, save in the fact that grape sugar is present in the urine in both.

In the light, or hepatogenic form, the urine contains sugar only when starchy food or sugar is taken, and not when the diet is composed solely of flesh and fat. The appearance of glycosuria in such cases is due to some disturbance of the glycogenic function of the liver, the hepatic cells being then no longer capable of transforming the sugar, absorbed by the intestines, to glycogen. Hence much sugar accumulates in the blood and is excreted in the urine. Indeed, in some of these cases, the interference with the liver function may be so slight that the appearance of sugar in the urine only takes place during digestion, while in the intervals the urine may contain no sugar.

The causes which give rise to this lighter form of diabetes are various. Many of them, such as traumatic and other nervous lesions, seem to act by interfering with the hepatic circulation. Glycosuria of this kind is also often associated with gout, and with various forms of dyspepsia. Beyond the fact that there is sugar in the urine, patients suffering from this form of diabetes do not exhibit the well marked and characteristic symptoms which are seen in the severe form.

The severe form of diabetes differs completely from the mild, not only in its symptoms and course, but also in its causation. In the lighter form, as has been said, the glycosuria disappears when the patient is put on a proper dietary, but in the severe form this is not so. In the latter, when all sugar and starchy foods are abstained from, the sugar in the urine may be diminished, but it does not disappear. The symptoms are also distinctive. There is much emaciation and loss of strength, much thirst, and an inordinate appetite. The face is usually brick-red, with some tendency to cyanosis, and the skin is harsh and dry.

It is assumed on reasonable grounds that the immediate cause of this form of diabetes is to be found in an altered metabolism in the muscle cells, and that this is in turn produced

by changes in the central nervous system. The muscle-glycogen is, in the muscles, converted into glucose, which, under normal circumstances, undergoes a further breaking up, which is lacking in cases of diabetes. The lesion which produces diabetes is also in some unknown way related to the functions of the pancreas, for destructive disease of that gland is found to lead to the severe form of diabetes.

### *Qualitative Tests for Sugar.*

The qualitative tests for urine containing sugar depend upon the coloration caused by boiling with caustic potash, upon the power grape sugar possesses of reducing hydrated oxide of copper, and upon the evolution of carbonic acid when fermentation is set up by the addition of yeast. In all cases albumin, if present, should be got rid of by coagulation and filtration before these tests are applied.

1. *The Caustic Potash Test* (Moore's).—The urine is mixed in a test-tube with an equal quantity of liquor potassæ, and the upper part of the mixed fluid heated to boiling in the flame of a spirit-lamp. If sugar is present, the heated portion will assume a dark-brown colour. Almost all urines, it must be remembered, darken slightly when thus treated; but the change is very marked when sugar is present. This test is not very delicate, but is readily performed, and is useful as a preliminary.

2. *Trommer's Test.*—To a small quantity of urine in a test-tube, one third of its volume of liquor potassæ is added, and then a drop or two of a solution of sulphate of copper. The precipitate which falls will redissolve (the more readily if sugar be present), and more of the copper solution must be added until a small quantity of the hydrated oxide remains as a precipitate. On boiling this mixture a yellow colour will show itself if sugar be present, and will pass into a reddish-yellow granular precipitate of the suboxide of copper.

3. *Test with Fehling's Solution.*—The method of preparing Fehling's solution will be described on p. 310. A small quantity of that solution is placed in a test-tube, heated to boiling, and

then a drop or two of urine added. If sugar be present, reduction of the copper in Fehling's solution will at once take place, giving rise to a red precipitate. Fehling's solution is liable to undergo decomposition when kept for some time, and it will then of itself become reduced on boiling. If, however, it be always boiled previous to the addition of the urine, no error can take place, for if the solution remain clear on boiling, it is in a fit state for use.

4. *Fermentation Test.*—Under the influence of yeast, grape sugar breaks up into alcohol and carbonic acid, and this evolution of carbonic acid has been made the basis of another qualitative test for the presence of sugar. It is most readily performed by taking two test-tubes or narrow phials, one filled with normal urine, and the other with the urine to be tested, which should be slightly acidulated with tartaric acid if it be alkaline, adding to each a small quantity of yeast, and inverting them over mercury. If sugar be present in the urine under investigation, carbonic acid gas will collect at the upper part of that test-tube. A few bubbles of gas may come from the yeast itself, but the second test-tube containing normal urine will show these also, so that any mistake is hardly possible.

Other tests, such as the phenyl-hydrazin test of von Jaksch (which is too delicate for ordinary clinical work), the Almen-Nylander test with bismuth, and the picric acid test, are sometimes employed.

*Quantitative Estimation of Sugar.*—A considerable number of methods have been devised for this purpose.

*Pavy's Method.*—The principle upon which Fehling's method for the volumetric analysis of sugar depends is the reducing action which that substance has upon hydrated oxide of copper, but the reaction is so much obscured by the red precipitate of the suboxide which is thrown down that the results are not very accurate. Pavy therefore devised the following method, in which ammonia is made use of to prevent the precipitation of the suboxide. If ammonia be added to Fehling's solution, and the mixture be boiled, a sufficiency of grape sugar may be added to the mixture to reduce all the copper and render

the solution colourless, without any precipitation taking place.

The preparation of the copper solution is carried out as follows:—An ordinary Fehling's solution is made by dissolving 34·639 grammes of pure sulphate of copper in water and diluting to 500 c.c. The solution so obtained is mixed with another solution prepared by dissolving 173 grammes of tartrate of potassium and sodium in water, mixing it with 100 c.c. of liquor sodæ (sp. gr. 1·34), and diluting the mixture to 500 c.c. When these two solutions, each of 500 c.c., are united, we obtain one litre of ordinary Fehling's solution. Of this solution 120 c.c. are now taken, mixed with 300 c.c. of strong ammonia (sp. gr. ·880), and diluted up to a litre with distilled water. This constitutes Pavy's standard solution, and of it 20 c.c. correspond to 0·01 gramme of grape sugar.

The analysis is carried out as follows:—A flask of about 80 c.c. capacity is taken and fitted with a cork, through which two holes are bored, one of which receives the delivery tube of a Mohr's burette, and into the other is adapted a bent glass tube to allow of the escape of air and steam. The burette, filled with the urine,[1] is fixed in its stand, and the flask, into which 20 c.c. of the copper solution have been measured is allowed to hang free, so that nothing may obstruct the full view of its contents. Heat is now to be applied to the flask, and after the solution has boiled for a few minutes, so that all air has been expelled from the flask, the urine is allowed to flow into it until the copper solution has become completely colourless. This marks the end of the reaction. The quantity of urine used contains 0·01 gramme of grape sugar.

*Method by Fermentation and Differential Density.*—The urine, if not already acid, should be rendered so by means of tartaric acid. Its specific gravity is to be carefully ascertained by means of an accurate urinometer, at the temperature for which the particular instrument used is constructed. To 200 c.c. of the urine about 2 grammes of dry yeast is added, and it is placed

[1] It is best in the first instance to dilute the urine in the proportion of 10 to 100.

in a flask closed by means of a stopper, through which a finely drawn glass tube passes, so as to allow of the escape of gas but not to permit evaporation. After standing in a warm place for twenty-four to forty-eight hours so that fermentation may be complete, the urine is to be filtered and its specific gravity again determined. The difference between the two readings of the urinometer, when multiplied by the empirical factor 230, gives the percentage of sugar in the urine.

*Method by Circular Polarisation.*—Grape sugar when in solution possesses this peculiar property, that if a beam of polarised light pass through it, the beam becomes rotated to the right, and the degree of this rotation is in exact proportion to the amount of sugar contained in solution, and the length of the column of solution which the light traverses. Several instruments have been devised for the purpose of measuring the degree of this right-handed rotation, and so estimating the quantity of grape sugar present. Of these, the best known is the saccharimeter of Soleil-Ventzke. Its construction is complicated, and I do not propose to describe it in detail. It consists of two short brass tubes lying in line, and containing various polarising prisms. Between these two tubes, fits in the tube containing the urine to be tested. By means of a milled head two quartz prisms are moved so as to compensate for the rotation effected by the sugar solution, and the amount of this movement is registered by means of an attached scale and vernier. When this scale stands at zero, and when no sugar solution is in the tube, the appearance presented on looking through the instrument is a circular field divided into two lateral halves, each of which presents the same tint. If now the tube containing diabetic urine be slipped into its place, the light becomes rotated, and, on account of the special arrangements of the instrument, the field of vision assumes a different colour on the two sides. By slowly moving the screw which commands the quartz prisms, these two colours become gradually altered in tint until they again exactly correspond to each other. The amount of movement required to effect this is now to be read off on the scale by means of the vernier, and by a simple calculation

we can learn the percentage of sugar in the urine in question. With a tube one decimetre long each degree of the scale represents 1 gramme of grape sugar in 100 c.c. of urine.

The urine must always be rendered perfectly clear by means of filtration before it is placed in the tube of the saccharimeter, and if it is highly coloured it is well to remove the pigment by precipitation with acetate of lead and filtration. Albumin rotates polarised light to the left, as has been already mentioned, hence it is absolutely necessary to get rid of this substance, if it be present, before the saccharimeter is used.

Laurent's polarimeter is more accurate in its readings, and is to be preferred. The calculation is the same as that of the Soleil-Ventzke saccharimeter.

Diabetic urine possesses, when the disease is fully developed, various well-marked characteristics. It is large in quantity, sometimes reaching so high a figure as 15 or 16 pints, and correspondingly pale, but nevertheless possesses a high specific gravity, ranging from 1040 to 1050, or even higher. The quantity of the nitrogenous substances excreted is usually, if not invariably, very much increased. The quantity of grape sugar excreted may, in severe cases, be as high as 25 or 30 ounces in twenty-four hours.

Other forms of sugar are also occasionally found in the urine, such as *Lactose* in the urine of women who are nursing, *Maltose*, and *Levulose*. The last of these is sometimes seen in diabetes. It rotates polarised light to the left, but its other reactions are those of glucose. *Inosit*, which is not a sugar, but belongs to the aromatic bodies, may appear in the urine of diabetes mellitus, but is not peculiar to this, occurring wherever there is polyuria, in diabetes insipidus and in cirrhotic Bright's disease, for example, and even in normal persons when much water has been taken.

In certain cases of diabetes there may be detected in the urine, towards the termination of the case, a peculiar etherial odour, which is often associated with the onset of diabetic coma. One or more of three allied substances are then to be found in

the urine, *aceton*, *di-acetic* acid and *β-oxybutyric acid.* These substances, which are found not only in the urine but also in the blood of diabetics, are believed to owe their origin to the largely increased breaking up of the albuminous tissues which occurs in that disease. Owing to the presence of these bodies, the blood and the tissues in cases of diabetic coma have an acid reaction, and it has been supposed that the phenomena of diabetic coma are due to this.

*Tests for Acetone.*—The urine is acidulated and distilled, and to the distillate is added a little caustic potash and a few drops of a strong solution of iodine in iodide of potassium. If acetone is present, crystals of iodoform at once separate. Other tests are sometimes used, such as that with nitro-prusside of sodium (which is not reliable), or that with freshly prepared yellow oxide of mercury which, when mixed with the distillate, gives a black colour on the addition of sulphide of ammonium.

*Tests for Di-acetic acid.*—Add to the urine drop by drop a solution of chloride of iron, so long as precipitation occurs. Then filter and add a few drops more. If di-acetic acid is present a Bordeau-red colour will be produced, but in as much as other substances in the urine, notably antipyrin, give this reaction, it is necessary to distil the urine after having acidulated it with sulphuric acid, and to repeat the test with the distillate.

*Test for Oxybutyric acid.*—This substance never occurs in the urine without di-acetic acid being also present. Having detected the latter, the urine may be subjected to fermentation, after which, if oxybutyric acid be present, it will be found that the fluid has the power of rotating polarised light to the left.

## CHAPTER XXXI.

## URINARY SYSTEM—(*continued*).

### URINARY SEDIMENTS.

In order to examine the sediment of a urine it is usual to allow the urine to stand covered for some hours in a conical glass, after which a drop or two of the sediment which has collected may be removed by means of a pipette and examined micro-

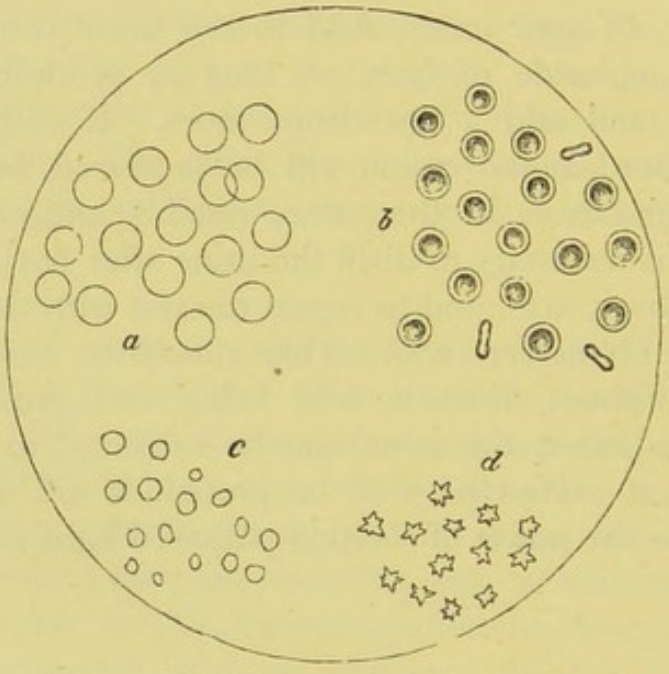



Fig. 70.—Blood Corpuscles in Urine. (Roberts.)

(*a*) Swollen up.
(*b*) Showing biconcave shape.
(*c*) Shrunken.
(*d*) Crenated.

scopically. A better method is to use a centrifuge, small instruments of this kind being specially made for the purpose.

Urinary deposits are divided into two classes—organic and inorganic. Of these the first is by far the most important.

*Organic Deposits.*

These include blood and pus corpuscles, epithelium, tube-casts, spermatozoa, and micro-organisms.

1. **Blood Corpuscles** are found in the urine in cases of hæmaturia (see p. 300). When the urine is acid the corpuscles may preserve for some time their normal appearance; but when it is alkaline, or very dilute, the red corpuscles swell up, lose

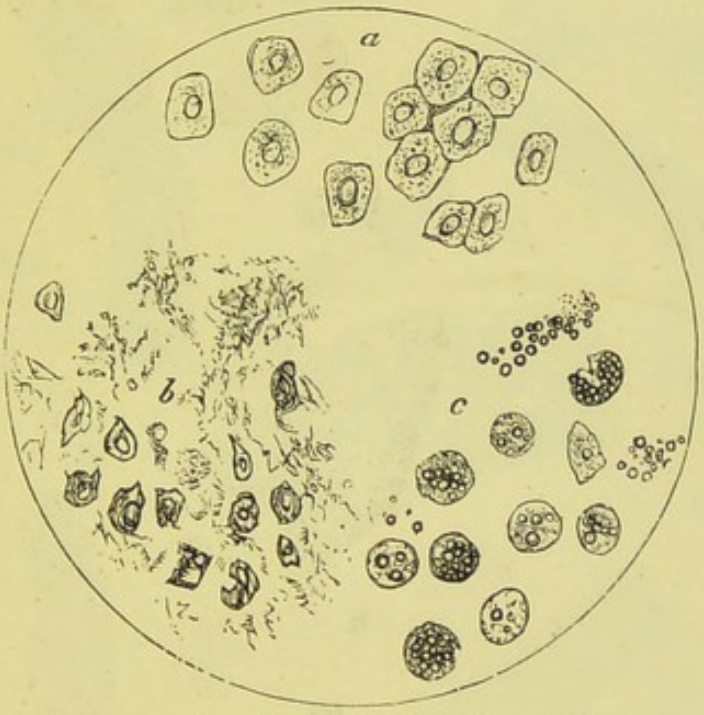



FIG.—71.—Renal Epithelium in Urine. (Roberts.)
(*a*) Natural appearance. | (*b*) Atrophied and Disintegrated.
(*c*) Fatty Degeneration.

their biconcave shape, and become pale. On the other hand, when the urine is concentrated they shrink up and become crenated. It is very rare to find rouleaux of corpuscles. They are only seen in cases of profuse bleeding from the bladder.

2. **Pus Corpuscles** when present in any quantity form a yellowish-white deposit, which is usually easily recognisable by the naked eye. Microscopically, the corpuscles present as a rule their normal appearance in acid and neutral urine, but

if the urine be alkaline, they swell up, become opaque, and tend to run together and form a homogeneous mass. If there be doubt as to whether a deposit consists of pus it is only necessary to add a small piece of caustic potash and to stir with a glass rod, when, if the sediment be formed of pus, it will become tenacious, glassy, and semi-solid.

The presence of pus in the urine is always a sure sign that

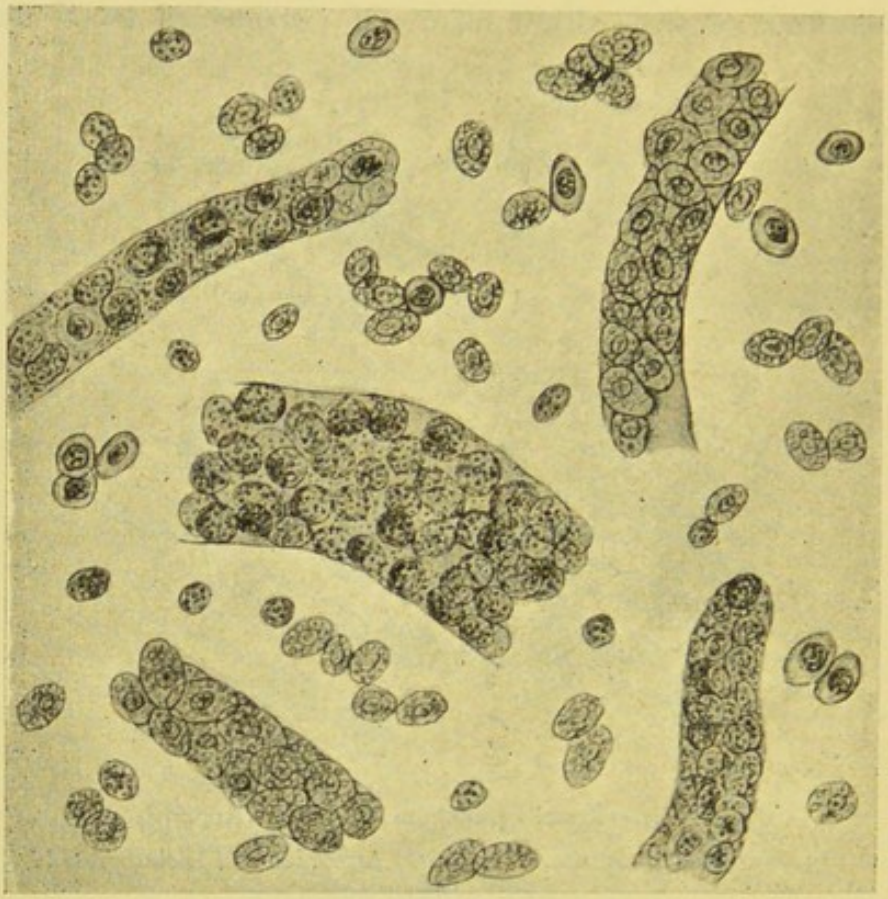


FIG. 72.—Epithelial and Pus Casts.

there exists an acute or chronic inflammation at some part of the urinary tract—renal abscess, pyelitis, cystitis, or urethritis. In women it must be remembered, pus flowing from the genital tract may become mixed with the urine.

3. **Epithelium.**—The epithelial cells found in the urine may be derived from any portion of the urinary tract. The epithelium of the urinary tubules consists of round or polygonal cells,

each having a large and sharply defined nucleus. Those of the pelvis of the kidney are conical, with one, or sometimes two, tail-like processes. The large irregular pavement epithelial cells which are often seen in the urine come from the bladder or vagina.

4. **Renal Tube-casts** are almost invariably associated with albuminuria, and most frequently with Bright's disease, but

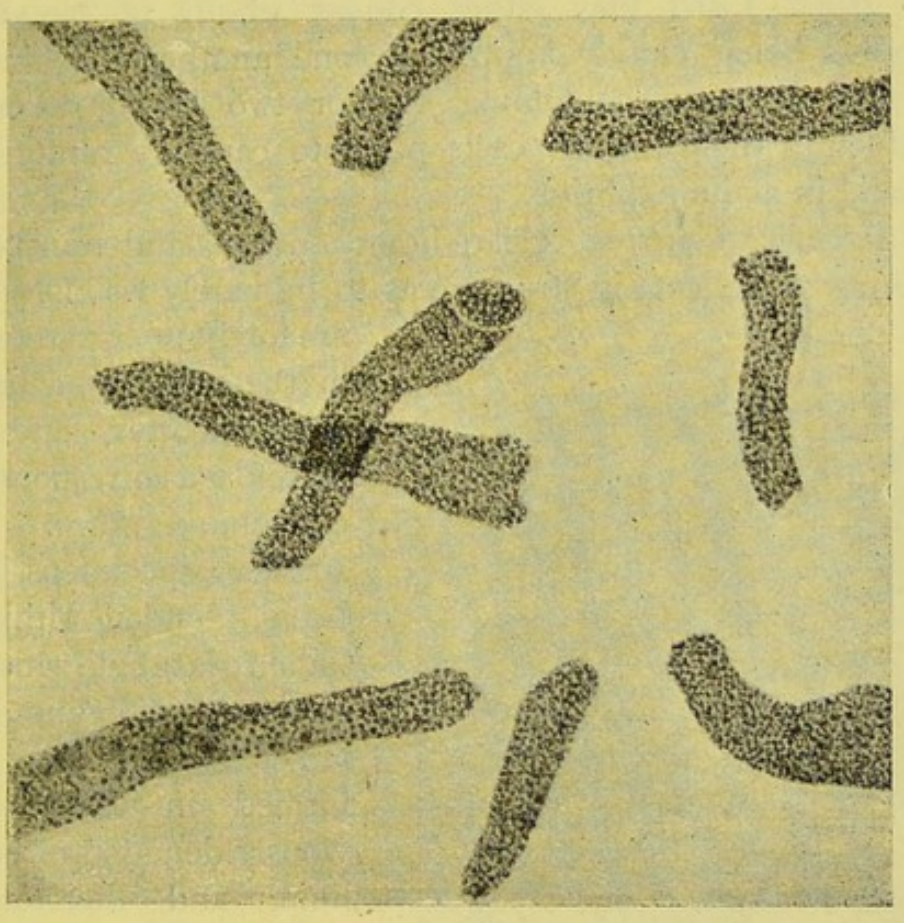


FIG. 73.—Granular Casts.

they occasionally occur when no albumin can be detected in the urine. They are, as their name implies, casts of the renal tubules, in the majority of cases of the convoluted tubules of the cortex. The chief forms of tube-casts are the following :—

(1.) *Epithelial Casts.*—In these the fibrinous cylinder has become covered over with epithelial cells which have been detached from the lining membrane of the tubule. These cells may be more or less cloudy and swollen.

(2.) *Pus Casts.*—Casts containing pus corpuscles embedded in them are sometimes met with.

(3.) *Fatty Casts.*—Very frequently casts are found studded over with oil globules. These globules are the result of fatty degeneration of the renal epithelium, and such casts are met with in the second stage of nephritis.

(4.) *Granular Casts.*—Dark opaque granular casts are also the result of epithelial degeneration in the renal tubules.

(5.) *Blood Casts* may either consist wholly of blood, the corpuscles being closely applied to one another, or fibrinous casts may be seen containing one or two blood corpuscles embedded in them. Such casts point to capillary rupture, and are found in acute nephritis.

(6.) *Hyaline Casts* are clear, homogeneous, and transparent, sometimes so delicate in structure as to be barely visible. They are for the most part formed in the convoluted tubules of the cortex, and have therefore a correspondingly convoluted form. The smaller specimens have been moulded within the lumen of a tubule which still retains its epithelium, while larger varieties have been formed in tubules previously denuded of epithelium, and therefore of greater capacity.

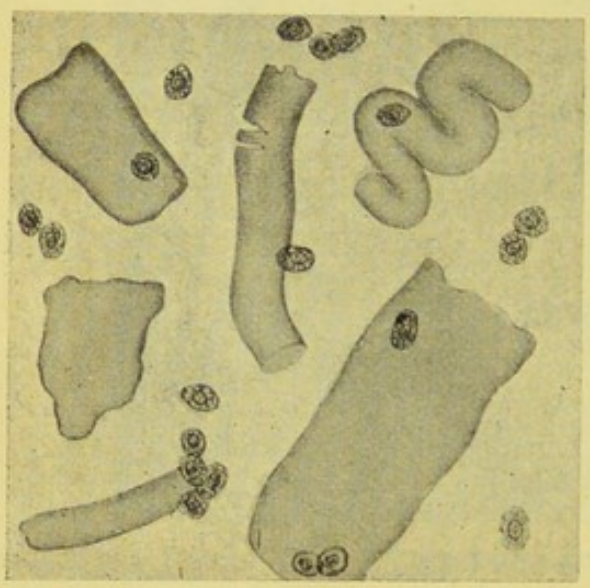


FIG. 74.—Hyaline Casts.

Occasionally hyaline casts may be found which exhibit the amyloid reaction, becoming reddish-brown on the addition of iodine, and dirty violet on the further addition of sulphuric acid; and giving also a beautiful violet with methyl-green, which tinges other casts green. Such waxy or amyloid casts are more strongly refractive than the ordinary hyaline variety, and being less flexible they exhibit deep fissures where

they have been torn asunder in passing through the straight tubules.

The student should be careful not to mistake for tube-casts these mucus-coagula which are so often found enclosing in their meshes whatever amorphous inorganic deposit the urine may happen to contain.

(7.) *Cylindroids*, ribbon-shaped structures, longer and larger than tube-casts, are occasionally found both in normal urine, and in cases of albuminuria. Their precise significance is not known.

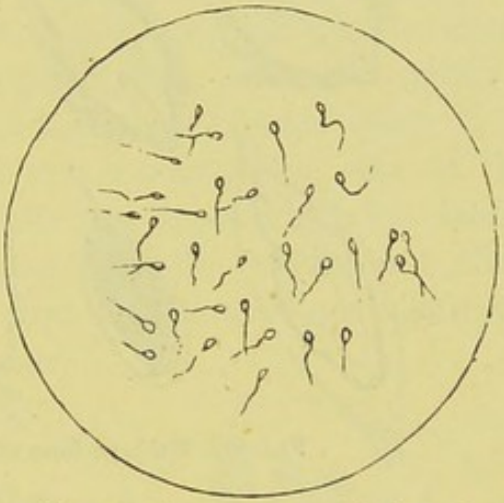


FIG. 75.—Spermatozoa. (Roberts.)

5. **Spermatozoa** are occasionally found in urine. They preserve their normal appearance for a considerable time. If the urine be very fresh, they may even be seen in active motion, but these movements are soon lost.

**6. Micro-Organisms.**—Very many forms of lower organisms are found in urine. Many of these only develop after the urine has been voided, and are derived from the atmosphere. Occasionally, however, urine as it leaves the body will be found to contain such organisms. These may be the result of the introduction of a catheter which has not been properly purified, but in other cases they are undoubtedly derived from the blood. The latter are most frequent in acute infectious disorder.

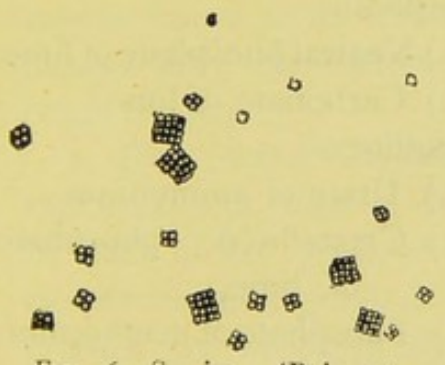


FIG. 76.—Sarcinæ. (Roberts.)

In cases of tubercular disease of the kidney tubercle bacilli may be found in the urine, the sediment being examined in the manner already described in connection with the sputum.

Occasionally the embryo forms of parasites, as for example

the Bilharzia hæmatobia, which infest the blood, are found in the urine.

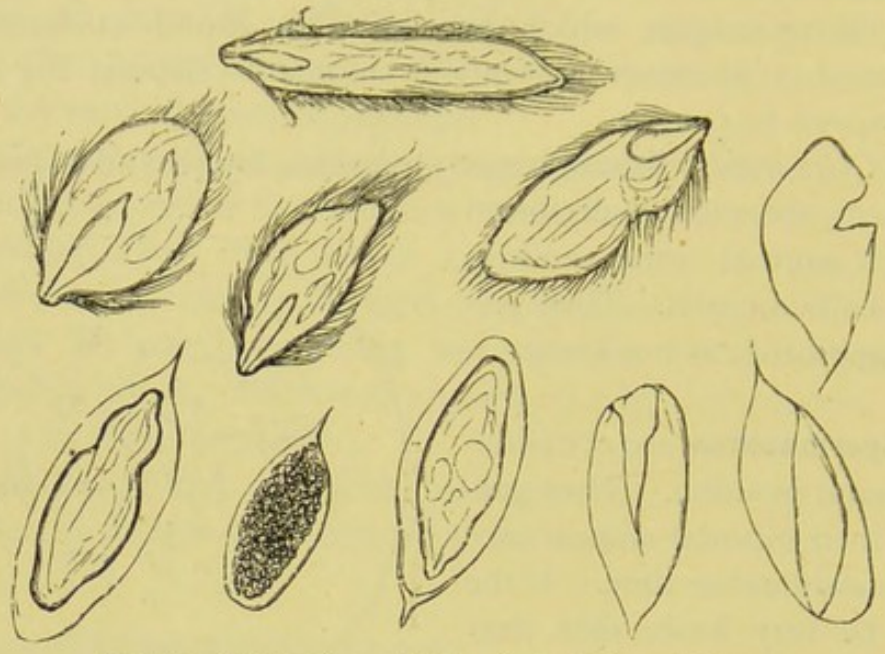


FIG. 77.—Embryo form of Bilharzia in Urine. (Roberts.)

### *Inorganic Sediments.*

The reaction of the urine in which the sediment is found gives an important indication as to its constitution, certain substances separating out only in acid urine, while others are only found when the reaction is alkaline. The following table shews what the physician may be prepared to meet with in each case :—

| *Acid Urine.* | *Alkaline Urine.* |
|---|---|
| Amorphous— | Amorphous— |
| Urates of potash and soda. | (*a.*) Neutral phosphate of lime. |
| | (*b.*) Carbonate of lime. |
| Crystalline— | Crystalline— |
| (*a.*) Uric acid. | (*a.*) Urate of ammonium. |
| (*b.*) Oxalate of lime. | (*b.*) Crystallised phosphate of lime. |
| (*c.*) Leucin. | (*c.*) Phosphate of magnesium. |
| (*d.*) Tyrosin. | (*d.*) Phosphate of ammonium and magnesium (triple-phosphate). |
| (*e.*) Cholesterin. | |
| (*f.*) Cystin. | |

## Sediments of Acid Urine.

1. *Urates.*—The amorphous deposit of urates, which is so frequently met with even in healthy urine, consists in the main of urate of soda, but may also contain urate of potash and of magnesia. These are, according to Roberts, in the form of quadri-urates. To the naked eye the deposit of amorphous urates has a reddish, brick-dust colour, due to pigmentation with uroerythrin. When the urine has been allowed to stand in a glass for some time, and deposit these urates, a peculiar bloom may be seen upon the sides of the glass when it is inclined, which is a characteristic and unmistakable sign of the presence of urates. Microscopically this deposit appears amorphous and finely granular. On warming the microscope slide the sediment becomes dissolved, and it separates out again on cooling, and the same reacton can be very readily seen with a larger quantity in a test-tube.

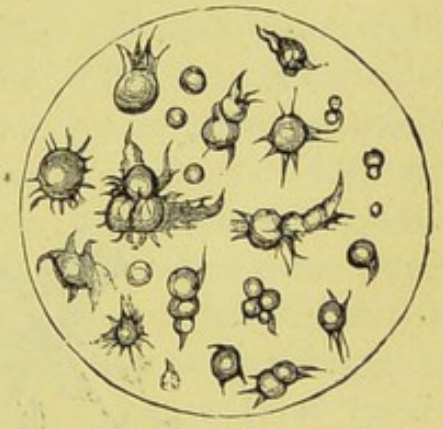


FIG. 78.—Urate of Soda. (Roberts.)

In health a deposit of urates often occurs after profuse sweating and violent exercise, in cold weather. Pathologically this deposit is found in all febrile conditions, in grave organic disease, particularly of the iver, and in dyspepsia. Very rarely a crystalline deposit of urate of soda may be found, in the form of irregular masses with spiny projections. These crystals are usually the result of gout.

2. *Uric Acid.*—Crystals of uric acid, when present in urine, can usually be seen as bright reddish-brown grains adhering to he sides of the glass, or forming a layer at the bottom. They closely resemble grains of cayenne pepper. Microscopically these crystals vary much in shape. They may take the form of four-sided tables, of six-sided rhombs, or they may be lozenge-shaped-ovoid, or barrel-shaped, or still more elongated and arranged in a stellate fashion. In whatever form uric acid appears the

crystals are always more or less yellow; and as no other crystal

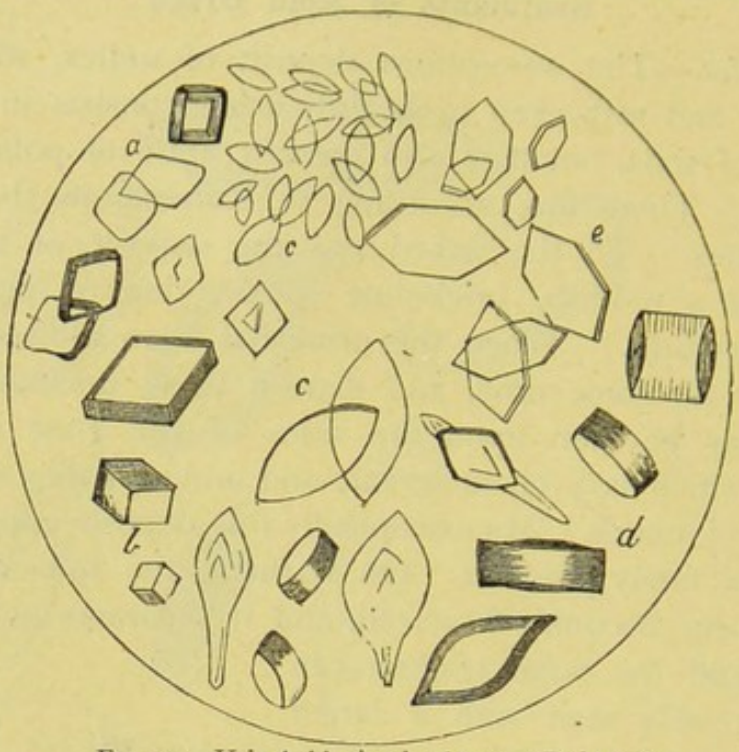



FIG. 79.—Uric Acid, simpler forms. (Roberts.)

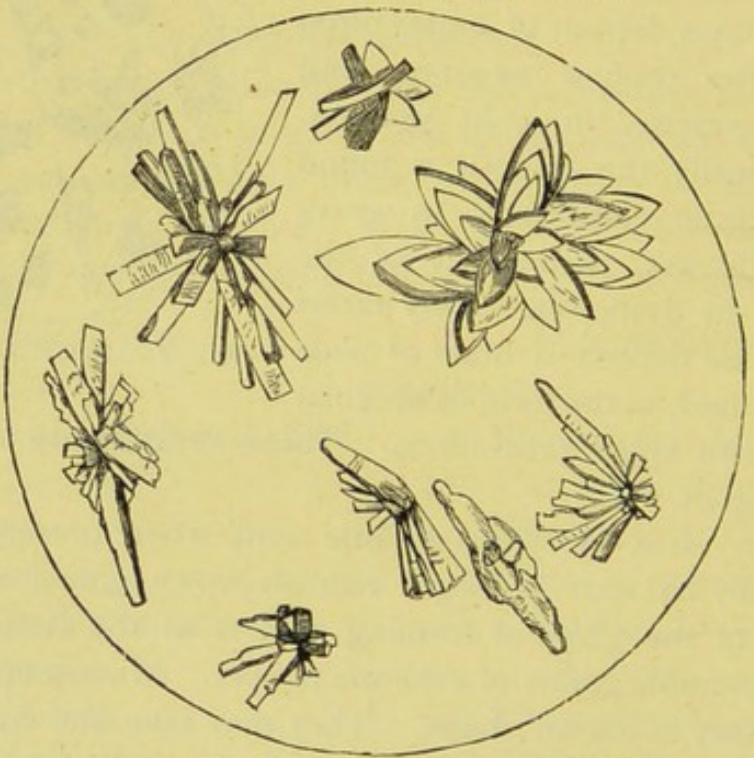


FIG. 80.—Uric Acid, stars and spikes. (Roberts.)

which spontaneously separates out from urine is so tinted, there can be no difficulty in its recognition.

3. *Oxalate of Lime* appears in the urine as small octahedra, which may be more or less elongated, or in the form of dumb-bells or small ovoids. To the naked eye the deposit appears as a white, undulating, clearly-defined layer, resting upon a greyer deposit beneath.

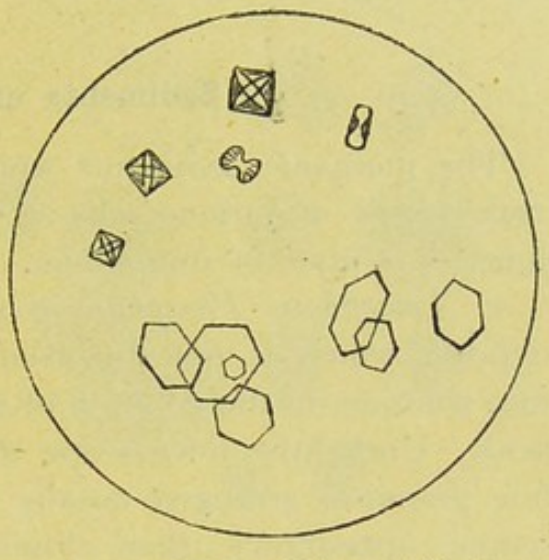


Fig. 81.—Oxalate of Lime and Cystin.

4. *Hippuric acid*, though always present in the urine (see p. 276), is but rarely found as a deposit. It crystallises in long rhomboidal prisms.

5. *Leucin and Tyrosin.*—Leucin appears in the urine microscopically in the form of larger or smaller yellowish-brown balls, which sometimes show distinct striation. Tyrosin, on the other hand, appears under the microscope as sheaves of silky glittering needle-shaped crystals. These two substances result from the decomposition of albumin and other nitrogenous bodies. Leucin and tyrosin are very rarely met with in urinary deposits. Their occurrence is almost confined to cases of acute yellow atrophy of the liver and of phosphorus poisoning.

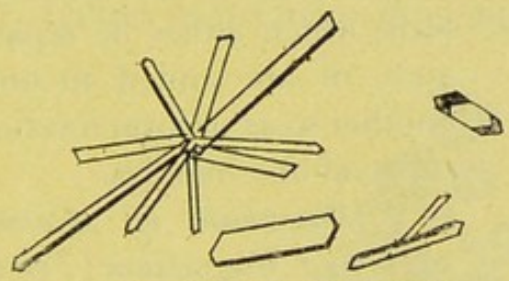


Fig. 82.—Hippuric Acid Crystals.

6. *Cholesterin* and other fats are found in the urine in cases of chyluria. The deposit consists of minute oil globules, and when dissolved in a mixture of alcohol and ether, and the solution allowed to evaporate, clear plates of cholesterin often crystallise out. Such plates are sometimes found in the urine in cases of cystitis.

7. *Cystin* appears in the urine in the form of six-sided plates, which are insoluble in water and in acetic acid, but which readily

dissolve in hydrochloric acid and ammonia. The pathology of cystinuria is very obscure.

### Sediments of Alkaline Urine.

The inorganic sediments which are found in alkaline urine may consist of various salts of phosphoric acid, of carbonate of lime, or of urate of ammonium.

1. *Amorphous Phosphate of Lime* forms a whitish flocculent deposit, which is not dissolved by heat, but at once passes into solution on the addition of a drop or two of acetic or nitric acid. Under the microscope this deposit is seen to consist of fine granules, arranged usually in irregular groups. In microscopic appearance they closely resemble amorphous urates, but the reaction of the urine will at once indicate their nature.

2. *Crystallised Phosphate of Lime* (*Stellar Phosphate*).—The crystals of this salt are found in the urine in the form of rods, which either lie separately or are united to one another so as to form rosettes or sheaf-like bundles.

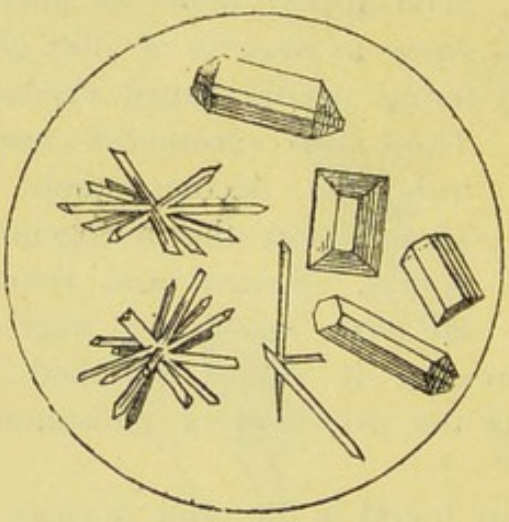


FIG. 83.—Triple Phosphate and Stellar Phosphate.

3. *Phosphate of Ammonium and Magnesium* (*Triple Phosphate*).—This salt forms comparatively large clear crystals which may frequently be recognised by the naked eye as bright sparkling points adhering to the sides of the glass. Examined microscopically, they are found to be of varying form, usually, however, having the shape of a triangular prism with bevelled ends, and presenting from above the appearance of a glass knife-rest. Most usually the deposition of these crystals is simply due to the ammoniacal decomposition of the urine.

4. *Phosphate of Magnesium* is occasionally, though rarely, encountered in the urine in form of crystals—flat tables elongated in shape, clear and glassy.

5. *Carbonate of Lime.*—In human urine carbonate of lime only occurs in an amorphous form. It dissolves in acetic acid with effervescence. In the urine of the horse it forms spheres marked with radiating lines.

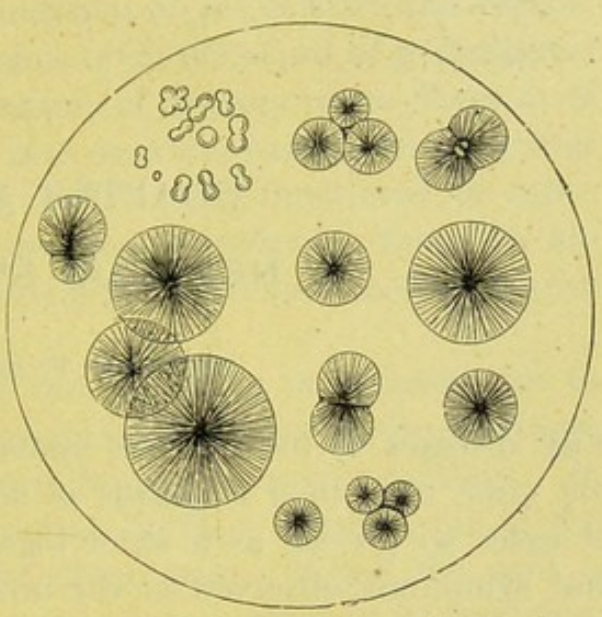


FIG. 84.—Carbonate of Lime (Roberts.)

6. *Urate of Ammonia* is found whenever the urine becomes strongly ammoniacal, in the form of opaque brownish spheres, which may be either smooth on the surface or may be covered with minute spikes. Sometimes this salt crystallises in the form of minute clear dumb-bells.

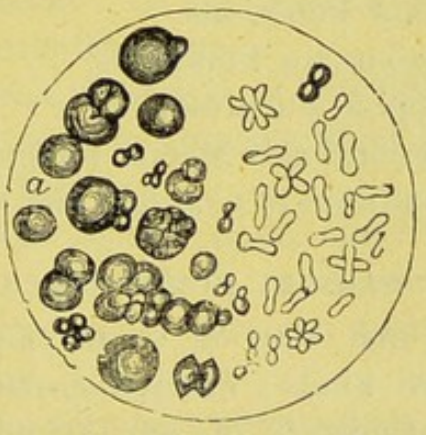



FIG. 85.—Urate of Ammonia. (Roberts.)

# CHAPTER XXXII.

## NERVOUS SYSTEM.

### SENSORY FUNCTIONS.

THE diseases of the nervous system are so complicated, and at the same time their diagnosis is now becoming in the majority of cases so precise as a strict logical deduction from the signs and symptoms observed at the bedside, that very special attention should be paid to the systematic arrangement of the inquiries which the physician has to make, and to the methods to be employed in testing the condition of the various functions of the nervous apparatus.

These functions will be considered in the following order :—

1. Sensory Functions.
2. Motor Functions.
3. Vaso-motor Functions.
4. Trophic Functions.
5. Cerebral and Mental Functions.
6. Condition of Cranium and Spine.

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### SENSORY FUNCTIONS.

The phenomena met with in connection with the sensory apparatus are of two kinds ; firstly, *subjective—i.e.*, such sensations as arise independently of any external irritation ; and, secondly, *objective—i.e.*, sensibility to external stimulation of various kinds. Although this classification is open to considerable criticism, it will be found to be convenient for purposes of description, and for clinical examination.

**Subjective Sensations.**—Among the many sensations of which patients complain, the following are the most important :—

1. *Pain.*[1]—Pain is simply an exaggeration of common sensibility. It may arise from stimulation of the sensory end organs, either by a physiological stimulus or as the result of pathological changes in the tissues. Irritation of sensory nerve fibres or of the posterior nerve roots will also occasion pain. An altogether different variety of pain may result from functional or other changes in the cortical sensory areas, rendering them so abnormally sensitive that physiological processes then give rise to sensations of pain.

Stimulation of the central sensory tracts is believed not to cause pain, and cerebral lesions, unless they involve the meninges or intracranial peripheral sensory fibres, are usually painless. In the same way spinal lesions, unless there is involvement of the posterior nerve roots, do not cause pain. When the pain is paroxysmal and follows the course of some nerve or its branches, it is termed *neuralgia*. It was first pointed out by Valleix that, when the nerve which is affected with neuralgia lies superficially, there can be found during an attack certain points upon the skin which are painful on pressure. Such painful points are usually situated where the nerve leaves an osseous canal or comes through a strong fascia. Of the many forms of neuralgia, the most commonly encountered are—

(*a.*) *Tic Douloureux*, neuralgia of the fifth nerve, which consists of paroxysms of pain corresponding in site to the nerve and its branches on the face.

(*b.*) *Intercostal Neuralgia* is, as its name implies, an affection of the intercostal nerves. It is to be carefully distinguished (as has already been said), from the pain of pleurisy and of muscular rheumatism.

(*c.*) *Sciatica.*—The pain here corresponds to the course of the sciatic nerve and its branches. It is usually localised between the tuber ischii and the great trochanter, and shoots downwards, sometimes as far as the heel.

(*d.*) *Visceral Neuralgiæ.*—These pains may be referred to the

[1] Many of the varieties of pain mentioned are not subjective, arising as they do from local causes, but they are classified here because they occur without any stimulation of the sensory apparatus on the part of the physician.

region of the heart and aorta (angina pectoris), of the stomach, intestines, liver, kidneys, uterus, or ovaries.

Besides these neuralgiæ there are various painful sensations which are met with in nervous diseases, and which must be looked for in such cases. Of these we may notice—

(*a.*) *Girdle Pain*, which is the sensation of having a cord or girdle tied tightly round the body. It may be felt at various levels, on the thorax, abdomen, or on the lower extremities at the knee or ankle. It is commonly met with in connection with inflammatory and degenerative changes in the cord, particularly in cases of locomotor ataxia, and is believed to result from excitation of the posterior nerve-roots.

(*b.*) *Lightning Pains.*—Among the most common symptoms in locomotor ataxia, are paroxysms of darting, lancinating pains, which shoot through the body, and which have received the name of lightning pains. They vary in position from minute to minute, now being felt in the area of one nerve, now of another. The skin over the affected areas is usually hyperæsthetic, and occasionally shews herpetic eruptions. These lightning pains are very characteristic of locomotor ataxia, though not absolutely pathognomonic of that affection, occasionally appearing under other conditions.

(*c.*) *Headache* in all its many forms.—Headache arises from a great variety of morbid conditions, such as vaso-motor changes within the cranium, abnormal composition of the blood, organic disease of the cranium or scalp, or of the brain or its membranes. The differential diagnosis of these different forms is to be found in special works on the subject. It is sufficient now to point out that the mode of invasion, the intensity, and the site of the headache must be exactly ascertained, as well as the presence of any obvious exciting cause.

2. *Paræsthesiæ*, or perverted sensations, are commonly met with among disorders of the nervous system.

(*a.*) *Sensations of Heat and Cold* (independently of actual elevation or depression of temperature as ascertained by the surface thermometer) are met with in intermittent fever, and also in various nervous diseases.

(*b.*) *Numbness.*—Any condition tending to depress the activity of the cutaneous sensibility may give rise to sensations of this kind, where the patient feels as if he were walking on a soft carpet, and has a tingling sensation up the limbs. It may also take the form of—

(*c.*) *Formication*, or the sensation of ants crawling over the skin. These two forms of paræsthesia are caused by affections of the nerve trunks (cold, mechanical injuries, &c.), or of the central organs (locomotor ataxia, hysteria, &c.), and perhaps sometimes of the peripheral terminations. Formication likewise arises occasionally after the administration of morphia, aconite, and ergot.

(*d.*) *Pruritus*, or itching, is caused by disease or irritation of the terminal end organs in the papilæ of the skin. It arises as a result of many skin diseases, particularly the parasitic varieties, and may also be caused by various chemical substances circulating in the blood—bile, sugar, hippuric acid, and perhaps xanthin and creatin.

3. *Giddiness* (*Vertigo*), is a sensation of swimming in the head, the body appearing to oscillate in different directions, and surrounding objects to rotate, and is accompanied with reeling and staggering. In Ménière's disease, and in disease of the cerebellum, vertigo is a frequent symptom; but it also arises from any condition which disturbs the circulation in the cranium, such as dyspepsia (*vertigo a stomacho læso*), heart disease, mental work, &c. &c. In many cases vertigo arises from paralysis of the muscles of the eye-ball, or from a contradiction between the impressions of external relations derived from two or more special senses.

4. *Abnormal Visceral Sensations.* — These comprise such sensory disturbances as pyrosis or water brash, boulimia or abnormal hunger, polydipsia or excessive thirst, and certain other similar symptoms. These have been already discussed in other parts of this work.

Such are the more important of the abnormal sensations complained of by patients suffering from nervous disorders.

We now turn to what is of much greater value in diagnosis, in that it admits of more precise determination—viz., the actual condition of the sensory functions as tested by the physician himself.

These sensory functions may be considered under two headings, viz., first, the cutaneous sensibility, and second, the sensibility of the deeper structures,—muscle, ligaments, joints, &c., although clinically, it is not always possible to separate rigidly the two sets of sensations.

In investigating these various sensory functions there are certain general points to be borne in mind. The methods about to be described are, many of them, of considerable delicacy, requiring on the part of the observer great care and patience as well as a certain amount of practice in the work, if the results are to be of value. But however careful the observer, no exactness of result will be arrived at with the more delicate methods, unless the patient can be induced to give his assistance. What is wanted should be carefully explained to him and his good-will secured. Unless the patient is fairly intelligent, and able to concentrate his attention, such methods as that of Weber, involving the recognition of two points, can hardly be employed with advantage.

No one of the methods of investigation about to be described is exact, and the results are in any case only approximate. The best results are obtained when the disorder of sensation is unilateral, for then the healthy side can be used for comparison.

In all cases it is well to test the patient's statements by means of blank control experiments. The eyes should be covered with a handkerchief in order to eliminate the sense of vision.

**A.—Cutaneous Sensibility.**—There are many forms of sensation connected with the skin, all independent of one another. We shall consider—(1) the sense of contact, (2) the sense of pressure, (3) the sense of pain, (4) the sense of temperature, (5) the sense of locality.

(1.) *Sense of Contact.*—This is most readily tested by touching the skin lightly with the tip of the finger, or with a feather.

The patient, whose eyes are covered, is asked if he feels; and occasionally blank control experiments where there is no touch are made to ascertain the correctness of his replies.

(2.) *Sense of Pressure.*—This may be roughly estimated by pressing more or less firmly with the tip of the finger, but more accurately by the method devised by E. H. Weber, which consists in the application of different weights over the portion of skin to be examined. In order to eliminate the muscular sense it is necessary that the part of the body to be examined should be carefully supported, and further, it is advisable to interpose some non-conducting substance, so as to prevent the temperature or size of the weight from being recognised, as these impressions might give some indication of its weight. Very rapid results can be obtained by the use of Eulenburg's *Baræsthesiometer.* This instrument is of simple construction, consisting essentially of a rod terminating in a vulcanite plate which is pressed upon the skin. The rod is so arranged that when it is pressed up into the frame in which it is held it comprises a spiral spring and at the same time indicates on a dial the amount of tension.

According to the observations of Weber a difference of weight in the proportion of 29 : 30 is appreciative on the finger tips, and on the forearm and leg of 18 : 20.

It is to be remembered that these results are not absolutely accurate, as the sensibility to pressure of the deeper structures cannot be eliminated.

(3.) *Sense of Pain.*—There is, unfortunately, no accurate means of estimating this sense. The method of pinching the skin by means of a graduated clamp is not satisfactory. It is better to prick the skin lightly with a fine needle. In health the slightest prick is felt as pain, and when, in disease, the prick is either not felt at all or is only felt as a touch, there is *Analgesia*, or loss of the sense of pain.

In the absence of a correct method it is fortunate for the observer that the electro-cutaneous sensibility runs fairly parallel with the sense of pain, and gives on the whole satisfactory indications, especially exact if the condition is unilateral and constant comparison be made with the sound side. One pole

of the faradic current, flat and well moistened, is placed on the sternum, and the other in the form of a brush is passed over the point of skin to be examined. The skin must be carefully dried before the observation is made. Beginning with a very weak current the secondary coil is brought nearer and nearer to the primary, until the point is reached at which the current is sufficiently strong to be felt. We should test, thus, point after point on the skin. The results obtained are of course only proportionate. Möbius gives the following figures as approximately correct :—

| | | | |
|---|---|---|---|
| Skin of face . . | 1·00 | Back of hand . . | 0·80 |
| Tongue . . . | 0·85 | Points of fingers . | 0·65 |
| Neck . . . | 0·95 | Thigh . . . | 0·80 |
| Chest . . . | 0·85 | Leg and back of foot | 0·75 |
| Back . . . | 0·80 | Sole of foot . . | 0·50 |
| Arm . . . | 0·85 | | |

(4.) *The Sense of Locality* is to be tested by pricking some portion of the patient's body when his eyes are closed, and making him indicate the site of the prick. In health the error is very small indeed, not greater than 2 c.m. Certainly an error of 4 or 5 c.m. is abnormal. The sense of locality may, further, be tested by ascertaining to what distance the two points of a pair of compasses, or of Sieveking's æsthesiometer, pressed upon the skin, must be separated before they can be recognised by the patient as distinct. Weber gives the following as the minimum distances to which the points must be separated, to be felt as different points, in health :—

| | | |
|---|---|---|
| On the point of the tongue, . . . . | 1·18 | m.m. |
| ,, palmar surface of last phalanx of finger, | 2·25 | m.m. |
| ,, ,, ,, 2nd ,, ,, | 4·5 | m.m. |
| ,, plantar ,, last ,, great toe, | 11·25 | m.m. |
| ,, back of the hand, . . . . | 31·5 | m.m. |
| ,, forearm and leg, and dorsum of foot, . | 40·5 | m.m. |
| ,, upper arm and thigh, . . . | 77·5 | m.m. |

(1 m.m. = 0·039 inch.)

To obtain reliable results with this method the two points should be made to touch the skin as nearly simultaneously as possible and with an equal pressure. They should be arranged so as to lie parallel with the axis of the body, not transversely. This method makes great demands on the exactness of the observer and on the intelligence of the patient.

(5.) *Sense of Temperature.*—Differences of temperature are very readily appreciated by the skin, especially so if the temperatures chosen lie between the limits of 27° C. and 33° C. In health, according to Nothnagel's observations, differences of 0·2° C. can be appreciated on the arm, of 0·2° - 0·4° on the cheek, 0·3° on the back of the hand, 0·5° - 0·6° on the thigh, and 0·4° on the dorsum of the foot. The best method of testing this sense clinically is to apply in succession to the skin two test-tubes, filled with water at different temperatures, and to ascertain whether the patient can appreciate the difference. Very great changes may be found in disease. Sometimes the patient feels cold well but not heat, or *vice versâ*, and, more rarely, cases may be encountered where heat is felt as cold, or cold as heat. This condition has been found to be associated with lesions of the Medulla and Pons.

Various other anomalies may be noted as occasionally met with in connection with cutaneous sensibility. There is, for example, the condition known as *Allocheiria*, in which the patient is ignorant from which side of the body a sensation comes, feeling a prick on the right leg as if it were on the left. In other cases the anomalous condition of *Polyæsthesia* may be encountered, in which the patient feels one point as two; two as three or four. Further, various *Perversions* of sensation may be encountered, a stimulus which in health produces one variety of impression, giving rise in the patient to another. For example a burning pain may be felt after the prick of a needle.

Any one of the different forms of cutaneous sensation may be delayed in transmission to the cerebral centres, an appreciable interval taking place between irritation and perception. In health the delay is but little over ·1 second, but in disease this period may be greatly overstepped. It is rare that the

sensations of touch or of temperature are delayed in transmission. The sense of pain is that most usually affected in this way, and then one may notice that the prick of a needle is felt at once as a touch, and only after an interval of a few seconds as a painful sensation.

**B.—Sensibility of the Deeper Structures.**—These structures include muscle, fasciæ, ligaments, joints, &c., and, as has been already said, their sensibility cannot, in the course of clinical examination, be entirely separated from that of the skin overlying them.

(1.) *Sensibility of Muscle.*—When one presses with the hand with moderate force over a muscle there is in health but little sensation. Under diseased condition, however, such pressure may develop pain.

(2.) *Muscular Sense.*—By this term is meant the power of appreciating the amount of muscular contraction during any action. It is usually tested by ascertaining how far the patient is able to detect the difference of various weights, using balls of the same size but of varying weight. These may be held in the patient's hand, or, if it is wished to test the muscular sense of the lower limbs, the weights may be slung in a towel over the ankle. According to Weber, a difference of $\frac{1}{40}$ can be appreciated in health. The results are complicated by the sensation derived from the pressure of the weight on the skin. Closely allied to this muscular sense is the—

(3.) *Power of appreciating the direction and extent of movements.*—This is best tested by making passive movements with the arm or leg of the patient while his eyes are shut, and asking him to indicate the position in which the limb has been placed. Or he may be asked to make certain movements, such as to touch the tip of his nose, ear, or great toe with his forefinger, while the eyes are closed. The power of appreciating weight and movement is often very materially affected, either by lesion of the psycho-motor centres in the cortex or (as in locomotor ataxia) from interference with the conduction of impressions upwards.

(4.) *Power of maintaining standing posture.*—This depends, as will be at once seen, on a very complicated mechanism, but it may be said generally that if, as so often happens in the earlier stages of locomotor ataxia, the patient can stand fairly well with his eyes open, but sways or falls if they are closed (Romberg's symptom), then the sensibility of the soles of the feet is diminished or lost. Interference with the sensations derived from muscles, joints, &c., may also produce this symptom.

In disease the various forms of cutaneous sensibility may be affected, either diminished (*hypæsthesia*), abolished (*anæsthesia*) or increased (*hyperæsthesia*) and they may be collectively or individually affected. When sensibility to pain is diminished the condition is termed *analgesia*, when it is increased, *hyperalgesia*. In the same way, we speak of *thermo-anæsthesia* and *thermo-hyperæsthesia*, when the sensibility to heat is diminished or increased; and other refinements of nomenclature have been devised by Eulenburg, such as *apselaphesia* and *hyperpselaphesia*, referring to tactile sensibility alone, the former indicating a decrease and the latter an increase. These partial affections of the cutaneous sensibility are frequently met with in cases of locomotor ataxia.

It is of importance to map out the area over which any disturbed sensibility prevails, and to ascertain whether it corresponds to the area of distribution of sensory nerves.

## CHAPTER XXXIII.

## NERVOUS SYSTEM—(*continued*).

### EXAMINATION OF THE SPECIAL SENSES.

### *Sight.*

For a full description of the various affections of sight, special works should be consulted. An outline may, however, be here given of the alterations which arise as the result of nervous affections, it being understood that all defects of vision caused by abnormalities in the refracting media and other local eye diseases are excluded from consideration.

In regard to the visual path, the reader may be reminded that the peripheral neuron lies entirely within the retina. From this start the fibres of the central neuron traversing the optic nerves to reach the chiasma where a partial decussation takes place, with the result that fibres from the right halves of both retinæ lie in the right optic tract, and those from the left halves in the left. Thus each optic tract contains fibres from both retinæ. Some are believed to run a long course, passing towards the external geniculate body and onwards through the posterior part of the internal capsule to the occipital cortex, but about these "direct" fibres there is some doubt. By far the most of the fibres in the optic tracts run a much shorter course terminating round the cells of the "primary optic centres" in the geniculate bodies, the Corpora Quadrigemina, and that portion of the Thalamus known as the Pulvinar. From these cells starts a third neuron the fibres of which, passing through the posterior part of the internal capsule, go to the occipital lobes (cuneus and cortex in calcarine fissure), spreading out in their course thither in a fan-shaped manner and being then known as the Optic Radi-

ation fibres of Gratiolet. The general course of these fibres may be seen in fig. 86.

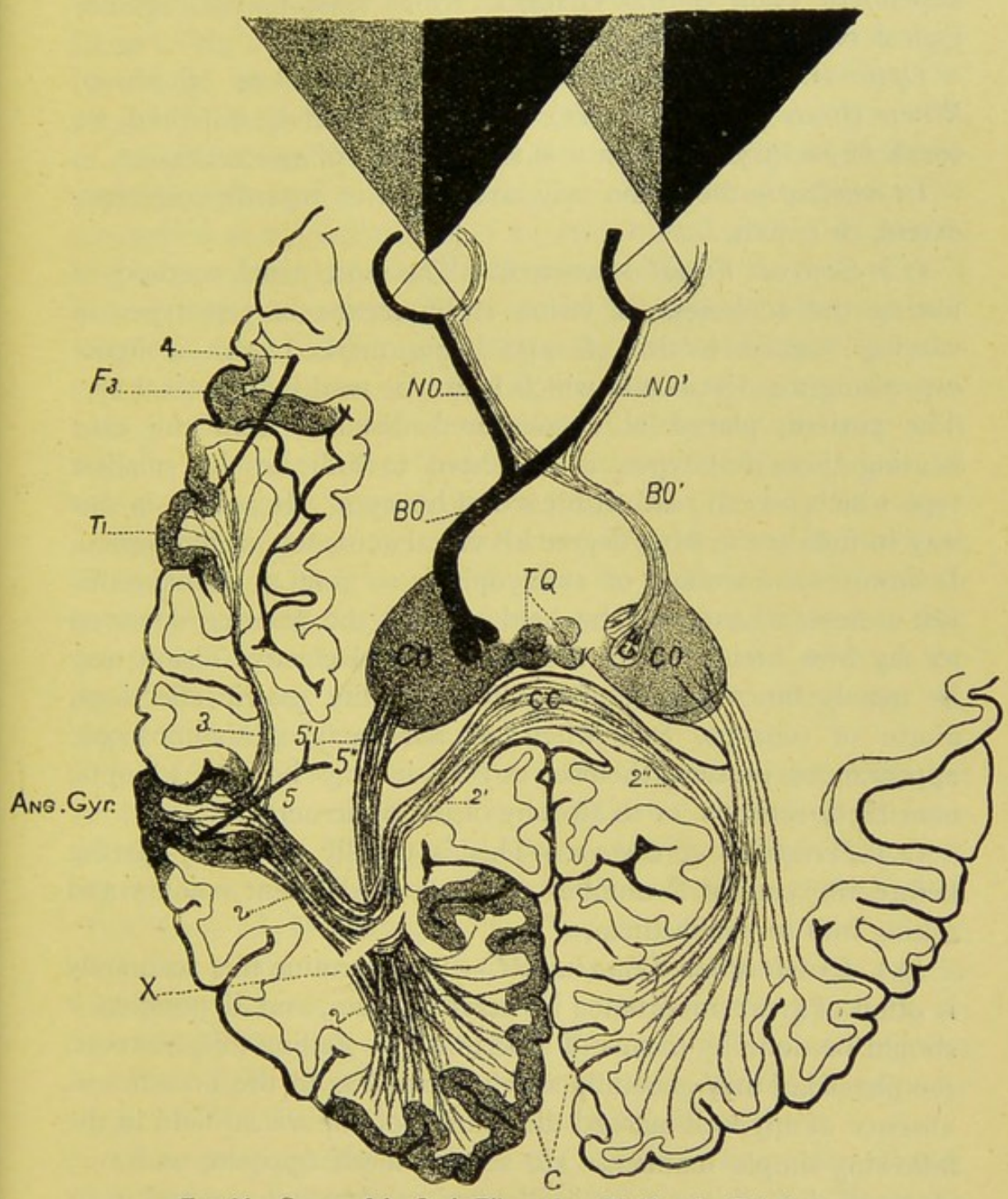



FIG. 86.—Course of the Optic Fibres (from Wyllie, after Dejerine).

*Optic Hyperæsthesia* and *Hyperalgesia*, the increase of the retinal sensibility — amounting to a painful sensation, with

intolerance of light—is common in meningitis and in cerebral hyperæmia, and illusions of sight often occur in insanity, dependent upon central changes. The most frequent pathological condition, however, is—

*Optic Anæsthesia*, the diminution or total loss of vision. When (from nervous causes) the sight is merely impaired, we speak of *amblyopia;* when it is entirely lost, of *amaurosis.*

In *amblyopia* the vision may be affected as regards acuteness, extent, or colour.

(1.) *Central Visual Acuteness.*—The most usual method of testing the acuteness of vision is by means of test types of varying size, each size of type being marked with a figure expressing the distance at which it can be read by a normal eye. The patient, placed at a measured distance from the card bearing these test types, is requested to indicate the smallest type which he can read with each of his eyes. It is easy in this way to indicate to what degree his visual acuteness is diminished. If however the amount of amblyopia is so great as to make this test useless, a hand may be held up and the patient requested to say how many fingers are extended. Defective vision may be merely functional, resulting from debility, gastric affections, abuse of tobacco, &c., when no abnormal ophthalmoscopic appearances present themselves; or it may be due to optic neuritis or retinitis, or to atrophy of the optic nerve.

(2.) *Perception of Colour.*—This is usually tested by placing before the patient a number of wools of different colours and asking him to match them.

(3.) *Extent of the Visual Field.*—To determine this accurately is often of great importance in nervous cases, and if possible it should be done by means of a perimeter. Failing this, however, the physician may generally satisfy himself as to the presence or absence of any well-marked diminution of the visual field in the following simple manner. He seats himself opposite and very close to the patient, who is directed to close one eye and to fix the other steadily on that of the observer. The latter should then move his hand from the periphere inwards from every direction, noting at what point it enters the visual field. Any

deviation from the normal condition can be thus readily detected. The visual field may be encroached upon either from the centre or from the margin. In the former case a dark spot (scotoma) forms in the centre and gradually enlarges. The scotoma may be steady; or scintillating, zig-zag, and brightly coloured, as in migraine. Such limitations of the field of vision are met with in functional amblyopia (especially from abuse of tobacco), in optic neuritis and in optic atrophy. The visual field for the perception of colours may also be encroached upon. In health it is to be noted that the area of vision differs in the case of each colour, being greatest for white—next yellow, then blue, red, and last of all, green. Colour-blind persons cannot distinguish red.

The field of vision may further be abolished as regards one-half of each of the retinæ, the line of demarcation being very sharply defined. This condition, known as Hemianopsia, is one of very great importance in diagnosis. It may be "horizontal," the upper or the lower halves of both visual fields being blotted out. This condition may be produced by a tumour pressing upon the upper or lower part of the chiasma. But much more commonly the dividing line is vertical and the right or the left half of each visual field is wanting (see fig. 86). This may take various forms.

(*a*) *Homonymous Hemianopsia.*—In this case either both left or both right halves of the visual field are more or less completely wanting. If both left halves are blotted out then both *right* halves of the retinæ have lost sensibility and *vice versâ.* This form of Hemianopsia may be caused by lesion of the occipital lobes, the optic radiations, the internal capsule, the primary optic centres, or the optic tracts. If the lesion be on the left side, then the right half of the visual field is wanting and we have right homonymous hemianopsia. When we come to speak of Aphasia this subject will be again alluded to.

In homonymous hemianopsia the condition is usually *absolute,* the portion of the visual field affected being dark. But occasionally cases of *relative* hemianopsia occur in which the perception of light remains while that for form and for colour has disappeared.

It has been said that homonymous hemianopsia may be due to any lesion which interferes with the visual tract at any point beyond the chiasma. Fortunately there is a means of localizing the site of the lesion a little more precisely, for, by means of Wernicke's symptom, it is possible to determine whether the part of the visual path affected is that which includes the reflex pupillary arc (in which case the lesion is in the optic tracts or in the primary optic centres), or whether this arc is not affected, the lesion then lying in the internal capsule, optic radiations or occipital lobes. To elicit Wernicke's symptom the patient is seated in a dark room with a lamp rather behind him. One eye is to be kept covered, while on the other some degree of light is to be thrown by a plane mirror. Taking now an ophthalmoscope mirror the observer reflects a strong beam of light through the pupil with sufficient obliquity that it falls only on the blind side of the retina. If this light causes the pupil to contract the inference is that the lesion lies beyond the primary optic centres, but if no contraction takes place, it is concluded that the portion of the visual part affected lies either in these primary centres or in the optic tract. As may be understood this symptom is not easily elicited and requires great care on the part of the observer.[1] A further localizing sign may be found in cases of relative hemianopsia, for this condition only arises in lesion of the occipital lobes. And, further, it may be pointed out that, according to Henschen, when homonymous hemianopsia is of cortical origin the patient may have hallucinations of vision in that portion of the visual field which has been blotted out.

Two other forms of hemianopsia remain to be noticed—

(*b.*) *Temporal hemianopsia.*—From the manner in which the fibres decussate in the chiasma any tumour pressure on the chiasma about the anterior or posterior angle may paralyse the function of the two inner retinal halves and cause temporal hemianopsia in both eyes.

(*c.*) *Nasal hemianopsia* is a rare condition. It may occur in

[1] In his *Klinische und anatomische Beiträge zur Pathologie des Gehirns*, Upsala, 1892-94 (*dritter Theil*, p. 108), Henschen discusses very fully Wernicke's reaction.

one eye from the pressure of a tumour on the outer side of the optic nerve, of the chiasma or of the optic tract.

**Movements of the Eyeball.**—Although strictly speaking this subject falls under the heading of the Motor Functions, it may for convenience be considered here.

The ocular muscles are supplied by three nerves,—the Oculo-motor or 3rd nerve, the Trochlear or 4th nerve, and the Abducens or 6th nerve. Affections producing irritation or paralysis in each of these give rise to spasm or paralysis in the corresponding muscles, and to consequent changes in the position and movements of the eyeball. These may be considered in turn.

1. **Paralysis of the Ocular Muscles.**—Without going into undue detail, certain general symptoms indicative of paralysis of the muscles moving the eyeball may be mentioned.

(1.) *Deviation.*—When the motion of the eyeball is limited, owing to paralysis of some particular muscle, we have what is called primary deviation. This is seen on the affected side. But in the healthy eye there may be secondary deviation, which is brought about thus. A very strong nerve impulse is sent to the affected muscle in order to get as much contraction as is possible in the paretic muscle. The same impulse sent to the healthy eye is much more than necessary. Hence the healthy muscle contracts too powerfully and secondary deviation is the result.

(2.) *Diplopia* or double vision. Owing to deviation, two images of a single object are formed, the true image in the healthy eye, the false in the paralysed eye. These two images, not falling, as they should do on corresponding parts of the retinæ, are seen separately and are not combined as in health.

(3.) *False Orientation.*—Another result of the ocular deviation is that an inaccurate idea is formed of the position of surrounding objects, and if the patient uses the paralysed eye in walking he is apt to take a zig-zag course.

(4.) *Vertigo* is not uncommon as a symptom of ocular paralysis. It is due to diplopia and false orientation. The contradictory impressions thereby produced give rise to an unsteady feeling which often develops into vertigo and sometimes even induces vomiting. The feeling of vertigo ceases at once when the paralysed eye is closed.

(5.) *Altered Carriage of Head.*—Patients suffering from ocular paralysis learn instinctively to carry the head in such a position as shall reduce the diplopia to a minimum. Hence each variety of paralysis shews a special attitude of the head, recognisable by a trained observer.

Of these signs, diplopia is the most important, and unless the paralysis is complete and the limitation of ocular movement therefore very distinct, it is necessary to test the diplopia by means of the method of double images. The patient should be seated in a dark room about five yards from a lighted candle. The sound eye being covered with a piece of red glass the true image will appear to be of that colour. The candle is now to be moved upwards and then downwards, to the right and then to the left, the relation which the two images bear to each other being noted at each position of the candle. This test obviously implies a considerable degree of intelligent co-operation on the part of the patient.

In the following pages diagrams[1] are given (modified from Fuchs) of the double images as they appear in the case of paralysis of each of the muscles moving the eyeball. These muscles are grouped under the heading of the nerves supplying them.

(1.) *Oculo-motor or 3rd Nerve.*—According as the paralysis is complete or incomplete, the whole, or only one or more of the following muscles are affected :—

(*a.*) *Levator Palpebræ Superioris.*—Paralysis causes drooping of the upper eye-lid—ptosis.

[1] In these figures the false image is indicated more faintly than the true.

(*b.*) *Superior Rectus.*—The eyeball turns downwards and slightly outwards when this nerve is paralysed, and there is in consequence diplopia or double vision, the result of the visual axis of the two eyes not being directed to the same object. As this divergence is increased on looking up, but does not exist when the eyeballs are both turned downwards, the patient instinctively carries the head well thrown back.

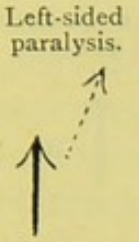



FIG. 87.

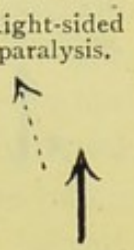



FIG. 88.

(*c.*) *Internal Rectus.*—Paralysis here gives rise to divergent strabismus (squint), with diplopia, the eyeball being rotated outwards on account of the unopposed action of the external rectus.

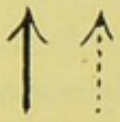


FIG. 89.

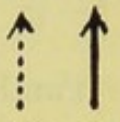


FIG. 90.

(*d.*) *Inferior Rectus.*—The affected eye is, in paralysis of this muscle, directed upwards and slightly outwards, and there is diplopia except when the object lies above the level of the eyes.

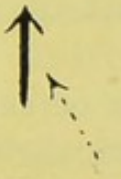


FIG. 91.

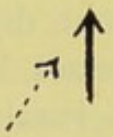


FIG. 92.

(*e.*) *Inferior Oblique.*—In paralysis of this muscle the eyeball is turned slightly downwards and inwards, but this condition is rarely observed, as paralysis of the inferior oblique as an isolated affection is exceedingly uncommon.



FIG. 93.

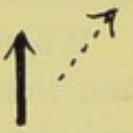


FIG. 94.

(*f.*) *Ciliary Muscle.*—The effect of paralysis of this muscle is that the patient is unable to accommodate the eye for distance. This point is subsequently alluded to.

When the 3rd nerve is paralysed as a whole all these actions combine, and the result is, that the lid droops, the eyeball is drawn downwards and outwards, and protrudes from its socket,

the pupil (as we shall presently see) is dilated and immobile, and there is defective power of accommodation.

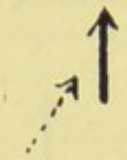



Fig. 95

(2.) *The Trochlear, or 4th Nerve*, supplies the superior oblique muscle, and when that is paralysed there is diplopia, the field of vision being moved downwards and outwards.

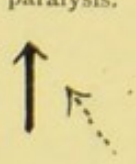



Fig. 96.

(3.) *The Abducens, or 6th Nerve*, supplies the external rectus, paralysis of which causes convergent strabismus, with consequent diplopia, there being no power of rotating the eyeball outwards beyond the middle line. This condition frequently gives rise to giddiness, nausea, and vomiting.

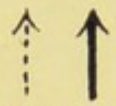


Fig. 97.

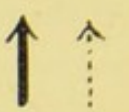


Fig. 98.

Paralysis of these ocular muscles may be either peripheral or central. The peripheral causes embrace many local affections within the orbit of interest chiefly to the oculist, but in addition paralysis of this kind is seen as the result of rheumatism, gout, diphtheria, influenza, neurasthenia, and various toxic processes, lead-poisoning, ptomaine poisoning, and the like.

Paralysis may, further, result from affections of the nerves in their course, in such diseases as meningitis (particularly the tubercular variety), hæmorrhage, tumours, syphilis, and the various results of middle ear disease. Ocular paralysis is also occasionally met with as a symptom of migraine.

When the paralysis is the result of disease of the nuclei of the three nerves (3rd, 4th, and 6th), the condition is termed ophthalmoplegia. This affection may take an acute form, analogous to acute anterior poliomyelitis in the cord, or it may be chronic. In this latter form it is not uncommon to meet with it in connection with locomotor ataxia, general paralysis of the insane, chronic bulbar paralysis, multiple cerebro-spinal sclerosis, progressive muscular atrophy, and syphilis. The frequency of ocular paralysis in locomotor ataxia is very considerable, and the paralysis is often transitory and one of the earliest symptoms of that disease.

Occasionally ocular paralysis is of cortical origin.

One other form of ocular paralysis remains to be noted, viz. :—

*Conjugate Deviation.*—When one desires to look at an object to right or left, both eyeballs move synchronously in the required direction, *i.e.*, the external rectus of one eye and the internal rectus of the other contract simultaneously. The impulse which produces this combined movement is believed to pass from the cortex to the nucleus of the 6th nerve, and thence through the posterior longitudinal fasciculus to the nucleus of the 3rd nerve on the opposite side.

Many gross lesions of the cerebrum interrupt this arc. If the lesion is a destructive one the eyes are turned away from the side of the body which is paralysed. For example, if there be conjugate deviation in a case of left hemiplegia from cerebral hæmorrhage, then the eyes will be turned to the right, the unaffected ocular muscles overpowering those whose nerve supply is interfered with. If, on the contrary, the lesion is an irritative one, the deviation is in an opposite direction, the eyes being then turned towards the side on which the muscular spasms occur.

This conjugate deviation is also seen in lesions of the Pons, and then the direction of the deviation is exactly the converse of that seen in cerebral lesions. If the lesion in the pons is a destructive one the eyes are turned towards the paralysed side, if it is an irritative lesion the eyes look away from the side on which the spasms shew themselves.

**Spasm of the Ocular Muscles** is much less common than paralysis. It is occasionally met with in hysteria, and in meningitis of the base, and, as has just been said, it is seen in connection with conjugate deviation of the eyes.

Clonic spasm of the muscles of the eyeball generally gives rise to a condition named *Nystagmus*, where the eyeball undergoes continuous oscillatory and rotatory movements which cannot be controlled by the will. It arises either from local abnormalities of the ocular structures, or from central nervous affections, such as locomotor ataxia, insular sclerosis, meningitis, hydrocephalus, &c. Coal-miners are frequently affected with nystagmus, owing to the constrained position and bad light in

which they have to work. In them the nystagmus is probably strictly analogous to writer's cramp in clerks, and to trade spasms generally.

**Intra-ocular Muscles.**—The condition of the pupil and its reflex actions, are of great importance in diagnosis. The size of the pupil varies with age, being large in the young, and small in the old; with the amount of light falling on the retinæ; with the nearness or remoteness of the object of vision and the convergence which is required; and with the excitability of the reflex centres. But in the normal state they are always equal. Irregularities in the shape of the pupil are apt to occur as the result of syphilitic iritis, and occasionally, though rarely, in locomotor ataxia.

To examine the pupils the patient should be placed opposite a window, in diffuse daylight (not sun-light), and, when the physician has examined them under this average amount of illumination and, if necessary, measured their size by means of a scale engraved on a slip of glass or other pupilometer, he should proceed to test the light reflexes. Of these there are two, the direct and the consensual.

*Direct Pupil Reflex.*—When the light falls on the retina, the stimulus thereby set up travels along the optic nerve, through the chiasma and the optic tract, and reaches the nucleus of the third nerve. Thence a centrifugal impulse passes down the third nerve to the circular fibres of the iris causing contraction of the pupil. To test this reflex, one eye of the patient should be covered, while on the other, which is being examined, the full light is allowed to fall. Having observed the size of the pupil, the eye should be screened for a few seconds by holding a card in front of it. The pupil will now be found to have dilated. When the card is removed it will contract to its former size.

*Consensual Pupil Reflex.*—The stimulus to which light, falling on one pupil, gives rise, travels, as has been said, down the optic nerve of that side. But, owing to the partial decussation in the chiasma and to the various commissural connections between

the corpora quadrigemnia on either side, and between the nuclei of the third pair of nerves, the motor impulse is a double one, and both pupils contract, even though only one retina has been stimulated by light. To test this consensual reflex the same process is gone through as that just described, with this important difference, that it is the pupil of the other eye, that which is kept carefully screened all through the observation, which the physician watches. Under normal conditions the shaded eye acts consensually, contracting and dilating simultaneously with the other on which the light is falling.

The observer should now determine another point, viz. :—

*Contraction of the Pupil with Convergence and Accommodation.*—Where the eye accommodates itself for near objects, the ciliary muscle contracts, the internal rectus contracts, and the iris contracts. In this way the lens becomes more convex, the eyeballs converge, and the pupils contract. This contraction can readily be observed by causing the patient to look first at a distant object, and then at a near one, such as the point of a pencil held near the eyes.

The active *dilating mechanism* of the pupil is under the control of the sympathetic and dilatation is probably brought about by the contraction of those muscular fibres of the iris which are placed radially. The centris lie in the cilio-spinal region of the cord, and in the medulla. Active dilatation of the pupil may be brought about by stimulating the skin of the neck by means of the faradic current. It also occurs under the influence of strong emotion, and of deep and forced respiration.

Certain of the diagnostic indications afforded by the reactions of the pupils may now be considered.

When one optic nerve is affected by disease and can no longer transmit impulses to the centres, the pupil on the diseased side no longer contracts when light falls on that retina. When, however, light acts on the healthy retina a consensual pupil-reflex will be observed on the diseased side. In the sound eye, on the other hand, the consensual reflex fails when light falls on the

paralysed retina. Both pupils react normally to accommodation and convergence.

When the lesion lies more centrally, affecting the fibres which connect the optic terminations with the oculo-motor nuclei (Meynert's fibres) the condition termed *reflex iridoplegia* (the Argyll-Robertson symptom) is brought about, in which the pupils no longer react to light, but do react in accommodation and convergence, a state of matters frequently met with in locomotor ataxia.

Passing still further round the reflex arc it is found that if the trunk or terminations of one oculo-motor nerve (say the right) is affected the following is the state of matters. The right pupil is larger than the left, and has lost both direct and consensual light reflexes, and also the contraction in convergence and accommodation.

The size of the pupil being as it were an expression of the balance between the dilating and the contracting mechanisms, and as either of these may under pathological conditions be so affected as to give rise either to spasm or to paralysis of the corresponding fibres in the iris, it is apparent that we may have four varieties of pupil change.

1. *Paralysis of Sphincter* (*paralytic mydriasis*) from lesion of the oculo-motor nerve or its nucleus. The pupil is dilated, and, as may be imagined, the direct and also the consensual reflexes are not to be obtained. This condition is produced by atropin, occurs occasionally as a form of diphtheritic paralysis, and is sometimes, though rarely, seen in locomotor ataxia.

2. *Spasm of Sphincter* (*spastic myosis*).—The pupils are in this case strongly contracted and no response can be obtained to light, accommodation, or sensory stimulation. This condition is seen where the intra-cranial pressure has been moderately increased as in cases of meningitis, tumour, etc.

3. *Paralysis of the Dilator* (*paralytic myosis*).—Here the pupils are moderately contracted. The light reactions are normal, but that occasioned by faradic stimulation of the skin of the neck cannot be obtained. This condition is seen when the nerve fibres passing to the dilator fibres of

the iris have been destroyed. This may occur from disease of the medulla, or of the spinal cord at its upper end (chiefly myelitis) or of the cervical sympathetic itself. This latter may be destroyed by the pressure of aneurisms, or other tumours.

4. *Spasm of the Dilator* (*spastic mydriasis*).—In this case the pupils are more or less widely dilated. The condition may result from any lesion irritating the cervical cord or the cervical sympathetic.

It is however not always possible to say to which of these four classes an abnormal pupil belongs. Speaking generally, the following are the chief conditions in which dilated and contracted pupils are met with :—

*Dilatation of the pupils.*

(1) In children and in neurasthenic persons where the reflex excitability is high.
(2) In certain affections of the eye—particularly myopia and glaucoma.
(3) From the action of atropine and other mydriatics.
(4) In all cases of coma save that following opium poisoning, or the result of a lesion involving the pons.[1]
(5) From irritation of the sympathetic nerve, or of the centres regulating the dilating mechanism.
(6) Atrophy of the optic nerve, the stimulus of light no longer acting to keep the pupil balanced.

*Contraction of the pupils.*

(1) In old persons.
(2) In certain affections of the eye, hypermetropia, retinitis, iritis, &c.
(3) From the action of such drugs as morphia, tobacco, physostigmin.
(4) From paralysis of the sympathetic, or destruction of the dilating centres in the cervical cord and medulla.

[1] We have also to except from this rule the case of coma in persons who from some cause already acting (such as locomotor ataxia), have strongly contracted pupils.

(5) From increased intracranial pressure—as in cases of cerebral tumours.
(6) From various lesions of the pons and medulla.
(7) In locomotor ataxia, general paralysis, &c.

Finally, there remains to be mentioned the curious condition of *hippus*, consisting in quickly and rhythmically alternating contraction and dilatation of the pupil, probably the result of clonic spasm of the sphincter of the iris. It is sometimes seen in various nerve affections, in oculo-motor paralysis, multiple cerebro-spinal sclerosis, epilepsy, neurasthenia, &c.

## CHAPTER XXXIV.

## NERVOUS SYSTEM—(*continued*).

### OPHTHALMOSCOPIC EXAMINATION.

The use of the ophthalmoscope ought to be a matter of routine in all cases of nervous disease, for the condition of the optic nerve and retina often throws much light on an otherwise obscure diagnosis. Of the many forms of apparatus which have been devised, probably the most convenient is that of Dr Gowers. It consists essentially of a concave mirror to throw the rays of light into the interior of the eye to be examined, pierced in the centre with a small hole through which the observer looks. Behind this mirror is fixed a disc bearing a series of convex and concave lenses, which can in turn be brought so as to lie over the aperture in the centre of the mirror, and thus come between the eye of the physician and that of his patient. There are also supplied along with the instrument two larger, bi-convex, lenses which are to be used as will be presently described. The ophthalmoscopic examination of the retina may be conducted in two ways, either with the simple mirror—giving an upright image,—the direct method, or with the mirror along with a lens—giving an inverted image, —the indirect method, as in fig. 99.

1. *Upright Image.*—The patient is placed in a dark room with a bright light at the side of his head on a level with the eye. The physician now takes up the ophthalmoscopic mirror, and resting its upper edge upon his eyebrow and looking through the central aperture, he inclines it so as to throw a strong beam of light into the eye of the patient. If the refraction of the eyes, both of the patient and of the observer, be normal, nothing

more is required than that the latter should bring his eye close to the patient and relax his accommodation, looking as it were at a distant object. The retina will then come into view as an upright image. Should the patient's eye be hypermetropic, it may become necessary, in order to obtain a distinct image, to employ a convex lens to counteract the divergence of the rays as they emerge from the hypermetropic eye. Suitable convex lenses are fixed in the rotating disc behind the mirror, and this disc should be turned, bringing lens after lens between the two eyes, until the one which suits the particular case has been found. In the same way in cases of myopia a concave lens must be interposed.

2. *The Inverted Image.*—To use the ophthalmoscope to obtain an inverted image of the retina, the observer places himself at a little distance from the patient, say about a foot off, and holding the mirror as before, he throws the light upon the eye to be examined. In his unemployed hand he holds a bi-convex lens between the thumb and index finger, and places it vertically between his eye and that of the patient, at a short distance, about $1\frac{3}{4}$-2 inches, from the latter. The lens will be held with greater steadiness if the small finger be allowed to rest on the eyebrow of the patient. In this way an inverted image of the retina is obtained.

Both these methods of using the ophthalmoscope should be made use of, as each has its special advantages. With the first, the area of the retina seen at one time is circumscribed, but is considerably magnified; with the second, the magnifying power is small, but a great deal more of the retina comes into view at one time. The use of atropine or homatropine to dilate the pupil may be necessary, but as a rule information sufficient for diagnostic purposes can be obtained without preliminary dilatation.

*Ophthalmoscopic appearance of the normal Fundus of the Eye.* The normal appearance of the fundus varies so much in different people, that it requires very considerable practice to be able to say that any individual eye is natural. It is best to make the

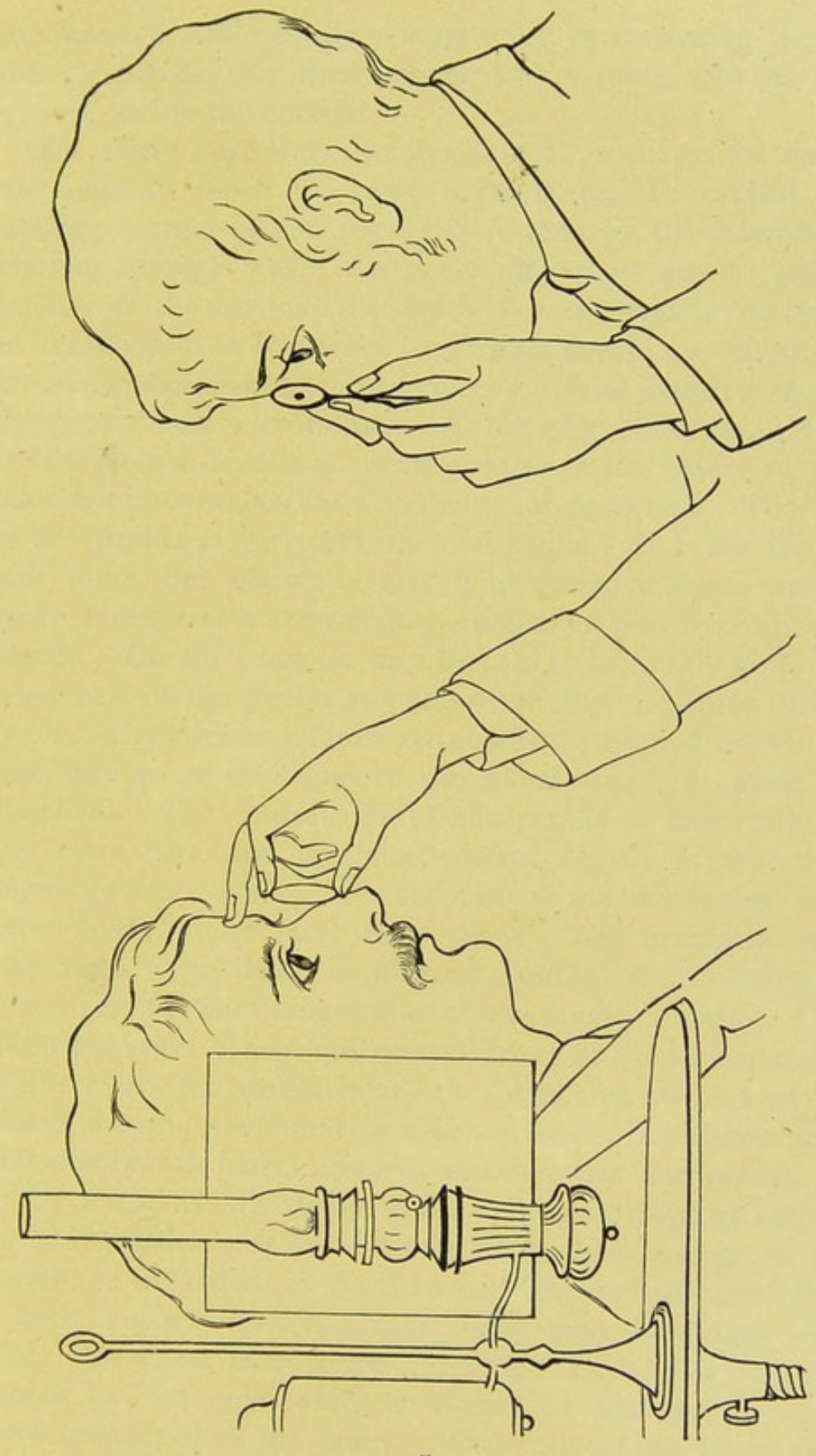


FIG. 99.—Indirect method of using the Ophthalmoscope.

examination methodically, commencing by inspecting the optic disc or papilla, and then passing to the retina with its blood-vessels, and to the choroid.

*The Optic Papilla* is usually elliptical in shape, the result of the angle at which the optic nerve enters the eyeball. The limiting margin of the papilla is formed by the sclerotic and choroid through which the nerve pierces its way to gain the interior of the eye, and inasmuch as most usually the aperture in the choroid is somewhat larger than that in the sclerotic, the latter membrane frequently shows as a faint bluish ring round the papilla—the sclerotic ring. At the edge of the choroid there is very often a deposit of pigment, more or less extensive, which must not be mistaken for a pathological condition. The *colour* of the papilla is very difficult to describe. It is for the most part white, but derives a very faint greenish lustre from the nerve fibres, and a somewhat more distinct pink from its blood-vessels. On the whole it may be said to be faintly pink. The outer half of the papilla is more white than the inner, owing to the fact that the nerve fibres are chiefly distributed on the inner side, and as a consequence the blood supply is there more abundant. The centre area of the papilla is more pale than any other part, and is almost always slightly excavated—the *physiological excavation*—on account of the divergence of the nerve fibres which there takes place. The excavation differs very much as to depth in different people, often showing at its lowest part the faint blue colour of the *lamina cribrosa*. Out of this excavation comes the central artery of the retina, and into it disappears the central vein—the branching of both of these vessels varying somewhat in different cases, but almost invariably one branch passes upwards and the other downwards.

*The Retina* is only visible to the ophthalmoscope by reason of its blood-vessels. It is perfectly transparent, except in some persons in the region of the macula. The retinal vessels follow a fairly uniform course, always being more abundant on the inner side where most nerve fibres lie. Each artery is accompanied by one vein, which is deeper in tint and about one-third broader than the artery. The walls of the blood-vessels

are invisible in the healthy fundus, it being only the column of blood which shews, but from the peculiar way the light is reflected from each red column, the edges appear as dark lines. The nerve fibres follow the same course as the arteries, but are almost always invisible, as they lose their sheath of myelin just as they enter the papilla. Occasionally, however, individuals are met with in whom some of the nerve fibres have retained their sheaths, and thus appear as brilliant white lines, stretching in patches, very much in the same line as the blood-vessels. In some persons the macula is invisible; in many, however, it appears dark-red, and surrounded with one or more oval white rings.

*The Choroid.*—The general tint of the fundus is chiefly due to the vessels and pigment of the choroid, the pigment giving rise to the more or less dark appearance of the ophthalmoscopic image. The amount of choroidal pigmentation varies much in different individuals, in dark-complexioned and dark-haired persons being deep, and in blondes showing little. In the latter case the pigment is so slightly deposited as to allow the blood-vessels of the choroid to appear, and this is still more marked in albinos.

### *Diseases of the Optic Nerves.*

The diseases of the optic nerves which can be recognised by means of the ophthalmoscope are mainly these—(1.) Congestion, (2.) Optic neuritis, (3.) Optic atrophy.

1. *Congestion of the Papilla* shows itself by an increased redness of the disc. The change of tint is, however, difficult to estimate, and the more important point is that the margin of the papilla loses its sharpness, and becomes more or less blurred and indistinct.

2. *Optic Neuritis.*—In this condition the red colour of the papilla deepens, and its edge becomes very indistinct, so much so as often to be unrecognisable. Along with these changes there is associated an œdematous swelling of the papilla (the so-called choked disc), the veins are much engorged and tortuous; the arteries, on the other hand, are distinctly reduced

in size. In well-marked cases the swollen papilla forms a prominent tumour with steep edges over which the blood-vessels disappear from view, to reappear in a different line on the neighbouring retina. Hæmorrhages frequently take place into the swollen papilla.

When the choked disc is present in both eyes it is an indication of increased intracranial pressure, and although this condition of double choked disc tells us nothing as to what the cause of the increased intracranial pressure is, yet in the great majority of cases it is due to cerebral tumour, and therefore, when choked discs are seen, we should at once look for other indications of tumour. It is to be remembered that this change in the optic nerves occurs early in cases of brain tumour, and that the discs may be very markedly affected before the patient shews any defect of vision. Indeed, if the vision is lost or greatly impaired early in the case, this would point to some factor acting on the optic nerves other than the mere increase of intracranial pressure. Early blindness along with choked discs would for example be produced by a tumour pressing on the chiasma, or interfering with the 3rd ventricle. In such cases the state of the pupil reflexes would give important indications (see p. 346). Choked discs may also be caused by hydrocephalus, by meningitis, especially of the tubercular form, and, rarely, by cerebral abscess.

Optic neuritis may also be occasioned by various pathological conditions within the orbit, by such acute diseases as typhus, pneumonia, or scarlet fever, and by poisons such as lead, alcohol and tobacco. There is a special form of optic neuritis met with in cases of Bright's disease, which will be mentioned in connection with albuminuric retinitis.

3. *Atrophy of the Optic Nerve.*—There are three varieties of this condition.

(*a.*) Primary optic atrophy, the result of locomotor ataxia, general paralysis, multiple cerebro-spinal sclerosis, or, more rarely, amyotrophic lateral sclerosis. On ophthalmoscopic examination the disc is found to present a greyish red or grey colour, with sharply marked edges and the vessels (at any rate

in the earlier stages), are normal in size. The appearances are in many cases not well marked, but when the field of vision is subsequently examined, it will be found that if atrophy is present, the visual field, particularly that for colour, is much contracted.

(*b.*) Secondary atrophy may arise from disease of the cortex (probably in or near the supra-marginal gyrus), or from pressure on the optic tracts, on the chiasma, or on the optic nerve itself. In this form the disc is very white, and the arteries and veins are contracted.

(*c.*) Consecutive atrophy is due to preceding optic neuritis, retinitis or choroiditis. The disc is white with indistinct margins, and while the arteries are contracted, the veins are frequently very large and tortuous, and at their edges they are marked with white lines.

### *Diseases of the Retina.*

1. *Papillo Retinitis.*—When the lesion of the papilla which has just been described as optic neuritis advances to a certain point of intensity, the retina itself becomes affected, being infiltrated with transudation. The veins are much engorged, and small hæmorrhages result. These lie, as a rule, in the neighbourhood of the disc, and though of no great size can be readily recognised. The retinal nerve fibres degenerate and become visible at scattered points of the fundus as opaque white patches, and, the perivascular tissue undergoing proliferation, the vessels become bounded with white lines.

2. *Albuminuric Retinitis.*—In cases of Bright's disease there is often found a degree of retinitis resembling very closely the papillo-retinitis already described. The cases are, however, peculiar in respect of the amount of hæmorrhage and the rapidity with which the extravasated blood undergoes fatty degeneration. There are thus left numerous white patches which are often arranged in a peculiar and characteristic stellate manner. A similar form of retinitis sometimes occurs in connection with diabetes.[1]

[1] There are several other retinal changes with which the physician ought to acquaint himself, but for these special works must be consulted, such as Gowers' *Medical Ophthalmoscopy*.

## CHAPTER XXXV.

## NERVOUS SYSTEM—(*continued*).

### HEARING.

In connection with this sense we often meet with subjective sensations, which from their persistence are frequently complained of by the patient. These consist of roaring, humming or ringing sounds (*tinnitus aurium*) which may or may not be accompanied with giddiness. In many cases such symptoms result from disease of the middle ear, and here fall within the domain of the surgeon. When such is not the case, they arise most frequently from affection of the inner ear, rarely from disease of the auditory nerve, or of the central organs in the brain. The first of these conditions is met with in cases of Ménière's disease, in which ringing in the ear, and intense vertigo occur in paroxysms. Although the nerve fibres engaged in transmission of sound impressions (cochlear nerve) and those in connection with the sense of equilibrium (vestibular nerve) run for some distance together, yet it sometimes occurs (as in locomotor ataxia) that the latter are alone affected, so that auditory vertigo results without any deafness. In the brain these two sets of nerves divide to proceed to different centres, and thus, when subjective sensations of hearing result from central brain causes, we find that when the auditory centre is affected we have tinnitus or deafness and no giddiness, whereas when the affection is in the cerebellum the conditions are reversed, giddiness and not deafness resulting. Ringing in the ears may be the result of over stimulation of the auditory apparatus by loud or long continued sounds, and it is frequently observed to result from the administration of quinine and sali-

cylic acid; and, finally, it must not be forgotten that tinnitus is common where there is anæmia, being then perhaps not purely subjective but probably resulting from the anæmic murmurs in the vessels to which reference has been made in a former chapter.

Passing now to the physical examination of the ear, it may be briefly said, that so far as affections of the outer and middle ear are concerned, the method of examination should be conducted by means of a speculum, light being reflected into the instrument from a concave mirror held by the examiner. For details of manipulation special works on the subject should be consulted; in which, also, information regarding changes in the appearance of the tympanic membrane will be found. The condition of the Eustachian tube should also be ascertained.

*Perception of Sound Waves.*—The degree of acuteness with which sound waves are perceived in an individual case is ascertained by means of a watch. The normal distance at which the ticking of the particular watch employed can be heard, should first be noted. The watch is then held at that distance, and gradually brought nearer to the patient's ear until the sound is perceived. Having ascertained this distance, it is then advisable to test the patient's power of perceiving a whispered voice—each ear being tested separately. For medical purposes, however, the third test is the most important. It is directed to ascertain the condition of the nervous apparatus in the inner ear, and is performed by means of a tuning-fork. If the stem of a vibrating fork be applied to the vertex or to the teeth, the vibrations are communicated through the bones directly to the labyrinth, and in normal conditions are perceived equally on both sides. If this is so, we may safely conclude that the terminal nerve organs of hearing are intact, and this even if deafness to ordinary sound exist. In such a case the deafness must be due to some affection of the middle or external ear, and the very obstruction which prevents sound from reaching the labyrinth equally prevents sound from escaping, so that when the fork is applied to the head, the sound is heard most loudly in the deaf ear. The converse, is, however, not invariably true. In persons below the age of forty we may

indeed conclude that if the tuning-fork is not well heard on the vertex, the auditory nervous apparatus is at fault, but after that age a degree of bluntness in the perception of these vibrations is not uncommon.

Hyperæsthesia of the auditory nerves is occasionally seen in hysteria, in acute febrile diseases, and in insanity. It may also result, according to some, from paralysis of the stapedius muscle with consequent over-tension of the *membrana tympani*, in cases of facial paralysis where the lesion lies above the origin of the branch to that muscle. More important clinically is the subject of auditory anæsthesia. In the great majority of cases deafness depends upon disease of the outer or the middle ear. We have already shown how nervous deafness is to be distinguished from these. The diagnosis of affections of the auditory nerve in its course, and of its centres can only be made by means of the other symptoms.

## Taste.

The sense of taste is located in the surface of the tongue, fauces, and back wall of the pharynx. The root of the tongue (circumvallate papillæ), fauces, and pharynx are supplied by the glossopharyngeal nerve. The fibres conveying the sensation of taste leave the glossopharyngeal to pass (according to Gowers) to the fifth nerve by the tympanic nerve and small superficial petrosal through the otic ganglion. The taste nerve for the anterior two-thirds of the tongue, on the other hand, is the lingual, and the majority, if not the whole of the sensory fibres of taste, pass from the lingual into the chorda tympani, and then to the facial, which nerve, however, they leave at the geniculate ganglion, to pass through the large superficial petrosal nerve to the fifth nerve, in the trunk of which they pass to the brain.

To test the sense of taste the patient should be made with closed eyes to protrude his tongue, on different points of which the substances in solution used in testing are to be deposited by means of a glass rod. For *bitter* tastes, solutions of quinine, or infusion of quassia may be employed; for *sweet*, syrup is the most convenient; *acid* taste will be produced by the application

of vinegar or dilute acids; and *saline* by means of solutions of common salt. Sweet tastes are best felt at the tip of the tongue, acid at the edges, and bitter at the root of the organ. To obtain correct results the patient must indicate what variety of taste he feels *before* he moves his tongue back into the mouth. The best way to accomplish this is to write on a slip of paper the words "sweet," "bitter," &c., and to ask the patient to point to the word expressing the taste he feels. After this the mouth should be carefully rinsed with water before the sense is again tested. These substances should be taken in the following order, sweet, saline, acid, bitter. Another and very convenient method of testing the sense of taste is by means of a galvanic current. Hyperæsthesia of the sense of taste is rarely met with, but it occurs occasionally in cases of hysteria. Paræsthesiæ or abnormal sensations of taste are sometimes met with in insanity.

*Anæsthesia* of taste may be peripheral, due to a coating of fur on the tongue, or abnormal dryness of the mouth, or to the action of heat or cold. It may be also due to defective conduction, from disease of the nerves of taste in their course. In this way it may arise from lesion of the glossopharyngeal, when the defect of taste will be limited to the root of the tongue and fauces, but owing to the nerve path already indicated, lesions of the nucleus of the glossopharyngeal nerve do not affect taste, whereas those of the nucleus of the 5th may do so. When the anæsthesia involves the anterior two-thirds of the tongue, it is due to the fibres of the lingual nerve, the course of which has been already pointed out. The affection of these fibres has considerable diagnostic value. 1. If taste is thus lost along with loss of ordinary tactile sensation in the tongue, and without other indications of affection of the fifth nerve, then the lesion is in the lingual. 2. When the chorda tympani is alone affected, taste is lost on the anterior two-thirds of the tongue without tactile sensation being affected. This often occurs in connection with diseases of the middle ear. 3. Where facial paralysis accompanies the loss of taste, then the lesion is situate on the nerve between the geniculate ganglion and the

point at which the chorda tympani leaves the nerve. 4. When symptoms of affection of the second division of the fifth nerve accompany the loss of taste, the lesion lies on the nerve tract, between the spheno-palatine ganglion and the gasserian ganglion. 5. When the loss of taste is accompanied by total anæsthesia over the region supplied by the fifth nerve, the lesion probably lies at the root of that nerve on the base of the skull.

It is to be remembered that the aroma of food, the bouquet of wine, etc., when appreciated in the mouth are really perceived by the olfactory sense.

## Smell.

The sense of smell is conveyed to the brain solely by the olfactory nerves. The branches of the fifth nerve distributed to the nasal mucous membrane have only to do with tactile sensation. To test the sense the patient may be made to smell various odoriferous substances, such as the essential oils, musk, camphor, valerian, &c.,[1] or to hold in the mouth such articles as cheese, wine, and liqueurs, which owe their agreeable flavour to the sense of smell (the latter test is particularly useful when the nostrils have become occluded). Hyperæsthesia of the sense of taste is occasionally seen in hysteria. Loss of smell may be due to any cause which prevents the access of the aromatic particles to the mucous membrane, such as polypus, catarrh, abnormal dryness of membrane (in paralysis of the fifth), or paralysis of the muscles necessary to the act of "sniffing" from paralysis of the seventh nerve. But apart from these causes, there is a true anæsthesia of the olfactory nerve (anosmia), which may occur in hysteria, tumour of the brain, embolism of the middle cerebral artery, blows on the head, and, as Althaus has pointed out, in locomotor ataxia.

[1] Such substances as ammonia and acetic acid should be avoided, as they stimulate the terminations of the fifth nerve.

## CHAPTER XXXVI.

## NERVOUS SYSTEM—(*continued*).

### MOTOR FUNCTIONS.

For practical purposes, the various motor functions may be arranged in the following manner:—

A. *Visceral Motor Functions.*

B. *Functions of Voluntary Muscles.*

C. *Vaso-Motor Functions.*

### A.—**Visceral Motor Functions.**

The movements of the viscera are regulated by means of so complex a nervous mechanism, and enter as yet so little within the scope of diagnosis, that they need only be very briefly alluded to here. There are, however, certain reflex actions which are of diagnostic value as indicative of the condition of that part of the spinal cord where their centres are situated.

(1.) *Deglutition* has already been spoken of under the heading of the "Alimentary system." The reflex contractions of the œsophagus are in the main under the control of a centre in the medulla, and cease when that centre is diseased—as, for example, in advanced bulbar-paralysis.

(2.) *Micturition and Defœcation.*—The mechanism of micturition is not as yet thoroughly understood, but, so far as our present knowledge goes, the following are the main points bearing on diagnosis.

There are two muscular mechanisms in opposition to each

other, the detrusor muscle (represented by the muscular wall of the bladder) by the contraction of which the viscus is emptied, and the sphincter (represented by more than one muscular arrangement) by which the outflow of urine is prevented. The centres for these lie in the lumbar region of the cord (II. to IV.), to which also pass the sensory nerves from the mucous membrane of the bladder. These centres in the cord are connected with and controlled by the centres in the cerebral cortex.

Under normal conditions, as the urine accumulates in the bladder and the pressure rises, a stimulus travels from the vesical mucous membrane to the sphincter centre, from which centre a centrifugal impulse passes to the sphincter closing the bladder tightly. When the pressure rises still further a sensory impression reaches the brain informing one that the bladder is full. If, now, the individual wishes to micturate, a stimulus is sent downwards from the cerebral cortex to the centres in the lumbar region, which causes relaxation of the sphincter and contraction of the detrusor. The bladder is thus emptied, and the flow of urine may be accelerated by the voluntary contraction of the abdominal muscles. If, on the contrary, the moment is not a suitable one for micturition, a stimulus is sent from the cortex which causes the sphincter to contract yet more powerfully and so retain the urine in the bladder.

A complex mechanism such as this may be affected by disease at various points and with correspondingly various results. If, for example, the sensory terminations in the mucous membrane of the bladder be abnormally sensitive (as in cases of cystitis), a far smaller stimulus (that is a lesser quantity of urine in the bladder), will give rise to such impressions as inform the centre and the cerebral cortex that the bladder must be emptied. Hence, in such cases, the calls to micturition are very frequent. A directly opposite result would be produced by destruction of these nerves.

Then, again, it not unfrequently happens that from some dulling of the sensorium, as, for example, in cases of coma, the sense of fulness of the bladder is not felt and the call

to micturition not perceived. The proper emptying of the bladder then depends on the integrity of the reflex centres in the cord.

When a transverse lesion in the upper part of the cord occurs, the paths between the cortex and the reflex centre are destroyed, and the patient is no longer informed when the bladder is full, nor is conscious micturition possible. In such cases it sometimes happens that the reflex centres are not affected, the bladder then filling regularly and being as regularly emptied without the patient's knowledge. But, more frequently in such cases, this perfect automatism does not take place, and we find that the bladder fills and the tension on its walls rises steadily until the sphincter can no longer control the opening. The urine then dribbles away and continues to do so, the bladder remaining full.

When, however, the centres themselves are affected, then both the detrusor and the sphincter are paralysed, and the urine dribbles out of the bladder as soon as it reaches it, the bladder therefore being at all times collapsed and nearly empty.

The act of *defœcation* is under a mechanism similar to that of micturition, with reflex centres in the same portion of the cord. What has been said of micturition under diseased conditions applies *mutatis mutandis* to defœcation.

(3.) *Sexual Functions.*—These functions are controlled by a reflex centre in the lumbar enlargement of the cord, so close to that for the cremasteric reflex that the condition of that reflex affords us a trustworthy indication of that of the sexual functions. If the control of the higher centres is cut off by disease in the upper part of the cord, the process becomes imperfect, and may sometimes be excessive (priapism). Disease of the sexual centre in the lumbar enlargement causes loss of sexual power.

(4.) *Respiration.*—The respiratory centre lies in the medulla close to and below the vaso-motor, the *nœud vital* of Fluerens. In advanced bulbar-paralysis, it is sometimes attacked, and with a necessarily fatal result.

## B.—**Motor Functions of Voluntary Muscles.**

In examining the condition of the muscular system the following points should be investigated:—

1. Nutrition of the Muscles.
2. Tonicity of the Muscles.
3. Voluntary Muscular Movement.
4. Abnormal Muscular Movements.
5. Mechanical Irritability.
6. Reflex Irritability.
7. Electrical Irritability—Electro-diagnosis.

### 1. *Nutrition of the Muscles.*

This subject will be considered subsequently under the head of the Trophic Functions.

### 2. *Tonicity of the Muscles.*

The tension of the muscles is readily appreciated by examination with the hand—a rigid muscle being hard, a flaccid one soft to the touch. When the muscles of a limb are abnormally tense, there is a degree of resistance to passive movement which is readily appreciated, and affords an important indication. The intimate connection between the tonicity of the muscles and the tendon-reflex causes alterations in the latter when rigidity exists. This subject will, however, be again referred to.

*Flaccidity* of the muscles occurs where there is muscular atrophy. It is best marked, perhaps, in spinal cases, where the anterior cornua of the cord are involved in the diseased process.

*Rigidity* of the muscles, when it assumes a well pronounced form, is termed *contracture*. Of this condition there are two forms—an active and a passive. Active contracture is found as a result of increased muscular tonus or of direct irritation of motor nerve fibres. It occurs as the result of two changes, firstly, the destruction of the fibres in the pyramidal tracts which inhibit the reflexes, and secondly, an increased irritability of these reflex centres. Active contracture is therefore an important sign of secondary descending degeneration of the pyramidal

*See page* 376.

EXPLANATION OF FIG 100.

1. Frontalis muscles.
2. Attrahens and attollens auriculam muscles.
3. Retrahens and attollens auriculam muscles.
4. Occipitalis muscle.
5. Facial nerve.
6. Posterior auricular branch of facial nerve.
7. Stylohyoid muscle.
8. Diagastric muscle.
9. Buccal branch of facial nerve.
10. Splenius capitis muscle.
11. Subcutaneous branches of inferior maxillary nerve.
12. External branch of spinal accessory nerve.
13. Sterno-mastoid muscle.
14. Cucullaris muscle.
15. Sterno-mastoid muscle.
16. Levator auguli scapulæ muscle.
17. Posterior thoracic nerve (Rhomboidei muscles).
18. Phrenic nerve.
19. Omohyoid muscle.
20. Lateral thoracic nerve (Serratus magnus).
21. Axillary nerve.
22. Branch of brachial plexus (musculo-cutaneous and part of median).
23. Anterior thoracic nerve (Pectoral muscles).
24. Corrugator supercilii muscles.
25. Compressor nasi and pyramidalis nasi muscles.
26. Orbicularis palpebrarum muscle.
27. Levator labii superioris alæque nasi muscle.
28. Levator labii superioris muscle.
29. Zygomaticus minor muscle.
30. Dilatator naris.
31. Zygomaticus major.[1]
32. Orbicularis oris.
33. Branch to triangularis and levator menti muscles.
34. Levator menti muscle.
35. Quadratus menti muscle.
36. Triengularis menti muscles.
37. Cervical branch of facial nerve.
38. Branch to platysma muscle.
39. Sterno-hyoid muscle.
40. Omohyoid muscle.
41. Sternothyroid muscle.
42. Sternohyoid muscle.

[1] The upper of the two lines that converge on 31 should have been directed to 30 as it applies to the Dilatator naris posterior muscle.

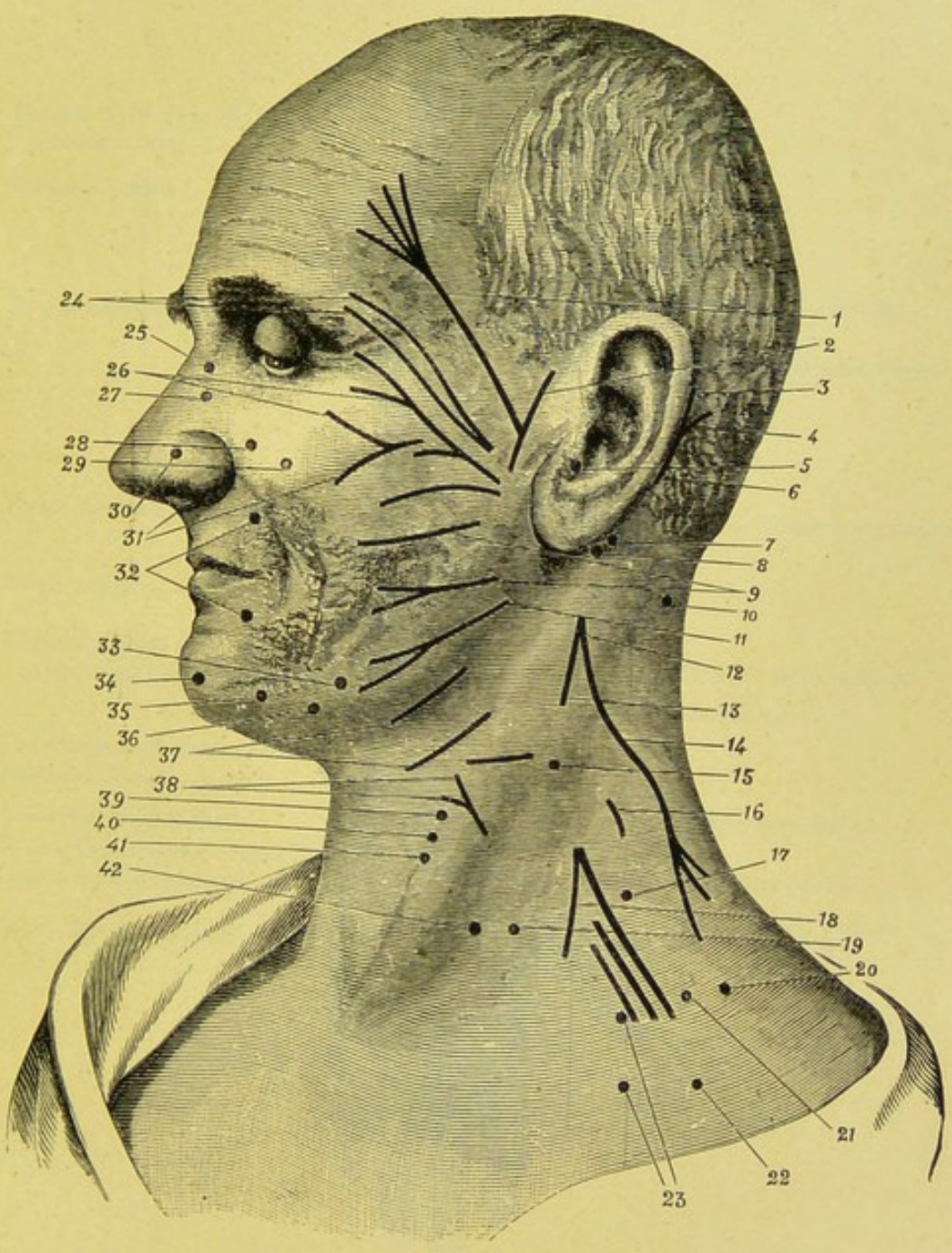



FIG. 100.—Motor Points of Head and Neck. (Ziemssen.)

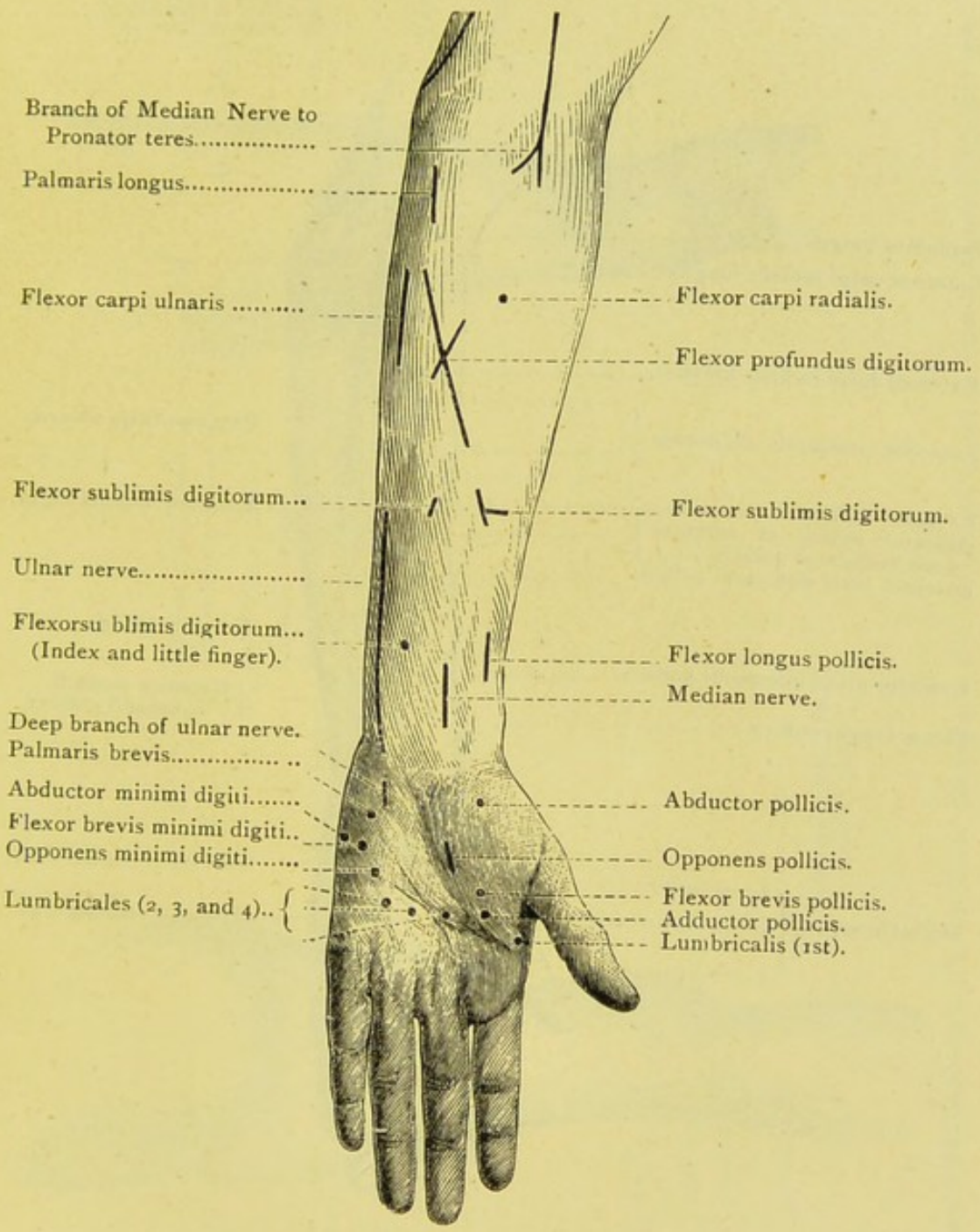



FIG. 101.—Motor Points on Flexor Surface of Forearm. (Ziemssen.)

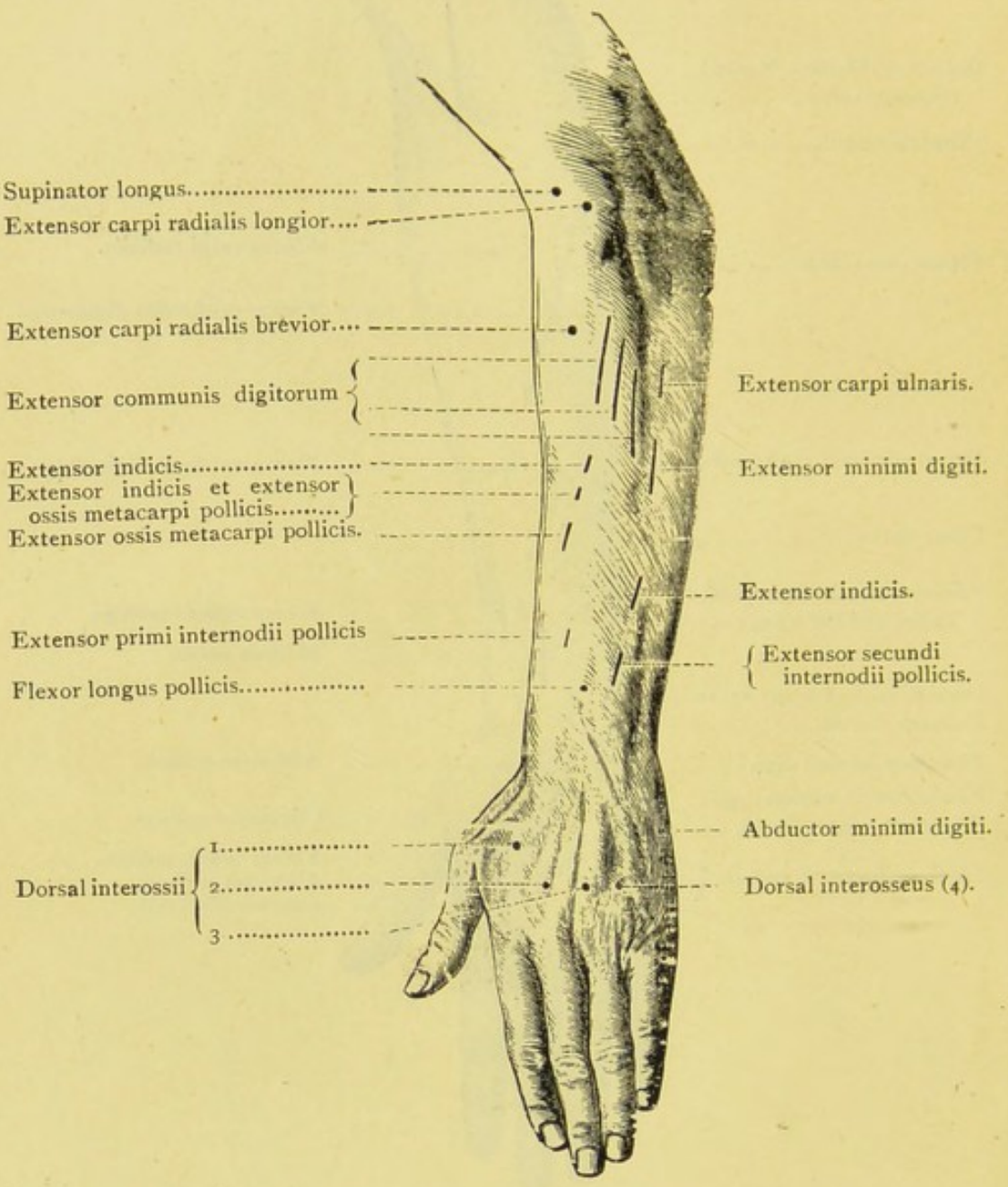



FIG. 102.—Motor Points on Extensor Surface of the Forearm. (Ziemssen.)

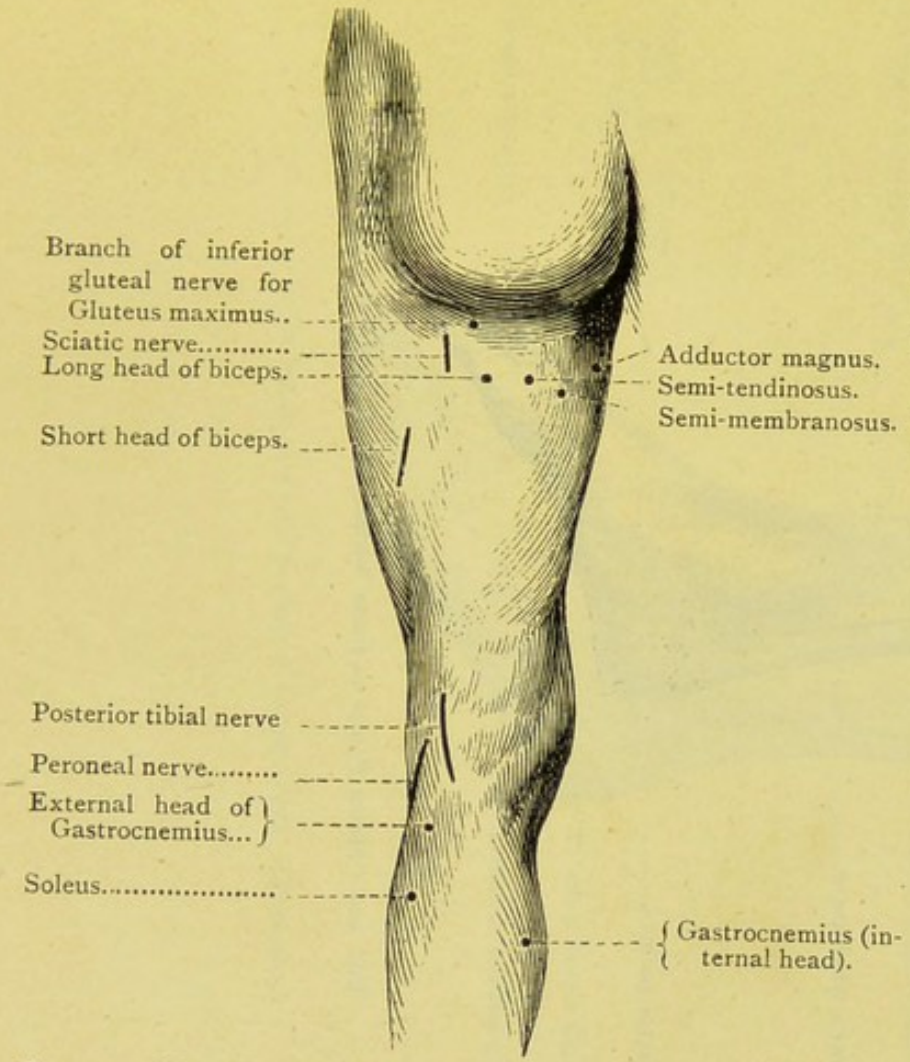



FIG. 103.—Motor Points on Posterior Aspect of Lower Limb. (Ziemssen.)

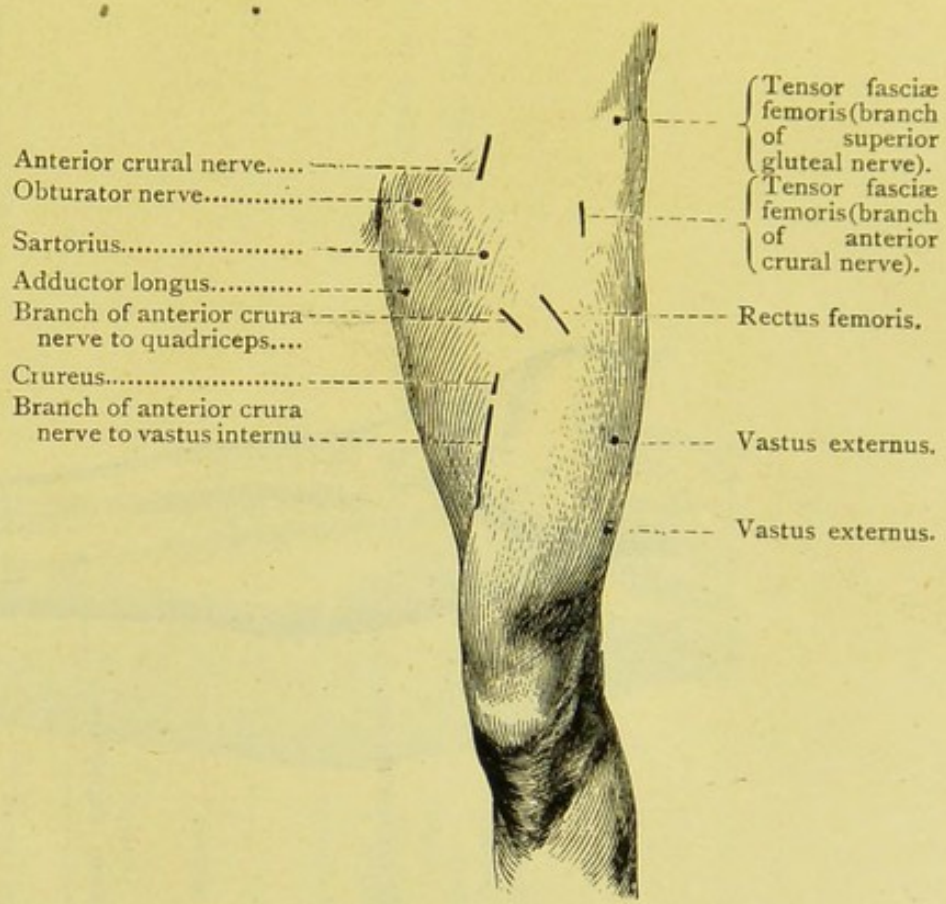



FIG. 104.—Motor Points on Anterior Aspect of Inferior Limb. (Ziemssen.)

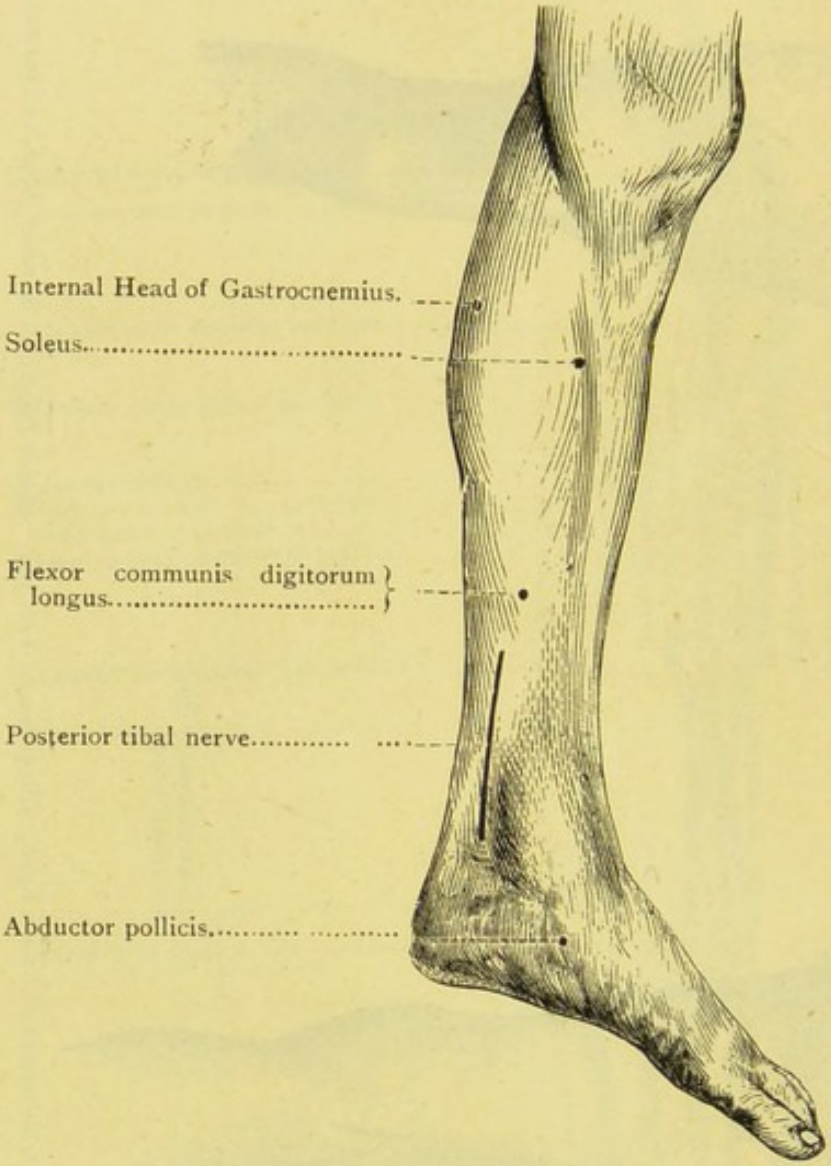



FIG. 105.—Motor Points on Inner Surface of Inferior Limb. (Ziemssen.)

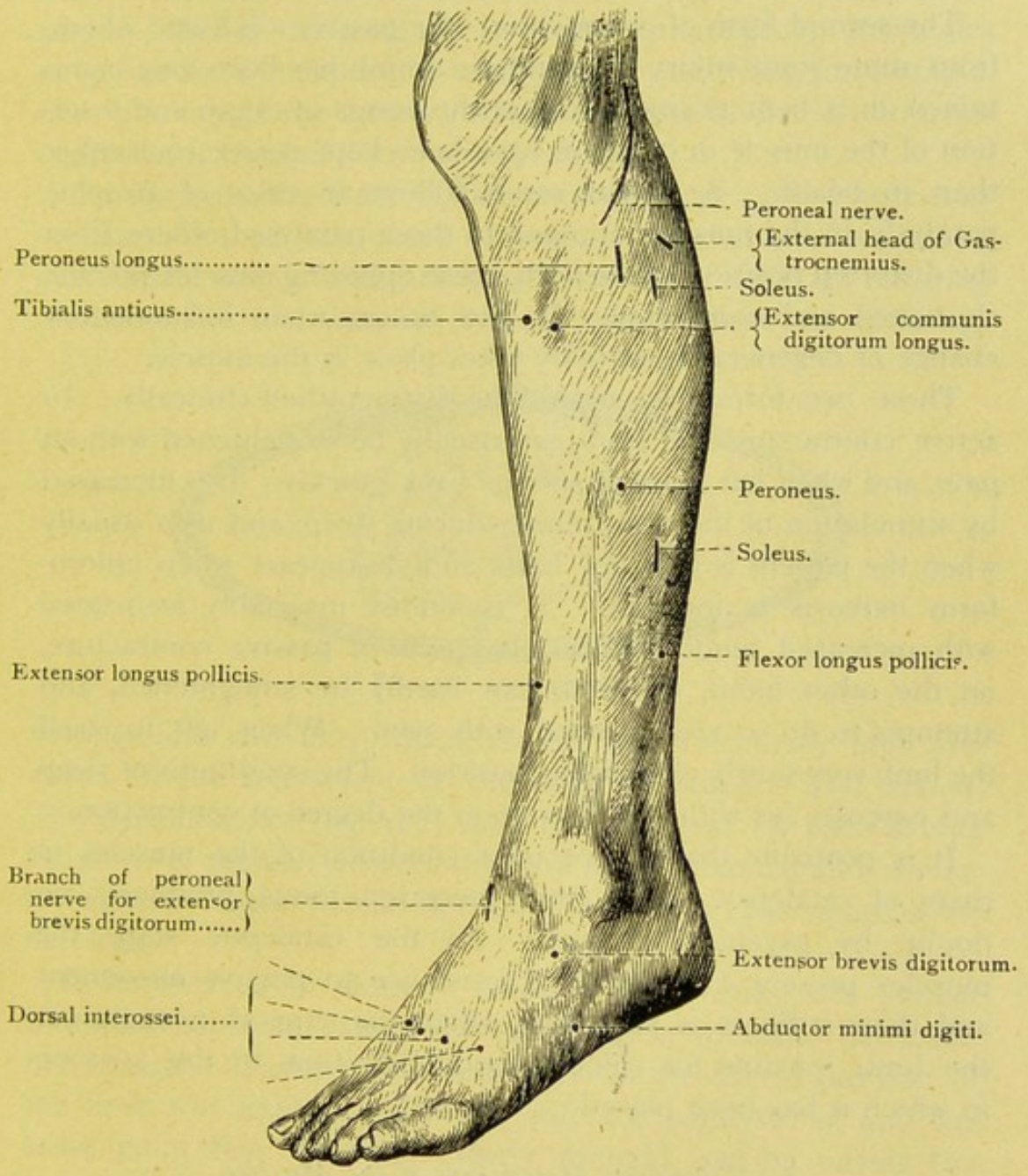



FIG. 106.—Motor Points on Outer Surface of Lower Limb. (Ziemssen.)

tracts, the result of cerebral disease or of transverse lesion of the cord higher up. It also occurs in cases of primary systemic disease of these tracts, in amyotrophic lateral sclerosis, and likewise in hysteria.

The second form of contracture—the passive—is found where, from some joint injury for example, a limb has been long maintained in a bent position, so that the points of origin and insertion of the muscle in question have been kept nearer each other than in health. A similar result follows in cases of atrophic paralysis, in the muscles opposed to those paralysed, where, from the distorted position of the limb, these opposing muscles become shortened and contracted. It also occurs when inflammatory change or degenerative atrophy takes place in the muscle.

These two forms can usually be distinguished clinically. In active contracture the limb can usually be straightened without pain, and when left alone it springs back quickly. It is increased by stimulation of the skin, relaxes during sleep, and also usually when the patient is in a hot bath, and disappears when chloroform narcosis is induced. It is almost invariably associated with increased tendon-reflex. In cases of passive contracture, on the other hand, the limb can hardly be straightened, and attempts to do so are attended with pain. When left to itself the limb very slowly recovers its position. The conditions of sleep and narcosis are without effect upon the degree of contraction.

It is probable that the peculiar condition of the muscles in cases of catalepsy results from increased tonus, produced, no doubt, by psychical changes. In the cataleptic state the muscles present a curious dull resistance to passive movement and their stiffness is such that, when the support is removed, the limb remains for some considerable time in the position in which it has been placed.

### 3. *Voluntary Movements of the Muscles.*

When a muscle or group of muscles fails to respond, by shortening in the normal way, to a normal stimulus—we speak of *paralysis* (akinesis)—the lesser grade of which is called *paresis* (hypokinesis).

*Paralysis* varies much in regard to its extent and distribution. It may be limited to one or two muscles, or it may affect all the muscles of one lateral half of the body (hemiplegia), or of both sides of the body symmetrically, usually both lower limbs (paraplegia). When the paralysis is limited to one group of muscles or to one limb, we speak of monoplegia; and when, for example, the right side of the face is paralysed along with the left arm, the condition is termed *crossed paralysis.*

To examine the state of the muscular system, it is necessary to cause the patient to go through all varieties of voluntary movement, simple and combined, standing, walking, stepping up upon a chair, &c., as well as such actions as speaking,

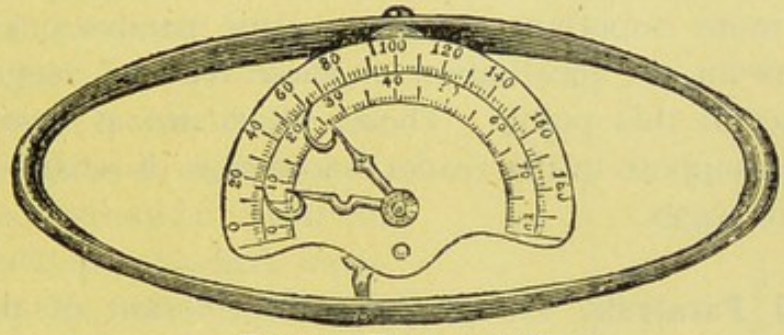


FIG. 107.—Dynamometer.

writing, and the like, which require great accuracy and precision in the movement of the muscles brought into action. The dynamometer (fig. 107) may be employed for ascertaining the force of the muscular contraction in the hands, and similar instruments have been devised for testing the muscles of the legs.

If, however, as in cases of cerebral hæmorrhage, the patient is unconscious, he cannot be called upon to make particular movements. Under these circumstances the flaccid position of the limb will indicate on which side the paralysis is, and the same point may be more clearly brought out by raising the two arms and noting which falls most completely and rapidly when released. In the case of the face, the absence of wrinkles on the paralysed side will at once attract attention, as well as the position of the mouth which will be drawn towards the sound side.

Paralysis is of two varieties,—organic and functional. The former is the result of some interruption in the motor tract. The latter is caused by some abnormality in the portions of the cerebral centres which subserve the mental functions, leading to mental conditions which inhibit the will. Functional paralysis is usually distinguishable without much difficulty by the facts that there are no symptoms pointing to organic disease, no muscular atrophy, no indications of trophic changes in skin or bone, no alterations of electrical reactions (*vide infra*), no ophthalmoscopic changes, and no diminution of the reflexes, which are indeed often increased. There are usually present other indications of hysteria, such as ovarian tenderness, hemianæsthesia, convulsions, and psychical changes.

Much more important are the organic paralyses, and some indications for the localisation of these may be useful to the practitioner at this point. These, which are at best only in outline, presuppose in the reader knowledge of what follows in subsequent pages.

**Central Paralysis**, *i.e.* paralysis from lesion of the upper neuron, which, starting from the cells of the motor cortical area and their arboresations, passes to the nuclei of the cerebral nerves and to the anterior horns of the spinal cord. The relations of the upper and lower motor-neurons are shewn in fig. 108. Speaking generally, in lesions of this tract, the paralysis is more or less general in distribution, involving all the muscles of the limb affected. These muscles are in a state of increased tonus, in a spastic condition, and there is rarely any atrophy. The deep reflexes are either normal or exaggerated, the superficial reflexes are usually present, rarely absent, and there is no qualitative change in the electrical reactions—*i.e.* no reaction of degeneration.

Lesions of the cortical motor region give rise to various monoplegias, according to the portion of the cortex affected, involvement of the lower central convolutions leading to facio-lingual monoplegia, of the middle convolutions to brachial monoplegia, and of the convolutions of the para-central lobule to

crural monoplegia. These are usually accompanied by indications of irritation, such as epileptiform attacks, chorea, ataxia, athetosis, and by anæsthesia.

When the lesion affects the pyramidal tracts in the posterior part of the internal capsule there is complete hemiplegia, often accompanied by hemianæsthesia. Affections of the corpora striata and optic thalami may indirectly occasion hemiplegia. Lesion of the crus causes hemiplegia, to which a crossed oculomotor paralysis is often added. That of the anterior portion of the pons gives rise to hemiplegia with crossed paralysis of the seventh, fifth and sixth nerves. When the pyramidal tracts in the medulla are attacked, hemiplegia results, which, if both sides are affected, may be double, all four limbs being paralysed, and, in addition there is usually crossed paralysis of the hypo-glossal and glosso-pharyngeal nerves, and interference with the functions of the vagus, the result being that there is paralysis of the tongue, the soft palate, the upper third of the œsophagus, interference with the heart and the respiration, and sometimes the urine is found to contain sugar and albumin. When one-half of the cord is affected we have what is called Brown-Séquard's paralysis—*i.e.* paralysis of the muscles on the affected side of the body and anæsthesia on the opposite side. This is

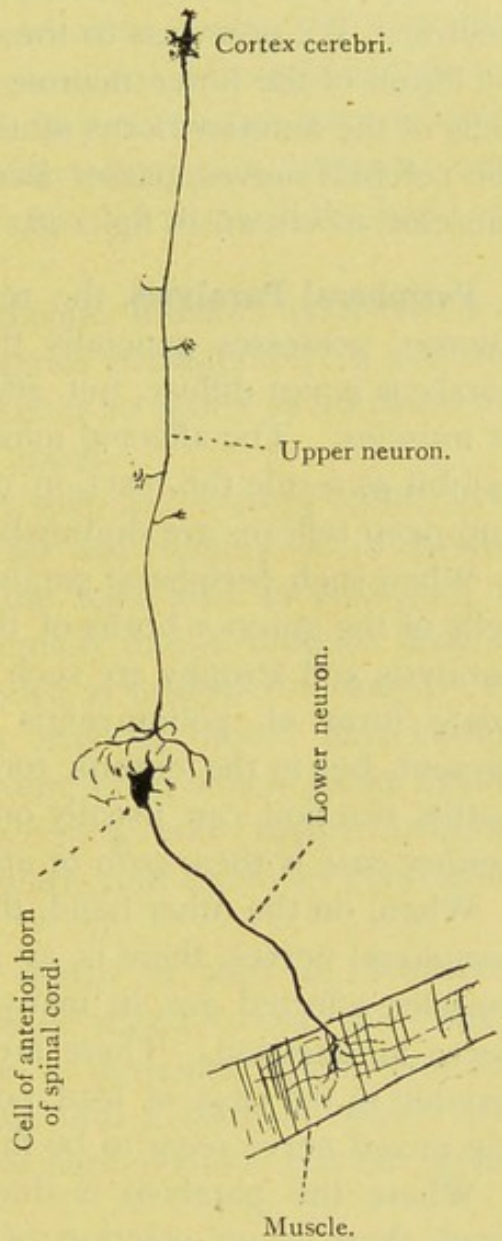



FIG. 108.—Diagram of upper and lower neuron (after Jakob).

accounted for by the fact that the sensory fibres when they enter the cord cross at once to the opposite side, while the motor fibres decussate in the medulla. When the pyramidal tracts in the cord are alone affected we have a pure spastic paralysis.

Such, very briefly stated, are the paralyses of the upper neuron. We now pass to the consideration of those occasioned by lesion of the lower neuron, which, starting from the ganglion cells of the anterior horns of the cord or of the motor nuclei of the cerebral nerves, passes along the motor nerves to end in the muscles, as shewn in fig. 108.

**Peripheral Paralysis**, the result of some lesion of this lower neuron, possesses generally the following characteristics. The paralysis is not diffuse, but affects individual muscles or groups of muscles. The affected muscles undergo a rapid atrophy, and exhibit as a rule the reaction of degeneration. Both superficial and deep reflexes are diminished or lost.

When such peripheral paralysis is due to lesion of the ganglion cells of the anterior horns of the cord, the muscles affected with paralysis and atrophy are such as are allied in function. In the acute form of poliomyelitis the reaction of degeneration is present, but in the chronic form—progressive muscular atrophy —this reaction can usually only be elicited with difficulty. In neither case is there pain or any other sensory disturbance.

When, on the other hand, the paralysis is due to lesion of the peripheral nerves, there is, as a rule, considerable pain, and the muscles affected are, in many cases, all those supplied by the nerve in question. There are, however, exceptions to this rule, notably in the case of lead poisoning, where the motor fibres of the mixed nerve seem to be first attacked.

Where the paralysis is due to lesion of the muscular fibre itself there is no reaction of degeneration, and there are no sensory symptoms nor fibrillary twitchings.

### 4. *Abnormal Muscular Movements.*

Under this heading is included the whole group of spasms or convulsive movements. The more important of these are as follows :—

(*a.*) *Clonic and Tonic Spasm.*—Spasm (hyperkinesis) of the voluntary muscles, may be defined as abnormal muscular contraction, the result either of pathological irritation, or of a physiological stimulus to which the resulting contraction is disproportionate. It is of two varieties—tonic and clonic—the former indicating a condition of muscular contraction (tetanus) which remains of nearly equal intensity for a lengthened period (minutes, hours, or days), while under the latter term (clonic) is understood a condition of rapidly alternating muscular contraction and relaxation, whereby particular parts of the body are set in motion.

Clonic spasm occurs during epileptic, uræmic, hysterical, and other forms of convulsion, and varies considerably in severity, sometimes being slight, at other times so severe as to cause the whole body to be violently tossed about.

Tonic spasm is most commonly seen as "cramp," continuous and painful contraction of muscles individually or in groups, especially those of the calf of the leg; also in catalepsy, in hysterical contracture, and occasionally in the muscles used in certain co-ordinated movements, such as in *writer's cramp*.

In connection with spasm, it may be noted that certain points are often to be found, pressure upon which either excites or arrests the spasm (motor exciting and motor arresting pressure points). This is particularly noticeable in connection with facial spasm.

A peculiar variety of tonic spasm is seen in *Thomsen's Disease*, the characteristic feature of which is that the patient is unable immediately to make a muscular movement, the muscles in question being thrown into a state of slight tonic spasm. Once, however, the movement commences, the spasm disappears.

The causes and mechanism of convulsions are, in many cases, very obscure. They may be produced by any lesion of the motor tract (in which case the spasm is spoken of as *direct*), or of the sensory tract or its end-organs (reflex spasm). Speaking generally, it may be said that in the former case the spasm is usually tonic, in the latter, clonic in character. Of these the

reflex causes are the most common, and they are of very various kinds. In susceptible children, the irritation caused by dentition, or by the presence of intestinal parasites, is often sufficient to cause convulsive attacks. Other sources of peripheral irritation may occur in connection with almost any organ of the body. Similarly, though much more rarely, irritation of a sensory nerve in its course, as by a cicatrix or by the pressure of a tumour, may lead to a like result. It does not appear that organic disease of the spinal cord gives rise to convulsions, but certainly tonic spasms of great severity have a spinal origin in the case of poisoning with strychnine and with the tetanus toxines. Pressure on motor nerves may cause irritation sufficient to produce tonic spasm in the muscles supplied. The convulsions so frequently seen in hysteria are probably sometimes of central origin, but are certainly, in many cases, due to peripheral irritation. Epilepsy, on the other hand, though sometimes peripheral, is probably more frequently due to central causes. Certainly this is the case as regards partial or Jacksonian Epilepsy where the convulsion is limited to some particular part of the body, and in which the lesion is one affecting the corresponding portion of the motor cerebral cortex.

(*b.*) *Tremor.*—Muscular tremor is often seen in health under conditions of great excitement, or where exhaustion has been produced by considerable muscular exertion or by lack of food.

When it occurs in disease it is probably due to an increased sensitiveness of the nervous system, such that tremor follows a stimulus which would have been insufficient in health to occasion it. This state of morbid sensitiveness is well seen in cases of hysteria and of neurasthenia, and in persons weakened by sexual excesses, or by recent severe illness.

In investigating cases of tremor note should be made of the particular parts of the body affected, of the rapidity, rhythm and extent of the movements, of the causes which appear to excite, to aggravate or to restrain the tremor, and in particular, whether the tremor takes place only or chiefly during voluntary movement. In special cases it may be well to record the movements by means of a graphic method.

Tremor is conspicuously seen in cases of multiple sclerosis (where it only occurs during voluntary movement), and of paralysis agitans, and it occurs as the result of poisoning by alcohol, mercury, tobacco, &c., and in persons under treatment for the morphia habit.

(*c.*) *Fibrillary Twitching.*—A sign of degenerative muscular atrophy, fibrillary twitching differs essentially from tremor. It consists in rapid contractions of the individual bundles of muscular fibres, the muscle not contracting as a whole. These fine twitchings cause sufficient motion in the overlying skin to render them visible on inspection. They may usually be increased by tapping the muscle or by cooling the skin by blowing on it. They are chiefly seen in the muscles of the hand and of the face, and are most marked in cases of progressive muscular atrophy.

(*d.*) *Choreic movements.*—The movements which are characteristic of chorea are well marked. They consist in sudden and irregular contractions of the muscles of the face and limbs which prevent the patient from resting when awake, and if severe may render sleep impossible. The brow wrinkles, the tongue is darted out, the mouth is pulled to one side or the other, the head is twisted suddenly, the shoulders shrugged, hands and feet thrown about irregularly. These movements are quite objectless. They are most usually seen in cases of chorea (St Vitus' Dance) in childhood, or in its chronic adult form, and occasionally in hysteria. In connection with hemiplegia, either before or more commonly after the attack, choreic movements may be observed.

(*e.*) *Athetosis.*—This rare disorder is characterised by slow regular movements of the hands and feet. The fingers are slowly stretched out one after another and then slowly flexed, the hands twisted, the toes extended and flexed. Sometimes similar movements are observed in connection with the head. Athetosis is often unilateral. It sometimes occurs as a congenital affection, and may be associated with epilepsy, or with hemiplegia.

Besides these abnormal forms of muscular movement there

are one or two others of greater rarity, such as the *co-ordinated spasms* (hysterical laughter, saltatory spasm, &c.), and the *associated movements* in which when, for example, one hand is moved voluntarily, the other hand or the feet pass into involuntary motion.

(*f.*) *Ataxic movements.*—These will be subsequently referred to.

### 5. *Mechanical Irritability.*

Over healthy muscles a blow of some force causes a local contraction of sufficient volume to show a swelling under the skin. In various diseased conditions (particularly phthisis) this irritability is so much increased that the slightest tap is followed by such contraction. This is of little or no diagnostic value, as it only indicates muscular exhaustion. The same phenomenon can be observed during the first hours after death.

Tapping over a motor nerve where it runs superficially may also give rise to sudden contraction in the muscles supplied by that nerve. This irritability is often much increased. Of this nature is *Chvostek's symptom*, which is so frequently met with in cases of tetany. It is elicited by tapping with the finger, or, better, with a percussion hammer, over the facial nerve, which at once causes contraction of the muscles which it supplies. A tap of this kind, made just under the zygomatic process calls forth, in cases of tetany, a sudden contraction of the lips and alæ nasi. Tapping over the frontal branch may, in like manner, give rise to contraction of the frontal muscle. While most frequently met with in tetany, Chvostek's symptom is also occasionally seen in other conditions, particularly in cases of early phthisis.

### 6. *Reflex irritability of Muscles.*

One of the most valuable diagnostic signs we possess in connection with the nervous system consists in the reflex movements of the muscles. These reflex movements may be excited by stimulation of the skin or mucous membrane (superficial reflexes), or by that of the tendons, faciæ, or periosteum (deep reflexes), or finally, may consist in the changes of the pupil caused by light, &c.

The value to the physician of these phenomena consists in the fact that their presence or absence gives important indications regarding the integrity of the reflex loop by means of which each individual movement is brought about, and in particular the state of the spinal cord at that level.

These reflexes, which are very numerous, fall into three groups :[1]—

1. *Complicated Reflexes, with special centres.* — This group includes sneezing on tickling the nasal mucous membrane, vomiting on tickling the fauces, swallowing movements on touching the back of the tongue, coughing on touching the opening of the larynx, and certain reflexes connected with the bladder and rectum, which have been discussed already.

These reflexes possess certain peculiarities, such as that there is a peculiar sensation connected with the action of the stimulus, that the stimulus must be very prolonged as compared with that of other forms of reflex, that the latent period is of considerable duration, and that the resulting movement is a very complicated one.

2. *The Superficial or Skin Reflexes.*—These are very numerous. They include the closure of the eyelids when an object is brought suddenly towards the eye, the various pupil reflexes (which have been already spoken of), and the contraction of the soft palate on irritation of the fauces. Of the others, those which are most commonly made use of in diagnosis are—

(1.) *Plantar Reflex.*—Tickling the skin of the sole gives rise to contraction of the muscles moving the toes, and, if the stimulus be continued, of those of the foot and leg : the centre lies in the lower part of the lumbar enlargement.

(2.) *Gluteal Reflex.* — Tickling the skin of the buttock determines in many persons a contraction of the gluteal muscles : the centre lies probably at the level of the 4th or 5th lumbar nerves.

(3.) *Cremasteric Reflex.*—Tickling of the skin on the inner aspect of the thigh is followed by drawing up of the testicle : the centre is at the level of the 1st and 2nd lumbar nerves.

[1] Jendràssik, *Deutsches Archiv.*, Bd. 52 (1894).

When the skin of the scrotum is stroked, or, better, touched with ice it shrivels from contraction of the smooth muscular fibres. This is known as the scrotal reflex.

(4.) *Abdominal Reflex.*—On stroking the skin of the abdomen, from the costal margins towards the iliac crests, the abdominal muscles contract: the centres lie between the 8th and the 12th dorsal nerves.

(5.) *Epigastric Reflex.*—Tickling or stroking the skin of the chest over the 4th, 5th, and 6th intercostal spaces, causes a dimpling of the epigastrium on the corresponding side: centres from the level of the 4th to the 6th or 7th dorsal nerves. The same region of the cord contains centres for the reflex contraction of the erectores spinæ, which occurs when the skin is stroked from the angle of the scapula down to the iliac crest.

(6.) *Scapular Reflex.*—Tickling of the skin in the inter-scapular region gives rise to contraction of the scapular muscles: the centre lies at the level of the lower two or three cervical, and the upper two or three dorsal nerves.

*Diagnostic value of the Superficial Reflexes.*—These reflexes are increased—(1) where the cerebral restraining influences are removed, as sometimes happens in cases of central paralysis and transverse myelitis; and (2) where the grey substance of the nerve centres is unusually excitable, as in cases of strychnine poisoning, tetanus, &c. Diminution or absence of the superficial reflexes results either from interference with the integrity of the reflex loop in question (disease of nerves, spinal nerve roots, white or grey substance of the cord) or from increase in the cerebral inhibitory influence. This latter condition arises in cases of cerebral mischief where there is irritation, and is often of value in distinguishing organic from purely functional hemiplegia.

3. *Deep Reflexes.*—These are produced by tapping over, or quickly rendering tense, tendons or fasciæ.[1] They consist in the rapid contraction of the muscle which is thus put on the stretch. The chief of these are—

(1.) *The Knee Jerk.*—If the knee be flexed, and the leg be allowed to hang down loosely, a tap over the patellar tendon

[1] Also by tapping over periosteum.

will give rise to a contraction of the quadriceps femoris and a consequent jerk forwards of the leg. If the contraction be doubtfully present it may be strengthened by directing the patient to interlace the fingers of each hand in the other and then to pull the hands strongly as if to separate them. Although this reflex is sometimes difficult to elicit it is very rarely altogether absent in healthy persons.

Similarly, an ankle jerk may be obtained by tapping the Achilles tendon when the foot is held at a right angle. In the arm a triceps reflex sometimes follows a tap on the tendon of that muscle just over the olecranon, and occasionally a similar result may follow a tap on the biceps tendon at the elbow. Other reflexes of this kind are met with, for example, in connection with the masseter muscles—but by far the most useful reflex from a diagnostic point of view is the knee jerk.

(2.) *Ankle Clonus.*—In certain nervous conditions, involving increase and muscle tonus, a sudden pressure upon the sole of the foot, which stretches the muscles of the calf, gives rise to a series of spasmodic contractions of these muscles which recur with great regularity, usually about 6 or 7 times per second. James has shewn that the rate varies with the height of the individual, and the consequent length of the motor nerve, a beautiful proof of the essential reflex character of the clonus. The clonus can also sometimes be excited by a tap on the muscles on the front of the leg. Similar phenomena may be observed in connection with the knee. Comparing the ankle jerk, the knee jerk, the wrist and elbow jerks, James has pointed out that the rapidity of their production is governed by the distance which lies between the individual muscle and its centre, that is by the length of its motor nerve, another proof, in addition to that just mentioned, of the reflex character of these jerks.

*Diagnostic value of the Deep Reflexes.*—The deep reflexes are increased—(1) in cases where there is an increased excitability of the grey matter of the cord, as in strychnine poisoning and in tetanus. Probably to this cause may be ascribed the increase observed in cases of phthisis, and occasionally in multiple

neuritis. Possibly also this may explain the fact that the knee jerk is sometimes unusually active in cases of disease of the ankle joint. The deep reflexes are increased (2) where the inhibitory cerebral influence is withdrawn. Hysterical paralysis acts in this way, as may also central organic paralysis, and, very specially, disease of the pyramidal tracts.

Abolition of the deep reflexes occurs when the reflex loop is interrupted. Any lesion, therefore, which affects the sensory nerves, the posterior roots, the postero-external columns, the grey matter, the anterior roots, or the motor nerves, will produce this effect. These reflexes are thus lost in such diseases as neuritis, poliomyelitis anterior acuta, progressive muscular atrophy, and disseminated sclerosis. Practically, however, the most important point is that the knee reflex disappears in locomotor ataxia, and that at an early stage of the disease.

### 7. *Electro-diagnosis.*

*Electric Currents* are of the utmost use in diagnosis, but the limits of this work prevent the description of the various forms of apparatus—batteries, electrodes, galvanometers, &c. For such information the reader is referred to special works on the subject. It will be sufficient here to indicate very briefly the inferences to be drawn from the information so obtained.

In using electric currents it is important to limit the effects as far as possible to individual muscles or nerves, going from one to another and comparing the results obtained. In diagnosis the polar method should always be employed, which consists in placing the pole of the battery, the action of which it is wished to determine, at the point to be stimulated, while the other electrode is placed at some distant part of the body, usually the sternum. Both electrodes, as well as the skin to which they are applied, should be thoroughly moistened. Proceeding in this way we may stimulate either the trunk of a motor nerve, which will cause contraction of all the muscles it supplies, or we may stimulate the muscle itself. In acting on the muscle the electrode is usually applied over the *motor point—i.e.*, the point at which the motor nerve branch enters the

muscle, when the muscle as a whole will contract. In order then to be able to make an intelligent use of electric currents in diagnosis it is necessary to know where the motor nerves lie sufficiently superficially to be affected by the current, as well as the position of the motor points of the muscles over the body generally. In figs. 100 to 106, which are derived from von Ziemssen's work, the more important of these are given.

The degree of resistance offered by the skin to the passage of electric currents varies. In cases of abnormally high resistance the muscular response is weakened, and therefore, to prevent error, it is needful to determine the amount of resistance, or in other words, the amount of the current which penetrates the tissues. This is done by means of a galvanometer introduced into the circuit.

The *Faradic* or *Induced Current* excites muscular contraction when the stimulus is applied to the motor nerve in its course, or over the muscle itself. The contraction so induced varies from a scarcely perceptible change up to tetanus, according to the strength of current used.

Having placed the electrodes, the current should be turned on, at first with the primary and secondary coils at a considerable distance apart, and the secondary coil should then be moved slowly towards the primary until a point is reached when there is a visible and distinct, though slight, response in the muscle. The distance between the coils should then be noted. If the lesion is unilateral it is easy by comparing the healthy side to ascertain if the muscle is responding to a normal current. If it is bilateral it is more difficult to determine this point. But even then it is usually possible to say whether the electrical irritability in either arm or leg is normal by bearing in mind, what Erb has pointed out, that in stimulation of the ulnar nerve slightly above the internal condyle of the humerus, and of the peroneal nerve over the head of the fibula, the muscles in question respond to the same strength of minimal current, or at any rate that the difference of the distance of the coils in the two cases does not exceed 10-15 m.m.

The *Galvanic* or *Continuous Current* only gives rise to con-

traction when the current is opened or closed, not when it is passing. The reaction of each pole should be separately investigated, the other being placed upon the sternum.

As with the Faradic, so now with the galvanic current, we proceed to ascertain the strength of current required to cause muscular contraction. It is sufficient for this purpose to employ the cathode, placing the anode over the sternum. The cathode is then held pressed over the nerve to be stimulated, and the current is gradually increased, the key being opened and closed with each increase, until a point is reached when on closing the circuit the muscle contracts. The galvanometer is then at once read. This reading gives the minimal current which will cause contraction at the cathode on closing the current, and consequently the quantitative reaction.[1] Whether this figure is too high or too low, is to be decided in a manner similar to that used in connection with the faradic current.

But while the cathode is the pole made use of for determining the quantitative reaction, the action of the positive pole or anode must also be investigated; and when this is done it will be found that in health considerable differences exist in the action of the two poles. The reactions obtained at either pole, for weak, medium and strong currents, are embodied in the following law:—

The *Law of Normal Contraction* for motor nerves is as follows:—

*Weak Currents*—

Positive pole (anode)—No contraction.

Negative pole (cathode)—Contraction when the current is closed, expressed by the formula C.C.C. (cathodal closing contraction); none when it is opened.

*Currents of Medium Strength*—

Positive pole—Slight contraction both on opening and on closing the current, expressed by the formulæ A.O.c. and A.C.c., anodal opening contraction and anodal

[1] Another figure is sometimes also used for this purpose, the current namely which is sufficient to cause tetanus at the cathode on closing the current.

closing contraction,—the small c. indicating that the contraction is slight.

Negative pole—Strong contraction on closing the current, expressed by the formula C.C.C′., cathodal closing contraction,—the accent on the last C′ indicating that the contraction is strong.

*Strong Currents*—

Positive pole—Contraction both on opening and on closing the current expressed by the formulæ A.O.C., A.C.C.

Negative pole—Tetanus when the current is closed, slight contraction when it is opened, expressed by the formulæ C.C.T., cathodal closing tetanus, and C.O.c., cathodal opening slight contraction.

Various forms of paralysis may be accurately classified, as Erb has shewn, by means of the electrical reactions of the muscles and nerves, as follows :—

*a. No Change in the Electric Excitability* with either form of current (cerebral paralysis before secondary degeneration occurs, and paralysis from disease of the white substance of the cord).

*b. Quantitative Change in the Electric Excitability*—

(*α.*) *Simple Increase.*—This condition is uncommon, but is sometimes found in the first stage of cerebral hemiplegia, in hemichorea, in certain forms of locomotor ataxia, and rarely and transitorily in peripheral paralysis at its commencement. It is characteristic of Tetany.

(*β.*) *Simple Diminution* occurs in all diseases which lead to simple muscular atrophy, such as hereditary muscular atrophy and the like. It is met with in pseudo-hypertrophic paralysis, progressive ascending paralysis, multiple sclerosis, myelitis, &c. It also occurs in muscles which have been paralysed from inaction.

*c. Quantitative and Qualitative Changes* ("Reaction of Degeneration,"—*Erb*).—These changes are much more important than the merely quantitative. The reactions of nerve and muscle are different, and must be separately stated as follows :—

*Nerves.*

Two or three days after the paralysis has begun, the excitability of the nerves to both currents diminishes, and gradually becomes lost. Should recovery take place the excitability reappears, but commonly it is later of being regained than voluntary motion.

*Muscles.*

To the Faradic current they behave much as the nerves do —the excitability being gradually lost, and as gradually regained on recovery taking place.

The galvanic excitability falls parallel with the Faradic for about a week, but in the course of the second week it begins to rise, until a point is reached when the muscles contract with stimuli which would have no apparent effect upon them in their normal condition. The muscular contraction thus induced is, however, slow and delayed instead of instantaneous as in health. A qualitative change has also taken place, the positive closing contraction increasing until it equals or surpasses in intensity the negative closing contraction, while the negative opening contraction becomes equal to or exceeds the positive. By comparing the normal law of contraction it will be seen that in this form of paralysis the conditions are exactly reversed. After a time this galvanic excitability of the muscles diminishes, and in incurable cases disappears; but when recovery takes place, the normal state of matters becomes gradually restored. In the accompanying diagrams, constructed by Erb, the electrical reactions in two of these forms of paralysis are graphically indicated. The two selected for illustration shew the differences between rapid and slow recovery.

The clinical significance of this "reaction of degeneration" is that it occurs in all atrophic paralysis depending on disease of the anterior horns of the cord or of the corresponding brain nuclei, of the anterior roots, of the nerves—in fact of any part of the peripheral neuron. It is not met with in paralysis of the upper neuron. It is typical, then, of all forms of peripheral paralysis—traumatic, rheumatic, diphtheritic, or due to neuritis in any form. Finally, it is important to bear in mind that the

reaction of degeneration is not met with in cases of paralysis from cerebral causes, in hysterical paralysis, or in paralysis due to muscular affections.[1]

It may be useful to give here a brief *resumé* of the electrical reactions in special forms of paralysis—cerebral, spinal, and peripheral.

(1.) *Paralysis from cerebral disease.*—When paralysis results from disease of brain, the electrical reactions are normal in most cases. There is sometimes, however, a slight *quantitative* increase of response which may be due either to irritation of the centres, or to removal of the cerebral inhibitory influence, and at other times a slight *diminution* of response when the muscles have become wasted from disease.

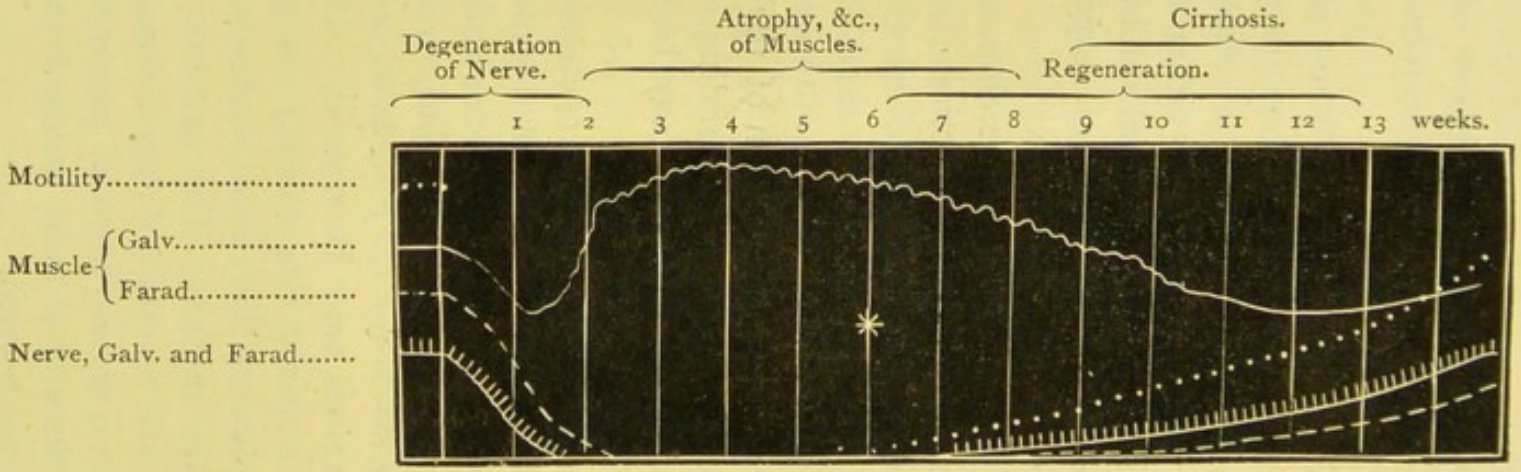
(2.) *Paralysis from disease of the white substance of the cord.*—As a rule in such cases the electrical reactions are normal. If, however, the upper part of the cord be so diseased as to cut off the inhibition of the brain, and at the same time the lower part remain healthy, there may be quantitative *increase*, and the same result follows acute inflammation of the cord from the irritation thereby occasioned. In long standing cases when the muscles have become atrophied, there may be simple quantitative diminution in the response.

(3.) *Paralysis from disease of the grey substance of the cord.*—In such cases we meet with the reaction of degeneration, already described. The area over which the muscles and nerves are so affected depends upon the extent of the cord involved.

(4.) *Paralysis from disease of the peripheral nerves.*—In slight cases the electric reactions may be normal. In severe forms there is a total loss of response when either the galvanic or the Faradic current is applied to the nerve, and when the Faradic current is applied to the muscle. The galvanic current acting upon the muscle gives the reaction of degeneration.

(5.) There is besides a certain form of paralysis which cannot as yet be classified under any of these heads, *hysterical paralysis*, in which the reactions are normal.

[1] Very rarely indeed exceptions to this rule have been encountered.




* Return of voluntary motor power.

FIG. 109.—Reaction of degeneration in mild peripheral lesion, with rapid recovery. (ERB.)

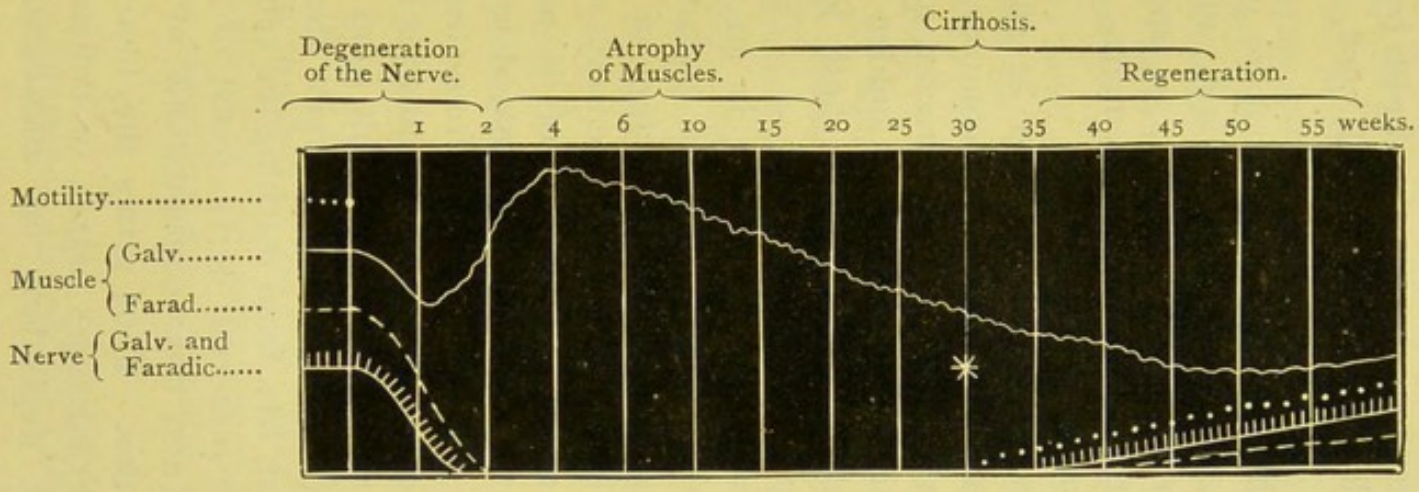



FIG. 110.—Reaction of degeneration in severe peripheral lesion, with slow recovery. (ERB.)

### III.—Affections of Co-ordination.

Such complex muscular adjustments as are involved in standing, walking, &c., are probably co-ordinated in the higher nervous centres. They may be interfered with by any affection of the brain, medulla oblongata, spinal cord, or peripheral apparatus.

When a patient (whose muscles need not be paralysed), can no longer carry out, as he formerly could, such combined movements as are involved in walking, in speaking, or in writing or making other fine hand movements, then we may say that there is inco-ordination.

The gait of such a patient, if the legs be affected, is distinctive and is well seen in cases of locomotor ataxia where the movements are stiff, jerky and ill-directed and where the heel is brought to the ground with a sudden stamp. The testing of the muscular co-ordination in both legs and arms has been already referred to.

The subject of ataxic inco-ordination is an obscure one, but I think that the following view tends to explain most cases. In order to the proper co-ordinated action of muscles, the individual must be informed through sensory nerves of the position of the limb and of the tension of its muscles. This information must be received and co-ordinated in some higher centre—probably in the cortex,—and from that centre the voluntary impulse which is to move the muscles must descend. It is therefore clear that ataxia may result from any lesion of the centripedal fibres which may prevent the impressions from skin, muscle, joint, &c., from reaching the centre. Lesions of sensory nerves, of posterior nerve roots, of the posterior columns of the cord and their continuations in the medulla, pons, &c., may do this. Thus arises the inco-ordination of locomotor ataxia, of Friedreich's ataxia, of bulbar disease, and generally of ataxias dependent on anæsthesia. Diseases affecting the co-ordinating centre may also cause ataxia, as we sometimes see in cases of cortical lesion. And finally, ataxia may result from some affection of the centrifugal fibres

by preventing the proper conduction of the stimuli to the muscles, or by producing an increased muscle tonus sufficient to interfere with co-ordination.

A special form of ataxia is that associated with disease of the cerebellum. It is rather a loss of the power of maintaining equilibrium than a true ataxia, and it manifests itself in a peculiar staggering gait resembling that of alcoholic intoxication, the patient when lying shewing no sign of muscular inco-ordination. It is usually accompanied with vertigo. Acting as the centre for maintaining equilibrium, the cerebellum only does so by means of peripheral impressions received from various sensory end organs. Among these are impressions proceeding from the skin, joints, &c., but in particular from the eye and from the semi-circular canals. Hence this form of ataxia—reeling, with vertigo and sometimes vomiting—is seen in cases of disease of these organs. Paralysis of the muscles of the eyeballs may cause it (as has already been said), and in Ménierè's disease and other forms of labyrinthine vertigo it occurs conspicuously. Apart however from interference with these peripheral impressions, cerebellar ataxia may occur from disease of the cerebellum itself, particularly of its central lobe.

Loss of muscular co-ordination in connection with speech will be subsequently considered.

### C.—**Vaso-motor Functions.**

As an index of the state of these functions we have to take the condition of the skin as regards pallor or redness, temperature, and the amount of the various secretions. Such changes in the tissues as sloughing, &c., are rather to be referred to the trophic nerves.

#### I.—*Cutaneous Vaso-Motor Affections.*

Diffused paleness or redness of the skin may be seen in persons in perfect health (blushing, &c.), but are often associated with nervous disorders such as epilepsy and hysteria, and

may be induced by various drugs, as, for example, the flushing which follows the inhalation of nitrite of amyl.

In fever the vaso-motor nerves of the skin appear to be in a condition of abnormal irritability. The hot, cold, and sweating stages of ague seem to depend upon general tonic contraction of the vessels of the skin, followed by general relaxation originating in all probability from the centre in the medulla.

More local changes may be brought on by mechanical or chemical irritation, but sometimes occur independently of such, as, for example, the local and circumscribed vaso-motor epileptic aura, which Nothnagel has described. Further, in various neuroses (epilepsy, Graves' disease, &c.), there are to be found scattered over the skin of chest and abdomen red blotches of congestion, to which Trousseau gave the name of *tâches cerebrales*, and which may sometimes be excited by drawing a pencil point over the skin. Various affections of the central nervous system are followed by vaso-motor changes in the skin. In paraplegia the temperature of the paralysed limbs frequently undergoes an increase, which is followed by a diminution. In cases of cerebral hemiplegia the temperature in the paralysed parts is almost invariably slightly elevated, and remains so for some time. Ultimately, however, it falls again, and in old standing cases not only is the temperature on the affected side lower than that on the healthy, but the pulse is smaller and more compressible, and the hand and foot pale and cold. Very high temperatures are sometimes met with in hysterical cases. Unilateral sweating and other vaso-motor disturbances are not uncommon in Graves' disease, epilepsy, and hysteria.

### II.—*Visceral Vaso-motor Affections.*

Lesion of the brain and medulla, and even passing psychical disturbances, often determine vaso-motor changes in internal organs (congestion of viscera in hemiplegia, disorders of menstruation from emotions, &c.). The secretions are sometimes affected from such causes. The urine, in particular, is liable to well-marked quantitative and qualitative changes; injury of

the 4th ventricle and other areas in medulla, cerebellum, and cord, giving rise to polyuria, albuminuria, and glycosuria. It seems also probable that certain forms of enlargement of the liver and spleen are dependent upon vaso-motor changes determined by affections of the central nervous system.

## CHAPTER XXXVII.

## NERVOUS SYSTEM—(*continued*).

### TROPHIC FUNCTIONS OF THE NERVOUS SYSTEM.

AMIDST a great deal that is uncertain in respect to these functions, there are several well ascertained facts which may be briefly alluded to here. The nutrition of all the tissues is under the control of the nervous system, and disease in this system may lead to serious and well-marked nutritional disturbance in various organs of the body. One or two of the more important of these trophic changes may be mentioned.

I. *Muscular Tropho-neuroses.*—Muscles may atrophy as the result of local causes, or as a consequence of long inaction (such as follows paralysis from cerebral hæmorrhage or embolism). Apart from these causes, when active neurotic atrophy of the muscles occurs, it is due either to disease of the nerve cells in the anterior horns of grey matter in the spinal cord or corresponding regions in the medulla, or to affections of those nerve fibres which connect these cells with the affected muscles. It is in such cases that electrical examination gives the "reaction of degeneration" already described, and hence its great importance as a means of diagnosis.

True muscular hypertrophy, as a pathological condition, is exceedingly rare, but it is seen in Thomsen's disease. With this must not be confused the pseudo-hypertrophy with increase of the size of the muscles, due to proliferation of interstitial connective tissue, which is seen in the pseudo-hypertrophic paralysis of children.

II. *Affections of Bones and Joints.*—In all nervous affections, whether peripheral or central, changes in the nutrition

of the bones and joints are liable to occur. Amongst the best known are those which follow locomotor ataxia, which may be acute articular swelling, closely resembling rheumatism, or chronic degenerative changes, leading to great deformities and strong predisposition to fractures. The important points to note in regard to these joint affections are that they are sudden in outset, and that they are not accompanied by fever or pain.

In acromegaly, a peculiar hypertrophy of nervous origin occurs in the bones of the hands, feet, and face.

III. *Affections of the Skin.*—Various eruptions, such as erythema, urticaria, eczema, herpes, may arise as the result of nervous disease. Paget has described an affection of the skin of the fingers, "glossy skin," which is due to the same cause. Still more important, as belonging to this category, are the acute and chronic bed-sores, which are so common and so troublesome in spinal cases. And, finally, there have to be noted the occurrence of pigmentation, and of affections of hair, nails, and cutaneous secretory apparatus, and the more profound lesions of lepra anæsthetica and the symmetrical gangrene of Raynaud's disease, all of which must be included among the cutaneous tropho-neuroses.

IV. *Affections of the Secretory Glands.*—Salivation and lacrymation, as well as the flow of the bile and other secretions, are under the influence of the nervous system, but do not give diagnostic indications further than has been already noted in other parts of this work. Sweating has been already alluded to.

## CHAPTER XXXVIII.

## NERVOUS SYSTEM—(*continued*).

### CEREBRAL AND MENTAL FUNCTIONS.

In many diseases of the nervous system the intellectual powers are affected. The powers of attention and memory of the patient are put to sufficient proof while the physician is informing himself regarding his history. Decadence of the power of judgment may betray itself in connection with the business transactions of the patient, which, if obviously irrational, will usually be communicated to the physician by his friends. The most striking interference with the intellectual powers is, however, loss of consciousness, or coma.

*Coma* is met with in simple fainting, in injuries of the head, in apoplexy (in which case it is accompanied with paralysis), in epilepsy (usually accompanied with convulsions), in hysterical attacks, in catalepsy, in uræmia, in severe attacks of fever of various kinds, and in narcotic poisoning. It is sometimes very difficult to establish a diagnosis between alcoholic poisoning, for example, and apoplexy. The state of the pupils, the smell of the breath, the condition of the heart, and the presence or absence of paralysis, will, however, usually make clear the nature of the case. Coma vigil, in which the patient lies unconscious, with the eyes wide open, is met with in cerebral diseases.

There are certain other disorders of intelligence which frequently occur in mental disease, and which must be noticed here.

*Illusions* are objective disorders of perception—a sound is heard, or an object seen; but both perceptions are misinterpreted.

*Hallucinations*, on the other hand, are subjective disorders of perception. The patient may imagine, for example, that rats are running over the bed-clothes, or that he hears people calling to him, when no foundation exists for either belief. These are hallucinations. But if he observes some dark object on the bed and takes it to be a rat, that would be an instance of an illusion.

*Delusions* have no relation to perception. They are purely mental, and are only met with in the insane. It is not uncommon, for example, to meet with such delusions as that the patient believes himself to be the Deity.

*Delirium*, or wandering of the mind, indicated by incoherent speech, may consist in low muttering or in wild and furious shouting. The former variety is most frequently met with in cases of nervous exhaustion, the result it may be of fever, or of any grave organic disease. The more noisy form of delirium occurs in meningitis, in acute mania, and as the result of some poisonous ingredient circulating in the blood, such as alcohol, fever poison, belladonna, carbonic acid, and other substances. Delirium may also be caused by reflex irritation in connection with such organs as the stomach or uterus. A variety is not uncommon in pneumonia, and is even occasionally met with in phthisis pulmonalis.

It is also important to note further the condition of the patient in regard to mental *emotions*, whether these are under full control or not. This is very obviously not the case in hysterical persons, and in many other nervous affections the patient may be observed to be emotional and excitable.

## CHAPTER XXXIX.

## NERVOUS SYSTEM—(*continued*).

### SPEECH.

In considering the subject of speech, we have first to deal with the disorders of the various cortical centres concerned in the production of spoken and written language (Aphasia), and second, with the anomalies of Articulation.

### Aphasia.

This subject is a very large and a very difficult one, and its complexity has been rendered greater by the fact that the terms used by various writers differ considerably. The terminology which I adopt in teaching is that of Wyllie, whose valuable work on the "Disorders of Speech" has been of great use to me in preparing this chapter.

If such an object as a bell, or an apple, let us say, is placed in the hands of a child, he acquires certain information about it. He notices its weight, its shape, colour and general appearance, the smoothness or roughness of its surface, the sound it gives in the case of the bell, its smell and taste in the case of the apple. Through every sense, then, impressions regarding the object he holds reach the centres in the child's brain and are perceived by him. But the impression thereby produced on each sense-centre is not merely a momentary one, it is more or less permanent, these centres storing up memories of the impressions made on them, and the child will never forget the taste of apple or the tone of a bell. And not only are these sense impressions stored up, but they are also linked together in such a way that when next the child tastes an apple the other sense memories arise, so

that he recognises that the taste is that of a thing which has formerly produced other impressions on his senses. The union or blending of all these impressions is what is called the *percept* of the apple.

As the child begins to learn language, he realises that the word "apple" is the name of the object with which he is already familiar, and the sound of this word, perceived in his cortical auditory centre, is stored there as a sound-memory. Presently he tries to reproduce this sound, which he does by means of the cortical motor centre which governs the utterance of spoken language, and when he has succeeded in doing so the memory of the mechanism required remains in the motor centre as a motor word-memory. The blending of these two memories, the memory of the sound of "apple" and the memory of how the word "apple" is spoken, constitute the *percept* of the word "apple," and whenever, in the future, thought desires to find expression in such a word, both the sensory auditory image and the motor image must be called up before the word can be used.

Further on in his education the child learns and stores up two other sets of memories, one a sensory, the memory of the appearance of words written or printed, and the other a motor, the memory of the actions necessary to write particular words.

Thus, there are four centres for the understanding and originating of language, written and spoken. Of these, two are sensory and two are motor, and all four are situated in the cortex of one hemisphere, in right-handed persons on the left side, in left-handed persons on the right. It is to the result of lesions of these centres that the term Aphasia is applied, and as will readily be understood, the manifestations of aphasia differ according to the particular centre which has been destroyed together with its memories. Of late years, partly as the result of experiment on monkeys, partly by means of clinical observation of the results of localised cortical lesions, the position and limits of these four speech centres have been determined with fair accuracy. These are indicated in fig. 111, where the centres are marked as follows :—

A. The auditory centre where are stored the memories of the sound of words, where, consequently, language heard is interpreted and understood. There also, when thought is striving to find expression, the auditory word-memories are called up in order to their subsequent motor expression. This centre corresponds to the posterior three-fourths of the first temporal convolution.

B. The motor speech centre, where are stored the motor word-memories, and whence originate the motor im-

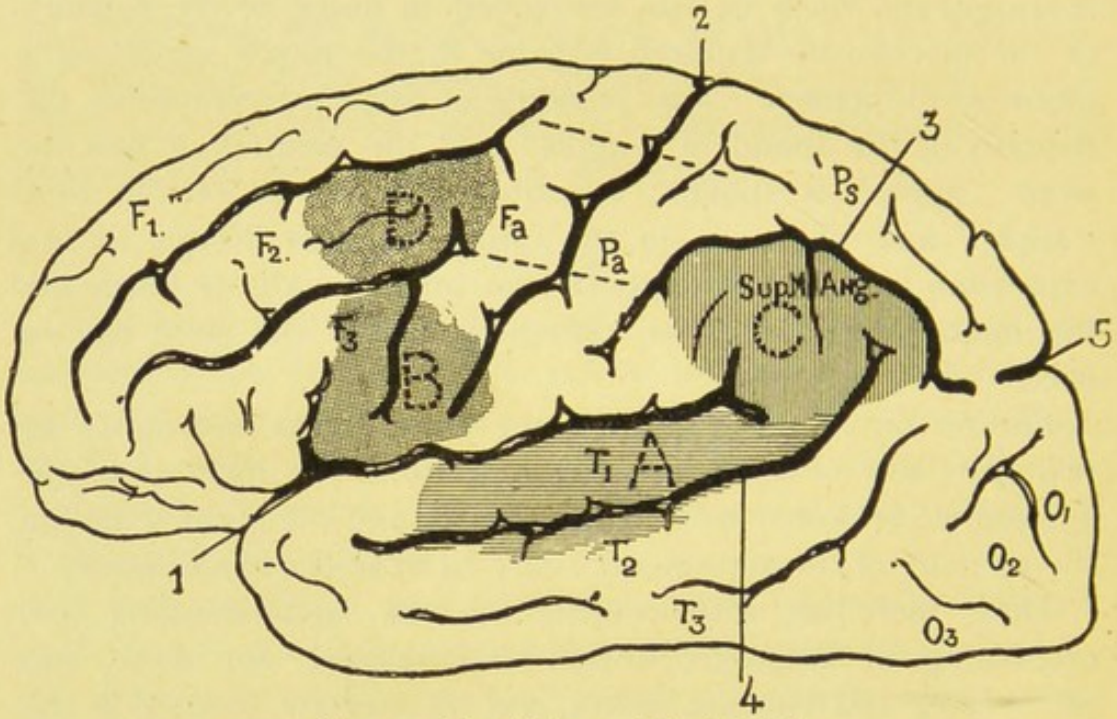



FIG. 111.—Speech Centres (after Wyllie).

pulses of speech. This centre, which is that originally described by Broca, corresponds to the foot of the third frontal, the foot of the ascending frontal, and part of the foot of the ascending parietal convolutions.

C. The visual centre, where are stored the memories of the appearance of written and printed words, and where consequently, resides the power of understanding and interpreting written language. This centre is found to correspond to the angular and supra-marginal convolutions.

D. The graphic-motor centre, where the motor memories of the movements required in the act of writing are stored. It lies at the posterior end of the third left frontal convolution, and is therefore very near in position to the motor centres for the right hand, and also close to Broca's centre.

These centres are all very closely connected to each other.

Using then the term Aphasia in the wider sense, and considering it to include all disorders of spoken or written language caused by disease of these cortical centres, there are four chief forms of that disorder.

1. **Auditory Aphasia**, sometimes known as "sensory aphasia," and due to lesion of the centre A. The effects of such a lesion are two-fold, according as the centre is viewed from the receptive or from the emissive standpoint. The memories of word-sounds are gone, and therefore the patient is "word deaf," that is, the words of spoken language are perceived by him merely as sounds, which sounds he can no longer understand. And even if the centre C is intact it is doubtful whether the power of reading remains after destruction of A, for the visual centre C appears to require the aid of the auditory centre in order to grasp the meaning of a word read.

On the emissive side also, that is in the production of language, destruction of the centre A has very great influence, for, as has been said already, when thought is seeking expression it calls up auditory word-images as well as motor word-images, and it is only when both are simultaneously present that perfect speech can be effected. The destruction of the centre A, although it may not entirely prevent speech, damages it considerably, there being more or less *amnesia verbalis*, *i.e.*, failure of the power to recollect words—particularly nouns—when the effort to speak is being made. There is also the condition of *paraphasia*, in which the patient uses words which he does not intend to employ, and which do not express his meaning, and being "word deaf" he cannot recognise his mistakes and correct them as he can do to some extent when the paraphasia arises in other

ways. Similar effects are produced on writing, and such a patient can no longer repeat correctly words which are spoken to him.

2. **Motor Aphasia**, to which the name of Aphemia may be given, results from the destruction of the centre B. This, of course, prevents the patient from calling up motor word-images, and from originating word-utterance. He can sometimes still utter one or two words, such as "yes" or "no," and sometimes makes use of oaths and other emotional expressions, but these are believed to be due to the action of the uneducated centre in the opposite hemisphere. But, as the auditory centre is uninjured, he can understand perfectly what is said to him. It has been pointed out that thought which is seeking expression requires that the motor images and the auditory should be simultaneously called up, and as in this case the former are destroyed, there is probably amnesia verbalis, and this is shewn by the fact that although the graphic motor centre is not destroyed, the patient cannot express his thoughts in writing, nor can he understand written language.

The fibres which connect the centres A and B run under the Fissure of Sylvius and the Island of Reil, and, when they are destroyed by a lesion situated there, a peculiar kind of aphasia is produced which has been called conduction aphasia: the "Leitungsaphasia" of Wernicke. Here the centre A is uninjured, therefore there is no word-deafness, the patient understanding all that is said to him. The centre B is uninjured, therefore there is no motor aphasia, the patient being able to speak. But, in that the connection between these two centres is cut, he is apt to use wrong words, those, that is, which do not express his meaning,—shewing therefore the phenomena of *paraphasia*.

3. **Visual Aphasia**, due to lesion of the centre C. Here we have destruction of the visual memories of words, which the patient has stored up there. He is therefore "word-blind." He can no longer read (alexia); nor can he write, because,

although the centre D is uninjured, he is not able to call up those visual memories of the appearance of words which are necessary to him before he can write them.

This inability to write appears to be always present in cases of destruction of the centre C. But some persons who are "word-blind" are found to be able still to express their thoughts in writing. In these cases the lesion is not in the cortical centre C, which is uninjured, but has cut the connections between that centre and the primary optic centres in the two occipital lobes. In speaking of homonymous hemianopsia (page 339) it was pointed out that these primary optic centres lie in the occipital cortex, in the neighbourhood of the cuneus, and that, if the optic radiations of Gratiolet, which pass to the left cuneus, be destroyed, right homonymous hemianopsia will be developed. Now if, in addition to the optic radiations and the fibres which connect the left cuneus with centre C, the lesion also destroys those fibres which unite the right cuneus with centre C, then the condition of word-blindness will be added, because all the connections between the retinæ and centre C are cut. But, as the centre itself is intact, the patient can still call up his visual memories of words, and, with the aid of centre D, he can still write. The elucidation of this point we owe in great measure to the work of Dejerine, and the following diagram which shews the position of a lesion causing word-blindness but not agraphia, is one contained in a paper which he communicated to the Société de Biologie, Feb. 27th, 1892.

In cases of word-blindness, it ocasionally occurs that a peculiar condition is also present which has been called "mind-blindness." In this the patient loses the power of recognising well-known objects, and fails to remember the faces of familiar friends.

4. **Graphic Motor Aphasia**, due to lesion of the centre D, is much less definitely understood than the other forms. This arises from the fact that there is very little clinical evidence bearing on the point, and that the experimental evidence is not very conclusive. There can, however, be no doubt that

the motor graphic memories are stored in this centre, and that, consequently, its destruction would involve the loss of

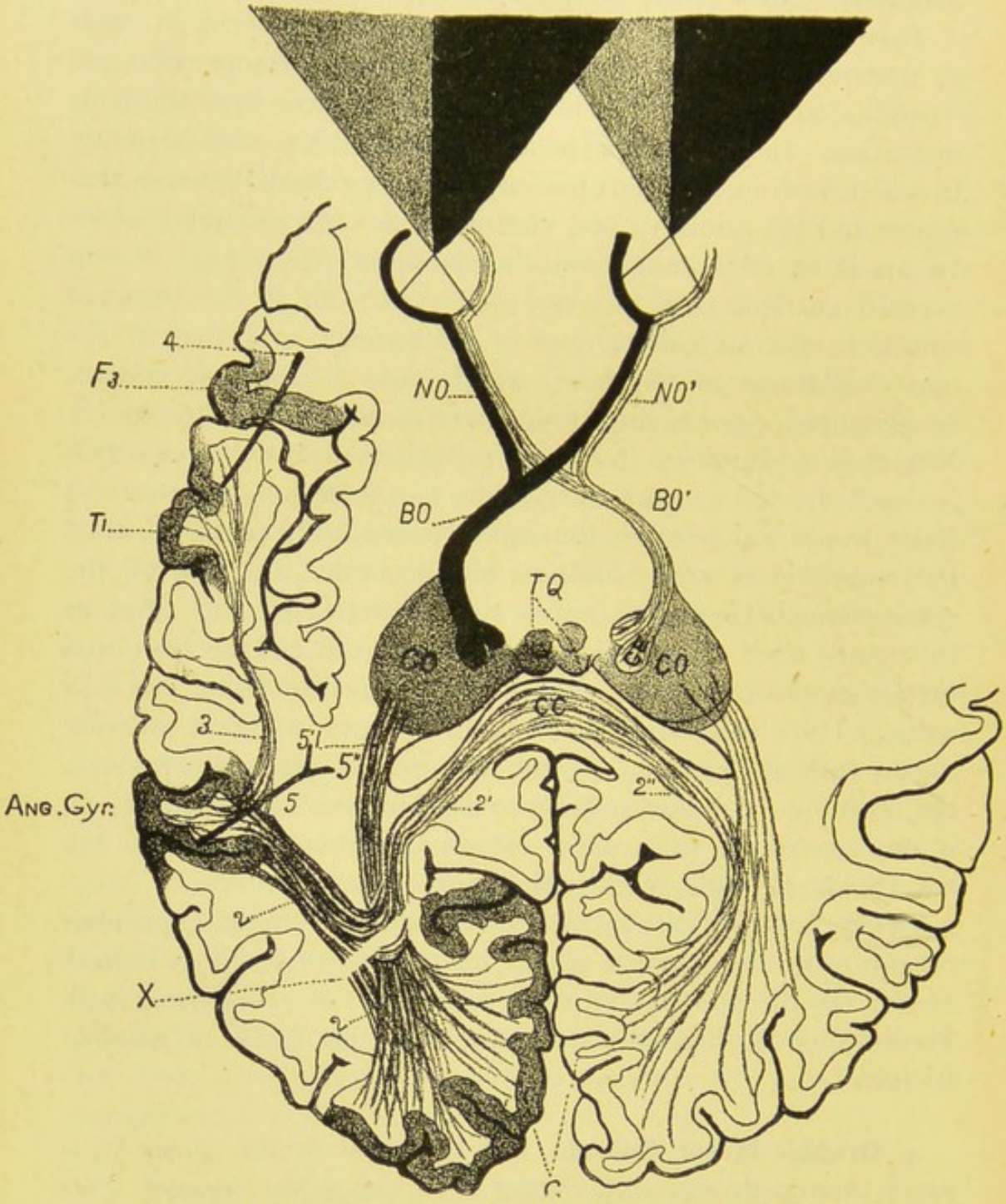



FIG. 112.—Course of the Optic Fibres (from Wyllie, after Dejerine).

these, and produce agraphia, at least in so far as the right hand is concerned.

Such are the simple forms of aphasia. For the more complex, special works on the subject should be consulted.

Passing now from the disorders of the speech centres, a word may be said as to the lesions which may interfere with the expression of speech by affecting the speech tract, either in its upper neuron which extends from the motor cortex through the internal capsule and crus to the nuclei of the cerebral nerves which innervate the laryngeal and oro-lingual mechanisms, or in its lower neuron, extending from the nuclei of these nerves to the muscles in question.

The fibres of the upper neuron are affected in multiple cerebro-spinal sclerosis where the well-marked "scanning speech" in which each syllable is pronounced separately, slowly and with effort, is believed to be due to interference of conduction produced by the pressure of proliferated connective tissue. The speech of Friedreich's disease closely resembles this.

In general paralysis the speech is also sometimes of a staccato or scanning character, but its characteristic is that the syllables are slurred, and are often misplaced, suggesting indeed rather a cortical origin, than one merely affecting the motor tract.

Affections of the motor nuclei in the pons and medulla, of which the type is bulbar paralysis, cause peripheral paralysis of the muscles of lips, tongue, &c., with consequent wasting, and impede articulation to so marked an extent that finally the patient may not be able to articulate at all.

Stammering is due to faulty co-ordination between the oral and the vocal executive mechanisms of speech.

**Sleep.**—The disorders of sleep are of considerable practical importance. They are mainly three—

1. *Somnolence.*—Apart from the natural aptitude for sleep possessed by persons of a lethargic temperament, the causes of somnolence are mainly as follows:—exposure to external cold, especially when combined with insufficient nutriment; overloading the stomach with food; dyspepsia; blood poisoning

(uræmia, fevers, poisoning with narcotics, alcohol, carbonic acid, &c.); disease of the brain.

2. *Insomnia*, or want of sleep, may be directly due to pain. It may further arise from excessive mental work, worry, anxiety, from dyspepsia, from the use of tea or coffee, from cerebral disease, from insanity, and from heart disease.

3. *Somnambulism.*—In this case also a definite cause, similar to the above, may usually be found.

## CHAPTER XL.

# NERVOUS SYSTEM—(*continued*).

### CONDITION OF CRANIUM AND SPINE.

**Cranium.**—The condition of the cranium sometimes affords important indications in nervous cases. Its shape and form should be observed, and if necessary measured, and if there be asymmetry (a sign of degeneration), this should be mapped out as in the case of the thorax (see p. 195). One of the most common alterations in the size and shape of the cranium is that of chronic hydrocephalus where the skull becomes more or less globular, the frontal bone prominent, the sutures open, and the fontanelles large and pulsating. On palpating the skull localized depressions may be found, the result of old depressed fracture, which, in the case of epilepsy, give valuable indications. The thin bones of cranio-tubes give a curious crackling under the finger.

On percussion of the skull sensitive points may be discovered, which, in the absence of hysteria, point to local organic cerebral mischief.

**Spine.**—Examination of the spine includes inspection, palpation, percussion, and the "hot sponge test."

*Inspection.*—The patient should, if practicable, be stripped, and be made to stand upright, with the feet close together and firmly planted. If an ink mark be made on the skin over the tip of each spinous process, the line of the spine will be rendered distinct, and any lateral curvature will readily be detected. At the same time, any displacement of the column caused by angular curvature will become apparent.

*Palpation* of the spinal column should be practised both

posteriorly and anteriorly through the abdominal walls. Tumours of the vertebræ may thus be detected, as well as the presence of local tenderness at any particular point.

*Percussion* of the spine posteriorly may cause pain when there is disease of the vertebræ or of the spinal membranes, in myelitis, in spinal irritation, &c.

*Hot Sponge Test.*—This test consists in passing down the spine a sponge which has been wrung out of warm water, and which is not so hot as to be unpleasant to the healthy skin. In certain cases, particularly in myelitis, pain is experienced by the patient as the sponge passes over the seat of the disease.

The employment of the X-rays in investigating the spine has proved to be of diagnostic value.

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