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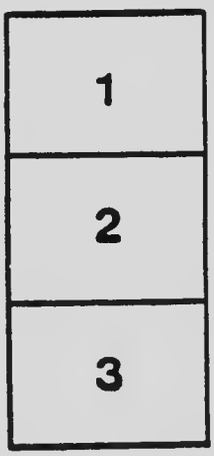

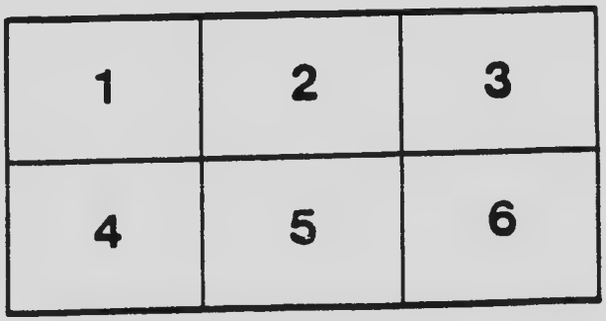

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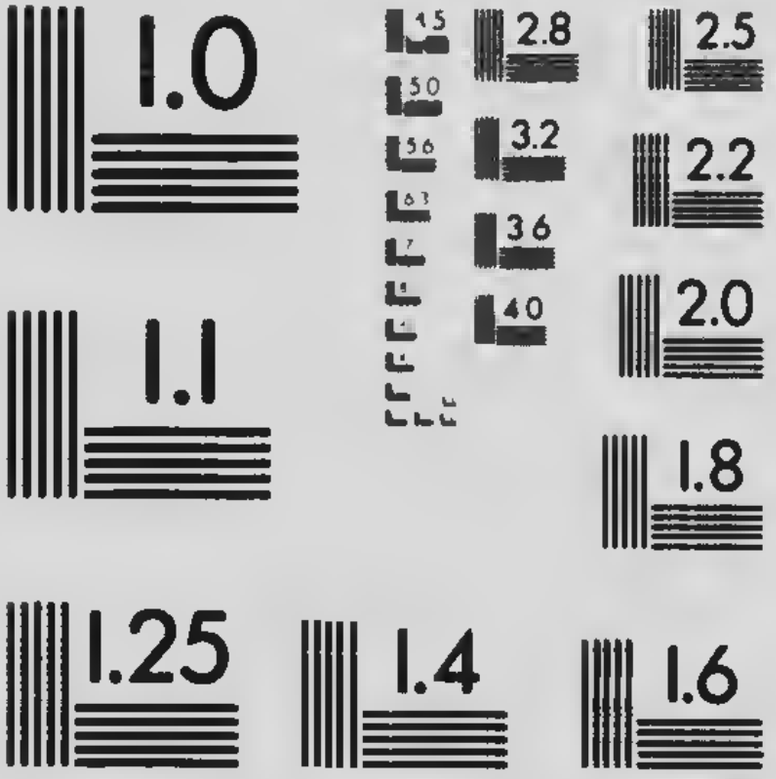

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A SHORT
PRACTICE OF MEDICINE

A SHORT PRACTICE OF MEDICINE

BY

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PREFACE TO SECOND EDITION

THE progress and important developments in the science of medicine made during the past five years have rendered necessary a second and revised edition of this book, and full advantage has been taken of the opportunity thus afforded of bringing the whole of the subject matter up to date. New paragraphs have been added on kala-azar, gastric neuroses, adenoids, suffocative catarrh of adults, the varieties of myelitis, diseases of the cauda equina, psychasthenia, and myositis—amongst others—while many of the descriptions in the first edition have been entirely recast.

Several new figures have been added and, where necessary, the original ones altered so as to bring them into accord with recently established facts. Although the book has necessarily increased in size, the original aims, in accordance with which the first edition was written, have been kept in view, and it is hoped that the extra pages will enhance the usefulness of the volume.

I am under a deep debt of gratitude to many authorities, whose experience and works I have freely consulted and utilised. It is one of the trials of writing a *short* text-book, that it is quite impossible to do justice by name to the authors from whom one has derived so much assistance.

To Mr. T. H. Graham my grateful thanks are due for revising the proof-sheets, and to Mr. J. Grieve for his painstaking care in reproducing the new illustrations.

I desire to take advantage of this opportunity of expressing my indebtedness to Messrs. J. & A. Churchill, the Publishers, and Messrs. R. & R. Clark, the Printers, for the uniform courtesy and help I have received at their hands, and to which the success of this book is in no small measure due.

ROBERT A. FLEMING

EDINBURGH, *January* 1912.

PREFACE TO THE ORIGINAL EDITION

The writing of a new book on the Practice of Medicine demands an apology, or at least an explanation. There are many excellent text-books, but there are few small-sized manuals which fill the place in the library which the present volume is intended to occupy.

Medical science has made such strides in the direction of specialism and along the lines of every system, that the time must come when students attending a class on general medicine will be armed with a text-book giving the general outline of each individual disease, and thus only special points, clinical cases in illustration, and hints on treatment will be left to be written down. Such a plan saves much pen-driving, and frees the teacher from dictating year in and year out statements which can be as well, if not better, mastered beforehand from a text-book.

Further, the student, burdened with innumerable classes, demands some guide as to what is necessary and what unnecessary for the all-important examinations which lie before him, and such a guide is offered in the following pages. For the busy practitioner it may also not be out of place to provide a volume which, if it does not exhaust the historical account and theories of every disease, contains a résumé of all that is most important.

No attempt has been made to group together clinical features in tabulated form, because such tables are more useful if drawn up by the reader himself, but a definite method has been followed, where possible, so as to aid the student's memory in mastering the clinical features of each disease.

It has been found impossible in the space at disposal to write, as originally intended, a brief account of the medical anatomy and physiology of each system, but figures have

been used to elucidate the difficulties of the structure and functions of the nervous system.

The methods of qualitative and quantitative testing of the normal and abnormal constituents of urine and stomach contents have been omitted, because they appertain more properly to the manual of clinical medicine, and for similar reasons pulse tracings with their diagnostic significance have not been described.

While the idea with which the book has been written is that everything should be mastered by the reader, the results of paralyses of spinal nerves have been given in detail for purposes of reference.

I am indebted to many writers from whose works hints and help have been obtained, and the experience gained during past years in the Edinburgh Royal Infirmary has not unnaturally coloured the descriptions of certain diseases.

I have also to thank, for friendly counsel and advice, several of my colleagues on the staff of the Edinburgh Royal Infirmary, with whom I have discussed numerous difficulties. In particular, I should mention Dr. J. J. Graham Brown and Major Marshall, whose kindly criticism of several special sections has been most helpful; and Mr. F. C. Nicholson, to whom I am indebted for revising the proof-sheets.

The charts and diagrams have been derived from various sources, but I wish specially to thank Professor Osler, who has permitted me to utilise several figures from his well-known text-book on the *Principles and Practice of Medicine*. Lastly, I desire to acknowledge the services of Mr. J. Grieve, who is responsible for the reproduction of the illustrations.

ROBERT A. FLEMING.

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SPECIFIC INFECTIOUS DISEASES

I. THE FEVER PROCESS

Fever or pyrexia implies chemical changes occurring in the body, by means of which the organism as a whole suffers more or less severely. The results of the fever process may be gathered from a glance at the clinical features tabulated under the different systems referred to later.

While we are specially studying the pyrexia of what are called the *continued fevers*, it is desirable to set down in tabular form the various causes of heightened temperature. It may be due, according to Adami, to -

(1) The infection of the individual with pathogenic organisms.

(2) The toxins which can cause pyrexia apart from the organisms which produce them.

(3) The products of a large internal haemorrhage which may cause pyrexia, although no decomposition changes be present in the effused blood. In part this is due to fibrin ferment.

(4) Sterile juices from tissues may also produce pyrexia. In this way, after a fracture or other exudation, temperature is produced.

(5) The action of certain drugs such as strychnine.

(6) The exposure to great heat.

(7) Nervous influences—reflex stimulation of the thermogenetic centre.

Fevers may be **classified** according to the **type** of pyrexia present.

1. *Continued fever* is pyrexia, in which the temperature remains for a certain number of days at a heightened level.

2. *Intermittent fever* is pyrexia in which the fall of temperature, generally occurring during the day, reaches the normal.

3. *Remittent fever* is pyrexia in which several rises of temperature occur, but the intervening fall does not reach normal.

4. *Relapsing fever* is a type of pyrexia in which periods of heightened temperature are succeeded by periods of apyrexia.

5. *Hectic fever* implies generally a nightly rise succeeded by a morning fall of the temperature to or about normal, and is usually associated with great sweating.

The normal skin temperature is stated as 98·4 or 98·6 F., but it is invariably higher in the mouth and rectum by half a degree to a degree. In old persons the temperature is slightly subnormal. The daily variations must be remembered. There is a trivial rise in the afternoon between 3 and 6 P.M., and a fall in the morning between 2 and 5 A.M.

There are heat-centres, probably situated in the medulla, which regulate in a remarkable way the temperature of the body. Heat is lost by the skin and lungs, and by an elaborate reflex mechanism the supply of blood to the skin may be increased or decreased by these heat-centres. There is not merely loss of heat from a dry skin, but sweating, with the resultant evaporation of water, causes very rapid loss of heat. To keep, therefore, a fair equilibrium, there are constrictor and dilator centres which govern the amount of blood sent to the skin and the secretion and excretion of sweat; and these centres are affected by the increased distribution of blood for special purposes to the stomach and other abdominal organs.

The mode of heat production has given rise to considerable differences of opinion, and naturally our explanation of the fever process is closely associated with the accepted theory. In the fever process is it the case that, according to Traube, (1) heat is generated to the same amount but not discharged; or (2) is there increased production, the discharge remaining the same; or (3) is there increased production far in excess of the increased discharge? Heat production is under the control of the nervous system, and the thermogenetic centres are probably situated, as already stated, in the medulla.

Experimental as well as clinical evidence has proved—(1) that in the febrile state there is increased metabolism altogether out of proportion to the food taken in and indi-

cating, therefore, an excess of breaking down processes. (2) that in fever, heat production exceeds heat discharge, and (3) that the balance between heat production and discharge is upset.

Clinical Features.—There is increased tissue waste, with defective excretion by the skin, kidneys, and bowels, and therefore a tendency to the retention and accumulation of waste products in the system.

Alimentary System.—The tongue is furred and dry, sordes are present in the mouth, there is marked loss of appetite, failure of the digestive processes, excessive thirst, and not infrequently vomiting. Constipation is the rule, although in certain fevers diarrhœa occurs. The liver is frequently swollen and painful.

Haemopoietic System.—The red blood corpuscles suffer both numerically and as regards their haemoglobin, while in some cases there is a leucocytosis and the spleen is acutely congested. In certain fevers the lymphatic glands are inflamed.

Circulatory System.—The heart's action is excited, the pulse full and bounding but also dicrotic, although in the late stages of many long-continued fevers degenerative changes occur in the myocardium, and thrombosis may occur in the veins.

Respiratory System.—The breathing is rapid, but agrees in ratio with the frequency of the heart's action, unless, as in certain cases, hypostatic congestion and other pulmonary complications or sequelae supervene, or the lung is specially involved as part of the fever process in question.

Integumentary System.—The skin is usually hot and dry, but in some types of fever, such as rheumatic fever, sweating is a special feature. Many fevers are characterised by definite rashes. In the late stages of any debilitating fever bed-sores are apt to occur.

Urinary System.—There is a diminished amount of urine, which deposits urates on standing, and is of high colour and specific gravity. There is often a deficiency of chlorides; and albumin, with tube-casts, may be present. Bladder difficulties, especially in connection with loss of consciousness, are common in severe cases of fever. Suppression of urine occasionally occurs.

Reproductive System.—Menorrhagia is frequent, and abortion is induced where the pyrexia is high, should pregnancy unfortunately complicate the case.

Nervous System.—Malaise is typical, and it includes headache and general discomfort, which in great measure is referable to this system. Sleeplessness, convulsions, delirium, coma vigil, and coma are features of severe types of fevers: while hyperpyrexia, by which is meant a temperature exceeding 106° F., and associated with delirium, may be included under nervous phenomena.

Locomotory System.—The muscles rapidly waste, and there is a stoppage of growth in the more serious fevers.

Stages of Fever.—Most of the eruptive fevers pass through definite stages, which include:—

1. The stage of *Incubation*, during which there are no symptoms.

2. The stage of *Invasion*, during which the first clinical features appear, including malaise, and generally more or less pyrexia.

3. The stage of *Advance* or *Eruption* is characterised by the appearance of the rash, where a rash is present, and the clinical features referable to the fever process are at their height.

4. The stage of *Defervescence* or *Resolution*, during which the fever diminishes either suddenly by *crisis* or gradually by *lysis*.

5. The stage of *Convalescence*, during which, in a favourable case, complete recovery occurs.

Complications or *sequelæ* may delay a favourable issue. In severe or malignant types of fever, death may occur at almost any stage after incubation is over. It sometimes happens that a malignant eruptive fever kills the patient before any eruption has appeared.

General Treatment of the Fever Process.—It is prudent to begin the treatment of a continued fever by careful attention to the alimentary canal, and a purge is therefore advantageous. Relief may also be obtained by stimulating the skin and kidneys to act freely. A hot bath and a dose of spiritus etheris nitrosi render the patient much more comfortable. The condition of the mouth and teeth should be considered from the outset and attended to daily. Boro-

glycerine forms an admirable antiseptic, and it should not be forgotten that the appetite is aided by a clean mouth, and digestion is at least not interfered with more than it is inevitably by the fever process.

The diet in fever is most important. As a general rule milk forms the chief part of a typical fever dietary, and if milk cannot be taken, beef-tea and soups of a simple nature may take its place. In not a few cases alcohol is a necessity, but it should rarely be given until other measures have first been tried. It will support life, but its administration should be deferred, if possible, until the more acute stage of the fever is passed. In alcoholic subjects its use is obviously objectionable.

The room selected for the fever patient should be light and airy, and where it is an infectious disease, all unnecessary furniture, hangings, and carpets should be removed. Insist on having a hair mattress, or, where necessary, a water-bed, but on no account permit a feather-bed to be used. In the more severe cases of a continued fever it is a great comfort to the patient to have two beds of similar height in the sick-room, so that he can be moved from one to the other occasionally. The bed should be placed in such a position that free access can be obtained to it. The utmost care should be taken to have sheets and blankets absolutely clean; and where the excreta are infective, antiseptic precautions must be taken so as to prevent any danger to others, and especially to the nurses and attendants. Carbolic acid, chloride of lime, and many other powerful antiseptics can be used for sterilising stools, etc. A good nurse is even more important than a carefully appointed sick-room; and, when desirable, night and day nurses should be procured. A most important point is to insist on the nurse carefully noting the amount of food given and the hours at which it has been administered; and, similarly, medicines and stimulants should be charted in an orderly and exact fashion.

It is hardly requisite to describe here the treatment of all the different symptoms of an ordinary fever, but it is desirable to refer in detail to the method of treating hyperpyrexia. Every fever patient should, if possible, have the skin sponged at least twice a day, as the temperature, even when tepid water is used, is considerably reduced by this procedure.

The following methods, in order of increasing severity, may be employed for the treatment of hyperpyrexia:—

1. Sponging the patient with tepid water or eau-de-cologne and water.

2. Sponging the patient with cold water or iced water, taking care to dry each part of the body that has been sponged before proceeding to the next part.

3. Wrapping the patient in a sheet or blanket wrung out of cold or even iced water. For this procedure the use of two beds is almost imperative. A waterproof sheet is spread on one bed, a dry blanket is placed on it, then the sheet or blanket wrung out of cold or iced water, in which the patient is wrapped, and blankets placed on the top. At intervals the temperature should be taken in the mouth so as to avoid too great reduction, and on no consideration should the temperature be brought down to normal. Before removing the patient to a dry bed care must be taken to prevent collapse by giving a stimulant when necessary.

4. The patient may be placed in a bath, the temperature of which should be about 80 Fahrenheit, and then by adding cold water or iced water it can be rapidly reduced to 50 or 60 Fahrenheit. A more severe method of treatment is to pour cold or iced water over the patient's head and back, whilst in the bath. Generally fifteen to thirty minutes is the maximum of the patient's endurance, and every precaution should be taken to prevent collapse, as already mentioned.

Many patients owe their lives to treatment on one or other of these lines, and the cold-bath or hydropathic treatment of typhoid fever has proved most successful, although from a patient's point of view it may be a great hardship.

No drugs, which can be wisely given, do much for the reduction of temperature, and on no consideration should antipyrine, antifebrin, or other antipyretic remedies of the group be administered for this object, because of their depressing effect on the heart, which the hydropathic treatment most certainly obviates. Perhaps the only drug which is an antipyretic, and which it is prudent to administer, is quinine in doses of 10 to 15 grains at bedtime, although its use is limited to certain fevers only.

Dr. Claude Ker considers that there is no advantage in checking a moderately high temperature, and holds the

opinion that the prognosis of serious fever cases is often more favourable when the pyrexia is in keeping with the severity of the disease. Needless interference with the temperature, whether by hydropathic or medicinal measures, is certainly to be condemned.

In any long-continued fever bed-sores are apt to occur. They may be guarded against by attention to cleanliness and by frequently washing the skin with soap and water. Hardening the skin with methylated spirit or whisky rubbed into and allowed to dry on the part is very useful, and a dusting powder of zinc oxide or boracic acid and starch may be freely employed.

Prophylaxis.—The day of the sulphur candle seems past, and yet sulphurous acid gas has proved a satisfactory disinfectant for many years. Clothes, blankets, etc., should be disinfected by means of steam where this is possible, and formaline spray (4 oz. to the gallon) can be used for the floors and walls of infected rooms.

II. TYPHOID FEVER

(Enteric Fever; Typhus Abdominalis)

An infectious fever characterised by a special involvement of Peyer's patches and solitary glands in the intestine, with usually the appearance of a limited rose-coloured rash, enlargement of the spleen and mesenteric glands, and, in most cases, a typical temperature chart.

Etiology.—The Typhoid bacillus of Eberth measures 3 to 4 μ in length by 1 μ in breadth, is flagellated and actively motile, and is found in the stools, the urine (25 to 30 per cent of cases), the blood, the spleen, and, it is said, in the spots. Less commonly it occurs in endocarditic vegetations, pleural and other exudates, and in meningeal effusions. It stains with aniline (basic) dyes, and in one of the rounded ends there is a refractile body which was thought, at one time, to be a spore.

Typhoid bacilli manufacture a toxin, but it does not appear to be separable from the bodies of the bacilli. As a result, however, of the presence of the typhoid bacillus, the body manufactures an antitoxin which interferes with the

motility of the bacilli. Widal's reaction consists in the action of the blood of a patient suffering from typhoid upon a culture of the typhoid bacillus in bouillon of twenty-four hours' growth, and it should produce rapid clumping of the organisms with cessation of movement. A dilution of 1 part of the patient's blood to 30 of sterile bouillon may be taken as the minimum standard, and the period of time within which this change should occur ought not to exceed sixty minutes. A typhoid patient gives a typical reaction after one week of the disease.

The disease is commoner before middle life. It is specially frequent in hot, dry weather, and is the direct result of the typhoid bacilli infecting the water-supply, milk, food, particularly oysters, vegetables which are eaten uncooked (such as lettuce), and ice, in which the bacilli for a long time retain their vitality.

Persons in perfect health, but who have had typhoid fever, may continue to pass typhoid bacilli for months or years, and such cases unquestionably explain the sudden appearance of typhoid fever without any history of previous case in the neighbourhood.

We are still uncertain as to the limit of time during which the bacilli remain active. The following figures are interesting: in garden earth 21 days, in filter-sand 82 days, in street dust 30 days, and on clothing 60 to 70 days. In the South African War many typhoid cases were unquestionably the result of inoculation of food by flies. It is extremely improbable that the organisms are transmissible by air.

The stools of a typhoid patient almost certainly increase in virulence during the process of decomposition.

Pathological Anatomy.—The intestinal lesions are the most typical.

During the *first week* Peyer's patches and the solitary glands of the lower part of the small intestine, and probably at first close to the ileo-cæcal valve, undergo enlargement and become of a pink or fawn-coloured appearance. There is not merely hyperæmia, but there is a rapid increase of lymphoid cells.

At the beginning of the *second week*, if resolution does not commence (an abortive attack), necrotic changes occur.

During the *third week* ulceration of these necrosed patches

commences, although it is most probable that even during the preceding week this process may have begun.

The ulcers are bile-stained, do not necessarily involve the whole of a Peyer's patch, and the depth varies in different cases. Sometimes the floor of the ulcer is the muscular coat, in more severe cases the peritoneum, with naturally an increased risk of perforation. The edges of the ulcer are usually swollen and often undermined. The base of the ulcer is smooth and clean. As already indicated, the older ulcers are to be found in the lower parts of the ileum.

Towards the end of the third week cicatrisation occurs, and, depending upon the amount of destruction, there may be more or less constriction of the bowel. The mesenteric glands are always inflamed, of reddish colour, and in certain cases they may suppurate and so cause peritonitis. The large intestine is not so frequently or so markedly affected, but the caecum naturally suffers to a greater extent than the colon. The jejunum may be involved right up to the duodenum, but the process is much less extensive than in the ileum.

The possible results of ulceration may be perforation, severe haemorrhage, and in rare cases stricture from cicatrisation.

The spleen is almost invariably enlarged and it is acutely congested, and occasionally infarction occurs. The liver and kidneys show cloudy swelling, and hypostatic congestion of the lungs occurs before death in fatal cases. In certain cases the bacillus attacks the endocardium or pericardium, the pleura, the cerebral meninges, and other parts, considerably modifying the clinical features from the usual type, and there are rare cases of typhoid fever in which there are no intestinal lesions, but in which the lung may be the primary site of infection.

Clinical Features.—The stage of *Incubation* is one to two weeks. The premonitory symptoms consist in lassitude, headache, nausea, epistaxis, bronchitis, sometimes abdominal pain, or even a rigor.

During the *first week* the temperature steadily rises until it reaches 103 or 104 F. Each evening the record is higher than the one preceding. The pulse is fairly rapid, full, and of low tension; but its frequency does not coincide with the temperature. There is generally constipation, the tongue being white and coated, and about the end of the first week the rash may appear. The rash, consisting of rose-

coloured spots, comes out first on the abdomen, and usually the spots number from six to twenty at a time. They are slightly raised, but disappear on pressure. They are, as a rule, confined to the trunk, and in about one-fifth to one-quarter of all cases they are absent altogether. The spleen becomes enlarged about the same time, and there is usually nocturnal delirium.

At the beginning of the *second week*, during which necrosis is occurring in the Peyer's patches, the tongue is dry, especially in the centre, and may be covered with sordes; the face is pale, with red, flushed cheeks; and although the patient is dull and listless, the eyes are brighter than in typhus. Diarrhœa is not infrequent, and if it has appeared already, it now

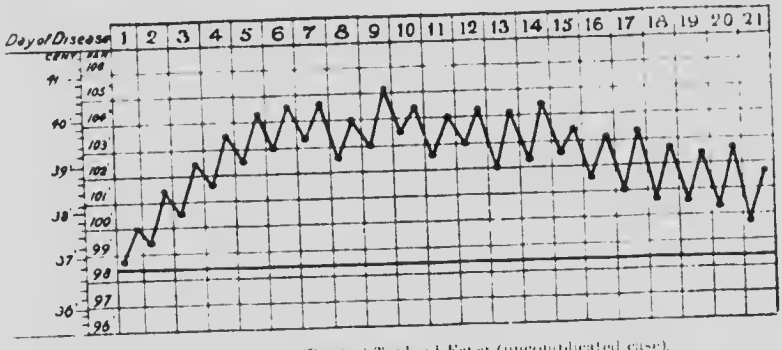


Fig. 1. Temperature Chart of Typhoid Fever (uncomplicated case).

becomes much more severe, with abdominal distension and tenderness. The stools are pea-soup-like, alkaline, have a horrible smell, and contain much débris, altered blood, and, of course, many bacilli. There are certainly cases of typhoid in which diarrhœa is entirely absent, but should necrotic changes occur in the patches, it is usual to find the typical stools.

Death is not uncommon during this week, but haemorrhage and perforation are likely to be met with during the stage of sloughing, which more probably occurs during the following week.

During the *third week* the clinical features depend on the amount of ulceration present. There may be much diarrhœa, great meteorism and gurgling in the right iliac region, but in a favourable case the temperature should have begun to fall, and the physician may be only called upon to combat the progressive weakness following a protracted, continued fever.

Great abdominal distension commonly precedes either haemorrhage or perforation.

Haemorrhage occurs in 3 to 5 per cent of all cases, and may be said to be the cause of death in about one-twentieth of those proving fatal. It is generally preceded by a feeling of faintness; a marked fall of temperature and death may result even before the blood has appeared in the stools.

Perforation occurs in from 2 to 3 per cent of all cases, and is the cause of death in 25 to 35 per cent of those which are fatal. Perforation causes abdominal pain which may be localised, more or less board-like rigidity of the abdominal wall, vomiting, disappearance of the liver dulness in the right nipple line, and all the other typical signs of peritonitis. A sudden fall in temperature often coincides with the collapse which indicates that perforation has occurred.

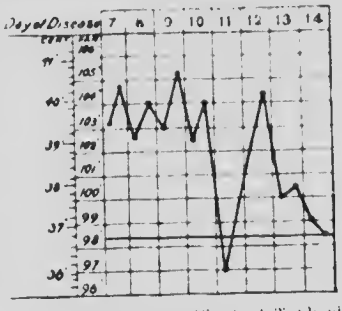


FIG. 2. Temperature Chart of Typhoid Fever showing sudden fall of temperature due to a severe haemorrhage.

Abdominal pain is present as a typical feature of the disease, but it may become specially severe as the result of great meteorism, inflammation of the mesenteric glands, and, of course peritonitis.

In the *Alimentary System* there is frequently a considerable amount of nausea and vomiting, and sometimes inflammation of the tonsils. Cholecystitis, due to infection of the gall-bladder, is by no means uncommon, and pain over that organ is a suggestive clinical feature.

In the *Haemopoietic System* there is practically never a leucocytosis in uncomplicated cases, and sometimes there may be a leucopenia (2000 to 4000 per c.mm.), but the red blood corpuscles and haemoglobin are diminished. The coagulability of the blood is diminished in the early stages, but much increased during convalescence.

In the *Circulatory System* the first sound of the heart is almost invariably feeble, and the pulse full, soft, and dicrotic, is proportionately very slow taking into account the temperature. In a recent case seen, the pulse did not exceed 80 although the temperature was 102·5 F. The feeble action

of the heart is due in part to the prolonged pyrexia and in part to the long-continued effect of the typhoid toxin.

In the *Respiratory System* there may be several complications, such as pleurisy, lobar pneumonia, and hypostatic congestion, while bronchitis and epistaxis have been already noted as among the early symptoms.

In the *Integumentary System* there are occasionally other rashes present, generally of an erythematous type, while a cadaveric odour is certainly not uncommon in cases which are about to terminate fatally. Profuse sweating occurs once the fever has begun to abate.

In the *Urinary System* the presence of albumin is almost constant, associated probably with the excretion of both toxins and organisms by the kidney. From the 6th to the 14th day Ehrlich's diazo-reaction may be obtained (see page 15).

In the *Nervous System* headache and slight nocturnal delirium, and not infrequently deafness, are the only symptoms in a mild case of typhoid fever. In severe cases there is a type of the fever to which the term *nervous* is applied. In it there are coma vigil, constant muttering delirium, subsultus tendinum, and plucking at the bedclothes, often terminating in profound coma and death. Hyperpyrexia, nearly always associated with marked delirium, is a grave danger in this type of typhoid fever. Peripheral neuritis, sometimes localised neuritis, or pain in connection with the muscles, may be associated with the disease, and more rarely a limited myelitis or other lesion of the cerebro-spinal system occurs.

During the *fourth week*, in a mild case, convalescence begins, and after a varying period of time the patient completely recovers. It is important to remember that the keen appetite of the patient should not be gratified, otherwise a favourable case may be suddenly rendered most unfavourable by the increase of ulceration in the intestine, and very probably perforation or haemorrhage.

It must not be forgotten that many cases do not run a typical course: resolution may commence in the Peyer's patches without any necrosis occurring: in many cases profound changes may develop in the intestine without giving rise to the usual clinical features, and serious relapses are by no means uncommon even in a mild case, although generally as the result of

a dietetic error. We must also remember that besides the local action of the bacilli on certain tissues, there may be a pronounced and very generalised action of the toxins on the body as a whole, and a typhoid septicæmia is by no means rare.

Perhaps one of the most serious developments is *relapse*, which may set in at almost any period of the disease, and after one, two, or three weeks of apyrexia. The temperature may once more rise with a return of the typical intestinal and other symptoms. It should also be remembered, however, that some trivial error in diet may cause a slight and evanescent rise of temperature. The more serious relapses are almost certainly the result of re-infection, either of the same or of fresh Peyer's patches.

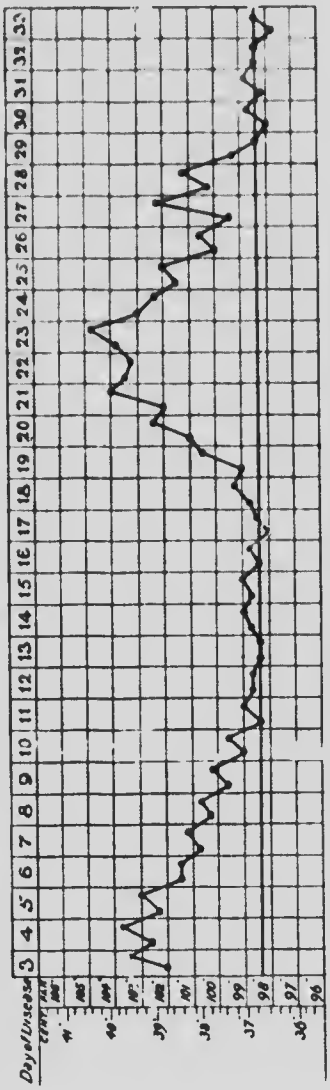


Fig. . Temperature Chart of a case of Typhoid Fever with relapse about 24th day.

Types. In a disease with such varied and complex groupings of clinical features many types may be described. Of these the most important are: —

(1) *A mild abortive type*, in which probably resolution occurs in or soon after the first week. The rash may be present, but the temperature falls with profuse sweating, and restoration to health speedily follows.

(2) *The ordinary type* of typhoid fever already described.

(3) *A gastro-intestinal type*, in which vomiting and diarrhœa are specially marked.

(4) *A malignant type*, in which, as a rule, the nervous symptoms are severe, or where death occurs prior to the definite development of the disease. So-called hæmorrhagic typhoid,

by which is meant haemorrhages from mucous surfaces, is a rare form.

(5) *The ambulatory* or *latent type* is perhaps one of the most serious forms, because the patient risks his life by carrying on his daily work in ignorance of the disease from which he is suffering, and may, by a dietetic error or over-exertion, readily cause the development of grave symptoms, such as perforation or haemorrhage.

(6) *Typhoid septicaemia* is the type already referred to, in which there is a general infection, often with no typical intestinal symptoms.

Complications.—The chief complications of the *Alimentary System* have already been described.

In the *Circulatory System* it should be kept in mind that the heart frequently is affected by the prolonged fever process. During the stage of convalescence thrombosis of the femoral veins, causing "white leg," may occur: the risk of separation of a portion of the clot must in such cases be guarded against.

In the *Respiratory System* lobar pneumonia, pleurisy, oedema of the lungs, and hypostatic congestion are of frequent occurrence.

In the *Integumentary System* the likelihood of bed-sores, especially in asthenic patients, should be kept in mind, and crops of boils sometimes annoy the patient during convalescence.

In the *Urinary System* there is a risk of acute nephritis, while retention of the urine in an unconscious patient may produce cystitis.

In the *Nervous System* most of the more important complications have already been mentioned, but the possibility of "typhoid spine," which is probably largely functional, and of inflammation of the eye or ear, although rare, should not be forgotten.

Diagnosis. Perhaps no fever presented such difficulties of diagnosis prior to the introduction of the test devised by Widal in 1896.

1. *The Widal Reaction.*—A culture of typhoid bacilli on agar of less than twenty-four hours' age is added to a drop of sterilised beef bouillon. A little blood is taken from the finger or ear of the patient, and the serum diluted thirty times with bouillon. A drop of the diluted blood-serum is added to a drop of the typhoid culture, and examined under the micro-

scope. In the event of the case being one of typhoid the bacilli soon cease to move actively, run into clumps (which is called agglutination), and this should occur certainly within one hour.[1] The reaction should be present after the end of the first week, and only fails in a small percentage of cases of typhoid. A similar reaction can be obtained in cholera, plague, glanders, and some other fevers by using a culture of the respective organisms in the same way.

2. *Isolation of Bacillus.*—In addition to the Widal test, we may endeavour to isolate the bacillus of Eberth from the stools, urine, or, best of all, from the spleen; but in many cases, notwithstanding the utmost care, the result is unsatisfactory.

3. *The Diazo-Reaction of the Urine*, described first by Ehrlich, has been largely replaced by Widal's serum-reaction. The test consists in the following: Two solutions are prepared, (1) a saturated solution of sulphanilic acid in 5 per cent hydrochloric acid in water, and (2) a freshly prepared $\frac{1}{2}$ per cent solution of sodium nitrite in water. To a few c.c. of the urine in a test-tube add equal parts of solution (1), and a few drops of solution (2). Shake the test-tube, and add enough ammonia to render the mixture alkaline. When sulphanilic acid is acted on by nitrous acid, a compound is formed which unites with aromatic compounds found in certain urines, and amongst others the urine of a typhoid patient, and it produces a brownish-red colour, the froth being specially tinged. The test, although commonly positive in typhoid fever from the 6th to the 14th day, is also present in cases of measles, in rapid tuberculosis, and in some other conditions.

These tests, together with the typical temperature chart, the presence of the rash, should it occur, and the enlargement of the spleen, are generally sufficient to prevent mistake; but the physician is more apt to consider his patient to be suffering from such diseases as *tubercular meningitis, acute miliary tuberculosis, lobar pneumonia, catarrhal pneumonia*, or *influenza*, and to ignore the possibility of typhoid altogether. It is a wise precaution to have the Widal reaction carried out in all doubtful cases, and more especially if the patient presents symptoms not in keeping with the original diagnosis. We

[1] The agglutination test can also be applied by a quick drop method, suspensions of dead typhoid bacilli being used with blood-serum from the patient.

have seen several cases diagnosed as catarrhal pneumonia or capillary bronchitis which turned out to be typhoid.

Prognosis.—The death-rate is rarely over 20 per cent, and generally only about 10 per cent; but the age, constitution, and habits of the patient greatly affect the chances of a satisfactory result, while it must be remembered that epidemics vary in their severity. Unfavourable conditions, such as excessive meteorism, haemorrhage, perforation, or marked nervous symptoms, should make the physician cautious, while older patients (after middle life), alcoholics, very debilitated subjects, and extremely stout persons run a much greater risk.

Treatment. *General.*—The patient should be placed in a well-ventilated sick-room, and the most absolute cleanliness is requisite, especially for the sake of those in attendance. A careful note of every little detail is of great value, such as a three- or four-hourly temperature chart: the times of feeding the patient, with the amount taken: the number of hours of sleep: the quantity of urine; and the number of stools passed. From the very commencement the condition of the skin should be thought of. Cleanliness is essential, and the local use of a non-irritating antiseptic, such as boracic acid, will be found of much value, while a water-bed is a great comfort in some instances. On no account should the patient be allowed to sit up for the evacuation of bowels or bladder, and in severe cases order cotton-waste to be used in place of the bed-pan. The patient should be kept absolutely at rest, free from excitement, and encouraged to sleep as much as possible. The condition of the mouth should receive attention at stated intervals. Especially where the patient is semi-conscious the mouth should be swabbed out with the glycerine of boracic acid every few hours, and in all cases the mouth should be washed out or swabbed with this or a similar antiseptic once or twice daily.

Diet.—There is much difference of opinion as to whether we should order milk, perhaps peptonised, with egg-flip and similar easily digested and nourishing foods, or whether the patient should be starved, strength being merely kept up by a little iced alcohol and water with the minimum of other nourishment. The principle governing the dietetic régime is to have as little waste matter reaching the inflamed portion of the intestine as possible, and probably a middle course is advisable,

For an adult 2 to 3½ oz. of milk may be given every two hours, and a pint of beef or chicken tea may be given in three equal portions at suitable times in the twenty-four hours, so as to break the monotony of the milk dietary. In most cases the patient should be encouraged to drink water freely. During convalescence the dietary should be most cautiously increased, and, as indicated, a careful watch kept on the stools, so as to detect any evidence of overfeeding.

Special Symptoms.—*Pyrexia and Hyperpyrexia.*—Many physicians, and especially Germans and Americans, have used the bath system of treatment with great benefit. This consists in immersing the patient in water about 70 to 75 Fahrenheit, and either pouring cold water over him or applying cold-water compresses to the chest and abdomen. The patient remains in the bath about fifteen minutes, during which he usually becomes extremely blue and shivers with the cold. The baths are repeated every two or three hours, if necessary. As many as 200 baths have been given to a patient during an attack of typhoid fever. After each bath the patient is rubbed dry, and, if somewhat collapsed, a little alcohol is administered. By this means continued pyrexia is prevented, and the death-rate in serious cases has certainly been diminished.

For hyperpyrexia similar treatment may be adopted, or one or other of the methods described in the treatment of fevers generally (p. 6) may be preferred.

Diarrhœa.—Where diarrhœa is profuse and tending to be excessive, the dietary of the patient should be carefully revised, and either bismuth subnitrate (gr. 30-40), with Dover's powder (gr. 5-15), or small doses of lead acetate gr. 2 given at frequent intervals. A starch and opium enema is often invaluable, and morphia suppositories (gr. ½-1) may be administered with great benefit. Occasionally an alteration of diet, and especially the substitution of boiled milk, relieves the patient.

Meteorism and abdominal pain are often treated by the application of either hot fomentations or cold compresses over the abdomen, and sometimes by the use of such counter-irritants as turpentine stupes. Tympanitis is frequently prevented by the administration of intestinal antiseptics, such as salol (gr. 20), wood charcoal (gr. 30-60), or β-naphthol. An enema containing turpentine or asafœtida is often of

service, and in very severe cases, where there is paralysis of the bowel, the introduction of a long, flexible rubber tube has given much-needed relief by drawing off some of the gas.

Hæmorrhage from the bowel is very frequently the result of excessive meteorism. Absolute rest should be enjoined, the quantity of food diminished and replaced by pieces of ice to suck, while full doses of acetate of lead with opium or 10 minim capsules of turpentine should be promptly administered. An ice-bag should be applied to the abdomen; very generally alcohol or other stimulant may have to be administered owing to collapse.

Perforation usually results from one or possibly more ulcers, and an attempt should invariably be made to treat the condition surgically.

Peritonitis is relieved by full doses of morphia administered hypodermically, and by the application of either warm poultices and fomentations, or an ice-bag. In some cases operative interference may yield a faint hope of relief.

Heart failure should be prevented by careful attention to the heart-sounds, and the early use of digitalis, strychnine, and certainly diffusible stimulants.

Bed-sores can usually be obviated by the attention to cleanliness previously recommended, while a water-bed and the process of hardening the skin referred to on p. 7 are of special value in a long-continued fever like typhoid.

Headache is sometimes so severe that it may call for special treatment, when the use of the ice-bag or the internal administration of phenacetin in 10-grain doses every two hours generally proves sufficient. The patient should be kept absolutely quiet, and the room darkened.

Insomnia is troublesome in many fevers, and especially in typhoid. Sulphonal (gr. 15-30), chloralamide (gr. 15-30), paraldehyde (♏ 60-120) are amongst the best hypnotics for a case of the kind.

Delirium is frequently associated with hyperpyrexia, although it may indicate that some grave nervous, respiratory, or other complication is present. Ice-bags applied to the head and the treatment for hyperpyrexia are invaluable.

Recently *Typhoid Vaccine* has been more systematically tried and apparently with some success. It is recommended

that the vaccine should be "autogenous," *i.e.* obtained from the patient's own typhoid bacilli, and that the treatment should be begun early.

Prophylaxis.—It is hardly necessary to refer to the importance of pure and uncontaminated drinking water, nor is it requisite to do more than mention the risks of sewage getting into wells. The stools, probably also the urine, and most assuredly the sheets from the patient's bed, should be disinfected, either by using carbolic acid of a strength of 1 in 20 parts, or corrosive sublimate of 1 in 1000, and the greatest attention and care should be paid by the nurse to the careful disinfection of her hands, rubber gloves being recommended when handling soiled sheets, etc.

Inoculation against Typhoid.—Wright's vaccine is a fluid containing dead typhoid bacilli, and the specific toxin which has been elaborated in the course of cultivation. About 1000 million dead typhoid bacilli constitute the first dose, and ten days later 2000 million are injected. It usually causes considerable discomfort to the patient, both malaise and heightened temperature being present for several days. The chance of taking typhoid fever is much reduced, and the death-rate appears to be lower. After inoculation the patient's blood agglutinates typhoid bacilli, so rendering the Widal reaction useless as a test for typhoid fever.

Paratyphoid

This remarkable fever closely resembles typhoid, but is apparently due to a distinct organism not very dissimilar to the colon bacillus. The fever process is like a mild attack of true typhoid with splenic enlargement, a rose-coloured rash, and evidences of intestinal involvement. The chief points of difference are—(1) that the essential lesion of typhoid in the Peyer's patches and solitary glands in the intestine is generally absent; (2) that a pathological condition more nearly resembling dysentery may be present; and (3) that the typical Widal's reaction is either not obtained or is doubtful. The only certain test is to cultivate the organism from the blood.

III. TYPHUS FEVER

(Spotted Fever)

An acute infectious fever occurring in epidemics, and due to a micro-organism associated with dirt and insanitary conditions of life.

Etiology.—Several organisms have been described, and amongst others a diplococcus was isolated by Balfour and Porter during the last severe epidemic in Edinburgh. Dirt, overcrowding, starvation, and probably fatigue of mind and body, are all predisposing factors. Improved sanitary arrangements have tended to stamp out this very fatal fever. It may be transmitted by contact and by the clothes of infected persons.

Pathological Anatomy.—The spleen is invariably congested and enlarged. The blood is dark, but there is no distinctive pathological lesion.

Clinical Features.—The stage of *Incubation* is generally about twelve days, although sometimes it may be much shorter. The stage of *Invasion* usually commences suddenly with a marked rigor, the temperature rapidly rises, the pulse is full, the tongue furred and dry, vomiting is common, and there is frequently pain in the back, limbs, and head. The face is flushed, the conjunctivae are injected, and there may be mild delirium at night.

The stage of *Eruption* appears from the 3rd to the 5th day. The rash comes out first on the trunk, on the backs of the hands and elbows, and later, and to a less extent, on the face. It is a dusky, mottled eruption, often described as of mulberry appearance, and at an early period little petechial haemorrhages develop in certain of the spots. The mulberry appearance is due to the fact that while some of the spots are superficial others are deeper seated, and these latter give rise to the subcuticular mottling which is so characteristic. The temperature remains high, from 103 to 105° F., the tongue becomes brown and dry, the urine is scanty, with a heavy uratic deposit and often a little albumin; the apathetic appearance of the patient is very typical. There is almost always a certain amount of delirium, especially at night, and dulness of hearing, which necessitates loud speaking when

attempting to communicate with the patient. In a severe case the face becomes dusky, and coma vigil and subsultus tendinum supervene.

There is a distinctive odour about the body of a typhus patient, which is certainly very unpleasant and is said to be of diagnostic value.

The termination by *crisis* generally begins before the end of the second week, the temperature usually falling somewhat slowly to normal within 2 or 3 days, and the patient at once becomes brighter and less apathetic. In fatal cases death may be due to excessive prostration or, in other words, the develop-

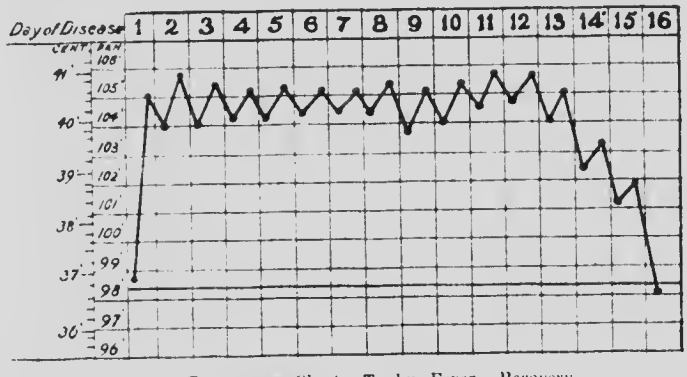


FIG. 4.—Temperature Chart. Typhus Fever. Recovery.

ment of the typhoid state, to toxaemia or to hyperpyrexia with wild delirium.

Complications.—Perhaps the most fatal complications are heart failure, broncho-pneumonia, gastro-intestinal catarrh, and Bright's disease. Occasionally phlebitis, sometimes paralysis (hemiplegia or peripheral neuritis), and even insanity, follow this fever.

It is hardly necessary to state that a malignant type of typhus is common.

Diagnosis.—The petechial rash, the delirium, and the continued high temperature rarely make a mistake possible. *Purpura* is not as a rule associated with an elevated temperature, while *flea-bites* show a marked puncture which can be easily seen with a magnifying-glass. In *measles* it is rare to find a haemorrhagic rash, and in *uraemia* there should be no rash at all. It is in isolated cases that the physician's skill is likely to be taxed.

Prognosis. -The death-rate varies considerably. The older the patient the higher it becomes. According to Dr. Claude Ker it is 2 per cent between 10 and 15 years, 30 per cent between 35 and 40 years, and 80 per cent in persons over 70. Alcoholism, the appearance of marked nervous symptoms, and pulmonary or cardiac complications render the prognosis grave.

Treatment. -The patient should be isolated along with those in attendance on him. Attention should be paid to the bladder, and as a rule the patient's skin should be sponged twice a day with some agreeable antiseptic lotion. The food is most important: a generous though simple diet must be ordered, and nourishment administered every two or three hours. The patient should be encouraged and, if necessary, constrained to drink freely of cold water. Paraldehyde (3 2 for adult , chloral, or the bromides should be given for delirium, the bowels should be attended to, and the headache relieved by the administration of caffeine citrate (gr. 5-10), phenacetin, or similar remedy.

The PROPHYLAXIS of the disease is of enormous importance. Sanitary improvements, the prevention of overcrowding, and the careful separation of infected cases, with subsequent disinfection of the houses from which the patients have come, have done much to eradicate the disease from our large towns.

IV. SCARLET FEVER

SCARLATINA.

AN acute infectious fever characterised by a brief stage of invasion, with sore throat, high temperature, and a typical rash.

Etiology. —It occurs generally in epidemics, often in late autumn, but sporadic cases frequently crop up where epidemics have previously existed, and they are due to the tenacity of life of the micro-organism. It is most common in childhood, rare in infancy, but adults do not escape. Females appear to suffer more often than males. Desquamation is probably the period of greatest infection, the scales of skin containing the organism, but the nasal and salivary secretions are also infective. Milk is often the medium of infection.

Klein has isolated a streptococcus from the blood of scarlet fever patients, which he believes to be the specific organism. It grows rapidly on gelatine and other media, forming whitish colonies, and it seems highly probable that this organism is the genuine treptococcus of scarlet fever. Other observers have isolated a similar organism. The streptococcus of scarlet fever is not, however, the only organism found in cases of scarlet fever. Where there is marked throat ulceration other streptococci are also present and in particular the Streptococcus pyogenes. This is a secondary infection superadded to the primary scarlet fever organism.

Pathological Anatomy.—It is remarkable how rapidly the swelling of the skin disappears after death. There are no

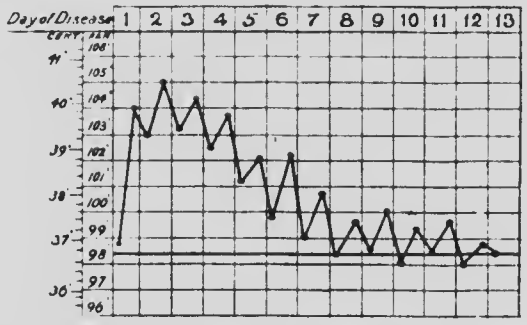


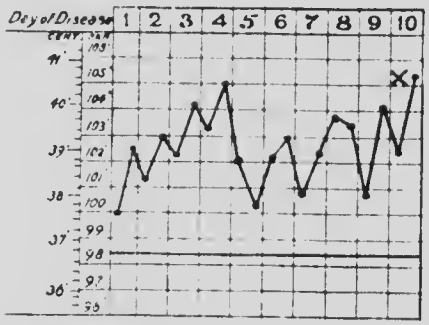
FIG. 3. Temperature Chart. Scarlet Fever. Case terminating favorably

constant changes, but the throat inflammation with tonsillar suppuration is usually present, with sometimes acute inflammatory oedema of the neck, acute suppuration of the neighbouring lymphatic glands, or occasionally a burrowing cellulitis. The spleen is often enlarged and soft. There may be endocarditis and pericarditis, and not infrequently there is evidence of pulmonary complications, such as broncho-pneumonia and hypostatic congestion, while in not a few cases acute nephritis is well marked at death. In the nervous system perforation of the tympanic membrane, the result of inflammation of the middle ear, is extremely common, and may lead to mastoid abscess, septic meningitis, and sometimes abscess in the brain. In the locomotory system the joints are often enlarged and may suppurate.

Clinical Features.—The stage of *Incubation* is 1 to 7 or 8 days, with no symptoms.

The stage of *Invasion* is rarely more than a day, and often only twelve hours, with malaise, vomiting, headache, sore throat, general'y a quick rise of temperature (102 -104 F.) and very rapid pulse, and in children it frequently sets in with a rigor. On examination the throat is found to be red, the tonsils and fauces being swollen and painful.

The stage of *Eruption.* Generally on the second day scattered red punctiform spots appear, first on the neck and chest, and then rapidly spread over the body. The chin, an area round the mouth, and the scalp usually escape. The whole skin is early hyperaemic, and later bright scarlet. Sometimes there are small papular elevations, occasionally sudamina, and in malignant cases petechial haemorrhages. The skin, especially in the neighbourhood of the joints, is so markedly swollen as to feel stiff and make the bending of the fingers difficult. There is comparatively little rash on the face although it is flushed. Pressure with the finger leaves a white mark for some seconds on the skin. A careful examination of the soft palate often demonstrates punctiform spots, which may precede the rash on the body.



Temperature Chart. Scarlet Fever. Throat complications with death on 10th day.

The tongue is at first covered with a whitish fur through which on the 2nd day the hyperaemic fungiform papillae shine like bright red spots dotted over its surface. This is the white strawberry tongue. When the fur is shed (3rd or 4th days), and the tongue becomes bright red with the fungiform papillae still showing prominently, it forms the red strawberry tongue. For a long time the patient's tongue remains typical.

During the stage of eruption the throat symptoms continue, there is pain on swallowing, the voice is altered, and the tonsils are markedly prominent, generally covered by a yellowish membrane, while frequently one or both of them suppurate. The temperature remains high (103°-105° F.)

until the rash begins to fade, which it does in 3 to 10 days. A considerable polymorphonuclear leucocytosis is usually present.

The stage of *Resolution* is generally by *lysis*.

During the stage of *Convalescence*, with the disappearance of the rash desquamation occurs, beginning on the face, neck, and chest, and lasting for 1 to 3 weeks or more. There may be fine or large scales of skin, and sometimes the desquamation is so complete that the skin of the hands may be shed like a glove. It may continue for 5 or 6 weeks. It is interesting to note that a transverse groove or furrow is almost always present on the nails of scarlet fever patients, indicating an arrest of development during the disease.

Types.—*Scarlatina Simplex.*—Eruption with no throat symptoms.

Scarlatina Latens.—Where there is no eruption, but desquamation, or possibly the development of sequelae, indicates the existence of the disease.

Scarlatina Anginosa.—Eruption with more or less severe throat inflammation. Where the throat suffers, the temperature swings more markedly, and even in a favourable case it falls very gradually.

Severe types of Scarlet Fever:—

(1) *Toxic Scarlatina* or Scarlatina Maligna is due to the patient getting huge doses of toxin in a virulent type of scarlet fever, death sometimes occurring before the rash appears at all with marked nervous symptoms and especially delirium.

(2) *Ataxic Scarlatina* is a slightly less acute type, but one in which nervous symptoms are very pronounced, delirium, subsultus tendinum and coma vigil being associated with a tendency to hyperpyrexia, vomiting, etc. Death occurs in 2 or 3 days.

(3) *A Typhoid Type* with great weakness may be a less severe or at least a more protracted variety of the last type.

In any of these types the rash is often not characteristic, and it may, although rarely, be haemorrhagic.

Septic Scarlatina is a severe variety of Scarlatina Anginosa. The throat attacked by secondary organisms (Streptococcus pyogenes) becomes markedly ulcerated, and extensive necrosis may occur. Middle-ear suppuration often follows, and broncho-pneumonia is a common and fatal sequel.

Complications and Sequelae.—In the *Alimentary System* there may be extensive phlegmonous inflammation of the tissues in the neighbourhood of the pharynx, and rarely there is marked gastro-intestinal catarrh.

In the *Haemopoietic System* the lymphatic glands of the neck may suffer as already indicated.

In the *Circulatory System* endocarditis, sometimes ulcerative, and pericarditis may be present, while myocarditis is by no means uncommon.

In the *Respiratory System* a persistent rhinitis and broncho-pneumonia are probably the most frequent complications.

In the *Integumentary System* bed-sores may form on points of pressure.

In the *Urinary System* an acute parenchymatous nephritis is by far the most important complication; it generally appears during the 2nd or 3rd week, and sometimes is the result of exposure to cold, and may follow in even the mildest case. Oedema, which may become very pronounced, and suppression or the passage of small quantities of highly albuminous urine with much blood, and many blood and epithelial casts, indicate the onset of the attack. In every case of scarlet fever a careful examination of the urine should be made at short intervals until the patient is fully restored to health.

In the *Nervous System* the most important complication of scarlet fever is middle-ear suppuration, the infection spreading up the Eustachian tube, and resulting often in perforation of the tympanic membrane.

In the *Locomotory System* the joints may swell and become extremely painful. The finger joints are most commonly affected and next in order the wrists, ankles, and knees.

There is a tendency for the bacillus of diphtheria to attack the throat after scarlet fever, and this important fact must not be forgotten in cases where a suspicious membrane develops during convalescence.

Diagnosis.—In *measles* the longer period of invasion extending to 3 or 4 days, the typical crescentic rash, and the presence of the marked coryzal phenomena, with the absence of the general hyperaemia of the skin, should enable a definite diagnosis to be made. In *German measles* the fever is of

a milder type, the rash may closely correspond, but is less extensive, and the cutaneous hyperaemia is not so marked; further, the glandular enlargement in German measles is usually out of proportion to the involvement of the throat.

In *scarlatiniform erythema* there may be a somewhat typical rash with resultant desquamation, but the absence of marked temperature and throat symptoms helps the diagnosis.

In *acute exfoliative dermatitis* there are, as a rule, no throat symptoms at all, while the desquamation is by far the most distinctive feature of the disease. *Diphtheria*, or diphtheroid inflammation, is often a sequel to scarlet fever, but the examination of a swab from the throat prevents any mistake being made. Certain *drug rashes* also resemble the eruption of scarlet fever, such as those due to copaiba, belladonna, quinine, and salicylate of soda, but with a little care there should be no room for doubt.

Prognosis.—The milder cases terminate in recovery, but it must be remembered that the death-rate is often as high as 15 per cent, a fatal result being frequent in young children under five years of age. The malignant types of scarlatina are generally fatal. Hyperpyrexia, early collapse, marked vomiting and diarrhœa, prominent nervous phenomena, and a severe attack of scarlatinal nephritis during convalescence are all grave features.

Treatment.—An ordinary fever treatment should be sufficient for a mild case, but the most rigid precautions must be taken to prevent the spread of the disease, and, if possible, patients should be sent to a fever hospital. Failing this, the sick-room selected should be large and airy; unnecessary furniture should be removed, along with all curtains, carpets, etc., and the doorway protected by a sheet soaked in carbolic lotion. The diet should consist largely of milk and beef-tea, while aerated waters and fruits, such as oranges and lemons, may be freely permitted. During the period of pyrexia, sponging the patient is most refreshing; and when desquamation has commenced, the skin should be rubbed over with thymol in vaseline (gr. 10 to the oz.), weak carbolic oil (2-5 per cent.), or carbolated vaseline of a similar strength. The patient should be warmly clad, and especially so if allowed up during the period of desquamation, remembering the risk of renal complications. It is well in almost every case to confine the

patient to bed for 2 to 3 weeks. The urine must be examined for albumin, as already mentioned, every few days, and similar care paid to the ears, especially in cases in which the throat is severely affected.

For the throat symptoms much benefit may be obtained by the application of the glycerinum acidi carbolici (1 part in 2 or 3 of glycerine); or solutions of chlorate of potash, permanganate of potash, and other antiseptics may be used for gargling. Cold compresses applied externally are useful, and the internal administration of quinine, tincture of the perchloride of iron, guaiac, salicylate of soda, and other remedies, is of great value.

Hyperpyrexia should be treated by sponging, the cold bath, and other methods mentioned on page 6, taking care to remember the frequent necessity for stimulation, once the temperature has been reduced. If the middle ear suppurates, it may be necessary to incise the tympanic membrane.

Other complications which may arise should be appropriately treated.

In children the hair should be cut short. It often comes out badly, and close cropping to some extent obviates or lessens this tendency.

Serum therapy has been tried, but the results obtained so far are inconclusive.

Prophylaxis.—The physician should wear an overall, and special care should be exercised by the nurses in attendance to avoid conveying infection by their clothing to other persons with whom they come in contact. Probably the patient is most infectious during desquamation, and therefore attention to the smearing of the skin with an antiseptic, and the subsequent use of baths to separate the scales of skin, are invariably insisted on. On no consideration should a scarlet fever patient be considered disinfected until a period of at least six weeks has elapsed since the rash appeared, and every care must be taken to disinfect the clothes and anything which has been used in the sick-room.

V. MEASLES

(Morbilli; Rubeola)

A contagious fever, characterised by an eruption of typical spots, and associated with catarrhal inflammation of mucous membranes, especially the eyes, nose, and respiratory tract.

Etiology.—It spreads in epidemics, which are rarely long absent from large towns, and especially attacks children, although it may occur at all ages. It is most prevalent in winter and early summer. One attack protects from a second, as a rule. It is probably most infectious during the stage of eruption, but the patient is infective to others from the earliest symptom. The virus is very short-lived, and therefore the risk of infection through a third person is greatly diminished.

Pathological Anatomy.—There is a specific inflammation of the mucous membranes of the respiratory and, to a less extent, of the intestinal tracts, but the organism is unknown.

There is an oedema of the cutis, to which, probably, the typical measles papule is due.

Clinical Features.—The stage of *Incubation* of about ten days is followed by the stage of *Invasion*, lasting 3 or 4 days. During this latter stage the temperature gradually rises, there is quickened pulse, malaise, and the typical nasal catarrh, with conjunctival congestion and more or less bronchitis. The temperature may slightly fall before the next stage appears. Occasionally a preliminary measles or scarlet-fever-like rash appears, which fades before the true eruption develops, and a leucocytosis of 10 to 14,000 leucocytes develops at an early stage and persists throughout the stages of invasion and eruption. The stage of *Advance* or *Eruption* extends for 4 to 6 days, when a purplish-red, raised papular rash appears, the spots uniting together to

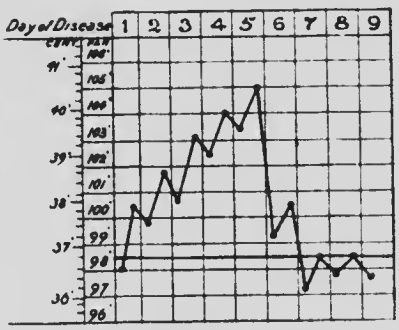


Fig. 7. Temperature Chart. Measles. Recovery.

form crescentic groups, commencing on the forehead and spreading over the face and body generally. Rarely is the rash haemorrhagic or do vesicles occur. The face has a blotchy, swollen look, and the whole skin presents a mottled appearance. The rash develops fully in 1 to 2 days, and, as a rule, the catarrhal symptoms gradually increase in severity. Koplik's spots, rose-pink in colour with a bluish centre, are to be found on the buccal mucosa in a large proportion of cases. They appear very early, and are therefore of diagnostic value. Later these spots coalesce, but many bluish-white specks can still be seen. The general rash fades on the 6th or 7th day, when the temperature falls by a *crisis* lasting for some 12 to 24 hours, and desquamation begins about the 8th day in the form of fine branny scales, very different from the large flakes of scarlatina. The sites of eruption can be recognised for some time by a pigmentary staining, which very gradually disappears. The stage of *Convalescence* is rapid if there are no respiratory complications.

Special Types.—The *Haemorrhagic Type* of measles, with a purpuric rash and haemorrhage from the mucous membranes is sometimes, though very rarely, met with, and the so-called *Typhoid Type* is applicable to cases of measles which show extreme prostration, or where an excessive dose of the poison is threatening the life of the patient.

Complications.—Capillary bronchitis leading to catarrhal pneumonia is to be dreaded, especially in delicate children, and frequently causes death. Laryngitis sometimes ensues. Eye and ear inflammation may occur, while cancrum oris is a rare sequel in delicate children. Persistent gastric or intestinal catarrh is not infrequent, and the disease may lead to permanent ill-health. Latent tubercular foci are apt to become active during convalescence.

Diagnosis.—From *scarlet fever*, the typical coryza, the character of the rash, and its presence round the mouth, and, lastly, the long period of invasion are absolutely diagnostic. From *German measles* the rash and the absence of the sore throat and also the absence of marked enlargement of the lymphatic glands are at least suggestive. The absence of Koplik's spots in both of these may be of diagnostic value in doubtful cases. In *typhus* there is always delirium, the face is little affected by the rash, and one obtains the history of

exposure to infection. The initial *small-pox* rash may be misleading, but rarely are the awful headache and backache of small-pox present in measles.

Prognosis.—Generally recovery from the fever, but the respiratory complications cause a large death-rate in young children. In certain epidemics the death-rate reaches a high percentage, but often a fatal result is due in part to the age of the child, the insanitary surroundings, and careless nursing. Diphtheria developing in a measles patient is always serious.

Treatment.—A mild case requires practically no medicine, and only ordinary dietetic and hygienic measures. Keep the child in bed in a fairly warm room, give a mild fever mixture (spiritis etheris nitrosi ♏ 15, and liquor ammonii acetatis *ad* ʒ 1), and feed on milk diet or beef-tea. There is a tendency to enteritis in some cases, and therefore a simple diet should be adhered to for at least a week after the crisis. Where there is much bronchitis a steam-kettle should be invariably used, and the utmost care taken to avoid the risk of extension downwards to the lungs. The eyes also require careful cleansing with a boric lotion.

It ought to be remembered that measles is contagious, and at least a fortnight must elapse before members of the family who have come in contact with the patient are free from the risk of infection.

VI. GERMAN MEASLES

(Rubella: Rötheln)

An infectious, eruptive fever possessing characteristics both of scarlet fever and measles.

Etiology.—There is much difference of opinion as to whether some cases are not scarlet fever and others measles, but our present position should be that it is due to a distinct organism. It is usually an affection of childhood, and one attack does not appear to confer any immunity, nor does an attack of this fever protect the patient from measles or scarlet fever. It is most commonly met with in epidemic form during springtime.

Pathological Anatomy.—The lymphatic glands of the neck are especially enlarged.

Clinical Features.—The stage of *Incubation* is 2 to 3 weeks. The stage of *Invasion* commences with malaise, headache, pains in the back and legs, coryza, sore throat, and rise of temperature, while nausea is not infrequent.

Usually about the 2nd or the 3rd day the stage of *Eruption* begins, the rash appearing first on the face and neck, or on the trunk, and rapidly spreading over the body. The spots are round or oval, discrete, or tending to coalesce to form irregular patches which somewhat simulate the crescentic rash of measles. The spots are not raised as in measles, and are not equally bright all over the body at once. At the same time the mucous membrane of the pharynx and, to a less extent, the nose and the conjunctivae inflame. The typical enlargement of the cervical lymphatic glands, and especially the glands behind the sterno-mastoid muscle, often precedes the rash.

In 1 to 3 days the eruption fades, the temperature, which may have reached 102° F., falls by *crisis*, and during the stage of *Convalescence* desquamation may occur to a slight extent.

Types.—The disease is usually extremely mild, and the patient may never be confined to bed, but malignant cases occasionally occur in which the patient dies prior even to the stage of eruption.

There are very few, if any, definite complications.

Diagnosis.—The rash resembles both *scarlet fever* and *measles*, but the stage of invasion is shorter than in measles, which ought to differentiate it from that disease; while the coryzal symptoms, which suggest measles, should prevent the diagnosis of scarlatina being made. The glandular enlargement is often helpful, especially in differentiating this fever from measles.

Prognosis.—Generally in recovery; severe epidemics are rare.

Treatment.—Where there is pyrexia the patient should be kept in bed; otherwise an ordinary fever mixture is all that is requisite. The diet should be on the usual lines, at least for the few days of pyrexia.

VII. SMALL-POX

(Variola)

An acute contagious fever with a typical pustular eruption.

Etiology. — It is due to a specific organism, probably a protozoon, which causes widespread and general epidemics unless in a community protected by vaccination.

The infection is conveyed by the secretion from the vesicles, and also by the scales of skin during convalescence. Guarnieri in 1892 described a sporozoon, to which he gave the name Cytoryctes Variolae, and which he believes to be present in small-pox and also in vaccinia, and the results of his researches have been added to by Councilman and others. The spores (1 μ) are first seen near the nuclei of the epithelial cells. They increase in size until each forms a body (10 μ) akin to the malarial zygote and containing many spores. The capsule bursts, and it is possible that the new spores are distributed by the blood stream throughout the body. Pustules also contain other organisms, mostly staphylococci, which are secondary and have nothing to do with the primary infective organism.

The inoculation of the child *in utero* has occurred. Small-pox is common at all ages, most fatal in the very young, and both sexes suffer equally. While more prevalent in the tropics, it is met with in all climates. Winter and spring are the usual periods for epidemics. Certain races seem to have a higher death-rate from small-pox than others. The Mexicans, for example, died by thousands in one epidemic of small-pox.

Pathological Anatomy.—The typical papule is found in the superficial layers of the rete mucosum close to the cutis vera. The epithelial cells are granular and oedematous, and two processes are described by Unna—a reticulating degeneration and an epithelial oedema. The process advances most rapidly at the periphery of the pock, causing a balloon-like appearance, the central part undergoing less-marked change, and thus producing the typical umbilication of the pock. There is always active inflammation round about the pock, and a rapid invasion of leucocytes, mostly polymorphonuclear, into the affected area towards the end of the papular stage, when the pock becomes hard and shot-like. Scarring depends upon

damage to the cutis vera by scratching or extension of suppuration downwards.

The spleen is enlarged. There is sometimes myocarditis, rarely endo- or pericarditis. Pocks may appear on the larynx, causing ulceration, while the bronchi may be involved, and even septic pneumonia results. The skin often shows intense congestion, and gangrene of the genitals may occur, especially where the patient is suffering from a coincident severe gonorrhœa. Bright's disease may develop during the stage of convalescence, and sometimes suppuration of joints.

In a malignant case the patient dies before the eruption comes out, or there may be extensive haemorrhage from serous membranes or into the pocks or elsewhere.

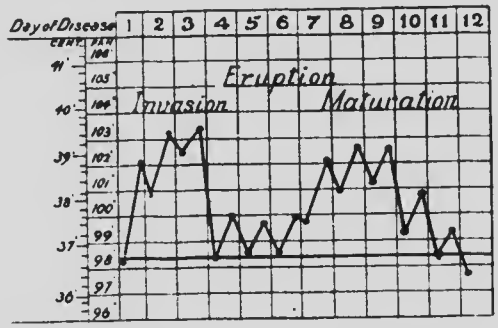


FIG. 8. Temperature Chart. Small-pox. Discrete case. Recovery.

Clinical Features.—The stage of *Incubation* is 11 to 14 days. The stage of *Incasion*, lasting from 2 to 3 days, is accompanied by very definite phenomena. There are rigors, severe frontal headache, pains in the back and epigastrium, vomiting, often delirium at night, and a temperature which rapidly rises each day to a higher point than the preceding day, and with only a slight morning fall. There are sometimes convulsions in children. The initial rash, resembling scarlet fever or measles, occasionally a mixture of both, and sometimes petechial, usually comes out on the lower region of the trunk, in particular the "bathing-drawers" area, the inner aspect of the thighs, and the lateral regions of the chest and axillæ. These rashes are uncertain as regards appearance, extent, and duration. A very remarkable rash is the "lobster rash" in which face and whole body become deep red. It is of very serious import, and generally precedes severe haemorrhagic small-pox.

In the stage of *Advance* or *Eruption*, at first the temperature falls often with sweating, and papules appear on the forehead, face, scalp, limbs (especially the wrists), and back, and they may be seen on the soles of the feet and palmar aspect of hands and fingers. The flexor aspects of the limbs are less affected than the extensor. On the 4th or 5th day the shot-like papules become vesicular, are as a rule discrete and umbilicated, but may even at this period show a tendency to be confluent. By the 7th or 8th day the vesicles become pustules, the umbilication tends to disappear, and the pustules are surrounded by a zone of inflammation. This is termed *maturation*, and the temperature rises once more during this stage, as indicated in the chart. The pocks are tense and painful, there is much swelling of the skin, and the features of the face may almost be unrecognisable. In a mild discrete case the temperature may only be up for one full day and then it falls.

In the *confluent* form the pocks may be first discrete and then later run together: they usually appear early, often before the 4th day, and the initial discomfort is much greater. During the stage of maturation the inflammation is more intense, the temperature much higher (104-105° F.), and delirium and other nervous phenomena are common. Diarrhœa at this time is very frequent, due largely to septic absorption.

When the pocks burst the patient becomes extremely odoriferous. About the 10th to the 18th day the pustules dry and form cicatrices, and soon afterwards desquamation begins. This process is often called *desiccation*, and the whole skin may be shed *en masse*. A leucocytosis of 12,000 to 16,000 develops about the 8th day. This is followed by a fall and later by a rise to 18,000 or 20,000 on the 12th to 14th day. A mononuclear increase has been considered favourable, but this lacks proof.

Resolution is by *lysis*, and for long afterwards the skin remains red and unsightly. During the process of cicatrisation the itching is apt to be intense. There is sometimes a prolonged period of ill-health, which may extend to months.

Salivation, vomiting, and constipation, more often diarrhœa, may be present. Hypostatic congestion of the lungs, pneumonia, and pleurisy are not infrequent, and sometimes the eruption attacks the larynx. There may be albumin in

the urine, and a degree of delirium is very common, while subsultus tendinum and coma vigil may be present in severe cases.

As already indicated, pocks may be found on the mucous membranes, and sometimes haemorrhage occurs in connection with the pocks.

Types.— *Variola Discreta*, where the pocks are isolated, has already been described.

Variola Confluens is much more serious, 50 per cent being fatal. The stage of maturation is very severe, and the pocks are specially confluent on the face and limbs. There is not the same remission of fever prior to maturation. The mucous membranes are more apt to be affected, and generally there are pocks on the conjunctiva and even the cornea. These may readily lead to panophthalmitis from perforation of the eyeball. The patient usually succumbs between the 10th and 28th days, and the constitutional phenomena are apt to be extremely severe. Vomiting, persistent diarrhœa, salivation, albuminuria, and various respiratory complications are present as a rule.

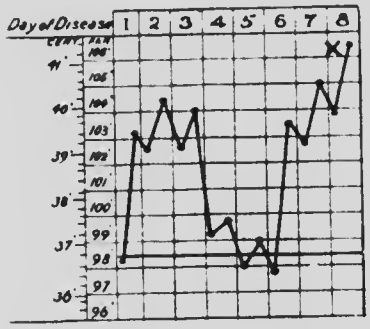


Fig. 9. Temperature Chart. Malignant Small-pox. Death on 8th day.

Variola Maligna.—In this type the patient may die from excessive doses of the poison prior to the stage of eruption (toxic small-pox). The preliminary rash may be the lobster-like rash, or excessive haemorrhage may occur into the pocks and from the mucous membranes (nose, gums, stomach, and bowels) and the kidneys, etc. A trivial haemorrhage into the pocks does not constitute haemorrhagic small-pox. The true haemorrhagic form is called black small-pox.

Varioloid or modified Small-pox is generally considered to be small-pox occurring in a vaccinated person. There is a well-marked stage of invasion, but the rash comes out very rapidly. The pocks are generally few in number, are present on the face and limbs, and at an early period become pustular, implying that maturation is earlier than in the ordinary case of small-pox. There are also cases in which the papules abort

without ever becoming really pustular. Occasionally the pocks come out in crops like chicken-pox. Varioloid is just as infectious to others, and hence the importance of a correct diagnosis.

Complications. *Alimentary System.*—The throat, tongue, and stomach may all be involved by the pocks. Gastric and intestinal catarrh is not uncommon, especially in children, and vomiting has been already referred to as a typical manifestation of the disease.

Haemopoietic System. The lymphatic glands are often enlarged.

Circulatory System.—Myocarditis, endocarditis, and pericarditis have been mentioned, and may occur as complications, while more frequently functional cardiac disturbances, especially palpitation, trouble the patient during convalescence.

Respiratory System.—Laryngitis, bronchitis, broncho-pneumonia, and hypostatic congestion are all of frequent occurrence.

Integumentary System.—Erysipelas is not uncommon, and even gangrene may involve the genital organs should there be a coincident gonorrhœa, while boils frequently occur during convalescence, due to interference with the sebaceous glands.

Genito-Urinary System. Bright's disease occasionally supervenes. There is often a peculiar variety of testicular involvement in which nodules develop in the testicle, and sometimes a typical orchitis is set up.

Nervous System.—The pocks on the conjunctiva may lead to ulceration of the cornea and cause blindness. Deafness, sometimes due to inflammation spreading up the Eustachian tube, may also be present. Delirium is common in severe cases, and coma, more rarely encephalitis, and paralyses of different types may occur.

Locomotory System.—Inflammation of joints sometimes develops during desquamation, and may lead to permanent lameness.

Diagnosis.—The backache, headache, and the nocturnal delirium are typical. The shotty hardness of the pocks and their appearance under the horny skin of feet and palmar aspect of fingers are also distinctive. Not merely may *varicella* be differentiated by attention to these features, but

the stage of invasion is preceded in that disease by no typical phenomena, and the rash comes out in twenty-four hours, usually in groups of spots on different days. In chicken-pox the rash appears first on the upper part of the trunk, while in small-pox the forehead and wrists are first affected. The initial rash in small-pox, measly or scarlet fever-like, must not be forgotten, and the sites of the initial rash should be carefully noted. Varioloid is certainly most apt to be confused with chicken-pox.

Prognosis.—In mild cases recovery is common, especially in cases modified by vaccination. In confluent and haemorrhagic small-pox at least 50 per cent die.

The following features are unfavourable:—

1) The quality and character of the eruption, especially if truly haemorrhagic.

2) The extent of mucous membrane involved in the eruption, if considerable.

3) The age of the patient, the disease being particularly fatal in the very young.

4) The marked nervous symptoms present, especially wild delirium, deep coma, or intractable sleeplessness.

5) The previous habits of the patient: alcoholism is peculiarly unfavourable.

6) Pregnancy nearly always leads to abortion or miscarriage, and so increases the risk to the patient.

7) Insanitary surroundings, especially dirt, an insufficiency of fresh air, and unsatisfactory diet and nursing.

On the other hand, the presence of well-marked vaccination scars is a good feature, as it greatly minimises the risk of a severe type of the fever.

Treatment.—In a mild case comparatively little treatment is necessary, but in a severe attack the utmost care by both nurse and doctor may alone save life, obviate disfigurement, and prevent much suffering. The sick-room should be large and well ventilated, or, better still, a tent or wooden shed should be used, but the temperature must be kept up to about 50 or 60 F. A water-bed should be provided, and extremely light but warm clothing. The skin is unusually sensitive, and the finest texture of night-gown and bed-linen adds greatly to the comfort of the sufferer. Bright sunlight should be excluded from the sick-room, but orange or red light, which theoretically should obviate the scarring (due to actinic

rays), renders the patient subjected to this treatment excessively depressed. Carpets and curtains and all unnecessary furniture should be removed. The diet should consist of milk, soups, custards, jellies, and similar easily digested food. Relieve the backache with opium, and order ice and aerated water for the intense thirst and vomiting. During the stage of maturation feed generously, and where possible, administer alcohol freely, because at that time the patient is exposed to the danger of septic absorption.

A warm bath soothes the itching, and the skin should be anointed with an antiseptic oil, the pustules pricked, especially those on the face, and cold water compresses used for allaying the discomfort. Lint is an excellent medium for use in applying the oily preparation selected, and it may be covered with oil silk so as to protect the night-dress and bedclothes. Watch the eyes carefully, washing frequently with boric lotion where pocks are present on, or near, the eyeball. Remember the necessity for abundance of fresh air for patient and attendants alike.

VIII. VACCINATION

(Vaccinia)

Vaccination was discovered by Dr. Edward Jenner in 1798, when he vaccinated with cow-pox a boy eight years of age.

Prior to this the inoculation method was practised. Inoculation consisted in the introduction of small-pox matter direct from a small-pox vesicle prior to the pustule stage. The patient had, as a result, an attack of small-pox which was usually mild, but was naturally extremely infectious, and could cause infection to others of possibly a severe type of the disease. The death-rate proved, however, to be extremely small, but the patient required to be confined in hospital during the process.

Vaccinia, or cow-pox, is probably small-pox passed through the calf. The vesicles in cow-pox are found on the teats or udders of cows, and the disease is transmitted to the hands of those milking the cows, and also from one cow to the other by contact. Jenner noticed that in an epidemic of small-pox, milkmaids, who had previously become accidentally inoculated with vaccinia, had a marvellous immunity from small-pox.

Primary Vaccination.—*Local Phenomena.*—2nd to 3rd day. Slight papular elevation.

5th to 6th day. Distinct vesicles of bluish-white colour, with raised edge and central depression.

7th to 8th day.—Areola forms round base, and spreads for two days. It is circular, 1 to 3 inches in diameter, and may be accompanied by hardness and swelling of subjacent tissues.

8th day.—Vesicle perfect, plump, distended with clear lymph, firm margin, with marked central cup.

10th to 13th day.—Areola fades and disappears.

14th to 15th day. Scab forms as result of drying of vesicle. This contracts and blackens.

20th to 25th day. Usually on 21st day scab falls off, leaving a cicatrix which is circular, depressed, and permanent.

Rise of Temperature.—Slight from 5th to 7th day greater from 8th to 10th day, *i.e.* during areola stage.

The Complications which may result from vaccination depend largely upon three factors: *Firstly*, the care with which the trivial operation is performed; *Secondly*, the health of the child at the period of vaccination; and, *Thirdly*, whether calf lymph or humanised lymph is used.

To obviate complications the following precautions should be observed:—

1. The site of vaccination should be carefully cleansed with an antiseptic lotion, then washed with boiled water, and the part covered after the operation with a pad of aseptic wool, and during the vesicle stage protected from injury.

2. The child should not be vaccinated unless it is in good health, and the period of teething should be, if possible, anticipated by previous vaccination.

3. Humanised lymph causes less reaction in the case of a delicate child, but the child from whom the lymph is taken must be healthy, and the possibilities of both tuberculosis and syphilis must be taken into account.

The chief complications of vaccination are the following:

Ulceration and *suppuration* at the site of the operation, with abnormal enlargement of the lymph glands in the armpit. This is generally due to carelessness. *Eczema* may also be caused where a child has a predisposition to this affection. *Erysipelas* is not infrequent, and is due to want of aseptic precautions. *Excessive constitutional disturbance* is only

present in delicate children, and especially when calf lymph and not humanised lymph is used. *Syphilis* and *tuberculosis* are rarely inoculated, and only when humanised lymph is used and insufficient care taken in the selection of a healthy child as a medium.

Generalised vaccinia is rare. By this is meant the appearance of the typical spots over the body generally, although more numerous as a rule on the limb which has been vaccinated.

Revaccination should be performed every seven years, and invariably after exposure to the risk of small-pox. A good mark ought to be visible on the arm or leg, and should be looked for before protection from small-pox can be assured. As a rule four sites of inoculation are considered necessary, and the insertion of the deltoid muscle on the left arm is the usual position selected.

IX. CHICKEN-POX

Varicella

An acute infectious fever most common in early life, consisting in the appearance of successive crops of vesicles; it is quite distinct from small-pox.

Etiology.—Generally epidemic, sometimes sporadic. It is a disease of childhood or infancy, and no specific organism has as yet been discovered. It is very contagious, but there is doubt as to the possibility of inoculation.

Pathological Anatomy.—Unna describes reticulative degeneration, and to a certain extent an oedematous change in the epithelial cells in the pock, resembling in a somewhat milder degree what occurs in a small-pox pustule. The vesicles are generally simple and not umbilicated.

Clinical Features.—Stage of *Incubation*, 10 to 20 days. Stage of *Invasion*, with slight fever and malaise, and occasionally an evanescent scarlatiniform or other rash, is followed in one, sometimes two days, by the stage of *Eruption*. Red, raised papules appear first on the trunk, forehead, and face, which in a few hours become vesicles, often typically oval in shape and containing clear fluid. They are very superficial, and they may, or may not, be surrounded by a slight area of hyperaemia. In one to two days the fluid becomes cloudy or even purulent,

and soon dries up, leaving no scar. Several crops appear on successive days, often about two to three hundred spots in all, and are spread more or less over the whole body. There is slight feverishness with the eruption, and specially so where much pustulation occurs, or where there is a tendency to a confluent form.

Diagnosis.—Differentiate from *small-pox* by the more superficial, clear, and less slotty pocks with little or no surrounding inflammation, and very rarely definite umbilication. They are numerous on the trunk, and do not as a rule involve the soles of the feet or the palmar aspect of hands or fingers. In small-pox the face and forearms are always specially affected with pocks. The stage of invasion is much shorter and much less marked in chicken-pox.

Prognosis.—Almost invariably in recovery. Very rarely do the lesions become gangrenous.

Treatment.—Relieve the itching in the same way as in small-pox. Prevent scratching, which may cause injury to the true skin and permanent disfigurement. A soothing ointment, such as zinc oxide, and muffling up the patient's hands, if it is a child, will usually obviate any trouble.

X. DIPHTHERIA

An acute infectious disease characterised by the development of a fibrinous exudation most generally on the pharynx, and in which the toxins produced by the organism cause marked constitutional symptoms.

Etiology. The Klebs-Löffler bacillus is the specific organism present, although very frequently other organisms may be associated. The bacillus is non-motile, measures from 2 to 3 μ in length and about ·5 μ in breadth, is very slightly curved, with rounded ends, and the ends sometimes stain in such a way as to suggest sporulation, which, however, is not present. The bacilli grow readily on solidified blood-serum at blood heat, and typical colonies may be seen as early as twelve hours after inoculation. Many variations in appearance are met with in the process of cultivation, but the bacilli almost always occur in the throat and elsewhere in irregular clumps, and they stain by Gram's, Neisser's, and other distinctive methods. Roux

and Yersin separated the toxin, which was found to be as toxic to animals as inoculation with the organism itself. The toxic substances, for there are several different ones, have also been studied: one which readily unites with, and is easily neutralised by antitoxin causes the local oedema, while another does not unite with antitoxin so completely or firmly, and it produces the paralytic phenomena. This latter, which has been called *toxone*, may by its incomplete combination with antitoxin explain why paralysis may follow in cases of diphtheria notwithstanding the use of antitoxin.

The disease is transmissible by actual contact and probably not by the air, and therefore the use of common drinking-cups and kissing an infected patient would be at least certain, if not the most common, modes of infection. The disease, however, has been gradually diminishing in frequency, owing to sanitary improvements, while the decrease in the death-rate depends to a great extent on the use of the antitoxin.

The specific organisms are found in the affected patches on the throat, and are sometimes discoverable on the fauces of perfectly healthy persons. Animals, especially cats, appear to suffer from diphtheria. Milk is a frequent mode of infection. As already indicated, a mixed infection is not uncommon, and there is a distinct tendency for diphtheria to be associated with scarlet fever, measles, and whooping-cough, but it must be remembered that the appearance of a membrane does not always signify the presence of Löffler's bacillus. The disease is endemic in cities, epidemic at certain seasons of the year, and is most frequently met with between the ages of 2 and 15, and especially between the ages of 2 and 5.

Pathological Anatomy.—The tonsils, soft palate, and uvula are usually affected: the membrane is at first adherent, and when scraped off leaves a raw bleeding surface (during life). In later stages, however, and most frequently at the time of death, the membrane is readily separated.

The membrane consists of the superficial layers of the mucosa which have undergone coagulation necrosis, and in addition there are leucocytes and red blood corpuscles, fibrin, and many colonies of the specific organism. The diphtheritic membrane may involve the larynx and spread down to the bronchi, and even to the lung, producing septic pneumonia,

while the lymphatic glands, which are always enlarged, may in rare cases undergo suppuration.

In some cases the membrane is slight in the pharynx and is extensive in the larynx.

In the *Alimentary System* there may be extensive involvement of the mouth, and even of the œsophagus and stomach.

In the *Hæmopoietic System* the spleen is usually enlarged and acutely congested.

In the *Circulatory System* an interstitial myocarditis has been described, while an atonic and fatty condition of the heart is a common result of the toxin.

In the *Respiratory System* septic pneumonia and pulmonary collapse may occur; while in the *Urinary System* acute nephritis is a somewhat rare complication.

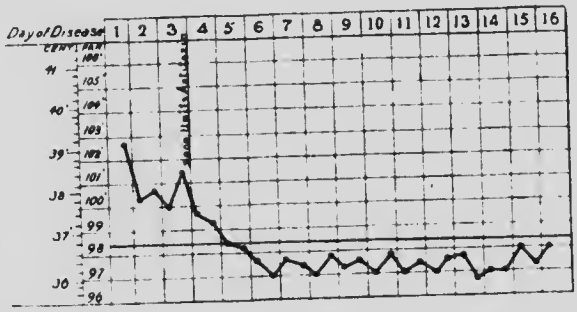


Fig. 16. Temperature Chart. Diphtheria, showing effect of antitoxin on temperature.

In the *Nervous System*, in which paralytic phenomena result from the toxin, there may be the changes met with in parenchymatous neuritis with consequent changes in the corresponding nerve-cells.

Hæmorrhages are not uncommon, just as in many malignant cases of fever.

Clinical Features.—Stage of *Incubation*, 2 to 7 days. The stage of *Invasion* begins with marked constitutional disturbance, malaise, nausea, fever, etc., and the temperature rises rapidly, reaching about 103° F. by the end of the 2nd day, at which time the typical patch on the throat has generally formed.

Diphtheritic patches appear on the tonsils, soft palate, and uvula, while in certain cases, without any pharyngeal affection, the larynx may be involved. The mucous membrane is first

reddened and swollen, and greyish-white membranous patch or patches appear, covering a raw bleeding area, and the fauces become of a dusky-red colour. Often within a few hours the membrane begins to develop, although in certain cases it may be delayed, and it is important to remember that as the membrane is formed in part from the mucosa itself, it is at an early stage firmly adherent. The tongue is thickly furred, the glands of the neck become swollen and tender, and chewing and swallowing are extremely painful, and there is generally a trace of albumin in the urine. Pain varies much in degree and depends on the amount of congestion: in some cases there is remarkably little discomfort. Probably pain, swelling, and certainly suppuration of neighbouring lymphatic glands depend on the presence of a mixed infection. In a favourable case the membrane may cease to spread and gradually separates about the end of the 4th day, while in a more severe case it may persist till the 8th to the 10th days. There may be little membrane formation and merely redness, and yet the case is diphtheritic, a fact which indicates the great value of bacteriological examination in every doubtful case.

In all cases of diphtheria there is some constitutional disturbance, but it varies in degree in different cases. The pulse is rapid, and particularly in severe cases the ashen-grey face is characteristic of the disease. An erythematous rash occasionally appears in the early stages of the fever. Laryngeal diphtheria gives rise to attacks of dyspnœa, often paroxysmal and occurring at night. The distress may rapidly increase until the patient is asphyxiated, or the attack may temporarily yield. Much relief is obtained if the obstructing membrane is in part coughed up, when the croupy character of the breathing disappears.

The toxin exerts its influence in certain cases more than in others, and mainly on the heart and certain peripheral nerves, especially those which innervate the soft palate, the laryngeal muscles, more rarely the muscles of the eyeball, and still more rarely other muscles of the body.

Various types of diphtheria have been described, depending much on the position of the false membrane, whether in the pharynx, larynx, or nose, or whether involving a wound or the anus or vulva. A malignant type of diphtheria is generally due either to (1) a tendency to severe haemorrhage from

mucous membranes and elsewhere: or (2) an intense toxaemia, which kills the patient; or (3) extension of the membrane, as, for example, to the lung, producing a very grave diphtheritic broncho-pneumonia.

Deep ulceration and gangrene are also sometimes met with.

Complications.—In the *Circulatory System* heart failure is to be dreaded. It is probably due to the action of the toxin on the heart causing dilatation but it may be the result of fatty degeneration, and a fatal syncope may follow even a slight effort, such as sitting up in bed, and rapid and irregular action of the heart with a tendency to the galop rhythm should be considered a serious phenomenon.

In the *Alimentary* and *Respiratory Systems* we have only to note again the tendency to the spread of the false membrane down the alimentary and respiratory tracts respectively: while the frequent development of oedema of the glottis must not be forgotten in cases of laryngeal diphtheria.

In the *Integumentary System* urticaria sometimes appears, and purpura may develop in cases of a malignant type.

In the *Urinary System* albuminuria is typical of the disease, and acute nephritis is an occasional complication.

In the *Nervous System* the leading complication is paralysis, to which reference has already been made. It is the result of the toxin, and occurs in 6 to 20 per cent of all cases of the disease. It usually appears 14 to 21 days after the attack, and may follow in the mildest case. The most common form is paralysis of the soft palate, causing a nasal type of voice and a tendency for fluids, which the patient drinks, to regurgitate down the nose. On inspection the soft palate is seen to hang absolutely motionless during both breathing and phonation, and in addition it is insensitive to the touch. The legs may be paralysed, next most frequently, according to Claude Ker's statistics. Sometimes there is an ataxic gait, with loss of knee-jerks, and there may be some analgesia. Less commonly there is paralysis of the muscles of the larynx, producing a tendency to asphyxia, because the openers of the glottis (the posterior crico-arytenoid muscles) are paralysed. Occasionally the eye muscles are attacked, causing squint, ptosis, and sometimes paralysis of the ciliary muscle: more rarely other cranial nerves are involved. The arms are much less frequently

affected, and only in a very few cases does paralysis of the intercostal muscles and diaphragm endanger life. The paralysis is, as already indicated, a parenchymatous neuritis, giving in marked cases a definite reaction of degeneration on testing the affected muscles with the galvanic current. There is a varying degree of anaesthesia with, at first, a feeling of numbness and the pins-and-needles sensation. The paralysis usually disappears in from 2 to 3 weeks, although it may last for a longer time.

Diagnosis.—In every suspicious case a swab from the throat should be examined for the specific organisms. Important as bacteriological examination is, it must be remembered that a culture takes 12 to 24 hours to grow, so that some delay is inevitable in making the diagnosis. In the throat of *scarlet fever*, in *follicular tonsillitis*, and in *thrush*, the diphtheritic bacillus is not present, but on the other hand it must not be forgotten that streptococci are often met with along with Löffler's bacillus. The membrane in thrush is whiter and more easily removed by scraping.

In diphtheria of the larynx the typical croup-like breathing, the albumin in the urine, and the obvious and increasing distress of the patient, render the diagnosis fairly easy even without a laryngoscopic examination.

Prognosis.—The death-rate is still high. Formerly 30 to 50 per cent, with antitoxin it is now 3 to 14 per cent. Death is most apt to occur from heart failure, asphyxia where the larynx is involved, or from extension of the membrane into the lungs; and in addition so-called malignant cases are nearly always fatal.

Cases of diphtheria are now treated by the antitoxin method, and if this treatment is applied at a sufficiently early period, the chances of recovery are greatly increased, the death-rate rising rapidly each day which passes before the antitoxin is injected; in cases treated on the 1st or 2nd day the death-rate is proportionately low.

Treatment.—The patient should be placed in a room with plenty of fresh air, and with the minimum of furniture; if possible for cases of laryngeal diphtheria, a continuous spray of steam should be played upon the patient's face. The throat should be first sprayed or painted with an alkaline solution, such as sodium bicarbonate 1 drachm to the ounce of water.

and then painted with the glycerinum acidi carbolici, diluted 1 part in 2 or 3 of glycerine, corrosive sublimate (1 in 1000 in water), chlorine water, or other antiseptic. In nasal cases the nostrils should be sprayed or washed out with antiseptics, and at an early period in most laryngeal cases tracheotomy or intubation must be performed, because asphyxia may rapidly develop and greatly endanger the success of the operation if delayed. Silver nitrate stick, or a 10 to 20 per cent solution in distilled water, applied to the affected part is of great value.

Quite as important as the local treatment is the careful feeding of the patient. Strong soups should be freely given, and alcohol, strychnine, and iron are frequently of great service. The risk of paralysis of the heart should ever be before the minds of doctor and nurse, and on no consideration should the patient be allowed to sit up in bed until all such risk has passed away. Paralysis of the voluntary muscles (after a short period of rest) should be treated by massage and a combination of galvanism and faradism. In rare cases, where there is paralysis of the respiratory muscles, artificial respiration has to be resorted to.

Antitoxin Treatment.—This consists in the injection of the blood-serum of animals immunised against diphtheria. It is obtained from the horse, and the so-called "unit" is 100 times the amount of serum required to neutralise the minimum lethal dose of the toxin injected into a guinea-pig weighing 300 grms. At one time 2000 to 3000 units were considered sufficient, but experience teaches that either this dose must be repeated more than once within twenty-four hours, or else a single dose of 5000 to 6000 units is administered, and a smaller quantity later, if necessary. Repeated doses of antitoxin may have to be given in severe cases, and there should be no hesitation in pushing the treatment, as improvement may not begin until some 20,000 units have been injected. The back between the shoulder-blades is often selected as the site of injection. It is introduced into the subcutaneous tissue. The antitoxin retains its properties for nearly a year. The death-rate has been diminished by one-third as the result of this treatment.

Occasionally, following the administration of the antitoxin urticarial or erythematous rashes appear on the skin, associated sometimes with fever and joint pains, and these may appear

from 1 to 3 weeks after the injection. As a rule the antitoxin causes the extension of the membrane to cease, and the patient rapidly improves.

Diphtheria bacilli are very tenacious of life, and for long after apparent complete cure the patient's pharynx may contain the bacilli capable of inoculating others with the disease. It is therefore desirable in *all* cases to use an antiseptic mouth-wash such as 1 in 10,000 corrosive sublimate for a considerable period after cure, and in patients who remain infective to others, special local applications of antiseptics should be regularly carried out.

Diphtheroid Inflammation

It has already been indicated that there are membranous inflammations involving the throat and larynx which are not due to the Klebs-Löffler bacillus. These may result from the presence of the Streptococcus pyogenes, and the clinical features may closely resemble true diphtheria. These cases sometimes originate in caseous deposits in the tonsillar crypts, or may be associated with one of the continued fevers, and especially scarlet. While many instances of so-called croup are in reality of diphtheritic nature, it is probable that cases in which a bacteriological examination has not been made may be wrongly called diphtheritic, and such cases belong to the diphtheroid group.

The clinical features closely resemble diphtheria, and a streptococcal infection may prove quite as fatal in delicate patients, although in the majority of cases so affected the symptoms are mild. It should be remembered that paralysis may follow a streptococcal infection, just as it may result from true diphtheria, and the treatment carried out should be quite as thorough, and much on the same lines as that recommended for the more serious disease.

Vincent's angina, due to specific bacilli and spirilla, may cause a membranous tonsillitis not unlike diphtheria. It is sometimes epidemic. Swab examinations at once determine the diagnosis.

XI. WHOOPING-COUGH

(Pertussis)

An acute infectious fever, characterised by peculiar spasmodic attacks of coughing, terminating in the typical "whoop," and usually preceded by a period of bronchial catarrh.

Etiology.—Generally epidemic. It occurs almost exclusively in childhood, and before the age of ten: it is less common in tropical countries, and it is frequently associated with an epidemic of measles. Girls are more commonly affected than boys, and it is said that with them the death-rate is higher.

Afanassjev has discovered a short, thick bacillus, capable of producing the typical spasms in animals, and which he believes to be the specific organism of whooping-cough. The infection is certainly carried by the nasal and bronchial mucus, and therefore the clothes of the patient should be considered as infectious to other children. The risks of infecting others diminishes after the early stages of the fever are past, and during the greater part of the spasmodic stage there is little risk of the patient transmitting the disease.

Pathological Anatomy.—Bronchitis is always present, and there may be extension of inflammation to the lung, causing catarrhal pneumonia. From the strain of the coughing emphysema frequently results.

An attempt has been made to lay the blame of the typical spasm upon the swelling of the bronchial glands, or possibly the thymus: while it has also been suggested that the effect of the poison on the vagi nerves may explain the hyperaemia of the bronchi and the spasmodic coughing.

Clinical Features.—The stage of incubation is about 1 to 2 weeks. The fever may be divided into three stages:—

Firstly, a stage of ordinary bronchial catarrh commonly lasting 7 to 10 days, during which the usual clinical features of bronchitis become more and more marked, and there is a pretty definite coryza. The temperature is also, as a rule, slightly raised. Stress has been laid by Crombie on a marked increase of lymphocytes appearing in the blood as an early sign of the disease.

Secondly, a stage when the typical spasm is superadded and remains present usually for a similar or somewhat longer

period of time. There are a great many short, spasmodic, expiratory coughs without any air entering the lung, the patient often becoming black in the face, followed by a long crowing inspiration, producing the typical "whoop" or "kink." A number of attacks follow each other, and generally relief is not obtained until either vomiting occurs or a considerable quantity of glairy mucus is expectorated. These paroxysms may be frequent, or only occasional: they come on at night-time before sleeping, often after meals, almost constantly if the patient is excited, and are liable to be induced by hearing another patient cough.

During the attacks many results of the great vascular strain may occur, such as epistaxis, expectoration of blood, conjunctival haemorrhage, rupture of the tympanic membrane, and convulsions. Emptying of the bladder and bowels is not uncommon, and even prolapse of the rectum and hernia may be induced, particularly in children. In an older patient there is a distinct danger of cerebral haemorrhage. In most cases the attacks are so severe that the child rushes to the nurse or holds on to the bed, and in the infant the sufferer's face manifests much fear during the paroxysm.

Stress has been laid upon the presence of a small ulcer under the tongue, due to the lower teeth cutting the fraenum during the spasm.

Thirdly, during the next ten days the spasm gradually disappears, and in a favourable case the child should be practically well after a period of 3 to 4 weeks in all. In certain patients there is a tendency for the spasm to persist, and should the child have a slight attack of bronchitis, an occasional typical whoop may reappear.

Complications.—The complication to be most dreaded is catarrhal pneumonia, and for the prevention of this a great deal depends upon the capable nursing and treatment of the patient.

Emphysema is a common sequel, and in particular it occurs in cases in which the whooping-cough is the direct cause of a persistent bronchitis.

Diagnosis.—There is nothing which imitates the whoop of this disease; even in *hysteria* the cough is rarely or never the same. During the first stage of whooping-cough diagnosis is impossible.

Prognosis.—The disease in itself is not serious, but when catarrhal pneumonia follows (especially in young children) it is very frequently fatal.

Treatment.—There are two methods of treating whooping-cough: (1) We can either, in perfectly strong children, treat them with fresh air, and in summer this is probably the most satisfactory plan. The child should merely be kept warm, properly fed, and otherwise be allowed to be as much in the open air as possible.

(2) In most cases, however, and certainly in cold weather, the patient should be kept exclusively in one room, or at least in one atmosphere. The air should be impregnated with a mixture containing creosote, guaiacol, or other soothing agent. Cresoline will be found admirably suited for this purpose, and the repeated vaporising with this or similar drugs should be persisted with night and day.

Internally much benefit is derived from giving fairly large doses of dilute hydrocyanic acid, bromides, chloral, and similar remedies. Bromoform in $\frac{1}{2}$ to 1 minim doses dropped upon a lump of sugar has been specially recommended for controlling the paroxysms of coughing.

Externally gentle friction with camphorated oil, and sometimes the use of linseed poultices, are helpful, and in every case the chest should be covered with a Gamgee tissue jacket. The child should be kept as free from excitement as possible, and the diet be such as to discourage the all too frequent tendency to vomiting.

XII. MUMPS

(Cynanche Parotidea; Epidemic Parotitis)

An acute, infectious, febrile disease, characterised by inflammation of the salivary glands, especially the parotid; often occurring in epidemics, and sometimes, by metastasis, involving the testicles in boys, the breasts, and more rarely the ovaries, in girls.

Etiology.—A disease of childhood and adolescence, which is prevalent in spring and autumn, and is probably due to the specific diplococcus (Laveran) which has been isolated from the saliva. It is highly contagious, and is said to be commoner

in males. The patient is infectious for a period of about three weeks after the onset of the disease.

Pathological Anatomy.—The parotid and other salivary glands swell, the oedema being rather interstitial than parenchymatous in position. Rarely does suppuration develop.

Clinical Features.—The stage of *Incubation* lasts from 2 to 3 weeks. The stage of *Invasion*, during which the fever is marked, the temperature rising to 103 or 104 F., is accompanied by aching pains, especially in the salivary glands, and limited, as a rule at first to one side of the face. The gland rapidly swells, not merely the part of the parotid in front of the ear being enlarged, but also the part behind the lobe of the ear. The gland has a somewhat elastic feeling on palpation, is tender to the touch, and is painful on moving the lower jaw, as in the act of eating. Other salivary glands may be affected either in whole or part. Salivation is painful, and therefore acids should not be introduced into the diet (oranges or lemons) or into any medicine administered. The pain shoots up to the ear, and sometimes tinnitus aurium is produced. One side may alone be involved, but generally the swelling spreads to the other side, and there is usually marked salivation. The tonsils often swell, the breath is fetid, and the tongue heavily coated. The pyrexia disappears about the 4th or 5th day, and the parotid swelling rapidly subsides.

Orchitis is generally the result of allowing the patient to be out of bed too soon. One or both testicles may swell and become extremely painful. The enlargement gradually subsides in a few days, and may be accompanied by a urethritic discharge. The orchitis is generally secondary to the parotid swelling, but it may be primary, or even occur without any parotid swelling at all. It only occurs in patients who have reached puberty.

Mastitis affects girls much in the same way as orchitis does boys, but ovarian involvement is less common.

A considerable number of complications and sequelae may follow, such as acute otitis, bronchitis, arthritis, and so forth, but with the exception of the complications due to metastasis the others are rare.

Diagnosis.—There are cases of parotitis which are not due to mumps. After almost any continued fever, after an abdominal injury or operation, and along with many septic

conditions, the parotid glands may inflame and even suppurate. Notwithstanding this, the disease is extremely typical, with the marked swelling in front of the ear on one side, while the fact that an epidemic is present should prevent any mistake.

Prognosis. Recovery in a few days, but orchitis, should it complicate the case, may result in atrophy of the testicle.

Treatment. As a rule the patient should be confined to bed and especially if there be any temperature. Metastasis is generally the result of exposure to cold or wet, but it also may be due to the patient's going about too freely even indoors.

The diet should be mostly fluid, as mastication is painful: attention must be paid to the bowels, and a fever mixture is often of value. Local fomentations with opium and chamomile flowers is an old-fashioned but useful remedy. Sometimes leeching has been employed, but it is rarely necessary. In convalescence the usual tonics should be administered.

XIII. RELAPSING, OR FAMINE, FEVER

A CONTAGIOUS epidemic fever in which there is no rash, but which is characterised by the presence of a specific organism in the blood, and by peculiar relapses of the fever after apyretic intervals.

Etiology. Is caused by the Spirillum of Obermeier, and is associated with starvation and overcrowding. The disease is infectious to persons well-fed and well-housed. The spirillum, which is about 2 to 6 times the diameter of a red blood corpuscle, is actively motile, and is found in the blood in great numbers. It stains well by Löffler's methylene-blue stain, but has so far resisted all attempts at cultivation, although inoculation with the blood of a patient may produce the disease. The organisms appear in varying numbers from day to day during the attack, and disappear altogether in the apyretic intervals.

Pathological Anatomy. The blood contains the spirillum, which multiplies rapidly, and the spleen is generally enlarged but the disease is seldom fatal.

Clinical Features.— The stage of *Incubation* is 1 to 14 days.

The stage of *Incasion* commences suddenly, with rigors and pains in the forehead, back, and legs. The temperature rises rapidly (103 -105 F.), and there is often vomiting, jaundice, and enlargement, with tenderness, of liver and spleen. The face is red, the eyes congested, the tongue moist and thickly furred, while the skin is hot and burning. The pains increase, causing sleeplessness and frequently delirium. Suddenly between the 3rd and 7th days the stage of *Resolution* occurs by *crisis*, the temperature falling very rapidly and with profuse sweating. During the attack a leucocytosis is usually present.

After a period of apyrexia, during which the patient may feel perfectly well, in about a week's time a second attack begins. Probably a third and even further relapses may occur,

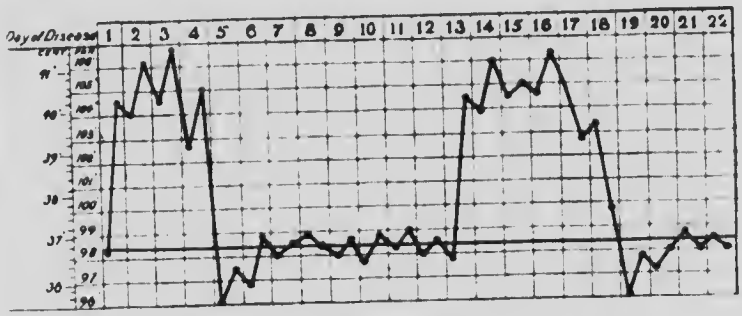


FIG. II. -Temperature Chart. Relapsing Fever. (After Murchison.)

with apyretic intervals in between. Usually the successive relapses do not last so long as the first attack, varying in duration from 2 to 5 days. The crisis in each case closely resembles that occurring in the initial attack. Pains in the joints, with, sometimes, effusions, and in severe cases pulmonary and other complications, may be present. Evanescent rashes occasionally appear, but are neither characteristic nor striking.

Diagnosis. -The recognition of the spirillum in the blood is the surest test, but the typical relapses should prevent any possible mistake, especially as the fever occurs in epidemic form.

Prognosis.—Generally recovery, except in old or debilitated persons.

Treatment. — Common-sense rules should guide the physician in the treatment of his patient; sponging the skin, attention to the diet, and the use of suitable remedies for vomiting, if excessive, and diarrhoea, if troublesome, are

generally sufficient. Quinine has been tried as a means of preventing the relapses, but without avail. Tonics should be administered during convalescence.

As a result of sanitary improvements, the disease is almost exterminated in our own country, and therefore little opportunity is now afforded of observing its clinical features.

XIV. SIMPLE CONTINUED FEVER

(Febricula)

A mild continued fever, probably due to many different causes, has to be described under this title. These cases of fever may be, *firstly*, mild, and indefinite attacks of one of the ordinary eruptive fevers, such as scarlet fever or measles; or, *secondly*, they may be the result of poisoning from sewer gas, or possibly from the ingestion of some toxic article of diet. In addition to these well-recognised types there are, *thirdly* cases of mild fever which follow a chill, generally disappearing in 1 to 2 days: and also, *fourthly*, cases of fever incidental to tropical countries, and perhaps due to exposure to the sun's rays. *Fifthly*, at the climacteric and in old persons mild attacks of fever without any definite cause are not uncommon.

The **Clinical Features** vary considerably, but rarely include pyrexia of more than 102° F., and the fever subsides in a few hours or days. A degree of malaise, headache, and loss of appetite are commonly present.

The **Diagnosis** is often important because of some infective fever being mistaken for this generally trivial ailment.

The **Prognosis** is favourable, and the **Treatment** consists in a simple diet and the usual febrile mixtures.

XV. INFLUENZA

(La Grippe: Epidemic Catarrhal Fever)

An infectious disease, due to a specific organism, and specially affecting the respiratory passages.

Etiology. Influenza has not infrequently followed attacks

of plague during the later centuries. The bacillus was discovered by Pfeiffer in 1892, and it is present in the respiratory mucus. It is highly contagious, is probably conveyed by the atmosphere, and causes widespread epidemics. The bacillus is very minute, ·5 μ in length and ·2 μ in breadth. It is non-motile, grows well in blood agar and is almost certainly the specific cause of influenza. While it spreads mostly by the air, it is quite probable that clothes and breath may communicate the disease to others. There is definite evidence that Pfeiffer's bacillus is not the only influenzal organism. It undoubtedly is the cause of true influenza, but there are other varieties of the disease which are closely allied as regards symptoms, but are dependent on different organisms.

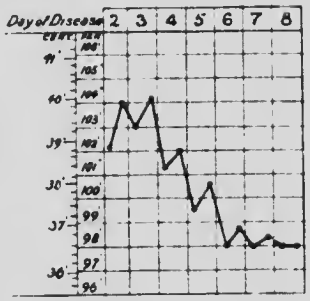


Fig. 12.—Temperature Chart. Influenza. Recovery.

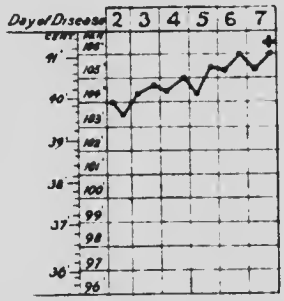


Fig. 13.—Temperature Chart. Influenza. Death from cardio-pulmonary complications.

Pathological Anatomy.—The post-mortem changes in influenza are largely due to complications, such as pneumonia, to which the attack of Pfeiffer's bacillus appears to render the patient specially susceptible. The heart is apt to suffer, undergoing fatty changes or sometimes atonic dilatation, while there is a distinct association between the ravages of the tubercle bacillus and influenza. In what might be termed uncomplicated cases, a catarrhal inflammation of the respiratory passages and gastro-intestinal tract is probably the only fairly constant lesion.

Clinical Features.—The stage of *Incubation* is 3 to 4 days, but possibly it may be a great deal shorter. The attack begins like an ordinary severe cold, with, however, more marked loss of taste and smell, often with bronchial catarrh and sore throat, with headache, malaise, considerable pyrexia, pains in the back and legs, and very generally gastro-intestinal

irritation. Certain patients suffer more in one way than another; some complain of photophobia, lachrymation, or earache, while others suffer from cough and bronchial catarrh, and others again from nausea and sickness, with sometimes diarrhœa. It is common to find severe neuralgia either associated with the disease itself or as a troublesome sequel to it.

Various types of the disease have been described, such as the *Respiratory* type, the *Gastro-intestinal* type, and types which indicate that the nervous system has specially suffered (*Nervous* type), or that the heart is seriously involved (*Cardiac* type).

Usually recovery occurs in a few days, but exposure to cold or wet is very likely to induce one or more of the following complications:—

Complications.—Pfeiffer's bacillus predisposes to the attack of Fränkel's pneumococcus, and lobar, or lobular, pneumonia is an extremely common complication. In not a few cases the physician finds the patient's lungs attacked by a virulent form of pulmonary tuberculosis.

Bradycardia (pulse-rate even 30-35 per minute) and cardiac failure, awful attacks of neuralgia, an excessive degree of prostration rendering all bodily or mental effort a serious tax, insanity, acute inflammation of the ear, and many other less common complications, may also be met with.

Diagnosis. Unfortunately it is difficult to find the bacillus of Pfeiffer, but the history of most cases gives a fairly sharp clinical picture. It should be remembered that there is rarely a leucocytosis in influenza, and therefore *pneumonia*, which usually is associated with an increase of these cells in the blood, is, if of influenzal origin, accompanied by even a leucopenia.

In the tropics *dengue* is not unfamiliar, and has frequently been called tropical influenza. The backache of *small-pox* is certainly not unlike the pain of influenza, but the eventual appearance of the rash and the history of an epidemic in the neighbourhood should prevent any confusion. A common *cold* is not attended by the extreme prostration which marks influenzal patients.

Prognosis. The death-rate from influenza itself is extremely small, only probably about 2 per cent, but it is

proportionately more fatal in infants and in old persons, and the complications are very serious. Not infrequently the constitution of the patient is undermined, and it may take a long time before the effects of the fever are finally thrown off.

Treatment.—For the relief of the attack probably there is nothing so satisfactory as to give the patient, after a preliminary hot bath or hot foot-bath, a large dose, or several repeated smaller doses, of one or other of the following drugs:—Antipyrine, quinine, salicylate of soda, antifebrin, or phenacetin. These are the best of a long list of somewhat similar remedies, and they speedily relieve the aching pains and bring down the temperature. At the same time plenty of nourishing but light food should be given, and where there is any suspicion that the heart is affected, cardiac tonics may be promptly administered. Alcohol, and in large doses when it can be safely taken by the patient, is of enormous value once the pyretic period is past. Where, however, it is undesirable to order the patient alcohol—strychnine, spirit of chloroform, and other stimulants and tonics should be freely exhibited.

The prostration and depression are often best alleviated by change of air, and, above all, by plenty of cheerful society.

PROPHYLAXIS.—During an influenzal epidemic many people are in the habit of taking ammoniated tincture of quinine in large doses, while eucalyptus and other volatile antiseptics may be smelt in the tramcar or the street. Certainly these remedies are advantageous, and relieve the anxiety of the persons who use them. A patient with influenza should be looked upon as infectious, and the attendants and all delicate persons should endeavour by generous diet to keep themselves well above par when the disease is prevalent.

XVI DENGUE

BREAK-BONE OR DANDY FEVER

An infectious fever, occurring in tropical and sub-tropical countries, associated with joint and muscular pains and typical eruptions.

Etiology. The specific organism of this disease is as yet

undiscovered, although the resemblance of dengue to influenza has suggested a close relationship between the two. It occurs during the hot weather, it spreads with great rapidity, attacking a large proportion of the inhabitants of any district, and, as with influenza, there is no protection against a second attack.

Pathological Anatomy.—The disease rarely endangers life, but joint effusions with pericarditis have been noted in a few cases which proved fatal.

Clinical Features.—The *Incubation* stage is uncertain; the stage of *Invasion* begins with great suddenness, and generally slight shivering is followed by high fever, great pains in the joints and muscles, and severe headache, while the eyes are congested, the pulse is rapid, and a diffuse erythematous rash appears all over the body. The joints specially involved are the knees, which become red and swollen, and the pains in the muscles and joints are increased by movement, thus giving rise to the terms "break-bone" and "dandy" fever.

In 1 to 3 days the fever subsides with sweating, but the pains in the joints persist somewhat, and after another period of 1 to 3 days' respite there is a rise of temperature, return of the pains, and a second rash, which may be measles-like or scarlatiniform, develops on the arms, legs, face, and trunk. After a few days it fades and desquamation ensues, sometimes accompanied by much itching and discomfort of the skin. The joints may remain stiff for some time, and occasionally the cervical glands enlarge.

Diagnosis.—The history of an epidemic, the two attacks of fever with apyretic interval and the two eruptions, should prevent any confusion with acute rheumatism; while in *scarlet fever* and *measles* the typical throat inflammation of the one and the absence of joint pains in the other, should render a mistake improbable. In *influenza*, eruptions are rare and catarrhal symptoms are much more common.

Prognosis.—The disease is rarely fatal, and recovery generally occurs in about a week to ten days from the period of onset.

Treatment.—Sponge the skin and administer analgesic remedies, and especially phenacetin, while the patient should be kept in bed and given the usual fever diet. Baths, electricity and massage do much for the relief of persistent

joint pains, while iodide of potash has been frequently commended.

XVII. ERYSIPELAS

An acute contagious inflammation of the skin, due to the Streptococcus erysipelatis (Fehleisen), which gives rise to a varying amount of constitutional disturbance.

Etiology. It is usually endemic, but epidemics are not unknown at certain seasons of the year. It is a disease of cold or temperate climates, and is most prevalent during the autumn and winter months. The *Traumatic* type is met with in those who have pre-existing skin lesions, and is apt to occur in surgical wards. The so-called *Idiopathic* form, which is much less common, is probably the result of insanitary dwellings. Persons who are extremely debilitated, alcoholic, or who are suffering from Bright's disease, and patients suffering from scarlet fever and small-pox, have a special predisposition. Certain individuals also appear to have a constitutional tendency to attacks. Infection is generally conveyed by direct contact.

Pathological Anatomy.—The specific organism closely resembles the Streptococcus pyogenes, but it differs inasmuch as it seldom produces suppuration, unless the organisms invade deeper tissues. The affected skin is inflamed, and specially so at the spreading margin where the streptococcus is present in large numbers. There is a large amount of inflammatory oedema with marked invasion by leucocytes. Blebs are not uncommon, but suppuration is probably due to secondary infection by suppurative organisms. The spleen is enlarged and congested, and infarcts are found in the lungs, spleen, and kidneys, where erysipelatous pyaemia has supervened. In this case the pleurae, pericardium, and even the endocardium may all be involved, and pneumonia and acute nephritis are not infrequent. Where the scalp is affected, the meninges may become inflamed, probably through the venous communications, and abscess formation is specially common in erysipelas in this region.

Clinical Features.—The stage of *Incubation* is 3 to 6 days. The stage of *Invasion* is ushered in by a rigor and rapid rise of temperature, and is accompanied by malaise,

often vomiting, headache, and constitutional disturbance. During the stage of *Advance* or *Eruption* the typical red, infiltrated area of skin appears round a pre-existing wound or abrasion. The area pits on pressure, and has a distinct rampart-like edge, and bullae may appear on the affected skin. The face is often the part affected, although any region of the body may be attacked. The temperature rises to about 104° F., with all the usual febrile phenomena. The lymphatic glands in the neighbourhood are often enlarged, and there is a marked leucocytosis, and the temperature after remaining fairly high for 3 to 5 or more days falls by *crisis*, sometimes by *lysis*. When the face is affected it often starts at the bridge of the nose, and the features may be almost unrecognisable from swelling near the eyes. The swelling may spread rapidly, always following the line of least resistance, and tending to be arrested where the subcutaneous tissues are not lax. Blebs sometimes form over the patches, and in cases where the inflammation is very intense, superficial necrosis may follow.

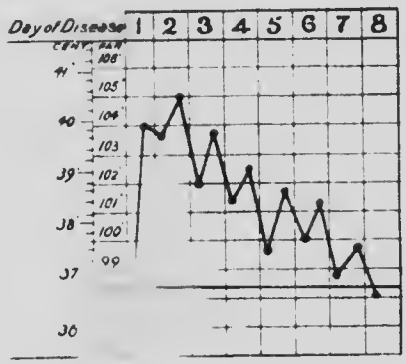


Fig. [illegible] Chart. Erysipelas [illegible]

[illegible] sleeplessness at night, which is specially severe in [illegible]. In alcoholic patients albuminuria is often present. When the mouth is attacked, the erysipelas may extend to the [illegible] with sudden dyspnoea and death, although this [illegible] are.

There is a [illegible] form of erysipelas called *Erysipelas migrans*, in which, as the name signifies, the rash spreads erratically over the body. It is a more chronic form, in which the temperature is very irregular.

Temporary alopecia is apt to follow erysipelas of the scalp. In certain persons repeated attacks occur, a pimple in the nose often being the exciting cause of a fresh outbreak.

Complications — These have mostly been mentioned already. Suppuration, pyaemia, meningitis, acute nephritis, and various pulmonary and cardiac complications are not

uncommon, and occasionally the involvement of the lymphatics may cause a condition resembling elephantiasis.

Diagnosis.—The rash is typical and characteristic, and can usually be diagnosed with certainty from *erythema nodosum*, *urticaria*, or *malignant pustule*.

The **Prognosis** is generally favourable, but likely to be fatal in debilitated or alcoholic subjects, in persons suffering from Bright's disease, and at the extremes of life.

Treatment.—*Firstly*, endeavour to keep up the patient's strength by giving a light but nutritious diet, and alcohol is sometimes of great value. *Secondly*, apply to the affected skin either an ointment containing ichthyol (zinc-ichthyol salve-muslin, Unna), or else, where possible, subacetate of lead lotion. Carbolic and boracic fomentations are also recommended. If an abscess develops it should be incised and evacuated at once. *Thirdly*, give internally full doses of tincture of the perchloride of iron (♏ 20 - 30). Some authorities condemn this treatment as useless, and certainly where the stomach is irritable it should not be persisted in, but we have generally ordered it and believe it to be of benefit. Where there is much delirium or excitement, order chloral (gr. 20-30) or opium (gr. $\frac{1}{3}$-1), and insomnia must be combated.

In our experience anti-streptococcic serum administered by the rectum in 1 to 2 c.c., repeated every 2 or 3 hours, rapidly reduces the temperature, and appears to counteract both the local and general effects of the streptococcus.

XVIII. ACUTE PNEUMONIA

(Croupous Pneumonia; Lobar Pneumonia

This is a continued fever, due to a specific micro-organism, in which there is a characteristic inflammation of the lungs, and often a widespread infection of other organs.

Etiology.—Fränkel's pneumococcus, or the Micrococcus lanceolatus, a diplococcus with a definite capsule, was recognised in 1884. It may be stained with carbol-fuchsin or gentian violet. There are certainly several strains of this diplococcus which appear to have different degrees of infectiveness and

virulence. The organism is found in the sputum, in the affected lung, in the blood, and in the pleuritic exudate, while it is present in connection with the pericarditis, endocarditis, peritonitis, meningitis, and the synovitis sometimes associated with this specific fever. It has been found in the saliva of healthy individuals.

Friedlander's pneumobacillus has also been described. It may be associated with the pneumococcus, but is more often found in broncho-pneumonias. It is also capsulated, but shows totally different characteristics from the pneumococcus when cultivated.

The Streptococcus pyogenes and various staphylococci are also present in some cases; mixed infections are not uncommon.

Pneumonia is more frequent in spring and winter, and seems to depend on exposure to cold and damp. Pneumonia may occur in epidemics varying greatly in virulence. Men naturally suffer more than women; and as regards age, from birth to the 10th year, and again from the 25th up to the end of life, pneumonia is of common occurrence. There are certain predisposing conditions, and pneumonia especially follows influenza or other fevers, and attacks persons who are debilitated and alcoholic. It may result from an injury to the chest, and it is apt to be the terminal stage in diabetes mellitus, chronic Bright's disease, and various grave nervous diseases, although in some of these cases the disease is really a broncho-pneumonia and not specific croupous pneumonia.

One attack predisposes to another.

Pathological Anatomy.—The distribution is generally lobar as regards the lungs, but a whole lung may be involved, the affection often beginning at the base and extending upwards. It may be double, and is more often basal than apical. The right lung suffers more frequently than the left. Sometimes a lobular distribution occurs, but the lobar type is the usual one. Occasionally it begins in the centre of a lobe gradually extending towards the pleura.

Four stages are described: (1) *Congestion*, which lasts probably 24 to 36 hours; towards its termination exudation begins. (2) *Red hepatisation* lasting 2 to 4 days, in which the affected part of the lung becomes solid, and on section resembles red granite. The air vesicles contain a firm, fibrinous

coagulum in which red blood corpuscles and degenerated endothelial cells are found. (3) *Grey hepatisation*, resembling grey granite, in which the lung becomes less vascular, and the coagula, which begin to separate, are invaded by numerous leucocytes. The lung is more friable, and some pathologists imply by the term grey hepatisation the stage in which breaking down of the lung is about to occur and not a stage preceding resolution. In both red and grey hepatisation the pleura is involved, and a varying amount of exudate may be present. 4. *Resolution*. In a favourable case the lung clears up, mostly by absorption (digestion by enzymes), and partly by expectoration. This stage may be safely completed in 6 to 9 or 11 days. But in unfavourable cases the lung breaks down, and the cut surface on scraping exudes a thick muco-purulent material. Actual abscess-formation may ensue, and even gangrene is possible.

In the affected lung pneumococci are present in great numbers. The bronchial glands are generally enlarged, and these may soften and break down in fatal cases. In many cases after death extensive clotting is found to have occurred in the right heart and extending into the pulmonary arteries. This clot is very firm, generally adherent, and is due to the great excess of fibrin in the blood. Purulent meningitis, pericarditis, endocarditis, and peritonitis may be present.

Clinical Features. The *Incubation stage* varies, but is probably about 1 to 2 days. *Invasion* begins suddenly, and there is usually a rigor followed by a continued high temperature, and often accompanied by severe pain in the side. The patient lies on his back or on the affected side. There is a marked malar flush, the eyes are bright, the face anxious, and the alae nasi work vigorously. There is marked dyspnoea and the breathing is shallow. The temperature is usually 103 to 104° F., and remains high, although it may swing gently with a slight rise at night, until *Resolution* is reached, when it falls very suddenly by *crisis*, often within a few hours, to normal. Occasionally the temperature falls more gradually or by *lysis*, and especially where the disease runs a longer course. In some patients a pseudo-crisis occurs about the 6th or 7th day, but the temperature rarely falls to normal, and usually rises again to its former level within a few hours.

Respiratory System. As a rule there is pain, and the

respirations may rise to 40 or 60 per minute, the ratio to the pulse-rate often reaching 1 to 2. Cough is painful and mostly ineffectual, although a little very viscid, rusty (blood-stained) sputum is with difficulty brought up. Later the sputum increases in amount. The amount of blood varies greatly and may be very considerable, suggesting a graver type of the disease. The sputum contains an excess of chlorides. It may become watery from oedema, and when the prune-juice character is assumed, the significance is grave. The organisms are usually found in great numbers in the sputum.

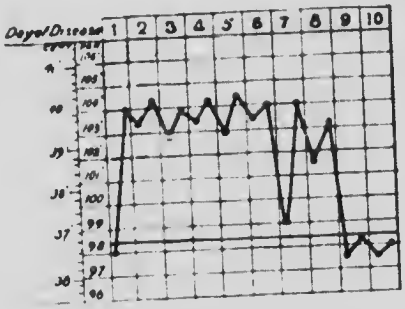


FIG. 15.—Temperature Chart. Croupous Pneumonia showing pseudo-crisis, later crisis, and eventual recovery.

Physical Signs.—During the stage of congestion the note is not so dull and the breathing is rather distant than of a definitely bronchial character. The physical signs of the consolidation stage are typical as a rule. *Inspection* shows the limited movement especially of the affected side, and on *Palpation* the increased vocal fremitus is easily recognised. On *Percussion* the note is dull over the consolidated area, but in the neighbourhood a tympanitic or Skodaic note (from relaxed lung) is often to be made out. *Auscultation* yields typical tubular breathing with, at first, very fine crepitations: these may disappear for a time, and later they reappear and become gradually coarser and bubbling (*crepitation redux*). The crepitations have a sharp, clicking character. Very fine crepitations are limited to the first stage, and are thought to be due to the cohesion and subsequent separation of the alveolar walls. The vocal resonance is increased, and may be aegophonic in parts. All the auscultatory phenomena are temporarily modified should mucus block the bronchus to the affected lung. Pleuritic friction is often made out, and there may be evidence of a more or less extensive pleural effusion which may become an empyema.

Alimentary System.—The tongue is furred, often dry, and the edges red. Digestion is disturbed, and vomiting may occur. There is always malaise. Occasionally the pain of

pneumonia is referred to the abdomen, and its presence there may prevent attention being paid to the lung.

Circulatory System.—The pulse is full and bounding in strong patients during the earlier stages, but later it may become weak and irregular. Its rate is usually 100 to 120, depending much on the age and constitution of the patient. The first sound in the mitral area should be carefully auscultated; it early becomes less clear, and is replaced by a systolic murmur if the heart dilates. As a rule the maximum blood-pressure falls 15 to 20 mm. of mercury. A fall of 30 or 40 mm., especially if it occurs suddenly, is of grave significance and demands the prompt exhibition of cardiac and general stimulants. Special attention should be paid to the veins in the neck. Their distension suggests that coagulation of blood may be occurring in the right heart.

Hæmopoietic System.—The blood tends to clot, as above implied, because it contains an excess of fibrin, and there may be 10 parts instead of the normal 4 per 1000. There is usually a leucocytosis (mostly polymorphonuclear) reaching 20,000 to 40,000 per cubic millimetre of blood. If the leucocytosis is excessive, it is a bad sign, because it may mean breaking down of the lung; and if slight or absent, it indicates the inability of the individual to cope with the organisms present.

Integumentary System.—A burning skin is present during the fever, and copious sweating accompanies the crisis. Herpes labialis is extremely common about the 2nd day. It is said, but without much reason, that its appearance is a favourable sign.

Urinary System.—The urine is high coloured, with a dense precipitate of urates; the chlorides are diminished or absent. There may be slight albuminuria.

Nervous System.—In children headache and even convulsions are common. Delirium is frequently noted, especially in alcoholic cases, and is very wild where hyperpyrexia ensues. Meningitis is often present in fatal cases, and may produce definite and suggestive phenomena.

Locomotory System.—The joints are occasionally attacked by the pneumococcus.

There are many types of lobar pneumonia differing in a remarkable way from the description given above. There are

MICROCOPY RESOLUTION TEST CHART

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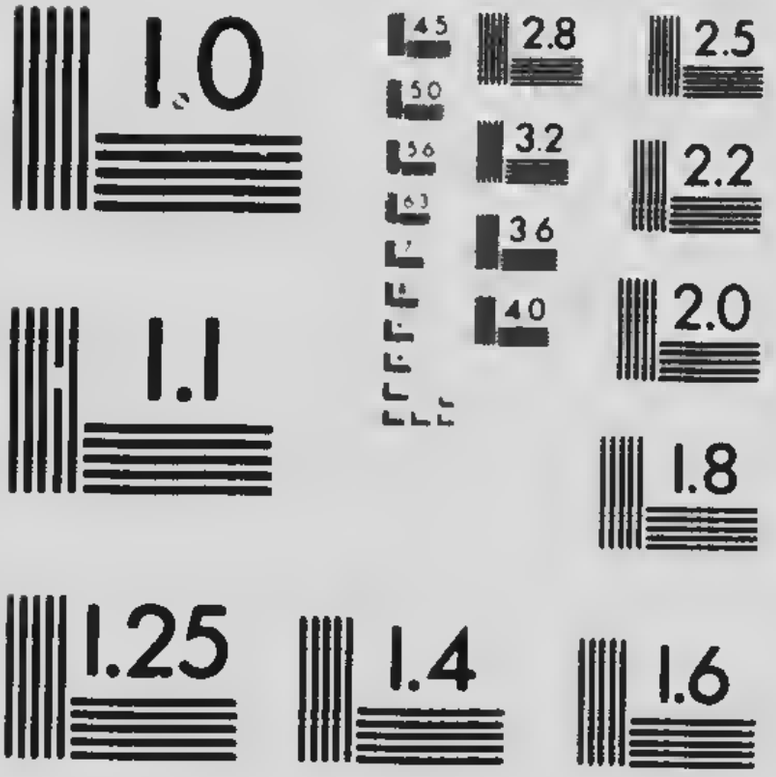

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Latent cases with little temperature: *Migrating* cases in which consolidation develops with extraordinary rapidity in one lobe, and as rapidly disappears, while another lobe becomes involved. In *Apical* pneumonias there are often marked cerebral symptoms, and in *Massive* pneumonias the exudation is very extensive, generally filling the bronchial tubes as well as the air vesicles, while the greater part of a whole lung is invaded.

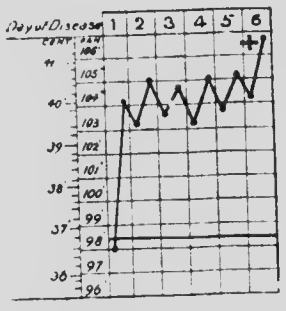


Fig. 16. Temperature Chart. Croupous Pneumonia, with death from hyperpyrexia on 6th day.

Complications and Sequelae.—It has been stated that an extensive pleural effusion, which may be purulent, sometimes occurs, and that changes may ensue in the affected part of the lung, where resolution does not take place. The physical signs of a pneumococcal effusion are sometimes very contradictory, and the introduction of an exploring needle may be required in order to confirm the diagnosis. The pneumonic lung may form an abscess, or gangrene may develop, due sometimes to thrombosis of the branch of the pulmonary artery supplying that area. Fibroid changes may also follow in pneumonic areas which do not clear up, and in certain instances the damaged lung becomes infected with tubercle. Reference might also be made to ulcerative endocarditis, peritonitis, enteritis, suppuration of the parotid gland, nephritis, and even neuritis, all due to the pneumococcus.

Diagnosis.—The definite physical signs characteristic of pneumonic consolidation with the discovery of the pneumococcus in the sputum are distinctive, but in the early stage, or where the consolidation is deep-seated, the differential diagnosis between pneumonia, *typhoid fever, acute tuberculosis*, and *meningitis* may be difficult. The Widal reaction in typhoid, the discovery of the tubercle bacillus, with the peculiar swinging or hectic temperature in cases of tuberculosis, are generally sufficient to decide the diagnosis in most cases. Meningitis early affects the cranial nerves, and where it is not simply a part of the pneumonic fever, a few days suffice as a rule to make the diagnosis definite.

Prognosis.—The death-rate varies 15-40 per cent

according to the virulence of the organism. The disease is more serious at the extremes of life, in alcoholic subjects, in delicate persons, and in those cases where the pneumonic attack is a final stage in some other serious disease, such as diabetes mellitus, Bright's disease, and cancer.

The following phenomena are of grave import in a case of croupous pneumonia:—a weakening heart with very rapid dicrotic pulse, hyperpyrexia, a scanty leucocytosis, and probably also an excessive leucocytosis, a rapid spread of the consolidation in the same or to the other lung, and the development of oedema or supervention of gangrene.

Treatment.—In the case of a strong patient no medicinal treatment may be required, and only the usual care with regard to food is needed, such as every severe infective fever necessitates, combined with attentive nursing.

Never let the patient sit up: and, if indicated, prescribe strophanthus or digitalis for the heart. Diffusible stimulants and especially alcohol, are often given just at or after the crisis, and ammonia is believed to hinder the tendency to blood-clotting which is apt to be present in this disease. Locally much relief may be afforded by one of two methods of treatment: (1) In strong patients, where the fever runs high, apply ice or a cold-water coil (Leiter's tubes) over the affected lung. (2) For less robust individuals moist heat in the form of a linseed poultice, with or without mustard, is much more helpful, and the pleuritic pain, with the ineffective and harassing cough, is greatly relieved by this method of treatment. It is also well in such cases to use a steam bronchitis kettle to moisten the air which the patient breathes. Oxygen inhalations greatly aid the physician in combating the disease.

The usual cough, diuretic, and febrifuge remedies may be administered, but antipyretics, such as antipyrine and phenacetin, should not be given freely, as they mask the true height of the fever and are very depressing to the heart. It is much wiser to control the temperature by sponging the patient, and, if there is hyperpyrexia, by wrapping him up in the sheet wrung out of cold or iced water, or using the cold-bath methods described fully on page 6, under the treatment of fevers generally.

Several curative sera have been recommended, but the success of the serum therapy of pneumonia is not yet assured.

Lately a vaccine consisting of 50 to 200 millions of sterilised pneumococci has been administered hypodermically with the object of expediting the crisis, and with some hopeful results.

During recovery, or where resolution is delayed, counter-irritate with iodine, and remember not merely to give tonics, but to order pulmonary gymnastics to help to re-expand the affected area of lung.

XIX. RHEUMATIC FEVER

(Acute Rheumatism)

An acute specific disease, characterised usually by joint swellings, sometimes by inflammation of fibrous tissues, and not infrequently accompanied by inflammation of the endo- or pericardium.

Etiology.—Acute rheumatism occurs most frequently during early adult life. More common in males than in females, it may occur at all ages and in both sexes. It is probable that its greater frequency in males is dependent upon exposure to cold and wet associated with occupations. There seems to be little doubt that a specific organism is present, and probably the diplococcus described by Poynton and Paine, and found in the blood and the synovial fluid of affected joints, is the pathogenic organism of rheumatic fever. Inoculation with this organism has produced endocarditis in animals with joint swelling. But inoculation with the specific organism is only one factor in the etiology. The predisposing causes of rheumatic fever are of great importance, and exposure to cold or wet, sudden changes of temperature, especially in connection with such employments as those of bakers, ironworkers, etc., may all act in this way. The disease is more frequent in damp climates, and is commoner in autumn and whenever the weather becomes wet and cold.

Much discussion has arisen as to the hereditary element in the etiology. There is certainly much to be said in favour of a rheumatic family history having, at all events, an important predisposing influence towards the disease.

A chemical theory for the production of the rheumatic fever has also been brought forward, and it was thought that

lactic acid might be the agent to which the pain and joint inflammation were due; but there is no evidence that any such chemical changes in metabolism have other than a subservient position.

Still a third theory, that the disease is one involving the trophic nerves, has little to commend it.

In conclusion, it seems probable that a specific organism is really present, and recent bacteriological research appears to support this view.

Pathological Anatomy.—There is congestion of the synovial membranes of the affected joints, with exudation of a synovial fluid, which may be clear or turbid, and which contains some leucocytes and an excess of fibrin. Only in the most

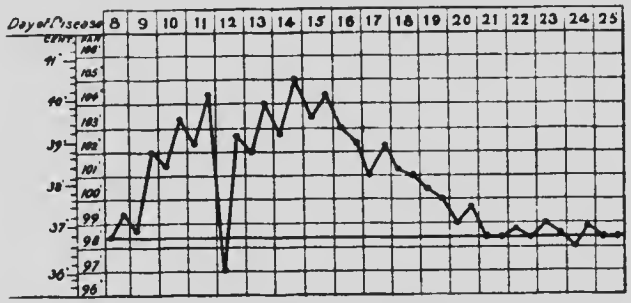


FIG. 17.—Temperature Chart. Rheumatic Fever, showing threatening of hyperpyrexia checked by cold bath. Recovery.

exceptional cases do the joints suppurate. The blood generally reveals a diminution in the number of red blood corpuscles, a slight increase of leucocytes, and an excessive amount of fibrin. The heart muscle is sometimes affected, probably as the result of the toxin, and this occurs apart altogether from the typical changes which are met with at the orifices in endocarditis. Pericarditis is also of frequent occurrence.

Clinical Features.—The patient begins to suffer from malaise, sometimes tonsillitis, and at an early period from pains especially affecting the joints of the arms and legs. The temperature rises to 102° or 103° F. or more, and usually there is a fairly definite morning fall. The pulse is bounding, the tongue moist and coated with white fur, while there is loss of appetite and great thirst. The joints most frequently affected are the knees, elbows, ankles, wrists, hands, and

shoulders; more rarely the hips, toes, and other joints suffer. The joints are swollen, the skin being reddened, and the effusion, while mainly in the joint cavity, also affects neighbouring tendon sheaths and fibrous tissues. The pain soon becomes extremely severe, absolutely preventing the slightest movement, and in a most remarkable way it may change its position from one set of joints to another—joints previously affected being practically free from pain, and the swelling also subsiding with great rapidity. Another typical feature is the profuse sweating which occurs all over the body. The peculiarly sour odour, unquestionably due to the presence of fermentation in the sweat, originated the idea that lactic acid might be the cause of the disease, but however profuse the perspiration may be, the acidity is due to accidental causes. Sudamina or clear vesicles from blockage of the orifices of the sweat-glands are not infrequently present over the trunk and especially the chest, and in certain instances some local irritation may arise round these sudamina.

Complications.—In the *Alimentary System* tonsillitis, as already noted, is by no means uncommon, while gastric disturbance and constipation are generally present. But the *Circulatory System* demands the most careful attention. Endocarditis most frequently involves the mitral, sometimes the aortic orifice, and from day to day the heart sounds should be studied with the utmost care. Increase in the rapidity of the heart's action, with perhaps a further rise of temperature, and very commonly palpitation, indicates in many cases that the endocardium is damaged; whilst the slightest change in the character of the heart-sounds, even before a definite murmur develops, is important presumptive evidence in favour of commencing endocarditis. Pericarditis is also common, and the myocardium may suffer either from myocarditis (sometimes associated with endo- or pericarditis), or as a result of atony, or from fatty degeneration. Endocarditis may be looked for in 50 per cent of cases of acute rheumatism, pericarditis in about 5 to 10 per cent. In the *Urinary System* diminution in the amount of urine and an excess of urates are features common to any febrile condition; the chlorides are usually diminished.

There are certain complications other than those involving the heart, which are often intimately associated with rheumatic

fever. In the *Respiratory System* pleurisy and bronchitis may be present; in the *Integumentary System* purpura, erythema nodosum, and eczema are by no means uncommon; while in the *Nervous System* there is a close association between chorea and rheumatism, especially in children. We sometimes find that the rheumatic pains disappear and well-marked choreic movements take their place; and it has been suggested, although it is by no means proved, that endocarditis being common in both diseases may explain this association, probably by causing small emboli. Hyperpyrexia is a very serious complication not infrequently coincident with the appearance of pericarditis, the temperature rising to 107-108 F. or more. It is accompanied by marked delirium and great restlessness, and, if it is not promptly checked, the patient soon passes into a stage of stupor ending in death. It has been called *cerebral rheumatism*, perhaps because with its appearance the joint pains markedly diminish.

Rheumatic nodules, which are small-shot or pea-like, subcutaneous, fibrous tumours, are found in the fascia or attached to tendons. They are certainly most common in children, and are met with on the fingers, hands and wrists, elbows, knees, spines of vertebrae, scapulae, back of the head, and more rarely elsewhere. They are composed of round and spindle cells; as a rule they are not painful to the touch, and while also met with in the less acute types of rheumatism, they bear a definite, although not very explicable, relationship to endocarditis.

An attack of acute rheumatism usually subsides in about 10 to 20 days if untreated, but there is great risk of relapse, and endocarditis may develop during the primary attack or during a relapse.

Diagnosis.—It is almost impossible to mistake *gout* and *rheumatoid arthritis* for acute rheumatism, provided sufficient care is taken. Occasionally an acute rheumatoid arthritis simulates acute rheumatism, but the protracted nature of the disease soon clears up the initial uncertainty. There is a close resemblance, however, between *osteomyelitis* and *pyaemia* and this disease; but as a rule the bone inflammation in osteomyelitis is associated with a very marked leucocytosis, and to begin with the disease is more localised than acute rheumatism, generally to the lower end of the femur or tibia and with

local oedema and tenderness not over a joint, while the migratory pains of acute rheumatism prevent confusion with the arthritis of pyaemia.

Prognosis.—Death is apt to be due to one of the complications so closely related to the disease, such as endocarditis, myocarditis, pericarditis, or hyperpyrexia, while in uncomplicated cases recovery is usual. The risk of a relapse must never be forgotten, and the damage to the heart resulting from the complications just mentioned may greatly interfere with the life and usefulness of the patient.

Treatment. The patient should be placed between blankets and clad in a flannel night-dress—these precautions being necessary to prevent chill from the excessive perspiration. The night-dress should be slit down the front and sleeves, so that it can be easily changed, when damp, without disturbing the patient. The bed should be soft, but not a spring mattress, the movements of which would greatly increase the agony of the patient. Absolute rest is essential, and the diet should consist almost exclusively of milk and occasionally plain soups, while plenty of alkaline mineral waters can be given to quench the thirst.

The proper medicinal treatment is much disputed.

(1) We may give the salicyl compounds, generally administering the sodium salicylate in 20-grain doses 3 or 4 times daily, and often in combination with sodium bicarbonate in 10-grain doses. Salol (gr. 15-30) or salicin (gr. 20) may be given in place of the salicylate. With this treatment the pain usually rapidly abates and endocarditis is possibly prevented, but the treatment is depressing, and a trifling erythematous eruption sometimes develops in persons who have a peculiar idiosyncrasy to the drug. Much may be done to prevent any unpleasant results by clearing out the bowels before administering the drug, but in cases in which the heart becomes markedly depressed it is wise to alter the treatment. (2) Alkalies [illegible] to be administered for the relief of rheumatism in the [illegible] of that the disease was due to some acid poison, and much benefit is certainly obtained, as already indicated, from the combination of an alkali with a salicyl compound. For children alkalies with salicin are most suitable.

Locally the joints may be treated (1) by wrapping up in cotton wool, or (2) by applying fomentations or poultices of a

soothing nature. A useful application consists in smearing the affected joints with salicylate of methyl, combined with equal parts of vaseline and lanoline, and covering over the joint with gutta-percha tissue in order to prevent the annoyance due to the penetrating odour of the drug. (3) Iodine may be painted on, either the ordinary tincture, the Edinburgh tincture, or equal parts of tincture and liniment. Counter-irritating liniments, or even blisters, may also be applied.

Nothing relieves the excessive pain of acute rheumatism so satisfactorily as a dose of Dover's powder, while phenacetin or kindred remedies may be cautiously administered.

Hyperpyrexia should be promptly treated; cold sponging, the application of the wet pack or the cold bath, must be resorted to without delay in order to save life.

In cases of endocarditis never give cardiac tonics unless when absolutely necessary; for serious affections of the heart or pericardium diffusible stimulants are more desirable, although they too should be given with caution.

Rheumatic fever invariably leaves the patient anaemic and prostrate, so that iron and general tonics, careful dieting, and the suitable clothing of the patient should all receive the attention of the physician.

Dr. Caton has recommended the application of small blisters along the course of the 3rd, 4th, 5th, and 6th dorsal nerves on the left side, in order to obviate or retard cardiac involvement, the patient being kept in bed for a period of six weeks.

XX. EPIDEMIC CEREBRO-SPINAL MENINGITIS

(Spotted Fever)

An acute febrile disease, characterised by nervous phenomena which indicate an inflammation of the cerebro-spinal meninges. This disease has attracted much attention lately owing to recent epidemics in America and in this country, and our knowledge of it has been much added to.

Etiology.—The Diplococcus intracellularis meningitidis of Weichselbaum is the specific agent. It resembles a small-sized gonococcus, and is found both in pus cells and also free in the

cerebro-spinal fluid. It is somewhat difficult to grow, and soon dies out if fresh cultures are not constantly made. The naso-pharynx is almost certainly a common habitat for the organism, and from there, in some way not at present very clear, it may and can infect the meninges. The disease occurs in epidemics as well as appearing sporadically and endemically. Children appear to be more susceptible than adults, and it is apt to occur during cold weather. Overcrowding seems to be an important factor in promoting epidemics.

Pathological Anatomy.—In acute cases a thick purulent exudate into the meninges of the brain and cord is present, with small haemorrhages in the cortex, congestion of many organs, and sometimes ecchymoses into serous membranes. The base of the brain and in particular the under surface of the pons and cerebellum are specially affected with the exudate.

Clinical Features.—There may be premonitory headache or malaise, but generally the sudden severe occipital headache, with rigors and pains in back and limbs, indicates the commencement of the fever. The head and neck are rigid and bent backward: there is great pain along the line of the spinal and cranial nerves, and often hyperaesthesia: convulsions, and finally coma and death, generally with a high temperature, terminate the scene. Kernig's sign (see p. 654) is usually present. Delirium is a common clinical feature, and may be of a wild type. Various skin eruptions, sometimes purpuric, sometimes herpetic, and sometimes erythematous, may appear. The purpuric spots, which vary in size, are more marked on the lower part of the trunk and limbs. Herpes on the lips is almost constantly seen in adult cases. There is occasionally optic neuritis, while there may be conjunctivitis, photophobia, and sometimes paralysis of various cranial nerves. Cerebral vomiting is extremely common, and is an early symptom. A marked leucocytosis (25,000-40,000 per c.mm.) is present. There is sometimes albuminuria.

Various types have been described, depending on the malignancy of the fever or on its intermittent or other characteristics.

It is not uncommon to find a case either chronic from the outset or which passes into a chronic stage. The patient emaciates rapidly, temperature and pulse vary from time to time

and the headache is sometimes extremely severe and sometimes very slight. The rigidity of neck muscles persists, and Kernig's sign should be easily elicited. Cerebral vomiting may also persist along with twitchings of muscles and even convulsions. There is apt to be considerable involvement of the cranial nerves, with, in particular, blindness, deafness, and spasm or paralysis of ocular muscles. Bed-sores at times develop, and the patient may gradually sink. Several relapses may occur in cases which appear to be on the way to recovery, and should recovery occur, there may be permanent damage to sight, hearing, etc.

The **Sequelae** are the results of interference with nerve centres and tracts, and include deafness, impaired vision, etc., while **Complications** such as pneumonia and pleurisy are not infrequent.

Diagnosis.—Lumbar puncture yields the most accurate information. The disease resembles *typhus*, especially as regards the rash, and *tubercular meningitis* from the nervous symptoms. The diplococcus is agglutinated by the blood serum of a patient suffering from epidemic cerebro-spinal meningitis.

Prognosis.—May be rapidly fatal; epidemics vary in mortality from 30 to 70 per cent.

Treatment.—The patient should be isolated in a dark, well-ventilated room, but otherwise the treatment is the same as for an ordinary meningitis. The hair should be shaved and ice-bags applied to the head, the nape of the neck, and even down the line of the spinal cord. Blisters are sometimes useful, and internal sedatives, such as chloral, are of great value for soothing pain and excitement. Perhaps no treatment affords so much relief as the hot bath. Flexner's serum has been used with marked success in Edinburgh by Dr. Claude Ker. The serum (15-30 c.c.) obtained from the horse is injected into the spinal canal after an equivalent amount of cerebro-spinal fluid has been removed. The dose may later be increased to even 300 c.c. Potassium iodide has also been recommended. It is desirable to disinfect with a suitable antiseptic the naso-pharynx of the patient and of all contacts.

XXI. 1. SAPRAEMIA 2. SEPTICAEMIA 3. PYAEMIA

However clear to the mind of the pathologist these terms may be, it is desirable that each should be carefully defined for the purposes of accuracy.

1. Sapraemia

This implies the presence of toxins, but no organisms, in the general circulation, the organisms being entirely limited to some local site, as, for example, in tetanus and in many cases where there is a local focus of inflammation due to some suppurative or pathogenic organism. The poison may act with excessive power, not merely locally, but throughout the whole body, even although the manufacture of the poison is limited to a local site, and it is possible that an organism which may produce a sapraemia at one time, may cause a septicaemia or a pyaemia under condi- which must be different, although the exact nature of these differences is unknown.

The **Clinical Features** of sapraemia are chill and rigor, malaise, headache, thirst, sickness, and the result may be fatal if the poison be of a virulent nature.

2. Septicaemia

This implies the presence of organisms, as well as their toxins, in the blood-stream. There is thus a more widespread condition with many foci where toxins can be manufactured. The common organisms are the Staphylococcus pyogenes albus and aureus, the Streptococcus pyogenes, the Pneumococcus and the Bacillus pyocyaneus. There may be a local wound, such as a post-mortem wound, with inflammation extending up the lymphatics of the arm, or there may be no definite site of any primary invasion. There are enlarged lymphatic glands in the one case: in the other there are no local phenomena to be noted.

Clinical Features.—There is greater malaise than in sapraemia, usually a higher temperature, which is sometimes hectic in character, a quicker pulse, and very generally marked

gastro-intestinal disturbance. There is often a leucocytosis, the spleen is enlarged, and we ought to obtain by culture the specific organism from the blood, although it may only be isolated with difficulty.

3. Pyaemia

This implies not merely the presence of organisms in the blood but large metastatic foci of organismal inflammation in different parts of the body. There may be pyaemic emboli in the lung, kidneys, spleen, brain, etc. The organisms may be the same as in septicaemia, and very frequently there is a septic wound, a phlebitis, a sloughing uterine wall in the puerperium, a carbuncle, an empyema, malignant endocarditis,

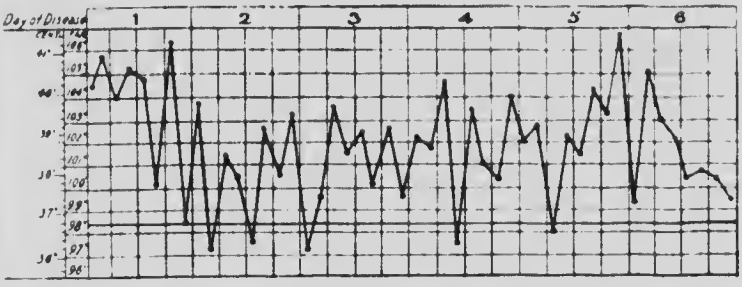


Fig. 18.—Temperature Chart. Pyaemia, showing the great variations of temperature.

an osteomyelitis, middle-ear disease, or an ulcer in the intestine as a starting-point.

The organisms may spread by the lymphatics, by direct extension along bile-ducts, ureter, etc., or by infective emboli by the blood-stream.

Clinical Features. Very similar to the last, but only more severe, and there is local pain where the septic emboli lodge, as for example in the spleen, kidneys, and lungs. The temperature is hectic, with marked rigors and sweating. There is malaise, there is always vomiting and diarrhoea, the appetite is lost, the tongue dry, and delirium precedes final prostration and collapse. Occasionally an erythematous rash is present, there should be a leucocytosis, and the spleen is enlarged.

The evidence of pyaemic emboli is the following: albumin and blood in the urine, with sometimes local pain over the kidney, pain and perisplenitis in connection with the spleen, and the signs of broncho-pneumonia and pleurisy in the lung,

while in cases where the endocardium is involved, fresh murmurs may develop.

It is rare to find the patient survive more than 7 to 10 days.

Diagnosis.—*Typhoid fever, acute miliary tuberculosis,* and *malarial fever* all somewhat resemble pyaemia; but Widal's reaction in typhoid, the examination of the blood, the benefit obtained from quinine in malaria, and the fact that in pyaemia a culture should be obtainable from the blood, ought to assist in clearing up the diagnosis. There will probably also be a leucocytosis in pyaemia (although this may not occur), and the spleen is usually enlarged. In all cases of pyaemia we should try to find the presumptive cause of the condition.

The **Prognosis** is essentially bad, and there is little hope of cure.

The **Treatment** of these three conditions—sapraemia, septicaemia, and pyaemia—is often most unsatisfactory. Where pus is present it should always be evacuated. In milder cases we endeavour to keep up the patient's strength, and iron in large doses and quinine have been given with apparent benefit in some instances. It is certainly worth trying anti-streptococcic serum, which, if it does no good, will at all events do no harm.

Terminal Septicaemia

Closely allied to the conditions above described is the infection by organisms—sometimes local, sometimes general—found not infrequently in the terminal stages of certain diseases, and especially Bright's disease, heart disease, leucocythaemia, Hodgkin's disease, chronic tuberculosis, cirrhosis of the liver, and pneumonia. Thus the pleura, pericardium, peritoneum, and meninges may be affected in chronic Bright's disease, heart disease, etc., while the parotid gland sometimes suppurates in pneumonia, and an enteritis occasionally is the cause of death in many of the conditions just indicated. The attack of the organism occurs when the individual is much below par, and there is probably some special reason for the particular site selected, although the reason is by no means always clear. The organisms present differ considerably, but appear to be not infrequently the ordinary suppurative microbes.

XXII. CHOLERA ASIATICA

An infectious epidemic fever, due to a specific organism and characterised by vomiting and purging, muscular cramps, and suppression of urine. It causes an extremely high death-rate.

Etiology.—The comma bacillus of Koch was discovered in 1884. It is a minute, actively motile spirillum 1·5 to 2 μ in length by ·5 μ in breadth, and several flagella of great length may sometimes be seen by special staining methods. The b. lus grows most actively between 30 and 40 C., and is arrested by a temperature of 15 C. on the one hand and about 45 C. on the other. The organism produces in animals a disease similar to Asiatic cholera, and it is found in large numbers in the stools of affected persons. It is not communicable by the air, but is usually propagated by water or milk.

The disease has been endemic in India for centuries; it has frequently spread to Europe, and this country has been visited by more than one severe epidemic. In the last great epidemic in Hamburg there were 18,000 cases (with 40 per cent deaths), due, it was believed, to the fact that the Elbe water, contaminated by sewage, was the main drinking-supply; while in the large suburb of Altona, where a better filtering system had been adopted, there were only 500 cases.

The disease has no respect for sex or age. It is certainly more common in tropical countries and during the warm season. As in many other fevers, drunkards present a much higher death-rate, and there is a current belief that the disease may be acquired to some extent by fear.

Pathological Anatomy.—Rigor mortis produces extraordinary effects after death. It comes on early, and often causes the limbs of the corpse to assume strange attitudes. A good deal depends on the stage at which death has occurred: but there is usually intense congestion of the intestinal tract, the bowel being full of rice-water-like material, often unstained by bile, and Peyer's patches are swollen. There is cloudy swelling in the kidneys, liver, etc., while during the stage of reaction in fatal cases there may be inflammations of pleura, lungs, pericardium, kidneys and other organs. The bladder is usually empty.

Clinical Features.—The stage of *Incubation* is 2 to 5 days. The attack itself may be divided into three stages.

In the *First Stage*, diarrhoea, becoming more and more excessive, is the first symptom, while vomiting, colicky pain, and headache usually begin later; but there is rarely much fever. Towards the end of this stage the stools become rice-water-like in appearance, alkaline in reaction, have a specific gravity of 1006 to 1010, and contain sodium chloride, albumin, and mucus, and of course numerous organisms. Borborygmi are frequent, and epigastric pain, and intense cramps occur in the legs, especially in the calves, and eventually may involve all the muscles.

The *Second Stage* begins in 2 or 3 hours, the diarrhoea is much worse, and vomiting of the rice-water-like material commences. The pain is greater, the thirst more intense, and the patient becomes rapidly exhausted and collapsed, his skin being shrivelled and of a ghastly bluish-white colour, the eyes sunken, and every feature demonstrating intense distress. The surface temperature may fall as low as 92° Fahrenheit, while in the rectum it may be 103 or more. The pulse is almost impalpable, the first sound of the heart extremely feeble, and syncope frequently occurs during this stage, and is sometimes fatal. The urine is suppressed, hiccough may begin, the breath is described as being cold, and the patient is reduced to the most abject state of misery conceivable. This stage lasts from 2 to 24 hours, and the symptoms are largely due to the great drain of fluid from the blood.

The *Third Stage*, or stage of reaction, follows upon the preceding stage, should the patient still survive. The skin becomes warm, and is often covered with an erythematous rash, the pulse and heart improve, the diarrhoea and vomiting cease, at least for the time being, and the kidneys begin to secrete once more. The patient, however, runs considerable risk of inflammation of kidneys, lungs, and intestine, while the temperature may rise to a great height, and the patient die with marked nervous phenomena, such as delirium, subsultus tendinum, or possibly uraemia. Sometimes the diarrhoea continues, the stools becoming excessively fetid, or a relapse occurs, and if so it is very commonly fatal.

The complications have been already indicated.

Diagnosis.—*British cholera, dysentery,* or even *severe*

diarrhœa where due to arsenic or to ptomaine poisoning, may be at first readily mistaken for cholera; but a cholera epidemic in the neighbourhood, and the recognition of the typical organism in the stools, would assist the diagnosis even in its first stage.

Prognosis.—The death-rate is 30 to 80 per cent. It is specially bad at the extremes of life, and debility and alcohol predispose patients both to the attack of the fever and also to a fatal termination.

Treatment.—The treatment of cholera resolves itself into two divisions: (1) prophylactic and (2) an attempt to relieve the symptoms.

(1) Prophylaxis.—Patients should be removed to cholera hospitals, and the most thorough disinfection of clothes, drains, and cisterns should be insisted on, so as to stamp out the source from which the infection occurred. Remembering that the stools are infective, they should be treated with carbolic acid or chloride of lime, and the condition of the water-supply of the community must be thoroughly investigated and any error promptly corrected.

A code of rules should be drawn up for the use of persons living in an infected district, and especially with regard to the boiling of water and milk, the cooking of vegetables and fruits, and careful attention to the prevention of diarrhœa. Everything must be done to discourage alcoholic excess and to improve the general health of the populace. Immunising experiments, as suggested by Haffkine, by injection with first attenuated and later virulent cultures of the cholera bacillus have yielded good results. They only protect for about one year.

2. The Treatment of the Symptoms.—The main thing is to check the diarrhœa, giving opium, bismuth, salol, or acetate of lead. Little benefit results from attempting to feed the patient during the stage of vomiting and diarrhœa, but iced water, effervescing drinks, and washing out the bowel and even the stomach have been suggested. Severe cramps frequently render this latter operation difficult and painful. Much benefit follows the intravenous injection of a saline solution (sodium chloride 1 drachm, and sodium carbonate $\frac{1}{2}$ drachm, to each quart of water), but it may be only temporary. During the stage of collapse apply heat by hot bottles and

practise friction of the limbs, rubbing the patient with dry, warm towels to remove the cold, clammy perspiration, and as soon as vomiting ceases, g. e iced champagne, beef-tea, etc. When the stage of reaction sets in, feed very cautiously, and try to keep down the temperature by sponging or similar means. General tonics and careful dieting are of much value during the stage of convalescence.

XXIII. SPRUE

(Psilosis)

A FEBRILE disease, characterised by irregular action of the bowels with frothy, pale, often pultaceous stools, flatulent dyspepsia, and a raw condition of the mucosa of mouth, tongue, and throat. It causes atrophic changes in the gastro-intestinal tract, with too frequently the eventual exhaustion and death of the patient.

Etiology.—This form of tropical diarrhoea is common in both the East and West Indies, and extends over a fairly wide range of country. No specific organism has been isolated, but the disease, common to both sexes, is almost entirely limited to the foreign population of the lands in which it occurs. It generally attacks Europeans after some years' residence, but no satisfactory explanation is at present available.

Pathological Anatomy.—There is atrophy of the mucosa of the bowel, the glandular structures appear to suffer specially, while occasionally ulceration may be present, and the disease extends to the oesophagus and even the stomach.

Clinical Features.—A European acquiring the disease develops, in the first place, an ordinary diarrhoea, which soon becomes very excessive, the stools being profuse, pale, frothy, and often pultaceous, with much flatulent distension of the abdomen and a typical stomatitis. The diarrhoea once started progresses slowly, gradually undermining the patient's strength, and rendering the sufferer totally unable for work, physical or mental. The tongue is glazed and often fissured, and becomes very painful. The liver dulness is usually diminished.

A mild intermittent type of the disease is sometimes present.

Diagnosis.—The typical stools, together with the condition of the mouth and the country in which the disease was acquired, should render the diagnosis easy.

The **Prognosis** depends partly on successful treatment, but mainly on the departure of the patient to a more temperate climate.

Treatment.—Rest in bed, with a rigid milk diet taken in small quantities, should be kept up for one to two months, and thereafter, during convalescence, a somewhat more generous diet may be permitted. Fresh fruit, and especially strawberries, have been commended. An occasional dose of castor oil is often efficacious in checking the diarrhœa. Opium, chalk mixture, and astringent remedies are valuable, but, above all, a change of climate greatly assists a permanent cure.

XXIV. BUBONIC PLAGUE

A CONTAGIOUS epidemic fever, generally confined to hot countries, with typical carbuncle-like swellings. It is probably the same as the Black Death of the fourteenth century, the name given to it on account of the haemorrhages with which it was associated.

Etiology.—There have been outbreaks of plague in Europe for centuries, and the great Plague of London in the seventeenth century was unquestionably a severe epidemic of this disease. It is associated with India, China, and the East, from some part of which it is rarely entirely absent. Kitasato and Yersin isolated the specific bacillus during the Chinese epidemic of 1894. It is a short non-motile rod with rounded ends, has a characteristic appearance in culture, and produces the disease when inoculated into animals. The bacilli are found in the blood, glands, and viscera, and many animals, such as rats and mice, may acquire and transmit the disease. The rat flea has been proved to be the means by which one rat infects another; man is infected from rats by being bitten by infected rat fleas, and infected rat fleas are conveyed in luggage, cargo, etc., to a fresh rat colony. It is possible that the bacillus may live in the soil, thus becoming endemic in certain regions. It is not very contagious, and faulty hygiene is

generally associated with most protracted outbreaks. The organisms find entrance by the respiratory and possibly the digestive tracts, and certainly in many cases by inoculation by rat fleas.

The disease attacks people of all ages, and most epidemics can be traced to rats which have brought the pestilence with them in ships from some infected port.

One attack appears to confer a certain amount of immunity.

Pathological Anatomy.—The lymphatic glands are inflamed, forming buboes. The spleen is enlarged and soft. Haemorrhages are common in connection with mucous and serous membranes. The post-mortem rigidity is often well marked, resembling in its effects the muscular contractions following death by cholera.

Clinical Features.—The stage of *Incubation* is 2 to 7 days.

The stage of *Invasion* follows, in which prostration, headache, and rigors are marked, and the temperature soon rises; sometimes nausea and vomiting occur. The tongue becomes brown and dry, the urinary secretion is suppressed, and in two-thirds of the cases glandular swellings appear in 2 or 3 days in the groin, axilla, or neck. These swellings are extremely painful, and generally suppurate in about a week, while boils appear over other parts of the body. Sometimes petechial haemorrhages come out on the skin in a severe case, and death may occur within the first week. The temperature during the later period of the fever may rise from 105° to 107° F., and there are frequently extensive haemorrhages from the nose, mouth, and elsewhere. The pulse, at first full, rapidly becomes feeble and dicrotic, and the spleen is usually enlarged.

The appearance of the glandular swellings frequently indicates that the stage of defervescence has commenced, and this occurs with profuse sweating. The glandular swellings discharge, and usually the central slough comes away. It is hardly necessary to add that *Resolution* is by *lysis*, and *Convalescence* may be retarded by prolonged suppuration and ulceration of the buboes, while complications and sequelae, such as suppuration of the necrotic glands, pneumonia, and sometimes insanity, occur.

A number of clinical types of the fever have been described:—

(1) *A Malignant Type*, in which death may occur within

twenty-four hours, with or without haemorrhage, and in which no buboes develop.

(2) *Pestis Minor*—mild cases of plague, but with marked buboes, usually leading to a satisfactory result.

(3) *Pestis Major*—simply a severe type of bubonic plague.

There are also certain cases in which the lungs suffer especially, and to which the term *pneumonic plague* has been given.

The urine and faeces contain the bacilli, and in the pneumonic form the sputum swarms with the organisms.

Diagnosis.—The existence of an epidemic, with the characteristic buboes, and the information gained by experimental inoculation of rats and other animals with the blood of the suspected patient, are usually sufficient to clear up any doubtful cases.

Prognosis.—The bubonic form is rather more favourable than the type in which no glandular swellings occur. Suppuration in the buboes is considered a good sign. The death-rate, however, is extremely high, from 40 to 80 per cent of cases proving fatal.

Treatment.—The patients should be isolated, their dwellings disinfected, and those who have been in contact with them quarantined. The findings of the recent Plague Commission prove beyond doubt the part the rat and its parasitic fleas play in the spread of plague, and therefore the extermination of rats should be carried out, at least in all plague-stricken districts.

As regards the patient, relieve the constipation, which is often severe, by a dose of calomel, and thereafter give opium or morphia, which will greatly aid in relieving the restlessness, delirium, and pain. Stimulants, and especially alcohol and strychnine, are of great value where the heart is threatening to fail, and in no disease does iced champagne or some alcoholic stimulant render such signal service. Above all, check the excessive pyrexia by cold sponging or other hydropathic treatment.

The buboes should be treated, in the first place, by smearing with alcoholic extract of belladonna and glycerine in equal parts, and later they should be incised just as an ordinary abscess.

Many attempts have been made to introduce a satisfactory

serum treatment, but without, at the present time, much success. On the other hand, the immunisation of unaffected persons in a plague-stricken district is certainly worthy of trial. Haffkine's serum consists of a sterilised bouillon culture, and its efficacy as a prophylactic agent has of late years been recognised.

XXV. YELLOW FEVER

An acute specific tropical fever, characterised by jaundice, severe gastro-intestinal disturbance, haemorrhages, albuminuria and sometimes suppression of urine. Yellow fever is specially common in the West Indies, in parts of South America, and West Tropical Africa.

Etiology.—Sanarelli has isolated a specific organism, which he describes as being a small ciliated bacillus producing a very virulent toxin, but his results have not been confirmed by any of the recent workers at the subject. The white population suffer more than the black, and strangers are peculiarly susceptible. Swampy, low-lying country, and especially insanitary and overcrowded towns suffer, and chiefly in hot weather. Apparently the organism possesses great resistance, and ordinary cold weather is not sufficient to exterminate it, the disease reappearing with any rise of temperature.

The mosquito (Stegomyia fasciata) has been proved to be the medium of inoculation, and the disease cannot be carried by clothes or acquired by contact with infected persons. The extermination of the mosquito from Havana in 1901 was successful in causing the total disappearance of yellow fever.

Pathological Anatomy.—There is intense jaundice of the skin, and many petechial and other haemorrhages are noted. There is marked fatty degeneration of the liver, often nephritis, and many red blood corpuscles are destroyed: otherwise the appearances are not distinctive.

Clinical Features.—The stage of *Incubation* is 1 to 7, generally 3 or 4 days.

The attack is usually sudden, or after severe headache and malaise it begins with rigors. Great frontal headache, pains in the back and joints, and pain over the stomach, with

nausea and vomiting, are prominent symptoms. The flushed face and very slight icteric tinge are early features and of diagnostic value. In 2 or 3 days marked jaundice appears, there is albumin in the urine, and the pulse instead of rising in rate with the temperature tends the second day and afterwards to become slower, and may not exceed 70 to the minute even with a temperature of 103° F. On the 4th day the fever remits, and the patient recovers, or a severe relapse may occur.

Especially in fatal cases, haemorrhage from the stomach (commonly called "black vomit") and bowels is common, and there may be suppression of urine, convulsions, and coma. One attack appears to confer immunity. Recovery is often delayed owing to complications and sequelae, such as protracted diarrhoea, inflammation of the parotid glands, nephritis, etc.

Diagnosis.—There is practically always an epidemic present in the district, but the appearance of the face, which is flushed and swollen, and the very definite jaundice, should help in the diagnosis. The fever most nearly resembling it is *blackwater fever*, but the previous history of malarial attacks renders a mistake unlikely.

Prognosis.—The death-rate is 10 to 75 per cent., generally nearer the higher figure.

Treatment.—The patient should be carefully treated, firstly, with the view of arresting the excessively painful vomiting, for which washing out the stomach, the application of small blisters, and the administration of such remedies as dilute hydrocyanic acid, creosote, or ice, are all recommended. Nutrient enemata should be given when vomiting prevents ingestion of food.

Haemorrhage may need to be checked, and ergot, large doses of the tincture of the perchloride of iron, and acetate of lead may all be recommended. Small doses of $\frac{1}{60}$th of a grain of perchloride of mercury, administered in iced water every hour, have been found of advantage, and the free use of alcohol, when necessary, helps to support the patient's strength. Quinine has been given with success.

The PROPHYLAXIS of the disease is a disputed matter. Unquestionably the extermination of the mosquito is the all-important method of stamping out the fever, and further, until this has been accomplished, no opportunity should be afforded

for mosquitoes becoming infected by biting patients suffering from the disease.

XXVI. MALTA FEVER

(Mediterranean Fever)

A specific fever, confined to places on the Mediterranean, and characterised by a long, somewhat tedious illness, with many partial relapses, the joints being specially involved, neuralgic pains very frequent, and the spleen enlarged.

Etiology.— The disease is due to a definite organism, the Micrococcus melitensis, discovered by Bruce in 1887. It is, or was, specially common in Malta, Gibraltar (Rock Fever), Naples, and Sicily. It is not contagious. Goats are extensively affected in districts where the fever is prevalent, and the organism is found in their milk. The micrococci are also present in large numbers in the urine of infected persons, and it is conceivable that the disease may be propagated by the contamination of water, etc., with sewage.

Pathological Anatomy.—The spleen and liver are enlarged, the former being markedly congested; otherwise there is nothing to note except the presence of many of the micrococci.

Clinical Features.—The stage of *Incubation* is 6 to 10 days. There are various types of the fever which so far differ from each other.

(1) There is a type in which the temperature rises by a gradual daily increase, associated with intense pain, constipation, and headache. In about a week the fever abates and a period of apyrexia follows, which in turn is succeeded by another relapse and a small daily increase of temperature much like the original attack. The temperature is often remittent, *i.e.* 104° F. at night and about 100° in the morning, and never reaching normal during the pyrexial attack. These relapses recur sometimes for many weeks or months: gradually they may become less severe, and the patient recovers.

Associated with the disease are joint pains and swellings, severe neuralgic pains, bronchitis, and other complications.

(2) A malignant type may also be recognised, in which the fever process is very intense, and the patient dies from

hyperpyrexia, cardiac failure, or possibly a pulmonary complication in a few days' time.

There are many quite atypical cases with fever only at night or totally irregular temperatures. Some of these are mild cases with few complications.

The **Complications** of the fever, some of which have been already indicated, are often very numerous and very severe: neuralgic pains, intractable insomnia, involvement of joints, orchitis, and many other less serious disturbances of the different systems which render convalescence slow and unsatisfactory.

Diagnosis.—No fever presented at one time such difficulty in regard to diagnosis, but the discovery of the micrococcus in the urine and the agglutination test (the organisms are agglutinated by a patient's serum in dilutions of 1 in 1000) have removed all difficulties. *Typhoid*, *malaria*, and *rheumatic fever* are amongst the conditions with which the disease is most readily confused.

Prognosis.—The death-rate is low, being somewhere about 2 per cent; but, on the other hand, the long, protracted convalescence renders the fever more formidable than many conditions with a much higher mortality.

Treatment.—Fluid diet, and, when the temperature is high, cold sponging or the cold bath are advisable. A vaccine has been prepared, but its use is only recommended in moderately severe cases. The greatest relief, as far as the joint pains are concerned, is obtained from hydropathy.

XXVII. AMOEBIC AND BACILLARY DYSENTERY

The term dysentery is applied to cases of diarrhoea, with severe tenesmus, in which there is a typical mucous or muco-purulent stool, practically always at one time or other blood-stained.

While many cases in the tropics are truly amoebic, we frequently meet with dysentery due to a bacillus, and it is extremely difficult to dissociate entirely the two groups. In more temperate climates severe enteritis, if involving the large intestine, may acquire the characteristics of dysentery. To many of these sporadic and epidemic cases, not due to the

amoeba, the term bacillary dysentery has been given, because they are due to a specific group of bacilli.

Etiology.—Amoebic Dysentery is the tropical form of the disease, due to the Amoeba dysenteriae, although some authorities still claim that the presence of the amoeba is merely accidental, and that some other organism or organisms may be the true cause of the condition. The amoeba is a unicellular, protoplasmic, motile organism, 15 to 30 μ in diameter, with a clear outer zone called the ectosare, and a granular inner zone called the endosare. It contains a nucleus, and one or more vacuoles.

The bacillus of **Bacillary Dysentery** has been isolated by Shiga of Japan, and others, but it is now certain that there are several different varieties all of which produce very similar symptoms. It is found in the stools, and is agglutinated by the blood-serum of the patient. The disease is endemic in many lands, and specially in damp, low-lying countries near the equator: it occurs as an epidemic especially where large numbers of persons are collected together under somewhat insanitary circumstances, and is therefore frequently a scourge in military campaigns. The incidence of the disease is certainly lessened by careful drainage, attention to the water-supply, freedom from overcrowding, and care with regard to feeding of troops and others likely to be affected.

Sporadic cases occur in both tropical and temperate climates, but where the specific organism, whatever it may be, suddenly attacks the individual, these cases are not so likely to prove truly infective to others. Probably the specific organism or organisms are introduced into the human subject in water and in the autumn, or towards the end of the tropical rainy season, epidemics are most apt to occur. Both sexes suffer, no race is immune, and children appear to be specially predisposed. One attack of the disease renders the patient liable to future attacks, especially as the result of sleeping on damp ground, imprudent diet, or excessive fatigue.

Pathological Anatomy.—(1) In **Amoebic Dysentery** the large intestine is generally involved, with inflammation of the submucosa, the muco s, and specially the solitary glands, and, as a rule, the caecum, the hepatic and sigmoid flexures, and the upper part of the rectum are most severely affected. Ulceration follows in the inflamed oedematous mucosa, and

sloughs separate, leaving small, punched-out ulcers representing the solitary glands, and larger ulcers due to the ulceration of extensive areas of mucosa. As a rule the ulcer does not involve more than the mucosa and submucosa, but the remaining wall of the bowel may be thinned, and perforation through the muscular coat and peritoneum is by no means uncommon. There is a remarkable absence of purulent inflammation. Sometimes curious undermined bridges of mucosa join neighbouring ulcers together, and in some cases the whole mucosa becomes gangrenous, or may even be covered with a diphtheritic membrane. In these more serious types of the disease large areas of the mucosa may slough, and perforation or adhesion of the affected bowel to neighbouring structures may occur.

In the more chronic form of the disease there is a great amount of pigmentation, especially of the floor of the ulcers, and the affected areas are often greatly thickened, in other parts thinned, causing much alteration in the size of the lumen of the bowel, and stricture of the gut may result.

Secondary abscesses in the liver are specially frequent in amoebic dysentery, and may occur a considerable time after the onset of the attack; more rarely abscesses develop in other organs. In some of these abscesses the amoebae may be recognised in the pus, which in the liver has a peculiar, gelatinous-looking, often reddish appearance (see p. 294).

The mesenteric glands are inflamed, and may even suppurate, and in chronic cases are generally pigmented.

2) In **Bacillary Dysentery** there is intense congestion of the large intestine especially, but no ulceration as a rule. Gangrene may, however, occur, the mucosa becoming of a purplish colour.

Clinical Features.—1. **Acute Catarrhal Dysentery, Amoebic** and **Bacillary.**—Malaise with a varying amount of fever, some abdominal discomfort, and diarrhoea. The diarrhoea increases, with colicky pain and severe tenesmus. The tongue is at first furred, but later red; nausea and vomiting are occasionally present, and there is excessive thirst. The stools are typical, and contain pus, blood, and glairy mucus; in the amoebic form the Amoeba coli is present, and in the bacillary form large numbers of the Bacillus dysenteriae. During the ulcerative stage of amoebic dysentery masses of stinking,

decomposing mucosa may be passed by the patient, and should gangrene ensue, the phenomena of acute obstruction are superadded in their worst form. These include intractable vomiting, the abdomen being swollen and excessively tender, and death terminates the attack in a few hours' time.

The number of stools varies greatly from 15 to 200 in the twenty-four hours (according to the severity and type of the case), but in the amoebic cases relatively fewer stools are passed by the patient. The abdominal tenderness and the degree of distension present depend much on the severity of the process and the amount of putrefactive change in the contents of the bowel. The temperature also varies greatly. In mild cases it may not be higher than 101 or 102 F., while in more severe cases it may rise to 104 .

Occasionally a diphtheritic form occurs, with grave typhoid-like symptoms and high fever, which generally proves fatal.

Improvement may set in after a few days in mild cases, but often in amoebic dysentery the disease continues for 6 to 12 weeks, eventually causing profound anaemia and debility. The bacillary form is very fatal when it is of a severe type.

2. **Chronic Amoebic Dysentery.**—The chronic form frequently succeeds the acute and has similar symptoms, although less severe; there is less tenesmus, fewer stools, and the pain and discomfort are associated with the period following digestion when the waste matters have reached the large intestine. The appetite is capricious, and the patient may become anaemic and even cachectic from the long-standing drain on the system.

The bacillary form may also occur as a subacute or chronic disease, but less frequently.

Diagnosis.—There is no difficulty in recognising cases of dysentery, although it may be excessively hard to say whether it is the result of some dietetic indiscretion or a true case of the amoebic or bacillary type. The amoebae and the bacilli should be searched for in the stools, and the fact that the bacilli are agglutinated by the patient's blood-serum should not be forgotten. A leucocytosis of 15,000 to 20,000 is suggestive of hepatic abscess. Digital examination of the rectum should invariably be carried out where *haemorrhoids, ulcer*, or other local affection of the rectum is probable.

Prognosis.—The disease in its most favourable type is one

of long duration, and it is extremely apt to recur, and therefore in all cases it is prudent to offer a guarded prognosis, although, if the sufferer leaves the country in which dysentery is common for a more temperate climate, the cure is likely to be rapid and permanent. The prognosis is seriously complicated by the addition of a liver abscess, the death-rate even in cases operated on, being high.

Treatment for both **Amoebic** and **Bacillary Dysentery.**—Enjoin absolute rest in bed, with the most careful and restricted dietary, feeding the patient entirely on food-stuffs which leave, as far as possible, no waste matters to reach the large intestine. Probably milk and beef-tea in small quantities are amongst the best articles of diet.

In **Amoebic Dysentery** intestinal antiseptics are of use because it is believed that other organisms are necessary for the growth of the amoeba, and full doses of quinine, small doses of calomel, and sometimes ipecacuanha have been recommended. The last named is certainly less useful in amoebic than in bacillary dysentery. The most successful treatment is, however, irrigation of the colon by large enemata of quinine (5i. to 3 pints of water), which should be so administered as to reach the caecum, and be retained for 15 to 20 minutes. Should enemata be found difficult an appendicostomy may be performed, and the colon irrigated at will. Pain may be relieved by opium or morphia.

The medicinal treatment of **Bacillary Dysentery** is by no means satisfactory. We generally commence operations by giving a mild purge, such as castor oil, salts, or a small dose of calomel, and then attempt by one of three methods to relieve the patient. (1) Ipecacuanha powder in large doses of 20 to 40 or even 60 grains is given in wafer paper, and to prevent vomiting it may be preceded by a fairly large dose of morphia. It should be taken on an empty stomach and while the patient is absolutely at rest, because the slightest effort induces the rejection of the powder by vomiting. If the maximum dose has been administered at one time, six hours may be allowed to elapse before repeating the remedy. (2) *Sedatives* such as bismuth, in quantities of at least 12 to 15 drachms in the twenty-four hours, or *antiseptics* such as salol, in 15 to 20 grain doses, or perchloride of mercury in $\frac{1}{100}$th grain doses every two hours, are often of signal benefit. (3) An attempt may be made to

attack the disease by rectal injections. After the administration of morphia or cocaine suppositories, irrigation of the bowel may be carried out with one of the following solutions: either 20 to 30 grains of silver nitrate to the pint of water, 2 to 3 pints being used, or antiseptic solutions of quinine, 1 part in 2000 of warm water, may be slowly injected into the bowel. It is hardly necessary to say that the silver nitrate injection is extremely painful, and the irrigation method may cause the utmost agony to the patient. Sometimes warm opium and starch enemata (60 minims of tincture of opium in 1 to 2 ounces of thin starch) are of great benefit in relieving the awful tenesmus, while washing out the rectum with warm boracic lotion often gives relief. Recently the administration of sodium sulphate in 1 to 2 drachm doses by the mouth three or four times a day has been found most satisfactory. Hot baths and hot fomentations applied over the abdomen are most soothing.

In chronic cases of both kinds of dysentery, if at all possible, insist on the patient having a long holiday, going for a considerable length of time to a more temperate climate.

XXVIII. BERI-BERI

An endemic and epidemic peripheral neuritis, occurring in tropical and subtropical countries, but differing from the other varieties of multiple neuritis by the special involvement of the vagus, phrenic, and vasomotor nerves, by great liability to oedema, and by a tendency to cardiac dilatation which may be fatal.

Etiology.—The disease is found throughout a large part of the tropical world, but particularly in Japan, in the north of South America, and in China, and it may also occur in more temperate regions. It is certainly due to an organism, possibly a fungus, although as yet undiscovered. It is as common in the town as in the country, and is certainly brought on mainly by the work or diet of those affected. Persons living largely on white rice run a special risk. This is rice which is husked by milling when husk, pericarp and surface layers of the grain are removed. Parboiling renders the rice innocuous: working among infected rice dust may

also cause the disease. Natives mostly suffer, males are more affected than females, and the usual age is from 16 to 25.

Pathological Anatomy.—There is marked nerve degeneration closely resembling what is found in peripheral neuritis, and the vagi, phrenics, the cardiac nerves, and the nerves of the limbs and even the trunk, may be affected. In the muscles supplied by these nerves fatty changes are prominent, and the heart is greatly dilated, especially on the right side. There are, in addition, the usual changes in the nerve-cells of the degenerated neurones which one would expect to be present.

Dropsy is a distinctive feature, and may be present in most of the serous cavities, especially the pericardial sac, and also generally throughout the body.

Clinical Features.—The period of *Incubation* is unknown, but may extend for a long time. There are often premonitory symptoms, such as depression, numbness, headache, stiffness, and cramps, and during this period slight oedema of the ankles and feet, with a puffy appearance of the face, develops. Later a definite and pronounced peripheral neuritis appears, which rapidly becomes worse, the muscles are tender, while the skin may be numb, burning, or tingling, and cramps are not infrequent. There are often patches of hyperaesthesia generally found on feet or ankles. The knee-jerks are lost, the affected muscles soon waste, and the patient becomes breathless and suffers from epigastric oppression and severe attacks of palpitation. The condition lasts for weeks or months, during which period the digestion may be unimpaired, the intellectual powers good, and there may be no fever of any kind. The affected muscles may show the reaction of degeneration until it is lost, and the patient frequently develops a typical ataxic gait with Romberg's phenomenon, but in any case, owing to the extent of muscular involvement, he becomes feeble and staggers when he walks. In raising the foot the heel is lifted first, and there is high-steppage gait. During this time the oedema and general weakness become more and more marked, but cases vary in the degree of involvement of the heart, of the muscles, and even in the amount of dropsy present, so that many types of the disease have been differentiated. One remarkable feature is the absence of bed-sores in nearly all varieties of beri-beri.

Death in fatal cases is generally due to the spread of the affection in marked degree to the vagus nerve or the phrenics, to cardiac failure, to extreme dropsical effusion into the plural or pericardial sacs, and, lastly, to prostration from the numerous relapses which may occur.

Diagnosis.—As may be gathered from a careful study of the preceding symptoms, the disease differs from ordinary *multiple neuritis*, which is the only condition to which it bears any obvious similarity, and of which it is in reality a special and specific form.

Prognosis.—The death-rate varies from 1 in 40 to 1 in 2 of the patients attacked. Apparently cases with excessive dropsy are apt to be more serious, but epidemics vary greatly in their mortality.

Treatment.—The food should be at once changed from rice, with which most authorities now associate the disease, to a diet composed of meat, fish, fresh milk, and fresh vegetables, while care should be taken to keep the patient from sleeping on the ground, and to ensure a dry, well-aired room. Drugs do not appear to be of any very great value, but quinine, salicylate of soda, arsenic, and many others have been recommended. Probably strychnine, administered by the mouth or given hypodermically, together with electrical treatment and massage, will do more for the affected nerves and muscles than anything else.

XXIX. TETANUS

(Lockjaw)

An acute infective disease, characterised by tonic spasms of the voluntary muscles, and due to the presence of the bacillus of tetanus.

Etiology.—The bacillus was discovered over twenty years ago, and has been cultivated by Nicolaier and later by Kitasato. It is an anaerobic bacillus growing in threads: the bacilli are sometimes motile and sometimes motionless. They are commonly known as the drumstick bacilli, because at one end of each bacillus there is a refractile spore, which rapidly develops and may be well seen in cultures of a certain age. The

bacilli occur in the soil, the spores are very resistant, and as a result of the growth of the bacillus there is a virulent tox-albumin, which is said to be 100 to 200 times more poisonous than strychnine. Different observers have separated other toxic bodies.

Inoculation of a wound by the bacillus readily produces the disease, the phenomena of which are due to the production of the toxin or toxins. Probably infection is local, although the toxic bodies affect the whole system. The bacilli are found in garden soil, and are said to be associated in some way with the manure of horses.

A separate group of cases of the disease is called *idiopathic*, and to this group the cases reported as occurring in hot countries, and cases said to be due to cold, must be referred. An epidemic form called *tetanus* or *trismus neonatorum* occurs amongst newly-born children in hot countries.

Pathological Anatomy.—It is frequently stated that there is inflammation at the site of inoculation (if a scratch or wound), but it is difficult in certain cases to assure oneself of the truth of this.

In the central nervous system there is congestion, and the changes are most marked in the region of the great motor centres. It has been suggested that in the idiopathic form the spores may be inhaled, and that therefore bronchial irritation is present in those cases.

Clinical Features.—Generally about ten days after the injury, slight stiffness of the neck and jaw muscles indicates the onset of the disease: the spasms occur repeatedly, especially affecting the muscles of mastication, and the *risus sardonicus*, or the typical and ghastly smile produced by the raised eyebrows and drawing outwards of the angles of the mouth, frequently develops. Gradually other muscles suffer, causing *opisthotonos*, during which the patient's body may be arched backwards, so that he rests on the back of his head and his heels. In other cases there may be lateral bending called *pleurothotonos*, or even bending forwards, to which the name of *emprosthotonos* has been applied. With the severe spasms the body is held as in a vice, the pain is agonising, breathing becomes difficult, the temperature rises often as high as 105 or 106 F., and death may occur from hyperpyrexia.

In other cases heart failure during the spasms, or asphyxia or sheer exhaustion, may bring about a fatal result. The slightest irritation, such as a heavy foot on the floor or shaking the bed, brings on the spasms, while a measure of rigidity persists all the time. The pulse-rate rapidly increases, and there is the utmost difficulty in swallowing, and therefore in keeping up the patient's strength, while sleeplessness is a troublesome feature.

KOPF-TETANUS is a term applied to the disease when it results from a wound or injury of one side of the head. Here the facial muscles on the side of the wound are paralysed, there is stiffness of the jaw and the back muscles, and those of the pharynx and larynx are specially affected by the spasm.

Diagnosis.—*Strychnine poisoning* most closely resembles tetanus, but in it the jaw muscles do not suffer so early, and there is no rigidity between the spasms. *Tetany* is too slight to render a mistake possible.

Prognosis.—In milder cases, especially with the antitoxin treatment, recovery may occur, but in severe cases the death-rate is very high. If the patient survives the first four days the prognosis is greatly improved.

Treatment.—Attention must be at once paid to the condition of the wound, which should be excised and treated with antiseptics. Keep the patient in a dark room, absolutely at rest. Morphia, chloral, belladonna, cannabis indica, curara, and bromides are all beneficial, while chloroform has been administered for the relief of the paroxysms.

The antitoxin treatment depends on the fact that Behring and Kitasato succeeded in rendering animals immune to the disease in a manner similar to what had previously been achieved in the case of diphtheria. 20 to 30 c.c. of the antitoxin are administered at once, preferably into the spinal canal after the withdrawal of a corresponding amount of cerebro-spinal fluid, by lumbar puncture, and later 15 to 20 c.c. should be given. The method is well worth a trial, and especially so because the antitoxin has proved of much value in veterinary surgery. Spinal anaesthesia by cocain, suggested many years ago, is also worth considering.

XXX. HYDROPHOBIA

(RABIES)

An acute disease depending on a specific virus inoculated by dogs, wolves, and more rarely other animals, into the human subject.

Etiology.—The virus of rabies, although extensively used in the Pasteur method of treatment, is the toxin of an organism as yet undiscovered. Protozoa-like bodies (4-10 μ in size) have been described by Negri in the cells of the cerebellum, cerebral cortex, pons, and cord in infected animals. It is a remarkable fact that whereas the virus is the same in all animals, and capable of causing hydrophobia, the bite of certain of these is more apt to be fatal than that of others. The wolf, fox, cat, and dog generally transmit hydrophobia, the wolf and the cat giving a more dangerous bite than the dog, the animal with which we most frequently associate the disease. The poison is in the saliva of the affected animal, and the certainty of inoculation depends greatly on whether the bite is on an exposed part of the skin, or whether much of the infective saliva is wiped off upon the clothes. A lacerated wound is more apt to be followed by the disease, and a lesion of an area of skin richly supplied with nerves is much more certain to produce the disease.

Many other animals besides those mentioned may become inoculated, such as cattle and horses.

Pathological Anatomy.—Changes are described in the cerebral cortex, medulla, and spinal cord, and especially in the medulla, in the region of the vagus, hypoglossal and spinal accessory nuclei. The changes referred to are dilatation of small vessels, minute haemorrhages, and aggregations of leucocytes while the mucosa of pharynx and oesophagus may show marked congestion.

Clinical Features.—The stage of *Incubation* is of very varying length (weeks or months), depending apparently on the nature of the wound, the animal by which inoculation is produced, and the age and degree of immunity of the patient. The period is shorter in children, while certain persons appear to possess a remarkable resistance to the virus.

Long after the wound has healed the stage of *Invasion* manifests itself by renewed redness and irritation at the site of the scar, loss of appetite, and mental depression, which is soon replaced by excitement. There is irritability, especially to bright lights or loud sounds, and some stiffness of the throat muscles.

The next stage of the disease is one of marked excitement, great restlessness and hyperaesthesia, with violent spasms produced by any peripheral irritation, and especially affecting the muscles of the mouth, larynx and pharynx: there is great difficulty in swallowing, because it produces spasm, and breathing is difficult. The saliva cannot be swallowed, and is spat out, possibly in the faces of those standing around, and the apparent bark with which the patient is popularly credited is really due to spasm. The temperature rises to 101° or 102° F. This period usually lasts for $1\frac{1}{2}$ to 3 days, and is succeeded by the paralytic stage, terminating in 6 to 18 hours with the death of the patient, often by heart failure.

During the greater part of the disease the mind is clear although hallucinations may be present in some cases.

Diagnosis.—The history of the bite many weeks or months before, together with the characteristic clinical features of the attack, is sufficiently suggestive, but it is always desirable that the supposed mad dog should be kept alive and under observation. If, however, the animal has been killed, then a rabbit should be inoculated under the dura mater with a small part of the dog's medulla. After a period of 2 or 3 weeks rabies develops, should hydrophobia have been present.

Prognosis.—The disease, once it commences, is generally fatal, and the success of the Pasteur method of treatment depends on the prevention of the attack rather than its cure. The bites of rabid wolves and cats are peculiarly fatal.

Treatment.—The wound must be at once cauterised and a ligature applied above the part bitten: while sucking the wound, if there is no abrasion of the mucous membrane of the mouth, should be energetically performed. As soon as possible the Pasteur method of immunisation should be carried out.

Pasteur's Method of Immunisation.—In 1885 Pasteur trephined a rabbit and inoculated under the dura mater a small amount of the spinal cord of a rabid dog. In fifteen

days rabies appeared; one rabbit was inoculated from another until the incubation stage was reduced to six days. In this way Pasteur obtained his *virus fixe*. The spinal cords of these rabbits were suspended in the air, and gradually lost their virulence. The cords kept for fifteen days are first used for inoculating the patient, and so on until a cord suspended for one day only is used, so as gradually to increase the immunity of the patient against the virus.

The risk that an individual who has not really been bitten by a mad dog at all might acquire the disease by inoculation is proved to be imaginary. The inoculation is generally performed into the abdominal wall, and is by no means free from pain, while pyrexia and malaise may result from the treatment.

The death-rate from hydrophobia has been greatly reduced, and recent statistics bear eloquent testimony to the efficacy of this treatment.

When the attack has developed, little can be done, with the exception of keeping the patient at rest and administering morphia and other sedatives, while the feeding of the patient presents the greatest difficulty.

During the period of hot weather, when rabies is most common, dogs ought to be muzzled, and animals found about the streets, and which are unclaimed, should be destroyed.

Pseudo-Rabies

It is important to remember that in hysterical patients hydrophobia may be simulated, and the better informed the patient is with regard to the clinical features of the disease, the more nearly are these imitated by the sufferer.

XXXI. GLANDERS

An infectious disease occurring in horses, and due to the Bacillus mallei, causes *glanders* when affecting the nasal and respiratory passages, and *farcy* in the skin. Grooms are infected direct from the horse.

Pathology and Pathological Anatomy.—The bacilli are short and non-motile, and are somewhat difficult to recognise

by staining methods. Typical swellings and nodules appear in the skin, mucous membrane, lymphatics, and also internally. These inflammatory nodules are composed of granulation tissue, and, dependent on their site, abscesses or ulcers result. The toxin causes the pyaemic symptoms.

Clinical Features. — *Acute Glanders.*— After an incubation stage of 3 to 4 days the temperature rises, and nodules, which rapidly ulcerate, are found in the nose, causing a copious purulent and fetid discharge. There is great swelling of the nose with erysipelatous redness, the neighbouring lymphatic glands are involved, and death follows in 1 to 2 weeks.

Chronic Glanders.—Closely resembles a severe coryza with ulceration which slowly spreads to the pharynx. It is rare and difficult of diagnosis.

Acute Farcy.—Follows on inoculation of the skin, and consists in a localised inflammatory infiltration, producing subcutaneous nodules along the line of the lymphatics (farcy buds), which rapidly suppurate. Neighbouring lymphatic glands and joints are involved, and abscesses in the muscles follow: death generally results in about two weeks often from pyaemic pneumonia or involvement of pleura, intestine, etc.

Chronic Farcy.—Similar subcutaneous nodules, but with much less inflammation, and suppuration is slower. The disease lasts for months or years with varying results.

In glanders a pustular eruption frequently appears on the face.

Diagnosis. There is usually direct evidence of infection from the horse, but it often runs a more chronic course in horses, and therefore may present some difficulties. In all cases a careful examination should be made of the secretion and inoculation experiments performed. Straus has pointed out that if the peritoneum of a guinea-pig be inoculated with the suspected matter a severe orchitis develops in 2 or 3 days, and the bacilli can be obtained from the fluid in the tunica vaginalis.

Prognosis.—In man it is a serious disease, only a small per cent of really acute cases recovering, and even in the chronic forms at least 50 per cent die.

Treatment.—Prompt disinfection of the wound is absolutely necessary, and an attempt may be made to apply antiseptics to the nasal passages.

Mallein, a sterilised culture of the Bacillus mallei in peptone bouillon, has been administered in minute doses to man, but it is as yet, impossible to say if it is beneficial.

XXXII. ANTHRAX

(Malignant Pustule. Woolsorters' Disease)

An acute infectious disease due to the inoculation with the Anthrax bacillus.

A wound may be inoculated from infected cattle, as in the case of slaughter-house men, butchers, tanners, woolsorters, and stablemen (malignant pustule), while in the case of woolsorters the bacilli may also be inhaled into the lungs, and there set up an internal and very virulent type of the disease. The disease seems to be endemic in certain countries such as Siberia, Russia, and Persia, and has in the past caused enormous destruction of cattle.

Pathology and Pathological Anatomy.—The bacillus varies greatly in size, and is often as big as 20 μ in length by 1 μ or more in breadth. They form spores which are very resistant, and are probably the cause of infection of the respiratory passages or alimentary tract. Spores do not form within the body. The organism produces most powerful toxins, to which many of the clinical features are due.

In malignant pustule there is a rapidly extending area of inflammation round the site of inoculation. The central part of the pustule becomes necrotic, and the lymphatic glands in the neighbourhood swell markedly.

In the lungs it causes a type of broncho-pneumonia, and in any internal form of the disease it is usual to find a widespread distribution of bacilli throughout the body.

The **Clinical Features** in the case of malignant pustule are a certain amount of pain, high temperature, and general malaise; while in the internal form the phenomena vary with the site of infection. In pulmonary anthrax (woolsorters' disease) there is local pain, severe cough, and at an early period in the illness great malaise and profound constitutional symptoms terminating in collapse. In some cases symptoms may be absent until shortly before death. Where the

alimentary tract is affected, vomiting and diarrhœa of an intractable type are superadded, cyanosis supervenes and rapid death is inevitable.

Diagnosis.—In malignant pustule it is not as a rule difficult to get a fairly suggestive history, while a culture of the bacillus anthracis should be made.

In the internal form the diagnosis is often extremely obscure.

Prognosis.—Prompt treatment of a malignant pustule yields the only satisfactory results. In internal cases there is little hope of cure.

Treatment. *Prophylaxis.* Much has been done to stamp out anthrax in cattle. Pasteur's method of rendering cattle immune by injecting an attenuated virus has been commended for use in districts in which the disease is endemic. In malignant pustule apply powerful antiseptics, such as liquefied carbolic acid, or a strong solution of chloride of zinc, or inject into the pustule a 10 per cent solution of iodoform in ether.

For the internal form support the patient's strength and prescribe quinine in large doses.

A serum has been prepared by Professor Sclavo. It should be injected intravenously in 10 to 60 c.c. doses, and has already yielded good results.

XXXIII. ACTINOMYCOSIS

THE actinomyces or ray-fungus is common amongst cattle and horses, and occasionally pigs, but it is rare in man. The exact mode of infection is still doubtful. The spores are very resistant, and it is probable that they may be inhaled or taken into the alimentary tract.

The organism has given rise to much discussion both as to its nature and development. It is usually found in the tissues as an interlacing mass of radiating threads which at a later period develop somewhat larger club-shaped extremities. There are also many spores. The fungus stains by Gram's method. In the areas involved by the fungus there is an extensive leucocytic invasion and small-cell proliferation, and if this occurs in non-resistant tissues, necrosis is apt to result. In some places the disease spreads with great rapidity

in others it is more or less limited, and pus formation is not uncommon, the pus containing the typical fungus.

The disease usually affects the tongue in animals, producing what is called "wooden tongue," and is most common in the ox, but may be found in other domestic animals. In man it may involve the jaw (lower or upper), but in certain cases is more marked in the lung, and may be met with throughout many of the internal organs.

The **Clinical Features** vary greatly, depending on the site and the extent of the infection. In the jaw the development of a suppurating abscess, which eventually discharges, is associated with a certain amount of pain, malaise, and temperature, but it is probable that the constitutional disturbances are mainly the result of the superadded attack by pyogenic organisms.

The tongue is not infrequently affected, but it is certainly in the lungs that most instances of the disease in men are met with. The pulmonary form may resemble a bronchopneumonic or a miliary tuberculosis, and sometimes simply a severe bronchitis. The bases of the lungs are more apt to be affected in actinomycosis than in pulmonary tuberculosis. When the fungus is found in the sputum the diagnosis becomes clear. The liver and other organs are much more rarely involved.

Diagnosis.—Finding the ray-fungus in the pus is usually sufficient, while the lymphatic glands suffer to a less extent in this disease than one would expect.

The **Prognosis** depends largely on the possibility of complete removal of the disease; otherwise it is eventually fatal.

Treatment.—Remove the disease by surgical means if possible. Internally the use of iodide of potassium (gr. 30-60 daily) has yielded satisfactory results in certain cases.

XXXIV. FOOT-AND-MOUTH DISEASE

An acute infectious disease occurring in cattle, sheep, and pigs, and more rarely other animals.

Transmission to man may be by milk or butter from infected animals, or sometimes by inoculation, as by a scratch. It spreads in cattle with great rapidity, and usually after a

short period of *Incubation* pyrexia sets in, and the mucosa of the mouth shows the typical inflammation.

The **Clinical Features** are inflammatory vesicles on the lips, mouth, and pharynx, which rapidly cause painful ulcers. There is fever, marked salivation, and generally considerable gastro-intestinal disturbance. In severe cases there is a tendency to haemorrhage.

The disease usually terminates favourably, and the treatment should consist partly in the isolation of infected cattle, and as regards the human subject attention to general principles.

XXXV. LEPROSY

A CHRONIC infectious disease, caused by the presence of the Bacillus leprae, and characterised by one or other of two definite types, either (1) a *tubercular* leprosy, in which tubercular-looking nodules appear in the skin and mucous membranes; or (2) an *anaesthetic* leprosy, in which the bacillus specially attacks the nerves, causing sensory, motor, and trophic changes. It not infrequently happens that a patient presenting the characteristics of the first type eventually develops the second, and *vice versa*.

Etiology.—The disease interests us specially because of its occurrence in India and many other of our dependencies, while in Europe, although less prevalent, it still exists in Norway and Sweden, and in parts of Russia, especially those parts bordering upon Asia Minor. In certain lands the disease appears to be much more infectious, as well as being endemic.

Hansen originally discovered the bacillus of leprosy, but even now its culture is extremely difficult, and the exact mode of infection in many cases is still mysterious. It can be inoculated, although this statement has been denied and again reasserted many times. It can certainly be conveyed by contact, and therefore the segregation of lepers is an extremely important point in prophylactic treatment. Persons living in contact with lepers frequently become infected, and the evidence afforded by the heroic life of Father Damien, who devoted himself to lepers and lived in their midst, thereby acquiring the dis- proves conclusively the con-

tagiousness of leprosy; and this is no isolated instance. It is, however, possible for medical men and nurses to attend daily to lepers without becoming infected.

A fish diet has been considered a possible means of causing leprosy in those predisposed, or else it confers the predisposition towards the attack of the organism. The evidence, however, in favour of this theory is inconclusive.

Pathological Anatomy—The typical tubercular nodules of leprosy consist of granulation tissue containing enormous numbers of the bacilli, partly intracellular and partly lying between the cells. These nodules involve the deeper parts of the skin and mucous membranes, and especially the conjunctiva, the cornea, and the larynx, and deep-seated ulceration is associated with the nodules. In the anaesthetic type of leprosy there is a form of neuritis due to the extensive involvement of the affected nerves by the bacilli, and so causing remarkable trophic changes in the skin and other tissues.

Clinical Features.—*Type I. Tubercular Leprosy.*—Preceded by attacks of fever and malaise there is a preliminary discoloration of areas of skin which are the future sites of the tubercular nodules. These are brownish-yellow in colour, and are sometimes hyperaesthetic: they are termed macular leprosy. In certain patients the pigment disappears and the skin becomes white, coincident with the appearance of local anaesthesia, while the hair over the affected parts falls out. At a later period the tubercular nodules develop, and their distribution gives the face the peculiar leonine expression so characteristic of the disease. The forehead, just above the eyes, and the lips suffer specially. At the same time similar nodules appear on the mucous membranes of the mouth and larynx. Very deep-seated ulceration may follow: in one leper seen, more than half of the face was eaten away, the patient presenting the most ghastly spectacle imaginable. The eyes are frequently affected and sight is lost. Preceding each fresh crop of nodules there is often considerable pyrexia.

Type II. Anaesthetic Leprosy.—In this form the bacilli attack the nerves, causing pains, hyperaesthesia, and eventually numbness. Soon trophic changes ensue in the affected skin, and extraordinary contractions of fingers and toes may result, and owing to necrotic changes the phalanges may be gradually

lost until the hand is completely mutilated. Bullae form over affected joints preparatory to this destructive process. Macular spots, similar to those seen in the first type, appear on the skin of the body as well as on the limbs; but although they give rise to anaesthetic areas, tubercular nodules do not develop as a general rule. The ulnar and peroneal nerve trunks are usually the first to become affected. This type runs an extremely slow course, and many patients live for years, gradually becoming more crippled and unable to use their hands and feet.

A combination of the two forms, as already indicated, is by no means rare.

Diagnosis.—The macular eruption is very typical, and especially when the anaesthetic stage has been reached. In the tubercular form of the disease the leonine appearance of the patient is almost distinctive. The absence of anaesthesia in *syphilitic* skin lesions prevents a mistake being made. In *syringomyelia* there are no maculae and no destruction of fingers and toes such as occur in anaesthetic leprosy. It is impossible, with care, to confuse the perforating ulcer of the foot of an *ataxic* patient with leprosy.

Prognosis.—In the anaesthetic type long life and even comparative usefulness is assured, while there is an idea, possibly erroneous, that this type is less infectious to others than the tubercular form, in which ulceration is so much more constantly present.

Treatment.—There is no remedy which can be truly called a specific for this disease. For many years certain of the essential oils peculiar to India and other lands have been recommended. These include gurjun and chaulmoogra oils used both internally and externally. Antivenene (Calmette) has been tried, and also X-rays, but it seems probable that successful treatment must depend on serum therapy. Leproline affords a means of diagnosis, by the reaction which occurs after its injection, similar to what is seen with tuberculin in tuberculosis, and its possible efficacy in treatment of leprosy is supported by some published reports of cases. Careful segregation and the prevention of marriage between lepers and healthy persons have done much to stamp out leprosy in Western Europe, although the problem remains as serious as ever in India and many other countries.

XXXVI. SYPHILIS

A DISEASE due to the Spirochaeta pallida discovered by Schaudinn. It may be inherited or acquired by infection, and both the ovum and spermatozoa are capable, the first of being infected from the mother, and the second of carrying infection. In syphilis there are three stages. The *primary* stage is only found in acquired cases, and the manifestation is the appearance of a local primary sore at the site of infection. The *secondary* stage follows in six weeks to two months' time, when rashes appear on the skin and superficial sores on the mucous membranes. The *third* or *tertiary* stage succeeds the secondary at an interval of six months to several years, and in it deep-seated granulomatous tumours develop in the muscles and in many parts of the body.

It should be stated definitely that syphilis is quite distinct from, although it may be associated with, soft sores (soft chancres) and gonorrhoea.

Etiology.—The Spirochaeta pallida is actively motile, very difficult to stain, and occurs in great numbers in the discharges and the tissues affected. It is 10 to 15 μ in length.

The modes of infection are certain, although very various. The disease is most often acquired during sexual intercourse probably due in some cases to a slight local abrasion, in others to infection through healthy mucous membrane; but kissing, sucking the nipple, vaginal examination where there is a lesion of skin on one of the examining fingers, circumcision where the operator is syphilitic and performs the rite with the teeth, and vaccination from a syphilitic child, are all fairly common methods of communicating the disease. Dirty dental instruments, drinking-cups, smoking the same pipe as a syphilitic patient, are also a few of the other ways by which infection may be conveyed. The primary sore, the condylomata, and the mucous plaques are intensely infective. Blood is probably capable of conveying infection, but saliva, tears, sweat, and milk are in themselves harmless unless mixed with discharge or blood. The semen is said to be innocuous, but this is very questionable, as the spermatozoa can almost certainly transmit the disease. In hereditary syphilis the discharges from syphilitic sores are infective. It is stated

that the secretions from tertiary sores are non-infective, but this is more than doubtful, although they are certainly not virulent. In inherited syphilis [illegible] the father or mother may give the infection, and a sy[illegible]e child may be born by a woman who is not herself [illegible]ected but in such a case, according to Colles' law, the mother is protected against syphilis, although the child in suckling may give the infection to a wet nurse. Similarly, a healthy child born of syphilitic parents is said to be immune, but this statement, made by Profeta, requires more circumstantial evidence to prove its truth. Syphilis acquired from the mother is always more severe than infection from the father.

There are some curious facts about syphilis and immunity. Certain peoples and races appear to be becoming saturated with the disease, so that in them it runs a less severe course. In other races not previously exposed to it, syphilis, if introduced, assumes a most malignant type, and where it is transmitted from an individual of one race to a member of an entirely different race, it is apt to appear in an aggravated form. Should an individual have one attack of syphilis, he is usually protected against a second attack, and, if reinfected, the manifestations are generally much milder; rarely is the converse of this true.

Pathological Anatomy.—I. The *Primary Sore*, or hard chancre, consists of a great infiltration of the deeper parts of the true skin with small round cells. There is proliferation of the connective-tissue cells, and the small blood-vessels undergo endarteritis obliterans. The cell proliferation appears to be greatest in and round these small vessels, and there is a hard indurated edge due to cell proliferation and exudation. These chancres tend to break down, and, as already noted, the discharge is very infective. The glands in the neighbourhood swell up and become indurated. In old chancres there may be seen the epithelioid cells and even the giant cells so often associated with tubercle nodules, but the latter are not common in syphilis.

II. The *Secondary Affections* are very various. There are superficial ulcers on the tonsils and pharynx: these are often symmetrical, and whitish patches appear on the tongue and lip, said to be most marked in smokers. These lesions depend on small-cell infiltrations, and they are due to the syphilitic

virus in the blood. The secondary roseolar rashes are due to hyperaemia, and infiltration with small round cells and pus-forming organisms may be present in addition, leading to pustular eruptions more or less local. Iritis is also a common secondary lesion. These secondary lesions are much less infective than the primary chancre, although they can transmit the disease, and a patient is not considered to be free from syphilis until at least two years have elapsed since the last manifestation disappeared.

III. The *Tertiary Affections* such as gummata are more rarely seen now, owing to prompt treatment at earlier stages.

The essential lesions are gummata or tumours, made up of granulation tissue, which may be distributed in a most impartial fashion throughout the body. They may occur anywhere, may be soft or hard, minute, or as large as a small orange. The centre tends to become gelatinous-looking, or even to caseate: the periphery is firm and fibrous. This tendency to central degenerative changes and thickening of the fibrous capsule is typical of older gummata, and causes the remarkably deep cicatrices seen, for example, in the liver. A number of these may occur together. They are common in muscles, skin, bones, periosteum, and internal organs, including the meninges of the brain. Superficial gummata are apt to break down and form ulcers: the internal ones remain long quiescent, and eventually may be absorbed, leaving a puckering or scar, or may become the site of calcareous deposition. Gummata tend to appear wherever the circulation of blood is least active. They are met with in the septa of muscles, and in fibrous structures in connection with joints. They may be found in the fibrous perichondrium of the larynx and in the median septum of the tongue. They also occur where there has been an injury; hence the syphilitic nodes on the tibia, and the development of gummata about a joint which has been bruised or overstrained.

In the skin tertiary lesions are deep seated, and lead to permanent disfigurement from cicatrisation.

In connection with the arteries there is endarteritis obliterans, in which the inner coat undergoes enormous thickening, usually at one side. The newly formed tissue of spindle-shaped cells develops just internal to the endothelial cells of the intima, and later there is a marked cellular invasion

of the media and adventitia. The arteries most generally affected are the cerebral vessels, and the disease may go on until thrombosis occurs from interference with the lumen. Endarteritis obliterans develops sometimes in hereditary syphilis, but generally in the acquired form and in the tertiary stage. In some cases a syphilitic periarteritis is present, in which there is gummatous formation, especially in the adventitia.

Amyloid or waxy degeneration is a condition commonly associated with tertiary syphilis, and about one-third of all cases of waxy degeneration are of syphilitic origin.

Lastly, there are many distinct but late results of syphilitic toxins which may be properly referred to here. These specially involve the nervous system, and include locomotor ataxia and general paralysis of the insane. Although some doubt still exists as to this relationship, there seem to be well-grounded reasons for associating not a few nervous diseases with syphilis.

Clinical Features of Hereditary Syphilis.—The child at birth may be hall-marked with the disease. The chief phenomena are snuffles, fissures at the angles of the mouth, or sometimes at the anus, and very generally an eruption on the skin. In most cases, however, the child is merely puny, and the characteristic features only develop about the 4th to the 12th weeks. In other instances the child is born healthy but does not grow well.

(1) Snuffles is always present, and is amongst the first and most obvious symptoms. It may be so severe as to impede breathing when the child is at the breast, and the nasal discharge may become purulent, and eventually the cartilages and bones necrose, which gives the peculiar flattening at the root of the nose so characteristic of congenital syphilis. Sometimes the Eustachian tubes are involved, and deafness results.

(2) Skin eruptions are seen. These include the typical fissures at the angles of the mouth and mucous patches on the lips, inside of cheek, pharynx, and tongue. The secretion from these is virulent, and may easily infect a wet nurse or other individual, although not the mother (Colles' law, *vide supra*). There are in addition typical copper-coloured, erythematous rashes, well seen about the nates, and the hair may fall out.

(3) The permanent upper incisor teeth show the peculiarities described by Hutchinson. They are peg-shaped, short, and have a notch or groove in the cutting edge, due to a defect in the enamel: this groove is deepest in the middle of the tooth. The upper canines may also be peg-shaped and shortened, but are not characteristic.

(4) The child soon looks old and stunted: the liver and spleen are found to be enlarged, but the lymphatic glands often escape or suffer slightly. There may be so-called syphilitic cirrhosis of the lung, but it is extremely rare. Bones, cartilages, and even joints may show definite changes. These consist in enlargements at the junction of epiphyses with the shafts of the long bones, ribs, and clavicles. There may also be periostitic gummata, but these develop at a later period; they cause great thickenings, which are often symmetrical, of the bones of the arms and legs. The joints more rarely undergo painless symmetrical swellings, which may subside or give rise to deformity. The knees are said to suffer most commonly.

(5) Keratitis is frequently seen, but it often develops later on. Both eyes are affected at once: the cornea has a ground-glass-like appearance, and there is marked ciliary congestion. Photophobia is present, and sight is greatly diminished. This may last for months, and rarely clears up entirely, generally leaving definite opacities behind. Iritis is sometimes present.

There is much difference of opinion as to whether these hereditary syphilitics should marry. Apparently they may, and do, have perfectly healthy children.

Clinical Features of Acquired Syphilis.—The stage of inoculation varies probably from 2 to 7 weeks, 5 being the average. Then the *primary* chancre or sore appears, beginning at the site of inoculation, and forming first a small papule, later a more marked swelling with flattened apex and indurated almost cartilaginous-like base. The degree of breaking down depends on secondary causes, such as local irritation, and, in fact, the primary chancre will not develop to the same marked extent in every case, cleanliness and freedom from irritation greatly limiting its size. In most cases the primary sore remains for 2 or 3 months, and then heals, leaving a scar.

The lymphatic glands in the groin swell and become hard, but they are painless, and do not suppurate unless irritated. Sometimes the line of infection may be traced by the palpable infiltrated lymphatics. The next or secondary stage is reached from 6 to 12 weeks after the primary chancre has appeared.

Secondary Stage.—(1) The rashes vary in extent and degree. There is usually a roseolar rash, which comes out coincidently with a degree of pyrexia, varying in different patients, and lasts for 2 or 3 weeks. In severe cases pains in head, back, and legs, general malaise and discomfort are present: in others these phenomena are slight or absent. (2) At the same time the throat is sore, superficial ulcers and mucous plaques appearing on the tonsils, tongue, and lips. These mucous patches are raised, moist, and whitish in colour, with sharply defined edges. (3) Condylomata or warts form most frequently about the anus or vulva, but may also be seen on the lips. (4) The skin eruptions, other than the typical early roseolar rash, have certain well-marked and classic features. They are polymorphous (by which is meant that papules, vesicles, pustules, etc., may all occur at one and the same time), coppery coloured, symmetrical in position, have a distinct tendency to form ring-like, crescentic, or serpiginous lesions, and imitate many other skin affections, while they have a special predilection for the inner surface of the thighs, the region of the anus, the flexor aspects of the limbs, the face, and the forehead. Occasionally pigmented areas mark the sites of old eruptions, but this is not constant. A form of alopecia is not uncommon, the hair often thinning, although it may fall out in patches, while syphilitic onychia, or inflammation of the nails of the hands and feet, may occur.

Laryngeal catarrh may cause pain on swallowing and aphonia. (5) Anaemia is almost always present; the patient suffers from the toxin, but the degree varies with each case. Often debility and loss of flesh accompany the anaemia. Certain writers lay much stress on the frequency of headache and sometimes trigeminal neuralgia in syphilis. These are probably associated with the anaemia and debility, and are commoner in females.

(6) Eye affections are very typical, and especially an exudative iritis which develops some weeks, at least, after the secondary stage has commenced. It is symmetrical, but one

eye suffers first, and there is ciliary congestion and generally photophobia and pain. There may be retinitis or choroiditis, but they are rare. Deafness may be due to the spread of inflammation from the throat up the Eustachian tube.

Although a number of signs and symptoms have been enumerated, it must not be supposed that all even of those numbered are necessarily present: sore throat and possibly a slight roseolar rash, with some falling out of the hair, may alone indicate the existence of the second stage of syphilis.

The *Tertiary Stage* in a treated case should never appear at all, but 6 or 8 months, and even a number of years after syphilis has been acquired, tertiary symptoms may develop notwithstanding careful attention. The 3rd or 4th years are the most frequent periods for tertiary phenomena. In this stage, as indicated under pathological anatomy, the (1) skin eruptions are deeper seated, (2) gummata develop, and in time (3) the arterial changes due to the disease manifest their presence clinically.

There is not the same symmetry about tertiary manifestations of syphilis, and the lesions are deeper seated, and therefore more serious in their effect, but they are less infective to other persons.

1. The skin eruptions include rupia, in which limpet-like crusts cover a deep-seated ulcer, and serpiginous syphilides which resemble lupus in their appearance, but have a more crescentic margin. The polymorphous character of syphilitic eruptions may be demonstrated by other types of eruption being present as well. Sometimes a psoriasis-like syphilide develops, especially on the legs and arms, but, unlike true psoriasis, the flexor aspects do not escape. Bullae also appear (called syphilitic pemphigus) on the arms and legs, but they become pustular, and unless carefully treated are indolent in healing.

2. Gummata develop in many internal organs, although they may appear also in subcutaneous tissues, as, for example, under the skin of the scalp, buttocks, breasts, and many other regions, where they form painless tumours, while sometimes they develop in bursae. They also grow in muscles, forming slightly painful, slowly enlarging tumours, which specially involve the tongue, sterno-mastoid, masseter, supra- and infraspinatus, gastrocnemius, and the rectus femoris muscles. Sometimes

gummata develop in connection with the joints, and the joints may swell up, as they occasionally do in the secondary stage. The periosteum is very frequently involved by gummata, and the tibia, ulna, clavicle, and sternum are specially apt to suffer, but any bone may be affected, depending much on a preceding injury.

(3) The syphilitic endarteritis obliterans, and the less common syphilitic periarteritis, which is a development of gummata in the tunica adventitia, yield most definite symptoms in connection with the nervous system, although the coronary arteries are sometimes also involved, causing serious degenerative changes in the myocardium. It should be mentioned that syphilis may predispose to aneurism by weakening the arterial walls.

Choroiditis is a common tertiary manifestation in syphilis. The syphilitic affections of the ocular nerves will be mentioned under the diseases of the nervous system.

Tertiary Syphilitic Manifestations in the different Systems (which have not as yet been mentioned).—1. *Alimentary System.*—Gummata of the *tongue* have already been described; they may lead to ulceration. Gummata may develop in the *pharynx*, rarely in the *œsophagus* and *stomach*, and occasionally in the *small intestine*, *cæcum*, and *rectum*. There is always a greater or less tendency to stenosis as the result of the cicatrisation.

In the *liver* there are (1) perihepatitis, which is sometimes of syphilitic origin, and (2) gummatous hepatitis, in which gummata of varying size develop in the interstitial tissue. Gummata caseate in the centre, and tend to produce marked scarring or cicatrisation, which may be palpable, and may interfere with portal vessels or bile-ducts, causing ascites or jaundice. They are often associated with waxy degeneration. This form of hepatitis is not uncommon in acquired, but may also occur in congenital syphilis. (3) Diffuse hepatitis, which is congenital in origin, consists of an extensive invasion of small cells, and the liver cells are much compressed. The organ is enlarged, and jaundice is a common result.

2. *Haemopoietic System.* The *spleen* is often waxy and may contain gummata. In such cases it is generally enlarged. The *lymphatic glands* are affected in secondary syphilis, as already mentioned, but even in tertiary syphilis there may be

induration secondary to gummata in the line of the lymphatic channels connected with the glands in question.

3. *Circulatory System.*—*Cardiac* gummata are not common, and are usually found in the wall of the left ventricle, but the heart wall may be degenerated if the coronary arteries are affected as already mentioned, and the form of degeneration is generally fibroid. Syphilitic *arteritis* has already been referred to under pathological anatomy. Aortic incompetence is not infrequently of syphilitic origin.

4. *Respiratory System.* The *nasal* syphilis so common in hereditary cases has been described. The *larynx* is frequently involved, gummata may form on the epiglottis, and there may be perichondritis of the arytenoids or the cartilages of Wrisberg. Extensive ulceration may develop, and the true cords, the ary-epiglottic folds, and almost the whole larynx may suffer, the epiglottis being often affected at its free edge and on its buccal surface. Stenosis of the glottis is a common result, often associated with the formation of subglottic membranes. The differential diagnosis between syphilitic and tubercular laryngitis will be described under the latter disease (p. 134), but it may be stated here that syphilitic ulceration is more painless, and the ulcerative process is very rapid. The pain of carcinoma greatly exceeds that felt in syphilis. With prompt treatment syphilitic disease of the larynx readily yields, although for the resultant stenosis it may be necessary to dilate with bougies.

The *trachea* and *bronchi* may be the site of gummata, and perichondritis, ulceration, and eventually stenosis may be caused. This leads to marked inspiratory stridor, and, in the case of the trachea, great dyspnœa.

The *lungs* may show two types of syphilitic affection. (1) White hepatisation or pneumonia, met with in the fœtus, in which the lung is in whole or part consolidated, due to a syphilitic fibrosis of the air-vesicle walls, while the arteries are markedly thickened, the lung being almost bloodless. If extensive, it is not compatible with life. (2) Gummata may form more often near the root of the lung, and associated with them is a syphilitic thickening of interstitial septa extending into the lung from the root, in which the gummata are to be found. The arteries often show syphilitic endarteritis, the neighbouring pleura may be thickened, and there is generally peribronchial thickening as well. The clinical features, such as cough and purulent

expectoration, closely simulate phthisis, excepting that the lesion is chiefly confined to the root of the lung, and there are no tubercle bacilli. Syphilitic disease of the lung is rarely diagnosed in the adult, partly because it only occurs in a very small percentage of cases of tertiary syphilis, and partly on account of the absence of really typical symptoms or signs.

5. *The Genito-urinary System.*—Gummata may occur in the *kidney*, but are rare. Waxy degeneration is not uncommon and may be associated with a form of nephritis which is possibly but not certainly of syphilitic origin (see p. 545).

The *testicle* is frequently involved, generally by gummatous masses, which invade the body of the organ, and not the epididymis, as in tubercle. It is painless and does not suppurate. Both testicles may suffer.

6. *The Nervous System.*—Gummata may occur varying in size from a pea to a small tangerine orange. They are almost invariably found growing from the *meninges*, although they may invade the *brain* tissue and usually involve the cerebrum while oculo-motor and other cranial *nerves* are often affected. Gummata in connection with the *cord* are much more rare. There may also be a gummatous development in the adventitia of the cerebral arteries called periarteritis nodosa. Endarteritis obliterans is a common tertiary lesion, and specially affects the arteries whose branches form the circle of Willis.

As the result of gummata we may note the clinical features of an intracranial tumour, with, in most cases, according to the writer's experience, optic neuritis. Secondary inflammation is not uncommon, encephalitis if in the brain, myelitis if in the cord, while gummata, involving cranial nerves, may cause paralysis more or less complete.

Patches of sclerosis develop as the direct result of the toxin or toxins often long after the primary infection, and locomotor ataxia is a good example of this.

Cerebral syphilis may thus directly or indirectly give rise to an intracranial tumour, a hemi- or mono-plegia, hemianopsia if the occipital lobe is involved, and paralysis of different cranial nerves, but especially the ocular ones. It may also produce great headache and giddiness from interference with the blood-supply of the brain, and convulsive seizures of the epileptiform type are not uncommon. There is little doubt

that general paralysis of the insane is now believed to be of syphilitic origin.

Spinal syphilis is similar in that gummata and endarteritis may be present, although less frequently than in the brain. Probably spinal meningitis is not uncommonly of syphilitic origin, and myelitis and patches of sclerosis in the cord may be due to the syphilitic toxin. It should also be repeated that besides locomotor ataxia, which is undoubtedly due to syphilis, spastic paralysis and other cord lesions, may in some cases be of specific origin. It is most important in diagnosis to remember the possibility of syphilis, to try to get a definite history from the patient, and in every doubtful case to try antisyphilitic treatment. Sometimes other evidences of syphilis may be noted, which are either typical in themselves or may support a syphilitic hypothesis.

Diagnosis of Syphilis. Many patients refuse to admit syphilis, and we may require to look for evidence of a primary chancre, or of secondary or tertiary lesions. A history of the hair falling out, with sore throat and skin rashes, suggests secondary phenomena, and the deeper-seated tertiary lesions leave scars. In women repeated miscarriages are very common, due to placental degeneration. Examine the tibiæ for nodes, the eyes for keratitis or choroiditis, and note if the testicles have suffered. Lumbar puncture should not be neglected in chronic syphilitic diseases of the central nervous system because a marked lymphocytosis in the cerebro-spinal fluid is usual. The Wassermann reaction, although positive in some other diseases according to various authorities, is valuable, and it has demonstrated that the supposed immune mother (Colles' law) and the supposed immune child (Profeta's law) are really suffering from latent syphilis (see p. 112).

Antisyphilitic treatment is so successful in specific cases that a diagnosis may only be definitely arrived at after a prolonged course has been carried out.

Prognosis.—In most cases a favourable result is assured if treatment has been carefully, thoroughly, and conscientiously attended to. In untreated patients tertiary manifestations are very apt to cause permanent damage.

Treatment. It is unnecessary to enter into a lengthy discussion of the prophylaxis of syphilis. Continence is possible, and, as Sir William Osler along with many others

has wisely remarked, hard work, physical and mental, does much to keep the lower side of human nature in check.

The question of State regulation of vice has caused many stormy scenes. Unfortunately the transgressor of the moral laws in question is not the only sufferer, for he may hand down a heavy load of misery to his children. Syphilis cannot be considered cured short of a period of two years from the last appearance of the rash or other manifestations, and therefore marriage, before that period has elapsed, is an inexcusable offence; but with all the care possible a recrudescence of syphilis may appear even after that period has passed. It is, however, a certain satisfaction to know that tertiary lesions are not nearly so infective. The extension of our knowledge as to cerebral and spinal syphilis leads us to hope that hitherto incurable diseases such as general paralysis of the insane and locomotor ataxia may no longer be relegated to that category.

For the primary chancre, cleanliness is the only treatment. The knife is rarely resorted to for the purpose of excision, because the futility of the operation is evident, the organisms passing through the body at a rapid rate and being in no sense limited to the primary site of inoculation.

For the secondary manifestations, mercury is the great remedy, and it may be used in several different ways, but it is wise to get the teeth overhauled beforehand by the dentist, and everything done to keep up the general health during treatment.

(1) *Internal Administration.*—Hydrargyrum cum creta may be ordered in small doses (gr. 1-2), with a similar quantity of Dover's powder, 4 times a day. The liquor hydrargyri perchloridi may be taken internally in drachm doses (gr. $\frac{1}{16}$) thrice daily, or the biniodide of mercury in the same dose may be taken in pill form. Such treatment, begun early and steadily persevered with for months, is most efficacious in aborting the secondary stage. Some authorities give mercury for 2 months, then a month without, then 2 months with and 3 months without, so that in 2 years the patient has 10 months' treatment and 14 months without (Fournier). In the later years 6 weeks' courses of mercury are given—4 courses in the 3rd and 4th, and 3 courses in the 5th year. Other authorities, such as Hutchinson, give long continuous treatment. Guard against salivation by careful attention to the teeth and by using

an astringent mouth-wash, and check diarrhœa by dieting and opium.

(2) *Inunction* is another favourite method, the ointment—often the unguentum hydrargyri—being rubbed into the skin night and morning, and about 40 to 50 grains of the ointment used at a time. Rub for thirty minutes, and, if the rubbing is not performed by the patient himself, the hands should be protected. The sides of chest and abdomen and the inner aspects of the thighs are often selected, and the area of application should be changed each time. Mercurial soaps are often used, the lather being well rubbed in and left to dry. The treatment may be dropped one day out of seven.

(3) *Fumigation.*—The patient sits on a chair covered with blankets so as to keep in the fumes. About 20 to 40 grains of calomel are volatilised in a small capsule over a flame, and boiling water is also used, so that the mercury is absorbed during a mild steam bath. About 20 to 30 minutes should suffice for the application.

(4) *Hypodermic injections* may be given deeply into the muscles, either the perchloride or biniodide being preferred gr. $\frac{1}{20}$-$\frac{1}{4}$; intravenous injections have been tried, but are not commended. Care should be taken with intramuscular injections to avoid the risk of local necrosis, due generally to the injection being too superficial. The injections are only given once every day, or every two or three days, depending on the dose administered.

For infants and children, the subjects of hereditary syphilis, inunction is one of the best methods of administering mercury, and the ointment may be applied on the child's binder. Hydrargyrum cum creta is another favourite in doses of $\frac{1}{20}$ to $\frac{1}{2}$ grain given internally thrice daily, the dose depending on the age of the patient.

For tertiary manifestations of all kinds potassium iodide should be administered, beginning with 10 grains, thrice daily, and increasing until twice or thrice that dose is being taken. Attention to the bowels and general health is usually sufficient to prevent iodism with its resultant sore throat and severe coryza, but there are patients who cannot take it. Sometimes doubling the dose lessens iodism; still in certain cases it is wise to stop treatment from time to time.

The new method of treatment devised by Ehrlich is the

use of "606" or Dioxy-diamido-arseno-benzol or salvarsan. It has an extraordinarily rapid action in secondary syphilitic manifestations, and evidence is accumulating that its action in tertiary syphilis is also curative. Cases of hereditary syphilis also benefit, but it is less satisfactory in the parasyphilitic lesions, such as tabes and general paralysis of the insane. It may be given either by deep intramuscular injections (into the buttock), which are painful and leave an indurated area for some weeks (·3 grm. to ·6 grm.), or intravenously in saline solution in doses of ·3 grm. to ·5 grm. The intramuscular injections leave a big dose of arsenic, which is slowly excreted, while the intravenous are got rid of in four days. Some cases have terminated fatally, and "606" is contraindicated where there is serious cardiac or renal disease, and where there is early optic neuritis or atrophy.

For syphilis involving the internal organs, besides the specific treatment just mentioned, symptoms such as dropsy, headache, etc., may call for our help.

Carefully regulated diet and a minimum of exercise are advisable, and the best holiday for a syphilitic patient is a sea-voyage. Marriage, as already stated, should be discouraged for long after the period of infection, and should be absolutely forbidden until two years after the last obvious symptom has disappeared.

It is doubtful to what extent syphilis, properly treated, shortens life, although neglected syphilis certainly has every chance of so doing. Many insurance offices add a few years to the age of the aspirant for insurance, others defer the petition until all trace of the disease has disappeared.

XXXVII. GONORRHOEAL INFECTION

It is only the medical aspects of gonorrhoea with which we have to deal here.

While gonorrhoeal rheumatism is most important because of its frequent occurrence, it must be remembered that the gonococcus may produce a general gonorrhoeal infection as well as a localised involvement of joints.

Etiology. The gonococcus infects mucous membranes, and is usually acquired by sexual intercourse, although the

conjunctivae, as in new-born children, may be the primary site of infection. The organism has been found in the blood.

Pathological Anatomy.—The gonococcus sometimes finds its way up the urinary tract, or by way of the Fallopian tubes it may infect the general peritoneal cavity. In the joints infected by the gonococcus there is an effusion, rarely purulent, but containing the organism, and there is often inflammation in the sheaths and tendons, and in connection with bursae: the endocardium is not infrequently affected, while the conjunctivae may also be involved.

I. *A General Gonorrhoeal Infection.*—In this condition the patient usually dies of acute gonorrhoeal septicaemia, and often very rapidly. Occasionally there are numerous pyaemic foci scattered throughout the body.

II. *Gonorrhoeal rheumatism* is more common in men than in women, and generally occurs before middle life. There are several types of the affection which vary considerably. Many joints may be involved, and often the smaller joints. In other cases a few joints alone suffer, while sometimes a single joint is affected. One peculiarity of gonorrhoeal rheumatism is the fact that certain joints suffer in gonorrhoeal arthritis which, as a rule, escape in other forms of rheumatism. These are the temporo maxillary, the sacro-iliac, the sterno-clavicular, and the intervertebral. The effusion does not, as already stated, generally become purulent, but the affection is long standing, and does not yield satisfactorily to treatment.

III. *Gonorrhoeal bursitis and gonorrhoeal involvement of tendons and fibrous tissues* are not uncommon, either associated with arthritis or distinct from it.

IV. *Gonorrhoeal conjunctivitis* is generally the result of infection, and may cause a severe iritis, possibly with ultimate loss of the eyeball.

The **Prognosis** is favourable as regards life, in most cases, except in general gonorrhoeal infection; but gonorrhoeal arthritis is always tedious and may cause great and permanent deformity.

Treatment.—Potassium iodide is certainly a remedy which should be tried, but salicylates are useless. Tonics such as iron, arsenic, and cod-liver oil are invaluable. Locally a good deal may be done by hydropathy, and in some successful cases the affected joints have been incised and washed out

with antiseptic lotions. Vaccine treatment has been found beneficial in gonorrhoeal arthritis. Serum therapy is not yet so certain in its results.

XXXVIII. TUBERCULOSIS

THERE is no disease so widespread or so fatal to the human subject as tuberculosis. It is specially common in cities and much less frequent in country districts. Fully 11 per cent of the total mortality is due to one or other of the many forms of tubercular disease.

Tuberculosis is to be found in all countries, but is certainly less prevalent in the tropics than in more temperate regions. At high altitudes where, owing to the excessive purity of the atmosphere, there is less risk of propagation of the organism, tuberculosis is rare, and were it not for the number of phthisical patients who frequent the Alpine health resorts, the local death-rate from the disease would be very low. Little can be said with regard to racial tendency to the disease, because it depends more on the sanitary conditions of home-life than on any difference as regards nationality. Tuberculosis has greatly diminished during the past decades, and there is no disease with regard to which such satisfactory progress has been made, due more to sanitary reform than to any new medicinal treatment.

Tubercle bacilli, discovered by Koch in 1882, are short rods 1·5 μ to 3·5 μ in length (a red blood corpuscle is about 8 μ and ·3 to ·5 μ in breadth. They are slightly curved, and vary in length according to the age of the culture. Some show clear, refractile spaces which do not stain. These are not spores, and by many authorities are thought to be signs of decay. Sometimes the bacilli occur in threads, and occasionally they have delicate, branching processes, which are cultural peculiarities.

The **staining method** usually employed is the Ziehl-Neelsen method—fuchsin grm. 1, phenol 5 per cent solution in distilled water grm. 100, absolute alcohol grm. 10. This should be raised to the point of steaming, with the cover-glass, film-side downwards, floating in it, and the cover-glass must be left in the stain for 2 minutes. Then, after washing with water, decolorise

in a 20 per cent solution of sulphuric acid and water; then wash in weak alcohol, and counterstain in a weak aqueous solution of methylene blue. The bacilli are acid-fast, which means that they retain the fuchsin in spite of the action of the sulphuric acid—a peculiarity which they possess along with a very few other organisms, such as the leprosy bacillus, timothy grass bacillus, smegma bacillus, etc.

Cultures may be made on blood agar, glycerin agar, or potato, and are kept at blood-heat. They grow slowly, and the colonies are greyish-white in colour.

There is no question of the existence of differences both in form and also in virulence of the tubercle bacillus.

The Virulence of the Tubercle Bacillus.—Many patients suffering from acute pulmonary tuberculosis spit millions of tubercle bacilli in the daily expectorations. Dried sputum remains virulent for months, although after a time it gradually loses its infective power. The chief danger is unquestionably in dried sputum, dust getting into the air, and dust which may contain the tubercle bacillus. Bacilli retain their virulence for several months in water, and freezing has no effect in destroying the organisms. Moist heat is more destructive than dry heat, and dried bacilli survive in a considerably higher temperature. Most authorities agree that at 70 Centigrade the tubercle bacillus can be killed in 5 to 10 minutes. Sunlight is one of the best natural sterilising agents for the bacilli. It destroys them in a period varying from minutes to hours, depending on the strength of the solar rays and the thickness of the infective material to be acted on. Exposure to the atmosphere apart from the sun kills the bacilli within a week.

Carbolic acid solution of 1 in 20 and corrosive sublimate of 1 in 1000 may be safely depended on for the purpose of destroying tubercle bacilli.

The Toxic Products of the Tubercle Bacillus.—It is hardly possible to discuss here the various toxic agents which have been separated from the tubercle bacillus by different workers. The dead bacilli have been found capable of stimulating a localised inflammation round about tubercular foci, and it is partly in this way that Koch's tuberculin acts.

The Distribution of the Bacilli.—The bacilli are found in greater or less numbers in all tubercular lesions, although

in those which are markedly caseous the number present may be very small. There is a tendency for the local spread of the bacilli chiefly by the lymphatics, although if the organisms enter the blood-stream, they may be diffused widely throughout the body. Pathologists are agreed that tubercle bacilli may remain for many years quiescent in an old tubercular focus, and that, given the opportunity, they may then multiply, and these foci may become once again active centres for the diffusion of the disease. The urine frequently contains bacilli in cases where the kidneys are involved, but milk and sweat have not been proved to be infective unless in the case of the former there is a tubercular lesion of the nipple or breast.

The Histology of the Tubercle Nodule.—There is nothing really distinctive in the tubercle nodule apart from the presence of the specific organisms.

According to Baumgarten the *tubercle bacilli*, having reached the focus where a tubercle nodule is about to develop, proliferate, and this is followed by a multiplication of connective-tissue cells, endothelial cells of blood-vessels, and possibly other cellular elements, all of which may form the typical *epithelioid cells*.

Numerous *lymphocytes* now appear, not merely in the nodule, but also round its periphery, and polymorphonuclear cells accumulate also in the neighbourhood of the nodule.

There is invariably a *reticular matrix* or network, which acts as a supporting structure, and which probably develops from connective tissue.

Lastly, in many tubercle nodules *giant cells* may be present. These are generally formed from fusion of several epithelioid cells together, and nuclei may be seen round about the periphery of the cell. Giant cells are more frequently present in older nodules, and in them, and also in the epithelioid cells, tubercle bacilli are often found.

A tubercle nodule tends to caseate because it is extravascular, and the process is really a coagulation necrosis. Very often in place of, or secondary to, caseation, fibrous tissue replaces the degenerating nodule, or calcareous deposition occurs in the caseous material.

Not infrequently tubercle nodules run together to form larger tubercular masses, and this process may be well studied in tuberculosis of the lung. Many secondary changes may be

induced in connection with tubercular nodules, such as a localised catarrhal pneumonia round about the tubercular foci in the lung, and often suppuration.

Modes of Infection.—1. *Intra-uterine Infection.*—So far as we are aware, (*a*) spermatozoa do not carry infection, and very rarely at any rate is (*b*) the ovum attacked by the tubercle bacillus before it reaches the uterus; (*c*) placental blood may certainly transmit the infection to the foetus. Probably there are relatively few cases of intra-uterine tuberculosis, hereditary predisposition being rather evidenced in later life by the phthisical type of chest and a lessened power of resistance to the tubercle bacillus.

2. *Inoculation.*—The fingers may be inoculated from infective meat or pus, and the verruca necrogenica of pathologists is an example. Butchers and others exposed to tubercle in dealing with tuberculous carcases are also liable to infection, and washerwomen may suffer from washing infected linen. The opponents of vaccination cite a few cases where tubercle has been inoculated with the lymph.

3. *Inhalation.*—Dried sputum containing tubercle bacilli, whether on floor, handkerchiefs, clothes, or beard and moustache, may readily set free in the air numerous organisms, and the dust of the floor in a sick-room inhabited by a consumptive patient often contains many bacilli. Inhalation is evidently the chief mode of infection, and persons in attendance upon consumptives whose habits are careless and dirty are often themselves infected.

4. *Infection by Meat and Milk.*—Meat is not generally eaten uncooked in this country, and, besides, tuberculous meat is condemned by the authorities and destroyed, but cows suffering from tubercular disease of the udders give infective milk, and the frequency of involvement of the tonsils, cervical and mesenteric glands in children, is most easily explained on the hypothesis of an infective milk-supply.

It is now generally admitted, as originally stated by Koch in 1901, that bovine and human tuberculosis are due to different varieties of the tubercle bacillus, and that the former is a common cause of abdominal tuberculosis.

Conditions which predispose.—Those who live in stuffy, dark, ill-aired dwellings, and work amongst insanitary surroundings, are much more liable to be attacked, and unquestionably

nuns, lunatics, and prisoners who are kept in close confinement fall victims to the bacillus in large numbers, the average of deaths among such persons due to tuberculosis being proportionally very high. In the past decade the fall in the death-rate from tubercular disease has improved 30 to 40 per cent owing to sanitary and hygienic reforms.

There is a "tubercular" constitution, or, more correctly, a hereditary predisposition. These persons have typically ill-made chests, alar, flat, or long-shaped, and often a combination of these together, and they have a lowered vitality which renders them less resistant to the attack of the bacillus. Two types of face are common in such persons. The one type are small, delicate-featured, with fine skin, short upper lip, long eyelashes, and they are usually of short stature. The other type have irregular features, with coarse skin, thick lips, and are clumsy and frequently tall in stature. These two types are very often seen among those who fall victims to tuberculosis.

Age.—While tubercular disease is met with at all ages, pulmonary tuberculosis is most frequent between 20 and 40, and in young children the alimentary system is more apt to be the site of involvement than it is in the adult.

Occupations.—Many trades which are responsible for dust diseases predispose to tubercle, and persons exposed to sudden changes from great heat to cold, or whose occupation necessitates getting wet, are specially susceptible.

Disease.—Many lung diseases predispose, but especially pleurisy, often in itself tubercular. In diabetes mellitus death is frequently due to a secondary pulmonary tuberculosis, and in any heart affection, such as pulmonary stenosis, in which the lung suffers from a deficient supply of blood, there is a tendency to tubercular infection. All debilitating diseases may be said to predispose. Excessive lactation certainly is injurious, and in tuberculous women pregnancy, while there may be a temporary arrest of the disease, is generally followed after parturition by a rapid advance.

Diagnosis.—There are several tuberculin tests which are of value in the diagnosis of tubercular lesions.

(1) Koch's original tuberculin, at first in 1 millegramme dose, is diluted and injected hypodermically. Later, 5 or even 10 milligrammes may have to be given, but rather for purposes of treatment than diagnosis. Tubercular subjects

show a definite reaction, as evidenced by a rise of 2 or 3 degrees of temperature Fahrenheit accompanied by some malaise.

(2) Calmette's ophthalmo-reaction. The solution consists of $\frac{1}{2}$ to 1 per cent solution of tuberculin free from glycerine, and it is instilled into the eye (1 or 2 drops). In 3 or 4 hours marked local conjunctivitis develops in tubercular subjects, and subsides in most cases within a day without detriment.

(3) Von Pirquet's cutaneous reaction. The solution consists of 2 drops of old tuberculin diluted with equal parts of 5 per cent phenol in glycerine and twice its volume of normal saline solution. Three small areas of skin are scarified on the arm and the tuberculin applied to the upper and lower, the middle area being left as a control. A zone of congestion should develop in 24 hours and persist for 2 or 3 days, the control remaining unaltered. The test may also be carried out by applying T.R. tuberculin to one of two small blistered areas on the patient's back. The reaction is not infrequently positive in doubtful cases. It is much more reliable in children under 13; in adults there is increasing scepticism as to its trustworthiness.

Treatment. — All the important tubercular affections referable to the different systems are described under the diseases of the systems in question, and in connection with most of these the diagnosis, prognosis, and treatment are discussed. It only remains, therefore, to refer to the tuberculin treatment of tuberculosis and the more recent modifications of that treatment, so far as it is possible to speak with certainty. The old tuberculin originally devised by Koch had been largely given up, but either the original preparation or modifications of it have received more attention recently.

Sir A. E. Wright recommends the use of Koch's tuberculin T.R. in order that the patient's leucocytes may acquire a greater capability for the destruction of tubercle bacilli by phagocytic action. The procedure is as follows:—

The opsonic index or phagocytic index of one's own leucocytes for dead tubercle bacilli must first be ascertained. Serum-free corpuscles are obtained by washing the blood in a mixture of 1 per cent citrate of soda in normal saline solution, and later in normal saline solution alone. Then mix an emulsion of dead tubercle bacilli with the prepared leucocytes

and the serum of the normal individual who is to be examined. Incubate the mixture at a temperature of 37 Centigrade for 15 minutes. Prepare films, and stain by the Ziehl-Neelsen method, counting the number of tubercle bacilli in each leucocyte. As a general rule about 5 bacilli will be found within each leucocyte, and this may be taken to be a *normal* opsonic index, or equivalent to 1.

In the case of the tubercular patient his serum is used in place of that of the normal subject, because it is upon the serum that the test depends, and where the patient is suffering from chronic tuberculosis there is a marked fall below normal in the opsonic index, often reaching ·5, ·6, or even lower. Wright found that the administration of tuberculin after an initial fall for about two days often raised this opsonic index greatly above normal, and that this was associated with a distinct local improvement in the patient's condition during some ten days when the high tide of immunity persisted. A gradual fall to the old level followed, but an injection say once a week kept up this high tide of immunity.

In acute cases, however, the opsonic index is almost always much above normal, and therefore there is no need for tuberculin injections. Care must be taken, as recently pointed out, to test the opsonic index of patients, either always while resting, or always after a definite amount of exercise, because there is a rise in the index associated with exercise.

The administration of fresh doses of tuberculin should be coincident with the rise in the opsonic index of the patient, commonly described as the positive phase, and the rise of positive phase follows a fall or negative phase, the latter directly resulting from the injection of tuberculin. Inoculation during the negative phase, or too soon for the case in question, might still further lower the opsonic index. It is well, in treating a case, to begin with small doses of tuberculin, and not to exceed $\frac{1}{5000}$th milligramme at first.

It should not be forgotten that the tubercular lesion in man may be due to bovine tuberculosis, and therefore bovine tuberculin may have to be used. The whole subject of tuberculin treatment is still *sub judice*, opinion tending, however, towards its use in chronic cases of tuberculosis medical and surgical, and Tuberculin Dispensaries have been in

stituted from which good results have already been obtained. Koch's old tuberculin, tuberculin T.R., Béreneck's tuberculin, and Marmoreck's serum have all their supporters.

XXXIX. MALARIAL FEVERS

A group of fevers, which may be benign or malignant in type, in which the parasite occurs in the red blood corpuscles, thus producing anaemia and changes in the spleen, liver, and other organs which are mainly the result of the deposition of blood pigment.

Malarial diseases depend for their existence on a particular kind or kinds of mosquitoes, and consequently warm climates, associated with marshy ground in which mosquitoes can breed, are essential factors. The mystery surrounding malarial fevers has been elucidated by the invaluable work of Laveran in 1880, and later by the investigations of Ross in 1898, and others, with regard to the *rôle* which the mosquito plays in connection with the disease. Different types of malaria are met with in different countries, varying considerably in their severity.

Etiology.—As already stated, marshy ground tends to increase the mosquitoes which act as the intermediate host for the parasites. Unquestionably the natives of a country suffer less markedly than those who are only temporary residents, and persons before middle life are apparently specially liable to attack.

Benign Malaria usually Intermittent in Type

The Parasites.—There are two forms of parasites which may occur in this fever: the one parasite is the *tertian*, the second is the *quartan*. These terms mean that the organism sporulates every forty-eight hours in the tertian form, whereas in the quartan, sporulation occurs every seventy-two hours. There may be two groups of tertian parasites, so that the patient may have sporulation occurring every day; and similarly there may be three groups of quartan parasites, so that it is possible to

have a daily attack of fever where the quartan parasite is present. This is called *quotidian ague*. The possession of several groups of organism implies that the individual has been several times infected by mosquitoes constituting double tertian, and double or treble quartan, as the case may be. It by no means always happens that two or more series of parasites sporulate at an interval of a day, because two groups may sporulate on the same day, and then a day may pass without any attack of fever.

The Tertian Parasite.—These are small, colourless, somewhat ring-like bodies, with a distinct amœboid-like movement, a well-defined nucleus, and generally there is at one part of the ring a small quantity of pigment. These colourless bodies increase in size, and the red blood corpuscles, in which they are, become swollen and pale, the haemoglobin of the corpuscle being utilised by the parasite for the production of the pigment granules. The granules of pigment show rapid movement, and they eventually occupy a central position. Towards the end of the forty-eight hours the hyaline-looking parasite becomes divided into 12 to 24 subdivisions, and when this segmentation has occurred the condition is termed sporulation. These segments or spores become free in the blood-stream, whenever the red blood corpuscle forming the host breaks up, and at the same time the pigment granules escape also. The spores are now ready to infect a fresh series of red blood corpuscles and so keep up the succession of attacks of the fever. This sporulation or segmentation occurs almost exactly at the end of each forty-eight hours, and co-existent with the sporulation is the typical attack of fever.

The Quartan Parasite.—In this case the original ring-like parasite shows at first little difference from the tertian organism, only the amœboid movements of the parasite are slower, the pigment granules are coarser and darker in colour, and the segmentation gives rise to the development of 6 to 12 spores or segments in place of 12 to 24.

The sporulating tertian and quartan parasites are the asexual organisms, and after a period of 7 or 8 days sexually mature organisms develop, which are the micro- and macro-gametocytes described in their further stages under the extra-corporeal cycle in the mosquito.

MALIGNANT TYPE OF MALARIA, SOMETIMES CALLED AESTIVO-AUTUMNAL FEVER, WHICH IS GENERALLY REMITTENT IN TYPE

This group includes the comparatively mild types of aestivo-autumnal malaria met with in Italy near the Maremma, and it also includes very malignant forms of malaria, such as those which prove so fatal on the West Coast of Africa and elsewhere. In some of the milder cases the presence of the parasite is not definitely made out, and the patient suffers rather from a feverish attack, with headache, and enlargement of the spleen, and in process of time considerable anaemia develops; but such attacks of fever may undermine the patient's health just as seriously as the more obvious benign or intermittent varieties already mentioned. Other cases belong to the pernicious or malignant forms of malaria, but the whole group is described as malignant, and fully deserves that title.

The Malignant or so-called Aestivo-Autumnal Parasite. Different descriptions have been given of the organisms concerned, although they may in reality be one organism whose virulence is modified by certain conditions. They are, (1) the pigmented quotidian, (2) the unpigmented quotidian, (3) the malignant tertian; in all these organisms there is a much more indefinite period at which sporulation occurs, and in the peripheral blood there may be little evidence of the parasite at all. The parasites are chiefly confined to the internal organs, especially the spleen and the marrow of the bones, in which, however, they are invariably found. There is nearly always the typical ring-like parasite with little pigment, and generally marked changes in the red blood corpuscles which form their hosts, in the direction of wrinkling of the corpuscles and rapid disappearance of haemoglobin. In their life-history, however, the intra-corporeal parasites, although they differ in certain details from those belonging to the intermittent group, still show a family resemblance to them, and outside the body the mosquito plays the same part as with the intermittent organism.

Crescent-shaped bodies are distinctive of the malignant or aestivo-autumnal parasite. The pigment may be either diffused throughout the crescent-shaped parasite, or may appear as a central accumulation. This is the sexually mature form of the parasite, which will be referred to later.

Staining Methods.—A blood film, prepared in the usual way, should be taken 8 to 12 hours before sporulation is due. Fix the film with ether and absolute alcohol in equal parts, or with absolute alcohol alone, then stain with Romanowski's or Leishman's modification of Romanowski's stain. Leishman's stain contains methyl-alcohol, which is a fixing agent, and the fixation and staining can be performed in one operation. The parasite is coloured blue, and should be examined with an oil-immersion lens, by preference $\frac{1}{12}$th or $\frac{1}{14}$th inch objective.

Extra-Corporeal Phase of the Malarial Parasite, or the Cycle of Development in the Mosquito

Ross, Manson, and many others discovered that it is by the mosquito, and the species called anopheles, that the malarial parasites are taken up from the blood of the human subject, and go through a stage of development entirely distinct from what is noted in the human subject.

The parasites of benign tertian and quartan ague, and the malignant types of parasites, occur in sexually mature forms in the human subject. These forms do not sporulate. The tertian and quartan mature, non-sporulating parasites are of two kinds—the hyaline or male organism called the micro-gametocyte, and the granular or female organism called the macro-gametocyte. In malignant types, due to the so-called aestivo-autumnal parasite, the crescentic bodies are the sexually mature forms.

No further change occurs in the human subject, but the process of impregnation of the female parasites by the male can be studied on a wet slide, and the further cycle of development in the stomach wall of the mosquito.

The male parasite, or micro-gametocyte, develops flagella, called micro-gametes, which move vigorously, become free from the parent organism, and seek out the macro-gametocyte or female organism. One enters at a papilla which the female organism projects, and after moving vigorously inside the cell disappears from further observation. The fertilised macro-gametocyte is called a zygote; this undergoes a change in shape, one end becoming pointed like a spear, which enables the zygote, now called an ookinet, to pierce the stomach wall of the mosquito. It reaches the muscular coat, where it

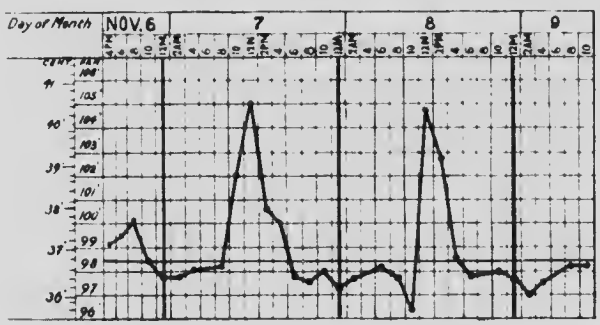
becomes encysted about the second or third day, and is called an oocyst. This grows rapidly, and may attain 70 μ to 80 μ in diameter. Inside the oocyst the small slender sporozoites develop in large numbers, and eventually the parent cell bursts: the sporozoites escape, and a certain proportion of them reach the veneno-salivary gland, ready to infect the next human being which the mosquito may bite.

The crescentic bodies of the malignant types undergo a similar cycle in the mosquito.

It is easy to distinguish the anopheles mosquito from the culex, because the body of the anophele is placed at right angles to the surface to which the animal is attached, whereas the body of the culex is parallel with the surface. The wing of the anopheles is spotted, and the palpi are also distinctive. The anopheles require a special kind of algae in the water in which they breed, and consequently they are not so widespread as are the culex mosquitoes. In addition anopheles require not merely sheets of water, but also a steady warm temperature.

Type I. Benign Ague, usually Intermittent in Type

Clinical Features.—There are three stages which are associated with the sporulation of a group of parasites.



Temperature Chart. Benign Ague; Intermittent Fever; Double Tertian or Quotidian. (After Osler.)

1. The *Cold Stage*, during which the patient shivers, looks blue and cold, and suffers from violent rigors; associated with these symptoms are headache, extreme languor, and often yawning and nausea. Notwithstanding the fact that the skin

temperature is reduced, the temperature in the rectum and the mouth will be found to be 103°, 104°, or 105° F. This stage lasts for some minutes up to about an hour.

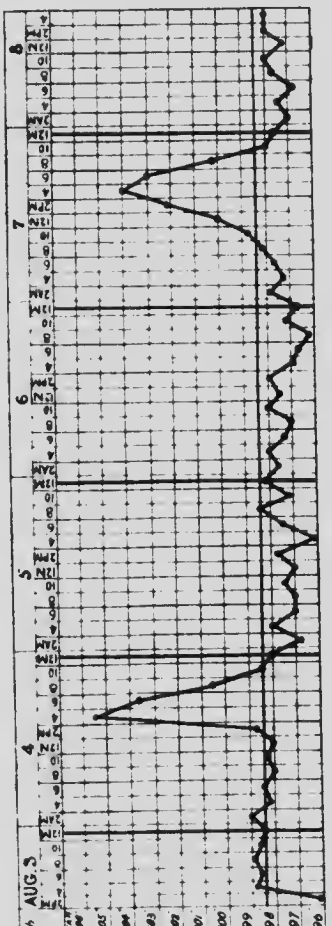


FIG. 20.—Temperature Chart. Benign Ague: Intermittent Fever: Simple Quartan type. (After Osler.)

2. The *Hot Stage* follows the cold. The patient soon feels warm and comfortable, and throws off the blankets with which during the preceding stage he has covered himself, but in a short time he begins to burn, his head throbs, and his pulse is full and bounding; he complains of thirst and severe headache, pains in the back and legs; not infrequently an element of delirium is superadded, perhaps especially in children. This stage lasts generally for a period of 2 to 4 hours, and is followed by—

3. The *Sweating Stage*, during which the temperature falls with profuse sweating, and the discomfort rapidly subsides. The duration of this stage varies; it may last for 1 or 2 hours.

These attacks occur at the same time, almost exactly forty-eight hours after the previous attack, unless there happens to be more than one group of organisms present in the same patient. The spleen invariably enlarges with an attack of malaria, and as attacks go on the enlargement may tend to increase. The liver also sometimes participates in this enlargement.

In certain cases the fever is of a remittent type.

Type II. Malignant Ague, sometimes called Remittent or Aestivo-Autumnal Fever

Clinical Features.—As we should expect from the description of the parasite, malignant ague does not follow the typical periodic characteristics of the benign form. The temperature may rise to a considerable height, but the three stages are not well marked. The hot stage lasts for twelve or more hours, and, as a rule, during the *Sweating Stage* the temperature does not fall to normal. In certain instances the fever is so continuous as to be termed a "continued" type of malaria. In other cases vomiting is peculiarly severe, and the term "bilious remittent" has been given to such cases.

Most of the irregular forms of malaria are instances of remittent fever, but it should be remembered that a pernicious type of malaria may occur in intermittent fever as well as in remittent, depending partly on the severity of the attacks, and partly, probably, on the ineffective resistance of the patient.

There are certain types of malaria more common in the remittent than in the intermittent forms, about which a few remarks must be made.

The Bilious Remittent Type.—In this form of malaria there is great alimentary disturbance, with specially marked epigastric pain, enlargement of spleen and liver, and much bilious vomiting. Sometimes diarrhœa occurs, although more often constipation. It may be a stage in remittent or intermittent fever, and the alimentary phenomena tend to become less marked after a time.

The Typhoid Type.—Some cases closely resemble typhoid fever from an early stage, and the clinical features designated "typhoid" are the result of excessive weakness, with usually a tendency to a low type of delirium or coma, a feeble pulse, a dry tongue, and frequent picking at the bedclothes—a group of symptoms often described as "typhoid" in connection with any febrile process. The typhoid type of malaria is, however, extremely dangerous; any malarial fever may pass into a condition of things which can be described only as "typhoid," and bed-sores, a low type of pneumonia, or sheer debility very commonly lead to a fatal termination of the case.

Pernicious Malaria.—This also is more common in remittent fevers. It is generally held to be due to the blood-

vessels being plugged by thrombi consisting mostly of pigment, perhaps in part of parasites. The results can easily be appreciated; there may be (1) head symptoms, sometimes with paralytic seizures, often with more acute delirium and a tendency to hyperpyrexia, while in certain cases convulsive seizures occur, and death only too frequently follows in twelve to twenty-four hours.

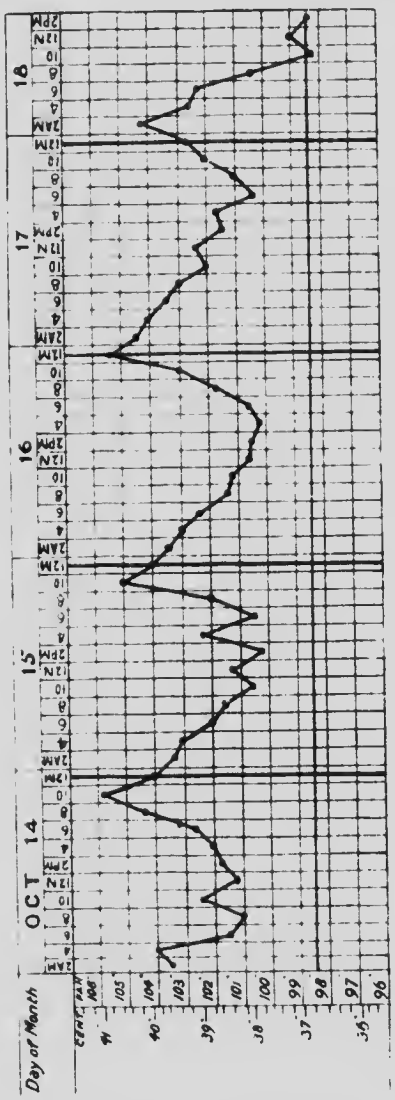


Fig. 21.—Temperature Chart. Malignant Ague; Remittent Fever; Quotidian attack.

(2) In other cases it seems as if the thrombi involved the alimentary tract. Profuse diarrhœa, with intense weakness and cholera-like cramps, soon place the patient in a very serious condition. In some of these cases the diarrhœa suggests dysentery, others more closely resemble cholera, and in still others the condition previously described by the term "typhoid" speedily develops.

(3) A third variety of pernicious malaria is well described by the term "haemorrhagic." In it blood may flow from any mucous surface, and not infrequently the urine contains, not red blood corpuscles, but haemoglobin. It is a paroxysmal haemoglobinuria, and when it is associated with vomiting of enormous quantities of blood and with bleeding from the bowel, nose, and elsewhere, the term "blackwater fever" has

been sometimes applied to it. This is the fever which we associate with the malaria of West Africa, and especially the Gold Coast, although it has been met with in many other malarial countries, and it should be remembered that any malarial fever may assume a haemorrhagic type under conditions which are as yet inadequately explained. It has been stated that quinine is responsible for some of these cases, but fortunately there is no proof that this is correct, and though quinine is not, as a rule, looked upon as such an efficacious remedy in these cases, its administration should not be neglected.

Malarial Cachexia.—When an individual has experienced many attacks of malarial fever, he becomes cachectic. By this is meant a profound anaemia, due to the destruction of enormous numbers of red blood corpuscles, and, in addition, there is a tendency to the accumulation of pigment in different parts of the body. Certain of these cases eventually become pernicious or malignant in type, but in others the patient simply suffers from the prolonged loss of red blood corpuscles, and his symptoms include those of severe anaemia with breathlessness, oedema of the legs, and huge enlargement of the spleen, which remains permanent, and to which the term "ague-cake" has been given. Added to these phenomena are not infrequently haemorrhages, a constant tinge of jaundice, and a tendency to scorbutus. The haemorrhage may be retinal, or from the nose, gums, mouth, and elsewhere, and the patient's skin acquires a typical brownish or greyish colour.

A number of cases of so-called malarial cachexia have recently been found to be due to the presence of Leishman-Donovan bodies in the splenic blood. These organisms are the cause of kala-azar (see p. 144).

The clinical features in malaria often suggest the special involvement of some particular system,—occasionally, for instance, severe sickness and vomiting are prominent, while in other cases headache and neuralgia of a very severe type constantly harass the sufferer.

It should be remembered in connection with all malarial fevers that the disease may simulate many other conditions—vomiting, dysentery, pleurisy, pneumonia, severe palpitation, syncopal attacks, apoplectiform seizures, and paroxysmal neuralgias may all develop along with, or as part of, a malarial

seizure, and in the malarial subject the possibility of part, at least, of any ailment being of malarial origin should never be overlooked.

Malarial attacks may last for a long time. They may, apparently, be cured in two or three weeks, but relapse is extremely common, and it is prudent, when possible, to induce the patients to change their place of residence to a country in which malaria is unknown, because repeated attacks of malaria induce malarial cachexia.

Diagnosis.—Even in the remittent forms of malaria there is a great tendency to periodicity, and in all forms of malaria the spleen is enlarged. During an attack, or rather just before sporulation, the blood should be examined for the parasite, and the remarkably beneficial effect of quinine in preventing attacks is an important point in the diagnosis.

Prognosis.—From the previous pages it will be gathered that malaria may be mild, or may be extremely fatal, and that remittent fever is more serious as a general rule than intermittent. Perhaps still more depends on the possibility of the individual attacked residing for the future in a non-malarial country, but there are many persons who stave off malarial seizures by carefully regulated doses of quinine. The malarial fevers acquired in West Africa are very commonly of a serious type.

Treatment.—PROPHYLAXIS.—Long before the relationship of the mosquito to the malaria parasite was understood, it was known that marshy ground predisposed to malaria. Certainly the draining of swamps where the anopheles can breed is of the utmost moment, and where infected mosquitoes exist, the mosquito curtain should invariably be used during the night. Much has been done by pouring kerosene oil over the surface of pools which cannot be easily drained, in order to destroy the larvae of the mosquito. It is wise, when an individual has to live in a malarial country, not merely to attend to the mosquito net, and to free the district immediately surrounding his home from mosquitoes by the use of kerosene oil and drainage, but also to avoid exposure in the evening when mosquitoes are apt to bite. Furthermore, after exposure in a specially malarial district, he should take a dose of quinine, sometimes 5, sometimes 10 grains, the amount depending on the personal equation of the patient.

It is best to give quinine, and to give it promptly, in treating ordinary cases of malaria. Ten grains must be taken at once, and then smaller doses of 4 or 5 grains thrice daily should be kept up for several days. It is well to endeavour to abort the periodic attacks of fever, in other words, to anticipate sporulation by giving quinine; and it should be remembered that in order to have the full effect of the drug, it must be administered on an empty stomach, and preferably with a little dilute sulphuric or hydrochloric acid, so that the quinine may be dissolved and thus be given every chance of acting. Koch recommends 5 grains of calomel, followed by a saline six to eight hours later, and then 15 grains of sulphate of quinine. The same dose of quinine is given five mornings running, and then 15 grains given on the 10th, 11th, and 12th days, and repeated every 10th day thereafter for three months. Much trouble, however, is experienced by the great tendency in many malarial patients to sickness and vomiting, and in these cases quinine may be given as an enema or even hypodermically,—the latter method, however, being undesirable, because an abscess not infrequently results unless great care is taken. Hypodermically the hydrochlorate of quinine ($7\frac{1}{2}$ grains thrice daily) is recommended by Manson. The injection should be made deeply; but as far as possible avoid the use of this method of administration altogether, because in several cases tetanus has supervened notwithstanding every possible care being taken.

Calomel and ipecacuanha are often of advantage in allaying the vomiting of malaria, while ice and effervescing mineral waters should not be forgotten. During the cold stage of an ordinary attack of intermittent fever give the patient hot drinks, apply hot bottles to his feet, and wrap him well up in blankets. During the hot stage sponge the skin and apply an ice-bag to the head, and when the sweating has commenced guard against chills, and during this stage give a dose of quinine.

It is not desirable, where there is a very high temperature, to give antipyrine, or one of that group of remedies, but rather to resort to cold affusions, the application of ice to the body, and similar methods. For haemorrhagic cases quinine should not be neglected, but calomel has proved of great value given in full doses, and tannin and other astringents are also

recommended. Never forget that malaria means anaemia, and that iron and sometimes arsenic are beneficial, while careful dieting and change of air and climate will do much to restore health.

The enlargement of the spleen is extremely difficult to treat satisfactorily: where it is painful, hot applications and certainly the use of quinine internally do much to reduce the size of the organ, but where ague-cake is present, no treatment seems to be efficacious.

There are many other remedies recommended in various countries for malaria, and methylene blue has been stated to be of value by one or two writers, while the anti-malarial remedies given in India and elsewhere consist largely of diaphoretics in addition to quinine. Baths are not infrequently of considerable advantage. Finally, it should be again stated that no remedy has such a remarkable effect on the malarial organism as quinine; it seems to have a peculiarly toxic action on the parasites, and while it can hardly be stated that, if sufficient quinine is administered, the patient will for ever after be free from malaria, the most remarkable benefit is derived from its careful and painstaking administration.

XL. KALA-AZAR

(Dum-dum or Black Fever)

A disease due to a definite protozoon — the Leishman-Donovan body—and characterised by marked enlargement of the spleen and an irregular and remittent type of fever with progressive emaciation and cachexia, terminating generally in some months' time in death.

Etiology.—The disease extends over a wide area of India, and it has also been found in China, Egypt, various parts of Northern Africa, and to a less extent elsewhere. The parasite was discovered in the splenic blood by Leishman in 1903. The disease is more common during the cooler months of the year, and its victims are usually under the age of 20.

Pathological Anatomy. The Leishman bodies are found mainly in the spleen, liver, and bone marrow. When

stained by the Romanowsky stain, or some modification of it, the parasite is seen as an oat-shaped body from 2 to 4 μ in diameter, with faintly stained protoplasm and two chromatin masses placed at opposite sides of the lesser diameter of the parasite. The larger mass or macro-nucleus is somewhat round in shape, while the smaller micro-nucleus is usually rod-shaped and is more deeply stained. The parasites become flagellated as one of the stages in their development. They are often found free in the splenic blood, and also in large macrophage cells, in each of which a number of the parasites may be present.

The parasites have as their intermediate host one of the forms of bed-bug, and it is almost certainly through the bites of these insects that this terrible disease is spread.

The spleen becomes greatly enlarged, is pigmented, is very friable, and owing to its great size is readily injured. The liver is enlarged, also pigmented and friable, and may contain many of the parasites. In the bone marrow the parasites are found in macrophage cells and occasionally in leucocytes and myelocytes.

The **Clinical Features** include high fever, often commencing with a rigor and general malaise. The pyrexia is of remittent type, and during the fever process the spleen rapidly enlarges. An apyretic period of varying length may follow the original febrile attack, and then repeated returns of fever follow until the patient becomes exhausted. There is progressive anaemia, often vomiting and diarrhoea, and not infrequently haemorrhage from gums, bowels, and elsewhere. The skin becomes deeply pigmented, giving to the fever the name of the "black fever." Night sweats are not uncommon, and the joint pains may be suggestive of rheumatism. The spleen soon reaches the level of the umbilicus, and the liver is also found to be enlarged. Both organs are often tender to pressure. The red blood corpuscles are reduced to about 2,000,000 per c.mm., and the haemoglobin to 35 or 40 per cent, and there is a marked leucopenia sometimes reaching 1000 per c.mm., with a relative increase of the lymphocytes.

Diagnosis.—The best method of diagnosis is to puncture the spleen or liver with a very fine needle and to stain the fluid obtained, but owing to the risk of haemorrhage it is

well to prepare the patient by administering calcium chloride or lactate for some days before making the puncture.

The **Prognosis** is very grave, over 90 per cent of cases proving fatal.

The **Treatment** is as yet most unsatisfactory. Quinine, according to some authorities, gives the best results, and its administration in enormous doses, such as 60 or even 90 grains, may be ordered.

It is obvious that the destruction of the bed-bug is most important, and the utmost care should be taken to prevent the subjects of the disease being bitten by these parasites and so leading to the infection of other persons.

XLI. SLEEPING SICKNESS

This is the result of the presence of parasites in the blood called trypanosomes. There are different varieties of this blood parasite, some of which may produce symptoms in animals, and when man is affected a form of lethargy called sleeping sickness may develop. The tsetse fly conveys the parasite from one animal to another, and the death-rate in horses and cattle, due, it was supposed, to the tsetse fly, is in reality the result of the trypanosome. In certain districts of Africa a large percentage of the population have trypanosomes in their blood, and therefore there may be no symptoms produced by the parasite; but in other cases sleeping sickness develops, and when it is remembered that the period of incubation, or at all events the period during which the trypanosomes produce no symptoms, may extend for a very long time, it is easy to understand the difficulties which surround the study of this disease.

The Glossina palpalis, the fly whose bite infects the human subject with trypanosomes, is the extra-corporeal host and the trypanosomes take twenty days before the fly becomes infective.

The cervical glands enlarge early in most cases, after infection, and the trypanosomes may be obtained from them, although the cerebro-spinal fluid affords an easier means of making a positive diagnosis. Bagshawe states that auto-agglutination of the red blood corpuscles is one of the most important signs

Sleeping sickness begins with the gradual development of lethargy, associated with difficulty in walking and speaking, considerable tremor in the limbs, and increasing pyrexia. The lethargy increases and the patient may die in a few months. The trypanosomes are found more easily in the cerebro-spinal fluid than in the blood.

The **Prognosis** is grave and the **Treatment** at present is still unsatisfactory. Atoxyl or sodium arsanilate has a wonderful effect on trypanosomes in animals, but its efficacy in sleeping sickness is still *sub judice*. Bruce in September 1910 stated that there is no absolute proof that a single person has recovered from the disease. Atoxyl has caused optic atrophy, and its administration requires careful supervision.

DISEASES OFTEN GROUPED AS CONSTITUTIONAL DISEASES

I. MUSCULAR RHEUMATISM

Myalgia

A PAINFUL affection of certain voluntary muscles and tendons.

Etiology.—It is generally the result of rheumatism, and is associated with cold and wet, although in gouty persons there is also a predisposition to this affection. In some cases of acute rheumatism the muscles may suffer as well as the joints.

Clinical Features.—Pain in the back muscles is termed *lumbago*, in the intercostal muscles *pleurodynia*, and in the neck muscles *torticollis*, or stiff neck. *Lumbago* is very common after exposure to cold or wet when one is overheated. It fixes the patient's body as if in a vice, and causes the utmost agony upon any movement of the affected muscles. *Pleurodynia* is a similar involvement of the intercostal, pectoral, and serratus magnus muscles, a deep breath causing pain, or any muscular movement, such as in coughing. *Torticollis*, or stiff neck, is an involvement of the muscles at the side of the neck, and specially the sterno-mastoid. Pains in other muscles have received different names, but the term *myalgia* can be used in all cases. The affected muscles are tender to the touch, and are often definitely swollen; they may be particularly painful when the patient becomes warm in bed, and a certain amount of pyrexia is not uncommon.

Diagnosis.—The pain on muscular movement distinguishes this condition from *neuralgia*, while digital pressure is not painful in *pleurisy*, with which pleurodynia may also readily be confused.

Prognosis.—In most cases prompt treatment is followed by equally rapid benefit, although there is a tendency to recurrence of myalgia.

Treatment.—Give one of the salicyl group of remedies along with an alkali, and remember that the prevention of movement of the affected muscles is the best means of securing comfort for the sufferer. Counter-irritation is sometimes beneficial, such liniments as liniment of turpentine being very efficacious, and blisters may also be used. Soothing applications, for instance the A. B. C. liniment, frequently give relief to the patient, and in certain cases acupuncture is followed by rapid improvement, the needles being left in position for five or ten minutes. A hot bath, hot fomentations, gentle massage, and, when necessary, the use of opium or morphia, are all useful methods of treatment.

II. CHRONIC RHEUMATISM

Etiology. There are obviously two distinct types of cases included under this title, the first being merely cases similar to the disease described as acute rheumatism, only with less marked pain, less acute joint involvement, and less general disturbance. But there are also many cases included under the term chronic rheumatism which are not the result of any specific organism, and with our present uncertain knowledge of the pathology of rheumatic fever it is obviously difficult to be dogmatic. In this latter group of cases, muscles, tendons, fibrous tissue, and not uncommonly the fibrous tissue of nerves, are the sites of the disease, and less commonly the joints.

There are three ways in which chronic rheumatism may be induced—(1) exposure to cold and wet; (2) by strain, such as tennis-elbow, golf-elbow, etc.; and (3) by the presence of toxins, such as cause the muscular pains in influenza and other fevers, and toxins due to over-indulgence in certain foods and liquors.

Chronic rheumatism is more common in persons who have reached or are past middle life, and who are exposed to cold and wet. It is closely associated with a rheumatic family history, and varies greatly in severity according to the weather

Poverty, hard manual labour, and insufficient food are also potent factors.

Pathological Anatomy.—In the articular form there is often great thickening of the capsule and ligaments of the joint, with a limited amount of effusion, and the joints which specially suffer are generally the larger ones, and less frequently the small joints of the hands and feet. The muscles in the neighbourhood of a severely affected joint sometimes waste, and very often the patient suffers from permanent ill-health. Cases in which special nerves are involved—as, for example, the sciatic—will be referred to under diseases of the nervous system.

Clinical Features.—The pain is worst at night, when the patient gets warm in bed; it is more severe in cold, damp weather, is often accompanied by creaking in the joint or joints, and becomes easier after exercise or rubbing. The swollen joints are generally tender on pressure.

More or less malaise may be associated with exacerbations, and considerable anæmia and debility result, and not infrequently deformity of the joint. There is rarely much temperature, but there is a considerable tendency for the heart to be involved, specially in articular cases.

Diagnosis.—It is difficult to distinguish this condition from *rheumatoid arthritis*, under which disease reference will be made to the differential diagnosis, but in *gonorrhœal rheumatism* the history of a urethral discharge, associated with the long-standing and persistent involvement of often a single large joint, generally renders the diagnosis easy.

Prognosis.—Chronic rheumatism is apt to become a fairly constant companion when once it has persistently attacked one or more joints. It recurs with any wet or cold weather but does not, as a rule, shorten the life of the sufferer.

Treatment.—It is wise in every case to try the salicyl group of remedies, and especially when renewed pain and swelling bring the patient to seek advice. Potassium iodide in 10 grain doses, and guaiacum resin in 5 to 10 grain doses, in capsules, are often, however, more efficacious than the salicyl group. Counter-irritation, especially with iodine, fly-blisters or the button cautery, is of the greatest value, while baths of all kinds, and in particular radiant-heat, electric, and alkaline baths, are beneficial; in fact, any hot-water baths associated

with massage and passive movement of the affected joints will yield satisfactory results. Where possible, recommend a warm, dry climate if the patient can afford to escape the severities of the home winter. Dress warmly, and study the dietary, remembering that in starved patients good feeding is of value, while in over-fed sufferers from this disease restriction in diet may be necessary.

III. RHEUMATOID ARTHRITIS

(Arthritis Deformans. Osteoid Arthritis)

A chronic affection of various joints, characterised by definite changes in the cartilages and synovial membranes, and generally followed by new bone formation, and always by marked and permanent deformity. The evidence that this condition is associated with a specific organism is not conclusive, and a chronic toxaemia, due to an error of metabolism or to absorption from intestinal mucous surfaces, has much to commend it.

Etiology.—Most patients suffer between the ages of 30 and 50, and as a rule the more acute cases occur in the younger persons. It is a disease which is certainly more frequent in women, and appears in them to be the result of any excessive strain such as repeated pregnancies or protracted lactation. Where one joint alone is involved, and where the condition is very chronic, a slightly larger proportion of men suffer than women.

The hereditary history is generally one of joint affection, sometimes of gout, and sometimes phthisis.

Local injury seems to predispose to the mono-articular form but cold, worry, fatigue of mind and body, and insufficient food are amongst the most common etiological factors in the polyarticular variety.

A small bacillus has been described by Bannatyne, Bloxall, and others, as occurring in the synovial fluid of affected joints, and experimental inoculation with a culture of the bacillus has yielded results in animals closely corresponding to those in man, although it is difficult to believe that in all cases a microbic origin can be proved. It should be remembered that

a definite gonorrhoeal history has been obtained in a considerable number of patients affected with this disease, and it is possible that in certain cases the gonococcus may really be the specific organism.

A number of authorities refer to what may be called the neural theory in attempting to explain the causation of the disease, and there is at first sight a similarity between the Charcot's joint affection of locomotor ataxia and rheumatoid arthritis, but the statement that the wasting of muscles in the neighbourhood of the affected joints is the result of a neuritis, which is a definite etiological factor in the disease, is improbable. It is true that lesions of the nervous system, such as occur in infantile paralysis, cause atrophic changes in muscles, bones, and skin, but only to a limited degree are joints affected.

Pathological Anatomy.—The first change in the **Polyarticular form** is probably in the cartilages of the affected joint, the cells of which proliferate, and fibrillation occurs at right angles to the articular surface of the joint. Soon afterwards the softened cartilage is rubbed away, and when the bony surfaces come in contact they become eburnated, and a considerable amount of creaking is produced in the joint on movement. Associated with this change is an increased vascularity and a coexistent development of fresh cartilage at the periphery of the joint. The synovial membrane is generally much thickened, and in some cases this thickening, together with periarticular effusions, may predominate over any bone lesion. The synovial fluid is increased in amount, and at an early period there is deposition of bone in the newly formed cartilage, causing "lipping" and greatly diminishing free movement. In the periosteum also fresh bone may form. This development of new bone in connection with cartilage and periosteum is called *osteophytic*. It can be easily understood how repeated attacks of inflammation, with increase of fluid and change in the cartilages, will in time cause marked disorganisation of the joint, while ankylosis is by no means uncommon. In a smaller number of these polyarticular cases the changes in bone and cartilage are mainly atrophic.

In the **Mono-articular form** of the disease there is apt to be absorption of bone, the existing cancelli being in part removed by osteoclasts.

In the form of the disease described as **Heberden's nodes,** small osteophytic outgrowths develop at the bases of the middle and terminal phalanges, usually lateral in position. When present they very frequently appear to limit the advance of the disease towards other joints.

In many cases there is muscular wasting in the neighbourhood of affected joints, and sometimes spasm of atrophied muscles may add to the deformities brought about in the joint cavities: hyperextension of the distal interphalangeal joints is not uncommon.

Clinical Features.—Type 1.—The Polyarticular Form may be acute, subacute, or chronic, and in the more acute cases it is specially common in young persons, in whom it may be associated with glandular enlargement. It commences in one joint, often one of the small joints of the hand, and rapidly spreads: it is generally symmetrical, and the joints are painful, and have a typical bulbous-like appearance with a considerable amount of local hyperaemia. Repeated attacks may occur, one attack subsiding, but usually leaving definite joint changes, and with a number of attacks much deformity inevitably results. Pain in the ball of the thumb has been described as an early symptom of the disease, and fresh attacks commonly result from any exposure to cold or any physical exhaustion.

There are four typical features which are often associated with the forms included in this type:—

1. Pigmentation is common over or near the affected joints, sometimes in other positions, and may even be an early and suggestive feature of the disease.

2. Sweating, especially of the palms of the hands and soles of the feet, is characteristic, and is more severe during the attacks.

3. A definite, fine tremor may be seen in the hands and feet; and

4. There is often an exaggeration of the tendon reflexes.

There is sometimes tachycardia, and neuralgic pain is common in the ball of the thumb and the ulnar side of the wrist.

In a certain proportion of patients the temporo-maxillary joints are affected, and occasionally the intervertebral joints, especially in the region of the neck.

Many patients suffer for years from the disease, but fortunately, in not a few cases a time arrives when the condition is arrested and there is immunity from renewed attacks. The usual twisting of joints is in the direction of flexion, with the exception of the terminal phalanges of the fingers, which are hyperextended, and the fingers and wrists are bent towards the ulnar side.

Recently Still has described this type of rheumatoid arthritis in children, and associated it with remarkable enlargement of lymph glands and spleen.

Type 2. –Mono-articular Form. This form is common in old persons, and affects the knee, hip, shoulder, and more rarely one of the intervertebral joints. Shortening of bone may be noted, and it tends to follow local injury. The general symptoms, such as pigmentation, local sweating, tremor, etc. are not present.

Type 3. In this type **Heberden's nodes** develop. As already noted, they form at the bases of the middle or terminal phalanges of the fingers, and are cartilaginous or osteophytic outgrowths from the joint cartilage. They are associated with a limited amount of swelling, pain and the usual changes in the cartilages described under Type 1. The disease is usually limited to the smaller joints just mentioned, and generally becomes arrested at an early period. Heberden's nodes are said to be more common in women.

Diagnosis. – *Gout, chronic rheumatism, and gonorrhœal rheumatism* may all be confused with this disease. In acute gout the usual joint first, and often alone, affected is the metatarso-phalangeal joint of the great toe, and the definite history of the attacks, with generally a hereditary gouty tendency should render the diagnosis easy. In chronic cases of either disease there is much more difficulty, although a careful study of the diet, of the urine, and of the history of the joint attacks is often very distinctive in a case of chronic gout.

In rheumatism relief is generally obtained with salicylate of soda, whereas in rheumatoid arthritis that drug is generally valueless.

Gonorrhœal rheumatism certainly resembles Types 1 and 2 of this disease, but the history of the case, if obtained, should prevent any mistake, and pigmentation, sweating, and tremor are not present except in cases of rheumatoid arthritis Type 1

It should be mentioned that the temporo-maxillary joint may be affected in gonorrhoeal rheumatism and in rheumatoid arthritis, while it is extremely uncommon to find it involved in gout or chronic rheumatism.

Prognosis.—At the best, a progressive case necessitates long and often energetic treatment before it is even safe to say that the disease is arrested. In the 2nd and 3rd Types, and especially the 3rd, the condition is limited, and the prognosis is more favourable.

Treatment. The etiological factors must be carefully considered. An insufficiently fed patient should be ordered a nourishing and easily digested dietary, and in no disease is the prudent use of port wine and malt liquors more satisfactory. Damp must be carefully guarded against, and if possible the patients should seek a dry and warm climate.

Internal treatment by drugs is hardly so successful. Cod-liver oil, malt extract, and similar agents are good, while iron and arsenic are often advantageous. Potassium iodide and carbonate of guaiacol have been strongly recommended.

The *local* treatment is more important; persistent painting of the affected joints with iodine is of very great value, while much benefit may result from the use of hot air baths, electric baths hot-water baths of all kinds, and the high-frequency currents. Douching and wet packs are amongst the numerous special hydropathic methods of treatment which suit different cases.

All forms of treatment should be combined with massage, and the massage should include movements which tend to restore the usefulness of the affected joints. In some cases excision of an ankylosed joint has been performed in order to render the limb more serviceable.

IV. GOUT

Podagra

A disease, probably due to faulty chemistry, the exact nature of which is as yet not clearly defined. It is associated with an excess of uric acid, possibly formed in the body as a result of the affection, and with the deposition of urate of soda

in certain joints, accompanied by typical attacks of arthritic pain. Excess of uric acid does not necessarily imply excessive production: it may be due to defective excretion. The disease may be (1) **Acute** or (2) **Chronic**, and there are special forms of gout to which the terms (3) **Suppressed** and (4) **Irregular** are given.

Etiology. There is a strong hereditary predisposition, and in gouty families the dangerous period is reached when middle life is passed. Probably in fully one-half of the cases there is a definite hereditary history of gout, although sometimes a generation is missed over and the legacy handed on to the grandchildren. The male line is credited with the stronger hereditary taint, and males suffer more markedly than females. It is most common after the age of 50, but it may occur at any time of life in those hereditarily predisposed, and 35 to 40 is often the age at which a first attack occurs.

An excessive amount of alcohol is certainly responsible for many cases of the disease, and especially the sweet, heavy ales affected by English palates as distinct from Scottish.

Overeating is another etiological factor in not a few cases and is apt to lead to gout if it is associated with a sedentary life and too little exercise. Lead poisoning is responsible for the production of some of the more chronic cases of gout, and gout is peculiarly common in persons whose work necessitates the use of lead.

There is often some definite exciting cause for an attack such as worry, overwork, illness of almost any kind, and especially confinement to bed for a surgical injury or operation.

Notwithstanding these fairly definite etiological factors, the pathology of gout is distinctly mysterious. Garrod has suggested that there is a lessened alkalinity of the blood, and that, as the result of diminished elimination, an excess of uric acid occurs. He asserts that urate of soda is not held in solution, and is suddenly deposited during the gouty paroxysm in the joints. Haig considers that the blood is less alkaline, and therefore less able to keep the uric acid in solution. There is no excessive manufacture of uric acid. Roberts considers that uric acid circulates as a quadriurate, which is freely soluble and easily eliminated by the kidney. If it lingers in the blood and should happen to come across sodium carbonate, it takes up an additional atom of base, becoming as a result a

biurate, which is much less soluble, and readily crystallises out in parts where the circulation is less rapid, and especially in the presence of sodium chloride, which is found in the lymph in the synovial sacs. Levison agrees with this statement, but adds that uric acid is obtained from the breaking up of white blood corpuscles, and therefore is proportionate to the leucocytosis present. Ord believes that there is a general or local disintegration which precedes the deposition of the urate of soda in the joint, and Ebstein considers that this joint process is a form of necrosis. Sir Dyce Duckworth holds that there is a peculiarity as regards the innervation of hereditarily gouty individuals, which influences the production and the elimination of uric acid.

None of these theories are altogether convincing, and it is now believed that uric acid circulates in the blood in combination with thyminic acid.

Fenner states that nitrogenous food oxidised in digestion forms proteins, paranucleins, and nucleins, and it is the nucleins which break up into nucleic acid and the purin bases. The purin bases are xanthin, hypoxanthin, etc., and it is due to the oxidation of these that uric acid is produced. Thyminic acid should also be produced in the same processes, and with it the uric acid forms a compound which is soluble. Uric acid may be precipitated, because this union does not occur, or because uric acid is formed synthetically in gout. If this view is correct, where thyminic acid does not combine or where, as in synthetically produced uric acid, it is not present, uric acid may be precipitated into the joints and an attack of gout result.

Pathological Anatomy.—There is a deposit of urate of soda in the cartilages of the affected joints in the form of acicular crystals, and these are also to be seen in the ligaments, in the bursæ, the tendon sheaths, in the skin over the joints, and in the cartilages of the ears, nose, and elsewhere. In the kidneys yellowish-white lines are seen, indicating the deposit of the crystals. There is apparently an excess of uric acid in the blood, although this is not peculiar to gout alone, and Garrod's experiment of the thread in blood-serum is not always positive even in cases of acute gout. It consists in obtaining a drachm of blood-serum from the patient, adding 2 or 3 minims of glacial acetic acid, and placing a thread in the

mixture for some 30 or 40 hours, when uric acid crystals should be found on the thread.

Unquestionably the metatarso-phalangeal joints of the big toes are the most frequently affected, although any joint may suffer. Arterio-sclerosis, with hypertrophy of the left ventricle and sometimes primary cirrhosis of the kidney, form a not infrequent combination in gouty patients, and more especially in cases of chronic gout.

Clinical Features. (1) **Acute Gout.**—There are nearly always premonitory phenomena, the patient feeling out of sorts, restless, dyspeptic, or peculiarly irritable. The acute attack begins with pain in, and tense shiny swelling of, the metatarso-phalangeal joint of the great toe, or more rarely some other joint. The veins in the skin become distended and the joint throbs much like a suppurative arthritis. There is marked dyspepsia, concentrated, highly-coloured uratic urine, and the attack comes on during the night or towards early morning. The pain is excruciating, and the affected joint swells rapidly; the temperature may reach 102° F. or higher, but towards morning the attack wears off. A return of the pain is probable during the next night, and for four or five consecutive nights there may be a considerable amount of suffering. As the attack passes off, the pain is less and less severe, but attacks are apt to recur from any trivial error of diet, and in severe cases at shorter and shorter intervals. Between the attacks both the uric acid and phosphoric acid excreted fall to a very low level, rising, however, as the attack develops. Frequently the patient feels much better after the attack, although repeated attacks of acute gout pull down the strength and render the patient extremely miserable. The skin over the affected joint may desquamate.

(2) **Chronic Gout.** In chronic gout the attacks are longer and more joints are affected. Deposition of urates occurs in the cartilages and ligaments around the joints, so that the joints become swollen and eventually much deformed and osteophytic deposits are very common in connection with them. The feet and hands, sometimes the elbows and knees, suffer, while gastric disturbance is extremely common, and generally arterio-sclerosis is present. It is in chronic gout that the uratic deposits in the skin called *tophi* are best seen—the so-called chalk-stones with which, if they occur in the

knuckles, the patient can write upon a slate. There is not infrequently a trace of albumin in the urine, and from time to time attacks of joint pain occur. There are many complications of gout, to which reference will be made under the heading of Irregular Gout.

3) **Suppressed Gout** implies serious gastric, intestinal, cardiac, or other disturbance, coincident with the disappearance of pain in the affected joints. Vomiting and pain are the most frequent gastric, and diarrhœa the most frequent intestinal symptoms, while dyspnœa with irregular action of the heart may occur as cardiac manifestations. In rare cases delirium, and sometimes even apoplexy, may be simulated in suppressed gout.

4) **Irregular Gout.**—In all gouty families there is a tendency for certain gouty manifestations to appear, and many of these are in reality the complications which may occur in any gouty patient. They include in the *Alimentary System* a tendency to gastric attacks of biliousness, in the *Circulatory System* palpitation, irregular action of the heart, and the development of arterio-sclerosis. In the *Respiratory System* bronchitis is very common; in the *Integumentary System* gouty eczemas and acne rosacea are peculiarly prevalent. In the *Urinary System* uric acid calculi are not infrequent, and associated with them or with the passage of gravel, urethritis may develop. In the *Nervous System* headache and especially neuralgia may annoy the patient. Apoplexy, probably as the result of the arterio-sclerosis, is not uncommon, and eye affections should not be forgotten. There may be inflammation, such as iritis or retinitis; glaucoma may occur, but is probably accidental, and the patient often suffers from attacks of episcleral congestion.

Many of the conditions mentioned under the different forms of gout, and especially in connection with irregular gout, might be more truly termed complications rather than clinical features of gout itself, and we are probably justified in placing inflammations of serous membranes, such as of the pleuræ, the pericardial sac, peritoneum, and meninges, under the head of complications; together with uræmia they are more apt to be associated with the final period of life of our gouty patients.

Diagnosis.—Much stress should be laid on the family

history, and also on the patient's habits as regards food and drink. Where tophi are present the typical acicular crystals of urate of soda may be seen under the microscope, if "chalk-stones" can be obtained and crushed. There is certainly an excess of uric acid in the blood, although the exact significance of its increase in the urine during an attack is by no means clearly demonstrated. The gouty attack is very typical, with the exception of chronic cases, in which the clinical features may be less definite. When it is remembered that irregular gout is almost invariably a hereditary legacy, the most indefinite clinical features may acquire their due significance and proportion when diagnosed as gout.

Prognosis.—The presence and degree of arterio-sclerosis with involvement of the kidneys constitute the most important point in prognosis. Acute gout is readily recovered from but there is a tendency to recurrence. Chronic gout leaves permanent changes in the joints, and irregular gout, notwithstanding frequent manifestations, is generally amenable to treatment. Suppressed gout is assuredly the most dangerous condition, and the sudden onset of severe gastro-intestinal disturbance, serious cardiac disease, or other manifestation replacing the paroxysmal pains of an attack of gout, should make the physician careful of giving a too favourable prognosis.

Treatment.—(1) *Treatment of the Attack.*—The affected limb should be elevated, and, if the patient is confined to bed the leg should be placed on a pillow: the joint or joints should be wrapped up in cotton wool, and fomentations, especially with lead and opium, will be found very soothing. Order a pretty vigorous purge, probably a dose of calomel or blue pill, to be followed by a saline draught in the morning. In most cases, provided the heart is satisfactory, a dose of tincture of colchicum seeds (♏ 15-20) should be at once administered and may be repeated twice or even thrice in the twenty-four hours. the patient may be encouraged to drink freely of lithia water or other alkaline mineral water. It is doubtful whether lithia is really of much value in the treatment of gout, in which disease it was supposed to act as a uric acid solvent. The diet should be of the simplest possible description, by preference milk diet alone, or plain soups, and on no account should sugar in any form be eaten. It is better to recommend the patient to use saccharin or saxin in place of sugar, both of

cookery and also for sweetening tea or coffee. An excess of starch must also be strictly prohibited. The patient may be permitted to have fresh fruit or vegetables, but oranges and fruits that are not too sweet should be preferred.

Where they can be obtained easily, hot-air or radiant-heat baths, applied locally to the affected limb, prove very efficacious, and once the affection has yielded and become less acute, a course of baths are advantageous; these are referred to later.

(2) *General Treatment of Gouty Patients.*— Persons liable to attacks of gout should be extremely careful with regard to diet. Their meals should be in strict moderation, care being taken to eliminate sugar, pastry, sweet fruits, especially preserved fruits, while both starchy articles and butcher meat must have a carefully balanced proportion in the patient's ordinary daily diet. Fish and white meat are good, but beef, pork, and salt meat should be sparingly eaten. Foods rich in purins, such as sweetbreads, and meat extracts containing excess of xanthin bodies, should be avoided. Potatoes, artichokes, and starchy vegetables are better eschewed, while cabbage, spinach, cauliflower, and tomatoes can be eaten freely.

A very important point in treatment is unquestionably the matter of liquor. A small quantity of whisky or spirit without sugar may be ordered when necessary. Certain Moselle wines, such as Bernecastler Doctor, are free from sugar, and may be used by the patient, while cider of the dry variety has been recently recommended, and bitter beer, although assuredly undesirable, is not so hurtful as sweet ales or porter. For wealthy patients dry champagne may be allowed in moderation but port wine, burgundy, and all heavy, sweet wines should be strictly prohibited. Roberts suggests that potassium chloride should be used in place of common salt at mealtimes only the taste is certainly far from agreeable. Elstein recommends the free use of fatty articles of food, such as butter. Most gouty patients benefit from alkaline mineral waters, although it is possible that the benefit is derived rather from the flushing of the system than from any ingredient the waters may contain. It is true that in the spas at home and abroad, where special springs are used in the treatment of gout, important factors are the careful dieting, freedom from home and business worries, and a healthy amount of exercise. Many baths have, however,

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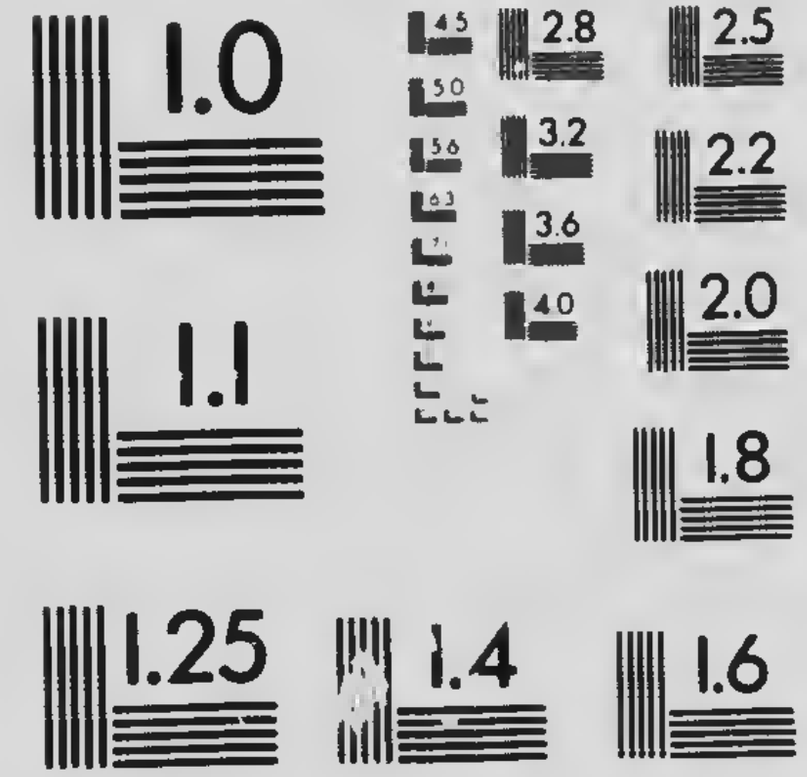

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obtained a thoroughly good and well-merited reputation, such as Bath, Strathpeffer, Buxton, and Harrogate in this country and Carlsbad, Aix-les-Bains, Contrexéville, Homburg, and many others abroad. For not a few patients the mere use of a daily purgative saline draught is good treatment, and it is probably better to substitute magnesium or potash salts for soda. Massage is useful where the joints have become more or less affected, and the careful dieting, together with the prescribed amount of exercise, is very helpful.

The clothing should be warm, but not excessive; probably flannel or wool should be worn next the skin. It is wise when possible, to send gouty patients, who are suffering from the rigour of our climate, to a suitable health resort abroad, as warmth is helpful in not a few cases.

In chronic gout, potassium iodide, guaiacum, and colchicum may be administered, while iron and arsenic are of advantage where the patient is anaemic.

No mention has been made of so-called uric acid solvents. Thyminic acid or solurol in 8-grain doses thrice daily is a remedy well worth a trial, and its success in many cases suggests that the theory propounded above, that it is the natural solvent of uric acid, is correct. Piperazine (gr. 15-30 daily) has a considerable reputation, but its effect is disappointing.

V. RICKETS

Rachitis

A DISEASE of infancy and early childhood, generally commencing before the end of the second year, and characterised by the excessive development of cartilage, especially in connection with the epiphyses, and by delayed and defective ossification of many of the bones of the body.

Etiology.—It is essentially the result of a dietetic error. It occurs where children are bottle-fed, and where the milk used is "condensed" or skimmed milk and poor in fat. There are, however, many children who are breast-fed, or fed upon fresh milk, but who in addition are given unsuitable articles of diet, such as tea, or part of "anything that is going," and these children are specially prone to develop rachitic tendencies if they are also placed in the midst of insanitary surroundings.

In the homes of the rich, a child may develop rickets who is being fed upon predigested food, and it is worth remembering that special foods of the starchy order are liable to produce a combination of scurvy and rickets. The excessive use of carbohydrates in the dietary may be the important factor in certain cases, but it should not be forgotten that the slightest dietetic error takes greater effect on delicate children and those who are not in hygienic homes.

Pathological Anatomy.—There is a great increase of cartilage between the shaft and epiphyses of the long bones, and it is abnormally vascular. Ossification is therefore delayed and is imperfect. The bones which show the earliest changes are the ribs, especially next the sternum, the lower end of the radius, and the bones of the skull. These and other affected bones are found to be unduly soft, being easily cut with a knife, and on section, especially near the epiphyses of the long bones, there is undue vascularity with irregular islands of ossification. The bones yield to pressure, and the typical changes seen in after-life are the result of the consequent twisting and bending. The periosteum is also unduly vascular, and probably this vascularity causes great delay in ossification in its neighbourhood. There is removal of bone already formed, and the medullary canal is found to be abnormally enlarged. The bones of the skull show characteristic changes. There is marked hypertrophic thickening over the outer table of the skull in certain positions, as over the frontal and parietal eminences, while parts of the parietal bones are thin and give the curious crackling sensation on pressure constituting the so-called *cranio-tabes*.

Clinical Features.—The disease, sometimes beginning before, but often at the 6th month, advances insidiously with marked debility, malnutrition, and nocturnal restlessness. The child is slightly feverish, sweats freely at night, and especially over the head, while dandling the child causes great pain. The little patient becomes emaciated, and soon the enlargement of the epiphyseal ends of the long bones is manifest. The delay in ossification causes important changes in the shape of the chest and the development of the body generally, while dentition is also unduly protracted.

The "rosary" ribs are extremely typical, and consist in a bead-like enlargement corresponding to the junction of cartilage

and bone. There is indrawing of the costal cartilages and the sternal ends of the ribs, tending to produce a keel-shaped sternum, while Harrison's sulcus, so commonly seen in phthisical chests with which rickets is not infrequently associated, is due to indrawing of the chest wall above the insertion of the diaphragm. The lower ends of the radius and of the tibia become enlarged, and contrast markedly with the small, poorly formed shafts of the bones. Should the child endeavour to walk, the typical bow-legged condition results, while if the child creeps about on the floor, the tibiae may be bent outwards and the feet slightly everted, because it trails the legs behind it much like the hind fins of a sea-lion. *Coxa vara*, or curvature of the neck of the femur downwards and forwards, giving to the patient a peculiar waddling gait, is an occasional result of this disease. The arm bones may also bend, should the child use the arms to assist in progression and the pelvis undergoes marked alteration, forming the typical rachitic or flat pelvis which causes such trouble in labour. The bones of the skull remain long unossified, and in any case the closure of the fontanelles is delayed. A rachitic head is square-shaped, and the frontal eminences are unduly prominent, giving the patient the appearance, at least, of great mental power. Sometimes cranio-tabes is present, by which is meant yielding of the bones of the skull to digital pressure and this is due to defective ossification.

Rickets is also responsible for knock-knee, flat-foot, and other deformities, but these results of the disease occur generally at a much later period than the phenomena just mentioned. Spinal curvature is often present, although the degree is not so great as in cases of tubercular caries. It depends partly on the softness of the bones and partly on the weakness of ligaments and muscles. In later life when ossification is completed, the malformations become permanent.

While these changes are developing the child suffers in other ways. Bronchitis is extremely common and may be very severe, the soft, easily-moulded chest wall readily yielding to the pressure of coughing. Most rachitic children are also anaemic, although the degree of anaemia in a simple case is not generally very extreme. Amongst the **Complications** of rickets may be mentioned laryngismus stridulus, convulsions

often associated with dentition, and tetany. As a rule the liver and spleen are enlarged in rickets. Teething is invariably delayed, often the first tooth only appearing after a year or fifteen months, and the teeth are apt to be irregular and have an early tendency to become carious.

There is a variety of rickets occurring at a later period than usual to which the term "late rickets" has been applied. It appears in children of 3 or 4 years old, sometimes even at a later period, and is associated with epiphyseal enlargements of bone, muscular weakness, and marked sweating. Generally various deformities result, such as flat-foot, knock-knee, or spinal curvature.

Diagnosis.—It is most important in children to keep before the mind the possibility of *scurvy-rickets* (really a combination of scurvy with rickets), because there is no other disease with which it is possible to confuse this condition. Bronchitis, anaemia, and many other permanent or temporary affections may depend to a certain extent on pre-existing rickets, and the rachitic element in these cases should not be overlooked.

Prognosis.—Death may occur as the result of some intercurrent disease, but in many cases the child survives, with more or less permanent deformity, and the deformity in question may even endanger life, as for example the flat or rachitic pelvis during labour.

Treatment.—The great causal factor in rickets indicates the cure. The child should be at once placed on suitable diet, and that means milk with plenty of cream, and he should at the same time have abundance of fresh air and sunshine, and should be clad warmly, although not excessively. In older children a proper proportion of nitrogenous and carbohydrate ingredients in the diet should be ordered. There are certain drugs which may be given in addition; these include cod-liver oil, syrup of the iodide of iron, the syrups of the phosphates, and arsenic in cases in which anaemia is very severe. Laryngismus stridulus must be treated on the lines suggested in describing that disease. All that need be said here is that antispasmodics, such as chloral and bromides, are of great benefit.

Care should be taken to prevent increasing deformity, and where possible to straighten twisted limbs. Sometimes splints can be intermittently used for this purpose: in other

cases wedges of bone may have to be removed. Lastly, no disease is more benefited by sea air than rickets, and if possible it is wise to have children so affected resident at the seaside, and as much out in the open air as the weather will permit.

VI. DIABETES MELLITUS

THE persistent excretion of sugar in the urine with definite deterioration of health. It is a matter of opinion whether the term should be applied to cases in which the passage of grape sugar is more or less temporary. It seems preferable to call such temporary excretion of sugar, glycosuria.

The carbohydrates of the food are largely stored up as glycogen in the liver, and it is difficult to ascertain what part the liver takes in the production of glycosuria. It seems certain that the internal secretion of the pancreas aids the tissues in some way with the destruction of sugar, thus preventing its excretion by the kidneys.[1] The injection of large quantities of sugar into the blood has not resulted in the production of glycosuria, and the removal of the liver, experimentally, causes the amount of sugar normally present in the blood to disappear. In diabetes mellitus there may be possibly (1) an over-production of sugar by the liver, or (2) a diminished destruction by the tissues, and in both of these supposed conditions the controlling influence of the pancreatic secretion may be the factor at fault. It is conceivable that the sugar brought to the liver in diabetes is not converted into glycogen, or that there may be an excessive conversion of glycogen into sugar (Hamilton). According to Seegen and von Noorden, if an excess of sugar reaches the tissues, some of it will remain in the blood.

Glycosuria of a *temporary* nature may be present as the result of the following conditions:—An excess of sugar ingested will cause a temporary glycosuria to appear, the amount needed depending much on the personal equation of the individual, and also on the existence of any disease from which he may be suffering. Sugar is also present in obesity

[1] Cohnheim has recently shown experimentally that glycogen is used up in the muscles, and that in muscle juice and in the pancreatic secretion (ferment) there are glycolytic agents which can only act together, and when they do so glycogen is converted into heat and energy.

especially in oldish persons, in chronic alcoholism, in exophthalmic goitre, and sometimes in certain of the continued fevers. The administration of phloridzin, opium, chloral, and other agents, especially in certain individuals, and chloroform and ether anaesthesia, may produce a temporary appearance of sugar in the urine. It must, however, be remembered that if the only test used for detecting sugar is copper reduction, there are other agents besides glucose which are capable of reducing the copper salt.

Etiology.—No disease presents such difficulty as regards an accurate etiology and pathology. There is sometimes a hereditary predisposition, and the disease is certainly commoner in males. Mental strain, gout, syphilis, and excesses—mental, moral, physical, or alcoholic—may all cause or predispose to the disease. Injury or disease of the brain or spinal cord, and especially in the region of the 4th ventricle, has resulted in diabetes, and quite a number of cases are definitely associated with atrophy or interference with the secretion of the pancreas. It has resulted from, or at least followed, an acute illness such as pneumonia or a continued fever, while in certain instances it is said to have ensued upon drinking cold water when overheated. Unfortunately in many cases there is no reliable history whatsoever.

It should be remembered that in stout persons, in those who are gouty, and especially after middle life, there is a tendency to a temporary or in other cases a permanent, but slight, degree of glycosuria.

Diabetes mellitus is not common, but it is increasing, and in Paris, for example, the death-rate from the disease is making rapid strides. It claims, proportionately, most victims among Jews, but the fact that glycosuria and diabetes mellitus are frequently considered synonymous, renders many series of statistics of little real value.

Pathological Anatomy.—There is no question that a tumour, cyst, or softening at or near the "sugar puncture" of Claude Bernard in the floor of the 4th ventricle may cause diabetes, and also that affections of the pancreas or the presence of a pancreatic cyst or tumour may act in the same way. The islands of Langerhans are the chief site of lesion in pancreatic diabetes.

There are, however, many cases of diabetes mellitus in

which the pathological anatomy is eminently unsatisfactory and most of the conditions to be described are either the results of the disease, or may be met with after death in many other totally dissimilar affections. Thickening of the membranes of the brain; enlargement of perivascular spaces; a tendency to colloid masses and corpora amylacea in brain and cord; degeneration, especially of the posterior columns of the cord; multiple neuritis and changes, which can hardly be called typical, in the cells of the semilunar ganglia, the sympathetic ganglia in the neck, and elsewhere, have all been described in the nervous system. In the heart there may be fatty degeneration; in the blood in a very few cases there is a great excess of fat, there is generally an increase in sugar, and a marked diminution in alkalinity, possibly due to the formation of diacetic and other acids, while glycogen occurs free in the leucocytes of the blood and in many organs of the body.

Many patients suffering from diabetes mellitus acquire tuberculosis, and may die of phthisis pulmonalis, while pneumonia of the lobar type sometimes develops, and gangrene of the lung, secondary to either phthisical or pneumonic inflammation, is by no means an uncommon mode of fatal termination. The liver, which we should expect from many researches in the past to show some definite and diagnostic change, may reveal nothing beyond evidences of glycogen in excess in the cells and fatty degeneration, while the kidneys frequently show merely a glycogenic infiltration.

Clinical Features.—When diabetes mellitus occurs in young persons the disease assumes a very acute form, whereas in older patients it is more chronic. The first symptoms are usually thirst and polyuria, and there is rapid loss of flesh, with progressive weakness. In certain cases a history of an apparently trivial glycosuria precedes the disease. The emaciation associated with the dry skin, the scanty hair, and the anxious expression of face, are very typical. In older patients emaciation may not be present, or the loss of flesh may be much more gradual.

Alimentary System.—The tongue is red and sometimes fissured, the mouth dry, the teeth bad, and often the breath has a peculiar odour of acetone, and the gums may be swollen and painful. There may be dyspepsia with acid eructations.

or the appetite may be large and digestion good. Constipation is almost always marked.

Haemopoietic System.—There is an excess of sugar in the blood, the normal 1 to 1·7 per 1000 being often increased to 2·7 up to 5·7 per 1000. Reference has already been made to the rare cases in which fat appears in the blood. The alkalinity is also reduced, probably as the result of the presence of β-oxybutyric or other acid. There is frequently anaemia with reduction of red cells and haemoglobin, or there may be a polycythaemia, due doubtless to the great drain on the blood-serum.

Circulatory System.—There is usually no marked change in the heart or pulse, unless a secondary nephritis has been set up or the patient has developed diabetic coma. In the former condition cardio-vascular changes may develop, and in the latter the pulse becomes excessively feeble.

Respiratory System.—With the exception of the complications of phthisis and pneumonia, to which many diabetic patients fall victims, there is only the remarkable dyspnoea to mention, which, however, is more appropriately described under diabetic coma.

Integumentary System.—The dry skin has already been referred to, and, probably as a result of the irritating qualities of sugar in the sweat, there is great liability to acne spots, boils, and carbuncles, perhaps more specially in the older diabetic patients. Eczema, due to the sugar in the urine, gives rise to much irritation and discomfort about the genitals, and a peculiar bronzing of the skin has been recently attributed to diabetes. There are other rare diseases of the integumentary system which may be present, such as xanthoma, but it is hardly necessary to refer to these here.

Urinary System.—The urine increases from the normal 50 oz. to 200 or even 300 oz. in the day. The colour is typically greenish-yellow; the specific gravity is almost always high (1030-1050). A low specific gravity may be present in milder cases, and too much importance should not be attributed to the daily readings of the urinometer. The amount of sugar present varies greatly, and may rise as high as 10 per cent, the total amount of sugar being 10 to 25 ounces. It has been found that the urea, which is also invariably increased, bears a direct proportion to the amount of sugar, in most

cases being about 1 part to 22 of sugar, and the total amount of urea secreted may reach 1000 or 2000 grams per day. Phosphates are often also in excess in the urine. The waste of sugar is greatly increased by an ordinary diet, and muscular exercise, and in fact any severe strain on the patient, usually raises the sugar excretion. Generally the urine passed during the day contains more sugar than that passed during the night; the latter, in mild cases, may even be free from sugar altogether. Sometimes, owing to the presence of the torula cerviseæ, the urine becomes cloudy, fermentative changes occurring in the sugar. The urine often contains acetone which gives a peculiar odour to it, and diacetic acid, both products of β-oxybutyric acid.

In connection with the estimation of the total amount of sugar, it is well to remember that the fermentation method is perhaps the most accurate, and that, provided there is no albumin present in the urine (which rotates the ray of light to the left), the polariscope also gives an extremely exact estimate of the percentage of sugar, owing to the fact that glucose rotates polarised light to the right.

Perhaps it is well again to remind the reader that there are other agents besides glucose which reduce copper solutions and among these should be mentioned glycuronic acid, alkapton pyrocatechin, hydrochinon, and even uric acid.

The Nervous System.—Practically all the phenomena referred to in this system are complications, many of which however, it is impossible to omit from our description of the disease.

1. Diabetic Coma. This was first described by Kussmaul and is an extremely common cause of death in *young* diabetics probably accounting for quite $\frac{9}{10}$ths of the death-rate in these cases. The onset is often gradual and insidious, the urine falling in amount and the sugar becoming greatly lessened, and it should be remembered that it may ensue on too energetically limiting the patient to a non-diabetic diet, or may result from constipation. The pulse becomes quick and feeble, the surface of the body cold and clammy, the patient nearly always complains of severe abdominal pain, followed or accompanied by headache, and generally there is a considerable amount of excitement. One prominent feature is *air-hunger*, the name given by Kussmaul to the extraordinary dyspnœa

associated with diabetic coma. Without any obstruction to the access of air, the patient seems totally unable to obtain sufficient oxygen.

It is difficult to say what the toxic agent present is, but probably β-oxybutyric acid in the blood is partly responsible. There are three other theories formulated to explain diabetic coma which should be mentioned: the *first*, that fat emboli occur in the lung; the *second*, that it is really due to uræmia, the result of a superadded Bright's disease; and the *third*, that it is an auto-intoxication from the bowel, and that constipation is largely responsible for precipitating an attack. Whatever the cause may be, unfortunately, in most cases, the patient sinks rapidly into coma unless treatment gives prompt relief.

(2) Peripheral Neuritis.—This develops in certain cases of diabetes, and it has been suggested that the neuralgic pains sometimes associated with the disease may have their origin in this way.

(3) Cord Affections.—It is a common feature of diabetes mellitus to observe that the knee-jerks are lost, and the wrist-jerks are usually also absent, while sometimes evidence of involvement of the posterior columns of the cord, as in locomotor ataxia, shows itself. Probably a number of these apparent cord affections are in reality the result of diabetic neuritis.

With regard to special senses, cataract is of frequent occurrence. It is generally of the soft type, while sometimes various defects of vision, such as paralysis of accommodation or central scotoma, make their appearance in the course of the disease.

Diagnosis.—As a rule the diagnosis is easy. It is far more difficult to diagnose a slight degree of glycosuria where it may chance that glycuronic acid or other copper-reducing agent is present. But the amount of sugar found in the urine in diabetes is sufficiently great to prevent any possible mistake of this kind.

To distinguish diabetes mellitus from glycosuria, it is only necessary to remember that in diabetes mellitus it is not possible that diet alone can remove the sugar from the urine, while in most cases of glycosuria a careful dietary will cure the condition, and even where it does not, there are none of the grave symptoms and signs which characterise a typical diabetic case.

The colour-reaction of eosin and also of methylene blue on the red blood corpuscles in diabetes has been used in diagnosing diabetic coma. With eosin they stain less deeply than do normal red cells, and methylene blue gives a yellowish-green colour which is distinctive.

Prognosis. Under the age of 40 the prognosis is grave but after that age it is more favourable. Rapid and permanent benefit from dietetic and other measures should also warrant a better prognosis. The appearance of nephritis, of pneumonia or phthisis pulmonalis, and, it need hardly be added, the development of diabetic coma are all of grave import, and, as already noted, diabetic coma is the chief cause of death in young patients. In patients under 40, even with the utmost care as regards diet and over-exertion, bodily or mental, a case will not probably live for more than a few years, possibly only months whereas in elderly persons the disease may rather be a persistent glycosuria than a true diabetes, and the patients may survive for many years.

Treatment. —The first point of importance is to find out the amounts of urine, of sugar, and of urea excreted by the patient, and if possible on an ordinary diet. After a few days or a week we should endeavour, in the second place, to find out the diminution in quantity which can be effected by a nitrogenous dietary. After a week on this diet, when it may be supposed that the average figures are fairly reliable, we can attempt treatment by drugs. It may be again stated that the complete withdrawal of carbohydrates from the dietary does not arrest the excretion of sugar in the urine in a genuine case of diabetes, although it greatly diminishes the total amount. It should not be forgotten that a rigid nitrogenous diet presents an element of danger, inasmuch as it appears to have conduced to the development of diabetic coma in many severe cases, and therefore it is wise to allow a little bread, a potato, or some such carbohydrate ingredient.

In studying the question of dietary we must endeavour to give the patient a diet upon which he will not lose weight and so far as possible the sugar will not increase in amount. R. T. Williamson suggests that it is desirable to study the heat-producing qualities of the different articles of diet preserved, and this is expressed in calories (a calorie is the amount of heat required to raise 1 kilogramme of water 1° C.). One

gramme of proteid and 1 gramme of carbohydrate each yields by oxidation about 4 calories, while 1 gramme of fat yields about 9 calories. The quantity of these three substances in the diabetic diet is calculated, and their value expressed in calories. Take from the total the value of the sugar lost in the urine, estimating by calories, and the remainder ought to be not less than 2300 calories daily. This is the value in calories of the food required daily by a healthy man not engaged in specially arduous physical work.

The following articles of diet may be permitted :—

Fish of all kinds, excepting those with large livers, which should be avoided, at least in excess; butcher meat of all kinds, excepting liver; poultry and game.

Soups: milk, butter, eggs, cheese, oils, and fats.

Green vegetables, tomatoes, radishes, asparagus, cucumbers, mushrooms, and most kinds of pickles (unsweetened).

The following substitutes for bread are more or less free from carbohydrates:—Gluten and bran bread, bread and biscuits made of almond or cocoa-nut flour. Soya beans may be used in several different ways; they are rich in proteids and poor in carbohydrates. Ordinary bread toasted is very frequently given, and may constitute the carbohydrate element of the diet of our patient; it is often preferable to gluten bread, which is tasteless and uninteresting, and causes the mouth to become tender and painful. Oatmeal has been strongly recommended by von Noorden, and later by others. Its use as a chief dietetic agent does not generally increase the amount of sugar excreted. Oatmeal and butter, with the white of half-a-dozen eggs, constitutes the oatmeal diet.

The following wines and spirits may be taken:—Unsweetened Moselles and many varieties of hock, claret, dry sherry, and unsweetened spirits.

Nuts, practically of all kinds, excepting chestnuts; oranges and lemons, and most fresh fruits, but in strict moderation.

Tea, coffee, and cocoa; lemonade and other aerated waters (all unsweetened).

The following articles of diet are prohibited :—

Liver, and soups thickened with flour or starch.

The following vegetables:—Potatoes, turnips, artichokes and practically all starchy vegetables.

Ordinary bread and all farinaceous food-stuffs, and especially

rice, maize, and wheat flour, which contain a large excess of carbohydrates.

All sweet wines, and especially those of the port-wine class, beer, ale, and porter.

Fruit syrups, fresh fruit cooked with sugar: excessive quantities of the sweeter fruits uncooked, and honey.

Chocolate and cocoa sweetened, and any aerated waters or other beverages containing sugar.

On glancing over the tables just given it is apparent that a large range of articles of diet may be partaken of by the patient, but it is prudent to keep in mind that of those articles permitted many, such as fresh fruits, should be eaten in great moderation. We can make up for the loss of starchy carbohydrates by giving fat, which is easily provided in the form of butter or of lard in cookery. It is wise to endeavour to limit the total amount of fluid which the patient drinks, at least to a certain extent. A strictly dry diet is dangerous, because, just as a diet with too little carbohydrates, it may induce diabetic coma. Hunger days have been recommended, but it is difficult to do more than say they sometimes are found useful in diminishing the output of sugar.

Order a healthy but not excessive amount of exercise for the patient, taking into account the age and capabilities of the individual, and also the type of diabetes present. Forbid all mental strain, and obviate worry and anxiety so far as possible. The patient should have a cold bath every morning, and should be clad warmly, by preference with wool next the skin, and the utmost care must be taken to keep up a free but not excessive daily action of the bowels. There are certain baths where patients improve greatly, partly by the diabetic régime, and partly perhaps by drinking the waters. Of these the best are Carlsbad, Neuenahr, and Vichy, while in winter certain of our patients will benefit by a few months in Egypt during the colder season at home.

The Medicinal Treatment.—Unfortunately, so long as the pathology of diabetes is in its present state, we can offer no satisfactory statement as regards drugs. Opium in the form of codein, morphine, or other of its alkaloids, has perhaps given the best results. The dose should be steadily increased from the equivalent of $\frac{1}{2}$ a grain of opium up to 3 grains thrice daily, and in successful cases marked benefit follows.

but, unfortunately, this is an exceptional experience. There is no possibility of giving internal pancreatic secretion in the same way as it is administered naturally, but duodenal extract or prosecretin (m 5-20) has been utilised for stimulating a sluggish pancreas to more energetic production of its internal secretion. Alkalies are certainly of value, and should be freely administered in the form of aerated alkaline mineral waters, or sodium bicarbonate and citrates can be given in fairly big doses. Rarely do they cause an increase of sugar excretion. For the treatment of diabetic coma alkalies are at least of more benefit than anything else. They may be administered as an intravenous injection, consisting of a 5 per cent solution of sodium bicarbonate in water, and over 400 oz. have been thus injected in thirty hours; certainly 2 or 3 pints should be given at one time. The median basilic vein is usually selected, and the solution should be warmed to blood-heat. Similar or stronger solutions have been used per rectum, but are not so efficacious, and we should remember that this treatment will be more successful if performed *before* diabetic coma actually develops. Arsenic, bromides (sometimes associated with the opium treatment), benzosol (gr. 15-20 thrice daily), sodium salicylate (gr. 15-20 thrice daily), and many other remedies, have been advocated from time to time, but experience does not bear out the special value of any one of these. Atropin has recently been strongly recommended, and may be administered in the form of the sulphate (gr. $\frac{1}{150}$, increased gradually), and calcium iodide has proved of at least temporary benefit in several cases under observation. Cod-liver oil is often a useful agent where emaciation is marked.

The following special symptoms demand a word in conclusion. Excessive thirst can be obviated by giving a little unsweetened lemon juice or some citric acid in water occasionally. Pruritus and eczema are largely prevented by cleanliness, and a sponge soaked in boracic lotion should be used for cleansing purposes after micturition. Remember that excessive fatigue, some cause of worry, and above all constipation, may bring on diabetic coma, and perhaps the best purgative to give is a saline, such as magnesium sulphate, or one of the purgative mineral waters of which the best are Carlsbad, Rubinat, and Hunyadi Janos.

VII. DIABETES INSIPIDUS

A FORM of polyuria associated with great thirst, but in which there is no sugar and no albumin in the urine. In many cases there is much difficulty in deciding as to the cause of the condition.

Etiology.—It is a disease of childhood or young adult life, but even then it is very rare. There is sometimes a hereditary history of the disease, but more commonly the history is tubercular.

Blows upon the head, excessive worry and anxiety, mental excitement, starvation and physical neglect, alcoholism, syphilis, many acute affections, including influenza, and perhaps not uncommonly exposure to cold, have all been stated to be exciting factors. There is no question that lesions in or near the floor of the 4th ventricle have occasionally produced this disease.

Pathological Anatomy.—It is a well-known fact that Claude Bernard, by puncturing the floor of the 4th ventricle at a slightly higher point than the so-called sugar puncture, was able to produce a temporary polyuria. While, therefore, we should expect to find in a certain proportion of cases a lesion at or near this particular point, there are many cases in which a post-mortem examination has yielded no definite pathological results. There is no doubt that exposure to cold weather induces in many persons a very free though temporary polyuria, and it is possible that a few cases may be due to some serious involvement of the vasomotor nerves, although how produced is unknown. Certainly the drinking of huge quantities of fluid, and especially of alcohol, has given rise to the disease, and in hysteria a polyuria is extremely common.

Clinical Features.—The quantity of urine passed by the patient may be from 3 to 10 times the usual amount. The urine is pale in colour, almost like water; the specific gravity is abnormally low, often not more than 1002 to 1005, while the quantity of urine passed may exceed the total amount of fluids drunk by the patients. There is no albumin and no glucose, and although inosite (muscle sugar) is sometimes present, it is probably accidental. In certain cases one finds an excess of phosphates, in others an excess of urates: to the

former the name of "phosphatic diabetes" has been sometimes applied.

In addition to the polyuria and thirst there are other typical features. The patient is emaciated and appears rapidly to lose flesh. The skin is dry. There is nothing worthy of note to be said about digestion, but constipation is a frequent phenomenon. Cardio-vascular changes, such as develop in cirrhosis of the kidneys, are not present.

There are many different types of the disease which might be described. In certain of these the amount of urine passed may not be so great as in others, and the degree of emaciation varies.

There is sometimes arrest, the polyuria diminishing or ceasing, and the patient improving in general health. Other cases, and especially those which are tubercular, may die, partly perhaps from the tubercular condition.

Diagnosis.—The absence of sugar differentiates this disease from diabetes mellitus; the absence of albumin and of cardio-vascular changes prevents confusion with chronic Bright's disease or primary cirrhosis of the kidney.

The **Prognosis** depends, as already indicated, on the type of the disease present and on the degree of improvement obtained from treatment. Very severe cases seem to resist all drugs, and a fatal termination usually occurs within a matter of months.

Treatment.—Every probable etiological factor must be thought of and, if possible, treated. With this object anti-syphilitic treatment, treatment for hysteria, the prevention of cold by warm clothing, should all have the attention of the physician. Try the effect of gradually limiting the amount of fluid taken by the patient. Treat with preparations of valerian, ergot, and opium, as all of these sometimes do good, and the first certainly in cases akin to hysteria. Nitro-glycerine is another useful remedy. Sometimes electrical treatment appears to be of distinct service.

VIII. OBESITY

Some persons have a hereditary tendency to stoutness, and in certain families a single member may become unusually

stout even from childhood. Stoutness is often the result of excessive eating, but many stout persons eat little, and want of exercise cannot always be accepted as a satisfactory explanation. Perhaps the most constant and well-recognised fa is the tendency of people who have passed middle life to become stout, and this tendency is often a hereditary one. The excessive ingestion of carbohydrates aids in the production of fat.

The **Clinical Features** include evidence of fatty infiltration of the heart and inability for prolonged effort or exercise. Stout people are bad subjects for anaesthetics, and it is prud when a general anaesthetic has to be administered, to study the sounds of the heart with special care. Stout persons suffer more severely from any pyrexial disease, because their powers of resistance and of recovery are below the average.

Treatment.—The dietary should be studied so as to eliminate any excess of carbohydrates or fat from the food. Fluids should be cut down to a minimum, and the famous method of Banting includes a great limitation in the quantity of food partaken of. Regular exercise is invaluable, and much good may be done by careful attention to the bowels and the administration of regular doses of purgatives. Many patients derive benefit from a residence at Carlsbad, or other spa, where the waters are purgative in nature. Thyroid extract is sometimes of service when taken for a long period in doses of not over 5 grains once or twice a day. The heart should be systematically examined during treatment, as it is apt to suffer. Arythmia is sometimes caused.

INTOXICATIONS

I. FOOD POISONING

(Ptomaine Poisoning and Grain Poisoning)

It is convenient to divide food poisoning into two groups of cases. In one group the substance eaten is poisonous in itself, and is only partaken of either with intent to kill or by mistake. This group includes poisonous mushrooms, and the flesh of animals or fishes which have before death partaken of foods poisonous to the human subject. The other group consists of cases in which food, not in itself poisonous, acquires poisonous qualities owing to decomposition.

1. Meat Poisoning

This is generally brought about by the eating of decomposing meat, and especially of sausages, pies, and similar partially prepared meat substances. In most of these cases the meat has become tainted, although, possibly, no unpleasant taste may be recognised.

Tinned meat sometimes decomposes, and where, without the application of heat, the jelly about the meat has liquefied, or there is the slightest tainted odour on opening the tin, the contents should be rejected.

The **Clinical Features** of meat poisoning vary considerably, depending a good deal on what organism is responsible for the decomposition of the meat, but more or less gastro-intestinal irritation is present in every case. A period of incubation of varying length may first occur, and then the patient complains of colicky pains with nausea and later vomiting, while rigors are not infrequent. The patient feels chilly, and the forehead is covered with beads of perspiration, diarrhœa

develops and becomes very persistent, and faintness or prostration soon follows in severe cases.

Milk poisoning, or poisoning by the decomposition products of milk, is also the result of a micro-organism, and the clinical features are very similar to those given above. Cheese and ice-cream have also been responsible for serious cases of poisoning, but little is known of the organisms present.

Many shell-fish are poisonous, mainly from the development of a toxin in the liver; a good example is the mussel. Shell-fish occasionally fasten on, and utilise as food, substances which may prove deadly to man.

2. Ergotism, or Poisoning by Ergot

Ergot is a fungus, the *Clavieeps purpurea*, which attacks certain grains, and especially rye, and poisoning generally results from infected grain being partaken of in large quantities as food. The black-looking fungus is easily seen on the heads of the rye-crop. The active principles are believed to be sphacelinic acid and cornutin.

Ergotism may produce (1) a *Gangrenous* and 2 *Spasmodic* type of poisoning.

(1) In the *Gangrenous* form gangrene attacks the extremities, and especially the toes and fingers, while numbness and tingling, sometimes spasms of muscles, and eventually anaesthesia precede the gangrene. Sphacelinic acid has been held responsible for this type of ergotism.

(2) The *Spasmodic* type is characterised by remarkable nervous phenomena, including cramps and spasms involving the muscles of the limbs, and coming on after a period of 1 to 2 weeks. Death occasionally occurs with delirium, and mental changes are extremely common. This variety is said to be due to the cornutin constituent of ergot.

In most cases of ergotism the two types are blended, as in chronic ergotism the pathological changes in the spinal cord resemble those found in ataxic paraplegia or locomotor ataxia.

3. Lathyrism

This is due to the eating of bread or food made from the "chick-pea," the seed of several kinds of vetch. It occurs

in countries such as India and North Africa, in which these seeds are used as food. It causes a transverse myelitis, with both sensory and motor symptoms, and the pathological lesion is a toxic sclerosis.

4. Pellagra

This is due to a disease affecting the maize crop, common in Italy, Spain, and some other maize-growing countries. There is at first weakness, spinal pain, sleeplessness, and the appearance of a typical rash called the "pellagra erythema." The skin is shed, and considerable desquamation and pigmentation occur in connection with it. By the end of 3 or 4 years paralytic symptoms develop resembling spastic paraplegia, sometimes with ataxia, and later still mental changes appear, the patient showing suicidal tendencies. No distinctive pathological change has been described, unless we include the sclerosis of posterior and lateral columns in the cord, although the disease not infrequently proves fatal.

Beri-beri is held by many authorities to be dependent on some organism or fungus associated with rice, and therefore it should be mentioned in connection with this subject. It is described elsewhere.

Treatment for the Preceding Conditions.—It is most important to remove as rapidly as possible any of the food which has caused the poisoning. If a considerable quantity has passed on into the intestine, a purge may be advisable, especially if diarrhoea has not commenced. Intestinal antiseptics, such as β-naphthol or salol, are often of advantage, especially where organisms constitute the chief danger to the patient. Stimulants may require to be freely administered, and where toxins cause contraction of the pupil, it has been suggested that atropine, which dilates the pupil, might therefore act as a suitable antagonistic agent. Certainly sedatives should be administered where convulsive seizures or spasms occur.

In the case of meat, sausages, and more especially tinned meat, rigorous examination should be made of stocks exposed for sale.

Where a rye-crop is seriously infected with ergot, it ought not to be used for food; and diseased maize is also dangerous.

II. ALCOHOLIC POISONING

1. Acute Alcoholism

Alcoholic poisoning produces symptoms with which most people are unfortunately too familiar. The flushed face, dilated pupils, mental excitement, full pulse, and deep respirations are the initial stage, and the loss of muscular co-ordination is soon followed by more or less deep coma. The smell of the breath is typical, and the other points in diagnosis are considered under chronic alcoholism: but the greatest care should be taken to avoid mistaking uraemia, or cerebral haemorrhage for drunkenness, and in all cases the patient should have the benefit of any doubt.

2. Delirium Tremens

This is apt to occur in an alcoholic individual who has been exceeding his usual limits, or who has had a severe strain or an illness such as pneumonia, for which he has been confined to bed. It has been known to develop in persons in apparently good health, but who, after months or years of constant excess, may be forced to lie up owing to some operation, and in them, apparently, the confinement to bed is largely responsible for the onset of the delirium.

Pathological Anatomy.—There is no very satisfactory change which can be called a pathological result of acute alcoholism. In most alcoholic individuals after death the membranes of the brain are milky in appearance, although such milkiness has been found present in the most abstemious persons. Congestion has been described and oedema, but it is possible, judging by recent research, that the oedema may occur post mortem.

Clinical Features.—The patient is sleepless, restless, extremely irritable, and complains of terrifying dreams, and his restlessness greatly increases towards night-time. There is a marked general tremulousness, especially of the tongue and hands. If the attack is severe, the patient soon begins to talk nonsense, discussing his business matters in an irrational or grotesque fashion, and passing from one subject to another

with a total absence of continuity. He has hallucinations of sight, and sees rats and creeping things of all kinds; when he has reached this stage the attack is at its height.

In the alimentary system one notices the furred tongue with marked dyspepsia, indicated by the total aversion to food. The patient's pulse is full, fast, and soft, later becoming feeble and weak. His skin is moist and he sweats profusely, while the temperature is found to be 102°, 103°, or may even rise to 104° F., falling from time to time with the profuse perspirations just mentioned. The urine is concentrated, and contains a trace of albumin. There are sometimes attacks of wild delirium which continue until death, and during these attacks the patient may be guilty of all sorts of impulsive and insane acts, while convulsive seizures, epileptic in nature, are by no means uncommon.

The attack may gradually pass off in 2 or 3 days. In fatal cases, as a result perhaps of continued excitement, the patient collapses; and death may occur from heart failure, from a low type of alcoholic pneumonia, or from sheer exhaustion. Each attack is more likely to kill the patient than its predecessor, a fact which should be used as a cogent argument in dealing with drunkards.

Diagnosis.—As a rule there is little difficulty, because the tremulousness, together with the history of the patient's habits, render a mistake improbable. In *meningitis* and *general paralysis of the insane* there are phenomena present which are absent here.

Prognosis.—Until the patient is really round the corner, it is impossible to make any definite statement.

Treatment.—It is difficult to say whether complete abstinence is better than a gradual diminution in the quantity of alcohol taken by the patient. As a general rule complete abstinence should be insisted on, and the utmost care taken to allay excitement and to prevent the patient doing himself harm during maniacal seizures. He may be either kept in bed with a sheet fastened over his chest, and if necessary a straight waistcoat put on, or he may be placed in a padded room and allowed to exhaust himself without risk of injury. Whichever plan is adopted, it is of the utmost importance to feed the patient regularly and fully. Give milk with two or three switched-up eggs and strong soup at frequent intervals. Try

by the use of bromide of potash gr. 20-40), chloral gr. 20-30), hyoscine (gr. $\frac{1}{100}$ hypodermically), or other sedative to soothe the excitement and restlessness. Care should be taken of the heart, which is so apt to fail, and digitalis or strophanthus must be administered whenever there is the slightest sign of weakening of the pulse. Aromatic spirit of ammonia, spirit of chloroform, and strychnine are invaluable when stimulants are necessary. The patient should be constantly watched, and it is wise as soon as possible to free him from restraint, because restraint impedes the action of the chest, and alcoholic pneumonia is one of the greatest dangers. Lastly, endeavour to keep the bowels open, and to pay attention to the kidneys if the quantity of urine is insufficient.

3. Chronic Alcoholism

There are many ailments which are the result of the constant excessive use of alcohol. All the alcohols act as poisons, and amylic alcohol or fusel oil is one of the most potent and dangerous members of the group. Alcoholic liquors contain very different amounts of absolute alcohol. Spirits contain, as a rule, from 50 to 60 per cent; wine, such as port and sherry, 15 to 20 per cent; light clarets about 10 per cent, and beers and porter from 4 to 6 per cent. Individuals vary greatly with regard to their liability to suffer from alcohol, and opinions differ in the medical profession as to what should be called the proper use of alcohol in health and in disease. Reference to the pathological anatomy will show the effect of alcohol, at least in excess, but it is improbable that in strict moderation it either shortens a man's life or lessens his mental or physical usefulness. The difficulty is to decide what that limit really amounts to, and it certainly varies in different individuals.

Pathological Anatomy.—There is always congestion of the brain in persons who die after great excess of alcohol, and reference has been made to this under delirium tremens. In chronic drinkers the nerve-cells show very marked degenerative changes. There is vacuolation of their protoplasm, with definite changes in the chromatic substance, and, as noted under peripheral neuritis of alcoholic origin, there may be parenchymatous as well as interstitial changes in the peri-

pheral nerves. In addition the cerebral membranes become thickened, and in arteries, kidneys, and heart there is the pathological condition implied by the term arterio-sclerosis, in which the arteries are thickened, the kidneys show primary cirrhosis, and the left ventricle of the heart becomes hypertrophied. Fatty changes are often found in the liver, while common or alcoholic cirrhosis is not infrequently met with in alcoholic subjects. The stomach may show evidence of chronic gastric catarrh, generally of the hypertrophic type.

There are many diseases, especially of the nervous system, which depend to a greater or less extent upon alcoholism: it is only necessary to mention as instances epilepsy and the various forms of alcoholic insanity.

Clinical Features. Chronic gastric catarrh with morning sickness is an almost constant feature in the subjects of chronic alcoholism. There are frequent attacks of jaundice, the face becomes bloated, and generally the body is loaded with excessive fat. Arterio-sclerosis is a common sequel to alcoholism, and occasionally myocardial degeneration. Acne rosacea is sometimes the result of alcoholism, and, when present, adds to the disreputable appearance of the patient's face. The legs are often oedematous, and the urine may contain a constant but small amount of albumin. The chief clinical features, however, are associated with the nervous system, and these include the typical tremor of hands and tongue, the irritable and capricious disposition, and the weakening of all mental powers, and chiefly of the will. The patient becomes excessively untruthful, especially when the matter under discussion is the quantity of liquor consumed or the means of obtaining it, and the constant craving for spirits completely overrules any transient resolution towards reform. The worst type of these patients is to be met with in women, who often indulge secretly and sometimes in enormous excess. It is in women that one meets most frequently with peripheral neuritis, which, although it seriously cripples the patient for the time being, has the advantage of offering a chance of cure from the alcoholic habit. The wrist-drop and foot-drop, with the muscular tenderness on palpation, and the history of cramps, should suggest to the physician the possibility of alcoholic neuritis.

The sequelae of chronic alcoholism are chiefly diseases of

the circulatory and nervous systems, and include arteri sclerosis, epilepsy, and alcoholic insanity, while prolonged excess in the use of alcohol greatly interferes with the vitality generally, and may therefore indirectly predispose to many diseases, such as pneumonia, erysipelas, and tuberculosis.

Diagnosis. It is easy to recognise an individual who is hopelessly drunk, but to prevent the possibility of error in diagnosis the iodoform test for alcohol in the urine is a good one. If a small quantity of urine be heated with some iodine and caustic potash, iodoform crystals will be precipitated in the presence of alcohol. Generally the odour from the breath and the smell of alcohol in the stomach contents prevent any mistake, although it is well to remember the possibility of *uræmia*, *diabetic coma*, *cerebral hæmorrhage*, or *concussion* giving rise to symptoms closely corresponding to drunken stupor.

It is often excessively difficult to secure a confession from the subjects of chronic alcoholism, because the want of truthfulness already mentioned, and the natural anxiety of the patient to conceal an alcoholic history, obscure the case, and much tact is necessary in dealing with women in order to arrive at an accurate opinion. The cross-examination of those living with the patient, and an effort to trace the source of supply of the alcohol, may be needed before the truth is elicited.

There are many causes of tremor other than alcoholism, but a little care will discriminate even an early case of *disseminated sclerosis*. *General paralysis of the insane* often closely resembles chronic alcoholism, but there is no morning sickness, and an examination of the pupils (irregularity of Argyll-Robertson pupil in general paralysis) should clear up the case.

Prognosis. There is always a tendency for patients who have been alcoholic to relapse, not once, but many times; and probably the greatest danger is the want of will-power, while it should be remembered that the craving for alcohol may become in reality an insane impulse absolutely beyond all possible control on the patient's part.

Treatment.—No treatment is so successful as shutting off the patient from all possible source of supply. This is best done in institutions or asylums, where alcoholic patients are admitted as voluntary inmates. The place of alcohol should

be taken by strong coffee, but not by the use of opium or other hypnotics, unless under exceptional circumstances. Hyoscine (gr. $\frac{1}{100}$) administered hypodermically is an admirable remedy for diminishing excitement and restlessness, while bromides and chloral are often of great value. The same care should be taken of the diet as was recommended under delirium tremens although, as a rule, the patient is not unwilling to eat fairly well. The gastric condition may be treated sometimes by washing out the stomach, sometimes by administering small doses of rhubarb root (gr. 2), sodium bicarbonate (gr. 10), and a carminative, such as powdered ginger gr. $\frac{1}{2}$). Strychnine, phosphorus, arsenic, and cinchona are amongst the most useful remedies which we can employ. The much-vaunted gold-cure consists in the injection hypodermically of about $\frac{1}{10}$ gr. of terchloride of gold in water. There is no particular virtue in the treatment, and probably much of the benefit is really derived from the care taken of the patient, and the endeavour to secure more healthy conditions of life. Persons who have been the subjects of chronic alcoholism and who have been cured should on no consideration be exposed to the risk of relapse, and it is desirable to prevent the use of alcohol in all forms in the homes of individuals who have once been dipsomaniacs.

III. MORPHINOMANIA

(Morphinism: Morphia Habit)

Acute Morphia Poisoning is readily recognised in most cases. The patient passes through a brief period of excitement into a deep coma, and death may result with great rapidity, the breathing becoming more difficult as the respiratory centres are narcotised. The pupils are contracted until immediately before death, when they may dilate.

Chronic Morphia Poisoning is the condition with which, from a physician's point of view, we have generally to do. Morphia, whether opium, morphia, or some other alkaloid of opium, may be taken by the mouth, injected hypodermically, or inhaled in an opium-pipe. Many persons acquire the habit of taking some preparation of opium for the relief of

pain, whilst others, discovering the soothing effects of the drug, acquire the habit in an insidious manner. Opium, if taken in small enough quantity, temporarily stimulates the brain to greater effort, and only produces the sedative and narcotic effects in larger doses. Unfortunately, the effect on the patient is by no means trivial or short-lived. The most intense depression, and moral as well as mental deterioration, soon render the victim of opium a miserable and wretched individual. Not infrequently alcohol is taken for the relief of the depression, and in order to produce the desired effect of the opium larger and larger doses of the drug require to be taken. There is much restlessness, itching of the skin, sleeplessness unless the drug is regularly taken, and general discomfort, while the skin of the face becomes sallow and muddy-looking, and the dress untidy and dirty, appearances which readily indicate the habitué of opium. Should the drug be suddenly stopped, the patient becomes intensely excited, very miserable, and the craving almost beyond endurance.

Treatment.—For an acute case of opium poisoning the stomach must be at once washed out, or an emetic given, and afterwards strong coffee administered to the patient. On no consideration should he be allowed to sleep, but he should be made to walk up and down for several hours after the drug has been removed from the stomach. The faradic current may be applied to the heart, where cardiac failure is threatening, and cutaneous stimulation is often necessary. Strychnine is one of the best remedial agents in opium poisoning.

In cases of chronic poisoning it is wise to withdraw the drug gradually, and, as a rule, to induce the opium-taker to become an inmate of an asylum for at least six months. During the period of inevitable discomfort much may be done by careful feeding, by the use of strong coffee and similar remedies to keep up the patient's strength, while other sedatives than opium have sometimes to be resorted to for the relief of the restlessness and the insomnia.

IV. COCAINE POISONING

(Cocainism)

This alkaloid, obtained from the *Erythroxylon coca*, is largely used in both surgical and medical practice, and a habit may be acquired similar to morphinism.

The initial stimulant effect is followed by great depression, for which alcohol is frequently taken by the patient. It is remarkable in how short a time the patient deteriorates mentally and physically, and the treatment for a case of cocainism should consist in seclusion, the patient being in this way unable to obtain the drug. Sometimes the depression stage is so severe as to demand the use of powerful stimulants, and it is during this period that death is apt to occur where an overdose has been administered.

V. TOBACCO POISONING

Persons unaccustomed to smoking, and those who smoke too strong tobacco and in too large quantities, may suffer from this kind of poisoning. It produces cardiac dilatation and often irregularity, a very feeble pulse being associated with intense nausea and a tendency to syncope. The respiratory centre is apparently paralysed before the cardiac centre, and for the treatment of cases of acute tobacco poisoning, artificial respiration is generally necessary.

VI. METALLIC POISONING

1. Lead Poisoning (Plumbism)

Lead poisoning is apt to occur amongst workers in certain trades, and especially plumbers, painters, glaziers, and lead-miners, while lead in considerable quantity may find its way into drinking water when conveyed by new lead pipes or stored in new lead cisterns, and in connection with this it should be stated that soft water is specially apt to become

contaminated when in contact with lead. Less frequently lead may be found in wines, ciders, and other alcoholic beverages, and certain hair dyes owe part, at all events, of their colouring properties to lead. Lead may thus enter the system by the alimentary tract, by the lungs, and also by the skin. Some persons possess a peculiar idiosyncrasy to lead, and poisoning may occur at any age.

Pathological Anatomy. — Lead is believed to have a special influence in the production of arterio-sclerosis; it also causes a form of peripheral neuritis, referred to later, and nerve-cells may show degenerative changes.

Acute lead poisoning is extremely rare, and it is therefore only necessary here to refer to the more usual chronic form.

Clinical Features.—(1) Lead Colic.—Colic is perhaps the most common result of lead poisoning. It in no way differs from ordinary colic, unless it be in its duration and the consequent prolonged agony from which the patient suffers. There may be vomiting, and sometimes acute attacks of diarrhœa.

(2) The blue line on the gums, which does not inconvenience the patient, is an extremely valuable clinical feature for the purpose of diagnosis. It is situated at the margin of the gum, and is due to a precipitate of sulphide of lead by the sulphuretted hydrogen derived from decomposition changes in connection with the teeth. It need hardly be remarked that the lead which forms the sulphide circulates in the blood, and is deposited in minute blue-black specks in the papillæ of the gum. The line may be seen for a long time after the source of poisoning has been stopped.

(3) Severe pains in the back and head are not infrequent in connection with lead poisoning. Marked mental changes often supervene in cases of lead-encephalopathy, and these patients tend to become asylum inmates. One case seen by the author developed nervous symptoms closely resembling general paralysis of the insane.

(4) Lead paralysis, which is described elsewhere under neuritis, and of which four types may be met with.

(5) Anæmia is often severe, and a lead cachexia is not uncommon. A number of observers have noted that the red blood corpuscles show a peculiar colour-reaction with certain stains, which is believed to indicate a basophilic degeneration.

In most cases of lead poisoning one or other of these

clinical features is present, but it is rare to find a case in which all are equally marked.

Diagnosis.—Lead is rapidly eliminated by the kidneys, and therefore may be obtained from the urine. The wrist-drop, when present, is extremely typical, and the blue line on the gums often prevents mistake.

Prognosis.—Recovery is probable in most cases in which the condition is recognised and the introduction of the metal into the system promptly stopped.

Treatment.—For the treatment of a case of lead poisoning potassium iodide is the most important remedy, and it should be administered in doses of 10 to 20 grains thrice daily, while magnesium sulphate is given every morning before breakfast in a dose sufficient to produce a free motion. By this means the lead is fairly rapidly eliminated from the system. Colic induced by lead demands similar treatment to other forms of that painful complaint, fomentations or poultices affording much comfort to the sufferer. The removal by lumbar puncture of a little fluid often relieves the extreme pain in the head and back, and in lead paralysis, treatment by strychnine hypodermically, by massage, and electricity, yields satisfactory results. The patient is always anaemic, and iron should be freely administered.

Prophylaxis.—Lead piping and lead cisterns should never be employed where the water is soft, unless it has been previously protected by producing a precipitate of carbonate of lead which forms a protecting skin. Where white or red lead is manufactured or extensively used, the hands should be thoroughly cleansed prior to eating, and where the lead-dust impregnates the atmosphere, a suitable mask should be worn by the workers.

2. Arsenic Poisoning

We have only to consider chronic arsenic poisoning here. It is apt to result from arsenical wall-paper, artificial flowers, or any coloured material used for dress or in connection with the house furniture, paper, or curtains in which an arsenical dye is used. Apparently arsenic may pass into the atmosphere in solid particles or in organic compounds, formed partly by fermentation of certain moulds. Recently quantities of arsenical beer have caused much trouble, due to contamination of the

glucose. The commonest cause of arsenical poisoning is the administration of arsenic to patients suffering from pernicious anaemia or chorea, the dose being carelessly increased until arsenical poisoning supervenes.

The **Clinical Features** include nausea and pain in the stomach, puffiness and congestion of the eyelids, and eventually vomiting and diarrhoea. The gums are apt to bleed, and salivation may occur, and later the phenomena of arsenical neuritis referred to under the nervous system may develop. Pigmentation of the skin and marked keratosis (thickening of the skin of the palms and soles, the thickened cuticle having a peculiar yellow colour) are often seen in cases of chronic arsenical poisoning.

The **Treatment** consists in the endeavour to remove the drug from the system, and probably potassium iodide is the best remedy. The arsenical paralysis should be appropriately treated.

3. Phosphorus Poisoning

Both acute and chronic forms of phosphorus poisoning may be met with in persons working at trades in which phosphorus is largely used, and especially in the making of matches.

Clinical Features.—The symptoms of **acute** phosphorus poisoning are severe gastric irritation, with pain over the liver and jaundice. The patient rapidly becomes weak, the pulse feeble, and death generally occurs in about a week, often from cardiac failure.

The symptoms of **chronic** phosphorus poisoning are profound ill-health with anaemia, frequently pain over the liver, and especially the development of phosphorus necrosis, commonly called "phossy-jaw." As a result of this the teeth may fall out, and the patient's health suffers seriously.

The **Treatment** consists in the removal of the phosphorus as rapidly as possible. In acute cases an emetic should be given if phosphorus is present in the stomach, and a weak solution of sulphate of copper will form a black phosphide of copper which is inert, although the remedy is mainly given to induce emesis.

In the more chronic cases careful dieting should be carried out and the surgical treatment of phossy-jaw must be thorough in order to be satisfactory.

VII. SNAKE-BITE

THERE are many poisonous snakes in hot countries, and in our own land the ordinary viper or adder (*Pelias berus*) is sometimes very deadly.

The active principles of snake venom may be divided into two bodies:—

(1) A coagulable proteid poison which causes disintegration of red blood corpuscles, and also acts directly on cardiac muscle.

(2) A non-coagulable proteid or albumose, which acts specially on the great nerve centres in the medulla, paralysing the respiratory centre and also the nerve-endings in muscle.

Snake venom differs widely as regards the amounts of these poisons present.

Sir Thomas Fraser and Calmette have obtained a serum called antivenene as the result of inducing immunity by repeated graduated doses of venom, and Calmette finds that the antivenene obtained by using venom of the cobra de capello is capable of counteracting all venoms, including that of scorpions.

Clinical Features.—There is rapid local swelling, with extensive haemorrhages owing to the disintegrating action of the coagulable proteid, and this is most marked in the affected limb. Cellulitis, and even gangrene, may ensue in cases of very poisonous bites. There is a tendency to heart failure, dyspnœa, and eventually coma and death.

The **Prognosis** depends (1) on the kind of snake which has inflicted the bite, (2) the strength of the individual bitten, (3) the part of the body bitten, whether the bite is on exposed skin or on skin covered by clothes, and (4) the possibility of prompt treatment.

Treatment.—Prevent the poison entering the system by applying a ligature *above* the part bitten, if it be a limb. Try to suck out the venom from the bite, taking care that there is no lesion of the buccal mucosa. Administer stimulants, and especially alcohol and ammonia, and apply strong liquor ammoniæ or permanganate of potash locally. Give antivenene if obtainable, and do so at once.

SUNSTROKE

(Heatstroke; Heat Apoplexy; Siriasis

This is a group of conditions attributed to the heat of the sun, exposure to excessive heat not necessarily solar, and especially exposure to heat when there is an undue amount of moisture in the atmosphere.

Etiology.—The predisposing causes are extremely important. Physical exhaustion, unsuitable clothing, an alcoholic habit, and bodily debility, all render the individual more liable to attack. The sun causes sunstroke in hot countries when either the head, neck, or back is exposed to its rays. It certainly seems more common near the sea-level and in valleys, whereas at high altitudes it is almost entirely unknown.

Pathological Anatomy.—Decomposition speedily follows death, there is much post-mortem staining of blood-vessels, and there is usually congestion of the brain and cord. It is very difficult to assert that there are definite central changes which can be called pathogenic.

Clinical Features.—There are two types of cases—those associated with fever, and those without fever, and both these types might be more elaborately subdivided.

The *Apyretic Type* is less severe; it consists in extreme exhaustion with threatening of syncope, a subnormal temperature, tendency to vomiting, dilated pupils, and a small, rapid pulse. Some cases show more dyspnœa than others, and profuse sweating is usually a prominent symptom. A subvariety frequently associated with fainting turns, has been termed syncopal, but the distinction is unimportant.

In the *Pyretic Type* of sunstroke there is hyperpyrexia, and to this form the term *Siriasis* is specially applied. It may kill in a few moments, or the patient may survive for some hours or days. There is usually excessive weakness, with

flushing of the face, an intensely dry skin, and a temperature which rapidly rises to 106°, 107°, or even 110° F. There is marked dyspnœa, and a quick, bounding pulse, while cramps occur in the muscles of the legs, and convulsive twitchings in the muscles of the face and sometimes of the limbs. There may be a trace of albumin and sometimes blood in the urine. Headache, often extremely severe, mental confusion, and even maniacal symptoms are sometimes associated with this type. These cases may rapidly die, or recovery may occur in those which are less severe, but convalescence is slow, and not infrequently, although the patient does recover, he is found to be unable to reside in a hot climate, and to be mentally unfitted for much, if any, strain,—in fact a degree of insanity is not uncommonly a sequel to sunstroke in those cases which do recover.

The **Diagnosis** is generally fairly simple: the history of exposure to great heat, whether to the sun's rays or in the stokehold of a steamer, together with any predisposing factor in the patient's history, is usually sufficient, while an ordinary *apoplectic seizure* almost always causes a definite and usually a unilateral paralysis.

Treatment.—In the *Apyretic Type* absolute rest is necessary, and the patient should be kept cool, while vigorous stimulation by alcohol, ether, or ammonia is at once resorted to. Strychnine is often of advantage, and ice may be administered.

For the *Pyretic Type* there is probably no remedy so satisfactory as the application of cold water either to the whole body or to the head and back. Iced cloths when obtainable result in rapid improvement. Strychnine and alcohol, and other stimulants, may need to be administered, but are not so necessary for this type. Sometimes the head requires to be shaved and blisters applied. The patient should be given a small dose of calomel and fed on a light diet.

Prophylaxis.—The prophylactic treatment is simple. Persons resident in hot climates should be enjoined to be temperate, especially as regards alcohol. The head, neck, and back must be protected from the sun's rays, and the sun helmets worn in tropical countries are of great value. On no consideration should individuals who have suffered from sunstroke be permitted to remain in the tropics if they find any exhaustion or mental aberration associated with exposure

to excessive heat. Probably there is no remedy so valuable for the prevention of sunstroke as water, but, unfortunately, it is difficult for troops on the march in a hot sun to find sufficient water to wet the head and back. The proper ventilation of stokeholds in steamers, has done much to obviate heatstroke occurring.

DISEASES DUE TO INTERNAL PARASITES

I. CESTODES

THERE are three important cestodes which in their adult state are found in man, namely, Taenia solium, Taenia mediocanellata, and Bothriocephalus latus. The head and neck, also called a scolex, the body or strobila, and the segments of that body or proglottides, are common to the group. The terminal segments are ripe and contain testes and uterus, and the latter is full of ova, which are generally evacuated. These ova are often ingested by a suitable host, the shell is digested, and a six-hooked embryo set free, which bores through the intestinal wall, and eventually forms a cyst-like bladder or cysticercus (except in the case of Bothriocephalus, in which the larval form remains free). When the cysticercus, or larval form, is eaten by man the adult worm develops in the intestine. Usually the head of the worm is situated high up in the small intestine.

1) **Taenia Solium** is common on the Continent, its cysticercus form being found in the pig. It is 8 to 15 feet in length. The head, which is about the size of a pin's head, has two rows of hooklets and four sucking discs. The genital pores open laterally, and generally on alternate sides in the proglottides. The uterus has about a dozen lateral offshoots which ramify considerably. The ova are 30 μ to 35 μ in diameter.

2) **Taenia Mediocanellata** or **Saginata** is the usual British form, as its cysticercus is found in the ox. It is 15 to 20 feet in length. The head is larger than the last and has four sucking discs, but no hooklets. The genital pores open laterally and have no tendency to alternate, and the uterus possesses often as many as thirty lateral branches, which

do not ramify. The ova are slightly larger than those of Taenia solium, and more oval.

(3) **Bothriocephalus Latus** is common in Russia, the Baltic, Switzerland, and elsewhere. Its larval form occurs in fresh-water fish, especially pike and trout. It is 20 to 30 feet in length. The head is long, with a long sucking groove on either side, and the segments are extremely broad, possessing a rosette-shaped uterus in the middle, and the genital pore opens in the centre of the rosette. The ova are about twice as large as those of Taenia solium.

The **Clinical Features** of the different forms of tapeworm are sometimes not distinctive, excepting that proglottides are found from time to time in the stools. The patient may not suffer at all, but colicky pain, dyspepsia, voracious or capricious appetite, and sometimes constipation, may be present. In some rare cases nervous phenomena appear—fits and even mania being noted. Bothriocephalus is credited with producing very profound anaemia. Tapeworms probably live for years in the host, and there may be several individuals present at once.

Diagnosis.—The position of the genital pore should render Bothriocephalus unmistakable, and the branching of the uterus in the more mature segments is sufficient to distinguish the other two commoner tapeworms from each other.

Treatment.—First starve the patient for some hours, give a purge, and follow with a dose of an anthelmintic, such as $1\frac{1}{2}$ to 2 drachms of filix-mas in a carminative infusion, such as cinnamon water. It is generally dispensed as an emulsion. Often smaller doses are administered every two hours, and in any case a purge must follow. Given on an empty stomach, the remedy is most efficacious, although it may require repeated doses to bring away the head. The head should be carefully searched for in the stools. Kousso, kamala, and other remedies may be substituted for filix-mas.

Visceral Cestodes

1. The **Cysticercus Cellulosae**, or cystic form of the Taenia solium, is sometimes found in man, when he becomes the intermediate host in place of the pig. It is not easy always to discover how the ova enter the stomach, and it has been suggested that in certain cases where the adult taenia is present

in the intestine, the proglottides may be forced upwards into the stomach during prolonged vomiting.

The cysticerci only cause trouble when they are found in certain parts of the body, and especially when they reach the brain or cord. There appears to be little evidence of general discomfort during the boring of the embryos through the stomach wall. In the brain or cord the cysticerci may give rise to the manifestations of tumour, or there may be no symptoms at all. In several recorded cases cysticerci have been found in the eyeball.

The cysticerci of Taenia mediocanellata have very rarely been found in man.

11. **Hydatid Disease.**—The adult tapeworm (Taenia echinococcus) occurs in the dog, but is extremely rare in this country. It is only some 4 mm. in length, consists of 4 segments, of which the head resembles the head of a tiny Taenia solium, with two rows of hooklets and four sucking discs. The terminal segment is alone mature, and contains several thousand ova.

If the ova are ingested by man, they develop into little six-hooked embryos which bore through the intestinal wall, and may reach the peritoneum, or a blood-vessel, or lymphatic. When finally located it forms a cyst which rapidly grows in size. The cyst wall is formed of a chitinous ectocyst and a granular endocyst or germinal membrane. The cyst contains a watery fluid 1005 to 1009 specific gravity, rich in sodium chloride. Soon buds appear on the germinal membrane and form daughter cysts: from the germinal membrane of these, grand-daughter cysts may develop, and the daughter and grand-daughter cysts become free. From the germinal membrane of all these cysts brood-capsules develop, each of which forms one or more embryo worms possessed of a head which resembles that of the parent, and which can be everted at will. These are called scolices, and these scolices can become adult worms if ingested by the dog. Sometimes the daughter cysts develop outside, and are called *exogenous*, while a rarer form occurs, in which each cyst after separating from the parent is surrounded by a thick capsule, forming eventually a multilocular cyst.

Cysts may be sterile, and many cysts contain hooklets and shreds of chitinous ectocyst, the result of degenerative changes. The cysts may grow to a huge size and cause much trouble.

They may suppurate, they may burst into various structures (sometimes where situated in a kidney, into the pelvis of the organ, and the daughter cysts cause renal colic when attempting to pass down the ureter).

Hydatid disease is most common in Iceland and parts of Australia, where dogs and men are very closely associated. The cysts occur in different organs in the following frequency: Of 1862 cases investigated, 953 were found in the liver, 165 were in the intestinal canal, 153 in the lung or pleura, 150 in connection with kidneys, bladder, and genitals, and 127 in the brain and spinal cord.

The **Clinical Features** depend on the organ in which they are situated.

Reference is made under diseases of the liver, kidney, and lung to the presence of hydatid cysts in these organs, with the clinical features in each case, but it must be remembered that hydatid cysts may be found in the brain, in connection with the peritoneum, and in almost any part of the body. The fact that the cysts may continue to grow, adds to the risk of pressure by the cyst on neighbouring structures, and suppuration from infection by pus-producing organisms has occurred in not a few instances.

Diagnosis.—The character of the hydatid fluid already described should, as a rule, prevent any mistake in diagnosis.

Treatment.—The evacuation of the cyst is the one satisfactory method of treatment. Sometimes aspiration is sufficient to effect a cure, while in other cases and especially where suppuration follows, it may be necessary to freely open and drain the cyst. The introduction of antiseptic or other fluids into the cyst, or the attempt by electrolysis to kill the embryos within, should not as a rule be attempted.

II. NEMATODE WORMS

These worms taper towards each end. The males, always smaller than the females, have a convoluted testis which opens at an orifice common also to the alimentary canal, while the female possesses two convoluted ovarian tubes which open into the vulva, placed anteriorly. Some of the group are oviparous, others viviparous or ovo-viviparous. The embryos in certain

instances pass through a cycle outside or in a different host—in others the ova are swallowed, and the embryos mature in the alimentary tract.

Oxyuris Vermicularis or **Thread-Worm.** This worm is about ¼ to ¾ inch in length, the female being 2 to 3 times larger than the male, and resembles a small piece of white thread. The ova are oval, and measure about .05 mm. × .02 mm. It is a widely distributed parasite. The habitat is the large intestine from the cæcum to the anus. Auto-infection is common, the ova being introduced into the patient's mouth. The worms often wriggle out of the anus, causing intense itching, and may even enter the vagina in females. They are found in large numbers in the stools, and are seen to move for some time after exit from the body.

The **Clinical Features** are intense itching of anus, sometimes nose-picking, colicky pain, and often reflex nervous phenomena, such as restlessness during sleep with grinding of the teeth, occasionally chorea, and sometimes convulsions. The appetite may be voracious.

The **Treatment** consists in repeated enemata of infusion of quassia, salt and water, lime water, or similar remedy, while internally 2 to 3 grains of santonin will be found of much service. Attend to the risks of auto-infection: a weak mercurial ointment may be used locally with benefit.

Ascaris Lumbricoides or **Round Worm.**—The ascaris is a common parasite in many lands, and especially in warm countries. It resembles a reddish earth-worm—is pointed at both ends, with four longitudinal bands and numerous transverse striations. The female is 7 to 12 inches, the male 4 to 8 inches in length. The ova, .07 mm. × .05 mm., are oval in shape. They possess a thick, chitinous shell, and are found in large numbers in the stools. The worms vary in number from 1 to 3 in a child, and 2 to 5 in an adult, but 50 or more have been present in one host.

The habitat is the upper part of the small intestine.

The **Clinical Features** vary: often some colicky pain or discomfort, picking the nose, and scratching the anus, not infrequently slight fever, and voracious or capricious appetite, are noted: while the reflex nervous phenomena may be more

marked than in the case of thread-worms, and twitchings convulsions, teeth-grinding, and restlessness at night may be more or less in evidence. A poison, which is possibly responsible for the intestinal symptoms, has been stated to be formed by the ascaris. In many cases there are no symptoms whatsoever. Very rarely an ascaris finds its way up into the stomach and is vomited, or blocks the ductus choledochus.

The **Treatment** consists in santonin, 1 to 5 grain doses, to be followed by a purge and preceded by rather low diet. Santonin should be administered with care, as it sometimes upsets the patient, producing yellow vision amongst other unpleasant phenomena, and a night or two should elapse before repeating the dose.

Anchylostoma Duodenale, Dochmius Duodenalis. Hook or Anaemia Worm.—The female is $\frac{1}{2}$ to 1 inch, the male about $\frac{1}{3}$ inch in length, and they are of a reddish-brown colour. The ova are numerous and measure ·06 mm. × ·04 mm. The mouth of the parasite is provided with sharp hooks or teeth by which the worm fixes on to the mucosa. The male possesses an umbrella-like expansion at the tail, distinguishing it from the female.

The worm is common in many warm countries, such as India, Brazil, and Egypt: it also occurs in Switzerland, and was especially baneful to the navvies when the St. Gothard tunnel was being pierced. It has recently been met with in Cornwall.

The habitat, as the name implies, is the duodenum and small intestine, and there may be numerous worms present.

The **Clinical Features** include dyspepsia with epigastric tenderness, very profound anaemia with its concomitant symptoms, such as langour, breathlessness, and increasing debility, and later vertigo and even syncope, while oedema of the ankles may develop. Colicky pain and diarrhoea are common, sometimes melaena, and death may result if the case is not treated. An eosinophilia is an important and fairly constant feature.

Diagnosis.—The ova in the stools render the diagnosis easy.

The **Treatment** should consist of 15 to 30 grains of thymol in wafer paper, to be taken on an empty stomach, repeated in

two hours, and followed by a purge. The thymol may require to be taken again a few days later. Filix-mas is sometimes preferred. The anæmia demands energetic treatment. The prophylactic treatment consists in guarding the water-supply from sewage contamination, and in destroying the dejecta of infected persons.

Trichocephalus Dispar or **Whip-Worm.**—This worm measures about 2 to 3 inches in length, the female being slightly larger. The whip-like anterior part of the worm is about $\frac{3}{5}$ths the length of the rest of the worm, and the posterior and thicker part is coiled up like a spring in the male parasite. The ova, ·05 mm. × ·02 mm., have a distinctive little node at either end, and possess a relatively thick capsule.

The worm is widely distributed, and especially in many warm countries.

The habitat is the cæcum and large intestine.

The **Clinical Features** are often *nil*, but there may be severe diarrhœa.

The **Diagnosis** is readily made from finding the ova in the fæces.

Treatment is usually not required, but, where necessary, thymol by the mouth and also by the rectum should be tried.

Trichina Spiralis.—The embryos are encysted in voluntary muscle, and the migration of the embryos from the intestinal canal to become so encysted, form the disease in man.

The adult worm is found in the small intestine, the female measuring 3 to 4 mm., the male 1·5 mm. in length. The encysted trichina is ·5 to 1 mm. in length, and is contained in a capsule which, after a time, is infiltrated with lime salts.

When trichinous pork (uncooked) is eaten by man, the capsules are digested, the worms maturing in three days. Hundreds or thousands of embryos are produced from each female about the 7th to the 9th days, and these embryos pierce the intestinal wall and find their way possibly by the lymph channels to the muscles, and in about two weeks become encysted. The cysts may be seen as minute white specks (when lime salts have been deposited) in the muscle. All the muscles may be affected, but perhaps most constantly the

abdominal muscles, the diaphragm, and the intercostal muscles. Raising meat to the boiling-point kills the encysted worms, and therefore it is smoked or imperfectly cooked meat which is dangerous. The pig is the infective agent, and the ratio of trichinous to healthy animals differs greatly, and is certainly rarely over 5 per 1000 in any country. Inspection of meat has diminished cases of the disease, but one infected carcase may lead to a local outbreak.

The **Clinical Features** are fever during the boring of embryos, with abdominal pain, diarrhoea, and nausea, and sometimes these phenomena begin early and go on for 7 to 10 days. Typhoid has not infrequently been suspected. In the muscles a very definite myositis results, with pain on pressure and movement, local swelling, and almost always marked and often general cutaneous oedema. A very characteristic oedema of the face is noted especially at the commencement of the myositis. The muscles become stiff, a leucocytosis reaching 30,000 with a remarkable eosinophilia (40 to 50 per cent of leucocytes) is fairly constant, and skin eruptions such as boils and urticaria sometimes occur. Dyspnoea depending on the involvement of the respiratory muscles, and difficulty in eating, speaking, and so forth, due to the same cause, may ensue, and the patient dies after a prolonged period of increasing ill-health.

The **Diagnosis** may be made by examining a piece of the affected muscle, while the worms are found in the stools up to the 10th day or thereby after ingestion of the infected meat. The blood should be examined for a leucocytosis and eosinophilia. Isolated cases are rare.

The **Prognosis** depends much on the degree of early intestinal irritation, because diarrhoea favours the removal of the trichina before maturing. The death-rate varies greatly, being from 2 to 25 per cent.

The **Treatment** consists in free purgation if the case is got at an early stage, and filix-mas, santonin, or thymol are also recommended as vermifuge remedies. General treatment for the myositis is all that can be suggested later. Prophylaxis consists in the systematic and careful inspection of pork.

Filaria Sanguinis Hominis.—There are four or more species of filaria whose embryos may appear in the blood of

man. These embryos are all long, 100 μ to 350 μ in length, and 5 μ to 12 μ in breadth. They taper posteriorly and have transverse striations. We do not know so much about the adult filaria as about the embryos.

The *Filaria Bancrofti* is 5 inches in length, the male being smaller, and resembles a whitish horse-hair: the worms are found in the blood-vessels and also in the lymphatics. They may cause no trouble, but as the result of their death filarial abscesses may develop, and they may block the thoracic duct or lymphatic trunks, and cause extensive varicosities of these channels. This may occur in many positions, and rupture leads to lymphorrhagia. If near the kidney, they cause chyluria; if in the groin, large tumour-like masses, and similarly lymph scrotum, and what is really elephantiasis of the leg are produced. It is difficult to explain the exact conditions under which elephantiasis and kindred affections arise. It may be from injury of the worm or worms, perhaps causing premature discharge of ova, and the ova may cause the blockage. In any case inflammation occurs with great local thickening of skin, and sometimes huge swellings. Where the embryos can enter the blood, *Filaria Nocturna* is found in great numbers. Where the filaria nocturna is prevalent in tropical and sub-tropical countries, it is often found in the blood of a large percentage of the inhabitants, and is probably transmitted from one to another by mosquitoes. This embryo has a long, translucent membrane which projects beyond the worm at both ends. The embryos appear in the peripheral blood-vessels at night from about 6 P.M. to 8 A.M., increasing in numbers up to midnight. In the day-time the embryos are found in the lungs and large internal blood-vessels. In the mosquito a cycle of development lasting from 2 to 3 weeks occurs: it develops a 4-lipped mouth and 3-lobed tail, grows much larger, and finds its way eventually to the proboscis, ready to pass into a fresh human host, in which it becomes a Filaria Bancrofti again.

The *Filaria Diurna* is the embryo of another species which is only found in the blood during the day. It closely resembles Filaria nocturna, and is found in the Niger country.

The *Filaria Perstans* is a smaller embryo, found in certain parts of West Africa, and especially the Congo, and has no special nocturnal or diurnal habits.

There are also other varieties of which our present knowledge is somewhat slender.

The **Treatment** is largely directed to the condition resulting from blockage of lymphatics (see Chyluria, p. 529).

Dracontiasis, Dracunculus Medinensis or **Guinea-worm.**—This worm is common in West Africa, India, Persia, Arabia, Brazil, and elsewhere. It inhabits the connective tissue of man.

The guinea-worm is 1 to 4 feet in length, and about $\frac{1}{10}$ inch in diameter. The male has not been found. Usually one worm is alone present. It enters by the stomach and finds its way into the subcutaneous tissue, and thence head first to the region of the ankle. The head pierces the skin, and a blister forms, which bursts, leaving an ulcer, in the floor of which the head of the worm may be seen. Meanwhile the uterus has become filled with hundreds of developing embryos, and it discharges them, when ready, in a whitish fluid. These embryos are ·6 mm. in length, and they develop in the fresh-water cyclops. Once the uterus is empty, the worm can be withdrawn by winding it day by day on to a piece of wood. The worm cannot be removed at an earlier period unless the body of the parasite is injected with 1 in 1000 perchloride of mercury, and its fracture with, in consequence, a portion of the worm left *in situ* is apt to lead to serious inflammation. It is prudent, therefore, to await the rupture of the uterus before attempting the withdrawal of the parasite.

III. DISTOMIASIS

While there are a number of different varieties of the distomum, there are only three which demand reference here.

1. The **Distomum Hepaticum.**—Of this group there are several subvarieties met with in different countries, and the parasites vary considerably in size, being from 10 to 20 mm. in length, and 2 to 5 mm. in breadth. The ova are proportionately extremely large, often measuring 30 $\mu \times 15\ \mu$. The phenomena produced by the presence of the distomum vary considerably, but pain over the liver, with gradual enlargement of the organ, diarrhœa, and occasionally jaundice have been described, and

ascites may ensue. The ova may be readily recognised in the stool

2. The **Distomum Westermanii.**—This parasite attacks the lung and bronchial tubes, and causes a disease more common in the far East. The parasite induces attacks of haemoptysis varying in severity, with generally a considerable degree of bronchitis.

3. **Bilharzia Haematobia.**—The worm is one of the few members of the group which have the two sexes separate. The female is carried in a special canal inside the male worm. This parasite is specially common in Africa, in parts of India, and in Egypt, and affects the kidneys, ureter, bladder, and rectum. The adult worms are found in the veins of the kidney, although they may also be present in the portal circulation. We do not know how the parasite enters the body, but it has been thought that the embryos may be drunk with water or eaten adhering to water-cresses. The worms are first found in the portal veins, but eventually reach the veins in the neighbourhood of the kidney and bladder. The ova, about $\frac{1}{180}$ inch in length, have a peculiar sharp spine, which enables them to pierce their way through the tissues and to reach the bladder, pelvis, or ureter; to a less extent they also pass into the bowel and escape with the stools.

Clinical Features.—The presence of the adult worms produces no trouble, but the ova piercing through the urinary mucous membrane cause considerable haemorrhage and much irritation, and the ova in the bladder may become the nuclei of stone in the bladder. In the rectum they cause haemorrhage and often considerable tenesmus. An eosinophilic leucocytosis is generally present.

The **Diagnosis** consists in the recognition of the typical spined ova in the urine and faeces.

The **Treatment** consists in the administration of turpentine in fairly full doses, and sometimes the use of filix-mas, but neither remedy is very successful. It is impossible to do anything to destroy the adult worms in the veins.

DISEASES OF THE ALIMENTARY SYSTEM

I. DISEASES OF THE MOUTH AND TONGUE

(1) Stomatitis

There are many organisms found in the mouth, some of which enter with the food; others are associated with dental caries, and others with slight inflammation of the tonsils. The fur of the tongue consists largely of food and desquamated epithelial cells, but in addition there are many organisms present, and it is known that even the pneumococcus and diphtheria bacillus may exist in the mouths of perfectly healthy persons.

a) **Simple Stomatitis.**—A slight inflammation of portions of the mucous membrane, causing redness, heat, and dryness of the mouth and furring of the tongue. It may be due to the action of bacteria or to irritants of various kinds, and it is associated with certain of the eruptive fevers. In children it may cause a trivial constitutional disturbance. The affection is often associated with dyspepsia, the use of too hot tobacco, the ingestion of over-spiced food, and in children it commonly accompanies dentition. There may be a considerable amount of discomfort rather than marked pain.

The affection is rapidly cured when the cause is removed.

Treatment.—Remove the cause, give the patient cold or tepid articles of diet, and paint on the inflamed surface either a 3 or 4 grain to the ounce solution of silver nitrate, or the pharmacopœial solution of borax in glycerine. An alkaline mouth wash (5 per cent sodium bicarbonate with 5 per cent boracic acid in water) is often very serviceable, either alone or before other treatment.

b) **Follicular Stomatitis, Aphthous,** or **Vesicular**

Stomatitis.—Small raised vesicles, with a reddened area round, appear in varying numbers on the lips, tongue tip and edges, and gums; they rupture, and leave small and very painful ulcers.

Etiology. Usually indigestion is directly responsible for the follicular ulcers. They are common in gouty persons, and also in children under the age of three.

The **Clinical Features** are pain, especially on eating or drinking anything hot, and often marked salivation. The ulcers heal rapidly with treatment, and only in rare cases are they troublesome.

Treatment.—Careful attention should be paid to any evidence of indigestion, and very generally an alkaline purgative gives relief. In children a dirty feeding-bottle, or some similar cause, should be sought for. Treat the condition locally by painting on silver nitrate solution similar in strength to that mentioned above, or by touching the ulcers with a modified lunar caustic pencil. In any powerful application to the ulcers care should be taken to dry the affected part of the mouth beforehand. Various antiseptics, and especially chlorate of potash, borax and glycerine, or permanganate of potash, are often beneficial when used as a mouth wash.

c) **Ulcerative Stomatitis** or **Fetid Stomatitis.**—A more extensive ulceration than the last, beginning at the margin of the gums, with greyish-white ulcers, often causing loosening of the teeth and even necrosis of the alveolar margin of the jaw.

Etiology.—The disease occurs in badly nourished children, after the first dentition, and especially where there is a scorbutic element present. It may be due to dental decay, but the fact that it has been found in epidemic form suggests a specific organism, although no such organism has as yet been isolated.

Clinical Features.—There is pain on chewing, and the swollen gums bleed readily. There is copious salivation, and the breath is foul-smelling. Generally some pyrexia is associated. The condition may go on for a long time, even for months, and produce considerable cachexia. There is painful enlargement of the neighbouring lymphatic glands.

Prognosis.—The disease is rarely fatal, but some of the teeth and part of the jaw may be lost.

Treatment.—The fact that a specific organism is almost certainly present suggests the necessity of using strong antiseptics. The mouth may be washed out with saturated chlorate of potash solution, a solution of permanganate of potash, listerine or similar mouth wash, while the patient should have good diet and plenty of open air, and probably it would be well to add lime-juice in most cases.

d **Parasitic Stomatitis, Thrush,** or **Muguet.**—A condition due to the presence of a fungus called Oidium albicans although in many cases other fungi or bacterial agents are present. The oidium is one of the yeast type of fungi, and forms long branching filaments, each segment having a nuclear-looking body at each end. Whitish patches form on the tongue, and, on scraping, a raw tender surface is exposed which often bleeds. It is most common in infants, is generally the result of a dirty bottle or other want of cleanliness, and causes local heat and pain, loss of appetite, and very frequently diarrhœa. Occasionally the patches are situated on the œsophagus or even the stomach, but this is rare. In adults we associate the appearance of thrush with patients in the later stages of any wasting disease, and especially phthisis and it may occur in a continued fever. In such cases it causes much discomfort and greatly interferes with the digestive processes.

Treatment. The condition is very amenable to treatment. It is well to give an alkali, such as bicarbonate of potash with bismuth, and to treat the mouth locally with the pharmacopœial borax and glycerine, silver nitrate solution (10 grains to the ounce), or, in adults, the glycerinum acidi carbolici diluted in two parts of glycerine.

e **Cancrum Oris, Noma,** or **Gangrenous Stomatitis.**—A rapidly spreading gangrene, due to the presence of one or more specific organisms, and commencing generally near the junction of the cheek and gum. It is practically confined to young children between the ages of 2 and 5, and very commonly follows an attack of measles. In a few cases epidemics have occurred. It runs a rapid course, generally involving the jaw and causing intense fetor, salivation, and a varying amount of pain, while vomiting and diarrhœa frequently indicate absorption of bacterial poisons. The disease is rapidly fatal in most cases.

The **Treatment** consists in the application of strong antiseptics, such as fuming nitric acid, to the site of the gangrenous ulcer, especially when, as generally happens, it has opened through the cheek. Give iron in large doses and chlorate of potash, feed well, and order wine and other stimulants. Possibly patients in whom the disease proved fatal might have been saved if the treatment employed had been more radical at the outset.

(*f*) **Mercurial Stomatitis.**—Certain patients are specially susceptible to the smallest dose of mercury, and a peculiar idiosyncrasy to the drug is apt to develop in persons suffering from Bright's disease. Patients who are getting large doses of mercury, cinnabar miners, and those who work at trades in which mercury is much used (mirror-makers and thermometer-makers), frequently develop mercurial stomatitis.

Clinical Features.—The gums and tongue are red, raw, and swollen, the teeth loosen, and there is always profuse salivation with dreadful fetor, and sometimes necrosis of the jaw follows.

The condition is amenable to treatment, although it may cause much temporary suffering.

Treatment.—Stop the mercury at once and give iodide of potassium with a saline purge. Atropine has been recommended, but it is of greater importance to keep the mouth aseptic, and to give the patient good nourishing food, which, however, should be partaken of cold or lukewarm.

2. Glossitis, Inflammation of the Tongue

The tongue may be inflamed as the result of an irritant, and perhaps most commonly from an injury, a septic wound, or the sting of a wasp or bee.

The swelling may be so excessive as to prevent swallowing, and the glands below the jaw are secondarily involved. This condition may be very serious or very trivial; in most cases the treatment consists in endeavouring to allay the inflammation, while in certain instances incisions may be necessary so as to reduce the inflammatory oedema.

(*a*) **Ichthyosis Linguae,** or **Leucoplakia Buccalis.**—This consists of whitish pearly patches, which develop on the dorsum of the tongue and resemble corns in appearance. The

disease is almost certainly due to tobacco, possibly to syphilis, and it yields in most cases to suitable treatment, unless it is very chronic, when it may develop into an epithelioma.

Treatment.—Order the patient to take the food cold, and to stop all over-spiced or irritating articles of diet, and paint the patches with silver nitrate solution from time to time. Where the patch persists and there is any suspicion of malignancy, it should be at once removed by operation.

(*b*) **Tuberculosis of the Tongue.** A rare condition almost invariably associated with a late stage of pulmonary phthisis. The ulcer which eventually forms has whitish thickened edges and is difficult to distinguish from a gummatous ulcer, except by the fact that it does not yield to antisyphilitic treatment.

Little can be done by way of radical cure, but the ulcer should be scraped and the mouth washed out with an antiseptic solution.

(*c*) **Syphilitic Ulceration of the Tongue.** Many authorities consider that ichthyosis linguae is of syphilitic origin. Typical syphilitic ulcers, however, affect the sides and the dorsum of the tongue near the tip, and they have somewhat thickened edges and a whitish floor. They may produce marked scarring, and are often associated with gummata. A trivial syphilitic ulceration may be associated with early secondary syphilis.

Treatment.—Give antisyphilitic remedies, including both mercury and iodide of potash, while a mercurial lotion of 1 in 5000 corrosive sublimate should be used as a mouth wash.

(*d*) **Cancerous Ulceration of the Tongue.**—Almost invariably cancer of the tongue is a squamous epithelioma originating at the root of the tongue and causing gradual infiltration, and usually before a definite ulcer has formed, it has involved the neighbouring lymphatic glands.

The **Treatment** consists in the early removal of the tumour.

The tongue may also be involved by warts and fibrous tumours which usually cause little pain, but which should be treated surgically, as such growths may eventually become malignant in character.

3 The Tongue in Dyspepsia

In many cases of dyspepsia the flabby, swollen tongue, thickly coated with fur and often showing marked furrowings on the dorsum and edges, is an important sign of prolonged and persistent dyspepsia. The tongue usually cleans first at the tip and edges, and the appearance of the tongue, especially the condition of its covering, often indicates some gastric disturbance. In acute gastric catarrh the tongue is thickly furred, the fur being white in colour and moist, but the tip and edges are bright red.

4 The Tongue in Fever

When there is any diminution in the secretion of saliva, such as usually results from pyrexia the tongue becomes dry, and if it is furred, the fur is often of a brownish or yellowish colour.

In scarlet fever, where there is inflammation of the tongue as well as inflammation of the skin generally, that organ is swollen and is covered with a white fur, through which the swollen fungiform papillae show distinctly as bright red spots. At a later stage of the fever the fur is shed, and the tongue becomes bright red in colour all over, and often dry and glazed. In all febrile conditions, in which the patient lapses into what is described as the typhoid state, the tongue may either be covered with brownish fur, or may be red and shining.

Tremulousness of the tongue is a symptom of many diseases; it is common in the typhoid stage of fever, in delirium tremens, and in other serious conditions.

Paralysis of the tongue will be referred to under Diseases of the Cranial Nerves.

II. DISEASES OF THE SALIVARY GLANDS

1 Excessive Salivation or Ptyalism

This condition has been mentioned as a feature of many forms of stomatitis. An excessive flow of saliva follows the use of certain drugs, in particular mercury, tobacco, and pilocarpine.

Ptyalism is not uncommon in certain mental cases, in small-pox, and occasionally in pregnancy. The only treatment consists in an endeavour to remove the cause of the condition, where that is possible.

2 Xerostomia

Wherever there is an excessive drain either from the kidneys, bowels, or skin, there may be a diminution in the flow of saliva, but the disease called xerostomia is very rare: in it there is no saliva, the mucosa is red and shining, and bread chewed by the patient becomes a dry powder. No curative treatment appears to avail in such cases.

3) Inflammations of the Salivary Glands

Mumps or parotitis is an infective inflammation of these glands, and is described under infectious diseases p. 52. But it must be remembered that the salivary glands may swell as the result of neighbouring inflammations, and in certain continued fevers suppuration occurs in these glands without apparently, any very satisfactory reason, unless it be septic infection.

In injuries to the peritoneum, and in abdominal and pelvic lesions of different kinds, the parotid may become inflamed.

4 Salivary Calculi

These may form in the ducts of the glands, and usually consist of carbonate or phosphate of lime. A fistula or abscess may result.

5 Cysts and Tumours of the Glands

A ranula is a cystic swelling due to blockage of one of the gland ducts. Gaseous tumours have been caused in the ducts and glands as the result of certain trades, such as glass-blowing, playing on wind instruments, etc., but they are very rare.

Tumours of various kinds are sometimes present, chiefly of the parotid gland, and they may be fibrous, malignant, or cartilaginous. These affections belong more properly to the domain of surgery.

III AFFECTIONS OF THE PHARYNX AND TONSILS

It is remarkable how often the pharynx is inflamed while the tonsils, comparatively speaking, escape, and *vice versa*. The tonsils are situated, however, in the pharynx, and may be affected with the rest of the pharyngeal mucosa. It should be remembered that the tonsils are lymphoid glands, and as such their cells act as phagocytes and should destroy organisms. They may in certain cases either fail in this function and be themselves attacked, or else the phagocytic cells may ingest organisms which are too strong for them, and in this way infective organisms either attack the surface of the tonsils, or invade and set up inflammation in their substance.

1 Acute Pharyngitis, Simple Inflammatory Sore Throat

Catarrhal inflammation of the whole pharyngeal mucous membrane, sometimes including the tonsils.

Etiology. Cold and exposure are frequently the exciting causes, and especially in rheumatic or gouty patients.

Clinical Features. The typical features are: heat, dryness in the throat, soreness on swallowing, and hoarseness if the larynx is involved. It often begins with coryza, and may be accompanied by slight fever while aching in the back and limbs is very generally present. There may be a short and constant cough with the hawking up of a little mucus. The Eustachian tubes, if interfered with, give rise to deafness, and the neighbouring lymphatic glands are often swollen. A varying amount of malaise, largely dependent on the degree of pyrexia present, adds to the general discomfort of the patient.

On examining the pharynx it is seen to be reddened, the uvula often showing this very markedly, and there may be a little glairy mucus present in the later stages.

In a few days the condition subsides.

Treatment consists in giving salicylate of soda if a rheumatic case, quinine, guaiac resin, or Dover's powder. Quinine is admirable in fairly full doses (gr. 3 10), and a single dose of Dover's powder, following a hot bath or footbath frequently aborts a severe pharyngitis. Tincture of aconite is another favourite remedy in cases with much

feverishness and should be given in 1 or 2 minim doses every two hours. Inhalations of steam afford the greatest relief, either alone or impregnated with pine oil, creosote, or guaiacol.

(2) Septic Pharyngitis or Hospital Throat

An acute form of pharyngitis in which the tonsils almost always participate, and characterised by patches of membrane due to suppuration of the lymph follicles.

Etiology.—Decaying organic matter and defective drains are often responsible, but individuals exhausted by hard work are apt to be affected by organisms obtained from many sources, and organisms which in persons above par would probably do no damage.

Clinical Features.—The throat is intensely inflamed and very painful, and patches of whitish-grey membrane, which is not easily removed by scraping, are seen on the tonsils and pharynx. The neighbouring glands are swollen, and there is always pyrexia and marked malaise with pains in the back and legs. In suspicious cases examine a swab for the diphtheria bacillus. The patient is often very sharply ill, but with suitable treatment improvement is rapid.

Treatment.—Apply at once to the patches either the glycerinum acidi carbolici diluted with two parts of glycerine, or a 1 in 3000 solution of corrosive sublimate. Gargle the throat with permanganate of potash solution, a saturated solution of chlorate of potash, or boracic acid, or other suitable antiseptic. Feed well, order stimulants freely, and in severe cases give ice to suck.

(3) Chronic Pharyngitis

A more chronic inflammation affecting the naso-pharynx and posterior pharyngeal wall.

Etiology.—It is the result of cold and damp, of excessive voice-strain in hawkers, clergymen, etc., and arises from the immoderate use of certain irritants, such as tobacco and alcohol. Gout and, to a less degree, rheumatism are sometimes responsible.

Pathological Anatomy.—There are often dilated venules and a granular-looking posterior pharyngeal wall, due to the presence of small grey or red nodules of lymphoid tissue.

Clinical Features.– There are no constitutional phenomena, but there is a tickling cough with tenacious and limited expectoration, and, as a rule, a relaxed uvula, which tends to trail on the tongue. Several varieties of the condition occur which have been given separate titles, such as **Pharyngitis sicca**, with total want of secretion: **Follicular pharyngitis**, where the follicles specially suffer: and **Ulcerative pharyngitis**, which needs no definition.

The **Prognosis** depends on the possibility of resting the voice and the success of treatment.

Treatment.—Stop any causal condition present. Paint the throat with nitrate of silver solution (gr. 3-10 to the oz. of water or chloride of zinc (gr. 5-10 to the oz. of water). Ammonium chloride or chlorate of potash in tabloid form is excellent. Where there are enlarged follicles try to destroy them with the galvano-cautery or with strong silver nitrate solution. A warm, dry climate is advisable, and many spas are recommended as much for the rest and change as for any special benefit likely to be obtained from a particular mineral water, unless perhaps in rheumatic or gouty subjects. Painting with Mandl's solution, the various strengths of which are given below, is often successful in cases in which all else has failed.[1]

4) Phlegmonous Pharyngitis

A diffuse suppurative inflammation involving the deeper tissues, and generally spreading from some superficial focus. It may be due to the streptococcus of erysipelas, and it is a grave and fortunately rare sequel of scarlatinal pharyngitis. It is a typically septic condition, and it rapidly proves fatal in most cases, and especially where it spreads and involves the larynx.

The **Treatment** must consist in an attempt to combat the tendency to septicaemia. Free incision, where necessary, should be promptly carried out.

Mandl's solutions. Three strengths

℞

		(1)	(2)	(3)
Potassii iodidi	gr.	25	50	75
Iodi	gr.	6	12	20
Glycerini	℥	1	1	1
Ol. menthae piperitae	℥	5	5	5

5) Tubercular and Syphilitic Ulcerations of the Pharynx

Tubercular ulcers are rare, and, as in tubercular ulceration in the mouth, are generally associated with grave pulmonary phthisis. They are often very painful and may render swallowing difficult. **Syphilitic** ulcers may be secondary, and if so, are shallow, or they may be tertiary and deep-seated. Syphilitic ulcers are rarely painful.

6 Acute Tonsillitis or Quinsy

An inflammation of one or both tonsils with occasional suppuration.

Etiology.—It is due to cold and wet, but it is probable that in many cases organisms emanating from decaying matter, perhaps, especially from drains, may enter the crypts of the tonsils and there set up acute inflammation. It bears a very close relationship to rheumatic fever, and the tonsils are apt to be involved in scarlet fever, diphtheria, and other infective fevers. There is a distinct predisposition to the affection in certain families and individuals, and persistent enlargement of the tonsils, often associated with the presence of adenoids, appears to predispose to acute attacks of tonsillitis.

Pathological Anatomy.—The tonsillar crypts become filled with a caseous material containing micro-organisms. Where an abscess forms it may be tonsillar or peritonsillar in position, and generally points towards the mouth.

Clinical Features.—The onset of tonsillitis is sudden and is generally associated with a rigor, with aching in the back and legs, and local pain on swallowing, which shoots up to the ear. The breath is bad, the tongue foul and furred, and on examination the tonsils, one or both, are seen to be red, lobulated, and swollen. They may touch in the middle line the uvula being in contact with both tonsils. The neighbouring lymphatic glands are often swollen: there is frequently salivation, and almost always a degree of malaise. The temperature varies from 102 to 103 F., and is generally more marked in cases which go on to suppuration and in septic pharyngitis, in which the tonsils specially suffer. There

is not infrequently formation of a membrane or superficial ulceration, especially where the tonsils come in contact, and the voice has a distinctive muffled or nasal tone, while in many cases the tonsillar inflammation interferes with the orifice of the Eustachian tube, causing deafness on the affected side. These phenomena become much more marked when suppuration has occurred. The recognition of an abscess may be made by digital examination, the finger to be introduced into the mouth being passed along and in contact with the lower teeth. Very rarely does the abscess open into an artery, and only in exceptional cases does the pus infiltrate the tissues of the neck.

Diagnosis.—*Diphtheria*, with which the disease may be confused, has a distinct membrane which may be absent in tonsillitis, and in the membrane the presence of the diphtheria bacilli should be easily demonstrated, while albuminuria is common in cases of diphtheria.

Prognosis.—Generally rapid recovery results, but with a tendency in certain persons to recurrence during cold damp weather. Only in very rare cases does a tonsillar abscess give rise to serious trouble, and then it is usually due to neglecting early incision.

Treatment.—Give ice or iced barley water, and paint the inflamed tonsils with a 10 to 20 per cent solution of cocaine, or with carbolic acid and glycerine; steaming and poulticing the throat is useful where an abscess is threatening. Invariably give a purge, and by preference salts or compound jalap powder (gr. 20-30); at an early stage quinine in 3 to 4 grain doses, guaiac resin (3 grains in lozenge form), and tincture of aconite (♏ 2-3) are often of great value.

When suppuration has occurred it is better not to delay incising the tonsil. After painting with cocaine, or giving the patient ice to suck, take a Syme's abscess knife, the edge of which should be guarded with sticking plaster up to within about ½ an inch from the point, and make a vertical incision, remembering the position of the internal carotid artery. Hæmorrhage after the operation is rare, and ice will usually check it if excessive. After most cases of tonsillitis the tonsils should be painted with Mandl's solution

In rheumatic patients give salicylate of soda or other agent of the same group.

(7) Chronic Tonsillitis

Chronic enlargement of the tonsils, not infrequently associated with hypertrophy of the adenoid tissue in the naso-pharynx.

Etiology.—It is very common in children, especially if they are unhealthy: it sometimes follows an acute attack, or it may be chronic from the outset. It is a sequel to scarlet fever, measles, whooping cough and diphtheria, and it is associated with the status lymphaticus in which there is an increase of all lymphoid tissue throughout the body.

Pathological Anatomy.—There is thickening of the fibrous stroma of the tonsil, and often increase of lymphoid tissue. The tonsils remain permanently swollen, are palpable externally, and may interfere very seriously with the orifices of the Eustachian tubes, thus causing deafness.

As a result of the condition in childhood the chest is badly developed and narrow, and generally the patient's mental growth appears to be seriously impeded, if not checked. In not a few cases of this disease it is found that the enlarged tonsils are either tubercular or are associated with tubercular glands, and there is a distinct tendency to the development of bronchial catarrh.

Clinical Features.—The visible and palpable enlargement of the tonsils, associated often, as already stated, with adenoids in the naso-pharynx, causes loud mouth-breathing with snoring at night, a peculiar change in the voice, deafness, and a special liability to subacute attacks of tonsillitis and to colds in the head. The child constantly keeps its mouth open, and may have more or less cough, while nocturnal paroxysms of dyspnœa sometimes occur. The existence of mental backwardness has already been noted. When a patient reaches the age of thirty, the tonsils often cease to give trouble. Refer also to description of adenoids, p. 426.

Prognosis.—The tonsils should be removed, even although there is the probability of their subsidence in adult life, and, in most cases, if this is thoroughly done, physical and mental development, together with freedom from attacks of tonsillitis and coryza, may be assured.

Treatment.—Remove the tonsils either with an ordinary bistoury or with the guillotine, and apply astringents or ice

to arrest the haemorrhage if excessive. Give in all cases in children cod-liver oil, iron, and other tonics; it is also wise after operation to use an antiseptic gargle for some days. Adenoids in the naso-pharynx should also be operated on if they interfere with breathing.

IV. AFFECTIONS ASSOCIATED WITH THE ERUPTION OF THE TEETH

TEETHING is likely to cause trouble during the *first* dentition, and especially in delicate children who are rickety or tubercular.

The chief **Clinical Features** are swelling and tenderness of the gums, stomatitis, gastric catarrh and diarrhoea, and very frequently eclamptic seizures or convulsions. Prolonged ill-health may result, the child becoming fractious and sickly. Improvement commences once teething is over.

With the *second* dentition there is usually no trouble, but should the patient be delicate, or the eruption of the teeth be accompanied by much local pain and swelling, nervous phenomena may develop, and especially reflex cough, irritability of temper and asthma, while sometimes gastro-intestinal disturbances are present.

Treatment.—In the infant it is usually only necessary to pay attention to the hygiene of the mouth and to the condition of the stomach and bowels, but if convulsions have occurred, the gums should be lanced.

For trouble during the second dentition similar treatment may be adopted.

V. DISEASES OF THE OESOPHAGUS

(1) ACUTE OESOPHAGITIS

AN acute catarrhal inflammation of the oesophagus.

Etiology.—It may be the result of (1) injury; (2) the extension of a catarrhal pharyngitis, and especially in certain continued fevers, such as diphtheria; (3) irritation due to the presence of a new growth or a foreign body; and (4)

the swallowing of a corrosive substance. Any focus of irritation in the neighbourhood of the œsophagus may set up œsophageal catarrh.

Clinical Features.—There is pain on swallowing, which may become so excessive as to cause complete inability to take food, and there may be spasm with regurgitation of what has been swallowed. The pain is marked along the line of the œsophagus, and is felt in the neck, under the sternum, and to a less extent in the back. In diphtheritic cases the presence of a membrane adds to the obstruction.

Diagnosis and **Prognosis.**—The diagnosis depends largely on the recognition of the nature and cause of the inflammation present, and the prognosis on the possibility of successful treatment.

Treatment.—Sucking ice and the administration of soothing drinks are most important, while rest to the inflamed surface may be obtained by feeding by the bowel, and limiting as far as possible the swallowing of saliva by the use of sedatives.

(2) Stricture of the Œsophagus

A. Spasmodic Stricture.—Spasmodic contraction of the muscular fibres of the œsophagus.

Etiology.—It is common in hysteria, and is associated with the neurotic habit, and sometimes with gout. It is one of the symptoms of hydrophobia.

Clinical Features.—There is inability to swallow solid or sometimes even fluid food. There may be pain and generally regurgitation of food. Great relief is experienced by the passage of a bougie, which, although stopped by the spasm, is eventually passed with ease, and produces a remarkable effect, perhaps partly mental, in alleviating the condition.

The **Diagnosis** is directed towards the discovery of any possible organic cause for the condition, and it must be remembered that with an organic stricture there is a great element of spasm superadded. Malignant stricture and aneurism should both be thought of.

Prognosis.—Recovery generally follows treatment.

Treatment. The occasional passage of a bougie with general tonic treatment is usually sufficient. Any constitutional element in the case, such as gout, should be treated.

B. Simple Stricture. A chronic thickening of connective tissue, causing narrowing of the oesophagus.

Etiology.—A rare condition, unless as the result of swallowing an irritant or corrosive fluid, in which case it follows from the process of healing or cicatrisation. Alcohol is said very occasionally to produce this condition. It should be remembered that aneurisms, enlargement of the thyroid, pericardial effusions, glandular swellings, and tumours of all kinds, may cause pressure on the oesophagus.

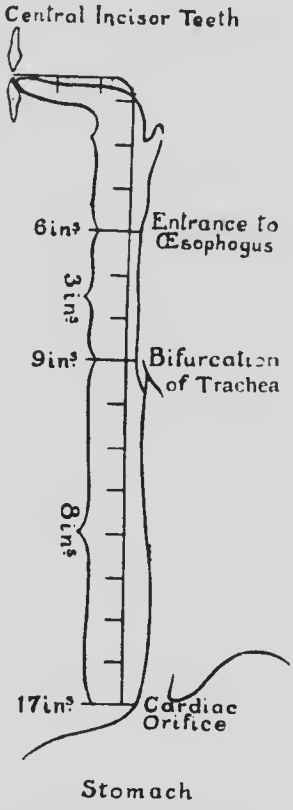

FIG. 22.—Shows length of Oesophagus in the adult, and indicates relationship to larynx (it begins at cricoid cartilage) and bifurcation of trachea

Clinical Features.—There is increased difficulty in swallowing with an element of spasm, the degree of which depends on the amount of obstruction. There may be regurgitation of food, and very frequently semi-fluid or fluid food passes down the gullet more easily than solids.

The **Diagnosis** and **Prognosis** depend on the recognition of the cause of the constriction and the possibility of doing something to relieve it.

The **Treatment**, in cases where there is actual stenosis due to cicatrisation, consists in an attempt at dilating the oesophageal stricture, while the element of spasm may be combated by such sedatives as dilute hydrocyanic acid (m 3). Very occasionally surgical interference may relieve pressure from tumours, but it depends entirely on the nature and site of the tumour.

C. Malignant Stricture.—The commonest form of tumour is the squamous epithelioma, although much difference of opinion exists as to where the tumours are generally situated, and each third of the oesophagus has been favoured by different authorities as the commonest position for such a tumour. Probably the upper and the lower thirds are more

frequently affected than the middle third, and malignant stricture is very common just at the bifurcation of the trachea. The growth may spread in an annular fashion round the oesophagus; it may infiltrate the walls, and extend upwards or downwards for a distance of 2 or 3 inches. The results of such a tumour may include the actual narrowing of the lumen of the gullet, and perforation through the wall of the oesophagus, often into the left bronchus, while infiltration of the muscle of the oesophageal wall interferes with normal peristaltic movement. Added to these, the presence of a tumour gives rise to spasm when food reaches the stricture, and dilatation of the oesophagus may occur above the stricture, due to food tending to accumulate; such a dilatation may empty itself reflexly from time to time. Perforation into the bronchi or trachea must inevitably cause septic broncho-pneumonia, while perforation into the pleura or elsewhere usually leads to serious and often fatal sequelae. Secondary deposits are common, involving the glands and other neighbouring structures, and thus death may be brought about, not merely by the starvation of the patient from blocking the oesophagus, but in many other ways.

The chief **Clinical Features** have been already indicated—emaciation and dysphagia, with frequently regurgitation of food while often the attempt at swallowing produces excessive pain. Generally the food regurgitated is found to be alkaline, indicating that it has not entered the stomach.[1] The patients in whom one finds the disease are usually past middle life.

Evidences of the septic broncho-pneumonia, empyema, and other results of perforation may follow as the disease advances.

Diagnosis.—The diagnosis depends partly on the age of the patient, partly on the information obtained by the passage of a bougie, and partly on any other evidence which may be obtainable of the existence of a tumour, and of the sequelae to which it may have given rise. For example, there may be portions of the tumour in the regurgitated matter, and a limited stricture, not due to aneurism or the pressure of a tumour from without, renders the diagnosis of malignant stricture almost certain. A small olive-headed probang, small enough to pass through the stricture once the initial spasm is re-

[1] This statement is not entirely accurate, because food in the stomach is at times alkaline for an appreciable length of time.

heved, will, when the head is being withdrawn, give rise to spasm again when it comes in contact with the lower end of the stricture, and by comparing the distances as indicated on the scale marked on the stem of the instrument, a definite idea will be obtained of the vertical measurement of the stricture. Such information may strongly point in favour of malignant stricture. Further, blood is very likely to be present in the regurgitated matter in a case of malignant stricture, although it may also occur in aneurism.

The utmost caution should be practised before passing a tube or probang, and aneurism must be excluded, because rupture might be induced and the death of the patient hastened.

Prognosis.—It is extremely unlikely that a malignant stricture can be cured. Whatever the nature of the tumour may be, if it is malignant, it is a question rather of prolonging for a time the patient's life than of attempting any definite cure by operation or other means. Gastrostomy may add months to the patient's lifetime, and sometimes the passage of a bougie through the stricture, or the temporary introduction into the stricture of a vulcanite tube, may cause dilatation of the lumen, and so permit of the passage of food with greater ease.

Treatment.—As already indicated, something may be done by the passage of a bougie, or by the introduction of a tube, and, where these fail, by performing gastrostomy. The treatment otherwise should consist largely in keeping up the patient's nutrition, and, when pain is present, administering sedatives and antispasmodics such as ice, dilute hydrocyanic acid, and morphia.

3. Dilatation and Diverticula

A **Dilatation** is frequently secondary to and above the level of a stricture, and it may involve the whole of the wall of the œsophagus.

Diverticula are either (1) **Pressure diverticula** or localised bulgings which may possibly be due to imperfect development, but are more likely to be the result of pressure from within, such as might be produced by a foreign body. The diverticulum is generally formed at the junction of the

pharynx and oesophagus; it projects *behind* the gullet, and may bulge on both sides of the neck. It may be palpable when filled with food, and may produce dysphagia from the pressure of the pouch on the oesophagus. (2) **Traction diverticula** occasionally develop. They are due to adhesion with glands often at the tracheal bifurcation. The adhesions cause traction on the *anterior* oesophageal wall. The condition is amenable to operation, and surgical text-books should be consulted with reference to the procedure.

4. Paralysis of the Oesophageal Muscles

This may occur in diphtheria and bulbar paralysis. It necessitates feeding the patient through an oesophageal tube, and the treatment, in diphtheritic cases at least, consists in electrical applications, the use of strychnine, etc.

5. Rupture of the Oesophagus

A rare condition, most frequently found after death and due to post-mortem digestion. During life it may result from a tumour, or from the effect of a powerful corrosive on the oesophageal wall; but very exceptional cases of rupture as the result of strain have also been recorded.

VI. AFFECTIONS OF DIGESTION

1. Normal Salivary and Peptic Digestion

The ptyalin of the saliva acts in an alkaline medium, and salivary digestion continues in the stomach for about half an hour after ingestion of a meal. By its action starch is converted into maltose and cane-sugar into glucose. Organic acids may be formed to some extent during salivary digestion. After about thirty minutes have elapsed, ptyalin digestion ceases, and hydrochloric acid and pepsin take its place. The gastric juice converts proteids into albumoses, and later the deutero-albumoses are converted into peptones; it curdles milk rennin, and it acts in virtue of its free hydrochloric acid as an antiseptic.

Free hydrochloric acid should normally reach a percentage of ·2 per cent. It is true that organic acids can take the place of hydrochloric acid in the process of digestion with pepsin, but they do so in a very imperfect manner. Lactic acid is normally largely formed from butcher meat and bread, but in cases of stagnation of stomach contents with diminished hydrochloric acid, the lactic acid is formed by the action of the Oppler-Boas bacillus which is present in great numbers. The flow of gastric juice is stimulated by the saliva. The peristaltic movements of the stomach greatly aid digestion. No change is effected by peptic digestion on carbohydrates or fats, but some of the products of salivary digestion may be absorbed by the stomach, such as maltose and grape-sugar, while salts and peptones are also taken up, the latter only to a very limited extent. Water is not absorbed by the stomach, while alcohol on the other hand is readily absorbed. Peptic digestion lasts for 4 to 6 hours, when the chyme passes onwards into the duodenum, but the time depends on the size and ingredients of the meal eaten.

Carbohydrates may begin to leave the stomach in 10 to 15 minutes, while proteids and fats are delayed much longer.

When the acid chyme reaches the duodenum it stimulates the production of secretin, which in turn stimulates the flow of pancreatic juice. Fats can probably also stimulate pancreatic secretion. The pancreatic juice acts only in an alkaline medium and it contains the following active agents: (1) Trypsinogen which is converted into trypsin by coming in contact with the enterokinase of the intestinal juice. Trypsin changes proteins into deutero-albumins and later into peptones, and part of the peptones become amido-acids and organic nitrogenous bases, part remaining as amphi-peptones; (2) Amylopsin which converts carbohydrates much in the same way as does ptyalin; and (3) Steapsin which hydrolises fats.

Bile aids steapsin in emulsifying fats. It dissolves lecithin and cholesterin, and so aids in their elimination from the body, and it has a definite purgative effect on the bowel.

The intestinal juice contains the all-important entero-kinase which converts trypsinogen into trypsin.

(2) Indigestion

There are many causes of indigestion, but the following tabulated list is suggestive:

1. Huge meals.

2. Badly masticated food, whether from the habit of bolting food or from bad teeth.

3. Excessive dilution of the stomach contents, as for instance by drinking too much water, thereby rendering the gastric juice less efficacious.

4. Exercise too soon after meals.

5. Unsuitable food, which may be due to *a* a badly proportioned dietary, whether the excess be of carbohydrates or nitrogenous ingredients, *b)* bad cooking, *c)* certain indigestible articles of diet, such as crabs and lobsters, which have very long muscle fibres, and *d)* excessive quantities of tea, etc.

6. Insufficient time between meals, especially if these meals are large.

7. Interference with stomach movements owing to tight lacing.

8. Excitement and emotion which may interfere with the process of digestion.

9. Existing pathological conditions constitute a numerous list. Bacteria in the mouth or about the teeth may interfere with both salivary and peptic digestion, anaemia and debility after any disease often predispose to indigestion, while local stomach affections, such as gastric ulcer, cancer, and gastrectasis or dilatation, are almost invariably associated with it.

10. Over-stimulation of the stomach by an excessive use of pepper, spices, pickles, and alcohol; closely associated as regards its detrimental effect is the consumption of undiluted spirits between meals.

VII. DISEASES OF THE STOMACH

(1) Acute Gastric Catarrh; Acute Catarrhal Gastritis; Acute Dyspepsia

An acute catarrhal inflammation of the mucous membrane of the stomach.

Etiology. — 1) The ingestion of irritant, toxic, or decom-

posing substances into the stomach. This group includes, in addition to corrosive poisons, alcohol in excess.

(2) Unsuitable food or articles of diet, which although digestible in moderate quantities, in excessive amounts may give rise to gastric irritation. A good example of this is carbohydrates in excess, and especially sugar, or vegetable fibre, such as one ingests in eating salad, cabbage, and many other green vegetables. Certain muscular fibres, such as those in the flesh of crabs, lobsters, cockles, etc., being of great length, are extremely difficult to digest, and in excess may overtax the stomach.

(3) In the subjects of rheumatism or gout, and in many persons who are said to have an "irritable stomach," there is a predisposition to acute gastric catarrh, and any indigestible meal may precipitate an attack.

(4) In many fevers with a cutaneous eruption, such as scarlet fever and measles, the gastric mucosa may to a certain extent participate with the skin in the inflammation.

(5) In chronic Bright's disease there is frequently gastric catarrh, due doubtless to the presence of some toxic agent.

(6) In any organic disease of the stomach, whether simple ulcer, cancer, or tuberculosis, there may be acute gastric catarrh associated with the condition.

Pathological Anatomy.—The mucous membrane is reddened, there is increased mucous secretion, and a diminished flow of gastric juice. From Beaumont's observations on the stomach of Alexis St. Martin, it is certain that there are also small erosions and haemorrhages. It is probable that the glandular epithelium, in place of secreting gastric juice, undergoes mucinoid degeneration. Should death occur, it is usual to find pigmentation present, possibly due to altered blood. There may be a definite change in the muscular coat, either atonic or atrophic, while in the neighbourhood of the pylorus the mucosa may acquire a mammillated appearance, which is due to a small-cell infiltration around the orifices of the glands and interfering seriously with their function. There is also great interference with the normal blood-supply to the stomach during digestion, and this fact, together with the structural alteration in the mucosa, will explain the diminution of both hydrochloric acid and pepsin, the former being generally more diminished in amount than the latter, although

both may be completely absent in a very acute case. As a result, fermentative changes rapidly take place in the stomach contents, and organic acids are formed in great excess, adding much to the discomfort of the sufferer.

Clinical Features.—A very typical attack of gastric catarrh follows either an imprudent meal or a bout of drinking. There is generally malaise with epigastric discomfort which may amount to severe pain, nausea, and vomiting. The tongue is thickly furred, there is loss of appetite, great thirst, and often a limited degree of pyrexia. The saliva may be increased in certain cases. The pulse is rapid, but soft, and as a part of the malaise already described, there are generally aching pains in the joints. Headache is a common symptom.

In many cases of acute gastric catarrh in children, the irritation extends downwards and causes diarrhoea, sometimes with colicky pain, but in adults constipation is the rule. The involvement of the duodenal mucosa, causing interference with the ductus choledochus, implies that a degree of catarrhal jaundice will follow the gastric catarrh in one or two days' time.

In many cases the condition might more properly be termed subacute than acute.

Diagnosis.—While the typical phenomena already described indicate an acute gastric catarrh, it is wise to ascertain whether some other condition may not also be present and *typhoid fever* should be thought of and excluded by the Widal reaction, where there is any doubt as to the diagnosis.

Prognosis. Generally in a few days the condition yields to treatment, but if too many attacks occur, a persistent chronic gastric catarrh will develop.

Treatment.—1. We should strive, in the first place, to get rid of any irritant present, such as unripe fruit, decomposing meat, etc. For this purpose an emetic, especially in the case of children, is of great value. Sometimes the stomach must be washed out either with a dilute alkali, such as sodium bicarbonate, or with a weak solution of permanganate of potash, this treatment being followed with a saline purge or a dose of Gregory's mixture. Should we suspect the presence of organisms, the internal administration of 10 to 15 grains of sodium hyposulphite is advisable.

2. We must next endeavour to soothe the stomach, and in

certain cases this should be done by giving it complete rest and feeding by the bowel. Hydrocyanic acid (♏3), bismuth subnitrate (gr. 10-20), oxalate of cerium (gr. 1-5), will be found of value in soothing pain, while opium and the use of ice, both internally and externally, are sometimes of advantage. It should be remembered that to certain patients the application of heat over the stomach is more helpful than the ice-bag.

3. Try to aid the stomach to return to its normal functions by giving predigested food, and later hydrochloric acid and pepsin in proper proportions, once the irritation of the mucosa has disappeared. Milk and soda-water (sometimes iced) is the best dietary.

(2) Acute Toxic Gastritis

A. This is a severe degree of the condition just described, and may be due to the swallowing of a corrosive poison. The inflammation set up is extremely acute, the vomited matter invariably contains blood, and from an examination of it an idea may be obtained of the substance swallowed. The patient is usually in a state of collapse, and in many cases there is in addition marked inflammation of the kidneys.

Most of these cases rapidly prove fatal unless promptly relieved, and may eventually give trouble even when some antidote has been promptly administered, partly as the result of necrotic changes in the œsophagus and stomach, and partly as the result of the tendency to cicatrisation causing stricture.

Sometimes emetics can be given, and if a suitable antidote is obtainable the patient's life may be saved.

B. Another form of toxic gastritis is due to eating decomposing meat. This also causes a severe type of acute gastric catarrh, and may produce most intense inflammation. A suppurative gastritis may ensue, dependent upon the nature of the organisms present, and the inflammation may spread to the intestine. Dreadful pain, vomiting, and diarrhœa, with early collapse, will follow, and death is only a matter of hours.

Even after a comparatively mild case, or where a very small amount of the decomposing food has been eaten, long protracted ill-health may result notwithstanding the most skilful treatment.

(3) Phlegmonous Gastritis

In certain fevers, and especially in septicaemia and pyaemia, there may be a widespread suppurative inflammation involving the wall of the stomach, or one or more abscesses may form as the result of the presence of simple or malignant ulcers. In acute toxic gastritis there may also be suppurative inflammation in the stomach wall.

Dreadful pain, vomiting, and generally diarrhoea rapidly terminate a case which is absolutely incurable. Very rarely does an abscess, by discharging into the stomach or some other viscera, lead to a favourable result, although such cases have been recorded.

(4) Membranous Gastritis

In diphtheria, and exceptionally in pyaemia, small-pox, typhoid, pneumonia, Bright's disease and many other conditions a membrane may form. A membrane is not uncommon in cases of toxic gastritis. The clinical features closely correspond to those referred to above under phlegmonous gastritis.

(5) Parasitic Gastritis

Thrush and favus have rarely been found growing in the stomach. Yeast fungi are more common. Occasionally the larvae of flies have been introduced into the stomach.

6. Chronic Gastritis

This is chronic gastric irritation, and is dependent on various conditions.

Etiology. — Acute gastric catarrh may give place to chronic irritation of the stomach, and many of the causes mentioned already under the heading of acute gastric catarrh may induce a lesser but protracted degree of inflammation, and so bring about chronic gastric catarrh.

The causes of indigestion detailed on page 228 may also lead to organic change in the mucosa of the stomach, and as the result of backward pressure from the heart or lung there may be engorgement of the portal vein, and with it its gastric tributaries.

Pathological Anatomy.—The mucous membrane has a brown or greyish colour owing to the deposit of iron-containing pigment in the cells and tissues. The mucosa is covered with a layer of thick tenacious mucus. In not a few cases there is a proliferation of the interglandular tissues or of the glandular elements, in both cases giving rise to a mamillated appearance of the mucosa with much thickening of the stomach wall. There may be considerable enlargement of the stomach, but in a small proportion of cases there is great diminution in size—the so-called sclerotic or atrophic form. In the latter type the increase of interstitial tissue produces great contraction of the wall of the organ with diminution in its capacity.

The **Clinical Features** include pain or gastric discomfort varying in degree, flatulence, occasional vomiting, and all of these are more marked when certain kinds of food have been ingested. The tongue is furred and the breath foul. There is a bad taste in the mouth and the appetite is poor or capricious. Morning vomiting, mainly of glairy mucus, is common, and as the disease progresses nausea becomes more and more persistent. Still later there is evidence of fermentation with acid eructations, water-brash, and gastralgia. The gastric contents contain much mucus, and the gastric juice is deficient in free hydrochloric acid. There is tenderness on palpation over the epigastric region, constipation is marked, and as time goes on the general nutrition of the patient suffers severely.

In connection with the nervous system the patient complains of headache, sleeplessness, depression, and incapacity for mental work.

The **Diagnosis** between *gastric cancer* and chronic gastritis depends on the history of the case. Haematemesis is absent in chronic gastritis, while it is only in long-standing cases of that disease that one finds the absence of free hydrochloric acid so typical of malignant disease.

The **Prognosis** depends on the possibility of removing the cause. In long-standing and severe cases one cannot hope for a complete cure. Digestion can be carried out in the intestine, and therefore any stagnation in the stomach, or in other words motor insufficiency, must be reckoned as a sign that medical must give place to surgical treatment.

The **Treatment** consists in the removal of any known and removable cause, and failing that gastro-enterostomy should be

performed. The diet must be carefully selected. It should consist chiefly of milk, soups, white fish, and only a moderate amount of carbohydrates, and it is often found advisable to administer the food in small quantities frequently. The stomach should be washed out if there is much mucus, and it is often advantageous towards the end of this operation to introduce water of colder temperature. Hydrochloric acid and pepsin are frequently necessary, and for fermentation creosote in capsules (m 2-3) will be found useful.

7 Dyspepsia

Acute dyspepsia so closely corresponds to acute gastric catarrh that no special description is required. Chronic dyspepsia is certainly often due to chronic gastritis, but there are many cases which do not belong to this category, although in time the pathological condition of the stomach known as chronic gastritis may result. Some of the clinical features of chronic dyspepsia have been included under gastric neuroses.

(8). Gastric Neuroses

A. Gastralgia or Gastric Pain.—A complete **Etiology** would include almost every gastric disease, but purely functional gastric pain is generally associated with hysteria or neurasthenia. In women it frequently occurs at the menstrual epoch or during the menopause. In men it is more common amongst Jews. It is often paroxysmal. It may be relieved by the ingestion of food or by vomiting, or it may come on at any hour of the night or day and quite apart from any meal. It is often burning, lancinating or boring in character, referred to the epigastrium but shooting through to the back and specially round the left side to the angle of the scapula. It may last for minutes or for several hours. Palpation demonstrates the existence of diffuse tenderness and not the single painful point so significant of gastric ulcer, but the gastralgia is sometimes relieved by pressure over the stomach. In a bad attack the face is pale, the pulse rapid, and the extremities cold.

The **Treatment** depends somewhat on the condition of the gastric contents, but bicarbonate of soda gr. 20-30 with

menthol (gr. $\frac{1}{2}$ - $1\frac{1}{2}$) often at once relieves the patient. Morphia is occasionally necessary, and belladonna, arsenic and silver nitrate have all been recommended.

B. Gastric Hyperaesthesia is not uncommon in neurasthenia. It is associated with eating, and may merely consist of a sense of fulness without much actual discomfort.

C. Hyperchlorhydria is a gastric juice containing an excess of free HCl (over the usual ·2 per cent) at the high tide of digestion. It is one of the common signs of both simple gastric ulcer and also of duodenal ulcer, but it may be present apart from these conditions.

It causes a burning pain which comes on from one to two hours after a meal, a pain often so severe as to make the patient voluntarily empty his stomach. The pain is usually relieved by taking more food, especially proteids, and it ceases at once on taking an alkali such as 20 to 40 grains of sodium bicarbonate. There may be acid eructations and water-brash, but there is not the flatulent distension associated with the presence of organic acids. The tongue is clean and does not suggest any gastric disturbance. The condition generally occurs in young persons, and the surest diagnostic test is the examination of the stomach contents.

The **Treatment** consists in lessening the carbohydrates and replacing with proteid food. 20 to 40 grains of bicarbonate of soda administered with or without the addition of menthol will usually at once relieve the discomfort. Cream has been recommended because it lessens the flow of hydrochloric acid, and for a similar reason oil or oily substances may be given. Our favourite treatment consists in directing the patient to take 10 minims of dilute hydrochloric acid immediately before food, and this method has one advantage in that its efficacy can be tested in one or two days' time.

D. Hypersecretion of Gastric Juice is closely allied to hyperchlorhydria, although there is no marked relative increase in the free hydrochloric acid. There are two forms—1) the *intermittent type*, which occurs in neurasthenic subjects, comes on without any relationship to food, and the patient soon vomits large quantities of sour stomach contents. The attack

lasts from 24 to 48 hours, and is often associated with severe headache, depression, and coldness of the extremities.

(2) The *continuous type*, sometimes called Reichmann's disease, is often a sequel to the last and begins much like hyperchlorhydria. The pain increases as digestion goes on and the stomach becomes greatly distended until the patient vomits large quantities of sour stomach contents, but with no undigested food and practically no organic acids.

The **Treatment** corresponds closely to the treatment for hyperchlorhydria.

E. Hypochlorhydria or Diminution of Free Hydrochloric Acid. – We know hydrochloric acid is usually absent in cancer of the stomach, in advanced cases of chronic gastritis and also in any atonic condition of the stomach. The symptoms are those of severe indigestion with much praecordial distress and fermentation of food, the stomach contents containing a varying amount of organic acids.

The **Treatment** consists in administering pepsin and hydrochloric acid, and a course of strychnine is very beneficial.

F. Peristaltic Unrest.—This occurs not infrequently in patients who either suffer from great motor activity or great motor weakness of the stomach. In other cases borborygmi or rumbling sounds are produced in the stomach when the patient has been perturbed by some mental or other cause which starts the peristalsis.

G. Pyloric Insufficiency.—In this case the contents of the stomach pass on at once into the duodenum, and bile tends to regurgitate into the stomach. The diagnosis is readily made by proving that the stomach is empty half an hour after a meal has been taken. The remedy which is perhaps more efficacious than any other is strychnine.

H. Pyloric Spasm is often due to hyperchlorhydria or hypersecretion, and it is also common in gastric ulcer and in cases where the stomach is unduly irritable as in acute gastric catarrh or where the stomach contents are of irritating nature. It is probably the factor which produces much of the pain in the first two conditions mentioned. Dietetic measures, gastric sedatives, and alkalis are often helpful in relieving the spasm.

I. Gastric Atony is present in slight degree in practically all cases of general debility, and it may follow chronic gastric catarrh. There is considerable praecordial distress sometimes even amounting to gastric pain. Flatulence is common, and as we should expect there is gastric dilatation. The stomach contents are poor in hydrochloric acid, and a test meal followed by the examination of the contents renders the diagnosis easy. The **Treatment** consists in careful dieting, massage to the abdomen, and strychnine.

J. Nervous Vomiting is the rejection of the stomach contents without sickness, much in the way an infant vomits. It is present in hysteria and neurasthenia, and is apt to be mistaken for cerebral vomiting. No treatment is so satisfactory as isolation on the principle of the Weir-Mitchell method.

Anorexia Nervosa is the condition in which there is absence of the usual sensation of hunger. **Bulimia** is the term applied to a voracious appetite, and **Pica** is the name given to the craving for indigestible things peculiar to certain patients.

9) Haematemesis; Gastrorrhagia

Vomiting of blood from the stomach.

Etiology. Ulcer of the stomach, simple or malignant, miliary aneurism, varicosity of the veins of the stomach, and acute congestion of the stomach wall, may all cause a varying amount of haemorrhage. Simple ulcer of the stomach may lead to very serious loss of blood owing to erosion of a considerable artery, and an abdominal aneurism may rupture through the stomach wall, death being due to the haemorrhage.

In certain toxic conditions gastric haemorrhage is common, as for example, in some of the continued fevers, especially yellow fever, and what may be termed the haemorrhagic type of small-pox, typhus, scarlet fever, measles, etc.

Corrosive poisons cause severe gastric haemorrhage, and it may result also from injury, such as might be produced by the careless use of the stomach-pump.

In some blood conditions and constitutional diseases, such as leucocythaemia, pernicious anaemia, haemophilia, scurvy, and other allied affections, we may find haematemesis.

It may occur also from backward pressure from the portal system, as in common cirrhosis of the liver.

Blood coming from the nose, mouth, or lung may be swallowed and then vomited.

Clinical Features.—The blood may either remain in the stomach until partial digestion has occurred, when the vomited matter is dark in colour, resembling hare-soup or coffee-grounds, or it may be vomited bright red just as it is effused, in which case the haemorrhage is more serious. Where bleeding has occurred some time before the blood is vomited, the patient may, prior to vomiting and coincidently with the haemorrhage feel sick and faint.

It should be remembered that if there is blood in the stomach, part of it will probably pass through the intestines, and that the stools will be of a deep black colour, similar to that resulting from the administration of iron; to blood in the stools the name *melaena* has been given.

The **Diagnosis** depends on the recognition of bleeding from the stomach as compared with haemorrhage from the *lung*. If the blood is from the stomach it has generally a definite acid reaction; blood from the lung is alkaline. In blood from the stomach there is often partial digestion, whereas in haemorrhage from the lung the blood is generally bright red in colour, unless it has been swallowed and then vomited. The blood in haemoptysis is usually bright red and frothy, in other words, is mixed with air, whereas blood from the stomach may be clotted or fluid, but contains practically no air. The history of the case, the physical examination of the lungs and heart (especially in mitral lesions), and the careful examination of the stomach ought to aid in the diagnosis.

The haemorrhage from a simple ulcer is generally more copious and less frequent than haemorrhage from a malignant ulcer of the stomach, and in addition the chemical investigation of the stomach contents often enables the physician to distinguish with fair accuracy between the two conditions.

Prognosis.—Excessive haemorrhage is of course dangerous and may lead to fatal syncope, while in other cases a limited haematemesis may be actually beneficial to the patient by relieving backward pressure, but the question of prognosis depends on the cause of the haemorrhage.

Treatment.—The general treatment of haematemesis is to

insist upon rest for the patient, and, above all, rest to the stomach. Food should be given by the bowel in severe cases, and only a little bit of ice to suck, or a few drops of iced water to relieve thirst, should be administered by the mouth. An ice-bag applied over the stomach may arrest haemorrhage, while various remedies are of value, such as acetate of lead in 1 to 4 grain doses, ergotin in 1 to 3 grain doses, or hypodermic injections of ergotin or ergotinine.

An attempt may be made to soothe the patient by hypodermic injections of morphia, where excitement and nervousness demand interference. When it is safe to allow food by the mouth, it should be given in small quantities and often predigested. It is hardly necessary to add that the treatment of haematemesis in all its details must depend largely upon the disease to which it is due.

(10) Erosions of the Gastric Mucosa

Haemorrhagic erosions are extremely common in cases of backward pressure, whether from the portal system, the lung, or the heart, and in many acute diseases similar erosions may be seen. They may bleed freely, and death has even occurred from the haemorrhage. The remarkable cases of haemorrhage following upon abdominal operation, and perhaps especially in cases in which a kidney has been incised, are often due to these haemorrhages.

While these erosions may form gastric ulcers, and are in reality superficial ulcers, they should be described separately.

(11) Gastric or Peptic Ulcer, and Duodenal Ulcer

Perforating ulcer of the stomach or duodenum.

Etiology.—Most common between the ages of twenty and thirty-five. Gastric ulcer is said to occur in about 5 per cent of all dead bodies, although not necessarily in an active condition, but this figure is apparently an exaggeration. Forty per cent are males, and 60 per cent of cases of gastric ulcer are females, and in men the age more nearly approaches middle life. It is dependent on gastric digestion with, probably, a local vascular cause (possibly thrombosis or embolism).

Trades in which pressure is exerted over the stomach are

said to predispose, as, for example, that of the shoemaker; and tight lacing in women is similarly prejudicial. Chlorosis and anaemia are often associated, and servant girls so affected are frequent victims of gastric ulcer.

Duodenal ulcers are found in the first part of the duodenum where the gastric contents pass in their acid condition prior to neutralisation.

Pathological Anatomy. — **Gastric Ulcer.** — Generally single or only 2 to 5 in number, usually situated on the posterior wall, near the pylorus, and specially near the lesser curvature. They are rarely found on the anterior wall, and when so placed are very dangerous, because they are apt to perforate without contracting any adhesions. The ulcers are round or oval, typically punched out, and generally terraced when chronic. This is due to the opening through the muscular coat being smaller than that through the mucosa, and that again smaller than the perforation through the peritoneum. They vary greatly in size, and may be very large, the larger ones being nearly always chronic. The ulcers may heal, become very chronic, break out again after healing, or they may perforate. Generally adhesions form before the peritoneal coat is eroded, and the pancreas, the left lobe of the liver, and the omentum often contract such adhesions, while sometimes the ulcer opens into the lesser omental sac. The pancreas or liver may be deeply eroded, and large vessels, either in the stomach wall or in the organs just mentioned, may give rise to very severe or even fatal haemorrhage, the pancreatic artery sometimes being opened into. Occasionally an aneurism forms in the floor of an ulcer. Following on perforation into the lesser omental sac there may be a subphrenic abscess and possibly even subcutaneous emphysema; more rarely the ulcer perforates into intestine, pericardium, or elsewhere. Sometimes cicatricial contraction produces various changes in the stomach, as, for example, hour-glass contraction, but more frequently it causes stenosis of the pylorus with secondary gastrectasis.

Pathology.—The stomach wall is often more or less digested post-mortem, and slight erosions occur in acute gastric catarrh; if therefore the stomach wall is locally damaged, an ulcer may result from the action of the gastric juice. The terraced ulcer is believed to correspond to the area of blood-supply of an end-artery in the stomach wall. (1) Virchow

thought that plugging such an artery with an embolus might be the explanation, while (2) Cohnheim contended that the diminished alkalinity of the blood in anæmia and chlorosis might permit the gastric juice to attack the stomach wall. (3) Gastric ulcer follows injury sometimes, but the theory of a preceding injury in every case is untenable, and no such theory, or for the matter of that, no theory as yet formulated, seems to offer a satisfactory explanation. (4) Bacterial necrosis has also been suggested by Martin, but the evidence is insufficient.

Duodenal Ulcers occur in the first part of the duodenum: they are more common in men, and they have been known to follow superficial burns over the region of the abdomen, although they are certainly also due to the same conditions which produce gastric ulcer. Surgical experience points to a much larger number of duodenal ulcers being in existence than our medical and pathological statistics seem to bear out.

Clinical Features.—In **Gastric Ulcer** there may be no symptoms, or there may be more or less long-standing dyspepsia of varying degree. Shooting or aching pain is, however, common either in the epigastrium, where it is increased by pressure, or about the 10th dorsal spine or the right shoulder blade. It is often relieved by altering the position of the patient so as to prevent the gastric contents from resting upon the ulcer, and it is generally increased after a meal. In some cases, curiously enough, pressure relieves the pain. The pain is probably due to the action of the gastric juice, which is stated by Riegel, Ewald, and others to contain a great excess of hydrochloric acid. Vomiting may be induced by the sufferer, to relieve the excessive discomfort. Nausea and vomiting are common, and haematemesis is a prominent symptom. Giddiness indicates haemorrhage into the stomach, and soon after, partially digested blood is vomited, producing the typical hare-soup or coffee-ground-like vomit. If a large artery has been eroded, bright arterial blood may be brought up. Blood is often passed in the stool, giving it the tarry appearance called melæna. Where haemorrhage is very slight there may be no haematemesis, and only melæna may be present. Care must be taken not to confuse melæna with stools coloured black by bismuth or iron sulphides. Palpation over the epigastrium may not merely induce or increase the

pain, but may yield definite evidence of a pyloric tumour largely due to thickening and cicatricial adhesions of an old ulcer.

Perforation may lead to peritonitis, in the first instance localised to the lesser omental sac, or to a general peritonitis, while perforation into the pancreas or liver may give rise to special symptoms and often severe haemorrhage.

Duodenal Ulcer.—Considerable attention has recently been directed to the clinical features of duodenal ulcer. There is no pain for 1½ to 2 or 3 hours after food, and then it begins. It is localised to the right of the middle line, and tends to shoot to the back or round the right side. The pain is often relieved by eating, just like the gastric pain of hyperchlorhydria which, in point of fact, is often present. There is rarely vomiting, and blood is usually only passed by the bowel (melæna). The symptoms may disappear in time or may become more marked, or may, after a period of improvement, reappear with greater virulence than at first.

The **Diagnosis** may be difficult for want of symptoms, but the presence of pain following immediately on the ingestion of food, and associated with an excess and not a diminution or absence of hydrochloric acid, should make one suspect the presence of a simple gastric ulcer. Blood in the vomited matter is very important. It is hardly possible for any length of time to mistake a *malignant ulcer* for a simple one, while *gall-stones* frequently cause not merely characteristic pain, but the occurrence of subsequent jaundice, and tenderness not so much over the stomach as over the liver.

When a gastric ulcer has ruptured, although general peritonitis and the typical symptoms of collapse taken with the previous history may render the diagnosis easy, there are cases in which much difficulty exists. The ulcer may rupture into the lesser omental sac, and it may be difficult to ascertain the cause of an abscess in that region. It is impossible, as a general rule, to diagnose with certainty the rupture of a gastric from a duodenal ulcer prior to operation.

The *gastric crises of locomotor ataxia* may occur without any ataxic or other cord phenomena, but these develop later, and the Argyll-Robertson pupil, lightning pains, ataxia and loss of knee-jerks rectify any mistake.

Prognosis.—In most cases it is good, especially if the patient be wisely treated, but the risk of perforation should

not be forgotten, and in the event of its occurring prompt surgical treatment is necessary. Serious haemorrhage is always dangerous. It is possible that a long-standing simple ulcer may acquire a malignant character, although this seems to be more rare than we might reasonably expect.

Treatment.—Absolute rest to the stomach is essential, and it is prudent to feed entirely by the bowel for several days, simply giving a small quantity of iced water by the mouth to allay the local discomfort. An ice-bag applied over the stomach is often of value, while bismuth subnitrate (gr. 10-30), dilute hydrocyanic acid (mj 3), and similar remedies may be administered for the relief of pain.

Haemorrhage is a most alarming symptom, and demands energetic treatment; and should ice and perhaps the administration of several hypodermic injections of ergotin not suffice, the aid of the surgeon must be sought, and the haemorrhage arrested by surgical interference.

Occasionally, it must be remembered, haemorrhage is from superficial erosions, and not from an ulcer at all.

When pain and haemorrhage have ceased, and it is probable that the ulcer has healed, feeding by the mouth should be cautiously and gradually resumed, and for a long time the patient should be enjoined to avoid all indigestible articles of food, to aid the stomach by careful mastication, and to attend carefully to the bowels.

The presence of an ulcer near the pylorus may cause stricture with gastric dilatation, and much benefit may be obtained from having gastro-enterostomy, or, better still, gastro-duodenostomy performed.

The treatment of duodenal ulcer demands the same care and much the same measures.

(12) Cancer of the Stomach

Etiology.—The stomach is a common site of primary cancer, and the pylorus is the most frequent part of the organ to be involved. More rarely the cardiac end of the stomach or middle of the viscus is affected.

The age of patients affected with cancer is usually after middle life, and in connection with the etiology of cancer in the stomach it is important to remember that a simple ulcer

may become malignant, and that sometimes definite gastric catarrh precedes the recognition of a tumour.

Sarcoma of the stomach is rarely primary, although secondary nodules may occur in the wall.

Pathological Anatomy.—The commonest varieties of cancer of the stomach are, in their order of frequency, medullary, scirrhous, adeno-carcinoma, and colloid cancer. The last form is really a degeneration of a medullary cancer.

The pylorus is the usual site of disease, and the lesser curvature more frequently than the greater. It is common to find adhesions between neighbouring viscera as the result of cancerous growths, and in these nodules suppurative changes may take place, causing eventually a general suppurative peritonitis in some instances.

The stomach is often greatly dilated where the tumour is near the pylorus, and the fungating growth may cause repeated and sometimes serious haemorrhage. If the tumour is near the cardiac orifice of the stomach, the viscus is usually of small size, while a neoplasm involving the middle region of the stomach may cause marked hour-glass constriction of the organ.

When dilatation occurs, the muscular coat of the stomach becomes weak, and almost invariably the gastric secretion is seriously affected, and it is usually stated that the hydrochloric acid diminishes almost to the vanishing point, thus permitting fermentative changes to occur in the stomach contents.

Occasionally cancer of the stomach is secondary to primary disease of the breast or of some other region; while the lymphatic glands, liver, peritoneum, intestine, lung, and pleura may all be involved secondarily when a primary tumour is situated in the stomach.

Perforation is not uncommon: it may occur into the general peritoneal cavity, sometimes into the lesser omental sac, or into the intestine, pleura, lung, or pericardium.

Clinical Features.—The *general* symptoms include progressive cachexia, with loss of weight and strength, while anaemia is usually dependent partly on the amount of interference with nutrition and partly on the loss of blood, which may be a feature of the case. There may be some pyrexia.

The *special* symptoms include pain, frequently shooting through to the back, and often of a gnawing or dragging character. It is sometimes increased by the ingestion of

food and by external pressure. According to Head, the skin area, referable to the stomach, and over which pain may be felt, is situated between the nipple and the umbilicus in front, and the 5th to the 12th dorsal vertebrae posteriorly. Severe dyspepsia is another prominent symptom, often with inability to take food, and very frequent vomiting. The vomited matter contains a minimum of hydrochloric acid, often none at all, and, if the stomach is dilated, the gastric contents ferment, and sarcinae and other organisms are associated, while blood is usually present. Haemorrhage is a frequent symptom: the blood may be coffee-ground-like, or of brighter colour, depending on whether it is partially digested or not. Haemorrhage is certainly present in a large proportion of cases at some time or other, although the nature of the tumour and the degree of ulceration are important factors.

In examining the stomach contents careful microscopic investigation should reveal sarcinae, yeast fungi, and many other fungi and bacteria, including the long thread-like bacillus of Boas and Oppler, which is responsible for the manufacture of lactic acid, although we rarely find small fragments of the tumour in the vomited matter.

Physical Signs.—*Inspection.*—The presence of peristalsis is probably the most definite and significant feature, which, however, is only noted in cases of dilated stomach. A visible tumour is present in about one-third of the cases, and it may be seen to descend with the diaphragm on inspiration. Osler suggests that distension of the stomach with carbonic acid gas in suitable cases may demonstrate the presence of a tumour, or, at all events, the existence of peristalsis. Sometimes secondary nodules appear in the neighbourhood of the umbilicus, sometimes in other parts of the peritoneum. *Palpation.*—Careful palpation reveals the existence of a tumour in a large proportion of cases, and the movement of the tumour with the diaphragm is often easily felt. It is a remarkable fact that not a few pyloric tumours are excessively mobile. In most cases of tumour in this region the mass is felt to be hard and nodular. *Percussion* demonstrates, especially if associated with auscultation, the outline of the stomach, and it may be again stated that a pyloric tumour is frequently associated with dilatation, while a tumour situated at the cardiac orifice causes atrophy of the organ. *Auscultation* may reveal gurgling

of gas through the pylorus and, where present, the existence of peritoneal friction.

Complications.—The most frequent complications are certainly due to secondary growths, and the lymph glands, liver, peritoneum, etc., may all be involved. Perforation has already been mentioned, while only in a small number of cases does a painful illness terminate by gangrene.

Diagnosis.—The recognition by palpation of a tumour in such a large percentage of cases renders the diagnosis easy. The absence of hydrochloric acid is of great value, especially when coupled with the age of the patient; *simple ulcer* is more common in young persons, and is associated with an excess of hydrochloric acid. In *pernicious anaemia* the examination of the blood is distinctive, because in most cases of tumour the anaemia is of secondary development, and shows none of the characteristics of the pernicious type. In doubtful cases surgical exploration should invariably be carried out if there is the remotest chance of benefit.

Prognosis.—It is rare to find patients with gastric cancer live over two years. The duration depends much on the interference with nutrition, but as a rule a period of some months is all that the patient can count upon, and occasionally an acute case terminates in a few weeks. It should be remembered that pyloric tumours are sometimes operable before secondary growths occur.

Treatment.—The only treatment possible is palliative except in cases where removal can be carried out. Give easily digested food, and wash out the stomach in cases of dilatation with very great care, using a soft oesophageal tube. Sometimes antiseptics have been given by the mouth, such as carbolic acid or creosote. In many cases morphia is requisite and for the comfort of the patient it should be freely administered. Sometimes the performance of gastro-enterostomy prolongs the life of the patient for many months, and greatly relieves pain and discomfort, while in cases of tumour near the cardiac orifice of the stomach gastrostomy is imperative.

(13) Dilatation of the Stomach; Gastrectasis

Etiology.—An **Acute** form is described, which is extremely rare, and which may be dependent on over-distension of the

stomach with solids or fluids, but most recorded cases have followed the use of a general anaesthetic for operation or injuries to the head and spine.

The **Chronic** or usual form is generally dependent on either (1) pyloric stenosis, often the result of gastric ulcer, simple or malignant, although sometimes due to simple thickening, or (2) loss of muscular tone, which is a common sequel to chronic gastric catarrh, general debility, anaemia, etc. (3) In addition to the two foregoing factors there is a third, namely, the distending force of the stomach contents, fluid, solid, or gaseous. The stomach is often greatly enlarged in patients who fill it to excess as in diabetics and in persons who consume enormous quantities of beer.

Pathological Anatomy.—In dilatation due to pyloric stricture, the stricture may be the result of an ulcer, simple or malignant, or may be due to adhesions between the pylorus and neighbouring organs; but whether the condition is due to pyloric stricture or weakening of the muscular wall of the stomach, there is great enlargement of the organ, which, in place of holding some 50 ounces, the capacity of the adult stomach in health, may hold 120 or more ounces. The shape of the organ varies considerably: the stomach may retain its usual form, the dimensions only being greater, or it may become U-shaped, the lesser curvature being enormously lengthened, and the greater curvature often reaching in a marked case to the symphysis pubis.

The stomach contents generally ferment as the result of the presence of abnormal vegetable and bacterial agents: there is usually a diminution in the normal constituents of the gastric juice, and in fact both the hydrochloric acid and the pepsin may be nearly absent. Butyric acid fermentation, due to the presence of the B. butyricus, lactic acid fermentation, the result of the Boas-Oppler bacillus, and acetic acid fermentation, due to the yeast fungus, may all be present, and Sarcinae ventriculi are almost always found in the stomach contents. Gases of various kinds are generated, and sometimes it is said that if a match is applied the emetated gas may even ignite (marsh gas when emanating from the mouth).

The **Clinical Features** are A *local*, and B *general* and *reflex*.

A. The *local symptoms* include epigastric discomfort often with pain, and, if dilatation is considerable the vomiting of

large quantities (5 or 6 pints) of fermenting stomach contents at intervals of 1 or 2 days, the contents containing sarcinæ and other organisms just referred to. Distressing eructations of ill-smelling gas and dreadful fetor of breath render the patient disagreeable to himself and to those about him. Peristaltic movements are often observable over the region of the organ and are easily stimulated by the application of anything cold to the skin, and colicky pain may be associated. The appetite is poor and thirst often great. The bowels are constipated, and the nutrition of the patient suffers greatly.

Physical Signs. *Inspection* shows generally the peristaltic movements already referred to, and sometimes bulging of the dilated stomach. *Palpation* reveals marked splashing, which is easily elicited if the stomach is full. *Percussion* gives a fairly definite outline of the greater curvature of the stomach, but should be combined with *Auscultation*, when the loud stomach note will be at once detected by the ear applied to the stethoscope even although the transverse colon covers part of the organ. The examination should be carried out with the patient lying on his back.

Other methods of defining the stomach consist in the introduction of carbonic acid gas into the organ by the administration of 20 grains of citric acid and a similar quantity of bicarbonate of soda in two separate solutions, or air may be pumped into the organ through an œsophageal tube. Another method has been suggested, namely, emptying the stomach and then pouring into it a measured quantity of fluid, and estimating the level of dulness which 20, 30, or 40 oz. of fluid reveal by percussion, the patient being examined standing. None of these methods, however, is as simple as combining percussion and auscultation the accuracy of which depends on the fact that the stomach is never empty of air or gas. Yet another plan is to wash out the stomach and then introduce a meal of bismuth porridge, and immediately after, examine the stomach by the X-rays, the patient standing up while it is being done.

The tundus of the stomach may interfere with the heart causing palpitation, which is relieved by eructations, and a functional albuminuria is sometimes associated with a marked degree of gastrectasis.

B. The *general* and *reflex phenomena* can be readily appreciated. They depend on the loss of nutrition and also on the

absorption of poisons from the fermenting stomach contents. They include emaciation, headache, and general discomfort, and tetany has been noted as a not uncommon result of marked gastric dilatation.

The **Diagnosis** depends rather on the discovery of the cause of the dilatation than on its recognition. Frequently a tumour at the pyloric orifice is palpable, and the degree of patency of the pylorus may be ascertained by measuring the amount of solids and fluids put into the stomach and the total amount vomited up, remembering the amount of saliva swallowed. In a case where no fluid contents pass through the pylorus, the total amount vomited will therefore probably exceed the amount taken in, because of the added saliva.

Prognosis.—In mild cases rapid improvement occurs with treatment.

The **Treatment** consists largely in careful dieting, and where necessary, washing out the stomach with an antiseptic solution such as boracic acid ¾ drachms to the pint; permanganate of potash 10 grains to the pint; hyposulphite of soda 1½ drachms to the pint; and other agents, and frequently following this operation by a limited and carefully selected meal. The preliminary use of a solution of bicarbonate of soda aids in dissolving the glairy mucus with which the stomach wall is coated. Massage over the region of the stomach is often of benefit, and general tonic treatment, consisting not merely in the use of iron and strychnine, but also in residence in bracing hill air, the use of cold shower baths, and the careful regulation of exercise, meals, and hours of sleep, should be tried. An abdominal bandage is often helpful; the internal application of electricity, sometimes one pole being introduced into the stomach, is of value, and the high-frequency currents appear to be of distinct advantage in some cases.

In cases of very marked dilatation, the stomach should, in the first instance, be treated by lavage, and it is often prudent to consider whether gastro-enterostomy or gastro-duodenostomy should not be performed, or whether it is possible to remove the pyloric tumour which is causing the stricture. While rest to the stomach is certainly beneficial, it is unlikely that in a very marked case mere feeding by the bowel will do much towards curing the patient.

Where there is fermentation present, antiseptics may be

given by the mouth, of which the best, probably, are hyposulphite of soda in 10 to 15 grain doses, creosote in 1 or 2 minim capsules, and small doses of carbolic acid.

In the acute form of dilatation treatment consists in emptying the stomach at once and repeatedly if necessary. In not a few cases the condition is rapidly fatal.

VIII CONSTIPATION

RETENTION of the faecal contents of the bowels due to sluggish action or other causes.

Etiology.—(1) It may be the result of habit. Inattention to the calls of nature is often due to careless upbringing and dates from childhood, but in many busy lives the rush to work in the morning is responsible for the omission. In certain cases the cause is a painful fissure at the anus or haemorrhoids, which renders defaecation more or less torturing to the patient, and so tempts him to put off the evil day. There are many constipated persons, and especially women who constantly use enemata, and in such individuals the deficient natural action is partly due to this habit.

(2) Constipation may be caused by diminished or altered secretion. Diminution may occur as the result of a drain otherwise, such as follows excessive sweating after violent exercise, or a great flow of urine, or a tendency to dropsical effusion into the tissues. A too dry diet favours constipation [illegible] fact which should be remembered. The secretion is sometimes altered, as in cases where bile, one of nature's purgatives [illegible] not enter the intestine or does so in diminished amount.

(3) Constipation may be due to deficient peristalsis of the [illegible] and also to laxness of the abdominal wall. Peristalsis is undoubtedly diminished where bile does not enter the intestine in sufficient quantity, as mentioned above, but more commonly a sedentary life is responsible. Active muscular exercise greatly aids normal peristalsis. Certain articles of diet are admirable peristaltic agents, such as oatmeal, brown bread, vegetables and fruits of many kinds, and their omission in proper amount from the dietary is apt to induce constipation. The abdominal wall, if lax, does not support the intestines, and so constitutes another common cause of constipation. This is most likely to

occur in women after many pregnancies, and also in persons once stout who have become thin. But a weak abdominal wall is also not infrequently seen in neurasthenic and debilitated patients.

The constipation due to the opium habit is the result both of diminished secretion and also deficient peristalsis.

(4) Lastly, there are cases in which there is obstruction. This may be the result of pelvic tumours or of a stricture of the gut frequently malignant in nature, and not uncommonly situated at the sigmoid or one of the other flexures of the large intestine. In certain cases constipation may occur from displacement (enteroptosis), ballooning, or other alteration or malformation of a part of the intestine, and generally it is the sigmoid or rectum which suffers in this way. The displacement or distension of the intestine is sometimes due to a long mesentery, or may be the direct result of long-standing constipation. The pregnant uterus interferes with the normal action of the bowels.

After severe attacks of enteritis or dysentery, spasm of part of the bowel and especially of the colon may be present, and leads to severe and troublesome constipation which is really a form of obstruction.

Pathological Anatomy.—While strictures and tumours may cause constipation, the changes in the bowel are a sequel to the condition; these include distension more or less marked and permanent, thinning of the wall, ulceration, especially in the caecum, and often associated with small pouchings of the wall, in which little scybalous masses are frequently found.

Clinical Features.—A huge number of persons suffer from constipation, and they may even neglect artificial means of relief without appearing to be much the worse, while others are rendered miserable if the bowels fail to act, naturally or with the help of medicine, for a period of forty-eight hours. The chief results are discomfort, bad breath, and thickly furred tongue, flatulence, sometimes colic, general loss of energy and depression of spirits, headache, irritability, and sometimes giddiness. Great stress has been placed on the evil effects of absorption from the bowel, but whether they cause chlorosis or not, certainly constipation and flatulence are not infrequently the predisposing causes of appendicitis. There are

also pressure results on sacral nerves, causing neuralgia, and on veins, causing haemorrhoids, and possibly even, in very extreme cases, oedema of the legs. A rectum blocked with hard faecal masses may give rise to great distension of the bowel with gas, and faecal impaction may be so great as to simulate peritonitis from the severity of the consequent colicky pains, while vomiting, eventually becoming faecal, may even commence if relief is not obtained. On abdominal palpation we can feel the hard faecal masses, mostly in the large intestine and caecum. Sometimes these masses are tunnelled, and diarrhoea due to irritation may actually coexist with constipation.

The **Diagnosis** is generally obtainable from the history alone, but a careful abdominal examination is invaluable, and often when in doubt as to a supposed tumour, the bowels are first cleared out so as to get rid of all faecal accumulations.

The **Prognosis** depends on the duration of the constipation and the amount of trouble the patient is willing to take so as to effect a cure.

Treatment.—A glance at the etiology enables the reader to anticipate the treatment. Try to inculcate the importance of going to stool *every* morning after breakfast whether inclined so to do or not, and the desire to go may be aided by a few minutes' sharp walk, or in men by a morning pipe, while not infrequently much may be accomplished by dietetic measures. Recommend the patient to drink freely of weak tea or coffee at breakfast, to eat brown or whole-meal bread, oatcakes, and perhaps, best of all, to take porridge made of fairly coarse oatmeal. Fruit at breakfast is invaluable, and especially oranges, pears, figs, and prunes, while a tumblerful of cold or warm water sipped whilst dressing is helpful. At other meals besides breakfast, study and revise, if necessary, the patient's dietary, introducing plenty of vegetables if an insufficient amount is being taken.

Exercise is important, whether walking, riding, or cycling.

A great deal of good can be done, where the abdominal wall is lax, by recommending an abdominal bandage, and by ordering daily abdominal massage. The rubbing should follow along the line of the colon so as to assist natural peristalsis, and wet compresses worn in the morning have a considerable effect in certain cases. Electricity applied to the abdominal wall is also good.

There are many drugs suitable for different types of cases, and probably cascara sagrada, either as fluid extract in 20 to 60 minim doses, or extract in pill or tabloid form (gr. 2-6), is one of the best purgatives. It acts specially on the large intestine, does not gripe in most cases, although it is a peristaltic agent, and the dose can be gradually reduced. A splendid pill consists of extract of nux vomica (gr. $\frac{1}{4}$), extract of belladonna (gr. $\frac{1}{2}$), and aloin (gr. 2), or compound rhubarb pill (gr. 2-4), and it may be taken at bedtime. These remedies tend to cure, although acting as purgatives, while, on the other hand, salts must too often be continued when once begun. Magnesium sulphate (℥ 1-4) in warm water just before breakfast, and sometimes combined with dilute sulphuric acid (♏ 20-30), acts well, and so do Carlsbad salts, Friedrichshall, Hunyadi Janos, Apenta, and many other aperient mineral waters. Salts clear out the intestine, and are, therefore, useful when beginning treatment in cases where the bowel is overloaded, but it is well to return as soon as possible to the type of remedies first mentioned.

Enemata of soap and water, sometimes preceded by an injection of olive oil, and small enemata of glycerine (℥ 1-2), are often helpful, and especially are big enemata beneficial where there is any faecal impaction. Inject 60 to 100 ounces of an enema made of Castile soap dissolved in water about blood-heat, and precede, where necessary, with 10 to 20 ounces of warmed olive oil, which the patient should try to retain as long as possible. Place the patient on his left side with the hips raised on a pillow before administering the enema, and give the soap and water very slowly. Enemata are apt to cause a habit, and they should not be used unless absolutely necessary. Sulphur and cream of tartar in equal parts, 1 to 2 teaspoonfuls of the mixture being taken at bedtime in a little water, is an old-fashioned but serviceable purgative, and amongst others which might be mentioned are castor oil, purgen (phenolphthalein gr. $\frac{1}{2}$-4), elaterium, compound jalap powder, and rhubarb in the well-known formula of Gregory's mixture.

Constipation in Infants

The causes of this condition are faulty diet, want of tone as regards the intestinal walls, and perhaps a deficient flow of

bile. Congenital strictures occasionally occur, and may call for operative interference, while sometimes there is only a trifling band or adhesion. Intussusception and obstruction from the development of a kink or constriction by adhesions cause constipation, but they are treated of under intestinal obstruction. Sucklings depend partly on the purgative effects of their mother's milk, and attention has to be paid so as to secure a suitable maternal dietary. A dose of castor oil taken by the mother operates through the milk on the child, and it is well to remember the ease with which constipation can be treated in the sucking by this means. A soap suppository is a well-known nursery remedy.

Imperforate anus, for which operative interference is the sole chance of relief, belongs to the domain of surgery.

IX. ENTERALGIA OR INTESTINAL COLIC

Irregular and spasmodic contractions of the intestine which give rise to pain. Renal and biliary colic will be described elsewhere. It is probable that in some cases of enteralgia the pain may be neuralgic, and not due to irregular peristalsis, but we have little definite evidence of this.

Enteralgia is a symptom of many different diseases, but it is a most important one, and as such it should have a separate description.

Etiology.—(1) It may be due to something decomposing, irritating, or indigestible in the contents of the bowel, and also to drugs acting as powerful peristaltic agents in large doses (colocynth and jalap). Food suitable for a person of strong digestion may cause colic in another, and food imperfectly digested in the stomach may become an irritant because it ferments. Chyle may undergo normal decomposition too far up the intestine, and so lead to colicky pain. Lastly, faecal masses in constipation set up enteralgia, the masses stimulating irregular peristalsis.

(2) Disease of the bowel of many kinds causes colic—thus enteritis, cancerous stricture, intussusception, appendicitis, dysentery, and ulcerations set up severe colicky pain which is one of the chief symptoms present.

(3) There are causes of colic which may be termed

nervous; probably lead colic is partly produced in this way, and nervousness and cold in susceptible persons may set up severe attacks of colic. Strychnine in large doses, and other toxic agents with a similar action, produce violent colic.

(4) Colic may be due in women to pelvic irritation, and is, in such cases, of reflex origin.

Clinical Features.—There are many varieties of colic, from a slight aching pain in the abdomen to a severe paroxysm which is almost unbearable, but in every case it varies in intensity from time to time during an attack. The colic described here is not the colic which results from complete obstruction. The pain may be due to spasms in the large or small intestine, and it is often localised near the umbilicus, but may be in any part of the abdomen. It is relieved by pressure with the hand where there is little distension with gas, but it is greatly increased if associated with the presence of much gas. The pain may follow the passage of the food into the intestine, being due to what is called intestinal dyspepsia; or it may occur just before the bowels act, and may be relieved by such action. Often the passage of a little flatus per rectum is followed by disappearance of the pain. Diarrhœa may follow an attack of colic, and thus the removal of the irritant is secured. In cases in which the colic is due to obstruction, visible peristaltic contractions of the intestine are often seen through the abdominal wall.

The patient's face in a fairly severe case is grey and anxious, his hands and feet are cold and clammy, and drops of perspiration may be seen on his forehead. His pulse is small and of low tension, but generally there is little or no pyrexia unless inflammation is present. The abdomen is rigid, and often the testicles are retracted, while frequency of micturition is common. Vomiting is occasionally present, but its presence should make the physician suspicious of a serious cause for the colic.

The **Diagnosis** depends on the recognition of the cause of the pain, and chiefly whether it is of trivial origin or points to a serious condition, calling for prompt operative interference. In trivial colic the pain is more spasmodic and less constant than in obstruction. Pressure generally relieves colic, where there is little distension with gas, while in obstruction it always aggravates the pain. On examining the abdomen

less rigidity is found with ordinary colic than with obstruction, while the absence of vomiting and temperature generally points to colic, and the history of the case should be suggestive. The pulse may be helpful—small and feeble in colic, it is rapid and often wiry in obstruction. Lastly, a purge eventually relieves colic, but intensifies the patient's suffering in obstruction.

The **Prognosis** is favourable in most cases.

The **Treatment** of a case of ordinary colic consists in clearing out the irritant, and giving a sedative at the same time. A full dose of castor oil, with 15 to 30 minims of laudanum, is most efficacious, and should be combined with hot fomentations, a linseed poultice, or, in mild cases, an indiarubber hot-water bottle applied to the abdomen. Salol, in doses of 20 to 40 grains, acts slowly but certainly, diminishing the fermentation and consequent distension of the intestine, and wood charcoal (gr. 10-20) may be given in cachets for the same purpose. In many cases an enema of 20 to 40 ounces of soap and water affords very rapid relief, and may be combined with the purgative and antiseptic internal treatment. In cases of very severe pain, opium by the mouth, or hypodermically, and an enema are most successful. Where a very severe irritant is in the intestine, a course of sedatives is necessary, but the chief point is to ascertain the cause of the colic, and then endeavour directly to treat it. Intestinal worms not infrequently produce much colicky pain, and the measures for their removal are stated elsewhere. Sometimes dietetic treatment is requisite, but the relationship of the pain to the passage of food through the pylorus gives a confirmative hint in these cases. Hysteria may be responsible for pain truly colicky in nature, or merely simulating it, and valerian or other anti-hysterical remedy is often of value in such cases.

X. DIARRHOEA

1. In Adults

This is a symptom of many diseases, and consists in excessive discharge from the bowels of too fluid faeces.

Etiology.—Diarrhoea may be due to—(1) a general con-

dition, and occurs in such diseases as pyaemia, septicaemia, etc.; or (2), and more commonly, to local changes, such as intestinal catarrh, ulceration, and the presence of irritants in the intestine.

There are three modes in which diarrhoea may be produced: (1) By increased peristalsis of the gut, as from the use of colocynth and similar drugs. (2) By increased outflow of fluid into the bowel from the mucosa, as from the administration of a dose of salts. It should be added that a somewhat similar condition obtains when too much fluid is taken in by the mouth. (3) Possibly there is in some cases diminished absorption of fluid by the intestinal wall.

There are endless causes of diarrhoea which fall under the first of these heads, and they include enteritis of all kinds, ulceration of the intestines, the ingestion of irritants, and local irritation at the anus or in the rectum, besides waxy degeneration of the whole or part of the alimentary tract.

Clinical Features.—Many varieties or types of diarrhoea depend for their characteristics on the cause of the condition, and all of the more important are described elsewhere. It is only necessary to mention summer diarrhoea, and the forms of diarrhoea which are due to some organism, and which occur in epidemic form, often the result of contamination of drinking-water with sewage. Dysentery, cholera, and similar diarrhoea-producing diseases call for no detailed reference. Apart altogether from these forms we should remember, however, that nervousness and sometimes chill may cause diarrhoea in individuals who are otherwise healthy, and these do not fall under any special group. It is well known that to a nervous person the prospect of an examination, or an appearance in the witness-box, may induce a well-defined, if brief, attack of diarrhoea. There are also individuals with very sensitive intestines, to whom a trifling quantity of a green vegetable would prove an irritant.

The number of motions varies greatly, and in excessive diarrhoea, as in severe forms of dysentery, they may number over a hundred in a day. Where there is much diarrhoea the motions are pale, and almost free from bile-staining, as in the evacuations of an Asiatic cholera patient. There may be colic or there may be tenesmus, by which is meant straining after the evacuation has taken place. The amount of colic present

varies with the cause of the condition, and the tenesmus is often associated with enteritis, or ulceration of the large intestine, and especially of the rectum.

Remember that diarrhœa may be in reality the result of constipation, and that sometimes the faecal masses are tunnelled, and a well-marked diarrhœa may develop from the irritation.

The **Prognosis** depends on the cause of the condition and the possibility of successful treatment.

Treatment.—Try to remove the irritant, if there is one, and give a purgative, such as castor oil, when necessary. To check diarrhœa give astringents and antacids. Bismuth carbonate in 10 to 40 grain doses, chalk mixture, catechu, kino and ipecacuanha are invaluable. Opium is often necessary, and there are many excellent pharmacopœial preparations, such as the pulvis cretae aromaticus cum opio (gr. 10-40), pulvis kino compositus (gr. 5-20), pulvis ipecacuanhae compositus (gr. 5-15). Where ulceration is present, rectal injections of either silver nitrate, or starch and laudanum are sometimes given, but much depends on the nature and site of the lesion.

Persons liable to diarrhœa from trivial causes should wear a cholera belt, and should have a carefully revised dietary, green vegetables and fresh fruits especially being limited to a very moderate amount.

2. Diarrhœa in Children

This is one of the great causes of infant mortality. A child is more likely to suffer from dietetic diarrhœa than an adult. In the hand-fed infant improper diet is a common cause of looseness of the bowels (milk from a cow suffering from mastitis is a good example), while a chill, and many other causes, are apt to induce attacks. Epidemic summer diarrhœa is common, and is treated of separately. An infant suffers seriously from any prolonged drain on the system, and the more delicate the infant, the more rapidly does the strength run down. He becomes flabby, pale, and pasty looking, with fontanelle sunk, face pinched, and expression anxious. The tongue is furred, and the child is often fretful and whining, much of the discomfort being due to the presence of some degree of colic. There are different types of infantile diarrhœa

depending on whether it is due to intestinal indigestion with or without fermentation, to enteritis simple or tubercular, to cholera nostras or to cholera infantum, types more or less distinctive. Some of these types are due to special micro-organisms, but probably simple diarrhœa in the infant may be due as much to poisons produced by such organisms as Bacillus coli communis and Bacillus aerogenes, which are always in the child's intestine, acting on the milk, and not to any inherent toxins present in the organisms themselves.

The **Prognosis** is only favourable if there is rapid improvement under treatment, dietetic or medicinal, as death is inevitable in a weakly child with protracted diarrhœa.

The **Treatment** varies with the nature of each case. Care should be taken to find out any error in diet, and in cases of fermentation, or where an organism is responsible, salol, charcoal, and similar remedies are of great value. For green stools in infants no medicinal agent appears to act so satisfactorily as dilute hydrochloric acid in 1 to 3 minim doses before meals. In many other cases a mild purge such as castor oil should be given, followed later by astringents. In place of castor oil the following powder might be substituted:—

℞

Pulveris Rhei Radicis	gr. 1-1½
Hydrargyri cum Creta	gr. 1-3
Pulveris Ipecacuanhae compositi . .	gr. 1-3
Pulveris Cinnamomi compositi . .	gr. 1

Fiat pulv.; tales 6.

Sig.: One powder to be taken at bedtime, and not repeated unless ordered.

XI. ENTERITIS

Inflammation of the intestines in whole or in part. There may be a duodenitis, a colitis (colon), a typhlitis (cæcum), an appendicitis, and a proctitis (rectum).

The following varieties of enteritis may be differentiated: (1) Catarrhal enteritis acute or chronic; (2) Diphtheritic or membranous enteritis, which generally implies a very intense inflammation with the formation of a membrane, such as may occur in diphtheria, although not necessarily in every case due to the diphtheria bacillus; (3) Ulcerative enteritis,

the ulcers being generally typhoid, tubercular, or dysenteric, though sometimes simple in origin; and (4) Mucous colitis.

(1) Catarrhal Enteritis in Adults

Varies much in degree, and may be the result of many different causes, and it should be remembered that a very severe type of catarrhal enteritis may lead to the formation of a "diphtheritic" membrane, while ulceration may follow as a sequel.

Etiology.—Irritants of all kinds, whether unripe fruit, decomposing meat, or arsenic, may set up enteritis. Excess of some article of food which, in smaller amount, would be wholesome, exposure to cold, and possibly even nervous causes, may all give rise to mild attacks. It is the experience of every physician that a mental shock, a period of great anxiety, and, perhaps most of all, excessive nervousness, can produce a catarrhal enteritis in susceptible individuals. But catarrhal enteritis may also be part of a specific fever, such as influenza, diphtheria, pneumonia, and typhoid, and it is even more commonly present in pyaemia and septicaemia. It may be a sequel to local inflammation such as an ulcer, tumour, or peritonitis. Lastly, in the terminal stages of many diseases, and especially Bright's disease, heart disease, common cirrhosis of the liver, leucocythaemia, Addison's disease, and cancer, a catarrhal enteritis may develop, probably due to an infection by organisms and allied in nature to pyaemia.

Pathological Anatomy.—Much depends on the stage of the condition, because mere hyperaemia disappears post-mortem, and only a marked degree of congestion is visible after death. The valvulae conniventes always show very definite injection where the jejunum is seriously involved, and the tips are specially congested and covered with thick glairy mucus often blood-stained. Peyer's patches and the solitary glands are often swollen, and in certain cases the mucosa is infiltrated with pus and has begun to become shreddy. Very commonly small superficial ulcers are seen after death. In chronic cases there may be a varying amount of slate-grey pigmentation due to altered blood pigment, and the wall of the affected part of the intestine may either be thickened or thinned.

Clinical Features. — In most ordinary cases of acute catarrhal enteritis due to the ingestion of an irritant there is marked abdominal pain, colicky in character, and associated with, or following it, severe diarrhoea which ceases when the inflammation subsides, as it does soon after the irritating ingesta are got rid of. There is often flatulence, and there may be nausea and vomiting. Where it is more severe, blood is seen in the stools (melaena) with much mucus, while in very bad cases the inflammation may lead to paralysis of the wall of the bowel and obstruction with constipation. If the *small* intestine is involved there is very severe colic and a fair amount of distension, but little or no tenesmus, while the evacuations will almost certainly contain undigested food. On the other hand, if the *large* intestine is chiefly affected there is more tenesmus (especially if it is the lower part of the colon and rectum), more borborygmi or rumblings of gas, more mucus in the stools, but little undigested food, and it is said that the abdominal distension is apt to be greater. The appetite is much more interfered with by a catarrhal inflammation higher up the intestine. The motions are less and less bile-stained according to the severity of the diarrhoea. Thirst is always great, and the degree of pyrexia indicates fairly clearly the gravity of the case.

Where the bowel is paralysed and peritonitis is threatening, or has actually commenced, as in strangulated hernia, etc., there is acute obstruction with its typical clinical features, namely, a rigid, board-like abdomen and vomiting, rapidly becoming faecal in character.

The enteritis associated with chronic Bright's disease, common cirrhosis of the liver, etc., is often very intractable, but although it rapidly kills the patient, there may be very little pain.

Lastly, a **duodenal** catarrh has distinctive features. It generally follows a gastric catarrh, and it is apt to cause catarrhal jaundice from the blocking of the ductus choledochus; in severe cases there is often much vomiting, but generally little diarrhoea, at least for a time; the appetite is lost, and should the patient drink so as to satisfy the intense thirst, the fluid is usually vomited.

Diagnosis.—Enough has been said to enable a diagnosis to be made between enteritis of the small and the large

intestine. It should be remembered that an *appendicitis* may be part of a more extensive catarrhal colitis, but the pain is not in that case limited to M'Burney's point.

The **Prognosis** depends on the type of catarrhal enteritis present and the success of treatment.

The **Treatment** is dietetic as well as medicinal. An irritant, if still present, must be removed, and it is often well to begin with a dose of castor oil and laudanum similar to that recommended for colic. Rest in bed is essential, and either hot fomentations or linseed poultices should be applied to the abdomen, or else the ice-bag. Bismuth subnitrate (gr. 10-30) with dilute hydrocyanic acid (m 3) is often insufficient, and morphia must be given, sometimes in the form of suppositories (gr. $\frac{1}{4}$-$\frac{1}{2}$), sometimes hypodermically (gr. $\frac{1}{8}$-$\frac{1}{4}$), or sometimes by the mouth. Starvation, with only little pieces of ice to suck, gives the inflamed intestine a chance of partial rest and recovery, and in cases of enteritis limited to the duodenum, or even the small intestine, an attempt may be made to feed by the bowel. Where food must be given by the mouth—and this is commonly the case—small quantities of milk and aerated water or concentrated beef-tea, chicken jelly, etc., should be administered, and either all given cold, and even iced, or else slightly warmed. On no consideration should hot and cold articles of food be partaken of by the patient during the same meal. Frequently chalk mixture, pulvis cretae aromaticus cum opio (gr. 10-40), pulvis ipecacuanhae compositi (gr. 5-15), and astringent mixtures containing the tinctures of kino, catechu, krameria (of each 3 $\frac{1}{2}$-1), etc., are beneficial. Salol (gr. 10-30), wood charcoal (gr. 10-20 in cachets), and other intestinal antiseptics are worth a trial in cases with much flatulence. Should diarrhoea continue, especially if associated with vomiting, it has been suggested that washing out stomach and bowel might be attempted, and in children this has proved successful. Once the patient has begun to improve, gradually increase the diet, adding fish cream or a similar easily digested semi-solid, and remember that it may be necessary to give partially digested food for a long time. The diarrhoea of chronic Bright's disease, common cirrhosis of the liver, etc., is very intractable, and all sorts of powerful astringents may have to be tried, such as the lead and opium pill (gr. 2-4).

Catarrhal Enteritis in Children

In young children the fault usually lies with the unsuitable food given, either sour milk or milk containing some bacterial agent, perhaps the most serious one being, not the tubercle bacillus, but the bacillus of Gaertner, which sets up an acute infective catarrhal enteritis. Many infants are fed on "what is going," which sooner or later in delicate children will cause enteritis, while others get an excess of some constituent of a normal dietary, such as milk sugar in condensed milk, to the exclusion of the fatty matters or cream of fresh milk. Catarrhal enteritis is more frequent in hot weather.

Pathological Anatomy.—Sir William Osler states that the normal stools of healthy, breast-fed children contain many organisms, but especially the B. lactis aerogenes and the B. coli communis, the others being more or less unimportant. In the diarrhœa of children the number and variety of potent organisms greatly increase. There may be not infrequently little follicular ulcers in the lower part of the small and upper part of the large intestine, related chiefly to the swollen solitary glands, but otherwise there is little to note.

Clinical Features.—In mild cases there are green stools, containing undigested milk and a varying amount of mucus, and the child suffers a little pain, and rapidly begins to fall below par, becoming flabby, pasty-faced, and emaciated, if not promptly treated. The sinking of the anterior fontanelle is a useful guide to an infant's general state of health. In more severe cases there is blood-stained mucus in the stools, which are very offensive; there is much pain and greater abdominal distension. Vomiting is common, there is frequently pyrexia, and, if not relieved, the child rapidly dies, sometimes in convulsions.

There are also chronic cases in which the symptoms are of longer standing and less urgent.

Treatment.—Attention must be at once paid to the milk. If the child is breast-fed, it may be getting suckled too often, or the mother's milk may be poor. Sometimes a wet-nurse is desirable, but often milk, as closely resembling healthy mothers' milk as possible, can be substituted. A mild purge is beneficial to remove all irritating contents of the bowel, and

directions about feeding may be all that the case requires. See that the milk is not allowed to turn sour, and that milk-cans and bottles are kept thoroughly clean. Raw meat juice is a valuable aid in bad cases, and after the preliminary purge, bismuth is often useful, while sometimes salol, β-naphthol, or wood charcoal controls fermentative changes. Remember that alcohol is necessary where exhaustion is extreme.

The following subvarieties of diarrhoeic conditions demand a brief reference.

Cholera Nostras and Cholera Infantum

(*a*) **Cholera Nostras.**—This form of diarrhoea is probably due to a specific organism or organisms of which we are yet uncertain. It is specially common in children, although adults do not escape. It is spasmodic, and is often due to some fermenting ingesta. The patient is suddenly seized with vomiting and diarrhoea, and the diarrhoea may resemble Asiatic cholera in its character and severity, and the patient may rapidly sink and die in one or two days, often with convulsions. The disease is closely allied to catarrhal enteritis. The treatment is troublesome, because the vomiting prevents the retention of most remedies. In severe cases hot bottles and, if collapse threatens, the intravenous injection of normal saline solution are the best kind of treatment. Where there is much temperature, cold water sponging or the wet pack may be tried. In mild cases sedatives and intestinal antiseptics should be administered. Warm saline enemata are often beneficial.

(*b*) **Cholera Infantum** is cholera nostras in infants. It is most common in bottle-fed babies in summer. The stools are offensive and greenish-brown, later almost odourless and alkaline and much like the rice-water stools of cholera. The child rapidly passes into the hydrocephaloid state, in which it lies comatous, with sunken features and fontanelles, cold clammy skin, but high internal temperature.

(2) Diphtheritic or Membranous Enteritis

The term "diphtheritic" means that there is necrosis of the mucosa with, in addition, an inflammatory exudation similar to what occurs in diphtheria, although the name does

not imply that Lofller's bacillus is in any way responsible for the membrane.

Etiology. It is found as a terminal infection in chronic Bright's disease, pyaemia, and common cirrhosis of the liver, and in many continued fevers, such as pneumonia and typhoid, and it may result from certain poisons, and especially arsenic, mercury, and lead.

Pathological Anatomy.—There is often very intense inflammation, and all the coats of the bowel may be affected. The muscular coat may be infiltrated with pus, and the bowel gangrenous. In other cases the amount of membrane is very limited, and may only involve the glandular structures or the tips of the valvulae conniventes. The membrane is the result of a bacterial necrosis, probably in both the severe and the milder types.

The **Clinical Features** vary according to the severity of the inflammation. There may, in cases of extensive necrosis of the intestinal wall, be all the symptoms of acute obstruction; in less extreme cases, profuse diarrhoea, with the passage of much blood and mucus. The other symptoms differ; occasionally much pain is experienced, while in other cases it is absent. Vomiting is generally more or less severe.

The **Treatment** consists in an attempt to allay the local inflammation and to check the diarrhoea.

(3) Ulcerative Enteritis

This may be due to a great variety of conditions.

Etiology.—Tubercle, typhoid, dysentery, and cancer all cause special forms of ulceration, described elsewhere under their respective headings. Superficial ulcers are common, affecting especially the Peyer's patches and solitary glands in the different forms of enteritis, and frequently occurring in children. The ulcers may be superficial and limited, or more extensive, and resemble dysenteric ulceration, although it may not be due to the specific organism or organisms of dysentery. There are ulcers in the caecum and colon which are due to hard scybalous masses, and the scybala may be found in small sacculi in the bowel, the mucosa of which is often ulcerated. Ulcers which do not appear to have any definite explanation may also develop in the large intestine. They are often single,

and may resemble in appearance a gastric ulcer, and, lastly, ulcers may open from without into the bowel, as in a localised suppurative peritonitis.

The **Clinical Features** vary greatly with the nature of the ulcerative process, but diarrhœa is common in nearly all cases. The motions are often offensive, and contain pus, mucus, blood, and sometimes shreds of necrotic mucosa. Pain in greater or less degree is frequently present, and ulceration of the lower part of the large intestine is accompanied by tenesmus. The results of ulceration include perforation, pylephlebitis suppurativa, hypertrophy or atrophy and sometimes constriction of the bowel.

The **Treatment** varies with the type present. Typhoid and dysenteric ulceration are described separately. Superficial ulceration should be treated on the lines laid down under catarrhal enteritis in adults and children, and ulcers due to scybala are generally cured when the masses have been cleared out. For ulcers in the large intestines the reader is referred to the treatment suggested for dysentery. The one principle which must be remembered in treating nearly all intestinal ulcers is rest to the bowel, and a carefully studied dietary, which should contain a minimum of waste matters. In ulcers situated in the small intestine, or even in the cæcum, an attempt may be made to feed by the rectum, provided, of course, there is little diarrhœa.

Tubercular Ulceration of Intestine may be *primary* in children chiefly, and the mesenteric glands are also involved and caseous. There is diarrhœa, with colic and often some degree of fever, and palpation, especially over the cæcal region, is painful. *Secondary* ulceration is very common in cases of pulmonary tuberculosis. The ileum, cæcum, and colon are specially involved, although the disease may be very widespread. The solitary glands and Peyer's patches are infected by tubercle nodules, and the ulcers tend to run in the transverse axis of the bowel, especially in the jejunum, cæcum and colon, while in the ileum they may be more ovoid in shape. The edges and base of the ulcers are infiltrated, and they often involve the muscular coat, while tubercle nodules are generally seen on the corresponding peritoneal surface. Tubercular peritonitis, tabes mesenterica, and, eventually, waxy

degeneration are frequently associated. Perforation is not uncommon, while healing generally implies some stenosis.

Ileo-cæcal tuberculosis often involves the appendix, and it may be mistaken for non-tubercular appendicitis. The definite tumour, often palpable and tender, is accompanied by periodic attacks of diarrhœa. The diagnosis of the condition is important, as it has been proved by Professor Caird to be one which can be operated on with every chance of cure.

(4) Mucous Colitis

This is a form of very chronic colitis, in which a large amount of mucus is passed by the patient, often as casts of the bowel.

Etiology.—It is commoner in women than in men, and is almost invariably associated with a marked type of neurasthenia.

The **Clinical Features** vary with the severity of the case. Generally attacks of mucous colitis develop from time to time, and the patient, after complaining of abdominal pain and discomfort, passes casts of the bowel, sometimes nearly a foot long, often only in bits, but always with a good deal of tenesmus. The colon is frequently distinctly palpable as a firm, cord-like structure which is tender to the touch. During the days of discomfort, while the pain continues, there is much fretfulness of temper, and sometimes great depression of spirits.

The **Diagnosis** should be directed to the elimination of cancer, or other condition associated with much mucus in the stools.

The **Prognosis** depends on the patient's general health as much as on anything else. The disease may last for months or even years, and give rise to constantly recurring periods of discomfort.

The **Treatment** consists partly in dietetic measures and care for the general health of the sufferer, and partly in an attempt to treat the condition by remedies which probably to some extent exert a local action. Meals should be given at regular hours, and quantity and quality must be studied. The following medicinal remedies are good:—Arsenic, sulphur, and cream of tartar: half a drachm of the last two remedies may be taken together at bedtime. Washing out the colon has

also been recommended, and is well worth a trial, and castor oil is another valuable remedy. During the paroxysms of pain, hot applications may require to be applied to the abdomen. In cases which defy all other treatment it may be necessary to perform appendicostomy and irrigate the colon through the opening.

XII. APPENDICITIS

The appendix is about three inches in length; it is a blind tube, and its walls have the same structure as the caecu It possesses lymphoid tissue and many mucous glands, and it has distinct mesentery, in which run an artery and a vein. The appendix usually lies just under the lower end of the ileum, pointing towards the umbilicus, but it may be situated under the caecum or hang over the pelvic brim, while it sometimes contracts adhesions with neighbouring structures. In about 12 per cent of c s of appendicitis there is a concretion in the lumen of the tube, either faec l, or more rarely a grape-seed, cherry-stone, or other small foreign body. Sometimes the appendix is thickened, and the lumen is often partially constricted near its b se or even obliterated, while a cystic distension of the tube beyond the constriction is usually associated. Ulceration is not uncommon, sometimes tubercular or typhoid. Suppuration is frequent in the cystic end of the appendix, the most virulent organisms present being the Bacillus coli communis, the Streptococcus pyogenes, and the Staphylococcus pyogenes aureus. Perforation may rapidly occur in what are called fulminating cases, and as a result either the general peritoneal sac may be infected or a localised abscess develops intra- or sometimes extra-peritoneal in position. Occasionally an artery or vein is opened into, or pylephlebitis may be set up. Gangrene may also ensue either from the intense local inflammation or from interference with the blood-supply in the mesentery of the appendix. Generally an attack of appendicitis subsides without any of these complications arising, but it leaves some evidence of its presence behind, and recurring attacks are common; on the other hand, complete obliteration of the whole lumen would imply a natural and probably permanent cure.

Etiology.—The cause of appendicitis is still disputed.

It certainly is in many cases due to constipation, although constipation alone is insufficient to produce it. Rheumatism, influenza, and oatmeal have been held responsible, but evidence is lacking. It is more common in the 2nd, 3rd, and, to a less extent, the 4th decades of life.

Clinical Features.—The anxious, flushed face and the drawn-up knees demonstrate the physical agony of the patient. The leading features are pain, sudden and colicky, generally in the right iliac region and increased by deep pressure over M'Burney's point. M'Burney's point is situated where a line joining the umbilicus and right anterior superior spine is crossed by the outer edge of the right rectus abdominis muscle. There is marked immobility of the abdominal wall over the affected region. Between the attacks, or where the condition is more chronic, the pain is dull and aching, but with the attack it becomes sharp and lancinating. The pain is possibly due in part to peristalsis of the appendix itself. Vomiting is an early feature, and is persistent during the attack. Any fluid drunk is generally returned at once, but only in very severe cases, amounting to acute obstruction, does the vomited matter become faecal. The temperature is raised (100°-102° F.), and the pulse, often soft and rapid, is a good index of the acuteness and gravity of an attack. On palpation a tumour-like mass is frequently felt, which is, often at least, due to the protecting abdominal muscles. When an attack is over, this resistance may disappear. There may be irritability of the bladder and sometimes albuminuria. The presence of an excess of indican in the urine has been noted. Where an abscess forms, a leucocytosis will probably develop, but a leucocyte count has not always yielded reliable results. An attack usually passes off in 2 or 3 days, leaving some aching pain behind.

Diagnosis.—*Renal* and *biliary colic*, *salpingitis*, *intussusception*, and *ordinary colic* can hardly be mistaken. Perhaps intussusception is most similar. Remember the red currant discharge from the bowel in the latter condition, and the fact that children usually suffer.

The pain localised to the right iliac region and the vomiting are practically constant symptoms of appendicitis, the former only disappearing, and that but temporarily, if gangrene ensues. Many authorities also place the heightened

temperature amongst the leading symptoms. *Perinephric* and *perityphlitic abscess* may simulate this affection, but perityphlitis is generally of appendicitic origin.

Prognosis.—Most cases recover without operation, and there may be no recurrence. Operative interference is good either within six hours of the commencement of an attack, or between attacks, and in every case of abscess formation operation is the only hope of cure. The death-rate should and would be low for operation cases, were medical men willing to get surgical aid promptly. Sometimes, however, general peritonitis is present from the first, and in certain cases the appendicitis is part of a general colitis, and operation is less likely to be of benefit. Where general peritonitis and gangrene are present, the prognosis is bad.

Treatment.—Ice locally, morphia hypodermically, and enemata of soap and water constitute the best medical treatment, but morphia should be used as seldom as possible, because by soothing pain it may prevent the surgical interference which in some cases of appendicitis can alone save life.

Surgical interference is generally desirable, and is urgent in fulminating cases, and the death-rate for operation in quiescent cases is extremely small, and, in fact, should be *nil*.

XIII. INTESTINAL OBSTRUCTION

As the name implies, this is interference with the lumen of the bowel, and it is usually associated with complete constipation, although sometimes from the site of the obstruction the bowel may empty itself below the stricture, and thus mask the true nature of the condition present.

It may be acute or chronic—terms not always absolutely distinct, because a chronic obstruction may become acute; and under slightly different circumstances the same cause may act in one case by producing acute, in another case chronic obstruction.

Etiology.—The obstruction may be (1) Extramural, (2) Mural, and (3) Intramural.

1. *Extramural Causes.*—**A.** Strangulation of the bowel

may be induced by bands or adhesions; by passing through apertures in the omentum, mesentery, or diaphragm; by passing into hernial sacs; by being caught by bands formed by persistence of foetal structures, such as Meckel's diverticulum, which is often connected with the umbilicus; by compression by tumours; and by peritoneal adhesions as the result of an appendicectomy, or some other abdominal operation in which the peritoneum has been exposed. The cause of the strangulation may be actual compression of the bowel or interference with the blood-supply to the part. This somewhat complex group includes a big percentage of all cases of acute obstruction, probably approximating 40 per cent.

B. Volvulus, twist, or kink is another cause, and is most generally met with at the sigmoid flexure, although twists may occur elsewhere.

C. Intussusception, or invagination of one part of the bowel into another, is not uncommon in children. It occurs most frequently at or near the ileo-caecal valve, that part of the small intestine being invaginated into the caecum. The invagination rapidly increases until the mesenteric blood-supply of the bowel is interfered with, and intense congestion and inflammatory oedema result, and if this is not relieved, gangrene will ensue. The invaginated bowel has even been known to reach the rectum, and to be palpable with the examining finger, but this would probably imply that the colon was invaginated into the colon or rectum. Occasionally sloughing occurs, and the invaginated part may be passed by the rectum, when natural cure might conceivably result, but this is very rare. In a case of the author's, a tumour at the ileo-caecal valve, and primarily of the ileum, formed the starting-point and exciting cause of the intussusception. A classic sign of intussusception is the presence of a red currant jelly-like discharge per rectum. It should be remembered that in children especially, but sometimes in adults, invaginations are found post-mortem which are easily reduced with the fingers, and which probably occurred *in articulo mortis*.

2. *Mural Causes.*—**A.** One important group of these is apt to be overlooked, but is none the less a common form of acute obstruction. It is the paralysis of the gut, which inevitably occurs as a result of very intense inflammation, whether there is actual suppurative infiltration of the wall of

the gut or not. In appendicitis the obstruction is often due to this cause.

P Strictures of the gut or tumours of the wall of the gut are more common causes of chronic obstruction, although at some stage in the affection the obstruction may become acute. They include rare congenital strictures which call for no detailed description, and cicatricial contraction of the bowel secondary to ulceration, especially tubercular and syphilitic and much more infrequently typhoid and dysenteric. The tumours are sometimes simple, and include papillomata, adenomata, fibromata, and lipomata, but far more commonly they are malignant and are chiefly carcinomata. Carcinomatous tumours are generally situated at the sigmoid flexure, although they may be found at the hepatic or splenic flexures or elsewhere. Tumours of this description occur as a rule after middle life.

3. *Intramural Causes.*—**A.** There are many foreign bodies which may cause obstruction, and these include coins, fruit-stones, gall-stones, sharp-pointed objects such as pins, nails, hooks, and many indigestible substances which may accumulate until a large mass is formed. It is remarkable how many large, pointed, or sharp objects, if accidentally swallowed, are readily passed per rectum, the best examples being pins, needles, and artificial teeth which, if not too large, may be evacuated without much delay. Should, however, a blockage occur from such an object, the caecum is the favourite site, although it may be at any part of the intestine: in the case of gall-stones it is often in the ileum.

Enteroliths or intestinal concretions are due to many causes. They may consist mainly of phosphate of lime and magnesia: they may be formed in part of husks of grain, of hair, or vegetable fibre, or they may be faecal masses impregnated with lime salts. Sometimes enteroliths are formed of some medicinal agent swallowed by the patient, such as salts of magnesium or bismuth. Enteroliths are, however, distinctly rare.

B. Faecal impaction is a frequent cause of obstruction, which is generally chronic, although it may become almost acute in the severity of pain and suffering to which it gives rise. A constipated habit, feeble muscular action of the intestines, and very probably badly masticated and unsuitable

food, are amongst the commoner causes. An elongated or misplaced sigmoid greatly favours faecal accumulations. Above these more chronic obstructions the bowel is usually distended with much gas. Faecal masses should be definitely palpable, and therefore the condition is easily recognised; it is most common in women, and often occurs between the ages of twenty and thirty.

Clinical Features of Acute Obstruction.—The attack itself is sudden, although there may be a history of chronic obstruction for some time previously. It sets in with acute and very severe abdominal pain, often felt at or near the umbilicus. The pain is at first colicky or spasmodic, then continuous, but with periods of greatly increased agony. Vomiting soon commences, first of stomach contents, then of bile, and later it is of a faecal character and is very persistent. There is generally absolute constipation, although the bowel may empty itself from the site of obstruction downwards, owing to peristalsis starting from the obstruction. The vomiting, it should be stated, is the result of reflex peristalsis starting from the site of the obstruction. Abdominal distension is not marked at first, but soon develops; it is due to gas, and the degree of such distension depends in part on the site of the obstruction, being usually greater when it is situated low down in the intestine. With the distension, local tenderness to touch greatly increases, and the slightest pressure may cause intense agony. At this period the patient's appearance and attitude are very typical. His face is anxious and pinched, cold clammy sweat is seen on his brow, his tongue is covered with dry, brownish fur, and his thirst is intense. He drinks incessantly if permitted, but at once vomits up all he has taken. The urine is often greatly reduced in amount. The temperature is not necessarily above the normal, but the pulse becomes rapid and feeble, and death may result from collapse if he is not relieved within three or four days. There may be a definite leucocytosis. The clinical picture closely corresponds with that of acute peritonitis, and peritonitis may be found post-mortem.

Clinical Features of Chronic Obstruction. There is no sudden onset, but rather a history of long-standing and very intractable constipation, with colicky pain from time to time and a good deal of general discomfort. There is frequently marked

dilatation of the intestine above the obstruction, and visible peristaltic movements may be made out, and can often be induced by the application of a cold hand to the abdominal wall. There may be vomiting, but it is not typical of this kind of obstruction. There is practically always more or less abdominal distension, much increased if no movement of the bowels has occurred for some days. Sometimes attacks of diarrhœa supervene, the bowel emptying itself below the obstruction, or tunnelling of faecal masses may occur. The tongue is furred, and there is a good deal of general malaise, but no increased temperature. The nature of the obstruction can sometimes be recognised by palpation and percussion, and frequently most valuable information as to the part of bowel which is distended, and which is almost always above the stricture, is obtained by carefully combined percussion and auscultation so as to trace the continuity of the distended bowel. The method is described under the heading of gastric dilatation (p. 248). Much help may be obtained from examining the motions. In stricture near the anus the motions are often small in calibre or flattened, although a big prostate may be alone responsible for this phenomenon. Again, the presence of blood, mucus, and shreds of tumour, etc., should be carefully noted. Not infrequently a chronic obstruction becomes acute, and then the symptoms mentioned under acute obstruction appear, and death rapidly follows if relief is not obtained: in any case the patient's health suffers from a comparatively slight chronic obstruction, and malnutrition, with its train of results, will ensue.

Diagnosis.—We must try to decide in a case of obstruction both where it is and what it is due to. Search first for a hernia, and then most thoroughly examine the patient's abdomen by careful inspection, palpation, percussion, and auscultation, remembering the importance of finding a localised board-like resistance or even a slight diminution in movement of the abdominal wall to the hand; and insist upon a careful rectal and, when necessary, a vaginal examination. Sometimes a ladder-like pattern of the intestinal coils, as described by Professor Wyllie, may be noted through the abdominal wall, but this is much more common in chronic than in acute cases. The site of the pain is always suggestive, and tenderness on palpation is often localised. Much information is certainly obtained by

one of the common methods of treatment, namely, the injection per rectum of large quantities of fluid, and noting the amount which can be introduced. Careful percussion and auscultation before and after big injections of water teach a great deal, and sometimes the rectum has been inflated with carbonic acid gas (tartaric acid and bicarbonate of soda) for similar diagnostic purposes.

Often with information which is not too definite we have to proceed at once to operative interference, but whenever possible we should endeavour to find out beforehand the cause of the obstruction. In very young children intussusception is relatively common, and we can often feel the sausage-like tumour through the abdominal wall, while blood-stained mucus is very frequently passed per rectum (in quite $\frac{3}{4}$ths of all cases). An appendicitis is recognised by the localised pain and the fairly typical clinical history of the case. Tumours and faecal obstructions are sometimes palpable.

The diagnosis between acute obstruction and other conditions is very important, but often very difficult.

(1) *Appendicitis* at a certain stage may lead to acute obstruction if not operated upon or relieved by treatment, but to begin with, it is not the same thing. In appendicitis there is the pain felt at M'Burney's point, while the vomiting very rarely becomes faecal—not indeed until the condition has actually become one of acute obstruction from paralysis of the bowel.

(2) *Haemorrhagic pancreatitis* is very difficult to diagnose: the symptoms are the same as in acute obstruction, but the pain would probably, in the first instance, be referable to the region of the pancreas and the lesser omental sac. Local tender points on the abdomen are common, but the pancreas seems to be the chief site of pain.

(3) *Peritonitis* is often closely associated with acute obstruction, and when, as the result of peritonitis, the bowel is paralysed, it is in fact a form of acute obstruction, although there may be no actual narrowing, but rather a huge distension of the affected bowel. In peritonitis, however, the temperature is nearly always raised, the pain is much more continuous and less colic-like, and in general peritonitis the whole abdominal wall is board-like and motionless.

The **Prognosis** varies greatly with the nature of each case. An acute obstruction is always serious until relief is obtained

by operation or other means, but many cases are necessarily fatal, and others are only saved by very prompt measures. The onset of gangrene is always grave, and death occurs in most cases. Chronic cases of obstruction may last for years, and even where a cancerous stricture is inoperable, as it too often is, the patient may live for months.

The **Treatment** must also depend a good deal on the nature of the obstruction. In many acute cases the intervention of the surgeon is imperative, and that without delay. It is difficult without operation to reduce a strangulated hernia, and it is specially difficult to undo kinks and to rectify an intussusception. Inflating the bowel with air or fluid, best done while the patient is under an anaesthetic, has sometimes succeeded in cases of volvulus or intussusception: it is most likely to succeed in children, but too long delay is always dangerous, and surgical aid should be sought early. The awful nausea and vomiting is greatly relieved by washing out the stomach, and Kussmaul, to whom this most excellent suggestion is due, recommends it for its effect in reducing the painful abdominal distension. Pain should be kept in check by opium (morphia, gr. $\frac{1}{8}$-$\frac{1}{4}$, hypodermically), often combined with atropine (gr. $\frac{1}{100}$-$\frac{1}{80}$), but it must not be forgotten that pain is a useful symptom, and its absence, artificially brought about, may cause a fatal delay in performing laparotomy. Certainly if enemata and inflation with air fail, they should not be too often repeated. From $\frac{1}{4}$ to $\frac{1}{2}$ of all cases of intussusception may be relieved by inflation, but it must be tried *early*—if delayed, inflation is more apt to cause rupture of the bowel, and much less likely to be successful. The death rate after operation for acute obstruction is still very high, certainly well over 50 per cent, but then many cases are seen too late, and in a proportion of them no operation could do any good. Once the bowel is gangrenous, or a definite general peritonitis has developed, operation is not nearly so successful.

The enormous distension of the abdomen may be relieved by tapping with a fine needle through the abdominal wall, or by passing a long rectal tube up the bowel. Feeding the patient is usually a work of difficulty, and naturally we cannot in an acute case feed by the stomach. Small nutrient enemata are advisable: merely give little bits of ice by the mouth to allay thirst and help to prevent vomiting.

In cases of chronic obstruction a great deal of benefit results from giving opium with atropine by the mouth or hypodermically, and soon afterwards large soap and water enemata, which may need to be repeated. For faecal impaction the faecal masses, if in the rectum, may require to be picked out with a spoon; but in most cases they are situated too high up, often in the caecum and colon, and then a preliminary enema of olive oil (5 6-12), combined in certain cases with half an ounce of turpentine, should be administered, and about half an hour after, a big soap and water enema. This may require to be repeated several times before the patient is completely cured.

It is never prudent in chronic obstruction to give purgatives too frequently: if one dose fails, it is preferable to resort to enemata. In certain cases where a tumour is present an artificial anus or a short circuit may be made so as to cut out the narrowed part of the bowel.

XIV. INFARCTION OF BOWEL

This is due to an embolus, most commonly from the heart in a case of ulcerative endocarditis, entering a branch of one of the mesenteric arteries. The result is gangrene of the loop of bowel affected. There are all the clinical features of acute obstruction with peritonitis, and the pain, vomiting, and rapid collapse are very marked. The bowel affected is found to be infiltrated with blood; there is no possibility of cure save by operative interference, at best a very slender hope.

Thrombosis of a mesenteric vein may cause the same results, although not quite so acutely.

XV. ENTEROPTOSIS

Glénard's Disease

Displacement of various abdominal organs, often the result of lengthening of the mesenteric or other attachments. The kidneys most frequently suffer, and movable kidney and float-

ing kidney are described separately. The liver and spleen are also sometimes displaced downwards, while the stomach, often as the result of tight lacing, may be considerably altered in position. Similarly, the colon may have a long mesentery, and sometimes intestinal obstruction is due to a kink occurring, owing to this long mesenteric attachment.

The **Clinical Features** vary considerably, but neurasthenia is not infrequently present in cases of enteroptosis.

The **Treatment** depends much on the organs involved and the degree of trouble which the displacement causes the patient. As noted elsewhere, the kidneys may be stitched back to the posterior abdominal wall, but it is more difficult by any abdominal operation to keep the liver or stomach in its proper position. Bandages and pads are often helpful, but the condition may cause much trouble to patient and physician alike.

XVI. DISEASES OF THE LIVER

(1) Jaundice

Jaundice used to be divided into obstructive or hepatogenous, and non-obstructive or haematogenous; but it is doubtful whether all forms of jaundice are not really obstructive in origin, because, where a poison such as tolylendiamin is experimentally given, there is, it is believed, an increased pressure in the bile-ducts, due largely to an alteration in the viscidity of the bile itself. The terms which we shall adopt are: **A.** Hepatogenous Jaundice, which includes all the forms previously grouped under Obstructive Jaundice; and **B.** Toxaemic, Haemolytic, or Haemohepatogenous Jaundice, which includes all the forms previously described as non-obstructive.

A. Hepatogenous Jaundice

Etiology.—There are many causes of jaundice which can truly be styled obstructive, and the obstruction may be situated inside or outside the bile-ducts.

As examples of obstruction within the ducts may be mentioned gall-stones, cancer, foreign bodies, worms, inspissation of bile, and other less frequent causes; catarrhal inflamma-

tion of the bile-duct close to its entrance into the duodenum is the cause of the mild attacks of jaundice in so many cases of gastro-duodenal catarrh.

As examples of obstruction of the bile-ducts by pressure may be mentioned tumours of all kinds, peritoneal bands or adhesions, and cicatrices as in syphilitic cirrhosis, which may occlude the ductus choledochus, or one of its larger branches.

There are also cases in which jaundice has been induced by anger, mental shock, or emotional excitement, and it has been supposed that the obstruction of the bile-ducts is, in these cases, due to spasm.

The **Clinical Features** of an ordinary case of obstructive jaundice are described under catarrhal jaundice, but in contradistinction to the transient tinging of skin and of urine by the bile, there may, in permanent cases, be very marked features which demand a word of description.

Permanent Jaundice.—The skin becomes dark greenish-yellow in colour, sometimes almost approximating to black, and the urine is deeply stained, the sweat more slightly.

The itching of the skin, often troublesome in catarrhal jaundice, is extremely irritating to the patient, and pruritus of a severe form may be induced, while other skin eruptions, such as nettle-rash and occur.

In the urine there is nearly always albumin present as well as bile, while in the *Alimentary System* the stools obstructive jaundice are absolutely colourless. Constipation, due to the loss of one of nature's best purgative agents, is the rule.

In the *Circulatory System* the slowing of the heart's action mentioned under catarrhal jaundice, becomes very marked where the jaundice is permanent, and is probably due to the action of bile salts on the cardiac centres.

Petechial haemorrhages are extremely common, and larger extravasations may also be present, while epistaxis occasionally occurs. There is often a difficulty in arresting haemorrhage during surgical operations.

In the *Nervous System* there is, in the first instance, irritability, and later great mental depression. Eventually definite evidence of poisoning sets in, the patient becoming delirious, or suffering from convulsions.

The **Prognosis** is always unfavourable, unless it is possible

by operative treatment to remove obstructions such as gall stones.

B. Toxaemic Haemolytic, or Haemohepatogenous Jaundice

This is always of toxaemic nature, and seems to depend on the rapid destruction of blood throwing a great excess of blood pigment into the liver, and thus causing an increase in the viscidity of the bile.

Etiology.—The common causes of this form of jaundice are (1) snake poisons, tolylendiamin, and the action of phosphorus and arsenic; (2) toxaemic jaundice may be present in certain fevers, and especially malarial fevers, yellow fever, sometimes scarlatina, typhus, etc., and (3) toxaemic jaundice may occur in certain rare infective conditions, of which some are, and some are not, epidemic, and these include acute yellow atrophy, Weil's disease, and other kindred conditions.

The **Clinical Features** of toxaemic jaundice differ somewhat from those of obstructive jaundice. There may be less pigmentation of the skin and of the urine, and there is generally some bile in the stools. On the other hand, there are always much more marked evidences of toxaemia, with fever, delirium, sometimes convulsions, and often suppression of urine, while numerous petechial haemorrhages may be noted.

2. Catarrhal Jaundice

Jaundice due to the blockage of the orifice of the ductus choledochus is secondary to catarrh of the duodenum which has extended from the stomach.

Etiology.—Gastro-duodenal catarrh may be secondary to many continued fevers, but is also a common result of any indiscretion in diet, and it is sometimes epidemic. Anger has been known to produce an attack. It may follow the passage of a gall-stone down the ductus choledochus, and it occurs in more chronic form in cases of backward pressure especially affecting the portal circulation.

The **Clinical Features** are those of obstructive jaundice with typical clay-coloured stools; jaundiced skin, conjunctivæ, and soft palate; and with bile in the urine, following on an attack of gastric catarrh. Not infrequently there is no very

definite history of gastro-duodenal disturbance, but close examination may elicit information as to some very indigestible article of diet which has been eaten. The skin may itch, due, it is thought, to the bile acids irritating peripheral nerve endings, and on examination the abdomen is often somewhat swollen, the liver slightly enlarged and tender on firm palpation, and the gall-bladder distended with bile. The pulse is slow, falling to about two-thirds of the rate normal to the individual, and the patient feels languid, sleepy and unfitted for any mental effort. Otherwise the symptoms depend on the gastric catarrh. The coloration gradually fades, and should completely disappear in a few days, although it may persist for 2 or 3 weeks. A longer duration, and the continued absence of bile in the stools, make the physician suspect either gall-stones or malignant disease blocking the common bile-duct.

The **Diagnosis** depends on the recognition of the cause, rather than on the existence of the jaundice. A simple catarrhal jaundice should be unaccompanied by pain or emaciation, and the jaundiced colour rarely becomes greenish, while petechial haemorrhages are not common. *Weil's disease* should be kept in mind: its epidemic character is helpful, differing in this respect from most cases of catarrhal jaundice.

The **Prognosis** is favourable in a simple case, and recovery should not be delayed over a few weeks at the longest.

The **Treatment** is directed to allaying gastro-duodenal catarrh. Careful dieting, mostly milk, and sedatives, such as bismuth subnitrate (gr. 10-30), and dilute hydrocyanic acid (♏ 3) in mucilage and water are usually all that is necessary.

Rest in bed and warmth, with a saline purge and a small dose of such cholagogues as calomel (gr. 3-5), euonymin (gr. 2-3), or podophyllin (gr. $\frac{1}{4}$-$\frac{1}{2}$) are beneficial, and sodium phosphate, given as a constituent of a saline draught, is one of the very best remedies. Fomentations, or the application of cold externally, are advisable where there is pain: in most instances fomentations or poultices are preferable.

(3) Icterus Neonatorum

In newly-born children there is common a mild degree of jaundice, which develops about the 2nd or 3rd day, when the urine may be bile-stained and the stools colourless. It is

difficult to offer a satisfactory explanation. It is said to be due to a catarrhal jaundice, which seems unlikely. It may be due to diminished tension in the portal vessels, a not very comprehensible explanation, or to destruction of the enormous excess of red blood corpuscles found in the child's blood after birth.

A much more severe type may follow congenital stricture of the ductus choledochus, syphilitic cirrhosis, and septic poisoning from the stump of the umbilical cord. In the latter case there is not infrequently haemorrhage from the stump of the cord.

(4) Acute Yellow atrophy of the liver (Icterus Gravis)

A form of malignant jaundice in which there is rapid destruction of liver cells and diminution in size of the organ, and in which the chief clinical symptoms are cerebral.

Etiology.—A rare disease, most common in women between 20 and 40, and certainly closely related to pregnancy. It is possible that common cirrhosis of the liver has a causal connection with this disease. Organisms and bacilli have been described in several cases, but their rôle is not yet clearly made out. The disease is almost certainly of toxic origin, although whether or not due to a specific bacillus, situated locally, is a matter of doubt.

Pathological Anatomy.—The liver is small, often under two pounds in weight, and its capsule is wrinkled. It is soft and flabby, and it is of a dark green colour. The typical patches, some of them dark red, others bright yellow, are best seen on section. In the yellow or more recent parts, there is marked necrosis of the liver cells, many of which are reduced to an almost unrecognisable, granular-looking condition. In the orange or red patches secondary fibrous changes have replaced the necrotic cells, the condition being really a cirrhosis, and the areas so affected have shrunk. Crystals of leucin and tyrosin may be seen in the liver, and may be obtained from the urine. Haemorrhages are common, as they are in all forms of toxic jaundice, and are sometimes subcutaneous, sometimes subserous. There is marked bile-staining of the internal organs, and the spleen is generally enlarged, while

the kidneys may show advanced catarrhal changes in the epithelium of the convoluted tubules.

Clinical Features.—The disease begins like an ordinary catarrhal jaundice with vomiting, but later head symptoms develop, sometimes convulsions, generally delirium, and these are followed eventually by coma. With these there is very deep jaundice, and subcutaneous haemorrhages appear, while the urine is albuminous, of course bile-stained, and contains leucin and tyrosin, got in a majority of cases after evaporation. Leucin occurs in yellowish spherical crystals, with both faint radial and also concentric striation, and the tyrosin in needle-shaped crystals, which appear in sheaf-like bundles. There is not necessarily much pyrexia—possibly there may be none at all—but the pulse is rapid and feeble, and the patient is obviously *in extremis*. The spleen is found to be enlarged, and on examination of the liver the percussion dulness anteriorly may be greatly reduced or almost absent.

As a rule the case rapidly goes downhill, and death follows in about a week.

Diagnosis.—Too much stress must not be laid on finding leucin and tyrosin in the urine, because they may be present in other diseases, although rarely, and they may be absent in acute yellow atrophy. An ordinary *malignant or permanent jaundice* is always associated with head symptoms, but here they are specially severe, and if there be diminution of hepatic dulness with leucin and tyrosin in the urine, we need have no doubt of the diagnosis. *Phosphorus poisoning* most closely resembles acute yellow atrophy with its clinical features of sickness, diminution in liver dulness, and the presence of head symptoms, but there should be a history, and leucin and tyrosin are rarely present.

The **Prognosis** is very bad, although a few apparently authentic cases of the disease have recovered.

The **Treatment** is directed to the relief of symptoms, and to supporting the patient's strength.

(5) Cholelithiasis: Gall-stones

Gall-stones are composed of (1) a nucleus which may be, and often is, inspissated bile pigment, or bacteria which have probably reached the gall-bladder from the duodenum, and

which are B. coli communis, the Typhoid bacillus, the Pneumococcus, and other organisms; and (2) stratified layers of cholesterin and lime formed round the nucleus, although other salts may also be present in smaller proportions. Cholesterin and lime are found in excess in connection with inflammation of mucous membranes, and although cholesterin is usually retained in solution in bile even when in excess, the presence of one or more "nuclei" may lead to its deposition, and so, in time, to the formation of gall-stones. The glycocholate and taurocholate of sodium, present in bile, help to keep cholesterin in solution. Their deficiency may mean its precipitation and this may be due in part to want of sufficient nitrogenous diet. Naunyn has conducted much research in connection with this subject, and has fairly definitely proved that fatty degeneration of cells in the mucosa may lead to excessive formation of cholesterin, and possibly a cholesterin which is specially apt to be precipitated. Where a part or the whole of the gall-bladder is absolutely shut off and contains no bile, but only mucus, the gall-stones are almost pure cholesterin, whatever the nucleus may prove to be.

Etiology.—There is a close relationship between micro-organisms and gall-stones, and it seems probable that microbes in the gall-bladder constitute the nuclei in many cases. In this connection it is worth remembering that such micro-organisms as the typhoid bacillus can live in the gall-bladder for months, and that typhoid fever is a frequent antecedent of gall-stones. Gall-stones are, however, most common in women. In the writer's collection of nearly 100 specimens more than three-quarters were obtained from women. They are certainly most frequent after middle life, and in women who have borne children. Tight lacing is held to be a predisposing factor, and this seems very probable. A sedentary life is certainly favourable to their development, and, in fact, anything as regards the habits or work of the patient which favours stagnation of bile in the gall-bladder or bile-ducts. Persons fed chiefly on carbohydrates appear to be more predisposed to gall-stones, which are less common in countries where meat is eaten as the staple diet. Perhaps their presence in gouty persons may be due to the limited meat dietary and the excess of carbohydrates. They are frequently met with in cases of cancer of the bile-ducts, although the question

arises as to whether the gall-stones are always the result of the malignant stricture of the bile-ducts and not sometimes the cause of the tumour.

Gall-stones—their Characters.—They vary greatly in size from minute calculi, which are composed mostly of inspissated bile, and are soft and very numerous, to large stones which occur singly and may be several inches in length. When there are several stones in the gall-bladder which can rub against each other, they are faceted. Occasionally several stones occur without being faceted, but this is rare. The colour depends much on the amount of bile pigment, and large stones often contain a considerable proportion of lime salts. Stones may be found in the bile-ducts which perhaps have formed there, or may have originated in the gall-bladder. Sometimes stones develop in the bile-ducts in the liver substance; these are generally small and ovoid, and mostly formed of bile pigment.

Clinical Features.—In many cases gall-stones are discovered after death which have never caused any symptoms during life, and the gall-bladder may even be full of gall-stones, forming a palpable tumour without producing one single attack of biliary colic. It is desirable, however, to describe a typical attack of biliary colic, and then to consider the other possible results of gall-stones.

Biliary colic is due to one or more gall-stones travelling from the gall-bladder along the cystic duct, and either passing down the common bile-duct, passing back again into the gall-bladder, or becoming temporarily or permanently encysted somewhere. The pain is intense and agonising, and occurs in paroxysms, usually referred to the region of the gall-bladder, and radiating to the right shoulder, often accompanied by vomiting, profuse cold sweating and pallor, and generally followed by an attack of jaundice due to the catarrhal inflammation consequent on irritation caused by the stone. There is aching pain felt after the attack has subsided, and the gall-bladder may be distended and painful on pressure. A temperature of 102°–103° F., or even higher, may be associated with the attack, but there is greater pyrexia if a cholecystitis develops. The presence of a definite attack of jaundice depends on whether the common bile-duct is involved in the journeyings of the stone, and will likely be absent when the cystic duct is alone affected. There is sometimes a transient albuminuria. It is difficult to offer a

good reason for the onset of an attack of biliary colic. Probably exercise, a sudden strain, and perhaps indigestion may all aid in starting a gall-stone on its wanderings, but, on the other hand, attacks are not infrequently nocturnal. The motions should be inspected after an attack, in case the gall-stone or stones have been passed by the bowel. Sometimes many stones are passed, either in one motion or in successive motions. The appearance of a big stone in the stools implies that it has ulcerated into the bowel. Should the stone be impacted in the cystic duct, the gall-bladder may greatly enlarge from distension with mucus and may be readily palpated. If the stone is impacted in the common bile-duct, not merely is the gall-bladder enlarged, but the liver also becomes swollen and painful, the bile-ducts being distended with bile. Permanent jaundice may develop, or the stone may not completely obstruct the duct and may permit of the passage of some bile. Occasionally the gall-bladder becomes enormously enlarged, generally after blockage of the cystic duct, forming a huge abdominal tumour. In such a case the gall-bladder may contain mucus or even pus, with very little, or no, bile.

It should not be forgotten that biliary colic may be very severe, as just described, or it may be comparatively mild, or it may be almost unrecognised by the patient as an attack at all. Generally, however, one attack is the precursor of others, and a number of attacks should make the diagnosis quite clear.

If a stone is present in the liver, it causes more marked rigors.

The Results of Gall-stones.—One of the chief results is certainly suppuration. It may occur in the gall-bladder, causing a cholecystitis, or it may occur in the common bile-duct and its branches in the liver. In the gall-bladder there is risk of rupture into the peritoneum: in the bile-ducts, when suppuration ensues, there are rigors, enlargement of the spleen, and eventually the rapid development of pyaemic phenomena.

As already noted, impaction of a gall-stone in the common bile-duct may lead to permanent jaundice; in other cases the calculus or calculi may ulcerate into the intestine or elsewhere. Fistulous openings are sometimes met with into the duodenum, rarely into the stomach, and sometimes into the peritoneal sac, while there may be communications with the portal vein or between the gall-bladder and the bile-ducts. Occasionally

fistulous openings are external, and gall-stones may be discharged in this way.

Intestinal obstruction may be due to blockage of the bowel with gall-stones.

Abscess of the liver is a rare result of gall-stones, and it implies the presence of suppurative organisms, and in a cholecystitis the pus has been known to perforate into the liver substance and set up a liver abscess.

A form of cirrhosis of the liver is due to permanent obstruction of the bile-ducts, and hence gall-stones may, although rarely, be responsible for this condition.

We have left until the last the important bearing of gall-stones on cancer. Almost certainly cancer of bile-ducts is in many cases due to the irritation of gall-stones, although stasis of bile, the result of a cancerous stricture, may also lead to the formation of calculi. The gall-bladder is not an uncommon site for primary carcinoma, and in about 95 per cent of these cases gall-stones are present.

Diagnosis.—There are a number of conditions in which abdominal pain occurs, but none are easily confused with biliary colic. In biliary colic the pain is local, shoots typically to the right shoulder, is accompanied by vomiting, and is associated with tenderness over the gall-bladder, which is generally distended and palpable. In *renal colic* the pain shoots down to the testicle in males, there is no palpable gall-bladder, and blood often appears in the urine. In *appendicitis* the pain is felt at M'Burney's point, and not over the gall-bladder, and there is generally constipation. The X-rays are not a likely means of recognising gall-stones because cholesterin does not obstruct the rays.

The **Prognosis** depends much on circumstances. An impacted gall-stone may be grave if the condition necessitates a serious surgical operation. Suppuration renders a case more anxious, and any suspicion of cancer is, of course, bad. Operation is much more hopeful than it used to be, and one attack of biliary colic generally means that other attacks are probable unless something radical can be done.

Treatment.—For the attack of colic at once apply fomentations or linseed poultices, or order the patient to take a hot bath, and aid in relaxing the spasm by giving morphia (gr. ¼–½) hypodermically, or resort to general chloroform anæsthesia.

Giving the patient large draughts of warm water to drink is sometimes useful, although they may be vomited.

Between the attacks the treatment is much less clear and defined. Can we dissolve the calculi present in the gall-bladder if any are there, and if not, should an operation be performed? Gall-stones certainly are the result of stasis of bile, and therefore a free flow of bile should be encouraged. Sodium phosphate, in drachm doses thrice daily, is found of great service in many cases, and nearly all forms of purgative saline waters are good. Gall-stones are, to a certain extent, soluble in oil, and olive oil (or salad oil) is sometimes taken by the mouth in the hope that, somehow or other, it may help in dissolving the gall-stones. Patients do improve occasionally under this treatment, but probably not on account of it alone. Abdominal massage over the liver has often been recommended. Many spas are of value, and especially Carlsbad, which has a great reputation for the treatment of gall-stones. Vichy and Nenenahr are also much favoured. Plenty of exercise and a careful dietary are necessary for individuals who are subject to biliary calculi.

There is no special danger in performing cholecystotomy nowadays, further than the risks accompanying any abdominal operation, so that if no relief follows medical treatment, and there is no doubt about the existence of gall-stones, the operation can be recommended with confidence. Stones in the cystic duct are easily removed as well as those which may be in the gall-bladder. Where there is suppuration (cholecystitis) operation and draining the gall-bladder are necessary.

Where the stone is in the common bile-duct, it may be crushed between the fingers or between the blades of a pair of forceps guarded with gauze, or it may be needled, or, lastly, it may be removed by incising the duct and stitching up the opening made.

Should it be found impossible to remove the obstruction from the cystic duct, the gall-bladder should be connected with the bowel (cholecyst-enterostomy). When there is a calculus in the liver, it is often more difficult to reach, and the operation is proportionately more dangerous.

If the patient suffers much from the itching of jaundice, he may sponge the skin with a solution of chloral hydrate 7.4 and liquor ammonii acetatis (5 2) in 10 ounces of water, or he may

use a dusting powder of camphor (5 1½), oxide of zinc (5 ½), and starch (5 1), as recommended by M'Call Anderson.

(6) Active Congestion or Hyperaemia of the Liver

In a slight degree it is physiological, and is the result of an ordinary meal, while in a more marked degree it follows the ingestion of too much food, irritating articles of food, and certainly alcohol in excess. In alcoholic subjects pain over the liver is very suggestive. In tropical countries, and probably as a part of many continued fevers, especially in tropical countries, there is apt to be hepatic hyperaemia. Where there is a sudden arrest of haemorrhage, as in profuse menstruation or in patients suffering from haemorrhoids, pain over the liver is thought by many authorities to indicate hyperaemia of that organ.

The **Treatment** consists in a careful and simple dietary, keeping the bowels open, and complete abstinence from alcohol.

(7) Passive Congestion or Passive Hyperaemia of the Liver

This is the result of backward pressure in the hepatic vein and its tributaries, and is generally due to cardiac and pulmonary disease. Mitral stenosis and incompetence among cardiac, and emphysema and cirrhosis of the lung among pulmonary diseases, together with intrathoracic tumours, are the chief causal conditions.

The liver is described by the pathological term "nutmeg," and the symptoms are those of backward pressure in the portal circulation. Nutmeg liver may lead to cyanotic atrophy in cases of long standing.

The **Clinical Features** have been described under the results of backward pressure in heart disease of which they form a part. They include chronic gastric catarrh, sometimes with haematemesis, intestinal dyspepsia, enlargement and, later, cyanotic atrophy of the spleen and ascites. As regards the liver, a slight jaundiced tinge is common, with discomfort and sometimes actual pain, while there may be enlargement of the liver, or in old-standing cases actual atrophy. Sometimes pulsating liver is noted, but it is rare. There is often bile in

the urine, and the stools may be somewhat clay-coloured. The spleen may be enlarged.

The **Treatment** consists in relieving the portal circulation, and in endeavouring to remove or alleviate the cause of the backward pressure whether cardiac or pulmonary. Sometimes a haematemesis diminishes the portal engorgement, but saline purgatives are especially useful, given in concentrated solution warm before breakfast. It is rarely desirable to bleed from the liver, although this has actually been done.

(8) Cirrhosis of the Liver

There are several different forms of this disease.

(*a*) Common Cirrhosis of the liver, or Alcoholic Cirrhosis, or Hobnailed Liver, is generally the result of alcohol, although other toxic agents must in certain cases play an important part.

(*b*) Hypertrophic Cirrhosis is believed to be due to a toxin involving the bile-ducts. It is doubtful whether this form of cirrhosis is not in reality a type of common cirrhosis.

(*c*) Cyanotic Atrophy, or cirrhosis due to long-standing engorgement of the hepatic veins.

(*d*) Capsular Cirrhosis, in which the fibrous tissue spreads into the liver substance from Glisson's capsule; it is generally secondary to perihepatitis.

(*e*) Syphilitic Cirrhosis, in which individual cells may be surrounded by connective tissue, although larger strands are also found frequently with gummata.

(*a*) **Common Cirrhosis; Alcoholic Cirrhosis: Gin-Drinker's Liver; Hobnailed Liver.**—In this form of cirrhosis groups of lobules are surrounded by fibrous tissue which spreads along the lines of the medium-sized portal spaces.

Etiology.—The disease is most common in middle life, and is the result of an excess of spirituous rather than of malt liquors. There are cases, however, of cirrhosis in children, in some of whom an alcoholic history may be obtainable, but in others the etiological factor remains uncertain.

Pathological Anatomy.—The liver shows a very typical appearance, and the nobnail-like surface is often palpable during life. On section the lobules are seen to be surrounded by thick bands of connective tissue, and fresh attacks are constantly made on the periphery of each little group of lobules by strands

of connective tissue, gradually diminishing the liver tissue as the disease progresses. A varying amount of fatty change may be seen in the hepatic lobules which are affected, and the liver on section is often tinged with bile. During the earlier stages the liver is of almost normal size, but as the disease advances it may become markedly smaller. The connective-tissue increase interferes greatly with the portal circulation, and as a result there is backward pressure in the organs which send their blood to the portal. The spleen is enlarged and shows chronic venous congestion. The stomach and intestines may show chronic catarrhal changes, and ascites is extremely common.

It is well to remember the accessory channels by means of which portal blood can find its way into the common venous circulation.

(1) By the round and suspensory ligaments of the liver the blood may find its way into the epigastric, mammary, and other neighbouring veins, and in so doing forms the typical *caput Medusae* seen surrounding the umbilicus in these cases.

(2) The œsophageal veins anastomose with the gastric veins at the region of the cardiac orifice, and by this means relief may be obtained for the engorged gastric veins.

(3) The portal veins communicate with the inferior mesenteric veins and form the haemorrhoidal plexus.

(4) The veins of Retzius unite the terminals of the portal veins in the intestines and mesentery with venous radicles belonging to the inferior cava, and a large quantity of blood is conveyed from the engorged portal circulation by means of these vessels.

Clinical Features.—For a long time there may be no symptoms and no discomfort experienced by the patient, but sooner or later the backward pressure in the portal system gives rise to definite and typical phenomena. These include chronic gastric catarrh, with nausea and vomiting, bleeding from the stomach and more rarely from the bowels, and the development of ascites preceding in its appearance any general dropsy—in fact, œdema of the legs is only induced when the ascitic effusion increases so much as to interfere, by pressure, with the inferior cava. Haemorrhoids are not uncommon and the *caput Medusae* may be seen round the umbilicus, while the spleen usually enlarges. There is nearly

always a slight degree of jaundice, and very frequently a trace of albumin in the urine, while the chronic gastric catarrh renders the patient's life very miserable.

The *Physical Signs* of the ascitic effusion are referred to under ascites, but the striae seen on the abdominal wall often far exceed both numerically and in depth those found in pregnancy. The fluid is typical dropsical fluid with no special characteristic. Bleeding from the gums, which are often spongy is frequently complained of by the patient.

Very many patients suffer also from definite toxaemic manifestations, usually becoming restless and delirious at night, and eventually passing into a stuporose condition. Death generally closely follows the development of such symptoms.

Diagnosis. The ascites is very suggestive, and if the patient's history includes a predilection for alcoholic excess there is rarely much difficulty. Sometimes the hobnail-like liver is easily felt: more frequently it cannot be determined during life. The possibility of *gummata* of the liver interfering with the portal vein should not be forgotten, and often a history of syphilis aids in the diagnosis of these cases.

The **Prognosis** is unfavourable, although in certain cases remarkable benefit has accrued from repeated tapping, but where there is permanent engorgement of the portal circulation improvement is less likely to occur. A very serious increase in the gravity of the case occurs should a thrombus form in the portal vein.

The **Treatment** consists in tapping ascitic effusions, or sometimes in an attempt to promote collateral circulation between the omentum and the abdominal wall. In certain cases after laparotomy the surgeon strives, by scraping or irritating the peritoneal surfaces, to induce as far as possible occlusion of the peritoneal sac.

b) **Hypertrophic Cirrhosis** or the **Cirrhosis of Hanot.**—Many authorities hold that this is merely a form of common cirrhosis, but it differs from most cases of alcoholic cirrhosis inasmuch as the liver is greatly enlarged and is deeply bile-stained, while jaundice is at least a typical feature in the case.

Etiology.—It occurs in young persons and generally in males. Alcohol plays an unimportant part, but there is no other known etiological factor.

Pathological Anatomy.—The liver is enlarged, the surface is smooth, the colour deep green. The cirrhosis is often described as being rather monolobular than polylobular. It is due to a catarrh of the finer bile capillaries, and the portal vessels escape. There is a remarkable development of recently formed bile passages. The spleen is enlarged, and is often of great size.

Clinical Features.—The disease is a chronic one, beginning generally with a mild form of jaundice, but developing into a permanent jaundice, associated with pyrexia and eventually delirium. There is bile in the urine, but the motions are also well coloured. The liver is painful on pressure, and there is the typical nausea and vomiting of a malignant jaundice. There is no ascites, but haemorrhages are by no means uncommon.

The *Physical Signs* include enlargement of the liver, which may reach to the umbilicus, and the spleen is also enlarged. Death generally results from malignant jaundice, and there is no treatment except that of symptoms.

c) **Cyanotic Atrophy**, or cirrhosis owing to long-standing engorgement of the hepatic veins, results from backward pressure often of cardiac, sometimes of pulmonary origin, and is due to an increase of connective tissue in relationship to the central or hepatic zone of the lobules. The liver is reduced in size, but otherwise there is no very marked characteristic.

(*d*) **Capsular Cirrhosis** or **Perihepatitis.**—There are many diseases in which perihepatitis may occur, as, for example, syphilis or cirrhosis. As a result, fibrous tissue penetrates from the capsule of the liver into its substance, and there may be very marked interference with the portal circulation in advanced cases. There is no treatment of any special value.

(*e*) **Syphilitic Cirrhosis.**—In this disease individual cells may be surrounded by connective tissue, although larger strands, frequently with gummata, may also be met with. Gummata, which have already been referred to under syphilis generally, may cause marked scarring and fissuring of the liver, and associated with the gummata we find a fibrous-tissue increase throughout the organ, radiating from the various gummata, and leading eventually to very marked cicatrisation. Not infrequently waxy degeneration is also present.

The **Clinical Features** depend upon the interference with bile-ducts or blood-vessels while the physical signs are often very suggestive, the fissuring of the liver being sometimes palpable to the hand.

9 Abscess of the Liver

A localised suppuration either in the liver substance, in the blood-vessels, or in the bile passages.

a **Tropical Abscess.** This occurs in cases of dysentery and is the result of the local presence of the dysenteric amoeba in the liver. In most cases there is a solitary abscess, although in others, abscesses may be scattered throughout the organ. The abscess contains greenish-yellow or reddish pus mixed with blood, and the wall is composed of necrosed liver tissue. Where an abscess has existed for some time, a fibrous capsule may form. Other organisms, besides the amoeba, may be present, and the abscess may perforate into the pleura, lung, peritoneum, or elsewhere.

There may be no **Clinical Features**, except hepatic enlargement, until rupture occurs, but often there is pyrexia with local pain. The fever suggests the presence of pus and may be of a hectic type. *Palpation* is painful, but it depends on the position and size of the abscess whether the condition can be recognised by the hand. The abscess is very frequently situated in the right lobe, but only occasionally is fluctuation detected. A leucocytosis should be expected, but is often absent. Where the abscess perforates grave phenomena may result; sometimes the patient expectorates the reddish or greenish viscid pus if perforation occurs into the lung.

The **Diagnosis** is greatly aided by the presence of the amoeba in the stools and the history of dysenteric diarrhoea; the **Treatment** should consist in the opening of the abscess often preceded by an exploratory puncture so as to localise the pus.

b **Suppurative Cholangitis.** Suppuration is noted as a frequent result of gall-stones, and it is termed *Suppurative Cholangitis*. The abscesses are sometimes multiple, or a great and continuous extent of the bile passages may be inflamed, the pus contained in the ducts being bile-stained. Inflamma-

tion of the bile-ducts may, however, result from other causes besides gall-stones; for example, the entrance of parasites, such as the Ascaris lumbricoides, or, more commonly, bacteria which have passed upwards from the duodenum.

The **Clinical Features** include hectic temperature, pain felt over the liver, and often severe jaundice.

The **Prognosis** is very grave, and the **Treatment** must consist in an attempt at draining the abscess.

c **Pylephlebitis Suppurativa.**—This is abscess formation due to infection by the portal vein; the infective agent enters from the intestine or from some organ which sends its blood into the portal circulation. The abscesses are multiple, and, in fact, a large extent of the portal vein in the liver may be distended with pus.

d **Pyaemic Abscess.** In this case the hepatic arteries are the channels by which the infective agent reaches the liver. The abscesses are generally multiple, and may vary in number and size.

In both pyaemic abscess and suppurative pylephlebitis, the liver is enlarged and painful, jaundice is common, and a leucocytosis is almost invariably present, while death generally occurs, and operative interference is hopeless.

Hydatid cysts sometimes suppurate, and parasites or foreign bodies, which may find their way into the bile-ducts (just as the Ascaris lumbricoides already mentioned), may set up a cholangitis. An injury sometimes causes abscess formation.

(10) Fatty Infiltration and Fatty Degeneration of the Liver

These processes are often associated. **Fatty Infiltration** is more marked in obesity; fat is also stored up in the liver cells in chronic alcoholism, and where, in the cachexia of phthisis, there is grave interference with oxidation processes. **Fatty Degeneration** is specially seen in phosphorus poisoning and in acute yellow atrophy of the liver, while it is present in localised parts of the liver in common or alcoholic cirrhosis.

In **Fatty Infiltration** the liver may attain a great size, reaching 8 to 12 pounds in weight, the colour is light yellow, and after section there are oil droplets on the blade of the knife. Curiously enough a liver so affected appears to discharge its

functions in a wonderfully satisfactory manner. The edge of a fatty liver lacks the sharpness present in waxy degeneration, and is smooth, with no nodules or cicatrices suggestive of cancer or syphilis. The skin is peculiarly greasy.

In **Fatty Degeneration** the organ is small.

In many cases, where limited fatty infiltration and degeneration occur together, there are no symptoms.

The **Prognosis** depends on the nature of the case. Fatty infiltration, if the result of obesity, is within the sphere of treatment.

The **Treatment** for obesity is given elsewhere (p. 1[illegible]), but it is, in brief, a dry diet with little fatty or fat-forming food, and a sufficiency of exercise suited to the patient's age and strength.

(11) Waxy or Amyloid Degeneration

The causes of waxy degeneration in other organs determine its appearance in the liver. These are syphilis, suppuration of a prolonged nature, and bone disease. The disease is seen first in the middle zone of the lobules, where it attacks the capillary walls, but it spreads to the interlobular vessels. The liver is of great size, 8 to 14 pounds, and has a smooth sharp edge (unless gummata are present, when cicatrices may cause indentations).

There are no **Clinical Features** referable to the disease in the liver, although the co-existence of enlargement of the spleen and evidence of waxy involvement of the kidneys and intestine help materially in the diagnosis.

The **Prognosis** is unfavourable, and nothing can be done by way of treatment, but the presence of waxy degeneration should make the physician cautious in ordering cod-liver oil and fatty articles of diet.

(12) Hydatid Cyst of the Liver

The liver is the commonest site for a hydatid cyst. The cyst or cysts may be in any part of the organ, but most commonly in the right lobe, and may grow to a large size. If superficial, the hydatid thrill may be obtained on palpation. Sometimes the cyst pushes up the diaphragm and greatly increases the liver dulness upwards, in other cases the rounded

cyst wall is palpable below the edge of the liver. Occasionally cysts suppurate and give rise to the clinical features of a hepatic abscess. Cysts may perforate into pleura or peritoneum, but are generally diagnosed and treated by tapping or stitching the cyst to the wound and draining.

(13) Tumours of the Liver

These may be simple, such as *angiomata*, which are fairly common, and occur as small vascular tumours about the size of a walnut. Malignant tumours are the most frequent, and are generally cancerous and secondary to a tumour elsewhere.

The **Etiology** of malignant tumours is a very difficult problem. The liver is the third organ in the body most commonly the site of such tumours, and in it they are, as already stated, generally secondary. Probably gall-stones play a very important part in the etiology of hepatic cancer, the disease commencing in the gall-bladder or bile-ducts. Injury may possibly be associated in some way with primary hepatic tumours, but it is easy to understand how a primary tumour in the stomach will readily induce secondary growths in the liver.

The Varieties of Malignant Tumour

1. **Cancer** may be **Primary**, but is far more frequently **Secondary**.

Primary Cancer is described as occurring in three forms.

Either *a* a nodular cancer scattered throughout the liver, one of these nodules, which may be larger, being the primary tumour. This form is almost indistinguishable from the common form of secondary cancer. Or (*b*) a massive cancer in which there is a huge, but sharply defined, mass of primary new growth which involves often a large extent of the liver. Or (*c*) a rare form with much cirrhosis, and which may be found in an organ which is not enlarged in size.

Secondary Cancer.—The primary sites are the stomach, in fully a quarter of all cases, the gall-bladder, the rectum, and the cervix uteri in women: women suffer rather more frequently than men (4 to 3).

The liver is often of a huge size, even reaching 20 pounds.

The masses vary greatly, from the size of a small pea to a cocoa-nut, and may be very numerous. The older ones undergo fatty degeneration centrally, and as a result umbilication is well marked in peripheral nodules; this characteristic feature may be felt on palpation. There is often pericapatitis, and not infrequently the diaphragm becomes infiltrated, or the abdominal peritoneum infected, nodules appearing first in the neighbourhood of the umbilicus.

The tumours may press upon bile-ducts and lead to local dilatations, or may interfere with vessels.

2. **Sarcoma** may be **primary** or **secondary**.

Primary Sarcoma is very rare.

Secondary Sarcoma is more frequent. One very marked variety is the so-called melanotic sarcoma, which should rather be termed a cancer. The primary tumour is either in the choroid of the eye or in the skin. Melanotic sarcoma may cause very great enlargement of the liver, and the nodules of new growth have varying shades of pigmentation. In other cases of secondary sarcoma the liver may be infiltrated with many nodules.

The **Clinical Features** vary with the number, size, and position of the tumour nodules. Generally speaking the liver is enlarged, often greatly, and there are evidences of cachexia with gradual, sometimes rapid, emaciation, loss of appetite often nausea, vomiting and jaundice. The degree of jaundice depends on whether nodules in the transverse fissure of the liver obstruct the bile-ducts, and such jaundice, once established, is apt to be permanent. Pain varies: sometimes it is better described by the term uneasiness, in other cases there is much actual sharp pain. Friction where perihepatitis is present generally causes pain. Occasionally the temperature rises to 100 or 101 F. Very often the feet swell, partly owing to the anaemia, partly to interference with the inferior vena cava.

The *Physical Signs* are usually definite. On *Inspection* the enlarged liver may be visible through the abdominal wall moving downwards with the diaphragm on inspiration, and even projections, due to nodules of new growth, may be seen and sometimes dilated veins indicate interference with portal or systemic veins. *Palpation* reveals the enlarged liver and frequently the presence of umbilicated nodules. *Percussion*

corroborates the enlargement, and on *Auscultation*, friction, due to perihepatitis, may occasionally be heard.

The **Diagnosis** largely depends on the possibility of palpating the umbilicated nodules, the recognition of a primary tumour in the stomach or elsewhere, and the cachexia and permanent jaundice (if present). *Gummata* have occasionally led to a mistaken diagnosis, but there is often a history to assist, and antisyphilitic treatment yields beneficial results not obtainable in cases of tumour. Time, too, usually clears up the diagnosis in a doubtful case.

The **Prognosis** of tumour is always grave, and the duration of life varies with the rapidity of growth of the neoplasm, but it is rarely longer than a year, and often much shorter.

The **Treatment** is purely palliative. It is necessary, where pain is present, to give morphia, and to apply fomentations or poultices locally.

(14) Cancer of the Gall-Bladder and Bile-Ducts

Cancer of the Gall-Bladder may be primary, and is probably due to the irritation of gall-stones (fully 90 per cent. of cases). There may be a history of preceding biliary colic, and as the tumour is apt to spread secondarily to the liver, the clinical features are indistinguishable from cancer of that organ.

Cancer of the Bile-Ducts is rarely primary, but may in such cases be due to gall-stones. It causes marked interference with the bile-ducts, with consequent dilatation and permanent jaundice.

It is possible that surgical interference, either draining away bile from the gall-bladder into the intestine, or making an external biliary fistula, may in certain cases afford much relief.

(15) Diseases of the Portal Vein

1. **Passive Hyperaemia** has already been described.

2. **Thrombosis of the Portal Vein**, or **Pylephlebitis Adhesiva**, occurs specially in cancer when it involves the veins, and in alcoholic cirrhosis of the liver. The clot may be extensive or limited, and it may only affect a part of the vein at any one

site. The affected part of the vein may become completely occluded and form a fibrous thread—hence the name Pylephlebitis adhæsiva. In order to carry on the portal circulation collateral anastomosis may provide channels, but if these fail or are insufficient, ascites and haematemesis are inevitable in really extensive cases.

Emboli in the portal circulation do not cause infarction owing to the free anastomosis with the hepatic artery.

3. **Suppurative Pylephlebitis** is described under Abscesses of the Liver.

16. Affections of the Hepatic Vein

1. **Passive Hyperaemia** has already been described with the consequent portal engorgement.

2. **Emboli** may, in rare cases, enter the hepatic vein, equally uncommon is it to find —

3. **Phlebitis** or other interference with the hepatic vein, unless from accidental involvement by tumour.

XVII. DISEASES OF THE PANCREAS

1. Acute Pancreatitis

The most common variety of this is **Acute Haemorrhagic Pancreatitis**. It is rare, although cases are met with from time to time in the post-mortem department of our large hospitals.

Etiology.—It is more frequent in men during adult life, and is said to depend on the presence of gall-stones and the following organisms—B. coli communis, Streptococcus pyogenes Staphylococcus pyogenes aureus, and the Typhoid bacillus.

Pathological Anatomy.—The pancreas shows many haemorrhages on section, and, at operation or post-mortem examination, little areas of fat necrosis may be often seen all over the abdomen, in the omentum and mesentery. Fat necrosis is due to the action of pancreatic secretion on fat and its presence implies an escape of the secretion into the general peritoneal cavity.

The **Clinical Features** are fairly typical. The patient is

suddenly seized with violent colicky pain in the upper part of the abdomen, and this is followed by nausea and vomiting and the early appearance of symptoms of collapse, death often occurring in two to four days with the abdomen distended and rigid. There may be definitely greater resistance in the region of the pancreas, and there is, certainly, greatly increased sensitiveness to pain upon pressure.

The **Diagnosis** lies between *intestinal obstruction* and acute pancreatitis, and a careful note should be made of any point in the history suggesting obstruction. In acute pancreatitis the symptoms come on suddenly in a previously healthy individual, and the localised area of pain and resistance suggests the pancreas. At the operation in a case of obstruction, we find, below the stricture, coils of intestine empty of gas, while in acute pancreatitis there are no depleted coils, and we note also the patches of fat necrosis.

The **Prognosis** is bad, but a few cases have recovered.

The **Treatment** is purely expectant. Operation is too often futile: pain can be soothed, and either heat or cold applied locally, while the patient's strength is supported with stimulants.

(2) Suppurative Pancreatitis

This rare disease is sometimes the result of trauma, but more often no cause can be assigned for it. There may be one, or several, suppurative foci. The clinical features vary: there may be local pain and vomiting, and sometimes definite resistance can be made out, on palpation, over the site of the pancreas. There may be fat in the stools, and sometimes sugar in the urine, and the inflammation may spread and set up jaundice. The abscess may open into a neighbouring viscus, and occasionally the portal vein becomes infected, causing thrombosis, and possibly pylephlebitis suppurativa (abscess formation in the liver in the line of the portal circulation).

The disease often tends to run a chronic course, and the only possible treatment is surgical, an attempt being made to drain the abscess.

(3) Gangrenous Pancreatitis

This may result from either of the preceding diseases or from injury. It is nearly always fatal. The clinical features

are very acute and resemble haemorrhagic pancreatitis. In one or two reported cases the necrotic pancreas has been passed by the bowel into which it had perforated.

(4) Cirrhosis and Atrophy of the Pancreas

There may be either (*a*) a thickening of the interstitial septa of the gland, or (*b*) the fibrous tissue may penetrate and involve the glandular structures themselves, namely, the islands of Langerhans. Both these conditions lead to hardening of the organ, and they may be combined. In diabetes there is often not merely an atrophy of the pancreas, which is a common condition in that disease, but the islands of Langerhans are specially affected by sclerosis. A chronic interstitial fibrosis of the pancreas may eventually involve the ductus choledochus and thus cause jaundice.

5. Pancreatic Concretions or Calculi

These are rare and are very difficult to diagnose, although when they are passed by the patient, or cause the formation of a cyst, their presence may be confirmed. Men suffer twice as frequently as women. These calculi occur in the duct of Wirsung, the branches of which are dilated. They are of a whitish colour, may be coral-like in structure, and consist of calcium carbonate or phosphate. Occasionally they cause suppuration and sometimes colic, but not as a rule. There may be fat in the stools owing to the blockage of the main duct, and indicating loss of the emulsifying ferment, and there may also be glycosuria, due to interference with the internal secretion of the organ.

There are often no clinical features except in severe cases the diagnosis may be confirmed by the X-rays, and the treatment is surgical.

(6) Pancreatic Cysts

Besides hydatid cysts, and cysts in the pancreas associated with cystic disease in the kidneys, liver, and brain, all of which are rare, there are cysts which are very difficult to account for. Plugging of the duct of Wirsung does not necessarily, or even commonly, cause cystic distension of

the duct behind the block, and although calculus and tumour may explain a certain number of retention cysts, they do not explain the larger and more important ones. There may be no block at all. Interstitial pancreatitis may cause blocking and subsequent distension of smaller branches of the duct. Some cysts seem explicable as the result of injury, and some of them are cystic adenomata formed in connection with the gland structures.

The cysts are generally of large size; the fluid is turbid, alkaline, and albuminous, and may possess the properties of a dilute pancreatic secretion. The cyst may contain 400 oz. of fluid, and so form an immense abdominal tumour. It may grow into the lesser sac of the omentum, and, pushing the stomach upwards, may appear between the stomach and transverse colon, or it may push the stomach downwards: but much depends upon the part of the pancreas with which it is connected and from which it grows. The cyst will in such cases be palpable, and is found to be fixed, and not movable with the diaphragm. The chief symptoms are due to the cyst pressing on the stomach and other neighbouring organs, and include dyspepsia and sometimes vomiting. There may, though rarely, be fatty diarrhœa; glycosuria is more common, and generally there is rapid emaciation. Hæmorrhage sometimes occurs into the cyst, and may lead to alarming collapse.

The **Treatment** consists either in tapping the cyst, an operation which may need to be repeated, or else in freely opening below the 12th rib and draining. The presence of persistent glycosuria is a grave feature.

Tumours of the Pancreas: Malignant Tumours

These may be primary or secondary.

I. **Primary Tumours.** 1. Carcinoma of the scirrhus variety is described as being not infrequent and as involving the head of the pancreas. In our experience it is extremely rare, and, with two exceptions, the cases at first believed to be primary tumours of the head of the pancreas were found not to involve the gland primarily. 2. Sarcoma is very rare.

II. **Secondary Tumours** are much more common, both carcinomatous and sarcomatous. Often the so-called primary

carcinomas of the head of the pancreas are really secondary and certainly involve by preference the head of the organ.

The **Clinical Features** are generally distinctive, and include epigastric pain, jaundice due to the involvement of the orifice of the ductus choledochus, a palpable tumour which is painful on pressure, rapid emaciation, and sometimes fatty diarrhœa and glycosuria. Sometimes the pylorus is interfered with and gastrectasis develops.

The **Diagnosis** is in many cases fairly easy.

The **Prognosis** is bad.

The **Treatment** is simply that of symptoms, pain often demanding the use of opium.

Gummata are sometimes met with in the pancreas, and occasionally tubercular tumours.

XVIII. DISEASES OF THE PERITONEUM

(1) Acute Inflammation of the Peritoneum

Etiology.—Acute peritonitis may be of **Primary** or **Secondary** origin.

Primary cases may be dismissed in a couple of words. They are distinctly rare; although called primary, they are the result of an organism or possibly a toxin, and occur in affections which are, more or less certainly, of infective or toxic nature, such as influenza, Bright's disease, sometimes acute rheumatism, and occasionally gout. It is probable that in every case, an organism is present, and that the peritonitis is often really due to a terminal infection with an organism. It is extremely unlikely that cold can alone be responsible for peritonitis.

Secondary peritonitis is by far the more important. It may be of local origin, and may be associated with a lesion of the stomach or some part of the intestine, abscess in the liver, glandular suppurations, pyosalpingitis, or other pelvic suppuration. Among the most common varieties of local peritonitis in connection with the intestine is that due to appendicitis. Many cases are generalised from the outset, where the inflammation is not limited by adhesions, but perforation through the posterior wall of the stomach may involve only the lesser

sac of the omentum if the foramen of Winslow is closed, whereas perforation through the anterior wall of the stomach will almost certainly set up a general peritonitis. Malignant tumours of stomach or bowel are sometimes responsible for the involvement of the peritoneum. Perforation of a suppurating gall-bladder and the rupture of an extra-uterine gestation constitute two possible, though not very common, causes of peritonitis.

Acute peritonitis may be associated with the distribution of acute miliary tubercles over the peritoneum, and in pneumonia, septicaemia, and pyaemia the peritoneum, as well as other serous membranes, may become infected.

Of 102 cases referred to by Osler, 56 were due to extension from some abdominal focus, 34 followed surgical procedures, and 12 were described as primary, and in all of these 12 an infective organism was present.

Pathological Anatomy.—The peritoneum becomes intensely congested, and both adhesive and serous or seropurulent lymph is rapidly poured out. Often matting together of loops of the bowel occurs. The bowel becomes distended, and in a severe case the muscle in the wall of the gut is paralysed, causing complete obstruction, because of the interference with ordinary peristaltic contractions as well as from the presence of adhesions. The fluid may amount to from 20 to 500 or more ounces; it may be sero-fibrinous or purulent, or in certain cases haemorrhagic, and sometimes it has a faecal odour. Occasionally the bowel and affected peritoneum become gangrenous.

The following organisms may be present: the Streptococcus pyogenes and the Bacillus coli communis, which are both extremely common, while the Staphylococcus aureus may infect the peritoneum after operation. In pneumonic cases the Pneumococcus may be the infective agent, and in gonorrhoeal salpingitis the Gonococcus may find its way into the general peritoneal sac. The Bacillus pyocyaneus, the Bacillus aerogenes capsulatus, and many other organisms have also been described.

Clinical Features.—(A) **Generalised Peritonitis.**—If the condition is severe and *general*, constant pain, which is increased by pressure, is experienced over the abdomen. A good deal depends on the mode of origin, but where

perforation has occurred, the onset is sudden and the pain intense, while a rigor is not infrequent. The site of the pain varies; it may be more marked at the umbilicus or in the region of the appendix, but may be generalised. The abdomen becomes rigid and motionless, diaphragmatic breathing being prevented by the patient so far as possible. Frequency of micturition is common, and an excess of indican has been noted in the urine. Vomiting soon occurs and is constant, first the contents of the stomach being brought up, and, later, faecal-smelling material from the intestine, while dreadful hiccough tortures the patient. The attitude is characteristic, the patient lying on his back with the knees drawn up, and hardly venturing to breathe, talk, or cough. The pulse is wiry and rapid, and the temperature soon rises, and may reach 104 or 105 F. There is almost always constipation, due to obstruction, although diarrhœa sometimes occurs, owing probably to the emptying of the bowel below the site of the obstruction. At a later period the abdomen becomes swollen from distension with gas; there is the same pain, which is greatly added to by the difficulty of breathing and the general discomfort. The cheeks sink, and if relief is not obtained, the pulse becomes thready and soft, and death rapidly occurs from collapse. The patient's facial expression demands a brief description. It is not merely pinched, but it is extremely anxious; the eyes, which are bright to begin with, become dull towards the stage of collapse.

Great care should be taken to investigate, by very light percussion, the condition of the abdomen and the amount of fluid present, although it is rather in cases where the peritonitis is limited by adhesions that the diagnosis is aided by careful physical examination. Where there is air or gas in the peritoneal sac, the liver dulness may completely disappear.

(B) **Localised Peritonitis.**—In this case the pain is generally limited to the side of the affection, and while it may occur in many different positions, it is perhaps more frequent either in the region of the appendix, in the pelvis, or in the lesser sac of the omentum, and the most thorough examination is often necessary to enable a diagnosis of the latter site of peritonitis to be made. A subphrenic abscess is a not infrequent result of peritonitis in the lesser omental sac, and the fact that the liver is displaced downwards is suggestive. In

localised cases of peritonitis, the symptoms, as a rule, are less severe, although there may be the same vomiting, intense pain, high temperature, and eventual death. One great risk is that the abscess may burst into the general peritoneal sac, while in certain cases it opens into the bowel, or may even make its way externally.

Forms of Localised Peritonitis. 1 *Appendicular Peritonitis.* In this case a localised peritonitis, or possibly a peritonitic abscess, may be associated with the inflamed appendix. The clinical features are those of appendicitis, and the position of the abscess is generally just behind the termination of the ileum.

2) *Pelvic Peritonitis.* Beyond stating that this form of peritonitis is much more common in women, and is generally due to salpingitis, it is hardly necessary to describe it here.

(3) *Subphrenic Peritonitis.*—Peritonitis may be localised to a part of the greater peritoneal cavity where it is related to the liver, and may be due to an abscess of the liver just under the right half of the diaphragm or to infection from the right pleura, or it may be confined to the lesser omental sac, and in cases of cancer or ulcer of the stomach or pancreatic inflammations, abscesses confined to this sac are by no means uncommon. In this case the abscess is bounded above by the left half of the diaphragm and on the right by the falciform ligament of the liver, while the lobulus Spigelii is exposed in the abscess cavity. The stomach is anterior and the pancreas bounds the abscess below. The left half of the vault of the diaphragm may be pushed upwards, greatly interfering with the heart. Sometimes the subphrenic abscess contains gas or air.

The **Clinical Features** of subphrenic abscess vary considerably; they include marked constitutional symptoms, and suggest sometimes merely localised pain, but sometimes the presence of a considerable effusion of pus. The heart is interfered with and often the breathing. Physical examination demonstrates the displacement of the liver downwards by the effusion where the abscess is on the right side. The symptoms depend, in part, on the cause of the condition. Unfortunately the prognosis is unfavourable in most cases.

The **Diagnosis** of peritonitis.—Where the peritonitis is general, the clinical features are extremely characteristic,

and the difficulty consists rather in the recognition of cases of acute localised peritonitis which may in time become generalised. In females, the possibility of a salpingitis should not be forgotten, and perforating appendicitis and gastric and duodenal ulcers should be kept in mind. In typhoid fever ulcers sometimes perforate, but there is, as a rule little difficulty in the diagnosis.

In *faecal impaction*, or other form of *intestinal obstruction* and in cases of *enteritis* the pain may suggest peritonitis, but in these cases it resembles ordinary colic, and in enteritis, at all events, diarrhœa is common. In *hysterical* subjects the pain of peritonitis may be simulated, and the typical picture of a peritonitis, if previously familiar to the patient, may be portrayed with great exactness and it is only from the knowledge of the patient's previous history and by careful observation of the case that a true diagnosis can be made.

The **Prognosis** is always grave in every case of peritonitis, generalised or localised, and where a gastric or intestinal ulcer has perforated, prompt surgical treatment can alone yield any hope of cure.

Treatment of Acute Peritonitis. In every case an attempt should be made to diagnose the cause of the condition, so that, where necessary, the patient may be operated on with the least possible delay.

The medical treatment of acute peritonitis consists in rest in bed; a cage being placed over the abdomen so as to remove the pressure of the bedclothes. Decide next whether to apply ice, or to use warm applications in the form of fomentations with lead and opium or poultices. In many cases ice affords the greatest relief, while it prevents, to a certain extent the awful vomiting which distresses the patient. Morphia should also be administered hypodermically to relieve pain. Where abdominal distension is very extreme, a long rectal tube may be passed into the bowel to draw off gas, or the abdominal wall may be punctured, and the gas taken directly from a distended coil. Sometimes the use of turpentine or assafœtida enemata gives relief in these cases. Saline purgatives have been strongly recommended so as to limit adhesions, but the administration presents difficulties, and there is some doubt as to their efficacy. Along with appropriate surgical treatment there is no question of the very great

value of normal saline injections, generally given almost continuously by a long tube and at low pressure into the bowel, but sometimes used intravenously. By means of an appendicostomy it has been suggested that normal saline can be introduced at will into the intestine. Lastly, for the sickness and nausea the stomach may be occasionally washed out.

The different forms of localised peritonitis demand treatment suitable for each condition, and beyond stating again that the ice-bag or the poultice may be applied locally, and that morphia hypodermically relieves pain, nothing need be added here.

2) Chronic Peritonitis

A chronic inflammation of the peritoneum which may be general or localised.

a) **Cancerous Peritonitis.**—This is usually *secondary* to cancer of the stomach or other organ, but *primary* peritoneal tumours, most of which are endotheliomata, are sometimes seen. In the majority of secondary cases, in addition to the primary lesion, there are numerous cancerous nodules, varying greatly in size, dotted over the peritoneum. There are many adhesions, while the mesenteric glands are enlarged, and the omentum is often puckered up, forming a large mass of new growth stretching across the abdomen. The amount and kind of the fluid present vary in different cases. It is often haemorrhagic, and sometimes owing to secondary infection it becomes purulent.

In colloid cancer the disease infiltrates the peritoneum to a remarkable extent, and large quantities of jelly-like material are present in the peritoneal sac.

The **Clinical Features** vary with the position of the primary lesion and the interference due to the peritoneal involvement. The presence of fluid can readily be made out, and the physical signs are referred to under ascites. Intestinal obstruction is a frequent sequel, and the amount of colicky pain is often very great.

In making a **Diagnosis** the cellular elements in the fluid should be examined; they may suggest the cancerous origin. The presence of an omental tumour is very striking, and cancerous nodules near the umbilicus frequently follow carcinoma hepatis.

The **Prognosis** is grave in the extreme.

(*b*) **Tuberculous Peritonitis.**—There may be either (1) an *acute miliary tuberculosis*, often with the presence of a haemorrhagic effusion, and really a form of acute, not chronic, peritonitis, or (2) *tubercular masses of a large size* causing great matting of the bowel, although in some cases what may be described as (3) a *chronic miliary tuberculosis*, with comparatively few large tumour masses, may also be met with.

The tubercular involvement of the peritoneum is often secondary to, or associated with, tubercular ulceration of the intestine, and in many cases the mesenteric glands are also extensively involved. Not infrequently, persons so affected have obvious pulmonary disease, or the glands in the neck are tubercular. While tubercular peritonitis must be described alone, it should be remembered that it often, but by no means always, occurs coincidently with tubercular enteritis and tabes mesenterica. Tubercular peritonitis is most common in children, and is certainly rare after middle life.

The **Clinical Features** of the acute miliary type closely resemble acute peritonitis already described. In the more common chronic cases the clinical features vary greatly, and depend much on the size of the tubercular tumours, the amount of matting of the intestine, and the presence or absence of ascites. In certain cases there is marked pyrexia of hectic type, with great abdominal tenderness, while in others there is little temperature and a comparatively small amount of pain. Intestinal dyspepsia is common, and the abdomen is often swollen, more from the presence of gas in the intestinal coils than from the fluid, which may also, however, be in considerable amount. In some cases the omentum forms a definite tumour infiltrated with tubercular masses, and its position, stretching across the abdomen, is very characteristic. Effusions are often sacculated, and definite tumour-like masses may be felt in different regions of the abdomen, while in many cases the enlarged mesenteric glands may be made out on careful palpation. These mesenteric glands are generally affected in younger patients, and are tender to the touch.

In **Diagnosis** it is desirable to investigate the family history, and to examine with the greatest care for evidences of tubercular involvement of the chest, although the source of infection may have been milk. It should not be forgotten

that in females a primary tuberculosis may be met with in the Fallopian tubes, and that a tubercular salpingitis can sometimes be definitely recognised by vaginal examination.

(*c*) **Proliferative Peritonitis.**—In this affection the peritoneum becomes greatly thickened, in fact, undergoes proliferative changes, and, as a result, the mesentery is shortened and the omentum pulled upwards, forming a more or less definite tumour. The thickening also affects the peritoneal covering of the liver and other organs, and there is frequently perihepatitis or perisplenitis. This thickening may be due to frequent tapping of ascitic effusions, but there are many cases in which the etiological factor is by no means clear. It occurs along with cirrhosis of the liver, and occasionally in alcoholic subjects.

The **Clinical Features** are sometimes obscure. There is generally considerable ascitic effusion, and not infrequently the localised peritoneal thickening interferes with intestinal peristalsis causing obstructive symptoms.

(*d*) **Local Chronic Peritonitis**, not included in the above three groups, is very common in connection with the liver and spleen, and also, though less frequently, with other abdominal organs. It may occur in cirrhosis of the liver or syphilitic disease of that organ, and it is not unusual in carcinoma hepatis. Perisplenitis is frequently associated with leucocythaemia, and is also a common result of splenic infarction.

The **Clinical Features** include pain, often with definite friction, palpable and audible, while bands of adhesions may constrict a loop of bowel and thus cause acute obstruction.

Treatment of the Foregoing Conditions.—In *Malignant* cases little can be done except to relieve the patient by occasional tapping and by local and internal sedatives. *Tubercular Peritonitis* yields satisfactory results, often simply from abdominal section, the operative interference apparently blighting the disease. Medicinal treatment includes the use of mercurial or potassium iodide ointments rubbed into the skin of the abdomen and sometimes applied on a binder. Tuberculin has yielded good results in chronic cases. Careful dieting is advantageous, and the better the condition in which the patient's general health can be placed, the more likely is improvement to follow.

Proliferative Peritonitis is incurable, although the removal of fluid when necessary adds greatly to the patient's comfort. *Local Chronic Peritonitis* should be treated either by the application of the ice-bag or by counter-irritation.

The method of performing the operation of tapping the peritoneum is described under ascites.

(3) Ascites

The effusion of more or less serous fluid into the peritoneal sac.

It is due to a variety of conditions, and is in reality rather a feature of several diseases than a disease in itself.

Etiology.—A. The fluid may be a *transudate* or dropsical lymph due to (1) backward pressure from heart disease: lung disease, as in very marked emphysema; affections of the portal circulation, especially in the liver, as in common cirrhosis of that organ; and tumours pressing on the inferior vena cava. (2) It may occur in the dropsy of Bright's disease, profound anaemia, etc.; and (3) it may result from obstruction of the lymphatic channels and thoracic duct.

B. The fluid may be an *exudate* of more inflammatory lymph, such as occurs in tubercular peritonitis and other acute and chronic forms of peritonitis, and in connection with cancerous growths of the peritoneum.

The fluid differs much in appearance and character in the two types of cases. A transudate or dropsical effusion has a low specific gravity, rarely exceeding 1012, and often about 1008, while in an inflammatory effusion it is rarely under 1018. Dropsical effusions or transudates are yellow or greenish, do not coagulate spontaneously, and contain few cells, while inflammatory effusions or exudates coagulate spontaneously, and contain a much larger number of cellular constituents. The fluid may become chylous from rupture of lacteals, as in filariasis, and it is not infrequently chylous or milky in tubercular and malignant peritonitis, owing, it is thought, to fatty degeneration of the cells lining the peritoneal sac, thus causing the milky appearance. The amount of the fluid varies greatly, from 10 or 20 oz. up to 400 oz., 600 oz., or more. There is probably always some fluid in the peritoneal sac, but transudation and absorption balance each other; in disease

this balance is upset. There is often blood in the fluid in tubercular and malignant cases, but this is not always to be depended upon: sometimes in malignant disease the cells in the fluid may be diagnostic. The peritoneum loses its smooth, glistening appearance, and in chronic cases becomes thickened and more opaque.

The **Clinical Features** depend on the amount of the fluid, and in what is termed globular ascites, where the dropsy is of portal origin, the appearance of the abdomen with, in the first instance, no oedema of the legs, is very typical and suggestive. The fluid tends to gravitate, and the intestinal coils generally float on the top. In a marked case on *Inspection* the skin may show recent striae or cracks, the umbilicus may be protruded like a hernia, and there may be enlarged veins indicative of the position of the pressure, either laterally, carrying blood from below up to the superior cava by the mammary veins (pressure on inferior vena cava), or by the distended veins round the umbilicus, the so-called *caput Medusae*, by which portal blood reaches the superficial abdominal veins by veins in the round ligament of the liver. In moderate effusions there is marked flattening in the flanks.

On *Palpation* a very definite thrill or fluctuation can be made out, if the fluid is not loculated. One hand is placed over one flank, and with the fingers of the other hand the opposite flank is flipped or tapped so as to produce a wave in the fluid. As the air-containing bowel sometimes transmits a pseudo-fluctuation, it is a great help to get the nurse to place a hand vertically, in the middle line of the abdomen, by which means this pseudo-fluctuation is prevented. Often by palpation a tumour, or enlarged liver, or other condition which bears an important relationship to the ascites, is revealed. On *Percussion* the fluid is found to follow the law of gravity, unless there are adhesions, so that with the patient in the dorsal position, the flanks are dull, and the intestines floating on the top of the fluid give a tympanitic note in the middle line, and it is only necessary, after percussing the flank and obtaining a dull note, to roll the patient on to his side and so get a clear tympanitic note where previously there was dulness. If the fluid is small in amount, place the patient in the knee-elbow position in bed, and percuss the most dependent part of the abdomen.

Only in very rare cases does the fluid cover over the

intestinal coils entirely, and especially when, as the result of proliferative peritonitis, the intestinal coils are bound back to the posterior abdominal wall.

Diagnosis.—The fact that the fluid alters its position sharply distinguishes it from fluid confined in a cyst, such as in an *ovarian tumour* or *hydatid cyst*. The nature of the jelly-like, brownish fluid in an ovarian cyst is very distinctive, and its specific gravity is generally high,—while a hydatid cyst contains clear watery fluid rich in sodium chloride, with little or no albumin, and often hooklets.

A mistake should never be made in connection with a *distended bladder*. The dribbling of urine is suggestive, and the passage of a catheter saves all further doubt.

The **Prognosis** must depend largely on the cause of the condition. Many inflammatory and some dropsical effusions are of very grave significance.

The **Treatment** varies with the nature of each case. It is often desirable, as well as necessary, to tap the effusion, but only a small amount may need to be removed so as to aid nature. Southey's tubes are generally used; sometimes, and certainly for effusions which do not flow freely, Potain's aspirator is preferable. Draw off the fluid slowly if an aspirator is used, and keep up abdominal pressure by means of a bandage, so as both to obviate shock and prevent the abdominal blood spaces distending, and the patient being bled into them with consequent fainting. Try diuretics, purgatives, and sometimes diaphoretics, and thus aid nature so far as possible, even if tapping may be also necessary. A dry diet is good practice in many cases, and it has been found that the elimination of sodium chloride is sometimes helpful.

In cases of common cirrhosis of the liver, patients have had enormous amounts of fluid drawn off, and occasionally with good results, the ascites gradually diminishing thereafter. Many times the patient's own weight may thus be removed in an illness of months or years. An abdomen may fill up at the rate of at least 10 to 15 oz. a day, with a consequent heavy drain of albumin, and hence many physicians prefer to delay tapping save when it is absolutely necessary. Never tap until the bladder has been emptied, and introduce the trochar and canula in the middle line and well below the umbilicus.

DISEASES OF THE HAEMOPOIETIC SYSTEM

I. GENERAL ANAEMIA

Anaemia may be primary or secondary.

(1) Primary Anaemia

A. Chlorosis ; B. Pernicious Anaemia. — These are described separately, and it is possible that there are cases which should be termed primary anaemias, and which yet do not fall under either of these two types.

A. Chlorosis ; "Green Sickness"

A form of anaemia in which, with a considerable diminution in the number of red cells, there is a proportionately greater decrease in the amount of haemoglobin.

Etiology.—This is the typical anaemia of girls at puberty, and is probably dependent on unsatisfactory conditions, as regards bedroom, work, and diet, which influence the general health at a time when the strain of development and the advent of menstruation demand the best hygienic surroundings and the greatest care of the general health. Many delicate girls are, however, exposed to insanitary surroundings at puberty, and only a small proportion become chlorotic. Constipation has, therefore, been considered a possible cause, with a resultant auto-intoxication, but many lifelong sufferers from constipation escape chlorosis entirely. In all cases of supposed chlorosis it is wise to keep in view the possibility of tuberculosis.

Sexual derangements may be associated, both as cause and as effect. On the other hand, there are cases which occur

years after puberty, and there are also cases associated with imperfect development of uterus and ovaries.

A hypoplasia or maldevelopment of the heart, and especially the aorta, has been described in a few exceptional cases of the disease, but this is too rare to be accredited with an important bearing on chlorosis generally.

In conclusion, the disease is, in all probability, a defective blood-formation, due largely to insanitary conditions, with unsatisfactory diet, and want of exercise and fresh air, at a time of life when development is most rapid.

Pathological Anatomy.—(1) The red cells fall to

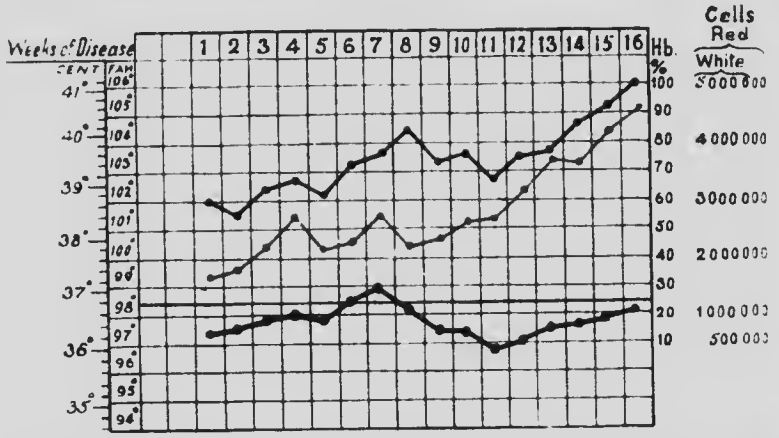

FIG. 20.—Blood Chart. Case of Chlorosis. Recovery. Black line indicates number of red blood corpuscles. Red line indicates haemoglobin percentage. Blue line indicates number of leucocytes.

2,500,000 or 3,000,000 per cubic millimetre of blood; and (2) the haemoglobin index falls to a proportionately much lower figure, perhaps to 20 to 30 per cent. There may be a degree of poikilocytosis in profound cases, but no constant alteration in the leucocytes. A few nucleated red cells may be present, indicating nature's attempt at remedying the defects.

Fatty degeneration of the heart muscle is not uncommon in protracted cases, and a similar change may occur in the liver. A remarkable feature is the plumpness of patients who are the subjects of chlorosis. A limited oedema of dependent parts is often present.

Clinical Features.—The typical changes in the blood have been described under the pathological anatomy. Patients

suffering from chlorosis are usually plump, but have a peculiar waxy-looking complexion, and often, as the name implies, may show a somewhat greenish tinge. The pallor is specially obvious in the palpebral conjunctivae, and in the gums and lips, while occasionally a deceptive coloration of the cheeks is present. The sclerotics have a markedly bluish colour. The patient complains of breathlessness, increased on the slightest exertion, and there is langour and inability both for physical and mental work. The face is sometimes puffy, and there may be slight oedema of the ankles.

Circulatory System.—The most important point is to examine for evidence of cardiac dilatation, and a systolic murmur may be heard perhaps most distinctly and commonly at the base of the heart, often also in the mitral area.

The haemic murmur may be explained by the theory that there is an atonic condition of the heart wall and of the fibres which surround the mitral orifice, thus leading to inadequacy so long as the anaemia persists. Its production at the pulmonary orifice may be due to unequal tension of cusps or of the arterial wall.

A *bruit de diable*, or humming-top murmur, is audible over the jugular vein at the root of the neck: in some cases it is a continuous hum, in others there is marked systolic accentuation. The *bruit de diable* is due, in part, to pressure of the cervical fascia on the bulb of the jugular vein, and it is louder when the head is turned away from the side which is being examined, because under these circumstances this pressure is increased.

The pulse is full, of low tension, and shows a tendency, on occlusion of the vessel with the finger, to the appearance of distal pulsation, owing to dilatation of vessels in connection with the palmar arch. There is a predisposition to thrombosis of veins, especially in the legs, and to a less extent in the cerebral sinuses. There may sometimes be a haemic bruit heard over the eyeball or over the lateral sinus.

Alimentary System.—There is generally loss of appetite with gastric pain, sometimes vomiting, but almost invariably persistent constipation. There seems to be a relationship between hyperacidity, so often present here, and the development of simple gastric ulcer. In certain cases the appetite is capricious, the patient longing for unusual articles of food.

In the other systems we find typical features, and

especially amenorrhoea or dysmenorrhoea, and very frequently headache, giddiness on suddenly assuming the erect posture, mental irritability, and even hysteria.

Diagnosis.—The examination of the blood is distinctive, although it may be difficult in certain cases to exclude the possibility of the condition being a *secondary anaemia*, and tubercular disease must be thought of in this connection.

Prognosis.—In the majority of cases, recovery can be definitely promised within a period of one or two months, but it should be remembered that what may be termed predisposing causes may bring about more than one relapse.

Treatment.—Try to eliminate any definite causal factor, and give iron, especially in the form of the protosalts; perhaps some preparation of ferrous carbonate is the best. Whatever salt of iron is administered, do not neglect the use of purgatives. The diet should be simple and nutritious, and the patient should have plenty of fresh air and sunshine, while in cases of cardiac dilatation, absolute rest in bed is an important part of the treatment. On no consideration allow the patient to return to work until the cardiac dilatation has disappeared and the haemoglobin has returned to its normal proportions.

B. Pernicious Anaemia

A form of primary anaemia in which there is marked diminution in the number of red blood corpuscles and also of haemoglobin, the haemoglobin index for each red blood corpuscle being, however, greater than in any other form of anaemia. There are all the clinical features of profound anaemia with, in most cases, a rapid and fatal termination.

Etiology.—The following diseases appear to bear a certain causal relationship to pernicious anaemia—the presence of the tape-worm Bothriocephalus latus, Anchylostoma duodenale or anaemia worm, and gastric dilatation and tumour. An unusually profound type of any other anaemia may eventually take on a pernicious tendency.

There is little doubt that some toxin is the real cause of pernicious anaemia, although we cannot always explain the nature or source of the toxin. We have seen a case in which pernicious anaemia developed and proved fatal in ten days after confinement, and at the *sectio* the uterus was found

to be gangrenous. The subject, aged 33, had, however, been anaemic for some time previous to the birth of the child.

Hunter considers that the disease is due to the entrance of organisms by the mouth in connection with carious teeth, while other authorities consider that intestinal sepsis has a direct bearing on the condition. After a prolonged haemorrhage, severe attack of fever, or excessive physical strain, pernicious anaemia has sometimes originated, although the causal relationship between the preceding conditions and the anaemia has not been proved. The disease is due to a

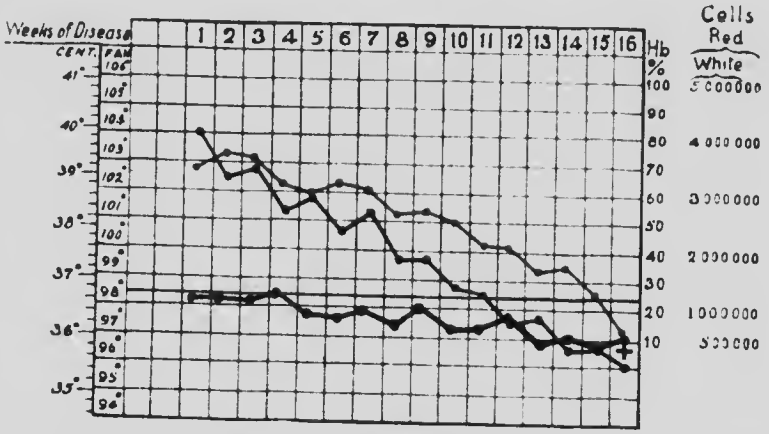

Fig. 24. Blood Chart. Case of Pernicious Anaemia terminating fatally. Black line indicates number of red blood corpuscles. Red line indicates haemoglobin percentage. Blue line indicates number of leucocytes.

haemolysis, although whether it occurs in the liver as Hunter believes, or in the bone marrow or elsewhere, we cannot definitely say. It is rather more common in men, and about middle life.

Pathological Anatomy.—The *blood* shows a marked diminution of red cells, the number often falling to 1,000,000 or 500,000, while in exceptional cases the figure may fall below 200,000 per cubic millimetre of blood. There is marked poikilocytosis, by which is meant a distortion in shape of the red cells; tailed cells, and cocked-hat-shaped cells being extremely common. The blood coagulates badly, and the red cells do not form the typical rouleaux. There are many megalocytes, measuring 12 to 15 μ, and also microcytes measuring 3 to 4 μ. Nucleated red cells or normoblasts are present at

intervals during the course of the disease, and often large nucleated reds or gigantoblasts, and the red cells have a special staining affinity for iodine. The leucocytes are generally diminished in number, especially in cases in which the disease is progressing unfavourably, and a relative increase of lymphocytes (35-40 per cent) has sometimes been noted. In some instances myelocytes may be found in the blood, but generally in small numbers. The blood-plates are diminished, and the specific gravity is markedly lowered.

Haemorrhages are common, mostly as small petechial haemorrhages, subserous, and more rarely subcutaneous in position, while small haemorrhages occur in the retinae, in connection with the meninges and elsewhere.

The *bone marrow* is usually deep red, and has an almost gelatinous appearance. It is unduly rich in cells, which are large and nucleated.

The *liver* shows fatty degeneration, and there is great excess of iron pigment in many organs of the body, including the liver, spleen, kidneys, and bone marrow; its presence is easily demonstrable by the ferrocyanide of iron reaction.

The *heart* shows marked fatty degeneration, and may be dilated, while there is in certain cases a sclerosis of the posterior columns of the *spinal cord*.

Clinical Features.—Cases may be (1) acute, when death occurs in a few days' time; (2) subacute, which may do well for a time but relapse, and generally prove fatal during the second or a subsequent attack; and (3) chronic, the common type which may go on for an indefinite time with many relapses. In the chronic cases the disease comes on slowly, the face soon becoming pale and bloodless, and the skin has a somewhat saffron or lemon-yellow colour. There is no great diminution in the amount of fat, but the pallor of lips, gums, and conjunctivae is most pronounced. There is marked dyspnoea, and as a rule cardiac dilatation, and the presence of loud haemic murmurs together with the usual *bruit de diable* in the neck, and palpitation is excited by any exertion. The pulse is feeble and rapid, and there is very commonly, in advanced cases, dyspepsia, vomiting, and not infrequently diarrhoea. A degree of pyrexia is present from time to time, no particular reason for the rise of temperature being forth-

coming. The urine contains an excess of urobilin, an evidence of the destruction of red blood corpuscles.

In the *Nervous System* one finds headache and giddiness, sleeplessness, and generally marked irritability, associated with languor and unfitness for continued mental effort. Retinal haemorrhages are frequently seen. Where sclerosis occurs in the spinal cord it involves the postero-lateral columns, and the patient suffers from numbness of the legs, and possibly in-co-ordination with marked Rombergism.

Amenorrhoea is very common, as can be readily understood.

Relapses are apt to occur, and apparent improvement is too often temporary. Blood crises have been described, in which numbers of nucleated reds enter the circulation and remain for a few days. They may represent an effort at cure.

Diagnosis.—The colour of the skin is very suggestive, while the clinical examination of the blood prevents any possible error being made.

Prognosis.—Very few cases recover, and it is doubtful whether even the few that do so are permanently cured. The presence of gigantoblasts is considered a grave feature.[1]

Treatment.—Attention should invariably be paid to any possible causal factors, and especially to the existence of intestinal parasites, gastric disease, and oral sepsis. If the patient has been losing blood by haemorrhoids or otherwise, promptly secure its arrest. It is a remarkable fact that anaemic patients of all kinds benefit greatly by hill air, and this should certainly be tried in cases of pernicious anaemia, together with a good and nutritious dietary.

The only drug which seems to be of genuine benefit is arsenic, and it should be given in 5-minim doses thrice daily, and gradually increased until 10 or 12 minims are being taken with each dose. It is doubtful whether iron does any good, and, in fact, the uselessness of iron has been suggested as a means of making the diagnosis certain.

Whatever our views with regard to oral sepsis and intestinal fermentation may be, it is wise to try salol or β-naphthol. An attempt may be made to give red bone marrow, although its efficacy is very doubtful.

[1] Gigantoblasts indicate that the bone marrow has assumed a foetal or embryonic character.

So profound does the anaemia become that transfusion of defibrinated blood or of saline solution has often been resorted to, but only in a few cases does appreciable benefit result. Oxygen inhalations are worth trying. It is certain that some toxin of unknown nature is present, and as yet we have no satisfactory means of treatment for most cases of this disease.

2. Secondary Anaemia

This may be the result of (1) a loss of blood, (2) a constant drain on the system, as in Bright's disease and prolonged pus formation, (3) insufficient ingestion of food or defective digestive processes, and (4) diseases or toxic conditions which interfere with the nutrition and the activity of the great vital processes.

(1) **Loss of Blood.** A haemorrhage of 3 to 4 lbs. weight of blood may prove fatal. Water and salts are quickly obtained from the gastro-intestinal tract, and even the albuminous constituents of the blood-serum may rapidly recover to more or less normal proportions, but the red blood corpuscles take weeks to return to their former numbers, and the haemoglobin index remains low.

Some hours must elapse after a haemorrhage before the anaemic characteristics appear in the blood. The haemoglobin is usually proportionately less than the red blood corpuscles, the ratio being often described as of a chlorotic type. There is marked poikilocytosis, a few nucleated red blood corpuscles (normoblasts) generally appear, and, as a rule, a leucocytosis of moderate degree develops soon after the haemorrhage, diminishing as recovery occurs. It is a grave feature to find the mononuclear leucocytes increased, and the multinuclear proportionately diminished, for it generally signifies a rapidly fatal termination.

(2) **Anaemia from constant Drain on the System as in Bright's Disease, prolonged Pus Formation, and similar Conditions.**—The drain in this case may be rather on the albuminous elements of the blood than on the red blood corpuscles themselves, but the phenomena are the same and the reduction in number of red blood corpuscles, in amount of the haemoglobin, together with the appearance of poikilocytosis, may exactly correspond to what obtains after a severe

loss of blood. Where, however, the drain is a continuous one, the appearance of nucleated red blood corpuscles (normoblasts) is infrequent.

(3) **Anaemia due to Insufficient Food.** — Inability to swallow food, or to digest food when swallowed, may account for inanition. There may be the failure to elaborate food when absorbed, circulatory defects preventing the proper utilisation of what has been elaborated, and respiratory disease preventing the aeration of the blood, and these may all indirectly, if not directly, lead up to the same result. In such cases the plasma is believed to suffer more markedly than the red blood corpuscles.

(4) **Toxic Anaemia** is the result of certain poisons, such as lead, mercury, syphilis, or malaria, which destroy the red blood corpuscles. Probably these and similar agents act more markedly in cases in which digestive and other errors are also existent. Nitrobenzol, ricin, and potassium chlorate are haemolytic agents of great potency, and they tend to produce a type of anaemia resembling that in pernicious anaemia. The red blood corpuscles have a high colour index, and there are many gigantoblasts present indicating that the bone marrow has reverted to the foetal type.

The **Clinical Features** of secondary anaemia, whether due to haemorrhage or not, are much the same in all cases. We find marked debility, languor, pallor of countenance, and a tendency to giddiness and faintness. There is inability for work, physical or mental. In the *Alimentary System*, the appetite is poor, and digestion unsatisfactory, and generally obstinate constipation is present. In the *Haemopoietic System*, besides the changes in the corpuscular elements in the blood, the haemoglobin index is always low. In the *Circulatory System*, the patient complains much of palpitation, and often of giddiness, which may be traced to circulatory weakness, and the extremities are apt to be cold and chilly. Haemic murmurs are common, and include a loud systolic murmur, well heard in the mitral, pulmonary, and sometimes the tricuspid areas, associated often with a loud *bruit de diable* in the veins at the root of the neck. In severe cases a definite degree of cardiac dilatation is present. These haemic murmurs, with the *bruit de diable*, have been described already under chlorosis. In the *Integumentary System*, the patient

frequently complains of swelling of the legs, and especially the ankles. Certain cases show a marked diminution of subcutaneous fat, and the patients may become rapidly emaciated if the anaemia is due to any wasting disease. In the *Reproductive System*, amenorrhoea and dysmenorrhoea are commonly present. In the *Nervous System*, neuralgia is a constant cause of complaint, and headache, especially in severe cases, is apt to harass the patient. Reference has already been made to the inability for mental work, and sleeplessness is extremely common, probably the result of irritation of brain centres from a deficient supply of normal blood. In the *Locomotory System*, mention must be made of the patient's debility and the muscular weakness.

The **Diagnosis** resolves itself largely into a question of the cause of the anaemia, and not its existence, and the careful investigation of the blood often helps in deciding this matter.

The **Prognosis** depends on the possibility of removing the cause of the condition. Certain cases do well, but it is always a serious matter to find an excess of lymphocytes in the blood, while gigantoblasts and macrocytes, so typical of the grave forms of pernicious anaemia, suggest a fatal termination.

Treatment.—Attempt to discover and then to relieve the cause of the condition. Should there be any alimentary error endeavour to correct it, investigate the condition of the teeth, the stomach, the intestine, and perhaps, above all, the diet which the patient is taking. Arrest haemorrhage by removing the causal factors where possible, and investigate in cases in which a toxic agent is suspected and try to eliminate it. Order plenty of fresh air, easily digested and suitable food, and, if possible, sunshine. As noted under chlorosis, the patient should be kept in bed if the heart is dilated, and until such dilatation has disappeared. The protosalts of iron have gained a well-deserved notoriety in connection with anaemia, and, in not a few cases, the addition of a small dose of arsenic is very helpful. It should not be forgotten that oxygen inhalations will greatly relieve the breathlessness and the discomfort of profound anaemia, even in cases in which satisfactory treatment is not possible.

II. LEUCOCYTHAEMIA

(LEUKAEMIA)

A DISEASE in which the white blood corpuscles greatly increase, the increase often being limited to one or more varieties of these corpuscles, and in one type myelocytes or marrow cells may be found in large numbers. There are also distinctive changes in the spleen, bone marrow and lymphatic glands, the degree varying in different types of cases. The disease was described by Hughes Bennett in 1845, and it is to him that we are indebted for the name leucocythaemia. From the large number of white blood corpuscles he described the disease as "suppuration of the blood."

There are two types of cases: (1) The Spleno-medullary Type, and (2) the Lymphatic Type.

1. The Spleno-Medullary Type

A chronic disease of the bone marrow in which, as the result of excessive proliferation of marrow cells, they overflow into the blood. It is possible that some of the myelocytes may be formed locally in other organs such as the liver, etc.

Etiology.—This type is more common during middle life, and apparently quite as often in males as in females. In women the climacteric is said to be the commonest period of attack. Malaria and syphilis are possible causes, and in some families there is a hereditary history, which, however, is not very satisfactorily proved. It is doubtful whether injury alone is responsible. A toxin might explain the condition, or the cell hyperplasia may partake of the nature of a neoplastic growth. In the meantime further research is necessary.

Pathological Anatomy.—The spleen is reddish-brown in colour, and is not inaptly described as resembling raw beef-steak. Its weight is usually 4 or 5 lbs., but may be anything from 2 to 18 or 20 lbs. There are generally some adhesions: on section the Malpighian bodies are invisible, the condition being a chronic hyperplasia, with thickening of the capsule. There are often numerous haemorrhages

in the cut surface, sometimes whitish lymphoid masses scattered throughout the organ, and infarcts may be seen in certain cases. The bone marrow presents a typical appearance generally resembling pus, although it may in some cases be reddish-brown in colour. The medullary canal is expanded, there is marked hyperplasia of the marrow instead of the usual amount of fat, and on microscopic examination, numerous myelocytes, nucleated red blood corpuscles, and eosinophile leucocytes are noticeable in addition to other cells. Many of the cells in the bone marrow show mitoses. The liver is generally enlarged, and the capillaries are seen on microscopic examination to be packed with leucocytes. The lung capillaries show similar changes. It is not so common, in this type of the disease, to find lymphoid nodules scattered throughout the organs of the body, although it is true that this type and the lymphatic type are sometimes coexistent.

The emaciation is extreme, and dropsy is sometimes present. After death the heart and veins are distended with clots, and these have a greenish-yellow appearance. There are not infrequently haemorrhages, sometimes petechial; retinal haemorrhages are not uncommon.

Clinical Features.—The onset is always insidious, the enlargement of the spleen, the dyspnoea, the palpitation, and the tendency to epistaxis being often responsible for bringing the case before the notice of the physician. The spleen is painful, and especially so on palpation, the splenic notch or notches can usually be distinctly felt, and the enlarged organ extends in the direction of the right iliac fossa. Perisplenitis is sometimes noted; and it is remarkable how the spleen may vary in size during the progress of the disease.

The blood is typical, the white cells increasing often from the normal 7, 8, or 10,000 in the cubic millimetre of blood to 50,000, 100,000, or many hundred thousands, and the proportion to the red blood corpuscles may be increased from the normal 1 in 500 or 1000 up to 1 in 10, 1 in 5, or even equal numbers. The number of white cells varies greatly from time to time. The characteristic white cells are unquestionably the myelocytes, which are large, mononuclear cells, containing generally neutrophile, sometimes oxyphile granules in the protoplasm. The nuclei are of large size in proportion to the cell, and of somewhat irregular shape

although generally oval. These cells constitute from 20 to 50 per cent of the leucocytes present, usually nearer the lower figure. Eosinophile or oxyphile leucocytes are increased absolutely, though not relatively. The ordinary polymorphonuclear leucocytes, while much increased in number absolutely, are often relatively diminished towards the terminal stages of the illness. Mast cells are always present, and usually in excess (3-12 per cent), and their appearance is the more striking, because they constitute a rare cell element in the blood.

The red blood corpuscles are reduced in number, usually to

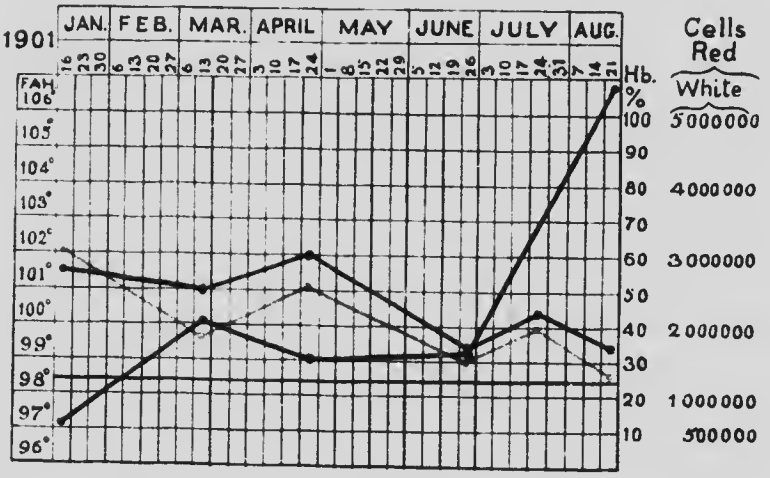

FIG. 25. - Blood Chart. Leucocythaemia. Spleno-medullary type. Fatal event. Black line indicates number of red blood corpuscles. Red line indicates haemoglobin percentage. Blue line indicates number of leucocytes.

about three millions in each cubic millimetre, while the presence of nucleated red cells, or normoblasts, and often also of gigantoblasts and red cells with fragmenting nuclei, is characteristic of this disease.

The blood-plates are sometimes stated to be much increased; they are certainly not reduced in number. The haemoglobin is proportionately diminished, the blood coagulates badly owing to the presence of albumoses; and leucin, xanthin, and other bodies due to the breaking-down of leucocytes are found in excess in the blood. Charcot-Leyden crystals may be seen in the blood in most cases, although they only precipitate on the slide. In the *Alimentary System* there is a tendency to nausea and vomiting, while diarrhoea is apt to be severe.

The liver is almost always enlarged. Bleeding may occur from gums, stomach, or other part of the alimentary tract. In the *Circulatory System*, the phenomena are mostly associated with the anaemia. There is palpitation, a rapid and soft pulse, and not infrequently oedema develops, although it is really of haemopoietic origin. In the *Respiratory System* there may be a final oedema of the lungs, and epistaxis is one of the common forms of haemorrhage met with in this disease. In the *Integumentary System* purpuric haemorrhages occur, and sometimes even local gangrene. In the *Urinary System* haematuria may develop, but the most important clinical feature is the presence of an excess of uric acid in the urine. In the *Nervous System* the headache, giddiness, and tendency to syncope are all due to the anaemia. Cerebral haemorrhage may occur, and leucocythaemic retinitis, often with haemorrhage, interferes seriously with the patient's vision. Deafness may also be present, due to haemorrhage or leucocytic effusions into the labyrinth.

A remarkable feature associated with the disease is the repeated occurrence of periods of pyrexia with apyretic intervals. The temperature, however, rarely exceeds 102° F.

Diagnosis.—An examination of the blood is amply sufficient in most cases, although it must be remembered that from time to time the number of leucocytes may greatly diminish, and apparently the spleen proportionately decreases in size.

Prognosis.—Life may be prolonged for a considerable time (generally 1 to 3 years), and recovery has even occurred, but the disease is a serious one, and a guarded opinion should always be given.

Treatment.—Arsenic is *the* remedy, and it should be administered in large doses, or rather in increasing doses, care being taken to obviate its irritant effect on the alimentary tract and the onset of neuritis. The best time to administer the drug is with food, and beginning with 5 minims of Fowler's solution of arsenic, the quantity may be pushed up to 15 minims thrice daily. Recently, good results have been obtained by applying the X-rays for 10 to 20 minutes every day, or twice or thrice weekly. The vacuum tube should be 4 to 6 in. distant from the spleen. It is important to allow time for the elimination of the products of destruction of the leucocytes

by stopping treatment occasionally. The bones or in other words the bone marrow, should also be exposed to the rays. Otherwise, the food, clothing, rest, and the general comfort of the patient, are the only matters which call for special care.

2. The Lymphatic Type

This type is met with in young persons, and was formerly considered to be of rare occurrence. There is no special etiological factor which is known to produce it.

There are two distinct varieties: (1) an *acute* form, in which the chief leucocytes present in the blood are the large hyaline lymphocytes; and (2) a *chronic* form, in which the small lymphocytes so greatly preponderate that hardly any other leucocytes may appear to be present in the film.

Pathological Anatomy.—While there may be some splenic enlargement, the chief lesion, other than in the blood, consists in enlargement of the lymphatic glands and the development of an abnormal amount of lymphoid tissue in many organs. These small lymphatic growths are well seen with the naked eye in the liver and kidneys. The bone marrow may also be replaced by lymphoid tissue.

Clinical Features.—The *acute* type of cases begins with weakness, often pyrexia and dyspnoea, and anaemia usually develops early. Haemorrhages are not uncommon, and the glandular enlargement if not great is definite. The cervical, axillary, and inguinal glands are involved. As a rule, in the acute cases the total number of leucocytes may not exceed 10,000 or 14,000 per c.mm., and in any case is rarely over 20,000. The cells are mostly large hyaline lymphocytes, although transitional cells may also be present, and such transitional cells are by no means uncommon in acute cases. Generally speaking, there are no myelocytes, and nucleated red blood corpuscles (normoblasts) are rare.

In the *chronic* type of case the onset is more insidious, and the weakness, dyspnoea, anaemia, haemorrhages, and glandular enlargements of slower development. The blood shows a great preponderance of small lymphocytes, and they may be so numerous that the leucocyte count may reach 60,000 to 100,000 per c.mm., and may even exceed the red blood

corpuscles in numbers. In the chronic type there are rarely transitional cells.

Diagnosis.—The presence of an excess of lymphocytes in the blood, especially if associated with enlargements of certain groups of lymphatic glands, renders the diagnosis clear.

The **Prognosis** is grave. The lymphatic type is always a serious one, and death may occur in *acute* cases in 6 to 8 weeks, and in the *chronic* in 6 months to 3 or 4 years.

Treatment must be on general lines. Probably arsenic should be tried, although results are not encouraging, and certainly X-ray treatment should be persevered with.

Chloroma

This condition is closely allied to the lymphatic type of leucocythaemia and also to sarcoma. The cases present the following characteristics:—(1) there are lymphoid deposits in the orbit, the periosteum of the skull, the temporal fossae, and other parts, causing exophthalmos and local swellings wherever the growths exist: 2 the glandular masses are bright grass-green in colour, although this somewhat startling appearance may be only noted after death, and the growths resemble in microscopic structure a lympho-sarcoma; (3) there is always profound anaemia of the lymphatic type, and haemorrhages into the skin and mucous surfaces are common; (4) there are lymphoid deposits in the bone marrow, in the lymphatic glands, and in the skin: the bronchial and mesenteric glands are almost always involved, other glands varying in this respect: (5) the age of the patient is invariably under 40 but the condition is most common in quite early life, while males have presented the larger proportion of cases.

The leading clinical features are palpitation, weakness, deafness with tinnitus aurium, facial paralysis, headache, giddiness, and sometimes apoplectiform attacks, and certain of these features are directly due to the growth of lymphoid deposits.

The result is always fatal, and generally within six months.

III. LYMPHADENOMA

(Hodgkin's Disease; Pseudo-Leukaemia; Malignant Lymphoma)

A disease in which the spleen and lymphatic glands undergo a peculiar hypertrophy, and in which secondary lymphatic growths develop in these and other organs.

Etiology.—The causation of this disease is still unknown. Syphilis, tubercle, local injury, exposure, over-exertion, and cold have been considered to be causes, and may predispose, although they cannot alone cause the condition. A toxin acting specially on lymph tissue seems a more probable factor. The disease is more common in young persons, and specially in males.

Pathological Anatomy.—The endothelioid and reticular cells of the lymph sinuses proliferate, and the newly formed spaces become packed with lymphoid cells. The lymphatic glands are greatly enlarged, and may be soft, but more often are hard and nodular; a relative excess of lymphocytes is associated with the cases in which the glands are soft. The spleen is enlarged, although not to the extent associated with leucocythaemia, and on section it shows numerous greyish-white lymphoid masses varying in size from a pea to a walnut. Not infrequently there is perisplenitis. Haemorrhages into the spleen are not uncommon, while sometimes infarctions occur. Lymphoid nodules may develop in many organs, including the lungs, kidneys, bone marrow, and alimentary tract.

Clinical Features.—The earliest manifestation of the disease consists in gradual loss of strength, with a degree of anaemia, while the glandular masses develop first in the neck, then in the axilla and groin, and eventually in the mediastinum and in other regions. At first the glands are discrete and freely movable, while later they become matted together and often adherent to the skin. These masses rarely suppurate, but they cause serious interference with many important structures by pressure, and in this way dyspnoea, difficulty in swallowing, grave interference with arteries, veins, and nerves, may rapidly develop. There is a tendency to epistaxis, to oedema of the feet, to bronzing of the skin in certain cases,

and to pains of a neuralgic nature, directly due to pressure on nerves. The tonsils, the thyroid, and the thymus are sometimes enlarged, and, as a result of the tonsillar hypertrophy, deafness may be induced by interference with the Eustachian tube. The lymphadenomatous masses differ from tubercular involvement of the glands by their tendency to push to one side important structures, such as the trachea, œsophagus, etc.

The spleen is found to be enlarged, although not to a great extent; the red cells in the blood are markedly diminished, and there may be a poikilocytosis, and even the presence of microcytes, with a proportionate diminution in haemoglobin, and a fall in the specific gravity of the blood, but the leucocytes are not increased in number, the only change being a relative increase of lymphocytes in a proportion of cases.

There is generally a tendency to pyrexial attacks, and gastro-intestinal disorders may be associated.

The **Diagnosis** is difficult, because the disease resembles *tubercle* and also *lympho-sarcoma*. In tubercle there is always caseation and the presence of tubercle bacilli, and, as a rule, tubercular glands do not displace important structures to anything like the same extent that lymphadenomatous growths do. A tuberculin test such as von Pirquet's should be positive in tubercular cases. As regards lympho-sarcoma, there are certainly cases in which it is difficult to distinguish between Hodgkin's disease and sarcoma. One sometimes sees lymphomatous growths, piercing the bronchial or tracheal walls in a manner suggestive of sarcoma, and the difference in histological appearances between this malignant form of Hodgkin's disease and lympho-sarcoma is not very apparent.

Prognosis. — Occasionally cases improve, and marked benefit may result from treatment, but not infrequently the disease remains incurable, and death may occur from exhaustion, or possibly as the result of pressure.

Treatment. — Arsenic is an important remedy. The X-rays should certainly be tried, and in a large number of cases their use has been very successful. Cod-liver oil and iron are also of value, and mercurial inunction has been employed, while Clifford Allbutt recommends tungstate of soda, which proved beneficial in one case, although it failed in others. Many

other remedies have been suggested, but none have proved of value.

IV. SCURVY

(SCORBUTUS)

IT is a morbid condition, characterised by increasing debility, anaemia, breathlessness on the slightest exertion, and sponginess of the gums, with a tendency to haemorrhages in various situations.

Etiology.—The disease appears to be the direct result of an insufficiency of fresh meat and vegetables, but it has occurred in almost epidemic form in Russia, and is said to be endemic in certain districts, which would suggest that it might be of infective origin. On the other hand, the sanitary conditions prevailing in the parts of Russia referred to, and the standard of hygiene of the people, leave much to be desired.

Whether the disease is due to the lack of potassium salts, or whether the absence of citrates, malates, and other organic compounds reduces the alkalinity of the blood, it is difficult to say with certainty. Such an explanation, however, appears to fit the circumstances of most cases of scurvy better than the theory that it may be due to a toxic agent in the meat or food partaken of, or a specific organism, as suggested by some Russian savants.

There seems little doubt that *predisposing* causes are ill-health, mental depression, as from confinement in prison or in besieged cities, and insanitary surroundings, as in poor and overcrowded parts of our great cities. The lack of vegetables alone is not sufficient to induce scurvy, although a modified form of the disease may result from a tea and bread dietary when associated with poverty and squalor.

Pathological Anatomy.—Haemorrhages are specially common and typical. They occur under the skin, under mucous and serous membranes, into muscles, under the periosteum, and sometimes even into joints. There is no microscopic or other discoverable lesion of the walls of the blood-vessels to account for these haemorrhages, and there is no leucocytosis, or any very definite change in the blood, besides

what has already been mentioned. The spleen is large, congested, and soft; there may be fatty degeneration of the heart muscle, the mucosa of the gums is spongy and ulcerated, and often the teeth are shed.

Clinical Features.—Debility and langnor are the earliest phenomena, and they may begin insidiously and advance slowly. Then the gums become spongy and bleed too readily, while the breath is fetid. The patient is pale and anæmic, the teeth become loose, and haemorrhages occur. These are petechial haemorrhages on the legs, sometimes larger haemorrhages into the muscles or under the periosteum, and they are often directly due to a blow or injury, the part injured becoming the site of an extravasation of blood. Coincidently, bleeding may occur from the mouth, nose, bowel, or kidney, and oedema of the ankles is a usual feature of this stage of the disease. Palpitation and dyspnœa on the slightest exertion are most troublesome.

Disturbances of the *Alimentary System* are not uncommon, especially dyspepsia, and sometimes diarrhœa. In the *Integumentary System* the brawniness of the skin over a large haemorrhage, even when deep-seated, is very characteristic: the petechial haemorrhages have already been noted. In the *Urinary System* the presence of albumin in the urine is common, but the phosphates are said in some cases to be increased, in others diminished. In the *Nervous System* the chief symptom is the depression of spirits almost amounting to melancholia, while in the *Locomotory System* there is always a tendency, when subperiosteal haemorrhages occur in young patients, for the shafts of the long bones or the epiphyses to be separated and to undergo necrosis, while joints may become fixed or absolutely ankylosed, as the result of haemorrhagic effusions. In cases which terminate fatally, extensive effusions, often haemorrhagic, may occur into pleuræ, pericardium, etc.

Diagnosis.—When scurvy appears on shipboard or in prisons, it is usual to find a number of the more weakly individuals attacked. In towns where insanitary surroundings, combined with a diet of tea and bread and butter, and no fresh meat and little milk, are responsible for the disease, the history of the dietary may help, while purpuric haemorrhages associated with spongy gums are distinctive.

The **Prognosis** is favourable, if proper treatment is carried

out, but when a case has gone very far downhill it may be difficult to effect a complete restitution to health.

The **Treatment** should consist of plenty of fresh meat (preferably given scraped owing to the state of the patient's teeth) and milk. Raw meat is more antiscorbutic than cooked. The mouth must be energetically treated with antiseptic solutions, such as permanganate of potash, chlorate of potash, or carbolic acid and glycerine, and in some cases a solution of silver nitrate (10 per cent) should be painted on the gums occasionally. Give lime or lemon juice not merely as a beverage, but as a medicine 3 or 4 times a day; also plenty of fresh fruit may be eaten, while later on fresh vegetables are of great value.

Scurvy is rare on shipboard, nowadays, when fresh meat can be carried in refrigerators, and the perpetual salt junk is no longer a feature of sea life, but scurvy can be prevented if, after ten days on salt meat, the crew of a vessel are placed on the regulation half-ounce of lime juice mixed with rum.

V. SCURVY-RICKETS

(INFANTILE SCURVY)

WHEN it is remembered that milk loses its antiscorbutic properties after it is boiled or peptonised, it is easy to understand how readily a child fed on Swiss milk, from which most of the antirachitic properties have also been removed, may develop a mixture of scurvy and rickets. Scorbutic women suckling their children are apt to induce the disease in their offspring. Many kinds of patent foods for infants contain no proper antiscorbutic elements.

A child showing the disease well developed is generally 6 to 18 months old. It is debilitated, peevish and fretful, and particularly resents being moved or taken on the knee. All attempts at handling the limbs and especially the legs cause great pain. There are swellings due to subperiosteal haemorrhages on the legs and thighs, on the ribs, and often near the orbit, and the epiphyses may be separated from the bones. There is profound anaemia, often very marked alimentary disturbance, and sometimes diarrhoea, while the

temperature may rise to 101 or 102° F. Sometimes the gums are spongy, but only, as a rule, after the teeth have appeared.

The **Prognosis** is good if the case is treated soon enough and energetically.

The **Treatment** consists in giving the child *fresh* milk and for a time at least, a teaspoonful or thereby of raw meat juice, or a little potato-cream, which consists of mashed potato made up with cream or milk, several times daily. A small allowance of orange juice or fresh fruit is also beneficial.

It is important to remember what has just been stated, that boiled and peptonised milk, and many patent foods, have little or no antiscorbutic properties, and therefore, where children require to be fed on such foods, they ought daily to get a little raw meat juice, fruit juice, or other agent rich in antiscorbutic properties.

VI. PURPURA

An affection of the blood or possibly the blood-vessels, in which haemorrhages occur under the skin, under serous membranes and elsewhere. They are due to toxic conditions; they are specially common in certain fevers, in connection with rheumatism, and almost any disease in which very profound anaemia or cachexia is produced.

Etiology.—Purpura is therefore not so much in itself a disease, as a sign of a number of different diseases. Such haemorrhages are most common in rheumatism (in which a special form may be met with), in pyaemia, malignant fevers and any serious blood disease such as pernicious anaemia and leucocythaemia, while many poisons are capable of producing generalised or local haemorrhages. Probably purpura neonatorum falls under the division of toxic purpuras. Where an individual is bitten by a deadly snake, one of the leading features is a remarkable diapedesis of red blood corpuscles into the tissues of the limb or part of the body bitten. Phosphorus-potassium iodide, sometimes mercury, quinine, and many other drugs, may produce a mild type of purpura. Cachexia, whether the result of tuberculosis, cancer, a severe type of Bright's disease, or other condition, may, and especially in certain

patients, give rise to purpuric haemorrhages. There is a form of purpura to which the term "neurotic" has been applied, and hysterical stigmata are instances of this remarkable manifestation.

A condition due to so many varied causes may be met with at almost any age, but is certainly more common during early life and adolescence.

Pathological Anatomy.—Many theories have been formulated by way of explanation for purpuric haemorrhages. They are probably the result of some lesion of the vessel walls, or a change in the blood; it has been suggested that the cement substance between the cells in the vessel walls may be defective, and may therefore permit of transudation of red blood corpuscles. But it is probable that the larger haemorrhages, met with in the more severe types of purpura, are due to actual rupture of blood-vessels. In cases of purpura most observers record a great diminution in the number of blood-plates. Recent researches show that the blood-plates contain prothrombin, which is necessary for the production of blood clot; but while the clot formed by the blood in purpura is less firm than the normal, there is little delay in the coagulation time even in cases with severe haemorrhage.

Anaemia is an almost constant feature, and the results of anaemia may be noted in the different organs, while, owing probably to the excretion of blood pigment by the kidney, nephritis of a mild degree is occasionally present.

Clinical Features of the different Types

1. *Purpura Simplex.*—In this condition small red spots appear on certain parts of the body, and especially over the legs. They are very common in children and in young persons. They appear to be due in some measure to exercise, and they develop under the skin of the most dependent parts of the body, that is, the legs. The little haemorrhages come out in crops, they are not raised above the skin, and on pressure the red spot does not disappear. The effused blood undergoes the usual pigmentary changes seen in an ordinary bruise. The patient may be slightly out of health, very generally owing to some other associated condition, and sometimes has a history of rheumatic pains in joints or muscles.

2. *Purpura Haemorrhagica.*—This is a much more serious form, in which the patient not infrequently dies of hæmorrhage. The hæmorrhages are extensive and the mucous membranes bleed freely. The nose, mouth, eyes, stomach, bowels, kidneys, and the female genital organs may all bleed; and not infrequently hæmorrhages are found in connection with pleuræ pericardium, meninges, and elsewhere. The disease is more often met with in young and delicate females, and is preceded by a few days of ill-health. The patient rapidly becomes anæmic, and suffers from severe palpitation and breathlessness. The temperature is generally raised and may reach a high figure. Occasionally hæmorrhages occur into the joints, associated with much arthritic pain: vomiting is also not uncommon, and albuminuria appears in an amount out of all proportion to the blood present in the urine. Many cases may be designated as mild purpura hæmorrhagica in which the hæmorrhage, although serious at the time, is not alarming, but in severe cases the prognosis is grave in the extreme.

3. *Purpura Rheumatica, or Peliosis Rheumatica or Schönlein's Disease.*—This type is specially associated with arthritic pains. With a varying degree of pyrexia, malaise, aching pains in the legs, and often a severe sore throat, bright red spots appear on the lower part of the legs. After 2 or 3 days another crop of spots develops with a recurrence of joint pain. The legs are often slightly swollen and tender, and the level of the spots may rise higher up the leg with each successive attack. There is not infrequently a tendency to urticarial wheals, and erythema nodosum is sometimes associated with this type of purpura. The condition is met with in patients suffering from acute or subacute joint pains, and generally between the ages of 20 and 40. We have generally been in the habit of considering these joint-pains as rheumatic, although many authorities hold that they are not of that nature and do not yield to treatment by salicylates. Further, it is very rare in cases of undoubted rheumatism to find peliosis or purpura rheumatica occurring.

4. *Henoch's Purpura.*—This is a form of purpura in which there are marked abdominal symptoms; vomiting, colic, and intestinal hæmorrhage being associated with purpura and joint swelling. Sometimes the affection begins with arthritis, in other cases the gastro-intestinal phenomena originate the

attack. The colic pains are intense, and there are repeated attacks lasting for weeks or months with intervals of rest. Sometimes albuminuria or even nephritis is present, and the purpuric spots, while most common on the extremities, may also occur on the body and face.

This type of purpura is apparently associated with rheumatism, and occurs more frequently in childhood and adolescence.

Diagnosis.—The account just given of the various types of purpura has not exhausted the list of possible varieties, and nothing has been said with regard to the purpuric eruptions due to *certain drugs* such as potassium iodide. It is necessary, in diagnosing the particular type of the affection present in any given case, carefully to exclude the probability of the affection being due to the administration of some drug. We must also investigate the condition of the blood, and the possibility of the haemorrhage being associated with the *haemorrhagic* or *malignant type* of *fevers*. *Scurvy* in which haemorrhages are so common must also be considered. If we take by themselves purpura simplex and purpura haemorrhagica, and exclude the possibility of their being due to rheumatism, they might be designated idiopathic, and the absence of any definite etiological factor would be an important point in the diagnosis of these two forms.

Prognosis.—In most cases recovery occurs, but certainly in 14 to 15 per cent of cases of purpura haemorrhagica death is inevitable, and a case of this kind is more grave the older the patient happens to be.

Treatment.—The patient should be confined to bed, and in rheumatic cases the salicylate group of remedies should be ordered. Probably turpentine administered as oleum terebinthinae in 10 to 20 minim capsules is one of the safest and best of remedies, while chloride of calcium in 20-grain doses, ergot, hamamelis, the mineral acids, the perchloride of iron, and lead salts, may all prove useful in different cases. The application of iced cloths, ice-bags, and sometimes pressure over bleeding points, arrests serious haemorrhage, and in the case of Henoch's purpura, while iron and arsenic should be administered internally, an ice-bag placed over the abdomen greatly relieves the severe colic pains.

Arsenic is worth a trial in many cases, and when cure has been effected, care should be taken to prevent any excessive

exercise, and to raise the standard of the patient's general health. We have found considerable advantage from the use of adrenalin and other forms of suprarenal extract in checking even severe cases of purpuric haemorrhage.

VII. HAEMOPHILIA

A CONSTITUTIONAL tendency to haemorrhage.

Etiology.—The hereditary tendency is transmitted through the female line to the male offspring, and may run through many generations. Occasionally isolated cases of the disease occur, and apparently it is associated with a peculiarly fine smooth type of skin. The proportion of males to females affected by the condition is about 9 to 1.

As regards race, Jews suffer in a remarkably large proportion of cases. The condition may show itself at an early period in childhood, as for example during the rite of circumcision, but as a rule it does not appear until the end of the first year.

Pathological Anatomy.—In the case of persons who die from excessive bleeding, perhaps as the result of a trivial operation, such as the extraction of a tooth, the post-mortem changes are generally negative; but there is little doubt that the cause of the condition is associated with delayed coagulation of the blood. The walls of the blood-vessels in this disease do not contain as much thrombokinase, which is an essential for blood-clot formation, as in health, and therefore clotting is greatly delayed, and it may also be that there is an embryonic error, most likely in the inner coat of the vessels. In a certain number of cases joint inflammations are met with.

Clinical Features.—(1) A cut or injury may cause excessive haemorrhage.

(2) Haemorrhage may occur, without previous injury, from the nose, mouth, stomach, bowels, urethra, lungs, etc.

According to Grandidier, of 334 cases of bleeding in haemophilia—

Epistaxis was present in	. . .	169 cases.
Bleeding from the mouth was present in		43 „
„ „ stomach	..	15 „

Bleeding from the bowels was present in			36	cases.
„	„	urethra	„ 16	„
„	„	lungs	„ 17	„

In many of these cases it was rather a capillary oozing than the rupture of one large vessel. The bleeding may last for hours or days, and, if not checked, has been known to cause death. As may be readily supposed, the slightest injury, such as a knock or bruise, may give rise to severe bleeding, and in extreme cases faintness may result, or a serious anaemia may follow repeated loss of blood.

Sometimes arthritis, especially of the larger joints, results from severe haemorrhage into the joints. The knees and elbows are most apt to be affected, and their condition may in time cripple the patient.

No special change has been noted in the blood, except delayed coagulation.

Diagnosis.—The family history, and especially a haemophilic history on the mother's side, is all-important. *Scurvy* and *purpura* may so far resemble this condition, but cannot well be mistaken unless in exceptionally mild cases.

Prognosis. Death may result from haemorrhage. Apparently the tendency may, to some extent, be outgrown.

Treatment.—Guard persons who suffer from this condition from injury of all kinds. Remember that dental and surgical operations, blistering, and even vaccination are attended by special risk. Try to brace up the general health by cold bathing, the administration of iron, cod-liver oil, and other tonics.

For the arrest of the bleeding, pressure will be found of most value. Styptics, such as tincture of the perchloride of iron, are useful, and solutions of gelatine, injected subcutaneously, have been recommended. Wright urges the use of calcium chloride or calcium lactate in 15-grain doses thrice daily, while ergot or ergotine should not be neglected in severe cases.

A somewhat thankless task, which, however, would do more than anything else to stamp out the disease, is the effort to dissuade the daughters of haemophilic families from marrying.

VIII. DISEASES OF THE SPLEEN

(1) Splenic Anaemia

A somewhat rare form of anaemia, characterised by enlargement of the spleen and liver, with marked diminution in the number of leucocytes, a tendency to haemorrhages and generally pyrexial attacks.

Etiology.—It seems probable that the disease is directly due to the spleen, because removal of the spleen has in certain cases resulted in cure. There is reason to suppose that the disease is due to a chronic intoxication. It is most common during adult life, and especially in males.

Pathological Anatomy.—The spleen in this disease usually weighs 3 to 5 lbs., although a much greater weight has been recorded. There is increase of fibrous tissue with atrophy of Malpighian bodies, while haemorrhages, infarctions, and perisplenitis are occasionally present. Cirrhosis of the liver has been recorded in cases described by Banti, and it is one of the later phenomena in splenic anaemia. There may be increase of erythroblastic tissue in the bone marrow similar to that seen in any severe anaemia. There is not the excess of free iron pigment in the liver and other organs which is associated with pernicious anaemia.

The blood generally shows a typical leucopenia or diminution in the number of white cells, while, as the result of the profound anaemia, there may not only be diminution in the red cells and haemoglobin, but also a poikilocytosis.

Clinical Features.—There is usually a slow and insidious onset, the gradually increasing anaemia being associated with enlargement and tenderness of the spleen, and as the disease becomes more advanced, haemorrhage is apt to occur from nose, stomach, bowels, and elsewhere. There is often gastro-intestinal irritation with severe dyspepsia and diarrhoea, and in the terminal stages, when the liver is cirrhotic, jaundice is not uncommon, and ascites develops. Rapid loss of strength with dyspnoea and palpitation, increasing during the periods of pyrexia, soon terminate the case.

Diagnosis and Prognosis.—The examination of the blood renders the diagnosis of this disease simple because the typical

characteristics of *pernicious anaemia* and *leucocythaemia* are absent, while in *Hodgkin's disease* the lymphatic glands are more markedly affected and the anaemia is not so profound. The association of an enlarged and painful spleen with leucopenia [in these cases in which the latter is undoubted] is almost alone sufficiently diagnostic, and prevents any confusion with *malignant disease*, *ague*, *syphilis*, *tubercle*, or other condition.

The prognosis is bad, the patient's tenure of life being rarely more than a matter of 1 or 2 years, unless in extremely chronic cases, when life may be protracted for years.

Treatment.—Arsenic should be tried, but in most cases splenectomy would be the accepted treatment, were it not such a dangerous operation. Soon after the removal of the spleen the leucocytes increase in number, but so high has the death-rate from the operation been in the past, that probably all other measures, such as those suggested under the treatment of Hodgkin's disease, etc., should in the first place be thought of. Recent statistics of splenectomies are, however, more favourable.

(2) Polycythaemia with Splenomegaly

In 1892 this symptom-complex was first described. It appears to be due to a hyperplasia of the erythroblastic bone marrow.

Etiology.—Nothing can be satisfactorily stated as regards the etiology, except that the disease occurs in both sexes, and generally during adult life.

The **Clinical Features** include the deep-purplish colour of the patient's face, and particularly the lips, which sometimes look strikingly cyanosed, and the evidences of cerebral congestion, such as headache and giddiness. The spleen is often enlarged, reaching to the umbilicus, is sometimes tender, and the patient may complain of dragging sensations due to its weight. The red blood corpuscles number 10 to 12 millions, and the haemoglobin is 120 to 130 per cent. There is sometimes a leucocytosis, with relative increase of polymorphs.

The **Diagnosis** is determined by the examination of the blood, and the **Prognosis**, although not immediately unfavourable, does not generally imply a life of over 5 or 6 years.

The **Treatment** is not satisfactory. Probably the X-rays will be found of more advantage than anything else.

Arsenic is not of much use, and although the iodides have been recommended, they have proved disappointing.

(3) Other Conditions

A *movable spleen* has been referred to under enteroptosis; it is not a very common lesion. Occasionally the spleen *ruptures*, more particularly where there is great enlargement of the organ, as in malaria, although sometimes, as the result of a crush, the capsule of the healthy spleen has been torn, causing serious and often fatal haemorrhage. *Splenic infarctions* may suppurate and cause peritonitis. *Enlargement of the spleen* is not uncommon in certain diseases, and especially in leucocythaemia, in Hodgkin's disease, in malaria, and kala-azar, while the enlarged spleen of a splenic anaemia is characteristic of that disease.

IX. DISEASES OF THE SUPRARENAL GLAND

(Addison's Disease)

A disease of the suprarenal glands, associated with pigmentation of skin, advancing debility, cardiac weakness, and marked gastro-intestinal phenomena. It was described by Addison in 1854.

Etiology.—It is commoner in males than females (2 to 1). The age is generally about thirty. The suprarenals most frequently show caseous tubercle in this disease, and they may be the only site of tubercle in the body. A strain or abdominal injury has been stated to be a possible cause.

Pathological Anatomy.—The suprarenals are found to be affected by:—(1) Caseating tubercle, the commonest condition present. Tubercle bacilli have been discovered in the nodules which are confined at first to the medullary part of the gland; later the cortex is destroyed, and the suprarenals are often adherent to neighbouring tissues. (2) Atrophy with or without interstitial inflammation. (3) Malignant disease involving directly or indirectly the suprarenal bodies. (4) Haemorrhage into the suprarenals. In a few cases the semilunar ganglia are said to be affected, with changes in the cells (pigmentation, atrophy, etc.), the suprarenals being healthy. The pigmenta-

tion of the skin and mucosa is found to be in the cells of the stratum Malpighii.

Pathology.—There must be an internal secretion from the suprarenals, and very probably, where the suprarenals are not found affected in cases of Addison's disease, the channels by which this secretion is conducted may be blocked. Possibly the abdominal sympathetic may be in some way responsible for the vomiting, feeble heart's action, and so forth.

Clinical Features.—A slow and insidious disease, with advancing debility and profound anaemia, feeble action of the heart, gastro-intestinal irritation, with marked attacks of nausea, vomiting and diarrhoea, and typical bronzing of the skin. The face, neck, backs of the hands and fingers, axillae, nipples, and genital regions are all dark, and the mucosa of the mouth and vagina is often affected. Pressure of a garter induces a greater degree of coloration, and irritation, such as that due to skin parasites or a carious tooth, will determine deeper local pigmentation. Moles are usually found on the arms, back, face, etc.: they consist in deeply pigmented spots which are not raised. Rarely patches of leucoderma are associated with the pigmentation. There is much palpitation, while sighing and persistent yawning, due to heart weakness, often occur. The blood-pressure is low. Pains in the back and headache are common.

Diagnosis.—The pigmentation of *pediculosis*, *pregnancy*, *pelvic disease*, *malignant disease of the abdomen*, *chronic phthisis*, and *malaria*, is never so extensive, and that due to *argyria* (silver poisoning) is quite distinctly bluish in colour. In *melanotic tumours* there is very exceptionally deep and general pigmentation of the skin, while in *exophthalmic goitre* and in *diabetes mellitus* pigmentation is sometimes induced. *Arsenic*, when taken for a protracted period, may cause intense pigmentation. It should be remembered that soap and water has removed pigmentation supposed at first to indicate Addison's disease.

Prognosis.—The duration is usually a matter of months, or rarely years with constant advance in the symptoms.

The **Treatment** is very unsatisfactory. Arsenic and strychnine are invaluable. Try, cautiously, some preparation of the suprarenal gland, preferably dried extract in tabloid form (gr. 1-3). Treat vomiting with bismuth and hydro-

cyanic acid: diarrhœa must be promptly checked with suitable remedies.

X. TUBERCULAR DISEASE OF LYMPHATIC GLANDS

THE tubercular glands so frequently seen in the neck belong to the domain of surgery, and do not demand any description here: but the involvement of mesenteric glands requires a brief description

Tabes Mesenterica. This condition is often associated with tubercular ulceration of the intestine and tubercular peritonitis. The glands involved are the mesenteric and also the retroperitoneal. The disease is common in children, and the clinical picture is very striking. There is great interference with nutrition, the patient is emaciated and anæmic, but often has the most extraordinary appetite for food. The abdomen may be distended or, less frequently, flattened. Generally definite nodules can be made out on palpation, and pain, or at least discomfort, is elicited to the touch. The child suffers from colicky pains, and diarrhœa is troublesome. The clinical features of tubercular peritonitis might be repeated here, because they are characteristic of tabes mesenterica.

The **Prognosis** is extremely bad, although the patient often lives for a wonderfully long time: the **Treatment** must be on general lines. Tuberculin is well worth trying in these cases.

XI. DISEASES OF THE THYROID GLAND

1. GOITRE

ENLARGEMENT of the thyroid gland, which may be *Sporadic* or *Endemic*. The *Sporadic* form is peculiar to women. The *Endemic* form occurs in certain districts, as, for example, in Derbyshire in England Derbyshire Neck in parts of the south of Scotland, in Switzerland, and Germany, and especially amongst the inhabitants of narrow valleys in mountainous regions The goitre varies in nature, but there is generally a great excess of colloid material, with much new formation of gland tissue In a second type of cases cysts form, often of

large size; and in a third group there may be great vascular dilatation, which constitutes the most obvious pathological feature.

The *Endemic* type is found in both men and women where the disease is prevalent, and persons going to a goitrous district may become affected just in the same way as the inhabitants. The explanation is doubtful; whether it is due to limestone water or to lack of sunshine during the long winter months, cannot yet be determined.

Clinical Features.—The enlargement often affects one lobe alone, or one lobe more than the other, or sometimes the isthmus of the gland. In many cases the goitre grows slowly, and, so long as it is small, causes no inconvenience to the patient, but where it is large it may compress the œsophagus or the trachea, and so give rise to great distress. It is certain that sudden death is more common in goitrous patients, and therefore in their case surgical operations should not be lightly undertaken.

Prognosis.—The disease is compatible with long life, although it must be remembered that a goitrous thyroid may be associated with myxœdema. Where the gland causes pressure, the prognosis is only favourable when operative interference is possible.

Treatment.—In most instances operation should be considered, because no drug is of much value. In cases in which the gland is not active, thyroid extract should be given, and many authorities recommend the use of iodine externally.

(2) Myxœdema

A constitutional disease, dependent on the loss of the function of the thyroid gland.

Etiology.—The disease may be due to complete, or almost complete, removal of the thyroid gland, or suppression of its normal function. It is present in cases of absence of thyroid, such as one finds in cretinism, while myxœdema may be associated with disease of the gland, such as goitre, and more rarely with exophthalmic goitre.

Women suffer more severely than do men (6 to 1 at least) and there is a tendency for the disease to run in families.

Pathological Anatomy.—The thyroid gland may be absent altogether, or it may be goitrous, or the site of a tumour or cyst. In most cases there is atrophy of the secreting gland structures, and the skin is markedly thickened, the connective tissue being increased in, or near, the hair follicles and also the sweat and sebaceous glands. This accounts for what has been aptly described as a solid oedema, and also for the loss of hair, want of perspiration, and the dryness of the skin generally.

Apparently thyroid secretion is essential for the activity of brain centres; and not merely do we find mental apathy, but slight degenerative changes have been found in cerebral cells.

The pathology of the condition, although not perfectly understood, appears to be fairly clear. The removal of the thyroid, experimentally, causes a condition closely resembling myxoedema, and the administration of thyroid extract, or transplantation of a portion of thyroid, remedies the condition. It should be remembered that there are para-thyroid glands, and that if these are active, they can carry on the thyroid functions, and it is possible that in cases where removal of the thyroid has not produced myxoedema, these glands have been left behind. The para-thyroids are involved in practically every disease in which the thyroid specially suffers.

Clinical Features.—The disease develops slowly, and after operation necessitating complete removal of the gland, weeks or months may elapse before myxoedema appears. The skin becomes swollen, but is firm and inelastic, and this causes loss of expression, dryness of the cuticle loss of hair, and a marked apathetic change in the demeanour of the patient. The features are coarser, the lower eyelids often have a baggy look, and there is a yellowish or waxy appearance of the face, with generally a reddish patch of colour on each cheek. The hands become spade-like owing to the swollen fingers; marked chilliness is usually complained of. The tongue becomes swollen, making speech difficult, and the voice is thick and monotonous, while the mental functions are slow and dull, and the memory extremely defective. Generally speaking, the pulse-rate falls below normal, the temperature is sub-normal, and there is a definite, if not very marked, degree of anaemia. One of the frequent peculiarities of the patient, when

the disease is well developed, is excessive irritability. Tetany may follow thyroidectomy.

The patient often falls a victim to pneumonia, tubercular disease, or other complications.

Diagnosis.—The appearance of the patient, and the solid oedema of the skin, are so distinctive as to render a mistake almost impossible.

Prognosis. The disease progresses slowly, and if untreated would eventually terminate in death.

The **Treatment** is most satisfactory. Thyroid extract should be administered in 3 to 10 grain doses thrice daily, beginning with 3 grains and increasing slowly and cautiously. The improvement is often rapid, the skin becomes softer, and all the functions greatly improve, so that, after a period of 6 or 8 weeks, it may be almost impossible to believe that it is the same patient who is being treated.

Care should be taken not to exceed the dose for which the patient is adapted, because a rise of temperature, cardiac weakness, and possible collapse, may result from any excess of thyroid extract. Once the patient is cured, the effect should be kept up by occasional small doses of thyroid extract every 2 or 3 days. It is well to bear in mind that much benefit accrues from massage of the skin, and from attention to diet, as well as to the general health and comfort of our patient.

(3) Cretinism

This disease corresponds to myxoedema, only it occurs at birth. There are two varieties: (1) *Sporadic*; (2) *Endemic*.

(1) *Sporadic Cretinism.*—In these cases there is atrophy or absence of the thyroid gland, and as a result an arrest of development both physical and mental. The disease appears to be due either to the absence or the atrophy of the thyroid, and the causes which might account for the condition are very varied, and include syphilis, alcoholism, and maternal impressions, together with delicacy and consanguinity of the parents.

(2) *Endemic Cretinism* is met with in certain districts, and especially where there are narrow valleys in which a minimum amount of sunshine is present during the winter months. Glacier water has also been described as a possible

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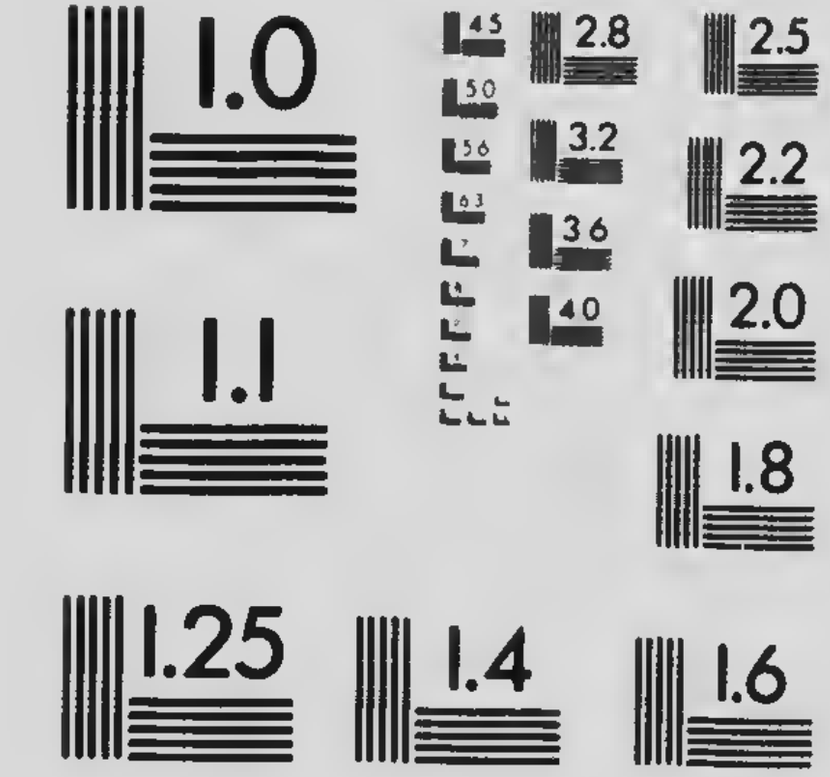
(ANSI and ISO TEST CHART No 2)



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factor; but there is undoubtedly, in addition, something in the consanguinity of parents even in these endemic cases. The disease is most prevalent in certain valleys in Swiss valleys, but is also common in the valleys of the Pyrenees, Italian mountains, and some other mountainous regions. It may be associated with the development of goitrous tumours.

The **Clinical Features** of these two types of cases practically correspond. The subject of cretinism is badly developed and stunted, and the disease manifests itself during the second year of life. The face is proportionately large, as are also the hands and feet. The alae nasi are thick, and the nose is broad, and often lacks a bridge. The tongue is large and may hang out of the mouth: the hair is thin and dry, and in older patients tends to fall out. The teeth form badly and decay at an early period: the skin has a peculiar yellow and waxy appearance, and is wrinkled: the abdomen is prominent and pendulous, and in very marked cases the patient may be unable to stand straight. The temperature is subnormal. There is another characteristic feature, namely, large pads of fat seen in the supraclavicular regions. The mind rarely develops, and imbecility is usually present. Some cretins are excessively irritable and capricious; others are more amenable to the influence of education. It is difficult to guess the age of a particular patient because growth ceases, leaving the individual stunted and dwarf-like. Life may be prolonged for many years, and death is generally due to some superadded disease.

The **Diagnosis** is easy in most cases, and it is chiefly in children, who merely show a slight degree of the disease, that there is any difficulty. *Achondroplasia* and other forms of *infantilism* have only superficial resemblances to cretinism.

The **Prognosis** depends largely on the age at which the patient comes under treatment.

The **Treatment** consists in the administration of thyroid extract, care being taken to avoid too constant administration. It should be remembered that the cretin may grow to a considerable extent, long after the age of puberty, under the influence of thyroid, and if time is not given for the bones to become strengthened, they are apt to give way under the increasing weight of the body.

4. Exophthalmic Goitre; Graves' Disease; Parry's or Basedow's Disease

A disease characterised by enlargement of the thyroid, prominence of the eyes or exophthalmos, and rapid action of the heart.

Etiology.—There may be a neurotic family history, but there is usually, in addition, a history of some mental or moral shock occurring in an individual below par physically. It is much more common in females, and specially between the ages of 20 and 30 years.

Pathology and Pathological Anatomy.—There is increased activity of the thyroid gland, and the typical clinical features can be produced, to a certain extent, by excessive doses of thyroid extract, while in a patient with the disease the symptoms are aggravated by the administration of thyroid. The clinical features are also exactly the antithesis of what is met with in myxoedema.

There is increased development of thyroid-gland tissue, with mucinoid change in the colloid contents of the gland spaces. The thyroid is enlarged, often more markedly on one side, accessory thyroids may also be enlarged if they are present, and the thymus frequently persists. The enlarged thyroid may be soft or firm, and there is a tendency to increase of connective tissue, which may in time almost entirely replace gland tissue, thus explaining cases which terminate with myxoedematous symptoms. There are no constant changes in the cervical sympathetic or in any other nerve centre. Cardiac dilatation is very common, and may hasten a fatal result.

Clinical Features.—It may develop slowly and insidiously, or with startling rapidity. The acute cases, which are rare, show, together with the typical features, intense gastro-intestinal disturbance and often delirium.

The chief clinical features are the following:—

1. *Tachycardia*, which usually develops early, but varies greatly in degree, and is much influenced by nervousness or excitement. The pulse-rate may be only 100, or it may be 150, 200, or more per minute. There is excited action of the heart, as shown by the visible cardiac pulsation and the throbbing arteries. Palpitation is associated and may be very distressing. The heart-sounds are loud, and there is

generally capillary pulsation, a wave being even seen in the peripheral veins. The heart often dilates owing to strain.

2. *Exophthalmos* develops soon after the tachycardia. Both eyes bulge, but generally one more than the other. The protrusion may be so extreme as to prevent the lids from covering the eyeballs during sleep, and the white sclerotics show above and below the iris.

Associated with the exophthalmos are the following phenomena: (*a*) Von Graefe's symptom, which is the lagging behind of the upper lid when the patient is asked to look downwards, and which is due, it has been thought, to the non-striped muscle fibres of Muller in the upper lid. (*b*) Stellwag's sign, which consists in the retraction of the upper lid, often very pronounced, and due to spasm of the levator palpebrae muscle. Closely connected with this is the fact that the patient winks very seldom as compared with a healthy individual. (*c*) Moebius' sign is the difficulty which the patient experiences in converging the eyes, when asked to look at a near point. (*d*) Joffroy's symptom is absence of wrinkling of the forehead, when the head is bent down and the patient looks upwards. It is very often present. There is rarely any defect of vision, and only in exceptional cases does conjunctivitis or corneal irritation appear. The prominence of the eyeballs has been variously explained by such theories as over-filled venous sinuses, excessive amount of fat in the orbit, and contraction of the non-striped muscle fibres of Müller in the membrane covering the spheno-maxillary fissure

3. The *enlargement of the thyroid*, as already stated, is often more obvious in one lobe. The gland frequently pulsates, and a loud hum, sometimes systolic, but usually continuous, with systolic increase of sound, is generally present. A palpable thrill is felt with the hand over the thyroid in these cases.

4. A *fine tremor* is typical, and is well seen in the arms and legs.

5. *Gastro-intestinal symptoms* are not uncommon, either gastric irritability with vomiting, or diarrhoea, or both at one time, and severe attacks may occur, sometimes threatening life.

There are many other less constant clinical features, including anaemia and excessive perspiration, and, associated with this, there is great reduction in electrical resistance.

Pigmentation of skin, trophic changes in skin, hair, and even the nails, slight albuminuria, sometimes glycosuria, and irritability of temper may also be present.

The first four clinical features are typical, although one or other may be awanting, and the results of cardiac dilatation are often present as the disease progresses.

Diagnosis.—The four typical symptoms render recognition of the disease easy, and even when one of these is absent, there should be little room for mistake.

Prognosis.—Mild cases often recover well, while chronic cases, after a period of definite symptoms, may undergo more or less complete cure. Advanced cases are very hopeless, and death may be due to cardiac or gastro-intestinal complications.

Treatment.—In mild cases, bodily and mental rest with the administration of tonics often effect a cure. Severe cases are most unsatisfactory from the point of view of treatment. Try absolute rest with freedom from excitement in every case. Give phosphate of soda in 1 drachm doses thrice daily, and persist with it for 6 weeks or 2 months. Digitalis and strophanthus are often beneficial for cardiac complications, and iron and tonics should be administered. Antithyroidin (the serum of thyroidectomised rams) and Rodagen (the dried milk of thyroidectomised goats) are very disappointing. Bromides, belladonna, ergot, and many other drugs have been recommended. Galvanism and faradism have been applied over the thyroid and the cervical sympathetic, but are rarely of much use. Do not operate until medical treatment, with rest, has been tried without success. The operative death-rate, until recently, has been high, but too long delay is undoubtedly responsible for part of this. The surgical procedures recommended include removal of the whole or a part of the gland, or ligature of the isthmus. Do not excise the superior cervical sympathetic ganglia, an operation which certainly removes the exophthalmos, but fails to relieve the other symptoms. An ice-bag applied over the heart is of great benefit for the palpitation in some cases.

XII. AFFECTIONS OF THE THYMUS GLAND

The thymus, which is apparently necessary for the child up to the end of the second year, gradually atrophies, and,

in most cases, cannot be recognised by the time puberty is reached.

Persistence of the gland has been held responsible for a number of affections, and more especially if there is actual hypertrophy. Laryngismus stridulus, asthma, and even whooping-cough are amongst the conditions which have been referred to the persistence and hypertrophy of the thymus, and an enlarged thymus can certainly produce difficulty in swallowing. It is a remarkable fact, in connection with hypertrophy of the gland, that sudden death has occurred in not a few persons in whom the thymus has not merely persisted, but was greatly hypertrophied, and it is possible that a hypertrophied thymus adds to the danger of general anaesthesia, by causing pressure on the bronchial tubes or on the vagi nerves.

Recently, enlargement of the thymus has been associated with exophthalmic goitre, although the relationship between the two affections has not yet been made clear.

Tumours of the thymus, sometimes sarcomatous, and abscesses in the gland have been recorded, but they are rare.

XIII. AFFECTIONS OF THE PITUITARY GLAND

Acromegaly

A chronic disease in which portions of the body, especially the hands, feet, lower jaw, and more rarely other parts, become enlarged.

Etiology.—It occurs in both sexes and in all races, and generally begins in the 3rd decade of life. Forty per cent of cases are above six feet in height, and about 20 per cent of giants are acromegalic, although acromegaly occurs occasionally in dwarfs. Rheumatism, mental worry, infective fevers, and many other conditions are said to have had some causal relationship with the disease, and a family predisposition has been recorded. The one constant pathological feature in cases of acromegaly is enlargement of the pituitary gland.

Pathological Anatomy.—The pituitary is greatly hypertrophied. It is often as large as a cherry, and in some cases may attain a much greater size. The anterior part of the gland appears to be specially involved, and in most cases there

is definite erosion of bone in the neighbourhood of the sella turcica.

In a typical case all the bones are more or less enlarged, but especially those of the extremities. It is chiefly a periosteal development of new bone more marked near the lower ends of the bones. The outer part is denser, but the inner portion of the bone is either normal or somewhat rarified. There are often osteophytic disturbances in connection with the occipital, mastoid, and other eminences on the skull. The orbital arch is large. The zygoma, malar bone, and nasal processes increase in size, and the lower jaw is enlarged in all directions. There is marked kyphosis in the cervical and dorsal regions, and with this is associated the tendency of the patient's head to rest on the sternum. The enlargement of the hands is not entirely due to bony hypertrophy, because the subcutaneous tissues are thickened by a new growth of connective tissue cells, although the hair and the cutaneous nerves escape injury. The heart, kidneys, liver, and spleen are often enlarged.

The pathology of the condition appears to be a hypersecretion of the pituitary gland.

Clinical Features.—The disease begins insidiously with increasing debility, headache, irritable temper, and not infrequently changes in sensation, such as tingling and numbness, of which the patient complains. Sweating is frequently noticed, and occasionally pigmentation of the skin. The typical feature is the gradual enlargement of the extremities, of the lower jaw, and often of the nose and upper lip. The brow is prominent, but the forehead recedes. The tongue may also be enlarged. In the foot the big toe may be proportionally much larger than the other toes, but, as a rule, the affected parts enlarge symmetrically, and can be freely used.

As the disease progresses, the patient notes the necessity for increased size of hats, gloves, and shoes, and the lower jaw may become so enormous as to be a prominent feature, while the teeth tend to separate from each other, because they do not increase in size with the jaw in which they are placed. The head leans forward, and the spinal curvature already referred to develops. The shoulders, the elbows, and the knees are sometimes also enlarged, but the fore-arm and the leg below the knee look thin and poor when compared with the size of the hands and feet. Epistaxis is sometimes complained of.

The heart is often hypertrophied, and in time such evidences of cardiac failure as breathlessness indicate the speedy termination of life.

One leading clinical feature is due to the pressure of the enlarged pituitary on the optic chiasma. It causes blindness in the two nasal halves of the retina, and therefore of the two temporal halves of the field of vision.

Mental changes often develop. They consist in loss of memory and slowness of cerebration, increasing irritability, and sometimes delusions. The speech becomes slow, and somnolence is often present.

The **Diagnosis** in most cases is easy. The enlargement of extremities and of bones is symmetrical. In *pulmonary osteo-arthropathy* there is invariably associated bronchiectasis or other respiratory disease, and the clubbing of fingers and toes, so commonly present, differentiates it sharply from acromegaly. By means of the X-rays the enlarged sella turcica, with the presence of the pituitary tumour, can often be made out, and in addition the enlargement of bones can be well demonstrated.

The **Prognosis**, while not immediately unfavourable, is bad because life is rarely a long one.

The **Treatment** for the condition is most unsatisfactory, and as we should expect, if the suggested pathology is correct, all treatment by administering pituitary extract is futile. Operative interference has been recently tried, and an attempt made to remove the pituitary tumour. The main duty of the surgeon is, however, directed to the relief of pressure by trephining so as to avert blindness. Cardiac tonics must be given where necessary, and potassium iodide has been found beneficial in some cases.

DISEASES OF THE CIRCULATORY SYSTEM

I. DISEASES OF THE PERICARDIUM

Pericarditis

An inflammatory infection of the lining membrane of the pericardium.

Etiology.—It is rather commoner in men, and in early adult life; it is caused generally by acute rheumatism (30-70 per cent of all cases), occasionally by Bright's disease, and may also be the result of tuberculosis, pneumonia, pyaemia, gonorrhoea, scarlet fever, small-pox, and other infective conditions. Sometimes it is due to aortic aneurism, trauma, cancer, hydatid cyst, abscess, etc. It may occur in many different diseases, and in mild degree, but the reason for its development is either a pathogenic organism or the presence of one or more toxins.

Pathological Anatomy.—There is congestion of serous surfaces of sac, and deposition of inflammatory lymph, and this is followed, in many cases, by an effusion of serous lymph. Where the infective agents are pus-forming organisms, the effusion is purulent.

There are three stages:—

1. The Plastic Stage, to which the condition may be limited. It is the stage of exudation of fibrinous lymph. In a severe case the heart is covered with a thick, shaggy-looking layer of fibrinous lymph, and the myocardium is often affected, definite myocarditis being present. The pericarditis in mild cases is localised in area, frequently near the root of the great vessels, sometimes over the right auricle or ventricle, and some pathologists consider that "milk spots" are the result of such limited pericarditis.

Clinical Features.—While there may be no symptoms at all, or, at any rate, none which point to an involvement of the pericardium, there is usually some pyrexia, with possibly an initial rigor, generally some præcordial pain with feeble, rapid, and often irregular pulse, and a varying amount of dyspnœa.

Physical Signs.—During this plastic stage *Inspection* may reveal nothing, *Palpation* possibly demonstrates distinct friction fremitus near the base of the heart, *Percussion* is often negative, while on *Auscultation* one hears a double rub. The friction murmurs are generally first heard at the base of the heart, change from time to time, are not synchronous with the heart-sounds, and often become louder on firm pressure with the stethoscope. The respiratory movements generally affect the loudness of the friction murmur, and one characteristic of importance is the limited area of propagation.

The differential **Diagnosis** between friction and a *double aortic murmur* is easy as a rule. The fact that the double rub does not correspond in time with the heart sounds, that it becomes louder on pressure, and lacks the lines of conduction of the double aortic murmurs, is conclusive. *Pleuritic friction*, when near the pericardium, may be in part caused by the cardiac movements, and occasionally the pericardial sac itself becomes secondarily affected. This is not uncommon in tubercular disease of the lungs and in catarrhal pneumonia, and careful and repeated auscultation may be required before it can be decided whether or not the pericardium is involved.

2. The Stage of Effusion.—In a certain number of cases the pericarditis passes from the first stage into a stage in which a serous and sometimes a purulent effusion occurs. The effusion may amount from 8 to 80 ounces. (See also Purulent and Haemorrhagic Pericarditis.)

Clinical Features.—Pain, if present, may be greater, and there is generally a sense of oppression or distress, which is increased on pressure being exerted over the lower part of the sternum. Dyspnœa is dependent on the amount of effusion. The pressure exerted by the pericardial sac may cause various phenomena:—the pulsus paradoxus from compression—during full inspiration—of the aorta at its commencement, aphonia from pressure on the left recurrent laryngeal nerve, and irritating cough due to the same cause, or from pressure on

the trachea or left bronchus. Lastly, the left lung may be compressed, and may suggest a pleural instead of a pericardial effusion. In a severe case, great restlessness, sleeplessness, and even delirium are generally present.

The *Physical Signs* are very definite. On *Inspection* there is praecordial bulging, and the interspaces are widened. The expansion of the left lung is obviously deficient, and there may be bulging and very generally diminished movement in the left half of the epigastric region on inspiration. On *Palpation* the apex beat is generally absent, and, if still palpable, is displaced upwards because the heart is pushed in that direction and away from the diaphragm by the increasing fluid. There may be friction fremitus at the base of the heart, but more probably, although once present, it will now have disappeared. On *Percussion* the cone-shaped area of dulness is very definite and distinctive; the apex may reach as high as the left clavicle, while the base is at the diaphragm. The apparent superficial cardiac dulness is greatly increased. Rotch has pointed out that the 5th right interspace near the sternum is dull, that in fact the angular area, which is usually resonant, bounded by the right border of the heart and by the liver, has become dull. On *Auscultation* the heart-sounds are always weak. There may be slight friction at the base. The only sound proportionately accentuated is the 2nd in the pulmonary area.

The differential **Diagnosis** is usually fairly easy in cases where the effusion is considerable.

It may be mistaken for (1) a *dilated heart*:—

Pericardial Effusion.	*Cardiac Dilatation.*
1. Impulse absent.	Impulse wavy.
2. No palpable shock.	Weak but definite shock.
3. Cone-shaped percussion dulness, apex above.	Percussion dulness of enlarged heart.
4. Tympanitic note in axilla.	No such note.
5. Sounds muffled.	Sounds sharp and clear, although gallop-rhythm may be present.

Unfortunately it not infrequently happens in rheumatic cases that dilatation of the heart is present, and if complicated with pericarditis with effusion, the diagnosis becomes more difficult. Paracentesis generally clears up the diagnosis, but it

is not an operation lightly undertaken for diagnostic purposes alone.

Or (2) a *left pleural effusion* which, if encapsulated, may resemble the pericardial dulness. In a pleural effusion, however the breath sounds are absent, and the heart will probably be merely displaced with distinct heart-sounds.

The **Prognosis** is usually favourable in fibrinous and sero-fibrinous effusions, but the greater the effusion the more guarded should be the opinion given. A considerable effusion may absorb quickly, or may run a very chronic course, and adhesions are inevitable. There may be endocarditis and myocarditis present, and pronounced cardiac dilatation adds to the gravity of the case. Purulent effusions are serious.

Treatment of Plastic Pericarditis and Pericardial Effusions.—For a plastic pericarditis enforce absolute rest in bed, and give in rheumatic cases one of the salicyl group combined with an alkali. Morph' (gr. $\frac{1}{4}$-$\frac{1}{6}$) hypodermically may be required for alleviating Much relief is obtained by local treatment, which may be varied according to the case. Either (1) apply an ice-bag, or (2) hot fomentations or poultices, or (3) blisters, such as cantharidis ($\frac{1}{2}$-2 in. square), or (4) 6 to 8 leeches over the praecordium. In most cases poulticing or blisters are to be preferred to the ice-bag, but for a vigorously acting heart the ice-bag yields very satisfactory results.

Where an effusion is present, tapping is sometimes necessary. Paracentesis is best performed either—(*a*) in the 5th interspace between the left mid-clavicular line and the sternum, or (*b*) in the left half of the epigastric region between the xiphi-sternum and costal margin, the needle being pushed upwards through the diaphragm. Never remove much fluid—10 to 20 oz. should suffice—and guard against shock by administering stimulants afterwards. Encourage absorption by appealing to the kidneys and bowels and by local counter-irritation.

In purulent cases tapping may be tried, but a freer incision should be considered. The prognosis in such cases is very bad.

Plastic pericarditis may completely disappear, but often adhesions remain, and in cases of serous effusion, should the patient survive, adhesions follow the absorption of the fluid.

In either case we may get the next or third stage of pericarditis.

3. ADHERENT PERICARDIUM. The may result either from plastic pericarditis or pericarditis with effusion. The size of the heart depends on whether the weakened wall permits of dilatation which in turn has to be compensated by hypertrophy, or whether from interference with the work of the ventricles enlargement occurs. In hearts where there are external adhesions between pericardial sac and other structures, hypertrophy is common. There is sometimes an associated chronic mediastinitis, less frequently chronic pleurisy and peritonitis, and more rarely a form of hepatic cirrhosis. Interstitial changes have also been noted in the lungs.

The **Clinical Features** may show evidences of dyspnœa and pain, but these are not very definite, and may be attributed to a coexistent valvular lesion.

The *Physical Signs* are often most distinctive. On *Inspection* bulging of the præcordial region may be seen, with a varying amount of systolic retraction of the left half of the diaphragm, and, according to Skoda and other authorities, also of the chest wall at the apex of the heart. The possible development of apical systolic retraction is open to serious question. On *Palpation* a diastolic shock felt at the base of the heart is characteristic; there is undulatory pulsation in cases of marked hypertrophy, and the apex of the heart is not altered in position by rolling the patient over on to the left side. *Auscultation* merely demonstrates the feebleness of the heart sounds. The pulse in cases of mediastinitis is often a typical pulsus paradoxus, especially with full respirations, and its importance is increased if there is inspiratory engorgement of the jugular veins in the neck.

Mediastinitis may eventually cause widespread phenomena, both pathological and clinical: for example, peritonitis may ensue, with ascites.

Treatment.—Lately by resection of ribs an attempt has been made to relieve the heart and permit of free use of the diaphragm on the left side where its action has been restricted. The treatment otherwise is to keep the heart acting properly, to give purgatives and diuretics when necessary, and to treat the ascites in those cases in which it develops.

Tuberculous Pericarditis

The pericardial sac may be infected by the tubercle bacillus in cases of general miliary tuberculosis. It is occasionally primary, but is generally secondary to a tuberculous lesion elsewhere.

The disease may manifest its presence in its primary site, wherever that may be, but the secondary involvement of the pericardial sac is nearly always insidious and not accompanied by many symptoms.

There is friction and in certain cases evidences of effusion, and the effusion is sometimes blood-stained. It is rarely purulent unless a pyogenic organism is superadded.

Purulent Pericarditis

This is common in pyaemic cases, and may be associated with tuberculosis or pneumonia. Pus-producing organisms may infect the pericardial sac in connection with other diseases, but the most frequent are those just mentioned. In connection with cancer, or as the result of an incised wound or infection from a neighbouring abscess, a purulent pericarditis may originate.

The clinical features are similar to sero-fibrinous effusions, only more acute, and the prognosis is less favourable.

Hydro-Pericardium

Dropsical effusion into the pericardial sac.

Etiology.—Hydro-pericardium may develop in any condition which causes general dropsy; most commonly it is of cardiac or renal origin. In cases of cancer and tuberculosis of the pericardium the effusion may be a dropsical one.

The **Clinical Features** in marked cases resemble pericarditis with effusion, but there is no fever and generally no pain. Dyspnoea and interference with the action of the heart are usually present.

The *Physical Signs* show increased praecordial dulness and feeble heart-sounds, and closely correspond with those of pericarditis with effusion.

The **Diagnosis** from an *inflammatory effusion* depends on the other clinical features present, and on the nature of the fluid in the sac.

Prognosis.—Hydro-pericardium aids a fatal termination and may occur just before death.

The **Treatment** must be directed to the cause of the dropsy, and attempting to remove the fluid present by appealing to the bowels and kidneys. Paracentesis may have to be resorted to.

Haemo-Pericardium

Blood in the pericardial sac.

Etiology.—It may result from an incised wound, from rupture of an aneurism into the pericardium, from cardiac rupture, tuberculosis, cancer, scurvy, Bright's disease, and purpura haemorrhagica.

The **Clinical Features** of a *severe* case are pallor, giddiness, dyspnoea, sense of great and increasing cardiac oppression, syncope, and often death. The needle is the only diagnostic agent, and cure is improbable.

Endeavour in **Treatment** to give absolute rest to the sufferer; but this is generally a hopeless task.

Pneumo-Pericardium

Air or gas in the pericardial sac, and it is always associated with effusion.

Etiology.—It may be due to perforation of a gastric ulcer into the pericardial sac, or to injury from a wound such as may occur in sword-swallowing. Rarely the presence of the Bacillus aerogenes capsulatus (Welch) accounts for the condition.

The **Clinical Features** include great cyanosis, distress, and irregular cardiac action, associated with churning movements heard on auscultation, and which are due to the movements of the heart. Sleeplessness is common, and death, with delirium, almost always occurs. A tympanitic note is obtained on percussion over the pericardial sac, which in time is modified by the accumulation of fluid rendering the lower part of the sac dull.

II. DISTURBANCES OF RATE AND RHYTHM

(1) Palpitation

Excited action of the heart and the recognition by the patient of the forcible cardiac pulsations.

Etiology.—It may be of nervous origin, as in hysterical patients, or may follow great excitement, when patients are specially highly strung; it is frequently met with at puberty, at the climacteric, and also in delicate girls at the menstrual epoch. In neurasthenia palpitation is common, and it is one of the typical phenomena in exophthalmic goitre.

Amongst other causes are gout, dyspepsia, pressure of a dilated stomach on the heart, and the action of certain toxic agents in excess, such as tea, tobacco, and alcohol. Soldiers on the march often fall out owing to excessive palpitation, the result of overstrain and consequent cardiac irritability. Lastly, palpitation occurs in some forms of cardiac disease, and also in fatty degeneration of the heart.

Clinical Features.—Sometimes the palpitation is so severe as to shake the patient's bed, and the pulse may become unduly rapid (tachycardia), although not necessarily. Often a temporary polyuria follows each attack. In gouty and dyspeptic patients there may be the tumbling heart, a phenomenon much more alarming to the patient than its significance warrants.

The **Prognosis** depends largely on the etiological factor present.

Treatment.—Avoid any known cause of the condition, and treat the dyspepsia or gout already referred to.

An ice-bag is admirable, and may be applied over the heart for hours, days, or even weeks. Brandy, ammonia, peppermint water, and similar remedies are often useful. Tincture of digitalis in 5 to 10 minim doses is beneficial in some cases. Occasionally valerian relieves, especially in hysteria. Sedatives, such as the bromides, are certainly worth a trial in severe cases. Divert the patient's mind from the condition: to attain this object, stretching the arms above the head while in bed and many similar devices are often helpful.

(2) Tachycardia

Rapid heart's action, and is due sometimes to shortening of the cardiac diastole, perhaps more often to the development of numerous extra-systoles. The pulse-rate may be 150, 200, or even 300 to the minute. It is one of the typical phenomena in exophthalmic goitre. There are cases of tachycardia in which a lesion of the vagi nerves, or a lesion in the neighbourhood of the great cardiac centres in the medulla, explains the hurried action of the heart. Fatigue is the etiological factor in some cases, abdominal affections and especially flatulence in others, and cardiac dilatation is present in not a few instances, although it may be impossible to say whether it is not an effect of the tachycardia. Dyspnœa and cardiac pain may be present, but the condition, when not too pronounced, is consistent with a long and useful life.

Many cases of tachycardia are paroxysmal, the paroxysm lasting for varying periods, and in many of these the explanation is the development of extra-systoles. Some of these cases are hysterical in origin, but any of the numerous causes of tachycardia may account for the paroxysmal type.

There is marked lowering of blood-pressure during the attack, and if very prolonged, cyanosis and venous stasis may appear.

The **Treatment** applicable to palpitation should be tried. In some cases bandaging the abdomen arrests the attack, and galvanism may be applied to the vagi nerves.

(3) Bradycardia

Slowness of heart's action. Bradycardia may be due to (1) irritation (direct or indirect) of the vagus or its centre, or to (2) myocardial changes whether degenerative or whether temporary or permanent loss of muscular tone. Not a few cases belonging to this second group are the direct result of disease of the coronary arteries which interferes with the blood-supply to the heart wall.

There are cases of slow heart's action which are less serious. Many famous men have very slow pulses, and the great Napoleon had a pulse-rate of only 40 to the minute. Bradycardia is, however, met with in cases of great exhaustion, and

therefore is common after a severe attack of a continued fever, and it is often present during the puerperium. In uraemia and some other toxaemic conditions, a slow pulse-rate is the rule. The pulse may be slowed down in certain nervous diseases, such as apoplexy, epilepsy, melancholia, and general paralysis, and in some other diseases such as anaemia, chlorosis, diabetes, gastric ulcer, and jaundice. Digitalis and many cardiac tonics act partly by slowing down the heart.

There are cases of bradycardia which are due to blocking of the auricular stimulus to contraction at the auriculo-ventricular fibres, so that only every second auricular contraction may succeed in stimulating a ventricular systole.[1]

Diagnosis.—Atropine paralyses the vagus terminations in the heart, and it may be possible by administering this drug to exclude cases due to vagus irritation by noting the increase of pulse-rate.

Prognosis.—A slow pulse, apart from the diseases above mentioned, generally suggests a phlegmatic constitution, and it is compatible with long life.

Treatment.—Attend to the general laws of health, and when necessary, give tonics, and especially strychnine and diffusible stimulants. Try nitroglycerine if there is a high blood-pressure, and atropine is sometimes of value.

(4) Stokes-Adams' Syndrome

A slow pulse with syncopal attacks, during which the heart-rate falls still lower. This is generally met with in old persons, and is associated with arterio-sclerosis and typical attacks of heart block with bradycardia and arhythmia. The

[1] The contraction of the heart commences at the venous orifices in the auricles, and the impulse passes from auricle to ventricle by means of a small bundle of fibres originating in the septum between the two auricles, passing downwards and forwards through what is called the trigonum fibrosum, and terminating close to the root of the aorta. Should the impulse be stopped by this band of fibres, ventricular systole does not occur, and the auricular impulse is blocked. In this way the auricular contraction may not be transmitted to the ventricle, although it should be stated that ventricular systole may occur apart from any stimulus from the auricle. Very frequently in bradycardia only every second auricular systole stimulates the contraction of the ventricles, and it is probable that feeble ventricular systoles may sometimes be explained by supposing an insufficient stimulus to contraction being transmitted through these auriculo-ventricular fibres.

pulse may only total 20 per minute, and the attacks of vertigo recur several times a day. In treatment remember to keep the head low and to try the effect of potassium iodide in all cases in which syphilis is even suspected.

(5) The Pulsus Paradoxus

In which the pulse-waves are more frequent, but less full, during the inspiratory act. This is due to pressure of the lungs during inspiration either on the aorta, in which case *both* radials will show the phenomenon, or on one subclavian, when only *one* pulse is implicated. Pericardial effusions and adhesions may cause the paradoxical pulse to appear at both wrists, while the subclavian, generally the right, may be involved by pleuritic adhesions in tubercular affections of the lung. It must not be forgotten that a weak and especially a dilated heart may show a slight degree of the pulsus paradoxus without any of the local affections just referred to.

(6) Arhythmia

Irregular action of the heart, of which there are many varieties. Of these the following special forms deserve mention:—

(*a*) The **Pulsus Bigeminus** and **Trigeminus** are often met with in mitral disease, or may follow an excessive dose of digitalis. They consist in ventricular systoles linked together, either in pairs or in threes.

(*b*) The **Intermittent Pulse** consists in an occasional, very weak contraction of the left ventricle, which records no wave at the wrists. This may be habitual and may be constant, or merely occasionally developed. It may indicate the debility following on any severe illness, and in such a case exercise, temporary excitement, or a meal may make the pulse regular for a time. There are, however, other causes, such as gout, fatty degeneration of the heart, excessive use of tea or tobacco, mental shock, and sometimes heart disease, which are responsible. The intermission occurs, generally with fair regularity, about every 4, 10, or 20 beats of the pulse. It may persist through life, or may, in a patient who is predisposed thereto, follow ingestion of an imprudent or too heavy meal.

The **Prognosis** depends much on the various elements in the case and their significance.

The **Treatment** consists in rest and tonics, and often the careful regulation of the dietary.

Anaesthetics are not contra-indicated by an intermittent pulse, but they should be administered with special care.

Comparatively recently the part played by extra-systoles in the production of intermittence has become recognised. Immediately after a contraction of the ventricle, there follows a phase during which the muscle is described as refractory or inexcitable. Sometimes this phase does not immediately follow contraction, and an extra-systole or second ventricular contraction occurs apart from the physiological stimulus of a preceding auricular systole. A long refractory or inexcitable phase follows, and the physiological stimulus of the next auricular systole passes unnoticed, and the inexcitable phase may not have passed off till the second auricular systole has occurred. Extra-systoles occur under the conditions mentioned above, and the prognosis is not necessarily very grave.

(*c*) **Foetal Heart-Sounds.**—The two normal foetal heart sounds are equal in intensity, and the pauses are of equal length. The adult heart sounds may acquire these foetal characteristics during a very severe illness, such as a bad attack of typhoid fever, and in the late stages of a fatal illness of any kind when death is impending. They are also present in grave cases of cardiac dilatation. The import is always serious.

(*d*) The **Gallop-Rhythm** is the condition sometimes met with in cases of arterio-sclerosis and chronic Bright's disease, and so forth, in which the heart-sounds resemble the rhythm of a cantering horse, and are divided into three parts. Either the 1st or, more generally, the 2nd sound is reduplicated.

(*e*) **Delirium Cordis** implies extremely rapid, often tumultuous, and totally irregular cardiac action. It is perhaps most commonly produced by excessive doses of a poison like digitalis.

III. PAIN IN THE REGION OF THE HEART

Such pain may be the result of muscular rheumatism, intercostal neuralgia, costal periostitis, or pleurisy, while flatulent distension of the stomach may displace the heart, causing very

definite pain. Hysteria is responsible for severe pain in this region, and one of the typical hyperaesthetic areas in that disease is situated just below the left breast. Neuralgia of the left phrenic nerve, or of the cardiac plexus, and a neuritis of the nerves in the wall of the aorta due to inflammatory changes there, closely simulate, if they are not the actual causes of, true angina pectoris.

Angina Pectoris: Cardiac Neuralgia

Paroxysms of pain in the region of the heart, dependent upon organic disease of aorta, coronary arteries, or heart.

Etiology.— It generally occurs in males and in persons who have reached adult life, and is associated with— 1. Disease of the coronary arteries, by which the myocardium is insufficiently supplied with blood. 2. Disease of the aorta, such as aortitis, arterio-sclerosis, and atheroma. 3. Heart conditions, such as fatty heart (not infrequently a direct result of coronary disease), hypertrophy of the heart with arterio-sclerosis, or a valvular lesion, especially aortic incompetence.

It is common in gout, and the *exciting* causes of an attack are nervous shock, peripheral irritation, and over-exertion (such as straining at stool), anger, and so forth.

Pathological Anatomy. — In fatal cases the heart is relaxed and full of blood. There is present one or more of the pathological conditions indicated above. Neuritis of the nerves in the wall of the aorta has been described, but obviously the coronary arteries, and their temporary (due to spasm) or permanent inadequacy to supply sufficient blood to the myocardium are most commonly at fault.

Clinical Features.— Sudden paroxysms of pain radiate from the praecordia to the left shoulder and arm, or the left side of neck, with feeling of suffocation and impending death: less frequently the pain radiates to the right side. The attack lasts from seconds to many minutes, during which the patient is pale, with ashy-grey face, and beads of cold perspiration are seen on the forehead. The chest is held as in a vice; the sufferer cannot move from the position in which the attack seizes him, and he generally experiences nausea afterwards. A very high blood-pressure may be associated with the attack. Occasionally eructations of gas and a flow of limpid urine follow

an attack. Dyspnœa of an asthmatic type, with wheezing, is sometimes present. These attacks may recur frequently, or the first may prove fatal.

It is difficult to explain the nature of true angina.

1. It has been thought to be a neuralgia either of the cardiac plexus, the 4th to the 9th intercostal nerves on the left side, or the left phrenic nerve. The pain is not akin to neuralgia elsewhere, but neuralgia involving the cardiac plexus would necessarily be unique in its peculiar severity.

2. Cramp or spasm of the left ventricle (Heberden). Such spasm would, however, be incompatible with life, and many patients have recurring attacks of angina.

3. Over-distension of the ventricles with blood (Traube).

4. Coronary disease, either producing anæmia of part of the heart wall, and so leading to local spasm, or vasomotor spasm of the coronaries, due to the diseased arteries, may temporarily deprive a part of the heart wall of blood. A defective blood-supply to the heart wall may be just sufficient until exercise or strain increases the demand for blood, when a sudden ischæmia of the affected area of heart wall occurs, and to this, whether it results in muscular spasm or not, the anginous pain may be due.

The 1st and especially the 4th theories are those most favoured.

Diagnosis. — Huchard distinguishes *pseudo-angina* (hysteria) from true angina.

True Angina.	*Pseudo-angina.*
1. Adult life.	Any age.
2. Males generally.	Females generally.
3. Attacks are not periodic.	Are periodic.
4. Awful pain.	Less severe.
5. Duration, seconds to minutes.	Hours.
6. Often fatal.	Never fatal.

Compare also other causes of præcordial pain.

The **Prognosis** should be most cautiously given. Very rarely is permanent recovery possible, but a first attack is not usually fatal.

Treatment.—For the attack, order nitrite of amyl in 3 to 5 minim capsules, which should be broken in the handkerchief and inhaled, and morphia hypodermically), or even a whiff of

chloroform might be tried, in the event of the nitrite of amyl failing to afford relief. Between the attacks, and especially if the blood-pressure is high, give nitroglycerine (1-2 minims of a 1 per cent solution), or nitrite of soda in 2 to 4 grain doses; in most cases order a course of potassium iodide. Arsenic and iron are often of value.

Remember that a quiet, well-regulated life, freedom from excitement, and the prohibition of physical and mental overstrain are essential to successful treatment.

IV. ACUTE ENDOCARDITIS

Inflammation of the endocardium, especially that part of it covering the valves. It may be acute or chronic, and the acute form is often described as being either (1) simple, or (2) malignant or ulcerative. Many of the chronic cases are closely associated with arterio-sclerosis.

Etiology.—Endocarditis is the result directly or indirectly of certain organisms. In acute rheumatism, and less acute but also rheumatic cases, in which the joints specially suffer, endocarditis commonly occurs. The *simple* form is more frequently the result of rheumatism than of anything else. In at least one-third of all cases of acute rheumatism, endocarditis develops; with certain fevers, and especially scarlet fever, it may occur, while tonsillitis, which is often associated with acute rheumatism, is not infrequently the precursor of inflammation of the endocardium. In chorea, endocarditis may develop, although the connection between the two diseases has given rise to considerable differences of opinion. In pneumonia, and occasionally in phthisis, endocarditis may be due to the organisms associated with these diseases.

It is usual to find that, where there is disease of the valves, whether originally inflammatory or degenerative, repeated attacks of simple endocarditis occur.

In *malignant* endocarditis similar etiological factors may be at work, but, in addition, the organisms found on the affected valves may be those of puerperal septicaemia, pyaemia, and erysipelas, in which group of cases the endocarditis has been termed secondary in place of primary, although such a classification is objectionable. We have no absolute border-line

between simple and malignant endocarditis; the simple form may assume, gradually or suddenly, malignant characteristics.

Pathological Anatomy.—1. *Simple Endocarditis.*—The inflammation commences with an invasion of the endothelial and subendothelial cells by the micro-organisms. They produce swelling and coagulation necrosis, and lead to a greyish appearance of the part affected. The endothelium is shed, and a small granulation forms, at first of blood platelets, and later of leucocytes and fibrin. In time, wart-like vegetations result often of small size, and usually limited to the lines of contact of sigmoid, and the auricular surfaces of auriculo-ventricular cusps. The aortic and mitral valves are far more commonly affected than are the pulmonary and tricuspid. The vegetations are often specially large in pneumococcal and gonococcal cases. On the sigmoid valves the proliferation of intimal cells and the exudation of leucocytes occur more slowly than on the auriculo-ventricular, owing to the fact that there are fewer vessels in the former than in the latter.

This inflammatory exudation, forming a primitive vegetation, may undergo the following changes:—

(1) It may be absorbed, leaving a perfectly healthy surface behind. (?)

(2) It may organise, and become almost entirely composed of connective tissue.

(3) It may grow, and if so, it may take on ulcerative characteristics, the necrotic being in excess of the reparative processes. Should this occur, portions of the vegetation may become detached, forming emboli in the blood-stream.

(4) The vegetation may disappear, but leave behind it cicatricial changes, causing a degree of sclerosis of the cusp, and perhaps interfering with the adequacy of the valve.

To what extent it is possible for a vegetation to disappear entirely, without leaving any evidence of its existence, is a point open to question. If it does occur, it is rare. It is certain that, along with thickening of cusps, contiguous edges may become adherent to each other, and this change is sometimes present in acute and also chronic cases.

2. *Malignant Endocarditis.*—In this form the reddish-grey vegetations tend to grow rapidly, but they also break down, and neighbouring portions of the heart or vessel wall may become infected, setting up limited areas of endocarditis or endarteritis

as the case may be. Not infrequently a valve segment is perforated, or an aneurism may develop in connection with a cusp. Sometimes the ulcerative process causes an erosion through the wall of the vessel, and in one recent case the author saw a communication, by means of such an opening, between the aorta and the pulmonary artery. Myocarditis is a common result of malignant endocarditis, and is generally localised.

Embolism in different organs, and especially in the kidneys, spleen, and brain, is an inevitable sequel to ulcerative endocarditis, while it is more rare in the simple form. In the ulcerative form, where many organisms are present in connection with the vegetations, numberless capillary emboli may be found in the kidneys, spleen, and elsewhere; whereas in other cases the areas of infarction are larger in size and less numerous. The nature of the organisms in the emboli determines whether the infarcts undergo acute necrosis, forming abscesses, or not, and the results differ greatly in different cases. Where the vegetation has originated in the right side of the heart, pulmonary infarction occurs.

The organisms most frequently found in endocarditis are, in addition to the Streptococcus pyogenes, the Staphylococcus pyogenes aureus, the Streptococcus parvus, the Pneumococcus, the Tubercle bacillus, the Gonococcus, the Diphtheria bacillus, the Influenza bacillus, the Micrococcus endocarditidis rugatus and capsulatus, and the Bacillus endocarditidis griseus.

Clinical Features.—1. *Acute Simple Endocarditis.*—In many patients there are no symptoms at all, but in other cases palpitation, pain, or, at least, a sense of cardiac oppression, a rise of temperature which may be somewhat trivial, and increased and irregular cardiac action, suggest that the endocardium has become involved. A bruit or murmur is not commonly heard in early cases, but slight impurity of a sound may convey important information to the experienced ear, and especially if such peculiarity of the sound alters from day to day to an appreciable extent.

Should an orifice become stenosed or incompetent, the clinical features to be described under the respective valvular lesions may be made out.

2. *Malignant or Ulcerative Endocarditis.*—Sometimes the symptoms, referable to infarctions, direct the physician's

attention to the fact that a simple endocarditis has become malignant. The general features are pyrexia, usually of hectic type, and associated with sweating, often distressing dyspnœa, and increasing weakness.

There are two types generally described: A) The *Typhoid* or common type, in which the temperature is irregular; there is early prostration, marked delirium, somnolence, and eventually coma; diarrhœa is also characteristic. The infarctions not infrequently suggest, in this type, the true nature of the case. B) The *Pyæmic* type is often due to puerperal conditions. The rigors, the profuse sweating, and the typical septic phenomena, with the development of jaundice, and abscesses in joints and elsewhere, are characteristic of this variety. Petechial haemorrhages are not uncommon.

Other types, such as *Cerebral* and *Cardiac*, have also been described. Malignant endocarditis varies greatly according to the organism present and the virulence of that organism. In certain cases the endocarditis may become quiescent, leaving serious valvular lesions as an evidence of pre-existing disease. In other cases a rapidly fatal result ensues.

Infarction of the *kidney* causes lumbar pain, and generally blood and albumin in the urine: *splenic* infarction is characterised by pain felt over the splenic region, and sometimes by the evidences of perisplenitis: embolism of the *brain* may cause extensive paralysis, probably a hemiplegia, together with coma and sometimes delirium: *pulmonary* embolism is described in connection with chronic valvular lesions, but it generally causes a localised area of dulness with the expectoration of blood-stained sputum, and considerable pleuritic pain (see page 497).

Diagnosis. As already indicated, the diagnosis of *simple* endocarditis may demand careful and repeated investigation. But in the *malignant* type, the disease assumes much graver proportions, and the evidence of cardiac auscultation generally points to the site of the disease. The rigors and hectic sweatings are peculiarly suggestive, and in not a few cases a definite leucocytosis is present. Occasionally the organism is obtained by culture from the blood.

Prognosis. — A cardiac lesion implies a damaged heart, but *simple* endocarditis may be compatible with a long and useful life, whereas the *malignant form* is always grave, and in certain cases proves rapidly fatal. The danger in this latter type is

invariably great, even in cases in which improvement with quiescence of the disease, occurs.

Treatment.—Absolute rest in bed is imperative, and wherever there is the least suspicion that the heart has become involved, the patient should be kept absolutely at rest, and not even allowed to sit up. In rheumatic cases the salicylates should be administered, although care must be taken to avoid the depressing effect of excessive doses. In ulcerative cases undoubted benefit follows the use of antistreptococcic serum, which may be administered in doses of 2 to 10 c.c. either hypodermically or by the rectum, note being taken of any fall in the temperature after each dose. With our knowledge of the value of vaccines a better method is to obtain a culture from the blood, and prepare a vaccine and administer that.

Locally, an ice-bag should be applied where there is much palpitation, and Caton's treatment, by small fly-blisters applied in the 4th, 5th, and 6th left interspaces, is sometimes advantageous. The treatment of endocarditis otherwise closely resembles the treatment of ordinary valvular lesions.

Chronic Endocarditis

In many cases of heart disease a simple endocarditis, of very chronic type, accounts for the valvular lesion present but it must be remembered that many valvular lesions are primarily sclerotic, and if endocarditic at all, are only secondarily so.

Etiology.—The causes which produce acute simple endocarditis may also produce the chronic variety, but factors, such as alcohol, syphilis, overstrain, and gout, which are mainly responsible for sclerotic or degenerative changes, may also take some part in the process. In many cases of valvular disease it is absolutely impossible to differentiate between processes primarily inflammatory and those which, from the commencement, are degenerative. In every instance where a valve segment has been damaged, there is a tendency for the development of degenerative changes in the affected structure, so that calcareous deposits as well as sclerotic changes are eventually superadded.

In the great majority of cases the left side of the heart is affected, and only in a very small proportion do the valves of the right heart suffer.

Pathological Anatomy.—Thickenings are apt to occur in the neighbourhood of the corpora Arantii, and small vegetations may develop. The changes, largely sclerotic in nature, cause considerable contraction of the valve segments and thickening of their free edges. Similarly, in the auriculo-ventricular cusps, thickenings may occur which involve not merely the edge of the valve curtain, but also the chordae tendineae and the apices of the papillary muscles. Such changes may be associated with marked inadequacy, or with comparatively slight interference with the heart's action, and a full description is given, where necessary, under the respective cardiac lesions at each orifice, so that further detail here is uncalled for.

V. VALVULAR LESIONS

Results of Valvular Disease

While it is requisite to describe, under the heading of each separate valvular lesion, the results of a valvular lesion, both as regards the heart and the organism as a whole, it is prudent to consider briefly some of those which are common to all heart lesions, and which render more intelligible the detailed consideration of each type of valvular disease.

In health, a chamber, such as the left ventricle, possesses energy sufficient for its complete and adequate contraction under ordinary circumstances: in addition, it has a residual energy capable of bringing about complete and satisfactory contraction, even under great strain. In valvular disease, there is not merely a great diminution of this residual energy, but the energy necessary for the ventricular systole during repose may even be inadequate. This is due to several causes:—

1) There is nearly always enlargement of certain chambers of the heart, as the result of a valvular lesion, and an enlarged chamber demands a greater amount of energy, so as to enable it to contract fully and perfectly.

2. In most cases of valvular disease, the chamber specially affected has a double duty to perform, because the disease of a valve generally either throws on the chamber the duty of

overcoming some obstruction to the onward flow of the blood in its own proper direction, or permits of abnormal regurgitation of blood through inadequate valves.

(3) In many cases, the wall of the enlarged chamber or chambers is badly supplied with blood, or is undergoing some degenerative or inflammatory change, so that, large and muscular as it may seem, its energy may be disproportionate to the work it has to do.

Valvular disease results in — (1) *Increased Backward Pressure*, and (2) *Diminished Forward Pressure*, and under these headings may be considered the immediate and remote results of any valvular lesion, while (3) *Embolism* and *Thrombosis* sometimes occur.

(1) *Increased Backward Pressure.* — If we take as an example mitral stenosis, the left auricle first becomes dilated and then hypertrophied. The lungs become engorged, and the resultant chronic venous congestion causes dyspnœa, cough with watery sputum (œdema), and not infrequently hydrothorax. The right heart next suffers; it dilates, and, in order to discharge its functions, becomes hypertrophied, and sooner or later the right auricle dilates and tricuspid incompetence results. The venæ cavæ are next engorged, and the liver is enlarged and painful, while the patient frequently develops slight jaundice. Following upon the engorgement of the hepatic vein in the liver the portal circulation is affected, and the organs which send their blood into that vein suffer from chronic venous congestion. In the stomach we find chronic gastric catarrh with dyspepsia, and often nausea and vomiting, while hæmatemesis sometimes results from rupture of a distended venous radicle. The pancreas is also affected, and probably chronic venous congestion of that organ shows itself by inducing an intestinal dyspepsia, which is also to some extent due to chronic venous congestion of the intestine. In many cases constipation is present, should there be deficiency of the succus enteriens. The spleen becomes enlarged and is occasionally painful; the peritoneum is also drained normally into the portal system, and therefore ascites develops. It should be remembered that the œsophageal veins anastomose with gastric veins at the cardiac orifice of the stomach, and that the portal circulation has a connection with the hæmorrhoidal plexus, the latter anastomosis resulting in most cases in

the development of haemorrhoids, which may cause the patient great discomfort.

Following down the inferior vena cava, we find that the kidneys send their venous blood into that vessel, and renal

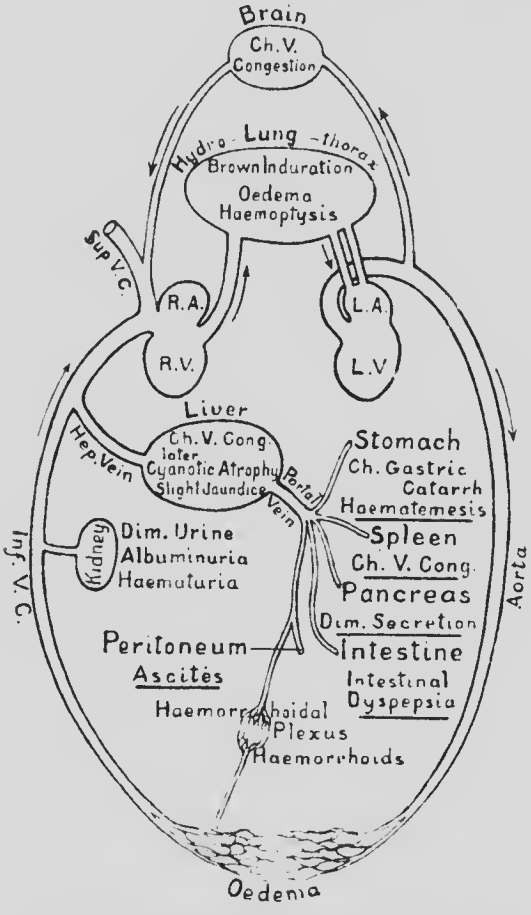

FIG. 26.—Diagrammatic Representation of Backward Pressure.

backward pressure causes a tendency to great diminution in the flow of urine with the presence of albumin and sometimes blood. Following the venous system still farther, we have only to note general dropsy, especially of the extremities, and cyanosis, which is obvious in the nose, ears, lips, and elsewhere. Backward pressure in the venous radicles of the brain tends to cause sleeplessness and sometimes nocturnal delirium.

A diagrammatic representation of the points just mentioned is shown on the opposite page, and the reader will note that, for purposes of simplicity, the right and left hearts have been dissociated.

(2) *Diminished Forward Pressure.*—This is more apt to occur in aortic incompetence, although wherever the left ventricle is feeble, from whatever cause, the same phenomena may be noted. These include anaemia of all organs, and specially of the brain, in connection with which it induces giddiness, headache, and sleeplessness, and perhaps also dyspnoea; in the *Circulatory* system, and in connection with the heart itself, it possibly explains the anginous pain so commonly noted; in the *Alimentary* system, it causes dyspepsia and feeble digestion; in all the other organs of the body, anaemia may seriously interfere with the discharge of normal functions.

(3) *Embolism* and *Thrombosis* are referred to elsewhere. It is only necessary to remind the reader that vegetations, when of ulcerative nature, give rise to emboli, and that, where the right auricle is enormously enlarged, clotting of blood is apt to occur in its appendix, so producing pulmonary embolism. Thrombosis is less commonly the result of valvular disease, but it is often directly dependent on feebleness of the circulation.

(1) Aortic Stenosis

An aortic systolic murmur may result from—

1. True aortic stenosis.

2. Dilatation of the aorta beyond the orifice, in other words, relative aortic stenosis.

3. Slight roughening of the cusps of the aortic orifice, due to vegetations or degenerative changes, and sufficient to give rise to an audible vibration in the blood-stream.

It is the *first* of these three conditions which we discuss here.

Etiology. This lesion is more frequently met with in men, and during middle and later life, an age suggestive of its degenerative origin. Chronic endocarditis accounts, however, for a certain number of cases.

Pathological Anatomy. The obstruction may be the result of vegetations or of thickening of the cusps, sometimes

with adhesion of one or more of the cusps at their contiguous edges. There is mostly adhesion of cusps so as to form a funnel, opening towards the aorta, and this may be associated with little, or with a very considerable degree of, thickening. A rare form of stenosis is subvalvular in position, and is of fœtal origin.

Atheroma of the aorta is commonly present in sclerotic cases, and the coronary arteries are generally also affected. The left ventricle is hypertrophied without, at first, much dilatation, and only at a later stage does the mitral valve become incompetent, and the right heart involved owing to backward pressure. Atheroma of the coronary arteries may produce fibroid and fatty changes in the myocardium.

Clinical Features.—At first there are no symptoms, then a varying degree of malnutrition, causing giddiness and faintness due to defective cerebral blood-supply, and later other phenomena, and particularly dyspnœa on exertion become prominent. Anginous pain is present in some cases, and may, indeed, be an ominous development indicating progressive disease.

Physical Signs.—On *Inspection* and *Palpation* the apex beat is displaced downwards and outwards, and may be diffuse and forcible; a thrill is generally present over the aortic area, and is often very marked. The *Percussion* dulness is increased to the left, but in a case of uncomplicated and pure stenosis the left ventricle may not greatly enlarge, and on *Auscultation* the murmur is rough and systolic in time, with its greatest intensity over the aortic cartilage, and it is propagated into the carotids in the neck and along the great arteries. The second sound in the aortic area is often well heard, but this varies with the condition of the cusps. The pulse is usually slow, small, and regular, and of good tension, until compensation is lost.

Diagnosis. *Dilatation of the first part of the aortic arch* generally yields an area of dulness to the right of the sternum, and a louder and more ringing second aortic sound, and the thrill, if present at all, is rarely so obvious as it is in true aortic stenosis.

Pressure on the aorta by *tumour* should be recognised by other evidences of the new growth, while in *patent ductus arteriosus* the murmur and thrill are late systolic in time, and

are loudest to the left of the pulmonary area, and the murmur is not propagated into the carotid.

Pulmonary stenosis is almost as rare a lesion as a patent ductus, and the murmur, although systolic in time is of course not propagated along the carotid arteries.

Prognosis.—While the lesion is compatible with life and work for many years, the degree of involvement of the coronaries and the resulting degenerative change in the myocardium, the development of aortic incompetence and involvement of the mitral orifice are the dominating factors in any given case.

2) Aortic Incompetence

In most cases there is not merely incompetence of the aortic orifice, but, in addition, either aortic obstruction, or, at least, the presence of an aortic systolic murmur. The lesion is by no means so common as mitral stenosis, but it is very much more grave in its consequences.

Etiology and Pathological Anatomy.—Aortic incompetence commonly occurs in two absolutely different ways:—(1) It may be the result of acute endocarditis with vegetations, not infrequently of a malignant type. This form of aortic incompetence is met with in persons generally before middle life, and is a common sequel to rheumatic, gonococcal, pneumococcal and other forms of infective endocarditis, although second in frequency to the involvement of the mitral orifice. (2) Aortic incompetence may be the result of sclerosis, specially affecting the aortic cusps, and such sclerosis is generally due to alcoholism, syphilis, or physical overstrain, or all three combined. It is frequently associated with atheroma, and tends to occur in persons who have passed middle life. These are the two most important forms. (3) Sometimes the aortic orifice is involved in a dilatation of the first part of the aortic arch, rendering the valves incompetent. (4) Aortic incompetence may also result from the rupture of a weakened cusp by strain, such as the lifting of a heavy weight, but it is rare to find such weakening of cusps apart from endocarditis. (5) The congenital union of cusps together, where, for example, there is a bicuspid condition of the aortic orifice, increases the tendency for inflammatory, and also for degenerative, processes to affect the abnormal cusp or cusps, so that mal

formation, although not the direct cause of aortic incompetence, may lead to its production.

Reference has been made, under aortic stenosis, to the frequent fusion at the contiguous edges of aortic cusps, and to the narrowing of the orifice which this fusion causes. It is extremely improbable that such stenosis can often occur without incompetence. Vegetations may also interfere with the adequate closure of the aortic cusps and, even where the valve curtains have not shrunk or become thickened, one or more vegetations may not merely prevent adequate apposition of the cusps, but may even cause a serious degree of incompetence.

In the endocarditic cases of aortic incompetence, where there is much ulceration, the affected cusps may be perforated, and infective vegetations are also found on the endocardium of the heart or on the intima of the aorta near the site of involvement. In the sclerotic type of aortic incompetence the valves are greatly thickened as well as retracted, and the thickening is generally best marked near their lines of attachment. There is usually atheroma of the wall of the aorta, and, in very many cases, the coronary arteries are also found to be seriously affected. The result is, that not merely do the sinuses of Valsalva fail to become distended with blood when the aortic valves are closed, but the coronary arteries, with their diminished lumen, do not convey a sufficient quantity of blood to the heart walls. The left ventricle hypertrophies in cases of marked aortic incompetence, and if the myocardium is dependent on an inadequate blood-supply, degenerative changes are inevitable. The left ventricle is sometimes so large that the heart has been termed the bovine or ox-like heart, and it may even attain the weight of 35 to 40 oz. As a result of the great enlargement of the chamber of the ventricle, mitral incompetence must sooner or later supervene, leading in turn to all the phenomena of backward pressure in the lung and right heart (dilatation, etc.).

Clinical Features. - The chief clinical features are a blowing aortic diastolic murmur propagated down the sternum, and a typical collapsing or water-hammer pulse. The patient's face is pale, and one notes the remarkable pulsation of vessels in the neck—a pulsation which seems sometimes to shake the whole body. A frequent complaint is that of pain, anginous in type, and accentuated with any slight over-

exertion, while giddiness and attacks of faintness are by no means uncommon.

Physical Signs.—On *Inspection* of the praecordial region, we note a forcible and diffuse apex-beat markedly displaced downwards and outwards, and there is a general heaving, which involves the whole praecordium. *Percussion* shows great increase of cardiac dulness to the left, and, where the right heart has begun to suffer, extension also to the right. On *Auscultation* a double aortic murmur is generally heard over the sternum. The systolic aortic bruit may be merely the result of roughening instead of actual stenosis of the orifice. The aortic diastolic is a blowing murmur, sometimes musical, and propagated down towards the xiphi-sternum, and also towards the mitral area. The point of greatest intensity is not infrequently near the base of the xiphi-sternum. Both systolic and diastolic murmurs are propagated upwards into the carotids. It is important to note the length of the diastolic murmur, because it may yield valuable information with regard to the degree of inadequacy of the orifice, a long murmur indicating less incompetence, as a rule, than a short murmur. A mitral systolic murmur is also recognised in cases in which, from dilatation of the mitral orifice, the cusps have become inadequate. Austin Flint stated that, as a result of aortic incompetence, the anterior mitral segment was pushed by the regurgitating stream of blood into closer apposition with its fellow, and that therefore a presystolic murmur might be produced by this narrowing of the mitral orifice. Much doubt has been expressed as to the correctness of this assertion; in many cases of aortic incompetence, in which a presystolic mitral murmur was also present during life, the existence of true mitral stenosis was proved on post-mortem examination.

The pulse has the typical water-hammer or collapsing character described by Corrigan, and this is readily explained by the fact that the pressure of blood in the aorta and arteries, due to the contraction of the left ventricle, rapidly falls when the blood regurgitates through the inadequate aortic orifice. The water-hammer character is well brought out when the arm is raised. Associated with the sudden fall of intra-vascular pressure, the phenomenon of capillary pulsation can be readily seen under the nails, sometimes in the lips, and at any part of the skin where it has been reddened by

friction. On ophthalmoscopic examination the retinal arteries are also seen to pulsate, a phenomenon not noted under normal conditions, and on placing a stethoscope over a large artery, such as the femoral, a double murmur may be recognised, due to the two waves passing the site of constriction and so setting up two murmurs. This is distinct from the conduction of the double murmur from the aortic orifice already noted as being audible in the carotid arteries. A sphygmogram of the pulse in aortic regurgitation shows the rapid rise, the short duration, and the rapid fall, while a predicrotic wave is often present in the down-stroke.

This description of the physical signs of aortic incompetence is typical of cases in younger persons, and which are the result of endocarditis. In older patients, there is usually a less evident water-hammer pulse, and the immediate prognosis is by no means so grave. A guide to the diagnosis of one type of aortic incompetence from the other may be obtained by comparing the time of the apex-beat of the heart with the radial pulse, when it is noted that in younger patients, in whom endocarditis is the cause of the condition, the delay in the radial pulse is very long, far longer than in health, and longer than the delay in patients whose aortic incompetence is due to sclerosis.[1]

Reference has already been made to the frequency of anginous pain, and this pain, as already hinted, is probably due to the degenerative changes in connection with the aortic orifice, and perhaps specially the coronary arteries. There is not infrequently cardiac dyspnoea, and attacks of palpitation are by no means uncommon, often indicating a very unsatisfactory condition of the myocardium of the heart.

In the *Haemopoietic System* one notes that anaemia is often present. In the *Respiratory System*, where backward pressure has begun to be in evidence, there is cough and dyspnoea of pulmonary origin, together with oedema into the air vesicles or into the pleural sacs. In the *Urinary System*, when backward pressure is marked, some albumin is present in the urine. In the *Nervous System* there are various important symptoms of frequent occurrence: these include a

[1] This observation of delay in time of the pulse-wave is ascribed by Mackenzie, but is supported by the authority of the late Dr. George W. Balfour and Sir William Broadbent

tendency to faintness especially on rising suddenly from the prone position, and headache owing to anæmia of the brain, while sleeplessness, probably from a similar cause, frequently demands careful treatment. Many patients complain of extraordinary and harassing dreams, dreams probably largely due to irritability from anæmia of brain centres.

It is very common in ulcerative cases to find evidence of emboli either in the spleen, where they may cause pain, sometimes with perisplenitis; in the kidney, where they give rise not infrequently to hæmaturia; or in the brain, where they may cause apoplectic phenomena; but much depends on the nature of the emboli in question. In cases in which the organisms associated with the endocarditis are of great virulence, the diffusion of emboli may produce pyæmic symptoms, while in other cases the emboli appear to be simple. Reference has already been made to the changes which result in the area of infarction.

Diagnosis.—The distinctive murmur and pulse generally prevent any possible mistake. A *pulmonary regurgitant* murmur is better heard to the left of the sternum, and, if propagated at all, is only propagated over the right ventricle, and not towards the apex of the left ventricle. There are cases, however, in which it may be extremely difficult to eliminate the possibility of pulmonary regurgitation coexisting with aortic.

Prognosis.—There is no valvular lesion, which is so likely to cause the sudden death of the patient, but much depends on the degree of incompetence and on the condition of the myocardium. Perhaps still more depends upon the possibility of the patient leading an easy life, free from physical strain as well as mental worry. A careful investigation should be made of the mitral orifice, the right heart, and the arteries of the body so far as they can be examined, and valuable information may be gained from noting the position of the apex-beat.

3) Aortic Obstruction and Incompetence

As already indicated, in considering the two preceding conditions, they are very frequently combined, and it is extremely rare to find aortic incompetence without a degree of aortic obstruction, although it should be remembered that the presence

of an aortic systolic murmur is by no means necessarily due to stenosis of the aortic orifice.

In some cases the lesion begins with obstruction, in other cases it begins with inadequacy, and in both the chief types of aortic incompetence that have been described, there may be eventually obstruction as well as incompetence. The existence of obstruction undoubtedly increases the work of the heart, already severely strained by the incompetence of the aortic orifice, and therefore an early loss of compensation, due to the great enlargement of the left ventricle, becomes extremely probable.

The **Diagnosis** of the double lesion is not always easy. A marked degree of obstruction interferes considerably with the typical water-hammer pulse, but the existence of a very definite aortic thrill, systolic in time, greatly increases the probability of stenosis being present.

The **Prognosis** of the double lesion is grave, although the fact that obstruction is so often a sequel to incompetence, renders cases of pure aortic incompetence extremely rare.

(4) Mitral Stenosis

Narrowing of the mitral orifice.

Etiology. — In a large proportion of cases it is the result of endocarditis. It is most common in early life, and is a disease affecting women in a larger proportion than men. Where endocarditis is present, it is the mitral valve which generally suffers, and stenosis usually results. Rheumatism and chorea are more frequent in girls, and mitral stenosis is often associated with these diseases. There are, however, many cases of mitral stenosis in which it is difficult to assign a satisfactory etiological factor. It is rarely a congenital lesion.

Pathological Anatomy. — When endocarditis attacks mitral cusps, it causes thickening and contraction, and as a rule the chordae tendineae suffer as well. Either (1) the disease causes a funnel-shaped orifice, due to the adjacent edges of the cusps becoming adherent and the chordae tendineae matted together and greatly shortened, the appearance suggesting that the papillary muscles are actually in contact with the valve curtains, while the apices of the papillary muscles become markedly fibrous; or (2) the button-hole type of stenosis may

be produced, in which the disease specially involves the base of the cusps, the cusps being drawn up and puckered, and in this type the chordæ tendineæ are less affected. Both these varieties may be complicated by atheroma. The first type is believed to be common in young persons, the second type being associated with the disease in more advanced life, and it is hardly necessary to add that there are many cases which represent a combination of the two types together. In an advanced case, especially of the funnel-shaped type, the opening of the orifice may be reduced to an excessively small size.

As a rule in uncomplicated cases of mitral stenosis the left ventricle is not enlarged, and the changes in the other chambers are the results of backward pressure. They include dilatation and slight hypertrophy of the left auricle, dilatation and hypertrophy of the right ventricle, and often great dilatation of the right auricle. There is usually tricuspid incompetence when compensation has failed, and brown induration of the lung with marked pulmonary œdema results. Clots, either from the right auricle or from the tricuspid valve, not infrequently lodge in a branch of the pulmonary artery and cause pulmonary embolism: sometimes large ante-mortem thrombi may be found in both auricles, and especially in their appendices.

Clinical Features.—For many years there may be no symptoms at all. Usually dyspnœa, especially on exertion, and palpitation, are amongst the first symptoms which suggest cardiac disease. In certain cases, during an attack of rheumatic fever there is evidence of endocarditis, eventually giving rise to stenosis of the mitral orifice. When compensation fails, great cardiac irregularity results, and the entire group of symptoms referable to backward pressure and described under mitral incompetence may be met with. Bronchitis is common, and the degree of œdema may be similar to what is found in mitral insufficiency.

Physical Signs.—Inspection.— The apex-beat, if displaced at all, is usually displaced somewhat outwards. In cases where compensation is lost, there may be distension of the veins in the neck, and there is sometimes pulsation over the præcordial region in the 2nd, 3rd, and 4th left interspaces, while epigastric pulsation may indicate enlargement of the right heart. *Palpation.*—A well-marked thrill, presystolic in time,

is generally felt in the mitral area; it is extremely rough, and certainly closely resembles the sensation obtained if the hand is applied to a cat's head when the animal is purring. In cases where the thrill is not very obvious it may be increased by exertion. The apex-beat is forcible, and there is sometimes a well-marked, although not so rough, diastolic thrill. There is usually epigastric pulsation, owing to the enlargement of the right ventricle. *Percussion.* There is increase of cardiac dulness to the right of the sternum sometimes the distended left auricle increases the area of dulness slightly upwards, but rarely does the left border of the heart extend much further outwards. *Auscultation.*—A rough, short, presystolic murmur leads up to the first sound. It is probable that the so-called first sound is not in reality due to the closure and stretching of cusps, which are, too often, so rigid as to render actual occlusion of the orifice absolutely impossible, but the first sound may be due to the muscular wall of the left ventricle contracting on the blood in its chamber. A systolic murmur of mitral incompetence is frequently associated, and in well-developed cases there is also a diastolic murmur. The diastolic murmur is unquestionably a suction murmur. It is due to the active relaxation of the left ventricle sucking blood through the stenosed mitral orifice, and hence, probably, the great length of the murmur, and also the fact that it is rarely so loud as the presystolic. There may, therefore, be a triple mitral murmur associated with mitral stenosis, and in some cases it seems as if the murmurs were continuous.

A reduplicated second sound is almost constantly associated with mitral stenosis, and it has been suggested that one part of this reduplication is actually produced at the mitral orifice, and may be, in effect, a short murmur, and it is noted that the apparently reduplicated second sound, heard in the mitral area, is often quite different from the reduplicated second heard at the base of the heart. The pulmonary second sound is invariably accentuated so long as compensation is maintained, and the forcible closure of the pulmonary cusps may even be palpable with the finger. The disappearance of a previously accentuated pulmonary second sound is one of the signs of loss of compensation.

The pulse may be slow and regular, but when compensa

tion is lost, great irregularity generally results. It is, however, of interest to note that the irregularity is not merely one of time, but also of character of each individual pulse wave. The force and quantity of blood in consecutive waves often show wide differences, and may constitute an important and valuable aid to diagnosis. It occasionally happens that the murmurs of mitral stenosis temporarily disappear, and an even more remarkable fact is sometimes observed, namely that a presystolic murmur suggesting mitral stenosis may be present, without any actual narrowing of the mitral orifice being in existence.

Diagnosis.—Should a presystolic murmur be lacking in doubtful cases, it is necessary to make the patient walk about, or alternately sit up and lie down once or twice in bed so as to bring out the auscultatory signs clearly and on the other hand, should the heart be very irregular and rapid, rest in bed for some time may enable a diagnosis to be made.

The **Prognosis** depends much on the degree of compensation present, and also on whether tricuspid stenosis is developing or has developed. Its appearance is invariably a danger signal, but its incidence with mitral stenosis, its commonest combination, is rare.

5 Mitral Incompetence

As the name implies, it is insufficiency of, or regurgitation through, the mitral valve.

Etiology.—1. It may be the result of atony, or dilatation of the muscular walls of the left ventricle, so that the valve segments are no longer able to cover the orifice. This condition is present in mild degree in anaemia, or after any debilitating disease, while great enlargement of the left ventricle may be the result of Bright's disease, or of a valvular lesion at the aortic or other orifice.

2. Endocarditis, when involving the mitral valve generally causes stenosis, but sometimes, with or without any very marked constriction of the orifice, the valve segments or the chordae tendineae may become contracted or the site of vegetations, and so prevent adequacy of the cusps. Very generally we find, secondary to endocarditis, a certain amount of calcareous deposition not infrequently at the base of one of

the cusps, sometimes even affecting the greater part of the cusp or cusps previously attacked by endocarditis, and in such a case it is extremely improbable that the rigid cusps can close the orifice.

3. There are also cases which are arterio-sclerotic in which no endocarditis is or has been present. In a mild degree this is a frequent result of age and hard work, and where it is in excess it causes marked thickening of the cusps with secondary deposition of lime salts.

Pathological Anatomy.—Dilatation of the orifice referred to under the first head, and endocarditis, with or without calcareous degeneration, mentioned under the second, have already been sufficiently described when we considered the etiology, but the results of incompetency of this orifice demand a brief reference here.

There is usually dilatation with hypertrophy of the left ventricle, and if the change is permanent, and not merely temporary, the left auricle speedily becomes dilated. Backward pressure in the lungs will follow next, causing brown induration, and not infrequently atheroma of the pulmonary arteries, and in a short time the right heart becomes involved. Tricuspid incompetence follows, the right auricle becomes dilated, and the general venous system is affected, this giving rise to general oedema with failure of urinary secretion. These changes imply loss of compensation, and the clinical features typical of the condition include dyspnoea, the expectoration of much watery fluid from the lung, the embarrassment of breathing due to pleural effusions, and sometimes, owing to pulmonary embolism, considerable haemoptysis.

Restoration of compensation implies the relief of these and other symptoms, and it is generally brought about by hypertrophy enabling the more affected chambers of the heart to resume their functions.

In chronic Bright's disease and in primary cirrhosis of the kidney, hypertrophy of the left ventricle may fail, in which case dilatation occurs, and the mitral orifice becomes incompetent; and in a case of adherent pericardium similar dilatation may result.

Clinical Features.—For a long period of time there may be no symptoms at all, and it must be remembered that so long as compensation is perfect, a considerable degree of

mitral incompetence may be present without discomfort to the patient.

Amongst the earliest manifestations will probably be a degree of cyanosis of lips and face, shortness of breath on any continued exertion, and a tendency to attacks of palpitation.

When compensation has failed, the following clinical features are present:—palpitation with marked cardiac irregularity, great dyspnœa with a definite degree of cyanosis; all the signs of backward pressure referable to the lung, such as watery expectoration frequently stained with blood; those referable to the stomach, such as vomiting and sickness; and those referable to the kidneys, as evidenced by the passage of scanty urine containing albumin and sometimes casts. One of the most noticeable signs of a mitral incompetence in which the patient has lost compensation, consists in the rapid development of general dropsy, beginning in dependent parts, and tending to involve the peritoneal sac, the pleurae, and even the pericardium.

Physical Signs.—Inspection usually shows, in young persons bulging of the præcordial region, and generally marked and diffuse pulsation. The apex-beat is displaced downwards and outwards, and there may be distension and pulsation in the veins of the neck. *Palpation.*— The apex-beat is generally diffuse, and is sometimes irregular when compensation has failed, but a thrill in the mitral area is distinctly rare with an uncomplicated lesion. *Percussion.*—The left border of the heart is displaced outwards, there is great increase both of relative and, as a rule, also superficial cardiac dulness, and when the right heart has become involved by the backward pressure, there is also marked increase of dulness to the right of the sternum. *Auscultation.*—The first sound is usually more or less replaced by a systolic murmur. The murmur is soft and blowing, is rarely musical, has its point of greatest intensity in the mitral area, and is propagated towards the inferior angle of the scapula, and sometimes upwards to a point just outside the pulmonary area. In rare cases it may be heard all over the chest. It should not be forgotten that a presystolic murmur, will be associated in patients who have mitral stenosis, and that, when tricuspid insufficiency is superadded, a systolic murmur, different, however, in character, should be

heard over the lower part of the sternum. The second sound in the pulmonary area is usually accentuated, although when compensation has failed, it is important to note how the previously accentuated sound becomes feeble. Not infrequently marked irregularity indicates commencing loss of compensation.

Diagnosis and Prognosis.—Recognition of the murmur is usually easy: the difficulty is to decide upon its significance. In temporary cases, such as in anaemia, the murmur rapidly disappears with treatment, whereas if it is the result of definite organic disease, and especially endocarditis, it is improbable that much change will result in its character. The degree of enlargement of the heart, and the evidence of stenosis of the mitral orifice, where they are present, are distinctly helpful. In conclusion, temporary incompetence disappears; permanent incompetence, although alleviated by rest and treatment, does not.

6) Tricuspid Stenosis

Constriction of the tricuspid orifice.

Etiology. This lesion, at one time believed to be of very rare occurrence, is met with in a small proportion of cases of mitral stenosis, which it follows as a sequel, although it rarely develops to an extreme degree. It may also be present as a congenital lesion, and if so, is likely to be associated with other cardiac lesions of similar origin.

Pathological Anatomy.—The affected orifice shows, but generally to a limited extent, the pathological conditions present in mitral stenosis. The orifice may be funnel-shaped, and there is always a certain amount of rigidity and sometimes vegetations. As regards the cardiac chambers, the right auricle is dilated and may even be slightly hypertrophied, although the muscular elements in the wall are rarely increased to the same extent as in the left auricle in mitral stenosis.

Clinical Features.—The symptoms presented by the patient are an exaggeration of those met with in mitral stenosis. There is more cyanosis and evidence of backward pressure; the veins in the neck become engorged, and may show distinct pulsation.

Physical Signs.—On *Inspection* one notes this engorgement

of the cervical veins, often with a very large auricular wave. *Palpation* may reveal a presystolic thrill felt over a wide area, and situated much to the right of the region where the mitral thrill is usually felt. *Percussion* demonstrates that the cardiac dulness is increased, particularly to the right of the sternum, and on *Auscultation* the murmur is usually a presystolic bruit with its point of greatest intensity in the tricuspid area, although it may be audible over the whole of the lower part of the sternum. It must, however, be remembered that the murmur may be absent, in which case the condition is probably never suspected during life. In many cases where tricuspid stenosis is present, it is diagnosed by the presystolic murmur, well marked in the mitral area, being audible over an unusually extensive region extending towards the right.

Very rarely is there a tricuspid diastolic murmur present.

There may be a polycythaemia, *i.e.* an increase of red cells in the blood, 7,000,000 to 9,000,000 per cubic millimetre being present.

The **Diagnosis** depends in most cases on whether, in addition to a mitral presystolic, there is a tricuspid as well. Careful auscultation, and above all the recognition of two points of maximum intensity of the presystolic murmur, are most important.

The **Prognosis** is bad, because the difficulties of the circulation are much increased owing to the tricuspid constriction being as a rule superadded to the mitral lesion so generally present.

7 Tricuspid Incompetence

Etiology. Regurgitation through the tricuspid orifice may be the result of:—(1) acute or chronic endocarditis involving the tricuspid segments, or much more commonly (2) backward pressure, the initial lesion being often found at another orifice. In this way mitral stenosis and incompetence will lead in time to inadequacy of the tricuspid orifice; but it should also be stated that disease of the lungs greatly increases the work of the right ventricle and may lead to its dilatation. Emphysema and extensive fibroid disease of the lungs sometimes produce tricuspid incompetence. Further, in all diseases in which the nutrition suffers temporarily or

permanently, enfeeblement of the heart muscle may result and lead to insufficiency of the tricuspid orifice.

Pathological Anatomy.—Where the cusps are puckered as the result of endocarditis, the condition in no way differs from what is found at the mitral orifice. Where there is dilatation, it is due to enlargement of the right ventricle, and the muscular ring surrounding the tricuspid orifice is certainly less capable of resisting pressure than that which aids in guarding the mitral orifice. As an inevitable sequel the right auricle dilates, and the degree of this dilatation may be extreme.

Backward pressure in lungs, liver, spleen, and kidneys is even more marked than in mitral disease.

Clinical Features.—In referring to mitral lesions, a brief description has already been given, not merely of the clinical features of tricuspid incompetence, but also of its results. In slight cases there may be comparatively trifling symptoms, but where compensation is lost the physical signs are very definite, and the symptoms include dyspnœa and a varying amount of cardiac distress.

Physical Signs.—On *Inspection* the pulsation in the neck, which may be auricular or ventricular in time, is well seen in the engorged jugular veins, and it may be distinguished in the internal as well as in the external jugulars. Auricular pulsation implies that each contraction of the right auricle is registered by a backward wave into the jugular, and a systolic wave implies that the contraction of the right ventricle can transmit, directly, or possibly through the stretched tricuspid valves, a definite wave into the vein. There are really three waves visible in a trace taken from the jugular vein, the auricular wave, a wave synchronous with the carotid pulse, and the ventricular wave proper, which immediately follows the ventricular systole. The appearance of a double wave in the vein visible to the naked eye indicates that both the auricle and ventricle register their contractions, but where the auricle is paralysed from excessive dilatation, the auricular element disappears, leaving only the ventricular pulsation, and in certain cases no visible pulsation is seen at all, but the jugulars appear turgid and almost motionless. The waves, to which reference has just been made, must not be confused with the normal undulation often noticed in jugular veins in health, and seen better in the external than in the internal jugulars.

The venous pulsations, which have just been referred to, are not merely extremely visible, but may actually be transmitted into many of the larger venous trunks, while sometimes pulsation of the liver may be made out, synchronous with the contraction of the right ventricle.[1] Cyanosis is often very pronounced, the lips, tip of nose, ears, and extremities being specially blue, and oedema of the legs and feet develops early. *Palpation* enables the finger to assist the eye in recognising the displacement of the apex beat outwards and also the presence of systolic epigastric pulsation, if the right ventricle is greatly enlarged. *Percussion* affords definite evidence of the enlargement of the heart to the right of the sternum, and on *Auscultation* a systolic tricuspid murmur can generally be heard over the tricuspid area and propagated over the lower part of the sternum. It is a soft, blowing murmur, often difficult to distinguish owing to the presence of a mitral systolic, but it is heard over the lower half of the sternum, and therefore differs in area from that which characterises a mitral systolic murmur.

The evidence of backward pressure in other organs is important, and may include pulsation in the liver, and the recognition of the primary lesion, whether in heart or lung, greatly aids the diagnosis.

The **Prognosis** is usually somewhat grave. It is true that a mild degree of tricuspid incompetence is not serious, but when it becomes a prominent feature, it indicates that compensation has broken down or is on the point of doing so.

A tricuspid murmur due to endocarditis is always grave because it almost invariably implies that other valves are also affected.

(8) Pulmonary Stenosis

Etiology.—It is generally congenital, but even then is extremely rare. It may be associated with a patent ductus arteriosus, or an imperfect inter-auricular or ventricular septum. In other cases it results from endocarditis, perhaps most

[1] Recently much attention has been directed to the significance of *auricular fibrillation*. Instead of one point where there is stimulus to contraction, there are numerous points of excitation to contraction of the auricle, and many small wavelets are seen in the jugular veins as a result. It is often noted in cases of great backward pressure in the right heart, and is not infrequently amenable to treatment by digitalis.

frequently occurring in young children and associated with a severe type of endocarditis in one of the continued fevers. In a few cases it is sclerotic in origin.

Pathological Anatomy.—The common form of stenosis is due to the fusion of the cusps together to form a funnel-shaped opening, but in other cases the cusps may be thickened and retracted, with or without the presence of vegetations, while a varying degree of ulceration is sometimes present.

Clinical Features.—There is always great dyspnœa and cyanosis, which is extreme in congenital cases, but more moderate in others. It is in these congenital cases that one notes the typical clubbing of the tips of the fingers and toes, so constantly associated with the morbus cæruleus, and there is often a pulsating liver.

Physical Signs.—*Inspection* shows displacement of the apex-beat outwards rather than downwards, and there is generally epigastric pulsation. On *Palpation* a systolic thrill is almost always felt over the pulmonary area. *Percussion* demonstrates an enlargement of the right heart in most cases, and on *Auscultation* a harsh systolic pulmonary murmur may be heard, limited to its own area, but in any case not propagated into the carotids.

The **Diagnosis** is easy. An *aortic systolic murmur* is propagated into the carotids; while in a *patent ductus arteriosus* the murmur is late systolic in time, and is best heard at a point in the second left intercostal space external to the pulmonary area.

It is the case that a pulmonary systolic is a common "functional" murmur in cases of anæmia, etc., but a true pulmonary stenosis gives rise to the most definite thrill, murmur, and clinical features, all of which sharply differentiate the one condition from the other.

The **Prognosis** is unfavourable, partly because there is a tendency, in cases in which the lung is badly supplied with blood, to the development of pulmonary tuberculosis.

9) Pulmonary Incompetence

Etiology.—This may be the result of conditions similar to those described under aortic incompetence, but sclerosis of the pulmonary cusps, comparable to what occurs at the aortic orifice, is very rare. On the other hand the pulmonary orifice

can be stretched in such a way as to render the cusps inadequate, and therefore, in conditions in which there is severe backward pressure, pulmonary incompetence may occur temporarily. Endocarditis of the pulmonary orifice is rare, but may develop in connection with acute rheumatism or one of the infective fevers.

Pathological Anatomy. The cusps may be thickened, twisted, and sometimes adherent, so as to give rise to stenosis, and not infrequently there are vegetations, often associated with ulceration. It should, however, be stated that in cases in which the incompetence is the result of over-distension, the valve cusps may be perfectly normal, and after death the orifice may have regained its normal size. A permanent pulmonary incompetence results in dilatation with hypertrophy of the right ventricle, and when the tricuspid orifice becomes in turn incompetent, dilatation of the right auricle ensues.

Clinical Features.—It is very difficult to give a satisfactory description of the results of pulmonary incompetence. Where it is of organic origin, it will certainly cause marked dyspnœa, cyanosis, and other evidences of an extreme degree of backward pressure.

Physical Signs. On *Inspection* the veins in the neck are seen to be distended and to pulsate. On *Palpation* the apex-beat is found to be displaced to the left, and there is epigastric pulsation due to the enlarged right ventricle. The *Percussion* dulness is increased both to the right and to the left, and on *Auscultation* a murmur, generally soft and blowing, occasionally harsh and rasping, is heard in the pulmonary area. It is diastolic in time, its point of greatest intensity is in the second left space close to the sternum, and its line of propagation is downwards towards the right ventricle. It is important to note whether the second sound, which ought to be produced in part at the pulmonary orifice, is completely replaced by the murmur, or is still audible.

The **Diagnosis** lies between *aortic incompetence* and pulmonary incompetence. In aortic incompetence, the murmur is propagated towards the apex of the heart, and in most cases a double aortic murmur may be heard in the carotids, whereas a pulmonary diastolic murmur is only propagated over the right ventricle, and is not, even when associated

with a pulmonary systolic murmur, propagated into the carotids. The murmur of pulmonary regurgitation is appreciably increased during expiration. The water-hammer pulse and capillary pulsation are present only in aortic incompetence. The chief difficulty arises in cases in which both lesions are coexistent.

The **Prognosis** is bad in all cases, and especially where the lesion is of a permanent nature.

Treatment of Heart Disease

A case of heart disease may be divided into three stages:

1. The stage before compensation is lost, and when very probably the patient does not present himself before the physician.

2. The stage when compensation is lost, a stage which may recur again and again during the life-history of any given cardiac case.

3. The stage when compensation after being lost has been regained.

Stage 1. During the first stage, should the cardiac condition be recognised, do not order any cardiac tonic, but rather endeavour to regulate the life and work of the individual so as to preserve compensation. Give strict directions as to moderation with regard to food, drink, and tobacco: in most cases forbid either too hot or excessively cold baths, and especially veto Turkish or Russian baths. Counsel the patient to avoid excitement, and remember the risk of sexual intercourse in serious cases of aortic incompetence, even at an early period in the disease. Should any drugs be ordered at all, give iron, arsenic, and general tonics.

Stage 2. When the second stage is reached, the first thing is to place the patient absolutely at rest in bed, and so aid the heart to regain compensation, if possible, without having to resort to any direct cardiac tonic. Should a cardiac tonic, however, be unavoidable, we may prescribe digitalis in the form of tincture (℥ 5-15), or the infusion (℥ 2-4). Digitalis acts powerfully on cardiac cases, causing a fuller contraction of the ventricles, and thus strengthening the action of a weak heart, and rendering it more regular, and, at the same time, slower. It increases the flow of urine, and of its glucosides

digitalin and digitoxin are powerful vasomotor stimulants while digitonin, which is believed to be present in larger amount in the infusion, is a valuable vasodilator agent, acting like saponin. There is much difference of opinion as to the reputed cumulative properties of digitalis but certainly it is wise to intermit the drug for a few days after its continuous administration for say 3 or 4 weeks. Like strophanthus, digitalis is apt to disagree with the patient's stomach, and may induce sickness, and it should be combined, as a rule, in such cases with a carminative. If given in excess, digitalis causes great rapidity and irregularity of cardiac action, and tends to arrest the renal functions.

Strophanthus is often preferable to digitalis, and especially where the vasoconstrictor action is not desired. It is better borne by the stomach, but should invariably, in cases where there is any tendency to irritability, be given in combination with a carminative, such as tincture of capsicum ℥ 1-5. Tincture of strophanthus is administered in doses of 5 to 15 minims; and although it is desirable to watch the effect with care, even when doses of only 10 minims thrice daily are being administered, it is, nevertheless, a drug which may be pushed in certain cases with great advantage. Strophanthin is sometimes preferred in doses of $\frac{1}{300}$th to $\frac{1}{100}$th of a grain.

Caffeine citrate is of special value after one or other of the preceding drugs has been administered, and when, perhaps owing to dyspepsia or other cause, it has been temporarily interrupted. The dose of caffeine citrate varies from 5 to 30 grains.

Indirect relief is afforded to the heart by (1) venesection, by appealing to the (2) bowels, (3) kidneys, and more rarely (4) to the skin, (5) by opening up the vascular channels by the administration of one of the nitrite group of remedies, and (6) by tapping dropsical effusions where they have become excessive.

(1) Bleeding is specially serviceable in extreme cases of backward pressure, with engorgement of the right heart. The median-basilic vein should be selected for the operation, and 15 to 30 ounces of blood removed. Follow the operation with stimulants or cardiac tonics.

(2) Depletion by the bowels is carried out by the administration of hydragogue cathartics, such as magnesium

sulphate (3 2-6), given as a concentrated solution in the morning before breakfast, or compound jalap powder (grs. 30-50).

(3) Depletion by the kidneys is aided by such diuretics as spirit of nitrous ether in drachm doses every 4 or 6 hours or by diuretin in 10 to 15 grain doses.

(4) Depletion by the skin is rarely safe practice, and diaphoretics, other than the simple liquor ammonii acetatis, should not be given.

(5) For dilating vessels $\frac{1}{100}$th of a grain (nitroglycerine generally administered in a solution (liquor trinitrini), is one of the best agents.

(6) The removal of dropsical effusions from pleura, peritoneal sac, and the subcutaneous tissue of dependent parts of the body, often gives great relief to the heart. Potain's aspirator is generally used for tapping the pleura and sometimes for an ascitic effusion, while Southey's tubes are usually employed for the abdomen, and almost invariably for draining fluid from the legs.

A very important matter in the treatment of cases of heart disease, is the proper quantity of fluids which should be allowed to the patient. A dry diet, by which is meant the limitation to 20 or 30 oz. of fluids a day, diminishes oedema, and throws less strain on an overworked heart.

There are also *Special Symptoms* which demand treatment. *Dyspnoea* is relieved by resting the patient in bed, by tapping the pleura, by giving such stimulants as equal parts of the spirits of ammonia, ether, and chloroform in drachm doses, by cupping the bases of the lungs, and by oxygen inhalations. Paroxysms of dyspnoea sometimes call for morphia. *Palpitation* should be treated by applying an ice-bag over the heart. *Dyspepsia* and *vomiting* require most careful dieting, and dilute hydrocyanic acid (♏ 3-5) and subnitrate of bismuth (gr. 10-30) are serviceable, while a mustard leaf or poultice applied over the region of the stomach is often of great value in allaying intractable vomiting; effervescing alkaline drinks, such as potash or soda water, are also good. *Cough* is relieved by such stimulants as spirit of chloroform or alcohol, and *sleeplessness* may require paraldehyde (♏ 30-90), or even morphia (gr. $\frac{1}{6}$-$\frac{1}{3}$) hypodermically.

Haemoptysis is beneficial when not excessive, but, unfor-

tunately, it begets fear, and then a mineral acid with the liquor morphinae ♏ 10-15, should be given to soothe the patient.

A case of *aortic incompetence* requires special directions; here such a cardiac tonic as digitalis, by lengthening the ventricular diastole, is said by some authorities to be hurtful and even dangerous; but given with care it will prove invaluable where compensation has failed (although we prefer strophanthus). Opium is frequently of great benefit, and this drug, together with potassium iodide, should invariably be accorded a fair trial, more especially in cases where anginous *pain* is present. Opium relieves the sleeplessness, dyspnœa, and cardiac asthma which are often present in this form of heart disease.

In a case of long-standing heart disease, whether valvular in origin or due to cardiac dilatation resulting from degenerative or fatty changes in the heart muscle, a time comes when rest and drugs are insufficient. In certain of these cases cautiously increasing the work of the heart seems to be of special value in toning up the diseased organ.

We have carried this out in the following way. The patient, after complete rest in bed and the usual cardiac tonics and other treatment, is subjected to 1 massage for 1 to 3 or more weeks; 2 passive exercises or movements of the limbs for a similar period; to be succeeded by 3) resistance exercises, more or less following the prescribed method of Dr. Schott of Nauheim; and then, lastly, 4 he is allowed to get up and walk for half an hour or thereby, and gradually day by day increases the personal effort.

The results of this treatment are excellent where the heart is still capable of improvement, and the consequent diminution in the size of the dilated chambers is extremely striking, while the improvement as regards the patient's general well-being is manifested by increased appetite, by better sleep at night, and by greater cheerfulness. Oertel's hill-climbing exercises are more applicable for cases of fatty infiltration of the heart.

The bath system of treatment, as carried out at Nauheim, deserves a few words of description:

(1) The patients begin with baths at a temperature of about 95° F., the bath containing 1 lb. of sodium chloride, which is gradually increased to 3 lbs., and 1½ oz. of calcium chloride gradually increased until 4½ oz. are present. The bath is 10

gallons in capacity, and the patient remains from 5 to 10 minutes in the water, gradually extending the duration of the bath until 20 minutes is reached, while the temperature is lowered to 84° F. These baths are taken on alternate days or one day out of three and a period of rest in the recumbent posture follows each bath. In two weeks the patients are ready for—

2. The effervescing baths, which contain the full strength solution of the salts mentioned above, and to which are added 2 oz. of sodium carbonate, gradually increased to 8 oz., and 3 oz. of hydrochloric acid, gradually increased to 12 oz.

3. Sometimes an undercurrent douche is used with the effervescing baths. The baths may be prepared artificially, as indicated by the quantities mentioned above, the solution of salts and of carbonic acid gas closely imitating the natural mineral waters of Nauheim and the strength in which these are used for treatment.

Stage 3. When the stage of restored compensation is reached, the duty of the physician is directed to preserving it; and, as suggested under stage 1, the diet, life, and work of the patient should be carefully arranged and regulated. Arsenic is a useful drug in many cases during this period, because it conserves the cardiac energy.

VI. CARDIAC HYPERTROPHY

It is doubtful whether cardiac hypertrophy ever occurs apart altogether from dilatation, and it seems most probable that although the hypertrophy may be the most prominent feature, dilatation invariably accompanies it.

Etiology.—A chamber of the heart hypertrophies when extra work has been gradually thrown upon it; wherever dilatation has occurred, hypertrophy is the process by which compensation is restored. Hypertrophy is the result of excessive stimulation, as by over-exercise, excitement, or the action of certain toxic agents in harmful amount, such as digitalis, alcohol, tea, etc. The *Left Ventricle* hypertrophies in Bright's disease, in which the arterial resistance becomes greatly increased, and also as the result of aortic and to a less extent, mitral lesions, which throw greater work on that chamber.

The *Right Ventricle* hypertrophies where backward pressure, however produced, renders the work of the chamber greater, and similarly the *Left*, and to a much less marked degree the *Right Auricle*, may hypertrophy where the mitral or tricuspid orifices respectively are narrowed, thereby increasing the effort necessary to empty the chamber. Pericardial adhesions may cause increase in size of the heart as a whole.

Pathological Anatomy Although hypertrophy has been specially referred to under the heading of etiology, as already stated it is always accompanied by more or less dilatation, and with the exception of the left ventricle the dilatation is always a more prominent characteristic than the hypertrophy. The wall of a hypertrophied left ventricle increases in thickness, the columnæ carneæ greatly increase in size, and in the auricles the musculi pectinati become much thicker. The capacity of the chamber is also increased, although this may be only slightly so in the case of the left ventricle.

Clinical Features Where hypertrophy predominates in the *Left Ventricle* as in chronic Bright's disease marked palpitation results, with, not infrequently, considerable headache and general discomfort.

The *Physical Signs* show enlargement of the left side of the heart and the displacement of the apex downwards and outwards, while the first sound in the mitral area has a peculiarly booming character. The pulse is often full and strong.

Hypertrophy of the *Right Ventricle* is evidenced by epigastric pulsation, extension of cardiac dulness to the right of the sternum, and the other phenomena due to the primary cause of the condition.

Hypertrophy of the *Auricles* gives rise to no special features.

The **Diagnosis** is rarely difficult so far as the left ventricle is concerned.

The **Prognosis** depends largely on the possibility of maintaining compensation. The greater the amount of concomitant dilatation the more apt is the enlarged heart to cause trouble.

The **Treatment** already described under palpitation yields the best results. Where there is no valvular cause for the hypertrophy, a quiet, well-regulated life, together with ac-

administration of sedatives, and especially tincture of aconite, will often yield beneficial results, while the patient should be discouraged from taking an undue interest in his cardiac condition.

Hypertrophy, where it merely compensates for the damage done by a valvular lesion, does not call for treatment.

VII. CARDIAC DILATATION WITH COMPENSATORY HYPERTROPHY

This is simply what obtains in a case of valvular or cardiac disease in which compensation has already been so far lost and is again restored. It is unnecessary to describe here the condition of the heart common to most forms of ordinary valvular disease, and the clinical features in a marked case are those of a patient whose compensation is barely maintained. There is palpitation, pain, breathlessness on exertion, and a tendency to cardiac failure after any excessive effort above the compensatory powers of the heart. The *physical signs* show enlargement of the chamber or chambers. In the case of the left side of the heart, percussion dulness demonstrates an extension both downwards and to the left, and where the right side of the heart is affected, the border is markedly displaced outwards to the right. The engorged veins, and the irregularity in both heart and pulse, yield significant signs, and the treatment consists in an attempt to diminish the dilatation and to increase the residual energy of the heart.

VIII. CARDIAC DILATATION

By this is meant enlargement of a chamber or chambers of the heart without proportionate and compensatory increase in their muscular walls.

Cardiac dilatation is a common result of any acute febrile process, as well as of profound anaemia in which the heart wall is poorly supplied with blood. It may also occur in diphtheria, or because of some toxin acting on the heart wall or possibly the cardiac centres: tobacco is an excellent example of an agent of this kind. Further, dilatation of the

heart is secondary to valvular lesions, myocardial degeneration, disease of the coronary arteries, Bright's disease, and many other conditions.

Overstrain is often an exciting cause of dilatation, and if the heart is damaged from valvular disease, malnutrition of the heart wall, the action of a toxin, or an unsatisfactory blood-supply, it acts with greater effect, and a smaller amount of effort produces a serious degree of distension.

A dilated chamber of the heart does not become completely emptied during its systole, and the residual blood tends by degrees to increase the dilatation. Very rarely rupture of a dilated heart occurs. As a general rule the muscle fibres are over-stretched, and the chamber does not recover its compensation until sufficient hypertrophy ensues.

The **Clinical Features** are those of loss of compensation, with the phenomena of both increased backward pressure and diminished forward pressure. There is much cardiac distress, often an irritative type of palpitation, and faintness is induced by the slightest effort. The digestion is poor, and constipation, more rarely diarrhœa, adds to the troubles of the patient. Sleeplessness is frequently complained of, and general dropsy soon becomes marked.

The *Physical Signs* are definite. There is not merely enlargement of the affected chambers of the heart, or the heart as a whole, but the apex-beat is feeble and often irregular, while on *Auscultation* the sounds of the heart are loud, but lack strength, and have been well described by the term "flapping." Further, the gallop-rhythm is very typical, and in many cases Cheyne-Stokes breathing is noted during sleep. The engorged veins in the neck which often pulsate, the enlargement of the liver, and the many other symptoms and physical signs referred to under increased backward pressure, may be observed.

The **Diagnosis** is comparatively easy, although sometimes a *pericardial effusion* closely simulates a dilated heart (page 359).

The **Prognosis** depends on the possibility of the restoration of compensation, and therefore, to a considerable extent on the primary cause of the dilatation. In older patients, or in long-standing cases of heart disease, dilatation is grave.

Treatment.—While everything might be repeated which

has already been stated under the treatment of valvular lesions, the following points are worthy of special attention :—

1. Endeavour by rest to relieve the heart and to reduce the dilatation.

2. Remember that diuretics and purgatives will indirectly afford relief to the overburdened heart.

3. Give cardiac tonics and diffusible stimulants, although these should be administered with care.

4. Endeavour to relieve symptoms as they occur : and

5. Much may be done in the way of toning up the heart by massage and other methods described on page 401, although obviously there is a limit to the possibility of improvement in advanced cases.

IX. DISEASES OF THE MYOCARDIUM

1 Myocarditis

An acute or chronic inflammation of the myocardium.

Etiology.—An acute diffuse myocarditis is sometimes a sequel to diphtheria, scarlatina, and more rarely other fevers, but generally, whether acute or chronic, it is secondary to an endo- or pericarditis, and is more or less localised. It may also occur in pyaemia from the infective organisms entering the coronary circulation.

Pathological Anatomy.—In the diffuse parenchymatous form, there is a small-cell infiltration mostly leucocytic, and the muscle fibres appear pale and cloudy. In myocarditis, secondary to inflammation of the endo- or pericardium, bands of fibrous tissue tend to spread between the muscle fibres. Ulcerative endocarditis is apt to invade the myocardium locally, and in pyaemia small abscesses may form in the heart wall.

Clinical Features.—In the parenchymatous form praecordial distress with "fluttering" heart's action, rapid and irregular pulse with a tendency to vomiting, restlessness, and even delirium are common. In all the other varieties the symptoms and signs are inseparable from those of the primary condition, but feeble and irregular heart's action is certainly suggestive, and especially if cardiac dilatation be present.

Fibroid myocarditis is essentially, in its chronic form, the same process as fibroid degeneration of the myocardium without much evidence of any pre-existing inflammation, and the condition is included under the heading of "Fibroid Degeneration of the Myocardium."

The **Diagnosis** must be guided by the advent of grave phenomena, suggesting a serious involvement of the heart muscle.

The **Prognosis** is always unfavourable, and death is generally due to cardiac failure.

The **Treatment** must be directed in part to the cause of the condition. Insist on complete rest in bed, give stimulants cautiously, and apply counter-irritants (mustard blisters) over the praecordia.

Parenchymatous Degeneration of the Myocardium is a generalised condition, due to the toxins of certain organisms, in which the muscle fibres become pale and soft, and cardiac failure almost inevitably terminates life.

(2) Fibroid Degeneration of the Myocardium

This is due to anything interfering with the blood-supply to the heart muscle, or it follows where great hypertrophy of the heart wall has developed. It is similar in its result to the myocardial changes which follow adherent pericardium. Possibly alcohol and syphilis may cause fibroid changes in the myocardium.

The **Clinical Features** vary with the degree of involvement, but feeble and rapid cardiac action with pain are the most common. Little can be done by way of treatment. Tonics and potassium iodide are worth a trial.

(3) Brown Atrophy of the Heart and Other Degenerations

Brown atrophy, so common in old age, waxy degeneration, calcareous degeneration, and the hyaline degeneration of Zenker, require no description here. Fatty degeneration is discussed later.

(4) Fatty Heart

(a) Fatty Infiltration of the Heart

A deposition of fat resulting from general obesity and associated, more generally, with adult or later life. It occurs on the surface, and also, in some instances, infiltrates the muscular substance separating the fibres which generally atrophy, and the fat may even reach to the endocardium.

The **Etiology** includes the many causes of obesity, and among these excessive drinking of malt liquors might be particularised.

Pathological Anatomy.—The fat is excessive in those positions in which it is normally present in moderate amount, for example, along the line of the interventricular septum, the auriculo-ventricular groove, at the apex of the heart, and elsewhere.

Clinical Features.—There is evidence of enfeebled circulation, with breathlessness especially on climbing stairs. The first sound is often weak, and the pulse is poor in intravascular pressure. Patients with fatty heart are bad subjects for operation, and syncopal attacks occasionally supervene. More or less dilatation of the heart may also be present.

Diagnosis. The general evidences of obesity should suffice.

Prognosis. Fatty infiltration is amenable to treatment in cases not too far advanced.

Treatment.—Careful dieting, with little fatty, starchy, or saccharine foods. As a dry diet is most important, limit the amount of fluid taken per day to a minimum, and advise regular exercise with, in certain cases, Oertel's hill-climbing exercises. Give such tonics as iron (where required), arsenic, and strychnine, but rarely digitalis.

(b) Fatty Degeneration of the Heart

A true fatty degeneration of the muscular fibres, most frequently affecting the left ventricle.

Etiology.— (1) Failing nutrition from old age; (2) wasting disease; (3) pernicious anaemia; and (4) certain poisons, and especially phosphorus may cause a generalised fatty

degeneration. It is due also to local causes which act locally, such as atheroma of the coronary arteries, pericarditis, and myocarditis. It seems as if the heart muscle needed more oxygen than other muscular tissues.

Pathological Anatomy.—The muscular fibres are pale and soft, and show minute fat globules in rows, replacing the muscle substance: it is most obvious in the left ventricle where the typical thrush-breast-like appearance is well seen. The muscle becomes soft and friable, and hence the risk of rupture of the heart.

The **Clinical Features** include enfeeblement of the circulation, cardiac irregularity, dyspnœa, often Cheyne-Stokes breathing, palpitation, and sometimes syncope. There is a tendency to yawning, sighing respirations, and giddiness; not infrequently a corresponding enfeeblement of the mental faculties and memory supervenes. A measure of dilatation of certain of the cardiac chambers may be present.

The **Diagnosis** can only be suspected by the nature of the case and the group of symptoms present.

The **Prognosis** is bad.

The **Treatment** must be on palliative lines. A quiet life should be ordered with the use of tonics such as strychnine and arsenic, and only with great caution is digitalis or strophanthus to be administered.

(5) Myomalacia Cordis: Acute Necrosis of Heart

This is due to blocking of a branch or branches of a coronary artery, generally due to arterial degeneration. The coronaries are end-arteries, and therefore necrotic changes follow in the area of heart muscle deprived of blood. The muscle fibres lose their striæ and nuclei, and the whole patch affected becomes pale and yellowish in colour, and very friable. On the endocardium covering the infarction blood-clot forms. It is usually met with near the apex of the left ventricle, and the weakened part may give way, leading to the formation of a cardiac aneurism with eventual rupture and death.

Under any circumstances acute softening is rapidly fatal.

X. CARDIAC ANEURISM

1. *Of a Valve.*—In ulcerative endocarditis this is not uncommon. The aneurism may perforate, and generally the aortic orifice is the one affected.

2. *Of the Heart Wall.*—This is rare. It may be due to a patch of acute necrosis (myomalacia cordis), or some other condition which weakens a part of the ventricular wall. It is most often met with near the apex of the left ventricle. The aneurism may grow to be nearly as large as the whole heart, and may even bulge the chest wall. It tends to rupture into the pericardial sac.

XI. RUPTURE OF THE HEART

This may result from fatty degeneration or infiltration, or from acute softening, or may be due to the rupture of a cardiac aneurism. It causes sudden death, with great distress and intense dyspnœa.

XII. CONGENITAL MALFORMATIONS OF HEART

An arrest of development, which may sometimes cause conditions incompatible with life. Endocarditis may also be present.

There may be—

1. Deficiency or absence of septa, either the interauricular (patent foramen ovale) or interventricular (often at or near the undefended spot).

2. Alteration in number or arrangement of cusps. Where there are only two well-formed aortic cusps, the third cusp may be represented by a minute, undeveloped structure. Such a malformation is apt to predispose to endocarditis or degenerative changes at the affected orifice, which is generally either the aortic or pulmonary.

3. Persistence of fœtal structures, of which the foramen ovale has already been mentioned; another is the ductus arteriosus, which conveys the blood in the fœtus from the

pulmonary artery into the aortic arch. An imperfect division of the bulbus arteriosus is more rarely present. Sometimes stenosis of an orifice such as the pulmonary occurs, from maldevelopment.

4. Fœtal endocarditis may cause lesions similar to those occurring in later life, only more frequently met with in the right heart. The type of endocarditis present is generally chronic.

The **Clinical Features** will depend on the gravity of the lesion present. In a case of a very widely patent foramen ovale, often associated with other congenital malformations, the cyanosis is very obvious, giving the typical *morbus cæruleus*. The extremities are blue and cold, the fingers and toes are clubbed at their extremities, and there may be a varying degree of dyspnœa. There is a tendency to great backward pressure in such a case, with venous engorgement or even pulsation in the veins of the neck, and often a pulsating liver. There is a predisposition to bronchitis, and, if the blood-supply to the lung is very defective, to pulmonary tuberculosis. In the cases of congenital heart disease showing cyanosis there is generally a polycythæmia or increase in number of the red blood corpuscles (8 to 9,000,000 per c.mm.), and the hæmoglobin is 125 to 135 per cent.

Stenosis of the pulmonary orifice gives rise to a palpable systolic thrill, increase of cardiac dulness to the right (hypertrophy of the right ventricle), and a loud systolic pulmonary murmur.

A patent foramen ovale may give rise to neither symptoms nor signs, but cyanosis is generally present, sometimes without and sometimes with a murmur or murmurs.

A patent ductus arteriosus is recognised by a loud, somewhat late systolic murmur which is heard most clearly just outside the pulmonary area, and which may be continued beyond the second sound. It is often accompanied by a thrill.

Where a murmur is present as the result of other congenital lesions, it is rarely accompanied by a thrill, but may be associated, either with no enlargement of the heart at all, or with marked enlargement of the right heart only.

The **Diagnosis** is easy in very obvious cases, which present the clinical features above mentioned and which do so at an early age.

The **Prognosis** depends much on the lesion present. In a case in which there is great interference with the aeration of the blood, death is not uncommon from bronchitis, pulmonary tuberculosis, or mere inability to stand the wear and tear of life.

The **Treatment** should consist in the very careful upbringing of the patient, and the physician should order warm clothing, good food, and general tonics, especially cod-liver oil and iron.

XIII. DISEASES OF ARTERIES

(1) Inflammations of Arteries

(a) *Acute Arteritis*

May be (1) *Local*, where, for example, an artery is involved in an abscess or where an embolus or thrombus is present; or (2) *General*, where, as in certain fevers, the arteries are extensively affected. The general form is met with during or after an infective fever, and especially diphtheria, typhoid, and influenza. Either the infective organisms or their toxins are believed to be responsible for the condition.

The **Pathological Anatomy** of the *general* form consists in gelatinous-looking patches of varying size which appear in certain arteries. These patches are composed of embryonic cells which are arranged in rows, and invade along the lines of the vasa vasorum.

Thrombosis may follow and even gangrene of the affected limb or limbs.

(b) *Endarteritis Obliterans*

A small-cell proliferation of the deeper layers of the intima. The lumen of the affected vessel may become absolutely blocked from thrombosis.

Etiology.—Syphilis certainly, and possibly alcoholism and malaria, are believed to be the commonest causes of the affection.

The **Clinical Features** vary. There will probably be pain, often intermittent, together with local cyanosis, cramps, and numbness of the extremities, while gangrene may eventually develop.

c. Syphilitic Arteritis

This specially affects arteries at the base of the brain, and the preceding form of arterial disease (endarteritis obliterans) is the commonest syphilitic variety.

Pathological Anatomy.—Greyish-white, opaque nodules of cartilaginous consistence develop. They are due to a small-cell infiltration of the intima and of the other coats, and gummata may be associated. A syphilitic periarteritis sometimes occurs.

The **Clinical Features** are generally fairly definite, headache being common, and also paralysis, the extent and nature of which depend on the part of the brain deprived of its blood-supply.

(d) Tuberculous Arteritis

Tuberculous arteritis may be due to infection (with tubercle bacilli) by the blood-stream, or it is met with in pulmonary cavities or areas of tubercular disease where tubercle nodules attack the wall of an artery, penetrating from without inwards, and eventually leading, in some cases, to diffusion of tubercle bacilli into the circulation.

(2) Arterio-sclerosis (Gull and Sutton)

A diffuse or localised thickening of the intima of the arteries, secondary to primary changes in the media and adventitia. The term arterio-sclerosis is very variously employed, and atheroma, which is often closely associated with it, has been regarded as one of the forms of the disease. Here, however, atheroma will be described separately, and the term arterio-sclerosis is used in a limited sense.

Etiology.—It may be the result, *firstly*, of senile changes in vessels, often occurring at an early age in certain families. It should be remembered that in this group of cases atheroma is an almost necessary part of the process, and is really inseparable from it. *Secondly*, it may follow long-standing abuse of alcohol, and also arise from the effects of lead, gout, and syphilis. *Thirdly*, overstrain, and almost certainly overeating, predispose to its development; and, *fourthly*, in chronic Bright's disease, arterio-sclerosis is an important sequel.

Pathological Anatomy.—There may be thickening of all the coats of the artery, and pathologists differ with regard to the coat or coats that are first affected. The thickening of the intima referred to in the opening sentence, is a small-cell infiltration which represents nature's effort at strengthening an artery wall, whose other coats (media and adventitia) have been weakened. This thickening may be limited, or may involve a considerable extent of the vessels. Where the disease is widespread, there is invariably associated hypertrophy of the left ventricle and a cirrhotic change in the kidneys, and primary cirrhosis of the kidney is in reality a part of the disease in which the arterio sclerosis in the vessels and the enlargement of the left ventricle are also leading features, although it is probable that the condition may be primary in the vessels.

A diffuse form of arterio-sclerosis is often seen in middle life, the change, mainly a hyaline one, beginning in the media and involving the muscular fibres and the elastic tissue, while the intima over the affected part undergoes thickening. A similar sclerosis may involve the veins in these cases.

Unquestionably a small-cell infiltration is the initial lesion, and it is thought that it passes along the lines of the vasa vasorum.

Clinical Features.—There is marked thickening, with localised dilatations and constrictions of many of the arteries, and it may be easily recognised on palpating the radial artery in a typical case. The pulse is one of high intravascular pressure (200-300 mm. of mercury), and the enlargement of the left ventricle is almost constant. The aortic second sound is loudly accentuated, and the mitral first is generally of a booming character. The thickening of the vessels may, however, be in part due to what has been called "hypertonus," by which is meant vasomotor constriction. This temporary increase of intravascular pressure, which results from the presence of ptomaines or poisons circulating in the blood, is a common feature in cases of constipation, even where the vessels are absolutely normal, but hypertonicity adds to the pressure in the vessels in arterio-sclerosis, in which condition toxins are apt to be present in the blood-stream. The patient suffers from headache, and often from sleeplessness, and there is always a risk of the combination of high pressure

with weakened vascular walls leading to cerebral haemorrhage. Sometimes thrombosis occurs, where the degenerative changes in the cerebral arteries are very pronounced. The urine is generally excessive, and closely corresponds, in amount and character, with the urine excreted in primary cirrhosis of the kidney (*see* page 553).

Intermittent claudication is sometimes seen in cases of arterio-sclerosis. It consists in painful cramps, occurring very often in one leg after the patient has walked for some distance. It is believed to be due to vascular spasm. The limp caused by the cramp-like pain passes off after a short rest.

The **Diagnosis** is rarely difficult, the examination of vessels and heart generally aiding in the recognition of a well-marked case.

The **Prognosis** largely depends on the possibility of counteracting the etiological factors responsible for the onset of the disease.

Treatment.—Purgatives should be freely administered, overstrain should be prohibited, and alcoholic tendencies kept in check. The possibility of syphilis and of lead poisoning should not be forgotten. Careful dieting is important, and a remedy of the greatest value is iodide of potash, while the nitrite group, as, for example, nitroglycerine in doses of $\frac{1}{100}$th of a grain, often relieves the patient's symptoms.

ATHEROMA

Atheroma cannot be entirely separated from the preceding condition of arterio-sclerosis, and is sometimes described under that disease. Notwithstanding the risk of being criticised as being old-fashioned we have retained the separate description given in the first edition. Atheroma is essentially a senile change, but many diseases, such as gout, alcoholism, and syphilis, and constant overstrain tend to produce or increase it.

Pathological Anatomy.—Slight degrees of atheroma are present in the arteries of all persons after, or even before, middle life. There is a thickening of the inner part of the intima, in which fatty and, eventually, calcareous deposition occurs, and lime salts may be deposited in very large quantities, the disease interfering seriously with the lumen of the vessel.

Sometimes atheromatous cysts or abscesses occur in connection with these patches, and the patches are best marked at the commencement of the aorta and in the larger arterial trunks. The middle coat of the affected vessels becomes weakened, so that an aneurism may result and sometimes a dissecting aneurism is caused by the blood passing behind a calcareous plate and penetrating between the coats of the vessel. In the smaller arteries atheroma is invariably associated with arterio-sclerosis, and there is a tendency to thrombosis which, in the vessels of the brain may cause extensive softening, and in the vessels of the extremities may produce gangrene.

The **Clinical Features** vary greatly. The tortuous, thickened radial arteries with calcareous plates in the walls, can often be well palpated with the finger, and the loss of expansile power, in a marked case, is very typical. There is generally a systolic aortic murmur frequently due to thickening and sclerosis of the aortic cusps. The interference with the blood-supply to the organs of the body, and also to the extremities, gives rise to symptoms and signs varying in different cases, and varying with the degree of the interference. Many old patients complain of coldness of the extremities, and gangrene as already mentioned is not infrequently a final phenomenon.

The **Diagnosis** is generally easy, and the **Prognosis** depends on the degree of the condition.

The **Treatment** is unfortunately *nil*. All that can be done is to preserve life, and to treat symptoms as they occur.

(3) Aneurism

There are various types of aneurism which require a few words of description.

1. A *True* aneurism is one in which the sac is formed by the coats of the vessel.

2. A *False* aneurism has its sac formed by tissues outside the artery, the walls of the vessel having been eroded.

3. A *Dissecting* aneurism is one in which the blood finds its way between the coats of the artery, and either passes back again into the vessel or ruptures externally.

4. A *Cirsoid* aneurism is one in which there is great dilatation and tortuosity of the arteries which form it, and in which there is free anastomosis.

5. An *Arterio-venous* aneurism is an aneurism which communicates with a vein.

In shape aneurisms may be *Fusiform* or *Saccular*; in the former the vessel coats give way all round, in the latter the bulging is at one weak spot, and does not necessarily involve the whole circumference of the vessel.

Etiology.—Aneurisms may result from two groups of causes: (1) Local weakness of the arterial wall, and (2) Increased blood pressure.

(1) Local weakness may be due to arterio-sclerosis, and especially before compensatory thickening occurs. The dilatation may be a general yielding of a larger or smaller stretch of vessel, or may be quite localised. Anything inducing arterial degeneration may cause weakening, and injuries are also sometimes responsible; occasionally pyaemic emboli produce local weakening of the vessel wall and later dilatation. (2) Increased blood pressure is usually the result of overstrain, and cirrhotic changes in the kidney may be an important factor in the case.

(a) Intrathoracic Aneurisms

Etiology.—They occur mostly in men between 35 and 45 years of age, and rarely begin before 20 or after 50.

1. *Aneurisms of the ascending part of the Aortic Arch.*—Such an aneurism may develop close to the orifice, and if so is generally in one of the sinuses of Valsalva, and is the result of ulcerative endocarditis. It is of small size, and may give rise to no symptoms until it ruptures into the pericardial sac, pulmonary artery, or elsewhere.

If the aneurism is higher up, it is larger, and may be either a fusiform dilatation, or more rarely saccular. It may erode ribs or sternum, and may bulge in the 2nd or 3rd intercostal space on the right side.

Physical Signs.—On *Inspection* a pulsating, expansile tumour may be seen. On *Palpation* an impulse is sometimes palpable, and is expansile, while a thrill may be felt. *Percussion* generally yields a definite increase of dulness to the right of the sternum in the 2nd and 3rd spaces. On *Auscultation* a systolic murmur may be present over the sac. There may in some cases be aortic incompetence, with an aortic regurgitant

murmur, but only where the aortic orifice is involved by the aneurism, or where there is aortic incompetence in addition to the aneurism. The left ventricle is enlarged if the aortic orifice is interfered with, but is generally unaffected as regards size, although the heart may be pushed downwards by the tumour. Rarely the aneurism presses on the superior vena cava, causing distension of the veins in the neck and perhaps also in the chest.

The *Respiratory System* is as a rule unaffected; sometimes the right bronchus is pressed upon, but this is rare.

In the *Integumentary System* there are localised changes in the skin covering the aneurism, should there be very marked external bulging. The skin becomes red and thin, and may be covered by blebs which burst and sometimes lead to ulceration. Rarely an aneurism bursts externally, although it sometimes does so, and there may be leaking of blood or weeping, as it is called, prior to the final rupture.

In connection with the *Nervous System* there may be pressure on the right recurrent laryngeal nerve causing paralysis of the right vocal cord.

Pain is usually only a marked feature when there is pressure on bone or nerves.

2. *Aneurism of the transverse part of the Aortic Arch.*—This aneurism often involves the innominate artery. Sometimes these aneurisms are saccular and bulge into the neck, sometimes they are very large, and may erode the manubrium sterni, and may form a large tumour with several accessory sacs.

Physical Signs.—On *Inspection* we note the pulsating expansile tumour in the neck or in the region of the manubrium. *Palpation* demonstrates the pulsation and sometimes also the presence of a thrill. *Percussion* generally yields a very definite area of dulness, and *Auscultation* may reveal a systolic bruit. The pulse is often very suggestive, should the right subclavian be weaker than the left. This may be the result of the innominate artery being interfered with in, or near, the sac in one of the following ways:— *a*) Its orifice may be blocked with clot. (*b*) the aneurism may compress the innominate or subclavian distally, or (*c*) by the large size of the sac there may be great loss of force as compared with the left subclavian, should it not be also involved in the sac. A sphygmographic trace of the two pulses is often distinctive and helpful.

In the *Respiratory System* there is frequently pressure on the trachea or left bronchus, and the aneurism may weep, and eventually rupture, into the bronchus. Dyspnœa is common, either from this tracheal compression, or from pressure on the vagus, the pulmonary plexus, or the left recurrent laryngeal nerve. The stridor and typical brassy cough are generally very marked, and sometimes the dyspnœa becomes paroxysmal. The brassy cough is due to irritation of the recurrent laryngeal, or, according to Semon, to contracture following paralysis. Should a low type of pneumonia ensue, purulent foci develop in the damaged areas of lung, while small bronchiectatic dilatations may result. Tracheal tugging is a valuable physical sign. The patient is asked to hold back the head, and the cricoid is grasped by the fingers and pushed upwards: with each contraction of the ventricle the aneurism drags on the trachea, especially if it is adherent to it, or the tracheal tugging may be due merely to the sac pushing down the left bronchus which passes under the aortic arch.

In the *Alimentary System* pressure on the œsophagus may lead to dysphagia.

The *Nervous System* is specially important. Pressure on the vagus and recurrent laryngeal nerves has already been referred to. The sympathetic may also be compressed. It supplies the dilator pupillæ, and therefore, if the nerve is irritated, dilatation of the pupil will occur on the same side: if paralysis, then contraction of the pupil ensues. The left phrenic nerve is occasionally involved, leading to paralysis of the left half of the diaphragm.

Pain must be a prominent symptom, due to pressure on some of these important structures mentioned above. It may shoot down the left arm and have a definite anginous character.

3. *Aneurism of the descending part of the Arch.* – Here the aorta is deep-seated and approaches the spinal column. It is therefore less likely that any evidence of the aneurism will be present on physical examination, unless the vertebral bodies are eroded (4-6 Dorsal), and unless there are definite signs of the tumour in the back. In such a case there may be dulness on percussion in the left interscapular region.

This is an aneurism of symptoms, and pressure symptoms will be more or less marked. The left bronchus, the œsophagus, and possibly some of the nerves mentioned above, may be

affected, and in any case pain is often present. Probably the eroding of the vertebral bodies is responsible for a considerable part of this, and in a few cases, including one which we saw post mortem, the blood makes its way into the spinal canal, causing complete paralysis of all four limbs.

The table appended gives a good idea of how aneurism cases terminate. Experience, however, teaches that they all kill by pressure more frequently than by rupturing internally or externally, and probably many aneurisms which are never diagnosed prove fatal in this way.

TERMINATION OF ANEURISMS OF AORTIC ARCH[1]

Crisp's Table

This table refers to rupture of aortic aneurisms into structures named, unless where otherwise indicated.

98 Cases. *Ascending Part.*			48 Cases. *Transverse Part.*			21 Cases. *Descending Part.*	
Pericardium		30	Trachea		4	L. Pleura	4
Externally		6	Pericardium		2	R. Pleura	1
Left Pleura		1	R. or L. Pleura		4	Oesophagus	5
Fatal Pressure on Trachea and Bronchi		7	Bronchus		2	L. Bronchus	2
Hydropericardium and Hydrothorax		6	Oesophagus		2	Pressure on Trachea and Bronchi	2
Oesophagus		2	Pressed fatally on Trachea and Bronchi		12		
Vessels	Pul. Art.	3	Suffocation		3		
	Sup. Cava	3	Hydrothorax		1		
Ventricles	Right	2					
	Left	1					
Trachea		1					

Diagnosis.—Definite physical signs are an enormous help. In a *solid tumour* the pressure is constant, unvarying in degree, and the pulsation is not expansile. In an aneurism the pressure varies; it is more like a living, constantly changing pressure, and the tumour, if palpable, is expansile. It is sometimes possible by pressing firmly over the upper part of the sternum, when the patient lies on his back, to detect this expansile character of the aneurism.

Age, sex, and kind of work may help in the diagnosis, and an X-ray examination is of enormous advantage.

An *encysted pleural effusion* can hardly be mistaken, and a pulsating empyema has features, described on page 513, which should prevent any error.

[1] Table quoted from the late Sir T. Grainger Stewart's *Lectures on Medicine*.

Prognosis.—An aneurism may be cured. It becomes hard, and does not pulsate so vigorously, which means that clotting has occurred. But there is always a danger that fresh sacs may develop. In an advanced case, never place the duration of life at more than a few months. It must not be forgotten, however, that a fusiform dilatation of the aorta is compatible with a long and useful life.

Treatment. Try to aid clotting in the sac, which may be done—

1. By rest, keeping the patient in bed for weeks or months, and even insisting on the supine position being constantly maintained during that time.

2. By low diet and a minimum of fluid. Tufnell's method of treatment consists in 10 oz. of solids and 8 oz. of total liquids per day. Tufnell combined this starvation diet with rest in bed for 2 to 3 months.

3. By administering potassium iodide in doses of from 10 to 90 grains thrice daily. Iodism is often prevented by doubling the dose the moment it threatens to appear, but with certain patients a considerable interval is necessary before the drug can be again taken.

4. By various surgical measures which have been tried to induce clotting. These include—the introduction into the sac of fine iron or gold wire, or horse-hair; scratching the inner wall of the sac with a fine needle: electrolysis, either the positive pole alone, or both poles being introduced into the sac of the aneurism.

Recently gelatine has been used hypodermically. 100 c.c. of a 2 per cent solution is administered, and the solution must be very carefully sterilised. At least 15 to 20 injections are necessary. Tetanus has followed in some cases.

Bleeding has been employed where there is much pain and pulsation in the aneurism (taking 10-15 oz. of blood from the arm), but the method is not very frequently resorted to.

Under treatment by rest, low diet, and potassium iodide, very great improvement results, pain disappears or is much relieved, pulsation lessens, and the aneurism tends to become harder. A few surgical authorities commend the wiring of the sac, the gelatine method having been found disappointing. An innominate aneurism may be treated by

ligaturing the subclavian and common carotid, an operation which has proved successful in some cases.

A few *special symptoms* must be referred to.

Pain, if very severe, demands the use of opium: *thirst*, when the dry diet is in force, is relieved by sipping a solution of citric or other suitable acid in water; and *sleeplessness* may call for the administration of bromides or other hypnotic.

Where an aneurism is bulging through the skin, elastic pressure on the tumour should be tried, while painting on collodion over cotton wool may help to support the sac.

(b) Abdominal Aneurism

It is most commonly situated at the coeliac axis branch just below the diaphragm—more rarely lower down at, or involving, the superior mesenteric or one of the renal arteries. The aneurism may be saccular or fusiform. Pain is common, either dull or aching, or sharp and lancinating, and is often due to involvement of the spinal nerves. The aneurism may erode the vertebral bodies, and sometimes it grows to a large size. Vomiting is often troublesome, and may prove a misleading symptom to the physician.

The *Physical Signs* include very definite pulsation, which is expansile; there may be a thrill, and often a systolic murmur. These physical signs are not easily made out in aneurism of, or at, the coeliac axis artery. Sometimes it is possible, by comparing the pulse in the femorals with that in the radial arteries, to recognise a distinct retardation and diminished tension in the former as compared with the latter.

The expansile character of the pulsation in the aneurism serves to distinguish it from a solid tumour.

The aneurisms may rupture into the peritoneum, and sometimes into the stomach, and are seldom cured.

Treatment.—Combine absolute and prolonged rest with a low diet and potassium iodide. If the aneurism is low enough in position, proximal pressure might be tried: but it is severe treatment, and frequently causes shock, and as the pressure must be kept up for hours, gangrene of the legs may result, while the pain is usually excruciating. Opium is generally requisite to soothe the patient during the application.

XIV. DISEASES OF THE VEINS

Varicose veins belong to the department of surgery, but it is necessary to refer briefly to phlebitis.

Phlebitis, or inflammation of the walls of a vein, may be of primary origin, and is believed to be more common in rheumatic or gouty patients. It is not infrequent in anaemia, and may be a very undesirable complication in one of the continued fevers. Very commonly it is secondary to pressure on the veins in the pelvis, or pressure on the vein above the level of the phlebitis, but an injury to the vein, such as a blow, is an important exciting factor in many cases, and such injuries are more apt to occur in connection with varicose veins. The vein wall is found to be painful, and generally thrombosis has occurred or is in process of occurring. The veins most frequently affected are the femoral or popliteal. The risk of a portion of clot separating from the thrombus and finding its way to the lung should ever be kept before the mind.

The **Prognosis** is favourable in most cases, unless the thrombus becomes purulent, when it forms an abscess in a most undesirable position.

Treatment.—Absolute rest is imperative, with the application of sedative lotions such as lead and opium. The limb should be supported, and if possible the affected part kept at a higher level than the patient's head. Bandages are of value, but in many cases the removal of the thrombosed and varicose veins is the wisest procedure. Should an abscess form, it must be opened in the usual way.

DISEASES OF THE RESPIRATORY SYSTEM

I. DISEASES OF THE NOSE

1. Coryza: Acute Nasal Catarrh

Etiology. — There is a hereditary predisposition in some families, while lifelong coddling usually results in numerous attacks of nasal catarrh. The exciting causes are exposure to cold, wet feet, irritating vapours, and, in many cases, infection from other sufferers.

Pathological Anatomy.—A number of specific organisms produce acute or chronic coryza. These include the Micrococcus catarrhalis, the Micrococcus paratetragenus, the Bacillus of Friedländer, the Bacillus septus, and the Bacillus of influenza, and many other organisms—staphylococci, streptococci, and so forth—are secondarily associated with them. There are hyperaemia and swelling of mucous membranes, with changes in the secretory glands, causing them first to cease secreting, and later to secrete a copious amount of clear mucus, which later still becomes muco-purulent. As a sequel, there may be a hypersensitive condition of the mucosa, with increased tendency to fresh attacks, and the catarrh may spread to the pharynx and gastro-intestinal tract, or to the larynx and bronchi.

Clinical Features.—A cold generally begins with sneezing, then dryness, loss of sense of smell, uneasiness and irritation in the nostrils, followed by a copious mucous, and eventually a muco-purulent secretion. The catarrh may spread to the pharynx, larynx, and conjunctivae. It may affect the frontal sinuses and Eustachian tubes, causing headache in the former case, and deafness in the latter, with possibly either suppuration from infection, or indrawing, and even rupture, of the tympanic membrane from exhaustion of the air in the middle ear. The

catarrh may spread to the bronchi (bronchitis) or to the alimentary tract. There is often some slight pyrexia and nearly always more or less general discomfort. Many patients have an associated herpes labialis coexistent with the attack of coryza.

The **Diagnosis** presents no difficulty. Occasionally a continued fever such as measles begins with coryza, while in cases really of *influenzal* origin, the aching bones and the exhaustion are typical of that disease.

Prognosis.—In acute cases, good: in chronic cases, or where there is hereditary predisposition, the condition may be obstinate.

The **Treatment** is frequently carried out without recourse to medical advice. If our help is sought, try to abort the attack by a hot bath or foot-bath, followed by a hot drink and, just before sleep, a dose of compound ipecacuanha powder (gr. 5-10). A saline purge in the morning completes the attempt at cure. Diaphoretics and diuretics are helpful, and locally adrenalin (1 part in 1000 of $\frac{1}{2}$ per cent chloretone), introduced into the nostrils on pledgets of wadding and sniffed up the nose, is of great value, while cocaine 5 to 10 per cent is sometimes sprayed or painted on. Inhaling carbolic acid, eucalyptus, or menthol is commended. Many people take tincture of camphor in drop doses on sugar.

The tendency to coryza may be largely averted by keeping up the general health at a high level, hardening the patient by cold sponging particularly applied to the chest, and by attention to clothing.

(2) Chronic Nasal Catarrh

This is simply a chronic form of the preceding, and it may require long and persistent local and general treatment to effect a cure. Often there is a *hypertrophic* swelling of the turbinated bones, tending to block the Eustachian tubes and so cause deafness, and, not infrequently, adenoids are associated, the patient becoming a mouth-breather, and snoring dreadfully when asleep. A second variety is the *atrophic*, which is often associated with a most offensive discharge (ozaena), and may be the sequel to hypertrophic catarrh, or may result from caries, syphilis, the presence of a foreign body, etc.

The **Treatment** consists in relieving the hyperaemia of the hypertrophic form either by the electric cautery, or by applying astringents (alum or silver nitrate solutions). In ozaena the nasal douche must be resorted to, and boracic acid, listerine, protargol, or other antiseptic lotion used regularly.

Ozaena belongs more properly to the domain of the surgeon. In it there is a peculiarly offensive discharge from the nose, and the sense of smell is lost. *Membranous Rhinitis* may be diphtheritic or due to staphylococcal infection, and is sometimes, though rarely, the result of the application of the cautery to the nasal mucosa.

(3) Adenoids

An increase of the lymphoid tissue in the roof and on the posterior wall of the naso-pharynx. In 50 per cent of cases it is associated with enlarged tonsils. The function of the adenoid tissue is to supply leucocytes to protect against organisms which may be inspired. In this way tubercle bacilli may attack the tonsils and adenoid tissue, and eventually involve the glands of the neck. It is also generally acknowledged that there is an increased risk of infection from exanthemata in cases of well-marked adenoids. Adenoid tissue consists of lobulated masses made up of a connective tissue network with many lymphoid cells and covered over with ciliated epithelium.

Etiology.—Adenoids constitute one of the diseases of early childhood, beginning usually about the 10th month and becoming well-marked by the 4th or 5th year. At puberty there is a tendency for the lymphoid tissue to retrogress, but by that time the patient's health may have suffered irreparable damage. There is a hereditary predisposition to adenoids, and a family history of tubercle is not uncommon. Adenoids are more common in cold damp climates, and are associated with repeated attacks of coryza.

Clinical Features. The facies is very suggestive—the pinched nose, the absence of movement of the alae nasi, the broad nasal bridge, the obliteration of the naso-labial folds, the tendency to mouth-breathing, and the vacant stupid appearance of the patient, partly due to deafness, are all characteristic. The child snores at night, and his speech is peculiarly modified. He tends to say *B* for *M*, and *D* for *N*, and not

infrequently stutters. There is a history of repeated attacks of nasal catarrh.

On examining the thorax, one notes the narrow chest, with a tendency to pigeon-breast. There is sometimes collapse of the lungs. Asthma is a direct result of adenoids, and the child with adenoids is restless at night, often suffering from night-terrors. Pallor, headache, and giddiness are frequently complained of, and even epilepsy seems to have originated from this disease.

In any doubtful case a thorough examination of the posterior nares should be made with the finger.

The **Diagnosis** is generally easy. In *syphilis* the nose is dry and fissures are present round both mouth and nose. *Polypi* generally present no diagnostic difficulty.

The **Prognosis** is good if the adenoids are removed by operation before permanent damage has been produced.

The **Treatment** consists in removing the adenoids and thereafter confining the patient to the house for the first four days, during which all food and drink consumed by the patient should be sterilised.

A thorough course of pulmonary gymnastics is the best after-treatment, and helps to obviate the reappearance of the adenoid growths.

4) Diseases of the Sinuses Associated with the Nose

The antrum frequently becomes inflamed as the result of a decaying tooth, the root of which may penetrate into its cavity. An abscess may thus form, readily recognised by the pain and the discomfort produced by the pus sometimes discharging by the nose when the head is held down, and also by the darkness of the affected side when a suitable electric lamp is introduced into the mouth, the patient being examined in a dark room. In such a case the empyema or abscess of the antrum demands treatment by drainage, and for this purpose the offending tooth should be extracted and a tube inserted, connecting the antrum with the mouth: by this tube the cavity may be washed out and thoroughly drained.

The frontal sinuses are sometimes affected as the result of an ordinary coryza, and this probably depends on the ease with which the infective organisms can pass upwards through

the channel of communication with the nose. It is rarely necessary to resort to any special treatment other than that recommended for the relief of coryza.

5) Epistaxis, or Bleeding at the Nose.

This is a symptom of many morbid conditions. It may be the result of some local lesion such as occurs from trauma, simple polypi, malignant tumours, local ulceration, or a varicose condition of the smaller vessels in the nostrils; or it may be one of the clinical features in a blood disease such as pernicious anaemia, chlorosis, leucocythaemia, or haemophilia. It is also a common feature in the early stages of certain of the continued fevers in children, and especially scarlet fever, measles, and typhoid. Vicarious menstruation sometimes causes epistaxis, and it is unquestionably a phenomenon met with in cases of marked backward pressure, whether from heart or lung disease, though of less frequent occurrence than we might expect. In certain families there is a tendency to nose-bleeding during adolescence, any slight strain apparently inducing a severe haemorrhage. Towards old age, or where the vessels are degenerated, there may also be alarming and frequent attacks of epistaxis. These are all the more alarming because at any time a cerebral vessel may be the one to give way. In a rarefied atmosphere epistaxis is common, and it adds much to the difficulties attending the ascent of very high mountains.

It is sufficient, as a general rule, to make the patient lie down on the floor or on a couch, to extend the arms above the head, and to apply something cold to the back of the neck. An ice-bag certainly acts most effectually. It is sometimes necessary to prevent the blood from flowing down the throat by turning the head towards the bleeding side so as to permit of the escape of the blood from the nostril. Plugging the nostrils with cotton wool soaked in a solution of suprarenal gland, such as adrenalin (1 in 1000), and also, where necessary, plugging the posterior nares, causes speedy arrest of the haemorrhage. In milder cases pressure applied to the bridge of the nose or application of cold to the same region may be sufficient. Remember that after plugging the nares, blood is apt to undergo decomposition changes, and that it is wise therefore to wash out the nasal cavity with an antiseptic

solution, and sometimes to use for the purpose of plugging, iodoform or other antiseptic gauze in preference to aseptic wool. Nasal plugs should never be left in position for over forty-eight hours. In many cases, the condition of the patient's blood requires vigorous treatment, by means of iron, arsenic, or other remedies.

(6) Nasal Polypi

These may be mucous or fibrous; the former are readily removed by means of the wire snare or even a pair of forceps. Fibrous polypi and malignant growths belong to the realm of surgery.

(7) Hay Fever

Etiology.—There are three factors, all of which may be present in a case of hay fever. These are: (1) A neurotic constitution, often hereditary; (2) A nasal abnormality consisting in a hypertrophic rhinitis, or at the least a great hypersensitiveness of the nasal mucosa; and (3) Hay pollen, to a less extent the pollen of flowers, or, in persons with a special idiosyncrasy, the dust from the horse, cat, or other animal.

There is a powerful albuminous poison in the pollen, and especially in that of some twenty-five different varieties of grasses. This has been isolated by Dunbar, and an antitoxin obtained from animals to whom the poison has been administered hypodermically.

Pathological Anatomy.—There is sometimes hypertrophic rhinitis.

Clinical Features.—The attack, usually associated with the season when hay seed is ripe, begins with a severe coryza, generally with tremendous fits of sneezing, the eyes stream, and the conjunctivae may become inflamed. Sometimes the frontal sinuses are affected and severe headache results, sometimes the bronchi participate and bronchitic asthma ensues, and indeed forms a distinct variety of hay fever. The condition lasts throughout the hay season; once established, it is hard to get rid of it, and it is apt to recur annually.

The **Diagnosis** requires no remark.

The **Prognosis** depends on the possibility of avoiding the cause, and on the success attending the attempt to cure the

hypersensitiveness and to strengthen the patient's general health.

Treatment.—Send the patient to the sea-side, or, best of all, on a sea voyage during the hay season; sometimes a high altitude is beneficial. For the attack, cocaine in 10 to 20 per cent solution, or adrenalin should be painted on, or introduced on pledgets of cotton wool into the nostrils. An attempt should be made to lessen the hypersensitiveness by the use of astringents, or by the galvano-cautery, by which many small vessels are obliterated, thus reducing the hyperaemia. Dunbar's antitoxin is beneficial in some cases, and is applied locally to the affected mucous membranes. Lastly, attend to the patient's general health, administering such tonics as arsenic, phosphorus, and strychnine.

II. DISEASES OF THE LARYNX

(1) Acute Laryngitis

An acute catarrhal inflammation of the larynx.

Etiology.—Exposure to cold, and especially a sudden change from overheated to cold air, excessive use of the voice, irritating vapours, entrance of irritating substances and fluids, and propagation of neighbouring inflammation (*e.g.* secondary to severe coryza or bronchial catarrh).

There is a hereditary predisposition to coryza, pharyngitis, and laryngitis: the coddling of children is apt to increase any such susceptibility.

Pathological Anatomy.—There is reddening of the larynx, specially of the posterior ends of the true cords, false cords, and inter-arytenoid fold, with increased secretion of mucus or muco-pus. Sometimes superficial ulcers form. It may go on to oedema under certain conditions.

Clinical Features.—These include dryness of throat, hoarseness, tickling cough, which is later often spasmodic, and occasionally pain, but only, as a rule, on swallowing, when the epiglottis may cause the discomfort. There is generally no fever. In children, inspiration is apt to be croupy, and laryngismus stridulus may develop.

Diagnosis. Little difficulty should be experienced in

diagnosing this condition from *laryngeal diphtheria*. The recognition of the organism and the presence of albumin in the urine ought to point to diphtheria.

Prognosis.—Generally recovery occurs, but the condition may become chronic; if oedema ensues it may cause death by asphyxia.

Treatment.—Rest to the voice absolutely, and keep the patient in one atmosphere, and if necessary, in bed. A diuretic and diaphoretic mixture may be given with a purge in the morning. Warm drinks, such sedatives as antimonial and ipecacuanha wine, are beneficial for the cough, and, above all, the inhalation of steam, with or without some agent added. Compound tincture of benzoin, the succus conii, and creosote are excellent for this purpose. Ammonium chloride is often inhaled with benefit. The wet pack is an effective remedy when used at bedtime. It consists of a piece of wet lint or a wet handkerchief wound round the throat, and covered with a piece of indiarubber tissue or oiled silk, and on the top a flannel bandage. In children, apomorphine gr. $\frac{1}{15}$-$\frac{1}{10}$ may be given hypodermically with the object of inducing sickness and so clearing the larynx of secretion.

An application of cocaine 10 per cent solution may abort the attack, and is worth a trial.

2 Oedema of the Glottis

There are two kinds of oedema of the glotti. A *inflammatory*, and B *non-inflammatory or dropsical*.

A. *Inflammatory Oedema* is the result of "septic" laryngitis, which may be of erysipelatous nature. The epiglottis, ary-epiglottic folds, and false cords become enormously swollen, and the oedema may be, although rarely, subglottic in position.

The **Clinical Features** are: the feeling as if a foreign body was in the throat, and alarming dyspnoea threatening complete asphyxia in addition to the phenomena of acute laryngitis; naturally pain is often a very pronounced symptom.

The **Diagnosis** can readily be made with the finger, by means of which the swollen oedematous epiglottis may be easily palpated.

The **Prognosis** should be guarded, and must depend on

immediate success of treatment. A great danger is the supervention of spasm.

The **Treatment** should be prompt and energetic. Scarify the epiglottis with a curved, sharp-pointed bistoury, the blade of which should be guarded with adhesive plaster: if this fail to afford relief, tracheotomy or intubation must be resorted to. During the early stages try to arrest the oedema by the use of ice to suck and prevent spasm by the inhalation of moist air and the exhibition of some drug such as the bromides, and give iron and quinine internally.

Oedema may sometimes develop in tubercular and syphilitic laryngitis giving rise to similar symptoms, and in such cases it calls for the same treatment.

B. *Non-Inflammatory Oedema* may occasionally occur in Bright's disease, sometimes in heart disease, and more rarely from other causes. It may require surgical treatment.

(3) Chronic Laryngitis

A chronic catarrhal inflammation of the larynx.

Etiology.—It may be the sequel to an acute attack, but is more often due to excessive use of the voice (hawkers, clergymen, and singers). It may result from cold, and together with the pharynx, from alcohol or tobacco in excess, and especially from cigarette smoking.

Pathological Anatomy.—Swelling and thickening of mucous and submucous coats of the true and false cords, the inter-arytenoid fold, and the epiglottis, frequently with superficial ulceration and a granular appearance of the true cords.

Clinical Features.—Similar to the acute form, but more hoarseness, dryness, and cough: it is often associated with pharyngitis.

Diagnosis.—Care should be taken to exclude *tubercular disease of the larynx*.

Prognosis. Recovery under favourable conditions, it is sometimes permanent; oedema rarely follows it.

The **Treatment** includes absolute rest for the voice, with a change of air, and especially departure from cold, damp fogs to the clear air of, for example, the Riviera, Egypt, or elsewhere. Sometimes the sea-side is beneficial, and sometimes a high altitude is preferable. Avoid any irritant which may help

to keep up the condition, and especially spirituous liquors, highly spiced foods, and tobacco. Occasionally it is found that our patients are mouth-breathers, some nasal affection preventing the air from entering and being duly heated in its passage through the nostrils. In this case attention should be directed to the nose. Various pigments may be applied to the larynx, such as tannic acid in 4 to 10 grains to the ounce solution, zinc sulphate 1 to 5 grains to the ounce, and in very obstinate cases, silver nitrate 10 to 50 grains to the ounce of distilled water. It is well to interpolate a word of caution about the use of silver nitrate, which in strong solutions always induces a marked reaction. Steam impregnated with oil of pine, turpentine, eucalyptus, and similar remedies may be inhaled with benefit.

(4) Laryngismus Stridulus; Spasmodic Croup. Child-Crowing.

Spasmodic contraction of the adductors of the glottis, often with carpo-pedal spasms, and in all probability due to the reflex irritation of the adductor centre in the brain.

Etiology.—Common in rachitic and weakly children, but is directly excited by irritation of the stomach, by dentition, and similar causes.

Clinical Features.—May commence with crowing inspiration, generally nocturnal, followed by cessation of breathing, early pallor and later cyanosis of face, and bending back of the head. The eyes stare wildly, and beads of perspiration may be seen on the forehead. The spasm yields with a loud crowing inspiration. There may be carpo-pedal contractions, while occasionally death results before free respiration is restored.

Prognosis.—Generally favourable.

Treatment.—(1) For the relief of an attack nothing is so helpful as a hot bath, or even a sponge wrung out of hot water placed over the upper part of the chest. In other cases cold water may be dashed on the face and chest and smelling salts held to the nose. Chloroform anæsthesia has been employed in very severe cases. Where all these measures fail tracheotomy may be performed. 2 Remove the exciting cause of the attack, whether it be stomach disorder or dental

irritation. (3) Give constitutional remedies such as cod-liver oil, syrup of the iodide of iron, and so forth.

(5) Phthisis Laryngea

In pulmonary tuberculosis from 13 to 97 per cent of patients have an affection of the larynx. The discrepancy between these figures is due to the fact that in the great majority of cases the laryngeal changes are of a trivial nature, and the authorities who accept the smaller percentage do not take such cases into consideration. Anaemia of the larynx and a catarrhal laryngitis, somewhat resistant to treatment, are common in pulmonary phthisis, and they make up the difference. The tubercular affections to which the term phthisis laryngea properly belongs are ulceration and infiltration, and sometimes a resultant perichondritis. Very rarely is the tubercular involvement primary: it is generally secondary to infection of the lungs. Laryngeal phthisis is most common between the ages of 20 and 40, and is rare in children.

Pathological Anatomy. Tubercles appear in mucous and submucous tissues, with surrounding inflammation, and they lead to ulceration. The sites of the disease are the inter-arytenoid fold, the false and true cords, the arytenoids, the ary-epiglottic folds, and the laryngeal surface of the epiglottis.

The **Clinical Features** closely resemble those of chronic laryngitis, with hoarseness of voice and short cough, but the special symptom is pain on swallowing. Aphonia may result from temporary paralysis, from fixation of an arytenoid or a cord, and, in advanced cases, from destructive ulceration. Where there is much oedema of the glottis, the dyspnoea is extreme.

The **Diagnosis** is usually fairly easy.

Tubercular Affections of the Larynx.	*Syphilitic Affections of the Larynx.*
1. Evidence of pulmonary tuberculosis.	Other evidences of syphilis and history of infection.
2. Pain.	Little pain.
3. Greyish white colour, especially of ulcers.	Bright red colour.
4. Breaks down later.	Breaks down earlier.
5. Laryngeal surface of the epiglottis affected.	Edge and lingual surface of the epiglottis affected.

The **Prognosis** is unfavourable in really marked cases, and the duration of life is often limited to six months; sometimes it extends to three years. The short duration of life is due to the fact that the lungs are often seriously involved before the larynx suffers, and also that tubercular ulceration heals badly, and relapses are apt to occur.

Treatment.—Pain on swallowing may be relieved by giving ice to suck, before administering food while liquids and semi-solids are more easily swallowed than solids. In very severe cases, a 10 per cent solution of cocaine should be painted on, or a quarter of a grain of morphia is sufflated in the form of a powder mixed with starch.

Try to secure healing of the ulcers. Some authorities recommend lactic acid applied in a strength of 20 to 80 per cent, others prefer the cautery but both methods are unsatisfactory. Antiseptics, such as carbolic acid, iodoform, guaiacol, or menthol in olive oil and similar remedies, may be applied locally.

In cases which are not too far advanced, pure, dry air affords the greatest comfort; either the high altitudes of the Swiss mountains or the warm sunny winter climate of Egypt or Algiers. Caution the patient about over-use of the voice, and recommend abstinence from stimulants and irritants so far as possible.

6. Syphilitic Disease of the Larynx

A catarrhal laryngitis is commonly present during the secondary stage but ulceration is generally a later manifestation. These ulcers may be superficial but if secondary to gummata are apt to be deep and to leave marked cicatrices when they heal. Gummata may occur on ary-epiglottidean folds, false and true cords, epiglottis, but are rarely subglottic in position.

Perichondritis sometimes results from ulceration.

Clinical Features.—There is loss of aphonia sometimes dysphagia and much less pain than in tubercular disease. Cicatrisation may follow and cause great trouble by interference with the glottis.

Diagnosis. See Phthisis Laryngea.

The **Prognosis** is good if treated but tertiary syphilis

not infrequently causes the formation of membranes and stenosis.

Treatment.—As serious syphilitic affections of the larynx are really of tertiary nature, iodide of potash is the best remedy, although mercury may also be administered. Where cicatrisation occurs, it may be found necessary to dilate with a laryngeal bougie, or even to perform tracheotomy.

III. DISEASES OF THE BRONCHIAL TUBES

1. Acute Bronchitis

An acute catarrhal inflammation of the mucous membrane of the bronchial tubes, which may involve either the large or the small tubes; in the latter case the condition is called capillary bronchitis.

Etiology.—One common *predisposing* cause is delicacy, especially in the young or the old, and perhaps chiefly in young persons the subjects of rickets. Gout, Bright's disease, heart disease, and many of the continued fevers may also be termed predisposing conditions, although in such fevers as measles and whooping-cough a bronchitis, due to the local action of the specific organism is a leading clinical feature of the fever, which might therefore be termed an exciting cause.

The *exciting* causes are cold and damp, irritating vapours, foreign bodies, and inflammations of the larynx and nose. Bronchitis frequently results from coryza, and is by no means an uncommon part of an attack of influenza.

Both pneumococci and streptococci may be found in the secretion of a bronchitis, and many other organisms such as the influenza bacillus. It is therefore sometimes an epidemic disease, and vaccines of the organism responsible for the bronchitis have been used with success in some cases.

Pathological Anatomy. There is catarrhal inflammation of the mucous membrane of the trachea and bronchi, and in the case of the finer tubes of the bronchioles. This is associated with swelling and congestion of the mucosa, with, at first, diminution in the secretion of mucus, and later the pouring out of a thin, and sometimes muco-purulent, sputum which is readily spat up by the patient.

Where the finer tubes are involved, small portions of lung collapse, and broncho-pneumonia frequently results.

On microscopic examination, in all forms of bronchitis there is found distension of vessels in the inner fibrous layer of the bronchial tube, the basement membrane becomes oedematous and swollen, and there is desquamation of cells with the formation of new ones prematurely by Debove's cells. There is also an infiltration of the surrounding tissues by leucocytes. The destruction of the ciliated epithelium of the bronchi greatly interferes with the natural action of the cilia.

Affecting the Large Tubes, or Ordinary Acute Bronchitis

Clinical Features. There is more or less pyrexia, with a temperature of from 100 to 102° F., coming on after exposure to cold or wet. During the *first stage* there is cough of an ineffective nature, because no mucous secretion is being poured out, and therefore there is nothing to be expectorated. There is severe pain behind the sternum, and a varying degree of general malaise and discomfort, and frequently pain in the back and legs.

During the *second stage* the patient coughs severely, but brings up only a small quantity of scanty, and very viscid, mucus; after an expectoration there may be temporary freedom from cough. During both these stages there is apt to be a considerable degree of spasm, generally more marked at night, and causing dyspnœa and orthopnœa in a severe case.

The *Physical Signs* of the two stages closely correspond. The breathing becomes harsh vesicular with expiration prolonged, and the accompaniments consist of rhonchi, generally sonorous, sometimes squeaking or sibilant. There may be some fremitus due to the rhonchi, and the percussion note is either unaltered, or is hyperresonant and suggestive of emphysema.

The *third stage* of bronchitis commences when the secretion becomes less viscid and often somewhat purulent; it is typically frothy, but comes up with great ease, and the cough is not so severe, and generally ceases temporarily after a fair amount of sputum has been got rid of. Rarely is the sputum tinged with blood, although not infrequently there may be particles of carbon intermixed with it. The only alteration

in the physical signs during this stage is the addition of bubbling, moist râles, especially heard during inspiration and sometimes expiration.

In the other systems there is little to be noted: the loss of appetite and the frequency of constipation are common phenomena in any febrile affection, and there may be a scanty secretion of urine, precipitating a copious deposit of urates on standing.

A result of repeated attacks of bronchitis is the development of emphysema, with marked backward pressure affecting the right side of the heart, and with a varying degree of dyspnœa resulting from it. Enlargement of the right heart may be recognised by epigastric pulsation, increase of cardiac dulness to the right of the sternum, accentuation of the second pulmonary sound, distension of the veins in the neck, and in certain cases the development of a tricuspid systolic murmur. The bases of the lungs should be examined for signs of œdema, in which case fine crepitations are heard. The duration of an attack depends much on the personal equation of the patient: but an ordinary attack of bronchitis usually subsides in a matter of ten days to a fortnight.

The **Diagnosis** is easy, although it may be difficult to say what the cause of the bronchitis is: and it is well to remember that a case of *measles*, *whooping-cough*, or *typhoid fever* may closely resemble a severe attack of bronchitis. In most cases tubercular lung disease is associated with bronchitis, in fact every case of phthisis pulmonalis at all active has necessarily some bronchial irritation present with it, and it may be a very prominent symptom. The physical signs of simple bronchitis are definite, and should prevent confusion with cases in which consolidation is present.

Prognosis.—At the extremes of life bronchitis is always serious, and also in patients who are debilitated, or in whom the condition of the right heart or the degree of pre-existing emphysema affords reason for anxiety. It is invariably of grave import when œdema of the bases of the lungs develops in a case of bronchitis, and if the inflammation spreads to the smaller tubes, the patient runs the additional risk of catarrhal pneumonia.

The **Complications** of bronchitis are emphysema, bronchorrhœa, bronchiectasis, cardiac failure from strain especially

upon the right heart, and, lastly, the risk of broncho-pneumonia where the small tubes become involved.

Treatment.—The *first stage* of the disease should be treated by sedatives, and perhaps most effectively by the use of steam inhalations and the external application of linseed poultices, fly-blisters, or mustard. Sometimes an early case of bronchitis may be aborted by giving Dover's powder (gr. 5-10), and following up with a saline purge next morning. A good deal may be achieved by the administration of such remedies as antimonial wine (♏ 5-15), and ipecacuanha wine (♏ 15-40); while a purgative, together with a fever mixture, such as the spirit of nitrous ether, in drachm doses, affords the greatest and most prompt relief. The patient should certainly be kept in bed until the temperature has fallen to normal.

During the *second stage*, when the secretion is viscid, much may be done to render it more easily expectorated, and sodium bicarbonate (gr. 5-15), and other alkalies, as well as small doses of the emetic group, such as apomorphine, are very serviceable, and are aided greatly in their action if poulticing or external counter-irritation be still kept up. A useful external rubefacient is the linimentum terebinthinae aceticum, either applied alone or with an equal part of the linimentum saponis. Towards the end of this stage a stimulating expectorant, such as ammonium carbonate (gr. 5-10), together with squill and senega, is often prescribed.

During the *third stage* an attempt should be made to lessen the secretion, and cough mixtures containing the mineral acids are specially useful for this purpose.

In many patients spasm predominates, and paroxysmal fits of coughing may cause great distress. The use of a tent over the bed into which steam is conducted, medicated with tincture of benzoin, terebene, creosote, or carbolic acid, is of great comfort to the sufferer, and apomorphine given by the mouth or hypodermically, although it induces sickness, may relax the spasm and clear the bronchial tubes. Spirit of chloroform and tincture of hyoscyamus are also useful antispasmodics.

Spirit of chloroform, spirit of ether, and aromatic spirit of ammonia, together with alcohol in the form of whisky or brandy, are frequently necessary to support the patient's

strength, and attention should be paid to the diet of the invalid during an attack.

For children a hot bath is an admirable method of commencing treatment, and where expectoration becomes difficult, an emetic, such as repeated large doses of ipecacuanha wine, clears the tubes at the same time as it empties the stomach of its contents.

Capillary Bronchitis, or Bronchitis affecting the Small Tubes

This form is common in childhood, and is generally secondary to fevers, and especially measles and whooping-cough. In adults, it is not infrequently due to the inhalation of irritating vapours, and is the precursor of septic pneumonia, which may arise from the inhalation of food particles, etc., in cases where the glottis cannot be closed, or in cases of cancer of the vocal cords. In patients suffering from excessive weakness, or where there is stricture of the bronchial tubes, often malignant in origin the resultant septic pneumonia begins with a capillary bronchitis.

Clinical Features.—Should the capillary bronchitis produce, as it frequently does, a secondary catarrhal pneumonia, the reader is referred for a description of the latter condition to p. 461; but, so far as capillary bronchitis itself is concerned, there is invariably greater pyrexia than in bronchitis of the large tubes (104 to 105 or 106° F.). There is also more intense dyspnœa, more pain on coughing, and more rapid exhaustion: the pulse-rate may vary from 120 to 150, the respirations from 30 to 50, or even more, and the rapidity is always greater in very young patients. The struggle for breath probably induces patches of the lung to become collapsed, and it has been stated that small areas of collapse form the stage between capillary bronchitis and catarrhal pneumonia, the patches themselves undergoing catarrhal inflammation. The proximity of the inflammation to the air vesicles greatly increases the patient's distress and dyspnœa, and there is always a grave depression of vital powers. Orthopnœa is invariably present where the child is old enough and sufficiently conscious; the extraordinary muscles of respiration and the alæ nasi act with great vigour, while the lower interspaces tend to be sucked in with each inspiration.

Physical Signs. On *Palpation* rhonchial fremitus, and sometimes fremitus due to moist râles, may be felt. The *Percussion* note varies considerably; it is dull, if consolidation develops or collapse ensues over any areas of involved lung. *Auscultation* may reveal little definite change as regards the breath murmurs, excepting that they are weakened and sometimes absent, and the latter phenomenon may depend upon collapse or blocking of bronchial tubes. The accompaniments, however, are distinctive, numerous sibilant rhonchi and fine, moist râles being present over the greater part of the chest; and in old persons, in cases where the vital functions are becoming depressed, and where there is septic pneumonia setting in, these moist râles are very evident over the bases of the lungs.

The cough is often harassing, and may occur in severe paroxysms. The complications and sequelæ have already been noted; they are broncho-pneumonia, sometimes septic in nature, and collapse of the lung.

The **Diagnosis** is generally easy; but it may be difficult, except in the case of children, to recognise the cause of the condition, and an effort should invariably be made to eliminate —(1) a possible neoplasm involving one or other bronchus; 2) a laryngeal affection either paralytic or malignant; and 3) by investigation of the history the possibility of the inhalation of an irritant gas or of acrid fumes. The question of the failure of vital powers being responsible for the condition may also be considered, because debilitated alcoholic subjects are specially prone to capillary bronchitis of septic type.

The **Prognosis** is always serious where the small tubes are involved, although it may depend partly on the age and strength of the patient. Extensive collapse and a definite catarrhal pneumonia increase the risks. The following clinical features are suggestive of grave danger: (1) Where, in the case of a child, the dyspnœa is so great as to prevent crying and feeding, and where coughing, previously in evidence, has entirely ceased. (2) A great rise of temperature, or on the other hand a fall to 99° F., or even to normal unaccompanied by improvement in the other features, generally precedes a fatal result. 3. When the child or adult becomes more markedly cyanosed, with a sudden and great increase in the

dyspnœa, it implies, either that the heart is failing or that collapse has occurred; and (4) in children, the violent throwing about of the arms invariably portends an early fatal result.

The **Treatment** consists in endeavouring to support the patient's strength and to prevent pulmonary collapse occurring. It is exactly similar to that given for catarrhal pneumonia, but it should be remembered that the use of oxygen inhalations, the early and free exhibition of stimulants, alcoholic and other, and in children the induction of vomiting so as to clear the tubes, are of peculiar value in this type of bronchitis, as distinct from the involvement of the larger tubes.

Suffocative Catarrh of Adults, as the name implies, is a type of capillary bronchitis not infrequently found in old people.

Clinical Features. It comes on with great suddenness, often killing the patient within 46 to 60 hours, or it may occasionally be protracted to ten days.

The clinical features are striking, marked cyanosis, great distension of the chest which progressively increases until no further movement appears to be possible, and the breathing is practically confined to the diaphragm. The life of the patient depends on the heart holding out. The sputum is frothy and gradually diminishes in amount as the heart fails. The temperature is rarely very high, while the pulse increases in rate as it falls in strength, and may eventually reach 130 to 150. The respirations are rapid, often being 40 to 50 to the minute. The feet and hands swell in a protracted case and albuminuria may be present.

The **Prognosis** is very grave, and the **Treatment** consists in oxygen and steam inhalations. A preliminary dose of calomel with a saline purgative to follow is of advantage, and a large jacket poultice should be applied as soon as possible to the chest. Alcohol and cardiac tonics are of great value in keeping up the strength, and spirit of chloroform (♏ 20-40) is not merely a stimulant but tends to relieve spasm. Sometimes compression of the chest by the hands placed over the bases of the lungs aids the patient's efforts at expiration. Bleeding from the external jugular vein (10 to 15 ozs.) is necessary in cases where there is great cyanosis and engorgement of the right heart.

No cough mixture is so helpful to the patient as one containing a considerable quantity of an alkali, and even antimonial wine might be ordered in certain cases in which the strength is sufficiently maintained to warrant its administration.

2. Chronic Bronchitis

A chronic inflammation of bronchial mucous membrane.

Etiology. The *predisposing* factors are old age, gout and rheumatism, alcoholism, Bright's disease, heart disease, certain cases of aortic aneurism, and chronic phthisis.

The *exciting* causes are cold and wet, as well as some of the above.

Pathological Anatomy.—The bronchial mucosa is thickened, granular, and infiltrated, and ulceration is not infrequent. In other cases the mucous membrane becomes thin, and there is invariably a tendency for the muscular coat, as well as the fibrous layers of the bronchial wall, to yield to pressure, causing dilatation of the tubes, while the constant attacks of the disease lead to emphysema, generally bilateral, and involving sometimes the whole lung, although often more particularly the anterior margins. The glandular structures in connection with the bronchial tubes are frequently atrophied.

Clinical Features.—Typical attacks of chronic bronchitis may be seen in old persons during cold weather, and are frequently called "winter cough." The appearance of the chest is that described under emphysema, and many of these patients have a typical barrel-shaped chest, while we get evidence of backward pressure due to the emphysema, causing dilatation and hypertrophy of the right heart, and, when compensation fails, dropsy of the extremities and all the other phenomena which we associate with loss of compensation of the right heart.

The amount of cough depends on the severity of the attack of chronic bronchitis, while the degree of dyspnœa, in part due to the condition of the right heart and the emphysema present, varies also with the extent of the bronchial tubes involved and the quantity of secretion present in them. There is usually an element of spasm: some patients suffer from severe spasms of coughing and wheezing during the night, others have their worst attacks in the morning upon wakening,

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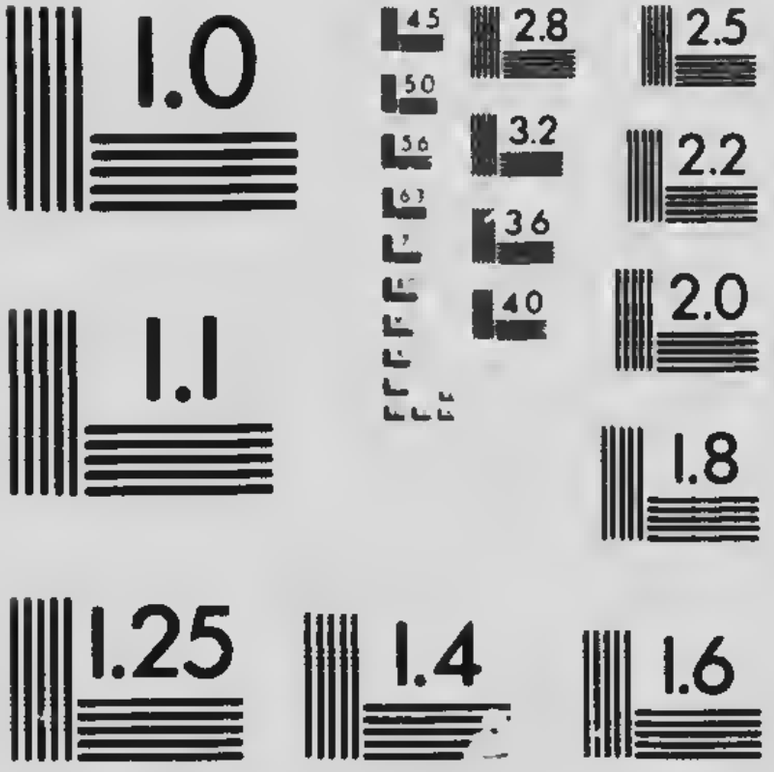

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when the tubes are full of secretion, and are only emptied after a severe bout of coughing.

Physical Signs.—On *Inspection*, as already noted, the chest is typically emphysematous, and there may be evidence of backward pressure, such as the distended veins in the neck and the smaller venules seen over the chest wall. The extraordinary muscles of respiration are generally called into action during a severe attack. On *Palpation* the vocal fremitus is diminished, while fremitus due to rhonchi and moist râles is sometimes present, rhonchi being in evidence during the earlier, and moist râles during the later stages of the attack. The *Percussion* note is hyperresonant. On *Auscultation* the breath sounds are harsh vesicular, with expiration prolonged, the degree of prolongation depending on the element of spasm, which is always expiratory in type. The accompaniments during the early stages are sonorous rhonchi, and during the later stages bubbling, medium, and large moist râles, which are heard sometimes over the larger tubes alone and sometimes over the whole lung. In older and weaker persons moist râles of a finer character are heard at the bases, and indicate a degree of oedema of the lungs. The vocal resonance is slightly diminished, or may be unchanged.

The other physical signs depend on the condition of the right heart and the presence or absence of tricuspid incompetence, with distension of the veins in the neck. Evidence of enlargement of the liver and spleen may be noted. Lastly, it should be mentioned that, as a result of extensive emphysema, the diaphragm is apt to be displaced downwards.

The **Complications** of chronic bronchitis, other than emphysema and enlargement of the right heart, depend on changes in the bronchial walls, which lead to bronchiectatic dilatations. Changes may also occur in the secretion contained in the bronchi, by which that secretion becomes excessive or fetid, or even gangrenous.

In *Bronchorrhœa* the sputum is excessive. It may be serous or mucous in character, and associated with marked oedema of the lungs (due to cardiac failure), the patient spitting 2 or 3 pints a day: or it may be very purulent, when it tends to become fetid, and is generally associated with bronchiectatic dilatations. Fetid sputum implies infection with organisms, and it is easy to understand how ulceration

may result in cavities containing such sputum, and how gangrene may ensue in the debilitated or alcoholic patient.

There is a mild form of chronic bronchitis in children, which may result in the same definite and permanent changes in the lungs and tubes as are associated with the disease in adults. They are, in reality, mild attacks of more acute bronchitis, from which the child may eventually recover when puberty is reached.

In older patients, there is sometimes a special form of chronic bronchitis in which no secretion occurs at all. This has been called *dry catarrh*, and it is invariably very resistent to treatment and is accompanied by marked emphysema.

Fibrinous or *plastic bronchitis* will be described separately.

The **Diagnosis** requires no reference, except the observation that it is wise to investigate the possibility of a tubercular element being present in cases which resist treatment.

The **Prognosis** depends much on the strength of the patient and the possibility of sending him to a warm climate during the winter weather. In every case, before an opinion is expressed, the condition of the right heart should receive special attention.

Treatment.—Of more importance than any drug is the question of climate. Bronchitic patients should be sent to warm, equable, and dry resorts, Egypt and the Riviera being almost ideal during good winters. It is not judicious to send patients so affected to a high altitude unless the heart can stand the strain. Many bronchitic patients derive the greatest benefit from drinking a glass of warm milk and water, or a cup of weak tea, immediately on waking, because by this means the usual fit of morning coughing is rendered more easy.

The following drugs appear to be of special value:—potassium iodide in 10 to 15 grain doses, ammonium carbonate in 5 to 10 grain doses, and sometimes ammonium chloride, while liquor arsenicalis (m 5), continued for a considerable time, is often beneficial. Where the sputum is slightly fetid, terebene or guaiacol should be given in capsules (m 15-30). With the same object inhalations of antiseptics may be used, either vaporising and inhaling carbolic acid, creosote, or guaiacol, or having in the sick-room and beside the patient's bed a

porcelain capsule, in which some volatile antiseptic, such as one of those just mentioned, may be slowly vaporised.

Much good may follow external counter-irritation, and the linimentum terebinthinae aceticum, equal parts of turpentine and olive oil, and many other rubefacients, are of great value. Where backward pressure is excessive, and the patient capable of standing it, the question of bleeding may be considered, and, lastly, the treatment given for emphysema is often applicable in cases of chronic bronchitis.

(3) Fibrinous or Plastic Bronchitis

An acute or subacute form of bronchitis, in which fibrinous casts of the bronchial tubes occur, and by their presence cause violent paroxysms of coughing.

Etiology.—Casts of the bronchi form in diphtheria, should the membrane extend down into the bronchi. Membranous casts sometimes develop in pneumonia and phthisis. It has been stated that after the inhalation of ammonia, alcohol and eau de Cologne, after the local application of lactic acid, and in some exceptional patients after the exhibition of potassium iodide, fibrinous casts may be formed in the course of a bronchitis which has otherwise nothing very peculiar in connection with it.

Clinical Features. — Paroxysmal attacks of coughing occur, after which the patient expectorates one or more of these casts generally with much mucus and blood; the casts may be formed in the larger or smaller tubes: they may, therefore, be of large or of small size, and usually are branched. They are hollow casts made of fibrin or mucin, and contain in addition many leucocytes, alveolar epithelial cells, frequently Charcot-Leyden crystals, and sometimes show the characteristics of Curschmann's spirals. There is great dyspnoea associated with the disease, with a tendency to inspiratory retraction of the chest, which, however, is relieved after the separation and expectoration of the casts.

The **Diagnosis** depends on the recognition of the casts in the sputum.

The **Prognosis** is bad in diphtheritic cases, and in any case this type of bronchitis is a tedious and severe one.

Treatment.—The casts are soluble in lime-water, and

Dixon has suggested the use of this in an atomiser; a solution of papain, which is said to have similar solvent properties, may also be tried. Emetics are not infrequently of value, and sometimes inhalations of ether have been advised. The disease is a rare one, and therefore the treatment must depend a good deal on the individual case.

(4) Bronchiectasis; Dilatation of Bronchial Tubes

It may be general or saccular, and it is due either to weakening of the bronchial wall, traction upon it, or increased intrabronchial pressure.

Etiology.—1. One of the common causes of bronchiectasis is weakening of the bronchial wall as the result of chronic inflammation, a condition frequently occurring in bronchitis. The muscular coat and the elastic tissue in the bronchial wall both yield, and associated with this weakening due to long-continued inflammation, there is also the important factor of intrathoracic pressure to be taken into account. Cough implies the closure of the glottis and sudden and powerful contraction of the expiratory muscles: therefore in cases where the bronchial wall is weakened, such pressure, if frequently brought into play, will result in dilatation of the tubes as a whole, or more likely of those parts where the resistance is weakest. In addition, however, to weakening of the wall and increased intrathoracic pressure there is a third factor present in certain cases, namely, the accumulation of secretion, which by its retention will tend to produce or increase local dilatations.

It is easy to understand how interference, such as might be caused by stricture of a bronchus from tumour, gumma, or other condition, will result in time in localised dilatation of the distal bronchial tubes, because it is largely the result of retained secretion.

2. Traction upon the bronchial walls is exercised by thickened septa in the lung, and especially where this condition attains a marked degree. In fibroid lung, and particularly that type associated with chronic tuberculosis, such traction may produce local dilatations, and it seems probable that pleuritic adhesions will aid in the process, because they give a definite fixed point from which traction may be exerted.

3. There is a rare congenital form of bronchiectasis, due to universal weakening of the bronchial walls, generally limited to one lung. It is probable that in still more exceptional cases such general dilatation of tubes may be met with in the adult, but it must imply an embryonic error in the structure of the bronchi.

Pathological Anatomy.—Saccular dilatations are certainly more common: they have smooth walls, unless ulcerative changes from retained secretion or tubercular processes are present. The muscular coat of the bronchus is atrophied, the elastic tissue weakened, and the fibres widely separated from one another. The shape of the cavity depends much upon the traction present. In some cases there may be saccular dilatations throughout the greater part of one lung, or part of a lung; in other cases one or two saccular dilatations may alone be present, associated with tubercular or other fibroid changes, and these are very common near the apices of the upper lobes. Surrounding the dilatations, there is generally some increase of fibrous tissue, but the amount depends largely on the nature of the case.

The **Clinical Features** vary according to whether the cavities are saccular, or whether the dilatation is more universal. There is generally the expectoration of large quantities of sputum, clearing the bronchiectatic cavities, and thus relief from the cough is obtained. These fits of coughing may be invariably looked for in the morning, when the patient awakes. The sputum, $\frac{1}{2}$ to 2 pints a day, tends to be fetid in odour and when placed in a glass vessel separates into three layers, of which that on the top is brownish, frothy, and muco-purulent, the middle layer is a clear, mucus-looking fluid, and in the foot of the glass there is a thick deposit, granular and yellowish in colour. This deposit is largely made up of pus cells, of fatty crystals, bacteria, and often elastic tissue of the lung where there is ulceration. Hæmorrhage is an occasional phenomenon in this disease. Dyspnœa varies, depending on the amount of interference with the lungs.

The *cylindrical or general* form of bronchiectasis closely resembles chronic bronchitis as regards the physical signs and clinical features, while in the *saccular* form, the physical signs are those of a cavity, and include the cracked-pot note on percussion, cavernous or amphoric breath murmurs, consonating

moist râles, and whispering pectoriloquy. There are very often evidences of fibroid changes in the lung, such as local retraction of the chest wall and the displacement of neighbouring organs, so commonly the result of such changes.

There is frequently clubbing of finger-tips and marked dyspnoea on exertion, in cases of bronchiectasis.

Complications.—There are four important complications or sequelae:—(1) the development of gangrene, (2) the not infrequent onset of severe haemorrhage, (3) the occasional association of rheumatoid joint pains with the disease, and (4) in rare cases, the metastatic cerebral abscess or abscesses which are secondary to the pulmonary condition. Further it is necessary to remark that fibroid lung throws considerable strain on the right heart.

Diagnosis.—In many cases the condition is merely suggested by the clinical features and physical signs, and accurate diagnosis is impossible. In most cases of fibroid lung saccular cavities are met with, and it is difficult to discriminate between the vomicae of chronic tuberculosis and bronchiectatic dilatations. In non-tubercular cases of bronchiectasis the cavities are not so likely to be situated near the apices of the upper lobes, and are more probably to be found at the base or at least in the lower part of the lung. The physical signs may suggest emphysema in some cases. A careful search for the tubercle bacillus in the sputum, and one or other tuberculin test should be carried out. In *empyema* which has opened into the lung the physical signs should prevent a mistake being made in most instances.

Prognosis.—Recovery is rare, but the condition is compatible with considerable prolongation of life. There is always the risk of septic absorption, and especially so where the contents of the cavities undergo decomposition changes.

The **Treatment** is not satisfactory. We may try to render the contents of the cavities a little less septic, but this is by no means easy, and it is the residual sputum which the patient does not cough up which is most septic. Probably the best intratracheal injection consists of guaiacol 2 parts, menthol 10 parts, in 88 parts of olive oil, and a drachm of this solution may be injected twice daily. It is impossible to ensure that this or any other injection should reach the affected part of the lung, but it is very difficult to adopt any other method.

Postural coughing is very advantageous. The patient should be made to hang his head over the bed during each bout of coughing, and so aid in the evacuation of the sputum. Direct injections of solutions of iodoform into the cavity through the chest wall are both painful and not very desirable, because a septic needle track on the removal of the syringe may give rise to much trouble. Inhalations of creosote, guaiacol, turpentine, thymol, and carbolic acid may be used in many different ways, both volatilised and by spray, but care should be taken that the patient does not suffer from toxic doses of the drug used. Chaplin's method of creosote inhalations is severe, but it aids the patient to bring up the residual sputum. The creosote is poured on a sand bath and vaporised. A small wooden shed may be used, if one is available, and as a rule twenty minutes to one hour represent the limits of endurance. The eyes require to be protected by watch-glasses from the irritating fumes, and the clothes and hair of the patient should also be covered so as to obviate the retention of the strong odour of the drug.

Every effort should be made to support the general strength, and to endeavour by patient medical treatment eventually to diminish the excessive secretion in the bronchiectatic dilatations.

The surgical treatment of bronchiectatic cavities is eminently unsatisfactory, unless adhesions have occurred between the layers of pleurae, and even then the risk is very great.

(5) Asthma

Recurrent attacks of expiratory dyspnoea, often extreme, and generally nocturnal.

Etiology.—It is undoubtedly hereditary, and especially common in neurotic families. It may follow bronchitis or whooping-cough in those who have a constitutional predisposition; gout and possibly syphilis are responsible for some cases. It occurs at all ages, more frequently in males.

The exciting causes of an attack are many—peripheral irritants, hay pollen, nasal polypi, adenoids, dyspepsia, mental irritation, fright and many other conditions of a varied nature.

Pathology and Pathological Anatomy.—That asthma is due to spasm of bronchial tubes is beyond question, but there

is much difference of opinion as to how this is brought about, and whether this is the sole lesion. There is frequently also catarrh of the mucosa associated with the condition constituting a sort of exudative bronchiolitis. Charcot-Leyden crystals and Curschmann's spirals are not limited to the sputum of asthmatic patients, and they may have no causal connection. A vasomotor paresis is a favourite theory, but there is nothing to prove its existence.

The anatomical changes present are those met with in bronchitis, or are mostly sequelae such as emphysema; while a marked eosinophilia usually present in the blood, does not offer any solution to the problem of causation.

Clinical Features. There are often premonitory phenomena, such as mental irritability, and then the attack develops. The patient awakes at 1 or 2 A.M. with a sense of great oppression and huge efforts at inspiration, while expiration is prolonged, wheezing, and difficult. He may be ashen-grey at first, but soon becomes cyanosed, and beads of sweat appear on his face, which wears an anxious expression. The spasm yields after a period of time, and during the attack the patient usually coughs up some viscid sputum containing the peculiar casts of small bronchial tubes called Curschmann's spirals. The spirals are only present for the first few days, and then disappear from the sputum. The attack generally recurs the following night, while during the day the patient may be fairly comfortable.

Physical Signs.—The chest is fixed at nearly full inspiration, and the inspiratory effort demands the aid of the extraordinary muscles of inspiration; the appearance of the chest closely resembles the type in emphysema, with the lower interspaces widely separated and indrawn on inspiration, while the chest as a whole is hoisted up rather than expanded. The shoulders are rounded and stoop, and there may be actual spinal curvat On *Palpation* there is usually rhonchial fremitus. C *Percussion* a hyperresonant note is obtained, while it is n d that the superficial cardiac dulness has disappeared, an the absolute liver dulness is much diminished. On *Auscultation* there is harsh vesicular breathing with great prolongation of the expiratory murmur, and the accompaniments are sibilant, rarely sonorous, rhonchi, and at later stages there may be moist râles. There is no pyrexia as a rule.

Diagnosis. — *Cardiac* and *renal* asthma are types of dyspnœa which have kindred characteristics, but they do not give the complete clinical picture of ordinary bronchial asthma, while in each case the recognition of cardiac or renal disease should help to solve the difficulties of diagnosis.

The **Prognosis** is good in children, in whom the disease may be arrested at, or by, puberty, but in adults the resultant changes in lungs and heart—emphysema and enlargement of right heart—render the outlook less favourable.

The **Treatment** of the attack consists in antispasmodics. Nitrite of amyl, in 3 minim capsules, broken in the handkerchief and inhaled, or nitroglycerine in doses of $\frac{1}{100}$th to $\frac{1}{75}$th grain; inhaling the vapours from burning stramonium, datula tatula, and nitre paper; and in certain cases with great caution chloroform anæsthesia, or morphia (gr. $\frac{1}{8}$-$\frac{1}{4}$) with atropine (gr. $\frac{1}{120}$-$\frac{1}{80}$), are all excellent remedies. Chloral, bromides, and many other sedatives and hypnotics may also be administered.

Much can be done to obviate the attacks by trying to remove any of the known exciting causes, such as dyspepsia, hay pollen, and so forth, and every case demands a careful dietary with a sufficiency, but no excess, of easily digested food, and the regulation of the meal hours, so that no heavy meal is taken towards bedtime. Warm clothing should be recommended, and, if possible, the patient should be advised to live, during the cold and damp months of the year, in a mild, dry climate. Inhaling compressed air is often beneficial, and big doses of potassium iodide are serviceable in some cases.

The neurotic element when present should not be forgotten, and suitable tonics should be prescribed.

IV. PULMONARY EMPHYSEMA

THE pathological term for abnormal dilatation of pulmonary alveoli and air vesicles.

The theories and the common varieties of emphysema form an excellent introduction to a study of the subject.

The *Theories* are three in number:—

(1) Primary weakness or acquired degeneration of the air-vesicle walls.

(2) An inspiratory theory of production:—the air not getting out, and each fresh inspiration distending the air vesicles still more (Gairdner).

(3) An expiratory theory:—cough and excessive expiratory effort certainly produce emphysema.

Probably combination of (3) and (1) explains most cases of emphysema.

There are three *Varieties* of emphysema:—

(1) A general type, often known as the large-lung type of emphysema.

(2) An atrophic or small-lung type, the result of atrophy, and met with in old age.

(3) Compensatory emphysema—where emphysema occurs in the same lung, or perhaps in the opposite lung, as the result of local interference with part of a lung.

Etiology.—*Age.*—It may occur at any age—the atrophic form in old persons. *Sex.*—It is commoner in men. *Trades.* It is met with in persons who blow wind instruments which tax the expiratory powers, such as the cornet, or in glass-blowing, and in all occupations which tend to induce bronchitis. *Disease.*—Bronchitis and whooping-cough unquestionably cause emphysema.

Whether there is a hereditary tendency is doubtful: certainly it seems to run in certain families. Possibly the inherent weakness may be in the elastic tissue of the lung.

Pathological Anatomy.—The lung affected is typically dry and bloodless; the air vesicles run together, with atrophy of the walls and obliteration of the capillaries. The anterior margins and apices of the lungs, the parts of the lung least well supported by the chest wall, suffer most, but compensatory emphysema may occur in any part of the lung. There is often considerable atheroma in the larger branches of the pulmonary artery. Large bullae sometimes develop under the pleura, and there may be interstitial emphysema in the lung substance. The pathological results of emphysema include enlargement of the right heart, with evidence of chronic venous congestion in the liver, the kidneys, and the venous circulation generally. Pneumothorax may result from rupture of one of the bullae referred to.

The **Clinical Features** of type (1), or *the usual Emphysema*. There is dyspnoea, especially on exertion, stooping, and even,

in marked cases, produced by talking. It may be so severe that the patient has to sit up in bed orthopnœa. Cough depending on bronchitis is often present. There is more or less cyanosis, and the veins in the neck may be engorged.

Physical Signs.—Inspection. The chest is very characteristic. It is barrel-shaped, with considerable increase in the antero-posterior diameter, the chest being fixed in the position of nearly full inspiration. The epigastric angle is abnormally obtuse the lower interspaces widened, and the upper narrowed. The shoulders stoop, there is often actual spinal curvature the supraclavicular fossæ are deep, and the neck appears short with well-developed sterno-mastoid, scalene, and the other muscles of extraordinary inspiration. The sternum is unduly prominent, and on inspiration there is little or no expansion but the chest is hoisted up as a whole, while the diaphragm contracts vigorously. There is a deep furrow marking the usual upper transverse abdominal line. Frequently venous radicles are seen on the chest, and especially along the line of insertion of the diaphragm. There is marked bulging in the supraclavicular regions on coughing.

Palpation reveals that the vocal fremitus is diminished.

On *Percussion* the note is hyperresonant; there is usually no superficial cardiac dulness, and the lungs may reach to the lowest limits of the pleural sacs, greatly diminishing the absolute liver dulness on the right side. The heart, and especially the liver, are displaced downwards. *Auscultation* reveals feeble breathing, unless bronchitis is present, with prolongation of the expiratory murmur. The vocal resonance is diminished, and in the absence of bronchitis or œdema there are no accompaniments.

The apex beat of the heart is not visible, and often not palpable. There may be epigastric pulsation due to a dilated and hypertrophied right ventricle, and the pulmonary 2nd sound is loudly accentuated.

Very rarely serious haemoptysis occurs, and death is generally due to cardiac or pulmonary complications.

(2) In the *Senile* or *Small-Lung Type* of emphysema, the chest is small, the sternum thrown forward, and the apices flattened. The note on percussion is hyperresonant, and the absolute cardiac dulness may or may not be diminished. There is little or no displacement of heart and liver.

Diagnosis.—It is almost impossible to mistake a well-marked case of general emphysema, while compensatory emphysema, being a common result of any interference with an area of lung, hardly demands much reference. The apices and anterior margins should always be examined in cases where emphysema is suspected.

The **Prognosis** of the general form is unsatisfactory. It is usually progressive, and if so, leads to ever-increasing interference with the circulation.

Treatment.—Prevent the condition from becoming worse. Treat the bronchitis, induce the patient to seek a warm, dry climate in winter, and keep up the general health. Stop any effort which throws great strain on the air vesicles, in other words, prevent forced respiratory efforts. Tonics, such as iron, strychnine, and especially arsenic, are of value; try also to relieve the backward pressure by diuretics and purgatives. Much benefit may be obtained by placing the patient in a pneumatic chamber, where he breathes condensed air, usually at a pressure of $\frac{1}{2}$ to $1\frac{1}{2}$ atmospheres over the ordinary. The treatment requires three hours. During the first hour the air is being gradually blown into the chamber, he then sits for 1 to 2 hours in the condensed air at full pressure, and lastly, for half an hour or so the air is slowly allowed to escape. This treatment causes deeper, fuller, and slower respirations after its use for some three weeks, and the emphysema is distinctly reduced in not a few cases. Expiring into rarefied air has also been commended, but is difficult to carry out. Pulmonary gymnastics, which help in expressing air out of the chest during the act of expiration, are certainly of value.

V. HAEMOPTYSIS

Pulmonary Haemorrhage

Haemoptysis is a sign of many pulmonary and other diseases, rather than a separate disease in itself.

Etiology.—Haemorrhage may occur from the nose or from the trachea, or one of the big bronchi, but it is haemorrhage from the lung with which we have specially to deal.

(1) It is certainly most commonly the result of phthisis, in connection with which disease it is described elsewhere.

(2) It is a result of pulmonary congestion, and is seen in acute lobar pneumonia and in lobular pneumonia, although in these conditions the amount of haemorrhage is not, as a rule, great.

(3) Haemoptysis may be a sequel to pulmonary abscesses of all kinds, and specially where gangrene is present, and also to pulmonary ulceration, such as occurs in stone-mason's lung. It is an important clinical feature in certain cases of cancer of the lung.

(4) Haemorrhage is one of the results of pulmonary infarction; the amount of blood expectorated varies greatly, and generally continues for some days.

(5) In the rare instances of the involvement of the bronchial tubes by the Distomum Westermannii, considerable haemorrhage may occur.

(6) Rupture of an aortic aneurism may take place into the lung or into a bronchus, and cause death from fatal haemorrhage.

(7) In a group of cases where, probably, there is little acute congestion of the lung, haemoptysis may occur: these include severe strain, as from lifting a heavy weight, and the haemoptysis sometimes associated with the commencement of a continued fever; it is also the result of backward pressure, as in common cirrhosis of the liver, and cases of backward pressure with engorgement of the pulmonary circulation.

(8) In certain blood diseases, such as purpura haemorrhagica, anaemia, haemophilia, and scorbutus, haemorrhage may take place.

(9) Injury to the chest frequently causes haemoptysis.

Vicarious menstruation is a rare cause of haemoptysis: and in a few cases a trivial haemorrhage has occurred, in elderly persons, probably from the rupture of pulmonary vessels whose walls have become degenerated.

Pathological Anatomy.— Blood in the lung undergoes the usual decomposition changes, and, therefore, if a haemorrhage has taken place, the patient will go on for some time expectorating sputum containing blood which has become altered in this way; the rusty sputum in pneumonia is a good example of this.

In tubercular disease of the lung haemorrhage often super-

venes, not only from the rupture of a vessel in a cavity, but from small miliary aneurisms formed in tubercular cavities. In tubercular cases a haemorrhage is often followed by a rapid spread of the disease.

Clinical Features.—The bleeding may be excessive or slight; it may be long continued, or there may be only one considerable attack of haemorrhage, lasting a comparatively short time.

A peculiar saltish taste in the mouth, sometimes with, sometimes without, cough, is generally the first indication to the patient that blood has covered the mouth, while a feeling of tightness, or even of pain, referred to the chest, is very common. The blood is bright and frothy, very different from the dark, partially digested blood which comes from the stomach. Clots may be coughed up, and the amount of blood depends on the cause of the haemorrhage. There is generally, in a severe haemorrhage, great palpitation and a feeling of faintness, the extremities become cold and clammy, and beads of perspiration appear on the forehead.

Very frequently careful examination of the chest with the stethoscope enables the physician to recognise the site of the haemorrhage, but the greatest care should be taken to avoid moving the patient, and so risking a renewal of the haemoptysis. The breathing over the area of haemorrhage may be somewhat bronchial in character, but the distinctive feature is the presence of fine or medium moist râles.

Diagnosis.—Blood from the *stomach* is generally dark and partially digested, and gives an acid reaction, while blood from the lung is bright red and alkaline. It must be remembered, however, that in haemorrhage from the lung some of the blood may be swallowed and then vomited. There is sometimes a feeling of pain or tightness over the chest, suggesting that the lung is the source of the haemorrhage, whilst the bright, frothy blood is typical. Bleeding from the *nose* is generally easily differentiated.

Prognosis. Whether the haemorrhage is severe or slight, it is prudent to assure the patient of the fact that he is in no immediate danger, because death rarely occurs as an immediate result of haemoptysis. It is hardly necessary to add, however, that where an aneurism of the arch of the aorta has ruptured, or where a miliary aneurism in the lung in a case of phthisis

has given way, a fatal result is by no means uncommon, and it may ensue with startling suddenness.

Treatment.—Where the haemorrhage is slight, it is only necessary to confine the patient to bed, to remove the pillows, to loosen any garments round the neck, and to enjoin absolute repose for a considerable period of time. An ice-bag applied over the chest, and, if possible, over the site of the haemorrhage, is of great value, and small pieces of ice to suck, while they may not avail greatly, give the patient something to do—a point of much importance in relieving his anxiety.

A haemorrhage frequently ceases when syncope occurs, the pressure in the vessels being then greatly lowered, and to aid in this lowering of pressure, aconite and other remedies are sometimes recommended. It is certain that opium, given in the form of the liquor morphinae (♏ 10-20), not merely soothes an excited heart, but helps also to allay the patient's anxiety. Styptics of different kinds should be administered, such as turpentine, in 15 to 30 minim doses in capsules, mineral acids, and especially dilute sulphuric acid (♏ 10-15), tincture of the perchloride of iron (♏ 20-40), gallic acid (gr. 10), acetate of lead (gr. 1-2), tincture of hamamelis (♏ 30-60), and chloride of calcium in doses of 10 to 20 grains three or four times daily. Some authorities have suggested that where the haemorrhage is continuous, and the patient does not faint, an attempt might be made to reduce the pressure in the pulmonary circulation by bleeding from the arm. This method is certainly open to considerable objection.

For a few days after the haemorrhage has occurred the patient should be confined to bed, and it must be kept in mind that a marked degree of pyrexia may follow, especially in tubercular cases. The food must be as simple as possible, and no alcohol should be allowed. All strain, especially physical, must be interdicted, and the lungs should be overhauled from time to time, so as to find out whether there has been complete absorption of the effused blood, because there is always a risk that a haemorrhage may induce a rapid spread of pulmonary tuberculosis, should it be associated with that disease.

VI. ACUTE PULMONARY CONGESTION

An active congestion of the lung and a condition similar to the first stage of lobar pneumonia.

Etiology.—It may be caused by the inhalation of irritating vapours or very hot air, and it may result from excessive action of the heart as in fevers or overstrain. In a localised form it is associated with many lung diseases, such as pneumonia, phthisis, and so forth.

The **Pathological Anatomy** is simply active engorgement of the lung with blood, general or local.

Clinical Features.—There is a varying degree of pyrexia, 102° F. or more, sometimes with haemoptysis. The physical signs are rather negative than positive. There is defective expansion, feeble breath murmurs, and diminished resonance. Otherwise the condition shows the characters of the disease of which, as a rule, it forms a part.

VII. PASSIVE PULMONARY CONGESTION

This specially involves the lower lobes and there is usually some oedematous effusion into the air vesicles.

Two types demand a brief description:—

1. *Hypostatic Congestion* is the result of gravity plus debility. The affected lung is of a dark purple colour, and is engorged with blood and oedematous fluid, and may even sink in water. The dependent parts alone suffer.

Clinical Features.—There is local dulness varying in degree, with typical moist râles and generally feeble bronchial breathing. There is always dyspnoea, and a low type of inflammation may supervene, which rapidly kills the patient.

The **Prognosis** is grave, and the **Treatment** consists in stimulation both by internal remedies, such as alcohol, aromatic spirit of ammonia, and spirit of chloroform, and also locally by poulticing and even counter-irritation of a more vigorous nature. Try to avoid the onset of this condition by altering the patient's position in bed from time to time.

2. *Backward Passive Congestion* is the pathological condition called brown induration of the lungs, in which the

alveolar capillaries become distended and tortuous, and there is much coloration with blood pigment of the alveolar epithelial cells. There may be blood or blood pigment in the sputum.

This type follows many valvular lesions of the heart. There is usually a considerable amount of oedema present.

The **Clinical Features** vary greatly and depend on the amount of oedematous exudation into the air vesicles.

The **Diagnosis** may therefore be rather a question of inference in milder cases.

The **Prognosis** depends on the causal condition, and the **Treatment** is directed to the relief of the backward pressure by kidneys and bowels, possibly by cardiac tonics, and in some cases by direct blood-letting, when 15 to 30 oz. may be taken from the arm.

VIII. PULMONARY OEDEMA

This is present to a very limited extent in active or acute congestion, but it is common in passive pulmonary congestion, and there is a so-called *acute idiopathic variety*, such as occurs in Bright's disease, and after the administration of pilocarpine, to which reference will be made later.

In ordinary pulmonary oedema the lung is full of watery fluid which may be expressed post-mortem from the cut section, and is coughed up during life in large quantities by the patient. It is greatest in the dependent parts of the lungs.

The **Clinical Features** include those of the heart disease or other causal condition, but also very marked dyspnœa, with cough and watery sputum. There are two reliable physical signs, namely, the dull note, sometimes with a tympanitic note over the neighbouring lung, and the moist râles of small size, very freely heard on coughing.

The ultimate **Prognosis** is never or rarely favourable, and the **Treatment** consists in local counter-irritation, free internal stimulation with alcohol, ammonia, and similar remedies, and an attempt at alleviating the conditions to which the oedema is due. Propping the patient up in bed is of special value.

Acute idiopathic oedema, not an uncommon cause of death in Bright's disease, may come on very rapidly. The clinical

features correspond to backward-pressure oedema, only the cause of the backward pressure with its characteristic features is wanting.

IX. CATARRHAL PNEUMONIA

(Broncho-Pneumonia; Lobular Pneumonia)

A catarrhal inflammation of localised, generally multiple, portions of the lung immediately consequent on capillary bronchitis.

Etiology.—(1) There is a form of broncho-pneumonia which is a lobular variety of croupous pneumonia, and which appears to be rather more frequently met with in children when subject to the attack of the pneumococcus.

(2) A large proportion of cases of broncho-pneumonia are, however, secondary to a capillary bronchitis. Many of these originate in an attack of whooping-cough, measles, influenza, or some other continued fever in children, and certainly the death-rate in measles and whooping-cough is not so much due to the primary disease itself, as to the broncho-pneumonia to which it may give rise. It is specially common under the age of five, and most fatal in children who have not yet reached their third year. The incidence of the broncho-pneumonia may depend partly on the time of year and partly on the debility of the patient.

In adults a broncho-pneumonia, resulting from a capillary bronchitis, is often associated with Bright's disease or any condition in which the vitality of the patient has seriously suffered. In diphtheria at all ages the membrane may infect the smaller tubes, and so give rise to a broncho-pneumonia of diphtheritic origin.

(3) There is yet another type of broncho-pneumonia which may be divided into two groups:—(*a*) In one, where there is paralysis of laryngeal muscles, food may be inspired, and a deglutition pneumonia results. A similar state of things follows in certain cases where the patient is comatose, or where there is paralysis of the vagi nerves, or in any condition in which a septic pneumonia may be set up by the inspiration of fetid mucus or of food particles. Purulent discharge in connection with the ulcerations due to malignant disease, and, in certain cases

the filling of the lung with blood, may both cause a septic pneumonia.

(b) The other subdivision of this group of broncho-pneumonias is the form which is associated with excessive weakness, and which often is a condition immediately preceding death. In it the mucus is not got rid of, and tends to decompose and to act as an irritant. This mucus is drawn farther into the smaller tubes with each inspiration, and may there set up a condition of things akin to a septic pneumonia. The debility is greatly aided by alcoholic tendencies in the patient, and death generally occurs before there are many patches of lobular pneumonia actually in existence.

The tubercle bacillus most frequently attacks the lung so as to set up a tubercular broncho-pneumonia, but this specialised form of the disease is described separately under pulmonary phthisis.

Pathological Anatomy.—The inflammation begins in connection with the little bronchus leading to a lobule, and is often preceded by the collapse of the lobule in which the consolidation is about to occur. The central bronchus is filled with leucocytes and swollen, granular, epithelial cells; the bronchial wall is inflamed, and the surrounding air vesicles are filled with leucocytes, many large mono-nuclear cells probably derived from the alveolar epithelium, red blood corpuscles, and a proportionately small amount of fibrinous exudation. At the post-mortem these pneumonic patches stand out prominently as small projections of the lung, and the pleura covering the patches is covered by a fibrinous exudate. On section the patches are reddish-grey in colour, and the lobules in the neighbourhood are often collapsed. Minute haemorrhages may occur under the pleura, and the areas of broncho-pneumonia may be seen, in many different positions, in the lung. In children, where it is a sequel to measles or whooping-cough they may be widely scattered over both lungs. In septic cases they are often met with in the lower parts of the lung.

The patches frequently resolve, but they may lead to suppuration or even gangrene, or may undergo a fibroid change. In septic cases the affected bronchi contain little plugs of pus, and the small lobular areas of hepatisation are seen to be undergoing softening.

Bacteriology.—In addition to the Pneumococcus of Fränkel

and the Pneumo-bacillus of Friedländer, there are many organisms which may be found in broncho-pneumonia: these are specially the Streptococcus pyogenes, the Staphylococcus aureus and albus, sometimes the Diphtheria bacillus, and, as already indicated, the Tubercle bacillus.

Clinical Features.—*Type* 1.—This type is simply the lobular form of croupous pneumonia: it corresponds in physical signs and clinical features to lobar pneumonia with, in addition, the characteristics of type 2, about to be described.

Type 2.—In a child who has had whooping-cough or measles, broncho-pneumonia may set in with severe rigors, or the temperature may gradually rise higher and higher until it reaches 105 or 106 F., the dyspnœa becoming much greater.

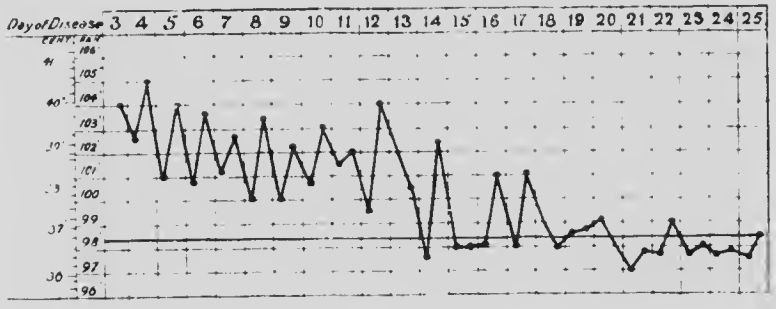

Fig. 27.—Temperature Chart. Broncho-pneumonia. Recovery.

the child markedly cyanosed, and the lower interspaces more and more sucked in. As broncho-pneumonia follows the antecedent capillary bronchitis, the child gradually becomes more seriously ill, and increase of the patient's distress usually indicates a rapid spread of the disease to a larger number of lobules of the lung. The pulse-rate may rise, in a child, to 150 or 200, and the respirations may reach 60 or 80 per minute; the face has a peculiarly anxious expression, and the cough, paroxysmal and exhausting, becomes less and less noticeable as the patient reaches the stage of stupor.

On examining the chest, we find evidences of small areas of consolidation which yield the phenomena already described under capillary bronchitis. As these areas are by no means always superficial, it may be difficult to elicit evidence of their presence by percussion. The breathing over the areas becomes tubular, and, what is even more typical, fine moist râles, sharp and clicking in character, are heard over the patches. The

phenomena are similar to what is noted in capillary bronchitis, but they are associated with a greater degree of distress. The condition may last for 1, 2, or 3 weeks, and then it will either terminate favourably or death may occur. The temperature falls in most cases by lysis; only in those patients in whom the broncho-pneumonia is the result of the pneumococcus or the pneumo-bacillus does the temperature sometimes fall by crisis.

Type 3. In septic broncho-pneumonia, where due to the inspiration of food, pressure on the bronchus, or other cause, the areas of consolidation are found in the lower parts of the lung. The dyspnœa is usually less extreme, the patient is in most cases an adult, and the disease may follow a less acute course. There may be increasing evidences of septic absorption, but very frequently the condition is rather suspected than actually diagnosed during life.

There are obviously cases of broncho-pneumonia which do not correspond closely to any of the types just mentioned, but sufficient has been said to make the leading clinical features of the condition recognisable.

A leucocytosis is usual, excepting in post-influenzal cases where it is often absent.

Diagnosis.—A careful examination should be made of the sputum so as to search for the presence of the pneumococcus, and it should be remembered that in children who are the subjects of a pneumococcal pneumonia, the distribution of the disease is apt to be lobular rather than lobar. The close association between measles, whooping-cough, and influenza and catarrhal pneumonia is often of great help in diagnosis, and a capillary bronchitis, from which the little patient does not show signs of recovering, generally implies the onset of catarrhal pneumonia. In such a case the greatly increased dyspnœa and cyanosis, with the indrawing of the lower interspaces, should make the physician extremely suspicious.

Typhoid fever may, at its commencement, show symptoms rather suggestive of a capillary bronchitis than of a typical case of the fever. Test doubtful cases for the Widal reaction, though it should be remembered that at least a week must elapse after typhoid fever has commenced, before the reaction may prove positive.

Many cases of *acute broncho-pneumonic tuberculosis* closely

resemble broncho-pneumonia, but it seems hardly necessary to remind the reader that the discovery of the tubercle bacillus in the sputum is the one certain method of diagnosis. In *miliary tuberculosis* of the lungs there are usually no lobular areas of dulness, and the tubular breathing generally heard over such patches does not occur. In addition, the moist râles in miliary tuberculosis are not, as a rule, clicking in character. Unfortunately, it is extremely common to find miliary tuberculosis associated with broncho-pneumonic tuberculosis, but in such a case the tubercle bacillus will sooner or later be obtainable in the sputum.

Tubercular meningitis sometimes closely resembles broncho-pneumonia in its general features, but the rigidity of the neck with the drawing back of the head, the involvement of cranial nerves, and the presence of Kernig's sign (p. 654) render the diagnosis of basal meningitis fairly clear. Lumbar puncture may also aid the physician, as the cerebro-spinal fluid should contain an excess of leucocytes and an increased proportion of albumin.

Prognosis.—The death-rate from broncho-pneumonia is very high in children, but much depends on the constitution and age of the patient: probably over 50 per cent of patients under the age of three terminate fatally. In the lobular form of pneumococcal pneumonia, the prognosis is favourable. It is hardly necessary to add that septic pneumonias of all kinds are very apt to prove fatal, and that where an irritant substance of any kind has been inhaled, the death-rate must be high.

Treatment.—The utmost care should be taken of children suffering from the fevers to which capillary bronchitis and broncho-pneumonia are common sequelæ. The child's strength should be carefully maintained, and every care taken to avoid chill and fatigue during convalescence from the fever in question. When it is evident that capillary bronchitis has set in, the main object of the physician should be, *First*, to give the patient moist air to breathe at a fairly warm temperature, and this is best done by covering in the top of the bed or crib with a "tent," into which a bronchitis kettle discharges steam; *Secondly*, endeavour to prevent the bronchial tubes from being filled up with mucus, by encouraging coughing, and, when necessary, giving an emetic to clear the tubes.

Much benefit results from altering the position of the child in bed, keeping the head high, and from time to time lifting the little patient, so that he may get change of position together with some stimulation; *Thirdly*, alkalies should be administered for the purpose of diminishing the viscidity of the sputum, and stimulants, such as spirit of chloroform, aromatic spirit of ammonia, and spirit of ether, will also be found valuable. Alcohol has an important place among the necessary remedies. The local application of stimulating liniments, such as camphorated oil, or an occasional linseed poultice, is excellent, and will often help in enabling the patient to clear the bronchi of secretion. A jacket poultice is good practice, but requires the greatest care in its application, because it readily exhausts the patient's strength, and must not, therefore, be used too frequently. The application of ice, or the ice-water jacket, may be useful in cases where the child's strength is well maintained, but it is doubtful whether such treatment is prudent as a general rule; *Fourthly*, perhaps there is no disease, with the exception of lobar pneumonia, in which oxygen inhalations prove such a valuable aid to the physician at a critical time: *Fifthly*, and finally, remember to keep a constant and careful watch on the temperature; it is wise to instruct those in attendance that the temperature should be kept *below* 103° F., and that, whenever it tends to rise higher, cold sponging, or the use of the cold bath, or other similar means for lowering the temperature, should be promptly resorted to. The number of cases of broncho-pneumonia which are lost because of the neglect of this precaution is very considerable.

It seems hardly necessary to refer in detail to the diet: it should be of the simplest, the child being fed with milk and beef-tea or egg-flip every 2 or 3 hours; and it is wise to procure free, but not excessive, action of the bowels, to help the kidneys in the discharge of their functions, and to endeavour to ensure for the patient as much sleep during the night as possible. Probably one of the first and best signs that the disease is yielding to treatment is the appearance of moisture on the skin coincident with a steady, but gradual, fall in temperature. For a long time after an attack of broncho-pneumonia the child's health and general well-being demand careful consideration.

X. PULMONARY COLLAPSE

Collapse of portions of the lung may occur either from the pressure of pleural effusions, from occlusion of trachea or a bronchus, or from some difficulty in the way of the air entering part of the lung (commonly found in the lower parts of the lung).

The **Etiology** of pleural effusions is described under the appropriate heading. Bronchial occlusion may result from the presence of a diphtheritic membrane, excessive bronchial secretion, especially in children, or the pressure of a tumour upon the bronchi. The form of collapse associated with the lower parts of the lung is a frequent result of excessive debility, and generally occurs shortly before death. Paralysis of the diaphragm may cause collapse, and physical conditions such as ascites and abdominal tumours may exert pressure from below. In cases of great cardiac enlargement collapse of the lower part of the left lung is by no means uncommon.

Pathological Anatomy.—The portion of lung collapsed is dry and airless; it is somewhat tough when cut with a knife, and is of a bluish-grey colour.

Clinical Features.—Collapse of lung may occur anywhere, but the lower and posterior portions of the lower lobes, the middle lobe of the right lung and the anterior margins are not infrequently affected. Where a large area of lung collapses, dyspnœa results, and may be associated with great cyanosis, which tends to increase until the death of the patient. The changes depend largely on the amount of lung affected. Local flattening and immobility of the affected part of the chest are typical, the percussion note is dull although the degree of dulness varies with the amount of lung involved, the breath murmurs are absent or greatly enfeebled, and the vocal resonance is much diminished. In certain cases it should be remembered that the collapsed lung may conduct breath murmurs better than normally, so that distinct bronchial breathing, with bronchophony, may be sometimes noted.[1]

[1] This depends on the bronchus remaining patent. Collapse with tubular breathing and bronchophony is not uncommon near the left base in cases where an enlarged heart or distended pericardial sac compresses the lung.

Diagnosis.—The great increase of dyspnœa, together with the immobility of the affected part of the chest, and the physical signs just mentioned, are diagnostic of collapse, but on the other hand, the collapse noted after death in debilitated patients, may give rise to little definite clinical phenomena.

The **Prognosis** is grave: it is only in cases where small areas of collapse are associated with broncho-pneumonia that a favourable result may be anticipated. Extensive areas of collapse occurring in young children in connection with capillary bronchitis or broncho-pneumonia are of very grave import.

The **Treatment** should consist of free stimulation, generally by the administration of alcohol and such drugs as ammonia, strychnine, etc., while the application of mustard poultices and counter-irritation with stimulating liniments should be persevered with. In young children collapse is frequently due to an excessive amount of secretion blocking the bronchial tubes. Encouraging the child to cough, or giving emetics, may tide him over a serious condition. When the stomach is emptied, the bronchial tubes are also cleared, but emetics are depressing, and therefore stimulation may need to be vigorously carried out after their administration.

Atelectasis

Deficient expansion of the lungs at birth. The anterior margins of both lungs and the lappet of the left lung which covers part of the heart expand last after birth. The want of expansion is sometimes the result of feebleness, and where it does not occur, the child remains cyanosed, and generally soon dies, sometimes with convulsive seizures. The usual treatment is to stimulate the surface of the skin by the alternate application of cold and hot water, and in most cases this treatment is alone sufficient to effect full expansion. The trachea or bronchi may be blocked with secretion, and it is easy, by mechanical means, sometimes by stimulating the child to cough or making him sick, to clear away the obstruction.

XI. PULMONARY TUBERCULOSIS

(1) Acute Tuberculosis of the Lungs

(a) Type 1. Lobar Pneumonic Tuberculosis

This is a rare form, and it is not always easy to ascertain by what channels the whole of one or more lobes have become involved. There are generally a few caseous foci which probably indicate an older disease, but the most of at least one lung is consolidated and closely resembles the grey hepatisation stage in an ordinary lobar pneumonia. On careful examination with the magnifying glass, numerous tubercle nodules are seen dotted in miliary arrangement throughout the whole of the consolidated lung, although it is quite probable that, in certain cases, the disease may be a combined attack by the tubercle bacillus, together with one or other of the organisms responsible for croupous pneumonia. There is generally some pleurisy, and tubercle nodules may be dotted over the pleura.

The **Clinical Features** begin with a sudden rise of temperature, with rigors, great dyspnœa, and pleuritic pain associated with cough, while the sputum is not very dissimilar to that in croupous pneumonia, being generally tenacious and blood-stained or rusty in appearance. There may be the typical malar flush, so commonly seen in croupous pneumonia, but soon after the period when the crisis should arrive, there is obvious evidence of destruction of lung tissue, and the sputum generally becomes extremely purulent, and on examination is found to contain numberless tubercle bacilli. The temperature swings, often reaching 103 or 104 F. at night, and falling to about 101 during the day, with considerable sweating; this tends to increase as the disease progresses.

The *Physical Signs* are those of lobar pneumonia, with, in addition, evidence of rapid excavation, and the breathing associated with this disease is just as typically tubular as in lobar pneumonia. Death occurs with great rapidity, often in 2 weeks, sometimes 4 to 6 weeks, and very rarely does the disease become chronic. Sometimes, in cases which probably are in reality a mixture of true pneumonia in addition to the

tubercular element, the consolidation may largely clear up, but it leaves a part of the lung evidently the site of tubercular disease.

Diagnosis.—There is a close resemblance between this condition and *lobar pneumonia*, only the temperature swings more in the tubercular form than in the other, and the presence of tubercle bacilli in the sputum, with early evidence of breaking down in the lung, as shown by the appearance of elastic tissue in the sputum, render the diagnosis easy, at least after a certain length of time has elapsed.

(b) Type 2. Acute Broncho-pneumonic Tuberculosis

This is another form of an extremely acute invasion of the lung by the tubercle bacillus. Probably in these cases the tubercle bacilli are inhaled, and we find the grape-like clusters corresponding to the distribution of the bronchial tubes. Round about the affected bronchial tubes there is a definite catarrhal pneumonic process, and in very acute cases the little caseous areas are apt to run together to form much larger masses. The lung between these masses is congested, and the pleura may show tuberculous pleurisy. Generally these areas are more marked in the upper lobe and near the apex, although any part of both lungs may be involved. Sometimes there is evidence of a much older focus of tubercular disease, from which the recent acute broncho-pneumonic involvement has originated. Cavity formation occurs early, and not infrequently the larynx and trachea are found to be affected.

Clinical Features.—The onset is sudden, starting with rigors, much like Type 1; but there are cases where an insidious commencement ushers in the attack. Not infrequently it is secondary to influenza. Sometimes after an insidious commencement, a considerable haemoptysis precedes the rapid development of the disease. There is loss of appetite, the tongue is furred, and there is vomiting; the sputum is muco-purulent, and sometimes blood-stained, and later it becomes markedly purulent. There are many tubercle bacilli, and there is much elastic tissue in the sputum; the temperature usually reaches at least 102°, possibly 104° F., and the clinical features soon demonstrate the rapidly advancing

and serious nature of the disease, vomiting and diarrhœa being common symptoms. A good deal depends on whether the process is widespread, or limited to one lobe or one lung; where there are many scattered areas of involvement, the results of septic absorption are much more pronounced.

The *Physical Signs* are those of small patches of consolidation, which, however, may run together to form larger and more extensive areas. *Inspection* may show diminished movement, very probably well marked at, or near, one or other of the apices. *Palpation* reveals increased vocal fremitus, and also the fremitus of accompaniments which are generally medium and coarse moist râles. On *Percussion* the areas of

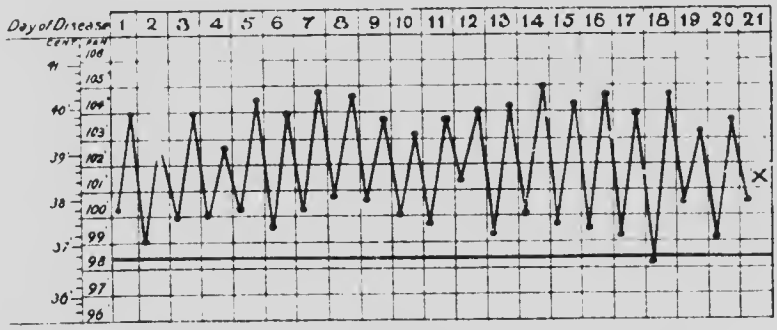

Fig. 28.—Temperature Chart. Pulmonary Tuberculosis. Hectic temperature; fatal termination.

dulness are sometimes easily mapped out, and where excavation has commenced, a tympanitic or cracked-pot note may be obtained. *Auscultation* yields harsh vesicular breathing, with expiration prolonged, often of a bronchial character, while over patches of consolidation the breath sounds are frankly bronchial. Over the cavities the breathing is cavernous, but one important feature of the breath murmurs in tubercular disease is the jerky or cog-wheel character, so frequently made out over less advanced areas of the disease. This jerky character does not disappear when the patient takes a deep breath. In addition there are numerous clicking,[1] moist sounds specially heard during inspiration, and which are increased on coughing. Should excavation be progressing, these moist râles become consonating. Vocal resonance is usually bronchophonic, and whispering pec-

[1] The clicking character of the moist rales is due to their conduction through consolidated lung.

toriboquy is often obtained over the cavities. The presence of friction is not always easily made out, owing to the existence of so many moist râles, but the friction sounds are not altered by coughing, while moist râles are distinctly modified. Friction sounds increase in distinctness when firm pressure is made with the chest piece of the stethoscope, and they are generally accompanied by pain, of which the patient usually complains. Friction is also, as a rule, better marked at the *end* of inspiration and the *beginning* of expiration, and becomes much more distinct when a deep breath is taken. The sputum usually contains many tubercle bacilli and much elastic tissue, although it must be remembered that death may occur before the disease has reached the stage of extensive lung destruction.

Diagnosis.—The rapid emaciation of the patient, the evidences of the spreading consolidation in the lung, extending not infrequently with alarming rapidity, and the typical temperature with hectic sweating, render the diagnosis easy even in young children who may swallow the sputum.

The **Prognosis** is always grave, and death may occur nearly as rapidly as in Type 1. Fortunately the disease can be arrested in some cases, and many months may elapse before death occurs, or the condition may pass into the chronic stage although with a lung very seriously damaged.

(c) Type 3. Miliary Tuberculosis of the Lungs

Pulmonary tuberculosis of the miliary type may be part of a general involvement, or it may be limited to one lung, or merely part of a lung. Sometimes a miliary tuberculosis is the result of ulceration through the wall of a pulmonary artery of one or more tubercle nodules, and thus the blood-stream to the part of the lung supplied by the artery in question may become infected by tubercle bacilli. In such a case the nodules are scattered along the line of the vessels but a miliary tuberculosis may be distributed by the lymphatic system. With this condition there is usually a varying degree of pulmonary congestion, and sometimes consolidation.

Clinical Features.—Where there is extensive involvement of the lungs, the phenomena met with in general miliary tuberculosis will be present. After a preceding period of ill health the attack begins with rigors, there is generally head

ache and malaise and marked loss of appetite, the face becomes flushed, and the temperature varies from 103 to 105 F., falling towards the early morning, with profuse sweating. There is a short, dry, hacking cough, and the respirations are generally from 50 to 80 per minute, while the patient usually shows marked dyspnœa, with increasing cyanosis. The sputum is often clear mucus, sometimes it is muco-purulent, and occasionally blood-stained.

The *Physical Signs* are chiefly negative; unless there is some patch of older tubercular disease, there may be nothing distinctive on inspection, palpation, percussion, or auscultation, except the evidence of a general bronchitis with a few moist râles. In some cases we find a trace of albumin in the

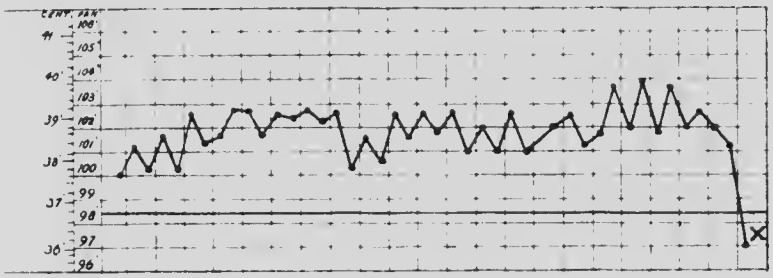

Fig. 94.—Temperature Chart. Miliary Tuberculosis of Lungs; fatal termination.

urine where tubercle nodules are deposited in the kidneys. Tubercles in the alimentary tract are not infrequently associated with marked changes, such as vomiting, sometimes diarrhœa, and occasionally waxy degeneration, and in certain cases definite tubercular ulceration in the lower part of the ileum. In the *Hæmopoietic System* the spleen is often enlarged and waxy, with sometimes the presence of miliary tubercles in the organ. In the *Circulatory System* there is nothing distinctive of the disease, but in the *Nervous System* there may be tubercular meningitis, and sometimes the appearance of miliary tubercles in the choroidal coat of the eyes. The clinical features of tubercular meningitis are described under the diseases of the Nervous System (p. 654).

The patient usually sinks early into the typhoid state, and the phenomena are generally governed by the degree of involvement of the meninges.

The **Diagnosis** is by no means easy: the possibility of

typhoid fever should invariably be kept before the mind, and Widal's reaction is of great value in some of these doubtful cases. It should be remembered that in many cases of miliary tuberculosis there are no tubercle bacilli in the sputum, and unfortunately tubercles in the choroid are rather the exception than the rule.

In *acute capillary bronchitis*, advancing to *catarrhal pneumonia*, there is sometimes doubt as to whether the case is not really one of miliary tuberculosis. Generally a few days will clear up the diagnosis, because the definite areas of dulness in catarrhal pneumonia are not present in most cases of miliary tuberculosis, and as a rule the temperature is less hectic in non-tubercular catarrhal pneumonia than in a miliary tuberculosis, although the temperature chart on the preceding page shows only slight hectic characteristics.

Prognosis.—It is rare for the patient to survive more than 3 or 4 weeks, and he rapidly loses flesh and strength, but sometimes life is prolonged for 3 to 4 months, and occasionally miliary tubercles become obsolete, and the patient recovers. Unfortunately this latter course is very exceptional.

The **Treatment** must be on general lines, and no suggestions can be offered as to particular methods likely to yield good results. If any points demand special mention, they are the value of oxygen inhalations, and the benefit which may be obtained from the application of cold to the chest wall.

(2) Subacute and Chronic Tuberculosis of the Lung

The majority of cases of pulmonary phthisis may be grouped under this heading, and for purposes of description an early case of the disease will be briefly considered in the first place, and afterwards the typical features of one more advanced.

Etiology.—There is little to be said with regard to the etiology of this form of tuberculosis that does not apply to the other forms just considered. There is, in the first place, a very important hereditary predisposition, a predisposition which rather implies insufficient development of the chest and a want of vitality of the individual as a whole, than the direct transmission from either parent of the tubercle bacillus. The types of chest associated with tubercular disease are either—(1) *alar*.

in which, along with anterior flattening, there is always marked prominence of the wings of the scapulae posteriorly, or (2) the chest may be *flattened*, with sloping shoulders and a distinct cervical stoop almost amounting to spinal curvature. With these peculiarities there is sometimes a long chest, with hollowed-out sternum, and a narrow subcostal angle. In the subjects so affected there are often evidences of rickets with the knob-like prominences at the junction of rib and cartilage, and a very marked indrawing of the chest, commonly known as Harrison's sulcus, which is just above the attachment of the diaphragm on either side, and which slopes downwards from near the sternum towards the mid-axillary line. In many of these cases there is bad nutrition, sometimes a history of very insufficient feeding and care, and not infrequently also inherent delicacy. Unquestionably the presence of adenoids neglected in youth may lead to the kind of defective development of the chest to which reference is made.

The disease generally manifests itself about the age of adolescence, or just after puberty, when probably the strain of life is greatest. In persons with a tubercular predisposition, exposure to cold or wet, sleeping in an ill-aired apartment, working at a dusty occupation, the strain of profuse menstruation, some debilitating illness and especially influenza, even a great mental worry—these, and similar causes may induce an attack of pulmonary tuberculosis. Reference has already been made in previous pages to the virulence of tuberculous sputum, and to the risk of tubercle bacilli becoming disseminated throughout the air of rooms or houses, and, given a suitable nidus, there is the greatest risk of the tubercle bacillus, inspired into the lung, setting up active disease. It is a remarkable fact that in asylums and other institutions, in which, for various reasons, free life in the open air is not possible for all the inmates, tubercular disease of the lungs is extremely common. Tubercular disease of the lungs is rare in persons living at high altitudes, and it is proportionately common in damp, cold, low-lying countries. Inclement weather, and especially cold and damp, are predisposing factors.

Pathological Anatomy.—In most cases pulmonary tuberculosis is met with first near the apex of the upper lobe of one lung, and it is probable that this seat of election is

due to the fact that there is less exchange of air in that portion of the lungs. A not infrequent position is the apex of the lower lobe, although this is generally a secondary involvement, the original focus being in the upper lobe. It is not the *extreme* apex of the upper lobe which is usually the first part invaded, but it is about an inch below the extreme apex. The right apex is slightly more frequently involved than the left, but in a large proportion of cases both apices are found to be affected. The distribution of the disease is often mainly in the line of the peribronchial lymphatics: it is therefore a chronic broncho-pneumonic tuberculosis, and the tubercle bacilli, once they have entered the lung, are distributed largely by the lymph-stream, although it is not uncommon to find, as the result of the invasion of the wall of one of the branches of the pulmonary artery, that a limited miliary tuberculosis is also present. The cavities found in the affected lung are chiefly the result of excavation of caseous areas. These are closely related to the bronchial tubes, and the cavities are lined with a pyogenic membrane often dotted over with miliary tubercles. These cavities have not rounded walls as a rule, but are irregular, one cavity sometimes appearing to have numerous branches corresponding to the divisions of the original bronchi. Most large cavities have septa running through them in which blood-vessels pass. These vessels are generally obliterated, as the result of endarteritis obliterans, but sometimes they remain patent, and are invaded by tubercle bacilli, as already mentioned, and occasionally these form fairly large, aneurismal dilations which may nearly fill the whole of the cavities in which they develop. Such aneurisms not infrequently give way, and may lead to fatal haemorrhage. The amount of destruction of the lung varies greatly; sometimes the whole of one lobe may be excavated, in other cases the cavities are of small size, and the old bronchial walls can be readily recognised. Some cavities are truly bronchiectatic, although ulcerations of their walls may occur secondarily. Coexistent with the more chronic varieties of the disease there is marked interstitial change—a fibrosis of the lung, which is the commonest process of cure of a tubercular lung. The degree of this fibroid change varies greatly: it may be very marked and very extensive; it may, in other cases, be comparatively limited, and associated with it there is almost

always more or less thickening of the pleura, sometimes with pleural effusions, and often with the presence of tubercle nodules on the pleura. Often where the disease becomes quiescent at a very early period, calcareous deposition occurs in caseous nodules, together with a limited fibroid change such as we have just described, but it is rare to find, even after many years of quiescence, that the tubercle bacilli have absolutely and finally disappeared. The glands at the root of affected lungs generally show caseous foci, and in these caseous areas calcareous deposition is very common, although sometimes the glands soften and form small abscesses.

Amongst changes in other organs to which chronic tuberculosis, primarily in the lungs, may give rise, the following may be mentioned: In cases where there is much suppuration, waxy degeneration of the liver, spleen, kidneys, and alimentary tract may ensue, leading to the rapid death of the patient. In other cases tubercular ulceration of the intestine may follow from the swallowing of tuberculous sputum, and as a secondary result the mesenteric glands may become affected. The larynx, frequently involved by a comparatively mild catarrh of the vocal cords, sometimes shows marked infiltration or ulceration, or both together, but far less frequently than one would expect. In connection with the circulatory system there is occasionally tubercular pericarditis and endocarditis, and it is only necessary to mention the fact that miliary tuberculosis or tubercular infection of other organs of the body may be a sequel to pulmonary tuberculosis.

Clinical Features of a Case of Early Pulmonary Tuberculosis.—There are many modes of onset of the disease. In not a few cases it begins insidiously, and only eventually is attention directed to the lungs. In a second group of cases a haemoptysis, which may be extremely severe, is the first indication to the patient of any pulmonary mischief: in another group of cases a severe bronchitis occurs, from which the patient does not completely recover. Pleurisy is yet another affection which appears to usher in pulmonary tuberculosis: there may or may not be effusion, but the cough from which the patient suffers remains behind, even after the effusion and friction have disappeared. It is an important point to remember that in young patients apparently suffering from anaemia, or from some alimentary disturbance, the real

cause of the trouble may be pulmonary tuberculosis. Tubercular glands in the neck may be the source of infection.

While it is true that the shape of the patient's chest, with the presence of cough and sputum, may yield important clues to the diagnosis of an early tuberculosis, there are only two or three clinical features specially typical of pulmonary tuberculosis at a very early stage:—

(1) The respiratory capacity of the lung in pulmonary tuberculosis always falls far short of the normal amount, and were it possible to use the spirometer, the information yielded would be of great value. A careful note, both by hand and by tape measure, should be made of even the slightest difference of movement between the two sides of the chest, although it is true that in right-handed persons the right side of a normal chest acts more vigorously than the left, and that sometimes from rickets or other cause the usual symmetry of the chest may be altered, and that such alteration may have little connection with phthisis pulmonalis.

(2) The breathing over a patch of early phthisis is sometimes cogwheel-like or wavy, and this peculiar character is not lost when the patient breathes deeply.

(3) There is generally some secretion, even although the amount of sputum may be extremely small, and a few clicking sounds are often detected over the affected lung when the patient takes a deep breath, and especially after coughing.

The Clinical Features of a more advanced Case of Pulmonary Tuberculosis.—Pain in the chest and cough are two early and important symptoms; the pain is often pleuritic, and may occur over an affected apex, or may be referred to one shoulder blade. The cough is generally distinctive; it is short and dry, with, at first, little expectoration, and indeed the appearance of much secretion is dependent on the presence of bronchitis or the commencement of disintegration processes in the lung. The cough frequently induces vomiting, and especially when it has a laryngeal as well as a pulmonary cause. It is a remarkable fact that although serious involvement of the larynx is by no means common in pulmonary tuberculosis, catarrh or simple anæmia of the laryngeal mucosa is associated with between 80 and 90 per cent of all cases.

The sputum is typical: it may vary from muco-purulent

to purulent, it may be nummular, and it may, or may not, contain blood, but sooner or later, in every case of chronic broncho-pneumonic tuberculosis, tubercle bacilli will be found, and in very many cases, elastic tissue from the air-vesicle walls is readily recognised. Tubercle bacilli should be stained by the Ziehl-Neelsen method described on p. 126. Elastic tissue must be looked for either by squeezing some sputum between two glass plates, and inspecting with a hand lens, and thereafter mounting on a slide, and examining with the microscope any suspicious portion, or by boiling with liquor potassæ B.P. solution, diluted with equal parts of water, and examining the deposit after allowing it to settle in a conical glass. The amount of sputum varies greatly; it may be fetid, or merely have the peculiar sweetish odour so characteristic of tuberculous sputum. Sometimes blood appears, either in large or in small amount. It is rare to find that a case of tuberculosis runs its whole course without any haemoptysis, but only in about half of the cases does the spitting of blood become an important feature. Such haemoptysis may be the result of severe strain, possibly merely the stress of coughing causing congestion or trivial oozing of blood; in other cases it is due to rapidly destructive processes in the lung; while in yet others, and these are cases of very severe haemorrhage, it may result from the rupture of a miliary aneurism in one of the cavities. It is very rare indeed to have a severe haemorrhage in the early stages of phthisis, although in the later stages the bleeding is apt to be more copious. Where there has been a considerable haemoptysis, blood is expectorated for several days afterwards, and clots and blood-casts of the bronchi may be coughed up. Sometimes old calcareous masses are found in the sputum. These indicate calcareous deposits in an old caseous nodule, and their appearance is generally the result of a local recrudescence of the disease permitting these little nodules to be separated and spat up in the sputum.

Dyspnœa depends largely upon the area of lung involved, and whereas, in some cases, breathlessness may merely follow from exertion, in other cases it is exceptionally severe, and is associated with more or less cyanosis.

Emaciation is usually marked, and the sharply cut features, so frequently noted in phthisical patients, depend largely on

loss of flesh. The temperature varies within wide limits; in mild cases a nocturnal rise of 1° or 2° may be the limit of the pyrexia, whereas in other cases the temperature may reach 102°, 103°, or 104° F., generally falling towards morning with excessive sweating. The temperature chart is often a useful guide in deciding whether the tubercular process in the lungs is extending, because a minimum temperature which never reaches the normal has been described by the term *remittent*, and generally indicates advancing disease (see Chart below). On the other hand, a daily variation of temperature of 2° or 3° is not incompatible with quiescence of a tubercular area. Where there is a tremendous fall, sometimes reaching normal, and associated with pronounced sweating, the tempera-

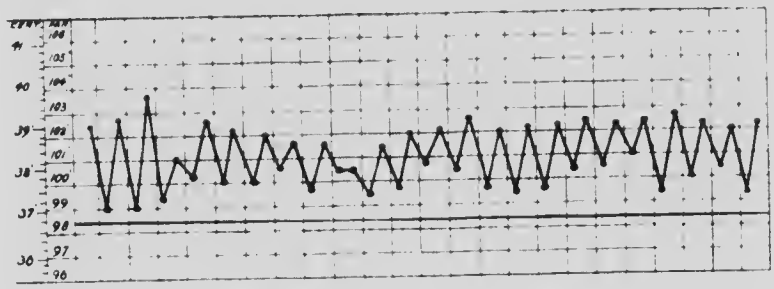

FIG. 30.—Temperature Chart. Caseating Pulmonary Tuberculosis. Advancing Disease.

ture is described as *hectic*, and implies septic absorption, and generally indicates an early and fatal termination of the illness (see Chart on p. 471). Improvement in the patient's condition in the direction of quiescence is usually demonstrated in the temperature chart by a lessened degree of pyrexia at night, and as a result diminished sweating. The perspirations, so typical of the phthisical patient, frequently saturate the night-dress, and render the use of flannel or woollen garments imperative. The sweating is to a large extent the result of toxins produced by the tubercle bacillus. There is almost always more or less malaise, the degree depending largely on the activity of the pulmonary disease.

Physical Signs associated with the Respiratory System.—*Inspection* generally reveals one or other of the types of phthisical chest, and the flattening, with marked lagging, of the affected apex or base is usually evident. The expansion of the affected part of the lung is invariably deficient, and the utmost

care should be taken to compare accurately the movements of the two apices, as viewed by looking over the patient's shoulders, so as to contrast the one side with the other. *Palpation* greatly aids in appreciating diminished expansion, and it is well to take a measurement of the amount of expansion of both sides of the chest on full inspiration. The lagging, referred to under inspection, is much more obvious to the hand on palpation. The vocal fremitus is generally increased if the area of disease enables the vibrations to be better conducted. The *Percussion* note over the affected lung is dull, and should be very carefully compared with the corresponding area on the other side: and not merely will the ear enable the observer to obtain a fairly accurate impression of the amount of consolidation, but the sense of resistance to the hammer finger is greatly increased. A tympanitic note is obtained over a superficial cavity, and when the patient opens the mouth and breathes deeply, the typical cracked-pot note is obtained during expiration. Cavities are most frequently to be found just below the clavicles and also at the apices of the lower lobes. Tidal percussion both at apices and bases should not be forgotten. It implies the inability of diseased lung to expand as it ought to do towards the extreme limits of the pleural sac on full inspiration.

Auscultation.—The breath-sounds vary greatly in different cases: they may be faint, from interference with the entrance of air into the affected part of the lung: they may be harsh vesicular with expiration prolonged, or they may be frankly bronchial: the inspiratory murmur is sometimes cogwheel-like, and where cavities are present the bronchial breath-sounds acquire a cavernous, or possibly amphoric, character. Tubular breathing is not so common as in the acute lobar pneumonic type of the disease. The accompaniments, generally best marked after coughing, have a peculiar clicking character, and they may be fine, medium, or large in size, while in cavities they acquire consonating qualities. Rhonchi are frequently met with, depending, however, on the amount of bronchitis present. The vocal resonance is increased and may be bronchophonic, and whispering pectoriloquy is obtained over cavities. Pleuritic friction is often present.

Cavities are generally easily diagnosed: the tympanitic or cracked-pot note, the cavernous or amphoric breathing, the consonating moist râles should secretion be present in or near

the vomica),[1] and lastly, whispering pectoriloquy, are all typical phenomena. It should, however, be remembered that a cavity may be full of secretion, in which case the typical signs will only be obtained when, after a fit of coughing, it has been emptied of sputum. Cavities may also be deep-seated, and therefore may not yield a definite tympanitic or cracked-pot note on percussion. It is necessary to point out that the size of a cavity cannot be gauged by the pitch of the tympanitic note obtained, because the quality of note depends greatly on the size of the opening of the bronchial tube into the cavity. The larger the opening, the higher in pitch does the note become, and the opposite also holds good, that the smaller the opening, the lower in pitch is the note on percussion. The coin test is applicable to large cavities, and can be used for the definition of their boundaries. It consists in tapping one coin on another coin placed on the chest wall over the cavity, while with the stethoscope, also placed over the cavity, the ringing sound obtained is conducted to the ear.

Very frequently there is evidence of displacement of neighbouring organs, and especially the heart, as the result of tubercular disease in the lung. This is common in cases where fibroid changes have developed, the heart being pulled over toward the affected side.

Complications and Sequelae referable to the Respiratory System.—The *larynx* is apt to be involved in a considerable percentage of cases: not merely the slight huskiness of voice already mentioned, but definite tubercular ulceration may occur. There may also be many miliary tubercles scattered over the mucosa of the trachea and larger bronchi, and there is always a tendency, where a tubercular area of lung exists, to find that the disease spreads by neighbouring and communicating bronchi to other parts of the same lobe or the same lung. This is probably due in many cases to sputum over-flowing into a neighbouring bronchus, along which it may be inspired, and such an accident is peculiarly liable to occur where there is much sputum, and when it gets a chance of accumulating during sleep. Haemorrhage is also a common mode of extension of the disease, blood containing tubercle bacilli, being not infrequently inspired

[1] The term "consonating" means a musical or ringing quality acquired by the rales because of overtones produced in the cavity.

into healthy lung, the haemorrhage having occurred in the tubercular area.

Emphysema is an almost inevitable sequel in cases of tubercular disease of the lung. It affects the healthier portions of the diseased lung, and sometimes the whole or the greater part of the opposite lung. In reality it is a compensatory process, and unless excessive, rarely causes much if any trouble to the patient.

Pneumothorax is one of the most serious sequelæ to which a phthisical patient is prone. Where a cavity near the pleura ulcerates into the pleura, a pneumothorax results, possibly limited by adhesions, and possibly involving the whole of the pleural sac. The patient becomes very dyspnœic, and death is hastened. Not infrequently a pyopneumothorax develops and adds much to the distress of the patient.

Pleurisy has already been described, and is a part of the tubercular process: but it may be mentioned that effusions are not uncommon, sometimes consisting of ordinary inflammatory lymph, and sometimes being a purulent effusion. Dry pleurisies, limited, as a rule, to the neighbourhood of the disease, are very common.

In a few cases of advanced pulmonary tuberculosis the lung becomes the site of invasion by *gangrene-producing organisms*, and death may follow, under conditions peculiarly trying for the patient and attendants alike.

A certain proportion of cases die from pneumonic involvement of the lung, sometimes by infection with the pneumococcus, and sometimes by the development of acute pneumonic tuberculosis, which has already been described.

Clinical Features associated with other Systems.

Alimentary System.—Dyspepsia is an almost constant feature in marked pulmonary tuberculosis, and not infrequently vomiting and diarrhœa ensue,—the former perhaps largely due to the stress of coughing, the latter often the result of tubercular ulceration or waxy degeneration of the intestinal tract. The tongue is typical; it is furred and generally moist, but in severe cases it may be covered with patches of thrush, which may also be seen on the gums and fauces. Where there is phthisis laryngea, pain on swallowing is very common, and adds greatly to the difficulty of feeding the patient.

Haemopoietic System.—Anæmia is frequent, but beyond

the fact that a leucocytosis generally develops where there is much pus formation, and that the blood-plates are said to be increased in number, the blood yields little useful information.

Circulatory System.—The displacement of the heart and the retraction of the anterior edge of the left lung are usual features in cases in which there is much cirrhosis. The pulse is generally of low tension and may be very dicrotic.

Integumentary System.—The skin of a phthisical patient is often suggestive; the hair is apt to become thin and dry, pigmentation of the skin may occur, and many phthisical patients show very marked freckling. It is common to find pityriasis versicolor on chest and back, due to the presence of the fungus, microsporon furfur, and probably largely induced by the excessive sweating. Very rarely do the patches cause itching or give any trouble. The tips of the fingers and toes may show clubbing, peculiarly evident in emaciated patients, and usually associated with extensive fibroid phthisis.

Urinary System.—The appearance of albumin in the urine may be significant of a secondary involvement of the kidneys by miliary tubercle or the advent of waxy degeneration; more frequently it is simply due to backward pressure.

Nervous System.—Many patients suffering from pulmonary phthisis develop tubercular meningitis. A much more unusual condition, and probably really the result of alcohol, or possibly some drug, such as arsenic, is the appearance of peripheral neuritis in the later stages of pulmonary phthisis. A curious feature of many phthisical patients is the happy, hopeful spirit to which the term *spes phthisica* has been applied. It not merely means that the patient is bright and cheerful, but implies a strong expectation on his part of final recovery, and the worse the disease, the more hopeful may he become.

A familiar phenomenon called myoidema may be noted on percussing the chest; the portion of intercostal muscle contracts after tapping, and remains so contracted, forming a local swelling for a considerable period of time. This phenomenon should more correctly be described under the locomotory system; the cause of this abnormal muscular irritability is unknown.

Course, and Mode of Termination of the Disease.—A case of pulmonary phthisis may be chronic from the outset, and

may, at any stage, become quiescent, the affected parts of the lung being gradually surrounded by fibrous tissue. In other cases the disease may slowly progress, the patient living in comparative comfort for many years. In yet other cases the disease becomes a fibroid phthisis, tubercular in nature, but owing to excessive suppuration it may finally kill the patient, either from septic absorption or waxy degeneration, or possibly simply from exhaustion. Chronic pulmonary tuberculosis presents itself to the physician under many different forms and types, and much experience is necessary in order to recognise the exact position of matters in the lungs and the probable course of the disease.

Not a few cases die of sheer exhaustion: others, as already noted, die of pneumothorax; others from severe pulmonary haemorrhage, or from the development of gangrene; and probably in a larger number of cases than our present knowledge suggests, death may be due, in part, to thrombosis occurring in important branches of the pulmonary artery.

Diagnosis.—There is no disease which, in an advanced stage, is so easily recognised as pulmonary tuberculosis. In most cases tubercle bacilli and elastic tissue are found in the sputum, and even where there is no cough and perhaps no sputum, the physical signs of tubercular consolidation are definite and typical. They may for a time be confused with *catarrhal pneumonia*, but the patches in catarrhal pneumonia clear up, whereas in tubercular disease they persist. In cases of *bronchitis* accompaniments are generally heard, either in larger or smaller tubes over the whole of both lungs, but in tubercular disease the accompaniments, generally clicking, moist sounds, are usually limited to the area affected.

Tuberculin has been suggested as a suitable means of testing the presence of tuberculosis, but its use for this purpose has certain objections, mainly its unreliability (see p. 131).

Prognosis.—There is no more difficult matter to decide than the prognosis of a case of pulmonary tuberculosis. Unquestionably freedom from hereditary taint has an important bearing, and the quiescence of the disease, without any attack of haemoptysis for at least several years, should make it possible to offer a more favourable prognosis. It should, however, be remembered that patients who have tubercular

foci in the lung possess a permanent and irradicable source of danger which should never be left out of account. The danger may be *nil* if the patient exercises prudent care, but there is always the chance of the disease being stimulated to renewed activity,—a chance which should ever be before the mind of the physician, when making suggestions as to the patient's future life and work.

It is wise to endeavour to prevent the marriage of persons suffering from pulmonary tuberculosis, although it is possible that, in the event of complete quiescence, there may be less tendency for children of the marriage to become tubercular. There is, however, a want of vitality, or of resistance, or it may be a congenitally weak chest, which can truly be described as the heritage of phthisical parents.

Treatment. *Prophylactic Treatment.*—It is of the greatest importance to impress upon patients who are the subjects of pulmonary tuberculosis that they are infectious, and that the chief danger is due to the sputum. There are many useful inventions by which the sputum can be collected and afterwards destroyed, such as by the aid of strong antiseptics or by fire. A portable flask of some kind must be obtained, and the one chosen should have an arrangement by which it can be thoroughly cleansed. In the bottom of the flask there should be a little 10 per cent solution of carbolic acid, and the patient should be enjoined always to use the flask for purposes of expectoration. Handkerchiefs should never be employed, and if, from the patient's weakness, something of the kind must be resorted to, rags, which can afterwards be burned should be used. The patient should sleep alone, and certainly if possible, either in a room by himself, or at any rate in a separate bed from healthy persons. It is a good precaution to enjoin the patient to wash out the mouth with permanganate of potash, or other antiseptic solution, where the sputum is excessive and teeming with bacilli. On no consideration should persons who have a hereditary predisposition to, or have previously suffered from, a chest affection, be permitted to nurse or even to live with phthisical patients.

Open air is probably the chief mode of prophylactic treatment, and where free access of fresh air is permitted there is less risk of infection to healthy persons. It is a remarkable fact that, in consumption hospitals, few of the

attendants become infected with the disease, unless they have a family tendency, or have allowed their state of health to fall much below par.

General Treatment.—The first essential in treatment is rest in all cases where there is any pyrexia at all. A temperature above 99° F. or 99·5° F. means that auto-inoculation is going on, and rest in bed is imperative until the temperature (taken three times a day) is normal all the time. Then, and not till then, the patient can get up and gradually begin walking, and later other exercises.

The second essential in treatment is open air. Where possible the patient should live, eat, and sleep in the open air. Some cases are not able, owing to the condition of their throats or lungs, to stand the cold, but even for them a modified treatment on the same lines can be easily carried out. Where the patient sleeps under cover, the windows must be wide open, and, if possible, the bedroom should have a south exposure. He should wear flannel next the skin, and, while a sufficiency of blankets is necessary, any excess of clothing, which might induce sweating, must be discouraged. A hot bottle in winter is a necessity, and there is no reason why the patient should suffer from excessive chilliness. Much may be done, however, to harden the patients by sponging the chest and neck with cold water every morning, summer and winter, and in time they become acclimatised even to the great cold of a high mountain health resort. The effects of the sun are most beneficial, and with plenty of fresh air and bright sunshine, a patient with a swinging temperature and a poor appetite often rapidly and steadily improves, and reaches the stage when life can once more be enjoyed. It is necessary to avoid draughts, and this is accomplished in many sanatoria by having revolving screens which shelter the patients from the prevailing winds.

Certain cases of phthisis improve to a remarkable extent when taken to a high altitude, and Davos Platz, St. Moritz, Arosa, Montana (all about 6000 feet), and many other Alpine health resorts, are famous for the purity and dryness of the atmosphere, and the large amount of sunshine even during the winter months. Many patients suffer considerably from bronchitis, and they do better at a lower altitude, such as may be obtained at one of the winter health resorts above Montreux

for instance, Château d'Oex, Caux, and many others. Patients who are extremely ill, and patients who suffer from repeated haemorrhage, should not be sent to a high altitude: most satisfactory patients are those whose cases are chronic. For persons to whom the rigour of an Alpine winter is unsuitable the warm, dry air of Egypt, and the bright sunshine of Algeria and the Mediterranean, may be commended; and many patients whose lives would be greatly shortened if they remained in our own land, live more comfortably, and for a longer time, especially during the colder months, at one or other of the innumerable Mediterranean resorts. It should, however, be remembered that phthisis is infectious, and that, if a patient is extremely ill, other persons associated with him in hotels and elsewhere may suffer, while the benefit he obtains personally is often slight. Under no circumstances should a dying patient be sent away from home, because even in the most luxurious hotels, and under the best possible conditions, he cannot have the comforts of home.

Diet. It is an important matter to make the patient eat, and eat well. It is unwise to stuff our patients, and this method of treatment has to a large extent been superseded, although forced feeding is useful in some cases. It consists in forcing the patient to take large quantities of milk, finely pounded meat (sometimes raw), fish, and other nutritious food, so prepared as to be readily digested; and where the patient will not eat, this method has occasionally proved satisfactory. Debove's method of forced feeding implies the administration of half-a-pound of raw meat thrice daily. Dyspepsia should be carefully treated; sometimes the use of pepsin or a bitter tonic may help a capricious appetite; and often the combination of fresh air with wholesome, easily digested food enables a patient to put on weight rapidly.

Medicinal Treatment. Cod-liver oil is one of the most important and best of remedies. It is often well taken in one big dose in the forenoon, midway between breakfast and lunch, or in small doses after food. Its taste is readily concealed by the use of a pinch of salt, a little bit of dry bread eaten after the dose, or a squeeze of lemon juice; and in the form of emulsions, cod-liver oil, although diluted, frequently acts well. It should never be given where waxy degeneration is present.

General tonics, such as iron, strychnine, arsenic, and the mineral acids, are extremely beneficial; but arsenic, which used to be given in enormous doses in the form of the insoluble cacodylate of soda, should be given with considerable caution.

Perhaps the most valued drugs are those which are believed to act as antiseptics. Guaiacol and creosote are admirable remedies, combined with the cod-liver oil, or given alone in capsules, the only trouble being that the stomach is apt to be upset, and that the taste comes back into the mouth many times after the capsule has been swallowed. Guaiacol carbonate, a tasteless powder, given in 20-grain doses thrice daily, sometimes exercises a salutary influence on the patient, but unfortunately its success is by no means certain. Turpentine, in 15 to 30 minim capsules, is to a large extent excreted by the lung, and has been found serviceable in some cases, while iodoform administered internally as a pill (gr. $\frac{3}{4}$) has been recommended as of distinct advantage.

Special Methods of Treatment and the Treatment of Symptoms.—While the use of tonics and the internal administration of guaiacol and creosote may benefit the lung, a further attempt should be made to treat the local condition. Intratracheal injections of guaiacol 2 parts, menthol 10 parts, in sterilised olive oil 88 parts, may be used, and about a drachm of the mixture injected twice or thrice a day. It is, however, only a chance if much, or any, of the injection reaches the affected part of the lung, and therefore it has been suggested that injections of iodoform or other antiseptics should be given through the chest wall. The objection to this procedure is obvious, that in addition to the conceivable risk of pneumothorax, there is always the possibility of a septic needle track being left when the syringe is withdrawn. Surgical procedure has in certain cases been attempted, but the results are not satisfactory. A useful method of administering iodoform is to give an emulsion of the drug as an intravenous injection, and the results, in certain chronic cases, have been favourable, but this method of treatment has lost popularity owing to the risk of phlebitis which may ensue after repeated injections. Almost immediately after the injection has been given, the patient's breath smells of iodoform, and coughing is induced.

Inhalations are of great value in attempting local treatment of the lung; they consist in finely divided particles of a

volatile antiseptic being inhaled by the patient, and amongst the best agents to use are the essential oil of cinnamon, the essential oil of peppermint, creosote, and guaiacol. Some of these are administered in such a way that the patient inhales the drug both through the nose and the mouth, and in the case of creosote and guaiacol the remedies are often mixed with paraffin or other agents for the purpose of dilution.

The tuberculin methods of treatment are mentioned under the treatment of tubercular disease generally, see page 131. Koch's original tuberculin, tuberculin T.R., Béreneck's tuberculin, Marmorek's serum, etc., have all their supporters. The present position is that in chronic cases, and perhaps very early cases, tuberculin is advantageous if carefully used, and if pronounced reactions are avoided. The founding of so-called Tuberculin Dispensaries is perhaps the best testimony to the value of tuberculin in suitable cases. It should not be lost sight of that bovine tuberculosis may be present in the human subject, and in these cases bovine tuberculin should be used.

Counter-irritation deserves continuous and careful attention. Iodine may be painted on over the affected side of the chest either the tincture, or a mixture of tincture and liniment, depending on whether the patient's skin is susceptible or not to the drug. Fly-blisters are also valuable, and sometimes the use of a mustard-leaf helps to relieve pain and cough.

Cough is often troublesome, and may require sedative cough mixtures. Perhaps one of the best is the Brompton mixture, which consists of dilute hydrocyanic acid ℥ 3, dilute nitric acid ℥ 10, liquor morphinae ℥ 5-10, syrup of tolu ℥ 1, and acid infusion of roses *ad* ½ oz. Such a remedy is of special value at night, so as to soothe irritation and aid in inducing sleep; and there are endless prescriptions of different kinds which are suitable for the alleviation of cough. It should be remembered that gargling the throat with a little cold water or drinking a cupful of warm milk or weak tea, frequently checks the most distressing bronchial irritation.

The *pyrexia* requires careful attention and the remedy almost universally given is quinine, but the necessity for it may be greatly lessened if cold sponging is resorted to, should the temperature rise above 102° F.; and, in fact, the sponging of a phthisical patient at night, not necessarily with water

which is absolutely cold, renders him much more comfortable, and frequently prevents the temperature from reaching a high level. On no account should the usual antipyretic group of remedies be employed unless after mature consideration. They tend to increase the amount of sweating, and in many cases probably do more harm than good.

The excessive *sweating* may demand special treatment. Probably atropine in doses of $\frac{1}{100}$th gr. to $\frac{1}{50}$th gr. is the best remedy in most cases. Quinine is also of value, but it is important to remember that, if the temperature be kept within bounds, the sweating is greatly diminished. Every phthisical patient should be clad in wool during the night, because, even in the mildest cases, there is apt to be greater perspiration than in health.

Should *hæmoptysis* occur, an ice-bag may be placed over the chest, and, if possible, over the bleeding point, while morphia and the mineral acids are valuable in soothing nervousness and excitement. Keep the patient absolutely at rest and on low diet. There are many remedies which may also be given, such as turpentine (℥ 10-20), hamamelis tincture (℥ 20-60), ergotin (gr. 2-5), but the best of all is calcium chloride or lactate in 20-grain doses 3 or 4 times a day. A purge is often of service in lessening the tendency to a recurrence of the bleeding.

Where the intestine becomes secondarily involved, *diarrhœa* frequently results. Its treatment is mentioned under enteritis, but a useful preparation is the lead and opium pill.

The clothing during the day should be warm without being excessive; many consumptives wear an abnormal number of woollen garments, and this should be discouraged, because it is of the greatest importance to harden, rather than to coddle, phthisical patients. Exercise must be graduated according to the patient's strength. It is advantageous to perform pulmonary gymnastics, but the expansion of the lungs is certainly better carried out by gentle exercise than by other means, and if the patient is living in a suitable climate, he should be able to take walking exercise nearly every day, and in bad weather pulmonary gymnastics could be substituted.

The subject of sanatoria is one which has given rise to much discussion; many patients improve greatly in these institutions, because in them they have the advantage of open

air and suitable feeding, together with other treatment; but there is little reason why the patient, if living in the country, may not be treated on the same lines at his own home, provided always that home is in good surroundings. On the other hand, the sanatoria situated at high altitudes are of undoubted advantage, and those at Davos Platz, Arosa, and many other places are arranged on specially hygienic principles. Excitement of all kinds is undesirable, and a quiet, well-regulated life should be enjoined, with freedom from overheated rooms and from excessive exercise, such as dancing, and even skating, but each case must be judged and treated on its own merits.

XII. PNEUMONOCONIOSIS

The term includes the group of diseases which are the result of the inhalation of dust: the commonest of these are anthracosis (coal-miner's lung), chalicosis or silicosis (stone-mason's lung), and siderosis (steel- or iron-grinder's lung).

Etiology. Dust particles of different kinds cause irritation. Normally such particles are taken up by the leucocytes and mucous corpuscles, and, aided by the ciliated epithelial cells, should be expelled in the mucus by the act of coughing. When the inspired dust is in excess of the protective capacity, then this disease commences, varying in character according to irritating qualities of the dust. These particles of dust may reach the air vesicles, and entering the lymphatics pass to the peribronchial lymphatics, or by lymphatic channels running along the interlobular septa they may reach the pleura, while many of the particles are also found in the lymph glands at the root of the lung. Probably steel, iron, and stone dust are among the most irritating particles, and, where present in large quantity in the lymphatic system, they are liable to cause ulceration, but the degree of ulceration depends on the irritating nature of the dust in question. In this way, although coal-dust is by no means a desirable substance in the lungs, it may cause no ulcerative lesion at all; on the other hand, in marked cases of siderosis or chalicosis ulceration is the rule, and, very frequently, the affected lung becomes secondarily involved by the tubercle bacillus.

Pathological Anatomy.—The process is really an inter

stitial pneumonia, with the distribution of the dust particles along the line of the peribronchial lymphatics and the interstitial tissue of the lung. It seems probable that where the mucous membrane of the bronchial tubes remains intact, no serious degree of pneumonoconiosis can result. There are many nodules scattered throughout the lung in the line of the interstitial septa and the peribronchial lymphatics. Depending on the irritating quality of the dust there is a greater or less degree of catarrhal inflammation, and in the case of stone or steel dust there is often ulceration and cavity formation. Stone-mason's lung is peculiarly common amongst workers who use covered sheds, or where the kind of stone being cut forms a very fine and irritating dust.

The **Clinical Features** depend greatly on the irritant qualities of the inhaled dust. Cough and gradual loss of health, with the formation of cavities, are the chief clinical features. Frequently particles of the dust in question may be seen under the microscope in the sputum along with elastic tissue, the evidence of disintegration of lung. Haemorrhage is not infrequent, and a varying amount of dyspnœa develops as the disease progresses.

Diagnosis.—The history of the patient's occupation, or previous occupation, together with a severe bronchitis and the physical signs of cavity formation, should suggest one of the more serious forms of pneumonoconiosis. In coal-miner's lung, the so-called black spit is very typical, although there may be little evidence of any ulcerative process.

The **Prognosis** depends on the variety of the disease from which the patient is suffering: the more serious forms are not infrequently fatal, although most probably in many cases death is hastened by the addition of the tubercle bacillus to the pre-existing disease.

The **Treatment** is practically the same as that recommended for chronic bronchitis, and in addition cod-liver oil, iron, and general tonics are of great advantage. The work from which the patient has suffered should be stopped, or means used to prevent the dust being inhaled. In many factories in which dust is almost inevitable, preventive measures are taken to render the dust harmless to the workers. Masks are sometimes worn, and, where possible, the dust is moistened so that it may not become mixed with the air.

It will be readily understood that besides the trades just mentioned, there are many others in which dust may enter the lung; one of these is associated with the dust from grain, in which spores of fungi may not infrequently be found. Apparently the fungus which most commonly causes trouble is the Aspergillus fumigatus, and the condition has been called *aspergillosis*. There are two trades in which aspergillosis may occur, namely, that of workers among human hair who use rice dust, and that of persons who train carrier pigeons, an occupation which is largely carried on in Paris, and by a method which is peculiarly deleterious to those employed at it. Grains of barley or rice are held between the lips, and the pigeon is taught to take the grain, but, unfortunately, spores of the fungi are not infrequently found adhering to many of the cereals, and as a result the trainer may get the spores introduced into the lung. The spores set up an excessively acute destructive process, closely resembling tubercular disease of the lung, but the prognosis is by no means so unfavourable as in that condition.

XIII. CIRRHOSIS OF THE LUNG

(Fibroid Phthisis)

Great increase of the fibrous tissue of the lung with consequent invasion of air vesicles. It may be a diffuse condition, or localised to a limited part of the lung previously involved by tuberculosis, pneumonia which has not properly cleared up, or any other disease which has, directly or indirectly, resulted in increased formation of connective tissue.

Etiology. (1) The most common cause of fibroid lung is certainly chronic tubercular disease. (2) It may in rare cases be secondary to lobar pneumonia, where the affected lung is replaced by fibrous tissue, forming a sequel to that disease. (3) It may be secondary to chronic bronchitis, with great thickening of the bronchial walls. In this there is gradual involvement of the air vesicles, either in one lobe or possibly in a whole lung, and it is a frequent result of chronic bronchopneumonia. (4) As a sequel to pneumonoconiosis, cirrhosis of the lung may occur. (5) Not infrequently, where there is pleurisy with thickening of the affected pleura, strands of

fibrous tissue penetrate the lung. 6 Syphilis of the lung causes a variety of cirrhosis, generally limited to the interstitial septa near the root of the lung.

It should also be added that wherever the lung, or a part of a lung, is interfered with, either by tumour or other cause, cirrhotic changes occur in the affected area, the amount varying, however, in different cases.

Pathological Anatomy.—Where a whole lung is affected, there is marked flattening of the corresponding side, with nearly always a definite degree of spinal curvature; the pleura is generally greatly thickened, and the air vesicles are encroached upon by the thickened fibrous septa. There is considerable dilatation of the bronchi, forming bronchiectatic cavities of varying size, and in tubercular cases there may be much ulceration associated. Neighbouring organs are drawn over towards the affected side, and generally, if one lung alone is involved, the other shows marked emphysema. Where the cirrhosis is secondary to broncho-pneumonia the areas are scattered, and may be separated by emphysematous lung. Occasionally small aneurisms are present on branches of the pulmonary artery in one or more of the cavities, and if there is much pus formation, waxy degeneration may be present in the liver, spleen, kidney, intestine, and possibly other organs.

Clinical Features. There is always a prolonged history leading up to the cirrhotic changes in the lung, which are very frequently of tubercular nature. In these cases the clinical features are those of chronic tuberculosis, in others there may be more thickening of the pleura, while bronchiectatic cavities give rise to the usual physical signs associated with them. There may be copious and purulent expectoration, and often great dyspnœa, but the degree depends largely on the pathological changes in the lung and the extent of its involvement. Bronchiectasis is a not infrequent sequel.

Physical Signs.—Inspection.—The immobility of the affected lung, and its marked retraction, are extremely typical. Generally the opposite lung is emphysematous, and frequently, as a result of the disease, there is spinal curvature. *Palpation* may show diminution or loss of vocal fremitus, or sometimes increase (depending on the presence or absence of thickening of the pleura), while on *Percussion* a dull note is obtained, with here and there evidence of the existence of cavities; a definite

tympanitic note is often much interfered with should there be greatly thickened pleura. On *Auscultation* the breath-murmurs are generally distant bronchial, or cavernous the vocal resonance is usually increased, and there may be whispering pectoriloquy while the quantity of the moist sounds depends on the amount of secretion present in the part of the lung under examination.

Displacement of neighbouring organs is always a marked feature, especially where the whole of one lung is fibroid. A fatal result is not infrequently due to exhaustion, sometimes to waxy degeneration, and occasionally to haemorrhage.

The **Diagnosis** is rarely difficult, although it is important to make out a tubercular element in the case, and, generally speaking, tubercle bacilli can be recognised in the sputum.

The **Prognosis** depends much on the degree of the condition and on its nature. If primarily tubercular, or if tubercle has secondarily involved the lung, the prognosis is more grave, but much depends on the capacity of the lung for carrying on the work of life. Bronchiectasis, the superaddition of waxy changes in the kidney, alimentary tract, and elsewhere, and signs of a failing heart add much to the gravity of a case.

The **Treatment** is largely directed to the relief of cough and to an attempt to cause drying up of the secretion, while if the sputum has become fetid, every effort must be made to render it aseptic. It is important to remember, where a patient is sufficiently wealthy to choose his own climate, that a warm, dry health resort, in winter, is beneficial, but that no amount of treatment, medicinal or climatic, can actually cure, in the sense of removing altogether, the condition from which the patient is suffering.

XIV. PULMONARY EMBOLISM AND THROMBOSIS

Pulmonary Embolism, Pulmonary Infarction

Where a clot or embolus enters one of the branches of the pulmonary artery, it causes pulmonary infarction.

Etiology.—It may be due to phlebitis of any systemic vein and the entrance of a clot into the venous circulation. Such a clot may originate from one of the uterine veins in

puerperal septicæmia. In valvular lesions of the heart and especially in mitral stenosis, an embolus may be separated from a clot in the right auricle or ventricle, or from vegetations at the tricuspid or pulmonary orifices. In fracture of the long bones, fat embolism may occur in the lung.

Pathological Anatomy.—A haemorrhagic infarct occurs, blood pouring into the affected area from surrounding vessels, and it may contain suppurative organisms, in which case the area of infarction breaks down and forms an abscess. Limited pleurisy is generally present, and in cases where the infarct breaks down, the abscess may open into the pleural sac.

The **Clinical Features** depend largely on the size of the vessel blocked. Where a large branch is occluded the patient becomes suddenly breathless and may die in a few minutes. When a smaller artery is blocked much depends on whether the infarction does, or does not, contain suppurative organisms. Often a definite area of dulness can be made out over which the breathing is bronchial, and crepitations can be heard. Haemoptysis lasting for several days is generally present, and pleuritic pain with audible friction is common. Dyspnœa may be severe if much lung is involved, and there may be symptoms indicating the breaking down of septic infarcts.

The **Diagnosis** is usually aided by the haemoptysis, the pleuritic pain, and the limited area or areas of dulness.

The **Prognosis** depends on the nature of the infarction. A simple infarction may actually relieve a heart case where backward pressure is extreme and the consequent haemoptysis is free. In septic cases the outlook is grave in the extreme.

The **Treatment** must be palliative.

Pulmonary Thrombosis

This sometimes occurs in croupous pneumonia, pulmonary tuberculosis, tumours, and other pulmonary lesions, and is always of grave import. Gangrene may ensue in some cases, and rapidly kill the patient.

XV. ABSCESS OF THE LUNG

Etiology.—(1) In pulmonary consolidation, whether it be lobar pneumonia, lobular pneumonia, pulmonary infarction, or

tuberculosis, the lung may, as the result of infection with suppurative organisms, and partly perhaps on account of diminution in the vitality of the tissue, break down and form an abscess. In the same group we may include abscess associated with cancer or other new growths, although it should be stated that tumour more commonly causes abscess formation as a secondary result by septic pneumonia, than from suppurative change occurring in connection with the tumour masses themselves.

(2) Pyaemia causes multiple abscess formation, either in the line of the pulmonary artery, where an ulcerative endocarditis specially involves the right side of the heart, or by the terminal branches of the general arteries which supply the bronchi, in which case the suppurative organisms are distributed from the left side of the heart. It should, however, be stated that as organisms, capable of causing suppurative foci, may pass through capillaries, in an ulcerative endocarditis of the left side of the heart, the organisms may readily reach the lung. The focus of distribution may be in other positions besides the cardiac valves; for example, it may be found in the septic clot from a phlebitis or in the uterine wall in puerperal septicaemia.

(3) Pulmonary abscess may also arise from perforation of a neighbouring abscess into the lung. This has occurred in cases of tropical or other abscess of the liver.

Pathological Anatomy.—The abscesses noted above differ greatly in size, position, and clinical features, and they may in certain cases result sooner or later in gangrene.

The **Clinical Features** vary greatly. There may be marked evidences of septic absorption, with a swinging temperature and much sweating, while in other less acute cases the patient suffers comparatively slightly. Not infrequently the abscess discharges by the bronchus or opens into the pleura.

The *Physical Signs* may resemble those of a cavity, associated often with the expectoration of a large quantity of purulent material.

The **Diagnosis** is by no means always easy. The tendency to fetor of the breath and the stinking character of the expectoration, with evident breaking down of lung, should make the physician suspect the presence of an abscess, although the history of each case is of great importance. Sometimes the

development of pleurisy reveals the fact that the abscess is in close proximity to the pleura.

The **Prognosis** in many cases is grave, and in pyaemia death is the only likely termination. There is also the risk of the abscess opening into the pleura and so inducing pyothorax.

The **Treatment** is mainly surgical, and although one hesitates to recommend surgical operations which involve the lung itself, it is the only method likely to result in benefit to the patient, and recent results are most encouraging. Quinine is, perhaps, the one drug which should be given, owing to its effect in counteracting septic absorption.

XVI. GANGRENE OF THE LUNG

GANGRENOUS destruction of one or more portions of lung.

Etiology.—Gangrene depends on the presence of certain putrefactive organisms, associated with a lowered tissue vitality which permits of their growth.

There are three groups of causes:—

1. It may be due, or secondary to the inspiration of food, infective blood, septic discharges from nose, mouth, or larynx, and possibly to the entrance of a foreign body.

2. It may be secondary to some pre-existing lung disease, and especially bronchiectasis, lobar pneumonia, septic pneumonia as from pressure of a tumour on trachea or bronchus, and, lastly, tubercular disease of lung. In croupous pneumonia and pulmonary phthisis, excessive debility and the devitalising influences of chronic alcoholism are important factors.

Thrombosis of a large branch of the pulmonary artery is sometimes associated with the diseases mentioned above, and may be the direct cause of the gangrene.

3. It may spread from neighbouring septic foci situated in or near the œsophagus, stomach, mediastinum, or pleura, and metastatic inflammation, resulting in gangrene, may occur as in pyaemia, the original lesion being a septic uterus, bed-sores etc. Usually in this latter group a septic embolus is the vehicle by which the organisms are conveyed to the lung.

Pathological Anatomy. The lung involved is greenish

brown or blackish, with most offensive smell, and generally there is breaking-down in the centre. It may be limited, or involve a whole lung. The gangrenous area is usually surrounded by a zone of extremely congested, often pneumonic, lung.

Clinical Features.—There is marked depression of vital powers, malaise, and frequently diarrhœa from septic absorption. It may be insidious in onset. Soon the appalling odour of breath and sputum aids in the diagnosis. The sputum separates into three layers. At the bottom of the vessel it is greenish-yellow or brown, and more solid, and contains much débris of lung (elastic tissue), fatty acids, micro-organisms, etc. The middle layer is opalescent, and on the top the sputum is covered with a brownish froth. Haemoptysis sometimes occurs. In rare cases there is less fetor, and the patient's strength is well sustained.

The *Physical Signs* vary greatly; perhaps the evidence of cavity formation is most constant, and moist râles occur in great abundance. The physical signs of the pre-existing disease may also be present.

The **Diagnosis** is usually amply clear, owing to the fetor and the large amount of elastic tissue in the sputum.

Prognosis.—The result is generally death from exhaustion and septic absorption, sometimes with the development of a pyopneumothorax, with consequent and very intense dyspnœa. Very rarely, and then only in cases in which the gangrene is limited, does recovery occur, when a local gangrenous abscess may open and be evacuated externally, or internally into a bronchus.

Treatment.—The patient's strength must be kept up by soups and stimulants of all kinds. Vaporise antiseptics in the room for the sake of both patient and attendants, using carbolic acid, creosote, sanitas, or similar agents. Try intratracheal injections of guaiacol 2 parts, menthol 10 parts, and sterilised olive oil 88 parts (5 i thrice daily). Give quinine, terebene, and remedies which may counteract the toxins, and possibly, as in the case of terebene, may exert a local antiseptic influence, however slight. Sometimes the antiseptic is introduced direct into the lung through the chest wall. Surgical interference has been disappointing, but should always be considered. Treat any symptom which may arise.

XVII. NEW GROWTHS OF THE LUNG

Primary sarcoma is generally met with in the form of lympho-sarcoma developing in connection with the glands at the root of the lung. Such a tumour will cause many pressure phenomena, and frequently pleural effusion; death is generally due to septic pneumonia.

Secondary sarcoma, while it may involve the lungs along with other organs, shows no very characteristic clinical features. The tumours may be cartilaginous in nature, but vary considerably, according to the type of the primary tumour.

Primary cancer of the lung is not so rare as primary sarcoma. It not infrequently develops in the neighbourhood of the root of the lung, probably from some glandular structure in the bronchial wall, and spreads along the bronchi, in time infiltrating the greater part of one lung. Tumours of this kind cause marked stridor from stricture of the bronchial tubes, and very frequently, owing to ulceration, there is a considerable amount of haemorrhage, the bleeding being rather continuous than excessive: the sputum is described as red-currant-jelly or sometimes prune-juice-like in character.

Secondary cancer of the lung is a not uncommon result of a primary tumour of the breast, the stomach, or some other organ. These tumours are often encephaloid, sometimes melanotic cancers (where the eye or skin is the site of the primary growth); usually there are numerous nodules varying greatly in size, some of them large, being about as big as a small orange, and others small, almost resembling miliary tubercles. They may involve the pleura and cause considerable pleural effusions; such pleural effusions are often haemorrhagic.

The **Clinical Features** have been so far referred to, and, as already indicated, they vary greatly, according to the site and nature of the different growths, but dyspnoea, pain, cough, and some definite change in the breath sounds, and often evidence of pleural effusion, may be said to be typical of most cases of malignant disease of the lungs.

The *Physical Signs* also vary considerably: there may be evidence of secondary growths in connection with the ribs,

or there may be dilated veins indicating pressure on the superior vena cava or some of its branches. The percussion note over the region of the affected lung is usually dull, and the stridor in the breath murmurs is very typical where there is partial occlusion of a bronchus.

The degree of general disturbance depends on the nature and distribution of the tumours. Emaciation may be rapid, or the patient may live in moderate comfort for a period of months.

Diagnosis.—Where the primary tumour, whether of the breast, stomach, or other organ, has been diagnosed, the involvement of the lung or pleura is easily recognised. There is no question that the presence of blood in the sputum, so characteristic of primary cases of cancer, should render it imperative to overhaul the lungs with special care, and the investigation of the percussion dulness and breath murmurs at the roots of the lungs may help when difficulty arises. Sometimes the cervical or other lymphatic glands show evidence of secondary growths.

Prognosis.—Death most frequently results from pressure, sometimes from emaciation, and certainly septic pneumonia is the final stage in many cases where the lung condition is mainly responsible for the death of the patient.

The **Treatment** must be purely palliative: the use of opium and attention to the tapping of the pleural sac, when there is much effusion, are the most important indications.

XVIII. SYPHILIS OF THE LUNG

Syphilis of the lung has been mentioned already in connection with syphilis generally: it is therefore only necessary to remind the reader that in congenital cases syphilitic cirrhosis of the lung—so-called white pneumonia or white hepatisation—may occur, and that in tertiary syphilis, gummata are not infrequent near the root of the lung. Further, stenosis of the trachea or bronchial tubes may result from syphilitic ulceration, generally with gummata. The clinical features are not distinctive: the white pneumonia of the infant is often incompatible with life, and the presence of gummata may rather be

guessed at than diagnosed by any special clinical feature. The treatment should be on antispecific lines.

XIX. HYDATID CYST OF THE LUNG

It is rare to find hydatid cysts in the lung: the liver and kidneys, however, being relatively more frequently affected. The cyst may be in the lung or pleura, and may grow to a considerable size. The symptoms are cough, pain in the chest, a varying amount of dyspnoea and sometimes haemoptysis; while there may be definite evidences of the cyst, such as local bulging of the chest wall and a dull note on percussion. Sometimes neighbouring organs are displaced.

The **Diagnosis** may be difficult, but is often cleared up after an exploratory puncture is made, the fluid being generally typical in its appearance, and it sometimes contains hydatid hooklets.

The **Prognosis** is always grave, because other cysts may be present, and suppuration and possibly gangrene may ensue.

The **Treatment** should certainly consist in the tapping of the cyst in the hope that it may not fill up again, but sometimes a free incision is called for.

XX. DISEASES OF THE PLEURA

(1) Acute Pleurisy

Inflammation of the pleura, which may be arrested at what has been called the *first* stage of pleurisy—the stage when fibrinous lymph is alone exuded. In many cases a further, or *second*, stage is reached in which a more serous effusion follows the fibrinous exudation. The *third* stage consists in the removal by absorption of the serous lymph, and practically corresponds in symptoms and signs to the first stage.

Etiology.—In a large number of cases pleurisy is of tubercular origin. At one time too little importance was attached to this association of tubercle and pleurisy, and there seems now to be a greater risk of associating too many cases of obviously non-tubercular pleurisy with the tubercle bacillus.

Pleurisy is a common result of exposure to cold or wet, and as such it may occur in a perfectly healthy individual, although it is more likely to develop in one with a tubercular tendency: it is possible that some of these cases ascribed to cold may be of rheumatic origin.

Pleurisy is also secondary to the following diseases—pneumonia, cancer, abscess, gangrene, and infarction. Not infrequently a pleurisy, which is rather more subacute than chronic, is associated with the terminal stages of Bright's disease, cirrhosis of the liver, and cancer other than that directly involving lung or pleura,—a group of causes, therefore, which may sometimes be toxic in nature, although in most cases the pleurisy is due to infection by an organism.

From what has already been said, it may be gathered that in many pleural effusions definite organisms are present to which the pleurisy is directly due; these include the Tubercle bacillus, Pneumococcus of Fränkel, the Diplobacillus of Friedländer, Streptococci and Staphylococci, and sometimes the Diphtheria bacillus, the Typhoid bacillus, the Bacillus coli, the Bacillus aerogenes capsulatus, and, less frequently, a number of other organisms.

In connection with any or all of these, an effusion may occur of more serous lymph.

Pathological Anatomy.—There is first congestion of the pleural surfaces, followed by the exudation of fibrinous lymph varying much in amount in different cases. The serous effusion is poured out later; it is pale yellow in colour, and may contain more or less flocculent material, and in some cases a considerable amount of blood. On microscopic examination, in addition to numerous endothelial cells there are also leucocytes, and much significance has recently been attached to the variety of leucocytes found in the fluid. Where the majority of the cells are lymphocytes, the pleurisy is believed to be closely associated with a tubercular cause, but in order to carry out the test it may be necessary to centrifuge some of the effusion so as to obtain the majority of the cells present. There is always a comparatively large amount of albumin, much larger than what is found in dropsical effusions. In certain cases the fluid forms a jelly outside the body, and this is common in cases in which it closely resembles in constituent parts the serum of the blood. Bile, sugar, and

sometimes uric acid and cholesterin may be found in the fluid, and when a suppurative organism is present, pus cells are seen under the microscope in enormous numbers. The quantity of serous lymph varies from 60 to 100 oz., or even more. Where there is much effusion, the lung is compressed, and is pushed upwards and backwards unless limited by previous adhesions. Should effusions remain long in position, layers of organised lymph may form on the visceral pleura, and greatly restrict possible re-expansion, and for this reason it is generally unwise to permit a large effusion to remain in the pleural sac longer than two or three weeks.

Where there are extensive effusions, there is always much displacement of neighbouring organs, and the mediastinum, and with it the heart, may be pushed over in an effusion of the left side, so that the apex-beat is discovered to be under the sternum. Such great displacements of heart and vessels not merely cause palpitation and discomfort, but might actually lead to a fatal result, although fortunately this is rare.

When the effusion has become absorbed, the fibrinous lymph on the visceral and costal pleurae may either completely disappear, may organise to a certain extent, or may form adhesions greatly interfering with the action of the affected lung. In these adhesions calcareous deposits sometimes occur, but more commonly great thickening of the pleura results, and an invasion of the neighbouring lung by the connective tissue is also seen along the lines of the interlobular septa.

Clinical Features.—There is generally a chill or rigor with severe pain in the side, although in a small proportion of cases a pleurisy may develop insidiously and without pain. Pleuritic pain is sharp, is often described as stabbing in character, is accompanied by cough, and is much increased on deep inspiration. As a rule it is localised, and its position suggests the site of the pleurisy; in exceptional cases the pain is referred to some distant point, and such referred pain is perhaps specially common in connection with diaphragmatic pleurisy. The temperature is generally found to be high, usually 102° or 103° F., but during the stage of serous effusion it may drop to normal.

The *Physical Signs* of this the *first* stage of pleurisy are usually simple and diagnostic. On *Inspection*, the efforts to

limit respiratory movement on the affected side are very obvious, the breathing being shallow and rapid. On *Palpation*, in certain cases friction fremitus may be made out. *Percussion* rarely yields any obvious increase of dulness during this early stage, and on *Auscultation* the breath murmurs are indistinct, rather from the patient's efforts at limiting movement, than from the fibrinous lymph interfering with the conduction of sounds. The chief auscultatory sign is friction, heard most distinctly at the end of inspiration and the beginning of expiration, greatly increased in distinctness by the patient taking a deep breath, and not altered in character by coughing. These features prevent confusion between friction and moist râles. Pressure with the stethoscope generally increases the friction sound. It should not be forgotten, however, that interlobar pleurisy and diaphragmatic pleurisy may, at all events to begin with, and sometimes throughout their whole course, give rise to no audible friction owing to the distance of the affected surfaces of pleura from the chest wall.

A pleurisy may never pass beyond this first stage, and the application of a blister or other simple treatment often completes the cure in a few days.

Sero-fibrinous Pleurisy. — This is the *second* stage of a pleurisy with effusion, and after the physical signs of a dry pleurisy have been more or less definitely made out, effusion occurs. It is usual to find that the pouring out of a serous effusion causes the disappearance of the pleuritic pain, because it separates the two layers of pleurae from each other, although friction is often still audible at the upper limits of the effusion.

The patient generally lies in bed either on his back or on the affected side. Not infrequently there is a considerable amount of dyspnœa.

Physical Signs. — On *Inspection*, where the effusion is considerable in amount, the affected side is seen to be immobile. It bulges and the lower interspaces are pushed outwards, and with the cyrtometer it is found that in extensive effusions the measurement of the affected side greatly exceeds that of the healthy side. In the epigastric region diminished movement of the diaphragm may be noted, and with it of the abdominal wall on the affected side. On *Palpation*, the diminished

movement is recognised, and also the absence of vocal fremitus. *Percussion* yields a note which is frequently described as wooden, although it is really more bone-like in character. Fluid is excessively resistant, and, with the hammer finger, it is easy to recognise the enormous increase of this resistance. Above the effusion, where the lung is pushed upwards, a tympanitic note is obtained, due to the air in the lung vibrating as one column; the phenomenon, often termed Skodaic resonance, is due to the relaxation of lung tissue. The upper limit of the effusion varies somewhat at different points on the chest. It is generally highest in the axillary region, and is at a somewhat lower level at the sternum, and at a still lower level close to the vertebral column, but a good deal depends on the amount of the effusion, and also on the position of the patient, because fluid in the pleura follows, to a certain extent, the laws of gravity. Posteriorly a triangle of dulness with its apex above can be made out on the *opposite* side from the effusion. It lies close to the vertebral column, and is due to the displacement of the mediastinum by the effusion. This triangular area of dulness has been called *Grocco's sign*. *Auscultation.*—While a friction rub may still be audible, and often is audible, at the upper limit of effusions, the breath murmurs are found to be absent altogether over the greater part of the area of the chest below the level of the fluid. In some cases where the effusion is very excessive the breath murmurs in the interscapular region of the same side may also be in abeyance. The vocal resonance is also lost, although towards the upper limit of the effusion the diminution becomes less marked, and at or near the upper limit a peculiar resonance called *ægophony* can generally be recognised. It is a nasal, squeaking, or punchinello-like sound, described by Laennec as resembling the bleating of a goat, and hence he gave it the name ægophony. It is believed to be due to a thin layer of fluid through which the voice sounds pass.

The physical signs, indicating displacement of heart and liver, should be carefully made out. It is always easy, when in doubt with regard to an effusion, to introduce a suitable hypodermic syringe, and draw off a little of the fluid, which operation has the advantage not merely of making the diagnosis of the presence of fluid certain, but also of permitting

an examination of the cell constituents, and so determining whether the fluid is of tubercular origin or not, and if it is becoming purulent.

As a general rule the fluid will become absorbed in one to two weeks, the level gradually falling, and as the layers of the pleurae come in contact once more, friction, very often excessively coarse, is audible and generally palpable. This has been termed *redux-friction*, and in a favourable case complete disappearance of the pleurisy may eventually result. In children, certainly, this is by no means uncommon, whereas in adults it is perhaps more likely that some evidence of the effusion such as thickening of the pleura will persist throughout life. A leucocytosis in the blood usually develops in the course of an effusion, and albumoses may appear in the urine during the stage of absorption.

Diaphragmatic Pleurisy is only peculiar inasmuch as initial pain is frequently referred to the corresponding shoulder the abdomen is kept extremely rigid, and an effusion may accumulate to a considerable extent before percussion reveals a definite area of dulness.

A *Latent Pleurisy* has been already mentioned; there is usually the history of a chill, but no pain, and the patient may seek relief on account of the dyspnœa from which he suffers upon any exertion.

An *Interlobar Pleurisy* and an *Encysted Pleurisy* may present considerable difficulty. *Interlobar pleurisy* is not infrequently secondary to pulmonary infarction, and pleuritic friction may only be heard some little time after pain and cough have made us suspect the presence of the pleurisy. An *Encysted Pleurisy* implies the existence of adhesions, and it may be necessary to use the needle before the nature of the case is cleared up.

An *Empyema* is not infrequently at first a sero-fibrinous pleurisy, and the reason why the fluid becomes purulent is due to its infection by suppurative organisms. The clinical features of empyema are described separately.

Diagnosis.—The physical signs of an effusion are sufficiently diagnostic, although where the pleura is greatly thickened there may be some difficulty. Remember, however, that a hypodermic needle can be used as a final and positive test. It is extremely unlikely that a *pneumonic* or *tubercular* con-

solidation can be mistaken for a pleural effusion, and it is only in rare cases that bronchial breathing and increased, in place of diminished or absent, vocal resonance are associated with pleural effusions. Where a *bronchial tube is blocked*, although the breath sounds are absent and the vocal resonance greatly diminished, it is unlikely that the percussion note will be so dull or the resistance so great as over a pleural effusion. The cause of the effusion is often elucidated by an investigation of the cells, by the amount of albumin, and sometimes by the presence of blood. A *hydatid cyst* in the lung may closely resemble a pleural effusion, and a similar difficulty may be associated with a hydatid cyst in the right lobe of the liver. Exploratory puncture should, however, yield definite data from which a diagnosis can be made.

Prognosis.—Generally speaking the prognosis is good; the only difficulty is the possible tubercular element in the case, and any other causal factor the significance of which may in itself be grave. Thus the pleural effusions associated with cancer are invariably of grave import.

Treatment.—For the first stage of pleurisy, or for cases of dry pleurisy, counter-irritation is probably the best possible mode of treatment. The application of one or two small fly-blisters, perhaps 1 in. × 2 ins., or painting on liquor epispasticus or strong iodine, and the simple but less efficacious mustard leaf, are excellent modes of combating the condition. Where there is much pain and irritating cough, fomentations or poultices may be preferred. Strapping the side with plaster so as to limit movement, and the application of a bandage, are often used where pain and cough are very severe. The ice-bag has been much commended, but it is more applicable in pneumonic pleurisies than in those due to most other causes.

As in every case of inflammation, a purgative forms a useful part of the treatment: perhaps a dose of salts is more suitable than anything else, while diuretics and, where necessary, diaphoretics may also be given. Should the cough be unusually troublesome, powerful sedatives are called for, and even the use of morphia should be entertained, if the cough prevents sleep. It is wise also to study any constitutional element which may be present, and hence the salicylic acid group should be given in cases with a rheumatic history.

Where an effusion is present, it is important to endeavour to secure its absorption as rapidly as possible and this should be done without tapping in the majority of cases. Purgatives, diuretics, diaphoretics, and limiting to a certain extent the amount of fluid drunk by the patient, will all be found of value. On the other hand, it should be remembered that an effusion must on no consideration be allowed to remain in the chest longer than two or three weeks, unless by the end of that time absorption has commenced. It is not necessary to remove the whole or even a large amount of the effusion in order to induce more rapid absorption. If there is the slightest suspicion of the fluid becoming purulent, the tapping should be done promptly and thoroughly, although it will probably prove to be insufficient.

Paracentesis.—Tapping should be performed in the 7th or 8th interspace, and either in the posterior axillary line, or better, slightly nearer the spinal column. Care should be taken that the needle puncture is below the level of the effusion and an attempt may be made to widen the interspaces by making the patient stretch over the arm on the side to be tapped, towards the opposite shoulder. Just before introducing the needle, tell the patient to take a deep breath. Potain's aspirator is probably the most suitable instrument: the needle and cannula should be thoroughly sterilised, while the skin at the site of the puncture should be washed first with soap and water, and thereafter with absolute alcohol, or should have a carbolic poultice placed over it for some hours before the operation is performed.

Holding the needle in the right hand, the operator should place the thumb of the left hand on the upper margin of the lower rib of the interspace selected, and then, before making the puncture, should pull upon the skin so as to make the opening valvular. The needle should be thrust boldly forwards until it passes through the pleura and into the fluid, when all resistance suddenly ceases. The fluid should be *slowly* aspirated into the bottle connected with the instrument, care being taken not to allow the end of the cannula to irritate the lung by rubbing against it, and thereby setting up troublesome cough. It is generally unwise to remove more than 50 to 70 oz. from one pleural sac, inasmuch as the removal of fluid permits of considerable cardiac excitability and over-action: it is therefore

prudent in every case, where a considerable amount of fluid has to be tapped, to bandage the chest *during* the operation, the bandage being gradually tightened while the fluid is being withdrawn.

The dangers connected with paracentesis are slight if the operation is carefully performed. Pneumothorax and subcutaneous emphysema are extremely rare, and generally are due to carelessness, while it is very unusual to find that even a puncture of the lung causes much escape of air into the pleural sac. Severe coughing of a paroxysmal nature is due either to the two pleural surfaces rubbing against each other, or to the cannula scraping against the visceral pleura; such coughing is only dangerous when it induces severe haemorrhage. Syncope is by no means uncommon, and should be guarded against by administering a small dose of alcohol to a weakly patient before, sometimes during, and always after the operation. Ether, in a hypodermic syringe, should also invariably be kept in reserve in case of need. Epileptic seizures have been described, but are very rare. Albuminous expectoration has been recorded as a sequel to paracentesis. It really constitutes a form of oedema of the lungs, causing intense dyspnoea, and may prove fatal; fortunately it is of rare occurrence.

After tapping, renewed and vigorous attempts at counter-irritation should be carried out, and much benefit will result from persistent painting with iodine over the affected side. Pulmonary gymnastics are certainly of great value: the patient may be made to blow water out of one bottle into another by means of a simple arrangement of glass and rubber tubes as in a Wolff's bottle, or he should simply take a specified number of deep breaths several times a day.

(2) Empyema: Pyothorax

Pus in the pleural cavity.

Etiology.—(1) It may result from a simple pleurisy with effusion, as the result of infection by a pus-producing organism. In many so-called sterile empyemas the cause of the condition is pulmonary tuberculosis, although few tubercle bacilli may be discoverable in the pus. (2) Empyema is a common complication of certain fevers, especially in children: in them

pleural effusions in scarlet fever, measles, and less frequently in other continued fevers are apt to be purulent. (3) Although pneumonia is a continued fever, it is better to keep its association with empyema distinct from the preceding class, because it frequently occurs in adults as well as in children. Probably a large number of cases of empyema are really associated with an unrecognised pneumonia. (4) Empyema is also the result of infection either from without or within; from *without*:—a fractured rib or an incised wound, malignant disease of the œsophagus, stomach, or some other organ communicating by ulceration with the pleural sac; from *within* the lung:—malignant tumours, abscesses, tubercular or pyæmic, and necrotic infarcts may give rise to empyema.

The bacteria associated with empyema are the following: so-called sterile empyemas are probably tubercular, and in many cases the Pneumococcus of Fränkel is found in pure culture, while Streptococci, Staphylococci, and other less common pus-producing organisms may be met with.

Pathological Anatomy.—The purulent discharge has a peculiar greenish-yellow colour with a heavy, sweetish odour unless in cases where, from infection by putrefactive organisms, it becomes fetid or stinking. There are erosions on the affected surfaces of the pleurae, and there may be evidences, post mortem, of the different ways in which pus seeks exit, if an operation has not been performed. There is marked collapse of the affected lung, which may be bound down by adhesions rendering any great degree of re-expansion extremely improbable.

Clinical Features.—The phenomena described under pleurisy with effusion are closely simulated where the effusion is purulent, but the following differences are worthy of note:—

There is frequently a swinging temperature which suggests pus formation, and sweating is common. There is almost invariably a leucocytosis in the blood, and there is apt to be more malaise, although not necessarily any greater amount of coughing and pain than with a non-purulent effusion.

Physical Signs.—The skin over the affected half of the chest is sometimes œdematous, and the degree of bulging of the interspaces may be very marked. The upper limit of

the effusion does not curve downwards from the axilla, but is usually horizontal. In children, tubular breathing is occasionally heard over an extensive purulent effusion, but the percussion note is just as dull and bone-like as in a more serous effusion. The dislocation of the heart and mediastinum, and of the liver, where the effusion is on the right side, is more marked in cases of empyema. Rarely does an empyema pulsate in consequence of the communication of the cardiac contractions to it, but a pulsating empyema is more commonly met with than a pulsating effusion which is non-purulent.

An empyema may undergo the following changes:—

1. Absorption of the fluid may occur, when extensive calcareous deposition generally results.

2. It may perforate in the following ways:—

(*a*) Very frequently the pus burrows outwards, either in one of the interspaces, frequently about the 5th or 6th, or it has even been known to make its way along the psoas muscle into the iliac fossa, simulating a psoas abscess. Such external perforation has been called *empyema necessitatis*.

(*b*) An empyema may either perforate into the lung by one or two considerable openings, or it may soak into the lung as the result of necrotic changes in the pulmonary pleura.

(*c*) An empyema may open into the stomach, peritoneum, or pericardial sac, and may thus cause disastrous results.

Diagnosis.—The hectic temperature, a great increase of leucocytes, more than we should expect in a simple serous effusion, and greater displacement of neighbouring organs, suggest the probability of an empyema, while its relative frequency in children who have scarlet fever or measles is also an important point. The removal of a little of the fluid with an exploring needle clears up any difficulty.

Prognosis. An empyema generally results in more or less sinking in of the affected side of the chest. It is decidedly a more grave condition than a simple pleurisy with effusion, and if it is untreated, not merely does it produce permanent collapse of the greater part or the whole of one lung, but the burrowing of the pus, either internally or externally, may cause serious mischief.

Treatment.— It is a difficult matter to urge resection of ribs as the only method of treatment, for in empyema in

children aspiration will, in a small number of cases, be found sufficient to effect a cure. In the great majority of instances, however, resection is necessary, and should not be delayed. Many efforts have been made to cause re-expansion of the affected lung by means of valvular tubes; and sometimes, by the use of a head of water fixed above the patient, and connected by a T-tube both with the patient's chest and with a bottle placed on the floor, an attempt has been made to aid re-expansion, and, further, by making the patient take deep inspirations to help the passage of the water from the reservoir above to the bottle placed below. The necessity for the irrigation of the cavity with antiseptics depends upon whether the fluid is fetid or not. It should never be undertaken without careful consideration owing to the risks involved. One extremely important point is the risk of death during the administration of the anaesthetic in empyema; as a rule, it is the result of carelessness in turning the patient over on to his healthy side, thereby interfering with the free entrance of air into the only serviceable lung. In many cases resection is performed under a local anaesthetic.

During the later stages of recovery after operation pulmonary gymnastics are of very great value.

(3) Chronic Pleurisy

Many cases of chronic pleurisy result from the more acute affection; sometimes they are associated with a considerable amount of effusion; in other cases there is no effusion, but firm pleuritic adhesions develop, and may eventually cause great thickening of the pleura. This latter condition is one mode of origin of cirrhosis of the lung.

It is unnecessary to refer specially to chronic pleural effusions, and the phenomena associated with a thickened pleura are generally those present in fibroid or cirrhotic lung. The flattening of the chest on the affected side, the diminished vocal fremitus, the presence of a dull note, almost suggestive of effusion of fluid, the absence of breath murmurs, and the absence or diminution of the vocal resonance, are the chief physical signs of thickened pleura. It should also be remembered that thickened pleura with cirrhotic lung causes traction on neighbouring organs towards the affected side.

Occasionally in cases of chronic pleurisy affecting the region of the right apex, the subclavian artery may be compressed by the adhesions, and a pulsus paradoxus develops in the right radial artery, while sometimes vasomotor phenomena, such as the flushing of one cheek, local sweating, or dilatation of the pupil, may indicate involvement of the cervical or dorsal sympathetic.

(4) Hydrothorax

Dropsical effusion into the pleural sac.

Etiology.—It is a result of backward pressure, often of cardiac or pulmonary origin, while local pressure on vessels may also cause the condition. Obstruction of the portal circulation may bring about hydrothorax as well as ascites, and pressure on the thoracic duct sometimes gives rise to a chylous effusion. In Bright's disease, in malignant disease of the lung or pleura, and in blood diseases, especially of the cachectic type, dropsical effusions may develop.

Pathological Anatomy.—The fluid transudes; there is sometimes slight thickening of the pleurae, but not to any marked extent, unless the effusion has been of long duration. The fluid contains much less albumin than does an inflammatory effusion; it is straw-coloured, unless it contains much chyle, when it becomes milky, and it varies greatly in amount from 2 or 3 to over 100 ounces. It is usually to be found at the lower parts of the pleural sac, if pleuritic adhesions are not present.

Clinical Features.—In cases of backward pressure of cardiac or pulmonary origin, and in cases of Bright's disease, there is generally very evident dyspnoea, rendered much more marked by the presence of an extensive hydrothorax, and the dyspnoea is dependent on the amount of the effusion. Pyrexia, if present, is not the direct result of a hydrothorax alone.

The *Physical Signs* resemble those of pleurisy with effusion except that the displacement of neighbouring organs is generally not so great, and it is said that the upper limit of the effusion forms a straight line in place of the curve, higher in the axillary region, which was described as being typical of an inflammatory pleural effusion. Too much importance must not, however, be attached to these points.

The **Diagnosis** depends partly on the recognition of the cause of the condition, and partly on the examination of the

fluid. Evidence of dropsy elsewhere, both in the lung itself and in the dependent parts of the body, is usually noted.

The **Prognosis** is often grave, because dropsical conditions frequently point to a causal factor, the treatment of which presents great difficulties, and in too many cases the benefit obtained is apt to be purely temporary.

The **Treatment** should be directed towards the removal of the causal condition, and the usual treatment for dropsy generally should be carefully carried out. This includes the use of purgatives, diuretics, diaphoretics in certain cases, and sometimes limiting the consumption of fluids by the patient. The removal of part of the effusion should be attempted in most cases by paracentesis. Occasionally venesection is indicated, but only in cases in which the right heart is tremendously engorged.

There is no special treatment for chylous effusions; in many cases it implies the presence of malignant disease.

5. Haemothorax

Haemorrhage into the pleural sac.

Sometimes an inflammatory pleural effusion contains an excessive amount of blood, and this may occur in patients who are the subjects of haemophilia, or rarely in tubercular disease. It is more commonly the result of trauma, owing to the lung being torn by a fractured rib, or of aneurism, where rupture has occurred into the pleural sac. It is an occasional and somewhat serious sequel to paracentesis, where a large blood-vessel has been punctured.

The **Clinical Features** are those of pleural effusion, and the **Prognosis** depends greatly on the nature of the case. The **Treatment** is sometimes directed to an attempt at arresting the haemorrhage, and for this purpose ergotin and the free use of ice are often of considerable help. Absolute rest should be enjoined, and in traumatic cases resection of the ribs and clearing out the clots have been followed by excellent results.

(6) Pneumothorax and Pyopneumothorax

Air or gas in the pleural sac, in many cases associated also with the presence of fluid often purulent.

Etiology.—(1) A perforating wound of the chest, as by a spear or sword thrust, or a bullet wound, may permit air to enter the pleural sac, and we may include in the same group cases in which, from cancerous or other ulceration, the oesophagus or stomach becomes connected with the pleural sac. (2) In many traumatic cases the visceral pleura is torn by a fractured rib, and so permits of the escape of air. Cavities of tubercular origin, if situated near the pleural sac, not infrequently ulcerate into it, forming a common non-traumatic cause of pneumothorax. In cases where bullae or blebs occur in emphysema, one or more of these may give way and cause a pneumothorax, which, unless associated with pleuritic effusions, may become generalised. (3) In a small proportion of cases gas forms in the pleural sac, and generally in connection with exudations previously present. The organism usually responsible is the Bacillus aerogenes capsulatus.

Pathological Anatomy.—A pneumothorax may be limited by adhesions, and this is frequently the case where tubercular cavities ulcerate into the pleural sac. The opening into the pleural sac, if made by a fractured rib, or by a dagger or sword thrust, may cause a valvular opening, so that the air can freely enter the pleural sac from the lung, but cannot escape during expiration, with the result that the pressure in the sac may rise far above the pressure of the external air, and cause alarming dyspnoea and great displacement of the mediastinum and heart, and also of the diaphragm. In many cases in which fluid and air are present together, the fluid is purulent, and the condition is termed pyopneumothorax. This is a condition of the pleura similar to what obtains in empyema, with, in addition, an opening either internally into the lung, or externally, by which opening the air has entered.

The lung collapses, the extent depending on the effusion, and in cases of pyopneumothorax the affected lung may not merely be collapsed, but is often bound down by firm pleuritic adhesions.

Clinical Features.—Sudden severe pain, with increasing dyspnoea, indicate the development of a pneumothorax, and even where it is limited in extent, as it may be in cases due to a tubercular cavity, the dyspnoea present is suddenly increased,

the patient sits up in bed panting for breath, cyanosis becomes marked, and the pulse feeble and rapid.

The *Physical Signs* of a case of pneumothorax involving the whole or a great part of one side of the chest are very characteristic. On *Inspection* there is great distension, with immobility of the affected side, the lower interspaces being widely dilated, and the diaphragm depressed. On *Palpation* the vocal fremitus is diminished or lost; on *Percussion* the note is tympanitic, and where there is an opening into the lung or externally through the chest wall, a very forcible stroke may elicit a somewhat cracked-pot resonance. Where the pressure rises to a very great extent in the cavity, the note got on percussion may become almost dull. *Auscultation* reveals loss of breath murmurs, and sometimes of vocal resonance, while consonating râles are only heard where moist râles are present in the lung, and are fairly near the stethoscope.

There are three phenomena which are typical of pyopneumothorax.

(1) *The coin test*, or *bruit d'airain* described by Trousseau, is obtained by tapping one coin used as a hammer on a second coin placed on the chest over the cavity, and at the same time auscultating over the cavity: the ringing sound is got where the coin-tapping and the stethoscope are *both* over the air-containing space. By this means the cavity may be sharply mapped out, and more accurately than by percussion alone. The coin test is also applicable to large cavities in the lung which are near the chest wall and of sufficient size. For the sake of practice the student is advised to carry out this mode of examination on the normal stomach.

(2) *The metallic tinkling of Laennec* is typical of a cavity in the lung, as well as of a mixture of pus and air in the pleural sac. It is believed to be due to the bursting of small bubbles of air in the cistern of fluid: it is sometimes got by shaking the patient, and it has been suggested that after the patient has been so shaken, drops of pus may fall from the roof of the cistern into the fluid below, and thus give rise to the tinkling sound described by Laennec. The characteristic tinkling depends on the production of over-tones in the cavity.

(3) *Hippocratic succussion* is got by violently shaking the patient, and at the same time applying the ear or stethoscope

to the affected side, when splashing of the fluid is audible. This phenomenon cannot be got in an ordinary pulmonary cavity.

In a general pneumothorax the enormous displacement of the heart and the diaphragm must not be forgotten; this displacement is not so great where there is a free communication between the outer air and the pleural sac.

The **Diagnosis** is easy unless the pneumothorax be very limited, when it may be impossible to discriminate between a large superficial lung *cavity* and a limited pneumothorax. There are certainly cases of *subphrenic abscess with air or gas superadded* which may require care in diagnosing from a pyopneumothorax, but the history suggests perforation of stomach, appendix, etc., and although the diaphragm is pushed upwards vesicular breathing will be heard as far as the lung extends downwards.

The **Prognosis** depends entirely on the cause of the condition. In the case of a fractured rib or an incised wound, or the rupture of an emphysematous bulla, recovery is by no means improbable: the air acts as a splint, and may cause no trouble unless it contains septic organisms. In many tubercular cases a pneumothorax hastens the death of the patient, and cannot be looked upon as other than a very grave complication, death sometimes supervening in a few days or even a few hours after its occurrence.

Treatment. Where fluid and air are present, resection of the ribs should be carried out, the surgical treatment for empyema being resorted to. In the case of a broken rib or an emphysematous bulla it is probably wise to leave the air alone unless the intrathoracic pressure rises too high, when tapping with a fine needle may have to be done to draw off some of the air. Keep the patient at rest, and enjoin shallow breathing, in the hope that the air acting as a splint may hasten the healing of the damaged pleura, after which the air will rapidly become absorbed. Some authorities have suggested that either sterilised air or nitrogen may be used to replace the air already in the pleural sac, but as any harm will have been done already, it seems to be a totally unnecessary interference with the patient. A carefully applied bandage often greatly adds to the comfort of the sufferer, and a scrupulous watch should be kept for the first appearance of subcutaneous

emphysema, which, however, may not require any special local interference.

Pain may call for the use of morphia; otherwise careful dieting alone is sufficient in most cases.

(7) Tubercular Involvement of the Pleura

Wherever there is extensive tubercular disease of the lung, there is always a tendency for the appearance of tubercles on the pleura, and in cases of miliary tuberculosis it is usual to find the visceral pleura almost as markedly affected as the lung itself. In some cases of miliary tuberculosis involving the pleura a haemorrhagic effusion is present.

It may be stated again that in many cases pleurisy with effusion, whether sero-fibrinous or purulent, is of tubercular origin.

(8) Tumours of the Pleura

In many cases of tumours involving the lungs, the pleura suffers to a greater or less extent, and not infrequently the effusions, sometimes haemorrhagic, are associated with such pleural involvement. It is not common to find the pleura secondarily invaded before the lung. Occasionally a pleural endothelioma is met with.

XXI. AFFECTIONS OF THE MEDIASTINUM

Tumours

Habe's table of 520 cases of mediastinal disease is most instructive, and a large proportion are tumours. Of these, 134 were cancer, 98 were sarcoma, mostly primary, 21 were lymphoma, 7 fibroma, 14 dermoid cysts, and 8 were hydatids.

The **Clinical Features** of an intrathoracic tumour are sometimes very distinctive, but in other cases there may be absolutely nothing of diagnostic value. Dyspnœa, cough, and difficulty in swallowing are common symptoms.

Physical Signs.—On *Inspection* and *Palpation* dilatation of veins may be noted, and especially if either the superior vena cava or the left innominate vein is compressed. In the

latter case the left side of the neck and left arm are oedematous. There is sometimes local bulging and possibly erosion of ribs or sternum. The tumour may pulsate, but is not expansile. It may displace the heart, and very generally interferes with the lungs by compressing one or other bronchus. *Percussion* often yields a definite localised area of dulness, and the note over the sternum is generally dull. *Auscultation* may be negative, or the heart sounds may be well conducted, or the breathing may be diminished, absent, or stridorous. Evidence of a septic pneumonia may point to compression of a bronchus. A pleural effusion is not uncommonly associated, and there may be symptoms of pressure on the oesophagus, or on some of the nerves.

Special care should be taken to examine the cervical glands, and to palpate each rib separately, and also the sternum, as small secondary growths may sometimes be recognised.

The thymus gland, when involved, may form a tumour palpable in the episternal notch, or may erode the manubrium sterni.

Tumours in the posterior mediastinum often cause pressure on the oesophagus and the recurrent laryngeal nerves, and give rise to much dyspnoea and cough.

The **Diagnosis** is obscure in the absence of definite symptoms and physical signs, but it is as a rule easy to differentiate between an *aneurism* and a solid tumour.

The following points are helpful:—The age, usually 35 to 45 in aneurism, at any time of life in tumour; the sex, usually male in aneurism, and either sex in tumour (although more cases do occur in males). Hard work suggests aneurism. There is no tracheal tugging in tumour, and no expansile pulsation. The pressure of an aneurism is a "living" pressure, or one that is ever varying: the pressure of a tumour is constant. Potassium iodide affords much relief in aneurism, but usually no relief at all in tumour. An X-ray examination is often helpful.

The **Treatment** must be purely palliative and on general principles.

Enlargement of mediastinal glands is often of tubercular nature, sometimes abscess formation occurs from tubercular or other causes, and very frequently along with adherent pericardium there is a mediastinitis associated.

DISEASES OF THE URINARY SYSTEM

I. ABNORMALITIES OF THE KIDNEY

(1) Movable Kidney; Floating Kidney; Nephroptosis

Normally, both kidneys are kept firmly applied to the posterior abdominal wall by the peritoneum, by their blood-vessels, and by a considerable amount of fat, which generally surrounds each of these organs. Should the kidney have a definite mesentery (mesonephron), there will be an amount of movement proportionate to the length of that mesentery. It also stands to reason that where a considerable amount of fat has surrounded the kidney, emaciation may lead to corresponding mobility; it is probable that the lifting of heavy weights, the enlargement of the kidney sometimes due to a tumour, and loss of the support of the abdominal wall, such as occurs after pregnancies, peritoneal effusions, and abdominal tumours may lead to a certain amount of mobility of the kidney.

It is well to retain the term "*floating kidney*" for those cases in which there is a well-marked mesonephron, and to describe as "*movable kidney*" cases in which the kidney is palpable, but with no wide range of movement.

Etiology.—Many of the etiological factors have already been suggested. Women suffer more commonly than men, 80 to 90 per cent of all cases occurring in females. The age is generally from 25 to 40 years, and statistics show that the right kidney is more frequently affected than the left, while in about one-sixth of the cases both kidneys suffer equally. Many authorities consider that tight-lacing has much to do with the development of floating kidney in women, and this may explain also its greater frequency on the right

side than on the left, from the relationship of the right kidney to the liver. Neurasthenia is often associated either as cause or effect.

Clinical Features.—In *movable* kidney there may be no symptoms, and the condition may not be discovered until the abdomen is accidentally palpated. In other cases there may be dragging pain in the lumbar region, sometimes described as neuralgic, but the chief symptoms are referable to dyspepsia occurring in a neurasthenic patient. Constipation is frequently associated with the condition. Such movable kidneys are palpable, sometimes with ease, the bi-manual method greatly aiding in the recognition of the lower border of the kidney, and forcible pressure over the organ causes a peculiar sickening kind of pain which is very typical.

In cases of *floating* kidney, while there may be no symptoms, severe attacks of renal pain frequently occur, due probably to twisting of the mesonephron: they may be associated with nausea, vomiting, and even collapse. These attacks have been termed "Dietl's crises," after the writer by whom they were originally described. In these paroxysms the affected kidney is specially tender.

In floating kidney one readily recognises on palpation the kidney-shaped tumour which passes freely downwards below the hand, when the patient takes a deep breath, and the condition is generally easily recognised.

Dilatation of the stomach has been described as being present in a small number of cases of movable and floating kidney.

Diagnosis.—The shape of the kidney, where it is definitely floating, prevents any possible error. The severe attacks of pain described by Dietl and associated with floating kidney certainly resemble *renal colic*, *biliary colic*, and sometimes the pain met with in *cancer*, but the history of the case, and the absence of the typical clinical features due to biliary and renal colic, generally prevent any mistake, and once the pain has subsided the kidney is easily palpated.

The **Prognosis** is favourable in most cases.

Treatment.—In many cases of *movable* kidney it is only necessary to advise the patient to wear a bandage, sometimes with a suitable pad, and to recommend the avoidance of severe physical strain, such as lifting heavy weights.

In *floating* kidney, while sometimes the bandage and pad may be amply sufficient, operative procedure is often advisable. It is better not to excise the kidney, but nephrorraphy, by which is meant the stitching of the kidney to its attachment posteriorly, has proved very successful. For the attacks of pain, morphia, local fomentations, and confinement to bed may be necessary.

There is often a marked neurasthenic factor which demands treatment, not merely for what may be termed the constitutional element present, but also for the accompanying dyspepsia.

(2) Malformations of Kidney

Malformations are, as a rule, unimportant, unless when operative interference, likely to interfere with the functions of one kidney, is under consideration. The following variations in size, shape, number, and situation may be briefly mentioned:

(1) *Size.*—There may be one large kidney practically taking up the duties of both; the second kidney may be absent altogether or extremely small in size.

(2) *Shape.*—A horseshoe-shaped kidney is not an infrequent abnormality; in it both kidneys are fused into one, the junction between the two being generally below. Sometimes the pelvis of the kidney with the ureter is abnormally situated.

(3) *Number.*—As already indicated one kidney may alone be present, sometimes both kidneys are fused together, as in the horseshoe-shaped kidney. Absence of both kidneys is not compatible with life, and need not be considered here.

(4) *Situation.* One or both kidneys may be displaced sometimes even being found situated in the pelvis, but very often there is merely increased mobility of one or both organs the condition described under movable or floating kidney.

Occasionally the ureters are blocked, and interference with a ureter may cause hydronephrosis. Sometimes there is great dilatation of one or both ureters, generally the result of some obstruction. Abnormal arrangement of the ureters, both as regards number and site of entrance into bladder, is rarely of much clinical significance.

II CERTAIN ABNORMAL CONSTITUENTS OF THE URINE

(1) Albuminuria; Albumin in the Urine

Serum albumin and serum globulin are generally present, less frequently albumoses, peptones, etc.

Type 1.—*Functional Albuminuria.*—This is apt to occur (1) after hard physical work, (2) as the result of fright, (3) after cold bathing in some persons, and (4) as the result of certain kinds of dyspepsia. In all of these cases the amount of albumin is small, and in many of them it is only present during a short period of the day. There may be tube casts, and if they are found along with the constant presence of albumin, there is generally a pathological change in the kidney, either in the walls of the glomerular capillaries, or in the lining cells of the glomerular tuft.

Nenrotic albuminuria, which might be included under this type, occurs after epileptic fits, in tetanus, frequently in exophthalmic goitre, and after blows on the head.

Type 2.—*Febrile Albuminuria.*—Albumin is often present in many of the continued fevers, and especially pneumonia, diphtheria, and typhoid, and it is probably due to marked cloudy swelling of the tubular epithelium. The variety of albumin is sometimes albumrose instead of serum albumin or globulin.

Type 3.—*Toxic Albuminuria and Albuminuria due to Changes in the Blood.*—The following are examples of this type:—syphilis, scurvy, purpura, lead and mercury poisoning, and the presence of bile pigment or sugar in the urine. Ether and chloroform, when administered as anaesthetics, sometimes cause a temporary albuminuria.

Type 4.—*Organic Albuminuria.*—By this is meant the presence of albumin in the urine as the result of Bright's disease, definite congestion of the kidney, and sometimes disease of the pelvis of the kidney, or of some other part of the urinary tract.

a) In acute and passive congestion of the kidney albumin is present, the former condition being a stage of Bright's

disease; included in this group are all forms of acute and chronic Bright's disease.

(*b*) In fatty and waxy kidney albumin is present, and also in cases of suppurative nephritis and tumour of the kidney.

(*c*) Very small amounts of albumin are present in primary cirrhosis of the kidney.

(*d*) The urine contains albumin in disease of the pelvis of the kidney, of the ureter, and of the bladder, and in these cases there is almost always pus present.

Albumosuria

The presence of the albumoses has generally little clinical value, but Bence-Jones' body, forming a precipitate with cold nitric acid which disappears temporarily on heating and reappears when the urine cools, is significant of myeloid tumours of bone and is of definite diagnostic value.

(2) Haematuria, or Blood in the Urine

Etiology.—There is a wide range of diseases in which blood appears in the urine. It may be present in many of the malignant types of continued fevers, and sometimes in leucocythaemia. There are numerous *renal causes*, such as acute Bright's disease, acute congestion of the kidney resulting from toxic agents and especially cantharides, turpentine, and carbolic acid, new growths and calculus, and in particular new growths situated in the calyces of the kidney. Sometimes in primary tuberculosis of the kidney, and commonly in cases of renal infarction, haemorrhage occurs; while in Bilharzia haematobia and the Filaria sanguinis hominis, haematuria is the rule, and these diseases constitute common tropical causes of the condition.

From the *urinary passages* blood may also enter the urine, and especially in cases of calculi, bilharzia, and tumour, and reference must be made to villous growth in the bladder which so frequently cause repeated and serious loss of blood. In not a few cases of severe abdominal injury the kidney, ureter, or even the bladder may be damaged, and as a result blood may appear in the urine.

Occasionally what has been termed renal epistaxis occurs:

this is sometimes a vicarious form of menstruation, but is more often really due to calculus, tumour, etc.

Diagnosis.—Much may be learned as to the origin of the haemorrhage by studying the urine and the time when the blood appears. From the *kidney* the blood is intimately mixed with the urine, which is of a smoky appearance. From the *pelvis of the kidney*, as for example where small tumours, frequently of malignant origin, bleed into the calyces or pelvis of the kidney, there is apt to be clotting of blood in the ureter, and consequent passage of small casts with the pains of renal colic. In haemorrhage from the *bladder* the blood is passed with the terminal portion of the urine, and from the *prostate* bleeding is apt to occur at the end of micturition. From the *urethra* blood comes at the commencement of the flow of urine.

Sometimes the examination of the cells present in the urine may yield important information, and especially if these cells should be of a malignant nature. Cystoscopy and the differential examination of the urine from the two kidneys often give important information as to the site of the haemorrhage.

The **Prognosis** and **Treatment** naturally depend upon the causal conditions present, and will be referred to under their respective heads.

(3) Haemoglobinuria

The presence of blood pigment, haemoglobin, or more usually methaemoglobin in the urine, with very few blood corpuscles or with none at all. The urine is reddish-brown or black, and has a thick reddish-brown deposit. There may be débris of red blood corpuscles, renal epithelium, and often a copious deposit of urates, while the urine always contains albumin.

Etiology.—1) *Toxic Haemoglobinuria.*—This is due to poisons which produce rapid destruction of the red blood corpuscles, and these include chlorate of potash, carbon monoxide, carbolic acid, strong mineral acids, such as hydrochloric acid and sulphuric acid, pyrogallic acid, naphthol, muscarine, and sometimes also the toxins of scarlet and other fevers. Exposure to excessive cold, great muscular exertion, a severe burn, and the administration of quinine to malarial patients, have all

been occasionally followed by this condition. It is a well-known fact that the blood of one animal transfused into another may lead to haemoglobinuria, and in the newly born there is an epidemic form associated with jaundice and certain nerve symptoms.

(2) *Paroxysmal Haemoglobinuria.*—This is found especially in Raynaud's disease, and it is reputed to occur also, though rarely, in malaria and syphilis.

Clinical Features.—The spectroscopic test for methaemoglobin is probably one of the most accurate. There are two absorption bands between D and E Fraunhofer's lines, and a deep, much more definite one between C and D.

In the *toxic* type of the disease the patient yawns, is excessively languid, frequently vomits, suffers from diarrhœa, and may pass speedily into stupor and death. Occasionally definite rigors are present.

The *paroxysmal* type is not, as a rule, fatal. It begins much like the toxic type, and often with rigors. There is sometimes vomiting, diarrhœa, and pain in the lumbar region, while abdominal discomfort is extremely common. There may be several paroxysms during the same day, but as soon as the paroxysm has passed off, the urine becomes clear. During the attack there is often frequency of micturition, and jaundice is an occasional sequel.

The **Prognosis** depends on the nature of the case, and, as already stated, the paroxysmal form is not dangerous like the toxic type, unless nephritis is superadded; but it must not be forgotten that tubules and glomeruli may be blocked with blood pigment. Paroxysms may recur for years, and render the patient very anæmic.

Treatment.—Direct the patient to avoid any cause known to produce the paroxysmal attacks. Recommend warm clothing, no exposure to cold, and residence in a warm climate. During the attack order rest in bed, give warm drinks and wrap up the patient warmly, while sedatives such as hyoscyamus, and astringents such as acetate of lead and gallic acid, may be given with advantage. In certain cases haemostatics have been administered, and cold may be applied, in place of heat, over the region of the kidneys. In syphilitic cases give the usual antispecific treatment.

(4. Pyuria; Pus in the Urine

Where the pus is of urethral origin, it appears with the first portion of the urine. Where it is due to cystitis, it is apt to be passed in considerable quantity towards the end of micturition. In cases of cystitis the urine is sometimes acid and sometimes alkaline; in men it is generally the latter, and contains a copious phosphatic deposit: in pyelitis the urine is more frequently acid in reaction. Pus in acid urine remains as a heavy deposit separate from the urine at the bottom of the glass, while pus in alkaline urine forms a ropy deposit similar to what obtains when an alkali is added to pus in a test-tube.

In diagnosis, epithelial elements should be carefully looked for, although tailed cells may come from other parts of the urinary tract than the pelvis of the kidney. The presence of great numbers of vaginal squames cannot fail to be recognised where the pus is of vaginal origin.

(5) Chyluria

Chyluria occurs in cases of filariasis, and chyle is also sometimes found in the urine in cases in which there is no satisfactory explanation for its presence. It is, in these instances, almost invariably mixed with blood.

(6) Glycosuria

There is nearly always a trace of sugar in the urine in health, but it is with its presence in excess that we have to do here. It should be remembered that there are agents, other than glucose, which reduce copper, for a full account of which the reader should consult a text-book on clinical medicine. Glycosuria is not uncommon in stout persons after middle life, and especially where there is a gouty element in the history. A temporary glycosuria is not infrequent after the inhalation of chloroform or ether in certain subjects; it is sometimes present in epilepsy, and sometimes during pregnancy, and very commonly it follows severe injuries to the head. Glycosuria which can be removed by dietetic treatment should not be termed true diabetes mellitus, and care should

be taken to obviate, by careful qualitative methods of examination, mistakes due to reducing agents in the urine, such as alcapton and glycuronic acid, both of which may be present in certain instances.

Other Constituents

Acetone, diacetic acid, and β-oxybutyric acid are all referred to in connection with diabetic coma, and do not require further notice here.

In rare cases of diabetes mellitus, fat has been found present in the urine.

III. NORMAL CONSTITUENTS OF THE URINE, BUT PRESENT IN EXCESS

1. Excess of Uric Acid, Lithuria

The conditions under which uric acid is precipitated are mentioned in connection with uric acid calculi, but it should be remembered that hyperacidity, a low pigmentary index, and poverty in mineral salts may produce a precipitate of uric acid which does not depend on any excess of uric acid in the urine. The reader is advised in connection with this subject to refer to the description of gout and the formation of uric acid calculi in that disease. It is only necessary to add here that some error of oxidation probably accounts for cases in which there is a true excessive formation of uric acid, and that excess in eating and drinking with insufficient exercise goes a long way towards the explanation of lithaemia. Associated with it there is not infrequently primary cirrhosis of the kidney with an arterio-sclerosis generalised throughout the arterial system.

2. Oxaluria

It is doubtful whether oxalates appear in the urine from other causes than the ingestion of oxalic acid with the food, but where there is an excess of oxalate of lime present in crystalline form in the urine, there is nearly always a marked degree of dyspepsia with profound depression of spirits. It is common in gouty persons and is often associated with

a deposit of uric acid. Many subjects of oxaluria are hypochondriacal and neurasthenic.

(3) Phosphaturia

By this is meant not the presence of the normal acid phosphate of soda, but the alkaline phosphates which render the urine alkaline, and which are often precipitated as earthy phosphates. When urine decomposes, the breaking up of urea renders the urine alkaline, but the alkalinity is volatile, because it is due to ammoniacal decomposition. Where, however, the alkalinity is fixed, it is generally due to a true excess of phosphates. This is certainly not uncommon in protracted ill-health, and especially in diseases of the nervous system, although it may be present with any debilitating illness. Cases have been described of phosphatic diabetes in which there is a great increase in the excretion of phosphates, but this is rather an excess of acid phosphates than alkaline. It is a rare ailment, and its exact significance is not clear.

IV. ANURIA

A condition occasionally met with in various morbid conditions, and consisting in arrest of urinary secretion.

1 The Non-obstructive Form

Etiology. Acute Bright's disease, especially scarlatinal nephritis and acute congestion of the kidney, whether associated with local pyogenic organisms or not, may cause suppression of urine. It may occur in the last stage of chronic Bright's disease, in the cold stage of cholera, after any severe shock, as from an internal injury, and also in collapse, while a less grave reflex form of anuria sometimes follows catheterisation.

Clinical Features.—In most cases the symptoms are grave from the first—vomiting, purging, convulsions, and frequent rigors being present, and death may occur in 2 to 3 days, with all the symptoms of toxic poisoning.

Diagnosis and Prognosis.—The passage of a catheter demonstrates the absence of urine in the bladder, while by

careful abdominal examination hydronephrosis and similar conditions must be excluded. Any urine which is passed is small in amount, and loaded with albumin, and usually contains blood and many casts. The prognosis is extremely grave unless prompt relief can be obtained.

Treatment.—The patient should be given a hot bath at once, and hot poultices or fomentations applied over the region of the kidneys, while the skin may be stimulated to vigorous action by hot-air or radiant-heat baths. Sometimes cupping over the kidneys and hot rectal douches are of great value.

(2) The Obstructive Form

Etiology.—This is generally the result of the impaction of a calculus in the ureters, or pressure on them by some morbid growth. Congenital malformation of the ureters, slight at first, may gradually cause increasing obstruction.

Clinical Features.—During the first few days of the obstruction the patient suffers little inconvenience; there is usually a flow, from time to time, of a little pale urine of low specific gravity and containing little or no albumin. By the end of a week uraemic phenomena develop—sickness, gradual loss of muscular strength, and muscular twitchings, while increasing vomiting and diarrhoea indicate the onset of the toxic phenomena which terminate just like the more serious non-obstructive form, unless relief is obtained. There may be a combination of drowsiness with marked delirium, total inability to eat, and profound prostration, and the phenomena practically correspond with those met with in uraemia.

Diagnosis.—It is often possible in hydronephrosis to recognise the presence of a renal swelling indicating the marked distension of the pelvis of the kidney, while the passage of a clear limpid urine in diminishing amount, co-existent with the onset of uraemic phenomena, is very suggestive.

Prognosis.—Death occurs in 9 to 11 days, unless relief is afforded by surgical treatment.

Treatment.—In a case of calculus, an attempt may be made by vigorous abdominal massage (the patient being under an anæsthetic) to displace the stone, while the sedative effects

of a hot bath and of tincture of hyoscyamus may permit of the relaxation of spasm, and so allow the calculus to proceed downwards. Where such treatment fails, an abdominal operation is indicated, as it offers the only chance, in a large majority of cases; but it may be found impossible to give the patient permanent relief in tumour cases.

V. URAEMIA

A TOXIC condition of the blood not infrequently found in chronic Bright's disease, and from which a considerable percentage of patients so affected eventually die. It is also the result of anuria. Unfortunately we cannot state, with certainty, what is the exact nature of the toxic substance or substances. The following theories have been put forward to explain the condition:—

1. *Toxic Agents.*—(*a*) Substances retained in the blood which ought to be excreted by the kidneys; (*b*) Abnormal decomposition in the blood, or possibly in the tissues, of such retained substances; (*c*) The formation of abnormal products of metabolism by the tissues.

The toxic agent does not seem to be urea, because urea can be administered experimentally without the production of uraemia. The blood may contain a considerable amount of nitrogenous extractives, principally urea, without the production of uraemia. Normally there is ·015 per cent of urea in the blood, while in renal disease without uraemia there is ·15 per cent of urea, in addition to a considerable amount being excreted by the kidneys. In acute uraemia the percentage rises to ·45 or ·5 per cent. While, therefore, the urea alone cannot be considered the sole cause of uraemia, the examination of the amount of urea excreted in the urine and the investigation of the amount of urea present in the blood yield definite information with regard to the probable onset of uraemic poisoning. A large amount of urea present in the blood indicates a distinct risk, and a marked diminution of the urea excreted by the kidneys is also associated with a tendency to uraemia. In making such calculations it must be borne in mind that the amount of urea and nitrogenous extractives

excreted by the kidneys varies directly with the dietary, and allowance must be made for a further diminution in cases where the patient is confined to bed.

2. *Œdema of the Brain.*—This is commonly present post mortem, but it is by no means confined to cases of uraemia.

Etiology.—Uraemia may develop in chronic Bright's disease, less frequently in the acute forms, also in cirrhosis of the kidney, in active and passive congestion of the kidneys, in waxy degeneration, in tubercular disease, in cases of calculus, in hydronephrosis, in cystic disease of the kidneys, and in association with suppression of urine where no kidney disease may be present at all. It should be stated that while the diseases above mentioned predispose to uraemia, there is often, in addition, some exciting factor, such as exposure to cold, overwork, imprudence in diet, or, in short, anything which throws a special strain on the renal organs.

Clinical Features. There are various types, each consisting of a group of symptoms which may be described separately, although a combination of these is not uncommon.

Type 1.—*Convulsions.*—These come on suddenly, generally with headache and great restlessness. The convulsions resemble Jacksonian epilepsy, or sometimes an ordinary epileptic seizure, but in place of one, or perhaps two fits occurring, there is a tendency to the status epilepticus: in the intervals between the fits the patient is practically unconscious, or else, in a few isolated cases, he may be highly excited and almost maniacal. Associated with these convulsions are marked twitchings of muscles, seen between the fits, and often indicating that a new seizure is about to occur. Temporary blindness and deafness are sometimes present with uraemic convulsions, while headache, sleeplessness, and alimentary disturbance may be noted in greater or less degree.

Type 2.—*The Maniacal Type.*—In this form there is intense restlessness and mental excitement, with absolute sleeplessness and eventually wild delirium. The attack may develop suddenly, and is apt to be rapidly fatal.

Type 3. *The Dyspnœic Type.*—This type resembles a form of asthma, the breathing, however, being of a peculiarly hissing character. There is marked orthopnœa, and in certain cases the breathing becomes Cheyne-Stokes in character. Sometimes the pupils contract and dilate rhythmically, and the pulse

may alter in speed. Associated with this type there are often some of the other manifestations of uraemia.

Type 4.—The Gastro-intestinal Type. In this type there is great nausea and incessant vomiting, often associated with severe abdominal pain. Hiccough, of a most persistent nature, frequently occurs, and intractable diarrhoea may be present. In certain cases the diarrhoea occurs without any gastric disturbance, but this is rare.

Type 5. The Comatose Type.—With or without preceding delirium the patient becomes comatose. There may be initial cramps or twitchings of muscles or gastro-intestinal phenomena, but with the advent of coma the patient soon dies. Probably this type is one of the commonest, and often constitutes the final stage of an acute uraemia.

Type 6. The Paralytic Type.—In certain cases of uraemia, perhaps more frequently in the chronic than in the acute form, a monoplegia or a hemiplegia develops, while there may be no haemorrhage or other gross lesion in the brain to account for its presence. Such cases are by no means uncommon, and their explanation is as yet unknown.

While uraemia has been described under these different types, many cases show phenomena referable to several or possibly to all of them, and it is well to remember that the most common features of any uraemic attack are the following: gastro-intestinal irritation, headache, uraemic dyspnoea, twitchings of muscles, and a distinct tendency to convulsive seizures, mental excitement, and coma. No reference has yet been made to pyrexia; sometimes there is a marked degree of temperature, although it is possible that it may be due, in not a few cases, to a pleurisy, pericarditis, meningitis, or other complication, rather than to the toxaemia.

Not infrequently a form of uraemia which might be designated *chronic* occurs, and although the symptoms are similar to those just mentioned they are less acute. They consist in headache, twitchings, attacks of dyspnoea, often rather suggestive of asthma and nocturnal in occurrence, restlessness and sleeplessness, sometimes somnolence and stupor, itching of the skin, and a varying amount of nausea, vomiting, and diarrhoea.

Diagnosis. The quantity of urea in the blood is probably one of the safest guides to the diagnosis of uraemia. The following conditions are liable to be confused with uraemia:

(1) Occasionally a *continued fever* may be mistaken for uraemia, perhaps especially *typhus* and *typhoid*, and it may be extremely difficult, unless after a careful consideration of the symptoms and signs, to offer an absolutely certain diagnosis. Unquestionably the presence of an excess of urea in the blood would be an important point.

(2) *Opium poisoning* and *alcoholic poisoning* may be mistaken for the coma met with in uraemia. Generally the presence of albumin in the urine is of the greatest importance, while the odour of the patient's breath may, on the other hand, suggest opium or alcoholic poisoning. The pin-point pupils in opium poisoning and the dilated pupils in alcoholic stupor may both be simulated in uraemic coma.

(3) Uraemic paralysis is sometimes extremely difficult to diagnose from a *monoplegia* or *hemiplegia* the result of an organic brain lesion. The examination of the blood and the urine may help in obscure cases.

If albuminuric retinitis is present, there is positive evidence of kidney disease, and in all doubtful cases an examination should be made of the condition of the fundus.

Prognosis.—Acute uraemia of any type is apt to be fatal, whereas the more chronic forms, although they may eventually lead up to a fatal result, rarely directly cause it.

The duration of life in a patient who has once had uraemia will depend largely on the state of the kidneys, and on the possibility of careful treatment, medicinal and dietetic, but so many cases terminate fatally during the first attack that the prognosis must always be grave.

The **Treatment** is practically that described in Bright's disease in its different forms, especially the use of purgatives and diaphoretics.

For the convulsions, chloroform anaesthesia is often advantageous, and, where the vascular tension is high, an attempt may be made to reduce it by means of nitroglycerine (gr. $\frac{1}{100}$-$\frac{1}{50}$) by careful attention to the free action of bowels and skin, and perhaps above all by a well-regulated and suitable non-nitrogenous dietary. Diaphoresis may be secured by the hot-air bath and pilocarpine administered in $\frac{1}{12}$th to $\frac{1}{6}$th grain dose hypodermically.

VI. ACUTE CONGESTION OF THE KIDNEY

This is an early stage of acute Bright's disease, but it may also be the result of the following conditions:—

1. The action of irritant substances on the kidney, such as excessive amounts of cantharides, carbolic acid, turpentine and copaiba, quinine, and mineral acids.

2. Toxins produced by organisms and excreted by the kidneys, and also the presence of certain organisms themselves. Probably the first stage of Bright's disease is often due to the irritant action of toxins.

3. Active congestion from dilatation of vessels. This is a doubtful cause, but it may possibly occur through the agency of the nervous system as a reflex nerve action, and it has been thought that by this means the renal congestion following catheterisation, and perhaps that following exposure to excessive cold, may be brought about. Experimentally it has been found that stimulating the central ends of the lower dorsal roots produces acute congestion of the kidneys.

Pathological Anatomy. — The kidney is swollen and engorged with blood, the glomeruli are bright red in colour, and there are frequently haemorrhages into them or into the tubules, while the tubular epithelium is swollen, showing cloudy swelling, and may even have reached the further stage of desquamation.

The **Clinical Features** resemble the early stages of acute Bright's disease with the presence of albumin, frequently blood, and generally epithelial and hyaline casts in the urine.

The **Treatment** is directed to the removal of the cause of the condition, and will be referred to further under acute Bright's disease.

VII. PASSIVE CONGESTION OF THE KIDNEY

Etiology.—It may be the result of heart disease, and of adherent pericardium, and it commonly follows as a sequel of such pulmonary conditions as bronchitis, emphysema, and phthisis. It may also be due to obstruction of the inferior vena cava, and may occur in cases of ascites, especially where,

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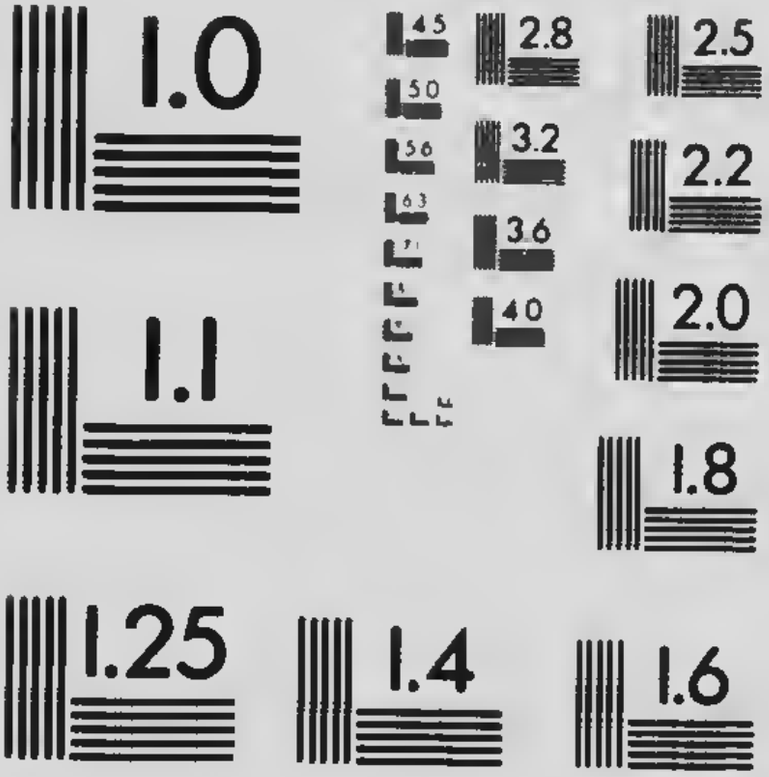

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as the result of cirrhosis of the liver, there is interference with the venous circulation. Malignant growths sometimes infiltrate the renal veins and thrombosis of these veins may occur in debility.

Pathological Anatomy.—The kidneys are large in size and dark red in colour. They are hard and firm, and engorged with blood, and the stellate veins are prominent. Interstitial changes are common in long-standing cases, and may be associated with thickening of vessels and of Bowman's capsules.

Clinical Features. There is often a reduced quantity of urine (10-20 oz.), and it is generally of high specific gravity, of a dark colour, and throws down a uratic deposit on cooling. The albumin varies in amount, being rarely large unless inflammatory changes are present in addition. There are hyaline and sometimes granular casts.

Diagnosis.—Is the albumin simply the result of passive congestion, or is there an inflammatory process present in addition? The albumin is invariably limited in passive congestion, and a large amount of albumin together with epithelial casts would suggest nephritis. An attempt should be made to discover the cause of the passive congestion.

The **Prognosis** depends on the possibility of relieving the backward pressure, and is distinctly more favourable if there is no inflammatory condition present.

Treatment.—Treat the heart, lungs, or other causal factor, and administer hydragogue purgatives and diuretics in almost every case. Much may also be done to afford relief by poulticing or dry-cupping the loins.

VIII. ACUTE NEPHRITIS

(Acute Bright's Disease)

An acute inflammation of the kidney, which may involve especially 1) the glomeruli, 2) the convoluted tubules, or 3) the interstitial tissue. In every case, although one or other of these may be chiefly affected, all suffer.

Etiology.—There are probably *predisposing* as well as *exciting* causes in most instances, and it is difficult to assign to each the due amount of blame.

1. Cold and wet are extremely common causes, and especially where perspiration is suddenly checked, or where alcoholic subjects or debilitated persons are exposed to sudden and great changes of temperature.

2. In some of the specific fevers, especially in scarlet fever small-pox, and yellow fever, and less frequently in typhoid, cholera, and malaria, acute nephritis may supervene as a complication. It may be due to the toxin, and perhaps, in certain cases, to the presence of the organisms themselves where they are excreted by the kidney. Associated with this group is the incidence of acute Bright's disease in cases of septicaemia, pyaemia, erysipelas, and acute tuberculosis.

3. Certain poisons, such as cantharides, turpentine, carbolic acid, and large doses of strong mineral acids, may produce acute nephritis; while lead and probably long-continued and oft-repeated smaller doses of the drugs just mentioned are capable of causing a more chronic form of the disease.

4. Certain substances excreted by the kidney may act as toxic agents: for example, sugar in diabetes mellitus, and bile in permanent jaundice.

5. In pregnancy, acute Bright's disease may supervene, perhaps as the result of some unknown toxin or from special strain being thrown upon the kidneys. The compression of the renal veins by the gravid uterus may be the cause of the congestion.

6. Acute Bright's disease may also follow extensive burns, especially of the trunk, persistent eczema and erythema involving large areas of the skin. Possibly this group depends on the absorption of some toxin or toxins, but it may also be due to reflex nerve stimulation.

7. Acute Bright's disease may result from a renal operation, or from injuries affecting the region of the kidney, and may be due to the introduction of some organism, or in certain cases to congestion from reflex irritation.

Acute Bright's disease is common in childhood and up to the age of 40, but is rare after middle life. It is more frequent in men than in women, the proportion being as 3 to 2.

It is doubtful whether heredity has much to do with a predisposition to Bright's disease, but certain families in successive generations have supplied an unusual percentage of cases. This probably depends rather on inherited weakness

of constitution than on anything else. Undoubtedly debility both of mind and body and intemperance predispose to acute Bright's disease.

Bright's disease is more usual in temperate climates.

Pathological Anatomy.—The appearance of the kidneys varies much according to whether the glomeruli, the tubules, or the interstitial tissue have specially suffered, and the congestion and inflammatory infiltration may be more or less localised to one or other of these structures, although it is often generalised. Where there is much interstitial exudation, there is considerable enlargement of the organ. The kidneys may be congested and engorged with blood, or the cortex may be pale and swollen, while the medulla is red and hyperaemic. The capsule strips freely.

(1) The glomeruli, when they are specially affected, may be seen with the naked eye as bright red points in the cortex. On microscopic examination, haemorrhages are frequently seen within Bowman's capsules, and the epithelial cells covering the tuft proliferate and begin to desquamate, while leucocytes may also be observed in considerable numbers. Sometimes the capsules and the capillaries show hyaline changes in their walls.

(2) The convoluted tubules, where they are chiefly affected, show marked cloudy swelling with commencing desquamation of the renal epithelial cells. The desquamated cells are seen to contain droplets of fat when examined with the microscope, while the nuclei lose their distinctness, and red blood corpuscles and leucocytes may be found inside the tubules.

(3) The interstitial changes consist of an exudation of inflammatory lymph with red blood corpuscles and leucocytes, specially between the convoluted tubules. These changes are frequently more or less localised to the neighbourhood of the larger vessels supplying the cortex, and may vary considerably in amount in different parts of the same kidney.

All toxic cases tend to show marked *glomerular* inflammation. Acute nephritis, the result of scarlet fever, of cantharides, or of carbolic acid poisoning, is a good example of this variety, while exposure to cold and wet produces chiefly *tubular* changes associated with a considerable amount of

interstitial exudation. It must be remembered that these limitations are by no means always adhered to, and that following on glomerular changes the tubules may be secondarily involved, and interstitial exudations may also occur. Swelling of the cortex depends largely on the involvement of tubular epithelium and on interstitial exudations, and in these cases the cortex of the kidneys is frequently swollen but pale. When interstitial exudation has been replaced by the formation of connective tissue, the convoluted tubules are liable to be compressed and local dilatations occur.

In the medulla of the kidneys in acute Bright's disease there is also congestion, frequently haemorrhages, and more or less small-cell infiltration, although it does not suffer to the same extent as the cortex.

Clinical Features.—*Type* 1. *Acute Nephritis, the Result of Exposure to Cold or Wet.*—The onset is generally sudden, oedema or puffiness of the face, and especially of the eyelids, develops within a few hours, and on examining the eyes oedematous fluid may be seen under the ocular conjunctiva, which has been aptly termed the "tear that never falls," and which gives to the eye a peculiar glistening appearance. The ankles may also show dropsical effusion. In some cases there is no dropsy present at all. There is usually pyrexia, and possibly rigors, the temperature reaching 101° or 102° F., while the patient suffers from considerable malaise and general discomfort.

Urinary System.—The urine at first is scanty, although micturition is frequent, and it contains albumin, blood, and hyaline, epithelial, and blood casts; later the urine becomes less "smoky," and contains a varying amount of albumin, often large, with much renal epithelium, and hyaline, epithelial, and granular casts. The urea diminishes to about half the usual quantity. On chemical examination both serum albumin and serum globulin are found to be present, and sometimes albumoses.

Alimentary System.—There is often acute dyspepsia with nausea and vomiting, the appetite is poor, and in certain cases persistent diarrhoea supervenes. One of the serious though rare complications of acute Bright's disease is peritonitis.

Haemopoietic System.—Anaemia develops at an early stage owing to the waste of albumin, and the pale, pasty face of a

patient suffering from acute Bright's disease is most characteristic. Sometimes epistaxis occurs.

Circulatory System.—In 3 to 6 weeks the cardio-vascular phenomena of Bright's disease may develop. There is often, to begin with, increased intravascular pressure of the radial artery owing to vasomotor spasm, the result of toxins circulating in the blood. At a later stage there is definite arterio-sclerosis with hypertrophy of the left ventricle. The complications in connection with this system include pericarditis and sometimes acute dilatation of the heart.

Respiratory System.—Oedema of the lungs is common in Bright's disease, and may occur to a serious extent in acute nephritis, while bronchitis sometimes develops. The complications are pleurisy, not infrequently with marked effusion, and sometimes pneumonia.

Integumentary System.—The skin is dry and perspiration scanty. Sometimes eruptions appear, but they are not typical.

Nervous System.—Uraemic phenomena may develop; certain of these belong to other systems, and have already been mentioned. They are nausea and vomiting, twitching of muscles, severe headache, epileptiform convulsions, amaurosis, and sometimes a very marked dyspnoea not entirely explained by the amount of oedema of the lungs. Retinitis and papillitis are more commonly found in chronic than in the acute forms of Bright's disease.

Type 2. Post-Scarlatinal Nephritis.—While many of the symptoms are the same, the following points are worthy of special note: The onset is more gradual, and convulsions are not infrequently present in the case of children, while the temperature, which had probably fallen to normal, once more reaches 102°, or possibly 103° F. The degree of dropsy varies considerably, but is generally great, and similarly the amount of blood in the urine varies. Uraemia is more frequent in post-scarlatinal than in any other form of acute nephritis. In other respects the description already given under Type 1 applies also to the post-scarlatinal form of Bright's disease.

Diagnosis.—The dropsy, when present, and especially that involving the face, is very characteristic, and if it is associated with a diminished quantity of urine and urea, a large amount of albumin, the presence of blood and of epithelial and other casts, the diagnosis becomes simple. In *passive congestion of*

the kidney there is diminished urine, but the albumin is small in amount. It must, however, be stated that there are insidious cases of the disease in which uraemic phenomena may first suggest the presence of serious nephritis. Cases of acute Bright's disease without the presence of albumin in the urine have been described, but must be exceptional. The nature of the tube-casts offers one of the best methods of diagnosing the variety of Bright's disease from which the patient is suffering.

In pregnancy it is wise towards the later months to examine the urine on several occasions.

The **Prognosis** is often favourable, although the condition may tend to become chronic. An acute nephritis after exposure to cold or wet is more likely to terminate favourably than a post-scarlatinal case. The mortality in young children is high, and $\frac{1}{3}$rd of all fatal cases occurs in them.

The duration of a case of acute nephritis varies considerably, but from 1 to 3 weeks generally elapse before marked improvement occurs, while in other cases it may be 6 or 8 weeks before the urine becomes free from albumin. Usually the dropsy disappears first and the albumin later. In not a few cases, unfortunately, the persistence of the albumin indicates a permanent change in the kidneys.

Treatment.—The patient should be placed in bed between blankets, and a flannel night-dress should be worn. This is desirable, because free diaphoresis forms perhaps the most important method of treatment, and chills must be strictly avoided.

The *Diet* should be limited to milk, and milk alone, and some authorities prefer buttermilk. Many patients require the addition of starchy foods such as arrowroot, sago, etc., but so far as possible meat and fish, even in the form of beef-tea and soups, should not be given. There are certainly a few cases in which a rigid milk or carbohydrate diet proves unsuitable, and to them a small quantity of beef-tea may have to be allowed. Recently it has been suggested that the albumin lost should be replaced by the diet given, and that a moderate amount of proteid does not increase the excretion of albumin. Oranges and lemons may be permitted, and alkaline mineral waters are of great value. As recovery occurs, spinach, cauliflower, and similar vegetables may be added to

the dietary, and bread may be freely eaten, but it is prudent to avoid nitrogenous articles of food until the albumin has disappeared.

The following medicinal measures are useful:—

1. *Purgation.*—Saline purgatives are very suitable, such as magnesium sulphate (ʒ 1-3), Carlsbad salts, Hunyadi Janos, and many purgative mineral waters. Amongst other hydragogue cathartics are compound jalap powder (gr. 20-30), and the pulvis elaterini compositus (gr. 1-4). Do not purge too freely unless uraemia is threatening, because a severe form of enteritis is not uncommon in Bright's disease.

2. *Diaphoresis.*—Hot baths, hot-air baths in which the temperature may be raised from 120 at first to 170 or 180 F., and wet packs (hot water), are admirable methods of treatment. Jaborandi (tincture ʒ $\frac{1}{2}$-1) or pilocarpine hypodermically (gr. $\frac{1}{12}$-$\frac{1}{6}$) are the best drugs to use for stimulating diaphoresis, but it is desirable to investigate the amount of pulmonary oedema present before giving pilocarpine, as it may induce oedema in a sudden and dangerous manner.

3. *Diuresis.*—It is certainly bad policy to stimulate an inflamed organ, and one object of eliminating waste matter by the bowels and skin is to save the kidneys as far as possible, but where uraemia is threatening, or where dropsy is very excessive, it may be necessary to have recourse to diuretics. In ordinary cases plain water or alkaline mineral waters form the best and simplest method of stimulating renal secretion, but the spirit of nitrous ether in 1 to 2 drachm doses may be administered occasionally, and often in association with the hot bath or hot-air bath.

The local treatment is very important, and especially if there is much lumbar pain and great haematuria. It consists in poulticing the loins and in very severe cases wet-cupping. A hot bath is an excellent method of treatment in children during the earlier stages of the disease as a means of relieving the congested kid[illegible]s.

There are c[illegible] in conditions which demand special treatment; sometime *dropsical effusions* require to be tapped, and Southey's tubes or Potain's aspirator may have to be used, the former for ascitic effusions and for great oedema of the legs, the latter for hydrothorax. Care must be taken to prevent irritation of the skin, in view of the peculiar tendency of patients

suffering from Bright's disease to succumb to the attack of the specific organism or organisms of erysipelas. A salt-free dietary has the definite effect in some cases of reducing the œdema. *Vomiting* should be treated with dilute hydrocyanic acid and bismuth, and sometimes with minute doses of carbolic acid or iodine, while the *dyspepsia* so commonly associated with the disease may require the use of pepsin and dilute hydrochloric acid. The *anæmia*, from which all Bright's disease patients suffer to a greater or less extent, should be treated with iron and sometimes with arsenic. Where albumin persists in the urine, various drugs have been recommended, although none of them seem to be of special efficacy. These are nitroglycerine in $\frac{1}{100}$th of a grain doses, tincture of the perchloride of iron, which is supposed to act as a powerful astringent, and rosaniline, which has the effect of colouring the urine in a marvellous fashion, although it does not appear to produce any phenomenal benefit. A warm, dry climate is of great advantage in the after-treatment of acute Bright's disease, while special care should be taken of the patient, to guard against chills and against any strain being put on the kidneys by imprudent diet. Should dilatation of the heart occur, strychnine and arsenic must be carefully administered with prolonged rest in bed. The cardio-vascular changes associated with Bright's disease are referred to under cirrhosis of the kidney (p. 553).

IX. SUBACUTE PARENCHYMATOUS NEPHRITIS

Two varieties of Bright's disease may be included under this head: (1) the form which is the so-called "large white kidney," or more correctly the large type of subacute parenchymatous nephritis; and (2) a subacute variety of Bright's disease, in which the kidney is smaller in size, and which is either secondary to acute Bright's or commences as a less acute variety.

Type I.

Etiology.—It is generally of insidious development, and is sometimes due to excessive alcoholism, and sometimes to the three great causes of waxy degeneration, *i.e.* syphilis, chronic

suppuration, and suppuration of bone. This form of Bright's disease may also be present in cases of tuberculosis, and possibly it may develop, in place of acute nephritis, after exposure to cold or after scarlet fever.

Pathological Anatomy.—The kidney is large the capsule is not markedly adherent, probably in most cases stripping freely, the cortex is pale and swollen, and the pyramids may be deeply congested. Microscopically the tubules are distended the epithelium is very fatty, the glomeruli are enlarged and their capsules thickened. The walls of the capillaries show evidence of hyaline degeneration, and the interstitial tissue is everywhere increased in amount. Where it is associated with amyloid degeneration, there are in addition marked waxy changes.

Clinical Features.—The urine is at first small in amount and very highly albuminous, although later polyuria may develop, especially in waxy cases; the urea is diminished. there is much degenerated renal epithelium, and many leucocytes, and hyaline, granular, epithelial, and fatty casts are present in the deposit. There is dropsy of the face and body generally, and the anaemia is profound, the face in particular being pale and pasty-looking. Gastro-intestinal symptoms are common, especially vomiting and diarrhoea, while ulceration of the colon may hasten a fatal result.

The cardio-vascular changes, while they may be present, do not develop so definitely as in chronic Bright's disease. Uraemia is of frequent occurrence and may cause death.

The **Diagnosis** between this form of Bright's disease and the second variety, to be mentioned immediately, is by no means easy. The insidious commencement, the frequent association with waxy disease, and the large number of fatty casts, suggest the probability of the large type of subacute kidney disease being present.

Prognosis.—Whether it is associated with waxy degeneration or not, the prognosis is always grave. Oedema is common. Inflammation of serous membranes frequently occurs, and uraemia is a constant danger. The coexistence of waxy degeneration adds much to the gravity of the case.

The **Treatment** must be on general lines, and little can be done in the way of attempting a cure.

Type II.

In this variety of Bright's disease, which is much more common than Type I., the kidney is of smaller size, the cortex shows marked fatty changes, and, as a rule, fatty casts in the urine are numerous. The capsule is thickened and somewhat adherent. The amount of albumin is invariably greater than would be expected in a case of more chronic Bright's disease, but it is so closely associated with chronic Bright's disease, of which it is really a more acute variety, that its description will be included under that heading. Where a patient, who has suffered from acute Bright's disease, does not recover, there are apt to be frequent acute or subacute exacerbations, and after death marked fatty changes in the kidney are usually seen.

X. CHRONIC BRIGHT'S DISEASE

(Chronic Inflammatory Nephritis; Chronic Interstitial Nephritis)

This form of Bright's disease may result from an acute case, or it may be chronic from the outset, although it is extremely unlikely that the type referred to as the large type of subacute parenchymatous kidney can ever become the "small white kidney" commonly met with in chronic interstitial nephritis.

Etiology.—The causes of acute Bright's disease may give rise to this variety, but probably toxic agents such as alcohol, lead, and gout play a prominent part in the cases which are chronic from the outset.

Pathological Anatomy.—This variety of kidney disease is frequently termed the "small white kidney," by which is meant that there are marked fatty changes in the cortex, and that the cortex is, as a rule, atrophied; but the accompanying with the development of interstitial tissue, and the kidney may therefore be, comparatively speaking, large, if it is in the stage of an acute or subacute affection, and may closely resemble the subacute parenchymatous kidney in appearance.[1] In many cases, however, the kidney is found to be small in size, closely

[1] These cases are practically the same as, and certainly indistinguishable from, Type II. of subacute parenchymatous nephritis.

resembling the small atrophied kidney of a primary cirrhosis from which it may with difficulty be differentiated by the naked eye, although as the result of the marked cicatricial changes secondary to interstitial exudation the interlobular vessels become extremely tortuous, whereas in primary cirrhosis the vascular markings are usually straight. Further, in a post inflammatory kidney, however advanced the cirrhotic change may be, there is always greater evidence of fatty degeneration in the tubular epithelium.

The vessels are thickened, the thickening being greater in the more cirrhotic forms; and the walls of the capillaries and Bowman's capsules show marked hyaline degeneration. The tubules of the cortex are irregularly dilated. Much of the epithelium has been shed, and there is evidence of fatty degeneration, as already stated.

The capsule, in the cases where there is little interstitial change, strips more freely, although there are always some adhesions, but in the cirrhotic cases it is very adherent, and leaves a granular surface when it has been removed, which closely resembles what is seen in primary cirrhosis.

Clinical Features.—In cases previously acute, the history of the symptoms will correspond to what has already been stated under acute Bright's disease. In less acute cases, or in chronic cases, there may be an insidious development with gradual loss of strength, progressive emaciation, and profound anæmia. The degree of dropsy varies greatly; in certain cases it may be very obvious, the face showing the typical puffy pasty-looking appearance of an acute Bright's case, while in the more cirrhotic forms dropsy may be entirely absent. Similarly, in the urine, where the kidney is of larger size, the quantity of urine will probably be scanty and the amount of albumin large; on the other hand, in the "small white kidney" the urine tends to be excessive and the albumin less copious. The urea is generally diminished, and the casts vary both in character and number. There are always granular and hyaline casts, but fatty casts are specially frequent in the larger variety of kidney.

Alimentary System.—The gastro-intestinal phenomena are usually well defined, a degree of nausea and vomiting being almost constant, and the vomiting is generally of the type which occurs in the morning before breakfast. Hiccough

is sometimes present and diar[illegible] is not infrequent, and it should be remembered that enteritis sometimes ensues as a complication, and consequently diarrhœa adds much to the anxiety of those in charge of a case of chronic Bright's disease.

Haemopoietic System.—Anæmia is the only prominent feature to be noted in this system.

Circulatory System.—Cardio-vascular changes are extremely marked, and especially in the small type of kidney. There is generally progressive, and often great, enlargement of the left ventricle, while the arterial walls are thickened and the pulse is of high tension. Partly as a result of these vascular changes, and partly on account of the presence of toxic substances in the blood, there is great tendency to haemorrhage. Such haemorrhages may occur from the gums, from the nose, from the kidney, and, not infrequently, as purpuric haemorrhages, especially seen on the legs.

Respiratory System.—Dyspnœa is present if much pulmonary œdema has occurred, and asthma, in reality a uræmic manifestation, may also be noted.

Integumentary System.—The dropsy and purpuric haemorrhages have already been referred to, and there is a peculiar liability to erysipelas where there is any irritation of the skin. Pigmentations are by no means uncommon, and eczema and sometimes erythema may be seen in certain cases.

Nervous System.—In referring to the nervous phenomena in acute Bright's disease it was stated that many of them had already been mentioned under other systems, although they were in reality of uræmic nature, and the same statement holds good here. The vomiting, nausea, and dyspnœa are all to a large extent of uræmic or, in other words, of cerebral origin. Headache is a prominent feature and is dependent on the changes in the vessels, and perhaps partly on the high blood-pressure in the cerebral arteries, and it is noteworthy that after a severe attack of epistaxis or renal haemorrhage, the headache is greatly relieved. Uræmic cramps and twitchings of muscles, Cheyne-Stokes breathing, and all the typical phenomena of uræmia referred to under that heading, are unfortunately only too common in chronic Bright's disease, and the slightest over-strain or over-exhaustion may precipitate a fatal attack: while the secondary inflammations of serous membranes which form such common complications in every variety of Bright's disease

may readily induce uraemia. Albuminuric retinitis is specially apt to develop in all forms of chronic Bright's disease. It is seen as stellate patches of fatty degeneration round the macula lutea, and often there is marked papillitis with numerous flame-shaped haemorrhages. As a result sight generally fails, and this may be the means of bringing the patient for the first time to seek medical advice. Cerebral haemorrhage, while not so frequent as in primary cirrhosis of the kidney, is also apt to occur, and is often associated with the presence of small miliary aneurisms on the cerebral arteries.

Complications.—The only complications to which special reference is necessary are inflammations of serous membranes, and in particular of the peritoneum, pleura, and the pericardium, while mention may again be made of enteritis, a virulent type of which may terminate the patient's life.

Diagnosis.—There is little probability of a mistake where dropsy and a large amount of albumin are evident clinical features. Uraemic manifestations should not be forgotten, and a patient, supposed to be suffering from asthma, may be found to be really the subject of chronic Bright's disease. On examining the urine it is very unlikely that a *physiological albuminuria* could be mistaken for this disease, because the kind of casts usually found present and the amount of albumin are distinctive. In physiological albuminuria there is little albumin in most cases, and only a few granular or hyaline casts, and diet and exercise may be the factors which predispose to the presence of albumin at certain periodic times in the day. The greatest stress should be laid on the careful examination of the heart and vessels, because cardio-vascular changes inevitably follow in a well-defined case of chronic Bright's disease.

Prognosis.—The patient is invariably in a position of great danger, because the slightest overstrain may induce uraemia, and there is a considerable predisposition towards cerebral haemorrhage. On the other hand, patients carefully treated and living under the best possible surroundings may enjoy long life. Dropsy is probably the feature which, above all others, with the exception of uraemia, is of grave significance.

Treatment.—Each case requires careful study. Find out the amount of the albumin, the number and variety of the casts, and the capacity of the kidney for carrying on the work

of life. There are cases in which it is futile to attempt vigorous treatment with any hope of cure, and excessive bathing may only lead to serious exhaustion of strength.

A dietary as simple as possible should be rigidly adhered to, but it is unwise to limit the patient to milk alone. Specially forbid an excess of meat and all heavy and indigestible food, and particular stress should be laid upon the importance of abstinence from alcohol, or its use in very limited quantity. Anaemia should be treated by giving large doses of iron: plenty of fresh air, but without undue exercise or fatigue, is most important. Where dropsy is present, purgatives and diaphoretics are requisite, but, while care should be taken to see that the bowels act freely, it is unwise to over-stimulate the alimentary tract.

In cases of Bright's disease it is undesirable to use drugs which, owing to renal inadequacy, can accumulate in the system, and therefore opium should only be administered under exceptional circumstances. Mercurial salts should also be prescribed with caution, because in Bright's disease there is a special tendency to mercurialism.

Stripping off the capsule of the kidney has been recommended in cases of chronic Bright's disease, with the view of relieving the vascular strain by inducing new anastomoses. In some cases the results have been favourable, but statistics do not enable us to express a definite opinion as to this procedure.

XI. PRIMARY CIRRHOSIS OF THE KIDNEY

(Granular Contracted Kidney: Small Red Kidney)

This form of cirrhosis of the kidney is in reality a part of arterio-sclerosis, inasmuch as there is invariably a definite change in the arteries generally, similar to what is found in the vessels in the kidney, and associated with this there is hypertrophy of the left ventricle.

Etiology.—There is sometimes a hereditary tendency to arterio-sclerosis, and the great causes of arterio-sclerosis are, and must be, causal factors in the production of cirrhosis of the kidney. These include syphilis, alcoholism, muscular over-strain, overeating especially of an over-rich diet, gout, and to a

less degree, mental overwork. Males suffer more than females, and it is a disease which rarely commences before adult life, while many of its victims suffer at, or after, the age of forty.

A mild form of what is, in reality, arterio-sclerosis, with cirrhosis of the kidney, may be found after death in those who have put too great a strain upon their vessels and kidneys from all sorts of causes, and it is frequently seen in persons who can hardly be termed alcoholic, although they have indulged in an amount of alcohol which has proved excessive for the welfare of their arteries and kidneys. It is an interesting fact that as a result of mental strain arterio-sclerosis is specially prevalent amongst asylum inmates.

A form of cirrhosis may also be the result of hydronephrosis, or of any condition where there is similar interference with the renal functions.

Pathological Anatomy.—The kidney is small in size, generally weighing only $1\frac{1}{2}$ to 2 oz.; it is often embedded in fat, sometimes to a remarkable extent, and when this is stripped off, the surface of the kidney presents the granular appearance from which it has derived its name. These granular swellings correspond to the lobules: the capsule is thickened and extremely adherent; the colour is deep red, the cortex, however, being somewhat mottled, if fatty changes are present. The cortex is greatly diminished in thickness, and there are numerous cysts, some of them subcapsular, and others scattered throughout the cortex. They are due to dilated and obstructed tubules: most of them are microscopic in size, but the larger ones are found to contain a yellowish fluid in which urea and other urinary constituents may be present.

The vessels show an extreme degree of thickening, often resembling endarteritis obliterans, and the vascular markings, although not so irregular as in the "small white kidney," are rarely perfectly straight. Bowman's capsules are greatly thickened and hyaline, and, as a result, the glomerular tufts are often completely atrophied. The interstitial changes are most advanced in the neighbourhood of the interlobular arteries, and therefore the glomeruli farthest away from these vessels are generally less affected than those in their immediate neighbourhood. The tubules have undergone remarkable changes: in parts they are greatly dilated, while the epithelium is often

granular and fatty; in some tubules the epithelium is extensively desquamated, and in others it has disappeared altogether. The changes in the heart and arteries have been more fully discussed under the heading of arterio-sclerosis (p. 414).

Clinical Features.—The disease begins insidiously, and may for a long time escape the attention of physician and patient. Amongst the earliest features are tiredness and headache, sleeplessness, dyspepsia, and the passage of an increasing amount of urine, which may necessitate the patient's rising several times during the night.

Urinary System.—The urine may reach 70 to 150 oz. per day; it is clear and of low specific gravity (1005-1010), with little or no albumin, and contains only a very few hyaline and possibly some granular casts. The urea is generally absolutely diminished. Albumoses are sometimes present in small amount, but their significance is uncertain. Blood is rarely noted unless an acute exacerbation supervenes. Uric acid crystals are sometimes precipitated, probably as a result of the very low pigmentary index and poorness of the urine in salts.

Circulatory System.—The clinical evidences of the arterio-sclerosis are definite; the larger arteries have thickened walls and are often atheromatous, but there is, in addition to actual thickening, a degree of hypertonus, by which is meant vaso-motor spasm, certainly the result of toxins circulating in the blood-stream. The intravascular pressure may be as high as 200 to well over 250,[1] and the left ventricle is found to be enlarged, the apex-beat displaced outwards and downwards, and on auscultation the first sound in the mitral area is booming in character, and the second sound in the aortic area markedly accentuated. Where enlargement of the left ventricle renders the mitral orifice incompetent, a mitral systolic murmur is heard, and in process of time the evidences of backward pressure appear both in the lungs and the right heart.

These cardio-vascular changes, with the exception of the mitral incompetence, may develop to a quite recognisable extent within six weeks.

Alimentary System.—The appetite is poor, and the long-continued dyspepsia, often associated with morning sickness,

[1] The normal sphygmomanometer reading is 120-130 millimetres of mercury; any increase above 140 implies abnormal intravascular pressure, but there is a rise of blood-pressure as age advances.

soon tells upon the patient's strength; diarrhœa is sometimes present.

Haemopoietic System.—Anæmia may gradually develop, and a chemical investigation of the blood shows the presence of excess of urea in cases in which uræmia is impending.

Respiratory System.—Epistaxis is one of the clinical features worthy of special reference; it may be both troublesome and excessive. Bronchitis is often present, sometimes of the asthmatic type, and if so it is very suggestive of uræmia.

Integumentary System.—The purpuric haemorrhages, already referred to, are not uncommonly seen under the skin of the legs, and where, with such haemorrhages, there is no rheumatic history, the possibility of primary cirrhosis of the kidney should not be forgotten. Pigmentations are also not infrequent in this variety of kidney affection. Dropsy is rare, except in the later stages, or in cases in which an attack of acute nephritis has been superadded.

Nervous System.—The arterio-sclerosis causes very definite cerebral phenomena. Headache, sleeplessness, and difficulty in cerebration are common, and it should be remembered that as many cases of this disease terminate fatally by the development of uræmia, all the uræmic phenomena may be present.

Albuminuric retinitis occurs in a considerable number of cases, sometimes with papillitis and flame-shaped haemorrhages. Very many cases of apoplexy occur in persons with primary cirrhosis of the kidney, and the prognosis in these cases is invariably more grave owing to the arterial and renal disease.

Diagnosis.—There is no disease so difficult to diagnose as early cirrhosis of the kidney, but in the advanced stages the cardio-vascular phenomena and the polyuria render recognition easy. It may, however, be impossible to determine whether the condition has been purely a primary cirrhosis, and not to some extent a post-inflammatory affection, and, perhaps, the study of the casts in the urine forms the best means of coming to a decision; dropsy is far more frequent in cases which are, or were, inflammatory.

In any case in which a patient comes to the physician complaining of overstrain, langnor, and dyspepsia, without any sufficient cause, the possibility of primary cirrhosis should not be overlooked.

Prognosis.—Granular kidney is absolutely incurable, but

a long life may be enjoyed by the patient provided the greatest care is taken to guard against strain, the effects of alcoholism, and imprudence as regards exposure to cold and wet.

Treatment.—A quiet life, with little mental and physical effort, must be enjoined upon all patients. Alcohol should be forbidden, and the diet reduced to one which will put little strain on the affected kidneys and vessels. A fairly warm climate and warm clothing are advisable.

Medically an attempt may be made to lower the blood-pressure where it is excessively high, and probably the nitro-glycerine group is the most valuable, because with the relief obtained the headache disappears, and frequently the patient's general well-being much improves. The liquor trinitrini in 1 to 3 minim doses, or $\frac{1}{100}$th to $\frac{1}{50}$th grain of nitroglycerine, should be given. The benefit obtained from potassium iodide in arterio-sclerosis should not be forgotten, and aconite administered in the form of the tincture in 2 to 5 minim doses often helps to lower the vascular tension. Where the heart is dilated and marked mitral incompetence has developed, digitalis and strophanthus should be ordered, while sometimes venesection or other measures for the relief of backward pressure, and especially the removal of dropsical effusions if they develop, may save the patient at a critical moment.

Purgatives, diaphoretics, and the cautious use of baths are advantageous in the general treatment of the case.

Anaemia should be treated by the free use of iron.

XII. WAXY DEGENERATION OF THE KIDNEY

Waxy degeneration, especially involving certain vessels in the kidney, is a part of a general condition, and other organs, such as the liver, and often the spleen and alimentary tract, may suffer equally.

Etiology.—Syphilis, long-continued suppuration, and bone disease are common causes of amyloid degeneration, although there are rare cases associated with a varying degree of cachexia which appear to be inexplicable.

Pathological Anatomy.—The vessels suffer first, and particularly the afferent and efferent arterioles, and also the glomerular tufts. At a later period the straight arteries and

larger vessels of the kidney may also be affected. The change is specially marked in the middle coat, and is chiefly limited to the fibrous tissue. At a later period the basement membrane of Bowman's capsules and the tubules become involved, and it is stated that the tubular epithelium may eventually suffer. There are very often fatty changes associated with the waxy degeneration, and especially is this the case where, in a subacute parenchymatous nephritis, waxy degeneration is superadded, not infrequently of syphilitic origin.

A waxy kidney is large in size, weighing 10 oz. to 1 lb., and on section has a glistening appearance; if iodine is poured over the cut section the glomeruli are picked out as mahogany-brown spots. In microscopic sections iodine may be used, or methyl-aniline violet, and in either case the waxy material acquires a distinctive stain, mahogany-brown as the result of the iodine, and rose-pink with methyl-aniline violet. It is rare to find waxy degeneration alone, and generally a degree of nephritis, often tubular, sometimes glomerular, is present in addition.

Clinical Features.—The urine is large in amount (70-100 oz.), is pale in colour, of low specific gravity (1006-1015), and sometimes contains a small amount of albumin; where there is fatty change as well, there may be much albumin. The tube casts are hyaline, and may be described as being of a "hard" hyaline nature, although it is doubtful whether they ever show the waxy reaction on staining. There are sometimes fatty and often granular casts present. The amount of urea in an uncomplicated case may be practically normal.

The dropsy varies greatly: it may be pronounced when profound anaemia and cachexia are present, but it is by no means invariably a prominent symptom. Cardio-vascular changes, retinitis, and uraemia are rarely present, and where they occur they result from changes in the kidney other than the waxy degeneration.

A careful investigation should be made so as to determine, *firstly*, the cause of the waxy degeneration: and, *secondly*, the presence of waxy changes in other organs with the respective clinical features.

Diagnosis.—It is obviously impossible by any examination of the urine alone to recognise positively the presence of waxy degeneration of the kidney, but it may be suspected.

where, along with a definite causal disease, the symptoms above mentioned are present.

The **Prognosis** is always unfavourable, and especially when a marked degree of dropsy appears. It naturally depends also to some extent upon the disease to which the condition is due.

The **Treatment** must be upon general lines; it is unwise to give much fatty material, either in the diet or as medicine, and therefore cod-liver oil is contra-indicated where waxy degeneration is present. Potassium iodide, iron, and careful dieting are probably the best methods of attempting to treat a very hopeless condition.

XIII. HYDRONEPHROSIS

Distension of the pelvis and calyces of the kidney with urine, generally the result of obstruction in some part of the urinary passages.

Etiology.—It is commoner in females. It may be congenital from obstruction of the ureter by folds, imperforate urethra, etc. It may occur in later life from impacted calculus, stricture of the ureter, abdominal and pelvic tumours, growths in the bladder, enlarged prostate, and urethral stricture. In a mild degree it is often associated with floating kidney.

Pathological Anatomy.—The pelvis enlarges at the expense of the kidney: the papillae of the kidney undergo atrophy, and there may be little kidney substance left. The ureter may participate in the dilatation.

The fluid consists of dilute urine, often with a little albumin: pus is present if there is any inflammation of the pelvis. Where one kidney is alone affected, the other tends to undergo compensatory hypertrophy.

Clinical Features.—Mild cases often escape recognition. If unilateral and more marked, it may only cause a cystic tumour to appear in the region of one kidney. Not infrequently this tumour disappears with the passage of the contents into the bladder; but generally reappearance soon takes place (the intermittent type).

If both kidneys are involved, in addition to suppression of urine, there is an early development of uraemic symptoms.

Rarely does the condition become purulent and form a pyonephrosis.

Diagnosis.—*Ovarian tumours* are generally recognised on vaginal examination, while the hydronephrotic cyst lies in the region of the kidney and is covered anteriorly by the ascending or descending colon. General or loculated *effusions into the peritoneum* may be excluded by examining the fluid after aspiration.

Prognosis.—A unilateral lesion causes little trouble except from the pressure exerted by the tumour, and the condition may even disappear in time without treatment. If the lesion is bilateral and complete, death from uraemia must inevitably ensue.

Treatment.—Do not operate on intermittent cases. A pad and bandage have been recommended and may be tried. Aspiration, keeping well behind the colon, is occasionally indicated, while sometimes draining by free incision or even removal of the kidney may be necessary.

XIV. TUBERCULAR DISEASE OF KIDNEY

Tubercular disease may be primary or secondary.

I. *Primary tuberculosis of the kidney* may involve the pelvis of kidney, the ureter, and the bladder, but rarely the prostate, and it is often a matter of difficulty to suggest its starting-point. It generally attacks one kidney first, and if both are involved, one is more extensively affected.

Etiology.—No definite cause for the site of the disease can be assigned; children appear to be less commonly the subjects of this form of tuberculosis.

Pathological Anatomy.—Tubercle nodules are deposited in the substance of the kidney, which apparently start in or near the pelvis or calyces; these coalesce after caseating, and thus form ragged abscess cavities which open into the pelvis. Lime salts may eventually be deposited in the caseous areas. The ureter is generally thickened, and may be almost occluded with tubercular deposit, and the mucosa may be ulcerated.

Sometimes the bladder, the prostate, the testicles, and the vesiculae seminales are involved.

Clinical Features.—If only one kidney is affected there

may be no symptoms, but usually the case simulates a pyelitis in the early stages, because of the severe lumbar pain, and there may be evidence of a renal tumour.

The urine, which is frequently acid, contains pus, and generally tubercle bacilli. Blood is rarely copious, the albumin depends on the pus present, and the amount of urea varies with the capabilities of the unaffected kidney. Later cystitis, with alkaline urine, may develop. Hectic fever with emaciation is present in more rapid cases.

Many instances of extensive renal tuberculosis, exactly similar to primary renal tuberculosis, are really secondary, but they differ in no way from the description given above.

The **Diagnosis** depends mainly on the detection of tubercle bacilli, and evidence of coincident tuberculosis in some other part of the genito-urinary tract, but cystoscopy and testing of the urine from each kidney by the use of Luys' separator or by catheterising the ureters may yield invaluable information.

Prognosis.—The condition is compatible with many months or even years of life, but is eventually fatal. Where active tuberculous pyelo-nephritis is present, the immediate outlook is grave.

Treatment.—The possibility of surgical interference should be determined. If the other kidney is healthy, nephrectomy is indicated. Tubercular cystitis contra-indicates operation. Treat otherwise on general lines, cod-liver oil and tonics being useful, while urotropine (gr. 5-10) might be tried as a urinary antiseptic. One of the tuberculins may be used, but only after due consideration, and a marked reaction should be obviated by giving small doses.

II. *Secondary Tuberculosis of Kidney.*—In acute general tuberculosis small tubercle nodules are often found dotted over the kidneys. Sometimes a slight albuminuria is present.

XV. RENAL CALCULUS

(Nephrolithiasis)

The following calculi are common :—

(1) Uric acid. Hard, round, smooth or finely granular, of reddish colour, generally numerous, poppy-seed to pea in size.

(2) Oxalate of lime (mulberry). Hard, very rough, dark colour, often single.

(3) Sodium urate. Soft, of moderate size.

(4) Mixed calcium and triple phosphates. Phosphates in alkaline urine deposited on a previous calculus (oxalate or uric acid), may be of huge size.

(5) Calcium phosphate. White, smooth, chalk-like, in size from pea to hen's egg.

While calculi are often found in the bladder, they may also occur in the kidney, and especially in the pelvis of the kidney and in its calyces. The most common calculi are mainly uric acid or oxalate of lime, but very frequently carbonate of lime, phosphates, and other salts may be present, forming some of the layers which go to make up the laminated structure of a stone. In time the calculus may form a coral-like stone filling the pelvis, and with branches extending into the calyces. Cystine and other rare constituents need not be mentioned here. Calculi consisting mostly of phosphates generally occur in the bladder, and not in connection with the kidney. The deposition of salts may be the result of some chemical change in the urine, or it may be associated with disease of pelvis or kidney. Most stones have a definite nucleus, the nuclei varying in nature, being sometimes uric acid or oxalate of lime, and sometimes a small blood-clot, or other foreign body. In many cases little uric acid calculi are passed in great numbers, without the definite clinical features which are associated with a large stone, and to this condition the term gravel or sand has been applied.

Etiology.—Calculi occur at all ages; they may be met with in young children, although they are more common after middle life. A good deal depends on the reaction of the urine whether the calculus is on the one hand a uric acid or oxalate of lime calculus, or, on the other, a phosphatic calculus; but it should be remembered that a calculus is always made up of layers superimposed the one on the other, and in these layers it is usual to find evidence of the urine having been from time to time sufficiently alkaline to precipitate carbonate of lime or phosphates.

In children and young persons the uric acid calculus is not uncommon. A urine of high acidity, with a small proportion of salts and a low colour index, tends to precipitate the

uric acid normally held in solution. While it is true that concentrated urine may contain an excess of uric acid, it often also contains a sufficient amount of saline constituents to prevent the uric acid from depositing.

In the case of oxalate of lime calculi a chemical error in digestion is probably an important factor, and excess of oxalate of lime will inevitably result in its precipitation from the urine. Green vegetables and fruits of all kinds contain a large quantity of oxalic acid, but certain patients appear to have a sufficient digestive error to explain the oxalate of lime apart from anything specially ingested.

Much stress has been laid upon the association of calculus with a pyelitis, trivial or severe, and it is held to be extremely probable that an excessive amount of mucus may in itself tend to produce the precipitation of certain crystalline bodies from the urine. A sedentary life is a strong predisposing factor in the production of calculus, and assuredly an unsuitable diet, and especially one apt to increase the amount of uric acid in the urine, plays an important part in those cases in which there is at best a tendency to an excessive production of uric acid. It is also true that a diet poor in salts may lead to the precipitation of uric acid. Calculus is much more common in men than in women, the proportion being as 5 to 1, and not infrequently, certainly in one-fifth of all cases, there is evidence of both kidneys being involved.

Pathological Anatomy.—A good deal depends on where the stone or stones are situated. If it is in the pelvis of the kidney, and if, as not infrequently happens, the calculus branches into the calyces, there is a degree of cirrhosis of the kidney induced, and very generally a hydronephrotic distension of the calyces and sometimes the pelvis. In many cases pyelitis is set up, and may even cause the destruction of the whole kidney.

In other cases a calculus may be present for years, and lead to no definite clinical feature which suggests its presence, or at most a slight pyelitis may be the only pathological result of the stone.

Clinical Features.—Pyelitis, and certainly pus formation in connection with the stone, lead to definite clinical features; but there are many instances of extensive branching calculi which have caused absolute atrophy of the affected kidney, which are only discovered accidentally after death, because

the remaining kidney may be adequate for double duty: small calculi frequently produce no symptoms.

An attack of *Renal Colic* indicates the attempt by the calculus to pass down the ureter. The pain is very typical: it commences in the loin and shoots downwards towards the scrotum, sometimes extending for a considerable distance down the inner side of the thigh. The pain is frightfully severe; it is accompanied by a feeling of sickness, often with vomiting, and the abdominal wall on the affected side is rigid and tender to pressure. The pain frequently occurs in paroxysms, although it may also be constant. The attack lasts for a varying time: it may cease when the stone has reached the bladder, or when it returns to the pelvis of the kidney from which it has been displaced, while in a few cases the calculus becomes encysted and the pain gradually wears itself out. In other cases the pain is located in the loin, and does not appear to shoot downwards towards the leg, and this is more likely to occur where a calculus in the pelvis of the kidney has become displaced but has not endeavoured to enter the ureter. In both these types of renal colic the temperature is frequently high, 102° or 103° F., while rigors are common, and they may become more marked if pyelitis or suppuration is in progress. Haematuria is an almost constant accompaniment of attacks of renal colic, although the amount of blood will depend much on the character of the stone and the irritation which it may set up. For the history and clinical features of stone in the bladder the reader is referred to surgical text-books.

If the stone becomes encysted, the passage of urine from the affected kidney may be arrested and hydronephrosis consequently results. Uraemia does not, however, occur unless both kidneys are seriously interfered with.

Diagnosis.—*Biliary* and *intestinal colic* must be differentiated from the colic due to a travelling renal calculus. In biliary colic the pain is associated with the region of the gall-bladder, generally with enlargement and local tenderness of that organ, and is very often followed by jaundice, while biliary calculi may appear in the stools after an attack is over. In renal colic the pain shoots, in most cases, towards the inner aspect of the thigh, and there is no special tenderness in connection with the gall-bladder. With enteralgia the pain is frequently better marked in the region of the umbilicus; there

is no local tenderness over either kidney, and the history of the attack, commencing with the ingestion of some intestinal irritant, is often definite. Neither in biliary colic nor enteralgia does blood appear in the urine.

In *lumbago* and *neuralgia* the pain may be paroxysmal, but is rarely so severe, and in place of shooting down the thigh would more probably be limited to the affected muscle, or would follow the line of the spinal nerve. There is also in both these affections definite evidence of superficial tenderness on pressure, while blood in the urine is not present.

Blood-clots, associated sometimes with *cancer* of the kidney, may also cause attacks of renal colic. There is commonly in malignant disease much more haematuria than with calculus, and sometimes bits of the tumour may be recognisable in the urinary deposit.

In *floating kidney* renal pain is not infrequent, but it does not shoot in the typical direction so often associated with a calculus, and blood in the urine would be exceptional.

It is wise in cases of supposed renal calculus to examine the patient by means of the X-rays, and a photograph often gives more definite and satisfactory information than the use of the screen.

Prognosis.—The patient may only have one attack, but in all probability recurrent seizures imply the formation of other calculi, or the renewed attempt of the original calculus to pass down the ureter. Pyelitis is a common result of renal calculus, while pyonephrosis is by no means an improbable sequel. It is open to question whether the constant irritation of a calculus may not induce the development of malignant disease, and, in any case, there is apt to be a cirrhotic change in the kidney with more or less interference with the renal function, and sometimes kidney destruction. In some cases surgical interference yields most satisfactory and permanent results.

Treatment.—For an attack of renal colic endeavour to relieve the spasm by hot baths, warm-water enemata, hot fomentations applied over the loins, and hypodermics of morphia (gr. $\frac{1}{8}$-$\frac{1}{4}$) and atropine (gr. $\frac{1}{100}$-$\frac{1}{50}$), while in very serious cases chloroform anaesthesia should be employed. Sometimes changing the position of the patient in bed gives prompt relief, and it may help the calculus either to return

to the pelvis of the kidney, or to make its way more easily into the bladder; copious draughts of warm water, weak tea, or aerated alkaline water, assist in washing the calculus downwards.

Between the attacks, and after finding out by careful examination of the urine the probable nature of the stone or stones, an attempt may be made to counteract the tendency of the urine to precipitate its constituent salts. In this way alkaline mineral waters, such as Ems, Vichy, Carlsbad, and other spas, have been found to yield good results: while the treatment of the patient with bicarbonate of potash, in 30 to 40 grain doses, or other alkali, is frequently beneficial. Piperazine has been recommended as a solvent of uric acid; it is given in 15 grain doses, but probably has an extremely feeble effect in its very weak solution in the urine.

Abdominal massage has been commended, but should be practised with the greatest caution.

As regards surgical treatment, all that need be said here is that phosphatic calculi, provided the urine is free from pus, are often best left alone; that where the X-rays show that the opening into the ureter is being occluded, operation is necessary; and that in all cases pyuria should indicate the desirability of surgical interference. Prior to operation it is advisable to determine by examination with the ureteric catheter that the other kidney is functionally sound. Cryoscopy, of which much was hoped, has not proved of practical value. It consists in comparing the normal freezing-points of blood and urine with those of the patient's. The larger the quantity of salts and urea in the blood the lower will the freezing-point be, and their presence in excess indicates renal inadequacy.

XVI. PYELITIS—PYELO-NEPHRITIS—PYONEPHROSIS

Inflammation of the pelvis of the kidney and the conditions which result from it.

Pyelo-nephritis implies that the kidney itself has also become involved, and pyonephrosis that the kidney has broken down and forms an abscess.

Etiology.—Pyelitis may be due to the following:—Calculi; decomposing urine (retained by pressure on ureter) in pelvis, or a sequel to cystitis; tubercle, cancer, and parasites. It may occur in some of the infectious fevers, such as scarlet, typhoid, small-pox, diphtheria, etc.; as the result of certain irritating drugs, such as cantharides and turpentine; and possibly from cold.

From this list of causes it will be seen that pyelitis may (1) begin in the pelvis of the kidney, as in the case of tubercle, in the infective fevers where the infective organisms are excreted by the kidney and so enabled to set up a local inflammation, in any severe illness where it is thought organisms may make their way to the pelvis possibly from the intestine, and rarely as the result of the excretion of such substances as turpentine, copaiba, and sugar. Pyelitis may (2) be associated with, and be a sequel to, acute nephritis; or it may (3) result from infection from below as in cystitis.

Pathological Anatomy.—There is catarrh sometimes with haemorrhages, ulceration, and occasionally extensive necrotic areas. In more chronic cases the pelvis and calyces may be distended and the walls greatly thickened. The distension is often sufficient to cause destruction of the kidney substance. Sometimes the kidney suppurates, causing pyonephrosis.

In "surgical kidneys" where secondary to cystitis a suppurative nephritis ensues, there is evidence of suppuration extending into the pyramids, and often small wedge-shaped abscesses under the capsule in the cortex develop

The **Clinical Features** depend on the stage and severity of the condition. Dull, aching pains in the loins, increased on palpation, with mucus and pus in the urine (which may be acid), are common, while the amount of albumin varies. Tailed cells, at one time considered to be diagnostic of pyelitis, cannot be depended on, as they may come from other parts of the urinary tract, and particularly the bladder. In an acute case rigors and hectic phenomena may occur. In chronic cases there is frequently much pus, which is sometimes intermittent from temporary blocking of the affected ureter, in which case a renal tumour is palpable prior to the discharge of pus. Suppurative nephritis or cystitis may add characteristic features to those already mentioned.

There is always a risk of uraemia developing.

Diagnosis.—The examination of the urine obtained from each kidney by the urinary separator is an invaluable help, and careful abdominal palpation should be carried out. The cystoscope may also yield important evidence. Tubercle bacilli should be repeatedly sought for, and one of the tuberculin tests should be employed.

Prognosis.—Mild cases and those occurring in fevers are favourable. Tubercular cases may become quiescent. Involvement of the kidney, whether it be a pyelo-nephritis or a pyonephrosis, increases the gravity of the case. Prolonged suppuration may cause waxy changes to develop and speedily kill the patient.

Treatment.—Complete rest in bed, and hot baths, poultices, and sometimes wet-cupping the loins, are advisable in acute cases. Allow plenty of diluent drinks.

Where there is much pus—alum, mineral acids, tincture of the perchloride of iron, and acetate of lead (gr. 3, thrice daily) may be tried, or, with caution, such remedies as oil of turpentine or sandal-wood oil. Urotropine (gr. 10) is frequently useful, and quinine may be given with benefit. Above all, support the patient's strength by milk diet in acute cases, by more generous diet in tubercular cases, and in the latter send the patient, if the disease is sufficiently quiescent, to the sea-side. Tuberculin treatment has yielded hopeful results in some instances. In certain unilateral cases of acute pyelo-nephritis and pyonephrosis, nephrectomy or nephrotomy is necessary, but only after carefully testing the adequacy of the unaffected kidney.

XVII. PERINEPHRIC ABSCESS

Suppuration around the kidney.

Etiology.—It may be due to (1) a blow or crush; (2) the spread of inflammation from kidney or pelvis outwards; (3) the spread of inflammation from a source outside the kidney, as from perforation of intestine, appendicitic abscess, empyema perforating diaphragm, or caries of spine; and (4) certain continued fevers in children.

Pathological Anatomy.—The pus may be in different

positions. It may have a faecal odour. It may burrow in different directions, sometimes simulating a psoas abscess, and it may set up peritonitis.

Clinical Features.—A tumour is felt, and pain is usually present, dull and aching, or sharp and increased on pressure. The leg of the affected side is often kept flexed, adduction is difficult, and the testicle is retracted.

Diagnosis.—Palpation may reveal a definite tumour, and almost always causes local pain. *Hip-Joint disease* may have to be excluded. An aspirating needle yields definite information.

The **Prognosis** varies with the nature of each case.

The **Treatment** is surgical, and the abscess should be opened and drained; rarely does the abscess discharge itself spontaneously into the bowel.

XVIII. CYSTS OF THE KIDNEY—CYSTIC DISEASE OF THE KIDNEY

1. **Simple Cysts.**—These vary in size from a pea to 3 or 4 inches in diameter, being occasionally even larger. They occur in the cortex, may project from the surface, and contain a clear, watery, or gelatinous fluid, in which there is a trace of albumin and salts, and sometimes urea or uric acid. These cysts are found in kidneys otherwise perfectly healthy, and are by no means of uncommon occurrence. They are probably formed in uriniferous tubules, and are due to the occlusion of these tubules at certain points. They are very rarely palpable, and are generally discovered after death. It has been suggested that possibly injury might explain the formation of some of these cysts, but there is little evidence in favour of this view.

2. **Cysts** occur also in *granular contracted kidney*, and are specially common in primary cirrhosis, although they may be present in any of the chronic forms of Bright's disease. They are of small size; sometimes they contain fluid, dark in colour, from the presence of blood pigment, and they cause no symptoms.

3. **Dermoid cysts** may occur in the kidney, although they are very rare.

4. **Hydatid cysts** are also found, to which reference is

made under the head of parasites of the kidney (p. 570), and also parasites generally (p. 199).

5. **Cystic Disease of the Kidney.**—This condition may occur in adults, or it may be congenital, although it is quite conceivable that the cases only recognised in adult life have their origin in foetal life. A hereditary predisposition has been occasionally traced. The whole organ is composed of cysts varying in size, containing a clear or opaque, sometimes viscid fluid, which may be reddish in colour, occasionally blackish. Albumin, blood, leucocytes, and cholesterin have been found in the fluid. All distinction between the cortex and the medulla of the kidney becomes lost, and it is difficult to detect any normal kidney substance in an advanced case. The kidneys are usually enlarged, sometimes very greatly, and the tumours formed by them may be readily palpable. The condition is bilateral in the form occurring in adults, and the enlargement in that form reaches a greater size than in the congenital variety.

Many theories have been suggested to explain cystic disease. Some authorities hold that the cysts are formed by obstruction of tubules, others that persistence of the Wolffian body, which has become mixed up with the kidney, may explain the condition, but in quite a number of cases the cystic disease is not limited to the kidneys only, the liver being sometimes affected, while cysts have been noted in the brain in patients who have well-marked cystic disease of the kidneys. It is possible, therefore, that cystic disease may be the result of a new growth in which cysts form, but this theory is open to question, and the cases in which cysts occur in other organs are sometimes classified separately from the cystic disease just described.

Clinical Features.—Many cases are only discovered after death, although death may be the result of uraemia with fairly typical symptoms. The clinical features, when present, are practically those of uraemic poisoning, and include nausea and vomiting, debility, ill-health, anaemia, and very generally the passage of a large quantity of pale urine of low specific gravity, and suggestive of cirrhotic kidneys. There may be a trace of albumin, but dropsy is certainly uncommon: other uraemic phenomena, such as dyspnoea, twitchings and cramps and coma, together with pyrexia, may develop towards the

termination of the disease. The *physical signs* would be suggestive were the abdomen palpated in suspicious cases, because in cystic disease in adults the kidneys are nearly always sufficiently large to permit of easy recognition. The cardio-vascular changes associated with cirrhosis of the kidney are not infrequently present in cystic disease.

Diagnosis.—As already noted, cystic disease is often missed entirely, but the presence of renal tumours, with the clinical features of cirrhosis of the kidneys, should make the diagnosis fairly clear.

The **Prognosis** depends on the adequacy of the kidneys for their vital functions, and also upon the amount of strain which the patient puts upon them. Any evidence of uraemia is of grave omen.

Treatment.—The symptoms may be combated, but otherwise little can be done. It has been suggested that a kidney might be removed, provided one only is involved, but generally the condition is bilateral and there seems no particular object to be gained by this procedure.

XIX. EMBOLISM OF THE KIDNEY

This is a common result of ulcerative endocarditis, but may occur where small clots, not necessarily containing pyogenic or other organisms, are broken off from aortic or mitral vegetations, or from calcareous plates on atheromatous arteries. The emboli are often multiple, and in cases of a pyaemic nature they may be found in the capillaries. The blood-supply of the affected area of kidney is arrested, and fatty changes ensue, and where suppurative organisms are present, acute necrosis, sometimes even gangrene, may result. In simple cases (where there are no suppurative organisms) the infarct gradually becomes absorbed, and is partially replaced by fibrous tissue, leaving a scar easily seen on the surface of the kidney. Infarctions are usually limited to the cortical portion of the organ.

Clinical Features.—There may be no symptoms at all, or the passage of an embolus to the kidney, with resulting infarction, may give rise to sudden local pain, and to the presence of albumin largely due to the appearance of blood in the urine, and in simple cases these symptoms may last for

one or two days. If organisms are present, rigors often ensue, and the pain is usually more severe. In pyaemic cases rapid death, mainly due to the general condition of the patient, is inevitable.

The **Diagnosis** is usually fairly easy where sudden pain is present, and where there is blood in the urine, together with evidences of valvular disease. In the absence of pain, and of blood in the urine, a simple infarction cannot be diagnosed.

The **Prognosis** depends on the presence or absence of organisms, and, if present, on their nature.

The **Treatment** is purely palliative.

Thrombosis of the renal veins may be part of a more general thrombosis. If not speedily fatal, the results as regard the kidney closely correspond to what follows passive congestion of the kidneys. The urine is diminished, is of high colour, and may contain blood.

Thrombosis of the renal artery may be due to injury or to disease of the vessel wall. Complete occlusion of the whole blood-supply implies necrosis. In cases of limited thrombosis it is associated with an embolus.

XX. PARASITES OF THE KIDNEY

The more important parasites have been referred to in a separate section (p. 197), but further reference is desirable to several of these.

1. **Hydatid Cysts.**—The cysts are generally found in the substance of the kidney: they may be under the capsule, or they may destroy the whole of one organ. They tend to discharge into the pelvis of the kidney, and may produce severe attacks of renal colic due to the vesicles which attempt to pass down the ureter. A large hydatid cyst may form a considerable abdominal tumour, readily palpable, although it is unlikely that the hydatid thrill, always of doubtful existence, can be definitely made out.

The **Diagnosis** frequently depends on aspiration, and the clear watery fluid of a specific gravity of 1005 to 1009, containing common salt, but no albumin, and often with hooklets and even scolices, is distinctive.

The **Treatment** consists in an attempt to empty the cyst, sometimes by aspiration, and sometimes by stitching it to the abdominal wall. A cyst may fill up after aspiration, or a neighbouring cyst may take the opportunity of growing rapidly. Unfortunately, hydatid cysts, if present in the kidney, are apt also to be in the peritoneum and elsewhere, and therefore the treatment is not invariably successful. If the cyst ruptures into the pelvis of the kidney, large quantities of fluid should be drunk by the patient, in order to aid the expulsion of daughter cysts by the ureter, and warm baths, together with the other treatment for renal colic, should be prescribed.

2. **Bilharzia Haematobia.**—This parasite is described on p. 207; it is a cause of haematuria, and generally the ova are passed from time to time in the urine, and, together with the ciliated embryos, may be readily recognised under the microscope. The ova may cause a tedious illness, with all the symptoms of cystitis. There is often haematuria and much discomfort, while calculi may form in both kidney and bladder. Peroneal fistula is not infrequent in chronic cases.

Treatment.—Mild cases may give rise to little trouble, but in many instances treatment is not very successful. Order alkaline drinks and oil of turpentine (♏ 15-20), or filix-mas (♏ 15-60).

3. **Filaria Sanguinis Hominis** is described on p. 204; it causes chyluria and haematuria, and may be associated with the development of elephantiasis.

4. **Eustrongylus Gigas.**—This is an enormous nematode, the female being 3 feet in length and the male 1 foot, of very rare occurrence in man. When it is present in the kidney, it causes complete disintegration of the organ.

5. **Pentastoma Denticulatum.**—The larval form of the Pentastoma taenioides, the latter being small worms a few inches in length, found in the frontal sinuses and nostrils of dogs, and very rarely in man. The larval form may occur in the kidney without apparently causing very much interference either with its structure or functions.

XXI. TUMOURS OF THE KIDNEY

1. **Simple Tumours** are by no means uncommon in the kidney. There may be small fibromata frequently seen near the apices of the pyramids, also lipomata and angiomata, while occasionally suprarenal gland tissue is enclosed inside the kidney substance. None of these tumours cause any symptoms as a rule, excepting the last, and then only in certain instances.

2. **Malignant Tumours.**—(1) *Sarcoma.*—*Primary* sarcoma is more common in children: it may grow to an enormous size, and sometimes a rhabdomyoma occurs which shows a remarkable similarity to striped muscle. Sarcomatous tumours may reach many pounds in weight—twelve pounds, even to thirty pounds—and may completely fill the whole abdomen. They grow rapidly and cause haematuria, a varying amount of pain sometimes due to the passage of clots down the ureter, and the development of a recognisable tumour on palpation.

Secondary sarcoma may infiltrate both kidneys, but does not give rise to such definite phenomena as a primary tumour.

(2) *Carcinoma.*—Cancers may be *primary* or *secondary*: the *primary* form is comparatively rare, but the tumour, when it does occur, may attain a great size, while *secondary* tumours cause comparatively slight symptoms. Cancers may be encephaloid and sometimes colloid.

(3) *Hypernephroma.*—These are tumours, generally malignant, which are in all probability formed from suprarenal "rests" or portions of suprarenal tissue enclosed in kidney substance. When they possess a malignant tendency they may simulate in structure carcinoma, sarcoma, etc., but the secondary growths in other organs bear a close resemblance to suprarenal tissue.

Clinical Features.—In nearly all malignant kidney tumours there are three cardinal symptoms: (1) pain often localised; (2) haematuria; and (3) the presence of a tumour on palpation situated in the kidney region.

The pain varies in different cases. It may be a dull ache increasing greatly on exercise, and it is sometimes of colicy nature due generally to blood-clots forming in the pelvis and ureter and passing down to the bladder. The

haematuria is peculiar. It is brought on by exercise, and when it commences it goes on for several days. A longer or shorter period may follow, during which there is no bleeding, and then it reappears. Some cases bleed periodically. The existence of a tumour is often easily made out on palpation, and it should be remembered that the colon lies in front of the kidney, and therefore in most cases a resonant note will be obtained over the renal neoplasm. Occasionally a renal tumour presses on the inferior vena cava or on the portal vein.

Diagnosis.—Palpation usually affords very definite information: the tumour growing from the region of the kidney, and lying behind the colon, is readily distinguished from an *enlarged spleen* which lies in front, or from *sarcomatous* involvement of the *retro-peritoneal glands*. In certain cases it may be difficult to distinguish a renal tumour from a *tumour* of the *liver*, but the liver tumour moves more freely downwards with the diaphragm, while a renal tumour is more definitely moored, and is not so readily displaced when the diaphragm contracts. The presence of blood in the urine, and blood often in great excess, and the passage of clots accompanied by colicky pain, generally render the diagnosis easy.

The **Prognosis** of malignant tumours is invariably serious; the duration of life depends, however, partly on the possibility of the affected kidney or kidneys carrying on the functions of nature, and partly on the exhaustion and emaciation which every malignant growth naturally causes. In certain cases renal arteries and veins are invaded by the tumour growth, and in this way a fatal result may be hastened.

The **Treatment** must be purely on palliative lines, the pain generally requiring sedatives and rest in bed. The application of ice locally, and administration of astringents internally, are often of value for haematuria. Order soothing liniments, and the use of belladonna or aconite internally, and, where absolutely necessary, opium, to alleviate the patient's suffering.

Surgical treatment is advisable in some cases, certainly only in primary tumours, and where there is a reasonable hope of the tumour being localised to the kidney, and to the kidney alone.

DISEASES OF THE NERVOUS SYSTEM

I. INTRODUCTORY

The Neurone.—The Nervous System is made up of neurones, and it is necessary to understand at the outset what a neurone is, and the different types of neurones which make up the central Nervous System. The neurone possesses a nerve-cell, an axone, and sometimes also grey processes or dendrites. The simplest conception of the nerve cell is a mass of protoplasm containing a nucleus in which there is a nucleolus. Both the cell and the nucleus possess a definite membrane. In the protoplasm of the cell there are bodies which take up basic stains, and which are termed "Nissl's granules." These granules are invisible *intra vitam*, but are easily stained and therefore seen after death, and apparently the functional activity of the cell depends much on the integrity of arrangement of these Nissl's granules. Of the processes, the axone or axis-cylinder is the chief path by which stimuli from or to the cell pass. Some cells have only a single axone or axis-cylinder, others, such as the cells of the posterior nerve-root ganglia, have a single axis-cylinder which, soon after leaving the cell, divides into two parts, one part stretching downwards into the nerve and the other upwards into the spinal cord. The axis-cylinder is made up of neuro-fibrillae, which have been comparatively recently discovered by special staining methods. These neuro-fibrillae which are present in the axis-cylinders do not all necessarily pass from a single neuronic cell, but some of them may reach the axis-cylinder process of one neurone although they originate in the cell-centre of another neurone, and it has been contended that these neuro-fibrillae can pass through the cell-centre of a neurone without any break in continuity, and might even thus be a direct link between one neurone and another

through the medium of a third neurone, which only acts as an intermediate link. Certainly some of the neuro-fibrillæ pass into the grey processes of the cells. The axone is surrounded by a sheath of myeline, which acts as an insulating medium, and all fibres which possess such a sheath are termed medullated. Practically all the nerve-fibres in the white matter of the brain and cord and in the peripheral nerves are medullated, and as the myeline sheath develops at different times in fœtal life, it is possible to distinguish certain tracts of the cord by embryological study. The myeline sheath is better supported in the peripheral nerves, where it is liable to suffer pressure from muscular contraction and other influences than in the central nervous system, and, further, the nerve-fibres of a peripheral nerve have a definite outer sheath termed the neurilemma sheath. This sheath is epithelial in nature, each envelope cell having a nucleus and being joined above and below by similar envelope cells. These cells, or at least their nuclei, have unquestionably a trophic influence on the axis-cylinder or axone, and in the process of regeneration of a divided or otherwise injured nerve it is unquestionably due to the attractive or, as it is called, chemiotactic influence of these neurilemma cells that the central axis-cylinder process or axone is guided into its old place in the peripheral portion of the nerve. The grey processes not present in posterior nerve-root ganglia cells, but which are an important feature in the cells of the anterior horn of the cord and in most of the nerve cells of the cortex of the brain, differ materially from the axone or axis-cylinder processes. They appear to be a part of the protoplasm of the cell extending into the neighbouring nerve-tissue, and Nissl's granules, which are such a prominent feature of the nerve-cell, extend for a short distance into the grey processes. Most of these grey processes branch and ramify in an intricate fashion, and doubtless help to bring the different neurones into functional union with each other.

Each neurone is probably a distinct entity, and it is doubtful whether either the axone or the grey processes actually anastomose with any neighbouring neurone, although, as already indicated, there is certain evidence in favour of such anastomosis. It has been contended by Sherrington that one neurone comes into indirect continuity with another

by a synapse. When a neurone is brought into close relationship with another neurone its axis-cylinder forms marked arborisations with processes of the second neurone.

Degenerative Changes in Neurones. According to Waller's Law an axis-cylinder process if separated from its trophic cell-body degenerates, and Waller proved that the cell-centre of each neurone is trophic for all the processes of that neurone. As a result of the division of a peripheral nerve from its trophic centres—if sensory, in the posterior nerve-root ganglia if motor, in the anterior horn of the cord, the peripheral portion degenerates. The axis-cylinders break up, the myeline sheath forms into numerous small balls, and the neurilemma nuclei proliferate. Marinesco asserts that the inner surface of the myeline sheath possesses a digestive ferment which aids in the complete removal of, at all events, the remains of the myeline.

The researches of Turck demonstrated degenerations in various tracts of the cord due to similar severance of nerve-fibres from their trophic cells. As a result of these degenerations certain of the tracts in cord and brain have been clearly differentiated.

It has also been shown that a process of a neurone cannot be divided without the cell-centre undergoing degenerative changes, and very similar changes result from the action of toxins or other detrimental influences, and even, to a less extent, by excessive fatigue. If a peripheral nerve be divided the following changes may be noted in the cells which are the neuronic centres: (1) the cell initially swells, although eventually it atrophies; (2) the grey processes, where present, diminish in number; (3) Nissl's granules undergo conspicuous alteration. Within twenty-four hours they become less definite, and the staining substance forming the granules tends to become diffused throughout the cell-body, a process termed *chromatolysis*. Within a few days after section, or after a very severe toxin has been in action, the chromatic substance may almost entirely disappear, not merely from around the nucleus, where chromatolysis usually begins, but from the whole of the cell. (4) The nucleus, which often becomes somewhat diminished in size, tends to assume an eccentric position, and may even be extruded entirely from the cell.

Most of the cells recover after such a lesion, although, of

course, where a toxin has produced these changes the virulence of the toxin is a very important factor.

There are various agencies by which neurones may undergo pathological change. (1) There may be separation of processes from neurone centres by haemorrhage, tumour, and so forth. (2) There may be toxins which act either in the blood or the lymph: a good example of such a toxin is that produced by the diphtheria bacillus. (3) Insufficient blood-supply to a part of the nervous system, whether by cardiac failure or disease of the arteries, is also a not infrequent cause of morbid change. (4) Insufficiency of oxygen, such as one finds commonly in anæmia or debility, may also affect the neurones in a detrimental way. (5) Amongst other possible morbid changes may be mentioned the presence of an excessive amount of carbonic acid or other waste products in the system. (6) Fatigue produces temporary changes which disappear after rest, but without sufficient rest such changes may become permanent. (7) Lastly it should be remembered that a congenital error or weakness may lead to far-reaching defects of the nervous system, especially when any strain is put upon it.

Regeneration of Axis-Cylinders.—The regeneration of axis-cylinders after division is a subject which cannot be discussed fully here. The accepted theory of regeneration is the *central* one, by which is meant that the central end of a divided nerve is the only one by which restoration of continuity can occur, by the down-growth of the axis-cylinder processes into the peripheral end of the nerve, and that the neurilemma nuclei in the peripheral end of the divided nerve are not at all neuroblastic, by which the regenerating axis-cylinders can be restored. It should be remembered however that both the central and peripheral theorists believe that the neurilemma nuclei have some attractive, partly chemiotactic influence, in aiding the central axis-cylinder in its growth outwards.

With a clear conception of what a neurone is, and how the life of the processes depends on the integrity of the cell centre, we can consider the various neurones in the central nervous system. There are (1) *sensory* neurones conveying impressions from skin, muscles, and so forth; (2) *motor* neurones which transmit motor impulses to muscles; (3) *association* neurones

which form a very large proportion of the neurones of brain and cord. These link together the various neurones, sensory and motor, and they are probably far more numerous than the sensory and motor neurones put together: (4) it has been suggested that there are also *intercalary* neurones by means of which all other neurones are brought into continuity, if not of substance, at least of function with each other.

Motor Fibres

The motor neurones may be divided into two groups—those belonging to the First or Upper Motor Neurone, and those belonging to the Second or Lower Motor Neurone.

I. The First or Upper Neurone, aptly called by Professor Wyllie "The First Trophic Realm," extends from the cell centre in the motor cerebral cortex lying just in front of the Fissure of Rolando, thence through the internal capsule, the crus, the pons, the medulla, and eventually reaches the cord. The greater number of the fibres pass downwards and decussate at the decussation of the pyramids in the medulla to reach the crossed pyramidal tract, while a smaller number pass down the same side of the cord by the direct pyramidal tract and decussate in the cord itself. A number of fibres of this upper motor neuronic system do not reach the cord, but pass to the nuclei of the motor cranial nerves in the pons and medulla.

II. The Second or Lower Motor Neurone or "Second Trophic Realm" has its cell centre either in the nuclei of the motor cranial nerves or in the anterior horns of the cord.

In a lesion of the First or Upper Motor Neurone, if it be irritative and involve the cortical motor areas, Jack-sonian epilepsy results, but if it be destructive it causes complete paralysis. A lesion of the pyramidal system in any part of its course is followed by degeneration of the pyramidal tract below the level of the lesion, and, provided the injury is not excessive, there is interference with voluntary control over the deep reflex arcs, and therefore exaggeration of the tendon reflexes will follow soon after the patient has recovered from the shock of the injury. This variety of paralysis is

termed *spastic*, and there is a great increase in the myotatic irritability.

A lesion of the pyramidal tracts causes diminution or loss of the superficial abdominal and epigastric reflexes, and there is extensor response instead of the usual flexion when the plantar reflex is tested (see p. 582). There may be considerable interference with the organic reflexes, sometimes retention and sometimes incontinence of urine and faeces.

In severe lesions of the spinal cord, such as may be induced by fracture of vertebrae or haemorrhage into the cord, the deep reflexes belonging to the distal segments of the cord are permanently abolished, and this is due to the loss of tonic influences usually carried by the rubro-spinal and vestibulo-spinal tracts which conduct important impulses from the cerebellum and the semicircular canals (see p. 584).

In a lesion of the First or Upper Motor Neurone the muscles do not waste except from disuse, and there are no qualitative changes in the electrical reactions, while with the exception of cases of severe transverse lesion just referred to, in which there may be flaccidity, there is a great tendency to spasm with contracture.

In a lesion of the Second or Lower Motor Neurone the lesion must either be situated in the nuclei of the motor cranial nerves or in the anterior horn of the cord, as in infantile paralysis, or it may involve the anterior nerve-roots or the motor fibres in the peripheral nerves. The nuclear cells or the cells in the anterior horn of the cord are trophic for the muscles which the nerve supplies, and therefore the muscles waste and the electrical reactions become greatly changed, and we find in fact the reaction of degeneration.

The Reaction of Degeneration.—The degenerated nerve is not stimulated by either the galvanic or faradic current, and the muscles cease to react to the faradic current after a very short time. Normally with the galvanic current applied to the muscle, on closing the circuit, the negative pole or cathode gives the first and strongest contraction, the positive pole or anode being distinctly weaker. This is expressed by the formula CCC is > AnCC. In the reaction of degeneration there is (1) a polar change, the anodal closing contraction being more powerful than the cathodal, so that AnCC is > CCC. With regard to the opening contractions, it is usually stated

that the anodal opening contraction is stronger than the cathodal and that in the reaction of degeneration this is reversed. In addition to the polar change (2) a weaker current causes contraction, and (3) the contraction, when obtained, is *much more sluggish* than it is in health. These phenomena constitute the reaction of degeneration, and they generally develop about seven to ten days after a lesion of the nerve supplying the muscle.

An *Incomplete Reaction of Degeneration* implies that the conducting capabilities of the nerve are not entirely destroyed. The faradic and galvanic excitability of the nerve may be only lessened, while in place of definite polar change, on testing the muscle, the closing contractions with cathode and anode may show equal excitability. Certainly the retention of faradic irritability is most important, and generally indicates that a rapid recovery may be anticipated.

The multipolar cells of the anterior horn of the cord are also centres of reflex action both for skin or superficial reflexes and also tendon or deep reflexes, while in the lumbar enlargement of the cord certain of these nerve-cells constitute the motor centres for the great organic reflexes of bladder and bowels. The deep reflex arcs are under the control of centres in the brain, and the tracts by which these controlling impulses pass downwards are in part the pyramidal tracts, and in part the rubro-spinal vestibulo-spinal, and perhaps other tracts. A lesion of the pyramidal tracts generally causes spastic paralysis with exaggerated knee-jerks, because inhibitory stimuli from the brain are interfered with. On the other hand, a lesion of the cells in the anterior horn of the cord, damaging the great trophic centres for muscles and also the motor centres of the reflex arcs, causes abolition of both superficial and deep reflexes. If the lesion involves the lumbar enlargement, the organic reflexes of bladder and bowels also suffer.

Myotatic Irritability.—It was formerly supposed that upon tapping a tendon such as that of the quadriceps extensor muscle a message was sent by way of sensory fibres to cells in the anterior horn of the cord, from which in turn a stimulus travelled by the sciatic nerve to the muscle, causing contraction. It has been proved that the time taken for the passage of these nerve stimuli exceeds in length the period between the tap on the patellar tendon and the eliciting of the resultant

knee-jerk. Gowers therefore suggested that the muscles might be kept in a state of tonus by the integrity of the reflex arc, and that it was not necessary for a message to travel upwards to the cord and downwards again to the muscle in order to bring about muscular contraction, but that the muscle, being in a state of tonus or myotatic irritability, contracted directly upon the tapping of its tendon.

Hypertonus implies spasticity and often rigidity of the muscles. It is less apparent when passive movements are slowly executed, but becomes greater if the movements are performed quickly and particularly if volition on the part of the patient enters into the movement. In cases of hypertonus the tendon responses are greatly exaggerated. Ankle clonus and patellar clonus are generally present, and should the condition depend on a lesion of the pyramidal tracts there is plantar extension. Rigidity is only present in organic cases, but exaggeration of the tendon responses without any rigidity may be seen in cases of neurasthenia, hysteria, etc., without any plantar extension.

Hypotonus or decrease of the tonus of the muscle is the exact opposite of hypertonus. There is atony and generally wasting of the affected muscles and a remarkable adaptability of the joints to passive movement and usually also diminution or absence of the tendon responses. The joint ligaments are so extraordinarily relaxed that hyperextension of joints can be easily accomplished, and the legs can be placed in many unnatural positions. The diminution or absence of the knee-jerk is a feature in these cases. Hypotonia is well seen in many patients with locomotor ataxia.

The Reflexes, Superficial and Deep

It is important to remember the segments of the cord to which the various superficial and deep reflex arcs belong, and in the short tables appended, reference is made to the mode of eliciting these reflexes.

[Table

Table of Superficial Reflexes

1.	Scapular reflex.	5th cervical to 1st dorsal segments.	Is elicited by drawing the finger over the scapula, causing contraction of the scapular muscles.
2.	Epigastric reflex.	5th dorsal to 9th dorsal segments.	Is elicited by drawing the finger across the hypochondriac region when the epigastrium is indrawn.
3.	Abdominal reflex.	10th dorsal to 12th dorsal segments.	Is elicited by drawing the finger down the lateral aspect of the abdominal wall, causing contraction of the abdominal wall.
4.	Cremasteric reflex.	1st lumbar to 2nd lumbar segments.	Is elicited by tickling the inner aspect of the thigh, causing contraction of the cremaster muscle with drawing up of the testicle.
5.	Gluteal reflex.	4th lumbar to 5th lumbar segments.	Is elicited by drawing the finger along the gluteal fold, causing contraction of the gluteal muscles.
6.	Plantar reflex.	1st sacral to 2nd sacral segments.	Is elicited by tickling the sole of the foot which causes flexion of the toes.
7.	Anal reflex.	5th sacral.	Pricking the skin near anus or introducing finger through sphincter.

The *Plantar Reflex* is so important that it requires a separate description. In health in all persons who have passed the age of infancy, drawing the finger nail or the handle of a pen along the outer side of the sole of the foot or across

the sole causes flexion of the great toe, and with a stronger stimulus, all the other toes, while the foot is inverted. The test should be carried out with the knee bent and all the muscles relaxed, and this can be done by making the patient lie on his side. The feet must also be warm. Where there is a lesion of the pyramidal tracts, the great toe is extended instead of being flexed (termed "plantar extension," and this constitutes the *positive Babinski sign*. Plantar extension is

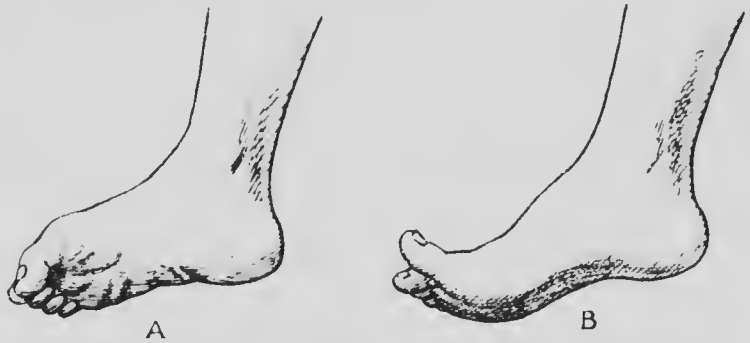


FIG. 31.—A shows normal flexion of great toe; B shows plantar extension (positive Babinski sign).

obtained in healthy infants before they have learnt to walk before the pyramidal tracts have become medullated, and, along with plantar extension of the great toe, there is often separation of the smaller toes. Gordon showed that plantar extension could be obtained in cases of lateral sclerosis by forcible pressure exerted on the calf muscles (the leg muscles being relaxed), and Oppenheim obtains the same result in similar cases by drawing the knuckle down the inner side of the tibia. It is claimed that both Gordon's and Oppenheim's methods of obtaining plantar extension show the alteration at an earlier period than by Babinski's modus operandi.

Table of Deep or Tendon Reflexes[1]

1.	Biceps-jerk.	5th cervical to 6th cervical segments.	Elicited by tapping the tendon of the muscle.
2.	Supinator longus-jerk.	6th cervical to 7th cervical segments.	Elicited by tapping the radius, the elbow being kept slightly flexed.
3.	Triceps-jerk.	6th cervical to 7th cervical segments.	Is elicited by tapping the triceps tendon with the arm flexed.
4.	Knee-jerk.	3rd lumbar to 4th lumbar segments.	Is elicited by tapping the patellar tendon, causing extension of the leg at the knee-joint.
5.	Achilles-jerk.	1st sacral to 2nd sacral segments.	Is elicited by tapping the Achilles tendon causing contraction of the calf muscles.
	Ankle clonus or rhythmic contraction and relaxation of calf muscles.	1st sacral to 2nd sacral segments.	Is elicited by suddenly putting these muscles on the stretch.

There are several *efferent* tracts in the cord which should be described because they render intelligible what has been stated about myotatic irritability and the reflexes.

1. The Vestibulo-Spinal Tract. The vestibular nerve terminates in Deiter's nucleus, and from the nuclear cells the vestibulo-spinal tract passes down the antero-lateral column of the cord to end in arborisations around the spinal motor neurones. They convey impressions from the semi-circular canals to the cervical, dorsal, lumbar, and sacral spinal motor

[1] The following reflexes are of less importance, but might be added:—(1) The Adductor-jerk is obtained by abducting the thigh and tapping the adductor magnus tendon. A crossed adductor-jerk has been sometimes noted. 2. The Jaw-jerk is elicited by making the patient open the mouth, placing one finger on the chin and tapping sharply on it, when the jaw muscles contract, should the jerk be present. It is absent normally.

neurones of the same side. These impressions have much to do with the maintenance of the tonus in muscles.

2. The Rubro-Spinal Tract. This tract arises from the red nucleus. The fibres decussate and pass down the lateral column of the cord as seen in the figure of the cord (Fig. 42) and form arborisations near the base of the anterior horn. These fibres probably convey cerebellar impulses to the spinal motor neurones.

3. Closely allied to the rubro-spinal fibres is another system which belongs to the posterior longitudinal bundle. The tract appears to be connected with the optic fibres, and it is probable that both the rubro-spinal and this bundle just referred to convey from the optic tracts impressions to the spinal motor neurones which help to maintain tonus.

4. A system of fibres arises in the corpora quadrigemina, which may be traced down as far as the sacral region, and it probably exercises an important function in controlling the bladder and rectum.

Sensory Fibres

Sensory fibres are infinitely more complicated in their arrangement than the motor. Not merely are there a number of different fibres which subserve different kinds of sensation, but the pathways pursued by these fibres are much more involved than is the case with fibres subserving motor impulses.

According to recent researches by Head, there are three varieties of sensory fibres which convey different sensations in the peripheral nervous system.

1. There are fibres which subserve *deep* sensibility. These fibres convey the impressions of *passive movement* and *position*, and may be tested by deep pressure, which, if excessive, causes pain. These fibres run with motor nerves and are distributed to muscles, joints and periosteum.

2. There are fibres which subserve what Head calls *protopathic sensibility*. Such fibres respond to *painful cutaneous stimuli* and to the *extremes* of *heat* and *cold* (−25° C. and +45° C.). They also endow the hairs of the skin with sensibility to painful stimuli. There is no appreciation of position, that is, accurate locali-ation, gained by protopathic sensibility. These fibres regenerate rapidly when the ends

of a divided nerve have been re-united, and conductivity returns in 7 to 10 weeks.

3. There are fibres which subserve *epicritic sensibility*. These fibres carry stimuli produced by the *lightest touch* and the *finer grades of temperature* (25° C. 45° C.) and they enable one to appreciate *accurate localisation* and to recognise the two points of a pair of compasses when placed in close apposition. These fibres regenerate very slowly and after suture of a divided nerve do not regain their normal conductivity until six months at least have elapsed.

After section of a peripheral nerve there is loss of protopathic sensibility over the area which corresponds to the cutaneous distribution of the nerve, while epicritic sensibility is lost over a distinctly larger area. In the overlapping epicritic zone there is great discomfort experienced from a painful stimulus, but the site stimulated cannot be localised. Where there is nerve injury but not complete division epicritic sensibility is generally most markedly affected.

The more centrally is the site of the injury situated, the smaller becomes the overlapping epicritic zone. In root lesions the protopathic involvement may even be greater than the epicritic. Head has therefore stated that the peripheral nerve represents the unit of epicritic sensibility, while the posterior nerve root is the unit of the protopathic.

The fibres which carry *pain* and *temperature* sense impressions decussate almost immediately on entering the spinal cord. Those conveying *tactile* and *pressure* impressions mainly cross, but the decussation is only effected after the fibres have passed upwards, for a varying distance on the side of the cord on which they have entered.

The Three Systems of Sensory Fibres

1. **The First System of Sensory Neurones.**—Each segment of the spinal cord is connected through the posterior nerve roots with a posterior nerve root ganglion, from which it receives by the posterior nerve roots fine and coarse fibres. *The fine fibres* probably conduct sensory impressions from the skin, and after entering the cord pass upwards in Lissauer's tract for a short distance before entering the grey matter of the

posterior horn of the same segment. They form the sensory limb of the *superficial reflex arcs*.

The coarse fibres conduct sensations from deeper structures such as muscle, bone, and so forth, and they may be divided into three groups: 1. Short fibres which pass through the posterior columns for a short distance prior to entering the posterior horn. They form the sensory limb of the *deep reflex arcs*. 2. Fibres of medium length which pass upwards in the postero-external column and eventually reach the cells of Clarke's column. Their function obviously concerns *afferent cerebellar impulses*. 3. There are long fibres which pass upwards in the postero-external and postero-internal columns.

The afferent fibres of the sensory cranial nerves also belong to this system.

II. **The Second System of Sensory Neurones** carries sensory impressions from the termination of the first system upwards mainly towards the optic thalamus. Sensory impressions are conveyed by this second system from the spinal cord by 1. the mesial fillet and 2. the spino-thalamic system.

(1) The fibres which have their origin in the nuclear structures in which the tracts of Goll and Burdach terminate, mostly decussate and enter the inter-olivary layer. The mesial fillet is the continuation of this layer, and passes through the medulla and pons to reach the optic thalamus. By means of this tract, sensory impulses conveyed by the posterior columns of the cord are conducted upwards. A lesion of the mesial fillet does not cause any interference with *tactile sensation* or *pain*.

2. The spino-thalamic system is made up of neurones the cell centres of which are in the posterior horns of the cord. The fibres decussate and pass up, in or near, the antero-lateral tract of Gowers' and terminate after passing through the medulla and pons in the ventral part of the optic thalamus. This tract receives contributory fibres from the sensory cranial nerves, and especially the 5th, during its passage through the medulla and pons. Impressions of *touch*, *pain*, and *temperature* are carried upwards to the optic thalamus by this tract.

III. **The Third System of the Sensory Neurones** includes those which carry sensory impulses from the optic thalamus

to the cerebral cortex. Some of these go to the ascending parietal convolution constituting probably the cortical termination of the mesial fillet. A second tract passes to the taleiform lobe and contains fibres which are situated in the posterior portion of the posterior half of the internal capsule close to the fibres forming the optic radiation of Gratiolet. This system of fibres conveys the impressions of *touch*, *pain*, and *temperature* conveyed by the tracts in the antero-lateral column.

Apparently all sensory tracts pass upwards to the ventro-lateral portion of the optic thalamus, because destruction of the optic thalamus causes complete loss of *all sensations* on the opposite side of the body, and in addition *loss of muscular sensation* and the *recognition of position* of the limbs.

II. DISEASES OF NERVES

1. Local Neuritis

Inflammation may arise in various structures of a nerve.

(1) In the outer fibrous sheath surrounding the funiculi called *perineuritis*.

(2) In the delicate connective-tissue fibres dividing up the constituent nerve elements in the funiculi, called *interstitial neuritis*; or

(3) An inflammation or degenerative change actually involving the nerve fibres themselves, called *parenchymatous neuritis*.

The term *interstitial neuritis* is generally used to include perineuritis.

It is rare to find parenchymatous neuritis alone, without a certain amount of interstitial change. Parenchymatous neuritis is best exemplified by multiple or peripheral neuritis, generally of toxic origin, and described separately.

Etiology. — A localised perineuritis commonly results from rheumatism, gout, or exposure to cold. A nerve may also be injured by wounds or bruises, fractures, tumours, abscesses and so forth, and in these cases a perineuritis is, at all events first produced, although later the funiculi may be involved. Toxic neuritis is referred to separately under multiple neuritis but where a nerve is affected by some local condition, which

alone might be insufficient to produce a neuritis, a toxin capable of acting on the peripheral nerves may specially devote its attention to those nerves which are below par. And in the same connection it may be stated that, in alcoholic neuritis, exposure of one or more limbs to great cold may specially predispose to an attack on the nerves of these limbs.

Pathological Anatomy. In perineuritis and interstitial neuritis there is generally an exudation into the connective tissue; sometimes it is of inflammatory lymph, occasionally there is an invasion of leucocytes, and in very acute cases haemorrhages are not infrequent.

Inside the funiculi similar interstitial changes may be found. The parenchymatous changes in the nerve fibres are described under multiple neuritis.

Clinical Features.—In some cases there is a marked degree of constitutional disturbance, but locally the chief feature is pain, and pain shooting down the line of the nerve. It increases with pressure over the nerve, and is generally more severe at night. The skin may be hyperaesthetic, and later in the affection, anaesthetic. Sometimes it is possible to make out on digital examination that the nerve is definitely swollen. Should the nerve carry motor fibres, the muscles become weakened and eventually paralysed, and muscular tenderness and sometimes fibrillary twitching may be noted.

The trophic changes vary considerably: the skin may be reddened and swollen, sometimes it is thin and glossy; the nails may become brittle and even fall off. Local sweating, herpetic eruptions, joint swellings, and other trophic changes are not uncommon.

Diagnosis.—In most cases the diagnosis is easy, although the exact nature of the etiological factor may not be clear. The possibility of rheumatism and gout, as well as syphilis, and, of course, exposure to cold, which is the most frequent cause, should be carefully investigated.

The **Prognosis** varies considerably. In some cases benefit rapidly ensues from rest and treatment. In other cases where there has been much exudation, a long period elapses before the nerve can resume its normal functions.

Treatment.—Rheumatism, gout, and syphilis should be appropriately treated; rest must be ordered for the affected part, and warm applications applied along the line of the

nerve. The pain may be so excessive as to demand the use of opium or morphia, and sometimes local counter-irritation (provided no trophic change is in evidence) may be applied; such remedies as iodine and fly-blisters being of great service. Potassium iodide is invaluable, and the constant current not merely relieves pain, but also helps to remove exudate. In some cases an attempt may be made to puncture the nerve by the method of acupuncture referred to under sciatica.

2. Ascending Neuritis or Neuritis Migrans

A remarkable form of neuritis in which the inflammatory process generally travels up the nerve, causing intense pain sometimes so excruciating as to determine the patient in favour of amputation. It is generally due to a septic wound or, at all events, a wound of the nerve peripheral to the ascending inflammation, and the presence of an irritant of some kind is essential.

In these cases the best treatment is to search at the site of the injury for a neuroma, or other possible source of irritation, or to divide the nerve; in some instances treatment fails, and amputation of the limb affected has to be performed.

3. Sympathetic Neuritis

This condition implies irritation or inflammation of the corresponding nerve on the opposite side of the body, secondary to the primary involvement of a nerve by an abscess, tumour or other lesion.

4. Multiple Neuritis or Peripheral Neuritis

This is a symmetrical and peripheral neuritis, the lesion being best marked near the peripheral end of the nerves. Although probably in most cases the whole neurones suffer, the first clinical evidences of involvement are found peripherally. Nearly all the forms are true parenchymatous neuritis.

Etiology. The following grouping of causes will be found helpful: 1. Diffusible stimulants, as alcohol, naphtha, bisulphide of carbon, dinitro-benzine, etc.; 2. Metallic poisons, as arsenic, lead, mercury; 3. Toxins from the micro-

organisms of diphtheria, typhoid, rheumatism, septicæmia, syphilis, pneumonia, tubercle, malaria, beri-beri, leprosy, etc. It may also occur in diabetes mellitus, possibly in gout, in cachexia, cancer, and sometimes in profound anæmia.

Cold has been described as a cause of the condition; probably it is an important exciting cause where alcohol or other predisposing factor is present. Similarly physical ill-health or mental depression has a potent influence. In the above groups certain of the lesions are primarily rather an *interstitial* than a parenchymatous neuritis.

Pathological Anatomy. The myelin and the axis-cylinders of the affected nerve fibres break up, the neurilemma nuclei proliferate, and there are almost invariably two associated conditions seen in fatal cases, namely, a marked degree of thickening of the coats of the arteries and capillaries in the nerve, and exudation inside the funiculi, sometimes hæmorrhagic, sometimes leucocytic but mostly inflammatory lymph. The cell centres belonging to the affected neurones show chromatolysis, eccentric position, and sometimes even extrusion, of nuclei.

In the muscles there are also marked secondary changes including loss of striation, fatty degeneration, and proliferation of nuclei. In cases of sufficiently long standing we believe there is evidence of peripheral regeneration of nerve fibres in the affected nerves.

Clinical Features.—As a type it is best to take alcoholic neuritis. This often occurs in women, and is common in drinkers of all classes, but co-existing ill-health is a frequent and most important predisposing cause.

There are different types—such as sensory, motor, etc.—indicating a predilection of the toxin for certain neurones, and the condition may be acute, subacute, or chronic. The early *Sensory* symptoms are pain, tingling, and numbness, and cramps and pains on pressure over nerves and muscles: the chief *Motor* symptom is paresis of the extensors of hands and feet causing wrist-drop and foot-drop.

The *Sensory Phenomena* are, besides the symptoms mentioned above, delay in conduction of sensations, the patient often crying out a few seconds after the muscle or nerve has been pressed, and sometimes alteration of the temperature sense.

The *Motor Phenomena* are, besides wrist-drop and foot-drop, high-steppage gait, weakness of muscles of back and neck, and tremor or rather tremulousness of the limbs. Death may be due to involvement of the phrenics and the intercostal nerves, sometimes the vagi. In the leg, the extensors, tibialis anticus, and peronei suffer specially and generally first of all. Inco-ordination is a marked feature in certain cases. Contracture is not an uncommon sequel involving the less affected muscles, such as those of the calf.

The *Reflexes.*—The knee-jerk and wrist-jerk are usually lost, but to begin with may be sometimes exaggerated. The superficial reflexes soon disappear, even if exaggerated at first. The organic reflexes are generally unaffected, except in very severe cases or as a late phenomenon of grave import.

The *Electrical Reactions* are diminution to faradic stimulation, and the reaction of degeneration in the affected muscles (see p. 579).

The *Vasomotor Phenomena* include pallor, redness of hands and feet, oedema of ankles, and a profuse and generally ill-smelling perspiration.

Trophic Functions.—The affected muscles waste, the affected skin may become glossy, the nails brittle, and bed-sores are to be dreaded in severe cases.

The *Special Senses* are usually normal.

Mental Condition.—There is great loss of memory, and hallucinations, often very vivid, are almost constantly present.

There are cases, generally more chronic, which belong to an ataxic type. This is due, in all probability, to the toxin involving more particularly the deep sensory fibres which subserve movement and the position of the limbs. The legs are chiefly affected. The epicritic and protopathic fibres are less involved in this type.

Cardiac weakness is a serious complication, and the lungs must be regularly examined because a low type of pneumonia may occur. Effusions into the serous sacs are not uncommon. The temperature varies: in acute cases it may be high, 102° to 103° F., in more chronic cases it may never be above normal. Insomnia is apt to give much trouble.

The duration is uncertain; in an acute case death may occur from involvement of respiratory muscles, vagi, or heart in 10 to 14 days, while a chronic case may take weeks to

reach its climax, and months before complete recovery is attained. Pain and tenderness disappear first, and sensation returns long before motor power.

Diagnosis. The symmetrical distribution and the pain on pressure over nerves and muscles usually render the diagnosis clear, because in *infantile paralysis, locomotor ataxia, Landry's paralysis* and *hysteria*, these phenomena do not correspond. On the other hand, in very acute cases the disease may be almost indistinguishable from Landry's paralysis.

Prognosis.—Very acute cases are generally fatal. Pulmonary and other complications must be guarded against, and their appearance is serious. Chronic cases are favourable, but may be extremely slow as regards recovery. Returning faradic irritability is a sure sign of restoration of function.

Treatment.—Remove the cause, absolutely confine the patient to bed for a time, and feed carefully.

Hot fomentations are most helpful, and sometimes hypodermics of cocaine or even morphia. Bromides and chloral may be given to soothe the mental excitement, and as soon as possible, *i.e.* when the inflammation in the nerve is arrested, begin massage, daily hypodermics of strychnine (gr. $\frac{1}{60}$), and electricity, both faradic and galvanic. Sand-bags are of value, where there is foot-drop, to correct any tendency to contracture.

2. Arsenical Neuritis.—(See Arsenical Poisoning, p. 191.)

The **Clinical Features** closely correspond to the alcoholic type, the legs suffering more than the arms. The earliest symptoms include paraesthesia and pains in the legs, and later paralysis and sometimes ataxia develop. The paralysis is symmetrical and affects the extensor muscles much more than the flexor. There is typical foot-drop and wrist-drop. The hyperaesthesia of muscles and nerves resembles what is found in alcoholic cases. The tendon reflexes are abolished.

The **Diagnosis** is usually easy and much help is gained from a knowledge of the other results of arsenic poisoning and specially the gastro-intestinal irritation and the pigmentation of the skin.

The resulting paralysis is apt to persist for a longer time than in alcoholic cases.

3. Lead Paralysis.—(See Lead Poisoning, p. 189.

The following types of paralysis occur, and the table appended may be useful for reference:—

(1) Antebrachial type, in which there is paralysis of the extensors of the fingers and the wrist; but the supinator longus and extensor of thumb escape. The typical wrist-drop results.

(2) Brachial type, including paralysis of the deltoid, biceps, brachialis anticus, and supinator longus, but the pectoral muscles escape.

(3) Duchenne-Aran type, in which the small hand-muscles are affected, as in progressive muscular atrophy.

(4) Peroneal type, in which the peronei, the extensor longus digitorum, and the extensor of the great toe are affected, causing foot-drop.

(5) Laryngeal type, in which the adductors of the glottis suffer.

Remember that the arms suffer most frequently, and that the supinator longus and the extensor muscles of the thumb usually escape.

4. Diabetic Neuritis.—The legs are more affected than the arms, and the external popliteal nerve is affected early. There is some pain on pressure over muscles and nerves, but less paraesthesia. Trophic sores sometimes develop on the feet, but are painful and therefore unlike the anaesthetic ulcers seen in locomotor ataxia.

There is little difficulty in the **Diagnosis.** The presence of sugar in the urine, the absence of the Argyll-Robertson pupil, and a fact recorded first by Williamson, that there is loss of the vibration sense, prevent confusion with locomotor ataxia.

5. Diphtheritic Neuritis.—This is a parenchymatous neuritis due to the neuro-toxin of the Klebs-Loffler bacillus. The motor neurones appear to suffer most, and the pathological changes in the nerves are best seen in the intra-muscular branches.

As stated on p. 46, the paralysis occurs in 6 to 20 per cent of all cases of diphtheria, but there is no relationship between the severity of the throat lesion and the probability of paralysis following. Unquestionably the early application of antitoxin treatment has greatly reduced the frequency of post-diphtheritic neuritis.

Clinical Features.—The soft palate is first paralysed, and as a result fluids tend to regurgitate through the nose. The

soft palate will be found to be insensitive to stimulation. In many cases nothing further follows, and the paralysis gradually disappears. Sometimes, however, abductor or other laryngeal paralysis develops, in the former case greatly endangering the patient's life.

Paralysis of the laryngeal muscles, the eye muscles especially the external rectus (6th nerve), paralysis of the limbs, and rarely a more extensive paralysis may either follow the palatal paralysis or replace it. Where death occurs due to paralysis, it is usually the result of the involvement of the heart muscle and sometimes of the diaphragm.

The deep reflexes are usually affected in severe cases, the knee-jerks being either diminished or lost.

The **Diagnosis** is generally easy. A definite history of sore throat, and particularly the infective type of sore throat, prevents any possible mistake.

The **Prognosis** is favourable in all slight cases. In the graver forms of paralysis, and especially where the heart muscle is much involved, the neuritis presents a grave danger.

The **Treatment** does not differ much from that already prescribed for alcoholic neuritis. The necessity for preventing fatigue has already been insisted on in the treatment of diphtheria, and especially preventing the patient sitting up in bed and so risking cardiac failure. Sometimes in cases of laryngeal paralysis it may be necessary to prevent food particles from entering the larynx, and the patient may require to be fed by a stomach tube. Strychnine hypodermically and, needless to say, the application of electricity, galvanic, faradic, or both, constitute the best treatment for most cases.

5. Tumours of Nerves

Neuromata.—These tumours either consist of nerve fibres, sometimes even of nerve cells, in which case they are called "true neuromata," or else they are composed of connective tissue constituting the far more frequent "false neuromata." True neuromata are usually found in connection with the sympathetic nervous system. They consist of nonmedullated nerve fibres and ganglion cells. They cause no clinical symptoms. Gliomatous tumours closely resemble true neuromata in appearance, although microscopically they are easily distinguished.

1. Multiple Neuromata are tumours intermediate between false and true neuromata. They occur on many of the nerves of the body, and occasionally give rise to considerable pain when the tumours are palpated.

2. Multiple Neurofibromata.—These growths, described by, and called after, von Recklinghausen, are amongst the most remarkable tumours in existence. The essential features are:—

(1) Numberless small fibrous nodules, some sessile, others pedunculated, which are situated in the skin. These vary in size, and may be extremely large.

(2) Tumours on the nerve trunks, very similar to those described under multiple neuromata, and they cause considerable pain, cramps, and even paralysis.

(3) Pigmentation of the skin, occurring in patches, which may be found on any part of the body. The patches vary considerably in size.

(4) There are many sensory and motor phenomena, such as pains, cramps, and paralysis, due to the presence of the nerve tumours; in addition, marked mental changes appear, including loss of intellectual power and difficulty in speaking.

The **Prognosis** depends on the possibility of the removal of the tumours which are causing the pain or paralysis just referred to.

3. Plexiform Neuromata are tumours made up of a tortuous bunch of nerve fibres, with often a considerable amount of myxomatous tissue around the constituent nerve bundles. They are frequently met with in connection with the 5th nerve, but occur on many of the nerves of the body. They are almost invariably the result of a developmental error.

4. Amputation Neuromata.—When a limb has been amputated, a bulbous swelling forms on the central end of the divided nerves. These swellings consist largely of axis-cylinder processes which have grown downwards and become convoluted, and they are surrounded by a network of fibrous tissue. The existing axis-cylinders at the terminal end of the divided nerve unquestionably form numerous young axis-cylinders by a process of fission, so that in the neuroma there are actually more axis-cylinders than in the nerve just above the level of section. These neuromata cause pain if they

so situated as to be pressed upon in the stump, and it is prudent in performing amputations to divide the nerves as high up as possible. Occasionally a painful amputation neuroma requires to be removed.

Carcinoma and sarcoma may also occur on nerves, but with the exception of the tumours above mentioned no other neoplasm requires reference.

6. Neuralgia

Nerve Pain. It may be associated with definite inflammation of the nerve affected, or there may be no recognisable pathological change present. It may, therefore be of organic or purely functional origin. Pressure on a nerve may also cause pain.

Etiology.—The functional cases are common in debilitated and anaemic persons, and in neurotic and gouty individuals, and they frequently follow in the train of a continued fever, and especially influenza: while the immediately exciting causes include a carious tooth, cold, the act of mastication, gastric disturbances, and so forth.

Pathological Anatomy.—Although there is usually no visible change in the affected nerves in so-called functional cases, there may be swelling of the nerve and even changes in the central cells.

Clinical Features.—The pain is aching or lancinating: it may be paroxysmal and almost unbearable, or in other cases comparatively slight. There are painful points corresponding to the positions of foramina through which the nerve emerges, and the affected skin may be swollen and hyperaesthetic. The hair belonging to the area may even change colour, and there may be local sweating. The pain lasts for hours or for a whole day, and there is often a periodicity about its recurrence.

Types of Neuralgia.—(1) Neuralgia of the 5th Nerve. Tic Douloureux.—(Compare figure of nerve distribution on page 629.) Any of the three divisions may be involved. When the *first* or *ophthalmic division* is affected, supra-orbital pain is the most common manifestation, and pressure over the supra-orbital notch greatly aggravates it. The conjunctiva is often inflamed, and tears may flow freely. Not infrequently

herpes frontalis (the lesion being in the Gasserian ganglion) is associated, and even iritis. Almost in every case the eyeball is painful. Where the *second* or *superior maxillary division* is involved, pain is felt over the point of emergence of the nerve at the infra-orbital foramen, and the upper teeth on the affected side are painful. In these cases, the existence of dental caries and the condition of the antrum should be investigated. Where the *third* or *inferior maxillary division* is involved, there is often pain felt either in connection with the mental foramen and lower teeth, or the auriculo-temporal branch behind the ear. Not uncommonly there is twitching of facial muscles associated with very severe cases.

Paroxysmal cases of trigeminal neuralgia are called *epileptiform*, and the almost continuous paroxysms of agonising torture give scope for the ingenuity of the physician in trying to afford relief.

The **Diagnosis** really implies in most cases an attempt at discovering the cause of the pain.

The **Prognosis** depends on the recognition of the cause and its treatment, successful or the reverse.

The **Treatment** is twofold. Find out any definite causal factor, and treat it. Give iron and arsenic for anaemia, salicylates for rheumatism, colchicum for gout, and quinine for malaria. Examine the nose and teeth, treat any gastric disturbance present, and attend to the general health.

The special remedies for the relief of the pain are the analgesic group, of which the best are—quinine (gr. 5-10), antipyrine (gr. 10-20), phenacetin (gr. 20), antifebrin (gr. 5-15), butyl-chloral hydras (gr. 10-20), and exalgin (gr. 2-3). Aconite, bromides, and tincture of gelsemium are also useful. Always be careful to avoid depressing the patient too much; in certain cases a diffusible stimulant, and especially alcohol, works wonders. Locally, chloral and camphor in equal parts may be painted on, or menthol, veratrine, or aconitine ointments rubbed gently over the site of the pain. Local hypodermics of osmic acid (1 or 2 drops of a 1 per cent solution), cocaine, or morphia are sometimes used. Galvanism is worth a trial, the positive pole being applied to the painful part, and the negative pole placed over the vertebral column. Schlosser has advocated the injection into the nerve of 80 per cent alcohol in water. By one

puncture through the gum just behind the wisdom tooth and dextrous alteration of the direction of the needle he has injected each of the three divisions of the nerve by one puncture.

In severe and persistent trigeminal neuralgia, nerve section, or even the removal of the Gasserian ganglion, has been recommended.

Neuralgia of Certain of the Spinal Nerves.—Many of the spinal nerves may give rise to neuralgic pain, the initial and causal lesion being tumours, injuries, pressure, syphilis, and, in some cases, exposure to cold. Certain of these neuralgias demand special reference, and many of them are in reality cases of neuritis.

(2) Neuralgia of the Breast or Mastodynia.—This is a neuralgia of the branches of the intercostal nerves supplying the skin or the gland structure of the breast. It may result from excessive stimulation, as in over-lactation or in pregnancy, but it is also not infrequently associated with menstrual irregularity or hysteria. There are generally tender points suggestive of the cutaneous distribution of the intercostal nerves. Common-sense treatment with the local use of sedatives, affords relief.

(3) Brachial Neuralgia.—This form of neuralgia is sometimes the result of an injury, but is more likely to occur in persons predisposed by rheumatism, gout, anaemia, or debility. The pain may be very excessive. It should be remembered that pianists' and telegraphists' and other trade cramps are often associated with neuralgic pain, specially experienced in the nerves of the brachial plexus, mostly confined to the shoulder, but sometimes shooting down to the fore and middle fingers. Rheumatoid arthritis should also be kept in mind as a possible causal factor. The treatment should be on general lines for neuralgia; sometimes local counter-irritants or sedatives are efficacious, although in many cases rest to the affected arm is of the greatest value.

(4) Intercostal Neuralgia is almost invariably diagnosed by the three painful points or areas which correspond to the posterior, lateral, and anterior cutaneous branches of the nerve. Very commonly herpes zoster is associated with the pain, and especially in cases of great severity and long duration.

(5) Lumbo-Sacral Neuralgia.—Just as in intercostal

neuralgia there are usually three painful points, so the cutaneous distribution of the nerves of the lumbar plexus often gives rise to pain in the three positions where the superficial branches come to the surface—that is to say, (1) close to the spinal column, (2) in the lateral region, and also (3) anteriorly, near the middle line.

The causes are various. There are generally *predisposing* causes, such as anaemia, ill-health, or neurasthenia, and also local or *exciting* causes, such as a pelvic tumour, constipation, etc.

The **Diagnosis** is by no means easy. The physician has to remember the possibility of some malignant growth, and that the neuralgia very frequently has an organic cause.

(6) SCIATICA may be either a purely functional neuralgia, or it may be a definite perineuritis or interstitial neuritis.

Etiology.—It is most common in adults and in males. It is closely associated with rheumatism and gout, and certainly exposure to cold or wet in a rheumatic subject is extremely apt to induce an attack. Many cases of sciatica are really interstitial neuritis and not merely a functional neuralgia.

Pressure on the sciatic nerve, whether in the pelvis or on the sacral nerve roots, or less usually in the line of the nerve, causes sciatica: but although the nerve may actually be compressed, the pain is generally of neuralgic nature, and not due to a definite neuritis. Constipation, pelvic tumours, and the pressure of the foetal head are common causes of such sciatic pain. It is also probable that, just as a carious tooth may set up a trifacial neuralgia, so a pelvic tumour may give rise to a reflex neuralgia of the sciatic nerve. It is doubtful whether syphilis is often an important factor.

Pathological Anatomy.—If there be neuritis, the nerve trunk is generally swollen, and the exudations already mentioned may be found interstitially either inside or outside the funiculi, while haemorrhages are not infrequent. These changes are more commonly met with at the sciatic notch, or in the middle of the thigh. Where the pain is the result of pressure, or is a true neuralgia, there may be no recognisable anatomical change.

Clinical Features.—The chief clinical feature is pain. It comes on gradually, increasing in violence towards nightfall. It is greatly increased by muscular effort, or by pressure over

the nerve. The patient cannot stoop, nor is he able to sit on a hard seat without suffering torture, and he walks with the affected knee bent. The pain may be burning, boring, or shooting, and may shoot down to the knee, possibly to the heel. There are certain specially painful points: (1) over the sacro-sciatic notch, (2) just behind the great trochanter, (3) posteriorly in the middle of the thigh and along the line of the nerve, (4) just behind the head of the fibula, (5) in the middle of the calf, (6) behind the external malleolus, and (7) over the dorsum of the foot. Cramps not infrequently occur in the affected muscles, and the muscles may waste. There may be a certain amount of cutaneous anaesthesia, which is a definite indication of an organic change in the nerve. Sciatica is an obstinate and long-standing affection, very refractory to treatment, and relapses occur notwithstanding every possible care. In severe cases the patient is confined to bed altogether.

The **Diagnosis** is aided by the digital investigation of the nerve, and the chief point of importance is to decide what is the cause of the condition. The utmost care should be taken to investigate the pelvis and also the hip-joint for tubercular disease or rheumatoid arthritis. Where there is a lesion of the cauda equina, the pain is generally bilateral, and there is involvement of the sphincters.

The **Prognosis** as a rule is favourable: but protracted cases generally cause muscular atrophy, and imply a damage to the nerve from which it may take many months to recover.

Treatment. - In an acute case the limb should be kept absolutely at rest, and, if possible, a splint applied. Locally much may be done by poultices, or sometimes by blisters, to relieve the aching pain. The button cautery is often of service; but in an obstinate case, where it is not simply a neuralgia, but probably a true interstitial neuritis, acupuncture yields by far the most satisfactory results. The line of the nerve should be marked out accurately, and the needles, half-a-dozen in number, should be rendered aseptic, and then inserted at intervals of about an inch into the nerve: the highest needle should not be above the fold of the buttock, and the lowest must not reach as far as the popliteal space. The needles may be left in position for half an hour or thereby, the limb being covered by a cage so as to remove

the pressure of the bed-clothes. The object of the treatment is undoubtedly to permit escape of some of the exudate from the nerve sheath; and when we remember that the lymphatics inside the nerve run for considerable distances without communicating externally, the value of this treatment, where an exudation is present, is obvious. Sometimes a 1 per cent solution of osmic acid (m 2-10) is injected into the nerve with good results as regards pain, although the conductivity of the nerve is thereby greatly affected for some time. Internally rheumatism should be treated by means of salicylate of soda, and gout or syphilis, if present, should be treated on the usual lines.

Baths are often of great value, peat-baths or warm mud-baths affording much comfort to the sufferer. In certain cases high frequency has proved efficacious, but galvanism is disappointing. Massage should be used for wasted muscles, but only after the inflammatory stage has subsided.

Lastly, reference must be made to the surgical procedures of removing a portion of the sciatic, dividing adhesions or nerve-stretching. It is probable that the removal of a part of the nerve permits of the escape of the exudate, and therefore in some cases is advantageous. Dr. Crawford Renton has repeatedly found benefit from dividing adhesions, and it is probable that the older operation of nerve-stretching was only efficacious in so far as by its means painful adhesions were broken down.

(7) Plantar Neuralgia.—This is not uncommon in flat-foot, the pain being largely due to pressure on one or other of the branches of the nerve in the sole of the foot.

(8) Metatarsal Neuralgia, or Morton's Affection of the Foot.—This consists in spasmodic attacks of pain starting at the base of the fourth, sometimes of the second toe, and shooting up the leg. It is increased by pressure over the head of the metatarsal bone.

The affection is due unquestionably to tight shoes, which exert pressure on the heads of the metatarsal bones, especially the fifth, and persons with a tendency to flat-foot often suffer. The treatment is very simple—broad shoes, the use of a special spring in the boot so as to raise the instep, and, where these measures fail, excision of the head of the offending metatarsal bone.

(9) Testicular Neuralgia, Coccygeal Neuralgia, etc.

—Neuralgic pain may also be felt in the region of the testicle or spermatic cord, in the rectum, and in the urethra, while a coccygeal neuralgia, which is often of great severity, is due to pressure on the coccyx. This coccygeal pain is apt to follow labour, or a severe injury to the coccyx. Many hysterical patients suffer from pains of this kind, and the treatment consists largely in an attempt to combat the hysteria and to apply sedatives locally. In several cases benefit has resulted from applications of the high-frequency currents to the region of the rectum.

(10) Visceral Neuralgias.—The crises in locomotor ataxia, gastric, œsophageal, etc., are neuralgias, and angina pectoris may also be neuralgic in nature. Pelvic neuralgia is not uncommon in women, especially in hysteria.

Erythromelalgia, or red neuralgia, implies pain, redness, and swelling, and affects either the hands or feet. It resembles in certain respects Raynaud's disease, and is described later.

7. Diseases of the Spinal Nerves

(1) *Paralysis of the Phrenic Nerve*

This may be the result of peripheral neuritis (diphtheritic or alcoholic), or it may be due to pressure on the nerve roots (3rd and 4th cervical) from injury, haemorrhage, meningitis, tumour or disease of the vertebrae, malignant or tubercular. The phrenic nerve arises from the 3rd and 4th cervical segments, and these may be compressed or injured.

Clinical Features.—Paralysis of the diaphragm in most cases is bilateral, and breathing is entirely confined to the thoracic muscles. In place of distension of the abdomen on inspiration there is retraction, and, on the slightest exertion, extreme dyspnœa is experienced. Coughing is difficult, and bronchitis is a common sequel. Defæcation is much interfered with. If the paralysis is unilateral, the absence of movement on the affected side of the epigastrium is very suggestive.

The **Diagnosis** is generally fairly easy, although a mild degree of paralysis may present considerable difficulty.

The **Prognosis** depends on the cause, but certainly in

cases of peripheral neuritis, whether of alcoholic or diphtheritic origin, a fatal result is extremely probable.

The **Treatment** may necessitate an attempt at artificial respiration, especially where, as in peripheral neuritis, the intercostal muscles also become involved. Oxygen inhalations are to be commended.

Hiccough.—Hiccough is the result of spasmodic contraction of the diaphragm. Peripheral stimuli, frequently from the stomach or from the region of the abdomen, act reflexly on the respiratory centre, while in other cases the stimulation may be described as central in origin, as is Bright's disease or diabetes. There are also cases of hiccough which are associated with hysteria and epilepsy, and cases in which there is local cerebral irritation. Whether the hiccough is due to peripheral or central stimuli, it is by the phrenic nerves that the motor impulses are conveyed. Associated with the diaphragmatic spasm is a sudden closure of the glottis induced by stimuli conveyed through the medium of the vagus.

Transient hiccough is readily cured by sipping cold water, a number of sips being taken as rapidly as possible without drawing breath. In serious cases, in which hiccough goes on for days or weeks until the patient is exhausted, the skill of the physician may be taxed. Sometimes the application of a mustard leaf or of an ice-bag over the stomach affords relief; in other cases it is requisite to administer powerful sedatives, such as morphia or the bromides. Washing out the stomach may be found to be successful in certain cases.

(2) *The Brachial Plexus*

There are many possible forms of injury which may damage one or more cords, or the whole of the brachial plexus, such as a severe blow or fall, or the fracture of ribs or clavicle, and, in a considerable number of cases, traction on the arm has resulted in the tearing through of certain of the spinal nerve roots. Such cases have been met with in factories, where an arm has been drawn into the machinery; and similar lesions have ensued from traction on the arm during child-birth. It is obviously the lower nerve roots belonging to the plexus which are most likely to suffer, the roots being tightly stretched over the lower edges of the ribs close to the spinal column. When the brachial

plexus is damaged as a whole there is paralysis of all the arm muscles and generally also the pectoral muscles. The serratus magnus, the levator anguli scapulæ and rhomboids escape. The area of anæsthesia includes the whole arm excepting the upper part of the inner surface of the upper arm, which derives its nerve supply from the cervical plexus.

a. ERB'S PARALYSIS, or the Upper Arm Type of Paralysis is a lesion of the 5th and 6th cervical roots.

Etiology.—It may be produced by injuries such as those just referred to by pressure above the clavicle as from carrying a heavy load on the shoulder, and by traction on the neck of the infant at birth.

The **Clinical Features** include paralysis of the following muscles:—The deltoid, biceps, brachialis anticus supinator longus, and sometimes the supinator brevis and the supra- and infra-spinatus muscles. Anæsthesia is limited to the outer side of the upper and lower arm, and depends on the sensory distribution of the circumflex and musculo-cutaneous nerves. The result of the lesion is that the arm cannot be raised or abducted at the shoulder joint, cannot be flexed at the elbow joint, and supination is impossible. Pain may also be present.

b. KLUMPKE'S PARALYSIS, or a Form of Lower Arm Type of Paralysis, is a lesion of the 8th cervical and 1st dorsal roots.

Etiology.—It is produced by tumours, syphilitic meningitis, sometimes by primary neuritis, and also by traction on the arm in accident cases.

The **Clinical Features** include paralysis of the small muscles of the hand and the flexors of the fingers in the fore-arm, and should the 6th and 7th cervical roots be also involved the extensors, triceps, and the pronators and flexors of the wrist will be affected. The results of the lesion are wasting of the muscles above mentioned, pain, hyperæsthesia or anæsthesia in the realm of the ulnar nerve, and from the affection of communicating root branches of the 1st dorsal root there is myosis sluggish pupil reaction, and diminution in size of the palpebral tissure, the eye appearing to be somewhat sunken.[1]

c. PARALYSIS OF THE LONG THORACIC NERVE, OR SERRATUS PARALYSIS.

[1] A *cervical rib* causes paralysis and atrophy of the small muscles of the hand, an area of anæsthesia limited to the ulnar side of fore-arm and hand, and often a good deal of pain and sometimes tingling. The use of the X rays clears up the diagnosis.

The nerve arises from the 4th, 5th, and 6th cervical nerves and innervates the serratus magnus muscle.

Etiology.—The nerve may be injured by pressure or by a punctured wound, by traction on the elevated arm, injuries to the shoulder, carrying heavy weights on the shoulder, and sometimes it is affected in neuritis.

The serratus magnus muscle fixes the scapula when the arm is raised above the horizontal, and carries the scapula outwards, forwards, and slightly upwards. When the arm is raised vertically, the scapula being fixed by the rhomboids and other muscles, the serratus can raise the ribs and help in expanding the chest. There is often paralysis of the trapezius as well. In paralysis with the arm at rest the scapula is higher than on the normal side, and its inner border is nearer the middle line posteriorly. The arm cannot be raised above the horizontal, and the scapula is not kept in its usual close apposition to the side. This fact makes it desirable for the patient to wear a sling for a considerable time, so as to avoid keeping the paralysed muscle on the stretch. There may be pain in the region of the neck and shoulder but no area of altered sensation. Complete recovery is likely to be long delayed.

(*d*) Paralysis of the Circumflex Nerve.—The nerve arises from the posterior cord of the brachial plexus and supplies the deltoid and the teres minor muscles. It may be paralysed as the result of a fall or blow on the shoulder, dislocation of the shoulder joint, compression of the nerve as in crutch palsy, and less frequently from lead poisoning, diabetes, and other toxic conditions. As a rule the deltoid muscle is chiefly affected. The patient cannot move the arm outwards, forwards, or backwards, and the arm cannot be abducted. Generally the shoulder hangs loosely, and the head of the humerus is subluxated. There may be an area of anaesthesia over the region of the deltoid and the upper part of the outer surface of the arm.

If the suprascapular nerve is also affected, the supra- and infra-spinatus muscles are paralysed, and the anaesthetic area extends to the outer border of the scapula.

(*e*) Paralysis of the Musculo-Spiral Nerve.—The nerve, derived from the 6th, 7th, and 8th cervical roots, arises from the posterior cord of the brachial plexus and supplies the triceps, brachialis anticus (in part), supinator longus, extensor

carpi radialis and ulnaris, the extensor communis digitorum, and the extensors of the thumb and the little finger. The skin area supplied by the nerve includes the outer side of the wrist, hand, and the thumb (see Fig. 32).

Etiology.—There is no nerve so liable to injury from its position, and from its relationship to the humerus and the muscles in the upper arm. Pressure exerted by the use of a crutch, or due to the patient hanging the arm over the back of a chair, frequently causes paralysis of this nerve, and particularly in alcoholic and other patients who have an inherent predisposition to neuritis. Heavy weights, violent action of muscles, especially suddenly extension, and sometimes

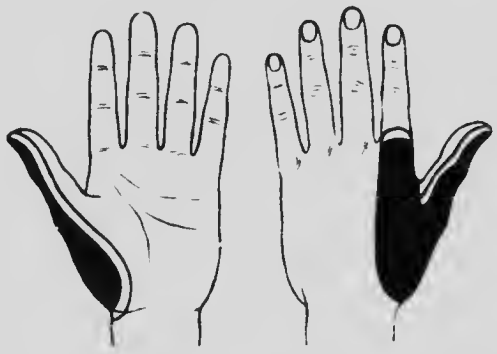


FIG. 32.—Area of anæsthesia after section of musculo-spiral nerve (in black). Epicritic overlap is indicated by the area between the black part and the outer line. (After Head.)

gunshot or other wounds and injuries to the arm, are also responsible for the paralysis. There are also toxic cases in which arsenic, lead, and alcohol are etiological factors, and more rarely the toxin of one of the infective fevers.

Clinical Features.—In the usual pressure paralysis the branch to the triceps escapes because the pressure is exerted below the level of its origin from the nerve. There is paralysis of the supinator longus, the extensors of the hand and fingers, including the extensor of the index finger, the little finger, and the thumb. The hand is flexed at the wrist-joint, the fingers and thumb flexed at the metacarpo-phalangeal joints. The hand cannot be extended, nor can the proximal phalanges, although the other two phalanges are not affected, as the interossei muscles are not supplied by this nerve. The grasp of the hand is weakened, although this is largely due

to posture. If the lesion is in the fore-arm the supinator and the extensors of the carpus are not affected.

The area of anaesthesia depends upon the position of the lesion. Should it include all the branches, the area of anaesthesia extends over the back of the hand corresponding to the two radial fingers and the back and outer side of the thumb, but is often limited as in Fig. 32. As usual the epicritic overlap is more extensive than the protopathic.

Should the radial nerve escape, there may be no sensory change at all, or only a limited area of anaesthesia may be present, confined to the outer side of the thumb.

The **Prognosis** in most cases is favourable, and especially where the cause has been pressure, but the electrical reactions, and in particular the retention of faradic excitability, constitute a most important prognostic point.

The **Treatment** consists in the prompt removal of the cause of pressure and in the application of electricity—galvanism, faradism, or both currents combined. In some cases nerve suture or muscle anastomosis has proved beneficial. For the weakened flexors of hand and fingers, Heusner has devised a form of glove, or rather an arrangement of straps which keep the wrist-joint slightly extended and the four fingers in a position of slight extension.

f) Paralysis of the Ulnar Nerve.—The nerve receives its fibres from the last cervical and 1st dorsal roots, and arises from the inner cord of the brachial plexus. It supplies the following muscles:—the ulnar half of the deep flexor of the fingers, the flexor carpi ulnaris, the interossei, the two ulnar lumbricales, the adductors and half the flexor brevis pollicis and the hypothenar muscles. The sensory supply corresponds to the palmar and dorsal surfaces of the ulnar half of the ring and the little fingers, including the front and back of the corresponding metacarpals.

Etiology. Injuries in the region of the elbow constitute a common cause of ulnar palsy, and in particular, fracture of the internal condyle of the humerus. After a fracture, the nerve may be involved by the callus which develops in the process of reunion. Toxic causes of paralysis are not uncommon.

Clinical Features.—Where the lesion occurs at the elbow-joint, paralysis of the muscles mentioned produces the most

characteristic of palsies. All the fingers, and specially the little and ring fingers, are flexed at the interphalangeal joints, while there is hyperextension of the little and ring fingers at the metacarpo-phalangeal joints. The fingers cannot be separated, and the interosseous spaces are deep owing to the wasting of the interossei muscles. Adduction of the thumb is impossible, and the hand soon assumes a typical clawlike appearance, should the paralysis be a serious one. There is anaesthesia over the sensory area of the nerve, with an epicritic overlap as shown in Fig. 33.

Where the lesion is at the wrist-joint and the muscles

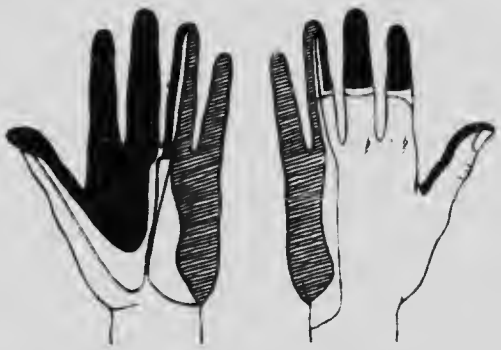


Fig. 33.—Areas of anaesthesia after section of ulnar nerve (transverse shading) and median nerve (in black). Epicritic overlap indicated as in Fig. 32. (After Head.)

supplied at a higher level escape, paralysis is limited to the small muscles of the hand.

(*g*) Paralysis of the Median Nerve.—The nerve composed of fibres proceeding from the last 3 cervical and 1st dorsal roots arises from the outer and inner cords of the brachial plexus. It supplies the superficial and deep (in part) flexor of the fingers, the long flexor of the thumb, the flexor carpi radialis, the two pronators, the abductor and opponens pollicis, and the short head of the flexor brevis pollicis. It supplies also the two radial lumbricales. The sensory area corresponds to the palmar aspect of the first two and a half fingers and thumb, and on the dorsal aspect the back of the first two and a half fingers from the base of the second phalanges to the finger-tips, thus sharing with the ulnar nerve in supplying the greater part of the skin area of the front and back of the hand (see Fig. 33).

Etiology.—The nerve may be injured by a lesion in the fore-arm, either an incised wound or fracture, and particularly in the neighbourhood of the wrist-joint. Should the injury be above the elbow-joint, other nerves frequently suffer.

Clinical Features.—If the lesion is above the elbow-joint, all the muscles referred to are paralysed and the patient cannot flex the fingers, and, in particular, the index finger and the terminal phalanx of the thumb. Pronation is feeble, and the wrist cannot be flexed except towards the ulnar side. The sensory loss corresponds to the area described. There is a considerable epicritic overlap.

Where the lesion occurs at the wrist-joint, the motor paralysis is limited to the thumb muscles and the two lumbricales, while the sensory loss remains as already described.

Pain and hyperaesthesia are not infrequently complained of by the patient.

(3) *The Lumbar Plexus*

The lumbar plexus arises from the ventral primary divisions of the first four lumbar roots, with a connecting branch from the 1st dorsal root. It is formed in the substance of the psoas muscle, and supplies the flexors and abductors of the thigh, the extensors of the knee, and the cremaster muscle. It supplies also the skin over the lower part of the abdomen, the region of the genital organs, the greater part of the internal and external surfaces of the thigh, and the internal surfaces of the leg and foot.

The lumbar plexus may be injured in whole or in part by disease of the spinal column, such as caries or tumour, by meningeal inflammations, by affections of the cord, and by lesions involving the roots or branches of the plexus. Neuritis is not uncommon, especially a neuritis secondary to tumours or other conditions.

(*a*) Paralysis of the Obturator Nerve sometimes occurs from pressure during labour, occasionally from tumours or obturator hernia. It causes loss of power in the adductors of the thigh with inability to cross the legs, while outward rotation is impossible.

(*b*) Paralysis of the Anterior Crural Nerve may occur as the result of tumour or abscess, dislocation of the hip joint and other conditions. The extensors of the knee are

paralysed, the knee-jerk lost, and there is anaesthesia of the anterior and inner part of the thigh [below the area supplied by the ileo-inguinal and genito-femoral nerves] and the inner side of the leg as far as the great toe. There is a considerable epicritic overlap.

(*c*) PARALYSIS OF THE EXTERNAL CUTANEOUS NERVE. - This nerve is mainly sensory, and is affected by neuritis, sometimes of alcoholic origin. Pains are felt along the lines of distribution of the nerve, and there is tenderness on pressure where it passes under Poupart's ligament just internal to the anterior superior iliac spine, and there may be altered sensation over the outer side of the thigh. So severe may be the pain associated with this neuralgia that division of the nerve has been practised for its relief.

4 *The Sacral Plexus*

The sacral plexus is formed by the 5th lumbar and the first 3 sacral nerves and part of the 4th. It supplies the extensors and rotators of the thigh, the flexors of the knee, and all the foot muscles, and the cutaneous area governed by these nerves includes the skin over the buttock, the back of the thigh, the outer and posterior aspects of the leg below the knee, and the greater part of the foot.

Etiology.—The sacral plexus is liable to be injured by tumours, pelvic inflammations, and injury during parturition.

(*a*) PARALYSIS OF THE SCIATIC NERVE.—The sciatic nerve is the chief branch of the sacral plexus, and it may be injured by conditions similar to those above mentioned, but it must be remembered that neuritis is a not infrequent cause of paralysis, and that injuries to the nerve from fracture of the thigh, tumours, and so forth, may also cause sciatic paralysis. The nerve supplies the extensors of the hip, the flexors of the knee, and all the muscles below the knee, but it depends on where the lesion is situated whether the branches supplying the thigh muscles may not escape. The branch for the hamstrings comes off some distance above the popliteal space, and so may readily escape in a lesion at a lower level. The sensory supply of the nerve includes all the skin below the knee excepting a strip on the inside of the leg (internal saphenous nerve).

Wasting of the affected muscles results, and there may not infrequently be trophic sores produced on the foot, and sometimes herpetic eruptions along the line of the nerve.

(*b*) Paralysis of the External Popliteal Nerve is generally the result of injuries or neuritis. The nerve supplies the long and short extensors of the toes, the peroneal muscles, and the tibialis anticus, while the sensory distribution includes the outer half of the front of the leg and the dorsum of the foot.

The **Clinical Features** include foot-drop with the characteristic high-steppage gait. In old-standing cases talipes equinus may develop.

(*c*) Paralysis of the Internal Popliteal Nerve.—The nerve supplies the posterior tibial muscle, the muscles of the calf, the sole of the foot, the poplitens, and the long flexors of the toes, while the sensory distribution includes the lower half of the back of the leg, and the sole of the foot and plantar aspect of the toes.

Etiology.—The nerve may be injured by a blow or wound, and neuritis is a not infrequent cause.

The **Clinical Features** include loss of extension at the ankle-joint: adduction of the foot becomes impossible, and the patient cannot rise on tiptoe, while talipes calcaneus may develop as the result of a long-standing paralysis.

Many of the lesions of the plexuses and nerves above mentioned are associated with more or less severe neuralgia, and these neuralgic pains may be the result of irritation by pressure or of the action of toxins, while more trivial cases may depend on deficient amount, or poor quality, of the blood-supply.

8. Diseases of the Cranial Nerves

(1) *1st Cranial Nerve—the Olfactory Nerve*

Anosmia, or loss of smell, may result from lesions of the nasal mucosa, the olfactory bulbs and the fine terminal nerve fibres which pierce the cribriform plate of the ethmoid bone, or of the accredited olfactory centres situated, according to Ferrier, in the uncinate convolutions.

Catarrhal affections of the mucosa of the nose and polypi are common causes of anosmia, while the olfactory bulbs and

their filaments may be damaged by fractures in the anterior fossa of the skull by blows on the head, falls on the occiput (by contrecoup), caries of neighbouring bones, meningitis, tumours, and rarely by neuritis or by primary degeneration such as occurs

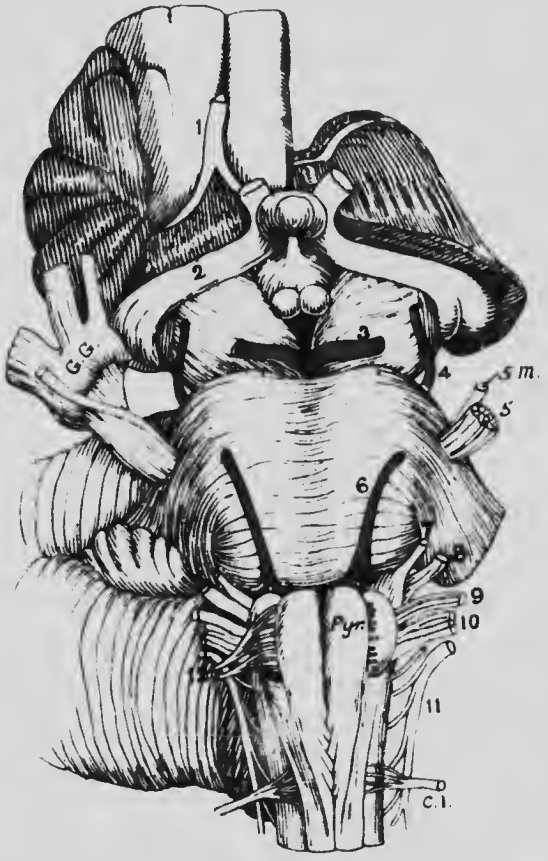


Fig. 34. Figure showing Base of Brain with emergence of Cranial Nerves. The 1st or olfactory, 2nd or optic, and 8th or auditory nerves are coloured yellow. The 3rd, 4th, and 6th nerves which supply the ocular muscles are coloured red. (After Quain.)

in locomotor ataxia. Lesions of the uncinate convolution cannot be considered as a common cause of anosmia.

Smell is responsible for much of the pleasure usually referred to the sense of taste. It may be tested by sniffing oil of peppermint or other essential oil, but care must be taken not to utilise agents which will act mainly on the 5th, the nerve of common sensation to the nasal mucosa.

The **Prognosis** is unfavourable in most cases, and little can be suggested by way of satisfactory treatment.

Hyperosmia, or excessive appreciation of smell, is peculiar to hysterical and insane persons, and *Parosmia*, or perverted sense of smell, is sometimes found in similar cases. In

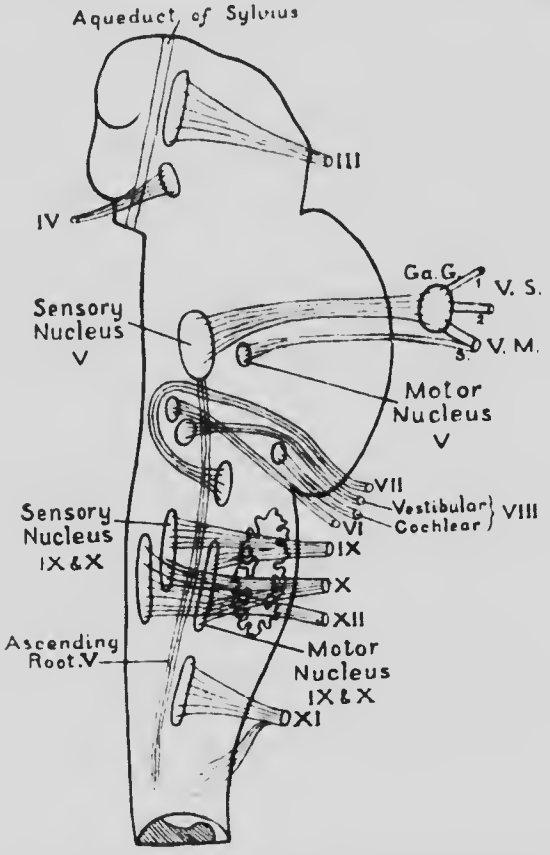


FIG. 35.—Diagrammatic Representation of the Nuclei of the Cranial Nerves. The descending root of the 5th Nerve has been purposely omitted.

epilepsy the aura may be olfactory, and may be an instance of parosmia.

2. *2nd Cranial Nerve, or Optic Nerve*

The course of the optic nerve fibres is best appreciated by the figure. They pass from the occipital lobe by the optic radiation of Gratiolet to three ganglia, the anterior corpus

quadrigeminum, the external geniculate body, and the pulvinar of the optic thalamus. From these ganglia the three bundles pass into the corresponding optic tract, and reach the optic commissure or chiasma. The outer half of the fibres passes to the outer half of the retina in the optic nerve of the same side, while the inner half of the fibres of each tract decussates in the commissure, and passes to the optic nerve and the inner

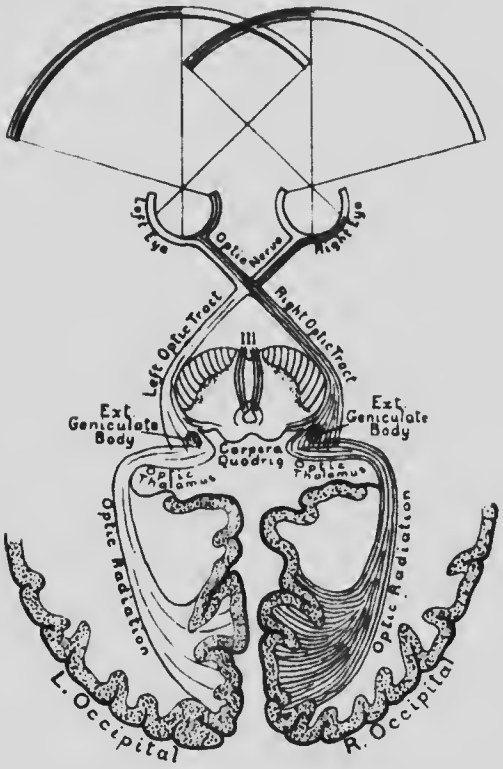


FIG. 36.—Figure showing Course of Visual Fibres.

half of the retina of the opposite side. Thus the *right* optic tract supplies the *right* halves of both retinae. The fibres supplying the macular portion of the retina, which originate in the neighbourhood of the calcarine fissure, also participate in this division at the chiasma, and pass in each optic nerve in a definite bundle—the papillo-macular bundle. The nerve fibres which pass through the external geniculate body are the most important sight fibres, while the bundle passing to the

anterior corpus quadrigeminum carries upward from the eye the impulses to form the sensory limb of the light-reflex arc, and from the anterior corpus quadrigeminum these impulses pass by Meynert's fibres to the nucleus of the 3rd nerve, to reach the part of the nucleus wherein lies the controlling centre for the sphincter pupillae muscle. The motor limb of the arc is the 3rd nerve. It is, however, absolutely certain that the ciliary ganglion contains the cells which functionate the sphincter pupillae muscle, and that in locomotor ataxia and general paralysis in which the Argyll-Robertson phenomenon is present, the lesion is found in the presiding cells in the ciliary ganglion and not, as was formerly supposed, in a primary degeneration of Meynert's fibres.

Wernicke's Pupil Reaction is the test used to determine whether this light-reflex arc is intact. In other words, it determines whether the lesion involves the optic tract, or is situated behind the anterior corpus quadrigeminum. The patient is seated in a dark room, and the ray of light must be thrown, by the ophthalmoscopic mirror on to the blind half of the retina. If the pupil contracts, the light-reflex arc is intact; if it does not, the lesion must be situated in the optic tract.

A lesion of one occipital lobe, of one optic radiation of Gratiolet, or of one optic tract, causes *homonymous hemianopsia*, the two corresponding halves of the retinae being involved and producing blindness of the opposite halves of the fields of vision.

A lesion through the middle of the chiasma, such as might result from the pressure of an aneurismal dilatation of one of the anterior cerebral arteries, may cause the involvement of the two nasal halves of the retinae which correspond to the two temporal halves of the fields of vision. A similar lesion is met with in cases of acromegaly (see p. 356). Where the two temporal halves of the retinae are affected, a double lesion must be present, one on either side of the chiasma, and involving the two halves of the optic tracts which do not decussate. Such a double lesion causes blindness in the two nasal halves of the field of vision. Where the two nasal or the two temporal halves of the retinae are involved, and corresponding blindness results, the lesions are termed *heteronymous hemianopsia*.

If the lesion involves the angular gyrus, the resulting

changes vary somewhat. There may be hemianopsia, sometimes crossed amblyopia, and, if the lesion be on the left side of the brain in a right-handed individual, there is visual aphasia or word-blindness.

It is impossible to refer here to all the forms of blindness in the visual field, but it may be desirable to remind the reader of the significance of the following terms:—

Scotoma is limitation of the field of vision. It may be peripheral or central. Peripheral scotoma is a common result of optic neuritis, whereas chronic retrobulbar neuritis often causes central scotoma. Generally the visual field for colour is seriously affected in cases where the field for white is limited. It is interesting to know that a great deal depends on the size of the test object used, and Bjerrum has, by his delicate methods of testing the vision field, greatly added to our accuracy of observation, the size of the test object used being an important feature in this method.

Amaurosis is complete blindness, temporary or permanent, without perceptible ocular lesion.

Amblyopia is loss of acuteness of vision, and is very frequently associated with toxic conditions such as tobacco poisoning.

Night-blindness is a peculiar functional condition, in which the patient fails to be able to see clearly at night.

Day-blindness is the opposite of the preceding, and is a condition in which objects cannot be seen comfortably in bright light.

(*a*) Optic Neuritis.—Inflammation of the optic nerve, and specially the papilla, often associated with a greater or less degree of retinitis.

Etiology.—It is most frequently the result of—

(1) Cerebral tumours, and occurs in 80 per cent of all such cases. Certain kinds of tumours are more apt to produce it than others, glioma taking the leading place, and the site of the tumour has a great influence. It is most common in cerebellar tumours and in neoplasms affecting the corpora quadrigemina, while it is rare in tumours of the medulla. The size of the tumour has little to do with its development. In a big tumour optic neuritis may be absent, and in a small tumour situated in the cerebellum, and no larger than a hazel-nut it may be very intense and of rapid development.

2 Meningitis is reputed to be the second most frequent cause, but this is doubtful. It is very rare in simple meningitis, more common in basal meningitis of tubercular origin, especially if there are definite tubercular tumours in the brain, and it is most common in syphilitic meningitis with gummata involving brain tissue.

3 In aneurisms of the internal carotid, in abscesses and cysts, optic neuritis may occur, and it is sometimes associated with thrombosis of the cavernous sinus.

4) It results from a group of cases to which the term toxic is applicable, and of which Bright's disease, generally the chronic form, is the best example; while in the same group we might include its rare occurrence in cases of continued fever, in lead poisoning, etc.

5) It results sometimes from local eye disease, to which the optic neuritis is secondary, and we should especially note here hypermetropia with astigmatism.

(6) In cord and brain disease, such as myelitis, disseminated sclerosis, cerebritis, and general paralysis, it very occasionally occurs.

7) In meningeal haemorrhage, where the intersheath space is involved, optic neuritis may develop secondarily.

(8 It occurs sometimes as the result of anaemia, chlorosis, and other blood diseases, and it may constitute a grave danger to the patient's sight in cases of menstrual irregularity.

Pathological Anatomy and Pathology.—There is sometimes definite "choked disc," but very often the papillitis or swelling of the papilla is moderate in degree. Exudation of inflammatory lymph is always present, and there is a certain, but as a rule a limited, number of inflammatory cells. In the optic nerve the increase of nuclei is not generally a leucocytic one, but is due to a proliferation of neuroglial cells. There is sometimes a great cellular proliferation in the intersheath space. There may be a great increase in the fluid contents of the intersheath space, but this is by no means always the rule: when it is present, it certainly tends to produce a very marked choking of the disc, from interference with the outflow of blood and lymph from the papilla. The retina always suffers, but the retinitis varies much in extent and degree. Where recovery does not occur, optic atrophy of the consecutive type supervenes.

The backward pressure theories of optic neuritis include venous pressure in the cavernous sinus and lymph pressure in the intersheath space and the lymph spaces which communicate with it. The free communication of the cavernous sinus with orbital and facial veins, and the fact that often there is no retrobulbar distension of the intersheath space put these theories out of court in most cases. Leber has suggested a toxic agent in the cerebro-spinal fluid, and Galezowski a toxic agent acting by continuity of tissue from the site of the tumour, abscess, etc., others have suggested a descending neuritis, or a meningitis, commencing near the tumour and extending by the meninges to the optic nerve. We favour the toxic theory owing to various facts concerning the neuroglial hypertrophy and proliferation which cannot be discussed here.

Clinical Features.—The opthalmoscopic appearances are very marked.

(1) At a very early stage there is simply congestion of the disc, which later forms a definite swelling, great or small in amount. The edge of the disc becomes blurred, and the sharp distinction between retina and papilla is lost, while the physiological cupping disappears, being filled up with inflammatory exudation.

2) The vessels are more or less covered with exudation, and, as the result of pressure, the veins become enlarged and tortuous, and the arteries narrower than normal. There may be a certain amount of exudation extending along the vessels into the retina for some distance.

3) Haemorrhages, either in the swollen disc or the retina near the disc, are not infrequent. They are generally flame-shaped, but, if in the nerve-cell layers of the retina, they may be round or oval.

The visual changes vary. Sight may be preserved for a marvellously long time after optic neuritis has commenced. It may fail gradually or suddenly. There is generally peripheral contraction of the field, and more rarely central scotoma. The colour vision is specially affected, green and red suffering most markedly.

These changes, ophthalmoscopic and visual, are generally bilateral, unless they are due to an ocular cause, when they may be limited to the affected eye. When bilateral, they are often more advanced in one eye, and in tumour cases are

sometimes worse on the side of the lesion. Recovery may take place even in severe cases if the cause of the condition is removed, but it is generally found that severe optic neuritis leads to consecutive atrophy and complete loss of sight, the disc becoming white and filled in, and the vessels diminishing in size, although the veins remain proportionately larger than the arteries.

The other clinical features depend on the cause of the condition, but headache is commonly present.

Diagnosis.—It is the cause of the optic neuritis which demands investigation. There is usually headache, vertigo and cerebral vomiting in cases of intracranial tumour, besides any localising phenomena which may be present. In Bright's disease the urine should afford a clue to the diagnosis, and in anaemia and other blood affections the examination of the blood ought to be sufficient. Hypermetropia with astigmatism is easily recognised, and menstrual irregularities afford a suggestive history.

The **Prognosis** is favourable where an intracranial tumour is operable and the optic neuritis is not too far advanced, and in syphilitic cases there is always a better prognosis if suitable medicinal treatment is commenced at a sufficiently early date.

Treatment.—In syphilitic cases give potassium iodide in 10 to 30 grain doses: mercury should also be tried. Operation must be attempted, where possible, for the relief of pressure although it is definitely known that in not a few cases pressure is not the chief or only cause of the optic neuritis. In inoperable cases it is therefore considered desirable to trephine, simply because, in a proportion of patients, benefit results as regards sight. Puncture of the intersheath space has been attempted but without much success, and lumbar puncture, where it is safe to attempt relieving pressure in this way, has yielded disappointing results. If it can be definitely proved that the condition is toxic and the toxin is recognised, something may be possible when our knowledge is more precise, but obviously many inoperable intracranial tumours must prove fatal at an early period, and the loss of sight often only precedes death by a short period of time.

b. Chronic Retrobulbar Neuritis.—This is the involvement by toxins of the special bundle of optic nerve fibres which supply the region of the yellow spot, and which are called the

papillo-macular bundle. It is due to tobacco and alcohol, but apparently some other factor is also necessary. It is found (1) in certain cases of cord disease (such as locomotor ataxia and disseminated sclerosis); (2) where tobacco and alcohol have been used in great excess: (3) it is present as the result of other toxic agents such as lead, diabetes, and the toxins present in syphilis: and (4) certain drugs, such as quinine. It is a parenchymatous degeneration of the nerve fibres, and it is bilateral. There is little change in the disc at first, but later the affected part (towards the temporal side) becomes swollen and blurred, and when atrophy has developed, it is extremely white.

There is marked central scotoma—the periphery of the fields being fairly normal. This form of optic neuritis has a favourable prognosis provided it is due to the use of alcohol and tobacco. Treat the condition by stopping the tobacco and alcohol absolutely, and give strychnine, iron and other tonics. Potassium iodide is also recommended.

(*c*) Optic Atrophy.—This may be (1) *consecutive*, (2) *primary*, or (3) *secondary*.

(1) *Consecutive* or *post-papillitic atrophy* is the result of optic neuritis where recovery does not occur.

The disc is filled in and white, but the edges are slightly blurred, and white lines along the vessels may indicate the site of old exudation. The veins are dilated and tortuous, although less so than in the pre-existing neuritis, while the arteries are even smaller in size than in that condition. There is complete blindness eventually, although there may be merely limited visual acuity for some time. The nasal half of the field of vision is usually first involved.

(2) *Primary atrophy* is often associated with certain cord diseases, and in particular locomotor ataxia and disseminated sclerosis, but it may develop in syphilis, diabetes, menstrual irregularities, and in cases where cold, tobacco, alcohol, and lead poisoning are the causal factors, while it may also be of congenital or hereditary origin. Retrobulbar neuritis, described above, might be often more correctly termed a primary atrophy limited to the papillo-macular bundle, while in some cases of primary atrophy this bundle escapes at least for a long time, and may enable the patient to retain his central vision wonderfully unimpaired. The ophthalmoscopic

picture is very different from consecutive atrophy. The disc is sharp to begin with, and of a peculiar greenish-grey colour; the lamina cribrosa is well seen in the floor of the physiological cup. The vessels, although eventually diminished in size, show no proportionate decrease in lumen. The field of vision often shows at first a central scotoma, and the colour vision is markedly interfered with even at an early stage.

(3) *Secondary atrophy* — a term which should be reserved for cases in which there is a true descending degeneration — is due to a lesion of optic nerve, tract, or commissure, with, as a secondary result, atrophy of the fibres cut off from their central trophic cells. It may be the result of a tumour, aneurism, suppuration, fracture, or, in fact, anything exerting pressure, one occasional cause being great distension of the 3rd ventricle, as in hydrocephalus, which may press seriously on the optic commissure. The ophthalmoscopic appearances are not very distinctive: the vessels are contracted, but the veins are proportionately larger than the arteries. The disc is sharp cut, and there are no white lines running along the vessels into the retina, but the physiological cup may be filled in. There is very rapid interference with vision, the degree and kind of scotoma being dependent on the fibres which are affected.

The **Diagnosis** of the different forms is usually not very difficult; the first and third forms cannot well be confused.

The **Prognosis** is unfavourable in most cases, but the duration of vision, however impaired, varies greatly: in primary atrophy it may be weeks, months, or even years.

The **Treatment** consists in trying to remove the cause of the condition where that is possible; in giving iron, strychnine, and other tonics; and certainly in trying the effect of anti-syphilitic remedies.

4. Retinitis.—Retinitis more properly belongs to the domain of the specialist, and only a very brief description of forms important to the physician will be mentioned here. The nerve fibres, when they leave the disc, pass into the retina, and, as stated under optic neuritis, inflammation of the optic disc must inevitably involve, to a certain extent, the neighbouring retina. In albuminuric retinitis, however, the retina appears to suffer primarily, and only in certain cases is there a definite papillitis.

Albuminuric Retinitis. This occurs in chronic Bright's disease, and sometimes in cirrhosis of the kidney, but is uncommon in the more acute kidney inflammations. It is believed to be due to vascular degeneration or arteriosclerosis, and as a result, fatty changes occur in the nerve fibres near the yellow spot owing to deprivation of blood. These fatty-looking fibres have a peculiar radial arrangement round about the yellow spot. Associated with this condition there is generally a degree of papillitis, often with many haemorrhages, both in the inflamed disc and in the retina.

Leucocythaemic Retinitis.—There are numerous retinal haemorrhages, and the remaining part of the retina is of a palish colour.

Syphilitic retinitis and *retinitis pigmentosa* cannot be described here, and in fact the latter condition is of too rare occurrence to demand reference.

3) *The Oculo-Motor Nerves, the 3rd, 4th, and 6th*

(*a*) PARALYSIS OF THE 3RD NERVE.—*Nucleus.* It contains five distinct cell groups, situated in the grey matter of the floor of the aqueduct of Sylvius, and opposite the anterior corpus quadrigeminum; its posterior extremity almost reaches the nucleus of the fourth.

Point of Exit from Brain. The two nerves come out together between the crura cerebri.

It supplies 1, Superior division—Superior rectus; levator palpebrae superioris. 2, Inferior division—Internal rectus; inferior rectus; inferior oblique.

The 3rd nerve also supplies, through the ciliary ganglion, the ciliary and the sphincter pupillae muscles.

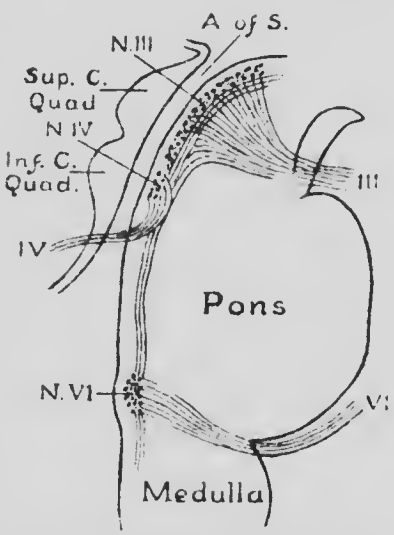


FIG. 37.—Shows position of nuclei of the 4th and 6th Cranial Nerves. A of S. = aqueduct of Sylvius.

The nucleus for convergence and certain of the other nuclei send fibres to both 3rd nerves.

Etiology.—The nucleus may be involved by tumour, haemorrhages, inflammations, such as acute polio-encephalitis, or degeneration, but is rarely affected without other nuclei suffering. The nerve may be involved in meningitis, gummata, and aneurism; in diphtheria and locomotor ataxia a neuritis of the nerve may be present.

The *Results of Paralysis* depend much on whether the whole nerve, one division, or merely certain muscles are affected. If complete, there is slight protrusion of the eyeball, diplopia, external strabismus, ptosis, dilatation of the pupil, and paralysis of accommodation, while the eyeball can only be moved by the superior oblique and external rectus downwards-outwards, and slightly rotated inwards.

For the effects of a lesion of the separate external ocular muscles, see table on p. 627.

Ptosis may be the result of: (1) a congenital paralysis of the levator palpebrae muscle, in which case the condition is permanent; (2) a more or less complete lesion of the 3rd nerve; (3) idiopathic muscular atrophy; (4) myasthenia gravis; (5) a lesion of the 1st dorsal root or the sympathetic fibres associated with it, which causes a pseudo-ptosis as the result of sinking in of the eyeball; (6) hysteria; (7) debility in weakly persons, especially females, in whom the condition is usually only marked on awakening from sleep.

Iridoplegia, or Paralysis of the Iris.—(A) The *Argyll Robertson Phenomenon*, or loss of the light-reflex, indicates a lesion of the reflex arc formed by fibres in the optic nerve, the optic tract passing to the anterior corpus quadrigeminum, and from there by fibres passing to the nucleus of the 3rd nerve; from the nucleus the motor impulses travel by the 3rd nerve through the ciliary ganglion, the cells of which include the trophic centre for the sphincter pupillae muscle, to the sphincter pupillae muscle. Light must be very carefully thrown on to the retina of each eye separately. The light-reflex is lost in locomotor ataxia and in general paralysis of the insane, and its absence may help also in localising a lesion of the optic nerve fibres *behind* the commissure. See Wernicke's pupil reaction, p. 616.

B *Accommodation Iridoplegia* is loss of contraction of the pupil on looking at a near object.

C) *Cutaneous Iridoplegia* is loss of the typical dilatation of the pupil on pinching the skin of the forehead.

Cycloplegia is loss of the power of accommodation, due to paralysis of the ciliary muscle; as a result the patient cannot read or see near objects clearly.

Paralysis of the 4th Nerve.—The nucleus lies in the floor of the aqueduct of Sylvius, and is just behind the 3rd nucleus (see Fig. 37). The nerve fibres, after leaving the nucleus, decussate in the valve of Vieussens, and winding round the outer surface of the crus, pass through the sphenoidal fissure to the superior oblique muscle.

Etiology.—Tumours, softening, etc., may involve the nucleus, while the nerve may be damaged by conditions similar to those from which the 3rd or 6th nerve may suffer.

Result of Paralysis. There is defective downward and inward movement and double vision when the patient looks downwards and outwards.

Paralysis of the 6th Nerve. *Nucleus.* Situated in the floor of the fourth ventricle above the striae acusticae.

Point of Emergence of Nerve. Between medulla and pons the fibres passing just external to, or may be through, the motor pyramidal fibres.

It supplies the external rectus muscle.

Etiology. The nerve, from its long course, is commonly affected by tumours, tubercular meningitis, gummata, and so forth.

The nuclear origin of the opposite *internal* rectus is connected with the 6th nucleus by a strand of fibres, and therefore in a nuclear lesion the opposite internal rectus is affected along with the external rectus of the side of the lesion. Further, because the fibres of the 7th nerve pass closely round the 6th nucleus there is often facial paralysis on the one side as the lesion.

If the nerve is paralysed, there is strabismus, diplopia and often considerable giddiness.

When examining paralysis of any of the muscles supplied by the 3rd, 4th, or 6th nerves, we should note the *primary deviation* of the affected eye and the *secondary deviation* of the

normal eye, the latter being due to the effort of the patient to fix the object with the affected eye. These deviations, when measured and compared, yield important information. The secondary deviation greatly exceeds the primary in cases of paralysis, while in strabismus resulting from an error of refraction or from spasm it is usually exactly the same as the primary.

Diplopia implies the existence of a false image. When the false image is on the same side as the eye with which it is seen, it is simple or homonymous. When on the opposite side, it is crossed. The false image is always projected in the direction in which the paralysed muscle ought normally to move the eye, and the false image is farthest from the true when the attempt is made to use the affected muscle. The patient, by turning the head, tries to avoid the diplopia and prevent the giddiness and sickness to which it often gives rise.

The following scheme explains itself: Remember that the obliques, acting round pulleys, pull the eyeball exactly in the opposite direction to what the terms superior and inferior would suggest. Both obliques move the eyeball *outwards*, and the superior and inferior recti, counteracting this, move them *inwards*; and while the two superior muscles (oblique and rectus) rotate the eyeball *inwards*, the two inferior muscles rotate the eyeball *outwards*. The right eye is taken as the affected one, and the arrow represents the false image.

Normal Movements

Muscles	Outwards. Inwards.	Upwards. Downwards.	Rotation, inwards or outwards.	Images. (Nasal, Temporal.)
Superior oblique	Outwards.	Downwards.	Inwards.	
Inferior oblique	Outwards.	Upwards.	Outwards.	
Superior rectus	Inwards.	Upwards	Inwards.	
Inferior rectus	Inwards.	Downwards.	Outwards	
External rectus	Outwards.			
Internal rectus	Inwards.			

To get the rotation, place a pencil first in the proper position as regards "outwards" or "inwards," and "upwards" or "downwards," and then if the rotation has to be inwards turn the point of the pencil towards the nose, if outwards, away from it.

To find the direction of the eyeball when any muscle or muscles are paralysed, delete the affected muscles; then, by working out the movements of the remaining muscles which counteract each other it is easy to obtain the final result.

External Ophthalmoplegia is the term applied to more or less complete paralysis of the external ocular muscles. It

may be due to a nuclear lesion or meningitis, but it is specially common in locomotor ataxia and general paralysis. It is often associated with *Internal Ophthalmoplegia*, which is paralysis of the iris and the ciliary muscle.

In conclusion, there may be a supranuclear involvement of the 3rd, 4th, or 6th nerves, which may, in rare cases, give rise to crossed paralysis.

Treatment of Ocular Paralysis.—Try to find out the cause, and if possible treat it. Antisyphilitic remedies should be administered, and an attempt made to arrest neuritis, if present, by counter-irritation behind the ear or over the temple. Electricity is of little use; strychnine may be ordered in certain cases, and by prisms, or by covering the affected eye, much may be done to counteract the diplopia.

(b) Spasms of Ocular Muscles.—These may be tonic or clonic.

Conjugate deviation is common in apoplexy, the patient looking upward towards his lesion in "destructive" cases, while in "irritative" cases he looks away from it. Spasms of ocular muscles may also occur in meningitis, or where there is basal irritation. In chorea there may be diplopia, and in hysteria and epilepsy there are often spasms, tonic and clonic, of ocular muscles. Serious errors of refraction may also cause spasm.

Nystagmus is clonic spasm, usually lateral, rarely vertical or rotatory. It may be intermittent and only appear when a weak muscle is put on the stretch. It varies from about 60 per minute to an uncountable rate, and the magnitude of the movements also varies between coarse and excessively fine oscillations. Its causes are: (1) purely ocular, as in high grades of refractive error, choroiditis, opacities in the lens, in albinism, and also in other congenital defects in the retina and choroid; (2) in certain lesions of the cerebellum, optic thalamus, or pons;[1] (3) in lesions of the semicircular canals; (4) in disseminated sclerosis and hereditary ataxia; (5) in certain trades, as miner's nystagmus, in which the eyes are strained in some given direction.

The **Treatment** of nystagmus is sometimes satisfactory

[1] The nucleus of Deiters has been demonstrated, by the researches of the Drs. Alexander Bruce, to be immediately connected with the 6th nucleus. As of Deiters' nucleus, one of its connecting fibres is often responsible for nysta

where it is the result of trade-spasm, rest often effects a complete cure.

4. *The 5th Nerve or the Trigeminal Nerve*

The course and distribution of the fibres of the 5th nerve, both sensory and motor, are shown in Fig. 39.

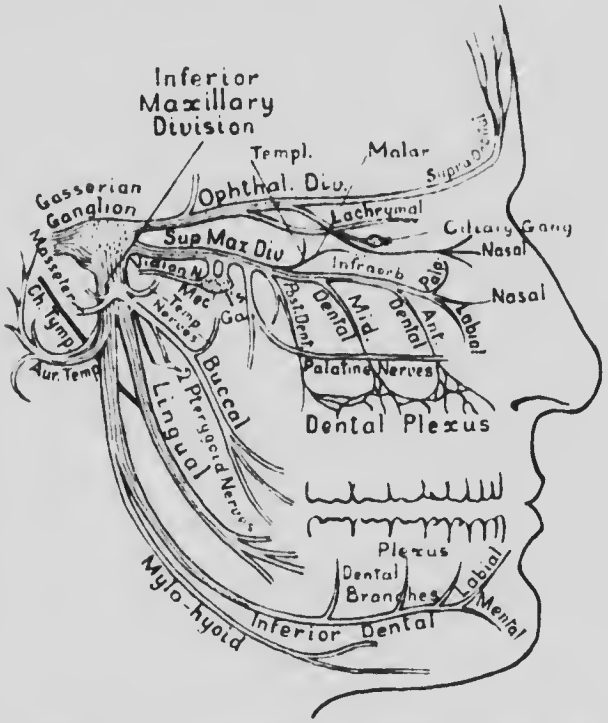


Fig. 39.—Schematic Representation of the Distribution of the 5th Nerve.

a PARALYSIS.—**Etiology.**— A lesion of the nerve may be due to the following conditions: *Outside the skull* a secondary neuritis is not uncommon, and may be the result of a tumour, or of irritation in the region of the parotid gland or the spheno-maxillary fossa. A primary neuritis due to cold, gout, syphilis, or other affection is rare.

At the base of the brain, tumours, meningitis, syphilis, and caries of bone may cause damage to the nerve.

In the pons, tumours, haemorrhage, softening, and sclerosis are not uncommon; and chronic diseases, such as bulbar

paralysis, tabes, and syringomyelia, may be associated with an involvement of the nerve.

The *first* division may be affected by pituitary tumours, aneurisms of the internal carotid and cellulitis in the orbit, while the *second* and *third* divisions more frequently suffer from a lesion in the spheno-maxillary fossa.

The sensory nuclei are widely separated, and part may escape where the lesion is nuclear. Supranuclear lesions also rarely involve the whole of the nerve, the motor fibres generally escaping. Paralysis of the motor fibres of the 5th nerve is often the result of a lesion of the nerve, and not of a lesion of the nucleus, while occasionally a cortical lesion results in a more or less complete involvement of the masticatory muscles.

Clinical Features. The nerve supplies common sensation to one-half of the face, a great part of the side of the head, the conjunctiva, the mucous surfaces of half of the mouth and tongue, and one nostril. In the figure the dark-shaded area is totally anaesthetic, the dotted part is only partially so, and is the area of epicritic overlap. A lesion, therefore, of the whole of the nerve will cause a hemianaesthesia of the region mentioned. It is, however, rare to find such an extensive lesion. Pain is extremely common. It may be sharp and shooting, and the painful points which indicate the foramina where branches of the nerve become superficial are typical of neuralgia of this nerve. When the patient drinks he only feels the cup with half of the lips, this giving him the impression of a bit being broken out of the cup. The nasal mucosa is affected because it becomes dry, and, therefore, the olfactory nerve-endings are incapable of receiving proper impressions. The secretion of saliva and of tears is lessened, and where there is any irritation of the nerve, ulceration is extremely common, and may cause inflammation of the cornea with eventual panophthalmitis, while the teeth may loosen

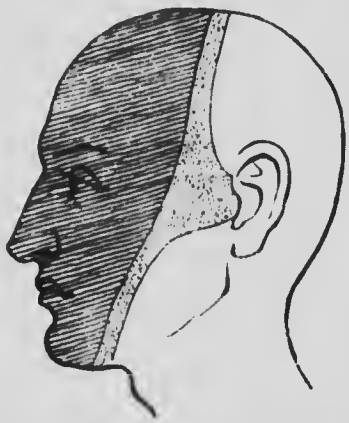


Diagram showing the sensory supply of the fifth nerve. Dark shading indicates the anaesthetic area after division of the nerve. Dotted shading shows partial anaesthesia or epicritic overlap.

and the gums ulcerate. At first it was thought that these trophic changes depended on interference with the Gasserian ganglion, but the ganglion has been removed for severe cases of trifacial neuralgia without any trophic changes occurring. On the other hand, herpes frequently involves the branches of this nerve, especially the supra-orbital branch of the first division, and undoubtedly it is due to trophic irritation.

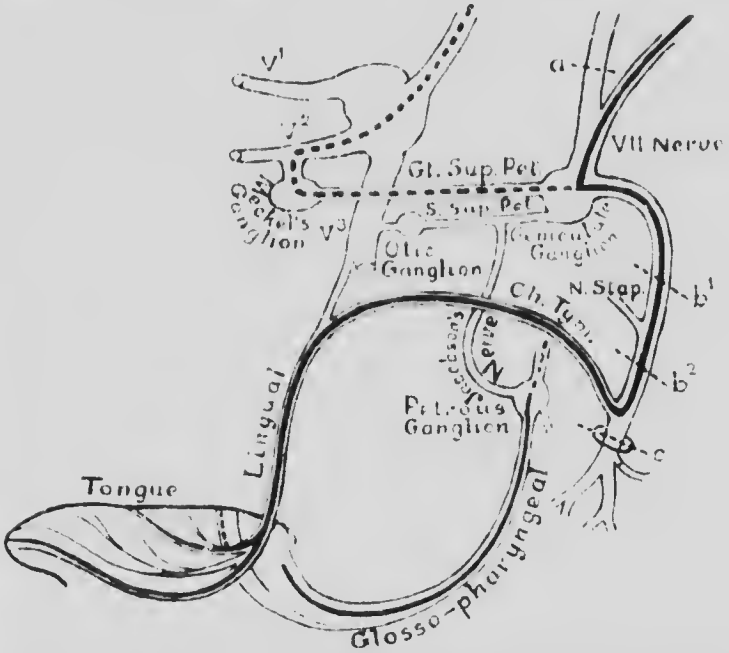


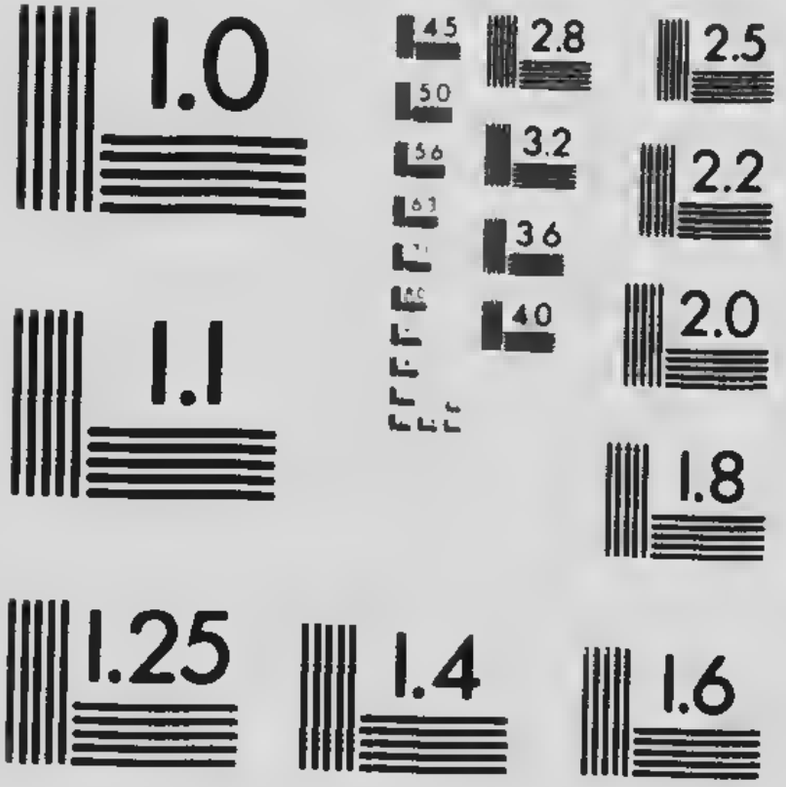
Fig. 168.—Shows probable routes of the [illegible] the gustatory fibres of the [illegible] (Chorda tympani) the gustatory area (the [illegible] the [illegible] and palatine arches. The chorda [illegible] shown [illegible] with the 7th Nerve, and leaving it [illegible] the geniculate ganglion by the nervus intermedius of Wrisberg (the sensory root of the 7th). The glossopharyngeal fibres may be traced to a ganglionic line to the petrous ganglion. The [illegible] the soft palate are indicated by [illegible] which they join with the [illegible]

Taste is supplied to the anterior two-thirds of the tongue by the chorda tympani nerve, which joins the lingual branch of the third division of the 5th, the taste fibres probably passing by the chorda tympani along the 7th nerve to the geniculate ganglion, and thence by the nervus intermedius of Wrisberg to the brain.

The motor portion of the 5th nerve supplies the muscles of mastication namely, the masseter temporal, and two pterygoids.

MICROCOPY RESOLUTION TEST CHART

(ANSI and ISO TEST CHART No 2)



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The motor root passes behind the Gasserian ganglion, and its position may be seen in the figure of the nerves at the base of the brain, while by reference to the sketch of the nuclei of the cranial nerves (Fig. 35), the relationship of the motor nucleus to the sensory will be made clear.

In paralysis of the whole motor root the patient cannot bite on the affected side, and the muscles are felt to be soft and flabby when he attempts to clench the jaws. Food tends to accumulate between the cheek and the gums on the affected side, and the three following alterations as regards movement and position may be noted:—

(1) The lower jaw cannot be moved toward the sound side.

(2) When the lower jaw is depressed, it is displaced towards the paralysed side, and

(3) When the month is wide open, the condyle of the lower jaw projects markedly on the paralysed side.

The **Diagnosis** of a lesion of the 5th nerve is easy, but it may be difficult to ascertain its exact position. If the Gasserian ganglion is the site of affection, all the divisions and often the motor root are involved. If the lesion is in the pons, it is usually bilateral, and if in the posterior limb of the internal capsule, there is generally hemianaesthesia.

The **Prognosis** will depend on the nature of the lesion.

Treatment.—Pain may have to be relieved by the use of morphia or local anaesthetic preparations. Syphilis should be appropriately treated, and sometimes strychnine, together with galvanism or faradism, proves beneficial in paralysis.

Trifacial neuralgia is discussed on p. 597.

(*b*) Spasm of the Muscles supplied by the 5th Nerve. —Masticatory Spasm.—Trismus.—Masticatory spasm may be *tonic* or *clonic*. The tonic form is exemplified in tetanus or lockjaw and the tonic spasm of an epileptic fit. Clonic spasms occur in rigors, in hysteria, and in the clonic stage of epilepsy.

(5) *Affections of Taste*

Taste is confined to the tongue, the soft palate, and the palatine arches in the neighbourhood of the tongue.

1. The anterior two-thirds of the tongue is supplied with taste through the chorda tympani nerve which joins the lingual branch of the fifth. Tracing the chorda tympani

towards the brain, it enters the aqueduct of Fallopius by the *posterior iter* and runs along with the 7th nerve to the geniculate ganglion. From the ganglion the taste fibres pass either by the great superficial petrosal nerve to the second division of the 5th, or more probably by the sensory root of the 7th nerve—in other words, the nervus intermedius of Wrisberg, to the pons.

2. The posterior third of the tongue is supplied with taste fibres through the glossopharyngeal nerve. Some of these fibres probably reach the brain through Jacobson's nerve and the small superficial petrosal to reach the otic ganglion on the third division of the 5th nerve, while other fibres may possibly, as in Fig. 41, pass upwards by the root of the glossopharyngeal nerve itself (see Fig. 41).

3. The soft palate and palatine arches are supplied by taste fibres which, traced towards the brain, pass through the geniculate ganglion and the great superficial petrosal to the second division of the 5th nerve, so that although the chorda tympani fibres pass by the nervus intermedius of Wrisberg, and probably not by the great superficial petrosal to the second division of the 5th nerve, the great superficial petrosal still carries some taste fibres.

The taste fibres pass eventually to the anterior end of the temporo-sphenoidal lobe, where there is a cortical centre in close apposition to the olfactory, but the pathway by which the fibres reach this centre is not yet satisfactorily determined.

Loss of taste is described as *Ageusia*, perversion of the sense of taste is called *Paragcusia*, and this latter condition is not infrequently due to hysteria or insanity.

Taste is closely associated with the sense of smell, and the appreciation of a good dinner and of the bouquet of wine is more dependent on the integrity of the olfactory nerve than of the gustatory.

In testing taste we must remember that by the gustatory nerve we can only distinguish (1) bitterness, (2) sweetness, (3) saltness, and (4) acidity, and it is easy by using quinine, sugar, common salt, and vinegar, to determine whether these four primary properties of the sense of taste can be appreciated by the patient. We must be careful to prevent the tongue from being drawn into the mouth after the test solution has been applied, and the patient should indicate

the taste he appreciates by writing instead of by speech. The well-known metallic taste produced by a feeble galvanic current passed through the tongue is a satisfactory test of the gustatory sense.

(6) *The 7th Nerve*

The motor area from which the facial fibres originate is situated in the lower part of the ascending frontal convolution. From this centre, the fibres pass downwards through the internal capsule, decussate, with the exception of a few fibres, in the middle of the pons, and terminate in the 7th nucleus, which is situated in the lower part of the pons, and just internal to the ascending root of the 5th nerve. From the nucleus the 7th nerve passes round the 6th nucleus, forming a loop, and emerges just external to the 6th nerve at the lower part of the pons. The nerve, in close relationship with the 8th, enters the internal auditory meatus, and passes through the petrous part of the temporal bone, emerging from the skull by the stylo-mastoid foramen. In the petrous part of the temporal bone the geniculate ganglion is situated on the 7th nerve, and at that point the chorda tympani fibres enter the nerve to leave it at the posterior iter and prior to its emergence at the stylo-mastoid foramen. The sensory division of this nerve is the nerve of Wrisberg, whose fibres have their cell centres in the geniculate ganglion. The area of skin supplied by the sensory division of the 7th nerve is limited to the region of the external auditory meatus and the skin just in front of the ear, and on this a herpetic eruption is sometimes seen, due to a lesion of the geniculate ganglion cells.

(*a*) PARALYSIS.—Paralysis of the muscles supplied by the 7th nerve may therefore be due to a lesion in several different positions: (1) The lesion may be cortical. (2) It may be in the internal capsule or in the pons, at any rate between the cortical centre and the nucleus. (3) The lesion may be in the nucleus of the nerve. (4) The lesion may be in the nerve itself—Bell's paralysis—and it may involve the nerve (*a*) proximal to the geniculate ganglion, (*b*) between the geniculate ganglion and the departure of the chorda tympani fibres to join the lingual branch of the 5th nerve, or (*c*) after these fibres have left the nerve (see Fig. 11).

In supranuclear lesions the orbicularis palpebrarum and

frontalis muscles are generally unaffected, and emotional movements are well preserved, while in nuclear and nerve lesions all such movements are lost. This escape of the upper face muscles may be due to their innervation in part by the fibres which do not decussate in the pons.

Etiology. The *cortical centre* may be injured by tumours, abscesses, softening, or haemorrhage, and in the pons similar lesions may occur. In the region of the internal capsule haemorrhage is most commonly situated in, or near, the lenticular nucleus, and as the face fibres pass down in the genu, it not infrequently happens that pressure on the internal capsule involves these fibres as well as those of the arm and leg. Thrombosis and embolism more frequently affect the cortical centre than either the tracts or the nucleus.

The *nucleus* may suffer along with other nuclei in cases of bulbar paralysis, and among the causal lesions may be chronic toxic conditions such as occur in bulbar palsy, encephalitis, softening, haemorrhage, or tumour.

The *nerve* is most commonly paralysed as the result of exposure to cold, and the resulting parenchymatous neuritis is of rheumatic origin. It is most marked in the nerve at, or distal to, the stylo-mastoid foramen. It is possible that these rheumatic cases may turn out to be of infective origin, because epidemics of Bell's paralysis have occurred, and several cases have been seen in the same family. Syphilis and diphtheria also induce neuritis of this nerve, but the second most common cause is suppuration in the middle ear. Tumours in the cerebello-pontine angle, meningitis, or haemorrhage are occasionally responsible for Bell's paralysis, while distally, operations on the lower jaw, parotid tumours, and tubercular and other glandular enlargement in the neck may also interfere with the nerve.

A bilateral lesion implies— (1) middle-ear disease on both sides, (2) a basal lesion, probably meningitis, and in most cases also the involvement of other nerves, (3) a bilateral lesion in the pons, and (4) a bilateral cortical lesion, which last is extremely rare.

Pathological Anatomy.—In cases of encephalitis the nuclear cells may show marked changes, similar to what is found in the motor cells of the anterior horn of the cord. No special description requires to be given of parenchymatous

or interstitial neuritis, the changes noted are similar to those described under these forms of neuritis. Where the nucleus or nerve is seriously involved, the affected muscles show marked atrophic changes: but, on the other hand, where the lesion is supranuclear, any wasting of muscles is the result of long-standing disuse.

Clinical Features.—In a case of Bell's paralysis, and in cases where the lesion involves the nucleus of the nerve, there is complete loss of power in the muscles affected, with as already noted, loss of the movements representing emotional expression. The skin is smooth and the wrinkles disappear on the affected side. The eye on the affected side cannot be closed, because the levator palpebrae muscle acts unopposed, and when the attempt is made, the eyeball is rotated upwards under the lid. In time, the lower eyelid droops, and tears are apt to run down the face, because the canaliculus is not kept in accurate apposition to the eyeball. The patient cannot whistle, show the teeth, wrinkle up the face, or move the ear, and he smiles with one side of the face only. Drinking is difficult, because of the impossibility of keeping the lip closely applied to the glass, and, on chewing, the food tends to collect between the cheek and the teeth. Speech is sometimes slightly indistinct owing to the difficulty of using the lips. The palate and uvula are not affected, because the levator palati and azygos uvulae muscles are innervated by another nerve.

Taste may, or may not, be abolished in the anterior two-thirds of the one side of the tongue, this depending on whether the nerve is affected after the taste fibres have joined the 7th nerve and before they have left it in the chorda tympani nerve, or not. The involvement of taste would therefore mean that the lesion is situated in the internal auditory canal, and would most probably be due to ear disease, or to extension of the inflammation upwards. The stapedius muscle is supplied by a branch from the 7th nerve, and where this branch is paralysed,[1] the tensor tympani acts unopposed, rendering the drum of the ear unduly sensitive to loud sounds.

Where the lesion involves the nucleus or nerve, the facial muscles supplied by the nerve waste and show after a period of seven to ten days a more or less pronounced reaction of

[1] In Fig. 11 b^1 shows position of lesion which involves stapedius branch, b does not.

degeneration, while faradic stimulation of both nerve and muscles should be lost. In less severe cases a modified reaction of degeneration is obtained, and recovery may be hoped for in one to two months (see p. 580).

There is often pain behind the ear, and very occasionally a herpetic eruption, indicating an inflammatory lesion in the geniculate ganglion, is present in the external auditory meatus and over an area in front of the ear. Still more rarely the herpes is wider in distribution.

The development of the paralysis is usually rapid when it follows exposure to a cold wind, and in most cases recovery results after a considerable number of weeks. Where only a modified recovery occurs, there may be a marked degree of contracture of the affected muscles, causing deep wrinkles on the paralysed side, and giving the suggestion, on a cursory examination of the patient, that the healthy side, being the more smooth, is the paralysed one. This contracture is sometimes extremely painful.

Should the nerve be involved by a lesion between the internal auditory meatus and the site of emergence of the nerve from the medulla, the auditory nerve amongst others is apt to suffer.

Where the lesion is supranuclear, the paralysis is incomplete, and usually involves the muscles of the lower part of the face. The patient can close the eye, although not quite perfectly; and the closed lid, if attempted to be raised, is found to be less resistant than that on the healthy side. Emotional movements are retained, and electrical changes are absent. If the lesion is cortical, there may be Jacksonian epileptic seizures, and if the pons is affected, crossed paralysis is sometimes present (p. 711).

Diagnosis.—There is little difficulty in the recognition of facial paralysis, and it is generally easy to diagnose the position of the lesion where it is cortical, near the internal capsule, or in the nerve once it has entered the internal auditory meatus; but it is sometimes difficult to be certain that the lesion is nuclear. It is true, however, that if one nucleus is affected, other neighbouring nuclei will likely suffer.

The **Prognosis** depends largely on the results of electrical examination. A modified or incomplete reaction of degeneration implies a speedy, and probably complete, recovery, whereas

a complete reaction of degeneration often indicates permanent paralysis.

Treatment. In most cases apply counter-irritation—either blisters or the button cautery—near the stylo-mastoid foramen. Perhaps the author favours this treatment because he has always believed many of the cases due to "cold" are interstitial neuritis, a view not generally held. In syphilitic cases iodide of potash should invariably be given. Once the inflammatory changes in the nerve have subsided, an attempt should be made to keep up the nutrition of the affected muscles. This may be done by applying the positive pole behind the ear, and the negative over the paralysed muscles, while massage may sometimes be used. A careful examination of the ear is also necessary. The best operative interference in cases in which improvement has ceased, is unquestionably to join the 7th nerve to the spinal part of the spinal accessory. Wonderfully hopeful results have followed this treatment, although movements of the arm may produce twitching of the face, owing to the stimuli overflowing into the 7th nerve as well as passing along the spinal accessory. The hypoglossal nerve is sometimes preferred for anastomosis.

(b) Facial Spasm.—This spasm is in part considered under the affection *tic convulsif*, and in part under such diseases as tetanus, tetany, epilepsy, and eclampsia. The movements may be twitching, clonic movements, or muscular spasms of a tonic nature.[1] They are often due to an irritant such as a carious tooth, or to exposure to cold, or may be the result of some emotional disturbance. It should, however, be remembered that there may be in the nerve itself some cause of irritation, such as a tumour or other irritative lesion.

7. *The 8th Nerve, or Auditory Nerve*

The 8th nerve consists of two separate divisions, the *cochlear* and the *vestibular* (see Fig. 255), the cochlear specially subserving the function of hearing, and the vestibular equilibration.

The cochlear division arises from the spiral ganglion of the cochlea. The cells of the ganglion are bipolar, the peripheral processes going to the organ of Corti, the central

[1] Twitching movements of the muscles round the eye are called *blepharospasm*.

constitute the cochlear root which joins the vestibular to form the 8th nerve. The common trunk consisting of the two divisions lies in front of the cerebellum. The fibres of the cochlear division end in the accessory nucleus and the acoustic tubercle. The striae acusticae seen on the floor of the 4th ventricle are auditory fibres, which pass from the acoustic tubercle over the restiform body on their way to the auditory cortical centre. The cochlear nucleus is connected with the posterior corpus quadrigeminum and the internal geniculate body, while the fibres eventually reach the cortical auditory centre in the superior temporo-sphenoidal convolution.

The vestibular division arises from a group of bipolar cells called Scarpa's ganglion situated near the outer end of the internal auditory meatus. Peripherally the fibres pass to the semicircular canals, centrally they join the cochlear nerve, and after entering the medulla arborise with the cells of the vestibular nucleus.

The vestibular nerve carries the nerve impulses from the semicircular canals to the brain, and in particular to the cerebellum. Lesions of the semicircular canals closely correspond in their effect with lesions of the vestibular nerve, and vertigo is the chief symptom. The impressions received from the semicircular canals enable the centre in the cerebellum to carry on the processes necessary for equilibration, and unquestionably the cerebellum is the dominating centre.

In testing the integrity of the semicircular canals, the vestibular nerve and the tracts communicating with the cerebellar centre, (1) examine for Rombergism, (2) watch the patient walk with eyes open, and eyes shut, (3) make him carry out various movements necessitating the co-ordination of muscles, and (4) practise the rotation tests.

1. **Rombergism** is tested by making the patient stand with feet close together and eyes closed, and if he has a lesion of any part of the vestibular apparatus, he tends to sway and may actually fall, the swaying movements being more marked towards the affected side. A more severe test is to make the patient stand on one foot.

2. **Gait.**—This is best tested by making him walk in a straight line first with the eyes open and then with the eyes closed. He tends to sway towards the affected side should there be a unilateral lesion.

(3) **Co-ordination of Movements.** This may be tested by making the patient carry out various movements which necessitate the co-ordination of muscles. Make him hop forwards or backwards with eyes open and then with eyes closed.

(4) **Rotation.**—The patient is seated on a revolving chair made to turn at the rate of ten revolutions in twenty seconds; then the rotation is suddenly stopped and the patient requested to look first towards the side from which he has been rotated, and then in the direction of rotation. After rotation there is horizontal nystagmus towards the side from which rotation has been made when the patient looks towards that side, while there is absence of nystagmus when he looks in the direction of the rotation. In a normal patient such nystagmus should last for twenty-five seconds or more. If there is a lesion of the right labyrinth, and the patient be rotated from left to right, the nystagmus produced after stopping the rotation is due chiefly to the left labyrinth and will continue for the normal twenty-five seconds, while if he be rotated from right to left it will only continue for 10 to 15 seconds.

(a) PARALYSIS.—A lesion of the *cortical auditory centre* on the left side causes word-deafness, and the reader is referred to the description of auditory aphasia on page 715. In addition to tumours, softening, or other affection of the cortex, the lesion may involve fibres from the cortex passing to the posterior corpus quadrigeminum, or the internal geniculate body, or the fibres from them to the cochlear nucleus.

A lesion of the *cochlear nucleus* is sometimes associated with bulbar paralysis, haemorrhages, or softening in the medulla.

A lesion of the *auditory nerve at the base of the brain* may be due to tumours, meningitis, haemorrhage, or fracture of the skull. In locomotor ataxia a primary degeneration of the nerve has been described, but primary neuritis does not commonly affect it.

Lesions of the *cochlear and vestibular nerves in the ear.*—Many alterations of hearing are dependent on a fault in the passages and not upon any nerve defect. A lesion of the tympanic membrane may produce either hyperacusis or auditory hyperaesthesia, or may cause a greater or less degree of deafness: associated with the tympanic membrane the ossicles must be considered, the free movement of

which is essential for perfect hearing. Where there is imperfect aeration of the middle ear, deafness is not uncommon on account of the drum being indrawn, and with thickenings, or perforation of the tympanic membrane, a degree of deafness is inevitable. Tinnitus aurium is hyperaesthesia, and is mainly due to the cochlear nerve, whereas Ménière's disease is associated with an involvement of the domain of the vestibular nerve, namely, the semicircular canals and sometimes the cochlea supplied by the cochlear nerve. In the latter case tinnitus is a prominent feature of the attacks.

In all cases where there is either tinnitus aurium or Ménière's disease, the ear should be carefully examined, and in cases in which there is any interference with the auditory sense, the acuteness of hearing should be tested by the watch, by words spoken, and by the tuning-fork. While it is unnecessary to describe fully the method of testing with the tuning-fork, it may be remarked that where bone conduction of sound is better appreciated by the patient in the deaf ear than aerial conduction, the fault is usually in the auditory passages, while the nerve and the auditory centres and connecting fibres in the brain are normal. Where, on the other hand, aerial conduction of sound is better appreciated by the patient than bone conduction, it may be concluded, with a fair degree of certainty, that the error is due to a lesion of the nerve, or possibly the auditory centre, and not to a fault in the passages.

(b) Tinnitus Aurium.—Hissing, whistling, chirruping, humming, or thumping sounds heard in one or both ears. They frequently increase when the patient is in noisy surroundings. The thumping or pulsating type of sounds is synchronous with the pulse, and this is very commonly the result of atheroma in the internal carotid. It should be remembered that small or large veins may cause rushing sounds in anaemia, and it is probable that the lateral sinus is largely responsible for these sounds, but changes in the labyrinthine fluid may permit of the labyrinthine circulation becoming audible to the patient, and the cinchonism due to quinine is of this nature. Amongst the common causes of tinnitus aurium are the presence of wax pressing on the drum, thickening of the drum, and ankylosis of the ossicles of the ear. Sometimes choreic twitching of the stapedius muscle becomes audible to the patient and in other cases, the gurgling sounds of fluid may be heard where an

effusion is present in the middle ear. Intracranial tumour may interfere with the circulation of the inner ear, and tumours of the cerebellum and the pons are specially associated with tinnitus aurium. It will be readily appreciated that in many conditions, such as thickening of the drum and ankylosis of the ossicles, deafness is frequently associated with the tinnitus.

There are, however, other cases in which a mental element is present, for example, where, in an insane individual, imaginary melodies or croaking human voices are in reality hallucinations of hearing.

(*c*) MÉNIÈRE'S DISEASE.—Sometimes with severe tinnitus aurium there is vertigo and a tendency to sickness and vomiting.

Etiology.—It is in many cases the result of a haemorrhage into the semicircular canals or other lesion of the inner ear, and it may be due to severe blow on the side of the head, while it sometimes occurs in persons exposed to excessively loud sounds, which may damage the inner ear. Hammermen, especially those working in the inside of boilers, and artillerists, are more frequently affected than other persons.

Clinical Features.—The attacks are paroxysmal; they occur at irregular intervals, perhaps several in one day, or an attack on successive days, or in other cases an isolated paroxysm. The patient has to sit or lie down in order to prevent himself from falling. There is generally tinnitus aurium, always vertigo, and a tendency to nausea and vomiting, while in most cases a certain degree of deafness results from the haemorrhage or other lesion. The attacks may pass off after rest, and sometimes after the total loss of hearing they gradually cease. Unfortunately this is not always the case, and, even with total deafness, the patient may be confined to bed owing to the constant dread of an attack occurring.

CHRONIC MÉNIÈRE'S DISEASE or symptomatic auditory vertigo may be present and yet may not produce the vomiting so commonly met with in acute cases.

Etiology.—It is not uncommonly the result of gout associated with arterio-sclerosis, and it may be a senile change of vascular origin (atheroma). In a number of cases of old suppurative middle-ear disease and thickening of the tympanic membrane, vertigo may also be present.

Clinical Features. There are practically always certain symptoms associated with labyrinthine vertigo which aid materially in the recognition of this symptom complex. These are a greater or less degree of deafness, tinnitus aurium, generally a nerve type of deafness, and there may be headache, often of congestive type.

Diagnosis. Labyrinthine vertigo is always associated with tinnitus. In some cases of epilepsy, vertigo is a typical feature, and epilepsy may complicate labyrinthine disease. The recognition of an *epileptic* element in the case is aided by loss of consciousness, although sometimes the differential diagnosis is very difficult. In *hysterical* cases of vertigo, deafness and tinnitus should not be present. In cases in which the cause of the vertigo is due to a tumour involving the auditory nerve, unilateral nerve deafness and tinnitus are generally associated. The recognition of the presence of a tumour, however, may have to be postponed until optic neuritis or the involvement of other important centres indicates the presence of a growing neoplasm.

Prognosis.—The prognosis should invariably be guarded, although much benefit may result from the removal of the semicircular canals on the affected side.

Treatment.—Probably the bromides in 20 to 30 grain doses is the best remedy, but should syphilis be suspected, potassium iodide may also be given. Quinine and salicylate of soda have been administered until cinchonism is produced, and ergot and cannabis indica are amongst the other remedies which have been suggested. Lastly, pilocarpine should be tried if all else fails; it sometimes produces the best possible results. In every case, a careful aural examination should be made, so as to determine whether the presence of wax, or defective aeration of the middle ear, may not greatly accentuate, if it does not entirely cause, the patient's distress.

(8) *The 9th Nerve, or the Glosso-pharyngeal Nerve*

This nerve supplies motor impulses to the stylo-pharyngeus muscle and the middle constrictor of the pharynx, while it also supplies sensory fibres to the upper region of the pharynx. It should be remembered that by the glosso-pharyngeal, taste

fibres are conveyed to the posterior third of the tongue, and secretory and vaso-dilator fibres to the parotid gland.

It is impossible to dissociate this nerve from the vagus nerve, the nuclei being closely related both in position and also in function; according to many authorities the glosso-pharyngeal forms a small and comparatively unimportant part of the vagus nucleus, or rather nuclei.

Paralysis of the nerve may be the result of a lesion in the medulla such as we find in bulbar paralysis. Sometimes the nerve is affected by tumours or by meningitis at the base of the brain.

Clinical Features.—There is anaesthesia of the upper part of the pharynx, and certainly difficulty in swallowing food while the loss of taste is confined to the posterior third of the tongue. The course of these taste fibres has been the subject of much discussion.

(9) *The 10th Nerve, Pneumogastric or Vagus Nerve*

This nerve supplies not merely an extensive area of the alimentary tract, but also the lungs and heart, and its involvement causes widespread changes throughout the body.

Paralysis of the Vagus.—**Etiology.**—A nuclear involvement is not uncommon in cases of bulbar paralysis, and is generally associated with a similar lesion of the nuclei of the glosso-pharyngeal, the spinal accessory, and the hypoglossal nerves. Meningitis may involve the nerve close to the brain, and tumours, aneurisms, and wounds in the neck may all cause paralysis of the nerve. Sometimes in the toxic forms of neuritis the vagus is affected shortly before death, and this may be the result of alcohol, diphtheria, and more rarely other toxins. Neuromata sometimes occur on the nerve, and may interfere with its functions.

Clinical Features.—It is impossible to consider all the branches of the vagus together, and therefore we describe the results of paralysis of the different branches separately.

1. *Branches to the Pharynx.*—The vagus supplies, along with the glosso-pharyngeal, the muscles of deglutition. There is therefore, in paralysis, difficulty in swallowing, and as a result the food tends to find its way into the larynx, where it excites spasm, or it may regurgitate upwards by the nose, especially if the soft palate is involved.

Spasm of pharyngeal muscles occurs in various diseases, as, for example, in hydrophobia.

2. *Branches to the Larynx.*—The superior laryngeal branch supplies sensation to the laryngeal mucosa, and is also the motor nerve for the crico-thyroid muscle. The recurrent laryngeal nerve supplies all the other muscles of the larynx, and these laryngeal fibres reach the brain by the spinal accessory root. The recurrent laryngeal nerve on the left side is frequently affected in cases of aneurism of the arch of the aorta, and on the right side it may be involved in pleuritic adhesions. In cases of laryngeal catarrh certain muscles tend to be paralysed, and the paralysis does not endanger life, but, on the other hand, toxic paralysis, as in diphtheria, and sometimes in rheumatism, involves the abductors of the glottis, and readily causes death by suffocation. Hysterical paralysis almost invariably affects muscles which do not endanger life.

(*a*) *Adductor Paralysis.*— This results from the involvement of the crico-arytenoideus lateralis muscle, or the arytenoideus muscle, or both, and it is commonly due to catarrh or hysteria. The laryngoscopic picture on attempted phonation shows total inability to oppose the cords together. Aphonia results.

(*b*) *Abductor Paralysis.*—This is paralysis of the crico-arytenoideus posticus, and it is commonly the result of the toxin of diphtheria. It may occur in bulbar paralysis, but is very rarely caused by hysteria or an ordinary laryngeal catarrh. The laryngoscopic picture shows total inability to open the glottis during inspiration, although there is no aphonia. This condition endangers life, as air cannot enter the chest, and there is invariably great stridor with inspiration.

Abductor paralysis may occur on one side only, in which case there is less tendency to suffocation, and, at least for a time, little hoarseness.

(*c*) *Paralysis of one Recurrent Laryngeal Nerve.*—This is the common result of aneurism involving the nerve on the left side. The abductor muscle is first paralysed, and the cord remains near the middle line, but later the adductors are also paralysed, and the cord becomes fixed midway between abduction and adduction in what is termed the "cadaveric" position. The sound cord crosses the middle line during attempted phonation, but the voice, as a rule, is hoarse, although there is not necessarily much difficulty of breathing.

Spasm of the muscles of the larynx is present in laryngismus stridulus, which is described separately on page 433, and also in locomotor ataxia, in which laryngeal crises are not uncommon.

3. *Branches to the Heart.*—The cardiac plexus is partly supplied by the vagus and partly by the sympathetic. The vagus retards the accelerator action of the sympathetic, and therefore in paralysis of the vagus the heart acts with abnormal rapidity; irritation of the nerve causes greater inhibition, and therefore slowing of the heart's action. It should be remembered that paralysis of one vagus is not necessarily sufficient to cause great acceleration of the heart, but when the vagi nerves are affected by the toxins of diphtheria, influenza, and other infective fevers, very rapid cardiac action may result.

The sensory fibres of the vagus which go to the cardiac plexus may possibly give rise to the pain felt in certain cases of angina pectoris.

4. *Branches to the Lungs.*—The pulmonary plexus is made up of branches from the vagus and also from the sympathetic system, and it is difficult to say how much influence the vagus alone has on the lungs. It is certainly true that pressure on the pulmonary plexus is apt to cause septic pneumonia. Probably the sensory fibres of the plexus supplying the bronchi are largely responsible for the production of the act of coughing.

5. *Branches to the Stomach and Oesophagus.*—Paralysis of the oesophageal branches causes great difficulty in swallowing, while spasm of oesophageal muscles is a recognised feature both in hysteria and also in association with tumour or other organic stricture of the oesophagus.

The vagus supplies both sensory and motor fibres to the stomach, and upon the discharge of their functions the process of digestion largely depends. It is a well-known fact that vomiting is apt to be produced by stimulation of branches of the vagus, sometimes in the stomach itself, although often in some other branch of the nerve. Gastralgia or cramp in the stomach, probably the result of irritation of the sensory fibres of the vagus, and the gastric crises in locomotor ataxia, are unquestionably due to a lesion of the vagus.

It is very difficult to determine to what extent the vagus has been paralysed, although reference to the different branches will help, and in an involvement of the nerve trunk all its branches are apt to suffer to some degree. The close associa

tion of the glosso-pharyngeal nucleus renders it improbable that the nucleus of the one could be affected without an involvement of the other,[1] and the hypoglossal nerve is closely related to these two nerves in the first part of its course.

Treatment.—Little need be said with regard to treatment. Where laryngeal muscles are paralysed, an attempt should be made to discover the cause of the condition, and if it be remediable, the affected muscle may be treated by massage or stimulated by electricity. Generally both poles are applied, one on either side of the larynx, and either the galvanic or faradic current may be used. In cases where there is bilateral abductor paralysis, and if the cords do not speedily assume the cadaveric position from the coincident involvement of the adductor muscles of the larynx, tracheotomy must be performed.

(10) *The 11th Nerve, or the Spinal Accessory Nerve*

The accessory or medullary portion of the nerve supplies the laryngeal muscles, and the fibres leaving the medullary nucleus form the internal and smaller division of the nerve. This portion of the nerve is often included with the vagus for descriptive purposes. The other nuclear cells are situated in the cervical cord, and the fibres from them form the larger and external portion of the nerve, and supply the sterno-mastoid and the upper third of the trapezius muscles. The medullary nucleus is apt to be involved in bulbar paralysis, and the spinal nuclear centre may be affected in various cord affections.

(*a*) PARALYSIS.—The nerve may be damaged by tumours, fractures, and other injuries involving the cervical vertebrae and the region of the neck. The muscles supplied by the nerve are often affected in progressive muscular atrophy. Sometimes neuritis involves the nerve, and it is due most commonly to exposure to cold and wet.

Paralysis of the laryngeal fibres has already been described in connection with the vagus nerve. In paralysis of the fibres supplying the sterno-mastoid and part of the trapezius the patient is unable to turn the head towards the opposite side,

[1] In the schematic figure on page 631 a common sensory and a common motor nucleus is figured for the 9th and 10th nerves.

and the head may be retained in a slightly twisted position. The involvement of the trapezius is not complete, but shoulder movements are affected, and shrugging of the shoulders is carried out with great difficulty. The shoulder droops, and, owing to the unantagonised action of the rhomboids and the levator anguli scapulae, the scapular angle is rotated inwards. When the arm is raised, the scapula moves as a whole, the trapezius failing to fix the bone so as to permit of leverage for the deltoid muscle. It is hardly necessary to add that as the trapezius is only partially supplied by this nerve, the degree of paralysis will vary in different patients.

A bilateral paralysis sometimes occurs, more generally as the result of progressive muscular atrophy; if both sterno-mastoids are affected the head falls back, but if both trapezius muscles are also paralysed it falls forward, giving the typical picture of an advanced progressive muscular atrophy. In any case the movement of the head is very slight, and the movements of both arms are also interfered with. The affected muscles are often flabby and wasted, and the electrical examination shows the typical reaction of degeneration.

It is probable that the soft palate is supplied by fibres of the internal or medullary division of the nerve, and where, as in bulbar paralysis, the nucleus is affected, the voice becomes nasal from paralysis of the soft palate, and fluid drunk is apt to pass down the nose.

The **Diagnosis** is usually fairly easy, and the **Prognosis** depends largely on the nature of the affection; in both bulbar paralysis and progressive muscular atrophy, it is by no means favourable.

Treatment.—In rheumatic neuritis an attempt may be made by counter-irritation and electricity to aid in the restoration of function.

(*b*) Torticollis. Wry-Neck.—The muscles which produce this spasm are not solely those functionated by the 11th nerve, although the sterno-mastoid and trapezius are mainly responsible, but include the splenius, scaleni, and deep muscles of the neck and at times the platysma and omohyoid. Rheumatic torticollis or stiff-neck is described under muscular rheumatism, and is not a true spasm, and it is doubtful whether the first of the two forms to be described here, has any spasmodic element associated with it. The two forms are:

1. *Congenital Torticollis.*—This is the result of shortening of the sterno-mastoid muscle on one side. It is noticed soon after birth, and was believed to be due to traction on the head. In any case the affected sterno-mastoid is shortened and atrophied, and it has been suggested that the condition is similar to the lesion in infantile paralysis, in which talipes of the foot so often occurs. Any lesion which causes contraction of the sterno-mastoid will produce this condition, whether occurring in childhood or later life. Tenotomy affords relief.

2. *Spasmodic Torticollis.*—This is an affection in which there are active tonic and clonic spasms of the sterno-mastoid and other neck muscles.

The **Etiology** of spasmodic torticollis is unsatisfactory. Blows on, and injuries to, the region of the neck, and exposure to cold, are amongst the reputed causes, while the family history of patients affected, often includes epilepsy and other nervous affections.

Both tonic and clonic movements may be present in the same patient, but, as a rule, this is not so.

In the *tonic form* of spasmodic wry-neck, the sterno-mastoid on one side is contracted, and draws the occiput backwards towards the affected shoulder, rotating the face towards the healthy side and tilting the chin upwards. Where other muscles participate in the tonic spasm, they may modify somewhat the position of the head, and not infrequently the trapezius muscle so participates, drawing the head somewhat more downwards. There may be a certain amount of pain accompanying the tonic spasm. The affected muscles tend to hypertrophy.

The *clonic form* of spasmodic wry-neck is much more serious, and while the sterno-mastoid is generally involved and sometimes the trapezius, other muscles of the neck, such as the splenius and the platysma, may also be affected. These clonic movements may occur almost constantly, although they cease during sleep. They are increased by nervousness or excitement, and they are often accompanied by considerable pain. The clonic movements may extend to other muscles in the neighbourhood, adding greatly to the patient's discomfort.

The **Diagnosis** is easy and the **Prognosis** is only favourable as a rule in tonic cases, and only in certain of these is it

possible to afford relief. In a very few of the clonic cases the movements cease in course of time.

The **Treatment** consists partly in an effort to soothe spasm by sedative remedies such as the bromides, hyoscine, chloral, and even morphia, and partly, in severe tonic cases and with doubtful success, in dividing the external or spinal portion of the spinal accessory or in dividing the offending muscles. We have tried rhythmic exercises with distinct benefit. In cases of clonic wry-neck, treatment is often useless, and division of the spinal accessory nerve rarely affords much relief.

(11) *The 12th Nerve, or the Hypoglossal Nerve*

This is the motor nerve for the tongue, and for the muscles which protrude the tongue and which are attached to the hyoid bone.

(*a*) PARALYSIS.—**Etiology.**—A lesion of the nucleus is not uncommon in bulbar paralysis, and may be the result of encephalitis, sclerosis, or haemorrhage. Where the lesion is supranuclear, the nerve fibres may be involved along with the motor fibres for the corresponding half of the body in the region of the internal capsule, or in the cortical centre for the tongue. The nerve may be affected by meningitis and tumours in the neighbourhood of the base of the skull, while wounds or injuries of the nerve are occasionally met with. A primary neuritis very rarely occurs.

Clinical Features.—Where the lesion is supranuclear, there is no marked wasting of the muscles supplied by the nerve, whereas if the lesion is nuclear or infranuclear, wasting is a prominent feature, and in addition fibrillary tremors may be seen in the paralysed muscles. In nuclear or nerve lesions speech becomes difficult, and mastication is interfered with, even when the lesion is unilateral. In bilateral paralysis speech and mastication are greatly impaired, and the tongue cannot be protruded from the mouth. Where the lesion is unilateral, the tongue is protruded towards the paralysed side, as the result of the unantagonised action of the healthy muscles.

The **Diagnosis** is easy, and when it is remembered that in a supranuclear lesion there is little wasting and no fibrillary tremor, it is generally a simple matter to distinguish between a supranuclear and a nuclear or infranuclear lesion.

The **Prognosis** is often unfavourable, although in supranuclear lesions considerable improvement may result.

The **Treatment** should consist in the administration of antisyphilitic remedies, while electricity has been applied with benefit in certain cases.

(*b*) Spasm of the Muscles of the Tongue.—This is not uncommon in persons who stutter, while it may be associated with various nervous affections, such as epilepsy, chorea, and so forth.

III. DISEASES OF THE CEREBRAL AND SPINAL MEMBRANES

(1) Pachymeningitis

Inflammation of the dura mater.

(*a*) Pachymeningitis Externa.—This variety of pachymeningitis may occur where there is a fracture of the skull or any neighbouring infective focus from which the outer surface of the dura becomes involved. It follows middle-ear disease and sometimes caries of bone.

As regards the spinal meninges, external pachymeningitis may occur as the result of tuberculosis, sometimes from malignant tumours of bone, and more rarely from aneurism.

The lesion may be limited to the outside of the dura, or it may spread to the inner surface, and the clinical features in most cases are those of irritation and later compression.

(*b*) Pachymeningitis Interna.—This may be secondary to external inflammation of the dura mater.

The most important variety of internal pachymeningitis is unquestionably the form which bears the special name of pachymeningitis interna haemorrhagica.

Pachymeningitis Interna Haemorrhagica.—It consists in the formation of a false membrane on the inner surface of the dura mater, into which haemorrhage occurs; repeated haemorrhages may add greatly to the thickness of the dura.

Etiology.—This affection is found in certain forms of insanity, as the result of alcoholism and other conditions, such as profound anaemia, which cannot be well grouped together, but in which its appearance is more or less occasional.

Pathological Anatomy.—Virchow holds that a vascular

membrane is formed in the first instance, into which the extravasions of blood occur.

The **Clinical Features** are indefinite. There may be no symptoms whatsoever, or headache, twitchings, and irritative phenomena may be present, and may in turn give place to paralysis and coma from compression of the brain. Giddiness is not uncommon, and in most cases intellect and memory suffer seriously.

Pachymeningitis Cervicalis Hypertrophica.—In the spinal meninges there is a tendency to a pachymeningitis involving the cervical region, and in many cases there is great pressure exercised on the spinal cord at this level. It resembles and probably is a syphilitic meningitis, and the other membranes of the cord participate with the dura. In some cases the central canal is dilated. The nerve roots are apt to be pressed upon, and severe pains may eventually be followed by definite anaesthesia and often muscular atrophy. The muscles most affected are naturally those of the arms, the flexors of the fingers suffering especially, and from unantagonised action of the extensors, a fairly typical *main en griffe* is produced. In the legs, as a result of descending changes in the pyramidal tracts, spastic phenomena may develop. The disease runs a chronic course, and may resemble in some respects syringomyelia. Antisyphilitic treatment should be tried.

(2) Leptomeningitis. Acute Leptomeningitis

Inflammation of the pia-arachnoid.

Etiology.—There are several special forms of meningitis, such as epidemic cerebro-spinal meningitis (referred to under infective fevers), tubercular meningitis, and posterior basic meningitis (which is probably sporadic epidemic cerebro-spinal meningitis), to both of which reference will be made separately: but in addition to these, meningitis may result from many causes—

(1) It may be secondary to fracture or caries of the bones of the skull, and this includes a number of cases of middle-ear disease in which caries of bone has occurred.

(2) In cases in which there is a neighbouring focus of infection, as in middle-ear disease, mastoid abscess, or inflamma-

tions of orbit or nose, the membranes may become secondarily affected without necessarily any caries of bone.

(3) In acute infective fevers, such as pneumonia, erysipelas, septicaemia, and more rarely influenza, typhoid, and small pox, meningitis may occur.

(4) In pyaemia, infective organisms may be carried, directly or indirectly, to the meninges of the brain and cord.

(5) Not infrequently, as a terminal stage of certain chronic diseases, such as Bright's disease, heart disease, etc., there may be infection of the meninges by pus-producing organisms, although the exact mode of attack is not always clear.

Pathological Anatomy.—The meningitis may be basal or cortical, the type depending on the cause of the condition and the original focus of infection, if any. In some cases it may be unilateral, as when secondary to ear disease, and as a general rule, unless free communication is interrupted, the spinal meninges suffer along with the cerebral. The effusion is generally purulent. It is usually sub-arachnoid in position, and it may cause very serious pressure on the cortex of the brain.

Clinical Features.—Along with the clinical features associated with the primary disease, the symptoms suggestive of the meninges having become involved are the following:—A temperature of about 103° F., with headache, sometimes great sensitiveness to light (photophobia) and sound, and often delirium, merging eventually into coma. There is rigidity of muscles, particularly those of the neck, and sometimes convulsions, while paralysis of certain muscles, and more especially those supplied by the cranial nerves, may be noted in cases in which the effusion is mostly basal. When the spinal meninges are specially affected opisthotonos may be noted. There is occasionally Cheyne-Stokes breathing, and sometimes the *tache cérébrale*. Cerebral vomiting is an early symptom, and may be associated with retraction of the abdomen. Optic neuritis is distinctly rare. Constipation is generally described as being a typical feature, and often certain areas of skin, especially on the neck and calves, become hyperaesthetic. The pupils may yield suggestive information. Irritation produces contraction, but later during the paralytic stage the pupils become widely dilated. The pulse and respirations are often irregular and abnormally slow, considering the degree of temperature present. Kernig's sign is generally

positive. It is carried out by flexing the leg on the thigh at right angles, the knee being bent, and then attempting to fully extend the leg. In health this can be easily done, but in cases of meningitis, spasm of the hamstring muscles prevents full extension. A leucocytosis is not uncommon, and lumbar puncture offers important evidence both of the presence of pus cells and of excess of albumin in the cerebro-spinal fluid. Occasionally cultures made from the fluid yield definite information.

Diagnosis.—Although cases differ greatly in degree and severity, the clinical features are fairly suggestive and typical, while the evidence obtained by lumbar puncture is most important.

Prognosis.—Death is extremely common. In non-purulent cases there may be recovery, but this is rare.

Treatment.—The head should be shaved and an ice-bag placed over it, while local blood-letting, by leeching over the mastoid, is often of great value where the patient can stand the operation. Counter-irritation is certainly useful, and blisters to the back of the neck are commended. Any suggestion offered by lumbar puncture should be followed up, and in certain cases antistreptococcic serum might be administered either hypodermically or by the rectum. The fluid removed by lumbar puncture (10 c.c.) often temporarily relieves the patient. Sometimes trephining is warranted, but it is improbable that much benefit will result from such treatment. The bowels should be freely opened, and the temperature kept in check by sponging. Opium is often necessary for the relief of pain, and the patient should be kept at absolute rest in a darkened room. In most cases the spinal meninges suffer secondarily.

(3) Tubercular Meningitis

Etiology.—It occurs most commonly in children, and usually under the age of seventeen, although sometimes it develops in adults. It is certainly very rarely primary.

Pathological Anatomy.—The membranes at the base of the brain are infiltrated with a yellowish, gelatinous exudation. Tubercles, often microscopic in size, may be seen following the course of the vessels, and especially in the neighbourhood of

the optic commissure and the Sylvian fissure. Frequently the lateral ventricles are greatly distended with turbid fluid,[1] and the convolutions are markedly flattened as a result of pressure. It is a special type of leptomeningitis.

The spinal meninges are also affected, although in some cases not quite to the same extent. There may be tubercle nodules in the choroid coat of the eyeballs, sometimes even involving the retina; a primary lesion, sometimes a gland in the abdomen, more frequently an involvement of the lungs or of the genito-urinary tract, may also be recognised.

Clinical Features.—There are often definite premonitory symptoms. The child becomes peevish and out of health, and the disease is specially liable to develop after a continued fever such as measles or whooping-cough.

During the *stage of irritation* many of the most typical phenomena develop, such as headache, which is intense, and causes the child to give utterance to a short, sharp cry, often called the "hydrocephalic" cry. There is cerebral vomiting and fever, the temperature sometimes falling towards morning with considerable sweating. The little patient is excessively restless, starting out of sleep, and muscular twitchings are very common. During stage the pupils are contracted. Various spasms, especially muscles supplied by intracranial nerves, may be noted, and, in particular, squinting, whil head is bent back, the child apparently boring the occiput in the pillow, and the abdomen is often markedly retracted. Kernig's sign is positive. The breathing and pulse are irregular, especially the latter, and the *tache cérébrale* is usually noted.

This stage of irritation is generally followed by a *stage of depression or paralysis*, during which an ocular squint may develop, but of paralytic origin; the patient tends to become comatose, and convulsive seizures occur from time to time. The pupils, no longer contracted, are widely dilated, and the child generally sinks into what is best described as the typhoid state. An extensor plantar response is often present in older patients. During this period the temperature remains high, and usually a leucocytosis, if not definitely present before, is noted now. Sugar may be present in the urine in the later stages. The cerebro-spinal fluid obtained

[1] A condition which used to be called *acute hydrocephalus*.

by lumbar puncture shows turbidity, often a considerable increase of lymphocytes and tubercle bacilli may be discovered.

Diagnosis.— The eyes should be invariably examined for the presence of choroidal tubercles, but it must be remembered that only in 50 per cent of cases of acute generalised tuberculosis do they occur. The classic phenomena, such as the retraction of the head, ocular squint (either spasmodic or paralytic), retraction of the abdomen, and the *tache cerebrale* are very suggestive, and not infrequently a tubercular family history and the discovery of the primary focus of disease greatly assist the physician.

Prognosis.— Occasionally a temporary improvement occurs and the hopes of doctor and friends alike may be raised, but too often this is followed by a relapse with more marked symptoms, and in every case a grave prognosis should be given. In only an isolated case does recovery occur, and post-mortem it is rare to find evidence accidentally of an old quiescent basal meningitis of tubercular origin.

Treatment. Unfortunately little benefit is derived even from the most careful treatment. An ice-bag over the head is advantageous, and sometimes the use of counter-irritants, but in every case the patient experiences relief from being kept absolutely quiet and in a darkened room.

4 Posterior Basic Meningitis

This is probably a sporadic form of epidemic cerebro-spinal meningitis. It is more common in early childhood, and it is peculiar because of the very limited area involved by the purulent exudate, and it is to this that the name "posterior basic" is due. Otherwise it comes on much like epidemic cerebro-spinal meningitis, and the clinical features and prognosis closely correspond.

5) Syphilitic Meningitis

When it is remembered that the syphilitic infection early involves the cerebro-spinal fluid, it is easily realised how often spinal cord and membranes suffer in syphilis. Among the common lesions is chronic meningo-myelitis. It commences 2 to 8 years after the primary infection. It is often preceded

by some sign of cerebral syphilis, such as hemiplegia, diplopia, or persistent headache. There is pain in the back and shooting down the legs due to the meningitis—the later manifestation being dependent on the involvement of the cord. Gummatous radicular neuritis is invariably accompanied by a localised meningitis, and in most syphilitic cord lesions the meninges suffer more or less severely.

IV. DISEASES OF THE SPINAL CORD

(1) Introductory

The position of the great ascending and descending tracts of the cord are seen in the figure, which shows a transverse section of the cord just at the lower part of the cervical enlargement (see Fig. 42).

The grey matter of the cord is made up of two anterior horns and two posterior horns, and each half is joined by a commissure of grey matter surrounding the central canal of the cord. In the anterior horn the cells are arranged in groups, but there is no uniformity about the grouping in the different segments, for in the upper cervical and dorsal regions of the cord there is only a single group of multipolar cells, whereas in the cervical and lumbar enlargements there are several groups. The motor nuclei in the anterior horn supply the muscles but do not represent centres for co-ordinated movement. Indeed most of the larger muscles are supplied with nerve energy by cells in more than one segment of the cord.

The group of cells in the intermedio-lateral horn, which is situated between the 8th cervical and the 2nd lumbar segments, was believed by the late Dr. Bruce to be an important controlling centre belonging to the sympathetic system.

The anterior nerve-roots contain mainly motor fibres. The very fine nerve fibres which are found mingled with the coarser ones (which undoubtedly pass to voluntary muscles) are almost certainly intended for the sympathetic nervous system which they reach by the rami communicantes.

Descending Tracts.—The *anterior or direct pyramidal tract* contains fibres which have not decussated in the medulla,

but decussate in the cord before reaching the anterior horn cells to which they conduct impulses. The tract terminates about the mid-dorsal region, and in a small proportion of cases it is absent altogether.

The *crossed pyramidal tract* is situated in the lateral columns and extends downwards from the top to the bottom of the spinal cord. Below the level of commencement of the tract (in the lower dorsal region) the crossed pyramidal tract reaches the periphery of the cord. Its axis-cylinder processes, having already decussated, arborise round the multipolar cells in the anterior horn of the cord on the same side. These tracts degenerate from above downwards after section or similar lesion. These are the two motor tracts.

The *vestibulo-spinal tract* commencing in the cells of Deiter's nucleus passes down the antero-lateral column of the cord to end in arborisations around spinal motor neurones.

The *rubro-spinal tract* arising from the opposite red nucleus passes down in the antero-lateral column just internal to the antero-lateral tract of Gowers. It carries down cerebellar impulses to the spinal motor neurones, and its fibres terminate in arborisations near the base of the anterior horn. The tract may be traced to the mid-dorsal region.

The *descending tract of Schultze* and the *septo-marginal tract*, both of which may be seen in the figure, are also descending tracts, and are probably of endogenous origin, by which is meant that the fibres arise and terminate in the cord.

Ascending Tracts.—The posterior columns of the cord are divided into two important tracts, the *postero-median* or *tract of Goll* and the *postero-external tract*, the former ending in the *nucleus gracilis* and the latter in the *nucleus cuneatus* in the medulla. Fibres in the postero-internal columns pass up mainly on the same side as that on which they enter, while many of the fibres in the postero-external tract leave the tract either to pass into the postero-internal or turn back into the grey matter of the posterior horn. A lesion of the posterior columns low down in the cord results in ascending degeneration which, immediately above the lesion, is found to involve both the postero-external and postero-internal tracts, but at a higher level healthy fibres from healthy posterior nerve-roots have filled the postero-external tract with undegenerated

fibres, and even in the postero-internal tract that part of it nearest the commissure contains many healthy fibres. Should the lesion, however, be situated in the cervical region of the cord, the postero-external columns are also extensively degenerated right up to the medulla.

The *direct cerebellar tract*, sometimes termed the dorsal spino-cerebellar tract, originates in the cells of Clarke's

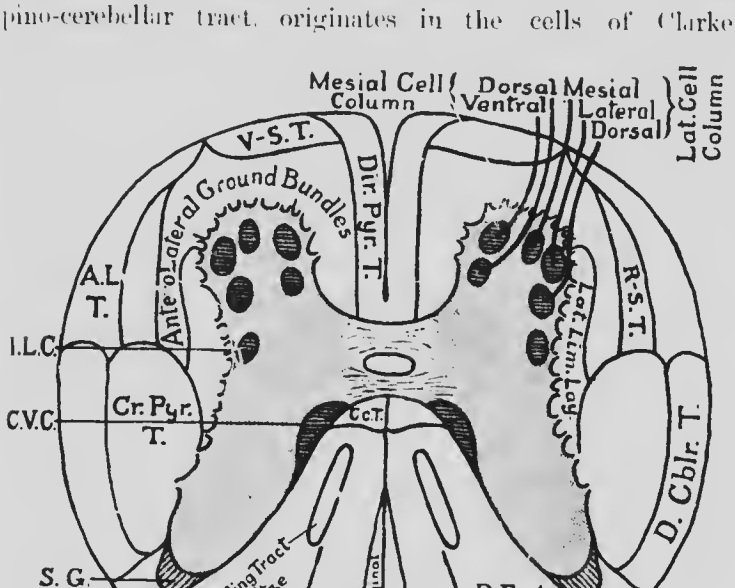


FIG. 42.—Diagrammatic Representation of Position of Tracts in the Spinal Cord. D.Pyr.T., Direct Pyramidal Tract. Cr.Pyr.T., Crossed Pyramidal Tract. P.Int.T., Postero-Internal Tract. P.Ext.T., Postero-External Tract. D.Cblr.T., Direct Cerebellar Tract. Lat.Lim.Lay., Lateral Limiting Layer. A.L.T., Antero-Lateral Tract or Comma-shaped Tract of Gowers. Lis.T., Lissauer's Tract. V-S.T., Vestibulo-spinal Tract. R.S.T., Rubro-spinal Tract. Cc.T., Comm-commissural Tract. S.G., Substantia Gelatinosa. C.V.C., Clarke's Vesicular Column. I.L.C., Cells of Intermedio-lateral horn.

vesicular column (which cells can be traced upwards from the upper lumbar region of the cord), and the direct cerebellar tract begins in the lower dorsal region. It unquestionably conveys up impulses to the cerebellum.

The *antero-lateral tract of Gowers*, or the ventral spino-cerebellar tract, arises from cells situated in both posterior horns, and the fibres pass into the medulla and eventually reach by way of the superior cerebellar peduncle the middle

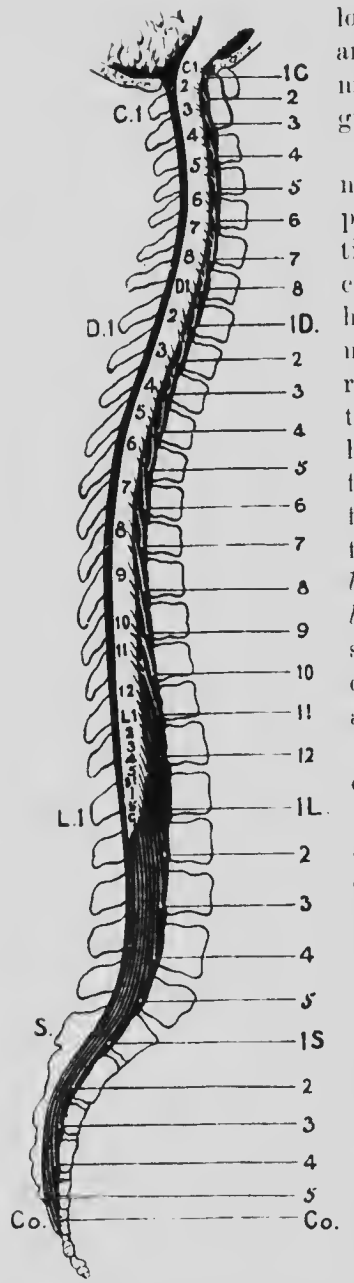


FIG. 43.—Figure showing relationship of the Segments of the Cord to the Vertebral Spines, and also the Termination of the Spinal Cord in the Canal.

lobe of the cerebellum. The antero-lateral tract of Gowers may be traced throughout the greater part of the cord.

The *tract of Lissauer* (which may be noted in the figure just posterior to the substantia gelatinosa capping the posterior horn) contains small-sized fibres which have entered at the posterior nerve-roots, and which, after running for a short distance in the tract, pass into the posterior horn. As previously noted, these fibres probably subserve the functions of the superficial reflexes. The *antero-lateral ground bundles* and the *lateral limiting layer* are also part of the white substance of the cord, and they contain mostly endogenous and association fibres.

In Fig. 43 the relationship of the segments of the cord to the spinal canal becomes clear, and especially the fact that the cord does not extend much below the first lumbar spine, so that in performing lumbar puncture a lower level is always selected. For a full description of the structure of the spinal cord reference must be made to special text-books.

Lumbar Puncture.—Lumbar puncture and the diagnostic value of the examination of the cerebro-spinal fluid are so important that a brief description is essential. Place the patient on his left side in bed with the

knees approximated to the chin so as to bend the back as far as possible. Draw a line across at the highest points of the iliac crests and remember that about half an inch below this line the disc between the 4th and 5th lumbar vertebrae (where the puncture should be made) is situated. Sterilise the skin and freeze if necessary with ethyl chloride. Introduce a steel or, if preferred, an iridium-pointed needle (which should be armed with a stillete) and of at least eight centimetres in length, just to the right of the middle line. Push the needle horizontally forwards and a little inwards and push through the ligamentum subflavum until the arachnoid sac is reached. It is often convenient to mount the needle on a syringe (not for purposes of suction but in order to render insertion easier). When the sac is punctured and the syringe removed, the fluid should drop fairly freely. 5 c.c. are sufficient for purposes of examination, although where the pressure is unduly high a larger quantity may sometimes be taken for therapeutic reasons. When the needle is withdrawn the wound is covered with collodion, and the patient should be kept in bed for the rest of the day so as to obviate any unpleasant consequences, of which the most common are headache, especially if too much fluid has been removed, and sciatic pain if the sciatic nerve roots have been pierced.

When examining the fluid, part of it should be centrifuged and some of the deposit placed on a slide and stained, and part of the fluid may be used for rough chemical analysis.

Normal cerebro-spinal fluid is clear like water, of low specific gravity, alkaline in reaction, and contains a small amount of serum globulin and a substance which reduces Fehling's Solution. There should be no cloud of albumin on boiling.

In pathological cases albumin is present in large amount in meningitis, and a distinct cloud may be obtained in most of the parasyphilitic affections. Sometimes blood is found in considerable amount in cases of haemorrhage. Where there is meningitis the fluid is opalescent, or may be purulent.

The cells found in *normal* cerebro-spinal fluid after centrifuging should be two or three lymphocytes and possibly a few large endothelial cells, in a single microscopic field (×550 diameters). *Pathologically*, a lymphocytic increase, which may number 100 to 200 or more, is found in cerebro-

spinal syphilis, in locomotor ataxia and general paralysis of the insane (parasyphilitic diseases), and in early cases of tubercular and some other types of meningitis. A polymorphonuclear leucocytosis is found in many cases of acute meningitis, sometimes in the later stages of tubercular meningitis, and in epidemic cerebro-spinal meningitis.

Lastly, a bacteriological examination is sometimes found to be of the greatest importance, and some of the fluid may be used for experimental inoculation.

(2) Brown-Séquard's Paralysis

If a lesion of the cord is unilateral, the following clinical features result:—Just immediately above the level of the lesion there is a zone of cutaneous hyperaesthesia limited to the side of the lesion. In the affected segment or segments there is, in the first place, unilateral anaesthesia, and, in the second place, motor paralysis with unilateral wasting of muscles and the reaction of degeneration—in other words, the phenomena met with in a lesion of the second motor neurone. These sensory and motor changes are limited to the side of the lesion.

Below the lesion and on the *same* side as the lesion there is motor paralysis, but without wasting of muscles or electrical changes, because the lesion involves the crossed pyramidal tract, and is therefore a lesion of the first motor neurone. The tendon reflexes, after being in abeyance, become markedly increased. The Babinski sign is positive (see page 583). There is impairment of muscular sense (by which is implied the sense of position of limbs and knowledge of movements of muscles), and often cutaneous hyperaesthesia and the temperature of the affected limb is raised.

On the *opposite* side and below the level of the lesion the muscular power and tendon reflexes are normal. There is cutaneous anaesthesia, at least for pain and temperature, but muscular sense is normal, and the temperature of the limb is not raised. A unilateral lesion is more commonly the result of trauma, such as a gunshot wound, or similar injury, than of any disease.

The phenomena above described are well shown in the following diagrammatic plan:

The Spinal Cord

Zone of Cutaneous Hyperæsthesia.		
Paralysis of muscles, with wasting and electrical changes. Abolition of reflexes. Zone of cutaneous anæsthesia.	Side of lesion.	Side unaffected.
Paralysis of muscles, but without wasting and electrical changes. Reflexes at first diminished, and later (in most cases) markedly increased. Babinski sign positive. Often cutaneous hyperæsthesia. Muscular sense impaired or lost. Temperature of skin raised.		No paralysis of muscles. No interference with reflex arcs. Babinski sign negative. Loss of pain and temperature sense. Tactile sensation sometimes impaired. No loss of muscular sense. No change in temperature of skin.

(3) Spastic Spinal Paralysis (Lateral Sclerosis)

This is clinically a well-defined disease. Many, indeed most, of the cases of so-called primary spastic paralysis may really belong to the wider group of disseminated sclerosis. Spastic paralysis follows most lesions of the pyramidal tracts, but the description here given is not intended to include the spastic changes which follow upon a hemiplegia, but only to cases in which the lesion is bilateral and apparently primary.

The **Etiology** is most unsatisfactory. Influenza or some toxin is probably the responsible agent, but in all cases the possibility of the spasticity being due to disseminated sclerosis, to pressure on the cord, to chronic myelitis or some other brain (such as hydrocephalus) or cord (such as amyotrophic lateral sclerosis) disease should not be lost sight of.

Pathological Anatomy.—There is sclerosis, and the sclerosis does not differ in any very obvious way from what is found in secondary degeneration. It is probably the result of a toxin whose action seems to be limited to certain groups of fibres. Unquestionably the legs are more affected than the

arms, and in certain cases the lesion extends to the trophic cells in the anterior horn of the cord (amyotrophic lateral sclerosis) or may even involve the second motor neurone; in other words, the anterior nerve roots and their extension into the peripheral nerves may also suffer.

Clinical Features. The chief symptom is weakness of the legs, which gradually increases, and in a matter of months interferes with the patient's ability to walk. When this occurs, he finds that he has inadequate control over movements which have now become spastic in nature. He describes his legs as being stiff and he tends to trip over any irregularities of the ground. The tendon reflexes are markedly exaggerated, and the Babinski sign is positive. In this test, on tickling the sole of the foot, the toes separate, and the great toe is extended in place of being flexed into the sole as in health. A lesion of the crossed pyramidal tract is always accompanied by a positive Babinski sign, but the converse does not always hold true. Ankle-clonus is almost invariably present, and there is commonly the clasp-knife rigidity, by which is meant that when the flexed leg is partially extended, the muscles complete the extension in a sudden and spasmodic fashion. Spasm of the adductor muscles of the thighs is not uncommon. There are no sensory changes, excepting sometimes pains in the joints. Rarely does primary optic atrophy develop, and there is no inco-ordination as a general rule.

Towards the final stages of the case, if a measure of quiescence is not established, there is a tendency to contracture of the affected limbs, while the organic reflexes may fail and cause much distress to the patient. Sometimes, though very rarely, the existence of a primary spinal lesion is suggested by the presence of a girdle sensation.

Diagnosis.—There is no difficulty in arriving at the diagnosis that the pyramidal tracts are affected, but it is by no means easy to prove that the lesion is primary. The absence of inco-ordination prevents confusion of this disease with *ataxic paraplegia*.

The **Prognosis** is unfavourable, although partial recovery, or at least a quiescent stage, may be reached, during which the patient remains for a long time in the same condition, and the duration of life is not infrequently prolonged for years.

Treatment.—Drugs are unsatisfactory. In every recent

case place the patient at absolute rest, and endeavour to keep up muscular nutrition by massage and electricity. Where there is even a suspicion of syphilis, administer potassium iodide and mercury. Sometimes sedatives are needful in order to relieve the spasmodic movements, and especially in cases where contracture has occurred.

(3) Acute Anterior Polio-Myelitis

(Infantile Paralysis—Atrophic Spinal Paralysis)

An acute or subacute inflammation of the anterior horn of the cord, the cell centre of the second motor neurone, but often selecting a limited area, or even a group or part of a group of multipolar cells. It is characterised by wasting of the muscles whose cell centres are involved (see p. 701).

Etiology.—Much difference of opinion has arisen as to whether the disease is infective, whether it is toxic, the toxin showing a peculiar selection of cells, or whether it may not be due to thrombosis or embolism of certain of the branches of the anterior spinal artery. It is common in childhood, and most generally during the 2nd and 3rd years of life. Boys were thought to suffer more frequently than girls, but this is doubtful, and there are many more cases in summer than in winter. There seems to be no doubt that definite epidemics have occurred, and if so, this favours the theory of an infective organism. The oft-repeated statement that a careless nurse letting the child fall was responsible for the illness, is probably inaccurate. The disease may attack children otherwise in perfect health, and it has occurred in adults, but in them it is by no means so common.

Pathological Anatomy.—The lesion consists of two definite changes: 1, a small cell proliferation chiefly round the small vessels in the grey matter of the anterior cornu, the cells resembling lymphocytes in appearance, and also a small cell proliferation round the multipolar cells, partly lymphocytic and partly neuroglial proliferation, and 2, atrophy of the multipolar cells with marked chromatolysis. A preliminary swelling of these cells may occur in slighter cases without palsy. In very acute cases haemorrhages may be present, and the lesion seems to resemble a myelitis localised chiefly to the anterior cornu. The anterior nerve roots in a marked

case become smaller, proportionate to the involvement of their cell centres, and there is descending degeneration in the anterior roots and the nerve fibres. The muscles supplied by these nerve fibres atrophy, and undergo fatty degeneration. The posterior horns may be similarly affected though to a less degree.

The enlargements of the cord, either cervical or lumbar, are the common sites of the foci of inflammation in the anterior horn. The disease may be unilateral or bilateral, sometimes both arm centres suffer more or less completely, sometimes an arm and a leg on the same side, or on different sides, and, as already indicated, the extent of inflammation is often limited to groups of muscles.

As development goes on, and should recovery not occur, the affected anterior nerve roots remain atrophied, and the corresponding half of the cord is seen to be smaller than its fellow. The cerebro-spinal fluid often shows a lymphocytosis.

Clinical Features.—Marked constitutional disturbance, with malaise, vomiting, fever, and sometimes pains in the back and legs, indicates the onset of the attack, which may be extremely sudden. When the acute symptoms have passed off, the paralysis is noticed. The paralysis is usually widespread to begin with, but the majority of the affected muscles or groups of muscles rapidly improve, whilst a limb or part of a limb remains paralysed. The muscles permanently affected rapidly waste, and the reaction of degeneration is readily obtained: where a limb remains paralysed, its growth and the development of its muscles are greatly interfered with, thus causing many familiar deformities, such as club foot, which result from the contracture and unresisted action of non-paralysed or less paralysed muscles. Pain, which may be referred to the part about to be paralysed, is sometimes an initial symptom: after the paralysis has developed, local tenderness of muscles and joints on handling or attempted movement may persist. It is probably of neuritic origin, but there are no other sensory changes. In very widespread cases all four limbs may be paralysed, and it is even possible to have paralysis of trunk muscles, the disease eventually proving fatal. As the lumbar enlargement is the centre for the organic reflexes governing the bladder and bowels, we occasionally find incontinence of urine and faeces.

In the arm there may be an upper-arm type of paralysis which includes the deltoid, biceps, brachialis anticus and supinator longus, or a lower-arm type of paralysis, which includes the extensors and flexors of the fingers and wrists, and occasionally the small muscles of the hand are specially affected.

In the legs the tibialis anticus and the extensor muscles are more generally affected than the hamstrings and gluteal muscles. The muscles of the face are rarely involved, unless the disease has attacked the nuclei of cranial nerves.

To the touch the affected muscles are flabby and soft, and the reaction of degeneration (page 579) should be obtained from 7 to 10 days after the attack commences, although in a favourable case, in which there is a prospect of recovery, only a partial reaction of degeneration is present. The superficial and deep reflexes of the affected limbs are generally absent. In cases where a whole limb is affected the growth of the limb is greatly interfered with, and a baby arm or a baby leg may result, often greatly handicapping the patient throughout life. There is no resultant change in the joints excepting the fact that bones may not be kept in their positions when the supporting muscles are completely paralysed, and subluxation sometimes occurs.

The **Diagnosis** presents no difficulty. The history of the case, with the age of the patient, the absence of sensory phenomena, except pain, and the presence of flaccid paralysis, and, lastly, the electrical reactions prevent any possible error. In *peripheral neuritis* pain is typical, and pain felt both in the muscles and nerves; in infantile paralysis pain may occur, although it is rare, but it does not persist for any length of time, and then peripheral neuritis is by no means a common disease in childhood. It is almost incredible that *hip-joint disease* could be mistaken for infantile paralysis.

Prognosis. There are very acute cases with extensive inflammation which are rapidly fatal, but these are rare. While it is hardly likely that even the smallest focus of inflammation can disappear without leaving some trace, a great diminution in the extent of the paralysis may be hoped for in most cases, and the electrical reactions should suggest a favourable prognosis, especially where an incomplete reaction of degeneration is present.

Treatment.—Give a purge and a fever mixture, should the case be seen in its early stages. Keep the patient absolutely at rest, and wrap the affected limb in cotton wool. It is unwise to apply fly-blisters or other severe counter-irritation to the region of the spine, because, although bed-sores are by no means common, no benefit has been found to follow this procedure. As soon as possible endeavour to keep up the nutrition of the affected muscles, and do this by massage and electricity. Where there is an incomplete reaction of degeneration, the affected muscles may be stimulated by the faradic current, and such treatment is most efficacious. Massage and electrical treatment should be kept up for months, and improvement may be hoped for during the whole of that time. In the later stages, when the chance of benefit from massage and electricity is lessened, endeavour to prevent deformity; much may be done by artificial supports for strengthening the limb, while transplantation of tendons, so as to make flexor muscles take the place of paralysed extensors, has yielded results which, if not brilliant, are at least extremely good.

There are few medicinal agents likely to be of much value; certainly strychnine in small doses is worth trying when the acute inflammatory stage in the cord has subsided; ergot and belladonna, amongst other remedies, have been administered with benefit in certain cases during the early stages. Too much, however, should not be expected from the use of any internal drug.

(5) Acute Ascending Paralysis (Landry's Paralysis)

This is a rapidly developing motor paralysis, travelling from below upwards, but without muscular atrophy or electrical changes and with little sensory alteration. It is usually fatal.

Etiology.—Cold and wet, syphilis, and possibly alcoholism, and such toxins as those of diphtheria and typhoid, have been considered as causes, but the evidence is not conclusive. Males suffer more than females, and it is generally a disease of adult life.

Pathological Anatomy.—Changes have been described in the grey matter of the anterior cornu, chromatolysis, etc., but

many authorities consider that no naked-eye lesion is present. A coccus has been found in the blood, and by some observers in the cerebro-spinal fluid, but in not a few cases bacteriological examination has been negative. Some believe that it is an ascending peripheral neuritis. It may be an affection, toxic in nature, of the whole lower motor neurones. The spleen is sometimes enlarged.

Clinical Features.—After some premonitory tingling, paralysis with loss of reflexes commences, travelling from the legs upwards. The arms suffer less completely than the legs, and with involvement of the other muscles, difficulty in respiration, deglutition, and speech are observed. The motor cranial nerves are sometimes involved. The affected muscles do not waste, nor show the reaction of degeneration, save in exceptional cases. The organic reflexes, sensation, and the trophic, vasomotor, and mental functions are normal as a rule. The spleen is frequently enlarged, and profuse sweating is common, but there is little or no pyrexia.

Diagnosis.—The advancing paralysis of muscles without pain aids in the diagnosis between this disease and *acute toxic polyneuritis*, but *acute ascending myelitis* and *acute poliomyelitis* must also be kept in mind. The almost complete absence of sensory changes in Landry's paralysis is an important diagnostic point.

Prognosis.—It rarely terminates in recovery, but generally after extension to the medulla death follows in 2 days to 2 weeks.

Treatment.—Hot baths may be tried at first, and also counter-irritation to the back. No drugs are of much value. Give ergot, strychnine, and perhaps salicylates, especially if the condition is due to cold and wet. Quinine and perchloride of iron may also be administered where there is any probability of the case being of septicaemic nature.

(6) Progressive Spinal Muscular Atrophy[1]

A lesion involving motor nerve cells in the anterior horn of the cord, probably the result of a toxin and characterised by progressive wasting, with fibrillary tremors in the muscles

[1] Compare Muscular Dystrophy, p. 806.

affected. It is a lesion, therefore, of the second motor neurone although it seems probable that the disease may spread upwards to the first motor neurone in certain cases.

Etiology.—It is commoner in males, and generally between the ages of 25 and 45. It is probably the result of overwork exposure, and worry, but in any case it must be due to the action of a toxic agent of some sort. A rare form of hereditary origin occurs in childhood.

Pathological Anatomy.—The ganglion cells in the anterior horn of the cord show the most marked changes. Certain of these become atrophied, and may even disappear, while the g[illegible] processes are less numerous, and the chromatic substance largely decreases in the affected cells. Increase of neuroglia takes the place of the cells which have become atrophied, and the anterior nerve roots in the affected segments of the cord are wasted, while the muscles supplied by the affected cells are pale and atrophied, and show indistinct striation, and often fatty, more rarely hyaline, degeneration.

In a special group of cases the pyramidal tracts are also involved, giving rise to spastic phenomena along with the typical wasting of certain groups of muscles. In not a few cases, before death, the disease affects nuclei in the medulla, and may therefore lead to the development of bulbar paralysis.

Clinical Features.—Wasting of certain muscles is the early [illegible] symptoms, and later gradual loss of power. Several types are described depending upon the muscles first and chiefly affected.

I. Duchenne-Aran Type.—The disease generally begins in the arms, the thenar and hypothenar eminences disappear, the interossei and lumbricales waste, and as a result of shortening of the extensors unresisted by the interossei, the griffin-like hand, called by Duchenne the *main en griffe*, is produced. Usually one hand is first affected, and the wasting extends to the flexors, followed by the extensors of the forearm; later the deltoid and biceps waste. Still later the lower two-thirds of the trapezius and certain of the scapular muscles become affected. The muscles of the trunk may follow: those of the neck and legs, if affected at all, generally succumbing at a late period. In all the muscles affected, fibrillary tremor or twitching is very common, there

is loss of electrical irritability, and there may be a partial reaction of degeneration. The tendon reflexes are gradually lost, but sensation is unaffected and the organic reflexes are normal.

II. The Peroneal Type. This is a less common form of progressive muscular atrophy, in which the peronei and anterior tibial muscles of the leg are first and most markedly affected. Double talipes varus may develop, and as the case progresses, the arms and the body generally become involved. Some neurologists deny the existence of this type of progressive muscular atrophy. Certainly many cases of peroneal muscular atrophy are really neuritic in origin, occurring in younger patients, and have a definite hereditary history.

Where, with wasting of the arm and shoulder muscles, the crossed pyramidal tract has also suffered, causing spastic paralysis of the leg, the condition has been termed *amyotrophic lateral sclerosis.*

Diagnosis. The history of the case, and the involvement of the interossei, the lumbricales, and the thenar and hypothenar eminences, are typical of the disease, and the absence of sensory phenomena prevents the possibility of mistaking progressive muscular atrophy for *syringomyelia.* In the *muscular dystrophies* there is a hereditary history and no fibrillary tremor and where the wasting is due to *cervical ribs* there are always sensory changes and the X-ray examination is conclusive. In *paralysis of the ulnar nerve* the whole of the interossei are not involved, and therefore it is easy to differentiate between the griffin-like hand of ulnar paralysis and this disease.

The **Prognosis** is unfavourable, although the disease may become arrested at any period.

The **Treatment** is most unsatisfactory: strychnine and arsenic, and certainly antisyphilitic remedies, should be tried. Probably strychnine is most efficacious when injected hypodermically into the affected muscles. Electricity, both galvanic and faradic, and massage are useful in preventing the rapid wasting of muscles. Attention should be paid to the feeding and general comfort of the patient, and as far as possible the deformity due to the action of muscles not antagonised, should be prevented.

(7) Amyotrophic Lateral Sclerosis

This consists in degeneration of the multipolar cells of the anterior cornua associated with sclerosis of the pyramidal tracts. It is a combination of progressive muscular atrophy with lateral sclerosis.

The **Etiology** is the same as that given for progressive muscular atrophy, and the pathological appearance of the anterior cornua also closely corresponds.

Clinical Features.—The atrophic symptoms specially involve the same muscles in the arms which suffer in progressive muscular atrophy, so that wasting of the small muscles of the hands, sometimes the muscles of the forearm, and usually at a later period the muscles of the shoulder, is associated with spastic paralysis of the legs. The atrophic changes and the spastic phenomena gradually increase, and muscular atrophy follows the spasticity in the lower limbs. By that time there is generally evidence of involvement of the nuclei in the medulla, in other words, bulbar paralysis supervenes.

There are marked fibrillary tremors in the wasting muscles. The tendon reflexes of the legs are exaggerated, ankle clonus and patellar clonus are present, the superficial abdominal reflexes are abolished, and the plantar response is extensor in type. The organic reflexes may suffer should the cells of the lumbar enlargement become affected, but this is not often the case.

The duration of life depends largely on the time which elapses before the bulbar phenomena appear, but 1 to 5 years may be considered the average duration of life.

(8) Locomotor Ataxia (Tabes Dorsalis)

A disease due to a degeneration of the posterior roots and part of the posterior columns of the cord; characterised by marked inco-ordination, loss of knee-jerks, shooting pains, difficulties with bladder and bowels, impairment of temperature, other sense impressions, and many other symptoms and signs indicative of degenerative change. There is a close relationship between tabes and general paralysis of the insane.

Etiology.—The disease is most common in males, and

during adult life. It is the result of syphilis, constituting one of the parasyphilitic affections, and is certainly due to inefficient treatment of the original syphilitic lesion. In all cases, whatever may be admitted by the patient with regard to a luetic history, lumbar puncture reveals the increase of lymphocytes so constantly present in the cerebro-spinal fluid in syphilis. Concussion of the spine, over-exertion, damp or cold, and alcoholic and other excesses have also been included as possible causes, and no doubt they may contribute somewhat to the production of the disease. In tabes in children hereditary syphilis is invariably present.

Pathological Anatomy and Pathology.—The posterior columns of the cord present the greatest changes. The sclerosis is most extensive in these columns in the lumbar region, and it diminishes as the cord is followed upwards. Above the lumbar enlargement the postero-external columns become gradually less and less affected, but the degree of involvement of those columns varies considerably in different cases. It is probable that the sclerosis commences in the posterior root zone in the lumbar region of the cord, and above that level of the cord the posterior roots are generally healthy. In cases where the arms are markedly ataxic, the sclerosis of the posterior roots is also present in the cervical enlargement of the cord, and associated with this is a profound sclerosis of the postero-external tracts generally throughout the entire length of the cord. The anterior part of the postero-external tracts, even in the lumbar region of the cord, usually escapes sclerosis. In the postero-internal tracts the fibres which enter these columns at different levels of the cord do so anteriorly, that is, next the commissure; and, therefore, in tracing the postero-internal tracts upwards in an ordinary case of locomotor ataxia, in which the lumbar enlargement presents the most marked sclerosis, the anterior part of these tracts is found to contain healthy fibres which have entered by healthy nerve roots.

A careful examination of the posterior cornua reveals that the sclerosis begins in the posterior nerve-root fibres, and that the tract of Lissauer almost invariably suffers, at least for a certain distance above the lumbar enlargement. It is probable that, as Marie states, the lesions of the spinal cord in locomotor ataxia occur by segments, and therefore

where degenerated nerve-root fibres cease to enter the cord tracts which are not continuous to the medulla will be found to be healthy. Following the posterior nerve-root fibres backwards, the ganglia may be found affected, although this is not always the case: sometimes the sclerosis has extended to the nerves.

Many other tracts and parts of the cord may suffer: sometimes the cells of Clarke's vesicular column are degenerated, and the direct cerebellar tracts which, according to many authorities, originate in these cells are often sclerosed. The comma-shaped tracts of Gowers, occasionally the pyramidal tracts, and sometimes the anterior cornual cells may also be involved.

In many cases there is chronic meningitis, and it has even been suggested that the disease may originate in connection with the pia mater, although this is unlikely.

Microscopically the nerve fibres in the degenerated tracts are markedly atrophied, and, as the name sclerosis implies, there is great increase of connective tissue.

Certain of the cranial nerves and special bundles of nerve fibres in the brain may undergo degeneration. In this way the optic nerves may be affected by a primary degeneration, and the 3rd, 4th, 6th, and sometimes other cranial nerves have been found involved. The sensory roots of the 5th nerve and the sympathetic fibres which supply the dilator pupillae muscle are often affected, particularly the latter. The Argyll-Robertson phenomenon (loss of the pupil contraction to light) depends on degeneration of the cells in the ciliary ganglia which supply the sphincter pupillae muscles.

In a small proportion of cases of the disease there is marked change in the bones and joints. The articular ends of some of the long bones, and especially the femur, undergo atrophy, while the joints swell up and there may be considerable disorganisation. New bone formation may develop around the joint much like the condition in osteoid arthritis, and fracture of long bones may occur owing to their excessive brittleness. In the cerebro-spinal fluid there is invariably increase of lymphocytes.

Probably the syphilitic lesion is the necessary element in every case of tabes, and the excess, accident or whatever it may

be, starts the degenerative change in the previously weakened neurones.

Clinical Features.—There are three more or less distinct stages of the disease, which seem more generally accurate than the classic *Pre-ataxic*, *Ataxic*, and *Paralytic* stages. It should, however, be stated that ataxia may appear very early and be very marked, and that no order or sequence can be dogmatically laid down for the chief clinical features and the stages in which they may occur.

Stage 1.—Commencing incoordination, numbness of the skin of the feet, shooting pains, especially in the legs and round the trunk in the line of the spinal nerves, loss of knee-jerks, the presence of the Argyll-Robertson phenomenon, pupil reacting to accommodation, but not to light, sometimes transient paralysis of an eye muscle, and sometimes incontinence of urine.

Stage 2.—In this stage there is marked ataxic gait, increased pains, myosis, contracted pupils, impotence, girdle sensation, sometimes deafness, and occasionally curious crises—laryngeal, gastric, vesical, and so forth.

Stage 3.—Ataxia and anæsthesia become still more marked, there is often ophthalmoplegia, and as time goes on the organic reflexes may give way, the patient becoming increasingly helpless and burdensome both to himself and to those in attendance on him.

Where trophic changes in the joints begin in the first, Charcot's disease results in the third stage.

The following is a brief description of the typical phenomena noted under their appropriate headings:—

1. *Sensory Phenomena.*—The *shooting* or *lightning* pains are very characteristic. They come on at night, occur in the legs and sometimes the trunk, are apt to be paroxysmal, and may last for hours and even days in one position, and may then change to some other part of the body. Boring pains, deeper-seated, may be referred to bones, joints, or muscles. Not infrequently the pains are modified by the weather, becoming worse during cold and damp, while with fatigue or indisposition their severity increases.

The *girdle sensation* of locomotor ataxia is a feeling of constriction resembling that produced by a tight belt, and its level is probably associated with that of the diseased segments.

Paraesthesia or abnormal sensations of different kinds are common, such as burning, pricking, numbness, and formication; they are apt to be felt in the feet, and less frequently in the hands. There is *anaesthesia* over areas in the feet and legs, and generally, even when sensations are appreciated, their conduction is delayed. In a few cases *polyaesthesia* is present, by which is meant that one touch is felt by the patient as many, and also *allocheiria*, which signifies that a touch on one side of the body may be referred to the same position on the opposite side.

In some cases, so insensible does the skin become, that a perforating ulcer on the sole of the foot occasions no pain to the patient, and the muscles and bones may be equally anaesthetic. There is loss of the sense of pain when the calf muscles are squeezed, temperature sense and sense of pain in the skin of the legs are much less acute than the normal, and often much delayed, while tactile sensation may be remarkably accurate.

There is marked loss of the *sense of position*. This is well tested by making the patient, when blindfolded, find with the hand his own foot, which may be brought within reach. He generally displays absolute ignorance of its position. It is noted that the distal parts show this loss of position-sense more markedly and earlier than the proximal.

(2) *Reflexes.*—The knee-jerks are always abolished, and in most cases the other important muscle reflexes in the legs are found to be absent or very feeble, the order of disappearance depending on the level of origin of the lesion in the posterior nerve roots. Where the arms are affected, the wrist-jerk, biceps-jerk, and other tendon reflexes will also be abolished.

The skin reflexes of the legs are naturally lessened in any case owing to the degree of anaesthesia usually present.

(3) *Motor Symptoms.*—The leading phenomenon is *ataxia* or *inco-ordination*, and this is in most cases (where the cervical enlargement of the cord is not involved) limited to the legs. Romberg's phenomenon is well marked, and may, in fact, be the first indication to the patient that something is wrong. When he stands, with heels together and eyes shut, he sways and tends to fall, and when washing in the morning and soaping the face he may reel against the wall or knock over the basin. Romberg noted that by this test the inco-ordination, which

develops gradually, is easily recognised. The gait is soon modified, mainly as the result of the ataxia. The alteration becomes more observable when the patient endeavours to walk without boots or shoes, and it is seen that he separates the legs so as to give himself a wider base of support. He raises his feet too high, he brings them down, heel first, with a thump on the floor, and it is found that when an angled chalk-line is drawn on the floor he cannot, with the great toe, trace it accurately. Notwithstanding these changes there is no muscular weakness, as a general rule, and the power of the patient may be unaffected.

In cases where the cervical part of the cord is involved, the arms also become ineo-ordinate, and in rare cases they may show ineo-ordination prior to its development in the legs. The ineo-ordination in the arms may be well demonstrated by making the patient, with eyes closed, endeavour to touch the tip of the nose with the forefinger, or to bring the tips of the fingers of the two hands into apposition.

There is marked loss of tone in the muscles of the affected limbs even if there be no great wasting, and the ligaments of joints may also participate in this. As a result hypotonia or over extension of joints develops, the knee-joints showing this clinical feature often very markedly.

(4) *Special Senses.* The *Argyll-Robertson phenomenon*, by which is meant the loss of pupil reaction (contraction of pupil when the eyes are exposed to the light after being closed), is present in five out of every six cases of the disease, if not in an even larger proportion. The phenomenon depends not on degeneration of Meynert's fibres, which join the anterior corpus quadrigeminum to that part of the nucleus of the 3rd nerve governing the sphincter pupillae muscle, but to degeneration of the cells in the ciliary ganglia which energise the sphincter pupillae muscle. It is also found that pinching the skin of the neck no longer dilates the pupil, and that, owing to the involvement of the sympathetic fibres in the cord which supply the dilator pupillae, there is generally persistent contraction of the pupil, termed *spinal myosis*. The two pupils are often unequal, a common feature of cerebro-spinal syphilis.

Primary atrophy of the optic nerve is certainly not common, and probably is not present in more than 1 out of 10 cases. It frequently begins during the first stage, and with the advent

of complete blindness there is often an apparent arrest of the disease in other directions. Certain of the *external ocular muscles* may be temporarily *paralysed*, and of these the levator palpebrae generally suffers, causing ptosis, while sometimes the ciliary muscle may also be affected. In later stages of the disease a permanent paralysis of one or other of these oculomotor muscles may develop. *Deafness*, together with *loss of the sense of smell* (anosmia), is sometimes present. Associated with the laryngeal type of crisis there may be a degree of paralysis of the abductor muscles of the glottis.

5 *Sphincters.* The bladder gives trouble at an early period in the disease. There may be *incontinence of urine*, sometimes retention, sometimes mere sluggish action, and owing to the cutaneous anaesthesia the patient may be ignorant of nature's calls. In some cases cystitis ensues during the later stages of the disease. The bowels may give similar trouble, the sphincter becoming weak, and should diarrhoea be present, much trouble is caused to the patient from *incontinence of faeces*. Fortunately constipation is common, and it is remarkable in how many cases, after control of the sphincter has been lost, it may be temporarily or permanently regained.

6 *Vasomotor and Trophic Functions.*—There is sometimes *local sweating*, sometimes alteration in the distribution of pigment in the skin and hair. The *perforating ulcer* of the foot has already been mentioned; it usually originates in a corn on the sole of the foot. The teeth may drop out, the bones, especially the long bones, may become brittle and the articular ends may be partially absorbed, while the joint changes, which perhaps most commonly affect the knees and which go by the name of *Charcot's joint disease*, develop during the third stage of locomotor ataxia. The resulting disorganisation of the joint often renders any hope of treatment absolutely futile, and even in the best conditions there may be new bone thrown out, so that ankylosis and deformity inevitably ensue.

7 *The Crises* are not common; in the *gastric* variety there is severe pain in the stomach, accompanied by vomiting of watery fluid which may contain bile or blood, and often by severe hiccough. The attack generally lasts for some hours. *Rectal* crises imply terrible tenesmus, and *urethral* crises a similar pain at the neck of the bladder. *Laryngeal* crises

are simply laryngeal spasms, which may give trouble and frighten the patient by the feeling of impending suffocation.

Complications.—It is only necessary to glance at the pathological anatomy in order to realise the many complications of the nervous system which may ensue, due to the spread of the sclerosis or to a myelitis being set up in some part of the cord. Myelitis, acute or chronic, sclerosis of the lateral columns of the cord progressive muscular atrophy, and general paralysis of the insane, which is etiologically closely allied to tabes, give some indication of complications which may render the diagnosis difficult.

It is also probable that other systems may be affected, and cardiac disease is not uncommon in cases of locomotor ataxia.

Diagnosis.—A typical case is easy, and the absence of knee-jerks, the presence of the Argyll-Robertson phenomenon, the ataxia and the pains, present a clinical picture different from any other cord lesion. Attention is directed to the differential diagnosis of *Friedreich's ataxia* and *ataxia aphasia* (see pp. 682 and 684), while in *cerebellar tumour* the presence of optic neuritis is practically constant.

Prognosis.—At an early stage in the disease there is hope of improvement from anti-syphilitic treatment. As already stated, the presence of optic atrophy, with the inevitable blindness to which it dooms the patient, is generally believed to precede the arrest of the disease otherwise.

The first stage may last for twenty years, or the disease may progress with much greater rapidity, at times becoming stationary and again advancing. Complications not infrequently are the cause of death, and should the patient have bed-ribbon, bed-sores are to be dreaded, and bladder troubles may result in kidney disease.

Treatment.—Invariably try anti-syphilitic remedies carefully and patiently: of these potassium iodide will probably be most lement, although mercurial inunction should be associated with it. Forbid all mental and bodily fatigue, and bear in mind that a period of rest in bed is often of great value. In mild cases recommend a sea-voyage, and regulate the patient's life, and especially the amount of alcohol and tobacco which he should use. Caution the patient against sexual excess during the early stages of the disease. In the treatment of symptoms remember that in certain cases nothing

affords so much relief for the pains as electricity, sometimes galvanism and sometimes high-frequency, while hot baths and a change to a warmer and drier climate are helpful. In some cases it is necessary to give hypodermic injections of cocaine or morphia. For the crises the nitrite group, and especially nitrite of amyl and nitro-glycerine, afford great relief. Resection of the 7th, 8th, 9th, and 10th dorsal nerve roots has been performed successfully for the arrest of the gastric crises. The operation is a serious one, but the gastric pains may be so severe as to warrant the risk being taken. Antipyrine, phenacetin, and kindred remedies are beneficial in arresting the pains of tabes.

For the disease itself there is no remedy which can honestly be called specific unless it be salvarsan (see page 124), from which much benefit has been anticipated. Arsenic, silver nitrate, quinine, strychnine, phosphorus, Calabar bean, or belladonna, and many other drugs have been administered but they are all unsatisfactory. Pay special attention to the bladder and never permit retention to occur, and guard very carefully by antiseptic precautions against the risk of inducing cystitis by passing a dirty catheter. It is wise also to warn the patient of the possibility of a perforating ulcer of the foot and with this object in view recommend attention to any corn or injury, and prevent the patient from attempting to cut or pare corns himself.

Two other methods of treatment demand a word: one is the outcome of a suggestion made some years ago to treat locomotor ataxia by stretching the spinal column, and with it the spinal cord. This has been done either by suspending the patient practically by his head, or with the patient lying on a couch, by bending the back in a bow-shaped fashion to bring the knees into as close contact with the face as the operator may consider advisable. The treatment has not yielded good results, and is certainly not free from risk.

It has, secondly, been found possible to do something for the cure of the ataxia by re-educating the patient to perform movements in a co-ordinate way, and much may be accomplished by training him to walk along a straight line with the artificial help of a go-carriage. It is to Frenkel that the credit of this re-education plan of treatment is due.

9) Friedreich's Ataxia—Hereditary Ataxia

A combined sclerosis of the spinal cord, involving both the posterior and lateral columns, and commencing at an early age. Professor Friedreich of Heidelberg first described the disease in 1861.

Etiology.—As the name implies, the disease is generally hereditary, several members of a family being, as a rule, affected, but direct transmission is rare. Alcoholism, syphilis, and consanguinity have been traced in certain cases in the parents, but the percentage is not convincing. The disease begins in early life, generally between the ages of 7 and 15, and very rarely after the age of 25. Males suffer more frequently than females.

Pathological Anatomy.—The sclerosis affects the posterior columns very markedly, especially the postero-internal tracts and to a less extent the lateral and anterior columns. The posterior columns are extensively sclerosed throughout the entire cord, while the sclerosis only involves the periphery of the lateral and anterior columns, extending forward almost to reach the anterior commissure. This implies the involvement of the direct cerebellar tract and associated with it, atrophy of Clarke's vesicular column, and the antero-lateral tract of Gowers, while the pyramidal tracts are less extensively sclerosed. The sclerosis is symmetrical, the posterior root zones largely escape, and the dorsal region of the cord is often the portion most affected.

The pathology of the condition is by no means clear. It seems most probable that it is an embryonic error in development owing to which the tracts affected cannot resist the strain of life. It is, however, difficult to assert that it is an early atrophy of these affected tracts. The microscopic appearance is that of proliferation of connective tissue.

Clinical Features.—As indicated under etiology the disease usually commences between the ages of 7 and 15. The ataxia begins in the legs, but soon also develops in the arms. The gait is peculiarly modified, somewhat resembling, in its staggering irregular movements, the walk of a drunken man. The movements of the arms, instead of being merely ataxic, have a choreiform and swaying character, and later there is unsteadiness of the head and trunk. Romberg's phenomenon

is well marked, the knee-jerks are absent, and the plantar reflex is extensor in type. The speech is peculiar, beginning somewhat slow, and suggesting sluggish cerebration, while nystagmus, and very exceptionally the Argyll-Robertson phenomenon, are present. There is often vertigo, but primary atrophy of the optic nerve does not occur. Sensory changes, trophic changes in the skin and joints, and incontinence of urine, are generally absent. As time goes on, certain deformities develop; the first of these is *pes cavus*, in which the foot appears to be shortened, the arch markedly high, the heel drawn up, and the toes dropped. The toes are hyperextended at the metatarso-phalangeal joints and the great toe is specially overextended. The patient may later tend to walk on the outer side of the foot. Lateral curvature of the spine is common.

Diagnosis.—The fact that it is a family disease, inasmuch as several members of the same generation are usually affected, is a great help in diagnosis, while the ataxia of the arms, and the peculiar choreiform or jerky movements of both arms and legs, the affected speech, well-marked nystagmus, and the absence of the Argyll-Robertson phenomenon prevent any confusion with *locomotor ataxia*. In addition the age of the patient is practically always under 25, while in locomotor ataxia except in the rare juvenile type, or in *ataxic paraplegia* the age is generally over 40. The gait may suggest *cerebellar tumour*, but optic neuritis is absent, and the other characteristic features of Friedreich's ataxia should prevent a mistake being made.

Prognosis.—The disease may last for many years with gradual advance, and is generally fatal by some intercurrent affection. Unfortunately there is a tendency to various deformities and contractures, as already noted, and these may cripple the patient and much interfere with his comfort.

The **Treatment** is practically *nil*. Attention to the general health is of course desirable, Frenkel's exercises may be tried, and much may be done to obviate deformity and contracture by careful massage, and in the later stages by division of tendons.

Marie described under the title "Hereditary Cerebellar Ataxia" a group of conditions in which there are the reeling staggering gait so characteristic of cerebellar lesions

2, a peculiar tremor or almost ataxic movement of the arms; and 3, a tottering speech.

Such lesions may be sub-divided into groups most of which are hereditary, but the cases referred to are so rare that further description is unnecessary. In one group, Sanger Brown has described a degeneration of the spino-cerebellar tracts, but although the direct cerebellar and the antero-lateral tract of Gowers usually suffer, the posterior columns do not escape.

10. Ataxic Paraplegia of Gowers.

This is a combination of sclerosis of the posterior and lateral columns developing in later life, and so is distinct from Friedreich's ataxia, while there is less extensive involvement of the posterior columns than in that disease.

Etiology.—It is a disease of males, the ages being generally from 30 to 40, although females are not exempt. The cause of the condition is doubtful; syphilis, over-work, cold, injury to the spine, and sexual excesses have all been considered as possible predisposing or exciting causes.

Pathological Anatomy. In the posterior columns the sclerosis is most marked in the dorsal region of the cord, while it is less intense in the lumbar region; the posterior root zone, which is so markedly affected in locomotor ataxia, escapes. In the lateral columns, the crossed pyramidal tracts, Gowers' tract, the direct cerebellar tract and the lateral limiting layer will be involved, and are more sclerosed in the dorsal than in the lumbar region of the cord. The meninges of the cord do not appear to be affected.

Clinical Features.—It is a slowly progressive disease, patient complaining, to begin with, merely of languor. The history suggests ordinary spastic paralysis. The legs go first, and Romberg's phenomenon is marked; but the knee-jerks are exaggerated and ankle clonus is present. Hence the root zones in the lumbar enlargement have escaped. There are no pains, there is no girdle sensation, and there is not the typical heel-thumping gait of locomotor ataxia. The arms may be ataxic, but there is usually no Argyll-Robertson phenomenon, and rarely optic atrophy. There may be slight difficulty in speaking, with tremulous

movements of the face, but these symptoms are not alw present. The Babinski sign is positive

As the disease advances, the patient becomes bed-ridd the condition closely resembling the later stages of a se case of spastic paralysis, and the organic reflexes may give wa

Diagnosis. The association of ataxic and spastic phen mena marks the disease out sharply from *tabes*, while fr *Friedreich's ataxia* it is equally clearly differentiated by t age of the patient, the exaggerated knee-jerks, the absen nystagmus and of the typical movements superadded to ataxic of the arms and legs which are present in Friedreich ataxia.

Prognosis.—The disease may last for years, and it is r arrested. Death is apt to be due to some intercurrent affect and may very probably be associated with the bladder

The **Treatment** is that appropriate for spastic paraly

(H) Myelitis

The term Myelitis has been applied to various affect *inflammatory* and *degenerative*, to which the spinal co liable. It should be limited to *inflammatory* proc although thrombotic conditions due to blocking of s blood-vessels, and generally the result of syphilis, and cases in which there is pressure on the cord by t vertebral caries or meningeal affections, are often incl Syphilitic myelitis and so-called pressure myelitis are des separately.

1. Infective Myelitis. **Etiology.** A rare di because, although many parts of the body become inf due to the presence of infective organisms, the spinal comparatively seldom suffers. It sometimes follows as plication of an infective disease such as small-pox, ty dysentery, gonorrhœa, tonsillitis, cystitis, and tuber Infective myelitis occurs in both sexes and at all although young adults provide the largest number of v Streptococci, staphylococci, pneumococci, and many organisms have been found in different cases.

Pathological Anatomy. The pathological appearan the same whether the myelitis is transverse, diffuse disseminated. The affected part of the cord is soften

oedematous, markedly hyperaemic and often contain extravasations of blood. The neighbouring meninges show congestion of blood vessels and not infrequently there is a purulent exudate. There is generally a great invasion of cells in the neighbourhood of the vessels, and the neuroglial cells round the area of myelitis proliferate. Both white and grey matter of the cord are involved, the posterior columns often presenting the most intense changes. As a result of the inflammation, the axis-cylinders break up and the nerve cells undergo first chromatolysis and later vacuolation and destruction. The myelin sheaths also break up and the affected areas of white matter cease to stain by the Weigert-Pal method. The Marchi method demonstrates the presence of fat throughout the area of myelitis. In time, sclerosis replaces the diseased tissue, and ascending and descending degenerations necessarily follow.

The **Clinical Features** depend on the site and extent of the myelitis, but in every case there are constitutional symptoms such as pyrexia, loss of appetite, and often vomiting, while pain in the back, and tingling or numbness in the lower limbs often precede the paralysis. The paralysis may come on rapidly or may be delayed; frequently retention of urine precedes any marked loss of power in the limbs.

Acute Transverse Myelitis is the most common variety and implies the partial or complete destruction of important structures in one or more segments of the cord, and the clinical features vary, depending on whether the lesion is situated in the cervical, dorsal, or lumbar region.

Above the level of the lesion there is a zone of hyperaesthesia.

At the level of the lesion there is 1. flaccid motor paralysis of the muscles supplied by the anterior horn cells; 2. abolition of all the reflexes, superficial and deep, belonging to the affected segments; and 3. loss of all kinds of sensation in the area of skin belonging to the affected segments.

Below the level of the lesion, and if the lesion is *complete*, there is (1) flaccid motor paralysis; (2) loss of superficial and deep reflexes; (3) loss of all forms of sensation; and 4. loss of control over bladder and bowels.

Where the lesion is *not complete* although the patient may have lost the power of voluntary motion, spastic paraly-

in time replaces flaccidity, and we get great exaggeration of the tendon reflexes below the level of the lesion, marked ankle and patellar clonus, abolition of the epigastric and abdominal reflexes, and plantar extension. There may be loss of control over the sphincters, and not infrequently involuntary spasmodic contractions of the muscles; while increasing contracture adds much to the discomfort of the patient. Trophic changes often lead to the formation of bed-sores over any points of pressure and specially over the sacrum, great trochanters, and heels.

Cervical Myelitis.—It rarely occurs above the cervical enlargement, but when it does, all four limbs are paralysed and death is apt to ensue, owing to paralysis of the respiratory muscles.

It is more usual to find the cervical enlargement involved, in which case all four limbs are paralysed, and very generally the oculo-pupillary fibres of the sympathetic cause spinal myosis and narrowing of the palpebral fissures, with pseudo-ptosis due to sinking in of the eyeballs.

Dorsal Myelitis.—Here the arms are not affected, and it is often easy, by locating the zone of hyperaesthesia above the upper limit of the lesion, to define its position in the spinal cord. As regards the bladder, there is first retention, and later incontinence.

Lumbar Myelitis.—Here the legs specially suffer, and it is not uncommon to find that the paralysis remains largely of flaccid type owing to the fact that the segments of the cord may be involved which contain the multipolar cells supplying the leg muscles. The fact that the centres for bladder and bowels are also situated in the lumbo-sacral region renders the prognosis, as regards even partial recovery and prolongation of life, by no means bright.

Prognosis. Absolute recovery in infective cases is rare, but in incomplete lesions there may be improvement as regards sensory conduction; and the development of spastic paralysis and in the most hopeful of cases a certain return of voluntary power are the best that can be expected.

The **Diagnosis** is not as a rule difficult, although it may be impossible to determine the exact cause of the condition.

Treatment. Rest of the most absolute description is imperative, and a water-bed should be obtained without delay

The application of ice to the back, mustard to the skin, and hot baths are useful methods of treatment, but only under special conditions. An initial hot bath in an acute case is certainly most beneficial. Ice used with care generally does no harm, but counter-irritation such as by mustard or blisters of any kind should never be carried out, without carefully considering the risk of damage to skin whose nutrition may be much below normal. It is well in acute cases to make the patient lie on his face for a time, because by this means the cord is kept as high as possible. The greatest care should be taken of the skin; the parts pressed upon should be washed with soap and water, and then with methylated spirits, and after being carefully dried a dusting powder should be applied, while pressure should be taken off by the use of air-pillows, nests of cotton wool, and similar devices. The bladder should be always remembered, and an aseptic catheter used for drawing off the urine at suitable intervals, while urotropine (gr. 5-10) aids in preventing decomposition occurring in the urine. Two drugs are of great advantage in many cases: these are potassium iodide and mercury, and with a specific history they may be pushed with much benefit to the patient. Ergot is of advantage where haemorrhage is occurring. Massage is sometimes useful, and also galvanism and faradism to keep up the nutrition of affected muscles, but on no consideration should the patient be disturbed during the acute stage of the disease. It is hardly necessary to add that the bowels should be kept freely open, and that the diet should consist largely of milk.

II. Acute Ascending Myelitis or Acute Disseminated Myelitis is the name employed where there are scattered patches of myelitis throughout the cord, scattered both as regards their presence in white and grey matter and also as regards their level. The patches closely resemble the description given under transverse myelitis.

The **Clinical Features** are distinctive because the disease commences suddenly, and after some feeling of numbness or constriction round chest or abdomen there is a rapid onset of motor and sensory paralysis.

There may at first be exaggeration of deep reflexes together with plantar extension, but later flaccid paralysis with complete loss of all deep and superficial reflexes develops, and

not infrequently, as the name implies, the paralysis *ascends* the cord, gradually including more and more of the segments from day to day. The bladder and bowels suffer just as in the other form of myelitis, and bed-sores are extremely common.

The **Prognosis** is very grave, and **Treatment** is unfortunately of little avail.

III. Syphilitic Myelitis.—It has been abundantly proved that syphilis is responsible for a very large proportion of all cases of myelitis, and it generally develops a few years after the primary infection.

The dorsal region of the cord appears to be the seat of election in place of the cervical and lumbar enlargements which are so frequently involved in infective myelitis. It is not improbable that exposure to cold, fatigue, or injury may act as exciting causes along with the syphilitic infection.

Pathological Anatomy.—The meninges invariably suffer along with the cord, and generally the dura mater is adherent to the other membranes. In the affected area of the cord, the blood-vessels are diseased, the intima being much thickened so as almost to occlude the lumen of the vessel. There are also changes in the other coats and often a small cell infiltration round about the vessels. Where there is actual thrombosis the neuroglial cells share with the nerve tissues in the general necrosis, but where the circulation is not completely occluded there may be proliferation of neuroglial cells. Nerve fibres and nerve cells undergo degenerative changes, axis-cylinders and myelin sheaths breaking up and chromatolysis of the cells occurring. The meninges in the neighbourhood of the affected part of the cord show a well-marked small cell infiltration. Ascending and descending degeneration are seen above and below the site of disease.

Clinical Features.—After a period during which the patient is out of health he begins to feel his legs heavy and weak, and he may have some bladder difficulties. Numbness comes on from the site of the lesion downwards, and paralysis of the legs gradually develops. The rapidity of the process varies, and although there is little actual inflammation or myelitis there may be marked interference with nerve tracts. Complete paralysis, motor and sensory, may develop within a few days. As already indicated, the bladder and bowels suffer: at first there is retention and later incontinence.

There is generally an increasing degree of spasticity of the leg muscles with ankle clonus and plantar extension indicating that the lesion is an incomplete one. Involuntary spasms of the leg muscles are often very troublesome, and increasing tendency to contracture soon follows.

An examination of the cerebro-spinal fluid obtained by lumbar puncture shows a great excess of lymphocytes, and the fluid becomes somewhat turbid on boiling. The Wassermann reaction is positive.

Prognosis.—These cases constitute the most hopeful forms of myelitis. Under proper antisyphilitic treatment remarkable benefit may be obtained.

(2) Pressure Paralysis or Compression Myelitis

Gradually increasing pressure on the spinal cord, resulting in a corresponding interference with functions.

Etiology.—It is generally the result of tubercular caries of the spine, which causes spinal curvature, and is commonly called Pott's disease. Pott's disease most commonly affects either the mid-dorsal or the cervical regions of the cord. No age is exempt, although the disease usually manifests itself in childhood or youth: probably in not a few cases an injury bears a definite relationship to the commencement of the tubercular process in the vertebral bodies.

The other lesions which may give rise to pressure are tumours of meninges or vertebrae, injury, aneurism, and more rarely the presence of hydatid cysts.

Pathological Anatomy.—There is often very marked angling of the spinal canal from collapse of several bodies of contiguous vertebrae, and, in addition, there may be extra-dural pressure on the spinal cord from tubercular débris. Sometimes meningitis is set up, with or without inflammation of the cord itself.

Clinical Features.—One or two vertebral spines are generally found to be much more prominent than the others, and tenderness may be felt on pressure. The deformity is gradual in development, and is often associated with pain radiating along the lines of spinal nerves, and due to involvement of the nerve roots. One of the first manifestations of compression of the cord is the development of spasticity in the

legs, due to descending degeneration in the pyramidal tracts. This gradually increases until paralysis occurs, but it is a paralysis of the first motor neurone or trophic realm, and therefore unaccompanied by rapid wasting or altered electrical reactions. The superficial abdominal reflexes are absent, and there is plantar extension (positive Babinski sign). In cases in which myelitis has been lighted up, its phenomena will be superadded, although it is not always easy to recognise the onset.

Where the lesion is in the *cervical* region of the cord, a retro-pharyngeal abscess is occasionally present, and difficulty may be experienced in moving the head, while sometimes evidence of involvement of the first dorsal nerve may be made out by the narrowing of the palpebral fissure, sinking in of the eyeball, and alteration in size of the pupil.

If the lesion is in the *dorsal* region of the cord, the curvature is often well marked, and the girdle pains may be very suggestive.

In the *lumbar* region of the cord the great organic reflex centres controlling bladder and bowels are apt to be involved. A psoas abscess may develop.

Considerable assistance may be afforded not merely by the site of shooting pains, but also by numbness or tingling in areas belonging to affected nerve roots.[1]

Diagnosis.—An examination of the back is often sufficient with percussion of the vertebral spines. The hot-sponge test is frequently of service in recognising a localised myelitis, and care should be taken to examine for the presence of tumours of breast or other region, or of tubercular manifestations in the chest or elsewhere.

Prognosis and Treatment.—The treatment of tubercular caries of the spine is essentially surgical. There are many cases in which, after a prolonged period of rest in bed, often with the application of splints and other devices to remove pressure from, and to keep at rest, the affected vertebrae, a process of cure results, which, if it does not imply the complete elimination of the tubercle bacillus, at all events permits of the patient leading a useful and often prolonged life with little physical discomfort. It is important, in cases of cervical

[1] Pressure on nerve roots may cause intensely painful areas of skin, which are nevertheless anaesthetic to tactile sensibility and common sensation. This is called *anaesthesia dolorosa*.

caries, to prevent the rotation of the head, and therefore sand-bags have been used with considerable benefit during the period of rest in bed, or a suspensory apparatus by means of which the weight of the head is supported, and rotatory movements largely prevented. Poroplastic jackets are often worn in cases of vertebral caries occurring at a lower level.

In very early cases operative treatment is not necessary, and rest is alone sufficient; but in cases in which there is marked angular curvature with, in addition, pressure of tuberculous débris on the outside of the dura mater, operation by laminectomy, provided the lesion is sufficiently localised and the patient fit for the operation, may alone yield any hope of cure. The spastic paralysis may gradually disappear, and recovery follow. The ordinary open-air, good food, and tonic treatment for tubercular subjects should be carried out in cases of tuberculosis of the vertebrae. It should be added that tuberculin might be considered as a possible method of treatment.

13. Haemorrhage into the Spinal Membranes (Haematorrhachis). Haemorrhage into the Spinal Cord (Haematomyelia)

1. Haemorrhage into the Spinal Membranes.—This may occur either outside the dura or inside between the membranes and the spinal cord. *Extra-meningeal haemorrhage* may be the result of injury, and the haemorrhage may press severely upon the dura and the spinal cord within. An aneurism of the third part of the arch or of the descending aorta has been known to erode the vertebral bodies and to rupture into the spinal canal, causing death by the great pressure which it produced.

Intra-meningeal haemorrhage is much more frequent, and may be due to blood passing down from the base of the brain. Such haemorrhage may originate in the ventricles from rupture of an ordinary intracerebral haemorrhage into the neighbouring lateral ventricle, or an aneurism, sometimes involving the circle of Willis, may rupture; while from fracture or other injury, severe haemorrhage, either subarachnoid or subdural, may be caused. In all of these cases the blood may flow downwards into the spinal canal. In death from tetanus, strychnine poisoning, or from a severe convulsive seizure, submeningeal haemorrhage may

occur. It is common to find in cases which may be classified as toxic, and especially in the infective fevers of malignant type, that numerous small haemorrhages are present, sometimes under the pia and sometimes subarachnoid in position, and these may be found in connection with both brain and cord.

The **Clinical Features** dependent on the meningeal haemorrhage vary considerably in different cases. There may be sharp and severe pain, with evidence of irritation of spinal nerve roots; in other cases the pressure is sufficient to induce paralysis, and the organic reflexes may be completely abolished. There is sometimes muscular spasm, sometimes complete paralysis, and in not a few cases there may be absolute coma.

A special form of internal pachymeningitis associated with haemorrhages not infrequently affects the cervical meninges. It is described under meningitis (p. 652).

11. Haemorrhage into the Spinal Cord.—This is a rare condition, although more common in males, and appears to be most frequently due to injury, such as a bullet wound or a severe blow, or even forcible bending of the neck. It has been described in women as occurring in labour, and in both sexes it may be associated with alcoholism, tetanus, strychnine poisoning, and severe convulsions. It seems likely that in a number of cases the haemorrhage is in reality secondary to, and occurring in, a glioma of the cord, and it is hardly necessary to add that in myelitis small haemorrhages are common.

Pathological Anatomy. — The haemorrhage varies in position and in extent; it is often situated in the grey matter of the cervical or lumbar enlargements; it may rupture through the cord, it may tear it up transversely, or it may burrow vertically, and especially is this vertical burrowing apt to occur in connection with glioma tous tumours.

The **Clinical Features** vary considerably. The onset is much more sudden than in meningeal haemorrhage, and the paralysis, sensory and motor, may involve the legs alone, or the arms and legs, according to the position of the lesion. If the haemorrhage is primary, a myelitis almost invariably follows, and death, if not due to the haemorrhage itself, is generally not long delayed. In slight cases the blood may be absorbed, and partial recovery results, generally with spastic gait, exaggerated tendon responses, and plantar extension of

the great toe. Spasmodic contractions of muscles are often troublesome.

The **Diagnosis** is usually easily made by the suddenness of onset and the rapid trophic changes which generally ensue. The pyrexia and constitutional symptoms, generally present at the commencement of a case of *poliomyelitis* or *myelitis*, are absent here. Lumbar puncture is of the greatest value in enabling the physician to diagnose the presence, not merely of a little blood in the cerebro-spinal fluid, but of an extravasation almost entirely haemorrhagic.

The **Prognosis** varies greatly. Recovery of sensation is always favourable. In haemorrhage into the membranes relief may sometimes be obtained by operation. Extensive haemorrhage into the cord is usually soon fatal.

Treatment.—The patient should be placed, and if possible kept, in the prone position. Ice should be applied along the line of the spines, and ergotine administered. Once the haemorrhage has ceased, it may be prudent to canvass the possibility of operation.

(14) Tumours of the Spinal Cord and Membranes

Etiology.—With the exception of syphilitic and tubercular cases the origin is doubtful; possibly injury may be an important factor in some instances.

Tumours of the Cord.—These are rare; they include gliomata, sarcomata, tubercular nodules, gummata, and hydatid cysts.

Tumours of the Membranes.—These include gummata, sarcomata, myxomata, tubercular nodules, hydatid cysts, fibromata, psammomata, and lipomata.

Tumours and tubercular disease of the vertebral bodies and aneurismal tumours may also cause pressure on the cord.

The **Clinical Features** vary greatly, depending much on the extent of the cord involved, the amount of pressure on the nerve roots, and the degree of meningitis present in each case. If the tumour is in the cord, it may interfere with grey and white matter, and may induce all kinds of changes, possibly a hemi-lesion, or more likely a complete transverse lesion, and the case, to begin with, may suggest syringomyelia. Sometimes, for example, pain and temperature sensations are lost

and tactile sense impressions retained. If the tumour is situated in the membranes, or grows from the vertebral bodies, there is apt to be pressure on the nerve roots, with pain along the line of the affected spinal nerves. There is generally a localised myelitis in the neighbourhood of the tumour, and in cases of glioma, haemorrhage often occurs into the tumour tissue, greatly shortening the patient's life. Sensory and motor symptoms will depend on the site, extent, and nature of the tumour. The motor changes are often suggestive: paralysis with rapid wasting of muscles belonging to the affected segments, while below the lesion there may be spastic phenomena.

Diagnosis.—A careful examination should invariably be made of the vertebral spines, and by tapping with a percussion hammer, pain may be elicited over one or more vertebrae indicating the position either of the lesion or of the myelitis to which it has given rise. A history of syphilis and evidences of tubercle in other parts of the body may be helpful, but often the diagnosis is difficult.

The **Prognosis** depends largely on the nature of the tumour; in certain cases surgical interference is possible. In other cases, and especially where the lesion is syphilitic, potassium iodide and mercury may be administered, while in tubercular disease of the vertebral bodies prolonged rest on the back and sometimes surgical treatment are beneficial. Tumours of the cord itself are of necessity very grave.

The **Treatment** has been indicated. In tubercular and syphilitic cases much may be done in the way of attempted cure, and in tumours of the membranes surgery offers some hope of relief. The bladder must be watched, and when necessary the urine drawn off. Bed-sores should be carefully obviated, and, where they develop, treated with suitable antiseptics. Sometimes the patient requires the administration of morphia to relieve pain.

(15) Subacute Combined Degeneration of the Spinal Cord

In pernicious and other profound anaemias, and perhaps more often from unknown causes, this "toxic" sclerosis occurs and most generally in patients between 50 and 60 years of age

Pathological Anatomy.—It is an increase of neuroglia replacing degenerated nerve fibres, and involving the peripheral parts of the posterior and lateral, and to a less extent, the anterior columns. Isolated patches of sclerosis appear at some distance from the surface and spread towards the periphery of the cord. There are only slight changes in the cells of the grey matter, excepting in Clarke's vesicular column, and there may be evidence of chromatolysis in the cells of brain and cerebellum. Endogenous tracts such as the septo-marginal do not participate.

Clinical Features.—After a preceding history of weakness and ill-health the patient begins to suffer from numbness of the limbs, going on later to anaesthesia and the appearance of spasticity of the legs with stiffness of muscles. The tendon reflexes are exaggerated, and there is plantar extension (positive Babinski sign). There may be increasing weakness of sphincter control. The last stage is flaccid paralysis with loss of tendon responses and sphincter control. Bed-sores, cystitis, mental deterioration, and delirium often ensue later. In other cases there are locomotor-like pains with loss of the knee-jerks at the commencement, without any spasticity.

Diagnosis.—The disease resembles *spastic paralysis, locomotor ataxia*, and *peripheral neuritis*, but the presence of anaesthesia in the spastic type of cases and the absence of the Argyll-Robertson pupil in the tabetic type render mistake unlikely.

The **Prognosis** is unfavourable, and the **Treatment** must be on general lines, iron and arsenic being of most benefit, while rest in bed should be enjoined.

(6) Caisson Disease—Diver's Paralysis
(Compressed-air Disease)

A disease due to high atmospheric pressure, of at least 1, generally 2 to 4 atmospheres.[1]

Etiology.—The *predisposing* causes are, any lowered vitality, especially alcoholism, obesity, and diseases of the heart and kidney. Inexperienced workmen suffer most, but

[1] Each 33 feet of salt or 34 feet of fresh water equals the pressure of 1 atmosphere. The record depth attained by divers is said to be 210 feet.

even those who are experienced cannot remain in a caisson at a great depth for more than a very limited time.

The phenomena are thought to be due to escape of nitrogen gas from the blood, when the pressure is taken off.

Pathological Anatomy.—Haemorrhage and congestion have been described in acute cases, and certainly myelitis in cases fatal after some time, but the gross lesions are very indefinite. Leyden has described spaces in the cord.

Clinical Features. At the onset there may be pains in the joints and muscles. The more serious symptoms are *cerebral* and *spinal*. The *cerebral* include faintness, with nausea and vomiting, headache and giddiness, sometimes coma, and occasionally apoplectiform seizures, which may be instantly fatal. The *spinal* include neuralgic pains, often very severe and often in paroxysms, felt in the legs, arms, back, etc., increasing weakness, and later paralysis.

In the majority of cases only certain of these symptoms appear, and they may do so immediately, or some time after the patient has left the compressed atmosphere.

Prognosis.—In most cases in recovery; where the more severe phenomena appear, paralysis or death.

Treatment.—No one should enter the caisson who feels seedy, and all newcomers should work for a very short time at first, and never at a great depth. The greater the pressure the shorter should the time of work be. For the intense pains give opium. Immediate recompression is of great value.

(17) Syringomyelia

The formation of a cavity or cavities in the spinal cord, associated with the presence of more or less glionatous embryonic tissue, in which the cavity formation may originate.

Etiology.—Men are more frequently affected than women and the age at which symptoms first appear is usually between the ages of 15 and 30. It is an increase of neuroglial tissue, and possibly a congenital condition dependent on the persistence of embryonic tissue, but it may also be due to haemorrhage into the spinal cord. In rare cases there may be evidence of a spina bifida.

Pathological Anatomy.—The cavity or cavities may be connected with the central canal, or may be distinct. They

may extend up into the medulla and pons, and in the cord are most commonly met with in the cervical enlargement or the upper dorsal region. Gliomatous tissue surrounds the cavity in varying amount, and the cavity usually enlarges, and when it does so, causes pressure on neighbouring structures. The simplest form of cavity is a dilatation of the existing canal of the cord, and is probably dependent on an embryonic error of development, although possibly also on an excess of cerebro-spinal fluid. The cavity is generally found in the grey matter, and the anterior or posterior horns may be invaded. The embryonic tissue surrounding the cavity or cavities is translucent in appearance and retains much of its embryonic character, and sometimes this embryonic tissue is found at a lower level than that to which the cavity extends.

Both grey and white matter are liable to pressure when the cavity enlarges, and it is to this pressure that the typical clinical features are due.

Clinical Features.—Between the 15th and 30th year the symptoms and signs of syringomyelia generally develop, and probably therefore at the time when the strain of life becomes greatest. Its onset is insidious, and the following symptoms are present. The clinical features may be unilateral, bilateral, or bilateral but more advanced on one side, dependent on the position of the cavity.

(1) Loss or diminution of temperature sense and pain, sometimes involving both arms, often more marked in one. In other cases, areas of cutaneous anaesthesia develop and gradually extend. Tactile and muscular sense impressions are usually unaffected.

(2) Atrophy of certain muscles frequently occurs, beginning in the small muscles of the hand and extending upwards, sometimes involving special groups of muscles, and dependent on the amount of interference with the anterior horn.

(3) Spastic phenomena develop in the legs, the result of pressure on the pyramidal tracts, with exaggerated tendon reflexes and ankle clonus, and the Babinski sign is positive.

(4) Marked trophic changes occur in certain bones and joints of the affected limb or limbs. The bones become brittle, and the joint changes may resemble what is sometimes met with in locomotor ataxia.

(5) In certain cases of syringomyelia, trophic changes also

occur in the skin of the hands and in the nails. The skin becomes glossy, the nails thickened, brittle, and with a tendency to chop off. Deep-seated ulcers or painless whitlows sometimes develop, and eczema, herpes, and other skin affections have been noted, while localised sweating is occasionally present. Cases of syringomyelia with painless whitlows have been termed Morvan's Disease, but there is no necessity for the special name.

The electrical examination may show the reaction of degeneration in wasted muscles; fibrillary twitchings and tremors may be present, similar to what is seen in progressive muscular atrophy. The spinal muscles may be weakened and scoliosis results.

If the lesion is situated in the lumbar enlargement, the organic reflexes may be abolished, and bed-sores, cystitis, and incontinence of the bowels occur. Where a cavity extends into the medulla and pons, bulbar phenomena are apt to appear, such as difficulty of swallowing and of respiration, and enfeeblement of heart's action.

Diagnosis. *Leprosy*, although it may be associated with painless ulcers, is easily distinguished. A localised *myelitis* or a *cervical pachymeningitis* may each cause symptoms so far resembling syringomyelia, but in cervical meningitis the retraction of the head is suggestive. A *cervical rib* should be easily recognised by palpation and X-ray examination, and no spinal symptoms are associated. It is possible that in *gliomatous tumours* of the cord cavities may arise, and so phenomena resembling syringomyelia may develop, but the slowly progressive clinical features of a typical case of syringomyelia are very different from the rapid advance of a gliomatous tumour.

Prognosis.—Sometimes the disease becomes stationary, not merely for months but for many years, and in such cases there may be a moderate improvement, and the trophic affections are more likely to be amenable to treatment.

The **Treatment** must be on general lines. No surgical procedure can effect a cure, and as the disease is embryonic in origin, drugs are of little avail. Attention should be paid to the treatment of trophic sores and skin affections.

18) Spina Bifida

It is important to consider spina bifida in its relationship to syringomyelia. It is defective closure of the bony canal containing the spinal cord. The membranes lining the cord bulge into the sac, which is distended with cerebro-spinal fluid. It is usually found in the lumbar region of the spine, and in a considerable proportion of cases the central canal of the cord is enlarged, in connection with the presence of the meningocele. This enlargement of the central canal is common above and sometimes below the site of the protrusion of the sac, but generally the central canal of the cord does not communicate with the sac, although in a few cases it does.

Clinical Features.—The symptoms depend on the interference, if any, with the spinal cord. These include paralysis, possibly atrophy, and perhaps sensory changes, as a rule involving the legs only. Talipes varus is the most frequent result of such muscular change, the anterior tibial muscles being generally affected.

The **Prognosis** depends on the possibility of surgical treatment.

The **Treatment** is purely surgical, and usually the sac is tapped. The cases in which the cord is also affected are eminently unsatisfactory.

(19) Diseases of the Cauda Equina

The spinal cord is considerably shorter than the spinal canal and terminates at the lower border of the first lumbar vertebra. As a result, there are many nerve roots which pass downwards below the level of the cord to their respective canals of exit. The *conus medullaris* is the term applied to the lowest portion of the spinal cord proper, and from the apex of this conus the filum terminale passes downwards for 17 to 18 cm. closely surrounded by the lower lumbar and sacral nerve roots to terminate opposite the second sacral vertebrae. The filum, with its surrounding nerve roots, constitutes the *cauda equina*, and a lesion in this position may cause extensive motor and sensory paralysis.

Etiology. Injury is the most important etiological factor. Should the first lumbar vertebra be injured, the conus

medullaris suffers, while in cases where the lesion is situated below that level the conus medullaris may escape, and the cauda equina is generally alone involved. Tumours of bone or meninges, haemorrhages, meningitis, and abscess may also produce lesions of the cauda equina.

Clinical Features.—Much depends on the level of involvement and the number of nerve roots which are affected, and the distribution of sensory and motor symptoms follows the distribution of a *root* rather than a *nerve* lesion. A complete lesion of the cauda equina results in flaccid paralysis and atrophy of all the muscles supplied by the sacral plexus and by the lumbar roots below the second, and there is high steppage gait due to marked foot-drop. A definite reaction of degeneration is obtained, and pain is not infrequently a prominent and very disturbing factor. There is anaesthesia over a saddle-shaped area in the gluteal region and at the back of the thighs, and also over the outer side of the leg and foot. There is anaesthesia of the genital regions, with incontinence of bladder and bowels, and loss of sexual power.

The Achilles, plantar, anal, and bulbo-cavernosi reflexes are abolished, while the knee-jerks are retained. According as the first, second, or third lumbar roots escape, so does the area of anaesthesia diminish from above downwards.

Diagnosis.—In lesions of the cauda equina the symptoms come on more slowly than is the case where the lesion involves the *conus medullaris*. There is also greater pain, and Lasègue's sign points to a lesion of the cauda equina, or at least of the associated nerve roots. This sign implies the production of pain on an attempt being made to flex the thigh on the pelvis with the leg extended. Want of symmetry in symptoms and signs favours the diagnosis of a caudal lesion, while it has been stated that anaesthesia favours a lesion of the conus medullaris, and especially anaesthesia to pain and temperature, but not to tactile sensations. The **Prognosis** is certainly better where the lesion is one involving the cauda equina. Anti-specific **Treatment** should invariably be tried, and in not a few cases operation has yielded satisfactory results.

V. DISEASES OF THE MEDULLA AND PONS

Glosso-Labio-Laryngeal Paralysis (Bulbar Paralysis)

The term bulbar paralysis implies a lesion of nerve centres in the medulla, although nuclei in the pons may also be involved. The result of the lesion is paralysis, more or less complete, of the muscles governing speech, including phonation, and of those which have to do with the acts of swallowing and mastication; while more rarely the eye muscles and other muscles innervated by cranial nerves become affected (see Fig. 35, p. 614).

The lesions in question include haemorrhages, acute inflammations, or patches of sclerosis involving the nerve nuclei, although perhaps the most important, because the most common, group of cases depends in reality upon the action of a toxin similar to that which causes progressive muscular atrophy. The action of this toxin is probably limited to the nerve cells. Such lesions may be secondary to progressive muscular atrophy, or may develop primarily in the medulla and pons.

(1) Acute Bulbar Paralysis

This appears to be sometimes the result of influenza or some other continued fever: it may be haemorrhagic in nature, or it may be a polio-encephalitis akin to polio-myelitis in the cord. Either the haemorrhage or the encephalitis may involve the higher nuclei, as, for example, those of the 3rd and 4th nerves, or may be limited to those in the medulla and pons: even in the cases where the lesion is probably an encephalitis, minute haemorrhages are extremely common.

The onset is acute and the muscles may be extensively involved; where the higher nuclei suffer, certain of the eye muscles are paralysed, while not infrequently there is nystagmus and a cerebellar gait. Where the lesion is in the pons or medulla, there is more or less paralysis, often of all four limbs, sometimes a hemiplegia and sometimes a crossed paralysis. The latter condition is dependent on a hemi-lesion in the lower half of the pons, by which the motor fibres to the face are affected on the side of the lesion, while the fibres to the

opposite arm and leg are also affected (see Fig. 50 on page 712). Extensive lesions of such an acute nature in the medulla or pons are incompatible with prolonged life, and the disease soon terminates fatally.

There are instances in which acute softening is the result of embolism or of syphilitic endarteritis in connection with some small nutrient artery supplying that region. In these cases the paralytic phenomena depend on the position of the patch of softening. There are generally marked headache, giddiness, and loss of consciousness, while vomiting and sometimes convulsions may occur. It is always possible to have paralysis of the respiratory and cardiac centres, which may suffer in the same way as the nuclei or fibres of the cranial nerves. Haemorrhages are by no means uncommon in connection with patches of softening, and either an extensive haemorrhage or a progressive thrombosis may rapidly terminate the patient's life. Many of these cases are of syphilitic origin, and where treatment is possible, they should be treated by the usual antisyphilitic remedies.

2 Chronic Bulbar Paralysis

Etiology.—A disease of middle life, more frequent in men, and generally in those of neurotic family history; possibly dependent on mental over-strain, exposure, syphilis, and lead poisoning. It generally develops as an extension upwards of the lesion of progressive muscular atrophy.

Pathological Anatomy.—There are marked degenerative changes in the nuclei of certain nerves in the medulla, and especially the hypoglossal, the facial, the glosso-pharyngeal, the spinal accessory, and the vagus. The nerve-endings in the affected muscles degenerate, and so do the nerves and the muscle fibres to which they are distributed. The nerve-cell changes in the nuclei are typical, the cells atrophy and certain of them disappear, while in others pigmentary changes have been described, and there are disappearance of the chromatic substance from the cell protoplasm and diminution in the number of grey processes. The affected nerve-roots are grey and shrivelled, and many nerve fibres in them are found to be degenerated.

Clinical Features.—The development of the bulbar

symptoms is insidious, and begins with indistinctness of speech from weakness of the tongue and lips; the letters *l*, *n*, *r*, and *t* are among the first to be affected, while later on *o*, *u*, *p*, *b*, and *m* are pronounced with difficulty. The palate becomes weak, fluids tend to regurgitate through the nostrils, and the patient speaks with a nasal tone. Soon afterwards swallowing becomes difficult, partly because the tongue is unable to perform its usual function in moving the bolus of food backwards towards the oesophagus, but also because the oesophageal muscles themselves are weakened. The glottis may not close, and in time, food tends to enter the larynx, either causing fits of coughing by which the foreign body is expelled, or else setting up a septic pneumonia if inspired. Paralysis of the cords indicates the involvement of the nuclear cells governing the laryngeal muscles, and the patient becomes aphonic. When this stage is reached the tongue is usually completely paralysed, cannot be protruded from the mouth, becomes wrinkled, and fibrillary tremors may be seen in it. The lower lip hangs down, and thick, tenacious saliva tends to dribble more or less constantly. In amyotrophic lateral sclerosis in which bulbar involvement is common the lips are often affected early. The electrical excitability of the affected muscles is diminished, and there may be a partial reaction of degeneration. The patient's face hardly demonstrates the fact that in mind and intelligence he is as acute as ever, and often keenly feels his disability as regards speech and deglutition. There is no change in sensation, and, as a rule, the organic reflexes are unaffected.

The disease is a progressive one, and serious involvement of the respiratory and cardiac centres in the medulla may lead to a fatal result, or septic pneumonia already mentioned may terminate the patient's life.

Diagnosis.—The lesion is one of the second trophic realm. It is localised, and usually the nuclei become involved in the order mentioned in describing the clinical features. In a considerable proportion of cases the disease has commenced with progressive muscular atrophy or amyotrophic lateral sclerosis, and only in the later stages do the bulbar phenomena develop. In *myasthenia gravis* there is more loss of power and less wasting of muscles, and the myasthenic reaction (p. 804) is present.

The **Prognosis** is bad, and as a general rule nothing can be done to prolong life.

Treatment.—Benefit might be hoped for, should the lesion be syphilitic in origin. Keep the patient at rest, and avoid excitement of all kinds. The feeding is most important, especially in cases where there is the slightest risk of food entering the larynx. Electrical treatment should be tried, and especially galvanism, while strychnine, arsenic, phosphorus and silver have proved beneficial in a very few cases.

VI. DISEASES OF THE BRAIN

Introductory.—A series of figures show the convolutions of the cerebrum, the blood-supply of these convolutions and

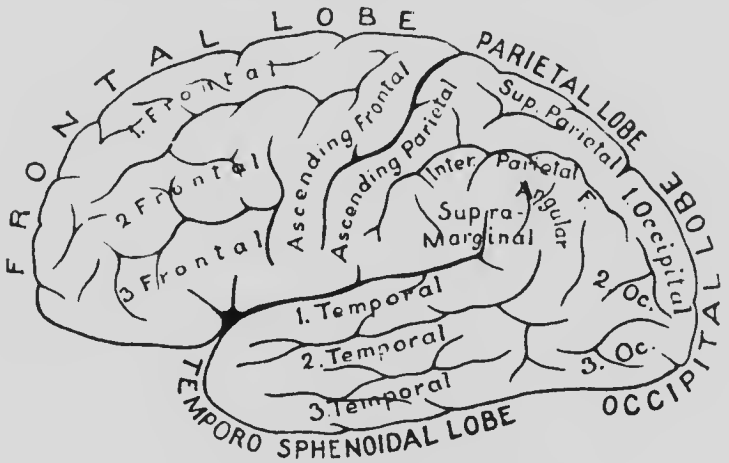


FIG. 44.—Figure showing Cerebral Convolutions.

the very important relationship of the blood-supply to the great motor and sensory areas of the cortex. A description is given under regional diagnosis of the results of cerebral lesions, with the position of the chief centres, while the sensory and motor centres for the reception of symbols and the evolution of speech are discussed under aphasia. A figure of the circle of Willis with the nutrient arteries which come off from it for the supply of the region of the internal capsule is shown on p. 721.

1. Regional or Topical Diagnosis of Cerebral Lesions

The Cerebral Cortex

Frontal Lobe.—The pre-frontal region has been termed a silent area, while in the posterior part of the superior,

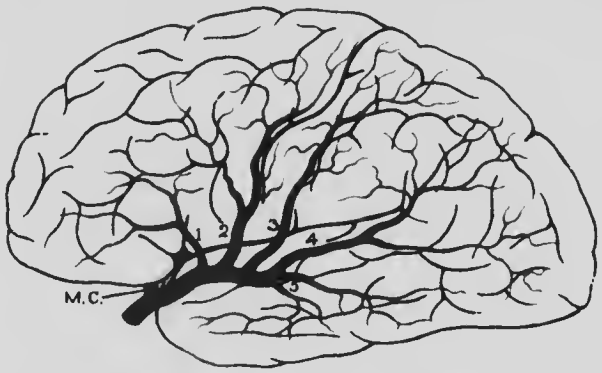


Fig. 75.—Cerebrum showing distribution of Middle Cerebral Artery.

middle and inferior frontal convolutions there is the psycho-motor area (to be referred to presently) and forming part of this area there is a centre for conjugate deviation of the head and eyes to the opposite side, and on the left side of the brain in right-handed people, the writing and speech centres.

Fig. 76.—Cerebrum, showing area of blood-supply from Anterior, Middle, and Posterior Cerebral Arteries.

The frontal lobe appears to be associated in some way with the intellect and memory, because disease of the frontal region causes failure of memory, hebetude, sleepiness, and in cases in which the affection is less marked a great inability to concentrate the mental faculties. It must, however, be admitted

that in certain cases extensive lesions of the frontal lobe have been present without any definite mental feebleness.

The following are the common lesions met with in the frontal lobe—tumour, abscess, and haemorrhage from the anterior cerebral artery; but the last named is rare, and is more commonly the result of trauma.

THE ROLANDIC REGION.—The motor or precentral area lies immediately in front of the fissure of Rolando, occupying the ascending frontal convolution, and in this area, as indicated in Fig. 49, the leg, trunk, arm, and face centres are situated. In this area there are many large pyramidal cells and many Betz cells, with the exception of

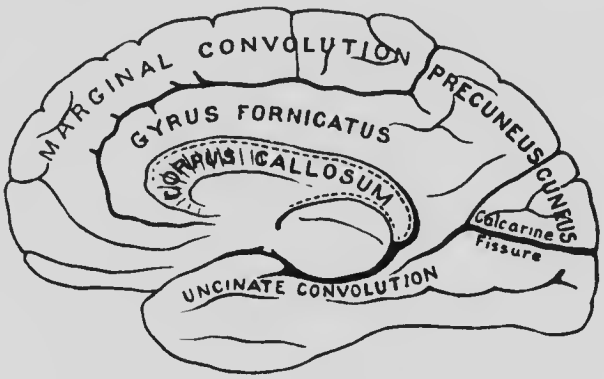


FIG. 47.—Internal surface of Cerebrum.

the face centre, in which no Betz cells are present. The Betz cells are the cell centres of the motor neurones. In front of this precentral motor area there is a psychomotor or intermediate precentral area, which extends into the posterior parts of the superior middle and inferior frontal convolutions. This psychomotor area is the portion of the brain in which the voluntary movements of muscles are controlled, and in this region is situated the writing centre, the conjugate deviation centre, and the motor speech centre (the writing and speech centres being only active on the left side of the brain in right-handed people).

(*a*) *An Irritative Lesion.*—This causes epileptiform convulsions on the opposite side of the body from the lesion, and it may be limited to one arm, one leg, one group of muscles, etc. (monospasm); in other cases the spasms become

general, and may be associated with loss of consciousness. There may be no organic lesion, unless some paralysis develops, but in most cases there is a subsequent and temporary motor paralysis, and sometimes tingling or numbness of the affected limb or part of a limb. The group of clinical features just described constitutes Jacksonian epilepsy, and the commonest causes are tumour, a syphilitic lesion such as a gumma, occasionally patches of softening, and more rarely haemorrhage.

(*b*) *A Destructive Lesion.*—This may be the sequel to an irritative lesion: a tumour or haemorrhage may first cause irritation and later paralysis. The paralytic phenomena involve the muscles on the opposite side of the body from

FIG. 48.—Internal surface of Cerebrum showing supply of Anterior Cerebral in yellow, Posterior Cerebral in blue, and Middle Cerebral uncoloured.

the cortical centre or centres which have been destroyed. This is a lesion of the first or upper motor neurone or first trophic realm, and therefore there is no electrical change and no rapid muscular wasting. The lesion, if extensive, closely corresponds to a lesion in the internal capsule, but in the region of the cortex a large area requires to be involved in order to produce a complete hemiplegia. There may be therefore a succession of attacks, with a gradual involvement of more and more of the cortical centres: paralysis of one limb alone (monoplegia) generally suggests that the lesion is cortical. Where the cortical lesion is on the left side of the brain and involves Broca's convolution, motor aphasia results if the individual is right-handed.

PARIETAL LOBE.—The ascending parietal or post-central convolution is the great cortical sensory centre, and in it probably impressions of *passive position of limbs*, of *muscular*

movement, of *tactile localisation*, of *pain*, and *temperature sense* are received. Immediately behind this great sensory centre there is a psycho-sensory or intermediate post-central area in which the intellectual processes for the utilisation of sensory impressions may be carried out. The superior parietal region was believed to be associated with the ability to recognise the shapes of things, or what is termed the *stereognostic sense*, but probably this is inaccurate. A lesion of the inferior parietal lobule may yield no definite sign, or there may be some ptosis-hemianopsia, and also, if it is on the left side of the

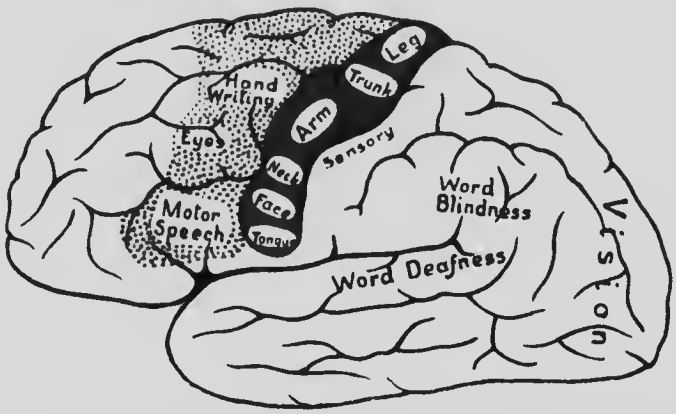


FIG. 49.—Left half of Brain showing Cortical Motor (in red) and Cortical Sensory (in yellow) areas. It also shows the position of the visual, auditory, and speech centres. The above areas of colour indicate the psycho-motor and psycho-sensory centres respectively.

brain, word-blindness. The relationship of ptosis to the occipital gyrus lacks confirmative evidence.

OCCIPITAL LOBE.—A lesion of the occipital lobe causes homonymous hemianopsia which may be associated with a hemiplegia and also a hemianaesthesia, provided the internal capsule is also involved; if the lesion extends to the left angular gyrus, then word-blindness is associated with the hemianopsia.

TEMPORAL LOBE.—There may be no symptoms, or deafness if the middle and posterior portions of the superior temporo-sphenoidal convolution are involved, while if the lesion is on the left side of the brain there is word-deafness. The cortical centres for smell and taste are believed to be located in the lower extremity of the temporal lobe.

THE HIPPOCAMPAL GYRUS (POSTERIORLY) AND THE GYRUS FORNICATUS.—These have been considered to be the centres for *tactile* and *general sensation*, and possibly also the centres for recognition of the *position of the limbs* and *muscles*, but the parietal region is probably the chief sensory centre.

THE CORPUS CALLOSUM.—The commissural fibres joining opposite cortical centres pass through this structure. Bristowe has described four cases of tumour in which the lesion was found to be in the corpus callosum. In all these cases they were (1) ingravescent: (2) gradual in onset, with hemiplegia, better marked on the one side, but also vaguely present on the other; they showed (3) drowsiness, apathy, difficulty in swallowing, and speechlessness: (4) no cranial nerves were involved; (5) death occurred from coma. There was no sickness, no headache, and in one case out of the four no optic neuritis.

CENTRUM OVALE AND INTERNAL CAPSULE.—Destructive lesions of the centrum ovale cannot be distinguished from those of the Rolandic cortex, but irritative lesions show certain differences. If they are cortical, there are tonic, followed by clonic, convulsions. If, on the other hand, the lesion is situated in the centrum ovale, there are only tonic and no clonic movements, probably because the stimulus must be continuous in order to produce clonic spasms. If a haemorrhage is immediately below the cortex, there may be a typical history of cerebral haemorrhage with, in addition, an element of spasm. Convulsions and early rigidity in cerebral haemorrhage may be due to pressure on the fronto-parietal fibres, and not to haemorrhage into the lateral ventricles.

The position of the motor fibres in the anterior two-thirds of the posterior half of the internal capsule, and of the sensory fibres which are supposed to be in the posterior one-third, as well as the relationship of the optic radiation of Gratiolet, may be seen in Fig. 53, and reference will be made to the usual site of a cerebral haemorrhage in connection with that affection.

CORPUS STRIATUM, OR THE BASAL GANGLIA.—Most lesions of the corpus striatum cause hemiplegia. The *caudate nucleus*, according to Ferrier, governs in some way the movements on the opposite side of the body: but softening or tumour has been found in both the *caudate* and *lenticular* nuclei, and yet

no paralysis resulted. A haemorrhage into the head of the caudate nucleus causes hyperpyrexia, and the haemorrhage is most generally from one of the antero-median nutrient arteries (see Fig. 54). One of the causes of pseudo-bulbar paralysis is a bilateral lesion of the corpus striatum. The diagnosis is simplified by remembering that pseudo-bulbar lesions are lesions of the upper motor neurone producing no rapid muscular wasting, while true bulbar lesions are lesions of the lower motor neurone with marked wasting of muscles.

Optic Thalamus.—The optic thalamus is one of the most important ganglionic structures in connection with sensory fibres. A lesion of the optic thalamus generally produces: (1) superficial and deep hemianaesthesia, especially the latter; (2) a slight hemiplegia, usually with contracture; (3) a slight hemiataxy, with astereognosis; (4) severe pain in the hemiplegic side, often paroxysmal; (5) athetoid movements of the paralysed limbs; and sometimes (6) paralysis of mimetic movements of the face.

The Crura Cerebri.—Lesions limited to the crura cerebri or to one crus are rare. Tumours, generally tubercular in nature, or gliomata, may invade the crura cerebri, and neoplasms growing from the base of the skull have sometimes involved this structure. Haemorrhages, thrombotic softenings, and abscesses are rare. The sensory fibres pass in the tegmentum, and the motor fibres in the middle portion of each crus. A form of alternate or crossed paralysis, consisting in the arm and leg and often the lower facial region being paralysed on the opposite side from the lesion and the 3rd nerve on the same side, may be produced by the involvement of one crus. The lesion of the 3rd nerve is not always complete.

Tumours involving the crura cerebri readily involve both 3rd nerves, and may also affect both motor tracts. A lesion of the tegmentum causes anaesthesia.

The Corpora Quadrigemina.—These structures are too small in extent to be involved alone by haemorrhages, tumours, or softening, and therefore it is only by a study of different cases that it is possible to arrive at the correct phenomena to which their involvement gives rise. There is an unsteady, reeling gait much like the drunken gait in a case of cerebellar tumour. There is often also ophthalmoplegia, generally incomplete, and due to an affection of the subjacent 3rd and 4th nuclei.

Irritation of the corpora quadrigemina on one side causes dilatation of both pupils, and especially that of the opposite eye; and it must be remembered that there are important connections between the fibres subserving vision and these bodies (p. 616).

The central tracts of the cochlear nerve ascend through the opposite lateral fillet, the posterior tubercle of the corpora quadrigemina, the brachium, the internal geniculate body, and thence to the temporal cortex. Therefore hearing should be tested in all cases of supposed lesion of the corpora quadrigemina, and, generally speaking, it will be involved on the side opposite to the tubercle affected (in 19 cases of tumour of the corpora quadrigemina, 9 showed a definite defect of hearing).

In tumour cases other structures are invariably involved, such as the optic thalamus, the tegmentum, the pons, or the superior cerebellar peduncles. In experimental cases tetanic spasms are caused by irritation of the corpora quadrigemina. The knee-jerks have been found to be either normal or more generally exaggerated; in certain cases they are abolished.

The Pons.—The symptoms vary according to the position and extent of the lesion. Tumours are occasionally met with, but gliomata may infiltrate the whole pons, and haemorrhages, unless small, rapidly prove fatal. A lesion of the pons tends to involve the 5th, 6th, and 7th nerves on the side of the lesion and the limbs on the opposite side.

The chief clinical features of a pontine lesion are profound coma, and complete relaxation of the muscular system, with, however, in some cases, a tendency to tonic or clonic spasms. There is often hyperpyrexia, the temperature even reaching 109 Fahrenheit, but only in cases where the lesion is sudden as in haemorrhage. Pin-point pupils are almost invariably present.

Crossed paralysis depends on the level of a lesion which is pontine and unilateral. The fibres from the face centre decussate in the middle of the pons on their way to the 7th nucleus, and therefore a lesion above the middle of the pons will cause paralysis of the face, arm, and leg on the opposite side from the lesion, whereas a lesion in the lower half of the pons will cause paralysis of the arm and leg on the opposite side from, and of the face fibres on the same side as, the lesion. Similarly the 5th and 6th nerves may be affected, as

well as the 7th on the same side as the lesion, and the limbs and tongue on the opposite side. The involvement of the tongue depends on whether the lesion extends to the fibres or nucleus of the 12th nerve in the medulla. Hearing is not infrequently affected on one or both sides, and from the small size of the pons and the close relationship of motor and sensory tracts hemianaesthesia or bilateral anaesthesia may be present. There is often hemianaesthesia of the body on the opposite side from, and of the face on the same side as, the lesion, depending on whether the lesion is above or below the junction of the ascending and descending roots of the 5th nerve. There are sometimes ataxic disorders associated with pontine lesions, but the position of the fibres to which the phenomena are due is somewhat doubtful. Articulation and deglutition may be affected; interference with articulation probably depends on the involvement of the central fibres of the 12th nerve above the nucleus.

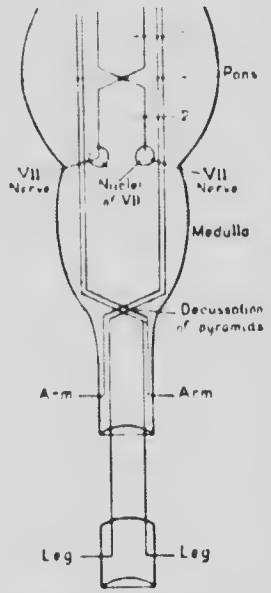


FIG. 50.—Schematic Figure showing Decussation of Face (7th) Fibres in the middle of the Pons and the Arm and Leg Fibres at the Pyramidal Decussation in the Medulla. Lesion 1 would give paralysis of face, arm, and leg on the opposite side from the lesion. Lesion 2 would give Crossed Paralysis, the face being paralysed on the same side as the lesion and the arm and leg on the opposite side.

CEREBELLUM.—The common lesions in the cerebellum are tumours, abscess (secondary to middle-ear disease), and in young persons, haemorrhage. The clinical features of cerebellar disease may be latent, but in most cases there are the following very definite and suggestive phenomena. Instability when attempting to stand or walk is peculiarly typical. The gait resembles that of a drunk man, although curiously enough in some cases, when the patient lies down, the finest movements may be possible without any inco-ordination. It is probable that the middle lobe of the cerebellum is the special centre for co-ordination. Rotatory movements believed to be associated with the cerebellar peduncles are sometimes present, but these movements are not constant and are of doubtful significance. The cerebellum governs combined and co-ordinated

muscular movement, and for this the cerebrum, the semicircular canals, and the cerebellum are needed and the integrity of the tracts joining these important centres with each other. Paralysis may occur in cerebellar lesions, but there is no special site, and it is probably the result of pressure. There is marked weakness of the limbs even where there is no paralysis, and tremor of head and limbs is often noted. Convulsions are not uncommon on the side of the lesion. The knee-jerks vary; they may be increased, diminished, or sometimes normal. The ocular symptoms include nystagmus which is extremely common, and optic neuritis which is practically constant in the case of cerebellar tumours. There may be skew-deviation of the eyes in patients suffering from tumour, the eye on the side of the tumour being turned downwards and inwards, and the opposite eye upwards and outwards. Hearing, smell, taste, and cutaneous sensation are generally unaffected.

2. Aphasia

There are two types of Aphasia, *motor* and *sensory*. Motor aphasia takes the form of inability either (*a*) to speak or (*b*) to write, while sensory aphasia includes inability to appreciate words spoken (or auditory aphasia), and inability to understand words seen with the eye (or visual aphasia). Aphasia does not necessarily include any lack of intellectual power. The term is reserved for cases in which the higher intellectual centres are capable of functioning, but in which the cortical centres subserving speech or writing (motor aphasia), or the centres for the reception of auditory or visual impressions (sensory aphasia) are affected.

The figure shows the position of the important centres. The speech centre is mainly situated in the posterior end of the inferior frontal convolution, (Broca's convolution). The writing centre is situated in the posterior part of the mid-frontal convolution. Both these centres are placed in what are termed the psychomotor areas, situated in front of the motor area proper of the brain. The speech centre is in close proximity to the motor area for face, mouth, etc., and the writing centre is just anterior to the motor area for the arm and hand. The auditory centre is situated in the posterior part of the superior temporo-sphenoidal convolution,

and probably a large part of this should be termed the psycho-auditory centre or the word-hearing centre. The higher visual centre or word-seeing centre is situated in the angular and supra-marginal convolutions. Connected with it are two half-vision centres, one in the region of the calcarine fissure on the same side of the brain, and the other in a similar region on the opposite side. These centres are, with the exception of one half-vision centre, all situated on the left side of the brain in a right-handed person.

The connecting links between these important sensory and motor centres are easily worked out. A child first learns the

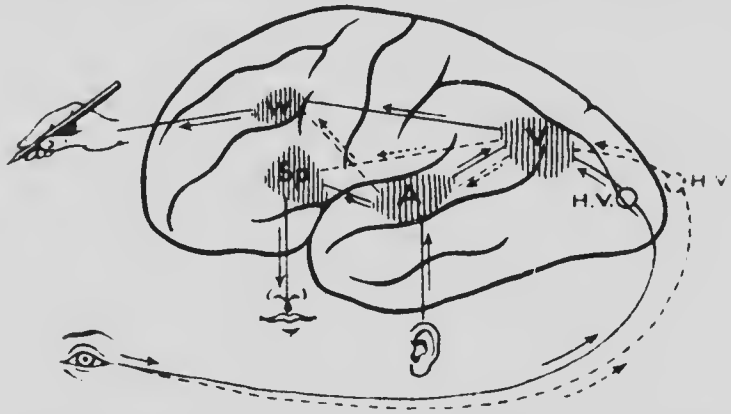


Fig. 51.—Sp, Speech centre; W, Writing centre; A, Auditory centre; V, Visual centre; H.V., H.2, Vision centres. The dotted lines, with the exception of the line connecting H.2 Vision centre, indicate the less usual paths of impulses. (After Bramwell.)

sound of a word by the ear and recognises in time what the meaning of the word is, and by imitating what he hears, the first important connection is demonstrated between auditory and speech centres. Later he sees words printed, and realises the meaning of these words by associating them with the impressions learned by the ear. In this way the auditory is connected with the visual centre, and in writing to dictation the visual is in turn connected with the writing centre. The other connections between these centres are shown by dotted lines which are intended to indicate that although impulses may pass up these channels, they do so less frequently and regularly. Most people use the auditory centre far more than the visual, and therefore a lesion involving the auditory centre directly or indirectly causes far greater disturbance than does

a lesion of the visual. It is probable that the store-houses of memory are to be found in the auditory and visual centres, but the frontal convolutions have invariably had assigned to them an important function as regards intelligence, and possibly the frontal lobes are the chief centres of the individual's memory as well as of his intellectual powers.

Motor Aphasia.—Here the patient is unable to express himself by words spoken. He may be able to repeat, probably by means of the corresponding speech centre on the opposite side of the brain, certain simple words, but to all intents and purposes he is speechless, and yet he is aware when wrong names are given to things shown to him. Such a lesion may be *cortical* or *sub-cortical*.

If *Cortical* he is not merely unable to speak, but as there is probably some association of ideas carried out in the speech centre, his appreciation of things said to him or shown to him in writing is considerably interfered with.

In *Sub-cortical* motor aphasia the centre is uninjured, but he cannot speak because the pathway outwards is blocked. He can, however, understand what is said to him and what is shown to him in writing, and he can write what he cannot speak aloud.

Cortical and Sub-Cortical Agraphia.—Such lesions have not been described in an isolated form. Agraphia is more usually associated with sensory aphasia and specially cortical word-blindness, but it may be associated with motor aphasia in certain cases.

Sensory aphasia may be either (1) *auditory*, or (2) *visual*.

1. **Auditory Aphasia,** or **Word-deafness.**—In this condition the patient is not necessarily deaf although he is word-deaf, by which is meant that he cannot understand the meaning of words spoken to him.

Cortical Auditory Aphasia or cortical word-deafness is due to a lesion of the cortical word-hearing centre, producing loss of the memories of words spoken. The motor speech centre obtains no guiding impulses from the auditory word centre. The patient is much confused intellectually, continued thought becomes impossible, and mistakes are made in reading as well as in speaking. Generally words become mixed up in a hopelessly confused jargon, the patient being totally unaware of the lack of intelligence which he demonstrates and the errors

of speech which he makes. The reason why the cortical centre for hearing is so important is due to the fact that we mostly get our knowledge of symbols by the ear. Some improvement may eventually occur when the centre on the opposite side of the brain becomes educated to take up the functions of the damaged area.

Sub-cortical auditory aphasia or *word deafness* implies that the centre is intact, and that the lesion interferes with the path by which impressions enter. Such a lesion must be, and is, very rare, but should it occur, the patient can read intelligently, and spontaneous speech remains unimpaired, and he can understand anything written because the auditory centre is intact, while in cortical word-deafness this is generally impossible.

2. **Visual Aphasia,** or **Word-blindness**, or **Alexia.**—Here the patient can see but cannot interpret what he sees.

Cortical visual aphasia or *word-blindness* implies the destruction of the visual word-centre. As a result he cannot write either from dictation or from copy, although in the case of an incomplete lesion he may rather confuse words and write wrong ones, than be totally unable to write at all.

Sub-cortical visual aphasia or *word blindness* implies the destruction of the path by which visual impressions are carried to the centre, the visual centre itself remaining intact. In this case the patient can write spontaneously and to dictation, but cannot read from a book or even read what he has written. To produce such sub-cortical word-blindness the lesion must divide the path from both half-vision centres (see Fig. 51).

It is rare to find a clear sharp type of aphasia, and it is easy to understand how seldom a lesion is limited to one centre or one path connecting one centre with another, and therefore in most cases several centres are damaged with results which are often difficult to interpret accurately. The fact that the left middle cerebral artery supplies practically all the great aphasia centres, and that a vascular lesion is one of the most common causes of aphasia, renders this statement easily appreciated. For a full description of this intricate subject the reader is referred to the classic works of Broca, Lichtheim, Wyllie, and many other writers.

Etiology.—Tumour, thrombosis, embolism, haemorrhage, gumma, or abscess can involve one or more of the cortical

areas, and certain of these lesions may cause damage to connecting paths. In addition it is probable that toxic agents may in some way produce temporary or permanent derangements, and it is only necessary to mention the effects of an excess of alcohol, morphia, or similar agent in upsetting the normal capabilities and co-relations of these centres with each other.

Pathological Anatomy.—In addition to the primary lesion, whatever that may be, degeneration occurs in tracts of nerve fibres severed from their trophic cells, and it should also be remembered that a comparatively small area of sclerosis may damage connecting paths between important centres without there being necessarily a lesion of the centres themselves.

The **Clinical Features** have already been described.

In examining an aphasic patient find out—

1. If he is right or left-handed.

2. By interrogating his friends, the state of his education as regards reading and writing.

3. His ability to hear, by asking him to touch his nose, put out his tongue, etc.

4. His ability to name objects shown to him, and if he cannot, determine whether this is due to visual or motor aphasia. If the former is suspected, prove that it is so by finding out if he knows what can be done with the object he is asked to name, and find out if he can name it by indirect suggestion.

5. His ability to understand spoken and written words. Can he speak connectedly or has he any memory defect?

6. His ability to read, write, repeat. Can he understand a written request?

Many terms have been used for different kinds of aphasia, but few have become really classic.

Paraphasia is employed to signify the condition in which the patient uses words absolutely senseless and inappropriate.

Enough has been said to show the difficulty often experienced in diagnosis; and in medico-legal cases the skill of the physician may be greatly taxed in deciding as to the amount of comprehension of which patients are capable for purposes of signing wills or other legal papers.

Prognosis.—Cases in which one centre alone is damaged may improve greatly, owing to the possibility of educating the corresponding centre on the opposite side, and it is hardly

necessary to state that in syphilis the use of suitable remedies often leads to marked amelioration. On the other hand, cases of cerebral tumour and cases of extensive thrombosis are generally grave.

The **Treatment** includes what might be said in connection with the management of tumours and other causal lesions. Where one centre alone is affected, and especially in young persons, the opposite side of the brain may be capable of being trained to do the work which ought properly to be conducted by the centres on the affected side. Re-education is always slow and even in the most favourable cases requires much patience and not a little skill.

Aphasia

A term which has been employed to mean a variety of different conditions mostly psychical. It signifies loss of the ability to carry out certain combined motor acts although he, the patient, is not ataxic nor aphasic. For instance he may know the name of a thing and be able to describe its uses but he cannot actually use it himself.

Dysarthria

By this term is implied speech defects due to lesions of the nerve cells governing the second motor neurones, their nerve fibres, and the muscles to which they go. All sorts of lesions, transient and permanent, in connection with the nose and mouth, pharynx and larynx, may also interfere with the pronunciation of words, and complete inability to articulate has been termed *anarthria*. Not infrequently it is only certain letters and sounds with which the patient has much difficulty. Amongst the common types of dysarthria are slurring of syllables, stumbling over syllables, undue separation of syllables and the reduplication of syllables, but the subject is a very wide one, and cannot be fully considered here.

3. Cerebral Congestion

1. *Acute Cerebral Congestion*

This is congestion of the vessels of the brain, and is in certain cases, an initial stage towards actual inflammation

of brain tissue: the term is also often employed to describe the condition which precedes the actual rupture of the vessel in the brain in persons who have suffered from a cerebral haemorrhage.

Etiology.—It is due to many causes, including excessive mental and physical strain, but perhaps most commonly to prolonged constipation, and it is associated with high arterial tension and arterial disease. Persons who have a high vascular tension, and who allow the bowels, kidneys, and skin to act defectively, render themselves liable to an attack of cerebral congestion. In a quite distinct class of cases, excessive mental overwork may induce an attack, even in those whose vessels are perfectly healthy.

Pathological Anatomy.—The nerve cells are over-stimulated by the excessive supply of blood, and not infrequently miliary or other aneurisms are found on the arteries in persons who are predisposed.

Clinical Features. Headache, complete unfitness for mental effort, excitement, and sleeplessness are common and in not a few cases, the phenomena suggest the probable onset of a cerebral haemorrhage, to which, indeed, this condition is often a premonitory stage. In some cases the patient may develop a regular attack of acute mania.

Prognosis.—Recovery under proper treatment, although not infrequently repeated attacks point to the probability of cerebral haemorrhage occurring sooner or later.

Treatment.—Absolute rest, with prompt attention to induce free action of the bowels, kidneys, and skin. Endeavour to reduce the high tension in the arteries, and curtail the dietary, especially in the direction of limiting the amount of butcher meat eaten and of alcohol consumed, and by so doing obviate the risk of future attacks in persons with arterio-sclerosis.

2. *Passive Cerebral Congestion*

The result of any obstruction to the circulation which interferes with the return of venous blood from the brain. Headache, unfitness for mental effort, and sleeplessness are all common clinical features, and the treatment consists in endeavouring to remove the obstruction: as this is often of

cardiac origin, it generally implies the administration of cardiac tonics, besides the use of purgatives and diuretics.

4. Cerebral Anaemia

Anaemia of the brain, whether from a deficient supply of blood or from the poor quality of the blood supplied.

Etiology.—It is not infrequent as the result of excessive haemorrhage. It is due to all the causes of profound anaemia, while it may also occur in aortic incompetence and in obstruction of arteries.

Pathological Anatomy.—The nerve cells are stimulated by the want of blood, and, probably, of the oxygen it should contain: where this condition persists, the cells cease to be able to discharge their functions.

Clinical Features.—In addition to the subjective and objective symptoms and signs of general anaemia, there are the following clinical features specially referable to anaemia of the brain. These are giddiness, more or less pain in the head, sometimes tinnitus aurium, dimness of vision, dilatation of pupils, sleeplessness, and, in very marked cases, syncope. There may be definite irritation phenomena in addition to those mentioned above, as, for example, muscular twitchings, and these may be followed by depression phenomena, including paralysis, coma, and death.

The **Prognosis** depends much on the degree and continuance of the condition.

The **Treatment** must obviously be directed to the removal of the cause where this is possible, and to the supply of more and better blood to the brain.

5. Cerebral Haemorrhage

This generally causes what is described as apoplexy although it should be remembered that an apoplectic seizure may also be the result of cerebral thrombosis or embolism. The vessels which give way are those which are least well supported, and have to sustain the greatest blood pressure. The antero-lateral nutrient arteries, and especially those of the left side, being in direct line with the left ventricle, are

more frequently affected than are any other arteries in the brain (see Fig. 52).

Etiology.— Cerebral haemorrhage is an affection of later life; it is more common after the age of 50, and especially in persons whose arteries are seriously degenerated. In all cases in which there is arterio-sclerosis, so often the result of alcoholism, syphilis, or overstrain, and in chronic Bright's disease, there is increased pressure in the arterial system as a whole, and the little nutrient arteries in the brain are apt to give way. In

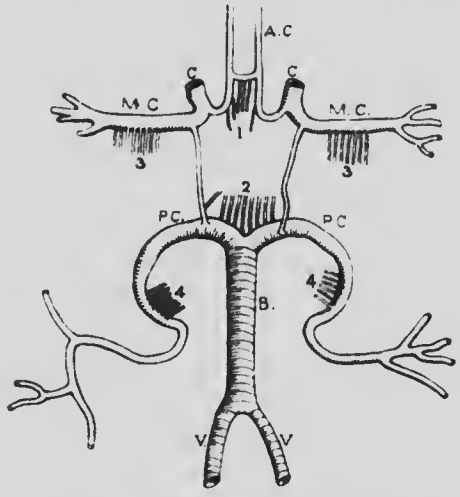


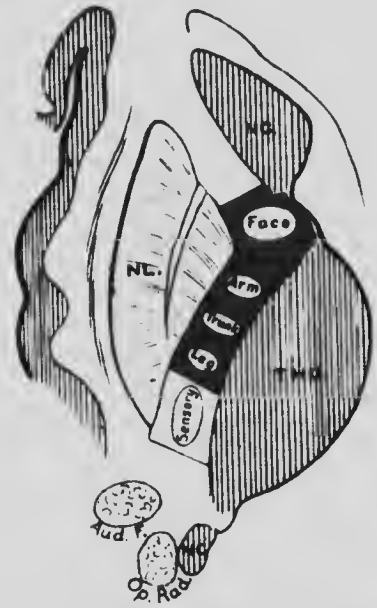
Fig. 52. Schematic representation of the Circle of Willis, with the nutrient Arteries supplying the region of the Basal Ganglia. C, Internal carotid Arteries; M.C. Middle cerebral arteries; A.C. Anterior cerebral arteries; P.C. Posterior cerebral arteries; B. Basilar artery; V, Vertebral arteries; 1, Antero-median nutrient arteries; 2, Postero-median nutrient arteries; 3, Antero-lateral nutrient arteries; 4, Postero-lateral nutrient arteries.

many cases minute miliary aneurisms form on the nutrient arteries, and it is to the rupture of one of these that cerebral haemorrhage may be due. There are many cases of haemorrhage in which no miliary aneurism is present, but arterial degeneration may lead to weakening of the vessel wall, and rupture may occur quite as readily as in cases where miliary aneurisms have developed. Persons of what may be termed apoplectic habit, and those whose vessels have suffered in the way just described, may readily bring about the rupture of a small miliary aneurism by any unaccustomed or severe strain, such as straining at stool, or the effort necessary for catching a train or tram-car, or even stooping to tie a boot-lace. Excessive

drinking, excitement, and especially anger, may also cause cerebral haemorrhage.

Haemorrhages are, however, sometimes associated with endocarditis, especially where emboli of an infective nature find their way into the cerebral arteries, and haemorrhages may also occur in leucocythaemia, pernicious anaemia, purpura, and scurvy.

Pathological Anatomy.—*The Site of the Haemorrhage.*



Internal Capsule—showing position of Motor (red) and Sensory (yellow) f
NC. Caudate nucleus. NL. Lenticular nucleus; THO. Optic thalamus; A
Auditory fibres. Op. Rad. Optic radiation.

A. *Intra-cerebral Haemorrhage.*—(1) The most frequent position is in the outer part of the lenticular nucleus, and the haemorrhage is due to rupture of the so-called "artery of cerebral haemorrhage," which is one of the largest and most posterior of the lenticulo-striate arteries (see Fig. 54). The effused blood may be small in amount, and may merely press on the internal capsule, generally involving the genu or knee and the posterior half of the internal capsule, and thus causing pressure on the motor fibres, a pressure which may be merely temporary if the blood is absorbed. In other cases the haemorrhage is greater, the brain tissue is torn through, and the blood may

burst into the lateral ventricle, or may make its way externally towards the cortex. Haemorrhage more frequently occurs on the left side of the brain owing to the direct line of blood pressure between the heart and the antero-lateral nutrient arteries on that side.

(2) More rarely haemorrhages occur from other nutrient arteries. Sometimes one of the antero-median nutrient arteries may rupture and cause haemorrhage into the frontal lobe.

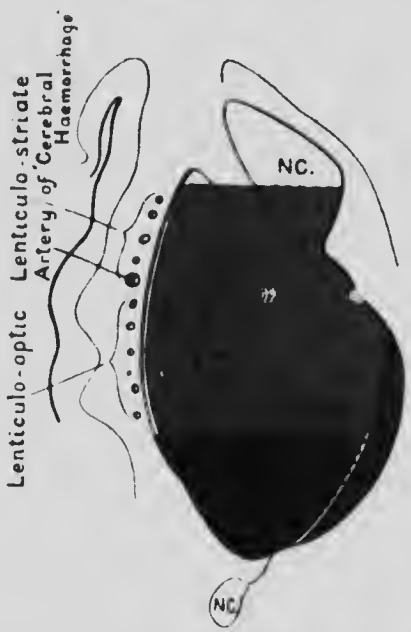


Fig. 54—Internal Capsule and Basal Ganglia, showing distribution of blood supply by the Nutrient Arteries. Area supplied by Antero-median in yellow, Antero-lateral in red, Postero-median in green, and Postero-lateral in blue. The internal and external antero-lateral nutrient arteries are shown, and the Artery of Cerebral Haemorrhage which is one of the largest of the latter group.

(3) Haemorrhage may also occur into the cerebellum, and usually proceeds from the superior cerebellar artery. It is met with in young persons generally under the age of 25, and the blood may burst into the fourth ventricle; in any case death ensues with great rapidity.

(4) Ventricular Haemorrhage.—This is rarely of primary origin, and is usually due to haemorrhage into the cerebral substance which has made its way into the ventricles. It may, however, occur from rupture of vessels of the choroid

plexus. Ventricular haemorrhage is said to be not uncommon in early life, and has occurred as the result of pressure on the head during birth. The haemorrhage may be limited to one ventricle; generally it is found in both lateral ventricles unless the channel of communication is closed by pressure, and it may fill the whole ventricular system.

5. Small haemorrhages scattered throughout the cerebral substance are met with in cases which may be described as toxic. They may be seen, for example, in patients who have died from hyperpyrexia of rheumatic origin.

B. *Meningeal haemorrhage* may be secondary to intra-cerebral haemorrhage, and the blood is very frequently subarachnoid in position, or may rupture through the pia-arachnoid and become subdural. In fracture of the skull owing to laceration of the brain, and generally as the result of contrecoup, subarachnoid haemorrhages may occur on the opposite side of the brain from the fracture. Aneurisms of one of the larger intracranial arteries may rupture and cause meningeal haemorrhage, and, lastly, meningeal haemorrhage is not infrequent from injury sustained during labour.

The blood, once effused, tends to become absorbed, and if it is in the brain tissue, a cyst may result, surrounded by a definite wall and containing haematoidin pigment. This definite wall is due to secondary and localised inflammation which is very generally a sequel to an intra-cerebral haemorrhage. In the mildest cases a somewhat pigmented scar may alone be noted.

One inevitable result of a cerebral haemorrhage where certain tracts of brain tissue have been damaged, is degenerative change, and therefore, where the motor part of the internal capsule is destroyed, descending degeneration occurs in the corresponding pyramidal tracts in the cord (the direct pyramidal on the same side and the crossed pyramidal tract on the opposite side from the lesion). Further, haemorrhage in the frontal lobe may cause degeneration of fibres joining the frontal lobe with the pons, and when the cerebellum is destroyed, degeneration of the brachium conjunctivum may result. A haemorrhage destroying the occipital lobe may produce degenerative changes in the optic radiation.

One important cause of intra-cerebral haemorrhage is unquestionably miliary aneurisms, and it is difficult to account

for their existence. They are about the size of a pinhead, and are probably associated with arterio-sclerosis; but they also depend on the fact that in brain tissue the blood-vessels are badly supported. In many cases of cerebral haemorrhage occurring after middle life these miliary aneurisms are present. The exact connection of syphilis with the presence of these aneurisms is doubtful.

The smaller multiple haemorrhages seen in toxic conditions and also found in purpura haemorrhagica, pernicious anaemia, and other diseases, are due to diapedesis of red blood corpuscles without actual rupture of the vessel walls.

Clinical Features.—*Premonitory Symptoms.*—Usually an apoplectic stroke is sudden, but not very infrequently the patient has suffered from what may be called *warnings*, which include a feeling of fulness in the head on stooping, ringing in the ears, or a sense of mental confusion, especially if the patient overeats or permits constipation to be present without treatment. Occasionally a localised numb feeling is felt in the limbs about to be paralysed, or choreic movements may appear, called prehemiplegic chorea.

The Apoplectic Seizure.—In many cases the attack is sudden, and as a rule the patient rapidly loses consciousness, although in other cases the coma may be more gradual. There is sometimes a definite cry before the patient sinks to the ground. The face may be blue, reddish, or ashen-grey; the pupils are generally dilated, and if there is deep coma they do not contract to any stimulus. Should the haemorrhage irritate in some way the part of the nucleus of the third nerve containing the centre for the sphincter pupillae muscles, then the pupils are pin-point. The respirations are typical, the breathing being stertorous, slow, and noisy, and the paralysed cheek is seen to be more puffed out than the one on the unaffected side; the pulse is found to be slow, full, and of high tension; the temperature at this time is generally normal. Although the patient is deeply unconscious, it is easy to tell which is the affected side by examining the limbs. The arm and leg on the opposite side from the lesion drop like a stone, whereas on the healthy side this absolute relaxation is not present. The abdominal and cremasteric superficial reflexes are absent on the affected side. Often the bladder and bowels are evacuated during the attack. There are no convulsions, but

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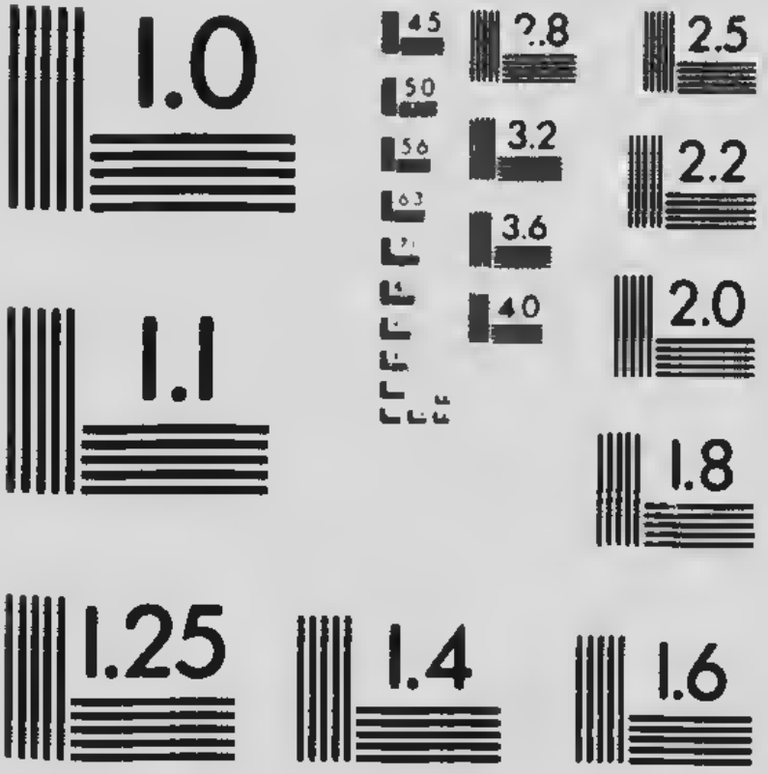

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on examining the eyes it is generally noted that the patient squints upwards and outwards towards the side of the haemorrhage: in other words, he looks towards his lesion. When early rigidity or convulsions occur, the deviation may be towards the opposite or sound side. The temperature generally falls 1 or 2 during the first hour and then rises to normal.

In a haemorrhage producing hemiplegia, we find on examining the face that it is not completely paralysed, that the muscles governing emotional movements are unaffected, but that the patient cannot quite close the eye or draw down the mouth on the affected so well as on the healthy side, and the tongue, when protruded, deviates towards the paralysed side, owing mainly to the unantagonised action of the normal genio-hyoglossus muscle. Interference with speech varies; it may be slight or very marked.

The arm is more paralysed than the leg. This is partly due to the fact that the leg muscles are supplied by fibres from both sides of the brain. The leg may even escape, and very rarely indeed is the leg more affected than the arm. In the *arm* the most affected muscles are those opposing the thumb, those rotating the arm outwards, and the openers of the hand. In the *leg*, the most affected muscles are the flexors of the leg and the dorsal flexors of the foot, and these muscles may remain paralysed long after the other leg muscles have begun to recover.

There is no muscular wasting unless from want of use, because the lesion is one of the first motor neurone, and not of the second, the trophic centres for the muscles remaining unaffected. The tendon reflexes are abolished immediately after the fit, but in a few days they become markedly exaggerated on the affected side and ankle-clonus is noted. The Babinski sign (page 583) is positive in the affected foot. The superficial reflexes on the paralysed side are diminished or lost.

An apoplectic shock may occur during the night, and the patient may waken to find himself paralysed on one side; or, as noted under the etiology, it may be the result of strain, such as straining at stool, or tying a boot-lace, or hurrying to catch a train. In the case of a small haemorrhage, hemiplegia may occur without any loss of consciousness, although this is certainly rare. On the other hand, where coma gradually

deepens to profound unconsciousness the term *ingravescent* is applied to the condition.

Later Changes.—Sometimes within two days after a seizure febrile reaction occurs, and the patient, who may have wakened up out of the coma, once more becomes comatose, and may remain in this condition for a week or longer, and possibly death may occur without any improvement. The febrile reaction depends probably on inflammatory changes round the site of the haemorrhage, and is by no means invariably present and it is during this period that what is termed *early rigidity* is apt to develop in the paralysed limbs, while trophic changes, causing bed-sores, may also make their appearance. The temperature may rise to 102° or 103° F., and the patient becomes extremely restless until the more acute inflammatory manifestations have passed off.

Recovery generally begins in the leg, and the face improves before the arm, while the patient finds it possible to govern the shoulder movements before he regains control of those of the lower arm.

Late rigidity is apt to appear in muscles and limbs in which recovery has ceased: the arm may become permanently flexed at the elbow and the fingers flexed upon the hand. Flexion is the result of this late rigidity, and with the flexion there is often much pain, especially in the joints, which have become more or less rigid. The late rigidity is largely due to secondary degeneration, and associated with it there is exaggeration of the tendon reflexes. The muscles of the affected limbs which do not recover their movements tend to waste as the result of inaction, and it is remarkable how, in some hemiplegic limbs, the skin becomes glossy, the nails of the affected fingers and toes brittle, and the superficial temperature much below normal. Sometimes acute atrophy occurs, and probably this implies some involvement of the cells in the anterior horn of the cord. Amongst the more unusual results of hemiplegia are tremors and choreiform movements in the affected limbs, the latter type of movements being commonly called athetosis, but athetosis is more common in hemiplegias dating from early childhood.

Do not despair if unconsciousness should persist for several weeks, so long as it does not become deeper, because even after six weeks considerable improvement may occur in the paralysed

limbs. The gait of a hemiplegic patient, if the leg does not completely recover, is very typical. As a rule the affected limb describes a semicircle, so as to prevent the toe of the boot catching on any irregularity of the ground.

Sensory phenomena are rare, and depend on the lesion involving the posterior third of the posterior limb of the internal capsule, when as a rule the hemianaesthesia is also associated with hemianopsia (from the close relationship of the fibres of the optic radiation. See Fig. 53).

One of the most distressing features associated with certain cases of hemiplegia is the mental change which may occur. A strong-minded man may become emotional like a child, and excessive irritability is frequently present in severe cases, rendering the nurse's post anything but a sinecure.

The typical description given above is that of the most common form of intra-cerebral haemorrhage; but the phenomena resulting from a haemorrhage depend on the site of the lesion. As already noted, if the posterior portion of the posterior half of the internal capsule is involved, hemianaesthesia results, but the haemorrhage may be in many other positions.

If the haemorrhage is in the crura cerebri, there may be crossed paralysis. If the artery to the nucleus of the third nerve ruptures, then the face, arm, and leg on the opposite side, and the third nerve on the same side are affected, and there is often some change also as regards sensation, while if the optic tract or the external geniculate body is involved, there may, in addition, be hemianopsia.

If the haemorrhage is in the pons, there may be paralysis of all four limbs with pin-point pupils, hyperpyrexia, vomiting, and convulsions, as noted under cerebral localisation, or there may be crossed paralysis depending on the position of the lesion, which must in that case be unilateral (see Fig. 50). In this way the 5th, 6th, and 7th nuclei or nerves may be affected. If the haemorrhage is in the posterior or upper part of the pons, then sugar or albumin sometimes appears in the urine.

If the haemorrhage is primarily or secondarily intra-ventricular, and is of large amount, there is sudden and deep coma with abolition of reflexes and complete relaxation of muscles. Death is generally rapid. The temperature generally rises before death.

If the haemorrhage is in the cerebellum, it usually

proceeds from the superior cerebellar artery, and is met with in young persons generally under the age of 25. The blood may burst into the fourth ventricle: there is deep unconsciousness but no hemiplegia, unless the pons is affected. Persistent vomiting, vertigo, skew-deviation of the eyeballs, and sometimes glycosuria may be present. Should the haemorrhage occur into the *lateral lobe*, there is marked nystagmus on movement of the eyes towards the affected side, and a typical cerebellar gait with a tendency to fall to the side of the lesion. In cases of haemorrhage into the *middle lobe* a tendency to opisthotonos has been noted. Small haemorrhages may occur in the cerebellum without causing any unconsciousnes

If the haemorrhage is into the meninges, there is sudden pain and often convulsions, which may be unilateral. Should the haemorrhage be basal, there is deep coma, pin-point pupils, hyperpyrexia, and heart and respirations soon fail.

Diagnosis.—In most cases there is little difficulty with regard to diagnosis. The evidence of arterio-sclerosis with hypertrophy of the left ventricle, and possibly a history suggesting cirrhosis of the kidneys, greatly assist in clearing up the case.

Where the onset is gradual, it is often difficult to say whether the lesion is a haemorrhage or a *thrombosis*, but haemorrhage is more likely to occur when the patient's cardio-vascular system corresponds to the description just given.

If the patient is comatose when seen, the lesion may be haemorrhage, *embolism*, or *thrombosis*, and in addition there are other possibilities which must not be forgotten. These include *alcoholic stupor*, *opium poisoning*, *uraemia*, and *diabetic coma*. Embolism and haemorrhage are generally accompanied by unconsciousness, while thrombosis, with a longer and somewhat suggestive history of premonitory phenomena, is more typically ingravescent, and not necessarily accompanied by coma. In cases of embolism we expect to find endocarditis with vegetations and clinical phenomena associated with that disease. In thrombosis the circulation is usually enfeebled.

Decide whether there is hemiplegia by carefully comparing the limbs and the face on the two sides. Opium poisoning produces pin-point pupils, and is simulated by a pontine haemorrhage; but convulsions are usually associated with a

haemorrhage into the pons, and the heightened temperature is often a help. In opium poisoning the breath, and very probably the contents of the stomach, may greatly assist in the diagnosis, and similarly, in eliminating alcoholic poisoning, a careful investigation should be made of any stomach contents obtainable. The urine should be tested carefully for albumin in cases in which uraemia is a possible diagnosis. It is more unlikely that diabetic coma can be mistaken, but if the urine is tested for sugar, and acetone or its derivatives, there is little chance of error.

It is important to remember that a patient may be both drunk and suffering from a cerebral haemorrhage, and rather to err on the safe side than to risk making a definite diagnosis. In cases in which there is a subarachnoid effusion, however produced, and which has come on *suddenly*, the writer has noted retinal haemorrhages in the eye of the affected side, and this may sometimes assist in forming a diagnosis.

Prognosis.—In ordinary cases of cerebral haemorrhage from one of the lenticulo-striate arteries much depends on the amount of blood effused, and also whether there is any recurrence of the effusion during the inflammatory or reaction stage. In favourable cases consciousness is either not lost or soon regained, and the paralysis, although possibly complete in the arm, is only partial in the leg, while within a matter of several weeks the patient regains control over many muscles originally powerless. In these cases a slight trace of the hemiplegia usually persists, but contracture is rarely marked. On the other hand, where the patient remains comatose for a long time, or where there is almost complete paralysis of the leg as well as the arm, persisting without improvement for a considerable time, recovery can only be partial, and contracture is nearly always present and marked. Grave phenomena associated with a case of haemorrhage are:—deepening coma, prolonged Cheyne-Stokes type of breathing, a considerable rise of temperature during the stage of reaction where it occurs at all, the return of unconsciousness with this reaction stage, and the development of acute bed-sores.

If the haemorrhage is situated in the cortex, it is very often due to injury, and recovery without any contracture is probable. Meningeal haemorrhage if not basal may be recovered from, and especially if by operative interference

the clots can be removed. Infantile meningeal haemorrhage is apt, however, to produce imbecility with spastic diplegia. Extensive haemorrhages into the corona radiata and rupture of the blood into the ventricles nearly always prove rapidly fatal.

Treatment.—The patient should at once be placed at rest in bed, care being taken to loosen garments about the neck and chest, and if possible the head should be kept high. The next thing to be done is to ensure free evacuation of the bowels, which may readily be effected by placing on the tongue a minim of croton oil mixed with one or two drops of glycerine. Then decide whether it is advisable to perform venesection. The object of this procedure is to endeavour to lower the blood pressure, and so to arrest haemorrhage if it be still continuing. An ice-bag may be placed over the head and hot bottles to the feet. In cases in which, from the deepening coma, the haemorrhage is probably progressing, an attempt may be made to compress or to ligature the carotid artery, but the latter operation should only be carried out in serious cases.

The patient will probably be confined to bed for a long time, and therefore it is wise to procure a water-bed, and to do all in one's power to obviate bed-sores by carefully cleansing the skin, and endeavouring to harden any part liable to pressure. This is efficiently managed by sponging the skin with spirit after careful ablution with soap and water. Always prevent the skin from being kept moist or the bed-clothes being soiled by discharges. The bladder should receive careful attention from the first, and if necessary an aseptic catheter should be regularly passed. The affected limbs should be wrapped in wadding, and massage should not be permitted until an interval of a week or ten days has elapsed.

No excitement should be allowed, and stimulants, unless absolutely necessary, are contraindicated. As far as possible the blood pressure should be kept low, and this can be efficiently carried out by free purgation, and sometimes by the administration of potassium iodide. During the stage of reaction it is specially desirable to endeavour to control the temperature, and to diminish the restlessness which may lead to further haemorrhage, and during this stage sedatives are sometimes necessary.

After about ten days, begin with gentle massage for brief periods of time, and go on later to faradic stimulation. For late rigidity, massage and passive movements are probably as efficacious as anything else, and careful but not excessive use of galvanism. It is worth while, during the stage when the effused blood is being absorbed, to try the effect of potassium iodide in regular doses, although it is unlikely that it will cause much increase in the natural processes.

6. Cerebral Embolism, or Embolism of Cerebral Arteries

Etiology.—The embolus may originate from a vegetation or deposition of fibrin on either the mitral or aortic valves or sometimes from a dilated left auricle, in the appendix of which clotting has occurred. Much more rarely does the embolus proceed from clot on the wall of an artery or aneurism. The majority of cases of cerebral embolism are the result of heart disease in which vegetations occur on valves chronically affected, and in the less frequent cases of ulcerative endocarditis, cerebral emboli are naturally not uncommon.

Pathological Anatomy.—Emboli more frequently enter one of the middle cerebral arteries, and often the left. There may be a limited amount of anastomosis, provided the embolus does not obstruct an end artery, but in most cases where, for example, several branches of the middle cerebral are blocked, a large area of softening inevitably occurs. There may be haemorrhagic infarction owing to the part deprived of blood becoming infiltrated from surrounding vessels, or on the other hand there may be white softening, commonly called anaemic infarction. In both cases the brain substance becomes soft and diffluent. There is a considerable amount of serous infiltration into the area, and if the embolus contains suppurative organisms an abscess may develop. All shades of colour may be seen in fairly old infarctions, from brownish-red to yellow to white, depending much on the amount of, and change in, the blood pigment present: but the typical yellow and white areas of infarction are more commonly the result of thrombosis than of embolism.

It sometimes happens that an embolus retracts, and the

circulation is partly opened up into the affected territory, but on the other hand it is not uncommon to find a considerable amount of thrombosis secondary to the lodgment of a large embolism.

The **Clinical Features** depend on the vessel blocked by the clot. As a rule there is no premonitory symptom, but there should be in most cases some evidence of preceding heart disease. The onset is sudden and there may be coma, although, where a smaller vessel is blocked, consciousness may not be lost, and there is more frequently hemiplegia without coma than is the case in cerebral haemorrhage.

If the whole of the *middle cerebral artery* is involved with most of its five branches, there is hemiplegia, and should the lesion occur on the left side of the brain, there is motor aphasia, word-deafness, and sometimes word-blindness: but the sensory forms of aphasia are more likely to occur in thrombosis than in embolism. If a *cerebral artery* is involved, it is the left one which generally suffers, and the phenomena are suggestive of acute bulbar paralysis.

If the *basilar artery* is blocked, there is generally bilateral paralysis of the four limbs, and bulbar phenomena may also be present along with clinical features suggesting a pontine lesion.

If the *posterior cerebral artery* is blocked, there will be hemianopsia and very generally hemianaesthesia.

If the *anterior cerebral* is blocked, there may be no phenomena, or there may be hebetude with intellectual dulness.

The reader is referred to the topical diagnosis of brain lesions for a fuller account of the phenomena met with in each region. It should be stated that with the exception of the middle cerebral arteries, emboli rarely affect other cerebral vessels, and that, in them, thrombosis is by far the more common lesion.

The **Diagnosis** has to some extent been treated of under cerebral haemorrhage, but it may be mentioned that an examination of the heart should, in most cases, demonstrate the presence of endocarditis.

The **Prognosis** is unfavourable in severe cases, and the **Treatment** must be on general lines.

7. Cerebral Thrombosis

Etiology.—It is generally the result of disease of the vessel walls, as in atheroma and endarteritis obliterans, but it may also occur in cases where the circulation becomes enfeebled, as in old age or in typhoid fever, and in many cases there is a combination of marked atheroma together with debility.

Pathological Anatomy.—A description of infarction has already been given under the head of embolism. It is probable that in thrombosis the anastomotic supply of blood to the affected area will not be so free as in embolism, and the patch of softened brain is usually yellow or greyish-white in colour and milky in consistence. Haemorrhage into a patch is not so common as in embolism.

All arteries of the brain may be involved by thrombosis, and in many cases numerous small patches of softening occur, which may be in silent areas, before a more important part of the brain is affected, giving rise to definite paralytic phenomena. In the brains of old persons, yellowish patches of softening, commonly called "*plaques jaunes*," may be seen in the grey matter, and as many as 20 or 30 may be counted in a case in which no very serious clinical phenomena have resulted from their development.

Clinical Features.—There are often premonitory symptoms such as vertigo or giddiness, and headache, and very frequently before an important area of brain is affected, the patient may have experienced numerous attacks of giddiness without loss of consciousness or experiencing any other trouble. Sometimes the patient, if walking on the street, may have to hold on to the railings, or even sit down for a few moments before he recovers.

When a serious thrombosis occurs, there are generally repeated attacks which lead up to the more extensive involvement of vessels. There may be gradually developing hemiplegia, one limb being affected after another, sometimes with a transient improvement in the affected limbs, in other cases without any marked recovery of movement. When a large vessel is blocked, such as the whole of one of the middle cerebral arteries, coma supervenes. The attack may occur during the night, and the patient wakens to find that he is hemiplegic

and if the lesion be on the left side of the brain, he may have motor aphasia, or some form of sensory aphasia.

Where the lesion is acute, by which is meant that a large vessel is suddenly and completely blocked, the condition closely resembles cerebral haemorrhage, and in all cases in which there are repeated attacks, or a large vessel becomes thrombosed, the patient's memory and mental powers suffer seriously.

Should the *internal carotid artery* be thrombosed, there is hemiplegia with deep coma and rapid death.

If the *posterior cerebral artery* is occluded, there is hemianopsia and hemianaesthesia and sometimes tremor of the limbs on the opposite side of the body.

Where the *basilar artery* is affected, there is a bilateral paralysis, but a good deal depends on the site and extent of the thrombosis, and it is possible, for instance, to have paralysis of the third nerve with a crossed hemiplegia.

Where the *anterior cerebral artery* is thrombosed, there is generally hebetude but little evidence of any motor or sensory paralysis.

Diagnosis.—A careful examination should be made of the condition of the peripheral vessels so as to ascertain the amount of atheroma present, and a history of syphilis is suggestive of endarteritis obliterans. There are cases in which it is extremely difficult to differentiate between thrombosis, *haemorrhage*, and *embolism*, but in most cases of thrombosis in older patients there have been numerous previous attacks of milder nature, while in haemorrhage and embolism the attack is more sudden and unexpected. In thrombosis, the patient not loses consciousness unless a large vessel is thrombosed. [illegible]orrhage, consciousness is generally lost, while in em- [illegible]oma more frequently supervenes than in thrombosis [illegible] attempt should be made in every case to decide with [illegible]to the diagnosis, because the treatment for haemorrhage [illegible] different from that for thrombosis.

[illegible]**ognosis** is bad in serious cases, because repeated [illegible]pt to occur and it is only in mild cases, or in the [illegible]ccur as the result of the debility associated with [illegible]r other fever, and in syphilitic cases, that a fairly [illegible]prognosis is warranted.

Treatment.—While rest is indicated, and just as absolute

rest as in a case of haemorrhage, stimulants should be gi
and probably ammonia and alcohol; further, cardiac tonic
such as strophanthus and digitalis, will be called for. Nitro-
glycerine has been suggested as a possible remedy for helpin
to open up a blocked vessel. In every syphilitic case giv
potassium iodide and sometimes mercury, and persist with thi
treatment for a long time. The treatment of the paralysed
limbs is exactly the same as in cases of hemiplegia the result
of haemorrhage.

8. Thrombosis of the Cerebral Sinuses and Veins

Etiology.—Thrombosis of the sinuses may be *primary*
or *secondary*.

The *primary* cases are due to any debilitating disease, an
blood condition in which thrombosis is apt to occur in veins
and in children suffering from a wasting disease such a
continuous and severe diarrhœa.

The *secondary* cases depend upon an infective organism
focus of inflammation in the neighbourhood of the sinus
vein, and the most common causes are middle-ear disease
erysipelas, parotitis, and tubercular caries of bone.

Pathological Anatomy.—Thrombosis is readily induce
especially in certain sinuses, such as the great longitudina
sinus, owing to its anatomical and physiological condition
Sometimes inflammation of the wall of a sinus is the direc
cause. The clot tends to become adherent and to organise.

The **Clinical Features** vary considerably according to th
sinus involved. There are generally irritation phenomen
often followed by depression or paralytic phenomena.

If the *longitudinal sinus* is thrombosed, there may be
phenomena, and the condition may only be found accidental
at a post-mortem examination, but in certain cases headach
mental apathy, possibly convulsions, vomiting, and drowsine
have all been reported to be the results of thrombosis
this sinus.

Thrombosis of the *lateral sinus* may also occur in debil
as a primary lesion, but is more commonly the result
middle-ear disease. There is generally a painful swelli
behind the ear in the region of the mastoid vein which em
this sinus; the internal jugular vein may be empty

blood and sometimes, in septic cases, the whole side of the neck on the affected side is swollen and painful. While headache, drowsiness, delirium and coma, muscular twitchings, nystagmus, strabismus and sometimes optic neuritis may be present.

Where the thrombosis in any simple or pyæmic case is secondary pyæmic abscesses will be met with in the lungs and elsewhere, and the clinical features are pyæmic in type.

Treatment. In simple primary cases, rest in bed with vigorous stimulation is indicated, and probably an attempt should be made to prevent obstruction to the flow of blood in the jugular veins by keeping the head almost flat in bed, in the hope that thereby the occlusion of vessels due to the bending of the neck may be prevented. Stimulants such as ammonia and alcohol may be given, and in certain cases potassium iodide and mercury have been recommended.

9. Aneurism of the Cerebral Arteries

This does not include miliary aneurisms, but refers only to larger aneurisms generally found on one of the branches of the circle of Willis.

Etiology.—It is not common. It is more frequent in men than in women, and generally at an age when aneurism elsewhere tends to develop. It is due to weakening of the arterial wall, and may be associated with syphilitic disease or possibly endarteritis, atheroma, or embolism. Osler's list of twelve cases includes one on the internal carotid, five on the middle cerebral, three on the basilar, and three on the anterior communicating. In other statistics nearly one-third of the cases have been found on the middle cerebral artery. In two cases seen post mortem by the writer, the anterior cerebral was involved by aneurisms about the size of a large pea.

Clinical Features.—The aneurism may rupture and so cause death, or it may act as a tumour and press upon various important structures. It may give rise to optic neuritis, or to paralysis of some of the cranial nerves, and as a rule its termination, where death is due to the aneurism, is by rupture. Occasionally audible murmurs may be heard on auscultating the skull, but in most cases the clinical evidence of the existence of an aneurism depends on interference with cranial nerves.

10. Intracranial Tumours

Tumours may develop from the brain, from the membranes, or from the bones of the skull, while aneurisms and cysts may produce the same symptoms as do solid intracranial tumours.

(1) *Gliomata* are probably the commonest tumours originating in the brain substance itself. They develop from the neuroglia and usually infiltrate the cerebral tissues, having therefore no definite wall, and in process of their growth a long time may elapse before the nerve fibres passing through the tumour area lose their function.

(2) *Sarcomata* more commonly develop from the membranes, and sometimes from the bone or periosteum of the skull. This is one of the largest and most rapidly spreading intracranial tumours, and numerous nodules may be found throughout the brain substance.

(3) *Tubercular tumours* vary greatly in size, and are most commonly found about the base of the brain or in the cerebellum. They may be single, but more generally are multiple, and in size vary from a pea to a walnut. They are frequently met with in young patients, are apt to undergo degenerative changes, and almost always have a sharply defined outline.

(4) *Gummata* are generally superficial, and are usually attached to the membranes; they are often single, but may be multiple, and as they sometimes involve the region of the pons both 3rd nerves are apt to be affected.

(5) *Carcinoma* is very rarely of primary origin in the brain, but secondary tumours may be met with where the primary lesion is in the breast or some other part of the body.

There are many other tumours, amongst the most interesting being *psammomata*, which are probably of sarcomatous nature. *Endotheliomata*, *angiomata*, and other rare tumours, simple and malignant, are occasionally met with. *Hydatid cysts* and the *Cysticercus cellulosae* are sometimes found in the brain, but more frequently in foreign countries than in our own.

Cysts, the result of haemorrhage and *porencephaly*, which is sometimes of developmental origin, may cause certain of the phenomena suggestive of tumour.

Clinical Features.—The following typical phenomena are met with in cerebral tumour:—

General Symptoms.—(1) Headache, dull and aching, or sharp and lancinating, and sometimes indicating the site of the tumour. Not infrequently, when the tumour presses on some sensory nerve, there are neuralgic pains shooting along the line of the branches affected and a neuralgia which resists treatment by ordinary remedies.

(2) Cerebral vomiting is a typical though not a constant clinical feature. By the term *cerebral* is meant vomiting not directly connected with the ingestion of food, but often associated with a special paroxysm of headache.

(3) Vertigo or giddiness, varying considerably in degree, is sometimes marked when the patient rises from the prone position or when he turns suddenly when standing up.

(4) Optic neuritis occurs in four-fifths of all cases of cerebral tumour. It is almost constantly present in cases in which the tumour is situated in the cerebellum, or when it involves the corpora quadrigemina, while it is less common in cases in which the tumour is limited to the pons or medulla. It is more frequent where there is a glioma, sarcoma, gumma, or cerebral cyst, and it is less common in tubercular tumours.

Less frequent phenomena are slowness of pulse and sometimes respirations, convulsive seizures, fainting attacks, wasting and bodily weakness, and occasionally mental manifestations, which are often very typical. As the tumour grows, the patient tends to become dull, sleepy, and slow of thought and speech, and the memory may fail. It is much more unusual to find mental excitement even of a transient nature.

Focal Symptoms.—The reader is referred to the description already given of cerebral localisation, p. 705, and to the brief synopsis of symptoms of tumours in special regions given later. In every case of supposed cerebral tumour the calvarium should be palpated and also percussed, because, where the tumour is superficial, a considerable amount of pain is elicited on pressure over it.

It not infrequently happens that the tumour presses on the veins of Galen, causing distension of the ventricles of the brain. This is more likely to occur where the tumour is situated near the basal ganglia. As a result the evidences

of pressure on the cerebral cortex and the important centre in the brain become more marked, and eventually the irritation phenomena, which include vomiting, giddiness, and headache, are replaced by coma and slowing of the pulse, suggestive of the paralytic stage being reached. There is no doubt that choked disc is more common where there is great intra-cranial pressure, but it is impossible in every case to deduce from the examination of the eyes satisfactory evidence of pressure on the veins of Galen. In children there is sometimes definite separation of the bones of the skull, but in the adult this is almost impossible. The tumour, if near the base of the brain, frequently presses on one or more of the cranial nerves, and this may aid in its localisation.

Towards the termination of a case of cerebral tumour there is usually greatly increased intra-cranial pressure resulting in coma, which may, however, be relieved by operation undertaken for the purpose of reducing the tension within the skull.

Tumours of Broca's convolution.—If Broca's convolution is involved on the left side of the brain, motor aphasia develops early, and gradually becomes more and more complete.

Tumours of the left temporal lobe may produce word deafness, amnesic aphasia and paraphasia, with not infrequently tinnitus and in certain cases hemianopsia, hemiplegia, and hemianaesthesia. In certain cases smell and taste are also affected.

Tumours of the left inferior parietal region may cause alexia and agraphia.

Tumours of the superior parietal region may cause loss of the stereognostic sense—or astereognosis—by which is meant inability to recognise objects by their shape.

Tumours in the grey matter of the cerebral hemispheres are apt to cause hemiplegia developing gradually and sometimes incompletely. There may be some signs of cortical irritation such as athetosis, tremor, and sometimes Jacksonian epilepsy. Generally speaking there is definite spastic paralysis with plantar extension of the great toe. There is sometimes hemianaesthesia and hemianopsia.

Tumours of the cerebellum may for a time remain latent as far as focal symptoms are concerned, but there is apt to be headache, vomiting, nystagmus, vertigo, ataxia of the cer-

bellar type (although this depends to some extent upon the site of the tumour), rigidity of the neck, and skew-diplopia.

Tumours involving the cerebello-pontine angle.—These tumours only *secondarily* involve the cerebellum and the pons by pressure. They grow sometimes from the 8th, sometimes from the 5th or 7th cranial nerves. The typical signs of intracerebral tumour are late of appearing, but headache is generally the first of these, and optic neuritis either does not appear at all or is very late of doing so. The chief symptoms depend on the involvement of the cranial nerves by the tumour, and only secondarily do we find evidence of pressure on the lateral lobe of the cerebellum and the pons. There may be more or less paralysis of the 6th, the 5th (in particular the sensory divisions), the 7th, and the 8th nerves, and with this there is often much tinnitus aurium, and giddiness.

On the side of the tumour there is some paresis with ataxia, and often atonia due to pressure upon the lateral lobe of the cerebellum. On the opposite side of the body from the tumour there may be a degree of spastic paralysis due to pressure on the pyramidal fibres prior to their decussation.

The gait is ataxic and also spastic: ataxia being more marked on the side of the tumour. There are no sensory changes in the body or limbs, and as one might expect the tendon reflexes are exaggerated on the side which is spastic. The abdominal reflexes are diminished on the spastic side, and there is typical plantar extension.

Diagnosis.—It is necessary to determine whether there is a tumour, and if so, what is the nature of the tumour and where it is situated. The presence of the general phenomena suggesting cerebral tumour, together with any evidence indicative of its site, helps greatly in the diagnosis. The *albuminuric retinitis* met with in Bright's disease differs considerably from the papillitis usually seen in cerebral tumour, and there is no particular reason why, in cerebral tumour, albumin should be present in the urine unless the tumour involves the region of the 4th ventricle. Sometimes cases of *general paralysis of the insane* closely resemble cerebral tumour, but the history of the case and the absence of optic neuritis should, as a rule, prevent mistake, and in *hysteria* it is rare to find the group of cardinal clinical features so commonly met with in intracranial tumour.

The physician must depend on the knowledge he possesse
of the functions of different parts of the brain to aid in th
diagnosis of the position, but with regard to the nature of th
tumour one is often greatly at a loss. In younger patient
tubercle, but also glioma and sarcoma are common, and th
diagnosis favours glioma or sarcoma if optic neuritis is presen
In older patients, and those with a syphilitic history, gumm
is by no means improbable.

The **Prognosis** is extremely unfavourable, with the ex
ception of syphilitic cases, and those in which the foc
phenomena are very distinct and suggest tumours which ar
operable. Many patients survive for years if the tumour
chronic, but the more probable duration is a matter o
months.

Treatment of Intra-cranial Tumour.—When a diagnos
has been made as regards the existence of an intracrani
tumour, its probable site, and its possible nature, it must ne
be decided whether medical treatment is advisable and whethe
—and if so when—operative interference should be carrie
out.

Provided the patient has not developed optic neuritis, an
there is, therefore, no immediate fear of sight being endangere
antisyphilitic treatment should be energetically carried ou
both potassium iodide and mercury being given; but durin
the whole course of the disease the optic discs should
systematically examined every week.

If antisyphilitic treatment fails, the case becomes surgic
and the surgeon must consider whether operation is to
limited to the relief of pressure and to save the patien
sight, or whether he should endeavour to remove the tumo
Where an exact diagnosis of position is impossible it is well
remove a large piece of bone either in front or behind t
Rolandic area—but not over it—in order to obviate t
possibility of a hemiplegia resulting. Pressure on a cerebe
hernia, should it consist in a protrusion of the motor ar
through the wound, is very apt to cause paresis if not paraly
By the operation referred to, pressure can be removed in
cases in which the growth is *above* the tentorium.

In cases in which the tumour lies *underneath*
tentorium the bone should be removed over the right or
side of the cerebellum, and where localisation is impossible

pieces of bone should be removed, one from either side, leaving a bridge in the middle line. It is sometimes desirable to delay incising the dura mater until a later date.

The danger of lumbar puncture in cases of subtentorial pressure is due to the possibility of the medulla with its important centres being forced down into the spinal canal whenever the excess of cerebro-spinal fluid in that canal is drawn off. Not infrequently cerebellar tumours are cystic, and tapping may be possible without actual removal of the tumour.

11. Encephalitis

The term implies inflammation of the brain, which may be acute or chronic, a somewhat trivial hyperaemia, or a serious and fatal infective suppuration. Many forms of encephalitis are certainly of toxic origin, and, as stated in the descriptions of acute bulbar paralysis and infantile paralysis, the lesions in these diseases are exactly similar to toxic encephalitis. There are also many forms of encephalitis which follow an acute infective fever or are due to direct infection of brain tissue as the result of fracture of the skull, infection from a cerebral abscess, caries of bone, or infective thrombosis. In ulcerative endocarditis and pyaemia there may be numerous small capillary emboli containing organisms and capable of setting up a corresponding number of local areas of cerebritis.

The affected brain tissue varies considerably in appearance, depending much on the amount of haemorrhage associated with the inflammation and the presence or absence of pus. In many forms of encephalitis the pathological changes are limited to hyperaemia with a certain amount of inflammatory oedema, while in other cases actual softening and even suppuration may result.

The **Clinical Features** depend on the type of inflammation present, but headache, delirium, convulsions, and eventually coma are extremely common, while all the symptoms included under the term malaise are generally present. The earlier phenomena due to the cerebral condition are *irritative*, and those which develop later are *paralytic*. Probably many cases of concussion of the brain are in reality encephalitis due to traumatic injury, and not infrequently in a

severe case where recovery occurs, the mental faculties are seriously affected, either temporarily or permanently.

Various names have been given to encephalitis in different parts of the brain; for example, *superior polio-encephalitis*, described by Wernicke, involves specially the nuclei of the ocular nerves, whereas *inferior polio-encephalitis* is the term given to cases in which the cranial nerve nuclei in the pons and medulla are affected. Encephalitis is also one of the common causes of acquired infantile hemiplegia or diplegia.

The **Prognosis** depends on the site and severity of the lesion, and the **Treatment** should be on general lines, special attention being paid to rest in bed, to purgation, to the administration of sedatives such as the bromides, and in certain cases to the use of iodide of potash and mercury.

12. Cerebral Abscess

An encephalitis of suppurative nature, and which may be found in the brain tissue itself or between the membranes and the brain.

Etiology.—Cerebral abscess may be the result of injury and especially fracture of the skull, the abscess being found in close relationship to the site of the lesion; foreign bodies such as bullets, sometimes carry into the brain septic organisms which may set up suppuration.

Disease of the middle ear is probably the commonest cause of abscess in the brain, and the abscess may be secondary to a septic thrombosis of the lateral sinus infected from the ear. It generally results from caries of bone, by which in time the meninges become involved. The most common site of perforation is the roof of the tympanum, when the temporo-sphenoidal lobe is specially apt to be the site of the abscess; the second in frequency is through the posterior wall of the antrum, the labyrinth, and internal auditory meatus, when the lateral sinus is apt to be infected and the abscess is more likely to be cerebellar. The pus may, however, find its way inwards by any of the venous or lymphatic channels of communication between the ear and the inside of the skull. Very rarely does suppuration of the outer ear extend by the lymphatics to the brain without the middle ear being involved.

Sometimes in diseases of the nose, caries of the ethmoid

bone occurs, and in this way the brain may become involved. The abscess is most frequently in one of the frontal lobes and in close proximity to the affected bone.

Very rarely does suppurative disease of the orbit extend to the brain, and pus formation only occasionally occurs in cerebral tumours, perhaps generally in tubercular cases.

Cerebral abscesses may be the result of pyaemia, and they are very common in cases of ulcerative endocarditis. In bronchiectasis, and more rarely in other conditions, metastatic abscesses have been found in the brain.

Abscess of the brain is sometimes a sequel to a continued fever, and there are cases in which a cerebral abscess has occurred without any recognised primary cause.

Pathological Anatomy. - A cerebral abscess is generally single, and varies considerably in size. The pus is often stinking in odour and greenish in colour, while there is usually in the less acute cases a definite capsule. Depending upon the size of the abscess, there is more or less flattening of the cerebral convolutions, and in some cases suppurative meningitis occurs.

The organisms most frequently found in cerebral abscess are Staphylococci and Streptococci, although many other may also be present.

The **Clinical Features** vary greatly in different cases; sometimes there is undoubted evidence, owing to the irritation phenomena present, that a cerebral abscess exists. In other cases there may be no symptoms at all, or, at all events, the symptoms present may not suggest the existence of an abscess. There may be headache, pain—sometimes increased on tapping over the site of the abscess,—cerebral vomiting, marked vertigo, and occasionally optic neuritis. Lethargy is often an early and prominent symptom, the patient spending most of the day in a stupid condition if not in sleep. The pulse is usually slow, it may be only 40 to 50, and the temperature is often subnormal: the respirations are slow, although sometimes they assume the Cheyne-Stokes rhythm. Focal symptoms of cerebral abscess are of the utmost importance, and depend on the site of the pus. The most careful examination should be made of the ears, of the skull, and of the nose, so as to determine a possible cause for a supposed abscess of the brain. It is also desirable to examine the

cerebro-spinal fluid by lumbar puncture. Usually an excess of polymorphs is found in a somewhat opalescent fluid, and micro-organisms may be detected by staining a film or by culture.

The reader should refer to the description of the phenomena which characterise a cerebellar lesion (p. 712), although it must be remembered that a cerebellar abscess may occur without any typical phenomena whatsoever. MacEwen considers that the percussion note obtained over the skull becomes more resonant when the lateral ventricles are distended with fluid, and this may be of assistance in a case of supposed cerebral abscess.

A *temporo-sphenoidal* abscess will, if on the left side in a right-handed individual, give rise to auditory aphasia, and on whichever side it may be the epigastric and abdominal reflexes of the *opposite* side to the abscess will be diminished or absent.

An abscess in the *lateral lobe of the cerebellum* will cause neck rigidity with head retraction, excessive giddiness on attempting to sit up in bed, and a cerebellar type of reeling gait. Nystagmus, especially *towards* the side of the abscess and inco-ordination of the limbs on the same side are generally present.

The **Diagnosis** is often of very great difficulty, especially where there are no focal symptoms and no definite evidence of the presence of an abscess at all. It is wise to investigate the blood so as to determine the presence or absence of a leucocytosis, and the optic discs should be examined with the greatest care. In the writer's opinion too much stress is laid on the supposed frequency of optic neuritis in cerebral abscess.

In *thrombosis of the lateral or other sinuses* of septic nature a careful examination should be made of the neck (the jugular vein), and also of any swelling over the mastoid region, the position of the mastoid vein which enters the lateral sinus being kept in mind.

The **Prognosis** depends on the possibility of successful operation, and on the probable absence of meningitis or of an extensive area of encephalitis.

The **Treatment** is purely surgical, and is therefore omitted. It is important to remember that early operation is desirable

and that opium or morphia should not be given where suggestive symptoms might be masked by its use.

13. Chronic Hydrocephalus

The term generally implies distension of the ventricles of the brain with cerebro-spinal fluid, although cases in which there is an excess of fluid between the dura mater and the brain have also been included, and the terms *internal* and *external* are accordingly used to distinguish the two varieties.

(1) **Internal Chronic Hydrocephalus. Etiology.** — It may occur in the foetus (congenital type), and interfere with normal labour, or it may develop after birth (acquired type); in both cases it is probably due to inflammation of the ependyma ventriculi. In certain instances this inflammation may be of tubercular origin, but in others, and especially some of the congenital cases, there is no satisfactory explanation obtainable.

In adults this form of chronic hydrocephalus is usually due to pressure on the veins of Galen by a tumour, or to occlusion of the foramen of Magendie; but sometimes inflammation of the meninges or of the ependyma accounts for the condition. It may be associated with spina bifida or syringomyelia.

Pathological Anatomy.—Where there is great distension of the ventricles, the brain is greatly thinned, the convolutions are flattened, and the sulci are almost obliterated. There may be from 5 to 20 pints of fluid in the lateral ventricles, and the bones of the skull are widely separated, while the appearance of the child's head described below is extremely typical. The fluid is generally clear like water, containing a trace of albumin, and the ependyma lining the ventricles is much thickened, opaque, and covered with greyish granulations. The presence of a tumour may be noted where it accounts for the condition. Sometimes the pressure is so great that the optic commissure is flattened by the distended 3rd ventricle.

Clinical Features.—The head is greatly enlarged, and in the child may measure 30 to 50 inches round the forehead. The frontal and temporal regions appear to overhang the small, pinched-looking face. The bones of the skull are widely separated, and *ossa triquetra* or Wormian bones may

form in the gap between the bones or over the fontanelles. The head may be so heavy that the patient is unable to raise it from the pillow, or he may have to support it between the hands. There may be few compression phenomena, or there may be blindness due to pressure on the optic commissure. The intelligence varies greatly, it is sometimes deficient, or it may be normal; but it is interesting to note that in the child if ossification of the bones is nearly completed, the pressure phenomena are more marked, and therefore severe headache and vomiting are extremely common. Nystagmus is frequently present in severe cases in children.

In adults the head cannot so easily enlarge, and in consequence the evidences of pressure are much more serious. Headache, vomiting, convulsions, and paralysis due to pressure on cranial nerves, are common. There is early loss of upward ocular movement and impairment of the pupillary light reflex and optic atrophy may develop without any preliminary neuritis. The optic atrophy is probably due to pressure of the distended 3rd ventricle. Certainly in cases of tumour optic neuritis and consecutive atrophy frequently develop.

Diagnosis.—In the young child a glance at the head is sufficient to render the diagnosis clear, while the patent fontanelles found in older hydrocephalic children will help in any doubtful case.

In adults it is usually easy to recognise increase of intracranial pressure, although the cause of that increase may be obscure.

Sometimes cases occur in which there is enlargement of the head commencing after adult life, and probably similar in nature to acromegaly, but this is extremely rare.

The **Prognosis** is unfavourable, although in not a few instances benefit is obtained by careful treatment. In some cases after tapping, the fluid does not reaccumulate rapidly, while in other cases the ventricles fill up in a very short time. In tumour cases the possibilty of removal of the neoplasm pressing on the veins of Galen is very problematical. Where there is chronic meningitis present, the prognosis in adults is more favourable, but even in these the percentage of recovery is extremely small.

Treatment.—The historic case in which the child with hydrocephalus fell down, struck its head upon a nail, which

perforated the skull and opened into one of the ventricles, is certainly encouraging, because in that case recovery occurred, and fortunately without sepsis developing.

In young children it is wise to endeavour to tap the ventricular effusion. This may be done in different ways, either (1) by repeated lumbar puncture, when, provided there is free communication, the excess of fluid can be greatly diminished; or (2) a child whose anterior fontanelle is still patent may have the operation performed by inserting the trochar and cannula at the outer angle of the fontanelle, care being taken to remove only a few ounces at a time; or (3) the skull may be trephined and the fluid removed either from time to time or by continuous drainage. The last operation is obviously the most suitable for adults. Watson Cheyne recommends an attempt at permanent drainage into the subdural space [illegible] to add that many neurologists do not [illegible] treatment especially for congenital cases [illegible] permanent drainage has often not been [illegible]

In children, after [illegible] by any [illegible] of these methods, the head should be [illegible] by an elastic bandage or strapping, but care should be [illegible] the appearance of either cerebral [illegible] due to too great pressure. A course of [illegible] mercurials has sometimes been found useful [illegible] child's health may be benefited by the administration [illegible] of strengthening food and tonics.

(2) **External Chronic Hydrocephalus.**—In this condition the fluid accumulates [illegible] cerebral convolutions, and is generally due to [illegible] adhesions although it may also depend upon [illegible] cerebro-spinal fluid is found in excess in cases [illegible] paralysis of the insane, and in all cases in which either does not develop properly or where it atrophies.

The **Clinical Features** [illegible] suggestive of atrophy of the brain, the intellectual [illegible] usually failing, while in not a few cases the typical manifestations of general paralysis appear. In idiots in whom the brain does not develop at the same rate as the skull, there are often no particular clinical features due to the presence of the fluid. Little can be done by way of treatment in these cases. In general paralysis it may be desirable to trephine the skull so as to relieve pressure.

14. Disseminated Sclerosis

(Multiple Cerebro-spinal Sclerosis—Insular Sclerosis)

A chronic disease affecting the brain and cord, in which patches of sclerosis replace the nerve elements; they may be situated in practically any position.

Etiology. It is a very difficult matter to assign the proper causes to this disease. In some cases there seems little doubt that cold and exposure, injury, excitement, lead, or other metallic poison, and sometimes an acute infective fever, are responsible for the condition. It is common in both sexes and occurs soon after puberty and also in middle life, and it seems not improbable that it really may be of infective origin. It should be stated that many authorities attribute to syphilis an important causal relationship.

Pathological Anatomy.—The patches of sclerosis vary greatly in size, they are not limited to any particular tract and the same patch may involve white and grey matter although the white matter is the seat of election. They are pinkish in appearance, but when older they become greyish-white, and they are often hard and cartilaginous to the touch. The patches are specially common in the region of the basal ganglia, in the pons and medulla, while in other cases the cord, and particularly the cervical region, may contain several of these areas. The microscopic structure of the patches is simply a great proliferation of neuroglia forming a dense, firm nodule, which eventually destroys the nerve fibres or cells in the affected region, although it is a remarkable fact that for a long time the axis-cylinder processes persist, and may, in an unsatisfactory way, discharge their functions. In consequence of this, less secondary degeneration results than might be expected. It is probable that the sclerosis is toxic; whether due to an organism or not it is impossible to say.

Clinical Features.—The disease is chronic, insidious and gradual in its development, and the phenomena may be divided into three stages. These three stages have been described by Charcot, who considers that the *first* stage terminates with the disablement of the patient, due to spastic rigidity of muscles; that the *second* stage, beginning with the rigidity, terminates with the loss of the organic

reflexes; and that the *third* stage, beginning with the loss of these reflexes, terminates with bed-sores and death. The stages may be long protracted, and there is no given time when the patient should pass from one stage to the next. Usually the disease begins with loss of power in the legs, and the patient complains of feebleness in movement, with gradually increasing stiffness and rigidity amounting to spastic paralysis, and associated with this there is a marked increase in the tendon reflexes.

There are eight important clinical features which are typical of the disease.[1]

1. *Intention Tremors.*—These are peculiar involuntary quick jerks observable on voluntary movements. They generally appear first in the arms or in one arm, are markedly increased by nervousness, and are not under the control of the patient. The more the patient endeavours to arrest the tremors the worse they become, but they cease entirely during sleep. If the patient is given a glass of water to drink, he tries to raise it to his lips, but the jerky, uncontrolled oscillations, which increase as he continues the movement, make him spill the bulk of the contents of the tumbler, and as his head participates to some extent in the same kind of movement, the tumbler may be brought with a sudden dash against the patient's teeth; once he begins to drink his arm becomes steadier.

Some loss of power accompanies this tremor, but the legs generally show more weakness with spastic paralysis than loss of co-ordination. The marked development of this spastic paralysis with increased knee-jerks and ankle-clonus constitutes the rigidity described by Charcot as terminating the first stage of the disease.

2. The *speech* should become *scanning, staccato*, and *syllabic*, with slow, monotonous, stammering articulation, and the ends of the words are cut short.

3. *Nystagmus*, generally lateral and rarely vertical, is almost always present on moving the eyeballs to one or other side.

4. *Headache* and *giddiness* are extremely frequent, and are often bitterly complained of by the patient.

5. At an early period in the disease there may be some

[1] It is remarkable how many atypical cases present themselves, and how often the distinctive intention tremors are absent. This depends on the site of the sclerotic patches.

bladder symptoms, such as difficulty in retaining the urine or in passing it

6. The *mental changes* include defective intelligence and the almost complete failure of memory, and sometimes insanity develops in the course of the disease.

7. *Absence of the abdominal reflexes* is an early and very important clinical feature of the disease.

8. There is often *pallor of the optic discs*, defective vision, and a central scotoma for colours. The fields of vision are generally affected centrally, although in some cases the limitation is peripheral.

The other clinical features vary, depending on the sites of the patches of sclerosis. The optic nerves may be affected by a patch involving the chiasma and causing secondary optic atrophy, and paralysis of some cranial nerve may result from a patch involving its nucleus: in this way the eye muscles may not infrequently be paralysed.

There is a varying degree of muscular weakness in practically all cases of the disease.

Sometimes patches of anaesthesia occur, although ordinary sensation is not, as a rule, much affected.

In the last stages of the disease the loss of control of the organic reflexes and the bed-sores rapidly lead to a fatal result.

Diagnosis.—The great clinical features are very typical if present: it must, however, be remembered that we may find disseminated sclerosis associated with other diseases, such as locomotor ataxia and general paralysis of the insane, and there is a large group of cases which are undoubtedly disseminated sclerosis and yet are very far from being typical.

The **Prognosis** is bad: the patient will probably live five or six years: in some cases the first stage may be even more protracted, and the question is rather the rate of progression downwards than anything else. In favourable cases the disease may be arrested for a considerable period of time.

Treatment.—Antisyphilitic remedies should certainly be tried: arsenic and silver nitrate have been commended, but no drug seems to be of much avail. Recently hydropathic and electrical treatment, and rhythmic exercises on the Swedish system, have received considerable attention, but the results are very disappointing. Probably rest is as important as any other form of treatment.

15. Infantile Hemiplegia

A small percentage of all cases of hemiplegia are found to be of congenital origin. Both sexes suffer equally, and inherent delicacy does not appear to be so important a factor as might be imagined. Opinion has varied greatly as to whether alcohol and syphilis in the parents have much to do with this form of hemiplegia in the child. Of the causal factors which are definitely known, injury at birth is unquestionably the most frequent, the head being compressed by the forceps in cases of instrumental delivery. In other cases, the hemiplegia develops during the early years of life, and probably infectious diseases which set up encephalitis are responsible for a large number of these cases. A convulsive seizure precedes the paralysis, and on recovery the hemiplegia is noted.

Pathological Anatomy.—In cases occurring at child-birth, haemorrhage or injury to the cortex of the brain produces the condition. Atrophy of the brain cortex, whether the result of meningeal haemorrhage or not, is associated with a large proportion of all cases of congenital hemiplegia. Sometimes there is more or less distension of the ventricles. A cystic formation, commonly called *porencephaly*, has been found present in a number of cases. These cavities are most probably of developmental origin; some of them are connected with one of the lateral ventricles.

In a small proportion of cases haemorrhage, thrombosis, and embolism may be present, similar in all respects to these conditions in the adult.

Clinical Features.—There are generally one or more convulsions, followed by the development of the hemiplegia, which is noted when the child recovers consciousness; but, as the pathological anatomy suggests, there are also cases in which the paralysis develops more slowly—cases in which atrophy or arrested development of one-half of the brain is present.

In some instances the hemiplegia gradually disappears; in others it persists more obviously in the arm than in the leg, and contracture is by no means infrequent, while athetosis is peculiarly common in these cases. The athetoid movements are slow, rhythmical, and involuntary flexion and extension movements of the fingers and thumb, while hyperextension occurs, and the fingers are widely separated; the

movements are increased by emotion, and may persist more or less during sleep. Spasm or paralysis of ocular muscles is not uncommon at the commencement of the attack. Probably the feature which is most striking in addition to the athetosis consists in the large number of cases in which mental weakness is present, and all stages and types of imbecility may be found in these patients. Epilepsy is also a fairly frequent sequel.

The **Diagnosis** is not difficult. The lesion belongs to the first motor neuronic system, and therefore the muscles do not waste unless from disuse; it is easy to distinguish between a lesion of the cells in the anterior horn of the cord in *infantile paralysis*, because in the latter case wasting is extreme, and there is abolition of reflexes, and also loss of electrical reactions: whereas in infantile hemiplegia not merely is there less wasting, but the reflexes tend to be exaggerated, and the electrical reactions are usually normal.

The **Prognosis** is generally unfavourable.

The **Treatment** is most unsatisfactory. Where a haemorrhage has just occurred under the membranes, it may be possible by surgical interference to do something for the patient; and sometimes good feeding and care, with massage to the affected limbs, may result in great improvement in a case which, at first sight, appears hopeless. Imbecility and epilepsy are very unfavourable clinical features, and little can be done surgically in such cases, while the later treatment consists in the careful training and education of the child.

16. Congenital Cerebral Diplegia

(including little's disease)

The disease begins before or just after birth. It is the result of a toxin sometimes hereditary in origin, but however it may be acquired, it causes the most profound interference with the development of the central nervous system. According to the particular part involved, so will the case show motor, sensory, or mental features.

There is generally atrophy of the cerebral convolutions with neuroglial proliferation, but with no evidence of any inflammatory or vascular lesion.

The **Clinical Features** include spastic paralysis with

rigidity. The lesion is almost always bilateral, but may be better developed on one side than the other. The term "Little's disease" is applied to cases in which there is spastic diplegia involving the legs only. There is generally marked mental enfeeblement, the mind apparently failing to develop, and there may be absolute idiocy.

The gait may be often cross-legged should the patient be able to walk at all. There is usually spinal curvature and in some cases athetosis. Occasionally optic atrophy is noted and sometimes there is paralysis of the 3rd, 6th, and less frequently other cranial nerves. There are no sensory changes, but epilepsy is not infrequently noted.

The **Prognosis** is most unfavourable, and no **Treatment** will probably yield much benefit.

17. Amaurotic Family Idiocy

A rare disease in which impairment of vision together with mental and physical weakness end, after a protracted but progressive illness, in death.

The cells in the cortex of the brain atrophy, and there is an associated degenerative change in the motor axis cylinder processes proceeding from these cells.

The disease begins about the fourth month of life with weakness of the muscles of the body; at first they are simply flaccid, later there is some spasticity. Coincidently the child becomes listless and impairment of vision develops. There is no involvement of the superficial or deep reflexes. There is atrophy of the optic discs with a bright red spot at the macula surrounded by a cloudy-looking halo. The child generally succumbs in about two years, and no treatment has been found of any avail.

18. General Paralysis—Dementia Paralytica

A primary degeneration of the brain, with progressive motor paresis and dementia. According to Dr. Mott it is the cortical association neurones which primarily suffer. It really constitutes the cerebral type of locomotor ataxia.

Etiology.—Syphilis is now recognised as the cause of

this disease, although worry, mental overstrain or continued excitement, and possibly physical overwork, alcoholism, and sexual excess, may all contribute to the production of the disease. Ford Robertson believes that syphilis plays only a secondary part, and by its effect on the nervous system permits the attack of other organisms, but his views still lack confirmation. Males of adult age suffer chiefly, and the disease is rare after 50.

Pathological Anatomy.—The brain is markedly atrophied, especially anteriorly, and there is a corresponding excess of cerebro-spinal fluid; the lateral ventricles are enlarged, and the ependyma is thickened. The cerebral membranes are also thickened, and *pachymeningitis interna haemorrhagica* may be present. The smaller arteries in the cortex are thickened and the middle coat of these vessels is markedly hyaline. The neuroglial cells of the cerebral convolutions are greatly hypertrophied, and have undergone proliferation, and the cortical nerve cells show degenerative changes with chromatolysis. The cord may present changes similar to the lesions in tabes.

Clinical Features.—No disease possesses so many different symptoms or types. The leading features common to all cases are:—

(1) Progressive mental changes, and especially dementia with excitement or depression.

(2) Progressive motor weakness with tremor, chiefly of the tongue and face.

(3) Hesitating, stammering, jerky speech, often with dropping of syllables, while labials, linguals, and dentals become difficult, such words as "British constitution" and "artillery" being specially troublesome to the patient.

(4) Writing is affected soon after speech, and words or syllables are omitted, while much confusion of ideas exists.

(5) The pupils are unequal, and usually show loss of the light-reflex (the Argyll-Robertson phenomenon) while reacting normally to accommodation.

(6) The knee-jerks are generally affected, in certain cases exaggerated, in others—in which tabetic phenomena have also developed—they are absent, while sometimes they differ on the two sides.

The tremor of face muscles is well seen on asking the

patient to show his teeth, and the fingers are often tremulous. Optic atrophy may be present in a small number of cases, and sometimes in tabetic cases there is paralysis of oculo-motor muscles, temporary or permanent.

Epileptiform attacks are not uncommon at a later stage in the disease, with or without loss of consciousness, but with twitchings, often marked convulsions, and almost always a marked rise of temperature. They may be frequent, or only occasional, and each leaves the patient worse than he was before. They sometimes simulate Jacksonian epilepsy.

The disease usually runs a slow progressive course, beginning with a definite mental change, loss of sequence of ideas, and lapses of memory, while the patient may develop sudden business energy, generally misdirected, and grandiose ideas and ambitions aims and projects, which land him too often in financial ruin. In other cases mental depression is more manifest, with suspicion of persons with whom he comes in contact.

In the last stages of the disease the patient is practically confined to bed, callous to the calls of nature, and absolutely unable to take any interest in his surroundings, and death may ensue from bed-sores or from pulmonary or other complications.

The cerebro-spinal fluid obtained by lumbar puncture contains a great excess of lymphocytes.

Diagnosis.—The recognition of the six cardinal clinical features which are generally present will help in the diagnosis of a case of this disease. In *disseminated sclerosis* the intention tremor (if present), nystagmus, the scanning speech, and the absence of the Argyll-Robertson phenomenon are usually sufficient. *Locomotor ataxia* is held to be closely associated with general paralysis, but the differential diagnosis is easy, unless where a combination of the two diseases exists.

The **Prognosis** is bad, but life may be prolonged for two or three years. The duration is much shorter in rapid cases. With care and treatment something may be done, but not much.

The **Treatment** consists in complete bodily and mental rest; and in the relief of symptoms, and especially mental exaltation and restlessness, by the administration of bromides, hyoscine, trional, etc. As in the case of locomotor ataxia

we are hoping much from the use of salvarsan (see p. 124). Antisyphilitic treatment on the old lines is indicated, but it has not yielded satisfactory results.

VII. DISEASES OF THE NERVOUS SYSTEM WITH NO CONSTANT OR WELL-RECOGNISED ORGANIC LESION.

1. Paralysis Agitans

(Parkinson's disease—shaking palsy)

A chronic disease of the nervous system, characterised by a peculiar tremor associated with muscular weakness and rigidity.

Etiology.—It generally occurs between the ages of 40 and 70, and somewhat more frequently in males. Hereditary predisposition is doubtful, but there is often a family history of nervous disease. Cold, damp, anxiety, mental strain, fright, an accident, and a continued fever have all been believed to be predisposing or exciting causes in different cases.

Pathological Anatomy and Pathology.—No constant pathological change is present, but minute patches of sclerosis have been described, situated around the vessels of the medulla and spinal cord, while pigmentation and vacuolation of ganglia cells were found in certain instances. Most of these might be due to senility. Very probably the most important changes are those in the nerve cells of the cerebral cortex, medulla and cord, but whether due to a toxin or not is not known.

Clinical Features.—The disease usually commences in an insidious manner, and the following typical phenomena are present :—

(1) *Tremor.*—This begins in one hand, with either pronator-supinator, or pill-rolling movements. As a rule the tremor is limited to the hand and wrist. In the leg the movements are best seen at the ankle-joint. Often one side is affected before the other, and the head movements, which are less common, are generally vertical, and not rotatory (as they are in senile palsy). The movements are about 5 per second, are at first under voluntary control, and are well seen when the limb in which they occur is raised, although the

do not entirely cease in the attitude of repose. They stop during sleep, and are apt to be exaggerated by emotion. The handwriting is at first good, but soon voluntary control only permits of single letters being formed at a time, and even these are tremulous. In later stages of the disease the movements become much exaggerated.

(2) *Muscular Weakness and Rigidity.*—Loss of power is very apparent, and may precede the tremor, although it generally advances simultaneously with it. Associated with this is rigidity, to which the slow, stiff, voluntary movements are due, and also the tendency to flexion of many joints, as well as the typical attitude and gait of the patient.

(3) *Attitude and Gait.* Rigidity and flexion are well seen in the attitude of the patient. His head is bent, the elbow is flexed, the face so expressionless as to be termed mask-like, and there is almost complete loss of ability to display any emotion, while the lips and tongue acquire a rigidity which renders speech slow and monotonous. Later the voice is high pitched. The intellectual sharpness is in marked contrast with the vacuous expression.

The gait is typical. He rises with difficulty, perhaps after several failures, from his chair, and with head bent forward he starts to walk with slow steps, which soon become faster and faster, as if he were running after his centre of gravity. This is termed *propulsion*, while by the term *retropulsion* is meant the result of impelling him backwards, when, after similar rapid but retrograde steps, he often ends his career by falling. The gait is also described by the term *festinant*. In the later stages of the disease the elbows, knees, and other joints may become ankylosed in the position of flexion.

There are no alterations in superficial and tendon reflexes, or in electrical reactions.

Certain sensory changes may be present, and especially a hypersensitiveness to cold, and a tendency to profuse perspiration. In very severe cases a considerable amount of pain is not infrequently complained of.

Diagnosis.—*Senile tremor* has not the characteristic rigidity nor the pronator-supinator movements, and the head is generally more markedly affected. *Double hemiplegia* and *bilateral degeneration of the cerebral cortex* resemble paralysis agitans for a time, but the definite increase in the tendon

reflexes with plantar extension and the absence of the tremor should prevent mistake. In *disseminated sclerosis* there is exaggeration of tendon reflexes, nystagmus, and scanning speech, and the jerky movements are quite distinctive.

Prognosis.—The disease is long protracted, and not necessarily immediately serious, but cure is rare. The duration might be safely reckoned as extending for years.

Treatment.—There is no satisfactory treatment. Try arsenic, and, in severe cases, hyoscyamine or hyoscine, and bromides. Recommend a period of physical and mental rest, and give general tonics. Locally massage is often of benefit, and galvanism has occasionally been used with temporary improvement. Pituitary extract (gr. 2-5) injected into the muscles is said to control the tremor, but it must be carefully watched. Should the blood-pressure rise too high it must be stopped for a time.

2. Epilepsy

Sudden loss of consciousness, with tonic and later clonic spasms.

Etiology.—It seems probable that there are predisposing as well as exciting causes, although many of the so-called exciting causes may be rather the result of the condition than related to it etiologically. The disease generally begins before the age of 20, and in many cases at even an earlier period; probably three-quarters of all cases of idiopathic epilepsy commence before that age. There are certainly two periods when epilepsy is most apt to commence: (1) during the early years when brain development is most active, and (2) at puberty, when reproductive development is most active. Males suffer rather more frequently than females.

There is a strong hereditary tendency to epilepsy, and especially in families who are rightly called neurotic, and in whom a family history of insanity exists. Alcoholism in the parents tends to produce epilepsy in the child, and undoubtedly syphilis, although not such a potent factor as at one time it was believed to be, is still responsible for a certain proportion of cases. The so-called exciting causes are:—fright, which probably rarely or never produces true epilepsy; injury; fevers; some peripheral irritation, such as a tight prepuce or inflammation

of the gums associated with dentition, and more rarely errors of refraction, a foreign body in the ear or nose, or the presence of worms in the rectum. These have all led to fits in young children, although such fits cannot be designated true epilepsy, inasmuch as they cease when the action of the irritant is removed.

Toxic agents produce a special type of epilepsy; for example, in uraemia the seizures are epileptic in nature, and in lead poisoning, epileptic manifestations have been observed.

Vascular and cardiac conditions leading to cerebral anaemia tend also to produce epileptiform seizures.

Organic lesions of the brain and membranes, defective development of the cerebral cortex, tumours, hydrocephalus, and a spicule or bony node growing from the inside of the skull have been found in cases of epilepsy, although more commonly in the type of epilepsy termed Jacksonian.

Pathological Anatomy.—There may be organic disease, such as tumour, abscess, gumma, etc. In cases in which dementia develops secondarily, there are patches of sclerosis seen especially in the region of the cornu ammonis, and less frequently there is atrophy of the optic thalamus, the cerebellum, or the medulla. Where the condition is *idiopathic* many varied microscopic lesions have been described, especially changes in cortical and other nerve cells, but none of these can be called constant. Bevan Lewis, for instance, has noted fatty changes in the nuclei, and vacuolation and disintegration of cells in the second layer of the cerebral cortex in a number of cases. A diminution in number and size of cortical cells may be seen, probably indicating faulty development, and also neuroglial hypertrophy with or without sclerosis, which is in all probability a result of the attacks. Much significance has been attached to the vascular changes, the engorged veins and tortuous arteries with hyalin-looking bodies lying free in the lumen of the blood-vessels (Turner).

A fit seems really to be a discharge of nerve energy, and the typical aura points to the cortex as the likely centre of the nerve storm. The theory that vasomotor spasm is the cause of the fit is probably incorrect.

The presence of some toxic substance in the blood or cerebro-spinal fluid has often been suggested, but there is no corroborative proof.

Clinical Features. Many types are described; of these the most important are:

1. The Grand Mal.—There is generally a definite *aura, or warning of the approach of the fit*, and this *aura* may indicate the site of disturbance and possibly even the means of treatment. The *aura* may be (*a*) sensory, sometimes a feeling of discomfort over the region of the stomach or abdomen, or a tingling or feeling of numbness in one hand, arm, or leg, or (*b*) it may be a special sense impression, such as a peculiar taste or a flash of light; while (*c*) a psychical *aura* is occasionally experienced. Good examples of this kind of *aura* are the appearance of some imaginary face, or a dreamy sensation of something morally wrong. (*d*) A motor *aura* is occasionally present, generally in the form of a spasm, associated with a certain group of muscles. The *aura* may give sufficient intimation to enable the patient to sit down or throw himself on a bed or chair so as to avoid injury.

Immediately before the fit occurs the patient emits a loud *cry* or *groan*, then suddenly loses consciousness, and falls down if he is standing, and the muscles of the body are thrown into a condition of violent *tonic spasm*, which is bilateral, but more marked on one side of the body. The spasm is very typical: the head is thrown back, or turned to the more affected side, the arms flexed, the hands clenched, the legs extended and rotated inwards, the respiratory muscles in a state of rigid spasm, and as a result the face, at first pale, soon becomes cyanosed, and the veins of the neck and face engorged with blood. The pupils are widely dilated, and the eyes are fixed in an unnatural stare, sometimes being turned upwards. This stage lasts a few seconds, and is followed by the stage of *clonic spasm*, during which jerking movements occur generally at the rate of about three a second. All the voluntary muscles of the body may participate in the clonic movements. The eyes roll, the eyelids open and shut, the tongue is apt to be bitten by the teeth, and the patient froths at the mouth, especially during the terminal clonic movements, which are usually of special severity. This stage lasts from two to three minutes, and often in this or the preceding stage the patient evacuates the bladder or bowels. The breathing which recommences during the clonic stage is peculiarly stertorous, and this continues until the clonic movements have subsided

and the patient passes into *the stage of somnolence or sleep*, in which he remains for one, two, or more hours. Occasionally coma follows the fit, but as a rule only in cases of great severity, in which fit follows fit, and in the intervals the patient remains absolutely unconscious; this condition is termed the *status epilepticus*. In the *status epilepticus* the temperature nearly always rises, and death may occur from exhaustion. Uraemic convulsions very frequently take the form of the *status epilepticus*.

Before a fit the patient is often drowsy, and has a feeling as if something was going to happen, while headache is not uncommon. After a fit is over, the patient is brighter, once the stage of sleep has passed off, although in certain cases, as the result of a fit, headache may persist for some time. Sometimes fits are associated with insane acts or hallucinations, and not infrequently assault and murder have been committed, while in other patients the post-epileptic condition resembles hysteria more than anything else.[1] It is true that the patient may remember nothing of the attack, and that peculiarities of temper are present after epileptic seizures, but unquestionably cases of post-epileptic insanity are by no means common.

The knee-jerks are frequently lost for a short time after a fit, but this loss is soon replaced by exaggeration; the plantar response is at first absent, then becomes extensor, and later changes to the usual flexor type. In certain cases there may be local paralysis or local anaesthesia, although rather in cases where the fits are of Jacksonian nature.

It is not uncommon to find traces of sugar and albumin in the urine after a fit, but their significance is not often important.

II. **Petit Mal.**—Most epileptic persons are subject to *petit mal*, and there are cases of epilepsy so mild that *petit mal* alone may be present. It consists in a *brief loss of consciousness*, in which frequently the patient is quite unaware that anything has happened. He may suddenly forget what he is saying when engaged in conversation: it may be, in fact, merely a sudden lapse of memory, and in a moment he resumes the interrupted conversation as if nothing had happened. Occasionally an *aura* is present with these minor fits, but as a rule there is merely the sudden unconsciousness, with pallor

[1] Post-epileptic automatism—as it is called—is much more frequently pulling at the clothes, scratching, or some equally inoffensive performance.

of face, fixity of eyes, and momentary cessation of breathing. Sometimes the bladder is emptied during a minor fit, but the tongue is never bitten, and very rarely does the patient fall down. The pupils, however, dilate even in this mild form of epilepsy, and there is often alternate dilatation and contraction for some little time after the fit is over. There are no distinct convulsive seizures, although a few tonic and clonic movements may be noted, while the lethargic state which so constantly follows the *grand mal* is not generally present.

Patients who suffer from either *grand mal* or *petit mal* very frequently begin to do so at night, and nocturnal enuresis (wetting the bed), or the patient wakening with a headache, may in the first instance, be the only indication that something unusual has occurred. Vomiting occasionally follows an epileptic fit, and may always do so in certain patients. The number of fits varies greatly; there may be only one seizure in a number of years, or there may be several attacks, both *grand mal* and *petit mal*, in one day. Some epileptics have their fits only during the night, whilst others have them also in the day-time. Not infrequently fits occur in a longer or shorter series, and then there is an interval during which the lesser type of fits alone troubles the patient: in women the menstrual epoch is a common period of attack.

The mental condition of epileptic patients forms an important and sometimes a distressing feature; some patients develop dementia, others show serious and increasing mental excitement amounting almost to mania, and in not a few cases delusional insanity is associated with the fits—in fact, a certain proportion of asylum inmates owe their mental condition to epilepsy. In other cases epilepsy merely dulls the mind, and causes considerable loss of memory, and it is only in the milder type of cases that these phenomena are unimportant and can be neglected. Possibly much of the mental deterioration is in reality the result of bromides given for too long a time and in excessive doses, but in every case of epilepsy it should be remembered that there is a tendency to mental change, and that homicidal, suicidal, and imbecile types may be met with.

There is a characteristic expression termed the *epileptic facies* which is peculiar to patients suffering from epilepsy of severe type. In some cases the extremities become blue

and cold, there is marked constipation, and often a large appetite. Headache, mental confusion, and vertigo are also frequently complained of.

III. Sometimes fits are associated with **Insane Acts** or **Hallucinations**, and while it is necessary, from a medico-legal standpoint, to lay special stress upon this type of epilepsy, it does not require any further description. Insane acts or hallucinations may occur after the fit, and for these the patient is quite irresponsible.

IV. **Jacksonian Epilepsy.**—This is the form in which, generally owing to a lesion of the cortex, there are localised spasms (mono-spasm), sometimes extending so as to become general. If localised, they may occur without loss of consciousness; if they become general, consciousness is nearly always lost. A localised and temporary paralysis may follow the attack, and sometimes it is associated with a localised numbness or tingling in the part affected. It should be remembered that this form of epilepsy is the type most frequently met with in uraemia and general paralysis of the insane. Jacksonian epilepsy is referred to under cerebral localisation and cerebral tumours, but it has a close connection with idiopathic epilepsy which renders it necessary to describe it here.

Diagnosis.—It is impossible to mistake an epileptic seizure which is typical, but nocturnal attacks are often overlooked. As already noted, enuresis, biting the tongue, mental confusion in the morning, and a headache, may suggest that a fit has occurred during sleep.

Faints of a syncopal nature are often simulated by fits of the *petit mal* type. It is important, in connection with diagnosis, to investigate carefully the existence of an *aura*, and if convulsions occur, to get an accurate description of the attack. In *hysteria*, the patient is emotional, and the movements are excessive in extent, while the patient never bites the tongue, and does not fall down, unless a couch or bed is in a convenient position. The duration of the hysterical attack extends for a far longer time than an epileptic seizure, and it is rare to find a period of sleep or stupor associated with the attack. A hysterical patient rarely suffers from enuresis, and defæcation does not occur during the attack. It should, however, be remembered that epileptics are apt to become

emotional, and that it is extremely common to find eventually a combination of hysteria and true epilepsy in the same case.

There is a form of epilepsy associated with an old haemorrhage or patch of sclerosis or softening in the brain. In these cases the attack begins like a Jacksonian seizure, and the paralysed side is the one more affected. The attack usually occurs where the lesion is of infantile development and the mental condition of the patient generally approaches idiocy.

Prognosis.—Death almost never occurs in an epileptic fit, but the possibility of cure depends on many things. It is distinctly more favourable when epilepsy begins after the age of 20, and when treatment is commenced before the fits have gone on for over a year. Unfavourable cases are those in which the mind has become impaired, and certainly where the family history includes epilepsy in the parents, or other serious and constitutional nervous disease. There is the risk also, in most cases, of the patient damaging himself or herself during a fit, and cases are recorded where fracture of the skull has resulted from a fit occurring in the street. It is important, therefore, to remember that the occupation of the patient may expose him to great danger, and will greatly affect the prognosis as regards long life.

Treatment.—Every effort should be made to try and find out some definite cause for the attacks. Remember that there is no better method of treatment for epileptic children, where the epilepsy is severe, than to have them trained up in surroundings in which they will be free from being teased, and will not constantly have their disabilities forced upon their notice. Certain cases should therefore be brought up in asylums or in epileptic colonies, whereas others should be taught at home, if this is possible. A healthy outdoor life with little excitement, plenty but not excess of good food, and complete abstinence from alcoholic beverages of every kind, should be laid down as the best method of treating adult patients. Epileptics who have an *aura* recognisable by the patient prior to a *grand mal* do not need the constant supervision to prevent injury which other patients who have no such *aura* may require, and occasionally a fit can be prevented by utilising the information obtained by the *aura*. For example, if the *aura* be a sensory impression passing up

one arm, compression of the arm may prevent the fit occurring. Unfortunately, in the majority of cases such an attempt at treatment is of little avail.

The medicinal remedies most frequently given for epilepsy are the bromides. The sodium, potassium, and ammonium salts in equal parts, and about 20 to 40 grains of the mixture taken thrice daily, act most satisfactorily. The potassium salt has the oldest reputation, but is considered more depressing than the others are. It is difficult to say whether one bromide acts better than another. Certain cases have benefited from the administration of strontium bromide, although it is doubtful whether it is in reality much better than the others. Many physicians prefer one big dose of bromide, such as a drachm at bed-time. In some cases it is given before, in other cases it is given after, food, but it must be remembered that bromides in large doses should never be persisted in for longer than 6 or 8 weeks without intermission, because there is no doubt that it dulls the intellect and affects the memory, even apart from the epilepsy, which acts in the same way. There is also a risk of bromide acne and bromism occurring when the drug has been taken for a considerable time, and especially in patients who have any idiosyncrasy to the drug. Sometimes the use of arsenic along with the bromide prevents this trouble; in other cases the drug should be stopped for some weeks. The author has had the advantage of watching many cases treated by Dr. Byrom Bramwell with bromides (gr. 20), biborate of soda (gr. 10), and arsenic, and the combination has proved far more certainly efficacious than bromides alone. A double dose at night is often ordered, and a printed sheet is given to each patient on which the date of every fit is charted.

Cod-liver oil and iron are probably as important as bromides, but after treatment with bromides has resulted in amelioration or cure, the case should be kept under observation, and bromides in lessened doses given from time to time; in women, for example, they should be given at the menstrual period.

Belladonna, chloral, and cannabis indica form a group of remedies of considerable value, and they may take the place of the bromides when these drugs have been intermitted. Amongst other remedies suggested are ergot, antipyrine, nitroglycerine (with the idea that a fit is the result of vasomotor

spasm), zinc and silver salts, borax, and many other drugs, but, excepting borax, it is difficult to recommend one of these more than another. Opium has also been given, but its administration should be kept under very strict supervision. For the status epilepticus, chloroform anaesthesia is the best treatment, although hyoscine hydrobromide (gr. $\frac{1}{50}$) hypodermically may also be tried.

No case of epilepsy in children should escape the most careful and painstaking search for some exciting cause; a tight prepuce, the presence of worms, any irritation in nose or ears, in short anything likely to act as a peripheral irritant, should claim the attention of the physician.

The surgical treatment of epilepsy is not very hopeful: sometimes the *aura* suggests a site of irritation which can be localised in the brain, or tapping with the finger on the patient's head may elicit pain over one part of the bone, and under these circumstances trephining is warranted. In Jacksonian epilepsy it is needless to say that operative interference is often of the greatest service.

Epileptic colonies have been long established on the Continent, and there are already several in England, such as that of Chalfont St. Peter, Buckinghamshire, and the Maghull Epileptic Colony near Liverpool, while the system of finding suitable work for epileptics and having them congregated together in one institution has proved of great benefit.

It should be stated that under no circumstances is the marriage of epileptic persons to be encouraged, because epileptic children are an extremely common result of the union of epileptic parents, and even where only one parent is so affected.

Eclampsia

This is closely allied to epilepsy. Eclamptic seizures are more common in children, but they differ from true epilepsy inasmuch as there is no tendency to many attacks. It generally occurs in children up to the age of two years, it is rare after five, and exceptional after seven.

Etiology.—The *predisposing* cause is unquestionably the unstable condition of the central nervous system in early childhood, an instability specially marked if the child be weakly, subject to rickets, or the offspring of highly neurotic

parents. Anything causing debility, and many continued fevers, such as scarlet fever, measles, pneumonia, and whooping-cough, may be associated with one or more eclamptic seizures, and in the continued fevers in children a fit is often the initial manifestation.

The *exciting* causes can sometimes be differentiated from the predisposing. The irritation of the first dentition, some gastric or intestinal disturbance, and the presence of worms, may cause fits; while a tight prepuce, inflammation of the ear or nose, a foreign body in the external auditory meatus, and even a full bladder, may be sufficient to produce one or more fits in children who are predisposed.

Clinical Features.—Without any warning the child begins to show twitching movements often associated with grinding of the teeth. There may be a definite tonic spasm during which the body becomes rigid, the breathing ceases, and the face, after being pale, becomes blue and cyanosed. Clonic spasms follow, the eyes roll, the mouth and face muscles twitch, and twitching movements may be noted in arms and legs. It is common to find in children what has been described as carpo-pedal spasms, by which is implied the bending inwards of the thumbs and the great toes; but such spasms, though they may occur in connection with the fits, are often distinct from true childish eclampsia. There may never be more than one fit, or there may be several, which, however, do not recur when the peripheral irritant has been removed.

The **Treatment** should consist in giving an emetic or purge where the alimentary tract is at fault, in examining and possibly lancing the gums, and in removing any foreign body from ear or nose, should such a foreign body be present. A hot bath is often most important, and a warm sponge placed over the chest gives relief in cases in which the fits recur frequently, while the administration of chloroform to produce general anaesthesia is sometimes called for. Occasionally anti-spasmodics require to be ordered, and especially bromides or chloral, which should be administered in small doses. The child's general health should receive careful attention, and tonics and good food must be given.

Eclamptic seizures also occur where there is meningitis or inflammation of the brain, but these attacks are not the

same as the reflex eclamptic seizures to which we have just referred.

3. Chorea

(St. Vitus's Dance—Sydenham's Chorea)

A disease associated with, as yet, no definite organic lesion, but characterised by involuntary, inco-ordinate, and jerky movements of one or more limbs and often of the facial muscles, and with a close relationship both to rheumatism and to endocarditis.

Etiology.—Chorea is much more common in females (3 to 1) and it occurs specially between the ages of 5 and 15. It may be the result of one of the following conditions:—

(1) Many cases of chorea are directly related to rheumatism. There is a family history of rheumatism and generally a previous record of muscular or joint pains, while at the time of attack, rheumatic manifestations may be replaced by the choreic movements. Both acute rheumatism and endocarditis of rheumatic origin are probably due to the specific Diplococcus rheumaticus of Poynton and Paine. Recently this organism has been obtained from the brains of cases of acute chorea dying during pregnancy.

(2) There is no question that a fright or a severe scolding may be sufficient to start chorea in a neurotic, and perhaps we should add also a *rheumatic*, subject, and we may include under this group the chorea so frequently met with in children who have been overworked at school. There are also cases in which imitation of a comrade affected with the disease is said to be responsible for the attack.

(3) Some peripheral irritant which, however, must be co-existent with a neurotic disposition, may be credited with being an exciting cause. Examples of such peripheral irritants are a long prepuce, the presence of thread-worms, and occasionally the irritation associated with dentition. These causes are more common in the child, whereas in the adult, pregnancy is sometimes responsible for one of the most severe types of chorea, in which the severity of the attack may even endanger the life of the patient. In such cases it is more than likely that a *rheumatic* element is present in every case, and that the peripheral irritant is of less importance than used to be thought.

Pathological Anatomy.—Much discussion has arisen with regard to the site of the supposed organic lesions, and whether they should be sought for in the cortical cerebral convolutions, in the region of the basal ganglia, and particularly the optic thalamus, or in the groups of motor nerve cells in the anterior horn of the cord. The theory that multiple emboli, due to endocarditis with vegetations, account for the majority of cases, is not supported by the results of pathological research. It is true that the introduction of fine particles of sand into the carotid blood-stream of the dog causes choreic movements, but in cases which have proved fatal in the human subject there is often no evidence of endocarditis with vegetations, and also no small patches of softening such as would be caused by the smallest emboli. An infective organism, a diplococcus, has been described by Messrs. Poynton and Paine, in connection with the joints and cardiac lesions in acute rheumatism, and chorea has been produced in animals in, at all events, certain cases in which this organism was injected. Rheumatic endocarditis especially affecting the mitral orifice and less commonly pericarditis may be present. The old-fashioned opinion that instability of important brain centres explains chorea most satisfactorily, has still many supporters, and has much to be urged in its favour.

Clinical Features.— The characteristic features of the movements in chorea are:—

(1) They are involuntary; they are increased by nervousness and by any voluntary effort.

(2) They are jerky, the jerkiness being of an inco-ordinate type, and they absolutely lack rhythm.

(3) They are generally unilateral, so that a hemichorea is the most common form, although the opposite arm and leg may be more slightly affected.

As regards the face movements, they may be described as jerky grimaces with winking of the eyelids and twitching movements of the corners of the mouth, more marked on the affected side: and if the patient is directed to put out the tongue, it is often jerked out and as quickly drawn back, while in certain cases the lower jaw has involuntarily closed on the tongue, biting it severely. The muscular weakness associated with the choreic movements may actually amount to a definite paralysis. As a general rule the patient is able to swallow

freely, and respirations, although slightly jerky and irregular, show no marked alteration otherwise. Sometimes deep sighing inspirations are noted, and in severe cases the contraction of the diaphragm may not be quite rhythmic with the movements of the intercostal muscles.

In very severe cases, which are much more common in older patients and specially in pregnancy, the movements of the limbs are violent, and if the patient is not protected by pillows, serious abrasions may be caused, and indeed in exceptional cases the movements are so violent as to necessitate the use of a mattress on the floor, so as to obviate the possibility of the patient tumbling out of bed. In such patients, speech is generally considerably involved, and not infrequently, in the more neurotic type of children affected with this disease, there may be great peculiarity of temper with much display of emotion, difficulty in speaking, and sometimes even difficulty in swallowing.

The inevitable result of a prolonged attack of chorea is the exhaustion of the patient, and when there is sufficient restlessness to prevent sleep, and a tendency to maniacal manifestations, the case is always grave. Generally there are no sensory changes at all and the reflexes are either unaffected or slightly exaggerated. There is often a remarkable prolongation of the jerk, the leg remaining in the position it reaches with the stimulus of the tap. In chorea associated with hemiplegia the deep reflexes on the affected (hemiplegic) side are of course exaggerated, and there will probably be a well-marked extensor plantar response. Very rarely the affected limbs are tender to touch. The temperature, excepting in cases of great severity, and especially where the disease is associated with pregnancy, is usually normal, and generally pyrexia only accompanies maniacal excitement.

The cardiac condition should invariably be investigated. It is usual to find haemic murmurs, especially where the patient is bloodless, but mitral regurgitation and stenosis are common, indicating the presence of endocarditis not necessarily of a recent nature. Irregularity of the cardiac rhythm has been described in chorea, and it has been suggested that it is due to choreic contractions of the papillary muscles of the heart, and that the same explanation may account for the murmurs so often present; but it may be pointed out that

it is practically the voluntary muscles alone which suffer, and therefore this theory is untenable, and the cardiac irregularity is more probably the result of nervous exhaustion than of anything else.

Attacks of chorea usually subside in 6 to 12 weeks, but a relapse is not uncommon, and the causal factors in each case have much to do with the probable duration.

Diagnosis.—It is easy to recognise chorea by making the patient extend the fingers and attempt to keep them at rest, to put buttons into buttonholes, or to tie knots on pieces of string. The latter movements demand accurate effort and perfect co-ordination, and the attempt to perform them brings into prominence the jerking movements peculiar to the disease.

Care must be taken not to confuse *athetosis* with chorea. Athetoid movements are slow, rhythmic, and involuntary, confined usually to one hand, possibly to one hand and foot, generally associated with infantile hemiplegia or some cerebral lesion and often accompanied by mental debility, while the fact that the affected limb or limbs show a more or less distinct paralysis generally prevents any difficulty in the diagnosis, because paralysis is rare in chorea.

Prognosis.—The only serious cases are those in which the movements are very violent, and these are not infrequently associated with pregnancy, but the cardiac condition may give rise to anxiety, especially if endocarditis is present and is acute and ulcerative. Cases with temperature, and therefore with more or less delirium, are less favourable, but a great deal depends upon skilful nursing, so that a sufficient amount of food and sleep may be obtained by the patient.

Treatment.—The stock treatment for chorea is arsenic, and probably there is no remedy of such value, and which can be given for almost any type of the disease, the dose varying with the age of the patient, but generally beginning with 3 to 5 minims three times a day *with* meals. Isolation behind screens is also most effective. The peripheral irritant, if there is one present, should be removed, and attention directed to ensuring as much rest as possible, and the prevention of damage to the patient, should the movements be violent. Generally in these cases a mattress placed on the floor and surrounded with pillows is better than any attempt to restrain the patient in bed, while a good deal may be done to limit the

movements by keeping the patients absolutely quiet, free from excitement, and in certain cases isolated. Food should be given in easily digested and easily administered forms, and fish cream, pounded meat, mince, and beef-tea, and of course milk, are all admirable, although it should be remembered that fluids may be swallowed with greater difficulty than semi-solids.

As regards drugs, with the exception of arsenic it is difficult to know what to recommend. Certainly in rheumatic cases salicylate of soda is of great value, or aspirin in 10 to 15 grain doses, and bromides sometimes alone, sometimes associated with chloral, have proved efficacious. Antipyrine in moderate doses has acquired a considerable reputation, but it should be used with care. Remember that although arsenic is probably the best remedy, it should never be given unless under strict supervision, because arsenical poisoning, if not arsenical neuritis, may be produced, and may cause much and prolonged suffering. The dose, therefore, while increased gradually, should, in most cases, never exceed 10 minims of Fowler's solution three times in the day.

Where there is intense restlessness and delirium, cold sponging and the use of the ice-bag are advantageous, and to ensure a certain amount of rest hydrobromide of hyoscine (gr. $\frac{1}{100}$-$\frac{1}{75}$) may be given hypodermically, or chloroform may be administered as a general anaesthetic.

Conditions allied to Chorea

(1) Chronic Chorea, Huntington's Chorea

There is a form of chronic chorea which is frequently called Huntington's chorea, and which is characterised by irregular, choreic-like movements with difficulty in speech, gradual and progressive dementia, and a strong hereditary tendency. It begins at or after middle life, affects both sexes, and occurs in the members of a particular family. Huntington, who first described the disease, has records of a case in which preceding generations had been treated by his father and grandfather, who were physicians before him. The speech is peculiarly slurring and the gait unsteady, but the movements described as choreic are perhaps rather inco-ordinate, while the progressive failure of the mental powers is a typical feature of

all cases. The patients rapidly become insane, although the insanity rarely assumes, even for a time, an acute stage. Little is known of the pathological anatomy of the disease, owing to its excessive rarity, but atrophy of the cerebral convolutions, and in other cases scattered patches of sclerosis have been described. It is hardly necessary to state that the prognosis is bad, and that treatment is hopeless.

(2) Senile Chorea

Occasionally in old persons choreic movements develop as life advances. There is no hereditary tendency and no mental weakness, but the movements, once started, generally continue and probably annoy those persons with whom the patient comes in contact more than the patients themselves.

(3) Chorea Major, or Dancing Mania, or Epidemic Chorea

This is the original St. Vitus's dance, a disease, if it can honestly be so called, which dates back to the Middle Ages. It was associated with periods of excessive mental excitement, and occurred in pilgrims attending the chapel of St. Vitus in the Rhine provinces. Dancing, jumping, and gesticulations of all kinds were associated with great religious excitement, and the disease was in reality a type of hysteria.

(4) Saltatory Spasm

This is a peculiar hysterical manifestation to which, however, a special name has been given. It is very similar to the so-called chorea major, except that the individuals affected jump or spring in the most remarkable fashion. Often associated with this jumping movement is a tendency to explosive utterances or to the repetition of some single word.

(5) Tics or Muscular Spasms

(including habit chorea or habit spasm, convulsive tic, head-nodding, impulsive tic, etc.)

Tic is a co-ordinated purposive act produced in an involuntary manner.

Tic is a new name by which a widely different variety of

affections can be grouped together, and we are chiefly indebted to Miege for the name and definition. Reference has been made to spasm of the masticatory muscles, to facial spasm, and to the different forms of torticollis under affections of the 5th, 7th and 11th nerves respectively, and yet the paragraphs referred to might perhaps more correctly have appeared under the heading, "Tics." But as everything rightly mentioned under masticatory and facial spasm is not tic, the references to these spasms have been left as in the last edition, although torticollis, which is not merely a spasm of the muscles supplied by the spinal part of the spinal accessory, but also includes muscles supplied by cervical nerves might more correctly be described here.

(*a*) *Habit Spasm or Habit Chorea (Convulsive Tic).*—Under this heading are included the most typical examples of "tic." It is extremely common in nervous girls and children. It consists in certain movements of the head or face muscles, such as winking, nodding, twitching of the corners of the mouth, shaking movements of the head, movements of the ears and scalp, and similar peculiarities which become a habit to the individual. Many of these are learned in odd ways, and once acquired they are very difficult to get rid of. They are apt to be more marked when the individual is fatigued or nervous, and in the case of winking and other facial contortions may become a matter of great disquietude to the patient and to his or her friends.

Similar spasms of the muscles of the legs, and in particular the quadriceps extensor, the adductors, and the calf muscles may become a habit in certain persons, and these jerking movements are generally met with in neurotic individuals, and are increased by nervousness and by fatigue. Among the numerous varieties of tics might be mentioned tonic diaphragmatic spasm, clonic diaphragmatic spasm or hiccough blepharospasm, and many others.

(*b*) *Head-nodding and Eclampsia Nutans.*—Head-nodding is an affection closely allied to habit spasm, in which there are jerking movements of the head, and in the case of eclampsia nutans, bowing movements of the whole body. These movements are most common in delicate children, and rickets appears to be associated with it in certain cases. Nystagmus is not infrequently present.

(*c*) *Impulsive Tic.*—There is a group of cases in which, as the result of a peculiar psychical affection, the patient not merely suffers from involuntary muscular movements of the face and limbs, but in addition may give forth explosive sounds, sometimes in the form of a cry, bark, or shout, or else the sudden and explosive utterance of an oath or some obscene word. Beside these movements and explosive utterances there is also a peculiar mental phase generally taking the form of delusions and to this group of clinical features Gilles de la Tourette, has given the name of "Impulsive Tic."

Treatment of Foregoing Conditions.—Sedatives are not of much avail. When the spasms are wearing out the patient, the bromides, chloral, hyoscin, and similar drugs may be necessary to enable the patient to obtain the necessary amount of sleep.

Isolation and hydropathy are sometimes useful, singly or combined. The best treatment consists in practising rhythmic exercises *slowly* with the affected muscles, beginning with a few minutes twice or thrice daily and extending the period to ten, fifteen, or even twenty minutes at a time.

Sometimes a child may be educated to give up a habit spasm by patient and careful management. Hypnotism is disappointing as a curative agent, and is not to be commended for other easily appreciated reasons.

4. Occupation Neuroses

(Writer's Cramp, Telegraphist's Cramp, Pianist's Cramp)

A group of nervous affections incident to various trades and occupations, and characterised by interference with certain muscular movements which form a large part of the professional work of the patient.

Etiology.—It is due to the excessive use of the muscles which become affected, sometimes owing to improper use, perhaps in certain cases merely as the result of great stimulation of one or more groups of cells.

Pathology.—There is rarely any obvious lesion in the cells of the nervous system governing the affected muscles, but the following varieties of the affection, some of which imply

definite pathological change, at least in the muscles, may be met with: —

(1) The condition may be a failure of co-ordinating power for a particular action constantly required in the daily work of the patient.

(2) A disturbance of, rather than a failure in, co-ordination associated with excessive irritability of groups of muscles which are not required for the carrying out of the action in question, but which happen to be near them anatomically.

(3) An association of the first or second type with wasting or paralysis of the muscles affected.

(4) Cramps or spasms, excessive tremor, and not infrequently pain may be associated with the affected group of muscles to a greater or less extent, and sometimes the spasm involves muscles other than those required for the movements in question, so that in writer's cramp, one of the most common types of the disease, the pen or pencil may be struck through the paper or jerked out of the hand.

The **Clinical Features** have been already partially stated. In writer's cramp there is often spasm with pain, and very generally tremor involving the muscles, which may in time waste. The patient may bring into play other muscles so as to get over the difficulty, but these muscles also soon become affected. There is sometimes, although rarely, a little anaesthesia, and the skin occasionally shows definite trophic changes, the fingers become glossy, while sometimes vaso-motor phenomena develop, the patient becoming subject to chilblains. Electrical irritability is often diminished in the affected muscles.

While this description specially refers to writer's cramp similar trade spasms are met with in violinists, pianists hammermen, and telegraphists, especially those who work the Morse instrument, in the use of which Gowers estimates that over 50,000 similar movements are made by the patient in one day. Closely allied to these trade spasms is miner's nystagmus.

The **Diagnosis** of writer's cramp or palsy is easy. There is nothing with which one can confuse it, but, on the other hand, nervous people are apt to imagine that they are the victims of the affection.

The **Prognosis** is unfavourable, because even if the left

hand be used for writing, or the patient learn to write from the shoulder, it is by no means certain that the disease will not attack the newly educated muscles.

Treatment.—Certainly it is desirable to interdict all writing, and it might be added all occupation likely to cause other forms of trade spasm. Rhythmic exercises together with galvanism have been recommended, and attention should be paid to the patient's general health.

5. Tetany

It consists in tonic spasms, generally limited, and chiefly affecting the extremities.

Etiology.—There are several distinct varieties of tetany to which reference must be made.

A very mild type may be present in children as a sequel to severe diarrhœa, rickets, or any severe continued fever. The more serious forms are seen in adults. Tetany may develop as the result of prolonged lactation or after any debilitating illness such as a severe attack of a continued fever. It occurs in cases of dilated stomach, and a form of tetany is due to extirpation of the thyroid and parathyroid glands. It sometimes follows the administration of chloroform or ether, and a variety of drugs such as ergot, lead, morphine, etc. Lastly, epidemics of tetany have been described, the exact nature of which seems somewhat doubtful. Such epidemics occurred in Paris in 1855 and 1876, and were more prevalent during the winter months.

Clinical Features.—In cases associated with debility there are what have already been described as carpo-pedal spasms, in which the fingers are slightly bent at the metacarpo-phalangeal joints, the terminal phalangeal joints being extended, while the thumb is flexed into the palm of the hand. The pointed fingers (except for the flexed thumb) resemble the attitude of the accoucheur's hand. The wrist is often flexed. When the legs are involved they become extended, the feet arched, and the toes adducted. Sometimes the muscles of the face and neck are affected, and there may be trismus. These attacks occur in paroxysms, and a paroxysm may be produced by pressure on the affected part, either on the nerves or on the chief blood-vessels, while tapping the

muscles tends to throw them into active contraction. These spasms may last for minutes or for hours, and although they come on suddenly they pass off gradually. The electrical reactions are often greatly increased, and there may be a certain amount of pain associated with the condition.

Diagnosis.—It is easy to recognise the carpo-pedal spasms so common in children, but many authorities hold that they are hardly analogous to tetany. It is impossible to confuse tetany with *tetanus*, and it is rare to find a *hysterical* imitation close enough to render a mistake possible.

The **Prognosis** is favourable and the **Treatment** for mild cases in children consists in the alternate use of hot and cold baths, sometimes the administration of bromides, and in very severe cases chloroform anaesthesia. The ice-bag, general massage, and the various forms of electricity have often proved of value; in cases where the thyroid has been extirpated, thyroid extract should invariably be administered. Where tetany occurs in cases of dilated stomach lavage may be tried, but it is desirable to operate as soon as possible (gastro-enterostomy) for the relief of gastrectasis.

6. Paramyoclonus Multiplex

A remarkable disease, in which clonic contractions of certain muscles, or groups of muscles, of the limbs occur, more rarely of the trunk, the contractions being sometimes constant. These contractions are apparently the result of fright or great emotional excitement, and in certain cases the most violent movements are met with. There is occasionally a hereditary element. The disease is rare and may be associated with other affections of the nervous system.

7. Migraine or Hemicrania

Paroxysmal attacks of headache, generally unilateral, with sickness, vomiting, and often peculiar disturbances of vision.

Etiology.—The *predisposing* causes are hereditary, and especially in the neurotic or gouty. Commoner in women, the headaches may begin in childhood, and continue throughout life, and they are more frequent at the menstrual epoch. The *exciting* causes are:—peripheral irritation from nose, eyes

(astigmatism especially associated with hypermetropia), teeth, or pelvis; fatigue; mental emotions; overwork; and gastric or intestinal disturbance.

The **Pathology** is still doubtful. Neuralgia of the 1st division of the 5th nerve, vasomotor spasm followed by dilatation, and a "nerve storm" in a sensory centre are all theoretical explanations which have been suggested.

Clinical Features.—Often after premonitory symptoms of malaise, depression, etc., a severe pain begins in one temple or other part of the head, rapidly increasing in intensity, generally with nausea, and in a large number of cases with dimness of vision, flashes of light, and occasionally hemianopsia. These visual disturbances often precede the onset of pain and resemble the aura of epilepsy. Occasionally anaesthesia or aphasia temporarily develops much in the same way. The attack lasts for some hours or a day, and frequently passes off with vomiting. The attacks may be definitely periodic.

Prognosis.—It tends in many cases to recur, but does not endanger life. As age advances the attacks lessen in severity and may cease entirely.

Treatment.—Remove any exciting cause and study the constitution of the patient. For an attack, order rest in bed, promote vomiting in gastric cases by ipecacuanha or other means, and then administer antipyrine, phenacetin, salicylate of soda, or similar drug. Opium and the inhalation of chloroform often relieve, but are dangerous remedies. On the assumption of the correctness of the vasomotor theory, nitroglycerine has been suggested and found useful in some instances.

8. Neurasthenia

Loss of nerve energy, together with irritability of many nerve centres.

Etiology.—The *predisposing* causes are hereditary influence, unwholesome life as regards the mode of upbringing and education, excessive excitement, and mental overstrain. Certain races, as for example the Jews, appear to be specially predisposed.

The *exciting* causes are overwork, and particularly mental overwork, emotional excitement and nervous exhaustion, fevers, any severe illness, and dissipation. Perhaps the most marked

cases of neurasthenia are met with in persons addicted to the drug habit, and especially morphia and cocaine. A mental shock, such as a severe accident, is more likely to leave its mark upon patients predisposed to neurasthenia than on healthy strong-minded persons, but even in the latter it may start a definite neurasthenia.

Clinical Features.—All the manifestations which one anticipates finding in general debility may be present, and in addition there is pain in the back, a feeling of oppression on the top of the head, vague sense of discomfort, often palpitation and pulsating abdominal aorta, headache, and very generally disorders of the mind and memory and sometimes of the special senses, while sleeplessness is peculiarly common. Loss of the power of concentration is a very characteristic feature, and any sustained effort such as letter writing or adding figures produces quite disproportionate exhaustion. Considerable emotional excitement is not infrequently present, while a number of suicidal cases are in reality neurasthenics who have lost hope and become excessively depressed. Many persons suffering from neurasthenia are retiring in disposition, and dread society, and not a few of them imagine they suffer from some serious disease. The peculiarities of vision include tiredness after any short effort of sight, and there may be a similar peculiarity with regard to hearing. Many of the typical phenomena are associated with the spine, and these include the pains already described, which may be peculiarly severe in patients who have the spinal type of neurasthenia, a type very common after a railway accident. Not infrequently there are peculiar vasomotor phenomena such as general or localised sweating, reddening of the skin over the chest, throbbing of vessels, and many uncomfortable visceral sensations. A painful testicle in men and corresponding ovarian tenderness in women are not uncommon.

Diagnosis. — Neurasthenia may be confused with a number of affections, mental and organic, and the differential diagnosis is important. (1) Early cases of *general paralysis of the insane* may closely resemble neurasthenia, but one expects at least some of the evidences of organic disease such as the Argyll-Robertson pupil, the tremors of lips, etc., in patients suffering from the former. Lumbar puncture, with the great

increase of lymphocytes in the cerebro-spinal fluid, clinches the diagnosis in favour of paralytic dementia. (2) *Mental disease*, whether of adolescence or middle life, sometimes presents close resemblances to neurasthenia, but the age of the patient in cases of adolescent insanity and the hallucinations in all cases are distinctive. (3) Cases of what is called *psychasthenia* (Janet) to some extent resemble and were formerly included under neurasthenia, while others belong more properly to the insanities. Psychasthenia includes a wide range of imaginary, emotional, and mental disturbances, in fact phobias of all kinds. The subjects are usually young, but not always. The persistence of fancies, fears, etc., as a rule render the diagnosis of these cases from neurasthenia fairly easy.

The **Prognosis** is often favourable and depends largely on proper treatment, and above all upon an effort to improve the patient's nutrition.

Treatment.—A long period of mental and physical rest should be enjoined, and the patient's hopes of cure kept alive by persistent encouragement even in the most intractable cases. Tonics of all kinds should be administered, and when the physical strength has been sufficiently restored, active exercises suitable for the age of the patient should be ordered. Change of air and scene are most beneficial, and in many cases the Weir-Mitchell treatment is indicated.

Iron, strychnine, and arsenic are of great value, and any cause for the neurasthenia should be sought for and, if present, vigorously combated. This is specially necessary in cases where the disease is due to the drug habit.

Psychasthenia

This is a form of neurasthenia which has been recently dissociated from neurasthenia proper. In it the patient complains of headache and giddiness, and there is associated much fear of some impending disaster, the exact nature of which the patient often cannot explain. It occurs between puberty and middle life, and is not infrequently found in patients whose near relatives have suffered from epilepsy, neurasthenia, alcoholism, and other forms of nervous disease.

The headache differs from that which afflicts an ordinary person in as much as the pain may be described as being a

"feeling of tightness" or a "sensation of emptiness" in the head. The patient is often sleepless, tends to suffer from dyspepsia, and is readily fatigued by any exercise of mind or body.

The fears and anxieties from which the patient suffers, include a dread of doing physical harm either to himself or to some one with whom he comes in contact, the dread of being alone, crossing a wide street, or of being in a crowd, a church, a train, or in fact in any situation in which there is the remotest likelihood of personal disaster happening.

Dreamy states have also been described in which the patient complains of feeling dazed and of a want of reality so far as concerns his surroundings.

There are no obvious signs of any organic disease of the nervous system, and except for the dyspepsia, a tendency to palpitation, flushing of the skin, and some tenderness over the top of the head and along the spinal column there is nothing which can be called definitely pathological.

The **Prognosis** is favourable provided the greatest care can be taken of the patient, but in almost every case the cure is a very protracted one.

The **Treatment** consists in the Weir-Mitchell method of isolation and in the administration of the same kinds of drugs as are given for hysteria. Later, exercise and change of scene are found beneficial. In a few cases hypnotism has been described as beneficial but one dreads recommending it.

9. Hypochondriasis

A condition in which the sufferer imagines illnesses, magnifies any trivial complaint into a most serious disease, and regards the state of his health as demanding the most anxious care.

Etiology.—A neurotic family history, and mental depression from business or other worries, together with gout and dyspepsia, often predispose to this affection. Many of these patients are hysterical, some of them suffer from considerable moral obliquity, and many cases are to be met with in asylum inmates.

Clinical Features.—In young persons hypochondriasis is frequently associated with sexual irritability or masturbation,

and in other cases imaginary impotence is an important factor.

In middle-aged persons the illness imagined may take many different forms. Some patients really suffer from ill-health, and they add to the true symptoms a large variety of imaginary affections which render their lives almost unbearable. Pains in the back and legs, in the head, and in almost every part of the body, are extremely common. A number of these patients suffer from sluggish action of the liver associated with dyspepsia, and they may feel suppositi-tions internal parasites, have definite evidence in their opinion of the existence of cancer, and so forth. In older patients impotence and the imaginary ill-results of indulgence in vice dur ng early years may render much aid to the development of hypochondriasis. Syphilophobia is also a common manifestation.

The **Diagnosis** demands considerable care in order to prevent a real affection being treated as an imaginary one.

The **Prognosis** is often unfavourable: the symptoms imagined by the patient may persist for many years, sometimes complete recovery occurs, while in the worst type of cases the patient may commit suicide or may become insane.

Treatment.—Order change of air and scene in every case where such treatment is possible. Endeavour to divert the patient's attention from the affected organ or organs of the body, and abstain from informing him that his symptoms are purely imaginary. The first step towards successful treatment is to obtain the confidence of the patient.

10. Hysteria

A functional affection depending on instability of the nervous system, but which may depend to a certain extent on organic disease. There may be sensory, motor, vasomotor, psychic, and other manifestations.

Etiology.—In many cases faulty upbringing and education have much to do with hysteria. A child, weakly in body and who has never been controlled, may develop, towards puberty, the mental instability which causes this disease. Mental shock, debility from long-continued ill-health, the effects of alcoholism, syphilis, or tuberculosis, nervous diseases of many kinds, including epilepsy and insanity in the paternal

or maternal history, are peculiarly apt to lead to an ill-balanced mental equilibrium in the children. Intermarriage is, very probably, another important factor in many cases.

Over-education, faulty methods of education, excessive excitement of any kind, precocious interest in sexual matters, indulgence in masturbation, especially during early years, are all apt to give rise to hysteria.

Granted that the patient is a suitable subject hereditarily and personally for the development of the disease, it is easy to understand how some unfortunate love-affair, mental strain, anxiety, fright, fear of an examination, etc., may upset the patient's mental balance.

Pathological Anatomy.—Unfortunately we have little to say with regard to the pathology of hysteria. It is extremely probable that there are minute, but certainly as yet unrecognised, changes in the cells of the higher centres of the nervous system.

Clinical Features.—Many pages might be written without exhausting the innumerable features met with in hysteria. It is perhaps better, therefore, to describe a hysterical seizure, and then to refer under suitable headings to the most typical phenomena which are present in different cases.

The *paroxysm* generally begins with premonitory nervous phenomena, such as depression or excitement, and then a more or less typical hystero-epileptic seizure occurs. There are tonic and clonic spasms, with often extremely marked opisthotonos, but the clonic movements are very extensive and the patient —often a female—hits out at any one near at hand, while, though falling down, she generally selects a suitable spot for the seizure, where a sofa or bed is available. After the spasm stage has passed off, there is sometimes a stage of contortions and cataleptic poses, followed by a stage during which the patient demonstrates great emotional excitement, the face being expressive of intense love, pain, desire, hate, or other emotion. Lastly, a stage of delirium or hallucinations may develop. There are areas called hystero-genetic on the front and back of the body, the most important of which are shown in the figures. Pressure over certain of these areas may bring on an attack, or arrest it, if it has begun. It should be noted that only certain of these areas are active in any given case. Sometimes somnambulism catalepsy, and trance form a definite part of the attack. The paroxysms occur when the

patient is surrounded by friends or has a suitable audience, and they last for a long time, especially when much sympathy is shown towards the sufferer. In many cases the patient can prevent an attack occurring, but once it has commenced it appears to be beyond the control of the patient's will-power. It is between these paroxysms that the many sensory, motor, visceral, and psychical symptoms occur, and there are cases in which no typical hystero-epileptic paroxysms are present at all.

Before describing some of the chief phenomena associated

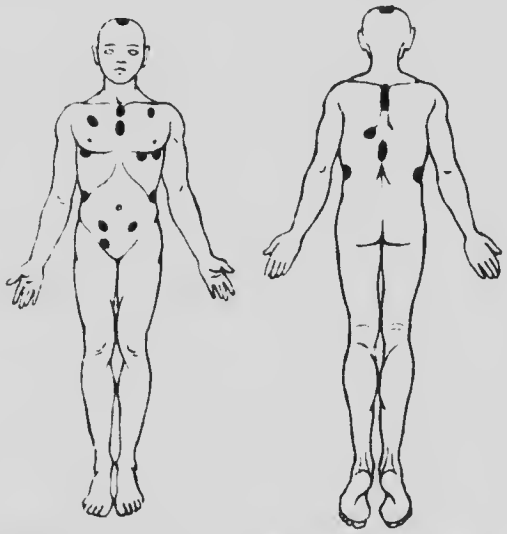

Fig. 55. Figures of chief Hystero-genetic Areas on anterior and posterior aspects of the body. (After Charcot.)

with hysteria it is desirable to state again that the patient invariably demands sympathy, and that without sympathy many of the most typical symptoms do not appear.

1. *Sensory Functions.* — Not infrequently the patient develops anaesthesia over one half of the body or over some part of it, such as the soft palate, and similarly, areas of hyperaesthesia may also be present. It should be noted that the hystero-genetic areas already referred to are areas of hyperaesthesia. The areas of anaesthesia may be of the "glove" type in the upper and the "stocking" type in the lower limb, that is, they are sharply limited by a line drawn round the limb, and the area involved does not correspond either to a nerve or to a segmental (cord) distribution, but are

obviously psychic in type. In addition it occasionally happens that certain joints become painful, the condition simulating inflammation or disease of these joints. It has been found possible, by means of magnets and other devices, to move the anaesthetic and hyperaesthetic areas from one side of the body to the other. Pains in the back, head, and indeed all over the body are not infrequent, and may be described by the sufferer as being of intense severity.

2. *Special Senses.*—Sight is not infrequently affected: the patient may be completely, although temporarily, blind, or may have one of various forms of scotoma, while colour-vision may be greatly limited. Sometimes the conjunctiva is peculiarly insensitive. The other special senses, hearing, taste, or smell, may also suffer.

3. *Motor Functions.*—All kinds of paralysis occur in hysteria, sometimes of a limb, sometimes merely of a group of muscles, while in other cases a hemiplegia is simulated with great exactness. The paralysis comes on suddenly and is accompanied by anaesthesia. The paralysed muscles do not waste, and the reaction of degeneration is not obtained where in cases of an organic nature these phenomena would be expected. There may also be paralysis with rigidity of the muscles, and it is easy to determine the functional nature of the affection, whether in arm or leg, by placing the hands over the muscles which ought to flex and extend the limb. Whichever group of muscles, whether flexors or extensors, the patient is told to put in action she *first* makes the antagonist muscles contract and *second* the prime movers (Beevor).

Aphonia or loss of the voice is perhaps one of the commonest forms of motor paralysis, and it is remarkable how in hysterical cases, recovery may follow some trivial procedure on the part of the physician, which could not possibly have any remedial effect whatsoever. Contractions and spasms are frequent, a limb may be flexed so as to be rendered almost useless, while the nails of the fingers may be firmly pressed into the palm of the hand. One of the most remarkable forms of spasm is that producing the so-called *phantom tumour* found in the abdomen, and resembling a neoplasm in appearance. Tremors of different degrees of coarseness are also common; the movements may be fast or slow, and may be kept up in a most convincing manner by the patient.

4. *The Reflexes.*—The tendon reflexes are active, but never absent. The superficial reflexors may be modified by any area of anaesthesia present, but there is no plantar extension (*i.e.* no Babinski sign). There may be some diminution of the corneal reflex, and the soft palate is generally anaesthetic. The organic reflexors are only functionally affected—retention of urine being often present, but often voluntarily.

5. *Visceral Phenomena.*—In this group there are many common hysterical manifestations. In the *Alimentary System* one finds typical spasm of the œsophagus, and the *globus hystericus* of which so many patients complain as passing up the gullet is a striking instance of this spasm. In not a few cases the patient describes the appetite as absolutely lost, while eating privately the food necessary to support life.[1] Phantom tumours have already been described, and it is probable that the remarkable rumblings, noisy eructations due to the retention of air behind the larynx or to swallowing it into the stomach, are generally of hysterical origin. Hysterical vomiting is not uncommon, but the food comes up with suspicious ease, although the nutrition of the patient suffers from the loss of food. It should not be forgotten that nervousness may bring on diarrhœa, and, therefore, considerable alimentary disturbance may be associated with hysteria. A remarkable phenomenon which may be noted here is excessive salivation, the patient sometimes secreting and expectorating an enormous amount of saliva.

In the *Circulatory System* palpitation is very common and may be accompanied by great discomfort and often cardiac pain; marked pulsation of the abdominal aorta is present in some cases, and another peculiarity is the absence of bleeding when an anaesthetic limb is pricked.

In the *Respiratory System* the hysterical cough is one of the best-recognised hysterical phenomena. It is a loud, barking cough much more annoying to those around than to the patient herself. Attacks of dyspnœa may sometimes develop, the rapidity of breathing being remarkable and often most distressing. The attack wears off after a short time, and is induced by excitement or any similar cause.

In the *Integumentary System* there may be urticaria or

[1] *Anorexia nervosa* is the term applied to absolute loss of appetite which may continue for long periods of time.

flushing of the skin either localised or very general over the trunk. In some remarkable cases stigmata or purpuric haemorrhages have appeared in the skin as the result of a hysterical attack. In very severe cases trophic changes may occur, and sometimes even the nails are shed.

In the *Genito-Urinary System* sexual irritability is not uncommon, while indecency and a love of obscene language and literature are occasionally present. The bladder often gives much trouble, the patient complaining of being totally unable to pass water, while in other cases polyuria is present after hysterical seizures.

6. *Mental Changes.*—The intense desire for sympathy already noted dominates the phenomena belonging to this group of symptoms. The patients are generally either excited or depressed: they will tell any number of untruths provided the sympathy desired is shown to them. They are unduly susceptible to mental impressions, and easily affected by any physical condition. They are childlike in their outbursts of temper and readily become passionate. The effect of alcohol is to produce a condition almost approaching mania, and the hallucinations associated with the attacks of hysteria may take many varied forms. The patient commonly manufactures ailments so as to attract the attention of those surrounding her, and not infrequently sugar or white of egg has been added to urine, or the patient has cut herself, introduced foreign bodies of different kinds into the nose, ear, vagina, etc., and in short has done everything she can think of to obtain the medical or other interest which her mental condition demands.

In some cases there is marked double consciousness. The patient being able to become what is really a second and very different personality, a personality in which she may think and act in a way quite unlike her usual self, in fact, a modified dual life like that of Dr. Jekyll and Mr. Hyde.

The emotional excitement, the stage of contortions and the stage of hallucinations so commonly associated with a hystero-epileptic seizure are all examples of mental phenomena. In the intervals between the attacks many psychical symptoms may develop, as, for example, astasia and abasia.[1]

[1] Astasia is inability to stand, abasia inability to walk, and yet with unaffected sensation, motor power, and co-ordination.

while the border-line between insanity and hysteria is often a very narrow one. Many patients develop hysterical manifestations in connection with religious excitement, and, just as in insanity, it is remarkable how a mixture of religious and sexual emotions may be present in the same case.

The temperature may become remarkably high, and apparent hyperpyrexia is a common manifestation of severe attacks, the patient in a mysterious way managing to raise the apparent temperature to 110°, 115° F., or even higher, although in most cases the hyperpyrexia is due to deliberate fraud.

It should be remembered that in patients suffering from true organic disease, and whose mental equilibrium has suffered from protracted ill-health, hysteria is not infrequently superadded to the clinical features characteristic of the disease in question.

Diagnosis.—As a general rule the patients are young females and possess the definite hystero-genetic areas already described. In the more severe cases anæsthesia of the posterior pharyngeal wall and other typical hysterical manifestations point to a definite diagnosis. A careful search must, however, be made for any organic lesion. Hysterical contractures and paralyses may often be proved to be functional by the administration of chloroform, and hysterical fits are much more lengthy than true *epileptic* seizures.

Prognosis.—Much depends on the zeal of the physician and the patient's obtaining good and efficient nursing. It is impossible to effect a rapid cure in most cases, but no case of hysteria, however severe, should be looked upon as hopeless.

Treatment.—The Weir-Mitchell method of treatment is probably the best. It consists in the isolation of the patient, depriving her of the sympathy which does so much to keep up the disease. At the same time she is kindly treated, is well fed, in fact is made to eat an excess of easily digested food, and is under the care of a strong-minded nurse or attendant. Massage is generally given and may be kept up daily for a length of time suitable to the patient's strength. Exercise is distinctly desirable where it can be taken, and electricity in all its forms, especially static electricity and faradism, have been found of great benefit. It is useless to contradict the patient by stating that symptoms

complained of are purely imaginary. Wise mental guidance and the cultivation of self-control and will-power are the proper lines of treatment.

The drugs generally administered consist of valerian, of which 30 minims of the ammoniated tincture may be ordered, or musk, and the ordinary tonics, but probably it is not so much the drug as the objectionable taste of certain remedies such as valerian and assafœtida which is responsible for the benefit obtained from their administration.

Unquestionably hypnotism is a powerful agent, but its use rather weakens than strengthens the patient's will-power and self-restraint. While, therefore, it may be prudent to treat the patient by suggestion, it is certainly foolish to resort to hypnotism in its ordinary form.

For hystero-epileptic fits the order to place the patient in a cold bath, should a subsequent fit be taken, or to pour cold water from a height on to her face, will often arrest a sequence of seizures, and the more uncomfortable the treatment the better it operates.

Prophylactic Treatment.—Parents should endeavour to enforce restraint on children, and particularly in the case of those who are delicate, physically or mentally. Self-control is probably better taught by parents and nurses during the early years of life than by any other means later on. Care should also be taken to prevent children allowing their minds to dwell too much upon one subject, however right and proper that subject may be, and variety of mental work with a sufficiency of physical exercise should be the régime for growing children. In older patients, and especially those who are delicate, it is wise to prevent an unhealthy imagination being wrongly stimulated by reading too many silly stories or by indulging in innumerable day-dreams, and the weaker physically and mentally these patients are, the more carefully regulated should their lives be as regards both mental and physical work.

11. Catalepsy

This is a rare affection. It is a hysterical manifestation in which the limbs remain for a long time in any position in which they are placed.

Etiology.—It forms a part of hysteria and may occur in all conditions in which hysteria appears, and it is a phenomenon sometimes present in meningitis and apoplexy. It is also the condition induced by hypnotism.

Clinical Features.—The attack is sudden; the patient usually a female, becomes rigid and may remain standing, sitting, or lying in exactly the same position until the seizure passes off. The limbs are at first so rigid as to be governed with difficulty, but soon they become plastic and remain for some time in any position in which they are placed. It is even possible to mould the features so as to make the patient's face demonstrate grief, surprise, or pleasure. The attack may last for several hours.

The **Prognosis** is that of hysteria and is generally favourable. The **Treatment** consists in anti-hysterical remedies. Give tonics and endeavour to improve the nutrition of the patient.

12. Trance

This is a condition closely allied to catalepsy in which for hours, days, or even months, the patient may remain rigid and to all appearance in a profound death-like sleep. The breathing is shallow, the pulse feeble, and nervous people imagine that death can be so closely simulated by trance that living persons may be buried alive. In the most marked instances trance has existed for years, and the patient is only awakened for brief periods, only more to lapse into slumber. Such cases are rare and not always terminate fatally.

In the more ordinary cases where feeding is difficult, where there is insensibility to pain and sometimes even temporary suppression of the urine, it is wise to send the patient to a home or asylum, for the attack closely simulates a hysterical manifestation. The treatment in an asylum not being complicated by excitement is generally promptly successful.

VIII. VASOMOTOR AND TROPHIC DISEASES, SOME OF WHICH ARE REFERABLE TO THE NERVOUS SYSTEM.

1. Angio-Neurotic Oedema

A transient form of local oedema, often hereditary, and which has in many cases a close relationship to attacks of colic.

Etiology.—It is sometimes associated with urticaria, sometimes with Raynaud's disease, and less frequently with purpura. It is impossible to account for all cases, and the condition may develop without apparently the remotest reason.

Pathological Anatomy.—It seems probable that it is due to a vascular lesion which permits of the transudation of lymph through the vessel walls, but the fact that it is a temporary condition greatly adds to the difficulty of discovering its exact nature.

Clinical Features.—A common site for the oedema is the face and especially the eyelids. The patient may awaken in the morning with both eyes so swollen as to prevent the possibility of the lids being opened, and similar oedema may involve the lips, cheeks, backs of the hands, the throat, and especially the larynx, the legs, and, indeed, almost any part of the body. The attacks are apt to come on at some particular time of the day, and they generally last for a few hours. There is, exceptionally, itching associated with the oedema, in which case the swelling is closely related to urticaria. The attacks may recur every few weeks or months, and they may be induced by a variety of causes. Very commonly severe colicky pains are present along with the oedema, in other cases vomiting, and in yet others joint-pains may be met with. The hereditary element in many cases is very marked, and several members of one family have died from oedema of the glottis of this nature.

The **Prognosis** is doubtful, but some patients are only slightly affected, and in them a cure may be anticipated.

The **Treatment** is eminently unsatisfactory. Give tonics, treat any anaemia or other constitutional condition present,

and try the effect of administering one of the nitrite group, and particularly nitro-glycerine.

2. Raynaud's Disease

(SYMMETRICAL GANGRENE, LOCAL ASPHYXIA)

As the title implies, this disease is a form of symmetrical asphyxia, sometimes resulting in gangrene; it is due to vasomotor spasm followed by vasomotor paresis.

Etiology.—The *predisposing* causes appear to be the existence of some nervous disease, severe anæmia resulting from malaria, scleroderma, and possibly injuries to the solar plexus and splanchnic ganglia, while the *exciting* cause is cold, often associated with excessive fatigue.

The disease is more common in women, it is sometimes directly associated with hysteria, and in a group of cases it is found to be congenital and sometimes hereditary.

Pathological Anatomy.—There is little doubt that a form of peripheral neuritis is present in not a few cases, and with this there have been described definite changes in the arteries and veins, which, however, appear to be more in the direction of spasm, followed by dilatation, than in any organic alteration. Recently the author found in a marked case in Dr. G. A. Gibson's wards, extensive endarteritis obliterans in the arteries of the affected limbs. No central lesion, common to a number of cases, has as yet been discovered.

Clinical Features.—The fingers, toes, and more rarely the nose and ears are the parts affected. They become during the *first stage* numb, waxy-looking, and bloodless—the condition generally described as *local syncope*. During this stage the fingers feel numb and may tingle, while at the same time the patient complains of chilliness or nausea. The stage lasts for a few minutes or a few hours, and, when it passes off, is apt to be replaced by the *second stage* or the stage of *local asphyxia*, in which the spasm of vessels relaxes, the vessels dilate and the affected parts become livid. This stage is accompanied by more or less pain, sometimes agonising in character, at other times merely a tingling sensation. In a limited number of instances a *third stage* is reached, namely, *local gangrene*, due as a rule to the circulation not becoming re-established, and the tips of the fingers or part of the nose or ears may slough off.

In certain cases either the first or second stage may be specially marked, while the third stage is rare. Paroxysmal haemoglobinuria is not infrequently associated with the attacks, and various other phenomena may develop; these include the presence of sugar in the urine, the association of scleroderma or erythema, and sometimes amblyopia and other eye changes. Nervous phenomena have also been described as being occasionally present, such as hemiplegia (sometimes with aphasia), coma, great depression, and more rarely mania. The blood in many cases is considerably altered, the corpuscles failing to run into rouleaux.

The typical attack, with or without paroxysmal haemoglobinuria, and with one or more of the other symptoms just mentioned, may be induced by exposure to cold, provided the individual is susceptible.

The **Prognosis** depends on the severity of individual cases. The disease may be eventually fatal from spreading gangrene or from intercurrent affections, but it is not infrequently amenable to treatment.

The **Treatment** consists in prophylactic measures to prevent attacks rather than in the use of any special curative drug. The clothing should be warm, the parts affected should be wrapped up in cotton wool, and probably there is no remedial agent so valuable as galvanism applied to the fingers or toes while immersed in water. Nitro-glycerine and the nitrite group should be tried, and for the pain associated with the second stage anodyne remedies are of value, and among the best of these are subacetate of lead lotion, and extract of belladonna in glycerine or vaseline.

3. Intermittent Claudication of Arteries

(INTERMITTENT LIMP)

An affection of the nervous system characterised by sensory and motor disturbances which are due to an insufficient supply of blood (claudication of arteries) when the individual indulges in active muscular exercise.

Etiology.—The causes of arteriosclerosis are generally present. There is always arteriosclerosis, and apparently spasm of narrowed vessels is induced by the exercise which constitutes the exciting factor in the case.

Clinical Features.—The legs suffer chiefly, either one or both. After a certain amount of exercise there is numbness, tingling, or pain, and paresis with cramps in the muscles follows. The arteries of the affected part cease to pulsate, and the leg and foot may become blue and cedematous.

Gangrene has developed in certain cases.

The **Prognosis** depends on the amount of benefit obtained by treatment, but the disease tends to progress.

The **Treatment** consists in rest, the administration of potassium iodide and nitro-glycerine, the use of massage, and where there are Raynaud-like symptoms the application of warmth.

4. Erythromelalgia

(Red Neuralgia)

This form of neuralgia has been mentioned along with neuralgia proper. There are three typical clinical features—*pain, redness,* and *swelling.* It affects the hands and feet and appears to be induced by cold, although this is not constantly the case. In one patient the attack was induced by continuously working in cold water during the winter months; in other cases cold relieves and hot weather makes the disease worse. The pathology of the condition is open to question, but it seems probable that debility together with a neurotic constitution are predisposing factors. There is certainly a vasomotor paresis, and changes have been described in the coats of the arteries. It is said to be the result of a neuritis, but this seems improbable.

In addition to the three phenomena just mentioned there are not infrequently definite trophic changes induced in the skin and nails of the affected limb, while the temperature of the part may be considerably heightened. Sometimes vesicles appear on the fingers or toes, but these are not common, and there may be considerable alteration in sensation both in the direction of anaesthesia but mainly hyperaesthesia.

Diagnosis.—The condition is not the same as *Raynaud's disease*: there is not the local syncope stage during which the affected part is deprived of blood as the result of vascular spasm, and in quite a number of cases of erythromelalgia the

affection is not symmetrical, and there is no haemoglobinuria, even occasionally, associated with it.

Prognosis.—Different cases vary with regard to duration. The attacks may pass off quickly, or on the other hand they may remain and recur, at intervals, for many years.

Treatment.—Probably rest and supporting the affected limb are the best methods to adopt. Care should be taken to prevent exposure to cold, where cold makes the condition worse. Various local remedies have been used for the relief of pain, such as the hypodermic injections of morphia or cocaine.

5. Scleroderma

This disease is certainly one coming under the group of tropho-neuroses. It belongs more properly to the Integumentary System, but inasmuch as, associated with the diffuse thickening of the deeper portions of the skin, there may be also similar changes in the muscles, a brief reference to the disease is desirable. It is more common in females than males, and is a disease of adult life. It may be limited, when the term *Circumscribed* has been applied to it, or it may be *Diffuse*, affecting the whole of the skin and greatly interfering with the usual movements.

In the *Circumscribed Type* the affected areas, often of small size, are brawny or hide-bound; they are sometimes pigmented, or in other cases show complete loss of colour. These patches are most frequently found on the neck or chest and have a peculiar life-history, sometimes appearing and then disappearing in a remarkably rapid way.

In the *Diffuse Type* the whole skin is hide-bound and is firmly adherent to the subjacent tissues. Movements become difficult, the face loses its expression, and various changes in the local circulation may be associated. Raynaud's disease has been described as coincident in several instances of diffuse scleroderma.

The skin is sometimes typically waxy in appearance. In other cases ulcerations or bullae develop, and there are a considerable number of modifications of the disease which may be met with.

The **Treatment** is unsatisfactory. Probably hot baths massage associated with injections of fibrolysin (♏ 40) every

2 or 3 days, either locally or into the buttock, and in certain cases the administration of thyroid extract will afford considerable relief.

6. Osteomalacia

(mollities osseum)

A rare disease which, in its typical form, occurs in adults, and consists in the softening of certain of the bones due to actual decalcification.

Etiology.—It is a disease of early adult life; it is commoner in women, and in them is associated in some way with pregnancy.

Pathological Anatomy.—The central parts of the affected bones are decalcified, leaving only an outer shell which readily yields under pressure, and may be easily cut with a knife.

Clinical Features.—The pelvic bones, the long bones of the legs, and to a less degree those of the arms, are specially affected, and marked distortion of the skeleton and shortening of the patient's stature result. The disease may be slowly progressive, lasting for years, and if the ribs and spinal column are involved, much misery ensues. Lime salts are said to be excreted in the urine during the progressive stages of the disease.

It is incurable, and there is no treatment, unless symptomatic.

A somewhat similar softening of the long bones and ribs may occur in the young, but it is extremely rare.

7. Hypertrophic Pulmonary Osteo-Arthropathy

This consists in enlargement of the hands and feet and the lower ends of the long bones, but the head does not suffer. It is associated with long-standing pulmonary disease, sometimes chronic bronchitis, empyema, asthma, or tubercular affections. The disease occurs in adults, and generally in males, and beyond the association of the enlargement of bones with the pulmonary affection the etiology is obscure.

8. Osteitis Deformans, or Paget's Disease

This is another rare disease, involving the shafts of the long bones, and also sometimes the cranial bones. The bones enlarge and soften, and become curved and misshapen from the weight of the body, frequently causing very great deformity. There is spinal curvature, the clavicles are prominent, the lower part of the thorax greatly widened and the abdomen diamond-shaped. It is probable that there is a relationship between this affection and malignant tumours of bone.

9. Achondroplasia

A remarkable form of dwarfism in which with ordinary size of head and trunk, the arms and legs are very short, the humerus and femur being proportionally shorter than the radius and ulna and the tibia. There is marked bending of the bones as in rickets, and the hands and feet are short and stumpy. The pathological process is mainly in the epiphyseal cartilages which are prevented from developing bone and hence the bones cannot grow in length.

10. Facial Hemiatrophy

In this peculiar affection there is wasting of one side of the face. The disease commences in childhood as a general rule. It may begin at one spot and gradually spreads, involving the skin, the bones, and to a less extent the muscles. This wasting is sharply limited to one-half of the face, and the upper part appears to be more affected than the lower. The hair and skin often change colour, and the teeth may fall out: sometimes the tongue participates in the hemiatrophy.

The pathology of the disease is very unsatisfactory. It has been considered to be due to an affection of the 5th nerve, and to involve specially the superior maxillary division.

A limited degree of asymmetry of the face is not uncommon, and is often associated with congenital wry-neck, scleroderma, infantile hemiplegia, and some other conditions.

DISEASES OF THE MUSCLES

1. MYOSITIS

There are three groups into which this disease may be divided: (1) *Primary Myositis*, which includes polymyositis, neuromyositis, tubercular and syphilitic myositis, and the myositis met with in trichinosis (page 203): (2) *Secondary Myositis*, which occurs in certain fevers such as small-pox, enteric, and typhus, and the infective myositis secondary to pyaemia, puerperal septicaemia, ulcerative endocarditis, erysipelas, etc.; (3) *Special forms of Myositis*, including *Myositis Ossificans* and *Myositis Fibrosa*.

(1) Primary Myositis

Acute Polymyositis has a very obscure etiology. It may be the result of a toxin, and it has been ascribed to eating crabs and possibly other shellfish in which decomposition had commenced. In diabetes and exceptionally in tubercular disease of the lungs and other organs, this form of polymyositis has occurred. Almost all the muscles of the body may be affected excepting the masseters and the eye-muscles, which usually escape. The muscular tissue has a somewhat yellowish colour, and many haemorrhages may be seen. The muscle fibres lose their striation, and there is a considerable interstitial infiltration of round cells between the muscle fibres. There is oedema especially of the extremities, and the muscles are tender to pressure, while the skin over them presents an erythematous blush. There is sometimes a definite purpuric or other rash. The disease begins insidiously with malaise, some rise of temperature, soon followed by cramp-like pains and tenderness in the muscles. Usually the limbs suffer first. The spleen is generally enlarged.

Atrophy of the muscles may follow with the usual

electrical changes, and the duration of the myositis may vary from one or two weeks to a period of months.

Diagnosis.—The muscular tenderness is distinctive, while in cases in which the trichina spiralis is responsible there is invariably abdominal pain during the period of boring, and oedema of the face and eyelids during the early stages of the disease.

Neuro-Myositis implies an involvement of nerves as well as the muscles. There is definite tenderness over the nerves, paraesthesia or anaesthesia, and loss of tendon responses. The muscles atrophy later, but are not generally so sensitive to pressure during the attack as in the previous type of myositis. The disease is a protracted one and recovery long delayed.

Syphilitic Myositis is divided into two forms, diffuse myositis and gummatous myositis. The diffuse form specially involves certain muscles such as the biceps, masseters, pectorals, and calf muscles. It consists in a plastic exudate deposited between the muscle fibres, and secondary cicatricial changes which cause marked muscular atrophy. The disease begins insidiously with tenderness upon pressure over the muscle and some redness of the skin. Movement of the muscle is painful, and contracture may result although it is stated that many cases appear to recover more or less completely.

Gummatous myositis is much more common. There is a small cell infiltration between the muscle fibres in the neighbourhood of the gumma, and in time cicatricial changes cause a corresponding amount of atrophy. A muscular gumma is only slightly painful, and it is found most commonly in the following muscles:—the quadriceps extensor, the calf muscles, the biceps, the fore-arm muscles, the masseters, and the pectorals.

The **Treatment** of syphilitic myositis consists in the administration of iodide of potash and in mercurial inunction.

(2) Secondary Myositis

Secondary Myositis demands only brief reference. There may be abscess formation due to suppurative organisms, or the change in the muscle may be rather degenerative than inflammatory. In most cases of infective secondary myositis the organisms in the muscle tissue are the same as those found in the primary lesion.

(3) Special Forms of Myositis

Myositis Ossificans is a rare disease in which the muscles actually undergo ossification. The disease begins much like an acute myositis followed by an increase of connective tissue, with great atrophy of the muscle fibres, and last of all, bone forms in the fibrous tissue. Males suffer more frequently than females; it generally commences in very early life.

The muscles swell, become painful, eventually hard, and later bone-like. Generally the back muscles suffer first. A varying degree of temperature may be associated with the preliminary myositis when it attacks each new group of muscles. A remarkable shortness of the toes and fingers, and more particularly the great toe, the thumb, and little fingers is not uncommonly present in these cases. No known treatment seems to be of any avail.

Myositis Fibrosa.—In this disease the muscles undergo fibrous change either locally or generally, and it is the general and progressive condition to which special reference is made here.

There is an increase of intermuscular connective tissue with atrophy of the muscle fibres, and this progresses until arms and legs and practically all the muscles of the body become completely fixed. There is no satisfactory explanation for the disease. The disease progresses slowly but without any arrest, and the prognosis is very hopeless.

By way of treatment, massage and electricity have been recommended. Baths of many kinds, and fibrolysin administered hypodermically may be of some avail, but one cannot refer with much enthusiasm to any known method of treatment.

2. MYASTHENIA GRAVIS

A rare disease, resembling bulbar paralysis, but without the anatomical changes generally associated with that disease. Careful investigation of an ever increasing number of cases has demonstrated a constant lesion, namely small deposits of lymphocytes in nearly all the tissues of the body. In a number of cases hypertrophy, degeneration, or new growth has been found in the thymus gland. The disease consists

in the rapid exhaustion of certain muscles by work and by faradic stimulation.

Etiology.—Possibly the etiological factors are wet and over-exertion. Pregnancy has a marked but temporary beneficial effect in many cases, but there is apt to be a fatal relapse after confinement.

Clinical Features.—The disease affects the muscles of the eyes the muscles of the face, the muscles of mastication and of the neck. later all the voluntary muscles may be involved. The affected muscles do not waste, at least to begin with, although they may do so later. The nasal speech, ptosis, and what is called the myasthenic reaction are very typical. This reaction implies the gradual loss of faradic excitability when the faradic current is applied to the affected muscles. Rest restores this excitability. The orbicularis palpebrarum is one of the muscles almost always markedly affected, the patient being unable to keep the eyes forcibly closed. The orbicularis oris is often weak and the patient cannot whistle. The muscles supporting the head are often affected, and indeed any or all the muscles of the body. The reflexes are generally preserved, and although neuralgia is not infrequent, other sensory changes are not commonly met with. Most of the recorded cases proved fatal, but it is a slowly advancing disease, even though incurable.

The **Treatment** consists in rest and massage. Strychnine and arsenic should be tried, but no remedy has so far proved of much avail.

3. MYOTONIA CONGENITA

(Thomsen's Disease)

A peculiar rigidity or tonic spasm of voluntary muscles which comes on when an effort is made to contract the muscles. The condition is transient, and only returns after a period of rest. It was described first by Thomsen, who was himself a sufferer from the disease, and in whose family it had existed for several generations.

Etiology.—The disease is a hereditary one. It shows itself in childhood, and males suffer rather more than females. It is commoner in certain countries, especially Norway, Sweden and Germany, but it is a rare disease.

Pathological Anatomy.—Portions of muscle which have been harpooned out during life show, according to some authorities, indistinct transverse striation, while the sarcolemma nuclei proliferate and the fibrils hypertrophy.

Clinical Features.—A tonic spasm of voluntary muscles occurs on commencing any muscular effort; it is best marked after rest, but the spasms soon pass off if the muscular effort is persevered in, and do not recur till after another period of rest. The spasm may begin when the patient endeavours to walk, but after trying for some little time he walks well for a long distance with no return of the spasm. The legs suffer more than the arms, and very rarely are the face muscles involved, although, apparently, in some cases few muscles can be said to be entirely free from the affection. The spasms increase under certain conditions, such as cold, damp, great heat, excitement, etc. Tapping a muscle causes a groove which lasts for some time, and, as one would expect, there is increased irritability to faradism, while what Erb has termed the *myotonic reaction* is generally present. This consists in the muscle, when stimulated by faradism or galvanism, contracting and relaxing slowly, a wave-like contraction passing from the cathode towards the anode.

There are no sensory changes, and rarely does the patient suffer more than slight, but constantly recurring, inconvenience from the disease.

Diagnosis and **Prognosis.**—The diagnosis is easy. Although cure never occurs, the condition is compatible with a long and useful life, and does not as a rule appear to become worse as time goes on.

The **Treatment** is *nil*. The patient learns how to avoid causing the spasms, and this he generally manages to do in an extremely adroit fashion.

4. AMYOTONIA CONGENITA

A very rare disease in which there is remarkable hypotonia—the joints being capable of being placed in all sorts of extraordinary positions owing to the relaxation of ligaments. The muscles are also flaccid, and the child cannot walk. The electrical irritability is diminished and the deep reflexes are lost. The disease is most probably allied to the myopathies.

5. THE MUSCULAR DYSTROPHIES

(Myopathy)

A group of diseases primarily of the muscles, and in which the nervous system only suffers secondarily, if at all. Most of these cases have a hereditary predisposition, and in all, whether the disease develops in early life or at puberty, the lesion is congenital. There is great weakness of the affected muscles, and some of them *atrophy* while others greatly increase in bulk although they weaken in strength (*pseudo-hypertrophy*).

(1) Pseudo-hypertrophic Paralysis

A form of muscular weakness, associated, however, with apparent enlargement of many of the weakened muscles and with atrophy of others. It has for long been considered as a disease essentially of the muscle fibres and not primarily of the nervous system, although in a few instances changes have been described in the motor cells of the anterior horn governing the affected muscles.

Etiology.—It is often hereditary, and is confined mostly to males, although it is transmitted by the female line. The disease commences soon after infancy, or between the ages of 4 and 6, although in some patients it may not develop till about the age of 15 or 16. In many cases no probable cause can be traced.

Pathological Anatomy.—At death the large muscles have generally become somewhat smaller than normal; the muscular fibres are narrowed, and are separated by fibrous tissue and large numbers of fat cells. During life, if a portion of affected muscle is harpooned out, it is found, on microscopic examination, to contain an excessive amount of fat, although the striation of the muscle fibres may in parts be indistinct, in parts fairly normal. Where there is indistinct striation, vacuolation may be present. In some cases there is a new formation of fibrous tissue, and not merely an increase of fat. The naked-eye appearance of the muscle shows an excess of fat.

It is difficult to offer a satisfactory theory to explain the condition. It probably implies an embryonic error and develops when strain is put upon the muscles.

As already stated, no constant changes have been noted in the nervous system to which the disease can in any way be referred.

Clinical Features.—The child appears to have well-formed muscles but the stout, muscular looking legs are surprisingly weak, and at an early period in the disease the child finds great difficulty in climbing stairs. The muscles affected first are the calf muscles, [illegible] muscles infra-spinatus, deltoid, etc., and to begin with these are usually hypertrophied, but the hypertrophy is accompanied by weakness and not greater strength. Other muscles waste, such as the latissimus dorsi, teres major, and the lower part of the pectoralis major, while the biceps and triceps sometimes hypertrophy and sometimes waste. Other muscles escape altogether such as the fore-arm and hand muscles, and the muscles which move the toes, but it is generally noted that the leg muscles as a whole are more affected than those of the arm, and that in the arm the muscles above the elbow are almost alone involved. The muscles originally enlarged often tend to atrophy later.

If the child be laid flat on the floor, and told to assume the erect posture, he has to "climb up his legs," the typical attitudes during the process being very suggestive of the disease. When standing, great difficulty is experienced in bringing the legs together if they are widely separated. The muscular weakness causes marked alteration in gait and posture, including lordosis and as time goes on contraction of the calf muscles occurs causing talipes equinus and later similar contraction of the flexors of the knee and elbow. The knee-jerks are gradually lost as the disease progresses, the electrical irritability to faradism and galvanism is diminished, but as a rule the organic reflexes do not fail until late in the disease. Sensation is unaffected.

Diagnosis.—The age of the patient, and the typical gait and appearance of the hypertrophied muscles, together with the history of several members of the same family being similarly affected, render the diagnosis easy.

Prognosis.—The disease is incurable, and generally progresses slowly, but surely, until the patient is bed-ridden. Death occurs from some intercurrent affection.

Treatment.—Little can be done by way of treatment, no drug affording any particular benefit. Attention to the general

health, massage of the muscles, and tenotomy when necessary, are all the methods of treatment which are commonly adopted.

(2) General Muscular Atrophy with no Hypertrophy

To this form the term "idiopathic muscular atrophy" is sometimes applied. The disease is congenital and starts in infancy. The wasting of the muscles is alone responsible for loss of tendon responses and great diminution of electrical irritability. There are no fibrillary tremors in the affected muscles which sharply differentiates this disease from the progressive spinal muscular atrophy of infants (Werdnig and Hoffmann). There are also cases of "idiopathic muscular atrophy" which commence in later life. Hypertrophy of muscle is not present in most cases.

(3) Erb's Juvenile Type

The disease begins at or soon after puberty. The muscles of the shoulder girdle suffer first, in particular the pectoralis major and latissimus dorsi. In the upper arm the biceps, supinator longus and generally the triceps atrophy, while the deltoid escapes or hypertrophies. The supra- and infra-spinati may also hypertrophy.

In the leg the glutei, the ilio-psoas, and most of the thigh muscles waste while the muscles below the knee escape. The trunk muscles and specially the erector spinae also suffer.

4 The Facio-Scapulo-Humeral Type of Landouzy and Déjerine

It begins at about the same age as the last type, and both sexes are affected. The face muscles suffer first and later those of the shoulder and upper arm. The orbicularis oris, the zygomatici, and often the orbicularis palpebrarum are affected, giving the face a peculiar expression. The lips project tapir-like, the eyes have a gaping appearance, and the features are immobile. The patient cannot whistle or blow out the cheeks. Either after or before the involvement of the face muscles the Erb type of shoulder and upper arm dystrophy develops.

Other types have been described, but the above are the most important.

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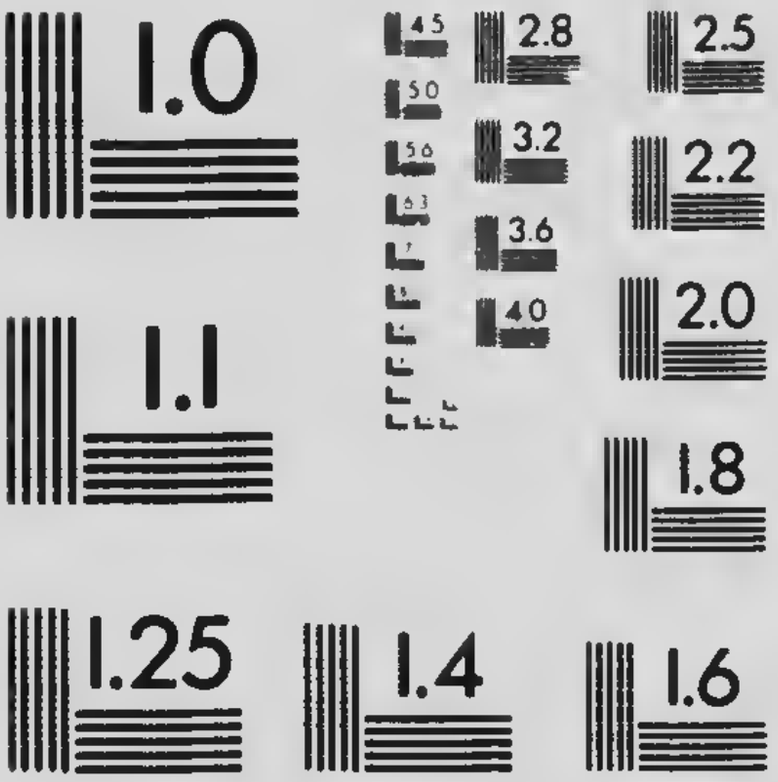

THE END

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