Medical diagnosis : a manual of clinical methods for practitioners and students.

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Publication/Creation

Edinburgh : W. Green, 1906.

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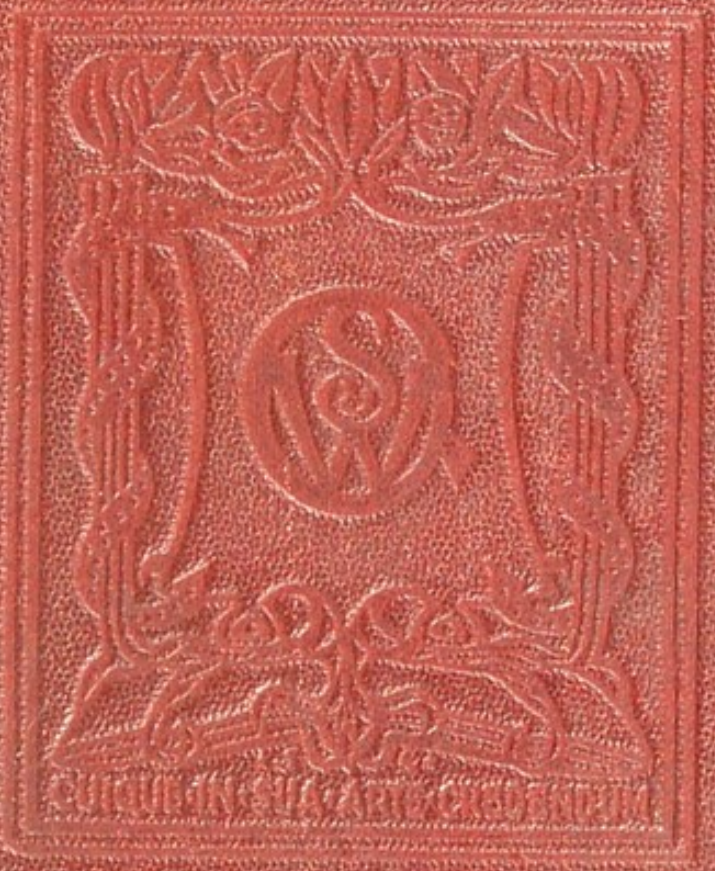
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MEDICAL DIAGNOSIS

MEDICAL DIAGNOSIS

A Manual of Clinical Methods for Practitioners and Students

FIFTH EDITION

GREATLY ENLARGED AND REVISED TO DATE

BY

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WITH 200 ILLUSTRATIONS AND 8 FULL-PAGE PLATES

EDINBURGH & LONDON

WILLIAM GREEN & SONS

MEDICAL PUBLISHERS

1906

PREFACE TO THE FIFTH EDITION

In preparing this edition for the press every section has been carefully revised, and in almost every case considerable additions have been made, which, it is believed, have brought the book in line with present knowledge.

This work of revision has been performed with great care and thoroughness by Dr W. T. Ritchie.

To him and to various other of my friends who have given me advice and assistance I wish to express my grateful thanks.

For the opportunity of making the photographs, Figs. 35 and 49, I am indebted to the courtesy of Major D. G. Marshall.

J. J. G. B.

Edinburgh, *September* 1906.

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MEDICAL DIAGNOSIS

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 the physician 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 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 inquiries 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, etc., 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 in which heredity is an important factor, 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, and to a lesser extent excessive con-

sumption of tea and tobacco. 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, hewers being more affected than builders.

(*b*) Poisonous Effects of Materials worked in—such as lead (workers in lead factories and in potteries, compositors and house painters); phosphorus (makers of lucifer matches); copper and zinc (brass founders); arsenic (makers of wall paper, sheep-dip, etc.); bisulphide of carbon (those engaged in vulcanising rubber goods); aniline (dyers); anthrax spores (those working with hides, wool and hair; rag sorters, slaughter-house butchers); etc.

(*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, etc. 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 or has demanded in former years.

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 (as constipation, tonsillitis, bronchitis, palpitation, etc.), present or previous residences or other circumstances which may have influenced the production or development of disease.

If there be reasonable grounds for suspecting *syphilis* in a male patient, it is better to ask, "When did you have syphilis?" rather than "Have you had syphilis?" If the patient be a female, it may be advisable to inquire into the condition of the reproductive functions, asking her whether she is "regular every month," inquiring as to "the change of life," and so forth.

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
    (1) Colour.
    (2) Perspiration.
4. Condition of the subcutaneous tissue—
    (1) Adiposity.
    (2) Œdema.
    (3) Myxœdema.
    (4) Emphysema.
5. Expression of the face.
6. Attitude.
7. Evidence of previous injury or disease.
8. 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. The following table gives the average weight of a man between the ages of thirty and forty years:—

5 ft.	2 in.	...	9 st.	9 lb.		5 ft.	9 in.	...	11 st.	10 lb.
5 ,,	3 ,,	...	9 ,,	12 ,,		5 ,,	10 ,,	...	12 ,,	2 ,,
5 ,,	4 ,,	...	10 ,,	2 ,,		5 ,,	11 ,,	...	12 ,,	7 ,,
5 ,,	5 ,,	..	10 ,,	5 ,,		6 ,,	0 ,,	...	12 ,,	13 ,,
5 ,,	6 ,,	...	10 ,,	9 ,,		6 ,,	1 ,,	...	13 ,,	6 ,,
5 ,,	7 ,,	...	11 ,,	0 ,,		6 ,,	2 ,,	...	13 ,,	13 ,,
5 ,,	8 ,,	...	11 ,,	5 ,,						

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 deposition 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: (1) 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), from any disease leading to loss of blood (as from hæmorrhage, menorrhagia, gastric ulcer, etc.), or causing hæmolysis (destruction of red blood corpuscles, as in pernicious anæmia, malaria, and other infective diseases), or from Bright's disease, malignant disease, etc., or finally, from any affection of the vascular system interfering with the proper propulsion of the blood (mental emotions, myocardial and valvular disease of the heart, etc.). 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. Pallor is also readily appreciated by examining the conjunctivæ and the mucous membrane of the 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

associated with inflammation and 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 certain congenital malformations of the heart, particularly in cases of constriction of the pulmonary orifice along with patency 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 most characteristically seen in such cases 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 (*icterus*), or yellow discoloration of the skin, results from obstruction to the outflow of bile into the duodenum and the consequent absorption of bile pigment into the circulation, and the staining of the tissues. Jaundice 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. Slight and transient jaundice is usually due to acute catarrh of the bile ducts, which may in some instances be caused by the passage of gall-stones; persistent and progressive jaundice of a severe degree is seen when the common bile duct is occluded by a calculus or by malignant disease. The question of jaundice will be more fully treated in connection with the urinary system (see p. 318).

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

(*e*) Bronzing occurs in Addison's disease (disease of the suprarenal capsules). The pigment is deposited in and between the cells of the Rete Malpighii, 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 discoloured. The patches of pigmentation are (except those caused by local irritation) arranged symmetrically, and their margins are rarely well defined,

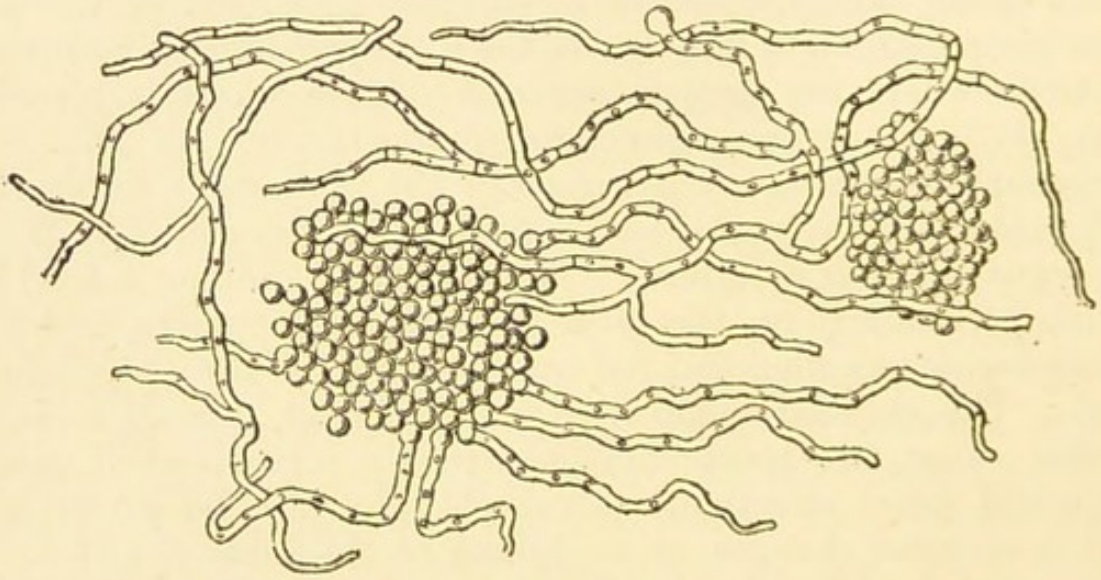


Fig. 1.—Microsporon furfur (after Neumann).

but rather shade off imperceptibly. Pigmentation of the skin, closely resembling Addison's disease, is met with in bronzed diabetes, and sometimes in chronic phthisis, 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. In bronzed diabetes, the pigmentation is diffuse, and the mucous membranes are unaffected. 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* (*Tinea*) *versicolor* the presence of the parasite

Microsporon furfur gives rise to a brown discoloration, readily distinguished by its situation on the trunk, its "fawn" or "liver" colour, and by microscopic examination of a scraping from the epidermis, which when treated with liquor potassæ presents interlacing mycelial threads and groups of spores (Fig. 1).

In some cases of pediculosis attended with much scratching, the

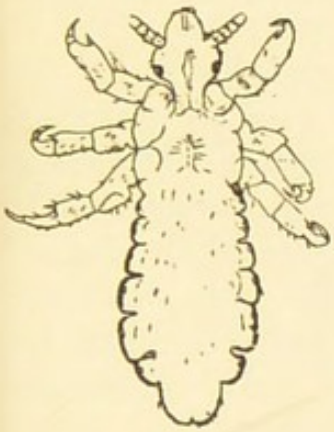


FIG. 2.
Pediculus capitis.

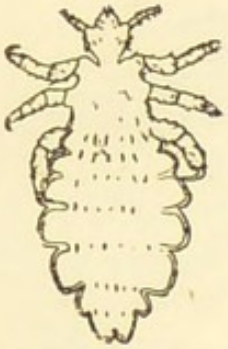


FIG. 3.
Pediculus corporis.

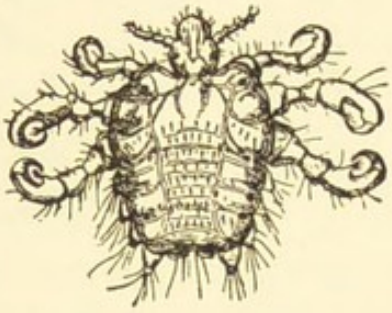


FIG. 4.
Pediculus pubis.

(After Max Braun.)

skin may be extensively and deeply pigmented. The detection of the *Pediculus corporis* (Fig. 3) will determine the diagnosis. This parasite must not be mistaken for the *Acarus scabiei* (Fig. 5).

(*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.

(2) **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 malaria is that of profuse perspiration, and the night sweats of the patient in the late stages of phthisis are among the most distressing symptoms of that disease. 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 bacteria.

Diminution of perspiration is seen locally in many chronic skin affections, and generally in almost all conditions of pyrexia

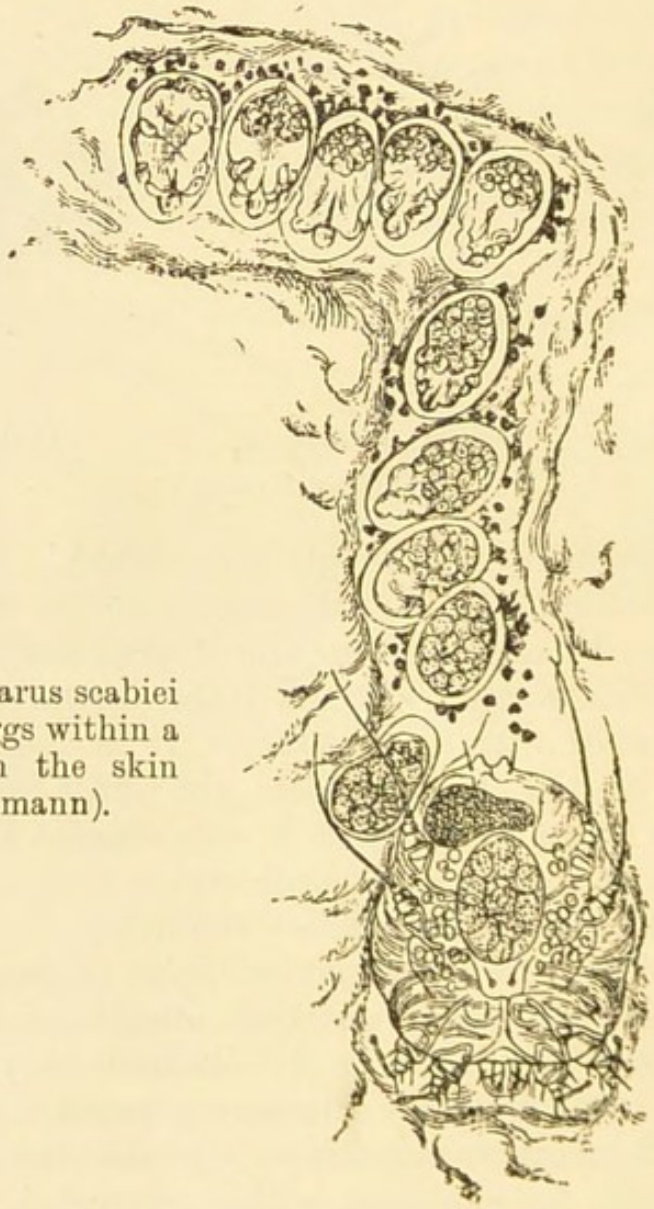


FIG. 5.—Acarus scabiei and its eggs within a burrow in the skin (after Neumann).

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, owing to pigmentation produced by *Bacillus pyocyaneus*.

4. **Condition of the Subcutaneous Tissue.**—There are four pathological states to be noted under this head—viz. (1) Adiposity; (2) Œdema; (3) Myxœdema; and (4) Emphysema.

(1) Adiposity

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 connective 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. Glucose 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.

(2) Œdema

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. If the finger be pressed upon the skin, a deep indentation remains after withdrawal of the pressure, and only slowly fills up. In well-marked cases the external genitals become greatly swollen (the ram's-horn penis). 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—

(*a*) From obstruction to the return of 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 may give rise to œdema of the lower limbs with associated ascites; obstruction of the veins of a limb, as by

thrombosis or the pressure of a tumour, results in œdema confined to the affected limb.

(*b*) 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, whether the result of superficial inflammation, or as an indication of inflammation in the deeper parts, is as a rule of greater interest to the surgeon than to the physician.

Elephantiasis, affecting one or both lower limbs, the scrotum, etc., may result from long-continued lymphatic obstruction. The affected part is much enlarged and very hard from inflammatory overgrowth of connective tissue.

(3) Myxœdema

In myxœdema the subcutaneous tissues are infiltrated with a peculiar mucoid substance, which causes a dropsy-like swelling all over the body. The swelling differs 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. The face is most characteristic, and the condition is therefore described more fully on p. 13.

(4) Subcutaneous Emphysema

Subcutaneous emphysema is produced by a collection of air or gas 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 subpleural rupture of one or more air vesicles in the lung, as a result of violent expiratory efforts. 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 or gases under the skin if these structures have been previously fixed to the abdominal wall by adhesions.

In rare instances the subcutaneous tissues are distended with gases formed by anaerobic bacilli—*Bacillus aerogenes capsulatus* (*Bacillus phlegmones emphysematosæ*), etc.

5. **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, etc. To some of these allusion has already been made; a few remarks, however, may be added.

The enlargement of the face in ACROMEGALY is very striking. The lower jaw is especially enlarged, the lower teeth project forwards, and the nose, ears and lips also undergo enlargement.

In MYXŒDEMA the face is swollen and expressionless. The nose and ears are thickened, the palpebral fissures are narrowed, and there is transverse wrinkling of the brow, owing to contraction of the occipito-frontalis muscle in the attempt to widen the palpebral apertures. In myxœdema there is moreover usually marked swelling of the dorsum of the hands and feet, and swelling in the supraclavicular fossæ. Together with these signs there is marked dryness and coldness of the skin, loss of hair, great hebetude of the nervous system, and in late stages, which are now rarely seen in consequence of the success of thyroid treatment, insanity is liable to develop.

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.

Excessive prominence of one *Eyeball* is seen in connection with tumours of the orbit. Bilateral prominence of the eyeballs (*Exophthalmos*) is observed in exophthalmic goitre; in such cases we look for increase in the width of the palpebral fissures (Stellwag's sign) due to tonic contraction of the levator palpebræ superioris, and for defective descent of the upper eyelids when the patient looks downwards (von Graefe's sign), so that the sclerotic is seen between the upper edge of the cornea and the upper lid.

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 visibly pulsate, and the mouth is usually half open on account of the dyspnœa. In aortic regurgitation, 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 laryngeal diphtheria, laryngismus stridulus, œdema glottidis, paralysis of the posterior crico-arytenoid muscles, etc.

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 drooping corner of the mouth, the eye is open and watery, and the whole skin of that side becomes smoothed out and loses its wrinkles. The unilateral paralysis is well seen when the patient is asked to laugh, elevate the eyebrows, shut the eyes, whistle, puff out the cheeks, and raise the upper lip as if to show the teeth.

Facies of Bulbar Paralysis.—The lower part of 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 Paralysis agitans.—The features are rigid and expressionless, like a mask, but the facial muscles are not paralysed, and there is consequently no drooping of the corners of the mouth. The eyes alone express the emotions.

Adenoid facies.—This is characterised by the drooping lower jaw, the separation of the lips, the marked naso-labial folds, the transverse broadening of the nose, and the dull and somewhat vacant expression of the face.

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.

6. **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 often lies with his knees drawn up; many patients with aneurism of the thoracic aorta lean forward with the arms resting on the bed-table; the patient suffering from pleurisy with effusion or pneumothorax lies on the affected side; and so with many diseases which compel the assumption of some more or less characteristic attitude.

7. **Evidence of Previous Injury or Disease.**—Apart from the obvious traces of such surgical affections as fractures, etc., 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 in the neck resulting from tuberculous disease of lymphatic glands, the large joints of those

who in their childhood have been subject to rickets, old-standing paralysis, the signs of syphilitic lesions (the fallen in bridge of the nose, perforation of the palate, corneal infiltrations, etc.), are among these indications.

8. **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] Each time before the thermometer is used, the index must be jerked down below the normal point.

It is most usual to take the temperature by placing the instrument in the axilla, and directing the patient to keep the arm in apposition with the chest wall. The skin of the axilla must previously be carefully dried, should there have been any perspiration. It is frequently more convenient to ascertain the temperature in the mouth. The bulb of the instrument, which has been washed in cold antiseptic solution, is placed under the tongue, and the patient is directed to close the lips. Accurate results are also obtained when the instrument is passed into the rectum. In infants this is the best method of taking the temperature.

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 mercury has reached its highest point.

In all cases of importance the temperature ought to be taken twice a day, morning (9 to 11 A.M.), and evening (5 to 7 P.M.), and in serious cases every two or four hours. From the data so obtained charts should be constructed in the usual manner.

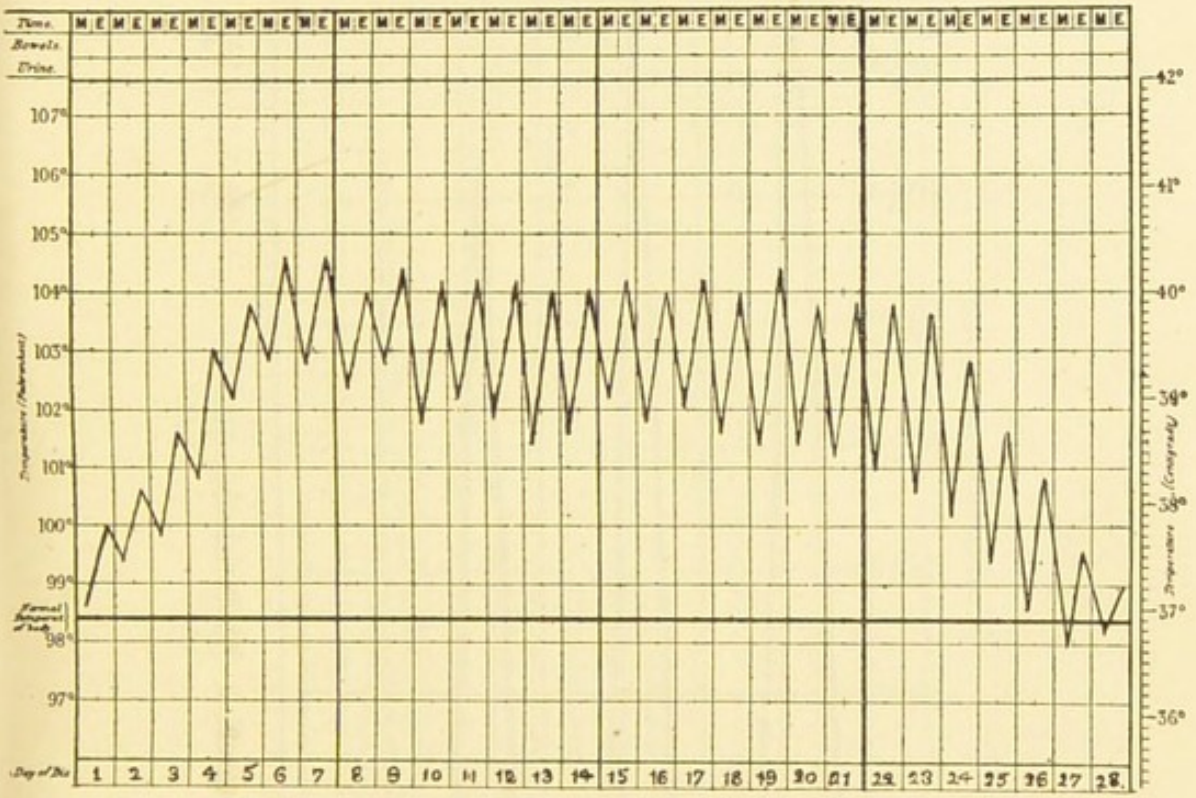
While the *mean normal temperature*, taken in the axilla, may be stated to be 98·4° F., yet diurnal variations are observed even in healthy persons. The daily minimum temperature (in the axilla, 97·2° F.) is observed between 2 A.M. and 8 A.M., the daily maximum temperature (98·7° F.) being attained between 3 P.M. and 5 P.M. The temperature taken in the mouth or rectum is from 0·5° F. to

[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.

0·8° F. higher than that in the axilla, the mean rectal temperature being 99·3° F.

The temperature of the body in disease may rise above, or fall below, the normal level.

Elevation of Temperature or Pyrexia is one of the chief manifestations of fever. Three stages in the course of the febrile temperature should be noted—(1) *The stage of rise* or *pyrogenetic stage*. A sudden continuous rise of temperature, to a given height, is often accompanied by sensation of coldness, or even a definite rigor, as in acute lobar pneumonia, typhus fever, small-pox, etc. Or the rise of temperature is gradual, and somewhat


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(1) *Accuminated.*—The temperature rises suddenly, remains at a high level for a short period, and falls rapidly.

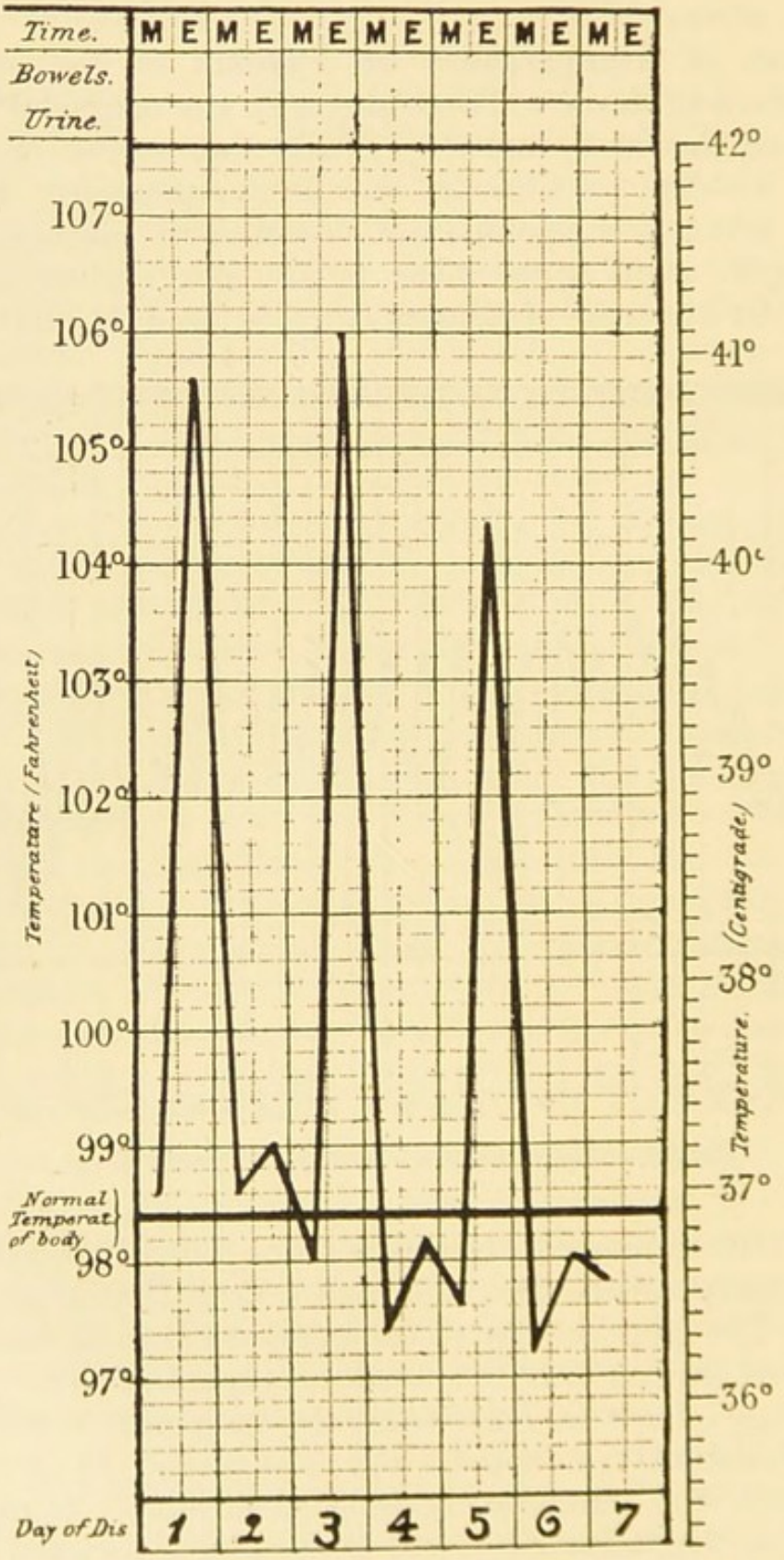


FIG. 7.—Temperature chart in tertian fever.

(2) *Continued.*—The temperature in the second stage remains elevated for a prolonged period, lasting several days or weeks, and

is subject only to diurnal variations, as in acute lobar pneumonia, typhus fever, and acute suppurative leptomeningitis.

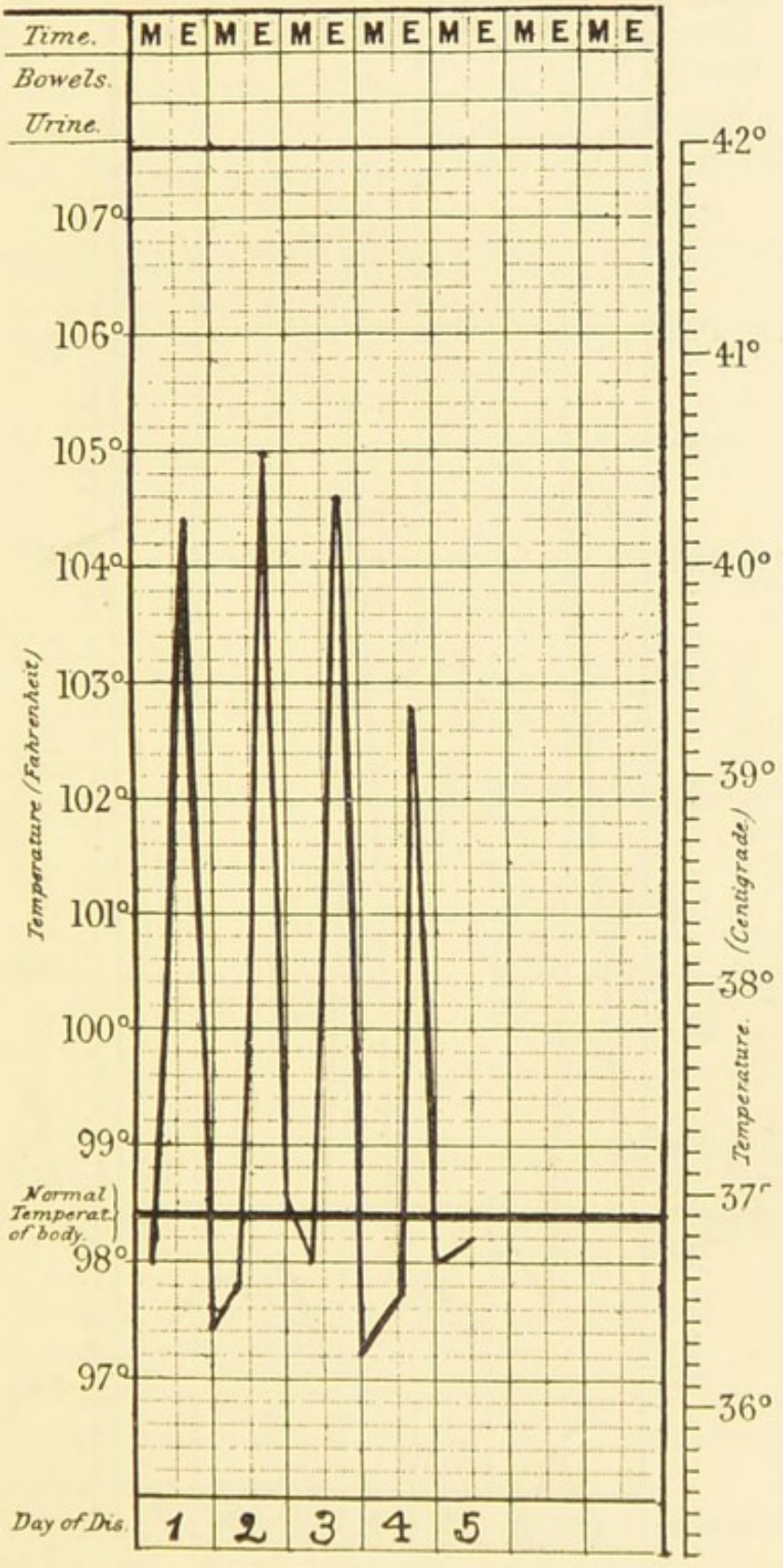


FIG. 8.—Temperature chart in double tertian (quotidian) fever.

(3) *Remittent.*—The difference between the morning and evening temperatures is considerable, say, 2° F. or more, but the minimum

temperatures during the second stage do not fall to the normal level. This type of temperature is observed in typhoid fever

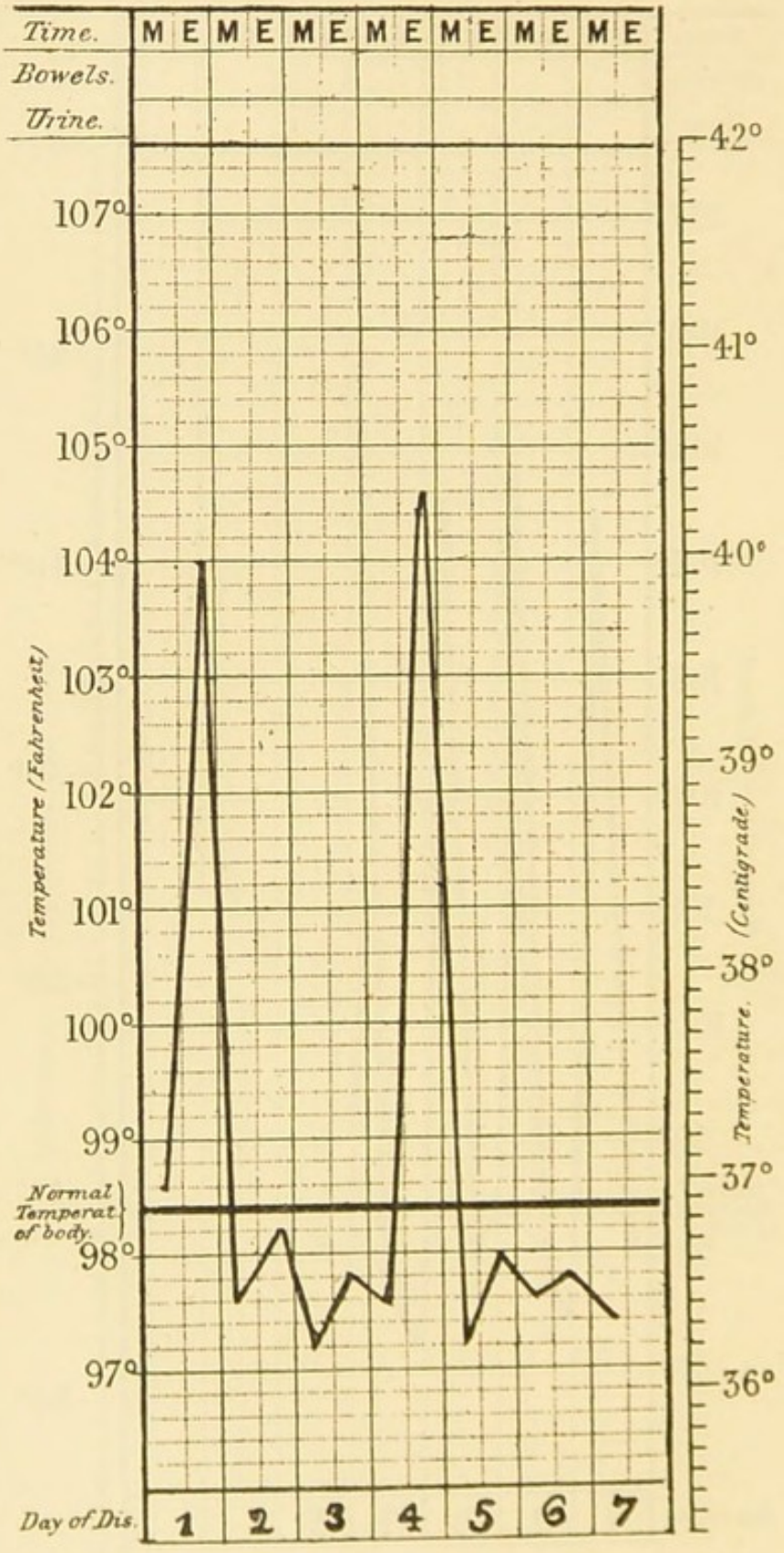


FIG. 9.—Temperature chart in quartan fever.

(Fig. 6), chronic tuberculosis (hectic fever), pyæmia, ulcerative endocarditis, etc. Occasionally (particularly in pulmonary tuber-

culosis) it is the morning temperature which is high, that in the evening being low (*Inverted* type of remittent temperature).

(4) *Intermittent.*—In this type, the temperature at each successive fall reaches the normal level, at which it remains for a longer or shorter period (intermission), then again the temperature rises as before, and so on. Examples of this type of temperature are seen in the fever of septic origin, and in malaria. In *malaria* the periodicity of the febrile paroxysm varies according to the

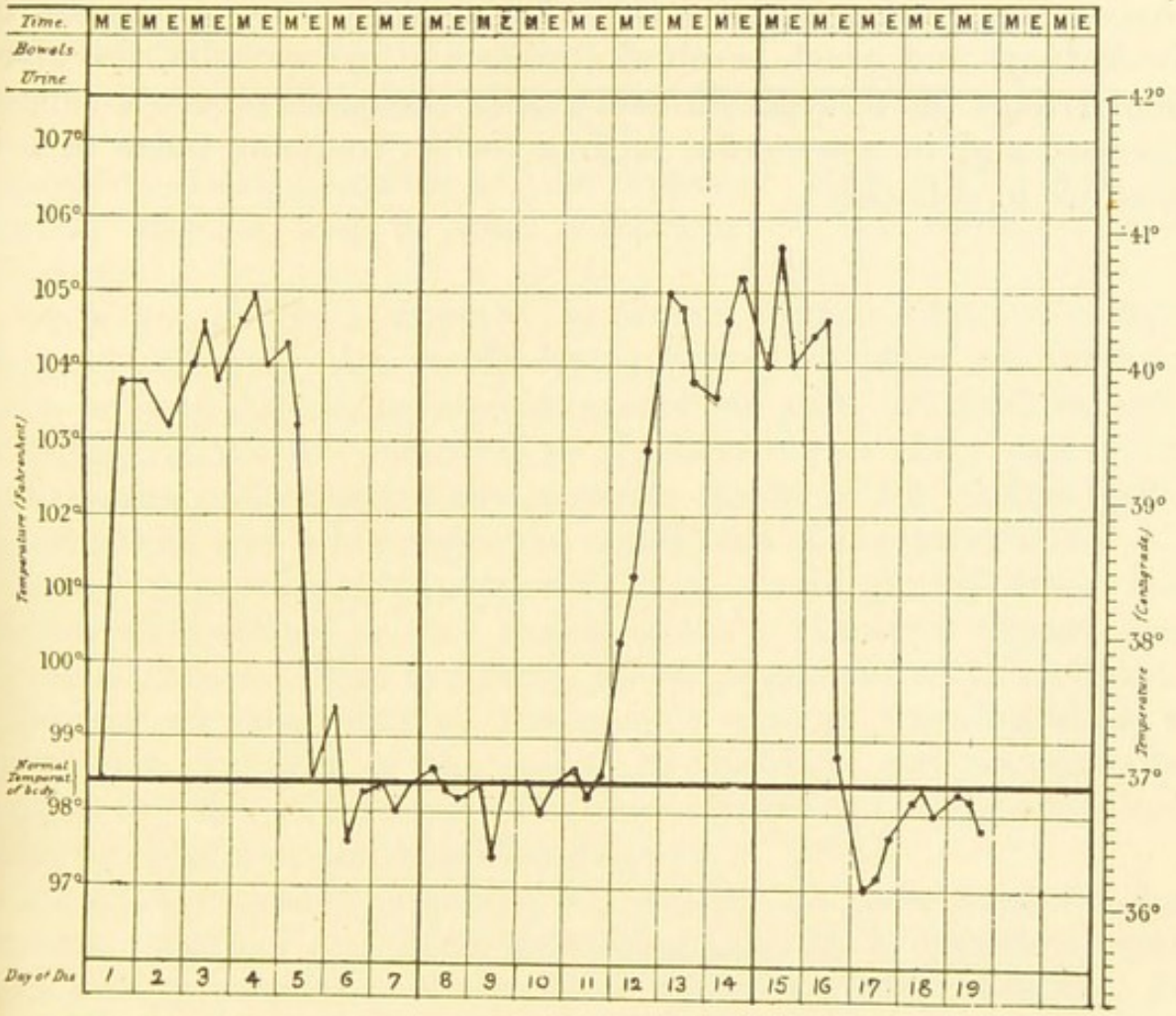


FIG. 10.—Temperature chart in relapsing fever (after Murchison).

species of parasite infecting the blood. In tertian fever there is a febrile attack every forty-eight hours (Fig. 7); if there be infection with two generations of tertian parasites there is an attack every day (double tertian fever, see Fig. 8); in quartan fever the febrile attack ensues every seventy-two hours (Fig. 9).

(5) *Relapsing or Recurrent Type.*—After a rigor the temperature rises to a high point, near which it remains for some days, and then falls by crisis to normal. An interval of apyrexia 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 (Fig. 10). These relapses may occur several times before the disease ceases.

Hyperpyrexia is the condition in which the temperature rises to 106° F., or to a still higher level.

In all febrile conditions, the pulse and respiration are increased in frequency, the appetite and digestion are impaired, headache and other cerebral phenomena are usually present, and there are changes in the blood and urinary secretion.

Subnormal Temperature.—This is observed as a constant phenomenon in many patients suffering from chronic disease of the kidneys and heart, cerebral abscess and melancholia, also after hæmorrhage (as in typhoid fever), after the crisis of acute febrile diseases, and in association with a feeble, frequent pulse of low pressure in collapse.

CHAPTER II

ALIMENTARY SYSTEM

SUBJECTIVE PHENOMENA OF DIGESTION

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 tape-worms in the intestines. The symptom 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 various 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 is an almost constant symptom whenever there is fever. 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 nervous dyspepsia there is sometimes 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 soon after food has entered the stomach. Hypersecretion 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 which arise after food is swallowed, and 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 in the epigastrium, and is increased by external pressure. In gastric ulcer there is not infrequently pain also in the back on the left side from the level of the tenth dorsal to the first lumbar vertebra.

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 tabes dorsalis.

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 epigas-

trium. It is due to abnormal digestion, and is frequently accompanied by acid eructations.

Flatulence and Eructation.—The stomach and intestines normally contain gases, namely swallowed air, and, especially in the intestines, gases resulting from fermentation. The excessive collection of gas in the stomach and bowels may result either from the swallowing of air in excessive quantities, or from abnormal fermentative changes in the contents of the viscera. Flatulence usually arises as a result of perversion of the digestive process in some of its various actions. Gaseous distension of the stomach and intestines occurs most readily when there is defective muscle tonus of their walls, as in peritonitis, 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 compressing 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, constituting the condition known as *water-brash*. If the regurgitating gastric contents be acid, we speak of *pyrosis*.

Nausea and Vomiting.—The expulsion of the contents of the stomach in vomiting is caused by the forcible contraction of the abdominal muscles, diaphragm, etc., 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 uræmia is due to a like cause. Perhaps the vomiting seen in the commencement of fevers may

also be thus explained, the toxins acting on the centre; (3) The centre may also be irritated by compression, a common result of the increase of intracranial 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, it seems probable that a stimulus may pass through the branches of the trigeminus to the reflex centre and cause 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 vomiting arises from cerebral causes, these premonitory symptoms are frequently absent.

Examination of the vomit (see p. 84) often yields important indications as to the activity of gastric digestion; and the detection of abnormal substances in the vomit is frequently of aid in the diagnosis of gastric diseases.

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 subside. 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, etc.

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 two or three 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, etc.

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, etc., looked for.

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

Constipation may result from—

(*a*) 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.

(*b*) Defective peristaltic action.

(*c*) 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 toxins 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 toxins 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.

3. Pathological conditions of the mucous membrane frequently lead to the pouring out of much secretion into the bowels. This group includes intestinal catarrh, all the grave affections of the intestinal tract, and also many general diseases, as cholera, typhoid fever, dysentery, and tuberculosis.

The examination of the fæces is described on p. 86.

EXAMINATION OF THE MOUTH AND FAUCES

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

(1) 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. This 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 below the normal standard, as in chlorosis and anæmia, or as in many cases of Bright's disease.

(2) 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

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

respiration, although frequently due to more or less complete obstruction of the nasal passages, as by adenoids, 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.

(3) 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 an example of this. The opposite condition is seen in the expressionless appearance which occurs in double facial paralysis, and in certain cases of muscular dystrophy.

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. This is very frequently a manifestation 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. 11). They are shorter and narrower

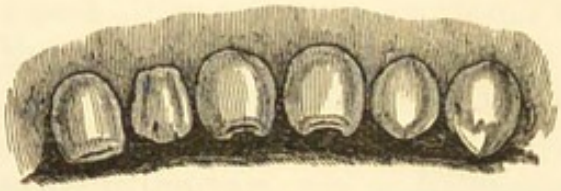


FIG. 11.—Shape of teeth in congenital syphilis (after Hutchinson).

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, etc. 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, Palate, and Mucous Membrane of the Cheeks.—The form of the palate should be noted. It may be highly arched, like an inverted V, or Gothic arch, and is then usually narrow from side to side. Or it may be V-shaped in the horizontal plane, the two aveolar processes forming an acute angle at the incisor teeth. Such defects may be associated with adenoids and mental deficiency.

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 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.

Atrophy of the tongue is produced by destructive lesions of the nucleus of the hypoglossal nerve, or of its fibres.

(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, and in bulbar paralysis.

Paralysis of the tongue is frequent in cerebral affections (hemiplegia from hæmorrhage, embolism, etc.), bulbar paralysis, and general paralysis of the insane. 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 temperature. 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 *débris* of food, of cast-off epithelial cells, and of masses of micro-organisms. The fungiform papillæ 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. The fur collects most where the tongue is roughest, and where the movement is least in amount—*i.e.*, the centre and posterior 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, etc.) paralysing the lingual muscles.

The mucous membrane of the mouth affords a nidus for other forms of vegetable organisms such as *Saccharomyces albicans* (*Oidium albicans*), the parasite found in that form of stomatitis known as thrush. This disease is usually met with in ill-nourished, bottle-fed infants, especially when ill-cleansed bottles

[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.

are in use, but also in the late stages of phthisis, in typhoid fever, etc. The affection is observed on 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 we find, in the midst of a mass of epithelium and salivary corpuscles, the fungus, consisting of long, segmented, branching filaments and shorter ovoid elements resembling yeast cells. In the meshes of the mycelium which the filaments form there are usually to be seen numerous free spores which are spherical and highly refractile.

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 other affections—mucous patches, syphilitic fissures, and ulcers are frequently met with, and cancer of the tongue is not uncommon.

The sense of taste will be considered under the nervous system (see p. 379).

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 nose or 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 bronchiectasis and gangrene.

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. Normal saliva is mixed with epithelium and leucocytes, and contains various bacteria, notably *Leptothrix buccalis*, but also pneumococci, *Micrococcus tetragenus* and various

bacilli. A small quantity of albumin is present in health, and may be much increased in disease. Certain medicines, in particular, the salts of iodine (see p. 83) and bromine, when administered internally, appear in the saliva. 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, tonsillitis, etc.
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 atropine.

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. If the fauces be hyperæsthetic, it is advisable to spray the throat with 10 per cent. solution of cocaine prior to making the examination.

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, tuberculous 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 may quickly spread 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 diphtheritic 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.

The differential diagnosis is best made 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. The bacteriological examination is considered in Chap. XXXVI.

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 the paralysis may also affect the ocular muscles, and 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 from the vago-accessory fibres. Occasionally the paralysis of the palate is of supra-nuclear origin, as in

hemiplegia, but more frequently it is due to some affection of the nucleus or of the nerve fibres below the nucleus. 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 muscles of the palate, 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 movements of the tongue are affected in paralysis of the hypoglossal nerve, for example, in bulbar paralysis. 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 (*trismus*), 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. The tonic spasm of the masseters is readily felt if the forefinger be passed along the inner sides of the cheeks.

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 malaria. 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, etc., 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.

The second stage of deglutition is interfered with in paralysis of the soft palate, food passing into the posterior nares. This 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 may occasion such dyspnœa as to require its removal by means of the finger, and fluids pass readily into the larynx. This paralysis 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, etc.), 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

(1) Palpation of the neck may yield information regarding a tumour or diverticulum of the œsophagus, or as to the presence of a foreign body within it.

(2) By Means of the Œsophageal Bougie, or the Stomach Tube (No. 20 or 21). The latter is the more satisfactory instrument. The patient should be seated, and his clothing should be protected by waterproof sheeting fastened around the neck. The instrument, having been washed in warm water and dipped in milk, is held in the right hand as one holds a pen, passed gently over the back of the patient's tongue to the posterior pharyngeal wall, 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 or 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—

(*a*) *Pain.*—If the pain be felt again and again at the same spot, it indicates a local process, probably of an inflammatory nature.

(*b*) *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 stomach tube was arrested should be noted.

When the tube has been withdrawn, it should be carefully examined for traces of blood or fragments of tissue adhering to it or blocking the eyelet. Tissue fragments should be preserved in 5 per cent. formalin for the purpose of microscopic examination. The presence of ulceration may be conjectured if the tube, however carefully it has been introduced, always comes away smeared with blood.

(3) Auscultation of the Œsophagus.—The stethoscope is to be applied a little to the left of the spinal column in the cervical or upper 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, œsophageal obstruction exists, this sound is prolonged, and altered in character below the seat of the stricture. This method is of lesser clinical value than examination by means of the stomach tube.

(4) Œsophagoscopy, or the inspection of the œsophagus by means of a special instrument and electric illumination, is also of little clinical value.

(5) Radiography is of aid in the localisation of foreign bodies and tumours of the œsophagus.

CHAPTER III

Alimentary System (*continued*)

EXAMINATION OF THE ABDOMEN

The abdominal surface is arbitrarily mapped out by two vertical and two horizontal lines (see Fig. 12). (1) The right and left *mammary lines* each pass from the middle of the clavicle to the mid-point of Poupart's ligament. (2) The *infracostal line* passes horizontally on a level with the lowest points of the tenth costal cartilages. (3) The *bi-iliac line* joins the most prominent points of the two iliac crests. These four lines demarcate the nine regions of the abdomen, as shown in Fig. 12.

The natural lines of the abdominal surface are (1) the *linea alba*, in the middle line, from the ensiform cartilage to the symphisis pubis; (2) the two curved *lineæ semilunares*, each passing from the ninth costal cartilage to the pubes, and corresponding to the outer border of the rectus abdominis muscle; and (3) the three *lineæ transversæ*, of which one is at a level with the tip of the ensiform cartilage, a second midway between the ensiform and the umbilicus, and a third at the level of the umbilicus.

The umbilicus is on a level with the disc between the third and fourth lumbar vertebræ, and lies about an inch and a half above the bi-iliac line.

The physical examination of the abdomen can be satisfactorily made only when the abdomen is completely exposed, and it is made with greatest advantage when the patient is in the recumbent posture. It is well to have the shoulders slightly elevated by means of a low pillow, so as to relax the abdominal muscles, which can be still further effected by causing the knees to be raised, and by directing the patient to breathe quietly, so that the glottis may be kept open. The examination is conducted by means of inspection, palpation, percussion, and auscultation, each of which will be considered in turn.

Further evidence is sometimes obtained by examination of the

patient in the erect, lateral recumbent, and genu-pectoral postures.

INSPECTION OF THE ABDOMEN

On inspection of the abdomen, we may notice, instead of the normal contour: (1) General Prominence; (2) General Retraction; (3) Local Prominence. We must further observe if there be (4) Any Abnormal Appearances of the Skin, and (5) The Movements of the Parieties.

I. **General Prominence** of the abdomen is usually due, as is said by Wyllie, to *fat, fluid, flatus, fœtus, or fæces.*

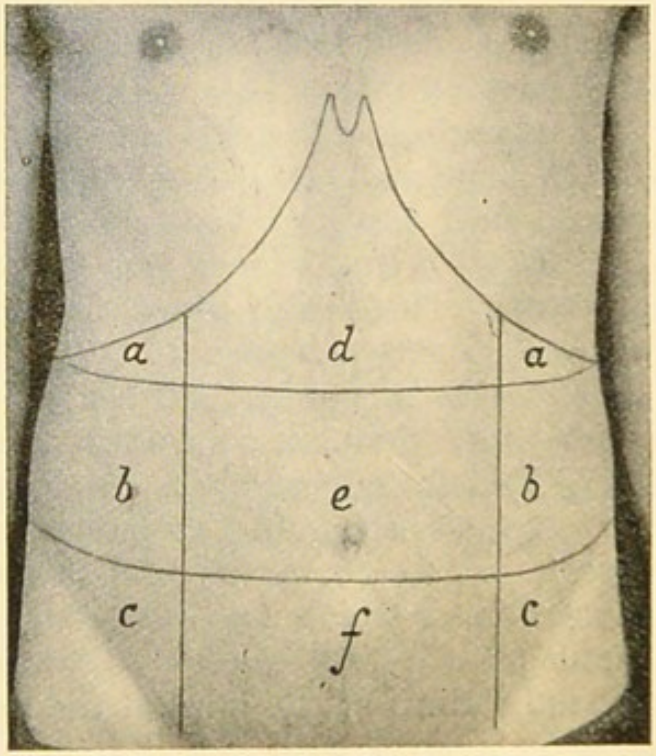


Fig. 12.—Regions of the abdomen.

a, Hypochondriac regions; *b*, Lumbar regions; *c*, Iliac regions; *d*, Epigastric region; *e*, Umbilical region; *f*, Hypogastric region.

1. Fat.—A full dietary and a corpulent habit cause general prominence of the abdomen, yet the umbilicus remains depressed below the surrounding level. The excess of subcutaneous fat is recognised when a fold of skin from the anterior abdominal wall is pinched up between the fingers. Pressure of the finger does not cause pitting, and the condition is thus readily distinguished from œdema of the abdominal wall.

2. Fluid.—(*a*) *Œdema* of the abdominal wall. The swelling is more obvious in the flanks and hypogastrium; the pressure of the

finger causes pitting; and there is œdema in other parts of the body.

(*b*) *Accumulation of fluid in the peritoneal sac.* Fluid when free in the peritoneal sac 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.

By the history of the patient, and by further examination, we determine whether the fluid accumulation is the result of dropsy (*ascites*) or is an inflammatory effusion, as in tuberculous peritonitis.

3. 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; whereas obstruction to the portal circulation occasions distension of the veins radiating around the umbilicus—this varicose condition (*Caput Medusæ*) 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,—in the iliac colon or in the rectum,—the whole of the large intestine, and often the small intestine also, presents an appearance of tumidity, shows peristaltic movements (passing, in the case of the colon, from right to left), 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, and because of the visible peristalsis in the small intestine, the abdomen then presents what Wyllie calls the "ladder pattern."

When the obstruction is at the pylorus the stomach only is distended, and it may show visible peristaltic movements, passing from left to right, its contour standing out in relief during the rigid spasms into which it is thrown from time to time. The waves of gastric peristalsis can in some instances of pyloric obstruction be elicited by sharp percussion, or by palpation, over the region of the stomach.

II. **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 tuberculous meningitis), constituting the scaphoid or boat-shaped abdomen, and is then attributable to contraction of the intestines, determined by the irritation at the base of the brain.

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

III. **Local Tumefaction** may be observed in the various regions of the abdomen. Swelling in the *epigastrium* is usually due to dilatation of the stomach, the swelling being then of oval outline, and extending chiefly towards the left. Visible epigastric prominence may also be due to tumours, usually carcinomata, of the stomach, and especially of the pyloric region, or to tumours of the left lobe of the liver. We must accordingly ascertain whether the patient's symptoms are indicative of disease of the stomach or liver, and must determine by palpation (see p. 55) with which of those organs the tumour is connected. Swelling in the epigastrium is occasionally caused by a tumour of the transverse colon, tumour or cyst of the pancreas, aneurism, etc.

Visible swelling in the *right hypochondrium*.—In the healthy adult the liver gives rise to no visible prominence of the abdominal walls. When hepatic 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, a tumour arising from that organ, or a distended gall bladder, when visible, may be seen to rise and fall with the respiratory

movements—an important diagnostic point. In infants 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.

Swelling in the *left hypochondrium* is less frequent, and is usually caused by dilatation or carcinoma of the stomach, splenic enlargement, carcinoma of the splenic flexure, or tumour of the left kidney. Swelling in the *umbilical region* is often due to gastroptosis (see p. 54), tuberculous disease of the mesenteric glands, and omentum (tabes mesenterica), tumour of the intestines, or retro-peritoneal tumour. Prominence in the *hypogastrium* is frequently caused by the enlargement of the pregnant uterus, by uterine fibroid tumour, and most frequently by a distended bladder. The possibility of a swelling visible or palpable in the lower part of the abdomen being due to a distended bladder must always be borne in mind. Swelling in the *right lumbar region* may be due to enlargement of the right kidney, as from hydronephrosis, tuberculous disease or tumour of the kidney, or to affections of the right lobe of the liver, as carcinoma or echinococcus cyst. Swelling of the *left lumbar region* may be caused by enlargement of the spleen, or by hydronephrosis, sarcoma, or other disease leading to great enlargement of the left kidney. A swelling in the *right iliac region* may be due to a variety of causes:—fæcal accumulation in the cæcum, appendicitis (abscess or chronic inflammatory thickening), malignant disease or tuberculosis of the ileo-cæcal region, intussusception, ovarian and parovarian cysts, pelvic abscess, and uterine fibroid tumour. In the *left iliac region*, a swelling may be due to fæcal accumulation, carcinoma of the iliac colon, volvulus, and tuberculous peritonitis, or to ovarian cyst or other morbid condition originating within the pelvis, as in the case of the right iliac region.

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

By inspection alone, we are seldom able to determine the nature of the localised tumefaction. We must carefully inquire when the swelling was first detected. Has the increase in size been continuous since then, or does the swelling vary in size from time to time (as, for example, in hydronephrosis)? Is there local pain, and, if so, to what part of the abdominal surface is the pain referred? Is the pain constant, or, if intermittent, how frequently does it recur, and is there any relation between the onset of pain

and the ingestion of food (as in carcinoma of the stomach), or movement on the part of the patient (as in the case of a distended gall-bladder containing gall-stones)? We must also inquire as to the character of the pain, whether dull and aching, burning, stabbing, etc. Careful inquiry regarding the previous health of the patient, the sexual history, if the patient be a female, and the detection of emaciation, jaundice, anæmia, or fever will often throw light on the nature of the swelling, but the most reliable information will be obtained by employment of other means of examination, and in particular that of palpation.

IV. **Abnormal Appearances of the Skin of the Abdominal Wall.**—Œdema and distension of the superficial veins, and the pigmentation in Addison's disease and in Tinea versicolor have been already considered (see p. 8). During pregnancy, pigmentation is observed, especially along the middle line, constituting the *linea nigra*. In certain infective diseases, the eruption may be most marked over, or even confined to, the abdomen. This is true of the rose spots appearing in successive crops in typhoid fever, and in many instances of the dusky red macular mottling seen in secondary syphilis. Striæ albicantes should be noted if present. They indicate antecedent abdominal distension (pregnancy, ascites, etc.).

V. **Abdominal movements** are (*a*) Respiratory, (*b*) Pulsatory, and (*c*) Peristaltic.

1. Respiratory Movements.—In health the upper part of the abdominal wall rises with inspiration and falls with expiration, the extent of movement being greater in the adult male than in the female patient. If the diaphragm be paralysed, the respiratory movements of the abdomen are reversed, there being a fall with inspiration and a rise with expiration.

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 lesser degree. Tumours of the stomach are not as a rule affected in this manner, unless adhesions to the liver or diaphragm have been formed. Tumours of the pancreas, intestine, omentum, and kidney do not move with respiration (see pp. 132, 133).

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

3. Peristaltic Movements may be seen in connection with the stomach (see p. 44) or intestines. Those of the intestines may be occasionally observed in persons in whom the abdominal walls

are abnormally thin, but more distinctly when the vermicular peristaltic movements are excessive, as in cases of chronic intestinal obstruction.

Having completed our inspection of the abdomen, we take an accurate **measurement** of the abdominal girth at the level of the umbilicus. Subsequent measurements will afford definite proof of increase or decrease in the size of the abdomen. Measurements are of much importance in cases of ascites, tuberculous peritonitis, etc.

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 a solid growth or fluid accumulation; 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 any necessity for it, the administration of a general anæsthetic will allow of a perfect exploration of the abdominal cavity.

The examining hand must be thoroughly warm. With the fingers approximated to one another, and in a position of extension,

the hand should first be passed with a gentle kneading movement over the abdominal surface in general. The regions of the abdomen are thereafter to be examined in succession, but it is well to palpate first those regions to which no pain is referred, and only thereafter to examine any painful or tender areas. The examining hand is kept in continuous contact with the abdominal surface, and should at first be allowed to rise and fall with the respiratory movements of the abdominal wall, whilst during expiration, pressure is made with the fingers, which are then to be flexed at the metacarpo-phalangeal articulations. All movements of the nature of tapping with the finger tips are to be avoided. Thereafter it is advantageous, in order, for example, to determine the mobility of an organ or tumour, to maintain firmer pressure with the hand during inspiration and expiration. Further information may be obtained by "dipping" movements, in which the fingers, which have been in close contact with the abdominal wall during the expiratory movement, are quickly depressed at the end of expiration, namely, when the parieties are most completely relaxed. That manipulation sometimes enables us to detect a deep-seated tumour more readily than by any other method, and is also useful in eliciting splashing sounds within a dilated stomach or colon. Palpation with one hand having been completed, we proceed to:

Bimanual Palpation.—To examine the right side of the abdomen, we sit on a low chair or kneel by the patient's right side, and palpate with the right hand laid on the anterior abdominal surface, the left hand meanwhile pushing up the posterior wall between the last rib and the iliac crest. When examining bimanually the left side of the abdomen, we sit by the patient's left side; our left hand is placed on the anterior, and our right hand on the posterior wall of the abdomen. Any solid body, such as a movable kidney, tumour of the colon, or masses of tuberculous glands, can usually be more readily detected by this method than by palpation with one hand alone.

PERCUSSION OF THE ABDOMEN[1]

When the dorsal surface of a finger of the left hand, laid on the umbilical region, is sharply struck by the middle finger of the right hand, a resonant sound is elicited from the underlying air-containing viscera, and the sound, as it resembles that obtainable from a drum, is termed tympanitic. If, however, we percuss over a solid organ such as the liver, we elicit a dull sound. The

[1] The theory of percussion will be considered in Chap. XVIII. (see p. 246).

change of sound as we percuss from the air-containing viscera towards and on to the liver dulness is usually sufficiently obvious. It is by such change of percussion sound that we define the outlines of the liver and spleen which, when healthy, it is not possible to ascertain by means of palpation. The pitch of the tympanitic note yielded by an air-containing viscus (see p. 256) depends on the size of the air-cavity, and the tension of the wall of the cavity. By ascertaining the variations in pitch of the percussion sounds as we percuss over the different regions of the abdomen in a given patient, we may consequently be able to demarcate the boundaries of the stomach, colon, and small intestine. It is important to remember, however, that the pitch of the percussion note of the stomach (and likewise that of the colon and small intestine) is by no means constant, but varies according to the distension of the viscus, and that the note obtained over the stomach is not infrequently indistinguishable from that of the transverse colon.

Three points of importance regarding the technique of percussion of the abdomen should be remembered:—*Firstly*, the finger (or pleximeter) receiving the percussion stroke must be laid parallel with the boundary we are seeking to define; *secondly*, we should percuss towards the organ the outline of which we wish to ascertain; and, *thirdly*, the force of the percussion stroke must be light, the only exceptions being on percussion of the upper (deep) borders of the liver and spleen.

AUSCULTATION OF THE ABDOMEN

This is mainly of value—

1. In the diagnosis of pregnancy. Auscultation over a point midway between the umbilicus and the left anterior iliac spine may permit the detection of the fœtal heart sounds;
2. In the diagnosis of aneurism of the abdominal aorta and its branches, which will be considered in connection with the circulatory system; and
3. In combination with percussion (auscultatory percussion) as a means of determining the size of the stomach (see p. 57).

EXAMINATION OF THE 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 p. 11) 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 affections 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 as to whether the swelling be 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 may be mistaken by the inexperienced for abdominal tumours. It is important to determine whether the swelling becomes more readily palpable, or less so, when the patient raises the head from the couch. When he does so, a thickening of the rectus is rendered more obvious, whereas an intra-abdominal tumour becomes less distinct.

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

THE PERITONEAL CAVITY

I. **Palpation.**—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 the 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 less acute localised peritonitis a characteristic doughy resistance is usually to be felt over the affected part, accompanied by some tenderness on pressure. These signs of localised peritonitis are most frequently observed in the right iliac region, as a result of appendicitis.

To detect by palpation the presence of **free fluid in the peritoneal cavity**, we must ascertain whether an *undulatory thrill* can be transmitted across the abdomen. The thrill is best appreciated by placing the palmar aspect of one hand firmly on one lumbar region, and giving a smart tap on the abdominal surface at a point diametrically opposite, while firm pressure is meanwhile made along the middle line of the abdomen by the ulnar edge of the hand of a third person, in order to prevent

transmission of any thrill along the fat of the abdominal wall. The impulse of the wave thus formed in the peritoneal fluid can usually be clearly felt when it reaches the opposite wall. If, however, the amount of fluid be small, no such undulatory thrill will be felt in the ordinary position. The patient may then be placed on his knees and elbows, when the fluid will gravitate to the anterior part of the sac, and the thrill may then be obtained.

Friction vibration can 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.

II. **Percussion.**—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 eases, where the sac is greatly distended with fluid 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 (Fig. 13). 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 encysted fluid, for example, an ovarian cyst.

It is not hard to distinguish ascites from meteorism, in which there is neither lateral dulness altering with position, nor the undulatory thrill mentioned on p. 50, 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

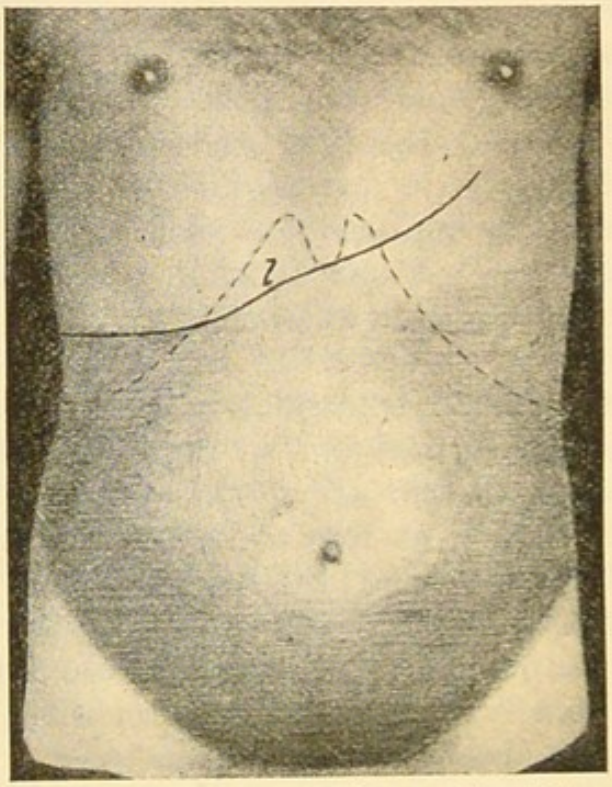


FIG. 13.—Ascites : the shaded area indicates the dulness. The liver (*l*) is displaced upwards.

by the position of the dulness, and, above all, by the auscultation of the fœtal heart.

The main points of difference between ascites and ovarian cystic tumour are given in the following table :—

ASCITES.	OVARIAN CYST.
1. *History.* No history of lateral development.	Tumour develops from one iliac fossa.
2. *Inspection.* 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.
3. *Percussion.* On percussion there is dulness in the flanks, and a clear note over the centre of the abdomen. 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. Change of position does not alter the line of dulness.

Examination by palpation and percussion, as described, enable us to detect the presence of free fluid in the peritoneal cavity. As the fluid may represent either a dropsical transudation, or an inflammatory effusion, we must base our diagnosis on the history of the patient, and on evidence of the primary disease—renal and cardiac disease, cirrhosis or carcinoma of the liver, tuberculous peritonitis, etc. In tuberculous peritonitis, palpation often enables us to detect areas of resistance or even definite solid masses of irregular form (tuberculous mesenteric glands, or tuberculous omentum) which are never felt in ascites.

THE STOMACH

Topographical Anatomy.—The stomach lies in the left hypochondrium and left half of the epigastrium. The *cardiac orifice* is situated four inches beneath a point corresponding to the seventh left costal cartilage, one inch from the edge of the sternum. The *pyloric orifice* of the empty stomach lies on a level with the first lumbar vertebra, one inch to the right of the mesial plane, and midway between the base of the ensiform cartilage and the umbilicus. The *fundus* rises to the level of the fifth rib in the mammary line; the *greater curvature* of the normal, distended stomach lies (in the mesial plane) at the junction of the lower and middle thirds of a line joining the base of the xiphisternum and the umbilicus.

The stomach, it must be remembered, is of constantly varying form, in accordance with the degree of its distension with food and gas. When empty, the stomach probably lies almost horizontally, with its long axis directed forwards and to the right, and is entirely under cover of the ribs, costal cartilages, and the left lobe of the liver. But when moderately distended, the stomach protrudes from beneath the left costal margin and the left lobe of the liver, and comes in contact with the anterior abdominal wall, whilst the pylorus passes to the right of the mesial line.

Inspection of the Stomach

The normal stomach, even when distended, causes no visible swelling of the abdominal surface.

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 the gastric tumour mass, having contracted adhesions to neighbouring parts, moves synchronously with the diaphragm. Such movements can be much more accurately studied by means of palpation (see p. 55).

Visible Peristalsis of the stomach (see p. 44) is always suggestive of pyloric stricture, whether due to simple cicatricial stenosis, tumour of the pylorus, or other cause.

Dilatation of the Stomach often 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 left costal margin downwards and to the right. In Gastroptosis—downward dislocation of the stomach—there may be a localised oval or crescentic swelling in or below the umbilical region.

Artificial Inflation of the Stomach.—The form and position of the stomach are most readily appreciated when it has been artificially inflated. This procedure is carried out by passing the stomach tube, and, having connected the free end with a Higginson's syringe, bicycle pump, or Politzer's bag, by pumping in air until the desired distension is produced, when the free end of the stomach tube may be clamped. Or Senorance's extractor (Fig. 16, p. 75) may be employed for inflation of the stomach with air. The tube having been introduced into the stomach and the aperture in the neck of the bottle being closed by a finger, the india-rubber ball is compressed, and air is thus driven into the stomach. Then successively clamp the stomach tube with the fingers, open the aperture, close it, relax the compression of the tube, and compress the india-rubber ball. This manipulation is repeated until the desired inflation of the stomach is effected, when the tube may be clamped. The advantages of inflation with air are the ease with which the degree of inflation can be regulated, and the possibility of releasing the air from the stomach at will. The disadvantage is the necessary introduction of the stomach tube.

Inflation with *carbonic acid gas* is performed by making the patient swallow successively solutions of a teaspoonful of sodium bicarbonate and of half a teaspoonful of tartaric acid. 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. Inflation by this method cannot be regulated so exactly, and is therefore not to be preferred to inflation with air.

By inflation of the stomach its form and position are rendered distinct; whilst in a case of gastroptosis the low situation of both

the greater and the lesser curvature may be rendered evident, especially if the patient be in the erect posture.

Inflation of the stomach is also of assistance in palpation and percussion of the viscus. The conditions which contra-indicate inflation of the stomach are marked asthenia, recent gastric hæmorrhage, cardiac disease, and aortic aneurism.

Palpation of the Stomach

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

The abdomen should be palpated with a view to the detection of a TUMOUR OF THE STOMACH. Carcinoma, the most frequent tumour, is more often at the pyloric end of the stomach, and when the tumour has attained moderate dimensions it can usually be felt in the epigastric region. But every tumour palpable in that region is not necessarily a gastric carcinoma. We must therefore endeavour to prove that the tumour is one of the stomach, and not of the transverse colon, duodenum, liver, gall-bladder or pancreas. The pyloric carcinoma is felt as a somewhat globular or irregular nodular mass, tender to pressure, freely movable for the most part, and but little affected in position by the rise and fall of the diaphragm, though this last feature is not reliable, for if the tumour has become adherent to the liver it moves with respiration. If a pyloric tumour, which has not contracted adhesions, be grasped between the fingers it may be prevented from passing upwards on expiration, whereas a tumour of the liver or gall-bladder or a tumour of the stomach or colon which is adherent to the liver cannot be prevented in that manner from passing upwards during expiration. The connection of the tumour with the stomach can sometimes be most satisfactorily determined by palpation of the stomach when it is inflated according to the method already described.

The absence of palpable tumour does not exclude the possibility of carcinoma of the stomach. No tumour can be felt in the early stages of the disease. A carcinoma of the lesser curvature or the cardiac end of the stomach is seldom palpable, as it lies deeply in the concavity of the diaphragm, under cover of the ribs.

We next proceed to ascertain whether gurgling or **splashing sounds** (succussion sounds) can be elicited within the stomach. The sounds, the *timbre* of which is characteristic, are produced when a cavity containing fluid and air is shaken. In some persons in whom the stomach is normal, splashing can be elicited during

the period of digestion over the area of the normal stomach. Splashing, however, must be regarded as a sign of disease if it can be elicited six or more hours after a meal (when the stomach should be empty), for example, in the morning before breakfast, or over a wider area than that corresponding to the normal stomach. Under such circumstances, splashing indicates either motor insufficiency or dilatation of the stomach, two conditions which usually co-exist.

To examine for succussion sound, the physician, being on the patient's right side, places one hand over the epigastrium, and the other over the left hypochondrium, and, during expiration, makes quick, sudden, dipping movements with the fingers. Another method is to strike, not too forcibly, the anterior abdominal wall along the mesial line successively from below upwards with the ulnar edge of the hand or the tips of the fingers, splashing being elicited when the hand strikes the abdominal wall over the stomach. Succussion sounds which are inaudible by either of these methods, because of rigidity or excessive adiposity of the abdominal wall, may, however, be detected by auscultation over the region of the stomach, whilst the examination, as described, is being performed. The succussion sounds obtained from a colon distended with fluid and gaseous contents may be mistaken for gastric splashing.

Percussion of the Stomach

When the stomach is filled with food, it is impossible to define its boundaries by means of percussion; but when the cavity of the viscus is moderately distended with air, it gives on percussion a tympanitic note of low pitch, and long duration, which, though varying according to the degree of distension of the stomach, may yet be 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, its walls, on percussion, are also thrown into vibration, and a metallic ring results, which renders the definition of the gastric outlines more difficult.

Method.—By percussion we can define only that portion of the stomach which is in contact with the abdominal or thoracic wall. Light percussion is required, and the patient must be in the supine position. First ascertain the stomach note by percussing over the left half of the epigastrium close to the left costal margin, or preferably over the seventh interspace or eighth

costal cartilage in the left mammary line. Then percuss along imaginary lines converging from various parts of the abdomen and the left axilla towards the stomach, and mark the points at which a percussion note identical with the stomach note is first obtained. Then confirm the accuracy of the outline of the stomach, as indicated by those points, by percussing from the stomach towards the surrounding viscera. The difference in pitch of the percussion note of the stomach from that of the intestines can usually be more distinctly recognised by means of *auscultatory percussion*, namely, by auscultating over the left half of the epigastrium, close to the costal margin, or over the seventh left intercostal space in the mammary line, whilst making light percussion towards the stomach, as described.

We thus ascertain the boundaries between the stomach on the one hand and the intestines, liver, lung and spleen on the other. Though the percussion outline of the stomach is constantly varying, **the lower boundary** of the moderately distended stomach usually lies, in the left parasternal line, from 3 to 7 cm. (but not less than 3 cm.) above the level of the umbilicus. **The upper boundary** is at the fifth intercostal space, in the left parasternal line — at the sixth rib, in the left mammary line — at the seventh or eighth rib in the left anterior axillary line. The resonant note of the stomach does not extend to the right of the mesial line.

Gastrectasis.—If the stomach be dilated, its percussion outline will extend beyond those limits. When the lower border of the stomach is below a transverse line drawn 3 cm. above the umbilicus, the organ may be assumed to be dilated. Dilatation of the stomach may indeed sometimes be so great that its lower boundary may reach almost to the pubes. The right boundary will then lie to the right of the mesial line.

The lower boundary of the stomach is displaced downwards not only in gastrectasis but also in **gastroptosis**. Further, it must be remembered that gastric dilatation is not uncommon in cases of gastroptosis. We must not be content, therefore, with having determined the position of the lower boundary of the stomach, but must percuss out its upper boundary too. In gastroptosis both upper and lower boundaries are displaced downwards; in gastrectasis the upper boundary is in normal position.

Traube's Semilunar Space is the tympanitic area bounded below by the left costal margin, on the right by the liver dulness, above by the convex margin of the left lung, and in the left axilla by the anterior margin of the spleen. The tympanitic note

over Traube's space is due to the fact that normally the stomach, or stomach and intestines, are there in contact with the thoracic wall. In a left-sided pleural effusion Traube's space is usually encroached on from above, owing to accumulation of fluid in the complementary sinus of the left pleura (see p. 219).

Gastrodiaphany or Electric transillumination of the stomach.—The patient is examined in the erect posture, in a darkened room and when the stomach is empty of food. The gastrodiaphane, the instrument employed, is a flexible tube with an incandescent electric lamp at the lower end; above the lamp is the lower aperture of the tube, and at its upper end are the two screws for

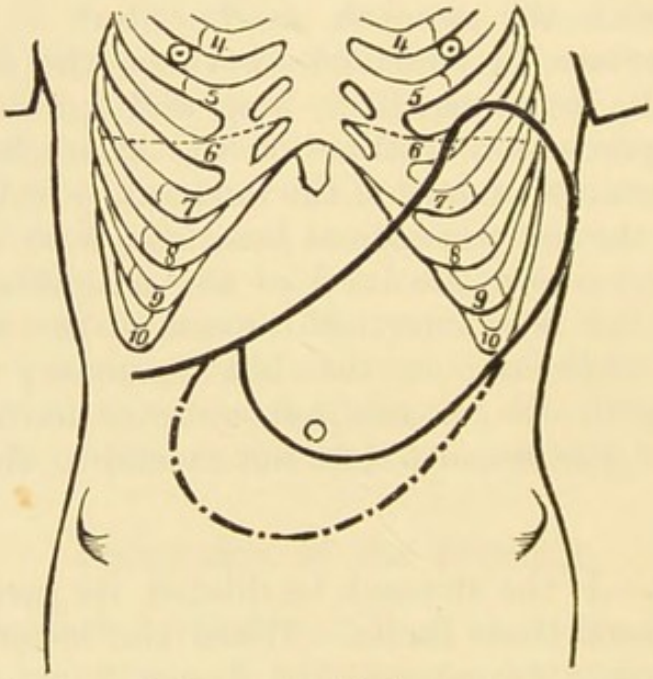


FIG. 14.—Area illuminated by gastrodiaphany: ——— when the normal stomach is empty; — - — - — when normal stomach is filled with water (after Meltzing).

attachment of the wires. The tube is passed into the stomach, about a quart of water is introduced through the tube, and the site and extent of the luminous area of the anterior abdominal wall is studied. Fig. 14 shows the luminous area in the case of the normal stomach when empty and when distended.

It cannot be said that the results hitherto obtained by gastrodiaphany have proved of much clinical value.

The examination of the gastric contents is discussed in Chap. IV., p. 74.

THE INTESTINES

Topographical Anatomy.—The cæcum lies in the right iliac region, the situation of the ileo-cæcal valve corresponding to a

point midway between the anterior superior iliac spine and the umbilicus. The hepatic flexure lies beneath the tenth right costal cartilage; the transverse colon, though inconstant in position, usually crosses the upper half of the umbilical region. The splenic flexure lies in the left hypochondrium behind the stomach.

Inspection, Palpation, and Percussion of the intestines have already been considered on pp. 42 to 49. We may here recall the fact that retention of fæces in the intestines, and chiefly in the large intestine, may give rise to localised swelling at various points. These nodular masses are of a doughy consistence, and to a large extent disappear after purgation. Catarrh and inflammation of the vermiform appendix and colon are apt to give rise to localised peritonitis, resulting in a swelling which is usually ill-defined, doughy, hard, and very tender on pressure. Such inflammatory swelling is frequently met with in the right iliac region. Cancerous and tuberculous masses may occasionally be felt at various parts of the colon; the cæcum and iliac colon being most commonly the seat of the disease. Peristaltic movements of the intestines are occasionally to be felt when the abdominal walls are thin, but much more distinctly when the movements are very energetic, as in stenosis of the bowel (see pp. 43 and 44).

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.

Gurgling or splashing sounds may be elicited within the transverse colon, and also the cæcum, when the contents are unduly fluid, and are undergoing fermentation, as in some cases of typhoid fever.

Over the intestines the percussion note is normally tympanitic, but is higher in pitch than over the stomach. When the bowels are distended with liquid or solid contents this note ceases to be heard.

Increase of resonance over the intestines is observed in meteorism. The colon may then lie in front of the right lobe of the liver, and the lowest limit of hepatic dulness may thus be considerably above the costal margin in the right mammary line. A similar diminution of the hepatic dulness may result from the presence of free gas in the peritoneal cavity, as after perforation or rupture of one of the hollow abdominal viscera.

The position and size of the colon is most satisfactorily ascertained by means of auscultatory percussion. As an aid to diagnosis the colon may previously be inflated with air by means of a Higginson's syringe introduced into the rectum.

Examination of the Rectum.—The readiest method is that by digital examination. The patient lies on the left side with the knees flexed on the abdomen. The examination is performed with the right fore-finger, which should be protected by means of a thin india-rubber finger-stall, failing which, soap is filled in under the free edge of the nail and the finger is smeared with vaseline. With the left hand the physician separates the gluteal folds, and he then inspects the anal region, noting the presence of hæmorrhoids or other abnormality. The lubricated fore-finger is then slowly, and with a rotatory movement, introduced through the anal canal, in which it should be firmly gripped. The finger is thereafter directed backwards and upwards so that the whole of the mucous surface within reach of the finger may be examined. Any tenderness is noted, likewise the presence of scybalous masses, tumours, ulcers, stricture, or polypi. The finger cannot detect internal hæmorrhoids. When the finger has been withdrawn it should be examined for any trace of blood, pus, etc., adhering to it.

Digital examination of the rectum enables us to examine the prostate and seminal vesicles in the male, whilst in the female patient the abdomino-rectal bimanual examination is especially useful.

Other methods of rectal examination lie more in the domain of the surgeon and gynæcologist, and need not be considered here.

The examination of the fæces is considered in Chap. IV., p. 86.

THE LIVER

Topographical Anatomy.—The liver lies mainly under cover of the ribs and costal cartilages, but the left lobe is in contact with the anterior abdominal wall in the upper part of the epigastric region. The upper limit of the organ is at the level of the fourth intercostal space in the right mammary line, and at the level of the sixth chrondro-sternal articulation in the mesial line. The lower border passes obliquely upwards from the tenth right costal cartilage to the eighth left costal cartilage, crossing the mesial line at the level of the first lumbar vertebra midway between the base of the ensiform cartilage and the umbilicus.

The upper part of the right lobe of the liver is overlapped by the basal margin of the lower lobe of the right lung. The fundus of the gall-bladder is opposite the ninth right costal cartilage, just to the outer side of the rectus abdominis muscle.

Inspection.—The normal liver causes no visible prominence of the abdominal walls. Visible enlargement of the organ has been already considered (see p. 44).

Palpation of the 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 right costal margin as to offer appreciable resistance to the fingers. In infants, the liver is of such size as to be readily examined by palpation.

Glénard's Procédé du Pouce—examination by means of the thumb—is often useful in palpating the edge of the liver. The patient lies in the supine position with the shoulders somewhat raised. The physician, sitting on the right edge of the couch, and facing the patient, applies the left hand to the right hypochondriac and lumbar regions in such a manner that the fingers press forward the two lowest ribs and the liver, whilst the left thumb is abducted and palpates the anterior abdominal wall in the right mammary line. The right hand is laid horizontally over the lower part of the abdomen, so that the palm lies over the hypogastric, the fingers over the right iliac region. While firm pressure is being made, the right hand is rotated so that the fingers come to point towards the right nipple. This manipulation pushes up the intestinal coils under the liver, and if the patient now takes a deep inspiration, the edge of the organ may be felt by the thumb of the left hand.

Either as a result of enlargement or of lowered position (due, for example, to the downward pressure of a pleural effusion, or as in some cases of enteroptosis) 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 change, fatty disease, 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. Marked irregularity may also be found in tertiary syphilis of the liver.

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 the fatty liver, still more so in the congested liver, 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 cyst 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 (chronic venous congestion, waxy disease, 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 transverse furrow marking off the lower portion of the right lobe. The constricted lobe may be mistaken for a floating kidney, but is more superficial, of less symmetrical form, is not so tender on pressure, and cannot be prevented from passing upwards on expiration. Still more obviously is the shape of the liver altered by the presence of a large tumour, as a carcinoma or hydatid cyst, 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 stomach, omentum, pancreas, colon, or kidney, unless they have become adherent to the liver or diaphragm.

[1] When a hydatid cyst lying near the surface of the liver is percussed, a peculiar tremor (hydatid thrill or fremitus) may sometimes 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. A similar thrill can sometimes be elicited in ascitic fluid or in other cysts, for example, a parovarian cyst.

Occasionally the **gall-bladder** may be felt as a small pear-shaped tumour projecting from beneath the lower edge of the liver opposite the ninth right costal cartilage. 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.

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 *superficial* or *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 firmly the right side of the thorax from above downwards, the level at which impairment of the reasonant percussion sound is first detected corresponds to the level at which the subjacent air space is being encroached upon and rendered shallower by the more deeply situated liver, and hence corresponds (in the normal condition) to the highest point to which the liver reaches under the diaphragm. At this level the *deep* or *relative hepatic dulness* commences.

Method of Percussion.—*Firstly*, define the lower margin of the liver. The tympanitic resonance of the neighbouring abdominal organs, which contain air, enables us to demarcate with comparative 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 transmission of the vibrations to parts in the vicinity. We therefore percuss very lightly from below upwards, first in the mammary line, and subsequently in other vertical lines, and note the points at which the resonance becomes impaired.

Secondly, define the upper limit of the superficial dulness, which is equivalent to defining the lower margin of the right lung. Percuss very lightly from below upwards, first in the right mammary, and thereafter in the right parasternal and other vertical lines, noting the points at which the absolute dulness first gives place to a slightly resonant sound.

Thirdly, define the upper boundary of the deep dulness. Use heavy percussion, and, commencing in the second interspace,

percuss downward in each interspace in the right mammary line. Note the level at which impairment of resonance is first detected. Then percuss downwards from rib to rib in the same vertical line, and thus determine the uppermost level (whether at rib or interspace) at which impairment of resonance commences. Lastly, percuss downwards from interspace to interspace, and from rib to rib, in the right parasternal line, axillary lines, and right scapular line.

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 midway between the base of the ensiform cartilage and the umbilicus, and joining the costal margin at an acute angle in the right 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 the superficial (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 chondro-sternal articulation; in the right mammary line it corresponds to the sixth rib, and in the right mid-axillary and scapular lines it reaches respectively the eighth and tenth ribs.

The upper limit of the deep or relative hepatic dulness lies at the fourth intercostal space or fifth rib in the right mammary line; at the seventh interspace in the right mid-axillary line, and at the ninth interspace in the right scapular line.

From the clinical standpoint, the most important percussion boundaries are those in the right mammary line, namely—

The deep dulness at the fourth interspace,

The superficial dulness at the sixth rib,

The lower boundary at the costal margin.

The position of the normal hepatic dulness, superficial and deep, is indicated in Fig. 15.

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 sinus occupied by pulmonary 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 gases, or a coil of the small intestine, may be forced upwards

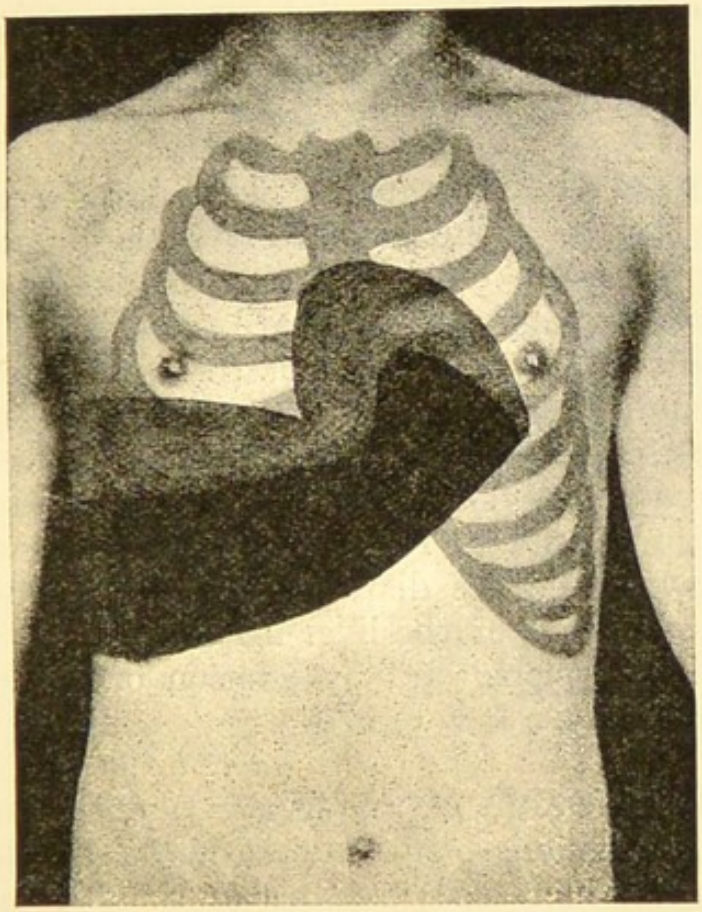


FIG. 15.—Cardiac and hepatic dulness. The lighter shading indicates the deep dulness, the darker shading the superficial dulness.

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. Similarly the hepatic dulness may be more or less abolished by the presence of free gas in the peritoneal cavity, as a result of perforation of one of the hollow abdominal viscera. 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.

Thus 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 in the right mammary line 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, etc.), but when percussion can determine the upper limit of 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 downwards than upwards. The causes of displacement **downwards** are (1) enteroptosis; (2) emphysema of the lungs, whereby both hepatic lobes are equally depressed; (3) pleuritic effusion on the right side, which causes depression of the right lobe of the liver, with perhaps slight elevation of the left lobe; (4) 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 (Fig. 13, p. 52), meteorism, ovarian cysts, etc. — and possibly also by fibroid contraction 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 cysts, carcinoma, and waxy disease. A lesser degree of enlargement is found in chronic venous congestion (as in cases of heart failure with tricuspid incompetence), occlusion of the bile ducts, fatty liver, certain cases of hepatic cirrhosis, and leucocythæmia. 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 determination of the lower border by percussion is prevented by the almost invariable presence of ascites at that advanced stage of the disease.

THE SPLEEN

Topographical Anatomy.—The organ lies obliquely in the left hypochondrium beneath the ninth, tenth, and eleventh ribs, and its long axis corresponds to that of the tenth rib. The *apex*, overlapped by the left lung, is one and a half inches from the middle line at the level of the tenth dorsal spine; the *anterior basal angle* (that portion which is most anterior) is beneath the tenth rib in the mid-axillary line. The *posterior basal angle* is in the eleventh intercostal space, four inches from, and at the level of the first lumbar spine. The *upper border* does not rise above the level of the ninth rib in the scapular line; the *posterior* or *lower border* lies beneath the eleventh intercostal space, passing from the apex to the posterior basal angle. The antero-inferior border is indicated by a line joining the anterior to the posterior basal angle.

Inspection.—It is only when the spleen is greatly enlarged that it causes visible prominence of the abdomen, and then mainly of the left hypochondrium.

Palpation.—In determining whether the spleen be enlarged, palpation is much more reliable than percussion.

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.

We sit at the right side of the patient, who lies on his back, and with one hand press forwards the tenth and eleventh ribs and the spleen. When the patient takes a deep inspiration, the spleen, if enlarged, may then be felt by the fingers of the other

hand palpating the left hypochondrium close to the costal margin.

As the organ increases in size it projects from beneath the margins of the ribs towards the umbilicus, and can be felt to rise and fall with the respiratory movements. The enlargement is proportionately the same in all diameters, and so the spleen retains its original shape. Its margin is sharp and smooth, and one or more notches, which can usually be felt on the anterior border, are important as a certain indication that the tumour with which we have to deal is splenic.

Percussion.—The patient should lie somewhat on his right side, so that the left hip and shoulder are slightly raised from the couch, and the left arm should lie across the front of the chest. If the patient lie too far over on the right side, the spleen tends to gravitate in that direction, and the splenic dulness diminishes or disappears. Percussion is made while the patient holds his breath in full expiration. In consequence of the proximity of the spleen to the large air-containing cavities of the stomach and colon, it is necessary to percuss very lightly when defining the anterior and lower limits of the splenic dulness, so that the note may not become obscured by reason of the tympanitic resonance of those organs.

(1) *To define the anterior margin.*—Commencing in the left mammary or anterior axillary line, percuss lightly backwards along the long axis of the tenth rib. The normal splenic dulness is found in the mid-axillary line.

(2) *To define the lower border.*—Commencing just above the iliac crest, midway between the posterior axillary and scapular lines, percuss vertically upwards. The splenic dulness is found at the eleventh rib.

(3) *To define the upper border.*—Commencing at the level of the angle of the scapula, midway between the posterior axillary and scapular lines, percuss vertically downwards from rib to rib, and from interspace to interspace. The deep splenic dulness is found at the ninth rib. The upper limit of the superficial splenic dulness varies according to the position of the lower margin of the lung.

Posteriorly the splenic dulness blends with the dulness of the erector spinæ muscle.

The spleen is of variable size, and, as age advances, it atrophies somewhat, and thus 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.

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 elicit.

Conditions similar to those which cause upward displacement of the liver (p. 66) 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.

Increase in size of the spleen takes place in numerous diseases, such as leucocythæmia, amyloid disease, recent syphilis, malaria, 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. 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 *consistence* 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 malaria 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.

THE KIDNEYS

I. **Topographical Anatomy**—*Anterior relations.*—The hilus of the right kidney and that of the left lie respectively two inches

to the right and one and a half inches to the left of the midpoint between the umbilicus and the base of the ensiform cartilage. The upper extremity of the right kidney is situated two inches from the middle line, at the level of the seventh costal cartilage; the lowest point is three inches from the middle line and one inch above the level of the umbilicus. The left kidney lies half an inch higher and half an inch nearer the middle line than the right kidney.

Posterior relations.—The upper extremity of the right kidney lies at the level of the eleventh dorsal spine. The lower end is one and a half inches above the crest of the ilium, three inches from the middle line. *Transversely* the kidney extends from the hilus (one and a half inches from the middle line) to a point four inches from the middle line. Thus nearly one-third of each kidney lies above the twelfth rib.

II. **Palpation.**—In their normal condition the kidneys are not within the range of palpation, but when they leave their normal position or increase greatly in size, they may be felt by means of the bimanual examination, as described on p. 48. Whilst palpation is being performed, the patient should be now and again directed to take a full inspiration.

The Movable or 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 the characteristic renal shape, its size, smooth surface and rounded edge. When the kidney is grasped between the hands, the patient often experiences a sensation of nausea and pain. If the range of mobility be slight, only the lower portion of the kidney may be palpable, and that only on deep inspiration. If the range be more extensive, the floating kidney may wander into various regions of the abdomen.

As excessive mobility of the kidney is ten times more frequent on the right than on the left side, the floating kidney is most likely to be mistaken for a distended gall-bladder, a constricted portion of the right lobe of the liver, or a mesenteric tumour. From a distended gall-bladder the movable kidney is distinguished by its mobility, by the ease with which it can be felt at one time, and at another by the difficulty experienced in its detection, and further, as was pointed out on p. 62, when speaking of the "corset liver," by the fact that when the floating kidney is grasped between the two hands, it can be prevented from passing upwards on expiration.

Enlargement of the Kidney occurs in hydronephrosis, hydatid

disease, tuberculosis, sarcoma, carcinoma, etc., and the tumour is smooth or nodular according to its nature. It is recognised by its position, immobility on respiration, and dulness on percussion. On the left side the enlarged kidney can hardly be mistaken for a splenic enlargement, as the edge of the former is neither sharp nor notched. Further, the colon lies in front of the kidney but behind the spleen, and consequently if, after inflation of the colon (see p. 60), a resonant note is obtained over the swelling, it cannot be the spleen.

Inflammatory thickening around the kidney and perinephritic abscess may also be detected by palpation.

III. **Percussion.**—The outline of the normal kidney cannot be defined by percussion. The most frequently occurring renal diseases are not accompanied by so great an amount of alteration in the dimensions of these organs as to render them appreciable by percussion. Percussion is therefore an aid in diagnosis only when there is great renal enlargement (hydronephrosis, tumour, etc.), and when, as already described, we seek to ascertain the relation of the swelling to the colon.

THE PANCREAS

I. **Topographical Anatomy.**—The pancreas crosses the middle line on a level midway between the xiphisternal junction and the umbilicus, and opposite the disc between the first and second lumbar vertebræ.

II. **Examination of the Pancreas.**—*Tumours* and *cysts* 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 stomach, intestines, and to some extent by the lower edge of the liver. An increased resistance or definite swelling may be felt to the right of, or in the middle line above the level of the umbilicus. The swelling is deeply seated, often nodular (if a tumour), or smooth and elastic (if a cyst), almost immobile and unaffected by the respiratory movements, but pulsation is usually transmitted through the tumour from the aorta beneath. Malignant disease of the pancreas is almost invariably carcinoma, and is seldom limited to the gland, but invades the retro-peritoneal lymphatic glands and other neighbouring parts.

In differentiating a tumour of the pancreas from one of the pylorus or colon, it is important to remember that the pancreas lies behind the stomach and colon. After inflation of the stomach

or colon, a pancreatic tumour will no longer be palpable, and any area of dulness it might have caused will have given place to a tympanitic note.

Though an accurate diagnosis of pancreatic disease is seldom possible by examination of the abdomen alone, there are other signs which are suggestive or confirmatory of pancreatic disease. They are—

1. *Diabetes*, and more especially when this is accompanied by bronzing of the skin—bronzed diabetes (see p. 8).

2. *Signs of disturbed intestinal digestion.*—The most obvious of those signs is *steatorrhœa* or the passage of fatty stools, which are usually bulky, pale, soft and often offensive. Fatty stools are, however, not constant and may be due to other causes (see p. 87). Again there may be impairment of proteid digestion, manifested by the presence of a large quantity of undigested nitrogenous food material in the stools (*azotorrhœa*).

3. *Jaundice*, which is insidious in onset, yet persistent and progressive. This sign is especially significant when there is much distension of the gall-bladder.

4. *Sahli's sign.*—The fasting patient is given one grain of iodoform enclosed within a glutoid capsule (gelatine hardened in formalin). The capsules designed for diagnostic purposes are to be employed in preference to the softer capsules intended for the therapeutic administration of drugs. The capsules are unaffected by gastric digestion, but are dissolved when they come in contact with the pancreatic juice, when the iodoform is liberated and absorbed. That event is recognised by the appearance of the iodine reaction in the saliva or the urine (see p. 83). Similarly several grains of salol in a glutoid capsule may be administered, and the urine tested for salicyluric acid (see pp. 83, 84). In normal persons, in whom there is neither motor insufficiency of the stomach, pancreatic disease, diarrhœa nor other intestinal derangement, the iodine or salicyluric reaction is obtained from four to six hours after administration of the capsules. A positive reaction within six hours contraindicates severe pancreatic disease, but a delayed or negative reaction does not necessarily indicate disturbance of pancreatic function. The test is therefore of limited value.

None of these signs are in themselves pathognomonic of disease of the pancreas, but when these signs are conjoined they indicate the probability of grave disease of that gland, and the diagnosis will be confirmed if a tumour, having the characters already described, can be detected in the epigastrium.

Omentum.—Tumours of the omentum are rare. They are of very various nature: cancerous, tuberculous, hydatid, etc., 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, tuberculous or other deposits. They form smooth, hard, movable tumours of regular form. Occasionally they become fused together, along with other neighbouring structures (loops of small intestine, retro-peritoneal glands, etc.), into masses of considerable size, which, overlying the aorta, may have imparted to them a pulsatile movement.

The **Urinary Bladder** does not enter the abdomen unless distended, when it forms a pyriform swelling lying in the middle line above the pubes, and giving a dull note. It may reach the level of the umbilicus.

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 have been already considered (see p. 52).

Uterine tumours, physiological as well as pathological, may be felt above the pubes. Their consideration belongs to the domain of the gynæcologist 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 IV

Alimentary System (*continued*)

EXAMINATION OF THE GASTRIC CONTENTS

Gastric digestion is best studied by examination of the gastric contents.

So as to be able to draw accurate conclusions as to the activity of digestion 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 is the **test-breakfast** of Ewald, which consists of two rolls or slices (70 grammes) of wheaten bread and a cup (400 c.c.) of weak tea, without milk or sugar. This meal should be taken about 8 a.m., and the stomach contents removed one hour thereafter.

More information is obtained after a **test-dinner**, consisting of soup, 6 ounces of minced beef, and 2 ounces of wheaten bread; the gastric contents being withdrawn four hours later.

Methods of Obtaining the Gastric Contents.—One uses a soft, india-rubber stomach tube, with a rounded blind end and a lateral eyelet. The tube is connected by a glass tube three inches long with another india-rubber tube, to the further end of which a glass funnel is attached. The stomach tube is passed according to the directions given on p. 39. The entrance of the tube into the stomach is usually indicated by the audible escape of gas. The funnel is now lowered, and if the patient be not already retching, he is directed to do so, or to cough. The gastric contents may then be forced through the tube, and are caught in a basin placed on the floor. Should this method fail to withdraw the gastric contents, the firm pressure of the hand over the epigastrium may be successful, or an aspirator, such as that of Boas or Senorance (see Fig. 16), may be used.

Investigation of Gastric Contents

Amount.—Under normal circumstances, the quantity of gastric contents obtained after the test-breakfast is about 30 to 50 c.c.; after a test-dinner the quantity is somewhat greater. If, in a case where there is no gastric dilatation, very much more than these quantities can be removed from the stomach, say 100 or 200 c.c., then hypersecretion may be suspected; whereas,

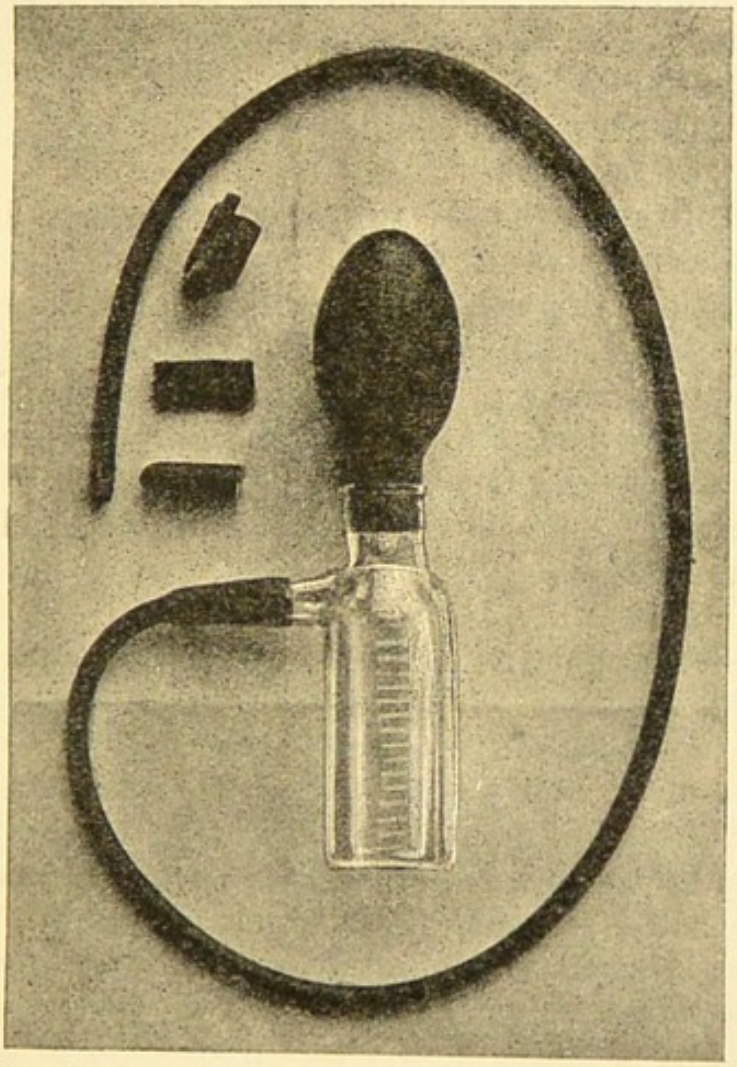


Fig. 16.—Senorance's extractor (one-fifth of the natural size).

if a considerable quantity of gastric contents containing food particles be obtainable six or seven hours after a meal, motor insufficiency may be inferred (see p. 83).

Colour.—The normal gastric contents after a test-breakfast are almost colourless; those after a test-dinner are usually greyish-yellow. Bile, if present, renders the colour yellowish-green (see p. 85); blood produces a colour varying from bright red to dark brown or black (see p. 85).

Odour.—Instead of the odour of normal gastric contents there may be an exceedingly unpleasant smell, the result of fermentation within the stomach. The odour of rancid butter indicates the presence of butyric acid; the odour of vinegar is due to acetous fermentation. A fæculent odour is indicative of intestinal obstruction.

Consistence.—Normally somewhat resembling thin gruel, the gastric contents may be gelatinous and stringy from an excess of mucus, as in acute gastric catarrh and in carcinoma of the stomach. An excess of mucus may also be due to tracheal or bronchial mucus previously swallowed.

General Appearances.—If the gastric contents be allowed to stand in a glass vessel, it will be found that in some cases of gastric dilatation the contents separate into three layers: an upper frothy layer composed of oil droplets, sarcinæ, etc. (see p. 86); a middle layer of turbid fluid; and a lowermost layer of undigested food particles. It is important to ascertain whether these are of proteid or carbohydrate nature (see p. 85).

The gastric contents should now be filtered and subjected to chemical examination.

Reaction.—The gastric contents, under normal circumstances, give an acid reaction when tested 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 hydrochloric acid in combination with proteid. Up to this time the hydrochloric acid secreted has all combined with the proteids of the food. Later on under normal circumstances, the amount of hydrochloric acid secreted has been more than sufficient to satisfy the affinities of the proteid in the food, and there is consequently free hydrochloric acid present in the stomach. The secretion of hydrochloric acid normally attains its maximum about an hour after a test-breakfast, and about four hours after a test-dinner. Under pathological conditions considerable variations occur, and the acidity of the contents, when withdrawn one hour after a test-breakfast or four hours after a test-dinner, may depend on the presence of hydrochloric acid, free or combined with proteids, on acid salts, particularly acid phosphates, or on organic acids.

Detection of Free Acid.—A strip of Congo-red test-paper is dipped into the filtered or unfiltered gastric contents. The red

colour is changed to blue if free acid be present; neither combined hydrochloric acid nor acid salts causes this change of colour.

Tropœolin, methyl-violet, and dimethylamidoazobenzol (0·5 per cent. alcoholic solution), may also be used as tests for the presence of free acid.

Detection of Free Hydrochloric Acid.—If Congo-red test-paper be turned blue, free hydrochloric acid is almost certainly present. It is advisable, however, to test with some reagent which gives a colour reaction only with free hydrochloric acid.

1. The best reagent is that of Günzburg; consisting of

Phloroglucin	2 grammes
Vanillin	1 gramme
Absolute alcohol	30 c.c.

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 free hydrochloric acid have been present. The solution does not keep well, becoming of a brownish colour, and an old solution may fail to react to free hydrochloric acid.

2. Boas' reagent; consisting of

Resorcin	5 grammes
Cane sugar	3 grammes
Alcohol, 50%	100 c.c.

It is used in the same manner as Günzburg's reagent, and gives a pink or cinnabar-red colour. Boas' reagent keeps well and is inexpensive.

Detection of Lactic Acid.—(1) Add one drop of Liquor ferri perchloridi to a test-tube full (about 15 c.c.) of distilled water, so as to produce an almost colourless solution. Take an equal quantity of this solution in two test-tubes, and to one add a few drops of the filtered gastric contents. The development of a greenish-yellow tint indicates the presence of lactic acid.

2. Uffelmann's reagent may be employed. It consists of

Carbolic acid (1 in 20)	10 c.c.
Distilled water	20 c.c.
Liquor ferri perchloridi	1 drop

The solution is of an amethyst-blue colour, and should be freshly prepared just before use. On the addition of gastric

contents, the colour is changed to canary or lemon-yellow if they contain much lactic acid, or to a faint greenish-yellow if the lactic acid be in small amount. Hydrochloric acid discharges the colour; acetic acid, grape sugar, and combined hydrochloric acid change the colour to yellowish-brown. It is therefore better, before performing either of these tests, to extract the gastric contents with ether (which takes up the organic acids), to evaporate the ether on a water-bath, and to dissolve the residue in water.

Detection of Volatile Fatty Acids—Butyric Acid and Acetic Acid.—The odour of rancid butter indicates the presence of butyric acid; acetic acid can also usually be recognised by its odour.

(1) 10 c.c. of gastric contents are heated in a test-tube and a piece of blue litmus paper, moistened in water, is held over the mouth of the tube. If butyric or acetic acid be present, the colour of the paper is changed to red.

(2) Uffelmann's Reagent.—Butyric acid discharges the colour and imparts to the solution a grey, opalescent appearance; acetic acid changes the amethyst-blue colour to yellowish-brown.

(3) If further proof be required, an ethereal extract of the gastric contents should be made, the ether evaporated, and the residue dissolved in a little water.

(*a*) To a portion of this residue add a little absolute alcohol and a few drops of strong sulphuric acid, and boil the mixture in a test-tube. The odour of butyric ether, like pineapple rum, indicates the presence of butyric acid. Or to a portion of the residue dissolved in water add a fragment of calcium chloride, and observe the formation of small droplets of butyric acid, which float like drops of oil on the surface of the fluid.

(*b*) Another portion of the residue, dissolved in water, is neutralised with a dilute solution of 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.

Total acidity of the Gastric Contents.—This is readily estimated by titration with a decinormal solution of caustic soda, ascertaining what quantity of the solution is required to neutralise a given quantity of stomach contents. To 10 c.c. of the filtered gastric contents, carefully measured into a white porcelain capsule, 40 c.c. of distilled water are added, and three or four drops of a 1 per cent. alcoholic solution of phenol-phthalein are added as an indicator. From a burette the decinormal solution of soda is

then allowed to fall into this mixture until the pink colour of the fluid no longer disappears on agitation. This 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 acidity is easily calculated. Thus if 6·4 c.c. have been required to neutralise 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, according to Ewald's notation, 64. The total acidity is more frequently expressed as percentage acidity in terms of hydrochloric acid. For example, if 6·4 c.c. of decinormal soda solution were required to neutralise 10 c.c. of gastric contents, 64 c.c. would be required to neutralise 100 c.c. of gastric contents. Now 1 c.c. of the decinormal soda solution corresponds to 0·00365 gramme of hydrochloric acid, therefore 64 c.c. corresponds to 0·2336 gramme of hydrochloric acid, and consequently the acidity of 100 c.c. of gastric contents is equal to 0·233 per cent.

The total acidity, after the test-breakfact, is normally from 0·18 to 0·29 per cent., or, according to Ewald's notation, from 50 to 75.

The determination of the total acidity does not, however, tell us on what that acidity depends. It may be due to free hydrochloric acid, to hydrochloric acid loosely combined with proteids, to acid salts (particularly acid phosphates), or to organic acids, lactic, butyric, acetic. If the total acidity be excessive (above 0·29 per cent.), and free hydrochloric acid be present, there cannot be more than traces of organic acids. We may therefore conclude that the hyperacidity is due to excessive hydrochloric acid. It is, however, better to estimate the amount of free hydrochloric acid.

Estimation of Free Hydrochloric Acid.—(*a*) Measure 10 c.c. of the gastric contents into a porcelain capsule, and add twenty-five drops of Günzburg's reagent. A drop of the mixture, removed with the platinum loop, gives the characteristic crimson colour on evaporation over the flame. To the mixture now add slowly decinormal solution of caustic soda from a burette until a drop of the mixture 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 described above.

(*b*) *By Töpfer's Method.*—To 10 c.c. of the gastric filtrate add two or three drops of a 0·5 per cent. alcoholic solution of dimethylamidoazobenzol. Then add decinormal solution of caustic soda from a burette until the red is entirely replaced by a yellow colour. The calculation is made as already described.

An hour after the test-breakfast the gastric filtrate contains from 0·07 to 0·20 per cent. of free hydrochloric acid.

Estimation of the Free and Combined Hydrochloric Acid.—There are many methods, but the majority are too complicated for clinical work. The simplest is that of Töpfer:—*Firstly*, estimate the total acidity. *Secondly*, estimate the free hydrochloric acid, using dimethylamidoazobenzol as an indicator. *Thirdly*, to 10 c.c. of the gastric filtrate add a few drops of a one per cent. aqueous solution of alizarin as an indicator, and titrate with decinormal solution of caustic soda until a violet colour is produced. All the factors causing the acidity, except combined hydrochloric acid, have now been neutralised. *The calculation* is made as follows:—If, for example, with alizarin as the indicator, 5 c.c. of the soda solution were used for 10 c.c. of gastric filtrate, 50 c.c. would be required for 100 c.c. of the filtrate. The total acidity (previously determined) being 60, the acidity due to combined hydrochloric acid = 60 − 50 = 10, and is therefore equal to 0·036 per cent. And if the free hydrochloric acid be 0·15 per cent., the acidity of the free and combined hydrochloric acid = 0·15 + 0·036, or 0·18 per cent. The difference between this and the total acidity (0·21 per cent.) represents the acidity due to acid phosphates and organic acids.

Estimation of lactic acid is of lesser clinical importance. It is sufficient to note the intensity of the colour reaction with weak perchloride of iron solutions or Uffelmann's reagent.

Clinical Significance of Hydrochloric and other Acids.—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 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 pathogenetic bacteria are destroyed. That the cholera vibrio is thus destroyed, Koch showed many years ago; and the same fate befalls most of the cocci and bacilli that gain entrance to the stomach with the saliva or the food. The bacilli of tuberculosis, anthrax, and typhoid fever 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 0·18 per cent. to 0·29 per cent., the amount of *free* hydrochloric acid being from 0·07 per cent. to 0·20 per cent. After a test-dinner, the total acidity is usually about 0·3 per cent. In certain diseases of the stomach, such as nervous dyspepsia, in simple atony of the muscular walls of the stomach, and occasionally in gastric ulcer, the normal amount of acid may be found.

Increased total acidity with excess of free hydrochloric acid constitutes the condition known as *Hyperchlorhydria.* This may be a pure neurosis, as is often seen in neurasthenia, or may be associated with organic disease, such as gastric ulcer or chlorosis. Hyperchlorhydria is also found in some cases of gastric hyper-secretion, for instance in the gastric crises of tabes dorsalis.

Diminished amount of hydrochloric acid (*Hypochlorhydria*) is found when 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 pulmonalis, in severe anæmia, in some, but not all, cases of gastric carcinoma, and as a neurosis, especially in neurasthenia. There is usually at the same time deficient secretion of pepsin.

Absence of free hydrochloric acid (*Achlorhydria*) is found in many cases of gastric carcinoma, and is suggestive but not pathognomonic 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.

Clinical Significance of Lactic and other Organic Acids.—During no stage of normal digestion do the gastric contents contain lactic, butyric, or acetic acid. These acids, of which lactic is the most important, are the products of fermentation. Lactic acid fermentation is inhibited by a mere trace of free hydrochloric acid, and the presence of lactic acid consequently indicates deficient secretion of hydrochloric acid and is found when, within the stomach, there is stagnation due to any cause, such as gastric carcinoma, cicatricial stenosis of the pylorus, or gastrectasis resulting from atony.

The presence of butyric and acetic acids is likewise due to abnormal stagnation and fermentation.

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 thin discs of white of egg, 1 cm. in diameter, cut with a cork-borer. If the flask is placed in an incubator at 37° 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 albumin in about three hours.

Quantitative Estimation of Pepsin.—This is performed by *Mett's method.* Several capillary tubes, 1 to 2 mm. in diameter, are filled with white of egg, the ends of the tubes are plugged with cotton-wool, and the albumin is thereafter coagulated by immersing the tubes in water at 95° C. for fifteen minutes. The tubes are then removed, and their ends are dipped in melted paraffin in order to prevent drying of the albumin. When required, the tubes are cut into portions 3 cm. long; two or three of these are put into a test-tube containing the gastric filtrate, and left in the incubator at 37° C. for ten hours. The length of the capillary tube, and that of the solid albumin still remaining within it, are then measured with the aid of a lens and millimetre scale. The difference represents the length of albumin digested, and the square of this length represents the relative amount of pepsin. For example, 2 mm. of albumin digested equals 4 parts of pepsin; 3·5 mm. digested equals 12·2 parts of pepsin. Normally from 5·5 mm. to 5·9 mm. are digested.

Nirenstein and Schiff's modification of Mett's Method.—The capillary tubes are placed for twenty-four hours in gastric filtrate which has been diluted sixteen times with $\frac{1}{20}$ normal solution of hydrochloric acid. The square of the length of albumin digested multiplied by 16 represents the amount of pepsin in the gastric juice. If more than 3·6 mm. of albumin be digested, the gastric filtrate should be diluted thirty-two times.

Rennet can be readily demonstrated by adding an equal quantity of gastric contents, previously neutralised with decinormal solution of caustic soda, to 10 c.c. of boiled, neutral or amphoteric, milk. In the incubator, curdling should take place in about a quarter of an hour.

Absence of pepsin and rennet is found only in very advanced organic disease of the stomach, such as carcinoma or amyloid disease. If pepsin be deficient there is almost invariably a relatively greater deficiency of hydrochloric acid.

Examination of Starch Digestion.—The digestion of starch by the diastatic ferment of the salivary glands continues within the

stomach until the percentage of hydrochloric acid amounts to 0·1. In cases of hyperchlorhydria and of gastric hypersecretion, amylolysis being defective or even entirely arrested, starch is not successively converted into erythrodextrin, achroödextrin, and maltose. Starch digestion is tested :—

(*a*) By microscopic examination of the gastric contents and detection of starch granules (p. 85).

(*b*) By testing with a solution of

Iodine	0·1 gramme
Potassium iodide	0·2 ,,
Distilled water	200 c.c.

If an excess of this be added to the gastric filtrate, a blue colour indicates unchanged starch ; a purple colour denotes erythrodextrin ; no change of colour is obtained with achroödextrin or maltose. The normal gastric filtrate, therefore, yields no blue or purple colour with this solution of iodine. If maltose be present, the gastric filtrate will reduce Fehling's solution.

The Rapidity of Absorption possessed by the Stomach may be tested by causing the patient to swallow a gelatine 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, etc., considerable delay may be noted, but the information obtained is not of great value.

The Motor Power of the Stomach is best tested by its state as to contents six or seven hours after a test-dinner, the determination being made by means of the *stomach tube.* At that time the stomach should be empty, but if, on the contrary, it is then found to contain any considerable amount of food, a loss of motor power may be inferred. The cause of the motor insufficiency, whether due to pyloric obstruction or atony of the gastric wall, must be sought for by other methods of examination.

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. If there be motor insufficiency, the first appearance of the reaction in the urine is considerably delayed.

The test may also be made with *iodipin*, which is decomposed by bile and pancreatic juice but not by gastric juice. Iodine should be found in the saliva fifteen minutes after the ingestion of iodipin. In motor insufficieney the reaction is not obtained until a period varying from forty-five minutes to several hours has elapsed.

EXAMINATION OF THE VOMIT

The vomit should first be subjected to careful **macroscopic examination**, the amount, colour, consistence and odour being noted. The vomiting of large amounts of sour vomit every few days will suggest the possibility of gastrectasis.

Of abnormal substances present, the most important is **blood**, which may be almost pure, but has been 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 (leucocythæmia), 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 in the vomit imparts to it a greenish tint; *mucus* in large

amount is observed in gastric catarrh. The vomit rarely contains *pus* or *fæcal matter*. The latter usually points to intestinal obstruction, although it may occur independently of mechanical obstruction, as when the bowel is paralysed as a consequence of peritonitis.

Chemical Examination.—After filtration, the vomit may be examined for free HCl, lactic acid, etc., as has been described in the previous pages when speaking of the gastric contents.

For the detection of **blood**, the direct application of the guaiac and ozonic ether test to the vomit is not satisfactory, because the latter may contain substances other than blood which give a similar colour reaction. The test is best performed according to *Rossel's method*:—Extract 5 c.c. of the vomit with 20 c.c. of ether. Having poured off the ether, and thus removed the fat, add to the vomit 3 c.c. of acetic acid and again extract with ether. Then test the acid ethereal extract with 10 drops of alcoholic solution of aloin and with ozonic ether. If blood were present, the aloin solution turns first pink and then cherry red. If no blood be present, the colour remains yellow for at least one hour.

The presence of blood may also be demonstrated by *Teichmann's test*, namely, by taking up some of the brown deposit and proceeding with the test as described on p. 317.

To detect blood in the vomit by *spectroscopic examination*, shake up a portion of the vomit with water, add a few drops of sulphuric acid, filter and extract with ether. If blood were present, the ethereal extract will present the spectrum of acid-hæmatin (see Fig. 52, p. 123).

Bile is recognised by Rosin's or Gmelin's tests, which are described on p. 319.

Microscopic Examination.—A little of the deposit is transferred by means of a pipette to three slides. (1) The first is examined under the low power for muscle fibres, connective tissue, elastic fibres, starch granules, vegetable cells, fat droplets, epithelium, etc.

(2) To the specimen on the second slide, add one or two drops of saline solution, and examine under the high power for torulæ (yeasts) and sarcinæ, both of which indicate abnormal fermentation within the stomach. Torulæ are seen as ovoid cells, usually in short chains, and show distinct evidence of budding. *Sarcina ventriculi* (Fig. 17) is a large round coccus arranged in groups of eight, or multiples thereof, so as to form yellow cubical packets resembling bales of wool. Bacilli of various kinds may also be

detected. In a case of suspected gastric carcinoma, search should be made for the *Oppler-Boas bacilli*—long filiform bacilli—

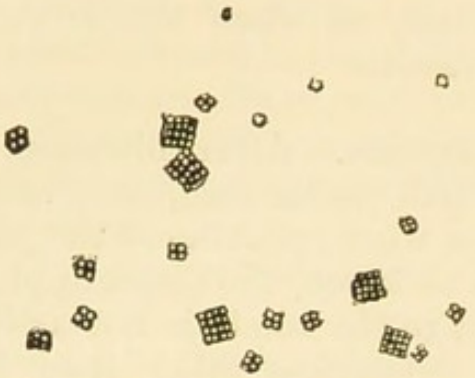


FIG. 17.—Sarcinæ (Roberts).

which are not infrequently present in those cases of carcinoma in which the vomit contains lactic acid but no free HCl.

(3) To the specimen on the third slide, add a drop of Lugol's solution (iodine, potassium iodide and water). This stains starch granules blue, sarcinæ mahogony-brown, and torulæ yellow, and facilitates their detection.

EXAMINATION OF THE FÆCES

I. **Macroscopic Examination.**—1. THE VOLUME of the fæces depends largely on the nature of the diet, being greater when the patient is on a vegetable than on an animal diet.

2. FORM AND CONSISTENCE.—The fæces, when of normal consistence, are of cylindrical form. They become much harder when constipation exists, their long delay in the intestine affording time for the more complete absorption of the watery constituents. Under such circumstances the stools take the form of isolated rounded fæcal masses (scybalous masses). When peristaltic action is increased, the fæces are of lesser consistence, like that of gruel. An increased secretion of the intestinal juices adds to this fluidity, and in practice we meet with all gradations of consistence, 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.

3. COLOUR.—The colour of the normal fæces is partly due to pigment (hydrobilirubin), but also depends on the nature of the food eaten: becoming yellowish or orange 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 tint.[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 Urine, p. 319) cannot be obtained; but when, from whatever cause, the peristaltic action of the small intestine is increased, this characteristic play of colours will be seen on the addition of impure 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 (tarry stools, or 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 vomit (see p. 85).

4. The Odour of normal fæces depends chiefly on the presence of indol and skatol. When much decomposition has occurred, the odour may be greatly increased. The normal fæces of a breast-fed infant are almost odourless.

5. Constituents recognisable to the Naked Eye.

(*a*) *Remains of Food.*—On account of their indigestibility, various articles of food, as portions of vegetables, berries, and fruit skins, may appear unaltered in the fæces. Recognisable fragments of connective tissue and animal flesh indicate respectively defective gastric and intestinal digestion. Fat in large amount, when not accounted for by the diet, indicates defective fat digestion, as in cases of obstruction of the common bile duct, pancreatic disease, or amyloid disease of the intestine.

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

(*b*) *Pathological Constituents—Mucus.*—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 conclude that the morbid process affects the lower part of the colon and the rectum. When mucus has a membranous form it is derived from the colon. 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 (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.

Pus.—When of intestinal origin, as in dysentery and carcinoma of the rectum, pus appears as greyish-white particles, mingled with the fæcal matter. The evacuation of large quantities of unaltered pus points to the rupture of an abscess into the lower part of the intestinal tract.

Blood and *Bile.*—To the presence of these substances, allusion has already been made (see p. 87).

Parasites.—If the presence of parasites be suspected, they must be searched for, and, if necessary, the fæces must be carefully washed on a fine sieve. The eggs are detected by microscopic examination.

Cestodes.—The common tapeworm of Great Britain is *Tænia saginata* (*Tænia mediocanellata*). Its head (Fig. 18), about the size of a pin's head, has four suckers but no hooks. The mature proglottides or segments, which are about 16 mm. long and 4 to 8 mm. broad, have each one prominent genital papilla, which alternates from right to left in successive proglottides. The uterus, which is rendered visible if a ripe proglottis be placed for a short time in glycerine and then compressed between a slide and large cover-glass, has on each side twenty to twenty-five lateral branches (Fig. 19). The eggs are shown in Fig. 20, and Fig. 34, p. 93.

Tænia solium is readily distinguished. The small head (Fig. 21) has four suckers and two circlets of hooks, about twenty-eight in number. The ripe segments from the end of the strobilus are seldom liberated singly, are somewhat smaller than those of Tænia saginata, and are recognised by there being only seven or eight lateral branches on each side of the uterus (Fig. 22).



FIG. 18.—Head of *T. saginata* in contracted condition (×8) (after Leuckart).

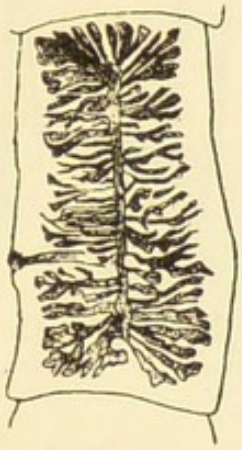


FIG. 19.—Ripe segment of *T. saginata* (×2) (after Leuckart).



FIG. 20.—Egg of *T. saginata* (×400) (after Looss).

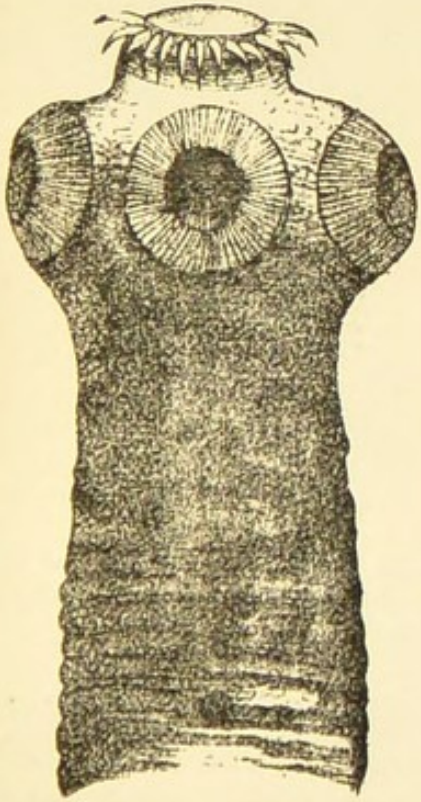


FIG. 21.—Head of *T. solium* (×45) (after Leuckart).

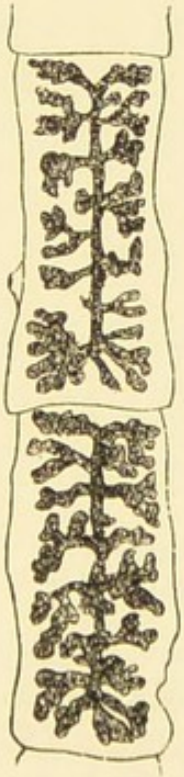


FIG. 22.—Two proglottides of *T. solium* with uterus (×2) (after Leuckart).



FIG. 23.—Egg of *T. solium* (×400) (after Looss).

Bothriocephalus latus, *Hymenolepsis nana*, and other tapeworms are very seldom found in Great Britain. Trematodes are very seldom detected in the fæces.

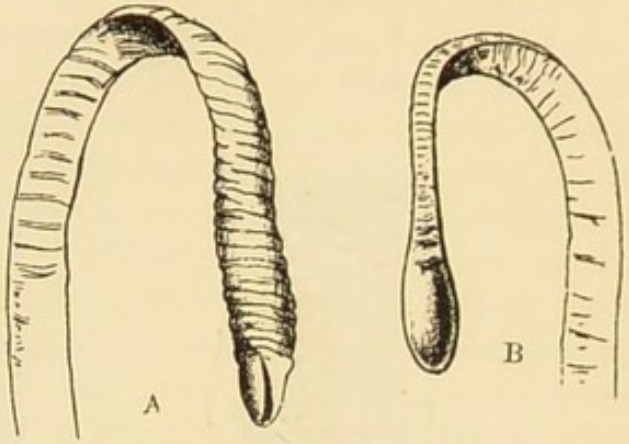


FIG. 24.—Head of *Bothriocephalus latus* (× 8). A, from the flat side; B, from the margin. (After Leuckart.)

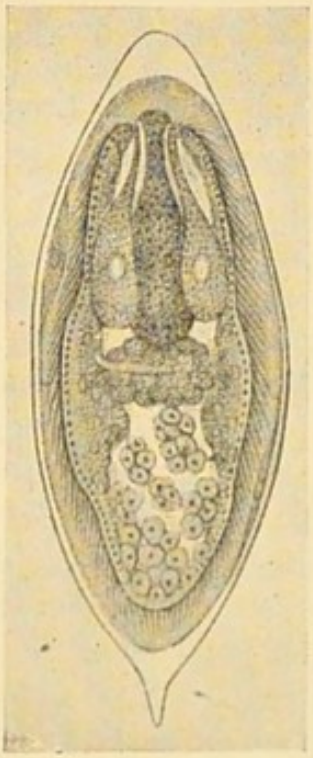


FIG. 25.—Egg of *Schistosomum hæmatobium*, usual form (after Looss).

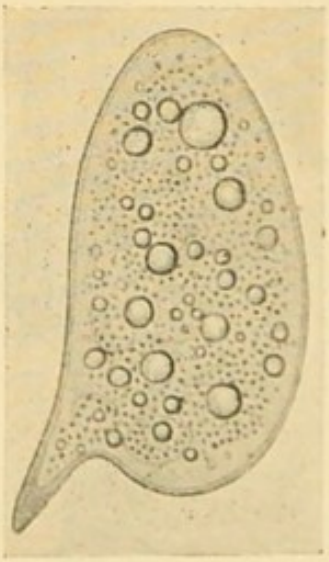


FIG. 26.—Abnormal form of egg of *Schistosomum hæmatobium* (after Looss).

NEMATODES or Round Worms.—*Ascaris lumbricoides* is one of the most frequent nematodes. The male ascaris is 6 to 8 inches long, the female 8 to 12 inches. They resemble the earth-worm. The eggs are shown in Fig. 27.

Oxyuris vermicularis, the thread-worm, is also a common parasite. The male (Fig. 28, B) is 3 to 5 mm. long, with the

caudal end ventrally curved. The female (Fig. 28, A) is 10 mm. long, with a pointed caudal extremity. The thread-worms inhabit the cæcum, colon, and rectum, whence they often migrate on to the skin of the anal region, causing intense nocturnal pruritus ani. The eggs are represented in Fig. 29.

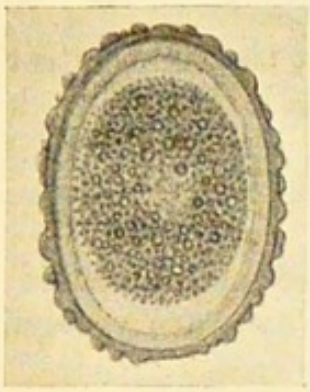


FIG. 27.—Egg of *Ascaris lumbricoides* (× 400) (after Looss).



FIG. 28.—*Oxyuris vermicularis.* A, the female; B, the male; natural size.



FIG. 29.—Egg of *Oxyuris vermicularis* (× 400) (after Looss).

Trichocephalus dispar (*Trichocephalus trichiurus*), the whip-worm (Fig. 30).—The male measures 4 cm., the female 5 cm. in length. The anterior three-fifths of each worm is thin, resembling the lash of a whip, and lies buried in the mucous membrane

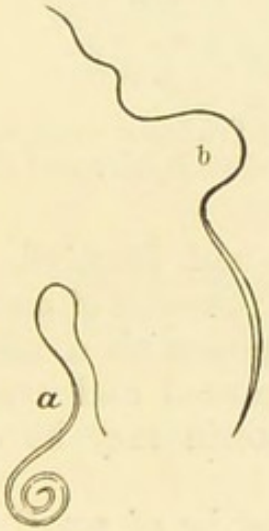


FIG. 30.—*Trichocephalus dispar*: *a*, male; *b*, female; natural size. (Manson, after Blanchard.)

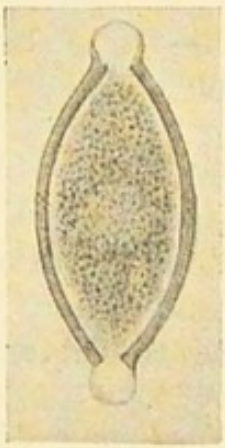


FIG. 31.—Egg of *Trichocephalus dispar* (× 400) (after Looss).

of the cæcum. These worms and their characteristic eggs (Fig. 31) may be detected in the fæces of persons without apparent intestinal disturbance; in other cases the worms cause prolonged diarrhœa and anæmia.

Ankylostoma duodenale.—The male is 6 to 10 mm. long; at the posterior end is a trilobed caudal bursa. The caudal end of the female, which is about 11 mm. long, is pointed. The mouth of both male and female is armed with chitinous teeth. As the worms are firmly attached to the mucosa of the duodenum and jejunum, the diagnosis of ankylostomiasis or miners' anæmia is usually based on the detection of the eggs in the fæces (Fig. 32).

Trichina spiralis.—The adult worms inhabit the small intestine. The male is 1·5 mm. long; the viviparous female is 4 mm. long. The embryos are carried by the blood stream into the voluntary muscles, where they are arrested, become coiled up, encapsuled, and may live for years.

Foreign Bodies and Concretions.—These are detected by washing the stools on a fine sieve. *Gall-stones* are recognised



Fig. 32.—Egg of *Ankylostoma duodenale* (× 400) (after Looss).

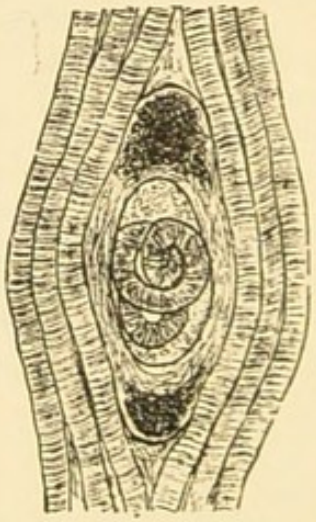


Fig. 33.—Trichinæ encapsuled in muscle (after Mosler and Peiper).

by their facets. A solitary gall-stone is not facetted, and for exact diagnosis it is necessary to prove the presence of cholesterin. This is done by extracting the crushed stone with a mixture of alcohol and ether, which is thereafter decanted and evaporated, when the characteristic crystals of cholesterin may be observed (see p. 341).

Intestinal concretions are rare. They may be of small size, yet abundant, constituting "intestinal sand," which is composed mainly of calcium and magnesium phosphate. This sand must be distinguished by microscopic or chemical examination from the extremely hard undigested vegetable sclerenchyma, of which a considerable quantity may be passed with the fæces, as after ingestion of pears. Larger intestinal concretions consist of phosphates around a central nucleus (gall-stone, fruit-stone, etc.),

of dried fæcal matter impregnated with phosphates, or of medicinal substances, as magnesia or bismuth salts.

II. **Microscopic Examination.**—Of a portion of the fæces, stirred up with water, a drop or two is placed on three slides. The specimen on the first slide is spread out under a cover-glass. To the second specimen add a drop of acetic acid, heat it over the flame, and cover it with a cover-glass. Before covering the third specimen, add to it a drop of Lugol's solution (iodine, potassium iodide, and water).

In Normal Fæces, we find in the first specimen—muscle and connective tissue fibres, vegetable cells, fat (mainly as yellow soap particles), other food particles, debris, bacteria, and various

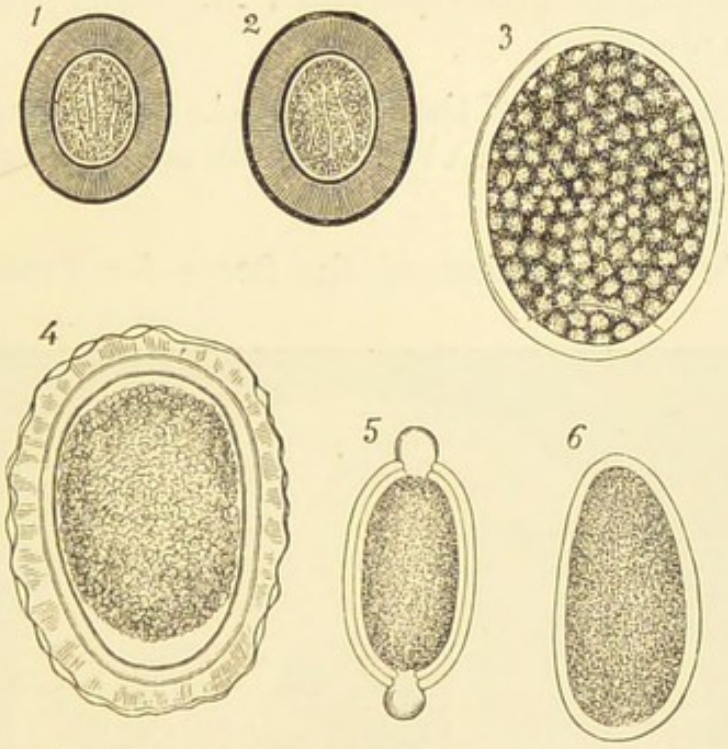


Fig. 34.—Eggs of the following parasites (× 400) :—
1, Tænia solium ; 2, Tænia mediocanellata ; 3, Bothriocephalus latus ; 4, Ascaris lumbricoides ; 5, Trichocephalus dispar ; 6, Oxyuris vermicularis.

crystals—triple phosphates, calcium phosphate, calcium oxalate, etc. If the second specimen be examined while it is still warm, the fatty particles are seen as droplets, whereas they solidify on cooling. In the third specimen, we observe vegetable particles, stained violet.

In Abnormal Stools.—In the first specimen we search for muscle and connective tissue fibres, fat droplets (neutral fat), free starch granules, crystals of fatty acids and of soaps, Charcot-Leyden crystals, hæmatoidin crystals, mucus, pus corpuscles, erythrocytes, fragments of tumours, eggs of parasites (see Fig. 34) and micro-organisms (see Chap. XXXVI.).

Any excess of fatty particles is detected on examining the second specimen. In the third specimen, starch granules stained blue should be looked for. In normal fæces, *free* starch granules are not seen. Their presence indicates catarrh of the small intestine.

III. **Chemical Examination**—(1) *Reaction.*—The fæces are normally of amphoteric reaction, but are alkaline in dysentery, typhoid fever, etc. In the acute catarrhal enteritis of children the stools are usually acid.

(2) *Schmidt's sublimate test.*—Add a portion of the fæces, previously stirred up with water, to a shallow vessel filled with saturated aqueous solution of corrosive sublimate. On examination twelve hours later, normal fæces, which contain hydrobilirubin, are stained red ; any green particles, indicating the presence of bilirubin, are abnormal.

IV. **Micro-organisms of the Fæces.**—As the bacteria of the fæces are dealt with in Chap. XXXVI., the protozoa alone are considered here.

Microscopic examination of the fæces for Protozoa.—From

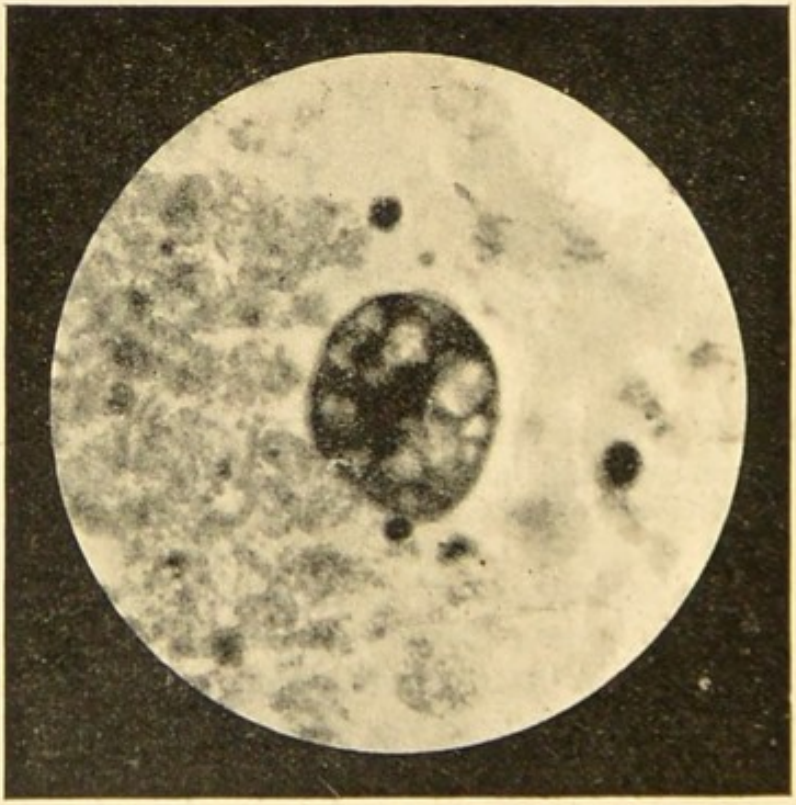


FIG. 35.—*Amœba dysenteriæ* (*Entamœba histolytica*) with vacuolated cytoplasm, in the pus of hepatic abscess (× 1000).

the freshly passed fæces, pick out a small mass of mucus, place it on a slide, and, protecting it with a cover-glass, examine it with

a high-power objective. The fresh specimen may be stained by adding a drop of methylene blue. The *amœbæ* of the fæces are 10 to 50 μ in diameter, nucleated, vacuolated, and capable of movement by means of short pseudopodia. *Amœba coli* may be found in the stools of healthy persons, and of cases of enteritis. In the fæces of some patients suffering from dysentery, and also in the pus of tropical abscess of the liver, *Amœba dysenteriæ* (*Entamœba*

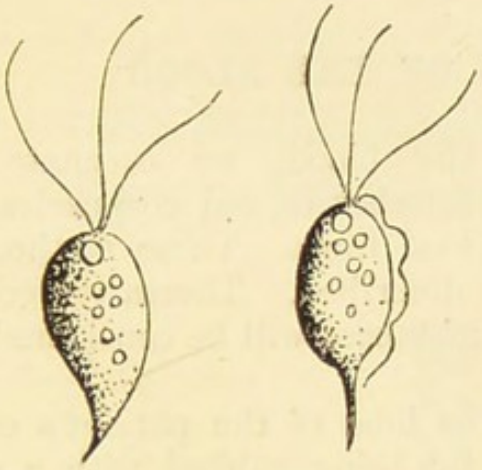


FIG. 36.—*Trichomonas hominis* (after Grassi).

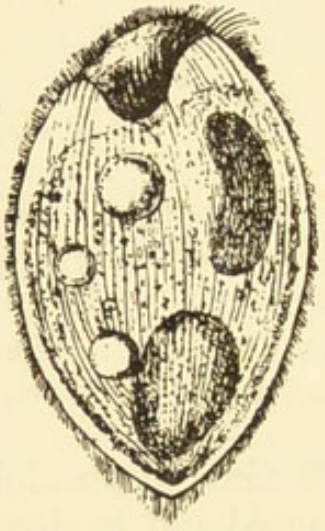


FIG. 37.—*Balantidium coli* (after Leuckart).

histolytica) may be detected (Fig. 35). In appearance it resembles the more common, saprophytic *Amœba coli.*

Trichomonas hominis (Fig. 36) is of pyriform shape, 4 to 10 μ long, and has three flagellæ. *Lamblia intestinalis* is also pyriform, 10 to 16 μ long, and has four pair of flagellæ. *Balantidium coli* (Fig. 37) is of ovoid shape, 40 to 110 μ long, and is covered with cilia. These three harmless saprophytes may be found in the stools in cases of diarrhœa.

CHAPTER V

THE EXAMINATION OF THE BLOOD

In the routine examination of the blood, we estimate the amount of hæmoglobin and the number of red corpuscles and leucocytes, and examine stained blood films. These methods of examination will therefore be first described. Thereafter certain other procedures required in some instances will be considered.

Method of obtaining Blood.—The lobe of the patient's ear is cleansed and rendered hyperæmic by being rubbed with a swab moistened with ether. When the ether has evaporated, the under surface of the lobe is quickly stabbed with a blood-pricker or small lancet which has also been cleansed with ether. If the blood does not flow freely from the puncture wound, another and deeper puncture must be made. The flow of blood must never be promoted by compression of the part, for lymph would thereby be squeezed out of the tissues and the drop of blood consequently diluted.

Before making the puncture, all the apparatus required in the investigation must be ready for use at the patient's bedside.

ESTIMATION OF HÆMOGLOBIN

This may be performed by various instruments:—

1. **Gowers' Hæmoglobinometer.**—This consists of two glass tubes of the same diameter, one of which contains a standard colour-solution[1] (glycerine jelly carefully tinted by means of picric acid and carminate 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

[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.

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. 38) having been placed upright in the small 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 ear in the manner already described, twenty cubic millimetres of the blood are carefully sucked up by means of the pipette (B). The point of the pipette is then wiped with a soft cloth and the blood injected into the distilled water in

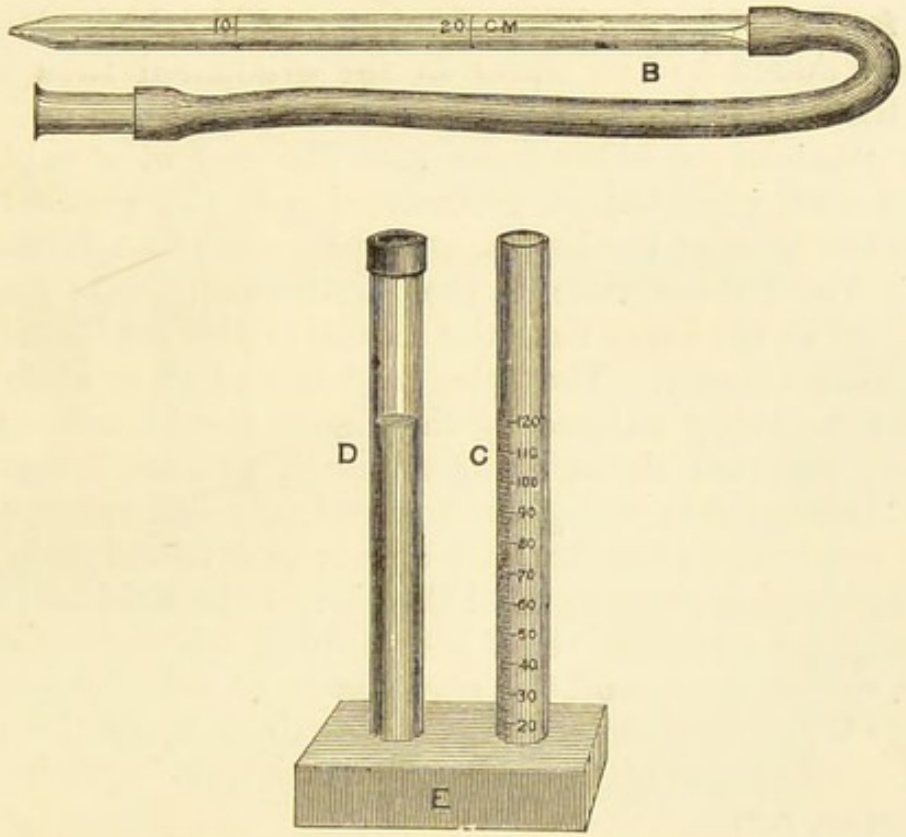


FIG. 38.—Gowers' apparatus for estimating hæmoglobin.

the graduated tube. To ensure the complete transference of the blood from the pipette to the graduated tube, water is now sucked up into the pipette and then injected into the graduated tube, which must then be gently shaken or stirred with a platinum needle to ensure thorough mixture. Care must be taken that no bubbles are formed on shaking. More distilled water is now added, drop by drop, and the tube shaken after the addition of each, until the tint of the diluted blood is the same as that of the standard.

The comparison of the tints can be satisfactorily performed only in daylight. The tubes are to be compared by reflected light, a sheet of white paper being held behind them.

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 the 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.

2. **Haldane's Hæmoglobinometer.**—Haldane's method is an improved modification of that of Gowers. The standard solution is a 1 per cent. solution of blood containing the same amount of hæmoglobin as does the blood of average healthy adult males.

METHOD.—Water is placed in the graduated tube to dilute the blood as far as safely possible. 20 c.mm. of blood, obtained in the pipette, are added to the water in the graduated tube, and the water is then sucked into the pipette two or three times to complete the removal of blood from it. The end of a rubber tube connected with a gas burner is inserted into the graduated tube nearly to the level of the liquid, and the gas turned on for a few seconds. The rubber tube is then withdrawn while gas is still passing, and at the same time the mouth of the graduated tube is closed with the finger. The tube, held in a cloth so as to prevent the fluid from being warmed by the fingers and therefore spurting out, is now inverted about a dozen times, without being shaken, and the hæmoglobin being thus completely saturated with CO, the fluid acquires a pink tint. Water is now added drop by drop from the dropping pipette, until the tint of the fluid matches that of the standard solution. Half a minute is allowed to elapse for the fluid to run down, and the percentage of hæmoglobin is then read off. Water is then added drop by drop, until the tints are unequal. The mean of the readings which give equality of tints is the correct result.

When the tints are being compared, the tubes are to be held against the light from the sky, or against an opal glass shade when artificial light is employed. While 100 per cent. on the hæmoglobin scale is the average reading for the normal adult male, the figures for healthy women and children are respectively 89 per cent. and 87 per cent.

3. **Hæmometer of v. Fleischl-Miescher.**—The apparatus is shown in Fig. 39. Blood is drawn into the pipette up to the mark $\frac{1}{2}$, and is at once diluted by filling the pipette up to the mark 200 with a 0·1 per cent. solution of sodium carbonate. The ends of the pipette being then closed by the fingers, it is thoroughly shaken; the diluting fluid in the capillary portion of the pipette is blown out, and the blood solution is then transferred

to one compartment (a') of the chamber (M, or M′) until a convex meniscus is formed. The other compartment (a) is similarly filled with a 0·1 per cent. solution of sodium carbonate. A special glass disc (D, D′) is then slipped over the chamber, and if its compartments were correctly filled there will now be no air-bubbles under the glass disc.

The next stage in the estimation is performed by candle-light, which is reflected from the disc (S). The light from an electric lamp or incandescent gas-burner is not suitable. Under the com-

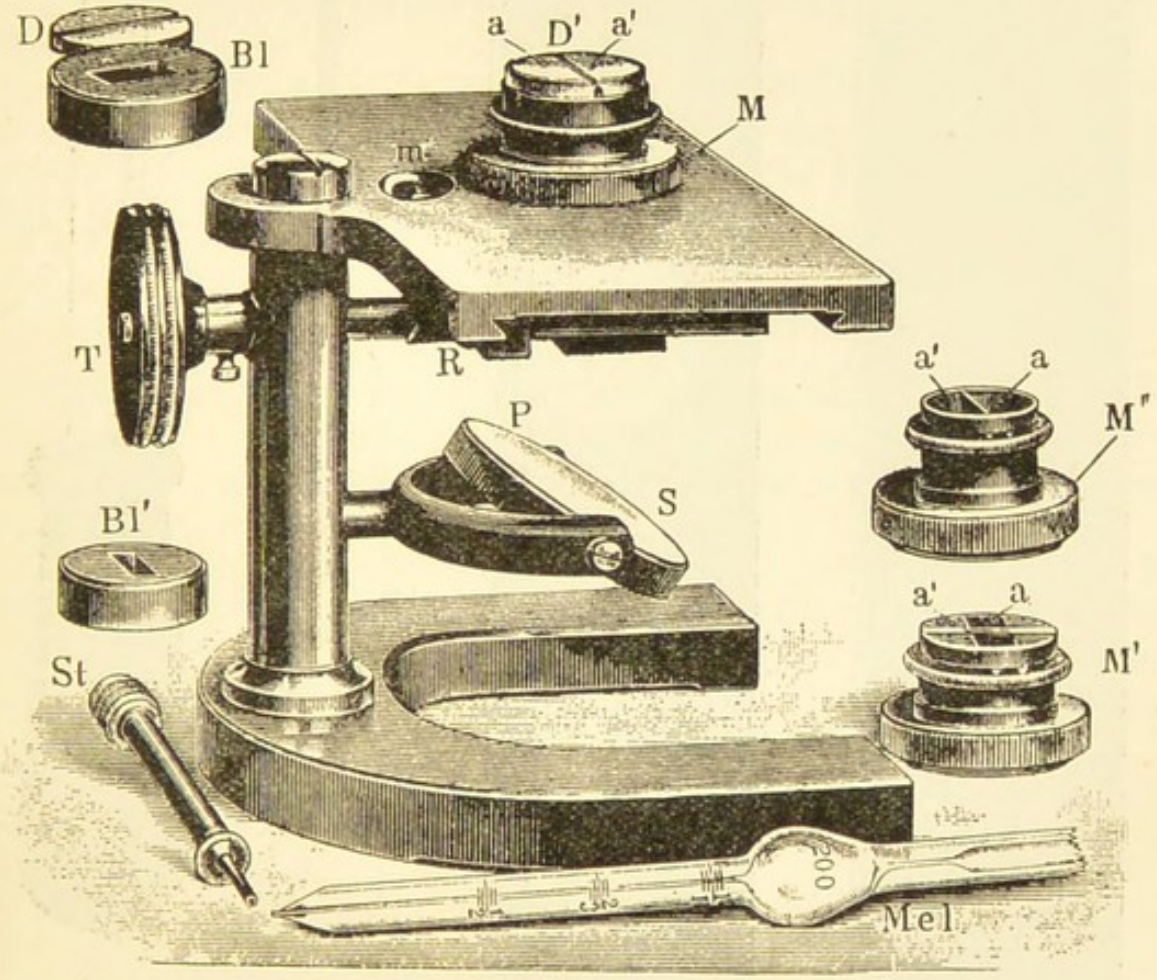


FIG. 39.—The v. Fleischl-Miescher hæmometer (after Grawitz).

partment a, which contains the soda solution, 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 solution. It is thus possible to match the colour of compartment a, containing soda solution, with that of compartment a', which contains the blood solution. The number on a graduated scale attached to the wedge is then read off, and the amount of hæmoglobin, expressed as grammes per cubic millimetre, can readily be calculated from the table furnished with the instrument.

4. **Oliver's Hæmoglobinometer.**—The capillary tube (*c*, Fig. 40), is accurately filled with blood, which is then washed into the cell

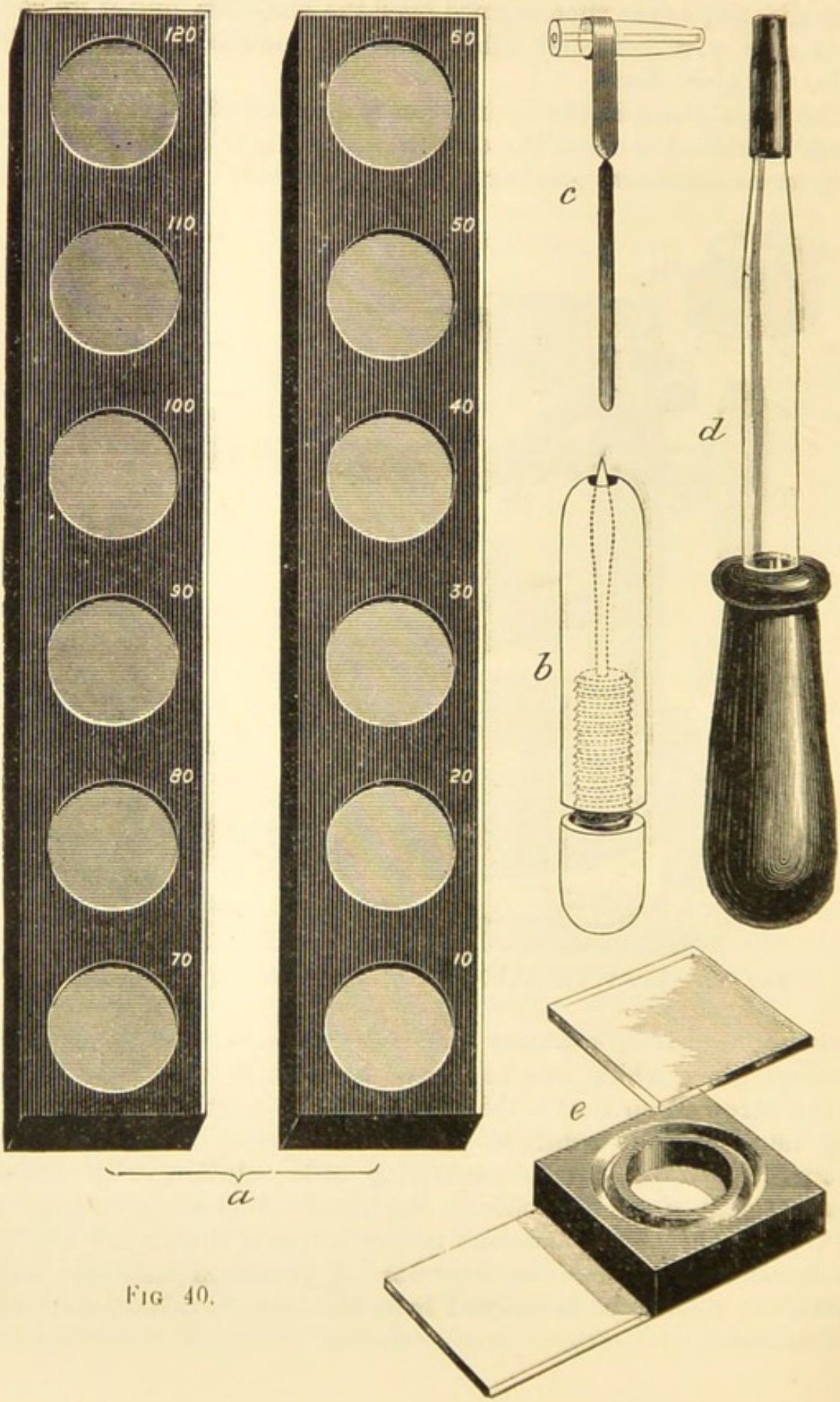


FIG 40.

(*e*) by means of the pipette (*d*) previously filled with distilled water. The solution in the cell is then stirred with the handle of the capillary tube, and distilled water is added so as to fill the cell. When the cover-glass is now applied, a small air bubble should form beneath it, indicating that the cell has not been overfilled. The cell is then placed under the camera tube (not shown in the figure) at the side of the standard discs (*a*) of red glass, and a comparison of the tints is made by candle-light. The figure attached to the disc, which matches in colour the solution in the cell, indicates the percentage of hæmoglobin in the patient's blood.

If the colour of the blood solution is intermediate between that of two discs, say between those marked 70 and 80, we lay one or more "riders" on the lower, *i.e.* paler, of those two discs, until the tint of the solution is exactly matched.

ESTIMATION OF BLOOD CORPUSCLES

This is performed by Thoma's hæmocytometer (Fig. 41), which consists of two mixing pipettes—one for estimating the red corpuscles, the other for the leucocytes—and a counting-chamber.

Estimation of Red Blood Corpuscles.—The blood is to be diluted in the pipette with saline solution (0·9 grms. NaCl in 100 c.c. of distilled water), or in one or other of the following diluting fluids: –

Gowers' Solution—

Sodium sulphate	gr. xxi.
Acetic acid	min. xv.
Distilled water	℥ i.

Hayem's Solution—

Perchloride of mercury	0·5 grm.
Sodium chloride	1·0 „
Sodium sulphate	5·0 „
Distilled water	200·0 c.c.

Toisson's Solution—

Sodium chloride	1·0 grm.
Sodium sulphate	8·0 „
Glycerine (pure)	30·0 c.c.
Distilled water	160·0 „
Methylviolet 5B	0·025 grm.

Suck up blood into the pipette (Fig. 41) as far as the mark 0·5, and wipe the end of the pipette with a soft linen cloth. Then suck up diluting fluid to the mark 101, and while doing so rotate the pipette or shake it gently, so that the fluid in the bulb of the pipette is well mixed, but be careful not to form air-bubbles in the fluid nor to draw the latter beyond the mark 101. Then

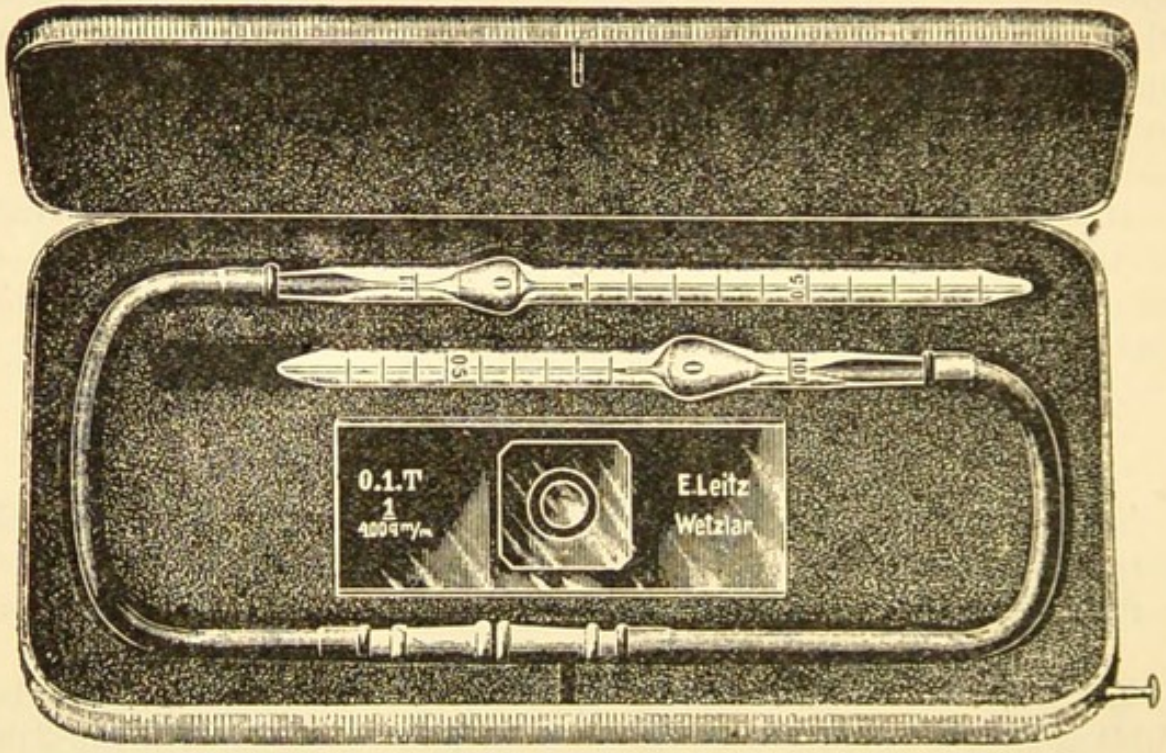


FIG. 41.—Thoma's hæmocytometer (after Grawitz).

hold the ends of the pipette between the thumb and middle finger and shake it briskly for about a minute to complete the thorough mixing of the blood and diluting fluid. Because the capillary tube of the pipette now contains diluting fluid only, a drop or two of the fluid is now blown out on to a cloth or filter paper, and then a small drop of the diluted blood is placed on the centre of



FIG. 42.—The counting-chamber of Thoma's hæmocytometer.

the ruled disc (Fig. 42, *a*) of the counting-chamber, which is clean, dry, and free from dust particles.

The cover-glass (Fig. 42, *b*) supplied with the instrument is then gently lowered until it rests on the glass slab (Fig. 42, *c*), and the drop should now cover the whole of the ruled disc. If the cover-glass has been correctly applied, Newton's colour rings will now be visible between the cover-glass and the slab. Pressure

on the cover-glass may render them more evident, but if they do not appear or do not persist after the pressure is withdrawn, the cover-glass should be lifted, the whole chamber thoroughly cleansed, and another drop from the pipette laid on the ruled disc. Only when Newton's colour rings are evident is the under surface of the cover-glass at the correct distance, namely 0·1 mm., from the upper surface of the disc.

A correct estimation is not possible if fluid flow off the ruled disc and in between the cover-glass and the slab, nor if there be any dust particles between them.

After the cover-glass has been satisfactorily applied, the chamber is set aside for four or five minutes, to permit the corpuscles to settle on the disc, and it is thereafter placed on the stage of the microscope, and examined under a magnification of about 200

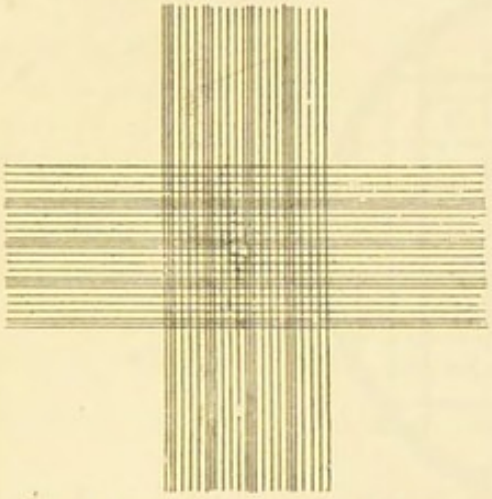


FIG. 43.—The ruling of Thoma's counting-chamber.

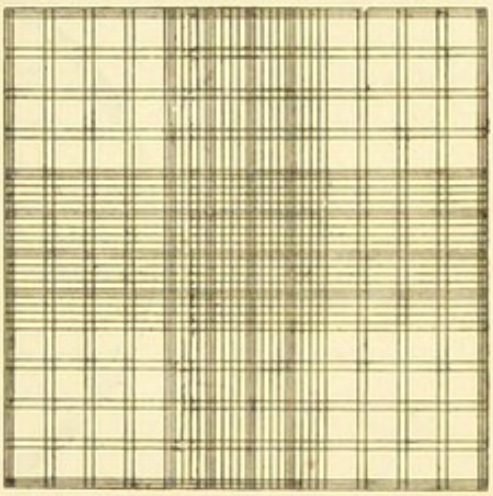


FIG. 44.—The ruling of Türk's counting-chamber.

diameters. The subsequent counting of the red corpuscles is greatly facilitated by the employment of a mechanical stage on the microscope.

At the centre of the disc there will be seen, under the microscope, a square millimetre subdivided into 400 small squares, as in Fig. 43. The number of red corpuscles in at least 100 of these small squares must be counted. The greater the number of corpuscles we count, the more accurate is our estimation.

We may count the red corpuscles in eight groups of 16 small squares, each group being enclosed by a zone of subdivided squares (see Fig. 43). It is better, however, to count the number of red corpuscles in from four to eight adjacent groups of 25 small squares. To do so, place in the field of the microscope the top left hand group of 25 small squares, as in Fig. 45. Then count

the red corpuscles in successive squares from 1 to 25. In the case of each square, any red corpuscles which lie on the left and upper lines are counted, whereas those on the lower and right lines are subsequently counted as if belonging to the adjacent square below or to the right. Then proceed to the next group of 25 small squares, and so on, until the red corpuscles in not less than four such groups, and at least 1000 corpuscles, have been counted.

The final calculation is then made as follows:—The dimension of each small square, namely, $\frac{1}{400}$ sq. mm., is marked on the counting chamber, and as the depth of the fluid was 0·1 mm., each square represents $\frac{1}{4000}$ of a cubic millimetre. If there are x red corpuscles in four groups of 25, *i.e.* in 100 small squares, there are x red corpuscles in $100 \times \frac{1}{4000}$ c.mm., and therefore

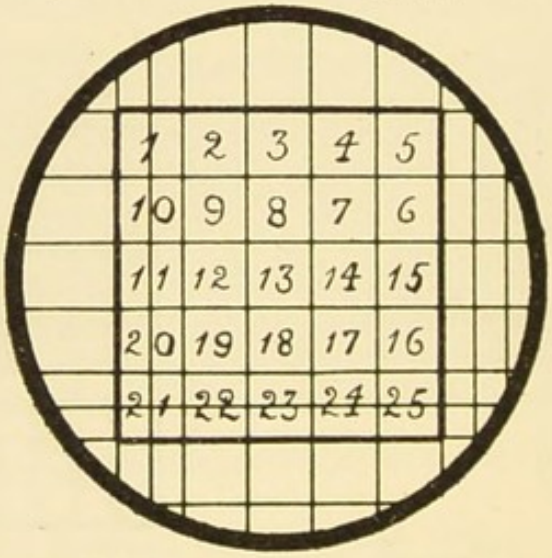


Fig. 45.—Group of 25 small squares in the counting-chamber.

40 x corpuscles in 1 c.mm. But the blood was diluted 200 times, and therefore the number of red corpuscles per c.mm. of blood is 8000 x.

Thus if the blood be diluted 200 times, the number of red corpuscles counted in 100 squares, multiplied by 8000, gives the number of red corpuscles per c.mm. of blood.

The calculation may also be readily made by dividing the total number of red corpuscles counted by the number of small squares examined, and multiplying the result by 4000, and by 200 or 100, according to the degree of dilution.

Estimation of Leucocytes.—Blood is sucked up to the mark 0·5 of the leucocytometer pipette (Fig. 41), and a diluting fluid which stains the leucocytes but renders the red corpuscles colourless is sucked up to the mark 11. The diluting fluid consists of—

Glacial acetic acid	1·0 c.c.
Distilled water	100·0 ,,
1 per cent. aqueous solution of Gentian-violet	1·0 ,,

The dilution of the blood in the pipette is thus 1 : 20. The pipette having been well shaken, and two drops of the diluting fluid blown out, a drop of the diluted blood is placed on the ruled disc and the cover-glass applied with the same precautions as when the red corpuscles were being counted. The leucocytes are then counted in the 400 small squares, *i.e.* over 1 square millimetre. If x be the number of leucocytes counted, and the dilution be 1 : 20, then $\frac{x \times 20 \times 4000}{400}$ = the number of leucocytes per c.mm. of blood. The error by this means of estimation may be as much as 10 per cent.

It is therefore preferable to replace the Thoma counting chamber by that of Zappert or Türk. The ruling of Türk's chamber (Fig. 44) enables us to count the leucocytes over 9 square millimetres. If x be the number of leucocytes counted and the dilution of the blood be 1 : 20, then $\frac{x \times 20 \times 4000}{3600} = \frac{x \times 200}{9}$ = the number of leucocytes per c.mm. of blood, *i.e.* the number of leucocytes counted over the 9 square millimetres $\times \frac{200}{9}$ = number of leucocytes per c.mm. of blood.

The central square millimetre of Zappert's or Türk's chamber (Fig. 44) is subdivided into 400 small squares, and it can therefore be employed, like the older Thoma chamber, for counting the red corpuscles.

The pipettes, after being used, are to be cleansed successively with water, absolute alcohol and ether, but the counting chamber is cleansed with water only, as the cement would be dissolved by alcohol or ether. If the pipette become blocked, it should be cleansed by means of a horse hair or liquor potassæ.

The average **number of red corpuscles** per cubic millimetre of blood is in the man 5,000,000; and in the woman 4,500,000. No doubt in very perfect health those figures are exceeded somewhat. An apparent increase in the number of red corpuscles is seen when there has been much loss of water from the blood, as in severe diarrhœa, dysentery, and cholera. An increased number of red corpuseles (Polycythæmia), perhaps to 8,000,000 per cubic

millimetre, is found in chronic venous congestion and cyanosis, *e.g.* in cases of uncompensated tricuspid regurgitation.

Diminution in the number of red corpuscles (Oligocythæmia) follows hæmorrhage, but is then of a temporary character. More permanent diminution is seen in chlorosis and all forms of anæmia, being most pronounced in pernicious anæmia, where the number of red corpuscles is frequently about 1,000,000 per cubic millimetre.

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 some secondary anæmias, where the percentage may fall very low, to 20 per cent. or even lower. In estimating the amount of benefit derived from the use of iron in any particular case, the hæmoglobinometer is of the utmost service.

The Colour Index.—When the red corpuscles have been estimated and the percentage of hæmoglobin ascertained, we are able to determine the amount of hæmoglobin each red corpuscle contains in comparison with the normal, *i.e.* to determine the colour index. A fraction, of which the numerator is the percentage of hæmoglobin, and the denominator the percentage of corpuscles, will express the colour index. For example, in a normal case with five million red corpuscles and 100 per cent. of hæmoglobin, the colour index is 1 ; whereas if the number of red corpuscles be 3,500,000 and the percentage of hæmoglobin be 42, the colour index is $\frac{42}{70}$, or 0·6, *i.e.*, each red corpuscle contains 0·6 of the normal amount of hæmoglobin.

The colour index is less than 1 in chlorosis and most secondary anæmias, *e.g.* after hæmorrhage, in cancer, etc., whereas in pernicious anæmia the colour index is greater than 1, being perhaps 1·2 or 1·4.

In health the average **number of leucocytes** per cubic millimetre of blood is from 7000 to 8000. 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 (Digestion leucocytosis). The increase, which may amount to 30 per cent., attains its maximum three or four hours after the meal, and depends largely on the amount of albuminous food consumed. Digestion leucocytosis is usually absent in malignant disease of the stomach and liver.

There is a leucocytosis in the newly-born infant, and usually, but not constantly, in pregnancy.

Pathologically, leucocytosis is observed in many conditions. It is found in nearly all acute infective diseases—staphylococcal, streptococcal, pneumococcal, etc. Thus it is seen in acute lobar pneumonia, pericarditis, meningitis, peritonitis, diphtheria, acute rheumatism, scarlet fever, etc. Leucocytosis is also observed in cases of malignant disease, after copious hæmorrhage, and shortly before death (Agonal leucocytosis). In all these conditions the increase in the number of leucocytes is due to increase of the polymorphonuclear leucocytes. In acute infective diseases, for example, acute lobar pneumonia, where an increase to twenty or thirty thousand or more is the rule, a low degree of leucocytosis is an extremely unfavourable sign.

In typhoid fever, measles, and malaria, there is no leucocytosis. In simple tuberculous infection, as in tuberculous peritonitis or meningitis, there is usually a slight leucocytosis, the excess of leucocytes being due to increase of the lymphocytes (*Lymphocytosis*); whereas in cases of tuberculosis with mixed infection, as in many cases of pulmonary phthisis, there is a polymorph leucocytosis. The most striking increase in the number of the white cells takes place in leucocythæmia, where the enumeration may exceed 500,000 in the cubic millimetre.

Decrease in the number of leucocytes constitutes the condition known as *leucopenia*. It is observed, for example, in uncomplicated cases of typhoid fever, and in pernicious anæmia.

MICROSCOPIC EXAMINATION OF FRESH BLOOD PREPARATIONS

The surface of a perfectly clean slide is brought into contact with a drop of blood from the patient's ear, and a clean cover-glass immediately applied to the preparation. The drop will spread out as a film under the cover-glass, to which however no pressure is to be applied. The specimen is immediately examined, not with an oil-immersion lens, but with an objective of low or intermediate power.

According as the preparation is thin or thick, the *red corpuscles*, or erythrocytes, are seen isolated or in rouleaux each of 10 to 30 corpuscles. Each red corpuscle is a biconcave disc, with an average diameter of 7·5 μ.[1] The pale central depression should be visible in each red corpuscle, otherwise the preparation is probably too thin, and the corpuscles are flattened by the pressure of the cover-glass. Abnormalities in the size and shape of the red corpuscles may be noticed in disease. These will be considered when describing the appearances of films which have been fixed

[1] $\frac{1}{1000}\mu$ is a thousandth part of a millimetre.

and stained. We must note, however, that abnormality of shape (Poikilocytosis) must not be confounded with crenation of the red corpuscles, which occurs when the film has begun to dry.

The *tint of the red corpuscles* should also be noted. The general pallor of the corpuscle and its larger central depression in chlorosis and most secondary anæmias is in marked contrast with the deep tint observed in pernicious anæmia.

The leucocytes appear as pale, colourless, and more or less refractile spheroidal cells. If a considerable degree of leucocytosis exist, it may with a little practice be easily recognised in the fresh preparation, but a differentiation of the varieties of leucocytes from one another is more satisfactorily performed in films which have been fixed and stained (see p. 115).

Blood plates, which are from 2 μ to 4 μ in diameter, may be recognised if the preparation be examined directly it is made. In a few minntes they coalesce, forming irregular groups, from which stellate fibrin threads may be seen to develop.

Some of the *parasites* of the blood, *e.g.* the embryonic filariæ, may also be recognised in fresh blood preparations, but they must also be studied in stained films, and are, therefore, considered on p. 118.

EXAMINATION OF BLOOD FILMS

The films may be prepared either on slides or cover-glasses. When slides are employed, good films are more easily spread, and the subsequent manipulations are more easily carried out. Whether slides or cover-glasses be used, they must be perfectly clean, and especially must be free from grease and dust. After having been washed first in water and then in alcohol, they should therefore be kept in equal parts of absolute alcohol and ether in a wide-mouthed stoppered jar or bottle, from which they are removed by means of a pair of clean forceps. They are then dried and polished with a soft clean linen cloth, and laid on a sheet of white notepaper. During these manipulations the fingers are never allowed to come in contact with the surface of the slide or cover-glass on which the blood film is to be spread.

Method of making Films.—1. *On slides.*—The slides must not be too large. Those 7·5 cm. by 2·5 cm., and of the best quality, should be used. A drop of blood is obtained on the surface of the slide, at one end, and is spread out on that slide by means of another applied at an angle of about 30 degrees, and drawn slowly over its surface, as in Fig. 46.

2. *On Cover-glasses.*—To permit of the film being examined with an oil-immersion objective, thin cover-glasses (No. 1) must be used. They may be three-quarters of an inch square, or 27 mm. by 18 mm. A drop of blood is touched with the surface of a

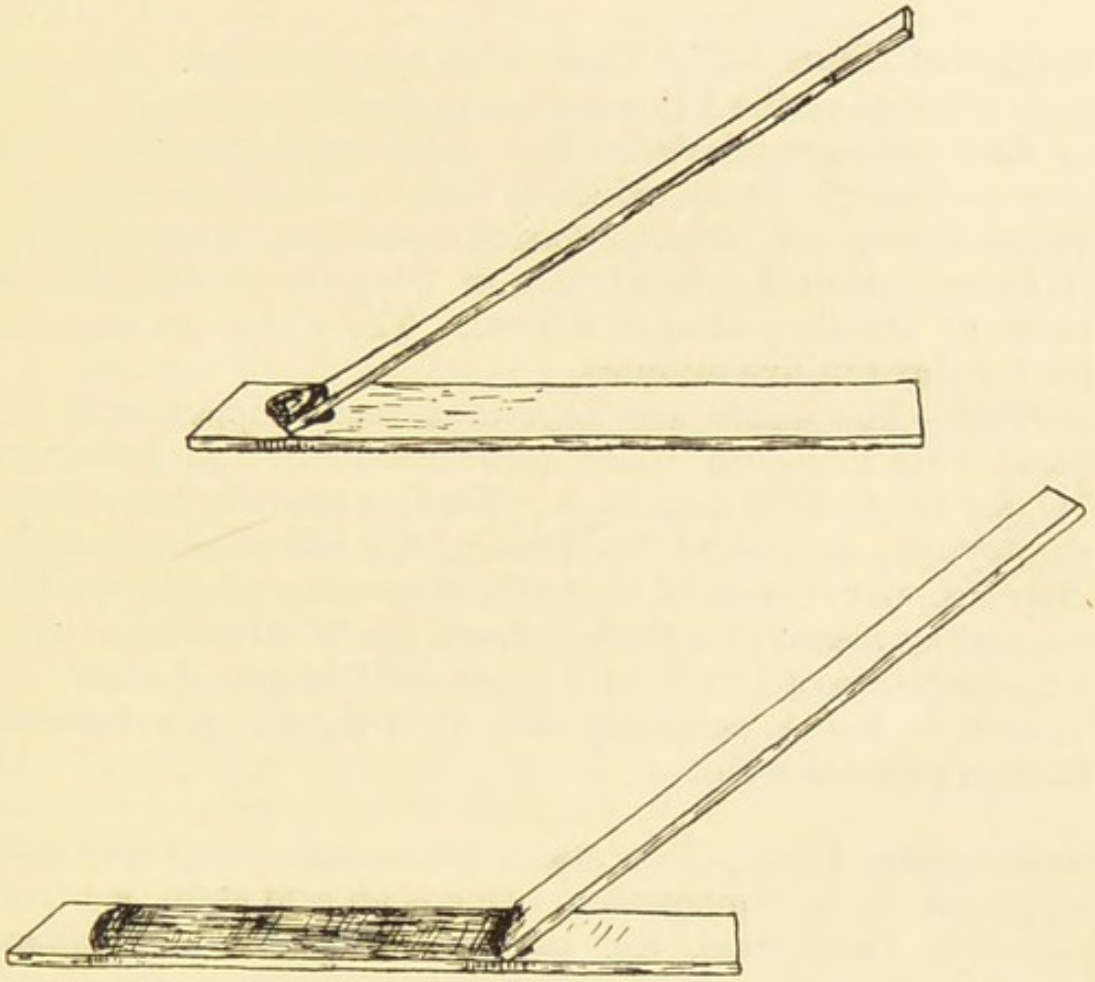


FIG. 46.—The method of making a blood film on a slide.

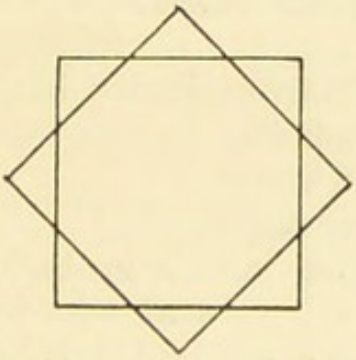


FIG. 47.—Method of making blood films on cover-glasses.

cover-glass, held by its margins between the fingers or by a pair of forceps. This cover-glass is at once laid on another in the manner shown in Fig. 47. The blood quickly spreads out between the two glasses, which are then quickly slid apart. There must be no pressure applied to the cover-glasses after they

are once in contact, and in sliding them apart the one must not be lifted off the other.

The two films thus prepared are allowed to dry in the air. If they are not too thick, they will dry in the course of a few seconds.

Fixation of Films.—The films must now be fixed. Of many methods of fixation, the following are in general use:—

(*a*) *Pure Methyl Alcohol.*—The slides are immersed for five minutes in methyl alcohol, which is kept in a wide-mouthed, well-stoppered bottle. A more prolonged immersion does no harm.

(*b*) *Formol Alcohol.*—A mixture of 10 parts of formalin and 90 parts of absolute alcohol is used. The slides are immersed in this for three to five minutes.

(*c*) *Heat.*—The slides are kept in a small copper oven at a temperature of 120°–125° C. for three-quarters of an hour, or of 135° C. for ten to fifteen minutes. Heating can also be performed by employing a special "toluol heater," the slides being placed on the upper copper surface of the bath, containing toluol; the lid is closed, and by means of a Bunsen flame the toluol is kept boiling (at a temperature of 110° C.) for the desired length of time.

Fixation by heat is necessary only when the film is to be stained by Ehrlich's triacid stain.

Staining the Film.—Two stains are usually employed either in succession or in combination. One is an acid stain, *e.g.* eosin, the other a basic stain, *e.g.* methylene-blue. Many different staining methods are in common use. We shall describe the following:—

(*a*) Eosin and Methylene-blue.—The film, having been fixed in methyl alcohol, is

1. Stained for three to five minutes in ½ per cent. solution of eosin in 60 per cent. alcohol.
2. Washed in distilled water.
3. Dried between filter-paper.
4. Stained for half to one minute in the following mixture freshly prepared:—20 drops of a ¼ per cent. aqueous solution of methylene-blue and 10 drops of the ½ per cent. eosin solution.
5. Washed in distilled water.
6. Dried in the air and mounted in xylol balsam.

(*b*) Jenner's Stain.—This is a compound eosin and methylene-blue stain, and is one of the best for general use. The stain [1] is

[1] Jenner's stain is prepared by mixing in an open basin equal parts of a 1·2 per cent.–1·25 per cent. solution of Grubler's water soluble yellow eosin

dissolved in methyl alcohol (0·5 grm. of the powder in 100 c.c. of methyl alcohol), and fixation and staining are therefore performed simultaneously, which is a great advantage. The procedure is as follows :—

1. The air-dried film is treated with the stain for one to three minutes. If slides are employed, they are immersed in the stain contained in a wide-mouthed bottle, which is kept stoppered to avoid evaporation and precipitation. If cover-glasses are used, the stain having been poured over them, they are covered with a watch-glass.

2. After staining for one to three minutes, wash the film at once in distilled water for five to ten seconds, *i.e.* until it acquires a pink colour.

3. Dry the film in the air and mount in xylol balsam.

In films fixed and stained by either of these methods, the red corpuscles are stained a terra-cotta colour, nuclei are blue, neutrophile granules are red, eosinophile granules bright rose red, and bacteria and parasites blue. Basophile granules appear dark violet in films treated with Jenner's stain.

(*c*) Leishman's Modification of Romanowsky's Method.—The solution used is 0·15 grm. of the dry powder [1] in 100 c.c. of pure methyl alcohol.

1. The air-dried film is evenly covered with a few drops of the stain and is left exposed to the air.

2. After half a minute add twice as much distilled water, and mix it with the stain by gently moving the slide. The staining is continued for five minutes.

3. Wash the film in distilled water, and then allow a few drops of the water to remain on the film for one minute in order to intensify the staining, to remove remains of the deposit, and to alter the tint of the red corpuscles from a greenish-blue to pale pink.

4. Dry the film without heating and mount in xylol balsam.

The red corpuscles are stained pale pink, the nuclei of leuco-

in distilled water and of a 1 per cent. solution of methylene-blue, also in distilled water. The mixture is left for twenty-four hours, then filtered, the residue is dried, collected, powdered, shaken up with distilled water, washed on a filter, again dried and powdered.

[1] Leishman's stain is prepared by taking a 1 per cent. solution of methylene-blue in distilled water, rendering it alkaline by the addition of a 0·5 per cent. solution of sodium carbonate, heating it to 65° C. for twelve hours, and leaving it at the room temperature for ten days. Methylene-azure is the stain formed by the addition of the alkali to the methylene-blue solution. Equal parts of this solution and of 0·01 per cent. aqueous solution of eosin are then mixed, and allowed to stand for twelve hours, being occasionally stirred. The precipitate is collected, washed with distilled water until of pale blue tint or colourless, and then dried and powdered.

cytes are ruby red, those of nucleated red corpuscles appear almost black, the granules of the polymorphonuclears and neutrophile myelocytes are stained red, eosinophile granules are pale pink, basophile granules are purple black.

(*d*) Ehrlich's Triacid Stain.—The stain is a neutral mixture of one basic and two acid stains. It is prepared by taking successively

Saturated aqueous solution of orange G	13–14 c.c.
,, ,, ,, acid fuchsin	6– 7 ,,
Distilled water	15 ,,
Absolute alcohol	15 ,,
Saturated aqueous solution of methyl-green	12.5 ,,
Absolute alcohol	10 ,,
Glycerine	10 ,,

After adding the methyl green the mixture is continuously shaken. The stain can be obtained prepared as above.

The film, fixed by being heated to 120°–125° C. for from thirty minutes to one hour, or to 135° C. for ten to fifteen minutes, is covered with the stain for five minutes, washed in water, dried, and mounted in xylol balsam.

The red corpuscles are stained of orange colour, nuclei are pale green or pale greenish-blue, the cytoplasm of the lymphocytes and large mononuclear leucocytes is reddish-yellow, neutrophile granules are violet, eosinophile granules are orange or copper-coloured. Basophile granules are not stained.

(*e*) Staining of Films for detection of the Iodophilic Reaction.—The film is allowed to dry in the air, but is not fixed. Then a drop of the following solution is applied :—

Iodine	1·0 grm.
Potassium iodide	3·0 ,,
Distilled water	100·0 c.c.

Gum arabic; enough to make the fluid of a thick syrupy consistence.

A cover-glass is lowered on to the preparation. A minute later the surplus fluid is removed by filter paper and the preparation is ready for microscopic examination.

An alternative method is to leave the slide for a few minutes in a wide-mouthed stoppered bottle containing iodine crystals, and thereafter to apply a drop of saturated lævulose solution and cover-glass.

DESCRIPTION OF PLATE I.

Blood corpuscles drawn to scale from films stained by Jenner's stain. The corpuscles of normal blood are those numbered 1, 15, 16, 17, 18, 19, 20, 21 and 22. The others are pathological forms.

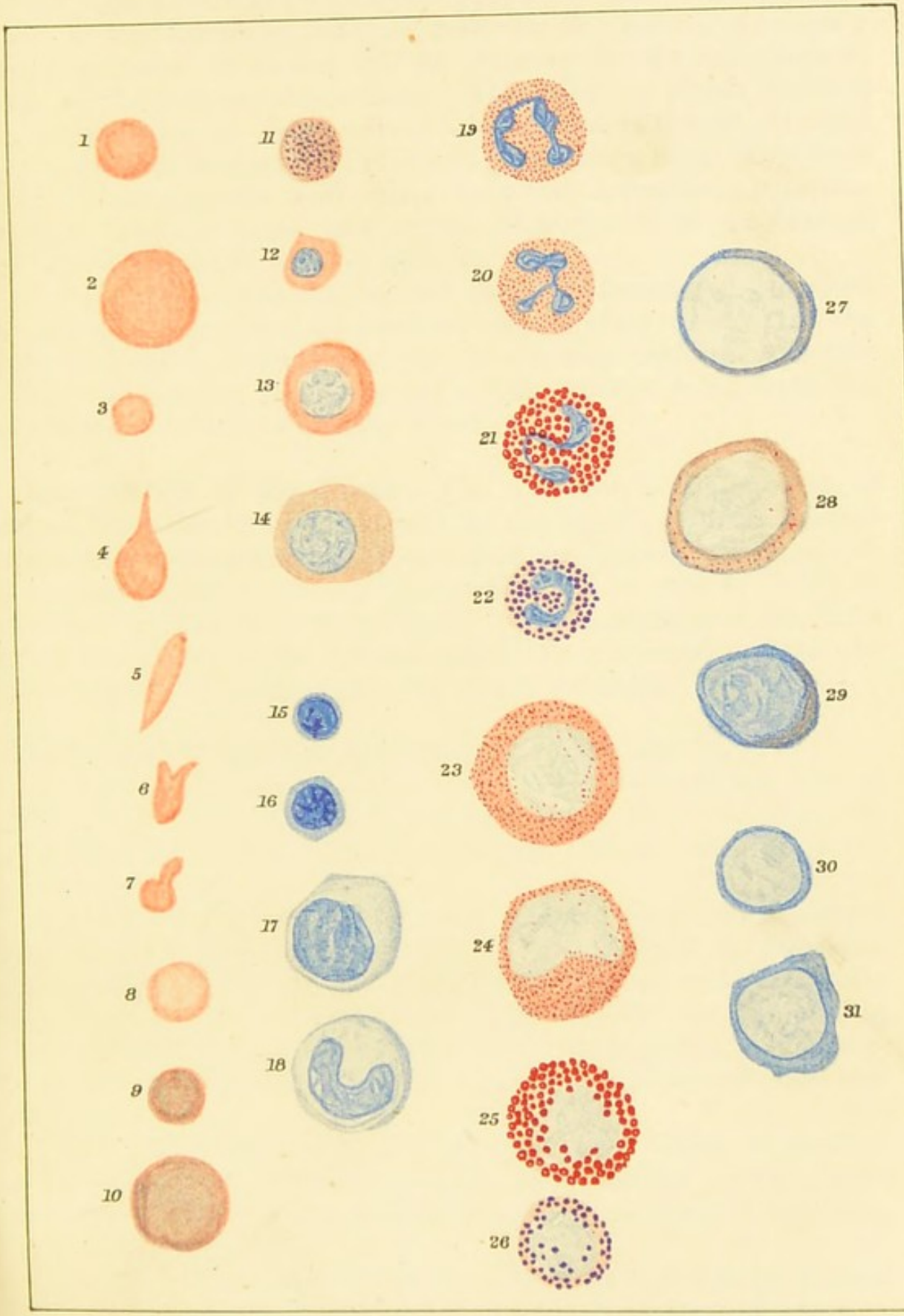
RED BLOOD CORPUSCLES.

1. Normal erythrocyte.
2. Macrocyte.
3. Microcyte.
4, 5, 6 and 7. Poikilocytes.
8. Pale red corpuscle from a case of chlorosis.
9. Polychromatophilic erythrocyte.
10. Polychromatophilic macrocyte.
11. Erythrocyte with granular degeneration.
12. Normoblast.
13. Megaloblast.
14. Polychromatophilic megaloblast.

LEUCOCYTES.

15 and 16. Lymphocytes of normal blood.
17. Large mononuclear leucocyte.
18. Transitional leucocyte.
19 and 20. Neutrophile polymorphonuclear leucocytes.
21. Eosinophile polymorphonuclear leucocyte.
22. Mast cell.
23 and 24. Neutrophile myelocytes.
25. Eosinophile myelocyte.
26. Basophile myelocyte.
27 and 28. Lymphoid marrow cells. 28 is a transitional form between lymphoid marrow cell and myelocyte.
29. Stimulation cell of Türk.
30 and 31. Lymphoid cells from a case of acute lymphatic leucocythæmia

PLATE I.



To face

The red corpuscles after such treatment with iodine appear yellow. In health the leucocytes appear of pale lemon yellow tint. If the iodophilic reaction, however, be present, mahogany brown granules or masses will be observed in the cytoplasm of some of the polymorphonuclears. This reaction, which depends on the presence in those cells of a loose combination of glycogen with a proteid, is observed in films of pus, and in blood films from cases of suppuration and other bacterial infections, toxæmia, anæmia, leucocythæmia, and severe disturbance of respiration, *e.g.* the dyspnœa of cardiac disease.

If fresh moist films be exposed to iodine fumes and examined immediately thereafter, the reaction may be observed even in the blood of healthy individuals; but in contrast with the reaction obtained in disease, the abnormal colour quickly fades from the polymorphonuclears of healthy blood.

Examination of the Films.—The microscopic examination of stained films should be performed by daylight. A magnification of about 500 diameters will often suffice for diagnosis, but an oil-immersion objective, giving a magnification of 800 to 1000, is necessary for a study of finer details. Those portions of the films where the corpuscles lie isolated should be examined; where the red corpuscles lie in rouleaux, the film is too thick.

Red Blood Corpuscles (Erythrocytes) (see Plate I.).

1. *Size.*—Even in health there is some difference in the size of the red corpuscles, but their average diameter is 7–7·5 μ. In anæmia, marked alterations in the size of the corpuscles are frequently seen.

Macrocytes (Plate I., 2) are red corpuscles of normal shape, but excessive size. They measure from 10–14 μ in diameter. Those which stain well are regarded as young red corpuscles which, while still immature, have passed into the circulating blood. Macrocytes which contain little hæmoglobin, and therefore stain feebly, are probably, however, degenerated red corpuscles.

Microcytes (Plate I., 3) are red corpuscles which, though of normal shape, are distinctly smaller than normal erythrocytes. They are frequently 3–6 μ in diameter, and arise from the shrinking of red corpuscles.

2. *Shape.*—The shape of the red corpuscles is apt to be altered in severe chlorosis and all forms of anæmia. All sorts of irregular forms (*Poikilocytes*) may then be encountered, the corpuscles appearing pear-shaped, kidney-shaped, flask-shaped, etc. (Plate I.,

4, 5, 6 and 7). Crenation of the red corpuscles must not be mistaken for poikilocytosis.

3. *Colour.*—In the stained film the relative amount of hæmoglobin in the corpuscles is recognised by the intensity of their coloration and the size of the central depression. If the corpuscle be of normal size, its central depression is increased in proportion to the deficiency of its hæmoglobin (Plate I., 8).

In disease the red corpuscles may have acquired an affinity for basic as well as for acid stains, and therefore appear of a colour varying from violet to blue. This alteration, known as *Polychromatophilia* or *Polychromasia* (Plate I., 9 and 10), is observed in degenerating erythrocytes, but more frequently in young erythrocytes, and especially in macrocytes and erythroblasts.

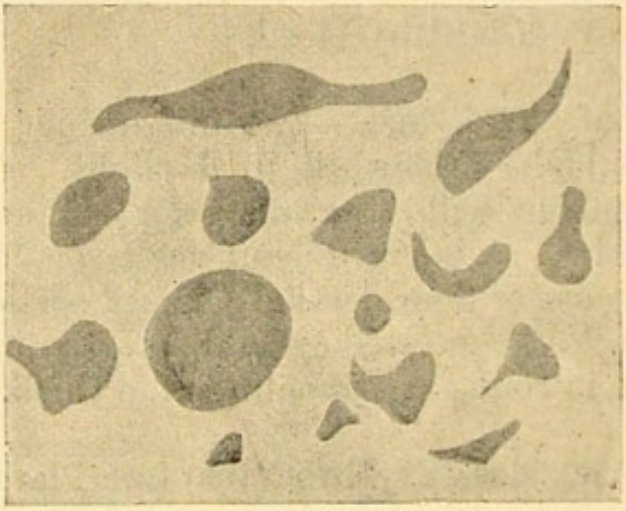


Fig. 48.—Poikilocytosis.

Granular Degeneration of Red Corpuscles.—Fine basophile granules may be observed scattered through some of the red corpuscles (Plate I., 11). The granules appear blue in films stained with eosin and methylene blue or with Jenner's stain, but are unstained by Ehrlich's stain. They are observed in anæmia, leucocythæmia, malarial and bacterial infections, and toxæmias, *e.g.* lead poisoning. The alteration is regarded by some as a sign of degeneration of the corpuscles, but the origin and significance of the granules is still a matter of dubiety.

4. *Nucleated red corpuscles* (*Erythroblasts*).—There are two forms of nucleated red corpuscles, namely the normoblast and megaloblast, both of which are found in normal bone marrow, but not in the normal blood.

The Normoblast is a nucleated red cell of normal size (Plate I., 12). The nucleus, which is usually circular or ovoid, stains deeply with the basic stain, and is frequently excentrically situated. The cytoplasm is not seldom polychromatophilic.

The Megaloblast (Plate I., 13) is a nucleated red corpuscle of excessive size. It is often three times as large as a normal red corpuscle. Another important characteristic of the megaloblast is the pale tint of its nucleus. The cytoplasm is frequently polychromatophilic (Plate I., 14).

The detection of nucleated red corpuscles in the blood is evidence of a pronounced affection of the bone marrow, from which immature red corpuscles are passing into the blood. Such a process of regeneration of red corpuscles is observed in severe anæmias, myelogenous leucocythæmia, and malignant disease of the bone marrow. In most of these diseases the chief erythroblast is the normoblast. In pernicious anæmia the megaloblast is more common, but it is not pathognomonic of that disease.

Leucocytes.—A study must be made of the various forms of leucocytes in the films, noting their size and shape, their nuclei, and the characters of any granules in their cytoplasm. Cytoplasmic granules are termed basophile when they have a marked affinity for basic stains, oxyphile or eosinophile when their affinity is for acid stains, and neutrophile when their affinity is for neutral stains. **A differential count** of the leucocytes must be made. With this object a large number, several hundreds, of leucocytes are counted. A mechanical stage is of great assistance in this enumeration.

The following list indicates the varieties of leucocytes and the proportion which each bears, under normal conditions, to the total number of leucocytes in the blood :—

Lymphocytes	20–25 per cent.
Large mononuclears and transitional leucocytes	3– 5 ,, ,,
Neutrophile polymorphonuclears .	70–75 ,, ,,
Eosinophile polymorphonuclears .	½– 4 ,, ,,
Mast cells	¼– ½ ,, ,,

Other forms of leucocytes may be observed in disease, and are considered on pages 116, 117.

1. Lymphocytes (Plate I., 15 and 16).—These are no larger than red blood corpuscles. Each has a circular nucleus which is deeply stained with the basic stain. Around the nucleus is a narrow zone of basophile cytoplasm which is not granular, except in films stained by Romanowsky's method or its modifications. The lymphocytes are enormously increased in lymphatic leucocythæmia, where the number of leucocytes may exceed 400,000, and 95 to 99 per cent. of these be lymphocytes.

2. Large Mononuclear Leucocytes.—These are from two to three times the size of a red corpuscle, have a large ovoid or circular pale nucleus in which a chromatin network is recognisable (Plate I., 17). Around the nucleus is a considerable extent of still paler, sometimes almost colourless, cytoplasm, which, like that of the lymphocytes, is non-granular.

Leucocytes having similar characters, except that the nucleus is indented or horse-shoe shaped, are termed transitional leucocytes (Plate I., 18).

3. Neutrophile Polymorphonuclear Leucocytes are usually termed *Polymorphs* (Plate I., 19 and 20). These are almost twice as large as the lymphocytes. Each has a multilobular irregular nucleus, which is frequently shaped like the letters S or U, and which stains deeply with the basic stain. The cytoplasmic granules are fine and neutrophile; by Jenner's stain they are red. In inflammatory and post-hæmorrhagic leucocytosis, and in that of malignant disease, these leucocytes are absolutely and relatively increased.

4. Eosinophile Polymorphonuclear Leucocytes, usually termed *Eosinophiles*.—These are slightly larger than the polymorphs. The nuclei are multilobular and stain less deeply than those of polymorphs, and the cytoplasm contains large spherical, refractile, oxyphile (eosinophile) granules (Plate I., 21). The eosinophiles are absolutely and relatively increased in cases of infection with animal parasites, of malignant disease, of many skin diseases, and during the stage of subsidence of acute inflammatory leucocytosis. The eosinophiles are absolutely but not always relatively increased in myelogenous leucocythæmia.

5. Mast Cells (Plate I., 22) are leucocytes somewhat smaller than polymorphs, each containing a multilobular, polymorphic, pale nucleus and cytoplasmic granules which are large and basophile, *i.e.* which stain with the basic stain. These granules are readily soluble in water, and are consequently not stained in films treated by eosin and methylene-blue or by Ehrlich's stain. But they are well seen when the stain used has been dissolved in methyl alcohol, *e.g.* Jenner's stain (see p. 110).

The mast cells are increased in myelogenous leucocythæmia.

Pathological Leucocytes.—These are cells never detected in normal blood, although some of them are normally present in the bone marrow as precursors of the leucocytes.

1. Myelocytes.—The essential characteristics of a myelocyte are that it is a cell with a single (not multilobular) nucleus and granular cytoplasm. Myelocytes differ in size; some are three times the size of a red corpuscle, others are not larger than

a polymorph. The nucleus is large, circular, ovoid or reniform, and stains less intensely than the nucleus of a polymorph. The granules in the cytoplasm are usually fine and neutrophile (neutrophile myelocytes, see Plate I., 23 and 24), but may be larger and eosinophile (eosinophile myelocytes, Plate I., 25), or basophile (Plate I., 26). Myelocytes with basophile granules are not larger than polymorphs.

Myelocytes constitute about 50 per cent. of the total leucocytes in cases of myelogenous leucocythæmia. In scanty numbers they may be detected in other diseases, *e.g.* pernicious anæmia and other conditions in which the bone marrow is markedly affected.

2. Lymphoid Marrow Cells. — These cells (Plate I., 27) differ, in the same manner as myelocytes, in size, but are usually about twice the size of a red corpuscle. The single, circular, or ovoid nucleus is pale and apparently structureless. It nearly fills the cell, and around it is a thin zone of non-granular cytoplasm, which is markedly basophile, and the outer edge of which is frequently irregular.

These cells are found in the blood in large numbers in some cases of myelogenous leucocythæmia, and are regarded as cells which have reverted to the embryonic type of cell from which myelocytes, red corpuscles, and leucocytes are descended. The lymphoid marrow cells have many synonyms: myeloblasts, indifferent lymphoid cells, etc.

In films of leucocythæmic blood containing these cells, others will be found representing all transitional stages between lymphoid marrow cells and myelocytes, *i.e.* cells which closely resemble the former, but the cytoplasm being less obviously basophile and containing a variable number of very fine granules (Plate I., 28).

3. Stimulation Cells of Türk.—These differ from lymphoid marrow cells in that the nucleus is smaller and stains better, and that the non-granular cytoplasm is intensely basophile and more deeply stained than the nucleus (Plate I., 29). These cells, which are derived from the bone marrow, may be found, usually with a few myelocytes and nucleated red corpuscles, in the blood after prolonged leucocytosis and in severe anæmia with cachexia.

4. Lymphoid Cells (Plate I., 30 and 31). — These closely resemble the lymphoid marrow cells in all but one respect—namely, that none manifest a transition to the granular myelocyte, *i.e.* none contain any granules in their cytoplasm. The entire absence of granules is best studied in films stained by Ehrlich's stain.

The lymphoid cells are derived from the fixed cells of

Flemming's germ centres in lymphadenoid tissue, and constitute from 50–99 per cent. of the leucocytes in large-celled lymphatic leucocythæmia (leuco-sarcomatosis).

PARASITES IN THE BLOOD

Filariæ.—*Filaria sanguinis hominis* (*Filaria nocturna*) is the embryo of the nematode worm Filaria bancrofti, which inhabits the lymphatic vessels. The embryos are found in the blood of persons suffering from chyluria, elephantiasis, lymph-scrotum, and

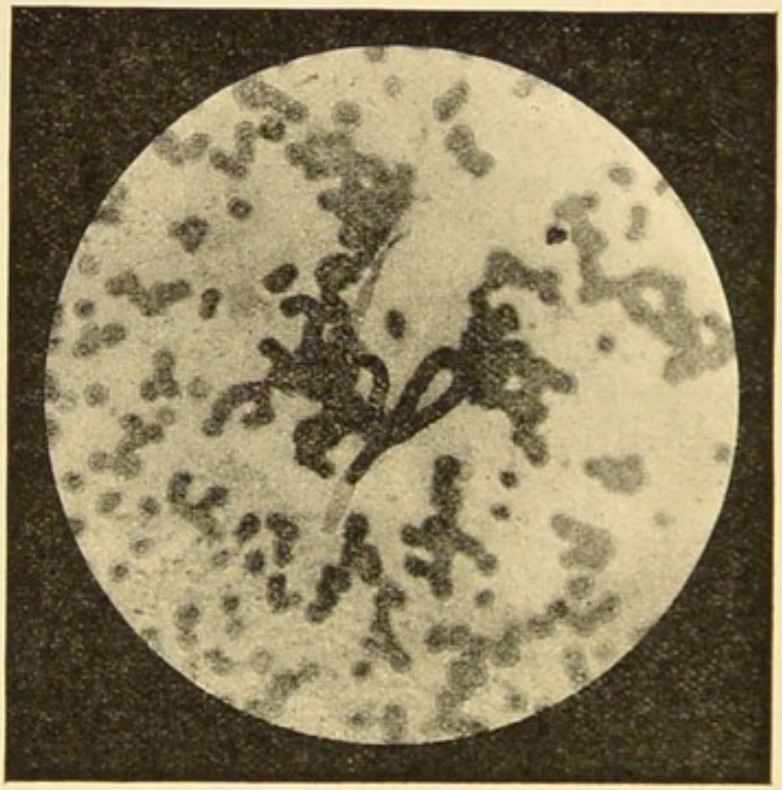


FIG. 49.—Blood film : *Filaria nocturna* retracted at both ends within its membrane (× 200).

other manifestations of Filariasis, the embryos appearing in the peripheral blood during the night, but disappearing therefrom during the day. For diagnosis the blood is examined during the early part of the night. Films are made, and stained with Jenner's or Leishman's stains, or with dilute fuchsin, methylene-blue, or gentian-violet. On microscopic examination of the preparations (Fig. 49), Filaria nocturna is seen to be a small worm, 270–340 μ long, by 7–11 μ broad, enclosed in a delicate membrane. In fresh blood preparations, the embryos are colourless and wriggle about, displacing the red corpuscles in their vicinity. The movements become more sluggish in the course of a few hours, and the shape of the worm is then more readily seen.

Filaria diurna differs from Filaria nocturna only in its periodicity, appearing in the blood during the day and disappearing at night.

Filaria perstans is found in the blood both during the day and night. It is smaller than Filaria nocturna, and in a fresh blood preparation the worm exhibits progressive movements for many hours.

Malarial Parasites.—These may be found on microscopic examination of fresh blood preparations. It is better, however, to examine blood films which are allowed to dry in the air, and then fixed and stained by Leishman's stain (see p. 111) or by *Giemsa's stain*[1] as follows :—

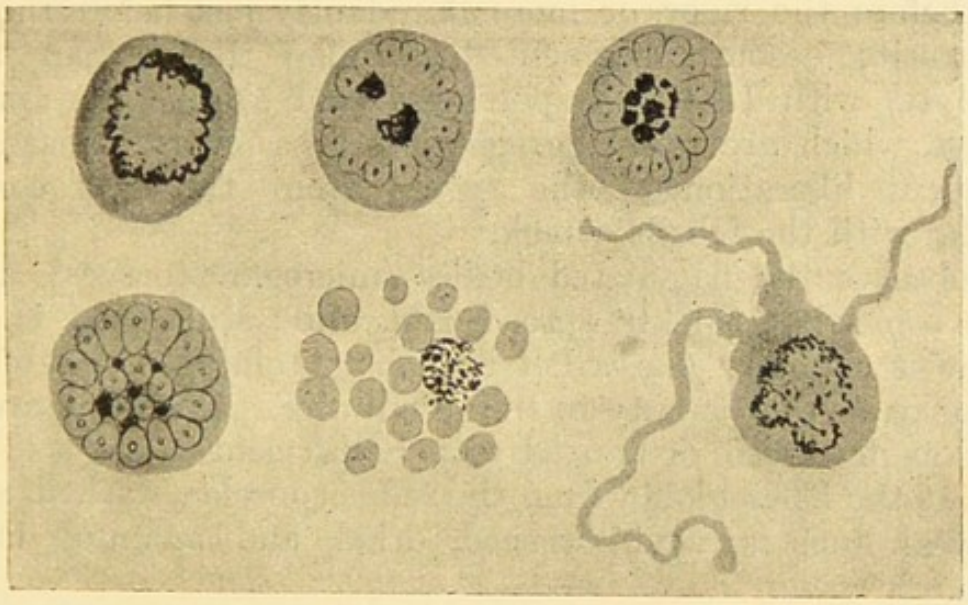


FIG. 50.—Malarial parasites (after Thayer and Hewetson).

1. The film is fixed with methyl alcohol for two to three minutes ; the excess is then removed by filter paper.
2. To 1 c.c. of distilled water, warmed to 30°–40° C., add one drop of Giemsa's stain.
3. Pour the freshly prepared dilute stain over the film, and leave it for ten to fifteen minutes.
4. Wash the film in a stream of water.
5. Dry and mount in xylol balsam.

The stained films are examined under a $\frac{1}{12}$-inch oil immersion objective.

[1] *Giemsa's stain* : 3·0 grm. of Azur II—Eosin and 0·8 grm. of Azur II are dried over sulphuric acid in an exsiccator, powdered, rubbed through a silk sieve, and dissolved in 250 c.c. of pure glycerine at 60° C. Add 250·0 c.c. of methyl alcohol, previously heated to 60° C., shake, leave at the room temperature for twenty-four hours, and filter.

The malarial parasites are unicellular, nucleated, and motile sporozoa. Their appearance in stained films depends not only on the species present, but on the stage in the cycle of development which has been reached at the time when the patient's blood was examined (Plate II.).

The Benign Tertian Parasite (*Plasmodium vivax*, *Hæmamœba vivax*).—The youngest form is a spheroidal or irregular body about 2 μ in diameter situated within a red blood corpuscle. In films stained as directed, the body of the parasite is blue, its nucleus red. A few hours after the rigor, the next stage—the ring form—is found, and the infected red corpuscle is swollen. The parasite then develops into a spheroidal pigmented body, which nearly fills the red corpuscle. Still later, three or four hours before the rigor, or more abundantly just before the rigor, the parasite becomes segmented, forming the tertian rosette (sporocyte) with 15 to 23 spores grouped around the pigment granules, which are now aggregated into a central mass. The subsequent liberation of the spores from the red corpuscle coincides with the febrile attack.

To observe the flagellated bodies (microgametoctyes), a blood film is somewhat thickly spread, and while still moist is placed in a moist chamber (*e.g.* a Petri's dish containing strips of moist blotting paper) for twenty to thirty minutes. The films are then stained as described, or treated with 10 per cent. acetic acid to dissolve out the hæmoglobin from the red corpuscles, washed, stained with weak fuchsin, again washed, dried, and mounted in xylol balsam.

The Quartan Parasite (*Plasmodium malariæ*, *Hæmamœba malariæ*) exhibits developmental changes similar to those of the benign tertian parasite. The main differences are that the cycle of development of the quartan parasite is completed in seventy-two hours, the infected red corpuscles are not decolorised nor swollen, the pigment granules are coarser and darker, and the number of spores in each rosette is from 6 to 12.

Malignant, Tropical, or Æstivo-autumnal Fever.—In the peripheral blood, there are found small and large signet-ring forms, and, about a week after the onset of the fever, pigmented crescents, the latter being early forms of the sexual cycle. Rosette forms (sporocytes) with 18 to 21 spores are found in the blood of the internal organs, rarely in the peripheral blood. If blood containing crescents be examined fresh under a cover glass, the crescents are seen to alter in shape, becoming spherical, and from some of the spheres (the microgametocytes) motile flagellae may be seen to develop and to agitate the adjacent red corpuscles.

DESCRIPTION OF PLATE II.

MALARIAL PARASITES.

Films stained by Romanowsky's method (×800) (after Bertrand and Klynens).

1. Simple Tertian Fever.—The parasite is young, of ring-shape, and stained blue; its nucleus is stained red.

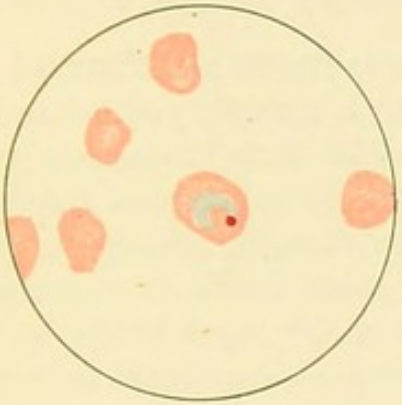
2. Simple Tertian Fever.—The cell containing the parasite (twelve to fifteen hours old) is enlarged and dotted over with fine red granules (Schüffner's dots), which are not found in other forms of malaria.

3. Simple Tertian Fever about Twenty-four Hours after a Paroxysm.—The parasite is pigmented. At the upper part of the figure there is a polychromatophilic red cell.

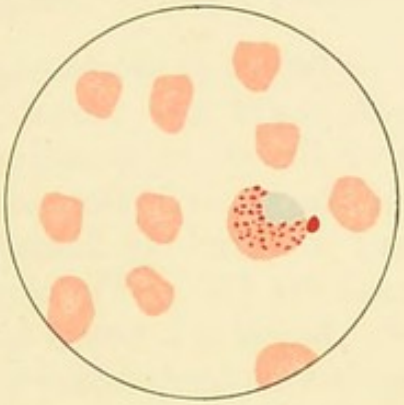
4. Simple Tertian Fever.—Segmentation of the parasite.

5. Simple Tertian Fever.—Gamete within a red cell. The parasite is pigmented and has a single nucleus.

6. Malignant Tertian Fever.—Female crescent (macrogamete).



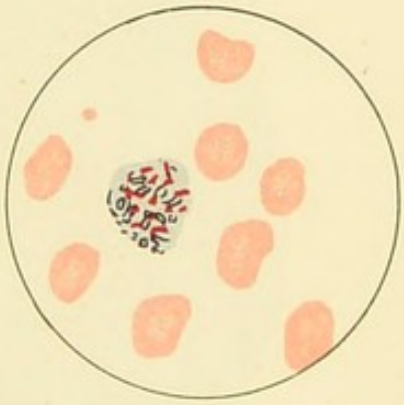
1



2



3



4



5



6

To face page 12

Malarial parasites must not be confounded with deposits of stain employed, vacuoles in red corpuscles, granular degeneration of red corpuscles, pigment in leucocytes, or blood plates.

Spirochæta obermeieri.—This spirochæte is found in the blood of patients suffering from relapsing fever. On examining a fresh preparation of such blood with the microscope, it will be seen that the red corpuscles are agitated by the actively motile spirochætes. For diagnosis, films are made and stained by Jenner's, Leishman's, Giemsa's, or other stain. The parasites (Fig. 51) are then seen to be of spiral form, 20 to 40 μ long and about 1 μ broad. When the blood is examined in cases of relapsing fever, these spirochætes begin to appear shortly before the temperature rises. On the second or

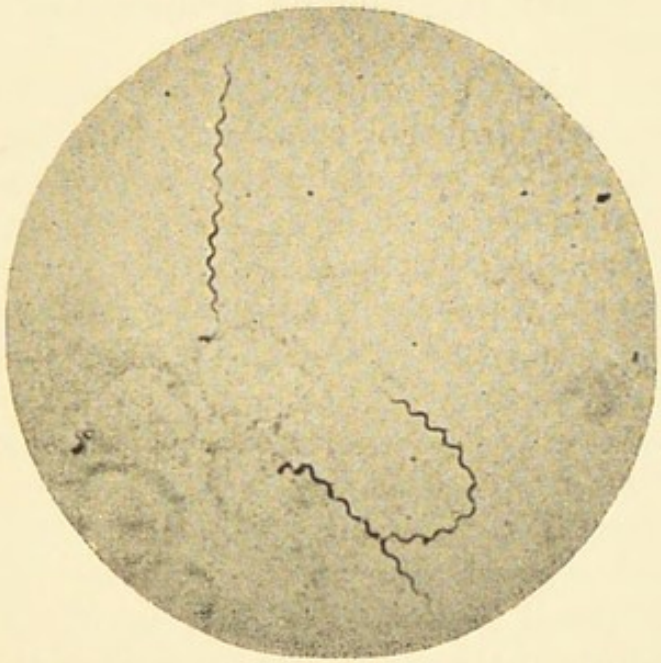


FIG. 51.—*Spirochæta obermeieri* in a blood film (× 1000) (after Günther).

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.

Specific Gravity.—This is tested by Hammerschlag's modification of Roy's method. A mixture of chloroform and benzol is made in such proportions as to have a specific gravity of about 1059. A small drop of blood is transferred from a capillary tube—*e.g.* the Thoma hæmocytometer pipette—to this mixture. 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 showing that the specific gravity of the liquid is just that of the blood. By means of an ordinary urinometer graduated up to 1065, this specific gravity may then be readily ascertained.

The normal specific gravity varies from 1058 to 1065. It depends largely on the amount of hæmoglobin in the blood, and is therefore much reduced in anæmia.

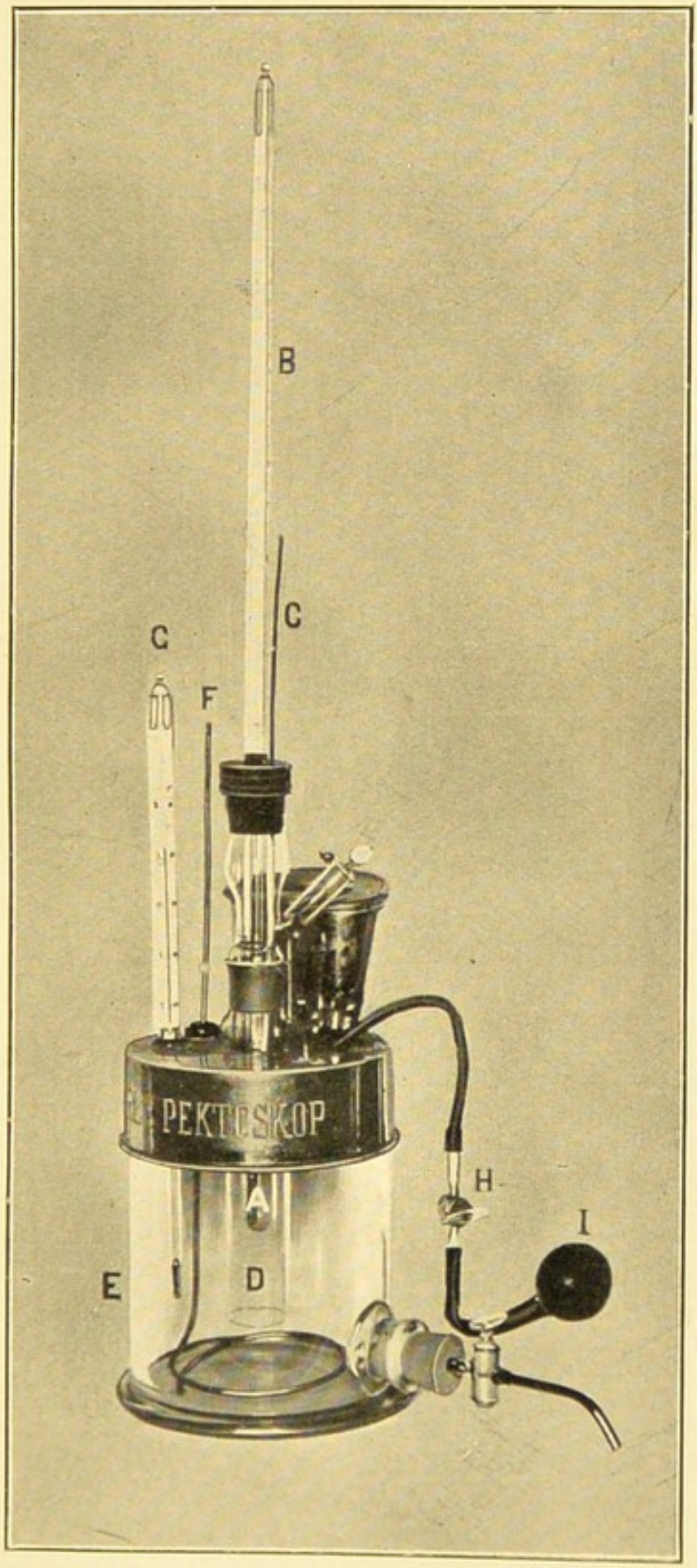
Cryoscopy.—The freezing-point of the blood is determined by means of the cryoscope. The apparatus of Beckmann, Lindemann, or Zikel are most frequently used. The following description applies to Zikel's cryoscope.

The cylindrical glass tube (A) (see Plate III.) contains the fluid to be tested, a thermometer (B) graduated in 0·01° C., and a stirrer (C). This tube is contained within a cylinder (D), which affords an air-mantel between the inner tube and the freezing mixture in the jar (E). The freezing mixture can be stirred by the stirrer (F), and its temperature ascertained by the thermometer (G).

Method.—The exact freezing-point of distilled water must first be determined on each occasion. Fill the jar (E) with a mixture of chipped ice, salt and water, so that the thermometer (G) records a temperature of from − 5° C. to − 6° C. Open the stopcock (H). Remove the inner tube (A) and fill it with distilled water sufficient to cover the bulb of the thermometer (B). Replace the tube within the cylinder (D), and from this moment onwards continue stirring by means of the stirrer (C). The mercury quickly falls in the thermometer (B), and when it has fallen to near the freezing-point, quickly compress the bulb (I) several times, and then close the stopcock (H). The freezing mixture is thereby blown out of the cylinder (D), and an air-mantel thus interposed between the fluid in the inner tube and the freezing mixture in the jar. The mercury in the thermometer (B) now falls more slowly, then rapidly rises and remains stationary at a certain point—the freezing-point—which is read off with the aid of a lens.

The tube (A) is then removed, dried, and filled with blood sufficient to cover the bulb of the thermometer. To obtain sufficient blood, a superficial vein of the forearm should have been punctured in the manner described on p. 465. The freezing-point of the blood is then determined in the same manner as in the preliminary determination of the freezing-point of distilled water.

The reading obtained must then be corrected for the difference between the freezing-point of distilled water and 0·00° C. For example, if the blood freezes at − 0·52° C., and distilled water at + 0·05° C., then the actual freezing-point of the blood is − 0·57° C. Again, if the blood freezes at − 0·58° C., and distilled water at − 0·02° C., then the freezing-point of the blood is − 0·56° C.



Zikel's cryoscope.

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The freezing-point of normal blood is in health constant between $-0{\cdot}55^{\circ}$ C. and $-0{\cdot}57^{\circ}$ C. It is lowered, perhaps to $-0{\cdot}71^{\circ}$ C., when the excretory functions of the kidneys are impaired, as in nephritis and uræmia, and also in conditions of cyanosis from cardiac disease, pneumonia, etc. Such an alteration in the freezing-point of the blood may be found when either one or both kidneys are affected by organic disease.

Williamson's Test for Diabetic Blood.—To 1 c.c. of water in a small test-tube add 20 c.mm. of blood (measured by means of the pipette of Gowers' hæmoglobinometer), 1 c.c. of aqueous

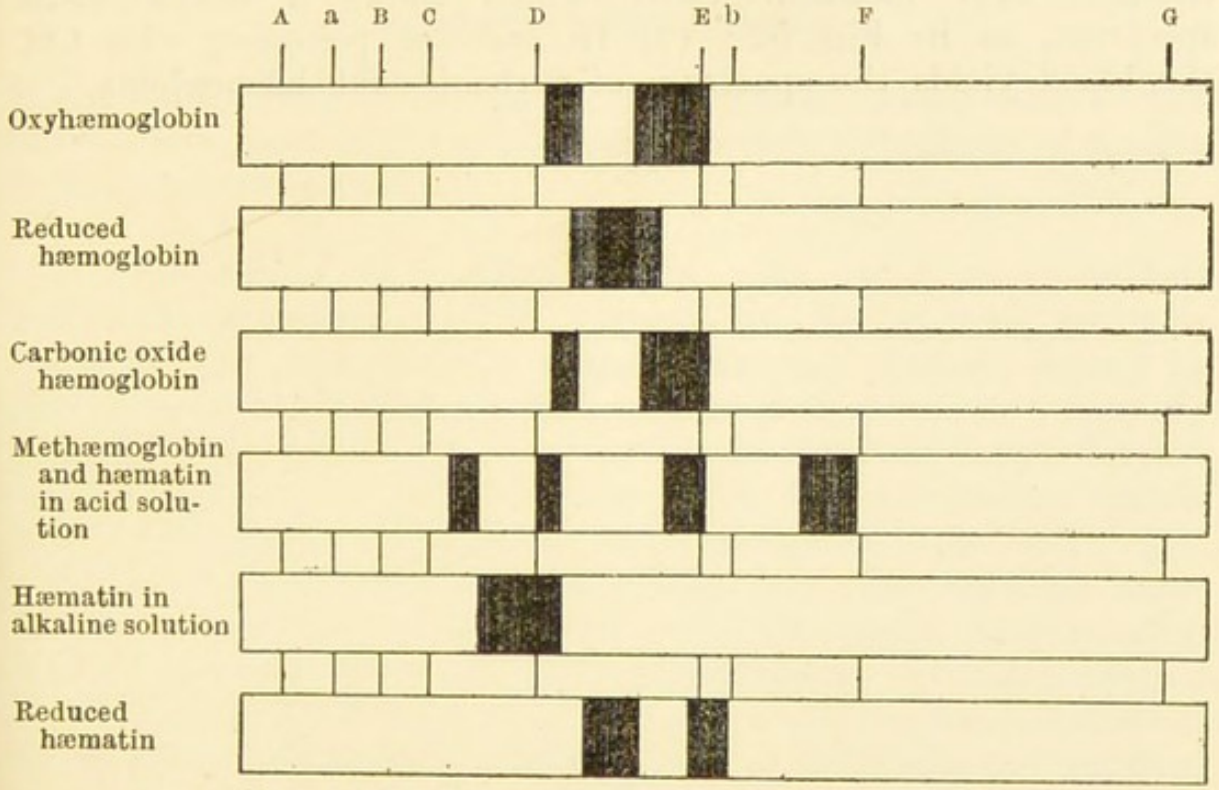


FIG. 52.—Absorption spectra of blood pigment.

solution of methylene-blue (1 : 6000), and 40 c.c. of liquor potassæ. The tube is now shaken, and then allowed to remain in a water-bath at 100° C. for four minutes. If the reaction be positive, the colour of the fluid is changed to a dirty pale yellow.

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. 52.

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 shows a characteristic spectrum, as in Fig. 52; (2) In cases of poisoning with CO, the blood yields the spectrum of carbonic-oxide-hæmoglobin.

CHAPTER VI

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.**—Pain of cardiac origin may be a more or less prominent symptom. It is frequent in the chronic toxæmia resulting from the abuse of tobacco, tea, and alcohol; it may be due to gout and infective diseases, and it is common in neurotic persons, and particularly in women. In organic heart disease, and in particular in disease of the aortic valves, coronary vessels and myocardium, pain is a not infrequent symptom. The abnormal afferent impulse, proceeding from the diseased heart to certain segments of the spinal cord, stimulates neighbouring sensory neurones belonging to those segments. That stimulation is referred, as Head says, "on the surface of the body instead of on to the organ actually affected," and so gives rise to "referred pain" which is segmental in distribution (see p. 352).

Cardiac pain in its most pronounced form—angina pectoris—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 may be of the most intense character and accompanied by a sense of impending death, radiates from the heart to the shoulders and down the ulnar side of the left arm (less frequently 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.

In every case of cardiac pain careful search should be made for

tactile hyperæsthesia, which is frequently co-extensive with the area in which pain is felt.

2. **Palpitation.**—The abnormal perception of excited pulsation in the heart or aorta is very frequently due to diminution of vagus control, and may be induced by emotional excitement, reflex disturbance, as dyspepsia, flatulence, and floating kidney, by anæmia, nervous debility, toxæmia, as of tobacco, tea, influenza, and exophthalmic goitre, and by physical strain. Palpitation 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, which she may speak of as "bumping," "wobbling," etc., 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.

Palpitation must not be mistaken for trembling of the heart or **Tremor cordis**, in which there is a fluttering sensation, or one as if the heart's action had stopped, with an almost imperceptible radial pulse. This condition is due to a series of extra systoles (see p. 190).

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, with shallow respiration and dilatation of the pupils.

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 symptom is common in 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.

TOPOGRAPHICAL ANATOMY

In order to determine the position of any particular point on the thoracic wall for the purpose of description or record, definite horizontal and vertical landmarks are required.

The Horizontal Landmarks are the ribs, costal cartilages and intercostal spaces. To find any particular rib, we first feel for the *Angulus Ludovici*—the prominent ridge at the junction of the manubrium and the body of the sternum. When the finger is drawn horizontally outwards from that ridge, it passes on to the second costal cartilage and rib. Whilst the forefinger remains pressed on the second rib, the middle finger feels for the second interspace and then for the third rib. Before the middle finger is moved downwards from the third rib, the forefinger should mark its position. Proceeding from rib to rib in this manner, the ribs and interspaces are identified.

Vertical Landmarks.—These are the following:—The *mid-sternal* line; the right and left *lateral sternal* lines; the right and left *mammary* lines, each passing vertically down from the mid-point of the clavicle to the midpoint of Poupart's ligament; the right and left *parasternal* lines midway between the lateral sternal and mammary lines; the anterior-, mid- and posterior-*axillary* lines passing vertically downwards from the anterior fold, the apex, and posterior fold of each axilla; and lastly the two *scapular* lines, passing vertically through the angle of each scapula.

The Relations of the Heart to the Anterior Wall of the Chest.—1. *The base*, formed by the right auricle and the conus arteriosus, lies behind the sternum at the level of the upper border of the third rib (or second intercostal space), and extends on each side half an inch external to the right and left lateral sternal lines. The greater part of the left auricle lies posteriorly, the auricular appendix alone coming to the front at the level of the second left intercostal space.

2. *The right border*, formed by the right auricle, is indicated by a line, convex outwards, joining a point on the upper margin of the third right costal cartilage, half an inch to the right of the

right lateral sternal line, with the sixth right chondro-sternal junction. This border is furthest from the middle line at the level of the fourth intercostal space, being then one and a half inches from the mid-sternal line.

3. *The left border*, formed by the left ventricle, is demarcated by a line, convex outwards, which passes from the upper margin of the third left costal cartilage (half an inch to the left of the left lateral sternal line) to the *apex* beneath the fifth left intercostal space, in the left mammary line.

4. *The lower border*, formed by the right ventricle and to a slight extent by the apical portion of the left ventricle, is indicated by a line passing from the apex to the sixth right chondro-sternal junction.

The præcordia (cardiac or præcordial region) which is thus marked out overlies not only the heart, but also the margins of the lungs which overlap it.

CHAPTER VII

Circulatory System (*continued*)

INSPECTION

On inspection, we study *first* the form of the præcordia, and *second*, the pulsations visible within and outside that region.

I. The Form of the Præcordia.—Whereas the two sides of the normal chest, viewed from in front, are symmetrical, a slight *bulging* of the præcordia is more readily detected by simple inspection than by means of measurement. The bulging 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 by 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 level of 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 also be caused by retraction of the left lung, the result of pleurisy or of fibroid contraction, whilst a mesial localised depression at the level of the sixth, seventh and eighth ribs is observed in cobblers.

II. Pulsations.—1. The Cardiac Impulse.—In health the visible cardiac impulse, due to the forward thrust of the apical portion of the left ventricle during systole, is a gentle impulse, occurring rhythmically about seventy-five times per minute, and visible in the fifth left interspace. The impulse extends about 1 inch horizontally, and extends outwards to a point ½ inch internal to the left mammary line, or 3½ inches from the mid sternal

line. That part of the impulse which is lowest down and furthest from the middle line is termed the *apex-beat.* The impulse just described is the only pulsation visible on the normal chest wall.

In childhood, and in persons who have a short and wide thorax, the cardiac impulse 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, in which direction the deflection may amount to 2 inches. Fixation of the apex-beat, so that its position is altered neither by deep respiration nor by change of posture, is one of the signs of chronic mediastino-pericarditis.

The cardiac impulse does not always make itself visible on the chest wall. This is usually due to great development of fat or muscle in the parieties, or to pulmonary emphysema. In such cases we can by palpation almost always ascertain 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 alterations in the character and position of the cardiac impulse are often visible, but as their detection must be confirmed by means of palpation, they will be considered under the head of palpation (see p. 137).

2. Other Pulsations visible on the Chest Wall.—Systolic pulsation of the inner end of the *second left interspace* close to the sternum is sometimes communicated to the surface from the subjacent pulmonary artery in cases of retraction of the anterior border of the left lung.

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. Pulsation may often be seen in the *third and fourth left interspaces* as well as in the fifth. That impulse is usually due to the right ventricle, but is better studied by means of palpation (see p. 136).

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 aortic 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 may also give rise to visible pulsation in the walls of the thorax.

Systolic indrawing of the thoracic wall occurs under various conditions. In healthy persons (particularly children), in whom the chest walls are unusually thin, systolic indrawing is sometimes observed in the third and fourth left interspaces, and is simply the result of that recession of the base of the heart which is synchronous with the forward movement of the apical portion during systole. 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.

Systolic recession of the third and fourth left interspaces close to the sternum, due to the emptying of the right ventricle during systole, may also be observed in some cases of dilated heart. Such recession of interspaces must not be confounded with visible *retraction of the ribs and interspaces* in the præcordia, which is one of the more reliable signs of chronic adhesive mediastino-pericarditis. In that disease we may also observe systolic retraction of the lower end of the sternum, and of the tenth and eleventh interspaces below the angle of the left scapula.

Diastolic indrawing of interspaces and ribs at or in the vicinity of the apex-beat, is pathognomonic of adhesive mediastino-pericarditis. Mere adhesion of the pericardial surfaces to each other without mediastinal adhesions does not necessarily give rise to either systolic or diastolic recession of the thoracic wall.

3. Pulsations at the Root of the Neck.—Pulsation in the *episternal notch*, when well marked, usually points to simple or aneurismal dilatation of the aorta.

Pulsation *external to the sternomastoid* may be arterial, venous, or both arterial and venous in origin. It is important to distinguish the venous from the arterial pulsation. If there be well-marked pulsation in the neck, but a feeble pulse in the radial arteries, the pulsation in the neck is almost certainly venous.

Venous Pulsation at the Root of the Neck is frequently observed only when the patient is recumbent, and tends to diminish, or

even disappear, when he sits up. Venous pulsation is more readily seen than felt; arterial pulsation is as readily detected by palpation as by inspection. Arterial pulsation is as nearly as possible systolic in time; when venous pulsation is systolic (ventricular venous pulse) the jugular veins are greatly distended; in the variety of venous pulsation more frequently seen (auricular venous pulse), the collapse of the vein coincides with ventricular systole.

The venous pulse, however, can be satisfactorily studied only by means of recording apparatus, such as Mackenzie's polygraph (see pp. 179, 187).

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.

Engorgement and pulsation of the jugular veins are found in cases in which there is some obstruction to the flow of blood from the veins into the heart, whether the obstacle be primarily 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 attended with pulsation, which is considered more fully on p. 187. It will suffice here to mention that systolic venous pulsation in the neck is one of the most important signs of tricuspid incompetence.

Sudden collapse of the distended jugular veins during ventricular diastole—Friedreich's sign of pericardial adhesion—does not occur in all such cases, nor is it a conclusive sign of that disease.

4. Epigastric Pulsation may be conveniently divided into two groups—(*a*) that which is synchronous with the ventricular systole, and (*b*) 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 down-

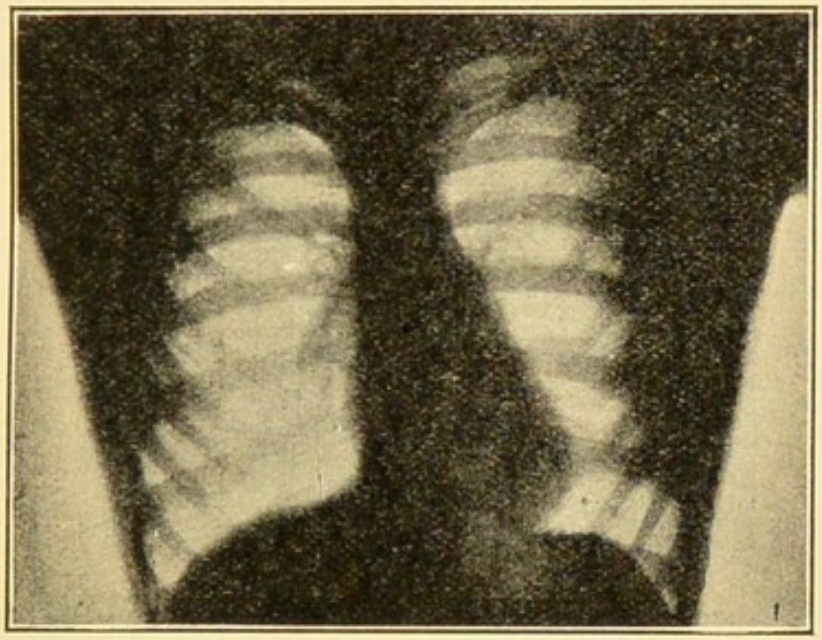


FIG. 53.– Skiagram of normal thorax (after Weintraud).

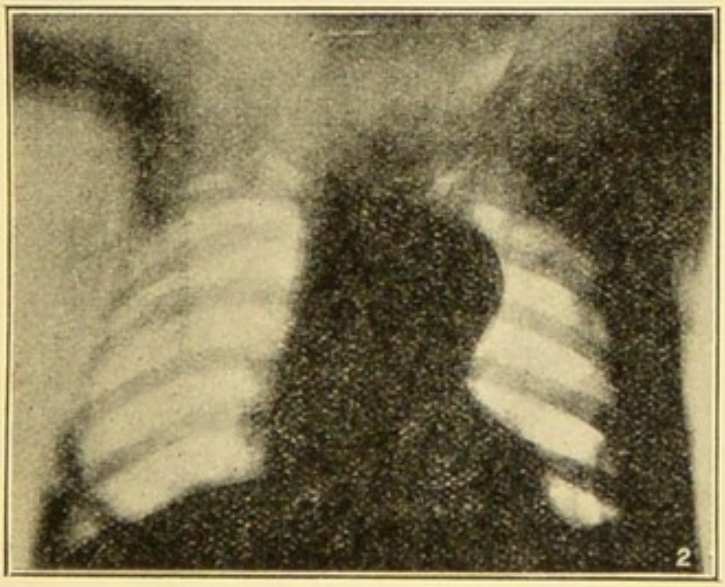


FIG. 54.—Aneurism of descending portion of the aortic arch (after Weintraud).

wards 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

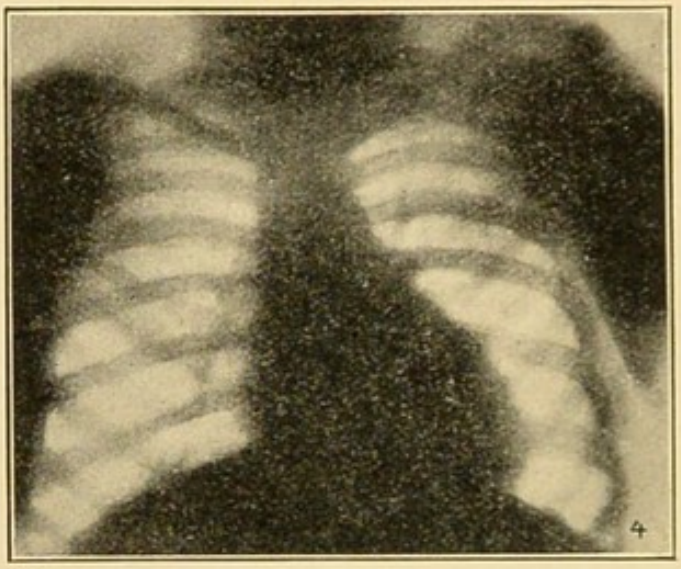


FIG. 55.—Aortic incompetence, with hypertrophy and dilatation of the left ventricle (after Weintraud).

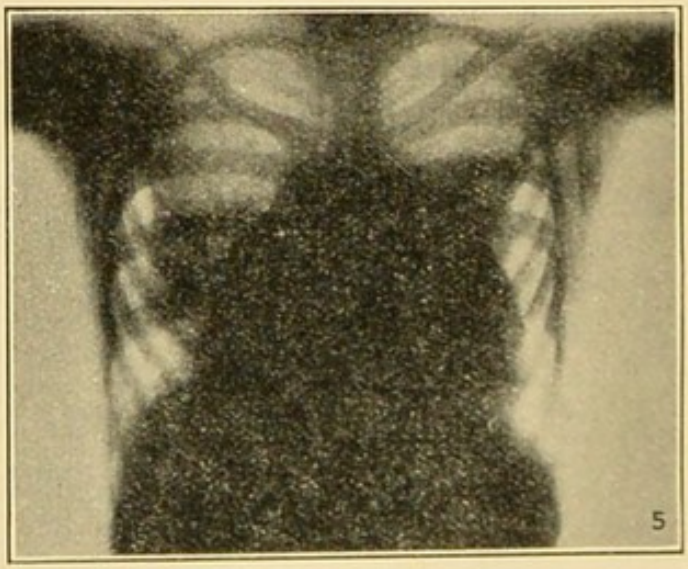


FIG. 56.—Mitral and tricuspid incompetence; marked dilatation of both ventricles (after Weintraud).

liver. If the hepatic veins be likewise affected, the pulsation in the liver becomes not merely heaving but distensile.[1]

Although in all these latter conditions the pulsation follows the apex-beat after a slight interval of time, it is usually somewhat

[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.

difficult to determine, by inspection and palpation, the exact nature of the pulsation. It may be much more accurately studied by means of recording apparatus (see p. 181).

The Examination of the Patient by means of the Röntgen Rays is sometimes of service in the diagnosis of aortic aneurism and of enlargement of the heart. (See Figs. 53, 54, 55, and 56.)

CHAPTER VIII

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.

Method of Palpation.—The examination of the patient's chest, which has been bared, should be made while he lies on his back, though it may also be performed while he sits or stands upright. The physician, being at the right side of the patient, lays the palmar aspect of the whole hand, which has been thoroughly warmed, across the præcordial region, so that the points of the fingers lie in the left anterior axillary line. If the left mamma of a female patient be so large as to interfere with palpation of the præcordia, it must be held upwards and inwards. The whole **cardiac impulse** is then felt, and its position, strength, rate, rhythm, and extent carefully noted. The examiner should then palpate with the point of one finger to find the lowest and most external point of the cardiac impulse, in other words, the *apex-beat*. If the cardiac impulse cannot be readily felt, it frequently becomes more obvious when the patient leans forwards.

In this manner we confirm and amplify evidence obtained by inspection, remembering that in health the cardiac impulse lies between the fifth and sixth ribs, $\frac{1}{2}$ inch internal to the left mammary line; 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 the impulse is slightly displaced outwards, and that when he lies on the right side the impulse may no longer be palpable.

Alterations in the Position of the Cardiac Impulse.—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 collections of liquid or of gas in one or both pleural cavities, an increase of the size 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 fibrosis of one lung, or to pulmonary contraction following the absorption of a pleural effusion, or by reason of increased abdominal pressure, the result of tumours, ascites, tympanites, etc., the apex-beat will be correspondingly elevated.

2. LATERALLY.—(*a*) *To the right.*—In cases of hypertrophy and dilatation of the right ventricle, the cardiac impulse is due to that cardiac chamber. The impulse may be felt as far inwards as the left lateral sternal line. In some cases the pulsation is greatest in the epigastrium, whilst in more marked cases the main impulse may be felt under the right edge of the sternum, or even further to the right. The condition just described must not be confounded with that in which the heart is pushed over towards the right side by effusion into the left pleural cavity or by tumour of the left lung. On the absorption of the effusion, 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 fibrosis, 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 displace it considerably to the left. In fibrosis 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, when they are of considerable volume, also displace the apex-beat to the left.

The Strength of the Cardiac Impulse varies much even in healthy individuals, owing to the variations in the thickness of

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

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 cardiac innervation, acute and chronic myocarditis, myocardial degenerations, fatty change of the myocardium, and lastly deficiency of proper blood supply.

2. *Extrinsic Causes.*—When the visceral and parietal layers of the pericardium become adherent, all evidence of the cardiac impulse may be lost. Accumulation of fluid or gas in the pericardium or in the left pleural cavity, as well as intervention of emphysematous lung tissue between the heart and the thoracic wall, all tend to weaken the cardiac impulse.

Increase in Force of the cardiac impulse may be due to any cause which increases the force of ventricular systole. It may be of neurotic origin; it may arise from violent exertion or from strong emotions; it may be a sign of fever. 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 is, moreover, never so great as that of the left ventricle.

The Rate and Rhythm of the cardiac impulse should be noted on palpating the præcordia. Abnormalities of rate and rhythm of the heart will be considered when dealing with the pulse (see p. 172).

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, etc.), 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.

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 by the time of the thrill in relation to the various phenomena of cardiac action.

Thrills in the mitral area (a circle with a radius of 1 inch round the apex-beat) are systolic or presystolic, 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. Presystolic 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 area, denoting stenosis of the pulmonary orifice, or a diastolic, indicative of regurgitation through that orifice.

Thrills, both systolic and diastolic, may be felt over the præcordia in congenital malformation of the heart; and systolic thrills may sometimes be felt over the manubrium sterni or to the right or left of the sternum in cases of aortic aneurism.

2. Pericardial Friction Fremitus caused by the rubbing during the heart's action of the two pericardial surfaces, which have been rendered rough and uneven by the deposition of fibrin, is more readily detected by auscultation than by palpation, and so too is the friction of pleural and of pleuro-pericardial origin.

Pulsations Palpable outside the Præcordia.—The pulsations which may be felt outside the præcordia have been already to some extent considered under the head of Inspection (see p. 130). The pulsation of an aortic aneurism is frequently best felt when bimanual palpation is employed, the palm of one hand being laid on the anterior, and that of the other hand on the posterior aspect of the upper part of the chest.

The information obtainable by palpating the epigastrium and pulsatile vessels at the root of the neck has been already considered on pages 131 and 132.

Tracheal Tugging (Oliver's sign).—When an aortic aneurism is adherent to the trachea, or presses against the left bronchus, the trachea may be pushed downwards each time the aorta is

distended with blood. To elicit tracheal tugging, the patient closes the mouth and elevates the chin, and the physician, standing behind the patient and pressing the two forefingers beneath the cricoid cartilage, pulls it upwards. The trachea may then be felt to be tugged downwards with each systole of the heart.

CHAPTER IX

Circulatory System (*continued*)

PERCUSSION

By percussion of the heart, evidence is obtained regarding:—(1) The Size and Position of the Heart; (2) The Extent to which it is Overlapped by the Lungs; (3) The Presence of Pericardial Effusion, and of (4) Aortic Aneurism.

The greater portion of the heart is separated from the chest wall by the overlapping lung, and therefore it is only over a small area corresponding to the uncovered part, which consists of right ventricle, that an absolutely dull note is obtained on percussion. That is the area of *superficial* or *absolute* cardiac dulness. The larger area, which represents the actual outline of the heart, is termed the area of *deep* or *relative* dulness.

As the indications regarding the size of the heart, obtained by examination of the area of absolute cardiac dulness, may be vitiated by various pulmonary conditions, we must seek to obtain the desired information mainly by percussing out the area of deep cardiac dulness.

Method of Percussing the Heart.—The patient should lie recumbent on his back. Commence by defining the area of **Deep Cardiac Dulness.** Using moderately strong percussion, we percuss towards the cardiac area. The note which is at first purely pulmonary in character grows gradually 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 (see p. 255).

It is by noting the point at which the first change in the note occurs that the position of the outer margin of the heart can be determined by percussion. In addition to the note elicited, the sense of resistance perceived during percussion is of great value.

Now, although it is in most cases not difficult to percuss out the entire margin of the 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.

(1) Percuss Vertically Downwards in the Left Parasternal Line, in order to avoid the dulness due to the aorta and pulmonary artery. Percussing downwards from the lower edge of the clavicle, and comparing rib with rib, and interspace with interspace, the

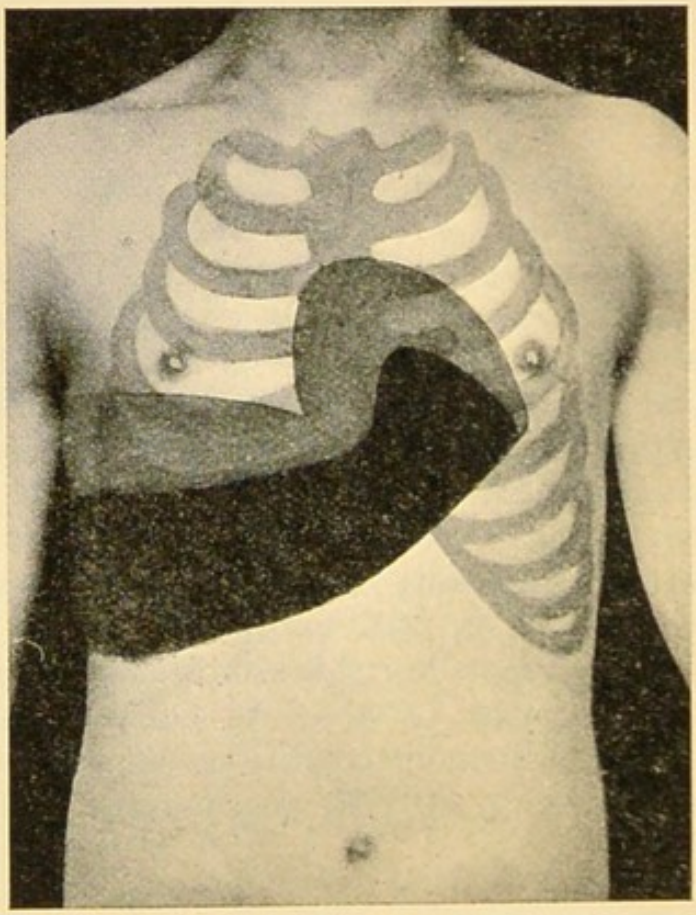


Fig. 57 —Deep and superficial dulness of normal heart and liver.

full note of the lung becomes impaired at about the upper edge of the third rib. This is, then, the limit of relative or deep cardiac dulness in this situation (Fig. 57).

(2) Percuss Transversely at the Level of the Fourth Rib.— To define the *left border* of the heart, commence in the left axilla, and keeping the finger, on which percussion is made, parallel with the left border of the heart, percuss from left to right along the fourth rib. The left border is normally found about $\frac{1}{4}$ inch external to the apex-beat, or from $3\frac{1}{2}$ to $3\frac{3}{4}$ inches from the mid-sternal line, at the level of the fourth and fifth interspaces, and 3 inches from the mid-sternal line at the level of the fourth rib.

To define the *right border* of the heart, commence in the right anterior axillary line, and percuss from right to left along the fourth rib. The right margin of deep dulness at that level is normally found in the right lateral sternal line, or 1 inch to the right of the mid-sternal line (Fig. 57).

The transverse extent of the deep cardiac dulness, at the level of the fourth interspace, is thus normally $4\frac{1}{2}$ inches. A convenient method of recording the limits of the deep cardiac dulness is that of Graham Steell. The normal limits would be recorded thus: $\frac{\text{III}}{1\text{–}3\frac{1}{2}}$. The Roman numeral III indicates the rib, to the upper edge of which the deep cardiac dulness extends in the left parasternal line. Beneath the horizontal line, the Arabic numerals record the distances, expressed in inches, of the right and left borders of the heart from the mid-sternal line.

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

The Superficial Cardiac Dulness.—Our aim being to map out that portion of the heart which is uncovered by lung, the anterior margins of the lungs are to be defined as far as is possible. Percussion is therefore made from the heart towards the lungs, in accordance with the rule that when seeking to define the margin of an organ we always percuss towards it. And the pulmonary margin being thin, it will not be detected except by very light percussion. Commence by percussing lightly over the fifth costal cartilage, midway between the left lateral sternal and left parasternal lines. Thence percuss upwards, to the right, and to the left, noting each point at which the absolutely dull note is first replaced by one slightly resonant.

The *right border* extends along the left lateral sternal line, from the level of the fourth to that of the sixth costal cartilage. This border does not lie in the same line as the anterior border of the right lung, namely, in the mid-sternal line, because on percussion over the sternum a resonant note is obtained, chiefly owing to the fact that the air in both lungs is thereby set in motion (see p. 262).

The *left border* is indicated by a line stretching from the fourth left chondro-sternal junction to a point $\frac{1}{2}$ inch within the apex. The *lower border* 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 to the sixth left chondro-sternal junction. The lower border usually measures $2\frac{1}{2}$ or 3 inches.

The area thus formed is, roughly, triangular in shape. Its

extent and position vary not only in accordance with the changes in the volume of the left lung on each respiration, but also with the posture of the patient. The variations to which the area of superficial cardiac dulness are liable, render it of comparatively little value as an index of the size of the heart.

Pathological Alterations of the Area of Cardiac Dulness

1. **Increase of the Area of Deep Cardiac Dulness.**—Increase to the **left** (Fig. 58), with corresponding displacement of the apex-beat,

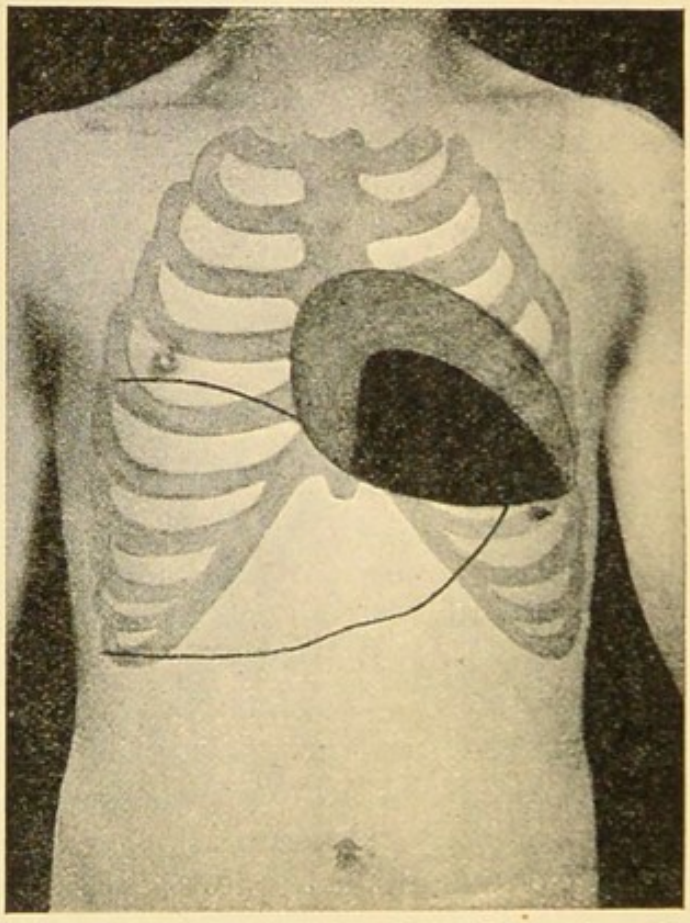


Fig. 58.—Superficial and deep cardiac dulness in enlargement of the left ventricle.

and provided that the whole heart be not displaced towards the left, indicates enlargement of the left, right, or both ventricles of the heart. Increase of dulness to the left, extending considerably beyond the apex-beat, is due to pericardial effusion (Fig. 60), provided that the dulness be not caused by pulmonary consolidation or pleural disease on the left side.

Increase to the **right** (Fig. 59), so that there is definite dulness to the right of the sternum, is due to enlargement of the right auricle, provided that the dulness be not the result of disease of the right lung or pleura, or of displacement of the whole heart.

Increase **upwards**, above the upper border of the third left rib (if not due to consolidation of the lung, aortic aneurism, nor upward displacement of the whole heart, as in ascites) arises from pericardial effusion.

Increase both to the **right** and **left** is observed when the right auricle and both ventricles are enlarged, as in some cases of mitral and tricuspid incompetence, and in pericarditis with effusion. In the latter condition the area of deep dulness is of pyramidal or pyriform shape, with its base downwards resting on the diaphragm

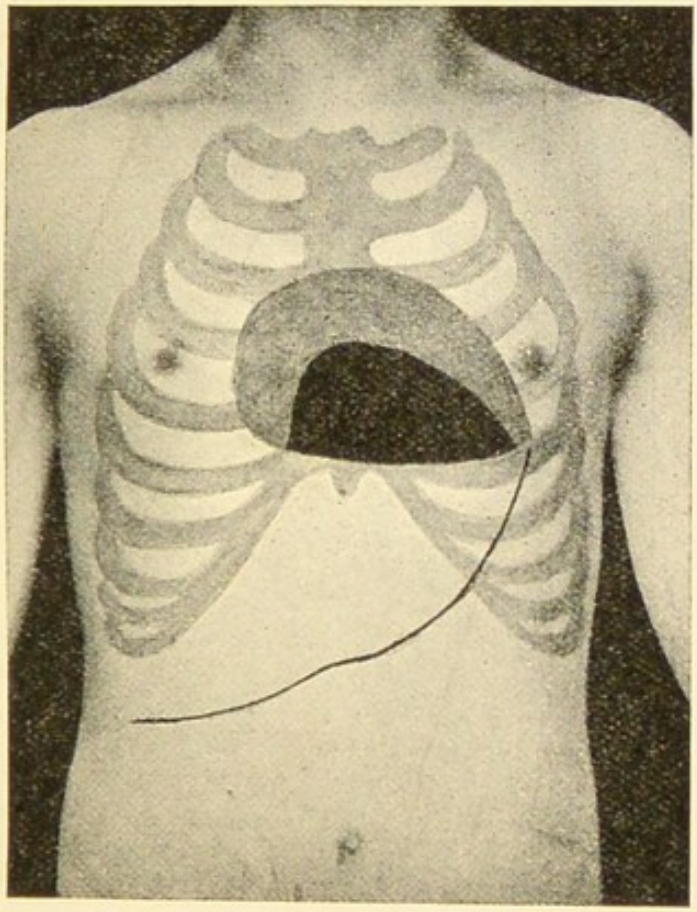


FIG. 59.—Superficial and deep cardiac dulness in enlargement of the right auricle.

(Fig. 60); the left border extends to the left of the apex-beat; there is dulness at the inner end of the fifth right interspace (*Rotch's sign*), and the right border forms a right or even obtuse angle, instead of the normal acute angle, with the upper margin of the deep hepatic dulness.

2. **Displacement of the Area of Deep Cardiac Dulness** without increase of its extent may occur (*a*) to the *right*, as in dextrocardia, or when the heart is displaced by a massive effusion into the left pleural cavity (Fig. 61), by left-sided pneumothorax, or by

contraction of the right lung. (*b*) Displacement to the *left* may be due to pleural effusion or pneumothorax on the right side, or to contraction of the left lung. (*c*) Displacement *upwards* is due to ascites, large abdominal tumour or other condition associated with increase of pressure within the abdomen. (*d*) Displacement *downwards* is usually the result of pulmonary emphysema.

3. **Increase of the Area of Superficial Cardiac Dulness,** associated with increase of the deep dulness (Figs. 58 and 59), is

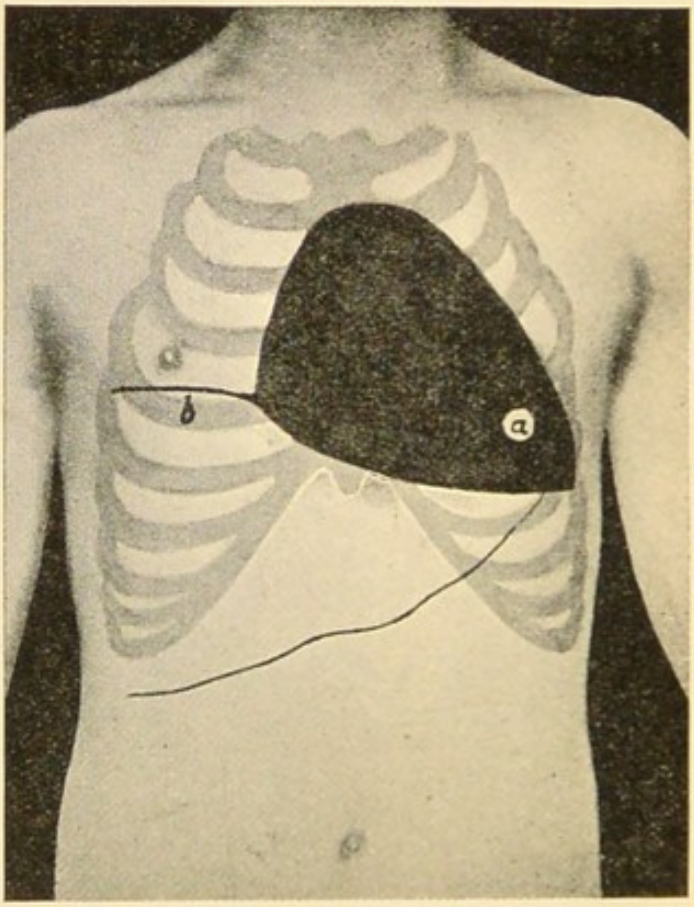


FIG. 60.—Area of cardiac dulness in case of pericarditis with effusion. *a*, the apex-beat; *b*, the upper margin of the liver.

observed in enlargements of the left and right ventricles and of the right auricle, and in pericardial effusion. When the area of superficial dulness is increased, but not that of the deep dulness, the usual cause is retraction of the borders of the lungs, resulting from fibroid contraction of the lung or pleuritic adhesions. In such cases a larger area of the heart is exposed, and hence there is an increase of dulness.

Increase of superficial cardiac dulness may be simulated by various pathological conditions of the neighbouring organs, such as infiltration of the margins of the lungs, pleuritic effusions, etc.

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 superficial cardiac dulness.

4. **The Area of Superficial (Absolute) Dulness is diminished,** or entirely lost, in—

(1) *Left pneumothorax*, where the collection of gas in the left pleural cavity is so great as to force the heart to the right. The

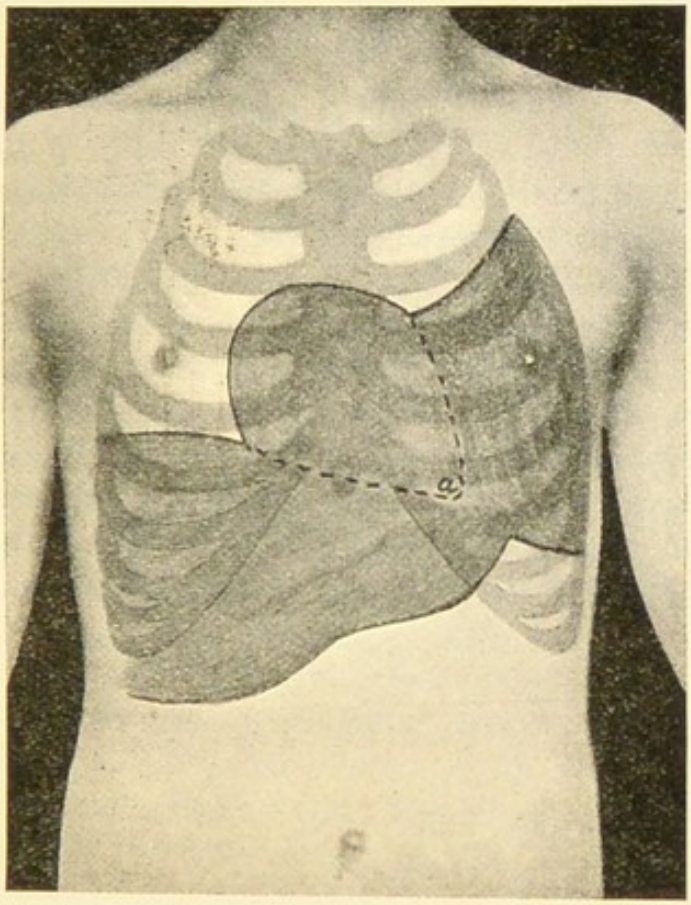


FIG. 61.—Displacement of the heart to the right by effusion in the left pleural cavity. *a*, the apex-beat. The liver is enlarged and displaced downwards.

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 the 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 those 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 peri-

cardium (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.

The Aortic Dulness exists in the normal condition simply as a slight rounded projection from the upper part of the deep cardiac dulness over the sternum. 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 sternum 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 of the vibration of the sternum which the firm pressure of the tumour occasions. 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 X

Circulatory System (*continued*)

AUSCULTATION OF THE HEART

Cardiac auscultation is almost invariably practised with the aid of a stethoscope. The best stethoscope is a simple wooden one; the ear-piece should be very slightly concave, while the chest-piece should not be more than 1 inch in diameter. Neither the binaural stethoscope nor the phonendoscope are so good as the simple wooden instrument; yet a binaural stethoscope may be employed with advantage when examining infants.

When we are auscultating, it is essential that the whole circumference of the chest-piece be in contact with the chest wall, and that the stethoscope do not press heavily on the patient.

On listening over the præcordia, two sounds, which may be represented phonetically by *lubb—dup*, 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 the cardiac orifices occupy in relation to the anterior thoracic wall (see Fig. 62).

The **Pulmonary Orifice** is situated horizontally behind the upper border of the third left costal cartilage and the corresponding half of the sternum.

The **Aortic Orifice** lies almost horizontally (but slightly lower and further to the right than the pulmonary) and corresponds to a line joining the middle of the sternum and the inner end of the lower border of the third left costal cartilage.

The **Mitral Orifice**, which is situated on a plane considerably posterior to those in which the other orifices lie, corresponds to an oblique line behind the left half of the sternum, at the level of the fourth rib.

The **Tricuspid Orifice** lies still more obliquely behind the middle of the sternum from the level of the fourth to that of the fifth costal cartilage. The relation of the tricuspid orifice to the chest wall is also indicated by the middle two-fourths of a line, which, representing the auriculo-ventricular groove,

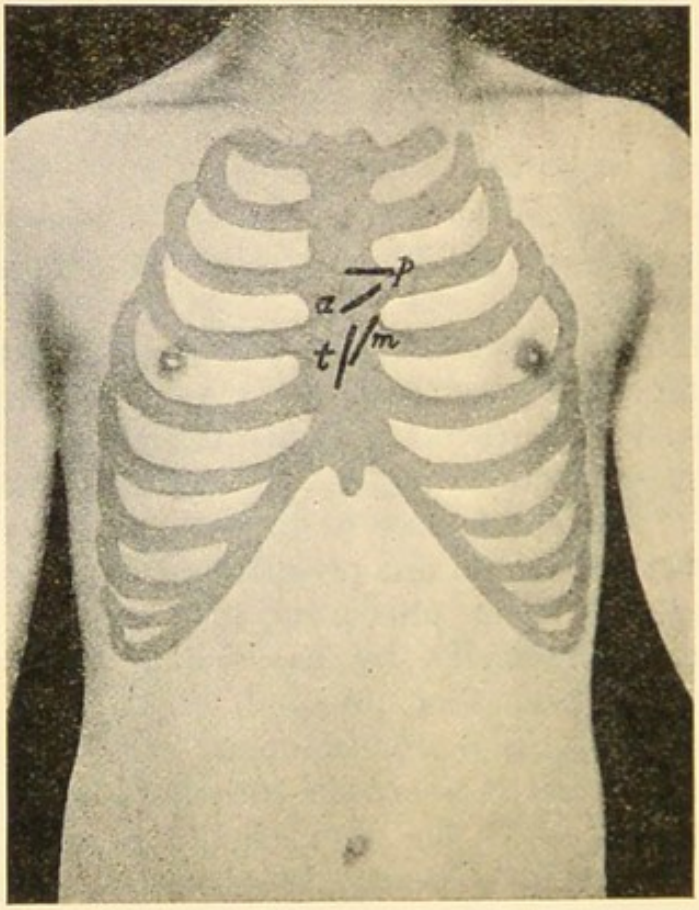


FIG. 62.—Position of the cardiac orifices in relation to the thoracic wall.

passes from a point in the mid-sternal line, at the level of the lower border of the third costal cartilage, to the sixth right chondro-sternal junction.

It will thus be seen that, in relation to the chest wall, the cardiac orifices and their valves lie very close to one another. The sounds produced in connection with each valve are, however, best heard, not immediately over the valve, but at that point on the chest wall at which the cavity, into which blood is flowing through that valvular orifice, approaches nearest to the surface. Naturally the point where the sound is most intense varies in the

case of each valve, and hence we have four areas for auscultation (Fig. 63).

The **Mitral Area** is a circle about 1 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-

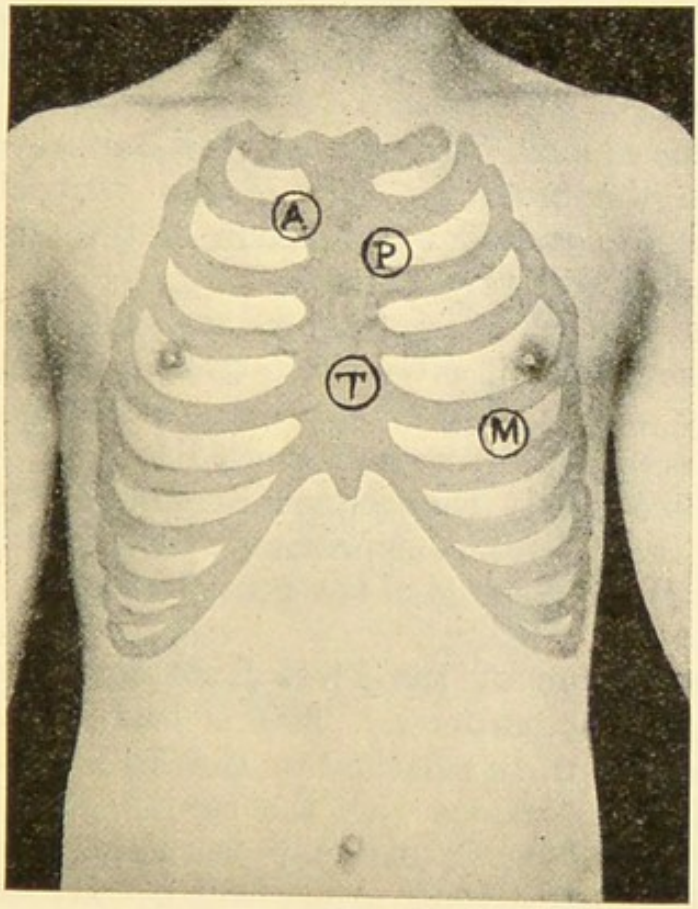


FIG. 63.—Auscultatory areas.

wall at the second right costal cartilage, and consequently the aortic area is situated at this point.

The **Pulmonary Area** corresponds to the inner end of the second left intercostal space.

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.

The **second sound** (diastolic) has its origin in the vibrations produced in the closed aortic and pulmonary valves, by the blood being forced back upon those valves during the first part of ventricular diastole.

It is evident that we have thus to deal with four sounds during the course of the cardiac cycle, 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 first or systolic and a second or 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 Differentiation of the First from the Second Sound is therefore of great importance. This is best effected by timing the two sounds in their relation to the cardiac impulse. The sound which is synchronous with the cardiac impulse is the first (systolic) sound. If the cardiac impulse cannot be palpated, the pulse in the common carotid artery will usually serve as a fairly reliable standard, the sound apparently synchronous with the carotid pulse being the first sound. The pulse in the radial artery is appreciably later than the commencement of ventricular systole, and therefore can not serve as a standard of time whereby to differentiate the two cardiac sounds (see Fig. 64).

The differentiation, however, is also aided by noting the relation of the two sounds to the two pauses. The first sound is that which is followed by the short pause, the second sound being followed by the long pause. This method fails us when the two pauses are of equal duration (pendulum and fœtal rhythm). Lastly, in health, at the mitral and tricuspid areas the accent falls on the first sound (*lūbb—dŭp*), whereas at the base of the heart the second sound is the louder (*lŭbb—dūp*).

From what has been already said, it will be evident that the mitral element of the first sound is best heard when we auscultate at the mitral area, whereas the tricuspid element is best heard at the tricuspid area, and that the aortic and pulmonary elements of the second sound are best heard at the aortic and pulmonary areas respectively.

The changes which disease may produce in the cardiac sounds 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, etc. It will suffice to consider such

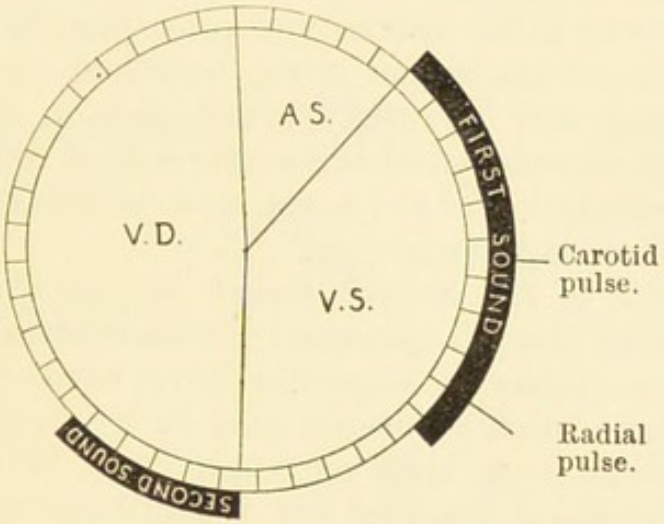


FIG. 64.—The cardiac cycle.

changes under the three following heads :—(1) Variations in Intensity ; (2) Reduplication and Alterations in Rhythm ; and (3) Impurity.

1. **Variations in Intensity.**—(*a*) THE SOUNDS MAY BE INTENSIFIED, *i.e.* ACCENTUATED.—This takes place with regard to all the heart sounds under the influence of mental or bodily excitement, pyrexia, etc., or may be the result of improved conduction, either because the chest walls have become thin, or from consolidation 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 pressure in the column of blood which presses back on the valves.

The first sound is accentuated with a dull, long, booming character in cases of hypertrophy of the left ventricle, as typically seen in arterio-sclerosis and 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 the second sound is accentuated in either of those areas, it is necessary to compare the sound in the two areas, always bearing in mind, however, that in normal adults the sound is louder in the aortic area than in the pulmonary.

Accentuation of the Second Sound in the Aortic Area arises from increase of the arterial blood pressure. This leads to more forcible vibrations of the aortic valves, and consequently to intensification of the sound, which is met with, therefore, in cases of general arterio-sclerosis and in nearly every case of chronic interstitial nephritis. Accentuation of the aortic second sound is also to be observed in dilatation of the aorta, and in aortic aneurism if the aortic valves are still competent.

Accentuation of the Second Sound in the Pulmonary Area is found whenever there is increased pressure within the pulmonary artery. 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 into 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, pericardial effusion, etc., or they may be audible with difficulty on account of loud sounds in the neighbouring lungs. Feebleness of the *first sound* is noticed 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.

Enfeeblement of the *second sound* in the aortic area is observed in mitral regurgitation, the explanation being that from the over-distended left auricle an excess of blood passes into the left ventricle, consequently in the early stage of ventricular diastole the pressure within the left ventricle is higher than normal and the pressure within the aorta, therefore, *relatively* low. In like manner the second sound in the pulmonary area is enfeebled in tricuspid incompetence.

2. **Reduplication and Alterations of Rhythm.—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 of the first sound may be represented phonetically by *tlubb—dup*, or if the two elements of the first sound are still more distinctly separate, as *talubb—dup*, with the accent on *lubb* or *dup*, according as we auscultate at the apex or base of the heart. Reduplication of the second sound is represented phonetically by *lubb—click*, or *lubb—tadup*, the accent being on *dup* when we auscultate at the base.

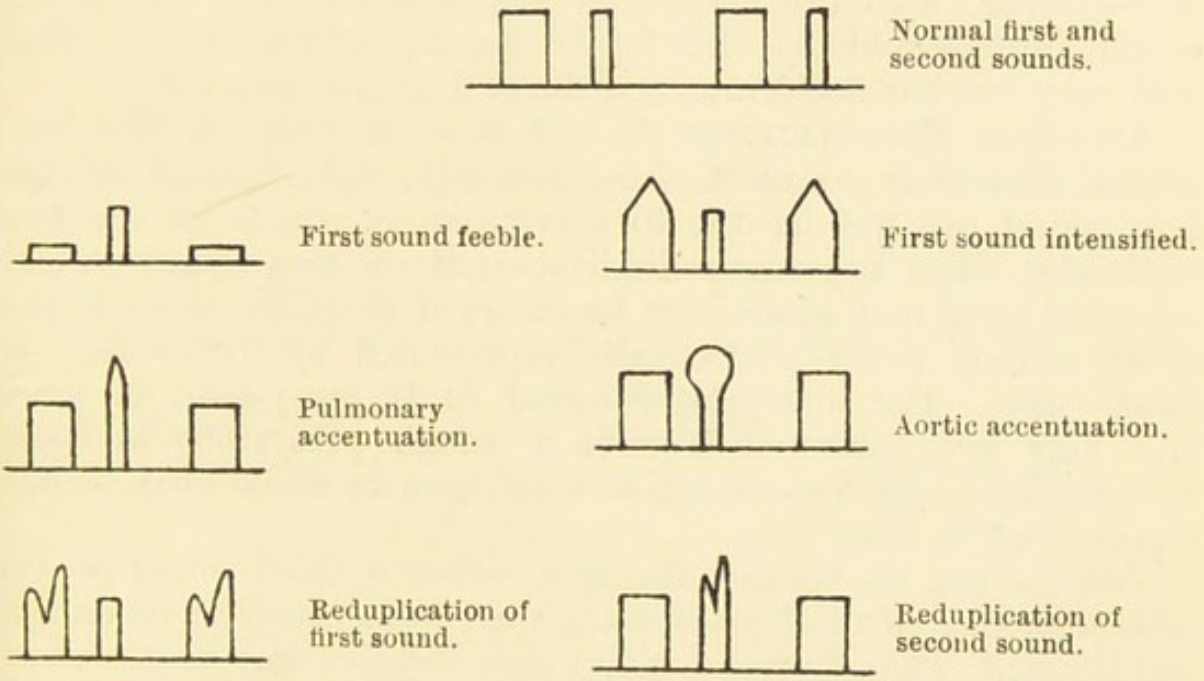


FIG. 65.—Diagram of modifications of heart sounds (after Wyllie).

REDUPLICATION OF THE SECOND SOUND is an indication of alteration of blood pressure either in the pulmonary or systemic circulation. In health the second sound may be reduplicated at the end of full inspiration and the beginning of expiration, but is then of no peculiar significance. In disease, when reduplication of the second sound occurs, it is more marked and constant. It is frequently found in cases of mitral constriction and lung disease. Reduplication of the second sound in the pulmonary area is usually explained by the theory which maintains that when there is abnormally high blood pressure in the pulmonary vessels, the closure of the pulmonary cusps is accelerated and the sound they emit is therefore in advance of that produced at the aortic cusps. As the truth of this theory is not above suspicion, some maintain that abnormally high pressure in the pulmonary vessels leads the

right ventricle to contract more slowly than the left, and that therefore the pulmonary element of the second sound follows that produced at the aortic valve.

According to the latter theory, when there is abnormally high blood pressure in the systemic arteries the aortic element of the second sound will follow the pulmonary element. Reduplication of the second sound is always best heard at the base of the heart.

In Fig. 65 these various modifications of the sounds of the heart are graphically represented.

Apparent Reduplication of the Second Sound at and near the Apex, but not at the base, may be heard in some cases of mitral stenosis. The heart sounds may then be expressed thus: *lubb—dupta*, with the accent on the first sound. The added sound, represented by the *ta* of *dupta*, is diastolic in time and may resolve itself into a definite diastolic murmur.

Apparent Reduplication of the First Sound.—As the ventricles invariably contract simultaneously, reduplication of the first sound can not be due to asynchronous systole of the two ventricles. But apparent reduplication of the first sound at and near the apex may sometimes be observed in mitral stenosis, the heart sounds being phonetically represented by *tlubb—dup*, or *talub—dup*. The interpolated sound *ta* is presystolic in time, and may give place to a definite rough presystolic murmur running up to the first sound, in which case we would hear sounds represented by *rrup—dup*.

The Canter or Gallop Rhythm.—Here a third sound is also interpolated. Of the three sounds, the accent usually falls on the middle one. The sound of a canter thus imitated may be expressed thus: *ta—lubb—dup*. The first of the three sounds is produced during diastole, and is ascribed to suddenly increased tension of the ventricular wall (Potain). It is a sign of increased intra-ventricular pressure.

Pendulum Rhythm.—The two pauses are of equal duration, owing to prolongation of the period of systole. This abnormality is usually associated with high arterial blood pressure, as in chronic nephritis.

In Fœtal Rhythm or Embryocardia, as may be observed in the terminal stages of heart disease, the two pauses are of equal duration (the diastolic phase being reduced), and the rate of the heart is greatly accelerated.

The Rhythm when Extra-systoles occur. — When an extra-systole occurs (giving rise to an "intermission" of the pulse) the two cardiac sounds are quickly followed by other two; then ensues a long compensatory pause (Fig. 66, 2). If the extra-systole

occur still earlier after the normal systole, the former may cause a first but no second sound (Fig. 66, 3), because the ventricles, at the moment when the extra-systole occurs, contain so little blood that ventricular systole does not suffice to open the semilunar valves. Examples of the altered rhythm of the cardiac sounds on the regular occurrence of extra-systoles are graphically represented in Fig. 66, 4 and 5.

3. **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,

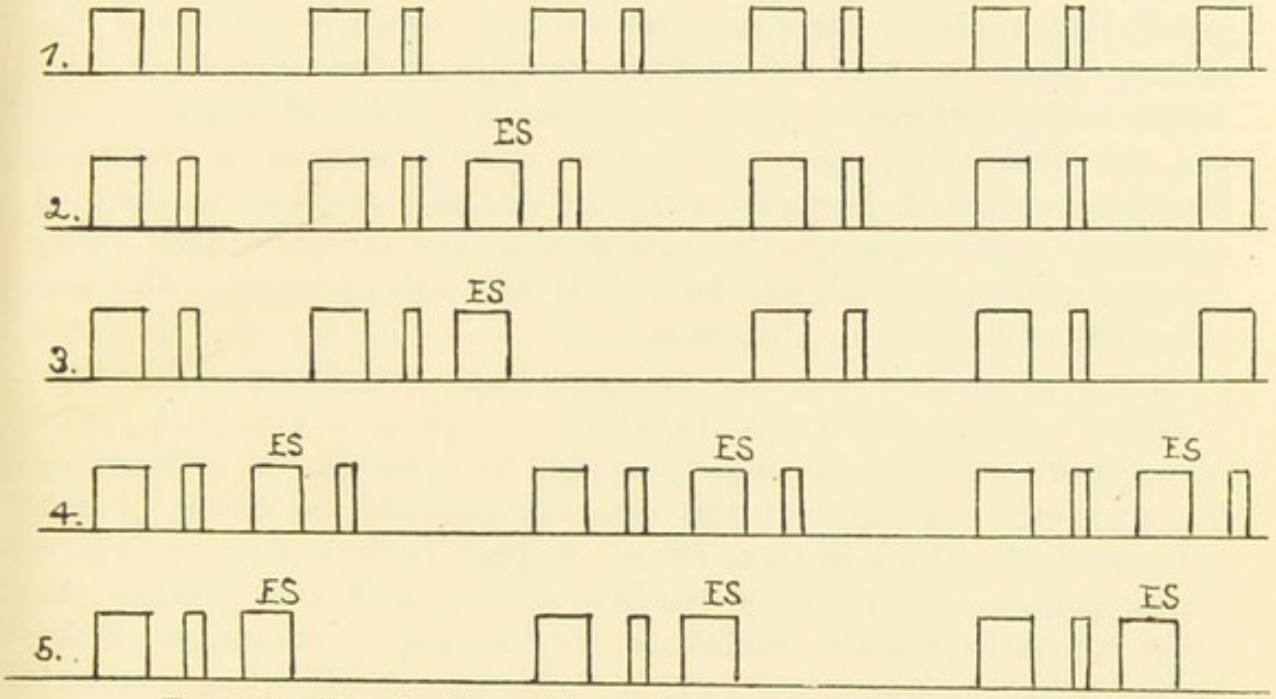


FIG. 66.—The rhythm of the cardiac sounds on the occurrence of extra-systoles.

but in practice an impure sound is not readily distinguished from one accompanied by a soft murmur. The impurity may give place to a definite murmur if the heart be stimulated to more vigorous contraction, as by directing the patient to walk across the room twice or thrice.

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

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 roughness 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 endocardial 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.

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 cycle 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 indicates 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 120 per minute, it may be difficult to time the murmur.

Murmurs which arise during the ventricular systole are termed *systolic*; those which occur during the ventricular diastole are known as *diastolic*. Diastolic murmurs may arise during the early, mid, or late phase of ventricular diastole. A late diastolic murmur occurs during the period of auricular systole, and as auricular systole precedes the ventricular, the late diastolic murmur is 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 valve 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 point of maximum intensity 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 frequently most intense at the lower part of the sternum, or at the fourth or fifth left interspaces, as far out, perhaps, as the apex.

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 murmur with its point of maximum intensity at the mitral area can only be one

of regurgitation through incompetence of the mitral 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 mitral systolic 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 systolic murmurs are heard over an area corresponding to the right ventricle, and are propagated to the right of the sternum. Aortic systolic murmurs are propagated up the manubrium sterni, and into the arteries of the neck. Aortic diastolic murmurs are propagated down the left edge of 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, etc.) 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 usually soft and blowing in character.

(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. It is not unusual to observe a murmur (for example, a mitral presystolic 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 an index of the cardiac vigour. This is not the case, however, as regards aortic regurgitation.

Having determined the rhythm and seat of the murmur, it is, as we 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.



Fig. 67.—Normal first and second sounds (after Wyllie).

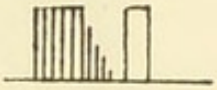


Fig. 68.—Mitral systolic murmur (after Wyllie).

Mitral Systolic Murmurs.—These murmurs, originating at the mitral valve during the ventricular systole, accompany or replace the first sound (see Fig. 68), are usually of blowing, less frequently of musical, character, have their point of maximum intensity at the apex of the heart, and are propagated towards the left axilla and angle of the left scapula (Fig. 69).

The murmur indicates that from some cause the valve does not completely close the orifice, but allows a part of the blood contained in the ventricle to be forced back into the left 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 rise of blood-pressure in the pulmonary vessels is that the rebound of the blood column upon the pulmonary valves after ventricular systole is rendered more forcible, and the second sound at the pulmonary area is consequently accentuated.

There are thus three chief physical signs to be looked for in cases of mitral incompetence—(1) the systolic murmur, (2) accentuation of the pulmonary second sound, and (3) displacement of the apex beat downwards and to the left, because of the dilatation and hypertrophy of the left ventricle. Evidence of enlargement of the right ventricle (see p. 132) should also be looked for.

Incompetence of the mitral valve is the result of one of two

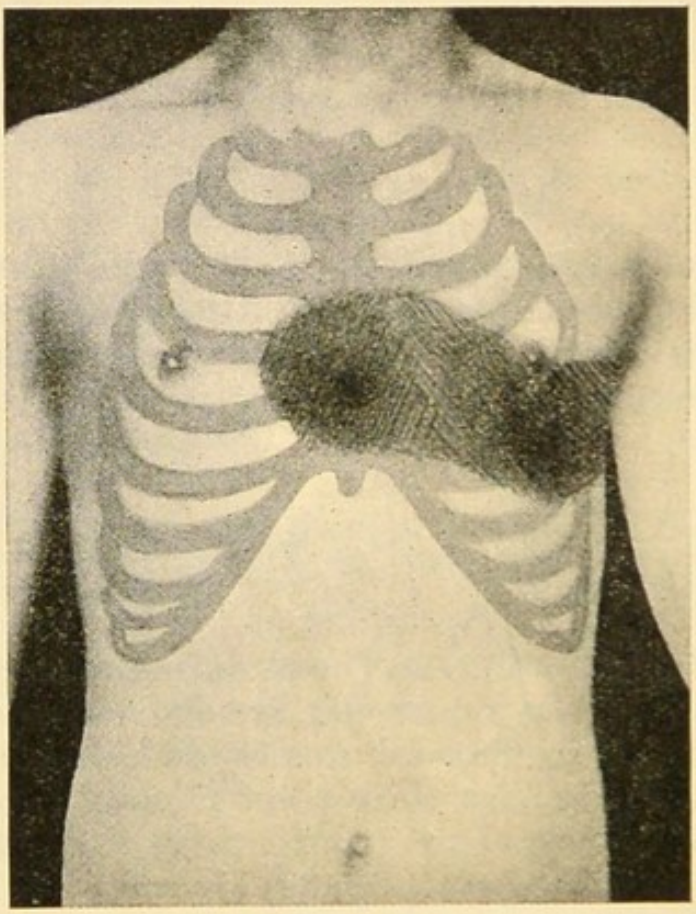


FIG. 69.—Mitral and tricuspid systolic murmurs.

The two shaded areas (which partly overlap) show the areas over which the murmurs are audible. The intensity of each murmur is indicated by the depth of the shading, and the point of maximum intensity by the black circle.

processes :—(1) the valve, with the chordæ tendineæ, has become thickened and shrunken so that it can no longer occlude the mitral orifice. Such structural alteration of the valve arises as a result of acute endocarditis in connection with rheumatism, scarlatina, etc., or of the chronic inflammatory and degenerative processes induced by physical strain and the toxæmias of syphilis, alcohol, etc. (2) The orifice has increased in size, or the papillary muscles lie so far apart, in consequence of dilatation of the left ventricle, that the valve, although structurally healthy, can no

longer close the orifice. This is the condition termed relative incompetence, and the murmur in those cases is termed "the bruit of disparity of size." It may be found in all diseases in which relaxation of the cardiac muscle takes place to a marked degree, as in typhus, typhoid, relapsing and rheumatic fevers; in scarlatina, measles, erysipelas, small-pox; and not least importantly, in chorea, exophthalmic goitre, and in various forms of anæmia, particularly in chlorosis.

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. In health the flow of blood into the ventricles takes place noiselessly; but when stenosis of the mitral orifice 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. The rapidity of flow is greatest at two periods of ventricular diastole, namely, at the commencement of diastole (when the suction of the ventricle is most powerful), and towards the end of ventricular diastole, when the rapidity of flow is accelerated by the occurrence of auricular systole. The murmur of mitral stenosis is therefore usually heard early in diastole (mitral diastolic murmur), or late in diastole (mitral presystolic, or auriculo-systolic murmur).

The mitral presystolic murmur usually has its point of maximum intensity a little above and within the apex. It is almost invariably rough in character, and as it increases in intensity up to the first sound, is often known as "the crescendo murmur of mitral stenosis." When this murmur is present the heart sounds may be represented phonetically by *rrup—dup*, and graphically as in Fig. 70. The mitral diastolic murmur is usually softer. It may be represented phonetically by *lubb—di-iff*, and graphically as in Fig. 71. The two murmurs often co-exist, as in Fig. 72, and may be separated from, or may run into, each other, so as to fill up the whole period of ventricular diastole.

At the apex the first sound has usually a peculiar thumping or slapping character, while in the pulmonary area there is accentuation and often reduplication of the second sound. The heart sounds, when there are presystolic and diastolic murmurs and reduplication of the second sound, would be represented

phonetically by *rrup—didi-iff*. There is usually a well-marked presystolic or diastolic thrill at the apex and evidence of enlargement of the right side of the heart. The cardiac rhythm is frequently irregular in the later stages of the disease. The disappearance of the presystolic murmur is an unfavourable sign, indicating that the dilated left auricle is probably contracting very feebly.

In every case of mitral stenosis we must search for evidence of

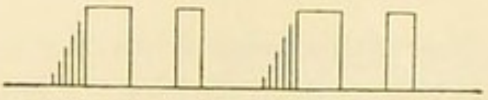


FIG. 70.—Mitral presystolic murmur.



FIG. 71.—Mitral diastolic murmur.



FIG. 72.—Mitral presystolic and diastolic murmurs.



FIG. 73.—Mitral presystolic and systolic murmurs (after Wyllie).

mitral incompetence, because regurgitation so frequently occurs through the stenosed orifice (Fig. 73).

The presystolic murmur audible at the mitral area in some cases of aortic incompetence, when the mitral valve is healthy, is considered on p. 166.

(*b*) TRICUSPID MURMURS

Tricuspid Systolic Murmurs replace or accompany the first sound, are of blowing character, have their point of maximum intensity at the lower end of the sternum, and are propagated to the right of the sternum (Fig. 69, p. 162). They indicate incompetence of the valve with consequent regurgitation of blood into the right auricle during ventricular systole. The incompetence may result from deformity of the valve produced by endocarditis. Relative incompetence (see p. 163), however, is much more frequent, being caused by fevers, anæmia, etc. (see p. 163), and still more frequently by dilatation of the right ventricle, the result of obstruction to the pulmonary circulation, produced most distinctly in cases of stenosis or incompetence of the mitral valve. The diagnosis of tricuspid regurgitation is confirmed by other signs, such as the ventricular venous pulse (see p. 189), general œdema (see p. 11), and enlargement of the right auricle (see p. 144).

Tricuspid Presystolic and Diastolic Murmurs are seldom met with, and rarely without other valvular complications.

They have their point of maximum intensity in the tricuspid area, are the result of stenosis of the tricuspid orifice, and the mechanism of their production is similar to that which produces the corresponding mitral murmurs.

(*c*) Aortic Murmurs

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

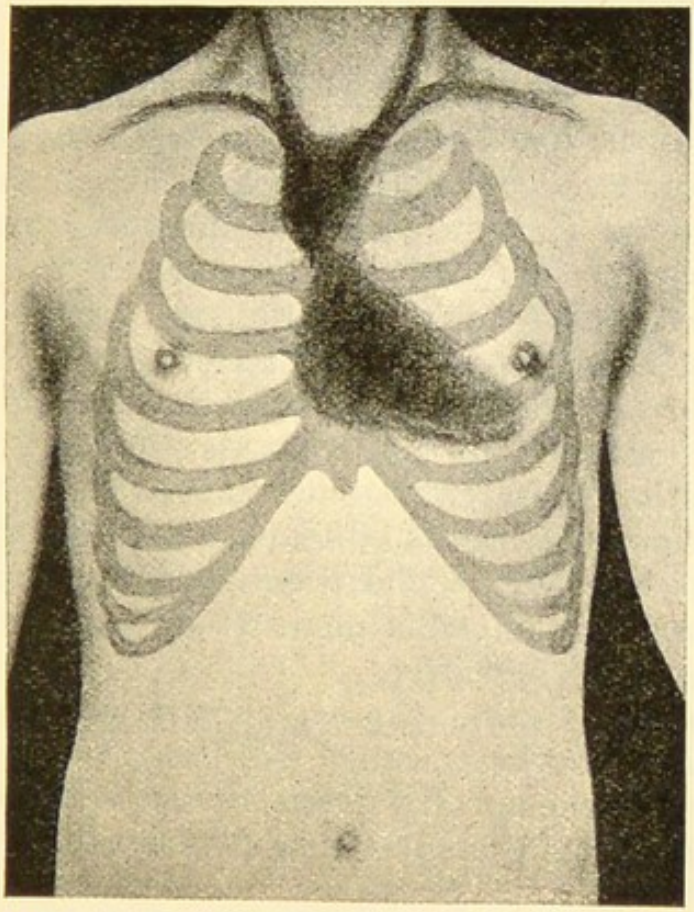


Fig. 74.—Aortic systolic and diastolic murmurs.

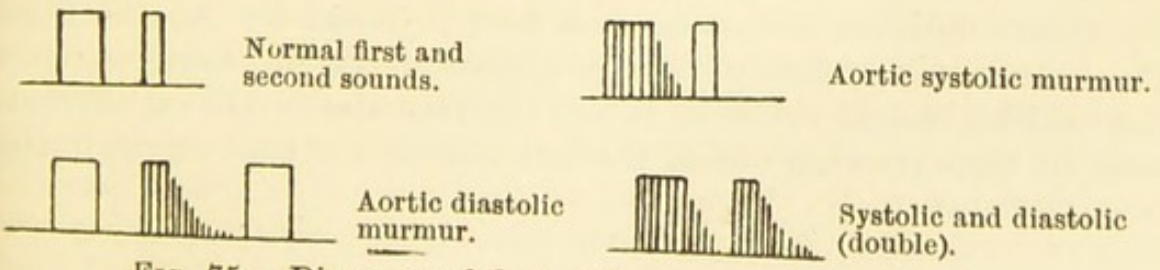


Fig. 75.—Diagrams of the aortic murmurs (after Wyllie).

Aortic Systolic Murmurs are usually loud and somewhat rough, occasionally musical, have their point of maximum intensity in the aortic area and are propagated up into the vessels of the

neck (Fig. 74). When the murmur is loud, it may be heard over the whole præcordia. The murmur arises when the aortic orifice is contracted, when the aortic cusps have become adherent to each other or roughened, and when there is atheroma or dilatation of the first part of the aortic arch. The murmur is therefore seldom due to actual stenosis of the aortic orifice. Confirmatory signs of aortic stenosis are a systolic thrill at and above the aortic area, marked hypertrophy of the left ventricle, owing to the extra resistance which the ventricle has to overcome in forcing the blood through the stenosed aortic orifice, and the small hard pulse which will be mentioned presently.

Aortic Diastolic Murmurs are the result of incompetence of the aortic valve, the blood regurgitating from the aorta into the left ventricle during ventricular diastole. The murmur is usually of blowing, but in some instances of musical, character. The point of maximum intensity of the murmur varies. In some cases it is best heard at the aortic area; more frequently it is loudest at the lower part, or a little to the left of the lower end of the sternum (Fig. 74); rarely the apex is the situation at which the murmur is most distinct, and it may then be confounded with a mitral diastolic bruit.

Aortic incompetence leads to dilatation and hypertrophy of the left ventricle (see p. 137). The peculiar pulse which is found in such cases (Corrigan's pulse) is of great importance in diagnosis, and will be described hereafter.

The two aortic murmurs, the systolic and the diastolic, usually co-exist, constituting the so-called *double aortic*, see-saw, to and fro murmur. For the aortic valve is rarely incompetent without there being conditions present which give rise to an aortic systolic murmur.

Flint's Murmur.—In certain instances aortic regurgitation also gives rise to a presystolic murmur audible at the mitral area. To this condition attention was first directed by Austin Flint. The murmur is probably due to vibrations of the aortic cusp of the healthy mitral valve, as it lies between the two blood currents, namely, that pouring out of the left auricle and that regurgitating from the aorta.

(*d*) Pulmonary Murmurs

Murmurs due to structural alterations of the pulmonary valve are of very rare occurrence. They are systolic and diastolic in rhythm.

Pulmonary Systolic Murmurs have their point of maximum intensity at the pulmonary area. They 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.

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

(*e*) Multiple Murmurs

In many instances, two or more endocardial murmurs co-exist. If the murmurs occur at different periods of the cardiac cycle, they are readily differentiated. If we find a systolic murmur having two points of maximum intensity, as, for example, at the mitral and tricuspid areas, we are obviously dealing not with one but with two murmurs. Any difference in the character of the murmurs is also of aid in their differentiation, as when rough aortic systolic and blowing mitral systolic murmurs co-exist. The propagation of the murmurs, and, lastly, all evidence regarding the size of the different chambers of the heart, the characters of the pulse and the patient's condition as regards œdema, cyanosis, etc., may be of assistance in diagnosis.

(*f*) Murmurs of Congenital Origin

The murmur due to *stenosis of the pulmonary artery*, the most frequent of the congenital malformations of the heart, has been already considered. The lesion is frequently associated with other malformations, such as: (1) *Patency of the ductus arteriosus*, which usually causes a loud systolic murmur having its point of maximum intensity in the second left intercostal space a short distance from the left lateral sternal line. The murmur is often accompanied by a systolic thrill. (2) *Patency of the foramen ovale*, with a murmur, presystolic or systolic in time, and best heard at the level of the third or fourth costal cartilages. (3) *Defect of the interventricular septum*, which produces a systolic bruit loudest over the upper half of the præcordia. In other cases there may be no audible murmur.

The differential diagnosis of a congenital from an acquired malformation is often difficult. It should be remembered that (1) diastolic bruits are rarely of congenital origin; (2) murmurs

heard loudly over the base but inaudible at the apex point to congenital rather than to acquired disease; (3) when the heart is of normal size, loud murmurs are almost exclusively of congenital origin; and (4) in children, murmurs which are associated with enlargement of the heart to the right and a feeble cardiac impulse are probably of congenital origin.

(*g*) Hæmic Murmurs

In many cases of anæmia, and especially of chlorosis, murmurs, which are invariably systolic and usually of soft blowing character, may be audible. In their order of relative frequency and intensity the murmurs are heard at the pulmonary, mitral, tricuspid and aortic areas. The mitral and tricuspid murmurs are due to relative incompetence (see p. 163) of the mitral and tricuspid valves.

There has been much speculation as to the mode of production of the most frequent hæmic murmur, namely, that best heard in the pulmonary area. Naunyn's theory, which is not generally accepted, is that the murmur is due to mitral regurgitation, the sonorous vibrations being conducted through the left auricular appendix, and having their point of maximum intensity at that point on the chest wall where the appendix approaches nearest to the surface—*i.e.* about $1\frac{1}{2}$ inch to the left of the pulmonary area. A more satisfactory explanation is that the murmur is due to dilatation of the conus arteriosus and pulmonary artery. Other possible factors in the production of the murmur are the increased volume of the blood in chlorosis (as established by Lorrain Smith), the increased rapidity of the blood current, and the lowered viscosity of the anæmic blood whereby it passes more readily into eddies and sonorous vibrations.

The differentiation of hæmic murmurs from murmurs of valvular disease is based partly on the presence of anæmia, on the systolic murmur having its point of maximum intensity at the pulmonary area, and on the co-existence of a *bruit de diable* over the jugular veins (see p. 177). Of greater importance, however, is the absence of all other evidence of organic disease of the heart—for example, signs of cardiac enlargement or enfeeblement.

2. **Exocardial Murmurs.**—Pericardial 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. The former are usually of rough, grating or brushing character. Their point of maximum intensity

does not coincide with any of the cardiac areas, but lies over the base of the heart at the left side of the sternum, or over the right ventricle. Friction murmurs are localised to the præcordia and are not propagated in the direction of the blood currents. The rhythm of the friction murmur is variable; it is not confined to any particular phase of the cardiac cycle, but appears to overlap the first and second sounds. The murmur is usually most distinct at two periods of the cycle, ventricular systole and diastole, and has therefore a to-and-fro rhythm; or it may be composed of three elements, one of which is presystolic in time. The intensity of the murmur may be increased by firm pressure of the stethoscope, or when the patient leans forwards.

Pericardial friction may be audible although there is considerable effusion in the pericardial cavity.

Pericardial friction can hardly be mistaken for the friction of pleurisy, the latter being synchronous with respiration. The movements of the heart, however, sometimes occasion friction on each other of the two inflamed surfaces of the pleura overlying the heart. Such *pleuro-pericardial* friction murmurs are of greater intensity on full inspiration, whereas if the friction murmur of pericarditis vary with respiration, it is enfeebled on full inspiration.

CHAPTER XI

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 (see p. 131). Still more marked is the pulsation when the left ventricle is greatly 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 examination of the pulse in the radial artery.

The Pulse.—Although the arteries are constantly filled with blood, the pressure within them—*i.e.* the arterial blood pressure—is constantly varying in accordance with the systole and diastole of the left ventricle. The pulse is the wave of increased pressure within the arteries due to the ventricular systole.

When the pulse is examined, the patient's arm should be supported on a level with the heart. If he be recumbent, the

forearm should lie on the bed; if he be standing or sitting, the arm and hand are supported at the level of the fourth costal cartilage. In either case, the patient's forearm being pronated, the physician presses the first three fingers of one hand on the radial artery as it lies on the lower end of the radius.

The radial pulse is, in health, equal on the two sides; but abnormal distribution, compression, or other pathological condition 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 from stenosis of the aortic orifice rendering the systole slow and difficult.

When examining the pulse, the physician should study (1) THE CONDITION OF THE ARTERY, (2) THE FREQUENCY, (3) RHYTHM, and (4) CHARACTERS OF THE PULSE.

(1) **Condition of the Artery.**—By rolling the fingers lightly across the vessel, we attempt to estimate its *size*, and then proceed to determine the *state of its wall*. This can be best appreciated by pressing on the vessel with the finger sufficiently to empty it, and then rolling it under the finger on the bone below. The radial artery is, normally, so soft and elastic, that it cannot be felt by this method of examination. But when the arterial wall is the seat of arterio-sclerosis and has become thickened and less elastic, the vessel can be felt, and when those changes are pronounced the artery feels hard and rigid. Tortuosity, and local irregularities of the arterial wall, imparting to it a "beaded character," are best recognised by passing the fingers up and down over the artery in its long axis.

The student should remember that although the radial arteries are palpably thickened the aorta is not necessarily diseased, and that there may be marked atheroma of the aorta and coronary arteries when the radials are unaffected.

(2) **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 more frequent 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.

Bradycardia.—A pulse rate of less than fifty beats per minute, with a corresponding infrequency of the heart's contractions, constitutes the symptom of bradycardia (see Fig. 97, p. 194). This may be simulated when only every alternate ventricular systole gives rise to a palpable pulse wave. Bradycardia is due to stimulation of the vagus or to disease of the heart. The symptom may be observed in jaundice, and during the convalescence from acute infective diseases, as influenza; it may be due to other toxic agents (lead, digitalis, etc.), to nervous causes (injuries to the cervical region of the spinal cord, cerebral tumour, etc.), and to arterio-sclerosis and myocardial degenerations. In order to ascertain whether the auricles as well as the ventricles beat with diminished frequency, or whether the ventricular rate alone is diminished (in consequence of a lesion at the auriculo-ventricular bundle, resulting in heart-block), the pulsations of the jugular vein must also be studied (see p. 194).

Tachycardia.—More frequently the pulse rate is increased. The frequent pulse of fever, of collapse, and of various cardiac neuroses, as neurasthenia and paroxysmal tachycardia, is well known. Very generally the pulse is rapid in diseases of the valves of the heart, particularly when there is failure of compensation.

(3) **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 variety of ways. Disturbance of rhythm may be shown by complete irregularity (*arhythmia*), or by rhythmic irregularity (*allorhythmia*, see Fig. 91). When the pulse beats are irregular in time they are frequently unequal in volume. Irregularity of rhythm, though readily detected by digital examination, is more precisely studied by means of the sphygmograph, and is therefore considered on p. 187. One of the most frequent forms of irregularity, namely, that due to a ventricular extra-systole, can be readily recognised, however, when counting the pulse beats, by continuing to count rhythmically and finding that the long pause (the so-called intermission) is exactly equal in time to two pulse beats, as in Fig. 91.

(4) **The Characters of the Pulse.**—(*a*) The Volume of the Pulse.—Under the influence of the pulse-wave, the vessel, partially flattened by the pressure of the fingers, tends to

return to its cylindrical form. The extent of this movement, constituting the volume of the pulse, depends on several factors. It is increased in proportion to the diameter of the artery and the volume of blood propelled into the aorta at each systole. This in turn is usually proportionate to the strength of the ventricular systole. Increased tonus (hypertonus) of the arterial walls and high arterial blood pressure diminish the volume of the pulse, whereas, other factors being equal, the volume is increased when there is hypotonus and low blood pressure.

When the pulse waves are of unequal volume, we speak of an *unequal pulse.*

The volume and frequency of the pulse are normally greater during inspiration than expiration. In the *paradoxical pulse* (*pulsus paradoxus*) the volume and frequency of the pulse in the arteries becomes lessened, or the pulse may even be imperceptible, during inspiration (Fig. 81, p. 183). This peculiarity of the pulse may be found in cases of adhesive mediastino-pericarditis (especially when there are fibrous adhesions around the origin of the great vessels), pericardial effusion, and cardiac dilatation—conditions in which ventricular systole is impeded during inspiration. Inspiratory diminution of the volume of the pulse in only one radial artery may be due to inflammatory adhesions between the pleura and the subclavian artery.

(*b*) The Blood Pressure within the Artery.—As has been already stated, the blood pressure within an artery is raised with each ventricular systole. We must therefore distinguish between (1) the systolic or maximum, (2) the diastolic or minimum, and (3) the mean arterial blood pressure. In order to estimate the blood pressure accurately, recourse must be had to one or other of the modern sphygmomanometers. By digital examination we can only approximately determine the systolic and diastolic pressures.

To determine the Systolic Pressure.—Lay three fingers over the radial artery, and while pressing firmly with the peripheral finger so as to occlude the vessel and thereby prevent any retrograde pulsation from reaching it, pressure is made with the middle finger so as to appreciate the full volume of the pulse. The pressure of the proximal finger is then gradually increased until the pulse is no longer felt by the middle finger. The degree of pressure applied by the proximal finger is the approximate index of the systolic pressure. One of the main sources of error is that the requisite pressure of the proximal finger varies not only according to the systolic pressure, but also according to the diameter of the artery, and therefore (see p. 172) to the volume of the pulse. Thus

the same pressure may be required to obliterate the pulse which is in one instance of high pressure and low volume, and in another of lower pressure but greater volume.

To estimate the Diastolic Pressure.—Applying gradually increasing pressure with two or three fingers, we find that the volume of the pulse gradually increases until it attains its maximum. It then gradually decreases if the pressure is still further increased. By determining the degree of pressure required to elicit the full volume of the pulse, we approximately estimate the diastolic pressure. For the volume of the pulse is greatest when the diastolic pressure within the artery is equalled by that external to it. High diastolic pressure is usually due to increased peripheral resistance—in other words, to increased obstruction to outflow through the arterioles, as in arterio-sclerosis and chronic interstitial nephritis. The pulse is then spoken of as *hard*, *tense* or *incompressible*; when the pressure is low, the pulse is termed *soft*, or *compressible*. The chief difficulty in correctly estimating the diastolic pressure is the simulated impression of high pressure given to the finger by a rigid condition of the arterial wall.

(*c*) Other Characters.—When the rise of pressure is sudden, the increased pressure ill-sustained, and the fall of pressure again sudden, giving to the finger the impression of a very quick stroke, or of a jerking and collapsing pulse, we term it a *water-hammer pulse*, *quick pulse*, or *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 *slow pulse*, or *pulsus tardus*, is distinguished by the slow manner in which the wave of pressure rises and falls. 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 arterio-sclerosis.

When the arterial blood pressure is low and the pulse of considerable volume, the dicrotic wave may be felt as a second wave after each beat of the pulse (*dicrotic pulse*). The significance of that wave is considered more fully on p. 186.

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

Auscultation of the Arteries.—As in cardiac auscultation, so also in auscultation of the arteries, we have to distinguish two phenomena—sounds and murmurs.

1. 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 relaxation of the artery. Of these the latter is simply the aortic element of the second sound conducted into the carotid, and it seems most probable that the sound coinciding with the arterial expansion originates in vibrations of the arterial wall. These two sounds can also generally be heard in the subclavian, and occasionally the sound corresponding to the arterial expansion 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 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 murmur (*Duroziez's sign*) may be produced in the femoral artery in cases of aortic incompetence by pressure with the stethoscope. The one murmur is caused by the pulse wave; the other, a softer murmur, is due to the returning backward wave, which is artificially produced in such cases and flows towards the heart during the arterial collapse.

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

Capillaries

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.

Veins

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 p. 131.

Undulation of the Veins of the Neck.—The pulsations in the cervical veins, which correspond to the movements of the heart, are described on pages 187 and 189. It only remains to mention the undulation which the respiratory movements sometimes produce in the jugular veins. When there is increased intrathoracic pressure and the cervical veins are overfilled, as in pulmonary emphysema, each inspiration diminishes the venous distension, while each expiration increases it, and so the veins show a constant undulation, synchronous with respiration. The expiratory swelling of the jugular veins becomes more marked on forcible expiration, as on coughing.

Inspiratory swelling of the cervical veins may occasionally be observed when there is obstruction to the flow of the venous blood within the thorax during inspiration, as in cases of mediastinal tumour and adhesive mediastino-pericarditis.

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 when auscultation is performed just external to the clavicular head of the right sterno-mastoid muscle. More rarely a murmur may be heard over the large intrathoracic venous trunks, the superior vena cava, and the innominate 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 subclavian, 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.

There are many theories to account for this venous murmur. The most important factors in its production appear to be: 1st, the increased volume of the blood in chlorosis; 2nd, increased rapidity of the blood current; 3rd, lowered viscosity of the blood; and 4th, the change in the calibre of the vein at any particular point (such as occurs in a marked manner at the jugular bulb).

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 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 hæmic 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 ventricular 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.

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. Examination of the pulse by means of the sphygmograph, however, cannot entirely replace the examination by means of the fingers. The sphygmograph is capable of eliciting very accurate information regarding the frequency and rhythm of the pulse, but relatively little concerning the condition of the artery, the volume and celerity of the pulse and the arterial blood pressure. The permanence of the records which may be obtained by the use of the sphygmograph, their value in illustrating the history of individual cases, together with the fact that instruments give results which are more purely objective than those obtainable by other methods, would alone render the sphygmograph an instrument of great value. Within recent years, however, the importance of the sphygmograph in clinical medicine has increased enormously, for tracings taken simultaneously from the radial artery and the jugular vein may afford information regarding disturbances of the rhythm and rate of the heart which can be obtained by no other means.

Sphygmograph.—Many varieties of sphygmograph have been devised and employed. The original instrument of Marey, in its latest modification, gives most satisfactory pulse tracings. The pad of the instrument rests on the radial artery, and the movements of the pad are communicated to a light lever the end of

which is in contact with a paper which has been smoked over a piece of burning camphor. The paper is driven by the clockwork of the sphygmograph.

Dudgeon's sphygmograph, though condemned by some writers, is the instrument most frequently used in this country. Both

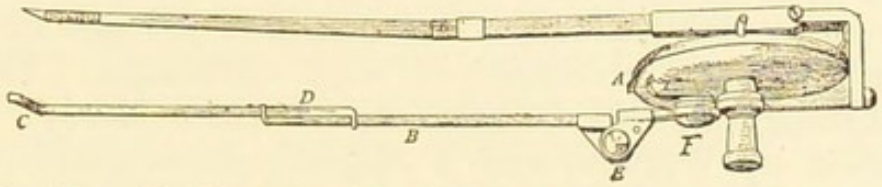


FIG. 76.—Mackenzie's clinical polygraph (after Nicholson).

these sphygmographs, however, are lacking in two essentials, namely, a time marker and apparatus for recording the cardiac impulse or jugular pulsations. At a small cost Dudgeon's sphygmograph can be fitted with Nicholson's time marker, which

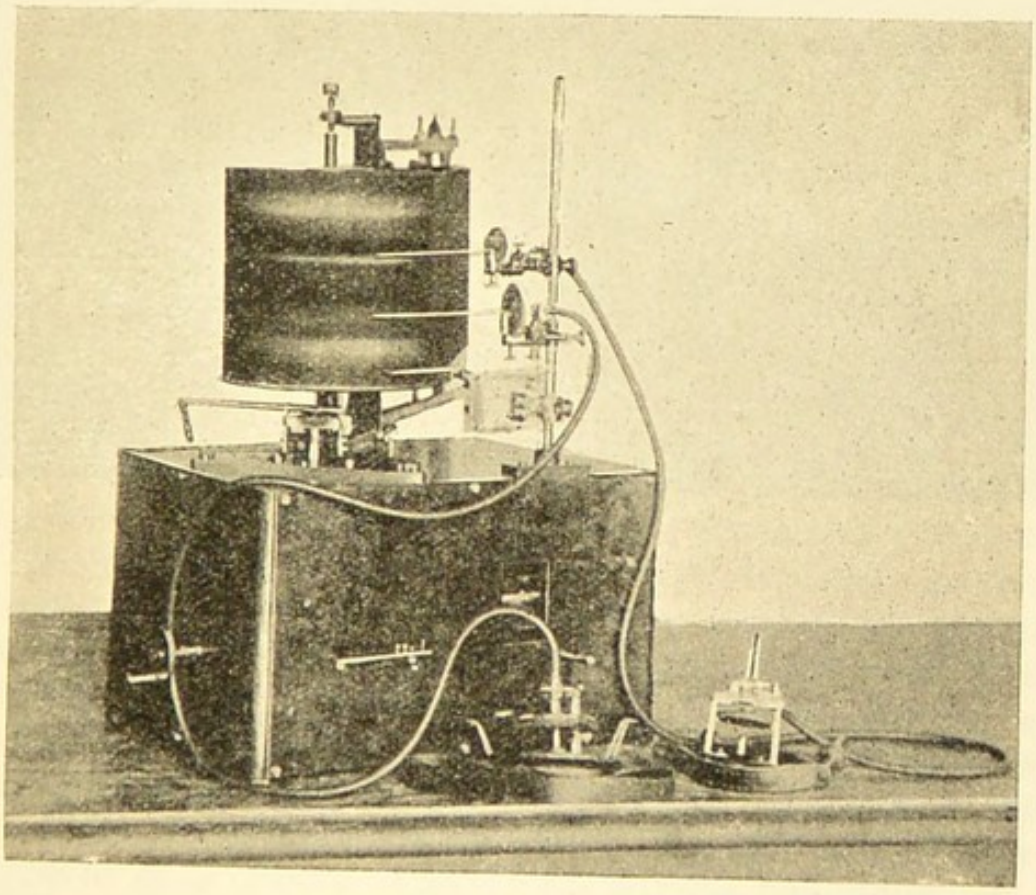


FIG. 77.—The Knoll-Hering kymograph.

records 0·2 of a second. When Mackenzie's polygraph—consisting of a small shallow cup or "receiver" connected by an india-rubber tube with a tambour (Fig. 76)—is also attached to Dudgeon's sphygmograph, we have an efficient and portable instrument with

which the radial and jugular pulsations, or radial pulse and apex-beat, can be simultaneously recorded.

The new model of Jaquet's sphygmograph has a time marker recording 0·2 of a second, and bears two tambours, so that the jugular pulsation and cardiac impulse can both be recorded simultaneously with the radial pulse.

When the portability of the instrument is of lesser importance, a kymograph may be employed with advantage. The Knoll-Hering kymograph (Fig. 77), fitted with three Marey's tambours (as modified by Waraus) and a Jaquet's chronograph recording either 1 or 0·2 second, is the most satisfactory.

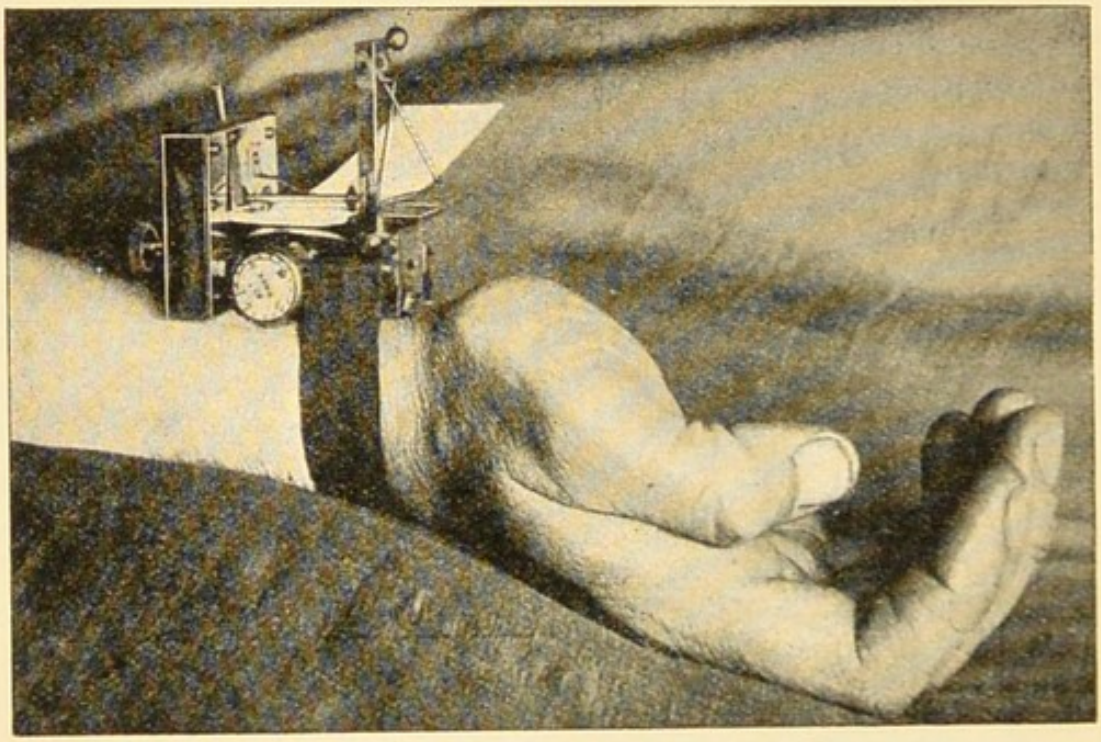


FIG. 78.—Dudgeon's sphygmograph.

METHOD OF OBTAINING TRACINGS WITH DUDGEON'S SPHYGMOGRAPH.—Mark the position of the right radial artery by means of a dermatograph pencil; wind up the clockwork of the sphygmograph and attach the instrument to the right wrist by means of the two elastic bands, so that the pad rests upon the artery (Fig. 78). Then insert the smoked paper between the wheels and the roller and set the clockwork in motion so as to drive the smoked paper onwards for about half an inch, namely, until the writing lever rests on it. Then stop the clockwork. Now tighten the elastic bands and increase or decrease the pressure of the spring until the oscillations of the writing lever are of maximum amplitude with each beat of the pulse. Then set the clockwork in motion; stop it when the end of the paper is within half an

inch of the lever. Having withdrawn the paper, write by means of a finely pointed instrument the patient's name, the vessel from which the tracing was taken, and the date. Then immerse the paper in photographic negative varnish,[1] and hang up the paper or lay it on blotting-paper until it is dry.

To obtain simultaneous Tracings of the Radial and Jugular Pulsations.—The tambour of the polygraph having been attached to Dudgeon's sphygmograph, the latter is applied to the right wrist in the manner already described, and the lever of the tambour is adjusted so that it writes just over the lever of the sphygmograph recording the radial pulse. The receiver is then pressed lightly over the lower end of the relaxed right sterno-mastoid muscle, or immediately to its outer side. The correct pressure is applied when the lever of the tambour oscillates most freely. The clockwork is now set in motion, and the two tracings simultaneously recorded on the smoked paper. Or the receiver may be applied to the apex-beat or the epigastrium and the movements of the left and right ventricle recorded.

The Sphygmogram in Health.—The systolic rise of blood pressure constituting the pulse wave is represented in a sphygmogram (Fig. 79) by a line ascending abruptly till it reaches its highest point (*p*), the apex of the percussion wave. The more oblique line of descent presents in succession the systolic, tidal or predicrotic wave (*s*), the dicrotic or aortic notch (*n*) and the dicrotic wave (*d*), and the line of descent then falls to its lowest level, although the fall may be further interrupted by secondary waves (see Fig. 80). As the commencement of the upstroke corresponds to the opening of the aortic valves, and the dicrotic notch to their closure, the aorta is, during the time represented by the interval between these events, in free communication with the interior of the ventricle. This interval is therefore termed the *systolic* period. During the time of the rest of the curve (the *diastolic* period) the aortic valves are closed. The sphygmogram is thus divided at the dicrotic notch into its two fundamental parts, the systolic and the diastolic.

The most satisfactory explanation of the appearance of the percussion wave, apart from the tidal (systolic) wave, is that the upward movement of the lever is greater than the corresponding movement of the arterial wall which produces it, and that when the lever has reached its highest point it falls until it is again

[1] The following is a good recipe for a varnish:—Dissolve 50 grammes of white shellac and 10 grammes of gum mastic in 600 c.c. of methylated spirit. The mixture is allowed to stand for two or three days, and is then filtered.

caught and raised by the wave of increased pressure which is only now attaining its maximum. When the arterial blood pressure is relatively low, the percussion and tidal waves are fused in one—the systolic wave.

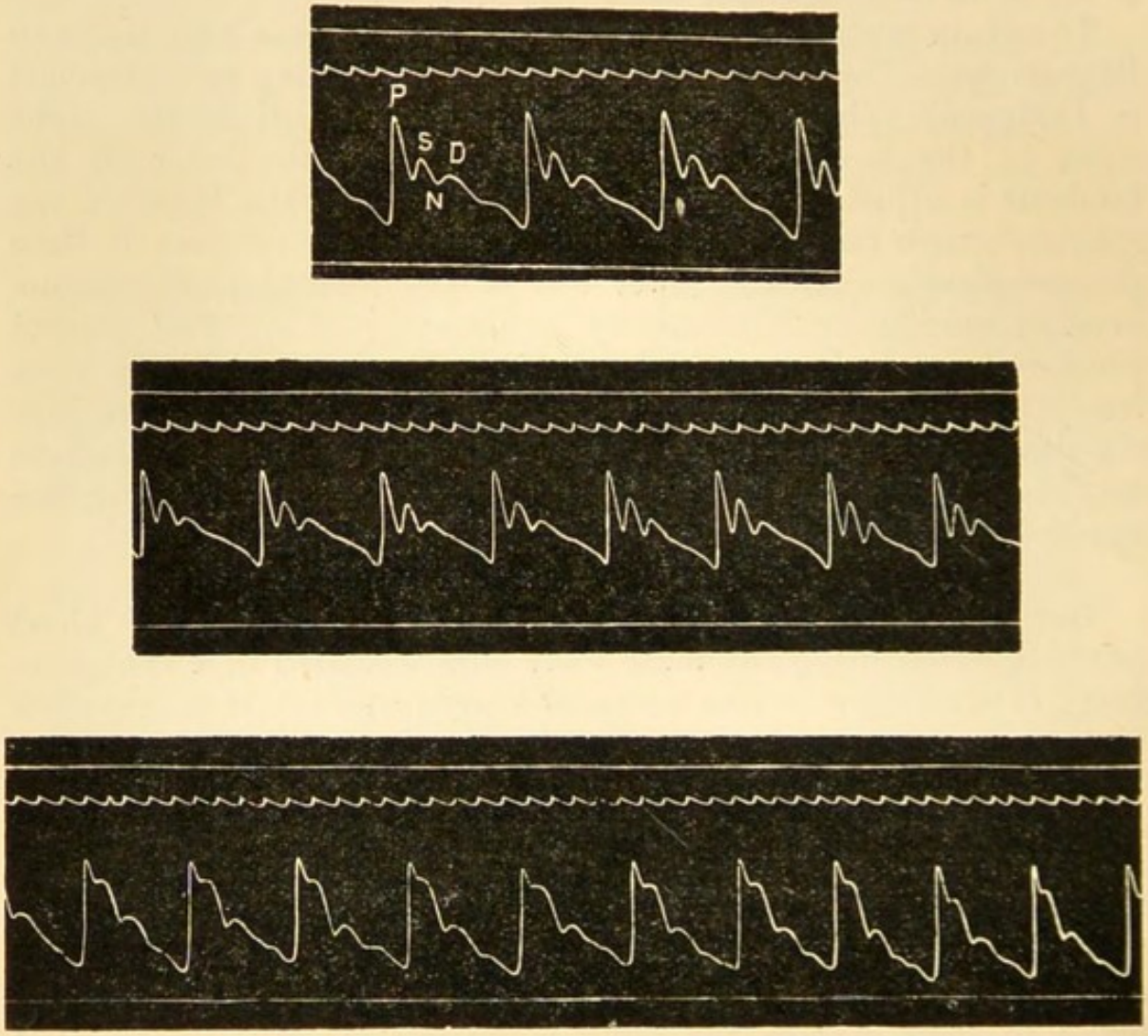


Fig. 79.—Normal pulse tracings.

The dicrotic wave is due to the elastic recoil of the aortic valves subsequent to their closure, while the secondary waves after the dicrotic wave are probably due to reflections of that wave.

In a sphygmogram the following points should be noted :—

1. **Rate** of the Pulse.—This can be accurately studied only when there is a simultaneous record of the time marker (as in Figs. 79, 80, etc.).

2. **Rhythm** of the Pulse.—This is best studied by measuring, with a pair of compasses the interval between the commencement

of each line of ascent. Irregularity of rhythm is considered on page 187. It must not be confounded with irregularity due to instrumental defect, when the rate at which the smoked paper travels is not uniform. The rate at which the paper travels may decrease too rapidly, so that there may apparently be an increase in the frequency of the pulse towards the terminal portion of the tracing. Or the paper may be partially arrested at times and

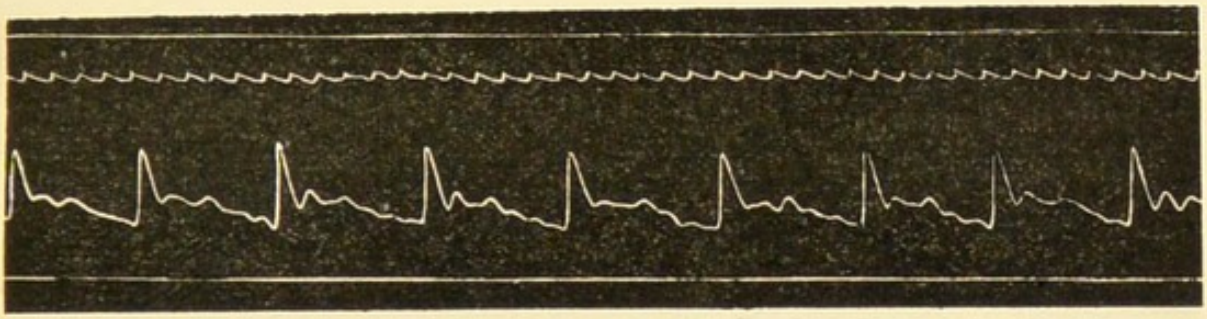


FIG. 80.—Pulse tracing showing secondary waves after the dicrotic wave.

arhythmia of the pulse thus simulated. The recognition of such instrumental defects is rendered easy by a study of the tracing recorded by the time marker.

3. **Volume** of the Pulse.—This is estimated by the height to which the tracing at each beat rises above the lowest level. Normally the height of the percussion wave is uniform throughout the tracing, except for such variations as are associated with respiration. But the volume of the pulse varies in different

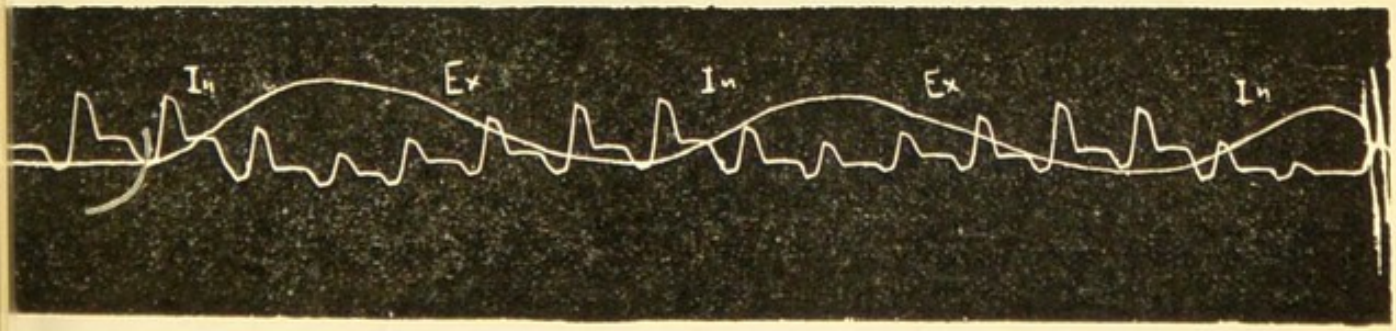


FIG. 81.—Inspiratory diminution of the pulse-curves in a slight case of spasmodic asthma. (Identical in appearance with the true pulsus paradoxus of mediastino-pericarditis) (after Nicholson).

tracings from any patient according to the tension of the elastic bands and the spring of the sphygmograph. A pulse of small volume may also be simulated if the pad of the sphygmograph be not placed exactly over the vessel. Inequality in the volume of the pulse is usually associated with irregularity of rhythm (see

p. 187). In the paradoxical pulse (see p. 173) the volume of the pulse is lessened during inspiration, as in Fig. 81.

4. **The Rise and Fall of Blood Pressure.**—In a tracing from a quick pulse, in which the rise and fall of blood pressure are both sudden, the line of ascent and that of descent are abrupt, as in Fig. 85, from a case of aortic incompetence. In a tracing from a slow pulse, on the other hand, the line of ascent rises

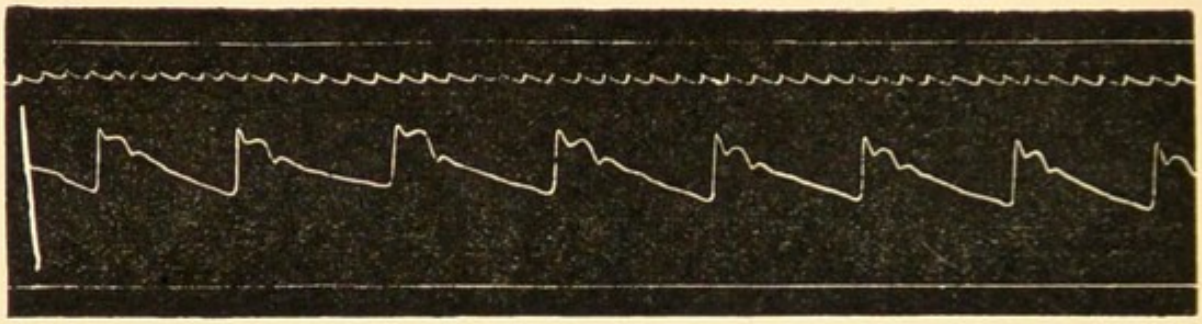


FIG. 82.—High arterial blood pressure.

gradually and the line of descent is usually gradual also, while the apex of the tracing may be rounded when the elasticity of the vessels is defective, as in arterio-sclerosis and atheroma.

5. **The Height of the Blood Pressure.**—The pressure of the spring of the sphygmograph necessary to obtain the maximum excursions of the lever affords no indication of the arterial blood

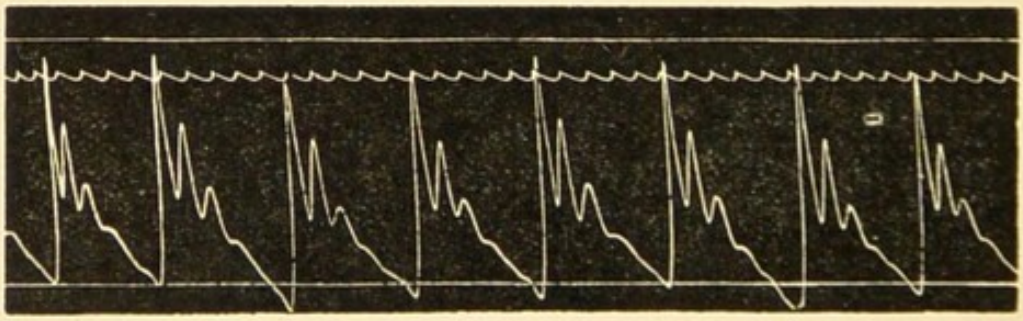


FIG. 83.—High arterial blood pressure (220 mm. Hg, Gärtner's tonometer), with forcibly acting left ventricle. From a case of chronic nephritis.

pressure. When the blood pressure is *high*, the systolic or tidal wave is usually well marked, but more significant is the fact that the dicrotic notch is high above the lowest level of the tracing. The dicrotic wave is usually small (Fig. 82). The line of ascent may be gradual, but when the left ventricle is contracting vigorously the line of ascent may be abrupt and the apex of the percussion wave be high above the lowest level of the tracing (Fig. 83).

When the blood pressure is *low*, the dicrotic notch is low down on the line of descent, the dicrotic wave is usually well marked—the dicrotic pulse—the line of ascent is abrupt, and the frequency of the pulse is usually increased (Figs. 84, 85, and 87).

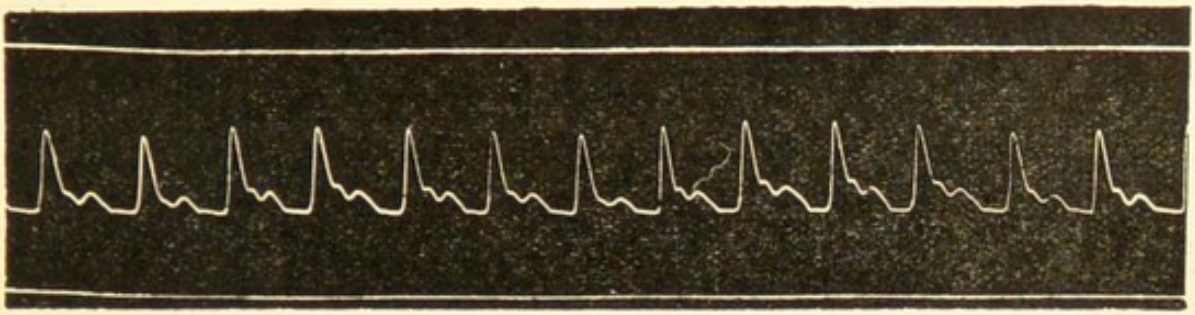


FIG. 84.—Low arterial blood pressure.

Although in a sphygmogram the arterial blood pressure is best estimated by noting the position of the dicrotic notch, this method is not nearly so precise or reliable as the estimation of the blood

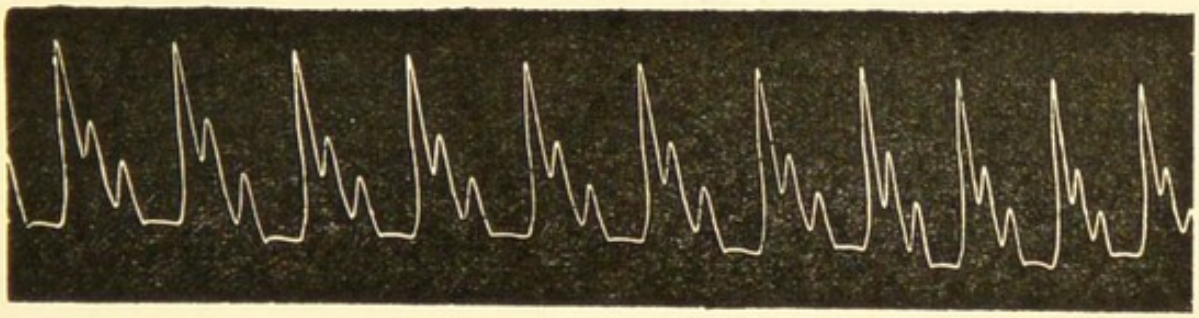


FIG. 85.—Low diastolic pressure. From a case of aortic incompetence.

pressure by means of the sphygmomanometer, which is therefore always to be preferred (see p. 195).

ANACROTIC PULSE.—Those pulse waves in which a more or less

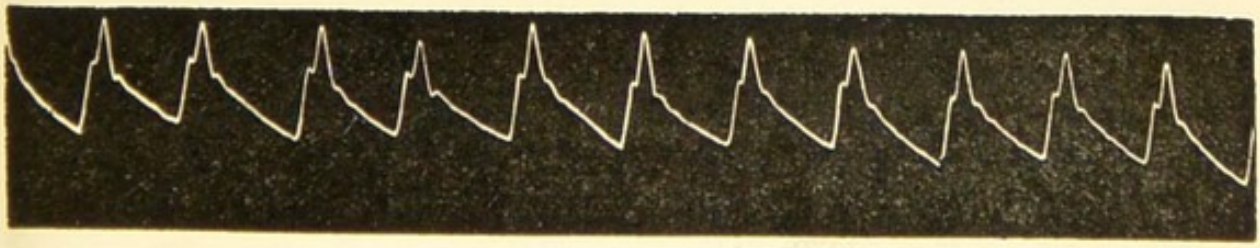


FIG. 86.—The modification of the systolic wave in aortic stenosis—the second summit forms the highest part of the tracing (after Nicholson).

well-marked notch occurs in the ascending line are termed anacrotic. The tracing (Fig. 86) annexed shows a typical example of the anacrotic pulse. It differs from the normal

mainly in that it is the tidal wave and not the percussion wave, which forms the highest point of the tracing. This form of pulse wave is found when there is much resistance opposing the contraction of the left ventricle. Under such circumstances the rise of pressure is more gradual than is normal. The anacrotic pulse may be found in some cases of aortic stenosis, or when there is increased peripheral resistance to the outflow of blood from the arteries, as in cases of peripheral arterio-sclerosis, chronic interstitial nephritis, and arterial hypertonus.

Dicrotic Pulse, Dicrotism.—These terms are applied to the pulse when there is exaggeration of the dicrotic wave (Fig. 87). 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, athough these latter contain relatively normal amount;

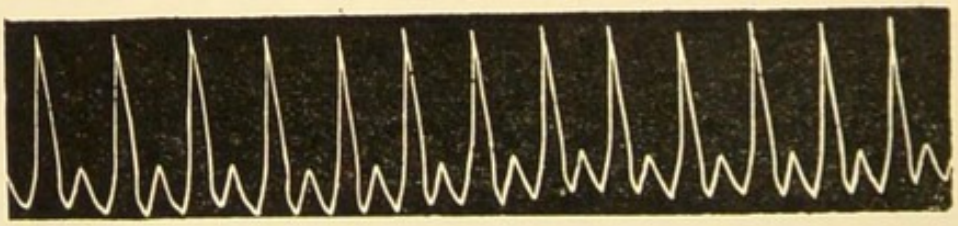


Fig. 87.—Typical dicrotic pulse (enteric fever, 20th day) (after Nicholson).

(*b*) in cases of unusual expansion of the arterioles and capillaries leading to a relatively rapid outflow from the arteries, as when there is fever, and 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. It is to be noted that the dicrotic pulse invariably results from abnormally low arterial pressure, the cause of which, in individual cases, it is rarely difficult to discover.

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 when the blood pressure is low and the rate of the pulse markedly accelerated, each successive cardiac systole follows its predecessor before the pressure within the artery has fallen below that which it presented at the dicrotic notch (Fig. 88).

The student is warned against the fallacy of supposing that

associated with each valvular lesion of the heart there is a characteristic sphygmogram. Thus the characters of a sphygmogram from a case of aortic incompetence may be indistinguishable from those of a tracing from another case in which, although the diastolic pressure is low, the aortic valves are competent.

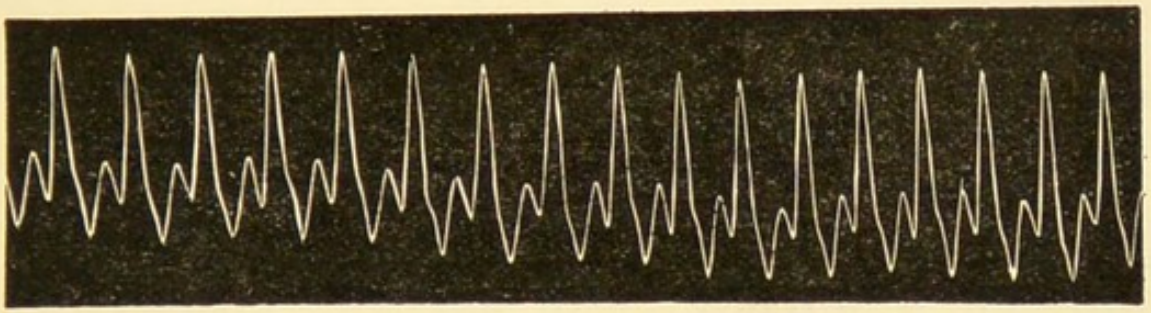


FIG. 88.—Hyperdicrotism of the pulse. This is largely the result of increased inertia of the needle of the sphygmograph and extreme rapidity of the pulse (after Nicholson).

Irregularity of the Pulse.—Reference has already been made to irregularity of the rhythm of the heart, and to the detection of certain forms of irregularity by means of auscultation (see p. 156). But the differentiation of the different forms of irregularity and the recognition of the causal disturbance of

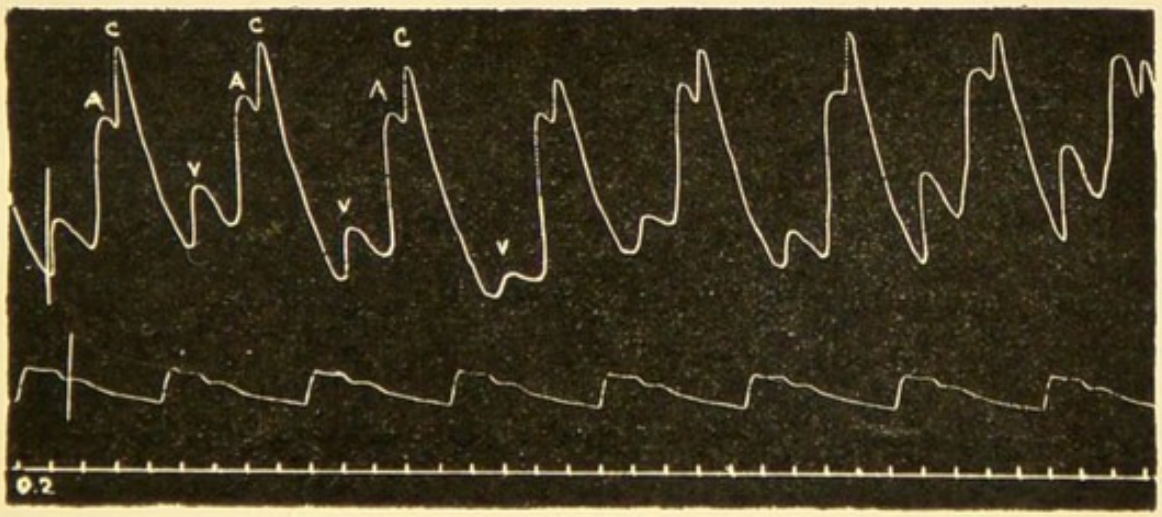


FIG. 89.—Tracings taken simultaneously from the jugular vein and brachial artery. Normal rhythm.

cardiac function is best effected by taking simultaneous tracings from the radial artery and the jugular vein, in the manner described on page 181.

THE EVENTS RECORDED IN A TRACING FROM THE JUGULAR VEIN WHEN THE CARDIAC RHYTHM IS NORMAL.—In Fig. 89 the upper tracing, from the jugular vein, presents three waves (*A*, *C*, and *V*)

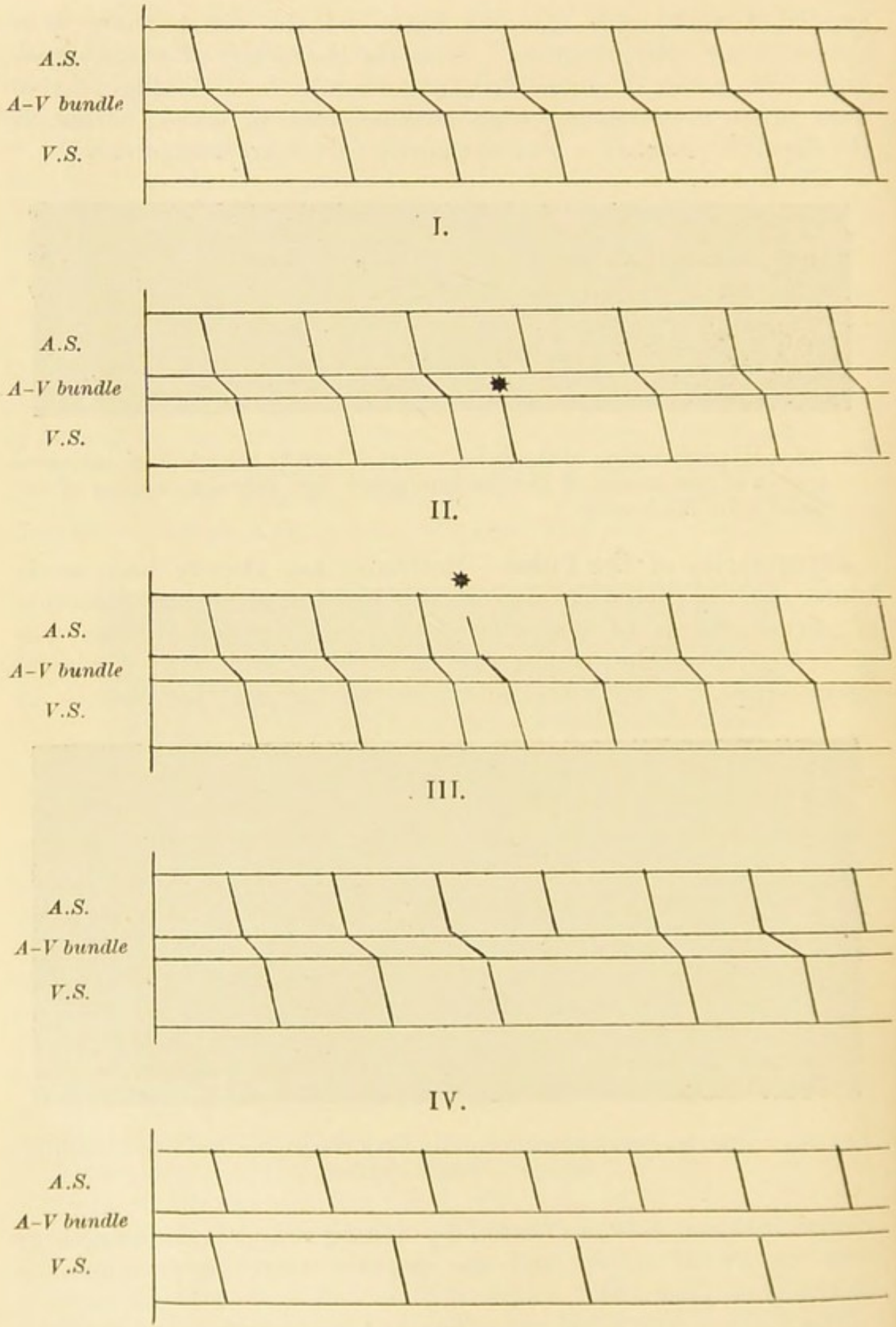

A.S.
A–V bundle
V.S.
I.
A.S.
A–V bundle
V.S.
II.
A.S.
A–V bundle
V.S.
III.
A.S.
A–V bundle
V.S.
IV.
A.S.
A–V bundle
V.S.
V.

Fig. 90.

occurring in succession. One of these waves is found, on measurement with a pair of compasses, to immediately precede the arterial pulse wave in the lower tracing, and is indicated by the letter *C*, as it represents the pulse wave transmitted through the vein from the adjacent carotid artery. The wave *C* varies considerably in size in different tracings. The wave *A* preceding the carotid wave is due to auricular systole, the rise of pressure in the right auricle when it contracts causing a wave of increased pressure within the vein. This wave is therefore termed the auricular wave. The third wave, *V*, following the carotid wave, is termed the ventricular wave. In the case of a normal heart this wave probably represents the increase of pressure which occurs within the vein when the right auricle becomes distended with blood in diastole. Whenever there is engorgement of the right auricle, the ventricular wave *V* becomes more pronounced, and occurs earlier in the cardiac cycle. In a case of well-marked tricuspid incompetence there is only one wave in the jugular tracing, namely, a wave synchronous with and due to the systole of the right ventricle (see Fig. 95).

For the two main forms of the jugular pulse the nomenclature suggested by Mackenzie is now universally adopted. When the tracing presents a well-marked auricular wave we speak of an "auricular venous pulse"; when the auricular wave is not present, and the sole wave is synchronous with ventricular systole, we speak of a "ventricular venous pulse" (see Fig. 95, p. 193).

In order to understand the various irregularities of the pulse, it must be remembered that inherent in the cardiac muscle

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FIG. 90.—Diagrams to illustrate some forms of pulse irregularity.

I. Represents the normal passage of the contraction stimulus along the auricular fibres (*A.S.*), the auriculo-ventricular bundle, and the ventricle (*V.S.*). Each auricular systole is followed by a ventricular systole.

II. The action of the heart on the occurrence of a ventricular extra-systole originating at *. The stimulus from the next auricular systole is not transmitted to the ventricles. The compensatory pause is complete.

III. The action of the heart on the occurrence of an auricular extra-systole. The abnormal stimulus originates in the auricular fibres and is transmitted to the ventricles. The compensatory pause is incomplete.

IV. Owing to depression of conductivity at the auriculo-ventricular bundle (indicated by the obliquity of the lines in the middle division of the diagram) some contraction stimuli are blocked at that point, and ventricular systoles are therefore occasionally dropped.

V. The block at the auriculo-ventricular bundle is complete. No stimulus passes from the auricles to the ventricles. The ventricles beat less frequently than the auricles, and with an independent rhythm.

fibres there are the functions of rhythmically creating contraction stimuli, receiving the stimuli (excitability), contracting in response to a stimulus (contractility), conducting the stimulus from one muscle fibre to another (conductivity), and maintaining a degree of tone. The normal contraction stimulus originates in the auricular muscle fibres at the venæ cavæ, is conducted from muscle fibre to muscle fibre along the auricular walls, and thence by the auriculo-ventricular bundle of His (Gaskell's bridge) to the muscle fibres of the ventricles. It must also be remembered that the different functions of the cardiac muscle fibres may be independently or conjointly modified by nervous influences.

The brilliant researches of Mackenzie and Wenckebach have led to a general recognition of the following conditions, which we have to consider briefly :—

I. Irregularity caused by an Extra-systole.—An extra-systole is a cardiac contraction which does not originate in the auricle at the venæ cavæ. It is followed by a more or less complete "compensatory pause," so that the physiological rhythm of the heart is, or tends to be, maintained.

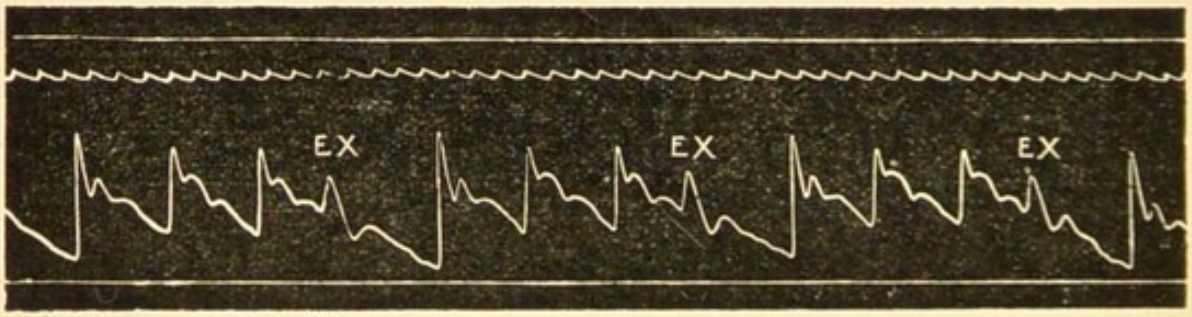


Fig. 91.—Pulse irregularity due to a ventricular extra-systole after every third beat.

Ventricular Extra-systole.—In this, which is the more frequent variety, the contraction originates in the muscle fibres of the auriculo-ventricular bundle. The resulting irregularity of the pulse is readily recognised in the sphygmogram (see Fig. 91). The extra-systole appears as a small wave occurring prematurely and followed by a long pause. By means of a pair of compasses, it is found that this compensatory pause is "complete," *i.e.* the time elapsing between the beat which precedes the extra-systole and that which follows it is exactly equal to the two preceding pulse-periods. The first pulse wave following the compensatory pause is of larger volume than those immediately succeeding it, because the ventricle during the long diastolic pause has acquired an unusual degree of contractility and also become more fully distended with blood than during a normal diastolic period.

If an extra-systole occurs very soon after a physiological systole, the former is not represented by a wave in the sphygmogram; there is merely an "intermission." The later the extra-systole

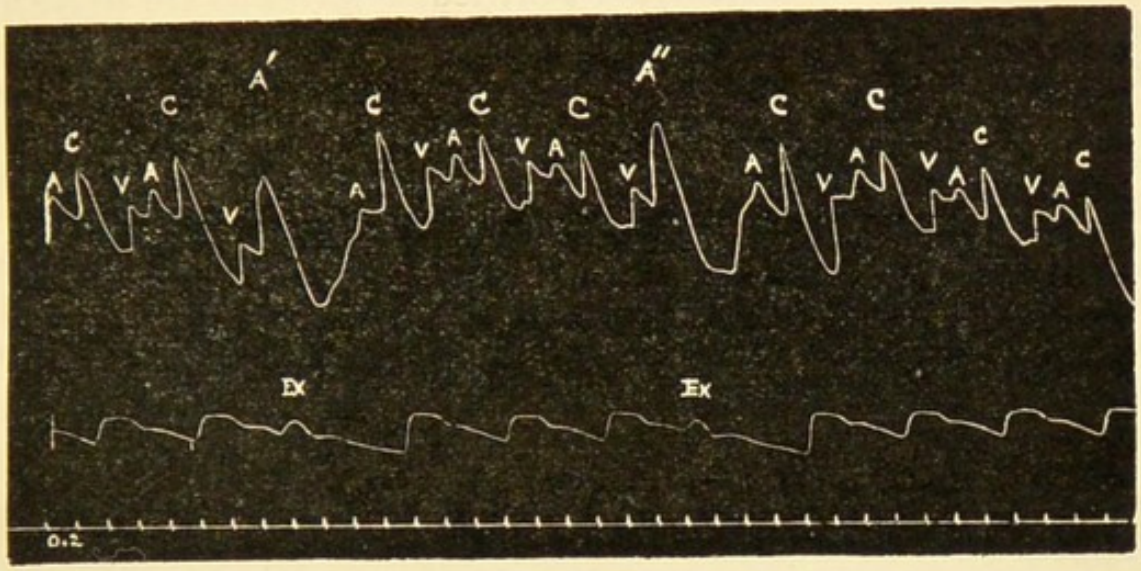


FIG. 92.—Simultaneous tracings from the jugular vein and brachial artery. Irregularity due to ventricular extra-systoles (Ex).

occurs, the larger is the wave representing it in the tracing (see Fig. 94).

In the duration of the compensatory pause we have a fairly reliable means of differentiating a ventricular extra-systole from

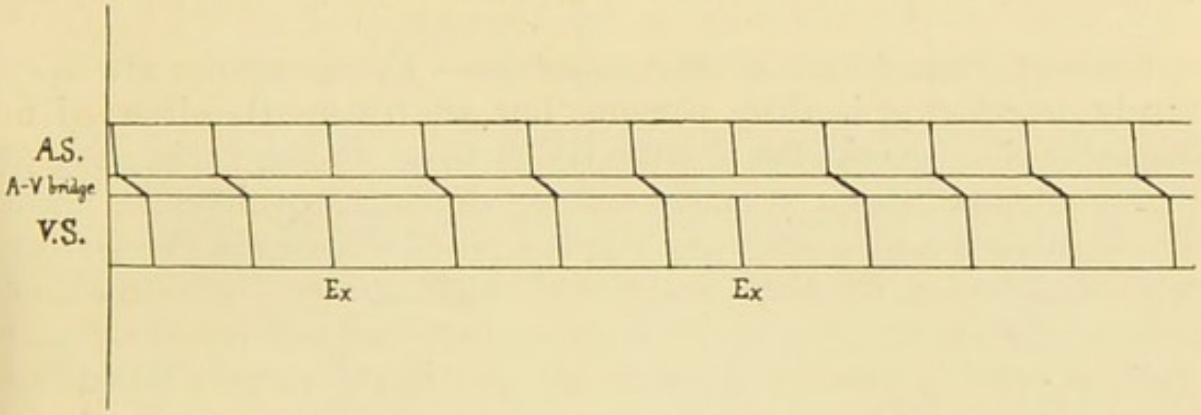


FIG. 93.—Diagram constructed to show the events recorded in Fig. 92. In the first two beats of the heart the contraction stimulus is transmitted from auricles (*A.S.*) over the auriculo-ventricular bundle to the ventricles, which respond by contracting (*V.S.*). At Ex there is a premature ventricular contraction, followed by a compensatory pause.

one of auricular origin. But in order to confirm the diagnosis, it is well to take simultaneous tracings from the jugular vein and the radial artery. In Fig. 92 the upper tracing is from the jugular vein, the middle tracing is from the arterial pulse, the lower tracing is that of the chronograph marking 0·2 of a second.

When the block at the auriculo-ventricular bundle is complete, so that no stimulus passes from auricle to ventricle, there is the condition of complete heart-block (Fig. 97 and Plate IV.). The auricles continue to contract rhythmically, whilst the ventricles, manifesting their power of rhythmically creating contraction stimuli, beat less frequently than the auricles and with an independent rhythm (see Fig. 90, V.). This is the condition found in those cases of bradycardia where the rate of the pulse is persistently about 30 per minute, or still less frequent.

Paroxysmal accesses of pronounced bradycardia, attended by syncopal or epileptiform attacks, constitute the *Stokes-Adams syndrome.*

IV. Disturbance of Contractility.—When contractility is the only cardiac function disturbed, the pulse may assume that form known as the *pulsus alternans*, in which every alternate beat is of smaller volume than that of the beats preceding and following it,

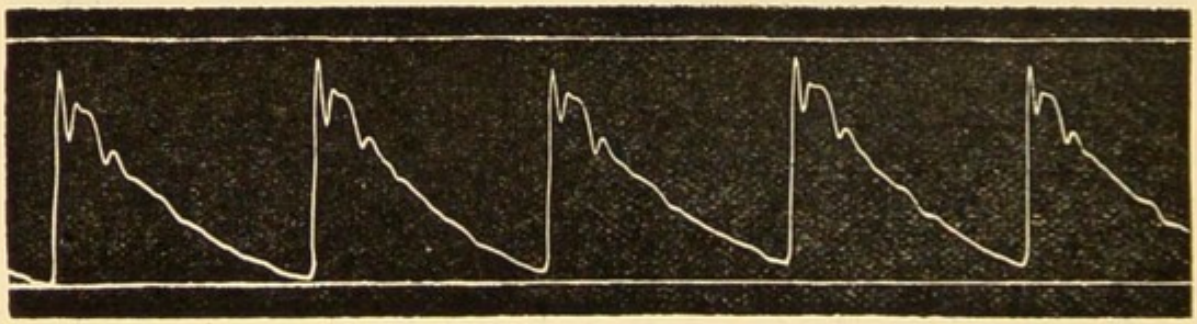
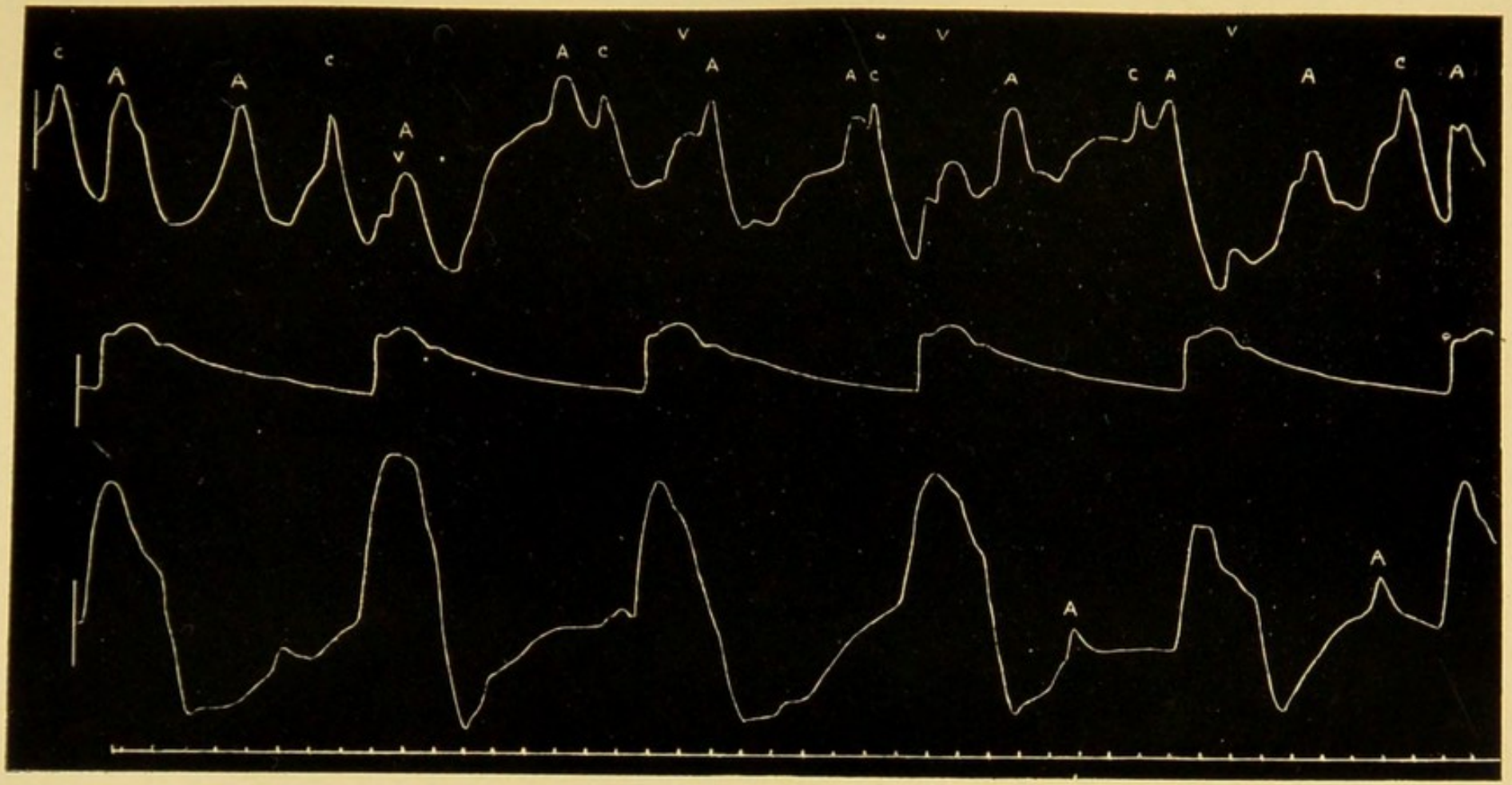


Fig. 97.—Bradycardia (pulse rate 34 per minute) due to heart-block.

but where the pulse waves occur at regular or almost regular intervals of time. This abnormality is rarely seen. It may be simulated when an extra-systole occurs after each normal beat of the pulse. But the true *pulsus alternans* is more permanent than the irregularity resulting from extra-systoles.

The Cardiogram.—Simultaneous tracings taken from the radial artery and apex-beat may confirm and amplify the information obtainable by a simultaneous record of the pulsations of the jugular vein and radial artery. In the normal cardiogram, taken from the apex-beat, we note the wave *a* (Fig. 98), due to the systole of the left auricle, the abrupt line of ascent (the space *D*) marking the commencement of ventricular systole, the space *E* ("the systolic plateau") corresponding to the maintenance of the cardiac apex against the parieties while the ventricle is emptying and the semilunar valves remain open; the space *F*, in which the line of the tracing descends owing to relaxation of the ven-



Simultaneous tracings from a case of heart-block. The upper tracing is from the jugular vein, the middle tracing from the brachial artery, the lower tracing from the apex-beat. Below the tracings the time is recorded in 0·2 of a second. The auricles contract at the rate of 59·3 beats in a minute, the ventricles contract at the rate of 32·8 beats per minute.

[*To face page* 194.

tricular muscle; and lastly the space *G*, during which the ventricle is filling, and the line of the tracing gradually ascends. The auricular wave in the cardiogram is always small, and in many cases there is no record of it.

SPHYGMOMANOMETRY, or the Instrumental Determination of Arterial Blood Pressure.

By means of a sphygmomanometer we can estimate the pressure which must be exerted on the outside of an artery in order to equalise that within the vessel. Some instruments record the systolic, others the diastolic, pressure within the vessel. Estimations made by von Basch's and the earlier forms of Oliver's instruments are less accurate than those obtained by the use of an instrument in which pressure is applied by means of an india-

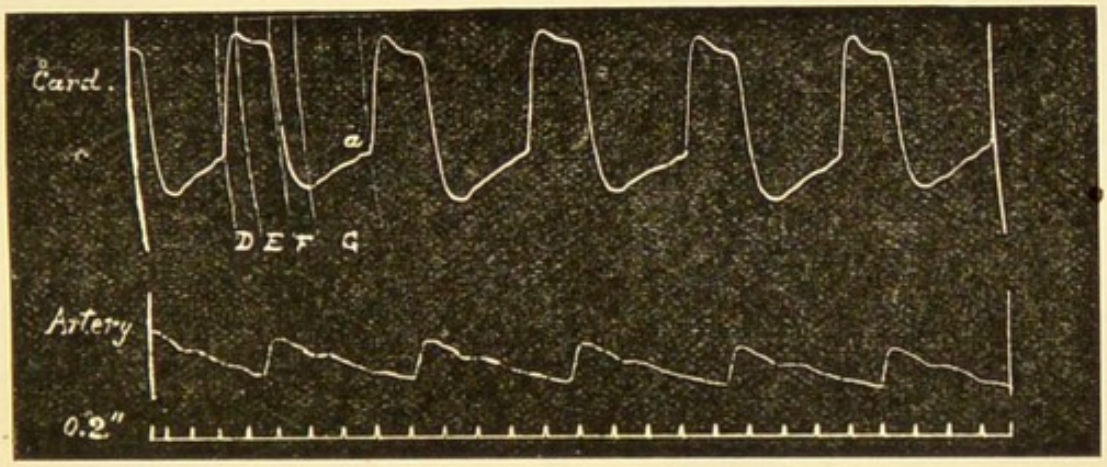


FIG. 98.—Cardiogram and sphygmogram taken simultaneously.

rubber bag encircling the arm or finger, so that the artery is equally compressed from all sides against the adjacent bone. Of the many sphygmomanometers, in which the method of circular compression by air is employed, we may mention the three most frequently employed.

1. RIVA-ROCCI'S SPHYGMOMANOMETER.—Of this excellent instrument there are many modifications. The cistern of the manometer (Fig. 99) is filled with mercury up to the zero mark. The armlet, which must be 12 cm. wide, is adjusted around the patient's right upper arm, which has been bared and is supported on a level with the heart. The physician places the fingers of his left hand on the right radial artery, and by means of the inflating bulb gradually raises the pressure in the apparatus until the radial pulse is no longer felt. The pressure is then gradually relaxed by turning the screw valve until the radial pulse

reappears, when the height of the column of mercury in the manometer is noted. The maximum systolic pressure in the brachial artery is thus estimated. It is normally between 125 and 135 mm. Hg.

2. Gärtner's Tonometer (Fig. 100) is the most compact and portable of reliable sphygmomanometers. Each time before it is employed the mercury index must be set at zero. To do so, turn the right-hand stopcock and the black knob of the left-hand (three-way) stopcock to "Einstellung," and tighten or relax the screw which acts on the indiarubber tube attached to the upper (or right-hand) end of the manometer, until the upper end of the

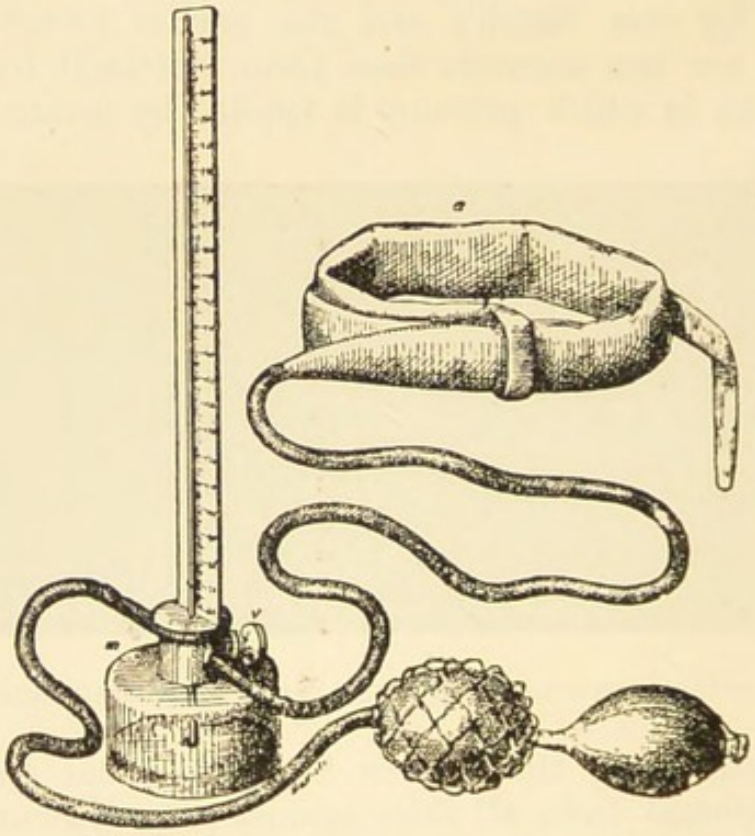


Fig. 99.—Sphygmomanometer of Riva-Rocci (after Luciani).

mercury index is at zero. Then turn the right-hand stopcock and the black knob of the left-hand stopcock to "Messung." Place the pneumatic ring around the second phalanx of a finger, which is maintained at the level of the heart. A stout indiarubber ring is then rolled over the forefinger as far as the last interphalangeal joint and the terminal phalanx thus rendered bloodless. The pressure in the pneumatic ring is then raised by means of the screw apparatus in the middle of the box, so as to arrest the circulation in the finger, *i.e.* until the manometer records a pressure of about 200 mm. Hg. Then withdraw the indiarubber ring from the finger, and by means of the screw apparatus

gradually release the pressure within the pneumatic ring until the blanched finger is observed to suddenly flush, the skin at the

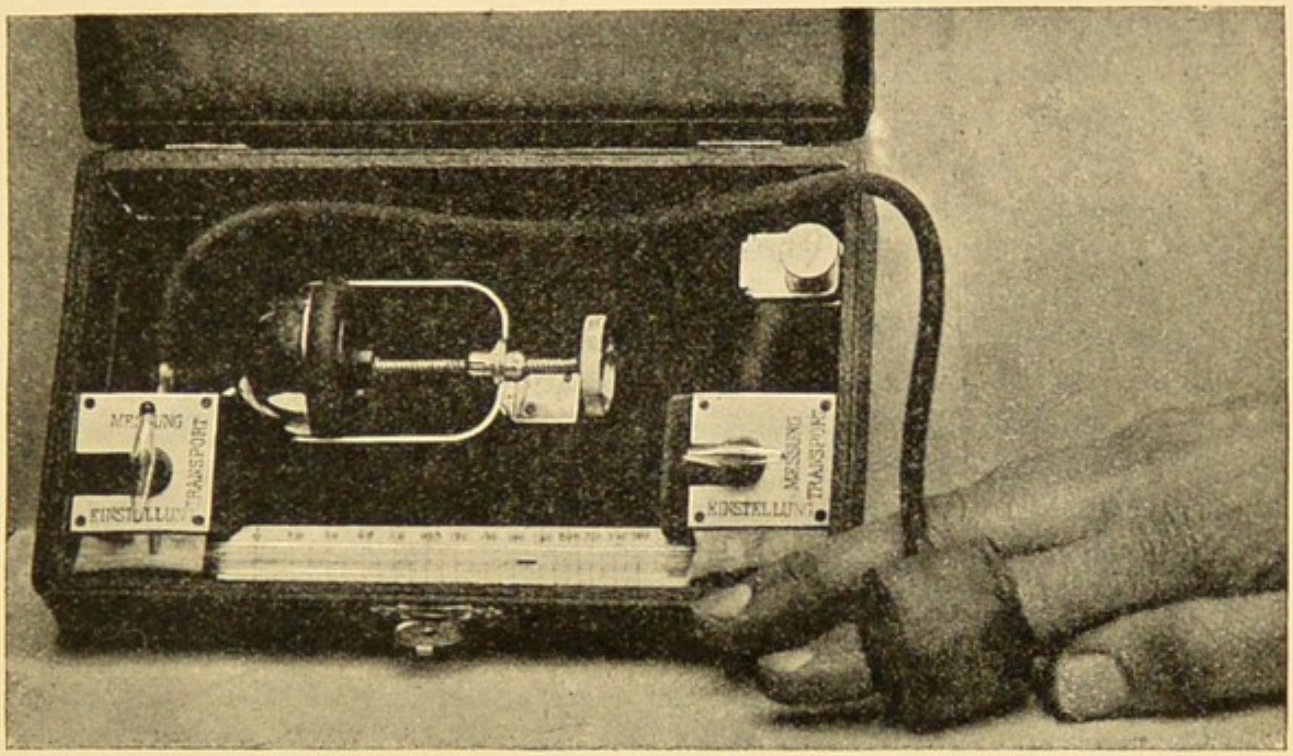


FIG. 100.—Gärtner's tonometer.

side of the finger nail becoming as red as, or redder than, the corresponding parts of the adjacent fingers. The level of the

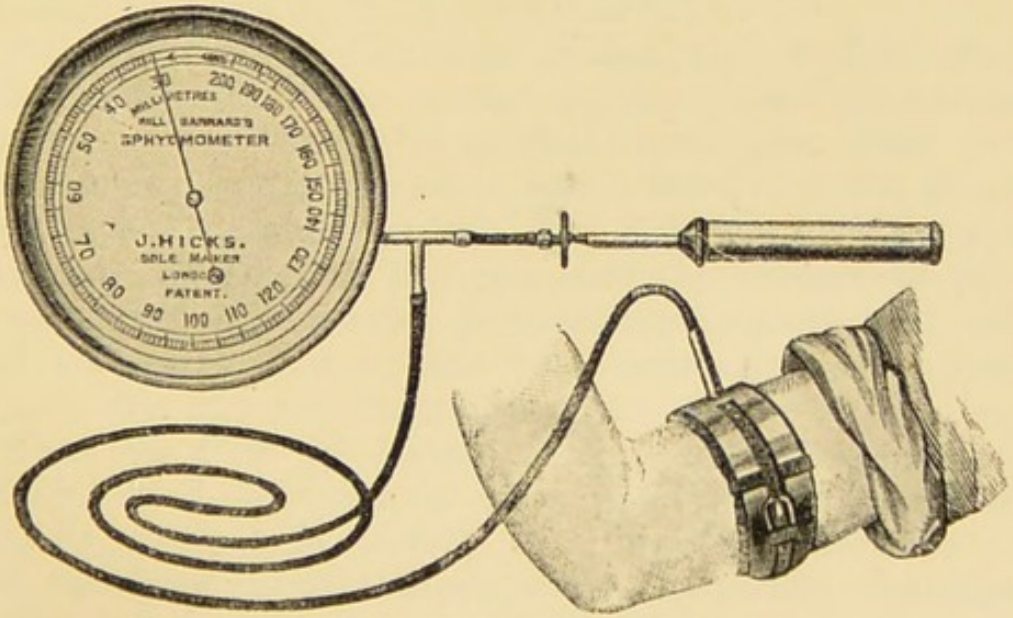


FIG. 101.—Hill and Barnard's sphygmometer.

mercury index is then read off, and corresponds to the maximum systolic pressure in the digital arteries. In normal adults the limits, with this instrument, are from 90 to 120 mm. Hg,

being somewhat lower in women than in men. High pressures much exceeding these, and running as high as 250 mm., are met with in such affections as chronic Bright's disease with cardiac hypertrophy, while in such conditions as pulmonary phthisis and other wasting diseases, typhoid fever, shock, and collapse, the blood pressure is found to be very low, 70 to 80 mm., or less.

3. Hill and Barnard's Sphygmometer.—The armlet (Fig. 101) is strapped around the bare upper arm and is connected by a T-tube with the spring manometer. By means of the air pump the pressure is gradually raised, until the oscillations of the manometer needle at each pulsation are maximal. The pressure is now raised above the systolic pressure and then gradually released at the valve, and the lowest pressure at which the oscillations are still maximal is noted. This should correspond with the former reading, and indicates, according to Hill, the mean pressure, according to others the diastolic pressure. The average pressure is equal to 120–125 mm. Hg. It is often difficult to determine the lowest pressure at which the oscillations are still maximal, and the possibilities of error are greater with this than with the other two sphygmomanometers.

CHAPTER XII

RESPIRATORY SYSTEM

Subjective Phenomena, such as pain, tickling, burning, etc., 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 abnormal pulmonary physical signs, except such alterations of the respiration as the pain occasions, will suffice to distinguish this affection from pleurisy.

(3) *From the Pain of Herpes Zoster.*—This eruption, which is radicular in distribution, is often preceded, accompanied, or followed 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.

[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.

(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. 225).

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 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 products as 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. 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 quiescent intervals. 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. Its Character.—This may vary very greatly. The cough may be dry, as in pleurisy, the first stage of phthisis, etc., or moist, as in chronic bronchitis, and in the late 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. 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. Notice if the paroxysm terminates 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.

SPUTUM

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, globulin, myosin, nuclein, glycogen, various fatty acids, leucin, tyrosin, etc., 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.

The amount of albumin is determined by shaking up the sputa with 3 per cent. acetic acid (to precipitate mucin), filtering, and estimating the percentage of albumin by means of an Esbach's tube, as when estimating albumin in urine (see p. 310). The amount of albumin is much greater in pulmonary tuberculosis than in bronchial catarrh. 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 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 leucocytes.

(2) Muco-Purulent Sputum may occur in almost every affection of the bronchi and lungs. 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 resemblance 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

[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.

of plasma. This variety of sputum is usually derived from suppurating cavities in the lung, or as the result of other collections of pus (for example, empyema) bursting into a bronchus.

(4) Serous Sputum is that form which is met with when copious transudation takes place from the pulmonary capillaries, as in œdema of the lungs. It has a characteristic thin, transparent appearance, and is usually copious and frothy.

(5) Sanguineous Sputum.—The sputum may be simply streaked with blood (as in the early stages of phthisis, etc.), 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, of chronic pulmonary congestion, and of acute 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 can, however, usually be recognised by examining the upper respiratory passages. It is not difficult, as a rule, 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 usually 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 (see p. 85).

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, etc. 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 micro-organisms are present the fœtid 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, saffron, or yellow, as the hæmoglobin becomes more and more altered into hæmatoidin. In gangrene of the lung, and in some cases of acute lobar pneumonia, the sputum has the colour of prune juice. 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. The sputum in asthma and in some forms of bronchitis contains a large number of eosinophile leucocytes.

(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, refractile, ovoid nucleus.

The columnar epithelium of the bronchial mucous membrane—both goblet and ciliated cells—is 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), the cytoplasm is granular, and each cell possesses 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 with blood pigment, and undergo fatty and myelin degeneration,—changes which the other varieties of epithelium 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 in many affections of the respiratory organs—in œdema, hypostatic congestion, hæmorrhagic infarction, pneumonia, and pulmonary tuberculosis. 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 granules of blood pigment, 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 sputum with an equal volume of

caustic soda solution for ten or fifteen minutes, until the mixture becomes thin and watery. The fluid is then centrifuged, and the deposit examined microscopically.

While the debris of lung tissue occurs 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,

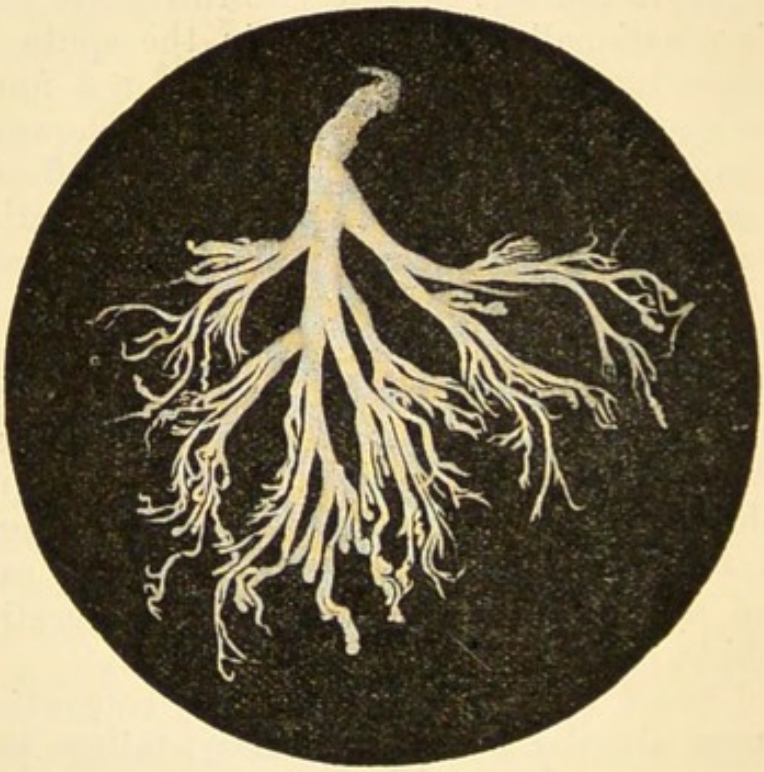


FIG. 102.—Bronchial cast, one-third of the natural size.

hence the great importance of this symptom in cases of phthisis where the physical signs are not distinct.

(4) FIBRINOUS BRONCHIAL CASTS.—Fibrinous casts (Fig. 102) of the bronchi or bronchioles may be found in the expectoration in fibrinous (plastic) bronchitis, acute lobar pneumonia, and very rarely in diphtheria. In the two former diseases the casts are solid; in diphtheria the cast has a hollow lumen. In the sputa of pneumonia and fibrinous bronchitis the white casts are rolled together, and only unroll and spread out when washed in water. The perfect way in which they reproduce the arrangement of the bronchi and bronchioles makes the recognition of these casts easy. In pneumonia the casts are usually derived only from the bronchioles, and are therefore small. They are most numerous in the

sputum on the third and fourth days of the disease, but they are of little clinical importance. More important are the large or small fibrinous casts in cases of fibrinous bronchitis. The terminal branches of the casts are of spiral form, and the sputum in that disease may also contain Curschmann's spirals (Fig. 103), Charcot-Leyden crystals (Fig. 105) and many eosinophile leucocytes.

In rare instances bronchial casts consist of mucus or blood.

(5) CURSCHMANN'S SPIRALS.—Spiral 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 twisted in spiral fashion around an axial or central fibrinous



FIG. 103.—Curschmann's spirals, in sputum, seen on black ground (after Curschmann).

thread. Embedded in the spiral are many eosinophile leucocytes. The spirals can often be detected with the naked eye (Fig. 103). They are formed in the bronchioles, and while asthma is the disease in which they are most frequently encountered, they are in no way characteristic of that affection, being often met with in bronchial catarrh arising from other causes.

(6) 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



FIG. 104.—Curschmann's spiral, magnified (Hartnack 4, Oc. 2) (Curschmann).

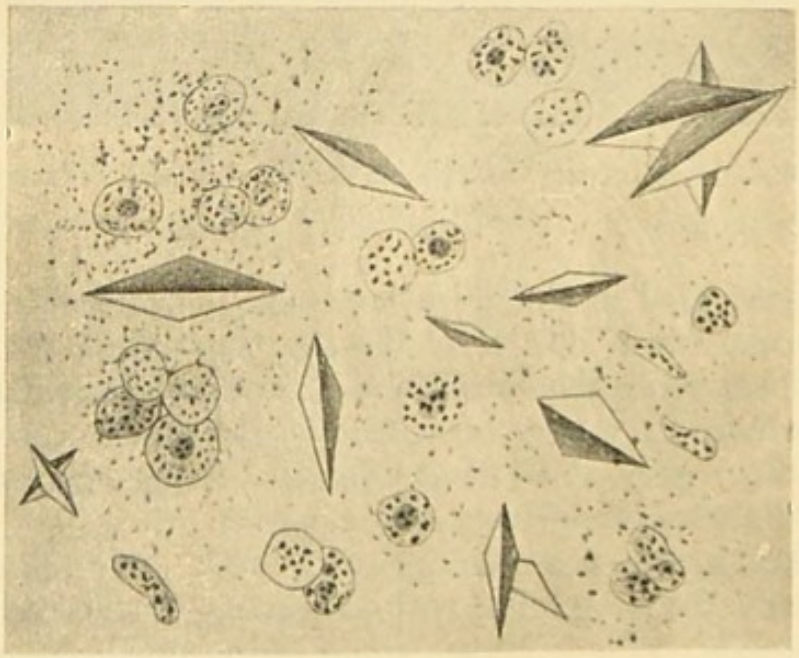


FIG. 105.—Charcot-Leyden crystals in sputum.

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, but are not, as was supposed, the exciting cause of the paroxysm.

Other crystals, such as cholesterin (Fig. 158, p. 341), hæmatoidin, leucin, and tyrosin (Fig. 157, p. 340), oxalates, and triple phosphate, occur in the sputum, but do not demand special notice here.

(7) Micro-organisms of various kinds may be found in the sputum. The methods of detecting micro-organisms in the expectoration and their significance are considered in Chap. XXXVI.

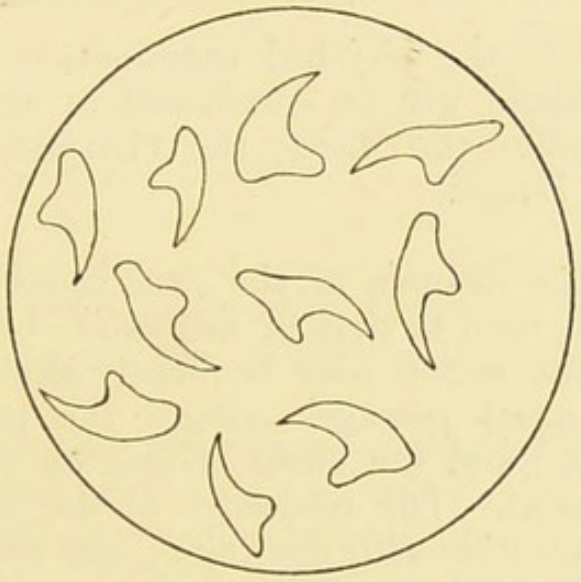


Fig. 106.—Echinococcus hooks, × 500.

(8) Animal Parasites.—When an echinococcus cyst, situated within the lung or invading it from the liver, ruptures into a bronchus, the sputum may be found to contain echinococcus scolices and their hooks (Fig. 106), brood capsules and portions of the cyst wall with its characteristic laminated ectocyst layer.

In Eastern Asia, and especially in Japan, the eggs of *Distoma pulmonale*—a trematode, parasitic in the lungs—may be found in large numbers in the blood-tinged expectoration.

(9) Other 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 XIII[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 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 adrenalin or cocain (5-10 per cent.) to the interior, which has the effect of contracting the mucous membrane, and thus giving a better view of the parts.

[1] Our thanks are due to Dr M'Kenzie Johnston, Consulting Surgeon to the Ear and Throat Department, Royal Infirmary of Edinburgh, for revision of certain parts of this chapter.

"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, etc., 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, etc.

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 transversus), 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 medulla.

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 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 metal 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 (Argand or incandescent burner) 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 close behind the patient's right shoulder, on a 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

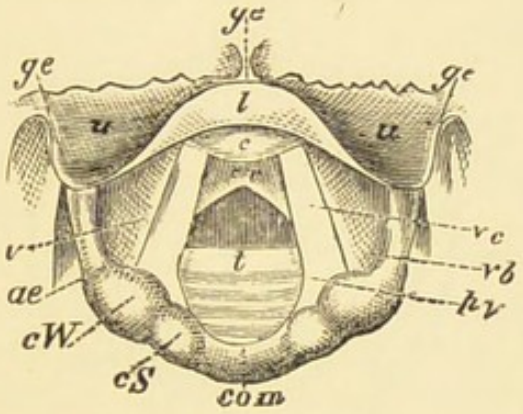


Fig. 107.—Diagram of laryngoscopic image in quiet inspiration (Morell Mackenzie).

(*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.
(*pv*) Processus vocalis.
(*cr*) Cricoid cartilage.
(*t*) Rings of trachea.

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, the surface of the mirror being kept horizontal. 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 metal rod of the mirror be pressed lightly outwards against the left 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 told 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. 107 and 108. 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

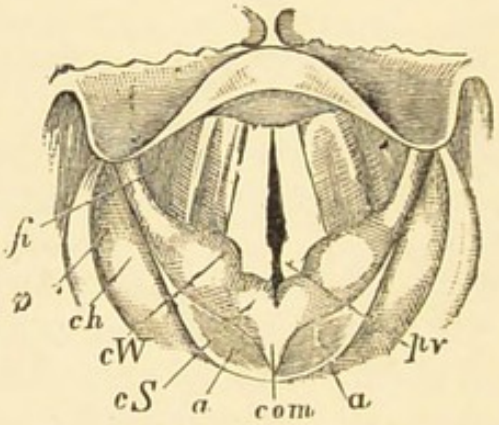


FIG. 108.—Diagram of laryngoscopic image in the act of vocalisation (Morell Mackenzie).

(*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.

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 per cent. solution), 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, etc., can generally be overcome by perseverance and practice. The laryngoscopic examination should be made methodically, 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 tuberculous 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) *Tumefaction.*—Swelling of 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, as from the sinus pyriformis.

(5) *Position and Movements of the Vocal Cords.*—The positions which the cords may assume are—(*a*) In quiet respiration (Fig. 107) 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. 108). (*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.

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, tabes dorsalis, syphilitic and other diseases of the brain. Most frequently 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.

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 œsophagus, etc., 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

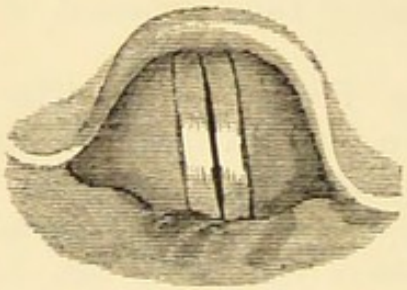


FIG. 109.—Unilateral recurrent paralysis—phonation (after Ziemssen).

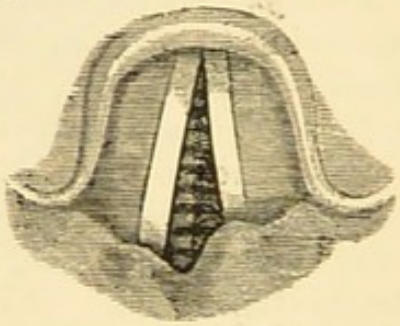


FIG. 110.—Unilateral recurrent paralysis—inspiration (after Ziemssen).

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.

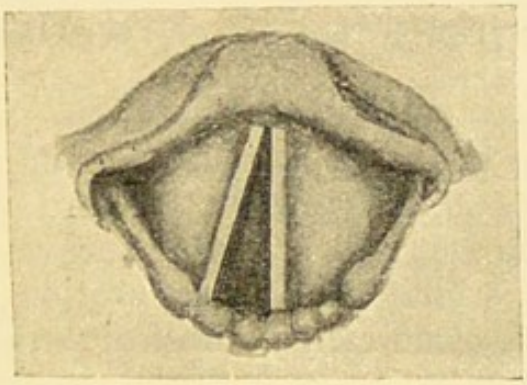


FIG. 111.—Abductor paralysis on left side—inspiration.

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.

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 aortic aneurism or intrathoracic tumour.

(2) ARYTENOIDEUS TRANSVERSUS 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.

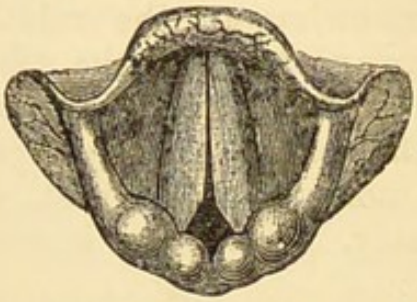


FIG. 112.—Paralysis of arytenoideus transversus muscle—phonation.

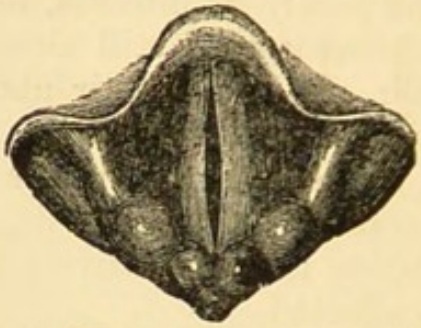


FIG. 113.—Paralysis of internal thyro-arytenoid muscle—phonation.

(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 bilateral, this excavation is of course found in both cords, and is often called elliptical paralysis.

CHAPTER XIV

Respiratory System (*continued*)

THE LUNGS

Topographical Anatomy. — The outlines of the lungs are considered on pp. 260, 261.

The Oblique Fissure of the lung is indicated by a line passing from the second dorsal spine to the sixth rib in the mammary line (Figs. 114 and 115). The transverse fissure of the right lung, demarcating the upper from the middle lobe, is represented by a line passing from the mid-point of the line representing the oblique fissure to the fourth right chondro-sternal junction (Fig. 114). The lower lobes are therefore represented to a very slight extent on the anterior surface of the thorax. When the arm is raised and the hand placed on the occiput, the vertebral border of the scapula corresponds, according to Cunningham, with the oblique fissure, and marks the upper limit of the lower lobe.

The Trachea bifurcates at a point corresponding posteriorly to the interval between the third and fourth dorsal spines, and anteriorly to the junction of the manubrium and the body of the sternum.

The Pleura.—The line of reflection of the right pleura anteriorly corresponds with the anterior margin of the right lung. On the left side the line of reflection of the pleura below the fourth costal cartilage is considerably nearer the middle line than is the margin of the left lung. The pleura extends downwards on each side below the lower margin of the lung, thus constituting the complementary pleural sinus. This extends down to the level of the seventh costal cartilage in the mammary line, the tenth rib in the mid-axillary line (its lowest point), and the upper border of the twelfth dorsal spine at the vertebral column (see Figs. 114 and 115).

Regions of the Thorax.—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.

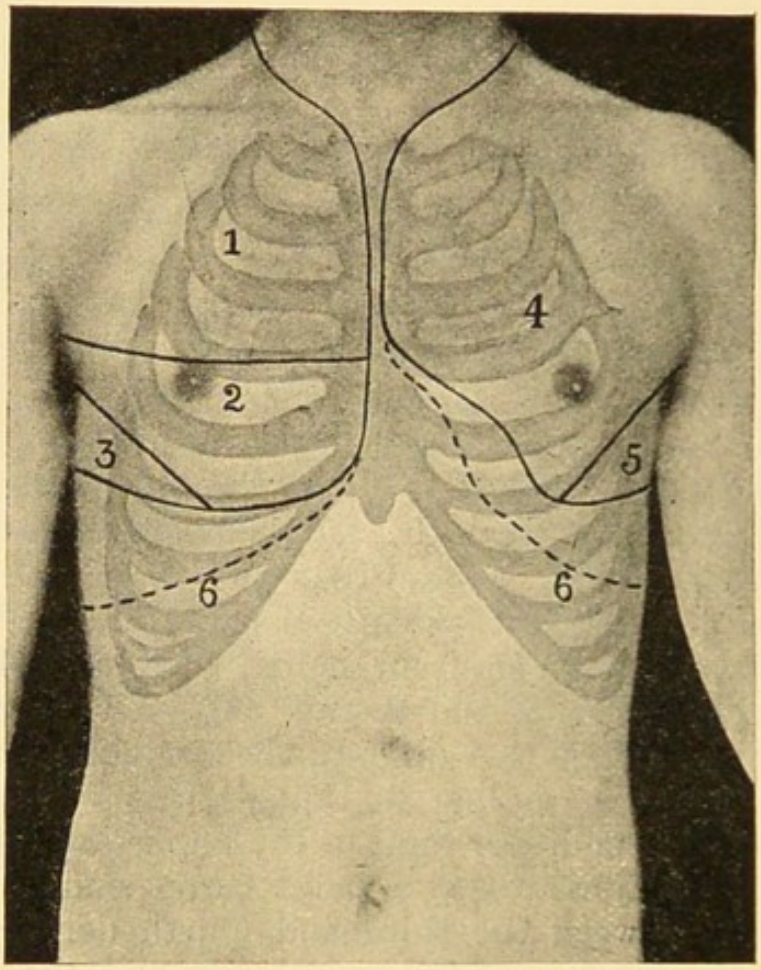


FIG. 114.—Percussion outlines of the lungs anteriorly. The continuous black lines indicate the percussion outlines of the lungs and the pulmonary fissures. The dotted lines (6) show the lines of reflection of the pleuræ. The figures 1, 2, 3 indicate the upper, middle, and lower lobes of the right lung ; 4 and 5 the upper and lower lobes of the left lung.

(*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.

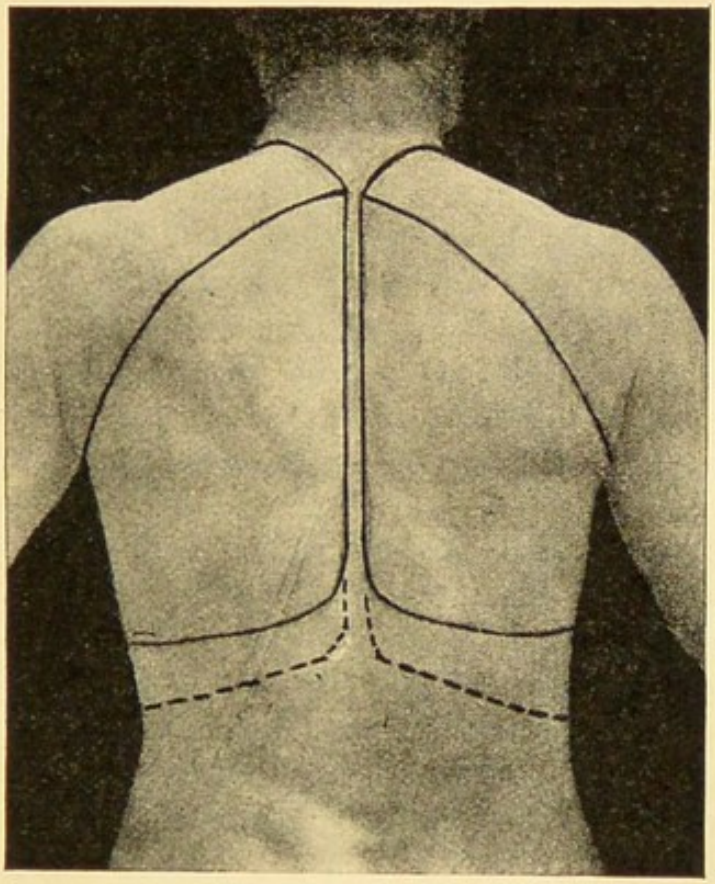


FIG. 115.—Percussion outlines of the lungs posteriorly. The continuous black lines indicate the percussion outlines of the lungs and the oblique fissure on either side. The dotted lines show the line of reflection of the pleuræ.

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 a line passing vertically downwards from the posterior fold of the axilla. 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 posterior fold of

the axilla, 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 OF THE THORAX

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 of the chest, and (2) the respiratory movements.

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 flattened. 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 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 is that associated with a tendency to phthisis. 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.

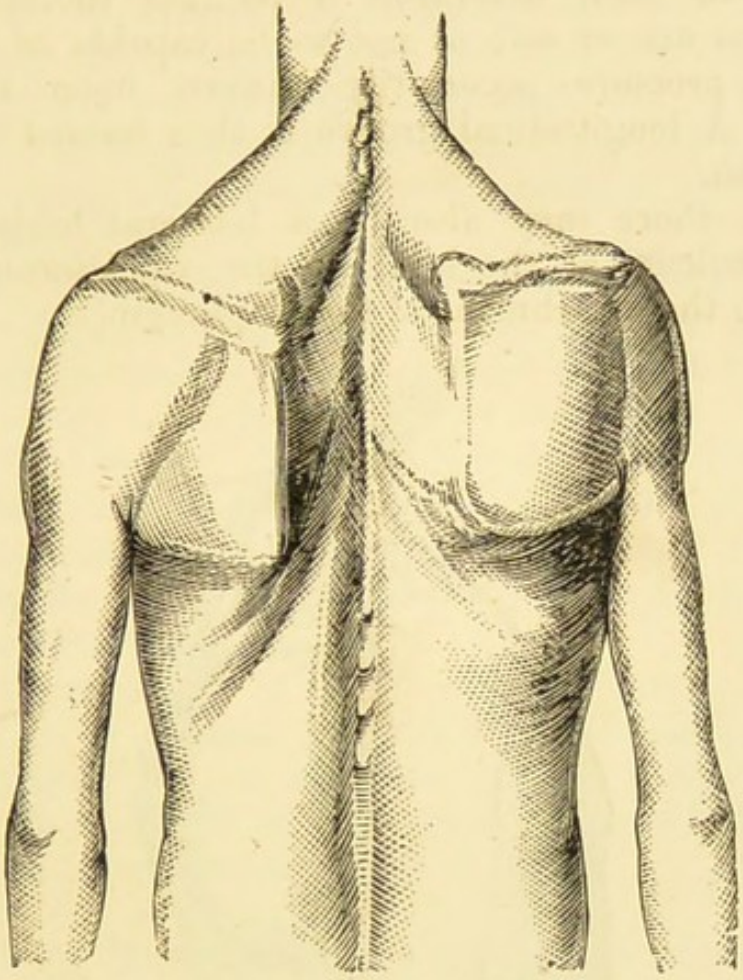


FIG. 116.—Alar chest (R. Thompson).

The occurrence of any obstruction to the respiration in childhood, along with rickets, tends to produce the "**pigeon-breast**,"

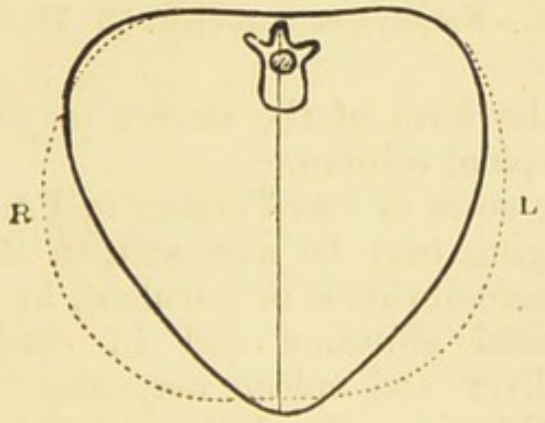


FIG. 117.—Pigeon-breast. A reduced tracing taken from a child of seven years. Dotted line indicates natural shape at same age. (After Gee.)

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, 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.

In infants there may also be a bilateral horizontal groove (**Harrison's sulcus**) at the level of the xiphisternum and corresponding to the attachment of the diaphragm.

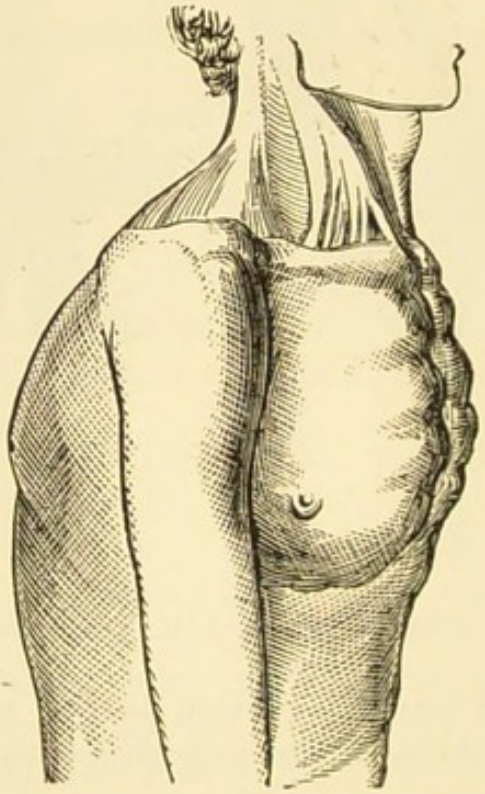


FIG. 118.—Emphysematous chest (R. Thompson).

Irregularity in the form of the thorax may also be caused by deformities of the spinal column.

CHANGES IN THE FORM OF THE THORAX IN PULMONARY DISEASES. —(1) **Local.**—Bulging may be met with in encapsuled pleural effusions, whether sero-fibrinous or purulent, in localised pneumothorax, in pericardial effusions and in cardiac hypertrophy. 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 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 fibrosis and as a result of continued pressure on the main bronchus, as by an aortic aneurism.

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, (6) the degree of excursion of the margins of the lungs.

(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, and is slightly more frequent in the female. It is increased in frequency 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 normally 1 : 4, but may vary in disease 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 (see p. 230).

(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. The relative length of inspiration to that of expiration is in the ratio of 5 : 6. 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 (Fig. 128), 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

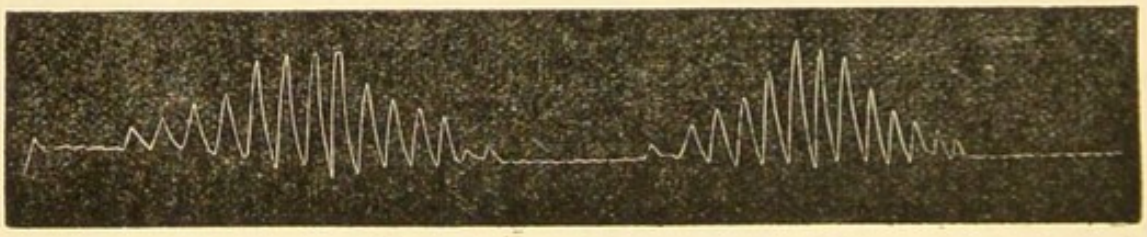


Fig. 119.—Respiratory tracing of Cheyne-Stokes respiration (after Weintraud).

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** (Fig. 119), 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 one-quarter to three-quarters 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 still quite obscure. Cheyne-Stokes breathing is met with in uræmia, in many cerebral diseases, in myocardial degeneration (attended, perhaps, with arterio-sclerosis),

and 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* or *abdomino-thoracic* 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* or *thoracic* type). In disease, however, the type of breathing may be changed, for anything which interferes with the movements of the diaphragm (such as paralysis of the diaphragm, ascites, peritonitis, and many other affections of the abdomen) will in a man change the type of breathing into the purely thoracic; whilst the latter type of breathing may be lost in a woman when there is some painful thoracic affection which obliges the respiration to be chiefly abdominal. The breathing is of the abdominal type when the intercostal muscles are paralysed.

(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 pulmonary 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 frequently 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. Tuberculous consolidation at the apices gives rise to deficient movement in the upper part of the chest, as compared with the lower; whereas, 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.

(6) The Degree of Excursion of the Margins of the Lungs may be determined by means of **Litten's Diaphragm Phenomenon.** To study this phenomenon, the patient bares his chest and lies on his back, with his feet towards the window. The couch is placed so that the light falls at an angle of about 45° on to the side of the patient's chest, as shown in Fig. 120. If the room be lit by other windows, they must be darkened; or all the windows may be darkened and a lamp placed near the foot of the couch. The observer, standing 3 or 4 feet from the patient's side, and with his back towards the source of light, inspects the lower part of

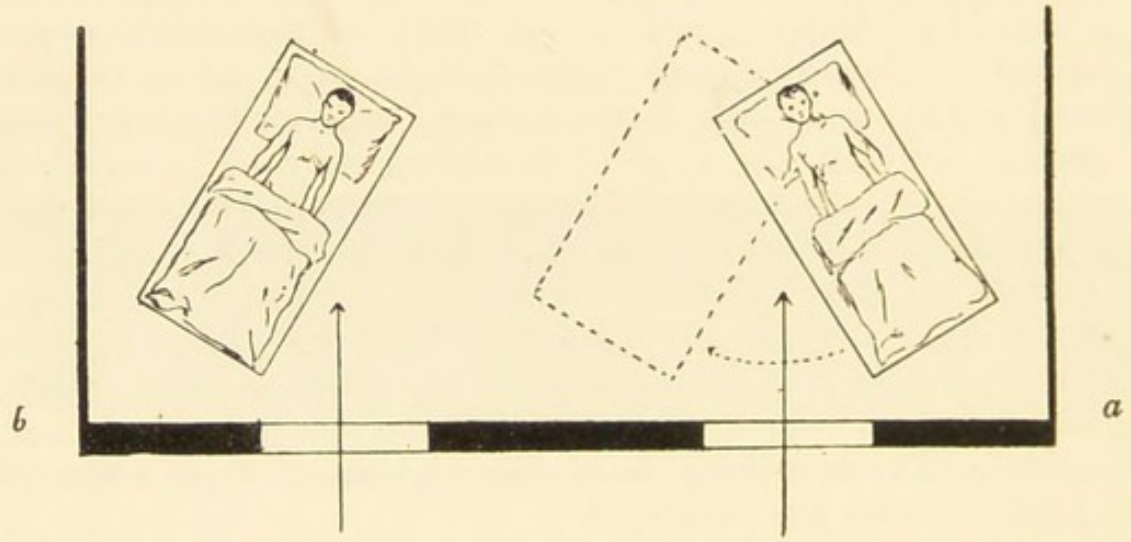


Fig. 120.—Litten's diaphragm phenomenon. *a*, the position of the couch in relation to the window when the right side of the thorax is examined; *b*, when the left side is examined (after Zabel).

the chest. When the patient takes a deep inspiration, Litten's phenomenon is observed, namely a narrow linear shadow around the thorax, and descending on deep inspiration from the level of the sixth or seventh rib to that of the ninth or tenth rib, and rising again to its former level on expiration. The degree of excursion of the shadow is normally about 6 or 7 cm.

The shadow is best seen between the anterior axillary and mammary lines. It has been ascribed to indrawing of intercostal spaces by the suction power of the diaphragm when it contracts. But it is more probable that on inspiration the intercostal spaces from above downwards are first depressed (because of diminished pressure within the thorax), and then bulge outwards (because of increased intra-abdominal pressure). The difference in the level of the thoracic surface, thus caused, gives rise to the shadow.

Litten's phenomenon can almost always be observed in normal persons, with the exception of those who are unduly stout. The phenomenon is not seen when there is effusion into the pleura, adhesions at the lower part of the pleural sac, consolidation of the lung involving its lower border, and advanced pulmonary emphysema.

The degree of excursion of the pulmonary margin should be confirmed by means of percussion, as described on page 261.

CHAPTER XV

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 shows 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 shows 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, acts as a stimulant on the respiratory centre, 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 oxygenate 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, trapezei, and pectorals, 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 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.

When 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

[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.

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 p. 223), is a common cause of certain of the varieties of deformity 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** shows 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 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 uræmia and in the "air hunger" of diabetic coma. 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 pulmonary emphysema, and in passive pulmonary congestion, resulting from heart disease, where the great engorgement of the pulmonary capillaries, and the consequent limitation of movement, may of itself give rise 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 shown to be the case in severe typhus, be of considerable moment. In cases of extreme nervous exhaustion the flapping of the alæ nasi, from loss of tone in their muscles, constitutes an impediment to respiration.

In the **pharynx**, the soft palate may strike against the posterior wall of the pharynx (Nasal Stertor), or the tongue may fall back on the palate, as in snoring, and cause Oral Stertor, 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 œdema 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 (see p. 217).

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 mediastinal tumour. An aortic aneurism, for example, may press on the trachea near its bifurcation, giving rise, both on inspiration and expiration, to tracheal stridor, the "leopard growl" as it is termed by Wyllie.

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 fibrinous bronchitis and in bronchial asthma, due in the latter disease, as Roy and the author showed, to spasm of the smooth muscular fibres in the walls of the bronchi. The obstruction in asthma is chiefly expiratory.

(4) **Dyspnœa from Diminished Alveolar Area.**—All forms of exudation into the alveoli of the lungs, whether passive, as in pulmonary œdema, 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 intrathoracic 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 respirations 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 Pulmonary Circulation.**—Though a normal quantity of oxygen is reaching the alveoli, the circulation of blood in the pulmonary capillaries may be so impaired 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 mitral valve, are apt to show this symptom. But, in many of these cases, not only is there present a more or less constant dyspnœa, but paroxysms of great breathlessness occasionally occur, which, from their resemblance to bronchial asthma, are usually known as "cardiac asthma." They generally come on during the night, or on physical exertion, and are mainly due to the defective reserve power of the heart. It is possible, however, that these attacks may be partly due to that spasmodic contraction of the bronchi which Roy and the author found to be produced, to a very marked extent, by defective aeration of the blood.

It should also be remembered that an embolus 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 defective formation), or are themselves deficient in hæmoglobin and consequently manifest impairment of power to take up and carry oxygen. The latter condition is well seen in cases of chlorosis and in leucocythæmia, 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 XVI

Respiratory System (*continued*)

PALPATION OF THE THORAX

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, both on tranquil and forced respiration. The hands must be laid symmetrically over corresponding parts of the two sides of the chest.

We first palpate the apices; and to do so we stand behind the patient and place each hand so that the fingers rest on the clavicle, the thumb on the supra-scapular region. Then, standing in front of the patient, we lay the palm of each hand over the infra-clavicular region, while the fingers lie over the clavicle and supra-clavicular region. The movements in the mammary, infra-mammary, and axillary regions having been successively ascertained, we grasp the chest from behind, pressing the fingers firmly over the axillary regions while the two thumbs meet over the vertebral column. When the patient takes a deep breath we estimate the extent to which the points of the thumbs are moved from the middle line.

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.

The intensity of the vocal fremitus at any part of the chest is ascertained by applying the palm of the hand firmly to the part and directing the patient to say with a loud voice "ninety-nine" or "one, one." The same hand is employed throughout, and the intensity of the fremitus is compared at corresponding regions on the two sides of the chest.

The thickness of the thoracic wall has an important influence on the intensity of the thrill, the fremitus 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. The fluid in the pleural cavity is a good conductor of the vocal fremitus, but when the effusion is extensive the bronchi are compressed, and the vocal fremitus is consequently diminished or may be entirely lost. 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, causing defective expansion of the lung, 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 a better conductor of the vocal vibrations, and, in consequence, the thrill becomes intensified. Such is the case, for example, in lobar pneumonia; and when 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 palpating 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 empyema. In this latter condition there is often some inflammatory œdema in the chest wall.

CHAPTER XVII

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 developments 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 32 or 33 inches, varying, however, considerably in different individuals. A full inspiration increases the figure by from 2 to 5 inches, while in quiet respiration the inspiratory measurement exceeds the expiratory 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.**—A simple cyrtometer may be constructed of two pieces of lead wire, joined together by means of a piece of india-rubber tubing. The apparatus, when closely applied around the thorax at the level of the nipples, and with the india-rubber tube over the spinal column, retains the curves given to it; and when it is removed and laid on a large sheet of paper, these curves can be marked out on the paper. A permanent record of the form of the chest in a horizontal plane is thus obtained.

4. **Perigraph.**—The author has devised this instrument for the purpose of obtaining tracings of the outline of the thorax more reliable than those the cyrotometer affords, and also tracings taken in planes where the cyrotometer 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. 121, 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 modification is necessary in order to enable the point C to move round a more or less cylindrical surface.

The modified instrument is shown in Fig. 122. 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

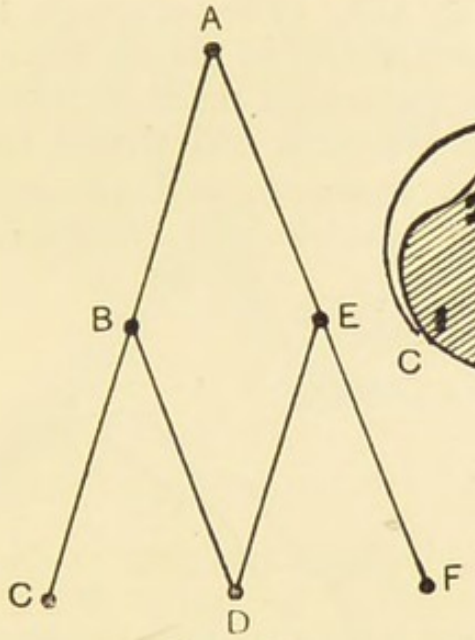


FIG. 121.

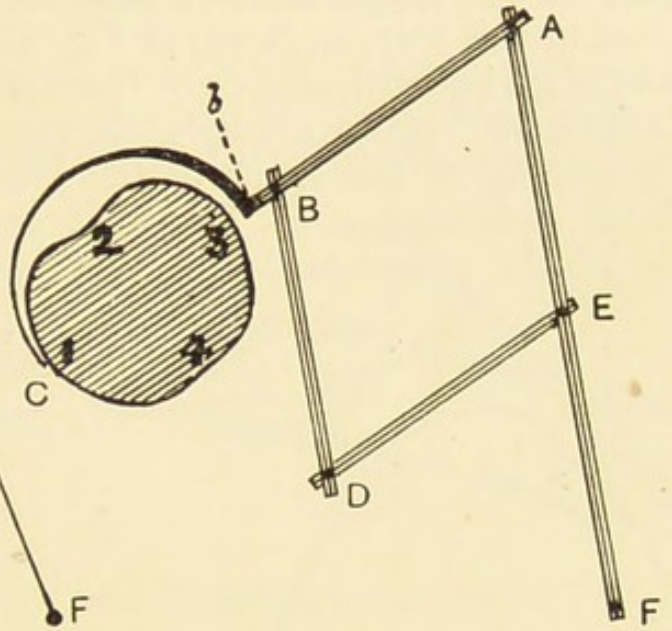


FIG. 122.

C is made to move round the thorax, the pencil at D will trace the chest form.

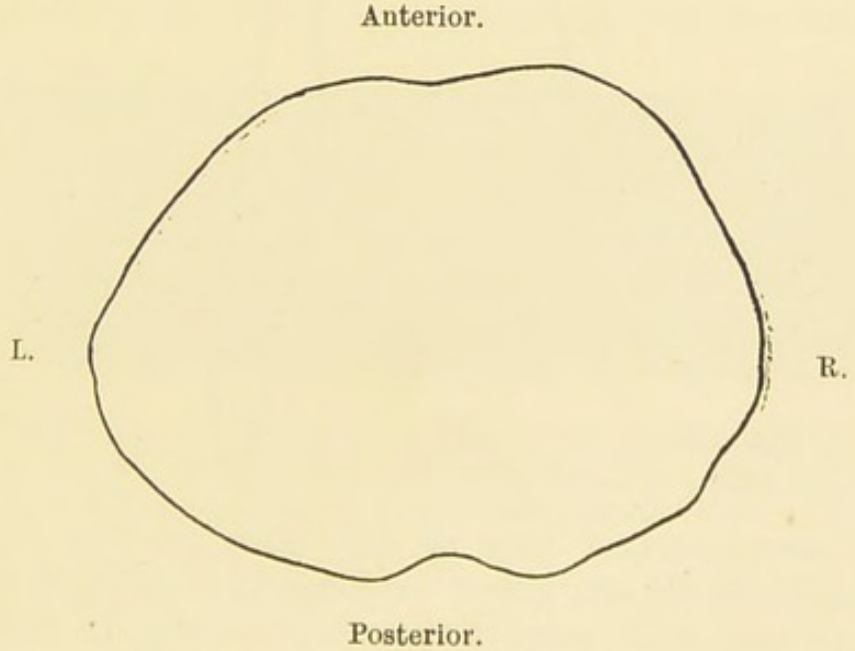


FIG. 123.—Outline of thorax.

Fig. 122 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° 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. A reduced diagram of the shape of the thorax in a very muscular subject, obtained in this way, is shown in Fig. 123. The perigraph is also capable of giving outlines of the chest shape, in planes other than the horizontal. 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

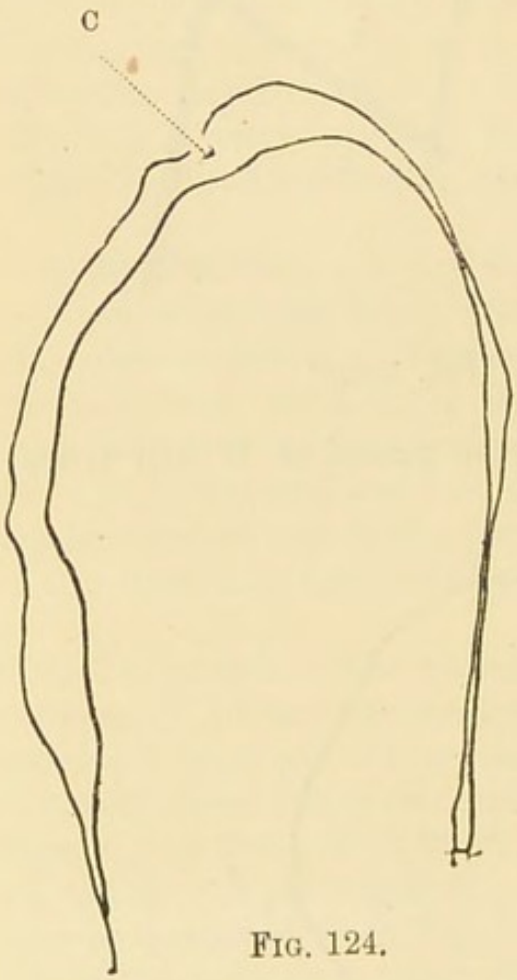


FIG. 124.

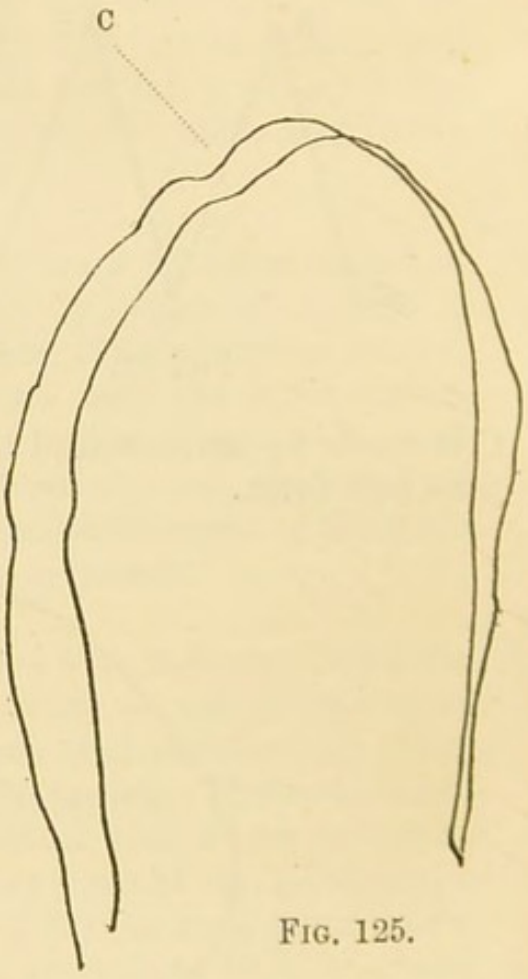


FIG. 125.

down the corresponding line of the back to the crest of the ilium. Such curves are given in Figs. 124 and 125, 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 is of interest to note the relative 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.

Pneumograph.—Marey's pneumograph is arranged so as to be tied around the chest. The instrument (Fig. 126) consists of a flexible steel plate which bends when the two side-arms are drawn

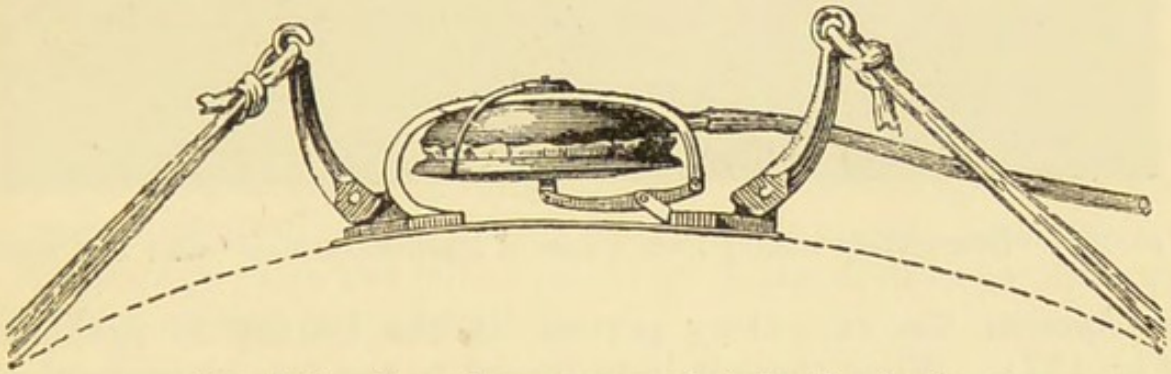


FIG. 126.—Marey's pneumograph (after Luciani).

apart on inspiration. The movement is transmitted by a lever from the steel plate to a tambour, and is conducted thence by a piece of tubing to a Marey's recording tambour, the lever of which writes on a smoked paper. Inspiration is represented by the descending line of the tracing, expiration by the ascending line.

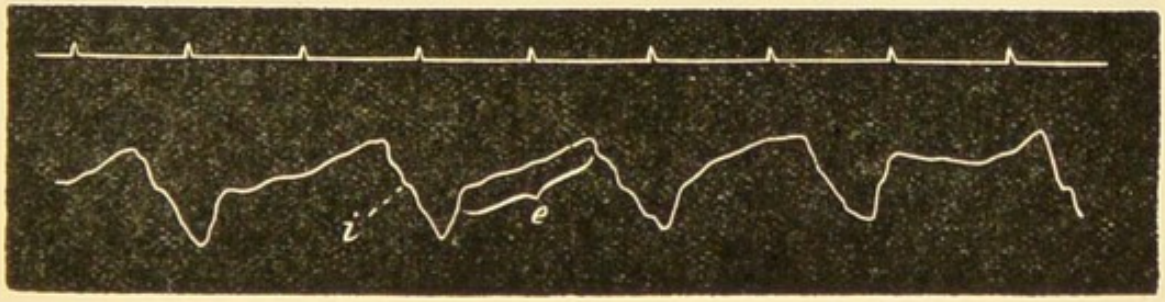


FIG. 127.—Respiratory tracing from a case of bronchial asthma (after Hofbauer). In this and the three following figures *i* marks the inspiration, *e* the expiration ; the time is recorded in seconds.

Or we may simply apply the "receiver" of Mackenzie's polygraph to the supra-clavicular fossa. Inspiration is again represented by the descending line of the tracing.

Or an india-rubber bag, about three inches long, and connected at one point with a piece of tubing, and thus with a Marey's tambour, may be strapped to the thorax. Inspiration is then represented by the ascending line of the tracing, expiration by the descending line.

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. The most striking changes are seen in the following conditions:—In *bronchial asthma* and in

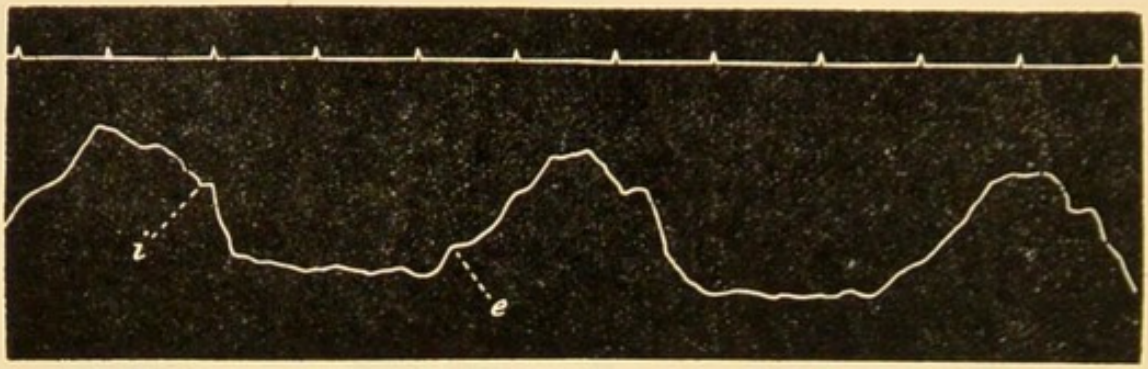


FIG. 128.—Respiratory tracing from a case of tracheal stenosis (after Hofbauer).

emphysema the expiratory portion of the tracing is prolonged (Fig. 127). When there is impediment to the free entrance of air, as from *stenosis of the larynx* or *trachea*, the inspiratory portion of

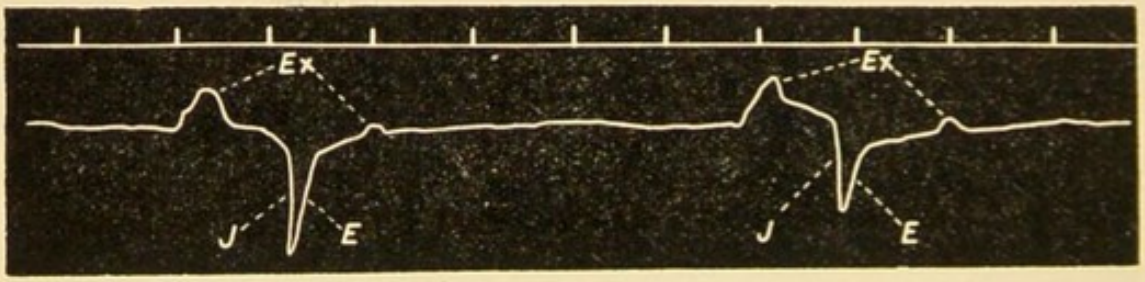


FIG. 129.—Respiratory tracing from a case of uræmic asthma (after Hofbauer).

the tracing is much prolonged (Fig. 128). In the *uræmic asthma* of uræmic coma (Fig. 129) the respiratory rate is infrequent, and there is an "extra-expiration" at the end of the expiratory portion

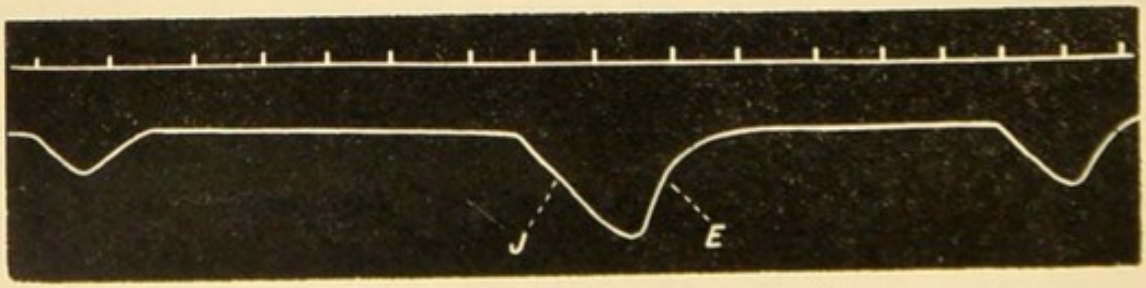


FIG. 130.—Respiratory tracing from a case of diabetic asthma (after Hofbauer).

of the tracing (Fig. 129, Ex.). In *diabetic coma* the respirations are infrequent, slow, and deep (Fig. 130). The characters of *Cheyne-Stokes* respirations are shown in Fig. 119, p. 226.

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.

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 when 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.

CHAPTER XVIII

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 plexímeter employed, and still more when the plexímeter 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 pulmonary 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 consolidated, 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 keyboard 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 we have already said, the length of some at least of these columns is determined by limiting pulmonary 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 XIX

Respiratory System (*continued*)

PERCUSSION OF THE THORAX

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.

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 almost 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 the middle finger of the right hand to give the stroke, and to employ the middle phalanx of 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 and metacarpo-phalangeal joint alone, and not from the shoulder or elbow, and the finger 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 we have indicated the theoretical basis on which we believe the practice of percussion may 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 Pulmonary 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, etc.); 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

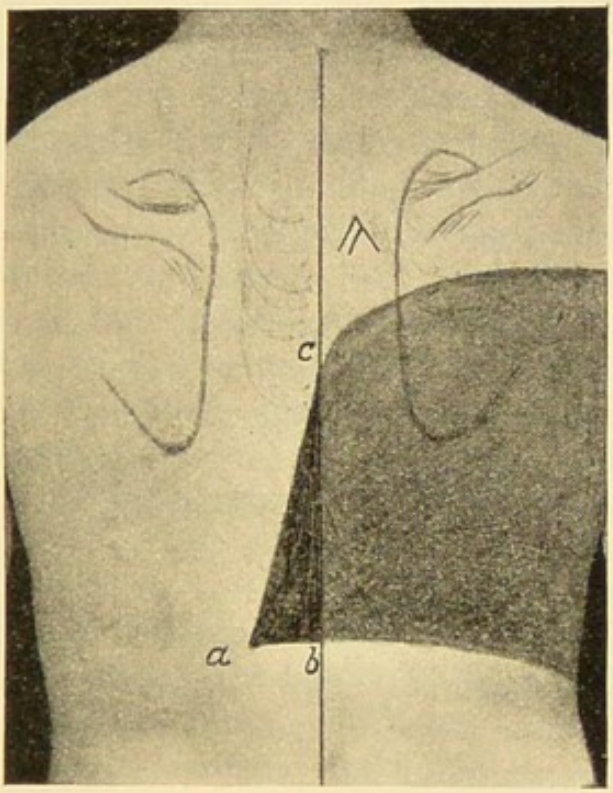


FIG. 131.—Dulness in right-sided pleural effusion. In cases of pleural effusion a triangular area of dulness (Grocco's paravertebral triangle, *a*, *b*, *c*) can frequently be detected on the opposite side of the chest posteriorly.

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. The adhesions also interfere with the proper expansion, and consequently with the resonance of the lung.

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 cases of chronic pulmonary 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 note depends 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, we have sometimes seen emphysema mistaken by the in-

experienced for pulmonary consolidation, owing to the heightened pitch.

2. The State of Tension of the Pulmonary 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 (see p. 257).

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 various stages 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 right mammary 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 pulmonary 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 (*Skodaic resonance*) 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 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 dependent 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.

[1] When the effusion is excessive, the lung tissue becomes compressed, and the tympanitic quality of the note is consequently lost.

3. Pulmonary Consolidation, allowing the Broncho-Tracheal Air Column to be set in Vibration.—Allusion has been already made 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 is the

Cracked-Pot Sound.—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 schoolboys, 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 we 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.

[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.

The last change in quality which we shall consider is

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 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 pleximeter, 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. The sounds simulate those of a hammer striking an anvil.

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, etc.), 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 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. When determining the outlines of the lungs by percussion, we must percuss at right angles towards the margin we wish to define, and the finger on which we percuss must be kept parallel to that margin. When percussing on the thorax, the plexímeter finger should lie along a rib or interspace, not across the ribs.

The Apices.—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. Percuss firmly downwards, from the level of the angle of the jaw, along the anterior border of the trapezius. In health the apex rises on each side to a point about $1\frac{1}{4}$ to 2 inches (3 to 5 centimetres) above the clavicle, being perhaps a trifle higher on the right side. The highest point of the apex corresponds posteriorly to the spine of the seventh cervical vertebra.

As noted years ago by Gee, the excursions of the aspices during respiration, as detected by percussion, are of some diagnostic value. Shrinking of the apices, both vertically and transversely, is one of the early physical signs of phthisis, and is therefore of great importance. In pulmonary emphysema, the limits above given may be overstepped to a considerable extent.

The Anterior Margin of the Right Lung is represented by a line (Fig. 132) which passes downwards from the apex along the clavicular portion of the sternomastoid muscle, passes through the right sternoclavicular articulation, reaches nearly to the middle line at the level of the first intercostal space, and then passes vertically downwards to the level of the sixth chondro-sternal junction. The portion of this border lying behind the sternum cannot be defined by percussion. The line representing the lower border of the right lung commences at the level of the sixth right chondro-sternal junction and passes outwards, being on tranquil respiration, at the level of the sixth rib in the right parasternal and mammary lines, at the eighth rib in the mid-axillary line, at the tenth rib in the scapular line, and at the eleventh rib at the vertebral column (see Fig. 115, p. 221).

The Anterior Margin of the Left Lung, as far down as the fourth left chondro-sternal junction, corresponds to that of the right lung (see Fig. 132). At this point the margin of the left lung curves outwards to the sixth rib midway between the left parasternal and mammary lines, thus forming the left border of the superficial cardiac dulness. The lower border of the lung

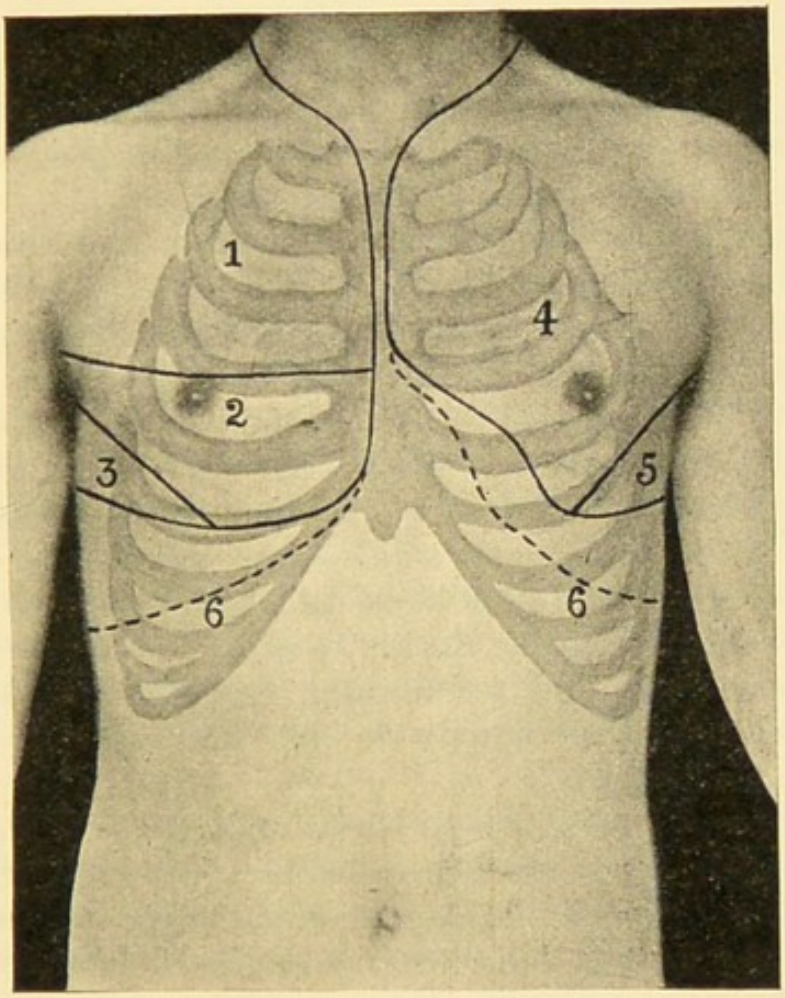


Fig. 132.—Percussion outlines of the lungs anteriorly. The continuous lines indicate the percussion outlines and fissures of the lungs. The dotted lines, 6, show the lines of reflection of the pleuræ ; 1, the upper lobe ; 2, the middle lobe ; 3, the lower lobe of the right lung ; 4, the upper lobe ; 5, the lower lobe of the left lung.

then passes outwards and backwards along a line corresponding to the lower border of the right lung.

The Inferior Margins should be ascertained by percussing very lightly from below upwards, during tranquil respiration and subsequently on full inspiration.

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. Defective movement on inspiration may also be due to chronic pleural adhesions.

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 (see p. 147).

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 (upper 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 may be 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 XX

Respiratory System (*continued*)

AUSCULTATION OF THE LUNGS

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 simple wooden stethoscope answers admirably for all ordinary cases, although sometimes, and especially in infants, a binaural stethoscope 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 physician, 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 physician 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.

When auscultating the chest at any point, we have to note—

1. The character of the breath sounds.

2. The presence or absence of accompaniments—adventitious sounds of various kinds.

3. The character of the vocal resonance (auscultation of the voice).

We auscultate first while the patient is breathing tranquilly, and then, after he has been made to cough and while he is breathing deeply. Accompaniments may now be audible which were not detected during tranquil respiration. Finally, to test the vocal resonance, we auscultate while the patient says with a loud voice "ninety-nine" or "one, one."

Breath Sounds.—The Laryngeal, Tracheal, or Bronchial Murmur.—If we auscultate over the larynx, trachea, or spines of the lower cervical vertebræ, we hear two blowing sounds, one synchronous with inspiration, the other with expiration. The double murmur may be phonetically represented by arranging the position of the mouth and tongue to utter the gutteral "Ch" and then breathing quietly in and out. The expiratory portion of the murmur is about as long as the inspiratory, and between the end of the inspiratory and the commencement of the expiratory murmur there is a short but quite appreciable pause.

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 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 that the respiratory murmur audible in healthy persons over the trachea and lower cervical vertebræ is produced.

The Vesicular Murmur.—On auscultation of the healthy chest, the blowing breath sounds produced at the glottis are nowhere audible, as they are not well conducted through the lung. What we do hear is 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 or four times as long as the second, and is softer and higher in pitch. There is no pause between the termination of the inspiratory murmur and the commencement of the expiratory murmur. The expiratory murmur is not unfrequently inaudible in healthy persons.

The vesicular murmur arises as the air streams through the bronchioles and infundibular passages into the pulmonary alveoli.[1] Whenever the vesicular breath sounds are heard, the pulmonary alveoli are fulfilling their function, and when it is absent, 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 (except in the interscapular region), 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.

Broncho-vesicular Breath Sounds. — In the inter-scapular region the large bronchi lie comparatively near the chest wall, and on auscultating that region of the healthy chest we hear a combination of the blowing murmur, produced at the glottis, and the vesicular murmur originating in the lung tissue overlying the large bronchi at the root of the lungs. The inspiratory murmur may be vesicular, and the expiratory murmur be of blowing character, or *vice versâ*, and there is no distinct pause at the end of the inspiratory murmur.

Modifications of the Vesicular Murmur in Disease.—From various causes the vesicular murmur may be absent. Thus it may be replaced by bronchial breath sounds, or it may be inaudible owing to the loudness of superadded sounds, but apart from these two conditions the common modifications of vesicular breath sounds 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** breath sound depends in part upon the thinness of the chest walls and the greater elasticity of the lung tissue.

[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.

In adults harsh vesicular breath sounds occur when, part of the respiratory surface having been thrown out of action by disease, the healthy portion is receiving a more than usually liberal supply of air. Over this healthy portion the breath sounds become of harsh vesicular character. In cases of pneumonia, pleural effusion, and many other conditions, harsh vesicular breath sounds may be heard over the unaffected parts of the lungs. The breath sounds may also be of harsh vesicular character when there is catarrh of the smaller bronchial tubes. The harshness in this instance results from the swelling of the bronchial mucous membrane, and the consequent increase of friction as the air passes into and out 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 all over the 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



FIG. 133.—Diagram of various types of vesicular breath sounds (after Wyllie).

which differs from that just referred to in two particulars, viz.: (*a*) it does not disappear with deep inspiration; and (*b*) 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 the expiratory murmur. When the expiratory murmur exceeds this length, we may conclude that the lung tissue has lost its elasticity (as in emphysema or early phthisis), or that there is some obstruction to the escape of air, as in bronchial asthma. 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 and adhesion of the pleural surfaces, or pleural effusion of limited amount, may also produce weakening of the vesicular murmur, because of the defective inspiratory expansion of the lung.

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.

The Bronchial Murmur in Disease.—As has been already said (p. 264), bronchial breath sounds, although audible over the



FIG. 134.—Diagram of bronchial breath sounds (after Wyllie).

larynx and trachea, cannot be heard over the chest generally, because inflated lung tissue is a very bad conductor of sound.

But in disease, bronchial breath sounds may be audible over the chest. According to the pitch of the sounds, we have to distinguish (*a*) **high-pitched** bronchial breath sounds (sometimes termed tubular breath sounds), which are phonetically represented by whispering *ich*, *ich*[1]; (*b*) **medium-pitched** bronchial breath sounds, which may be imitated by whispering *ach*, *ach*; and (*c*) **low-pitched** bronchial breath sounds (or **cavernous** breath sounds), which are imitated by whispering *och*, *och*.

Bronchial breath sounds become audible under two varieties of pathological conditions as follows:—

(1) WHEN THE LUNG TISSUE BECOMES CONSOLIDATED—provided that the consolidation 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 consolidation, and the bronchial murmur is conducted to the surface and becomes audible. These conditions are fulfilled in the case of acute pneu-

[1] These sounds are to be pronounced in the German fashion.

monia (stage of hepatisation), and in all the varieties of chronic phthisis. Bronchial breath sounds are therefore heard over hepatised lung, and whenever tuberculous consolidation is of considerable extent. They also occur 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. High-pitched bronchial

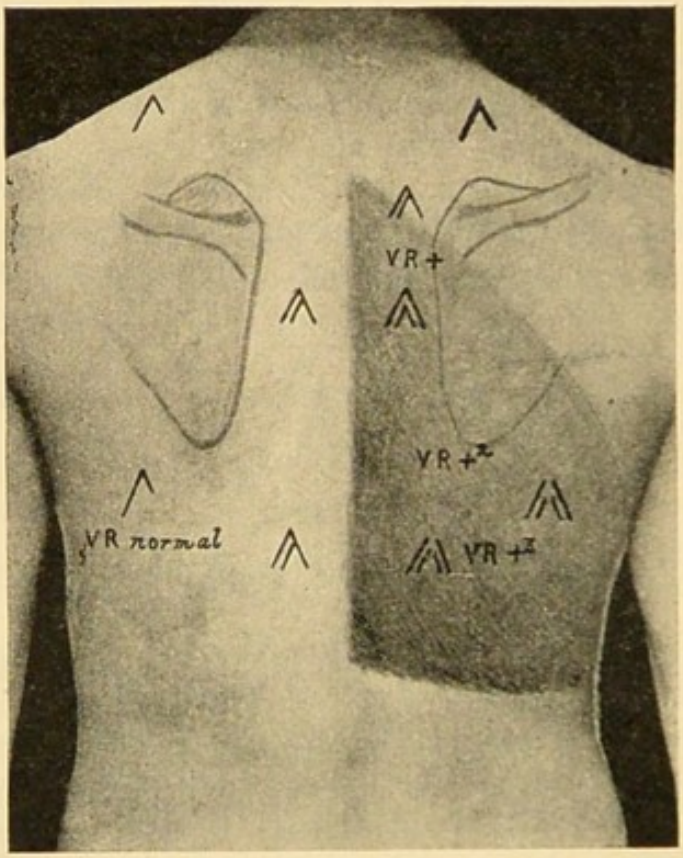


FIG. 135.—Acute lobar pneumonia of the right lower lobe. The shaded area indicates the dulness.

breath sounds are most characteristically heard over the consolidated lung in acute lobar pneumonia.

(2) IN PULMONARY CAVITIES.—Bronchial breath sounds of the low-pitched variety (cavernous breath sounds) may be heard over vomicæ, provided that they are superficial, have smooth walls, are surrounded by consolidated tissue, and freely communicate by means of a bronchus with the air in the trachea. These breath sounds are, therefore, heard over pulmonary cavities and over the dilated bronchi of bronchiectasis. 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.

Amphoric Breath Sounds.—The peculiar character of amphoric breath sounds is well reproduced by blowing into an empty jar or bottle, and their mode of origin is similar to that of the sound so obtained. Amphoric breath sounds occur under two pathological conditions—(1) pulmonary excavation, and (2) pneumothorax, as follows :—

(1) Pulmonary Excavation.—In order that amphoric breath sounds 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.

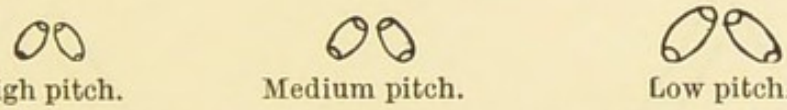


Fig. 136.—Diagram of amphoric breath sounds (after Wyllie).

(2) Pneumothorax.—When air escapes into the pleural sac and distends it, the lung becomes collapsed; and if the pressure be sufficient not only to drive air out of the alveoli, but also to cause collapse of the bronchi, no amphoric breath sounds are audible, nor are they audible if the fistula by which the air has entered the pleural cavity becomes closed.

But if the bronchi remain patent and in free communication through the fistula with the air in the pleural cavity, the bronchial breath sounds 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, etc.), 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.

Broncho-vesicular Breath Sounds.—As has been said on page 265, this murmur may be audible in healthy persons when we auscultate near 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 occasionally 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. It is then an indication of pulmonary consolidation or collapse, which is either incomplete or of very limited extent, so that although the respiratory murmur produced at the glottis is in part conducted to the parieties, it is mingled with a vesicular murmur.

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 stage of their course, there become audible certain abnormal, adventitious sounds. Inasmuch as certain of these 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

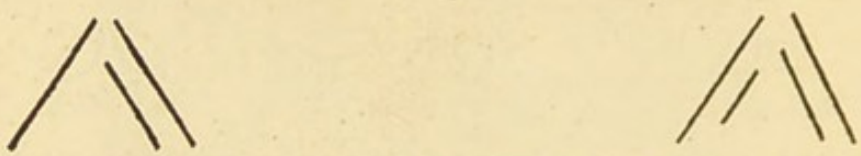


Fig. 137.—Diagram of broncho-vesicular or "indeterminate" breath sounds (after Wyllie).

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.

Physicians differ much in regard to the nomenclature of these adventitious sounds, and as a rule they have been too minutely subdivided. For practical purposes the following classification suffices :—

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.

(1) **Moist Râles.**—(*a*) 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 imitated by firmly pressing the moistened thumb against the forefinger, and then suddenly separating the two surfaces, close to the ear. Fine crepitation may also be imitated by the sound produced on rolling a good cigar between the finger and thumb, or on slightly compressing and relaxing an india-rubber sponge.

Although fine crepitation is probably sometimes due to the bursting of fine bubbles in the very smallest bronchioles, it commonly arises during inspiration 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



Fig. 138.—Symbols of various forms of moist râle, for noting on outline diagrams of the thorax (after Wyllie).

most important sign. It also occurs occasionally in pulmonary collapse and œdema.

Fine 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.

(*b*) Medium Crepitation. (*c*) 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 larger 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 will be found to be 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.**[1] 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). 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 breath sounds 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

[1] The difference between ordinary (toneless or non-resonant) crepitations and resonant crepitations is perceived on comparing the sounds produced by the bubbling of soda water in a glass and in the soda water bottle, which is less than half full, and the mouth of which is held close to the ear.

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.

(2) **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**), and when in the smaller tubes they have a whistling character (**sibilant râles**). All varieties of rhonchi occur chiefly during inspiration, the sonorous râles at its commencement, the sibilant not till towards its termination.

Rhonchi 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.

(3) **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 (**fine** friction) 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 (**coarse** friction) 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 limited to the apex of the lung it points with great probability to the pleurisy being secondary to pulmonary phthisis.

With regard to differential diagnosis, the fine 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 (see also p. 169).

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, so that the auscultator can recognise the words spoken, and can hear whispered words, 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 parieties, 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 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 breath sounds, in all diseases which lead to consolidation of the lung—for example, in acute pneumonia and in pulmonary 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. Pectoriloquy may also be heard over consolidated lung tissue, apart from cavity formation.

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 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 is 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 vocal resonance 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.

[1] With the exceptions to be presently mentioned.

Amphoric Vocal Resonance.—In cases of pneumothorax, when the bronchi remain patent and in free communication with the pleural cavity, the vocal resonance is of amphoric character.

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 partially filled with fluid.

CHAPTER XXI

URINARY SYSTEM

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 towards the end of, or immediately after, micturition, because the stone is then compressed against the trigone—the most sensitive part of 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 angioma (urethral caruncle).

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 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 ureter to the testicle and inner side of the thigh (renal colic).

The nervous mechanism of micturition will be considered in a future chapter (see p. 383).

Frequency of Micturition.—Whenever the urine is large in quantity, as in diabetes and waxy disease of the kidneys, for example, there is increased frequency of micturition. This symptom, however, also occurs in many other urinary disorders. In all inflammatory conditions of the prostate and bladder, in pyelitis, 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, (5) freezing-point, and (6) reaction.

1. **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, or, on an average, about 1500 c.c.

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, etc. Further, there is scanty urine in the inflam-

matory 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 patients affected with granular contracted kidney, the quantity of urine secreted is augmented in the later stages, when the heart has become hypertrophied and the blood pressure increased. On the other hand, in waxy disease of the kidneys, polyuria is an early symptom, often occurring even before the presence of albumin can be detected. An increase in the quantity of urine is sometimes met with in nervous affections, particularly in hysteria.

2. **Colour of the Urine.**—In order to obtain uniform results as to colour, the urine should be examined, by transmitted light, in a glass, the diameter of which is about 2 inches; and if not absolutely clear, the urine should be filtered before its colour is noted. 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 comparison the various tints of normal urine are grouped as follows:—pale straw, pale amber, and dark amber. Under abnormal conditions the urine may be colourless, or may be yellowish-red, red, "smoky," reddish-brown, greenish-brown, or brownish-black.

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.

The colour of normal urine is due to a pigment, **urochrome**, which is probably a derivative of hæmoglobin, and which gives no characteristic spectrum.

Urobilin.—Normal urine contains a small quantity of the colourless chromogen of urobilin, which is readily oxidised and

converted into urobilin when the urine is allowed to stand. Urobilin closely resembles hydrobilirubin both in its reactions and in its spectrum (Fig. 139), but it is believed that the two substances are not identical.

The detection of urobilin is usually easy, and depends chiefly on the three following points :—(1) When examined in a conical glass, the urine containing much urobilin is of a rich orange colour with a pinkish tint towards the apex of the cone. (2) Examined with the spectroscope, the acid urine which contains urobilin shows 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 ethereal solution of the pigment will show the spectrum clearly (see Fig. 139). (3) To 10 c.c. of the urine add

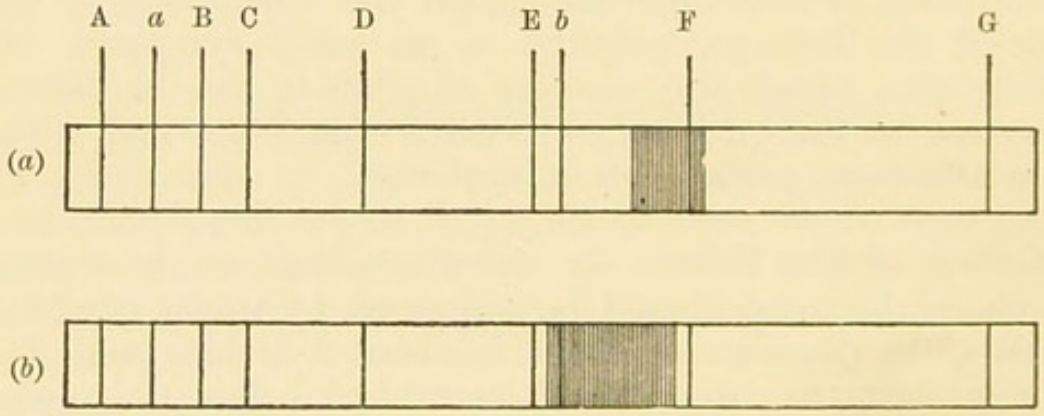


FIG. 139.—Absorption bands of urobilin ; (*a*) in acid solution, (*b*) in ammoniacal solution after the addition of chloride of zinc.

a few drops of hydrochloric acid and from 5 to 10 drops of amylic alcohol, and shake gently. To the amylic extract add a few drops of a 1 per cent. alcoholic solution of zinc chloride and a few drops of ammonia. A green fluorescence appears if urobilin be present.

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 is being absorbed, and also in such diseases as lead to hæmolysis. Thus urobilin is found in the urine in cases of fever, of pernicious anæmia, and as the result of various poisons. It is still uncertain whether in such cases the hæmoglobin is first converted in the liver into bile pigment from which urobilin is subsequently formed by bacterial action in the intestine, or whether urobilin is directly formed from hæmoglobin within the liver. Urobilin is also found in the urine in diseases of the liver such as cirrhosis and venous congestion.

If the intestinal origin of urobilin be accepted, it may be supposed that the healthy liver arrests urobilin, whereas the diseased liver permits it to pass and thus to appear in the urine. The intestinal origin of urobilin is supported by the fact that, if owing to complete occlusion of the common bile duct, no bile enters the duodenum, the urine contains no urobilin.

Uroerythrin is a pinkish-red pigment which often appears in the urines of fever, and of cirrhosis and congestion 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 causes leading to functional disturbance of the liver.

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. Whenever such albuminous putrefaction is taking place, indican appears, in more or less considerable quantities, in the urine. There are mainly two sets of conditions under which this may occur. There are, firstly, those causes which lead to increased putrefaction in the contents of the intestine, such as constipation, obstruction, typhoid fever, etc.; and secondly, there are the conditions under which collections of pus, either in abscess cavities or in one of the serous sacs, are undergoing decomposition. 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 gently 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 be added it causes a further oxidation of the indigo which prevents the reaction. To obviate this the modification suggested by Obermayer may be made use of. In this process a 20 per cent. 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-blue 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.

Indigo-red is isomeric with indigo-blue, and may impart a reddish colour to the urine. Indigo-red may be detected by Jaffé's test, or by adding nitric acid to the urine and boiling, when a burgundy red colour will be produced. A red colour is also obtained with nitric acid after administration of iodides or bromides.

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.

Urorosein is present in some urines as a chromogen. It forms a red colour on the addition of nitric or hydrochloric acid to the urine. The red colour disappears on adding an alkali, whereby urorosein is differentiated from indigo-red.

Hæmatoporphyrin, a derivative of hæmatin, but containing no iron, and isomeric with bilirubin, occurs frequently in healthy urine though in very minute amount. This pigment, when present in some quantity, imparts to the urine a dark port wine or even almost black colour. When thin layers are examined, the tint is found to vary from yellowish-red to violet. Hæmatoporphyrin 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, being then of grave significance. According to the observations of Stokvis the pigment also appears in the urine in cases when blood, which has been poured out into the digestive tract, is being absorbed.

The detection of hæmatoporphyrin is best carried out by means of the method described by Garrod. The urine is precipitated with potassium or sodium hydrate, and the precipitate extracted with acidulated alcohol. The spectrum is characteristic. In acid solution, hæmatoporphyrin 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 shows 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.

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 and administration of salol, the urine becomes of a dark greenish-brown colour, due to the presence of an oxidation product of hydrochinon (see p. 300). Rhubarb, senna, cascara sagrada, and chrysarobin (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 (see pp. 315, 319).

Froth, which is formed abundantly when the urine is shaken, and which remains longer than that of normal urine, points to the presence of albumin. If the urine contain bile, the froth is of greenish-yellow tint.

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 febrile diseases, a dense cloud of urates forms after cooling has taken place, which, as well as other urinary sediments, such as phosphates, pus, bacteria, etc. which render urine cloudy, will be considered in Chapter XXVII.

3. **Odour.**—Freshly passed normal urine has a faint odour peculiar to itself, which gradually disappears. When urine 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 may have 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 should develop, comes to resemble that of chloroform.

4. **Specific Gravity.**—The specific gravity is usually and most conveniently estimated by means of a urinometer. The instru-

ment 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 Hæser, which consists in multiplying the two right-hand figures by 0·233, the result being the amount of the solids in grammes per 100 c.c. of the urine. For example, if the specific gravity be 1022, then, $22 \times 0{\cdot}233 = 5{\cdot}1$ per cent of solids.

The proportion of solids in normal urine is about 4 per cent.

As the specific gravity of 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 of the importance 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.

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 febrile 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 glucose.

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, etc.) or of general interference with nutrition (anæmia, cachexia, etc.), in both cases arising directly from the defective elimination of the urinary salts, particularly urea and other products of nitrogenous metabolism.

5. **Cryoscopy of Urine.**—The freezing-point of urine is an index of its molecular concentration, and varies in health from 0·9° C. to 2·5° C. below that of distilled water. The freezing-point of urine being so inconstant, more valuable evidence regarding the excretory power of the kidneys is obtained by ascertaining the freezing-point of the blood, which in health is constant at from 0·55° C. to 0·57° C. below that of distilled water (see p. 122).

6. **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 (NaH_2PO_4). During the process of digestion, on account of the outpouring of hydrochloric acid into the stomach,[1] the urine loses in acidity, sometimes becoming amphoteric, or even alkaline, owing to the presence of disodic phosphate (Na_2HPO_4) in large amount; 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 mineral acids produces an acid reaction, whereas that of alkalies and of salts of organic acids (which are converted into and excreted as carbonates) renders the urine alkaline.

Alkalinity of the freshly passed urine may either be due 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.

fixed alkali[1] or to the presence of *ammonia*, resulting from the breaking down 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 associated with the presence of *Proteus vulgaris*, *Micrococcus ureæ*, etc., and with certain lesions of the spinal cord, and may have been induced by the use of a catheter which had 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. As a result of the rapid absorption of transudates, 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 when the urine is concentrated, or after administration of acids.

The amphoteric reaction, that in which the urine turns red litmus blue, and 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, 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

[1] 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.

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 (see p. 343).

Quantitative Estimation of Acidity.—This is seldom required in clinical work, and moreover can not be satisfactorily performed. An approximate estimation may be made as follows :—10 c.c. of urine are placed in a porcelain capsule, and 90 c.c. of water and a few drops of a 1 per cent. alcoholic solution of phenol-phthalein are added. This mixture is then titrated with decinormal soda solution, until a permanent red colour remains on agitating the capsule. 1 c.c. of decinormal soda solution neutralises 0·0063 gramme of oxalic acid, hence the number of cubic centimetres of soda solution employed × 0·0063 × 10 = the acidity of 100 c.c. of urine in terms of oxalic acid.

CHAPTER XXII

Urinary System (*continued*)

UREA AND OTHER PRODUCTS OF NITROGENOUS METABOLISM.

Nitrogen, to the amount of 15 grammes in twenty-four hours, is eliminated in the urine in several forms. About 85·5 per cent. is excreted in the form of urea; 5 per cent. as ammonia; about 1 per cent. as creatinin; from 1 to 3 per cent as uric acid and other purin bodies; the remaining nitrogen being in the form of hippuric acid, indol, skatol, etc.

As is well known, these substances are not formed in the kidney, repeated observations having shown that when both kidneys are removed in animals, urea accumulates in the blood rapidly, and to considerable amount. The products of nitrogenous metabolism leave the tissues chiefly in the form of lactate of ammonia, and this, passing to the liver, is there oxidised to form ammonia compounds, from which, by synthesis in the liver cells, urea is formed. The accuracy of this supposition rests on both experimental and clinical observation.

It is known that in frogs, after removal of the liver, the formation of urea almost entirely ceases, and it is replaced by ammonia. After removal of the liver from birds, nitrogen is excreted in the form of ammonium lactate instead of uric acid as is usual in birds. In mammals, the liver may be thrown almost entirely out of action by means of an Eck's fistula, *i.e.* by uniting the portal vein and inferior vena cava. The excretion of urea is then greatly diminished and that of ammonia greatly increased. It may also be mentioned that in dogs, when serum containing ammonium lactate is perfused through the liver, either after it has been removed from the body or short-circuited, 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.

Uric Acid and other Purin Bodies.—Nucleic acid is derived from nucleo-proteid and nuclein, and its decomposition leads to the formation of the purin bodies, substances constructed on the nucleus $C_5H_4N_4$. The purin bodies, which include the following—uric acid, hypoxanthin, xanthin, adenin, and methyl-xanthins—are, with the exception of uric acid, contained in small amount in many vegetables as oats and potato, in larger amount in beef, mutton, chicken, etc., and in still larger amount in the thymus (sweetbread) and pancreas. The purin bodies of the urine are derived from two sources. They may firstly represent exogenous purins of little nutritive value, ingested with the food; and secondly, they arise endogenously within the body, by the breaking down of tissue cells and leucocytes, and particularly of the nuclei of cells. The purin bodies are in part excreted in the urine as xanthin and methyl-xanthins, and in part oxidised to form uric acid.

The ultimate fate of the uric acid 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 down of cells, because those organs in which this process occurs in largest measure lie within the portal circulation.

Normally about 15 grammes of nitrogen are excreted in the urine in twenty-four hours. 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 chronic interstitial nephritis. As a rule (to which, however, acute yellow atrophy of the liver is a marked exception), the total nitrogenous elimination lies fairly parallel to that of urea.

Estimation of Total Nitrogen in the Urine.—The best method 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 about 1 gramme of copper sulphate, which hastens the reaction, and 10 c.c. of pure sulphuric acid. The mixture is then gently boiled for 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, the ammonia has combined with the sulphuric acid, 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 100 c.c. of a saturated solution of 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 50 c.c. of decinormal sulphuric acid. The distillation requires to be continued for some time, usually about one hour, 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 titrating the sulphuric acid solution with decinormal soda solution, using cochineal as an indicator. Then subtract the number of c.c. of soda solution used from 50, the number of c.c. of sulphuric acid employed, and multiply the difference by 0·0014, because 1 c.c. of decinormal sulphuric acid combines with the quantity of ammonia corresponding to 0·0014 gramme of nitrogen.

The result obtained is the amount of nitrogen, expressed in grammes, in 5 c.c. of urine, and from this the total nitrogen excreted in twenty-hours hours can be easily calculated.

Urea.—Urea is the chief product of nitrogen metabolism, and the increase or decrease of urea excreted is an index of the amount of such metabolism.

Detection of Urea. — Place a few drops of the fluid on a microscope slide, add one drop of nitric acid, and gently warm over the flame. On subsequent cooling, rhombic or six-sided plates of nitrate of urea are formed. They often lie one above another.

Estimation of Urea.—This is performed by the hypobromite method, which depends upon the fact that urea, when treated with hypobromite of soda, breaks down into nitrogen, water, and

carbon dioxide, the last of which is absorbed in the alkaline solution, while the nitrogen comes off as free gas. 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

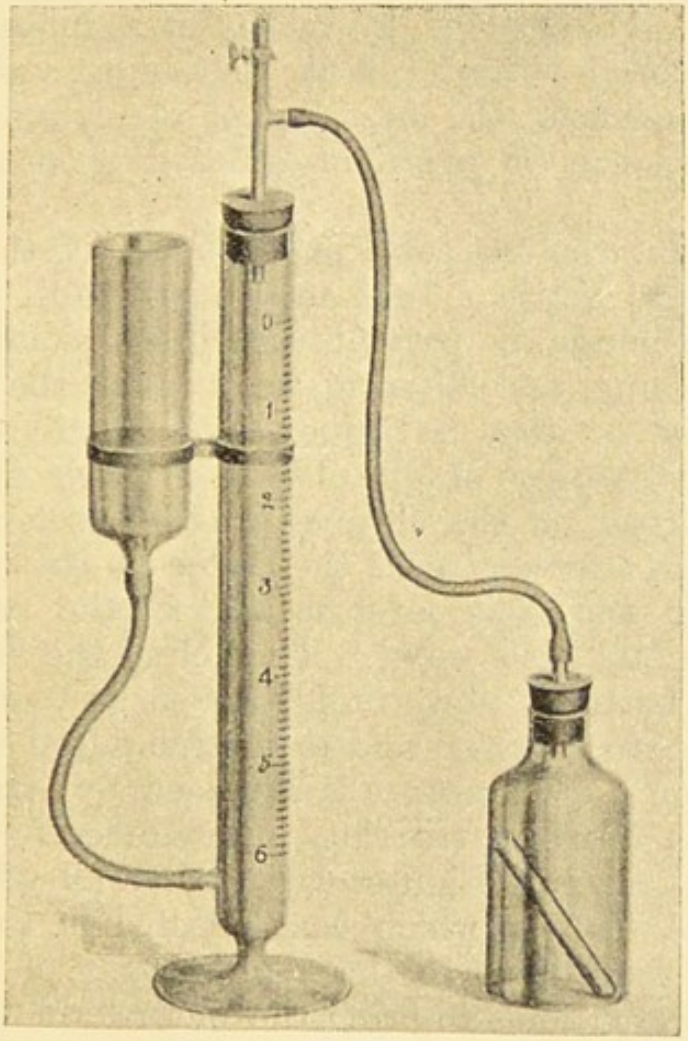


FIG. 140.—Gerrard's apparatus for estimating urea.

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

[1] In theory it should give somewhat more.

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.

Many forms of apparatus are used for the analysis :—

(1) Ureometer of Gerrard (see Fig. 140) is a modification of that originally described by Graham Steele. It consists of a glass cylinder graduated in percentage of urea; a T-tube passes through the rubber stopper closing the upper end of the cylinder. The vertical limb of the tube can be closed by a stop-cock; the horizontal limb is connected by means of a rubber tube with a small wide-mouthed bottle or flask containing a short test-tube. A rubber tube connects the lower end of the cylinder with a glass reservoir, which can be raised or lowered upon the cylinder as desired.

After removing the test-tube, pour 25 c.c. of the hypobromite solution into the bottle. Take in the test-tube, 5 c.c. of urine (from which albumin if present has been removed by adding acetic acid, boiling and filtering) and lower the tube into the bottle carefully, so that no urine mixes with the hypobromite solution. The stopper of the bottle is then firmly replaced. Open the stop-cock on the T-tube, fill the reservoir with water, and elevate the reservoir until the water in the cylinder stands at zero, and at the same level as that in the reservoir, which should now contain little water. Then close the stop-cock on the T-tube. The bottle 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. The mixture is attended by a rapid giving off of gas, and the nitrogen entering the cylinder forces water into the reservoir. Fifteen minutes later, after all effervescence has ceased, and the nitrogen which has collected in the cylinder has had time to cool down to the temperature of the room, the reservoir is lowered so as to bring the water-level within it to the same height as that in the cylinder. This point is read off and represents the percentage of urea.

(2) Ureometer of Doremus.— Of this there are two varieties, that of Southall (see Fig. 141, A) and that of Hind (see Fig. 141, B). If the former be employed, pour in the hypobromite solution so as to nearly fill the wide bulb. Then gradually tilt the tube over so as to fill the vertical limb and bend of the tube, and then restore the tube to the vertical position. Then take exactly 1 c.c. of urine (freed of any albumin) in the curved pipette, wipe the outer surface of the pipette, and introduce its nozzle end as far as possible into the vertical limb of the ureometer tube, slowly compress the rubber nipple until all the urine is driven out of

the pipette, withdraw the pipette, and after withdrawal, but not till then, relax the compression of the rubber nipple.

If Hind's apparatus be used, close the stop-cock on the side-tube, and then fill the vertical limb and bend with hypobromite solution as already described. Then fill the side-tube with urine to the mark zero; slowly turn the stop-cock so as to run in exactly 1 c.c. of urine.

In either form of Doremus tube the nitrogen collects at the upper part of the vertical limb, and the amount can be read off after about fifteen minutes. A scale on one side of the vertical

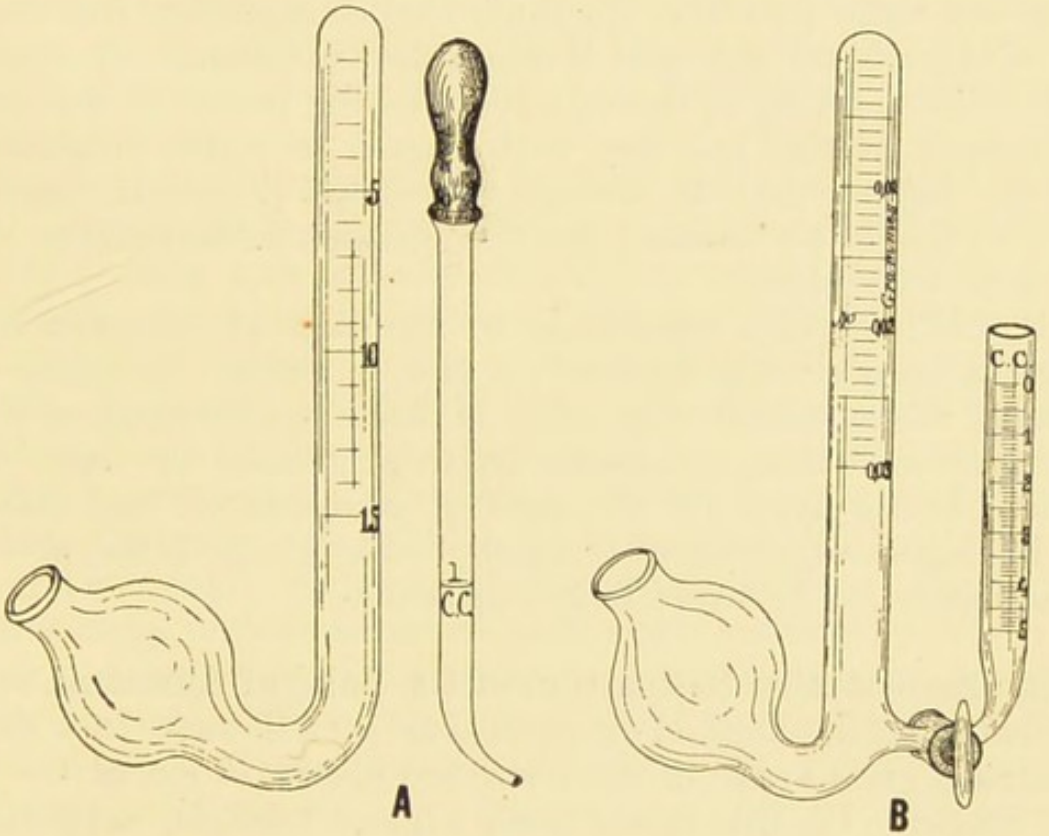


FIG. 141.—A, Southall's ureometer; B, Hind's ureometer.

limb represents grains of urea per ounce of urine; the scale on the other side represents centigrammes and milligrammes of urea per c.c. of urine. For example, if the reading be 0·023 gramme, then that is the amount of urea in 1 c.c. of urine, and consequently the urine contains 2·3 per cent of urea.

If it be found that the urine contains more than 3 per cent. of urea, use urine diluted with an equal quantity of water, and multiply by two the reading obtained.

In order to draw reliable conclusions from the quantity of urea excreted in the twenty-four hours, it is necessary to know the amount of nitrogen taken with the food during that period. In

a healthy man on an ordinary mixed diet, however, the quantity of urea excreted in twenty-four hours averages about 25 to 30 grammes, say 450 grains, which is equal to about 2 per cent., or about 9 grains per ounce. It is increased after a full meal of animal food. In almost all diseases attended with fever the urea elimination is increased. Thus in typhus, pneumonia, pleurisy, and acute rheumatism, the amount of urea excreted is usually much above normal. The excretion of urea is also increased in chronic infective diseases such as chronic tuberculosis and syphilis, and in chronic wasting diseases such as carcinoma. In diabetes mellitus the daily quantity may be very high. This is 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 chronic interstitial nephritis, especially when, to the latter disease, parenchymatous inflammation of the kidney is superadded.

The relation which urea bears to the salts of ammonia in the urine has been already referred to (see p. 288). 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 decinormal 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 decinormal soda solution, cochineal being used as an indicator. 1 c.c. of decinormal sulphuric acid combines with 0·0017 gramme of ammonia, therefore the difference between the number of c.c. of sulphuric acid solution and that of decinormal soda solution required, multiplied by 0·0017, equals the amount of ammonia in grammes in 25 c.c. of urine.

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, are quadriurates), 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. The change occurs either spontaneously in acid urine or after the addition of hydrochloric acid. The uric acid then deposits itself in crystalline form, the character of which will be described when we come to speak of urinary sediments (see p. 339). It must be remembered that a deposition of uric acid is no indication of its increased excretion.

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 *murexide 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 murexide (ammonium purpurate) develops; the addition of a few drops of caustic potash produces a violet-blue colour.

The Quantitative Estimation of Uric Acid may be carried out by the method of Hopkins. 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 estimation of uric acid is not so valuable as the estimation of the total purin bodies (see p. 297).

The average quantity of uric acid excreted in twenty-four hours is about 0·4 to 0·7 gramme. It may be increased to 2 grammes by an animal diet, especially one rich in nucleins, such as liver or sweetbread (thymus). On a diet of milk and vegetables, the quantity of uric acid excreted is much diminished. Apart from such an exogenous source of nucleins, there is an increased excretion of uric acid, usually parallel with that of urea, whenever there is excessive breaking down of cells or tissues, especially of those rich in nucleins, such as the spleen and the leucocytes. In most feverish conditions the excretion of uric acid is augmented, especially in pneumonia after the critical fall of temperature. Between the attacks of gout the excretion is diminished, but during the paroxysm it undergoes some increase. It is increased in many hepatic affections, in acute rheumatism, and sometimes in cases of simple dyspepsia. From what has been said as to the formation of uric acid from nucleins (see p. 289), it will be readily understood that in 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, or fed on thymus or pancreas, allantoin appears in some quantity in the urine. 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.

Other Purin Bodies.—These have been referred to on page 289, and it has been pointed out that they are derived from nucleins, 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 as xanthin and methyl-xanthins. They are constantly present in normal urine to an amount varying from 0·1 to 0·01 gramme *per diem*. As may be understood from what has been already said, these bodies are much increased in the urine under the influence of a diet rich in nucleins, as meat

extracts, sweetbread and butcher meat, and in all cases where there is excessive tissue metabolism, as in fever.

Estimation of the Total Purin Bodies (including uric acid) by the method of Walker Hall. Two solutions are required. No. I. consists of:—Ludwig's magnesium mixture (magnesium sulphate, 1 part; ammonium chloride, 2 parts; ammonia solution, 4 parts; distilled water, 8 parts), 100 c.c.; ammonia solution (20 per cent.), 100 c.c.; and talc, 10 grammes. No. II. solution consists of:—Silver nitrate, 1 gramme; strong ammonia, 100 c.c.; talc, 5 grammes; and distilled water, 100 c.c. The apparatus, the purinometer, consists of (1) a closed tube graduated in c.c.; (2) a stop-cock, with a bore of the same diameter as the upper tube; and (3) a small glass reservoir of known capacity. The method of procedure is described by Walker Hall as follows:—With the stop-cock at a right angle to the tube, urine is poured in up to 90 c.c. The stop-cock is then turned parallel with the tube, and the lower chamber and the bore of the tap become filled with the urine. 20 c.c. of solution No. I. are then added and the precipitate allowed to settle. The precipitate of phosphates sinks into the lower chamber of the purinometer, and immediately this has happened the tap is again turned at right angles. To the clear fluid now remaining in the upper tube, solution No. II. is added to make the total fluid 100 c.c. The resultant precipitate consists of a mixture of silver chloride and silver purin. The apparatus is then inclined backwards and forwards until, the silver chloride becoming dissolved by the excess of ammonia, the precipitate is yellowish-white. The instrument is now allowed to stand for twenty-four hours, when the amount of purin may be read off at the upper level of the precipitate. The number of c.c. of precipitate multiplied by 1·5 and 0·0011 gives the percentage quantity of purin nitrogen. For example: $7 \times 1{\cdot}5 \times 0{\cdot}0011 = 0{\cdot}01$ per cent. of purin nitrogen.

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 about the same quantity as uric acid. Its presence is readily detected by adding to about 5 c.c. 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, which soon passes into deep straw yellow. Acetone gives a similar reaction, but, being volatile, acetone can be removed by previously boiling the urine.

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 rapidily 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, 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, shows a precipitate of the mercury salt of creatinin. 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 1 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 XXIII

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 alkaptonuria. 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 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: in the rabbit, however, this does not appear to be the case.

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.

The quantitative estimation of hippuric acid is performed 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 hydrochloric 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, phenol is formed. 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 mainly as salts of ether-sulphuric acid. In this way the poisonous phenols are rendered harmless and their excretion provided for. These aromatic substances are also excreted as conjugated glycuronates (see p. 328).

The 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, and pyæmia, where putrefaction has taken place in abscess cavities or on absorbing surfaces.

The phenols may be detected by adding Millon's reagent (mercuric nitrate and nitric acid) to the urine, and warming the mixture, when a deep red colour is produced.

The analysis and separation of these substances depend on the fact that boiling the urine with dilute mineral acids causes 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 pyrocatechin 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 Chapter XXIV.

Hydrochinon is not present in normal urine, but appears in cases of carbolic acid poisoning, and it is to the oxidation of hydrochinon and of pyrocatechin that the dark colour of "carbolic urine," which has been standing exposed to the air, 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, partly as conjugated sulphates, 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.

For the method of detection and estimation of these acids, the reader is referred to works on chemical analysis.

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, nor ferment with yeast. Alkaptonuria may occur in persons apparently healthy. The peculiar characters of the urine are probably due to 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 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.

Another substance which has been found in the urine in alkaptonuria is uroleucic acid, which was first discovered by Kirk. It differs from homogentisic acid in that it reduces not only Fehling's solution, but also alkaline bismuth solutions.

CHAPTER XXIV

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 sodium chloride, 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 exudate,—as in acute pneumonia, or in pleurisy with effusion,—a considerable quantity of salt leaves the blood to pass into the exudate. 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 an exudate much salt is

taken up by the blood, and, being excreted, raises the amount of chlorides in the urine. A similar train of reasoning shows 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 partly on the small quantity of food taken under such circumstances.

Detection of Chlorides.—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 (to prevent the precipitation of phosphates), 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 Hammarsten, 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 precipitate 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* (29·055 grammes in one litre of water), 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.*

Take 10 c.c. of urine in a beaker, neutralise with a little calcium carbonate, and add 90 c.c. of water. Add the chromate solution until a yellowish tint is produced. The silver solution is then 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 number of c.c. of the silver solution which have been used is now read off, and 1 c.c. is deducted therefrom, because urine contains other substances which have a greater affinity for silver than the chromate has. Each remaining cubic centimetre corresponds to 10 milligrammes of sodium chloride.

For example, if 12 c.c. of silver solution were used, deduct 1 c.c., and then $11 \times 10 = 110$ milligrammes of NaCl in 10 c.c. of urine.

The average quantity of sodium chloride excreted in the urine, in the healthy state, in twenty-four hours may be taken to be about 12 or 15 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 three forms—firstly, in combination with alkalies; secondly, in combination with aromatic substances as conjugated or ethereal sulphates (chiefly phenyl-sulphate and indoxyl-sulphate, and other aromatic bodies already considered); and thirdly, as "neutral sulphur." The ethereal sulphates, 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 ethereal 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 0·1 to 0·2 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 has 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, 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, potassium, and ammonium (alkaline phosphates), and of calcium and magnesium (earthy phosphates). It may also appear in the form of glycero-phosphoric acid, and lecithin. Phosphoric acid is derived chiefly from the food, but also in part from the breaking down of tissues of the body which contain phosphorus, principally the cell nuclei, 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 (see p. 341).

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 uranium acetate, 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 ferrocyanide 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 acetate, 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 ferrocyanide of potassium not too concentrated.

To 50 c.c. of the urine are added 5 c.c. of the sodium acetate solution, and the mixture is placed in a beaker, and warmed on 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 ferrocyanide 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. On a diet containing a small amount of lime salts—namely, an animal diet—there is much phosphoric acid excreted in the urine. On a vegetable diet—one rich in lime salts—relatively little phosphoric acid is excreted in the urine, whilst much is contained in the stools. There is increased excretion of phosphoric acid after a diet rich in nucleins, sweetbread, for example (see also Uric Acid, p. 296), and also when there is excessive breaking down of tissues rich in nucleins, as in leucocythæmia. Other tissue changes also influence the excretion of phosphoric acid. In chronic nervous diseases and in meningitis 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. In all conditions associated with fever (except malaria) the phosphates are at first diminished, but when convalescence sets in their amount in the urine is increased to a point above normal.

Oxalic Acid, in the form of calcium oxalate, occurs in the normal urine in small quantities (0·01 gramme in twenty-four hours). It is probable that increased excretion of oxalic acid is never due to pathological causes, and that the quantity excreted is mainly

dependent on the amount ingested. The excretion is greatly increased by the ingestion of such vegetables—spinage, asparagus, tomatoes, celery, etc.—as contain oxalic acid, and to a lesser extent by various fruits, honey, tea and cocoa. The precipitation of calcium oxalate in the urine is due to the slight solubility of this salt, and the precipitation can be checked by the administration of magnesia.

CHAPTER XXV

Urinary System (*continued*)

THE PROTEIDS OF THE URINE

Various proteid substances may make their appearance in the urine, under pathological conditions. Those most commonly encountered are serum-albumin, globulin, albumoses, and fibrin.

Normal urine may contain a very minute trace of albumin, derived possibly from the kidney, possibly from the lower urinary passages, but these minute traces are not recognisable by ordinary clinical tests.

Albuminuria, or the presence in the urine of albumin in sufficient quantity to be detected by ordinary clinical tests, is never seen in normal persons, but is a pathological condition.

Detection of Albumin (serum-albumin and 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.—Fill a test-tube half full with urine, and heat the upper part of the fluid 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 to 10 c.c. of urine are placed in a test-tube and acidulated with acetic acid, and $\frac{1}{6}$th 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 globulin are precipitated, but not the albumoses or peptone.

(2) Heller's Nitric Acid Test.—A test-tube is filled to a depth of about one inch with nitric acid. The tube is then inclined, and by means of a pipette the urine is slowly poured down the side of the tube on to the surface of the nitric acid, in such a way that when the tube is again held in an upright position the acid forms a distinct layer at the bottom. If albumin be present in the urine, a sharply defined white ring forms at the line of junction of the two fluids, and is not dissolved on heating.

Heller's test coagulates serum-albumin, globulin, chondro- and nucleo-albumins, and proto-albumose, but not peptone. A cloud or ring may also be due to the presence of urates or resins.

(*a*) *Urates.*—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 heating will suffice to distinguish it.

(*b*) *Resins.*—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 heating.

(*c*) *Chondro- and Nucleo-albumins* are precipitated by the addition of acetic acid alone (see p. 314).

(*d*) *Proto-albumose.*—The precipitate obtained by Heller's test dissolves on heating (see also Albumosuria, p. 313).

(3) The Ferrocyanide 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 ferrocyanide of potassium. If albumin be present, a white flocculent precipitate will separate out at once in the cold. This test coagulates all the proteids except peptone. In some normal urines a cloud is formed by the mere addition of acetic acid. The significance of this is dealt with on page 314.

(4) The Salicyl-Sulphonic Acid Test.—The urine must be acid. If not acid, add a few drops of acetic acid. Then to the urine in a test-tube add about ten drops of a 20 per cent. solution of salicyl-sulphonic acid. If a small amount of albumin be present, the fluid in the tube becomes turbid; a white flaky precipitate is formed if a larger amount of albumin be present. The test precipitates albumin, albumoses, and peptone. The two latter are soluble on warming, whereas albumin remains unchanged. The test is very delicate and reliable.

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 Serum Albumin and Globulins.—In ordinary clinical work the term "albumin" is understood as meaning the mixture of serum-albumin and globulin 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 neutralise 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 globulins separate 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.

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 globulin together.

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 serum-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 0·4 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 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.

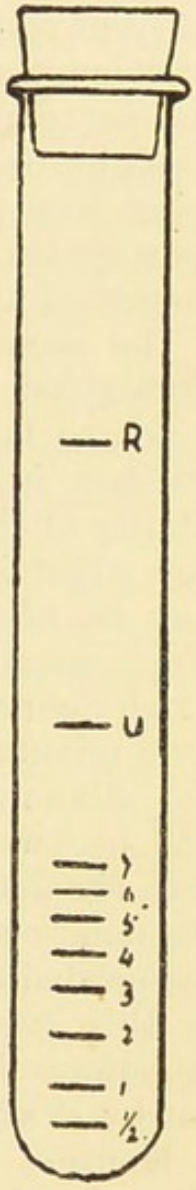


FIG. 142. Esbach's albuminometer.

Still more simply and quickly may the estimation of the serum-albumin and globulin be carried out by taking the coagulum, after washing as above, and estimating its nitrogen by Kjeldahl's method (see p. 289). 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.

Significance of Albuminuria.—There are two great factors in the causation of albuminuria, and most of the pathological con-

ditions 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 altered in such a way as to allow the proteids 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 toxins in the blood 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, or an epileptic fit, or that which Schreiber 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, wasting diseases, and cachexia, the morbid quality of the blood produces a like result. Probably to this category may also be referred the albuminuria which sometimes occurs in exophthalmic goitre, a symptom first pointed out by Warburton Begbie.

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, or after exertion, the quantity present may be very considerable. The changes which produce this condition are unknown, but that they are not of great gravity is shown 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 neurasthenia.

While in the parenchymatous forms of Bright's disease the albumin present is always considerable in quantity, in the chronic interstitial form the quantity is, in the early stages at least, very small, and, out of several specimens examined, albumin may only

be found in some. The changes which occur in such cases in the circulatory system (hypertrophy of the left ventricle, with peripheral arterio-sclerosis and high blood pressure) do not occur in cases of cyclic albuminuria. 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 the chief proteids in the urine are serum-albumin and 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 the globulin, although in febrile albuminuria the amount of globulin may equal that of serum-albumin. The clinical significance of globulin is not known. It does not appear to occur alone, or, at any rate, very rarely so.

But besides these two, other proteids may be encountered in the urine.

Albumoses and peptone are the results of the hydration and splitting 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. True peptone has appeared in the urine in a few recorded cases; but the proteid substance which is found in so-called "peptonuria" is a mixture of albumoses.

Detection of Albumose.—If albumin be present, it must first be removed by adding acetate of soda and ferric chloride, neutralising, boiling, cooling, and filtering. The filtrate is tested for albumose by applying the tests given below. An alternative method of separating albumose is to boil the urine for half an hour with a saturated solution of ammonium sulphate. Albumin, globulin, and albumose are precipitated, and obtained on a filter, but albumose alone is redissolved on adding boiling water to the precipitate. The solution so obtained is therefore tested for albumose.

Tests.—(1) Albumose is not precipitated by boiling. (2) The precipitate obtained by the salicyl-sulphonic acid test (p. 310) is soluble on heating, that of albumin is insoluble. (3) Albumose yields a pink colour with the Biuret test—the addition of one

drop of copper sulphate solution and as much caustic potash solution as urine. The Biuret reaction alone is, however, insufficient evidence of the presence of albumose, for urobilin also gives a pink colour with that test. (4) Nitric acid precipitates proto-albumose; the precipitate dissolves on heating and reappears on cooling. Nitric acid does not precipitate deutero-albumose unless sodium chloride were previously added.

Albumosuria is found in many conditions attended with the breaking down of albumin within the body. It occurs in acute infective diseases, as scarlet fever and measles, and in pulmonary tuberculosis, the main variety of albumose present being deutero-albumose. Albumosuria is also found during the stage of resorption of richly cellular exudates, as in the resolution stage of acute lobar pneumonia, in empyema, and likewise in acute yellow atrophy of the liver, phosphorus poisoning, intestinal ulceration, leucocythæmia, and many other diseases. The determination of the variety of albumose is at present of no clinical importance.

Bence-Jones' Proteid.—In cases of multiple myeloma of the bone marrow, the urine may contain a proteid, the identity of which with hetero-albumose is now denied, and which is regarded as more closely related to albumin. The Bence-Jones proteid is detected by warming the acid urine in a test-tube. If the proteid be present, a milky-looking turbidity or precipitate is formed when the temperature has been raised to about 60° C., *i.e.* some considerable time before the urine begins to boil. The precipitate is dissolved by boiling, and reappears on subsequent cooling, and consequently is readily distinguished from serum-albumin.

Albumin Compounds in Normal Urine.—As already stated, normal urine may contain a trace of albumin, which is not recognisable by the ordinary tests. But a cloudiness is sometimes observed in normal urine on the addition of acetic acid alone. This cloudiness is due to the presence of traces of albumin and of chondroitin-sulphuric acid and nucleic acid, these acids forming, on the addition of acetic acid, insoluble compounds with the albumin. Taurocholic acid in the urine of jaundice may combine with albumin in the same manner.

Fibrin.—Fibrin may be formed only after the urine has been passed, when it separates as a 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 it may occasionally be found in cases of hæmaturia.

Mucinous Substance.—The cloud which forms in the urine on standing contains a mucin-like substance. To demonstrate its presence, filter the urine, dissolve the residue on the filter-paper with weak ammonia, add acetic acid, and extract by shaking up with chloroform. After evaporation of the chloroform, a substance remains which gives the colour reactions of a proteid, and is precipitated from aqueous solutions by acetic acid.

Ferments in the Urine.—Pepsin and ptyalin may be found in the urine. The presence of pepsin may be detected in the following manner:—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, covered with a 0·2 per cent. solution of hydrochloric acid, and kept for a little time at a temperature of 37° 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 37° C. for half an hour, and then tested with iodine solution, when, if ptyalin were present in the urine, the blue colour of starch is not obtained.

Blood may be found in the urine as such (hæmaturia), 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 (see p. 300), while in the latter they are absent. The admixture of even a very small quantity of blood (1 in 1500) 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 detected, 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 (Fig. 143). 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 (Fig. 143).

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 Test for Blood.—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

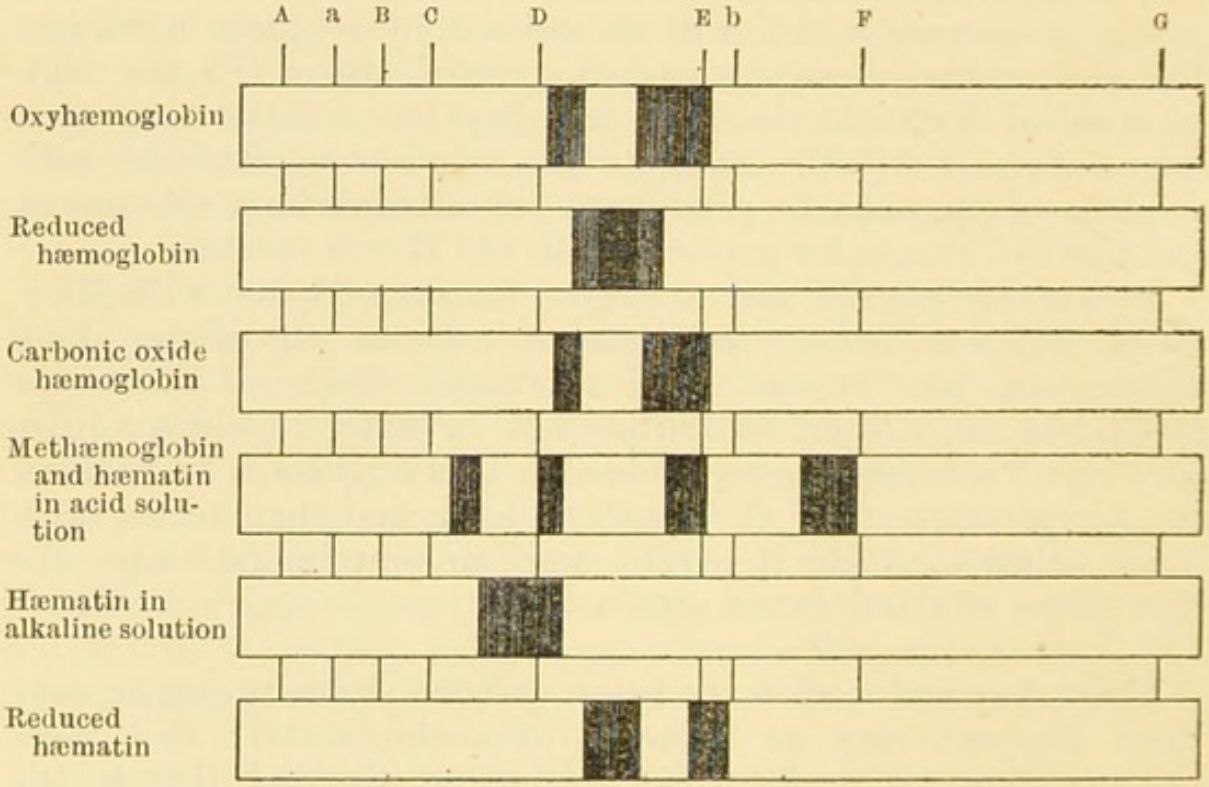


Fig. 143.—Absorption spectra of blood pigment.

circumstances this precipitate is white in colour, but if blood pigment be present it is converted into hæmatin by the alkali, and imparts a more or less deep red appearance to the precipitate. This test is a delicate one. After the administration of rhubarb, senna, cascara sagrada, and chrysarobin, however, a similar colour may be produced.

(2) Guaiacum Test.—To about 5 c.c. of urine in a test-tube add a couple of drops of freshly-prepared tincture of guaiacum, shake the mixture, and then about 5 c.c. of ozonic ether are poured down the side of the test-tube, and the whole is shaken gently. If blood be present, a blue colour quickly appears at the junction of the two fluids. The hæmoglobin has transferred

ozone from the ozonic ether to the guaiacum, which, becoming oxidized, has acquired a blue colour. Saliva and pus also give this reaction, and so does the urine of patients taking potassium iodide. In the latter case, the blue colour develops more slowly.

(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



Fig. 144.—Hæmin crystals.

the urine. These crystals are oblique rhombic prisms, and have a reddish brown colour.

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 tuberculous affections of the kidney; calculus in the pelvis of the kidney; filaria and other blood parasites.

Hæmoglobinuria may appear 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 and is excreted as methæmoglobin. After transfusion of blood a similar lysis of red blood corpuscles takes place, and also as the result of poisoning with carbolic, pyrogallic, hydrochloric, and sulphuric acids, chlorate of potash, and other substances.

Hæmoglobinuria also occurs in the disease termed paroxysmal hæmoglobinuria. In the sufferers from this disease, the paroxysms are usually excited by exposure to cold or by exertion.

Bile in the Urine.—Bilirubin, the pigment of bile, is not a normal constituent of urine, but appears therein in cases of jaundice.

Bile pigment is formed from hæmoglobin. When red blood corpuscles undergo lysis, hæmatin is formed in the liver by destruction of the hæmoglobin. The hæmatin, taking up water and losing its iron, is changed into bilirubin, which is the pigment which appears in the freshly passed urine of jaundice. When the urine is allowed to stand exposed to the air, biliverdin is formed by oxidation; and when the urine undergoes decomposition, bilifuscin and other derivatives are formed.

Jaundice is usually dependent on obstruction to the outflow of bile from the liver. 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. Jaundice may also occur in conditions of toxæmia, as in poisoning with snake venom, phosphorus and other drugs, or in yellow fever,

malaria, pyæmia, typhoid fever, acute lobar pneumonia, and acute yellow atrophy of the liver. In such cases, whether there be excessive hæmolysis or not, the jaundice, although possibly due to perverted function of the liver cells, is usually ascribed to obstruction of the smaller bile ducts by viscid mucus. As the transformation of hæmoglobin into bilirubin does not take place outside the liver, there is no such condition as that formerly termed hæmatogenous jaundice.

Detection of Bile Pigment in the Urine.—The urine of jaundice 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. If the somewhat similar dark greenish-brown urine, passed after absorption of carbolic acid or continued administration of salol (see p. 300), be shaken, the froth is not coloured.

Rosin's Test.—Let dilute tincture of iodine flow gently on to the surface of urine in a test-tube. A green ring forms at the line of contact of the two fluids if bilirubin be present, owing to its conversion into biliverdin by oxidation. This is a more delicate test than

Gmelin's Test.—If a little (about 5 c.c.) nitric acid (which has been allowed to stand exposed to light for some time, and is therefore mixed with nitrous acid) be placed in a test-tube or conical glass, and an equal quantity of urine containing bile pigment be allowed to flow gently on to the surface of the acid, a series of coloured rings will form, from oxidation of the bilirubin, in the following order from below upwards:—yellow, red, violet, blue, and green. The test is not a delicate one, and is positive only if a green colour be produced. The red and purplish colours may be produced by oxidation of urinary chromogens.

Rosenbach's Modification of this test is more delicate. Filter the urine in quantity several times through the same filter-paper, and then allow a drop of nitric and nitrous acid to fall on the surface of the paper. 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 it from the urine with milk of lime. The precipitate, collected on a filter, will show the play of colours when a drop of impure nitric acid is allowed to fall on it.

Bile Acids in Urine.—Even in cases of severe jaundice the salts of these acids are found in the urine only in very small quantity, and urine may contain other substances, including the phenols,

pyrocatechin, and cholesterin, which give a similar colour reaction with Pettenkofer's test, which is therefore not directly applicable to urine. It is necessary to isolate the salts of the bile acids before they can be detected. Their varying amount in the urine is, however, of no clinical significance so far as present knowledge goes.

CHAPTER XXVI

Urinary System (*continued*)

SUGAR AND ALLIED BODIES IN URINE

Sugar.—Even in normal urine a small quantity of glucose (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. 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 is excreted by the kidneys.

In some patients the presence of glucose in the urine is almost the only, if not the sole, evidence of disturbance of metabolism. Such cases are said to present the symptom of glycosuria. In others, the presence of glucose in the urine is only one of many symptoms—intense thirst, progressive emaciation, etc.—which collectively characterise the disease termed diabetes mellitus. It is true that in some patients glycosuria may for long be the sole evidence of disease, and yet the case develop, at a later period, into one of diabetes mellitus. The differentiation of that disease from simple glycosuria may consequently, in certain instances, be a matter of difficulty, but in general the diagnostic features are sufficiently obvious.

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 cir-

culation, and is excreted in the urine. In such persons, however, it is only excessive consumption of sugar, not of starchy food, which causes the glycosuria. Therein such cases of dietetic glycosuria differ from true diabetes.

In other cases the urine contains glucose only when starchy food or sugar is taken, but not when the diet is composed solely of flesh or fat. The appearance of glycosuria in such cases is probably 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 into 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 glycosuria of this kind are often associated with gout, and with various forms of dyspepsia. Beyond the fact that there is sugar in the urine, the patients do not exhibit the well-marked and characteristic symptoms which are seen in diabetes mellitus.

Diabetes mellitus.—In this disease the glycosuria is attended with other pronounced symptoms. There is progressive 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. The severity of the disease varies. In the mild forms, the glycosuria disappears when the patient is put on a proper dietary, but in the severe forms 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.

It is assumed that the immediate cause of diabetes mellitus consists in excessive conversion of glycogen into sugar. In health, the pancreas forms a substance which acts as a check on the reconversion of glycogen into sugar in the liver. In disease, the excessive conversion of glycogen into sugar may be due (1) to pancreatic disease (the checking action of the pancreas being impaired or lost); or (2) to continued stimulation of the liver cells either directly or through the nervous system (the checking action of the normal pancreas being insufficient), in which case there is diabetes in spite of a healthy pancreas.

Qualitative Tests for Glucose.—In all cases albumin, if present, should be got rid of by addition of acetic acid, boiling, and filtration before the following 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. If sugar be present, the heated portion will assume a dark-brown colour and an odour of caramel is given off. 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.

2. Trommer's Test.—To a small quantity of urine in a test-tube add one-third of its volume of liquor potassæ. Then add a drop or two of a 10 per cent. solution of copper sulphate, shaking the mixture after the addition of each drop, until a small quantity of hydrated cupric oxide remains undissolved. If the mixture be now of a deep blue colour, glucose is probably present. On heating the mixture until it begins to boil, a yellow colour (cuprous hydrate) will show itself if sugar be present, and will pass into a reddish granular precipitate of cuprous oxide. The test is neither very delicate nor very reliable. A small quantity of glucose in the urine may cause a yellow colour but no distinct precipitate, whereas if an excessive amount of copper sulphate has been originally added, black cupric hydrate is formed on heating. The fallacies of Trommer's test are described when dealing with those of Fehling's test.

3. Test with Fehling's Solution.—The method of preparing Fehling's solution will be described on page 325. A small quantity of that solution is placed in a test-tube, heated to boiling, and then a drop or two of urine added. A better method of procedure is to simultaneously boil Fehling's solution in one tube and the urine in another, and, twenty seconds after cessation of the boiling, to pour a little of the urine into the hot Fehling's solution, so that the reduction of the hydrated cupric oxide may take place at a temperature below 100° C. If the test be performed in either manner, and if glucose be present, reduction of the copper in Fehling's solution will take place, giving rise to a yellow or a red precipitate of cuprous oxide. Fehling's solution is liable to undergo decomposition when kept for some time, and will then of itself become reduced on boiling. If, however, Fehling's solution be always boiled previous to the addition of the urine, this error cannot take place, for if the solution remain clear on boiling, it is in a fit state for use.

Fehling's test is, like Trommer's test, not free of **fallacies.** Lactose and lævulose both reduce hydrated cupric oxide. After the administration of many drugs, as salicine, morphine, antipyrine, and chloral hydrate, the urine, containing these drugs in combina-

tion with glycuronic acid, reduces hydrated cupric oxide even at a temperature somewhat below 100° C. Other conjugated glycuronates (phenol-indoxyl and scatoxyl compounds), uric acid, hydrochinon (p. 300), and alkapton (p. 301) can also reduce hydrated cupric oxide.

When urine is tested, in the manner described, with Fehling's solution, a greenish colour is not infrequently produced. This discoloration probably depends on the creatinin contained in the urine modifying the action of some reducing substance—glucose, uric acid, etc. If the influence of drugs can be excluded, a distinct precipitation occurring at a temperature of about 80° C. probably indicates glucose; whilst a delayed precipitation, especially if the mixture have been long boiled, is not due to glucose, but to some other reducing substance.

4. Fermentation Test.—Under the influence of yeast, glucose breaks up into alcohol and carbonic acid, and this evolution of carbonic acid is the basis of the most reliable qualitative test for the presence of glucose. It is most readily performed by taking two Doremus ureometer tubes, 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 placing the tubes in an incubator. The tubes are examined twenty-four hours later. If sugar were present in the urine under investigation, carbonic acid gas will have collected at the upper part of the 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.

5. Phenylhydrazin Test.—This can be performed in a number of ways:

(*a*) A test-tube is filled for half an inch with crystals of phenylhydrazin hydrochloride, and for another half inch with sodium acetate. Then add the urine until the test-tube is half full, and boil in a water bath over a flame for half an hour. A bright yellow crystalline deposit of phenylglucosazone appears, if glucose be present. When the deposit is examined microscopically it is seen to consist of bright yellow needle-shaped crystals, arranged in sheaves or rosettes.

(*b*) Mix in a test-tube five drops of pure phenylhydrazin and ten drops of acetic acid, add about 1 c.c. of a saturated solution of sodium chloride, and about 4 c.c. of urine. Gently boil the mixture over the flame for about thirty minutes. Bright yellow crystals of phenylglucosazone will be seen if glucose be present.

The test is a delicate one. If no crystals be formed, the urine

does not contain glucose. But a crystalline deposit may also be obtained with lactose (p. 328), pentose, and conjugated glycuronates. With the latter, however, the crystals are shorter, thicker, and have not the characteristic arrangement of phenylglucosazone crystals.

Quantitative Estimation of Glucose.—1. Fehling's Method.—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, 10 c.c. of which are exactly reduced by 0·05 gramme (0·77 grain) of glucose. Take 10 c.c. of Fehling's solution in a porcelain capsule, add about 40 c.c. of water, and boil over a flame. Into this boiling solution allow urine, diluted to 1 in 20 (5 c.c. of urine and 95 c.c. of water), to flow from a burette graduated in tenths of a c.c. A reddish precipitate of cuprous oxide will form, and as more diluted urine is added, the blue colour of the fluid is discharged. The amount of diluted urine which entirely discharges the blue colour contains 0·05 gramme of glucose. If, for example, the amount of diluted urine required be 20 c.c., then 20 c.c. of diluted urine contain 0·05 gramme of glucose. But the urine having been diluted twenty times, 20 c.c. of urine contain 1·0 gramme of glucose, and the urine therefore contains 5 per cent. of glucose.

It is quite unnecessary to express the glucose in grains per ounce, but if this be thought desirable, one has to remember that 10 c.c. of Fehling's solution equal 0·77 grains of glucose, and that one fluid ounce equals 28·395 c.c.

The reaction in Fehling's method is so much obscured by the red precipitate of cuprous oxide which is thrown down that the results are not very accurate. It is better, therefore, to employ

2. Pavy's Method.—In this method ammonia is made use of to prevent the precipitation of the cuprous oxide. 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.

Pavy's solution is prepared as follows:—120 c.c. of Fehling's solution are 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. Through one hole passes a tube connected with a Mohr's burette, and into the other is adapted a bent glass tube to allow of the escape of air, steam, and ammonia fumes, which may thus be led off into a beaker containing a weak solution of acid. The burette, filled with the urine,[1] is fixed in its stand, and the flask, into which 20 c.c. of Pavy's 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.

3. 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 are added, and it is placed 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 multipled by the empirical factor 0·234, gives the percentage of sugar in the urine. For example, if the readings were 1035 and 1015, the percentage of glucose in the urine = $20 \times 0{\cdot}234 = 4{\cdot}68$.

4. Method by Circular Polarisation.—Glucose, when in solution, possesses the 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

[1] It is best, in the first instance, to dilute the urine in the proportion of 10 to 100.

present. Of these the best known is the saccharimeter of Soleil-Ventzke. Its construction is complicated, and we 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 1 decimetre long each degree of the scale represents 1 gramme of glucose in 100 c.c. of urine.

The urine must always be rendered perfectly clear by means of filtration through dry filter-paper 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, hence it is absolutely necessary to get rid of this substance, if it be present, before the saccharimeter is used.

Laurent's polarimeter and other modern polarimeters are more accurate in their readings, and are to be preferred. With Laurent's instrument 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 in twenty-four hours, and correspondingly pale, but nevertheless possesses a high specific gravity, ranging from 1030 to 1050, or even higher. The quantity of the nitrogenous substances excreted is usually, if not invariably, very much increased. The quantity of glucose 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.

Lactose.—This is sometimes found in the urine of women *post partum*, and when, during the period of lactation, bottle feeding is substituted for the maternal milk. Lactose reduces hydrated cupric oxide and rotates a beam of polarised light to the right, but differs from glucose in yielding no gas with the fermentation test, and in giving yellow crystals (of phenyl-lactosazone) with the phenylhydrazin test (p. 324), only when the heated fluid cools. The crystals are redissolved by warming the tube.

Lævulose is sometimes seen together with glucose in the urine of diabetes mellitus. Alimentary lævulosuria has been observed in diseases of the liver. Lævulose gives reactions similar to glucose when tested with Fehling's solution, phenylhydrazin, and the fermentation test, but rotates the polarised ray to the left.

No particular clinical significance is attached to **isomaltose**, **pentose**, and **inosite.**

Conjugated Glycuronates.—Traces of these (see p. 300) appear in normal urine. Larger amounts appear when there is much decomposition within the intestine. Glycuronic acid is also excreted in combination with a number of drugs, such as salicine, antipyrine, morphine, chloral hydrate, menthol, etc. When the urine contains a considerable amount of conjugated glycuronates, it reduces Fehling's solution, rotates the polarised ray to the left, and forms short thick crystals with phenylhydrazin; it is not fermented by yeast.

In certain cases of diabetes mellitus there may be detected in the urine, towards the termination of the case, **acetone**, **diacetic acid** and **β-oxybutyric acid.**

Acetone, normally found in traces in the urine, is greatly increased in diabetes mellitus (especially if the patient be on a strict animal diet), in fevers, in starvation, and after chloroform anæsthesia. When in grave cases of diabetes mellitus diacetic acid and β-oxybutyric acid are contained in the urine, the alkalinity of the blood is found to be reduced. The dyspnœa and other symptoms of diabetic coma are therefore the manifestations of an acidosis or acid intoxication, due chiefly to β-oxybutyric acid and β-amido-butyric acid. The amount of β-oxybutyric acid contained in the urine of twenty-four hours may amount to 20–30 grammes. Although it is thought that these acids are probably derived within the body from glucose, the subject is still one of dubiety.

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 and iodide of potassium. If acetone be present, yellow crystals of iodoform at once separate. Other tests are sometimes used, such as that with nitro-prusside of sodium, which, however, is not reliable.

Tests for Diacetic Acid.—Add to the urine drop by drop a solution of chloride of iron, so long as precipitation of phosphates occurs. Then filter and add a few drops more. If diacetic acid be present a Bordeau-red colour will be produced. But inasmuch as urine after administration of certain substances, notably antipyrine, phenacetine, and salicylates, gives a similar reaction, it is necessary to boil the urine for three to five minutes, and after cooling to apply the chloride of iron test. Diacetic acid is volatile, and hence the urine which has been boiled no longer gives the Bordeau-red colour, whereas if the colour were due to antipyrine or other drug, the reaction is obtained even after the urine has been boiled.

Test for β-oxybutyric Acid.—This substance never occurs in the urine without diacetic acid being also present. Having detected the latter, the urine may be subjected to fermentation with yeast, and then clarified by the addition of lead acetate solution and filtration, after which, if β-oxybutyric acid be present, it will be found that the fluid has the power of rotating polarised light to the left.

Diazo Reaction.—Two solutions are required :—(A) 0·1 gramme of sulphanilic acid, 5 c.c. of hydrochloric acid, and water to 100 c.c. (B) A 0·5 per cent. solution of sodium nitrite. Take an equal quantity of urine and solution A in a test-tube, add a few drops of solution B, a little ammonia, and shake thoroughly. With normal urine, the colour obtained is yellow or orange; the fluid and the froth are of deep red colour in many cases of typhoid fever, tuberculosis, and other diseases, but the reaction is of no diagnostic value.

CHAPTER XXVII

Urinary System (*continued*)

URINARY SEDIMENTS.

The best method of examining the sediment of a urine is to take, by means of a pipette, from the lowest part of the urine glass, urine and any heavy sediment already deposited. From the pipette fill two or four tubes of a centrifugal machine to within half an inch of the top, place the tubes in the centrifuge, and put the latter in action. The tubes are revolved for two or three minutes, and after they have gradually ceased to revolve, they are taken out of the centrifuge and the deposit within them is transferred by means of a pipette to a microscope slide. The specimen is then covered with a cover-glass, and examined microscopically, at first with a low power and subsequently with a high power objective.

If a centrifuge be not available, the urine must be allowed to stand for some hours in a covered conical glass, after which a drop or two of the sediment which has collected is removed by means of a pipette and examined microscopically.

The best method of preserving any sediment for future use is to place it in a small test tube, and shake it up with an equal quantity of glycerine jelly (50 grammes of gelatine, 50 grammes of pure glycerine, and 0·5 gramme of thymol).

Urinary deposits are divided into two classes—organised and unorganised. Of these, the first is by far the more important.

Organised Deposits

These include blood and pus corpuscles, epithelium, tube-casts, oil droplets, spermatozoa, animal parasites or their eggs, and micro-organisms.

1. **Blood Corpuscles** are found in the urine in cases of hæmaturia (see p. 317). 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 their biconcave shape, and become pale. On the other hand, when the urine is concentrated they shrink 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. **Leucocytes and Pus Corpuscles.**—A few leucocytes can be detected in normal urine, and especially in that of female patients. Pus cells, 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



FIG. 145.—Pus and epithelial cells from the urinary tract.

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 more or less homogeneous mass. The addition of acetic acid renders more evident the form of the pus cells and their nuclei.

A less reliable method of determining that the deposit consists of pus is to fill a test-tube to the depth of about an inch with the sediment, to add an equal quantity of liquor potassæ, and to shake the mixture, when, if the sediment be formed of pus, it will become tenacious, glassy, and semi-solid. The altered consistence is readily recognised if the mixture be poured from one test-tube into another.

The presence of pus in the urine is always a sure sign that there exists an acute or chronic inflammation at some part of the

urinary tract—renal abscess, pyelitis, cystitis, urethritis, or prostatic abscess. In women it must be remembered that pus flowing from the genital tract may become mixed with the urine. The urine containing pus should be carefully examined by bacteriological methods to ascertain what bacteria are present.

The reaction of a urine containing pus may be alkaline or acid. If alkaline, the deposit also contains phosphates. Pus in an acid urine is most frequently found in cases of renal tuberculosis, or of cystitis or pyelitis due to *Bacillus coli communis.*



FIG. 146.—Epithelial and pus casts, and pus cells.

3. **Epithelium.**—The epithelial cells found in the urine may be derived from any portion of the urinary tract. The cells are usually more abundant in the urine of female than of male patients. The epithelium of the urinary tubules consists of polygonal cells, each having a large and sharply-defined nucleus. Those of the deeper layers of the pelvis of the kidney and the bladder cannot be distinguished from one another, being from both situations conical or pear-shaped, with one, or sometimes two, tail-like processes. The large irregular squamous epithelial

cells which are often seen in the urine come from the superficial epithelium layer of the bladder or from the vagina (see Fig. 145).

4. **Renal Tube-casts** are almost invariably associated with albuminuria, and most frequently with Bright's disease, but 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 :--

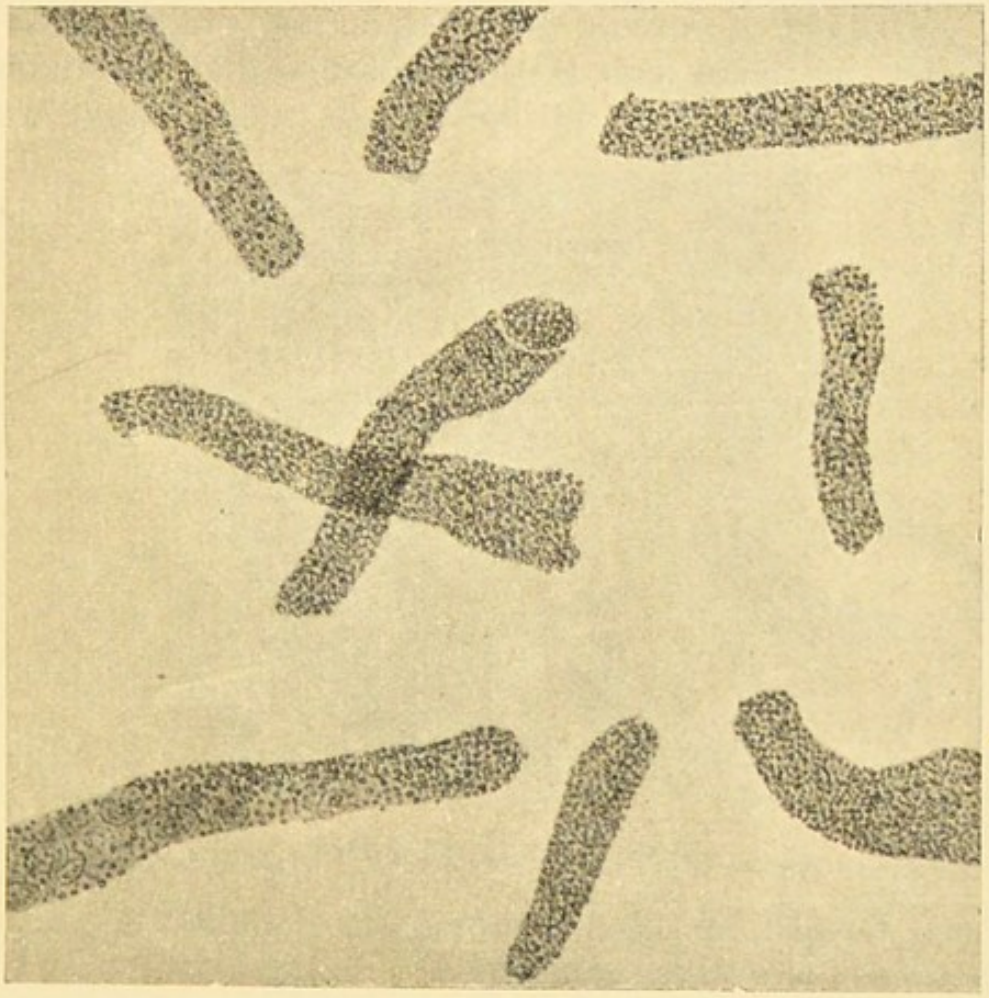


FIG. 147.—Granular casts.

(*a*) *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.

(*b*) *Pus Casts.*—Casts containing pus corpuscles embedded in them are sometimes met with.

(*c*) *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 chronic parenchymatous nephritis.

(*d*) *Granular Casts.*—Dark opaque granular casts are also the result of degeneration in the epithelium of the renal tubules.

(*e*) *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 glomerulo-nephritis.

(*f*) *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

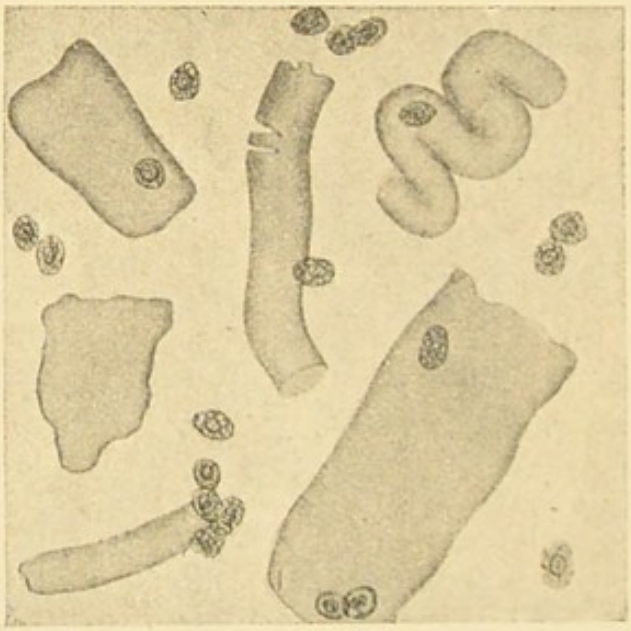


FIG. 148.—Hyaline casts.

have been formed in tubules previously denuded of epithelium, and, therefore, of greater capacity.

(*g*) *Waxy Casts.*—Occasionally homogeneous casts may be found which resemble hyaline casts, but yield the amyloid reaction, becoming mahogany-brown on the addition of iodine solution, and giving a beautiful pink colour with methyl violet, which tinges other casts violet. Such waxy or amyloid casts are more highly refractile than the ordinary hyaline casts, 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 those mucus-coagula which are so often found enclosing in their meshes whatever amorphous inorganic deposit the urine may happen to contain.

5. **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.

6. **Oil Droplets** may be found in the urine (chyluria) in diabetes mellitus, and after fracture of the long bones. In chyluria the urine is of milky appearance, the oil droplets being very minutely subdivided. If chylous urine contain fibrin and be allowed to stand, it may solidify into a mass like *blanc-mange*.

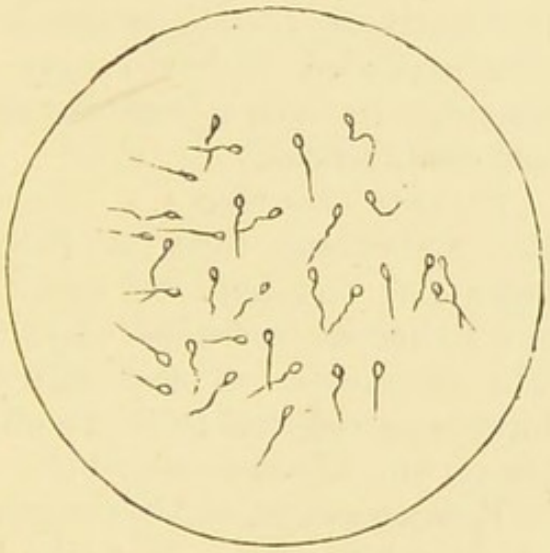


FIG. 149.—Spermatozoa (Roberts).

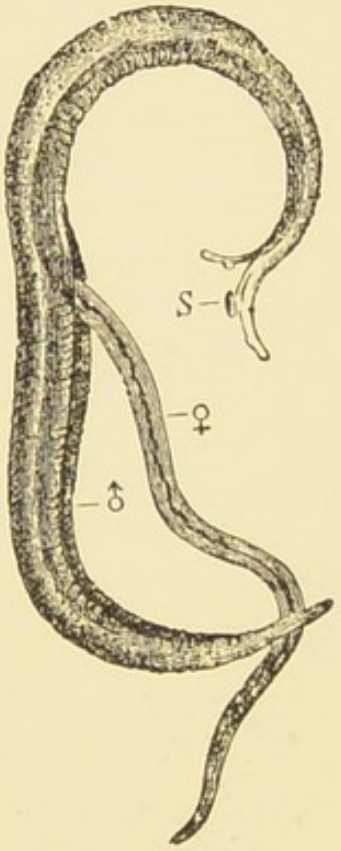


FIG. 150. — *Schistosomum hæmatobium*: S, posterior ventral sucker of male. (After Blanchard.)

7. **Spermatozoa** (Fig. 149) 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.

8. **Parasites.**—The only parasites which need be referred to in connection with the urine are *Schistosomum hæmatobium*, *Filaria nocturna*, and *Echinococcus*.

SCHISTOSOMUM HÆMATOBIUM (DISTOMUM HÆMATOBIUM, BILHARZIA HÆMATOBIA).—The adult male and female worms (Fig. 150) inhabit the rectal and vesical tributaries of the portal vein. The

eggs, escaping from the venous channels of the vesical mucous membrane, appear in the urine, which also contains blood clots, red blood corpuscles, pus cells, epithelial cells, and debris. The eggs (Fig. 151) are spindle-shaped; each is 110–120 μ long, about 50 μ broad, and has a characteristic spine projecting from the posterior pole. In some instances the spine is situated laterally (Fig. 26, p. 90).

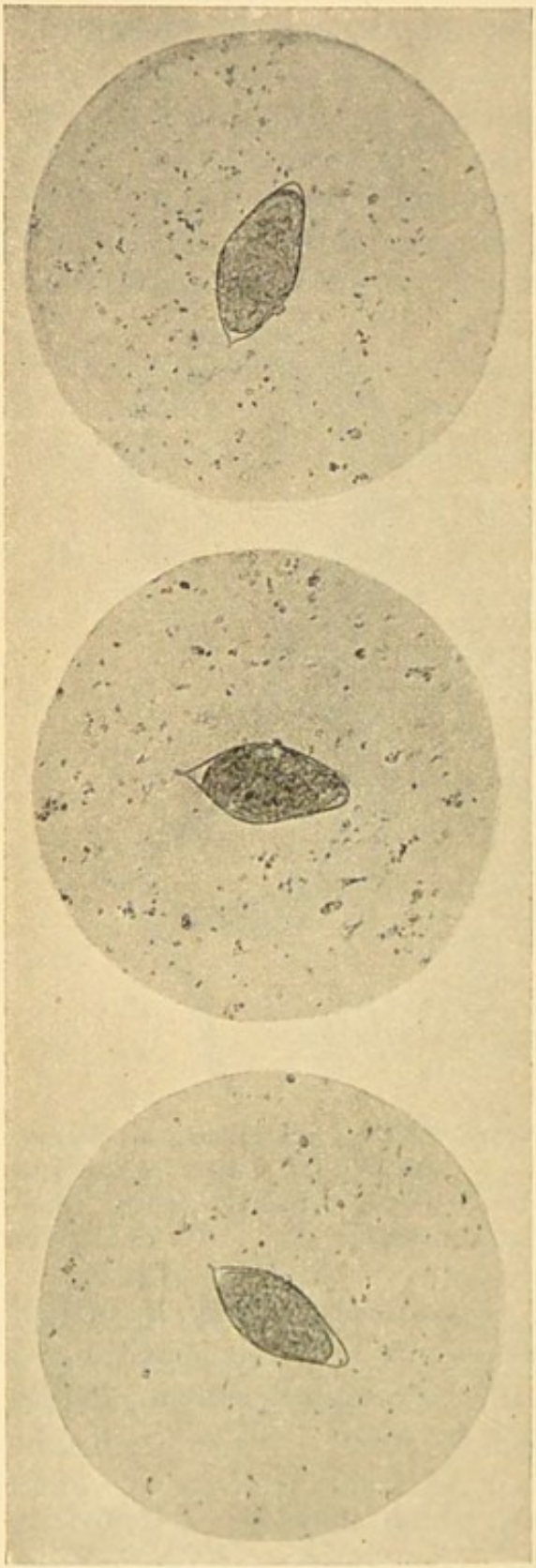


FIG. 151. — Microphotographs of eggs of *Schistosomum hæmatobium* in urinary deposit.

If some of the urine containing the eggs be placed in water in a watch-glass and observed under a low power of the microscope, the actively motile miracidium (the so-called embryo) may be observed to escape from its shell and swim about.

FILARIA NOCTURNA. — This is the embryonic form of *Filaria bancrofti*, and may be found in the urine in cases of chyluria. The characters of the embryos have been referred to in the chapter on the Blood (see p. 118).

ECHINOCOCCUS.—After rupture of an echinococcus cyst (the larval stage of *Tænia echinococcus* of the dog) into the urinary tract, the urine may contain scolices, hooks, or portions of the cyst wall. The hooks (Fig. 106, p. 209) are recognised on microscopic examination by their small size and their slender root-processes; a portion of cyst wall is recognised by the detection of the laminated ectocyst.

9. **Micro-organisms.**—Normal urine when freshly passed may contain a few bacteria derived from the lower urinary passages.

Every urine after having stood exposed to the air is found to be swarming with a variety of bacteria, and consequently a bacteriological examination of urine to be of any value must be performed with certain precautions, which are considered on page 458. The most important bacteria found in the urine in conditions of disease are the gonococcus, staphylococci, streptococci, *Bacillus coli communis*, *Bacillus typhosus*, and the tubercle bacillus (see Chapter XXXVI.).

Unorganised 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 shows 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 reaction can be very readily seen with a larger quantity in a test-tube.

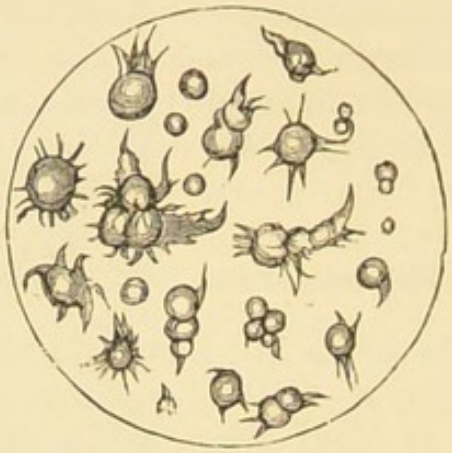


FIG. 152.—Crystalline urate of soda (Roberts).

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, par-

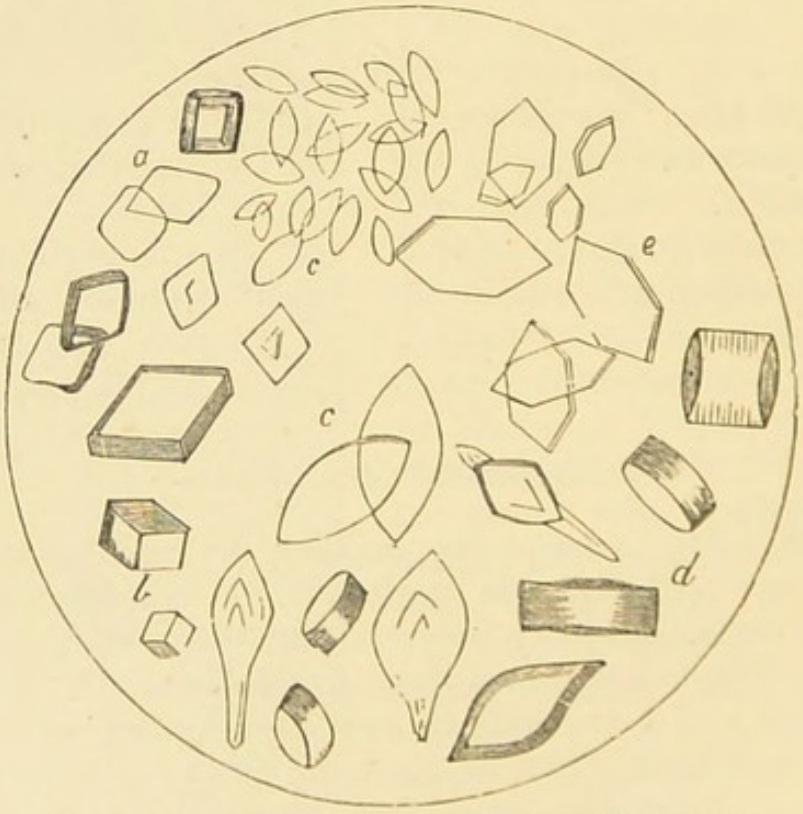


FIG. 153.—Uric acid, simpler form (Roberts).

ticularly of the liver, and in dyspepsia. Very rarely a crystalline deposit of urate of soda may be found, in the form of irregular masses with spiny projections (Fig. 152).

2. **Uric Acid.**—Crystals of uric acid, when present in urine, can usually be seen as bright reddish-brown grains adhering to the 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, or 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, being pigmented with uroerythrin or urochrome, are always more or less yellow; and as no other crystal which spon-

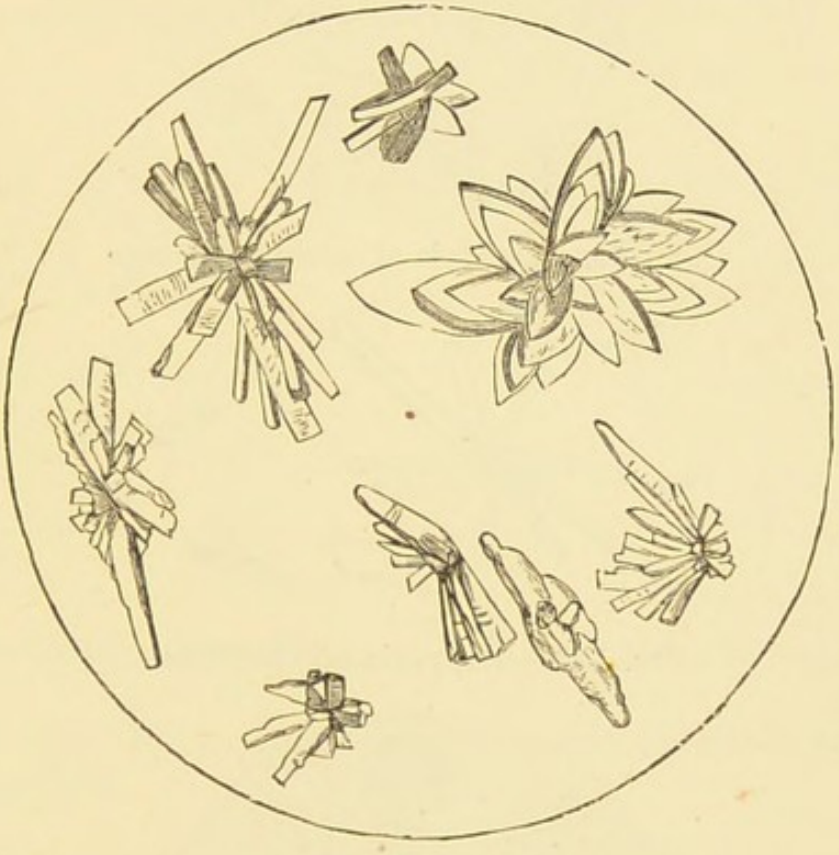


FIG. 154.—Uric acid, stars and spikes (Roberts).

taneously separates out from urine is so tinted, there can be no difficulty in its recognition. As deposition of uric acid crystals is due mainly to increased acidity of the urine and decreased excretion of salts, increased excretion of uric acid cannot be inferred from the deposition of uric acid crystals.

3. **Oxalate of Lime** appears in the urine as small octahedra (Fig. 155), 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.

4. **Hippuric Acid**, though always present in the urine (see p. 299), is but rarely found as a deposit. It crystallises in long rhomboidal prisms (Fig. 156).

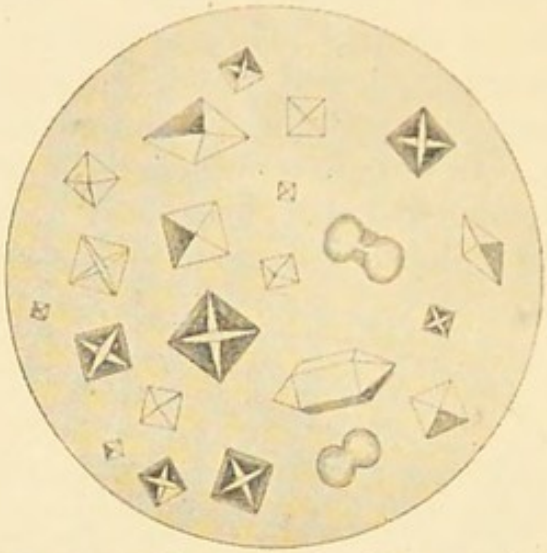


Fig. 155.—Crystals of calcium oxalate.

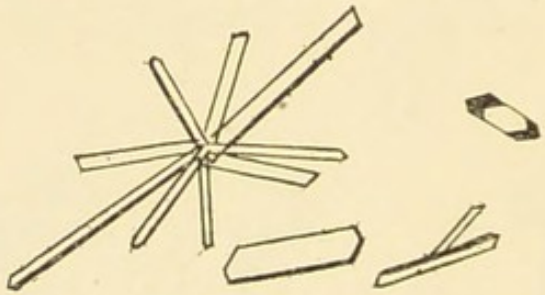


Fig. 156.—Hippuric acid crystals.

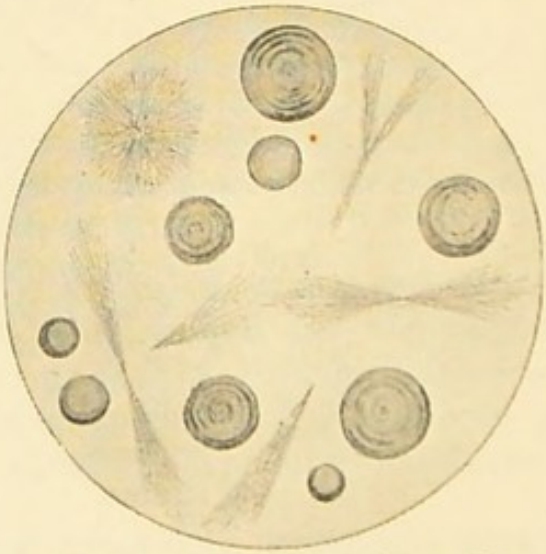


Fig. 157.—Leucin and tyrosin.

5. **Leucin and Tyrosin.** — Leucin appears in the urine microscopically in the form of larger or smaller yellowish-brown balls, which sometimes show distinct concentric striation. Tyro-

sine, on the other hand, appears under the microscope as sheaves of silky glittering needle-shaped crystals (Fig. 157). 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.

6. **Cholesterin** and 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 (Fig. 158) often

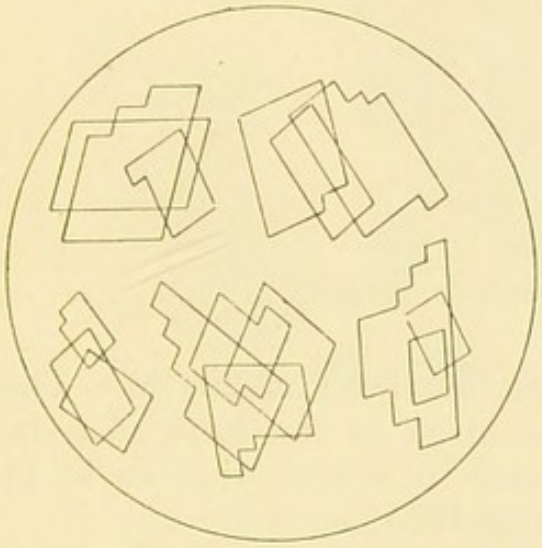


FIG. 158.—Cholesterin crystals.

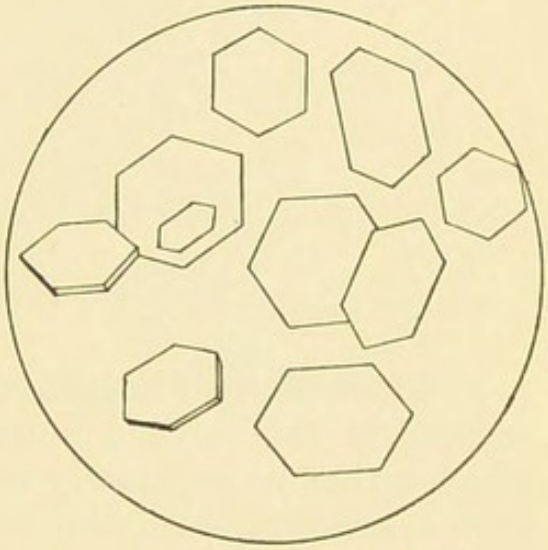


FIG. 159.—Cystin.

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 (Fig. 159), 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. **Armorphous 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.

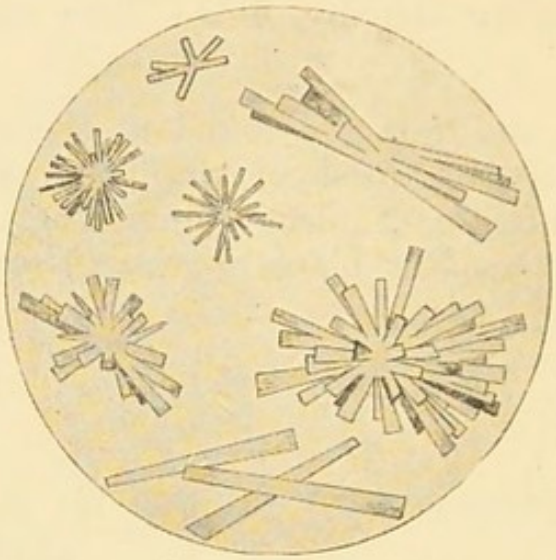
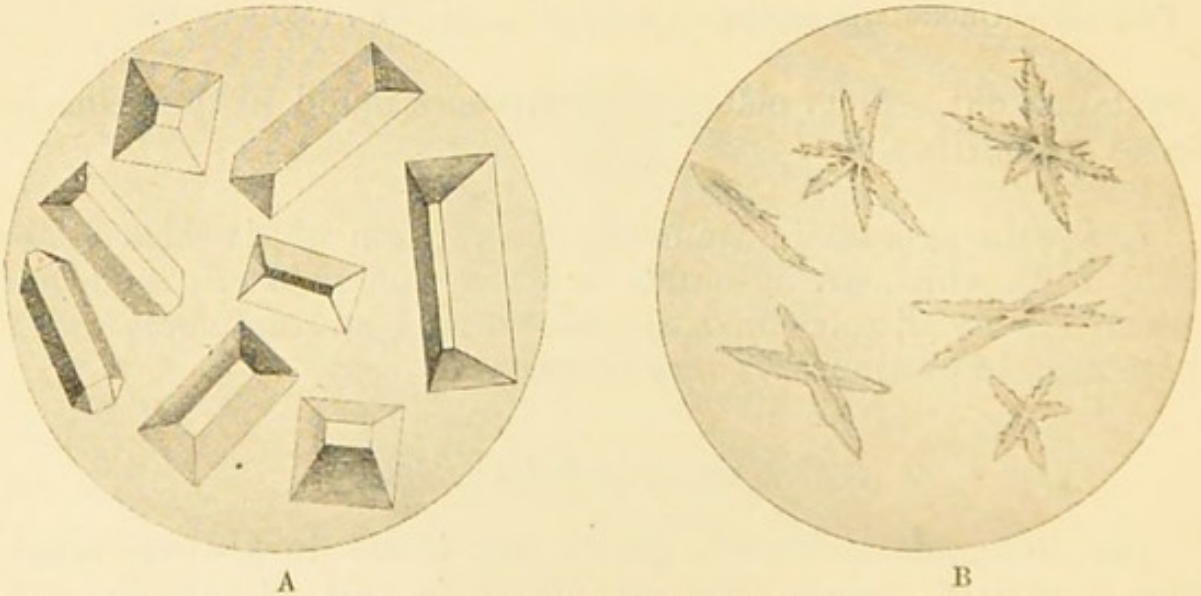


FIG. 160.—Calcium phosphate crystals.

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. 160).



A B

FIG. 161.—Triple phosphates.

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 have usually the shape of a triangular prism with bevelled ends, and present from above the appearance of a glass knife-rest or coffin-lid (Fig. 161, A). Less frequent are forms resembling the star-fish, the so called "feathery phosphates" (Fig. 161, B). The deposition of these crystals is, as a rule, simply due to ammoniacal decomposition of the urine.

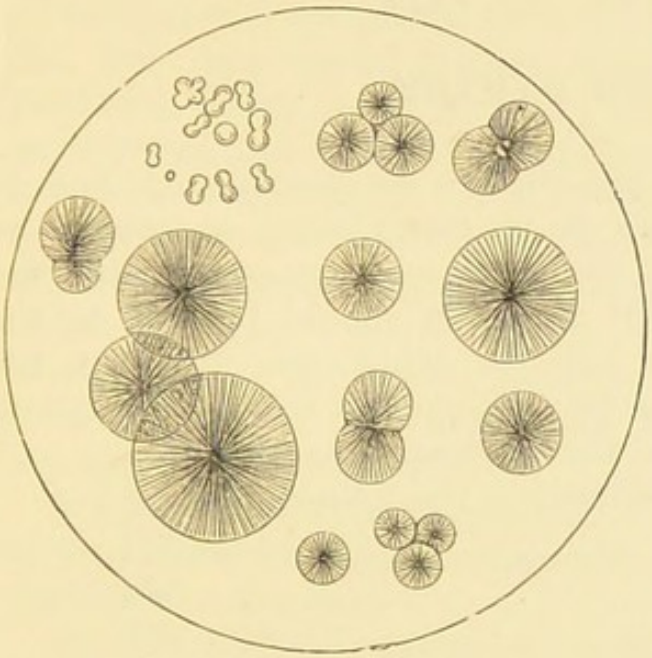


Fig. 162.—Carbonate of lime (Roberts).



Fig. 163.—Urate of ammonia (Roberts).

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. 162).

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. 163).

CHAPTER XXVIII

NERVOUS SYSTEM

The diseases of the nervous system are so complicated, and at the same time their diagnosis is in many 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 . . . . *page* 344.
2. Motor functions . . . . „ 383.
3. Vaso-motor functions . . . „ 420.
4. Trophic functions . . . . „ 422.
5. Cerebral and mental functions . . „ 424.
6. Condition of cranium and spine . „ 434.

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 may 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

[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.

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 (see p. 199) 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.*—The pain associated with disease of the heart and aorta (angina pectoris), of the stomach, intestines, liver, kidneys, uterus, ovaries, and other internal organs is referred to the surface of the body. The explanation is that impulses passing along the sympathetic nerves from a diseased organ to a certain segment of the spinal cord there come into relation with the sensory nerves connecting that segment with the surface of the body, and, possibly as the result of irradiation from one set of neurones to the other, the pain is referred to the surface of the body instead of to the diseased organ. The pain is consequently referred to the area of distribution of one or more of the posterior nerve roots (see Plates V. and VI.).

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 as of having a cord or girdle tied tightly round the body. It may be felt at various levels, on the thorax, abdomen, or the extremities. It is commonly met with in connection with inflammatory and degenerative changes in the cord, particularly in cases of tabes dorsalis, and is believed to result from excitation of the posterior nerve roots.

The distribution of the pain should be carefully ascertained, as it is of great value in localising the level at which the lesion of the spinal cord is situated.

(*b*) *Lightning Pains.*—Among the most common symptoms in tabes dorsalis 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 distribution of one posterior nerve root, now of another. The skin over the affected areas is usually hyperæsthetic, and occasionally shows herpetic eruptions. Lightning pains are not pathognomonic of tabes dorsalis; they may be met whenever there is implication of the posterior nerve roots, as in cases of spinal pachymeningitis, extra-dural new-growth, etc.

(*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 should 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 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. Paræsthesia 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, etc.) or of the central organs (tabes dorsalis, hysteria, etc.), 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 papillæ of the skin. It arises as the 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 creatinin.

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 may also arise from various toxic conditions, such as dyspepsia (*vertigo a stomacho læso*), or from disturbance of the cerebral circulation, as in heart disease. In many cases vertigo arises from paralysis of the muscles of the eye-ball (see p. 363), from excessive eye-strain, 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 gastric crises, pyrosis or waterbrash, 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**,—muscles, ligaments, joints, etc., 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 may be, 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.

Cutaneous Sensibility

There are many forms of sensation connected with the skin, all independent of one another. We shall consider—(1) the sense of touch, (2) the sense of pain, (3) the sense of temperature, and then proceed to discuss (4) the power of localising sensory impressions.

1. **The Sense of Touch.**—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 directed to say "now" or "yes" whenever he feels the touch; and occasionally blank experiments are made to ascertain the correctness of his replies. When sensibility to touch is abolished there is the condition of **tactual anæsthesia**; when it is increased there is **tactual hyperæsthesia.**

The Quantitative Estimation of Sensibility to Touch is best performed by means of the author's *Æsthesiometer*. This simple instrument (Fig. 164) consists essentially of a screw, A, to be rotated by the milled and graduated head, B, and pressing at its lower end on the steel plate, G, and thereby on the plunger, H, in which are fixed six small metal rods, E. The plunger is kept in contact with the screw by a spiral spring, D. By turning the screw downwards, the six rods can be made to project from the smooth metal surface, F, and the degree of their projection is indicated on the graduated head, B.

If the rods be made to project about 0·01 mm., the surface of the instrument feels rough when it is passed over the pulp of the finger. A projection of 0·1 mm. is required before the roughness can be appreciated at the wrist, of 0·25 mm. to 0·3 mm. at the

forearm, and of 0·4 mm. at the upper arm, while still further projection of the rods is required before the roughness is appreciated elsewhere, *e.g.* on the back or the neck.

In using the æsthesiometer certain precautions are necessary. The skin to be tested must not be moist, and observations are to be made on comparatively hairless parts. The temperature of the metal should be as nearly as possible equal to that of the skin; the pressure of the instrument at any two points should be as nearly as possible equal, and the instrument is to be moved on the skin in one direction only and at a uniform rate.

Sensibility to touch may also to some extent be estimated by utilising the sensation of pressure, as in 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

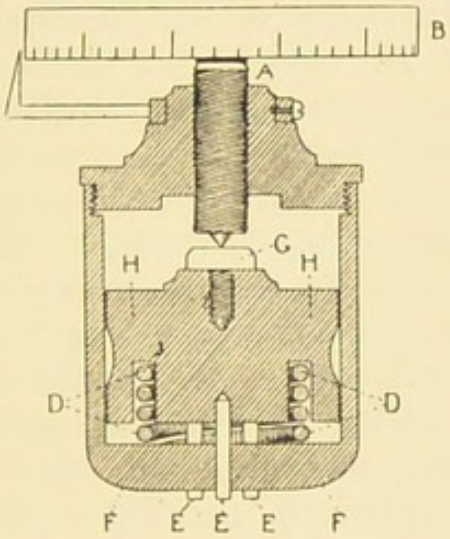


FIG. 164.—Æsthesiometer.

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.

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.

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.

2. **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. When that sense is exaggerated, there is the condition of **hyperalgesia**.

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, varying according to the type of apparatus employed and the strength of the current. According to Bernhardt we may distinguish nine zones of cutaneous sensibility to the Faradic current: I. *The tongue zone* = 16·6 cm. (including the point of the tongue, 17·5, and that of the nose, 15·7); II. *Face zone* = 15·05 (eyelids, gums, red portion of the lips, cheeks); III. *Frontal zone* = 14·45 (lips and forehead); IV. *Shoulder zone* = 13·7; V. *Trunk zone* = 12·8 (upper arm, chest, neck, abdomen, occiput, thighs and forearm); VI. *Thigh zone* = 12·21 (sacral region, thighs, dorsum of first phalanx, dorsum of foot); VII. *Hand zone* = 11·6 (dorsum of hand, leg, palmar aspect of terminal phalanges); VIII. *Patellar zone* = 11·1 (patellar region, dorsal aspect of terminal phalanges); IX. *Toe zone* = 10·45 (points of toes, palm of hand).

3. **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 cannot distinguish between heat and cold (**thermanæsthesia**), or he feels cold well but not heat, or *vice versâ*. More rarely, cases may be encountered where heat is felt as cold, or cold as heat.

In some cases the anæsthesia is a partial one, one or more varieties of sensation being abolished, others remaining intact This constitutes the condition known as **dissociated anæsthesia,** of which the most familiar example is that found in syringomyelia, in which disease there is diminution of sensibility to thermal and painful impressions, while there is preservation of the sense of touch.

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.

4. **The Power of Localising Sensory Impressions** is to be tested by touching or pricking some portion of the patient's body when his eyes are closed, and asking him to indicate the site pricked or touched. In health the error is very small indeed, not greater than 2 cm. Certainly an error of 4 or 5 cm. is abnormal.

The ability to localise sensory impressions may further be investigated by testing the "spacing sense," *i.e.* 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, ...	1·12 mm.
,, palmar surface of last phalanx of finger, . .	2·25 ,,
,, palmar surface of 2nd phalanx of finger, . .	4·50 ,,

On the plantar surface of last phalanx of great toe,	.	11·25 mm.
,, back of the hand,	.	31·5 ,,
,, forearm and leg, and dorsum of foot,	.	40·5 ,,
,, upper arm and thigh,	.	67·6 ,,

(1 mm. = 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.

Delayed Conduction of Sensory Impressions.—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 0·1 second, but in disease this period may be greatly overstepped. It is rare that the sensation of touch is materially 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.

Distribution of Areas of Disturbed Sensibility and their Topographical Significance.—It is important to map out the area over which any disturbed sensibility such as tactual anæsthesia or hyperæsthesia prevails. The area may then be found to correspond to the distribution of a peripheral nerve or a branch of the same, or to the surface distribution of one or more posterior nerve roots, or to correspond to the sensory disturbance resulting from a cerebral lesion or from functional disease.

1. Disturbed sensibility over an area corresponding to the distribution of one or more peripheral nerves indicates that the lesion affects the nerves in question. The surface distribution of sensory nerves of the head, hand, and foot is shown in Fig. 165.

2. Disturbed sensibility over an area corresponding to that innervated by one or more posterior nerve roots (see Plates V. and VI.).—The recognition of disturbed sensibility over such areas (*Segmental areas*) is of great importance in determining that the lesion is one of the spinal cord or its posterior roots and not of the peripheral nerves. It is also important in localising a focal lesion in the cord, for the distribution of anæsthesia, hyperæsthesia, girdle pains, and all other root pain aids us in deciding at what level of the cord the lesion is situated.

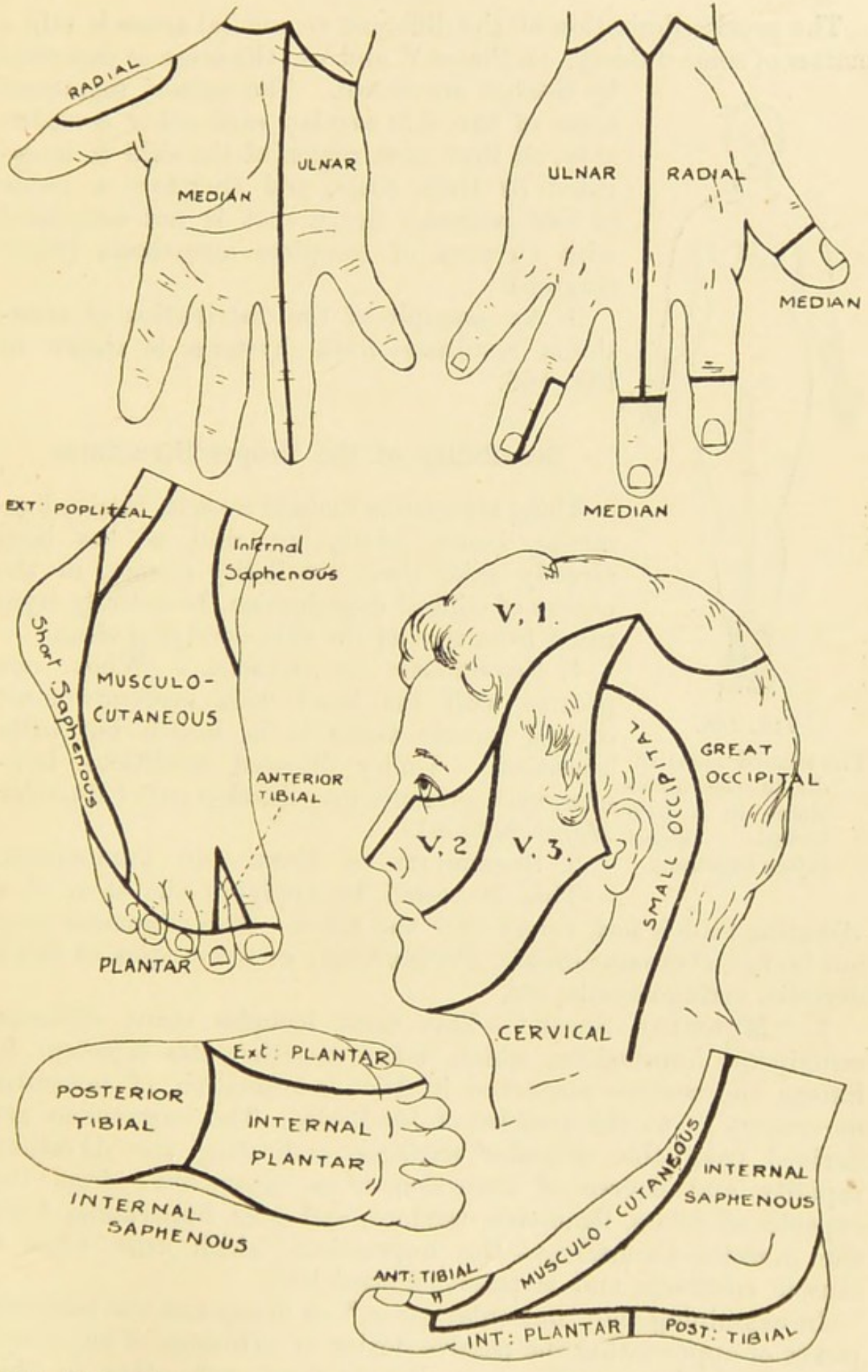


FIG. 165.—Cutaneous distribution of the sensory nerves of the head, hand, and foot (after Bernhardt).

The precise limitation of the different segmental areas is still a matter of some dubiety. In Plates V. and VI., the areas as described by Kocher are shown. The sensory segmental areas of the skin overlap each other considerably, so that each region of the skin is innervated by three roots, and therefore a lesion of one posterior nerve root is not associated with an area of complete anæsthesia (Sherrington).

3. An example of the distribution of anæsthesia resulting from hysteria is shown in Fig. 166.

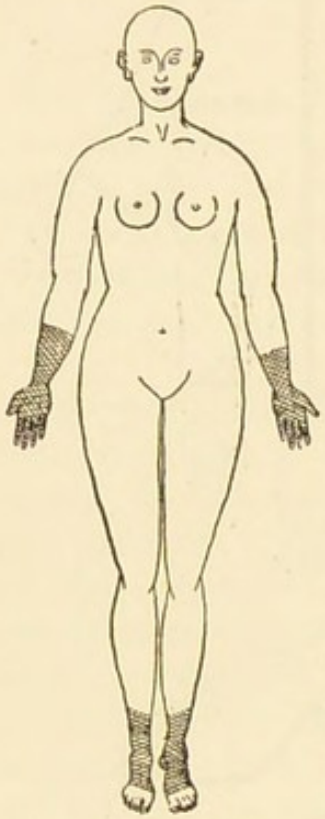


FIG. 166.

The shaded areas indicate the anæsthesia in a hysterical subject (after Dejerine).

Sensibility of the Deeper Structures

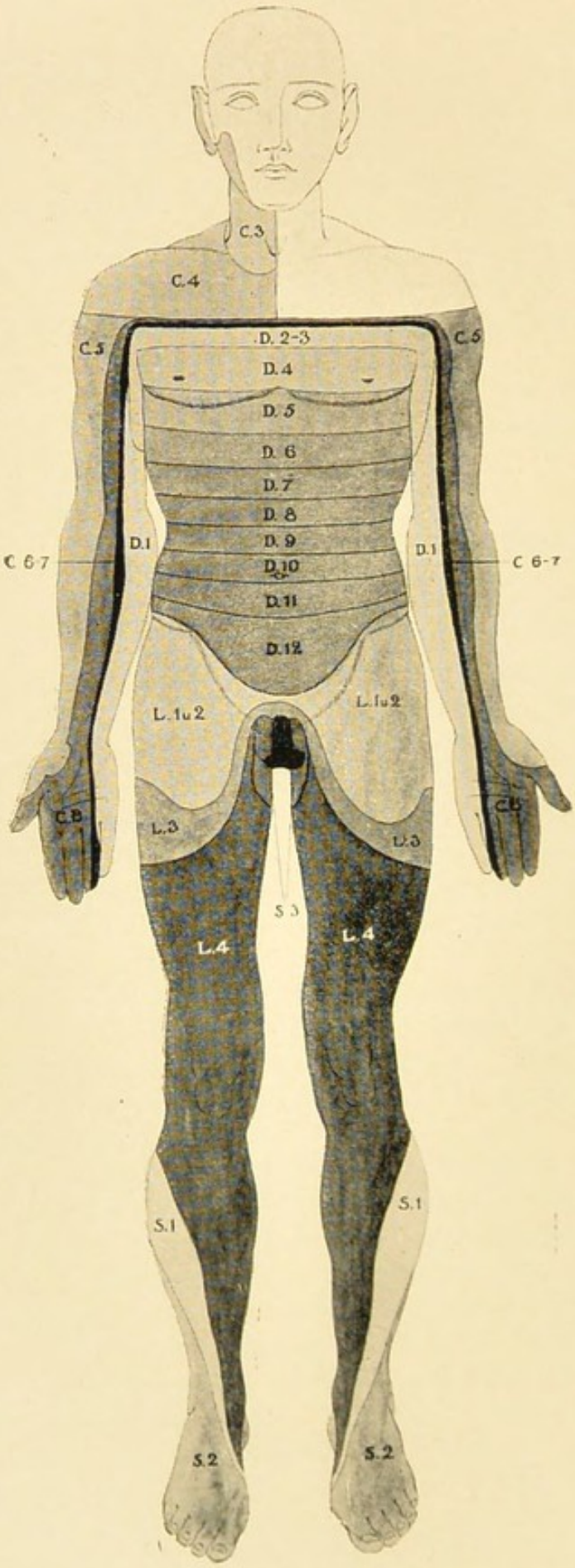
These structures include muscle, fasciæ, ligaments, bones, joints, etc., and, as has been already said, their sensibility cannot, in the course of clinical examination, be entirely separated from that of the skin overlying them.

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 conditions, however, such pressure may develop pain (*Muscular Hyperæsthesia*).

2. SENSIBILITY OF BONE AND PERIOSTEUM. —This is tested by applying the stem of a vibrating tuning fork firmly over the bones. The vibrations may not be felt (*Osteoanæsthesia*, *Pallæsthesia*) in some cases of tabes dorsalis, syringomyelia, etc.

3. "MUSCULAR SENSE."—This term includes many different centripetal impressions, which, taken together, are sufficient to inform the centres concerned in the co-ordination of muscular movement as to the position of the limbs. The impressions are derived from the articular surfaces—indicating the direction, rapidity and degree of excursion of a movement; from the capsules of joints, ligaments, tendons, and most importantly from the muscles themselves—the impressions which arise when a muscle contracts and is passively stretched.

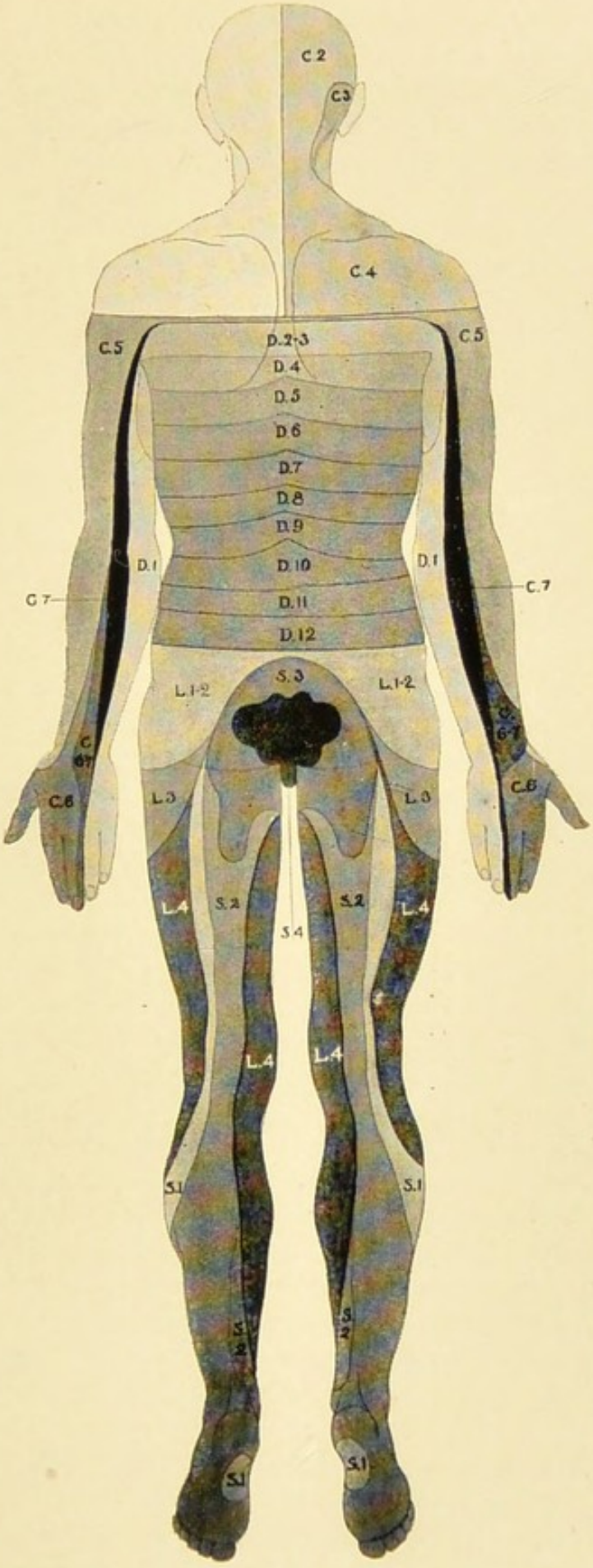
In examining the "muscular sense," we firstly test the patient's power of appreciating the passive flexion or extension of any joint. The various joints differ very slightly from each other in this respect, but it may be said generally that an angular movement



FRONT

Segmental areas (after Kocher).

[*To face p.* 354.



BACK

Segmental areas (after Kocher).

[*To face p.* 354

in connection with any joint of the body corresponding to about a degree and a half ought to be appreciated by a healthy man. In certain of the joints a much lesser degree of movement is appreciated by the patient.

We then test the patient's power of appreciation of active movements. He is asked to make certain movements, such as to touch the tip of his nose, ear, or great toe with his forefinger, or the knee with the heel of the other lower limb, while the eyes are closed. We thus ascertain whether the patient's movements are correctly co-ordinated, or whether there is inco-ordination, that is, whether the patient is ataxic.

A test sometimes used is the determination of the patient's ability 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 seriously complicated by the sensation derived from the pressure of the weight on the skin.

Affections of co-ordination are considered more fully on page 418. Here we may note that the power of appreciating weight and the direction and extent of passive and active movements are often very materially affected, either by lesion of the psycho-motor centres in the cortex or (as in tabes dorsalis) from interference with the conduction of impressions upwards.

4. Stereognostic Sense.—This is the power of recognising objects, such as small cubes, balls, coins, etc., by feeling them while the eyes are closed. The power is greater in the hands than in the feet. It is a judgment or faculty rather than a simple or special sense, and depends mainly on the power of localising tactual impressions, the ability to distinguish two or more simultaneous impressions, and the muscular sense. The various impressions obtained by feeling an object are combined or correlated in the cerebral cortex, possibly in a special "stereognostic area" in the superior parietal region behind the motor areas. It is probable, however, that the impressions derived from handling an object must call into action the visual centres (*i.e.* the visual memory of the object must be called up) before the object is fully recognised.

Astereognosis or loss of the stereognostic sense may occur, firstly, from loss of one or more of the elementary sensory functions, *e.g.* the power of localising tactual impressions, the muscular sense, etc., as may occur in disease of the peripheral nerves, spinal cord (as in tabes dorsalis) or brain, and secondly from a cortical lesion

(tumour, etc.) involving the stereognostic area or a lesion involving the tracts connecting that centre with the visual centres.

According to the recent researches of Head, every part of the limbs and surface of the body possesses three systems of afferent fibres which subserve (1) *Deep Sensibility*, (2) *Protopathic Sensibility*, and (3) *Epicritic Sensibility*.

Complete division of all the sensory nerves to the skin leaves the part sensitive to those stimuli commonly employed as a test for sensibility to touch. All forms of pressure can be appreciated and localised with considerable accuracy. Two points applied successively can be recognised, but, if applied simultaneously, the patient entirely fails to discriminate them, even when the compasses are widely separated. The denervated part is insensitive to heat and cold. Pain can only be evoked by pressure, and then has that peculiar aching character associated with a crush or contusion. This *deep sensibility* is restored rapidly, and seems to reach the part by way of the tendons and fibrous structures connected with them.

Moreover, if a nerve has been completely severed, recovery of sensation does not take place, as is usually believed, by a gradual increase in sensibility, beginning in parts where sensation has never been lost entirely, but the part first acquires *protopathic sensibility*: every stimulus to which the part reacts produces a sensation that radiates widely (there is defective localisation) and is accompanied by a peculiar tingling quality. Gradually there is return of *epicritic sensibility*, *i.e.* a return of sensibility to light touch, together with recovery of sensation to intermediate temperatures and with the discrimination of the two points of the compasses.

The afferent fibres in the peripheral nerves can therefore be divided, according to Head, into three systems:

(1) Those which subserve deep sensibility and conduct the impulses produced by pressure.—The fibres of this system run mainly with the motor nerves, and are not destroyed by division of all the sensory nerves to the skin.

(2) Those which subserve protopathic sensibility.—This system of fibres and end organs responds to painful cutaneous stimuli, and to the extremes of heat and cold. It also endows the hairs with the power of reacting to painful stimulation. In any peripheral nerve the distribution of the protopathic fibres usually overlaps greatly the area supplied by the fibres of the adjacent nerves.

(3) Those which subserve epicritic sensibility.—The nerve fibres and end organs of this system endow the part with the power of responding to light touch with a well-localised sensation. The existence of this system enables us to discriminate two points and to appreciate the finer grades of temperature called cool and warm. The distribution of these fibres in the larger peripheral nerves, such as the median and ulnar, overlaps little compared with the great overlapping of the protopathic supply.

After reunion of a divided nerve the fibres subserving epicritic sensibility regenerate more slowly than those which subserve protopathic sensibility.

CHAPTER XXIX

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 neurone lies entirely within the retina. From this start the fibres of the second neurone. These, running in the optic nerves, reach the chiasma, where a partial decursation takes place (Fig. 167), 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. Most of the fibres in the optic tracts run a much shorter course, terminating around the cells of the "primary optic centres" in the geniculate bodies, and also around cells in the corpora quadrigemina, and that portion of the thalamus known as the pulvinar. From the geniculate bodies starts a third neurone, 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 radiations of Gratiolet. The general course of these fibres may be seen in Fig. 167.

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,

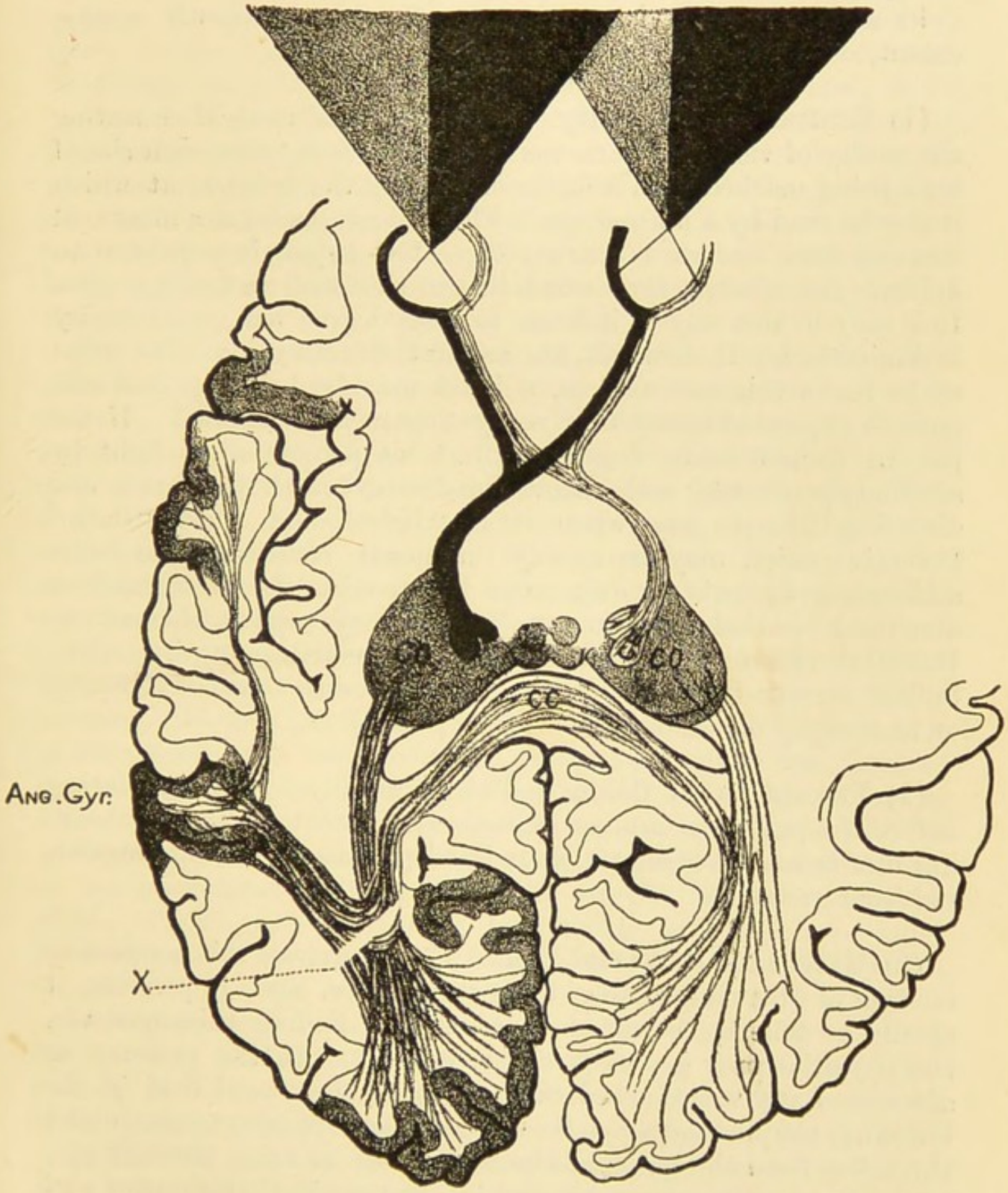


Fig. 167.—Course of the optic fibres (after Dejerine).

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 acuity, extent, or colour.

(1) **Central Visual Acuity.**—The most usual method of testing the acuity of vision is by means of Snellen's test types, 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 acuity 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. If the patient cannot count fingers, we test his perception of light by alternately covering and uncovering the eye with the hand, and directing him to say when it is "light" and when "dark." Defective vision may be merely functional, resulting from neurasthenia or hysteria, or may occur in uræmia. In these cases no abnormal ophthalmoscopic appearances may present themselves. Defective vision may also be due to optic neuritis, retinitis, retrobulbar neuritis (toxic amblyopia, usually due to abuse of tobacco), or to atrophy of the optic nerve (see p. 375).

(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. Colour-blind persons cannot distinguish red from green.

(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 to and about three feet from the patient, who is directed to close the left eye, while the observer closes his right. The patient's right eye and the observer's left being now fixed steadily on each other, the observer moves his hand from the periphery inwards from every direction, keeping it always on a plane midway between the patient and himself, and noting at what point it enters the patient's visual field. Any deviation from the normal condition can be thus readily

detected. When the patient's left eye is to be tested, his right eye and the observer's left eye are to be closed.

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. 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 blue, then yellow, red, and, last of all, green.

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. 167). 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 lesions of the half-vision centre in the occipital lobe, the optic radiations (as in lesions of the posterior part of the internal capsule), the primary optic centres, or the optic tract. 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 (see Chapter XXXIV.).

In homonymous hemianopsia the condition is usually *absolute*, the portion of the visual field affected being dark. But occasionally cases of *hemiachromatopsia* occur in which the perception of light remains while that for colour has disappeared (see p. 362.)

It has been said that homonymous hemianopsia may be due to any lesion which interferes with the visual path at any point behind the chiasma. Fortunately there is a means of localising the site of the lesion a little more precisely, for, by means of **Wernicke's symptom**, it is sometimes 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 optic radiations, or in the 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 behind the primary optic centres, but if no contraction takes place, it is concluded that the portion of the visual path 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. A further localising sign may be found in cases of *hemiachromatopsia*—the condition in which there is loss of colour sense over one-half of each visual field. This condition only arises in a lesion of the occipital lobe on one side. And, further, it may be pointed out that, 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 pressing 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 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 oculomotor 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.**—Paralysis of one or more ocular muscles results in deviation (*primary deviation*) of the affected eye from its correct position. Without going into

undue detail, certain general symptoms indicative of paralysis of the muscles moving the eyeball may be mentioned.

(1) 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.

(2) 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.

(3) 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.

(4) Altered Carriage of Head.—Patients suffering from ocular paralysis learn instinctively to carry the head turned towards the side of the paralysed muscle, so as to reduce the diplopia to a minimum. Hence each variety of paralysis shows a special attitude of the head, recognisable by a trained observer. For example, if the right external rectus be paralysed, the head is turned to the right; if the right internal rectus be paralysed, the head is turned to the left.

In examining a case of paralysis of ocular muscles, it will be found that when the affected eye fixes, the deviation of the healthy eye (the *secondary deviation*) is greater than the primary deviation (that of the affected eye when the healthy eye fixes). The explanation is that a very strong impulse is sent to the affected muscle in order to get as much contraction as is possible in the paretic muscle (*e.g.* the right external rectus). The same impulse being sent to the healthy eye, is much more than necessary. Hence the healthy muscle (*i.e.* the left internal rectus) contracts too powerfully, and secondary deviation is the result.

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 Third 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 eyelid—ptosis.

(*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.

Left-sided paralysis.



Fig. 168.

Right-sided paralysis.



Fig. 169.

(*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.

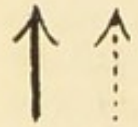


Fig. 170.

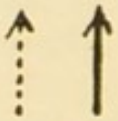


Fig. 171.

(*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. 172.

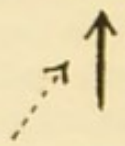


Fig. 173.

(*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. 174.

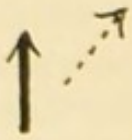


Fig. 175.

(*f*) *Ciliary Muscle.*—The effect of paralysis of this muscle is that the patient is unable to accommodate the eye for a near object. This point is subsequently alluded to.

[1] In these figures the false image is indicated more faintly than the true.

When the third nerve is paralysed as a whole all these actions combine, and the result is, that the upper lid droops, the eyeball is rotated downwards and outwards, and protrudes slightly from its socket, the pupil (as we shall presently see) is dilated and immobile, and there is defective power of accommodation.

Left-sided paralysis.

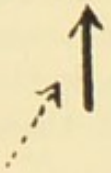


Fig. 176.

(2) The Trochlear, or Fourth Nerve, supplies the superior oblique muscle, and when that is paralysed there is diplopia, which is most apparent when an attempt is made to move the eyeball downwards and inwards.

Right-sided paralysis.



Fig. 177

(3) The Abducens, or Sixth 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.

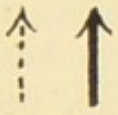


Fig. 178.

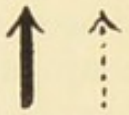


Fig. 179.

When the false image (that formed in the paralysed eye) lies on the corresponding side, as in Figs. 176 and 177, the diplopia is termed **homonymous**, and indicates relatively too great convergence.

Crossed diplopia, as in Figs. 168–173, indicates divergence of the visual axes.

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 ophthalmic surgeon, 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 tuberculous 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 tabes dorsalis, general paralysis of the insane, chronic bulbar paralysis, multiple cerebro-spinal sclerosis, and syphilis. The frequency of ocular paralysis in tabes dorsalis 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 sixth nerve, and thence through the posterior longitudinal fasciculus to the nucleus of the third nerve.

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.

Conjugate deviation is also seen in lesions of the pons, but the direction of the deviation is then 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 show themselves.

When there is paralysis of conjugate deviation, the internal rectus rarely fails to contract on convergence.

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 disseminated sclerosis. 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 iritis, and occasionally in general paralysis of the insane and in tabes dorsalis.

To examine the pupils the patient should be placed opposite a window, in diffuse daylight (not sunlight), 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 light falls on the retina, a stimulus travels along the optic nerve, through the chiasma and the optic tract to the corpora quadrigemina. The path thence to the nucleus of the third nerve is not definitely known. 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 quadrigemina 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.—When the eye accommodates for a near object, 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 centres 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 on convergence.

The Argyll-Robertson Symptom—that condition, namely, in which the pupils no longer react to light, but do react in accommodation and convergence—is very frequently met with in tabes dorsalis, and not uncommonly in general paralysis of the insane.

If the trunk or terminations of one oculo-motor nerve (say the right) are 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 atropine, occurs as a result of lesions involving the third nerve at the base of the brain, occurs occasionally as a form of diphtheritic paralysis, and is sometimes, though rarely, seen in tabes dorsalis.

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 intracranial 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 tumours.

4. Spasm of the Dilator (Spastic Mydriasis).—In this case the pupils are more or less widely dilated, but contract somewhat to light and on convergence. 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.

[1] We have also to except from this rule the case of coma in persons who, from some cause already acting (such as tabes dorsalis), have strongly contracted pupils.

Contraction of the pupils.

(1) In old persons.
(2) In certain affections of the eye, hypermetropia, retinitis, iritis, etc.
(3) From the action of such drugs as morphia, tobacco, physostigmine.
(4) From paralysis of the sympathetic, or destruction of the dilating centres in the cervical cord and medulla.
(5) From increased intracranial pressure—as in cases of cerebral tumours.
(6) From various lesions of the pons and medulla.
(7) In tabes dorsalis, general paralysis of the insane, etc.

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, etc.

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.

The ophthalmoscope consists essentially of two centrally perforated concave mirrors, of different size and different focal length, for the reflection of rays of light into the interior of the eye to be examined. Either mirror can be brought over an eyehole through which the observer looks. A series of convex and concave lenses 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 is also supplied with the instrument a large biconvex lens, which is to be used as will be presently described.

The ophthalmoscopic examination may be conducted in two ways, either with the smaller mirror alone—giving an upright image—the direct method, or with the larger mirror and the biconvex lens—giving an inverted image—the indirect method.

I. **The Direct Method: the Upright Image.**—The patient being seated in a dark room, let us suppose that his right eye is to be examined. A bright lamp is placed at his right side, on a level with his eye. The physician sits close to the right side of the

patient, so that his right eye and that of the patient can subsequently be brought to within 2 inches of each other. The smaller mirror is brought opposite the eye-hole of the instrument, and the mirror is rotated, so that when the ophthalmoscope is held vertically the mirror will be directed towards the lamp. The physician and patient both incline the head towards the right shoulder, and the patient is directed to relax his accommodation completely. With the right hand the physician now holds the ophthalmoscope vertically against his right cheek, resting the upper edge upon the eyebrow, and, looking with his right eye through the central aperture, he inclines the mirror so as to throw a strong beam of light into the right 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 will be 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. If the observer's eye be hypermetropic or myopic, a convex or concave lens will likewise be required before the image is seen distinctly.

To examine the patient's left eye, the physician's left eye is employed. He sits at the patient's left side, places the lamp at the left side of the patient, and holds the ophthalmoscope vertically in the left hand. Both the patient and the physician incline the head to the left shoulder.

II. **The Indirect Method: the Inverted Image.**—The lamp is placed at the left side of the patient, close to the left ear. The observer sits about 2 feet in front of and slightly to the left side of the patient, and advances his left shoulder in front of the right. The larger mirror of the ophthalmoscope is employed. The instrument, held horizontally between the thumb and first three fingers of the right hand, is now placed in front of the observer's right eye. If the patient's right eye is to be examined, he is directed to look at the point of the observer's little finger which is extended beyond the handle of the ophthalmoscope; if the patient's left eye is to be examined, he looks at the tip of the

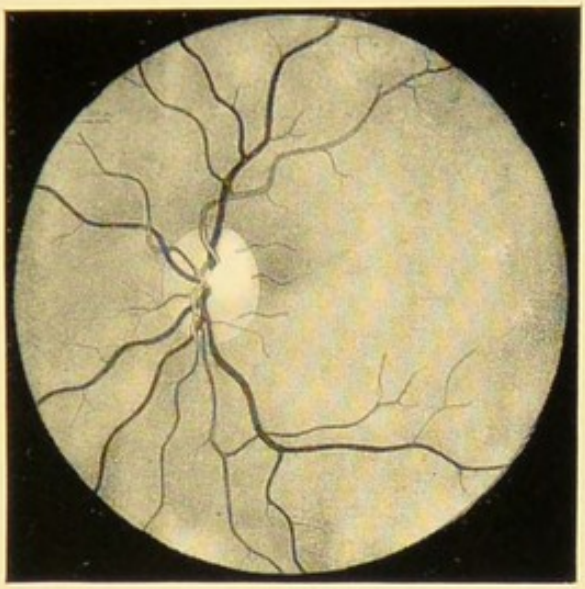
observer's left ear. When the light is now reflected into the patient's eye, a red glare from the fundus will be seen in the pupil. The observer, holding a biconvex lens (of about 2 inches focus) between the thumb and the index finger of the left hand, places it vertically between his eye and that of the patient, at a distance of about 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 fundus is obtained. If the observer have any error of refraction he should correct it by means of a suitable lens placed over the aperture of the mirror.

As it is often desirable to obtain a somewhat larger image, we may with advantage hold a weaker biconvex lens—one of 4 inches focus—in front of the patient's eye, and at the same time bring a +2 D lens behind the mirror.

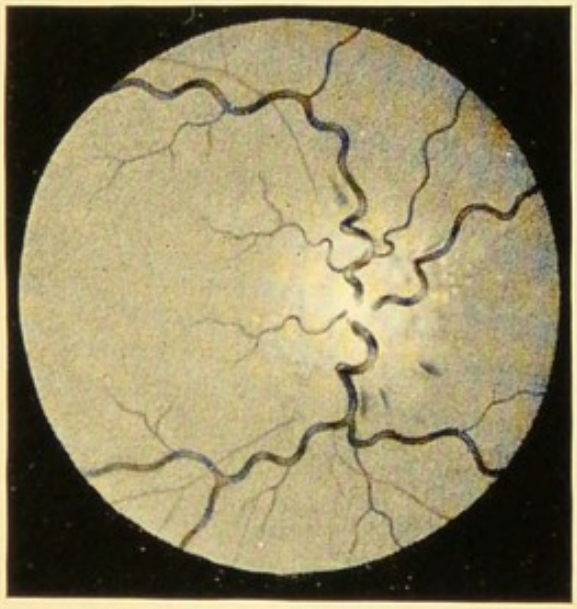
Both the direct and indirect method of using the ophthalmoscope should be made use of, as each has its special advantages. With the first, the area of the retina seen at one time is circumscribed, but is considerably magnified; with the second, the magnifying power is small, but a great deal more of 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 examination methodically by inspecting (1) the optic disc or papilla. To do so, the patient directs his eye somewhat to the nasal side; if the right eye is being examined by the indirect method, he looks at the point of the little finger of the observer's right hand; if the left, he looks at the tip of the observer's left ear; (2) the periphery of the fundus, noting the retina with its blood-vessels, and the choroid. The periphery is seen when the patient looks upwards, downwards, and to the right and left; (3) the macular region: the patient should look at the central aperture of the mirror or at the observer's forehead. It is often difficult to see the macula because of marked contraction of the pupil.

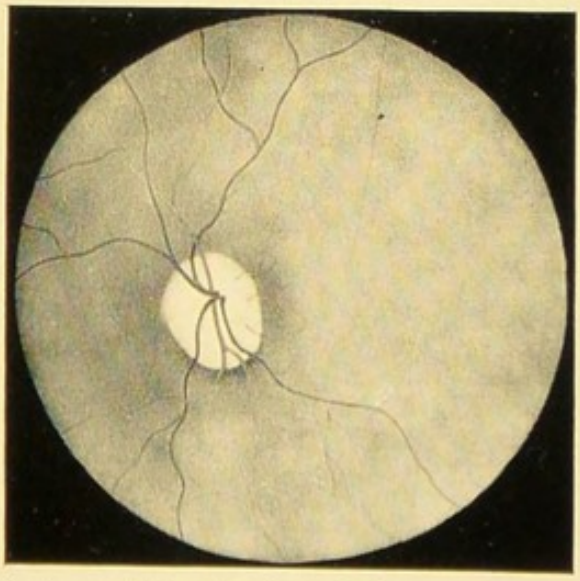
The Optic Papilla is usually elliptical in shape, the result of the angle at which the optic nerve enters the eyeball. The limiting margin of the papilla is formed by the sclerotic and



The normal fundus.



Optic neuritis (after de Wecker and Masselon).



Post-neuritic atrophy of the optic nerve.

[*To face page* 372.

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. By contrast with the rest of the fundus, the papilla appears paler in persons of dark complexion than in blondes. 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 central 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 shows, 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.

The Macula is recognised by being of deeper red than the rest of the fundus, and by the absence of retinal blood-vessels; in

some cases it appears by the indirect method to be surrounded by a bright oval ring or halo.

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 swelling 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.

The swelling of the papilla is best recognised by the direct method of examination. The retinal vessels beyond the papilla having been sharply focussed, a convex lens must be brought behind the central aperture of the mirror in order to bring the vessels on the papilla into focus. The lens required for that purpose is an index of the degree of swelling. For each mm. of swelling a + 3 D lens is required.

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 shows 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 third ventricle. In such cases the state of the pupil reflexes would give important indications (see p. 368). 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 and scarlet fever, by chlorosis and by poisons such as lead and alcohol. 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.**—This may occur under different conditions.

(*a*) Simple Atrophy is the form which is not preceded by recognisable inflammation. Two varieties may be distinguished, namely, *Primary Atrophy*, the result of tabes dorsalis, general paralysis of the insane, or disseminated sclerosis, and *Secondary Atrophy*, due to a lesion of the optic centres, to pressure on the optic tracts, on the chiasma, or on the optic nerve itself. On ophthalmoscopic examination the disc is found to present a greyish 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*) Post-neuritic Atrophy or Consecutive Atrophy is that form which ensues as a result of inflammation of the trunk or intra-ocular end of the nerve. The disc becomes white and its margins may be sharply defined, the swelling of the disc may eventually be replaced by excavation, the vessels become narrowed, and white lines can often be traced along their margins for a considerable distance from the disc.

(*c*) Atrophy following Choroiditis and Disease of the

Retina.—Optic atrophy may result from retinitis pigmentosa or from congenital or acquired syphilitic choroido-retinitis. The disc is pale, its margin is slightly blurred, and the size of the retinal vessels is markedly diminished. If the condition be due to choroido-retinitis, round, white, atrophic areas of different size, surrounded by accumulated pigment, can be observed, mainly in the peripheral part of the fundus.

Diseases of the Retina

1. **Papillo-retinitis.**—When the lesion of the papilla which has 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, are usually flame-shaped, 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 XXX

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 equilibration (vestibular nerve) run for some distance together, yet it sometimes occurs (as in tabes dorsalis) 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 centres (in the superior temporo-sphenoidal convolutions) are affected there is tinnitus or deafness and no giddiness, whereas when the affection is in the middle lobe of 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 salicylic 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 hæmic 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 acuity 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 (**Weber's 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 posterior 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 (see Fig. 180) 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 nerve of taste for the anterior two-thirds of the tongue, on the other hand, is the chorda tympani. This nerve runs with the facial as far as the geniculate ganglion, at which point the fibres conveying taste pass, according to some, through the large superficial petrosal nerve (see Fig. 180) to the second division of the fifth nerve, in the trunk of which they pass to the brain. Others believe that the fibres of taste reach the brain through the *pars intermedia* of Wrisberg.

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," etc., 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 of peripheral origin, 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.

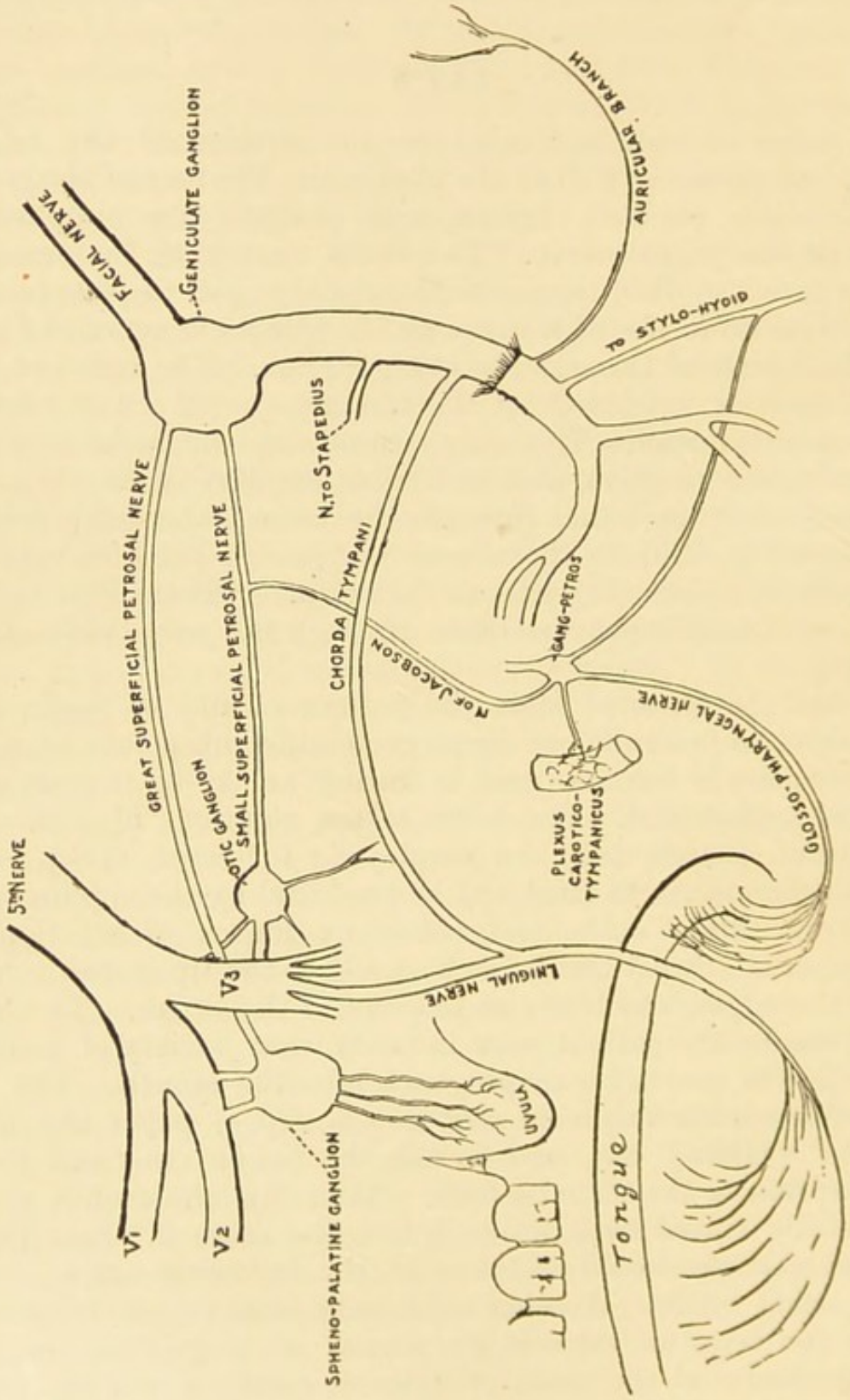


FIG. 180.—Diagram to illustrate the course of the fibres of taste (after Bernhardt.)

In this way it may arise from lesion of the glosso-pharyngeal, 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 glosso-pharyngeal nerve do not affect taste, whereas those of the nucleus of the fifth may do so. When the anæsthesia involves the anterior two-thirds of the tongue, it is due to the fibres of the chorda tympani, 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 situated on the nerve between the geniculate ganglion and the point at which the chorda tympani joins 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.[1]

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, etc.,[2] 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

[1] Considerable doubt exists as to the accuracy of the conclusions stated in 3 and 4.

[2] Such substances as ammonia and acetic acid should be avoided, as they stimulate the terminations of the fifth nerve.

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 tabes dorsalis.

CHAPTER XXXI

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

There are certain reflex actions which are of diagnostic value as indicating the condition of the various neurones concerned in each.

(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 Defæcation.**—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, according to some, in the second and third lumbar and third and fourth sacral segments of the spinal cord (to which also pass the sensory nerves from the mucous membrane of the bladder), whereas, according to others,

the centres for micturition, defæcation, and the sexual functions lie in the sympathetic system. By means of fibres in the spinal cord, these centres are connected with and controlled by 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 question, 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 is produced by degeneration of the afferent sensory fibres passing from the bladder, as so frequently occurs in tabes dorsalis; in such cases the vesical anæsthesia often leads to the bladder becoming overfilled. 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. If micturition occurs automatically in such cases, it does so under the control of the reflex centres only.

When a tranverse 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 there is retention of urine, the bladder filling and the tension on its walls rising 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 **defæcation** is under a mechanism similar to that of micturition, with reflex centres, either in the same portion of the spinal cord or in the sacral part of the sympathetic system. What has been said of micturition under diseased conditions applies, *mutatis mutandis*, to defæcation.

(3) **Sexual Functions.**—The reflex centres for erection and ejaculation are probably located in the second and third sacral segments. If the control of the higher centres is cut off by disease in the upper part of the cord, these functions become imperfect, and may sometimes be excessive (priapism). Disease of the sexual centres in the sacral segments 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

The cells of the **upper motor neurone** lie in the motor areas of the cerebral cortex. The position of the cortical motor areas in the precentral gyrus on the left side is shown in Fig. 181. The axones or axis-cylinders of those cells pass down the motor or pyramidal tracts, traversing in turn the corona radiata, the anterior two-thirds of the posterior limb of the internal capsule, the crura cerebri, the pons, the medulla oblongata (where the majority cross the mesial plane in the decussation), and the pyramidal tracts (direct and crossed) of the spinal cord. The fibres destined for the cranial nerves likewise cross the mesial plane; those, for example, coming from the cortical face centre cross in the upper part of the pons. The fibres of the upper motor neurone are believed to end in arborisations around the

cells of the lower motor neurone in the anterior horns of the cord, and in the corresponding nuclei of the motor cranial nerves.

The **lower motor neurone** consists of the nerve-cell lying in the anterior cornu of the spinal cord, or in the corresponding medullary or cranial nuclei, with its protoplasm, its nucleus, and the arborisation of its dentrites, of its axis-cylinder or axone prolonged to become the anterior root, and the efferent nerve-fibre, and of its terminal arborisation connected with the substance of the muscular fibres which it innervates.

The course of the motor path is represented in Fig. 182, and the position of the tracts of the spinal cord on transverse section is shown in Fig. 183.

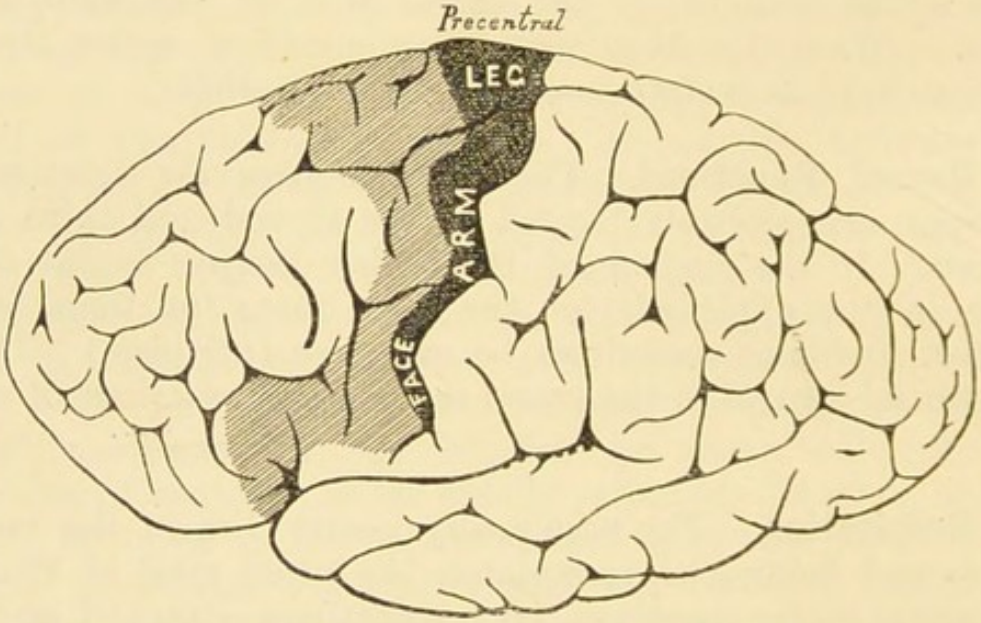


FIG. 181.—Lateral surface of the left hemisphere (modified after A. W. Campbell). The darkly shaded area represents the precentral field, that more lightly shaded corresponds to the intermediate-precentral area.

The motor cells in the Rolandic area are the trophic centres for the fibres of the upper motor neurone. Any lesion destroying the cell in the cortex causes the neurone as a whole to degenerate, and any lesion which cuts the axone causes the peripheral part of that fibre to degenerate, because it is thereby separated from its nutritional centre, the cell in the motor cortex. In either case there is a descending degeneration of the axones in the upper motor neurone in the brain, medulla and cord.

The motor cells of the anterior horn are the trophic centres for the fibres of the lower neurone and the associated muscles, and when there is a lesion of this neurone, whether in the anterior horn, anterior root, or peripheral nerve, the fibres peripheral to the

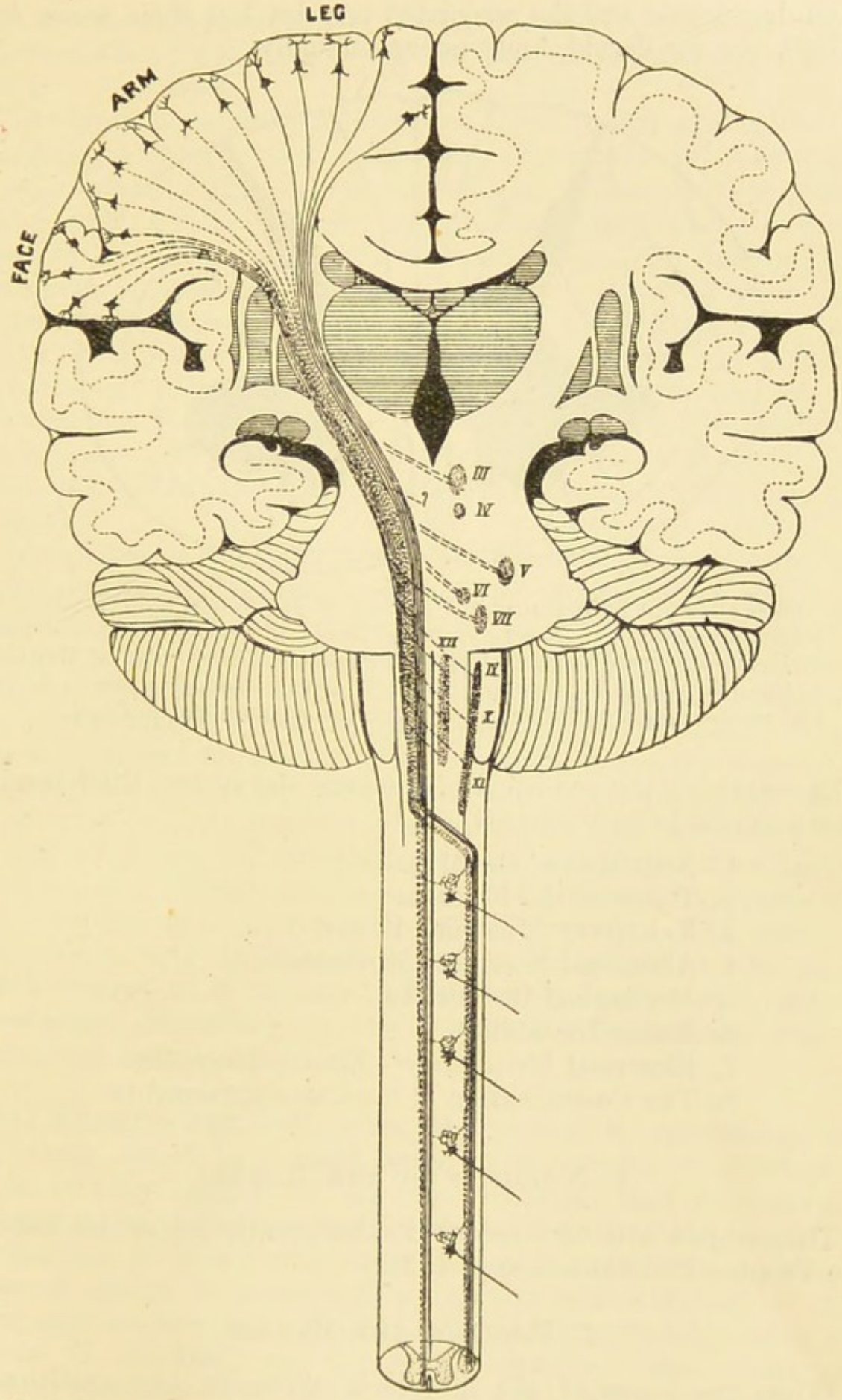


FIG. 182.—Diagram of the motor path from the right side of the brain (after Van Gehuchten (modified)).

lesion degenerate and the associated muscles lose their tonus (see p. 389), become flaccid, and undergo atrophy.

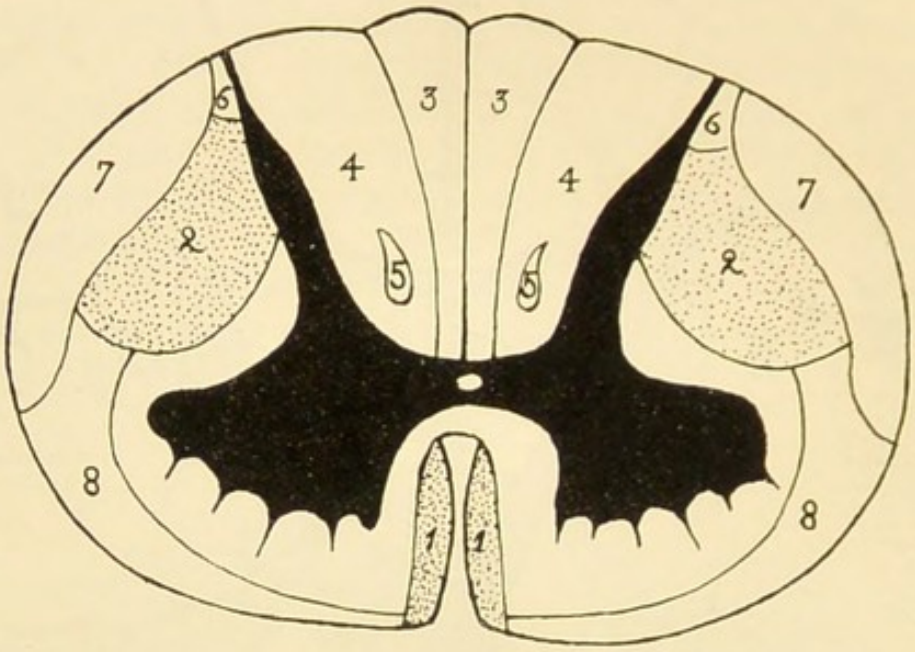


FIG. 183.—Diagram of a transverse section of the spinal cord to show the principal tracts. 1, Direct pyramidal tract; 2, Crossed pyramidal tract; 3, Postero-internal tract of Goll; 4, Postero-external tract of Burdach; 5, Comma tract; 6, Lissauer's tract; 7, Direct cerebellar tract; 8, Ventral cerebellar tract of Gowers. (After Van Gehuchten, modified.)

In examining the condition of the muscular system the following points should be investigated :—

1. Nutrition of the Muscles.
2. Tonus of the Muscles.
3. Voluntary Muscular Power.
4. Abnormal Muscular Movements.
5. Mechanical Irritability.
6. Reflex Irritability.
7. Electrical Irritability—Electro-diagnosis.
8. The Co-ordination of muscular movements.

1. NUTRITION OF THE MUSCLES

This subject will be considered subsequently under the head of the Trophic Functions (see p. 422).

2. TONUS OF THE MUSCLES

When the tonus of the muscle is excessive, the condition is termed **hypertonus**; when it is decreased, we speak of the condition as **hypotonus**. When hypertonus is pronounced the

affected muscles, examined by the hand, are tense, rigid, or spastic, whereas when hypotonus is well marked the muscles are soft and flaccid to the touch.

The tonus of muscles is, however, more readily appreciated by ascertaining the degree of passive mobility at the different joints; in other words, by ascertaining the degree of resistance to passive movements. When there is hypertonus or spasticity the resistance is increased; when there is hypotonus or flaccidity the resistance is decreased. When the tonus of the muscles is being tested, the patient's attention must be diverted from the part under examination, as otherwise active resistance may simulate hypertonus. The intimate connection between the tonus of the muscles and the tendon reflexes results in increase of the latter when there is hypertonus, and in their decrease or absence when there is hypotonus. This subject will be again referred to (see p. 404).

Hypotonus or flaccidity of the muscles occurs when there is a lesion destroying the continuity of the spinal reflex arc, as in peripheral neuritis, tabes dorsalis, and acute anterior poliomyelitis.

Hypertonus occurs as the result of two changes: firstly, impairment or destruction of the fibres of the upper motor neurone in the pyramidal tracts which normally control the reflexes. Hypertonus is therefore an important sign of secondary descending degeneration of the pyramidal tracts, the result of cerebral disease or of an incomplete transverse lesion of the cord above the level of the reflex arc, *e.g.* myelitis, pressure paraplegia, disseminated sclerosis, etc. It also occurs in cases of primary degeneration of this neurone, as in amyotrophic lateral sclerosis. And secondly, hypertonus may be due to increased irritability of the spinal reflex centres, as in tetanus and strychnine poisoning. Special phenomena depending on the existence of hypertonus may be mentioned here.

(1) **Kernig's Sign.**—If, when the patient is recumbent, with the thigh flexed to a right angle to the trunk, an attempt is made to fully extend the leg on the thigh that movement is prevented by reflex contraction of the flexors of the leg. The explanation of this phenomenon, which is present in nearly all cases of acute leptomeningitis, is that inflammation of the meninges renders the spinal roots unduly irritable, and the flexion of the thigh on the trunk stretches those roots and thereby increases their irritability, so that reflex contraction of the flexors of the leg occurs when an attempt is made to passively extend the leg.

(2) **The Sign of Flexion of the Thigh on the Trunk.**—To elicit this sign the patient, who is lying on his back with his lower limbs extended, is directed to raise himself to a sitting posture without the aid of his arms. In health, and in functional paralysis of one lower limb, neither limb is raised from the couch. In paralysis of one limb, due to organic lesion of the pyramidal fibres—*e.g.* in hemiplegia—the affected limb is raised from the couch when the patient sits up or attempts to do so.

(3) **The Tibialis Phenomenon of Strümpell** consists in contraction of the tibialis anticus on flexion of the leg. It is a sign of organic lesion of the pyramidal tract.

Contracture.—When the passive mobility of a joint is markedly decreased as a result of hypertonus of the muscles, there is a condition of *active contracture.*

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 by the physician without causing pain, and when left alone it springs back quickly. This contracture 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 Muscular Power

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).

To examine the patient's voluntary motor power, it is necessary to cause him to go through a variety of voluntary movements, simple and combined, standing, walking, etc. The dynamometer (Fig. 184) 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.

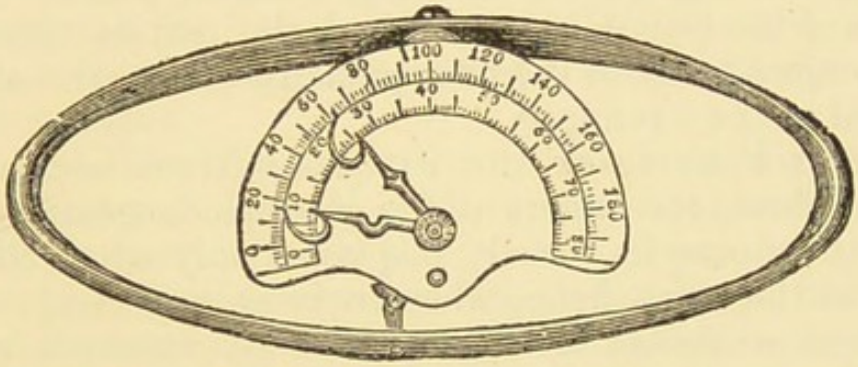


Fig. 184.—Dynamometer.

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 more 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.

Apparent defect of motor power may be simulated by painful affections of the joints, muscles, etc., by ankylosis of joints, or by affections of co-ordination (see p. 418).

It is often necessary to make a systematic examination of individual muscles and groups of muscles; the patient is made to perform various movements while the physician offers passive resistance thereto.

Interossei and Lumbricles.—These muscles abduct and adduct the fingers, flex them at the metacarpo-phalangeal joints and extend them at the inter-phalangeal joints. When these muscles are paralysed, those movements cannot be performed, and there results a characteristic deformity of the hand, the **claw hand** or

main en griffe, in which the first phalanges are hyper-extended, the more distal phalanges flexed, in consequence of unopposed action of the extensor communis digitorum and the long flexors of the fingers. This deformity occurs in cases of paralysis of the ulnar nerve, Klumpke's paralysis (lesion of the eighth cervical and first dorsal nerve roots), and diseases of the spinal cord involving the anterior horns of the eighth cervical and first dorsal segments (progressive muscular atrophy, syringomyelia, etc.).

Muscles of the Thenar Eminence.—The opponens pollicis and abductor brevis pollicis are to be tested by directing the patient to touch the tip of the ring and little fingers with the thumb, without flexing the fingers at the inter-phalangeal joints. When the adductors of the thumb are paralysed, the patient cannot firmly grasp an object between the first phalanx of the thumb and the metacarpal of the forefinger.

The Long Flexors of the Fingers.—These are tested when the patient flexes the fingers at the inter-phalangeal joints. The action of these muscles is fully displayed only when the hand is extended at the wrist, hence when there is wrist-drop there may be apparent weakness of the long flexors, which, however, disappears when the hand is passively extended at the wrist.

The Long Extensors of the Fingers.—When these muscles, which normally extend the fingers at the metacarpo-phalangeal joints, are paralysed, extension at those joints cannot be performed, and because of the flexed position of the fingers, full abduction of the fingers by the interossei cannot be performed until the fingers have been passively extended.

The Flexors of the Wrist are tested when the patient, having the forearm supinated, flexes the hand towards the forearm.

The Extensors of the Wrist.—When these muscles are paralysed there is **wrist-drop**, and the patient cannot perform extension (dorsiflexion) at the wrist joint, *e.g.* as the result of paralysis of the musculo-spiral nerve.

The Pronators and Supinators of the hand and forearm are to be tested while the forearm is semi-flexed and the upper arm is being grasped by the physician.

The Contraction of the Biceps can be both seen and felt when the patient flexes the forearm against resistance. The contraction of the Supinator longus can also be seen and felt at the radial side of the bend of the elbow, when the patient flexes the pronated forearm against resistance.

The Triceps is tested by the physician grasping the patient's forearm, flexing it, and offering passive resistance to its extension.

PECTORAL MUSCLES.—The patient stretches out the arms horizontally in front and presses the palms of the hands firmly against each other, the physician meanwhile noting the contraction of the pectorals at the anterior folds of the axillæ, and the ease or difficulty with which he can separate the hands from each other.

DELTOID.—The physician places one hand over the muscle and directs the patient to elevate the arm to the level of the shoulder. If the muscle be paralysed it is neither seen nor felt to contract.

SERRATUS MAGNUS.—The patient stretches out the arm horizontally in front and pushes forwards against resistance. When the serratus magnus is paralysed the digitations of that muscle in the axilla are not seen, but there is marked projection of the scapula, and especially of its vertebral border, from the thorax, and the scapula as a whole does not move forwards as does the scapula on the unaffected side.

TRAPEZIUS.—The middle portion of the trapezius is tested by asking the patient to elevate or shrug the shoulder against resistance. When that portion of the muscle is paralysed, the point of the shoulder droops and lies further forward than the unaffected shoulder; the scapula lies obliquely with its lower angle approximated to the vertebral column; elevation of the arm above the horizontal is defective.

The LATISSIMUS DORSI is tested (*a*) by estimating the resistance the patient offers to passive elevation of the arm; (*b*) by elevating the patient's arm and asking him to depress it against resistance; (*c*) by grasping the posterior axillary fold between the fore-finger and thumb and feeling the contraction of the muscle when the patient coughs.

MUSCLES OF THE LOWER LIMBS.—The extensors and flexors of the joints are tested by directing the patient to perform movements of flexion and extension at the joints of the toes, ankle, knee, and hip. The PERONEAL MUSCLES are tested by asking the patient to evert the foot; the adductors of the thigh, by abducting at the hip and asking the patient to adduct the limb; the abductors of the thigh, by crossing the limb over its fellow and directing the patient to abduct the limb. Passive resistance is in each instance offered to the movement.

To test the STERNO-MASTOID, ask the patient to turn the head towards the opposite shoulder.

The ERECTOR SPINÆ is tested by directing the patient to raise the trunk from the stooping posture. Bilateral weakness of the erector spinæ results in lordosis when the patient stands or walks; the lordosis disappears when the patient lies down; when he sits

he obviates the tendency for the trunk to fall forwards by resting the arms on some support.

Abdominal Muscles.—Weakness of those muscles also causes lordosis; when the patient lies on his back there is inability to rise into the sitting posture without the aid of the arms.

The Facial Muscles (see p. 14) and the ocular muscles (p. 362) have been already considered.

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.*

Paralysis is of two varieties—organic and functional. The former is the result of some interruption in the motor tract (upper or lower motor neurone), or of some disease of the muscles. 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.

I. Functional Paralysis is usually distinguishable without much difficulty by the facts that there are no symptoms pointing to organic disease, *e.g.* no muscular atrophy, no indications of trophic changes in skin or bone, no alterations of electrical reactions (*vide infra*), and no ophthalmoscopic changes. There are usually present other indications of hysteria, such as ovarian tenderness, hemianæsthesia, convulsions, and psychical changes.

On the other hand, there may be signs which conclusively indicate that the paralysis is organic and not functional. Of such signs we may mention the extensor response in the great toe (p. 401), the sign of flexion of the thigh on the trunk (p. 390), and the Argyll-Robertson pupil (p. 368).

II. Organic Paralysis may be the result of a lesion in the upper or in the lower motor neurone.

(*a*) *Paralysis from Lesion in the Upper Motor Neurone.*—The paralysis is usually more or less general in distribution, involving all the muscles of the limb affected. The tonus of the paralysed muscles is increased, the tendon reflexes are exaggerated, and clonus is often present, *i.e.* there is a spastic paralysis. The superficial reflexes are usually present, rarely absent, Babinski's sign (the extensor response in the great toe) is present; there is rarely atrophy of the paralysed muscles, and there is no

qualitative change in the electrical reactions—*i.e.* no reaction of degeneration.

Lesions of the cortical motor region (Fig. 181) give rise to various monoplegias, according to the portion of the cortex affected, involvement of the lower part of the precentral gyrus leading to facio-lingual monoplegia, of the middle portion to brachial monoplegia, and of the upper portion to crural monoplegia. These are usually accompanied by indications of irritation, such as Jacksonian epilepsy, less frequently by choreiform movements and athetosis, and by anæsthesia of the paralysed part.

When the lesion affects the pyramidal fibres in the posterior part of the internal capsule there is complete hemiplegia, often accompanied by hemianæsthesia. Affections of the corpus striatum and optic thalamus may indirectly occasion hemiplegia. Lesion of the crus causes hemiplegia, to which a crossed oculomotor paralysis is often added. That of the lower 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 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.

(*b*) *Paralysis from Lesion in the Lower Motor Neurone, or Peripheral Paralysis.*—The paralysis is not diffuse, but affects individual muscles or groups of muscles. The reflex arc being destroyed by the lesion, there is hypotonus of the affected muscles, and the tendon reflexes are lost, *i.e.* there is a flaccid paralysis; the superficial reflexes are abolished; the affected muscles rapidly atrophy, and exhibit, as a rule, the reaction of degeneration (see p. 415).

When such peripheral paralysis is due to lesion of the 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 a peripheral nerve, there is, as a rule, considerable pain, and the muscles affected are, in many cases, all those supplied by the nerve in question.

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 (see p. 398).

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 tetanus and tetany, in meningitis (causing head retraction or cervical opisthotonos), 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 *Myotonia congenita* (*Thomsen's Disease*), the characteristic feature of which is that the patient is unable immediately to make a voluntary muscular movement, the muscles associated with the desired movement and their antagonists being thrown into a state of slight tonic spasm. Once, however, the movement commences, the spasm disappears.

In the great majority of cases, convulsions are due to some irritation of the cerebral cortex. In the so-called idiopathic epilepsy we may assume that the Betz cells possess a certain degree of hyper-excitability which leads them to ready explosive discharge. The proximate cause in most of these cases is probably toxic, the result, for example, of gastro-intestinal disorders. In other instances the evidence of toxic action is more obvious, as in the convulsions of uræmia.

That variety of epilepsy which is known as Jacksonian, and which is often a mono- or a hemi-spasm without loss of consciousness, is indicative of a gross irritative lesion of the cerebral cortex on the side opposite that on which the spasms occur. These mono-spasms are of a very considerable diagnostic value, because from irritation of a cortical motor centre, the convulsion begins in some special group of muscles, or even in one particular muscle, and subsequently, from radiation of irritation to other cortical centres (see Fig. 181), involves other groups of muscles or becomes a generalised convulsion. The site of the primary irritation may thus be localised on the cortex.

In a few instances the starting-point of the irritation may be peripheral, from an old cicatrix, for example. The convulsions which may occur in children in association with intestinal worms may arise in this manner, or, on the other hand, they may be explained by the assumption of a toxic cause.

The convulsions of tetanus and strychnine poisoning are probably of spinal origin.

(*b*) **Tremor.**—Muscular tremor, or involuntary oscillation of any part of the body, 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 sometimes 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 other instances, tremor may result from a deficient motor impulse.

In investigating cases of tremor, note should be made of the particular parts of the body affected, of the rapidity, rhythm, and amplitude 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 during volitional movement (**intention tremor**), or persists when the body is at rest. In special cases it may be well to record the movements by means of a graphic method.

Intention tremor is conspicuously seen in cases of multiple sclerosis, and is well elicited while the patient raises the hand to the mouth. In paralysis agitans the tremor is most marked in the hands, is of slow rate (four to six oscillations per second), and often simulates the movement of rolling a cigarette between the fingers and thumb. The tremor is present during repose, and tends to decrease during the performance of a volitional movement. Tremor also occurs as a result of poisoning by alcohol, mercury, tobacco, etc., and is observed in persons under treatment for the morphia habit, and in senility, hysteria, and exophthalmic goitre.

(*c*) **Fibrillary Twitching.**—This differs essentially from tremor, and 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 those cases of muscular degeneration and atrophy which result from a lesion of the anterior horn of the spinal cord, *e.g.* progressive muscular atrophy and bulbar paralysis. Fibrillary twitchings are not observed in idiopathic muscular atrophy (muscular dystrophy).

(*d*) **Choreic Movements.**—The movements which are characteristic of chorea are well marked. They consist in involuntary, 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, choreiform movements may be observed on one side of the body.

(*e*) **Athetosis.**—This rare symptom is characterised by involuntary 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 usually unilateral, occurring in cases of cerebral palsy in children or hemiplegia in adults. Athetosis sometimes occurs as a bilateral congenital affection.

Besides these abnormal forms of muscular movement there are one or two others of great rarity, such as the **co-ordinated spasms** (hysterical laughter, saltatory spasm, etc.), 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 (see p. 419).

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 condition, **myoidema**, 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 occipito-frontalis 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, fasciæ, or periosteum (deep reflexes), or, finally, may consist in the changes of the pupil caused by light, etc.

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 arc 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. **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 mucous membrane of the larynx, and certain reflexes connected with the bladder and rectum, which have been discussed already (see p. 383).

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.—When this reflex is to be examined, the patient should lie supine with the lower limb rotated outwards and semi-flexed at the hip and ankle, so as to ensure muscular

relaxation. The skin of the foot must be warm and dry. When the observer gently scratches the plantar region, especially on its outer aspect, there is in health a "flexor response"—a simultaneous flexion of the toes on the metatarsus, and the foot may be simultaneously dorsiflexed and inverted at the ankle.

The *extensor response* or *Babinski's sign* on stimulation of the plantar region is characterised by a slow deliberate extension of the great toe. This may be followed by extension of the other toes, the tensor fasciæ femoris does not contract early as it does in health, and there is lessened dorsiflexion at the ankle. The extensor response indicates a lesion of the pyramidal fibres and is not observed in functional paralysis. The extensor response is probably dependent on relative hypertonus in certain muscles, including the extensors of the toes.

The normal plantar reflex in infants is characterised by brisk extension and abduction of the toes, with eversion of the foot and dorsiflexion of the ankle. This "infantile response" can hardly be confounded with the extensor response of adult patients.

The spinal segments concerned in the reflex arc for the plantar reflex are the 1st and 2nd sacral.

(2) Gluteal Reflex.—Tickling the skin of the buttock determines in many persons a contraction of the gluteal muscles: the spinal segments concerned are the 4th and 5th lumbar.

(3) Cremasteric Reflex.—Tickling of the skin on the inner aspect of the thigh is followed by drawing up of the testicle; the spinal segments concerned are the 1st and 2nd lumbar. When the skin of the scrotum is stroked, or, better, touched with ice it shrivels from contraction of the muscular fibres of the dartos. This is known as the scrotal reflex. It depends on the integrity of the 2nd lumbar segment.

(4) Abdominal Reflex.—On stroking the skin of the abdomen, from the costal margins towards the iliac crests, the abdominal muscles contract: the spinal segments concerned are those from the 8th to the 12th dorsal.

(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; the segments concerned are those from the 4th to the 6th dorsal. 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 interscapular region gives rise to contraction of the scapular muscles;

the 4th and 5th cervical and the 1st dorsal segments are those concerned.

Diagnostic Value of the Superficial Reflexes.—The plantar reflex has been already considered. As regards the other superficial reflexes, they are increased—(1) when the cerebral restraining influence is lessened, as sometimes happens in cases of paralysis due to a lesion of the upper motor neurone; and (2) when the grey substance of the nerve centres is unusually excitable, as in cases of strychnine poisoning, tetanus, etc. Diminution or absence of the superficial reflexes results either from interference with the integrity of the reflex arc in question (disease of nerves, spinal nerve roots, white or grey substance of the cord) or from increase in the cerebral restraining influence. This latter condition arises in cases of an irritative cerebral lesion, and is often of value in distinguishing organic from purely functional hemiplegia.

3. **Deep Reflexes.**—These are tested by tapping over tendons or quickly rendering them tense, and consist in the rapid contraction of the muscle, which is thus put on the stretch. Deep reflexes may also be elicited by tapping over periosteum. The tendon-jerks are possibly not true reflexes, yet they are dependent on the integrity of a reflex arc. Evidence of the reflex character of these jerks is furnished by comparing the ankle, knee, wrist, and elbow jerks, for James has pointed out that the rapidity of their production is governed by the distance which lies between the individual muscle and its spinal centre, that is, by the length of its reflex arc.

When any of the tendon-jerks are to be tested, the patient must completely relax the muscles of the limb, which is to be placed in such a position that the muscle or muscles in question are passively stretched to a slight degree. A tap is then given, preferably with a percussion hammer, over the tendon, and the contraction of the muscle is looked for. The movement of the limb resulting from the muscular contraction affords less delicate and reliable evidence of the reflex.

If the jerk appear to be absent, an attempt must again be made to elicit it whilst "**reinforcement**" is employed; for example, when the knee jerk is being tested the patient looks upwards, and, with the fingers of each hand interlaced, pulls the hands strongly as if to separate them. Other additional means may be employed to divert the patient's attention from the part which is being examined.

The chief deep reflexes are—

(1) The Knee Jerk.—This may be tested in a variety of ways.

If the patient sits on a chair, the limb, flexed at the knee, is either crossed over the opposite limb or is supported by the observer, so that the leg hangs down loosely. A tap is then given over the patellar tendon, and the contraction of the quadriceps femoris, and especially of the vastus internus, is to be looked for and felt; so, too, is the consequent jerk forwards of the leg. Or the jerk may be tested while the patient, sitting on a chair, advances the legs as far as possible, the heel and sole of each foot being yet in contact with the ground; or while he sits on a table so that the legs are dependent. If the contraction be doubtfully present it may be elicited when reinforcement is employed, as already described. If the patient be in bed, he is to lie on his back; we then slightly flex the limb at the knee and tap over the patellar tendon.

The spinal segments concerned are the 2nd, 3rd, and 4th lumbar. Although varying in activity in different persons, and sometimes difficult to elicit, the knee jerk is constantly present in health, and is equally active on the two sides.

Increased activity of the knee jerk is manfested when there is excessive response to a slight tap, or when the single muscular contraction of health is replaced by clonic spasm (*Knee clonus* or *Patellar clonus*). Knee clonus may be best elicited by grasping the patella between the thumb and forefinger and pushing it suddenly downwards towards the foot: the clonic spasm of the quadriceps femoris continues until the fingers release the patella.

(2) Tendo-Achillis-Jerk (5th lumbar and 1st sacral segments).—The tendo-Achillis is tapped while the patient is kneeling on a chair with his feet dependent over the edge. A brisk plantar flexion, equal on the two sides, is observed in health. If the patient be in bed, let him turn on one side and flex the knees; we then grasp the foot, dorsiflex it, and tap the tendo-Achillis. Reinforcement may be necessary.

The jerk is constantly present in health up to the age of fifty years.

If the Achilles jerk be exaggerated, clonic spasm of the calf muscles may be induced by tapping on the tendo-Achillis in the manner described. This phenomenon—*Ankle clonus*—is, however, best elicited by slightly flexing the knee, suddenly dorsiflexing the foot, and maintaining the pressure on the sole. This results in clonic contractions of the calf-muscles, which recur with great regularity, usually about six or seven times per second. James has shown that the rate varies with the height of the individual and the consequent length of the reflex arc. The clonus continues until the pressure on the sole is relaxed.

(3) Adductor Jerk.—Abduct the limb and tap upon the inner condyle of the femur or the tendon of the adductor magnus. A contraction of the adductors follows. The response is not constant, even in health.

(4) Jaw Jerk.—The patient having opened his mouth slightly, a tap on a tongue depressor laid on the teeth of the lower jaw, or on a finger laid on the chin, causes an upward movement of the lower jaw. This jerk is not constantly present in healthy persons.

(5) Triceps Jerk (7th and 8th cervical segments).—Support the upper arm so as to let the forearm hang dependent, and tap with the percussion hammer upon the triceps tendon just above the olecranon.

(6) Biceps Jerk (5th and 6th cervical segments).—While the patient's arm is semi-flexed at the elbow and the dorsum of his hand rests against the physician's chest, the latter grasps the elbow and strikes upon his thumb, which is laid on the biceps tendon just above the bend of the elbow.

(7) Supinator Jerk (5th and 6th cervical segments).—The patient's forearm is supported in a semi-flexed and semi-pronated position, and a tap is administered to the radius a little above its styloid process. The consequent contraction of the supinator longus may cause a movement of flexion of the forearm.

The triceps, biceps, and supinator jerks are not constantly present in healthy individuals, and their absence is not, therefore, a sign of disease.

Diagnostic Value of the Deep Reflexes.—The Achilles jerks and the knee jerks are constantly present in health; the other jerks mentioned above are not constant. We have to note—

I. The presence of a normally active deep reflex indicates the integrity of the reflex arc in question.

II. The deep reflexes are increased—(*a*) in cases where there is increased excitability of the motor cells in the grey matter of the cord, as in strychnine poisoning and in tetanus. Probably to the same cause may be ascribed the increase sometimes observed in cases of phthisis, and occasionally in the early stage of 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 also increased (*b*) when the restraining cerebral influence is withdrawn, as in cases of lesion of the pyramidal fibres, and in some cases of hysteria and neurasthenia.

III. Diminution or abolition of the deep reflexes occurs when

the reflex arc is interrupted. Thus any lesion which affects the peripheral nerves (as multiple neuritis), the posterior roots (*e.g.* tabes dorsalis and Friedreich's ataxia), the anterior horns of the cord (*e.g.* acute anterior poliomyelitis, or progressive muscular atrophy), or the anterior roots, will produce this effect. The deep reflexes are, moreover, diminished in muscular dystrophy, and are abolished in shock and coma, and in the hypotonic muscles in cases of *total* transverse lesion of the spinal cord, as from a fracture dislocation of the vertebræ.

It is of great practical importance to remember that the Achilles jerks and knee jerks are lost at an early stage of tabes dorsalis, and that in this disease the abolition of the Achilles jerks usually precedes that of the knee jerks.

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, etc. 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 with a solution of salt and water. 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. 185 to 191, which are derived from von Ziemssen's work, the more important of these are given.

EXPLANATION OF FIG. 185.

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. Stylo-hyoid 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. Trapezius muscle.
15. Sterno-mastoid muscle.
16. Levator auguli scapulæ muscle.
17. Posterior thoracic nerve (rhomboid muscles).
18. Phrenic nerve.
19. Omo-hyoid muscle.
20. Posterior 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. Triangularis menti muscle.
37. Cervical branch of facial nerve.
38. Branch to platysma muscle.
39. Sterno-hyoid muscle.
40. Omo-hyoid muscle.
41. Sterno-thyroid muscle.
42. Sterno-hyoid muscle.

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[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.

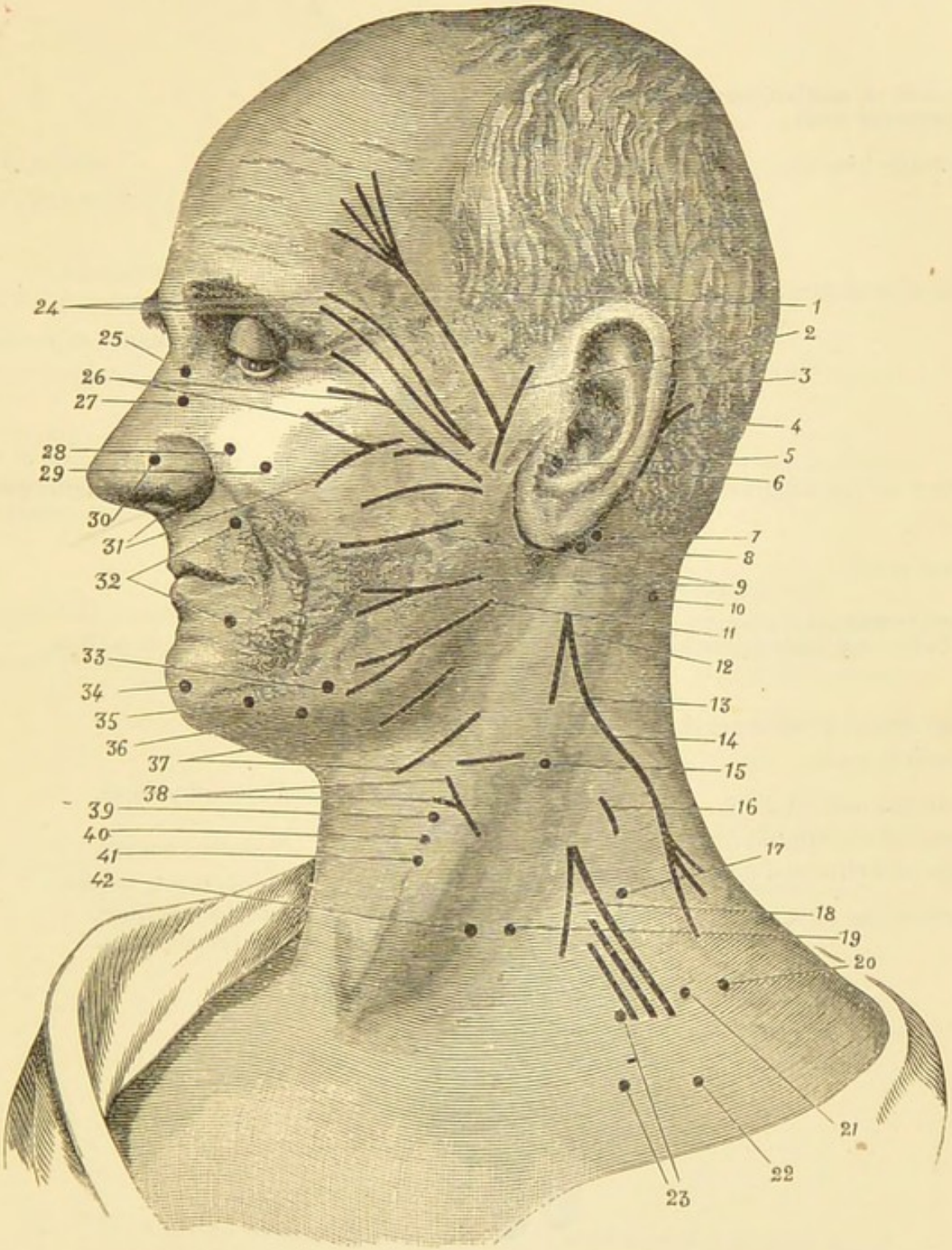


FIG. 185.—Motor points of head and neck (Ziemssen).

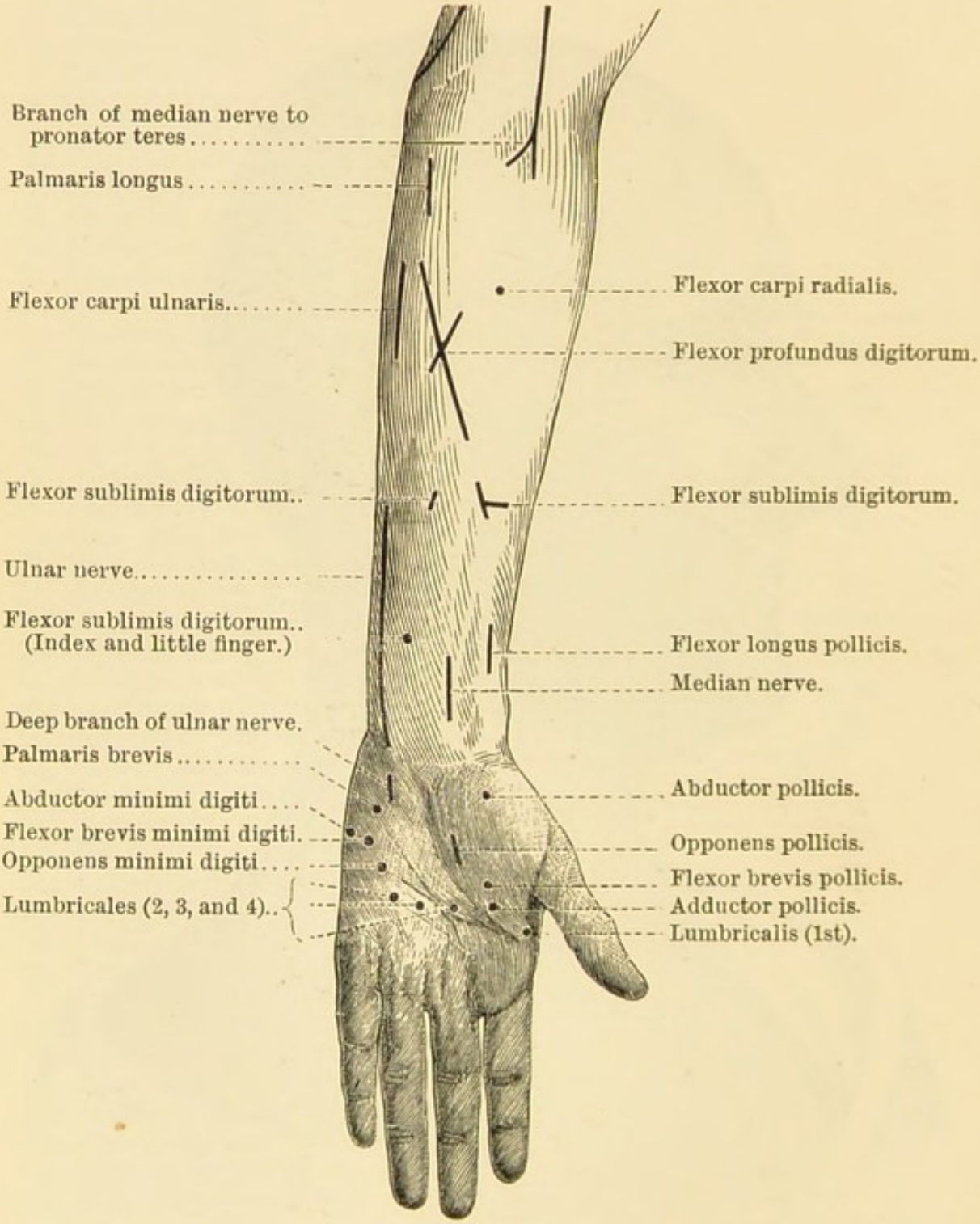


FIG. 186.—Motor points on flexor surface of forearm (Ziemssen).

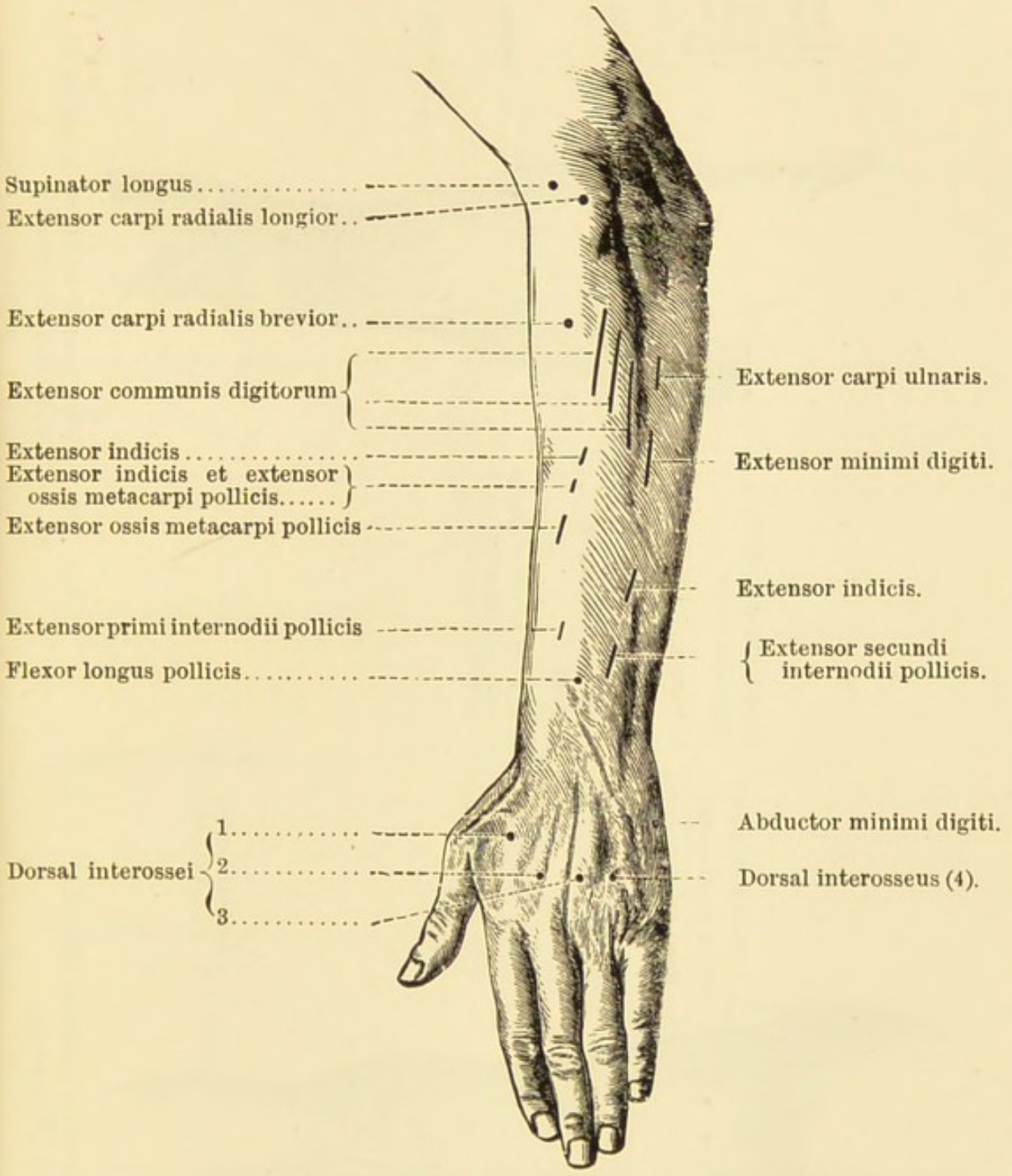


FIG. 187.—Motor points on extensor surface of forearm (Ziemssen).

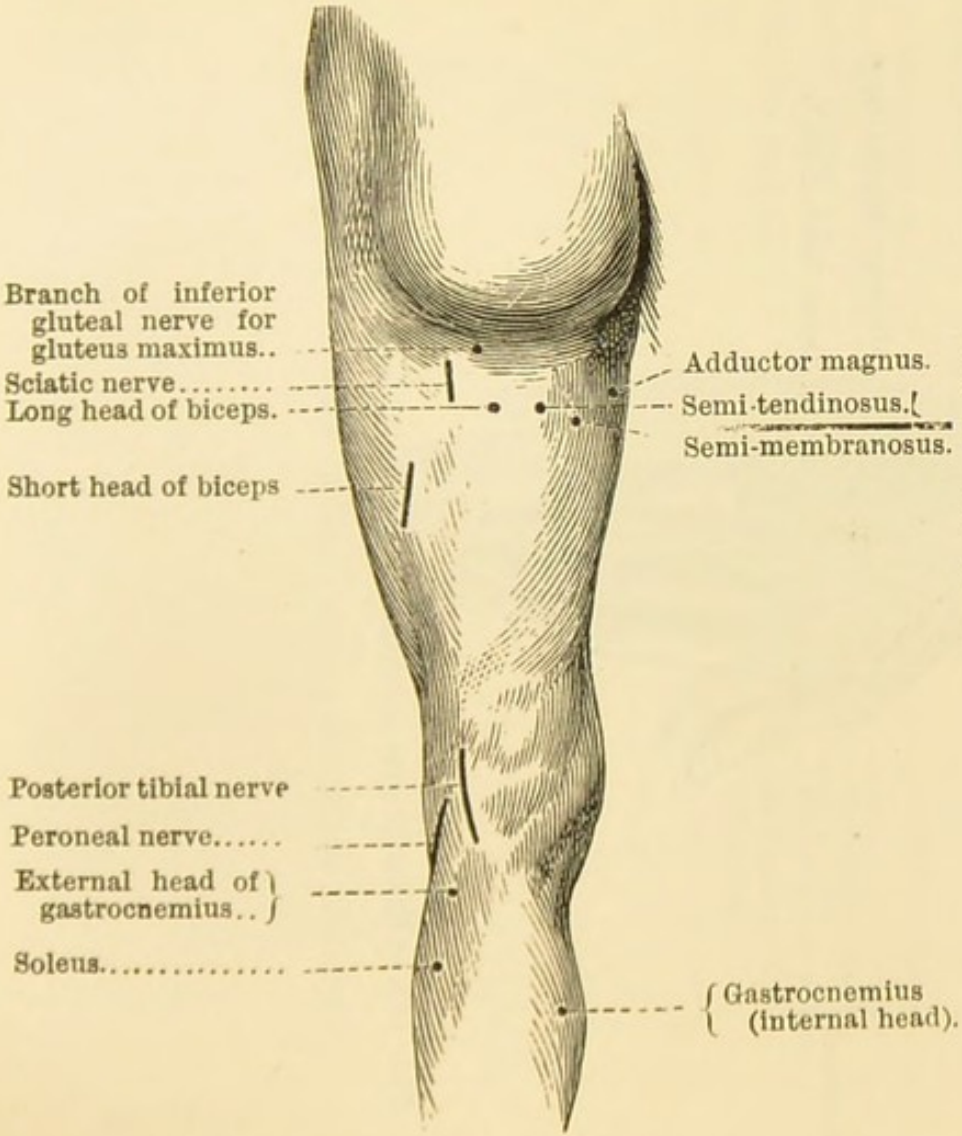


FIG. 188.—Motor points on posterior aspect of lower limb (Ziemssen).

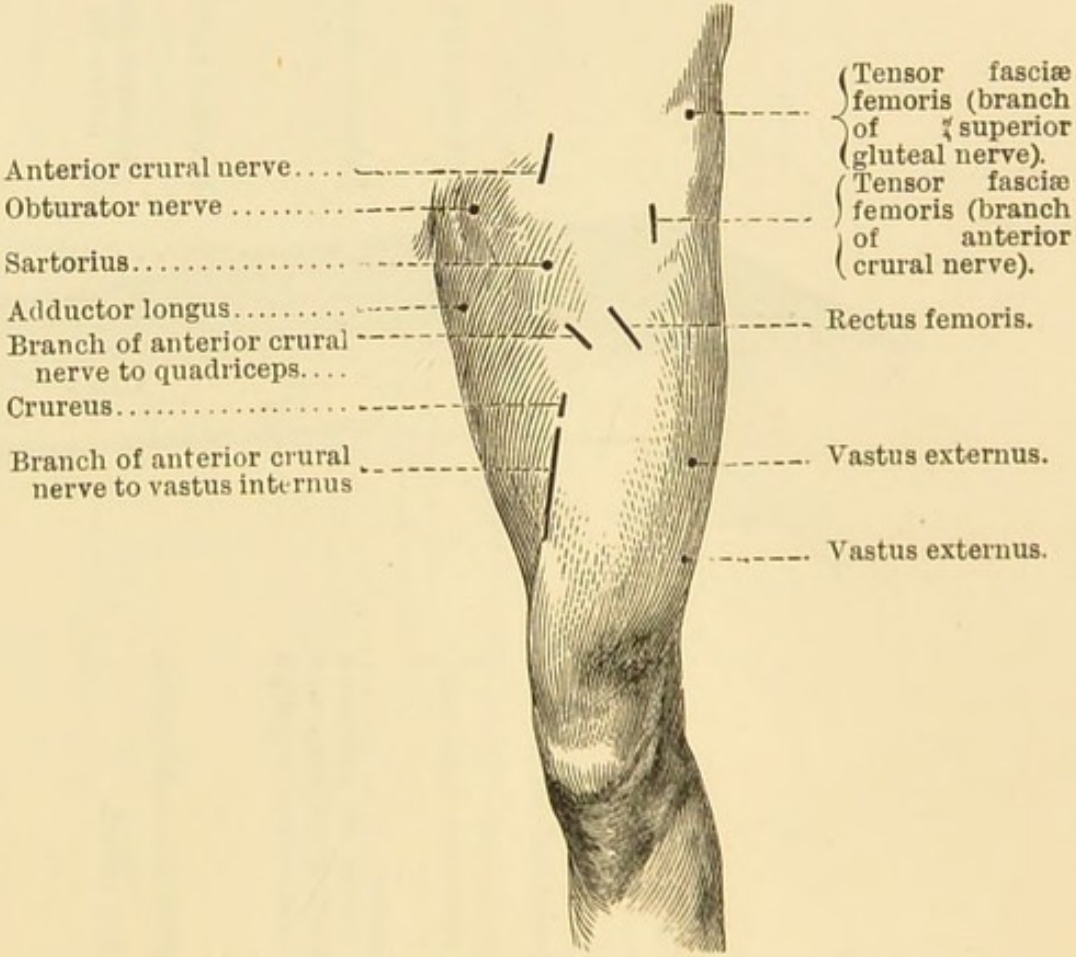


FIG. 189.—Motor points on anterior aspect of lower limb (Ziemssen).

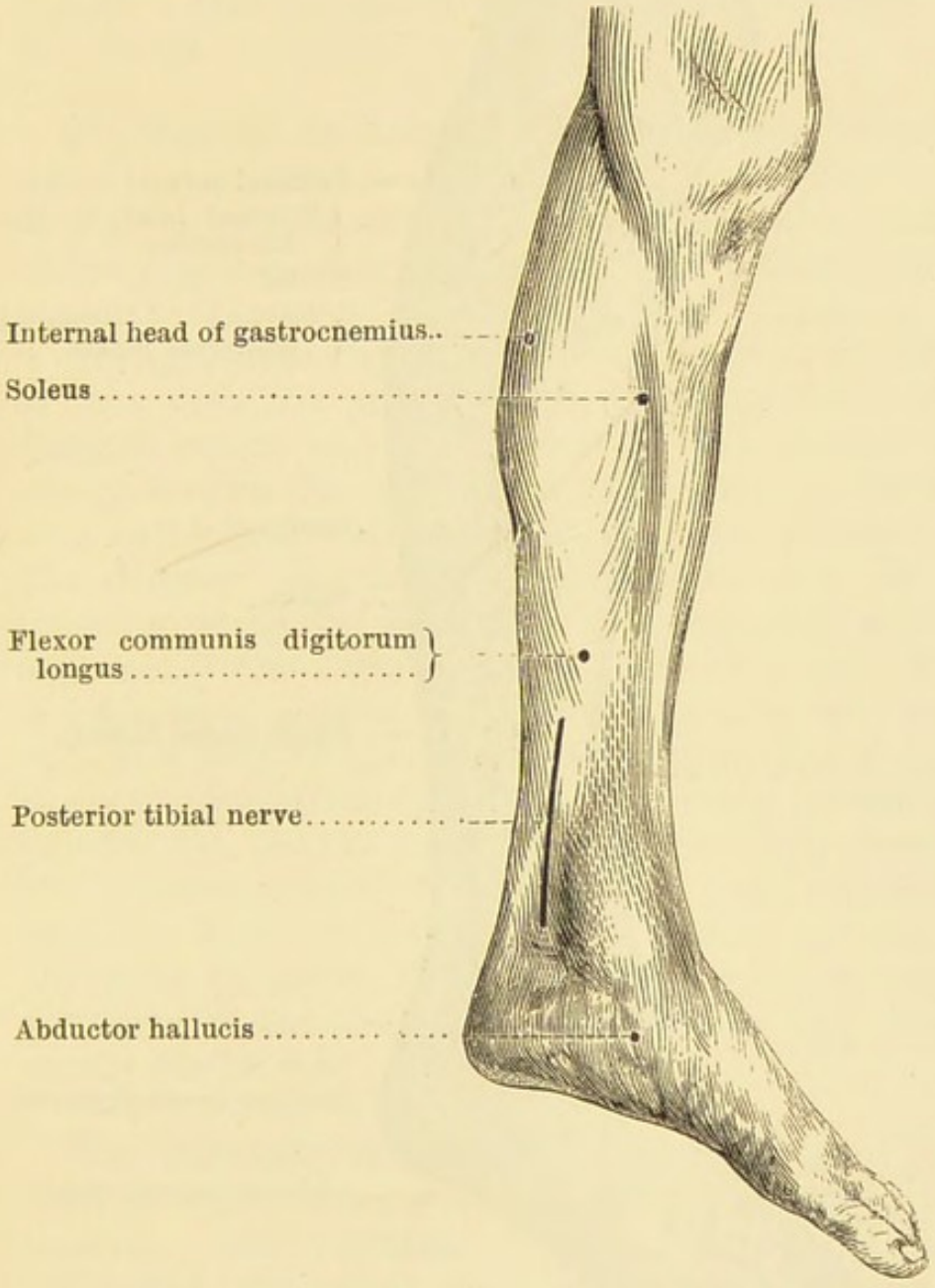


FIG. 190.—Motor points on inner surface of lower limb (Ziemssen).

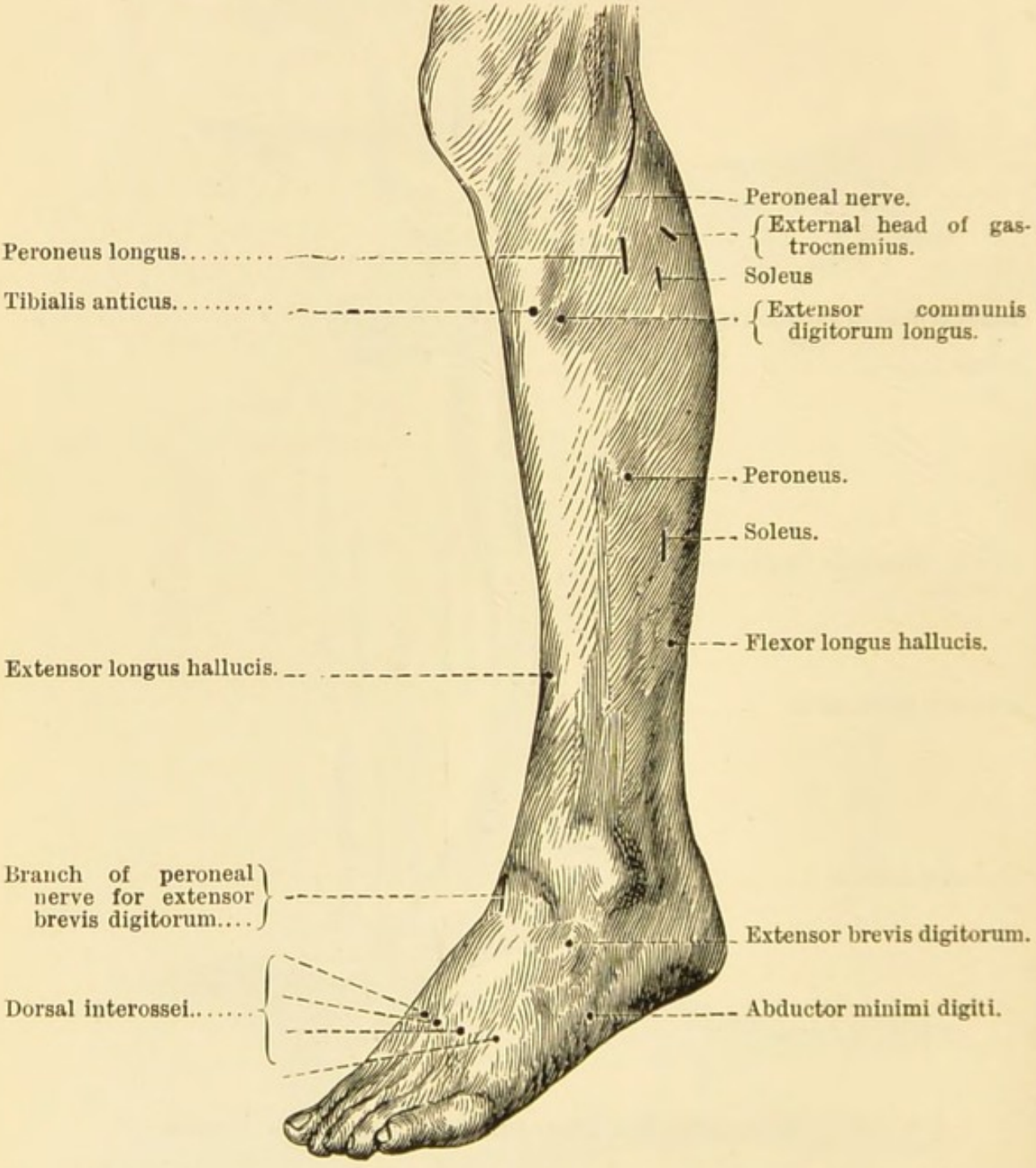


FIG. 191.—Motor points on outer surface of lower limb (Ziemssen).

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 excitability 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 mm.

The Galvanic or Continuous Current only gives rise to contraction 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 K.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 closing contraction,—the small c indicating that the contraction is slight.

Negative pole.—Strong contraction on closing the current, expressed by the formula K.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 K.C.T., cathodal closing tetanus, and K.O.c., cathodal opening slight contraction.

Various forms of paralysis may be accurately classified, as Erb has shown, by means of the electrical reactions of the muscles and nerves, as follows:—

1. *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).

[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.

2. *Quantitative Change in the Electric Excitability*—

(*a*) *Simple Increase.*—This condition is uncommon, but is sometimes found in the first stage of hemiplegia, and rarely and transitorily in peripheral paralysis at its commencement. It is characteristic of tetany.

(*b*) *Simple Diminution* occurs in all diseases which lead to simple muscular atrophy, such as muscular dystrophy and the like. It also occurs in muscles which have atrophied from inaction.

3. *Quantitative and Qualitative Changes* ("**Reaction of Degeneration**").—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 show the differences between rapid and slow recovery (Figs. 192 and 193).

The Clinical Significance of the Reaction of Degeneration is that it occurs in all organic paralyses depending on disease of the anterior horns of the cord, or of the corresponding nuclei of cranial nerves, of the anterior nerve roots—in fact, of any part

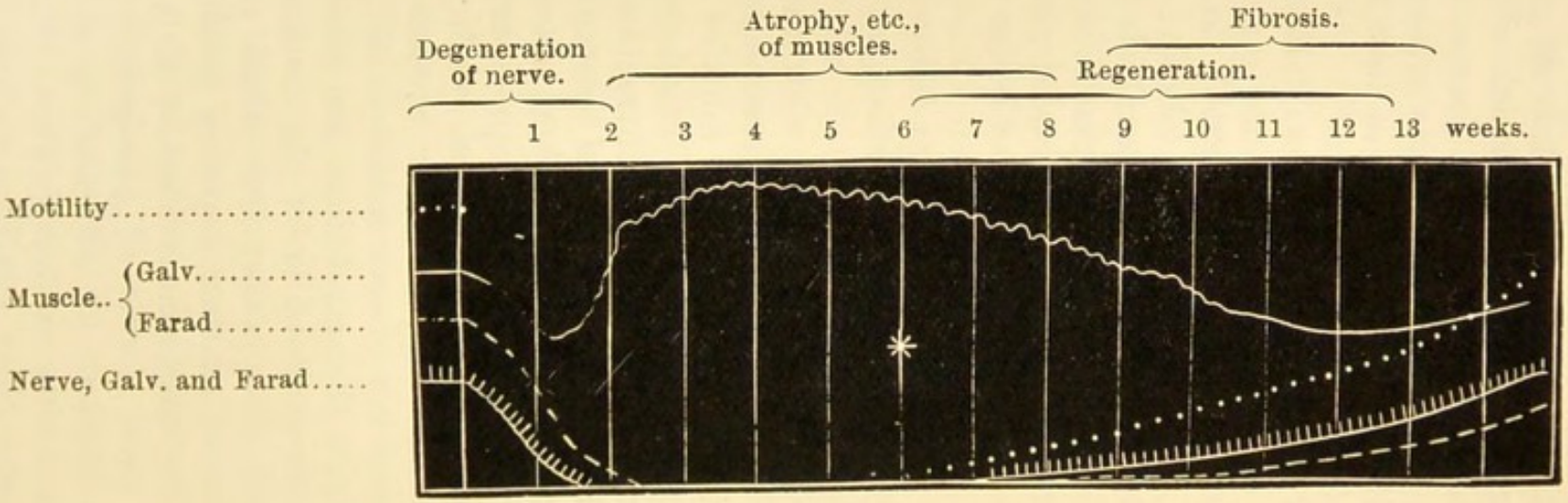
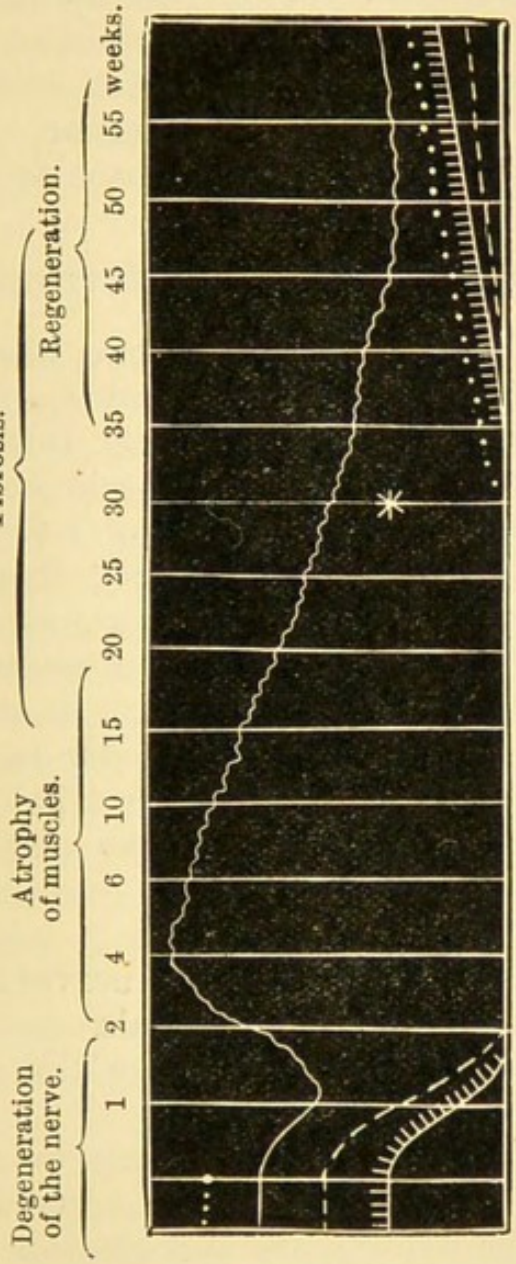


FIG. 192.—Reaction of degeneration in mild peripheral lesion, with rapid recovery (Erb).



* Return of voluntary motor power.

FIG. 193.—Reaction of degeneration in severe peripheral lesion, with slow recovery (Erb).

of the lower motor neurone. It is not met with in paralysis of the upper motor neurone. 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 organic paralysis of the upper motor neurone, in hysterical paralysis, or in paralysis due to idiopathic muscular atrophy.

8. Co-ordination of Muscular Movements

By a co-ordinated movement is meant one which fully and exactly fulfils its purpose without any undue expenditure of nervous or muscular energy. To arrive at this precision of result it is necessary that certain muscles should be set in action in a certain order and with a certain degree of force. No action is performed by means of one muscle alone; there are always others which come into play either by contraction or relaxation. The centres concerned in the co-ordination of muscular movements lie mainly in three places—in the spinal cord, in the cerebellum and various nuclei in its vicinity, and finally in the cerebral cortex. The chief ways in which those centres gain information as shall enable co-ordinated movements to be performed are as follows:—

1. The optic nerve.
2. The vestibular branch of the eighth nerve bringing impressions from the semicircular canals.
3. The neurones conveying "muscular sense" impressions from the periphery (see p. 354).

Of these, the optic nerve gives impressions as to the direction, degree, and rapidity of a movement. The vestibular neurones give information as to the movements and position of the head, and in sitting, standing and walking, of the body generally, such information being derived from the end-organs in the semicircular canals. The neurones conveying muscular sense impressions give information as to the position of the limbs, the degree of flexion of the joints, the tension of muscles. For the acts of standing and walking the healthy man does not require to use optical impressions; the vestibular and muscular sense impressions are sufficient. It is only when one of these is impaired that the help of the eyes is required for muscular co-ordination.

When there is a lesion at any point in the course of the neurones conveying centripetal impressions of muscular sense (peripheral nerves, posterior nerve roots, fibres in the posterior columns of the cord, etc.) the centres do not know with accuracy

the momentary position of the limbs either at the commencement or during the progress of a movement. Hence they cannot appreciate the distance through which a limb has to be moved in order to attain the object of the movement, and hence come exaggerated and defective movements, or the various muscles do not pass into contraction in the exact order which is necessary, or the stimulus passes to the wrong muscles. In any case the movement is no longer a co-ordinated one, there is muscular inco-ordination or ataxia.

To test the power of co-ordination the patient is instructed to perform certain movements while the eyes are closed; to touch the tip of the nose with the forefinger, or one knee with the heel of the other limb, to walk along a straight line on the floor, describe a circle on the floor with the great toe, etc. When an ataxic patient stands with his feet together, he may stand fairly steadily so long as his eyes are open, but he will at once sway, or even fall, if they are closed (**Romberg's sign**). If the ataxia be extreme, the patient may be unable to maintain the standing posture even when the eyes are open.

The gait of an ataxic patient, if the legs be affected, is distinctive. It is well seen in cases of tabes dorsalis, where the patient walks with a broad base, the feet being kept far apart, the excursions of the lower limbs are needlessly great, and the heel is brought to the ground with a sudden stamp. The patient's gaze is directed to the ground, and he experiences special difficulty in turning.

As has been said, lesions of sensory nerves, of posterior nerve roots, of the posterior columns of the cord and their continuations in the medulla, pons, etc., may cause ataxia. Thus arises the inco-ordination of tabes dorsalis, of Friedreich's ataxia, of bulbar disease, and generally of ataxias dependent on disturbance of muscular sense. Diseases affecting the co-ordinating centres may also cause ataxia, as we sometimes see in cases of cortical lesion.

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. 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, etc., 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 (see p. 363), and in Ménière'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, etc., are rather to be referred to the trophic nerves.

1. **Cutaneous Vaso-motor Affections.** — Diffused paleness or redness of the skin may be seen in persons in perfect health (blushing, etc.), 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 amyl nitrite.

In fever, vaso-motor affections of the skin may usually be observed. In the cold and sweating stages of malaria, for example, there is a 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 diseases of the nervous system (epilepsy, tuberculous meningitis, etc.) 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 cérébrales*, 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.

2. **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, etc.). The secretions are sometimes affected from such causes. The urine, in particular, is liable to well-marked quantitative and qualitative changes; injury of the fourth 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 XXXII

Nervous System (*continued*)

TROPHIC FUNCTIONS OF THE NERVOUS SYSTEM

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 long inaction from whatever cause. In cases of chronic arthritis, the wasting of the muscles which act on the diseased joint is sometimes ascribed not to inaction alone, but is regarded as a "reflex atrophy."

Apart from these conditions, in which the atrophied muscles do not exhibit the reaction of degeneration, muscular atrophy may be due to disease of the nerve cells in the anterior horns of the cord or corresponding regions in the medulla, to affections of those nerve fibres which connect these cells with the affected muscles, or to disease primarily of the muscles themselves (myopathy or muscular dystrophy). In distinguishing a myopathic atrophy from muscular atrophy of nuclear origin, it must be remembered that the former is a family disease, other members of the family being affected, that the disease appears insidiously in early life, that the muscles mainly affected are usually those of the arm, shoulder, or hip, and that fibrillary twitchings and the reaction of degeneration are not present.

True muscular hypertrophy, as a pathological condition, is exceedingly rare, but it is seen in myotonia congenita (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 fibrous and adipose tissue, which is seen in the pseudo-hypertrophic variety of myopathy, although even here a few muscle fibres may be hypertrophied.

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. Among the best known are the affections occurring in tabes dorsalis and syringomyelia. The affection may be acute articular swelling (arthropathy) closely resembling rheumatoid arthritis, or chronic degenerative changes, leading to great deformities and strong predisposition to fractures. The important points to note in regard to these joint affections (Charcot's joints) are that they are sudden in onset, and are not accompanied by fever or pain, and that the joints most frequently affected are the knee and hip in tabes dorsalis, the shoulder, elbow, and wrist in syringomyelia.

III. Affections of the Skin.—Various eruptions, such as erythema, urticaria, eczema, herpes, may arise as the result of nervous disease. In herpes zoster, in which disease there is hæmorrhage and inflammation, probably of infective origin, in the ganglia of the posterior nerve roots, the eruption is distributed over an area supplied by one or more posterior nerve roots (see Plates V. and VI.). The affection of the skin of the fingers, termed "glossy skin," is also of trophic origin. Still more important, as belonging to this category, are the acute and chronic bed-sores, which are so common and so troublesome in bed-ridden cases of transverse myelitis and pressure paraplegia, and the perforating ulcers of the foot in tabes dorsalis. And, finally, there have to be noted the occurrence of abnormal pigmentation, and of affections of hair, nails, and cutaneous secretory apparatus, the bullæ, ulcers and necroses of syringomyelia, the 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 XXXIII

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 the late stages of meningitis and cerebral tumour, in epilepsy (usually accompanied with convulsions), in hysterical attacks, in catalepsy, in uræmia, in diabetic coma, 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, bacterial toxines in acute infective diseases, 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 XXXIV

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 and reception of spoken and written language (aphasia), and second, with the anomalies of articulation.

Aphasia

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 in another centre the sensory memories of the appearance of words written and printed, with their interpretation; and eventually the child learns how to express thought in written speech, though the motor-writing centre is probably not a true speech centre.

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 centres have been determined with fair accuracy. These are indicated in Fig. 194, where the centres are marked as follows:—

A. The auditory-speech 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 impulses of speech. This centre, which is that originally described by Broca, corresponds to the foot of the third frontal and the foot of the ascending frontal convolution.

C. The visual-speech 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 gyrus and adjacent part of the supra-marginal convolution.

D. The graphic-motor-speech centre or motor-writing centre.—Many believe that there is no separate motor-writing centre apart from the cortical centres for the hand and wrist, but it seems reasonable to conclude that that portion of the intermediate-precentral area which lies anterior to the centre for the right hand (see Fig. 181, p. 386) contains the motor cortical mechanism for written speech.

These centres are all very closely associated with each other by means of connecting and commissural fibres.

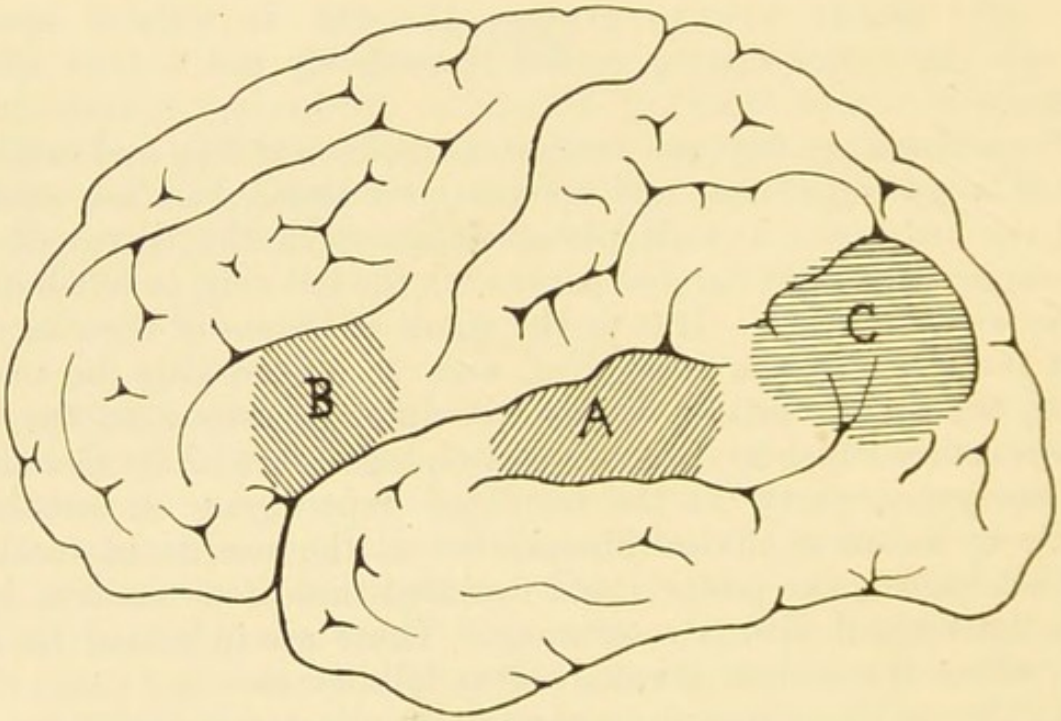


FIG. 194.—Cortical speech centres.

The term **Aphasia**, in its wider sense, includes all defects or loss of capacity for expressing propositions in speech or writing, or for comprehending spoken or written speech, the derangement being due to an affection of the higher cerebral centres concerned with speech, and not to defect of intelligence or articulation nor to disease of the eye or ear.

There are two main forms of aphasia—*motor* and *sensory*. Of each of these there are two varieties: (*a*) *Cortical*, when the lesion is in the speech centre, and (*b*) *Subcortical*, when the lesion involves, not the centre, but the fibres passing either towards a sensory or from a motor-speech centre.

1. AUDITORY APHASIA or WORD DEAFNESS.—The patient hears sounds well; but he is "word deaf," that is, the words of spoken

language are perceived by him merely as sounds, which he can no longer understand, and he is in the position, as it were, of one who in a foreign land does not understand the language he hears.

(*a*) *Subcortical Word Deafness.*—Due to a lesion at 1; Fig. 195. There is word deafness, and consequently inability to repeat spoken words and to write from dictation. But as the auditory-speech centre is intact, the patient is able to write spontaneously and to comprehend and intelligently copy written speech.

(*b*) *Cortical Word Deafness.*—Due to a lesion at A in Figs. 194 and 195. The effects of such a lesion are twofold, according as the centre is viewed from the receptive or from the emissive standpoint. The memories of word-sounds are gone, and therefore the

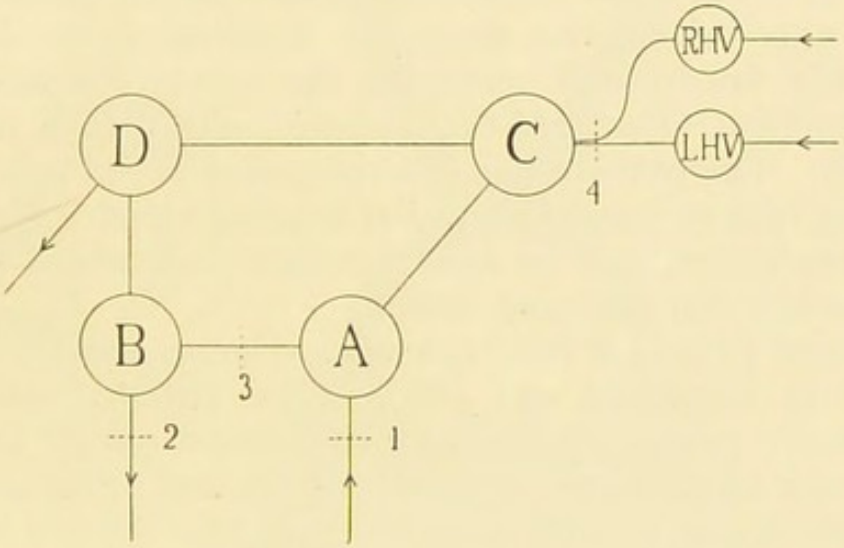


FIG. 195.—Scheme of the cortical speech centres. A, the auditory-speech centre; B, the motor-speech centre; C, the visual-speech centre; D, the motor-writing centre; LHV, the left half-vision centre; RHV, the right half-vision centre.

patient is "word deaf," and he cannot correctly repeat spoken language nor write from dictation. And even if the visual-speech centre be intact, the power of understanding written speech is usually impaired, for the visual-speech centre requires the aid of the auditory-speech 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 auditory-speech centre 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 auditory-speech centre, 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.

2. Motor Aphasia or Aphemia.—The patient has lost more or less completely the power of vocal speech:—he cannot speak spontaneously, repeat spoken language, nor read aloud. But his ability to comprehend spoken and written speech is little if at all impaired. 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.

(*a*) *Subcortical* (*Pure*) *Motor Aphasia.*—Due to a lesion at 2 in Fig. 195. The patient has lost the power of voluntary speech, of repeating spoken language, and of reading aloud. But internal speech is unaffected, and he can therefore understand all that is said to him and can read and write.

(*b*) *Cortical* (*True*) *Motor Aphasia.*—Due to a lesion at Broca's centre (B in Figs. 194 and 195). The patient cannot speak spontaneously, repeat spoken language, read aloud, write spontaneously, nor write from dictation. His power of comprehending written speech is usually impaired; so too, though to a lesser degree, is the power of understanding spoken language.

Conduction Aphasia.—The fibres which connect the auditory-speech and the motor-speech centres run under the Fissure of Sylvius and the island of Reil, and when they are destroyed by a lesion situated there (at 3, Fig. 195), a variety of aphasia is produced which has been called conduction aphasia. Here the auditory-speech centre is uninjured, therefore there is no word-deafness, the patient understanding all that is said to him. The motor-speech centre 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,—showing therefore the phenomenon of *paraphasia*.

3. Visual Aphasia, Alexia, or Word Blindness.—The patient can see well, but can no longer read; in some instances he cannot recognise even single letters.

(*a*) *Subcortical* (*Pure*) *Word Blindness.*—Due to a lesion cutting the fibres between the visual-speech centre in the angular gyrus and the two half-vision centres in the occipital lobes (at X, Fig. 196;

4, Fig. 195). The patient can no longer read. He can write spontaneously and from dictation, but can only mechanically copy

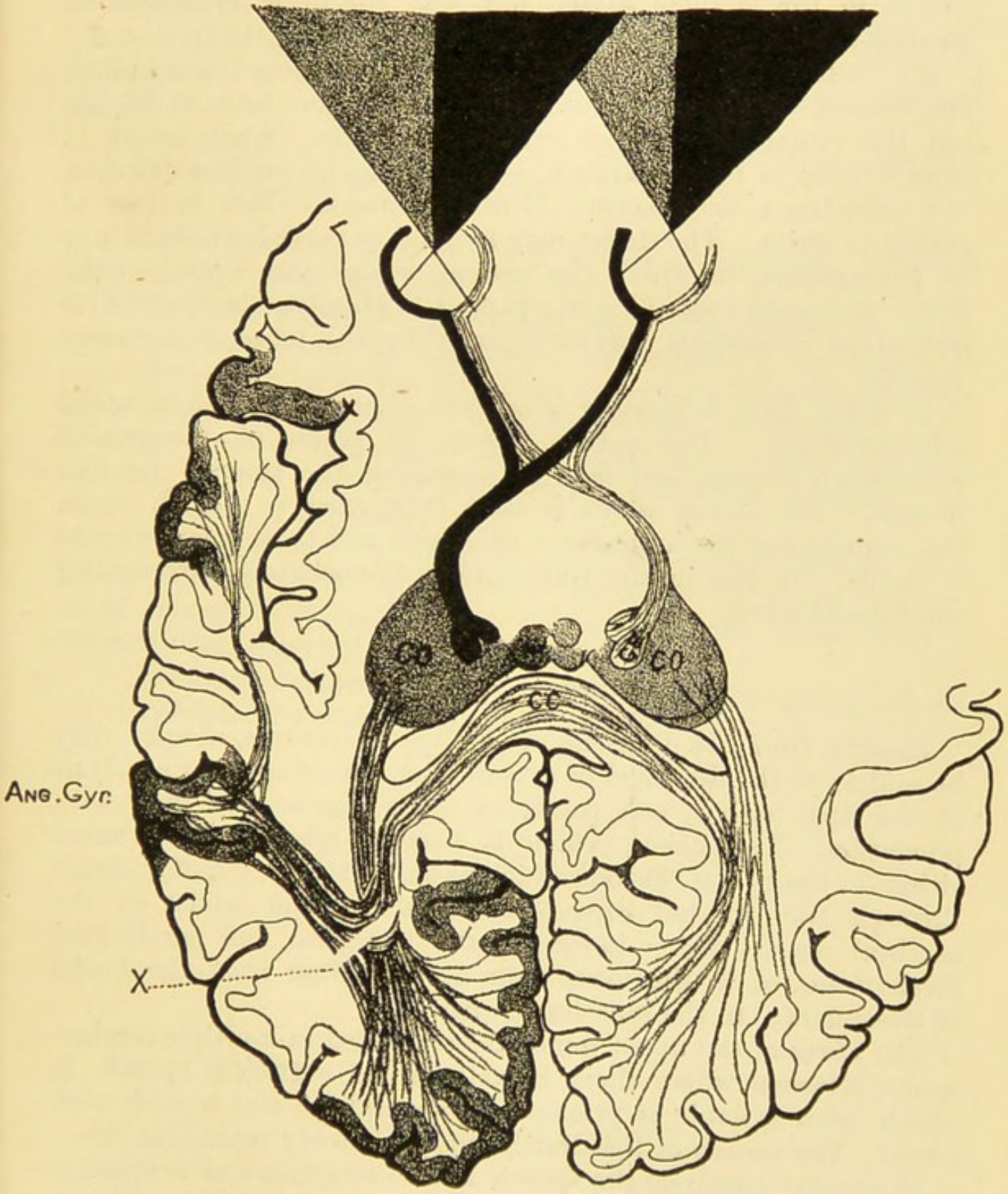


Fig. 196.—Course of the optic fibres (after Dejerine).

printed or written speech, in the same manner as he would copy a drawing. There is no defect in the production of motor speech,

nor in the understanding of spoken speech. Right homonymous hemianopsia is nearly always present, for the lesion destroys not only the fibres passing from the left and the right cuneus to the centre in the angular gyrus, but also the optic radiations of Gratiolet which pass to the left cuneus (see Fig. 196).

(*b*) *Cortical (True) Word Blindness.*—The patient is word blind, and cannot write spontaneously or from dictation, because he has lost the visual memories of letters and words. When asked to copy written or printed speech, he does so as he would a drawing.

4. Agraphia or Graphic Motor Aphasia.—This is loss of ability to write. The defect may be total or partial, or there may be paragraphia, in which the patient writes the wrong words. Even in complete agraphia the patient is usually able to write his own name. Agraphia may be observed in both motor and sensory aphasia.

In some cases a peculiar condition is present which is called *mind-blindness.* The patient loses the power of recognising well-known objects, and fails to remember the faces of familiar friends. The centre which is here implicated is that in which the memories of the appearance of objects and their interpretation is stored. It lies in the convolutions immediately surrounding the calcarine area.

Disorders of Articulation

Passing from the disorders of the speech centres, a word may be said as to the disorders of articulation, resulting from paralysis of the lips, tongue, soft palate or other organs concerned with phonation. The lesion, which is usually bilateral, is situated either in the upper neurone 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 the lower neurone, extending from the nuclei of these nerves to the muscles in question.

The fibres of the upper neurone are affected in multiple cerebro-spinal sclerosis where there is well-marked **scanning speech**, in which each syllable is pronounced separately, slowly and with effort. 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. To detect **slurring speech**, the patient should be told to say "British Constitution," "Hippopotamus," "Royal Irish Constabulary," etc.

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, etc., with consequent wasting, and impede articulation to so marked an extent that finally the patient may not be able to articulate at all. Similar defect of articulation is observed in pseudo-bulbar paralysis of cerebral origin.

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, etc.); 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 XXXV

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. 240). One of the most common alterations in the size and shape of the cranium is that of *chronic hydrocephalus*, where the skull becomes enlarged, more or less globular, the frontal bone prominent, the sutures open, and the fontanelles large and pulsating. The defective ossification in *rickets* is well seen in the cranium, the anterior fontanelle not closing during the second year of life.

On palpating the skull, localised depressions may be found, the result of old depressed fracture, which, in the case of epilepsy, give valuable indications. The thin bones of *cranio-tabes* 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 disease, as cerebral tumour, mastoid inflammation, etc.

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 (*Scoliosis*) will readily be detected. At the same time, any antero-posterior curvature will be apparent, whether it be posterior curvature without vertebral caries, as in rickets, old age, etc., or the angular curvature of Pott's disease, or finally the exaggeration of the normal lumbar curve (*lordosis*).

The mobility of the spinal column must also be noted on inspection. The patient, standing with his feet together, stoops forwards and bends from side to side. Rigidity of the vertebral column and pain on movement may thus be elicited.

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. Tenderness of the vertebræ may also be elicited by pressing upon the patient's shoulders while he is standing upright.

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, etc.

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 Röntgen-rays in investigating the spine often proves to be of diagnostic value.

CHAPTER XXXVI

CLINICAL BACTERIOLOGY

The recognition of micro-organisms in the blood, pathological fluids, sputum, etc., is of great importance in clinical medicine, from the standpoint not only of diagnosis, but also of prognosis and treatment. In many instances simple microscopic examination of suitably stained preparations, as of sputum, enables us to ascertain the presence of bacteria, and to form a fairly accurate diagnosis of the species present. As a rule, however, bacteria cannot be identified by microscopic examination alone, and they must then be cultivated on nutrient media and a study made of the cultures. In some instances the experimental inoculation of animals is required for diagnosis.

METHODS OF PREPARING AND STAINING FILMS

Preparation of Films.—Films may be prepared either on cover-glasses or slides; the former are preferable and must be perfectly clean and dry. The cover-glasses (No. 1, $\frac{3}{4}$ inch square) are therefore kept in a well-stoppered jar of alcohol and ether; one is removed with forceps, dried, polished with a soft linen cloth, and laid on a sheet of note or filter paper.

By means of a platinum loop, allowed to cool after being sterilised in a Bunsen flame, a single loopful of the fluid to be examined is transferred to the surface of the cover-glass, and there spread into an even, thin film by means of the loop. If the material be of more solid consistence, it is well, in order to obtain an even film, to place on the cover-glass first one or two loopfuls of distilled water, and then a single loopful of the material, which can now be rubbed up with the water to form a thin, even film. Films are allowed to dry in the air.

Fixation.—When the film has dried, the cover-glass, held film side upwards in a pair of forceps, is passed three times through

the Bunsen flame in order to fix the film to the glass. The rate at which the preparation is passed through the flame is equal to about one foot per second. If it passes too slowly, the film will be charred; if too rapidly, it will not be fixed to the glass.

Staining.—A single basic aniline stain is sufficient in some instances. In others it is advisable to employ a differential stain, or some special methods to demonstrate spores, capsules, etc.

The aniline stains most frequently used are fuchsin, gentian-violet, and methylene-blue. Saturated solutions of these stains in absolute alcohol should be kept in small bottles, which are fitted with pipette stoppers. For use, we dilute the stain, taking 1 part of the alcoholic solution to 10 of distilled water. The staining may be performed in watch-glasses, or the fluids may be dropped from pipettes on to the film while the cover-glass is held in a pair of Cornet's forceps. The former method is the cleaner.

Method of Staining Films with a Single Stain.—The film, dried and fixed, is treated with the diluted stain for fifteen to thirty seconds, washed in water, allowed to dry in the air, then passed three times through the Bunsen flame in the manner described for the fixation of films, and finally mounted in xylol balsam.

Gram's Method of Staining.—An aniline-water gentian-violet solution is first prepared. Shake up in a test tube 4 parts of aniline (aniline oil) and 100 of distilled water. It is sufficiently accurate if 2 or 3 drops of aniline oil are shaken up in a test tube half full of distilled water. The fluid is then passed through a filter paper which has been moistened with water, and to the aniline-water, thus prepared, add a tenth part of saturated alcoholic solution of gentian-violet. The method of staining is as follows:—

1. Stain the film in freshly prepared aniline-water gentian-violet for three to five minutes.
2. Transfer it directly to iodine and potassium iodide solution (iodine, 1 grm.; potassium iodide, 2 grm.; distilled water, 300 c.c.) for two to three minutes.
3. Wash in absolute alcohol until no more gentian-violet can be removed from the film (usually for one to two minutes).
4. Counterstain in dilute fuchsin solution.
5. Wash in water; then allow the film to dry in the air.
6. Pass the cover-glass three times through the flame.
7. Mount in xylol balsam.

An aqueous solution of carbolic acid (1 in 20) may be substituted for the aniline-water.

Gram's method is of great value, and should always be employed whenever films of pus, pleural fluid, urine, etc., are to be examined for bacteria. The chief value of the method consists in the fact that certain bacteria retain the gentian-violet when treated by Gram's method, *i.e.* they are Gram positive; whereas other bacteria, pus and tissue cells do not retain the gentian-violet, *i.e.* are Gram negative. Thus in preparations treated as described, Gram positive bacteria are of dark purple tint, Gram negative bacteria are counterstained red.

Of the common pathogenetic bacteria, those which are Gram positive are staphylococci, streptococci, pneumococci, *B. diphtheriæ*, tetanus bacillus, tubercle bacillus and streptothrices. Those which are Gram negative are the gonococcus, *Micrococcus meningitidis cerebrospinalis*, *Micrococcus catarrhalis*, *Micrococcus melitensis*, *B. typhosus*, *Bacillus coli communis*, influenza bacillus, *B. pneumoniæ*, *B. pestis*, *B. mallei* (glanders), the bacilli of dysentery, and the vibrio or spirillum of Asiatic cholera.

Differential Stains for Acid-Proof Bacteria.—

(*a*) *Ziehl-Neelsen Method* (modified).—

1. Stain the dried and fixed film in hot (steaming) carbol-fuchsin (fuchsin, 1 grm.; 5 per cent. aqueous solution of carbolic acid, 100 c.c.; absolute alcohol, 10 c.c.) for three to five minutes. The stain is heated, until it steams, by means of a small Bunsen flame.
2. Decolorise in 25 per cent. aqueous solution of sulphuric acid for two minutes.
3. Wash in absolute alcohol for two minutes, or longer, until the film has lost all trace of red colour.
4. Counterstain in dilute methylene-blue solution.
5. Wash in water, dry in the air, pass three times through the flame, and mount in xylol balsam.

(*b*) *Weichselbaum's method.*—

1. Stain the dried and fixed film in steaming carbol-fuchsin for three to five minutes.
2. Transfer it directly to a watch-glass full of concentrated solution of methylene-blue in absolute alcohol. Here it remains for five to ten minutes.
3. The film is washed in water, dried in the air, passed thrice through the flame, and mounted in xylol balsam.

Tubercle bacilli and certain other bacteria resist decolorisation with acid and alcohol, and are stained red, whereas all other

bacteria and tissue cells, not being acid-proof, take on the blue counterstain (Plate VIII., 4).

Differential Staining of Spores.—

1. Stain the dried and fixed film with steaming carbol-fuchsin for three to five minutes.
2. Decolorise in 3 per cent. hydrochloric acid alcohol (HCl, 3 c.c.; absolute alcohol, 100 c.c.) for one minute.
3. Wash in water.
4. Counterstain with dilute methylene-blue solution.
5. Wash in water, dry in the air, pass thrice through the flame, and mount in xylol balsam.

Spores retain the red stain; the bodies of the bacilli, having been decolorised by the acid-alcohol, are stained blue.

Staining of Bacterial Capsules.—The capsules of bacteria may frequently be stained when the film is treated with a single basic aniline stain. Of methods of differential staining, one of the best is

Richard Muir's Method.—A special mordant is used—namely, 20 per cent. aqueous solution of tannic acid, 2 parts; saturated aqueous solution of mercuric chloride, 2 parts; saturated aqueous solution of potash alum, 5 parts. A thin film is spread and dried over the Bunsen flame. Then

1. Filter on the mordant, and leave it for two minutes.
2. Wash thoroughly in water, then in methylated spirit, and again in water.
3. Stain in steaming carbol-fuchsin for two to three minutes.
4. Wash in water.
5. Filter on the mordant, and leave it for two minutes.
6. Wash in water, in methylated spirit, and again in water.
7. Stain in saturated aqueous solution of methylene-blue for two minutes.
8. Wash in water.
9. Differentiate with methylated spirit.
10. Dehydrate in absolute alcohol, clear in xylol, and mount in xylol balsam.

The method is suitable for demonstrating pneumococci and their capsules in sero-fibrinous exudates. The bodies of the pneumococci are stained bright red, their capsules are blue.

Neisser's Method of staining Metachromatic Granules.—

1. Stain the dried and fixed film for ten to fifteen seconds in the following fluid:—Methylene-blue, 1 grm; absolute

alcohol, 20 c.c. ; distilled water, 950 c.c. ; glacial acetic acid, 50 c.c.

2. Wash in water.
3. Counterstain for ten seconds with 2 per cent. aqueous solution of Bismarck brown.
4. Wash in water, dry in the air, pass thrice through the flame, and mount in xylol balsam.

The bodies of bacteria are stained brown. Metachromatic granules are stained blue. Thus the *B. diphtheriæ* appears brown, with a blue granule usually at each pole (see Plate VIII., 5).

When bacteria are to be examined with the microscope, a $\frac{1}{12}$-inch oil-immersion objective and a No. 4 ocular should be used. Light is reflected from the *plane* mirror through the condenser, which must be in position beneath the stage. A drop of special cedar oil (refractive index, 1·51) is placed on the cover-glass, and the tube of the microscope lowered until the objective is seen to touch the drop of oil. The observer now looks down the microscope, and first focusses the object in the film, and then raises or lowers the condenser and simultaneously moves the mirror until the field of the microscope is illuminated as brightly as possible. The iris diaphragm is to be widely open when stained films are being examined.

Examination of Unstained Bacteria in a Hanging-drop.—This is the simplest method of determining whether the bacteria in

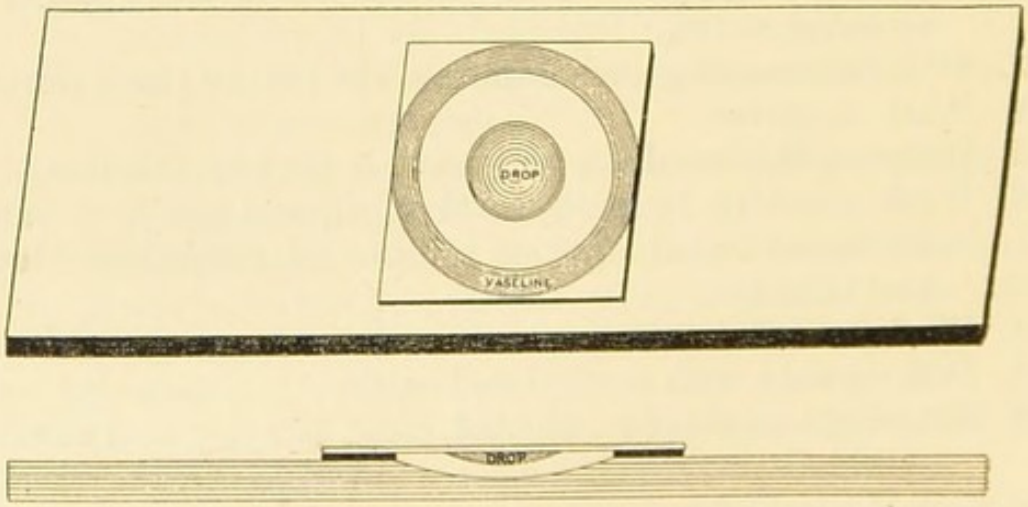


FIG. 197.—Hanging-drop preparation.

question are motile. A hollow-ground miscroscope slide is first ringed round with vaseline. A loopful of the fluid to be examined is then placed on the centre of a clean, dry No. 1 cover-glass, and the prepared slide is inverted and lowered on to the cover-glass.

When the preparation is now reinverted, the drop of fluid depends from the cover-glass, as in Fig. 197.

To examine the preparation, place a drop of special cedar oil on the centre of the cover-glass, and, while using a low-power objective, bring the edge of the hanging-drop into the centre of the field. Then rotate the oil-immersion objective into position, lower the microscope tube until the objective is seen to touch the oil, and focus the preparation first with the coarse and then with the fine adjustment. The iris diaphragm is to be contracted, because unstained bacteria suspended in fluid have to be recognised mainly by the difference of their refractive index from that of the fluid containing them.

The Brownian movement of bacteria suspended in a fluid must not be mistaken for independent motility. The latter is recognised with certainty only when the movement of the bacteria is progressive, so that their position in relation to that of others is constantly changing.

CULTIVATION OF BACTERIA

Preparation of Media.—For the preparation of sterile nutrient media a steam steriliser is required, while a hot-air steriliser is necessary for the sterilisation of test tubes—Petri's dishes and other glass apparatus—which should be exposed to a temperature of 170° C. for one hour. Of the liquid and solid media employed in the cultivation of bacteria, the most generally useful are bouillon, gelatine, and agar. But others are often indispensable.

Bouillon.—Take 500 grm. of lean minced beef or horseflesh, add 1 litre of distilled water, stir thoroughly, and leave standing for twenty-four hours in a cool place. Skim off any fat, filter through a fine linen cloth into a 2-litre flask, and by the hand or a meat-press express the juice from the residue until the flask contains 1 litre of "meat extract." An alternative method of preparing meat extract is to dissolve 10 grm. of Liebig's extract in 1 litre of distilled water.

To 1 litre of meat extract add 10 grm. of Witte's peptone and 5 grm. of sodium chloride, dissolve by heating, render feebly alkaline to litmus by adding caustic soda solution drop by drop, and heat in the steam steriliser at 100° C. for one to two hours. Filter when cold through a double layer of moistened filter paper into a flask. The bouillon, thus prepared, must when cold be absolutely clear. Clean, dry test tubes are filled each about one-third full of bouillon, the tubes are plugged with cotton wool, the mouth of the flask containing the remainder of the bouillon not immediately required is also plugged with wool, and the tubes and flask and their contents are then sterilised by being heated to 100° C. in the steam steriliser for fifteen to twenty minutes on three successive days.

Gelatine.—Take 1 litre of meat extract, prepared as above, in a 2-litre flask, add 100 grm. of gelatine, 10 grm. of Witte's peptone and 5 grm. of sodium chloride. Dissolve by heating to 40–50° C., render feebly alkaline to

litmus by addition of caustic soda solution, heat to 100° C. in the steam steriliser for 1 to 1½ hours, filter through a double layer of moistened filter paper, and again test the reaction to litmus paper. If necessary add caustic soda solution. The gelatine thus prepared must be absolutely clear when cold. If not so, clarify by adding the white of an egg, boiling for 1½ hours in the steriliser, and filtering. The prepared gelatine is filled into test tubes, which are then plugged and sterilised on three successive days, in the same manner as the bouillon tubes.

AGAR.—To 1 litre of meat extract add 10 grm. of peptone and 5 grm. of sodium chloride, heat to 100° C. for one hour, filter when cold through filter paper, add 15 grm. of agar, dissolve it by boiling in the steam steriliser, render feebly alkaline by adding caustic soda solution, boil again in the steriliser for several hours, filter (in the steriliser), and fill into test tubes, which are then plugged and sterilised in the same manner as are those filled with bouillon or gelatine.

The agar medium dissolves at 98° C. and solidifies at 40° C.

GLUCOSE AGAR.—This is ordinary medium, to which, before the final boiling, 0·3–0·5 per cent. of glucose is added. It is specially required for the cultivation of anaerobes, and for ascertaining whether a given bacterium causes fermentation of glucose and production of gas.

BLOOD AGAR.—This is required for the cultivation of the influenza bacillus. It is prepared by smearing one or two loopfuls of sterile blood over the surface of the agar medium in a Petri's dish, or of agar sloped in a test tube.

SERUM AGAR.—Dissolve ordinary agar medium (containing 2 per cent. of agar), allow it to cool to 42° C., and then mix it with an equal part of sterile human blood serum, hydrocele fluid, or ascitic fluid (heated to about 40° C.). The mixture must be immediately poured out into a sterile Petri's dish, or allowed to solidify in the tube while the latter is in a sloped position. Serum agar is specially required for the cultivation of the gonococcus.

GLYCERINE AGAR is ordinary agar medium to which while liquid 5 per cent. pure glycerine has been added ; the medium is then sterilised.

PEPTONE WATER.—Dissolve 10 grm. of Witte's peptone and 10 grm. of sodium chloride in 1 litre of water, boil for one hour in the steam steriliser, filter and fill into test tubes, which are then plugged and sterilised in the same manner as bouillon tubes. Peptone water is specially required for testing the formation of indol by bacteria.

MILK.—Test tubes are fllled about one-third full of fresh skimmed milk. The tubes are plugged and sterilised by being placed in the steam steriliser for one hour at 100° C. on three successive days.

LÖFFLER'S BLOOD SERUM.—Three parts of blood serum, obtained from the calf or sheep, are mixed with 1 part of neutralised bouillon (containing 1 per cent. peptone, 0·5 per cent. sodium chloride, and 1 per cent. glucose). Fill the mixture into test tubes, which are then plugged, laid on their sides and placed in the steam steriliser at 100° C., for fifteen to twenty minutes on three successive days. The medium is thus solidified and sterilised in a sloped position. Or about 10 c.c. of this fluid medium may be placed in a Petri's dish, and solidified and sterilised in the steam steriliser. Löffler's blood serum is the best medium for the cultivation of the diphtheria bacillus.

POTATO.—With a cork borer cut out a cylinder from a raw peeled potato, and divide it obliquely into two wedges, and wash them in water. Then insert each wedge into a special culture tube which is constricted near its lower end, and contains a few drops of water. Plug the tubes and sterilise them and their contents in the steam steriliser for forty-five minutes on the first day, and for fifteen to twenty minutes on the two succeeding days.

DESCRIPTION OF PLATE VIII.

Bacteria drawn from films seen under a $\frac{1}{12}$-inch objective and No. IV. eyepiece (Leitz).

1. Pneumococcus in pleural effusion. Stained by Gram's method, with fuchsin as counterstain.

2. Meningococcus in cerebro-spinal fluid. Stained by Gram's method, with fuchsin as counterstain.

3. Influenza bacilli in sputum. Stained by Gram's method, with fuchsin as counterstain.

4. Tubercle bacilli in sputum. Stained by Ziehl-Neelsen's method, with methylene-blue as counterstain.

5. *Bacillus diphtheriæ* from a twenty-four hours' growth on Löffler's blood serum. Stained by Neisser's method.

6. *Bacillus pestis* in the pus of a bubo. Stained with methylene-blue.



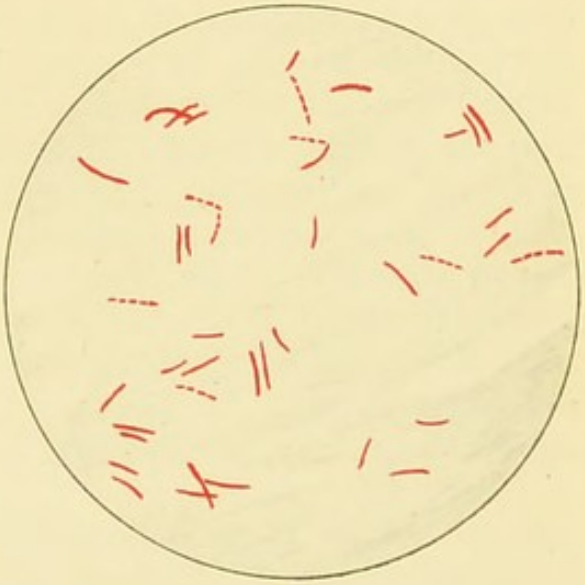
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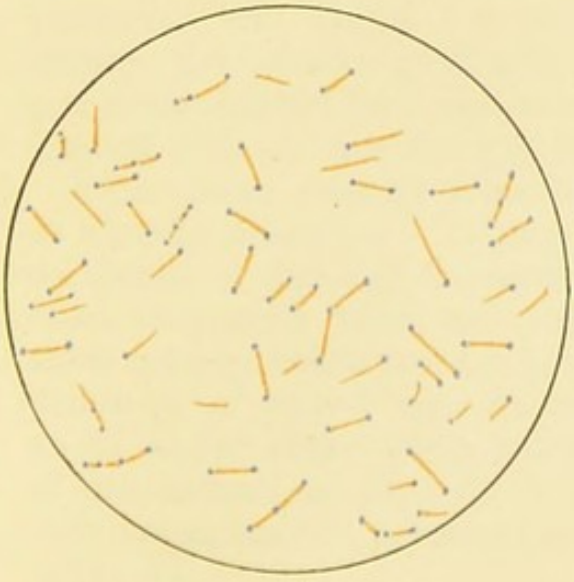
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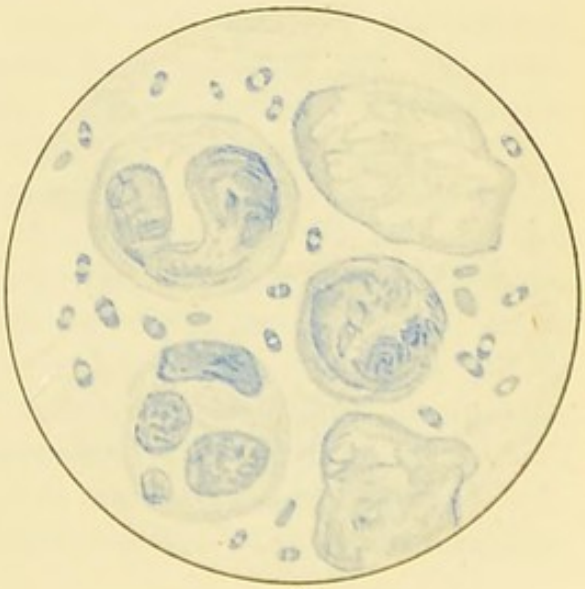
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To face page 442

Cultivation and Isolation of Bacteria

The initial cultivations should, almost without exception, be made on a solid medium, and usually on agar.

Cultivation on Agar.—A tube of agar is placed in boiling water, the medium is dissolved, allowed to cool down to 42° C., the plug is withdrawn, the mouth of the tube is sterilised by being rotated for several seconds in a Bunsen flame, and the liquid agar is then poured out into a Petri's dish, which has been previously sterilised by being exposed in the hot air steriliser to a temperature of 170° C. for one hour. After the agar has solidified, it is inoculated. The tube containing the material to be examined is held almost horizontally between the left thumb and forefinger, the mouth of the tube being directed towards the palmar surface. A platinum loop is taken in the right hand, is sterilised in a Bunsen flame, and allowed to cool. The cotton wool plug of the tube is now grasped between the forefinger and thumb of the right hand, is withdrawn by a rotatory movement, and placed between the ring and little fingers of the left hand in such a manner that the plug is held only by its upper end. A loopful of the material is then removed from the tube, the plug is reinserted, and the tube placed in a rack. The cover of the Petri's dish is raised by the left hand, the loopful of material is laid on the surface of the agar, and then spread out in parallel strokes by means of a small platinum spatula, which has been previously sterilised in the Bunsen flame and allowed to cool.

The preparation is placed in the incubator at 37° C. for twenty-four hours and then examined. It will probably be found that along the line corresponding to the first stroke the colonies are very numerous and dense, but that along the lines of succeeding strokes the colonies gradually diminish in number and lie more and more isolated from one another. These isolated colonies are studied with the unaided eye and with the microscope.

From these colonies films are prepared, stained, and examined as already described. It may be found that all the colonies are composed of one and the same bacterium, *i.e.* it is a pure culture. More frequently there are colonies of different bacteria; from these colonies subcultures are made on the surface of agar in Petri's dishes or of sloped agar in test tubes, and if after incubation at 37° C. for twenty-four hours a subculture contains a growth of one bacterium only—*i.e.* is a pure culture—then further subcultures as may be necessary for diagnosis can be made in bouillon, milk, etc.

If Petri's dishes are not available, the initial cultivation may be made on to the surface of sloped agar in tubes. The tube containing the material to be examined, and three tubes of sloped agar, are held between the left thumb and forefinger, the platinum loop is sterilised in the flame, the plugs are removed from the tubes and held between the fingers of the left hand, the mouths of the tubes are held for several seconds in the flame, the loop is charged once with the material and then smeared over the agar of the first and then of the second and third tubes. The plugs are reinserted and the tubes are placed in the incubator.

Whenever the platinum loop, needle or spatula have been in use, they must always, before being laid down, be sterilised in the Bunsen flame.

All Petri's dishes and tubes after being inoculated should be numbered and dated with a special pencil adapted for writing on glass.

Cultivation of Bacteria in Gelatine Plates.—Three sterilised Petri's dishes and three tubes of gelatine, liquefied and then allowed to cool to about 30°–40° C., are required. The tube containing the material for examination and the three gelatine tubes are held almost horizontally between the left thumb and forefinger, the platinum loop is sterilised, and the plugs are removed from the tubes. That of the tube containing the material to be examined is held between the left little and ring fingers, the other plugs are thrown away. One loopful of the material is transferred to the first gelatine tube and thoroughly mixed with the medium. The loop is then sterilised, allowed to cool, and three loopfuls are transferred from the first to the second gelatine tube; after the loop has been again sterilised, three loopfuls are transferred from the second to the third gelatine tube. The gelatine of each tube is at once poured into a Petri's dish, the three dishes are marked I., II. and III., and allowed to stand at the room temperature (18°–20° C.) for twenty-four hours or longer until the colonies develop. Many will do so in the first Petri's dish, fewer in the second, and still fewer in the third. The colonies lie both in the gelatine and on its surface. The latter, the surface colonies, are the more characteristic, and should be specially studied and sub-cultured.

Gelatine Stab Cultures.—While a gelatine tube and that containing the material to be examined are held in the left hand, in the manner already described, the sterilised platinum needle is charged and then inserted right down through the middle of the solid gelatine.

When a fluid medium is to be inoculated, a loopful of the material is to be thoroughly mixed in the medium. In testing gas formation in agar containing glucose, lactose, or other sugar, the medium is liquefied, allowed to cool down to 42° C., inoculated, and then held in a stream of cold water so that the medium may quickly solidify.

It must be remembered that before the medium in a tube is inoculated, or a culture taken from it, the mouth of the tube must be sterilised by being rotated in the Bunsen flame for two or three seconds after removal of the cotton wool plug.

Cultivation of Anaerobes.—The methods hitherto described have dealt with the cultivation of aerobic bacteria. In some instances, however, it is necessary to cultivate anaerobically.

The best medium for the cultivation of anaerobes is one containing a reducing substance, such as glucose. Anaerobes, for example, may often be cultivated by inoculating glucose agar which has been well boiled and allowed to cool down to 42° C. But in order to obtain surface growths of anaerobes, or a culture in a fluid medium, the inoculated tube must be placed in a vessel from which all the oxygen can be removed. This may be effected either by Buchner's method—namely, by placing the culture tube within a well-stoppered cylinder and absorbing the oxygen therefrom by means of pyrogallic acid and 10 per cent. aqueous solution of caustic potash—or else by replacing the air of the chamber in which the tube or Petri's dish is situated by means of hydrogen generated in a Kipp's apparatus, or by the use of a suction pump. These methods may with advantage be employed in combination.

SPUTUM

For bacteriological examination, sputum should be obtained fresh, in a sterilised vessel. If we are about to examine for tubercle bacilli alone, the vessel need not necessarily be a sterilised one, but if other bacteria are to be sought for, the patient must first wash out the mouth repeatedly with sterilised water; he is then told to cough, and the expectoration is received into a sterilised covered dish, *e.g.* a Petri's dish.

Examination for Tubercle Bacilli.—It is best to examine the sputum expectorated in the morning when the patient has just wakened. The sputum, if obtained in an ordinary sputum dish, is poured into a Petri's dish laid on a black surface. With the aid of two sterilised platinum loops, or a loop and spatula, one of

the opaque, yellowish particles is removed, and from it two films are prepared, fixed, stained by the modified Ziehl-Neelsen method, as described on page 438, and examined with the microscope.

If tubercle bacilli are present, they are seen as bright red bacilli, often slightly curved and beaded, whereas all else is stained blue (Plate VIII., 4). If in a suspected case no red bacilli are detected, more films should be prepared from other particles of the sputum. If these likewise fail to reveal the presence of tubercle bacilli, we employ the method of liquefaction and sedimentation.

Biedert's Method.—To sputum in a beaker or porcelain capsule add (according to the consistence of the sputum) 1 to 10 parts of 0·2 per cent. solution of caustic soda, warm the mixture over a Bunsen flame, and stir it with a glass rod until the fluid is homogeneous; the fluid is then centrifuged, and from the deposit in the centrifuge tubes films are prepared, fixed, and stained in the manner already described.

An alternative, but less satisfactory method, is to thoroughly shake up for several minutes 1 part of the sputum with 10 parts of carbolic acid solution (1 : 20). The mixture is allowed to stand in a covered conical glass or burette for twenty-four hours, or is at once centrifuged, and films are prepared from the deposit.

If bright red bacilli are found in the sputum they are almost certainly tubercle bacilli. Other acid and alcohol proof bacilli and streptothrices may occasionally be detected in the sputum, *e.g.* in cases of streptothrix infection, gangrene of the lung, and bronchiectasis. If there be any doubt, the diagnosis should be confirmed or disproved by washing the sputum (*vide infra*), and then making intraperitoneal inoculations into guinea-pigs, and by cultivating on sloped glycerine agar, which is to be incubated at 37° C. Tubercle bacilli grow more slowly than the other acid and alcohol proof bacteria, or may entirely fail to grow on the inoculated media.

Examination of Sputum for other Bacteria.—The sputum should be received into a sterilised Petri's dish after the patient has repeatedly washed out the mouth with sterilised water. The dish having been placed on a black surface, the denser, more opaque particles are, by means of sterilised platinum loops or spatulæ, transferred in succession to three tubes containing sterilised water or peptone water, and thoroughly shaken up in each tube. The sputum particles are thus mainly, if not entirely, freed from the bacteria with which they became contaminated during their passage through the patient's pharynx and mouth.

From the washed particles of sputum a film is prepared, fixed and stained by Gram's method, dilute fuchsin being used as the counterstain. Microscopic examination of the film may enable us to diagnose with considerable exactitude such bacteria as the pneumococcus, *Micrococcus catarrhalis*, streptococci, staphylococci, and the influenza bacillus. Other bacteria are less readily identified by miscroscopic examination alone.

In order to establish the diagnosis, one or more culture media should always be inoculated from the particles of washed sputum. It is well to make stroke cultures on the surface of serum agar and of blood agar. The former is the best medium for the pneumococcus, the latter is required for the growth of the influenza bacillus.

Pneumococcus (*Diplococcus pneumoniæ*).—This coccus is frequently found in the sputum. In acute lobar pneumonia, and in many cases of acute broncho-pneumonia and acute and chronic bronchitis, the sputum contains these diplococci abundantly and in pure culture. In these cases the pneumococcus is the exciting cause of the disease. It is also frequently found in cases of pulmonary mixed infection, *e.g.* in that of pulmonary tuberculosis, bronchiectasis, and abscess of the lung.

In film preparations of sputum, stained by Gram's method, as described on page 437, pneumococci are seen as oval or lancet-shaped diplococci, lying end to end, of dark purple colour (Gram positive), and surrounded by either a clear unstained oval area, or by a pink stained oval capsule (Plate VIII., 1). Short chains composed of two or three pairs of cocci are sometimes seen. The cocci are mainly, if not entirely, extra-cellular, *i.e.* do not usually lie within the leucocytes.

The pneumococcus grows at 37° C., but not at the room temperature, and consequently does not grow on gelatine. It grows best on serum agar, but also grows well on blood agar, agar, and Löffler's blood serum. To the naked eye the colonies are small, colourless, and translucent, like minute dewdrops. With the low power of the miscroscope, the colonies appear very finely granular, somewhat flat, and almost colourless. They are most likely to be mistaken for colonies of streptococci or of *B. influenzæ*. Bouillon subcultures twenty-four hours after inoculation with pneumococci show a faint diffuse turbidity, and a film therefrom presents the cocci, mainly in pairs, though some may be arranged to form short chains of two or three or more diplococci. Milk is usually rendered acid and coagulated. No growth occurs in a gelatine stab, nor on potato.

Streptococcus pyogenes.—Streptococci are frequently found in the sputum, and especially in the mixed infection of phthisis pulmonalis, bronchiectasis, and chronic bronchitis.

In films, streptococci are seen as rounded cocci, arranged in long or short spiral chains. The cocci are Gram positive and extra-cellular. They grow at 37° C. and at the room temperature. On agar the colonies appear to the naked eye as small grey points. On microscopic examination they are yellowish-brown, coarsely granular, and at their margin the coiled chains of cocci may be seen with the high-power objective. In gelatine stab a greyish-white growth occurs (after two, three or four days) along the needle track only. The medium is not liquefied. In bouillon, streptococci usually form long chains, which settle at the bottom of the tube in the form of a flocculent whitish sediment. In some instances (*Streptococcus brevis*), when the cocci form short chains, the bouillon is diffusely turbid. Milk is rendered acid and becomes coagulated.

The differentiation of *Streptococcus brevis* from pneumococci is sometimes attended with difficulty.

Staphylococci are less frequent in washed sputum, and when present the case is usually one of mixed infection. In films, staphylococci are seen as round Gram positive cocci, arranged usually in clusters, sometimes as diplococci or monococci. They are extra-cellular. For diagnosis, gelatine cultures are the most satisfactory.

In gelatine plates (p. 444) the surface colonies, after about two days' growth, are larger than those of streptococci or pneumococci, being several millimetres in diameter and greyish-white. When seen with the microscope, the colonies are dark-brown and finely granular, and the margin is sharp and regular. The gelatine becomes liquefied in the course of a few days, and, according to the species under cultivation (*Staphylococcus pyogenes aureus*, *S. pyogenes albus*, *S. citreus*), the colour of the growth becomes orange, white, or lemon yellow. In a gelatine stab culture, there is at first growth on the surface and along the needle track; after a few days liquefaction commences, and the sediment at the bottom of the liquefied medium is either white, or of orange or yellow colour, according to the species of staphylococcus present. Bouillon is rendered diffusely turbid. Films from it show the cocci arranged in clusters. Milk is coagulated.

Micrococcus catarrhalis.—This is not an uncommon coccus in bronchitic sputum. In some cases it is found in pure culture.

Morphologically the cocci resemble gonococci and meningococci. In films stained by Gram's method, as described on page 437, *Micrococcus catarrhalis* appears as diplococci with their adjacent surfaces flattened; the cocci are Gram negative, and lie within the cytoplasm of the leucocytes. From pneumococci they are distinguished by their shape, their intracellular position, and their reaction to Gram's stain.

The initial cultivations are best made on serum agar or blood agar. To the naked eye the colonies are greyish white; when twenty-four hours old they are about the same size as colonies of streptococci. Under the low power of the microscope the centre of the colony appears of yellowish-brown colour and coarsely granular, the margin being somewhat irregular. Later the central portion of the colony is elevated, whilst the periphery is translucent with a wavy margin. Subcultures can be obtained on agar and gelatine, and in milk and bouillon.

On the surface of agar the colonies resemble those on serum agar, but are somewhat smaller.

In bouillon, growth occurs as a thin surface pellicle which forms in the course of a few days, provided that the tube be not disturbed. A finely granular sediment settles at the bottom of the tube, while the medium remain clear or becomes slightly turbid.

In gelatine stab, after five or six days' growth there is a greyish-white growth on the surface, and a faint growth along the upper part of the needle track. Milk is not coagulated.

The chief cultural differences between *Micrococcus catarrhalis* and *Micrococcus meningitidis cerebrospinalis* (meningococcus, see p. 464) are that on agar the colonies of the former coccus are denser and whiter, their centre is more coarsely granular, and their margin less regular. When touched with the platinum loop, the colonies of *Micrococcus catarrhalis* can be pushed along the surface of the medium, and are thereby broken up, like mortar, into fragments. Further, the coccus grows in gelatine (at 20° C.), whereas the meningococcus does not.

Bacillus influenzæ is frequently present in the sputum in cases of epidemic and sporadic influenzal bronchitis and pneumonia, and may not infrequently be found in cases of pulmonary tuberculosis. Film preparations of sputum should be stained by Gram's method, dilute fuchsin being used as a counterstain. In typical cases the film is swarming with minute bacilli (about 0·2–0·5 μ in length), which are extra-cellular and also lie within the leucocytes (Plate VIII. 3). The bacilli are Gram negative, and

sometimes exhibit polar staining. They are most likely to be mistaken for *B. pyocyaneus* (which is very rarely found in sputum), but also for *Micrococcus catarrhalis*, and for pneumococci, which have undergone degeneration and therefore do not retain the gentian-violet by Gram's stain.

The diagnosis is made by cultivating on blood agar (p. 442). The colonies of *B. influenzæ*, when twenty-four hours old, are minute and colourless, like tiny dewdrops; later they may somewhat exceed 1 mm. in diameter, and may also become less translucent. Under the microscope each colony (twenty-four hours old) is seen to have a coarsely granular centre, while the periphery appears colourless and homogeneous. The diagnosis is confirmed by making two subcultures, one on ordinary agar, the other on blood agar. Growth occurs on the latter but not on the former.

Bacillus pneumoniæ (**Pneumobacillus** of Friedländer) is found in the sputum in a few cases of pneumonia and bronchitis, and may be associated with other bacteria. In film preparations of sputum the bacilli are seen in pairs, lying end to end, and enclosed in a capsule. They are usually of definite bacillary form, but are sometimes so short as to resemble diplococci. The capsule may be difficult to demonstrate, and is not seen in films made from cultures. The bacilli are extracellular and Gram negative.

On agar and on gelatine the colonies are cream-coloured, convex and moist, and when touched with a platinum needle are observed to be markedly viscous. A gelatine stab culture acquires the form of a round-headed nail: along the needle track there is a whitish growth, and on the surface a markedly convex growth develops. Bouillon becomes diffusely turbid, while a pellicle is formed on its surface. In a hanging-drop preparation made from the bouillon culture the bacilli are non-motile. Milk is usually coagulated, and gas is formed in agar containing glucose or certain other sugars.

Among the more important points in the differentiation from *B. coli communis* are the presence of capsules in the initial films, the absence of motility, the nail-like growth in a gelatine stab culture, and the formation of a pellicle on the surface of bouillon.

Bacillus pestis is contained in the sputum in cases of pneumonic plague, and, in some instances, in the terminal stages of bubonic plague. In films prepared from the sputum the specific bacilli (Plate VIII. 6) may be seen in great numbers. They are short and ovoid, with rounded ends, and are extra-cellular. Two films

are prepared; in one, stained by Gram's method, the bacilli are seen to be Gram negative. The other film is stained with dilute methylene-blue or with borax-methylene-blue (borax, 5 per cent., methylene-blue, 2 per cent. in water) for half a minute, to demonstrate the polar staining of the bacilli.

If bacilli with these characters are found, cultures must be made to establish the diagnosis. Colonies of *B. pestis* on agar are small and of greyish tint to the naked eye. When seen with the low power of the microscope the colony is brownish-yellow, its margin is homogeneous and irregularly indented, its centre is granular. Subcultures may be made on gelatine and in bouillon, but for diagnosis the agar culture should be inoculated subcutaneously into rats and guinea-pigs. The animals die in one to three days, and the characteristic bacilli can then be obtained in film preparations and in cultures made from the hæmorrhagic œdema at the site of inoculation, or from the enlarged lymphatic glands.

Actinomyces.—In cases of pulmonary actinomycosis the sputum may contain small greyish granules (colonies) of the actinomyces or ray fungus, which is one of the streptothrix group of the higher bacteria. These granules, which may be visible to the naked eye, are best demonstrated by adding dilute solution of caustic potash to the sputum on a microscope slide, and examining the preparation with a low-power objective. At the margins of the granules the characteristic club-shaped forms may be seen. Specimens containing intact granules may be stained with picrocarmin or eosin, and mounted in Farrant's solution or in glycerine jelly. In other preparations the granules should be broken up, and the films dried, fixed, and stained with dilute fuchsin, or by Gram's method, to demonstrate the filaments composing the central portion of the granule.

Other streptothrices, differing from actinomyces in morphological and cultural characters, may occasionally be isolated from the sputum, and it is important to remember that the filaments may be so short as to resemble bacilli, and, being acid-proof, may be mistaken for tubercle bacilli (see p. 446).

THE THROAT

Bacillus diphtheriæ.—Bacteriological examination of the fauces is frequently required, and especially for the detection of *B. diphtheriæ* (Klebs-Löffler bacillus), the specific micro-organism of diphtheria.

A swab is prepared by tightly rolling a small piece of cotton wool around the end of a stout copper wire; the latter is contained in a test-tube, the mouth of which is filled by a cotton wool plug attached to the upper end of the wire. The tube and its contents are sterilised in the hot air steriliser at 170° C.

While the tongue is depressed by means of a spatula, the sterilised swab is withdrawn from the tube, rubbed over the tonsil, soft palate, or other affected part of the throat, and thereafter replaced within the tube.

The first step in the examination consists in making cultivations from the swab, by means of the platinum loop, on the surface of Löffler's blood serum (p. 442), contained in a tube or a Petri's dish,

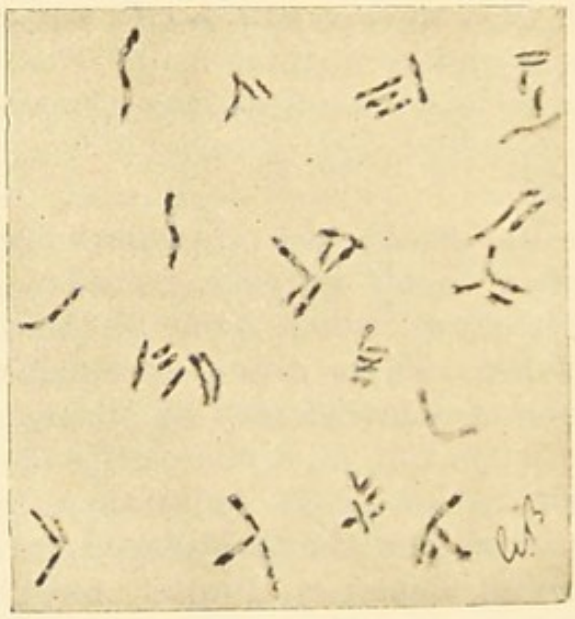


FIG. 198.—Bacillus diphtheriæ, from twenty-four hours' culture, stained with methylene-blue.

which is then placed in the incubator. Or the swab itself may be smeared over the surface of the medium. Two film preparations are then made directly from the swab. One is stained by Gram's method (p. 437), the other with dilute methylene-blue. In the former, search should be made for Gram positive bacilli, about as long as tubercle bacilli, but somewhat thicker. In the second film, stained with methylene-blue, we search for bacilli which are irregularly stained, and therefore have the appearance of being segmented (Fig. 198).

In some instances the diagnosis of diphtheria is rendered probable by the detection in the films of numerous bacilli presenting the above-mentioned characters. In other cases of undoubted diphtheria, no bacilli definitely recognisable as

diphtheria bacilli can be detected in the films. But in any case a definite diagnosis cannot be made until the cultures have been examined, after about twenty-four hours' growth in the incubator.

On Löffler's blood serum (which is the best medium for their growth) diphtheria bacilli form small greyish colonies (about $\frac{1}{2}$ to 1 mm. in diameter), or, if the colonies be abundant, a granular greyish-white growth is seen on the surface of the medium. The culture is often impure from the presence of other bacteria—cocci and bacilli. Two films are prepared from the culture. One is stained by Neisser's method (p. 439), the other by Gram's method. If the appearances of the culture to the naked eye correspond with those of *B. diphtheriæ*, and if the bacilli from the culture, twenty-four hours old, are (1) Gram positive, and (2) manifest blue polar granules (see Plate VIII. 5), in the film stained by Neisser's method, a positive diagnosis of *B. diphtheriæ* is justified. In cultures more than one day old, club-shaped and pear-shaped involution forms of the diphtheria bacilli will be found.

It must be borne in mind that in not a few cases which are clinically cases of diphtheria no diphtheria bacilli can be cultivated from the swab. *B. diphtheriæ* forms acid in bouillon subcultures. Inoculated into guinea-pigs, the bacilli may prove virulent or avirulent.

B. diphtheriæ has to be differentiated from pseudo-diphtheria bacilli and from *Xerosis bacillus*. Pseudo-diphtheria bacilli (Hoffmann's bacilli), which are often present both in healthy and diseased throats, are distinguished from true diphtheria bacilli by the larger size of the colonies, the absence of blue granules in films prepared from Löffler's blood serum and stained by Neisser's method, and by the production of alkali in bouillon; from virulent diphtheria bacilli they are also distinguished by being avirulent on inoculation into guinea-pigs. *Xerosis bacillus* is frequently found in the conjunctival secretion both in health and disease. Its colonies closely resemble those of the diphtheria bacillus, it presents blue granules by Neisser's stain, but bouillon is not rendered acid. *Xerosis bacillus*, moreover, is not pathogenetic to the guinea-pig.

The most important of the other bacteria which may be obtained from swabs taken from the throat are *Streptococcus pyogenes* (p. 448), *Staphylococcus pyogenes aureus* (p. 448), especially in cases of follicular tonsillitis, pneumococci (p. 447), *B. influenzæ* (p. 449), and *Bacillus fusiformis* (in cases of ulcerative stomatitis and tonsillitis). The last-mentioned bacillus is long, tapering at both ends, and often curved. It is Gram negative, and in

film preparations is usually associated with spirochætes, which are recognised by their spiral form. *B. fusiformis* does not grow on culture media.

FÆCES

In films prepared from the fæces of normal adults, micro-organisms in large numbers can be recognised—bacilli, spirilla, cocci, moulds, and yeasts. Of these micro-organisms, some are strictly anaerobic, others (chiefly the Gram positive bacilli) can be cultivated only on media of acid reaction. We shall, however, consider only the chief bacteria which are of importance to the clinician.

Bacillus coli communis.—This bacillus is one of the chief members of the normal intestinal flora. It is constantly present in the contents of the large intestine and in the fæces of healthy persons. It is also frequently found in the purulent exudate of localised and general peritonitis, and may also cause inflammation of the bile ducts and gall-bladder, of the pelvis of the kidney, and of the urinary bladder. The bacillus may, moreover, be found in causal association with inflammatory processes elsewhere, as in the urethra, uterus, lungs, etc.

The bacilli are of variable length. When young they may resemble cocci; older bacilli from fluid media are often of filamentous form. Their motility is usually recognisable in suitably prepared specimens (see p. 440). The bacillus is Gram negative. To the naked eye the superficial colonies on gelatine are large (3 to 4 mm. in diameter), greyish-white or bluish-white and flat, and their margins are wavy. In gelatine stab culture there is a greyish-white growth along the needle tract and a flat surface growth of like colour. The medium is not liquefied. On agar the colonies are less characteristic. Bouillon is rendered diffusely turbid. Milk is rendered acid and is usually coagulated. Peptone water cultures twenty-four hours old yield the indol reaction, *i.e.* after the addition of a few drops of a 0·02 per cent. aqueous solution of potassium nitrite the culture is shaken and a little pure concentrated sulphuric acid is added, when a rose-pink colour is produced. The appearance of the reaction is frequently delayed for about five minutes, and the colour is best developed if, after the addition of the reagents, the tube is gently warmed for several minutes. The bacillus produces gas in media containing glucose, lactose, or maltose.

With the above-mentioned ordinary media the diagnosis can usually be satisfactorily determined, but as an aid in the differ-

entiation of *B. coli communis* from *B. typhosus* there are two media[1] which are particularly useful, namely, (1) MacConkey's taurocholate-litmus-bouillon, in which *B. coli communis* forms both acid and gas (whereas *B. typhosus* produces acid, but no gas), and (2) v. Drigalski and Conradi's nutrose- lactose- litmus-agar containing crystal-violet. On this medium colonies of *B. coli communis* are red, while those of *B. typhosus* are smaller, colourless, or of pale bluish tint, and transparent.

Bacillus lactis ærogenes is found in the stools of infants, and occasionally in peritoneal pus, and in the urine of cystitis and pyelitis. Resembling *B. coli communis* morphologically, *B. lactis ærogenes* is readily differentiated by means of cultures. But being non-motile, forming on agar and gelatine colonies which are convex and whitish, and producing gas in media containing glucose or lactose, *B. lactis ærogenes* closely resembles *B. pneumoniæ*, and the differential diagnosis is often attended with difficulty.

Bacillus typhosus, the specific virus of typhoid fever, may be isolated from the fæces and urine of patients suffering from that disease. In clinical work, however, the bacteriological diagnosis of typhoid fever is not based on the detection of *B. typhosus*, but on the agglutination of the specific micro-organism by the blood serum of the patient. Post-mortem, the bacillus should be sought for in the spleen and in the bile within the gall-bladder.

B. typhosus is usually short (1 to 3 μ in length), but in fluid media it forms longer filaments. It is Gram negative; in alkaline media it is actively motile.

The surface colonies on gelatine are somewhat like those of *B. coli communis*, but smaller and bluish-white. When seen with a low-power objective of the microscope the margin of the colony is wavy, and its surface is venated so that it resembles a vine leaf. On agar the colonies are less characteristic. In a gelatine stab culture there is growth along the needle tract and a flat surface growth; the medium is not liquefied. Bouillon is rendered diffusely turbid; milk is rendered acid but is not coagulated; no gas is formed in media containing glucose or lactose; and no indol is produced in twenty-four hours in peptone water. Reference has already been made to the differential diagnosis from *B. coli communis* by means of MacConkey's bouillon and v. Drigalski and Conradi medium. The diagnosis of *B. typhosus* should be con-

[1] For the mode of preparing these two media, the reader should consult a text-book of bacteriology.

firmed by the use of a specific agglutinating serum, obtained from a case of typhoid fever.

From *B. fæcalis alkaligenes*, the typhoid bacillus is differentiated by the fact that the former produces alkali, the latter acid. This point is conveniently determined by cultivating in bouillon or whey to which litmus had previously been added.

Bacillus dysenteriæ (Shiga, Kruse, and Flexner) — the virus of epidemic dysentery—resembles the typhoid bacillus morphologically, in its reaction to Gram's stain, and in its cultural characters. It must be differentiated by the use of specific agglutinating serum. *B. dysenteriæ* is agglutinated by the serum of cases of epidemic dysentery, but not of typhoid fever.



FIG. 199.—Cholera vibrios.

Cholera Vibrio.—For the diagnosis of cholera a bacteriological examination is required, and this is especially the case at the commencement of an epidemic.

Cholera vibrios (Koch's comma bacilli) are short micro-organisms about 1·5 or 2 μ in length by about 0·5 μ in breadth, are curved so as to resemble a comma in shape, and usually lie singly, though some adhere to one another so as to resemble the letter S in form. The organism usually possesses one terminal flagellum; it is actively motile and Gram negative.

The method of diagnosis at the commencement of an epidemic is as follows :—From the flakes of mucus and epithelium floating in the fluid stools, films should be prepared and stained with diluted carbol-fuchsin. In many cases the curved organisms may be seen in large numbers, and apparently in pure culture. One or more of the flakes should be rubbed up with a little peptone water, and from the mixture a hanging-drop preparation (see p. 440) is made, in order to ascertain whether the organisms are motile. Four or five tubes of peptone water should then be inoculated with flakes from the dejecta, and examined at intervals

after six to twelve hours' incubation at 37° C. The medium is rendered diffusely turbid, while the organisms multiply most freely at the surface of the fluid, on which a thin pellicle, composed of vibrios, is formed. Two loopfuls of fluid are therefore taken from the surface of the peptone water culture, one for the purpose of making a film, which is stained and examined microscopically, whereas a hanging drop is made from the second loopful in order to ascertain whether the vibrios are motile.

From the tube which appears to contain the vibrios most abundantly, gelatine and agar plates are inoculated. The former should be incubated at 22° C. for eighteen to twenty hours, the latter at 37° C. for twelve to eighteen hours. The vibrios grow more rapidly on agar, but the colonies are more characteristic on gelatine. After about twenty hours' growth on the latter medium, the surface colonies appear to the naked eye as minute, refractile points, and when they are examined with the lower power of the microscope, their surface presents an appearance as if they were composed of tiny fragments of broken glass. On the second or third day the gelatine becomes liquefied around each colony, which sinks into the medium.

When a pure culture of the cholera vibrio has been obtained, a gelatine stab cultivation should be made. After its incubation at 22° C. for four or five days the gelatine at the surface of the medium becomes liquefied, and as evaporation occurs simultaneously, there is a cavity formed resembling an air bubble. Below this there is a funnel-shaped area of liquefied gelatine, and below this again a whitish line of growth along the needle track.

From the pure culture peptone water should be inoculated, incubated at 37° C. for twenty-four hours, and then tested for the cholera-red reaction (nitroso-indol reaction). This depends on the fact that by its growth the cholera vibrio forms indol from peptone and reduces nitrates to nitrites. The latter are decomposed by the addition of strong sulphuric acid, and the free nitric acid thereby formed acts on the indol so as to give a red colour. To the peptone water culture a few drops of pure sulphuric acid are therefore added, and the appearance of a reddish-pink tint is looked for. The reaction is important clinically, because, though many organisms (e.g. *Bacillus coli communis*) produce indol, very few produce at the same time the nitrite, the presence of which is necessary for the reaction.

The cholera vibrio is pathogenetic on intraperitoneal injection into guinea-pigs, which die in about twelve hours, signs of collapse preceding the fatal issue. In the peritoneal fluid, obtained after the death of the animals, the actively motile vibrios are obtained

in enormous numbers. The diagnosis of cholera is fairly well established if organisms are found corresponding in all respects with the points above mentioned, but as the cultural characters of other vibrios may closely resemble those of the cholera vibrio, the organism which has been isolated must be tested with a specific agglutinating serum (after the manner of Widal's reaction for enteric fever), and also for Pfeiffer's reaction.

For Pfeiffer's reaction three guinea-pigs are required. The first is injected intraperitoneally with a mixture of 1 c.c. of bouillon (containing one loopful of a culture) eighteen hours old, and one loopful of a bacteriolytic anti-cholera serum. The second guinea-pig is injected intraperitoneally with the same mixture, except that normal serum is used instead of anti-cholera serum. The third guinea-pig is injected intraperitoneally with one quarter of a loopful of the agar culture. By means of capillary glass tubes a little of the peritoneal fluid is withdrawn from each guinea-pig twenty minutes, and again one hour, after injection. If the vibrios injected were cholera vibrios, they are now observed in the peritoneal fluid from the first animal to be motionless and granular, and they may eventually be dissolved; whereas in the peritoneal fluid from the second and third guinea-pigs the vibrios are found to be still actively motile.

Tubercle bacilli may be found in the fæces. It is best to examine any particles of pus or mucus the latter may contain. The films are stained by the modified Ziehl-Neelsen method, as described on page 438. If acid- and alcohol-proof bacilli are found in large numbers, they are probably derived either from tuberculous sputum which has been swallowed, or from tuberculous ulcers of the intestines. Isolated acid-proof bacilli are not of great significance, as they might have been derived from the milk taken by the patient.

Various other bacilli, also streptococci and staphylococci, may be found in the fæces in some cases of enteritis, but are of importance only when very abundant.

URINE

Unless special precautions are taken, even normal urine, while being passed, may become more or less contaminated with bacteria derived from the urethra. Hence if the urine is to be submitted to bacteriological examination, the glans penis should first be washed and the urethra syringed with an antiseptic lotion. Thereafter the patient micturates, or, as is preferable, the urine is withdrawn by means of a sterilised catheter. From female

patients the urine should be obtained by means of a sterilised catheter after the external genitals have been washed with an antiseptic. In any instance, the first portion of the urine is probably contaminated with bacteria from the urethra, and, therefore, only the second portion is collected in a sterile flask.

In cases of pyuria the urine contains large numbers of pus cells and usually abundant bacteria. If the former are scanty and yet the latter abundant, the condition is termed bacteriuria. In about 20 per cent. of cases of typhoid fever *B. typhosus* appears in the urine after the seventh day of the disease, and may sometimes persist in large numbers until the late stage of convalescence.

In general, however, bacteria should be sought for in the small shreds or threads which the urine may contain, and it is always well to centrifuge the urine. From the deposit, films are prepared and stained by appropriate methods, as described on pages 437 and 438. Of various bacteria which may be found, the following are the most important :—

Tubercle bacilli.—In cases of tuberculosis of the kidney or bladder many films have usually to be prepared before any acid-proof bacilli are detected. Such bacilli may be either tubercle bacilli or smegma bacilli, the latter being normally saprophytic in the urethra. The only reliable means of differentiating these two bacilli is by experimental inoculation of animals. Intra-peritoneal injection of guinea-pigs is the most satisfactory method.

In cases of cystitis and pyelitis various other bacteria may also be found in the urine. The most important are *Bacillus coli communis* (especially in cystitis with urine of acid reaction), paracolon bacilli, *Bacillus lactis ærogenes*, typhoid bacilli, staphylococci, streptococci, gonococcus, and *Proteus vulgaris*. Gonococci are less readily obtained from urine than from urethral secretion, and they are therefore considered on page 460.

Paracolon bacilli resemble *Bacillus coli communis* in their morphological and most of their cultural characters, but do not ferment glucose or lactose, and therefore produce no gas in media containing those sugars.

Proteus vulgaris resembles *Bacillus coli communis* morphologically, is actively motile and Gram negative. It, however, liquefies gelatine, first coagulates milk and then liquefies the clot; and urine, bouillon or other medium in which the organism is cultivated becomes markedly alkaline.

The *Diplococcus rheumaticus*, obtained from the urine in some cases of acute rheumatism, is one of the streptococci, but its causal relationship to the disease in not yet proven.

URETHRAL SECRETION

As has been already mentioned, the urethra always contains saprophytic bacteria, one of the most frequent being the smegma bacillus (see p. 459). Among the most frequent of the bacteria obtained in cultures are staphylococci and diphtheroid bacilli, the latter resembling more or less closely the pseudodiphtheria bacilli of the fauces. A bacteriological examination of the urethral secretion is seldom required except for the diagnosis of gonorrhœa.

Gonococcus (*Micrococcus gonorrhœæ*).—It is well to prepare at least two films from the urethral secretion, so that one can be stained by Gram's method, in order to demonstrate the staining reaction of the cocci, while the second film is stained by Leishman's (p. 111), Jenner's (p. 110), or Giemsa's (p. 119) stain to show the intracellular position of the organisms.

The gonococcus is a diplococcus, the adjacent sides of the two cocci being flattened or even concave, so that the diploeoccus resembles two coffee beans lying side by side. In consequence of phagocytosis the cocci lie within the leucocytes. In urethral secretion the intracellular position is very characteristic of the gonococcus, and in films treated by Gram's method gonococci are found to be Gram negative. Hence if in films of urethral secretion cocci are found which are of diplococcus form, of coffee-bean shape, intracellular, and Gram negative, a diagnosis of gonorrhœa is established. When we are dealing with urethral secretion, cultures are not required for diagnosis, but they are necessary in determining that the vulvo-vaginitis of young girls is the result of gonorrhœal infection, and also when the material under examination is of extra-genital origin, *e.g.* is derived from the eye.

For the cultivation of the gonococcus from purulent fluids, a loopful of the latter should be smeared over the surface of serum agar (see p. 442). When the medium is examined after twenty-four hours' growth at 37° C., colonies of the gonococcus, if present, appear to the naked eye as minute, translucent points, which are of faint greyish tint. After forty-eight hours' growth at 37° C., the colonies, seen with the microscope, present a wavy margin and a brownish coarsely granular centre. In films prepared from the culture, many of the cocci are found to be degenerated, being somewhat swollen and staining poorly. Subcultures may be obtained on serum agar, but not on agar or gelatine.

FLUIDS FROM SEROUS CAVITIES

Pathological fluids from the serous cavities are usually obtained either by means of exploratory puncture or aspiration. The skin at the site chosen for puncture should have been previously cleansed with soap and water, and ether or alcohol, and a moist antiseptic dressing applied. The exploring syringe should have a capacity of not less than 10 c.c. ; the needle should be about 2 or $2\frac{1}{2}$ inches long, with a large bore, and should fit the nozzle of the syringe accurately. The construction of the instrument should enable it to withstand boiling without suffering damage, for it must be boiled for ten to fifteen minutes over a Bunsen flame, or in a steam steriliser, immediately before being used.

If the pleural cavity is to be explored, the needle must be introduced at a point at which the dulness, enfeeblement of breath sounds and of vocal resonance and fremitus are well marked. The best spot at which to puncture is usually the eighth intercostal space, slightly anterior to the scapular line.

If puncture of the pericardial sac has to be undertaken, the needle should be introduced through the fifth left interspace, 1 inch to the left of the sternum. Puncture at that spot is attended with less risk of injury to the heart and internal mammary artery than elsewhere.

When the peritoneal cavity is to be explored, the bladder should first be emptied, and the needle is then introduced at a spot where the dulness caused by the accumulation of fluid in the peritoneal cavity is well marked. The spot chosen is usually in the mesial line, midway between the umbilicus and the pubis, or above Poupart's ligament in the left iliac region, midway between the anterior superior iliac spine and the symphysis pubis.

Having obtained fluid, the needle is withdrawn, and a little cotton wool and collodion applied to the skin at the site of puncture.

A portion of the fluid is inoculated on to nutrient media, and films are also prepared. If the bacteria be scanty, part of the fluid should be centrifuged and films made from the deposit. The amount of fluid used for inoculation of the nutrient media depends on the number of bacteria contained in the fluid. If they are abundant (*e.g.* in some cases of empyema), a single loopful should be smeared over the medium, whereas if they are scanty it is well to mix 3 to 5 c.c. of the fluid with 10 c.c. of agar (previously liquefied and then cooled to 42° C.) and to pour the mixture into a Petri's dish.

The absence of any growth on the inoculated media may be due to a variety of causes :—

(*a*) The fluid may be sterile.

(*b*) The bacteria may be obligatory anaerobes, as is sometimes the case when we are dealing with fœtid fluids.

(*c*) The bacteria may be devitalised in consequence of the bactericidal action of the fluid. In such cases, however, scanty bacteria (*e.g.* pneumococci, streptococci, etc.) which stain poorly can usually be recognised in films prepared directly from the fluid.

(*d*) The exudate may be of tuberculous origin. If the clinical features of the case suggest the possibility of tuberculosis, search must be made for tubercle bacilli in the fluid. Before making films the fluid should be centrifuged for not less than fifteen minutes. In many instances, however, the fluid will have coagulated before the bacteriological examination can be commenced, and in such instances the clot (in which bacteria, if present, will be entangled) should be removed from the fluid and subjected to artificial digestion with pepsin and 0·2 per cent. HCl in the incubator for several hours. The fluid thus obtained is thereafter centrifuged ; films are prepared from the deposit and stained by Ziehl-Neelsen's method (see p. 438).

The result following the intraperitoneal inoculation of guinea-pigs may, however, indicate that the fluid was tuberculous when microscopic examination had failed to reveal the presence of tubercle bacilli.

In pleuritic and pericardial exudates, the most frequently encountered bacteria are streptococci, pneumococci, tubercle bacilli, and staphylococci. Influenza bacilli and *B. coli communis* are less common.

In peritoneal fluids, *B. coli communis* and streptococci are more frequent than staphylococci, tubercle bacilli, or gonococci. In fœtid pus (*e.g.* in some cases of appendicitis) *B. coli communis* or obligatory anaerobes may be found. Pneumococcal peritonitis is rare, and the cases recorded have usually occurred in female children.

Lumbar Puncture

Cerebro-spinal fluid is obtained by means of lumbar puncture—the introduction of an exploring needle between the spines of the 4th and 5th lumbar vertebræ into the subarachnoid space, which, it will be remembered, is continued down to the level of the 2nd sacral vertebra, whilst the spinal cord terminates opposite the 2nd lumbar vertebra.

The needle for use in adults should be stout, sharp, about 10 cm. long, and have a bore of 0·8 mm. For children a smaller instrument suffices. A small india-rubber tube is attached to the needle, and the whole is sterilised by boiling immediately before it is to be used. The skin of the lumbar region is also thoroughly cleansed and disinfected.

The patient should lie on his left side, at the edge of the bed, the thighs being flexed, and the back bent well forwards so as to increase the width of the intervertebral spaces. The physician, having carefully cleansed his hands, now determines the point corresponding to the space between the spines of the 4th and 5th lumbar vertebræ. That point is readily ascertained, as it lies at the level of a line joining the highest points of the iliac crests on each side. The physician places the left index finger on the fourth lumbar spine, and introduces the needle (held as one would a pen) in the fourth interspace 1 cm. to the right of the mesial line, and pushes it onwards and at the same time slightly upwards and towards the mesial line, and thus into the subarachnoid space. In adults the subarachnoid space is usually entered at a depth of about 5 to 6 cm. from the surface ; in children, at a depth of 2 to 3 cm. If bone be struck by the needle, it must be partly withdrawn and again introduced at a slightly lower level.

The cerebro-spinal fluid issuing from the tube attached to the needle is collected in a sterilised centrifuge tube. About 10 c.c. may usually be obtained, sometimes considerably more. A few drops are also allowed to fall directly on to the surface of sloped serum agar. The fluid may come away slowly drop by drop as in health ; but when the fluid is under increased pressure (as in meningitis, etc.) it may flow out in a stream of considerable force. If no fluid comes away, a sterilised wire should be passed through the needle to remove the flake of fibrin or other obstruction in the bore. After the needle has been withdrawn, a little cotton wool and collodion are applied to the site of puncture. The patient should retain the horizontal posture for one or two hours after the puncture, in order to obviate the headache which may otherwise ensue.

Some of the fluid should be at once inoculated on to the surface of blood agar, previously solidified in a Petri's dish, and this, together with the inoculated tubes of serum agar, is then incubated at 37° C. For the detection of the tubercle bacillus, 2 to 3 c.c. of the fluid should be injected intraperitoneally into a guinea-pig, which is killed and examined three weeks later. The remainder of the fluid is centrifuged for at least ten to fifteen minutes, after which the fluid is poured out of the centrifuge tube,

and from the deposit, collected with a capillary tube or platinum loop, films are prepared and stained.

In cases of tuberculous meningitis, tubercle bacilli can seldom be detected in films prepared from the cerebro-spinal fluid. In other forms of meningitis the bacteria most commonly encountered in films and cultures are the pneumococcus, meningococcus, streptococci, and staphylococci.

Micrococcus meningitidis cerebrospinalis (*Diplococcus intracellularis meningitidis*; meningococcus).—This coccus is the specific virus of epidemic cerebro-spinal meningitis; it is also found in cases of posterior basic meningitis of infants, and may occasionally be isolated from the naso-pharynx and accessory nasal sinuses of persons who have been seen in contact with patients suffering from cerebro-spinal meningitis.

The organism is a diplococcus closely resembling *Micrococcus gonorrhœæ* and *Micrococcus catarrhalis*, both in form and in being usually intracellular (see Plate VIII. 2). It is Gram negative, and in the films some of the diplococci are usually less deeply stained than others. To the naked eye the colonies on agar are small and greyish-white; under the low power of the microscope they each present a yellowish-brown finely granular centre and a translucent, almost colourless, periphery. On serum agar the colonies are somewhat larger. A faint diffuse turbidity is produced in bouillon; on its surface a thin pellicle may be formed, but only if the tube be not disturbed. No growth occurs on media kept at or below a temperature of 20° C., this being one of the features which differentiates the organism from *Micrococcus catarrhalis* (see p. 449).

It sometimes happens that although cocci having the characters of meningococci are found in the films prepared directly from the cerebro-spinal fluid, the vitality of the cocci is somewhat impaired, and consequently no growth is obtained on the inoculated media.

THE BLOOD

It is now universally recognised that in the course of many acute infective diseases the specific bacteria may enter the blood stream, and be found therein in greater or lesser numbers. This is true not only of such diseases as septicæmia, pyæmia, septicæmic plague, and malignant endocarditis, but of typhoid fever, acute lobar pneumonia, osteomyelitis, etc. As blood, however, contains bactericidal substances, there is a constant tendency for bacteria which invade it to be rendered innocuous and to undergo

destruction. Hence it follows that special methods of investigation are usually required for the detection of bacteria in the blood.

In grave cases, and especially during the period preceding the death of the patient, bacteria may occasionally be present in the blood in such large numbers that they may be detected merely by microscopic examination of blood films which have been fixed and stained, for example, by Leishman's stain (see p. 111). But from negative results obtained by such methods it cannot be inferred that the blood does not contain bacteria, because they are so often present in small numbers, and in every instance it is well to examine by cultural methods a considerable amount of blood taken with strict antiseptic precautions from one of the superficial veins of the arm. The procedure is not only easily carried out, but is attended with practically no risk to the patient, and often yields valuable evidence regarding diagnosis and prognosis.

Method.—The skin at the bend of the elbow having been carefully cleansed with soap and water, and with alcohol or ether, a wet antiseptic dressing is applied to the part for at least one hour. A syringe with a capacity of 10 c.c., and with a well-fitting sharp needle, is boiled immediately before it is to be used. The dressing is then removed from the elbow, and the skin of the part is again cleansed with alcohol or ether so as to remove every trace of antiseptic.

A bandage is then firmly applied around the upper arm so as to render the superficial veins at the elbow prominent. The vein chosen for puncture is usually the median basilic. The syringe is held in such a manner that the point of the needle is directed towards the patient's hand, while the long axis of the needle lies parallel to that of the turgid vein. The needle is then thrust into the lumen of the vein, and when gentle suction is made, blood will flow into the syringe. If it fails to do so, the needle is not within the lumen of the vein, and should, therefore, be partially removed and reintroduced.

When the syringe is full of blood, the bandage is first removed from the upper arm, and the needle then withdrawn; a sterilised dressing and a firm bandage are thereafter applied to the elbow.

A portion of the blood thus obtained should be immediately mixed with bouillon. The best method of procedure is to have at hand three or four small flasks, each containing 50 c.c. of bouillon. To each of these 1–2 c.c. of blood are added, and the flasks are then well shaken and put in the incubator. By this procedure the bactericidal power of the blood is held in abeyance, and any bacteria which may be present are maintained under conditions which are more favourable for their growth than can be obtained

by inoculation of solid nutrient media with the blood. After the bouillon has been incubated for twenty-four hours the bacteria will have multiplied greatly, and small quantities of the bouillon are transferred to other media—agar, gelatine, etc.—in order that the bacteria may be identified. In many cases, however, no growth is obtainable, and if the examination has been carefully performed, it may be inferred that there were no bacteria in the blood.

It is evident that by inoculation of bouillon with blood we acquire information merely as to the presence or absence of bacteria, but obtain no indication of their numbers in the blood. But in grave cases proceeding to a fatal issue, the blood may contain bacteria in abundance, and in order to form an estimate of their number, and thereby to judge of the prognosis, it is advisable to inoculate another portion of the blood either on to the surface of agar, or to mix the blood with agar which has been liquefied and then cooled to 42° C., to pour the mixture into Petri's dishes and incubate at 37° C. for twenty-four hours. The number of colonies which develop on the agar furnishes an indication of the relative number of bacteria in the blood. This method is especially applicable in cases of acute lobar pneumonia.

By the employment of these simple methods the blood may be found to contain streptococci in cases of septic disease, *e.g.* ulcerative endocarditis and septicæmia. The detection of staphylococci in cultures from the blood is always of doubtful significance; they usually represent contamination from the patient's skin, and are not derived from the blood itself.

Pneumococci may be found in the blood in almost all cases of acute lobar pneumonia, whether mild or grave. In the latter, when a fatal termination threatens, the blood may contain pneumococci in abundance, as may be determined in the manner already described.

In typhoid fever, *B. typhosus* may be isolated from the blood taken from a vein and inoculated in bouillon. As a further aid to diagnosis in doubtful cases, a drop of serum and blood may be obtained by incision of one of the roseolar spots, and bouillon inoculated therewith. Typhoid bacilli may sometimes be detected by this procedure before a positive Widal's reaction is obtainable.

Methods of Testing the Agglutinative Power of the Blood Serum

Although the agglutination reaction had been accurately described by Gruber and Durham, its clinical application to the diagnosis of typhoid fever was first introduced by Widal in 1896

and by Grünbaum. The serum diagnosis of typhoid fever is therefore commonly called Widal's test.

The reaction consists in the fact that when diluted serum of a patient suffering from typhoid fever is mixed with typhoid bacilli they lose their motility, and, adhering to one another, become aggregated into clumps. The reaction is dependent on the development of definite bodies, termed agglutinins, in the serum of the patient during the course of the disease.

TECHNIQUE OF THE METHOD.—We should have ready for use—

1. An agar culture of *B. typhosus*, incubated for twenty-four hours at 37° C.;
2. A tube of bouillon or of peptone water (see p. 442) to be used for diluting the serum;
3. One or more pipettes in which to collect the blood;
4. Two or more microscope slides with a central depression around which we place a ring of vaseline; and
5. Clean cover-glasses, a platinum loop, and a microscope giving a magnification of about 400 diameters.

The lobe of the patient's ear, after having been cleansed with a mixture of ether and alcohol, is punctured with a blood-pricker. The blood flowing from the puncture is collected in a leucocytometer pipette, a simple capillary tube, or a Wright's capsule (Fig. 200).

(1) If a leucocytometer pipette (see Fig. 41, p. 102) be used, we draw blood up to the mark 1, and immediately thereafter, bouillon or peptone water up to the mark 11. A dilution of 1 in 10 is thus obtained. If the subsequent manipulations cannot be immediately performed, as, for example, when the sample has to be sent to a laboratory at a distance, the blood should be collected according to one or other of the following methods.

(2) A simple capillary tube about three inches long having been half filled with blood, its ends are sealed in a small flame, care being meanwhile taken not to heat the blood and thus destroy its agglutinative power. The tube is now set aside for one hour or longer until the serum separates from the clot. One end of the tube is then broken off, and when the air within the far end of the tube is caused to expand by gently warming it over a small flame, the serum and clot will be expressed from the tube and should be received on to a perfectly clean microscope slide.

(3) If a Wright's capsule be used, the orifice of its shorter limb (*x*, Fig. 200) should be inserted into the drop of blood, which will run into the tube under the combined influence of capillarity and gravity. When the shorter limb has been filled, the upper

portion of the longer limb is gently warmed over a small flame and its orifice (*y*, Fig. 200) immediately sealed. When the air in the longer limb which has been rarefied by warming contracts on cooling, the blood is drawn into the body of the capsule, after which the orifice *x* is sealed in the flame. The capsule, after having cooled, is inverted and suspended by means of its curved arm in the centrifuge, and when the latter is revolved, the blood is driven into the longer limb of the capsule (Fig. 200, *b*), which is thereafter left at rest for some minutes until the serum begins to separate. It is then again centrifuged; the coagulum settles in the lower narrow portion of the capsule, while the serum rises to the top (see Fig. 200, *c*). The capsule is cut at about the level indicated by the letter L in Fig. 200, *c*), broken across, and the serum removed by means of a capillary pipette.

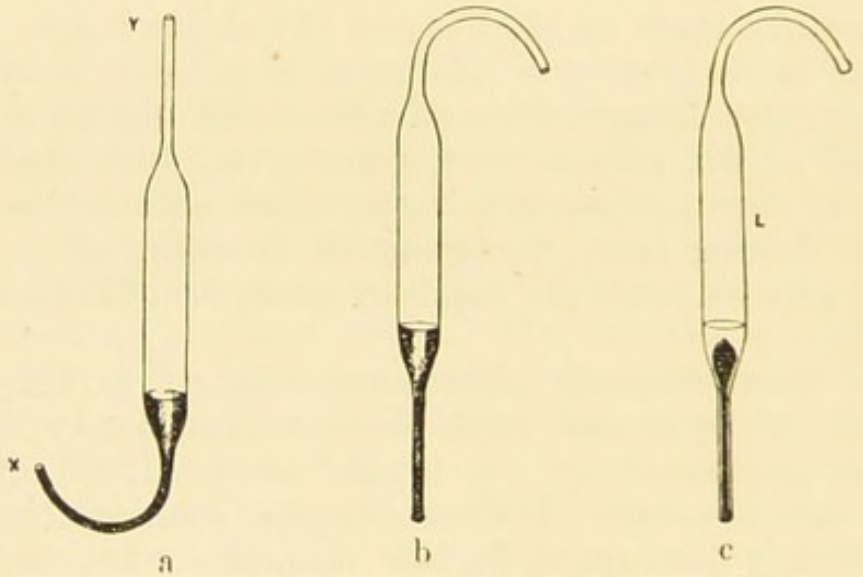


FIG. 200.—Wright's capsules (one-half natural size).

If the serum has been obtained by methods (2) or (3), it must now be diluted. If we have obtained a good deal of serum, the dilution can be carried out by means of a leucocytometer pipette, blood being drawn up to the level of the first graduation mark (0.1), and bouillon or peptone water up to the mark 1. Or if a larger quantity of serum be available, we may draw serum up to the mark 1 on the leucocytometer pipette and bouillon up to the mark 11.

An alternative method of diluting the serum is that performed by means of a platinum loop. A single loopful of the serum is placed on a perfectly clean slide, and nine loopfuls of bouillon having been laid on the slide around it, the whole are mixed together.

By the employment of methods (2) and (3) we obtain the serum

in an exact dilution of 1 in 10; in the case of method (1) the degree of dilution is rather greater, because nine parts of bouillon were added, not to one part of serum, but to one of blood.

By means of a sterilised platinum loop, we next take one loopful of the twenty-four hours' culture of *B. typhosus* on agar, and mix it thoroughly with two or three loopfuls of bouillon on a perfectly clean slide, until a bacterial emulsion of uniformly thick turbidity is produced. The platinum loop is then sterilised in the flame, and allowed to cool; one loopful of the diluted serum is placed on a clean slide, and after the loop has been again sterilised and allowed to cool, four loopfuls of the bacterial emulsion are placed around the loopful of diluted serum, and the whole is thoroughly mixed. The dilution of the serum is now 1 in 50. One or two loopfuls of the mixture are transferred to the centre of a clean cover-glass. A slide, the central depression of which has been ringed around with vaseline, is inverted and lowered on to the cover-glass; and when the slide, with the cover-glass adhering to it, is now reinverted, the drop depends from the under surface of the cover-glass as in Fig. 197, page 440.

If it be desired to employ a degree of dilution other than 1 in 50, we may obtain a dilution of 1 in 20 by mixing 1 loopful of serum (diluted to 1 in 10) with 1 loopful of the bacterial emulsion; a dilution of 1 in 30 is got by mixing 1 loopful of serum (diluted to 1 in 10) with 2 loopfuls of the bacterial emulsion; and a dilution of 1 in 100 is effected by mixing 1 loopful of serum (diluted to 1 in 10) with 9 loopfuls of the emulsion. But as a general rule a dilution of 1 in 50 is the best for diagnostic purposes.

The preparation is now examined under the microscope with a magnification of about 400 diameters. The presence of a *few* red blood corpuscles in the field does not interfere with the reaction, and greatly facilitates the focussing of the drop. As the bacteria are unstained, the iris diaphragm must be contracted for the reason explained on page 441.

If the serum were not derived from a case of typhoid fever, the bacilli are observed to be scattered uniformly throughout the drop and to move actively through it; they preserve their motility unimpaired for hours. In this case there is no agglutination reaction; in other words, Widal's reaction is not present.

But if the patient from whom the serum was obtained be a case of typhoid fever, the bacilli soon lose their motility, and adhere to one another so as to form clumps, which are small at first, but soon increase in size. When the clumping is complete

no motile bacilli can be found in the fluid intervening between the clumps. The agglutination test (Widal's reaction) is regarded as positive if, within one hour, there is complete clumping of the bacilli by means of a serum in a dilution of 1 in 50, or within thirty minutes when the degree of dilution was 1 in 30.

A control test, with serum taken from a normal person and used in the same dilution, should always be performed simultaneously. In the control there should be no agglutination of the bacilli.

In interpreting the results of the agglutination test there are a number of important facts to be borne in mind.

All strains of typhoid bacilli are not agglutinated with equal readiness by the sera of typhoid patients. We should therefore obtain a culture which is known to give a good reaction with typhoid serum. We make a subculture once a week, incubate it for twenty-four hours at 37° C., and afterwards keep it at the room temperature.

While a positive reaction is obtainable in 90 per cent. of cases of typhoid fever, it is seldom obtained until the seventh day of the disease; occasionally it is not obtained until the third or even the fourth week, and in exceptional cases not until convalescence has set in. But, in general, the agglutination reaction becomes more marked as the disease advances.

The severity of the case cannot be deduced from the degree of dilution in which the serum will still agglutinate typhoid bacilli. In some grave cases the reaction is not so pronounced as in others of lesser severity.

A normal serum in low dilution may agglutinate typhoid bacilli, and consequently to justify a diagnosis of typhoid fever, the serum should produce complete clumping in a dilution of 1 in 30 within half an hour, or within one hour when used in a dilution of 1 in 50.

It is also important to remember that for several months after recovery from typhoid fever the patient's serum may agglutinate *B. typhosus*, and, further, that a positive reaction may occasionally be obtained with the serum of patients suffering from other diseases than typhoid fever.

In paratyphoid fever, due to infection with *Bacillus paratyphosus*, where the clinical symptoms are closely similar to those of typhoid fever, the serum may agglutinate either *B. paratyphosus* or *B. typhosus*, or may fail to agglutinate one or other of these organisms. It may, therefore, be advisable to determine the diagnosis by isolation of the typhoid or paratyphoid bacilli, from the patient's urine, or preferably from blood obtained by

incision of one of the roseolar spots. The chief cultural differences between paratyphoid bacilli and typhoid bacilli are the brown growth of the former on potato and their thicker and more opaque colonies on gelatine.

Widal's reaction may also be performed by using a culture of *B. typhosus* which has been killed by the addition of formalin. One drop of the latter is added to every 150 drops of a bouillon culture, and the fluid having been filtered in order to remove any clumps of bacilli, is then stored in glass tubes. With either living or killed cultures the test may also be performed by observing the appearance of *sedimentation*—the microscopic evidence of clumping—in capillary tubes filled with bacterial emulsion and diluted serum.

The agglutinative power of the serum on other organisms may be tested in a manner similar to that employed in the diagnosis of typhoid fever. We may thus determine the agglutination of cholera vibrios in cases of cholera, of *B. coli communis* in cases of general infection with that bacillus, and of pneumococci in cases of acute lobar pneumonia and of pneumococcal septicæmia. The agglutination of *Micrococcus melitensis* may be regarded as conclusive evidence of Mediterranean fever (Malta fever), for this coccus is not agglutinated by the sera of patients suffering from other diseases.

Method of Estimating the Opsonic Index

In order to comprehend and appreciate the significance of the opsonic index, a certain knowledge is required of the defensive mechanism of the body to bacterial infection.

We know that in certain diseases, *e.g.* diphtheria and tetanus, a toxæmia arises when the specific toxins elaborated by the bacteria enter into chemical combination with some of the cells of the body. Those cellular elements may respond by producing antitoxin, which enters into chemical combination with the toxin and thereby renders it inert.

In other diseases, *e.g.* cholera, the serum of the convalescent patient contains not only a normal pre-existent element termed *complement*, but newly-formed substances known as *lysins*, and the lysin and complement in combination effect the disintegration (lysis) of the invading organisms. With antitoxins and with bacteriolysins we are, however, not now concerned.

The question we have to consider is the defensive mechanism of the body against such bacteria as *B. tuberculosis*, *B. coli communis*, staphylococci, gonococci, etc. It has not yet been

proved that either antitoxins or lysins are formed in the course of the infective diseases caused by the bacteria just mentioned.

Although Metchnikoff's observations on phagocytosis are undoubtedly correct in so far as certain cells of the body, notably polymorphonuclear neutrophile leucocytes and endothelial cells, may ingest and subsequently digest invading bacteria, our conception of the processes involved in phagocytosis have been materially modified since the phagocytic theory was formulated in 1884. It has been demonstrated by Buchner and others that cell-free exudates, plasma, or serum may possess a bactericidal action in virtue of the element termed *complement* or *alexin* contained therein, and for several years the theory has prevailed that complement exerts a primary prejudicial influence on the vitality of invading bacteria, whereby they are the more easily ingested by phagocytic cells. According to this theory, which was advanced by Bouchard, and by Buchner and Hahn, phagocytosis is a secondary defensive process, not a primary one.

In a series of recent experiments, Douglas and Wright, Bulloch, and others, have confirmed the fact that the ingestion of bacteria by phagocytes is a secondary process. They contend, however, that the primary effect on the invading bacteria is not produced by complement, but by other substances which Wright has termed opsonins (from Latin *opsono*, I prepare food for). Opsonins, apart from any bactericidal action, modify bacteria in a manner which renders them a ready prey to phagocytic cells.

The opsonic action of plasma or serum on various bacteria has been extensively studied. Briefly stated, the procedure, which is a modification of Leishman's method of measuring the phagocytic power of the blood, consists in mixing certain volumes of serum, bacterial emulsion and washed leucocytes, and after incubation of the mixture at 37° C., preparing and staining films, counting the number of bacteria ingested in a certain number of polymorphonuclear neutrophile leucocytes, and striking an average per leucocyte. As it is impossible to compare the results of two tests when different emulsions of one and the same bacterium are employed, a control test must be carried out, using the same stock of leucocytes and of bacterial emulsion, but the serum of a normal individual instead of that of the patient; an average of bacteria per leucocyte is again struck. Both numbers are now divided by the average number of bacteria per leucocyte in the second (*i.e.* normal) film. The result is the opsonic index. For example, if the average number of bacteria per leucocyte in presence of the patient's serum be 3·3, and if

6·5 be the corresponding number per leucocyte in presence of normal serum, the index $= \frac{3 \cdot 3}{6 \cdot 5} = 0 \cdot 5$.

It is immaterial whether the leucocytes employed in the tests be derived from a normal person or not, and as the same bacterial emulsion is employed in both preparations, it is obvious that when the phagocytic activity of different blood sera is compared it is the serum which is the variable factor.

Technique of the Method.—The articles required are—

1. Capsules (see Fig. 200, p. 468) for collecting the sample of blood to be examined.

2. A pure culture of *B. tuberculosis*, staphylococci, or other micro-organism, according as we are going to estimate the opsonic index for tubercle bacilli, staphylococci, etc.

3. A centrifuge, centrifuge tubes, a sterile aqueous solution of 0·85 per cent. sodium chloride, and another containing 0·85 per cent. NaCl and 1 per cent. sodium citrate, for preparing washed leucocytes.

4. An agate pestle and mortar for preparing emulsions of tubercle bacilli.

5. Capillary pipettes fitted with india-rubber teats.

6. Microscope slides, watch glasses, and reagents for Ziehl-Neelsen's stain and for Leishman's or Jenner's stains.

In the capillary pipettes we are going to mix measured volumes of serum, bacterial emulsion and leucocytes. These are prepared as follows :—

I. *Serum.*—The patient's blood having been drawn into one capsule, and blood from a normal individual into another, and clotting having occurred, the two sera are obtained in the same manner as when obtaining blood for Widal's reaction (see p. 468). In performing the control test it is sometimes advisable, instead of using serum from only one normal individual, to take blood from two or three normal persons into separate capsules, to mix their sera, and to use the pooled serum in the control test.

II. *Bacterial Emulsion.*—If we are about to test the opsonic index for tubercle bacilli, a small quantity of a sterilised culture of *B. tuberculosis* is ground up with a little sterile 0·85 per cent. sodium chloride solution in an agate mortar, until a milky fluid of uniform turbidity is obtained. This is then drawn into a glass capsule, such as is shown in Fig. 200, and centrifuged for four or five minutes, so that all clumps of bacilli may be deposited and only isolated bacilli remain suspended in the fluid, which then constitutes the bacterial emulsion. If we desire to test the opsonic index for staphylococci, gonococci, etc., we prepare the bacterial

emulsion by shaking up an agar culture of the bacterium in question in normal saline solution, and afterwards centrifuge the fluid so as to deposit all clumps of bacteria.

III. *Washed Leucocytes.*—Allow about ten drops of blood to fall into a centrifuge tube nearly filled with a sterile aqueous solution of 0·85 per cent. sodium chloride and 1 per cent. sodium citrate, mix thoroughly and centrifuge. Pipette off the clear fluid, and having filled the tube with 0·85 per cent. NaCl solution, mix and again centrifuge. Draw off the supernatant clear fluid, and then draw the upper layer of the deposit, containing a large proportion of leucocytes, into a capillary pipette, and transfer therefrom to a watch-glass. As has been already stated, it is immaterial whether the leucocytes be taken from one individual or from another.

The patient's serum, bacterial emulsion and washed corpuscles have now to be mixed. Into a graduated capillary pipette fitted with a rubber teat, draw three volumes of serum, one of bacterial emulsion, and three of washed corpuscles. These are mixed by being blown out on to a clean slide and re-aspirated several times in succession. Finally having drawn the mixture into the pipette, and having sealed its orifice in the flame, place it in the incubator at 37° C. for fifteen minutes. The point of the pipette is then broken off, the mixture is blown out on to a clean slide, and films are made on slides in the manner described on page 108.

The same procedure is again carried out, but with normal serum instead of the patient's serum, and films are prepared from the incubated mixture.

The films should now be fixed and stained. If we have used a bacterial emulsion of tubercle bacilli, the staining is performed by Ziehl-Neelsen's method, as described on page 438. When the emulsion was one of bacteria other than tubercle bacilli, the films are treated according to Leishman's or Jenner's methods (see pp. 110, 111). The films are then examined with a $\frac{1}{12}$-inch oil-immersion objective. The number of bacteria are counted in fifty polymorphonuclear leucocytes in both preparations—namely, in a film prepared from the mixture containing the patient's serum, and in another prepared from the mixture containing normal serum—and an average per leucocyte is struck in each preparation. We then divide the number of bacilli ingested per leucocyte in the presence of the patient's serum by the number ingested per leucocyte in the presence of normal serum. The resulting figure is the opsonic index.

For example, if there be an average of 2·5 bacteria per leucocyte

in the presence of the patient's serum, and of 5·5 per leucocyte in the presence of normal serum, the opsonic index $= \frac{2{\cdot}5}{5{\cdot}5} = 0{\cdot}45$.

In healthy individuals the opsonic index varies, as Bulloch has shown, only within small limits. Using tubercle bacilli as the test material, the minimal index of healthy persons is 0·8, and the maximal 1·2, the average being 0·97. A tuberculo-opsonic index below 0·8 or above 1·2 is regarded as abnormal. In many cases of tuberculosis the index is either too high or too low. In slight, early cases of phthisis, and also in many cases apparently cured, the tuberculo-opsonic index is usually found to be abnormally low, *e.g.* 0·4. In acute and in advanced cases of phthisis the index is often a fluctuating one.

Further diagnostic information may be obtained by ascertaining what changes the index undergoes after the patient has been injected with tuberculin T R. After an injection the index in all instances is at first lowered—there is a "negative phase" of opsonic power—but about twenty-four hours later this should be followed by a heightened index, indicating an increase of opsonic power. The depth and duration of the negative phase after injection is of especial diagnostic significance, for a negative phase which is unduly prolonged is very suggestive of tuberculosis.

The opsonic index for staphylococci, gonococci, etc., may be studied in a similar fashion, and the negative phase observed after the patients have received injections of dead cultures of those micro-organisms.

The opsonic index may thus be of considerable value in diagnosis, and the effect of injection of vaccines—tuberculin T R, dead cultures of staphylococci, etc.—can be controlled by determining the index at intervals before and after the injections are given.

CHAPTER XXXVII

EXAMINATION OF FLUIDS—CYTODIAGNOSIS.

By a study of the cellular elements in exudates, transudates, and cerebro-spinal fluid, considerable light may often be shed on the nature of the disease from which the patient is suffering. This method of investigation has attained increasing importance since Widal first drew attention to its diagnostic value in 1900.

Pleural Fluid

Methods of Examination.—The fluid which has been obtained from the pleural cavity by means of an exploring syringe or aspirator, in the manner described on page 461, should be examined as soon as possible after it has been drawn off. If it has not yet coagulated, a portion is immediately centrifuged, preferably in tubes which taper towards the bottom. In order to prevent coagulation it is well to add a few drops of a 5 per cent. aqueous solution of ammonium oxalate, or 1 per cent. sodium citrate, to a portion of the fluid as soon as it is obtained. If coagulation, however, has taken place, the cells will be entangled in the clot, and should be liberated by thoroughly shaking up the clot with glass beads in a tube; but this method is not entirely satisfactory, because polymorphonuclear leucocytes may be broken up or may not be disentangled. It is therefore better either to centrifuge the fluid before it coagulates, or to add one of the above-mentioned fluids to prevent coagulation.

After having been well centrifuged, the supernatant fluid is decanted, and the deposit at the bottom of the tube is collected in a capillary pipette. Several thin films are then made either on slides or cover-glasses, the method of procedure being the same as that for making blood films (see pp. 108–110). The films are allowed to dry in the air, and are thereafter fixed and stained by Jenner's method, as described on page 110. Leishman's stain

is hardly so satisfactory, as it does not demonstrate the cytoplasmic granules of the polymorphonuclear leucocytes so well.

In order to demonstrate fatty degeneration of the cellular elements, which is of common occurrence, some of the films should be treated for five to ten minutes with either Sudan III or Scharlach roth (saturated solutions in 70 per cent. alcohol), which stain fat droplets red. The films are afterwards counterstained with a weak alcoholic solution of methylene-blue, in order to bring out the nuclei of the cells, washed and dehydrated in absolute alcohol, cleared in xylol, and mounted in xylol balsam. Or hämatein may be used as the counterstain in place of methylene-blue.

The films should be examined with a $\frac{1}{12}$-inch oil-immersion objective, although one giving a somewhat lesser magnification may be sufficient for diagnosis. Every part of the film should be systematically subjected to examination, so that the proportion which the different cells bear to one another may be ascertained. In making the differential count of the cells, a mechanical stage is of considerable assistance.

The chief cells which may be found in films prepared from pleural fluids are cells derived from the blood—polymorphonuclear neutrophile leucocytes, lymphocytes, large mononuclears and red blood corpuscles—and endothelial cells derived from the wall of the serous cavity.

Polymorphonuclear neutrophile leucocytes.—These cells may be identical in appearance with the polymorphonuclear neutrophiles of normal blood (see Plate I., 19 and 20), but have often undergone degenerative changes. The most important of those alterations are—

(1) *Fatty degeneration of the cytoplasm.*—If the film has been stained by Jenner's or Leishman's methods, the cytoplasm appears to contain a number of small vacuoles; but if the films were stained with Sudan III or Scharlach roth, the fatty droplets are stained red.

(2) *Fragmentation of the nucleus.*—The nucleus often appears to lose its intracellular network and to stain both more diffusely and more deeply than normal. At the same time the nucleus commonly breaks up into globular fragments, each being surrounded by a zone of cytoplasm in which no granules can be detected, and which is therefore homogeneously stained with the eosin of the staining fluid. Such fragmented polymorphonuclear leucocytes may be mistaken for lymphocytes, and are sometimes spoken of as "pseudolymphocytes."

The red blood corpuscles and *lymphocytes* resemble those of

the blood (see Plate I.). The same holds good for the *large mononuclear leucocytes*, except that they are not infrequently seen to contain bacteria which they have ingested. These cells seldom undergo degenerative changes.

Endothelial cells from the surface of the pleura.—Although varying considerably in size, these cells are usually two or three times as large as the polymorphonuclear leucocytes, and of round, oval, or somewhat angular form. Unless it has become markedly degenerated, each endothelial cell is seen to contain a single nucleus with a well-defined chromatin network and two nucleoli. The amount of cytoplasm around the nucleus is relatively large, but has often undergone fatty degeneration. Evidence of the phagocytic activity of these cells may be discernible. Endothelial cells may be mistaken for large mononuclear leucocytes, and, when much degenerated, for lymphocytes.

The absolute and relative number of the different cells varies according to the nature and intensity of the disease.

If the number of cellular elements be scanty, and if they are almost all endothelial cells, the fluid is probably a dropsical **transudate**, such as is obtained from a case of hydrothorax due to cardiac failure or to Bright's disease.

As regards pleuritic **exudates**, the relative proportion of the different leucocytes depends on the stage and degree of the inflammatory reaction rather than on the cause. Thus while lymphocytes are usually the chief cells in an exudate of *tuberculous* origin, polymorphonuclears may predominate during the first two days of the disease, or, if the inflammation be acute and intense, these cells may be more numerous than any others for a longer period of time. In tuberculous pleurisy a preponderance of polymorphonuclear leucocytes does not necessarily imply that there is a mixed infection, as was formerly thought to be the case, because, as a rule, it is merely evidence of an inflammatory reaction more acute and intense than is observed in most cases of tuberculous pleurisy.

In about 80 per cent. of *non-tuberculous* pleuritic exudates, *e.g.* those due to pneumococci and streptococci, the polymorphonuclear neutrophile leucocytes are in great excess of all other cellular elements, and their number is usually proportionate to the severity of the disease. In the late stages of the disease, proceeding to a cure, the absolute and relative number of polymorphonuclear leucocytes gradually decreases, while that of the lymphocytes increases simultaneously. In purulent exudates the cells are found to be markedly degenerated.

Cytodiagnosis is thus an aid in the investigation of the nature

of the pleural effusion, but as the interpretation of the results furnished by a single examination may be somewhat uncertain, the examination should be repeated at intervals of about a week, and the information thus obtained should be controlled by bacteriological examination of the fluid (see p. 461), and by a careful inquiry into the patient's history, symptoms, and physical signs.

In **malignant pleurisy** the picture which the film may present is somewhat variable. In some instances polymorphonuclear neutrophile leucocytes and red corpuscles are the predominant cells; in others we may find large polymorphic cells which are often markedly degenerated and which are derived from the pleural endothelium. In yet other instances the appearance of these large cells suggests the probability of their origin from the tumour itself, and rarely these cells are grouped together so as to form small masses.

Pericardial Fluid

This should be obtained in the manner described on page 461. The technique of the examination is identical with that of pleural fluids, and in their interpretation the results are comparable to those obtained from cytological examination of pleural fluid.

Peritoneal Fluid

The mode of obtaining the fluid has been described on page 461; the methods of examination are the same as those for pleural fluids.

The interpretation of the results is somewhat uncertain and indefinite. In *ascites* the predominance of endothelial cells may be largely counteracted by the presence of many lymphocytes and polymorphonuclear neutrophile leucocytes. In *tuberculous peritonitis* either of these two varieties of leucocyte may be the chief cellular element. In establishing a diagnosis of the nature of the fluid, more help is usually furnished by bacteriological than by cytological examination, and especially so in the case of purulent fluids.

Cerebro-Spinal Fluid

The examination of the cerebro-spinal fluid is often of great assistance in diagnosis. The method of obtaining the fluid by means of lumbar puncture has been already described on pages

462 and 463. The rate at which the fluid flows from the exploring needle should be noted. It varies with the bore of the needle and with the position of the patient, but if the bore of the needle be constant and the puncture is made while the patient is lying horizontally on his left side, the rate at which the fluid flows is proportionate to the pressure in the subarachnoid space. In meningitis, for example, the fluid may gush out in a stream of considerable force.

Colour.—This is the first point to note after obtaining the fluid. Normal cerebro-spinal fluid is colourless, like water. It may be tinged with blood, as in cases of intra-cerebral or of meningeal hæmorrhage. It is possible, however, that the admixture of blood may be due to the needle having injured some of the subdural veins. In such instances usually only the first portion of the fluid is markedly sanguinolent, while the remainder, received into separate tubes, is not blood-tinged. If, on the other hand, the fluid in all the tubes is blood-stained, it is almost certain that the cerebro-spinal fluid does contain blood, and that the latter is not due to the exploratory puncture. Another means of ascertaining the source of the blood is to note whether the fluid coagulates in the tube. It does so only when, as an accidental result of the puncture, fresh blood has mingled with the fluid.

The fluid may have a dark amber colour when it is withdrawn from two to eighteen days after a cerebral hæmorrhage. A yellowish or greenish-yellow colour may be observed in some cases of chronic meningitis and of tumour of the spinal cord. In jaundice the fluid is not usually bile-stained.

Transparency.—Normal cerebro-spinal fluid is perfectly clear. Turbidity is a sign of meningeal inflammation. In purulent meningitis the turbidity is well marked; in tuberculous meningitis there is very slight turbidity, or the fluid may even be quite clear.

Chemical Examination.—There is only a trace of albumin in the normal fluid, but a well-marked cloudiness is usually produced on boiling the fluid taken from cases of meningitis, hydrocephalus, cerebral tumour, tabes and general paralysis of the insane. In meningitis the amount of albumin may be very considerable; but in all the above-mentioned diseases it may sometimes be found that the cloudiness produced on boiling is not appreciably greater than that obtained with normal fluid.

The recognition of the reducing substance contained in small amount in the cerebro-spinal fluid is of no diagnostic significance.

Bacteriological Examination.—This has been already considered on pages 463 and 464. Here we may note that in sleeping sickness, *Trypanosoma gambiense* is found in the centrifuged cerebro-spinal fluid. The films should be stained with Leishman's or Giemsa's stains. The *T. gambiense* is an actively motile protozoan parasite. It has a spindle-shaped body, terminating at one end in a long flagellum, and has on one aspect a delicate undulating membrane, prolonged into the latter. The large macronucleus is situated near the centre of the body.

Cytological Examination, Cytodiagnosis.—This is the method of examination which is in general of greatest diagnostic value. The fluid having been centrifuged for ten to fifteen minutes, is decanted, and, while the tube is still inverted, the deposit is drawn into a capillary pipette, and transferred therefrom to two or three slides. The films are allowed to dry in the air, and then fixed and stained by Jenner's, Leishman's, or other stain, as described on pages 110–112. The preparations should be examined with a $\frac{1}{12}$-inch oil-immersion objective.

In a preparation of normal cerebro-spinal fluid, very few cells are visible—not more than two or three in each field.

If they are more numerous, there is some departure from the normal. We often find that nearly all the cells are lymphocytes, with an occasional polymorphonuclear leucocyte and endothelial cell, this being the condition known as a *lymphocytosis*. In other instances the polymorphonuclear neutrophile leucocytes predominate; in other words there is a *polymorphonuclear leucocytosis*. As in the case of the pleura, so with the meninges the relative number of lymphocytes and polymorphonuclear leucocytes depends on the stage and still more on the intensity of the inflammatory process. At the commencement of a tuberculous meningitis there is a transient polymorphonuclear leucocytosis, which, however, is soon replaced by a lymphocytosis. This is subsequently constant. In non-tuberculous meningitis, on the other hand, the inflammatory reaction being more acute, there is a well-marked polymorphonuclear leucocytosis throughout, the only exceptions being those rare cases in which the patient recovers, when the improvement in the patient's condition is attended with a gradual disappearance of the polymorphonuclears and a simultaneous increase in the number of lymphocytes. In cases of meningitis the examination of the cerebro-spinal fluid should be repeated at intervals of two or three days in order to study the absolute and relative number of the cellular elements.

A lymphocytosis is constant in syphilitic disease of the central nervous system and in general paralysis of the insane, and is almost invariable in tabes. The lymphocytosis is one of the earliest signs of those diseases, and its recognition is of considerable value in their differentiation from multiple neuritis, epilepsy, and the various neuroses and psychoses in which the cytological examination of the fluid does not reveal any abnormality.

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