MEDICAL DIAGNOSIS

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PREFACE

MEDICINE is year by year receiving additional help, more especially in diagnosis, from the ancillary sciences. In the ordinary text-books on medicine space does not permit of adequate reference to a number of laboratory methods —whether bacteriological, chemical or physical—side by side with sufficient clinical and pathological detail. Our publishers informed us of their opinion that a book, in which all the clinical information and the more ordinary laboratory details, necessary for the purpose of making a scientific diagnosis in medical cases, were arranged in a concise and accessible form, was constantly sought for both by the student and the practitioner. At their request we have made an attempt in the following pages to meet this demand. We have availed ourselves freely of existing text-books and current medical literature ; but do not feel that an extensive bibliography would serve any useful purpose in a book of this size and scope.

We take this opportunity of expressing our indebtedness to Dr. Thomas Lewis for his permission to make free use of his book on " Clinical Disorders of the Heart Beat " in the section dealing with certain forms of cardiac irregularity ; to Dr. Charles Slater and Dr. E. J. Spitta for permission to reproduce thirteen micro-photographs from their atlas of bacteriology ; to Dr. Lindley Scott for permission to use fourteen plates from his atlas of urinary deposits ; to Dr. J. W. Linnell for the pulse-tracings in the chapter on diseases of the heart ; to Dr. John Parkinson for the electro-cardiogram illustrating heart-block ; and to Miss Seymour,

of St. George's Hospital, for the preparation of a number of temperature charts, the design of which is due to Miss Fullerton.

Messrs. Churchill have supplied the coloured plates of the malarial parasite from Dr. Panton's work on Clinical Pathology, and Figs. 71 and 72 from Dr. Aldren Turner and Dr. T. Grainger Stewart's text-book on Nervous Diseases.

We have also to thank Dr. H. W. Warden, house-surgeon at St. George's Hospital, for the beautiful original drawings which illustrate the diseases of the blood and the diseases of the nervous system, etc., and, finally, Messrs. Churchill for their long-suffering patience and courtesy.

A. L.
J. A. T.

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MEDICAL DIAGNOSIS

PART I

CHAPTER I

SPECIFIC INFECTIVE DISEASES

I. ACTINOMYCOSIS.

Definition. A chronic infective disease due to the Actinomyces bovis (a streptothrix organism) and characterised by the presence of granulomatous abscesses.

Diagnosis. The infection usually occurs in the mouth, particularly in relation to a carious tooth, consequently the abscess is most frequently seen in the jaw or neck; but in view of the fact that the organism may be inspired or swallowed, the lesion may be met with in any organ of the body. A case has been recorded of actinomycotic disease of the liver secondary to infection per vaginam.

In making a diagnosis the history of exposure to infection is very important, since the disease is almost always contracted by chewing infected grain, and is therefore usually met with in farm labourers and cattle tenders or in threshers, who may inhale the infected dust. The specific lesion, if superficial, is seen as a brawny, indurated swelling which ultimately suppurates and breaks down with the discharge of pus, in which are golden-brown granules, and the formation of intractable sinuses.

The diagnosis can only be established with certainty by the microscopical examination of the pus from one of the abscesses, preferably by teasing out one of the minute granules mentioned above, when the streptothrix filaments will be seen as Gram-positive branching threads in the film

preparations. Prior to bacteriological examination actinomycosis of the mouth or neck may be indistinguishable from sarcoma, tertiary syphilis, chronic septic infection or tuberculous periostitis. In such cases valuable information may sometimes be gained by excision of a small piece of the affected tissues for microscopical examination (biopsy), by the performance of the Wassermann reaction, or by the use of the special methods for the diagnosis of tuberculosis.

In addition to the ordinary form of actinomycosis described

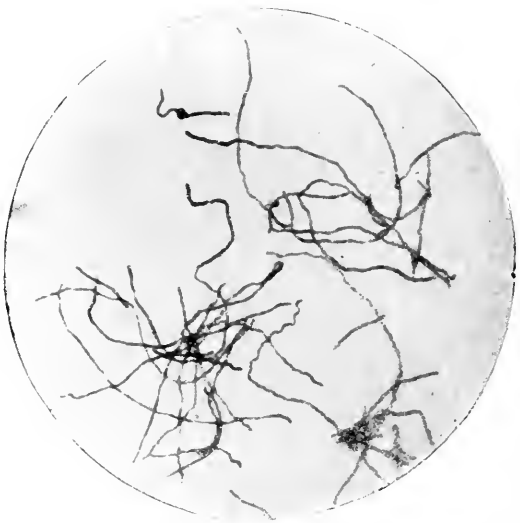


Fig. 1.—Actinomyces Hominis in pus from an actinomycotic abscess. Magnification × 1000.

above, visceral actinomycosis may be met with in the following varieties :—

Respiratory actinomycosis, which may simulate bronchitis, tuberculosis, or bronchiectasis, and in which the diagnosis depends on an examination of the sputum ; while the causative organism of an empyema can be discovered by the bacteriological examination of the pus.

Alimentary tract actinomycosis, which usually commences by ulceration near the cæcum and which secondarily may lead to *liver abscess*. The diagnosis here can only be made by exploratory laparotomy or by acupuncture.

Cerebral actinomycosis, which is indistinguishable from any other form of brain abscess or tumour.

Cutaneous actinomycosis, which may simulate a cutaneous tuberculide or syphilitic ulceration. The estimation of the opsonic index before and after exercise and the performance of Wassermann's reaction may assist the diagnosis in these cases.

II. ANTHRAX.

Definition. A specific infective disease caused by the Bacillus anthracis, which is a long motile Gram-positive organism.

Varieties. (i.) Malignant pustule, acquired by inoculation.

(ii.) Wool-sorters' disease, acquired by inhalation.

(iii.) Splenic fever, acquired by ingestion.

The incubation period is from three to six days.

Diagnosis. The history is important in so far as anthrax is a disease of herbivorous animals and is conveyed to man through infected wool, hides and meat; it is therefore especially met with in wool-sorters, tanners and butchers.

(i.) The *Malignant pustule* is a single lesion which starts as a papule, soon vesiculates, and in about thirty-six hours dries to a black scab, which is situated in the middle of a red œdematous area and is surrounded by a ring of secondary vesicles.

Suppuration does not occur. Lymphangitis and glandular enlargement follow, but there are no constitutional disturbances for from two to four days, when rigors, pyrexia, and vomiting indicate a general blood infection. In untreated cases death occurs in about one week, but for some time before the end the fever abates, while the absence of pain throughout is an important feature.

The characteristic bacilli are abundant in the vesicles, and later on can often be recovered from the blood. Occasionally the distinctive pustule may be absent, a diffuse brawny induration alone being present; these cases closely resemble ordinary cellulitis, or acute spreading œdema. The absence of pus and the falling temperature are important diagnostic points; while the most likely cause for spreading œdema is the Bacillus aerogenes, in which case the formation of gas causes crepitation in the tissues.

(ii.) *Wool-sorters' disease* starts suddenly with chill, prostration, severe pains in the chest and back, and a temperature of 102°—103°; there is some bronchitis, dyspnœa, cough and not infrequently a blood-stained expectoration, while the pulse is rapid and feeble. The prostration is more severe than the signs would appear to warrant, and death may ensue in from twenty-four hours to one week.

(iii.) *Splenic fever.* In addition to the symptoms of profound general toxæmia described above, the patient will have severe abdominal pain, diarrhœa, vomiting, and a rapidly enlarging spleen. Hæmorrhages under the skin and from the mucous surfaces may precede death.

Fig. 2.—Bacillus Anthracis from Bouillon culture, showing spore formation. Magnification × 1000.

In the differential diagnosis of the two latter varieties the history of possible exposure to infection is of paramount importance; the blood and urine should be examined for the bacilli, and if the result is negative some of the blood should be injected into a white mouse, when, if the disease is anthrax, the animal will die in a few days and the bacilli can be recovered from its blood.

III. CEREBRO-SPINAL MENINGITIS.

A specific infective disease characterised by inflammation of the meninges of the spinal cord, and more especially

of the base of the brain, and caused by the Diplococcus intracellularis of Weichselbaum.

Under this heading will be included—

(i.) The epidemic form, which is sometimes accompanied by a purpuric rash, and has therefore been called "spotted fever."

(ii.) The sporadic form, which is particularly likely to attack young infants, and which is then commonly known as "posterior basic meningitis."

Diagnosis. (i.) The *epidemic form*. The onset is usually sudden, with very severe headache, vomiting, and pains and stiffness in the back of the neck and in the limbs. There is not infrequently a rigor, and the temperature rapidly rises to 103° or more, and subsequently runs a course that is strikingly irregular. The patient is usually a child or a young adult. Herpes labialis is common, while the presence of a petechial rash is suggestive. There is early retraction of the head, which rapidly passes into opisthotonos; at the same time muscular rigidity and tremors become evident. Photophobia and ocular paralyses, usually of the external recti, are common, and the superficial reflexes are very often diminished or absent. Mental symptoms, even maniacal delirium, are of frequent occurrence, but commonly coma supervenes after three or four days.

There is an invariable leucocytosis, which persists throughout the entire course of the disease. The duration of the illness may be from a few hours to several months, and the younger the patient the more probable is a fatal issue.

Several clinical varieties are recognised in addition to the ordinary form described above.

(*a*) A *malignant form*, in which the pyrexia is moderate and the pulse slow, while a purpuric rash is particularly common. In this variety death occurs in from twelve to seventy-two hours.

(*b*) An *abortive form*, in which the patient enters upon a rapid convalescence about the fifth day, notwithstanding previous meningeal symptoms of extreme severity.

(*c*) An *intermittent form*, in which there are periodic exacerbations of pain and temperature for many weeks.

(ii.) The *sporadic form* or *posterior basic meningitis*.

Apart from the absence of an epidemic, the infrequency of a skin eruption, and the fact that the vast majority of the cases occur in infants, the course of this disease is very similar to that of the epidemic form described above. Although the mortality is extremely high, the course of the disease tends to be chronic. Extreme emaciation of the patient is a distressing feature of these cases. In patients who are sufficiently young the bulging anterior fontanelle bears witness to the rapid rise in intracranial tension. Optic neuritis is rare.

Kernig's sign is said to be of special diagnostic import in basic meningitis. This sign consists in the inability of the patient to extend the legs on the thighs when the thighs have been first flexed on the abdomen. In our experience the value of this sign has been overestimated and is largely restricted to cases in which the patient is a young child.

Lumbar puncture is the quickest and most satisfactory method of establishing the diagnosis. The object of this procedure is to withdraw some of the cerebro-spinal fluid from the lumbar region of the theca spinalis. The cerebro-spinal fluid is under pressure and turbid or even purulent. It should be collected in a sterile vessel and submitted to microscopical examination without delay. The cellular content is found to be much increased, the cells are polymorphonuclear leucocytes, and the characteristic organism, an intracellular Gram-negative diplococcus, will usually be visible in the films of the centrifugalised deposit. If some of the fluid is inoculated on to the surface of an agar tube, freshly smeared with human blood and incubated at 37° C., a growth of the organism can usually be obtained.

The diseases most likely to be confounded with cerebro-spinal meningitis are the following :—

Tuberculous Meningitis. This is not so common in the first year of life and does not occur apart from tuberculosis elsewhere, the signs of which may be susceptible of recognition : retraction of the head is less marked, and the course is usually shorter than in cerebro-spinal meningitis ; further, tubercles may be present on the choroid, and the fluid from the lumbar puncture is limpid and sterile and contains an excess of lymphocytes.

Enteric Fever. The absence of a leucocytosis and the presence of Widal's reaction at the end of the first week serve readily to differentiate this disease from cerebro-spinal meningitis.

Pneumonia usually presents suggestive signs in the chest, and such extreme rapidity of respiration is rare in meningitis ; but pneumonia, and especially apical pneumonia in children, is often accompanied by meningismus, which may closely simulate meningitis ; further, pneumococcal pneumonia may be accompanied by pneumococcal meningitis, and cerebro-spinal meningitis is occasionally complicated by pneumonia due to the Diplococcus intracellularis.

Lumbar puncture will settle the diagnosis, for the cultural characteristics of the two organisms differ widely, though polymorphonuclear leucocytes will be present in both cases.

Septic meningitis does not occur apart from some septic focus, such as otitis media or mastoid disease.

IV. CHICKEN-POX.

Definition. A specific infective disease, the causative organism being as yet undiscovered, which is characterised by a vesicular eruption.

Incubation Period. The incubation period of chicken-pox is rather variable, but fourteen days may be considered an average time.

Diagnosis. The disease usually attacks children of from two to six years, and one attack nearly always confers complete immunity. There is sometimes a trifling malaise or pyrexia for about twenty-four hours, but often the first thing observed is the appearance of a sparse papular eruption, most evident about the thorax and abdomen. In a few hours the papules develop into vesicles, the contents of which become turbid after two days and dry up into brown scabs, without suppuration, a few days later. These scabs drop off about the tenth day, and do not leave a scar unless secondary infection has been permitted, as by scratching. The spots, which do not become confluent, appear in little showers at intervals of about twelve hours, and may thus be

seen in all stages of development at one and the same time. They frequently occur on the mucous membranes, and vary in total number from ten or twelve to several hundreds. When the spots are very numerous the severe itching causes great distress. A slight rise in temperature is liable to occur as each successive crop of spots makes its appearance.

In very exceptional cases the vesicles of chicken-pox may become bullous and simulate pemphigus for a few days. A grave but happily rare sequel of chicken-pox is a condition known as "Gangrenous Dermatitis," in which there is a spreading superficial gangrene of the skin. A fatal result, due presumably to septic absorption, ensues in the majority of such cases.

It should be remembered that when chicken-pox attacks an adult it may be attended by much more severe constitutional disturbance; a temperature of 104° is by no means uncommon, and the spots may be so numerous as practically to cover the whole body.

Differential Diagnosis. Chicken-pox must not be confounded with the following conditions:—

(i.) *Small-pox.* Attention to the following points should serve to prevent this mistake:—

In the absence of an epidemic small-pox is most unlikely. In small-pox the onset is attended by severe constitutional disturbance; the rash appears on the third day and all at once, while it does not become vesicular till the sixth day, and it is most marked on the face and hands. Prior to their appearance the papules can be felt as shotty nodules underneath the skin. After the rash has appeared the temperature falls for a while until suppuration in the vesicles causes it to rise again on the eighth or ninth day, while just before the vesicles become purulent they are definitely umbilicated. Lastly, there is a red inflammatory areola around the pocks.

(ii.) *Varioloid*, or *Small-pox in the vaccinated.* In this condition the presence of vaccination marks and an epidemic of small-pox is of importance, while the invasion lasts longer than in chicken-pox and the rise in temperature is more abrupt. The vesicles are preceded by shotty papules and are most likely to occur in one eruption on the face and hands, while the temperature usually falls by crisis as soon as the rash

has appeared and does not show slight exacerbations with the appearance of successive crops as happens in varicella.

(iii.) *Herpes Zoster*. The vesicles here follow accurately the distribution of a sensory nerve-root, and there is considerable local pain.

V. DIPHTHERIA.

Definition. A specific infective disease due to the Klebs-Loeffler bacillus, characterised by inflammation of the naso-pharynx, fauces or larynx, and leading to coagulation necrosis and the formation of a false membrane at the site of infection.

Etiology. Diphtheria is particularly a disease of children under ten years of age. It occurs in epidemics, but is also endemic in all large towns, while a certain number of sporadic cases occur in which all efforts to trace the source of infection are fruitless.

The Klebs-Loeffler bacillus is a small rod-shaped bacillus which stains irregularly with all ordinary stains. It grows well on all ordinary culture media, but especially well on Loeffler's inspissated blood serum. The bacillus does not enter the blood stream; the symptoms therefore are due to toxæmia.

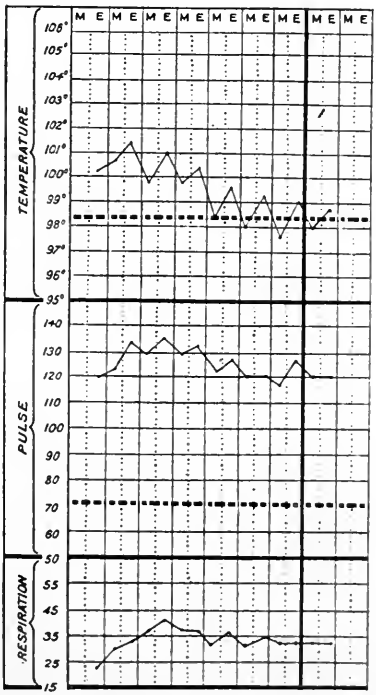


FIG. 3.—Chart from a case of Diphtheria treated with antitoxin at the beginning of the second day. Note the rapidity of the pulse in contrast to the slight pyrexia.

Incubation Period. Usually two days (thirty-six hours to seven days).

Course. The invasion may be abrupt, with shivering, vomiting, etc., but more often it is gradual, with malaise, headache, and loss of appetite.

Soreness of the throat may be trifling at first ; indeed, it is frequently not complained of at all by small children. Examination will reveal swelling and redness of the fauces, with often a patch of dirty, greyish-white membrane on a tonsil or on the soft palate or faucial pillars. The membrane shows a great tendency to spread to neighbouring parts. The glands about the angles of the jaw tend to be enlarged. If the membrane is picked off, a bleeding surface is exposed, which rapidly becomes re-covered by membrane.

The child soon presents the dull, earthy pallor of profound toxæmia.

The temperature is not high, usually between 101° and 102°, but the pulse is very rapid, about 135 per minute, and of poor quality. The importance of this pulse temperature ratio is very great.

Myocardial degeneration, as shown by cardiac dilatation, may be evident at an early stage.

The urine is of the febrile type, high coloured and scanty, and slight albuminuria is so common as to be regarded as a usual manifestation of the disease.

The knee-jerks at first are increased, but in a few days become diminished or even absent.

Varieties. (i.) *Faucial*, as described above.

(ii.) *Laryngeal.* Usually a complication of the faucial variety, but it may occur as the sole affection. The signs of laryngeal involvement are laryngeal stridor, increasing cyanosis and dyspnœa, restlessness and increasing rapidity of the pulse rate, and intercostal recession and sucking-in of the lower ribs on each inspiration.

(iii.) *Nasal diphtheria* is usually an accompaniment of ordinary or faucial diphtheria, but may exist alone.

The constitutional disturbance in this last variety may be slight ; a nasal discharge, and especially a blood-stained nasal discharge, may be the only sign. It is not too much to say that every chronic nasal discharge in children should be cultivated for the Klebs-Loeffler bacillus. The course tends to be very protracted.

(iv.) *Diphtheria of the Conjunctiva.* A membranous conjunctivitis may be due to diphtheria.

Complications. Complications are not numerous: the most important are :—

(i.) *Heart Failure.* This may occur as early as the third day of the disease, but is most to be feared in the second week. It is due at this stage to toxic myocardial degeneration and may manifest itself with alarming suddenness; arrhythmia develops, with increasing rapidity and feebleness of the pulse, and is quickly followed by the signs of extreme cardiac dilatation (*vide* p. 236).

The effect of the toxins on the heart muscle may be

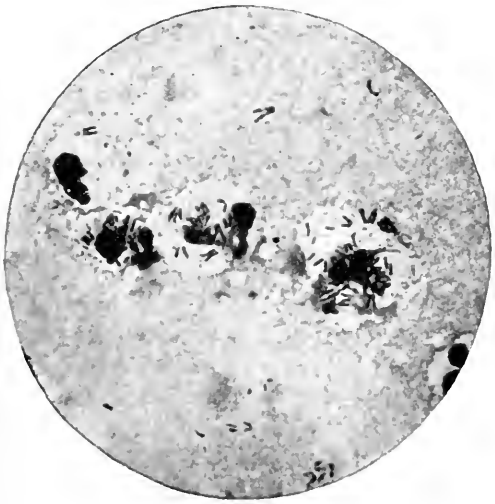


Fig. 4.—Bacillus Diphtheriæ in Nasal Secretion. Magnification × 1000.

apparent for many weeks in a case in which the course has otherwise been satisfactory. The symptoms here will be irregularity of the cardiac action, especially after exertion, alteration of the spacing of the heart sounds either in the direction of a "tic-tac" rhythm, caused by prolongation of systole at the expense of diastole, or the opposite state of affairs when the sounds come close together and the diastolic pause is unduly prolonged.

After the third week heart failure may manifest itself in yet another manner—that is to say by neuritis or

post-diphtheritic paralysis affecting the vagus nerve. It is extremely rare for this to occur at all, and rarer still for it to happen as an isolated palsy.

(ii.) *Post-diphtheritic paralysis* occurs in 15 per cent. of all cases, and its incidence is in no way determined by either the severity of the initial infection or the use of antitoxin. It appears as a rule about the end of the third or during the fourth week.

The muscles paralysed are usually affected in the following order :—

(*a*) *The Soft Palate.* In severe cases this permits regurgitation of liquids through the nose, and in milder cases imparts a nasal tone to the voice, most noticed in such words as "pig," which is pronounced as if spelled pihgh.

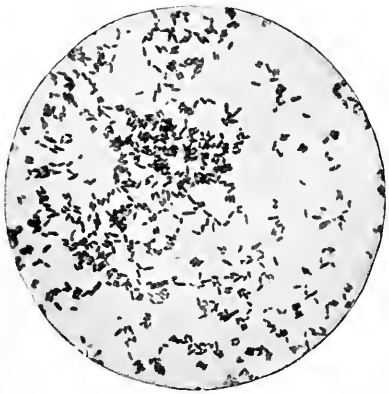


Fig. 5.—Bacillus Diphtheriæ. Seventy-two hours culture on gelatine at 21° C. Magnification × 1000.

(*b*) *The Internal Ocular Muscles.* This results in loss of power of accommodation of the pupil.

(*c*) *The Extensors of the Legs.* This produces a drop foot, and may prevent standing altogether.

(*d*) *The External Ocular Muscles.* This causes strabismus (usually convergent).

(*e*) *Involvement of the phrenic nerves, the intercostals, and the vagus.* This condition is a rare one.

It should be noted that diphtheria is not infrequently first diagnosed when paralysis occurs, the faucial affection having been so mild as to escape notice.

(iii.) *Broncho-pneumonia*, caused by inhalation of septic particles or by spread of the membrane down the trachea. It is most frequent in laryngeal cases and gives the usual signs of Broncho-pneumonia (*q.v.*).

(iv.) *Otitis Media.* This is liable to occur after any septic infection of the fauces, and the possibility of the Klebs-

Loeffler bacillus being present in a case of otorrhœa must be borne in mind.

(v.) *Acute Nephritis.* This complication is extremely rare and must not be confounded with albuminuria, which is almost invariable. The presence of blood and casts in the urine renders mistake impossible.

Diagnosis. The history of exposure to infection, or of other "sore throats" in the same house, is important; but the only certain method of diagnosing diphtheria is to isolate the characteristic organism from the nose or throat. For this purpose a swab is taken from the throat, or if any membrane is present a piece is picked off with forceps.

With this an even film is spread at once on a clean glass slide by gently rubbing the swab or membrane backwards and forwards; it is fixed by passage through a Bunsen flame and stained by Neisser's method, or with methylene blue, and examined under an oil-immersion lens.

In many cases a skilled observer can make a diagnosis from the fresh films, but the bacilli become so pleomorphic when older than twenty-four hours that considerable experience is necessary for their identification. Again, numbers of other organisms are always present. It is therefore usually desirable to obtain a culture of the organisms present. Accordingly a culture tube of inspissated blood serum is inoculated from the swab or membrane and incubated for twelve to fifteen hours at 37° C., at the end of which time a practically pure growth of Klebs-Loeffler bacilli will be obtained if diphtheria is present.

A membranous infection of the fauces may be produced by either streptococci or pneumococci, and conversely in true diphtheria membrane is sometimes entirely absent, while the *bacillus of Hoffmann* may produce a condition clinically indistinguishable from diphtheria. Bacteriologically Hoffmann's bacillus does not grow quite so quickly on Loeffler's blood serum as the diphtheria bacillus; it is shorter and broader, it has not got so definitely beaded an appearance and usually shows a central septum. It does not give polar staining by Neisser's method. Streptococci and pneumococci do not appear in culture on Loeffler's

blood serum for six or eight hours after the colonies of the diphtheria bacillus are present.

Follicular tonsillitis (*vide* also p. 316) has a more sudden onset than diphtheria; the exudate is usually yellowish and localised to the tonsillar crypts; the patches of exudate do not specially tend to run together. The flushed face, stiff and painful neck, and high temperature are all unlike diphtheria, and the toxæmia is not nearly so profound. A seemingly simple tonsillitis, however, may really be due to the diphtheria bacillus, and all such cases should be examined bacteriologically.

After the operation of removal of the tonsils the stumps are coated for some days with a greyish slough, which may cause apprehension in the mind of one not familiar with the fact.

Vincent's angina, which is commonly mistaken for diphtheria, presents the following picture:—A deep sloughy ulcer covered with a yellowish membrane, situated as a rule on or behind one tonsil and often accompanied by severe constitutional disturbance and a temperature of 103° or more. The diagnosis can readily be established by examining a fresh film stained with methylene blue, when the presence of large fusiform bacilli and numerous spirilla prove the case to be one of Vincent's angina.

Scarlet fever in the early stages may be difficult to diagnose from diphtheria, and it should be remembered that the two diseases may co-exist.

Bacteriological examination of the throat will settle the question. The more sudden onset with rigor and vomiting, the higher temperature, and the even more rapid pulse (temperature 104°; pulse 160) are greatly in favour of scarlet fever, while in thirty-six hours the characteristic rash should make its appearance (*vide* also "Scarlet Fever," p. 64).

VI. ENTERIC FEVER.

Definition. A specific infective disease due to the Bacillus typhosus of Eberth, and characterised by a septicæmia associated with ulceration of the intestine, inflammation of the mesenteric glands, enlargement of the spleen, bronchitis, and a papular eruption.

Incubation Period. Fourteen days (three to twenty-three days).

Course. The onset is nearly always gradual, the patient complaining of headache, chilliness, and general malaise. Epistaxis is fairly common, while vague abdominal discomfort and diarrhœa are frequently met with. In three or

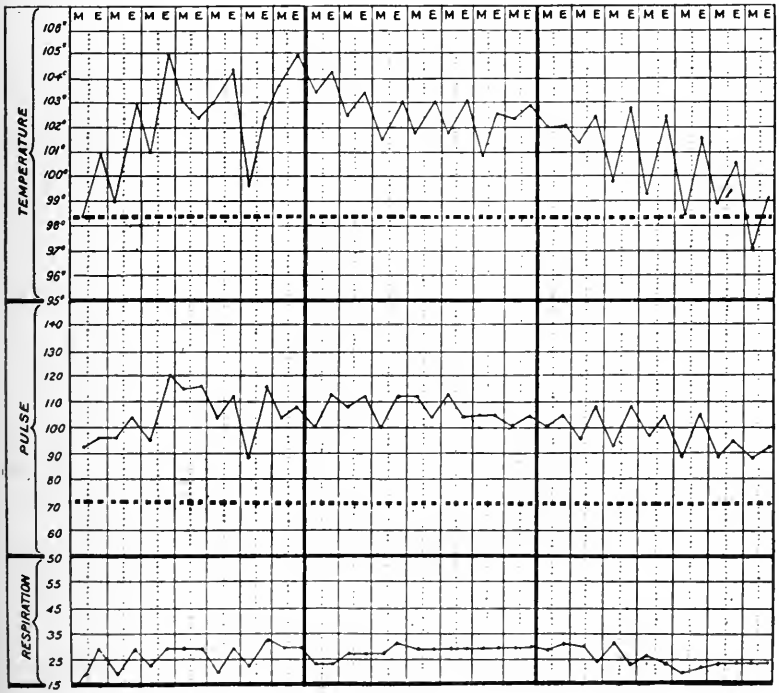


FIG. 6.—Typical Chart of a mild case of Enteric Fever.

four days the patient will feel so ill that he is compelled to go to bed. The temperature rises two degrees each evening, remitting one of these the following morning, until a height of 103° or 104° is reached. The pulse, at first full and bounding, has by now become remarkably soft (even dicrotic), and its rate is about 100 per minute. The tongue at first is moist, with thick white fur on the dorsum and a red tip and sides, but later on it becomes dry and cracked. The abdomen is usually somewhat distended,

and there may be some tenderness in the neighbourhood of the cæcum. At the end of the first week it is often possible to feel the spleen at or below the costal margin. More or less bronchitis is almost invariable.

From the fifth to the seventh day a scarlatiniform erythema may occasionally complicate the diagnosis, but the *characteristic* rash is rarely met with until the seventh day. It consists of small round or oval "lenticular" papules, rose-pink in colour, which appear up to the number of ten or twelve on the back, abdomen, chest and shoulders. In rare cases the spots may be very numerous.

The spots last about five or six days and occur in successive crops ; they fade on pressure throughout their existence except when, as rarely happens, they are surmounted by a minute vesicle.

During the *second week* the symptoms are all intensified ; the patient seems too exhausted to care what becomes of him ; the pulse is more rapid (110—120) and so soft as often to become dicrotic ; the temperature remains about 103°, but shows a distinct morning remission. In many cases diarrhœa is persistent, the motions being very offensive and described as "pea-soup," but in others constipation supervenes. A low muttering delirium is common at night.

About the end of the *third week* the sloughs separate from the ulcerated patches in the intestine and more or less blood may appear in the stools. When separation of the sloughs is complete, the temperature falls by lysis, usually returning to the normal about the end of the fourth week. During the fifth and sixth weeks relapses may occur ; these are due to an infection of hitherto uninfected Peyer's patches, and are characterised by a recurrence of symptoms and signs, which do not, however, last for more than a week or ten days.

Varieties. In addition to the ordinary form described above the following varieties are recognised :—

(i.) *Abortive.* Though starting with what appears to be the ordinary severity, the disease may terminate by crisis during the second week.

(ii.) *Ambulatory.* In this peculiarly grave variety the

patient, either from obstinacy or because he does not feel sufficiently ill, does not take to his bed, and may not infrequently persist in his ordinary vocation until during the third week perforation of an ulcer leads to general peritonitis and death. Such cases are often first diagnosed on the operating table or at the autopsy.

(iii.) *Enteric Febricula.* An extremely mild variety, in which the symptoms are trivial and the morning temperature often normal.

(iv.) *Nephritic.* This rare form starts with an acute nephritis, which is so severe as to mask the underlying enteric fever.

Although the main varieties of enteric fever have been briefly outlined above, it cannot too strongly be represented that clinically the disease rarely conforms strictly to type, thus :—

The course may be prolonged for many weeks.

The temperature may vary within wide margins.

The eruption may not appear at all or it may be very profuse.

Three or more relapses may occur for no very apparent reason.

Profuse perspirations may suggest septicæmia. The disproportionate severity of the concomitant bronchitis may simulate some primary pulmonary affection. An inflamed throat and an erythematous eruption may for the moment resemble scarlet fever.

Complications. (i.) *The Alimentary System.* It is here that the three most important complications are met with :—

(*a*) *Hæmorrhage.* This occurs when the sloughs separate, usually at the end of the third week. It can readily be diagnosed by the symptoms of collapse, namely, an abrupt fall in the temperature, an increased rapidity of the pulse-rate, great restlessness and thirst, combined eventually with the passage of large quantities of blood per rectum.

(*b*) *Perforation.* This occurs at the same period as hæmorrhage, and the signs, at first, are much the same, with the exception that there is no loss of blood. The

diagnosis of perforation may be extremely difficult in the early stages, for the classical signs of acute peritonitis are absent for a considerable time, presumably because the exhausted condition of the patient delays the ordinary reaction of the peritoneum to the irritating intestinal contents. In conjunction with the weak, thready pulse and a sudden drop in temperature, perhaps to 95°, increasing distension of the abdomen and a diminution in the area of liver dulness (due to free gas in the peritoneum), as estimated by percussion in the right mid-axillary line, will justify the diagnosis of perforation, especially if a blood examination shows a commencing leucocytosis.

(c) *Meteorism.* Abnormal abdominal distension, with which may usually be observed the presence of undigested curds in the motions, indicates an unsuitable diet and is of grave import, because by hampering the action of the diaphragm and compressing the thoracic viscera additional strain is thrown upon an already enfeebled heart, while perforation of an ulcer is favoured by the mechanical dilatation of the gut.

(ii.) *Cardiac.* Myocardial degeneration, with its resulting dilatation, is met with to a greater or less extent in every case of enteric fever; endocarditis leading to permanent valvular disorganisation may occur; pericarditis can be diagnosed by the to-and-fro rub heard on auscultation.

(iii.) *Thrombosis.* This is by no means uncommon: the usual site is the left femoral vein, but cases are recorded in which the inferior vena cava has been affected. Thrombosis is most often met with in the third week, but may occur at any time during convalescence.

(iv.) *The Respiratory System.* Pleurisy and pneumonia (lobar and lobular) may complicate enteric fever.

(v.) *The Nervous System.* Neuritis and neuralgia, *e.g.*, the so-called "Typhoid Spine," are common in the later stages, while acute mania may be met with at the height of the disease.

(vi.) *Acute nephritis* and *suppression of urine* sometimes occur during the second or third weeks, but are more often an initial manifestation.

(vii.) *The Bony System.* Cold sub-periostial abscesses may occur (the typhoid node); they are of importance because they may harbour the active bacillus for many years.

Diagnosis. (i.) *General Considerations.* Enteric fever is a disease of children and young adults; it is most frequent in the autumn, and is liable to occur in epidemic form, though it is endemic in all large towns. For a correct diagnosis we are compelled to rely on the grouping together of several individual symptoms or signs, for, with the exception of Widal's reaction, no single one can be regarded as pathognomonic. The most important guides will be :—

(*a*) The gradual onset.

(*b*) The step-ladder type of temperature during the first week and its subsequent remittent character.

(*c*) The combination of a temperature of 102° or 103° with a relatively slow pulse (about 100) (cf. "Tuberculosis," p. 84).

(*d*) The nature of the pulse, which is so soft as often to be dicrotic.

(*e*) The prominent abdomen.

(*f*) The splenic enlargement.

(*g*) The presence or history of diarrhœa.

(*h*) The presence of bronchitis.

(*i*) The characteristic eruption at the end of the first week.

Of these diagnostic features perhaps the most valuable are the history of the mode of onset, the appearance of the characteristic rose spots, and the discovery of an enlarged spleen.

(ii.) *Special Methods of Investigation.* (*a*) *The Blood Count.* There is no leucocytosis in enteric fever—indeed, there is nearly always a definite leucopenia, though the count may show a *relative* lymphocytosis. A leucocytosis is present in every other exanthematous fever except chicken-pox, measles, and German measles.

(*b*) *Ehrlich's Diazo-reaction.*—For this is required first a ·5 per cent. solution of sodium nitrite in distilled water, and secondly a saturated solution of sulphanilic acid in 5 per cent. hydrochloric acid. These solutions must be kept separate, and when the reaction is to be performed the

test solution is made by adding one part of the sodium nitrite solution to one hundred parts of the sulphanilic acid solution. If some of this be mixed with an equal volume of fresh urine and an excess of strong ammonia be allowed to run gently down on to the mixture, a deep red band is formed at the junction of the ammonia and the mixture: if shaken a rose-pink foam is produced, while a green precipitate is formed after several hours. This reaction is practically constant in enteric fever after the fourth day, but has been stated to occur in various other conditions such as pneumonia, malaria, and miliary tuberculosis. Its value is therefore largely negative—that is to say, its persistent absence excludes the diagnosis of typhoid.

(*c*) *Widal's Reaction.* This reaction depends on the presence of agglutinins in the blood serum of persons suffering from enteric fever. The serum of such a person possesses the power of causing the bacilli in an emulsion of typhoid organisms to run together into clumps. The reaction is positive or negative, according to the dilution of the serum which is necessary to cause this clumping and the time it takes in doing so. To perform the test there are required (*a*) 15 or 20 drops of the patient's blood, and (*b*) an emulsion made from an eighteen to twenty-four hours old culture of virulent typhoid bacilli.

The patient's finger is pricked and a small blood capsule (Widal tube) filled with blood.

The emulsion is made by scraping the growth off the surface of the culture medium with a platinum loop and mixing it thoroughly with a few drops of water in a clean watch-glass.

It is necessary to be sure that the emulsion is of a suitable consistence and also that the organisms are active and not already clumped. Therefore a hanging-drop preparation of the emulsion is first examined under the microscope to ensure that each field contains a sufficiency of active unclumped bacilli but is not too thick with them. If it is too thick a little more water must be added. A very little practice will ensure the preparation of a suitable emulsion. The blood tube is now centrifugalised (if the serum has not already separated by standing) and a loopful of the serum placed on

a clean glass slide. Round this drop of serum, but not touching it, are now placed four loopfuls (the same loop must be used) of sterile broth. These are now mixed thoroughly together and the result is a serum dilution of 1 in 5.

A loopful of this mixture is placed on another slide and mixed in the same way with four more loopfuls of broth and a serum dilution of 1 in 25 is produced. In the like manner a loopful of this second mixture is placed on a third slide and a serum dilution of 1 in 50 is effected by the addition of one loopful of broth. To a loopful of each of these dilutions 1 in 5, 1 in 25, and 1 in 50 is added a loopful of the bacillary emulsion, thus producing final serum dilutions of 1 in 10, 1 in 50, and 1 in 100.

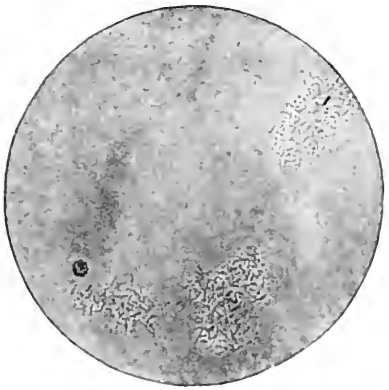


Fig. 7.—Widal Reaction showing Agglutination of Bacillus Typhosus (unstained) by the blood serum of a patient suffering from Enteric Fever. Magnification × 1000.

Hanging-drop preparations of these three ultimate mixtures are at once made and examined microscopically.

A hanging-drop is not essential — ordinary wet preparations on a flat slide with a cover-glass dropped on to the fluid are quite satisfactory; in fact, the bacilli are easier to focus.

If the patient is not suffering from enteric fever, the bacilli will continue to be evenly diffused throughout the film and to be actively motile.

If the patient is suffering from enteric fever, the movements of the bacilli will be seen gradually to cease and they will become aggregated together into little clumps.

The reaction may be considered positive if there is agglutination in thirty minutes with a dilution of 1 in 50.

The reason for advising the use of three dilutions is as follows:—

If agglutination occurs at all quickly with a dilution of 1 in 100, the diagnosis is very positive.

If no agglutination occurs with a dilution of 1 in 10, a negative diagnosis is probable, but since some healthy serums show a certain amount of agglutination of typhoid bacilli in this small dilution, it is not justifiable to give a positive diagnosis on this alone.

It is not essential to use a live culture for the performance of the Widal reaction. Dead cultures are also agglutinated by the serum of enteric fever patients.

Dead cultures, if kept long in emulsion form, tend to become clumped spontaneously, and this may spoil the experiment. At the same time the use of a standard emulsion of dead bacilli is very much more within the scope of the general practitioner, and affords excellent results provided that the uniformity of suspension is tested before each experiment.

The dead bacilli may be used exactly as described above, or, if preferred, the macroscopic method may be employed.

For this purpose a rather wide bore " capillary pipette " about 6 inches long is used. A mark is made on this about $1\frac{1}{4}$ inches from the end. The tube is filled with *diluted* serum to the mark ; a bubble of air is next allowed to enter the pipette, which is then filled to the mark with the bacillary emulsion. The emulsion and serum are well mixed by blowing them out several times into a watch-glass and sucking them back into the pipette. Finally, the bottom of the pipette is sealed off and the whole thing is left for twenty-four hours in an upright position. A positive reaction is shown by the presence of a *flocculent* granular deposit at the bottom of the tube, the supernatant fluid being clear.

A control should always be employed. This shows a uniform even layer of bacilli at the bottom of the tube, an appearance which is, of course, also given in a negative reaction.

The macroscopic reaction can equally well be employed with live bacilli, and the result is even easier to read, since in a negative reaction the whole tube remains turbid, while in a positive reaction the flocculent masses of agglutinated organisms settle to the bottom of the tube just as in the case of the dead bacilli. With the exception of cultivation of the Bacillus typhosus from the patient's blood, a positive Widal reaction is the most certain means at our disposal

for the diagnosis of enteric fever ; but it is most necessary to remember that the reaction is not commonly present till the eighth day of the disease, and also that patients who have once had enteric fever may give the reaction for several years.

(*d*) *The Recovery of the Specific Organisms from the Blood, Urine and Fæces.* It is sometimes of importance, especially in military encampments and in the tropics, to make a positive diagnosis before the eighth day, and it has been found possible to grow the bacillus from the blood of infected patients as early as the third or fourth day. This means of diagnosis is simple and very accurate (*vide* p. 157) ; it should be attempted in every case, *as a positive diagnosis can be made earlier than in any other way.* The bacilli may usually be found in the urine and fæces, though their identification from the latter material is complicated by the multitude of similar organisms which are normally present.

Differential Diagnosis. Unless blood cultivation is performed, it may be extremely difficult to distinguish enteric fever from the following conditions until the eighth day, when Widal's reaction can be employed :—

(i.) *Septicæmia and Ulcerative Endocarditis.* A primary septic focus may be found or obviously active endocarditis may be present ; frequent perspirations and rigors, or the occurrence of infarction as well as a prominent leucocytosis may help in the diagnosis, while the temperature is apt to show greater variations than is the case in enteric fever.

(ii.) *Miliary Tuberculosis.* The patient may present some obvious tuberculous lesion, the temperature may well be intermittent or even of the inverse type, while splenic enlargement is unusual and the pulse-rate is apt to be rather higher than in enteric fever.

(iii.) *A Tuberculous Storm in the Mesenteric Glands.* This condition may so exactly simulate enteric fever that diagnosis is impossible until a negative Widal has been obtained, though a wide variation in the opsonic index to tubercle when the temperature is at its highest and lowest respectively is likely to be found, while if the patient be under six years of age von Pirquet's tuberculo-cutaneous reaction is of value.

(iv.) *Appendicitis.* The sudden onset, the localising symptoms, the possible history of previous attacks, the increasing pulse-rate, the frequency of vomiting, and a leucocytosis will usually lead to a correct diagnosis.

(v.) *Lobar Pneumonia.* The rapidity of the respirations and the high leucocyte count, as well as the aspect of the patient and the possibility of herpes labialis, will usually prevent a mistake being made, but it must be remembered that lobar pneumonia and enteric fever may co-exist and also that in *psittacosis* (a disease conveyed to man by infected parrots) a lobar pneumonia is present at the same time as a chronic enteritis, and further, that in this disease the patient's serum will agglutinate the Bacillus typhosus. The diagnosis here must depend on the proximity of a sick parrot.

(vi.) *Malaria* does not give a true Diazo-reaction and the plasmodium can be found in blood-films from the patient, otherwise some remittent forms of malaria may closely resemble enteric fever.

(vii.) *Para-typhoid Infection.* The condition produced by this bacillus may be absolutely indistinguishable from enteric fever until the agglutination test shows that the patient's serum will clump one of the group of para-typhoid bacilli and not the Bacillus typhosus.

(viii.) *Influenza.* The abdominal type of influenza may easily be mistaken for enteric fever, but as a rule the sudden onset, the rapid rise of temperature, the absence of splenic enlargement, as well as the more pronounced character of the abdominal symptoms, should prevent such a mistake.

From the foregoing considerations the importance of Widal's reaction will be apparent; if properly performed the margin of error is probably less than three per cent.

VII. ERYSIPELAS.

Definition. A specific infective disease characterised by a spreading dermatitis and caused by the Streptococcus pyogenes.

Incubation Period. From two to seven days.

Course. The onset is abrupt, with rigors, headaches, and vomiting, while the temperature rises to 103° or 104°.

The inflammation, except in traumatic cases, usually starts at a muco-cutaneous junction, and spreads along the skin rather than the mucous membrane, though both may be involved. The affected skin is red, swollen and painful, while bullæ frequently appear on its surface. The inflamed area is surrounded by a sharply-defined, bright-red raised margin, which tends to spread by the lymphatics, either rapidly or slowly, so as to include an increasing area of hitherto uninfected skin. While the infection is extending peripherally the parts first involved may be recovering. Delirium is frequent, while in elderly or debilitated persons a condition of profound toxæmic exhaustion may rapidly supervene. The termination is usually by crisis on the seventh to tenth day.

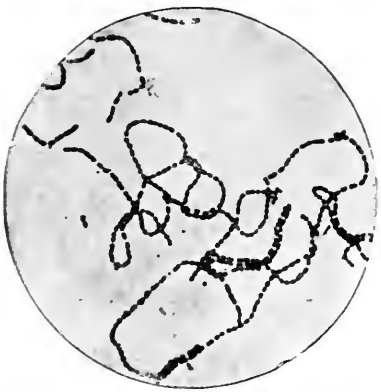


Fig. 8.—Film preparation of Streptococcus Pyogenes. Magnification × 1000.

Varieties. (*a*) An *acute form*, as described above.

(*b*) A *chronic form*, or *erysipelas migrans*, in which the constitutional disturbance is slight and the inflammation slowly wanders from one part of the body to another.

Complications. Complications are most likely to be seen when the infection attacks the mucous membrane of the mouth or fauces.

Œdema of the glottis may then occur, producing respiratory obstruction, as shown by laryngeal stridor and even asphyxiation.

Broncho-pneumonia may develop from the inhalation of septic particles.

Cellulitis, with pus formation, is yet another complication, and is of particular gravity should it affect the fauces, pharynx or orbit, in which latter case a spread of infection to the cerebral meninges has been recorded.

Erysipelas of the scalp is commonly followed by baldness.

Diagnosis. There is no disease except erysipelas which presents the above-mentioned features. Syphilitics and women during the puerperium seem peculiarly liable to this infection, while certain other individuals may suffer repeated attacks for no apparent reason.

The complication of pus formation can be diagnosed

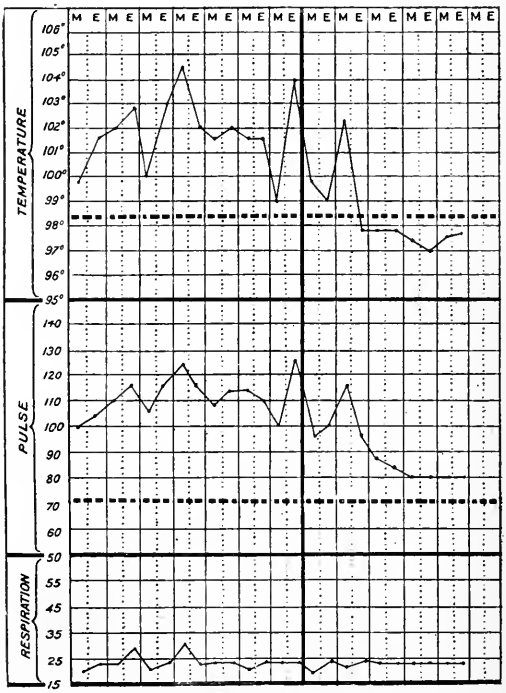


FIG. 9.—Chart from a case of Erysipelas.

by the brawny, boggy, or œdematous appearance of the affected parts. Occasionally the inflammation round a *vaccination pustule* may at first suggest erysipelas; the characteristic edge is, however, absent, and the fact that vaccination had been performed would suggest that true erysipelas was not present. Of course, erysipelas may follow vaccination, so a guarded prognosis is necessary for a few days. The early stages of *malignant pustule*

may simulate erysipelas, but the central black scar with its ring of secondary vesicles would soon settle the diagnosis.

VIII. GERMAN MEASLES.

Definition. A specific infective disease of unknown causation, characterised by a red rash and glandular enlargement, most commonly met with in children.

Incubation Period. Twelve to twenty-one days, usually eighteen.

Course. Constitutional disturbance is slight or absent. There may be trifling malaise and headache or mild catarrhal symptoms and sore throat. The temperature, though often normal, will sometimes rise to 100° for forty-eight hours.

In twenty-four hours the rash appears, and this is often the first sign of the infection. The rash is composed of rather bright-red papules, which usually appear first on the face, and particularly about the mouth and nose. Within a very few hours the trunk and arms are covered; the legs are usually involved after a short interval. The papules tend to run together, but do not commonly cause such a blotchy appearance as in true measles.

Within twelve hours from its appearance the rash begins to fade and is completely gone in three days; a fine branny desquamation usually follows. Itching is absent or slight.

At the same time as the appearance of the rash the fauces will be found to be infected, and not infrequently macular spots may be found in the throat. There is often a slight generalised glandular enlargement. This is most apparent in the posterior triangles of the neck.

Complications are practically unknown.

Differential Diagnosis. (i.) *From Measles* (*vide* p. 41).

(ii.) *From Scarlet Fever* (*vide* p. 64).

(iii.) *From Food Poisoning.* Shell-fish, strawberries, etc., may in susceptible people produce a rash which resembles that of German measles. Such rashes usually cause considerable irritation. The diagnosis must depend on the history of similar previous attacks, on the individual's

known liability to such occurrences, and on the absence of glandular enlargements.

True ptomaine poisoning is accompanied by such severe constitutional disturbance, *e.g.*, diarrhœa and vomiting, that mistake is unlikely.

(iv.) *From Drug Rashes.* Copaiba, bromide, iodides, belladonna, salicylates, etc., may sometimes produce a rash like that of German measles. They are, however, usually more or less localised in their distribution, and inquiry will reveal the fact that some such drug has recently been taken, while in the case of belladonna, iodides, or salicylic acid the dilated pupils, profuse coryza, and buzzing in the ears respectively will help to clinch the diagnosis.

After administration of an enema a measly or scarlatiniform rash will sometimes occur ; it appears first on the thighs and abdomen, and is often localised to these parts.

IX. HYDROPHOBIA.

Definition. A specific infective disease conveyed to man by the bites of rabid wolves, cats, and dogs. The causative organism has not yet been demonstrated.

Incubation Period. From three weeks to two years. Usually from six to eight weeks.

Course. The original bite is usually completely healed. The patient becomes restless, depressed, irritable, and has a well-marked sense of impending disaster ; at the same time there is generally a sense of discomfort, tingling, and even pain in the scar of the bite. In a few days there is a choking feeling in the throat, a huskiness in the voice, and a difficulty in swallowing. The next manifestations of the disease are spasms of the muscles of deglutition and respiration, which are excited by the attempt to drink or swallow, and later by the most trivial causes, such as a breath of air or a slight noise.

The spasmodic convulsions soon spread to other parts of the body, and at the same time there is intense cutaneous hyperæsthesia. The expression becomes one of acute anxiety and terror, the temperature is raised, delirium and

mania ensue, and the patient dies in from two to three days from syncope or asphyxia.

Diagnosis. When the characteristic symptoms of the disease have appeared the diagnosis is simple ; the important point is to determine whether or no an animal that has bitten people is rabid. The suspected animal should be isolated for observation, when, if rabid, it will die in five days.

If through over-zeal the animal has been destroyed, a portion of its medulla oblongata should be emulsified and injected into the dura mater of a rabbit. The rabbit will die of rabies in from fifteen to twenty-five days.

It has been stated by Negri that small irregular-shaped protozoa can be demonstrated in the brain, pons, spinal cord, and cornu ammonis of rabid animals, and that by this means a reliable diagnosis can be made within twenty-four hours.

Hydrophobia must not be mistaken for :—

(i). *Lyssophobia*. This is a hysterical manifestation occurring in nervous persons who have been bitten by a dog.

The premonitory symptoms of hydrophobia are accurately simulated, but there is no pyrexia, and the symptoms never go further than a professed inability to swallow.

This condition may persist much longer than would true rabies, and is amenable to treatment in the nature of electricity and moral persuasion.

(ii.) *Tetanus*. In hydrophobia there is no tonic rigidity, opisthotonos, or trismus. In tetanus there is no mental disorder, though should tetanus follow a wound on the head there will be spasms of the pharynx ; there will, however, also be facial paralysis, which does not occur in hydrophobia. Lastly, it is very rare for tetanus to follow the bite of a dog.

X. GLANDERS.

Definition. A specific infective disease of horses, conveyed by them to man, due to the Bacillus mallei, and characterised by the formation of multiple granulomatous

abscesses in the nose or along the course of the cutaneous lymphatics.

Varieties. (i.) *Glanders*, which is primarily an infection of the nasal mucosa.

(ii.) *Farcy*, which is primarily a cutaneous or lymphatic infection.

Either variety in the acute form tends to become complicated by the other, whilst chronic forms of both glanders and farcy are described.

Incubation Period. Up to four days. In rare cases it may be much longer.

Course. (i.) *Acute Glanders.* The invasion is sudden, with headache, rigors, and prostration ; joint and muscle pains are common.

About the second day the nose becomes swollen and painful, and shortly afterwards there is a profuse blood-stained nasal discharge ; at the same time the cervical lymph glands are much enlarged. Towards the end of the week an eruption appears about the face, trunk and joints ; this, at first papular, rapidly pustulates, and may closely resemble small-pox. Death ensues during the second week, and is often preceded by a sub-acute pneumonia.

Chronic Glanders. The constitutional disturbance is much less acute ; there is a chronic nasal discharge and ulceration of the nose, but a skin rash is uncommon.

The duration may be several months and recovery may take place.

(ii.) *Acute Farcy.* The nose is not affected and the skin eruption is absent. Instead there is an intense lymphangitis, with nodular masses of granulation tissue distributed along the course of the lymphatics. These nodules eventually suppurate. Joint pains are common, and abscesses may form in the muscles.

It will be seen that the disease bears a striking resemblance to an acute pyæmia.

Death usually occurs at the end of the second week.

Chronic Farcy. In this variety there are localised abscesses which are situated usually in the extremities and bear no definite relation to the lymphatics.

True pyæmia, or even acute glanders, may supervene ;

otherwise the disease drags out a protracted course for months or years and may ultimately end in recovery.

Diagnosis. In view of the fact that primarily the disease is one of horses, the history of employment and exposure to infection is of paramount importance.

The absolute diagnosis depends on the recovery of the specific bacillus from the nasal discharge or from one of the lesions.

The glanders bacillus is a small, straight or slightly-curved rod with rounded ends. It is rather thicker than a tubercle bacillus, but about the same length. The protoplasm is often very granular. The shape and size of the organisms are not constant. The bacillus is non-motile, and does not form spores. It stains readily with ordinary dilute basic stains, and is decolourised by the method of Gram.

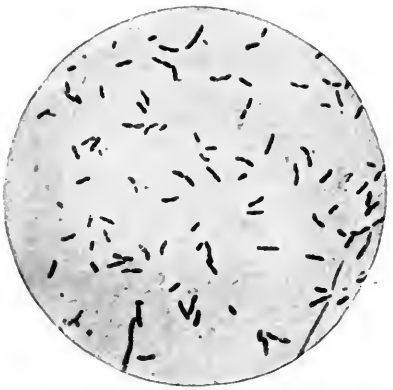


FIG. 10.—Bacillus Mallei from 48 hours culture on potato. Magnification × 1000.

In films of pus from a patient suffering from glanders the organisms are nearly all extra-cellular.

The best culture medium for the glanders bacillus is blood serum on which medium round, rather transparent, drop-like colonies become visible in twenty-four hours.

It is in *chronic* cases that the greatest diagnostic difficulty will be experienced. Even though the bacillus itself may not be visible either on cultivation or in film preparations, nevertheless films made from the pus of a glandered subject will often show a peculiar granular appearance, due to disintegration of nuclei, such as is rarely met with in other conditions.

To establish the diagnosis, however, it is best to inject a small quantity of the suspected material into the peritoneum of a male guinea-pig. At the end of two days, if the case be one of glanders, the testicles of the guinea-pig will be

distinctly swollen. This swelling increases, suppuration ensues, and in three weeks the animal dies with generalised glanderous nodules in the viscera.

The following diseases may be confounded with glanders before the inoculation of the guinea-pig has verified the diagnosis :—

(i.) *Small-pox.* The swelling of the nose and lymph glands and the nasal discharge in glanders, as well as the greater rapidity of pustulation, should prevent this mistake.

(ii.) *Sporo-trichosis* may resemble farcy, but the extreme chronicity of this disease, with little or no impairment of general health and the tendency of the nodules to remain unbroken for a long period, as well as the presence of a streptothrix organism, will assist in the diagnosis.

(iii.) *Chronic Coryza.* This may exactly simulate a mild case of chronic glanders. Examination of the nose will usually reveal multiple small ulcers, and in any doubtful case injection of a guinea-pig must at once be undertaken.

To diagnose glanders in horses mallein, prepared and used in the same way as Koch's old tuberculin (*q.v.*), is highly recommended.

XI. GONORRHŒA.

Definition. A specific infective disease caused by the gonococcus and characterised by local inflammation at the site of infection and sometimes accompanied by various metastatic inflammatory phenomena.

Incubation Period. Two to nine days. The incubation period is usually shorter in a second or third attack, but with the first infection symptoms usually become manifest on the fourth or fifth day.

Manifestations and **Diagnosis.** The purulent urethritis, with the more usual complications of epidydimitis, orchitis, prostatic abscess, cystitis, and inguinal bubo, are appropriately described in text-books on surgery. Similarly, an account of the pelvic and peritoneal complications of gonorrhœa in the female will be found in manuals on Diseases of Women.

We are here concerned only with those manifestations of

gonorrhœa which indicate that the organisms have entered the blood-stream and produced a condition of septicæmia.

(i.) *Arthritis.* This usually occurs during an acute attack, though it is more likely to happen with a subsequent attack than at the first infection. Nevertheless, it may complicate ophthalmia neonatorum, or it may only become evident when the urethritis has quieted down into a chronic gleet. Any one of the joints in the body may be affected, but it is noteworthy that the temporo-mandibular, sterno-clavicular, and spinal articulations are frequently involved. It is extremely rare for these joints to be affected by rheumatism. The inflammation may be mostly peri-articular, or it may be a practically painless hydrarthrosis. Suppuration may occur, but is uncommon. Whatever the manifestation of the disease, its extreme chronicity and resistance to treatment are important diagnostic points.

Any inflammation attacking tendons and tendon sheaths and bursæ may well be gonorrhœal; flat-foot is common, and a local periosteal thickening with exostosis on the under surface of the os calcis may give rise to great pain and lameness.

The great difficulty in diagnosis is to distinguish gonorrhœal arthritis from acute or sub-acute rheumatism. The diagnosis will rest on the following points:—

(*a*) The history of an active urethritis, or even of a chronic gleet (in the case of women the vaginal discharge should be examined for gonococci).

(*b*) The sudden appearance of pain and swelling in several joints, with the absence of that flitting from joint to joint which is so characteristic of acute rheumatism.

(*c*) A constitutional disturbance more slight and a less high temperature than the severity of the joint pains might appear to warrant.

(*d*) The absence of profuse perspirations, cardiac lesions, and rheumatic nodules.

(*e*) And, possibly most important of all, the fact that the symptoms are not ameliorated by the exhibition of salicylates.

(ii.) *Conjunctivitis and Iritis.* A purulent inflammation of the conjunctiva is commonly conveyed by direct infection

of gonorrhœal pus on fingers, towels, etc., but it may also be a metastatic phenomenon in the course of a gonococcal septicæmia.

(iii.) *Endocarditis and Pericarditis* will give the usual signs of such conditions.

(iv.) *Neuritis.* A symmetrical polyneuritis may sometimes occur with the symptoms of pain, numbness, and tingling followed by paralysis. Though the neuritis is often localised, it may give rise to a rapidly ascending paralysis, until death ensues, with the involvement of the phrenic nerves, in a few days from the onset.

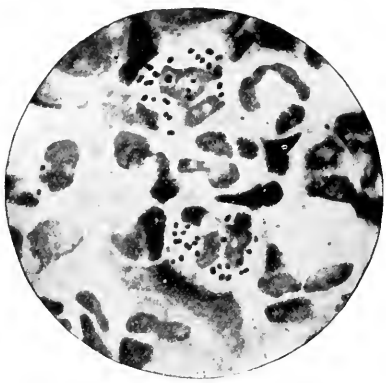


Fig. 11.—Gonococcus in Urethral pus. Magnification 1000.

Bacteriology. The gonococcus is a small coccus usually found in pairs (diplococcus). The adjacent surfaces of the cocci are often indented, thus giving rise to an appearance as of two beans placed side by side. It stains well with all basic aniline dyes, but is decolourised by Gram's method. It is abundant in the urethral discharge, the organisms being seen for the most part within the leucocytes. It can often be demonstrated in the fluid of a gonorrhœal joint, and in severe cases of gonorrhœal septicæmia may be grown from the blood of the patient.

The organism is best cultivated on a medium of inspissated human or rabbit's blood serum, though an ordinary agar tube freshly smeared with human blood will answer the purpose. The culture medium should be lightly rubbed with a platinum loop containing the suspected pus, or in the case of effusion from a joint with a portion of the centrifugalised deposit. The tube should then be incubated at the body temperature, when in from twenty-four to forty-eight hours small, roughly circular, opaque white colonies of gonococci will be apparent.

It is a noteworthy point that in the adult female the gonococcus does not ordinarily affect the vaginal mucous membrane; it chooses rather the urethra or the cervix uteri. This is not so in the case of children, and a large proportion of the cases of vulvo-vaginitis in young girls is due to the gonococcus.

XII. INFLUENZA.

Definition. A specific infective disease of an extremely infectious nature occurring at irregular intervals in widespread epidemics, and in the meanwhile apparently remaining endemic throughout the civilised world.

Bacteriology. Influenza is caused by a specific microorganism, the Bacillus influenzæ of Pfeiffer. This is a minute rod-shaped bacillus, which stains feebly with the basic aniline dyes and is best demonstrated by staining with 10 per cent. carbol-fuchsin. It is decolourised by Grám's method. It is somewhat difficult to cultivate except in the presence of hæmoglobin. Fresh blood agar is therefore the best culture medium.

Clinically indistinguishable conditions may be caused by the Micrococcus catarrhalis, the Pneumococcus, etc., or a combination of such organisms.

Incubation Period. Usually three or four days.

Course and Varieties. The manifestations of influenza are so varied that it is convenient clinically to describe the disease as occurring in the following five varieties. It has been noticed that one or other form is likely to predominate in any given epidemic :—

(i.) *Respiratory.* This is certainly the most common form of influenza. The onset is sudden, with severe pain in the head, particularly behind the eyes, in the back and in the limbs. Vomiting may occur and the temperature rises to 102° or 103°. The pulse is full and bounding but not very rapid, usually from 90 to 100 per minute. There is coryza, with catarrh of the respiratory tract, bronchitis, cough, and not infrequently a green nummular expectoration. A sore throat with injection and inflammation of the faucial pillars is often found.

There is a tendency for the symptoms to abate in the morning and to recur each evening for several days.

The prostration of the patient is extreme.

(ii.) *Gastro-intestinal.* Severe abdominal pain with vomiting and diarrhœa may take the place of the respiratory disturbances as an accompaniment to the general constitutional symptoms.

(iii.) *The Cerebral form.* In this variety catarrhal symptoms may be entirely absent. The headache is extreme, delirium is usual, and the occurrence of convulsions combined with obstinate vomiting may closely simulate meningitis.

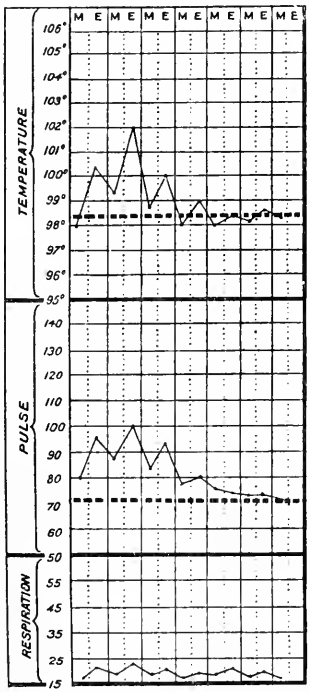


Fig. 12.—Chart from a typical case of uncomplicated Influenza.

(iv.) *The Febrile form.* In rare cases fever, either remittent or continued, even for several weeks, may be the only manifestation of influenza.

(v.) *Chronic Influenza.* This variety is often the result of a series of acute attacks occurring within a few weeks or months. It is frequently afebrile and accompanied by a slow pulse.

Though chronic bronchial catarrh is a usual accompaniment of chronic influenza, it is nevertheless by no means uncommon for severe prostration, mental and physical, to be the only sign.

Complications. It is by reason of its complications, as well as by its predilection for elderly and debilitated subjects, that influenza is so dangerous.

The common complications are :—

(i.) *Pulmonary.* Capillary bronchitis and broncho-pneumonia are often seen in the respiratory form of influenza. Lobar pneumonia is extremely rare, though the tendency for the patches of lobular inflammation to fuse into large

solid areas may simulate a lobar distribution. Should the inflamed area reach the surface, a pleurisy will follow, but primary pleurisy is exceptional. Empyema may occur in the same manner as pleurisy.

(ii.) *Cardiac.* Endocarditis and pericarditis are occasionally found. Disordered action of the heart from toxic myocardial degeneration is frequent. This usually takes the form of an obstinate bradycardia, though tachycardia, especially an undue "exercise acceleration," may persist for weeks.

(iii.) *Nervous System.* Meningitis is rare. Neuritis is common, both in the isolated or interstitial form and also in the parenchymatous variety. Obstinate depression, or even melancholia, is a frequent sequel.

(iv.) *Osteo-arthritic System.* Arthritis with fluid effusions, rarely becoming purulent, may occur. Otorrhœa is very common, and occasionally the mastoid cells may be involved.

Diagnosis. The only certain method of diagnosing influenza is to recover the bacillus from the sputum or nasal secretion. When bronchitis is present the sputum frequently contains small green purulent masses; one of these should be spread on to films and stained with carbol-fuchsin as described above, and also by the method of Gram. Culture tubes of blood agar should be inoculated, and if necessary sub-cultures made in forty-eight hours. When respiratory catarrh is absent it will be impossible to demonstrate the specific organism except in the graver septicæmic forms in which endocarditis and arthritis are present. In such cases the bacillus may sometimes be recovered from the blood. Apart from bacteriological investigations, the diagnosis will depend on the presence of an epidemic, the possible history of exposure to infection, the sudden onset with *headache and severe pains in the back and limbs*, high temperature combined with a fairly slow pulse, and an appearance of prostration out of all proportion to the signs and symptoms present. There is no leucocytosis in influenza.

Combined with these general features there will be symptoms pointing to the involvement of either the respiratory, gastro-intestinal, or cerebral systems.

The complications of influenza by themselves present no special features by which their causative organism can be recognised, and the history of the illness is all-important. At the same time a persistent fine-tube bronchitis, the physical signs of which are often strictly localised, is suggestive. In such cases every care must be taken to exclude tuberculosis.

Differential diagnosis. (i.) A thorough microscopical examination of the sputum will settle whether pulmonary or bronchial lesions are caused by influenza, tubercle, or other bacilli.

(ii.) Lumbar puncture, with cytological and bacteriological examination of the cerebro-spinal fluid, will settle whether meningitis is present or not.

(iii.) Careful examination of the abdomen, showing the absence of rigidity, local tenderness, and fixation should exclude such acute abdominal conditions as appendicitis or perforated gastric ulcer. The gradual onset, abdominal distension, enlarging spleen, and, at the end of the week, the rose spots and positive Widal reaction, will settle the question in favour of enteric fever as against the abdominal types of influenza.

XIII. MEASLES.

Definition. An acute infective disease characterised by catarrh of the respiratory tract, pyrexia, and a skin eruption.

Bacteriology. The specific organism has not yet been discovered. The contagion is present in the blood, in the skin, and more especially in the nasal and buccal secretions. The poison is probably conveyed in dust from dried particles of sputum, hence infection by fomites is common.

Incubation Period. Usually fourteen days (seven to eighteen).

Course. The onset, though occasionally insidious, is more often abrupt, with headache, malaise, running at the eyes and nose, and bronchitis. The temperature rises during the first twenty-four hours to a height of 103° to 104°; the pulse becomes rapid and full and the skin hot and dry.

The constitutional symptoms increase in severity till the rash is *fully* developed at the end of the fifth day, when the temperature falls rapidly, almost by crisis, and a speedy convalescence takes place. The rash of measles occurs on the fourth day, though in a few cases it is preceded by a prodromal eruption, usually in the nature of a scattered blotchy erythema.

The true rash starts as minute red papules in the roots of the hair along the forehead and behind the ears; these spread rapidly over the face, down the trunk and arms, and later along the thighs and legs. The papules increase in size and run together, forming irregularly crescentic blotches separated from each other by areas of healthy skin.

Sometimes the papules are surmounted by minute miliary vesicles.

The rash is not fully developed for twenty-four hours after its first manifestation; it has a shotty feel to the examining finger and fades on pressure, except when, as rarely happens, it is the site of small petechial hæmorrhages.

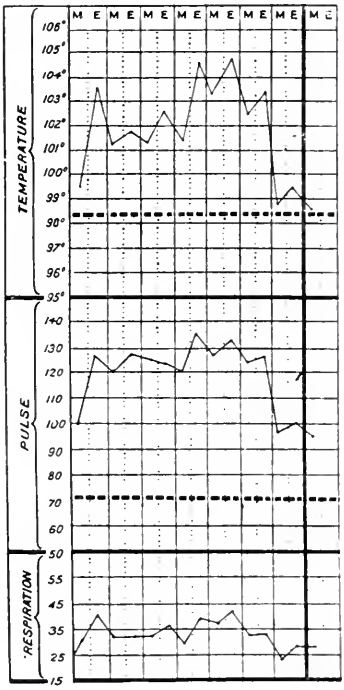


FIG. 13.—Chart from a case of Measles showing termination by crisis at the end of the sixth day and also increased fever after the appearance of the eruption on the fourth and fifth days.

The mucous membrane of the mouth is often affected by the eruption. The fauces are always injected. The rash fades gradually from the seventh or eighth day, and is followed by a fine branny desquamation lasting from one to two weeks.

Before the appearance of the rash—that is to say, as early as the second or third day—*Koplik's spots* may be recognised in 90 per cent. of all cases. The spots consist of red areolæ

with a bluish-white centre situated on the mucous membrane of the cheek opposite the level of the mandibular premolars.

There is no leucocytosis in measles, but myelocytes are often present in small numbers.

Varieties. The ordinary form of measles may be mild or severe, but in addition a malignant or black form is described in which hæmorrhages take place into the skin and from the mucous membranes, and death ensues before the end of the week with every sign of profound cardiac toxæmia.

Complications. The most serious complications are those of the respiratory tract. Laryngitis is not infrequent; it is most common during the second or third weeks, giving rise to a husky voice, croupy cough, and even to stridor, intercostal recession, cyanosis, and the other signs of severe laryngeal obstruction. Tracheotomy may be necessary, but as the acute signs do not often persist for more than thirty-six hours, palliative measures, or possibly intubation, will usually suffice.

Bronchitis, which is an ordinary manifestation of the disease, may affect the capillary bronchioles and produce a suffocative catarrh, while true broncho-pneumonia, both in the acute stage and during convalescence, is a frequent complication.

Owing to the lowered resistance of the tissues as the result of these respiratory complications, pulmonary tuberculosis, in the form of a tuberculous broncho-pneumonia, is a common sequel.

Otitis media, with perforation of the tympanic membranes and intractable otorrhœa, is very common.

Stomatitis, and even cancrum oris (massive necrosis and gangrene of the cheek and gums), may occur, and is most likely to be met with during the third week.

Acute nephritis is more common than has been stated.

Endocarditis, arthritis, hemiplegia, paraplegia, and polyneuritis, though rare, are sometimes met with during the height of the attack.

Diagnosis. Measles is a disease to which all ages are liable, but it is rare for babies under three months to be affected. By far the greatest number of cases are seen in children under twelve years of age. This is largely due to

the fact that the widespread incidence of the disease causes the majority of persons to be infected in childhood and so to acquire the relative immunity conferred by one attack.

The history of exposure to infection is important ; for the rest the diagnosis will depend on :—

(i.) A three days' coryza and bronchial catarrh of sudden onset with high temperature.

(ii.) Koplik's spots on the second or third day.

(iii.) The characteristic rash on the fourth day.

(iv.) The absence of any leucocytosis and the presence of myelocytes in the blood.

Differential Diagnosis. (i.) *Small-pox.* The nodular character of the rash and its early appearance on the face, especially in adults, may lead to this mistake, but in small-pox oculo-nasal catarrh and Koplik's spots are absent, and the general condition of the patient, as well as the onset with rigor, vomiting, and backache, is usually distinctive. Furthermore, in small-pox there is always a well-markéd leucocytosis.

(ii.) *Scarlet Fever.* In scarlet fever an onset with rigor is almost invariable. The pulse-rate is more rapid than that of measles (160 per minute) ; the throat is affected rather than the mouth, as in measles. The rash appears on the second day, starts at the root of the neck, and consists of a diffuse uniform erythema with no areas of healthy skin, such as are seen in measles. Again, coryza is absent, and there is a leucocytosis.

(iii.) *German Measles.* Here the onset is less severe ; the temperature as a rule is not above 100°. The rash appears on the first day ; the papules are more discrete, there are enlarged glands in the posterior triangles of the neck, and a red macular eruption in the *fauces* is a constant symptom.

(iv.) *Erythema Multiforme.* In appearance this may closely resemble the rash of measles, but coryza and constitutional symptoms will be wanting, or possibly definite signs of rheumatism may be present.

(v.) *Drug Rashes* (*vide* also p. 612). Copaiba and the injection of anti-toxic sera may produce rashes which are indistinguishable from measles, but in the former cases there will be no constitutional disturbance, and in the latter the

presence of profuse perspirations and joint pains will usually be noticed. In any case the knowledge that copaiba or serum has been used will usually prevent any error.

The erythematous eruptions of septicæmia may simulate measles, but are more evanescent and do *not* appear first at the root of the hair. Enema rashes are usually limited to the trunk and are more often scarlatiniform than morbilliform.

XIV. MUMPS.

Definition. A specific infective disease characterised by painful swelling of the salivary glands and mild pyrexia.

Bacteriology. The specific organism has not yet been discovered. The disease is highly infectious and confers immunity.

Incubation Period. Forty-eight hours to three weeks.

Course. After a few hours of slight fever and malaise there is pain and swelling in the region of one parotid gland; the swelling rapidly increases, until in twenty-four hours the mouth can only be opened with difficulty.

In the majority of cases the opposite gland will become similarly affected within two or three days. After a week or ten days the swelling gradually subsides. The temperature remains about 100° or 101° throughout the course of the disease. Slight deafness, earache, or even otitis media, may accompany the attack. Suppuration is extremely rare.

Although mumps nearly always selects the parotid gland, it may occasionally be found in the submaxillary or sublingual glands, while the testicles in men and the ovaries in women or the breasts are sometimes the site of metastatic mumps.

These extra-salivary affections are extremely rare before puberty.

The metastatic swellings are most likely to occur about the eighth day; sometimes the orchitis precedes the parotiditis, and in rare cases it may be the sole manifestation of the disease.

Diagnosis. Mumps is a disease of childhood and adoles-

cence; infants and adults are rarely affected. It is most frequent in autumn and spring.

A non-suppurative swelling of the parotid gland, passing forward in front of the ear and backwards beneath the sterno-mastoid with elevation of the lobe of the ear, starting on one side, soon becoming bilateral, accompanied by mild fever and occurring in a child, will usually permit of no other diagnosis.

It must be remembered that in rare cases, and more especially in older people, mumps may be peculiarly severe. High fever, delirium, and extreme prostration may then be met with.

Differential Diagnosis. Any other parotid tumour is rare in childhood.

Parotid endothelioma is unilateral, of slow growth, much harder than mumps, and, at first, unattended by any constitutional disturbance.

Infective parotiditis occurs during the course of an acute fever, and also as a complication of any abdominal condition, such as gastric ulcer, where mouth feeding may be suspended. It is caused primarily by the unusually dry condition of the mouth. Such swellings often suppurate. This fact in conjunction with the presence of some other definite pathological condition will suffice to prevent the diagnosis of mumps.

XV. LOBAR PNEUMONIA.

Definition. A specific infective disease characterised by inflammation of one or more lobes of the lungs and associated with profound toxæmia.

Bacteriology. Acute inflammation of a lung with a lobar distribution is not always due to the pneumococcus, but the predominance of this organism as the causative factor is so great that the term "lobar pneumonia" is generally understood to comprise this variety only.

The pneumococcus of Fraenkel is a micrococcus the shape of which is that of a flattened oval. It is usually arranged in pairs, the two flattened surfaces being adjacent. There is a definite capsule. It stains with basic aniline dyes and is not decolourised by the method of Gram.

In common with most other so-called diplococci, it is best cultivated at the body temperature on the medium of fresh blood agar, when in thirty-six hours characteristic small, almost transparent, circular colonies make their appearance. These run together into a filmy pellicle with isolated colonies at its margin. It is difficult, and sometimes impossible, to get a pure culture of a pneumococcus direct from the sputum of a patient, hence it is sometimes necessary to inoculate a mouse subcutaneously with a small

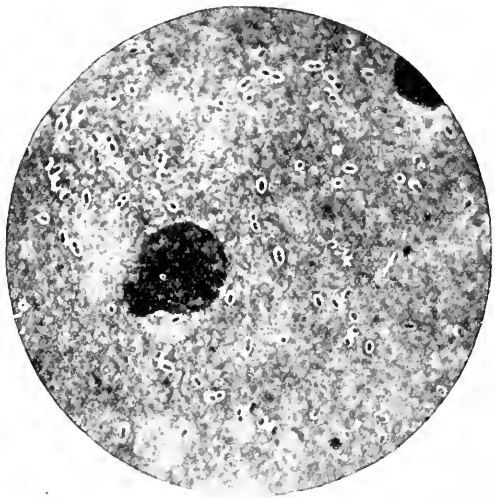


FIG. 14.—Diplococcus Pneumoniæ from the peritoneal fluid of an inoculated animal. Magnification × 1000.

portion of the suspected material. In forty-eight hours the animal will die of a virulent septicæmia and the capsulated diplococcus can readily be recovered from its blood.

Friedländer's pneumo-bacillus, a capsulated Gram-negative bacillus, is sometimes found in the sputum of pneumonic patients ; it is very rare for it to occur alone ; more often it is in association with the pneumococcus, for which, however, it should not be mistaken.

Incubation Period. From a few hours to a few days, but, since case to case infection is unusual, the incubation period cannot readily be determined.

Course. The invasion is abrupt, usually with vomiting and a rigor, or, in the case of young children, convulsions. In a few hours there is a sharp stabbing pain in the chest, worse on inspiration, increased rapidity of respiration, and a rise of temperature to from 102° to 104°. A short dry cough, at first unproductive, will soon be apparent. By the second or third day the clinical picture is usually characteristic. The patient lies flat on his back, or possibly on the affected side, the face is flushed except for a circum-oral pallor; the eyes are bright, the expression anxious; the alæ nasi dilate with each inspiration; a labial herpes is very common; there is a short, distressing cough which produces a scanty and very tenacious sputum, at first tinged with bright blood and later rusty in colour. Percussion usually reveals a slightly enlarged spleen. The temperature is steady, with a slight daily remission, at or about 104°. The pulse is relatively slow (105—115), while the respirations are extremely rapid, usually about 40 per minute for an adult, 60 to 80 for a child. The pleuritic pain often persists till the third or fourth day. The severity of the symptoms and the prostration of the patient increase till somewhere between the seventh and tenth days, when the temperature returns to normal by crisis, the respiration rate falls, the patient breaks out into a brisk perspiration, and usually drops into a healthy sleep, from which he wakes some ten or twelve

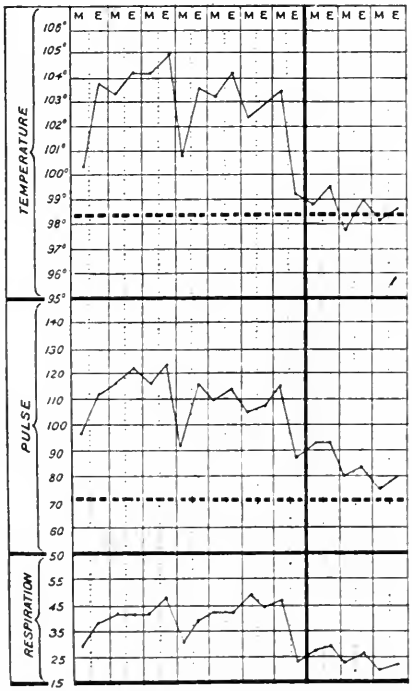


FIG. 15.—Chart from a typical case of Lobar Pneumonia.

hours later, weak, but free from all distressing symptoms. In a certain proportion of cases the crisis may occur at an earlier date, more especially in the case of children.

Physical Signs in the Chest. The earliest physical sign may be restricted movement of one side of the chest. When the pleuritic pain has appeared a friction rub can often be heard. The percussion note at first is usually "boxy" or hyper-resonant; this is due to the fact that the consolidation is apt to start in the centre of the lung, thus leaving a thin layer of healthy lung between the affected portion and the pleura. As the consolidation reaches the surface so will the percussion note become dull and woodeny. The area of dulness, if accurately delimited, may be found to correspond more or less closely with the anatomical boundaries of one or more lobes of the lung. On auscultation, in addition to the pleuritic sounds already mentioned, there will be localised râles of fine and medium size. The air entry will progressively diminish, and, as consolidation advances, the breath sounds will acquire a tubular character, until at the height of the disease there will be no sound audible over the affected area, except the tubular breath sounds of the larynx and trachea, which are admirably conducted to the listening ear by the consolidated lung. It must be remembered, however, that tubular breathing is often not detected during the early days of the attack. Medium and coarse râles, indicating bronchitis, may generally be heard over those areas of lung which are adjacent to the pneumonic patch, and often in the other lung as well.

In some cases the bronchus leading to the consolidated patch may be temporarily plugged with mucous; under these circumstances tubular breathing is absent.

Resolution is often delayed for several days after the crisis; it is effected by the liberation of autolytic enzymes from the disintegrating leucocytes. The inflammatory mass is thus softened by ferment action and is for the most part absorbed by the lymphatics, though a certain proportion is coughed up, as is evidenced by the profuse rusty-coloured sputum which is expectorated during this stage of the disease. The process of resolution is revealed to auscultation by the reappearance over the affected area of fine sticky

crepitations, which gradually increase in size and moistness. These so-called redux crepitations are due to the separation of the alveolar walls and the opening up of the terminal bronchioles by the re-entering stream of inspired air.

Varieties. The following varieties of pneumonia are recognised clinically :—

(i.) *Basal Pneumonia.* This is the most common variety and affects the lower lobe of one or other lung. The right lung is more commonly affected than the left.

(ii.) *Double Pneumonia.* In this both lungs are involved. It is unusual for both lungs to be affected simultaneously, the second lung usually being attacked after an interval of several days.

(iii.) *Apical Pneumonia.* In this variety it is the upper lobe which is affected. It is far more common on the right side than on the left, and is most frequently seen in children. Delirium and meningismus are particularly liable to occur in this variety.

(iv.) *Central Pneumonia.* In this variety consolidation is restricted to the central portion of the lobe and may not reach the surface at all. As a result the physical signs in the chest may be few or none, while the clinical picture will be that of undoubted pneumonia.

(v.) *Migratory or Creeping Pneumonia.* In this somewhat rare variety the signs of resolution in one part will be accompanied by those of developing consolidation in an adjacent area. In consequence the temperature chart is likely to show a number of pseudo-crises, and the disease will appear to have terminated by lysis. This variety is more frequently associated with the influenza bacillus than with the pneumococcus.

(vi.) *Terminal Pneumonia.* Lobar pneumonia may sometimes occur as a terminal event in elderly or debilitated subjects, though it is not so common in this connection as is a lobular type of inflammation. The initial weakness of the patient may prevent an adequate reaction to the infection—that is to say, there may be little or no increase in either the rapidity of the respiration, or the height of the temperature, while the primary illness may make

systematic examination difficult, with the inevitable result that this condition may easily escape recognition during life. It is probable that other organisms than the pneumococcus are often the cause of this variety of lobar pneumonia.

(vii.) *Asthenic Pneumonia.* In addition to the terminal variety it may sometimes happen that a given individual, though apparently healthy, may be affected with lobar pneumonia and yet fail to show the usual temperature and respiration reaction. At the same time the ordinary leucocytosis will not be found. This variety is particularly fatal, and chronic alcoholism should be suspected in such cases.

Complications. Two main classes of complications can be recognised in lobar pneumonia.

First, local complications affecting the thoracic viscera and secondly metastatic inflammations indicating a profound septicæmia.

(i.) Local Complications. (*a*) *Delayed Resolution.* The crisis may not occur until the third week. On the other hand, even when the crisis has occurred at the normal time, the physical signs of consolidation may persist for several weeks. In many such cases there will be little or no constitutional disturbance, but in others there may be persistent pleuritic pain, dry, hacking cough, with scanty expectoration, sweatings, and irregular pyrexia. In a very small proportion of cases fibrosis of the inflammatory exudate may lead to contraction of the chest, and ultimately a fibroid lung may develop with the probable accompaniment of bronchiectasis.

(*b*) *Abscess of the Lung and Gangrene of the Lung.* It occasionally happens that the patient is deficient in the power of manufacturing autolytic enzymes. Resolution is therefore prevented, while the collection of dead and dying leucocytes affords a favourable pabulum for the multiplication of secondary organisms such as strepto- and staphylococci. The natural result is the formation of an abscess cavity within the lung. In more severe cases gangrene may take place in the affected part. Clinically the supervention of a septic type of temperature, with profuse perspirations, etc., combined with the appearance of a foul purulent expectora-

tion and the persistence of a dull area in the lung, may lead to the diagnosis of the abscess. In the case of gangrene the sputum will be profuse, watery, and of a dark plum colour (so-called "prune-juice"); this, with its peculiar and unforgettable odour, which is also imparted to the breath of the patient, will leave no room for error.

(*c*) *Pleurisy.* A fibrinous pleurisy is the rule in lobar pneumonia. A few ounces of turbid yellow fluid are usually poured out. Occasionally the effusion may be much more abundant; the ordinary signs of pleural effusion will then be apparent. Sometimes so much fibrin is formed that the lung sounds are more or less completely cut off.

(*d*) *Empyema.* During the second week of pneumonia, or later, pneumococcal empyema may occur. If the crisis has occurred, the temperature will rise again and become irregular in type. Rigors may occur, and the patient will be worried by a frequent throaty cough and repeated perspirations. The physical signs will be considered under "Differential Diagnosis" (p. 53).

(*e*) *Pericarditis.* This is usually a complication of left-sided basal pneumonia, the infection spreading by direct extension through the parietal pericardium. Since the inflammation is usually purulent from the commencement, pericardial friction may not be audible. The diagnosis is therefore extremely difficult, and will depend upon increasing dyspnœa, tachycardia, and the other signs of cardiac dilatation, with possibly muffling of the heart sounds and a triangular area of cardiac dulness as the effusion increases in amount. Pericarditis may also occur as a metastatic septicæmic inflammation apart from direct extension.

(ii.) Metastatic or Septicæmic Complications. (*a*) *Ulcerative Endocarditis.* The symptoms of this condition will be the same as those of ulcerative endocarditis starting in any other manner. The pneumococcus is second only to the streptococcus in the frequency with which it produces this disease. In the majority of cases it is possible to recover the organism from the blood.

(*b*) *Pericarditis* (*vide supra*).

(*c*) *Arthritis.* Pain and swelling of one or more joints, with a rapid effusion which often becomes purulent, occurring

in the course of a lobar pneumonia will leave little doubt as to the nature of the organism at fault.

(*d*) *Meningitis.* Meningitis in the course of a pneumonic septicæmia is rapidly fatal. The inflammation is usually vertical. The intractable headache and vomiting, the tendency to convulsions and paralyses, together with the rapid development of unconsciousness, will suggest the diagnosis. Lumbar puncture will show an excess of polymorphonuclear leucocytes, while the pneumococcus may be demonstrated in the film preparations and may be cultivated either directly or after inoculation of a mouse.

In this connection it must not be forgotten that pneumonia, and especially *apical pneumonia in children*, may from pure toxæmia present a clinical picture of so-called meningismus which will very closely resemble true meningitis.

In such cases the cerebral symptoms will completely disappear with the crisis, while, if necessary, the diagnosis can be settled at any time by lumbar puncture, when normal cerebro-spinal fluid will be obtained.

(*e*) *Peritonitis.* This complication is rare; it is most frequently seen in children. It is of remarkably sudden onset, the peritoneum appearing to fill up with pus in a few hours. There will usually be more or less abdominal pain, often referred to the umbilicus and accompanied by diarrhœa. The rapid supervention of extreme distension and rigidity will excite suspicion as to the true state of affairs. It must, however, be clearly understood that in many cases of plain, uncomplicated lobar pneumonia the earliest symptoms may be abdominal pain and diarrhœa.

Diagnosis. Attention should be paid to the following characteristics of an attack of pneumonia :—

(i.) *The History.* The sudden onset with rigor and vomiting, followed in a few hours by cough and a pain in the side.

(ii.) *The Appearance.* The flushed face and bright eyes, labial herpes, rapid respirations, and the attitude (low down in the bed, either on the back or side).

(iii.) *The Temperature.* The temperature rises suddenly to 103° or 104°; it remains at this level with a slight remission for from seven to ten days, when it falls by crisis to below normal. In many cases there is a rise of one or two

degrees on the day following the crisis, after which the temperature returns to normal and remains there.

(iv.) *The Respiration.* The frequency of the respirations is much increased, more so in children than adults (35 to 60), but there is no true difficulty of respiration.

(v.) *The Pulse.* The pulse (about 110) is relatively slow considering the temperature. The pulse respiration ratio is much diminished—that is to say, instead of, as in healthy people, being about 4 to 1, in pneumonia it is usually about 5 to 2.

(vi.) *The Blood.* There is a well-marked polymorphonuclear leucocytosis, the white cells usually number from 30,000 to 40,000 per cubic mm. This leucocytosis appears within the first twenty-four hours and persists till shortly after the crisis.

(vii.) *The Urine.* The urine is concentrated, scanty and high coloured. The chlorides are much diminished and often entirely absent.

This retention of the chlorides occurs to some extent in many acute febrile disorders and also in starvation, but not to the same extent as in pneumonia.

(viii.) *Measurement.* The affected side of the chest will usually measure from $\frac{1}{3}$ inch to $\frac{1}{2}$ inch more than the sound side. The measurement is best taken from the tip of the fifth dorsal spine to the middle line in front.

(ix.) *The Sputum.* For twenty-four hours there is but little sputum, and what there is is thick, sticky, and tenacious. On the second and third days the sputum is more abundant, viscid, and often streaked with brightish blood; for the next three or four days the sputum becomes freer, more copious and contains more blood. The blood is more diffused and of a darker colour. As resolution takes place the sputum becomes very abundant, more fluid, and is often of a uniform dark red-brown coloration.

Differential Diagnosis. (i.) FROM OTHER ACUTE SPECIFIC FEVERS. At the outset it may be difficult, and especially so in children, to diagnose pneumonia from scarlet fever, measles, etc. The pulse respiration ratio is quite different, while the early appearance of pain in the side, or pleuritic rub on the one hand, and sooner or later of the eruption on

the other, will usually be sufficient. In those cases of abortive pneumonia where no physical signs in the chest are present throughout the illness the diagnosis of pneumonia may depend entirely on the rapidity of the respirations, the aspect and attitude of the patient, the possible presence of herpes, and the diminution of chlorides in the urine.

(ii.) From Enteric Fever (*vide* p. 24).

(iii.) From Cerebral Conditions. (*a*) *Meningitis* (*vide* p. 7).

(*b*) *Delirium Tremens.* Though delirium tremens does not bear any resemblance clinically to lobar pneumonia, nevertheless it is well worthy of note that in chronic alcoholics delirium tremens is a particularly common complication of pneumonia, and may indeed be the first visible sign of this affection ; it is therefore particularly important to make a thorough examination of the lungs in all cases of delirium tremens.

(iv.) From other Pulmonary Conditions. (*a*) *Pleurisy, without Effusion.* The constitutional symptoms are not so severe. The temperature is more irregular, the affected side is not increased in girth, the signs of consolidation do not appear.

Since a large proportion of cases of primary pleurisy are in reality due to tuberculosis, the special tests for this condition (*vide* p. 105) will often give a positive result.

(*b*) Pleural Effusion. Where there is fluid in the pleural sac the cardiac apex beat is displaced towards the sound side. The percussion note over fluid is more absolutely dull than over the solid lung ; it is woolly rather than wooden. The breath sounds are cut off partially or entirely, while tubular breathing, if present, is usually only to be heard at the upper limit of the dull area. The area of dulness does not follow in shape the anatomical boundaries of the pulmonary lobes. Above the dull area will be a skodaic percussion note, and here the voice sounds will have a nasal twang. An encysted collection of fluid may be more difficult to diagnose, since it is more likely to have been present for a considerable time ; under these circumstances secondary collapse of the lung may lead to contraction of

that side of the chest, and the apex beat, so far from being displaced to the sound side, may actually be pulled over towards the lesion. By very reason of its chronicity the clinical picture of pneumonia will be wanting, the respirations will not be hurried, and the patient may not appear *acutely* ill. In any case of doubt the diagnosis may be settled by exploration with a hollow needle. The point of maximum dulness should be selected for the puncture (*vide* also "Pleural Effusion," p. 306).

(*c*) *Empyema.* The above remarks *re* pleural effusion apply for the most part to empyema, but it must be remembered that empyema is often localised and frequently starts between the lobes of the lung; further, tubular breathing is often more extensive over a purulent collection than over a serous effusion.

The following features may prove of service in the diagnosis of empyema as against pneumonia :—

(i.) Œdema of the chest wall.

(ii.) An irregular or septic temperature.

(iii.) Frequent perspirations.

(iv.) A lower leucocyte count, viz., 15,000 to 20,000.

(v.) A throaty and unproductive cough.

(vi.) An X-ray examination will show a more complete opacity and a more complete immobility of the diaphragm on the affected side where pus or fluid is present than where the lung is solid.

It remains to say that it is often impossible to settle the diagnosis except by exploratory puncture.

(*d*) *Collapse of Lung.* Extensive collapse of lung may give the same physical signs as inflammatory consolidation. It may occur quite suddenly in the course of a bronchopneumonia. Again, pericarditis, or anything which interferes with the proper action of the diaphragm, *e.g.*, peritonitis, may cause extensive basal collapse in one or both lungs. Except when the collapse is caused by pleural effusion or pneumothorax, the affected side of the chest will measure *less* than the sound side. In most cases of pulmonary collapse there will be no leucocytosis, neither will the patient appear so acutely ill as in pneumonia.

(*e*) *Acute Pneumonic Pulmonary Tuberculosis.* This affec-

tion is most frequently seen in the upper lobe, and exactly resembles an apical lobar pneumonia in its mode of onset, physical signs, and in the aspect of the patient. It is indeed an acute inflammation of the lung with a lobar distribution, only it is caused by the tubercle bacillus, not by the pneumococcus.

The following points may lead the observer to suspect the true state of affairs, though it may be impossible to verify the diagnosis for several days :—

(i.) The pulse respiration ratio may not be so profoundly altered, and the absolute pulse-rate is often under 100 per minute.

(ii.) The temperature may show greater variations than in true pneumonia.

(iii.) Herpes labialis is not so common.

(iv.) There may be little or no sputum at first.

(v.) There will be no crisis.

(vi.) During the second week, if the patient survives, the temperature will show an extreme daily variation ; there are likely to be perspirations, and later the patient may begin to expectorate large quantities of liquid purulent sputum in which enormous numbers of tubercle bacilli can be found. By this time definite signs of cavitation may be manifest.

(v.) From Abdominal Conditions. The first symptoms of lobar pneumonia may be abdominal in character. Pain, diarrhœa, and vomiting are not infrequent. In children the pain may be referred to the abdomen throughout the disease. In many cases this is due to the pleuritic involvement, causing the pain to be referred along the course of the intercostal nerves.

Should the disease be on the right side, appendicitis may be suspected.

The diagnosis can usually be settled by a careful examination of the lungs as well as of the abdomen. It will be found that there are no objective signs in the abdomen, and if rigidity be present it will be voluntary rather than reflex, whereas there will be restricted movement on one side of the chest, even if there is no more definite indications of pneumonia. The leucocytosis is higher in pneumonia than

in acute abdominal conditions, and in the latter there is no diminution in urinary chlorides unless the patient has been starved.

The shallow nature of the quickened respirations, as well as the attitude of the patient, should afford confirmatory evidence of the true site of the lesion.

XVI. RELAPSING FEVER.

Definition. A specific infective disease characterised by sudden onset, high temperature, crisis and a relapse occurring one week later.

Bacteriology. The disease is due to the Spirillum or Spirochæta Obermeieri, a member of the protozoa, the length of which is about three times the diameter of a red blood cell.

During the height of the fever the organism is present in the blood-stream, from which it disappears after the crisis.

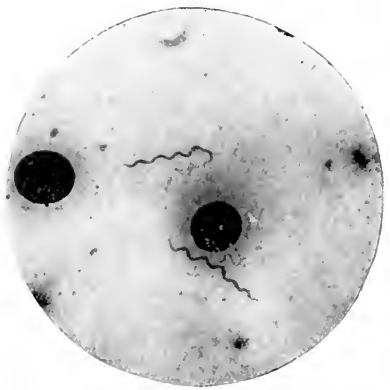


Fig. 16.—Spirillum Obermeieri in the blood of a patient infected with Relapsing Fever. Magnification × 1000.

It has not yet been cultivated, but can readily be demonstrated in ordinary blood films stained by the method of Giemsa (*vide* p. 155).

Incubation Period. From a few hours to three weeks.

Course. The onset is sudden, with rigor, headache, backache, giddiness, and vomiting. Joint and muscle pains are frequent. Though constipation is the rule, diarrhœa may be troublesome.

The temperature quickly rises to from 104° to 108°. The skin is jaundiced; the liver and spleen are enlarged and tender. Occasionally there is an eruption of small rose-pink papules on the trunk and shoulders. The temperature remains high for about a week, when it falls by crisis, usually to 96° or 97°. At the time of crisis there are profuse perspira-

tions, diarrhœa and diuresis, while occasionally there may be bleeding from the mucous membranes.

In a few days the temperature has risen to normal and the patient feels quite well, but about the seventh day from the crisis there is another attack in all respects similar to the first, except that it is usually about two days shorter. Commonly there is but one relapse, but there may be several.

Complications. Hæmorrhage from the mucous membranes may be severe, while the diarrhœa may assume a dysenteric form ; otherwise the complications are those of any other acute fever.

Diagnosis. The history of an epidemic or of exposure amid surroundings of squalor and destitution are important. Adolescence is the most likely age for infection to occur.

The course of the disease, with its relapse on the fourteenth day, is quite characteristic, while the discovery of the organism in the blood of the patient will leave no further room for doubt.

XVII. RHEUMATIC FEVER.

Acute and Subacute Rheumatism.

Definition. A specific infective disease characterised by irregular pyrexia, perspirations and inflammation of fibrous tissues, particularly in the joints and heart.

Bacteriology. Its specific organism has not with certainty been identified, but Poynton and Paine have described a diplococcus which they claim to have recovered from the blood, heart, joints, etc., of rheumatic patients.

It is probable that the infection enters the blood-stream *viâ* the tonsils.

Course. The onset is usually abrupt, with headache, malaise and a rapid rise of temperature, *but rigors are extremely rare.* At the same time there is pain, swelling, and tenderness in a joint, usually one of the larger joints, such as the knee, shoulder, or ankle. In a few hours the pain and swelling will leave the joint first affected and appear in another one and so on. The temperature remains irregular, and the patient suffers from repeated profuse perspirations.

The tongue is moist and covered with a thick white fur ; the urine is high-coloured, febrile, and scanty.

The heart is affected in two ways :—First, the severity of the infection produces a myocarditis, with consequent dilatation at some period during the course in the great majority of cases. Secondly, about 30 per cent. of all cases of rheumatic fever which start with arthritis develop a true vegetative endocarditis of one or more valves, the result being permanent valvular deformity to a great or less extent. Anæmia develops early in the course of the disease, and a well-marked leucocytosis is the rule. The course of rheumatic fever extends throughout six to ten weeks, a period that is not appreciably shortened by treatment, though the temperature is reduced and the patient kept free from pain by the use of salicylates.

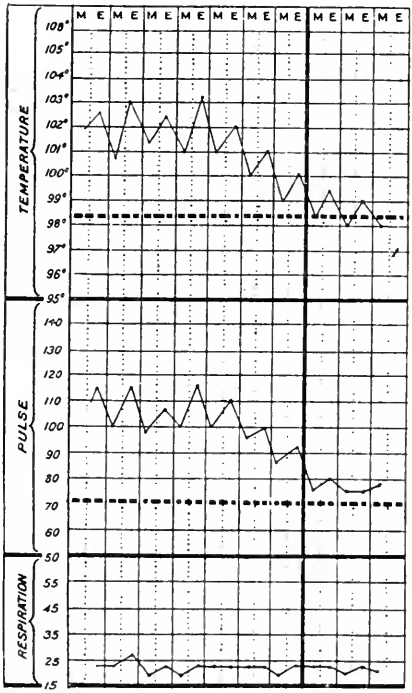


FIG. 17.—Chart from a case of Acute Rheumatism in which Salicylates were not administered till the fourth day.

It is worthy of emphasis that arthritis is not a necessary accompaniment of rheumatic fever ; indeed, the younger the patient the more likely is it for myo- and endo- carditis to be the most prominent features of the case.

Other Manifestations of Rheumatism. (i.) *Sore Throat.* Both tonsillitis and catarrhal inflammation of the fauces and pharynx are commonly met with.

(ii.) *Rheumatic Nodules.* These are small masses of fibrous tissue attached to the periosteum or tendon sheaths

close under the skin. They are seen particularly on the elbows, knuckles, knees and other bony prominences ; they usually persist for a few weeks only. It is very rare for them to occur in the first attack of rheumatic fever ; they are therefore of grave prognosis.

(iii.) *Skin Eruptions.* Sweat rashes are common, but are not specifically rheumatic.

Erythema multiforme is, however, frequently seen in children, though it is not common in adults. Purpura rheumatica (Schoenlein's disease) consists of showers of minute subcutaneous hæmorrhages about the trunk and limbs.

(iv.) *Pericarditis.* This may occur at any stage of rheumatic fever ; it may indeed be the first manifestation of the disease. The inflammation is usually sero-fibrinous, and an adherent pericardium is a common sequel.

(v.) *Pleurisy* is not common ; there is usually little or no effusion.

(vi.) *Chorea.* This is usually regarded as a rheumatic infection of the cerebral cortex ; certainly its age incidence is that of rheumatic fever, while it is frequently seen in association with rheumatism, and it is often complicated by endocarditis.

Complications. (i.) *Hyperpyrexia*—that is to say, a temperature remaining for more than a few minutes at a higher level than 106°—is a serious complication of rheumatism, though, fortunately, it is rare. It is more common in men than women, and in the first attack of rheumatism. It does not occur in children.

(ii.) *Cerebral Rheumatism.* By this is meant rheumatism accompanied by marked delirium or other cerebral symptoms. It is very rare, and when present is not infrequently accompanied by hyperpyrexia. It must be remembered that salicylates may give rise to delirium in susceptible subjects.

Diagnosis. In adults the diagnosis depends upon the sudden onset of illness, with pain and inflammation flitting from joint to joint, profuse acid perspirations with their characteristic odour, the irregular pyrexia, and the rapid development of anæmia.

It must be remembered that rheumatic fever is a disease

of children and young adults, and that it is rare for a *first* attack to occur after the age of thirty.

In children there may be more difficulty in establishing the diagnosis, for although there will usually be a history of transient limb pains, in many cases all joint inflammation will be absent ; the acid perspiration is well marked, and the anæmia is even more severe than in adults. Again, examination will frequently reveal a tonsillitis or some cardiac lesion. Any case of prolonged or oft-recurring pyrexia without obvious cause in a child should arouse suspicion of rheumatism. In all cases a careful history should be obtained, for there seems little doubt that heredity plays an important part in the rheumatic diathesis, and it is extremely common for brothers and sisters to be affected.

Lastly, the history of a previous attack of rheumatism is important, as one attack undoubtedly predisposes to another.

Special Features. (i.) *The Joints.* The larger joints are usually affected ; their characteristic serial involvement has already been mentioned. The affected joints are swollen, puffy, hot and extremely tender. Ordinarily there is little or no alteration in colour, though there may be a faint pink blush. They contain a slightly turbid fluid, but do not suppurate ; neither is there any permanent deformity.

(ii.) *The Heart.* Rheumatism is by far the commonest cause of so-called "Simple Endocarditis." This is likely to occur during the height of the attack. Since the mitral is the valve most often affected, mitral murmurs, systolic or diastolic, are commonly the first signs that endocarditis is present. It is, however, necessary to remember that cardiac dilatation is very often present from myocardial intoxication, and that a systolic murmur of passive mitral regurgitation is likely to be met with without there being of necessity any structural valve change. It will be impossible to differentiate these conditions until the end of the illness, by which time the heart will have recovered from any passive dilatation, or will have accommodated itself to meet the requirements of a damaged valve leaving permanent signs thereof, such as hypertrophy and persistent murmurs.

Lastly, a true hæmic bruit, systolic in time and of maximum intensity at the pulmonary area, may occur as the direct result of the anæmia and will disappear as the anæmia is relieved.

Differential Diagnosis. (i.) *From Acute Gout.* Gout attacks much older people ; it affects smaller joints, which are shiny, œdematous and purple, often with distended veins running over them.

(ii.) *From Gonorrhœal Arthritis* (*vide* p. 33).

(iii.) *From Acute Rheumatoid Arthritis.* Here the smaller joints are likely to be affected, and there is not the same tendency for the swelling to wander from joint to joint. Permanent peri-articular thickening commonly results. The particular odour of rheumatic perspiration is likely to be wanting.

(iv.) *From Pyæmia.* A septic focus is commonly present, the metastases are not necessarily limited to joints, and when joints are affected they are likely to suppurate.

(v.) *From Scarlatinal Arthritis.* Clinically these joints may be indistinguishable from those of rheumatism, but as a general rule the presence of peeling or history of scarlet fever should prevent error.

(vi.) *From Osteomyelitis.* This is likely to occur at the lower end of the femur or the upper end of the humerus. There is often a history of injury. The affected part may be red and œdematous, rigors are probable (these *do not* occur in rheumatism), and careful examination will reveal the fact that the joint itself is not affected by the inflammatory process.

It remains to say that the differential diagnosis of multiple arthritis is often extremely difficult and that the diagnosis of rheumatism can sometimes only be made after the tentative exhibition of salicylates, which seem to exert a specific action in relieving the pain and reducing the inflammation of joints affected by this disease.

XVIII. SCARLET FEVER.

Definition. A specific infective disease particularly affecting children, characterised by a sudden onset, a sore throat, and a red rash.

Bacteriology. The specific micro-organism has not yet been identified, but it has long been thought to be a streptococcus. An additional evidence in favour of this view is furnished in the favourable results reported from the treatment of certain of the more severe cases of septic scarlet fever by the administration of protective sera that have been

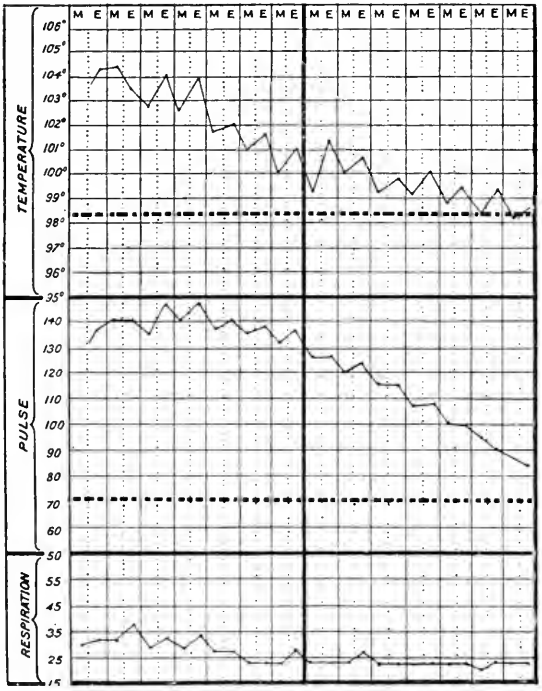


FIG. 18.—Chart from a case of Scarlet Fever.

obtained from animals immunised against streptococci from the throats of scarlet fever patients.

Incubation Period. One to seven days (usually two or three).

Course. The invasion is abrupt, with rigor, vomiting, and sore throat. In very young children there may be convulsions. The temperature rises on the first day to 103°—104°. The skin is hot and pungent, and the pulse is *extremely* rapid, 160 to 180 per minute. On the second

day the rash appears; it consists of two parts—a diffuse blush and minute punctate papules. It appears first at the root of the neck and spreads rapidly over the entire body. On the face, the palms of the hands, and the soles of the feet the papular element is absent, the blush alone being present, while the skin around the mouth escapes altogether (circum-oral pallor).

The rash starts to fade about the fourth day, and as it fades a desquamation commences which may continue for weeks or even months.

The fauces are intensely injected, and on the tonsils there is usually a yellowish exudate; the cervical glands are generally enlarged. The tongue at first is moist and covered with white fur, through which project the swollen papillæ; but by the second or third day the fur has disappeared, leaving a glazed surface on which the elevated papillæ are distinctly visible. The constitutional symptoms increase in severity till the fourth day, when as the rash begins to fade so does the temperature come down by lysis and the patient feels better.

A slight febrile albuminuria is common during the height of the disease. A definite leucocytosis is invariable.

Varieties. In addition to the ordinary form described above the following varieties of scarlet fever may be met with.

(i.) *Septic or Anginose Variety.* In this form there is extensive ulceration of the throat, the secondary cervical adenitis is extremely severe, the glands often break down, and ulceration and necrosis of the cutaneous tissues in the neck may follow.

(ii.) *The Toxic or Malignant Variety.* Here there are symptoms of profound toxæmia, and delirium is common, while hæmorrhagic spots and even death may precede the true eruption.

Complications. (i.) *Otitis Media.* This is due to the spread of infection up the eustachian tube. The chief symptom is earache, and in a little while examination of the ear reveals a bulging membrana tympani. If the membrane is not incised, it will rupture and a purulent otorrhœa will result.

(ii.) *Pulmonary Complications.* Pleurisy, pneumonia, and even empyema may in rare cases follow scarlet fever.

(iii.) *Cardiac Complications.* Endocarditis and pericarditis have been described. In the septic variety an ulcerative endocarditis, just as in septicæmia from any other cause, may supervene.

(iv.) *Nephritis.* An acute glomerular nephritis is a common complication in the second, third, or fourth weeks of scarlet fever. It must not be confounded with the febrile albuminuria of the first week.

The nephritis may be of such severity that there is suppression of urine, and the child dies in a few days with all the symptoms of acute uræmia. More commonly there is œdema of the face and extremities, the urine is smoky and contains tube casts and albumin, and the case becomes one of chronic parenchymatous nephritis. With proper treatment, however, recovery occurs in the majority of cases.

It is important to remember that in certain epidemics of scarlet fever there may be cases of nephritis due to the scarlet fever toxin, but occurring in patients who have shown no other sign of the disease.

(v.) *Arthritis.* (*a*) An acute suppurative pyæmic joint, part of a septicæmia, and so to be looked for in the septic type of case.

(*b*) A multiple arthritis analogous to rheumatism or gonorrhœal infection occurring in the second or third week and tending to affect many joints, particularly the smaller joints of the hands and feet.

Diagnosis. The tendency of scarlet fever to affect children between the ages of one and ten is significant; babies under one usually escape. The history of exposure to infection is important.

In the early cases the diagnosis must rest on the sudden onset, particularly with vomiting, the rapid rise of temperature, the *extremely* rapid pulse (temperature, 104°; pulse, 160), the dry and pungent skin, and the presence of an injected throat.

The appearance of the characteristic rash on the second day will clinch the diagnosis, but the type of rash is by no means constant. The papular element may be entirely

absent. The severity and persistence of the rash in the groins and axillæ are important points. While the tongue is furred, it is of no special diagnostic value, but a glazed papillated tongue combined with bright red injected fauces and tonsillar exudate is of great value. It is not too much to say that any erythematous eruption occurring in a child on the second day after the onset of sudden illness with vomiting, should be regarded as potentially scarlet fever.

In late cases, but in the absence of desquamation, there are often present circumscribed dark-brown raised patches on the outer aspects of the shins and upper arms ; these are considered to be diagnostic of scarlet fever and are said to be present in 50 per cent. of all cases.

When there is desquamation it is important to consider the nature of the peeling, for many conditions other than scarlet fever are followed by this process.

The following types of desquamation are more than suggestive of scarlet fever :—

(i.) *Glove Peeling*—that is to say, massive shedding of the cuticle of fingers and toes.

(ii.) *Ring Peeling.* This is best seen on the chest and shoulders.

(iii.) Desquamation of any sort starting on the hands or feet from two to three weeks after a generalised eruption.

Differential Diagnosis. (i.) *From Measles* (*vide* "Measles," p. 41).

(ii.) *From German Measles.* In this disorder there is usually little or no constitutional disturbance, and even if the temperature is high it is not accompanied by such an extremely rapid pulse, as in scarlet fever. Again, the rash appears first on the face, where papules can easily be recognised, whereas in scarlet fever the rash is first seen on the chest and neck, while the papular element is altogether wanting from the face. Diffuse glandular enlargement, especially in the posterior cervical triangles, is almost invariable in German measles, while in scarlet fever the enlargement is limited to the submaxillary glands.

(iii.) *From Diphtheria.* It must be remembered that a toxic erythema may occur in diphtheria, that there may be a severe membranous inflammation of the fauces in scarlet

fever, and lastly that diphtheria and scarlet fever may coexist. As a general rule it may be stated that a dull earthy toxæmic appearance with a *low* temperature is in favour of diphtheria, while a flushed face and dry pungent skin and a *high* temperature is in favour of scarlet fever. Further, the erythema of diphtheria is limited to the trunk, is less persistent, and of a darker colour than the rash of scarlet fever. Bacteriological examination of the throat will settle whether diphtheria is present or not (*vide* also "Diphtheria," p. 13).

(iv.) *From Follicular Tonsillitis.* This disease is not very common in young children ; it may, however, be associated with a patchy erythema.

Though the onset is abrupt, initial vomiting is rare. If seen early, yellowish points may be seen localised to the tonsillar crypts of one side ; there is extreme dysphagia and a severe aching pain down both sides of the neck. Lastly, in follicular tonsillitis the glazed papillated tongue is always absent and the erythema which is occasionally present is but rarely followed by desquamation.

(v.) *From Small-pox.* In this disease a rosy eruption will occasionally be seen on the second day ; the rash is, however, usually of a purple tint and confined to the lower abdomen, groins and thighs. Throat symptoms are absent. It should not be forgotten that hæmorrhagic small-pox and hæmorrhagic scarlet fever are indistinguishable from one another, since in both cases intense toxæmia accompanied by a purpuric rash may cause death before the characteristic symptoms of the true infection have had time to manifest themselves. The presence of an epidemic of one or other disease may be the only clue available.

(vi.) *From Drug Rashes.* Certain drugs and therapeutic agents may produce a scarlatiniform eruption ; the most important of these are belladonna, quinine, salicylates, chloral, boracic acid, and the enema rash. For the most part any accompanying symptoms will be referable to the toxic action of the drugs concerned—for example, delirium and dilated pupils from belladonna, deafness and tinnitis from quinine and salicylates.

(vii.) *From Erythema Scarlatiniforme* (Acute Exfoliative-

Dermatitis). At first this condition may be indistinguishable from scarlet fever, but peeling starts much earlier, often on the second day, and the eruption tends to persist for several weeks (*vide* p. 607).

XIX. SMALL-POX.

Definition. An acute infective disease occurring in epidemics and characterised by a sudden onset and an erup-

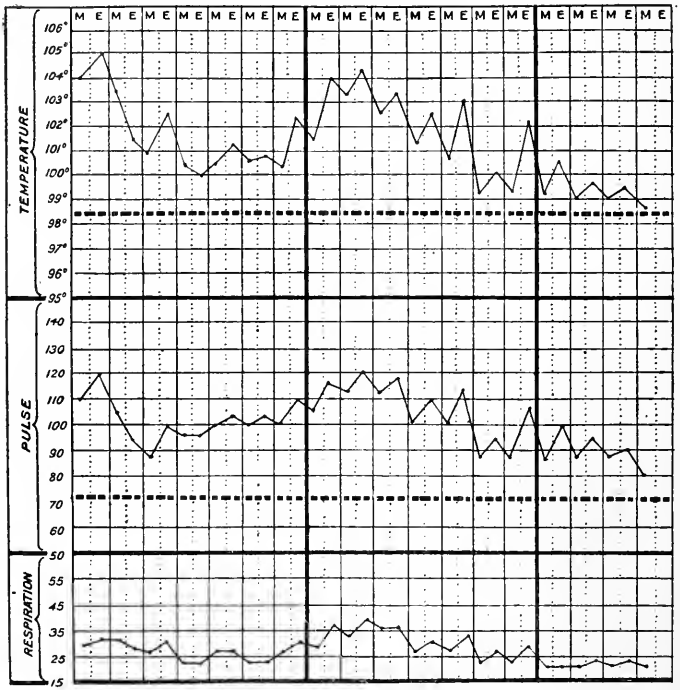


FIG. 19.—Chart from a case of Small-pox showing remission on the third day when the eruption appears.

tion which passes through the stages of papule, vesicle, and pustule.

Bacteriology. No specific micro-organism has been identified, but the researches of Guarnieri, Councilman and others tend to support the view that the causative factor *may* be a protozoon, the Cytoryctes Variolæ, which is found in the skin lesions.

Incubation Period. Seven to fifteen days, usually twelve.

Course. The invasion is abrupt, with rigors, vomiting, headache, and severe pain in the loins. In children the rigor is replaced by convulsions.

The temperature rises to 103° on the first day. Sometimes there is a prodromal rash, morbilliform, scarlatiniform or petechial, which is often localised to the bathing-drawers area or to the extensor surfaces of the arms and legs.

The true small-pox eruption appears on the third day, and with its appearance the constitutional symptoms abate.

The rash is seen as discrete round or oval papules. These are chiefly found on the face and wrists and gradually extend to the trunk and mucous membranes. The papules can be felt as small nodules in the skin before they are visible.

On the sixth day the papules vesiculate; the vesicles are multilocular, and so have an umbilicated appearance.

On the ninth day the vesicles suppurate, the temperature rises, and constitutional symptoms return; the pustules increase in size and become surrounded by a red inflamed areola.

On the twelfth day the pustules dry up with or without rupture and crusts are formed, which drop off during the next three or four weeks, to leave either no mark or the characteristic pitted scar, according to the depth of skin which has been involved in the inflammation.

The temperature declines by lysis from the fourteenth to eighteenth days. There is a definite leucocytosis from the eighth day onwards.

Varieties. (i.) ORDINARY SMALL-POX. This may be divided into two forms—the *discrete*, as described, and the *confluent*. In the latter form the remission of symptoms as the rash appears is less marked, the papules are set close together, and when the stage of suppuration is reached the pocks run together and form large scabby blotches. The earlier the first appearance of the rash the more likely is it to become confluent.

(ii.) HÆMORRHAGIC SMALL-POX. This is described as occurring in two forms :—

(*a*) *Black or Malignant Small-pox* (Purpura Variolosa).

In this form an erythematous rash and hæmorrhages beneath the skin and from the mucous membranes precede the papular eruption and death ensues in from two to six days.

(*b*) *Purpura Hæmorrhagica Pustulosa.* In this variety the hæmorrhages do not occur until the vesicular or pustular stage, and take place at first into the areolæ surrounding the pocks. Mucous hæmorrhages soon follow and death usually occurs during the second week.

(iii.) VARIOLOID, or small-pox occurring in the vaccinated. This as a rule is a comparatively mild infection. The onset is abrupt and the temperature reaches 103°; the papules appear on the third day and are often confined to the face and hands. The disease often aborts in the vesicular stage, and secondary fever is slight or absent.

Complications. (i.) *Laryngitis.* This is the result of the pocks originating in the mucosa of the larynx. Œdema of the glottis, or even perichondritis, may follow. Aspiration broncho-pneumonia is predisposed to by the laryngeal affections.

(ii.) Pleurisy, empyema, and broncho-pneumonia may occur.

(iii.) *Conjunctivitis* was very common in former years. Keratitis and blindness not infrequently resulted. It is probable that the pocks do not occur on the conjunctiva or cornea.

Diagnosis. If there is an epidemic, the sudden onset of illness, with rigor, vomiting, and pain in the back, should suggest small-pox. It may, however, be difficult for a time to differentiate Small-pox from :—

(i.) *Chicken-pox* (*vide* p. 8).

(ii.) *Typhus* (*vide* p. 113).

(iii.) *Measles* (*vide* p. 41).

(iv.) *Cerebro-spinal Meningitis.* When associated with marked purpuric symptoms this disease may be mistaken for a hæmorrhagic form of small-pox. The characteristic fluid withdrawn by lumbar puncture will determine the point (*vide* p. 512).

(v.) *Pustular Syphilides.* These are usually scanty on the face and are not preceded by such severe initial

symptoms as in small-pox. Wassermann's reaction (*vide* p. 76) will furnish important evidence.

(vi.) *Pustular Glanders* (*vide* p. 32).

XX. SYPHILIS.

Definition. A specific infective disease characterised by three more or less definite stages and lesions of extreme diversity, persisting or recurring throughout many years.

Bacteriology. The causative organism is a protozoon, the Treponema pallidum, which is actively motile and has the appearance of a tightly rolled cork-screw. The length of the organism is about 15 μ, and there are at least five spirals to a length equivalent to the diameter of a red blood-cell. The Treponema can easily be recognised in the discharges from primary and secondary syphilitic lesions, in the viscera of congenitally syphilitic babies, and occasionally in gummata.

It has only recently been cultivated outside the body, and syphilis has resulted from its inoculation into man and also the higher apes.

Incubation Period. Up to eight weeks (most commonly twenty to thirty days).

Varieties. (i.) *Acquired Syphilis.*

(ii.) *Congenital Syphilis.*

Course. (i.) *Acquired Syphilis.*

Primary Syphilis. The primary sore commences as a small red, raised papule at the site of inoculation, which is commonly about the corona glandis, often near the frenum, in the male, and not infrequently on the labium minus in the female. Extra-genital chancres are, however, common, and there is no part of the body that may not be so affected.

The papule grows in extent, and in a few days develops into a shallow ulcer with a flat or slightly excavated top and a hard irregular margin. The base feels like either parchment or gristle, according to its thickness; there is usually some slight secretion, which may dry into a scab, but occasionally a red papery papule is the only sign of the disease. There is a painless enlargement of the neighbouring

inguinal glands, and sometimes the lymphatics on the dorsum of the penis become palpable.

In ten or twelve weeks, if untreated, the sore heals, leaving a thin papery scar.

Secondary Syphilis. Secondary symptoms become manifest about six weeks after the appearance of the primary sore. They comprise :—

(*a*) Constitutional disturbance ; this may be severe, but more usually is limited to transient pyrexia and malaise.

(*b*) *Cutaneous Syphilides.* Any form of skin eruption may occur. The most common is a roseolar rash, best seen on the abdomen and forehead, the palms of the hands and soles of the feet ; papular rashes are not infrequent ; squamous rashes resembling psoriasis occur, but the scales are less shiny, the underlying surface is more coppery, and the knees and elbows are not so frequently involved. The pustular syphilide, apart from the history of the disease, may closely resemble the pustules of small-pox.

It is important to note that secondary syphilitic eruptions do not itch, are usually symmetrical, leave a coppery pigmentation on fading, and tend to come and go at intervals of weeks or months during the two or three years following infection.

(*c*) *Affections of Mucous Membranes and Muco-cutaneous Junctions.* A sore throat is the most common manifestation of secondary syphilis. There may be simple injection of the fauces, tonsillitis, or painless symmetrical, white, superficial, "snail-track" ulcers on the palate and fauces.

Mucous patches are localised inflammatory areas in the mucosa of the mouth, nose, anus, or vulva. In these regions the papillæ are frequently hypertrophied, forming the so-called "syphilitic wart" ; if from excess of moisture these œdematous papillæ fuse together, a sodden mass or "condyloma" is the result.

(*d*) *Affections of the Lymphatic Glands.* There is commonly a diffuse, moderate enlargement of the lymphatic glands throughout the body. They are hard,

discrete and shotty. Of special significance is enlargement of the gland above the inner condyle of the humerus and of those in the posterior cervical triangles.

(*e*) *Other Manifestations.* Iritis, if present, usually occurs in the first six months of the disease ; both eyes are generally involved, one soon after the other.

Osteo-copic pains are shooting pains in the long bones and are most complained of in the night. Painless effusion into the joints may occasionally be present.

Loss of hair (alopecia) and chronic inflammation of the nails may be seen, while anæmia may be severe. As a general rule secondary symptoms cease after about eighteen months.

TERTIARY SYPHILIS. Tertiary lesions or gummata commonly occur about three or four years after infection. This form of the disease may affect any tissue in the body, and is prone to recur, often at long intervals, throughout a great many years.

The typical gumma is a greyish-yellow semi-translucent mass, surrounded by granulation tissue and fibrous tissue, and tending to undergo softening and caseation in the centre. If the skin or mucous membranes are affected, deep indolent ulcers will result. Not infrequently a tough yellow slough may be visible at the bottom of such an ulcer. Occasionally the scabs will collect one above the other on a syphilitic ulcer and produce limpet-shaped crusts or " rupia."

Ordinarily there is an interval of immunity from symptoms between the secondary and tertiary manifestations, but sometimes there are frequent slight relapses or " reminders."

Some special manifestations of tertiary syphilis may here be mentioned.

(*a*) *Periostitis.* Syphilitic nodes are localised inflammatory masses occurring in the periosteum, usually on the shafts of the long bones. Secondary infection may lead to necrosis and abscess formations, or the swelling may quiet down into a hard bony nodule.

(*b*) *Circulatory System.* Gummatous mesarteritis is a fertile source of atheroma, and so plays an important part in the etiology of aneurysm.

Endarteritis obliterans is perhaps most marked in the

cerebral and coronary arteries. It consists in proliferation of the intima and consequent occlusion of the vessels. It often results in cerebral thrombosis or fibroid disease of the heart. Hemiplegia from syphilitic cerebral thrombosis is most common within ten years of the primary infection.

(*c*) *The Respiratory System.* Gummatous ulceration of the larynx usually occurs in the later stages of the disease. It is characterised by a thick, husky voice and freedom from pain.

Gumma of the lung is uncommon, but stenosis of a bronchus or of the trachea, the result of syphilitic ulceration, is more often seen.

Primary necrosis of the nasal bones with secondary involvement of the mucous membranes and cartilages causes nasal obstruction and a foul discharge, while serious deformity is a natural result.

(*d*) *The Alimentary System.* The most frequent manifestation here is stricture of the rectum. It is most common in women, is usually quite near the anus, and results from the cicatrisation of gummata which have originated in the sub-mucous tissues.

Gummata in the liver may yield no signs or symptoms ; sometimes, however, a nodular enlargement of the liver may be apparent, and in exceptional cases jaundice or ascites may result from pressure on the hepatic duct or portal veins respectively.

(*e*) *Nervous System.* In addition to the arterial disease mentioned above, gummata may develop in the pia mater. They may be either single or multiple.

Pressure on the brain or spinal cord will produce the characteristic symptoms of tumour in these regions. There may also be diffuse gummatous meningitis, which tends to involve the nerve roots where they leave the brain stem or cord. This inflammation usually involves the pia mater around the brain, but in the cord the dura mater is especially affected, the resulting condition being known as " Pachy-meningitis " (*vide* also p. 542).

Parasyphilitic Affections. Tabes dorsalis, general paralysis of the insane, and certain forms of spinal paralysis

have been shown to occur only in people who have had syphilis. These diseases are called parasyphilitic affections, because the patients may have been free of any clinical sign of syphilis for very many years; at the same time the spirochæte has been demonstrated in the brain and cord of patients with general paralysis, and is probably present in tabes also.

Lardaceous disease may be a sequel of acquired syphilis if proper treatment has not been carried out.

(ii.) *Congenital Syphilis.* There is no manifestation of acquired syphilis which may not be met with in the congenital form, including, in rare cases, tabes and general paralysis of the insane.

As a general rule the child is born healthy, though in exceptional cases bullous and other eruptions may be visible at birth. More commonly the first sign is "snuffles," occurring in the second to eighth week of life and probably accompanied by a red scaly rash on the buttocks, feet, and other parts. The rash often has a coppery red colour and is sometimes of a papulo-squamous variety, and is then likened to raw ham.

The snuffles may proceed to ulceration and necrosis of the nasal bones, while mucous patches are commonly present about the anus, mouth and eyes. As these heal fissured cracks are formed ("rhagades"), and permanent scarring may result.

Periosteal nodes, especially about the cranial fontanelles, are common, as also is deficient ossification in the centres of the membraneous portions of the skull.

There may be acute inflammation of the epiphyses of the long bones, which may become separated from the shafts.

The liver and spleen are often enlarged, and the child soon presents the dull, earthy, anæmic appearance of a severe toxæmia.

These signs usually disappear in a year or eighteen months; the later phenomena are:—

(*a*) *Hutchinson's Teeth.* The permanent central upper incisors are peg-shaped, being broader towards the gums than at their free edge, and possess a notch, which is a

large segment of a small circle, in the centre of their cutting edges. The other teeth are often dwarfed as well.

(*b*) *Interstitial Keratitis and Disseminated Choroiditis.* These usually appear about puberty, but are sometimes evident about the ninth or tenth year.

(*c*) *Deafness.* This is most likely to happen between the ages of fourteen and twenty-one.

(*d*) *Chronic synovitis,* especially of the large joints, such as the knee, as well as periostitis of the long bones, are other phenomena which may be met with during adolescence.

Both gummata and lardaceous disease are rare in congenital syphilis.

Diagnosis. (i.) *The Primary Sore.* The initial appearance three or four weeks after exposure to infection is important, but it is often impossible to obtain a reliable history.

The syphilitic chancre is nearly always single, but there may be two or three. Further, its presence may be masked by an herpetic eruption or by the coexistence of soft sores ; the typical induration is, however, met with in no other condition.

Indolent buboes in the groin form corroborative evidence.

(ii.) *Secondary Syphilis.* If this is suspected, evidence of a primary chancre must be carefully sought ; for the rest the clinical diagnosis must depend on the pleomorphic, symmetrical character of the rash, the coppery stains where the rash has faded, the evidence or history of sore throat, and the diffuse glandular enlargement. A scaly rash on the palms of the hands and soles of the feet is said to be pathognomonic.

(iii.) *Tertiary Syphilis.* The manifestations of tertiary syphilis are so diverse in their character that the diagnosis may well be impossible. In any case of obscure disease syphilis must be suspected, and in this connection it is important to remember that no station in life is immune, and that unfortunately there is no disease about which it is more difficult to extract a truthful history from the patient.

In the case of women it is doubly difficult, for the primary

lesion may well have been overlooked and secondary manifestations are often extremely slight.

Careful inquiry should therefore be made as to any skin eruption or sore throat, any loss of hair, and especially a history of miscarriages and at what month they occurred.

(iv.) *Congenital Syphilis.* The stigmata of congenital syphilis—namely, the stunted appearance, the earthy complexion, the depressed bridge to the nose, the scars round the mouth, the bossed skull, the hazy corneæ of interstitial keratitis, and the Hutchinson's teeth—present a sufficiently striking picture.

In many cases, however, the majority of these signs are wanting—for example, the rapid onset of deafness about the age of puberty may be the only manifestation. In such cases Wassermann's reaction (*vide infra*) is of great value, though positive reactions are not obtained in quite such a high percentage of congenital cases as in acquired cases. The Wassermann reactions of the parents of suspected congenital syphilitis may afford valuable corroborative evidence, even if the patient himself gives a negative reaction.

Special Methods of Diagnosis. Until recently it was often impossible to establish the diagnosis of syphilis except by the therapeutic method—that is to say, until exhibition of antisyphilitic treatment had cured the disease; but nowadays it is possible to diagnose active syphilis in any stage by one or other of the following methods :—

(*a*) *The Ultra-Microscope.* By this means it is possible to demonstrate the Treponema in the discharges of primary and secondary lesions. The primary chancre and mucous patches are particularly favourable. The lesion is kept free from antiseptics for three or four days; it is then thoroughly washed with plain water, and dried and finally scraped with a blunt scapula (an ordinary pen-nib is useful for this purpose). The first discharge is carefully wiped off, when in a few seconds a bead of serum will exude from the excoriated surface. This is taken on a platinum loop and mixed with a drop of distilled water on a microscope slide, a thin cover-slip is super-imposed, and the preparation is promptly examined with a $\frac{1}{12}$ inch

immersion lens with a dark ground illumination. The ultra-microscope is a mechanism of prisms which can be attached to an ordinary microscope, the result is that any refractile particle is thrown into strong relief against a black background. A drop of cedar-wood oil is placed on the upper surface of the condenser and this is racked up to touch the under surface of the slide. The Treponema will appear as a silvery filament with eight or ten tight spirals moving, often rapidly, across the black background.

The Spirochæta refringens, a normal inhabitant of preputial secretions, may by accident be present and must not be mistaken for the Treponema ; it is a larger organism, with much wider and more irregular spirals.

(*b*) *Wassermann's Reaction.* This reaction depends on properties of the blood serum of syphilitic patients. It cannot be obtained till six weeks after the first appearance of the primary sore, but thereafter it persists so long as there is active syphilis.

To perform the reaction it is necessary to procure the following :—

(i.) The patient's serum, 5 cc. of which can be obtained by veni-puncture (*vide* p. 157).

(ii.) An extract of syphilitic liver. This can best be obtained from a syphilitic fœtus.

(iii.) Healthy guinea-pig's serum, containing a known quantity of complement.

(iv.) The blood serum of an animal, say a rabbit, that has been immunised against the red corpuscles of some other animal, say a sheep. This is obtained by injecting the rabbit with sheep's corpuscles, whereby a body is elaborated in the blood serum of the rabbit which is capable of destroying by hæmolysis the red cells of a sheep.

(v.) A suspension in saline of washed sheep's corpuscles.

Since there is a variable amount of complement present in all blood serum, it is necessary to remove this from the patient's serum and the hæmolytic rabbit's serum. This can be done by heating to 57° C. for two hours.

(i.), (ii.) and (iii.) are now added together and incubated at 37° C. for two hours. If the patient is syphilitic,

his blood serum contains syphilitic antitoxins ; these will unite with the syphilitic toxins in the liver extract by the aid of the complement of the guinea-pig's serum, and in so doing the complement will be fixed and rendered incapable of further action.

(iv.) and (v.) are now mixed together and added to the first mixture, the whole being put into the incubator at 37° C. for half an hour ; but the hæmolytic power of the rabbit's serum for the sheep's blood-cells can only be exercised in the presence of complement ; therefore if the only complement present, namely that of the guinea-pig serum, has already been fixed by the syphilitic toxins and anti-toxins, there will now be no liberation of hæmoglobin from the sheep's blood corpuscles, which will settle to the bottom of the tube leaving clear fluid above. The reaction is then said to be positive.

If, on the other hand, the patient is not syphilitic, there will be no syphilitic antitoxins in his blood serum ; therefore the complement in the guinea-pig's serum will still be free to enable the hæmolytic action of the rabbit's blood on the sheep's corpuscles to take place. As a result the hæmoglobin will be liberated from the sheep's corpuscles and the contents of the tube will assume a uniform pink coloration. The reaction is then said to be negative.

It is found in practice that it is not necessary to use an extract of syphilitic liver, equally good results being obtained from the use of an alcoholic extract of guinea-pig's or rabbit's heart. It is possible that colloidal change is at the bottom of the various interactions outlined above.

The diagnostic value of Wassermann's reaction cannot well be overestimated. When properly performed with control tubes of known syphilitic and known healthy serum the margin of error is probably less than ten per cent. ; further, it is possible to gauge the success of the treatment employed by the rapidity through which successive Wassermann tests pass through stages of no hæmolysis, slight hæmolysis, and full hæmolysis.

The reaction is present in congenital syphilis and also in the great majority of cases of parasyphilitic disease. It must be mentioned, however, that the reaction can be

obtained in certain other diseases which are due to protozoon infections, such as yaws and sleeping-sickness, and also during the acute stages of some of the specific infections, such as pneumonia and sometimes in scarlet fever.

XXI. TETANUS.

Definition. A specific infective disease characterised by tonic spasms of the muscles.

Bacteriology. The causative organism (Bacillus tetani) is a long, slender flagellated rod, one end of which is often occupied by a spherical spore. This produces the characteristic "drum-stick" appearance. The bacillus is slightly motile and grows well at the body temperature, but is anaerobic. This latter quality makes it easier for the tetanus bacilli to grow in a wound which is also infected with some aerobic organisms, such as the Staphylococcus pyogenes, which use up the oxygen in the neighbourhood.

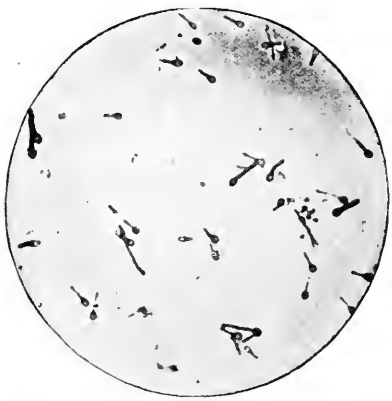


FIG. 20.—Bacillus Tetani showing spore formation. Magnification 1000.

The organism stains well with basic aniline dyes and is Gram-positive. It grows on ordinary culture media, and a stab culture on gelatine produces what is known as the "inverted fir-tree" appearance.

Tetanus is a true toxæmia, the bacilli being strictly localised to the site of the lesion. The toxins manufactured by the bacilli spread by the blood-stream and eventually travel upwards to the central nervous system along the course of the peripheral nerves.

The natural habitat of the tetanus bacillus is the intestinal tract of herbivorous animals and soil, particularly soil that has been well manured, where it is believed to pass a saprophytic stage in its life history.

Incubation Period. Five to fifteen days (usually ten or twelve). The longer the incubation period, the less severe is the attack.

Course. The first symptom is stiffness in the muscles of the neck, though sometimes shivering or rigors may precede this by a few hours.

The stiffness soon involves the muscles of mastication, and gradually a tonic spasm supervenes in the affected parts. The eyebrows are raised and the angles of the mouth drawn outwards, thus causing the characteristic "risus sardonicus."

The spasm soon passes to the other muscles of the body, the back muscles being particularly involved, so that a position of opisthotonos is likely to be assumed.

Although the muscular spasm never relaxes completely, it is liable to profound exacerbations, which may be produced by the most trifling causes. The paroxysms may be accompanied by extreme pain, and the patient perspires freely. As the disease progresses the temperature usually rises (though it may be high from the start), and before death, which usually occurs within four days in those acute cases which have a short incubation period, the thermometer will not infrequently register 108° or 109°. Death may be due to heart failure, asphyxia, or exhaustion.

Varieties. In addition to the ordinary form described above the following varieties may be mentioned :—

(i.) *Chronic Tetanus.* This variety is likely to have a long incubation period (ten to fourteen days). The spasms may be confined to the neck and jaws ; there is little or no pyrexia, and the course may be protracted for several weeks with intervals of comparative comfort. Recovery is usual.

(ii.) *Cephalic Tetanus.* This variety usually follows a lesion to the scalp. The chief features will be *facial paralysis* and pharyngeal spasm. (*Vide* also Hydrophobia.)

(iii.) *Tetanus Neonatorum* or *Tetanus of Newly-born Infants.* This is due to infection of the umbilicus.

(iv.) *Idiopathic Tetanus.* This almost certainly does not exist ; cases so described were probably those in which the abrasion through which the infection was acquired was so small as to escape notice.

Diagnosis. The history of a wound, particularly of a septic wound, in the preceding fortnight is important, but it must be remembered that the site of infection may be completely healed before the onset of symptoms.

Where there is an open wound the organism may sometimes be seen in film preparations of the discharge. If such examination is inconclusive, it is advisable to inoculate a deep glucose-gelatine tube with the suspected material. Incubation of such a tube for forty-eight hours will often show spore-bearing tetanus bacilli mixed with the other organisms. Again, mice or guinea-pigs may be inoculated subcutaneously with the material from the wound, when if tetanus bacilli are present the animal will shortly manifest tetanic symptoms.

The onset of stiffness and spasm in the muscles of the neck and jaws, with the subsequent course of the disease, will usually leave no doubt as to the correctness of the diagnosis.

It is, however, necessary to exclude the following conditions :—

(i.) *Trismus* from other causes :—

(*a*) *Hysteria*. Hysterical trismus is not accompanied by rigidity of the neck, and though there may be an irregular opisthotonos, this latter is transient and only seen in conjunction with a definite hysterical seizure. There is no true risus sardonicus.

(*b*) *Dental irritation*, such as impacted wisdom tooth, etc. Here there is never a risus sardonicus, and a little investigation will reveal the tooth which is causing the trouble.

(*c*) *Acute*, or, more commonly, *Chronic meningitis* or *Intracranial tumour*, causing irritation of the motor part of the fifth nerve. The other signs of meningitis or cranial tumour will be present.

(ii.) *Strychnine Poisoning*. Here there is a sudden onset; the spasm affects the whole body and is never seen first in the neck and jaws, while between the convulsive attacks there is complete relaxation of the muscles.

(iii.) *Hydrophobia* (*vide* p. 29).

(iv.) *Tetany*. Tetany is seen in rickety children and nursing women; it is usually limited to the hands and feet,

which are in a position of adduction and flexor spasm. Trismus is of extremely rare occurrence.

XXII. TUBERCULOSIS.

Definition. A specific infective disease, dependent on the presence of the tubercle bacillus in one or more organs of the body and of the circulation in the blood-stream of the toxins elaborated in the tuberculous lesion.

Bacteriology. The specific micro-organism is a slender non-motile rod-shaped bacillus from 3 to 4 μ in length ; it is often slightly curved, and when stained appears to be beaded. It can be cultivated at a temperature of 37° C. on glycerine agar, glycerinated potato, and other kindred media in the form of yellowish-brown crusts, but it does not grow readily outside the body, and there is usually considerable difficulty in obtaining cultures from tuberculous sputum or the discharges from other tuberculous lesions in man. For purposes of cultivation some of the tuberculous material should be injected subcutaneously into a guinea-pig. In six weeks the animal will be in a condition of tuberculous septicæmia with profuse visceral tuberculosis. The spleen is especially involved and cultures can readily be made from the lesions in this organ.

Owing to the presence of a waxy material in the sheath of the tubercle bacillus, powerful and prolonged staining is necessary for its demonstration. For this purpose advantage is taken of the fact that the tubercle bacillus, in common with the leprosy bacillus, the bacillus of smegma, the butter bacillus, and the timothy-grass bacillus, is acid and alcohol fast—that is to say, that the colour is not lost if the preparation is dipped in acid or alcohol after first being stained with a strong solution of carbol-fuchsin.

It is most frequently necessary to demonstrate the bacillus in sputum or urine. For this purpose the following technique may be adopted :—

The sputum is poured into a shallow dish and a purulent particle is picked out with clean forceps and placed on a slide. This is then spread in a fairly thin uniform film either

by rubbing with a needle or by flattening out with another slide. In the case of urine the specimen should be obtained by catheterisation. It is then centrifugalised and a small portion of the deposit is spread as a film on the surface of a slide. In both cases the film is dried in the air and fixed by passing two or three times through the flame of a Bunsen burner or spirit lamp. Some filtered Ziehl-Neelson carbol-fuchsin solution is now heated to boiling point in a test-tube and poured on to the slides. At the end of three minutes the carbol-fuchsin is washed off and the slides are immersed for a few seconds in thirty-three per cent. nitric acid and again washed in water. This process is repeated until there is just no return of pink coloration when the nitric acid is washed off. A half per cent. aqueous solution of methylene blue is now added for twenty seconds; the preparation is then washed and dried and is ready for examination. If permanent preparations are desired, a cover-slip may be affixed with Canada balsam in the usual manner, but for ordinary purposes a drop of cedar-wood oil may be placed directly on the film and a $\frac{1}{12}$ inch objective used in the ordinary way. When stained in this manner tubercle bacilli are seen as bright-red rods, while any other structures (organisms, cells, fibrin, etc.) that may be present are coloured a pale blue.*

There are three varieties of tubercle bacilli—avian, bovine, and human. It is, however, possible that the differences in these varieties depend on the different environment and that a few generations of growth may be sufficient to convert one form into another. Certainly there is evidence that

* Various sedimentation tests have been devised for the demonstration of tubercle bacilli in the sputum. The best of these is the method of Ellerman and Erlandsen, which is conducted as follows:

(i.) One volume of sputum is mixed with half a volume of 0·6 per cent. sodium carbonate solution in a corked glass, and placed in the incubator at 37° C. for twenty-four hours.

(ii.) The greater part of the supernatant fluid is then poured off, and the remainder is centrifuged.

(iii.) To the deposit so obtained, four volumes of 0·25 per cent. sodium hydrate solution are added, and after thorough mixing, the fluid is boiled.

(iv.) The resulting solution is again centrifuged.

(v.) Films are made from the deposit obtained, and stained in the usual manner.

It is probable that a slightly higher percentage of positive results is obtained with sputa examined by this method as compared with the more usual method described in the text.

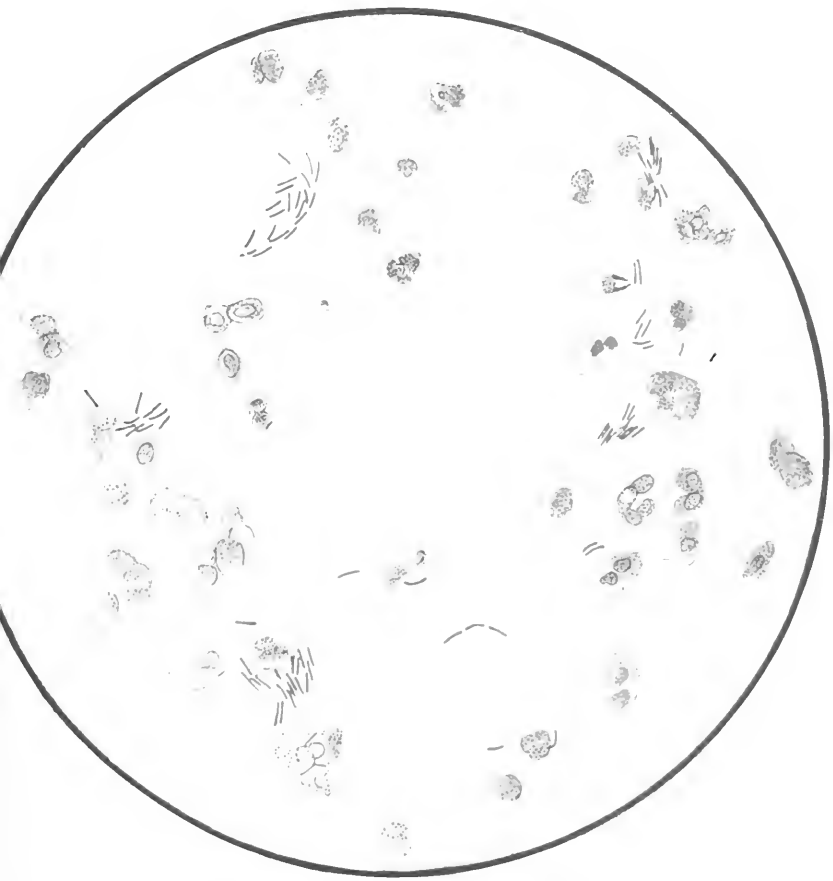


FIG. 21.—Tubercle Bacilli in Urine. Ziehl-Neelson Method Magnification × 1000.

human beings, and especially children, are susceptible to infection from bovine tubercle bacilli.

Since tuberculous lesions may occur in any organ of the body, it will be convenient to consider separately the effects produced in the different anatomical regions.

1. ACUTE MILIARY TUBERCULOSIS.

This disease depends on the introduction into the blood-stream of overwhelming numbers of tubercle bacilli: the result is the formation of countless so-called miliary tubercles widely diffused throughout the viscera. Each tubercle is deposited on the walls of the smaller arteries. They are particularly well seen in the pleuræ, lungs, liver, spleen, and kidneys and in the pia mater at the base of the brain. It is inconceivable that such enormous numbers of bacilli should enter the blood-stream except from a pre-existing tuberculous lesion. The most frequent primary lesions are tuberculous glands, particularly bronchial glands, which may ulcerate into a vein; or fibro-caseous disease of the lungs. Possibly the ulceration of a tuberculous deposit in the intima of a blood-vessel may sometimes be the primary focus. It is obvious that the miliary tubercles are too small *per se* to give rise to any physical signs, and in the case of a primary glandular lesion this, too, may not be susceptible of clinical recognition. So that in many cases of acute miliary tuberculosis the diagnosis must depend largely upon the signs and symptoms of the profound toxæmia which is produced.

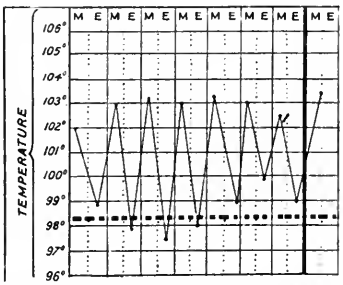


FIG. 22.—Chart to show inverse type of Pyrexia in some cases of Miliary Tuberculosis.

In a considerable proportion of cases, however, the basal meninges are affected, and it is not too much to say that general tuberculosis is often not diagnosed until the characteristic signs of tuberculous meningitis make their appearance. It must further be borne in mind that in this, the

most acute form of tubercular infection, the protective mechanism of the tissues appears to be completely paralysed, and this may account for the fact that the various diagnostic reactions—*e.g.*, von Pirquet, Calmette, the injection of tuberculin, and the estimation of the opsonic index—so often give a negative result in such cases.

Clinically it is possible to differentiate three forms of acute miliary tuberculosis.

(*a*) **The Typhoid Form.** The onset is usually gradual, with rising temperature and a rapid feeble pulse. There is early delirium and a definitely accelerated respiration rate, whilst the patient soon passes into a condition of coma. The temperature is strikingly irregular; often it is of the inverse type—that is to say, it rises in the morning and falls in the evening.

In rare cases there may be abdominal distension and diarrhœa; constipation, however, is the rule. The spleen is often enlarged and albuminuria may be present.

The diagnosis of this form of tuberculosis from enteric fever may be extremely difficult. The following table shows the essential differences:—

Miliary Tuberculosis (Typhoid Form).	*Enteric Fever*
Epistaxis rare.	Epistaxis common.
Pulse rapid.	Pulse slow in proportion to temperature.
Spleen less constantly enlarged and at a later date.	Spleen enlarged early.
Widal negative throughout.	Widal positive after first week.
Herpes fairly common.	Herpes rare.
Rash (if any) an irregular erythema.	Rash papular and in crops.
Moderate leucocytosis with excess of lymphocytes.	Leucopenia.
Possible evidence of local tuberculosis, *e.g.*, lungs or choroid.	Probable presence of Bacillus Typhosus in the blood and urine.

(*b*) **The Pulmonary Type.** This form is most likely to occur as a sequel of chronic pulmonary tuberculosis. In children it not uncommonly follows measles or whooping-

cough. Under these circumstances it is accompanied by a true tuberculous broncho-pneumonia, and it is to this latter condition that the physical signs are to be attributed.

There is always cough, with muco-purulent expectoration, very marked cyanosis, extreme rapidity of respiration, and dyspnœa. Percussion will reveal a uniform impairment of resonance at the bases or scattered dull areas due to broncho-pneumonic consolidation, or, what is perhaps equally significant, widespread areas of hyper-resonance due to a more or less acute compensatory emphysema. With the stethoscope there will be heard fine crepitant râles, widely distributed, while tubular breathing from either

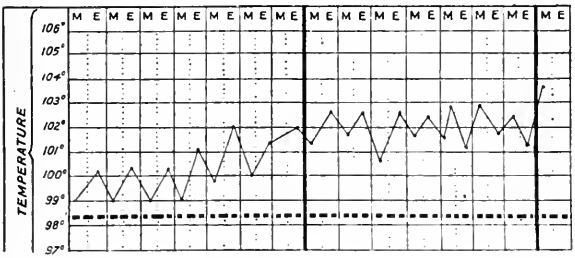


FIG. 23.—Temperature Chart showing continuous type of Pyrexia in certain cases of Miliary Tuberculosis.

consolidation or collapse is relatively common. The sputum may contain tubercle bacilli, but this is by no means invariable. The spleen is usually enlarged. The diagnosis will be seen therefore to depend largely upon the occurrence of cyanosis and dyspnœa, with the signs of capillary bronchitis, or even broncho-pneumonia, occurring in a patient who is known to have fibro-caseous pulmonary tuberculosis, or in the case of a child to have recently suffered from measles or whooping-cough.

(*c*) **The Meningeal Form (Tuberculous Meningitis).** Both the typhoid and the pulmonary forms may be accompanied by tuberculous meningitis, a condition which is only met with in cases of a generalised tuberculosis; but frequently there are no symptoms by which the general infection can be diagnosed until the meninges have become involved.

Tuberculous meningitis is much more common in children

than in adults, though it is rare in the first year of life. The primary focus is frequently glandular, the most common glands being the infra-tracheal, bronchial, or mesenteric. The inflammation affects the base of the brain, the miliary tubercles being deposited along the course of the blood-

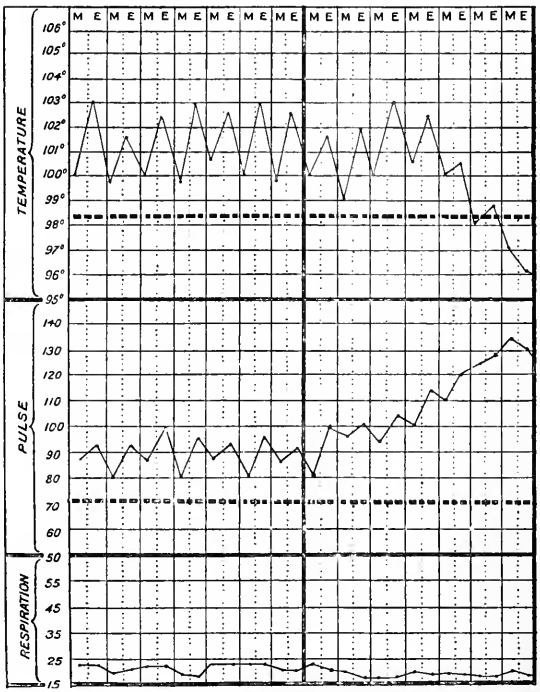


FIG. 21.—Chart from a case of generalised Tuberculosis with Tuberculous Meningitis.

vessels in the pia mater of the inter-peduncular space and the Sylvian fissures.

Course. Commonly there is malaise and general debility for several weeks. Starting from the onset of definite symptoms, three somewhat indefinite stages may be recognised.

First, the *stage of irritation.* This stage may be ushered in by convulsions, followed by severe headache, vomiting, and pyrexia. The child is usually constipated and gives

frequent utterance to the short, sharp hydrocephalic cry; the pupils are contracted, the pulse is slow and irregular, but there is no rapidity of respiration, unless there is a coincident pulmonary lesion. Kernig's sign (see p. 6) is often present.

Secondly, the *stage of depression*. This stage depends upon the gradual increase in intra-cranial tension. The temperature is lower, the headache is less marked, the child lies quiet and appears to be drowsy; the vomiting may cease, but the neck is stiff and the head tends to be slightly retracted, the fontanelle bulges if the child is young enough, the abdomen is retracted, and commonly there is a convergent strabismus. The irregularity of the pulse persists, and the drowsiness is really the onset of coma.

Thirdly, the *stage of paralysis or coma*. In this stage the child is persistently unconscious and cannot be roused. Convulsions are common, strabismus is more marked, and the pupils are dilated and often unequal. The pulse becomes rapid, but the temperature is strikingly variable; it may be sub-normal or it may be high throughout, whilst just before death there is often hyperpyrexia.

Optic neuritis is said to be present before the close in 20 per cent. of all cases, but this is perhaps a rather high estimate. Choroidal tubercles may occasionally be discovered. It is extremely rare for life to be prolonged for more than four weeks from the onset of symptoms.

Although individual cases show great variability in their course, the diagnosis of tuberculous meningitis is not difficult. The slow and irregular pulse, the headache, the vomiting, the ocular phenomena as described above, and the stiffness of the neck are all important points. If any doubt exists lumbar puncture should be performed. The cerebro-spinal fluid is under tension and escapes with a spurt; it is clear, it is sterile, and it contains an excessive number of *lymphocytes*. Further, its power of reducing copper is diminished or absent. In very exceptional cases tubercle bacilli may be present.

Tuberculous meningitis must not be mistaken for cerebro-spinal meningitis (*vide* p. 6).

II. TUBERCULOSIS OF THE LARYNX.

With hardly an exception this is a late manifestation in the course of pulmonary tuberculosis; a few cases of primary disease of the larynx are on record.

The tubercles are formed in the sub-epithelial layer of the mucosa. Their most frequent situations are the true and false vocal cords, the epiglottis, and the inter-arytenoid folds.

Diagnosis. The principal symptoms are hoarseness or aphonia and an irritating cough. When ulceration is present there is both difficulty and pain in swallowing. If the vocal cords are extensively involved the cough may become altered in character.

The laryngoscope is necessary to establish the diagnosis. The mucous membrane, at first pale, becomes thickened with inflammatory exudation, and when ulceration is present the diseased parts present a worm-eaten appearance. The individual ulcers are irregular and shallow with grey bases. The cords are often fixed, either by infiltration or by paralysis of the recurrent laryngeal nerve. When the fixation is due to paralysis the palsy is likely to be of the abductor type. The paralysis is not due to the laryngeal tuberculosis, but to some involvement of the nerve lower down. The right nerve is most commonly affected, and this is thought to be due to its involvement in pleuritic thickening at the apex of the right lung.

Paralysis of the left nerve may sometimes be accounted for by pressure of enlarged glands at the root of the left lung.

Differential Diagnosis. (*a*) *Chronic Simple Laryngitis.* Dysphagia is absent, and the laryngoscope shows swollen and injected mucous membrane, but no ulceration.

(*b*) *Syphilitic Laryngitis.* Secondary syphilis may produce symmetrical superficial, serpiginous, whitish ulcers on the cords or ventricular bands; there is no dysphagia.

Tertiary syphilis may produce diffuse gummatous inflammation going on to deep ulceration and healing in tight fibrous bands. Gummatous deposits are perhaps most common on the base of the epiglottis. There is no dysphagia,

but the voice is very thick and husky. Complete aphonia is rare.

(*c*) *Carcinoma of Larynx.* This is rare before the age of forty and is six times commoner in men than women. In carcinoma starting within the larynx hoarseness slowly increasing in severity may be the only symptom for months. With the laryngoscope the growth appears as a small circumscribed thickening, with a broad base and often with an excoriated surface. Sooner or later deep ulceration will occur and the base of the ulcer is likely to present a nodular appearance.

Carcinoma causes early and extreme dysphagia ; pain is more or less constant, and is often referred to the ear and occiput. In tuberculous laryngitis pain is usually absent except on swallowing, talking, or coughing.

Where there is any doubt some of the secretion from the affected part should be examined microscopically for tubercle bacilli, or a small piece of an ulcer may be excised for histological examination.

III. TUBERCULOSIS OF THE LUNGS.

Pulmonary tuberculosis is met with in four varieties :—

(*a*) Acute lobar-pneumonic tuberculosis.
(*b*) Acute broncho-pneumonic tuberculosis.
(*c*) Fibro-caseous tuberculosis.
(*d*) Fibroid tuberculosis.

(*a*) **Acute Lobar-pneumonic Tuberculosis.** This consists of the sudden invasion of an entire lobe or even an entire lung by the tubercle bacillus ; the result is a massive inflammation in all respects comparable with that of lobar pneumonia due to the pneumococcus.

The onset with rigor, pain in the side and cough, as well as the physical signs of consolidation, make the diagnosis from pneumococcal lobar pneumonia impossible in the earlier stages. A previous history of pulmonary tuberculosis is, however, suggestive, and in a considerable proportion of cases the pulse-rate is not so accelerated as in ordinary pneumonia ; consequently pneumonia with a pulse-rate below a hundred should suggest the possibility of tuberculosis. Ordinarily

the true nature of the malady is not suspected until in place of the expected crisis between the seventh and the tenth days the temperature tends to assume a more remittent character and the sputum becomes muco-purulent and green. By the end of the second week tubercle bacilli and elastic tissue may be present in the sputum, and during the third week there are likely to be the physical signs of softening and cavity formation. A number of patients, especially children, recover with cicatricial fibrosis and contraction of the affected lobe. Possibly in a larger proportion of cases the patient dies of cachexia and exhaustion at the end of eight or nine weeks. Death may, however, take place during the first week from profound toxæmia. The differential diagnosis between tuberculous and pneumococcal lobar pneumonia will depend upon the absence of crisis, the more remittent temperature, the tendency to a less rapid rate of respiration and slower pulse-rate, the liability to perspiration, and lastly upon careful and repeated examinations of the sputum for tubercle bacilli (*vide* also p. 81).

(*b*) **Acute Broncho-pneumonic Tuberculosis.** This variety is more common than the lobar form : it is of frequent occurrence in children and also as a terminal event in fibro-caseous pulmonary tuberculosis. It is rarely primary, being commonly produced by ulceration into a bronchus of a tuberculous gland, or as a sequel of measles and whooping-cough. Like the other varieties of broncho-pneumonia, the onset tends to be gradual rather than acute. The physical signs are those of broncho-pneumonia, fine râles of capillary bronchitis, and often dull areas of varying size, over which tubular breathing may be present. Tubercle bacilli and elastic tissue may be discovered in the sputum, which is usually profuse, muco-purulent, and green. Death as a rule occurs in a few weeks, though in very exceptional cases, especially if the disease happens to have been limited to one lung, chronic fibroid disease may result. The relative frequency with which tuberculous broncho-pneumonia may be found associated with generalised tuberculosis has been indicated in the discussion of the latter (*vide* " General Tuberculosis, Pulmonary Form," p. 84). It must be understood, however, that though the two conditions may well arise from the same

primary lesion (*e.g.*, a caseous bronchial gland), nevertheless their distribution is essentially different, the one being conveyed by the blood-stream, the other by the lymphatics and air passages.

(*c*) **Fibro-caseous Tuberculosis.** This is by far the commonest variety of pulmonary tuberculosis. It is most commonly met with between the ages of 15 and 25, while after the age of 35 the liability to infection seems to be definitely diminished.

Anatomy. The apices of the lungs are first affected in the majority of cases, and the right lung is more liable than the left. The primary lesion is usually about 1½ inches from the extreme apex and nearer the back than the front, a fact which shows the necessity of examining the back of the lungs. The infection spreads downwards, both by direct extension and by the formation of small outlying tubercles conveyed from the primary focus by the lymph-stream. As the disease progresses further foci are formed by the aspiration of tuberculous material into healthy bronchioles. After infection of the right upper lobe secondary infiltration is likely to take place at the apex of the right lower lobe, and then at the apex of the left upper lobe, and conversely should the primary lesion be on the left side. Wherever the primary site, the infection starts with the formation of one or more histological tubercles, round which there is a circumscribed area of reactionary inflammation and consolidation comparable to pneumonia. There is an attempt on the part of nature to shut off the diseased area by the protective formation of fibrous tissue, and since the "tubercle" is essentially avascular there is a tendency for softening and necrosis to occur in its centre. It will thus be seen that from the very outset of the disease there is a perpetual warfare between the tendency of the tuberculous process to spread peripherally, and the attempt of the protective mechanism of the body to limit the destructive process by the interposition of barriers of fibrous tissue.

The chronicity of an individual case depends in a great measure on which of these two processes gains the upper hand. In some cases the tuberculous focus is shut off almost at

its commencement, in others the disease progresses until a bronchus is involved of sufficient calibre to permit the evacuation of the necrotic material in the centre of the lesion; in this manner the formation of a cavity takes place. Stagnation of the secretion in the vomica favours secondary infection with other organisms, and it is to the presence of pneumococci strepto- and staphylo-cocci and other organisms in the more advanced cases of fibro-caseous disease that many of the objective symptoms are to be attributed.

Diagnosis. (1) HISTORY. Though the possibility of direct hereditary transmission may be neglected, a history of existing disease in the parents or brothers and sisters is of importance because parents are likely to infect their children, and the house where tuberculous patients live will become infective unless the most scrupulous precautions be observed.

(2) SYMPTOMS. The onset is usually gradual, and the early symptoms are debility, *easy fatigue* and loss of appetite extending over weeks or months. Progressive *loss of weight* is the rule. Not infrequently the patient will seek advice because of dyspepsia or anæmia. This mode of onset is perhaps more common in women, who are also likely to complain of amenorrhœa. *Night sweats* are an important early symptom: the patient wakes at night drenched with a *cold* perspiration. In other cases pain in the chest, the result of a dry pleurisy, may be the earliest sign. More commonly the patient will seek advice because of a *persistent cough* often dating from a cold in the chest some few weeks or months previously. Along with the cough there will be expectoration, unless it is the dry hacking cough of pleurisy, and possibly some hæmoptysis.

Hæmoptysis may be the first symptom to make the patient think he is ill; early hæmoptysis is usually slight, the sputum is streaked with bright-red blood, or there may be small quantities of nearly pure blood, frothy from admixture with air or in the form of clots. This hæmorrhage is produced by diapedesis from the congestion of the capillaries, which is the result of the early inflammatory reaction; it is comparable to the hæmorrhage in the early days of pneumonia.

Late hæmoptysis often results from the rupture of a blood-vessel in the wall of a vomica. Those vessels which cross a

eavity from one side to the other are nearly always thrombosed, and so do not cause hæmorrhage ; but those which run in the wall of a cavity are likely to form localised aneurysms at their most unsupported parts. These aneurysms project into the cavity and are prone to rupture with the production of more or less profuse hæmoptysis.

The sputum in the early stages is muco-purulent ; as the disease progresses it becomes more and more purulent, eventually assuming the form of isolated greenish-grey flattened discs which sink in water. In our opinion too much importance has been attributed to this nummular form of sputum in the diagnosis of tuberculosis ; the real importance of the sputum lies in the fact that it can be examined for tubercle bacilli and elastic tissue. To examine for tubercle bacilli, *vide* p. 81.

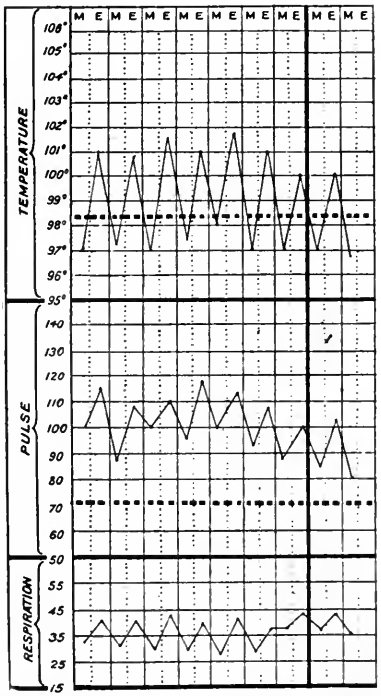


Fig. 25.– Chart from a case of rapidly progressing Fibro-caseous Tuberculosis of the Lungs.

To examine for elastic tissue one of the purulent particles should be squeezed out between two cover-slips. If examined with a hand lens against a black background, elastic tissue can often be recognised as small yellow shreds arranged in a network. If it cannot be recognised in this manner, some sputum should be boiled with an equal quantity of caustic soda solution (20 gr. to 1 oz.) until the mixture is liquid. The fluid should then be centrifugalised and the deposit examined microscopically. For certain diagnosis the alveolar arrangement of the elastic tissue fibres should be made out.

Dyspnœa is not a striking feature of early cases, but as the disease advances there is often considerable breathlessness after exertion. In very chronic cases, where there is considerable fibrosis of one or both lungs, true cardiac dyspnœa occurs from embarrassment of the right heart.

Fever is present in the great majority of all cases; the pyrexia is regular, showing an evening rise and falling to below normal during the early hours of each morning. A small percentage of cases show the inverse type of temperature with a morning rise and an evening fall, which is probably due to the formation of fresh miliary tubercles. The

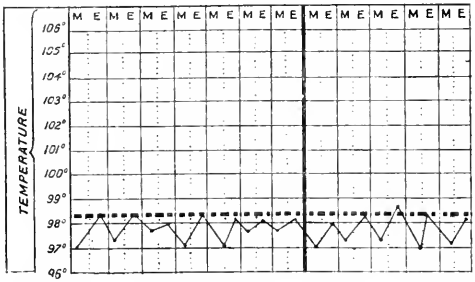


Fig. 26.—Chart of the Temperature in a case of quiescent Fibro-caseous Pulmonary Tuberculosis.

swinging type of temperature is of great importance in the diagnosis of tuberculosis; it is very constant, and even in those cases in which the maximum temperature is never more than 99° the temperature will generally show a daily variation of more than one degree, and so conform to type.

It is important that the temperature should be taken every four hours, or even more often, as if the temperature be taken only in the morning or evening the characteristic variation may not be so apparent. A persistent rise of temperature after exertion is of especial value both in diagnosis and in gauging the effect of treatment.

The pulse is usually accelerated in proportion to the temperature—it is often full and soft; except in advancing disease, it is usually below ninety. A persistent rise in frequency after exercise, even though unaccompanied by fever, is a sign that the patient has done too much.

The blood pressure is nearly always subnormal.

(3) Physical Signs. *Inspection* of the chest may reveal a long flat narrow thorax with wide intercostal space and narrow costal angle. The supra- and infra-clavicular fossæ are often well marked, while the scapulæ may be prominent or winged. Asymmetry of the upper chest, particularly in the direction of flattening or retraction at one apex, is important; impaired mobility of one side, again particularly the upper part, is often of early occurrence. Lastly, displacement of the cardiac impulse may result from the heart being pulled in the direction of a fibrosed area.

Palpation with the flat of the hand will show impaired mobility, and comparison of the vocal fremitus in different regions will show an increase in the vicinity of the affected parts. It must be remembered that vocal fremitus is normally better appreciated at the right apex than the left, but any marked difference is of significance.

Percussion may give a relatively dull note either from diminished air entry, consolidation, thickened pleura, or a cavity filled with secretion. Valuable information may be gained by direct percussion on the clavicles. A tympanitic note may be obtained where there is central cavitation covered by crepitant lung, or over an empty cavity if near the surface. A cavity communicating with an open bronchus may give a "crack-pot" sound if the patient has his mouth open at the moment of percussion, but it should be remembered that this sound may often be obtained in healthy children if they are crying. In early cases the percussion signs may be most indefinite, and it is then that significance must be attached to any *relative* difference on the two sides of the chest. It is important to remember that normally the percussion note is rather higher pitched at the right apex than at the left.

Auscultation. Diminished air-entry, and especially a prolongation of expiration with a feeble or nearly inaudible inspiration, are amongst the earliest signs. As the disease becomes more advanced adventitious sounds may appear. A localised fine-tube bronchitis is significant. When softening has occurred there are likely to be fine, hard

crepitations, often possessed of a metallic character; these are best heard during and at the end of inspiration. They may often be heard as a shower of harsh clicks accompanying inspiration if the patient has just previously been made to cough. Persistence or increase after coughing of harsh crackles over a localised area of lung is perhaps the most important diagnostic point, and such crackles are termed post-tussive. Consolidation, if at all extensive, will cause broncho-vesicular or tubular breathing, while amphoric phenomena will be obtained over an empty cavity of sufficient size.

Vocal resonance is likely to be increased throughout the diseased area, while bronchophony and pectoriloquy are usual over both solid lung and cavities.

Pleurisy is the rule at some stage or other, so that a respiratory rub may be audible at any period of the disease; it is often one of the earliest phenomena.

In many cases the physical signs as above described will be so obvious as to make the diagnosis a matter of no difficulty; in other cases, however, a very careful examination will be needed before the affected area can be discovered. In such cases it is of special importance to remember the *most likely situations* for the presence of early lesions and carefully to contrast suspected spots with the corresponding areas of the other lung. The apical lesions are most readily detected in the supra-spinous fossæ behind, and just below the middle of the clavicle in front. Below the outer third of the clavicle in front is another likely situation. Lesions starting in the upper part of the lower lobes can best be detected by examining at the level of the fourth and fifth dorsal spines behind and about two inches from the middle line. The interlobar septum must also be examined; the line for this can be judged by placing the patient's hand on his other shoulder and examining along the lower border of the scapula.

It remains to be said that sometimes the most painstaking examination may reveal no trace of the disease; for such cases certain special methods of diagnosis must be employed (*vide* pp. 105—110).

(*d*) **Fibroid Tuberculosis.** A fibroid lung is not neces-

sarily tuberculous, but chronic cases of fibro-caseous disease often become fibroid. Fibrosis may also follow tuberculous pleurisy and the pneumonic forms of pulmonary tuberculosis should the patient survive. On the other hand, tuberculosis may occur as a secondary infection in a lung fibrosed from other causes, such as non-tuberculous pleurisy, with or without effusion, unresolved lobar pneumonia, chronic broncho-pneumonia, and the late stages of the various forms of pneumono-koniosis.

The diagnosis of a fibroid lung is easy, but it is only possible to say whether it is fibroid tuberculosis by demonstrating tubercle bacilli in the sputum. They are generally present if there is active tuberculosis, but sometimes the tuberculous factor may become obliterated as the fibrosis

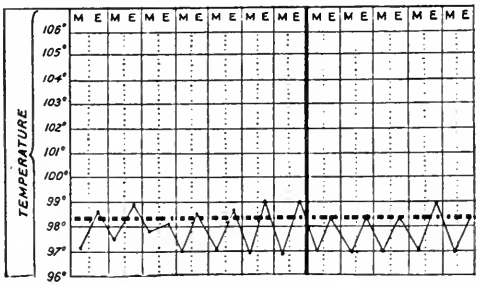


FIG. 27.—Chart to show the sub-normal temperature seen in many cases of Fibroid Tuberculosis.

advances. At the same time it must be remembered that tuberculosis is the commonest cause of fibroid lung.

The symptoms of fibroid tuberculosis are cough, which is often paroxysmal, and increasing dyspnœa on exertion. The sputum is purulent and varies in amount according to the degree of co-existent bronchiectasis. Hæmoptysis is common. There is very gradual loss of weight and strength; pyrexia is usually absent.

The physical signs are shrinking and retraction of the affected side with marked impairment of mobility; there is dulness to percussion, but tactile fremitus and vocal resonance are usually increased. The heart is displaced towards the lesion, and bronchial or tubular breathing is often heard, especially at the apex. (See also "Lungs, Fibrosis of," p. 294.)

IV. TUBERCULOSIS OF THE ALIMENTARY TRACT.

It is probable that the frequency with which the cervical lymph glands become affected with tuberculosis is due to the passage of tubercle bacilli through the tonsils. The bacilli can be demonstrated in a fair proportion of all tonsils removed for chronic hypertrophy, even though they have not formed a local lesion. Sometimes, however, it happens that a local deposit is formed, in which case caseation and ulceration result.

The tongue, pharynx, and palate may occasionally be affected in the late stages of pulmonary tuberculosis. A ragged spreading ulcer with a rough and sometimes caseous base is formed. The presence of signs of tuberculosis elsewhere will suggest the diagnosis. If there is any doubt a small piece of the ulcer may be excised and examined histologically, films may be made from scrapings and stained for the bacilli, or guinea-pigs may be inoculated with an emulsion in saline of material scraped from the floor of the ulcer.

The intestine may be infected secondarily from the sputum of patients with pulmonary tuberculosis; it may also be infected primarily in children. The parts commonly affected are the Peyer's patches in the lower three feet of the ileum and the solitary follicles in the cæcum and in the first part of the colon.

The symptoms of intestinal tuberculosis are very variable; diarrhœa is common, sometimes there is melaena, and profuse hæmorrhages are occasionally seen.

If the disease is localised to the cæcum the symptoms may simulate appendicitis. In an adult the presence of pulmonary tuberculosis might suggest the correct diagnosis. Where there are no signs of tuberculosis elsewhere a blood examination may prove of value, a lymphocytosis, relative or absolute, being a common accompaniment of tuberculous lesions. The special methods for the diagnosis of tuberculosis (p. 105) should also be employed. Perforation or peritonitis, either local or diffuse, are possible sequelæ.

Fistula *in ano* and chronic ischio-rectal abscess are frequently tuberculous in origin.

V. TUBERCULOSIS OF SEROUS MEMBRANES.

Pleurisy due to tuberculous infection may be primary or secondary; miliary tubercles are often found on the pleura without any signs of pleurisy in cases of generalised tuberculosis. In secondary cases there may be the physical signs of the underlying pulmonary condition.

The symptoms and physical signs of a tuberculous pleurisy do not differ from those of pleurisy produced from other causes (*vide* p. 304). Tuberculous pleurisy may be dry or sero-fibrinous; in secondary cases it may occasionally be purulent.

The diagnosis of tuberculous from other pleurisies will dépend upon the evidence of tuberculosis elsewhere, the special methods for diagnosing tuberculosis (p. 105), and, if there is effusion, on the examination of the fluid withdrawn by an exploring syringe or by aspiration. The fluid so obtained should be placed in a sterile bottle; it is usually clear or slightly turbid and of a pronounced yellow colour; it is highly albuminous. The fluid should be centrifugalised and cultures made from the deposit on various nutrient media; films should also be spread for immediate examination, and it may be desirable to inoculate some of the deposit into the peritoneum of a guinea-pig.

The film preparations will show a considerable number of lymphocytes, a variable number of endothelial cells from the pleura, and a complete absence of all organisms. In very rare cases tubercle bacilli may be found. The culture-tubes will yield no growth, but the guinea-pig will generally be found to be tuberculous if killed after an interval of six weeks. It is important to remember that apparently primary pleurisies with the characters above described, even though the inoculation test be negative, must be regarded as tuberculous. The truth of this is emphasised by the frequency with which patients presenting themselves with signs of early pulmonary tuberculosis will give a history of pleurisy some few years previously.

The peritoneum is commonly affected, primarily in children *viâ* the intestinal canal, and secondarily in adults through the swallowing of tuberculous sputum or possibly by the spread of infection through the Fallopian tubes or epididymes and seminal vesicles. In primary cases the mesenteric glands are always involved as well. Two varieties are described, one in which ascites is a prominent feature and the other in which there is a prominent doughy abdomen, often with palpable masses of omentum or glands, and little if any effusion.

The symptoms are extremely variable—indeed, they may be entirely wanting; in like manner great variations in the acuteness of the disease may be encountered. In the more severe cases there will be general ill-health with gradual wasting, abdominal pain and tenderness, diarrhœa, and a swinging temperature. Examination will reveal a prominent and distended abdomen, sometimes with effusion, but more commonly of a doughy or lumpy consistency. The disease is often very chronic, and in such cases a persistently subnormal temperature may be found. At the same time the temperature is likely to show a definite daily variation. In children the diagnosis does not as a rule present much difficulty: enteric fever can be distinguished by the Widal reaction; cirrhosis of the liver is rare in children, as also are cancerous and other tumours in the abdomen. Cœliac disease must always be excluded (*vide* p. 366).

In adults the presence of other tuberculous lesions is of extreme importance. The history and general appearance may suggest cirrhosis. In cases where there is a palpable tumour there may be evidence of a primary growth in the stomach or pelvis. Where there is a sacculated effusion in tuberculous peritonitis the diagnosis from ovarian cyst may be impossible; the irregular temperature, progressive emaciation, and gastro-intestinal disturbance would be in favour of tuberculosis, while a careful bimanual examination might help to establish the diagnosis of ovarian cyst.

Acute septic peritonitis is usually accompanied by constipation, vomiting, and a pulse more rapid and thread-like than that of tuberculous peritonitis; further, the abdomen is likely to be rigid, fixed and not moving with respiration.

At the same time tuberculous peritonitis *may* be very acute ; it is almost the only condition which ever produces a red, fluctuating swelling pointing at the umbilicus though obviously of intra-abdominal origin. In doubtful cases valuable information may be gained from the special tests discussed on p. 105.

VI. TUBERCULOSIS OF THE LYMPHATIC GLANDS.

The younger the patient the more liability is there to tuberculous adenitis. The most common glands to be affected are those in the anterior cervical triangles, the mesenteric and the bronchial glands, but the axillary and inguinal may also be involved. The process is often very indolent, the glands remaining permanently enlarged to a greater or less extent and very subject to periodical exacerbations. There is a tendency for neighbouring glands to become fused together into a nodular mass ; whilst sooner or later softening is apt to occur and give rise to fluctuation. Eventually there is ulceration, and in the case of subcutaneous glands tuberculous sinuses are formed, while the bronchial glands may ulcerate into a bronchus or a blood-vessel, producing tuberculous broncho-pneumonia or generalised tuberculosis respectively. *The mesenteric glands* do not commonly break down ; they may be met with as the sole infection or in conjunction with tuberculous peritonitis. In the former case there may be no symptoms or there may be evidence of obscure toxæmia ; sometimes there are definite abdominal symptoms, such as diarrhœa and distension, and in such cases the condition may be confounded with enteric fever (*vide* p. 23). The glands are rarely palpable, but sometimes they may form a definite tumour. Tuberculosis of the *bronchial glands* may never be suspected ; it may, however, give rise to general symptoms of pyrexia, debility, and wasting, and if the glands become very large they may obstruct the bronchi or implicate the recurrent laryngeal nerve, giving rise to spasmodic cough, stridor, bronchitis, collapse of lung, and laryngeal palsy. Sometimes they may

be detected by means of an unwonted dulness to percussion over the upper part of the sternum and in the absence of physical signs they may sometimes be recognised as dark shadows in X-ray photographs of the thorax.

Tuberculosis of the *cutaneous* lymph glands must not be mistaken for the following conditions :—

(1) *Septic Adenitis.* The presence of a septic focus, such as tonsillitis, pediculosis capitis, or sores about the hands or feet, etc., should prevent this mistake so far as the corresponding glands are concerned.

(2) *Lymphadenoma* (*vide* p. 169).

(3) *Lymphatic Leukæmia.* A systematic examination of the blood will establish the diagnosis of this condition (*vide* p. 165).

(4) *Lymphosarcoma.* The blood picture here may possibly approximate to that of leukæmia ; softening is extremely rare, and there is often evidence that the mediastinal glands are involved. If there is any doubt a gland should be exeised and submitted to microscopical examination.

(5) *Secondary Carcinomatous Adenitis.* Careful examination should reveal a primary growth.

It is to be noted that glandular tuberculosis is the most favourable form of tuberculous infection. Many cases undergo spontaneous cure even after a long period.

VII. TUBERCULOSIS OF THE URINARY SYSTEM.

(*a*) **Renal Tuberculosis.** The kidney may theoretically be infected in one of several manners :—

(1) By the blood-stream, in which case the bacilli are deposited either in the walls of the blood-vessels, or they may escape from the blood-vessels through the glomerular tuft into the convoluted tubules, whence they may be excreted or they may lodge in the pyramids.

(2) By an ascending infection from the bladder along the ureter to the pelvis. It is probable that this method is rare.

(3) By extension from the lower urinary tract along the lymphatics.

It is now generally accepted that the first, or hæmatogenous, route is by far the most common. We are not now concerned with the deposit of miliary tubercles in the course of a generalised tuberculosis, but rather with those cases in which the lesion in the kidney, at first at any rate, is localised.

Diagnosis. The condition is likely to be met with in young adults. The earliest *symptoms* will be polyuria with frequency of micturition, which at first is painless, slight albuminuria with the presence of a few pus cells but no casts in acid urine and occasional hæmaturia with the passage of small clots of blood. It is significant that the above symptoms are often remittent for a considerable time.

In more advanced cases there may be pain in the loin with an enlarged tender and palpable kidney and a certain amount of dysuria, at the same time as pyrexia, wasting and general ill-health. As a first step towards establishing the diagnosis the urine must be tested for the presence of tubercle bacilli (*vide* p. 81). If no bacilli are found, it is advisable to inoculate a guinea-pig with some of the centrifugalised urinary deposit. At the same time assistance may be gained from von Pirquet's test and the estimation of the opsonic index (*vide* pp. 107—108). The cystoscope should now be employed. In skilful hands a positive diagnosis can be made with certainty at a very early stage by the use of this instrument.

The cystoscope will show the presence of tuberculosis of the bladder or of the ureteric orifices; and, further, it is possible to estimate the health of the corresponding kidney by the appearance of each ureteric opening and the size, regularity, and frequency of the jets of urine which escape from it. The quality of the urine produced by each kidney can be observed, and if necessary the urines can be collected separately by means of ureteric catheterisation. An abnormal ureteric orifice, particularly in the direction of congestion, enlargement or erosion, practically always means an abnormal kidney on the affected side. A normal ureter shoots into the bladder a jet of from 10 to 15 drops every thirty to forty seconds. If the kidney is irritated, the urine escapes more frequently and in less amount; if, however, the kidney is compensating by over-activity for its diseased fellow, the jets are frequent, prolonged and

copious. Proof positive may often be furnished by the sight of pus or blood escaping from one ureter.

(*b*) **Tuberculosis of the Bladder.** This is practically never primary, it may be secondary on the one hand to renal tuberculosis, or on the other hand to tuberculosis of the seminal vesicles, which in turn become infected from the epididymis. The symptoms are those of chronic cystitis with pus, albumin, and epithelial cells in acid urine ; there is often but little pain. The discovery of tubercle bacilli and the use of the cystoscope make diagnosis easy. An examination with X-rays should prevent either renal or vesical tubercle being mistaken for calculus in these organs.

It is noteworthy that tuberculosis and the presence of pyelitis or cystitis due to the Bacillus coli communis are the most common causes of pus in acid urine.

VIII. TUBERCULOSIS OF THE SKIN.

Tuberculous skin lesions may arise in three ways—by direct infection through the skin ; by the spread of infection from breaking down of infected lymphatic glands ; and by the circulation of tuberculous toxins generated in some visceral lesion.

Lupus Vulgaris is the most common cutaneous tuberculide. This condition is most frequently met with on the face, often starting in the mucous membrane or skin of the nose. The earliest manifestation is a small raised yellowish-brown translucent nodule ; several of these nodules run together, forming a patch in the centre of which ulceration occurs. The ulcer spreads slowly, healing in one part while extending at another and eroding as it goes all tissues except bone. The disease nearly always starts in childhood or adolescence and runs an extremely chronic course. Serious deformities may be produced by the cicatricial contraction. The appearance of the spreading ulcer with ragged edges, on which the " apple-jelly " nodules are visible, is sufficiently characteristic.

Syphilis may be distinguished by the Wassermann reaction and *Lupus erythematosus* by its late onset, its symmetrical appearance, and the absence of ulceration.

If there is any doubt a small piece of the edge of the ulcer may be excised and the histological characteristics of tuberculosis demonstrated microscopically. This last manœuvre (biopsy) will also serve to distinguish readily lupus vulgaris from rodent ulcer.

Cutaneous Tuberculides due to direct inoculation are usually seen as greyish-white, warty outgrowths; ulceration is rare, but pustules not infrequently form as the result of secondary pyogenic infection. The processes may be single or grouped together, and sometimes spread peripherally while healing at the centre. Microscopical examination after excision will show the characteristic giant-cell systems of tuberculous infiltration.

The Toxic Cutaneous Tuberculide is an important manifestation, since it may sometimes be the first evidence that there is active tuberculosis elsewhere. The lesions are commonly met with on the legs or forearms; they occur at first as painless but sometimes irritable localised red swellings. They are apt to spread until considerable areas are involved, thus causing a slightly-raised, soft, shiny red patch. In the centre of this patch necrosis may occur, causing a punched-out ulcer with a yellowish-grey base; this ulcer may spread till it becomes of considerable size. No tubercle bacilli are present in this last variety, though they can generally be found in lupus and in the tuberculous warts.

IX. SPECIAL METHODS OF USE IN THE DIAGNOSIS OF TUBERCULOSIS.

(1) **X-ray Diagnosis.** This method may be of service in cases of pulmonary tuberculosis and tuberculous enlargement of the thoracic glands, but in no case does it tell us whether the disease is active or quiescent. In all cases screen examinations should first be made and subsequently short-exposure photographs taken of the thorax in different planes. Tuberculous infiltration of the lungs can often be detected as mottled areas of partial opacity either localised or diffuse, according to the distribution of the lesions. Even in very early lesions there is likely to be restricted movement of the

affected lung, and the consequent diminution in the excursion of the diaphragm on that side is well shown by the fluoroscopic screen. Enlarged glands may be seen as dark shadows about the root of the lung and along the course of the main bronchi. An enlarged kidney may occasionally be recognised in a photograph before it can be detected by palpation.

(2) **Diagnosis by the Hypodermic Injection of Koch's Old Tuberculin.** It is preferable to use tuberculin A.F., as this has the same value as old tuberculin, but, being free from albumoses, is less likely to lead to anaphylactic phenomena which may give rise to ambiguous results. A reaction, if it occurs, usually follows an injection in twelve hours, but may be delayed to thirty-six hours. It may be either (*a*) local, *i.e.*, redness and infiltration at the site of injection ; (*b*) general, *i.e.*, malaise, headache, pains in the limbs; (*c*) febrile to the extent of 1° F. or more, or (*d*) focal, *i.e.*, causing changes, chiefly due to hyperæmia in the lesion itself, which in the case of pulmonary disease is shown by an increase of adventitious sounds and an increase in expectoration. Any or all of these reactions may occur in a given patient. As a rule a reaction seldom lasts more than twenty-four hours. A positive reaction shows the presence of a tuberculous lesion, but gives no evidence as to whether the lesion is active or quiescent. At the same time a reaction, and especially a focal reaction, may do much in a given case to strengthen suspicion of the presence of tuberculosis.

The patient should be kept in bed for forty-eight hours before an injection for the purpose of obtaining a four-hourly record of the temperature. The first injection should be ·001 cc. tuberculin A.F. ; if no reaction occur, ·05 cc. may be injected at the end of forty-eight hours, and if no reaction follow now, ·01 cc. may be injected at the end of a further forty-eight hours. If this causes no reaction, tuberculosis can be excluded. In young children or weakly people the first dose should be smaller ; if there is some disturbance after an injection, but not sufficient to be called a reaction, the increase in dosage should be smaller.

The test should be confined to cases in which the evidence

of disease is slight, and should never be employed when fever is present, when signs of extensive disease are to be found, and when the heart and kidneys are not quite sound.

(3) **Von Pirquet's Cutaneous Tuberculin Reaction.** For this purpose a 50 per cent. solution of old tuberculin is made in normal saline : two separate drops of this are placed upon the forearm of the patient ; the skin is now scarified through the drops just as in ordinary calf-lymph vaccination. After three or four minutes a sterile but not antiseptic dressing is applied. At the same time a control observation is made in a precisely similar manner on the other arm, using normal saline instead of the old tuberculin. On both arms there will be seen an immediate traumatic reaction, which consists of a red areola surrounding a red papule. This disappears in about twenty-four hours in negative cases, but if the reaction be positive there is a definite inflammatory reaction in the one arm only which does not start till after a latent period of from three to thirty-six hours. Ordinarily, in a positive reaction, there is a well-marked papule surrounded by a red areola at the end of twenty-four hours ; the reaction reaches its height in about forty-eight hours and then gradually fades.

It has been shown that a positive von Pirquet reaction occurs both in cases of active tuberculosis and also in cases where the lesion is healed ; therefore the reaction need only signify that the patient has had tuberculosis somewhere at some time. The reaction is not obtained in cases of acute generalised tuberculosis, neither is it always present in cases of tuberculous peritonitis. The reaction may also be absent shortly before death.

In conclusion we may say that a positive von Pirquet reaction in itself can never determine the question whether we are dealing with active tuberculosis, but that a negative reaction, as indicating freedom from tuberculosis, may be of the utmost value.

(4) **Calmette's Ophthalmo-Tuberculin Reaction.** Two drops of a ·5 per cent. solution of old tuberculin in distilled water are allowed to fall into the conjunctival sac close to the inner canthus of the eye. The eyelids should be

held open for a few seconds so as to permit of the circulation of the fluid. A positive reaction consists of a distinct conjunctivitis with inflammation of the caruncle. The inflammation reaches its maximum in from twelve to eighteen hours, after which time it gradually subsides and has usually completely disappeared by the third day. In non-tuberculous people there is sometimes a slight reddening of the conjunctiva for two or three hours after the instillation. The test must never be employed unless the eye is absolutely healthy to start with.

The diagnostic scope of the reaction is identical with that of the von Pirquet, but it is scarcely so safe a procedure as this latter.

(5) **Moro's Inunction Tests.** The ointment used consists of equal parts of old tuberculin and anhydrous lanoline. A piece of ointment the size of a pea is rubbed lightly into the skin of the chest or abdomen over an area of a few square inches. A positive reaction is indicated by the appearance on the second day of small red papules at the site of inunction.

(6) **Diagnosis by the Determination of the Opsonic Index.** The researches of Wright and Douglas tend to show that there is circulating in the blood serum a substance called "Opsonin," the function of which is to react upon any micro-organisms that may be present in such a manner as to make them more susceptible to the phagocytic action of the leucocytes. It is probable that there are not an indefinite number of specific opsonins to correspond with every different variety of bacillus, but that there is normally present a single substance which can be activated by different bacilli to form the specific opsonin which is indicated.

The opsonic index is the ratio between the opsonin content of the blood of a suspected person with that of the blood of a healthy person. The required ratio is determined practically by mixing together in a capillary pipette equal volumes of washed leucocytes, emulsion of the required bacilli, and the suspected serum. A similar procedure is adopted in another pipette, substituting healthy for the suspected serum. Both pipettes are now inoculated at 37° C. for fifteen minutes, in order to permit the opsonin present in each tube to sensitise as many bacilli as possible.

Films are then spread in the ordinary manner (*vide* p. 155) and stained by the method appropriate to the micro organism that is being investigated. The films are now examined with an oil immersion lens and the number of bacilli counted which are contained in one hundred leucocytes. This number in the case of the healthy serum is taken as unity ; the opsonic index of the suspected person is therefore the number of bacilli contained in the hundred cells from the pipette containing the suspected serum divided by the number contained in the hundred cells from the control.

It is necessary to allow a considerable margin of error due to the technical difficulties in estimating an opsonic index ; and it is customary to consider any index between ·8 and 1·2 as being normal. A low opsonic index must be considered as suggestive of disease, especially of localised tuberculosis such as lupus or arthritis, but at the same time a persistently high index is quite compatible with an active infection to which there is a pronounced reaction.

For the purposes of diagnosis it is advisable to make at least two determinations of the opsonic index, one before exercise (the amount of which must vary with the condition of the patient) and one immediately after, or in the case of localised lesions, such as joint affections or tuberculous peritonitis, one before massage of the affected parts and one immediately after. It is best to have a third observation, viz., after two hours' complete rest, following immediately on the exercise taken.

If the patient is so ill as to render exercise out of the question, one determination may be made when the temperature is at its maximum and another when it is at its minimum. A wide variation between the two readings obtained as above outlined is very suggestive of a tuberculous infection.

Another method is to take the opsonic index at intervals of three or four hours for a period of two days, making also control observations with a healthy serum. It will be found that there will be very much greater variations in the indices of a tuberculous person than of a healthy one, a fact which can readily be shown by plotting the two series of observations in curve form on the same chart.

(7) **The Therapeutic Method of Diagnosis.** This method is

applicable to those cases in which there are no objective signs of disease, but the patient feels ill, is losing weight, and has a persistent daily rise of temperature in spite of prolonged rest and other forms of treatment.

Therapeutic doses of tuberculin T.R. are administered either by the mouth or by subcutaneous injection. A good initial dose is $\frac{1}{500,000}$ mgm. The dose is gradually increased having due regard to the temperature chart, that dose being desired at each injection which is sufficiently small to avoid a marked negative phase (as shown by a rise in temperature immediately following the injection) and yet large enough to ensure a distinct and prolonged positive phase (as shown by the subsequent flattening and lowering of the temperature). If at the end of some weeks of such treatment the temperature is permanently flat and at the same time the patient feels better and is gaining weight, then assuredly a positive diagnosis of tuberculosis is justifiable.

Summary of the Diagnosis of Tuberculous Disease. When there are obvious physical signs supplemented by the presence of tubercle bacilli the diagnosis is simple. In the absence of tubercle bacilli, as shown by *repeated* examinations, the only certain evidence we have of the presence of *active* tuberculosis is fluctuations of the opsonic index, and as the estimation of this is difficult it cannot be relied upon as a general rule in ordinary practice. Other tests, such as the various tuberculin tests or examination with X-rays, may show evidence of tuberculosis, but give no answer to the question whether it is active or quiescent—that is to say, whether the patient requires treatment. In the great majority of doubtful cases we have evidence of toxæmia such as slight fever, best shown by the rectal temperature, or a raised temperature after exertion which does not return to the normal after half an hour's rest, night sweats, loss of appetite and weight, easy fatigue, and loss of energy, etc. If we are able to exclude toxæmias of other origin, such as occur in pyorrhœa, chronic appendicitis, etc., the probability that we are dealing with early tuberculosis is great, in view of the wide distribution of the disease. A tuberculin test or the use of X-rays may increase our suspicions. The history of the case may do the same—for example, in pulmonary disease a history of

previous pleurisy a few months or years ago or of an attack of hæmoptysis makes the diagnosis in association with the symptoms described almost certain. In many cases it is necessary to keep the patient under observation for some weeks and then come to a conclusion on the basis of clinical observation and laboratory examinations. The diagnosis here cannot always be proved, but is a verdict based on circumstantial evidence.

XXIII. TYPHUS.

Definition. An acute infective disease characterised by sudden onset, macular eruption, and great prostration, and terminating by crisis.

Bacteriology. No specific micro-organism has been identified, but there is evidence to suggest that the contagion is spread through the medium of the pediculus corporis.

Incubation Period. About twelve days.

Course. Occasionally there is slight malaise for a few days, but usually the onset is abrupt, with rigor, headache, and pains in the back and legs. The temperature rises abruptly to 103° or 104°, though it not infrequently attains its maximum on the fourth day. Prostration is early and severe. The pulse is rapid; at first full and bounding, towards the end of the first week it becomes soft and feeble. Persistent vomiting is a distressing feature.

On the fourth or fifth day delirium supervenes, and on the fifth day the characteristic rash appears. This consists of two elements—a dusky red subcuticular mottling and distinct rose-red papules, which soon become the site of petechial hæmorrhages. The rash appears first on the abdomen and upper part of the wrists; the face often escapes, but in the course of two days the rest of the body is covered. During the second week the symptoms are intensified though the headache often disappears.

On or about the fourteenth day the temperature falls by crisis and convalescence ensues.

There is usually a slight leucocytosis.

Complications. The most important complications are broncho-pneumonia, lobar pneumonia and hypostatic congestion of the lungs. Suppression of urine and uræmia

may prove rapidly fatal, while bed-sores and thrombosis, both venous and arterial, are by no means uncommon.

Diagnosis. Typhus fever is now of such rare occurrence in this country that the history of an epidemic or of exposure to infection is of paramount importance. Otherwise the diagnosis must depend on the sudden onset, the rise of the

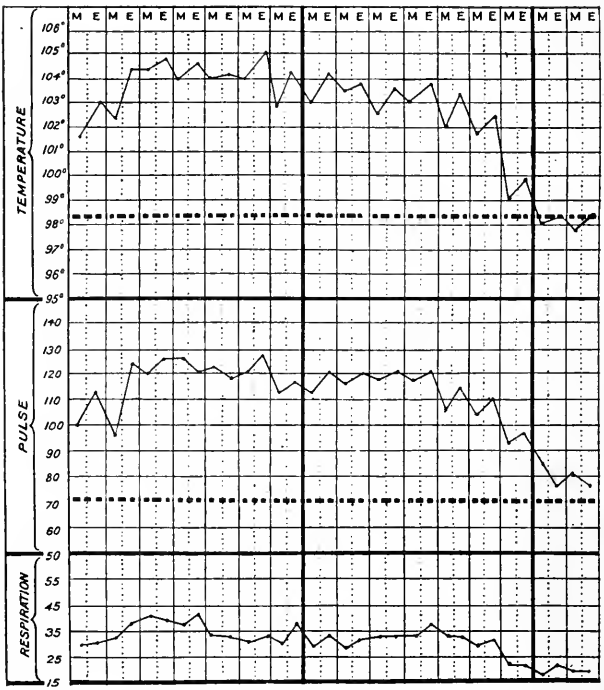


Fig. 28.—Chart from a case of Typhus Fever showing termination by crisis on the fourteenth day.

temperature, the appearance on the fifth day of the characteristic eruption, and the termination by crisis.

Typhus must be differentiated from :—

(i.) *Enteric Fever.* In enteric fever the onset is gradual ; there is often diarrhœa ; the rash appears later (seventh day) and in successive crops, it is limited in distribution, and does not become petechial, whilst subcuticular mottling is very rare. Early prostration is not so marked as in

typhus, whilst the termination is by lysis and not by crisis, and a leucopenia is the rule.

At the end of the first week Widal's reaction will settle the diagnosis.

(ii.) *Measles.* In measles there is well-marked coryza; the rash appears on the fourth day, and is first seen on the face. The temperature begins to fall on the sixth day and the severe prostration of typhus is absent throughout.

(iii.) *Small-pox.* Malignant small-pox, or purpura variolosa, may resemble severe typhus, but the greater extent of the hæmorrhages and the severe bleeding from mucous membranes should prevent mistake for more than a few days.

XXIV. WHOOPING-COUGH.

Definition. A specific infective disease characterised by respiratory catarrh and paroxysms of coughing which terminate in a characteristic whoop.

Bacteriology. The causative organism has only recently been isolated (Bordet).

Incubation Period. Usually from seven to fourteen days.

Course. (i.) *The Catarrhal Stage.* The initial symptoms are usually those of an ordinary cold. Bronchitis is present to a variable extent, but in the majority of cases there is a troublesome and persistent cough. In from ten days to a fortnight the paroxysmal stage is commenced.

(ii.) *The Paroxysmal Stage.* The cough instead of abating becomes more severe and tends to assume a definitely convulsive character. Each paroxysm consists of from fifteen to twenty consecutive short coughs, between which no inspiration is possible; finally, when the child is more or less deeply cyanosed there is a deep inspiration, the noise of which causes the characteristic whoop.

At the end of each paroxysm a small quantity of tenacious mucus is expectorated. Not infrequently there is vomiting.

The paroxysms vary in frequency from two or three to fifty or sixty in the day. The child is conscious that a paroxysm is imminent and displays considerable alarm, usually running to its nurse or mother for support.

In severe cases the strain of the coughing causes puffy swellings to appear about the face and eyes. There is definite leucocytosis (sometimes up to 50,000), with eosinophilia and marked relative lymphocytosis.

In from four to six weeks the paroxysmal stage is over and convalescence is usually rapid.

Complications. (i.) *Hæmorrhage.* There is extreme venous congestion as the result of the coughing. Small subcutaneous hæmorrhages may be met with, particularly about the forehead. Subconjunctival ecchymoses and epistaxis are common. In rare cases sub-dural or intra-cerebral hæmorrhage may occur ; if these latter do not cause death, paralysis, usually a hemiplegia, may result.

(ii.) *Convulsions.* The extreme cerebral congestion may give rise to convulsions. Not infrequently in babies spasm of the glottis may prevent the child making the necessary inspiratory effort at the close of a paroxysm ; it rapidly becomes black in the face, and death will occur if someone is not at hand to plunge it into a warm bath or perform artificial respiration.

(iii.) *Respiratory Complications.* Broncho-pneumonia is a very frequent complication of whooping-cough, and is responsible for a large number of deaths in the first few years of life.

Interstitial emphysema, true pulmonary emphysema, or even pneumo-thorax may result from the strain of coughing.

Acute bronchiectasis (the honeycomb lung of childhood) may complicate or follow whooping-cough. Pulmonary tuberculosis, usually of the broncho-pneumonic type, is a not infrequent sequel.

Enlargement of the bronchial glands may be so great as to give rise to an area of percussion-dulness over the manubrium. In adults whooping-cough is sometimes followed by periodic attacks of bronchial asthma.

(iv.) *Renal Complications* are rare, but the urine is often diminished in amount and may contain albumin, sugar, and excess of acid.

Diagnosis. Though adults are not exempt, whooping-cough is specially a disease of young children. History of exposure to infection is of course important. If the

characteristic whoop is heard, the diagnosis is certain, but the whoop is sometimes absent, more especially in adults. The diagnosis then rests upon the paroxysmal nature of the attacks of coughing, especially if these are followed by vomiting. In such cases a marked lymphocytosis with eosinophilia proves of diagnostic value.

The puffy face, especially if albuminuria is present, may lead to a diagnosis of acute nephritis, but the absence of casts and the presence of a paroxysmal cough should prevent this error.

CHAPTER II

TROPICAL DISEASES

I. BERI-BERI.

Beri-beri is a specific form of polyneuritis in which there is a special tendency for the involvement of the phrenic and vagus nerves, and also for the occurrence of œdema and serous effusions.

The incidence of beri-beri is practically limited to the areas between 45° N. and 35° S. (Manson).

All races seem liable to contract beri-beri, and the sexes are equally affected. Europeans, however, and children generally escape.

The nature of the poison which causes beri-beri is unknown. It can be conveyed by man from place to place, but probably is not infectious from case to case. It has been suggested that beri-beri is contracted by eating rice which has been husked without preliminary steaming, but the evidence in favour of this view is far from conclusive.

The average time at which symptoms appear after commencing to reside in a beri-beri infected building (*e.g.*, a prison) is three to four months.

The first conspicuous symptoms are those of peripheral neuritis (*vide* p. 508), but there are often (though not always) premonitory symptoms for some weeks, such as headache, lassitude, cramp, numbness, and tingling.

The neuritis affects the legs and arms at first, and is soon accompanied by œdema of the ankles, hands and face. Breathlessness, palpitations, sub-sternal oppression and tachycardia indicate involvement of the vagus nerve. The diaphragm may act feebly or not at all if the phrenic is affected. After several weeks the œdema disappears, and it is then obvious that extreme wasting has occurred. Convalescence is very protracted and tedious. Relapses are not uncommon.

All grades of severity may occur. In the worst forms a bilateral vagus neuritis may cause death in a few hours; in the milder form there is only slight loss of power and trifling œdema of the shins.

There is never any mental impairment.

Two main classes are distinguished clinically :—

(i.) *The dry* or *atrophic form*, in which the neuritic atrophy is very pronounced and the œdematous symptoms are relatively slight.

(ii.) *The wet* or *œdematous form*, in which the atrophy is relatively slight or is masked by the very prominent œdema, with which are associated effusions into the serous membranes.

The mortality is considerably higher in the wet form than in the dry, and also varies with different epidemics. It is highest at the beginning of an epidemic, and in all is less than 10 per cent.

The diagnosis depends on the association of peripheral neuritis with œdema. There can be little difficulty in the case of an epidemic, which could only be simulated by chronic arsenical poisoning from adulterated food or drink. The characteristic cutaneous and gastro-intestinal symptoms of chronic arsenical poisoning are not seen in beri-beri.

Isolated cases of beri-beri may present considerable difficulty in diagnosis if they are of the dry form. The type of neuritis may exactly simulate the ataxic form of alcoholic neuritis, but there is no mental change.

Reliance must be placed on the possible history of alcohol and evidence of the vagus or phrenic nerves being involved. Œdema, to a greater extent than a slight puffiness of the shins, is in favour of beri-beri.

II. CHOLERA.

A specific infective disease characterised by vomiting, purging, muscular cramps, and suppression of urine. Cholera is endemic in certain parts of Asia, but is also liable to occur in widespread epidemics.

Bacteriology. The bacillus of cholera was discovered by Koch in 1883. It is a small, comma-shaped vibrio, sometimes assuming a complete spirillum form. It is highly

motile, flagellated, and does not form spores. It grows well on ordinary media; on gelatine plates at 22° C. small yellowish-white highly refractile colonies with scalloped edges develop in twenty-one hours. Eventually the gelatine liquefies and the colonies sink into its substance. In dilute peptone the vibrios grow on the surface as a pellicle. Preferably a faintly-alkaline medium should be used for the cultivation of the cholera bacillus.

The vibrio stains well with ordinary dyes (dilute carbol-fuchsin is the best) and is Gram-negative. It possesses the property of becoming agglutinated by a specific cholera serum. Agglutination within twenty minutes by a serum diluted 500 times with normal saline is considered positive. This test is performed in a converse manner to the Widal reaction (cf. p. 20)—that is to say, peptone cultures are made from the dejecta of a suspected patient and a fragment of a colony obtained by sub-culture is added to the diluted *specific cholera serum*, which only agglutinates true cholera vibrios.

Fig. 29.—Spirillum of Cholera from Agar culture, 48 hours. Magnification × 1000.

In the very great majority of cases infection occurs through water which is used for cooking or drinking or washing utensils and which has been contaminated by dejecta from cholera patients.

As far as is known the infective material must be swallowed.

Incubation Period. The incubation period of cholera is probably two to five days.

Course. Individual cases vary greatly in severity as do different epidemics. The following account may be regarded as an average case. Four stages are recognised :—

(i.) *Premonitory Stage.* This is by no means always

present, but, if it occurs, consists in gastro-intestinal disturbance with diarrhœa of varying severity. It lasts for a few hours or even several days.

(ii.) *The Evacuation Stage.* This may be the first symptom, and is characterised by violent purging, with the passage of copious watery stools (rice-water evacuations), vomiting, cramps in the calves and the abdominal muscles, and exhaustion. There is no tenesmus. Both the vomited matter and the stools may be flecked with blood in the later stages. The cutaneous temperature becomes subnormal as this stage proceeds, but the rectal temperature may show high fever.

The average duration of the evacuation stage is three to twelve hours.

(iii.) *The Collapse Stage.* The surface is cold and clammy; the eyes are sunken; the voice husky or even lost; the pulse feeble and rapid; and the urine scanty, albuminous, or suppressed. The cramps may be prolonged into this stage, but usually cease, as do the profuse evacuations. This stage lasts from three to forty-eight hours.

(iv.) *The Stage of Reaction.* In this there is a gradual restoration of the functions, the pulse returns at the wrist, the skin becomes warm, and urine is once more secreted and the motions resume their fæculent character. In favourable cases, beyond weakness, there may be no adverse symptoms after three days.

In a proportion of cases, however, the reactionary stage only proceeds a short way and the fatal tepid phase is manifested, in which the temperature remains subnormal, vomiting and purging continue; the patient remains semi-comatose, and dies of exhaustion in a few hours.

The total mortality of cholera is usually given as 50 per cent.

The following atypical forms of cholera may be mentioned:

(*a*) An ambulatory form, in which the disease is aborted in the premonitory stage.

(*b*) A form in which the second stage is very slight and the entire trouble is over in twenty-four hours (cholerine).

(*c*) Very occasionally the stage of collapse is reached without the preliminary vomiting, purging or cramps. These

cases present very great difficulties in diagnosis, but fortunately are extremely rare.

(*d*) Some cases show uræmic signs from prolonged suppression of urine ; a fatal issue is to be apprehended in this form.

(*e*) A considerable number of cases end fatally from profound toxæmia in a few hours, even before the evacuation stage has properly developed.

The diagnosis of cholera depends upon a history of possible infection, such as the presence of a local epidemic, etc., upon the characteristic features of the disease as described above, and upon the discovery of the specific organism in the stools.

The identity of the vibrio is established by cultural characteristics aided by the agglutination test as described above.

Cholera may be simulated to some extent by food-poisoning (especially mushroom poisoning), by malarial diarrhœa, and possibly by very acute bacillary dysentery.

Malaria may be distinguished by its reaction to quinine and by the examination of the blood for the parasites and for leucocytosis. (There is a leucocytosis in cholera, up to 30,000 to 50,000, but none in malaria ; further, in cholera there is polycythæmia).

Dysentery does not really resemble cholera ; it may be distinguished by the character of the motions, the passage of blood, the tenesmus, the griping pains, and the absence of muscular cramps.

Food poisoning may be suspected from the history of a limited number of persons only being affected, all of whom have eaten some common dish, and by the absence of the cholera vibrio in the dejecta.

III. DYSENTERY.

Definition. An ulcerative infection of the intestinal mucous membrane, affecting, as a rule, the lower part of the large gut, but often involving the whole colon and the cæcum and sometimes the small gut also.

The ulcers start in the lymph nodules ; they spread in a most irregular manner, depending somewhat on the severity

of the infection, and are often connected with each other by sub-mucous tracks.

Bacteriology. Two forms are recognised—first the bacillary type, which is caused by Shiga's Bacillus dysentericus, and secondly the amœbic type, which depends on the presence of the Amœba dysenteriæ (Entamœba histolytica).

Shiga's bacillus resembles the Bacillus typhosus rather closely, except that it is not so slender, is very slightly if at all motile, and does not form spores. It stains well with all ordinary stains and is Gram-negative. It grows fairly readily on ordinary media, and can be separated from other organisms if a plate culture is made from the suspected dejecta and subcultures made from those colonies which appear *later than* twelve hours. It does not ferment sugars, neither does it form gas in the usual media.

Bacillary dysentery can be produced by organisms similar to but not identical with Shiga's bacillus. Individual epidemics are probably due to one particular organism.

The serum of dysenteric patients agglutinates the *appropriate bacillus* in dilutions of 1 in 50 or less.

Bacillary dysentery is the epidemic disease which afflicts armies, caravans, pilgrimages, and lunatic asylums. Sporadic cases, however, are not infrequent. Relapses are uncommon, and chronic dysentery is comparatively seldom of this type.

Amœbic dysentery is endemic in many parts of the East (such as the Philippine Islands); it may cause illness almost if not quite as severe as the bacillary form, but tends rather to produce the chronic and relapsing variety; indeed, a fair proportion of the cases are sub-acute or chronic from the start.

The amœba of dysentery is a protozoon about 30 μ in diameter; it consists of ectosarc, endosarc, and granular protoplasm, in which red blood-cells may be visible. The ectosarc is highly refractile. There is a nucleus some 5 or 6 μ in diameter, but this is difficult to see while the creature is alive. The amœba should be searched for *in fresh dejecta*, and in fragments of mucus, not in portions of fæcal matter. The slide must be examined on a warm stage

in a wet preparation, and should not be identified unless definite pseudopodial action can be observed.

Certain amœbæ may be present in any person's colon, especially after a dose of magnesium sulphate. These amœbæ coli are not pathogenic; they are smaller, less refactile, less vacuolated, and less active than the Entamœba histolytica. Nevertheless, considerable experience is necessary to differentiate quickly and with certainty the simple from the pathogenic forms.

The symptoms of acute dysentery, whether bacillary or amœbic, are as follows :—

Violent purging, with the passage of loose motions every few minutes till all fæcal matter is expelled, and then the equally frequent evacuation of blood and mucus, usually in small quantities at a time, but accompanied by severe tenesmus and also griping pains in the abdomen, due to painful contractions and spasm of the colon.

Vomiting is generally absent. Thirst is excessive and the other signs of collapse are marked.

There is usually great tenderness to palpation along the course of the colon.

Fever is usually present in the bacillary form, but not in the amœbic. The onset in the epidemic form may be alarmingly sudden, but in the milder varieties the disease is commonly preceded by what seems at first to be a mild diarrhœa.

The symptoms of chronic dysentery are identical with those of ulcerative colitis (*vide* p. 367), from which it can only be distinguished by a previous history of dysentery or the recovery of the amœba (or rarely the bacillus) from the stools.

The occurrence of a liver abscess is proof of *amœbic* dysentery.

The diagnosis of acute dysentery is usually easy. Residence in the tropics or under conditions favourable to an outbreak are important points.

The recovery of the specific organism is conclusive, as is a positive agglutination test.

Enteric fever has a gradual onset (though it may be relatively quite sudden in the tropics); there is no tenesmus and blood is not passed for many days. The Widal

reaction and the cultivation of the bacillus from the blood are conclusive.

Cholera has copious watery motions, cramps, and vomiting, but neither tenesmus nor the passage of blood. The onset is likely to be even more sudden than in acute dysentery.

Malaria may produce a variety of clinical dysentery, but the malaria parasite can be demonstrated in the blood of patients with active malaria, though, if a patient with active malaria contracted dysentery at the same time, the diagnosis might depend on the agglutination test or the presence of the dysentery organism. At the same time, for malaria to simulate dysentery is relatively uncommon.

IV. KALA-AZAR (Dum-Dum Fever).

Kala-Azar is a febrile disease characterised by progressive enlargement of the spleen, extreme emaciation, and a tendency to dropsical effusions.

The cause is believed to be one of the "flagellata," the Leishman-Donovan body, which can be recovered from the spleen of patients and also from the blood and other organs; they are minute ovoid bodies (2·5 μ in diameter) with vacuolated protoplasm and two distinct nuclear masses. When cultivated in vitro these bodies can be seen to develop in six days into typical flagellates.

Kala-azar is most prevalent in Assam, but also occurs in China, India, Burma, Egypt, and Algiers. It can be communicated by human intercourse, but how this occurs is not known, though it is suggested that the bed-bug may be the medium.

Clinical Features. Kala-azar is a very chronic disease, and its earliest manifestations are not fully described. Probably the first sign is fever, which may be either intermittent or remittent, and which lasts from two to six weeks. During this fever the spleen and liver enlarge.

After a period of remission there is another febrile attack, and after this a shorter remission, till at last there is continuous pyrexia of remittent type.

After the first attack the enlargement of the spleen and liver disappears, but after several bouts of fever the enlargement is permanent.

The period of continued fever lasts about nine or ten months and is succeeded by a phase of cachexia, often with a subnormal temperature and dropsical effusions, which continues until death takes place, usually from some intercurrent malady.

Some cases terminate fatally in a few months, others last for about two years. The mortality is over 90 per cent.

An important feature of kala-azar is the tendency for hæmorrhages from mucous surfaces and under the skin.

The blood picture is striking; there is a slight anæmia, with a colour index rather below unity, and also there is a very marked leucopenia (2,000 or less), with which, however a relative lymphocytosis can be recognised.

The diagnosis depends upon the history of the febrile attacks, with hæmorrhages and transient œdemas as described above, the large spleen and liver, the characteristic blood picture, the absence of the *malaria* parasite from the blood, and the demonstration of the Leishman-Donovan bodies in the material derived from splenic or hepatic puncture.

Leishman lays stress on the importance of slightly stirring up the splenic tissue with the needle point to break up some of the cells and permit the escape of the parasite into the blood before withdrawing it into the syringe; the same authority considers that hepatic puncture is preferable to splenic puncture as being less dangerous.

V. LEPROSY.

Leprosy is a disease characterised by the formation of granulomatous masses in the skin, mucous membranes, and nerves. It is a specific infection and is due to the Bacillus lepræ. It may be met with all over the world, but is especially prevalent in Asia. It flourished throughout Europe in the Middle Ages.

Bacteriology. The bacillus of leprosy closely resembles the tubercle bacillus in appearance and in staining reactions; it has not been cultivated outside the body, and recent claims that this has been accomplished still lack confirmation.

The organism is present in large numbers in the skin of leprous nodules and in the discharges from ulcerative lesions.

Leprosy is presumed to be contagious, but very prolonged contact and a suitable "soil" are probably necessary.

Artificial inoculation experiments have scarcely ever been unequivocally successful, and the lower animals appear to be immune.

Incubation Period. The incubation period is necessarily very uncertain; many years may elapse after leaving a leprosy district before the disease becomes apparent.

Course. The patient is liable to vague pains and indefinite febrile attacks for several months, or even years, before any objective signs appear.

Clinically the earliest signs are a succession of erythematous spots, which are often pigmented and are particularly prone to appear on the ears, nose, forehead, forearms, and the backs of the hands; at first these patches are hyperæsthetic and are transient, but later some of them tend to become permanent anæsthetic macules. Some of these lose their pigment and become peculiarly white and shiny.

The next phenomenon is usually the development of granulomatous nodules in the skin, mucous membranes, and nerves. These tend to appear first in the regions where the macular eruption has been; later ulceration may occur, with considerable tissue loss.

The last stage may be divided clinically into three groups;

(i.) *The nodular variety* (which, indeed, may develop without any preceding macular stage), in which hard, flat, subcutaneous nodules appear in the skin, especially about the face, ears, and forearms. This form causes the "leonine" aspect said to be characteristic of leprosy.

(ii.) *The anæsthetic variety* is produced by an extensive peripheral neuritis, due to the local action of the bacilli on the nerves. It is often, but not always, preceded by a macular eruption, but this is often best marked on the abdomen, back and buttocks, and is not so characteristically distributed over the face and ears as in the nodular form. Anæsthesia, muscular atrophy, bullæ, ulceration, gangrene and contractions are the features of this variety.

The median, ulnar, radial, anterior tibial, and peroneal nerves are the first to be affected, and are thickened and in early stages tender to the touch.

(iii.) *The mixed variety* is a combination of the other two forms; it is probable that most cases are in reality mixed, though in any given case one type may largely predominate.

The diagnosis may be difficult in the macular stage, but the tendency to central anæsthesia in the patches and the absence of perspiration over them when pilocarpine has been injected, as well as their characteristic distribution, should prevent mistake.

In the later forms it is only necessary to excise a portion of a nodule or a fragment of nerve in an anæsthetic patch and to stain sections of the material by the Ziehl-Neelson stain as for tubercle bacilli, when clumps of red, beaded bacilli can be shown lying between the cells of the granulation tissue. The purely anæsthetic type may present resemblances to syringomyelia (especially Morvan's type), but the history of preceding macular eruptions and the thickening of the nerves, as well as the type and distribution of the sensory disturbance, should suggest the diagnosis.

VI. MALARIA.

Malaria is a disease characterised by the occurrence of febrile attacks which may be periodic, irregular, or continuous, according to the variety of the causative Plasmodium. In all cases the Plasmodium enters the blood-stream of the patient as the result of the bite of a mosquito (Culex Anopheles). Only the female mosquito conveys malaria, and she only bites at night.

Varieties. Three types of malaria are recognised :—

(i.) *Tertian fever*, caused by the Plasmodium vivax, and characterised by the onset of fever every forty-eight hours.

(ii.) *Quartan fever*, caused by the Plasmodium malariæ, and characterised by the onset of fever every seventy-two hours.

(iii.) *Æstivo-autumnal fever*, caused by the Laverania malariæ, and characterised by the onset of fever at irregular intervals.

Parasitology. (i.) *The Tertian parasite* first occurs in the red blood corpuscles as a small colourless body of indefinite

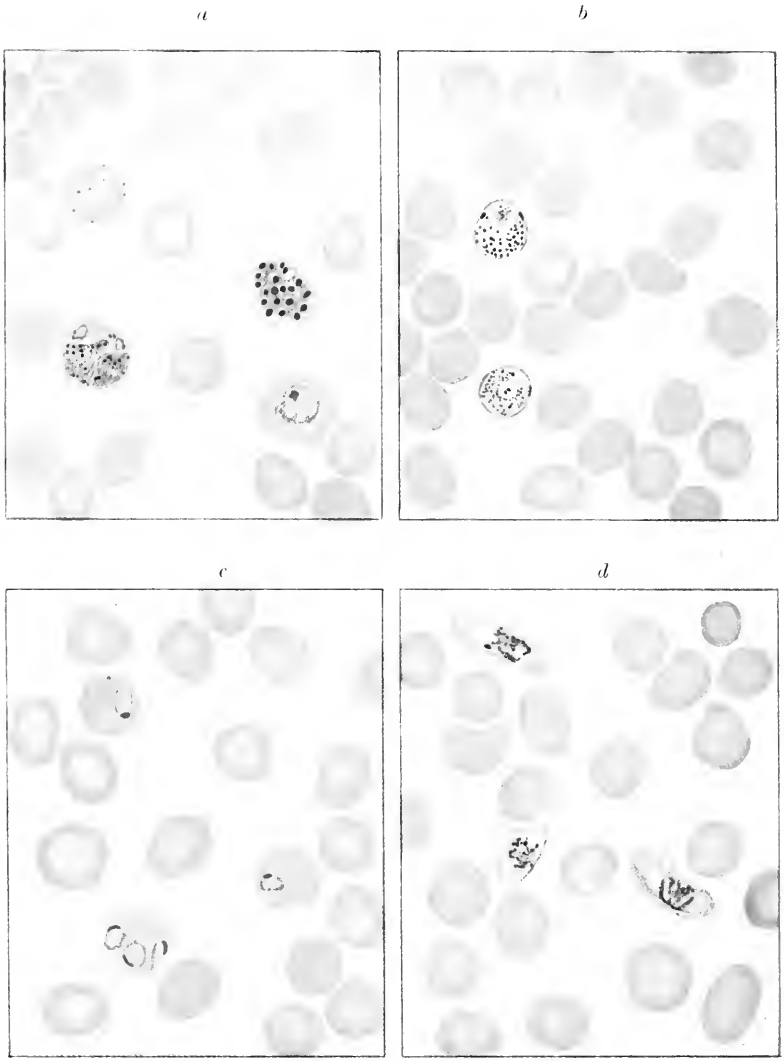


FIG. 30. Blood Films showing Malarial Parasites.

(*a*) Benign Tertian
(*b*) Quartan.
(*c*) Ring-Forms in "Malignant" Tertian Fever.
(*d*) Crescents in "Malignant" Tertian Fever.

shape. In a few hours it increases in size and develops granular pigment. In twenty-four hours it fills the corpuscle, which is swollen and bloated. This parasite now becomes either a sporocyte or a gametocyte. The sporocytes undergo segmentation and divide by radial lines into from fifteen to thirty spores. The corpuscle now bursts and the spores are liberated into the circulation. The liberated spores attack fresh red corpuscles, and the same asexual cycle is gone through again and again.

A feature of the development of those young spores which are *not* going to assume a sexual form is the stage of vacuolation, which causes a characteristic appearance like a signet-ring. The gametocytes differentiate into male and female forms, but cannot undergo sexual reproduction until they have been withdrawn from the human host by a mosquito.

In the stomach of the mosquito the male and female gametocytes escape from the red blood corpuscles, and the male gametocyte detaches from itself flagella-like microgametes which enter into and fertilise the female gametocyte, which is now called a zygote.

The zygote becomes capsulated, penetrates the wall of the mosquito's stomach, and discharges large numbers of thin spindle-shaped sporozoites into the blood-stream of the mosquito. These sporozoites reach the salivary glands of the insect and are injected into the blood of the next person bitten by the mosquito.

The asexual cycle in the human blood-stream takes *forty-eight* hours, and the disruption of the red cells, with the liberation of swarms of spores, coincides with the occurrence of a febrile paroxysm.

(ii.) The life history and general features of the *Quartan parasite* are similar to those of the tertian parasite, with the exception that it has a more definite outline and coarser and blacker pigment, causes the red corpuscles to shrink rather than to swell, has an asexual phase of *seventy-two* hours, and only forms from six to twelve spores.

(iii.) *The Æstivo-autumnal parasite* is much smaller and less pigmented than the other forms. The later stages of the asexual phase cannot be observed in the peripheral blood-stream, since they are carried out in the blood of

the spleen and bone marrow. After about six days, however, sexual forms appear in the peripheral circulation; these are crescentic or sausage-shaped bodies with pigment granules; they cannot sporulate in their human host, but readily do so on reaching the stomach of the mosquito.

Clinical Features of Malaria. (i.) The Tertian Form. *The incubation period*—that is to say, the interval between infection and a febrile paroxysm—is about fourteen days. This depends on the number of sporozoites injected, since it will take a considerable time for sufficient red cells to become infected to discharge enough spores into the circulation to produce a definite reactionary fever. There may be slight general malaise for two to three days before a paroxysm.

The febrile attacks last about eleven hours and are divided into three stages:—

(*a*) *The Cold Stage.* This is accompanied by headache, lassitude, giddiness, yawning, and sometimes sickness; after a few premonitory chills a severe rigor sets in, which lasts up to an hour.

(*b*) *The Hot Stage.* The temperature rises to from 103° to 105° F. The patient feels very hot and complains bitterly of headache, backache, giddiness, and often of tinnitus. Delirium is not infrequent. The spleen may enlarge rapidly. The hot stage lasts four or five hours.

(*c*) *The Sweating Stage.* The onset of profuse perspiration brings relief of the symptoms; the temperature falls steadily and is subnormal in about four hours.

In forty-eight hours from the onset of the cold stage the entire process is gone through again: the patient feels quite well in the interval.

If, as is usually the case, there is a double infection with asexual forms maturing at different times, there will be a daily paroxysm; careful analysis of the temperature chart will show that there is forty-eight hours interval between the *alternate* paroxysms, and examination of the blood shows two distinct generations of parasites at different stages of development. In a similar way multiple tertian infections can be diagnosed.

(ii.) The Quartan Form. This is much rarer than the

tertian. The paroxysms occur every seventy-two hours, unless there is a double infection, when there is a paroxysm for two successive days and then a free day. Of course a triple quartan infection gives a paroxysm every day. The individual paroxysms are in all respects similar to those described as occurring in the tertian form.

Repeated attacks of tertian or quartan fever, if untreated, may result in grave cachexia; as a rule, however, spontaneous recovery takes place.

(iii.) The Æstivo-Autumnal Form. This variety is especially met with in the real tropics. The tendency is for a continuous remittent fever to develop, which may be explained by the prevalence of multiple infections and also by the arrangement of the parasites in groups with a long period of segmentation.

The simple cycle of asexual development of the æstivo-autumnal parasite is forty-eight hours; hence a "malignant" tertian fever is not uncommon.

An important point is the great length of the individual paroxysms (when such can be recognised), often up to twenty-five or twenty-six hours. The initial chill is not so common as in the other forms, but the general constitutional symptoms are very severe, and many cases show a marked similarity to enteric fever in their general appearance.

If æstivo-autumnal malaria is neglected very grave symptoms (so-called pernicious) are likely to develop. For example, the patient may become comatose with slight jaundice and small punctate hæmorrhages, or meningitis may be simulated (especially in children), or gastro-intestinal disturbance may be so profound as to suggest cholera.

Those who have had the largest experience of malaria agree that there are few diseases that may not be imitated by the pernicious forms of the Æstivo-autumnal disease.

The Diagnosis of malaria depends upon the following points:—

(i.) The history of previous attacks or of exposure to infection. Recrudescences (especially of the tertian fever) may occur more than a year after removal from a malarious district.

(ii.) The absence of leucocytosis and the relative increase in large mononuclear cells.

(iii.) The character of the febrile paroxysms (in tertian or quartan fever).

(iv.) The most important diagnostic point is, beyond doubt the discovery of the parasite or its products in the blood. In tertian and quartan fevers the parasites are less abundant in the peripheral blood-stream just before and during a paroxysm. They may be detected in a fresh preparation or in stained films.

(*a*) To make a fresh preparation prick the patient's finger and place a *small* drop of blood on a clean slide; drop a thin cover-slip on to the blood and allow this to spread out by capillary attraction under the weight of the cover-slip; finally ring the cover slip with vaseline.

A $\frac{1}{12}$ inch oil immersion lens is used for the examination.

The parasites appear as indistinct, irregular bodies in the centre of the red blood corpuscles.

Tertian parasites *show active amœboid movements;* quartan parasites are more sluggish. Except when they are very young, both forms show pigment granules around their periphery or collected at their centres in the later stages.

In tertian cases the affected blood-cells are large and pale; in quartan cases they tend to be small and of rather deeper colour than those which do not contain a parasite.

The quartan parasite is more highly refractile than the tertian.

If the blood is taken shortly before a paroxysm is due, segmentation by radial lines can be perceived in most of the parasites.

(*b*) *To Stain Films for Malaria.* It is essential to spread a very thin film, which may be stained by Giemsa's or Leishman's method (*vide* p. 155), Jenner's method, carbol-thionin, etc., as preferred. Giemsa's and Leishman's methods stain the sporocyte blue with red spots of chromatin; fully developed spores are stained blue with a red centre. In cases of doubt the essential things to look for are the *red* spots of chromatin in the erythrocytes.

In æstivo-autumnal fever ring-shaped bodies in

shrunken, crenated corpuscles can be seen in the blood taken at the height of a paroxysm. Segmenting bodies are rare in the peripheral circulation, but can be seen in great numbers if splenic puncture is performed.

During the remissions small hyaline pigmented forms may be present in fair numbers. After from five to fourteen days the characteristic crescent-shaped bodies with centrally arranged coarse pigment granules can usually be recognised with ease.

An important point is that even if there are no parasites to be discovered in the blood about the time of the paroxysm, nevertheless leucocytes containing masses of pigment can generally be seen.

Jaundice, anæmia, and an enlarged spleen are all points in favour of æstivo-autumnal malarial infection.

VII. BLACKWATER FEVER.

This disease is characterised by febrile paroxysms and the passage of hæmoglobin in the urine.

Blackwater fever only occurs in patients who have had malaria, and nearly always in those who have had repeated attacks.

Experimentally quinine may induce hæmoglobinuria and clinically cases of malaria have been noted in which hæmoglobinuria coincided with the administration of quinine ; on the other hand, there is no doubt that blackwater fever *may* occasionally occur apart from the taking of quinine. Probably the combination of *malaria and quinine* is responsible for the very great majority of all cases of blackwater fever.

Although it sometimes happens that the passage of blackish urine is the first manifestation of the disease, as a general rule there is an accompanying febrile attack not unlike those seen in malaria. There is pain in the loins, spleen and liver, jaundice, and often severe bilious vomiting.

The fever lasts a variable time, and as it subsides the urine resumes its natural colour.

Relapses may occur, and some of the more serious cases progress with no intermission till a fatal result ensues from exhaustion and collapse.

VIII. MALTA FEVER (Mediterranean Fever).

Malta fever is a specific infective disease, endemic to the shores of the Mediterranean Sea, and characterised by pyrexia, perspirations, constipation, swelling of the joints, enlargement of the spleen, and a tendency to relapses.

Bacteriology. The specific micro-organism, the Micrococcus melitensis, was discovered by Bruce in 1887, and is a small coccus often arranged in pairs and tending to grow in short chains when cultivated in broth. A bacillary form is sometimes met with.

The micrococcus stains well with ordinary dyes and is agglutinated by the serum of Malta fever patients.

The organism is conveyed to man in the milk of infected goats in the very great majority of cases, but indirect contagion by fomites is certainly theoretically possible.

Incubation Period. The incubation period is about fifteen days (six to twenty days).

Course. The invasion may be sudden or gradual. The early symptoms are those of general constitutional disturbance, especially fever, headache, backache, and dyspepsia. Vomiting or rigors are rare at the outset, but nausea and vomiting may occur after a few days. The general debility increases, the spleen becomes tender and enlarged, the tongue is coated and furred, and constipation is common. Bronchitis and even pneumonia are not infrequent. These symptoms continue for from one to three weeks, the temperature remaining elevated all the time, and then for a few days there is very great amelioration, and the patient appears to be on the way to convalescence. After two or three days of well-being the symptoms all recur again ; the spleen becomes especially enlarged and anæmia is striking. In this stage constipation may give way to diarrhœa, sometimes with blood-stained motions. Orchitis, effusions into various joints, or even endocarditis, may occur in the more severe cases. Several such relapses may occur, and the total duration of the illness varies from three weeks to several months. The mortality is not more than two per cent., but during the illness the prostration, apathy, and pallor

indicate the severity of the toxæmia. There is no leucocytosis in Malta fever.

The Diagnosis depends on the locality in which the case occurs, the type of fever, with relapses and splenic enlargement, the absence of leucocytosis, and finally on the agglutination power of the patient's blood serum.

IX. PLAGUE.

Plague is a specific infective disease produced by the Bacillus pestis. It occurs in epidemics, is infectious from case to case, but is largely conveyed by a flea which ordinarily infests rats, but which may spread from them to human beings. In many eastern cities plague is now endemic.

Bacteriology. The plague bacillus as seen in the blood or buboes of infected persons is very polymorphous; the most usual form is a short oval rod, but long rods and oval involution forms are also seen. The organism grows well on ordinary culture media; its optimum temperature for growth is low, being only 30° C. It stains well with carbol-thionin or dilute fuchsin, and shows marked polar staining. It is Gram-negative.

Incubation Period. The incubation period is from three to ten days.

Varieties. Clinically four forms may be recognised :—

(i.) Mild Bubonic Plague (Pestis Minor).

(ii.) Severe Bubonic Plague (Pestis Major).

(iii.) Pneumonic Plague.

(iv.) Septicæmic Plague.

Clinical Features. (i.) Pestis Minor is never fatal; it may, however, occur in epidemic form shortly before an epidemic of Pestis major and is characterised by mild fever and the occurrence of glandular swellings, which, however, do not often suppurate.

(ii.) Pestis Major. (*a*) *Prodromal Stage.* Headache, giddiness, staggering gait, mental apathy, and often bilious vomiting, or sometimes hæmatemesis. This stage lasts up to twenty-four hours, and towards the end the temperature rises steadily.

(*b*) *The Febrile Stage.* A severe rigor is followed by a

temperature of 103° to 106° F., which reaches its maximum on the second or third day. Prostration is extreme; delirium may occur, but lethargy, or even coma, is more common. A loss of power of articulation is said by Dr. Jennings to be very constant and of great diagnostic importance. After about five days the temperature falls, often almost by crisis.

The characteristic buboes develop on the second, third, or fourth day of fever and occur in nearly 80 per cent. of cases. The inguinal or femoral glands are the most constantly involved, and after these the axillary. Internal glands are found to be affected in fatal cases. The probable cause of the glandular enlargement is the dissemination of the bacilli throughout the body tissues.

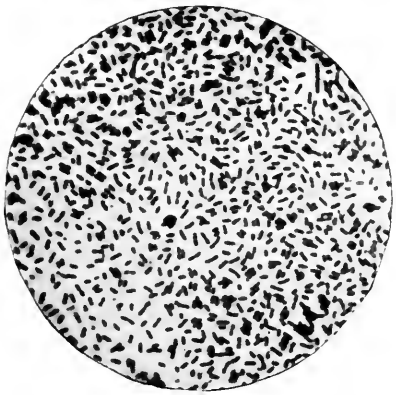


Fig. 31.—Bacillus Pestis from Agar culture, 48 hours. Magnification × 1000.

Suppuration of the buboes is a late sign and is often of favourable significance. Hæmorrhages into the skin and from mucous surfaces are not uncommon. There is an extreme leucocytosis, often up to 90,000 or even higher.

Very severe cases may end fatally within twenty-four hours—that is to say, before any buboes have developed. The total mortality varies with different epidemics and at different stages of the same epidemic, but all in all, probably averages fifty to sixty per cent.

(iii.) Pneumonic Plague. This must not be confounded with secondary broncho-pneumonia, which is a frequent complication of pestis major.

The first sign of pneumonic plague is usually a rigor; the general symptoms of pestis major then occur, but in addition there is cough, blood-stained sputum, and pleuritic pain. There is no primary bubo formation.

Physical examination soon shows a lobar type of pneu-

monic consolidation, the sputum swarms with plague bacilli, and death occurs from heart failure within a week. This form is virulently infectious and tends to reproduce the same variety of disease.

(iv.) Septicæmic Plague. This can hardly be distinguished from the very rapidly fatal form of pestis major mentioned above, in which death occurs within three days and in which no buboes have time to develop. There are, however, large numbers of circulating bacilli in the blood-stream.

The diagnosis of plague presents little difficulty in the presence of an epidemic or if the characteristic buboes are present. The bacilli can easily be demonstrated in the material removed from a bubo or in the sputum of the pneumonic form; but it is not so easy to find them in the blood-stream, except in the septicæmic type, and for this purpose it is advisable to perform a blood-culture experiment as described on p. 157.

If bacteriological methods are not available and there are no glandular enlargements, reliance must be placed on the sudden onset, the injected conjunctiva, the coated tongue with a red margin, and the extreme prostration, as well as the peculiar mental condition of apathy and unwillingness or inability to answer questions.

X. SLEEPING SICKNESS.

Sleeping sickness is an endemic disease limited to certain parts of Africa in the neighbourhood of the Equator, and characterised by increasing mental and physical lethargy, a hectic temperature, and a fatal issue.

The cause of sleeping sickness is a protozoon of the variety known as a Trypanosome, which is conveyed to human beings and also to animals by the bite of the tsetse fly (Glossina palpalis). The pathological lesions, according to Mott, are chronic meningo-encephalitis and meningo-myelitis.

Clinical Features. The onset is very insidious, and the prodronal stage may last from several months up to two years. The earliest symptoms are a change in mental attitude, disinclination to do things, and a tendency to

drowsiness. The face may become a little puffy and the expression is distinctly apathetic. The gait is shuffling, as if it was too much effort to raise the feet, and a tremor of the tongue can often be observed. The most important diagnostic point at this stage is a rise of temperature each evening up to 101° or 102° F. and a corresponding increase in the pulse-rate up to 130 per minute. There is usually a diffuse glandular enlargement.

As the disease progresses drowsiness, apathy, and muscular weakness become more marked; the tremors increase and involve the hands and feet; the skin becomes dry and coarse, and death is preceded by a state of complete coma.

In acute cases death may take place in six weeks from the case coming under observation; in chronic cases the duration is much longer, and temporary remissions are common.

The diagnosis depends upon the presence of signs as described above added to a history of residence in an area where the disease is endemic.

The Trypanosomes can be demonstrated in the cerebro-spinal fluid, in the blood, and especially in the material removed by puncture of an enlarged gland. For this purpose films stained by Leishman's stain are very satisfactory.

The discovery of these protozoa serves definitely to exclude cerebral syphilis or general paralysis, unless of course, there is a double infection.

XI. SPRUE (Psilosis).

The causation of sprue is unknown, but it is in all probability a specific infective disease.

It affects principally Europeans who reside in the tropics, and is characterised by diarrhœa with the passage of numerous pale, frothy, malodorous stools and by superficial ulceration of the mucous membrane of the mouth and tongue, and probably of portions of the mucosa along the entire alimentary tract, which eventually becomes extensively atrophied.

The result of this inflammation and atrophy is deficient assimilation, so that there is progressive starvation and emaciation.

Sprue is an extremely chronic disease and starts very insidiously; the only symptoms for long periods may be those of flatulent dyspepsia and *irregularity* of the bowels. When the disease is fully developed the chief symptoms are :—

(i.) The passage of copious pale, frothy, drab-coloured offensive motions.

(ii.) Tenderness and soreness of the tongue, cheeks and fauces, produced by loss of epithelium and the formation of vesicles or even small superficial grey ulcers.

(iii.) Flatulence.

(iv.) Wasting.

(v.) Anæmia.

(vi.) Cutaneous pigmentation.

Unless efficient treatment is employed the disease is very slowly but steadily progressive, with various brief remissions and relapses till death takes place.

The diagnosis of sprue rests upon a history of residence in the tropics, an insidious diarrhœic condition, with *typical stools as described above* associated with a sore, ulcerated mouth, anæmia, wasting, and pigmentation.

XII. YELLOW FEVER.

A specific infective disease characterised by fever, jaundice, black vomit, suppression of urine, and derangement of the nervous system.

Yellow fever is endemic in the West Indies, part of the West Coast of Africa, and in Brazil.

The specific micro-organism has not been discovered, but it has been conclusively proved that the disease is conveyed to man by the bite of the tiger mosquito (Stegomyia fasciata). The virus is only present in the blood of a patient for the first three days of the disease. A mosquito fed on yellow fever blood cannot communicate the disease for twelve to fourteen days. The infected mosquito may live one hundred to one hundred and fifty days, and may communicate the disease all this time; it is also probable that the offspring of an infected mosquito may also be infectious, though how far this goes is as yet uncertain.

Incubation Period. The incubation period of yellow fever is usually two to five days.

Course. The onset is sudden: the first symptom is a chill or rigor, but this may be absent; headache, pains in the limbs, pallor of the skin and fever next appear. The temperature rapidly rises to from 103° to 106° F., the face becomes red and swollen, the eyes watery, and the pains in the loins and limbs increase.

In twenty-four to thirty-six hours the maximum of the fever and constitutional symptoms is reached and the urine becomes albuminous, scanty, or even suppressed. On the third day the temperature falls and the symptoms abate, and some cases proceed to steady convalescence. About the commencement of this remission yellowing of the conjunctivæ can be noticed.

After a quiescent interval of forty-eight hours in many cases the gastric symptoms return and the patient becomes diffusely jaundiced. The vomiting is persistent, and instead of clear or bilious fluid the vomited material becomes black from altered blood. Epistaxis and subcutaneous hæmorrhages are fairly common.

The temperature may rise again or may remain subnormal; the urine once more becomes diminished or suppressed and loaded with albumin.

If the patient has not succumbed from toxæmia and exhaustion the symptoms gradually disappear from the fifth to the seventh day of the relapse. The mental condition is usually dull and apathetic; sometimes the mind remains clear, sometimes there is delirium; nearly always the patient fails to realise how ill he is.

There is a moderate leucopenia in yellow fever (6,000 to 3,000 white cells per cubic mm.) and a slight *relative* increase in the large lymphocytes.

Diagnosis. In typical cases, in an endemic area, or during an epidemic the diagnosis is easy. *Malaria* can be diagnosed by the presence of the parasite in the blood, though, of course, a double infection may exist.

Blackwater fever is proved by the presence of *hæmoglobin* in the water (*hæmaturia* may sometimes happen in yellow fever), and even in the absence of the spectroscope the

porter-colour of the urine, the fact that it settles into two layers, the upper clear and port-wine coloured, and the pink tinge imparted to the froth by shaking will settle the question against yellow fever.

Relapsing fever can be distinguished by the enlarged spleen, the leucocytosis, and the demonstration of the characteristic spirillum in the blood.

For the differential diagnosis of *Weil's disease* from yellow fever see p. 384.

CHAPTER III

CERTAIN ANIMAL PARASITES

For a detailed description of the various parasites which may occur in man reference should be made to text-books of pathology. Only the more common varieties will be described here.

I. **Cestoda** (Tapeworms). The following tapeworms are commonly found in man :—

(i.) Tænia Saginata (the beef worm).

(ii.) Tænia Solium (the pork worm).

(iii.) Bothriocephalus Latus (the fish worm).

(iv.) Tænia Echinococcus (in cysticercus form only).

(i.) *Tænia Saginata.* The intermediate hosts are cattle, which swallow the ova. In the stomach of the ox the envelope of the ovum is dissolved and the embryos burrow through the stomach wall and become encysted in the muscles, with the formation of a fibrous capsule (cysticercus stage). The cysticercus is about 1 cm. in diameter, and if it is not destroyed by the heat of cooking and is swallowed by man the head becomes attached to the mucous membrane of the intestines, the cyst wall is lost, and a tapeworm develops with the following characteristics :—

The head is about 2 mm. in diameter, and is furnished with four suckers but no rostellum or hooklets. The neck is about $\frac{1}{2}$ inch in length.

The fully-developed worm may be 30 feet long and have as many as a thousand segments or proglottides, which increase in size as the distance from the head and neck increases until the full width of about $\frac{1}{2}$ inch is reached.

Physiologically active proglottides are not reached till about the 200th segment, they are bisexual and contain a tubular uterus which branches dichotomously, two ovaries situated near the posterior part of the segment, and testes which are visible as scattered vesicles throughout the

segment. The uterus, the ovaries and the testes communicate with the exterior by a genital pore which is situated on the border of each segment. The proglottides are fully mature about the 600th segment and the uterus is now the most conspicuous feature, the other sexual apparatus having done its work and faded away. About eight of the terminal proglottides are discharged every day loaded with ova.

(ii.) *Tænia Solium.* The life history is the same as for Tænia Saginata ; the intermediate host is the pig.

The head is very small (about the size of a pin's head) and the neck is long (about one inch). The segments are altogether smaller than in Tænia Saginata, and the whole worm is rarely more than 10 feet long.

The head has a beak or rostellum, four suckers, and twenty-six chitinous hooklets, which are arranged in two rows.

The uterus has fewer lateral off-shoots (10 to 12) than Tænia Saginata (20 to 30), and does not branch dichotomously.

The cysticercus stage may occasionally be found in man, causing small cystic swellings in the brain, muscles, or other tissues. When very numerous in the muscles rheumatic pains are complained of, and if in the brain the signs are those of cerebral tumour.

(iii.) *Bothriocephalus Latus.* The intermediate host is a fish, usually the pike. The worm may reach 25 feet in length and have 3,000 to 4,000 segments.

The head has neither suckers nor hooklets, but is furnished with two lateral grooves.

This worm is sometimes met with in England and America, but is most often seen in Switzerland and Northern Russia.

The presence of a tapeworm in man does not often give rise to symptoms. The passage of proglottides per rectum is usually the first sign.

Bothriocephalus Latus may, however, cause a grave anæmia, with a blood picture resembling that of pernicious anæmia. Insomnia, loss of appetite, and vague dyspeptic symptoms are sometimes attributable to tapeworms, and in children convulsions and meningismus may occur, but are not so common as when the parasite is a round worm.

In treating cases of tapeworm the head must be carefully

sought, as unless this is passed the worm is sure to grow again in a few months' time.

Tænia Saginata is by far the most common tapeworm in Britain.

(iv.) *Tænia Echinococcus.* The cysticercus stage is the only one that is found in man ; other and more usual intermediate hosts are sheep, pigs, and cattle. The flesh of these animals is eaten by dogs, wolves, or jackals, and the worm

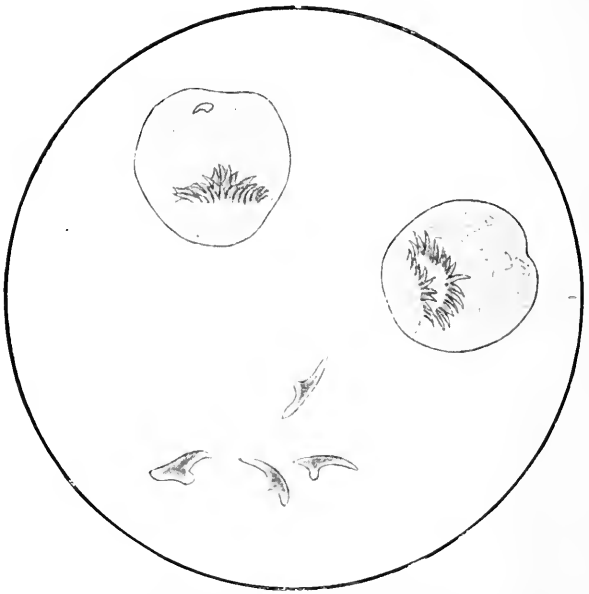


Fig. 32.—Fluid from an Hydatid Cyst, showing—Hooklets, magnification × 400; Scolices, magnification × 260.

develops in them. The eggs are passed daily and lie about on the grass, where they are taken up again by the herbivora. Man may be infected by eating contaminated greenstuff such as watercress, or directly from dogs, as these may carry the ova about their lips and mouths.

The worm is about $\frac{1}{6}$ inch long and consists of a head and three or four segments, the last one only being mature.

The head is provided with four suckers, a rostellum, and a double arch of from 30 to 50 hooklets.

When an ovum is received into the human stomach it

loses its shell and the small six-hooked embryo burrows its way through the intestinal mucous membrane; it often reaches the blood-stream, and may be carried to any part of the body. The liver is the most frequent resting-place; fifty per cent. of hydatid cysts are situated in this organ.

Wherever the embryo is deposited it loses its hooks and becomes converted into a double-walled cyst (hydatid cyst). The outer layer of the cyst is composed of laminated chitinous material; the inner layer is granular and frequently develops secondary daughter cysts, which project into its cavity like buds. In each of these daughter cysts a larval head (scolex) develops. These larvæ also develop directly from the wall of the parent cyst without the formation of daughter cysts, so that a single cyst is responsible for enormous numbers of scolices. It sometimes happens (especially in animals other than man) that the daughter cysts project externally from the mother cyst and in time become quite separated from the parent structure.

The fluid contents of an hydatid cyst consist of a clear non-albuminous fluid in which hooklets or even scolices can usually be found.

Symptoms may be entirely absent; when present they are the same as those of any other tumour in the particular organ involved.

Suppuration in an hydatid cyst gives symptoms of septic absorption.

Rupture of an hydatid cyst is usually fatal, though rupture into a lung or pleura has been survived.

The diagnosis of Hydatid Disease is helped by a history of close association with dogs, residence in Iceland or Australia, *the presence of eosinophilia*, and, in the case of the liver, a smooth, painless enlargement which *may* give a wavy thrill on percussion. If fluid is withdrawn by aspiration the presence of hooklets is pathognomonic.

In cases of doubt the patient's blood serum should be tested with fluid from a cyst for the precipitin reaction, which is positive in a considerable proportion of cases.

II. **Nematodes** (Round Worms). (i.) *Ascaris Lumbricoides*. This parasite inhabits the small intestine as a rule, but may wander into the stomach and be vomited or into the

large intestine and be voided per rectum. It may occasionally reach the bronchus (*via* the larynx), or even the accessory sinuses of the nose ; it may invade the bile ducts and cause jaundice.

There are usually two worms present (male and female), but sometimes larger numbers are found.

Children from three to ten years are mostly affected.

The female worm is about 10 inches long and looks very like an earthworm ; she is pointed at each end and has four longitudinal lines down her sides ; the genital pore is about the middle of the body.

The male is shorter, being about five or six inches long ; the genital pore is situated near the hinder end and is guarded by two chitinous spines.

There is no intermediate host, and the ova are conveyed in contaminated water or by re-infection from scratching the anus.

There may be no symptoms, but, on the other hand, very curious toxic states can be produced by these worms.

General malaise and vague dyspeptic signs are common.

If a worm is in the stomach repeated vomiting may occur until it is vomited or passed out through the pylorus. Anæmia, meningismus, convulsions, giddiness, and irregular pyrexia are not infrequent. Vacancies or faints in young children usually mean round worms or epilepsy.

It is obvious that none of these symptoms are conclusive, but their occurrence should not be forgotten, and a timely examination of the fæces for the ova of these parasites may clear up the diagnosis in a case of anomalous symptoms in a sick child. Of course, the passage of a worm by a rectum or in the vomit is conclusive.

The ova are small, brownish-red oval bodies about ·075 mm. in length ; their shell is thick and rough, and an ill-defined granular embryo can be seen within.

(ii.) *Oxyuris Vermicularis* (*Threadworm*). These parasites infest the whole of the large bowel. They resemble to the naked eye minute threads of cotton. The female is about 10 mm. long and the male rather less than half that length.

During the night the worms tend to migrate, and often emerge at the anus in large numbers.

The only symptoms are anal itching and general irritability. The diagnosis cannot be overlooked if a cursory examination is made of the fæces.

(iii.) *Trichina Spiralis.* The development of trichiniasis depends on the ingestion of raw or undercooked pork, in the muscles of which are deposited the encysted larvæ of the Trichina Spiralis. The parasite is killed at the temperature of boiling water ; hence the disease is most prevalent in Germany, where the habit of eating raw or imperfectly cooked flesh is general.

The embryo is relieved of its cyst in the stomach of the new host and passes into the small intestine, where it reaches its full development in three days. The adult worms produce large numbers of embryos, which about the eighth or ninth day have penetrated the walls of the intestine (unless indeed the female worm herself penetrates the intestine before laying her eggs) and are carried by the blood-stream to the various muscles of the body.

The embryos lodge in the muscle fibres and there become encapsulated. The cyst is about $\frac{3}{4}$ mm. in diameter ; the wall is translucent at first, but later becomes infiltrated with lime salts. The embryo can be seen coiled up in the interior of the cyst. The symptoms coincide with the appearance of the embryo in the muscle fibres and are :—

(*a*) Gastro-intestinal disturbance ;

(*b*) Fever ;

(*c*) Muscular pains, stiffness and tenderness ;

(*d*) High leucocytosis, with marked eosinophilia (50 per cent.) ;

(*e*) Œdema of the face, with perspirations, tingling and itching.

In most cases the symptoms gradually abate and recovery takes place in from two to eight weeks.

The diagnosis depends on the history of the food taken about a week previously, the characteristic muscle symptoms, the œdema of the face, and the eosinophilia.

The adult worms can often be seen as silvery threads in

the fæces if these are examined with a lens against a dark background.

Enteric fever is excluded by the leucocytosis and, later, by the negative Widal reaction ; rheumatism by the absence of joint involvement and the failure to react to salicylates.

(iv.) *Ankylostoma Duodenale* (*Hookworm*). This parasite is prevalent in southern latitudes, especially Egypt, but it also occurs all over the continent of Europe and in England and America ; it has repeatedly caused small epidemics in miners (Miners' anæmia).

The worms live in the duodenum and jejunum ; the female is rather more than $\frac{1}{2}$ inch in length and the male about $\frac{1}{4}$ inch. They have a mouth which is supplied with teeth, by which they fix themselves to the mucous membrane. The ova are discharged in the fæces, and the disease is presumably spread by contaminated water.

The worms suck blood from their host and so cause the anæmia which is the outstanding symptom of their presence. The anæmia is of the chlorotic type. Associated symptoms are diarrhœa, colic, dyspnœa, and œdema.

There is a marked leucocytosis and high eosinophilia.

For diagnostic purposes an anæmia with high leucocytosis and eosinophilia should lead to the examination of the fæces, when hundreds of small oval eggs (·6 mm. by ·4 mm.) can easily be recognised with a lens if the stools are compressed against a black background.

(v.) *Filaria*. The Filaria Bancrofti is the worm the presence of which in the lymphatics is a frequent cause of elephantiasis and chyluria in certain tropical districts. The female is some 3 inches and the male about $1\frac{1}{2}$ inches in length ; both are pale in colour and very slender, so that they resemble white horsehairs in appearance. These adult worms may live for years in the lymphatics of man ; the female is viviparous and discharges enormous numbers of very slender, highly motile filiform embryos, each of which is loosely surrounded by a transparent membranous shell. Each embryo is about $\frac{1}{90}$ inch in length and the same width as an erythrocyte, so that they can circulate freely in all parts of the blood-stream. Clinically they are found in the peripheral circulation only at night, though if the

patient sleeps in the daytime the parasites are then found only in the daytime. The embryos require a sojourn in the blood of a mosquito for full development, and are conveyed back to man when the insect next bites. Those embryos which have passed through a mosquito phase are the only ones to reach maturity in man.

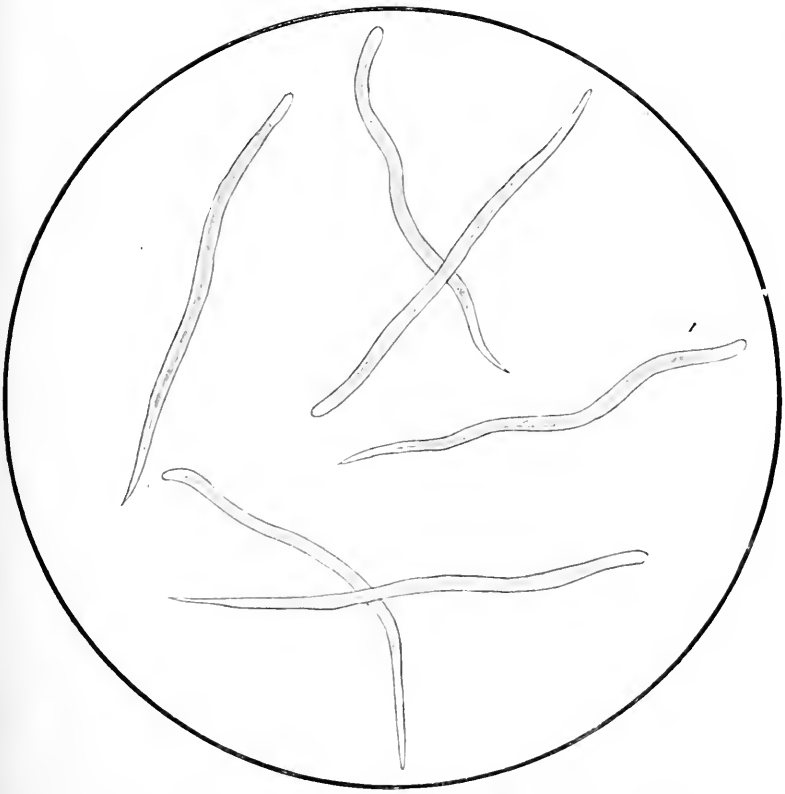


Fig. 33.—Embryos of Filaria Bancrofti. Magnification × 360.

Filariasis may exist without causing symptoms, and when symptoms do occur it is by the more or less accidental blocking of lymphatic channels by the new-laid ova, or possibly by the adult ones.

It has been suggested that elephantiasis only results when the female worm aborts, for the immature embryos are

shorter, thicker, and less motile than the fully-grown embryo and so more likely to block the lymphatic channels.

Clinically filariasis shows itself by enormous distension of the lymphatics distal to the blockage.

The most common clinical manifestation is the lymph-scrotum, which may enlarge so much as to weigh many pounds. The tissues are in a state of brawny œdema, but do not readily pit on pressure. This distinguishes them from ordinary cardiac or renal œdema. The legs may be affected in a similar manner.

If the peri-renal lymphatics are blocked chyluria results; the urine is milky in appearance and contains fat droplets; blood is often present also.

If the subperitoneal lymph plexuses are blocked chylous ascites results.

The diagnosis of filariasis depends on the locality in which the case occurs, the characteristic symptoms, and, above all, the presence of the embryos in the blood-stream at stated intervals. In fresh preparations a low-power magnification is sufficient to show the embryos, which look like snakes wriggling about among the blood-cells. The embryos stain well with dilute fuchsin or eosin, but do not show any very definite internal structure.

Various other forms of filaria have been described in the blood of humans and animals, but it is doubtful if they are pathological.

Since the clinical signs (when present) are purely mechanical, similar signs may be found if the lymphatics are blocked from any other cause, such as chronic lymphangitis, new growth, etc., so that it is necessary to discover the embryo in the blood before making a positive diagnosis of filariasis.

(vi.) *Dracontiasis* (*Filaria Medinensis : the Guinea Worm*). This parasite is prevalent in the East Indies and in parts of Africa. Only the female worm is known: she is some 2 to 3 feet long and about $\frac{1}{12}$ inch in diameter.

The embryos gain access to the human stomach in drinking water incorporated with a "cyclops," which is their intermediate host. The male embryos die; the females burrow through the stomach wall and other tissues until they reach a position in the subcutaneous tissues of the leg. Here the

female completes her growth and eventually discharges her embryos through an ulcer on the surface ; she can be felt underneath the skin like a bunch of string.

III. **Trematodes** (Flukes). Sheep-rot is caused by the *Distoma hepaticum ;* these flukes may very rarely be the cause of cholangitis and jaundice in man.

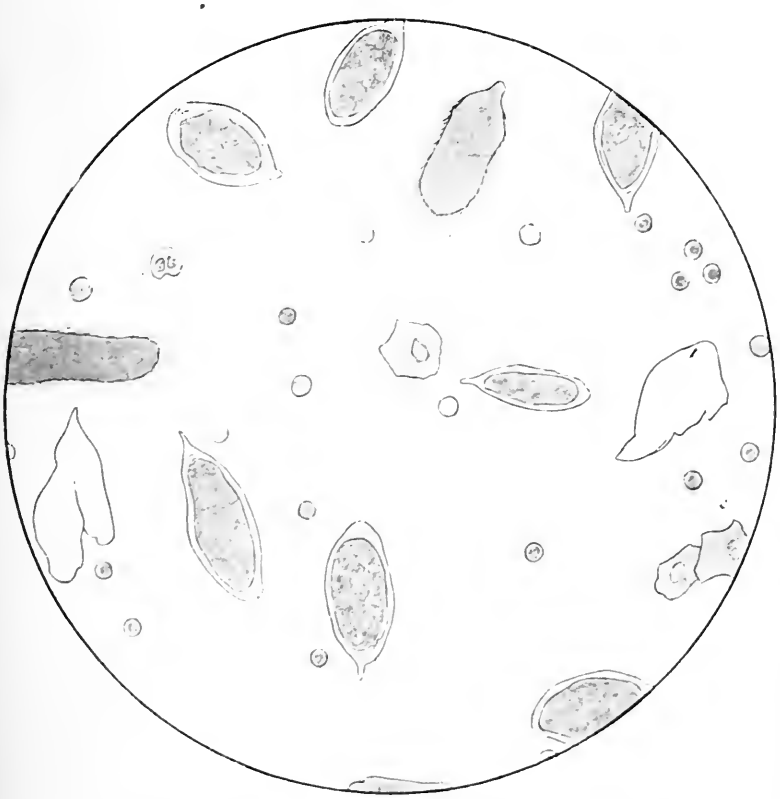


Fig. 34.—Ova and Embryos of Bilharzia from urinary deposit. Magnification × 216.

Distoma pulmonale, a parasite about $\frac{3}{5}$ inch long, inhabits the bronchial tubes and is the cause of endemic hæmoptysis in Corea and Japan. The ova can be recognised in the sputum.

Distoma hæmatobium (Bilharzia) is the most important of the flukes that may cause symptoms in man.

This parasite is endemic in Egypt, North and South Africa, Persia, and parts of India.

The sexes are distinct; the male (about 1 cm. long) is broader and flatter than the female (2 cm. long) and is furnished with a groove which receives the female during coitus. The eggs are oval bodies ·16 mm. by ·06 mm. in size, and one end is furnished with a definite spine. Sometimes the spine is lateral instead of terminal, and it is said that those eggs which are deposited in the rectum have the lateral spine while those that go to the bladder have the terminal spine.

The mode of infection is uncertain, but, since the ova hatch in water, contaminated drinking water is the probable source. The adult worms live in the portal veins, but tend to wander towards the bladder and rectum, in the tissues of which viscera the eggs are laid. The sharp-spined eggs behave like other foreign bodies and tend to cause irritation and hæmorrhage. They may be the starting point of vesical calculi, and may cause cystitis, proctitis, and polypoid masses in either rectum or bladder.

No symptoms may be caused; on the other hand, frequency of micturition, hæmaturia, and cystitis may be found. An aching pain in the perinæum is not uncommon. Tenesmus with the passage of blood and mucus may lead to the discovery of the ova in the rectum. The loss of blood is rarely so great as to cause more than a slight anæmia.

Suspicion of this disease may be aroused if the above symptoms appear in one who has visited the districts above mentioned, but as none of the symptoms are pathognomonic, the diagnosis can only be made when the characteristic spined ova have been discovered in the urine or in the fæces.

1. Primitive Hæmopoietic Cell of the Bone-marrow (never seen in the circulating blood).
2. Promyelocyte or Myeloblast.
3. Neutrophile Myelocyte.
4. Eosinophile Myelocyte.
5. Basophile Myelocyte.
6. Polymorphonuclear Leucocyte.
7. Mast Cell.
8. Eosinophile Leucocyte.
9. Megaloblast and Normoblast.
10. Erythrocytes and Blood Platelets.
11. Large Mononuclear Cell.
12. Large Lymphocyte.
13. Small Lymphocytes.

FIG. 35 — This picture represents all the blood cells (stained by Leishman's stain) which may be met with in health or disease, and also a primitive bone-marrow cell which does not occur in the peripheral circulation but which hypothetically may be the ancestor of all the blood cells, except the lymphocytes which are manufactured in the lymphatic glands.

The large mononuclear, the large and small lymphocytes, and all the cells in the bottom row are met with in normal blood. The remainder are only found in disease.

The normoblast is the parent of the erythrocyte and the megaloblast is the parent of the normoblast.

The three forms of granular cell with their *respective myelocyte parents* are shown on the right of the diagram, and all these three types of myelocyte are descended from the single pre-myelocyte figured above them.

The origin of the large mononuclear cell is uncertain; it is probably of endothelial origin, and it is not thought that it develops into a lymphocyte or any other cell.

1. Primitive Hæmopoietic Cell of the Bone-marrow (never seen in the circulating blood).
2. Pre-myelocyte or Myeloblast.
3. Neutrophile Myelocyte.
4. Basophile Myelocyte.
5. Eosinophile Myelocyte.
6. Polymorphonuclear Leucocyte.
7. Mast Cells.
8. Eosinophile Leucocyte.
9. Megaloblast and Normoblast.
10. Erythrocytes and Blood Platelets.
11. Large Mononuclear Cell.
12. Large Lymphocyte.
13. Small Lymphocytes.

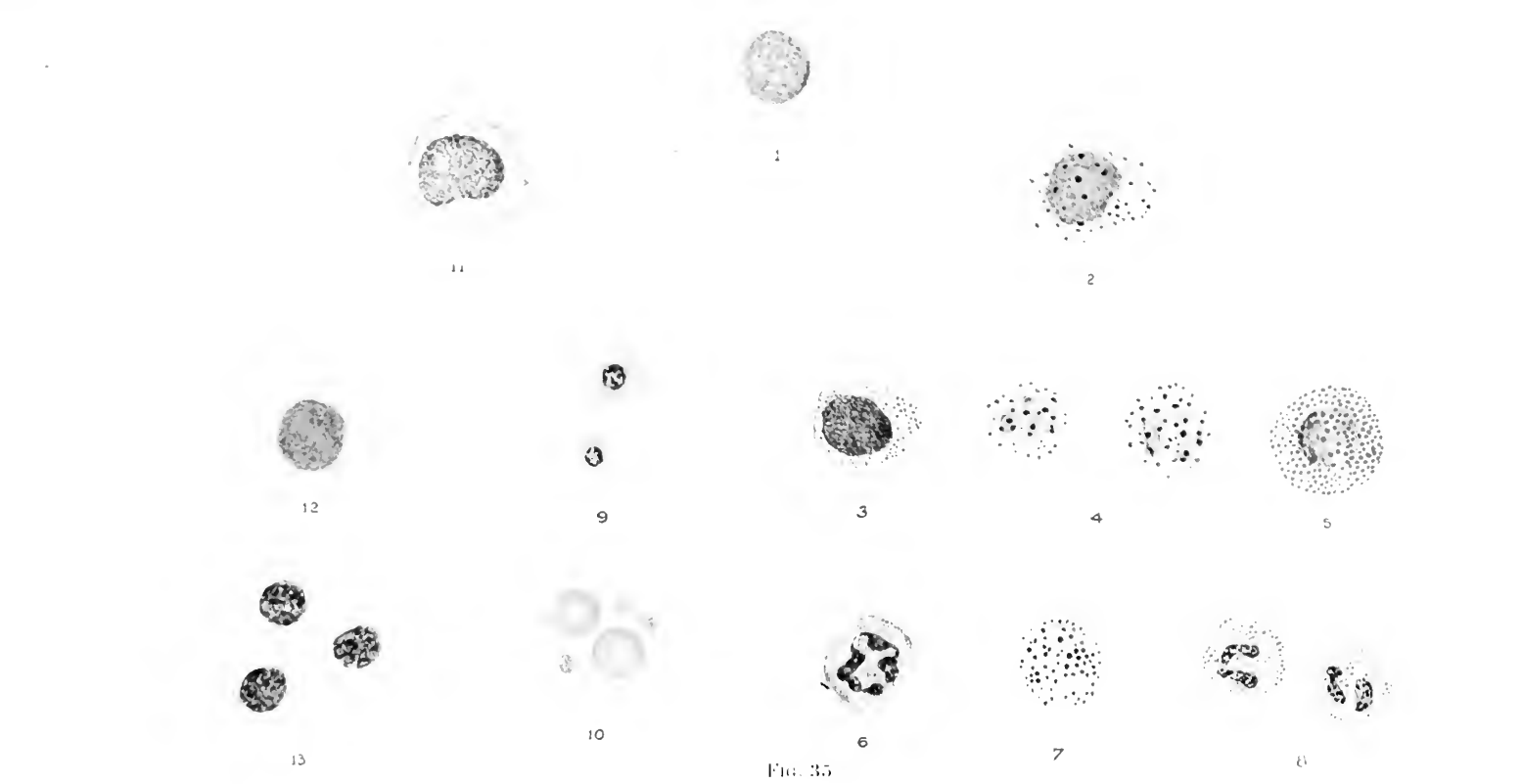


Fig. 35

PART II

CHAPTER I

DISEASES OF THE BLOOD AND BLOOD-FORMING ORGANS

I. EXAMINATION OF THE BLOOD.

The blood consists of a complex solution in which are suspended various cellular bodies. These are divided into two main groups—red and white cells.

The red cells, or erythrocytes, consist of un-nucleated, round or oval, bi-concave discs about 7 μ in diameter. They are manufactured in the red marrow of the bones and are present to the number of 5,000,000 in every cubic millimetre of blood. In women there are rather less, usually about 4,500,000 per cubic millimetre.

The white cells, or leucocytes, are present to the number of 7,800 in every cubic millimetre, and comprise various different cells in healthy blood. The morphological characters described below are those which become evident when Leishman's or Jenner's stains are used (*vide* Fig. 35) :—

A. **Granular cells** which are manufactured in the bone marrow :—

(i.) *Polymorphonuclear leucocytes.* These cells have a multilobular nucleus (three lobes at least can be seen) which stains blue and has colourless surrounding protoplasm in which are numerous fine neutrophile (brick-red) granules (60—75 per cent.).

(ii.) *Eosinophile leucocytes*, which resemble the polymorphonuclear cells except that the nucleus rarely shows more than two lobes and the granules are larger, more rounded, and take on a *bright-red* colour when stained (1—3 per cent.).

(iii.) *Mast cells,* which have a deeply-staining, irregular nucleus and very coarse basophile (blue) granules (1 per cent.).

B. **Non-Granular cells** :—

(i.) *Small or ordinary lymphocytes*, which are manufactured in the germinal centres of the lymph glands. They are rounded cells with a deep-blue circular nucleus which almost fills the cell, leaving only a very small margin of paler blue protoplasm (20—25 per cent.).

(ii.) *Large lymphocytes*, which closely resemble the small lymphocytes, except that they are definitely larger in size (3—6 per cent.).

(iii.) *Large mononuclear cells* (hyaline cells), which are possibly of endothelial origin, which have a definitely phagocytic action, which are the largest cells seen in blood films, and which are recognised by the lighter blue colour of their irregular (often indented) nucleus and the pale blue of the ample surrounding protoplasm (2—4 per cent.).

In addition to red and white cells *blood platelets* are also found. These are small bodies without nuclei, which are seen in clumps in stained blood films and which look like débris. They are present to the number of about 250,000 per cubic millimetre, are notably diminished in purpura, and are probably concerned with the thrombotic properties of blood.

The red colour of the blood is due to the hæmoglobin or oxygen carrier which is contained in the red cells. The *Colour Index* is the ratio between the red cells and their hæmoglobin content $\left(i.e., \frac{\text{amount of hæmoglobin}}{\text{number of red cells}}\right)$. The standard of 100 per cent. represents the amount of hæmoglobin present in the blood-cells of a healthy man. Since there is relatively less hæmoglobin in the blood of a woman, the normal colour index for women is only ·9, whilst for man it is unity.

For a complete blood examination it is necessary :—

(i.) To enumerate the red cells and the white cells.

(ii.) To estimate the relative proportions of the various white cells, any abnormality in the red cells, and to note the presence of any cells which are not found in health.

(iii.) To estimate the colour index.

(iv.) In some cases to perform a blood culture experiment.

(1) **To enumerate the cells** a Thoma-Zeiss hæmocytometer is necessary. This instrument consists of a glass slide in the centre of which is a circular platform surrounded by a moat and depressed below the level of the rest of the slide, so that when covered by a cover-slip a carefully measured distance ($\frac{1}{10}$ mm.) exists between the top of the platform and the bottom of the cover-slip. On the centre of the platform are ruled 400 small squares; they are arranged in groups of sixteen squares, and each square is $\frac{1}{400}$ of a square millimetre. Therefore the space between each square and the cover-slip represents exactly $\frac{1}{4000}$ of a cubic millimetre.

Two graduated pipettes are provided for collecting the blood. The red cell pipette provides for diluting the blood either one hundred or two hundred times. The white cell pipette for dilutions of either ten or twenty times. Toisson's solution * can be used as a diluent for both red and white cells. If preferred Hayem's solution † can be used for the red cells and ·5 per cent. glacial acetic acid tinged with methyl violet for the white cells.

The blood is obtained by pricking the lobe of the ear or the finger at the base of the nail with a needle that is flat or triangular and has a cutting edge as well as a sharp point. The part should be cleaned with warm water and alcohol before puncture, but no pressure should be used to make the blood flow, as this affects the number of white cells.

The red cell pipette is now filled to the mark 5, wiped quickly, and filled to the mark 101 with the diluent. A dilution of 1 in 200 is thus secured. If Toisson's solution is used for the white cell count, the red cell pipette is used

* Toisson's solution is :—

Methyl violet,	·025	parts.
Sod. chloride,	1	,,
Sod. sulphate,	8	,,
Neutral glycerine,	30	,,
Distilled water,	160	,,

† Hayem's solution is :—

Mercuric chloride,	·25	parts.
Sod. chloride,	·5	,,
Sod. sulphate,	2·5	,,
Distilled water,	100	,,

for collecting the blood—that is to say, the same dilution does for both red and white cells. If, however, ·5 per cent. acetic acid is used for the white cells, the larger bore pipette is filled in precisely the same manner with blood up to the 5 mark and with diluent up to the 11 mark, thus giving a dilution of 1 in 20. The pipette must be shaken gently to mix the blood well.

A few drops are now expelled from the pipette to ensure reaching the mixture in the bulb and a small drop is placed on the platform and covered with the cover-slip. This drop must not be so big as to overflow into the moat. It is essential that both counting stage and cover-slip are absolutely clean, dry, and free from dust. If the proper care has been observed, concentric prismatic rings of light are visible on the cover-slip (Newton's rings). A No. 2 eye-piece and a $\frac{1}{6}$ inch objective are used to count the cells.

For the red cells the number is counted in four lots of sixteen small squares. This number divided by 64 gives the number in each small square. If the number in each small square be multiplied by 4,000 the number of cells in a cubic millimetre will be obtained; but the blood has been diluted 200 times, therefore the result must be multiplied by 200 to get the number of cells in a cubic millimetre of undiluted blood.

Thus, if x be the number of cells in each small square, then the number of cells in a cubic millimetre of blood $= x \times 4{,}000 \times 200$.

For the white cells it is best to count in fields instead of squares; for this purpose the draw-tube of the microscope is drawn out until the diameter of the field is exactly eight small squares. Since the area of a circle is πr^2, the area of the field will now equal almost exactly 50 small squares. The number of white cells in 100 consecutive fields should be ascertained. This number divided by 100 and again divided by 50 gives the number of white cells in each small square. If the number so obtained be multiplied by 4,000 and also by the dilution of the blood, the result will be the number of white cells in a cubic millimetre of blood.

Thus, if x = number of white cells in 100 fields, then the

number of white cells in one small square $= \frac{x}{50 \times 100}$, and the number of white cells in a cubic millimetre of blood $= \frac{x \times 4{,}000 \times \text{dilution}}{50 \times 100}$.

The calculation is simplified if Toisson's solution and a dilution of 1 in 100 be used. Under these circumstances the number of white cells per cubic millimetre of blood is obtained by multiplying the total number of leucocytes in 80 *fields* by 100.

The reason why it is necessary to use larger dilutions with Toisson's solution as a diluent is that with small dilutions so many red cells are present that the white cells are concealed. If, however, the acetic acid diluent is used, the red cells are destroyed, so that it is possible to work with much smaller dilutions.

(2) **To estimate the relative number of the different white cells present** it is necessary to examine a stained blood film.

To make a blood film a small drop of blood is placed at one end of a glass slide ; the edge of a *narrower* glass slide is used as a spreader (preferably the spreading edge should be slightly convex). The film is spread with a slightly irregular or jerky movement. When spread the film is allowed to dry in the air.

The best methods of staining the film are those of Jenner, Leishman, or Giemsa, of which the two former involve the use of an alcoholic solution of methylene blue and eosin and are used for fixing the film as well as staining it.

Jenner's Method. Flood the dry but unfixed film with Jenner's stain. After two to three minutes wash off the stain with a brisk stream of distilled water from a wash-bottle ; allow the film to dry in the air, and mount in Canada balsam. It is essential that the stain should not be allowed to dry on the film.

Leishman's Method. Pour 10 drops of Leishman's stain on to the dried but unfixed film and leave for thirty seconds ; add 50 drops of distilled water and leave for five to ten minutes. Wash in distilled water, dry with filtre paper and mount in Canada balsam.

Giemsa's Method. Cover the dried but unfixed film with

absolute alcohol for thirty minutes. Wash off the alcohol with distilled water and dry lightly with filtre paper. Pour on to the film 2 c.c. of distilled water to which two drops of Giemsa's stain have recently been added. Leave for forty minutes and wash off in a brisk stream of distilled water. Dry between filtre paper and mount in Canada balsam.

Each of these methods gives admirable results :—

The red cells are stained pink, nuclei are stained blue, the granules of the polymorphonuclear leucocytes and eosinophiles are stained different shades of red, any basophile granules (*e.g.*, those of mast cells) are stained blue, and the groundwork of the lymphocytes is stained pale blue.

In making a differential count at least 300 white cells must be counted. It is to be noted that they tend to collect at the edge of the film.

Malarial parasites and bacilli are stained blue by these stains.

(3) **To estimate the amount of Hæmoglobin.** Haldane's hæmoglobinometer should be used. This consists of an empty tube with a scale on it and a closed tube of the same size containing a standard solution of carboxy-hæmoglobin corresponding to healthy blood. There is also a pipette with a mark on it to measure 20 cubic millimetres.

A small quantity of distilled water is placed in the empty tube, the pipette is filled to the mark with blood, and the 20 cubic mm. of blood so obtained are blown into the test-tube, where they sink to the bottom of the distilled water.

A stream of coal gas is now allowed to run into the tube for two or three minutes ; it is not allowed to bubble through the water. When the tube is thoroughly filled with gas it is closed by the thumb and gently inverted to permit of the conversion of the oxyhæmoglobin into carboxyhæmoglobin. The mixture is now gradually diluted with distilled water until its colour exactly matches that of the standard in the control tube. After each addition of distilled water the contents must be mixed by gentle inversion of the tube and compared with the standard.

When the colours are identical the height of the fluid is read on the scale, which is graduated to show percentages of hæmoglobin.

(4) **Blood Culture Experiment.** By this is meant the attempt to cultivate any micro-organisms that may be circulating in the blood. It is advisable to withdraw 10 or 15 c.c. of blood from a vein, and it is essential to avoid contamination with skin cocci. When no vein of sufficient size is available, the ball of the great toe may be thoroughly cleaned with soap and water, alcohol and ether, and pricked with a needle and the blood collected in a sterile capillary pipette.

If a vein is used the antecubital fossa on one side should be carefully cleaned with soap and water and then covered with a solution of iodine in rectified spirit. A bandage is now applied to the upper arm so as to distend the peripheral veins. A syringe of 10 c.c. capacity containing half a drachm of 2 per cent. citrate of sodium solution to prevent clotting, and fitted with a hollow needle 1½ inches long and of medium calibre, is sterilised in an autoclave and is used for collecting the blood. In order to ensure that the syringe and needle are sterile when brought to the bedside it is convenient to have a screw adjustment to the base of the barrel of the syringe and to screw this into a special glass cylinder (like a large test-tube) which is long enough to take the syringe with its needle already attached.

The syringe is screwed into the cylinder ready for use and then the whole is sterilised by dry heat. The apparatus can now travel any reasonable distance without becoming contaminated, and the syringe is not withdrawn from the cylinder until everything is ready at the bedside.

The syringe is filled with blood by puncture of one of the distended veins. The needle is now removed, the nozzle of the syringe passed swiftly through the flame of a spirit lamp, and the blood straightway expelled into a flask containing 50 c.c. of sterile bouillon. Care must be taken in expelling the blood that it goes straight into the culture fluid and does not trickle down the neck of the flask. It is a good plan to put 5 c.c. of blood into each of two flasks of broth, which thus serve as controls on each other.

The flask containing the blood and the broth is incubated at 37° C. for seventy-two hours. Preparations should be made every twenty-four hours to see if any growth has

resulted. If there is any growth, subcultures on agar agar, blood agar, gelatine, and other media must be made in order to establish the identity of the organism.

Staphylococci must be regarded with more suspicion than other organisms, since the blood can easily be contaminated from the skin.

White staphylococci are almost certainly contamination. Streptococci or pneumococci are important as indicating septicæmia. Anthrax bacilli, gonococci, and Bacillus coli may be demonstrated in appropriate cases. Bacillus typhosus and Bacillus paratyphosus can nearly always be shown about the third day in cases of enteric fever or paratyphoid infection. It sometimes happens that the blood-serum contains so much anti-body that the growth of the organisms is inhibited unless the serum is enormously diluted by the culture medium. This is most likely to happen in such diseases as enteric fever, and it is for this reason that some authorities advise the inoculation of a series of ten or more broth-tubes with different amounts of the suspected blood from one drop to several c.c.'s, in order to arrive at that dilution which is most favourable to the growth of the particular organism.

II. ABNORMALITIES OF THE BLOOD.

(a) **The Red Cells and Hæmoglobin.** These are both diminished in amount in cases of anæmia. They are not as a rule diminished proportionately; hence there may be anæmia with a low colour index (chlorotic type) or anæmia with a high colour index (pernicious type).

Where there has been great destruction of red cells imperfectly developed red cells are seen in the blood. These parent cells are called *normoblasts*, and resemble the ordinary red cell except that they are nucleated. In severe cases the parent cell of the normoblast, namely the *megaloblast*, may appear. This is a large cell with an irregular nucleus which stains variably and is often split up into two or three parts. The megaloblast is only found in pernicious anæmia, in certain parasitic infections, in nitrobenzol poisoning, and possibly in the last stages of cancerous cachexia. Its presence is of grave significance.

Great variability in shape and size of the red cells (poikilocytosis) and in the way they take the stain (polychromatophilia) is a feature of pernicious forms of anæmia. In polychromatophilia some of the red cells are orange, some are grey, and some are stippled with little blue points.

But little importance can be attached to variations in the colour index or in the number, shape, size, and varieties of red cells found in infancy.

There may be an increase in the total number of red cells (polycythæmia) in the following conditions :—(i.) Pregnancy ; (ii.) High altitudes ; (iii.) Carbon-monoxide poisoning ; (iv.) With certain forms of splenomegaly ; (v.) Congenital heart-disease ; (vi.) New-born infants ; (vii.) Severe cholera.

It is sometimes desirable to estimate the fragility of the red cells, for example, in acholuric jaundice, but the results are not altogether reliable.

To perform the experiment suspensions of the patient's red cells are made in saline solution of different strengths from ·1 per cent. to 1 per cent., and the strongest salt solution noted in which hæmolysis occurs. Healthy blood cells do not hæmolyse in stronger saline than ·45 per cent.

A set of control tubes should always be put up containing known healthy red cells.

(b) The White Cells. An increase in the number of the white cells is known as a *leucocytosis*. A physiological leucocytosis up to about 10,000 or 11,000 is commonly found shortly after a large meal, and during the latter months of pregnancy. In infancy the white cell count is often between 20,000 and 30,000.

Pathological leucocytosis occurs in all forms of inflammation except a localised abscess which is draining freely.

It is also seen in nearly all the specific infective diseases except :—

Enteric Fever.
Tuberculosis (except Tuberculous Meningitis).
Measles and German Measles.
Malta Fever.
Mumps.
Chicken-pox.

Influenza.
Yellow Fever.
Malaria.
Kala Azar.
Syphilis.

In the varieties of leucocytosis hitherto considered the increase is principally in the polymorphonuclear leucocytes. It is important to remember that in all varieties of tuberculosis there is likely to be a *relative* increase in the number of lymphocytes.

Extreme leucocytosis (above 100,000) is only met with in cases of leukæmia, either Spleno-medullary or Lymphatic, or very occasionally in whooping-cough or plague.

In spleno-medullary leukæmia the increase is mostly in polymorphonuclear leucocytes and their parent cells, the *myelocytes;* the lymphocytes are also increased absolutely, though diminished relatively. The myelocyte is a large cell with a faintly-staining, often indented nucleus, and numbers of granules which may be neutrophile, eosinophile, or basophile. It is not present in health.

In lymphatic leukæmia the increase is entirely in the lymphocytes.

Leucopenia, or diminution in the number of white cells below 6,000, is most often seen in pernicious anæmia, enteric fever, splenic anæmia, influenza, malaria, yellow fever, and kala azar.

III. ANÆMIA.

The classification of the anæmias into primary and secondary is not satisfactory. It is probable that all anæmias (except anæmia from hæmorrhage) are secondary to the action of toxins (bacterial or chemical) manufactured elsewhere than in the blood-stream or blood-forming organs, and therefore that anæmias are all secondary, even though in many cases no primary cause can be established either clinically or post mortem.

(*a*) Anæmia after Hæmorrhage. The total quantity of fluid is made up in a few hours. The number of red cells returns to the normal in about three weeks, but the deficiency in hæmoglobin is not made good for two or three months

after a severe hæmorrhage. The appearance of poikilocytes and normoblasts is common during the stages of repair, and a distinct leucocytosis is the rule during the early stages.

(*b*) Chlorosis. This is a common disease of girls and young women. It starts at or shortly after puberty, and is generally cured by marriage and childbirth.

The pathology is obscure, but it seems probable that the ovarian internal secretion and auto-intoxication from constipation may each play a part. The predisposing causes are defective hygiene and lack of sunshine, exercise, and good food.

Symptoms and signs. The girl is often well nourished but flabby. She may have a red flush on the cheeks, sometimes (if a brunette) she is a definite greenish-yellow colour. In all cases the mucous membranes are pale.

The cardinal symptoms are dyspnœa on exertion, palpitations, indigestion, constipation, disordered menstruation (usually scanty, sometimes increased), a tendency to faintness and swelling of the feet in the evenings.

A hæmic murmur is frequently heard over the pulmonary area, and in some cases the myocardial debility is so great as to permit of dilatation of the mitral ring.

The *bruit de diable*, a continuous buzzing sound in the jugular veins, is described in connection with chlorosis. This is of little diagnostic value, as it so largely depends on the pressure of the stethoscope.

The Blood. The total quantity of the blood serum is increased. The hæmoglobin is markedly diminished, often down to 30 or 40 per cent. The red cells are moderately diminished, usually 70 to 80 per cent., but in view of the hydræmic plethora the diminution in the *total* number of red cells is but slight. The colour index is low, usually about ·6. The white cells are unaltered, but there may be a slight relative lymphocytosis. In very severe cases a few normoblasts may be seen.

Complications. Gastric ulcer, headaches, neuralgia, venous thrombosis (sometimes of the cerebral sinuses), and occasionally optic neuritis are the most important complications.

The diagnosis of chlorosis depends on the blood picture,

the age and sex of the patient, the signs and symptoms above described, and the absence of any discoverable cause for the anæmia. Every case should be carefully examined with a view to the possibility of early pulmonary tuberculosis.

(*c*) Pernicious Anæmia (Idiopathic Anæmia of Addison). The pathology of this disease is very obscure, but it is probable that the error lies in excessive destruction of red cells through some toxic agency rather than in the diminished activity of the hæmopoietic centres. The origin of the toxins is thought by some to lie in oral or gastro-intestinal sepsis, but the evidence is not conclusive. The destruction of the red cells is accompanied by the deposit of hæmosiderin in the liver and kidneys, and sometimes in the spleen.

Pernicious anæmia affects men rather more frequently than women ; it usually starts between the ages of 30 and 50 and develops very insidiously. It is unusual for the patient to seek advice until the disease is well advanced.

Symptoms and Signs. The symptoms are lassitude, muscular weakness and shortness of breath ; often there is abdominal pain, and vomiting is not infrequent.

Examination of the patient will show a peculiar lemon yellow coloration of the skin ; the subcutaneous tissues are well preserved. In severe cases there will be evidence of fatty degeneration in the heart muscle (*vide* p. 237). The activity of the hæmopoietic tissues may be shown by tenderness in the shafts of the long bones and in the sternum. There is usually a moderate enlargement of the spleen. Hæmorrhages are frequent in the later stages, both from the mucous membranes and into the retinæ. An extremely important phenomenon is the occurrence of sclerosis in the posterior and lateral columns of the spinal cord, which may, in some cases, give rise to ataxy and paresis. This degeneration is identical with that found in "Subacute Combined Degeneration of the Cord" (*vide* p. 556), and it is possible that the two diseases are produced by a common cause.

The temperature shows periodic waves of moderate fever, each wave lasting for two or three weeks and alternating with an apyrexial period of varying length.

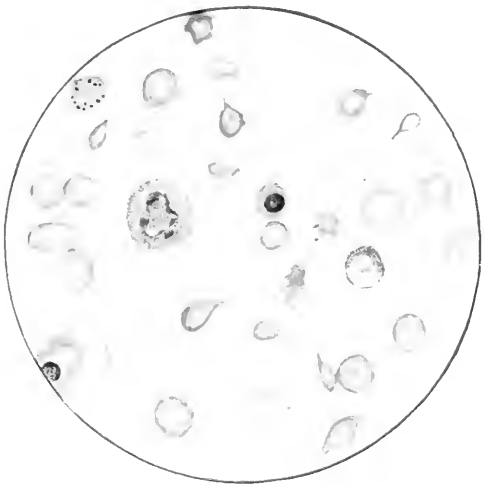


Fig. 36.

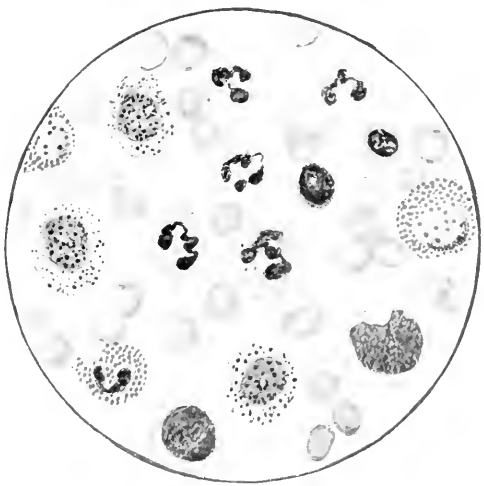


Fig. 37.

FIG. 36.—This picture represents a blood film from a case of *Pernicious Anæmia,* stained by Leishman's stain.

A typical blood count in such a case is as follows:

Red Cells . . 1,500,000, including normoblasts and megaloblasts.
Hæmoglobin . 45 per cent.
Colour Index . 1·4.
White Cells. . 4,500.

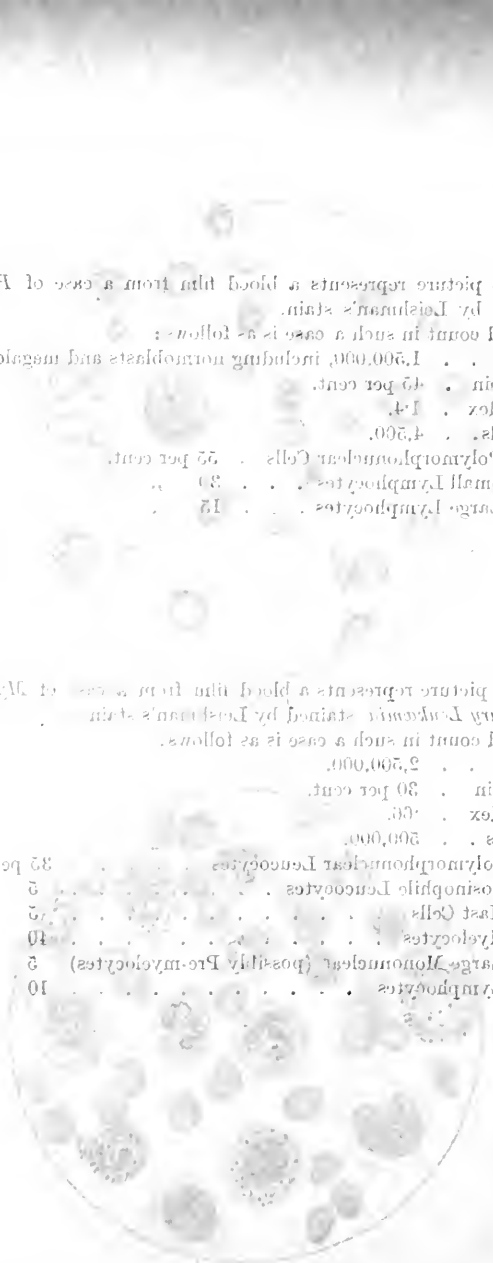
Polymorphonuclear Cells .	55 per cent.
Small Lymphocytes . . .	30 ,,
Large Lymphocytes . . .	15 ,,

FIG. 37.—This picture represents a blood film from a case of *Myelogenous* or *Spleno-medullary Leukæmia* stained by Leishman's stain.

A typical blood count in such a case is as follows:

Red Cells . . 2,500,000.
Hæmoglobin . 30 per cent.
Colour Index . ·66.
White Cells . . 500,000.

Polymorphonuclear Leucocytes	35 per cent.
Eosinophile Leucocytes	5 ,,
Mast Cells	5 ,,
Myelocytes	40 ,,
Large Mononuclear (possibly Pre-myelocytes)	5 ,,
Lymphocytes	10 ,,



In the majority of cases there are one, two, or more remissions in which there is an amelioration of all symptoms and a great improvement in the anæmia. Such remissions may last for six months or longer, but they are never permanent.

The urine contains an excess of urobilin, but no bilirubin ; there is often albuminuria.

The Blood (*vide* Fig. 36) shows very characteristic changes:— The red cells are reduced to between 20 and 40 per cent., or even lower. There is poikilocytosis and polychromatophilia. Normoblasts and megaloblasts are present. The hæmoglobin is reduced to from 50 to 60 per cent. ; consequently there is a *high colour index*. The white cells are diminished in amount, often below 5,000, but there is frequently a slight *relative* lymphocytosis.

Diagnosis. The blood picture is characteristic, and in conjunction with the symptoms and physical signs makes the diagnosis easy.

In very early cases there may be a low colour index, and the same thing may occur during one of the remissions.

The important features of the blood picture are megaloblasts, high colour index, and leucopenia.

A megaloblastic type of blood is sometimes seen in cases of *intestinal parasites* (ankylostoma duodenale, bothriocephalus latus, etc.), but there is no leukopenia in these conditions, and there is usually a marked increase in the number of eosinophile cells.

Cancer of the stomach may produce a somewhat similar red cell picture, but is usually accompanied by leucocytosis, while investigation of the gastric contents (*vide* p. 327) and examination of the abdomen should prevent error.

Nitrobenzol poisoning gives a similar picture except for the leucopenia but can be diagnosed from the history.

Chronic plumbism causes a somewhat similar anæmia, but the colour index is rarely above unity, and there is more basophilic degeneration of the red cells. Megaloblasts are usually absent, while the history and physical signs, such as blue line, wrist drop, colic, constipation, headache, and high blood pressure, should be conclusive.

The aplastic type of pernicious anæmia occurs more

often in women than men, and favours the young adult rather than the middle-aged.

It suggests a failure on the part of the hæmopoietic bone marrow to respond to the call made on it for the formation of new red cells by the action of toxins allied to those of pernicious anæmia.

The result is a great diminution in red cells, with a colour index of about unity and no nucleated forms, poikilocytosis or polychromatophilia.

Treatment has no effect and the disease runs a rapidly fatal course.

IV. LEUKÆMIA.

Two main forms of leukæmia are recognised :—the Spleno-medullary and the Lymphatic. In some few cases these varieties may both be present in the same person, giving rise to a mixed leukæmia. In both forms the symptoms are much the same—an insidious onset, with weakness, dyspnœa, lassitude, emaciation, gastro-intestinal disturbance, and often hæmorrhages from the mucous surfaces, especially menorrhagia and epistaxis. The temperature, as in pernicious anæmia, shows waves of elevation alternating with apyrexial periods.

(*a*) Spleno-medullary (Myelogenous) Leukæmia. The spleen is enormously enlarged, often extending to the pelvis. Occasionally there is tenderness over the sternum and long bones. The liver is often considerably enlarged.

The Blood (Fig. 37) shows very characteristic changes. There is a red-cell anæmia of chlorotic type. There is a leucocytosis up to 500,000, due to an increase in polymorphonuclear cells and their parent cells, the myelocytes. These two cells are often present in about equal numbers, together forming from 80 to 90 per cent. of the total white cells. In some cases the increase is almost entirely myelocytic. At the same time there is an increase in the eosinophile cells and in mast cells. The lymphocytes are absolutely increased, though relatively normal or diminished.

The diagnostic features are the abundance of myelocytes, which are not present at all in health (they may be neutro-

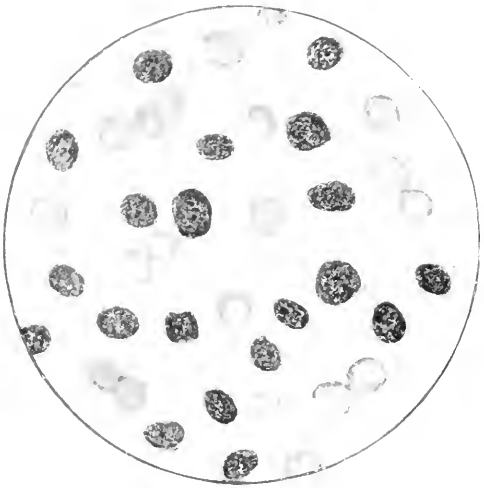


Fig. 38.

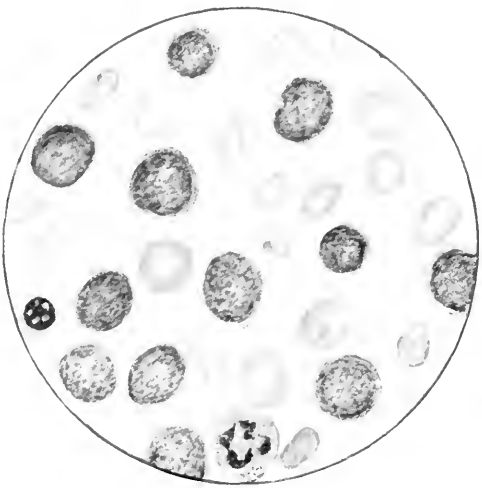


Fig. 39

Fig. 38.—This picture represents a blood film from a case of *Chronic Lymphatic Leukæmia* stained by Leishman's stain.

A typical blood count in such a case is as follows:

Red Cells	. .	2,000,000.
Hæmoglobin	.	20 per cent.
Colour Index	.	·5.
White Cells	. .	150,000.
Polymorphonuclear Cells	.	5 per cent.
Small Lymphocytes	. . .	90 ,,
Large Lymphocytes	. .	5 ,,

Fig. 39.—This picture represents a blood film from a case of *Acute Lymphatic Leukæmia*, stained by Leishman's stain.

A typical blood count in such a case is as follows:

Red Cells	. .	800,000.
Hæmoglobin	.	10 per cent.
Colour Index	.	·6.
White Cells	.	50,000.
Large Lymphocytes	. . .	85 per cent.
Small Lymphocytes	. . .	10 ,,
Polymorphonuclear Cells	.	5 ,,

Fig. 38.—This picture represents a blood film from a case of Chronic Lymphatic Leukæmia, stained by Leishman's stain.

A typical blood count in such a case is as follows:

Red Cells	2,000,000.
Hæmoglobin	20 per cent.
Colour Index	·5.
White Cells	150,000.
Polymorphonuclear Cells	5 per cent.
Small Lymphocytes	90 ,,
Large Lymphocytes	5 ,,

Fig. 39.—This picture represents a blood film from a case of Acute Lymphatic Leukæmia, stained by Leishman's stain.

A typical blood count in such a case is as follows:

Red Cells	800,000.
Hæmoglobin	10 per cent.
Colour Index	·6.
White Cells	50,000.
Large Lymphocytes	85 per cent.
Small Lymphocytes	10 ,,
Polymorphonuclear Cells	5 ,,

phile, eosinophile, or basophile, but these latter are relatively scanty), and the increase in mast cells.

(*b*) Lymphatic Leukæmia (Chronic form). The lymphatic glands throughout the body are enlarged, the individual tumours usually remaining discrete and freely movable. As a result of enlargement of the thoracic glands pressure effects are not uncommon.

The spleen is considerably enlarged, but not as a rule to the same extent as in the spleno-medullary form. The liver is moderately enlarged.

The Blood (Fig. 38) is very characteristic. There is a red cell anæmia with a low colour index. There is a white cell count of from 150,000 to 200,000 and of these cells 95 per cent. are small lymphocytes. No myelocytes are present.

(*c*) Lymphatic Leukæmia (Acute form). This disease occurs chiefly in childhood and adolescence ; it is associated with a septic (often gangrenous) inflammation of the mouth and fauces. There is a septic type of temperature, and the patients have a peculiar waxy-yellow appearance, paler than the colour of pernicious anæmia. *Hæmorrhages* from the mucous surfaces under the skin and into the retina are the rule. There is enlargement (usually slight) of the cervical glands, but the glands elsewhere often have no time to grow big before death ends the case. The spleen is generally palpable, and the liver may be slightly enlarged.

The Blood (Fig. 39) shows a very severe grade of red-cell anæmia (600,000 red cells is no uncommon count), with a low colour index. There is a white count of 40,000 to 60,000 (not often higher), and of these white cells about 95 per cent. are *large lymphocytes.*

The cases generally end fatally in from ten days to two months.

(*d*) Pseudo-Leukæmia. This term should be restricted to those cases associated with rickets, gastro-intestinal disturbances, etc., *occurring in infants,* in which there is splenic enlargement accompanied by a definite red-cell anæmia and a big leucocytosis. The colour index is usually low, but in some few cases it may be high. Blood films show poikilocytosis and polychromatophilia and many

normoblasts, some with double nuclei. Megaloblasts are present in scanty numbers. There is great variability in the white cells present; not infrequently a considerable number of myelocytes may be found.

(*e*) Leukanæmia. This name has been given to certain cases presenting the blood pictures of both spleno-medullary leukæmia and pernicious anæmia. There is a megaloblastic type of blood, and at the same time a great leucocytosis, consisting largely of myelocytes. This condition must not be regarded as a separate clinical entity; the majority of cases are slightly atypical spleno-medullary leukæmias, and the minority are late stages of pernicious anæmia.

V. PURPURA.

This consists of multiple hæmorrhages into the skin or mucous membranes; it is an expression of some toxic or septic process, and is therefore symptomatic. Clinically it is customary to recognise several forms of purpura :—

(*a*) Purpura Simplex, where the hæmorrhages are into the skin.

(*b*) Purpura Hæmorrhagica, where there is bleeding from the mucous surfaces.

Both these forms are liable to occur in septicæmia, pyæmia, leukæmia, and Bright's disease, and in the malignant forms of the specific infective diseases, such as small-pox, scarlet fever, measles, etc.

(*c*) Purpura Rheumatica (Schoenlein's Disease). It is still doubtful whether this condition is really connected with true rheumatism. It is met with in children, and consists in minute subcutaneous hæmorrhages, most commonly on the extremities, accompanied by pain and swelling in one or more joints. Inflammation of the fauces is not uncommon and slight pyrexia is the rule.

(*d*) Henoch's Purpura. Children are principally affected; there are purpuric patches on the skin, swelling of the joints, and severe abdominal pain, with vomiting and constipation. Later on there are hæmorrhages from the mucous membranes, especially of the bowel. Splenic enlargement and hæmorrhagic nephritis, or at least hæmaturia, are not uncommon

accompaniments of this malady. The abdominal symptoms are produced by hæmorrhage into the muscle coat of the intestine. Henoch's purpura has been mistaken for intussusception, which, indeed, it may cause. The joint pains, hæmaturia, and subcutaneous hæmorrhages should, however, prevent this mistake.

(*e*) Purpura Fulminans. This is a rare form of purpura affecting infants in the first few weeks of life. There are spreading subcutaneous hæmorrhages which soon involve an entire limb or even several limbs. These become swollen, tender and purple or even black. There are no hæmorrhages from the mucous membranes. The child dies in a few days with signs of severe septic infection.

Diagnosis. The diagnosis of purpura is easy, but it is not always so simple to demonstrate the cause of the purpura. A blood examination will show such conditions as *Leukæmia* or *Pernicious anæmia*.

Scurvy is indicated by the condition of the gums and the pain and swelling in the calves of the legs.

Septicæmia will be diagnosed by some primary focus, such as osteomyelitis or ulcerative endocarditis; and *Bright's disease* by the condition of the urine and the heart.

The hæmorrhagic or malignant forms of the specific infective diseases can often only be diagnosed by the presence of an epidemic, the history of exposure to infection, or the fact that some one else becomes infected from the patient.

In all forms of purpura it can be shown that there is a great diminution in the number of the blood platelets. Normally there are about 250,000 of these in each cubic millimetre of blood; in purpura they fall to 80,000, or even less.

There is but little information to be gained by counting platelets, since purpura is a self-evident fact, but if it be desired to enumerate them the following technique can be adopted:—

Carefully clean the patient's finger and place on it a drop of 10 per cent. sodium metaphosphate. Prick the finger through the drop and allow some blood to mix with the solution. Examine a drop of the mixture on the Thoma-Zeiss blood stage, if necessary diluting further, and note the

relative number of blood platelets and red blood cells present. If now an ordinary red blood cell count be done the number of platelets can readily be calculated.

VI. SCURVY.

A disease produced by prolonged deprivation of fresh food. The pathology is not understood; but the incidence of scurvy can be checked by the administration of lemons and limes to those people who are prevented from obtaining a sufficiency of fresh food and vegetables.

Symptoms. Gradual onset of muscular and mental debility, swelling and sponginess of the gums, with hæmorrhages from them; loosening of the teeth and foul breath; hæmorrhages into the skin and sometimes from the mucous membranes; painful swellings of the calves and beneath the periosteum of the shafts of the long bones, and nyctalopia (night-blindness). There is no pyrexia and no leucocytosis, but anæmia is well marked.

Diagnosis. The diagnosis can usually be made with certainty if there is a history of defective food supply and if the characteristic changes in the gums and in the calves of the legs are present.

Acute lymphatic leukæmia can be distinguished by its blood picture.

Mercurial poisoning can be diagnosed from the history.

Congenital syphilis can be eliminated by performing a Wassermann reaction on both the child and its parents.

Infantile scurvy (Scurvy rickets) is a disease occurring principally, but not exclusively, in children who have been fed on artificial foods. It may be associated with rickets. The symptoms and signs are those of ordinary scurvy with the addition sometimes of separation of the epiphyses.

VII. SPLENIC ANÆMIA.

This disease consists of a steadily progressive red-cell anæmia with a low colour index and a gradually increasing enlargement of the spleen.

The most characteristic feature of the blood picture is a

leucopenia ; the white cells usually number under 4,000 per cubic millimetre.

In the later stages hæmorrhages from the mucous surfaces (especially the stomach) are common.

Three types of splenic anæmia are sometimes described—the adult type, the infantile type, and Gaucher's type.

In the Adult type men seem to be affected more often than women ; the disease usually starts between twenty and forty, and the tendency is towards a chronic course.

The Infantile type (so called) is quite a different condition, and has been described under the heading of "Pseudo-Leukæmia." At the same time the adult type can and does occur in childhood, sometimes with a familial incidence.

Gaucher's type is, in all probability, a primary endothelioma of the spleen.

For the differential diagnosis of Splenic anæmia *vide* Spleen, Diseases of, p. 179.

VIII. LYMPHADENOMA (Hodgkin's Disease).

The clinical features of lymphadenoma are a progressive and diffuse enlargement of the lymphatic glands throughout the body, enlargement of the spleen, and anæmia of the chlorotic type.

All ages are liable, but the majority of cases occur in the first half of life. Men are more often affected than women.

The glandular enlargements are usually first noticed in the cervical region, then in the axilla, and then in the groin. The mediastinal and lumbar glands are frequently involved. At first the glands remain discrete, but after a while they fuse into large masses. Suppuration is very rare.

The splenic enlargement is rarely so great as in splenic anæmia or myelogenous leukæmia.

The blood is not characteristic ; there is a progressive anæmia with a low colour index. The white cells may be unaltered, or they may show a relative lymphocytosis ; sometimes there is a distinct polymorphonuclear leucocytosis, but this may be attributable to secondary infection. Death may occur from exhaustion or from pressure effects on

the trachea, œsophagus, etc., by the enlarged mediastinal glands.

The pathology of lymphadenoma is obscure ; it has been thought by Continental observers to be due to the tubercle bacillus, but this view is not accepted, though it is established that tuberculosis sometimes follows lymphadenoma.

Quite recently a bacillus of lymphadenoma has been described morphologically resembling the tubercle bacillus, but this work lacks confirmation up to the present.

The diagnosis of lymphadenoma from other varieties of lymphatic gland enlargement may be difficult. When practicable a gland should be excised and submitted to microscopical examination.

The structure of a lymphadenomatous gland is fairly characteristic : there is great increase in fibrous gland reticulum, a diminution of lymphoid elements, and the presence of big cells containing two or four central nuclei (lymphadenoma cells). Large mononuclear cells are also seen. Tuberculosis and lymphosarcoma can also be readily distinguished by this method (biopsy) ; indeed, in early cases it may be impossible to differentiate tuberculous adenitis from lymphadenoma by any other means. As a general rule there is less anæmia in tubercle, the spleen is not enlarged, and the glandular affection is more likely to be restricted to one group of glands. In later cases the tendency for tuberculous glands to break down is a very important diagnostic point (*vide* also p. 102).

IX. HÆMOPHILIA.

In this disease there is a tendency towards excessive bleeding after any injury however trivial, and also for the occurrence of apparently spontaneous hæmorrhages into the joints.

The condition is hereditary : it is conveyed by the daughters of bleeders, who do not themselves suffer ; on the other hand, the male bleeder does not transmit the tendency.

Hæmophilia is supposed to depend upon a deficient coagulation power in the blood and a congenital delicacy

in the blood vessels. In our opinion the methods now in use for testing the coagulation point of the blood are not of sufficient delicacy to be of diagnostic value.

The diagnosis is easy, especially if the patient is a man and there is a history of prolonged bleeding after tooth extraction or shaving cuts or other trifling injury.

An inquiry as to possible hæmophilia may elucidate some cases of sudden and severe swelling of a joint accompanied by great pain from distension.

CHAPTER II

DISEASES OF THE DUCTLESS GLANDS

I. DISEASES OF THE THYROID.

A. Myxœdema. The characteristic symptoms of this condition are produced by deficiency in the internal secretion of the thyroid gland.

In the majority of cases the thyroid gland is smaller than normal; occasionally it may be enlarged by cyst formation or excess of fibrous tissue, but in all cases there is loss of secretory tissue. Complete thyroidectomy is followed by myxœdema in a considerable proportion of cases. The disease is much more common in women than men, it usually appears between the ages of 30 and 50, and is predisposed to by excessive child-bearing.

An hereditary influence can be traced in some of the cases. Occasionally exophthalmic goitre may be followed or even accompanied by myxœdema.

The Signs and Symptoms are :—A malar flush, but pallor elsewhere, dryness of the skin, absence of perspiration and loss of hair. There is a gradual increase in general bulkiness, with swelling of the skin of the face, of the tongue, mouth, nose and lips. There is broadening of the hands and feet; sluggishness of movements; diminished mentality and powers of speech; thick, husky voice; a subnormal temperature associated with a definite feeling of chilliness; loss of memory; and in extreme cases definite delusional insanity. Despite the general swelling of the subcutaneous tissues, there is no true œdema and *no pitting on pressure.*

The course of myxœdema is very chronic; even if untreated, the patient usually dies of some intercurrent malady.

The diagnosis is not difficult in well-marked cases. Chronic parenchymatous nephritis should be distinguished

by the true œdema and the urinary signs, though it must be remembered that albuminuria is often present in myxœdema.

The mental dulness, the dry skin, and the loss of hair are not present in Bright's disease.

Acromegaly can be distinguished by the definite *bony* enlargements of the hands, feet, and lower jaw, as well as by the visual phenomena, and the absence of the dry skin, scanty hair, and thick speech (*vide* also p. 181).

Should any doubt exist as to the diagnosis, thyroid extract (1-5 gr.) should be exhibited; the manner in which myxœdema reacts to treatment is highly characteristic.

B. CRETINISM is due to a loss of thyroid function which is either congenital or appears before puberty. It is rarely noticed before the age of six months. Predisposing factors are residence in goitrous districts, consanguinity of parents, and a family taint of insanity.

The Signs and Symptoms are :—Impaired development, both mental and physical; dry skin and dry, brittle, scanty hair; swollen, bloated face with puffy lips and eyelids; large tongue, often protruding; domed palate; blobby nose, with depressed bridge; short, thick limbs; open fontanelles and delayed dentition; large, prominent abdomen, and marked lordosis. Those cases which develop about the age of four or five after a healthy infancy may well, as suggested by Parker, be called "juvenile myxœdema."

The diagnosis of cretinism is not as a rule difficult. Very slight or early cases can sometimes only be diagnosed after a course of treatment with thyroid gland.

Congenital syphilis usually presents some characteristic features (*vide* p. 73), but, if necessary, a Wassermann reaction both of the child and its parents should be done to clinch the diagnosis.

Achondroplasia may at first sight be mistaken for cretinism, but the lack of mental impairment and the preservation of muscular power are important points against cretinism. Further, the extreme shortness of the limbs, especially of the proximal segments, the equal length of

the fingers and tri-radiate appearance of the hands, as well as the curved limbs and the contracted pelvis, should prevent error.

Mongolian Idiots sometimes present a superficial resemblance to cretins; their oblique eyes, deep epicanthic folds, and fissured tongues serve to distinguish them as a rule.

C. Exophthalmic Goitre (Graves' Disease). This condition is due to increased activity of the thyroid gland and is the exact converse of myxœdema.

Women are very much more frequently affected than men; the onset of symptoms usually occurs between the ages of 18 and 30. Predisposing causes may be fright or violent emotion.

The Signs and Symptoms are :—Increased activity of the skin, with ready perspiration, acceleration of the cardiac action, with palpitation, and throbbing of the arteries in the neck; a fine involuntary tremor of the extremities; progressive emaciation; *diffuse enlargement* of the thyroid gland, and exophthalmos. Obvious enlargement of the thyroid and exophthalmos may be delayed for some time after the other symptoms are well marked.

In connection with the protrusion of the eyeballs the following signs are described :—

Von Graefe's Sign. When the eyeball is rotated downwards the descent of the upper lid is delayed, with the result that a band of sclerotic is visible between the iris and the upper eye-lid.

Stellwag's Sign. Retraction of the upper lid to such an extent that it does not descend at all.

Möbius' Sign. Lack of power of convergence for accommodation purposes.

The tachycardia leads to dilatation of the heart; systolic murmurs, both apical and basal, are often heard.

Certain complications are of importance. Diarrhœa and vomiting may be very troublesome, as may pruritus. Pigmentation of the skin, either localised or diffuse, is often seen; patches of solid œdema, usually transient, have been described. The nervousness, irritability of temper, and excitability which are symptomatic of the

disease may culminate in acute mania. Sclerodermia or myxœdema may follow Graves' disease.

Glycosuria and albuminuria are sometimes present.

The Diagnosis presents no difficulty when the signs are well marked; it is important to remember that exophthalmos and thyroid enlargement are not invariably present.

The temporary thyroid enlargement, with a slight feeling of choking, which is sometimes present about the age of puberty, should not be mistaken for Graves' disease, though it may develop into it.

A thyroid adenoma is more likely to be recognisable as a tumour in some part of the thyroid gland, whilst the exophthalmic goitre is a diffuse enlargement of the whole gland, including the isthmus. In a very few cases the enlargement has been limited to one lobe of the gland.

An " endemic goitre " consists of a diffuse parenchymatous enlargement of the thyroid gland, but is not accompanied by any of the signs of Graves' disease. Such goitres may be simple, cystic, or fibrous.

Very early cases of exophthalmic goitre may present great difficulty. Great importance must be paid to the tremor and the cardio-vascular signs. Both tachycardia and tremor may, however, be due to hysteria.

The disease tends to run a very chronic course; in about 20 per cent. of all cases there is a complete recovery, and in a further 30 per cent. the disease becomes arrested after a certain point; in the remainder the patients continue to get slowly worse.

Very occasionally an acute form of Graves' disease is met with in which the disease progresses so rapidly that death takes place in a few months, or even weeks, from the onset of symptoms.

II. DISEASES OF THE THYMUS.

At birth the thymus gland weighs 13 grms., under ordinary circumstances it increases in size for the first twelve or fifteen years of life, at which age its weight is about 27 grms. After this it gradually atrophies, until by the twenty-fifth year it has practically disappeared. Under

certain circumstances it may persist or be of abnormal size.

One of the most important conditions associated with an enlarged thymus is "Status Lymphaticus," in which there is lymphoid hyperplasia throughout the entire body.

Status lymphaticus can hardly be diagnosed clinically unless there is enlargement of the cutaneous or mesenteric glands. In rare cases the thymus may be so enlarged as to give an increased area of dulness over the manubrium sterni, and sometimes it is supposed to cause dyspnœic attacks from pressure on the trachea (thymic asthma). It is a recognised cause of sudden death, and also of death while under the influence of an anæsthetic. Patients with status lymphaticus nearly always have globular dilated hearts.

The thymus persists in cases of exophthalmic goitre, myasthenia gravis and usually epilepsy, acromegaly, and lymphatic leukæmia.

It has been suggested that mediastinal lymphosarcoma may often arise in a persistent thymus gland or in the remnants of the thymus gland.

III. DISEASES OF THE SUPRARENAL BODIES.

A. **Addison's Disease** is in the great majority of cases due to tuberculosis of the suprarenal glands; very rarely it may be produced by simple atrophy, malignant disease, or pressure from without. There are two theories as to the pathology of the condition:—

(i.) That it depends on a loss of function of the suprarenal bodies, with the result that the body is deprived of their internal secretion.

(ii.) That it is an affection of the abdominal sympathetic nervous system.

Men are more liable than women, and the disease usually starts in the third or fourth decades of life.

Signs and Symptoms. The onset is gradual, with debility and muscular weakness, pigmentation of the skin, vomiting, and a weak, poorly-sustained pulse. The pigmentation is most characteristic: it varies from light yellow to deep bronze; it is most marked on the exposed parts, such as the hands

and face, on the parts that are normally inclined to be pigmented, such as the genitals, nipples, etc., and where there has been constant pressure, such as the corset area in women or those parts in contact with the braces in men. The mucous membranes do not escape, and pigmented areas should always be searched for in the mouth and conjunctivæ and, if necessary, in the vagina.

The muscular weakness is so marked as in time to prevent walking about. Giddiness and syncope are common. The blood pressure is extremely low, often from 60 to 80 mm. of mercury.

The disease runs a course of varying length; patients who suffer from persistent vomiting sometimes die in a few months, others may survive for several years. Death is usually from exhaustion or from the supervention of tuberculosis elsewhere.

The diagnosis may be very difficult in the early stages; the most important signs will be asthenia, pigmentation and low blood pressure. Sometimes there is an increased carbohydrate tolerance so that glycosuria does not result from very large doses of sugar (*e.g.*, 300—350 grms.).

There are many other causes of pigmentation which must be excluded before making a positive diagnosis of Addison's disease in the absence of other symptoms, such as :—

Argyria. Here there is a history of silver, whilst the coloration of the pigmented areas is more grey.

Arsenic, especially in cases of *pernicious anæmia;* this can be diagnosed by the history and the blood picture.

Bright's Disease. Patients with red granular kidney may become very deeply pigmented indeed. The high blood pressure and the absence of pigment in the mucous membranes will prevent a diagnosis of Addison's disease being made.

Chloasma uterinum, e.g., in pregnancy or uterine disease, can be detected by appropriate examination.

Cirrhosis of the liver in the later stages is often accompanied by extensive pigmentation, but it is usually due to an obvious jaundice, as shown by the sclerotics and the urine.

Exophthalmic goitre may be accompanied by considerable

pigmentation, but the characteristic signs of the disease will be present.

Pediculosis and *dirt* may produce extreme discoloration of the skin (not the mucous membranes). This is only met with in tramps and in the lowest social grades.

Abdominal tumours may be accompanied by deep pigmentation, but careful examination may reveal the tumour, and the history will not be that of Addison's disease.

Certain women, especially brunettes, become pigmented for no apparent reason and without impairment of health; more often slight pigmentation in women may be attributed to constipation and auto-intoxication or, if their age is appropriate, to the menopause.

In cases of doubt the special tests for tuberculosis may be of assistance (*vide* p. 105).

B. Malignant tumours of the suprarenal bodies are rare, but when they do occur are sometimes accompanied by striking signs of over-activity of these glands, namely, hyper-trichosis, extreme sexual precocity, high blood-pressure and glycosuria. These phenomena, developing in a young child, might suggest the correct diagnosis.

C. Hæmorrhage into the suprarenal bodies (acute hæmorrhagic adrenalitis) is a rare cause of death; the clinical features are asthenia, emaciation, low blood-pressure and exhaustion.

IV. DISEASES OF THE SPLEEN.

The spleen is situated beneath the ninth, tenth and eleventh left ribs, separated from them by the diaphragm; its long axis is along the tenth rib, and occupies the middle third of the axilla. It is not palpable in health. Small enlargements can sometimes be made out by careful percussion; moderate enlargements can be appreciated by pushing the tips of the fingers up underneath the left costal margin, when the lower pole of the spleen may be felt.

Great enlargements cause a tumour of varying size to appear in the abdomen: it proceeds from under the ribs downwards and forwards, keeping mostly to the left of

the abdomen, but often reaching the umbilicus ; its lower extremity is rounded, and there is a notch in its anterior border. It is not possible to get above it ; it seems very superficial ; it is dull to percussion, and the dulness is continuous with the ordinary splenic dulness.

A splenic tumour moves with respiration.

The most likely tumour to mistake for splenic enlargement is a renal tumour. This is more deeply situated in the loin ; it does not move so freely with respiration (unless it be a movable kidney, when it may be possible to get above it), and there is usually a resonant band, due to a distended colon over the tumour. There is no notch, and, unless it is very large, the tumour does not appear to grow out from under the ribs as does the enlarged spleen.

Carcinoma of the fundus of the stomach may suggest a splenic tumour ; its great irregularity and the absence of notch, as well as the symptoms of malignant disease and the examination of the gastric contents, will usually suffice to establish the diagnosis.

The following table suggests the more common causes of splenic enlargement and the means of diagnosing them :—

A. *Slight Enlargements.* Specific Infective Diseases, as :—	
Enteric fever	Evidence of the various infections concerned.
Pneumonia	
Malta Fever	
Typhus	
Intestinal Anthrax (splenic fever)	
Relapsing fever	
Chronic Venous Congestion ..	Apart from coexistent infarction, the spleen is but little enlarged from this cause.
B. *Moderate Enlargements.*	
Pernicious Anæmia	Blood examination.
Acute Lymphatic Leukæmia	And Glandular swellings.
Lymphadenoma (may be great enlargement) ..	
Infarction	Evidence of cardiac disease.

B. *Moderate Enlargements*—continued.

Thrombosed Splenic or Portal Veins	Sudden onset of splenomegaly with ascites and hæmatemasis.
Accompanying Cirrhosis of Liver. Portal / Biliary	Evidence of cirrhosis.
Weil's Disease	(*vide* p. 384).
Rachitis .. .. .. / Congenital Syphilis	Evidence of primary disease.
Malignant Disease (rare) ..	Evidence of primary focus (primary malignant disease in spleen is extremely rare).
Tuberculosis (rare)	Evidence of generalised tuberculosis or of tuberculous peritonitis.
Malaria (may be great enlargement)..	History: signs of Malaria and presence of parasites in the blood.
Splenomegaly with Acholuric Jaundice (rare)	Negative blood except for increased friability of red cells, yellow coloration of skin, absence of bilirubin in urine, familial incidence.

C. *Great Enlargements.*

Splenic Anæmia .. .. / Spleno-medullary Leukæmia / Chronic Lymphatic Leukæmia	Blood Examination.
Splenomegaly with Polycythæmia	And Cyanosis.
Trypanosomiasis	Residence in West Africa: presence of trypanosomes in blood.
Kala Azar	History of residence abroad (especially Assam), splenic puncture, and discovery of Leishman-Donovan bodies in the blood.

V. DISEASES OF THE PITUITARY BODY.

A. **Acromegaly.** It is convenient first to describe acromegaly, which is clinically the best known of the pituitary disorders. This is a condition associated with disease of the "pituitary body" and believed to be due to hyper-pituitarism that is to say, to excess of some internal secretion of this gland.

The disease usually shows itself in the third or fourth decades, and practically never starts after the age of forty. It is more common in women than in men.

The characteristic signs are dependent upon an overgrowth or diffuse hypertrophy, including *all the tissues*, of the hands and feet and certain other parts. The wrists and ankles are involved, but not the forearms or legs. The result is a curious spade-like appearance of the hands and feet. The greatest increase in size is often in the thumbs and great toes. The head is increased in size as a whole, but the change is particularly marked in the lower jaw, which sticks out in front of the upper one, while the teeth become separated from each other. The tongue, eyelids, and nose may be very much hypertrophied. The skin tends to be greasy, and the natural lines in it are greatly accentuated.

In 50 per cent. of all cases there are definite visual changes, the most common being bi-temporal hemianopia or optic atrophy. In extreme cases the ribs, sternum, and clavicles may be hypertrophied. Kyphosis is common. Gigantism is generally identical with acromegaly which has developed during the period of natural growth.

The clinical picture of acromegaly is fairly distinctive; it must not be mistaken for the following:—

(i.) *Osteitis Deformans* (Paget's Disease). This is a rare disease of unknown causation, characterised by a rarefying osteitis of certain bones. The skull becomes of great size, and causes the face to have a triangular shape with the base of the triangle upwards.

There is also a dorso-cervical kyphosis and hypertrophy (apparent) of the clavicles, humeri, and femora, the latter becoming bent forwards. There appears to be a tendency for sufferers from this disease to develop malignant tumours;

if they live long enough there is a very definite diminution in height, due to the kyphosis and the bending of the thigh bones.

There is some evidence that osteitis deformans is in some way connected with syphilis ; certainly many patients give a positive Wassermann reaction.

(ii.) *Leontiasis Ossea.* A rare condition characterised by overgrowth of the bones of the head and face, especially of the superciliary ridges and malar bones.

The pathology is unknown ; occasionally it may coexist with osteitis deformans.

(iii.) *Hypertrophic Pulmonary Osteo-arthropathy.* This condition is associated with chronic pulmonary disorders, such as empyema, bronchial asthma, emphysema, and the like. Occasionally it may follow syphilis and heart disease. It is characterised by overgrowth of the hands and feet and also the ends of the long bones. The most striking change is in the terminal phalanges, which become noticeably thickened and splayed out ; the nails are large and curved and show longitudinal striation.

The localisation and appearance of the overgrowth is not in the least like acromegaly, while the presence of a pulmonary or cardio-vascular lesion should settle the diagnosis.

(iv.) *Myxœdema* (*vide* p. 172).

B. Pituitary Disorders as a whole.

The *Pituitary body* consists of three parts :—

(i.) An anterior lobe, which is formed by an up-growth from the pharynx.

(ii.) A posterior lobe, which is a down-growth from the central nervous system.

(iii.) A "pars intermedia," which connects the anterior and posterior lobes, but which, histologically, is the anterior part of the posterior lobe.

Clinically it is convenient to include in the comprehensive term " Dyspituitarism " all the various conditions which are known or believed to depend on alterations in the functions of the pituitary gland.

It is obvious that the gland may err in the direction of either over- or under- activity, just as does the thyroid gland,

but in the case of the pituitary body the outlook is obscured at present by lack of accurate knowledge concerning the respective functions of the anterior and posterior lobes and the different ways in which these may be affected by disease. For example, it is possible that we might get over-action of the anterior lobe combined with deficiency of the posterior lobe, or *vice versâ*, or both lobes might be either too vigorous or atrophic at one and the same time.

Nevertheless, it is probable that the pituitary body is *usually* affected as a whole, and it is possible to give a list of signs and symptoms which may indicate respectively hyper- and hypo- pituitarism.

It is important to remember that, apart from invasion of the pituitary body by malignant growths (in which case progressive hypo-pituitarism may be the first local sign), the initial symptoms in the commonest forms of pituitary disease (struma or adenoma) are likely to be indicative of over action, but that as the disease progresses the essential properties of the gland are likely to be abolished by pressure or atrophy, so that hypo-pituitarism may supervene.

This may explain those cases of acromegaly in which no pituitary enlargement has been found after death, for, if the over-action has continued sufficiently long to produce bony changes, these will persist even when the under-action becomes apparent and so the actual hypo-pituitarism of the later stages of the disease may be missed.

The symptoms of *hyper-pituitarism* are in the main those that have been described under the heading "Acromegaly"; while if the change occurs before the epiphyses have united, gigantism precedes the acromegaly, which will develop later unless spontaneous arrest of the morbid process takes place.

These acromegalic signs are believed to indicate an increased function of the anterior lobe of the pituitary body.

Hyper-trichosis, sexual precocity, high blood pressure, and excessive activity in the direction of carbohydrate metabolism (leading to hyper-glycæmia and glycosuria) are possible signs of over-activity of the posterior lobe.

Hypo-pituitarism may present the following features (cf. the Fat Boy of "Pickwick"):—

Adiposity, drowsiness, smooth skin, loss of hair in axillæ

and pubic region, slow pulse, low blood pressure, and greatly increased carbohydrate tolerance.

These symptoms are probably due to insufficiency of the posterior lobe.

A low temperature and susceptibility to cold may indicate anterior lobe insufficiency.

A striking feature in many cases of hypo-pituitarism is the change in sexual characteristics. Adults lose their sexual power and develop reversive changes in their sexual apparatus, while children remain infantile in growth and appearance and never develop the adult sexual features. The male sex assumes many of the physical attributes of the female.

Hitherto we have mentioned only the glandular signs of pituitary tumour.

The *General signs* vary somewhat from the classical signs of other cerebral tumours. Headache is frequently present at some time or another, but is not so marked or so constant as in ordinary cerebral growths. Vomiting is comparatively rare.

Optic neuritis may occur, but is not nearly so common as optic atrophy. Sometimes a choked disc follows the optic atrophy.

Certainly the most important diagnostic point is *bi-temporal hemianopia;* this is constant and may be preceded by a similar loss of colour vision.

CHAPTER III

DISORDERS OF METABOLISM AND CONSTITUTIONAL DISEASES

I. DIABETES MELLITUS.

Definition. A wasting disease characterised by profound disturbance of carbohydrate metabolism, and by the appearance of glucose in the urine.

Etiology. Diabetes is predisposed to by a sedentary life, excessive brain work, and over-indulgence in rich food; it is most frequently seen in the second half of life, in the male sex, and it is common among Eastern races, especially the Jews. There is a strong hereditary influence. The exact pathology of diabetes is still unknown, but certain facts stand out, viz :—

(i.) Diabetes is accompanied by hyper-glycæmia, that is, more than ·2 per cent. of sugar in the blood.

(ii.) There is a disability to derive heat-energy from the circulating sugar by the exercise of those glycolytic functions which occur in healthy persons.

(iii.) The demand for sugar by the tissues continues, notwithstanding that they cannot utilise it when they have got it, and the disability of the tissues is fostered, if not produced, by the presence of hyper-glycæmia.

(iv.) A certain amount of heat is necessary for continued existence—the bulk of this is normally furnished by carbohydrates; therefore, in diabetes, when carbohydrates fail other food-stuffs are pressed into service for heat production. If enough heat is not provided by the food taken in, the heat is obtained by breaking up the body tissues themselves, and the strain of this abnormal metabolism is first felt by the fats and later by the proteids also. The wasting of diabetes is partly explained in this manner.

(v.) There is a danger that in these processes of abnormal

metabolism certain bodies may be formed which normally are either absent or only present in very small amounts, and that the excess of these bodies may be directly harmful or even lethal to the patient. These toxic bodies are largely formed in diabetes by excessive or abnormal fat destruction, and circulate in the blood as acetone, diacetic acid, and β amido-oxybutyric acid. The presence of this latter is directly or indirectly the cause of diabetic coma.

(vi.) The most constant lesion in diabetes (though it is not invariable) is a fibrosis of the pancreas. Experimental extirpation of the pancreas is followed by diabetes.

(vii.) Clinical diabetes may also be associated with cirrhosis or other extensive disease of the liver and lesions of the brain, especially when situated in the vicinity of the fourth ventricle and medulla.

(viii.) Every person has a sugar toleration point beyond which the ingestion of further sugar is followed by glycosuria. The average amount of sugar that can be taken by an adult without glycosuria resulting is 100—200 grms. Some people have a very low sugar toleration point, but they have not necessarily got diabetes ; this may partly explain why it is not infrequent to find transient glycosuria in elderly people who are gouty, arterio-sclerotic, and who " do themselves well," though possibly arterio-sclerosis is itself a factor, since it is well-nigh universal in true diabetes.

(ix.) A certain amount of sugar passes straight into the circulation without storage (as glycogen) in the liver.

It is accepted that, in health, the glycogen stored up by the liver is set free into the circulation as sugar, and that this sugar is broken up by glycolytic enzymes into CO_2 and water and so furnishes heat energy to the body. These enzymes are manufactured largely by the muscles, and to some extent also by the liver and possibly other organs, but in order that the enzyme may exercise its glycolytic action on the sugar it must first be activated by a " hormone." Possibly this hormone is supplied by the internal secretion of the pancreas.

In diabetes, if the pancreas is extensively diseased, the hormone is not forthcoming and glycolysis is greatly interfered with. Even if the pancreas is not demonstrably diseased, its nervous control may be impaired with the same

result. Cases of cerebral lesions accompanied by glycosuria may perhaps be explained in this manner.

It is obvious that the liver may, theoretically, produce diabetes by turning its glycogen into sugar and discharging it into the circulation more quickly than it can be broken up by the tissues and in this way producing a hyper-glycæmia. Or possibly the glycogenic function of the liver may be upset, and sugar may pass, in excess, straight into the blood.

Hence an alternative theory has been set forth which assumes that diabetes is due to excessive mobilisation of sugar by the liver and not to any disability of the organism to use circulating sugar. According to this view the output of sugar by the liver is stimulated by the internal secretion of the suprarenal glands and the activity of this suprarenal secretion is checked by the internal secretion of the pancreas. Since the suprarenal bodies have similar properties to the posterior lobe of the pituitary body, pituitary glycosuria can readily be explained on this hypothesis. Further the internal secretion of the thyroid body is supposed to counteract the action of the pancreas in checking suprarenal activity so that the occurrence of glycosuria in some cases of thyroid disease can be explained.

This theory is attractive, but, up to the present, we consider that the balance of evidence is in favour of the older view.

The Cardinal Symptoms of Diabetes are :—Thirst, polyuria, wasting, and hunger. The first three are almost invariable, though there may be latent periods or remissions in which they may not be striking. The other manifestations and symptoms are numerous, and they are of special importance, since they may give the first clue to a correct diagnosis : they are :—

(i.) *Of the Eye.* Cataract, neuro-retinitis, and hæmorrhages.

(ii.) *Of the Skin and Mucous Membranes.* Dryness, eczema, pruritus, carbuncles, boils, and gangrene. The tongue is dry, glazed and "beefy."

(iii.) *Of the Nervous System.* Coma, neuritis, headache, drowsiness, insomnia and paraplegia.

(iv.) *Of the Sexual Organs.* Impotence and abortion.

(v.) *Of the Muscles.* Cramp and great weakness.

(vi.) *Of the Lungs.* Tuberculosis and chronic bronchitis.

(vii.) *Of the Blood-vessels.* Arterio-sclerosis.

(viii.) *Of the Kidneys.* Albuminuria.

The temperature of diabetics tends to be subnormal, and they are nearly always constipated.

The *Urine* is greatly increased in amount (it may even be passed to the extent of ten to fifteen pints daily); it is clear, pale and of a high specific gravity (1030—1040). Glucose is present in amounts varying from a trace to 15 per cent. In severe cases there may be a smell of acetone, and both this and diacetic acid may be shown by appropriate tests (*vide* pp. 429—431). Often, however, the patient's breath may smell of acetone without there being any present in the urine. Not infrequently there is a slight albuminuria.

The outstanding complication of diabetes is *coma*, which may develop with great suddenness, especially after undue muscular exertion. Coma is, however, more often ushered in with the important symptoms of drowsiness, air hunger, and abdominal pain; whilst the fatty acids can be found in the urine even though, as often happens, there is a marked fall in the output of sugar.

The diagnosis of diabetes does not present any difficulty provided that a routine examination of the urine is made in every case. When a reducing body is present in the urine it is first necessary to be sure that it is sugar (*vide* section on "Urinary Analysis"). Having established the fact that sugar is present, the case must be regarded as potential diabetes until adequate investigation has been carried out for at least several weeks. If sugar is present only on certain occasions, especially after a heavy meal of carbohydrate, and the patient is otherwise in perfect health, or if there is a small amount of sugar in the urine of an elderly and fat, gouty, or arterio-sclerotic gourmand, then a very favourable view may be taken, *quâ* diabetes, especially if, in the latter case, the sugar can be controlled by a trifling adjustment of the diet.

Cæteris paribus, the presence of sugar without polyuria

and in a urine of low specific gravity is favourable. Scientifically there can be drawn no hard and fast line between glycosuria and diabetes, and only time and experience can show which cases are of clinical significance.

Prognosis. Nearly all the diabetics *can* make use of *some* carbohydrates, and the prognosis varies directly with the amount which the patient can, with proper treatment, be educated to utilise. For the rest the prognosis varies with the age, the older the patient the better the prognosis. In children and young people diabetes is nearly always quickly fatal. "Acidosis" is always of serious import, as are carbuncles or gangrene; at the same time elderly patients may live for years with a constant acetonuria provided there are no other complications and adequate treatment can be employed.

II. DIABETES INSIPIDUS.

This is an obscure disease characterised by the passage of very large amounts (up to fifty pints daily) of clear, pale, watery urine of low specific gravity, free from sugar (unless in the merest traces) or albumin, and without any increase of solids.

Two groups are recognised:—First, those in which there is a definite lesion, such as cerebral syphilis, a tumour of the brain, or a head injury; and, secondly, those in which there is no discoverable lesion (so-called "idiopathic" form). Occasionally an hereditary influence may be observed.

As would be expected from the depletion of the system by the passage of so much water, thirst is the outstanding symptom of diabetes insipidus.

The diagnosis is obvious in marked cases, but in the slighter forms care must be taken to exclude diabetes mellitus, red granular kidney, hysterical polyuria, and even hydronephrosis.

The prognosis in the symptomatic form depends entirely on the causative lesion; in the idiopathic form it is good as regards life but most uncertain as regards cure. Some cases recover spontaneously; others persist without notable impairment of health for many years.

III. GOUT.

Definition. A disorder of metabolism characterised by a tendency to arthritis with the deposition of sodium biurate in the connective tissues of and around the joints, and also by certain visceral manifestations.

Etiology. The etiology of gout is imperfectly understood, but the disease would appear to depend upon defective metabolism (probably excessive oxidation) plus impaired excretory power. It is more common in men than in women, and occurs principally between the ages of 40 and 65.

Certain factors stand out as predisposing causes, notably heredity, over-indulgence in food and alcohol, and lead poisoning. There is in addition a nervous or mental factor, especially in the determination of an acute attack. Certain climates (*e.g.*, India and to a less extent America) seem unfavourable to the development of gout.

Uric acid is manufactured in the body, partly exogenously by oxidation of the purin bases, which are in turn derived from ingested nucleo-proteid (uric acid itself is tri-oxy-purin), and partly endogenously, from the by-products of tissue katabolism.

According to Sir William Roberts the bulk of the uric acid, however formed, circulates in the blood as sodium quadriurate, which is a soluble salt but unstable, since it is always ready to take up another atom of sodium and to become sodium biurate, which is stable but highly insoluble. Further, while sodium biurate is soluble in distilled water in the proportion of 1 in 1,000, this solubility decreases rapidly if sodium salts be added to the solution till in water containing ·7 per cent. of sodium bicarbonate the biurate is practicably insoluble.

If, then, there is a temporary or permanent impairment of the excretory functions, there will be an excess of circulating quadriurate, which will attach to itself an atom of sodium from the existing sodium carbonate of the plasma. It is reasonable to suppose that this transformation of quadriurate into biurate is most likely to occur in those regions where there is most sodium carbonate, and there is experimental evidence to show that cartilage and the synovial

fluids are especially rich in this substance. The prevalence of uratic deposits in the joints may thus be in part explained. Another factor is the stagnation of synovial fluid, which physically favours the deposition of circulating salts.

The above theory is given for what it is worth ; but it is only fair to say that but little is *known* concerning the pathology of gout, although it is generally accepted that there is an excess of circulating uric acid (in some form or another) in gouty persons (*vide* also p. 414). The inflammatory changes in gouty joints are thought to be secondary to the deposition of the sodium biurate, and not *vice versâ*.

Clinical Features. Clinically gout may be *acute*, *chronic*, or *metastatic*.

(i.) *Acute Gout.* For some days there is likely to be irritability of temper, depression, and possibly twinges of pain in the smaller joints. The real acute attack usually starts with extreme suddenness, often in the middle of the night. The most common joint to be affected is the metatarso-phalangeal joint of the right great toe, but other joints may be selected.

The pain is agonising ; the joint swells up and becomes purple, shiny, and often œdematous. At the same time there will be constitutional signs, such as moderate fever, furred tongue, constipation, and anorexia. The urine is scanty and high coloured, while the output of uric acid is temporarily increased. After a few hours the pain subsides somewhat, only to return the next night. The pain and swelling commonly disappear by the end of the week. The joint recovers its mobility perfectly after the first attack, though repeated attacks leave permanent structural deformity.

After an attack of acute gout the general health is particularly good for a while, but each attack renders a subsequent one more likely, and the intervals between successive attacks grow less.

An acute gouty joint must not be mistaken for :—

(*a*) *Acute Rheumatism* (*vide* p. 60).

(*b*) *Septic Arthritis and Cellulitis.* The age and history of the patient, the absolutely sudden onset, and the absence of a septic focus will suggest gout ; clinically, however,

the resemblance may be very striking. The presence of definite fluctuation would suggest pus, as would rigors and sweating.

(ii.) *Chronic Gout.* This is the sequel of multiple acute attacks. The joints are deformed by massive deposits of sodium biurate, above which the skin may ulcerate exposing hard, chalky masses or else soft, pultaceous, gruelly material. The muscles waste from disuse, and gross deformities occur by over-action of certain muscle groups.

Chronic gouty arthritis can be mistaken for nothing else, for even in the earliest cases there will be the typical history of acute attacks.

(iii.) *Metastatic Gout.* Under this heading may be grouped all the diverse affections of various organs which occur, *par excellence*, in gouty persons and many of which may be distinctly benefited by treatment directed at the underlying gouty state. These manifestations are naturally important in so far as they may suggest the diagnosis of gout apart from an acute attack ; they will be considered briefly under the various systems which they attack.

(*a*) *The Eye.* Iritis may be of gouty origin. Retinal hæmorrhages are common in the gouty because of the associated renal and cardio-vascular changes.

Glaucoma has been recorded.

(*b*) *The Skin.* Eczema, pruritus, and boils may be mentioned, in addition to gouty tophi in the ears and elsewhere.

(*c*) *The Cardio-vascular System.* The heart is hypertrophied because of the renal fibrosis and high blood pressure. Fibroid and fatty changes in the myocardium are common, as also are attacks of angina pectoris.

(*d*) *The Respiratory System.* Laryngitis may be caused by the deposition of biurate in the cartilages of the larynx. Dyspnœa of a paroxysmal nature clinically resembling true bronchial asthma and not directly attributable to cardiac insufficiency may occur. A renal (uræmic) origin for this seems probable.

(*e*) *The Nervous System.* Neuritis is not uncommon, and hemiplegia from cerebral hæmorrhage is a frequent cause of death in gouty persons. Headaches, muscular cramps, and even gouty meningitis are described.

(*f*) *The Urinary System.* Arterio-sclerosis and red granular kidneys are the rule, with all the symptoms that attend them (*vide* pp. 446—448).

An intermittent glycosuria is common.

The association of urinary calculus and gout has long been recognised.

(*g*) *The Alimentary Tract.* Chronic pharyngitis and nasal catarrh are often attributed to gout.

Chronic dyspepsia, with acute exacerbations (bilious attacks) and obstinate constipation, are of frequent occurrence.

IV. RICKETS (RACHITIS).

Definition. A disorder of metabolism affecting young children, showing itself by erroneous bony development and general malnutrition and produced by a faulty food supply and defective hygiene.

Etiology. Both sexes are affected equally. Symptoms usually manifest themselves between the ages of six months and two years. Late rickets has been described in which the onset is delayed until the ninth year; such cases must be very rare.

The great majority of cases occur in infants that have been fed on artificial foods, though *very prolonged* breast feeding is said to favour the development of rickets.

The foods which are most frequently responsible for rickets are those in which there is a deficiency in both fat and proteid and consequently an excess of carbohydrate.

There is in addition an imperfect assimilation of lime salts.

(i.) **The general symptoms** are malaise and malnutrition; the child is restless and tender to the touch; it often screams when handled; it sweats at night, especially about the head, and kicks the bedclothes off. The complexion becomes dull and earthy, the appetite fails, and there is often diarrhœa and a tendency to vomiting. The muscular power is lost and the muscles themselves are shrunken and flabby. Convulsions are not uncommon, and there is a notable tendency to reflex spasms, such as laryngismus stridulus.

(ii.) **The Special Signs** are *Bony* and *Visceral.*

(*a*) *Bony Changes.* The head is large for the body; the fontanelle remains unduly patent; bosses form in the parietal and frontal regions. The squamous parts of the cranial bones may be thinned like parchment (craniotabes).

The milk teeth erupt late and are prone to decay. The thorax shows a deep groove running transversely from the lower end of the sternum to the anterior axillary line (Harrison's sulcus)—this causes the xiphoid and lower ribs to appear splayed out like an umbrella—while the sternum, as a whole, becomes depressed.

The ribs present a characteristic bony knob at the junction of the costal cartilages with the bony ribs (rickety rosary).

The spine often shows a curve with the concavity forwards.

The long bones show curves which are usually exaggerations of their natural curves.

Both knock-knee and bow-legs are common.

The epiphyses of the long bones are knobbly and thickened: this is particularly evident at the lower end of the femur and at the wrist.

The pelvis tends to fall in and assume a tri-radiate type.

(*b*) *Visceral Changes.* Bronchitis is both common and resistant to treatment in rickety children.

The abdomen is protuberant and enlarged; the liver and spleen are often greatly increased in size.

The blood shows an anæmia of " secondary " type.

The diagnosis of rickets is easy in advanced cases; the slighter forms may well be overlooked.

Congenital syphilis must be excluded, if necessary by a Wassermann reaction both of infant and mother, though, of course, the two diseases may co-exist.

Scurvy may be suspected where tenderness is very marked (*vide* p. 168). The special features of *Achondroplasia* (p. 173) should prevent this condition being mistaken for rickets.

Paraplegia of cerebral origin has sometimes been diagnosed in cases of rickets where the muscular weakness is unusually pronounced; but even if the rickety child cannot stand he can still kick his legs about freely when he is lying on his back—in other words, there is no paralysis.

V. ARTHRITIS DEFORMANS.

The synonyms for and varieties of this condition are so numerous, and accurate differentiation between them is so difficult, that considerable confusion may result in the mind of anyone studying the literature of the subject.

It is convenient, clinically, to recognise the two main groups—*Rheumatoid Arthritis* and *Osteo-arthritis*—but it must be clearly understood that no hard-and-fast line can be drawn between them and that often both conditions are present in the same person.

(a) **Rheumatoid Arthritis.** This disease is produced by the toxins of infective bacteria, or possibly sometimes by the micro-organisms themselves. There is always a focus somewhere in the body where these bacteria are settled and whence they discharge their poisonous products into the circulation; sometimes, indeed, it would appear that the bacteria themselves enter the blood-stream. Possible foci of this nature are—the gums and tooth sockets, as in pyorrhœa; the accessory sinuses of the nose; the naso-pharynx; the middle ear; the alimentary canal, and the genito-urinary tract.

Although the condition must be regarded as "infective" it is not "specific," since similar results appear to be obtained from the action of many different organisms, to most of which only a generic name (often some form of streptococcus) can be given bacteriologically. Rheumatoid arthritis usually starts before the age of 40, often in the third decade.

The characteristic lesions are *periarthritic* inflammations; the fibrous tissues around the joints are the structures principally affected; the essential articular elements often escape, but may be involved secondarily. More often than not there is no effusion into the joint, but, owing to the swelling round about it, the joint as a whole *looks* swollen and puffy and, of course, movement of it is painful. The spindle-shape of affected joints is best seen in chronic cases where, from disuse, wasting of the muscles above and below the joint has supervened. The smaller joints, such as the fingers and wrists, are generally attacked first, but the knees

and elbows are often involved also; the hips, shoulders, and spine more often escape.

Once started the disease tends to be progressive, and associated trophic changes are often conspicuous, such as glossy skin, fibrillation of the nails, and neuritis.

The disease may be acute in onset, and at first hardly to be distinguished from acute rheumatism except by its failure to react to salicylates; more often it develops insidiously, but is liable to subacute exacerbations.

(b) **Osteo-arthritis.** This disease partakes more of the nature of a degeneration than an infection; the articular structures themselves, especially the cartilages and synovial membranes, are principally affected. There may be much or little effusion. Sometimes there seems to be a marked reactionary and perverted overgrowth of the affected tissues, resulting in the so-called hypertrophic form with lipping of the cartilages, osteophytic formation, and exuberance of pulpy synovial membrane; in other cases the process is entirely atrophic, and creaking and grating in the affected joints are the principal features.

A Charcot's joint in a case of tabes may be considered analogous to the hypertrophic form of osteo-arthritis except for its painlessness and the greatly increased mobility, which contrasts sharply with the limitation of movement, or even fibrous ankylosis, of osteo-arthritis.

The victims of osteo-arthritis are older than those of rheumatoid arthritis; it is particularly common in women about the menopause. The larger joints are most concerned, such as the knees, hips, and shoulders; but no joint is immune, and the fingers, spine, and temporo-mandibular articulation are frequently affected.

Trophic changes, as in rheumatoid arthritis, with free perspiration of the affected parts, may be expected. Ulnar deviation of the hand is not uncommon, and can be distinguished from ulnar paralysis by the associated joint changes.

Certain special varieties of arthritis deformans may be mentioned :—

(i.) *Still's Disease.* This seems to be a form of *rheumatoid arthritis* occurring in children. One joint (usually a knee) is first attacked, but in a little time one or several more become

involved. At the same time the lymphatic glands throughout the body, but particularly those near the affected joints, and the spleen become enlarged.

This train of signs seems to point to an infective origin, since the glands and spleen in children are affected by lesser causes than are necessary to produce the same result in adults.

(ii.) *Acute Mon-articular Osteo-arthritis.* A variety of osteo-arthritis with pain, creaking, and grating, occurring suddenly in old people and strictly limited to one joint, nearly always a shoulder or a hip. Trauma is often responsible for this type.

(iii.) *Spondylitis Deformans.* Osteo-arthritis affecting the spine only. The spine becomes rigid and curved forwards, so that the patient's chin is approximated to his sternum, and when placed on his back his occiput is many inches off the bed. Pain in this form is often severe from involvement of nerve roots, and there is commonly an associated ascending degeneration of the posterior and anterior columns of the spinal cord with muscular atrophy and anæsthesia.

(iv.) *Spondylose Rhizomelique.* Osteo-arthritis of the spine, hips, and shoulders. In this variety the lesion is usually limited to one region of the spinal column, and nervous symptoms are slight or absent.

(v.) *Heberdens nodes* are bony outgrowths from the base of the distal phalanges of the fingers ; they are diagnostic of osteo-arthritis, and are significant inasmuch as the other manifestations of the disease are likely to be of *relative* mildness.

VI. FIBROSITIS (MUSCULAR RHEUMATISM).

Muscular pains of varying intensity may result from a toxic inflammation of the fibrous tissues between various muscle bundles. The tendency for these pains to be induced by cold and damp is well known.

The pathology of the condition is obscure, and though rheumatism is often blamed it is doubtful if it really has much to do with it except possibly in cases of torticollis. It seems more likely that in many cases the toxins have a

similar nature and origin to those which are responsible for rheumatoid arthritis, though a certain proportion of cases appear to be gouty.

The clinical features of fibrositis are :—

(i.) Pain in certain muscles, especially the lumbar muscles (lumbago), the intercostals (pleurodynia), the neck muscles (torticollis), and the shoulder muscles (omodynia).

(ii.) The pain, as would be expected, is worse on movement of the affected muscle, but also is often increased by warmth, as in bed ; it is localised to the affected part and does not radiate like the pain of neuritis.

(iii.) Tender points can often be made out in the affected muscles.

(iv.) A characteristic attitude, varying with the site of the lesion, which is the result of an endeavour to avoid the use of the affected muscles.

(v.) There is rarely pyrexia or constitutional disturbance.

Lumbago is nearly always bilateral, and not infrequently is accompanied by sciatica on one side. There is extreme pain on attempting to resume the erect posture after stooping. Lumbago must not be mistaken for stone in the kidney, which can be distinguished by the characteristic referred pains, the urinary changes, and, if necessary, by the X-rays.

Pleurodynia must be distinguished from pleurisy by the local tenderness and the absence of friction, or indeed of any pulmonary abnormality, if the patient can be induced to breathe deeply.

Omodynia usually has definite muscle tenderness and pain. There is no evidence of neuritis (*vide* p. 505), and the shoulder-joint can be shown to move quite freely if the affected muscles are supported. If there is any doubt about the shoulder-joint the X-rays must be used.

Fibrositis can be distinguished from neuralgia by the periodic nature of neuralgic pain, the absence of stiffness, and the fact that movement has little or no effect on it.

CHAPTER IV

SUNSTROKE AND CERTAIN INTOXICATIONS

I. SUNSTROKE (INSOLATION).

The clinical effects of insolation may result from the direct exposure to the rays of the sun or from exposure to high temperatures in the shade. A considerable proportion of cases first manifest symptoms in the night. An impure atmosphere with moisture is more apt to produce sunstroke than a higher temperature with a pure, dry atmosphere. Sunstroke is most common when the thermometer registers from 90° to 110° F., but any arbitrary limit is impossible.

The clinical effects of high atmospheric temperatures may be considered under two headings :—

(a) Syncope from Exhaustion. This is well seen in soldiers on the march, stokers in the tropics, etc.

The condition is essentially one of syncope ; the patient is cold and pale and the pulse is feeble and fluttering. Recovery generally ensues in a few hours and there are no evil consequences. Sometimes high fever develops (thermic fever) when the patient has apparently recovered from the syncope, and this may terminate fatally or prove so intractable as to necessitate removal to a colder climate. More commonly the fever gradually subsides and a good recovery is made, though, even so, a change of climate is often advisable.

(b) Sunstroke Proper (Coup de Soleil). This usually results from the effect of the sun's rays on the back of the neck and head. The body temperature rises to 106° to 108° or even 110° F. and profound asphyxial symptoms occur.

Premonitory symptoms, such as restlessness, headache, gasping respiration, nausea, and a sense of dread may in cases of continued exposure to a high temperature precede the actual stroke for a variable period. These are followed by unconsciousness, stertor, lividity, and cyanosis. On the

other hand, the "stroke" may occur without warning. Death ensues if means are not taken to lower the temperature in from twelve to forty-eight hours.

Relapses are not infrequent, and persons who have had sunstroke of this type are often particularly intolerant of even moderate heat and humidity for many years afterwards. Meningitis occasionally develops in the course of insolation, and a goodly number of those who recover from a severe sunstroke develop later signs of permanent cerebral changes, such as epilepsy, loss of memory, deafness, blindness, paralysis, dementia, or even mania.

II. ALCOHOLISM.

The ordinary manifestations of moderate drunkenness do not need description here. Alcoholism will be considered under three headings :—

(i.) Acute Alcoholism.

(ii.) Chronic Alcoholism.

(iii.) Delirium Tremens.

(i.) **Acute Alcoholic Poisoning.** The result of an overdose of alcohol may produce a condition of stupor or unconsciousness which it is important to diagnose correctly. There may be a history of recent excess, but too much attention should not be paid to this, for the temporary rise in blood pressure induced by alcohol may suffice to rupture a cerebral artery ; in a like manner the smell of alcohol in the patient's breath is of some value, but far from conclusive, since the administration of brandy is usually the first step taken by the laity in any form of sudden illness.

The following points may prove of importance :—

(*a*) The breathing is slow and deep, but rarely stertorous.

(*b*) The pupils are equal and dilated.

(*c*) The unconsciousness is hardly ever so deep that the patient cannot be temporarily roused by shouting or pinching the skin of the neck.

(*d*) There is no evidence of paralysis.

(*e*) There are neither convulsions or albuminuria.

If narcotic poisoning is suspected, or indeed in any case

where the presumptive evidence is against a cerebral lesion, it is advisable to wash out the stomach. The obvious presence of large quantities of alcohol in the wash-out is a strong point, but the material should also be tested for poisons, since morphia or chloral, etc., may have been taken while under the influence of alcohol.

In all cases some of the stomach wash-out should be kept till the diagnosis is absolutely clear.

The speedy return to consciousness after free gastric lavage is striking in cases of simple alcohol poisoning.

Some unfortunate persons suffer from severe toxic gastro-intestinal disturbance with extreme prostration as the result of alcohol without ever showing any of the more usual signs of inebriety. In such cases the vomiting sets in early, but instead of ceasing when the stomach is empty it continues at very frequent intervals, perhaps for thirty-six to forty-eight hours, until the patient is seriously collapsed by the loss of fluid and exhausted from the repeated strain.

The differential diagnosis of alcoholic stupor, from narcotic poisoning, uræmia, and cerebral lesions is discussed on p. 460.

(ii.) **Chronic Alcoholism.** The effects of chronic alcoholic excess (even though the subject rarely or never gets drunk) are generally fairly obvious to those who are brought into contact with him. The following signs may be mentioned :—

(*a*) *General Appearance.* Shifty manner, yellow conjunctivæ, tremulous lips, red nose, acne rosacea, or rhinophyma.

(*b*) *The Digestive System.* Flabby, furred tongue ; nasty taste in the mouth ; chronic gastritis ; morning vomiting of bile-stained mucous ; no appetite for breakfast ; looseness of the bowels ; piles, and even positive evidence of cirrhosis of the liver.

(*c*) *Mental Symptoms.* The mental and moral qualities may change completely : the patient becomes untruthful and unmoral ; he loses his memory and power of concentration ; he may be exalted with grandiose ideas, or he may be depressed to the verge of melancholia. He is usually suspicious of his family and friends, while delusions

either transient or permanent are not infrequent, and may be so extreme as to necessitate certification.

(*d*) *Nervous System.* Beyond occasional peripheral neuritis (*vide* p. 508), there is usually no evidence of organic disease ; but tremor of the tongue, lips, and extremities is particularly common.

(*e*) *The Kidneys.* Chronic interstitial nephritis is often the result of chronic alcoholism.

(*f*) *The Heart* commonly shows fibro-fatty change.

(iii.) **Delirium Tremens** is only seen in the chronic alcoholic ; in such it may follow on some extra indulgence, on any intercurrent accident (*e.g.*, broken leg) or acute illness (*e.g.*, pneumonia), on any profound mental shock, or on the abrupt withdrawal of all forms of alcohol.

The first sign is tremor, with restlessness, mental depression, and insomnia. In about twenty-four hours there is noisy delirium ; the expression is anxious, the eyes are bright and glistening, and in a short while definite hallucinations appear. The patient often imagines he is in danger of being done to death and sees curious and imaginary animals about the room or crawling on the bed. He naturally becomes extremely violent, and restraint is frequently necessary. The question of restraint is important ; it should never be employed unless absolutely necessary, and it is very striking how often it is possible to keep a patient comparatively quiet for a long time by sitting with him and talking to him and even attempting to reason with him.

The exhaustion produced by struggling against forcible restraint often proves fatal, especially in the cases following illness or accident.

An interesting feature of many cases of delirium tremens is the tendency for the hallucinations to be closely connected with the daily life of the individual—for example, a bus-driver will imagine himself to be driving his bus along its accustomed route, and if he is given a pair of reins tied on to the end of his bed will often drive away contentedly for hours.

An ordinary case of delirium tremens wears itself out in about three nights. A high temperature is a bad sign. There is a great tendency for pulmonary affections, such as

tuberculosis or broncho-pneumonia, to develop in those who are chronic alcoholics. Apical pneumonia is peculiarly liable to be associated with delirium tremens. In all cases the lungs should be carefully and repeatedly examined.

The differential diagnosis of Chronic Alcoholism from General Paralysis is indicated on p. 550.

III. MORPHINISM.

Enormous quantities of opium or its alkaloid morphia may be taken by those addicted to the habit. Over 300 grains a day were taken by De Quincey.

A morphinist appears older than his years : his complexion has a pale, dull, earthy look ; his eyes are sunk (the pupils may be small, but this depends rather upon how recently a dose has been taken; in the intervals they are often dilated) ; his expression vacant or dreamy, and his body emaciated. When not under the influence of the drug the patient is depressed, irritable, and restless ; he has a great sense of mental and bodily weariness, and as the time for a dose draws near he has acute abdominal pain, nausea, and vomiting. The effect of a sufficient dose is marvellous ; there is an almost instantaneous recovery of mental and physical powers while the patient looks years younger for the time being.

One of the most significant features of the morphia habit is the effect on the moral qualities of the victim ; he is always untruthful and furtive, especially so in relation to his vice, but often in all other matters as well. It is no uncommon thing for a patient voluntarily to present himself for treatment in a nursing home with large quantities of the drug secreted about his person.

Itching of the dry skin is common, and this causes repeated rubbing of the nose, which has been emphasised by some writers as a diagnostic feature.

The diagnosis of morphinism is not difficult provided sufficient observation of the patient can be employed.

The appearance, the mental character, and the alternations of vigour and apathy are sufficiently striking.

Confirmation may be afforded by the discovery of numerous puncture marks, especially on the left forearm or right thigh.

The cocaine habit presents a very similar clinical syndrome to that described under "Morphinism." Itching is an even more constant feature. The pupils may be widely dilated, and certainly are not contracted.

IV. LEAD POISONING (PLUMBISM).

Lead poisoning may be acquired by contamination of the water supply, in the course of various industrial occupations, such as painting, plumbing, type-setting, etc., or wilfully (as by eating diachylon plaster) to produce abortion.

The following are the principal features of plumbism :—

(i.) *Anæmia and Cachexia.* There is a severe anæmia, of which the features are :

(*a*) Great diminution in red cells.

(*b*) Corresponding decrease in hæmoglobin, so that the colour index is not low.

(*c*) Basophile stippling of the red cells.

(*d*) Poikilocytosis.

There is no leucopenia, and the blood does not show megaloblasts so constantly as in pernicious anæmia. A certain number of normoblasts are nearly always to be found.

(ii.) *Blue Line on the Gums.* This is a punctate or intermittent line on the edge of the gums, best marked on the papillæ between the teeth. It does not appear readily in persons who have scrupulously clean mouths, and on the other hand must not be confounded with the continuous dirty, grey-blue line of those who have advanced pyorrhœa alveolaris.

(iii.) *Colic and Constipation.* A true colicky pain, often extremely severe, of maximum intensity at the umbilicus, but often to be traced along the course of the colon. Constipation is generally intractable.

(iv.) *Encephalopathy.* This may vary from simple headache to convulsions, delirium, and even coma.

Optic neuritis may be present. Delusional insanity has been recorded.

(v.) *Gout and granular kidney* are particularly frequent in those who suffer from plumbism.

(vi.) *Miscarriages* are the rule in lead poisoning.

(vii.) *Neuritis* (*vide* p. 509).

The diagnosis of lead poisoning rests on a judicious analysis of such of the preceding signs as may be present in any case. The history is very important except in cases in which lead has been taken to procure abortion.

The differential diagnosis of such individual signs as neuritis, colic, etc., is dealt with elsewhere under these headings. In cases of doubt, lead should be looked for in the urine, though the discovery of lead is not proof positive of lead-poisoning.

V. CHRONIC ARSENICAL POISONING.

This may be acquired by workers in certain trades, such as wall-paper makers, workers in artificial flowers, etc., but the Board of Trade Regulations have largely minimised these risks. It may also be acquired by the contamination of food supplies, as in the recent outbreak at Manchéster due to contaminated beer, by overdosage in medicine and, lastly, it may be administered with homicidal intent.

The Symptoms are :—

(i.) Gastro-intestinal disturbances, such as colicky pains, nausea, vomiting, and diarrhœa.

(ii.) Flushing of the skin, with puffiness of the eyes.

(iii.) Conjunctivitis and catarrh of the respiratory tract.

(iv.) Numbness and tingling of the extremities and neuritis (*vide* p. 509).

(v.) Pigmentation and thickening of the skin (keratosis), especially of the palms of the hands and the soles of the feet.

(vi.) Epithelioma has been described as a late development of the keratosis.

The diagnosis of chronic arsenical poisoning depends on the signs enumerated above and a history of exposure to the influence of the poison.

The danger of exposing patients with such chronic diseases as lymphadenoma or pernicious anæmia to the risk of arsenical poisoning in the course of treatment should always be borne in mind.

Acute arsenical poisoning gives the signs of an acute

irritant poison ; abdominal pain, vomiting, diarrhœa, tenesmus, and collapse ; in addition there are muscle cramps and sometimes convulsions or paralysis.

VI. CHRONIC MERCURIAL POISONING.

This occurs in those who work in mercury or in mercury mines.

The outstanding sign is a rather coarse tremor, at first volitional, later constant, but always increased by "intention." The tremor starts in the face and tongue, and then spreads to the upper limbs and finally to the legs. It may be so severe as to prevent speech or walking.

A history of exposure to mercury vapour can always be obtained ; apart from this the diagnosis must be made from paralysis agitans and disseminated sclerosis.

In *paralysis agitans* the tremor can, at first, be controlled by will power, and continues when the patient is at rest. The tongue is not often affected, and certainly not in the early stages. The characteristic rigidity of paralysis agitans is not met with in mercurialism (*vide* also p. 570).

In *disseminated sclerosis* there is nystagmus and some evidence of an upper motor neuron lesion (*vide* p. 552).

PART III

CHAPTER I

DISEASES OF THE CARDIO-VASCULAR SYSTEM AND PERICARDIUM

I. THE NORMAL HEART.

The boundaries of the heart as outlined on the front of the chest in a healthy individual may be represented by the following four lines :—

The upper border is formed by a line joining the two second costal cartilages and extending about 1 inch to either side of the sternum. The right border reaches from the right extremity of the upper or base line to the chondro-sternal junction of the sixth rib on the right side. This border is slightly convex towards the right, the greatest convexity being reached at the level of the fourth rib, at which spot the right limit of the heart is about $1\frac{1}{2}$ inches from the middle line. The lower border of the heart is represented by a line joining the sixth right chondro-sternal junction with the apex beat. This border merges with the liver dulness and is scarcely to be distinguished by percussion. The left border runs from the left extremity of the base line to the apex beat ; it is definitely curved with its convexity outwards. The apex beat is situated in the fifth left space $\frac{1}{2}$ inch internal to the mid-clavicular line and about $3\frac{1}{2}$ inches from the mid-sternal line. The area thus mapped out is known as the deep cardiac dulness. It consists almost entirely of right ventricle and right auricle except for a narrow strip of the left ventricle towards the left and the left auricular appendix, which is situated behind the inner portion of the third left chondral cartilage.

The greater proportion of the heart is separated from the chest wall by the lungs ; that part which lies directly beneath the thoracic parietes corresponds to the area of

superficial cardiac dulness. This is a small area the right border of which is the mid-line of the sternum, whilst the upper limit is usually about the level of the upper border of the fourth rib and the left border follows a line convexly from the fourth chondro-sternal junction to the apex.

The sounds of the heart are two in number: the first sound is formed partly by the muscle sound of the contracting ventricles and partly by the tension of the auricular-ventricular valves; the second sound is produced entirely by the closure-tension of the aortic and pulmonary valves. The first sound is best heard at the apex; the second sound at the base.

In children the pulmonary second sound is louder than the aortic second sound; in adults the converse usually obtains.

Cardiac systole extends from the commencement of the first sound to the commencement of the second sound, diastole from the commencement of the second sound to the commencement of the first. The systolic interval is shorter than the diastolic, the first sound is of longer duration and of less tension than the second.

The four cardiac valves are grouped together within a very small area; it is therefore convenient to listen at places to which the individual valve sounds are best conducted, when it is desirable to investigate the sound generated at a particular valve. These auscultation areas are known by the names of the valves whose sounds are conducted thither. Thus, aortic valve sounds are best heard at the aortic area, which is the second right costal cartilage; the pulmonary valve sounds at the inner end of the third left space; the mitral valve sounds at the apex, and the tricuspid valve sounds in the fifth or sixth left space close to the sternum.

The cardiac impulse can be seen or felt in the majority of healthy people as a circumscribed systolic thrust in the neighbourhood of the heart's apex, but it should not occupy a greater area than one square inch. The cardiac impulse does not really correspond to the anatomical apex of the heart, but to a spot situated at or about the junction of the middle and lower thirds of the left ventricle.

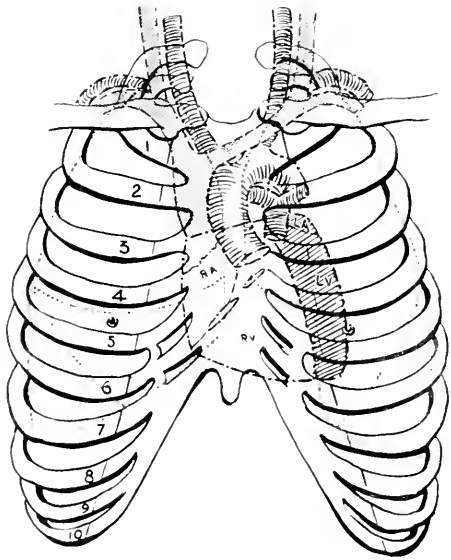


Fig. 40.—Diagram to shew the relation of the Heart and Great Vessels to the Chest Wall and to the Lungs. The dotted red lines indicate the surface marking of the Lungs and their inter-lobar septa.

II. THE SIGNIFICANCE OF DISPLACEMENT OF THE APEX BEAT AND ALTERATION IN THE CHARACTER OF THE CARDIAC IMPULSE.

In children the heart is relatively wider laterally than in adults, hence the apex beat is found in the fourth space instead of the fifth and often slightly external to the mid-clavicular line. Where there is marked emphysema, or where there is much fat or thick musculature, it may be impossible to locate the apex beat, a condition which may also be produced by a very feeble heart, œdema of the chest wall, and sometimes by pericardial effusion. The position of the apex may be modified by various intra-thoracic conditions without there being any cardiac lesion. A pleural effusion or pneumo-thorax will displace the heart towards the sound side, and a pulmonary neoplasm may have the same effect.

Fibrosis of a lung will pull the heart towards the affected side, and if the fibrosis is principally in the left upper lobe the apex may be drawn up into the fourth space. Various intra-abdominal conditions, such as subphrenic abscess, ascites, etc., may push up the diaphragm and so displace the heart upwards.

Hypertrophy of the left ventricle will displace the apex downwards and slightly outwards ; hypertrophy of the right ventricle does not tend to produce much alteration of the position of the apex, since the effect is rather to rotate the heart on its own axis, thus pushing the left ventricle away from the chest wall and causing the cardiac impulse to be formed by the right ventricle internal to the mid-clavicular line. Where there is very great hypertrophy of the right ventricle the apex may be displaced considerably to the left, and a heaving impulse is also present below the costal margin. Hypertrophy of both ventricles displaces the apex downwards and outwards.

Both cardiac hypertrophy and dilatation produce an increase in the area of the cardiac impulse ; but in the former case the thrust is heaving and powerful, whereas in the latter

it is weak, indeterminate, and diffuse. An extensive heaving impulse, easily felt below the costal margin, is the rule in mitral reflux : a short, sharp slappy impulse is one of the most important signs of mitral stenosis.

Adherent pericardium gives rise to a diffuse wavy impulse, and often to systolic retraction of the intercostal spaces in addition. Pericardial effusion may occasionally push the impulse up into the fourth space.

Pulsations not caused by the Apex Beat. Pulsation in the neighbourhood of the manubrium is suggestive of aortic aneurysm ; it may also sometimes be met with in cases of mediastinal new growth or empyemia. Pulsation in the third and fourth left spaces may be found in healthy people after violent exercise or in the forcibly acting hearts of nervous people ; more generally, however, it is met with in cardiac dilatation, fibrosis of the left lung, or severe anæmia.

III. THE SIGNIFICANCE OF ALTERATIONS IN CARDIAC DULNESS.

The superficial cardiac dulness may be increased by the enlargement of the heart, either from hypertrophy or dilatation, which pushes aside the lung fringes, by retraction of the lung from collapse or fibrosis, and by pericardial effusion.

Theoretically more valuable information will be gained by mapping out the area of deep than of superficial cardiac dulness, and if it could always be accurately delimited this would be true practically. Unfortunately, however, the deep dulness cannot always be made out. In such cases we may be compelled to rely on the superficial area.

The borders of the heart should be investigated from two points of view—first, abnormalities in position, and, secondly abnormalities in shape.

Enlargement of the left ventricle displaces the left border of the heart outwards to a variable extent, but it also causes it to be continued downwards even to the seventh space.

Enlargement of the right ventricle increases the area of

deep cardiac dulness a variable distance to the right of the sternum, and also produces a powerful heaving impulse below the xiphoid and left costal margin. The right border, which is formed by the right auricle, may assume a greater convexity from enlargement of this chamber. At the same time the left border of the heart may be displaced outwards, but will usually maintain its ordinary shape. Enlargement of both ventricles will produce a combination of these features.

Enlargement of the left auricle, as seen in some cases of mitral disease, will produce an increase upwards of the cardiac dulness to the left of the sternum ; the line may have a marked convexity upwards and to the left.

Enlargement of the right auricle will affect the right border of the heart and will not be met with apart from enlargement of the right ventricle, except possibly in those rare cases when tricuspid stenosis is the most prominent valvular lesion.

An increase of dulness to the right of the sternum in the first and second spaces is suggestive of aortic aneurysm or of dilatation of the ascending aorta.

A triangular area of dulness with the base downwards, and particularly with the right border showing *no convexity*, is suggestive of pericardial effusion.

A very marked increase in the area of deep cardiac dulness is caused by adherent pericardium.

In percussing out the areas of cardiac dulness it is well to remember that the finger should be placed firmly on the chest wall and parallel to the assumed direction of the line that is being investigated and that percussion should be light yet firm, should be performed with a free wrist and should not be performed alternately on rib and interspace.

Præcordial bulging, apart from bony deformities, such as rickets or spinal caries, is commonly due to aortic aneurysm or to new growth. It may be caused by extreme cardiac enlargement, especially if such date from childhood. Lastly, it may occur in large pericardial effusions, particularly in young patients.

IV. SIGNIFICANCE OF ALTERATION IN THE CARDIAC SOUNDS AND THEIR SPACING.

In health the cardiac rhythm should be regular, even though the rate is greatly accelerated by violent exercise. In young children, however, the rhythm is often habitually irregular, but this does not necessarily indicate any pathological condition (*vide* p. 240).

Since the first sound is partly generated in the ventricular muscles, undue prominence of it must mean an increase in the activity of one or other ventricle, and betokens hypertrophy from one or another cause.

The first sound in hypertrophy may be described as low-pitched and muffled. When, however, the hypertrophied ventricle is beginning to be unequal to the strain imposed upon it, the first sound at first becomes rather indistinct, and when there is distinct dilatation the muscle-sound appears to be lost and the first sound becomes short and sharp like an ordinary second sound.

The short, sharp first sound of mitral stenosis is not due to ventricular dilatation, but to the forcible contraction of the left ventricle before it is completely filled.

A reduplication of the first sound is taken to mean asynchronism of the two ventricles, with the result that the tricuspid and mitral valves do not become tense at the same time.

Intermission of the first sound every few beats may occur as a constant feature in certain perfectly healthy hearts; it is, however, sometimes of serious significance where there is cardiac fibrosis (*vide* p. 246).

Accentuation of the second sound indicates high pressure in the systemic or pulmonary arteries respectively—*e.g.*, the ringing aortic second sound of chronic interstitial nephritis and the accentuated pulmonary second sound of mitral stenosis. So long as the second sound is thus accentuated the ventricles are capable of performing the work required of them. When one of the ventricles begins to be unequal to its task, or when from force of circumstances one ventricle is acting relatively much more power-

fully than the other, the second sound at the base is likely to become reduplicated, and in severe dilatation the second sound gets shorter and weaker until in some cases it may be inaudible.

Apparent reduplication of the apical second sound when there is no reduplication at the base is a common feature in mitral stenosis, and is in reality a mid-diastolic murmur which simulates a doubling of the second sound.

The spacing of the heart sounds may be altered in two ways. Systole may be prolonged at the expense of diastole till both are of equal length. This "tic-tac" rhythm, as it is called, suggests a heart which is no longer capable of dealing with some increased strain which it has been called upon to face. It is met with in cases of arterio-sclerosis when the limit of hypertrophy has been reached. In other cases both systole and diastole may be greatly shortened, causing the sounds to appear unduly close together (*bruit de galop*); this condition is significant of grave myocardial asthenia.

V. THE SIGNIFICANCE OF CARDIAC MURMURS AND THRILLS.

A. **Murmurs.** A murmur may be defined as an endocardial sound generated at one of the cardiac valves and produced by an abnormal relationship between that valve and its neighbouring structures, or at some undue constriction in the tubular arrangement of the cardio-vascular system resulting from some congenital defect in the development of the heart and vessels. In the majority of cases a thrill is a palpable murmur.

For the purpose of diagnosis murmurs are divided into two main groups—(*a*) Functional, and (*b*) Organic.

(*a*) *Functional Murmurs* are met with in conditions of anæmia and debility; they are not associated with any impairment of valvular function or with any congenital defect. They are always systolic in time and originate at either the aortic or pulmonary valves. The most common

hæmic murmur is due to dilatation of the conus arteriosus, thus causing a *purely relative* stenosis of the pulmonary valve. The murmur is usually soft, but may be distinctly vibratory ; its point of maximum intensity is at the inner end of the third left space. It is commonly conducted up towards the left clavicle across to the aortic area and down towards the apex. Sometimes it is limited to the pulmonary and aortic bases, and sometimes it can only be heard just to the left of the sternum in the third, fourth, and fifth spaces.

More rarely a hæmic murmur is generated at the aortic valve ; it is then supposed to be due to dilatation of the upper part of the left ventricle immediately below the aortic ring, thus producing a *purely relative* stenosis of the aortic valve. The point of maximum intensity of this murmur is at or about the second right costal cartilage. It is conducted across to the pulmonary area, up towards the right sterno-clavicular articulation, and to a varying extent down towards the apex. Hæmic murmurs are most distinct when the patient is lying down ; they may sometimes disappear completely when the erect position is assumed.

It is customary to describe as functional systolic murmurs limited to the mitral or tricuspid areas and occurring in people who are suffering from toxic conditions, anæmia, and the like. Such murmurs indicate slight dilatation of the mitral and tricuspid rings resulting from æsthenia or toxic myocarditis ; as the patient recovers so do the murmurs disappear, but while they last they indicate a true though transient inadequacy of the auriculo-ventricular valves.

(*b*) *Organic Murmurs* indicate structural deformity of the valve concerned. Each valve may be affected in two ways ; it may be obstructed or it may be incompetent and so permit of regurgitation through its orifice. In many cases both incompetence and obstruction are present at the same time.

By estimating the time of the cardiac cycle occupied by the murmur or murmurs, and also their points of maximum intensity and lines of conduction, it is possible to state with considerable accuracy what valves are affected and what is the nature of their lesions. With regard to the

determination of the extent or severity of the particular lesions, murmurs by themselves are of little value; the loudest murmurs may be produced by the slightest lesions.

The following list will indicate the particular murmurs associated with the different forms of valvular disease :—

(i.) Aortic Reflux. (*a*) A diastolic murmur, usually soft and blowing, accompanying or replacing the second sound, and best heard at the aortic cartilage or over the middle of the sternum between the second cartilages, or sometimes in the pulmonary area or down the left border of the sternum. The lines of conduction are across to the pulmonary base down both sides of the sternum and to the apex. (*b*) A presystolic aortic murmur is often heard at the mitral area (Flint's murmur). This is due to the impinging of the refluent blood-stream through the aortic valve on to the anterior curtain of the mitral valve, thus producing a slight obstruction at the mitral valve. (*c*) A systolic murmur accompanying the first sound and of maximum intensity at the aortic area is very frequently heard in cases of aortic reflux. It exactly resembles the murmur of aortic stenosis, but in the majority of cases is due to thickening or roughening of the aortic cusps and not to true stenosis.

(ii.) Aortic Stenosis. A systolic murmur best heard at the aortic area and conducted up into the vessels of the neck. It is often to be heard behind between the left scapula and the mid-line. This murmur accompanies or replaces the first sound and is usually rough and vibratory. It must be remembered that an aortic systolic murmur is a very common murmur, but that aortic stenosis is a rare condition. This apparent anomaly is accounted for by the fact that dilatation of the first part of the aorta and slight atheroma of the aortic valves are both common conditions and will both produce a systolic murmur.

(iii.) Mitral Reflux. A soft blowing systolic murmur best heard at the mitral area, conducted out into the axilla, and often also audible between the angle of the scapula and the vertebral groove behind.

(iv.) Mitral Stenosis. (*a*) A rough presystolic murmur running up to the first sound, best heard just internal to the apex and localised to this neighbourhood. (*b*) A murmur

occupying the whole or any part of diastole, but most frequently a short mid-diastolic murmur. (*c*) A systolic mitral murmur, poorly conducted and not heard behind (Graham Steel's murmur).

(v.) PULMONARY REFLUX. A diastolic murmur of maximum intensity at the pulmonary area and conducted down the edges of the sternum. This lesion is extremely rare.

(vi.) PULMONARY STENOSIS. A rough systolic murmur best heard at the pulmonary area and conducted up towards the left clavicle. This lesion is extremely rare except as a congenital deformity.

(vii.) TRICUSPID REFLUX. A soft systolic murmur, often inaudible, best heard at the tricuspid area and conducted out for a short distance only towards the apex.

(viii.) TRICUSPID STENOSIS. A presystolic murmur localised to the tricuspid area. Tricuspid stenosis does not occur apart from mitral stenosis, and its recognition is likely to be obscured by the predominant signs of the latter condition.

Valvular lesions are often combined, in which case double or multiple murmurs will result. The most common double lesions are :—

(1) Mitral Stenosis and Reflux.
(2) Aortic Reflux and Mitral Reflux.
(3) Aortic Reflux and Aortic Stenosis.

The auriculo-ventricular valves are liable to passive dilatation consequent upon enlargement of the heart produced by other lesions, or by hypertrophy consequent upon increased peripheral resistance. This condition produces the blowing systolic murmur, indicative of regurgitation through the valve which is so affected. In children there is also a diastolic mitral dilatation murmur. Common examples of passive dilatation are :—

(1) Mitral dilatation secondary to aortic valve disease or to hypertrophy from arterio-sclerosis.

(2) Tricuspid dilatation secondary to mitral diseases or to aortic and mitral disease, or to hypertrophy of the right ventricle consequent upon a persistent increase in the pulmonary blood tension in cases of emphysema.

Arterial Murmurs not conducted from the heart may occasionally be met with in anæmia. The most likely

murmur from this cause is a soft systolic bruit in the left subclavian artery.

Where the aorta is congenitally narrowed at the level of the ductus arteriosus (coarctation of the aorta) a systolic murmur may be heard over the manubrium and also just to the left of the vertebral column behind.

Aneurysms may produce a systolic murmur or occasionally a double murmur. The murmurs are heard best over the aortic area and manubrium, and are not conducted down the sternum. They are only likely to be audible when the aneurysm is of the ascending aorta. The systolic murmur by itself is of no value in the diagnosis of aortic aneurysm.

Venous Murmurs (*bruit de diable*) in the great veins of the neck are common in anæmia, but are by no means restricted to this condition. They are modified by pressure with the stethoscope, and are heard as a continuous buzzing sound varying in intensity with the respiration.

Exocardial Sounds must not be mistaken for true murmurs. The most likely sources of error are :—

(i.) *Pericardial Friction.* This may be mistaken for a double aortic murmur. It is, however, not conducted, as is the aortic murmur, along the lines of the blood-stream ; it is more scratchy in character, more superficial, and can sometimes be modified by firm pressure with the stethoscope ; further, it is continuous.

(ii.) *Pleuro-pericardial Friction.* This is commonly heard at the upper part of the left border of the heart ; it is modified by pressure and also by cessation of respiration.

In conclusion we would emphasise that though the presence of a cardiac murmur indicates either a blood dyscrasia or a cardiac lesion and the study of the murmur shows which of these conditions is present, nevertheless in the latter case the fact that there is a murmur is no indication that the heart is not thoroughly competent to do the work required of it and that many other factors must be considered, both symptoms and signs, before condemning a patient to invalidity merely because a cardiac murmur happens to be present.

B. Thrills. The most common thrill is the presystolic or diastolic thrill felt at the apex in cases of mitral stenosis.

More rarely there is a systolic thrill in mitral regurgitation.

A systolic thrill at the aortic area and transmitted to the arteries in the neck is suggestive of aortic stenosis, and similarly a systolic pulmonary thrill is indicative of pulmonary stenosis.

A diastolic thrill following the lines of conduction of an aortic diastolic murmur is not very unusual in cases of aortic reflux.

A soft, tickling, wavy thrill is often present during late diastole over the diffuse impulse of adherent pericardium.

A systolic basal thrill may occasionally be produced by an aortic aneurysm.

VI. THE PULSE AND BLOOD PRESSURE.

(i.) **The Pulse.** Three fingers should be used for examining at the same time the pulse and the quality of the radial arteries ; the *frequency* of the pulse and its *regularity*, both of force and rhythm, are readily ascertained. *The quality* of the pulse is made up of three factors—namely, the size, the tension, and the mode of expansion. Considerable experience is necessary for the correct estimation of these qualities. The tension and the mode of expansion are estimated by gradually obliterating the pulse with the topmost finger. If this is found to be impossible it is due to a free anastomosis with the ulnar artery, and this return of blood must be prevented by exercising the pressure with the lower-most finger. When the pulse has been obliterated changes in the arterial wall can be appreciated by rolling the empty vessel beneath the middle finger.

The frequency of the pulse is increased by exercise and also by fever, the ordinary rate of increase being ten beats per minute for every degree rise in temperature. When the heart is extremely dilated many of the systolic contractions may be so feeble as never to reach the wrist ; when there is the slightest possibility of this being the case the radial frequency should be carefully checked by auscultation of the cardiac impulse. Rapidity of action combined with irregularity are significant of cardiac insufficiency, but they may also occur as neurotic features of hearts the valves and

muscles of which are perfectly healthy and also in condition of chronic toxæmia.

The size of the pulse is very variable ; it is large in febrile conditions, in some cases of increased peripheral resistance, and often when there is aortic reflux. It is small when the heart is failing and in the valvular lesions of aortic and mitral stenosis.

The pulse tension is high—that is to say, the wave is not readily obliterated—when from any cause the peripheral resistance is increased either by alteration in the quality of the blood or by arterial spasm, or when the heart is hypertrophied. A pulse that is at the same time small, hard, and rapid (wiry) is significant of peritonitis. A pulse of low tension—that is to say, one that is readily obliterated—is significant of myo-cardial debility, relaxation of the arterioles, or any grave debilitating disease such as enteric fever or chronic pulmonary tuberculosis.

The dicrotic pulse is produced by an abnormal increase in the dicrotic wave; this causes an apparent doubling of the pulse and is an expression of extreme low tension. It is most commonly observed in enteric fever. The dicrotic notch in a healthy pulse is produced by the elastic recoil of the aortic walls at the commencement of diastole. Since the aortic valves are closed at this time, a secondary impulse is conveyed to the blood in the arteries. For the production of a dicrotic pulse a short, feeble systole and a low peripheral resistance are requisite.

For the proper appreciation of the mode of expansion of a pulse it is necessary to understand the normal component parts of the pulse wave (Fig. 41). For this purpose the tracings made by the sphygmograph are of value. The normal pulse tracing shows an uninterrupted and nearly vertical upstroke or percussion wave and a gradually sloping downstroke on which are seen two definite irregularities. The upper irregularity is produced by the tidal wave which represents the actual blood pressure within the vessel. The fact that this is lower than the primary or percussion wave is due to the momentum imparted to the lever by the ventricular systole carrying the pointer too high. The lower irregularity or dicrotic notch corresponds

to the closure of the aortic valves ; the rise just after this notch is called the dicrotic wave, and is due to the elastic recoil of the aorta at the beginning of diastole following its distension at the moment of ventricular contraction.

It will thus be seen that cardiac systole is represented on the pulse tracing as the line between the base line and the tidal notch, while the curve from the tidal notch back to the base line represents the cardiac diastole.

A pulse of normal or low tension is described as a katacrotic pulse—that is to say, the percussion stroke ascends to its highest point in an unbroken line. Where, however, there is a mean high tension, as from greatly increased peripheral resistance and a strongly acting heart, the pulse

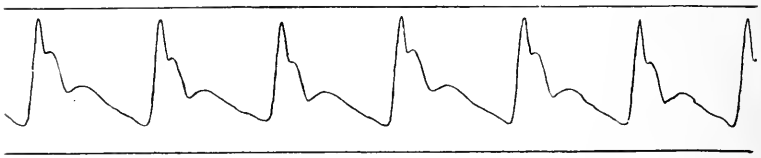


Fig. 41.—Tracing to show the component parts of the normal Radial Pulse Wave.

may become anacrotic and the tidal wave may be as high or higher than the percussion wave ; the result of this is a plateau formation on the pulse tracing. This plateau formation is also seen in aortic stenosis.

The pulse of low tension shows an exaggeration of the dicrotic wave, while the tidal wave is slight or absent.

The alternation of large and small beats (bigeminal pulse) in all probability is due to the occurrence of an extra ventricular contraction following the regular beat, and is likely to be caused by undue excitability of the ventricles (*vide* p. 244).

More marked irregularity of the pulse in which no two consecutive beats are alike is commonly seen in the later stages of organic heart lesions either muscular or valvular (*vide* Auricular Fibrillation, p. 251).

The alterations in the length of the systole so commonly met with in children are probably due to retardation impulses of vagus origin (*vide* p. 240).

The pulsus paradoxus consists in an enfeeblement, or even

disappearance, of the radial pulse during deep inspiration. It is present in some cases of adhesive mediastinitis, adherent pericardium, and even of large pericardial effusion, and is presumably due to hampering of an unusually weak cardiac systole by the increased intra-thoracic pressure of inspiration.

The features of the pulses belonging to the different valvular diseases will be described under the lesions with which they are connected.

Venous Pulse. As the right auricle contracts there is normally a suction wave manifested in the great veins of the neck; a ventricular systolic pulsation in the neck veins is, however, diagnostic of tricuspid reflux. Under such circumstances the veins, if compressed, will fill up from *below*. Transmitted pulsation from the external carotid must not be mistaken for a true venous pulsation.

(ii.) **The Blood Pressure** depends upon the peripheral resistance, the force of the heart's systole, the volume of the blood, and the elasticity of the vessels. The maximum systolic pressure can be measured in terms of millimetres of mercury by means of the sphygmo-manometer. This apparatus consists of a hollow armlet which is connected with a U-tube containing mercury on which there is a millimetre scale, and also with a small ball pump. The armlet is buckled to the upper arm of the patient and filled with air until the radial pulse has disappeared. Air is now permitted gradually to re-enter the armlet by means of a screw valve, and at the moment when the return of the radial pulse is first perceived the height of the column of mercury is read on the scale. This reading is the maximum systolic blood pressure.

In performing the experiment the arm of the patient should be kept on a level with his heart.

It is obvious that impairment of elasticity by calcification or rigidity in the radial artery must influence to a certain extent the readings obtained. There is no unanimity of opinion as to the extent of error that may be so produced, but it is probably equivalent to not more than ten millimetres of mercury.

The blood pressure may rise with exercise or excitement

and also after meals; in health the normal blood pressure of an infant is about 75 mm. of mercury, of children up to the age of 14 about 90—100 mm., of young adults about 115 mm., and from the age of 30 it is said to rise from 8 to 10 mm. for every decade up to 50 or 60 years.

High readings are obtained in cases of nephritis, arterio-sclerosis, gout, lead poisoning, and the like; low readings in acute infective diseases, debilitated conditions, and in cases of shock or hæmorrhage.

A high reading is usually obtained in cases of aortic regurgitation, which is misleading because the *mean* blood pressure in this disease must be low; hence it is a pity that it is so difficult to measure the diastolic pressure and so to establish the mean blood pressure, a reading which would be of more value than that which is now obtained.[1]

VII. ACUTE ENDOCARDITIS.

By acute endocarditis is meant microbic invasion of the endocardium. The primary site of infection is practically always on the valve cusps about $\frac{1}{12}$ inch from their free edges. Endocarditis is divided arbitrarily into Simple and Ulcerative, though there is no essential difference in the pathology of the two conditions.

Simple endocarditis is a much milder infection than the ulcerative form. As a rule the lesions are limited to a fringe of bead-like vegetations along the valve. Ulceration of these vegetations is unnecessary and indeed unusual. In the ulcerative variety, on the other hand, the infection is much more severe; the vegetations are much larger and often spread as fungating masses to the walls of the neighbouring heart cavities.

Simple endocarditis in the great majority of cases is caused by the micro-organism of rheumatism, though it may occasionally accompany scarlet fever, measles, influenza, etc. Ulcerative endocarditis is caused by streptococci, pneumococci, staphylococci, and occasionally gonococci or the bacilli of typhoid fever.

[1] Many sphygmo-manometers are on the market; the one described is the Riva-Rocci pattern.

(i.) **Simple Acute Endocarditis.** The symptoms are likely to be obscured by those of the primary disease. In the case of rheumatism continued pyrexia, despite the use of salicylates, may be suggestive. For the most part, however, the diagnosis must depend on the appearance of physical signs denoting impaired function of one or more of the cardiac valves. The mitral valve is most commonly affected, then the aortic and the mitral, and lastly the aortic alone ; it is very rare for the right heart to be affected primarily. Since, then, a cardiac murmur is likely to be the most important evidence of endocarditis, it is necessary clearly to understand what other explanations there may be of such a murmur.

The murmur may indicate an old valvular deformity resulting from a previous endocarditis ; in such a case the hypertrophy of the heart, as well as the history of previous attacks of rheumatism and perhaps the presence of rheumatic nodules, may assist the diagnosis.

A hæmic murmur is often met with during an attack of rheumatism : the point of maximum intensity of this murmur is in the pulmonary area, and the knowledge that the pulmonary valve is practically never affected in simple endocarditis should prevent any mistake.

A systolic murmur of mitral reflux due to passive dilatation from myocarditis is very common in rheumatism. This condition can only be diagnosed at the close of the illness, when as the heart muscle recovers its tone the murmur disappears, whereas true endocarditis will produce permanent valvular deformity and a lasting murmur will result. The immediate lesions produced by simple endocarditis are nearly always regurgitative, though as the vegetations become replaced by cicatricial tissue stenosis may develop.

The probability of there being active endocarditis is enhanced if there is a distinct alteration, as from blowing to musical, of a mitral systolic murmur, and also if there is a persistent mid-diastolic mitral murmur. This latter murmur does occur sometimes from dilatation, but it rarely persists for more than a few days unless there is thickening of the edges of the mitral curtains.

Obscuring of the aortic second sound followed by the soft diastolic murmur of aortic reflux and occurring in a child

can hardly mean anything else than active endocarditis of the aortic valves. The rapid, irregular pulse, dyspnœa, palpitations, and increased area of precordial dulness which are present in acute endocarditis are only significant of cardiac dilatation, and are therefore of but little service from the point of view of *differential* diagnosis.

(ii.) **Ulcerative Endocarditis.** This condition is a true septicæmia with a coincident inflammatory infection of the cardiac valves and endocardium. Although the left heart is affected much more often than the right, nevertheless the right heart is not so immune as in the case of simple endocarditis. The disease is most common in the third and fourth decades, and valves already damaged by previous inflammation are more liable to infection.

The Signs and Symptoms fall readily into two groups :—

(*a*) General symptoms of septicæmia, such as rigors, sweating, wasting, severe anæmia, purpuric rashes, an extremely irregular, intermittent type of temperature, and the presence of micro-organisms in the blood.

(*b*) Signs of cardiac involvement, such as dilatation, valvular disease, and particularly shifting and variable murmurs, indicating either alteration in existing lesions or the involvement of hitherto unaffected valves, and lastly *evidence of embolism.*

In very exceptional cases there may be no evidence of valvular disease ; we can recall two cases of mitral stenosis with ulceration of the left auricle and ball-clot formation, in which no murmurs were heard throughout the illness though signs of cardiac dilatation were present. Variability of murmurs from day to day is pathognomonic, but unfortunately is not a constant feature.

Embolism, and especially infective embolism, affords important corroborative evidence, though it is necessary to remember that simple infarction is common in mitral stenosis, and that in this condition pulmonary infarcts are particularly frequent.

The symptoms of the more common forms of infarction may briefly be considered in this connection :—

(i.) *Pulmonary Embolism* is commonly produced by the dislocation of clot formed in the right auricular appendix ;

sometimes it follows systemic venous thrombosis or an infective lesion of the pulmonary valve.

A large pulmonary embolus produces sudden precordial pain, extreme dyspnœa without obvious impairment of air entry, cyanosis, and a rapid, irregular action of the heart, followed by pallor, convulsions, and death in a few moments.

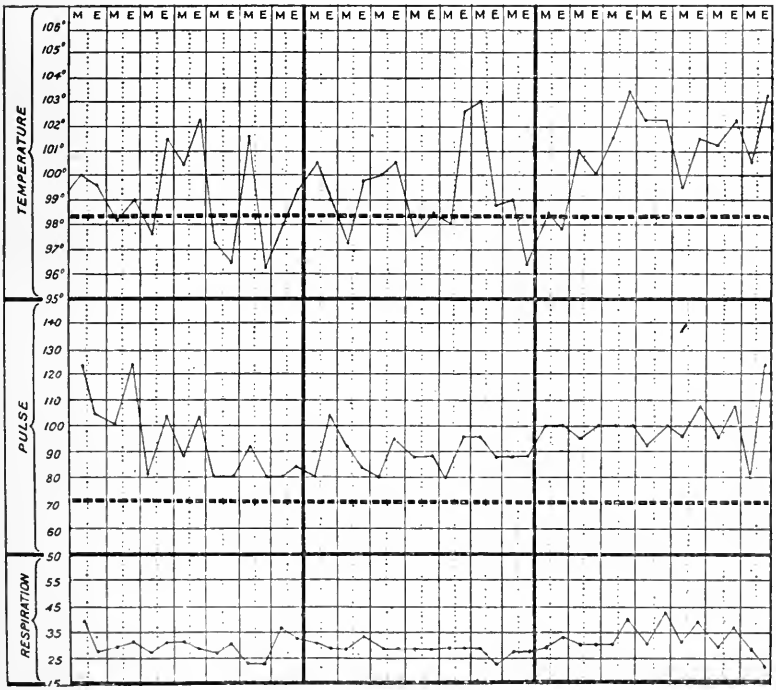


Fig. 42.—Chart from a case of Ulcerative Endocarditis.

Infarction of the smaller vessels causes sudden pain in the chest, with cough, hæmoptysis, dyspnœa, and moderate pyrexia. Often there is localised pleurisy with or without effusion, and there may be evidence of a small area of solid lung.

Sometimes hæmoptysis is the only symptom, while if the infaret is very small there may be no evidence of its existence.

Pulmonary apoplexies are not always embolic ; they may sometimes be produced by gradual arterial thrombosis in cases of chronic cardiac insufficiency, especially when there is mitral obstruction.

(ii.) *Splenic Embolism* may give rise to no symptoms ; commonly there is enlargement of the spleen, and often there is a sharp pain in the lower part of the left chest and a sudden rise in temperature, while a friction rub may be audible in this situation. Enlargement of the spleen from toxæmia is of frequent occurrence in infective endocarditis.

(iii.) *Renal Embolism* may cause severe pain in the loin, and is often accompanied by hæmaturia and pyrexia.

(iv.) *Cerebral Embolism* usually produces a sudden hemiplegia, but the extent of the paralysis naturally depends on the situation of the embolus. Consciousness is not necessarily lost. The left side of the brain is more liable to embolism than the right.

(v.) *Mesenteric Embolism* is accompanied by sudden severe abdominal pain, quickly followed by vomiting and tympanites. There is often malæna after a short interval.

(vi.) *Embolism of the Peripheral Arteries* usually causes sudden pain in the affected limb with obliteration of the pulse beyond the lesion. The limb may be pale and cold, but there is not often œdema, and the collateral circulation is usually established after a short interval. In rare cases gangrene may result, but not unless there is extensive arterial disease co-existing.

Differential Diagnosis. Ulcerative endocarditis may present widely diverse clinical pictures. The diseases with which it is most likely to be confounded are :—

(i.) *Enteric Fever* (*vide* p. 23).

(ii.) *Generalised Tuberculosis.* In the absence of variable cardiac murmurs (for a patient with tuberculosis may quite possibly have *old* valvular disease) or definite evidence of a "tuberculous lesion," the diagnosis may be extremely difficult. A high polymorphonuclear leucocyte count would favour septicæmia, while a preponderance of lymphocytes would favour tuberculosis. A positive blood culture experiment would of course settle the question (*vide infra*).

(iii.) *Malaria.* Occasionally for a few days the tempera-

ture of infective endocarditis may remit in a manner so regular as to simulate malaria. The absence of leucocytosis and the reaction to quinine, as well as the discovery of the plasmodium in the blood, should prevent error for more than a short while.

(iv.) *Basal Meningitis.* In the absence of cardiac murmurs this condition may be simulated. Lumbar puncture with the examination of the cerebro-spinal fluid will quickly settle the question (*vide* p. 512).

In all cases the attempt should be made to cultivate the causative micro-organism from the blood of the patient, both from the point of view of establishing the diagnosis and also in order to prepare a vaccine for purposes of treatment.

At least 10 c.c. of blood should be withdrawn by venipuncture and promptly added to 50 c.c., of sterile broth. The mixture should be incubated at 37° C., and, if any growth result, sub-cultures on agar tubes and other nutrient media may be made for the identification of the organism. The original broth should be kept in the incubator for seventy-two hours before hope is abandoned, since some of the streptococci may take this time to grow (*vide* also p. 157).

The isolation in this manner of an organism from the blood-stream of a patient is proof positive of septicæmia, but does not necessarily indicate ulcerative endocarditis. In the absence of any discoverable septic focus, even if there be no very definite cardiac sign, the diagnosis of ulcerative endocarditis is strongly suggested if a definite organism can be cultivated from the blood, and especially if infarction occurs.

VIII. THE DIAGNOSIS OF VALVULAR LESIONS (Chronic Endocarditis).

In consideration of the individual valvular lesions it must be remembered that both mitral and aortic lesions occurring in young people are nearly always due to rheumatic endocarditis, but that rheumatism occurring for the first time in adult life is not so prone to be associated with permanent cardiac affections. Therefore the

onset of valvular disease in the second half of life is likely to be atributable to other causes, such as fibrosis of the heart musele, arterio-sclerosis, syphilis, or strain.

Tricuspid reflux is a natural sequel of mitral disease or of chronic pulmonary lesions ; it is probable also that to a certain extent it is a physiological occurrence at times of severe physical exertion.

Tricuspid stenosis is not common and does not occur apart from mitral stenosis, the signs of which are so much more prominent that the former condition may not be detected.

Pulmonary regurgitation is so rare as to be negligible ; it may be produced by ulcerative endocarditis. Pulmonary stenosis is only met with as a congenital lesion or possibly in infective endocarditis.

The valvular lesions will now be considered individually.

(i.) Mitral Reflux. **Symptoms.** There are no symptoms so long as the cardiac muscle is able to hypertrophy sufficiently to perform adequately the increased work it is called upon to do.

The earliest symptom to appear will be an abnormal breathlessness on exertion, the result of the inability of the right heart to cope with any *extra* stress ; at the same time the acceleration of the pulse after exercise will take longer to subside than in health. In more advanced stages the symptoms will still point to engorgement and hypertension of the pulmonary blood system, and there will be permanent dyspnœa and cough, sometimes with hæmoptysis.

As the right ventricle fails under the increasing strain the right auricle becomes engorged and the congestion is communicated to the systemic veins : œdema is the result. The cough becomes more marked, the dyspnœa becomes orthopnœa, the liver becomes swollen and painful and may pulsate, and the urine becomes scanty, high coloured, and albuminous.

Physical Signs. The cardiac dulness is increased to the left and also to the right of the sternum ; the impulse is displaced outwards and slightly downwards from enlargement of both ventricles, and except in slight cases it is more diffuse than usual. The pulsation is forcible and heaving and is often well marked in the epigastrium below the

costal margin, when it is largely produced by the right ventricle.

The murmur is systolic in time; it is best heard at the apex, is conducted out into the axilla, and can be picked up again between the scapular angle and the vertebral column. It may accompany the first sound or it may replace it; it is often soft and blowing, but may be harsh or musical.

The pulmonary second sound is accentuated and may be reduplicated. As the right ventricle fails the accentuation of the second sound disappears.

The pulse is strikingly irregular when the lesion is very marked.

Free regurgitation through the mitral ring is indicated by:—

(1) Great enlargement of both ventricles.

(2) Replacement of the first sound by the murmur.

(3) Great accentuation of the pulmonary second sound.

(4) A forcible impulse with a weak small pulse, showing that the blood cannot all be driven into the arteries, and therefore that a good deal of it must go back into the left auricle.

(ii.) Mitral Stenosis. **Symptoms.** The symptoms are the same as those of mitral reflux, with the exception that there is more liability to hæmoptysis from the more sustained high pressure in the pulmonary arteries, and also to embolism, both pulmonary and systemic. Œdema does not occur so readily as in mitral reflux, but ascites is peculiarly likely.

Physical Signs. The right ventricle is enlarged, and sometimes an enlargement of the left auricle can be appreciated by percussion. The impulse is slapping in character, and is not displaced unless it be very slightly to the left.

The most common murmur is presystolic in time. It is a short, rough murmur, runs up to a sharp first sound, is best heard internal to the impulse, and is not conducted out into the axilla. Other diastolic murmurs may be present at the mitral area—viz., the mid-diastolic, which is a short murmur caused by ventricular suction and which resembles very closely a reduplicated second sound, and the early diastolic murmur, which may perhaps be produced by recoil of the pulmonary veins. If all these murmurs are present at the same time a continuous diastolic rumble will result.

A systolic murmur at the mitral area, poorly conducted and not audible behind, is often present; indeed, according to Graham Steel, it is a very common murmur of mitral stenosis.

There is often a thrill, presystolic or diastolic; the pulmonary second sound is exaggerated and often reduplicated.

The pulse is regular, but small and of high tension; it is sometimes anacrotic, and the downstroke is long and gradual.

The signs of mitral stenosis are by no means constant. No murmur may be present at certain stages, and, again, the second sound may disappear entirely at the apex.

It will be convenient to mention the three stages of mitral stenosis as described by Broadbent, though they do not occur in every case.

First Stage. Presystolic murmur, first sound, and second sound.

Second Stage. Presystolic murmur and short, sharp first sound, but no second sound.

Third Stage. Short, sharp first sound, no murmur and no second sound.

The short, sharp first sound is often sufficient by itself for the diagnosis of mitral stenosis; it is probably produced by the ventricular wall being thrown into a sudden state of tension by contracting before its cavity is full of blood.

The disappearance of the second sound is perhaps due to the large right ventricle rotating the left ventricle away from the chest wall, and partly also to the poor quality of the aortic second sound.

The disappearance of the presystolic murmur is due to distension of the auricle hampering or even abolishing its power of contraction.

It is important to time the sounds and murmurs carefully, otherwise when the second sound has gone the shortened first sound may be mistaken for a normal second sound and the presystolic murmur for a systolic one.

(iii.) Aortic Reflux. **Symptoms.** Muscular weakness, pallor, faintness, dyspnœa, both on exertion and paroxysmal, insomnia, and sometimes precordial pain or even angina

pectoris. Dyspepsia is common as in all cardiac disease, and vomiting may be very troublesome. In those cases in which the patients do not die of syncope or exhaustion the mitral valve may become dilated from the great size of the left ventricle, and all the phenomena of back pressure, as in the later stages of mitral reflux, may ensue.

Physical Signs. The heart is enlarged downwards and to the left; the impulse may be in the sixth or seventh space, and is extremely forcible. The characteristic murmur is a soft diastolic murmur, best heard at the aortic cartilage, over the centre of the manubrium, or in the pulmonary area, or even at the lower end of the sternum on either side. It is conducted across the base of the heart down either side of the sternum and towards the apex, where, indeed, it is often audible. Although the murmur is usually soft, it may be quite loud, and in those cases which are caused by sudden rupture of the cusp the murmur can often be heard several feet away from the patient.

The diastolic murmur accompanies or closely follows or entirely replaces the aortic second sound. There is often a loud systolic aortic murmur conducted up into the vessels of the neck, not necessarily due to stenosis, but more often to roughening or puckering of the aortic cusps or to dilatation of the first part of the aorta. When both systolic and diastolic murmurs are present the characteristic to-and-fro double aortic murmur is produced. The presystolic aortic reflux murmur of Flint has been described (p. 215).

The aortic second sound is feeble, impure, or absent.

The arteries as a whole, but particularly in the neck, can be seen to throb violently, and capillary pulsation is often marked. This can be demonstrated by pressing a glass slide on the mucous membrane of the lip, when the pulsations can be seen at the margin of the compressed area.

The pulse is delayed and has the very significant collapsing or "waterhammer" character. It is large in volume and can be felt to come forcibly up against the finger, but it is very poorly sustained, and the artery can be felt to empty completely between the beats. The characteristics of this pulse are best demonstrated by raising the patient's hand above his head and by completely encircling his wrist with

the hand and fingers so as to appreciate the ulnar pulse as well as the radial.

The pulse of children with aortic reflux is not nearly so characteristic as that of adults. This is due to the greater elasticity of the blood-vessels of young people.

The features of the pulse may be masked if the arteries are markedly thickened or if there is co-existing aortic stenosis.

Extensive regurgitation is indicated by :—

(1) Much arterial pulsation.

(2) Very collapsing pulse.

(3) Replacement of aortic second sound by murmur and consequent absence of the second sound in the neck.

(4) Great enlargement of the left ventricle.

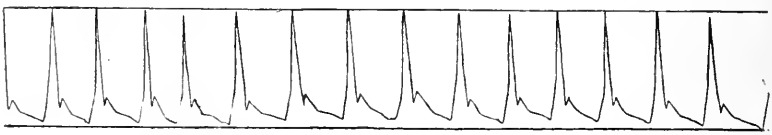


Fig. 43.—Radial pulse tracing from a case of Aortic Reflux.

The maximum systolic blood pressure as measured with a sphygmo-manometer is often high, but since the diastolic pressure must be extremely low, it follows that the mean pressure is below the normal. An important point is the wide difference in the blood pressure readings in the arm and leg in this disease ; the leg reading may be 100 mm. or more higher than that of the arm.

(iv.) Aortic Stenosis. **Symptoms.** Pallor, muscular weakness, dyspnœa, and faintness. Later on when the left ventricle dilates the signs and symptoms of mitral disease will be superadded.

Physical Signs. In the early stages there is pure hypertrophy of the left ventricle ; later on there is dilatation also. The impulse is displaced downwards and very slightly outwards ; it is powerful and somewhat diffused.

The murmur is a rough systolic murmur, heard best at the aortic cartilage and conducted up into the vessels of the neck, and, by reason of its loudness, often heard over the

entire præcordium. It is usually accompanied by a systolic thrill localised to the aortic area.

The pulse is small and often anacrotic, with a gradual rise and a distinct systolic plateau.

Since true aortic stenosis is a rare condition, it is not possible to base a diagnosis of it on the presence of the murmur alone. The size and shape of the left ventricle, the presence of a thrill, the nature of the second sound, and, above all, the character of the pulse must all be carefully considered. As before mentioned, an aortic systolic murmur is very common in advanced life, and may be produced by roughening of the aortic cusps, by atheroma, or by dilatation of the ascending aorta. If there is real stenosis, the cusps are likely to be so bound down that the second sound becomes feeble and dulled.

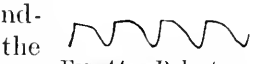


Fig. 44.—Pulse tracing from a case of Aortic Stenosis. Note the rounded systolic plateau and the gradual descent.

In young people an aortic systolic murmur may only be significant of anæmia.

(v.) Pulmonary Reflux. This lesion does not produce any definite symptoms. The murmur is a diastolic murmur accompanying or replacing the pulmonary second sound, best heard in the third left space and conducted down the left border of the sternum. The right ventricle is hypertrophied. The character of the aortic second sound, the size of the left ventricle, and the condition of the pulse and arteries should prevent confusion between aortic and pulmonary reflux.

(vi.) Pulmonary Stenosis. *Vide* "Congenital Heart Lesions," p. 255.

(vii.) Tricuspid Reflux. This condition is practically always secondary to other cardiac lesions, but in addition to the signs and symptoms of the primary lesion tricuspid reflux can be diagnosed by the systemic venous engorgement and the systolic venous pulse in the veins of the neck, which fill up from below. Another certain sign is a pulsating liver, which, however, must not be confused with a liver which receives a transmitted pulsation from an hypertrophied right ventricle. The expansion of a pulsating liver can be appreciated by bimanual examination with the

left hand in the loin and the right hand over the costal margin.

The murmur of tricuspid reflux is systolic in time and is best heard in the fifth left space close to the sternum ; it is conducted out towards the apex as far as the left border of the right ventricle, but no further. This murmur is not always present. General anasarca is a common accompaniment of tricuspid reflux, but is not a necessary adjunct.

(viii.) Tricuspid Stenosis is usually masked by the coincident mitral stenosis.

The murmur, if audible, is presystolic in time and localised to the tricuspid area. Chronic distension of the jugular veins without systolic pulsation might be an important sign.

IX. THE DIAGNOSIS OF HYPERTROPHY AND DILATATION.

(i.) **Hypertrophy.** This must not be regarded as a pathological condition; it is in all cases beneficial, since it indicates that the heart is able to deal with the work imposed upon it. Of course, the fact that the heart is hypertrophied is a sure indication that the work done is in excess of the normal, and the cause of this necessity for increased work is, in many cases, pathological.

The Left Ventricle will become hypertrophied from the following causes :—

(*a*) Valvular disease ..	Aortic reflux. Aortic stenosis. Mitral reflux.
(*b*) Other cardiac lesions	Coronary artery obstruction. Cardiac fibrosis. Adherent pericardium.
(*c*) High blood pressure	Renal disease. Arterio-sclerosis.
(*d*) Imperfect nervous control, *e.g.*, Tachycardia in Graves' disease, etc.	
(*e*) Prolonged strain and over-exertion ..	Soldiers. Athletes.

The Left Auricle is hypertrophied in mitral disease, but especially in mitral stenosis.

The Right Ventricle is hypertrophied in :—

(*a*) Valvular disease ..	Mitral stenosis. Mitral reflux. Tricuspid reflux. Pulmonary stenosis. Pulmonary reflux.
(*b*) Other cardiac lesions	Adherent pericardium. Fibrosis of heart.
(*c*) Chronic pulmonary lesions	Chronic bronchitis. Emphysema. Fibroid lung.

The Right Auricle is hypertrophied :—

(*a*) Secondary to right ventricle hypertrophy from any cause and consequent tricuspid reflux.

(*b*) Primary tricuspid reflux ; tricuspid stenosis.

Symptoms of hypertrophy are usually absent. There may be consciousness of the heart's action and more or less insomnia. When symptoms appear it usually means that there is commencing dilatation.

The Physical Signs of hypertrophy of the *left ventricle* are :—

(*a*) Displacement of impulse *downwards* and outwards.

(*b*) Increased percussion area downwards and to the left.

(*c*) Powerful, heaving and rather diffuse impulse.

(*d*) Prolonged, low-pitched first sound at apex and at aortic area ; accentuated ringing and sometimes reduplicated aortic second sound.

(*e*) The pulse varies with the cause of the hypertrophy. Except in certain valvular diseases the pulse will be of high tension and incompressible, with a gradual rise and a rounded summit. The artery is likely to be easily palpable between the beats.

Hypertrophy of the *right ventricle* is shown by reduplication or accentuation of the pulmonary second sound ; it is not easy to percuss out an hypertrophied right ventricle unless there is dilatation as well, but a diffuse heaving

impulse under the xiphisternum is suggestive of this condition.

(ii.) **Dilatation** of the heart may be beneficial up to a point; indeed, it is necessary in most cases of valvular disease. When, however, the dilatation is in excess of the accompanying hypertrophy grave issues are at hand.

Any condition which tends to require an increasing amount of cardiac hypertrophy will sooner or later produce a condition of clinical dilatation.

Dilatation may, however, be produced primarily by the action on the myocardium of the toxins of infectious diseases, such as diphtheria, enteric fever, rheumatism, etc., or certain chemical poisons, such as alcohol and tobacco, by unaccustomed and protracted exertion, and lastly by severe emotional crises.

The Early Symptoms of cardiac dilatation are:—Debility, both mental and physical, insomnia, bad dreams, and especially breathlessness on any exertion. Later on paroxysmal dyspnœa, palpitation, cyanosis, coldness of the extremities, cardiac œdema, enlargement of the liver, and scanty urine are likely to be met with.

The Physical Signs are:—An increase horizontally in the area of cardiac dulness, a diffuse indeterminate impulse, a short, sharp first sound, and a rapid, irregular, and soft pulse.

Such cardiac murmurs as are present may indicate some primary valvular lesion, or they may be systolic dilatation murmurs. In the earlier stages of dilatation the systolic period is likely to be prolonged at the expense of diastole producing a "tic-tac" rhythm; but in the more severe stages the heart sounds appear too close together, and at the same time the diastole is shortened.

Œdema of the lungs, effusions into the serous cavities, embolism, gastric catarrh, and Cheyne-Stokes respiration are all significant accompaniments of extreme cardiac dilatation.

For the differential diagnosis of Dilated Heart from Pericardial Effusion, *vide* p. 259.

X. MYOCARDIAL DEGENERATIONS.

These may be divided into Toxic and Degenerative. In the former group are included the results of the infective

diseases, such as rheumatism, influenza, diphtheria, as well as the grave anæmias, phosphorous poisoning, etc.; in the latter the results of gummatous deposits, subacute myocarditis, and the occlusion of the coronary arteries by atheroma or by endarteritis, by embolism or thrombosis.

The acute fevers produce cloudy swelling, with more or less granular degeneration and sometimes a certain amount of fatty change.

The grave anæmias, phosphorous, arsenic, and alcohol are likely to produce extensive fatty degeneration.

Coronary obstruction, when of gradual occurrence, is followed by fibrous transformation and also by fatty degeneration in varying proportions.

Coronary embolism, in the absence of sudden death, is of course followed by infarction of the heart wall. Softening in the infarct is likely to produce an aneurysm of the heart.

The symptoms of the cardiac degeneration are those of myocardial insufficiency and dilated heart. They include dyspnœa, palpitations, precordial pain, often paroxysmal, sense of oppression, and frequently more or less cardiac œdema.

The Physical Signs are:—An increase horizontally in the area of cardiac dulness, a diffuse feeble impulse, arrhythmia, and a shortening of the first sound. Soft dilatation murmurs may be heard. Later on the first sound may disappear, and serous effusions and pulmonary œdema may become manifest. The pulse is often slow, but in practically all cases the outstanding feature is its irregularity.

Both fibroid and fatty hearts are liable to cause sudden death with or without a preceding illness, and also syncopal, apoplectic, and epileptiform seizures.

Diagnosis. The obvious signs of severe cardiac incompetence, in the absence of primary valvular disease, make the diagnosis easy in the later stages, but the condition may well be overlooked at its commencement. Danger signals are the appearance in an elderly man of undue dyspnœa on exertion, irregularity of the heart's action after slight exertion, shortening of the first sound, spacing of the heart sounds, and an irregular, small, soft pulse, possibly in a thickened artery.

It is not possible clinically to differentiate with certainty between fibroid and fatty hearts. Poor circulation with cyanosed extremities in an obese individual who is prone to sleep in the daytime, together with absence of obvious arterial degeneration, would suggest fatty rather than fibroid heart, provided that the other signs and symptoms of cardiac degeneration were present.

XI. SOME FORMS OF CARDIAC IRREGULARITY.

In the last few years the work of Mackenzie, Lewis, and others with the polygraph and the electrocardiograph has made it possible to speak with some certainty as to the actual mechanism which causes irregular heart-beats. We are also enabled to estimate to a better extent than before the clinical significance of some of the more common irregularities, and the result is that the prognosis of heart disease has been placed altogether on a more sure basis than was formerly the case.

The Polygraph is an instrument which gives simultaneous tracings of the auricular and ventricular beatings, and on the same ribbon is a time lever marking fifths of a second. Considerable practice is required to interpret correctly the tracings of a polygraph, but most accurate information can be obtained from the instrument by one skilled in its use.

The Electrocardiograph affords similar and even more accurate information, but its use is necessarily restricted to the electrical department of large hospitals.

In the present work it is not proposed to discuss further the polygraph or the electrocardiograph, but rather to describe shortly the clinical aspects of some of the cardiac disorders which have been elucidated by their means.

Before proceeding to the irregularities of rhythm it is necessary to refer briefly to the normal mechanism of the heart-beat. The heart beats as the result of a series of rhythmical impulses which arise in a small mass of tissue called the sino-auricular node. This node is situated at the end of the sulcus terminalis close to the junction of the superior vena cava and the right auricle. The impulses

pass through the muscle of both auricles and thence to the auriculo-ventricular bundle of His, which runs from the right auricle near the coronary sinus forwards and downwards in the inter-ventricular septum, to be distributed throughout the muscle cells of each ventricle. The contractions of the auricular and ventricular chambers follow a perfectly regular and definite sequence, the wave of contraction starting at the sino-auricular node, spreading through both auricles and thence down the auriculo-ventricular bundle to the ventricles.

Every auricular contraction is followed, after a definite and constant interval, by a ventricular contraction, and the commencement of ventricular contraction coincides approximately with the termination of auricular contraction.

The period elapsing between the inception of auricular contraction and the inception of ventricular contraction is called the " As-Vs " interval ; it is an important measurement, as will become apparent later, and can readily be determined by the polygraph.

The ventricle in diastole is always ready to receive from the auricle a stimulus to contract ; if, however, the auricle sends a stimulus to the ventricle when the latter is in systole (an event which can only occur in disease), the ventricle cannot respond and that stimulus is lost.

An important point is the tendency for the auricle to continue beating with perfect regularity, quite regardless of what the ventricle may be doing, so long as there is no auricular disease.

The cardiac nerve plexuses ramify amply in the sino-auricular node, and but for one deterrent factor the auricular rate would be considerably more rapid than it is ; the deterring factor is the vagus nerve, which exerts a constant inhibitory influence on the frequency of the stimuli sent out by the sino-auricular node.

Adopting the classification of Lewis, the following irregularities in cardiac rhythm may now be considered separately :—

(1) Sinus irregularity.
(2) Heart-Block.
(3) Premature Contractions.

(4) Simple Paroxysmal Tachycardia.
(5) Auricular Flutter.
(6) Auricular Fibrillation.
(7) Pulsus Alternans.

(1) **Sinus Irregularity.** In sinus irregularity each phase of the cardiac cycle occurs absolutely normally; the As—Vs interval is always precisely the same, but the individual heart-beats do not follow each other at identical intervals. Clinically this is perceived by pauses of unequal length between the pulse as felt at the wrist or at the cardiac impulse, though each beat when it comes is of similar quality to its fellows.

Sinus irregularities are due to vagal influences and are often rhythmical.

The following types may be recognised :—

(*a*) Tachycardia the result of large doses of atropine, which paralyses the vagus endings in the heart and permits of a pulse-rate of 100 to 160 per minute.

(*b*) Definite slowing of the heart is a common manifestation of aortic stenosis, jaundice, high blood pressure, pregnancy, convalescence from specific infections, and prolonged muscular endeavour.

(*c*) *Respiratory Influences.* In children and young adults forced inspiration quickens the rate of the pulse, which again becomes slowed when the lungs empty.

In older people this effect is not commonly obtained.

In young adults *ordinary* respiration has no perceptible effect on the pulse-rate, but in children very often there are one or two quite long pauses at the end of ordinary expiration.

(*d*) Periodic and wave-like variations in pulse-rate lasting perhaps twenty to thirty seconds may follow large doses of digitalis.

The diagnosis of sinus irregularities depends on the following points :—

(i.) The age of the Patient. Practically all irregularities in young children (up to ten years) are of this nature.

(ii.) Evidence of respiratory influence; this, when present, points conclusively to sinus irregularity.

(iii.) The uniformity of the pulse waves and of the cardiac

impulses, as well as the correspondence of radial pulse and apex beat, are strong points in favour of sinus irregularity.

(iv.) The disappearance of the irregularity if the pulse-rate becomes raised, as by exercise or in febrile conditions.

(2) **Heart-Block**. This condition depends upon some obstruction to the passage along the auriculo-ventricular bundle of the impulse to contract, which normally passes from the right auricle to the ventricles. Erlanger has shown that all grades of heart-block may be produced experimentally by clamping the auriculo-ventricular bundle at different pressures. Such obstruction may be caused by myocardial intoxication from specific infections, such as rheumatism, diphtheria, influenza, enteric fever, septicæmia (of these rheumatism is undoubtedly the most important), or to myocardial degeneration from fibrous replacement, the result of syphilis or of arterio-sclerosis from any cause.

It is understood that for heart-block to occur the lesion must actually involve the auriculo-ventricular bundle, though, of course, this is often only a local manifestation of a much more widely diffused condition. The bundle may be rendered entirely functionless, or its conductivity may only be impaired to a greater or less extent.

The earliest result of an incomplete lesion is to hamper the impulses in their passage from the auricle to the ventricle, and this causes delay in their transmission, so that there is a prolongation of the As — Vs interval.

Now it must be remembered that all the while the auricle goes on steadily contracting at its normal and regular rate, so that as the As—Vs interval gets longer so does the commencement of each auricular contraction get closer and closer to the end of the previous ventricular systole, with the result that eventually an auricular stimulus reaches the ventricle when it is in systole and so unable to respond ; at this juncture there is a ventricular silence or dropped beat and the ventricle is enabled to rest before responding to the next auricular stimulus.

The further fact has been established that, as a rule, the As—Vs intervals lengthen progressively up to a point, but that the intervals immediately preceding the ventricular silence shorten up again quite definitely, and, further, the

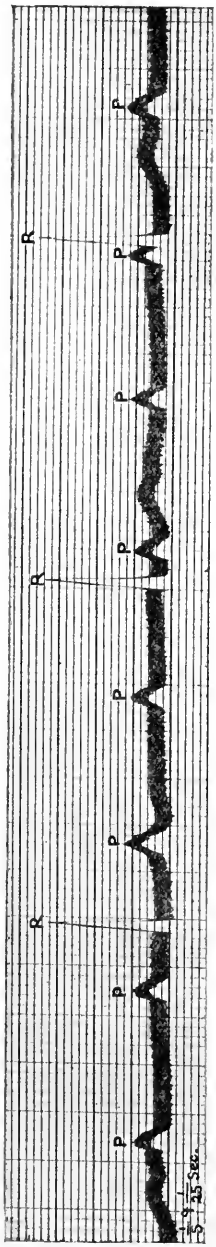


Fig. 45.—Electro-cardiogram from a case of complete Heart-block.
P = Auricular systole.
R = Ventricular systole.
Note the complete dissociation of ventricle from auricle and also the regularity of action of each chamber

As—Vs interval immediately following the silence is notably shortened. The result of this is that the long silence is rather shorter than the length of two ordinary pauses, and there is a definite quickening of the ventricle just before and just after the silence.

As the severity of the heart-block increases, characteristic rhythms are sometimes found in which the ventricle is enabled to respond to every alternate auricular stimulus (a 2 : 1 heart-block) or to every third auricular stimulus (a 3 : 1 heart-block), etc. The effect of these latter rhythms is to produce a slow but perfectly regular pulse the rate of which is a definite fraction, one-half, one-third, or one-quarter, as the case may be, of the auricular rate, which usually remains about 72 per minute.

In complete heart-block no stimuli at all reach the ventricle from the auricle. Under such circumstances the ventricle institutes a rhythm of its own which is perfectly regular but very slow, usually 28 to 30 per minute ; the ventricle and auricle are now beating independently of each other, both quite regularly, but at widely different rates (Fig. 45).

The Clinical Diagnosis of Heart-Block. It may be quite impossible to diagnose heart-block

without recourse to special graphic methods; it may, however, be suspected if attention is paid to the following points :—

(i.) When the heart-block is of such mild grade that there are no dropped beats, the widening of the As—Vs interval may lead to audible recognition of the sound of auricular systole, which becomes apparent as a reduplication of either the first or second cardiac sound, according as to whether the auricular and ventricular systoles are slightly or more widely separated.

Similarly in mitral stenosis the murmur of auricular systole may become displaced backwards till it occupies early diastole and not "pre-systole."

(ii.) When there is an occasional long pause in the radial pulse and examination of the cardiac impulse shows that these pauses coincide with a ventricular silence and the pauses are not connected with the respiratory cycle, it is justifiable to diagnose partial heart-block, especially if there is a recognisable quickening of the pulse-rate just before and just after the pause, so that the pause is not equal to two ordinary beats, though, even if the pause is equal to two ordinary beats, heart-block is the probably explanation.

(iii.) When the pulse-rate becomes suddenly half or one-third its previous rate, heart-block is the probable cause.

(iv.) A ventricular rate of 35 or less, especially when quite regular, justifies the diagnosis of complete heart-block. In such cases occasional first sounds are louder than their neighbours owing to coincidence of auricular and ventricular systoles, and occasional first sounds appear reduplicated when the auricular and ventricular systoles follow each other very closely. Regular pulsations, corresponding to the auricular systoles, may be detected in the jugular veins, and these venous pulsations wax and wane independently of respiration.

(v.) Large doses of digitalis and similar drugs may produce heart-block.

The clinical significance of heart-block depends very largely on the condition of the heart muscle as a whole and also on the soundness or otherwise of the valves.

If the damage is confined to the auriculo-ventricular bundle, heart-block seems little or no bar to an active

and vigorous existence; neither is it incompatible with longevity.

Only too often, however, the particular damage which causes heart-block is but part of a widespread myocardial and valvular disease, and in such cases the outlook is correspondingly serious, but more from the ordinary results of heart failure than from the heart-block.

As a matter of clinical interest it may be mentioned here that unconsciousness is the rule when there is no ventricular systole for from three to seven seconds, when the heart-beats fall notably below twenty per minute, or when they accelerate to 300 per minute, as may very occasionally happen in Auricular Flutter (*vide* p. 249).

Stokes-Adams Syndrome. This name is given to a group of cases in which there is a severe grade of heart-block associated with a certain definite train of symptoms. All cases of Stokes-Adams syndrome have heart-block, but all cases of heart-block do not have Stokes-Adams syndrome. The especial signs described are most liable to occur when the block is practically complete. They consist of:—

(i.) Attacks of giddiness
(ii.) Syncopal attacks ..
(iii.) Epileptiform attacks

} Probably due to cerebral anæmia.

(iv.) Venous pulsation in the neck, which does not synchronise with the cardiac impulse.

(v.) A ventricular rate of 35 or less.

In addition to the above, paroxysmal dyspnœa or cardiac asthma may be met with, and (in the older patients) there is likely to be arterial thickening.

The full Stokes-Adams syndrome is most often seen in men over 50 years of age.

(3) **Premature Contractions or Extrasystoles.** These are contractions occurring before the proper rhythmical time which originate abruptly in some other spot, either auricular or ventricular, than the sino-auricular node, and which do not tend to initiate a definite rhythmical series of contractions.

Premature contractions may be either ventricular or auricular in origin, and are the usual cause of "intermittent pulse."

(*a*) A *Ventricular Premature Contraction* occurs after an unusually short pause ; it is usually weak, does not reach the wrist, and often fails to raise the aortic and pulmonary valves sufficiently for there to be a second sound. Nevertheless, the ventricle is in systole when the next auricular impulse reaches it ; consequently it is unable to respond and there is a long pause. The ventricle takes up the next auricular impulse in precisely the manner and at precisely the time as it would if there had been no disturbance.

The time from the commencement of the ventricular systole before the premature contraction to the commencement of the ventricular systole next after the premature

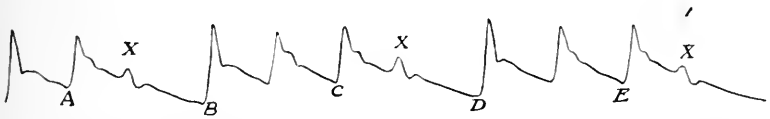


FIG. 46.—Ventricular tracing to show Premature Contractions marked *X*. Note that the interval *A B* = the interval *B C*, and likewise *C D* = *D E*, which suggests that the extrasystoles are ventricular in origin.

contraction is exactly double the ordinary cardiac cycle —that is to say, there has been interpolated a premature and ineffective ventricular contraction instead of a normal one, and beyond that nothing is altered.

(*b*) In *Auricular Premature Contraction* the auricle contracts before its time, and this premature contraction is followed by a ventricular response in precisely the same manner as in a normal auricular contraction. Both chambers are momentarily out of gear with the normal sino-auricular rhythm.

The period between the commencement of the premature systole and the commencement of the next or normal systole is longer than the ordinary inter-systolic interval, but the interval between the commencement of the normal systole before the premature contraction and the first normal systole subsequent to this is *not* quite so long as

two ordinary cardiac cycles, as is the case in premature ventricular contractions.

By definition it is agreed that the premature contractions are the result of stimuli originating elsewhere in the heart muscle than in the sino-auricular node. The cause of such stimuli is not very certain ; nevertheless, it has been shown that age is an important factor, since premature contractions are most frequent between the ages of 50 and 70 and are hardly ever found before the age of 10 years. Further, nearly 60 per cent. of these cases are associated with grave cardiac lesions either valvular or myocardial.

Over-indulgence in tobacco is a fairly frequent temporary cause of these extrasystoles, especially in young men. At the same time a large group is left in which, beyond the occurrence of premature contractions, the patient has no demonstrable lesion: in such it does not seem justifiable to attach much importance to the phenomenon.

Again, when there is obvious cardiac disease as well as premature contractions, the prognosis is necessarily very guarded, quite apart from the latter.

From these considerations it may be argued that premature contractions *per se* are of little or no significance, though probably adding somewhat to the gravity of a case with obvious cardiac disease. On the other hand, premature contractions are necessarily evidence of some pathological process, and, as such, are always worthy of attention in the direction of periodical re-examination, for they may be the first stage in a progressive lesion in so far as there is a common pathological basis for premature contractions and the more serious irregularities, such as paroxysmal tachycardia, auricular flutter, and auricular fibrillation.

The Clinical Diagnosis of Premature Contractions.

(i.) They may be induced by the digitalis group of drugs.

(ii.) They are rare when the pulse-rate is over 100 per minute, and they are temporarily abolished by anything (*e.g.*, exercise or fever) which raises the pulse-rate to this frequency or over.

(iii.) They may be induced by forced holding of the breath.

(iv.) They tend to disappear when the patient lies down.

(v.) A long pause in the radial pulse, during which a ventricular systole can be felt at the cardiac impulse, is suggestive of a premature contraction of insufficient force to raise the aortic valves.

The auscultatory sign of this is an apparent grouping of three sounds at the time of the extrasystole, these being the first and second sounds of a normal heart-beat closely followed by the extra contraction.

(vi.) If the contraction is strong enough to send blood into the arteries, there is an extra first and second sound, so that the heart sounds at the moment appear grouped in fours. In this variety there is not a complete pause in the radial pulse, but there is instead a hurried and feeble beat (the premature contraction) followed by a rather longer pause than usual.

(vii.) If the extrasystoles are sufficiently numerous to alternate with the ordinary rhythmical beats, the heart sounds are arranged constantly in "groups of three," provided that the aortic valves are not raised by the premature contraction, and the heart-rate is double the radial pulse-rate.

If the aortic valves are raised, the radial pulse is in pairs, one strong and one feeble, and the heart-beats are in fours.

This last condition is *not* the same as pulsus alternans, in which the interval between the beats is practically equal.

(viii.) Apart from a pulse tracing it may be impossible to distinguish between auricular and ventricular premature contraction, neither is this differentiation of importance. In the former there is disturbance of the normal sino-auricular rhythm, in the latter there is not. This may sometimes be appreciated by estimating with the finger or the ear whether the area of disturbance—that is to say, the interval between the commencement of the systoles next before and next after the extra one—is equal to or less than two ordinary cardiac cycles. In the former case the disturbance is ventricular, in the latter it is auricular. Other diagnostic features may be :—(*a*) If the disturbance is ventricular, the premature contraction may, and is indeed likely to, coincide with the next normal auricular

systole; the result of this is to force the blood into the veins of the neck with a jerk that can easily be recognised. (*b*) The same coincidence of auricular and ventricular systoles tends sometimes to exaggerate that particular first sound.

(ix.) Aortic murmurs in premature contractions depend on whether the aortic valves are raised or not.

Mitral systolic murmurs are present with the extrasystole. Mitral presystolic murmurs are, of course, absent when the premature contraction is ventricular, and usually also when it is auricular, probably because the extra contraction is too feeble to generate a murmur.

(x.) Premature contractions are often unnoticed by the patient, but he may complain of palpitations, of a sense of oppression during the long pause, or of feeling his heart miss a beat.

(4) **Simple Paroxysmal Tachycardia.** By this is meant the periodical domination of the normal sino-auricular rhythm by a series of impulses which originate in some other part of the heart muscle, usually in the auricle, but sometimes in the ventricle. These impulses are fired off from 110 to 200 times a minute, or more commonly 140 to 190. The condition is comparable to a series of consecutive premature contractions occurring so rapidly as to block all the normal sino-auricular impulses for the time being.

When the impulses originate in the ventricle it is to be presumed that the stimulus spreads in a retrograde manner to the auricles, which then occupy the *second* stage of the cardiac cycle with their systoles.

When the paroxysm terminates there is a longer pause than there is between two normal beats, just as after a single premature contraction, but the intervals between the successive beats of a paroxysm are of equal length, which indicates a single focus of elaboration for the abnormal stimuli.

Nearly half these cases have obvious myocardial degeneration or mitral disease, and a certain number have arteriosclerosis or renal disease, while it seems certain that, whether or not there are signs or symptoms, all these cases depend upon a definite myocardial change. It will be noted that

tachycardia from increase of activity in the sino-auricular node is a totally distinct condition.

Simple paroxysmal tachycardia is met with at any age after 10 years, it is perhaps most frequent between 20 and 30, and occurs more often in men than women.

Individual paroxysms may last from a few seconds to upwards of two weeks.

The diagnosis depends on the following points :—

(i.) The abrupt onset and equally abrupt termination of an attack.

(ii.) The rate of heart-beat. Any adult heart with a rate of 160 or more is almost certainly deriving its impulses from an abnormal site.

(iii.) The absolute lack of influence of change of posture or respiration on the rate of the heart-beats (this helps to exclude sinus tachycardia).

(iv.) The fact that paroxysms may be elicited by emotional factors, dyspepsia, and such trivial things as the adoption of certain postures.

The symptoms vary greatly, and depend to a large extent on the duration of the attack. Palpitation, exhaustion, coldness, and sweating are commonly noted ; anginal pain is not rare, and in more serious cases an increasing area of cardiac dulness is accompanied by dyspnœa, with cyanosis, venous engorgement, œdema of the feet, pulsating liver, and pulmonary œdema, which may cause great anxiety and even terminate fatally.

Even when the patient is apparently *in extremis* the attack may terminate in the usual abrupt manner, and recovery is then peculiarly rapid.

Such conditions as alcoholism and Graves' disease, which cause tachycardia, can usually be excluded by their appropriate physical signs, by the lack of the paroxysmal element, and by the reaction of the pulse-rate in these patients to sedatives and recumbency.

(5) **Auricular Flutter.** This can be regarded as a development of simple paroxysmal tachycardia when the new rhythm has completely dominated the situation but wherein the auricular rate is very much more rapid than in paroxysmal tachycardia.

The rate of the auricular beat has been settled arbitrarily (for clinical purposes) as from 200 to 350 per minute. The reason for this arbitrary distinction lies in the fact that after the rate of 200 per minute is reached the ventricle does not get sufficient length of latent periods and becomes unable to keep up ; the result is heart-block, which is practically always present in cases of auricular flutter.

When this perverse rhythm is once established the tendency is for it to continue for many months, though sometimes paroxysmal auricular flutter may be found.

A common type of case is for the auricle to beat say 300 to 320 times per minute and the ventricle at half this rate (2 : 1 heart-block). Less commonly a 3 : 1 or 4 : 1 heart-block is met with, and sometimes complete heart-block may be found in cases of flutter.

Except for the rate of the auricle, all that was said in the section on " Heart-Block " applies to flutter also, and a very striking point is the absolute regularity of the auricular contractions and their frequency throughout weeks and weeks. In a like manner, since the ventricular contractions (even when not in simple ratio to the auricular contractions) appear in definite cycles at stated intervals, the frequency of the cardiac impulses has the same striking uniformity for the same patient.

Auricular flutter must be regarded as due to myocardial degeneration ; it is more common in men than women and is most often found between the ages of 50 and 70 years.

It occasionally happens that for a few moments the ventricle picks up each of the auricular contractions ; this induces unconsciousness if the rate approaches 300 per minute, and is speedily fatal if it persists.

The diagnosis of auricular flutter cannot always be made without the polygraph or electrocardiograph ; especially is this the case when the ventricular rate is slow and regular, for, unless it is possible to perceive some venous fluttering in the neck, there is no way of appreciating what is happening in the auricles. Nevertheless, a slow ventricle is the exception rather than the rule, so that auricular flutter may often be suspected under the following circumstances :—

(i.) A *regular* and *persistent* pulse of 130 to 160 per minute in an elderly man, especially if a history of previous attacks of tachycardia can be elicited.

(ii.) The constant repetition of the *same high* pulse-rate throughout weeks and months.

(iii.) No alteration in rate with altered position, rest or exercise.

(iv.) Occasional brief crises, possibly with loss of consciousness, due to a very transient increase of ventricular rate to that of the auricle.

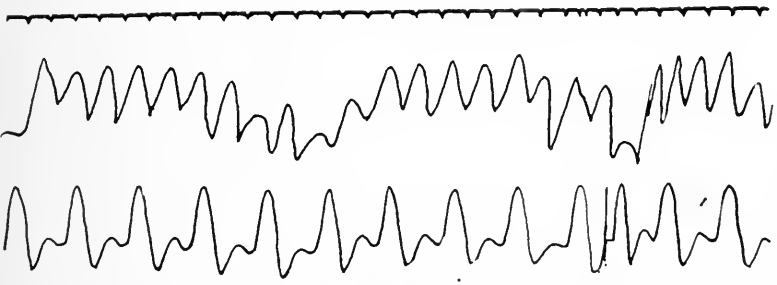


Fig. 47.—Polygraph tracing to show Auricular Flutter combined with a 2 : 1 Heart-block. The upper tracing shows the auricular wave; the lower tracing is the radial pulse.

Note the rapidity and regularity of the auricular systoles, also that the auricle beats twice for each ventricular contraction. The topmost line is a time record showing fifths of a second.

(v.) The fact that digitalis both slows the pulse by further blocking the auricular impulses and also makes it irregular.

The symptoms of auricular flutter are strikingly few. When the flutter is paroxysmal the symptoms are those of flaroxysmal tachycardia ; when, as is usually the case, the putter is well established and a well-defined heart-block is present, even though the ventricular rate is 160 per minute, the symptoms are often only those of slight myocardial degeneration—that is to say, easy fatigue and dyspnœa on exertion.

Of course, if the heart muscle is profoundly altered, more urgent congestive symptoms are likely to be present.

(6) **Auricular Fibrillation.** In this condition the normal sino-auricular rhythm is replaced by impulses arising at very many different points in the auricular muscle. Any

orderly auricular contraction becomes impossible, since many different small parts of the auricle are constantly contracting independently. An auricle seen in a state of fibrillation appears to be distended in diastole; closer examination shows that it is " twittering " all over, but each contraction is so minute and so local that there is no result on the auricle as a whole.

The ventricle picks up as many of these auricular impulses as it can, but does so in a most haphazard manner; consequently the pulse becomes both rapid and extremely irregular.

It has been suggested that the nature of the changes in the auricular musculature which cause fibrillation is akin to that which causes a single premature auricular contraction, with the addition that in the latter there are multiple pathological foci and in the former only one.

The number of effective impulses reaching the ventricle seems to vary with the efficiency of the auriculo-ventricular bundle; if this is quite healthy the ventricular rate approaches its potential maximum of 200 per minute: conversely heart-block is not incompatible with auricular fibrillation. The most usual rate is between 90 and 140 per minute.

Auricular fibrillation is considerably more frequent in men than in women and the etiological influence of rheumatism can be traced in over 60 per cent. of cases. As would be expected, mitral stenosis is the lesion *par excellence* to be associated with fibrillation, and this connection has been traced in slightly more than 50 per cent. of cases.

Considering that, according to Lewis, more than 60 per cent. of all cases of " heart failure " admitted into a hospital can be shown to have auricular fibrillation, it is important to recognise the clinical features of this condition.

Diagnosis. Apart from the polygraph or electrocardiograph, the diagnosis of auricular fibrillation depends on the following considerations :—

(i.) The extreme irregularity of the heart's action; and, since many beats may fail to reach the wrist, it is advisable to examine the cardiac impulse. No two beats are alike, no series of beats can be said to resemble any other series,

no two pauses are the same length, and the force of the impulse bears no apparent relation to the length of preceding pause. Clinically the key to the diagnosis of auricular fibrillation is the very characteristic *disorderly rhythm*.

(ii.) The rate of the heart-beats is from 100 to 160 in the most typical cases. When the rate is slower the irregularity requires more care in its detection, but, if suspected, should be discovered.

(iii.) Since many contractions are ineffective, the heart sounds and murmurs may present some of the features of premature ventricular contraction (p. 245).

(iv.) In mitral stenosis the rough *presystolic* murmur disappears when fibrillation sets in, but instead of this a softer diastolic murmur appears early in diastole. This

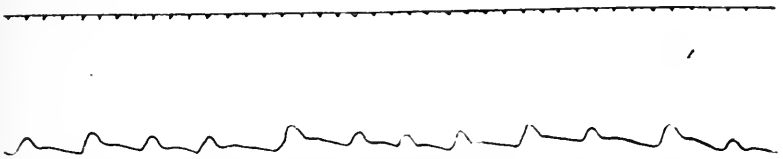


Fig. 48.—Pulse tracing to show the characteristic disorderly rhythm of Auricular Fibrillation.

murmur starts just after the second sound, but does not accompany this; the more rapidly the heart is beating the more of the diastole is occupied by the murmur. With a paralysed auricle the rate of flow from auricle to ventricle is greatest at the commencement of ventricular diastole.

(v.) Exertion increases the irregularity in auricular fibrillation in contrast to its effect in premature contractions and partial heart-block.

(vi.) Irregularity due to fibrillation persists as a rule until death takes place.

The symptoms of Auricular fibrillation are not characteristic, being practically those of myocardial degeneration.

With regard to **prognosis,** it may be said that an irregular pulse-rate of more than 120 is of grave significance and that the greater the frequency the graver the prognosis; this is

of especial importance, in view of the fact that adequate digitalis administration is capable of reducing the pulse-rate very markedly in fibrillation cases through blocking many of the impulses from the auricle and so resting the ventricle, though it does not commonly cure the fibrillation.

The ventricle may undoubtedly go into a state of fibrillation, in which case death rapidly takes place.

It is probable that many cases of sudden death in myocardial fibrosis are due to ventricular fibrillation, and it is the immediate cause of death in lightning stroke and electrocution.

(7) **Pulsus Alternans.** By this is meant a perfectly-spaced pulse, but one in which the strength of the ventricular contractions alternates, a powerful systole being followed by a weak one.

Pulsus alternans may occur when the heart is acting very rapidly, as in paroxysmal tachycardia ; it is not then of special significance : it is, however, a sign of the greatest import when occurring in hearts of moderate rate, and indicates definitely that the heart is emphatically not equal to the work it is called upon to perform.

As would be expected, it is most often met with in cases of fibroid heart, arterio-sclerosis, etc., but it may occur in the course of the specific infections, such as pneumonia, when a fatal issue can be apprehended.

The diagnosis of pulsus alternans is usually impossible apart from graphic methods (an ordinary sphygmographic tracing shows it very well) ; it may be appreciated by the finger, but will more often be overlooked, since the difference in the vigour of the contractions is not excessive.

Pulsus alternans may not be constant and each cycle of alternation is often initiated by a premature contraction, and, since the prognostic value of alternations is very high (though unfortunately in a bad sense), it is especially necessary to examine for this condition any elderly person with high blood pressure, renal disease, fibroid heart, etc., who is also liable to premature contractions.

Pulsus alternans must not be mistaken for—

(i.) *Dicrotic Pulse.* In this the apparent radial pulse rate is double that of the apex beat.

(ii.) *Alternating Premature Contractions* (*vide* p. 247). In this the smaller beat is followed by a definitely longer pause than the more forcible beat. In pulsus alternans the spaces between the beats are practically equal, though if *very* accurate measurements be taken (as with a very quickly-moving paper), it will be found that the smaller beat is followed by a *slightly shorter* pause than the more forcible one.

In any case of Pulsus Alternans the outlook is grave; when, however, the alternations are persistent, it is not too much to say that the prospects of life may be reckoned in months or weeks rather than years.

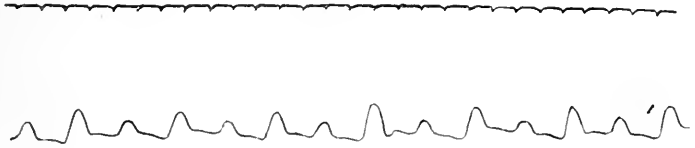


FIG. 49.—Polygraph tracing to show Pulsus Alternans.
Note the regular spacing of the beats despite their unequal force.

XII. CONGENITAL HEART DISEASE.

This may be produced by fœtal endocarditis or by imperfect development. In the latter group especially there are many possible varieties which are not compatible with life. The most common abnormality is a patent foramen ovale. This is unlikely to produce either signs or symptoms and is therefore of but little or no pathological significance when occurring as an isolated lesion. It is, however, very likely to be met with in combination with other abnormalities.

The following are the more usual congenital heart lesions and the murmurs accompanying them :—

(i.) *Patent Inter-ventricular Septum.* The murmur is very loud and rough, systolic in time, widely distributed, but of

maximum intensity in the third and fourth spaces to the left of the sternum. There is no thrill.

(ii.) *Pulmonary Stenosis.* A loud, rough systolic murmur, widely distributed, but of maximum intensity in the third left space 1 inch from the sternum. There is in addition a systolic thrill over the pulmonary area.

Pulmonary stenosis and patency of the interventricular septum are often concomitant affections.

(iii.) *Patent Ductus Arteriosus.* A persistent loud murmur running all through the cardiac cycle, increasing through systole and waning through diastole, best heard in the third left space close to the sternum. There is no thrill as a rule.

(iv.) Congenital lesions of the aortic, mitral, and tricuspid valves are relatively rare and present the same signs as do acquired lesions of the same valves.

The symptoms of congenital heart disease are fairly constant :—

(*a*) Cyanosis.
(*b*) Clubbing of the fingers and toes.
(*c*) Dyspnœa, either continuous or paroxysmal.
(*d*) Convulsive seizures.

The cyanosis is very striking when present, but it is not always to be demonstrated. In those cases in which there is cyanosis there is an increased number of red cells, an increase of hæmoglobin, and an increased specific gravity of the blood. This increased viscosity is possibly one factor in the production of the cyanosis, another factor being the necessarily poor pulmonary circulation in pulmonary stenosis.

Congenital heart disease is likely to be associated with other congenital abnormalities, both physical and mental, and also with malnutrition, dwarfed stature, and imperfect growth.

The diagnosis of congenital heart disease as a whole does not as a rule present any difficulty, but it may be impossible to distinguish between congenital lesions and hæmic murmurs in young children if there is no cyanosis, and it is often impossible to be dogmatic on the variety of congenital lesion that may be present.

XIII. ANGINA PECTORIS.

The pathology of the condition is still obscure; clinically it presents a definite symptom complex. In the majority of cases which have been examined post mortem, coronary artery disease has been revealed with coincident fibrosis or fatty degeneration; in other cases aortic valve lesions (stenosis or regurgitation) have been present, and in others acute inflammatory affections, or possibly aneurysm, of the first part of the aorta; while in a very few cases careful search has shown no lesion at all.

The great majority of sufferers are men of advanced years. Heredity, gout, and high arterial tension appear to be of etiological significance.

Each attack is characterised by sudden acute pain in the præcordial region, often radiating up into the left side of the neck and down the inner side of the left arm along the course of the intercosto-humeral nerve, or sometimes along the ulnar nerve. The right side may occasionally be implicated.

The patient is usually livid, anxious-looking, and covered with a clammy perspiration; at the same time he has a strong sensation of impending death.

The pulse is irregular, small and weak, and the arteries are often in a condition of tonic spasm.

Each attack lasts from a few seconds to a few minutes, or may recur in waves for several hours, and while it lasts the patient leans forward scarcely daring to breathe and supported by any convenient article. There is a copious eructation of wind at the close of a paroxysm, and sometimes a large quantity of pale urine is voided. The first attack is generally excited by exercise. Gradually the attacks become more and more frequent and are excited by increasingly trivial causes (sometimes, indeed, there is no apparent cause at all), such as indigestion, chill, or sudden movement.

The fact that true angina is very rare in women should help to differentiate it from attacks of præcordial pain and palpitations, which are so common in women round about the climacteric and those who are sufferers from atonic

dyspepsia and neurasthenia, especially if it is remembered that the first attack nearly always occurs after exercise and that the sense of impending death is a constant feature.

The combination of a dilated stomach and a persistently high arterial tension may closely simulate true angina, and may well be associated with a fatal syncope. A careful inquiry into the precise nature of the attacks of pain and the mode of origin of the initial attacks will usually suffice to prove the absence of angina pectoris.

XIV. AFFECTIONS OF THE PERICARDIUM.

Affections of the pericardium will be considered under three headings: (i.) Acute Fibrinous Pericarditis.

(ii.) Suppurative Pericarditis.

(iii.) Adherent Pericardium.

(i.) **Acute Fibrinous Pericarditis.** When this occurs in childhood, it is likely to be due to rheumatism, or possibly scarlet fever or even enteric fever; in older people gout, nephritis, or tuberculosis, are more likely causes.

The inflammation is accompanied by the formation of more or less sero-fibrinous effusion, which resolves satisfactorily in the majority of cases.

Symptoms. Owing to the extensive involvement of the myocardium in the inflammatory process the symptoms will include those of cardiac dilatation—that is to say, dyspnœa, an anxious expression, and a rapid, feeble pulse. Additional symptoms may be—

Pyrexia. This is an almost constant phenomenon.

Pain. Præcordial pain is common, but in young children it is often absent.

Dysphagia, from irritation of the œsophagus.

Cough, from pressure on the trachea.

Vomiting is a symptom of evil prognosis.

Delirium is common.

The Physical Signs:—

(1.) Before there is much effusion—

(*a*) An increase in the lateral extent of the cardiac dulness due to dilatation.

(*b*) A Friction Rub. This is a superficial scratchy,

continuous to-and-fro rubbing sound, not conducted in any particular direction and often modified by pressure with the stethoscope. It is first heard at the base in the majority of cases; then it can be detected at the apex and down the margins of the sternum. In a few cases it may only be audible during systole, and sometimes it is palpable as friction fremitus. As effusion develops the rub disappears, often to reappear as the effusion is absorbed.

(2.) When there is considerable effusion:—

(*a*) In cases that have been watched from their commencement the disappearance of the rub is the first evidence that there may be effusion. It must be remembered, however, that the rub will disappear from adhesion between the visceral and parietal layers of the pericardium, and also that the rub may continue to be audible at the base when there is a large effusion at the back of the pericardium.

(*b*) An increase of the already great area of cardiac dulness and an alteration in its shape. When the increase is entirely due to dilatation, the outline is more or less rounded; when there is much effusion, the outline becomes triangular, with the base of the triangle downwards. Important points are an *increase in dulness upwards* and *obliteration of the cardio-hepatic angle.*

(*c*) Muffling of the Heart Sounds. This is often absent, since there is a tendency for the heart to float up against the chest wall.

(*d*) Displacement upwards and a little outwards of the cardiac impulse. This sign, again, is by no means constant.

(*e*) The appearance of signs of compression of the lung below the angle of the left scapula, viz., dulness, impaired air entry, bronchophony, and often tubular breathing.

This area of compression does not extend far forward into the axilla and is important evidence of pericarditis, since it does not appear to be produced by pure dilatation of the heart. It is, however, often present in dry pericarditis, and is then due to under action of the left half of the diaphragm.

(*f*) Bulging of the præcordium and intercostal spaces to the left of the sternum. This is only likely to occur in children, and may also be caused by extreme dilatation.

(*g*) A pulsus paradoxus may be present (*vide* p. 220). In large effusions there is a tendency for the fluid to collect behind and below the heart. If the urgency of the case seems to warrant exploration either from a diagnostic or a therapeutic point of view, we have to decide where to explore.

The site usually recommended for paracentesis is the fifth left interspace three-quarters of an inch from the sternum. In our opinion it is more likely that a dilated right ventricle will be tapped in this situation, and we advise that exploration should be carried out either in the sixth left space at the junction of the anterior and middle thirds of the axilla or else in the sixth right space half an inch from the sternum in the cardio-hepatic angle, or perhaps in the chondro-xiphoid angle on the left side through the diaphragm and above the peritoneum.

It must be remembered that in very few cases of serofibrinous effusion is exploration justified.

Pericardial friction must be distinguished from :—

(*a*) *Pleuro-pericardial Friction.* This is produced by inflammation between the pleura covering the lung fringes and the pericardium that is in contact with them. There is a double rhythm in pleuro-pericardial friction, partly cardiac and partly respiratory; hence it is modified, or even abolished, if the patient holds his breath. It is usually heard along the left-hand border of the superficial cardiac dulness, and is not likely to be audible to the right of the middle line as well as in this region. True pericardial friction is nearly always heard to the right of the middle line at the base of the heart.

(*b*) *Aortic Valve Disease.* A double aortic murmur may sometimes be mistaken for pericardial friction, but its definite lines of conduction, the fact that it is not modified by pressure, its identity with the heart's sounds, and its greater remoteness should prevent this mistake.

The diagnosis of tuberculous from rheumatic pericarditis must rest upon the existence of tuberculosis elsewhere, especially in the left apex, and the lack of rheumatic history or valvular disease. In tuberculous pericarditis the effusion is sometimes blood-stained.

(ii.) **Suppurative Pericarditis.** This condition may be met with in pneumonia (either by direct extension or by metastasis), in septicæmia, pyæmia, and in mediastinal suppuration.

The diagnosis is but rarely made, since the effusion is purulent from the outset, and consequently, there may be no fibrin formation and so no rub.

The condition may be suspected in a case of pneumonia or pyæmia if there is evidence of pericardial effusion, such as abrupt increase in cardiac dulness with a more or less triangular outline and at the same time marked pulse acceleration and dyspnœa.

If pyo-pericardium is suspected the best way to clinch the diagnosis is to explore through an incision made parallel to and just below the costal margin on the left side about one inch from the base of the xiphisternum. The pericardium can be approached through the diaphragm, and if pus is present efficient drainage can be assured. An alternative method is to excise a portion of the sixth left rib close to the costo-chondral junction.

(iii.) **Adherent Pericardium.** This condition falls naturally into two groups :—

(*a*) Where the visceral and parietal layers of pericardium are tightly bound together, but where there is no undue adhesion between the outside of the pericardium and the chest wall or thoracic viscera.

(*b*) Where there are adhesions between the pericardium and the chest wall and the thoracic viscera. This condition is practically a chronic adhesive mediastinitis.

In the first group the significance depends largely upon the age of the patient. In an adult whose heart has done growing there may be no signs or symptoms of the condition until there is eventually some call for hypertrophy which the heart is unable to meet. In children, however, the development of the heart is seriously hampered and symp-

toms of dilatation and impairment of function are likely to be produced from the outset. In any case a heart so restricted cannot face any extra call so readily as when the pericardial space is not obliterated.

In the second group the condition is more serious, for the ordinary action of the heart is interfered with to a greater or less extent. Again, it is children who suffer most.

Symptoms of Adherent Pericardium. The symptoms are those of cardiac hypertrophy and dilatation, with eventually failure of the right ventricle. In any case where evidence of endocarditis, past or present, does not seem to be sufficient to account for the severity of the cardiac symptoms present, adherent pericardium should be thought of and the following physical signs carefully looked for.

Physical Signs of Adherent Pericardium :—

(*a*) An increase in the size of the heart (as evidenced by the increase in cardiac dulness) greater than can be explained by any valvular disease that may be present.

(*b*) A very diffuse wavy impulse, often with a soft tickling diastolic thrill.

(*c*) Fixation of the Apex Beat. This is tested by palpating the apex and at the same time rolling the patient over on to his left side. In normal hearts the apex will move out from 1 to 2 inches. This sign is of restricted value in children, since there is less room in their chests for the heart to swing.

(*d*) Systolic sucking in of the lower sternum and left costal cartilages.

(*e*) Systolic recession of the intercostal spaces in the lower chest at the apex, and also at the back and in the axilla. Of these the most conclusive is recession below the angle of the scapula in the tenth and eleventh spaces.

N.B. Both (*d*) and (*e*) may be produced by very large hearts in children in the absence of an adherent pericardium.

(*f*) A diastolic shock may be felt from the recoil of the chest wall after the systole is over.

(*g*) A presystolic or diastolic murmur, not accompanied by other evidence of valvular disease.

(*h*) If the right auricle is obstructed by band formation, progressive anasarca may be an early feature.

Despite the above list of signs, it remains to say that a positive diagnosis is often impossible, since many of the above signs may be absent in adherent pericardium or present when there is no lesion other than cardiac hypertrophy and dilatation.

XV. ARTERIO-SCLEROSIS (ATHERO-SCLEROSIS).

The pathology of this condition is obscure, but it would appear possible to recognise two main groups—the Toxic and Degenerative.

The *Toxic form* may be produced either by the direct actions of certain toxins on the arterial wall, acting presumably through the vasa vasorum ; or else the toxins may act indirectly by causing spasm in the muscle coat of the arteries, the result of which is to raise the blood pressure.

The *Degenerative form* may be considered as more or less physiological, since it has been suggested by Jorès that the capacity of the arteries for compensatory hypertrophy fails at the age of 40 to keep pace with the steadily rising blood pressure. Hence after this age more or less degeneration is to be expected at the points of maximum pressure (the ascending aorta) and the points which are structurally weakest (the points of bifurcation).

With the exception of the obliterating endarteritis of syphilis it seems probable that the primary change is in the middle coat of the vessel and that the obvious secondary changes in the intima are due to necrosis following lack of nutriment. It has been shown that the arteries are not nourished at all by the stream of blood passing through them.

Clinically arterio-sclerosis may be recognised by a high-tension pulse, a hypertrophied heart, and a persistently raised blood pressure. At the same time the arteries may be palpably thickened, hard, or tortuous.

There may be no **Symptoms** for years, but when there are symptoms they fall into three groups—Cardiac, Cerebral, and Renal.

The *cardiac manifestations* are breathlessness on exertion, coldness of the extremities, fainting attacks, and anginoid pains.

The *cerebral manifestations* are vertigo, headaches, epileptiform seizures, transient paralysis, noises in the head, and insomnia.

The *renal manifestations* are those of interstitial nephritis (*vide* p. 446).

XVI. AORTIC ANEURYSM.

It is customary to describe aneurysms as being either fusiform, saccular, or dissecting. In the great majority of cases when aneurysm is discussed "saccular" aneurysm is meant. The fusiform variety is rare, since a fusiform dilatation of the ascending aorta is very seldom worthy of being considered an aneurysm either clinically or pathologically. Dissecting aneurysm is also rare; it is caused by a rent in the intima followed by an inrush of blood into the media, which splits the wall of the aorta longitudinally for a varying distance. Under these circumstances life is rarely prolonged for more than a few hours, death being preceded by rupture of the external coats either into the pericardium or elsewhere. The only symptom is agonising precordial pain, which may be felt to spread along the course of the aorta.

The following remarks apply principally to saccular aneurysms of the aorta:—

The signs and symptoms of aortic aneurysm differ very greatly according to what part of the aorta is affected. It is best therefore to consider the general symptomatology of aneurysm and then to discuss shortly the individual aneurysms of the different parts of the aorta.

(i.) **General Symptomatology.** The symptoms produced are due to the pressure of the aneurysm on various adjacent structures and it is impossible to dissociate completely symptoms from signs.

Pain may be dull and aching from pressure on the chest wall, it may be neuralgic from pressure on the nerve trunks, or it may be lancinating and paroxysmal

from erosion of the vertebræ and implication of the posterior nerve roots.

Anginoid pain (*vide* p. 257) may be caused by aneurysm of the intra-pericardial portion of the aorta.

Pressure of the sac upon the trachea may produce cough, as well as dyspnœa, stridor, bronchitis, bronchorrhœa, and hæmoptysis.

Pressure on the root of the lung may produce cough and pulmonary collapse.

Pressure on the left recurrent laryngeal nerve may produce a brassy cough, hoarseness, or loss of voice from abductor paralysis of the left vocal cord ; or irritation of the same nerve may produce severe paroxysmal dyspnœa.

Pressure on the vagus nerve may produce dyspnœa, vomiting, or hiccough.

Pressure on the left phrenic nerve may paralyse the left half of the diaphragm.

Pressure on the superior vena cava or left innominate vein may produce venous engorgement without pulsation or respiratory modification. This phenomenon is unilateral when the left innominate vein is concerned.

Pressure on the sympathetic nerve fibres may produce unequal pupils (dilated from irritation and contracted from paralysis), and also unilateral flushing, sweating and bristling of the hair on the face.

Pressure on the œsophagus may produce dysphagia.

A consideration of the anatomy of the aorta makes it obvious that the majority of these pressure effects can only be produced by an aneurysm of the transverse part of the arch or one at the top of the descending atora. These parts of the aorta are, however, deeply situated, and aneurysms of them are not so likely to give rise to physical signs as are aneurysms of the ascending aorta which is more superficial and also has more room for expansion.

(ii.) **The Physical Signs of Aortic Aneurysm** may be :—

(*a*) *Visible or Palpable Pulsation.*—This is evident above the base of the heart, more often to the right of the sternum than to the left ; it should be expansile, and may only be apparent on careful examination made tangentially and in a good light. On the other hand,

there may be an obvious pulsatile tumour projecting through the chest wall.

(*b*) *An Increased Area of Dulness*, especially to the right of the sternum in the first, second, and third spaces.

(*c*) *A Thrill*, systolic in time, over the upper part of the sternum.

(*d*) *A Low-pitched Ringing Second Sound* over the aortic area. This is one of the most important signs of aneurysm. It is produced in the same manner as is the next sign.

(*e*) *A Diastolic Shock*, due to the recoil of the distended sac.

(*f*) *A Systolic Murmur.* The chief importance of the systolic murmur is that it should be heard over the aorta rather than at the aortic valve area.

(*g*) *A Tracheal Tug.* To elicit this the patient should sit in a chair with his neck extended, and the observer should stand behind the chair and place his thumbs, one from each side, under the patient's cricoid cartilage which should be firmly but gently pressed upwards. The tug will be felt as a definite systolic pull. The cardiac pulsation may sometimes be felt in this manner when there is no aneurysm, and many aneurysms of the transverse arch fail to give the sign.

(*h*) *Inequality of the Pulses.* The left radial pulse may be retarded and of smaller amplitude than the right where there is an aneurysm distal to the innominate artery but proximal to or involving the left subclavian. This phenomenon should be checked by observation of the brachial and carotid pulses, in order to exclude an aberrant radial artery.

(*i*) *Signs of Collapse* in some part of the left lung from obstruction to the left bronchus (the right lung is more rarely affected).

(*j*) Unless there is aortic valve disease, the heart is not enlarged to percussion and the position of the impulse is unaltered.

In reviewing the signs and symptoms detailed above it is evident that, with the exception of an expansile tumour and possibly the altered second sound and the diastolic shock, there are none that are pathognomonic of aneurysm.

When, in addition, we remember that in many cases of aneurysm there are neither signs nor symptoms to guide us it is plain that the diagnosis of this condition may be of the utmost difficulty.

(iii.) **Aneurysm of the Intrapericardial Portion of the Aorta.** There may be no symptoms or signs. Death often occurs at an early stage from rupture into the pericardium. Occasionally the aneurysm ulcerates into the superior vena cava or into the pulmonary artery. In the former case there is sudden extreme congestion of the veins in the head and neck, with severe throbbing headache, œdema, and dyspnœa: a continuous buzzing murmur, sometimes

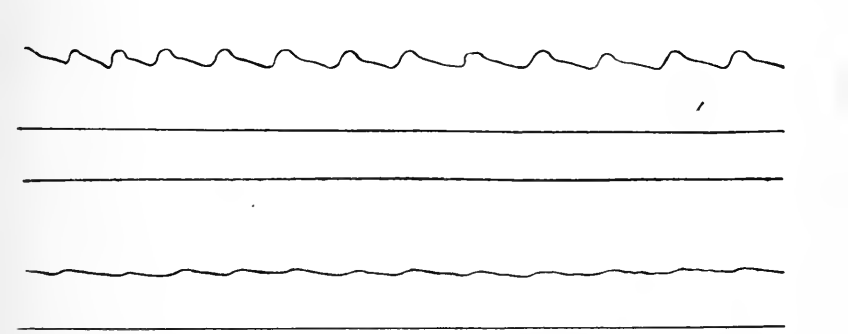


FIG. 50.—Pulse tracings from the right and left Radial Arteries of a man suffering from Aneurysm of the transverse aortic arch. The upper tracing is from the right wrist. Note the inequality of the tracings and that the left pulse is nearly obliterated.

accompanied by a thrill, may be apparent in the aortic area. In the latter case there is sudden precordial pain with urgent dyspnœa; at the same time a continuous murmur like that described as occurring with patent ductus arteriosus is audible.

The most likely manifestations of aneurysm in this situation are—angina pectoris, evidence of aortic valve disease, especially aortic reflux, an increased area of dulness in the second left space, and pressure on the superior vena cava.

(iv.) **Aneurysm of the Ascending Aorta between the Pericardium and the Innominate Artery.** This is the aneurysm

of physical signs, such as increased dulness to the right of the sternum in the second and third spaces, a pulsating expansile tumour in the second right space, a low-pitched ringing aortic second sound, and a diastolic shock. A systolic murmur is often present, but by itself it is of no diagnostic value. Possible pressure effects are pain from pressure on the chest wall, venous congestion from pressure on the superior vena cava, and occasionally collapse of lung as shown by loss of breath sounds, with at first unaltered percussion note from pressure on the root of the right lung.

(v.) **Aneurysm of the Transverse Aortic Arch.** This is the aneurysm of symptoms, due to pressure effects on the trachea, left bronchus, œsophagus, left recurrent laryngeal nerve, sympathetic nerves, or even the left innominate vein. If the aneurysm grows to a large size, there may be a heaving pulsation at or above the manubrium sterni, with a considerable increase of dulness to percussion in the second spaces. A systolic murmur audible about the episternal notch and not heard at the aortic area is suggestive, as is a diastolic shock. Unequal pulses and a tracheal tug are important diagnostic points of aneurysm in this position.

(vi.) **Aneurysm of the Descending Aortic Arch.** Here there are often neither signs nor symptoms for a long time. This aneurysm may give a tracheal tug, signs of pressure on the left bronchus, left recurrent laryngeal nerve or œsophagus, and, most important of all, severe root pains, indicative of erosion of the fourth and fifth dorsal vertebræ.

(vii.) **Aneurysm of the Descending Thoracic Aorta** gives the same signs as the above, except that tracheal tug and pressure on the left bronchus or recurrent laryngeal nerve are not met with, and the root pains will indicate involvement of lower vertebræ. In very rare cases a pulsating tumour has appeared in the back.

(viii.) **Aneurysm of the Abdominal Aorta.** This is usually situated opposite the second lumbar vertebra where the cœlic axis is given off.

A definite pulsating tumour may be formed if the bulge is forwards. It is important not to mistake transmitted pulsation from an excitable aorta; the test is to ascertain

whether the pulsation is expansile. Not infrequently the aneurysm grows from the back of the aorta and erodes the vertebral column, when root pains are the first evidence of illness.

Differential Diagnosis. In considering the differential diagnosis of aneurysm it is important to remember the factor played by syphilis in its production and the rarity with which women are affected. Over 90 per cent. of aortic aneurysms occur in syphilitic patients, so that a *negative* Wassermann reaction is very much against the diagnosis of aneurysm. The most important method of diagnosis at our disposal is undoubtedly the fluoroscope. By means of the X-ray screen it is possible to detect a *pulsating tumour* in the course of the aorta in nearly every case of aneurysm.

Thoracic aneurysm may be indistinguishable clinically from mediastinal new growth. The X-rays may settle the question at once, but if they do not do so it is possible to estimate to some extent the probabilities of the case by the consideration of the following points :—

(*a*) A low-pitched ringing aortic second sound and a diastolic shock are both very much in favour of aneurysm.

(*b*) Relief of symptoms, such as pain and dyspnœa, by rest in bed, and especially the re-aeration of hitherto collapsed lung, is in favour of aneurysm.

(*c*) The relief of pain by potassium iodide is only slightly in favour of aneurysm, since many inflammatory processes are temporarily benefited by this drug.

(*d*) Abductor paralysis of the left vocal cord is slightly in favour of aneurysm.

(*e*) Retardation and diminution of the left radial pulse, in the absence of enlarged axillary glands, is in favour of aneurysm.

(*f*) A definite tracheal tug is greatly in favour of aneurysm.

(*g*) Enlarged supra-clavicular or axillary glands or the evidence of primary malignant disease anywhere is in favour of new growth.

(*h*) Enlarged superficial thoracic veins, with the blood-stream running from above downwards, is slightly in favour of new growth.

(*i*) Displacement of the heart is in favour of new growth.

(*j*) Pleurisy is in favour of new growth.

(*k*) Persistent pain in the chest, not lancinating root pain, is slightly in favour of new growth.

(*l*) A negative Wassermann reaction is very much against aneurysm.

(*m*) The age of the patient, the evidence of arterial degeneration, and the length of history, may afford considerable help in excluding new growth.

Aortic Regurgitation with marked pulsation of the aorta may sometimes be mistaken for aneurysm. The X-ray screen will show whether there is dilatation of the ascending aorta or whether true aneurysm is present. It should be remembered that *per se* aneurysm does not cause cardiac hypertrophy, so that marked enlargement of the left ventricle would favour primary aortic reflux.

It should be remembered that an aneurysm of the first part of the aorta may affect the aortic valves and so give rise to reflux.

Gastric Crises and *Girdle Pains* may suggest the possibility of abdominal aneurysm. The other signs of tabes dorsalis should, however, prevent error. The fluoroscope should prevent root pains due to primary vertebral or meningeal causes being attributed to aneurysm.

CHAPTER II

DISEASES OF THE RESPIRATORY TRACT AND PLEURA

I. ANATOMY.

The trachea is $4\frac{1}{2}$ inches long; it commences at the cricoid cartilage, which is at the level of the sixth cervical vertebra, and terminates opposite the fourth dorsal vertebra by dividing into the right and left bronchi. Its course is downwards and distinctly backwards, a fact which is emphasised in babies and is of importance in tracheotomy. The arch of the aorta crosses in front of the lower part of the trachea and turns downwards above and behind the left bronchus. The left pulmonary artery forms at first a superior and then an anterior relation of the left bronchus. The left recurrent laryngeal nerve, as it winds round the arch of the aorta from before backwards, forms a superior relation of the left bronchus. The apices of the lungs reach for about $1\frac{1}{4}$ inches above the clavicles; the bases are somewhat hollowed out by the domes of the diaphragm. The anterior borders of the lungs run from the apex—that is to say, a point towards the posterior border of the sterno-mastoid muscle $1\frac{1}{4}$ inches above the clavicle—down through the sterno-clavicular articulations to reach the mid-point of the sternum at the level of the second cartilages. From this point the lines run side by side till the level of the fourth costal cartilages is reached, where they diverge. The right-hand line is continued down the middle of the sternum to the level of the fifth cartilage, when it curves rather abruptly outwards to the sixth right chondro-sternal articulation. The left-hand line curves, with its convexity forwards, out to the fourth left chondro-sternal junction, and thence, following the edge of the superficial cardiac dulness, proceeds in another curve to the sixth left costo-chondral junction (*vide* Fig. 40).

The lower borders of the lungs are represented by slightly

convex lines running from the sixth right chondro-sternal junction and the sixth left costo-chondral junction respectively to the spinous process of the tenth dorsal vertebra. The lower borders of the lungs will be at the seventh rib in the anterior axillary line, at the eighth rib in the posterior axillary line, and at the tenth rib in the line of the scapular angle.

The line dividing the upper from the lower lobe is obtained by joining the spine of the second dorsal vertebra with the sixth rib in the nipple line. The upper border of the middle lobe on the right side is indicated by a line joining the mid-point of the interlobar line above mentioned and the fourth right chondro-sternal junction.

The pleura extends for at least one inch below the level of the lung all round.

II. EXAMINATION OF THE CHEST IN HEALTH AND DISEASE.

(a) **Inspection.** The spherical chest of infancy develops into an ellipse with its long axis transverse by the third year. Both halves of the chest should be symmetrical one with the other, though the circumference of the right side may be a quarter of an inch more than the left.

Asymmetry of the chest can be demonstrated by the cyrtometer, which is composed of two lengths of malleable lead tubing hinged together. The hinge is applied to a spinous process and the lead is accurately moulded to the chest. The instrument can be removed and the outline traced on paper for permanent record.

The chest may be symmetrical and yet show the following deviations from the normal :—

The Flat Chest shows a diminution in the antero-posterior diameter ; the costal cartilages are unusually straight, and sometimes the sternum is more or less depressed. The condition is congenital and appears to predispose to pulmonary tuberculosis.

The Winged Chest is small in all dimensions, but especially antero-posteriorly ; the ribs and shoulders drop and the

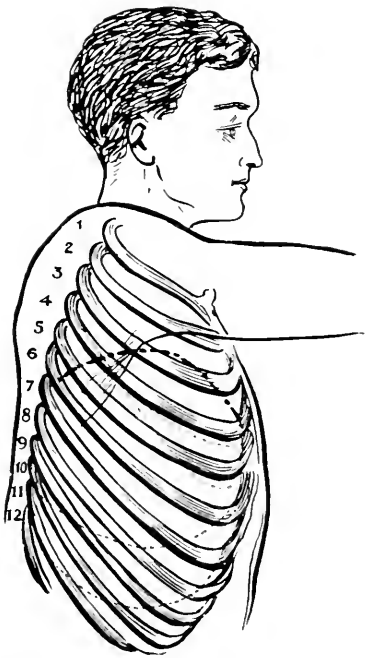


Fig. 51. – Diagram to show :

(i.) The curve assumed by the upper level of free fluid in the Pleura (Damoiseau's curve) : Dotted black line.

(ii.) The lines dividing the lobes of the right lung : Dotted red lines

(iii.) The lower level of the Lung : Upper blue line.

(iv.) The lower level of the Pleura ; Lower blue line.

scapulæ are thrown outwards. The condition is congenital, and is sometimes associated with pulmonary tuberculosis.

The Pigeon Chest is almost triangular in transverse section owing to undue prominence of the sternum; it is often associated with *Harrison's sulcus*, which is a deep transverse groove at the level of the fifth, sixth, and seventh ribs. Both these conditions are due to forced inspiration during infancy and childhood when the bony structures are still soft. Predisposing causes are whooping-cough, broncho-pneumonia, and rachitis.

Rachitis tends to produce a vertical groove at the costo-chondral junctions owing to the pull of the diaphragm on the softened epiphyses of the ribs and also a beading of the ribs (rickety rosary) at the same place.

The tendency of long-continued forced inspirations is to increase the capacity of the chest to its maximum. This is well seen in emphysema, where the chest becomes circular or barrel shaped and the cervico-dorsal region of the spinal column becomes bowed forwards.

Unilateral deformities of the chest may be produced by spinal curvatures, by chronic pulmonary disease, or by over-action of one lung; localised bulgings by aneurysm or new growth, general bulging by a large pleural effusion, especially if purulent or by pneumothorax.

Shrinking or wasting is especially noticeable at the apices, and is significant of fibro-caseous tuberculosis, fibroid lung, or adherent pleura. Complete collapse of one lung, as from pressure by an aneurysm or tumour, may produce a distinct falling in of one half of the chest.

Inspection of the chest also shows the movements and method of expansion. The ordinary respiration rate is 16 to 18 per minute for men and 20 to 22 for women: in children it is more rapid; in infancy it is 30 to 35 per minute. Men breathe largely by the exercise of the diaphragm (abdominal type of respiration); young children use this method almost exclusively; women, on the other hand, possibly owing to the use of corsets, breathe noticeably with their intercostal muscles (costal type). Hence bilateral impairment of expansion at the

apices is more significant in women than in men. The maximum expansion of the chest should be at least one twelfth of its mean girth greater than the minimum expansion.

Local impairment of expansion suggests local disease of the lung or pleura ; it must be remembered, however, that in old or debilitated people, especially those who are bed-ridden, there is often a very limited apical expansion from prolonged disuse.

The nerves to the intercostal muscles may be paralysed from spinal lesions, with the result that more strain is thrown on the diaphragm, and conversely the action of the diaphragm may be seriously interfered with by ascites, sub-phrenic abscess, abdominal tumours, etc., with the result that the accessory muscles of respiration are called into play.

Dyspnœa signifies difficulty of respiration and is a term that is used rather loosely ; thus in lobar pneumonia, though there is always a great increase in the number of the respirations (tachypnœa), there is very often no difficulty in drawing the breath. In cardiac conditions and renal disease, on the other hand, true dyspnœa is extremely common.

Dyspnœa may be inspiratory or expiratory ; in the former case, if accompanied by stridor, it signifies some definite obstruction to air entry. Expiratory dyspnœa is seen principally in emphysema and bronchial asthma and bronchopneumonia in children. Orthopnœa is an advanced stage of dyspnœa in which the patient cannot breathe unless he is sitting up.

Air-hunger is seen in certain toxic conditions, such as diabetic coma and other forms of acidosis, and is characterised by very deep and prolonged inspirations.

In adults the normal respiratory cycle consists of inspiration, expiration, and then a pause. In infants the cycle is expiration, inspiration, and then pause.

This infantile type of respiration persists for about six months, but any child up to two years or more who gets an acute pulmonary lesion often reverts to the infantile type of respiration for the time being.

Obstructive stridor, as mentioned above, is mainly inspiratory, and must not be confounded with the *expiratory grunt*, which is so characteristic a feature of broncho-pneumonia in young children.

Cheyne-Stokes respiration consists of a series of shallow respirations which gradually become more and more frequent, deeper and noisier, till they are positively distressing; they then subside in the inverse order and terminate in a complete cessation of respiration (apnœa) which may last for twelve to fourteen seconds. The whole cycle, including the pause, usually occupies about fifty seconds. The phenomenon is seen in uræmia, coma, cerebral disease, myocardial disease, opium poisoning, sometimes in acute infections, and occasionally in healthy children who are asleep. As a rule Cheyne-Stokes respiration is an indication that death is at hand.

Slowing of the respiration rate is common in unconsciousness and collapse; it is also a manifestation of digitalis and other drug poisoning.

Increased frequency of respiration is usual in fevers; it is also common where there is dyspnœa that is not obstructive, in pulmonary, cardiac and renal disease, and in many neurotic conditions.

(**b**) **Palpation**. This is carried out with the flat of the hand: it is of use in confirming variations in degrees of mobility as suspected by inspection, and also for testing the tactile vocal fremitus. Sometimes friction sounds may be palpable (just as are certain cardiac murmurs), and coarse rhonchi can often be felt.

The *tactile vocal fremitus* represents the vibrations of the spoken voice conducted from the larynx through the air in the bronchial tubes to the chest wall and so to the examining hand. In health it is slightly greater over the right lung than over the left.

In women and children it may be very slight. For eliciting it, resonant words such as nine, ninety-nine, and nine hundred and ninety-nine are the best.

Tactile vocal fremitus is increased over solid lung if the solid tissues are homogeneous; thus it is loud in pneumonia, but often diminished over a tumour; it is, of course, not well

perceived unless the bronchi communicating with the particular affected area are patent. It may be increased over a cavity which is near the surface and which communicates with a bronchus.

In pleural effusion, empyema, pneumo-thorax and very thickened pleura it may be absent.

It is sometimes more convenient to use the ulnar border rather than the flat of the hand for investigating the tactile vocal fremitus.

In all cases bilateral variations from the normal are not nearly so important as unilateral, and it must be borne in mind that extreme adiposity may completely cut off the vibrations.

(c) **Percussion**. The middle finger of the left hand (except for left-handed individuals) is the best pleximeter and the middle finger of the right hand is the best plessor. The pleximeter finger must be firmly applied to the chest wall and must be struck at right angles on the part between the base of the nail and the terminal joint by the pulp of the plessor, the nail of which should be kept very short. The blow should be short and staccato, delivered with a free wrist, from the wrist only, and never from the elbow or shoulder; it must be light yet firm.

The force of the blow must vary in different cases, necessarily being greater in fat or muscular subjects, but as a general rule, and always in children, light percussion affords more information than heavy.

In outlining an organ the pleximeter finger must be kept parallel to the assumed edge of the viscus. The object of percussion is to compare the note obtained (1) with the normal note for that position, (2) with the corresponding area of the opposite side, but, in addition to this, valuable information is gained from the feeling of resistance obtained by the act of percussion in different conditions of health and disease.

Percussion sounds are described as being resonant, hyperresonant, tympanitic, dull or flat.

A resonant note is obtained over healthy lung tissue and is heard at its best towards the apex of the axilla.

A hyper-resonant note is characteristically obtained over emphysematous lungs.

A tympanitic note differs from hyper-resonance in that the air producing it is not sub-divided into numerous small compartments as in healthy lungs ; this imparts a drum-like character to the percussion note which is best appreciated in pneumo-thorax, or over a cavity which is near the surface and which communicates with a patent bronchus.

Where the air has no outlet, as in closed cavities and some cases of pneumo-thorax, the pitch of the note varies directly with the tension of the walls. The result may be a dull note in cases where the tension is very high.

Skodaic resonance or sub-tympany is a peculiar boxy note, rather like that normally obtained over the trachea when the mouth is open ; it is characteristically present over the lung above a good-sized pleural effusion and also where there is a central consolidation separated from the chest wall by a thinnish layer of aerated lung, as in the early stages of many cases of pneumonia.

A completely dull note, as normally heard over the liver, depends for its generation entirely on the impact of the percussion blow ; there is no vibration imparted to the air as when the healthy lungs are percussed. In addition the resiliency or power of vibration of the chest wall may be completely or partially destroyed, and it is this latter factor which explains the difference (even more of feel than of sound) which obtains when percussion is made over solid lung and over pleural effusion. Both notes may be absolutely dull, but the sensation where there is fluid is that of striking a very thick blanket, and where there is consolidation of striking a very thick board. The dead, toneless sound of the note over fluid is called "flat" and may be nearly approximated to by percussing the thigh muscles.

The *bruit de pot fêlé* or crack-pot sound is a metallic chinking sometimes heard when percussion is made over a cavity which communicates with a bronchus when the patient's mouth is open. It is also to be heard on percussing the chests of healthy infants who are crying.

In percussing a chest it is important to remember that

the note over the right apex is normally rather higher pitched (duller) than that over the left, and that the liver dulness is perceived at the sixth rib on the right side in front, at the eighth rib in the mid-axillary line, and at the ninth rib in the line of the scapular angle.

On the left side the presence of the heart must be allowed for (*vide* p. 207), and possibly also the spleen (*vide* p. 178), while in certain cases a distended stomach may produce a tympanitic note extending quite high up into the left axilla.

The lower border of lung resonance behind reaches to the tenth rib in expiration and to the eleventh rib in forced inspiration on the left side and about half an inch higher on the right side. In emphysema these limits may be considerably exceeded.

If the vertebræ are percussed it will be found that the upper four dorsal vertebræ are dull, while the lower eight give a more or less resonant note (osteal resonance).

(**d**) **Auscultation**. Three classes of phenomena should be investigated by auscultation :—

(i.) Breath Sounds.

(ii.) Voice Sounds.

(iii.) Adventitious Sounds.

It does not matter what method of auscultation is employed ; for most purposes the binaural stethoscope, provided that the ear-pieces are of sufficient size and the pressure in the ears enough but not too much, is the best. It is well to remember that direct auscultation will often reveal as much as a stethoscope, and sometimes more, especially in the case of children.

(i.) Breath Sounds. *The vesicular breath sound* as heard over healthy lung tissue is composed partly of the breath sounds generated at the glottis and modified by conduction through the spongy lungs, and partly of the murmur manufactured locally in the terminal bronchioles and pulmonary alveoli. It consists of a soft, rustling inspiratory sound as long as the act of inspiration, followed *without any pause* by a still softer and much shorter expiratory sound the length of which is not more than one-third that of the expiratory act.

Bronchial breathing is heard normally over the trachea and large bronchi and pathologically over areas of consolidation or cavities. It represents the unmodified glottic breath sounds conducted by the air in the trachea and main bronchi. It consists of an inspiratory and expiratory sound of equal length, of higher pitch than the normal, and separated by a distinct interval. It can be heard in healthy chests over the bronchi at their origin from the trachea.

Tubular breathing, or extreme bronchial breathing, is heard over the larynx in health and over superficial consolidations and cavities or collapsed lung to which a patent bronchus runs. It is higher pitched than true bronchial breathing and has a characteristic whiffing sound. The expiration may be even longer than the inspiration, and is frequently of considerably higher pitch. When consolidation is developing a "tubular whiff" imparted to a lengthened expiratory sound and a pause between inspiration and expiration are often the first detectable signs.

Prolongation of expiration without alteration of pitch and without a pause is characteristic of emphysema.

Diminution or absence of breath sounds may occur in the very fat or the very muscular, but if it still persists in spite of forced respiration it is likely to be caused by thickened pleura, pleural effusion, pneumo-thorax, collapse of lung, new growth, or, in some cases, by massive consolidation where the main bronchi are all choked up.

Cavernous breathing is bronchial breathing of very low pitch and hollow quality. It indicates an empty cavity of a size at least that of a walnut.

Amphoric breathing resembles cavernous save that there is a resonant quality which can be simulated by blowing across the mouth of an empty bottle. It is significant of pneumo-thorax or a large superficial cavity communicating with a patent bronchus. It may be accompanied by a definite metallic tinkle, which is accentuated on speaking or coughing.

Broncho-vesicular breathing, as its name implies, indicates a mixture of bronchial and vesicular breath sounds. It can normally be heard in the second interspace close to the

sternum and better on the right side than on the left. This type of breathing when heard elsewhere is often significant of early tuberculous change, and tuberculosis is often diagnosed in healthy people because the normal occurrence of broncho-vesicular breathing in the second space near to the sternum is not sufficiently borne in mind.

Harsh breathing is ordinarily present in young children, otherwise it may be one of the earliest signs of inflammatory change. It must not be forgotten that the breath sounds at the right apex are normally rather harsher and of slightly higher pitch than are those at the left.

(ii.) Voice Sounds. The sound of the spoken voice is generated at the glottic aperture and, as heard through the stethoscope, is modified very much by the conditions of the lung or pleura. In health, though loud and resonant, it is indistinct and remote. The voice sounds are increased where there is solid lung and also where there is an empty cavity communicating with a patent bronchus. They are diminished where there is pleural effusion, pneumo-thorax, pulmonary œdema, or collapse of lung.

Bronchophony, or abnormal loudness and apparent nearness of the voice sounds, either spoken or whispered, is characteristic of an inflammatory consolidation or an open cavity.

Pectoriloquy is really an extreme degree of bronchophony in which the spoken or whispered voice in addition to being loud and close is also particularly clear and distinct. It is defined as representing the clear transmission of articulate sound. It is most likely to be heard over an open and superficial cavity or a superficial patch of consolidation.

A *metallic tinkle* accompanying the spoken voice is only heard in cases of pneumo-thorax or very large and quite superficial cavities.

Ægophony is a nasal twang or bleating sound imparted to the voice about the upper level of a pleural effusion and also sometimes in the neighbourhood of the advancing edge of an inflammatory process, such as pneumonia.

(iii.) Adventitious Sounds. *Rhonchi* are produced during

the respiratory cycle by the forcible passage of air past some mucous plug which is obstructing the lumen of the tube and temporarily converts it into a whistle. The result is a snoring or whistling sound devoid of all character of moisture. Rhonchi are therefore classified into sonorous or sibilant, as the case may be.

Râles are produced by the passage of air through moist secretion ; hence they are essentially wet, bubbly sounds and may be coarse or fine, according to the calibre of the tube concerned.

Crepitations are produced by the foreing open of the finest tubes and alveolar spaces the walls of which are stuck together by more or less sticky secretion.

III. COUGH AND SPUTUM.

(a) **Cough**. The ordinary purpose of a cough is to remove from the upper air passages—that is to say, fauces, larynx, trachea, or main bronchi—either a foreign body or excess of secretory products. The desire to cough is also induced by irritation, as from inflammation or pressure by new growth in these situations, before there is any material to be removed, or reflexly by irritation in regions which are remote from the larynx, trachea, or bronchi. It is worthy of note that morbid processes affecting the intrinsic lung tissue do not *per se* cause cough, and it is when, as the result of such morbid processes, the bronchi become involved that cough supervenes.

In distinct contrast to the above is the cough which is at once induced reflexly by any lesion of the pleura. Other varieties of the reflex cough may be instanced :—The stomach cough of chronic gastritis, which, however, is not infrequently due to associated pharyngitis ; the barking cough of puberty ; the cough sometimes present in association with lesions of the external auditory meatus, which is due to irritation of the auditory branch of the vagus nerve ; the cough not infrequently present when there is turbinal hyperæsthesia ; and, lastly, the purely hysterical cough of the bashful.

It is convenient sometimes to classify cough into two main

groups :—(1) The cough productive, and (2) the cough unproductive. Both varieties may be seen in the different stages of an ordinary lobar pneumonia. In the early stages there is an unproductive cough because of the existing pleurisy ; in the later stages the cough is productive and is one of the methods by which the disintegrating products are got rid of during the stages of resolution. In general it may be stated that the cause of a productive cough will be found by examination of the thorax, such conditions as bronchitis, bronchiectasis, œdema, and tuberculosis each possessing fairly characteristic physical signs.

Those coughs which are produced by pressure, as from new growth or aneurysm, at first are commonly unproductive, but later, when stenosis has occurred and permitted more or less damming up of secretion, expectoration may be profuse. Under such circumstances the physical signs may be localised to one lung or to part of one lung.

In conclusion, certain common types of cough may be enumerated :—The paroxysmal cough of pertussis, which terminates in the characteristic whoop ; the brassy cough of aneurysm and sometimes of mediastinal tumour ; the hoarse barking cough of laryngitis ; and the hollow racking cough of advanced tuberculosis.

(b) **Sputum.** For diagnostic purposes examination of the sputum must be considered from four points of view :— (i.) Naked-eye appearances ; (ii.) amount ; (iii.) odour ; (iv.) microscopical characteristics.

The sputum may be mucoid, muco-purulent or purulent ; it may be white, yellow, black, or mixed in varying proportions with blood ; it may be frothy or entirely free from admixed air. If placed in water it may sink to the bottom in nummular masses, a feature which is commonly observed in tuberculosis and resolving pneumonia. In chronic bronchitis and emphysema the sputum is muco-purulent and characteristically white and frothy. In bronchiectasis and advanced tuberculosis it is profuse, purulent, and usually malodorous. In œdema of the lungs the sputum is profuse, watery, and stained the colour of prune juice ; while in cases of advanced back pressure from mitral disability cells may be present

which contain numerous granules of brown pigment. In gangrene of the lung the sputum is profuse and has so characteristic an odour that mistake is well-nigh impossible. In malignant disease of the lung the sputum is said to resemble red-currant jelly, and it is likened to anchovy sauce in those cases in which a tropical abscess of the liver has ulcerated into a bronchus.

The expectoration of the white fibrinous cast of a bronchial tree is only met with in fibrinous bronchitis. The sputum of those who live in towns is always more or less blackened by carbonaceous particles, a characteristic which is emphasised in miners, coalheavers, stokers, etc.

The microscopical appearance of sputum may be of the utmost diagnostic value; by this means the causative organism—*e.g.*, pneumococcus, streptococcus, or micrococcus catarrhalis—may be determined after suitable cultivation, while the tubercle bacillus can often be demonstrated in film preparations.

In addition the following features may be noted. Curschmann's spirals, which are mucinous casts of the small bronchioles, and eosinophile cells are commonly present in bronchial asthma. Dittrich's plugs, which are minute, yellowish, malodorous masses of fatty acids and bacteria, are characteristic of advanced decomposition either in lung tissue or in old-standing cavities. Elastic fibres, which may be demonstrated after boiling sputum in 10 per cent. caustic potash, are pathognomonic of gangrene or tuberculosis. Calcareous particles may be expectorated in cases of healed tuberculosis.

(c) **Hæmoptysis.** In considering a case of hæmoptysis it is necessary first to establish whether the blood is really derived from the lungs; for this purpose a careful examination of the mouth, nose, fauces, and naso-pharynx is necessary. In this manner it may be possible to exclude gum-sucking, a common trick of malingerers; hæmorrhages from a pharyngeal vein; new growth of the mouth, fauces, pharynx and accessory sinuses; ulceration about the posterior nares; and lastly adenoid vegetations, which are a common cause of blood-stained sputum and "blood on the pillow" in young children.

It is also necessary to establish that the condition is not hæmatemesis. Careful questioning will usually suffice ; but it is important to remember that in profuse hæmatemesis the blood is often bright red at first, and also that in true hæmoptysis much blood may be swallowed, to be vomited subsequently in a black and altered condition. As a general rule the blood in hæmoptysis is bright red, frothy from admixed air and more or less mixed with sputum ; its reaction to litmus paper is alkaline, whereas in hæmatemesis it is acid from admixed gastric secretions. In its mildest form there will only be an occasional bright-red streak on various portions of phlegm. In more severe cases the patient will usually describe the first symptom as a tickling in the throat, which is followed almost immediately by a gush of bright blood from the mouth.

The blood in hæmoptysis is not always bright red ; a tuberculous cavity may be filled with blood-clot, which in time becomes altered and may be coughed up later in black jelly-like masses ; and the same effect is produced in the later stages of extensive pulmonary infarction. By far the most common cause of true hæmoptysis is pulmonary tuberculosis ; it is, however, well to remember some other if less common causes.

Slight hæmoptysis of the streaky variety is by no means rare in bronchitis, both acute and chronic, while in bronchiectasis there may be quite severe hæmorrhage. The rusty sputum of lobar pneumonia has been described elsewhere. Ulceration of the trachea or bronchi, whether simple, syphilitic, or malignant, may give rise to hæmoptysis.

Thoracic aneurysm may leak into the trachea producing hæmorrhage, which though usually fatal is not necessarily so at once ; while hæmoptysis from back pressure or infarction in cases of mitral disease is a matter of every-day experience.

IV. DISEASES OF THE LARYNX.

(i.) **Simple acute laryngitis** is associated with more or less severe hoarseness, sometimes proceeding to entire loss of voice and unproductive painful cough. The disease is

usually of fairly rapid onset, and the laryngoscope shows no changes other than a varying degree of congestion of the laryngeal mucosa. There is commonly an associated catarrh of the naso-pharynx, trachea, and bronchi.

(ii.) **Simple chronic laryngitis** is usually found in those who habitually overuse their voices, and especially in those who have no knowledge of elocution. Predisposing factors undoubtedly are excess of alcohol or tobacco and chronic nasal obstruction. The symptoms are a chronic hawking cough and a more or less permanent huskiness of voice.

Tuberculosis, syphilis, and malignant disease of the larynx produce a chronic laryngitis and have been considered on p. 88.

(iii.) **Œdema of the larynx** is a secondary infection and is most commonly met with in cases of faucial cellulitis; it may also occur as a sequel of any variety of ulcerative laryngitis, the ingestion of corrosive substances, the inhalation of steam, as from sucking the spout of a kettle, the sting of a wasp or a bee, the continued administration of potassium iodide, in angio-neurotic œdema, as a complication of the exanthems, and as a terminal event in nephritis.

The symptoms are those of laryngeal obstruction—that is to say, increasing dyspnœa, inspiratory stridor, intercostal recession, cyanosis, and a rising pulse rate. Inspection with or without a laryngoscope will show a red œdematous epiglottis and swollen aryteno-epiglottidean folds.

(iv.) **Spasmodic laryngitis** (*Laryngitis stridulosa*) is a condition peculiar to infants and young children. The glottis in infancy is relatively smaller than in adult life; hence a trivial degree of inflammation is apt to produce a disproportionate amount of distress in the very young, and when there is any constitutional disturbance such as rickets in addition, and sometimes when there is not, a recurrent reflex laryngeal spasm is apt to supervene. The clinical picture presented is that of a mild laryngitis with a hoarse voice, a crowing inspiration, and attacks of spasmodic coughing, to which may be added transient but total apnœa from laryngeal spasm. The spasmodic attacks tend chiefly to occur at night and may persist for days or weeks.

This condition should not be confused with laryngismus stridulus.

(v.) **Laryngismus stridulus**, or child crowing, is simply an expression of the tendency to reflex spasm so commonly present in infancy when the nervous mechanism is upset by rickets, indigestion, teething, or worms. In mild cases there may be merely a periodical bout of crowing inspirations, but in more severe cases suffocation seems imminent owing to sudden and violent adductor spasm of the laryngeal muscles and not infrequently of the other respiratory muscles also. Practically always the spasms will terminate spontaneously, and the air then rushes into the lungs with a characteristic crowing sound. There is no associated laryngitis, and the condition must be regarded as purely functional.

V. BRONCHITIS.

(i.) Acute Bronchitis. This, as the name implies, is an inflammatory process affecting the mucous membrane of the bronchial tubes. The inflammation is caused by one or more of such micro-organisms as micrococcus catarrhalis, streptococcus, staphylococcus, the pneumococcus, the pneumo-bacillus, or the influenza bacillus, except in those cases where the inhalation of some irritating vapour may have caused an œdematous condition of the lining membrane of the bronchial tubes. In these latter cases it is probable that secondary infection with micro-organisms is not long delayed. One attack of bronchitis disposes to another, and the disease is particularly likely to occur in alcoholics and those who suffer from renal disease, mitral disease, or pulmonary tuberculosis. It is an invariable accompaniment of bronchial asthma.

The prognosis in acute bronchitis may be said to vary directly with the size of the bronchial tubes affected; the smaller the tube the more serious the outlook. When the capillary bronchioles are involved the condition practically amounts to one of lobular or broncho-pneumonia, under which heading it will be considered. In the present section acute bronchitis is taken to mean only those cases in which the large and medium bronchi are affected.

Course. The onset is usually gradual, and it is often possible to trace the spread of the infection down from the nose or the larynx through the trachea until the bronchi themselves are involved. Commonly there is a tight or raw feeling across the chest with general malaise, a slight rise of temperature—which usually does not amount to more than 101° in adults, though in children this level may be greatly exceeded—and a dry, painful, unproductive cough. In two or three days a scanty, viscid secretion becomes sufficient to be expectorated in glairy pellets. At this stage an occasional faint streak of blood need cause no alarm, though it is the exception rather than the rule. Gradually the cough becomes looser, the expectoration more abundant and muco-purulent instead of viscid, and in a week or so the attack may be over.

Signs and Symptoms. At the outset there will be no physical signs except a few scattered rhonchi, either sibilant or sonorous, probably to be heard best at the base of the lungs behind. Occasionally these rhonchi are so creaky in character and so localised that they may be confused with a pleuritic rub. As the disease progresses the rhonchi will be accompanied or replaced by râles of various sizes; these may be audible over the entire thorax, but are usually most numerous in the lower lobes.

In a severe attack there may be a certain amount of cyanosis and dyspnœa. These symptoms are important and may mean one of two things:—First, that the right heart is not responding well to the additional strain: this is likely to be the case in a person who suffers from chronic bronchitis with acute exacerbations. In such a case there will probably be evidence of co-existing emphysema and also of right-sided cardiac hypertrophy; possibly also there may be clubbing of the fingers. Secondly, they may mean an involvement of tubes so fine that there is mechanical difficulty in the passage of air into the alveoli, and consequently imperfect aeration of the blood. In these latter cases the râles will be of a fine character, and crepitations will be audible.

The diagnosis of acute bronchitis is not difficult; it should not, however, be mistaken for œdema of the lungs,

in which there are symmetrical areas of impaired resonance in the most dependent portions of the lungs; numerous fine crepitations, both inspiratory and expiratory, over the dull areas; profuse watery expectoration; and, lastly, some evident cause for the œdema, such as a failing heart, enforced recumbency in aged people, and the like.

Unilateral bronchitis and bronchitis limited to the upper lobes should at once excite a suspicion of possible tuberculosis, and steps should be taken to exclude the latter disease. At the same time it must be borne in mind that streptococcal and influenzal infections are often strictly localised and extremely resistant to treatment. In such cases careful bacteriological examination of the sputum is of the utmost value.

(ii.) Chronic Bronchitis. This is more particularly a disease of advanced life, but it is by no means infrequent in childhood. It may result from repeated attacks of acute bronchitis and in children may often be traced to an attack of whooping-cough or measles.

The continued inhalation of irritating material, cardiac and renal disease, and any chronic pulmonary lesion are predisposing factors. It is nearly always at its worst in the winter, and may indeed disappear temporarily each summer.

The diagnosis of chronic bronchitis depends on the history of constant or periodic cough, which in the great majority of instances is accompanied by the fairly free expectoration of muco-purulent secretion. The sputum is often described as white and frothy. This character depends on admixed air and is the result of the emphysema, which is a constant accompaniment of chronic bronchitis.

Râles and rhonchi can be heard in the chest, especially at the bases of the lungs, while the special physical signs of emphysema (*vide* p. 290) are usually present. As a rule there is singularly little constitutional disturbance, and what there is can usually be attributed to the emphysema rather than the bronchitis, or to the underlying factor, such as cardiac or renal disease, which is the cause of the bronchitis. It is of the greatest importance not to overlook cases of

pulmonary tuberculosis when the predominant features are those of chronic bronchitis. To exclude the former repeated examinations of the sputum may be necessary.

The prognosis in the case of chronic bronchitis depends almost entirely on the underlying cause or, if no underlying cause can be established, on the condition of the right ventricle at the time of examination.

Fetid Bronchitis is a name somewhat arbitrarily given to cases in which the sputum is unusually offensive. The majority of such cases will eventually prove to have some super-added factor, such as abscess of the lung, pulmonary gangrene, or bronchiectasis.

(iii.) Fibrinous Bronchitis. This is a rare condition, more common in men than women, and is characterised by the expectoration of a firm white fibrinous cast of the whole or part of one of the bronchial trees. The condition has occasionally been noticed in connection with one of the infectious fevers; more commonly it is of a chronic type, the paroxysms occurring at regular intervals, often throughout many years. Each paroxysm starts rather like an attack of acute bronchitis, only there is more dyspnœa and general distress; the symptoms increase in severity for a varying number of hours until eventually a cast is expelled with an unusually severe attack of coughing.

There is some danger of suffocation during the more severe paroxysms. The condition is never diagnosed until a cast has been brought up, but when once this has occurred mistake is impossible.

VI. EMPHYSEMA.

Emphysema is produced as the result of violent expiratory efforts with a closed glottis or when there is some permanent obstruction to free expiration. Long-continued cough is by far the most common cause. The strain of oft-repeated coughing is most felt by the weakest parts of the lung—namely, the alveoli. The greatest effect is produced in those regions in which the thoracic parietes offer the least support to the lungs—that is to say, at the

apices, along the anterior borders, and where the lungs thin out between the diaphragm and the ribs.

For clinical purposes emphysema may be divided into four classes :—Large-lunged or hypertrophic emphysema, small-lunged or atrophic emphysema, compensatory emphysema, and interstitial or surgical emphysema.

When the term "emphysema" is used clinically it is the first group that is usually meant.

The diagnosis of *hypertrophic emphysema*, when advanced, does not permit of mistake. The patient is usually more or less cyanosed and the accessory muscles of respiration are freely used. The chest is barrel-shaped with a considerable increase in its antero-posterior diameter, and there is usually a slight kyphosis of the upper dorsal vertebræ. Clubbing of the terminal phalanges may often be observed. The whole chest is hyper-resonant to percussion, the superficial cardiac dulness may be diminished or obliterated, and lung resonance may be obtained as low as the eleventh intercostal spaces. On auscultation there is a characteristic harshening and prolongation of expiration, while associated bronchitis is a constant feature. The long-drawn expiration will be best detected at the apices and along the anterior borders of the lungs ; indeed, in mild cases it may be localised to these situations. Owing to the rupture from their points of attachment of the inter-alveolar walls with splaying out of the infundibular terminations and the consequent obliteration of many of the terminal pulmonary arterioles, there is very great obstruction to the pulmonary circulation. The result of this is to throw an increased strain on the right ventricle, and it is failure of the right heart to meet this increased demand that is the chief complication of emphysema. This danger of right-heart failure is greatly enhanced by the liability of these patients to recurrent attacks of acute bronchitis.

Small-lunged or *atrophic emphysema* is of pathological rather than clinical interest. It results from atrophy of the alveolar walls, occurs only in the aged, and is simply a local expression of the diffuse tissue atrophy which occurs in those of advanced age.

Compensatory emphysema is produced in the neighbour-

hood of chronic pulmonary lesions, and is possibly the result of a local attempt of the healthy lung tissue to compensate for the impaired activity in the neighbourhood.

Interstitial emphysema is a surgical affection whereby air is forced into the mediastinal tissues. It is usually produced by trauma, such as fractured rib, tracheotomy, etc., but has been described in cases of whooping-cough.

VII. BRONCHIECTASIS.

The same factors which predispose to the development of emphysema will produce bronchiectasis instead, provided that, as the result of previous inflammation, the bronchial walls have become so softened in certain places as to render these spots the weakest portions of the pulmonary system, for it is always the weakest place which yields to prolonged strain. The old idea that bronchiectatic cavities were produced directly by the pull of contracting bands of fibrous tissue is almost certainly fallacious, and here, just as in emphysema, the essential cause is a series of violent expiratory efforts with a closed glottis.

In order to produce the requisite areas of local bronchial softening more or less damming up of infected secretion is necessary, and this will necessarily be effected by anything which produces a partial occlusion of a bronchus or bronchiole; again, diseases, such as tuberculosis and pneumonokoniosis, which permit of extensive fibrous tissue formation in a given area of lung tissue are likely to produce pouching or pocketing, whereby infected secretion may lie stagnant for a considerable period even though there is no obstruction to the outflow, with the result that bronchiectasis eventually supervenes.

Partial occlusion of a bronchus may be produced from within by the inhalation of a foreign body, by ulceration (simple, tuberculous or syphilitic) or by new growth; or from without by the pressure of a mediastinal tumour or aneurysm.

Clinically bronchiectasis may be divided into acute and chronic.

Acute Bronchiectasis is relatively rare and might perhaps

more correctly be called bronchiolectasis. Its incidence is practically confined to young children, and it is seen as a complication (frequently fatal) of whooping-cough, measles, or capillary bronchitis. In these cases there is a diffuse softening of the bronchiolar walls throughout both lungs; the strain of the severe coughing causes widespread dilatation of these softened areas and an appearance not unlike that of a honeycomb results. The condition is not likely to be diagnosed apart from co-existing capillary bronchitis, but it may possibly be suspected from the unusually profuse and offensive expectoration and the prevalence of large moist râles.

Chronic Bronchiectasis will often depend for diagnosis rather upon the history of the case and character of the expectoration than on any very definite physical signs. There will usually be a history of chronic bronchitis for a varying number of years; this may have developed insidiously, but not infrequently it may be traced to whooping-cough or measles in childhood or to pulmonary tuberculosis, broncho-pneumonia, unresolved lobar-pneumonia, or empyema. Gradually the patient will notice a progressive loss of strength and weight, a general feeling of malaise with tendency to perspirations, a notable increase in the amount of sputum, especially on waking in the morning or on change of posture, and the fact that the sputum has become extremely malodorous. There may or may not be clubbing of the fingers (pulmonary osteoarthropathy).

If the sputum is collected and allowed to stand in a tall glass jar it will be found to settle in three layers: the upper one frothy, the second clear, and the lowest one purulent. Dittrich's plugs can often be identified, and bacteriological examination will demonstrate an extreme multiplicity of organisms, including pneumococci, streptococci, staphylococci, micrococcus catarrhalis, B. proteus, B. coli, etc.

The odour of the sputum and of the patient's breath, though not so appalling as in cases of pulmonary gangrene, is nevertheless sufficiently characteristic to be remembered when once it has been experienced.

The physical signs are very variable; there will always

be evidence of chronic bronchitis, and this may be definitely more severe in one spot, usually towards the base of one lung. Here there may be local dulness, possibly with signs of cavitation or consolidation, and in very marked cases loud, slushy gurgles may be audible. If one lung only is affected, that side of the chest will be flattened and shrunk, with poorer expansion, and of less measurement than the other, and the heart may be pulled over to the affected area.

The differential diagnosis of bronchiectasis presents some interesting features. It must be remembered that tuberculosis is the most common cause of fibroid lung, and that fibroid lung is nearly always accompanied by more or less bronchiectasis. At the same time extensive fibrosis is the method by which tuberculosis becomes healed; hence it is quite possible to have bronchiectasis which was primarily produced by a tuberculous process but in which all active tuberculosis has ceased. Conversely, tuberculosis is often grafted secondarily on to a bronchiectatic lung which has originated from some other cause.

Repeated examinations for tubercle bacilli should be made in all cases.

From a purely clinical point of view it may be said that the restriction of the signs to the bases of the lungs is against tuberculosis, but, since there is often widespread bronchitis at the same time, it may be impossible to say whether the apices are free or not. On the other hand apical bronchiectasis is almost certainly tuberculous. An empyema that has ruptured into the lung may easily be mistaken for bronchiectasis, but the history is likely to be much shorter, and as a rule it is possible to get evidence of the illness from which the empyema dated.

Unresolved or resolving pneumonia may closely simulate bronchiectasis at times, more especially in children who may have lobar pneumonia without the obvious symptoms which occur in adults. The course of the case will prevent mistake for more than a few days.

VIII. FIBROSIS OF THE LUNGS.

A fibrous replacement of more or less of the lung tissues may occur in a variety of conditions as follows :—

(i.) In pulmonary tuberculosis (*vide* p. 97).

(ii.) In bronchiectasis (*vide* p. 291).

(iii.) As a sequel to unresolved lobar pneumonia (*vide* p. 48).

(iv.) As a sequel to broncho-pneumonia.

(v.) As a sequel to chronic dry pleurisy.

(vi.) As a sequel to unrelieved partial collapse of the lung from any cause.

(vii.) In syphilis, either as a sequel to the white pneumonia of congenital syphilis or as the result of gummatous deposits in acquired syphilis. It should be noted that syphilis of the lung is extraordinarily rare.

(viii.) In the form of pneumonokoniosis which develops as a sequel to the constant inhalation of irritant particles in the course of certain occupations, for example :—

(*a*) Anthracosis, in coal miners.

(*b*) Silicosis, in stonemasons and gold miners on the South African reefs.

(*c*) Siderosis, in those who work in iron.

The most common cause of fibrosis of the lungs is tuberculosis, though the incidence of pneumonokoniosis is extremely high in certain occupations ; thus 80 per cent. of the miners engaged in blasting operations on the South African gold reefs acquire " miner's phthisis " within three or four years, though probably not more than 10 per cent. of these are infected with the tubercle bacillus. At the same time any form of pulmonary fibrosis favours the development of tuberculosis.

Symptoms. The symptoms attributable to fibrosis of the lungs, from whatever cause, are cough and dyspnœa, especially dyspnœa on exertion. The expectoration may be foul if there is any bronchiectasis, hæmoptysis is not uncommon, and in cases of anthracosis the sputum is often quite black, while in silicosis bright particles of silica may be detected with the microscope.

Physical Signs. The affected part of the chest is retracted

and moves badly or not at all, the heart is pulled over towards the fibroid area and there is dulness on percussion. Vocal fremitus and resonance are increased locally and bronchial breathing is generally heard. Clubbing of the fingers may develop in old-standing cases.

There are nearly always more or less diffuse bronchitis and compensatory emphysema in the healthy parts of the lungs. Where the fibrosis is of slight extent and widely diffused throughout both lungs, as may happen in the earlier stages of pneumonokoniosis, the physical signs are merely those of chronic bronchitis and emphysema.

Diagnosis. The diagnosis depends on the history and the physical signs: it may be impossible to exclude fibroid phthisis except by the repeated absence of tubercle bacilli from the sputum, though, on the whole, it may be said that signs restricted to the base of a lung are in favour of fibrosis from some cause other than tuberculosis.

IX. MEDIASTINAL TUMOUR, INCLUDING NEW GROWTH OF LUNG.

The symptoms of this condition will depend almost entirely on pressure exerted on surrounding structures and will therefore vary with the precise situation of the tumour. The earliest thing to attract attention is usually cough and dyspnœa together with a sense of oppression in the chest.

The cough may be produced by obstruction or ulceration of a bronchus, or it may be the reflex cough of pleuritic irritation; if the recurrent laryngeal nerve becomes involved a brassy cough like that of aneurysm will result, and laryngeal inspection will show an abductor palsy of the affected vocal cord (usually the left).

Pain may be an early symptom, or it may be absent till the later stages; when present it may be a neuralgic pain from irritation of nerve terminals, a stabbing pain from pleurisy, or the agonising pain which follows erosion of the spinal vertebræ and affection of the posterior nerve roots.

The tumour may come to the surface and be recognised

as a round or irregular swelling which does not possess an expansile pulsation, though pulsation of a sort may be present in certain very vascular sarcomata, or may be transmitted from the heart should this organ be near by.

Pressure on the great veins may cause great congestion of one or both sides of the head and neck, while dilated and tortuous veins on the surface of the chest are frequently developed in order to establish collateral circulation through the inferior vena cava when the superior vena cava has become completely or partially obliterated.

Irritation of, or extension of the growth to, the pleura is likely to cause effusion, and this may well be blood-stained, while microscopical examination of the aspirated fluid may show groups of cells which have the characteristic features of the new growth. If the growth has originated in or extensively involved the lung, communication between a bronchus and the pleura may be established and empyema or pyo-pneumo-thorax will follow. Involvement of the lung or bronchi will also produce bronchitis and bronchiectasis with profuse expectoration, sometimes likened to red-currant jelly from intimate admixture with blood. Severe hæmoptysis is not unknown.

The first sign of mediastinal new growth may be progressive collapse of the whole or part of one lung, as shown by increasing diminution of air entry, followed by increasing dulness and tubular breathing, bronchophony, and the classical signs of consolidation.

The heart may be pushed over to a surprising extent, and the aorta or, more commonly, one of the pulmonary arteries may be compressed until the characteristic murmurs of obstruction are produced.

It will be seen from the foregoing that a great variety of signs may present themselves, but that no single one is conclusive evidence of mediastinal growth ; hence the signs must be reviewed as a whole and considered in conjunction with the history and appearance of the patient.

Valuable assistance may be obtained from the use of X-rays. The possibility of gumma must never be forgotten, and in all cases a Wassermann reaction should be performed.

The differential diagnosis between tumour and aneurysm has been discussed on p. 269.

Mediastinal Lymphadenoma will be recognised by the enlarged glands in the neck, groins or axillæ, and by the big spleen and liver.

In conclusion we may say that a true mediastinal neoplasm is most likely to be lymphosarcoma or endothelioma. A new growth of the lung may be primary, in which case carcinoma is far more common than sarcoma, or secondary, in which case sarcoma is probably more common than carcinoma, though primary carcinoma of the mamma is responsible for a considerable number of secondary growths in the lungs.

X. BRONCHO-PNEUMONIA AND CAPILLARY BRONCHITIS.

(For "Tuberculous Broncho-pneumonia," *vide* p. 90.)

This disease is most frequent at the extremities of life, affecting principally infants and young children or else old and debilitated persons.

Broncho-pneumonia is the immediate cause of death in many cases of injury or disease affecting the skull or brain, because those who are unconscious are peculiarly liable, first to inhale septic particles from the mouth and fauces, and secondly to be unable to remove thoroughly, by coughing, material which has been so inhaled.

For a like reason those who suffer from disease of the pharynx, larynx, or upper air passages are especially liable to contract this complaint.

This variety of broncho-pneumonia is called septic or aspiration pneumonia, but it is important to remember that practically all cases of broncho-pneumonia are produced by aspiration, and that it is not a hæmatogenous infection, as is lobar pneumonia.[1]

Since, then, broncho-pneumonia is produced by the spread

[1] The possibility of the pneumococcus, or some other organism, causing a lobular instead of a lobar infection *viâ* the blood-stream must be admitted, but in our opinion such cases are rare and when they occur produce a condition which clinically resembles lobar pneumonia rather than broncho-pneumonia.

of infection down the bronchi into the bronchioles and ultimately into the alveoli, it would be natural to expect a patchy distribution of inflammatory areas throughout the lungs and also great diversity of causative organisms. Further, in a strict sense, the disease will always be secondary to some infection higher up in the respiratory tract.

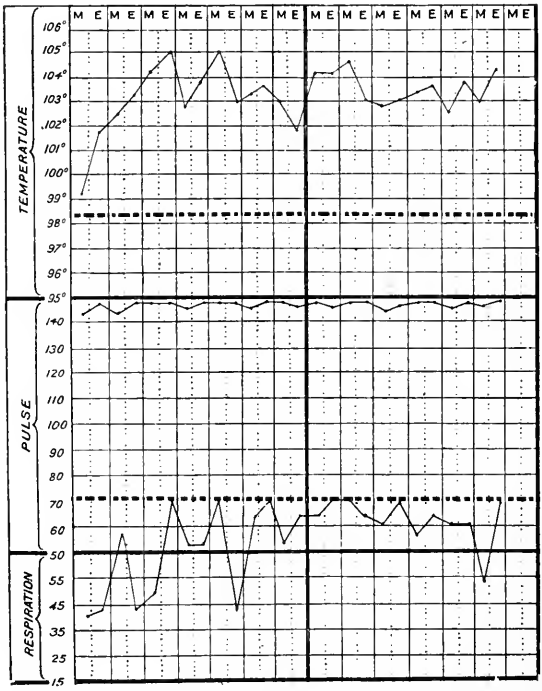


Fig. 52.—Chart from a fatal case of Broncho-Pneumonia in a child of two years. Note the extreme rapidity of both pulse and respirations.

These conclusions are justified by post-mortem and bacteriological findings, as well as by clinical observation. The organisms of broncho-pneumonia include staphylo- and strepto- cocci, Friedlander's bacillus, the bacillus of influenza, the micrococcus catarrhalis, the pneumococcus, and many others. The onset is usually gradual: many cases follow measles and whooping-cough, while often the infection can be traced to an ordinary bronchitis extending to the smaller

tubes until the capillary bronchioles are affected and broncho-pneumonia results.

The clinical picture of broncho-pneumonia varies greatly with the age of the patient ; in the very old the appearances are often masked or overshadowed by some primary disease and the pulmonary condition may easily be overlooked. There is, however, as a rule, increasing dyspnœa and cyanosis, a rapid pulse, an accelerated respiration rate, and a moderate rise in temperature, while a certain amount of cough and expectoration is inevitable.

The onset in infants is usually gradual, extending over days or even weeks of what is apparently an ordinary but fairly severe bronchitis, and then suddenly the child becomes acutely ill. Less commonly there may be obvious broncho-pneumonia within a few hours of the first symptom.

The physical signs may only be those of fine tube basal bronchitis, though there may well be one or more patches of recognisable consolidation. Pulmonary œdema is a frequent accompaniment. It is, however, in infants and young children that broncho-pneumonia is seen in its most characteristic form.

The expression is anxious, the face livid and often cyanosed, the accessory muscles of respiration are at work, the respirations rapid (60 to 80 per minute) and often of the inverted type with an expiratory grunt, and a pause at the end of inspiration. Vomiting and mild convulsions are common, but really severe fits are not usual. The pulse is rapid (120 to 180 per minute) and feeble, and the temperature runs a markedly irregular course, ranging as a rule between 100° and 103° F., though much higher readings are far from uncommon.

On examining the chest the diaphragm will be seen to be working strongly and sucking in the lower ribs with each breath.

Percussion will not often show much abnormality ; there may be relative dulness at one or both bases or between the scapulæ, but more often there are areas of almost hyper-resonance from local emphysema. In well-marked cases there may be definite areas of dulness caused by fusion of many small consolidated areas. Auscultation will show

diffuse râles and rhonchi, with, especially at the bases and in the interscapular regions, localised patches of fine crepitations ("consonating" râles), and possibly here and there an area in which tubular breathing can be heard.

Commonly the physical signs are more marked in one lung than the other; indeed, one lung may be normal throughout the illness save for compensatory over-action and emphysema. Occasionally the whole of one lobe may become solid through fusion of many patches; more often signs suggestive of extensive consolidation are due to collapse of the lung.

The above description is that of a well-marked case, but the diagnosis of broncho-pneumonia is warranted whenever the presence of very fine crepitations in the lungs is combined with cyanosis, dyspnœa, fever, a rapid pulse, very rapid respirations, and an expiratory grunt. The ordinary duration of an uncomplicated case is from two to six weeks, the temperature eventually falling by lysis. As a general rule the more abrupt the onset the shorter will the illness be.

The differential diagnosis does not present any difficulty as a rule.

Acute pericarditis may cause a similar anxious expression with dyspnœa and cough, but there will be no evidence of fine tube bronchitis, and the characteristic pericardial friction will be audible.

Pleural empyema may cause some doubt because it may develop in the course of broncho-pneumonia, or after the acute stage of broncho-pneumonia is over, although considerable bronchitis may still be present.

The absolute dulness, together with diminished tactile and vocal fremitus, the presence of ægophony, and, above all, the displacement of the heart will usually be sufficiently suggestive to warrant an exploratory puncture with a needle. Further, in empyema the condition is not so acute; the dyspnœa is not so urgent as in broncho-pneumonia; a longer history of illness is probable and also a history of repeated sweats. The girth of the chest is increased on the affected side in empyema (unless of very old date, in which case it is diminished), and sometimes there may be bulging of the intercostal spaces.

The differential diagnosis of simple from tuberculous broncho-pneumonia may be impossible clinically: the tuberculous variety is often more insidious in onset, and, for a time at least, the child is not so *acutely* ill as the rapid extension of pulmonary signs might appear to warrant, and it is very unusual for any of the signs to clear up, even temporarily.

The sputum should be examined for tubercle bacilli, and if the patient is too young to spit he may be made to vomit; fragments of the swallowed sputum may then be picked out of the stomach contents for microscopical investigation.

XI. ABSCESS OF THE LUNG.

Two varieties of pulmonary abscess may be met with:—First, the pyæmic abscess, usually small and multiple, depending on the presence of an infective focus in the right heart or in a systemic vein; and secondly, a single lung abscess which may occur in the course of bronchiectasis, empyema, unresolved pneumonia, broncho-pneumonia, or tuberculosis in which secondary infection has taken place; or by the spread of infection from below the diaphragm, as in the case of subphrenic abscess or liver abscess. The embolic forms of lung abscess are especially liable to be accompanied by empyema.

The diagnosis of Miliary Pyæmic Lung Abscesses is usually impossible, though they may be suspected whenever a septicæmia results from right heart endocarditis or from septic phlebitis which is not restricted to the portal system. Occasionally a septic embolus may be of sufficient size to produce a lung abscess big enough to cause physical signs. The first signs in such a case will be those of infarction—viz., sudden dyspnœa, pain in the chest, pleurisy and cough. Hæmoptysis may follow and later on the signs of lung abscess as mentioned below.

The diagnosis of Single Pulmonary Abscess will depend on the combination of symptoms of septic poisoning with more or less indefinite localising signs in one or other lung. If the abscess communicates with a bronchus the expectoration of quantities of pus will help the diagnosis; otherwise the signs

are likely to suggest a localised empyema or even an area of consolidation. Exploratory puncture may reveal the true state of affairs, or X-rays may enable us to clear up the diagnosis.

XII. GANGRENE OF THE LUNG.

This condition is produced in the same manner as is abscess of the lung. It may be added that it is particularly likely to be found in cases of diabetes and other wasting disorders of metabolism.

The physical signs are the same as for abscess, but the peculiar odour of the sputum usually leaves no room for mistaken diagnosis.

In both gangrene and open abscess, examination of the sputum will show shreds of elastic tissue produced by disintegration of the lung substance (*vide* p. 93).

XIII. ŒDEMA OF THE LUNGS.

This may occur as part of a general dropsy, whether cardiac or renal, in debilitated elderly people from static causes should a supine position be enforced, or in certain cases of mediastinal tumour in which the pulmonary circulation may be especially embarrassed. In all these cases the condition is of gradual development.

Pulmonary œdema may, in rare cases, develop quite acutely and be the cause of sudden death; the pathology of the phenomenon is not understood beyond that it must be caused by some reflex vaso-motor disturbance. It is more frequent in women than men and has more than once been recorded during coitus.

The diagnosis of pulmonary œdema will rest on the general condition, the presence of profuse watery sputum, and symmetrical areas of relative dulness at the bases of the lungs over which a poor air entry is combined with numerous fine, crackling râles.

XIV. BRONCHIAL ASTHMA.

This affection often starts in childhood, but no age is immune; it is always accompanied by more or less chronic

bronchitis and emphysema, and is characterised by paroxysms of urgent expiratory dyspnœa in which the sufferer leans forward with a livid, clammy face and with his chest fixed in a position of forced inspiration. After a time, varying from minutes to two or three hours, the attack terminates in a fit of coughing, during which a small amount of mucus, often in the form of pellets, is generally coughed up. Each attack is usually of sudden onset, and often a definite aura is described, generally of a psychic nature.

There are no characteristic physical signs, but bronchitis and emphysema are invariable. The sputum generally contains the colourless octohedral crystals of spermin phosphate (Charcot-Leyden), Curschmann's spirals which are mucinous casts of the bronchioles, and a number of eosinophil cells. In addition, during the attack there is a definite eosinophilia in the blood.

The attacks of asthma are most frequent at night; they may occur apart from any discoverable exciting cause, or they may be induced by a great variety of apparently inconsequent factors, such as a heavy meal, the wearing of flannel, sleeping in a feather bed, various smells, etc., etc.

Certain organic causes should always be investigated, such as the presence of enlarged tonsils or adenoids, hypertrophied turbinal bones or other forms of nasal obstruction, and even errors of refraction.

As a general rule asthmatic patients have fewer attacks in towns than in the country, a fact which can be explained by the irritating effect of the dust of various plants in the country.

Asthma is regarded as a neurosis; the immediate cause is probably hyperæmia of the bronchial mucosa and spasm of the bronchioles themselves. These are produced reflexly by some such cause as those just discussed.

There is no doubt of the diagnosis if an attack can be observed, and in other cases the history is sufficient.

Certain other forms of asthma are described and must not be confounded with true bronchial asthma.

Thymic Asthma. A condition which is liable to cause sudden death in infants and young children. It is produced by the presence of an enlarged or unduly persistent thymus

gland which may cause dyspnœa from pressure on the trachea, on the great vessels, or on the vago-sympathetic nervous system. There is a further possibility that the abnormal persistence of a functional thymus gland after ten or twelve years (when it should commence to atrophy) may permit the circulation of certain toxins which may be deleterious to the health of the patient. The condition is but rarely suspected during life.

Cardiac Asthma. Paroxysmal dyspnœa may be a notable symptom in patients suffering from advanced myocardial degeneration or heart failure secondary to valvular disease. The individual attacks may closely resemble true bronchial asthma, but examination of the heart should prevent mistake.

Renal Asthma. Paroxysmal dyspnœa is often seen in patients with severe renal disease; in such cases it may be an uræmic phenomenon or it may indicate a failing heart. Examination of the urine will prevent such a case being mistaken for bronchial asthma.

XV. PLEURISY.

Pleurisy may be primary or secondary, dry or with effusion, and the effusion may be simple or purulent.

Primary pleurisies may be produced by tuberculosis, rheumatism or new growth. It is important to remember that the great majority of all cases are tuberculous.

Secondary pleurisies may result from any pulmonary affection that extends to the surface of the lung and so involves the pleura, or they may occur in the course of a septicæmia and in certain constitutional metabolic disorders such as Bright's disease. They may be secondary to some infection below the diaphragm, or they may occur in the course of such specific infections as enteric or scarlet fever. Again, tuberculosis is probably the most common cause, though pleuro-pneumonia is responsible for many cases.

(i.) Dry Pleurisy.—**The symptoms** of dry pleurisy are a sudden sharp, stabbing pain in the chest (which is greatly aggravated by drawing a deep breath or by coughing) a dry, hacking, unproductive cough, and a sense of general malaise.

There is usually a moderate pyrexia which shows a distinct morning remission.

Examination of the thorax will show a relative immobility of one side of the chest, caused by the instinctive desire to avoid the pain of movement.

Auscultation will show the characteristic to-and-fro rub over the site of the pleurisy ; this rub may be coarse or fine—it sounds rather like two pieces of damp leather being rubbed together when coarse, and like hair being rubbed between the fingers when of the fine variety. It is audible during both inspiration and expiration and it does not disappear or alter after coughing. The deeper the respiration the more audible the rub.

Pleuritic friction may be heard over any part of the chest ; it is most frequently detected in the posterior part of the axilla.

It is important to remember that the pain of pleurisy is often referred along the course of the intercostal nerves even to the abdomen, and is not always situated at the site of the inflamed portion of the pleura.

Diagnosis. When there is a well-marked rub the diagnosis of dry pleurisy is simple enough, but the rub is often evanescent, and the case may not be seen till the rub has gone and the pain also. Under such circumstances the description of the pain may be all there is to go upon. If the patient is known to be suffering from some pulmonary disease, such as tuberculosis, in which pleurisy is common, an additional probability is present. In some cases the friction disappears quickly because the two layers of pleura become separated by a thin layer of fluid ; as this fluid is absorbed the rub may re-appear.

Dry pleurisy must be differentiated from pleurodynia or intercostal myalgia, in which, of course, there is never any rub or true impairment of air entry, while the pain tends to be more boring or aching than sharp and stabbing, and there is no constitutional disturbance. Both these conditions are associated with local tenderness to a greater extent and more commonly than pleurisy.

Intra-pulmonary sounds (fine râles) or creaky rhonchi may be mistaken for pleuritic friction, especially at the fringes

of the lungs ; they can, however, nearly always be greatly modified by coughing and deep breathing, while they are often confined to the inspiratory part of the respiratory cycle.

Pericarditis may be mistaken for pleuro-pericarditis or inflammation of that part of the pleura which lies on the pericardium. The differences have been considered on p. 260.

The pain of pleurisy, especially of diaphragmatic pleurisy, is often referred to the abdomen, and an acute abdominal condition may be simulated. A careful examination will nearly always show some abnormality of the thoracic action or physical signs which will suggest that the trouble is above the diaphragm, while there is no *true* immobility, tenderness, or rigidity of the abdominal wall.

(ii.) Simple Pleural Effusion. **The symptoms** of this condition depend rather on the rapidity with which the effusion has collected. The more rapid the effusion the more marked will be the breathlessness and general discomfort, while if the fluid has been poured out very slowly, a large amount may be present without marked dyspnœa. The characteristic pain of the dry stage of the pleurisy disappears with the onset of effusion. There need be no pyrexia, but there is usually a slightly accelerated respiration rate.

The Classical Signs of free fluid in the pleura are :—

(1) Impaired mobility of the affected side.

(2) Displacement of the heart to the sound side.

(3) Absolute dulness to percussion below a certain level.

(4) A skodaic or "boxy" percussion note above the level of the fluid (this is usually absent in large effusions).

(5) Cutting off or great diminution in breath sounds, voice sounds, and tactile fremitus over the area of dulness.

(6) A band of ægophony at the upper level of the fluid.

(7) The upper level of the dull area is not horizontal, but forms a definite curve (Damoiseau's curve) as is shown in Fig. 51.

(8) An increased measurement on the side of the effusion.

In addition to the above the following may be noted in certain cases :—

(1) Bulging of the intercostal spaces on the affected side when there is much fluid.

(2) Immobility of the diaphragm and opacity of the lower thorax, as shown by X-rays.

(3) Downward displacement of the liver or spleen, according to which side is affected.

(4) A small area of tubular breathing about the upper level of the fluid behind, depending on collapsed lung.

(5) Grocco's triangle. This is a triangle of dulness *on the sound side* formed by joining that vertebral spine which is opposite the upper limit of the fluid with a point 8 cm. from the middle line along the twelfth rib. The remaining sides of the triangle are the twelfth rib and the mid-line behind.

(6) Dilatation of the pupil on the affected side from irritation of the sympathetic nerve by distension of the pleura (this is a rare sign and is more commonly met with in empyema).

It is well to remember that fluid in the pleura does not cause a positive pressure until it is as high as the third rib, and that, in an average man, this is equivalent to the presence of about three pints of fluid.

Change in posture may cause a trifling alteration in the shape and position of the dull area, especially when the effusion is not large.

Difficulty in diagnosis may occur when the effusion is small and, as sometimes happens, spread out into a thin layer. In all cases the most important signs are :—

(1) The displacement of the heart.

(2) The peculiar dead, woolly percussion note.

(3) Diminution in breath sounds, voice sounds, and fremitus.

(4) Ægophony.

In rare cases a non-purulent effusion may be loculated ; the physical signs may then be rather anomalous—for example, the dulness may be strictly localised and not at the extreme base of the lung, while there will often be much more diffuse tubular breathing than is ordinarily heard.

Much more commonly, however, a loculated effusion will be found to be purulent (*vide* next section).

The differential diagnosis of pleural effusion from solid lung and from pneumo-thorax are considered on pp. 52 and 313 respectively.

Collapsed lung may simulate an effusion, but the heart, if displaced at all, is moved towards the affected side ; the intercostal spaces tend to show inspiratory recession, the percussion note is not so flat as in effusion, while the auscultatory signs may vary from complete absence of breath sounds to loud tubular breathing, and adventitious sounds (râles and crepitations) are often heard so long as any aeration of lung persists.

Further, some cause for possible collapse may be evident, such as mediastinal tumour, deformity of the chest, an extremely debilitated condition, rickets, broncho-pneumonia, or bronchial obstruction.

Hydrothorax gives the same physical signs as does pleural effusion ; it is commonly first seen on the right side, a fact which may be explained by the anatomy of the vena azygos major, and tends to become bilateral.

Hydrothorax may be anticipated in cases of right heart insufficiency, where there is any mechanical obstruction to the venous return within the thorax, as in new growth, lymphadenoma, etc., and in chronic parenchymatous nephritis as a part of the general œdema.

(iii.) Empyema. In addition to the general signs of pleural effusion empyema may be expected to present some of the following features :—

(1) Toxic appearance, with irregular temperature, rapid pulse and respirations, and tendency to free perspirations.

(2) A definite leucocytosis, usually about 18,000 per cubic millimetre.

(3) A history of some antecedent illness, such as pneumonia or broncho-pneumonia.

(4) A more extensive area of tubular breathing than is usual in simple effusion.

(5) The absence of a typical Damoiseau's curve, owing to the tendency for the formation of adhesions.

(6) Possibly some œdema of chest wall on the affected side.

The possibility of an empyema being loculated should always be remembered, and to this end careful examination must be made along the interlobar septa whenever empyema is suspected, for it not infrequently happens that an empyema is formed between the lobes of the lung. The adjacent lung substance is readily compressed, but the pus itself may remain at some distance from the chest wall. A patch of dulness and a variable amount of tubular breathing may be the only physical signs, but the fact that these are found along the course of the interlobar septum is suggestive of empyema when the corroborative symptoms of septic absorption are present.

Paracentesis Thoracis. In the majority of cases it is possible to be sure of the presence or absence of fluid in a pleural cavity by a careful consideration of the signs and symptoms; occasionally, however, the signs may be so anomalous that the diagnosis can only be settled by exploration with a needle. This manœuvre is practically free from risk, and should certainly be adopted when there is any possibility of the effusion being purulent.

The best instrument is a stout hollow needle attached to a syringe of at least 20 c.c. capacity; or, if preferred, a Potain's aspirator may be employed.

For diagnostic purposes the site of election is the point of maximum dulness, always provided that the situation of the heart, liver, spleen, and stomach are borne in mind.

As a general rule, when the fluid is free, exploration is best performed in the seventh or eight spaces in or just behind the line of the scapular angle; some authorities advise the sixth space in the mid-axillary line.

Where pus is suspected and the signs suggest that it is loculated, one must be guided entirely by the situation of the dulness and the tubular breathing, if any be present.

The best method of performing paracentesis is to have the patient sitting up, or, if he is too ill to sit up, lying on the sound side with his arm raised and his hand on the opposite shoulder. The left thumb-nail is then placed on the upper edge of the rib next below the point where exploration is indicated

and the needle, attached to the syringe and guarded about one inch from its point by the right forefinger, thrust swiftly along the thumb-nail and through the intercostal space into the thorax until stopped by the right forefinger. The needle is now thrust slowly forwards—as nearly as possible at right angles to the thoracic parietes—at the same time as the piston is slowly withdrawn to create a negative pressure in the barrel of the syringe.

As soon as pus or fluid is reached it will appear in the syringe.

The first syringe-full should be placed in a sterile vessel for subsequent examination.

It is essential that the operation of paracentesis should be performed with all antiseptic precautions.

Examination of the fluid withdrawn should be directed to the following points :—

(1) The presence of organisms, and, if present, their nature and whether they are alive.

In the great majority of cases the presence of bacteria is an indication for prompt resection of rib with free drainage, but occasionally it may happen that only dead pneumococci are found ; under these circumstances aspiration of the fluid may be all that is necessary, though in our opinion it is better for such cases to be drained.

(2) The presence of cells and their nature.

In empyema the great majority of the cells are pus cells or polymorphonuclear leucocytes.

In simple pleurisy with effusion (tuberculous) the majority of the cells are lymphocytes, though a certain number of uni-nuclear endothelial cells are usually present.

In passive exudates the number of endothelial cells is increased and many of them are likely to be disintegrating and showing fatty degeneration.

In new growth fragments of growth may be seen. Blood-cells are commonly present and large multi-nucleate endothelial cells are not infrequent, in varying stages of degeneration, if the examination is made immediately. Rivalta's test is a simple method of estimating whether an effusion is passive or inflammatory : about half a drachm of effusion is thrown into a good-sized beaker containing very dilute

acetic acid; a white cloud is formed by inflammatory effusion, but not by passive exudates.

It is a safe rule that all primary lymphocytic effusions are tuberculous in origin, though a very small proportion may be due to other causes, such as rheumatism, gonorrhœa, etc.

Unfortunately the proof of this hypothesis is not easy, for the reason that tubercle bacilli are but rarely demonstrable in the aspirated fluid. When they are present it is a curious fact that a large proportion of the cells present are frequently polymorphonuclear leucocytes.

A better method of proving the nature of a given pleural effusion is to inject a considerable quantity of the fluid into the peritoneum of a healthy rabbit. If the rabbit develops glandular and splenic tuberculosis in eight weeks, the effusion was tuberculous.

Another proceeding of value is to compare the opsonic indices for tuberculosis of the patient's blood serum, known healthy blood serum, and the effusion. It is suggestive of tuberculosis if the reading of the effusion varies 40 per cent. from that of the patient's blood serum, while a wide difference between the two blood serums is also suspicious. A tuberculous effusion commonly gives a much lower reading than the patient's blood serum.

XVI. PNEUMOTHORAX.

The causes of pneumothorax are as follows :—

(i.) Pulmonary tuberculosis.

(ii.) Ulceration of an empyema into the lung.

(iii.) Rupture of a lung abscess into the pleura.

(iv.) Rupture of an emphysematous bulla.

(v.) Trauma of the chest, such as a fractured rib driven into the lung, stab wounds, or exploratory paracentesis.

(vi.) Gastro- or intestino- pleural fistula.

(vii.) Whooping-cough.

The vast majority of all cases are tuberculous.

The pneumothorax may be produced suddenly or the air may leak in gradually; in some cases there is valvular communication into the pleural cavity, which may get more and more distended with each inspiration until it is as tight as a drum.

In all open cases the air in the pleura is at the atmospheric pressure, but in closed cases the pressure varies greatly, usually being about 7 mm. of Hg. (Osler). This is of course amply sufficient to cause complete collapse of the lung on that side, as the normal intra-pleural pressure is not more than − 3 mm. In course of time the passage into the pleura becomes sealed and the air in the pleura is absorbed, thus permitting re-expansion of the lung. Before this happens, however, there is practically always a pleural effusion from irritation of the pleural endothelium, and often this effusion becomes purulent from infection *viâ* the communication with the lung. Thus pneumothorax usually develops into hydro- or pyo- pneumothorax in a comparatively short time.

Symptoms. At the moment of rupture the patient often complains that something has "given way" in the chest; there is severe pain in the side, cyanosis, dyspnœa, and shock. A person who is cachectic and debilitated may well succumb to the immediate effects of pneumothorax, but more commonly the shock and pain pass off in a few hours and only a varying amount of dyspnœa remains.

It may very exceptionally happen that a pneumothorax may develop so insidiously that all urgent symptoms are obviated.

Physical Signs. The affected side of the chest is immobile and often the intercostal spaces bulge.

The heart is displaced to the sound side.

The percussion note is usually tympanitic; if the air is under great tension the note is so high-pitched as to be dull, but it is never as flat as when fluid is present.

Palpation shows complete absence of tactile fremitus.

Auscultation shows great enfeeblement or even absence of voice and breath sounds, and a characteristic metallic tinkle on coughing or speaking can usually be detected.

This metallic tinkle can often be beautifully demonstrated by using one coin as plessor and another as pleximeter and simultaneously listening with a stethoscope in their vicinity. The noise of metallic contact has a strikingly clear and bell-like note, and is known as the coin sound.

When fluid is present (hydro- or pyo- pneumothorax)

there will be a dull note at the base of the lung over an area the upper limit of which is readily altered by change of posture, and a splash will be heard if the patient sits up and his chest is shaken. This splash (succussion) sound may be audible at some distance, but should be listened for with a stethoscope.

Amphoric breathing may be heard in cases of open pneumothorax. As the fluid increases the air grows less and eventually only a bubble remains, and, since the patient is usually kept lying down, this bubble floats up to the front of the chest and can often be mapped out as a resonant area between the nipple and the clavicle. Gradually this resonant area decreases in size until all the air is absorbed.

Diagnosis. From the foregoing physical signs it is evident that the diagnosis of a large pneumothorax does not present much difficulty. A greatly pushed-over heart combined with tympanitic percussion note and a metallic tinkle must mean pneumothorax.

A very large superficial cavity communicating with an open bronchus might suggest pneumothorax, but the heart would, if moved at all, be drawn towards the affected side.

A diaphragmatic hernia may present difficulties of diagnosis, as, for example, when the stomach is situated mostly in the thorax owing to congenital deficiency of part of the diaphragm. It will be noted in such a case that the ingestion of food or water effects a profound alteration in the percussion note, and that borborygmi are audible independently of movement. An X-ray examination after a bismuth meal would be conclusive.

Simple pleural effusion may be simulated in those cases where the tension is so high that a dull note is heard on percussion. The metallic tinkle and the relatively great displacement of the heart will suggest the true diagnosis.

CHAPTER III

DISEASES OF THE MOUTH, FAUCES, ŒSOPHAGUS AND ALIMENTARY TRACT

I. THE TONGUE AND MOUTH.

(*a*) **The Tongue.** The tongue is an index of the well-being of the stomach and intestines, and the gastric functions are disturbed reflexly in all febrile disorders and in many other conditions of ill-health.

Normally the tongue should be pink, clean, and moist.

In *chronic gastritis* the tongue is pale, flabby, covered with fur, and indented by the teeth.

In *gastric ulcer* unassociated with gastritis the tongue is clean.

In *chronic constipation* the tongue is thickly coated and the fur is sometimes yellowish in colour.

In *thrush* the tongue may be covered by a white mould (oidium albicans).

In *fevers* the tongue is dry and coated. The strawberry tongue of *scarlet fever* has been described on p. 62.

In *enteric fever* the tongue is moist in the early days, with a slab of white fur on each side of the mid-line and a bright red line at each lateral margin and often down the centre ; in the later stages, as in any prolonged fever with toxæmia and exhaustion, the tongue is dry and cracked.

In *diabetes* the tongue is large and beefy in appearance,

In *chronic alcoholism* and in *general paralysis of the insane* the tongue is finely tremulous.

In *disseminated sclerosis* and in *chorea* there is a coarse tremor on protrusion of the tongue. In the latter disorder the tongue, instead of being withdrawn in the usual manner, is suddenly snapped back into the mouth.

In *cretins* the tongue is too big for the mouth, while in

mongolian idiots it shows deep longitudinal fissures on its surface.

In most cases of *hemiplegia* the tongue is protruded towards the side of the paralysed limbs : it may be coated on one side only.

In *bulbar paralysis* the tongue is wasted, shrivelled, and cannot be protruded.

In *facial paralysis* there is loss of taste for the anterior two-thirds of the corresponding half of the tongue provided that the lesion of the nerve is situated at that part of its course wherein it is accompanied by the chorda tympani nerve, that is to say between the geniculate ganglion and a point about $\frac{1}{4}$ inch above the stylo-mastoid foramen.

Epilepsy may be suggested by scarring of the tongue.

Naso-pharyngeal disease, *adenoids*, *mouth breathing*, *dental caries*, and a *milk diet* all tend to cause a coated tongue.

Syphilis may be shown either by gummatous ulceration, fissured scars, or the dense, white, smooth, raised, non-ulcerated though sometimes fissured patches of leucoplakia. Similar patches may, however, be caused by smoking, spices, or alcohol.

Addison's disease may cause brown pigmentation of the tongue.

Epithelioma must not be mistaken for gumma. A malignant ulcer usually has a horny edge ; infiltration causes early fixation of the tongue, and glandular involvement is common in the sub-maxillary triangles.

(*b*) **The Gums and Cheeks.** Pyorrhœa alveolaris causes a line of hyperæmia at the gum margin, while pressure will cause pus to exude from between the gum and the tooth. Slight cases can only be detected by most laborious examination of each tooth socket with a fine probe. The importance of the part played by pyorrhœa in the production of rheumatoid arthritis, pernicious anæmia, and obscure toxæmia is still debated, but it is certain that it *can* produce such or similar conditions, and even septicæmia has been initiated by this disease.

Sloughing gums with gangrenous tonsillitis are highly suggestive of acute lymphatic leukæmia or aplastic anæmia

when they occur in young people. In older persons pernicious anæmia is more probable. In the absence of tonsillitis scurvy must be considered, as well as purpura hæmorrhagica and chronic mercurial poisoning.

Gangrenous stomatitis may also occur in children after the acute exanthems (especially measles), and is likely to be accompanied or followed by *noma oris, i.e.*, a patch of gangrene starting on the buccal aspect of the cheek, which soon involves its entire thickness, spreads steadily, and is practically always attended by a fatal issue.

Ulcerative stomatitis occurs in two forms. In the mild or *follicular* form small crops of vesicles occur on the lips, cheeks, and tongue ; these break and small circular ulcers are left. Infants and young children are the principal sufferers from this condition. In the more severe form (*fetid stomatitis*) young and ill-cared-for children are again chiefly affected ; the ulcers are larger and tend to spread ; both gums and cheeks are involved, and, for a few days, the constitutional symptoms are severe.

II. THE TONSILS.

A. **Acute Follicular Tonsillitis.** The onset of symptoms is abrupt. There is shivering or even a rigor, with a sudden rise of temperature to from 102° to 104° N. The pulse is full and bounding, but not extremely rapid. The face is flushed, the eyes are bright, but there is no circum-oral pallor. Sometimes a patchy erythema may appear about the limbs or trunk ; this has no characteristic features and tends to fade rapidly. There is dysphagia with a sense of soreness or stiffness down the neck, and the submaxillary glands become enlarged. Inspection of the throat will show that one or both tonsils are enlarged and inflamed, while yellowish-white plugs can be seen projecting from the tonsillar crypts. Sometimes the secretion from the crypts runs together in the form of a localised membrane, which, however, can readily be removed with a swab.

This condition must not be mistaken for diphtheria (*vide* p. 14), scarlet fever (*vide* p. 65), or for Vincent's angina (*vide* p. 14). In all cases a swab should be taken for bac-

teriological examination. A mixed growth of strepto- and staphylo- cocci will generally be obtained from a case of follicular tonsillitis.

B. **Acute Parenchymatous Tonsillitis.** The symptoms are identical with those of follicular tonsillitis, but this form is more likely to be associated with acute inflammation of the faucial pillars and pharynx; it is usually streptococcal in origin and closely resembles faucial erysipelas.

The tonsils are enlarged and dusky red in colour, and often they have a rather glazed appearance. There is little or no follicular exudate.

The constitutional symptoms are similar to those of follicular inflammation, but the disease is more severe and has a longer course. The differential diagnosis is the same.

It must be remembered that this form of tonsillitis, even more than the follicular form, is likely to be a manifestation of rheumatism.

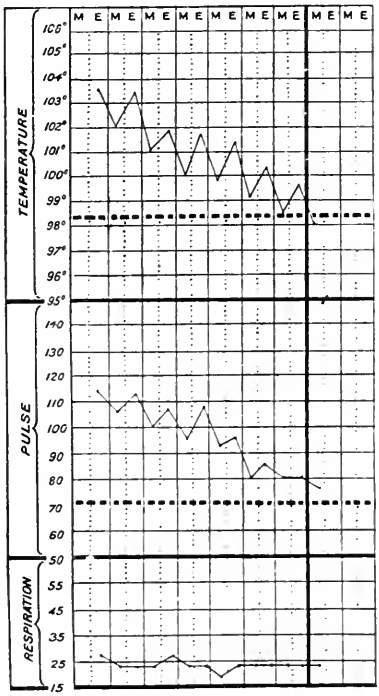


FIG. 53.—Chart from a case of Follicular Tonsillitis. Note the relative slowness of the pulse as compared with Diphtheria or Scarlet Fever.

C. **Acute Peritonsillar Inflammation (Quinsy).** This condition terminates in the formation of an abscess behind one or other tonsil. The onset is not abrupt, though the symptoms become severe in from twelve to thirty-six hours. At the outset there is increasing pain and soreness down one side of the throat; soon dysphagia becomes acute, the temperature rises to 103° to 104°, and a lancinating pain suggests that pus formation is in progress.

Examination shows an inflamed tonsil, which is pushed forwards and towards the middle line by a red, brawny, sometimes œdematous swelling of the peritonsillar tissues.

The faucial aperture may be well-nigh obliterated, and dyspnœa may be alarming. Eventually fluctuation will appear.

After several days, if incision be withheld, the abscess bursts into the mouth, and recovery is thenceforward rapid, though occasionally the cavity fills up again with pus owing to imperfect drainage. Sometimes, and especially if the quinsy bursts during sleep, septic pneumonia or even sudden death from asphyxia may result.

The diagnosis of quinsy presents no difficulty; it must not be confounded with retropharyngeal glandular abscess, which causes a bulge on the posterior pharyngeal wall and is practically limited to small children, or with retropharyngeal abscess due to caries of the cervical spine, which may point to one or other side of the mid-line and which can usually be diagnosed by the pain caused on manipulating of the cervical vertebræ.

D. **Gangrenous tonsillitis** may occur as part of a septicæmic infection, in acute lymphatic leukæmia, in purpura hæmorrhagica, in aplastic anæmia, or in the late stages of ordinary pernicious anæmia.

III. THE PHARYNX.

A. **Acute pharyngitis** is often secondary to a faucial or nasal infection, as in rheumatism, scarlet fever or diphtheria, but it may occur primarily as an acute catarrhal affection or from direct irritation, such as the inhalation of noxious vapours. The constitutional disturbance is but slight; there is dysphagia and a raw or sore feeling in the naso-pharynx.

Inspection shows an inflamed mucosa, which is sometimes coated with a glairy muco-purulent discharge.

It should be remembered that *recurrent* acute pharyngitis is probably due to nasal or retro-nasal trouble, such as adenoid vegetations, hypertrophied turbinal bones, chronic rhinitis, or sinusitis.

B. **Chronic pharyngitis** is usually the result of too much speaking without the necessary knowledge of how to use the voice, too much alcohol, or too much tobacco; it is also likely to accompany chronic nasal catarrh or chronic affections of the nasal sinuses.

The most prominent symptom is a constant "hawking cough" with a perpetual desire to "clear the throat."

Chronic laryngitis is a fairly common accompaniment, and in these circumstances the voice may be husky or even lost. Inspection shows that the pharyngeal mucosa is injected: prominent venules may sometimes be observed on it, as well as raised red patches of hypertrophied lymph tissue.

In other cases the chronic irritation causes atrophy of the secreting structures and a smooth, dry surface results.

C. **Retropharyngeal Abscess.** (i.) *Originating in lymph nodules situated in front of the prevertebral fascia.* Presumably this infection is conveyed through the posterior pharyngeal wall. These abscesses are not tuberculous, and, except in traumatic cases, are chiefly met with in infancy. The symptoms are fever, dysphagia, and dyspnœa, for which no cause can be found in the lungs or heart. Examination of the throat may show a rounded swelling bulging from the back wall of the pharynx; sometimes this may be hidden by the soft palate, but it can always be detected by the *examining finger*. The abscess nearly always points at or about the mid-line.

(ii.) *Originating in tuberculous caries of the upper cervical vertebræ.* These abscesses commence behind the prevertebral fascia and are seen in older children than the first variety. The bulging which they produce is lateral rather than central. The symptoms are chronic rather than acute.

The tenderness of the cervical spine with fixation of the head and hectic temperature should suggest the diagnosis.

It is important not to mistake for each other these two varieties of retropharyngeal abscess, since the treatment is different. The tuberculous abscess is opened externally behind the sterno-mastoid, while the other form is opened into the pharynx.

IV. THE ŒSOPHAGUS.

A. **Œsophageal Pouches.** These may be congenital or acquired; they are usually situated at the upper end of the œsophagus about the level of the cricoid cartilage or else at the level of the tracheal bifurcation.

They may give rise to no symptoms, but food may be caught up in them, giving rise to choking sensations, dysphagia, and retching.

They cannot readily be diagnosed except with the X-rays after a bismuth meal, or by the passage of an œsophageal bougie.

B. **Œsophageal Varix.** A sudden profuse hæmatemesis in a cirrhotic individual who has no history suggesting gastric ulcer may lead to a correct diagnosis, especially if the blood is bright red. It must be remembered that rupture of an œsophageal pile is often followed by collapse and death without hæmatemesis; in such cases a complete blood-cast of the stomach is found post mortem.

Unless an œsophageal varix leaks, there are no signs or symptoms to suggest its presence.

C. **Œsophageal Obstruction.** This may be produced by stenosis after ulceration, which may be simple (as from the swallowing of some corrosive), syphilitic, or malignant. The gullet may also be obstructed by pressure from without as by an aneurysm or a mediastinal tumour, or there may be impaction of some foreign body, such as a halfpenny or a collar-stud, in the lumen of the tube. Such foreign bodies are usually impacted (*a*) at the level of the cricoid, (*b*) opposite the tracheal bifurcation, or (*c*) at the cardiac opening of the stomach.

The chief symptom of œsophageal obstruction is progressive difficulty in swallowing solids. Fluids are generally taken without discomfort till a considerably later date. The usual description given is that the food seems to stick at a certain point (most often referred to the episternal notch). Violent retching is immediately initiated and the food is returned, often accompanied by a white frothy fluid. Wasting is always rapid, more so in malignant cases, in which cachexia is added to simple starvation.

In making a **differential diagnosis** the following points should be borne in mind :—

(i.) After the age of 30 years 80 per cent. of all cases of œsophageal obstruction are malignant. The presence of enlarged glands above the clavicles indicates a new growth with certainty.

(ii.) In *children* the commonest causes are impaction of a foreign body, swallowing a corrosive, or diphtheritic œsophagitis. Inquiry will generally elicit the fact that something may have been swallowed (this should be confirmed by the X-rays). Inspection of the fauces will show burning from corrosive, and probably membranous inflammation in diphtheritic cases.

(iii.) In *young women* hysterical spasm may closely simulate organic obstruction. Other signs of hysteria may nearly always be found, such as hemi-anæsthesia, monoplegia, aphonia, etc.

Anorexia nervosa is usually seen in young women, and immediate vomiting or regurgitation of all solid food is a fairly constant symptom of this condition. There is, however, no true obstruction and the food passes without delay into the stomach. This can be demonstrated readily by means of the X-rays and food impregnated with bismuth (*vide* also p. 588).

(iv.) *Aneurysm and mediastinal tumour* commonly give their own characteristic signs (*vide* p. 269). Further, pressure from these causes is but rarely sufficient to cause such definite obstruction as does organic stenosis ; indeed, an aortic aneurysm not infrequently causes death by ulcerating into the œsophagus without ever causing clinical obstruction.

(v.) *Syphilis* must always be remembered, though it is a very uncommon cause of œsophageal obstruction. If the Wassermann reaction is positive the exhibition of antisyphilitic treatment is justified. In any case a course of potassium iodide may cause temporary improvement.

(vi.) The passage of *œsophageal bougies* will at once show the presence of œsophageal obstruction. It is, however, not justifiable to use these instruments as a means of diagnosis unless there is suspicion amounting practically to cer-

tainty that the case is hysterical. There is a very grave risk of passing a bougie through the wall of an aneurysm, through the floor of an epitheliomatous ulcer, or through an œsophageal diverticulum, and the results of such a proceeding may well prove disastrous. If it is considered desirable to pass an œsophageal bougie the following technique may be adopted :—

Sit the patient up in a hard chair and direct him to extend his head as much as he can with comfort. Instruct him to open his mouth and to breathe steadily, deeply, and persistently through the nose. Now, having smeared the bougie with glycerine, pass it, unaided by a finger in the mouth, boldly down the posterior pharyngeal wall. The patient should be instructed to swallow as the instrument touches the wall of the pharynx. A slight obstruction will be noticed as the point enters the œsophagus at the level of the cricoid cartilage. Hysterical spasm will produce a *temporary* obstruction. Organic obstruction produces a *permanent* obstruction, the level of which can be estimated by noticing the distance the bougie has passed from the patient's front teeth. Force should never be used.

(vii.) If a semi-solid bolus of bismuth and oatmeal be swallowed, its course can be followed with a fluoroscopic screen, and any delay or obstruction can be accurately noted as well as the position where this occurs.

This method is more informative and vastly safer than the use of a bougie.

(viii.) Epithelioma of the œsophagus is most common at the level of the tracheal bifurcation, next at the cardiac orifice, and lastly at the level of the cricoid cartilage.

V. THE STOMACH.

(*a*) **Preliminary Remarks.** At birth the capacity of the stomach is only 1 oz. to 1½ oz. ; in the adult it is from 2 to 3 pints.

The position of the stomach is variable, since there is only one fixed point, the cardiac orifice, which is situated 1 inch to the left of the sternum along the seventh costal cartilage.

The pylorus should be about 2 inches above and 1 inch

to the right of the umbilicus, that is to say, 1 inch to the right of the mid-point of the line joining the episternal notch to the pubic symphysis.

The lesser curvature of the stomach is nearly vertical and its long axis is about 20° removed from the vertical. The fundus occupies a considerable part of the left "bed" of the diaphragm and extends as high as the fifth intercostal space in the mid-clavicular line, while to the left it is in contact with the spleen.

The lower level of the greater curvature of the stomach does not quite reach the transverse plane of the umbilicus in health.

(*b*) **Physical Examination of the Stomach.** *Inspection* shows no evidence of a healthy stomach. A dilated stomach may be apparent as a definite bulge with stomach outline in the epigastrium and left hypochondrium. If the dilatation is due to pyloric obstruction, peristalsis may be visible from left to right. Rarely reverse peristalsis may be detected; when present this is pathognomonic of organic obstruction. It is perhaps most often seen in congenital hypertrophic stenosis of the pylorus.

Peristalsis may be elicited sometimes (but not in health) by tapping and light "dipping" with the pulp of the fingers in the left hypochondrium.

Certain tumours, especially of the pylorus, may be visible and may be observed to move with respiration.

Palpation may reveal the presence of a tumour, which may be hard or soft, fixed or mobile.

Pyloric tumours are the ones most easily felt, and until adhesions are formed which bind them to the liver and other neighbouring viscera they are freely moveable both on respiration and manipulation.

Palpation also shows whether there is any tenderness or not. The size and position of any tender area should be established.

In chronic gastritis there is slight diffuse tenderness in the epigastrium, sometimes with an area of maximum intensity about 1½ inches below the xiphoid.

In gastric ulcer there is often a very small area of acute tenderness just to one side or other (usually the left) of

the mid-line about 2 inches above the umbilicus, but the points of tenderness are necessarily very variable, as the ulcers may be situated anywhere.

In examining for gastric tenderness palpation should be very light and performed with the pulps of the fingers and with *warm* hands.

Cutaneous hyperæsthesia is often very marked to the left of the mid-line in cases of gastric ulcer.

Succussion (stomach-splash) is elicited by placing the palm of the left hand over the left lower ribs behind and jolting smartly with the flat of the right fingers in the left hypochondrium. Succussion is a confirmatory sign of gastric dilatation, but is only of value in conjunction with other signs and when it is elicited at times when the stomach should be empty, that is to say, six or more hours after a meal.

Percussion. The stomach note varies greatly with the amount of gas in the organ. When gas is present the note is very tympanitic, even more so than that over the distended colon. When the stomach is quite empty of gas (which but rarely happens) the note is dull, and under these conditions inflation must be practised before percussion can be of value.

A glass of soda-water is often sufficient to distend the stomach, but a surer method is to administer half a drachm of sodium bicarbonate dissolved in a little water followed immediately by a like amount of tartaric acid. Perhaps the best method is to pass a gastric tube to which a pump is attached and gently to inflate the viscus by this means.

Even with an inflated stomach ordinary percussion is not reliable, but auscultatory percussion may be of great value. To perform auscultatory percussion the chest piece of the stethoscope is placed over a piece of undoubted stomach, say 2 inches below and to the left of the xiphisternum: using this as a centre light percussion is made along lines radiating from it and that point noted on each line where the *pitch* of the note changes as heard through the stethoscope. The lower border of the stomach can be mapped out with considerable accuracy by this means.

Merely drawing the finger along the skin radially from the bell of the stethoscope gives almost equally good results.

The upper limits of the stomach are apt to be obscured by liver and lung; the lower border should be a curve the lowest point of which does not reach the transverse umbilical plane. By means of auscultatory percussion displacements of the stomach as well as dilatation may be recognised. Doubtful tumours may be suspected to be of the stomach wall if the gastric note runs right up the mass with *no* preliminary change.

Inflation of the stomach will often render a tumour of the fundus more apparent than before, or even bring into notice one that was hitherto unsuspected.

(*c*) **Certain Investigations in the Diagnosis of Gastric Disease.** (i.) THE GASTRIC SECRETIONS AND THE CONSIDERATION OF VOMITED MATTER. The flow of gastric secretion is immediately determined by the presence of food in the stomach, but it can also be excited by the smell and probably also by the mere sight of food.

The secretion of hydrochloric acid is abundant during the half hour directly following the ingestion of food; this acid at once enters into combination with the albuminous constituents of the food and prepares the way for the eventual production of peptones.

Thus for half an hour or perhaps rather longer after a meal there is no free hydrochloric acid in the stomach and the fermentation of the carbohydrate portion of the meal proceeds unchecked. When, however, the acid affinities of the proteid bases are satisfied (forty-five minutes), the continued secretion of HCl causes inhibition of fermentation and the appearance of *free acid*. A large amount of proteid in a meal delays the appearance of free acid, and much carbohydrate has the opposite effect.

Lactic acid is never present at the same time as *free* hydrochloric acid; indeed, except for such as may be taken in with the food, lactic acid is not found in the healthy stomach.

Pepsin is always present where there is any free HCl, and very often when there is not; the secretion of this ferment is much less easily inhibited than is that of the acid.

Vomiting may be due to a great many different causes such as :—

(*a*) *Local* inflammation of the stomach, whether acute or chronic ; dilatation of the stomach, whether obstructive or atonic.

(*b*) *Reflex* causes, such as obstruction of the intestine, peritonitis, biliary or renal colic.

(*c*) *Emotional* causes, such as fright, rage, or disgust.

(*d*) *Toxic* causes. Certain drugs, such as alcohol, tobacco, opium or chloroform, and morbid blood conditions, such as uræmia, the vomiting of pregnancy or the periodic vomiting which is probably due to "acidosis."

(*e*) *Diseases of the Central Nervous System*, such as cerebral tumour, meningitis, concussion, locomotor ataxy, and migraine.

The vomit is acid unless (1) it is mixed with intestinal contents ; (2) it comes from an œsophageal pouch ; (3) in a very few cases of (*a*) cancer of the stomach, (*b*) achylia.

In cases of gastric carcinoma the acidity is due to bound acid, which is but rarely completely absent, though commonly there is no free acid to be found.

The type of vomiting may assist the diagnosis. Thus cerebral vomiting is free from effort, not preceded by nausea, and independent of food.

In dilatation of the stomach the vomit is often copious and offensive from the presence of fatty acids ; it is brought up at longish intervals of, say, twenty-four to thirty-six hours and contains food which was swallowed many hours previously.

Vomiting at a more or less constant hour each day is seen in pregnancy, and the morning vomit of the chronic alcoholic is well known.

In gastric ulcer the vomiting often occurs twenty to thirty minutes after the food is swallowed and is preceded by pain, which is relieved when the stomach is emptied ; the material vomited is likely to show advanced proteid digestion for the time it has been in the stomach.

In chronic gastritis and in simple hypo-acidity the vomit may contain much mucus and poorly-digested or even decomposing proteid. Of course all stomachs secrete a good

deal of mucus, but this is largely digested in a medium so acid as the healthy gastric juice.

Fæcal vomiting indicates intestinal obstruction (usually complete obstruction), and the further the obstruction from the pylorus the greater the scope for true fæcal vomiting.

Blood which has been partly digested in the stomach before being vomited assumes the characteristic coffee-ground appearance.

The examination of vomited matter chemically and microscopically does not provide very trustworthy information ; for these purposes a test meal should be employed.

(ii.) Test Meals and their Analysis. Since the gastric secretions are profoundly modified by diseases of the stomach and elsewhere much information can be gained by withdrawing the stomach contents and analysing them at certain intervals after a meal of known composition has been eaten.

A variety of test meals have from time to time been advocated and used by different observers ; but the precise composition of the meal does not very much matter provided that the following points are remembered :—First, do not introduce into the stomach any appreciable amount of any of the substances for which you are afterwards going to test the gastric contents—for example, do not give a pint of milk if you are anxious as to the subsequent presence of lactic acid. Secondly, arrange your meal with great care so as to approximate to a certain extent in quality, quantity, and time of administration to the ordinary physiological processes and habits of the particular individual. Thus, if a man habitually never takes any breakfast do not give him a test meal of egg, bread and butter, and tea at 8.30 in the morning, for by so doing you will call on his stomach for certain activities quite foreign to its own particular physiological expressions, and the results may well be most misleading. Thirdly, remove the meal at the time when the maximum activity of gastric secretion is in progress ; this varies greatly with the individual and depends on the motility of the stomach to some extent. Hence, several test meals may be necessary to determine the optimum time of removal for the particular patient.

Several standard test meals are described, but we think that the poor results, from a diagnostic point of view, which are so often experienced are largely due to too slavishly adhering to one particular meal regardless of the habits of the patient.

Ewald's test breakfast is the meal most commonly employed in this country. It consists of 40 grms. of white bread and 400 cc. of water or weak tea without milk or sugar. This meal should be removed about one hour after administration. Only from 40 to 80 cc. should be recoverable ; any considerable excess indicates hypersecretion or motor insufficiency.

Riegel's test meal consists of 200 grms. of lean beef, 150 grms. of mashed potatoes, and a plate of bouillon. This meal offers a much stronger stimulus to the secretory mechanism of the stomach than Ewald's. It should not be removed for three or four hours.

Fischer's test meal is a combination of both of the above and is probably as good as any. It consists of 40 grms. of white bread, 200 grms. of lean beef, and 400 cc. of water. It should be removed in three hours.

It is a good plan to estimate the motility of the stomach by watching the passage of bismuth out of the organ with a fluoroscopic screen instead of settling arbitrarily the time when a given test meal shall be withdrawn ; the optimum time for removal can be gauged in this manner. Better results are obtained if the stomach tube is passed once or twice previously in order to accustom the patient to its use. Lastly, in cases in which there is a strong nervous element and the symptoms are definitely periodic, it is well to administer the test meal at such a time that it can be withdrawn when the usual symptoms are at their height.

To withdraw the meal the soft stomach tube is passed exactly as described for the œsophageal bougie (p. 322) ; the patient is then instructed to lean forward, to keep on breathing through his nose, and to contract forcibly his abdominal muscles ; this is usually sufficient to return the gastric contents. If not, milk the tube gently with the fingers, withdraw it a little, and push it down into the stomach again. It is not advisable to pour water down the

tube for the purpose of "siphoning" off the contents, as this alters the acid values.

The following examinations can now be made :—

(1) *Total Volume of Material Removed.* In the case of Ewald's test breakfast only one-sixth of the original 400 cc. should be regained (say 50 to 70 cc.).

If the stomach was washed out (as it should be) and left empty the night before, 100 cc. at the very outside should be allowed for healthy gastric secretion throughout the night, so that the removal of more than 170 cc. of material after the test breakfast indicates hypersecretion or motor insufficiency ; on the other hand, in the majority of healthy stomachs there is practically no secretion so long as food is neither swallowed nor smelled, so that the removal of so little as 40 to 70 cc. must not be regarded as evidence of hyposecretion.

(2) *The Presence of Free Hydrochloric Acid.*

(*a*) If a Congo-red test paper is dipped into the material a bright-blue colour is produced by *free* mineral acid. Free lactic acid also turns this paper blue, but the colour is much less pronounced.

(*b*) Gunzberg's test is the one usually adopted. The solution is vanillin 1 part, phloroglucin 2 parts, and alcohol 30 parts ; it should be kept in the dark and tightly corked. A few drops of this solution are evaporated slowly just to dryness in a porcelain capsule ; a drop of the gastric content is now run up to the edge of the deposit and further gentle heat applied. At the point of contact a crimson line is formed if free mineral acid is present to the extent of ·005 per cent.

Absence of free HCl at the times when it should be present may occur in malignant disease, achylia, or in true chronic gastritis ; it should be present forty minutes after a carbohydrate meal or seventy-five minutes after a proteid meal.

(3) *The Total Acidity.* To estimate this the gastric contents should be filtered, preferably by a suction filter : 10 cc. of the filtrate are titrated with decinormal caustic soda, the indicator being a few drops of ½ per cent. phenolphthalein in alcohol. The number of cc. of caustic

solution necessary to turn the indicator is multiplied by 10, and the result is the total acidity per cent. For example, if 5 cc. of caustic are used to neutralise the 10 cc. of gastric juice the total acidity is expressed as 50 acidity per cent.

In health the total acidity per cent. varies from 40 to 60 one hour after the test breakfast and from 100 to 120 three hours after the Riegel test meal.

If desired the total acidity can be expressed gravimetrically as the percentage acidity of HCl in the gastric juice. Clinically it is sufficient to remember that an *acidity per cent.* of 55 is equivalent to the average healthy gastric acidity of ·2 per cent.

(4) *To Estimate the Free HCl*, titration with decinormal soda is carried out until the test for free acid is negative, for it is assumed that the *free acid* is neutralised by the soda solution before the bound acid. Accordingly during the titration repeated investigations are made with Gunzberg's reagent and a drop of the gastric juice, which is removed on a glass rod after stirring in the freshly added soda.

This process is rather tedious, but is reasonably accurate.

(5) *Lactic Acid.* This acid is tested for with Uffelmann's reagent. This consists of 2 per cent. carbolic acid, to 20 cc. of which one drop of 10 per cent. ferric chloride is added just before use.

The amethyst colour of this solution becomes canary-yellow in the presence of lactic acid.

Mineral acids destroy the purple tint of the reagent, leaving a perfectly colourless solution.

(6) *Pepsin.* This is almost always present if there is any free hydrochloric acid, so that practically it need only be investigated in cases in which there is no free acid, and even in these it is nearly always found.

To examine for it, it is necessary to add sufficient HCl to the gastric contents till there is free acid present, as shown by the Congo-red test paper. A disc of boiled white of egg is now put into the solution and the whole placed in an incubator at 37° C. If pepsin is present the edges of the disc of egg white will become rounded by the ferment action in from half an hour to three hours.

(7) *Microscopical Examination.* A wet preparation should be used, that is to say, a few drops of the gastric contents on which a cover-slip is floated. One drop of dilute watery methylene blue may conveniently be mixed in with the solution. Excess of moisture should be removed by blotting-paper and the cover-slip ringed with vaseline or, better, paraffin wax for examination with an oil immersion lens.

The following points may be elicited.

(*a*) *Boas-Oppler Bacillus.* This bacillus is said only to be present in cases of malignant gastric tumours, or possibly in cases of true chronic gastritis, that is to say, where there is no free HCl.

It is a large bacillus, often growing into long threads or chains like the anthrax bacillus, it is Gram-positive, does not form spores, and is easily cultivated on media which have been acidified with lactic acid.

(*b*) *Sarcinæ,* groups of cocci (Gram-positive) occurring in groups of eight, formed by division into three planes at right angles to each other.

Sarcinæ are very characteristic of dilated stomach, but are seldom seen in cases of malignant disease.

(*c*) *Moulds, Yeasts, and other Varieties of Bacteria.* Moulds and yeasts are present where there is free HCl.

Bacteria only occur in large numbers when there is no free acid.

(*d*) *Pus Cells* are but rarely present ; they may occur where there is an ulcerating new growth or in cases of phlegmonous gastritis.

(*e*) *Very occasionally* fragments of carcinoma or sarcoma may be visible.

(8) *The Presence of Blood.* The presence of blood may of course be obvious, or the amount may be so small that it can only be demonstrated by the occult blood test.

The Occult Blood Test. The best test for very minute traces of blood is that described by Colwell and MacCormack :—

To prepare the reagent :

Dissolve ·1 grm. benzidene and ·1 grm. of sodium perborate in 10 cc. of glacial acetic acid.

This solution should be freshly made for each day's work.

To prepare an etheral extract of the suspected material:

Take 5 cc. of the material to be tested (vomit, fæces or urine) and shake for two minutes with 5 cc. of glacial acetic acid in a *stoppered vessel*. Add to this 10 cc. of ether and shake again. Now add 2 or 3 cc. of water and allow the etheral extract to rise to the top of the fluid by standing the vessel for a few minutes.

To perform the test:

Place 5 cc. of the etheral extract in an evaporating dish and allow the ether to evaporate. Add 1 cc. of the benzidene perborate solution. If blood is present an intense blue colour appears. If the colour comes up slowly or is greenish rather than blue the blood is present in the most minute traces.

A positive test is given by any form of fresh animal food, even fish, and also green vegetable matter, so that the patient must be fed for forty-eight or seventy-two hours on milk, eggs, tea, bread and butter, and potatoes before the test can be profitably applied.

Iodides, which give a positive reaction when peroxide of hydrogen is used, are negative to perborate.

The test can of course be applied directly to the suspected material, but the use of an etheral extract gives very much more reliable information.*

Conclusions to be drawn from the Examination of Test Meals. It is obvious that the most important facts to be ascertained from test meals are first the presence or absence of free HCl, and secondly the proportion of free and combined acid in regard to the total acidity. The results of the remaining investigations vary almost directly with the amount of free acid present and therefore are mainly confirmatory in their scope. (Some observers place more reliance on the presence of lactic acid than on the absence of free HCl in the diagnosis of malignant disease.)

In the consideration of gastric disease cases can be sharply

* In the case of solid fæces an etheral extract is best made by rubbing up a small piece of fæcal matter with glacial acetic acid in a watch-glass, diluting with water, and then adding the ether as above described.

divided into two classes—the *Innocent* and the *Malignant*: it is always of prime importance to exclude the latter.

The justifiable deductions to be drawn from the acidity of the stomach contents, based on the summary by Sahli, are as follows :—

(*a*) *With Normal Acid Secretion* there may be :

(1) Some cases of ulcer of the stomach (more often chronic than acute).

(2) Gastric Neuroses.

(3) Simple atony.

(*b*) *With Increased Free HCl* (that is, more than ·25 per cent. and total acidity per cent. of more than 70) :

(1) Most cases of simple ulcer, either gastric or duodenal.

(2) Hypersecretion, either continuous or only during digestion, but not the increased secretion (apparent) of impaired motility.

(3) Gastroxynsis, or the paroxysmal hypersecretion which occurs in some forms of neurosis and is accompanied by copious acid vomit.

(4) Some cases of chlorosis.

(5) The initial stages of chronic gastric catarrh.

(*c*) *With Diminished Acidity* :

(1) Most febrile conditions.

(2) Severe anæmias.

(3) The later stages of chronic gastric catarrh.

(4) Any state of profound debility.

(5) The later stages of heart disease.

(*d*) *With the Repeated Absence of Free HCl* :

(1) More advanced stages of group (*c*).

(2) The acute specific infective diseases.

(3) Malignant disease of the stomach.

(4) The late stages of malignant disease elsewhere.

(5) Pernicious anæmia.

The complete absence of free HCl, as shown by several examinations, is always of significance ; for the rest it is important to remember that average acidities vary within considerable limits with the nationality and also with the individual.

(iii.) The Motility of the Stomach. Undoubtedly the

best method of investigating the motility of the stomach is to make the patient swallow 6 oz. of semi-solid bismuth paste and to watch with a fluoroscopic screen the movement of the stomach and the passage of its contents into the duodenum.

If the use of the X-rays is not practicable a meal consisting of meat, bread, and tea may be given without lavage at 7 o'clock in the evening.

The stomach contents should then be removed (before any further food has been taken) at 7 o'clock in the morning. The presence of food in the material removed denotes great impairment of motility.

It is probable that there is impaired motility if a fasting stomach contains *more than* 100 cc. of secretion first thing in the morning.

After an ordinary "mixed" meal the healthy stomach is empty within seven hours.

Excessive motility for the most part accompanies hyperacidity; it is therefore common in cases of gastric ulcer, duodenal ulcer, and gastric neurosis.

Impaired motility is more serious, and practically always indicates more or less dilatation of the stomach; this may be due to simple atony of the musculature or to some obstruction to the outflow through the pylorus. In the latter case there may be definite muscle hypertrophy combined with the dilatation, the result of an attempt to overcome the obstruction.

(iv.) The Absorptive Power of the Stomach. This function is not commonly of great importance, since healthy intestines can easily compensate for the absence of normal gastric absorption, especially if the motility of the stomach is unimpaired. Further, the absorption from the stomach is diminished in practically every gastric disorder and abolished temporarily in acute infections.

If it is desired to test the absorptive power of the stomach a gelatine capsule containing 3 gr. of potassium iodide should be administered (preferably with an Ewald's test breakfast in order to ensure constant conditions (Sahli)). Iodine should appear in the saliva in from five to fifteen minutes. It should also be present in the urine within half an hour. The test is made by adding to some saliva

(or urine) a little starch and a drop or two of strong HCl; iodine is shown by a blue coloration. This test may be tried with the saliva every few minutes until it is positive. The practical value of this test is very small.

(*d*) **Clinical Varieties of Gastric Disorders.** The symptomatology of the various gastric disturbances is so anomalous and the physical signs are often so vague that accurate diagnosis becomes a matter of the utmost difficulty. It is especially important always to remember that gastric symptoms are present in very many diseases which in themselves are quite distinct from any primary stomach affection. In such cases it may be fatal to focus the attention on the gastric organ or even on the alimentary tract at all. It is important to cultivate a comprehensive outlook in all cases of disease, but especially so in cases in which the chief symptoms are referred to the stomach. To illustrate this truth it is only necessary to mention the dyspeptic symptoms of early pulmonary tuberculosis and old-standing valvular disease of the heart, the gastric crises of tabes dorsalis, the vomiting of uræmia, and the sickness and abdominal pain of Addison's disease. In any of these a diagnosis of gastritis would be nothing more than a confession of ignorance, and treatment based on such a diagnosis must prove far from satisfactory.

Disorders of the stomach may be considered under certain definite headings, but it must be understood that any sharp classification is impossible in view of the inevitable overlapping that must occur from the association of two or more of the under-mentioned conditions :—

- A. Inflammation
  - Simple Acute Gastritis.
  - Phlegmonous Gastritis.
  - Toxic Gastritis.
  - Chronic Gastritis.
  - Atrophic Gastritis.
- B. Gastric Ulcer.
- C. Gastrectasis
  - Simple or atonic.
  - Obstructive.
    - Simple.
    - Malignant.
    - Congenital Pyloric Obstruction.

D. New Growth.

E. Disorders of Secretion .. { Achylia. / Hypersecretion. / Hyper-acidity. / Hypo-acidity.

F. Essential Neuroses .. { Anorexia Nervosa. / Bulimia. / Pica.

A. Inflammation. (i.) *Acute Gastritis.* This condition is produced by the ingestion of irritating or toxic material—for example, excess of alcohol (in those who are unused to it), unsuitable or highly-spiced foods or those that are tainted, and sometimes, in susceptible persons, by simple chill.

A second group of cases are found in the later stages of many chronic diseases, such as tuberculosis, exophthalmic goitre, pernicious anæmia, etc.

Lastly, an apparent acute gastritis may be the first symptom of an acute infection, such as pneumonia or scarlet fever, especially when the patient is a child.

The symptoms are loss of appetite, vomiting, headache, and usually more or less epigastric pain. There is a furred tongue and generally constipation, though sometimes there is an associated enteritis with diarrhœa.

The mucous membrane of the stomach is inflamed and coated with mucus, the vomit contains undigested and often fermenting food, as well as slimy mucus ; it is practically always deficient in acidity and free HCl may well be entirely absent. When the retching is persistent the vomit may contain bile.

The diagnosis does not commonly present much difficulty. Gastric crisis may be excluded by examining for the other signs of Tabes (*vide* p. 544).

The acute exanthems in children may always be suspected and their appropriate features sought for.

In the majority of cases the history of the recently-taken food and drink affords a valuable clue.

The promptness with which primary acute gastritis yields to starvation and a mercurial purge is worthy of note.

(ii.) *Acute Phlegmonous Gastritis.* This condition is,

happily, rare ; it is an acute streptococcal inflammation of all the coats of the stomach. It may, but usually does not, go on to abscess formation. The etiology is obscure.

The symptoms are very acute epigastric pain, nearly always accompanied by voimting. The vomited matter is usually totally anacid, and even the ferments may be absent. There is always evidence of profound constitutional disturbance and of septic absorption. The condition is rapidly fatal except in a few cases. In these the inflammation localises with the formation of an abscess which ruptures into the stomach (a rare event).

(iii.) *Toxic Gastritis.* By this is meant an acute gastritis, depending on the swallowing of some definitely poisonous material, such as oxalic or mineral acid, caustic, liniment, arsenic, or the like.

The symptoms are those a of a very acute gastritis, associated with profound collapse, which often proceeds to coma.

The diagnosis depends on the history of swallowing some poison and of the onset of symptoms with appalling suddenness, the possible evidence of burns about the mouth or pharynx, the presence of blood and shreds of mucus in the vomited material, the smell of this and its analysis for such poisons as may be suspected.

(iv.) *Chronic Gastritis.* Essentially this is a chronic catarrhal affection of the gastric mucous membranes, associated with the production of excessive mucus and a deficiency in free HCl. A common cause is the prolonged ingestion of irritating and unsuitable articles of diet, particularly alcohol ; again, a diet that is in itself harmless may be rendered irritating by imperfect mastication. Another cause is chronic venous engorgement ; this variety is a conspicuous feature of advanced valvular disease of the heart and also of portal cirrhosis.

Chronic gastritis is often secondary to local carcinoma and is met with in the later stages of any chronic debilitating disease.

The symptoms are very variable, and include :—

(*a*) Slight general malaise with a tendency to headache.

(*b*) Epigastric pain with slight diffuse tenderness,

most apparent from one and a half to three hours after a meal. The pain is sometimes described as going through the chest to the interscapular region, mostly on the left side.

(*c*) Vomiting and nausea; frequently immediately on rising in the morning, sometimes two to three hours after a meal.

(*d*) Constipation is the rule, but by no means invariable; indeed, alcoholics are often prone to diarrhœa.

(*e*) The tongue is furred and flabby, and there is often an unpleasant taste in the mouth.

(*f*) Flatulent distension and gaseous eructations are common.

The vomited material or the wash-out from a test meal shows excess of mucus, diminution or absence of free HCl, undigested and often fermented food and the presence of organic acids.

The total acidity is diminished as a rule, as is the total secretion.

The morning vomit of the alcoholic consists entirely of bile-stained mucus.

The symptoms depend to a certain extent on the motility of the stomach. When this is normal or increased, the symptoms are slight, because the food is passed on rapidly to the intestine, where compensatory digestion takes place. When the motility is diminished, the symptoms are aggravated. As a rule the motility is somewhat diminished.

The differential diagnosis may be far from simple. The history is important, and primary diseases must be excluded. When this has been done the chief trouble is to exclude gastric ulcer, malignant disease of the stomach, and atonic gastrectasis.

In *Gastric Ulcer* the pain is more severe and occurs sooner after food, the tenderness is more acute and often localised, while a test meal shows hyperacidity and absence of mucus.

Definite hæmatemesis (not capillary oozing) is of the utmost importance as indicating ulcer provided cirrhosis of the liver and carcinoma can be excluded.

In *Gastric Carcinoma* the pain is more severe and more

constant; vomiting is more frequent. A mass may be felt and cachectic symptoms are not long delayed. Further, obstructive symptoms may be present, and the Boas-Oppler bacillus may be found in the stomach contents as well as lactic acid, but no free HCl.

In *Atonic Gastrectasis* the dilated stomach can be diagnosed by auscultatory percussion, X-rays, and the test meal, which, with the absence of visible peristalsis or a palpable tumour, will help to exclude organic obstruction.

(v.) *Atrophic Gastritis.* This consists in atrophy of the gastric mucous membrane and is most frequently seen as the result of long continued chronic gastric catarrh; it also occurs as the result of infiltrating gastric carcinoma, and sometimes when the malignant growth is quite localised.

Achylia is a necessary accompaniment to atrophic gastritis, but is sometimes found as a pure neurosis.

The chief symptom is repeated vomiting soon after meals; in cases in which the motility is good there may be no symptoms. A test meal will surely demonstrate achylia, and so will help in the diagnosis of atrophic gastritis. The food is returned practically unchanged, there is no free acid, the total acidity per cent. is under 5, there is no organic acid, and pepsin is absent.

These findings do not necessarily exclude carcinoma. But the absence of obstruction as shown by X-rays would suggest the presence of simple atrophy rather than growth.

It should be remembered that the atrophy of the gastric mucosa is secondary to some other condition in the great majority of cases and is therefore a symptom rather than a disease; also that the presence of achylia in itself is not sufficient to warrant the diagnosis of atrophic gastritis, since all gastric secretions may be suspended from purely functional nervous causes.

B. Gastric Ulcer. **Etiology.** Gastric ulcer is much more common in women than men and is most often seen between the ages of 20 and 35 years, but practically no age is immune. Its causation is obscure, but the fact that it is associated with hyperacidity and very frequently with anæmia seems rather to support the idea that minute venous thromboses in the gastric submucosa may be the first stage and that

auto-digestion of the mucous membrane overlying these "infarcted" areas is the second stage. Possibly bacteria may play a part in the production of the gastric ulcer; certainly oral sepsis and extreme constipation are very often features of such cases, and these are both potential sources of bacterial invasion of the stomach.

Situation and Varieties. The acute ulcers are often multiple; they are most often to be found on the lesser curvature of the stomach and rather near the pylorus, but may well occur in any part of the stomach. They may all heal completely, or one or more may persist and gradually develop into a chronic ulcer. This is usually single and often bound by dense adhesions to neighbouring structures, especially the pancreas.

Any ulcer may perforate, but the so-called "acute perforating ulcer" is nearly always situated on the unsupported anterior wall of the stomach. Those ulcers which are on the lesser curvature readily form adhesions to other viscera and so do not commonly perforate suddenly, though not infrequently they leak slowly and form a subphrenic abscess.

The actual rupture of an "acute perforating ulcer" and of a chronic ulcer may be equally sudden, but in the former case the perforation may be almost the first sign of gastric ulcer, and certainly may occur within a few hours of the very first gastric symptom, whereas the chronic ulcer gives evidence of its presence for months or years before the sudden symptoms of perforation supervene.

Malignant disease (carcinoma) develops in a considerable proportion of chronic ulcers; hence the title "malignant ulcer."

Course. Considering the difficulty in accurately diagnosing many cases of early gastric ulcer and the impossibility of satisfactory home treatment in the majority of cases, it is perhaps fortunate that perforation takes place in only a very small proportion of all cases.

The tendency is for rapid improvement to follow appropriate treatment in acute cases, but, owing to a renewal of the same circumstances which originally caused the first ulcer, when the patient resumes her ordinary life the majority of cases relapse, not once only, but again and again.

Of course, in the upper classes, where prolonged treatment can be carried out and a hygienic life persisted in, complete cure is more usual; but gastric ulcer is not common in the upper classes—it is shop hands and domestic servants that seem chiefly to be affected.

Since, then, so many cases relapse, a chronic ulcer is often the result within a few years of the first symptom, and though acute ulcers respond well to medical treatment, chronic ones do not; indeed, the symptoms frequently return as soon as a diet of solid food is permitted. The likelihood of a chronic gastric ulcer becoming malignant has been mentioned, and this affords another cogent reason for advocating operative interference (gastro-jejunostomy) as soon as *chronic* gastric ulcer has been diagnosed. Acute ulcers should not be submitted to operation, partly because they tend to heal if given a chance, and still more because they are so often multiple.

Symptoms and Signs. The chief symptoms of gastric ulcer are as follows:—

(*a*) *A burning pain* in the epigastrium (often localised to a definite area) and occurring from twenty minutes to two hours after a meal—that is to say, at the height of acid secretion. The pain may radiate to the back and left scapula and often varies with change of posture. It ceases when the stomach becomes empty and is the more severe the nearer the ulcer is to the pylorus.

(*b*) *Nausea* accompanies the pain, and in about 20 per cent. of all cases there is *frequent vomiting*. When the patient vomits the pain is relieved. In about 25 per cent. of all cases there will be hæmatemesis sooner or later.

(*c*) *Tenderness*. This depends on the site of the ulcer, but is usually found over an area of 1 to 2 square inches about 2 inches above the umbilicus and rather to the left of the middle line. This localisation of tenderness is an important point.

(*d*) *Cutaneous hyperæsthesia* may be present over the same area as that just described.

(*e*) *Hyperacidity* and *increased motility* are almost constant features of acute ulcers; normal acidity is possible in chronic ulcers.

(*f*) *Anæmia* and *constipation* are the rule ; the tongue, however, is clean if there is no associated gastritis.

(*g*) *Hæmorrhage.* This may be almost the first sign of ulcer or it may be long delayed. It may be slight in amount or very copious. When very profuse it may be fairly bright red ; more often it is altered by the stomach contents and becomes black and broken up (coffee-grounds). Sometimes there is considerable hæmorrhage without vomiting ; under these circumstances the blood is passed per rectum only (melæna).

Differential diagnosis of gastric ulcer :—

From *Chronic Gastritis* (*vide* p. 338).

From *Malignant Disease.* The age of the patient as well as the length of the history and the absence of cachexia may be important points. If there be hæmatemesis it is likely to be much more profuse and less frequent in simple ulcer. The pain in ulcer is more paroxysmal and is absent when the stomach is empty. No mass can be detected. The test meal in simple ulcer shows increased total acidity and ample free acid ; in carcinoma there is no free acid, the total acidity is greatly diminished, and the Boas-Oppler bacillus may be found in the stomach contents. Definite obstruction, as shown by X-rays, is more common in malignant disease than in adhesions from simple ulceration.

From *Gastrostaxis.* By gastrostaxis is meant hæmorrhage from the stomach when there is no ulceration or erosion to account for it. This condition may not be very common, but there can be no doubt that it does occur, since there have been fatal cases where a most laborious examination of the stomach has failed to show any source for the bleeding.

Like gastric ulcer, gastrostaxis is a disease of young women, and a positive differential diagnosis between the two conditions is impossible. At the same time gastrostaxis may be suspected if the following features are present :—

(*a*) Absence of any gastric symptoms before the hæmorrhage.

(*b*) Occurrence of the hæmorrhage at or about the time of the menstrual period.

(*c*) Very rapid recovery from the anæmia which results

from the loss of blood. In gastrostaxis cases the hæmoglobin is restored to normal in four or five weeks; in gastric ulcer it often remains reduced for several months.

(*d*) The total acidity is normal or diminished.

From *Duodenal Ulcer*. An ulcer situated at or about the pyloric ridge may be, anatomically, either gastric or duodenal, but, clinically, differentiation is impossible. The following points are characteristic of true duodenal ulcer :—

(*a*) The male sex is more liable than the female.

(*b*) The pain is not noticed for from three to four hours after food, and is relieved by the next meal (hunger pain).

(*c*) The pain and tenderness extend definitely to the right of the mid-line: sometimes the pain is referred to the right scapula.

(*d*) A common tender spot is at the junction of the lower and middle thirds of the line joining the umbilicus to the tip of the ninth rib.

(*e*) Melæna is common in duodenal ulcer. Hæmatemesis is rare, as indeed is vomiting or nausea.

C. Gastrectasis (Dilatation of the Stomach).

(i.) *Acute Paralytic Distension of the Stomach*. This rare condition appears to be caused by extreme overloading of the stomach with food and drink; it is not likely to occur unless there is a certain amount of previous atony and is most frequently seen in advanced life. The result may be death from heart failure, especially if the heart is already damaged by fibrosis.

The correct diagnosis may be suspected if collapse occurs shortly after a very heavy meal.

The physical signs are obscure, but there may be a great increase of stomach resonance; there is no visible peristalsis, but often there is repeated unproductive retching.

The symptoms are those of cardiac embarrassment.

The correct treatment is to empty the stomach; five or six pints may be recovered, and relief is often pronounced.

(ii.) *Atonic or Non-obstructive Dilatation of the Stomach (Chronic)*. This condition is the result of prolonged chronic gastritis, usually with more or less atrophy and always with impaired motility (continuous hypersecretion is an infrequent

cause). Food is permitted to delay in the stomach unduly, and gradually dilatation of the viscus takes place. It seems probable that impaired motility is the most essential factor.

The symptoms are mainly those of flatulent dyspepsia; there is constant epigastric oppression and discomfort with a sense of distension, except on the rare occasions when the stomach is quite empty. The smallest amount of food produces an exacerbation of these symptoms, and violent eructations are a distressing feature of the case.

General neurasthenic symptoms are often present. True pain (apart from a sense of distension) is not necessarily found, but may occur in paroxysms associated with peristaltic efforts (possibly visible) shortly after the taking of food.

Vomiting is a prominent feature when any considerable amount of dilatation has developed; it occurs at infrequent intervals (twenty-four, thirty-six, or forty-eight hours) and is of large amount and frothy appearance, while the food present may be recognised as having been taken many hours before. The total acidity is variable; often it is slightly sub-normal: if high, active ulceration (simple) may be suspected; if very low, malignant disease is a possibility unless achylia can be demonstrated.

Sarcinæ are usually present in the gastric contents.

Physical Signs. The important physical signs of a dilated stomach are :—

(*a*) Increase of stomach resonance to auscultatory percussion (if necessary, after inflation of the organ).

(*b*) Succussion, or stomach splash, elicited six or more hours after a meal.

(*c*) Peristalsis, if present, is almost pathognomonic.

(*d*) Visible distension with obvious stomach outline (this is only present in extreme cases).

The diagnosis may be clinched by examination with the X-rays, or by the use of the stomach tube, which will demonstrate the presence of residual contents when the stomach should be empty.

(iii.) *Obstructive Gastrectasis.* This may be *simple* or *malignant.* The greater number of cases are due to carcinoma of the pylorus, but cicatricial contraction during the healing

of a simple ulcer is not infrequent. A third group occurs as Congenital Hypertrophy of the Pylorus (*vide infra*).

In atonic gastrectasis the stomach wall is thinned and feeble; in obstructive gastrectasis it is, at first at least, vigorous and hypertrophied from its attempts to overcome the obstruction.

The signs and symptoms of the actual dilatation will be the same as those described in the previous section, but in organic obstruction vomiting is more persistent and more regular; visible peristalsis is common (reverse peristalsis is pathognomonic), and the pylorus may be palpable. In addition, the other features of the underlying cause may be present, and, of course, the test meal reactions will vary with the pathological condition.

(iv.) *Congenital Hypertrophic Pyloric Stenosis.* As the name implies, this disease is produced by narrowing of the exit from the stomach from muscular overgrowth of the pyloric ring. It is a congenital abnormality and symptoms generally appear the third or fourth week of life.

The diagnostic points are :—

(*a*) The age of the patient.

(*b*) The type of vomiting, which has aptly been termed "explosive"; so vigorous is it that the stomach contents are thrown a considerable distance.

(*c*) Constipation. After a few days this may become absolute, but this of course varies with the completeness of the pyloric obstruction.

(*d*) Visible gastric peristalsis, not infrequently of reverse type.

(*e*) A palpable pylorus.

(*f*) A larger stomach than is usual for the age of the child; this can be shown by gentle lavage with warm bicarbonate solution.

Spasm of the Pylorus may occur in infancy without true hypertrophy of the pylorus; the signs and symptoms are the same, except that the pylorus is not so likely to be palpable. The two conditions can only be distinguished by the effect of medical treatment, which is often successful in spasm, but which rarely cures a case of hypertrophic stenosis.

D. New Growth. The stomach may be affected primarily by malignant disease, or the growth may invade the stomach by direct extension, as from the head of the pancreas or elsewhere. Metastatic growths are rare. Stomach growths are practically always carcinomatous; sarcoma has occasionally been recorded, and very rarely adenomyomata have been described.

The pylorus is the usual site for new growth, next the lesser curvature, then the cardiac orifice, and lastly the fundus of the stomach.

It is deplorable that it is still the exception rather than the rule for a positive diagnosis of malignant disease of the stomach to be made in the absence of a palpable tumour. Much has been hoped for from the chemical analysis of the gastric contents after test meals, and much help is often afforded by these means; but the findings are, as yet, not sufficiently constant or exclusive in the very early cases, and it is only the early cases that can be cured by gastrectomy.

At the same time many cases are overlooked because the importance of two or three cardinal symptoms is not sufficiently recognised. These symptoms are not, in themselves, pathognomonic, but they are sufficiently striking to warrant the employment of all other available methods of examination. When every possible investigation has been carried out a careful consideration of the data thus afforded, together with the history and clinical phenomena, will often lead to exploratory operation and sometimes to successful excision of a growth which otherwise might have been treated symptomatically till too late.

Especially unfortunate are those individuals in whom carcinoma develops in a chronic gastric ulcer. Often they have been known to have gastric ulceration for years, and they are treated again and again for this condition until the presence of a large mass simply compels the revision of diagnosis. As already stated, every case of *chronic* gastric ulcer should be submitted to operation as soon as diagnosed.

Early symptoms which should excite suspicion of a possible gastric neoplasm are :—

(*a*) The occurrence of dyspeptic symptoms in a person over 40 years of age who was previously not afflicted with indigestion.

(*b*) Alteration in habits of defæcation in any one over 40 years of age—that is to say, constipation in one who was not costive before, or diarrhœa in one habitually constipated, is often an early sign of either rectal or gastric carcinoma. The rectum and sigmoid can be examined with a sigmoidoscope and, if free, attention can be focussed on the stomach.

(*c*) Rapid loss of weight, combined with any dyspeptic symptoms.

The signs and symptoms of gastric carcinoma are as follows :—

(*a*) *Cachexia.* This, of course, only suggests malignant disease somewhere ; it is, however, peculiarly early in gastric cancer. It is shown by an earthy pallor and a yellow, inelastic skin.

(*b*) *Palpable Tumour.* This is strongly corroborative, but is not absolutely pathognomonic, since a pylorus bound by dense adhesions to the gall-bladder will be readily palpable.

(*c*) *Pain.* This is a constant symptom ; it is generally a persistent, dull ache in the epigastrium liable to exacerbations at varying times after food.

(*d*) *Tenderness.* This is very variable and often conspicuously slight.

(*e*) *Vomiting.* This is present in 90 per cent. of all cases. It is most usual about two hours after food. The vomited material is important : meat is badly digested, starchy foods are well digested. Blood is very often present, rarely in large amount, and is frequently only detected by the *occult blood test* (*vide* p. 331).

(*f*) *Melæna.* Blood, often in minute traces only, is present in upwards of 90 per cent. of all cases.

(*g*) *The Blood.* A secondary anæmia is the rule, with a definite leucocytosis if the growth is ulcerating or if there are many metastatic deposits.

(*h*) *Fever.* In late cases there is an intermittent pyrexia, presumably due to septic absorption.

(*i*) *Gastrectasis.* This frequently develops if the growth is at the pylorus (*vide* p. 344).

(*j*) *Analysis of Gastric Contents after a Test Meal.* Riegel's or Fischer's meal should be employed. The absence of free HCl is an important sign. It happens very early while the total acidity is still normal, though in later cases as the secretion diminishes the total acidity falls also. Though important, the absence of free HCl is not *per se* conclusive, since it is also lost in advanced chronic gastritis, in achylia, and in pernicious anæmia. Achylia can be shown by the absence of pepsin in the gastric juice and pernicious anæmia by the blood examination; for no matter how closely the red cells and hæmoglobin in carcinoma may occasionally resemble those in pernicious anæmia, in carcinoma there is not a leucopenia.

The presence of lactic acid is regarded by some as almost as important as the absence of free HCl; it is of great service in helping to exclude chronic gastritis, since it is not found in this condition. It may also occur (but not constantly) in atony with hyperacidity, and also in atrophy with pyloric stenosis.

The Boas-Oppler bacillus is but rarely found in any non-malignant disease of the stomach. Sarcinæ are very rare in malignant disease. Fragments of tumour are of course pathognomonic, but are scarcely ever found.

Summary. In conclusion we may summarise the more important diagnostic points :—

(*a*) Rapid emaciation and cachexia in persons over 40.

(*b*) Vomited material which contains altered blood (coffee-grounds).

(*c*) The presence of blood in the fæces.

(*d*) Absence of free HCl, with the presence of lactic acid and the Boas-Oppler bacillus after a Riegel's test meal.

(*e*) Palpable tumour.

The differential diagnosis of gastric carcinoma must be made from :—

(*a*) Gastric Ulcer (*vide* p. 342).

(*b*) Chronic Gastritis (*vide* p. 338).

(*c*) Pernicious Anæmia (*vide* p. 163).

(*d*) Nervous Achylia (*vide* p. 349).

E. Disorders of Secretion. (i.) *Achylia.* As mentioned when discussing atrophic gastritis, achylia may be an accompaniment of this condition; it may also be a manifestation of coincident gastric carcinoma. In both these types the symptoms of the primary disorder are present and the achylia is a purely secondary affair.

There is, however, a form of achylia which occurs simply as a manifestation of nervous instability; it may be persistent or paroxysmal or may terminate spontaneously without obvious reason. Only adults are affected.

The symptoms of achylia itself are variable; they may be very slight in those cases in which the motility of the stomach is unimpaired and the food is passed rapidly into the intestine. Where the motility of the stomach is impaired there may be a sense of heaviness and distension in the epigastrium soon after taking food; vomiting is uncommon, but when present the food is returned practically as it was swallowed, except for some starch digestion by the ptyalin of the saliva.

The diagnosis is readily made by a test meal which shows complete absence of HCl and also of pepsin and rennin.

(ii.) *Hypersecretion.* This condition is of the nature of an over-active reflex action. It is common in cases of gastric ulcer, and probably does not occur apart from hyperacidity unless as a gastric crisis of tabes.

Two forms are described—first, continuous hypersecretion (Teichmann's disease, or gastro-succorrhœa), and secondly, paroxysmal hypersecretion (Rosbach's disease, or gastroxynsis).

As a general rule hypersecretion is a symptom only, and the objective features are really those of hyperacidity (*vide* p. 350).

The diagnosis of hypersecretion can be made as follows:—Wash the stomach empty over night and give a *test breakfast* at 7 a.m. the next morning. Remove the breakfast in forty minutes. If more than 200 c.c. are returned there is hypersecretion, *provided that* the stomach motility is normal and that there is no pyloric obstruction.

In all cases of paroxysmal gastroxynsis it is of the first

importance to exclude tabes dorsalis ; if this can be done gastric ulcer with pyloric spasm may be suspected.

(iii.) *Hyperacidity*—that is to say, more than 2 per cent. of acid in the gastric juice during digestion. Continuous hyperacidity is practically equivalent to continuous hypersecretion.

Hyperacidity is in itself very suggestive of simple gastric ulcer.

The symptoms are—a sense of weight in the epigastrium, heartburn, the eructation of acid mouthfuls (pyrosis), and sometimes the vomiting of an acid tasting material. The distressing symptoms usually appear about two hours after a meal, are relieved by food (if of a proteid nature), and are *promptly removed by alkalies*. The tongue is clean.

There is not often any doubt as to the presence of hyperacidity; but an absolute diagnosis can be made from the high acidity figures in the gastric contents either when vomited or after a test meal. It is well to consider the possibility of gastric ulcer in any person suffering from persistent hyperacidity, though it is fairly frequently found as an apparently simple neurosis. Probably the gastric mucous membrane of such individuals is abnormally sensitive to reflex stimulation.

(iv.) *Hypo-acidity* is not likely *per se* to cause symptoms, but when discovered by a test meal analysis it is an important sign of—chronic gastritis, atrophic gastritis, and carcinoma of the stomach.

The *repeated complete absence of* HCl is only likely to be found in malignant disease and achylia (whether nervous or symptomatic). It may, however, also be met with in pernicious anæmia.

F. Gastric Neuroses. (i.) *Anorexia Nervosa*. More properly this should be considered as a manifestation of hysteria or neurasthenia, since there is no real pathological condition of the stomach or its juices except in, and then rarely, achylia.

The complaint is practically limited to the female sex and is most common between the ages of 20 and 30.

There is complete lack of all appetite, and the sight or thought of any form of food is absolutely repulsive. A

constant feature is the immediate vomiting (practically regurgitation) of any food that is taken. Starvation ensues and most extreme emaciation may be met with. The absence of any discoverable organic disease should suggest the diagnosis.

The vomiting of pregnancy and of œsophageal obstruction must be excluded (*vide* also p. 321).

(ii.) *Bulimia* (Excessive Appetite). What constitutes bulimia must depend largely on the individual and his mode of life, but a sudden change in the direction of greatly-increased appetite without a corresponding increase in the amount of exercise should arouse suspicion.

This condition is most often met with in diabetes mellitus, but may also occur in Graves' disease and certain cerebral lesions, of which perhaps the most usual is epilepsy. It may also occur as a pure neurosis.

(iii.) *Pica*. The craving for unusual articles of diet or even for filthy material is met with in hysteria, in children, in imbeciles, and in pregnant women, though in the latter it does not often assume any more extreme degree than a desire for pickles, acids, etc.

In children it is often the expression of a need for some material which is absent from their allotted diet or present in insufficient amount to fulfil their metabolic requirements.

VI. THE INTESTINES.

A. **Examination of the Fæces.** A prolonged or elaborate examination of the fæces is beyond the scope of the practitioner; further, the information gained by such procedure is but rarely of sufficient value to justify the investigation.

In obscure cases a correct diagnosis may sometimes be indicated by this means alone, but as a routine method it is unnecessary.

The following points, however, are important and may be elicited without trouble or difficulty :—

(*a*) The gross character of the motions, including weight, size, frequency, form, consistence, and colour;

also the presence of blood or mucus, as well as parasites, foreign bodies, calculi, or obvious undigested food.

(*b*) The microscopical examination of portions of the fæces may show certain parasites or their eggs, muscle fibres from undigested meat, starch granules, pus cells and red blood cells, and fat.

For purposes of investigation the fæces are collected in a covered vessel ; if they are too solid they are diluted with water and they are examined by passing them through a wire sieve. Selected portions can be examined as ordinary wet preparations with the microscope.

The natural brown colour of the fæces is largely due to hydrobilirubin (reduced bilirubin) ; if this is absent obstruction to the biliary apparatus is probable.

In health about 200 grms. of fæces are passed daily ; of this nearly one-third by weight is composed of bacteria—the remainder consists of undigested cellulose, salts, and small amounts of intestinal epithelium.

The presence of muscle fibres, starch granules, and much fat indicates impairment of digestive processes or a considerable increase in the motility of the intestines.

The presence of blood in the fæces is always pathological. If it comes from the rectum or descending colon, it is bright red, and, especially if from the rectum, is liable to coat the outside of otherwise natural stools. If from the small intestine, it is dark in colour and intimately mixed with the fæces, unless the hæmorrhage is very profuse (*e.g.*, some cases of enteric fever). The higher up the source of the bleeding the darker do the motions become. The passage of blood and slimy mucus without any fæcal matter is almost pathognomonic of either intussusception or volvulus of the large gut.

Any considerable amount of blood in the fæces is obvious to the naked eye ; small traces can only be detected by the occult blood test (*vide* p. 331). For this test the patient should be on simple diet, since meat and even fish and green vegetables give a positive reaction.

Mucus in obvious amount shows catarrh of the intestine—of the small gut if mixed with the stool and of the colon if the fæces are coated with it.

Pus is rarely found except in a lesion of the rectum or colon.

Bacilli. The chief organisms are the B. coli group ; they are mostly Gram-negative ; it is noteworthy that in certain diseases associated with metabolic disturbance (*e.g.*, cœliac disease) Gram-positive bacilli predominate.

The bacillus typhosus, the cholera vibrio and Shiga's bacillus of dysentery can all be demonstrated in the stools of patients suffering from these diseases.

The amœba of dysentery can also be found in cases of this type of dysentery.

Parasites. A large number of parasites have been described. The common ones are tape-worm, round-worm, and thread-worm. The ova of various parasites can only be detected microscopically and need an expert knowledge for their differentiation.

An egg which it is important to recognise, since its presence is associated with intestinal hæmorrhage, is that of the Bilharzia hæmatobium. Although this parasite is not endemic in England, many English people visit South Africa and Egypt and may there acquire the disease.

The worm usually attacks the bladder, but sometimes is found in the intestine. The eggs can easily be recognised ; they are oval in shape, about ·16 mm. in diameter, and show a lateral spine. When the eggs are in the bladder the spine is situated terminally (*vide* Fig. 34).

B. **Diarrhœa.** This usually indicates enteritis, whether of the small or large gut. Irritant matter may be swallowed, it may be elaborated in the intestines by the action of bacteria, or it may be manufactured elsewhere and excreted into the intestine. Again, certain infections such as dysentery and enteric fever cause local lesions in the walls of the intestine, and the irritation of fæcal matter passing over these excites peristalsis and so produces diarrhœa.

Diarrhœa may also be produced as a compensation for diminished activity of other organs, as in kidney and liver disease ; under such circumstances many toxins are eliminated in the diarrhœic motions which would otherwise prove harmful to the general system. It is unwise to check too swiftly the diarrhœa which often accompanies cirrhosis of

the liver, since watery evacuations tend to relieve portal congestion.

Another cause of diarrhœa is disturbance of the proper nervous control; in these cases the diarrhœa is often paroxysmal (*e.g.*, the candidate for an examination or one about to run a race), but a large group of cases with neurasthenia have a tendency to chronic diarrhœa, which often takes the form of mucous colitis (*vide* p. 368).

When lardaceous disease affects the intestines there is severe diarrhœa, but the intestines are not affected till after the spleen, liver, and kidneys, so that there is ample evidence on which to form an accurate diagnosis.

Constipation as a cause of diarrhœa must not be forgotten: the presence of scybalous masses in the large gut may so irritate the mucous membrane as to initiate a spurious diarrhœa in which soft or watery motions are passed; but all the time the scybala which cause the trouble remain *in situ*. A series of large enemata will settle the diagnosis of such cases and cure them at the same time.

One of the first signs of new growth of the intestine or stomach may be diarrhœa (often alternating with constipation); hence the sudden onset of intermittent diarrhœa in a middle-aged or elderly person requires the most thorough investigation.

In itself diarrhœa is often beneficial, since it helps quickly to eliminate toxic matter from the system, but continued diarrhœa is most exhausting, and collapse (due to loss of fluid from the body) soon develops in the old, the debilitated, and in children.

The exact appearance of the evacuations is not often of any particular help in forming a diagnosis; in obscure cases examination of the fæces (as indicated on p. 351) may be of assistance. For the rest the following types of motion are found to be associated with more or less definite conditions:—

The pea-soup stool—with enteric fever or allied infections.

The rice-water stool—with Asiatic cholera.

Very frequent small stools consisting chiefly of blood and mucus and passed with great pain—with dysentery.

Small stools coated with mucus—with mucous colitis.

Foul green fluid stools looking like chopped parsley and often flecked with blood—with epidemic enteritis of infancy.

Bulky pale porridge-like stools—with cœliac disease.

C. **Constipation, Intestinal Stasis, and Intestinal Obstruction.** The bowels of an ordinary healthy person should be opened once every day. Many persons habitually have two motions daily, and three is by no means unusual.

On the other hand, certain people only have their bowels opened every second or even every third day, and yet they appear to enjoy perfect health. A few cases are recorded when only one motion weekly has not produced any untoward symptoms.

It is obvious that the term "constipation," like diarrhœa, must only be used after due consideration of individual peculiarities.

At the same time it is probable that those cases in which perfect health continues in spite of infrequent evacuations of the rectum are examples of the adaptability of the particular individual to unnatural circumstances rather than evidence of the advisability of the procedure.

The normal stool is formed but soft; any undue hardness indicates constipation, because the consistence varies with the amount of water present, and if too much water has been abstracted it is evident that the fæces have delayed too long in the large intestine.

A very constipated motion consists of extremely hard, dark-coloured rounded masses, often about the size and shape of a walnut.

As mentioned in the previous section, constipation may be present even while there is apparent diarrhœa, since the scybala may not be passed themselves even though they irritate the bowel sufficiently to promote a flow of watery mucus which is passed with a certain proportion of fæcal matter.

Under ideal conditions the rectum should always be empty; the moment any fæces enter it the rectal reflex is aroused and there is a call to stool: if the call is obeyed, the colon as far as the splenic flexure or higher is emptied. Such calls should never be neglected. Unfortunately the

artificial existences that we are compelled to lead often prevent us from obeying the call to stool; as a result the reflex becomes dulled and inactive, the fæces stagnate in the rectum, they lose water and become hard, and in this manner the most common form of constipation is developed.

The proper way to prevent this type of constipation is to encourage the bowels to act at the same hour every day and to let nothing interfere with this daily rite from the earliest days of childhood.

Another common form of constipation is due to intestinal stasis, especially in the cæcum, ascending and transverse colon.

The delay in certain instances may be extreme (three, four, or more days) and yet the sufferer may indignantly deny constipation because his bowels are open every day—so indeed they may be, but the daily motions do not even begin to empty the large gut as it should be emptied and a progressive accumulation of fæces is the result.

The cause of this form of constipation is, usually, atony of the muscular coats of the bowel. Not infrequently in these cases there is hyper-motility of the stomach and small gut, so that the chyme passes much too rapidly into the cæcum and is delayed there and in the ascending colon, possibly to permit of the completion vicariously of certain digestive processes which should already have been finished in the ileum. This delay is achieved in part by exaggerated reverse peristaltic waves in the descending colon, and so large gut stasis is initiated. This may terminate in pronounced atony of the muscle wall and is attended by the bacterial elaboration of those toxins, which, when absorbed into the system, may produce such diverse clinical conditions as pernicious anæmia, rheumatoid arthritis, pyelitis, or even septicæmia should the bacilli themselves reach the blood-stream.

Patients who suffer from intestinal stasis of this type often present the symptoms of abdominal neurasthenia—that is to say, their attention is focussed on their abdomens; and they accentuate their vague dyspeptic troubles as well as manifesting the more ordinary mental and physical signs. At the same time there is generally evidence of auto-intoxication, as shown by gastric derangement, furred

tongue, cold extremities, pigmentation of the skin, headaches, easy fatigue, and, not infrequently, persistent slight fever.

In investigating such a case it is important to remember that there may well be an organic cause at the bottom of the syndrome, and it is especially necessary to exclude such conditions as chronic or latent appendicitis, ileo-cæcal or ileo-colic bands, Jacksonian membranes and gastro-colic or gastro-hepatic adhesions, as well as the more ordinary abdominal diseases, such as duodenal ulcer or gall-stones and pelvic conditions in women.

Careful attention to the history of the case, which should go back as far as possible, combined with a detailed physical examination, will often suggest such conditions as appendicitis, duodenal ulcer, gall-stones and pelvic disease.

To establish the Diagnosis of intestinal stasis and to suggest the presence of adhesions it is important to have a series of X-ray photographs taken after a bismuth meal.

The progress of the bismuth can be watched with a screen from the time it enters the stomach until it leaves the sigmoid, and plates can be taken at appropriate intervals. The site of delay can be demonstrated, and any kink or band can often be accurately localised.

Partial Obstruction in a more definite sense than is implied in the previous paragraphs necessarily produces constipation sooner or later; the motions become increasingly scanty and are passed at infrequent intervals, though there may be occasional attacks of spurious diarrhœa. If the obstruction is in the rectum or sigmoid, the motions often assume a narrowed calibre (pipe-stem) and may be streaked with blood. A spastic condition of the colon from irritation also gives rise to pipe-stem motions, so that their absolute diagnostic value is restricted.

If the obstruction is higher up blood may be more intimately mixed with the fæcal matter and of darker colour, while the narrowing of the fæces may not be apparent when they are passed, though necessarily there is narrowing as the actual obstruction is passed; this may be demonstrated by the X-rays. Such obstruction may result from new growth, bands, adhesions, enlarged glands, simple stricture, or impacted fæces.

New growth may be in the gut wall, or the gut may be pressed on by tumours in adjacent structures.

The only symptoms of such a condition of partial obstruction as the above may be a certain amount of flatulent distension with general abdominal uneasiness, or the patient may complain of his constipation or that he passes blood.

Malignant disease may give signs of cachexia before any obstruction is noted, but in intestinal cancer cachexia is often long delayed. In investigating such a case the appearance of the patient and a detailed history are most important, as is careful palpation of the abdomen, noting the presence or absence of distension or visible peristalsis, and last, and by no means least, an examination of the rectum. If necessary the sigmoid can be inspected with a sigmoidoscope.

A tumour anywhere in the colon may be fæcal in nature and should not lead too quickly to a diagnosis of new growth. Fæces can often be felt to pit on pressure. A series of enemata will settle the question by causing such a mass to disappear.

Complete Obstruction, as by new growth, bands, kinks, hernia, etc., is, of course, accompanied by *Absolute Constipation* and presents a sufficiently striking clinical picture.

In this connection it is essential to remember that a patient (probably a woman) may have no evacuation of the bowels from constipation for two or three weeks and yet there will be no urgent symptoms at all; but mechanical obstruction of the gut lumen with interruption of the blood supply invariably causes acute symptoms within a few hours. There may have been chronic obstruction for months with no acute symptoms, but the moment the obstruction is made absolute the patient's condition becomes urgent.

The outstanding features of acute obstruction, as it is called, are :—

(i.) *Abdominal Pain*, at first colicky and paroxysmal, but soon constant though still liable to exacerbations. The maximum intensity is usually umbilical or over the actual obstruction.

(ii.) *Distension*. This develops rapidly, but is limited to the parts above the obstruction, so that if the obstruction is in the duodenum only the stomach itself is distended.

The distension progresses from below upwards ; in rectal obstruction the colon becomes distended first and may be visible as a horse-shoe surrounding the belly. Often, however, there is a paralytic ballooning of the rectum *below* a rectal or sigmoid obstruction.

Central distension is usual in small gut obstruction. The site of the obstruction may often be localised with considerable accuracy by noting the type of the distension.

(iii.) *Vomiting.* This is persistent until the obstruction is relieved ; at first the vomited matter consists of stomach contents, then bilious material, and lastly stercoraceous matter. The lower the obstruction the longer is the vomiting delayed, though it is usually a prominent feature when once it has appeared. The higher the obstruction the quicker does the vomiting appear and the sooner does it assume a fæcal character.

(iv.) *Obstruction.* This is absolute : flatus even does not pass. It must be remembered, however, that the contents of the bowel below the obstruction may be evacuated naturally after the obstruction has occurred, and will certainly be returned if an enema be given.

(v.) *Peristalsis.* This is often seen in the distended coils of gut before they have become paralysed. Reverse peristalsis is fairly common, and is even more pathognomonic of obstruction than the ordinary variety.

(vi.) *Collapse.* Cold, damp skin, pinched face, small, quick pulse, subnormal temperature, and dry tongue.

The diagnosis of the site of obstruction has been outlined in the preceding remarks ; the cause of the obstruction may be impossible to determine : nevertheless an effort should be made despite the fact that immediate operation is the only treatment for acute obstruction.

In babies acute obstruction is most frequently due to intussusception (*vide* p. 365), in children to tuberculous glands in the abdomen. The presence of a hernia at any age would suggest strangulation.

A history of old tuberculous peritonitis or of appendicular or of pelvic disease would suggest adhesions from these causes. In persons who are more than 40 years old malignant disease becomes increasingly probable, and since

the rectum and sigmoid are the most common situations, examination of these structures often leads to an accurate diagnosis.

An impacted gall-stone would scarcely happen without a history of biliary colic and probably jaundice.

D. **Intestinal Colic.** This is produced by painful and irregular contractions of the bowel (disordered peristalsis).

Colic is only a symptom and great care must be exercised in its interpretation. It may be met with under the following circumstances :—

(*a*) The ingestion of irritating foodstuffs.

(*b*) Lead poisoning and most forms of irritant poisoning.

(*c*) In any acute inflammatory condition of the bowels, such as appendicitis.

(*d*) In intestinal obstruction, from whatever cause.

(*e*) After certain purgatives.

(*f*) In peritonitis.

Diagnosis. The presence of diarrhœa or constipation accompanying the colic is of importance, as diarrhœa excludes acute obstruction and is rather against acute inflammatory processes. Lead poisoning is accompanied by constipation, but should give its own special signs. A normal pulse and temperature, and an absence of all shock as well as a normal white cell count, are in favour of simple colic. The history of what has recently been eaten is naturally of great importance, but may be very misleading.

In distinguishing between simple colic and "the acute abdomen" stress may be laid on two features, namely, the attitude of the patient and his pulse. In simple colic the patient writhes and wriggles as the spasms seize him : in the acute abdomen he lies *very still on his back* and often has one or both legs drawn up. In colic the pulse is quiet and slow, except during the paroxysms : in the acute abdomen the pulse generally gets increasingly rapid and wiry. Further, in simple colic the abdomen is quite soft between the spasms.

The characteristic situation and distribution of the pain in renal and biliary colic (*vide* p. 397 and p. 456) should prevent these being mistaken for simple intestinal colic.

E. **Affections of the Duodenum.** (i.) Catarrhal Duo-

DENITIS. This is likely to occur whenever there is gastritis, and its symptoms may be masked by those of the latter. Bile-stained vomit indicates that the inflammation has passed through the pylorus.

Duodenal inflammation is of special interest, since it is probable that it is a necessary precursor to any ascending infection of the bile passages. Gall-stones are produced in this manner. Catarrhal jaundice may possibly result from the blocking of the opening of the ampulla of Vater by the excessive mucus produced by duodenal catarrh (*vide* also p. 383).

(ii.) DUODENAL ULCER. The pathology of the peptic duodenal ulcer is, in the main, similar to that of gastric ulcer. Men are very much more frequently affected than women, and the ordinary age incidence is 25 to 50 years.

Perforation occurs relatively more frequently than in gastric ulcer, and is attended by a much higher mortality even when an operation is undertaken without delay.

The Symptoms and Signs of duodenal ulcer are :—

(*a*) *General slight dyspeptic symptoms.* These are attributable to the hyperchlorhydria which seems to be constantly present.

(*b*) *Epigastric Pain.* This occurs in bouts, is most usual three to four hours after a meal, is promptly relieved by food and gradually by alkalies, and includes the right hypochondrium more than gastric ulcer does.

Sometimes there are both pain and tenderness over the right lower ribs behind.

(*c*) *Tenderness.* This is characteristically situated over a small area to the right of the middle line ; the maximum intensity is often at the junction of the lower and middle thirds of a line joining the umbilicus with the tip of the ninth rib.

(*d*) *Cutaneous Hyperæsthesia* is sometimes present over the same area as the tenderness.

(*e*) *Rigidity.* There may be more or less rigidity of the upper segment of the right rectus abdominis muscle.

(*f*) *Vomiting.* Unless the ulcer is just by the pyloric ridge, vomiting is unusual.

The same applies to hæmatemesis.

(*g*) *Melæna.* The passage of dark blood per rectum

intimately mixed with the fæces, if no other cause can be made out, is strongly suggestive of duodenal ulcer.

The hæmorrhage may be very slight and may require the occult blood test (*vide* p. 331) for its recognition. On the other hand, very grave anæmia may be caused in this manner.

(*h*) In nearly every case a test meal gives high acid values, even higher than in gastric ulcer.

Duodenal ulcer must be distinguished from :—

(1) *Gastric Ulcer* (*vide* p. 343).

(2) *Chronic Gastritis* is suggested by morning sickness, furred tongue, much mucus after gastric lavage, flatulency, lack of localised tenderness, severe pain or melæna, and the fact that food does not markedly relieve the distress.

(3) *Gall-Stones.* Biliary colic is usually more severe than the pain of duodenal ulcer; it is definitely situated in the right hypochondrium and goes through to the right shoulder-blade. The pain is definitely paroxysmal, and though exacerbations may occur about three to four hours after food, they do not constantly do so and they are not relieved by alkalies or by food. The abdominal tenderness is higher up towards the point of the ninth rib. Jaundice is frequent, and there is no melæna.

(4) *Appendicitis.* There is no hunger pain, the lower rectus is rigid, the pain is usually traceable down to the iliac fossa even in those cases in which its maximum intensity is above the umbilical level. There is no melæna.

(iii.) Other Ulcerative Conditions of the Duodenum. Duodenal ulcers may be found in the following conditions :—

(1) *Bright's Disease.* Examination of the renal system may suggest this cause for melæna. In albuminuric ulceration of the intestine the duodenum is the most common site.

(2) *Leukæmia, Pernicious Anæmia,* and *Purpura* do not cause ulceration in the duodenum more frequently than in the rest of the small gut, and not so frequently as in the large intestine.

(3) *Gall-Stones.* These may ulcerate into the duodenum.

(4) *Carcinoma.* This may develop in a chronic peptic ulcer, or it may spread from a primary growth of the head of the pancreas, of the common bileduct, or of the gall-bladder.

(5) *After Extensive Burns.* Duodenal ulcers have been recorded. Examination of post-mortem records has convinced us that this cause must be extraordinarily rare nowadays, though probably when burns were kept less aseptic the proportion was higher.

F. **Certain Special Forms of Intestinal Disease.** (i.) *Acute Enteritis.* This is usually produced by some indiscretion of diet, especially by eating tainted food. The stomach is often involved also, and then the term "gastro-enteritis" is more accurate.

Apart from food, acute enteritis is produced by many poisons, especially arsenic and antimony, and the beneficial action of a large number of purgatives is the direct result of the mild enteritis caused by the drug.

Debilitated individuals are prone to attacks of enteritis from causes which have no effect on healthy persons.

Certain diseases—*e.g.*, exophthalmic goitre and Bright's disease—are liable to be complicated by severe enteritis.

Some apparently healthy persons seem to possess idiosyncrasies for certain articles of food, and when they eat such things always develop enteritis. These cases frequently have urticaria also; possibly, therefore, this type of enteritis is due to urticaria on the bowel wall.

The severity of ordinary enteritis such as results in healthy persons from unsuitable food varies very much with the age of the patient. The attack is always of more serious import in young children than in older persons.

Enteritis which results from tainted food may be of a relatively simple form, or it may be due to certain alkaloidal products of bacterial action known as ptomaines. Some fish, for example mussels, develop ptomaines physiologically at certain seasons of the year. The enteritis caused by ptomaine poisoning is always severe, and the prognosis seems to depend on three factors:—

(*a*) The amount and nature of the ptomaine taken.

(*b*) The question of whether vomiting occurs early or

late, and so of whether most of the poisons are expelled before absorption.

(*c*) The personal equation, which appears to be a thing quite apart from our understanding, since two quite healthy people may eat the same amounts of the same tainted food and one may die while the other may suffer only the most trifling inconvenience. In fatal cases the onset of symptoms is usually delayed for about thirty-six hours after taking the food.

The symptoms of simple acute enteritis vary very much in their intensity : in ordinary cases colicky pains in the abdomen accompanied by diarrhœa are all that are to be observed. If gastritis co-exists there will be vomiting as well.

The motions vary somewhat according as to whether the large or small intestine is chiefly affected. In a small-gut enteritis there is often partly-digested food ; in a large-gut affection there is much slimy mucus. In both cases the motions become increasingly watery as the disease advances.

When all the irritating material has been expelled the symptoms gradually subside.

In more acute cases the diarrhœa and sickness are so severe that collapse quickly ensues, with pinched features, rapid, feeble pulse, and subnormal temperature.

Such cases are particularly likely to occur in hot weather, and are perhaps due to a bacillary infection (Cholera Nostras). Recovery when the intoxication has run its course is just as rapid as was the collapse at the onset.

In true ptomaine poisoning severe symptoms are commonly delayed for at least twenty-four to thirty-six hours.

(ii.) *Epidemic Enteritis of Infants.* As stated in the previous section, simple acute enteritis due to improper feeding is a much more severe affection in young children than in adults.

Epidemic Enteritis is, however, quite a different matter ; it is almost certainly a specific infective disease, though quite possibly more than one individual organism may be capable of producing it. The intestinal flora is so diverse, that hitherto these organisms have not been identified with precision.

The determining factors in the production of epidemic

enteritis are—hot weather, flies, dust, and the contamination of milk and feeding-bottles. When once started the infectivity is great, and even in hospital wards, where the utmost care is exercised, the disease often spreads from bed to bed with alarming rapidity.

In London the disease seems to be diminishing steadily; there has been no bad epidemic since 1906 and 1907. This may perhaps be explained as follows:—There have not been very hot summers of late, the science of infant feeding among the poor has made great strides, and lastly, with the enormous increase of motor traffic and consequent decline in *horse*-drawn vehicles, flies, dust, and road dirt have greatly diminished.

The patients are mostly infants who are being artificially fed; young children up to three and four may also be affected, but in them the infection is not so severe.

Two types of the disease may be recognised—the afebrile and the febrile. In either the child may die within twenty-four hours of the first symptom, but, on the whole, the febrile form with a temperature of 103° to 105° is even more quickly fatal than the other.

The symptoms are diarrhœa with frequent foul, green, watery stools, and vomiting. Blood may often be present in the motions. Infants cannot stand the loss of much fluid; they quickly become shrunken and collapsed and die from exhaustion. All grades of the disease are met with, and in the more chronic forms the outlook is more hopeful.

The diagnosis presents no difficulty; the passage of slimy motions streaked with blood may suggest intussusception, but in the latter there is no fæcal matter and a tumour can be felt in the abdomen.

(iii.) *Acute Intussusception*, or invagination of part of the intestine into itself, occurs principally in children from six months to two years old.

The ordinary form of intussusception is the ileo-colic, but sometimes ileo-cæcal or cæco-colic forms may occur.

The symptoms are those of acute intestinal obstruction; paroxysmal abdominal pain, possibly with vomiting, and the speedy disappearance of all fæcal matter from the motions, which soon consist entirely of blood and slimy mucus. In

eighteen to twenty-four hours distension develops and vomiting becomes more frequent. In the early stages shock, though it is apparent to the careful observer, need not be marked. The intussuscepted portion of gut can nearly always be felt as a tumour (frequently sausage-shaped) within the abdomen; sometimes it has travelled so far round the colon that it can be felt per rectum, or it may even present at this orifice.

Most often the tumour is situated in the right half of the abdomen; occasionally it gets tucked up under the liver, and is then hard to feel.

We believe that in all cases the tumour can be felt if the abdominal walls are relaxed; if they are rigid an anæsthetic must be given.

The appreciation of the fact that an abdominal mass can be palpated is of the first importance, for it sometimes happens that the passage of blood is delayed for twelve or even eighteen hours, and every hour is of value if the child's life is to be saved. At least 80 per cent. of the patients recover if they are operated on within eighteen to twenty hours.

Another point of diagnostic value is the fact that babies who get intussusception appear to be unusually healthy and robust. It is more common in boys than in girls.

Intussusception must not be mistaken for :—

Enteritis (*vide* p. 365).

Henoch's Purpura (*vide* p. 166).

(iv.) *Cœliac Disease.* This disease develops in children between the ages of one and three years. The principal sign is the passage of numerous bulky, offensive, pale-coloured motions, which are often likened to porridge by the parents. The patients waste steadily and show great muscular weakness, and yet no organic lesion can be discovered. Tetany is an occasional complication, and infantilism is sometimes a sequel in severe cases. The course tends to be chronic and relapses are common.

The disease appears to result from an inability properly to digest fats and starches; in some cases (but not all) there is a notable excess of unaltered fat in the fæces.

The chief difficulty in the diagnosis of cœliac disease is

to eliminate tuberculous peritonitis or tuberculosis of the mesenteric glands.

The character of the motions, as described above, is the most important feature.

The appearance of the abdomen is also important, for there are neither ascites, masses of omentum, nor palpable glands.

The special tests for the diagnosis of tuberculosis are of value in these cases.

The therapeutic test is also of value, for the benefit shown by many cases of cœliac disease from a diet which consists almost entirely of raw minced meat or raw meat juice is striking.

(v.) *Hirschsprung's Disease* (Idiopathic Dilatation and Hypertrophy of the Colon). This is a congenital affection of unknown causation. The principal features are obstinate constipation and a manifestly dilated colon. As in gross dilatation of other viscera, tetany is a not uncommon complication.

Many children with this disease show imperfect development, or even infantilism.

The most urgent danger is acute intestinal obstruction.

The diagnosis depends on the presence of obstinate constipation from birth, with a soft but enormously distended belly, in which the huge colon is usually very apparent. The percussion note over the colon is tympanitic, and peristalsis can frequently be observed.

(vi.) Such specific infective diseases of the intestines as enteric fever, tuberculous enteritis, dysentery, cholera, and the gastro-intestinal form of influenza are considered elsewhere.

(vii.) *Ulcerative Colitis.* It seems artificial and unnecessary to distinguish between chronic dysentery and ulcerative colitis, since pathologically the lesions are identical and primary ulcerative colitis is certainly an infective process even though it may not be due to any of the specific organisms known to be responsible for acute dysentery.

Ulcerative colitis may, however, occur as a manifestation or complication of certain diseases, of which the most important are Bright's disease, pernicious anæmia, and

leukæmia, or occasionally tuberculosis and septicæmia. Hence in investigating a case of ulcerative colitis it is first necessary to exclude the above diseases.

Diagnosis. The mere diagnosis of ulcerative colitis is usually obvious, since diarrhœa with tenesmus and the passage of red blood and slimy mucus must mean an ulcerative process in the large gut. At the same time there is progressive emaciation, colicky abdominal pain, and more or less constant tenderness over the colon, which is most marked in the sigmoid and cæcal regions. There may be severe toxæmia, and pyrexia is the rule.

A *careful* examination of the rectum and sigmoid with a finger and the sigmoidoscope will eliminate such conditions as carcinoma, fistula, or simple septic proctitis, and the sigmoidoscope (which must, however, be used with the utmost gentleness) will usually show definite ulcerated areas.

If as the result of all possible clinical investigations it seems certain that the case is one of primary ulcerative colitis, it is worth while endeavouring to isolate some organism from the fæces which may be responsible.

This proceeding is very uncertain, but plate cultures may show some organism (*e.g.*, the pneumococcus) which is normally absent, or some normal inhabitant may be present in excess. In the latter case the patient's opsonic index may be taken in respect of the suspected organisms and compared with that of a healthy person. A distinct difference in the readings would justify the use of an autogenous vaccine.

(viii.) *Mucous Colitis.* A condition characterised by an excessive secretion of mucus in the large intestine and the consequent passage of this in the motions. At the same time the large gut isi rritable and liable to spasm, so that painful contractions of the colon are common. Very rarely a little blood may be noticed in streaks on the mucus.

The etiology of mucous colitis is obscure ; in itself it can only be a symptom, and, since many sufferers from neurasthenia also have mucous colitis, some authorities regard it as a pure neurosis.

At the same time there is no doubt that in a large proportion of cases there is an organic foundation for the

condition, and, in investigating a case of mucous colitis every effort should be made to discover the organic lesion if one be present.

Mucous colitis is more common in women than in men. This can possibly be explained by the greater frequency of constipation in the female sex, for constipation either precedes or accompanies mucous colitis in a large majority of instances. Frequently there is habitual constipation with an attack of pseudo-diarrhœa every three or four days, during which much mucous is passed ; sometimes a mucinous cast of a portion of intestine can be recognised. Even during the periods of constipation the small hard scybala are often coated with mucus.

The patient is generally definitely hypochondriacal, and, because of the associated abdominal pain and discomfort, has often convinced herself that she is a martyr to dyspepsia and has curtailed her diet so greatly as to impair her nutrition.

Test meals usually show a perfectly healthy gastric secretion. Associated conditions may be a movable kidney or general visceroptosis, and not infrequently a colon bacilluria.

The most important organic lesion which may produce mucous colitis is chronic appendicitis with or without adhesions ; next to this adhesions about the hepatic or splenic flexures of the colon, round the pelvic colon, or across the cæco-colic junction. The precise manner in which these conditions cause mucous colitis is uncertain ; possibly the explanation is partly that they favour constipation and intestinal stasis and partly that by interfering with the proper peristaltic movements of the colon this viscus is rendered irritable and so responds abnormally to the nervous stimuli it receives, the visible result of which is excess of mucus.

The diagnosis of bands and adhesions has been considered briefly on p. 357; the diagnosis of chronic appendicitis depends largely on the history of acute or subacute attacks (possibly after an interval of many years) and the presence of deep tenderness in the right iliac fossa, not necessarily constant. Further, in chronic appendicitis of this type

reflex gastric disturbance (such as vomiting or other dyspeptic phenomena) is often present.

No case of mucous colitis should be labelled as simple or neurotic till every effort has been made to exclude definitely these possible organic causes.

VII. THE PERITONEUM.

A. **Ascites.** The presence of fluid in the peritoneal cavity is only a symptom of some underlying disease.

Peritoneal effusions may be serous or purulent, but the term " ascites " is restricted to the simple variety, because a purulent effusion gives rise to a totally different clinical picture with the signs and symptoms of acute peritonitis and requires immediate surgical interference. At the same time it is possible that in the majority of cases of ascites there is a certain amount of chronic peritonitis as well, since it is difficult to produce ascites experimentally in animals by simple obstruction to the venous return from the portal system.

The more common diseases in which ascites is found are :—

(i.) Myocardial insufficiency of the right heart, whether due to cardiac fibrosis, left-sided valvular disease, or to chronic pulmonary disease.

(ii.) Acute or chronic parenchymatous nephritis.

(iii.) Cirrhosis of the liver (portal type).

(iv.) Perihepatitis.

(v.) Tuberculosis of the peritoneum.

(vi.) Diffuse carcinomatosis or sarcomatosis of the peritoneum.

(vii.) New growth obstructing the portal vein (ascites not inevitable).

(viii.) Thrombosis of the portal vein (ascites not inevitable).

(ix.) Rupture of an ovarian cyst (especially the proliferating papillary ovarian cyst).

Sometimes the fluid instead of being serous in appearance is milky or opalescent : the milkiness has sometimes been shown to be due to intraperitoneal rupture of the recepta-

culum chyli or the lymphatic duct and so to depend on the presence of true fat globules (chylous ascites), but in other cases it is due to certain lipoids the origin of which is obscure.

Filaria sanguinis hominum may cause chylous ascites.

The fluid may be free in the peritoneum or occasionally it may be encysted or loculated.

Loculated ascites suggests tuberculosis or malignant disease, but sometimes occurs as the result of frequent "tappings."

The positive diagnosis of free fluid in the peritoneum is usually easy and is based on the following signs :—

(i.) Increasing abdominal size with a maximum girth at or about the level of the umbilicus ; in extreme cases the umbilicus is flattened out from within.

(ii.) An area of dulness to percussion which shifts with changes of posture. When the patient is half sitting up there is a horse-shoe of dulness over the abdomen with the horns of the horse-shoe pointing up in the flanks. This dulness above the pubes and each flank is important, and if the patient now turns on to one side, the flank which is raised off the bed becomes resonant, while there is a *deeper band of dulness than before* in the other flank.

(iii.) A fluid thrill can be felt if the flat of the fingers be laid on one dull flank and the opposite dull flank be sharply tapped with the fingers of the other hand. In performing this test the vibrations of the abdominal wall and intraperitoneal fat should be cut off by pressure in the mid-line of the patient's abdomen with the ulnar border of an assistant's hand. It is said that there may be too much fluid sometimes for a thrill to be perceived ; we have not seen such a case.

(iv.) The presence of a central or upper central area of resonance, due to the floating upwards of the gas-containing intestine.

(v.) The sensation of displacing fluid if sharp dipping movements are made with the fingers on to the liver.

A careful observer can hardly mistake free fluid for any other condition. The following possible sources of error may be mentioned, though they are more applicable to encysted ascites :—

(i.) *A Full Bladder.* This causes a central dull area, and micturition or the passage of a catheter will prevent mistake.

(ii.) *Ovarian Cyst.* The dull area is central or a little to one side of the mid-line. The flanks are resonant; the maximum girth is below the umbilicus. A pelvic examination may assist; but in so far as an ovarian cyst will rise out of the pelvis as it grows in size a pelvic examination may be negative, and under these circumstances an ovarian cyst may be clinically indistinguishable from *encysted ascites*, though it should not be mistaken for *free fluid*. Further, it must be remembered that an ovarian cyst may rupture, so that there may be *free fluid* plus *ovarian cyst*, and this possibility should always be borne in mind where the fluid is present in a young woman who shows no primary cardiac or renal trouble and in whom the history does not favour tuberculous peritonitis.

(iii.) A hydatid cyst may simulate ascites; a history of a sojourn in Australia or close association with dogs is suggestive. The characteristic evidence of free fluid is not present, and in most cases the effusion may be connected directly with an enlarged liver. The fluid from a hydatid cyst is clear, free from albumin, and of low specific gravity (1,010); it may contain sugar and chlorides and the characteristic hooklets should be demonstrable.

A blood examination shows definite eosinophilia.

The signs of loculated ascites are variable and depend very much on the size of the effusion. There is usually a constant dull area somewhere or other, most often in the lower half of the abdomen; frequently a thrill may be present over the dull patch. The size and shape of the dull area does not alter, or only alters very slightly, with change of posture.

A single loculus such as described above is very rare, and, if an ovarian cyst can be excluded, is nearly always due to tuberculous peritonitis, signs of which (such as rolls of doughy omentum) may be found as well. More often loculated ascites is formed at the same time as a collection of free fluid, and the loculation is only suspected when peracentesis fails to empty the abdomen.

The differential diagnosis of the cause of any case of ascites involves a detailed consideration of the symptoms and signs of the list of diseases on p. 370, but may be discussed briefly here.

Ascites may be divided into two groups, depending on whether it is caused *mainly* through obstruction to the portal venous system or *mainly* through some peritoneal affection.

The first group (obstructive) comprises :—

(i.) *Valvular and Muscular Heart Lesions.* These give their own conclusive signs, and ascites, in such conditions, is usually part of a general anasarca. Mitral stenosis, however, often causes ascites without subcutaneous œdema.

(ii.) *Large Pericardial Effusions.* Acute pericarditis, calcareous pericarditis, adherent pericardium, and chronic mediastinitis are all likely to be associated with ascites from embarrassment of the heart's action either toxic or mechanical.

The signs of pericardial or mediastinal disease are generally apparent and will indicate the cause of the ascites.

(iii.) *Thrombosis of the Portal Vein* (*vide* also p. 386) causes ascites, enlargement of the spleen, and hæmatemesis and is likely to be mistaken for cirrhosis, but may be suspected if the above signs develop suddenly in a patient with some debilitating disease which is likely to be complicated by venous thrombosis.

(iv.) *Cirrhosis of the Liver* (*vide* also p. 392). The patient may give a history of chronic gastritis and may show signs of chronic alcoholism (*vide* p. 201).

The liver may be increased in size, and the surface may feel nodular to palpation. The age is suggestive (30 to 50 years).

The second group (principally peritoneal) comprises :—

(i.) *Renal Disease.* This may be diagnosed by urinary examination.

(ii.) *Perihepatitis.* This may give no other sign than recurrent ascites ; if due to syphilis (as is often the case) the Wassermann reaction may help the diagnosis.

(iii.) *Tuberculous Peritonitis* has been discussed on p. 100.

(iv.) *Peritoneal New Growth.* There should be evidence of some primary focus (such as stomach or ovary). Cancerous cachexia is not long delayed. Intra-abdominal masses may be palpable, and secondary nodules in the skin round the umbilicus are not uncommon.

(v.) *New Growth Obstructing the Portal Vein* usually causes jaundice also from obstruction of the bileduct, and there are likely to be further signs referable to the growth itself, such as pyloric obstruction or pancreatic disturbance.

When paracentesis becomes necessary information may be gained from the fluid withdrawn. Ordinary ascitic fluid is a clear straw-coloured liquid with a specific gravity of 1,015; it contains albumin, but does not clot spontaneously.

The fluids removed from purely passive effusions and from renal disease or cirrhosis show a preponderance of endothelial cells. In malignant disease the fluid may be blood-stained, it contains endothelial cells, lymphocytes and polymorphonuclear leucocytes, while occasionally fragments of tumour may be demonstrated.

Lymphocytic effusions are most likely to be found in cases of tuberculous peritonitis.

The presence of cholesterin crystals indicates cyst formation, probably ovarian, possibly pancreatic.

The presence of a thick mucinous fluid that will not run even through a big cannula is pathognomonic of one variety of ovarian cyst (pseudo-mucinous).

B. **Peritonitis.** (*a*) Acute General Peritonitis. Apart from traumatic causes, acute generalised peritonitis is usually the result either of perforation of a hollow viscus or of acute inflammatory change in part of the intestinal canal (*e.g.*, the appendix or a strangulation) without necessarily perforation, for the coats of the intestine may be so damaged as to permit the passage of bacteria from the lumen of the gut—a condition found also at times in carcinoma. Other causes may be salpingitis (much more often this causes inflammation limited to the pelvic peritoneum), spread through the diaphragm of septic intra-thoracic

processes (*e.g.*, empyema), and rarely as a metastatic infection in pneumonia or septicæmia.

A subacute generalised peritonitis may be found in the terminal stages of any prolonged debilitating condition, especially Bright's disease.

The diagnosis of generalised peritonitis depends on the following signs and symptoms :—

(i.) *Abdominal pain :* sudden in onset, severe in degree, at first diffuse, then more localised to the umbilicus, and later still sometimes to the site of the lesion, in inflammatory as opposed to perforation cases.

(ii.) *Vomiting.* This usually occurs at the outset, but then often passes off until the inflammation has become very extensive, when it sets in again and persists with hiccups till the end : after a time the vomited matter becomes dark brown and has a fæcal odour (stercóraceous vomit).

(iii.) *Rigidity.* The muscles are held stiff ; this occurs at first over the lesion, but as the inflammation spreads so does the rigidity.

(iv.) *Immobility.* The respiration becomes shallow and of the thoracic type, the abdomen moving little or not at all.

(v.) *Attitude.* The patient lies on his back and does not willingly move at all ; one or both legs may be drawn up.

(vi.) *Pulse.* The pulse becomes small, rapid and hard (wiry).

(vii.) *Temperature.* In perforation of stomach or duodenum the temperature at first is often subnormal ; in appendicitis there is more often moderate pyrexia.

(viii.) *Distension.* Peritonitis is quickly followed by paralysis of the gut and great distension.

(ix.) *Tenderness.* There is usually well-marked diffuse tenderness all over the inflamed area.

(x.) *Facies.* The hippocratic face appears early and is really evidence of collapse ; it includes anxious expression, sunk eyes, sharp nose, and pinched appearance generally.

(xi.) *Constipation.* This is the rule, but it is by no means absolute ; pneumoccocal peritonitis, in especial, is quite often accompanied by diarrhœa.

(xii.) *Blood Examination.* A leucocytosis (polymorphonuclear) up to about 18,000 is usual.

In typical cases the diagnosis presents little difficulty, especially as there will often be a history pointing to some disease of which peritonitis is a recognised complication. Many cases, however, do not present the classical features as described above. Especially true is this of perforated gastric or duodenal ulcer. These catastrophes are often ushered in by an acute and sudden epigastric pain which is followed by the phenomena of severe shock; definite signs of peritonitis may be absent for some hours. The pulse especially is sometimes most misleading; more than once have we seen a perforated gastric ulcer with a fairly soft steady pulse of between 80 and 90 per minute.

As a rule, however, the actual perforation is followed quickly by local abdominal signs indicative of peritoneal involvement, and if the case is seen in this stage the condition is not likely to be overlooked. After a few hours, however, there is a period of reactionary calm in which all the symptoms and signs are very much diminished and the general condition seems greatly improved. If the case is seen for the first time in this reactionary stage, or quiescent interval as it may be called, the diagnosis becomes very difficult and will depend on the history, the facies, and the pulse, aided, of course, by abdominal examination. The quiescent interval does not last very many hours, and at the end of it the symptoms of generalised peritonitis quickly become unmistakable, but from all points of view it is undesirable to postpone the correct diagnosis as late as this. This quiescent interval is also seen (but to a less extent) in acute obstruction.

The presence of free gas in the peritoneum is proof positive of a perforated viscus, but it is not very easy to establish.

The sign always described is a diminution of liver dulness in the right axillary region. This sign is of value if the liver dulness has been mapped out shortly before the perforation (which is only likely to be done in enteric fever), but the liver dulness varies so much with the alterations in distension of the colon that practically the value of the sign is not very great.

In rupture of a duodenal ulcer the tendency is for the

contents of the intestine to flow into the right iliac fossa and into the right loin, so that for a time at least the peritonitis is principally right sided.

If a ruptured duodenal ulcer remains unseen for thirty-six hours or so, the appearance may exactly simulate a perforative appendicitis. In perforated gastric ulcer, except when the ulcer is right on the pylorus, the tendency is more for the gastric contents to sweep downwards into Douglas' pouch and the signs are bilateral from the outset.

Acute generalised peritonitis may be confused with the following :—

(i.) *Intestinal Colic* (*vide* p. 360).

(ii.) *Acute Obstruction.* Here there is less rigidity and tenderness, more absolute constipation, earlier and more persistent vomiting, and no pyrexia.

(iii.) *Hysteria.* This may exactly simulate peritonitis, and unfortunately the woman who is affected this way not infrequently is under observation for gastric or abdominal symptoms. The most valuable differential points in these very difficult cases are the pulse, which is usually quite good in functional cases, the absence of *constant* diffuse tenderness if a lengthy examination is made, and the fact that the hysterical patient does not display such a disinclination to move about in bed as does one suffering from peritonitis. In addition there may be obvious signs of hysteria which may suggest the diagnosis.

(*b*) Acute Localised Peritonitis. The chief medical interest in localised acute peritonitis lies in a subphrenic abscess. This is the result of a leaking gastric or duodenal ulcer, or it may occur secondarily to disease of the liver or gall-bladder, or from downward extension of intrathoracic suppuration.

Other forms of localised acute peritonitis are ulcerating new growth, appendicitis, and perimetritis, which are more appropriately considered in text-books on surgery or diseases of women respectively.

A *Subphrenic Abscess* may be one of the most difficult diagnostic problems in medicine.

On the right side disease of the liver or appendix or a

duodenal ulcer is the usual cause, on the left side a gastric ulcer is more common.

When due to a perforated viscus there is usually gas as well as pus in the abscess cavity.

The general symptoms are those of septic absorption.

The local signs are :—

(i.) Embarrassment of respiration from impaired mobility of the corresponding half of the diaphragm. This is best shown by X-rays, but may also be detected clinically by noting the absence of pull on the costal margin of the affected side.

(ii.) Dulness at the base of the lungs, though if there is much gas the signs rather suggest localised pyo-pneumothorax.

(iii.) On the right side the liver may be pushed down.

(iv.) On the left side the stomach resonance may be impaired. It may, however, be increased if there is much gas in the abscess.

(v.) Diaphragmatic pleurisy may be diagnosed from the presence of friction and the nature of the pain.

(vi.) Rigidity of the upper rectus on the affected side is fairly common, but need not be marked.

On the whole it may be said that unless there is a history pointing to gastric or duodenal ulcer, to liver or gall-bladder disease, or to appendicitis a subphrenic abscess is more than likely to be diagnosed as a basal empyema or pyo-pneumothorax.

Indeed, this diagnosis is often correct, for subphrenic abscesses spread upwards through the diaphragm and involve the pleura and bases of the lungs very readily indeed.

(*c*) Chronic Peritonitis. One result of chronic localised peritonitis is the formation of bands and adhesions (*vide* p. 357). Chronic generalised peritonitis has been considered under the heading "Ascites" with the exception of the disease known as chronic polyorrhomenitis or chronic polyserositis, a condition characterised by multiple and recurrent serous effusions into pleuræ, pericardium, and peritoneum.

Sometimes the tubercle bacillus is responsible for this disease ; in other cases the etiology is obscure.

Chronic peritonitis is occasionally dry throughout, and in such cases, and sometimes in the early stages of the wet forms, a definite friction rub can be heard with a stethoscope, or felt manually over different regions of the abdomen, particularly over the liver or spleen when perihepatitis or perisplenitis is in active progress.

PART IV

CHAPTER I

DISEASES OF THE LIVER, BILIARY APPARATUS, AND PANCREAS

DISEASES OF THE LIVER AND BILE PASSAGES

I. GENERAL CONSIDERATIONS

The right lobe of the liver, which comprises four-fifths of the entire organ, occupies the right hypochondrium and the upper part of the epigastrium as far as the middle line. The smaller left lobe is continued across the epigastrium and gradually tapers off in the left hypochondrium, above and rather anterior to the stomach.

The functions of the liver are numerous and variable and of such vital importance that life ceases in a few minutes when the liver is extirpated. The vital functions are not the formation and excretion of bile, since life may continue for an indefinite period with obstruction to the biliary outflow.

The chief functions of the liver may be enumerated as follows :—

(i.) *Carbohydrate Metabolism.* The sugar which is brought to the liver in the portal vein is converted into glycogen and stored as such in the individual liver cells. When the body tissues require heat energy some of this glycogen is reconverted into sugar and discharged as such into the circulation.

(ii.) *Glycolytic Function.* A considerable amount of heat is furnished by the power of the liver to break down circulating sugar into CO_2 and water.

(iii.) *Proteid Metabolism.* Toxic substances, the result of proteid katabolism, are brought to the liver in the portal vein. They are converted by the liver into harmless or

even beneficial bodies, which are either used by the tissues or excreted by the kidneys in the form of urea.

The bile acids and pigments are to some extent by-products of this proteolytic function.

(iv.) *Fat Metabolism.* The liver possesses the power of both storing and splitting up fat.

(v.) *The Secretion of Bile.* In part the outpouring of bile is the means by which the liver removes some of the waste products of its own cellular activity, but at the same time the bile itself exercises several distinctly beneficial functions—it neutralises the acid gastric juice and so assists intestinal digestion, it promotes the absorption of fats by its effect on the surface tension, it has a definite though slight amylolytic action of its own, it promotes intestinal peristalsis, and it is possibly a mild antiseptic.

In health the liver dulness reaches as high as the lower border of the sixth rib in the right mammary line, the eighth rib in the mid-axillary line, and the tenth rib in the line of the scapular angle. To the left of the sternum the liver dulness is indistinguishable from the cardiac dulness with which it merges. The left lobe of the liver cannot be perceived clinically unless it is enlarged, but it extends nearly as far to the left as the left nipple line.

The lower border of the liver is not usually palpable below the right costal margin, but in the mid-line it reaches downwards for about 3 inches below the base of the xiphisternum.

In infants and young children the liver is relatively very much larger than in adults.

Various deformities of the liver are described, such as tightlacer's liver, which has a deep transverse furrow; and extra lobes, of which the most common is a tongue-like process (Riedel's lobe) reaching downwards from the neighbourhood of the gall-bladder.

The liver may be displaced downwards by thoracic disease, by subphrenic abscess, or in general visceroptosis; upwards by increase in intra-abdominal tension, or its position may be transposed in "situs inversus" or transposition of viscera.

The size of the liver varies greatly in different diseases, both general and of the organ itself.

II. JAUNDICE

By jaundice is meant a yellow coloration of the skin, mucous membranes, and conjunctivæ, occurring as a result of the deposition of bile pigments in these tissues.

All jaundice is the result of obstruction to the proper flow of bile, but this obstruction may be partial or complete, and may occur in the hepatic or common bile-ducts or in the bile capillaries within the liver. These latter cases may be styled "intra-hepatic," and include the majority of so-called "toxic" or "hæmatogenous" cases of jaundice in contra-distinction to the more obvious "extra-hepatic" or "obstructive" types. One reason why such apparently slight degrees of intra-hepatic obstruction may cause jaundice is the fact that normally bile is secreted at very low tension, so that a very slight alteration of pressure is sufficient to permit of the absorption of bile pigments into the blood.

In the toxic group are included the jaundice of certain poisons, such as phosphorus, arsenic, and snake venom; of certain specific infections, such as yellow fever, malaria, scarlet fever, or typhus; of severe toxæmias, such as acute yellow atrophy, epidemic jaundice, Weil's disease, and pernicious anæmia. It is presumed that in all these cases, except perhaps acute yellow atrophy, in which the pronounced destruction of liver cells may be sufficient to explain the jaundice, there is hæmolysis (destruction of red cells), and that the waste products so formed, especially hæmoglobin, are excreted in the form of bile pigments, which are therefore present in the bile in excess. This excess of bile pigment causes increased viscosity of the bile, which in itself is sufficient, with or without a concomitant angio-cholitis, to alter the tension in the bile-ducts enough to permit the absorption of bile into the blood-stream.

It may be remarked that often in such cases the fæces are not pale and the urine does not contain bilirubin, as is the case in the more straightforward cases of jaundice. At

the same time in pernicious anæmia and in acholuric family jaundice the urine commonly contains *urobilin* in very much larger amount than is present in health.

It is then apparent that jaundice itself is only a symptom of some underlying pathological condition; nevertheless it is convenient to discuss briefly certain clinical forms of jaundice.

(*a*) **Catarrhal Jaundice.** This is usually an affection of young people, though no age is immune. The symptoms are commonly those of an ordinary bilious attack (gastro-duodenitis), and it has been suggested that the duodenal catarrh produces enough sticky mucous to block up the opening of the ampulla of Vater and so obstruct the flow of bile. It has further been suggested that in many cases the catarrhal infection spreads through the bile papilla and produces a similar inflammation in the common bile-duct. More recently the view has been advanced that the real trouble is in the head of the pancreas, which becomes swollen from catarrh spreading up the pancreatic duct from the duodenum. Since in 60 per cent. of all cases the common bileduct traverses the head of the pancreas it is reasonable to suppose that swelling of the pancreas will obstruct this duct. As the catarrh subsides so does the jaundice abate.

In many cases the jaundice is the first sign; in others the jaundice is preceded by acute gastritis with anorexia, furred tongue and vomiting. The bowels are usually confined. There is little or no fever. As the jaundice deepens the liver often becomes enlarged and tender. The motions are clay coloured, the urine is dark and contains bilirubin.

The gastric symptoms generally subside in a few days, but they may persist for much longer and they may recur if an unsuitable diet is permitted or if the patient takes a "chill."

The prognosis is uniformly good, though a chronic catarrhal jaundice may persist, in rare cases, until some such operation as chole-cyst-enterostomy is performed.

It is important to remember that catarrhal jaundice may occur in the course of malignant disease of the liver or pancreas and may improve with suitable treatment; as

a rule, however, in malignant cases the jaundice gets progressively deeper until the end.

Jaundice is often associated with itching of the skin and a slow pulse ; both these phenomena are more marked in the malignant cases than in simple catarrh.

(*b*) **Epidemic Catarrhal Jaundice.** It sometimes happens that catarrhal jaundice occurs in a definitely epidemic form and spreads from case to case just like specific infection. Even in apparently sporadic cases, careful inquiry will often reveal other cases in the same house or family at the same time. Nevertheless, there is not at present sufficient evidence to justify the opinion that catarrhal jaundice is a specific infection, though it is probable that occasionally it may be so.

(*c*) **Weil's Disease.** A form of infectious disease in which jaundice is a prominent symptom ; there is no real evidence that there is any specificity of infectious agent, though the Bacillus proteus fluorescens has been credited with being the cause.

It is possible that tainted meat may be responsible for certain outbreaks of this disease, and in one series of thirteen cases no fewer than nine of the patients were employed in a slaughterhouse.

The onset is sudden, often with a rigor, the temperature rises quickly to 104°, and there are pains all over, but especially in the calves of the legs.

The jaundice develops on the second or third day and the liver becomes swollen and tender.

The spleen is always enlarged, and there is a coincident nephritis.

Insomnia and delirium are common during the height of the disease, but the prognosis is fairly good, since most cases are convalescent by the end of a fortnight, though there is always a tendency to relapse. The disease may be mistaken for relapsing fever or for a mild case of yellow fever : in the former case the characteristic spirillum can be detected in the blood ; in the latter the locality in which the disease occurs is an important point.

(*d*) **Icterus Neonatorum.** The majority of all infants

show a mild jaundice soon after birth which passes away completely in a few days.

This form of jaundice is attended by no unpleasant symptoms and must be regarded as physiological.

Possibly it may be due to the destruction during the first week of extra-uterine life of the excess of red blood cells which is present in the newly born.

(*e*) **Infective Jaundice in the newly born.** This may result from a bacterial infection *viâ* the umbilical vein and may be regarded as an acute infective hepatitis, which, however, is usually but one manifestation of a true septicæmia.

Infective jaundice of infants may, however, originate in the intestine, and is then said to be due to the Bacillus coli or Bacillus lacticus.

The clinical features are cyanosis, jaundice, and diarrhœa.

The mortality is high, and the duration of the disease about ten days.

This variety of jaundice is probably extremely infectious, and the stools should be as strictly treated as in a case of enteric fever.

III. ACUTE YELLOW ATROPHY OF THE LIVER (Icterus Gravis).

This is an extremely rare condition and is manifested by a profound toxæmia which affects principally the liver. The result is an extensive degeneration of the hepatic cells, severe and progressive delirium, coma and death.

The similarity of the clinical appearances in cases of phosphorus poisoning is too striking to be overlooked, but there are definite differences, and the only justifiable deduction is that the toxins of acute yellow atrophy act on the human organisms in a somewhat similar manner to phosphorus.

It may be mentioned here that in phosphorus poisoning the liver is increased in size and contains ten times the normal percentage of fat, while in acute yellow atrophy the liver is small and shrunk and contains the normal proportion of fat.

Acute yellow atrophy is twice as common in women as

in men, and is noticeably most often seen in combination with pregnancy.

The age incidence is 20 to 40 years, but cases have developed in childhood.

The symptoms are at first those of catarrhal jaundice, but after five or six days obstinate vomiting sets in and the jaundice assumes a greenish tint. Hæmatemesis is frequent, blood is often passed in the motions and other hæmorrhages are common. Within the next twenty-four hours the patient becomes delirious and then semi-conscious; leucine and tyrosine are found in the urine, the pulse becomes very rapid and feeble, and death takes place in two or three days.

The diagnosis must be made from *Phosphorus Poisoning* and *Portal Pyæmia*.

In *Phosphorus Poisoning* it should be possible to get a history of exposure to this poison or of its accidental or intentional ingestion; the *earliest symptom* is vomiting, and there is usually a quiescent interval between the vomiting stage and the graver toxic stage. Phosphorus can be detected in the vomit and the liver dulness is increased.

In *Portal Pyæmia* there is a history of some septic process in the portal area such as appendicitis; the jaundice is less deep and rigors are a feature of the case.

IV. PYLEPHLEBITIS.

Inflammatory changes in the wall of the portal vein may result either in thrombosis or in suppuration.

(i.) **Adhesive Pylephlebitis (pylethrombosis).** This may occur in any of the conditions which favour venous thrombosis elsewhere, and it is probable that both bacteria and a damaged condition of the vessel wall are necessary for its production. The condition is rare and usually fatal in a short time.

The diagnosis of portal thrombosis can scarcely ever be made, since the cardinal symptoms of ascites, enlargement of the spleen and hæmatemesis, are so commonly present in uncomplicated portal cirrhosis.

Sudden abdominal pain with the passage of blood by the

bowel, followed shortly by ascites and an enlarged spleen, may suggest the possibility of pylethrombosis.

(ii.) **Suppurative Pylephlebitis.** This is the result of a more severe infection than the preceding, with the consequence that multiple abscesses are formed in the liver substance round the branches of the portal vein. The portal vein itself is generally filled with purulent bloodstained matter, but this is not always the case, since the small abscesses may be formed by infective emboli direct from the primary site of infection.

Appendicitis is responsible for 40 per cent. of all cases, but ulceration anywhere in the area drained by the portal system may be the cause.

Recovery has occurred in a small number of cases, but death usually takes place in a few weeks.

The symptoms are a high temperature of septic type, rigors and profuse perspirations, with the addition of abdominal pain, vomiting and distension, enlargement of the liver and usually of the spleen, and very often a slight amount of jaundice.

The diagnosis is only likely to be made when the above symptoms develop during the course of some such condition as ulcerative appendicitis or some septic rectal disease.

Suppurative Pylephlebitis must be distinguished from—

(*a*) *Suppurative Cholangitis.* The general symptoms are similar, but the jaundice is more marked and the spleen does not enlarge.

(*b*) *Tropical Abscess.* The symptoms are less severe; there is no splenic enlargement, but there is generally a much more localised swelling of the liver, and there is always a history of amœbic dysentery.

(*c*) *Subphrenic Abscess.* The symptoms may be very similar, but the diaphragm is much more immobilised and at a higher level, rigors are not so constant and jaundice is the exception; further, a history pointing to previous gastric or duodenal ulcer would be strongly in favour of subphrenic abscess.

V. SINGLE LIVER ABSCESS (Tropical Abscess).

This condition is only met with as a sequel to amœbic dysentery; a fact of primary importance in its differential diagnosis.

The abscess is usually situated in the right lobe of the liver.

The symptoms are a dull pain in the liver region, which becomes acute as the abscess nears the surface, an irregular temperature, and the general phenomena of septic absorption.

The signs are variable—the most constant is an increase in the size of the liver; there may be a localised swelling, but this depends on the situation of the abscess.

Jaundice is an important point, but is often absent. Peritoneal friction may be audible if the inflammatory process reaches the surface.

Impairment of the movement of the corresponding half of the diaphragm can be demonstrated by the X-rays, and there is usually evidence of collapse of the base of the lung on the affected side. For this reason empyema is often suspected, and indeed is often present, since the liver abscess commonly ruptures into the pleura or lung.

There is usually a high leucocytosis.

A Hydatid Cyst of the liver may give rise to the same physical signs as a single abscess, and a suppurating hydatid cyst causes the same symptoms also.

As a general rule it may be said that a localised cystic swelling is strongly in favour of hydatid disease, especially if a thrill can be elicited on careful palpation. In hydatid disease there is nearly always a definite eosinophilia.

Liver abscess may be indistinguishable from subphrenic abscess, and only most careful inquiry into the previous history of the patient as regards amœbic dysentery or gastric or duodenal ulcer will lead to a correct diagnosis.

VI. MULTIPLE LIVER ABSCESS (*vide* "Suppurative Pylephlebitis").

VII. CONGESTION OF THE LIVER.

(i.) **Passive Congestion.** The so-called nutmeg liver results from imperfect function of the right heart, whether due primarily to pulmonary or cardiac disease.

The flow of blood through the hepatic veins is impeded; the blood is dammed up in the liver and secondarily in the entire portal system.

The liver becomes enlarged and tender; it may pulsate; and ascites is common.

The symptoms are chiefly gastro-intestinal and can be attributed to the catarrh set up in the alimentary tract by the portal congestion.

The liver is disturbed in its functions, and the slight jaundice so often seen in the later stages of cardiac disease may be attributed to this cause.

The diagnosis of a nutmeg liver is not required separately; the condition can be inferred when the diagnosis of chronic right heart disability has been made.

(ii.) **Active Congestion.** This is undoubtedly produced by certain infective agents, especially certain tropical diseases, such as malaria and dysentery, and it is seen most typically in those who have lived for many years in the East.

At the same time any dietetic errors which induce chronic gastro-intestinal catarrh, especially alcoholism, permit the formation of abnormal or excessive metabolic products, which in their turn result in an added flow of portal blood to the liver.

The symptoms of active congestion of the liver fall into two groups—first, those of gastro-intestinal catarrh; and, secondly, those referable more directly to the liver itself—namely, pain, tenderness, and enlargement of the organ, slight jaundice, headaches, giddiness, bad temper, and mental depression.

In the tropics the above picture is met with in a very intense form with the addition of moderate pyrexia, and it is then justifiable to consider the condition to be one of acute hepatitis.

VIII. CIRRHOSIS OF THE LIVER.

The following varieties of cirrhosis will be considered; the classification is not altogether satisfactory, but in the

present state of our knowledge it is best to consider the subject from the standpoint of morbid anatomy :—

(i.) Multilobular Cirrhosis (atrophic, portal, Laennec's cirrhosis, hob-nailed liver).

(ii.) Unilobular Cirrhosis (hypertrophic, biliary, Hanot's cirrhosis).

(iii.) Obstructive biliary cirrhosis.

(iv.) Pericellular Cirrhosis (congenital syphilitic cirrhosis).

(i.) Multilobular or Portal Cirrhosis. This disease affects men much more commonly than women, and is most often seen between the ages of 30 and 50. Children are sometimes affected.

There can be no doubt that portal cirrhosis results from the action of poisons conveyed to the liver by the portal vein. The nature of these poisons is quite unknown, but it is reasonable to suppose that they are elaborated in the stomach or intestines, and that their formation is favoured by certain articles of diet whch are unsuitable to the particular individual. Such evidence as there is favours somewhat the idea that the toxins are the result of bacterial action rather than of plain katabolism, and that the particular foodstuffs act by producing a type of gastro-intestinal catarrh which permits the growth of a group of organisms. These manufacture the particular toxins which have such a disastrous effect on the liver.

With the exception of alcohol, no special foodstuffs can be mentioned which, for certain, are capable of causing cirrhosis. The action of alcohol is unproven ; it has been suggested that it may act by inhibiting that function of the liver which detoxicates the products of bacterial activity. Certain it is that alcohol *may* produce cirrhosis, but it is equally certain that it need not do so, and that cirrhosis quite often occurs in teetotallers.

Morbid Anatomy. The essential change consists in the formation of fibrous tissue round the ramifications of the portal vein in the liver substance ; hence the fibrous strands tend to include several lobules at a time. The contraction of the fibrous tissue squeezes out the liver substance on the surface of the organ in the form of rounded

nodules (hob-nails) and may result in considerable distortion.

The liver itself may be enlarged or shrunken at the time of death ; it is probable that it is large at first and tends to shrink if the patient survives long enough.

The interference with the blood flow through the liver causes dilatation of the portal vein, and the stagnation of the blood-stream may result in portal thrombosis.

In old standing cases there is often considerable fatty change in the liver cells.

Diagnosis. The symptoms and signs of portal cirrhosis fall naturally into two groups—first, the symptoms which depend on the primary gastro-intestinal catarrh, and, secondly, those which are referable to the liver itself. The second group are often absent for a considerable time, and when they have developed the diagnosis is not difficult : at the same time the prognosis is very bad, and death can generally be anticipated in a few months.

On the other hand, the signs of gastro-intestinal catarrh do not necessarily indicate cirrhosis of the liver, so that in its early stages the diagnosis is extremely difficult if not impossible. An early stage of cirrhosis may be suspected in a man of from 30 to 50 years who has the aspect and history of alcoholic excess, and who complains of dyspepsia, morning sickness, lack of appetite (especially for breakfast), and looseness of the bowels. The tongue is furred, there is a yellowish tint to the sclerotics, and often there is a slight œdema of the shins.

It is obvious that the above phenomena must be persistent in order to justify a diagnosis of cirrhosis, a diagnosis which is supported if a large liver can be demonstrated.

As the disease progresses signs more directly referable to portal obstruction and to the liver become manifest ; these include :—

(*a*) Evidence of attempted anastomosis between the portal and systemic circulation, as shown by enlarged veins around the umbilicus and by œsophageal and rectal piles, which may burst and give rise to hæmatemesis and melæna.

(*b*) *Ascites*, which cannot be considered as purely

mechanical, as it possibly depends in part on a low grade of peritonitis and in part on an increased toxicity of the blood which damages the capillary walls.

(*c*) *Jaundice.* This is rarely present to any extent; at the same time a sallow, yellowish complexion is the rule and depends on the toxæmia and malnutrition.

(*d*) *Liver.* The liver itself may be large or small: a large, nobbly liver is a great help in diagnosis; the presence of a small liver can rarely be proved with certainty.

Course. When once ascites has developed the expectation of life is not much more than a year, and when once the ascites has become so marked as to require tapping the expectation of life is less than six months.

At the same time cases occur from time to time in which cirrhosis of the liver is present and which are tapped as many as fifty or sixty times without appearing much the worse for it. It is possible that in such cases the cirrhosis is at a much earlier stage than usually causes ascites, and that the ascites depends mainly on some other factor, such as chronic peritonitis, which is often of syphilitic origin.

Differential Diagnosis. This depends rather on excluding other possible causes for any prominent symptom. For example, hæmatemesis requires the exclusion of gastric ulcer and also of gastric carcinoma. A test meal (*vide* p. 327) may be of value in this respect; while a blood count would exclude hæmatemesis from splenic anæmia, leukæmia, or pernicious anæmia.

The differential diagnosis of ascites has been dealt with (*vide* p. 373).

In children cirrhosis of the liver is at first usually mistaken for tuberculous peritonitis. If paracentesis is performed an excess of endothelial cells indicates cirrhosis, while in tuberculosis the effusion is lymphocytic.

(ii.) Biliary Cirrhosis of Hanot (Unilobular Cirrhosis). This disease is rarely or never met with in England. It is true that the fibrous tissue not infrequently assumes a unilobular distribution in certain parts of the liver (especially under the capsule) of very advanced cases of

portal cirrhosis, but there is no reason for supposing that primarily they are anything but portal cirrhosis.

True biliary cirrhosis is supposed to be due to toxins which are absorbed into the blood and which, in process of excretion into the bile, excite a descending cholangitis. The absence of duodenal catarrh, as well as the fact that the spleenis often enlarged before there is evidence of cholangitis, seems to be much against the suggestion that the condition is an ascending inflammation of the bileducts.

The fibrosis occurs round the bile channels, and so includes but one lobule of liver substance at a time.

It is worthy of notice that in all probability certain cases of biliary cirrhosis are complicated in their later stages by superadded portal cirrhosis. In such cases the primary lesion may well be overlooked unless the patients have been under observation for a long period.

Biliary cirrhosis affects men much more frequently than women ; the usual age is 20 to 35 years, but children are by no means immune. The disease is probably always fatal, but it runs a course of several years—ten or twelve years is not at all unusual.

The clinical features are as follows :—

(*a*) *Jaundice.* This is usually definite, but not very deep ; it may vary in intensity, but never disappears. There is bile in the urine, but the motions are not clay coloured.

(*b*) *Enlarged Spleen.* This may be the earliest sign ; the spleen may become very large, and usually shows more or less perisplenitis.

(*c*) *Enlarged Liver.* The liver becomes very large ; it remains smooth and firm, and is often quite tender throughout the illness.

(*d*) *Abdominal Pain.* The large liver may cause a dull dragging pain, but, quite apart from this, there is a liability to paroxysms of severe abdominal pain, which are accompanied by deepening jaundice and an increase in the size of both liver and spleen. They do not last long, but recur with increasing frequency.

Biliary cirrhosis must be distinguished from :—

(1) *Portal Cirrhosis.* In this jaundice is rare and

ascites is common ; the patient is older and the duration of the established disease is much shorter.

(2) *Malignant Disease.* The age of the patient, the shape of the liver, the evidence of malignant disease elsewhere, the frequency of an obstructive type of jaundice, and the very short course of the case should not present much difficulty.

(3) *Gall-Stones.* The liver is not usually much enlarged, the spleen is rarely enlarged at all ; the jaundice is much deeper, and the stools are, for a time at least, clay coloured.

(4) *Syphilis of the Liver.* The liver is not often uniformly enlarged ; the spleen need not be enlarged at all, and there should be other evidence of syphilis. A Wassermann reaction must be performed.

(iii.) Obstructive Biliary Cirrhosis. In certain cases of biliary obstruction a unilobular type of cirrhosis may develop as a secondary condition.

The symptoms are those of primary biliary obstruction.

(iv.) Pericellular Cirrhosis. One manifestation of congenital syphilis may be an acute diffuse hepatitis. If the infant survives long enough a diffuse intra-lobular or pericellular fibrosis results.

The diagnosis may be made from the discovery of a large, hard, smooth liver, with, or more rarely without, other manifestations of congenital syphilis. Jaundice may develop and is an unfavourable sign.

IX. NEW GROWTHS OF THE LIVER.

Innocent neoplasms of the liver are not of clinical significance.

Angeiomas are frequently found post mortem.

Malignant disease of the liver may be primary or secondary; in either case the growth may be a sarcoma or a carcinoma.

Secondary growths in the liver are very common, and carcinoma is much more often seen than sarcoma.

Primary growths in the liver are very rare, but when present are nearly always carcinomatous. The growths are usually secondary to a primary focus in the portal area, especially the stomach and head of the pancreas.

The symptoms are, first, those common to malignant disease elsewhere (and here it may be stated that a large number of patients die of carcinoma elsewhere and have, post mortem, secondary deposits in the liver, and yet during life there was no reason to suspect that the liver was involved); and, secondly, symptoms more directly referable to the liver, such as an obstructive type of jaundice and a large, nobbly or irregular liver.

Ascites is not infrequent, but it is not so common as jaundice.

In malignant disease of the liver the jaundice often depends on the presence of a malignant gland in the portal fissure, or carcinoma of the head of the pancreas. The jaundice deepens steadily to a deep olive-green colour, hardly ever remits, and is often accompanied by severe itching of the skin and by a very slow pulse.

The urine contains bile, and the motions are pale, greasy, and very offensive.

Other signs which may occur are secondary nodules of growth at the umbilicus, audible friction from perihepatitis when a nodule reaches the surface of the liver, and an irregular pyrexia.

Diagnosis. Great importance has been laid on the occurrence of manifest enlargement of the gall-bladder, since it is said that this does not occur with jaundice unless the cause is malignant disease.

Though there are many causes of enlarged liver, these usually present certain characteristic clinical features, and do not enter into the practical diagnosis of malignant disease of the liver.

Great difficulty may, however, arise, when there is a large liver and no discoverable primary lesion, in distinguishing between malignant disease, cirrhosis, and syphilis.

In addition to the history and aspect of the patient the following may assist :—

If the liver can be felt the size of the nodules is important, since the cirrhotic nodules are always small.

The occurrence of jaundice is somewhat in favour of malignant disease, but the character of the jaundice, with

enlarged gall-bladder and clay-coloured stools, as described above, is very important evidence.

Enlargement of the spleen is distinctly in favour of cirrhosis.

An umbilical nodule or a gland above the clavicle may be considered proof positive of malignant disease.

The cicatrisation of gummata in the liver may produce deformities closely resembling malignant disease. Syphilis however, is not often accompanied by jaundice, neither is ascites a likely occurrence.

In any case of doubt a Wassermann reaction should be performed, and, although a person with malignant disease of the liver may well have syphilis also and so give a positive reaction, the result of treatment by mercury and potassium iodide will soon settle the question.

X. GALL-STONES.

Women are much more prone to gall-stones than men; they usually develop in the second half of life, but no age is immune.

The formation of these calculi is influenced to some extent by diet, since they are relatively infrequent in people who eat a great deal of meat.

The common basis for gall-stones is cholesterin; occasionally pure pigment calculi are met with. Cholesterin is secreted by all mucous surfaces and therefore by the gall-bladder, so that stagnation of bile predisposes to an excess of cholesterin in the gall-bladder. This excess of cholesterin may be precipitated by any factor tending to produce a deficiency in bile-salts, which normally help to keep it in solution.

In addition to stagnation of bile, some inflammatory change in the epithelium of the gall-bladder is probably necessary for the formation of calculi; catarrhal inflammation may be sufficient, but in most cases, possibly in all, there is a bacterial element at the bottom of the inflammatory process. The Bacillus coli, Bacillus typhosus, staphylo- and strepto- cocci have been shown to be capable of pro-

ducing calculi, and Robson believes that the influenza bacillus may have a similar power.

The calculi vary in size from sand-like particles to masses the size of a hen's egg or larger; they are soft and light, and when multiple show facets from friction against each other.

Symptoms. The calculi may remain in the gall-bladder for years without ever giving rise to symptoms: in such cases a tumour is rare, since the gall-bladder tends to contract.

On the other hand, even though the calculi remain *in situ*, urgent symptoms may develop as the result of cholecystitis, which is, of course, more prone to occur when gall-stones are present.

If the calculi are expelled from the gall-bladder, biliary colic is usually very obvious while they are passing down the cystic duct and again while they are traversing the ampulla of Vater. If they are of any size, they also cause pain while in the common duct, especially at first; and if the common duct is blocked by a calculus, obstructive jaundice is produced.

Rarely a calculus may ulcerate through the wall of the gall-bladder or of the common bileduct and may reach the intestine in this manner; it may even cause intestinal obstruction as practically a first sign of its existence. The more prominent features of cholelithiasis are as follows:—

(i.) *Attacks of Biliary Colic.* These come on suddenly, usually for no apparent reason—sometimes two to three hours after food; the pain is most acute and is referred for maximum intensity to the right hypochondrium, but radiates over the abdomen and also to the right shoulder-blade.

Each attack lasts from a few minutes to several hours, and as a rule is accompanied by vomiting. The stone may slip back into the gall-bladder or be passed into the common duct or into the intestine, when the pain ceases.

(ii.) *Intermittent Pyrexia.* Shivering attacks are of frequent occurrence in gall-stones, and may simulate malaria. It is doubtful whether they are due to toxic absorption or to nervous influences; certainly a marked cholecystitis or cholangitis is not a necessary concomitant. Probably

they are most often met with when the stones are in the common duct, and Osler considers the following association of symptoms to be practically pathognomonic of calculi in the common bileduct :—

(*a*) Jaundice of varying intensity, persisting for a long time.

(*b*) Ague-like paroxysms, after each of which the jaundice deepens for a time.

(*c*) Pains in the region of the liver and general gastric disturbance at the time of each febrile paroxysm.

(iii.) *Jaundice.* So long as the stones remain in the gall-bladder there will be no jaundice, but when they reach the common duct there is likely to be some obstruction to the flow of bile, and so jaundice occurs. If the stone floats in the common duct, there need be no jaundice. If the obstruction remains complete, the jaundice steadily deepens until the characteristic olive-green colour develops. If the stone passes on into the duodenum, the jaundice clears up.

(iv.) *Enlargement of the Gall-Bladder.* It is rare for the gall-bladder to enlarge when the common duct is obstructed by gall-stones, whereas enlargement commonly occurs when the common duct is obstructed by new growth ; presumably the reason is that calculi in the gall-bladder generally cause it to shrink and atrophy so that it becomes incapable of dilatation.

This rule is known as Courvoisier's law and, though not invariable, affords great help in the differential diagnosis.

(v.) *Collapse.* Persons suffering from biliary colic are often profoundly collapsed ; indeed, feeble individuals may succumb during the course of an attack.

(vi.) *Tenderness.* In nearly all cases definite tenderness can be elicited by pressing firmly along the line joining the ninth costo-chondral junction with the umbilicus, often about the junction of the lower and middle thirds of this line.

(vii.) Sometimes the presence of gall-stones is shown by no more definite signs than attacks of pain in the upper abdomen (not necessarily right sided) associated with distressing flatulence, and possibly dark urine and occasional pale motions but no jaundice and no typical biliary colic.

Diagnosis. A careful consideration of the above features will usually make the diagnosis clear, but at first sight difficulty may arise in the following cases :—

(*a*) *Appendicular Colic.* There is no pain about the right scapula, and there is nearly always marked tenderness in the right iliac fossa, especially over McBurney's spot (the junction of outer and middle thirds of the line joining the anterior superior spine of the ilium to the umbilicus).

A retro-cæcal appendix may cause considerable pain above the transverse umbilical level, but there is nearly always tenderness as well in the right iliac fossa.

(*b*) *Duodenal Ulcer.* The pain occurs about three hours after food and is relieved by food and by alkali. Shivering attacks are not present.

(*c*) *Tabetic Crises.* There is no jaundice and no shivering, while other signs of tabes dorsalis will be found on careful examination.

(*d*) *Intestinal Colic.* There are no true paroxysms, as in biliary colic, rather a constant pain with exacerbations ; the pain is not so localised as in biliary colic.

(*e*) *New Growth in Liver* (*vide* p. 395).

XI. CHOLECYSTITIS.

The gall-bladder, especially when it contains stones, is liable to attacks of inflammation which closely resemble affections of the appendix vermiformis and which vary in severity from simple catarrh to gangrene and perforation. Not only is the pathology similar, but the symptoms are identical, if we remember that we are dealing with the right hypochondrium instead of the right iliac fossa.

XII. CHOLANGITIS.

(i.) *Catarrhal Cholangitis* has been considered under the heading of "Jaundice."

(ii.) *Infective Cholangitis* is usually the result of a non-suppurating bacterial infection of the bileducts supervening on the presence of calculi in the common bileduct. The calculi are not usually impacted, so that they act as an intermittent ball-valve.

Clinically there are a number of attacks of biliary colic, usually without jaundice; until after one such paroyxsm there is a rigor. Subsequently rigors occur at irregular or regular intervals until the calculi pass or are removed or until death occurs from the development of a suppurative process or from toxæmia.

Malaria must be excluded by the history and by examination of the blood for the parasite.

(iii.) *Suppurative Cholangitis* usually follows gall-stones, but may occur during the course of the specific infective diseases, especially enteric fever, or as an accompaniment to carcinoma or hydatid cyst.

The following symptoms are met with :—

(*a*) Evidence of septic absorption, such as irregular swinging temperature, perspirations, rigors, and leucocytosis.

(*b*) Steady increase in size of the liver, often painless, unless gall-stones are present, when paroxysmal pain is usual.

If the trouble is not due to gall-stones, the gall-bladder is enlarged also.

(*c*) *Jaundice*, which is persistent and severe.

Such cases present a great similarity to suppurative pylephlebitis, which can, however, usually be distinguished by the lesser degreee of jaundice and a source of infection being present in the area drained by the portal vein.

DISEASES OF THE PANCREAS.

Inflammatory affections of the pancreas may be acute or chronic. Acute inflammations may be hæmorrhagic or non-hæmorrhagic. Chronic inflammation is followed by diffuse fibrosis.

I. **Acute Hæmorrhagic Pancreatitis.** Traumatic causes may induce sudden bleeding into the substance of the pancreas, but apart from these the cause of such hæmorrhage is obscure. Gall-stones are present in some of these cases, and it has been suggested that one factor may be the regurgitation of bile up the canal of Wirsung. The presence of bile in the pancreatic duct certainly can cause acute inflammatory changes.

Theoretically, at any rate, hæmorrhage may follow the liberation of the pancreatic juice into the gland interstitia, by reason of its digestive powers, and such liberation may conceivably occur in simple inflammatory pancreatitis as a result of rupture of small retention cysts.

Arterial degeneration and any of the morbid states which favour hæmorrhage may be factors in some of the cases.

Most of the patients are fat. Men are more frequently affected than women, and the average age is 30 to 50 years.

Symptoms. The first sign is a sudden and extremely severe abdominal pain followed shortly by collapse: the pain is most marked in the epigastrium or round the umbilicus. In this neighbourhood there is great tenderness and rigidity, while distension of the abdomen, but especially of the upper part, develops in a few hours. Vomiting sets in almost at once and is likely to continue, but does not become fæcal in character. The bowels are confined. There is little or no pyrexia, and the pulse rarely rises to more than 100 per minute. A high leucocytosis is the rule.

If the loss of blood is very great, the signs of collapse are especially marked. Death usually occurs from exhaustion about the third day.

Hæmorrhagic pancreatitis may be mistaken for :—

(i.) *Acute Intestinal Obstruction.* At first differentiation may be impossible, but after a time the appearance of fæcal vomiting and the very rapid pulse would suggest the diagnosis.

Epigastric distension and possibly a palpable epigastric tumour from the effused blood are much in favour of pancreatitis.

(ii.) *Superior Mesenteric Infarction.* The presence of a cardiac lesion and the passage of blood by the bowel may help in this case, otherwise the early symptoms are identical.

(iii.) *Rupture of a Hollow Viscus.* The tenderness and rigidity are more diffuse, while unequivocal evidence of peritonitis is not long delayed.

(iv.) *Biliary Colic.* Jaundice, if present, is a valuable aid, and the general symptoms are but rarely so severe as in hæmorrhagic pancreatitis.

II. **Acute Non-hæmorrhagic Pancreatitis.** The etiology of this condition is obscure ; it may be associated with gall-stones (about 40 per cent.), with certain specific infections (mumps or enteric fever), with embolism, and with pancreatic calculi. A certain number of cases may be due to the spread of organisms up the canal of Wirsung in the course of a duodenal inflammation.

Men are more frequently affected than women.

All stages of inflammation may occur, from simple leucocytic infiltration to gangrene.

The symptoms and signs resemble those of the hæmorrhagic form, but are not quite so sudden in onset. They often take some hours to reach their maximum, and there may be relative intermissions.

Rigors are not unlikely in this variety ; there is usually distinct pyrexia, and the pulse tends to be rapid.

The urine occasionally contains sugar, but not sufficiently often for its presence to be of clinical value.

The gland may become so swollen as to obstruct the common bileduct and so cause jaundice. A palpable epigastric tumour, sometimes sausage-shaped, may become apparent.

The differential diagnosis is practically the same as for the hæmorrhagic form.

Some cases are subacute from the outset ; others start acutely and merge into a subacute or chronic form.

III. **Chronic Pancreatitis.** Some of these cases are definitely infective, and have been shown to be due to the Bacillus typhosus or to the Bacillus coli ; others must be regarded as toxic, and in all probability the same factors which produce cirrhosis of the liver can produce a similar change in the pancreas. It is thought that the toxins of the tubercle bacillus may be eliminated in the pancreatic juice and may cause irritation of the organ during their excretion.

Pancreatic calculi may cause fibrosis by damming up secretion just as gall-stones may cause obstructive biliary cirrhosis.

Lastly, in common with all other organs of the body, the pancreas may become fibrosed from simple senile vascular changes.

The sexes seem to be affected equally, and the disease is most usual in the second half of life.

Symptoms. Many cases give rise to no symptoms and are only discovered after death.

Such symptoms as may be present are wasting, lack of appetite, and alteration in the character of the fæces. Jaundice is frequently present, and is likely to be accompanied by a palpable gall-bladder. There are likely to be general dyspeptic phenomena, and bouts of severe abdominal pain are not infrequent.

A certain proportion of cases of chronic pancreatitis ultimately develop diabetes.

Since disturbance of the pancreatic function results in disordered fat digestion, the motions contain an excess of fat and are pale, bulky, offensive and greasy. The important point seems to be an increased proportion of neutral fat as compared with fatty acids.

A lack of bile produces a similar but less marked condition, but the motions in this case are generally acid, whereas they are alkaline when the pancreas is at fault. Further, pancreatic disability is shown by the presence of undigested muscle fibres in the fæces.

Cammidge's test is thought by some to furnish reliable evidence of active pancreatic disease. It is said not to occur in cases of simple fibrosis or of blocked secretion.

It appears to depend on the presence in the urine of these cases of a substance which is probably "pentose." In its simplest form the reaction may be performed as follows :—

Filter a twenty-four hours specimen of urine, and remove albumin and sugar if such be present.

Add to 20 cc. of the filtrate 1 cc. of strong hydrochloric acid. Boil gently for ten minutes and cool under the tap. Make up the solution to 20 cc. with distilled water and add 4 grms. of lead carbonate.

Shake till no more gas is evolved and filter.

Add 4 grms. of tribasic lead acetate.

Shake : allow to stand for a few minutes and filter.

Add 2 grms. sodium sulphate.

Shake for several minutes and then bring slowly to boiling point on a sand bath.

Cool under the tap and filter yet again.

Of this filtrate take 10 cc. and make up to 18cc. with distilled water.

Add to this solution ·8 grm. phenylhydrazine hydrochlorate, 2 grms. sodium acetate, 1 cc. of 50 per cent. acetic acid.

Boil gently on a sand bath for ten minutes.

Filter through filter paper moistened with boiling water.

Measure the filtrate, and if it is less than 15 cc. make up to this bulk with distilled water.

Place this final solution in an ice-chest for four hours.

In a positive reaction there will be an ample deposit of circular tuft-like ozazone crystals, which should be examined microscopically and which should disappear in ten to fifteen seconds if some 33 per cent. sulphuric acid be run under the cover-slip.

A positive Cammidge reaction, like glycosuria, is confirmatory evidence of pancreatitis either acute or chronic; a negative reaction, like the absence of sugar from the urine, is of little or no value.

Cammidge has also evolved a satisfactory technique for the estimation of neutral fat and fatty acids in the fæces, but the technique, which involves very accurate gravimetric processes, is hardly practicable except in a fully equipped laboratory.

The following test has recently been elaborated in the effort to arrive at some means of more accurately gauging the efficiency of the pancreas: it is likely to prove of considerable value in the diagnosis of certain cases of pancreatic disease and also in certain cases of renal disease:—

The diastase reaction. There is normally present in the blood a certain amount of diastase some of which is excreted with the urine.

In health the quantity in the urine is fairly constant, but in disease, especially of the pancreas and the kidneys, there is much variation in the amount found.

Technique. Three solutions are required:—

(i.) A 1 per cent. solution of sodium chloride.

(ii.) A 0·1 per cent. solution of "soluble starch" (Kahlbaum). The solution is prepared by stirring the

starch into the water as it boils in a beaker. It should be prepared fresh every day, as the starch tends to separate out on standing.

(iii.) A $\frac{N}{50}$ solution of iodine. This is an unstable solution. It should be made up fresh as required from a decinormal solution which is stable.

A series of ten test tubes is taken.

With a graduated 1 cc. pipette the following amounts of urine (24 hours specimen) are delivered :—

(i.) 0·6 cc. urine.
(ii.) 0·5 cc. ,,
(iii.) 0·4 cc. ,,
(iv.) 0·3 cc. ,,
(v.) 0·2 cc. ,,
(vi.) 0·1 cc. ,,
(vii.) 0·09 cc. ,,
(viii.) 0·08 cc. ,,
(ix.) 0·07 cc. ,,
(x.) 0·06 cc. ,,

These latter small amounts are best added by means of a 1 in 10 dilution of urine, adding instead of 0·09 cc. of undiluted urine 0·9 cc. of the dilution, etc.

The amount in each tube is made up to 1 cc. with the saline solution and to each tube is added 2 cc. of the starch solution. The salt and starch are best delivered from burettes graduated in tenths of a cubic centimetre.

The tubes are now labelled and placed in a water bath at 39° C. for half an hour. They are then placed in cold water for a few minutes to stop the ferment action, and to each tube is added a drop of the $\frac{N}{50}$ iodine solution.

The tube containing the greatest amount of urine in which a mauve colour is produced is noted ; it is called the " limit tube." In a normal urine this is commonly the 0·1 cc tube. In the tube next above this (the 0·2 cc. tube) all the starch has been converted by the ferment.

0·2 cc. of urine will convert 2 cc. of the starch solution : therefore, 1 cc. of urine will convert 10 cc. of the starch solution. This number 10 is known as the diastase number.

In health the diastase number varies somewhere between 10 and 30.

The test is particularly applicable to disease of the pancreas in which there is some obstruction to the duct (in which case there is more diastase passed into the blood and so into the urine), and to renal disease where there is difficulty of elimination due to organic change in the kidney (in which case less diastase appears in the urine).

It has been used as a test of renal function in comparing specimens from ureteric catheterisation of both kidneys, always provided that the two ureters are catheterised simultaneously.

The features which suggest pancreatic disease have been indicated above; but it will be obvious that it may be very difficult to exclude certain affections of the liver and bile-ducts. Again, when pancreatic disease has been diagnosed it may be difficult to say whether it is innocent or malignant.

IV. **Tumours of the Pancreas.** (i.) *Cystic Tumours.* So-called pancreatic cysts are usually peri-pancreatic and due to the collection of fluid in the lesser sac of the peritoneum after blocking of the foramen of Winslow.

True retention cysts of the pancreas do, however, occur, especially in conjunction with chronic pancreatitis.

Some writers hold that all pancreatic cysts are really hæmorrhagic in origin.

The symptoms vary with the causes of the cyst; in many cases a tumour is the first thing complained of.

The characteristic tumour is smooth and rounded, it moves slightly, is dull to percussion, and may give a transmitted impulse from the aorta, but is not expansile and does not give a thrill.

It first presents below the left ribs, and may, as it increases in size, fill the whole of the upper part of the abdomen.

If the size of the tumour increases rapidly, hæmorrhage into it is probable. When very large, it may interfere with the diaphragm and press on adjacent structures, such as the portal vein and intestines, which indeed may become obstructed.

The cyst does not often reach any great size without causing pain in the epigastrium and marked digestive disturbance.

Those cases which are due to retention cysts resulting from

chronic pancreatitis give the clinical manifestations of this condition often before the tumour is apparent (*vide* p. 402), and where the main duct is obstructed there is likely to be jaundice vomiting and paroxysmal pain.

When once the tumour can be recognised its diagnosis is not difficult ; it generally appears between the stomach and the colon, and these organs can be demonstrated after inflation lying respectively above and below the dull globular mass.

(ii.) *Solid Tumours of the Pancreas.* Clinically the only important solid tumour of the pancreas is carcinoma.

This is usually situated in the head of the pancreas and is for a long time painless.

In addition to the constitutional signs common to all malignant disease carcinoma of the pancreas is generally associated with the clinical phenomena of pancreatitis (*vide* p. 402), and also with jaundice of obstructive type and with an enlarged gall-bladder. In addition to the enlarged gall-bladder the main growth may be palpable as a tumour in the neighbourhood of the pyloric end of the stomach, from which it is often indistinguishable, and which, indeed, is often implicated by direct extension.

The liver is generally the site of secondary deposits, so that the features of carcinoma of the liver may be superadded.

In the earliest stages the diagnosis rests upon :—

(*a*) Rapid emaciation and increasing muscular weakness.

(*b*) Painless jaundice.

(*c*) Palpable gall-bladder.

The short history will usually serve to exclude chronic pancreatitis.

A positive diagnosis is of value, since when it is possible a patient may be spared a useless laparotomy.

CHAPTER II

EXAMINATION AND ANALYSIS OF URINE

I. GENERAL CONSIDERATIONS.

The diagnostic value of urinary examination can hardly be over-estimated; it is a procedure which should never be omitted, no matter how improbable it may appear that anything abnormal will be found.

In the present chapter the chief properties of the urine, normal and pathological, will each be considered briefly from the point of view of their diagnostic significance, and the simple tests which are needful for their respective investigations will be discussed.

Whenever possible a sample of the mixed specimens of all the urine passed during the twenty-four hours should be used for all examinations, except microscopical. In the latter case the urine should be examined as soon as possible after being passed. If the urine has to be sent away for examination it should be placed in a sterile stoppered bottle and a few cubic centimetres of chloroform added to it for chemical examinations, while for examination of sediments a few drops of formalin may be substituted. For bacteriological purposes a catheter specimen should be received in a sterile vessel and sealed up immediately without the addition of any preservative.

The amount of urine passed in twenty-four hours varies between 1,000 and 2,000 cc.; the average usually given is 1,500 cc., though this is probably rather high.

In health more urine is secreted in the daytime than at night, but the converse obtains in cases of interstitial nephritis.

The urinary output depends on the nature of the solids to be excreted and also on the rate of the blood flow through

the renal arteries; high blood pressure only causes polyuria if it increases the rate of the blood flow.

The amount of urine is increased in chronic interstitial nephritis, diabetes mellitus, diabetes insipidus, hysteria and certain cerebral lesions; also in cold weather, after the ingestion of much fluid, and during convalescence from acute infections. It is decreased in acute nephritis, chronic tubal nephritis, where there is anasarca or a serous exudate, in high fever, and when there is very free perspiration.

Specific Gravity. If water is 1,000, the normal specific gravity of urine is 1,020 at the ordinary room temperature. The urine of infants is normally about 1,007.

The specific gravity is high in concentrated urines, and if there are abnormal constituents present, such as sugar or albumin, except when, in the latter case, the high solid content is more than counterbalanced by the increase of watery matter. In the case of sugar the polyuria is rarely sufficient to induce a low specific gravity. Low specific gravities depend almost entirely on an excessive output of watery urine.

If the last two figures of the specific gravity are multiplied by the arbitrary coefficient 2·3, an approximate estimate of the solids in the urine will be found expressed in grammes per litre.

Colour. The natural colour of urine is some variety of yellow, and the more watery the urine the paler the colour, except in diabetes. Acid urines are usually darker than alkaline, and the urine of uræmia is often peculiarly pale. Febrile urines are dark owing to concentration.

The urinary pigments normally present are urochrome, urobilin, and hæmatoporphyrin. Urobilin is not present as such when the urine is passed, but is soon formed by the action of light on urobilinogen. Uroerythrin is frequently present in urines which are rich in urates. Urobilin may be tested for as follows:—

(i.) Add excess of ammonia and then a few drops of a 1 per cent. solution of zinc chloride: a green fluorescence indicates urobilin.

(ii.) Add a few drops of tincture of iodine to 10 cc. of

the urine and examine with the spectroscope: a band between the green and blue indicates urobilin.

The colour of urine may be affected by the presence of the following abnormal constituents:—

Blood and its Derivatives. Urine which contains blood varies in colour, according to the amount of blood present, from reddish-brown to a faint smoky tinge. Methæmoglobin causes a brown rather than a red tinge, and hæmoglobin, as seen in blackwater fever and paroxysmal hæmoglobinuria, causes the urine to appear greenish black when present in large amount. Hæmatoporphyrin, when present in considerable quantities, as in cases of sulphonal poisoning, imparts a dark violet colour to the urine.

Bile. When jaundice is at all marked bilirubin and biliverdin are usually present in the urine, and the colour is brown, green, or even greenish-black. If the urine is shaken, even small quantities of bile will give a yellow coloration to the foam which is characteristic.

Melanin. This pigment has been found in certain cases of melanotic growths in the viscera, especially in the liver; it is not commonly present when the urine is passed, but is formed soon after passing by oxidation of its precursor melanogen.

Ferric chloride causes immediate darkening of such a urine, and a grey precipitate is formed which is soluble in excess of the reagent (von Jaksch).

Drugs. When the urine is of a striking colour careful inquiry should be directed to the recent drug history. Methylene blue, even in minute doses, imparts a greenish blue colour to the urine. Carbolic acid and other coal-tar derivatives often cause marked darkening of the urine; the coloration may not appear till the urine has stood a long time and has become alkaline. The colouring matter is usually pyrocatechin or hydroquinone. These bodies reduce copper, but not bismuth.

Rhubarb, santonin, senna, and other drugs containing chrysophanic acid impart a bright yellow colour to acid and a reddish tinge to alkaline urine.

Reaction. Urine is normally acid, owing to the presence of acid sodium phosphate, except for a short time about

two hours after a good meal, when the reaction is temporarily alkaline (the alkaline tide).

The acidity is rarely very great and varies markedly with the proteid intake. (To estimate the total acidity, *vide* p. 413.)

Alkalinity of the urine, if caused by volatile alkali (ammonia), is always due to bacterial decomposition. Red litmus will be turned blue if hung over the mouth of a bottle containing urine which is alkaline from excess of ammonia; but this colour change is not produced by fixed alkali unless the litmus paper is dropped into the urine or allowed to remain suspended above it for a prolonged time.

Smell. The odour of decomposing alkaline urine is ammoniacal; normal urine has but little odour. Acetone imparts a striking smell of apples and chloroform to the urine. The smell of the oleo-resins can readily be appreciated in the urine, and a smell of violets is produced by the ingestion of turpentine.

Translucency. Healthy urine is clear when freshly passed; as it cools urates may be deposited, pink in adults, but often white in childhood, or a cloud of mucus may appear in the upper layers of the fluid. Definite cloudiness when passed may be due to an excess of phosphates in alkaline urine, to pus, or to bacteria. Pus and phosphates will settle to the bottom of the vessel, but bacteria will keep the urine permanently cloudy.

II. NORMAL URINARY CONSTITUENTS.

(i.) **Nitrogenous Bodies.** The nitrogen output is made up in four ways, and the average on a full proteid diet is as follows (Folin):—

Urea	87 per cent.
Ammonia	3 ,, ,,
Uric acid	2 ,, ,,
Extractives	8 ,, ,,

The total amount is about 16 grms. of nitrogen daily.

The nitrogen output is high in fevers, in cachexia, in diabetes, after protoplasmic poisons such as phosphorus or

arsenic, and also where large inflammatory exudates are in process of absorption.

The nitrogen output is notably diminished in nephritis, though in such cases nitrogen equilibrium may, to some extent, be maintained by an increased output in the fæces.

The estimation of the total nitrogen output involves a somewhat laborious technique (Kjeldahl's method) and is not often necessary in clinical work. The urea estimation (so called) is generally a sufficient guide.

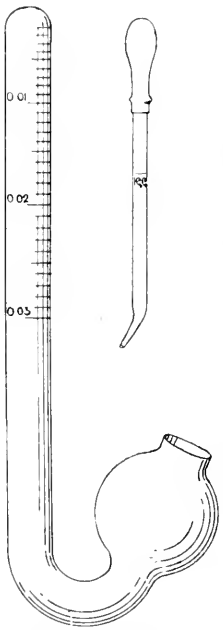


Fig. 54. — The Ureometer of Doremus.

Urea. The daily output of urea varies from 20 to 40 grms. and averages about 2 per cent. of the urine. It depends largely on the nature of the diet, and it should always be borne in mind that this may influence the value of a urea estimation.

The most convenient methods, for clinical use, of investigating the urea output are those of Gerrard or Doremus, though other nitrogen than that of urea is included in the estimate. More accurate investigation must be left to a highly-skilled chemist.

Both Gerrard's and Doremus's methods depend on the decomposition of urea by an alkaline hypobromite solution and the consequent liberation of CO_2, which is allowed to displace water in a vessel graduated in such a way as to permit of the resulting fluid level being read off as the percentage of urea in the urine.

The hypobromite solution must be freshly made for each experiment by adding 2 cc. of liquid bromine to 23 cc. of 40 per cent. aqueous sodium hydrate solution.

The necessary amount of urine (5 cc. for Gerrard's test) is carefully measured from a twenty-four hours specimen before being added to the hypobromite in the specially-devised apparatus.

The instrument devised by Doremus is easy of application

and reasonably accurate. It consists (Fig. 54) of a graduated tube closed at one end and having the other end bent in U-shape and expanded to a bulb. There is also a pipette graduated to contain 1 cc.

The bulb is filled with hypobromite solution, being tilted the while to expel all air. The pipette is filled with urine to the mark, and this urine is expelled into the long limb of the ureometer. The liberated nitrogen collects at the top of the tube and the level of the gas is read off on the scale, which is so graduated as to read either the percentage of urea or the amount in grains per ounce.

Although the urea percentage permits of wide variations in health it is not advisable to perform any major surgical operation when the reading is under ·6 per cent.

Ammonia Nitrogen. The importance of this method of nitrogen excretion is becoming more widely recognised. The normal amount of ammonia in the urine is approximately ·7 grm., or 4 per cent. of total nitrogen : this is increased on a very fatty diet and after the intake of organic and sometimes of inorganic acids ; it is often high in diabetes and any case of oxygen starvation. Any big increase is accompanied by toxic symptoms, and in fatal cases focal necrosis has been found in the liver.

Perhaps the most practical use of the above has been found in cases of pernicious vomiting of pregnancy ; in these there is a very high ammonia output, whereas in other forms of vomiting associated with pregnancy there is not. Hence there is a definite indication for emptying the uterus when the ammonia nitrogen percentage reaches 10 to 12 per cent.

Fortunately there is a fairly simple method for estimating the ammonia content of the urine. The first stage is that usually adopted for estimating the total acidity and is as follows :—To 25 cc. of freshly-voided urine add an equal bulk of distilled water, 15 grms. of neutral potassium oxalate, and ·5 cc. of phenolphthalein solution ; shake the mixture thoroughly till the oxalate is dissolved and then titrate with decinormal caustic soda until a very faint pink colour appears. The number of cubic centimetres of caustic soda used represents the total acidity of the urine. For the

estimation of the ammonia first neutralise 10 cc. of 20 per cent. formalin with the phenolphthalein and caustic soda solution and add this to the already neutralised urine. This causes the pink colour to disappear, and a further quantity of the standard soda solution must now be added to cause the reappearance of the pink tint. The extra amount of caustic soda which it has been necessary to use (in cubic centimetres) multiplied by ·0014 represents the amount of nitrogen present as ammonia in the original 25 cc. of urine taken.

N.B. The end points in these reactions are rendered more distinct by the precipitation of the calcium phosphate by the preliminary addition of oxalate; it is also important to add not less than ·5 cc. of the indicator.

Uric Acid. This substance is only present to the amount of about ·7 grm. daily, and is produced for the most part by oxidation of nuclein bases, both endogenous and exogenous. Very little is known concerning the significance of its increased production, and in all probability little or no information is gained by its quantitative estimation. It is increased *pari passu* with urea in febrile states, and also in leukæmia, diabetes, and cirrhosis of liver. With regard to gout, the uric acid output is lessened between the attacks but above normal during the exacerbations.

The endogenous uric acid is the important factor, and in order that any value whatever may be elicited by the quantitative estimation of the uric acid output the patient should be on a nuclein-free dietary so as to control the exogenous production.

The presence of uric acid may be demonstrated by the murexide test:—Evaporate some urine in a porcelain basin and dissolve some of the residue in a few drops of nitric acid; again evaporate slowly to dryness, when a red coloration occurs. If ammonia now be added the red colour becomes tinged with purple.

An accurate quantitative determination requires a very complicated technique, but a rough estimation may be made as follows:—Divide the lower half of a test tube into seven equal parts; fill up to the second mark with pure nitric acid; pour the urine previously freed from

albumin gently on to the nitric acid till it reaches the seventh mark. If the cloudy ring of urates appears above the line of junction before five minutes has elapsed, the uric acid is increased; if the cloud is delayed much more than five minutes, the uric acid is diminished (Heller).

The amount of uric acid seems to vary inversely with that of the purin bases.

(ii.) **Chlorides**. From 10 to 15 grms. of chlorides are excreted daily in the urine. The chief importance of the chlorides seems to be their relation to serous and fibrinous exudations. They are diminished or absent in cases of exudation, notably in pneumonia, but their output soon returns to normal as the exudate commences to be absorbed.

This chloride retention is very remarkable and has not been fully explained; there is evidence to show that the chlorides are accumulated in the tissues rather than in the blood. These facts have led to the advocacy of salt-free diet in cases of cardiac and renal œdema, and in certain cases there is no doubt that the œdema can be lessened if chlorides are withheld from the food.

An accurate quantitative determination of chlorides is not of clinical importance, but the fact of their diminution or otherwise can readily be estimated as follows:—First free the urine from albumin by boiling and filtration; then add to a test tube half-full of this about 8 drops of nitric acid; and finally add 2 or 3 drops of 10 per cent. silver nitrate solution. If the chlorides are normal, a thick, flocculent precipitate is formed; if decreased, the precipitate is much less thick, or only a cloudiness may be noticed. It is convenient to perform a control experiment with a known normal urine.

This question of chloride diminution is often of importance in the diagnosis of pneumonia, but it must be remembered that it is not pathognomonic, for pleurisy with effusion or any serous exudate may produce almost the same effect.

(iii.) **Phosphates**. These may occur up to 5 grms. daily. Both the earthy phosphates of calcium and magnesium and the alkaline sodium and ammonium phosphates are found. Organic phosphates also occur in minute amounts.

The presence of phosphates is not of any special diagnostic

significance, though, of late, it has been found that some cases of neurasthenia are associated with a largely increased output, and in some of these the *phosphoric acid* is decreased while the output of calcium is greatly increased. These facts, if true, are of great interest in that appropriate treatment readily suggests itself.

Calcium phosphate is precipitated by heat and may be mistaken for albumin; the precipitate, however, at once disappears when a drop of acid is added.

(iv.) **Sulphates.** These are present to the extent of about 2·5 grms. daily in the urine. As their production depends on the metabolism of proteid matter they commonly bear a constant ratio to the nitrogen output.

Their most interesting aspect lies in the fact that a varying proportion of them (about one tenth normally) is excreted in combination with an aromatic base. The amount of these so-called ethereal sulphates is thought to furnish a direct index of the amount of *intestinal* decomposition that is taking place. *Gastric* disturbances do not seem to affect them: alkalies increase them, while acids and intestinal antiseptics, except possibly carbolic acid, diminish their production.

The procedure for the estimation of ethereal sulphates in the urine is chemically too technical to be described here; but an idea as to whether they are increased or not can be formed by the consideration of the urinary pigment indoxyl sulphate, or indican as it is sometimes called.

The presence of this body can be shown by Jaffé's test. Any albumin that is present must be removed by boiling and filtering. Then into 10 cc. of urine is rapidly poured a like amount of concentrated hydrochloric acid; at the same time a drop of fresh concentrated bleaching powder is added (the $Ca(OCl)_2$ may be placed on the lip of the tube containing the HCl and carried with the latter as it is poured into the urine), and the whole mixed together by turning up the test tube. If now 10 to 12 drops of chloroform are added, the indigo blue that is formed if indoxyl sulphate is present is dissolved.

It is essential that too much hypochlorite is not used, as this will convert the indigo blue into yellow isatin.

The depth of the blue colour imparted to the chloroform provides a sufficient index, for ordinary purposes, to the amount of indican present. With this test potassium iodide, if present in the urine, yields a deep carmine colour.

III. ABNORMAL URINARY CONSTITUENTS.

I. Bile Pigments and Acids. (*a*) **Bile Pigments.** Bilirubin, hydrobilirubin, biliverdin, etc., are found in the urine in cases of jaundice, though it is probable that only bilirubin is present in freshly-voided urine, the others being formed by oxidation after standing.

Gmelin's test is sufficient in most cases, but is fallacious if there is much indican present, and also if antipyrin is being taken by the patient. To perform this test run about an inch of urine on to a like amount of *crude* nitric acid in a test tube. If bilirubin is present the following rings of colour will be seen in the urine—from above downwards green, blue, violet, red, and yellow. The green ring is the most important and is sufficient for diagnosis.

An even more delicate test is to run on to some of the urine, acidified with acetic acid, a layer of a 1 per cent. solution of iodine in alcohol. If bile is present a green ring occurs in less than a minute.

(*b*) **Bile Acids.** These are often present in the urine of jaundiced persons, and may be tested for as follows—Add 1 drop of ·1 per cent. watery furfurol to 1 cc. of the suspected urine and super-impose this on 1 cc. of concentrated sulphuric acid, cooling the while; a red colour is formed if bile acid is present, which deepens in colour on standing.

The property of bile salts of lowering the surface tension of fluids is utilised in Hay's test, which is said to be positive in so weak a dilution as 1 in 100,000. The test is very simple:—Sprinkle on to the *cooled* urine a little finely-powdered sulphur, if this sinks in one minute bile salts are present in the proportion of 1 : 40,000.

II. Proteids. The urine may contain albumins, globulins, albumoses, or mucein. Any of these except mucein must

be regarded as pathological until they have been thoroughly investigated (*vide infra*).

Mucein is found in two forms—an insoluble form, which occurs as a cloud in the upper layers of a urine that has been standing, and a soluble form, which is precipitated by a drop of acetic acid, only to dissove in excess.

It has no special significance, but an excess would indicate a catarrh of the urinary tract.

Albumin. The presence of albumin is one of the leading symptoms of nephritis and the coincident appearance of casts is highly suggestive of a definite renal lesion.

The albumin is derived from the serum albumin of the blood, and the fact that it is so much more common than serum globulin can be explained by its much smaller molecule. For a similar reason fibrinogen is hardly ever found in urine ; when it is present the urine solidifies on standing.

Clinical Albuminuria may occur in the following circumstances other than serious organic renal change :—

(i.) Contamination from vaginal secretion : the likelihood of this renders imperative the catheterisation of any female suspected of albuminuria. Similarly prostatic or urethral disease must be considered in the male.

(ii.) After strenuous muscular endeavour a large percentage of athletes have a transient albuminuria. This lasts for three to four hours, and not infrequently is accompanied by a few granular casts and red blood cells.

Although this does not appear to lead to any permanent renal disability, nevertheless we cannot but feel that for the time being the kidney is undoubtedly in a pathological condition.

A similar albuminuria is sometimes seen after prolonged mental strain such as sitting for the I.C.S. examination.

(iii.) Cold baths, if sufficiently prolonged, induce a slight and transient albuminuria.

(iv.) For the first ten days of life albuminuria is common.

(v.) Pregnant women frequently have albuminuria during the latter part of the pregnancy.

(vi.) The essential albuminuria of Posner. This includes :—

A. Albuminuria of adolescence, in which there is apparently a slight renal insufficiency at puberty and for a few years after, but which disappears completely as maturity proceeds.

B. Postural albuminuria, in which the urine is free from albumin only while a horizontal position is maintained. As a rule no other evidence of disease can be discovered, but sometimes there will be a movable kidney, definite neurasthenia, etc.; and a certain proportion of cases of progressive nephritis first manifest themselves in this way. Granular casts are present, but in very scanty numbers.

C. About 2 per cent. of all people show a slight albuminuria. When this is not evidence of commencing nephritis it can perhaps be considered as a congenital abnormality in the glomerular or tubal epithelium, whereby a slightly increased amount of albumin to the normal is permitted to escape.

All urines contain a minute trace of albumin, but not sufficient to be detected by the *ordinary* chemical tests.

(vii.) The albuminuria of fevers and of cardiac insufficiency. These, of course, are pathological, but, provided the cause be removed, the kidney seems to exhibit perfect recuperative powers.

In conclusion we would emphasise the fact that albuminuria is only a symptom and that the amount of albumin present may serve as an idea of the importance of the renal lesion, but that in no case does the organism suffer directly from the loss of the albumin passed in the urine.

Tests for the Presence of Albumin. To examine urine for albumin it is best to test samples passed at different times of the day in order to exclude the postural or cyclical forms of albuminuria, but if only one examination is to be made, then take the urine passed at the close of the day's work and examine it as fresh as possible. If the urine is cloudy it should be filtered, and if concentrated it should be diluted until its specific gravity is less than 1,010, for albumin may easily be missed if the urine is of very high specific gravity.

For clinical purposes the two following tests are sufficiently accurate, and both should be performed, each as a control on the other :—

(i.) *Heat and Acetic Acid.* Fill a test tube three-quarters full with the filtered and, if necessary, diluted urine ; hold the tube by the bottom and heat the upper part of the column of urine in a spirit flame (gas flames are apt to form a condensation on the wall of the tube).

Albumin is precipitated by heat, and even very faint clouds may be detected if the tube is held against a black background.

Whether there is a precipitate or not, 2 or 3 drops of 20 per cent. acetic acid should be added to the urine after heating, for calcium phosphate and calcium carbonate are both precipitated by heat as well as albumin. If, however, the precipitate is albumin, it will be unchanged, or even increased, by the acid, whereas the phosphates and carbonates will disappear, the latter with effervescence. Apart from this it is essential to add the acid, because the original urine may not have been acid enough for precipitation owing to the presence of albumin in the form of alkali-albumin, and no cloud may appear until the acetic acid is added.

It is well to add the acid drop by drop and to boil the top of the urine again between each drop.

Very occasionally the urine may be too acid to permit the precipitation of the albumin owing to the presence of albumin in the form of acid-albumin ; a drop of caustic soda should then be added.

On no account should *too much* acid be added, as soluble acid-albumin may be produced. Hence it is not always possible to be sure that a cloud formed by heat which disappears with acid is due to phosphates ; but the minute trace of acid necessary to remove the phosphates makes this objection theoretical rather than practical, and in our opinion the above test is the best of all for the estimation of the presence of albumin.

Nucleo-albumin will be thrown down by the above test ; it is, however, also precipitated by acetic acid in the cold : it is not a normal constituent of the urine, and is most likely

to occur in those conditions in which nucleo-proteid proper is present as well. If any doubt exists on this point Heller's test (*vide infra*) will settle the question.

(ii.) *Heller's Test.* Place about 1 inch of *colourless* concentrated nitric acid into a test tube ; run gently on to this from a pipette a like amount of the filtered *and diluted* urine, being very careful not to mix the two fluids. If there is albuminuria a white ring will be formed *at the junction* of the urine and the acid. If no ring has formed within three minutes, there is less than ·003 per cent. of albumin.

The following fallacies must be borne in mind :—

A. *Urates.* These will be precipitated from all concentrated urines ; the ring, however, is broader than the albumin ring and is well above the line of contact. It is not formed in diluted urine and disappears on heating.

B. *Urea Nitrate.* This is a solid crystalline crust and should not cause confusion. It does not form in diluted urine.

C. *Nucleo-Albumin.* This ring is rather above the line of contact, is slightly opalescent, and is dissolved on shaking the tube so as gently to mix the two fluids.

D. *Albumoses* are precipitated at the line of contact ; they, however, disappear on heating.

E. *Resinous Acids,* in those who are taking the oleo-balsams, give a dense ring which partly dissolves on warming and is completely soluble in excess of ether.

Of the two tests just described the heat and acetic acid test is the more delicate, but both should be performed in each case, and if proper care in the technique is observed, there is little chance of error.

(iii.) *Quantitative Determination of Albumin.* The use of *Esbach's tubes* is sufficiently reliable for most clinical purposes, though really there is a very large margin of error. The only accurate method is to weigh the precipitate.

In using Esbach's method four essential points are of prime importance if the optimum result is desired :—

(*a*) Dilute the urine till its specific gravity is below 1,008.

(*b*) Acidify the urine with acetic acid.

(*c*) Always perform the test in a room kept at approximately the same temperature.

(*d*) If the reading is above 4 grms. per litre, do the test again with a weaker dilution of urine until the reading is less than this amount. Of course the dilution must be allowed for in the final calculation.

To perform the test. Fill one of the tubes up to the mark U with the properly diluted and acidified urine and then add the reagent till the mark R is reached : now insert the cork and invert the tube three or four times (avoiding shaking) till the fluids are mixed ; allow the tube to stand for exactly twenty-four hours in a constant temperature and read the height of the precipitate against the scale on the tube. This scale is marked to read grammes of albumin per litre. The original dilution of the urine must finally be allowed for.

Serum Globulin. The significance of an increased proportion of globulin to albumin in nephritis is not established ; our view is that the more acute the lesion the larger the proportion of globulin.

Globulins are precipitated by the addition to the urine of an equal amount of saturated sulphate solution.

Since they are also insoluble in distilled water, a cloud will be formed if a few drops of globulin-containing urine be thrown into a beaker of distilled water.

Albumoses may be found in the urine accompanying the albumin of an acute, or less commonly a chronic, tubal nephritis ; they may also occur when there is resorption of some extensive exudate, as in pneumonia, after the ingestion of unusually large amounts of albumoses, in phosphorus poisoning, in gangrene, in cancer, and even in insanity. Albumosuria is thought by some to be especially common in syphilitic nephritis and to be induced by a smaller intake of albumose if there is gastric or intestinal ulceration.

The best test for albumose is to saturate the urine with ammonium sulphate, when a flocculent white precipitate is thrown down. The urine must first be freed from albumin by adding an excess of sodium acetate and concentrated ferric chloride, neutralising or leaving faintly acid, boiling and, finally, filtering (Hofmeister).

The diagnostic value of albumosuria is very limited.

Bence-Jones' Body. The chemical identity of this body has not yet finally been established ; it is closely allied to the albumoses and, according to some workers, even more nearly related to albumin.

The occurrence of Bence-Jones' proteid in the urine is of the utmost rarity, and seems practically to be limited to cases of multiple myelomatosis.

The tests for this body are as follows :—

(i.) On heating the acidified urine a precipitate is thrown down at about 60° C., which dissolves when a higher temperature is reached.

(ii.) Heller's test with nitric acid gives a yellowish ring which disappears on warming.

(iii.) In common with the true albumoses, a Biuret reaction (a rose-pink colour with a trace of copper sulphate and excess of strong caustic potash) can be obtained by Bence-Jones' body.

III. Blood and its Derivatives. **A. Hæmaturia.** Blood in the urine may be a symptom of the following conditions :—

(i.) Acute nephritis, whether primary or following the ingestion of such poisons as turpentine or cantharides, or chronic hæmorrhagic nephritis.

(ii.) New growth of the kidney, ureter, bladder, prostate, or urethra.

(iii.) Calculus of the kidney, bladder, or prostate.

(iv.) Tuberculosis of the kidney or bladder.

(v.) Filaria, bilharzia, or even echinococcus.

(vi.) Infarction of the kidney, and more rarely venous thrombosis.

(vii.) Hæmorrhagic forms of the exanthems, especially scarlet fever, measles, small pox, enteric fever.

(viii.) Purpura (especially Henoch's purpura), scurvy and hæmophilia.

(ix.) Malaria.

(x.) Renal epistaxis, which is hæmorrhage, usually, from one kidney, intermittent, and associated with no histological lesion in those cases in which the kidney has been removed.

(xi.) Pernicious anæmia and leukæmia (rare).

(xii.) Trauma, as from faulty catheterisation.

If the blood comes from the kidney, it is intimately mixed with the urine and clots are unlikely ; if from the bladder, the urine passed at the end of micturition contains most blood, and clots are frequently present. Cystoscopy should be employed to determine the source of hæmorrhage in doubtful cases.

If the colour of the urine is not conclusive the following tests may be employed to demonstrate the presence of blood in the urine :—

(*a*) *The Microscope.* If the urine is allowed to settle and a drop of the deposit examined fresh with a $\frac{1}{6}$ inch objective, the red blood cells will at once be seen. The presence of white cells is of course insufficient, as they may be pus cells or on the way to become such.

(*b*) *The Guaiacum and Ozonic Ether Test.* If necessary the urine is acidified with acetic acid and a few drops of a 20 per cent. solution of guaicum resin in alcohol added ; this mixture is boiled and when cool overlaid with some ozonic ether. A blue colour is imparted to the lower part of the ozonic ether if blood is present.

This test is extremely delicate, and if it is negative blood may be assumed to be absent. Certain other organic substances may give a positive reaction, and if the boiling is omitted pus will be found a fruitful source of error (cf. p. 331, " Occult Blood Test ").

(*c*) *The Spectroscope.* If the blood is fresh, the spectrum of oxyhæmoglobin will be obtained. In nephritis, however, and in hæmoglobinuria the spectrum of methæmoglobin is more likely.

B. Hæmoglobinuria. The presence of hæmoglobin in the urine is due to the destruction of red blood cells with liberation of hæmoglobin. It may occur from the following causes :—

(i.) Blackwater fever : here it is not certain whether it is the result of malaria, or of excessive quinine, or both (*vide* p. 131).

(ii.) Certain poisonous materials, such as quinine, potassium chlorate, carbon-monoxide, arsenuretted hydrogen, etc.

(iii.) The malignant form of certain exanthems, as scarlet fever.

(iv.) As a rare manifestation of "serum sickness."

(v.) After burns or prolonged exposure to cold.

(vi.) As a vasometer disturbance associated sometimes with Raynaud's disease.

The presence of hæmoglobin may be demonstrated as follows :—

(*a*) By the guaiacum and ozonic ether reaction being positive and yet no blood cells being shown microscopically.

(*b*) By the spectroscope. A spectrum, sometimes mixed, of oxy- reduced and methæmoglobin is obtained, but usually the spectrum of methæmoglobin predominates.

N.B. If bacteria are present oxidation will result in the production of the spectrum of pure oxyhæmoglobin.

(*c*) The greenish-black colour of the urine.

IV. Pyuria. Suppuration anywhere in the urinary tract will result in the appearance of pus in the urine ; but a few pus cells are commonly present in tubal nephritis, and, of course, whenever blood is present leucocytes will also be found.

Urine which contains pus is usually alkaline, but a few bacilli, such as Bacillus tuberculosus, Bacillus coli and the gonococcus, cause pus in acid urine. If an equal volume of strong caustic potash solution is added to urine which contains pus a sticky gelatinous mixture results.

Perhaps the simplest test for pus is to examine microscopically some of the deposit after centrifugalisation ; the pus cells will readily be recognised, though any nucleus may be unrecognisable unless acetic acid is added. The more alkaline the urine the more swollen and glassy do the pus cells appear.

Both pus and blood give positive albumin tests, and Posner has estimated that ·1 per cent. of albumin is produced by 50,000 leucocytes per cubic centimetre of shaken urine.

Pus cells should not be mistaken for epithelial cells ; the different shape and size of the cells and the different appearance of the nucleus (if this is visible it is usually poly-

morphous in pus cells and is not vacuolated) should prevent mistake.

V. Glycosuria. In normal urine rather more than 2 grms. of carbohydrate are excreted daily in the form of glucose, glycuronic acid, chondroitin-sulphuric acid, maltose, etc. Every individual has a toleration point for sugar ingestion beyond which clinical glycosuria results. As an average 150 grms. of sugar can be taken without an appreciable amount appearing in the urine.

In certain diseases, notably cirrhosis of the liver and acute infectious diseases, the toleration point is low; while at all times milk-sugar is less well tolerated than dextrose or cane-sugar.

If glycosuria results from the ingestion of starch it is probable that true diabetes is present. Temporary glycosuria in cases of extreme malnutrition and starvation is common.

Glycosuria recognisable by the ordinary tests is the result of the presence of more than ·2 per cent. of sugar in the blood.

In phloridzin diabetes, but in no other case, the glycosuria occurs with a hypo-glycæmia (less than ·2 per cent. of sugar in the circulating blood), and is caused by direct injury of the renal epithelium. In addition to phloridzin and starvation glycosuria, sugar may occur in the urine in the following conditions :—

(i.) Diabetes mellitus.

(ii.) Certain head injuries or diseases of the brain (especially in the neighbourhood of the fourth ventricle).

(iii.) Arterio-sclerosis and certain affections of the liver.

(iv.) After the ingestion of large amounts of sugar or starch.

(v.) In pancreatitis.

(vi.) After anæsthesia by chloroform.

(vii.) Certain cases of disease of the supra-renal bodies associated with hypersecretion of adrenalin.

(viii.) A few cases of Exophthalmic Goitre.

(ix.) Certain cases of Hyper-pituitarism.

(x.) In nursing women (Lactosuria).

The presence of sugar in the urine may be shown in the following ways :—

(1) *The Reduction of Fehling's Solution.*[1] It is essential to use freshly-prepared Fehling's solution. It is best therefore to mix equal volumes of Fehling A and Fehling B in a test tube and boil ; a few drops of urine are then added and the mixture brought to the boil again ; if sugar is present to the amount of ·3 per cent. a yellowish-red precipitate of cuprous oxide is thrown down.

The following points should be remembered :—

(*a*) Never add more urine than half the amount of mixed Fehling's solution.

(*b*) Avoid prolonged boiling.

(*c*) Suspect a precipitate that only appears on standing.

(*d*) Remove albumin before doing the test if more than a trace is present, as this hinders precipitation.

(*e*) Dilute the urine, if it is of very high specific gravity, till the specific gravity is about 1,010.

(*f*) Remember that excess of urates may cause a positive reaction, as may glycuronic acid compounds, pyrocatechin, or the intake of the following drugs :— Camphor, chloroform, chloral, morphia, phenol, menthol, salicylic acid, benzoic acid, rhubarb, copaiba, sulphonal, santonin, etc. These fallacies are minimised by dilution of the urine.

(2) *The Fermentation Test.* A small piece of yeast is placed in the urine, which is then gently shaken and poured into a fermentation tube.

If gas is formed it may be assumed that a sugar containing three or a multiple of three carbon atoms is present.

Two controls should be put up, one of a urine which is known to contain glucose and one of a known normal urine. The former proves that the yeast is active and the latter that there is not auto-fermentation of the yeast.

(3) *The Polariscope.* With this instrument use is made of the fact that glucose rotates the plane of polarised light to the right. Albumin is slightly lævo-rotatory and

[1] Fehling A = Copper Sulphate, 34·639 grms.; Distilled Water, 200 cc. Fehling B = Rochelle Salt, 173 grms.; Caustic Soda (14 per cent.), 600 cc.

should therefore be removed. Glycuronic acid is lævotatory, as, of course, is lævulose.

If cloudy the urine should be cleared by filtration after the addition of lead acetate.

(Each of these three methods is of value, and No. 2 or No. 3 may well be used as controls on the first. From a clinical point of view the fermentation test is easy, free from fallacy, and therefore probably the best for the practitioner.)

(4) *Quantitative Estimation of Sugar.* For the quantitative estimation of sugar either of the three tests already described may be used, but with none is it very easy to ensure great accuracy. If a precise determination be required it is best to weigh the precipitate thrown down by Fehling's test on a chemical balance after drying.

For ordinary purposes it is enough to know that 10 cc. of Fehling's solution is reduced by ·05 grm. of dextrose and to discover how much urine (added drop by drop from a burette after free dilution) is required to decolorise 10 cc. of the boiling solution. From this the calculation of the sugar percentage is simple, and, if the amount of urine passed in the twenty-four hours is known, the daily output of sugar can be estimated.

Many authorities prefer to use Pavy's solution [1] to simple Fehling for the quantitative estimation of sugar in the urine. The advantage is that the presence of strong ammonia prevents the formation of any precipitate, so that the end-point of the reaction is indicated by the disappearance of the blue colour from the solution. It is important to remember that 10 cc. of Pavy's solution are only equal to ·005 grm. of glucose.

To estimate the amount of sugar by the fermentation test an Einhorn saccharometer is necessary. A piece of yeast the size of a bean is put into the urine, to which a little tartaric acid has been added to prevent alkalisation, and the saccharometer filled with the mixture. This is allowed to stand for twenty-four hours at a temperature of 20° to 40° C.

[1] Pavy's solution = Copper sulphate 4·158 grms.
Rochelle salt 20·4 grms.
Caustic potash 2·4 grms
Strong ammonia 300 cc.
Water to 1,000 cc.

and the volume of gas then read off on the scale, which is so graduated as to read the equivalent percentage of sugar.

As fermentation proceeds the specific gravity of the urine becomes lowered. This fact can be utilised to give a rough idea of the amount of sugar present. If a piece of yeast is placed in a jar of urine and the jar allowed to stand for twenty-four hours, then each degree of specific gravity lost by the urine is roughly equivalent to 2 per cent. of glucose.

The amount of gas evolved depends considerably on the activity and to a small extent on the amount of the yeast used; hence this method is not conducive to great accuracy.

If the polarimeter is used for the quantitative estimation of sugar it is essential to clear the urine and to free it from albumin. The instrument is filled with the urine and rotated until equal illumination is observed on each half of the field. The amount of rotation is now read on the scale and the amount of sugar calculated by means of the tables supplied with the instrument. Thus a 100 per cent. solution of glucose has a rotatory power of 52·5° to the right, while lævulose of similar percentage strength rotates the plane of the light through 93·8° to the left.

In filling the urine tube care must be taken to exclude air bubbles.

Normal urine is slightly lævo-rotatory.

VI. Acetone in the Urine. This body is present in small amounts in the normal urine; it may be found increased in cases of ordinary starvation, during the treatment (by starvation) of gastric ulcer, in cases of cyclical vomiting, in the terminal stages of any profound cachexia, in auto-intoxication, in chloroform poisoning, and especially in the more advanced stages of diabetes mellitus, when there is insufficient alkali circulating to neutralise the acid products of abnormal metabolism (probably fat metabolism). Its appearance in the urine of diabetics possesses therefore, as a rule, a certain grave significance. It imparts to the urine a sweetish odour as of apples, and this smell may generally be observed in the breath of the patient at the same time.

In the case of diabetes the presence of acetone is scarcely so important as the presence of diacetic acid, since if diacetic acid is present acetone is bound to be there also, and the test

for diacetic acid is easy, whereas some of the tests for acetone are very unsatisfactory; further, the clinical significance of diacetic acid in diabetes is greater than that of acetone.

At the same time there are conditions when acetone is

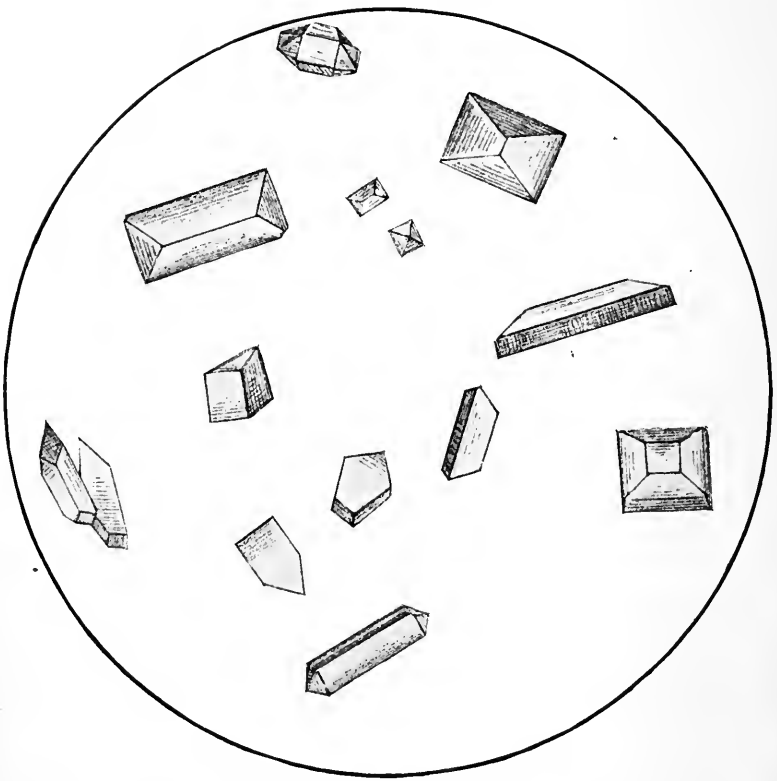


Fig. 55.—Crystals of Ammonio-Magnesium Phosphate. Magnification × 216.

present and in which diacetic acid is not found; therefore a reliable test for acetone is important.

The only test for acetone described here is Rothera's; it is easy of application, accurate and free from fallacies.

Rothera's Test (Garrod's modification). Add 5 cc. of a saturated solution of ammonium sulphate to 5 cc. of the urine; next add 3 drops of *freshly-prepared* saturated sodium nitro-prusside solution, and lastly add 2 cc. of ammonia.

Acetone gives a slowly-developing permanganate colour which is quite unmistakable and does not seem to be given by other substances.

VII. Diacetic Acid. As stated above, this body is more important than acetone and is produced under similar circumstances.

It is readily tested for by Gerhardt's test :—Add to some of the suspected urine drop by drop a solution of ferric chloride until precipitation ceases ; filter and add to the filtrate a few more drops of the ferric chloride : a claret-coloured solution will result if diacetic acid be present.

The fallacies in this test are that the colour will be produced by salicylates, antipyrin, and a few other bodies ; but in these cases the colour still appears after boiling the urine, whereas diacetic acid is decomposed and evaporated by boiling, so that the test is then negative.

VIII. Oxybutyric Acid. Just as diacetic acid is the parent substance of acetone, so is oxybutyric acid the precursor of diacetic acid. Its presence is hard to prove because of its ready dissociation into diacetic acid and acetone, but it is probably the all-important factor as indicating the onset of diabetic coma.

This acid is lævo-rotatory, and its presence may be guessed at if the urine *after fermentation* be found to be more definitely lævo-rotatory than normal urine.

IV. URINARY DEPOSITS.

For the purpose of examining urinary deposits it is desirable to obtain the urine as fresh as possible, while a centrifuge greatly facilitates the work. A few drops of the deposit after centrifugalisation or standing in a conical glass are sucked into a clean pipette and expelled from this on to a glass slide. A cover-slip may now be superimposed and the preparation examined with a $\frac{2}{3}$ inch and $\frac{1}{6}$ inch objective.

Crystalline Deposits.

(i.) *Phosphates* (Fig. 55). The white deposit of phosphates dissolves at once if acid is added ; this distinguishes it from pus. Phosphates are only deposited in alkaline or very weakly acid urine. Their microscopical appearances are variable,

but the most usual is the coffin-lid or pyramidal shape of ammonio-magnesium phosphate (triple phosphates). Calcium phosphate is of more rare occurrence, and is usually composed of wedge-shape masses radiating in all directions from a common centre.

(ii.) *Oxalates* (Fig. 56). The clinical significance of oxaluria

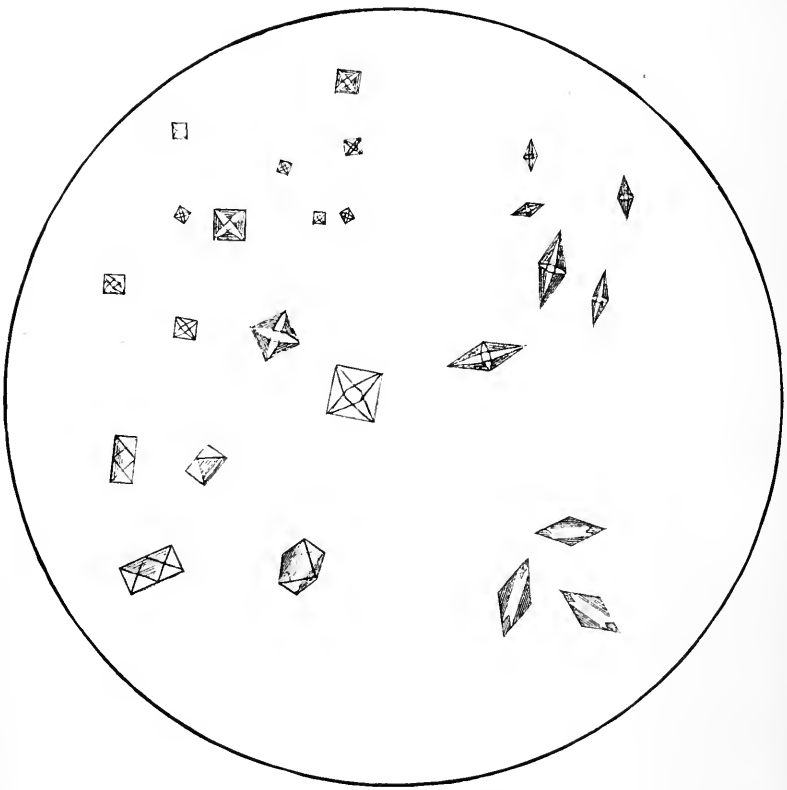


Fig. 56.—Crystals of Oxalate of Lime. Magnification × 216.

is very doubtful, but in so far as calcium oxalate is an important factor in renal calculi the persistent and excessive appearance of these crystals in the urine may be of some importance. An excessive number of these crystals may also give rise to a slight albuminuria. Their frequency in neurasthenia is suggestive.

Oxalate crystals may be found in both acid and alkaline urine; their usual shape is that of definite octahedra (envelope shape), but they are often seen as spheres or ovals, either grooved or showing radial striations.

(iii.) *Sulphates* are only precipitated from very acid urines; they usually take the form of clusters of thin needles or tablets.

(iv.) *Cystin* (Fig. 57). Cystinuria is a rare familial condition

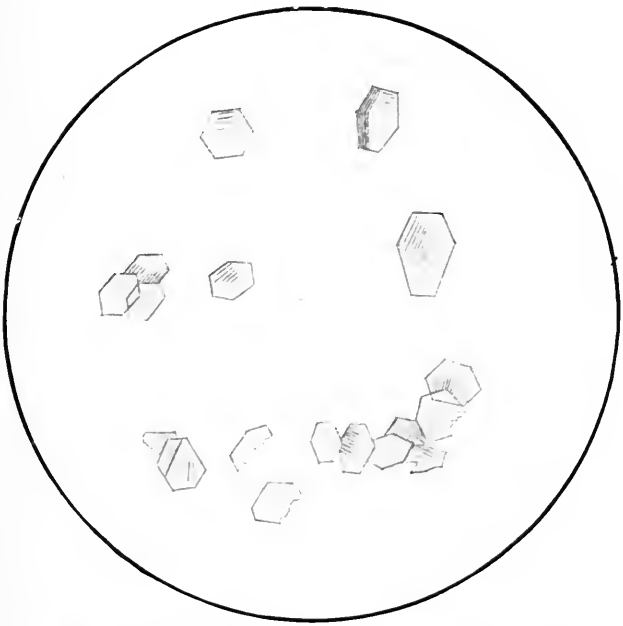


Fig. 57.—Crystals of Cystin. Magnification × 216.

and does not appear to be of pathological import. The crystals are beautiful hexagonal plates.

(v.) *Cholesterin* (Fig. 58) is but rarely found in the urine, though it has been noted in cases of chronic catarrhal cystitis. The characteristic appearance is that of flat, square or oblong crystals with one corner chipped out.

(vi.) *Uric Acid* (Fig. 59). These crystals are recognised by their yellowish-brown colour. Their most usual shape is that of whetstones, from disappearance of the corners of

the original rhombic forms. They may also look like barrels or needles, and sometimes greatly resemble cystin.

(vii.) *Urates* (Fig. 60) are not usually crystalline in appearance, being an amorphous mass. The fact that they disappear with heat serves for their ready recognition. They settle as a pink mass when the urine cools, but in the urine of children they are usually white.

(viii.) *Leucin and Tyrosin* (Fig. 61) are practically diagnostic

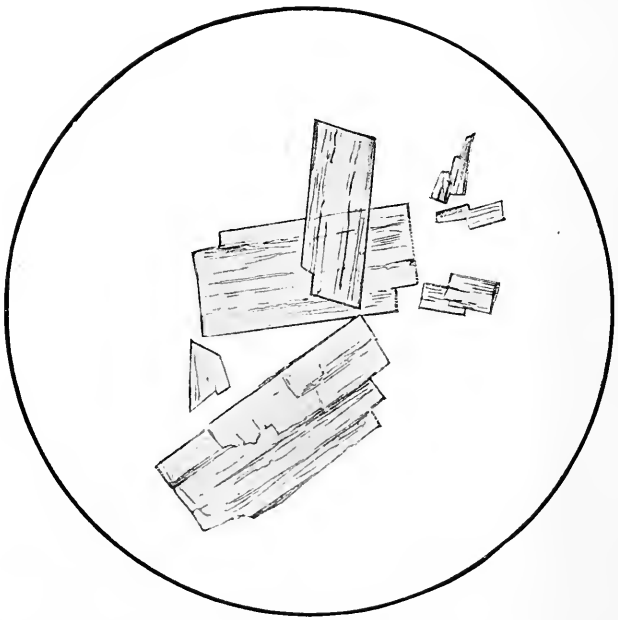


Fig. 58.—Crystals of Cholesterin. Magnification × 216.

of either acute yellow atrophy or phosphorus poisoning. When present leucin consists of small, clear-cut spheres, while tyrosin is likened to sheaves of wheat.

Non-Crystalline Deposits. (i.) Cells. Three types of cells may be found in urine :—

(*a*) *Epithelial Cells.* Renal epithelium cannot with certainty be distinguished from bladder epithelium, though the kidney cells are cubical and tend to show a tail-like process at one end, while the bladder cells are larger, more flattened, and of more irregular shape.

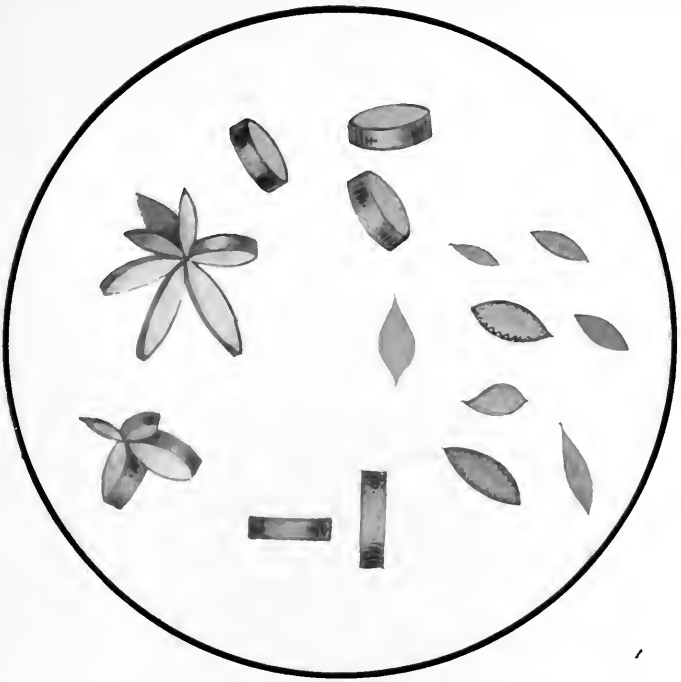


Fig. 59. — Crystals of Uric Acid. Magnification 216.

Fig. 60. — Crystals of Ammonium Urate. Magnification × 216.

The nuclei of kidney cells are large and vesicular, while those of bladder cells are small and highly refractile.

Urethral cells are cylindrical or transitional. Squamous cells indicate a vaginal or preputial origin.

(*b*) *Red Blood Cells.* Red blood cells indicate hæmaturia, the causes of which have been considered on p. 432.

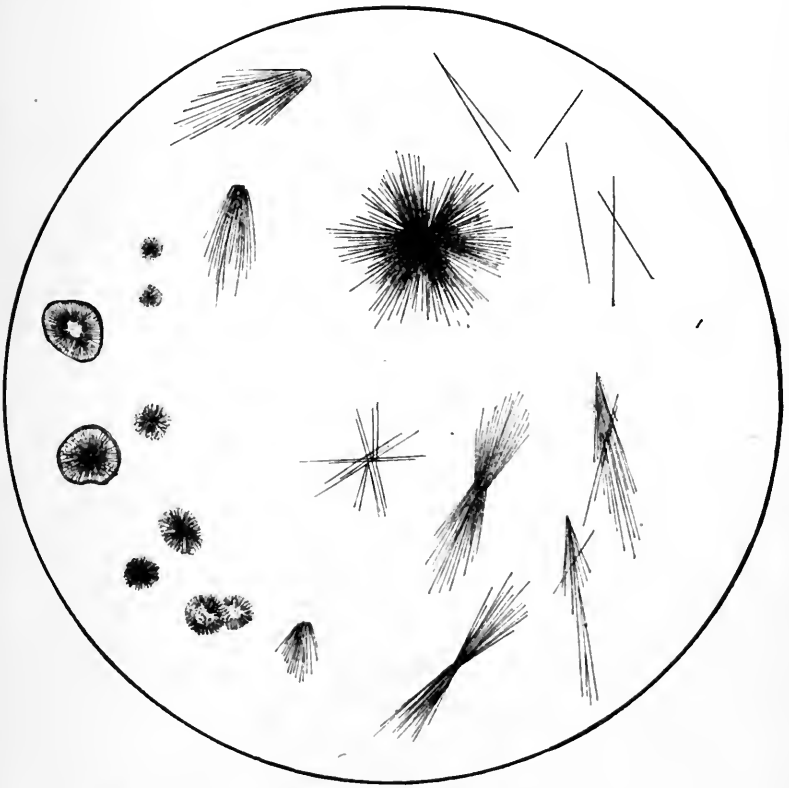


FIG. 61.—Crystals of Leucin and Tyrosin. Magnification × 216

(*c*) *White Blood Cells* may be present in their normal proportions to red blood cells in cases of hæmaturia; when, however, polymorphonuclear leucocytes are present in excess or without red blood cells they indicate pyuria. This may be produced by suppuration in any part of the urinary tract. Under such circumstances

a bacteriological examination is often advisable to clear up the diagnosis.

(ii.) Casts (Figs. 62 and 63). It is customary to think that the presence of casts in the urine necessarily indicates a true nephritis; this is not so, for casts may occur in any of the "so-called" physiological albuminurias, and even, when there is no albumin at all, in the urine of presumably healthy

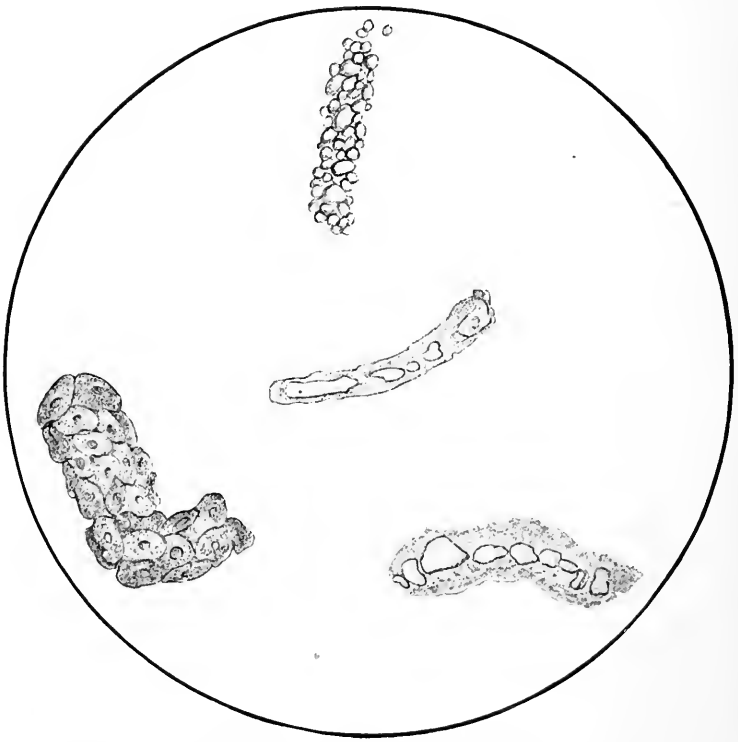


Fig. 62.—Epithelial and Fatty Casts. Magnification × 216.

persons. Hence it is probable that casts *need* only mean a temporary functional impairment of renal epithelium. Their continued presence, however, should certainly excite suspicion, especially if other signs of nephritis are present.

As a general rule it may be stated that the persistence of casts after the albumin has disappeared is suggestive of

a granular kidney, while the converse obtains in cases of parenchymatous inflammation.

In searching for casts the urine should be examined immediately after it is passed, for casts readily disappear

Fig 63.—Waxy Casts. Magnification × 216.

if the urine is allowed to stand for even a few hours before examination.

The procedure is the same as for all other urinary deposits, and no stain is necessary.

A cast, as its name implies, is a mould of part of a urinary tubule; as a rule comparatively short lengths only are to be seen. They are cylindrical masses the length of which is perhaps from three to six times their breadth. The surface

varies with the nature of the cast. The following types are described :—

(*a*) *Epithelial Casts.* These are formed of renal epithelium, probably shed piece-meal and passed *en masse*, though occasionally the epithelium of a length of tubule may be shed as a whole. The individual cells may be well preserved or in any stage of degeneration.

(*b*) *Fatty Casts.* These are epithelial casts the cells of which have undergone such extensive fatty metamorphosis as to leave no cell-bodies to be distinguished apart from the numerous fatty globules.

(*c*) *Granular Casts.* In these the epithelial cells have undergone granular degeneration.

(*d*) *Blood Casts* are suggestive of acute nephritis or of trauma to the kidney ; they are formed by the effusion of blood into the renal tubules.

(*e*) *Hyaline Casts.* These are pale, structureless casts of a very regular shape and *low* refractive index ; they are not necessarily indicative of any severe pathological change and must not be confused with the following.

(*f*) *Waxy Casts.* These are *highly refractile*, whitish, dense-looking casts, sometimes without structure, sometimes finely granular, and sometimes tending to split transversely.

They occur in acute or chronic nephritis, just as do epithelial, fatty, and granular casts.

The term " waxy " has no connection with lardaceous disease when applied to urinary casts.

Casts are most numerous in chronic parenchymatous nephritis, then in acute tubal nephritis, and fewest in chronic interstitial nephritis. The better the preservation of the epithelium forming a cast the more acute is the nephritis.

It is not easy to mistake any other structure for casts, but it is as well to mention the following possibilities :—

(1) *Cylindroids :* these are long, *tapering*, mucinous threads. Casts are much shorter than cylindroids and have blunt extremities.

(2) *Fragments of Thread, Wool, Linen,* etc. : these should not be present unless the vessel used for receiving the urine is dried with a duster. The student is advised

to familiarise himself with the microscopical appearances of such foreign bodies.

V. THE BACTERIOLOGICAL EXAMINATION OF THE URINE.

For this purpose catheter specimens of urine should be received into sterile vessels and examined as soon as possible. The urine should be examined directly, and cultures should also be made.

For the direct examination the urine may be diluted with an equal volume of alcohol, in order to lower its specific gravity and favour the deposit of the bacilli on centrifugalisation. After centrifuging the tube is rapidly inverted and emptied : a portion of the deposit is now transferred with a platinum loop to a sterile clean glass slide and spread into an even film ; this is allowed to dry in the air and fixed by passage through a Bunsen flame.

Some authorities advise that a drop of egg albumin be first placed on the slide, and this certainly prevents the loss of the film when it is washed. For ordinary purposes Löffler's methylene blue is the best stain ; it should be used cold and allowed to act for seven to ten minutes : the film is then well washed in tap-water, dried between filtre paper, and examined directly with an oil immersion lens.

It is well to make a second preparation stained by the method of Gram, and of course, if tuberculosis is suspected, carbol-fuchsin must be used, as described on p. 81.

The great value of a film examination lies first in the fact that it is the only method of quickly demonstrating the tubercle bacillus, and secondly that the morphological appearances of any bacilli that are present afford valuable information as to the proper culture media to employ for their further investigation.

Thus diplococci, such as gonococci or pneumococci, grow best on fresh blood-agar : the Bacillus coli and Bacillus typhosus grow well on ordinary nutrient agar, but may be differentiated by cultures in some such medium as McConkey's bile-salt-litmus-glucose solution. Staphylococci

should be planted on nutrient agar, while streptococci may be cultivated in broth. If no clue as to the identity of the organism can be gathered from the film examination, the best medium to use is blood-agar ; the colonies should be examined as they appear and subcultures made on appropriate media.

If tubercle bacilli are suspected but cannot be seen, some of the centrifugalised deposit may be injected into a guinea-pig, as described on p. 81.

The smegma bacillus possesses very similar staining reactions to the tubercle bacillus, except that it is more liable to decolorisation with alcohol than the latter. It should, however, be excluded if the utmost care and cleanliness are used in passing the catheter for the obtaining of the specimen.

In an ordinary alkaline cystitis, such as occurs behind a stricture of the urethra, in cases of paralysis and after repeated catheterisations, many organisms may be met with, such as staphylo- and strepto- cocci and various forms of bacillus.

If the urine is acid and yet contains bacilli, the Bacillus coli is the most probable organism, but the gonococcus, Bacillus typhosus, and the tubercle bacillus are also found in acid urine. The Bacillus coli may occur in large numbers in the urine without the presence of pus ; it often persists for many years after a pyelitis has quieted down. The tubercle bacillus and many other organisms also may occur without pus when there is a blood infection by the particular organism concerned : typhoid fever is a striking example of this fact.

The only parasites of importance in connection with the urine are the following :—

(i.) Bilharzia hæmatobium, which, in countries where it is endemic, is responsible for many cases of hæmaturia. The eggs of this creature are passed in the urine and may be recognised in the sediment as small oval eggs ·16 mm. in diameter, containing an indefinite embryo which is just visible through a transparent shell. Each egg has a spinous process at one end of it (*vide* p. 149).

(ii.) Echinococcus. Hydatid cysts are rare in connection

with the kidney. Hooklets and scolices may sometimes be found in the urine (*vide* p. 142).

(iii.) Filaria Sanguinis Hominum may cause chyluria and the embryos may be discovered in the chylous urine (*vide* p. 147).

CHAPTER III

DISEASES OF THE KIDNEY AND URINARY APPARATUS

I. NEPHRITIS.

The etiology of nephritis is still a matter of extreme obscurity, and until more accurate knowledge has been acquired of this most important subject it is best to adhere to the older classifications, which were based primarily on the morbid anatomy and histology of kidneys as seen in the post-mortem room. The affections are described as being either acute or chronic, and they are also distinguished according as to whether the morbid process has principally involved the renal parenchyma or the interstitial tissue. As a working basis the following table may be employed. Its limitations are many, for in all cases of nephritis both parenchymatous and interstitial tissue are involved, and therefore a clear-cut-type case is the exception rather than the rule.

Acute Nephritis	(1) Parenchymatous	Acute Tubal Nephritis.	
	(2) Interstitial	(*a*) Acute Glomerular Nephritis (scarlatinal).	
		(*b*) Acute Pyelonephritis from an ascending infection of the bladder (Surgical Kidney).	
Chronic Nephritis	(1) Chronic Parenchymatous Nephritis	(*a*) Large White Kidney.	
		(*b*) Large Mottled Kidney (hæmorrhagic).	
	(2) Chronic Interstitial Nephritis	(*a*) Red Granular Kidney.	
		(*b*) Arterio-sclerotic Kidney.	
		(*c*) White Granular Kidney.[1]	

[1] Since there is so pronounced an involvement of the parenchyma in the white granular kidney, it might with equal force be included in the group of chronic parenchymatous nephritis.

A. **Acute Tubal Nephritis.** This may occur during any specific infective disease, such as enteric fever or pneumonia, but it is commonly seen after a simple chill or exposure to wet. Another fertile cause is the ingestion of irritants, such as cantharides or turpentine. The disease may start suddenly, but an insidious onset is more common. There may be malaise and headache as well as an aching pain in the loins, but quite often these are absent, and the first thing noticed is puffiness of the face, hands and feet, or even of the whole body. In some cases hæmaturia, in others uræmic convulsions, may be the first sign. The temperature may be slightly raised at the outset, but it is often normal throughout. There is often some frequency of micturition at the outset, but the total amount of urine passed is diminished until convalescence is established, when there will be polyuria, which often persists for a considerable time. The urine contains blood, albumin, and casts (epithelial, granular, blood, and hyaline). The amount of urea is markedly diminished. The reaction is usually acid, unless a very large amount of blood is present and the specific gravity slightly raised, so long as albumin is abundant.

The blood pressure is moderately raised (140 to 160 mm. in an adult) and the aortic second sound is accentuated.

As the disease advances there is noticeable anæmia.

The following may be considered complications of acute nephritis :—

(i.) *Retinal Hæmorrhages.* These are rare, especially when compared with the frequency with which they occur in chronic forms of nephritis.

(ii.) *Amaurosis*, either with or without retinal œdema. The prognosis is good as regards recovery of the vision if the renal mischief is checked.

(iii.) *Suppression of Urine.* This rarely lasts for more than thirty-six to forty-eight hours provided suitable treatment (hot-air baths, warm packs, diaphoretics) is employed, but, if persistent, death occurs towards the middle of the second week from so-called "latent uræmia."

(iv.) *Serous Effusions.* These may form with the

utmost rapidity; hence a thorough daily examination is essential.

(v.) *Œdema of the Glottis* may occur in association with general anasarca or as a separate complication. Tracheotomy may be necessary.

(vi.) *Pericarditis* is often a terminal event, but is more common in chronic nephritis.

(vii.) *Cardiac Dilatation.*

(viii.) *Bronchitis.*

(ix.) *Uræmia* (*vide* special section, p. 458).

B. **Acute Glomerular Nephritis.** The description given above of acute tubal nephritis applies with equal force to the variety now under consideration, but scarlet fever is particularly prone to be complicated by an acute nephritis which primarily affects the glomerular apparatus.

The mortality of post-scarlatinal nephritis is high (20 to 30 per cent.). In other forms of acute nephritis the mortality seems to depend very largely on whether there is any pre-existing kidney lesion.

It should never be forgotten that acute or sub-acute exacerbations are especially liable to occur in kidneys already damaged by chronic tubal or interstitial change, and in these the outlook is correspondingly grave.

Every effort should therefore be made to discover whether the kidneys were healthy previous to a particular attack. For this purpose the history is most important, but much can be learned by examination of the heart and blood-vessels, for all chronic renal inflammations are inevitably accompanied by cardiac hypertrophy and increased blood pressure, greater than can be explained by a first attack of acute nephritis. The fundus oculi should also be examined for hæmorrhages, old or recent, for the more chronic the lesion the greater the likelihood of these being present.

The diagnosis of Acute Nephritis, whether *tubal* or *glomerular*, does not present many difficulties; the following sources of error may, however, be mentioned:—

(i.) The albuminuria of acute febrile conditions, especially of diphtheria.

(ii.) The albuminuria of chronic heart disease which is the result of passive congestion of the kidneys.

(iii.) Infarction of the Kidneys. An examination of the heart will reveal some lesion of the mitral or aortic valves, whilst the hæmaturia is not accompanied by renal œdema and is often preceded by a sharp pain in one or other loin.

(iv.) Lardaceous Disease. The presence of some possible focus, such as tuberculosis or sepsis, together with a *low* blood pressure and probably an enlarged liver and spleen, will suggest the true diagnosis.

(vi.) Renal Epistaxis. Here there is no œdema or cardiac hypertrophy, and cystoscopy will show that the blood is coming from only one ureter.

C. **Acute Pyelonephritis.** Chronic pyuria and cystitis (usually alkaline), together with an enlarged prostate or an old urethral stricture or paraplegia from any cause, will suggest the possibility of the infection, sooner or later, extending up the ureters to the kidneys. For a long time there may be no indication that this has happened, but eventually a fatal uræmia will develop (*vide* p. 458).

D. **Large White Kidney.** In some cases this condition may directly follow an attack of acute nephritis, especially scarlatinal nephritis. More often, we feel convinced, it arises quite independently and for no known cause. It progresses steadily to its appointed end, which is death. This is not often delayed for more than a year from the onset of symptoms, though this does not imply that there may not have been a much longer course *before* symptoms developed.

In the main the symptoms and signs are the same as in acute nephritis—headache, nausea, anæmia, œdema, and serous effusions. The large white face of the patient with a large white kidney has become an aphorism. There is more cardiac hypertrophy and a higher blood pressure than in acute nephritis, but neither need be extreme. The urine is scanty, and does not contain blood unless a subacute attack is superadded to the chronic affection. The urea is diminished and albumin is copious; casts are always present, especially the epithelial, fatty, and waxy varieties. The specific gravity is high unless the albumin is removed, when it is low owing to the diminution in total solids.

The complications are the same as for acute nephritis, and uræmia is the inevitable conclusion unless some intercurrent malady, such as bronchitis or pulmonary œdema, proves fatal first

E. **Chronic Hæmorrhagic Nephritis** (Large Mottled Kidney). The etiology, course, and prognosis of this variety are the same as in large white kidney. There would seem to be a greater tendency to hæmaturia and hæmorrhages beneath the skin and from the mucous membranes, and, post mortem, the appearances suggest a blend of acute and chronic tubal change. Possibly this variety is merely a large white kidney in which the acute exacerbations follow each other with unusual frequency.

F. **Red Granular Kidney.** Here we must pre-suppose the long-continued action of certain poisons or toxins which exercise a selective action on the renal arterial system causing a gradual peri-arterial fibrous tissue replacement and overgrowth. The effect of this is to diminish the area of functional renal tissue, with the result that it is necessary to force more blood than normal through the kidneys in order to maintain the proper elimination of the waste products of metabolism. Hence there will be a secondary and compensatory cardiac hypertrophy to meet the increased demand on the circulatory apparatus. The result will be a raised blood pressure and progressive arterial degeneration when the limit of hypertrophy of the tunica media has been reached. In like manner the heart muscle will in time become the seat of fibrous transformation and cardiac dilatation will ensue.

The toxins responsible for the inception of this vicious circle have not been identified, but, be that as it may, red granular kidney is predisposed to by gout, syphilis (here heredity must play a part), long-continued strain, both mental and physical, high living, alcohol, sexual excesses, lead poisoning, and the like.

If the clinical evidence of parenchymatous nephritis is fairly obvious the opposite is true of the red granular kidney, which is so insidious in its onset and progress as often to be unsuspected until its last phases, or even

until the autopsy. Attention may be drawn to the kidneys in the following ways :—

(i.) Symptoms immediately referable to the cardio-vascular system may develop and the hypertrophy of the heart will cause suspicion to fall on the kidneys.

(ii.) An attack of subacute or acute nephritis may occur.

(iii.) The onset of uræmic symptoms may be the first sign.

Not infrequently patients with red granular kidneys enjoy extremely good health till an advanced age and are able to lead active and vigorous lives.

Those patients who seek advice are likely to be over 40 years of age and to complain of increasing muscular weakness, with dyspnœa on exertion (often paroxysmal in type), headaches, noises in the head, insomnia, gastro-intestinal disturbances, and, more rarely, failing vision. Inquiry will elicit the fact that the urinary output is increased and that there is frequency of micturition, especially at night. The urine is pale, clear and of low specific gravity (1,003 to 1,012); sometimes there is a trace of albumin, but more often there is not, and from time to time a few granular casts are to be found. The urea output is diminished. The left ventricle of the heart is hypertrophied, the impulse is diffuse and heaving, and the aortic second sound is accentuated. The blood pressure is high (160 to 220 mm. of mercury) and the arteries are thickened and often tortuous. There is often a noticeable anæmia. Pigmentation of the skin may be so extreme as to simulate Addison's Disease.

The ophthalmoscope may show old or recent hæmorrhages, and in this connection it must be remembered that other hæmorrhages, such as cerebral hæmorrhage, petechiæ, or melæna, are far from rare.

Œdema is conspicuous by its absence unless heart failure with dilatation and tricuspid incompetence is present, when there will be the ordinary " cardiac " œdema into the dependent parts of the body. Occasionally, however, there is a very noticeable puffiness in the lower eyelids on rising in the morning, which passes off during the day, leaving baggy, wrinkled pouches below the eyes.

It is in red granular kidney that a clear understanding of

the many and diverse lesser manifestations of uræmia is essential for accurate diagnosis, since all the symptoms which are not referable to the cardio-vascular system have almost certainly an uræmic origin.

G. **The Arterio-sclerotic Kidney.** Any cause of arterial degeneration and persistent high blood pressure will sooner or later show itself by a fibrous tissue increase in the renal interstitia, and the arterio-sclerotic kidney is the result. Just as a red granular kidney causes a high blood pressure, so does a high blood pressure tend to cause a granular kidney, and, except from the point of view of morbid anatomy, the lesions are identical. Clinically no differences can be recognised other than those of degree.

H. **White Granular Kidney.** Very diverse opinions have from time to time been expressed regarding the etiology of this disease. Formerly it was taught that the large white kidney would, if the patient lived long enough, contract into the white granular form ; others deny that this ever takes place, and consider that the white granular kidney is always a distinct entity and never an advanced stage of a different type of nephritis.

Few persons of wide experience in morbid anatomy will deny that there are sometimes found at post-mortem examinations kidneys which present an intermediate appearance between the large and small white kidneys ; but such cases are rare, and, in our experience, the great majority of cases of white granular kidney are such from the outset, and it is quite exceptional for them to be preceded by a chronic tubal nephritis.

Histologically there is extensive change in both the interstitial and parenchymatous elements of these kidneys.

In investigating the records of patients dying from white granular kidneys the following features are noteworthy :—

(i.) The youthful age of the patient (15 to 35 years), the great bulk occurring in the third decade.

(ii.) The absence of previous scarlet fever in any greater frequency than in healthy people.

(iii.) The absence of œdema throughout the illness, unless there is cardiac failure or an incidental attack of acute nephritis (both of which are likely towards the end).

(iv.) The prevalence of albuminuric retinitis, which is more frequent than in any other form of nephritis.

(v.) The short course of the disease, with its fatal issue within two years of the first symptom.

The above rather suggests that these cases start in a similar manner to the red granular kidney, but that for some unexplained reason the particular toxins involve the parenchyma (especially the glomerular epithelium) as well as the interstitia, with the result that the renal efficiency is interfered with at a very early date and uræmia is ever at hand.

As would be expected, the onset is insidious, and the first thing complained of is often headache, general debility, or failing vision. Examination shows albuminuric retinitis, cardiac hypertrophy, a very high blood pressure (often over 200 mm.) and urine, which is abundant, of rather low specific gravity and containing a fair number of granular and fatty casts, a moderate amount of albumin (·3 per cent.), and diminished urea.

The progress is often terribly rapid, death occurring in the majority of cases from uræmia, though heart failure may sometimes be the predominant factor.

Pericarditis and dry pleurisy are common at the end, but serous effusions apart from heart failure are not frequently met with.

The diagnosis of white granular kidney presents no difficulty if a proper examination is made, but many of the cases are treated symptomatically for simple headache, and do not receive adequate investigation.

In conclusion we must emphasise the fact that apparently obvious cases of white granular kidney may be found to possess some other variety of lesion (sometimes even no very obvious lesion at all), and conversely cases which clinically suggest the presence of large white kidneys may, occasionally, at the autopsy prove to be due to the presence of white granular kidneys.

Morbid Anatomy and Histology of Nephritis.

In view of the fact that the classification of nephritis adopted in the preceding pages depends on the morbid

anatomy of the kidneys concerned, a short description of the chief naked-eye and microscopical appearances of the varieties of diseased kidney is, perhaps, desirable.

A. **Acute Tubal Nephritis**. (i.) *Naked Eye.* The kidney is large, rounded and congested ; the capsule strips easily, exposing dilated venules on the surface of the organ. On section the kidney drips blood ; the pyramids are deep purplish-red and stand out distinctly ; the cortex is ample in width and striated by fine red lines. The pelvis is natural.

(ii.) *Microscopically.* The vessels are engorged with blood. The tubal epithelium is swollen and cloudy, while in places it can be seen to have desquamated, so that the tubules, which also contain blood, appear to be choked up with cells.

B. **Acute Glomerular Nephritis**. (i.) *Naked Eye.* But little change may be visible ; in severe cases the kidney is large and engorged, the capsule strips readily and the glomeruli can be seen on the surface of the organ as bright injected dots. On section the cortex is abundant, the pyramids are distinct, and the whole kidney contains an excess of blood.

(ii.) *Microscopically.* The most striking feature is the small round-celled infiltration of the glomerular tuft, with dilatation of the blood-vessels and the presence of blood-cells and cellular débris within Bowman's capsule. The tubal epithelium may be secondarily involved.

C. **Acute Pyelonephritis** (the Surgical Kidney). (i.) *Naked Eye.* The kidney is often bulky and of a mottled yellow colour, and abscesses may be seen through the capsule, which usually strips fairly easily. On section the pelvis is acutely inflamed and often contains pus, whilst its size is increased from atrophy of kidney substance if back pressure has been a feature of the case. From the pelvis yellowish lines can be seen running up amongst the pyramids ; sometimes the abscesses are macroscopic, but frequently the microscope is needed for their detection. In early cases the inflammation may not have progressed as far as abscess formation. Occasionally the kidney is converted partially or completely into a bag of pus (pyo-nephrosis).

(ii.) *Microscopically.* The feature will be extensive round-

celled infiltration, peri-tubular and peri-vascular, with a varying number of necrotic areas of different sizes. The tubular epithelium is disorganised and many of the tubules are disintegrated. According to the duration of the disease so will there be more or less interstitial increase.

D. **Large White Kidney**. (i.) *Naked Eye.* The kidney is large, pale yellowish-white, and greasy-looking ; the capsule strips readily, and on the fatty surface stellate veins can be observed. On section the cortex is increased in depth and is sharply distinguished from the pinkish pyramids. The pelvis is natural.

(ii.) *Microscopically.* There is definite and diffuse increase in the interstitial tissue. The tubules are choked with the débris of fatty and granular epithelium. The vessels are moderately hypertrophied and are not engorged with blood.

E. **Chronic Hæmorrhagic Nephritis** (Large Mottled Kidney). (i.) *Naked Eye.* The kidney is large and mottled red and yellow in colour ; the capsule strips readily, and the kidney can be seen to be somewhat engorged with blood. On section the cortex is deep and the pyramids very distinct and of a much darker colour than in the large white kidney ; there is often a certain amount of cortical striation and the whole organ contains a good deal of blood.

(ii.) *Microscopically.* The appearances are those of the large white kidney, except that the vessels are engorged and there is often blood in the tubules.

F. **Red Granular Kidney**. (i.) *Naked Eye.* The kidney is small, red, shrunken and tough : cortical cysts are often visible through the capsule ; the capsule may be adherent and small fragments of renal cortex may be left sticking to it when it is stripped off, but in many cases the capsule strips quite easily if there has been no subacute nephritis as a terminal complication.

The surface of the kidney is very granular and often dotted with small cysts ; the granules are fine, somewhat resembling coarse sand-paper. On section the cortex is shrunken till the pyramids appear almost to reach the surface, and the excess of fibrous tissue has caused a loss of sharp differentiation between the pyramids and the rest of the organ. The pelvis always contains an excess of fat.

(ii.) *Microscopically.* The arteries are greatly thickened from hypertrophy of the media; wedge-shaped masses of fibrous tissue can be seen replacing the parenchyma throughout the organ, but between the fibrous wedges the kidney substance is relatively healthy. In the diseased areas the tubular epithelium is atrophied and lost, the tubules are obliterated or distorted into small cystic spaces, and the glomeruli are converted into fibrous masses both from pressure from without and also from becoming filled up by fibrous tissue laid down round the vascular tufts.

G. **Arteriosclerotic Kidney.** (i.) *Naked Eye.* This kidney is not necessarily smaller than the normal, but in all other respects it possesses the same characteristics as the red granular kidney.

(ii.) *Microscopically.* The changes are those of the red granular kidney, with the exception that they are not so intense in degree and tend to be more diffuse in distribution.

H. **White Granular Kidney.** (i.) *Naked Eye.* This kidney is small, pale yellowish-white, tough and shrunken; cortical cysts are often present. The capsule generally strips cleanly, but may be adherent in places. The surface is irregular, but the "granules" are very coarse and resemble rather irregular, lumpy projections than the coarse sandpaper of the red granular kidney. On section the appearances are identical with those of the red granular kidney except for the yellowish colour, which probably depends on fatty change in the tubules.

(ii.) *Microscopically.* The arteries are much thickened by hypertrophy of the media; the kidney substance is the seat of *diffuse* fibrous-tissue replacement. Such tubules as remain show epithelial proliferation, desquamation, and fatty change. The glomeruli are in time converted into fibrous masses, but this is accomplished as much by proliferation of the epithelium lining Bowman's capsules as by the methods described as occurring in the red granular kidney. This proliferation of the lining epithelium of the Bowman's capsules and subsequent conversion of the cells into fibrous tissue, together with the tubal changes, constitute the characteristic features of this form of nephritis.

II. DEGENERATIONS OF THE KIDNEY.

(*a*) **Lardaceous Disease.** It is rare for the kidney to be the only organ affected; the spleen and liver are nearly always involved as well. The causes are prolonged suppuration, especially without drainage, tuberculosis (especially of bone), and syphilis.

The symptoms are those of the primary cause plus persistent albuminuria; the urine is at first plentiful, but in the later stages it is diminished; casts (fatty and waxy) are present, but only in small numbers, and the amount of solids is diminished. Albuminoid casts—that is to say, casts giving the distinctive staining reactions of lardacein—have often been described, but, in our experience, they do not occur. The blood pressure is low and the cardio-vascular changes of true nephritis are wanting. It has been stated that persistent albuminuria with a low blood pressure is diagnostic of lardaceous kidneys, and certainly it is very suggestive; but there is no doubt that chronic tubal nephritis can occur in the subjects of wasting diseases, such as tuberculosis, cancer, etc., without the ordinary cardio-vascular sequelæ developing.

A lardaceous kidney is large, smooth, tough, rounded and bulky; the capsule strips readily. On section the rounded edges are striking, as is the smeary, par-boiled appearance of the organ, with lack of clear differentiation between its component parts. If a piece of lardaceous kidney be placed for a few minutes in a solution of iodine and then washed in water the lardaceous glomeruli will appear as mahogany-brown points.

Microscopically the chief feature will be the presence of structureless unstained glomerular patches, unless methyl violet is used as a stain, when lardaceous material stains a bright magenta colour.

The lardaceous matter itself is said to be a compound of chondroitin-sulphuric acid with an albumin; it is thought to be *formed locally* and is deposited as white crystalline plates in the adventitia of the arterioles. In the kidney the arteries to the glomeruli are the first to be affected.

(*b*) **Fatty Degeneration.** This condition is not likely to be

recognised clinically; it occurs in cases of prolonged toxic or septic absorption, after the ingestion of certain protoplasmic poisons, such as phosphorus, and in the course of the graver blood diseases, such as pernicious anæmia or leukæmia. It is possible that the large white kidney is really more a degeneration (fatty) than an inflammation.

(*c*) **Granular Degeneration** (cloudy swelling). This condition occurs temporarily whenever there is high fever; it is therefore a common manifestation of the acute specific infections. A slight albuminuria is sometimes found, but casts are not the rule. Complete recovery is usual, but if the primary cause is unduly prolonged fatty degeneration may ensue.

III. VASCULAR DISTURBANCES OF THE KIDNEYS.

(*a*) **Active Congestion**. This must occur as the result of irritation, whether direct or reflex, and is best seen in the early stages of acute nephritis, to which condition the symptoms, if any, will point.

(*b*) **Passive Congestion**. This is produced in its most typical form by heart failure in mitral disease. Mechanical obstruction of the renal veins by tumour, ascites, or ovarian cysts are less frequent causes. The urine in such cases is apt to be scanty, high-coloured and albuminous, but the urea is not notably diminished.

(*c*) **Infarction**. A renal infarct may be accompanied by pain in one or other loin, but quite often hæmaturia is the only symptom. A few blood casts can usually be found. The diagnosis is suggested by the discovery of endocarditis of the left heart.

IV. PYELITIS.

Consecutive Pyelitis due to an ascending infarction from the bladder is but a stage in the course of pyelo-nephritis which has already been considered.

In *Calculus Pyelitis* the symptoms of calculus may overshadow those of pyelitis, but not infrequently it is the pyelitis which claims attention, the calculus being found subsequently by the X-rays.

Tuberculous Pyelitis has been considered in the section on "Tuberculosis."

Primary Pyelitis is generally the result of an infection with Bacillus coli. In the majority of cases the infection is probably hæmatogenous; a lymphatic route is a possibility, as also is an ascending infection from the bladder, but this last is improbable so long as the mechanism of micturition is in no way impaired—that is to say, so long as there is no obstruction, either mechanical or paralytic, to the free voiding of the urine.

Acute Pyelitis is seen principally in two great classes, first,

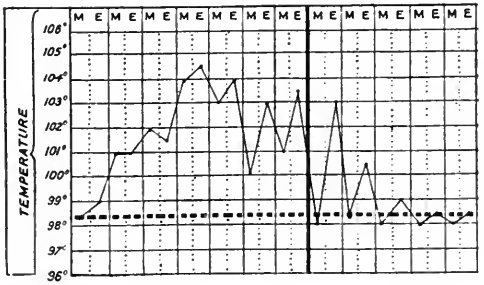


FIG. 64.—Chart from a case of Acute Pyelitis due to infection with Bacillus coli.

women after childbirth, and secondly, infants and young children.

The symptoms are those of any acute infection—shivering, malaise, headache, and fever. At the same time there will usually be severe pain in the loins (one or both) and definite tenderness in the angle between the spine and the twelfth rib. The urine contains many pus cells and bacilli; albumin and sometimes blood are present, as well as epithelial cells from the pelvis of the kidney. The amount of urine is diminished in severe cases, but there are no casts. In the majority of cases one kidney only is affected. The infection may be so severe as to necessitate nephrectomy, but in the generality of cases the symptoms subside under appropriate treatment in the course of a week or two.

In children cure is usually complete, but in adults recovery is often imperfect, and a chronic pyelitis or chronic bacilluria is left behind to prove a source of worry for many years.

Chronic Pyelitis, as stated above, may follow the acute variety ; it may also develop insidiously. There may be a dull aching pain in the back, but not infrequently the most prominent symptoms are those of neurasthenia. A movable kidney is found in some cases ; in others it is enlarged and tender. The urine is acid and contains pus cells and bacilli, but not, as a rule, more albumin than can be explained by the amount of pus present.

In the mildest type of all no pus cells are present in the urine—indeed, nothing abnormal can be found except numbers of bacilli.

The diagnosis of pyelitis rests on the urinary examination, reinforced, if necessary, by cystoscopy to examine the effluent from each ureter, the ureteric orifices, and the condition of the mucous membrane of the bladder.

Calculus must be excluded by X-ray examination.

Acute nephritis can be excluded by the excess of pus cells and the presence of bacilli.

V. URINARY CALCULUS.

A stone may lie imbedded in the kidney or renal pelvis for years without giving rise to symptoms. The usual symptoms of *renal calculus* are : pain and hæmaturia, though, as stated above, the symptoms of acute pyelitis may be the first clue to the diagnosis of stone. The pain is referred to the loin of the affected side, but also radiates to the groin, testicle, and inner side of the thigh on the same side.

If the calculus is in the ureter, renal colic is a likely event.

The pain of renal colic is probably as severe as any known form of pain ; it has the same distribution as the pain of renal calculus described above, but occurs in paroxysms of the utmost violence and is associated with vomiting, sweating, collapse, retraction of the testicle, and, nearly always, hæmaturia in acid urine. If the calculus becomes impacted at the opening of the ureter into the bladder no urine will be secreted by the affected kidney, and if the other kidney is diseased reflex suppression of urine may occur on that side also. The result is complete anuria.

Renal Colic due to stone must be distinguished from the following :—

(i.) *Acute Pyelitis*, in which the pain may be so severe as to simulate renal colic.

(ii.) *Dietl's Crises*, which are, in effect, attacks of renal colic associated with a movable kidney and periodic kinking of the ureter; the palpable kidney and the absence of hæmaturia may suggest the diagnosis.

(iii.) *Intestinal Colic*, in which the pain has a quite different distribution and is usually accompanied by flatulent distension of the bowel.

(iv.) *Biliary Colic*, in which the pain is referred principally to the right hypochondrium and through the right chest to the right shoulder, and in which the history will generally point to the liver or bile apparatus as a probable focus.

In cases of doubt an adequate X-ray examination will generally enable Renal Calculus to be excluded.

Stone in the Bladder is usually accompanied by alkaline urine, and there are frequency of micturition, strangury, hæmaturia and pyuria, while the pain from irritation of the trigone is referred to the end of the penis as well as to the perinæum and neck of the bladder.

VI. ANURIA.

Urine may fail to be secreted in the following circumstances :—

(i.) *Acute Nephritis*. Here the suppression is rarely complete for more than thirty-six hours.

(ii.) *Ureteric Obstruction*, either by calculus or by masses of pus and epithelial débris. The obstruction may be bilateral, but anuria may follow unilateral obstruction if the other kidney is diseased.

(iii.) *Acute Pyelitis*.

(iv.) *Massive Necrosis of the Renal Cortex*. This condition has been recorded about a dozen times. It occurs in association with parturition, or sometimes with eclampsia, or it may be associated with no apparent cause. In some cases there has been thrombosis of the ovarian and renal vessels

but more often an obliterative lesion has been found in the smaller renal arteries.

Where anuria persists the symptoms are relatively slight; the most prominent are vomiting, dyspnœa and towards the end slight twitching. The mental faculties are unimpaired, but death occurs quite suddenly from the seventh to the twelfth day of suppression. A striking feature of these cases of anuria is the absence of ordinary uræmic phenomena.

VII. URÆMIA.

By uræmia is meant the symptomatic expression of serious renal insufficiency. A variety of explanations have been advanced from time to time to account for these symptoms, but so far nothing altogether satisfactory has been evolved. The physical explanations of Rees and Ascoli respectively that uræmia depends upon œdema of the nervous system and meningeal inflammation are not supported by pathological findings. Of chemical theories the following may be mentioned :—

(i.) *Retention of Normal Products of Metabolism.* Retention of urine does not cause uræmia, and although urine is toxic if injected into animals the symptoms are not those of uræmia ; again, the urine in uræmia is not *less toxic* than in health.

(ii.) *Retention of the Products of Abnormal Metabolism* or their interaction to form some definitely toxic body. On this hypothesis the following bodies have been suggested :—

(*a*) *Urea.* This, however, is only toxic in very large amounts.

(*b*) *Ammonium Carbonate.* This does not produce uræmia when injected.

(*c*) *Potassium Salts.* These do not produce uræmia when injected.

(*d*) *Creatinin ; Pigments ; Chlorides ; Uric Acid.* All these are present in certain diseases (without producing the symptoms of uræmia) in greater amounts than are found in uræmia.

(iii.) *Acidosis.* The occurrence of coma in both uræmia and diabetes, coupled with the proved hypo-alkalinity of

the blood in the latter, has suggested the possibility of some analogous "acidosis" occurring in uræmia. The remaining symptoms of uræmia are, however, widely different from those of the acid intoxications, and, further, the treatment which is successful in the latter is without avail in uræmia.

(iv.) *Trimethylamine.* This body is, chemically, closely related to neurine, and the symptoms which follow the painting of the cerebral cortex with neurine somewhat resemble the symptoms found in certain cases of severe uræmia. Further, the characteristic odour of uræmic patients is not unlike that of trimethylamine, and it has been shown by Golla that the blood of uræmic patients contains as much as ten times the normal content of trimethylamine.

(v.) The internal secretion of the kidney has been shown by Bradford to exercise an inhibitory action on proteid metabolism. If, therefore, the renal disease causes a diminution of internal secretion, there will be a corresponding increase in the blood and tissues of the bye-products of proteid metabolism. This may help to explain how the specific uræmic toxin is formed, but it does not tell us what it is.

Clinically uræmia may be divided into acute, chronic, and latent forms.

(*a*) **Acute Uræmia.** Here the symptoms are mainly of nervous origin and may show themselves with the suddenness of an apoplexy. Convulsions, coma, mania, and delusional insanity are all common; the convulsions may be followed by amaurosis.

Paralysis either of isolated muscle groups or even hemiplegia may occur, while headache and giddiness are the rule. Gastro-intestinal symptoms, such as obstinate vomiting and diarrhœa or severe abdominal pain, may be present, and respiratory symptoms, such as dyspnœa (either continuous or paroxysmal) and Cheyne-Stokes breathing, are of frequent occurrence.

Other manifestations are formication, erythematous eruptions, and intense itching of the skin. The temperature is not characteristic, but is more likely to be low than high except when the convulsions have been of extreme severity.

(*b*) **Chronic Uræmia.** The symptoms are the same as in

the acute form but they are less severe and come on more gradually. Often one symptom or group of symptoms may be the only evidence of uræmia for many weeks.

(c) *Latent Urœmia.* This has been considered under the heading "Anuria." Beyond slight twitching and a tendency to vomiting and dyspnœa no symptoms are to be observed.

The diagnosis of uræmia may be a matter of extreme difficulty. In chronic cases the examination of the urine combined with a consideration of the cardio-vascular system should at least prove the existence of nephritis and hence suggest a cause for any seemingly obscure symptom; but it must be confessed that, at present, we cannot determine from an analysis of the urine whether uræmia is present or even imminent.

The principal difficulty, however, arises in the case of patients who are first seen when in an unconscious condition, and it is especially important to attempt to differentiate uræmia from the following :—

(i.) *Acute Alcoholism.* Here the odour of the breath may be suggestive, but it must be remembered that brandy is usually administered "on sight" to any unconscious person. An *uræmic* odour is important. The pupils in alcoholic coma are usually dilated; in uræmia they are variable, but generally are either medium or dilated. The alcoholic can usually be roused temporarily. If there is any doubt a catheter specimen of urine should be drawn off and examined for albumin and casts.

(ii.) *Opium Poisoning.* Here the pupils are pin-point and there is no albuminuria, while the characteristic odour of opium may be perceived in the breath.

(iii.) "*Apoplexy.*" The face is blue and swollen; the stertor is profound; the pupils are variable, they may be unequal and fixed; there may be conjugate deviation of the head and eyes and definite paralysis on one side of the body.

(iv.) *Diabetic Coma.* The smell of apples may be noticed in the patient's breath and sugar is present in the urine.

(v.) *Epilepsy.* The fits are brief and usually followed by sleep; there is usually no albuminuria.

VIII. MOVABLE KIDNEY.

This affection is much more common in women than men and in the spare than in the stout. The right kidney is practically always the first to be affected, though the left may follow suit. When both are affected there is usually a condition of general visceroptosis. The symptoms of movable kidney may be general or local.

The general symptoms are those of neurasthenia which may develop into melancholia or delusional insanity. The local symptoms are a dragging pain in the back or loin and sometimes the periodical occurrence of Dietl's crises, which are identical with attacks of renal colic. Dyspepsia is very commonly present and menorrhagia is far from rare.

The diagnosis of movable kidney can be made by physical examination, preferably with the patient half sitting up. One hand should be placed at the back and the other below the costal margin. If the hands are now approximated the kidney can be grasped between them and will be found to move up and down with respiration and also by the action of gravity. The contour of the organ can usually be well appreciated, and thus it can be distinguished from a renal tumour in the majority of instances.

IX. RENAL TUMOURS

(Including enlargements not due to new growth).

The general characteristics by which an abdominal tumour may be suspected to be of renal origin are as follows :—

(i.) The mass seems to be growing from the deep tissues of the loin forwards into the abdominal cavity.

(ii.) It can be grasped between the palms of the hands, if one be placed behind over the twelfth rib and the other in front below the costal margin.

(iii.) It moves definitely on respiration.

(iv.) Unless of very large size, a band of resonance may be detected superficial to the mass so long as there is gas in the colon.

(v.) Variations in size coincident with the passage of large amounts of (*a*) urine, point to hydronephrosis, or (*b*) pus, to pyonephrosis.

On the left side a renal tumour must be distinguished from spleen, fundus of stomach, splenic flexure of descending colon, retro-peritoneal sarcoma, and perinephric abscess: on the right side from liver, gall-bladder, pylorus, hepatic flexure of colon, retro-peritoneal sarcoma, and perinephric abscess.

In all cases examination of the urine may be of the greatest assistance, but the following points should be remembered :—

(*a*) *A Perinephric Abscess* should be suspected if there is marked dulness in the loin, œdema, and the constitutional disturbance of sepsis.

(*b*) *A Retro-peritoneal Sarcoma* is often indistinguishable from a renal tumour, but rapid growth in all directions or the occurrence of such a mass in childhood and without hæmaturia is suggestive.

(*c*) *Spleen.* An enlarged spleen appears to be much more superficial than a renal tumour; it seems to be growing from beneath the costal margin, it is dull to percussion, and the dulness merges into the ordinary splenic dulness. A blood examination should be made.

(*d*) *Colon.* Such masses are either very freely movable or else definitely fixed owing to the infiltration of surrounding structures. Their shape is most irregular; there are usually symptoms of chronic intestinal obstruction, and they are noticeably more superficial than a renal tumour.

(*e*) *Liver and Gall Bladder.* These again are more superficial than renal tumours, while a gall-bladder may present a characteristic oval shape. They are dull to percussion and the dulness merges into the normal liver dulness, but the edge of the liver can usually be felt above the neck of the gall bladder. Symptoms referable to the liver or bile apparatus may be present.

(*f*) None of these tumours, except *possibly* an enlarged spleen, can be so definitely grasped between the hands as can a renal tumour.

Renal Tumours not due to New Growth:—

(i.) *Hydronephrosis.* This condition is produced by any intermittent obstruction to the outflow of urine from the ureter. A ureteric calculus is a common cause. The

physical signs are the presence of a renal tumour, which often preserves closely the outline of a normal kidney and which varies in size from day to day, diminution occurring simultaneously with the voiding of large quantities of pale urine.

The general health may be quite good, but a history of renal or ureteric calculus is not improbable. In a few cases the hydronephrotic sac may be so large as to simulate free fluid in the peritoneum or an ovarian cyst.

(ii.) *Pyonephrosis.* This condition is precisely analogous to hydronephrosis except for the contents of the sacculated kidney, which are purulent. Therefore the physical signs will be the same except for pyuria, but the symptoms will be those of septic absorption.

(iii.) *Congenital Cystic Disease.* This is a bilateral affection in which the kidneys are converted into cystic masses which grow bigger with advancing years. They are discovered accidentally and perhaps not till the fourth or fifth decades of life. Patients suffering from them usually perish of uræmia before the age of fifty. The urine generally approximates in type to that of a red granular kidney. Hæmaturia is not infrequent.

The diagnosis of congenital cystic kidney should prove a contra-indication to any surgical operation if this can possibly be avoided, for uræmia is only too likely to follow the operation.

Renal New Growths. These may be innocent or malignant, and the latter may be primary or secondary.

A. Innocent Tumours. (i.) *Hypernephroma.* These tumours are said to arise in misplaced fragments of adrenal tissue; they are potentially malignant, but often remain "innocent" throughout their course. The presence of a renal tumour may first suggest the diagnosis unless the growth has encroached on the pelvis of the kidney, in which case hæmaturia may be the first sign. Cystoscopy will show that the blood is coming from one kidney only, and if blood diseases, tuberculosis, and calculus are excluded the only diagnosis left is new growth or renal epistaxis. This question and the nature of the tumour can only be settled by exploratory operation.

(ii.) *Adenomata* call for no special description ; they rarely reach sufficient size to give rise to symptoms.

B. Malignant Tumours. These may be carcinoma or sarcoma. The latter are not uncommon in small children.

The remarks made on hypernephroma apply with equal force to malignant renal tumours, but removal by operation is necessarily not so hopeful in the latter case.

In cases in which the growth is secondary no operation should be attempted.

Hæmaturia is an early sign in more than 50 per cent. of all malignant renal tumours.

X. THE ESTIMATION OF THE RENAL FUNCTION.

Various methods have been devised with a view to estimating the functional activity of the kidneys, but it must be admitted that from a purely medical aspect the results are of little value, either from a diagnostic or prognostic point of view, in cases of nephritis.

From a surgical point of view, however, some of these tests are of great importance since, if it is proposed to remove one kidney, it is desirable to know that the remaining kidney is likely to prove equal to the increased demands that will be made upon it.

It is not too much to say that a nephrectomy should never be performed without a preliminary cystoscopy and approximate estimation of the renal function.

The following investigations should be made :—

(*a*) *Cryoscopic determinations of the blood and of the urine*, obtained by catheterisation of the ureters.

The freezing point of a fluid is lowered to an extent proportional with the number of molecules of solid substances which are dissolved in it. By this means it can be shown whether the blood contains an excess of solids, and if this is so it is suggestive of a total renal insufficiency. The freezing points of the separated urines will show which kidney is principally at fault.

It must be remembered that the technique is extremely difficult and altogether beyond the scope of the prac-

titioner, but if properly carried out it affords some indication of the osmotic efficiency of the kidneys.

(*b*) *Injection of Phloridzin.* This experiment depends on the fact that after the subcutaneous injection of phloridzin a healthy kidney will excrete an amount of sugar proportional to the amount of phloridzin injected, and that the sugar will appear in the urine within a certain time of injection and will cease a certain time later.

Accordingly cystoscopy is performed, and catheters left in the ureters (practically it is sufficient to catheterise one ureter and collect the urine from the other kidney *viâ* the bladder), and at the same time ·005 gm. of phloridzin is injected beneath the skin.

The effluent from each kidney is received into sterile bottles, which are changed every thirty minutes for three hours. A healthy kidney shows sugar in its urine after half an hour, and secretes from ·5 to 2·5 grms. of sugar during the next two hours, after which no more sugar is found.

Should the urine contain less than ·5 gm. a serious renal inadequacy may be presumed. This may be confirmed by urea estimation.

(*c*) *The Diastase Reaction, vide* p. 404.

(*d*) *Simple Cystoscopy* may show that there is no kidney on one side or that (in cases of tuberculosis) both kidneys are affected and that an operation should be avoided. (*Vide* also article on "Tuberculosis," p. 102.)

XI. INFLAMMATION OF THE BLADDER (CYSTITIS).

A bacterial invasion of the bladder is the immediate cause of cystitis, but there may be, and often is, bacilluria without cystitis. Additional factors which encourage any bacteria that may be present to set up active inflammation are either traumatic—for example, the presence of a calculus or the ova of bilharzia; the faulty use of a catheter; the presence of a new growth; or anything which allows the urine to stagnate, such as retention from stricture or enlarged

prostate, in cases of paraplegia, or when there is a vesical saccule. Some cases appear to be spontaneous and follow a chill or exposure.

Cystitis is usually divided into acute and chronic forms, but all grades of inflammation may be met with from catarrh to gangrene.

Acute Cystitis is characterised by severe pain in the hypogastrium, frequency of micturition, strangury and the passage of blood, pus and epithelium. The urine is usually acid. The pain is often referred to the perinæum, and in men to the glans penis.

Chronic Cystitis. This may follow an acute attack or develop insidiously.

The symptoms are the same as those of acute cystitis, but usually much less severe ; indeed, in mild cases there may be none at all. The urine contains pus, ropy mucous and epithelium, and is alkaline unless the organism is the tubercle bacillus, a member of the Bacillus coli group, or sometimes the gonococcus.

When cystitis has existed for a long time the bladder walls become thickened and contracted and are often encrusted with phosphates.

Diagnosis of Cystitis. The presence of pus in the urine means inflammation of the kidney or bladder, if urethral or prostatic affections can be excluded.

If there is no more albumin present than would be explained by the pus, and if the urea output is normal, the probability is that the kidney is sound (unless possibly there is pyelitis : *vide* p. 454). Pain in the hypogastrium, perinæum and penis strongly suggests inflammation of the bladder, as does the presence of much mucus in the urine.

In cases which do not clear up quickly and for which no obvious cause can be found it is advisable to examine the bladder with a cystoscope, as by this means the condition of the mucous membrane can be determined, as well as the presence of calculi, sacculations, and new growths. Further, the condition of the kidneys can be estimated by looking at the ureteric orifices and by observing the jets of urine from each.

Tuberculosis can be excluded by cystoscopy or by

repeatedly examining the urine for tubercle bacilli, or by injecting some of the urinary sediment into a guinea-pig.

Vesical calculus can be detected by the passage of a sound, by X-rays or, in children, by bimanual examination of the abdomen with one finger in the rectum.

PART V

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS.

I. **The Tracts in the Central Nervous System.** The physiological function of the nervous system is to receive impulses or stimuli (conscious or unconscious) from the various organs and from the periphery generally, to convey these afferent stimuli to appropriate cell centres in the brain or cord, and to respond to them by such efferent impulses as may be required.

Just as the afferent impulses may be conscious or unconscious, so may the efferent ; indeed, practically the whole of the processes necessary for adequate metabolism and ordinary existence are carried out unconsciously. The processes that take place between the reception of an afferent impulse and the resulting performance of a voluntary action are, perhaps, the simplest form of thought.

In order to make a correct diagnosis in many cases of disease of the nervous system it is first essential to locate the position of the lesion or lesions as accurately as possible. This can only be accomplished by a satisfactory understanding of the anatomy of the nervous system both central and peripheral.

The Central Nervous System consists of the Brain and Spinal Cord. The brain is the site of the most highly specialised groups of cells and is the seat of consciousness. The spinal cord is a mass of conducting fibres between the brain and the periphery, but contains, in addition, certain cell stations between individual conducting fibres and also the cell nuclei for all the motor portions of the spinal nerves.

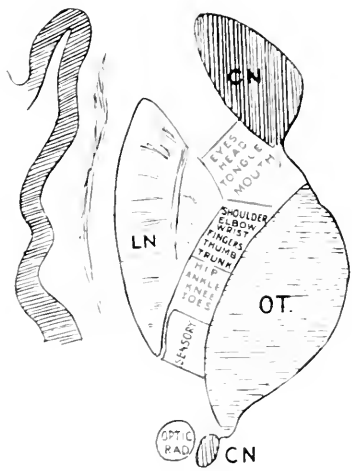


Fig. 65.—Diagram to show the arrangement of the motor and sensory fibres in the Internal Capsule.

CN = Caudate Nucleus.
LN = Lenticular Nucleus.
OT = Optic Thalamus.

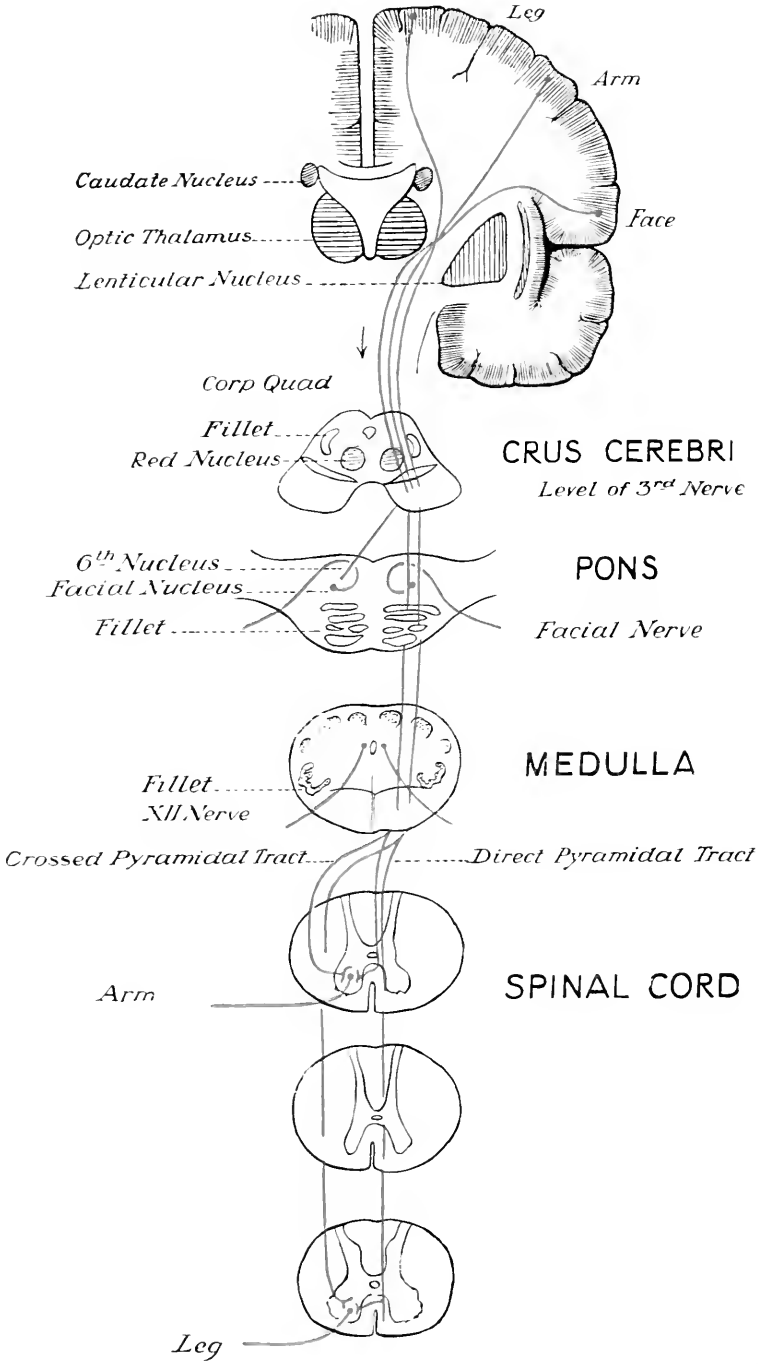


FIG. 66. Diagrammatic representation of the Motor Path from the Cortex to the Anterior Horn Cells.

THE MOTOR PATH. The cells responsible for the initiation of all voluntary movements are situated in the cortex of the precentral area of each cerebral hemisphere, that is to say, immediately in front of the fissure of Rolando. The characteristic large pyramidal cells of Betz are only found in the motor area of the cortex.

In front of the precentral area is the intermediate precentral area of Campbell, which governs the more highly specialised movements of the body as opposed to the crude movements which originate in the precentral area proper. Both the precentral and the intermediate precentral areas are represented on the mesial as well as on the superficial aspects of the cerebral hemispheres.

The nerve fibres originating in the giant Betz cells form the pyramidal tract; they pass through the centrum ovale to become part of the corona radiata, and converge together into a fairly compact bundle by the time the internal capsule is reached. In the internal capsule they occupy the "genu" and the anterior third of the posterior limb (Fig. 65).

After traversing the internal capsule the pyramidal tracts pass through the crura cerebri and ventral aspect of the pons to form the pyramids of the medulla oblongata, but before this they have parted company with the fibres for the nuclei of the third, fifth, sixth, seventh, and twelfth cranial nerves and with some to the nucleus ambiguus for the motor parts of the ninth, tenth, and eleventh cranial nerves. In addition to this some fibres are connected with the red nucleus, the thalamus, and the pontine nuclei, whence arise rubro-spinal, ponto-spinal and thalamo-spinal tracts, which may be assumed to form, under certain circumstances, alternative routes for the transmission of certain motor impulses.

At the pyramids of the medulla the pyramidal tracts decussate with each other and cross to the other side to form the crossed pyramidal or lateral tracts of the cord, except for a small number which are continued down the anterior aspect of the spinal cord on the same side to form the direct pyramidal tract of Turk. The fibres of the direct pyramidal tract, however, cross *viâ* the anterior commissure, at intervals

all the way down the cord so that all motor fibres are ultimately crossed.

Eventually the fibres of the crossed and direct pyramidal tracts arborise round the anterior horn cells all the way down the cord.

The path from the cerebral cortex to the anterior horn cells of the opposite side is known as the Upper Motor Neuron System, and any lesion of this path produces a paralysis of the corresponding muscles of the *opposite* side of the body if the lesion is above the decussation of the pyramids, and of the muscles on the *same* side if the lesion is in the cord.

The Lower Motor Neuron System comprises the motor nerve fibres from the anterior horn cells, or their bulbar analogues, to their termination in the muscles.

The Sensory Path. This is more complex and less fully worked out than the motor path. Certain sensory paths cross before reaching the brain, but others are homo-lateral. The main cortical sensory area is situated in the post-central area just behind the fissure of Rolando, and the individual localities are believed to correspond fairly closely to the adjacent motor areas.

The centres for Pain and Temperature are probably rather posterior to those just described, but they may be situated in the gyrus fornicatus.

Sensation can be subdivided into three main groups—Protopathic, Epicritic, and Deep.

Protopathic Sensation includes pain and extremes of heat and cold.

Epicritic Sensation comprises light touch, localisation, tactile discrimination, and slight degrees of heat and cold.

Deep Sensation includes sense of passive position (comprising impulses from joints, muscles, bones, and tendons), sense of pressure or contact, and sense of painful pressure.

All forms of sensation enter the cord through the posterior nerve roots, and it is worthy of note that if a peripheral nerve is cut there is a greater area of epicritic sensory loss than of protopathic.

This epicritic overlap is less marked the nearer the injury to the cord, so that Head considers the posterior nerve root

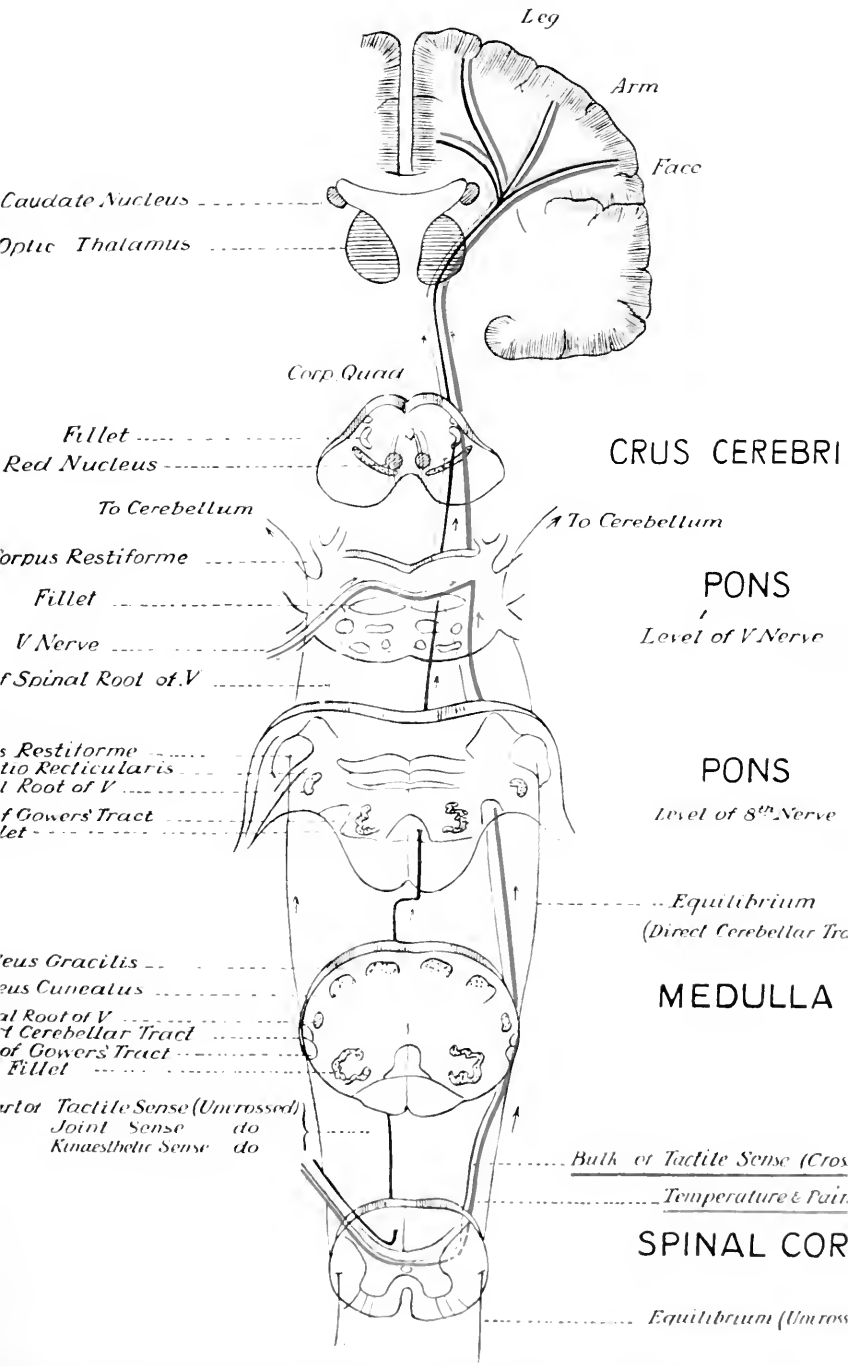


FIG. 67.—Diagrammatic representation of the paths taken by the Sensory Impulses in Cord and Brain

to be the unit of protopathic sensibility and the peripheral nerve to be the unit of epicritic sensibility.

The fibres for deep sensation run with the motor nerves, though, of course, they enter the cord by the posterior root. The cells of origin of the sensory spinal nerves are in the posterior root ganglia. On entering the cord by the posterior root the sensory fibres may behave as follows :—

(*a*) The long fibres pass into the columns of Burdach and Goll and run up in these columns homo-laterally to arborise round cells in the cuneate and gracile nuclei of the medulla respectively. The lowest spinal fibres get squeezed more and more towards the mid-line by the fibres which enter later, so that the lower spinal fibres form the column of Goll and the upper spinal fibres form the column of Burdach. This differentiation usually results in those fibres from below the fourth dorsal segment forming Goll's column.

The cells of the gracile and cuneate nuclei give off fresh fibres, which decussate with their fellows of the opposite side in the fillet and are continued as the mesial fillet through the medulla and pons to arborise round cells in the ventral aspect of the optic thalamus.

From the optic thalamus the third and last relay starts and the fibres run through the posterior part of the posterior limb of the internal capsule to the post-central cortical sensory area.

The following sensory impulses travel in the manner just described :—

Sense of Passive Position.
Sense of Tactile Discrimination.
Part of Ordinary Touch.
Part of Localisation.

Before setting out on their upward course these long fibres give off a branch which passes downwards for a short distance in the posterior horn of the same side as the comma tract of Schultze.

(*b*) The short fibres arborise round cells in the posterior horn immediately on entering the cord ; from these cells fibres are given off which may cross immediately to the

other side of the cord by the posterior commissure or which may run a variable distance in the posterior columns before thus crossing. In either event when crossed the fibres run up the cord as the spino-thalamic tract in close apposition to the ventral cerebellar tract, but leave this tract when it passes into the superior cerebellar peduncle and are themselves continued straight to the cells in the ventral aspect of the optic thalamus. From here fibres are given off which run through the internal capsule to the appropriate cortical sensory areas.

The sensory impulses which adopt this route are :—

Sense of Pain and painful pressure.
Sense of Heat and Cold both moderate and extreme.
Most of the Sense of Touch.
Part of the Sense of Localisation.

(*c*) The medium fibres take one of two courses :—(i.) To the cells of Clarke's column on the same side, from which are given off the fibres forming the dorsal or direct cerebellar tract of Flechsig. The direct cerebellar tract runs up on the same side through the medulla and pons to enter the cerebellum *viâ* the restiform body ; these fibres terminate on the dorsal aspect of the vermis or middle lobe of the cerebellum. (ii.) To certain cells in the posterior horn, from which arise the fibres forming the ventral cerebellar tract of Gowers. This runs up on the same side of the cord to enter the cerebellum through its superior peduncle and terminates on the ventral aspect of its middle lobe.

Since the functions of the cerebellum are largely connected with equilibration and co-ordination it is probable that certain impulses from muscles, bones, joints, etc., travel in these columns and enable the cerebellum to establish equilibration and stability.

(*d*) Some fibres entering by the posterior root run straight across to arborise round certain anterior horn cells of the same side and so complete the various segmental reflex arcs.

It is quite impossible in the present state of our knowledge to be didactic as to the various sensory paths. That the above description is probably correct for the majority of cases is shown by the clinical phenomena which are seen

after hemisection of the cord (Brown-Sequard's paralysis) as shown by the following table :—

On the Side of the Lesion.	*On the Side Opposite to the Lesion.*
Motor paralysis.	No motor paralysis.
Generally no impairment of touch, light pressure or cutaneous localisation.	Generally impairment of touch, light pressure or cutaneous localisation.
Pain and temperature sensations normal ; painful pressure normal.	Abolition of pain, temperature and painful pressure sensations.
Impairment of tactile discrimination and sense of passive position.	Persistence of sense of passive position and tactile discrimination.

At the same time the above phenomena are not constantly found when the lesions would suggest that they ought to be present, so that the preceding remarks as to the sensory paths must only be regarded as a sound working hypothesis until more accurate knowledge can be obtained.

The Cerebellum does not initiate impulses on its own account and appears to be chiefly concerned with the preservation of equilibrium in both standing and walking. For this purpose stimuli are constantly passing to the cerebellum from the periphery conveying impressions of alteration in the position of any particular part of the body in relation to its environment. These afferent stimuli call forth corresponding efferent impulses regulating the proper co-ordination of certain muscle groups, and generally determining the strength of the nervous impulses passing to the muscles concerned from the cerebral cortex.

The afferent impulses to the cerebellum are conveyed from the periphery, and mostly from the muscles of the back and extremities, to the cerebellum by way of the spino-cerebellar tracts and the columns of Flechsig and Gowers, which are homo-lateral[1] and enter the cerebellum *viâ* the restiform body and the superior peduncle respectively ; they terminate respectively on the dorsum and venter of the vermis or middle lobe of the cerebellum. There are also fibres running from the olive to the vermis.

[1] Gower's column *may* receive certain fibres from the cells of the contra-lateral posterior horn.

Another cerebellar path is by way of the vestibular nerve, by means of which impulses from the ampulla, utricle and saccule of the internal ear are conveyed to the vestibular nucleus of the cerebellum, thence to Deiter's nucleus, and finally to the vermis. The sensations of perception of space and proper orientation depend largely on an intact vestibular path. The nuclei of the ocular muscles also have free communication with Deiter's nucleus, and form in this way a supplementary vestibular system, without which our power of estimating distances would be very deficient.

The cerebellar tracts just described (Fig. 68) should be considered as the afferent limbs of a most important reflex system. The efferent paths of the cerebellar reflex system are as follows :—

(i.) From Deiter's nucleus (which thus appears to be both motor and sensory) *viâ* the dorsal longitudinal bundle with the oculo-motor nuclei.

(ii.) From Deiter's nucleus arises the vestibulo-spinal tract, which runs through the medulla, down the periphery of the spinal cord on the same side for its entire length.

Presumably this tract is the route adopted by the controlling impulses from the cerebellum to the homo-lateral trunk and limb muscles.

(iii.) From the corpus dentatum *viâ* the middle cerebellar peduncle fibres pass to the pontine nucleus and tegmentum cruris of the opposite side.

(iv.) From the corpus dentatum arises the brachium conjunctivum, which passes through the superior peduncle to the tegmentum, where it decussates and terminates in the red nucleus and optic thalamus of the opposite side.

It is possible that by means of these last two paths the cerebellum regulates the synergic action of the motor impulses passing down the ponto-spinal, rubro-spinal and thalamo-spinal tracts ; it is also possible that there is an indirect connection between one cerebellar lobe and the opposite cerebral cortex *viâ* the brachium conjunctivum, the optic thalamus, the internal capsule, and the centrum ovale.

The decussation of the fibres from the corpus dentatum to the red nuclei does not affect the homo-lateral influence

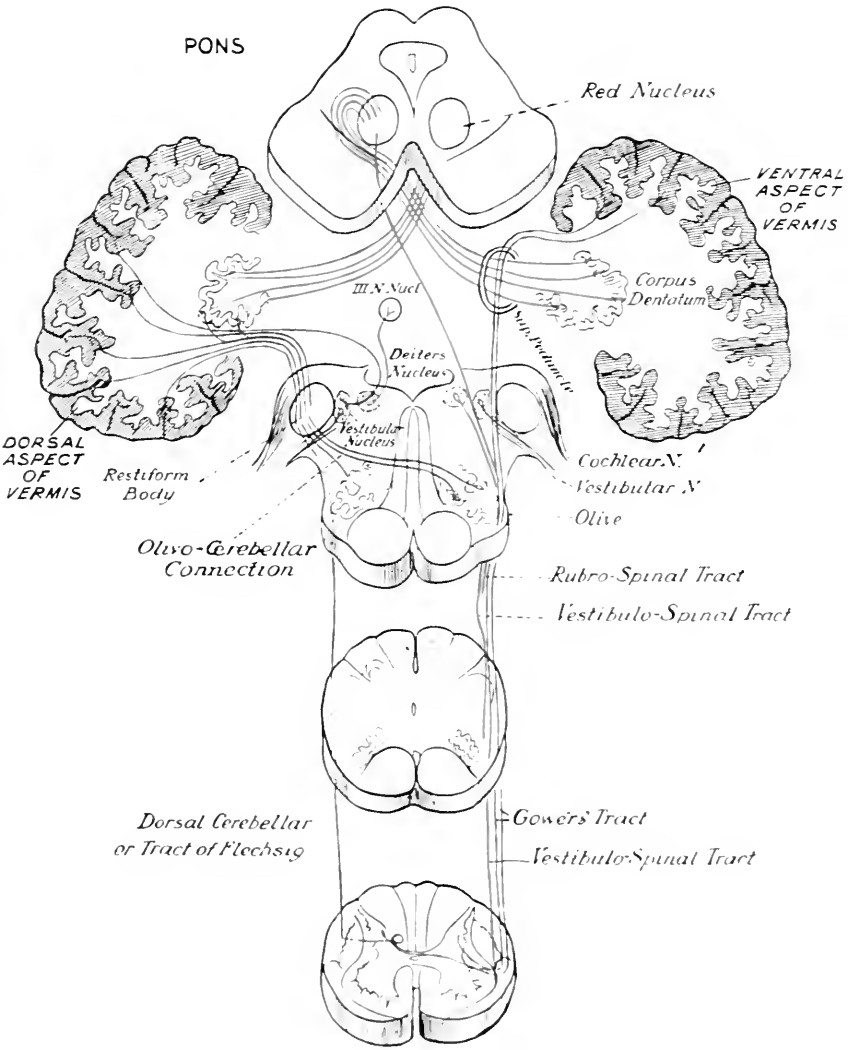


FIG. 68.—Diagrammatic representation of the Main Cerebellar connections.

of the cerebellum on the muscles of the trunk and limbs, because the rubro-spinal tract (Monakow's bundle) itself decussates soon after its inception and these two decussations naturally neutralise each other.

These anatomical facts will be appreciated more readily if they are considered in conjunction with Figs. 65—68.

II. **Reflex Actions.** In addition to the cerebellar reflexes considered in the last section three types of reflex may be distinguished :—

(i.) Superficial, obtained by stroking or light scratching of the skin ;

(ii.) Deep reflexes, obtained by tapping a tendon which is in a state of slight tension ; and

(iii.) Organic reflexes, which comprise such acts as swallowing, micturition, defæcation, and parturition.

These reflex actions take place quite automatically under certain circumstances and can occur independently of any action of the brain, though such organic reflexes as swallowing or micturition become more or less subject to cerebral control in process of evolution and education. Furthermore, the deep reflexes are subject to constant inhibitory impulses from the brain, so that their vigour is modified in certain brain and cord lesions which do not actually involve the reflex path concerned.

The superficial reflexes are also influenced by the brain, but the nature of the cerebral control is uncertain.

The anatomical path for the performance of a spinal reflex consists of an afferent limb by which the stimulus is conveyed to the cord *viâ* the posterior nerve root, a connecting fibre which consists in the arborisation of one of the short posterior root fibres round the corresponding anterior horn cells of the same spinal segment, and an efferent limb by which the responsive impulse is conveyed to the muscle concerned. The controlling or inhibitory impulse for deep reflexes from the cerebrum travels in the lateral pyramidal tract (*vide* Fig. 69).

Since even in sleep the majority of the muscles are never absolutely flaccid, there is a necessity for a constant series of afferent impulses from the muscles requisitioning efferent impulses to regulate the muscle tone. This tonic path for

different muscles is identical with the reflex path just described, and the deep reflexes are thought to depend largely on the state of muscular tonus.

The following reflexes are the ones most usually examined :

A. The Superficial Reflexes :—

Corneal and Conjunctival. Blowing upon or gently touching the cornea or conjunctiva causes the eyelid to shut.

Palatal. Touching the soft palate causes the uvula to be drawn up.

Pharyngeal. Touching the posterior pharyngeal wall causes the throat muscles to contract.

Epigastric and Abdominal. Stroking the skin below the rib margins and stroking the outer border of the rectus abdominis causes contraction of the abdominal muscles in the quadrant tested.

Scapular. Stroking the skin between the shoulder blades causes the scapular muscles to contract.

Anal. Scratching the perinæum causes the sphincter ani to contract.

Cremasteric. Stroking the skin over Scarpa's triangle causes the testicle to retract.

The Plantar Reflexes. To elicit the plantar response successfully it is necessary to have the leg somewhat everted at the hip and flexed at the knee with the ankle completely relaxed ; the foot must be warm and dry. The skin of the sole is now stroked rather firmly (as with a penholder) along its outer border.

In health there is flexion of the toes, including the big toe, and a tendency to inversion of the foot.

In Lesions of the Upper Motor Neuron and in infants who have not yet walked a totally different response takes place ; the foot tends to be everted and the great toe becomes extended (Babinski's phenomenon).

The essential part of this test is the behaviour of the great toe ; when properly performed it is an invaluable aid to diagnosis.

Even apart from the plantar reflex the diagnostic value of the superficial reflexes is considerable, though perhaps not so great as in the case of the deep reflexes.

Exaggeration of superficial reflexes does not imply organic disease.

Absence or great depression of the superficial reflexes is found in upper motor neuron lesions and often in cases of increased intra-cranial tension, but is very rarely if ever seen in functional disease.

Disease of the nerve roots may cause absence of all reflexes for the segments concerned.

B. The Deep Reflexes :—

The Knee Jerk. One leg is crossed over the other and allowed to hang quite loosely; the patellar tendon is now struck smartly towards its inner edge—the quadriceps muscle contracts and the foot is kicked up in consequence.

This test may be reinforced by supporting the toes of the hanging foot with the hand of the examiner or by instructing the patient to shut his eyes and clench both hands as tightly as he can.

Reinforcement should always be employed before the knee jerk is said to be absent.

The Achilles Jerk. The patient is asked to kneel on a chair or couch, with his feet projecting over the edge; the tendon Achilles is smartly struck, and the calf-muscles contract and extend the foot at the ankle-joint.

Supinator Jerk. The elbow is flexed to about 120° and the arm supported in this position with the hand hanging down. The back of the radius is now struck just above the styloid process and the hand is jerked slightly upwards.

Triceps Jerk. The elbow is flexed to a right angle and the arm supported; the triceps tendon is struck above the olecranon process, and the triceps muscle can be seen and felt to contract.

Clonus. This occurs when there is great exaggeration of deep reflexes due to lack of cerebral inhibition, as in an upper motor neuron lesion.

Pseudo-clonus may occur in hysteria.

Ankle Clonus is elicited by placing the leg in the position described for the plantar reflex and suddenly dorsiflexing the foot, which has been grasped round the toes.

Patellar Clonus is elicited by extending the leg so that the patella moves freely, and then suddenly pushing the patella downwards so as to stretch the quadriceps tendon.

The deep reflexes are never entirely absent in health; such a condition must mean a lesion somewhere in the reflex arc or in the muscles themselves.

Exaggeration of the deep reflexes occurs in hysteria, but is significant of upper motor neuron lesion if it is accompanied by spasm, if it is unilateral, or if it is only present below a certain level.

III. **The Vaso-motor System.** The vaso-constricting fibres arise in the lateral sympathetic ganglia, while vaso-dilating fibres, when present, are in the other ganglion systems incorporated in the sympathetic nervous system.

The constricting fibres travel in the ordinary mixed nerves and reach these structures in the grey rami communicantes which run from the sympathetic ganglia to the segmental nerves. There are, however, spinal vaso-constricting cells in the centre of the anterior horn cells, and from these are given off white rami communicantes which run to the lateral sympathetic ganglia (*vide* Fig. 69).

The spinal vaso-motor centres are controlled by a vaso-motor centre in the medulla, and probably this, in turn, is under the influence of some part of the precentral cortex.

The fibres from the medullary centre to the spinal cell-stations run in the lateral pyramidal tracts.

IV. **Cerebral Localisation and Blood Supply.** The cortical localisation of the various functions of the brain is not yet completely worked out. A consideration of the previous pages, in conjunction with Fig. 70, will assist in mastering what is known up to the present on this subject.

The Blood Supply of the Brain. A knowledge of the blood supply of the brain is of importance in view of the fact that vascular lesions are so common in the cerebral blood-vessels.

The circle of Willis is situated in the inter-peduncular space at the base of the brain. The circle is formed behind by the two posterior cerebral arteries which are terminal branches of the basilar artery (formed by the union of the two vertebral arteries). In front the two internal carotid

Fig. 69.—Diagrammatic representation of the Spinal Cord in transverse section at the level of the eighth dorsal vertebra, showing the principal tracts, the paths taken by the sensory fibres entering by the posterior nerve root, the reflex path for tendon reflexes, and the vasomotor path (*vide* p. 478), etc.

The tracts are seen on the left of the figure.

A. Direct pyramidal tract.
B. Crossed pyramidal or lateral tract.
C. Ventral cerebellar tract of Gowers.
D. Dorsal cerebellar tract of Flechsig.
E. Burdach's column.
F. Goll's column.
G. Comma tract.
H. Vestibulo-spinal tract.
I. Clark's column.
K. Lissauer's tract.
L. Spino-Thalamic tract.

The fibres are chiefly on the right of the figure.

1 and 2. Motor nerve fibres leaving the anterior horn cells.
2a and 4. Fibres conveying cerebral impulses to 1 and 2 *viâ* the lateral tract and the direct pyramidal tract respectively.
3. Vasomotor fibre running from the medulla in the lateral tract to control (7).
4. (*Vide supra*).
5. Fibres from Clarke's cell-column going to form the direct cerebellar tract.
6 and 6a. The motor and sensory limbs respectively for the ordinary deep reflex arc.
7. The white ramus communicans which leaves in the anterior nerve root and runs to the sympathetic ganglion X.
8. Grey ramus communicans running from the sympathetic ganglion X to the mixed nerve.
9. The path for temperature, most of touch, and pain sensations crossing the cord to join the contra-lateral spino-thalamic tract.
10 and 10a. Equilibration fibres running to the ventral cerebellar tract.
11 and 12. Fibres for joint and muscle sense, etc., running to the columns of Goll and Burdach.

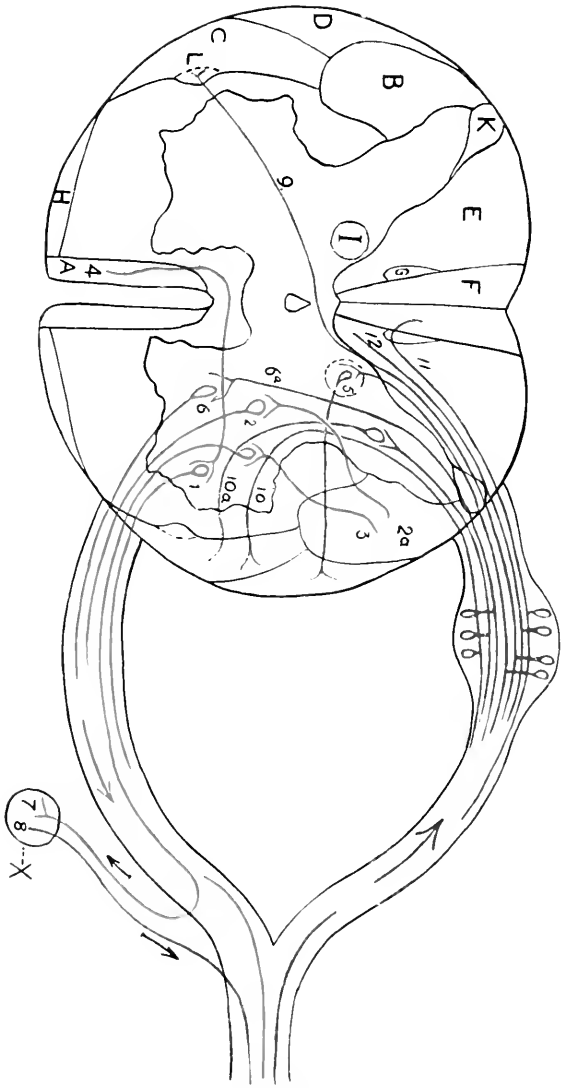
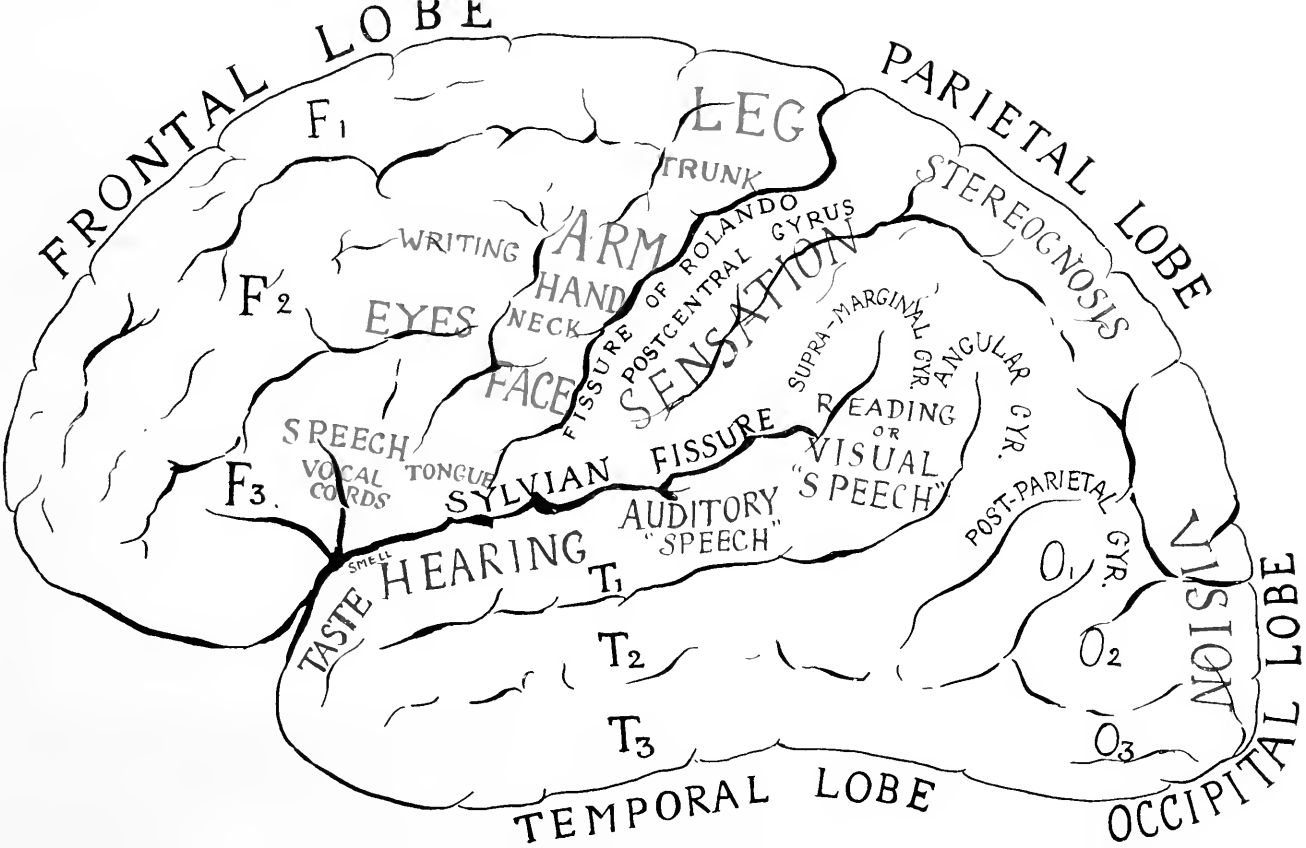


Fig. 69.


FRONTAL LOBE
PARIETAL LOBE
TEMPORAL LOBE
OCCIPITAL LOBE
F1
F2
F3
LEG
TRUNK
ARM
HAND
NECK
FACE
WRITING
EYES
SPEECH
VOCAL CORDS
TONGUE
SYLVIAN FISSURE
FISSURE OF ROLANDO
POSTCENTRAL GYRUS
SENSATION
STEREOGNOSIS
SUPRA-MARGINAL GYR.
ANGULAR GYR.
READING OR VISUAL "SPEECH"
AUDITORY "SPEECH"
POST-PARIETAL GYR.
SMELL
TASTE
HEARING
T1
T2
T3
O1
O2
O3
VISION

arteries divide into the middle cerebral arteries and the anterior cerebral arteries.

Joining the two anterior cerebral arteries is the anterior communicating artery, and each posterior cerebral artery is joined with the termination of the corresponding internal carotid artery by a posterior communicating artery.

Each cerebral artery supplies both superficial and deep aspects of the brain.

The Anterior Cerebral Artery supplies the external surface as far backwards as the middle of the parietal lobe and as far downwards as the superior frontal sulcus; mesially it runs as far backwards as the parieto-occipital fissure and includes the corpus callosum and gyrus fornicatus. The deep branches of the anterior cerebral artery supply the anterior limb of the internal capsule and the anterior parts of the lenticular and caudate nuclei, as well as the floor of the third ventricle.

The Middle Cerebral Artery supplies the external surface from the superior frontal sulcus to the anterior occipital sulcus, while it extends downwards to the second temporal sulcus. It gives no branches to the mesial surface. The deep branches of the middle cerebral artery supply the remainder of the caudate and lenticular neucleus; the middle part of the optic thalamus (lenticulo-optic artery), the centrum ovale, the posterior limb of the internal capsule, the external capsule, and the outer and upper half of the optic radiations.

The Posterior Cerebral Artery supplies the rest of the external surface of the brain except the uncinate lobe, which is supplied by the anterior choroidal artery, so that mesially it runs up to the anterior cerebral artery and externally up to the middle cerebral artery.

The deep branches of the posterior cerebral artery supply the posterior half of the optic thalamus, the pulvinar, the red nucleus, and the lower inner half of the optic radiations.

The Anterior Choroidal Artery is given off from the internal carotid artery just before its bifurcation; it supplies the uncinate lobe superficially and, deeply, the anterior part of the optic thalamus, the corpora quadrigemina, the internal geniculate bodies, most of the fornix, and the

posterior part of the posterior limb of the internal capsule.

The Cerebellum is supplied by three vessels—superior, middle, and inferior cerebellar arteries, the two former arising from the basilar and the last from the vertebral artery.

The Pons and Medulla are supplied by the numerous branches of the basilar artery.

It is important to remember that the terminal branches of the cerebral arteries do not anastomose with their fellows from the same trunk, though terminals from the middle cerebral anastomose to some extent with terminals from both anterior and posterior cerebral arteries where these abut.

Further, the superficial and deep branches in no place anastomose with each other.

V. **Electrical Reactions of Muscles.** A. *In Health.* Faradic stimulation causes a brisk and sustained contraction. The electrode should be placed as near as possible to that part of the muscle into which its nerve passes.

Galvanic stimulation causes a single contraction when the current is made or broken, but no contraction while the current is passing. The force of the contraction varies according to whether the anode or the kathode is applied to the muscle and also according to whether the current is made or broken.

The magnitude of the healthy response is represented as follows :—

$$KCC > ACC : \quad AOC > KOC,$$

where K = Kathode, A = Anode, CC = closing contraction, and OC = opening contraction.

Stimulation of the nerve leading to the muscle also causes a contraction both with faradism and galvanism.

B. *In Lesions of the Lower Motor Neurons* the so-called reaction of degeneration takes place—that is to say, the response to faradism becomes diminished or lost and the response to galvanism becomes sluggish instead of brisk, and in addition the qualitative arrangement of the responses becomes notably altered. For example :—

ACC becomes equal to or greater than KCC and KOC becomes greater than AOC.

In extreme cases no response to either faradism or galvanism can be elicited, whether the current is applied to the nerve or to the muscle. More usually the nerve loses its power of response completely while the galvanic reactions of the muscles show the above qualitative changes.

It is worthy of note that in ordinary cases the *opening contractions* for the galvanic current require so strong a current that so much pain is caused as to negative the use of this portion of the test.

VI. **Evidence of Lesions in the Motor Path.** A. *Lesions of the Upper Motor Neuron System* produce a spastic motor paralysis for all voluntary muscles below the lesion, the affected muscles being on the opposite side of the body if the lesion is above the decussation of the pyramids.

At the same time the deep reflexes for the affected area are increased and the superficial reflexes are abolished or very much diminished; the plantar reflex gives an extensor response and clonus can be elicited. The muscles are rigid and stiff, but there is no real loss of muscle power, no wasting, and no reaction of degeneration, because the lower motor neurons are intact.

B. *Lesions of the Lower Motor Neuron System* produce a flaccid paralysis of the muscles supplied from the affected anterior horn cells or by the damaged motor nerve.

All reflexes are abolished in the affected area, the muscles waste, there is marked reaction of degeneration, and often trophic change.

N.B. The extensor plantar response is the most valuable evidence of an organic lesion involving the upper motor neuron system. It is not met with in functional disease and cannot easily be maintained by malingerers. The only circumstances other than organic disease of the upper motor neuron path in which an extensor plantar response may occur are :—

(i.) Anterior poliomyelitis, picking out only the cells for the flexor muscles of the toes, so that if any plantar response occurs it must be extensor. This condition must be so rare as to be negligible clinically. Obvious poliomyelitis elsewhere with an extensor response might

suggest the above explanation, provided that there was no other evidence of upper motor tract involvement.

(ii.) Coma and even sleep have been reported as being occasionally associated with an extensor plantar response. Much more usually, we feel sure, is there an absence of all response under such circumstances, and we have not observed a constant extensor response in any such case when the reflex has been repeatedly examined in the proper manner (*vide* p. 476).

A lesion of the cord above the centres for the reflex emptying of the bladder and rectum (S 3—S 4) usually entails involuntary micturition as soon as the viscus is sufficiently distended by urine to excite the reflex act. The rectum does not behave in quite the same manner, since there is generally constipation owing to reflex spasm of the sphincter.

When the lesion is in the sacral region and involves the centres themselves there is usually permanent incontinence of both urine and fæces, the bladder and rectum merely serving as open passage-ways ; not infrequently, however, there is sufficient elasticity in the neck of the bladder to permit of considerable distension before overflow dribbling away takes place (retention with overflow).

The sexual apparatus (S 1—S 4) behaves in an analogous way ; lesions above the centre often produce more or less priapism and a tendency for an exaggerated effect from trifling stimuli, while lesions involving the centre cause complete impotence.

VII. **Evidence of Lesions in the Sensory Paths.** A. A posterior root lesion diminishes all forms of sensory perception for the region concerned. Since, however, no area sends its sensory impulses exclusively to one posterior nerve root, complete loss of all sensation implies a lesion of more than one nerve root (provided, of course, that the distribution of sensory disturbance is segmental and not attributable to a damaged peripheral sensory nerve).

B. Ataxia and astereognosis are present in posterior root lesions provided that a sufficiently extensive area is involved to permit of their demonstration.

C. Complete loss of tactile sensation implies a lesion of—

(i.) A peripheral nerve ;

(ii.) Two or more posterior nerve roots, or root zones within the cord ; or

(iii.) Both homolateral posterior and contra-lateral spino-thalamic columns in the cord.

D. Loss of pain and heat sensation implies as a rule a les on of the grey matter or of the contra-lateral spino-thalamic tract, provided the posterior roots and peripheral nerves are intact.

E. Ataxia of cerebellar type occurs in lesions of the ascending cerebellar tracts or, of course, of the cerebellum itself.

F. Before complete destruction of the cells and fibres concerned in a lesion of the sensory paths takes place irritation phenomena often become manifest. These take the form of pain, numbness, tingling, itching, etc., and are referred to the segmental area in the case of posterior root lesions and to the area of peripheral supply in the case of a peripheral nerve—that is to say, to the region of the sensory end organs of the damaged fibres.

VIII. **Segmental Distribution.** As an aid to regional diagnosis it is necessary to be familiar with the cutaneous distribution of the fibres forming the individual posterior roots, and also to understand which muscles receive their nerve fibres from the cells in any given spinal segments.

The distribution of the peripheral nerves, both motor and sensory, is described in every text-book of anatomy and will not be discussed here.

The sensory segmental distribution can be understood by reference to Figs. 71 and 72, which need no description. It is, however, important to remember that the diagrams indicate the principal sensory supply only, since, as stated above, there is considerable overlapping from the segments next above and below.

The motor segmental distribution for the principal muscles is as follows :—

Muscles of the Upper Extremity.

Supraspinatus and teres minor . .	C. 5
Deltoid, infraspinatus, subscapularis, biceps, brachialis anticus, and supinator longus	C. 5—6

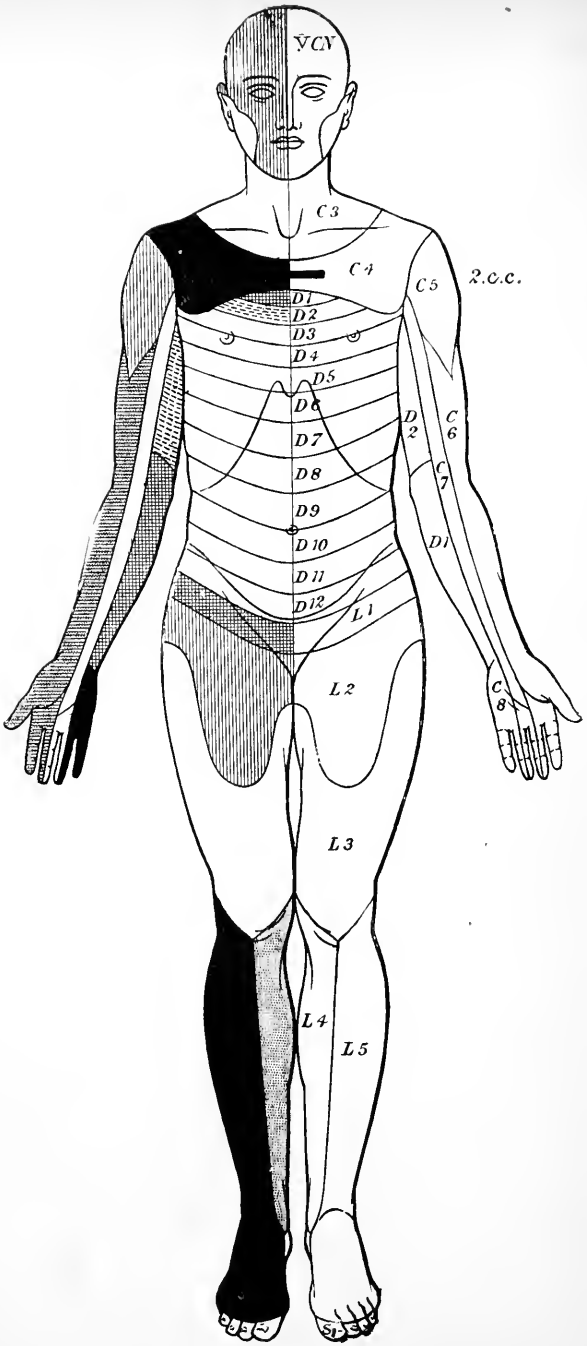


Fig. 71.—Diagram to show the segmental distribution of the Sensory Nerves.

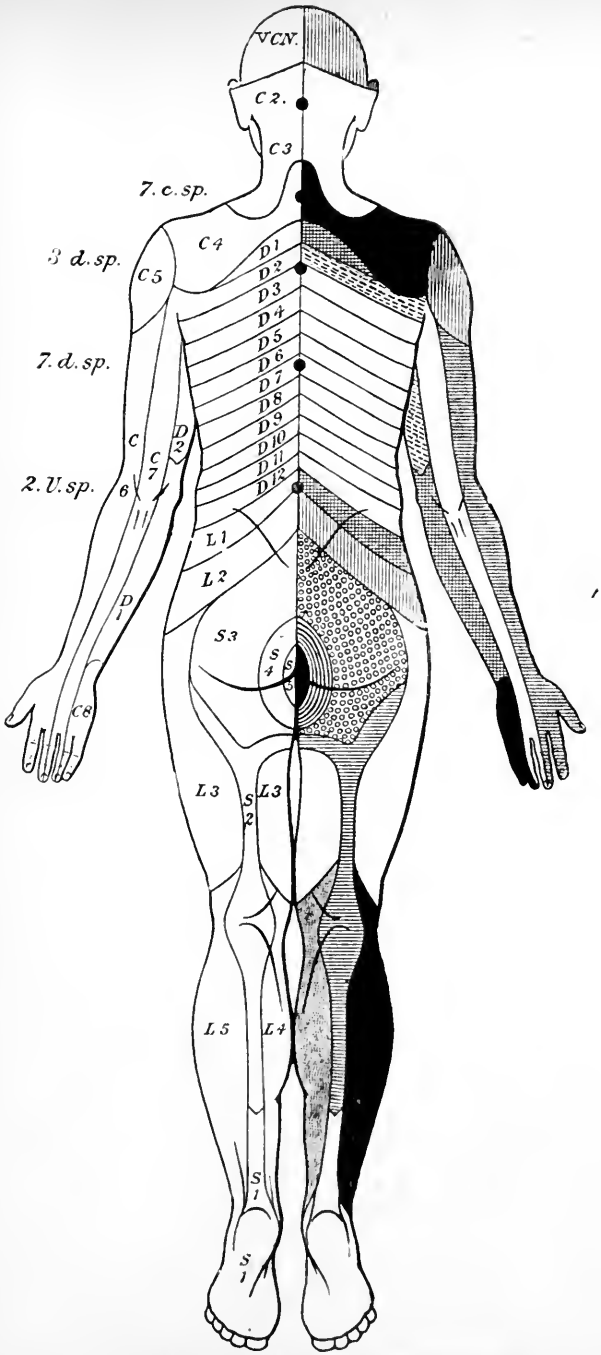


Fig. 72.—Diagram to show the segmental distribution of the Sensory Nerves.

Muscles of the Upper Extremity—continued.

Teres major, supinator brevis, extensor carpi radialis longior and brevior	C. 5—7
Coraco-brachialis, pronator radii teres, flexor carpi radialis, flexor longus pollicis, abductor pollicis, extensor brevis pollicis, flexor brevis pollicis, opponens pollicis	C. 6—7
Triceps, extensor longus pollicis, extensor communis digitorum, extensor indicis, extensor carpi ulnaris, extensor minimi digiti	C. 6—8
Anconæus	C. 7—8
Flexor sublimis digitorum, flexor profundus digitorum, flexor carpi ulnaris, pronator quadratus, palmaris longus, flexor minimi digiti, opponens minimi digiti	C. 7—D. 1
Adductor pollicis, palmaris brevis, abductor minimi digiti, lumbricales, interossei	C. 8—D. 1

Muscles of the Lower Extremity.

Psoas	D. 12—L. 4
Sartorius, pectineus, adductor longus	L. 2—L. 3
Gracilis, adductor brevis, quadriceps	L. 2—L. 4
Adductor magnus, adductor minimus, obturator externus	L. 3—L. 4
Tensor fasciæ femoris, tibialis anticus	L. 4—L. 5
Glutæus medius, glutæus minimus, quadratus femoris, gemellus inferior, semi-tendinosus, semi-membranosus, extensor longus hallucis, extensor longus digitorum, popliteus, plantaris	L. 4—S. 1
Biceps, soleus, gastrocnemius, glutæus maximus, gemellus superior.	L. 4—S. 2
Peroneus longus, peroneus brevis, adductor obliquus hallucis	L. 5—S. 1

Muscles of the Lower Extremity—continued.

Obturator internus, tibialis posticus, flexor longus digitorum, flexor longus hallucis, flexor brevis hallucis, lumbricales	L. 5—S. 2
Pyriformis, abductor hallucis, abductor minimi digiti, flexor brevis minimi digiti, opponens minimi digiti, interossei	S. 1—S. 2

The Trunk Muscles.

Short deep cervical muscles . . .	C. 1—C. 2
Splenius, scaleni	C. 3—C. 8
Trapezius	C. 2—C. 4
Latissimus dorsi	C. 6—C. 8
Levator anguli scapulæ . . .	C. 3—C. 5
Rhomboidæi	C. 4—C. 5
Longus capitis	C. 1—C. 4
Longus colli	C. 5—C. 8
Pectoralis major	C. 5—D. 1
Subclavius	C. 5—C. 6
Pectoralis minor	C. 7—D. 1
Serratus magnus	C. 5—C. 7
Diaphragm	C. 3—C. 5
Rectus abdominis and external oblique	D. 5—D 12
Transversalis abdominis . . .	D. 7—L. 1
Internal oblique	D. 8—L. 1
Quadratus lumborum	D. 10—L. 4
Levator ani, sphincter ani, etc. . .	S. 3—S. 5

CHAPTER II

THE CRANIAL NERVES

I. **The Olfactory Nerves.** The olfactory nerves enter the skull through the cribriform plate in the ethmoid and join the olfactory bulb from which the olfactory tracts run to the brain. Each tract divides into two parts, one of which crosses to the other side by the anterior commissure and the other runs towards the temporal lobe of the same side. The cortical smell centre is probably in the uncinate gyrus.

Anosmia. Loss of sense of smell is not usually a sign of organic nerve lesions ; more often it depends on some local condition in the nose.

Anosmia may be found in fractures of the anterior fossa of the skull if the olfactory tracts are damaged or for a similar reason in tumour or inflammation of the lower surface of the frontal lobe ; it may also be a manifestation of hysteria.

Parosmia (perverse sensations of smell) may occur in insanity, as an aura in epilepsy, or in tumours of the temporal lobe which involve the cortical centre.

II. **The Optic Nerve.** The optic nerves run backwards from the retinæ, and the fibres from the nasal half of each retina decussate at the optic chiasma, while the fibres from the temporal halves of the retinæ bend outwards again into the optic tract of the *same* side. The result is that each optic tract corresponds to the temporal half of the homolateral retina and to the nasal half of the contra-lateral retina. Fibres from the actual macula appear to separate, part decussating and part remaining homolateral. The optic tracts terminate in three cell stations. The greatest number of fibres go to the external geniculate body, a smaller number go to the superior corpus quadrigeminum and to the pulvinar of the optic thalamus. From these three cell stations the optic radiations run backwards through the

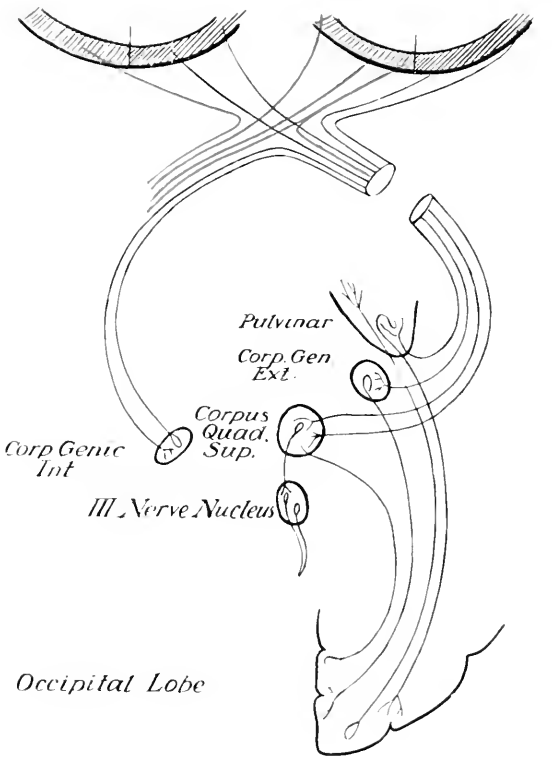


Fig. 73. Diagram to show the arrangement of the Optic Nerves, Tracts, and Radiations

hindmost part of the internal capsule to the cortical visual centres in the occipital lobes (*vide* Fig. 73).

The half-vision centre is situated on the mesial aspect of the occipital lobe round the calcarine fissure.

It is noteworthy that the calcarine artery has probably the poorest anastomosis of any cerebral artery, so that hemianopia due to thrombosis of this vessel hardly ever shows any improvement in vision.

The pupillary light reflex is accomplished by the passage of a stimulus along the optic nerve and tract to the superior corpus quadrigeminum, thence to the nucleus of the third nerve by an association tract, and so *viâ* the ciliary ganglion to the sphincter pupillæ.

Dilatation of the pupil is governed by the inferior cervical sympathetic ganglion *viâ* the long ciliary branches of the ophthalmic branch of the fifth cranial nerve (*vide* also p. 495).

From the preceding remarks it is clear that a lesion of one optic nerve causes blindness of the corresponding eye; that a lesion at the centre of the optic chiasma causes bitemporal hemianopia (loss of vision in the nasal half of each retina); that a symmetrical lesion of the outside fibres of the optic chiasma would be necessary to produce binasal hemianopia; and that a lesion anywhere behind the optic chiasma, between it and the cortical visual centre, causes homonymous hemianopia (loss of vision for the nasal half of the retina of the opposite eye and for the temporal half of the retina of the eye on the same side as the lesion).

If, however, the lesion causing homonymous hemianopia is at or in front of the superior corpus quadrigeminum the pupil will not contract when a pencil of light is projected on to the *blind half* of the retina, whereas if the lesion is in the occipital pole or optic radiations the pupil will contract under the above circumstances because the reflex path is intact (Wernicke's hemiopic pupillary reaction).

In homonymous hemianopia the blind area of the retina does not, as a rule, include the macula. This is explained by the fact that the macula has a double cortical representation by reason of the partial decussation of its fibres at the chiasma.

Optic Neuritis. This may be produced by anything which

causes increase in intracranial pressure such as tumour or abscess, or, more rarely, meningitis; it is also seen in morbid blood states, such as chronic nephritis, grave anæmia, lead poisoning, etc.

It is remarkable what good vision can be preserved with even high degrees of swelling; blurring of vision and slight contraction of the visual fields are all that can be detected in many cases until optic atrophy supervenes, when, of course, permanent blindness develops. Hence the importance of a routine examination with the ophthalmoscope.

Optic Atrophy. This may develop in three ways and can readily be diagnosed by the ophthalmoscope :—

(i.) Primary optic atrophy as seen in tabes, general paralysis of the insane, disseminated sclerosis, amaurotic family idiocy, etc.

(ii.) Secondary optic atrophy, which follows some injury to the optic nerve, as in fractured base of the skull, or some other lesion which is not accompanied by optic neuritis.

(iii.) Consecutive optic atrophy, which follows unrelieved optic neuritis.

Primary optic atrophy shows a clear-cut greyish-white disc with normal blood-vessels.

Consecutive optic atrophy shows a white disc with blurred edges and small arteries.

III. **The Oculo-Motor Nerves (Third, Fourth and Sixth).** The third nerve supplies the levator palpebræ superioris, the contractor fibres to the iris, and all the external muscles of the eye except the superior oblique and the external rectus, which are supplied by the fourth and sixth nerves respectively.

The nuclei for the third nerve are situated beneath the floor of the fourth ventricle and the front part of the Sylvian aqueduct and extend in a vertico-caudal direction for a considerable distance.

Three main groups can be recognised, the foremost of which sends fibres to the ciliary ganglion and is the centre for the sphincter pupillæ; the next, or mesial nucleus, is the centre for accommodation; while the hindermost nuclear mass is itself composed of five small nuclei, which, though closely adjacent to each other, are functionally quite

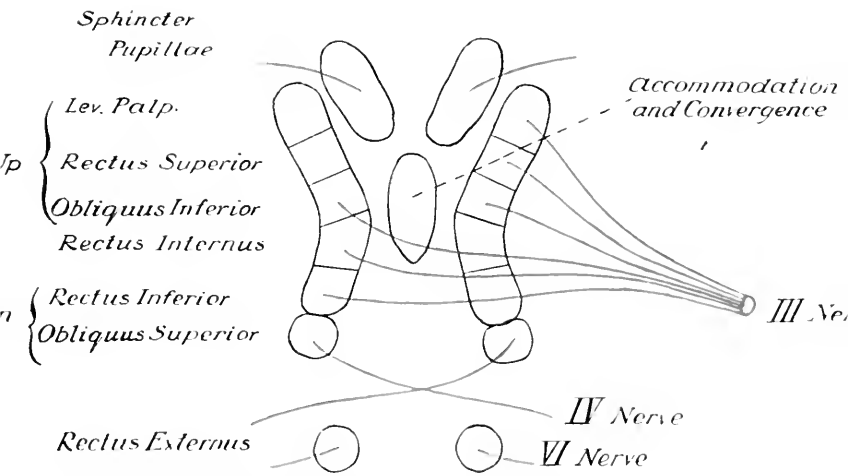


Fig. 74. Diagrammatic representation of the Oculo-motor Nucleus.

distinct. From before backwards these five small nuclei are as follows (*vide* Fig. 74) :—

(i.) The centre for levator palpebræ superioris
(ii.) The centre for superior rectus
(iii.) The centre for inferior oblique
} *i.e.*, looking upwards.

(iv.) The centre for internal rectus
(v.) The centre for inferior rectus .. (*i.e.*, looking downwards).

Immediately behind this third nerve nucleus lies that for the fourth nerve, the function of which is to move the cornea downwards and outwards by means of the superior oblique muscle. It is noteworthy that the fibres of the fourth nerve decussate completely *after* leaving their nuclei. It is probable that the fibres from the centres for internal rectus, inferior oblique, and inferior rectus undergo a partial decussation after leaving their nuclei, as represented in the diagram.

A considerable distance behind the fourth nerve nucleus—that is to say, lower down the pons, but in the same plane—is the sixth nerve nucleus, which supplies the external rectus muscle for the same side, but also sends a fibre to the nucleus for the internal rectus of the opposite side in order to produce synergic lateral deviation of the eyes.

Oculo-motor Paralysis may occur as a congenital affection or it may be acquired in the following conditions :—

(i.) Systemic nervous diseases, such as tabes dorsalis or disseminated sclerosis.

(ii.) Polio-encephalitis affecting the brain stem.

(iii.) New growth or vascular lesions in the brain stem.

(iv.) Basal meningitis due to syphilis or tuberculosis or following fractures of the sphenoidal fissure or orbit.

(v.) Toxic neuritis, as in diphtheria.

(vi.) Cortical lesions may disturb conjugate deviation.

(vii.) Hysteria and migraine.

(viii.) Any condition which produces a great increase in intracranial tension may produce a partial ophthalmoplegia. The most usual phenomenon is an external rectus palsy (single or double). This may be explained in part by the long intracranial course of the sixth nerve and its

extreme antero-posterior direction, which renders it particularly liable to be pulled upon when the brain sinks backwards and the medulla is depressed into the foramen magnum in the effort to make room for the increased amount of cerebro-spinal fluid.

Hence it is obvious that an external rectus palsy by itself is not often of great value as a localising sign of intracranial disease.

Complete Ophthalmoplegia includes the third, fourth, and sixth nerves.

The eye is immobile, the pupil is dilated (sympathetic) and central, and the upper lid is dropped.

Ophthalmoplegia Interna includes only the internal ocular muscles. If the third nerve only is affected the eye is turned downwards and outwards by the action of the superior oblique and external rectus muscles.

A *Partial Ophthalmoplegia* gives rise to the following signs and symptoms :—

(i.) Loss of movement of the eye in the direction of action of the paralysed muscle.

(ii.) Diplopia for such positions of the eyes as would involve the action of the paralysed muscle.

The diplopia is crossed in cases of divergent strabismus, that is to say, the false image is on the side of the sound eye ; the converse obtains in convergent strabismus.

(iii.) *Secondary Deviation.* If the paralysed eye is covered and an object fixed with the other eye, the paralysed eye can be observed to " swing " as the other eye fixes ; this is " primary " deviation.

If the converse experiment is now performed by covering the sound eye and fixing with the paralysed eye, the sound eye also swings when the paralysed eye fixes ; this is " secondary " deviation, and " secondary " deviation is greater than " primary " deviation except in the congenital affection known as *Concomitant Strabismus*, when the two deviations are equal in amplitude of swing.

(iv.) *Strabismus*, or squint, is simply faulty convergence of the visual axes and may be so slight as to escape observation.

(v.) *Erroneous Projection of the Visual Field.* If the

patient is asked to touch an object which is held in such a position as to make him try to use his paralysed muscle when he looks at it, he will always make a faulty estimate of the position of the object and place his finger beyond the object *in the direction of action* of the paralysed action.

It is not always obvious which muscle is paralysed ; this can, however, usually be ascertained in the following manner. First place a different coloured glass in front of each eye so that it is possible to say at once which image belongs to which eye ; next note whether the diplopia is crossed or homonymous—for example, if there is a red glass over the right eye and a blue glass over the left, the diplopia is homonymous if the red image is on the patient's right and the blue image on his left.

We can now apply the rule that " that eye is affected in the direction of the image of which the diplopia increases." For example, suppose the distance between the images increases when the patient looks to his right and the diplopia is homonymous, then the right eye is the affected one ; if the diplopia is crossed, then the right image corresponds to the left eye and therefore the left eye is affected.

Having in this manner ascertained in which eye the paralysed muscle is situated, we can apply the second of Landolt's laws, which tells us that the false image (that is to say, the image corresponding to the affected eye) bears that position in relation to the true image which the affected muscle would give to the eye. For example, if the false image is above and to the left of the true image and also with its upper end tilted to the left, and if it is known that the right eye is affected, then the right superior rectus is the affected muscle, for this muscle moves the eye upwards and also rotates it somewhat obliquely to the left.

As a general rule it may be stated that the cortical innervation of the ocular muscles is so largely bilateral that hemiplegic supranuclear lesions do not cause ophthalmoplegia. Cortical lesions sometimes cause a contra-lateral levator palpebræ paralysis, and may also cause conjugate deviation of the eye to the side of the lesion in a destructive lesion, or to the opposite side in an irritative lesion, through the opposite sixth nucleus. In like manner an irritative

lesion of the pons may cause conjugate deviation to the same side, or a destructive lesion to the opposite side. In these cases there may be involvement of the facial nerve as well, because the sixth nucleus is surrounded by the intramedullary root fibres of the facial nerve.

Nuclear lesions of the oculo-motor system are rare, but may sometimes be seen in polio-encephalitis; though owing to the extensive nuclear area the entire nucleus is not often affected. In contrast to an infra-nuclear lesion, the orbicularis palpebrarum is paralysed in nuclear lesions, since this muscle is supplied from the third nucleus *viâ* the seventh nerve.

A nuclear lesion of the sixth nerve in addition to the external rectus muscle of the same side affects the opposite internal rectus as far as conjugate deviation is concerned, but does not interfere with this muscle when used for accommodation.

In addition, sixth-nerve nuclear palsy is often accompanied by facial paralysis of infra-nuclear type owing to the proximity of the facial nerve during its intra-pontine course.

Gross lesions of the brain stem are, of course, liable to involve the oculo-motor nerves and their nuclei, but the hemiplegic or diplegic signs will be present also. In this manner a lesion of the crus may cause paralysis of the opposite face and limbs and of the oculo-motor nerve on the same side.

In most cases of oculo-motor paralysis associated with gross brain stem lesions the oculo-motor paralysis is of the "lower" or degenerative type, and is due to involvement of the nuclei or the nerves below the nuclei.

Unilateral ophthalmoplegias, whether partial or complete, are, in the majority of cases, due to lesions at the base of the brain, *e.g.*, tuberculoma or gummatous meningitis.

It is worthy of note that the oculo-motor nerves may be the subject of a paralysis which is etiologically identical with Bell's palsy as seen affecting the seventh nerve.

Nystagmus. By nystagmus is meant rhythmical oscillation of the eyeball; it may be rotary, lateral or vertical, and may be constantly present or only to be seen when certain muscles are put on the stretch.

Nystagmus is commonly due to lack of proper co-ordinating

control. and is therefore found in lesions of the cerebellum or of the mid-brain and tegmentum pontis if the dorsal longitudinal bundle is involved. In these cases the nystagmus is most marked, or perhaps only found on looking towards the side of the lesion.

Douching the ears with cold water is apt to cause nystagmus to the opposite side, while if hot water is used the nystagmus is to the same side. These impulses are conveyed from the labyrinth *viâ* the vestibular system to the dorsal longitudinal bundle. Inflammatory affections of the internal ear can produce nystagmus in a like manner.

In disseminated sclerosis and Friedreich's disease the nystagmus may be regarded as akin to the intention tremor, which is so marked a feature of these diseases.

Nystagmus may also be met with under the following circumstances :—

(i.) Occupations imposing constant lateral strain on the eye, *e.g.*, coal miner.

(ii.) In toxic neuritis, *e.g.*, diphtheria.

(iii.) In congenital ocular defect, such as optic atrophy, albinism, congenital cataract, etc., in which the child has never seen well enough to learn to "fix" objects.

(iv.) In central defects of congenital nature and obscure pathology, often familial, and sometimes accompanied by other tremors, such as spasmus nutans or head-nodding.

The Pupillary Light Reflex. The path for this reflex is probably as follows :—From the retina through the optic nerve and tract to the superior corpus quadrigeminum, thence by association fibres to the anterior portion of the third nerve nucleus and so to the ciliary ganglion, and thence to the sphincter pupillæ muscle. It is probable that the centre of control for this reflex lies in the ciliary ganglion.

The dilating mechanism is under the control of the cervical sympathetic system ; the path for reflex dilatation runs from the retina to the brain stem, then down the cord to the first and second dorsal segments, where it leaves the cord in the white rami communicantes and runs to the inferior cervical ganglion, and subsequently to the long ciliary branches of the ophthalmic branch of the fifth cranial nerve and so to the iris.

IV. **The Fifth Cranial Nerve** (Trigeminal). The gasserian ganglion is the sensory cell nucleus for the fifth nerve; the motor nucleus is formed in part by a mass of cells which is situated laterally in the tegmentum pontis and in part by a strip of cells running down to the cervical cord (spinal root).

The motor root supplies the muscles of mastication; the sensory branches supply the skin of the face (except the auricle and the greater part of the lower jaw), the conjunctiva and the mucous membrane of the tongue as far as the circumvallate papillæ and of the mouth and cheeks as far as the anterior pillars of the fauces, and lastly all the teeth.

The chorda tympani conveys taste fibres from the anterior two-thirds of the tongue and leaves the tongue in the lingual branch of the fifth, but soon leaves this nerve to join the facial nerve.

Some writers hold that these taste fibres enter the brain in the fifth nerve, and assume that they leave the facial nerve at its geniculate ganglion and run in the great superficial petrosal nerve to Meckel's ganglion and so to the second division of the fifth nerve.

In like manner it is possible to construct a path to the fifth nerve for the taste fibres of the posterior part of the tongue which leave in the glosso-pharyngeal nerve; they may be said to run in the tympanic branch of the glosso-pharyngeal to the small superficial petrosal nerve, and so to the otic ganglion, and finally to the third division of the fifth nerve.

Since, however, excision of the gasserian ganglion, and even section of the entire fifth nerve, is only very rarely followed by loss of taste for the corresponding half of the tongue, it is probable that the taste fibres really enter the brain through the glosso-pharyngeal nerve and the sensory root of the facial respectively.

The course of the taste fibres in the brain is not known; it is probable that eventually they reach the anterior pole of the temporo-sphenoidal lobe. They do not pass through the internal capsule.

Paralysis of the fifth nerve may be caused by lesions in

the pons affecting the nuclei, by lesions at the base of the brain affecting the nerve roots, or by lesions of the peripheral course of the nerves.

If the motor part of the nerve is affected the jaw swings to the paralysed side when the mouth is opened.

If the sensory part of the nerve is affected there may be complete anæsthesia of its area of cutaneous distribution. When this happens trophic ulcers of the cornea are commonly found.

Trigeminal Neuralgia consists of violent pain, sometimes persistent, but more often paroxysmal and oft repeated, referred to the area supplied by one or more sensory branches of the fifth nerve. The affected part may be swollen, glazed, and tender. The paroxysms appear to be started by trivial reflex causes, such as smiling, eating, etc.

The condition is often characterised by tender spots where the branches of the nerve become subcutaneous, and the pain appears to start at these spots and thence to radiate peripherally along the branch.

Certain of these cases are due to reflex causes, such as carious teeth, refractive errors or naso-pharyngeal disease, and these must always be carefully excluded, but in many no cause can be found, though possibly there may be some interstitial inflammation in the gasserian ganglion.

V. **The Seventh Cranial Nerve (the Facial).** The motor nucleus for this nerve is situated beneath the floor of the fourth ventricle. The sensory part of the nerve arises in the geniculate ganglion and runs into the brain as the " pars intermedia " of Wrisberg.

The motor supply is to all the muscles of facial expression excepting levator palpebræ superioris and to the occipito-frontalis muscle, the muscles of the pinna, and also to the stapedius muscle ; the sensory fibres probably form part of the supply to the skin of the external auditory meatus and the front of the external ear. The pars intermedia anastomoses with the trigeminal nerve, and from this the corda tympani is formed, which contains salivary and taste fibres.

Although the orbicularis oris and the orbicularis palpebrarum are supplied by fibres of the facial nerve, it is probable

that these muscles are not supplied from the seventh nucleus, but from the hypoglossal and third nerve nuclei respectively, the communications being with the facial nerve after it leaves its nucleus or with the upper part of the facial nucleus itself *viâ* the posterior longitudinal bundle.

The facial nerve has a very long intra-pontine course, and it winds round the sixth nucleus before emerging on the ventro-lateral aspect of the pons between the olivary and restiform bodies close to the cerebello-pontine angle.

The facial nerve now enters the internal auditory meatus with the eighth nerve, passes through the aqueductus Fallopii, across the roof of the middle ear, and emerges through the stylo-mastoid foramen. The upper part of the facial nucleus receives a bilateral cortical supply ; this comprises the frontalis and possibly also, as indicated above, the orbicularis palpebrarum. The upper motor neuron to the facial nucleus decussates about the middle of the pons.

Lesions causing a facial paralysis may be situated anywhere from the cerebral cortex to the peripheral distribution of the nerve. It is usually possible to localise the lesion with considerable accuracy.

(i.) *Infra-nuclear Lesions.* The most common cause is exposure to chill or draught, which sets up a parenchymatous neuritis at or about the lower end of the aqueduct ; other causes may be middle-ear disease, operations on the mastoid, birth injuries from forceps, syphilis, basal meningitis, and tumours of the cerebello-pontine angle. If facial palsy is produced *spontaneously* from mastoid disease the disease is usually tuberculosis.

Infra-nuclear facial paralysis is generally unilateral, unless due to basal meningitis. The signs are characteristic :

All the muscles of that side of the face are paralysed, and it consequently becomes smoothed and free from wrinkles ; the patient is unable to shut his eye, frown, whistle, or smile ; tears overflow on to the cheek, and food collects in the cheek and tends to run out of the corner of the mouth.

The following additional points should be remembered :—

(*a*) Lesions at the base of the brain (syphilitic meningitis or tumour) usually involve other cranial nerves as well,

especially 6, 9, 10, 11, and 12, and are often associated with general cerebral symptoms, such as headache and vomiting.

(*b*) Lesions in the aqueductus Fallopii proximal to the junction of the chorda tympani and below the geniculate ganglion give loss of taste for the anterior two-thirds of the homolateral half-tongue and also diminished salivary secretion on that side. Further, if the lesion is proximal to the nerve to the stapedius, that muscle is paralysed, with the result that there is hyperacusis on that side from the ability to detect notes of fewer vibrations than is normally the case.

(*c*) Lesions between the internal auditory meatus and the geniculate ganglion cause diminished salivation but no loss of taste; there is, however, often nerve deafness from implication of the eighth nerve, and this, when present, masks the hyperacusis which can otherwise be recognised.

(ii.) *Nuclear Lesions.* Nuclear lesions of the facial nerve alone are rare, but may be seen occasionally in poliomyelitis acuta.

Chronic lesions in the course of bulbar paralysis are not uncommon and are associated with paralysis of other bulbar nerves, often as a late stage of progressive muscular atrophy (*vide* p. 554).

In a pure nuclear lesion the upper part of the nucleus may escape and the orbicularis palpebrarum may be unaffected owing to its probable supply from the third nucleus.

Lesions in the pons may, of course, affect the nuclei, and also the nerve after it leaves its nucleus but before it leaves the pons. Such cases often leave the upper face unaffected, and are usually accompanied by a sixth nerve palsy on the same side, and often by a paralysis of the limbs on the opposite side from pyramidal involvement, or a crossed hemi-anæsthesia from interference with the sensory fibres in the fillet.

(iii.) *Supra-nuclear Lesions.* In these the paralysis is crossed and the lower face only is obviously paralysed, though there is usually demonstrable weakness of the upper face muscles as well. The reason why the upper face escapes

almost completely is that the upper muscles are always used together with their fellows of the opposite side (even winking requires assiduous practice and is not a natural act) and therefore receive a bilateral cortical supply. Consequently so long as the nuclei and the peripheral nerve-tracts are intact the muscles will work provided that any impulse can reach them from *either* cortex.

Emotional movements, such as laughing or smiling, remain relatively unimpaired in supra-nuclear lesions provided that the optic thalamus is not involved.

By reason of the close proximity of other pyramidal fibres supra-nuclear facial palsy is associated with paralysis of the homolateral arm and leg. A cortical lesion which involves the face area alone is of theoretical rather than practical interest.

(iv.) *Herpes* of the external auditory meatus may be combined with facial paralysis and is then supposed to be due to an inflammatory process affecting the geniculate ganglion.

VI. **The Eighth Cranial Nerve.** The eighth nerve is made up of two distinct sets of fibres, one of which, the vestibular nerve, originates in the vestibular ganglion and terminates peripherally in the sensory epithelium of the utricle, saccule and ampulla. Centrally these fibres end in the vestibular nucleus, whence they connect with Deiter's nucleus and so run to the vermis of the cerebellum. They are concerned with equilibration and form one of the main afferent cerebellar paths.

The cochlear nerve is the true nerve of hearing and arises in the spiral ganglion of the cochlea. Peripherally the cochlear fibres terminate in the organ of corti; centrally they run to the cochlear nucleus in the cerebello-pontine angle. From the cochlear nucleus two routes are taken :—

(*a*) The dorsal portion passes under the floor of the fourth ventricle through the fillet to the inferior corpus quadrigeminum and the internal geniculate body of the opposite side.

(*b*) The ventral portion runs through the base of the tegmentum pontis to the olive and thence also to the opposite corpus quadrigeminum and geniculate body.

From these structures the final auditory path runs directly to the auditory centre in the superior temporal convolution.

Lesions of the vestibular nerve cause a cerebellar type of ataxy, attacks of giddiness, and a tendency to fall towards the damaged side.

Lesions of the cochlear nerve cause nerve deafness which can be diagnosed by the tests of Rinné, Weber, and Schwabach.

(i.) *Rinné's Test.* With normal hearing it is possible to hear a vibrating tuning-fork held close to the ear after all sound has ceased to be perceived with the butt of the fork held on the mastoid process.

In nerve deafness the same effect is noticed, but in deafness due to ear disease no sound is heard when the fork is removed from the mastoid and held close to the ear.

(ii.) *Weber's Test.* A vibrating tuning-fork is held against the vertex and a healthy person hears the sound in both ears : if now one ear is stopped up he hears more sound in the ear so stopped, *i.e.*, he lateralises to the side on which air conduction has been interrupted.

A patient with nerve deafness lateralises of his own accord to the sound side, but one who is deaf from ear disease lateralises to the side of the deaf ear.

(iii.) *Schwabach's Test* is of great service when deafness is bilateral.

The test consists in comparing the time during which (*a*) the patient and (*b*) a normal person can hear a vibrating tuning-fork which is held to his vertex.

In nerve deafness the length of time sound can be heard by "bone conduction" is diminished or even absent; in deafness due to ear disease the length of time is increased beyond the normal.

Nerve deafness due to tumours in the cerebello-pontine angle or in the sheath of the nerve is associated with general signs of intracranial tumour and also with paralysis of neighbouring nerves, especially the fifth and seventh, as well as cerebellar signs, such as nystagmus, giddiness, and ataxy.

Nerve deafness may occasionally be due to cortical lesions on the left side affecting the superior temporal gyrus ;

more commonly word deafness only is the result of such a lesion.

Menière's Disease. (*a*) *Acute Labyrinthitis.* This is due to acute inflammation or hæmorrhage.

The symptoms are giddiness, vomiting, nystagmus towards the sound side, and such loss of equilibration from a sense of horizontal rotation that recumbency is necessary.

The patient is only comfortable when lying on the sound side.

The acute symptoms generally pass off within a week.

(*a*) *Chronic Labyrinthitis.* This may be secondary to very chronic middle-ear disease or to vascular changes, such as arterio-sclerosis.

In the former variety the chief symptoms are tinnitus and middle-ear deafness, followed after a distinct interval by paroxysmal vertigo, during which there is a sense of rotation of the body or of external objects.

In the latter variety there is also tinnitus and paroxysmal vertigo, and practically always more or less "nerve deafness," and usually a tendency to vomit after the attack of giddiness. Mild cases of this type may closely resemble "*petit mal*," but in epilepsy some alteration of consciousness is the rule, whereas it is rare in labyrinthitis, which is also favoured by demonstrable nerve deafness and the occurrence of vomiting.

VII. **The Ninth and Tenth Cranial Nerves.** The glosso-pharyngeal and vagus are in reality one mixed nerve, the motor part of which springs from the nucleus ambiguus in the formatis reticularis of the medulla.

The sensory parts terminate in the fasciculus solitarius and in the posterior vago-glosso-pharyngeal nucleus in the medulla.

The glosso-pharyngeal nerve does not appear to be paralysed by itself; when, however, it is affected in common with other nerves, as in bulbar paralysis, the symptoms are difficulty in swallowing from anæsthesia in the pharynx and, possibly, loss of taste for the posterior third of the tongue.

The stylo-pharyngeus muscle is supplied by the glosso-pharyngeal nerve, but paralysis of this muscle cannot be recognised clinically.

The vagus nerve supplies the pharynx, œsophagus, larynx, lungs, heart, stomach, and part of the intestines, it also sends branches to the dura mater and to the skin at the back of the external auditory meatus.

Hence complete bilateral vagus paralysis is immediately fatal. If, however, only one vagus is affected, there is little or no effect on the heart's action or on the respiration, and the most notable signs are unilateral paralysis of the soft palate with nasal voice and complete palsy for the half-larynx.

The recurrent laryngeal nerve supplies all the muscles of the larynx except the crico-thyroid, and this nerve is frequently affected by intra-thoracic tumours. The result is usually an abductor paralysis of the vocal cord on the left side. It is noteworthy that double adductor paralysis of the vocal cords is always functional.

VIII. **The Eleventh Cranial Nerve** (the Spinal Accessory). The bulbar or accessory part of this nerve is simply part of the vagus and arises from part of the vagus nucleus.

The Spinal part takes origin from the grey matter of the cord as far down as the sixth cervical segment.

This nerve supplies the sterno-mastoid muscle and combines with the cervical nerves proper to supply the upper part of trapezius. Disease of the upper two cervical vertebræ may involve this nerve, with the result that the head cannot be properly turned to the opposite side and the shoulder on the affected side cannot be shrugged, and tends to "drop."

IX. **The Twelfth Cranial Nerve** (the Hypoglossal). This nerve arises from a nucleus in the dorsal region of the medulla and emerges from the anterior surface of the bulb just external to the pyramids.

The real function of the hypoglossal nerve is to supply the muscles of the tongue; the sterno-hyoid, sterno-thyroid, and omo-hyoid are supplied through the anser hypoglossi by fibres coming from the upper cervical nerves.

Paralysis of this nerve from nuclear or infra-nuclear lesions causes wasting and wrinkling of the corresponding part of the tongue, while if both nerves are affected, as in

bulbar paralysis, it may be impossible to protrude the tongue at all.

In supra-nuclear lesions there is, of course, no wasting and, probably owing to bilateral cortical control, the deviation of the protruded tongue to the side of the paralysed limbs soon passes off.

Nuclear lesions may occur in bulbar paralysis, tabes, and syringo-myelia.

The nerve roots are frequently involved in basal meningitis, and the nerve may be affected peripherally in tumours of the neck.

CHAPTER III

NEURITIS

THE peripheral nerves may be affected in two main ways—first, by interstitial, and, secondly, by parenchymatous inflammation.

The sheath of the nerve may be affected (perineuritis), but this may be included in the interstitial group.

The above classification is perhaps rather arbitrary and may be misleading, since in interstitial neuritis there is secondary parenchymatous change and in parenchymatous neuritis the interstitial tissues may be affected also.

I. **Interstitial Neuritis** (Isolated Neuritis). This usually affects a single mixed nerve trunk and may result from trauma or from toxins from the alimentary tract, or it may occur in the course of prolonged and debilitating disease, such as cancer or tuberculosis, or in such other diseases as syphilis, rheumatism and gout, and lastly by prolonged pressure, as from tumours or in crutch palsy.

The symptoms are :—

(*a*) *Sensory*, varying from such subjective sensations as tingling or numbness to severe and intractable pain in the area of distribution of the affected nerve.

(*b*) *Motor*. Partial or complete flaccid paralysis of the muscles supplied by the nerve, with loss of reflexes, the reaction of degeneration, and later, possibly, contracture of the unparalysed muscle groups in the neighbourhood.

(*c*) *Trophic and Vaso-motor*. Thinning and glossiness of the skin, atrophy of the bones and loss of hair and nails, and ulceration. Frequently there is over-activity of the vaso-motor system in the early stages and cold, blue, dry skin in the later.

The diagnosis is not difficult if a tender nerve trunk can be felt and if the sensory disturbance corresponds to the anatomical distribution of the nerve. As stated previously,

there is considerable sensory overlap, so that the actual area of sensory disturbance is smaller than the full anatomical distribution of the nerve, and, further, the area of protopathic loss is negligible if cutaneous nerves alone are affected.

In all cases the neighbouring joints must be carefully examined to exclude the muscular paralysis that may follow from disuse in cases of arthritis: the electrical reactions are unaltered in these cases.

Certain special forms of interstitial neuritis may be discussed separately :—

A. Sciatica. The great majority of cases of true sciatica depend on a perineuritis somewhere in the course of the sciatic nerve. The disease is often hereditary and appears to be connected with gout, rheumatism, and exposure.

The symptoms and signs are :—

(*a*) Pain, usually dull and continuous for considerable periods, along the course of one sciatic nerve or its branches.

(*b*) Definite tender points along the course of the nerve, especially over the sacro-sciatic notch, behind the great trochanter, in the centre of the posterior aspect of the thigh, behind the head of the fibula, and behind the external malleolus.

(*c*) Definite increase of the pain when the sciatic nerve is stretched either voluntarily or passively.

(*d*) In severe cases there are contractions (flexion at the hip and knee, with the toes pointed to keep the heel off the ground) and also muscular wasting and more or less loss of epicritic sensibility over the outer side of the foot and leg. These phenomena of course depend on true neuritis and are not present in simple sciatic neuralgia.

The reflexes are preserved—indeed, they are often brisk unless there is a severe secondary parenchymatous neuritis.

Sciatica is nearly always unilateral, and the occurrence of bilateral sciatic pain should excite the suspicion of the possibility of some lesion in the pelvis or cauda equina.

In all cases of apparent sciatica the following conditions must be carefully excluded :—

(i.) *Malignant Disease of the Pelvic Viscera.* Abdominal palpation combined with digital and sigmoidoscopic

examination of the rectum, etc., will usually ensure a correct diagnosis.

(ii.) *Osteo-arthritis of the Hip-Joint.* The condition is frequently associated with sciatic pain and can nearly always be diagnosed by the X-rays.

(iii.) *Lesions of the Cauda Equina.* These produce characteristic *segmental* sensory disturbances, *flaccid* motor paralysis of muscles supplied by the particular *roots* involved, and complete incontinence.

(iv.) *The Lightning Pains of Tabes Dorsalis* can be diagnosed by the other signs of this disease.

B. Brachial Neuritis. This appears to be closely analogous to sciatica ; the pain may be severe, especially in the shoulder, and runs down the arm often to the tips of the first and middle fingers, though the whole hand may be affected.

Other sensory manifestations may vary from numbness and tingling in the extremities to peripheral anæsthesia.

Objective sensory loss is, however, relatively rare, and the same applies to motor symptoms, though slight paresis and wasting may be found.

The diagnosis of brachial neuritis depends on the exclusion of the following conditions :—

(i.) Aneurysm or cardiac lesions that could produce angina pectoris give their own physical signs.

(ii.) Osteo-arthritis of the shoulder and also of the spine can be excluded by the X-rays.

(iii.) Cervical rib can often be seen by the X-rays ; it tends to give a forearm paralysis, with special wasting of the small muscles of the hand owing to the involvement of the lower cord of the brachial plexus.

(iv.) Tabes dorsalis (*vide* p. 544) may give lightning pains limited to the arms.

(v.) Cervical caries can be detected by physical examination of the spine aided by X-rays.

(vi.) New growth of the bones forming the shoulder joint can usually be suspected by manipulation and confirmed by radiography.

C. The Anterior Crural Nerve may be affected in the same way as the sciatic nerve ; a tender spot can often be elicited in Scarpa's triangle.

II. **Parenchymatous Neuritis** (Multiple Neuritis). This is presumably a blood infection ; it is symmetrical, tends to affect many nerves, and may be produced by many poisons as follows :—

(*a*) *Metallic Poisons*, such as lead, silver, and arsenic.

(*b*) *Organic Poisons*, such as alcohol, carbon-bisulphide, and carbon-monoxide.

(*c*) *Toxins of Specific Infective Diseases*, such as diphtheria, beri-beri, measles, septicæmia, enteric fever, influenza, scarlet fever, gonorrhœa, and small-pox.

(*d*) *Morbid Blood States*, such as cancer, diabetes, and anæmia.

Since the clinical picture differs somewhat with the various toxic products which are the cause of multiple neuritis, the following varieties may be described briefly :—

(i.) Alcoholic Neuritis. This is the most common form of polyneuritis in England. It affects women more often than men, and is especially frequent in the secret toper as opposed to the periodic drunkard.

Three clinical varieties are described.

(*a*) *The Sensory Type.* Numbness and tingling are first noticed in the feet and later in the hands. These symptoms give way to or are later accompanied by cramps in the calf muscles, which become so severe as to entail the recumbent position. The muscles weaken and waste and trophic changes appear in the skin, though bed-sores are rare. The feet become dropped and the wrists flexed, since the extensors are weaker than the flexors.

The calf muscles are conspicuously tender.

There is peripheral diminution of all forms of cutaneous sensibility (sock and glove anæsthesia).

The deep reflexes are sluggish and finally disappear.

The superficial reflexes are sometimes increased at the outset, but tend to become diminished later.

The sphincters always escape, though wilful incontinence is sometimes met with in those cases that show marked mental change.

The cranial nerves nearly always escape, though the eye muscles show easy fatigue and sometimes a pseudo-nystagmus.

Tremor of the hands, lips, and tongue is frequent.

An important feature is the tendency (especially in women) for a curious mental change to supervene. This is known as Korsakow's syndrome, and consists in a blunting of moral sense and general mental inertia, combined with a loss of memory for recent events and a peculiar lack of appreciation of time and place.

(*b*) *The Motor Type.* The earliest symptoms are peripheral muscular weakness and sense of easy fatigue, while a "high stepping" gait is often adopted to prevent the foot dragging.

Peripheral paræsthesiae are usually present, though not obtrusive, but muscle tenderness, especially in the calves, is characteristically severe.

The reflex system behaves as in the sensory form, but trophic changes are less common and occur later.

(*c*) *The Ataxic Type.* The most striking feature of this variety is a loss of protopathic sensation as shown by inco-ordination of movements, loss of sense of passive position, and muscular hypotonus. Romberg's sign is well marked in these cases.

Muscular tenderness is always present, and this, together with the retention of the pupillary light reflex, serves to exclude tabes dorsalis.

It is important to remember the liability of patients suffering from alcoholic neuritis to suffer from cardiac, gastro-intestinal, pulmonary, and renal complications. There is also a special tendency towards the development of serous effusions, so that it may be stated that the most urgent risk in these cases arises from intercurrent maladies.

(ii.) Arsenical Neuritis closely resembles the alcoholic form as far as the nervous system is concerned; it may, however, be diagnosed from the history of exposure to infection, the severe gastro-intestinal symptoms, the pigmentation of the skin (not mucous membranes), and the cutaneous thickening of the palms of the hands and soles of the feet (*vide* also p. 205).

(iii.) Lead Neuritis. The motor nerves only are affected; the arms are involved earlier and more severely than the legs, while in many cases the posterior interosseous nerve

seems to be exclusively picked out. When the legs are affected the anterior tibial muscles are usually selected.

A dropped wrist from extensor paralysis is thus the most conspicuous feature, though very often *tremor of the hands* may be the earliest sign.

Other evidence of lead poisoning can usually be obtained (*vide* p. 204).

(iv.) Diphtheritic Paralysis. It is probable that the parenchymatous change is secondary to an anterior horn-cell involvement.

The characteristic selection of certain muscle groups has been described on p. 12.

(v.) Diabetic Neuritis. The legs are always affected earlier and more severely than the arms.

Subjective sensory changes are slight, so that the condition may escape notice, but investigation shows muscular tenderness and wasting, ataxy, and sluggish or absent reflexes.

The presence of glycosuria is an essential point in the diagnosis of this form of neuritis.

(vi.) Beri-beri (*vide* p. 116).

III. **Acute Toxic Multiple Neuritis.** The toxins of certain specific infections, such as gonorrhœa or influenza, as well as the exanthems and other less definite infective disorders, may occasionally produce an acute multiple neuritis characterised at first by peripheral sensory paræsthesiæ. These are shortly followed by muscular paresis, which can scarcely be described as peripheral rather than proximal, so extensive is its distribution. The trunk and face are often involved in this motor weakness.

In two or three days muscular wasting is apparent, and by now objective sensory changes have developed in the direction of muscle pains and tenderness combined with a peripheral loss of all forms of sensibility.

The sensory changes serve to distinguish this condition from Landry's paralysis, which it closely resembles in its initial stages.

CHAPTER IV

LUMBAR PUNCTURE AND THE CEREBRO-SPINAL FLUID

The cerebro-spinal fluid is secreted by the choroid plexuses; it fills the ventricles of the brain and occupies the pia-arachnoid space at the base of the brain and all down the spinal cord. The fluid gradually drains away along the nerve roots as they leave the spinal column.

The cerebro-spinal fluid is clear and watery in health; it is slightly alkaline and contains a trace of globulin, a trace of cholin, a trace of chlorides, a body (glucose) which reduces Fehling's solution, and one or two lymphocytes to the cubic millimetre.

An examination of the cerebro-spinal fluid is often of the greatest diagnostic value. The fluid is obtained by lumbar puncture, which is performed as follows:—

The patient, if well enough, is made to sit on the edge of the bed with his shoulders bent forwards as far as possible. If the patient is too ill to sit up he lies on his side and again bends forwards as much as possible. Babies must be forcibly flexed by pressure on the neck and buttocks.

A line is now taken joining the highest points of the iliac crests; this passes through the middle line at the level of the spinous process of the fourth lumbar vertebra, and from this landmark the third or fourth lumbar inter-vertebral spaces can be located. The skin is sterilised and a stout needle 4 inches long is passed at the level of one of these spaces (preferably the fourth) and $\frac{1}{2}$ inch from the middle line in a direction upward, inwards, and forwards till the back of the body of a vertebra is felt; the needle is now withdrawn $\frac{1}{4}$ inch and the fluid allowed to escape by its own pressure.

The first few drops may be neglected, especially if blood-

stained, but the rest should be collected in a sterile vessel for examination.

The diagnosis of the following diseases may be simplified by the examination of the cerebro-spinal fluid :—

(i.) *Tuberculous Meningitis.* The fluid is under pressure ; it is clear or faintly opalescent ; it is sterile, and shows no organisms ; it contains a considerable excess of lymphocytes (sometimes several hundred per cubic millimetre) ; there is sometimes a trace of albumin, and the reducing body is diminished but not often absent.

In very acute cases many polymorphonuclear leucocytes may be present instead of lymphocytes, and exceptionally a few tubercle bacilli may be seen in films.

(ii.) *Cerebro-Spinal Meningitis, Pneumococcal Meningitis, and Septic Meningitis.* The fluid is under pressure, turbid, contains an excess of polymorphonuclear leucocytes, definite albumin, and usually shows complete absence of reducing body. In addition the appropriate organisms can usually be seen in the films and cultivated on suitable media.

(iii.) *Hydrocephalus.* The fluid may be under pressure, and often contains much albumin but no cells.

(iv.) *Cerebral Syphilis.* The fluid contains an excess of lymphocytes, but gives a negative Wassermann reaction in over 70 per cent. of cases.

(v.) *Tabes Dorsalis.* The fluid contains an excess of lymphocytes and also gives a positive Wassermann reaction in 60 per cent. of cases. The globulin content is markedly increased.

(vi.) *General Paralysis.* The fluid contains an excess of lymphocytes and gives a positive Wassermann reaction in 95 per cent. of all cases. The globulin content is markedly increased.

(vii.) *Sleeping Sickness.* The trypanosomes can be demonstrated in films of the cerebro-spinal fluid (*vide* p. 135).

CHAPTER V

DISEASES OF THE BRAIN

I. **Hydrocephalus.** By this is meant damming up of cerebro-spinal fluid within the ventricles. Hydrocephalus may occur as a congenital abnormality; it may develop spontaneously at any time; or it may be secondary to intra-cranial disease, such as meningitis or tumour.

(*a*) *The Congenital Type.* The pathology of the congenital form is not known. The diagnosis is easy and can be made from the following signs :—

(i.) A large vault to the skull with a bulging forehead overhanging a wizened face in which the eyes are turned downwards.

(ii.) Patent sutures in the skull and prominent veins in the scalp.

(iii.) Impaired vision, and not infrequently optic atrophy.

(iv.) Fits and spastic motor paralysis.

(v.) Mental deficiency, should the child live long enough for this to be recognised.

(*b*) *The Acquired Type.* This appears to be due to an inflammation of the cells covering the choroid plexuses.

If it occurs in a young child the signs will be much as described for the congenital form; if, however, it does not occur until the growth of the skull has finished, the diagnosis is difficult. The classical features of cerebral tumour (headache, vomiting, and optic neuritis) are present; but hydrocephalus may sometimes be suspected by the occurrence of blindness at an earlier stage than is usual in cerebral tumour—that is to say, while the neuritis is acute and before there is any optic atrophy, by the early impairment of the power of upward movement of the eyes, by the impairment of the light reflex, by the fact that convulsive attacks, if present, do not always start in the same locality, as would be

expected in tumour, and by the general diffuse spasticity of the limbs.

(*c*) *Secondary Hydrocephalus.* In this the signs of the primary lesion rather overshadow the signs of the hydrocephalus, though in cases of tumour it may be possible to recognise the development of the special symptoms described as occurring in primary hydrocephalus. The essential features are the characteristic visual, oculo-motor and pupillary phenomena and the bilateral spasticity.

II. **Vascular Lesions.** (*a*) Intra-cranial Aneurysm. Two forms of aneurysm are found in the cerebral blood-vessels—first, multiple or miliary aneurysms, and, secondly, a single aneurysm of definite proportions.

Miliary Aneurysms are most frequent in the vessels supplying the basal ganglia, but may occur anywhere; they are not often met with before 40 years of age and do not give rise to any symptoms. They are, however, prone to rupture and so become a frequent cause of cerebral hæmorrhage.

If the aneurysm which ruptures is situated right at the base of the brain and not in its substance, the blood rapidly infiltrates the pia-arachnoid and spreads round the brain to the vertex, often without ploughing up the brain substance to any appreciable extent. In such cases there is little resistance to continued hæmorrhage and the rise in intra-cranial tension is very rapid, so that death often takes place with extreme suddenness.

Single Aneurysms do not give rise to any special symptoms by which they can be diagnosed with certainty. If they become of sufficient size there may be both general and localising signs of intra-cranial tumour. Any other symptoms that may be present can only be referred to concomitant arterio-sclerosis of the cerebral vessels.

(*b*) Cerebral Hæmorrhage. It seems certain that a healthy artery will not rupture no matter how high the blood pressure rises; therefore, in the mechanics of cerebral hæmorrhage, one must postulate a damaged vessel in addition to a raised blood pressure. Occasionally it may happen that a particular vessel is so diseased as to rupture with a subnormal blood pressure; but such cases must be rare, since

the chief cause of arterial damage is high blood pressure, and conversely any diffuse arterial lesion is likely to be followed by high blood pressure to compensate for impaired elasticity in the walls of the damaged arteries.

In more than half the cases of cerebral hæmorrhage there is a history of previous vascular cerebral troubles. In most cases these are probably thrombotic in nature, and the subsequent hæmorrhage is facilitated by the lack of support to the vessels which results from softening in the thrombotic area.

Hæmorrhage may occur in any part of the brain, but is most common at the base, and the arteries generally affected are the lenticulo-striate or lenticulo-optic branches of the middle cerebral artery.

The effect of a hæmorrhage within the skull is that of a foreign body or tumour, but one that has developed so quickly that no compensatory toleration is possible. In addition to the mechanical destruction of nervous elements, there is a pressure obliteration of veins and capillaries around the lesion ; this causes a noteworthy local increase in tension (probably to the height of the arterial pressure). Thus the symptoms of cerebral hæmorrhage are considered by Leonard Hill to be due to compression, which is itself the result of cerebral anæmia.

Pursuing this argument, it is obvious that the danger in these cases is anæmia of the medulla oblongata, and that, in fact, this bulbar anæmia is the cause of death in all cases of compression.

Cerebral hæmorrhage is more common in men than in women, and is most frequent between the ages of 45 and 55.

There may be premonitory symptoms, such as giddiness, throbbing in the head, headache (sometimes unilateral), or epistaxis ; often, however, the apoplectic stroke is the first sign.

More often than not the patient becomes unconscious at once, but a certain number of cases exhibit convulsive attacks for a varying time before consciousness is lost.

In all but the slightest cases the coma deepens until there is a completely flaccid condition of all four limbs with absence of all reflexes. The pulse is slow and full, the breathing is

stertorous, and at this time it is impossible to tell the sound from the paralysed side. The pupils may be unequal; that on the side of the brain lesion is usually the smaller.

The great majority of cases of cerebral hæmorrhage end fatally, often within a few hours, from rupture of the effused blood-clot into the lateral ventricle. When this rupture takes place unconsciousness becomes even deeper, there is often a convulsion and vomiting, the temperature rises quickly, the pupils contract, the breathing becomes of Cheyne-Stokes type, and death soon ensues.

Pontine Hæmorrhage is usually attended by very sudden and profound unconsciousness, though sometimes bilateral convulsions are noted. There is a bilateral flaccid paralysis and usually a high temperature. The pupils are generally tightly contracted, but towards the end they may dilate and assume an oval outline. Death usually occurs in from a few minutes to six or eight hours.

Cerebellar Hæmorrhage is relatively infrequent. A large hæmorrhage is accompanied by unconsciousness, but not by hemiplegia. Vomiting is persistent and other cerebellar signs, such as "rotation" and skew deviation of the eyeballs, may be noted. Small cerebellar hæmorrhages may be suspected by the sudden onset of cerebellar symptoms in a person free from internal ear disease.

(*c*) Cerebral Thrombosis. The essential factor for the occurrence of cerebral thrombosis is vascular degeneration, next to this a low blood pressure and feeble heart, and, lastly, morbid blood states, such as anæmia.

Two main classes of case can be recognised clinically—first, the syphilitic, in which a thrombotic hemiplegia occurs between the ages of 25 and 40 years (often within six years of the luetic infection), and, secondly the old and debilitated person with diseased vessels, feeble circulation, and low blood pressure.

Thrombosis tends to affect the same vessels as hæmorrhage with the noteworthy addition of the arteries supplying the temporo-sphenoidal lobe.

On the whole it may be stated that thrombosis is likely to be a more gradual affair than hæmorrhage, but this progressive train of symptoms often terminates in a sudden

stroke when the lumen of the vessel becomes completely blocked.

As in hæmorrhage, many of the premonitory symptoms are really referable to general arterio-sclerosis, but such symptoms as transient numbness or paresis may be attributed either to minute focal thrombosis, or more probably to temporary local cerebral anæmia from poor circulation in the damaged vessels.

Headache, drowsiness, torpor, and giddiness are not infrequently described a few hours before the absolute thrombosis takes place.

Loss of consciousness is by no means necessary in thrombosis; it may be absent throughout, or it may develop insidiously, keeping pace with a spreading hemiplegia. Convulsions are less frequent than in hæmorrhage.

Such localising signs as may be present depend upón the particular vessels affected: for example, thrombosis of—

(i.) The *middle cerebral artery* causes complete crossed hemiplegia and hemianæsthesia, and complete aphasia (sensory and motor) if on the left side.

(ii.) The *first branch of the left middle cerebral artery* causes paralysis of the opposite face and tongue as well as motor aphasia.

(iii.) The *second branch of the middle cerebral artery* causes paralysis of the opposite face and arm.

(iv.) The *anterior cerebral artery* causes progressive dementia.

(v.) The *basilar artery* usually causes paralysis of all four limbs and the muscles of articulate speech, while not infrequently the fifth, sixth, and seventh cranial nerves are involved also.

(vi.) The *posterior cerebral artery* causes hemianopia and crossed hemianæsthesia.

(*d*) Cerebral Embolism. Active endocarditis of the left heart is the most frequent cause of cerebral embolism; occasionally it may follow septic pulmonary conditions (*e.g.*, bronchiectasis). The left side of the brain is more often affected than the right.

The onset is sudden, and convulsions are much more frequent than in thrombosis or hæmorrhage.

The localisation is the same as discussed under "Thrombosis." As a rule only a small embolism is dislodged, so that the infarcted area of brain is small.

The diagnosis depends upon the recognition of a cardiac or (more rarely) pulmonary lesion, which is likely to be connected with embolism.

(*e*) Hemiplegia. Paralysis of one side of the body is the usual manifestation of an apoplectic stroke, whether it is due to hæmorrhage, thrombosis, or embolism.

The recognition of hemiplegia does not commonly present much difficulty, but at first (if the patient is profoundly unconscious) it may not be easy to say which side is paralysed, though this can usually be recognised by lifting the limbs and then letting them drop, when the *complete* loss of tone on the paralysed side is apparent.

Since the lesion is an upper motor neuron one, the ultimate paralysis will be of spastic type, but at first there is nearly always complete flaccidity with loss of all reflexes. Muscular rigidity does not commonly appear for two or three weeks. After the shock of the apoplexy has passed off the deep reflexes become exaggerated and an extensor plantar response appears. As degeneration of the pyramidal tract below the lesion progresses, the spasticity of the paralysed muscles increases until, in severe cases, the characteristic hemiplegic attitude is adopted. This attitude consists in adduction of the upper arm, flexion of the elbow, flexion and pronation of the wrist, and flexion of the fingers and thumb, the thigh is adducted at the hip, the knee is extended, the foot is inverted and the heel is raised from the ground.

Since the motor cortex of one side is often cut off completely from the brain stem and cord in cases of hemiplegia, there is an apparent anomaly in the fact that the trunk muscles, the ocular muscles, and the upper part of the face escape almost entirely. The explanation is that these muscles are accustomed to synergic action with their fellows of the opposite side and so receive a bilateral cortical innervation. The fact that emotional movement of the face is not lost to the same extent as voluntary movement depends on whether or not the posterior part of the optic thalamus is damaged.

Recovery of power in those who survive is always very

much greater than the extent of the initial paralysis might lead one to suspect; the leg is usually less affected and recovers more completely than the arm, whilst the fine movements of the hand are often permanently lost.

A certain number of cases exhibit post-hemiplegic athetosis (curious spontaneous movements of the hands and forearms, consisting in repeated rhythmical hyper-extension of the digits with a flexed wrist). Athetosis is rare in adult hemiplegias, but is common after cerebral paralyses of children; it probably depends on a lesion of the optic thalamus and indicates that no further recovery is likely.

The extent and distribution of the paralysis in a case of hemiplegia, as well as the presence of anæsthesia, aphasia or hemianopia, depends on the precise situation and extent of the lesion: for example—

(i.) Cortical or sub-cortical lesions are likely to produce a restricted paralysis with convulsions.

(ii.) Capsular lesions produce a complete contra-lateral hemiplegia.

(iii.) Aphasia can only be produced in right-handed persons by a left-sided brain lesion.

(iv.) Lesions far back in the internal capsule produce a hemianæsthesia and a hemianopia.

(v.) Lesions below the middle of the pons may cause a paralysis of the contra-lateral limbs with a peripheral type of facial palsy on the side of the lesion.

(vi.) Lesions in the crus may cause a contra-lateral paralysis of limbs and lower face with a homolateral oculo-motor palsy.

Differential Diagnosis. In attempting to diagnose the cause of a hemiplegia a general and comprehensive review must be taken of the case as a whole. The possibility of uræmia must be borne in mind and a catheter specimen of the urine examined for casts and albumin. The fundus oculi may show albuminuric retinitis, and it is noteworthy that a retinal hæmorrhage often occurs shortly before cerebral hæmorrhage and thrombosis.

Marked optic neuritis might suggest the possibility of a cerebral tumour, hæmorrhage into which may cause a sudden hemiplegia. Examination of the heart should be made to

exclude embolism, and, if there is marked hypertrophy, will confirm a diagnosis of chronic nephritis and so favour to some extent a diagnosis of hæmorrhage.

The state of the arteries and the blood pressure must be determined, though it must be remembered that any sudden intra-cranial disturbance is likely to be accompanied by an increased blood pressure, so that a high blood pressure, *per se* by no means excludes thrombosis.

The history of the manner in which the stroke occurred is all-important when it can be obtained ; the slower the onset and the slighter the damage the greater the probability of thrombosis. If there is little or no loss of consciousness, hæmorrhage is unlikely, while the younger the patient the more probable is it that the lesion is thrombotic ; and again, the very old and feeble are more liable to thrombosis than to hæmorrhage.

Lastly, two facts should be borne in mind—first, the great majority of cases of hemiplegia are due to thrombosis ; and, secondly, the great majority of cases of cerebral hæmorrhage are quickly fatal.

When a patient is seen for the first time in a state of profound unconsciousness and no reliable history can be obtained it may not be possible to demonstrate conclusively that the condition is one of hemiplegia, and the following possibilities must not be overlooked :—

(i.) Uræmic Coma.
(ii.) Diabetic Coma.
(iii.) Alcoholic Poisoning.
(iv.) Narcotic Poisoning.
(v.) Cerebral Tumour or Abscess.

Urœmia or *Diabetes* are suggested by an examination of the urine as well as by the smell of the patient.

Alcoholism gives dilated, equal pupils, and the patient can usually be roused by shouting or pinching the neck ; his breathing is slow, deep and regular, but very rarely stertorous.

Narcotic Poisoning (opium, etc.) often gives a smell of the drug used, the pupils are pin-point and equal ; the patient can sometimes be roused temporarily by shouting.

Cerebral Tumour or *Abscess* is suggested by a choked

disc, while abscess may possibly be suggested by otitis media.

(*f*) Local Cerebral Anæmia. When the cerebral arteries are greatly diseased the circulation through them becomes much hampered, with the result that a temporary lowering of blood pressure may cause a corresponding anæmia of that part of the brain which is supplied by the most degenerate vessels.

In this fact lies the explanation of those cases of transient hemi- or mono-plegia or aphasia which often last but a few hours and then recover completely.

The recognition of these cases is important, for they are especially likely to be followed by a real thrombotic attack sooner or later, and much can be done by judicious treatment to ward off such a disaster.

III. **Cerebral Palsies of Infancy and Childhood.** All the factors which cause hemiplegia in adults produce similar lesions in children: hæmorrhage, however, is peculiarly rare; embolism is much more common, since ulcerative endocarditis is not infrequent in children; thrombosis is not often seen, but may occur in association with any severe marantic disorder. It is probably the principal cause of the rare condition known as "porencephaly," when as the result of softening and subsequent growth of skull and brain large cyst-like cavities may be formed in the brain substance.

Apart from the above, infantile cerebral palsies may be divided into two groups—congenital and acquired.

(*a*) Congenital Cerebral Diplegia. This condition results from imperfect development of one or more neuron systems in the brain; the causes are quite unknown, though sometimes there is a familial tendency, but the signs may be present at birth or (more rarely) may not develop until later.

A bilateral spastic paresis is present, and this may affect the face and all four limbs. Both sides of the body need not be equally affected.

Mental deficiency to a greater or less extent is practically always found.

Optic atrophy is a fairly common accompaniment, as

is nystagmus, and other cranial nerves are sometimes affected.

The spasticity is often so marked that progression, when possible at all, is frequently by means of the " cross-legged " or " scissor " gait.

Athetoid movements of the hands and arms are nearly always a prominent feature of these cases, and probably depend on lesions of the optic thalamus or its connections.

(*b*) Acquired Cerebral Palsies. (i.) *Birth Palsies.* These, as the name denotes, are the result of injury at birth, sometimes by the blades of the forceps used in delivery.

The more permanent and severe forms are usually the result of meningeal hæmorrhage produced in this manner.

(ii.) *Encephalitis.* This is by far the most common form of acquired cerebral paralysis in infancy or childhood, and if the congenital, embolic, and thrombotic forms can be excluded it is the probable cause of all such cases.

Encephalitis is an acute but non-suppurative inflammation around the blood-vessels of the cerebral cortex. The micro-organism which causes this is the same as that which causes acute poliomyelitis in the cord, the only difference being the regions affected.

The most common age for encephalitis is up to three years.

The symptoms are those of any acute infection ; vomiting, pyrexia, and always convulsions ; the majority of patients lose consciousness, and coma may last for days or even weeks.

As the acute stage passes off more or less paralysis becomes evident, and this is of the upper motor neuron type. Perhaps the most common form is a hemiplegia or diplegia, but it is obvious that any variety and extent of cerebral paralysis may be met with, since any part or all of the brain may be affected. Athetoid movements are common in the cases in which permanent paralysis ensues.

Whether any of the paralysis is permanent or not depends on whether the inflammation was severe enough completely to destroy the nerve cells.

A very large proportion of children who have encephalitis become epileptic in later years, the epilepsy often being rather of Jacksonian type, in so far as the convulsions

are chiefly seen, or, at any rate, nearly always commence in the same place, that is to say, the paralysed limb.

IV. **Tumours of the Brain.** In children tuberculomas are the most common variety of cerebral tumour; in adults gliomas are more frequently present, and are followed in diminishing order of frequency by sarcomas, endotheliomas, tuberculomas, gummas, and carcinomas.

Cysts (echinococcal or cysticercal) may occur in connection with the ventricles, but are rarely seen in England.

Intra-cranial tumours can be divided into two main groups according to their anatomical position—first, supra-tentorial or anterior, and, secondly, infra-tentorial or posterior. The former are most common in adults, the latter in children.

Apart from special localising signs which will be mentioned later, it may be stated that tumours of the anterior chamber give rise to mental symptoms very much earlier and more frequently than those of the posterior chamber, and also that optic neuritis may more often be recognised as commencing on the side of the tumour in these cases.

The earliest sign of a tumour in the anterior chamber may be a loss of the superficial abdominal reflex and a tendency to briskness of the deep reflexes, on the opposite side of the body. Later on many of these cases give a contra-lateral extensor plantar response.

In tumours of the posterior chambers the spinal canal gets shut off from the cranium by the formation of a pressure cone, which forces the medulla into the foramen magnum earlier than in the anterior chamber tumours, with the result that internal hydrocephalus is more usual in these cases and failure of the medullary cardiac and respiratory centres is a more imminent danger. The deep reflexes may be brisk in the case of posterior chamber tumours, but the superficial are usually unaltered.

The signs and symptoms of brain tumours are divided into two groups—(i.) General, and (ii.) Localising.

(i.) General Symptoms—(*a*) *Headache.* This is probably the most constant symptom—it need not be continuous, and indeed may cease with extreme suddenness from time to time. The character of the pain varies—sometimes it is

lancinating, at others a dull ache. The situation of the pain is of but little value as a localising sign, though an occipital pain suggests a cerebellar rather than a cerebral tumour.

(*b*) *Vomiting.* This is not such a constant feature as headache; in its typical form it occurs quite suddenly without nausea or pain and independently of food.

(*c*) *Optic Neuritis.* This is the result of the cerebro-spinal fluid being forced by the increasing pressure into the sheaths of the optic nerves. It develops early in tumours of the cerebellum, later and more gradually in tumours of the cerebrum, and is not infrequently absent in tumours of the pons unless the ventricles themselves become obstructed by the growth.

(*d*) *Giddiness.* This may frequently accompany periodic attacks of headache and vomiting.

(*e*) *Mental Symptoms.* These are commonly present in tumours of the anterior chamber when the increased tension has become communicated to the opposite cerebral hemisphere. Irritability of temper and mental hebetude are perhaps the most usual signs. Of course a frontal tumour may give psychical symptoms as its earliest phenomena if such can be recognised.

(*f*) *Convulsions.* These vary from sensations of faintness to typical epileptiform seizures (Jacksonian or generalised), and are present in a fair proportion of cases, especially when the tumour is above the tentorium.

(ii.) Localising Signs.—(*a*) *Frontal Tumours.* The headache is sometimes frontal; optic neuritis appears late and is most marked on the side of the growth. Mental symptoms of all kinds, from lack of power of concentration to delusional insanity, are very liable to be present.

Convulsive attacks may start with jerking movements of the head and eyes to the opposite side; generalised convulsions with loss of consciousness may follow, but are not inevitable.

Attacks of pure motor aphasia, in which the patient utters unintelligible jargon, may occur in left-sided frontal tumours either alone or as precursors of general convulsions. Though the convulsive attacks may closely resemble epilepsy there is never a sensory aura.

(*b*) *Tumours of the Motor Area.* The general signs of tumour are usually well marked.

The localising signs are principally convulsive attacks whose commencement is local but which become general later; in the later stages such irritative phenomena always give way to signs of spastic paralysis in the contra-lateral limbs and face, according to the particular motor cells affected.

If the tumour is sub-cortical the convulsive attacks are less liable to occur.

Both psychic and sensory signs may well be present also, owing to the propinquity of the centres for these functions to the true motor area.

(*c*) *Tumours of the Sensory Area.* In addition to the general signs of tumour, there may be loss of sense of movement (active and passive) and loss of tactile sensibility. These losses are contra-lateral and most evident in the peripheral part of the limbs, and do not follow the segmental or the cutaneous distribution of the sensory nerves.

The remaining sensory appreciations may be affected little or not at all.

Convulsive attacks are Jacksonian in type and correspond to the adjacent precentral motor centres; they are preceded by local *numbness and tingling*, which shows that the disturbance commences in the post-central area.

(*d*) *Tumours of the Occipital Region.* The general symptoms are present; the headache is often occipital and the optic neuritis is often acute.

The localising signs are homonymous visual disturbance, culminating in hemianopia. The early signs are subjective and may simulate migraine. If the tumour grows forwards both word blindness (from involvement of the angular gyrus) and hemi-anæsthesia (from involvement of the posterior part of the internal capsule) may develop.

Convulsions, if present, may have a visual aura.

(*e*) *Tumours of the Temporo-sphenoidal Lobe.* The general symptoms are usually present, but are, perhaps, less constant than in the regions already dealt with.

It may be impossible to localise a cerebral tumour in

the temporo-sphenoidal lobe, but if certain parts of the lobe are involved the following signs may be present :—

(i.) Seizures or convulsions starting with an aura of smell (and usually an unpleasant smell) suggest a lesion of the uncinate lobe. Temporary loss of both smell and taste may follow the attacks.

(ii.) Subjective sensations of sound suggest lesions of the superior temporal gyrus ; if convulsions occur they may be limited to the opposite face and arm and may be followed by incomplete deafness in the opposite ear.

If the lesion is on the left side word deafness may be present.

(*f*) *Tumours of the Mid-Brain.* In addition to the general signs the following localising signs may be present :—

(i.) Contra-lateral hemiplegia, sometimes hemi-anæsthesia, and sometimes bilateral motor paralysis.

(ii.) Contra-lateral ataxy and rhythmical tremor.

(iii.) Defective light reflex and eccentric pupils.

(iv.) Weakness of upward movement of eyeballs.

(v.) Paralysis of third and fifth cranial nerves.

(*g*) *Tumours of the Pons.* General symptoms are striking in their absence or late appearance ; this is due to the fact that a slow-growing, diffuse glioma is the most common new growth in this region.

The localising signs may be—

(i.) Optic Atrophy without previous optic neuritis ; this is produced by pressure on the chiasma by a distended third ventricle.

(ii.) Paralysis of the fifth and twelfth cranial nerves.

(iii.) Eccentric pupils.

(iv.) Bilateral spastic paralysis, accompanied by ataxy.

(v.) Shivering attacks.

(vi.) Hemi-anæsthesia (not constant).

(vii.) Progressive hydrocephalus is frequently observed in children who have pontine tumours.

(*h*) *Tumours of the Cerebellum.*

(i.) Intra-cerebellar Tumours (lateral lobe). With the exception of mental symptoms and convulsions, the general signs of intra-cranial tumour are prominent ; the headache is severe, often occipital, and sometimes extending

down the neck. Optic neuritis appears early and advances very rapidly in both eyes. Giddiness is frequent, and generally there is a sensation that both the patient and external objects are rotating away from the side of the lesion. Nystagmus is always present; occasionally there is skew deviation of the eyeballs, but this is usually transient.

The nystagmus is, as a rule, only present on conjugate lateral movement; the movements are slow and deliberate (coarse) towards the side of the lesion, fine and hurried towards the opposite side.

The remaining cranial nerves are not specifically affected, except possibly the external rectus on the side of the lesion, which may show slight paresis.

Motor symptoms. There is slight general muscular paresis and distinct loss of muscle tone on the side of the lesion. There is ataxia on the side of the lesion. This results from loss of co-ordinating power for synergic movements; it is only present on movement and is most obvious in the trunk and legs.

The gait is reeling, and the patient tends to fall to the side of the lesion; to correct this he often rotates his body to the sound side, with the result that he may over-correct and so appear to stumble to the sound side. The attitude may be suggestive; the shoulder on the side of the lesion is raised and the occiput tilted down towards the raised shoulder, so that the chin is turned slightly upwards and outwards towards the sound side.

Sensory symptoms. These are absent.

Reflexes. The superficial reflexes are unaltered. The deep reflexes tend to be brisk, but there is not an extensor plantar response unless there is secondary pressure on the pyramidal tract.

(ii.) Intra-cerebellar Tumours (middle lobe). In these cases there is no lateralisation of symptoms. The tendency is to fall forwards or backwards; the nystagmus is equal to either side, and the motor symptoms are bilateral.

Owing to the proximity of the fourth ventricle spas-

ticity, tremors, internal hydrocephalus, and glycosuria may be added to the more definite cerebellar signs.

(iii.) Tumours of the Cerebello-pontine Angle. The eighth cranial nerve is usually the site of origin for these tumours. They grow very slowly, and may give rise to no signs till they press upon the cerebellum and pons. Hence headache appears late, and optic neuritis may be absent or inconspicuous for a long time.

The fifth, sixth, seventh, and eighth cranial nerves are very likely to be affected before there are general signs of intra-cranial tumour. Nerve deafness is often the first sign and facial paralysis the second.

When pressure is definite on both pons and cerebellum the signs are likely to be those of a homolateral cerebellar lesion combined with a contra-lateral spastic paralysis from involvement of the pyramidal fibres in the pons.

The sensory system usually escapes entirely.

The reflexes are important because the deep reflexes are increased, but especially so on the opposite side to the lesion, where also an extensor plantar response is found. The superficial reflexes are absent on the opposite side and quite brisk on the side of the lesion.

The combination of spasticity on one side and ataxia on the other is sufficiently striking to suggest the diagnosis. It is stated that these cases differ from intra-cerebellar tumours in the apparent direction of rotation of the patient during the vertiginous attacks. In intra-cerebellar lesions it will be remembered that the external objects appear to rotate towards the sound side, as does the patient himself, but in extra-cerebellar tumours the external objects rotate from the affected side to the sound side as before, although the patient feels as if he himself were rotating towards the side of the lesion.

V. **Abscess of the Brain.** This may be caused in the following manners :—

(i.) Trauma of the scalp or skull, and in this connection it is necessary to remember that direct injury of the brain or even fracture of the skull are not essential antecedents to the formation of an abscess in the brain.

(ii.) Suppuration in the middle or internal ear ; the

former is most likely to cause a temporo-sphenoidal abscess, the latter a cerebellar abscess.

(iii.) Suppurative processes within the nose, naso-pharynx, and accessory sinuses; these are more likely to produce a purulent basal meningitis, but may cause a frontal abscess also.

(iv.) Local septic processes (non-traumatic) of the scalp and skull—for example erysipelas.

(v.) General infections, such as septicæmia and more rarely the exanthems.

(vi.) Septic pulmonary conditions, such as bronchiectasis and empyema.

Brain abscess may be acute or chronic according to the virulence and nature of the causative organisms.

The temporo-sphenoidal lobe is affected more frequently than all the other parts of the brain put together, and the cerebrum is affected about twice as often as the cerebellum.

The *general symptoms* of brain abscess are in the main those of brain tumour, with the possible differences that mental symptoms are early and very constant and optic neuritis absent or late in appearing.

The temperature is variable; in many cases it is subnormal throughout or until the abscess bursts into the lateral ventricle.

High pyrexia is exceptional.

The pulse is slow and regular, even when there is distinct fever.

The initial *mental symptoms* of irritability and emotional instability gradually pass into mental hebetude and drowsiness, and eventually into coma.

The cerebro-spinal fluid may show an excess of polymorphonuclear leucocytes and even organisms, though the presence of these would suggest a coincident meningitis.

In diagnosing either the presence or position of a brain abscess a most important point is the existence of one of the conditions enumerated above as likely to be followed by abscess; for the rest the localising signs are very much those described under "Intra-cranial Tumour." We may again emphasise the early loss of the opposite superficial abdominal reflex in cerebral abscess. Retraction of the

head and pain down the neck are frequently seen in cerebellar abscess, and it is stated that one of the earliest signs of left temporo-sphenoidal abscess is the inability of the patient to name objects even though he knows their functions (one form of auditory aphasia).

VI. **Aphasia.** The art of speech is acquired coincidently with the development of certain centres in the brain. In right-handed persons these centres are situated in the left cerebral hemisphere. These special centres permit of the storing up of visual impressions, that is, impressions of objects seen and also auditory impressions, that is, the sound of words heard. In addition to these there must be a centre for the reception of impressions of the muscular movements concerned in the production of certain sounds, by means of which are correlated the sound of a word and the movements of the articulatory muscles necessary to produce that sound. These three centres thus comprise the sensory branch of speech.

The motor branch comprises the ordinary centres in the precentral region for the articulatory muscles and their connections with their corresponding lower motor neurons, and also higher centres situated in the intermediate precentral area, which are psycho-motor centres for spoken and written language.

The precise localisation in the left cerebral hemisphere of the various centres concerned in speech may be summarised as follows :—

(*a*) *Sensory* :—

(i.) Visual Word Centre, in the angular and supramarginal gyri.

(ii.) Auditory Word Centre, in the posterior part of the first temporal gyrus.

(iii.) Centre for Sense of Articulatory Muscle movement in the post-central gyrus fairly close to the Sylvian fissure.

(*b*) *Motor* :—

(i.) Primary motor centres for tongue, lips, larynx, hand, fingers, etc., in the precentral gyrus.

(ii.) Higher centre for spoken words (Broca's area) towards the base of the third frontal convolution.

(iii.) Higher centre for writing in the posterior part of the second frontal convolution.

The bulk of these centres (especially the sensory ones) lie in close relation to the Sylvian fissure, so that a thrombotic lesion of the Sylvian artery is certain to cause extensive aphasia, though the actual muscles concerned in speech are not affected.

Aphasia can be divided into two groups, first, Sensory, and secondly Motor, though a mixed aphasia may quite well occur.

A. Sensory aphasia may be found in lesions of either the visual or auditory word centres or in lesions involving the commissural fibres connecting these centres with each other or with the other speech centres.

Lesions of the Visual Word Centre cause word blindness, with inability to read, to write to dictation, or to copy printed or written words.

Nearly always, in addition, there is some difficulty in writing spontaneously, though this can often be accomplished without more serious error than the occasional use of wrong words.

If the path from the visual to the auditory word centre is interrupted, the patient is unable to read aloud unless he is a very strong visual and has a well-developed connection from the visual word centre to Broca's centre.

The connections from the visual word centre to the precentral and intermediate precentral motor speech centres are concerned respectively with copying and transferring printed to written language.

Lesions of the Auditory Word Centre cause word deafness—that is to say, inability to understand what is said though being able to hear quite well. A secondary result of word deafness is to produce unintelligible speech, not because there is any real motor aphasia, but because there is no storehouse of words to draw upon; the result is a medley of incomprehensible sounds which seems to the patient to be ordinary speech.

When the auditory word centre itself is intact, lesions of the commissural fibres connecting this centre with the other centres may produce aphasic symptoms as follows:—

Lesions of the path from the auditory word centre:—

(i.) To the chief visual centre in the occipital lobe causes inability to pick out named objects.

(ii.) To the *visual word centre* causes inability to pick out words named from a sheet of print or writing; at the same time the patient can read aloud.

(iii.) To the precentral motor areas causes inability to repeat spoken words.

(iv.) To Broca's area causes complete inability to carry on conversation, because, although the patient can understand what is said to him, the responsive impulses aroused in the auditory word centre are cut off from the higher centre for their spoken expression.

In like manner interference with the path to the higher centre for writing (cheiro-kinæsthetic centre) causes inability to write to dictation.

B. Motor Aphasia. In complete motor aphasia there is inability to speak or write, although the patient can understand what is said to him and can read.

(i.) Lesions in Broca's area cause inability to speak words, though there is no paralysis of the articulatory muscles.

(ii.) Lesions in the posterior part of the second frontal convolution cause inability to write *spontaneously*. Copying may still be possible.

(iii.) Lesions in the primary motor centres of the precentral gyrus cause a dysarthria from simple paralysis of the articulatory muscles.

When the speech centres are destroyed by a lesion of the left cerebral hemisphere before the age of three years there is a good prospect of developing, by education, similar compensatory centres in the right half of the brain.

After the age of three years this does not often occur.

VII. **Apraxia.** By this is meant the inability to use, properly, certain objects the name and use of which are perfectly understood by a patient who shows no signs of motor, sensory, or mental impairment. Apraxia is most evident in the case of objects which require considerable co-ordination of small muscles and muscle groups for their proper use, for example playing the violin or type-setting. Apraxia has been noted in lesions of the Corpus Callosum and also, more rarely, of the frontal lobes.

CHAPTER VI

DISEASES OF THE SPINAL CORD

I. **Hæmatomyelia** (Spinal Apoplexy). As a primary condition hæmorrhage into the spinal cord is nearly always traumatic. Secondary hæmorrhage may take place into a syringo-myelic cavity, into a myelitic area, or into a new growth.

The onset is sudden, and the signs become fully established within a few hours. There are neither convulsions nor unconsciousness.

The first sign is pain referred to the root areas which correspond to the lesion ; this may pass off in a few days.

Within a few hours (often a few minutes) there is complete sensory and motor paralysis (flaccid) below the level of the lesion. As time goes on the motor paralysis assumes the spastic form associated with pyramidal tract lesions, and tactile sensation, together with some sense of passive position, tends to return. Trophic changes are troublesome, particularly when the hæmorrhage is in the lumbar enlargement.

At the level of the lesion there is permanent flaccid paralysis from anterior horn destruction. This results in contracture deformities, which are especially well shown in the hands when, as is most often the case, the hæmorrhage is in the cervical enlargement.

All cases are not so extreme as implied in the above description ; sometimes only half the cord is affected, and in such cases Brown-Sequard's type of paralysis may be expected.

The chief difficulties in diagnosis may arise in cases of :—

(i.) *Acute Myelitis.* Here there is a more gradual onset, with some preliminary constitutional disturbance and no history of trauma.

(ii.) *Acute Poliomyelitis.* Here there is constitutional disturbance at the outset and usually a limitation of the

paralysis to the motor system ; further, the symptoms are not usually localised to a definite segmental level.

II. **Syringo-myelia.** By syringo-myelia is usually meant the development of cavities in the spinal cord as the result of degeneration in a diffuse gliosis of the grey matter of the cord.

It is a disease of young people (10 to 30 years), and in a considerable proportion of cases there is evidence of spina bifida to a greater or less extent.

Cystic degeneration may also occur in gliomas or sarcomas of the spinal cord ; in these cases there are the signs of intra-medullary tumour before those of syringo-myelia develop.

The principal seat of the gliosis is found in the great majority of cases to be the cervico-dorsal region of the cord, and the characteristic signs are to be looked for in the arms and chest. If, however, the growth spreads upwards bulbar symptoms are likely.

The diagnosis rests upon the following features :—

(i.) *At the level of the lesion* :

(*a*) Bilateral impairment or loss of the sensations of heat and cold and pain (not necessarily strictly segmental), with preservation of tactile sensibility. In very late cases all forms of sensation may be abolished.

(*b*) Bilateral atrophic paralysis of various muscles or muscle groups, the weakness being often in excess of the atrophy. This paralysis has no constant distribution or order of development, but is most often seen in the small muscles of the hands, the ulnar border of the forearms, and the shoulders. There may be no muscular atrophy even at a late stage of the disease.

(*c*) Trophic and vaso-motor changes. There is often passive hyperæmia with a partial sweat secretion. This œdematous condition produces the "*main esculente*" or succulent hand, which is of considerable diagnostic importance.

In other cases painless whitlows appear, and may produce a most extensive cellulitis of the hand and forearm (Morvan's disease). Perforating ulcers, arthropathies similar to Charcot's joints, and spontaneous fractures are other common trophic changes.

Dorsal scoliosis from weakness of the spinal muscles is nearly always present at some stage of the disease.

(ii.) *Below the Lesion.* There is slowly developing spastic paralysis, often with progressive loss of sensation. Some grade of spastic paraplegia from pressure on the pyramidal tracts can nearly always be detected, though, unless the lumbar enlargement is affected, there is rarely any sphincter trouble.

In many cases the earliest symptoms of all are subjective sensations of heat and cold in the arms, which precede the objective changes for a short time.

It must be remembered that different cases vary very much in the exact nature and distribution of their dissociated anæsthesiæ ; for example, there may be no loss of pain sensibility, or if present it may not coincide in distribution with the area of thermal changes. Again, the distal changes which result from the interruption of afferent and efferent tracts is commonly much more marked on one side of the body than the other, owing to the asymmetry of the new tissue formation in the cord.

There is usually no difficulty in diagnosing a case of syringo-myelia, since there is no other condition which causes the same symptoms extending over so long a period of time.

Cervical rib is unilateral and causes no cord symptoms, while the sensory loss is of the peripheral type.

Intramedullary tumour may closely resemble syringomyelia, but the symptoms are largely unilateral for some time, and unilateral spastic phenomena below the lesion, with extensor plantar response and sphincter disturbance, are much more prominent.

III. **Acute Myelitis.** This is a rare condition and signifies an inflammatory affection of the actual tissues of the cord, the result of a definitely infective process, though not due to any one specific infection : for example, it may occur primarily following a chill or for no known cause, or it may be secondary to such infections as small-pox, influenza, enteric fever, or gonorrhœa. An interesting form of myelitis is that which sometimes develops during the course of a severe cystitis. The term " Myelitis " is not conspicuously accurate, for it is evident that the meninges are nearly

always involved also in view of the extreme frequency of pain as an early symptom.

The most common condition to which the name myelitis is applied is the result of thrombosis in the spinal arteries secondary to syphilitic endarteritis; this condition is described in the section dealing with syphilis of the central nervous system.

Acute myelitis occurs in two main forms—the focal type, or Acute Transverse Myelitis, and the disseminated type, or Acute Ascending Myelitis.

It sometimes happens that the infection is so acute that it proceeds to abscess formation within the cord.

(i.) *Acute Transverse Myelitis.* There are the general symptoms of an infective disorder, fever, malaise, and pain in the back, which merge rapidly (that is to say in a few hours) into the clinical picture of myelitis.

The earliest special symptoms are subjective sensory symptoms, pain at the level of the lesion, and numbness and tingling of the feet and legs.

These sensations are replaced by a rapidly progressive loss of sensation and muscle power below the lesion, with complete loss of sphincter control. The motor paralysis is of the completely flaccid type with absence of all reflexes. Bed-sores and trophic changes are particularly troublesome.

The sensory and motor changes in the limbs usually proceed hand in hand, but sometimes one may be obviously in advance of the other. Cramp-like pains in the legs are by no means uncommon while the paralysis is developing.

The inflammatory process usually involves two or three spinal segments about the level of the 9th dorsal vertebra, and at the upper level of the lesion there may be persistent hyperæsthesia and constricting girdle pains.

If the lesion does not affect the entire thickness of the cord the paralysis of the limbs tends to be spastic and the plantar response extensor.

In the few cases in which the patient survives a complete acute transverse myelitis, a typical spastic paraplegia develops, with a notable tendency for the development of contractures of the adductor and flexor muscles.

(ii.) *Acute Ascending Myelitis.* The onset and symptoms

are the same as in acute transverse myelitis, but the disease does not remain limited to a definite level in the cord and can be observed to progress segment by segment higher up the cord.

The great majority of these cases end fatally by extension upwards of the inflammation to the bulb. Occasionally the upward spread appears to stop suddenly, and in such cases a fair recovery may be made.

Buzzard considers that the essential lesion in these cases is a spinal lymphangitis.

Post mortem the inflammatory areas may be focal and disseminated or diffuse and continuous. Clinically it has been noted that the progress is not always steadily up the cord segment by segment, but that occasionally several segments are skipped.

IV. **Tumours of the Spinal Cord.** (Intra-medullary Tumours.) Sarcomas, gliomas, tuberculomas, and gummas may be found in the spinal cord. Gummas do not arise in the actual tissues of the cord, but in the membranes or around the blood vessels.

The symptoms of an intra-medullary tumour naturally vary with the position of the tumour, but, speaking generally, are as follows :—

(i.) Segmental sensory dissociation, as in syringo-myelia.

(ii.) Unilateral cord symptoms, often of the Brown-Sequard type.

(iii.) Atrophic muscular paralysis of segmental distribution, with atrophy in excess of weakness (*cf.*, syringo-myelia, in which the converse obtains).

(iv.) Early sphincter trouble and extensor plantar response.

As the disease progresses bilateral spastic paraplegia develops, with progressive sensory loss below the level of the tumour.

The resemblance of intra-medullary tumour to syringo-myelia is striking but its duration is very much less, being rarely more than two to three years. The persistence of *unilateral* cord symptoms for a considerable time is an important differential point. The trophic changes and scoliosis of syringo-myelia should also be remembered.

V. **Diver's Paralysis** (Caisson Disease). This is the result of a too rapid return to the normal atmospheric pressure after exposure to a markedly increased pressure as in a diving-bell. While in the diving-bell the diver's blood becomes super-saturated with gases by reason of the extra pressure, and bubbles of gas (probably nitrogen) escape from the capillaries into the spinal cord if the diver is not permitted to return to normal pressure very gradually.

The effect of free gas in the spinal cord is to destroy the nervous tissues, and the symptoms vary with the amount of gas that is liberated. The principal changes are usually in the dorsal region.

In severe cases there are the signs and symptoms of a complete transverse lesion of the cord.

In less severe cases there are numbness and tingling of the extremities, severe pains in the joints, sometimes girdle pains and vomiting, motor weakness in the legs, and sometimes diffuse blunting of sensation.

Headache, giddiness, and deafness are commonly complained of in addition to the more special spinal symptoms.

VI. **Compression of the Spinal Cord.** The spinal cord may be compressed by fractures or dislocations of the vertebral column by new growth or tuberculous caries of the vertebræ or by new growth of the membranes.

The symptoms of compression of the cord fall into three groups, the prominence of each group varying with the nature of the primary lesion :—

(i.) *Symptoms due to disturbance of Nerve Roots.* A common symptom is intense pain over the area supplied by the damaged root. Cutaneous hyperæsthesia may be present as well, but sometimes this gives way to tactile anæsthesia, even though the pains persist.

If the anterior nerve roots are involved there is weakness and wasting of muscles supplied by these roots ; the anterior root symptoms are, however, often unobtrusive. Pain, though a constant symptom, is in many cases not referred to the areas typical of nerve root distribution ; it often resembles rheumatic pains in the neighbourhood, or it may be localised to the spine, to a joint, or to the breast.

(ii.) *Cord Symptoms.* Progressive paraplegia, most

marked at first on the side of the root symptoms, with sphincter disturbance and gradual loss of sensation below the lesion as the pressure increases.

The paralysis is spastic in type and the plantar reflex is extensor, but if the cord becomes completely destroyed physiologically the paralysis becomes flaccid.

(iii.) *Signs of Local Bone Disease.* Spinal curvatures and angular deformities are common in tuberculous disease. A tumour of the vertebræ may be palpable.

Local tenderness on percussion or manipulation, stiffness, and limitation of movement are very constant phenomena in caries, while severe local pain, much increased by movement, is often found in vertebral new growth.

As would be expected, these signs of local bone disease are rarely found in meningeal tumours unless the upper cervical region is involved.

It is worthy of note that in cases of paraplegia due to compression from tuberculous disease of the spine subjective root symptoms are often conspicuously absent.

The differential diagnosis between extra-thecal and intra-thecal spinal meningeal tumours is usually impossible, but it may be stated that, as a rule, an intra-thecal tumour causes early root symptoms and early compression, which for a time is to some extent unilateral, while with extra-thecal tumours compression is of slower onset, and when it does occur is not so likely to give unilateral signs.

Meningeal tumours must be differentiated from—

(*a*) *Aortic Aneurysm* by the physical signs of this disease and by the X-rays.

(*b*) *Intra-medullary Tumours* by the early dissociation of tactile and thermal sensibility, by the absence of root pains, and by the more conspicuous muscular atrophy.

(*c*) *Tuberculous Disease of the Spine* by the rigidity, impaired mobility or deformity of the spine, by the *local* tenderness of the spine to percussion, by the excess of motor over sensory symptoms, by the tendency to abscess formation, and by the younger age of the patient and possible evidence of tuberculous disease elsewhere.

(*d*) *Malignant Disease of the Vertebræ* by the possible history of a previous operation for carcinoma, since verte-

bral growths are usually secondary, by the extreme local pain and tenderness on manipulation, by the severity of the root pains, which are often increased by movement, and possibly by the presence of palpable nodules of growth. The remission of the pain with absolute rest is striking feature of many of these cases.

CHAPTER VII

SYPHILITIC DISEASES OF THE NERVOUS SYSTEM

THE frequency with which hemiplegia and kindred conditions may result from thrombosis of the cerebral arteries due to syphilitic endarteritis has already been discussed, but, apart from this, syphilis is the cause of several other definite clinical nervous diseases which will now be considered separately under the following headings :—

I. Cerebro-spinal Syphilis (excluding Hypertrophic Pachymeningitis and Meningo-myelitis).

II. Syphilitic Spinal Pachymeningitis (Hypertrophic Pachymeningitis).

III. Syphilitic Meningo-myelitis.

IV. Tabes Dorsalis (Locomotor Ataxy).

V. General Paralysis of the Insane (Progressive Paralytic Dementia).

I. CEREBRO-SPINAL SYPHILIS.

The essential lesion is gummatous formation.

(*a*) **Localised Cerebro-spinal Syphilis.** A single gumma is rare, but may cause symptoms of tumour either of the brain or spinal meninges.

Lepto-meningitis in the skull may cause severe headache and, if situated over the motor cortex, Jacksonian convulsions or even muscular paralysis.

Lepto-meningitis in the cord is generally associated with, and indeed may cause, local myelitis ; occasionally it may simulate an intra-thecal tumour and cause compression paraplegia by damming up the cerebro-spinal fluid.

Pachymeningitis in the skull is seldom strictly localised ; more often it is diffused over the base of the brain. In

the spinal cord it is commonly localised to the cervical and lumbar enlargements (*vide infra*).

(*b*) **Diffuse Cerebro-spinal Syphilis.** This results from a more or less diffuse gummatous infiltration of the membranes; the affection is often limited to the membranes within the skull, but signs of spinal meningitis may be present also.

The most striking and important signs are paralysis of one or more of the cranial nerves: the third and sixth nerves are specially picked out; unequal, irregular pupils with imperfect reaction to light are nearly always present.

Convulsive attacks, often of Jacksonian type, are common, as are the more general signs of gross intra-cranial disease, headache, vomiting, mental impairment, and even optic neuritis.

The resemblance to general paralysis of the insane is often striking, but the peculiar mental change is lacking, as is the tremor and the slurring speech. Further, cranial nerve paralyses are evident much earlier and the cerebro-spinal fluid gives a negative Wassermann reaction in the majority of cases.

II. SPINAL PACHYMENINGITIS (HYPERTROPHIC PACHYMENINGITIS).

This is usually limited to the cervical and lumbar enlargement, often to the former alone.

The earliest symptoms are sensory, and consist of pain in the back of the neck or lumbar region, worse on movement, and root pains owing to the involvement of the issuing posterior nerve roots. These pains at first are most marked on one side, but soon become bilateral. Hyperæsthesia is usually present, and in advanced cases anæsthesia may develop.

After a varying time, sometimes not for months, atrophic paralyses develop, at first in the small muscles of the hands and feet and in the flexor groups. A characteristic contracture of the hands often develops; the wrist and fingers are extended and the interphalangeal joints are flexed.

In severe cases signs of spastic paraplegia occur from compression of the cord.

There is a lymphocytosis in the cerebro-spinal fluid in this as in all syphilitic affections of the central-nervous system.

III. SYPHILITIC MENINGO-MYELITIS.

Although there is always some co-existing spinal lepto-meningitis, the essential cause of this condition is myelitic softening, the result of thrombosis in the spinal arteries. The lower dorsal region is most often attacked.

There are nearly always preliminary subjective sensory symptoms, such as numbness and tingling in the feet and legs; these, however, may be very slight, and the fact that they are present may only be ascertained by careful questioning. The striking feature of these cases is the onset of paraplegia, which usually develops quite suddenly.

In the great majority of cases the paralysis is spastic in type, the deep reflexes are increased, and the plantar reflex is extensor. If the lesion is so extensive as to amount to a complete transverse lesion, the paralysis is utterly flaccid, but this is rare.

At the level of the lesion there is a band of hyperæsthesia, and all forms of sensation are impaired or lost below this. The sharp limiting line between normal and disturbed sensation is rather characteristic.

Sphincter trouble (retention of urine and incontinence of fæces) develops with the paraplegia. Bedsores are likely to appear and in the more extensive lesions may prove very troublesome. The prognosis is fairly good, but as recovery takes place muscular spasms are often unpleasantly prominent.

Syphilitic Meningomyelitis can be distinguished from—

(i.) *Acute Transverse Myelitis* by the absence of constitutional disturbance, the probability of the paralysis being spastic from the commencement, and the relative absence of pain.

(ii.) *Disseminated Sclerosis* by the absence of nystagmus, altered speech and intentional tremor, and the presence of sensory changes.

(iii.) *Subacute Combined Degeneration* by the character

and distribution of the sensory changes and by the blood picture (*vide* p. 556).

Lastly, as in all forms of cerebro-spinal syphilis, the characteristic eccentric, unequal, sluggish pupils may be found. The Wassermann reaction to the blood and cerebro-spinal fluid is not as informative as might be expected, since a considerable proportion of presumably syphilitic cases give negative reactions to both. There is, however, a considerably higher percentage of positive reactions to the blood in meningo-myelitis than in ordinary cerebral syphilis.

IV. TABES DORSALIS.

The essential change in this disease is a progressive degeneration of the sensory fibres as they enter the cord through the posterior nerve roots and as they travel up the cord.

The disease starts in the lumbo-sacral region as a rule, but the cervical region is commonly affected also, as well as the intervening dorsal region.

The degeneration is most marked in the posterior columns of the cord. The posterior root ganglia escape, as do the nerve roots outside the cord. The anterior columns are affected to a certain extent in a small proportion of cases.

The manner in which syphilis produces this characteristic lesion is not yet understood ; men are much more frequently affected than women (10 : 1), and the common age incidence is 30 to 40 years.

The clinical manifestations of tabes dorsalis are very numerous ; no single one is absolutely indispensable, and as their order of development is not altogether characteristic they are best considered independently.

(i.) **The Cranial Nerves.** Temporary or permanent, partial or complete, ophthalmoplegia is sometimes present.

Laryngeal paralyses (abductor) are common, as is anæsthesia of the palate.

By far the most important cranial nerve sign is connected with the pupil. As in general paralysis and cerebro-spinal syphilis, the pupils often show irregularity of outline and inequality. In addition there is impairment or loss of the

light reflex, while the power of contracting to accommodation is preserved (the Argyll-Robertson reaction). This phenomenon is present in about 70 per cent. of all cases of tabes. It is to be noted that the consensual light reflex may be present when the direct reflex is gone.

The essential point is a sluggish (not necessarily absent) contraction to light in a pupil of irregular outline, provided that the irregularity is not due to old iritis. The eyes must be tested separately, and a good light must be used.

Primary optic atrophy occurs in 10 per cent. of all cases of tabes.

(ii.) **Motor System.** *True wasting* from anterior horn-cell degeneration occurs in a small proportion of cases.

Hypotonus of the muscles is a conspicuous feature of most cases. The ligaments are affected as well, and hyperextension of the limbs with general increased arthritic mobility can nearly always be demonstrated.

Ataxy may develop early or late; the muscular incoordination is the result of the loss of sense of position of the muscles and joints. At first this is compensated for by increased visual activity (the patient looks where his limbs are), and therefore is most noticed in the dark.

Ataxy is shown by inability to walk along a straight line; a stamping, unsteady, exaggerated gait, in which the legs are kept wide apart and the feet raised too high; inability to stand with the feet together and the eyes shut (Romberg's sign), and inability to perform the finer movements which require most muscular co-ordination, such as writing, threading needles, fastening buttons, etc.

(iii.) **Sensory System.** (*a*) Subjective. "*Lightning*" *pains* of paroxysmal nature shooting down the arms and legs; these appear to be just beneath the skin and may affect the joints as well as the limbs.

Deep pains referred to the bones or muscles, which are not paroxysmal, but are often persistent for months.

Girdle pains: band-like sensations of compression, pain, heat, and numbness.

(*b*) Objective. Loss of painful pressure sense, loss of sense of position, loss or blunting of cutaneous pain, and to a much less extent of tactile sense, most evident along

the ulnar border of the forearms, the thorax, the perinæum, and the legs and feet.

(iv.) **Reflexes.** *The superficial abdominal reflexes* are preserved.

The Tendon Reflexes in the lower limbs are diminished at first and eventually lost.

The Tendon Reflexes of the arm are lost if the cervical cord is affected.

The Organic Reflexes. There is often a frequent and urgent desire to pass water, but when the patient tries he is at first unable and then soon after passes it in a hurry, often involuntarily.

The Generative Functions. At first there is increased sexual desire and capacity; this does not last long and is followed by impotence and lack of all desire.

(v.) **Visceral Crises.** (*a*) *Gastric Crises* consist of attacks of nausea, vomiting, and severe abdominal pain, which may last for several days.

(*b*) *Laryngeal Crises* consist of sudden laryngeal spasms, with pain, stridor and a feeling of suffocation.

(*c*) Rectal, vesical, intestinal, cardiac, and respiratory crises are also described.

(vi.) **Trophic Changes.** (*a*) Progressive emaciation is nearly always to be recognised.

(*b*) *Charcot's Joints.* Joint changes follow relaxation of the ligaments and trifling injuries. The pathological changes closely resemble those in osteo-arthritis, with the addition of large effusions into the joints. There is very increased mobility of the joints, and the limb may become absolutely flail-like. Dislocations may occur. There is no pain.

(*c*) *The Tabetic Foot.* Changes in the tarsal ligaments cause the arch of the foot to drop and the foot to become everted.

(*d*) *Perforating Ulcers.* These commence as a corn, usually at the base of the great toe or the little toe. A painless ulcer next forms which extends deeply into the tissues of the foot and eventually exposes the bone. Similar ulcers sometimes occur in syringo-myelia and diabetes.

(*e*) Spontaneous fractures of the long bones are sometimes found.

(vii.) **The Cerebro-spinal Fluid** contains a large excess of lymphocytes (about 150 per cubic millimetre), but only gives a positive Wassermann reaction in 60 per cent. of all cases (*vide* p. 511).

The diagnosis of tabes dorsalis does not commonly present much difficulty provided that a conscientious examination of the entire nervous system is made.

It must not be imagined that every case of tabes gives all or even many of the signs just described, but some can practically always be found which are sufficiently characteristic for a positive diagnosis.

The most constant and earliest as well as most important signs are probably—

(i.) Impairment of light reflex (especially in an eccentric pupil).

(ii.) Impairment or absence of knee-jerks.

(iii.) Loss of deep muscle sensibility in the calves of the legs.

(iv.) Some impairment of pain sensibility, especially in the ulnar border of the forearms.

(v.) Urgency or precipitancy of micturition.

(vi.) Romberg's sign, as well as other evidence of ataxy, is very significant when present, but *may* not develop till late.

Lightning pains must not be mistaken for rheumatism or neuritis. The "neuralgic" symptoms may continue for months or years with no other symptoms, but pupil changes are nearly always present.

Gastric crises have before now led to laparotomy, but other signs of tabes can be found if sought for.

There are some grounds for believing that the cases in which optic atrophy develops early may remain relatively free from ataxic signs for long periods.

Juvenile Tabes Dorsalis develops in a very small proportion of cases of congenital syphilis or of syphilis acquired in infancy.

The disease occurs more frequently in girls than boys and starts just before puberty. The clinical manifestations are the same as in the adult form, with the exception that optic atrophy is of much more common occurrence.

Congenital tabes may at first be mistaken for Friedreich's disease, but in this there are no pupil changes and there are nystagmus, scoliosis, club-foot, and an extensor plantar response (*vide* also p. 563).

V. GENERAL PARALYSIS OF THE INSANE (Progressive Paralytic Dementia).

As tabes dorsalis is to the spinal cord so is general paralysis to the brain—that is to say, there is a progressive degeneration of the cortical cells and neurons, the result of syphilis, though often not developing for many years after the luetic infection.

The spirochæte of syphilis has been demonstrated in the cortex of the brain, and practically all (over 95 per cent.) of these cases give a positive Wassermann reaction to both blood serum and cerebro-spinal fluid.

The association in the same patient of general paralysis with tabes dorsalis is by no means infrequent (taboparalysis).

Men are affected considerably more often than women; the age incidence is that of tabes (35 to 50 years).

Microscopically there is degeneration of the cortical tissues, of the basal ganglia, and also of the pons, medulla and cord. The arterioles are thickened, and often there is a perivascular infiltration of small round cells.

The cerebro-spinal fluid contains an excess of lymphocytes.

The symptoms are best considered under the headings of the various systems :—

(i.) *Mental.* These are usually the first to appear, and consist in an alteration of the pre-existing mental habit, which is most striking when a person of orderly behaviour becomes guilty of gross immorality, petty thieving, or the like.

Very striking and often early to appear are inability to concentrate the attention, a condition of exaltation, a sense of self-satisfaction, and a ridiculous desire to spend or give away large sums of money.

Rather less commonly hesitation, irritability, depression, and even melancholia are the leading features.

The mental changes progress steadily until a state of complete dementia with entire loss of memory and lack of interest in all surroundings is reached.

(ii.) *Cranial Nerves.* The pupil changes described under "Tabes Dorsalis" are found in general paralysis, with the exception that the *complete* Argyll-Robertson phenomenon is rather less common. Optic atrophy is liable to occur, as well as, occasionally, partial or complete ophthalmoplegia (from basal meningitis).

(iii.) *Motor System.* A fine, rapid tremor develops early in the lips and tongue and spreads later to the hands and arms. The speech becomes slurred and elisive, a feature which is best shown by asking the patient to say sentences involving the use of tongue and lips, such as "The Royal Irish Constabulary extinguishes the conflagration." Voluntary power gradually diminishes and becomes completely absent in the latest stages, though at first there tends to be some muscular hypertonus.

Epileptiform seizures are a prominent feature of many cases; they may be Jacksonian in type, generalised or resembling *petit mal*, and followed by asphasia.

Each convulsive attack tends to leave the general mental condition worse than before.

(iv.) *Sensory Symptoms.* There is no constant sensory change, but diffuse blunting of pain sensibility may be observed.

(v.) *The Reflex System.* The knee jerks may be increased throughout, or after an initial increase they may diminish later. In tabo-paralysis they are usually absent.

The superficial reflexes are preserved and the plantar reflex is of the flexor type.

The sphincters are affected quite early, as in tabes.

Sexual power may be notably increased, but equally often is lost.

Diagnosis. It will be seen that the clinical picture is characteristic in well-marked cases. Difficulty may arise in the following cases:—

(*a*) *Neurasthenia.* There are no pupillary changes and no speech alteration.

(*b*) *Cerebral Syphilis.* There is no exaltation, no speech

alteration, and usually there are definite cranial nerve palsies.

(*c*) *Tumours of the Frontal Lobe* (*vide* p. 524).

(*d*) *Chronic Alcoholism.* The history of alcohol and the condition of the pupils are the most important points.

In any case of doubt, examination of the cerebro-spinal fluid should be undertaken, since it affords positive information of the presence of either general paralysis, tabes dorsalis, or cerebral syphilis (*vide* p. 511).

Juvenile General Paraylsis. This presents all the features of the adult disease ; it usually develops between the ages of 8 and 18, and always in congenital syphilitics.

The stigmata of congenital syphilis (*vide* p. 73) can generally be recognised, and in addition there is often more or less mental deficiency.

CHAPTER VIII

GENERAL DISEASES OF THE NERVOUS SYSTEM

I. **Disseminated Sclerosis.** This is a chronic progressive disease, the essential lesion being the occurrence of scattered patches of sclerosis throughout the brain and cord.

The disease usually starts in young adult life, but may not begin until after 40 years of age.

The sexes are affected almost equally, men being slightly more liable than women.

The pathology is obscure; the individual patches of sclerosis are sharply defined and are most commonly situated in the mid-brain, centrum ovale, and about the lateral region of the spinal cord, though they may develop anywhere and both white and grey matter may be affected.

Microscopically the most striking features of these plaques are—a dense network of neuroglial fibres, absence of the myelin sheaths of the nerves, and retention of normal axis-cylinder processes.

The symptoms and signs, of course, vary with the different distribution of the patches of sclerosis, but certain features are especially constant and therefore of particular diagnostic importance, namely :—

(i.) Nystagmus.

(ii.) Intention tremor.

(iii.) Scanning or staccato speech.

(iv.) More or less spastic paraplegia with an extensor plantar response.

(v.) Loss of superficial abdominal reflexes.

An important point is the tendency for remission of the earliest symptoms—for example, a patient may suddenly develop diplopia or amblyopia or a monoplegia, which may completely pass off in a few days.

The following are the more common manifestations as they affect the different systems :—

(i.) *Mental.* The patients (especially the younger ones) are extremely emotional, and often quite happy and optimistic ; they are easily moved to laughter or to tears. At the same time there is usually definite blunting of the higher faculties.

(ii.) *Cranial Nerves.* Amblyopia, hemianopia, and central scotoma are frequent.

Optic atrophy occurs in nearly 50 per cent. of all cases.

The pupils are natural and react to light unless there is optic atrophy.

Strabismus may occur from partial ophthalmoplegia.

Coarse, irregular nystagmus is nearly always present, and is best seen on lateral movement.

Inco-ordination of the articulatory muscles causes the characteristic staccato or syllabic speech.

(iii.) *Sensory System.* Subjective sensations (numbness, tingling, etc.) are often present. Objective sensory changes are inconspicuous.

(iv.) *Motor System.* Spastic paralysis (upper motor neuron) is the rule sooner or later ; it is most evident in the lower limbs, where it assumes a paraplegic form.

Tremor is, perhaps, the most constant feature. It is not evident when the patient is at rest, but at once appears on voluntary movement (intention tremor). The hands and arms show the tremor best, but the head and trunk possess it also to some extent.

(v.) *Reflexes.* The superficial abdominal reflexes are generally lost early. The deep reflexes are never lost ; indeed, they are usually increased, and the plantar reflex becomes extensor as soon as there is any upper motor neuron lesion. The sphincters are not constantly affected unless there is marked paraplegia. Precipitancy or retention may occur, and constipation is apt to be troublesome.

(vi.) *Trophic changes* are rare.

The diagnosis is easy in a well-developed case, but difficulty may arise in early or atypical forms. The most important conditions to be distinguished from disseminated sclerosis are :—

(*a*) *Hysteria.* A transient monoplegia or diplopia is apt to be regarded as functional. The most important points in settling that a case of this sort is not functional are :—

(i.) The presence of nystagmus.

(ii.) The absence of a superficial abdominal reflex.

(iii.) The presence of an extensor plantar response.

(iv.) The presence of optic atrophy.

Any of these phenomena are important evidence against hysteria ; on the other hand, well-marked evidence of hysteria may be present in cases that are not organic (*vide* p. 585).

(*b*) *Traumatic Neurasthenia.* A certain proportion of patients with disseminated sclerosis date the onset of symptoms from an accident, and it may well happen that inability to walk properly after an accident may be attributed to traumatic neurasthenia when in reality the case is one of early disseminated sclerosis.

(*c*) *Compression Paraplegia.* This can be distinguished by the sensory loss and paralysis up to a certain level and the absence of nystagmus or tremor.

(*d*) *Subacute Combined Degeneration.* The age incidence is later. Sensory changes, subjective and objective, are well marked ; there is no nystagmus nor optic atrophy, and usually there is a typical blood picture (*vide* p. 556).

(*e*) *Extra-cerebellar Tumour.* The nystagmus is unequal, being coarse to the side of the lesion ; there is likely to be ataxy on the side of the lesion and spasticity on the other side ; optic neuritis may be present.

(*f*) *Friedreich's Disease* (*vide* p. 563).

II. **Amyotrophic Lateral Sclerosis.** This is a chronic sclerosing degeneration of both upper and lower motor neurons.

In most cases the affection is very much more marked in the lower neurons than in the upper, and quite often clinically there is little or no evidence of upper neuron involvement. This condition is called *Progressive Muscular Atrophy.* When the nuclei of the cranial nerves are involved by extension of the disease up the cord *Bulbar Paralysis* results.

Since the clinical signs vary considerably with the situa-

tion of the lesion, the disease can conveniently be described under three headings :—

(i.) Progressive Muscular Atrophy. This is a disease which affects men more often than women, and rarely starts before 30 years of age.

The first sign is a progressive wasting of the small muscles of the hand. This is usually quite marked before there is any very conspicuous loss of power, but after a time weakness of the affected muscles is a prominent feature.

The muscles of the thenar and hypothenar eminences, the interossei, and the lumbricals are the first to be affected, but after a time the muscles of the forearm and shoulder are involved, and later those of the neck and also of the lower limbs.

One hand may be affected before the other, but when the case comes under observation the condition is usually bilateral.

Fibrillary tremor in the wasted muscles is a very characteristic feature of the disease.

In well-marked cases the wasted hands assume a striking "claw-like" appearance, with the thumbs retracted into the same plane as the fingers, the metacarpo-phalangeal joints extended, and the interphalangeal joint flexed.

The cranial nerves escape unless extension of the disease up the cord causes bulbar paralysis.

The sensory system is unaffected.

The reflexes gradually disappear in the affected muscles ; the superficial reflexes are unaltered.

The sphincters escape.

The diagnosis must be made from :—

(*a*) *Cervical Rib.* Here the lesion is unilateral, the atrophy does not progress, there are sensory changes, and an X-ray photograph may show the rib.

(*b*) *Muscular Dystrophies.* Here there is a familial history, a younger age incidence, different distribution of the wasting, and no fibrillation.

(*c*) *Peroneal Atrophy.* Except that there is fibrillation, the same remarks apply as in muscular dystrophy, and in addition there is sensory change (*vide* also p. 562).

(*d*) *Peripheral Nerve Lesions.* In these pain and other

sensory disturbances are constant if a mixed nerve is affected, and the paralysis is of peripheral type, not segmental.

(ii.) Amyotrophic Lateral Sclerosis. In addition to the features of anterior horn-cell degeneration, as described above, there is a degeneration in the pyramidal tracts which usually develops coincidently with the muscular atrophy.

If, then, we add to the picture of progressive muscular atrophy that of spastic paralysis of the legs and arms with exaggeration of deep reflexes, absence of superficial abdominal reflexes and extensor plantar responses we have the characteristic appearance of amyotrophic lateral sclerosis. There is no sensory change and usually no further sphincter trouble than slightly-delayed micturition and constipation.

Very rarely it happens that the paraplegic phenomena are much in excess of the atrophic ones, and it is probably to such cases that the term " primary lateral sclerosis " was formerly applied.

The diagnosis presents no difficulties. The lack of *sensory changes* excludes syringo-myelia and tumours whether intra- or extra- medullary.

(iii.) Chronic Bulbar Paralysis. This is merely a topical manifestation of progressive muscular atrophy in the nuclei of the cranial nerves ; it is, however, practically always associated with pyramidal tract involvement, and so is usually an accompaniment of amyotrophic lateral sclerosis.

If the upper cranial nerve nuclei are affected chronic ophthalmoplegia is added to the picture of bulbar palsy, or it may exist alone.

The special signs of bulbar paralysis are weakness and wasting of the tongue, the muscles of the palate and pharynx, the trapezius and sternomastoid and sometimes the muscles of the face. In a well-developed case the patient cannot put out his tongue, swallow, close his lips, articulate or even whistle.

The diagnosis must be made from :—

(*a*) *Acute Bulbar Paralysis*, which results from a vascular lesion, by the slow and insidious onset.

(*b*) *Myasthenia Gravis* by the greater atrophy, the absence of remissions in the signs, the absence of the

myasthenic reaction (*vide* p. 567), and the presence of an evident upper motor neuron lesion.

(*c*) *Former Double Hemiplegia* by the lower motor type of lesion with atrophy and reaction of degeneration.

III. **Subacute Combined Degeneration of the Spinal Cord.** This disease consists of a degeneration of the white matter of the spinal cord, which generally starts in the posterior columns, attacks next the lateral tracts, and may in the later stages spread round the entire white matter of the cord. The signs, however, point to especial involvement of the posterior and lateral columns.

The degeneration most often commences in the lumbar region. Associated with the nervous lesions is a progressive anæmia, which is generally of the type seen in Pernicious Anæmia.

Women are affected more often than men, and the age incidence is 40 to 65 years.

The earliest symptoms are the subjective sensory phenomena of numbness, cold and tingling in the extremities; these are usually felt in the feet before the hands, and may last for many months before any other signs appear.

The next symptoms are progressive spastic paralysis in the legs and anæmia. As the spasticity develops there are often cramps and flexor spasms. The sphincters are affected to a *slight* extent about this time, but there is not incontinence at this stage. An absent superficial abdominal reflex and an extensor plantar response are, as usual, likely to be the earliest signs of commencing paraplegia.

By the time that there is definite paraplegia there is usually objective sensory loss up to a definite segmental level, although subjective phenomena of numbness, sense of contraction, etc., may be present also.

The upper level of the lesion can often be traced in its advance up the cord by the increase upwards, segment by segment, of the anæsthesia.

When this stage is reached death is not far off, but before this happens there is a change in the type of the paraplegia, which becomes utterly flaccid, with loss of all reflexes, except possibly the extensor plantar response which may persist till the end, and complete incontinence.

Before the patient is bedridden the picture is one of ataxic paraplegia from the predominance of the "postero-lateral" sclerosis.

The ordinary duration of the disease is not more than two or three years and may be much less.

Differential diagnosis must be made from—

(*a*) *Tabes Dorsalis.* By the absence of eye change or *special* loss of muscle sensibility and the presence of an extensor plantar response.

(*b*) *Disseminated Sclerosis* (*vide* p. 553).

(*c*) *Syphilitic Meningo-myelitis.* By the absence of any eye signs or of lymphocytosis in the cerebro-spinal fluid, by the blood picture, and by the character and distribution of the sensory changes.

IV. **Acute Poliomyelitis.** This is a specific infective disease occurring in epidemics (especially in Northern Europe), but also sporadically, and affecting children and young adults.

The micro-organism is believed to be an ultra-microscopic coccus; the virus has been isolated and used to produce the typical disease in animals.

The lesions characteristic of the disease are acute inflammatory foci round the blood-vessels in the grey matter of the cord anywhere throughout its entire length. The anterior horn-cells are those principally affected, but there is in fatal cases, and possibly in all, a similar change locally in the vessels of the pia-arachnoid. Acute encephalitis is the same disease affecting the cortex of the brain.

The cerebro-spinal fluid, except in the mildest cases, shows a definite lymphocytosis.

The symptoms start abruptly, with the usual constitutional disturbance of specific infections, fever, malaise and pains in the head and limbs; vomiting and convulsions occur in the more severe cases.

In addition to the pains in the limbs the muscles are often tender.

The constitutional disturbance may be so slight as to be overlooked by inattentive parents and, anyhow, subsides in a few days.

Within a short time of the onset of the illness there is a flaccid paralysis of some of the voluntary muscles.

The paralysed muscles may be few or many ; most often the limb muscles are picked out ; the trunk muscles are quite often affected and the muscles supplied by the cranial nerves much more rarely.

The muscles are affected according to their segmental cord supply, thus showing clearly that the lesions are in the anterior horns.

There is considerable wasting of the paralysed muscles, with loss of reflexes and reaction of degeneration.

The initial paralysis is always far greater than the permanent ; this can be explained by the ultimate recovery of many nerve cells which are temporarily put out of action by adjacent inflammatory exudate. There are, however, practically always some permanent muscular paralyses left when the maximum of recovery has been made.

As the child grows up contracture deformities are prone to develop, usually from over-action of the unparalysed muscle groups.

The Sensory System usually escapes, but this is by no means always the case, and the presence of sensory phenomena pointing to a posterior horn lesion should not vitiate the diagnosis provided that the motor signs are those of poliomyelitis.

The sphincters are but rarely affected, though retention of urine may occur at the outset.

Vaso-motor and *trophic changes* show themselves by coldness and lividity of the affected parts, and by a tendency for impaired growth subsequently in severe cases.

If the bulbar muscles are affected an acute bulbar paralysis results, and must be diagnosed by the constitutional disturbance and possibly by the evidence of poliomyelitis elsewhere. As a rule the diagnosis is not difficult if the age of the patient and the presence of the initial constitutional disturbances are remembered.

Landry's Paralysis affects adults, starts gradually, and spreads steadily upwards from the lower limbs ; there is no atrophy and but little constitutional disturbance.

In *Acute Toxic Polyneuritis* adults are affected, there is usually no constitutional disturbance, but marked sensory

symptoms of *peripheral* type occur. A nuclear arrangement of the paralysed muscles is not present.

V. **Landry's Paralysis.** This is thought to be due to the action of some toxin on the anterior horn-cells of the spinal cord. It occurs between the ages of 20 and 40 years and affects men more than women.

The onset is attended by slight constitutional disturbance. In a few hours there is a sense of heaviness in one or both legs, which soon develops into a flaccid paralysis. The paralysis may affect the proximal segments of the limb before the distal, and in very rare cases the arms are attacked before the legs.

The motor paralysis is complete for all muscles, and spreads steadily up the trunk until the respiratory muscles are affected and even the muscles supplied by the cranial nerves.

Death usually takes place in from two to five days, but sometimes the upward course of the disease stops suddenly, and in these cases recovery may be complete.

The deep reflexes are, of course, absent over the paralysed area. The superficial reflexes may be preserved.

The sphincters are not affected.

There is no sensory loss.

The differential diagnosis must be made from—

(*a*) Acute toxic polyneuritis (*vide* p. 510).

(*b*) Acute poliomyelitis (*vide* p. 558).

CHAPTER IX

FAMILIAL DISEASES

In this chapter will be included those diseases of the nervous system which are congenital and those which have a familial incidence.

I. **The Muscular Dystrophies.** The name "Dystrophy" is given to a group of cases in which there is a congenital muscular defect whereby certain muscles are unable to develop in proportion with the rest of the body or in which certain muscles or parts of certain muscles are absent at birth. There is nearly always evidence of a familial distribution and the disease is transmitted by an unaffected mother. Clinically the following varieties of muscular dystrophy may be recognised :—

(i.) *Pseudo-hypertrophic Muscular Paralysis.* This affects males more than females and usually is first noticed at the age of four to five years; an onset later than twelve years is rare.

Difficulty and unsteadiness in walking are the first signs; the child falls readily and gets up with difficulty. It may be noticed that his gait becomes waddling owing to the wide separation of the feet. There is marked lumbar lordosis, and the shoulders are thrown back in order to relieve the quadriceps femoris as much as possible.

The mode of rising from the floor by climbing up himself is very characteristic; the child rolls over on to his hands and knees, he extends his knees so that his toes and hands only are on the floor, and then places one hand above its corresponding knee and gradually works it up the thigh as the sole of that foot becomes flat on the ground. A similar manœuvre is now gone through with the other hand, until with a jerk the shoulders are thrown back and he is standing up.

Certain muscles appear to be hypertrophied in the earlier

stages, though the hypertrophy is unreal and is made up of fat and fibrous tissue. These muscles are: the calf muscles, the extensors of the knee, the glutæi, the lumbar muscles, the supra- and infra- spinati, the deltoid, the triceps and biceps.

Certain muscles waste without any preliminary pseudo-hypertrophy or may be congenitally absent. These are: the latissimus dorsi, the teres major, the clavicular half of the sterno-mastoid, and the lower part of the pectoralis major.

The child is unable to depress his arms against resistance and cannot be lifted up by hands placed in his axillæ, without tending to slip.

The reflexes diminish as the muscle fibres waste: the electrical responses in the affected muscles do likewise.

The sphincters are not involved, and there is no sensory change.

There is no fibrillary tremor in the affected muscles.

In some cases the muscles affected are the same as described above, but the disease is atrophic from the start.

(ii.) *Erb's Jurenile Type of Muscular Dystrophy.* In this variety the onset occurs between the ages of 12 and 20 years; the condition is usually atrophic from the first, but may show pseudo-hypertrophy sometimes.

The first changes are seen in the muscles of the shoulder-girdle, namely, the lower part of pectoralis major, the trapezius, the serratus magnus, the latissimus dorsi, and the rhomboids. The following arm muscles are affected also: the biceps, the triceps, and the supinator longus. The spinati and the deltoid are generally unaffected. Later the muscles of the pelvic girdle and the back and trunk become involved.

(iii.) *The Facio-scapulo-humeral Type of Landouzy-Dejerine.* This variety affects the sexes equally and starts at or soon after puberty. The first muscles to be affected are the face muscles, especially the risorii, orbiculares, levatores menti, and zygomatici. The naso-labial folds are obliterated, the lips turn out and cannot be "pursed" as in whistling, and the articulation of labials is impossible.

Later the muscles of the shoulder girdle and upper arm become affected also.

Other rarer and less important varieties are described, such as a *Distal type*, in which the small muscles of the extremities are first affected, and the *Pelvic type*, in which weakness of the psoas muscles causes difficulty in walking upstairs before any other symptoms are apparent.

It may be stated that, in whatever form the disease starts, there is a steadily progressive tendency, and more and more muscle groups are likely to be involved before death takes place. This is usually due to some intercurrent pulmonary affection, probably from ten to twenty years after the first symptom.

The diagnosis must be made from—

(*a*) *Peripheral Nerve Lesions*, by the absence of sensory symptoms, the age of onset, and the distribution of the atrophy.

(*b*) *Progressive Muscular Atrophy*, by the familial history, the earlier age, the different distribution, the gait, and the absence of fibrillation or reaction of degeneration.

(*c*) *Myasthenia Gravis* (*vide* p. 567).

II. **Peroneal Muscular Atrophy**. This disease is not a muscular dystrophy ; it is included in this chapter because of its familial incidence. Pathologically the principal changes are those of degeneration of the mixed nerves and also of the anterior horn-cells, but there is degeneration to a lesser extent in the posterior roots and the posterior columns.

The course is very chronic ; symptoms are usually first noticed between the ages of 15 and 35 years, and males are more often affected than females.

The first signs are wasting of the peroneal muscles, the small muscles of the feet, and to some extent the long extensors of the toes. Later the calf muscles may become involved.

The appearance of the leg is characteristic : the leg is wasted below the knee and the outer border is notably flat, the thigh muscles are large and give to the leg rather the appearance of an Indian club.

The toes are pointed and the foot turned in ; often there may be talipes equinus or equino-varus.

Fibrillary twitching and some reaction of degeneration can generally be demonstrated.

The nerves are not tender, but occasionally pain is complained of, and some blunting of sensation can practically always be found on the peroneal aspect of the leg.

The sphincters escape.

A late manifestation of the disease may be the appearance of similar atrophy in the muscles of the hands and forearms.

The age of onset, the family history, and the very characteristic distribution should prevent any error in diagnosis.

III. **Friedreich's Disease.** This disease is usually familial, but apparently sporadic cases may occur. It is not as a rule transmitted directly by an affected parent. Males are attacked more often than females.

The first signs generally appear from 7 to 17 years of age. The essential pathology is a progressive sclerosis in the spino-cerebellar tracts and in the posterior and lateral columns of the cord.

The characteristic features of Friedreich's Disease are :—

(*a*) Cerebellar type of ataxy, reeling gait, and incoordination of voluntary movements.

(*b*) Tremor of hands, head, and trunk (mostly volitional).

(*c*) Nystagmus, but no alteration of light reflex except in the rare cases in which optic atrophy develops.

(*d*) Deliberate, scanning, explosive speech.

(*e*) Scoliosis.

(*f*) Pes cavus, with conspicuous over-extension of the great toe.

(*g*) Absent knee jerks and usually absent superficial abdominal reflexes.

(*h*) Extensor plantar response.

As a rule there is no sphincter trouble or sensory disturbance, but sometimes cramp-like pains are described. The mental condition frequently shows a slight dulness.

In a typical case the clinical picture is sufficiently obvious, but it must be remembered that the disease is extremely chronic and that only one or two signs may be present for considerable periods. Thus pes cavus may be the first sign in one case and clumsiness in walking in another or nystagmus in a third.

The combination of nystagmus, scoliosis, pes cavus, and

an extensor plantar response with absent knee jerks is more than suggestive.

It happens occasionally that Friedreich's disease may first manifest itself by ataxic paraplegia, increased deep reflexes, nystagmus, scanning speech, and even sphincter trouble. Except for the probable younger age of the patient such cases cannot be distinguished from disseminated sclerosis. In time, however, the development of scoliosis and pes cavus and the loss of the deep reflexes will enable a correct diagnosis to be made. In such cases a careful investigation into the family history might suggest the true state of affairs.

IV. **Cerebellar Ataxia.** Under this title may be grouped a variety of rare conditions some of which are hereditary or familial.

The outstanding clinical features are :—

(*a*) A cerebellar ataxy, as shown by the reeling gait.

(*b*) Inco-ordination or tremor of the arms.

(*c*) Scanning or explosive speech.

(*d*) Usually nystagmus.

V. **Familial Spastic Paraplegia.** This hereditary or familial disease is due to progressive degeneration in the lateral tracts of the cord. It usually develops between the ages of 7 and 10 years. The symptoms are those of pure spastic paraplegia, but sphincter disturbance is slight. Occasionally the arms are involved later. The family history, the later onset, the progressive course, and the absence of mental impairment distinguish this condition from the paraplegic type of cerebral diplegia.

VI. **Huntingdon's Chorea.** This rare disease is strongly hereditary. It develops between the ages of 30 and 40. The symptoms are choreiform movements combined with a steadily progressive mental impairment, leading eventually to complete dementia. A considerable number of patients who suffer from this disease commit suicide.

VII. **Amaurotic Family Idiocy.** This disease is often familial and appears only to occur in the Jewish race. The child is born healthy, but when a few months old develops optic atrophy and a spastic paralysis of the limbs, especially noticeable in the hands. The optic discs show a cherry red spot at the macula. Death occurs within two years.

VIII. **Myotonia Congenita** (Thomsen's Disease). This familial disease develops in childhood and shows itself by a delay in the relaxation of voluntary muscles after contraction or in making an initial movement after a period of rest. The muscles are well developed and show an increased excitability to stimulation of all sorts. After a certain number of movements have been performed the stiffness and rigidity wears off for the rest of the time that the exercise is continued.

IX. **Amyotonia Congenita.**—This rare condition shows itself soon after birth by a definite lack of muscular tone. All sorts of abnormal attitudes are assumed with ease. Sucking may be impossible, and in later years hypotonus of the back muscles may make the sitting posture difficult.

X. **Myotonia Atrophica**. This rare condition does not develop till after puberty; there is difficulty in relaxing the muscles of the extremities, face, and neck after contraction. The muscles are wasted, and this fact, with the later age of onset, serves to exclude Thomsen's disease.

CHAPTER X

DISORDERS OF MUSCULAR FUNCTION

I. **Myasthenia Gravis.** This disease is characterised by the development of easy fatigue in certain muscle groups. It may commence at any age after childhood, is not familial, and seems to affect the sexes equally.

The pathology is obscure; collections of small round cells have been described in the affected muscles and also in the liver, kidneys, and around the nerve cells of the bulbar nuclei. The thymus is persistent.

Course. The condition is subacute or chronic; mild cases may last many years: on the other hand, when many muscles are affected death usually occurs within two or three years.

Symptoms. The earliest and most constant symptom is a weakness of the facial and external ocular muscles, which is induced or markedly increased by exertion and is worse at the end of the day. The muscles of the palate and of articulation may be affected in the same way, so that slurring speech and difficulty in swallowing may be present. The tongue is soft and flabby and shows three longitudinal furrows, one central and two lateral.

Any special occupation may determine the onset of myasthenic symptoms elsewhere than in the muscles supplied by the cranial nerves, but these will become affected before long.

The early symptoms are often conspicuously remittent and may for this reason lead to a diagnosis of hysteria. In more severe cases the affection spreads to the muscles of the neck and shoulders, while in really advanced cases the muscles of the back, trunk and extremities are affected also.

The sensory system is not affected.

The reflexes show no characteristic change, but the deep

reflexes may temporarily fail to respond after repeated elicitation.

The vaso-motor system seems to be excitable, since flushing and sweating are common.

The mental state is not profoundly altered, but myasthenic patients are usually somewhat emotional.

Special Diagnostic Features :—

(*a*) *The Character and Distribution of the Symptoms.* The weakness of certain groups of muscles without notable wasting, and the fact that after rest these muscles are capable of considerable effort whilst use soon tires them out.

(*b*) *The Facies.* There is usually some ptosis ; the forehead is smooth and the head tilted back : the expression appears to be sneering ; the lips are slightly parted, the upper one is retracted and the lower one slightly everted, while the corners of the mouth droop.

The patient cannot wrinkle his forehead, shut his eyes tightly, or "purse" his lips.

(*c*) *Electrical Reactions.* A faradic current excites a brisk contraction, but this soon fades and disappears if the current is continued. After a definite period of rest the muscles can again respond to renewed excitation in the same manner.

This is known as the myasthenic reaction.

(*d*) *Weakness of the Shoulder Muscles* makes it impossible to keep the arms raised from the side for any appreciable time, and after this has been essayed once or twice the arms cannot be raised at all without a long rest.

Myasthenia gravis must be distinguished from—

(i.) *Facio-Scapulo-Humeral* type of muscular dystrophy, by the absence of atrophy or of partial reaction of degeneration and by the history of remissions and the presence of the myasthenic electrical reaction.

(ii.) *Hysteria*, by the absence of any hysterical stigmata, by the onset of the symptoms at night or after exercise, and by the myasthenic reaction.

(iii.) *Bulbar Palsy*, by the absence of atrophy and by the history of remissions.

II. **Paramyoclonus Multiplex.** This rare disease is

characterised by the occurrence of frequent "shock-like" contractions in single muscles or portions of muscles or, very occasionally, in groups of muscles.

The muscles of the upper arms or thighs are most often affected and the condition is bilateral. The contractions cease in sleep. There is no associated sensory or mental change.

The pathology is unknown.

CHAPTER XI

DISORDERS OF MOTION

I. **Paralysis Agitans** (Parkinson's Disease). The pathology of this disease is obscure. The onset between the ages of 40 and 70 years suggests the possibility of there being some underlying degenerative factor, possibly in the muscles.

The characteristic clinical features are :—

(i.) Muscular rigidity.

(ii.) Coarse rhythmical tremor.

In a given case either of these features may be present to the exclusion of the other ; usually they are both well marked.

(i.) *Muscular Rigidity.* This is usually the first sign ; it is widespread, but does not amount to spasticity, and an extensor plantar response is never present. With the rigidity is associated a slight muscular weakness. As a direct consequence of the muscular rigidity and weakness the following signs develop : —

(*a*) Mask-like Expression (Parkinson's Mask). Immobility of the facial muscles and replacement of ordinary emotional feature-play by a tense, anxious expression.

(*b*) Monotonous deliberate articulation.

(*c*) Attitude. This is very characteristic. The head is bent forwards, the back is held stiff and curved forwards, the elbows carried close to each other in front of the abdomen, with the fingers bent at the metacarpo-phalangeal joints, but extended at the interphalangeal joints. The knees are slightly flexed and the thighs tend to be adducted.

(*d*) The gait is slow and hesitating, but when movement has started it tends to become more rapid owing to displacement of the centre of gravity and an apparent attempt to catch this up again (festination).

(ii.) *Tremor.* This is coarse and rhythmical and at first best seen in the thumb and forefinger, which perform the typical "bread-crumbling" movements. Analogous movements may be performed at the wrist and ankle joints. Head nodding is often observed. Movements of the jaws are less common. In the lower limb the tremors tend to be proximal rather than distal at the outset. The tremors continue when the patient is at rest, but cease with voluntary movement and can, in early cases, be controlled by an effort of will.

The tremor usually starts in one hand and affects the leg of the same side before spreading to the opposite hand.

The reflexes are brisk but well within normal limits; there is no sphincter loss.

There is no objective sensory loss, but subjective sensations of temperature change and vague aching are not uncommon.

In well-developed cases the clinical picture of paralysis agitans does not commonly leave room for mistaken diagnosis; the following conditions should, however, be excluded:—

(1) *Bilateral Cortical Degeneration.* In this the progressive bilateral rigidity and fixed expression closely resemble Parkinson's disease; the progressive mental deterioration and eventually the development of an extensor plantar response should prevent mistake.

(2) *Senile Tremor* occurs at an even later age; it involves especially the head, is bilateral from the outset, and is not associated with rigidity.

(3) *Double Hemiplegia* gives clear evidence of organic disease, as shown by the alteration in the reflexes.

II. **Chorea.** Chorea is a disease of childhood and adolescence. That it is a manifestation of rheumatism seems no longer open to doubt. More than 30 per cent of all cases have endocarditis, while more than 80 per cent. give a history of ordinary rheumatism or show other signs of rheumatic infection. Girls are affected more frequently than boys.

An interesting point is the tendency for chorea and epilepsy to predispose to one another.

Symptoms. The earliest symptom is an alteration in the mental condition of the child; she becomes irritable, nervous, emotional, and unable to fix the attention. The

next symptom is usually clumsiness, with a tendency to drop things and to fidget excessively ; this is due to muscular inco-ordination.

It is not unnatural that these early symptoms should often be ascribed to naughtiness and the child punished unjustly.

The characteristic choreiform movements usually appear first in the hands and arms, next in the face, and lastly in the legs and trunk. Sometimes the movements are unilateral (hemi-chorea) throughout. The actual movements are produced by sudden, jerky, irregular contractions of *groups* of muscles ; they are not limited to one group, but seem haphazard. The contractions are not clonic. The movements are more marked when the child is excited or when she is being observed ; they usually subside during sleep. Occasionally the movements are so continuous and wild that a padded bed is necessary to prevent injury.

There is no atrophy of the muscles, but there is definite motor weakness, which may be so extreme as to simulate paralysis.

The muscular inco-ordination often produces an alteration in the speech which becomes hurried, indistinct, and interrupted.

The Mental Condition is not often more altered than indicated above, but in very severe cases a condition of maniacal excitement may supervene.

The expression is characteristically bright, eager, and full of nervous tension.

The Sensory System is unaffected. Pains in the limbs, if present, are due to rheumatism.

The Reflexes are brisk ; the knee jerks are well sustained in many cases. There is no sphincter trouble unless mania develops.

The diagnosis of chorea presents no difficulty as a rule ; very mild cases may be overlooked unless a careful history is obtained, and it is important to remember that the paralytic form in which there is marked muscular weakness may show but little of the typical movements.

Habit spasms may be mistaken for chorea, but a habit spasm is the constant repetition of the same "purposive"

co-ordinated movement, whilst choreic movements are not really purposive, and the same movement is only reproduced by accident amongst many others. At the same time children with chorea are rather liable to have some form of "tic" or habit spasm as well.

In *Paramyoclonus Multiplex* the individual contractions are much more violent; they are more likely to affect a single muscle than a muscle group, and tend specially to pick out certain muscles. There is no mental change.

Chorea occurring in pregnancy shows the same features as ordinary chorea, and is equally a manifestation of rheumatism.

III. **Tetany** (Carpo-pedal Spasm). Tetany is shown by the occurrence of bilateral tonic spasms in the muscles of the extremities, and sometimes of the face and trunk.

It is a disease of childhood as a rule, but is also met with sometimes during lactation.

It is most often associated with chronic dilatation of the stomach or intestine, but it may follow removal of the thyroid gland. In the latter case the parathyroids are supposed to be at fault, as excision of the parathyroids in certain animals may induce tetany.

The actual spasms are preceded by numbness and tingling in the hands and feet.

During the tonic spasms the fingers are flexed at the metacarpo-phalangeal joints and extended at the inter-phalangeal joints; the thumb is adducted and its tip meets the finger tips. The wrist is flexed, the forearm pronated and adducted, and the elbows flexed. The feet are arched and inverted; the legs are extended and adducted.

The spasms start suddenly and relax gradually after some minutes or possibly not for many hours.

The following signs may be mentioned :—

(i.) *Trousseau's Sign.* Compression of the blood-vessels to a limb may induce an attack.

(ii.) *Chvostek's Sign.* Tapping on the facial nerve may induce a spasm of the facial muscles.

The typical carpo-pedal spasm is not simulated by any other condition.

In tetanus there is a history of some lesion and the earliest

signs are in the neck and jaw muscles. Trismus never occurs in tetany.

IV. **Occupation Neuroses.** The oft-repeated performance of the same actions, especially actions involving the small muscles of the hand, sometimes induces cramp-like contractions in these muscles, which may be so severe as to prevent the further performance of the specific acts.

These conditions are not to be regarded as due to any organic lesion and are always associated with general symptoms of neurasthenia. Tremor of the hand is a constant accompaniment.

They must not be confounded with craft palsies (*vide infra*).

The following examples of occupation neurosis may be mentioned :—

(i.) *Writer's cramp.* This affects the flexors of the thumb and first fingers, but may involve the wrist and forearm. Much writing may induce a temporary "fatigue cramp" in anybody, but this passes off with a little rest, whereas in true writer's cramp the spasms start again as soon as writing is once more attempted, even after prolonged rest.

(ii.) *Tailors and sempstresses* may have very similar affections.

(iii.) *Pianists, telegraphists, and stenographers* are liable to cramps in the extensors of the wrists, *violinists* in the thumb and finger muscles of the left hand.

(iv.) *Hammermen* may suffer from analogous cramps in the deltoid and triceps muscles.

Craft Palsies are due to a true pressure neuritis, the result of long-continued over-exertion of certain muscles or muscle groups, especially in the case of the small muscles of the hands.

Muscular atrophy as well as the subjective sensory changes of true neuritis are present.

Platers, locksmiths, and cigar or cigarette rollers are perhaps most frequently affected.

CHAPTER XII

CERTAIN FUNCTIONAL DISEASES

A. Epilepsy.

Epilepsy is a chronic progressive disease characterised by recurrent seizures, always associated with impairment of consciousness and very often with convulsions. Progressive mental deterioration is common.

There is a definite hereditary factor in epilepsy, about half the cases giving a family history of epilepsy, insanity, or alcoholism. Males are more often affected than females. The majority of cases seem to start either in the first eight or nine years of life or between the ages of 14 and 21 years, but no age is immune.

The pathology of epilepsy is not understood.

For convenience in description Jacksonian or focal epilepsy is included under the heading "Epilepsy," but it must clearly be understood that Jacksonian epilepsy is produced by some definite and organic intra-cranial lesion, whereas in idiopathic epilepsy no such condition can be demonstrated.

Clinically Epilepsy may be considered under five headings :

(i.) Jacksonian or Focal Epilepsy.
(ii.) Major Epilepsy (*Grand Mal*).
(iii.) Minor Epilepsy (*Petit Mal*).
(iv.) Status Epilepticus.
(v.) Psychical Epileptic Phenomena.

I. **Jacksonian Epilepsy**. Any organic lesion which involves the cerebral cortex either directly or indirectly is likely to be accompanied by convulsive attacks which correspond in their nature and distribution to the affected part of the brain. The more strictly cortical is the lesion, the more localised are the convulsions.

The following are the characteristic features of Jacksonian epilepsy :—

(*a*) The attacks always start in the same manner and in the same part of the body.

(*b*) If the attacks become generalised, the order of progression is constant.

(*c*) There is no loss of consciousness unless the attacks become generalised.

If the lesion is in the motor area there are clonic contractions of the corresponding muscle groups on the opposite side of the body. In severe cases a tonic stage may be apparent. The convulsions may be limited to a certain area (*e.g.*, arm and face), or they may spread till a considerably wider region or even the whole body becomes involved.

If the lesion is in the sensory area the first signs of an attack are paræsthetic sensations in a particular locality; these sensory phenomena are generally, but not always, followed by convulsions in the corresponding motor area.

If the lesion is in the psychical area the earliest and often the only sign is a transient loss of consciousness strictly comparable to *petit mal*.

II. **Major Epilepsy.** The majority of attacks are preceded by a sensory or psychical warning which is called an "aura."

The following are the most common varieties of aura:—

(*a*) Abnormal sensation in the epigastrium or thorax, such as cramps, spasms or feelings as of suffocation.

(*b*) Curious sensation in the head.

(*c*) Subjective sensations of the special senses, visual, olfactory, or gustatory.

(*d*) Psychical phenomena, such as sensations of dread or of fear, or dreamy conditions.

(*e*) Sensory phenomena of all sorts affecting any part of the limbs, trunk, or head.

After the aura has developed, if one be present, the patient utters a cry, loses consciousness, and falls to the ground in a state of general tonic muscular spasm. He does not hear the cry he utters. His arms are abducted and flexed at the elbows; his legs are extended and inverted. His head and eyes show conjugate deviation; the pupils are fixed and dilated. He becomes increasingly cyanosed, since respiration has ceased. After a time, varying from a few seconds to a minute and a half, *the tonic stage* gives

way to *the clonic stage* and respiration is re-established. This stage lasts perhaps for three or four minutes, and as it wears off the patient passes into a condition of stupor which generally merges into deep sleep. The deep reflexes are usually increased for a short time after a fit. The patient may suffer considerable injury from his fall and he may bite his tongue or cheeks during the fit ; or he may suffocate himself by falling face downwards on a pillow or into a pool of water. He not infrequently passes both water and fæces while unconscious.

The fits may occur many times daily, or they may be separated from each other by many months ; sometimes the fits only happen at night (nocturnal epilepsy). In this form the patient may die of suffocation, owing to the fact that the fit leaves him in such a position that he cannot breathe.

III. **Minor Epilepsy.** Two forms of minor epilepsy can be recognised—first, where a characteristic "aura" is experienced but where no further manifestation in the direction of convulsions or unconsciousness occurs, and, secondly, where there is no aura but where there are recurrent attacks of what is described as "fainting," "loss of memory," "falling over" with or without a cry, etc., etc.

All these phenomena are very brief, but it is of especial importance that their true significance should be recognised, as they respond not infrequently to treatment.

The post-epileptic phenomenon of "automatism" is particularly likely to follow this latter variety of minor epilepsy.

IV. **Status Epilepticus.** This may supervene in any variety of epilepsy, and sometimes it develops if bromides are left off too suddenly. The fits follow each other practically without cessation and with little or no interval of consciousness.

If the fits do not stop the temperature rises and death takes place in about forty-eight hours as a rule.

V. **Psychical Epileptic Phenomena.** It sometimes happens that epileptics are men of undoubted genius ; more often there is obvious mental impairment with defective judgment, imperfect memory, and religious enthusiasm, which does not coincide with their daily life.

Many patients show progressive dementia and eventually become hopelessly imbecile.

Psychical phenomena occurring before a paroxysm should be regarded as part of an "aura" which is unduly prolonged. Psychical phenomena occurring after a paroxysm may take the following forms :—

(i.) *Acute Dementia.* This sometimes develops when there have been an unusual number of fits in a short time ; it is a temporary condition.

(ii.) *Acute Mania.* This is rare and generally lasts for ten or twelve hours.

(iii.) *Delusions.* A fit or series of fits may sometimes be followed by a definitely delusional state which may last several days.

(iv.) *Automatism.* This is most noticeable after minor attacks ; any form of automatic action may be performed. The duration of the automatic stage is doubtful, and many crimes have been attributed to post-epileptic automatism which were probably not due to this at all.

Psychical Epileptic Equivalents. It is possible that psychic phenomena may replace the more usual motor signs in certain cases of epilepsy, though how far such equivalents differ from automatism and other psychic manifestations of minor epilepsy seems doubtful.

The diagnosis of Epilepsy is not always easy. It is important to remember that typical attacks of *grand mal* may be produced by gross intra-cranial disease, and such diseases as cerebral tumour must always be excluded before diagnosing epilepsy (*vide* p. 523). In like manner uræmic fits should be eliminated.

Major Epilepsy, especially if the attacks are not seen by the physician, may present some resemblance to hysteria. The following table based on that drawn up by Sir William Gowers presents the chief differential points :—

	Epilepsy.	*Hysteria.*
Apparent cause.	None.	Emotion.
Warning.	Especially one of the auræ described above.	Palpitation, malaise, choking, drumming on the floor with the feet.
Onset.	Always sudden.	Often gradual.

	Epilepsy.	*Hysteria.*
Scream.	At onset.	During course.
Convulsions.	Tonic, then clonic.	Rigidity, struggling, arching of back.
Micturition.	Frequent.	Never.
Duration.	Three to four minutes.	Ten minutes or longer.
Restraint necessary.	To prevent patient being hurt.	To prevent patient hurting other people.
Termination.	Spontaneous.	Can be induced (as by slapping the face, pulling the hair, drenching with cold water, or applying a strong faradic current).

Nocturnal epilepsy may escape recognition for a long time, but suspicion should be aroused if a patient complains of waking with a sense of muscular fatigue, a sore tongue, a wet bed, or blood on the pillow.

Minor epilepsy is often overlooked in childhood ; it should be remembered that it is probably the most frequent cause of repeated so-called "fainting" attacks in young children, though similar phenomena may be produced by intestinal parasites, especially round worms. Cardiac lesions are an extremely rare cause of fainting in childhood.

A patient with sufficient cardiac damage to account for syncope may also be a minor epileptic ; the epileptic attacks, however, are attended by complete and sudden loss of consciousness and are followed by some automatism. The pulse is not affected, whereas in true syncopal attacks the loss of consciousness is gradual, there is marked pallor, and the pulse becomes distinctly feeble.

In conclusion we would emphasise the fact that it is scarcely justifiable to diagnose epilepsy without there being at some time (not necessarily with every seizure) some loss or impairment of consciousness. Apart from this perhaps the most conclusive signs are a fall, tonic spasms followed by clonic, involuntary micturition, and biting of the tongue.

B. Migraine (Periodic Headache).

It seems unnecessary to distinguish between cases presenting all the classical phenomena of migraine and those in

which a periodic headache, with no discoverable cause, is the only feature.

Periodic headaches are more frequent in women than in men; they usually commence in childhood and tend to diminish or cease after the climacteric. There is a strong hereditary influence in most cases.

The pathology of these headaches is not understood, but there seems to be a relationship between migraine and epilepsy; the children of epileptics may have migraine, and attacks of migraine may be replaced by epileptic fits.

Symptoms. The most constant feature is headache; this is often unilateral, but varies in position with different cases; the frontal region is most often affected. The pain may be either boring, piercing, or throbbing in character.

The most typical cases have a definite "aura," which precedes the headache by a short time.

The usual aura is visual in nature and may consist of flashes of light, sparks of light, zig-zag streaks of light (teichopsia), a central bright spot or a central dark spot (scotoma) which extends to the periphery of the visual field, but as it spreads clears up in the centre. Homonymous hemianopia is another common visual aura.

Less common warnings are feelings of numbness or tingling referred to the arms, face, tongue and lips, and occasionally paraphasia (use of the wrong word or inability to find the word wanted).

The average duration of the warning symptoms is twenty minutes and as they pass away the headache develops. The pain lasts for twelve to twenty-four hours, and in most cases terminates by vomiting or nausea; in other cases the vomiting may be present all through the period of headache.

It must be understood that the course of events differs greatly in different cases, but tends to follow the same general sequence for the same patient.

The individual attacks may be precipitated by certain factors which vary in different cases; the tendency for the menstrual period to be accompanied by this type of headache is well known.

Other common exciting causes are digestive disturbances, overwork, over-worry, over-fatigue and over-excitement.

Persons who are "run-down" or "anæmic" are more liable to migrainous attacks, as are those with naso-pharyngeal obstruction or refractive errors, especially the lesser degrees of astigmatism.

The diagnosis of migraine rests upon the character of the attacks and the exclusion of gross organic disease (cerebral tumour or nephritis) and of any remediable reflex cause (refractive error or adenoids). The diagnosis is strengthened if the attacks started in childhood or adolescence and if the patient is a female.

C. The Tics, or Habit Spasms.

A habit spasm is the involuntary and automatic performance of a co-ordinated purposive act. In addition to these, psychical tics are met with in which the subject is compelled to perform certain acts in a special manner or to precede certain actions by the utterance of certain words or numbers.

Ordinary habit spasms often start in adolescence, and not infrequently develop from repeated voluntary performance of certain acts; for example, a boy who kept rabbits imitated the wriggling movements of their noses and upper lips until he acquired this action as a tic.

The following common forms of Motor Tic may be mentioned:

I. **Facial Tics.** Blinking, winking, smacking the lips, "pulling faces," smiling, etc.

II. **Neck Tics.** Tossing and nodding the head and spasmodic torticollis, which is one of the most common forms of tic. The essential features of torticollis are:

(*a*) The movement is one which can be performed voluntarily.

(*b*) The movement can be controlled in the early stages.

(*c*) There is, in the fully-developed condition, an uncontrollable impulse for the performance of the movement.

Many patients with torticollis habitually control the spasmodic movements by carrying one or two fingers in apposition to their chins; as soon as the hand is removed the

spasms take place, and yet the hand is in no way used for *holding* the head steady.

III. **Arm Tics.** Shrugging the shoulders and abducting or adducting the arms.

IV. **Leg Tics.** Many unusual gaits are of the nature of a tic.

V. **Speech Tics.** The uncontrollable emission of words or phrases which have no logical bearing on the environment.

VI. **Convulsive Tics.** This rare condition is characterised by three phenomena :—

(*a*) Spasmodic jerking movement.

(*b*) Emission of irrelevant words or phrases (frequently obscene or blasphemous).

(*c*) Obsessive ideas which lead to all sorts of foolish or dangerous acts.

Diagnosis. The most important diagnostic features of the tics are :—

(i.) The fact that the movements can be controlled voluntarily to some extent and that the exercise of such control causes definite mental distress.

(ii.) The movements are notably lessened when the patient's attention and interest are fully occupied.

(iii.) Psychical instability, or at least a history of such in the family—for example, insanity, alcoholism, epilepsy, chorea.

(iv.) The movements cease during sleep.

It is quite possible to mistake some forms of habit spasm for chorea (*vide* p. 571) if the first two of the above characteristics are not borne in mind.

In paramyoclonus multiplex the movements are mostly of certain of the leg muscles ; they are bilateral, they are not purposive acts, and they are absolutely uncontrollable by will.

CHAPTER XIII

VASO-MOTOR AND TROPHIC DISEASES

1. **Raynaud's Disease.** This is more common in women than men ; it may be induced by cold or by shock, and appears to depend upon a vaso-motor disturbance which produces a spasm in the arteries of the extremities.

The disease is nearly always bilateral and in addition to the hands and feet the nose and ears may be affected.

Clinically three stages may be recognised :—

(i.) *Local Syncope.* A numbness and feeling of deadness in the fingers, accompanied by pallor and anæmia and obvious coldness. After a period of minutes or hours the arteries relax and the ordinary appearances and sensations are restored.

(ii.) *Local Asphyxia.* This may follow the local syncope or may be a primary stage. A temporary blanching is followed by extreme congestion with marked discoloration of the parts, which become purple and show distended veins. The hands and feet are usually affected. The similarity to chilblains is striking.

After a matter of hours or days these phenomena tend to subside or may proceed to the third stage.

(iii.) *Local Gangrene.* If recovery does not follow the asphyxial or even the syncopal stages localised dry gangrene ensues. The gangrenous patches may be very small or may involve whole fingers, though as a rule the bone escapes.

An essential feature of Raynaud's disease is the paroxysmal nature of the manifestations, which serves to exclude such conditions as senile gangrene or congenital heart disease.

Some cases of Raynaud's disease show the very interesting complication of paroxysmal hæmoglobinuria ; in others recurrent paralyses, presumably due to temporary ischæmia in the cerebral vessels, have been described.

II. **Intermittent Claudication** (Limping). This rare con-

dition usually develops during muscular exercise and is dependent upon the temporary withdrawal of blood from certain leg muscles owing to spasm of the arteries.

During the attack no pulse can be felt in the dorsalis pedis or posterior tibial arteries and the limb becomes cold.

The condition is usually unilateral and tends to be progressive. During the attacks there are cramps in the muscles, and the leg feels as if it were going to burst; it may be swollen and discoloured.

III. **Erythromelalgia.** A rare chronic disease affecting women more than men, and mostly seen in the Jewish race. It is characterised by severe local pain with redness, swelling, and increased temperature, usually in one or other foot.

The pathology is thought to be a combination of vaso-motor derangement with diseased blood-vessels, and possibly peripheral nerve degeneration also.

IV. **Angio-neurotic Œdema.** This condition results from vaso-motor disturbance and manifests itself by the occurrence of local œdematous patches, which are definitely circumscribed but which may involve the mucous membranes equally with the skin.

The appearance of the patches may be strikingly periodic, and each patch is often very brawny in consistence.

If the larynx is involved, tracheotomy may be necessary. If the stomach or intestines are affected, disturbance of their functions may be a feature of the case.

V. **Facial Hemiatrophy.** This rare disease nearly always starts in childhood and shows itself by a progressive wasting of all the tissues of one half of the face.

The cause is believed to be due to an interstitial neuritis of the fifth nerve, but as there is no sensory disturbance it is hard to believe that this is the only factor.

VI. **Scleroderma.** This chronic condition is characterised by a local or diffuse induration of the skin. In the affected areas the secretion of sweat is abolished, so that the skin appears smooth, dry, and glossy.

Occasionally Raynaud's disease and scleroderma are associated.

When the fingers are especially affected (sclerodactylie) they may become notably pointed, deformed, atrophied, and shortened ; the nails may completely disappear.

Scleroderma is regarded as a tropho-neurosis, a view which, however, does little to explain satisfactorily its pathology.

CHAPTER XIV

HYSTERIA AND NEURASTHENIA

HYSTERIA is a psychical disorder whereby, according to Janet, there is a dissociation of certain mental processes from the main consciousness. It is most common in the female sex and is most frequently seen in adolescence; it may be started by trauma, shock, or prolonged strain or ill-health. Hysterical persons are unduly susceptible to impressions from without, whether psychical or physical.

The manifestations of hysteria are extremely diverse; the following are some of the more important:—

I. **Hysterical Seizures.** (*a*) *Minor Attacks.* These are likely to follow some form of emotional disturbance, and consist of such phenomena as palpitations, a lump rising from the stomach to the throat, and feelings of suffocation. The patient gets excited and fights for breath and finally bursts into tears or laughter, or even "faints." The attack may terminate in eructations or with passage of large quantities of urine.

(*b*) *Major Attacks.* These usually develop from some form of minor attack as described above; eventually the patient reaches the ground, sometimes she really seems to fall suddenly, but more often she subsides gradually: she may appear to be unconscious. She often assumes a crucifixion attitude with arms and legs outstretched; she becomes rigid and exhibits convulsive movements (bilateral), and often shows opisthotonos. The eyelids are often tightly shut and the globes turned upwards and inwards. These performances frequently continue for several hours; they rarely last for less than fifteen minutes.

There is no mental disorder or automatism after the attacks and their termination is quite abrupt, but the patient, as a rule, has little or no knowledge of what has happened during the "fit."

The diagnosis from epilepsy has been described (p. 577).

(*c*) *Hystero-Epilepsy* of French writers. This is possibly an exaggeration of the major attacks. Real unconsciousness may develop, but throughout the attack the movements appear to be dominated by some definite subconscious idea. After the attack there may be hallucinations, contractures, paralyses, or even delirium.

(*d*) *Catalepsy.* This consists of profound sleep, in which the limbs assume a plastic rigidity and remain as they are put. The breathing is superficial and the surface temperature subnormal.

II. **Hysterical Affections of the Cranial Nerves.** (*a*) Smell and taste may be abolished.

(*b*) Concentric contraction of the visual fields, either bilateral or unilateral. The disturbance may be for all forms of vision or chiefly for colour vision, in which case the red field is conspicuously well preserved, whereas in organic lesions the red and green fields are affected early.

(*c*) Deafness similar to cortical deafness is fairly common.

(*d*) Anæsthesia of the face, if hysterical, includes the lower jaw, which is really supplied by the cervical nerves.

(*e*) Spasms of the face muscles and the tongue are usually hysterical.

(*f*) In hysterical aphonia the patient can barely whisper. The cords are in the abducted position.

III. **Motor Signs of Hysteria.** (*a*) *Flaccid Paralysis.* This is nearly always accompanied by complete anæsthesia of the affected part or of half the body. A limb may be affected or there may be paraplegia or hemiplegia. There may be general wasting, but there is no reaction of degeneration, no hypotonia, and the reflexes are present—indeed, they are often brisk.

If, in hysterical hemiplegia, the patient is told to sit up in bed the paralysed leg remains quite flat on the bed. In organic hemiplegia under like circumstances the paralysed leg is lifted higher off the bed than the sound one, as the abdominal muscles contract.

(*b*) *Paralysis with Stiffness.* There is usually some power of movement and the stiffness does not amount to true

spasticity. The performance of a voluntary movement is attended by great apparent effort.

A feature of these cases is that when the affected limb is voluntarily moved in a specific manner the *opposing* muscles undergo a slight contraction *before* the muscles concerned in the main movement commence to contract. This does not happen in organic paralysis.

If the legs are affected the gait is shuffling, but does not resemble the gait of a similar organic lesion; commonly the toes are pointed and the dorsum of the foot rests on the floor.

(*c*) *Hysterical Contractures* are due to strong muscular contractions; their nature can be diagnosed by the fact that they disappear spontaneously under an anæsthetic, which does not happen to organic contractures.

(*d*) *Hysterical Tremors.* Almost any form of tremor may be a manifestation of hysteria.

IV. **Sensory Manifestations of Hysteria.** (*a*) *Subjective.* Pain and hyperæsthesia may be complained of, especially in the head, back, side of the chest and left breast.

Local tenderness to pressure is peculiarly well marked.

The clavus hystericus (a feeling as of a nail being driven into the vertex of the skull) is a common manifestation of hysteria.

(*b*) *Objective.* Anæsthesia is one of the most common phenomena in hysteria; it may be the only manifestation or may accompany any of the other signs. It is commonly not known to the patient till her attention is drawn to it.

The site of the anæsthesia is not constant; it shifts from day to day or even from hour to hour.

The loss of sensation may be partial or complete; often the loss is confined to the cutaneous sensibilities. The distribution is rarely in conformity with any possible nervous distribution, either segmental or peripheral.

There is nearly always a definite circular upper limit if a limb is concerned, and in the case of the trunk the loss often ceases absolutely at the mid-line.

Hemianæsthesia is common, as is a quadrantic distribution—for example, the left arm and the right leg.

V. **The Reflexes in Hysteria.** The deep reflexes are usually

brisk. They are never abolished. A pseudo-clonus may be elicited, but not when there is any rigidity. The abdominal reflexes are not lost, though if there is abdominal anæsthesia it may be very difficult to obtain them. There is never a plantar reflex of extensor type.

There is never any further sphincter trouble than retention of urine.

VI. **Special Manifestations of Hysteria.** (*a*) *Anorexia Nervosa.* This condition has been described on p. 350. It may occur alone or with other definite stigmata of hysteria. The chief feature is a persistent refusal of food, often accompanied by prompt regurgitation, or vomiting, of any that may be taken. Emaciation is profound and death from starvation may occur.

The onset of such symptoms during puberty should excite suspicion of a functional cause. This is confirmed by the absence of any œsophageal obstruction or other evidence of organic disease (*vide* also "Œsophageal Spasm," p. 321).

(*b*) *Hysterical vomiting* without anorexia is not uncommon; a feature of this is the absence of nausea or pain and also the fact that in many cases there is little or no loss of flesh. Organic cerebral disease must be excluded.

(*c*) *Air-swallowing with subsequent eructations* is a common manifestation of hysteria; the diagnosis is self-evident.

(*d*) *Hysterical Spine.* Pain in the spine (usually the lower part), with tenderness on pressure, may be hysterical. There is no curvature except possibly a lateral scoliosis from muscular weakness, which immediately disappears on bending down. The tenderness is usually as well marked on light pressure as on deep, and is not sharply localised.

These points and the absence of any root symptoms may serve to distinguish tuberculous disease and also malignant disease, which is unusual before the age of 40 years.

(*e*) *Hysterical Joints.* Tuberculous joints (especially the hip joint) may be closely simulated. There is, however, never *real* shortening, and an X-ray examination would put the matter beyond question.

In conclusion it may be stated that a diagnosis of hysteria is only justifiable when every means has been taken to exclude

organic disease and after a very comprehensive review of the case as a whole.

It must never be forgotten that many cases of organic disease may show hysterical signs also, and that the first signs of organic lesions may be indistinguishable from hysteria.

The special points in diagnosis between hysteria and epilepsy and disseminated sclerosis, have been mentioned under the headings of these latter diseases.

NEURASTHENIA.

Neurasthenia is a condition of fatigue both mental and physical, generally accompanied by pain and other subjective sensory phenomena, but not by any evidence of organic disease of the central nervous system.

The most common age for neurasthenia is 30 to 50 years. Men suffer more often than women, and the predisposing causes are overwork, worry, lack of exercise, certain specific infections, especially influenza and gonorrhœa, and visceroptosis, or more particularly movable kidney.

Excessive child-bearing and the menopause are frequent causes of neurasthenia in women.

Injury or accident may determine the start of neurasthenic symptoms (traumatic neurasthenia), and these may continue indefinitely, or they may clear up when compensation has been paid. Such patients are not necessarily malingerers.

The outstanding Symptoms of neurasthenia are :—

(i.) Irritability and inability to focus the attention or to concentrate on any particular matter or to make up the mind on any given question.

(ii.) Insomnia.

(iii.) Extreme depression, often amounting to a sense of complete hopelessness bordering on melancholia.

(iv.) A tendency to worry about trifles that ordinarily would be brushed aside as of no importance.

(v.) Easy physical fatigue and sense of inability to make any bodily effort.

(vi.) Introspection so overwhelming that the patient can talk of nothing but his own symptoms.

(vii.) Pains in the head, back and limbs ; there is often a tingling sensation in the scalp and down the spine.

Different cases exhibit different clinical types. One will have a gastric type of neurasthenia with prominent dyspeptic phenomena which are associated with secretory neuroses of the stomach or with visceroptosis ; another will have a sexual type and will be convinced that he is impotent or unfit to marry, usually because he has had gonorrhœa or syphilis, or else because he is worried by nocturnal emissions.

Most patients suffering from neurasthenia lose weight, have a subnormal temperature, and are constipated. The urine commonly shows an excess of phosphates, and sometimes there is a definite oxaluria.

It is convenient to consider as a special class of neurasthenia those cases which are more accurately labelled "psychasthenia" and which manifest a definite obsessive idea, such as a fear of the dark, a fear of a crowd, a fear of being alone, a fear of open spaces, etc., etc.

Well-marked cases of psychasthenia are also liable to attacks of giddiness and to curious dreamy states, in which the most prominent feature is a sense of unreality of the surroundings, but in which there is no true unconsciousness, as in epilepsy.

The diagnosis of neurasthenia, as of hysteria, must only be made when the most careful and repeated investigation has failed to show any evidence of organic nervous disease. Movable kidney (especially in women) must always be looked for, not for the purpose of excluding but rather of confirming the possibility of neurasthenia.

It is especially important to exclude the following :—

(i.) *Dementia Præcox.* Neurasthenia does not occur at the time of puberty and is extraordinarily rare before the age of 21 or 22 ; hence neurasthenic symptoms before 20 years of age should arouse suspicion of mental disorder.

(ii.) *General Paralysis.* This may commence with purely neurasthenic symptoms.

There should, however, be some organic signs. If there is doubt an examination of the cerebro-spinal fluid should be made (*vide* p. 512).

PART VI

CERTAIN DISEASES OF THE SKIN

A CERTAIN number of skin affections have already been discussed in so far as they are manifestations of some underlying morbid process—for example, the eruptions associated with some of the specific infective fevers have been described under the headings of those diseases, and the cutaneous phenomena which may be met with in syphilis and tuberculosis have been similarly dealt with. There remains a large number of what may be termed primary affections of the skin in which the local condition is the only, or at any rate the principal, evidence of disease.

An accurate classification of diseases of the skin is impossible, because the pathology of many of these conditions is at present far from clear.

In the short description which follows the more important skin diseases are divided roughly into four groups, an arrangement which is convenient rather than scientifically accurate.

I. Disorders of the Cutaneous Functions.

II. Toxic and Organic Diseases of the Skin.

III. New Growths of the Skin.

IV. Parasitic Affections of the Skin.

The primary lesions which are met with in diseases of the skin may be defined as follows :—

(*a*) *Macule.* A macule is a stain or spot due to pigmentation (*e.g.*, a freckle or the appearance seen after a syphilitic eruption) : it does not fade on pressure.

(*b*) *Papule.* A papule is a small solid elevation of the cuticle with an inflamed base : the size varies from that of a pin's head to that of a pea.

(*c*) *Nodule.* A nodule is a solid elevation of the skin of larger size than a papule.

(*d*) *Vesicle.* A vesicle is a *small* blister-like elevation

of the cuticle: it contains fluid which may be clear or turbid but is not purulent.

(*e*) *Bulla*. A bulla is merely a *large* vesicle.

(*f*) *Pustule*. A pustule is a vesicle with purulent contents.

(*g*) *Pomphus*. A pomphus or wheal is a rounded elevation due to acute inflammatory œdema.

(*h*) *Erythema*. Erythema (strictly speaking) is merely a local congestion or superficial redness of the skin which fades on pressure.

I. DISORDERS OF THE CUTANEOUS FUNCTIONS.

I. **Affections of the Skin.** (i.) PRURITUS. By pruritus is meant the subjective sensation of itching without any further objective change in the skin than may be caused by scratching.

Pruritus may be local or general: the former is usually limited to the rectum or to the female genitals; the latter is seen in the very old, in cases of jaundice, glycosuria or gout, in morphino-maniacs, and also sometimes in cold weather (pruritus hiemalis).

A curious variety of pruritus is that known as "bath pruritus," in which contact with water is followed by intense itching of the part wetted.

The diagnosis of pruritus depends upon the absence of any local changes such as might result from scabies or pediculosis.

The examination of the urine to exclude diabetes must never be forgotten in cases of pruritus, and in young children the fæces must be investigated for the presence of thread worms, which are a frequent cause of localised pruritus.

(ii.) PIGMENTARY CHANGES. (*a*) *Albinism* is a congenital absence of pigment from the skin, eyes, and hair. The eyes are pink and the hair is white. Vision is usually defective, and nystagmus is nearly always present. The condition is often familial.

(*b*) *Lentigo* (Ephelis or Freckles). Freckles are usually most numerous in red-headed persons; they are induced or increased by the rays of the sun and are rare in infancy.

(*c*) *Ephelis ab igne* is the brown, mottled, or marbled

appearance which is seen on the shins, or more rarely on the wrists and forearms, of cooks and persons who are constantly exposed to the heat of a large fire.

(*d*) *Leucoderma* (Vitiligo). In leucoderma there are irregular but sharply-defined pale areas of skin which are usually surrounded by darker skin than normal. The patches may be large or small, and if the scalp is affected the hair of the pale areas is itself colourless.

(*e*) *Chloasma*. This is shown by patches of yellow or brown discoloration (larger than freckles) about the neck and forehead of certain women, especially during pregnancy or at times of menstrual disturbance.

II. **Disorders of the Hair.** (i.) *Alopecia* (Baldness) is a natural manifestation of advancing age, but occurs prematurely in males to a very large extent. In some cases it is hereditary or constitutional, but more often it depends upon a bacterial infection of the hair follicles and sebaceous glands. Partial baldness sometimes follows acute fevers, and is a common manifestation of syphilis.

(ii.) *Alopecia Areata*. This condition is characterised by a rapid and complete baldness over certain well-defined and sharply-delimited areas of the scalp.

In most cases the hair eventually grows again, but the new crop may be white.

The diagnosis must be made from ringworm by the absence of the broken-off stumps, in which the fungus of this latter disease can be demonstrated microscopically.

(iii.) *Canities* or Greyness, is due to loss of pigment from the hairs ; it may be partial or complete, local or diffuse, and, like baldness, is a natural phenomenon of increasing age, though often seen prematurely.

III. **Disorders of the Sebaceous Glands.** (i.) *Milium*. This is a very small, round white tumour in the skin due to the retention of sebaceous matter in a sebaceous gland. The tumours are generally multiple and may be very numerous. They are most common on the eyelids, cheeks, scrotum, and penis. If the tumours are punctured the sebaceous matter can be squeezed out.

(ii.) *Comedo* (Blackhead). This is caused by distension of a hair follicle with sebaceous matter. The opening of the

follicle on the skin is choked with greasy sebum, which soon becomes blackened with dirt and so gives the characteristic appearance.

(iii.) *Seborrhœa.* This is due to excessive secretion of sebaceous matter, and occurs in two main forms—seborrhœa sicca and seborrhœa oleosa.

Seborrhœa sicca always occurs first in the scalp : it is essentially an infective process. An invasion by the bottle bacillus of Unna is the first stage ; this causes epidermal desquamation, which is shown clinically by dry scurfy flakes in the hair.

The second stage commonly occurs about puberty and depends upon an infection with the Staphylococcus epidermidis albus (grey skin-coccus). The scurf becomes more greasy and the hair tends to fall out.

The third stage is one of oily seborrhœa and depends upon the invasion of the fatty matter in the ducts of the sebaceous glands by the acne bacillus.

Primary oily seborrhœa is more common about the face.

Both forms of seborrhœa may be found on the trunk and limbs. Clinically the lesions on the scalp vary greatly in intensity ; there may be nothing more than an excess of scurf, which drops out on the coat-collar, and is especially noticeable on brushing the hair, or there may be definite papular crops round an area of hyperæmia, surmounted by a cake of greasy scales. More or less itching is usually complained of.

Seborrhœa is common in infancy, and if neglected may be followed by eczema.

On the trunk the chief lesions are crops of papules. These run together into scaly patches which are more or less hyperæmic ; they spread peripherally and tend to improve centrally, so that rings are formed. The limbs are rarely affected.

The diagnosis must be made from psoriasis by the softer, greasier, and less glistening character of the scales, by the distribution (psoriasis starts about the knees and elbows), and by the evidence of seborrhœa in the scalp ; and from pityriasis rosea by the absence of the initial papules in this

latter, as well as by its relative preference for the limbs (*vide* also p. 608).

IV. **Disorders of the Sweat Glands.** (i.) *Anidrosis.* Absence of perspiration may occur generally in myxœdema or such wasting diseases as diabetes or locally in ichthyosis, scleroderma, psoriasis, eczema, and the anæsthetic form of leprosy.

(ii.) *Hyperidrosis.* Excess of perspiration may occur in certain febrile wasting diseases such as tuberculosis, in metabolic disorders such as Graves' disease, in certain lesions of the nervous system, or as a constitutional condition.

(iii.) *Bromidrosis.* Foul-smelling perspiration is usually, but not always, associated with excessive secretion. It is seen in certain diseases such as rheumatic fever and scurvy, but also occurs in certain unfortunate persons who are perfectly healthy otherwise. In these cases the bromidrosis is generally localised to the feet. The perspiration does not smell unduly when it is first secreted, but the subsequent odour depends on the presence of the Bacillus fœtidus.

II. TOXIC AND ORGANIC DISEASES OF THE SKIN.

I. **Acne.** (*a*) *Acne Vulgaris.* The essential lesion in acne vulgaris is a red papule which may or may not pustulate. The papules are the direct result of inflammation in blocked sebaceous glands or hair follicles.

The inflammation is produced by the action of the acne bacillus or the staphylococcus epidermidis albus (grey coccus).

The lesions are most prevalent on the face, shoulders, back, and thorax; those that suppurate often leave minute white scars.

Acne vulgaris usually develops about the age of puberty and often disappears at 25 or thereabouts.

Persons with coarse, greasy skins are principally affected.

The diagnosis does not present any difficulty; the distribution of the eruption, the associated comedones, and the age of the patient are usually characteristic, though the possibility

of a pustular syphilide should be borne in mind in doubtful cases and other evidence of syphilis sought for.

(*b*) *Acne Rosacea.* This disease affects the skin of the middle third of the face. It varies in intensity from a simple hyperæmia to a condition of rhinophyma, in which there is hypertrophy and lobulation of the skin of the nose, the result of chronic inflammation around over-secreting or blocked sebaceous glands. There is no tendency to ulceration.

Acne rosacea is more common in women than men, and usually develops in the third or fourth decade ; in either sex dyspepsia and alcoholism are predisposing factors.

The diagnosis is easy. Lupus erythematous can be distinguished by its raised spreading edge and its scaliness ; tertiary syphilis by its symmetry and tendency to ulceration ; acne vulgaris by the younger age, the wider distribution, and the presence of comedones.

II. **Cheiropompholyx.** This condition manifests itself by a symmetrical eruption of vesicles about the extremities, especially about the hands. Women are more affected than men, and the eruption is usually only present in hot weather.

Clinically the first symptom is burning and itching. The vesicles appear after a short interval ; at first they are deeply imbedded in the skin, but as they reach the surface they run together and form large bullæ. The bullæ gradually dry up and leave a crust, which drops off and exposes a very tender area of pink new skin. Relapses and recurrences are common.

Cheiropompholyx can be diagnosed by the characteristic development of the bullæ and their *localisation to the extremities ;* in addition it is distinguished from eczema by the absence of weeping, from pemphigus by the fusion of the vesicles into bullæ, and from ringworm by the absence of the fungus in the scrapings from the lesions.

III. **Eczema.** Eczema is defined by Sir Malcolm Morris[1] as "a catarrhal inflammation of the skin, originating without visible external irritation and characterised by serous exudation at some stage of its evolution." The same author points out that this definition excludes skin lesions produced by chemical or mechanical irritants,

[1] "Disease of the Skin" (Cassell, 1911).

although such lesions clinically and anatomically may be indistinguishable from true eczema.

Description. An attack of eczema is preceded by tingling and itching in the part about to be affected.

The primary lesions may be papular, vesicular, or erythematous. All writers agree that vesicles are the most constant phenomena although they may be extremely small individually.

In the papular type a minute vesicle can often be recognised at the summit of the papule, and these when scratched off may be replaced by a small scab of blood.

There is a great tendency for the disease to spread peripherally while healing centrally, and to heal up temporarily only to break out again in the same or in some other part.

Most cases of eczema present the following more or less ill-defined stages (all of which may be present simultaneously):—(*a*) Erythema. (*b*) Vesiculation. (*c*) The exudation of a clear fluid (which stiffens linen) from a red angry surface. (*d*) Scabbing, in which the discharge forms greyish-yellow crusts. (*e*) Desquamation after the crust formation has ceased.

It is easy to understand that the possibility of secondary infections with pyogenic micro-organisms is very great, and this is enhanced when it is remembered that the itching is often so intolerable that it is out of the question to refrain from scratching. Such secondary infections may modify the primary features of the lesions very considerably; pustules and boils are common, so that it may be said that there is no skin disease which may present such varied appearances as eczema.

The distribution of eczema presents certain important features, though there is no part of the body that may not be affected. There appears to be an especial predilection for the *flexor aspects of joints*, the grooves behind the ears, the scalp, the breasts of women, and the palms of the hands and soles of the feet. The genitals, the arms, the umbilicus, and the nipples are not infrequently attacked.

Eczema is especially common in infancy and in old age. In infants the scalp and face are particularly prone to attack, but the whole body may be involved.

Seborrhœic eczema, which is probably nothing more than eczema which has developed on a chronic seborrhœa, is the variety usually met with in infancy, childhood, and at puberty.

An interesting clinical point is the tendency for chronic eczema to alternate with certain chronic conditions, especially bronchial asthma and gout; when the patient has eczema he is free from asthma or gout, and *vice versâ*. In like manner it is only too frequently found that an infant with acute eczema recovers from the eczema only to perish forthwith from acute pneumonia; this distressing sequel has especially been observed in those babies whose eczema has yielded exceptionally quickly to treatment.

Diagnosis. The diagnosis can usually be established if a careful examination of all the lesions is made with a lens after bathing off all crusts and secretions.

Erythema multiforme presents neither scales nor weeping.

Secondary syphilis does not itch—there are other evidences of syphilis; the treponema can be demonstrated in scrapings from the lesions, and the Wassermann reaction is positive.

Erysipelas is generally accompanied by severe constitutional disturbance and shows a typical raised edge (*vide* p. 25).

Scabies should be suspected from the distribution (*vide* p. 616) and from the linear arrangement of the lesions. Proof positive is afforded by the discovery of the characteristic burrows in the skin.

Tinea cruris and *Tinea circinata* can be diagnosed by the discovery of the fungus.

Psoriasis never "weeps" and has a much more definite edge than eczema; its predilection for the extensor aspects of the elbows and knees is in distinct contrast to most cases of eczema and the scales are much more white and "silvery."

Lichen planus never discharges or forms crusts, and the individual papules are a much darker red than those of eczema.

Favus has cup-shaped sulphur-yellow crusts and possesses a strong odour of mice; further, the specific fungus can readily be demonstrated.

Pityriasis rubra does not itch, does not "weep," and does not form crusts.

The frequency with which eczema (especially of the genitals) is found as a manifestation of diabetes indicates the necessity for examining the urine for sugar in all cases. The possibility of an underlying gouty diathesis must also be borne in mind.

IV. **Erythema.** Two main groups of erythema can be recognised—first, hyperæmic erythema, and, secondly, inflammatory erythema. In the hyperæmic form the initial active congestion is followed by passive congestion from vaso-motor paralysis, and the bright red colour is replaced by a livid purple as the sensation of heat disappears and the local temperature falls.

In the inflammatory form there is stasis of the blood-stream, with the possibility of subcutaneous hæmorrhages and the occurrence of vesicles, œdema, pigmentation, etc.

Presumably erythema is the result of vaso-motor instability, whereby there is an excessive response to stimulation, whether direct (heat, cold, fomentations, insect bites, etc.) or indirect (toxins of rheumatism, gout, intestinal stasis, etc.).

Hyperæmic Erythema. The following varieties are described :—

(*a*) *Erythema Simplex.* Hot, red patches, which may occur anywhere, but are most frequent on the face and other exposed parts. There is itching and a sensation of heat. After a variable time the colour fades, the heat disappears and a branny desquamation often follows. There is no constitutional disturbance and no well-defined edge as in erysipelas.

(*b*) *Erythema Fugax.* Except for its transient character, erythema fugax is identical with erythema simplex. It is especially liable to develop in children from the reflex irritation associated with gastro-intestinal disturbance, teething, worms, etc.

Inflammatory Erythema. The following varieties are described :—

(*a*) *Erythema Intertrigo*, which develops where opposing skin surfaces are in contact, especially in the groins, thighs, axillæ, and under pendulous breasts. Eczema often supervenes in these cases.

The napkin rash of infants is an example of this form

of erythema ; it may closely resemble a congenital syphilitic rash, but is strictly confined to the area covered by the napkin, whereas the syphilitic rash is not so limited.

(*b*) *Erythema Paratrimma* is the livid discoloration which precedes the formation of a bedsore.

(*c*) *Erythema Lœve* is the dusky appearance which is so often seen in œdematous legs and which is sometimes a warning that sloughing is about to occur.

(*d*) *Erythema Pernio* (Chilblain) is met with in cold weather, and principally in children and the aged. The characteristic lesion is the formation of small dusky, red or even bluish patches on the hands and feet, though the ears and nose may be affected also.

The dorsum and sides of the fingers and the heel and outer edge of the foot and the little toe are the most common sites. Itching is extreme and there is great local tenderness ; ulceration is not uncommon.

(*e*) *Erythema Multiforme.* This condition is a toxic inflammation of the skin : the nature of the toxin or toxins is uncertain ; probably many can produce the same result. There is no evidence that rheumatism is especially associated with this condition.

The lesions may be papular, vesicular, œdematous, nodular, hæmorrhagic, or bullous.

There is usually constitutional disturbance at the outset, such as fever and malaise, as well as sore throat, joint pains, and gastro-intestinal disturbance.

The eruption is usually first seen on the backs of the hands or the dorsum of the feet, and spreads thence up the limbs to the trunk and face.

The primitive lesion is generally a crop of tiny bright-red papules. The skin surrounding the individual lesions is congested.

The patches often subside in the centre while spreading peripherally, so that ring and serpentine forms are common (erythema annulare ; erythema gyratum).

Vesicles and bullæ may form at the margins of the lesions and hæmorrhages may occur into the papules.

The average duration of erythema multiforme is four to six weeks, but recurrences are very common.

Erythema Iris is a variety of erythema multiforme, but it is frequently the only manifestation. The appearances are characteristic : there is a central papule surmounted by a vesicle and surrounded by an inflammatory areola on which a ring of secondary vesicles appears, whilst outside these again there is a second and even a third ring of vesicles.

The primary central vesicle dries up and forms a scab which drops off.

Occasionally bullæ replace the vesicles in erythema iris.

(*f*) *Erythema Nodosum*. The characteristic feature of this condition is the development of multiple node-like swellings on the legs and feet and less often on the thighs, forearms, and shoulders. The nodes are roughly oval in outline and tend to have their long axes in that of the limb ; they vary in size from that of an almond to that of a hen's egg, and are very tender but not painful. At first they are bright red in colour ; later they go through all the colour changes of a bruise ; they appear to soften almost to the point of fluctuation, but never suppurate.

Each node lasts for about fourteen days, but successive crops appear at short intervals, so that the duration of the illness is about four to six weeks.

Children and young adults are most frequently affected, and the female sex predominates.

There is sometimes a mild constitutional disturbance in the early stages and nearly always pain in the joints and down the legs. It was formerly thought that erythema nodosum was a manifestation of rheumatism, but careful analysis of many cases has shown that this hypothesis is probably incorrect.

The nature of the toxin which is responsible for this form of erythema remains unknown, but it is significant that a very large percentage of cases give a positive cutaneo-tuberculin (von Pirquet) reaction.

The diagnosis of erythema nodosum is usually quite obvious. Septic conditions such as osteomyelitis are accompanied by pain and severe constitutional disturbance. The early stages of nodular leprosy can be diagnosed by the presence of anæsthesia in or about the nodules ; gummatous

formation is not accompanied by joint pains and tends to ulcerate.

(*g*) *Erythema Scarlatiniforme.* This condition may simulate scarlet fever very closely; there is febrile disturbance, followed in a few hours by a vivid red erythema which may invade the whole body. The tongue is foul with prominent papillæ and the throat is injected. Relapses are common, and the duration of illness may be many weeks.

A positive diagnosis between this condition and scarlet fever is impossible in the early stages; the most striking difference is the early onset of desquamation (often on the second day) in erythema scarlatiniforme.

The rash of scarlet fever never lasts more than eight to ten days, whereas in erythema scarlatiniforme it may persist for several weeks.

V. **Herpes.** (*a*) *Herpes Facialis.* This is characterised by an eruption of vesicles on a red hyperæmic base. The usual site is the muco-cutaneous junction of the lips and nose; the mucous membrane of the cheeks or tongue may be affected. The condition is often bilateral and is commonly seen in ordinary colds. The occurrence of labial herpes in lobar pneumonia and cerebro-spinal meningitis is a striking feature of these diseases.

(*b*) *Herpes Genitalis* is a similar condition to the above which affects the prepuce and glans penis in men or the labia and cervix uteri in women.

(*c*) *Herpes Zoster* (Shingles). This affection is characterised by the eruption of clusters of vesicles on an erythematous background along the area of cutaneous distribution of one or more posterior nerve roots. There is often severe pain before, during, and even after the eruption.

Herpes zoster is nearly always unilateral, hardly ever recurs, and depends on some inflammatory or hæmorrhagic lesion in the corresponding posterior root ganglia.

VI. **Dermatitis Herpetiformis.** This is a chronic affection of the skin characterised by polymorphous lesions, tending to herpetiform, pemphigoid, and urticarial types, accompanied by severe itching, but not as a rule attended by any considerable constitutional disturbance. Scarring and pigmentation are common when the lesions heal.

The affection is bilateral, and both skin and mucous membranes may be involved. The sexes are equally liable and all ages are attacked.

An interesting feature is the presence of an excess of eosinophile cells both in the blood and in the fluid from the bullæ.

The diagnosis depends upon the diverse character of the lesion, the intense itching, the frequent relapses, and the bilateral distribution.

Herpes zoster is distinguished by its accurate localisation to certain nerve root areas and its unilateral distribution.

VII. **Impetigo Contagiosa.** The disease is seen as a rule in the children of the poor who are neglected and underfed. It is caused by a streptococcal infection of the skin and manifests itself as a pustular eruption, usually about the head and face.

The primary lesion is vesicular, but the vesicules soon pustulate.

The pustules dry up into scabs, but often coalesce first, so that large crusts are formed.

A striking feature is the absence of any hyperæmia around the pustules or scabs.

The neighbouring lymph glands are generally enlarged, but the constitutional disturbances are relatively slight. The disease is readily inoculable by means of the discharges.

The only difficulty in diagnosis lies in excluding a pustular form of eczema. The chief points are that there is no inflamed skin round the individual lesions in impetigo, that there is little or no itching, that there is no attempt at symmetry, and that the disease clears up with great rapidity if suitable treatment (*e.g.*, white precipitate ointment) is applied.

VIII. **Lichen Ruber Planus.** The characteristic lesions in this affection are small, irregularly-shaped papules, generally with flat tops though sometimes umbilicated.

The papules are purple in colour, and in the centre of each is a small scale. They tend to arrange themselves in rings or lines, are very closely packed, and often confluent.

The usual situations are the flexor aspects of the wrists, the popliteal spaces, and the limbs generally, but the whole

body may be affected, and in a considerable proportion of cases the mucous membranes are involved also.

The rash is always dry and as it fades leaves dark stains which gradually fade into white patches. True scars are never met with. Itching is often severe.

Lichen planus may occur at any age, though children and the very old usually escape. The causation is unknown, but it may follow profound emotional disturbance.

The diagnosis must be made from—

(*a*) *Papular Eczema* (*vide* p. 598).

(*b*) *Psoriasis* by the fact that the individual papules do not spread out into scaly patches and that the general scaliness is much less marked (*vide* p. 610).

(*c*) *Papular Syphilides* by the dryness of the papules and the Wassermann reaction.

(*d*) *Pityriasis Rubra Pilaris* (*vide* p. 608).

IX. **Lupus Erythematosus.** This is an inflammatory affection of the skin which usually is confined to the cheeks, nose, and ears, though other parts may be affected. In many cases there is a pre-existing chronic seborrhœa. Women are much more commonly affected than men, and the disease usually starts between the ages of 25 and 45. It runs a very chronic course and tends to die out in from fifteen to twenty years, leaving, however, permanent atrophic deformity of the affected skin.

The primary lesions are minute red papules which increase and coalesce to form rounded patches. They usually appear first on the cheeks and spread centrally to join each other over the bridge of the nose, thus causing the typical "bats-wing" appearance. The primary spots have red, elevated and thickened borders, which may be covered by a thin scab or by papery scales. Around the patches, in those parts in which there are sebaceous glands, comedones can be observed to a greater or less extent.

The lesions of lupus erythematosus are usually symmetrical. Sometimes the disease advances by the peripheral spread of the primary patches (discoid form), in others by the appearance of fresh crops of spots. The mucous membranes may be involved, but usually only by direct extension.

The diagnosis depends upon the chronicity of the disease combined with the characteristic appearance of the lesions, which show an atrophic centre, a raised red edge, and a girdle of sebaceous plugs. The process is always superficial and never ulcerates.

In *Lupus Vulgaris* the primary lesion is a soft nodule, the disease invades deeper structures, ulceration is common, and symptoms nearly always appear before puberty.

In *Acne Rosacea* there is no central scar and no surface scab.

X. **Pellagra.** A disease of obscure causation, endemic in Italy, Roumania, Egypt, etc. It probably has a far wider incidence really, since cases have been recorded in England, France, and Spain, and it is by no means uncommon in America.

It was for a long time thought that pellagra resulted from eating overmuch maize, especially decomposed or fermenting maize, but recently Dr. Sambon claims to have discovered a protozoon which is the cause of pellagra and which is conveyed to man by the bite of a small black fly. The question as yet remains uncertain.

The course of pellagra is fairly constant ; the first symptoms are usually noticed in the spring, and consist of pain in the back, limbs, and joints, with fever, gastro-intestinal disturbance, and general malaise. A cutaneous eruption soon appears, chiefly on the exposed parts of the arms and hands : it is primarily erythematous in character but petechiæ and bullæ are often seen ; the bullæ rupture and leave chronic ulcers. The eruption clears up in about two weeks with free desquamation, leaving thickening and yellowish-brown coloration of the underlying skin. At the same time a spastic paresis develops, particularly in the lower limbs.

Each spring there is a further similar outbreak, and eventually the skin where the rash appears becomes dry, wizened, and atrophic.

The paralytic phenomena progress slowly but steadily, and mental changes comparable to those of general paralysis develop. Many patients become melancholic with strong suicidal tendencies and eventually find their way into

asylums. Life is prolonged from from five to fifteen years after the first attack.

XI. **Pemphigus.** This consists in a bullous eruption developing on previously healthy skin.

Three varieties are described :—

(*a*) *Pemphigus Vulgaris.* There is usually some febrile disturbance at the outset. The bullæ develop in a few hours into tense hemi-spherical blisters ; they vary greatly in size and number, and have no surrounding areola of inflammation or characteristic distribution. The contents are clear at first, but soon become opaque, and in about three days the bullæ dry up to leave a brownish scab which drops off and exposes a purple patch of new skin. This, however, eventually assumes a quite normal appearance. The bullæ appear in successive crops, so that the condition may persist for weeks or months.

The mucous membranes are very rarely involved, neither do the bullæ fuse with their neighbours as a rule. Sometimes there is hæmorrhage into some of the bullæ.

Relapses are fairly common in pemphigus vulgaris, and sometimes the disease becomes practically continuous. It sometimes happens that the eruption is so extensive and the constitutional disturbance so severe that death takes place in a few weeks.

(*b*) *Pemphigus Foliacus.* This variety is rare : the initial bullæ are not rounded and tense, but more or less flaccid from the outset ; they break readily and form yellowish crusts, which become separated and expose an excoriated, angry-looking surface. The lesions progress steadily, until after many months the entire skin as well as large areas of the mucous membrane may become involved. There is constant fever and extreme discomfort and distress. Most cases end fatally from exhaustion and toxic absorption.

(*c*) *Pemphigus Vegetans.* This variety is extremely rare : the initial lesions are small bullæ, which gradually infiltrate and elevate the surrounding epidermis. Excoriation readily takes place and leaves a bare patch, on which a condylomatous growth is formed in four or five days. This papillary outgrowth discharges a thin, foul-smelling secretion and is surrounded by a ring of secondary bullæ.

The female genitals, the axillæ, the mouth, the hands, and the feet are usually the first parts to be attacked; but gradually large areas of skin are involved, the epidermis strips off in large sheets, and superficial gangrene eventually develops. A fatal result is to be expected.

In all forms of pemphigus there is a well-marked eosinophilia.

Diagnosis. *Pemphigus Vulgaris* is distinguished from dermatitis herpetiformis and bullous varieties of urticaria or erythema by the single character of the lesion, with the absence of erythema or urticaria around the bullæ; further, the pigmented areas which represent earlier bullæ are quite characteristic.

Pemphigus Foliaceus is distinguished from eczema by its larger scales and wider distribution.

Pemphigus Vegetans, even more than the other varieties of pemphigus, must be distinguished from a syphilitic condition. In pemphigus vegetans the surface of the papillary outgrowths is excoriated and warty; in a syphilitic condyloma the surface is smooth and even. A ring of bullæ round the papillary outgrowth is very much against a diagnosis of syphilis. In all doubtful cases a Wassermann reaction should be performed.

XII. **Pityriasis Rubra** (Exfoliative Dermatitis). This condition may be primary or it may be secondary to such pre-existing skin diseases as eczema, psoriasis, or lichen planus.

The onset is sudden with more or less malaise, shortly followed by the appearance of symmetrical red patches anywhere on the body. These patches spread with great rapidity and fuse with their neighbours, until within a few hours the entire body is involved from head to foot.

The skin is bright scarlet, but soon becomes covered with thin, papery scales, which overlap each other and vary considerably in size. There is no crust formation and but rarely any exudation, though sometimes there is a certain amount of perspiration. There is little or no itching. Desquamation is naturally most abundant.

The diagnosis (according to Morris) depends upon "the vivid redness of the eruption, the rapidity of its effusion its

universality, the profuse desquamation with papery scales and sheets, the frequent absence of itching, and the tendency for there to be serious impairment of health or even death."

XIII. **Pityriasis Rosea.** This condition is characterised by the occurrence of slightly raised, pink patches or circles which are thinly covered with small scales.

The primary patch usually appears on the abdomen; it spreads at its edge and fades at its centre, and in about a week secondary patches develop in other places.

The disease spreads rapidly and in a few weeks may cover the trunk; the forearms and legs below the knee usually escape.

In from one to two months the process terminates spontaneously. There is little or no itching.

Pityriasis rosea must be distinguished from—

Seborrhœa Corporis by the absence of the initial papules, the distribution and rapid spread (seborrhœa corporis avoids the limbs and prefers the back of the trunk to the front), the dryness of the scales, and the absence of the bottle bacillus.

Psoriasis by its more rapid development, different distribution, and relatively slight scaliness.

XIV. **Pityriasis Rubra Pilaris** (Lichen Ruber Acuminatus). The characteristic feature is the development of small hard, dry, red, conical papules at the orifices of hair follicles. Each papule has a single atrophic hair in its centre. The papules are very small and impart a definite roughness to the skin. They are most abundant on the backs of the fingers, on the outer side of the forearms, and on the thighs and buttocks—that is to say, where there is most hair. Itching is slight or absent.

The papules tend to coalesce to form pale yellowish-red patches covered with small papery scales.

The diagnosis depends upon the discovery of the small conical papules with the single hair plugging the mouth of a follicle. If these papules are pulled off small pits are left.

Lichen planus can be recognised by the itching, the distribution, the flat or umbilicated papule, and the beneficial reaction to arsenic.

XV. **Prurigo.** (*a*) *Simple Prurigo.* Some writers consider that the papular rash with subsequent roughening

of the skin is merely the reaction to scratching in a case of pruritus. Most authorities, however, consider that the eruption is an essential feature of the disease.

Simple prurigo may develop in childhood, but is more common between the ages of 20 and 30. There is intense local itching, which is intermittent and which is followed by the development of a crop of papules. These tend to group themselves into three concentric zones and then form what is called a "plaque." Prurigo generally attacks the neck, the thighs, the loins, the axillary and popliteal folds, the genitals, or the palms of the hands and the soles of the feet, but any part may be affected. Eventually the affected skin becomes thickened and wrinkled (lichenification) and often assumes a brownish tint. Relapses are very common.

The diagnosis is based upon the extreme itching, the long duration, the character of the papules, and the lichenification.

(*b*) *Hebra's Prurigo.* This commences in the first year of life and tends to persist till death. The characteristic feature is an eruption of papules on the extensor aspects of the limbs and on the chest, back, abdomen, and buttocks.

Itching is very severe. The papules are at first of the same colour as the skin, but after they have been scratched they become red and are generally surmounted by a blood crust.

The lymphatic glands in the groins and axillæ are nearly always enlarged.

Eventually the affected skin, especially on the legs and forearms, acquires a roughness which gives almost the sensation of touching canvas.

This peculiar feeling of the skin, together with the papular rash on the extensor aspects of the limbs, the age of onset, the persistence of the disease, and the enlarged lymphatic glands, is generally sufficient to ensure a correct diagnosis.

XVI. **Psoriasis.** This disease is characterised by the occurrence on the skin of dry patches which are covered by shining white scales. The amount of the scales varies form a thin layer to a large heaped-up mass. If the scales (which are quite adherent) are pulled off a smooth hyperæmic surface is left on which are dotted a variable number of

deep-red spots. These are the summits of congested papillæ and bleed readily.

The individual patches are sharply circumscribed, and even when they are actively spreading there is only a very narrow zone of hyperæmia around them. As the patches fade they can be observed to clear up first in the centres, so that ring forms with a gradually diminishing border are common.

Psoriasis lasts for months or years, but often clears up completely for a time, only to recur with renewed vigour later on. All parts of the body may be affected, but the face usually escapes. In practically every instance the disease *starts* on the tips of the elbows and on the front of the knees below the patellae. The extensor aspects of the limbs are preferred to the flexors, and the lesions are nearly always symmetrical.

The scalp is very often affected, and typical lesions on the palms and soles are common. The nails may become involved, and even pushed out of their beds, as the psoriasis develops in the matrix. There is no itching.

Diagnosis. This depends upon the site of origin on the elbows and knees, the preference for the extensor surfaces of the limbs, the silvery scales, the sharp border of the lesions, the absence of exudation, and the chronicity.

Eczema weeps sooner or later, commences as vesicles, prefers the flexor aspects, has no well-defined margin, and tends to form crusts.

Lichen Planus prefers the flexor surfaces, has no scales like those of psoriasis, and is formed by an aggregation of multiple bluish-red papules, whereas psoriasis is formed by the peripheral spreading of single bright-red papules (the papules are bright-red until they are covered up by the scales).

Pityriasis Rubra develops very rapidly, involves the entire body, and is covered by thin wafery scales through which the red skin is clearly seen.

Pityriasis Rosea (*vide* p. 608).

Syphilis may cause a psoriasiform eruption identical with true psoriasis. Other signs of syphilis must be sought for, and if necessary a Wassermann reaction performed.

XVII. **Sycosis.** This disease is due to a skin infection with Staphylococcus pyogenes aureus limited to the hairy parts of the face.

The essential lesions are papules which form round the hairs and speedily pustulate. Each pustule is pierced by a hair. They spread rapidly, but never advance to the non-hairy parts. The pus dries into adherent yellowish-brown crusts, and the hairs become loose as their follicles become filled with pus, but baldness does not follow. There is little or no itching.

The diagnosis rests upon an obvious inflammatory lesion starting in the hair follicles and never spreading away from the hairy parts.

In *eczema* the inflammation is less acute, the follicles are only involved secondarily, and the process is not limited to the hairy parts; in *Tinea barbæ* the primary lesion is a round red, scaly patch (ringworm) and the fungus can be discovered in the affected hairs.

XVIII. **Urticaria.** This condition is characterised by the appearance of wheals on the skin or mucous membranes. These raised areas of skin are at first bright red and are often surrounded by an erythematous border; later they become white and anæmic in the centre. The individual wheals vary greatly in size; they develop quite suddenly, itch intensely, and last from a few minutes to a few hours; fresh crops may, however, appear, and the whole attack may last for days, months, or even years.

Any part of the skin may be affected, and there is no tendency towards symmetrical arrangement.

Sometimes the individual wheals are small and leave papules as they subside (lichen urticatus or urticaria papulosa), sometimes there is hæmorrhage into the wheals (purpura urticans), and sometimes bullæ develop on the surface of the wheals (urticaria bullosa).

It has been suggested that urticaria is due to over susceptibility to foreign albuminoid bodies; certainly it is produced very readily in certain people by special articles of diet.

The diagnosis depends upon the sudden onset, the characteristic wheals, and the rapid subsidence of the individual lesions.

XIX. **Certain Drug Eruptions.** (1) *Acetanilide.* A slate-blue discoloration of the skin.

(2) *Anti-toxin.* Erythema and urticaria.

(3) *Arsenic.* Erythema, urticaria, eczema, herpes zoster and keratosis.

(4) *Belladonna.* Scarlatiniform, erythemato-papular rash.

(5) *Borax.* Psoriasiform eruption.

(6) *Bromides.* Erythema, urticaria, or acneiform rash.

(7) *Chloral hydrate.* Erythema.

(8) *Copaiba and similar oleo-resins.* Erythematous patches which itch severely.

(9) *Digitalis.* Urticarial or papular rash.

(10) *Enemata.* Scarlatiniform eruption.

(11) *Iodide or iodoform.* Erythematous, bullous, purpuric, or acneiform rash.

(12) *Opium.* Erythema or urticaria.

(13) *Phenazone.* Erythema or papular rash, sometimes morbilliform.

(14) *Quinine.* Scarlatiniform, morbilliform, or urticarial rash.

(15) *Salicylates.* Scarlatiniform, morbilliform, or urticarial rash.

(16) *Santonin.* Urticaria (and xanthopsia).

III. CERTAIN NEW FORMATIONS.

Innocent growths only will be described. For a description of malignant disease of the skin the student is referred to text-books of Surgery and Pathology.

I. **Corn** (Clavus). A corn is an overgrowth of epidermis, produced as a rule by pressure irritation. The deeper part of the corn in the form of a cone presses inwards on the corium, which may become inflamed. Corns may be hard or soft, according as to whether there is local moisture or not. If a bursa develops underneath a corn a bunion is produced.

A callosity differs from a corn in that the epidermal thickening is purely superficial and there is no core pressing downwards upon the corium.

II. **Wart** (Verruca). A wart is produced by local over-

growth of cutaneous papillæ. Warts are the result of chronic irritation or of an infective process (*e.g.*, gonococcus), though the nature of the infection is in many cases obscure.

Warts may be either protuberant or flat: the former variety are common about the hands and fingers; the latter are often seen on the forehead, back, shoulders, and on the soles of the feet in the form of a flat, raised surface which looks rather like a callosity, but which can be shown to have papillary processes extending quite deeply into the true skin.

III. **Cheloid.** A cheloid is a fibrous tissue overgrowth originating in a scar. It usually appears as a white or pinkish swelling of irregular shape, sometimes with a depressed centre, tending to extend laterally by claw-like processes. The sternum, trunk, face, and head are the most usual sites for cheloid growth.

IV. **Fibroma.** (*a*) *Fibroma Molluscum.* Multiple-rounded or pyriform swellings, softish in consistence, usually pedunculated, but sometimes sessile and covered by smooth skin. They vary in size from that of a hemp-seed to that of an orange.

(*b*) *Von Recklinghausen's Disease.* This disease is characterised by multiple cutaneous fibromas, pigmentation of the skin in the neighbourhood of the tumours, and sometimes by diffuse fibrous tissue overgrowth in the nerves, especially the nerves of the upper extremity.

The cutaneous fibromas may be definitely in association with various nerve trunks, and when this is the case they are composed of both fibrous and nervous tissues (neurofibroma). Arthritis deformans and papillary tumours are recorded as rare manifestations or complications of von Recklinghausen's disease.

(*c*) *Hard Fibromas.* These tumours usually arise in the corium, but sometimes in the nerve sheaths. They are usually multiple, grow slowly, and may reach a large size.

V. **Molluscum Contagiosum.** These tumours are rounded mother-of-pearl-like growths about the size of a pea, with a depression at the summit in which there is a small hole leading to the centre of the tumour.

Molluscum contagiosum is most often seen in children and

on the eyelids and face, but the neck, limbs, and genitals may also be affected.

The pathology is obscure; under certain circumstances inoculation experiments have been successful, so that possibly the disease is infective. Chickens and other birds often suffer from molluscum contagiosum, so that it is conceivably acquired from them in some cases.

The diagnosis is easy; if necessary the growth may be squeezed and the milk-white fluid which is expressed can be examined for so-called "molluscum bodies," which are clear oval bodies the result of degeneration in epithelial cells.

VI. **Xanthoma.** This consists in the formation of yellowish-white plates, or more rarely nodules, which are imbedded in the corium.

Xanthoma usually affects the upper eyelid, sometimes the lower eyelid also, and more rarely the skin in other parts, or even the mucous membranes.

The formation appears to consist of pigmented skin overlying a loose fibrous-tissue reticulum in which are imbedded numerous multi-nuclear connective tissue cells filled with fat droplets.

Xanthoma is associated with diabetes in many instances, but may be a congenital or familial condition.

The diabetic variety differs somewhat from the other forms in that the tumours are red at first and there is a predilection for their development on the extensor surfaces of the forearms.

VII. **Ichthyosis.** This is a congenital abnormality (often familial), characterised by hypertrophy of the papillary layers of the skin and brittleness of the epidermis, which cracks in a lozenge-shaped manner.

In mild cases dryness and roughness of the skin is all that can be noticed (xerodermia); in well-marked cases the skin resembles that of a reptile.

IV. PARASITIC AFFECTIONS OF THE SKIN.

A. Diseases due to Vegetable Parasites.

I. **Ringworm.** The ringworm fungus may be large-spored or small-spored. The former has two principal varieties—

Trychophyton endo- or ecto-thrix—according as to whether it is found inside or outside the hairs ; the latter is always outside the hairs.

(*a*) *Ringworm of the Scalp* (Tinea tonsurans). In the great majority of cases in England this is due to the small-spored fungus (microsporon Audouini) ; sometimes, however, the Trychophyton endothrix is the cause. Ringworm of the scalp is practically confined to children, and is very rarely seen after the age of puberty. It is extremely contagious. Clinically it first manifests itself as a small bare spot on the scalp, on which such hairs as remain are broken, bent, and inelastic. The skin is often scaly and reddened.

The diagnosis can be made with certainty by pulling out some of the affected hairs, soaking them in ether to remove grease, and examining them microscopically in a drop of liquor potassæ. The small spores and narrow mycelial fragments are arranged irregularly around the hair.

(*b*) *Kerion*. This is a variety of ringworm which produces such a severe inflammatory reaction in the hair follicles that the affected patch becomes swollen and boggy and the follicles ooze pus. Kerion is due to infection with a trychophyton.

(*c*) *Ringworm of the Body* (Tinea Circinata). Any of the ringworm fungi may cause an infection of the skin of the body, but an endothrix trychophyton is the usual cause.

Circular red patches develop on the skin, often covered with scales, sometimes with vesicles or pustules. They tend to heal centrally, so that ring forms are produced.

The diagnosis is confirmed by the demonstration of the fungus or by the rapidity with which the patches disappear under treatment.

(*d*) *Ringworm of the Beard* (Tinea barbæ). This is usually caused by an ectothrix trychophyton derived from the horse. The first sign is the development of a red scaly patch ; in a short while pustules develop in the hair follicles on this patch, while other similar patches form locally and become confluent.

The condition must not be confounded with coccogenic sycosis (*vide* p. 611).

(*e*) *Ringworm of the Nails* (Tinea Unguium). This is a

rare and obstinate condition and always due to a trychophyton derived from an animal. The nails become thick, opaque, and brittle.

II. **Favus** (Tinea favosa). The fungus of this disease is the achorion Schonleinii. Children are the chief sufferers, but amongst lower animals mice are extremely susceptible. The fungus nearly always attacks the scalp, but the nails and smooth skin may be affected.

On the scalp the hair follicles are picked out and cup-shaped crusts of sulphur-yellow colour are formed on them. There is a distinct odour of mice.

The appearance is fairly characteristic, but the diagnosis can be made certain by the demonstration of the spores and mycelium on the affected hairs and in the epidermis.

III. **Tinea Versicolor.** This is due to the presence in the cuticle of the microsporon furfur. This fungus shows itself by the formation of roundish, slightly-raised brown scaly patches on the trunk. The patches are extremely superficial and can often be scraped off to a large extent with the finger-nail. There is little or no itching unless the patient sweats a great deal.

IV. **Erythrasma.** This disease is produced by the microsporon minutissimum. It forms reddish-brown patches, not unlike those of tinea versicolor, in the genito-crural region. It is rare in England.

B. Animal Parasites of the Skin.

I. **Acarus Scabiei** (Sarcoptes Hominis: Itch Mite). Scabies is a dermatitis with secondary effects from scratching and sepsis. The cause is the itch mite, or rather the female itch mite, which excites the dermatitis while making a burrow in the skin in which to lay her eggs.

The burrow is about $\frac{1}{4}$ to $\frac{1}{2}$ inch long, and in it are deposited about fifty eggs in the course of two months. The eggs take a week to hatch and then the embryos crawl out of the burrow; the male parasite does not live in the burrow.

The female is larger than the male, being just visible to the naked eye as a whitish speck; she has a circular body and eight legs, of which the four front ones have suckers and the four hind ones bristles. The two median hind legs of the male have suckers as well as the four front legs.

The lesions of scabies are seen chiefly where the skin is thinnest—that is to say, the webs of the fingers, the front of the wrists, the ankles, genitals, breasts, etc.

The only characteristic lesions are the burrows, each with a vesicle at its entrance, and, if these have been destroyed by scratching, a linear arrangement of the scars and scabs.

Apart from the above any lesion that can be produced by inflammatory reaction may be present.

A pustular eruption on the hands should always arouse suspicion. The only subjective symptom is itching, and this is intense.

II. **Pediculus Capitis** (the Head Louse). This is an oval insect about $\frac{1}{10}$ inch long with six clawed legs. The male has a penis on its back and the posterior end of the female is notched. The female is distinctly larger than the male.

The eggs (nits) are deposited on the hairs, to which they are tightly adherent; they can be seen as semi-transparent oval bodies sticking to the hairs; they hatch in five days.

These pediculi live by sucking the blood, and the irritation caused by this and by their perambulations causes the only symptom, namely, itching. Scratching, with secondary infection of the lesions so produced, may result in an extensive pus infection of the scalp. Enlargement of the lymphatic glands in the posterior triangles of the neck is common.

III. **Pediculus Vestimentorum aut Corporis** (the Clothes Louse). This insect is longer and slimmer than the head louse; it lives in the folds of the clothing, especially the neck-band of the shirt, and there the female lays her eggs, which are minute, spherical yellow bodies.

The clothes louse, like both other varieties of pediculus, lives by inserting its proboscis into the mouth of a sweat gland and sucking blood therefrom. This blood suction causes the minute hæmorrhagic points which are so characteristic of pediculosis.

The only symptom is itching, but, of course, secondary dermatitis is common.

Vagabond's Disease, a peculiar harshness of the skin with more or less deep pigmentation, is probably attributable to pediculosis, dirt, and other parasites.

IV. **Pediculus Pubis** (the Crab Louse). This insect is

shorter and broader than either of those just mentioned—indeed, it bears a distinct resemblance to a crab. It may occur all over the hairy parts of the body, but greatly prefers the pubic region, and is rarely or never found in the scalp.

The ova are attached to the hairs close to the skin. Itching is severe, and a papular (pruriginous) eruption is not uncommon.

Characteristic patches of steel-grey discoloration (maculæ cæruleæ) are seen on the skin of persons who have harboured pubic pediculi for any length of time. These are thought to be due to a colouring matter present in the thorax of these lice, which is presumably injected under the skin during the process of blood-sucking. The patches soon disappear when the parasites are destroyed.

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