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MEDICINE

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MEDICINE

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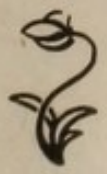


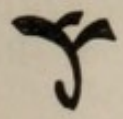
EDINBURGH:
E. & S. LIVINGSTONE, 17 TEVIOT PLACE

CATECHISM SERIES

MEDICINE

PART I.

SECOND EDITION



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CONTENTS

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MEDICINE

PART I.

I. SPECIFIC INFECTIOUS DISEASES OF TEMPERATE CLIMATES.

What is a Specific Infectious Disease?

A disease due to the action of a specific micro-organism or its toxin upon the tissues and fluids of the body, and to the reaction of the body to the invasion of such microbe or toxin.

What are the Common Characteristics of the Acute Infections?

They all run a typical course, differing in detail, but in each divisible into three periods :—

(1) A period following exposure to infection, during which symptoms are latent, or only vaguely indicated. This is the period of *Incubation*, in which the invading organism multiplies in the blood or tissues.

(2) A period of developed symptoms, characteristic for each disease, and expressive of the microbic intoxication and the bodily reaction. This is the *Fastigium*, of which the chief symptom common to the acute infections is *fever*, with the changes arising out of it.

(3) A period of *Defervescence* or decline, the result of neutralisation of the toxins or organisms by the development of *antibodies*. In this stage a rapid fall of temperature

(*crisis*) or a gradual fall (*lysis*) leads on to the establishment of convalescence.

What are the General Symptoms of the Febrile State?

1. Pyrexia, or elevation of temperature.
2. Rapidity of the heart's action, and alterations in the character of the pulse, which is often at first full and bounding, but later becomes soft and dicrotic.
3. Rapidity of respiration, and later a tendency to hypostatic congestion of the lungs.
4. Disorders of digestion :—the tongue is furred and the mouth dry ; appetite is impaired and thirst is pronounced ; there may be vomiting at the outset ; and constipation is usual.
5. The skin may be dry or moist ; in many cases there is a characteristic rash.
6. Headache, insomnia, and delirium show the implication of the nervous system.
7. The urine is concentrated, high-coloured, and of high specific gravity. On standing it throws down a precipitate of pink urates ; and it may contain albumin. Urea is increased, and chlorides are diminished.

What Morbid Changes characterise the Febrile State?

1. The blood is dark and fluid, and there is destruction of its red cells. The leucocytes are usually increased (*leucocytosis*), but in some fevers, such as enteric, they are diminished (*leucopenia*).
2. The muscles show granular degeneration (*cloudy swelling*). The cardiac muscle is softened, and the heart may be dilated.
3. There is also softening of the viscera, which are at the same time congested, particularly the spleen, liver and kidneys. Congestion of the lungs, above referred to, is aided by the patient's dorsal position (hypostatic congestion).

What are the Terms applied to the Different Degrees of Temperature?

1. Collapse temperatures: below 96·8° F.
2. Subnormal temperatures: between 96·8° and 98° F.
3. Normal temperatures: 98·4° or 98·6° F.
4. Subfebrile temperatures: 99° to 101° F.
5. Moderately febrile temperatures: 101° to 103·5° F.
6. Highly febrile temperatures: 103·5° to 105° F.
7. Hyperpyrexia: above 105° F.

(4–6: Pyrexia.)

What are the Different Types of Fever?

1. Continued fever: in which the temperature is persistently above normal, and has comparatively slight daily fluctuations.

2. Remittent fever: in which, though the temperature never falls to normal, the morning temperature approaches it, and the daily fluctuation is considerable.

3. Intermittent fever: in which, for some part of each period of twenty-four hours, the temperature is normal or subnormal.

What is the Typhoid State?

A condition occurring in the later stages of the graver acute infections.

Describe its Characteristic Symptoms.

There is marked prostration, frequently with tremor or subsultus tendinum; the patient tends to sink to the foot of the bed; delirium is of a low, muttering type, or there may be a semi-comatose condition with dilated pupils (*coma vigil*); the tongue is dry, brown, and tremulous; the lips and teeth are coated with sordes; and the pulse is rapid and soft. Picking at the bedclothes (*carphologia*) is a sign of bad omen.

To what is a Natural Recovery from an Acute Infection due?

To an increased capacity of the cells or fluids of the body to neutralise the toxins or destroy the organisms. This capacity is acquired in the course of the disease, and is known as *Acquired Immunity*. Its duration is for some diseases brief, for others lengthened, and for some it may be life-long.

What are the Varieties of Immunity?

Natural immunity, by which is meant a condition of insusceptibility to a particular infection, however frequent may be the exposure; and *acquired* immunity. Immunity may be acquired as the result of an attack of infectious disease, or artificially. Artificial immunity may be *active* or *passive*.

How is Active Immunity to a Particular Organism produced?

1. By the repeated injection of attenuated living cultures of the organism, or of sub-lethal doses of non-attenuated cultures.
2. By the repeated injection of the dead organisms in gradually increasing doses.
3. By the repeated injection of sub-lethal doses of the filtered toxins.

How is Passive Immunity produced?

By injections of the *serum* of an animal which has already been actively immunised. Such a serum, if taken from an animal which has been immunised by the injection of toxins, is *antitoxic, i.e.* it protects against the toxins, but has little bactericidal effect. If the animal has been immunised by the injection of organisms, its serum is *antibacterial* or *bactericidal*, but confers little protection against the toxins.

What are the Substances in Immune Serum which give rise to its Protective Properties?

They are known as *antibodies*; and the toxins or organisms upon which they act are called *antigens*. In the case of toxins the antibody is *antitoxin*; in the case of organisms there are several antibodies, bacteriolysins, *opsonins*, *agglutinins*, and precipitins.

Have the Bacteriolytic Substances an Independent Action?

No; they act only in the presence of *complement*, an unstable body contained in normal serum. The effect of an immune serum depends upon the *immune body* (bacteriolytic substance) which it contains, in conjunction with normal complement. The immune body *links* this complement to the organism to be attacked, and hence is also called *amboceptor* or *copula*.

How do Opsonins produce their Effect?

They are substances in the serum which *sensitise* organisms introduced into it in such a way that they become more readily ingested and destroyed by the polymorpho-nuclear leucocytes (phagocytes). This process is known as phagocytosis. Opsonins exist in normal serum, but are more numerous in immune serum.

What is the Action of Agglutinins?

Agglutinins are substances developed in an immune serum. When such a serum is added to an emulsion of the living organisms, these, instead of remaining separate, tend to run together and form clumps. The serum of those who suffer from a particular infection agglutinates the causative organism.

What is the Action of Precipitins?

These bodies, also found in an immune serum, precipitate the bacterial substances from the clear filtrate obtained

from a culture of the organism against which the animal has been immunised.

Are any of these Facts of Immunity applicable to Treatment?

Passive immunity may be produced by the injection of an immune serum, active immunity by the injection of dead bacterial cultures (vaccines). On these facts are based serum treatment and vaccine treatment. The sera used in treatment are for the most part antitoxic (diphtheria, tetanus); vaccine treatment is bactericidal, and depends for its effect largely upon the stimulus which the introduction of dead organisms gives to the formation of opsonins, and therefore to phagocytosis.

How do the Reactions of Immunity aid in Diagnosis?

The agglutinin reaction is that which is chiefly employed for this purpose. The serum of a patient suffering from enteric fever, Mediterranean fever, or whooping cough, for example, agglutinates the organisms in an emulsion made from a culture of the appropriate bacillus or coccus.

What are the General Principles of Treatment in the Acute Infections?

1. The patient must be isolated to prevent the infection of others. He should have a large, well-ventilated room, preferably at the top of the house, and all furniture that can be dispensed with, all hangings and carpets should be removed from it. His excretions must be disinfected. All dishes, etc., which are used for his food or drink, must be kept for himself, and disinfected before they leave the room. In all the severe infections a good nurse is essential.

2. To stimulate the production of antibodies, the patient's strength must be in every possible way maintained. This is to be done mainly by diet, which should be light but nutritious. Milk is the staple article of food in all such cases: but white of egg, beef-soups, and jellies may in

many instances be added. Stimulants should be used only to meet special indications, not as a matter of routine.

3. Elimination of the toxins should be promoted. The channels of elimination are the bowels, kidneys, and skin. Laxatives, diuretics, and diaphoretics are therefore the main drugs employed, but they should never be used to a depressant extent.

4. Fever that is moderate in degree and not unduly prolonged need not be interfered with; it is the expression of the protective reaction. Measures for the relief of headache, light coverings to the bed, and tepid sponging are sufficient. Where hyperpyrexia is present, or where the fever is so prolonged as to be itself a danger, hydrotherapy—cold sponging, cold applications to the head, cold packs or baths—is better than drug treatment. Do not use depressant antipyretics such as antipyrin (phenazone); if drugs must be used, quinine is the most reliable.

5. Wherever possible, endeavour to neutralise the specific poison by specific therapy—diphtheric antitoxin, antitetanic serum, etc. The use of vaccines in acute infections is combatted upon theoretical grounds, but in some cases would seem to have given striking results.

How are the Principal Febrile Diseases divided?

Into (*a*) *continued fevers*, such as enteric, typhus, and relapsing fever; (*b*) eruptive fevers or *exanthemata*, such as small-pox, scarlet fever, etc.; and (*c*) *remittent* or *intermittent* fevers, such as malaria and pyæmia.

ENTERIC OR TYPHOID FEVER.

Define Enteric Fever.

It is a specific infectious disease characterised by a protracted course of twenty-one days or more, accompanied

by diarrhœa and by the presence of a scanty eruption appearing in successive crops, and ending by lysis. There are characteristic ulcerations of the small intestine, and the spleen and mesenteric glands are enlarged.

What are the Characters of the Organism?

The *bacillus typhosus*, or bacillus of Eberth, is a short, thick, mobile bacillus with rounded ends and numerous flagella. It has a negative reaction to Gram's stain. It occurs in the rose-spots, the stools, the urine, and can be cultivated from the peripheral blood, at least in the earlier stages of the disease. After death it is also to be found in the intestinal ulcers, mesenteric glands, spleen, and gall-bladder.

How does the Organism enter the Body?

Principally by way of the alimentary tract. Infected stools, disposed of by imperfect drains, contaminate the rain-water of the soil, and may thus pollute a water supply. Dairy utensils washed by such water become infected, and the milk they contain is then a source of disease. Uncooked vegetables grown on infected soils, shell-fish from polluted river beds, food on which flies have rested after alighting on the excreta, may also spread the disease, and infection may follow direct contact with the stools or with infected linen. *Milk and water*, even in the shape of ice, are the most frequent vehicles.

Are there any other Sources of Infection?

If the exposed stools become dry, their dust may be disseminated by the air. Those who have recovered from enteric often harbour the bacillus for long periods, especially in the gall-bladder, and their stools may be a constant source of danger. Such individuals are called "typhoid carriers."

What are the Predisposing Causes?

The disease occurs at all ages, and in all climates and seasons, but it is most frequent in the autumn, and in young adults.

Describe the Morbid Changes.

Besides those due to the febrile state, there is an inflammatory condition of the lower part of the small intestine, with ulceration of Peyer's patches and of the solitary follicles.

In the first week, Peyer's patches are swollen, infiltrated with leucocytes, elevated above the surface of the bowel, and fawn-coloured. The infiltration may extend to the deeper coats.

In the second week, sloughs form upon the patches.

In the third week, the sloughs separate and leave typhoid ulcers, the long diameter of which lies in the long axis of the bowel. The edges are undermined, and the floor of the ulcer may be the submucous, muscular, or peritoneal coat. Healing, which is often slow, begins about the end of the week. It does not cause constriction of the lumen. Perforation is common.

The *mesenteric glands* are swollen and tender, and may caseate. The *spleen* is enlarged and soft.

What are the Symptoms of a Typical Case?

After a lengthy *incubation period* (ten to fourteen days), the disease sets in insidiously. Malaise, slight headache, slight diarrhœa, and increasing weakness are the first symptoms, and there may be epistaxis. The patient at last takes to bed, and when the disease is established, the main symptoms are diarrhœa with characteristic stools; a distinctive rash, which may be very scanty; pain and tenderness in the right iliac fossa, where there is often gurgling; enlargement of the spleen, emaciation, increasing prostration, passing, in grave cases, into the typhoid state, and fever.

What is the Course of the Temperature?

In a typical case the temperature rises gradually during the first week, the height in the evening being about 2° F. higher than in the morning. During the night there is a fall of about 1°, and thus the temperature each evening is about 1° higher than that of the evening before. At the end of the week a level of 103° or 104° is reached in the evening, and this is maintained through the second week with small daily fluctuations. In the third week the evening temperature begins to fall, the chart assumes a remittent type, and the normal level is reached about the end of the week. Defervescence takes place by *lysis*. In the fourth week the temperature is subnormal. The pulse rate in enteric is less rapid than the height of the fever would suggest. (*See* Chart p. 90).

Are there any Deviations from this Type?

In a few cases the onset is sudden, generally with head symptoms; in some the fever may abort at the end of the second week; in many its duration is considerably longer than three weeks. Relapses and recrudescences are common.

What are the Characters of the Rash?

It appears comparatively late, from the sixth to the tenth day, and may be delayed till the twelfth. It consists of small very slightly elevated rose-coloured spots, fading on pressure, and seated on the abdomen and chest. They may also appear on the back, and sometimes there only. The spots come out in successive crops, each of which lasts about four days, and they may continue to appear till the end of the disease. They may be very scanty, and *the rash is often absent.*

Describe the Stools of Enteric Fever.

These are at first brownish or greenish, and of a semi-solid consistency, but after a few days they assume a typical "pea-soupy" appearance, are loose and bulky, and have a characteristic odour. In the later stages they may contain sloughs. When hæmorrhage has occurred they become black, sticky, and tarry in appearance. In some cases constipation takes the place of diarrhœa.

Is there Leucocytosis in Enteric Fever?

No; the leucocytes are *diminished* in number (*leucopenia*). Leucocytosis suggests the presence of an inflammatory complication.

What are the Chief Complications?

Hæmorrhage from the *bowel*: other hæmorrhages (epistaxis) may also occur.

Perforation of the bowel, leading to peritonitis; or peritonitis without perforation.

Other complications are:—meteorism or tympanitic distension of the abdomen; bronchitis and broncho-pneumonia (lobar pneumonia may occur); thrombosis of the femoral vein in the late stages; neuritis, and sometimes meningitis; bed-sores; and early development of the typhoid state.

How do you recognise Hæmorrhage from the Bowel?

The tarry appearance of the stools, already described, is characteristic; it may be preceded by a pinkish tinge. If a severe hæmorrhage has occurred, there may be a sudden fall in temperature before any blood is present in the stools, the pulse becomes unduly rapid, and the face is pale.

What are the Symptoms of Perforation?

Intense abdominal pain, setting in *suddenly*, with tenderness, rigidity of the abdominal muscles, and abdominal distension. The percussion note is tympanitic all over, and the hepatic and splenic dulness may disappear. The respiration is hurried, and thoracic in type; the face is anxious; the pulse is rapid and wiry; and there are signs of shock.

What other Diseases may resemble Enteric Fever?

Acute miliary tuberculosis, malignant endocarditis, pyæmia, and influenza.

In acute miliary tuberculosis the fever is irregular, diarrhœa is usually absent, there are no rose spots, and there may be localising signs pointing to the lungs or meninges. Where there is meningeal tuberculosis, retraction of the head, squinting, or other cranial nerve paralysis, slowness or irregularity of the pulse, vomiting, and retraction of the abdomen are to be found.

In malignant endocarditis and in pyæmia the temperature is irregular, and of a remittent or intermittent type. In endocarditis there are cardiac murmurs, irregularity of the heart, and evidence of embolism in one or other organ. In pyæmia there are abundant sweats and rigors, often with jaundice, and local signs of abscess.

The onset of influenza is more sudden, the temperature tends to fall after the first few days, the spleen is not enlarged, and there are no rose-spots.

What are the Special Diagnostic Signs of Enteric Fever?

In the early stages, the cultivation of the bacillus from the blood, stools, or urine; later (after the seventh day), Widal's reaction.

What is Widal's Reaction?

It depends upon the agglutination of the typhoid

organisms by the serum of a patient. The blood is collected from a puncture in the finger or ear by means of a small pipette or Wright's capsule, in which it is allowed to coagulate. The serum is then blown out, and diluted with sterile bouillon to the desired degree (1 in 20 to 1 in 100). A loopful of the dilution is then mixed with a loopful of a thin emulsion of a young culture of living typhoid bacilli, and the mixture is examined as a hanging-drop preparation under the microscope. If the reaction is negative, the organisms move freely about the field ; if positive, they become agglutinated into clumps. The reaction does not occur till the end of the first week, and a negative reaction does not absolutely exclude enteric fever. Widal's reaction may persist for months or years after convalescence.

What is the General Treatment of Enteric Fever?

It is mainly dietetic. Strength must be maintained by feeding, but nothing must be given to increase the risk of perforation. Milk is therefore the chief food, and may be given in doses of ℥v every two hours, diluted with barley water or lime water. Soda water tends to produce flatulence. *The milk must not be given to relieve thirst*, for which water is used, but at regular intervals, and in definite doses. If curd is passed in the stools, the milk must be peptonised. Beef-juice, beef-tea, chicken-broth, etc., may be given if diarrhœa is not troublesome. *All food should be strained.*

If fever is high it may be met by cold sponging, cold packs, or cold baths, repeated systematically ; or quinine may be used.

Intestinal antiseptics have no influence on the course of the disease, but they reduce the offensiveness of the stools and check meteorism. Salol, calomel in minute doses, β-naphthol, etc., may be used.

Diarrhœa must be checked if there are more than four

motions daily. Bismuth and opium, or starch and opium enemata may be given.

Constipation lasting more than two days should be met by a simple enema. *Purgatives must not be used.*

Meteorism may be relieved by turpentine enemata.

How would you treat Hæmorrhage from the Bowel?

By absolute rest on the back, ice-bags over the cæcum, ice to suck, morphine hypodermically, or opium, lead acetate, or calcium lactate internally. Ergotine or adrenalin may also be given. Feed, if necessary, by the bowel.

What is the Treatment of Perforation?

Immediate laparotomy affords the only real hope : if for any reason this is impossible, opium in full doses, ice to the abdomen, and rectal feeding are the alternatives.

What is the Specific Treatment of Enteric Fever?

Both serum treatment and vaccine treatment are advocated, but as yet their value is unsettled. Chantemesse's serum is employed in France, and vaccines seem to have given good results. They are undoubtedly useful in prophylaxis.

In all cases of enteric fever the excreta must be rigorously disinfected, and the linen must be changed and disinfected as soon as it is soiled. Urotropin may be given throughout the disease as a urinary antiseptic, but the urine must none the less be disinfected before it is disposed of.

TYPHUS FEVER.

What is Typhus Fever?

An acute specific infection, characterised by a sudden onset, with marked nervous symptoms; a macular rash; and a termination by crisis after a fortnight's course.

Mention the Etiological Factors.

The organism is not yet known, and is probably ultra-microscopic. The disease is highly contagious, and may apparently be transmitted by the body-louse. Poverty, dirt, overcrowding, famine, and defective hygiene are contributory causes. Those between ten and thirty are oftenest attacked. Infection is supposed to be transmitted by emanations from the skin and breath.

Owing to improved sanitation, typhus is now rare.

Describe the Morbid Changes.

They are those found after death from pyrexia (*vide supra*). Fatty degeneration of the heart is common. The petechial rash remains after death.

What are the Chief Symptoms?

After an *incubation period* of about twelve days, the illness sets in suddenly, often with a rigor or rigors. Intense headache, nausea and vomiting, and rapid rise of temperature are also present. The pupils are contracted, the tongue furred, the bowels constipated, and the skin gives off a mousy odour. Delirium comes on early, and is at first noisy. The face and conjunctivæ are congested. The rash appears on the fifth day, and the patient about this time has a dull, heavy appearance, and becomes apathetic. In the second week the typhoid state may appear; the delirium is of the muttering type, and the

pupils are now dilated; urine may be retained or the sphincters paralysed; and death may be preceded by coma vigil, or may be due to a complication. When recovery takes place, the temperature falls *by crisis* (perhaps after an *epicritical* rise) about the fourteenth day, and this is accompanied by profuse sweating and the so-called critical diarrhœa. Convalescence is rapid.

What are the Characters of the Rash?

It consists of two elements:—(1) a dusky-red "subcuticular" mottling which fades on pressure (*measly eruption*); (2) papules, at first like rose-spots, but after a day or two becoming *petechial.* The petechiæ do not disappear on pressure or after death. The eruption appears first on the chest and abdomen and the back of the hands and wrists, spreads over the trunk, and extends to the extremities.

Describe the Course of the Fever.

The temperature rises abruptly, and even on the first day may reach 103° F. or more. In the course of the first week it reaches perhaps 105° or higher, and remains at that level, with little morning fall. In the second week it may fall slightly, but often a slight recrudescence precedes the crisis. The disappearance of fever is abrupt, the temperature falling on the fourteenth or fifteenth day as much as 6° or 7° F. (*See* Chart p. 91.)

Mention the Chief Complications.

Retention of urine; bronchitis, broncho-pneumonia, and hypostatic congestion; bed-sores, and gangrene; parotid bubo and suppuration of the joints; femoral thrombosis.

What is the Treatment?

That of fever in general, with special attention to head symptoms (shave the head, cold applications, ice), retention

of urine, and the prevention of bed-sores. Free ventilation is essential, and stimulants may be called for. Systematic hydrotherapy (packs, baths) is advised by some, but milder measures (sponging) are more satisfactory.

RELAPSING FEVER.

Give a Definition of Relapsing Fever.

A specific infectious disease, due to the *spirochæte Obermeieri*. It is characterised by fever terminating abruptly on the sixth or seventh day, and followed after a week's interval by a relapse of about four days' duration.

Describe the Causative Organism.

The *spirillum* or *spirochæte Obermeieri* is a long slender spiral with pointed ends, varying in length from 10 to 40 μ, and in constant lashing movement. It is found in the blood during the febrile attacks, and in the spleen in the interval.

What are the other Etiological Factors?

The disease occurs epidemically in circumstances of poverty, dirt, overcrowding, and especially *famine* ("famine fever"). It is endemic in parts of India, but does not now occur in Britain. It is contagious, and may be carried by *fomites* (clothing, etc., which has been in contact with the patient). It is probably transmitted by the bed-bug.

What are the Morbid Changes?

Those of fever (*vide supra*), with marked enlargement of the spleen; there are no specific lesions.

Describe the Symptoms.

The *incubation period* varies from one to twenty-one

days, but is usually less than nine. The onset is sudden, with rigor, headache, backache, and rapid rise of temperature, which may reach 104° F. on the first day. The pulse is very rapid, the tongue is furred, and thirst is great. The spleen is enlarged and tender; jaundice is frequent. Towards the end of the first week the symptoms become more severe; delirium is marked, and the fever rises still higher (even to 106° or 107° F.). On the seventh day or earlier a sharp crisis occurs; the temperature falls to normal, convalescence is rapid, and the patient may be out of bed in four days. About the fourteenth day, however, a relapse occurs, which repeats the features of the first attack, and ends by crisis, usually after four or five days. The mortality is comparatively low. There is no specific rash. (*See* Chart, p. 92.)

What are the Complications?

Pneumonia, dysenteric diarrhœa, jaundice, ophthalmia, and hæmorrhages in pregnant women. Abortion usually occurs.

What is the Treatment?

The general treatment of fever; stimulants may be necessary for collapse following the crisis. Salvarsan intravenously (grm. 0·2 to 0·3) is said to arrest the disease and prevent relapse.

VARIOLA or SMALL-POX.

What is Small-pox?

An acute infectious disease of sudden onset, with a characteristic rash appearing on the third day in the form of vesicles which afterwards become pustular.

What is its Etiology?

The specific organism is not certainly known. Small-pox is highly contagious, spreading rapidly from person to person among the unprotected. It is transmitted by the breath, and also by the contents of pustules, fragments of scabs, or fomites. It is infectious before the eruption appears, and the body is infectious after death. It attacks all ages and both sexes; the fœtus of an infected mother may be infected. A recent successful vaccination confers complete immunity, an old or partially successful vaccination modifies the attack.

What are the Morbid Changes?

Save the rash, there are no specific morbid phenomena. The changes are those of fever, and in hæmorrhagic small-pox hæmorrhages may occur in internal organs, into the skin, or upon mucous surfaces.

The *rash* has three stages:—

1. The papular stage: proliferation of the cells of the rete mucosum, accompanied by a serous exudation, which compresses the cells into trabeculæ, and thus leads into—

2. The vesicular stage. The vesicles are divided by the trabeculæ into loculi. Coagulation necrosis occurs at their centre, and they thus become umbilicated. The cutis vera shares in the inflammation, and is infiltrated by leucocytes. Thus arises—

3. The pustular stage. The pustule dries up, with the formation of a scab, and when this separates a permanent pitting is left behind.

Leucocytosis is marked, the chief increase being in the *lymphocytes*.

Describe the Symptoms of a Typical Case.

After an *incubation period* of eleven to twelve days, the onset is abrupt. Rigors, frontal headache, violent backache,

vomiting, and sharp fever are its chief characters. In children convulsions may replace the rigors. The temperature rises until the third day, when the rash appears in its papular form. This is followed by a decline of the fever, but the temperature remains above normal till about the sixth day, when it begins to rise again, and attains a second maximum on the eighth day, coincidently with the maturation of the pustules. From this point it assumes a septic type, with considerable oscillations, and gradually declines until, about a week or ten days later, the scabs fall off, and convalescence is established. The two febrile periods are known as primary and secondary fever.

In bad cases the secondary stage is marked by rigors, great swelling of the face, and the development of the typhoid state.

A characteristic greasy *odour* is given off from a case of small-pox. (*See* Chart, p. 93.)

What are the Characters of the Rash?

It appears on the third day, first on the forehead, face, and scalp, in the form of elevated red papules. These can be felt even before they are visible, and impart a *shotty* feeling to the skin. It next appears on the back of the wrists, then on the trunk and arms, and finally on the legs. The mucous membranes suffer, and papules are visible in the mouth.

On the sixth day the papules become vesicular. The vesicles are loculated, depressed in the centre (*umbilicated*), and filled with a fluid at first clear, afterwards turbid.

On the eight or ninth day the vesicles become pustular, and are surrounded by a red inflammatory ring which causes much swelling, especially about the face. About the eleventh day the pustules begin to dry in, and the scabs fall off about the eighteenth or twentieth day, leaving reddish cicatrices which ultimately result in depressed pits.

Do any other Rashes occur in Small-pox?

Yes; there may be *prodromal* rashes, appearing about the second day. These are:—

1. *Erythematous*, resembling measles or scarlet fever.

2. A *hæmorrhagic* or *petechial* rash on the lower half of the abdomen, forming a triangle with its apex downwards.

3. *Petechio-erythematous*, occupying the abdomen and the sides of the chest.

The erythematous rash indicates a slight attack; with the others the outlook is more grave.

What are the Varieties of Small-pox?

1. Discrete: the pocks are few and separate, and the attack is mild.

2. Confluent: on the face, hands, and feet the pocks run together. The symptoms are severe; the face and hands are swollen, the temperature falls only slightly when the rash comes out, there is noisy delirium, and death often occurs from the ninth to the eleventh day.

3. Hæmorrhagic: in which either (1) the eruption may be preceded by a hæmorrhagic prodromal rash, and hæmorrhages from mucous membranes also occur (*purpura variolosa* or malignant small-pox); or (2) hæmorrhage may take place *into the pocks*.

4. Corymbose: in which the pocks are arranged in grape-like clusters.

5. Varioloid, or modified small-pox, the result either of vaccination or of a previous attack. The symptoms are slight and there is no secondary fever.

What are its Chief Complications?

Conjunctivitis, keratitis, and ulceration of the cornea; respiratory affections such as laryngitis, bronchitis, and bronchopneumonia; hæmorrhage; gangrene, abscesses, and erysipelas; orchitis, and purulent affections of the joints.

Small-pox may be *followed* by blindness, inflammation of the middle ear, recurrent boils, or permanent alopecia.

What are the Prognostic Indications?

1. The character of the rash: discrete small-pox is seldom fatal unless through complications, while confluent and hæmorrhagic cases are always severe, and often fatal. Prodromal rashes, except the erythematous type, mean a severe case. Varioloid is a mild disease.

2. The age and habits of the patient; small-pox is very fatal at both extremes of life, and in alcoholics. Pregnant women usually abort.

3. The presence of complications increases the gravity of the prognosis.

How would you treat a Case of Small-pox?

The hair and beard must be cut close; the skin should be sponged daily with tepid water, and anointed with carbolised vaseline to relieve itching; the eyelids should be covered with cold compresses and the conjunctivæ frequently irrigated. The room should be somewhat darkened, or red curtains may be used in the windows. A ten per cent. solution of iodine in glycerine is said to be useful in preventing pitting; other applications have little effect. Stimulants may be needed in the graver cases; and in convalescence, iron and quinine.

A suspected case must be isolated from the outset, and all "contacts" must be vaccinated *at once*.

VACCINATION.

What is Vaccinia?

An eruptive disease occurring in the udder and teats of the cow, and causing pustulation. It is also known as cow-

pox. It is considered to be small-pox modified by its occurrence in the cow. Human subjects inoculated with the lymph taken from the pustules develope immunity to small-pox.

How is Vaccination performed?

It may be done either with lymph taken from a vaccinated person (humanised lymph), or by direct inoculation from the calf. The second is the usual method; it is more apt to cause irritation of the arm, but avoids the risk of transmitting human diseases. Glycerinated calf lymph is the preparation generally employed.

What are the Effects of Vaccination?

On the second or third day a papule forms at the site of inoculation (usually on the upper arm), and this becomes a vesicle on the fifth or sixth day, and a pustule on the eighth or ninth. The pustule is umbilicated and surrounded by an inflammatory areola. It dries in, and the scab falls off about the end of the third week, leaving a permanent pitted scar. The glands in the arm-pit may be enlarged, the arm is swollen at the seat of inoculation, and there may be some fever.

At what Age must Children be vaccinated?

Before the age of six months, unless there is a "conscientious objection."

In what Circumstances should Vaccination be postponed?

If the child is feverish, if it suffers from a specific infectious disease, from skin disease, or from diarrhœa. If it has been exposed to small-pox it must be vaccinated at once, unless there is serious acute disease.

Is the Protection lifelong?

No; it tends to become less in course of time. Revaccination should be done about the age of seven or eight, and adults should be revaccinated when there is an epidemic of small-pox. Much depends upon the efficiency of the vaccination.

VARICELLA or CHICKEN-POX.

What is Chicken-pox?

An acute infectious disease characterised by a vesicular eruption which occurs in successive crops and begins on the first day.

What is its Etiology?

The specific organism is not known. The disease, which is usually trivial, is most common in childhood, and is contagious. Epidemics are very frequent.

What are the Symptoms?

There is usually slight fever with a furred tongue and malaise. The rash is often noticed before other symptoms. It begins on the neck and chest, and spreads thence to the rest of the body. It appears on the first day in the shape of pink, slightly raised papules, which become vesicular within twenty-four hours. The vesicles are rounded, *not umbilicated, and not loculated.* In a day or two their contents, at first clear, become turbid, but they rupture about the fifth day and dry up without suppurating. The scab falls off in about ten days, leaving little or no scarring. The eruption appears in successive crops, and this process continues for about a week. There may be modified spots upon the buccal mucous membrane.

The average *incubation period* is fourteen days.

How do you distinguish Varicella from Varioloid?

In chicken-pox the eruption appears on the first day, and occurs in successive crops; in modified small-pox it appears on the third day, in one crop.

In chicken-pox it begins on the chest and neck; in varioloid on the face and wrists.

In chicken-pox fever is slight, but does not fall as the rash appears; in varioloid it is more marked, but declines when the rash comes out.

In chicken-pox the vesicles are not umbilicated or loculated, and they are not surrounded by an inflammatory areola. They do not leave scars or pits unless they have been ruptured by scratching.

What is the Treatment?

That of mild fever. Itching should be soothed by a 1 in 20 or 1 in 40 carbolic lotion, and scratching, especially about the face, should be prevented, if need be by cotton gloves.

SCARLATINA or SCARLET FEVER.

Define the Disease.

An acute infectious disease characterised by fever, sore throat, and an erythematous rash, ending in desquamation, and often complicated by otitis or nephritis.

Mention the Etiological Factors.

The disease is most common in children, but attacks all ages. It occurs in frequent epidemics, oftenest in the second half of the year. It is spread through the air, by direct contact, and also by clothing, which may remain infective for long periods. Third persons may carry it; and it is often transmitted by milk.

Various organisms have been described, but none is admittedly specific.

What is the Morbid Anatomy?

That of fever, and that of the complications. The inflammatory affection of the throat is accompanied by adenitis which may go on to suppuration; endocarditis and pericarditis are common, and also nephritis.

Describe the Symptoms of a Typical Case.

After an *incubation period* varying from one to five days, but usually two to three, the disease sets in suddenly with headache, sore throat, vomiting, rheumatic pains, and fever. The fever also sets in suddenly at the same time as the sore throat. In children convulsions may occur. The pulse is very rapid. The sore throat affects the tonsils and pharynx, and is accompanied by glandular swelling and stiffness of the neck. The rash comes out on the *second* day, at first on the chest, whence it spreads over the face, body, and limbs, sparing the skin round the mouth. The temperature does not fall as the rash appears, but begins to decline about the third day, and is normal by the end of the week. (*See* Chart, p. 94.)

The sore throat may increase in severity, the tonsils being much enlarged, and covered with yellowish points which may coalesce so as to form a membranous patch. In ordinary cases it improves as the rash fades, and the sore throat, rash, and fever may have all vanished a week or so from the onset.

The tongue is at first white and thickly coated, with red papillæ projecting through the white fur (white strawberry tongue); afterwards the fur disappears, leaving the red papillæ standing out upon a red ground (red strawberry tongue).

The rash varies in severity. In some cases it may be absent, in others present only in the flexures of the limbs.

It consists of two elements—small red *punctate spots*, surrounded by a diffuse *erythema* which disappears on pressure.

Albuminuria is common, even in the absence of nephritis. There is usually a marked polymorphonuclear leucocytosis.

Desquamation begins towards the end of the first week, usually in the form of fine branny scales. It may not be complete for six weeks or more, and lasts longest on the palms and soles, where the skin may peel in large flakes.

What are the Varieties of Scarlet Fever?

1. *Latent* scarlatina; throat affection, rash, and fever are so slight as to pass unnoticed. The disease may be diagnosed at a later stage, by desquamation or nephritis.

2. The benign form, or *scarlatina simplex*, described above.

3. *Scarlatina anginosa*, or septic scarlet fever. The throat affection is severe, a membranous exudate covers the tonsils and palate, and the tissues of the throat may slough. The glands at the angle of the jaw may suppurate. The local lesion is dangerous in itself, and also through septic absorption.

4. *Scarlatina maligna*, or toxic scarlet fever. The symptoms are grave from the outset, and the rash is abundant, but the throat affection is often slight. Death takes place within a week.

What are the most Frequent Complications?

1. Otitis media, due to extension from the throat along the Eustachian tube. It may cause perforation of the drum, and sometimes, at a later stage, permanent deafness, facial paralysis, meningitis, or cerebral abscess.

2. Albuminuria.

3. Acute nephritis, most common during the stage of

desquamation, about the third week. It is accompanied by œdema or anasarca; the urine is high-coloured, scanty, albuminous, and contains blood and casts.

4. Scarlatinal arthritis, taking the forms of (*a*) scarlatinal rheumatism, which may be followed by cardiac mischief; (*b*) suppuration of the larger joints; (*c*) generalised pyæmic infection.

5. Endocarditis and pericarditis.

6. Bronchopneumonia.

Mention any Special Points in Treatment.

1. Isolation. Of late the plan of anointing the whole body with eucalyptus oil at first twice, and after a week once, daily from the outset of the disease, has been extensively used, and it is claimed that patients so treated may be liberated after ten days, though they are still desquamating freely. The results are very doubtful, and it is much safer to insist on isolation for at least four to six weeks, and longer if there is any discharge from the ear or nose, which must have ceased before the patient is set free.

2. The skin must be daily attended to by tepid sponging, and later by tepid baths. Inunction of eucalyptus should also be practised as above.

3. Examine the urine daily for signs of nephritis; and watch the condition of the ears.

4. To prevent nephritis, as little nitrogenous food as possible should be given, excepting milk.

5. Specific treatment. Injections of the serum of convalescents (dose 10 to 20 c.c.) have been employed, also polyvalent antistreptococcic sera, and lately polyvalent streptococcus vaccines, the various strains being obtained from the throats of scarlatinal patients.

6. The throat may be treated by a chlorine gargle or antiseptic sprays, and hot fomentations externally. Avoid

caustic applications. Brawny swelling of the neck may require incision.

7. Nephritis requires milk diet, hydragogue purgatives, and hot baths or packs. Uræmic convulsions; croton oil, pilocarpine, venesection.

MORBILLI OR MEASLES.

What is Measles?

An acute infectious disease attended by catarrh of the upper respiratory passages, and by a papular eruption.

What are its Causes?

The disease principally affects children, and is most common in spring and autumn. The organism has not been discovered, but infection is communicated by the breath and nasal secretions, and by fomites.

Measles is infectious both *before* and after the rash appears. Second attacks are rare.

What is the Morbid Anatomy?

Catarrhal inflammation of the nasal, bronchial, and intestinal mucosa, and of the conjunctiva; no other specific changes. In the eruptive stage, there is *leucopenia*, not leucocytosis.

Describe the Symptoms.

After an *incubation period* of from eight to fourteen days, the disease sets in with smart fever, coryza, watering and redness of the eyes, and photophobia. In infants there may be a convulsion. The catarrh extends to the bronchi, causing cough, and often to the intestinal tract, causing diarrhœa. The temperature falls to some extent

on the second or third days, but rises again with the appearance of the rash on the *fourth* day, remains high while the rash is out, and falls rapidly as it fades at the end of the week. The rash is succeeded by a fine branny desquamation, which lasts about a fortnight. (*See* Chart, p. 94.)

What are the Characters of the Rash?

It begins on the fourth day at the roots of the hair and on the forehead and face, and spreads thence over the body and limbs. It consists of small, flat blotchy papules raised above the skin, of a purplish red colour, and tending to be grouped in crescentic patches. The eruption imparts a velvety feeling to the skin. Its colour fades on pressure. It remains prominent for about three days, then quickly fades, leaving transitory brownish stains which are succeeded by a slight branny desquamation, most common on the face.

Are there any other Diagnostic Indications?

Yes: *Koplik's spots.* These are small rounded red spots with a whitish or bluish centre, which appear on the buccal mucous membrane one to three days before the rash. They are commonest at the angles of the mouth and opposite the molar teeth. They must be looked for in a good light, but are present in about 90 per cent. of all cases.

What are the Varieties of Measles?

1. Simple, as above described.

2. Hæmorrhagic. Bleeding may occur into the spots, and also from mucous surfaces, or from the kidney. This form is rare.

3. Adynamic. The symptoms are grave from the outset, though there are no hæmorrhages, and the typhoid state appears early.

Mention the Chief Complications.

1. Pulmonary affections (bronchopneumonia and collapse of the lung) due to extension of the bronchial catarrh.
2. Ophthalmia, otitis media, parotitis.
3. Gangrene of skin or vulva : cancrum oris.

Whooping-cough may follow measles; so also may tuberculosis. Chronic endocarditis is an occasional consequence.

Indicate the Main Lines of Treatment.

Isolation and careful nursing are necessary, and avoidance of any exposure to chill while the catarrhal period lasts. Tonics are needed in convalescence (Syr. Fer. Iodid., Syr. Hypophosph., Ol. Morrhuæ). If all catarrhal symptoms have gone, cases may be released at the end of three weeks.

RUBELLA, RÖTHELN, or GERMAN MEASLES.

Give a Definition of German Measles.

An acute infectious disease resembling both measles and scarlatina, but without marked catarrhal symptoms.

What are its Symptoms ?

After an incubation period which may be as long as twenty-one days, but *averages* about fourteen, slight catarrhal symptoms (coryza, sore throat) may appear, along with slight fever. This prodromal stage may be absent, and in any case does not last more than half a day. The cervical and occipital glands are at the same time swollen and tender. The rash, which appears on the *first* or *second* day, may be the first symptom noticed. It consists of round or oval pinkish spots, slightly raised, and usually discrete, though they may be closely set. It

appears first on the face and rapidly spreads to the body and limbs; it lasts from two to four days; and it may be followed by slight desquamation. Fever is often entirely absent, and the whole illness is over within a week.

How do you distinguish Rubella from Measles?

By the absence or slightness of the prodromal stage; the appearance of the rash on the first or second day; the absence of the crescentic grouping of the papules, and their lighter colour; and the absence of Koplik's spots.

How do you distinguish it from Scarlatina?

By the larger size of the spots; the slightness of the fever; and the absence of marked sore throat. In scarlatina the rash on the face spares the region round the mouth, which is pale in contrast to the surrounding skin; in rubella this region is not spared.

What is the Treatment?

No special treatment is needed; the patient is free from infection ten days after the appearance of the rash.

DIFFERENTIAL DIAGNOSIS OF THE EXANTHEMATA.

Disease	Length of Incubation Period in Days	Rash			Temperature	Special Symptoms
		Day of Appearance	Situation where first seen	Characters		
Rötheln	14 or more	1st or 2nd	Face	Round or oval pink papules	99°-100° F.; fever may be absent	Cervical and occipital glands swollen
Scarlatina	1-5	2nd	Chest and neck	Punctiform red spots in a diffuse erythema	103°-105° F.	Strawberry tongue; sore throat; frequent pulse; vomiting
Measles	8-14	4th	Forehead and face	Dusky-red papules; crescentic grouping	101°-104° F.; rises as rash comes out	Coryza and photophobia
Varicella	14	1st	Chest	Papules succeeded by vesicles; not umbilicated; successive crops	100°-102° F.	Constitutional disturbance slight
Variola	11-12	3rd	Forehead, face, back of wrists	Shotty papules, becoming vesicles, then pustules; umbilicated; one crop	104°-105° F.; falls as rash comes out; secondary fever	Pain in back; marked constitutional disturbance
Typhus	12-14	5th	Trunk	Dusky-red mottling, and papules which become petechial; not affected by pressure; one crop	103°-105° F.	Headache; marked nervous symptoms, and prostration
Enteric	10-14 or more	6th-10th	Abdomen and back	Scanty rose spots, disappearing on pressure; successive crops	103°-104° F.; characteristic method of rise	Diarrhœa with characteristic stools; enlargement of spleen

MUMPS or EPIDEMIC PAROTITIS.

What is Mumps?

An acute infectious disease, characterised by an interstitial inflammation of the parotid glands.

What is its Etiology?

The organism is unknown. The disease attacks children and young adults, and is often epidemic, attacking an entire school.

Describe the Symptoms.

The incubation period varies from one to three weeks. Thereafter headache and pain about the angle of the jaw, with stiffness of the neck, set in, associated with fever, which may last three or four days. The parotid on one side becomes swollen, elastic, and tender, but not fluctuant, and the submaxillary and sublingual glands may also become swollen. After one or two days the other side is involved, and the swellings then form a collar which may be so large that the mouth can be only partially opened. Salivation is common, and the tongue is furred. The swelling resolves in about nine to twelve days, leaving the patient well. The glands do not suppurate.

What are the Complications?

Metastatic orchitis, occurring as the parotitis subsides. It is unilateral, and is most common in adults. It subsides in a few days, but may be followed by atrophy. In women mastitis, ovaritis, or œdema of the vulva may occur. Meningitis and endocarditis have been recorded.

Beyond a purge at the outset, fomentations, and an antiseptic mouth wash no special treatment is required.

PERTUSSIS or WHOOPING-COUGH.

What is Whooping-Cough?

A specific infectious disease, characterised by a paroxysmal cough ending in a long noisy inspiration.

What are its Causes?

The micro-organism is a bacillus recently discovered by Bordet & Gengou. It is a minute oval rod, Gram-negative, and growing freely on blood agar. It is abundant in the sputum during the first week.

Whooping-cough is highly contagious, and may be carried by fomites. The sputum is the infective agent. Children are chiefly attacked, but adults may suffer. It is most infectious in the first week, and the infectivity lessens as the case goes on. Five weeks from the onset the child may be considered non-infectious.

Describe the Symptoms.

The average incubation period is about eight days. The *stage of invasion* is characterised by feverishness, bronchial catarrh, and coryza, and lasts a week or more. Then follows the *paroxysmal or spasmodic stage*, in which a series of short coughs, without any intervening inspiration, is followed by a deep prolonged inspiratory effort with the characteristic crow or *whoop*. Three or four of these paroxysms succeed each other, and a plug of mucus is expelled, or vomiting takes place. In the intervals the child is perfectly well, but during the paroxysm the face becomes blue and the eyeballs protrude, and if the attacks are frequent there may be some œdema of the face. Nasal and subconjunctival hæmorrhages are common, and there may be ulceration of the frænum linguæ. Râles are audible during the paroxysms, but in

the intervals there may be no physical signs, unless complications are present. There is a marked leucocytosis, affecting chiefly the lymphocytes.

The duration of the paroxysmal stage is from three to six weeks. It is succeeded by a *period of decline*, in which the paroxysms become gradually less frequent, and finally disappear. Convalescence is slow, and during its progress patients are unusually liable to tuberculosis.

What are the Chief Complications?

Bronchitis and bronchopneumonia. Subcutaneous emphysema sometimes occurs, from rupture of the air-cells during the paroxysms. In young children convulsions; rarely cerebral hæmorrhage.

What is the Prognosis?

Favourable, save for the complications. Apart from these it depends on the number of the paroxysms. There may be sixty or more in the twenty-four hours, and such cases are grave; in most cases there are less than forty, and in very many less than thirty.

On what does the Diagnosis depend?

On the paroxysmal nature of the cough. Even if the whoop is not heard, a prolonged illness in a child, marked by violent cough *attended with vomiting*, is almost conclusive of whooping-cough. The discovery of the bacillus in the sputum, or the agglutination of cultures by the patient's serum, is conclusive evidence.

What are the Lines of Treatment?

The child must be isolated, and during the febrile stage must be in bed. The preliminary catarrh may be benefited by a mixture containing Tinct. Camph. Co.

There is no specific remedy; in the paroxysmal stage all the antispasmodics have been tried, and the best of them is belladonna, which children take well. Heroin, antipyrin, hydrocyanic acid, chloral, and bromide of potassium are also used. The room must be well ventilated, and may be impregnated with antiseptic vapours (creosote, carbolic acid, etc.). In convalescence tonics are needed.

INFLUENZA.

Define the Disease.

An acute specific infectious disease, with local symptoms mainly affecting the respiratory and nervous systems, and followed by prolonged prostration.

What is the Etiology?

The *bacillus influenzæ* is a minute rod, reacting negatively to Gram's stain, and forming very small colonies on blood agar. It is found in the secretions of the respiratory tract, and the disease is spread by the nasal discharges or the expectoration. Epidemics are commonest in the winter months.

What are the Symptoms?

The usual incubation period is two to four days. The onset is sudden, with headache and backache, pains in the bones, prostration, and smart fever (102°-104° F.). There may be coryza or sore throat. In mild cases the temperature falls in a day or two, and convalescence begins, but a sense of prostration remains for some time; in others defervescence is protracted, and may extend over more than a week (febrile type). The spleen may be slightly enlarged.

Other types of the disease are :—

I. Respiratory : with dyspnœa, pain in the chest, cough, and signs of bronchitis, bronchopneumonia, or less commonly lobar pneumonia.

II. Gastro-intestinal or abdominal : with abdominal pain, diarrhœa or vomiting, and occasionally jaundice.

III. Nervous : with severe neuralgia, drowsiness or insomnia, and enduring prostration. Cardiac irregularity and anginoid pain may follow, and in bad cases there may be coma or delirium.

What are the Complications ?

Almost any inflammatory affection may complicate influenza, but in addition to the respiratory inflammations, neuritis, malignant endocarditis, meningitis, and otitis media may be specially mentioned. In elderly patients the disease is a grave one.

How is Influenza to be treated ?

Even a mild case must be at once sent to bed, and kept there for a day or two after defervescence. The sputum and nasal discharges must be disinfected. A saline purge may be given at the outset, and the pains should be treated by salicylate of soda (gr xv every four hours), aspirin (gr x), or phenacetin. Stimulants may be required for the elderly. Much care must be taken in convalescence, and at this stage tonics containing strychnine are valuable.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

What is this Disease ?

An acute infection due to a specific micro-organism, and characterised by inflammation of the cerebro-spinal meninges.

What is the Causal Factor?

The organism is the *diploccus intracellularis meningitidis* or *meningococcus*, a Gram-negative organism growing on serum-agar, and found chiefly within the polynuclear cells of the inflammatory exudate. Young children are chiefly affected, but young adults may also suffer. The organism is found in the naso-pharyngeal secretions, and may be carried by convalescents or by contacts for long periods of time.

Describe the Morbid Changes.

The meninges of both brain and cord may be merely intensely congested when death has been rapid, but in most cases there is an abundant fibrino-purulent exudate on the convexity, at the base, and in the lumbar cord. The membranes are thickened, and the ventricles contain turbid fluid. In long-standing cases there may be hydrocephalus.

What are the Symptoms?

The onset is sudden, with intense occipital headache and pain down the neck and spine, vomiting, and sometimes a rigor. The neck is stiff, and *the head strongly retracted*, while the limbs and trunk are rigid, and the body may be arched backwards (*opisthotonos*). Vertigo may be prominent; photophobia is marked; and there may be paralysis of ocular muscles. The pupils vary, and may be unequal; optic neuritis may occur. *Kernig's sign* (when the thigh is flexed perpendicularly to the abdomen, the leg cannot be fully extended at the knee) is present. The spleen is enlarged, and there is a high leucocytosis (25,000 to 40,000 per cmm.). Fever is variable; in some cases the temperature rises to 104° F. or more, in others there may be little rise. Delirium may be present from the start, or may appear later; in fatal cases it is followed

by coma. Convulsions are common in children. Convalescence, when it occurs, is slow, and the child may be left blind, deaf, or hydrocephalic.

What are the Rashes of Cerebro-spinal Meningitis?

Herpes of the face, or herpes zoster. In some cases there is a specific petechial rash, usually beginning on the legs ("spotted fever").

What are the Diagnostic Features?

The sudden onset, the high leucocytosis, the presence of Kernig's sign (which may occur in other forms of meningitis), the skin eruptions, and the results of lumbar puncture. The cerebro-spinal fluid so withdrawn is turbid, the leucocytes are polymorphonuclear, and the meningococcus can be found in them.

How would you treat such a Case?

By ice to the head and back of the neck, and opium or morphia if necessary for the pains. Cold sponging may aid to reduce the fever. Repeated removal of cerebro-spinal fluid by lumbar puncture will relieve the pressure and mitigate the symptoms. The specific treatment is the use of an antiserum, preferably Flexner's; 50 cc. of cerebro-spinal fluid being withdrawn, 30 cc. of the serum are injected in its place, and this may be repeated daily or every second day.

DIPHTHERIA.

What is Diphtheria?

A specific infectious disease, with local lesions, characterised by the presence of a membranous exudation in the upper respiratory passages, and often followed by transitory paralyses.

Describe the Specific Micro-organism.

The *bacillus diphtheriæ* is a slender rod presenting both long and short forms. It is clubbed at one end and often slightly curved, and when stained with Loeffler's methylene blue appears granular or beaded. It is not decolorised by Gram's method. It is found in the false membrane, where it is often associated with pyogenic organisms; but it rarely penetrates the mucosa.

What are the Causes of Infection?

The organism is disseminated in the secretions of the mouth and throat, in the clothing of the sick, and by "carriers," who may be either convalescents or healthy people in whose throats it has found a lodgment without causing disease. Milk is readily infected, and epidemics are often traceable to a contaminated milk-supply, and also to school infection. The disease is most common in children, but may occur at any age. Measles and scarlet fever predispose to it. Epidemics are most frequent in autumn.

Describe the Morbid Changes.

The false membrane consists of a network of fibrin, entangling leucocytes, the specific bacilli, and pyogenic organisms. The subjacent mucous membrane is inflamed, necrotic, and in the fauces and nares the false membrane adheres to it. In the larynx the necrosis is superficial, and the membrane can be stripped off.

The general changes are produced by the diphtherial toxins, not by the bacilli, which remain local. They include fatty degeneration of the heart, cloudy swelling of the kidney, or nephritis, and at a later stage peripheral neuritis. The cervical lymphatic glands are usually swollen, and the lungs are very liable to broncho-

pneumonia. The conjunctiva, or abraded surfaces of skin, may be infected, with formation of false membrane.

What are the Symptoms ?

The brief incubation period—two to seven days—is succeeded by symptoms of malaise with slight fever, headache, and a rapid, feeble pulse. Sore throat is complained of, the glands at the angle of the jaw become enlarged and tender, and the fauces and soft palate are deeply congested. Soon afterwards one or more whitish patches appear on the tonsils or uvula. The patches coalesce, and become wash-leather-like in colour, forming a membrane which may extend all over the fauces and uvula, and is firmly adherent, leaving a bleeding surface when stripped off. The temperature is seldom above 103° F., and more usually is about 101°. Occasionally it is subnormal. Prostration and anæmia are marked. Albuminuria, febrile or due to nephritis, is frequent, and appears early.

What is the further Course of the Disease ?

In mild cases there may be no further extension, and a gradual convalescence follows ; in others death may be due to cardiac failure. The disease may also originate in the nose or larynx, or may extend to these parts.

Nasal diphtheria is attended by a thin mucoid discharge, epistaxis, and a nasal twang of voice. The passages are obstructed, and the patient breathes through the mouth.

Laryngeal diphtheria is accompanied by stridor, brassy cough, dyspnœa, and sucking-in of the lower intercostal spaces on inspiration. The membrane may extend to the bronchi. Death is due to asphyxia or cardiac failure.

What are the Complications ?

Bronchitis and bronchopneumonia ; cardiac failure ;

nephritis; suppuration of the cervical glands; and septic conditions due to mixed infection (the pyogenic cocci).

What is the Nature of Post-diphtheritic Paralysis?

It is rather a sequela than a complication, and, like the cardiac failure, is due to the diphtheritic toxins. When these affect the peripheral nerves, they set up a neuritis, which comes on a fortnight or three weeks after apparent recovery. The soft palate is first affected, becoming anæsthetic and paralysed, so that fluids regurgitate through the nose, and the voice is nasal. There may also be strabismus, paralysis of accommodation or ptosis, and weakness of the lower limbs. The knee-jerk is early lost. Most cases recover within a few months, but the neuritis may extend to the phrenic or vagus, causing death through diaphragmatic or cardiac paralysis.

Diagnosis, in the early stages, rests upon the bacteriological examination of swabs rubbed over the affected part.

How is a Case of Diphtheria to be treated?

1. By injections of antitoxic serum, given as follows. Mild cases, 4,000-8,000 units, one dose; moderate cases, 12,000-16,000 units, one or two doses; severe cases, 20,000-50,000 units, two or three doses (M'Combie). The dose depends on the severity of the case, not on age. In favourable cases the temperature falls, and the membrane begins to separate within twenty-four hours.

2. Local treatment consists in swabbing or spraying with antiseptic solutions (carbolic acid 1 in 40, formalin 1 in 200, or Loeffler's solution, containing menthol 10 grammes dissolved in toluol to 36 cc., Liq. Ferri Sesquichlor 4 cc., absolute alcohol, 60 cc.) In nasal diphtheria these solutions may be used as a nasal douche; in laryngeal diphtheria hot sponges to the larynx and medicated inhalations may relieve symptoms,

but everything must be in readiness for intubation or tracheotomy.

3. General treatment must be stimulating. The diet is to be fluid but nutritious; tonics such as quinine or perchloride of iron are indicated; and stimulation is called for in the later stages. Cardiac failure may be met by digitalis; and for the paralysis strychnine and electricity are needed, and in some cases feeding by the nasal tube.

4. Isolation must be maintained for at least three weeks, or until organisms can no longer be cultivated from the throat or nose. Contacts should be kept in quarantine for twelve days.

ERYSIPELAS.

What is Erysipelas?

A specific infection characterised by a spreading inflammation of the skin, and due to the *streptococcus erysipelatis*.

What are its Causes?

The organism is morphologically indistinguishable from the streptococcus pyogenes, but nevertheless invariably produces erysipelas. It is found in the lymphatics at the advancing edge of the eruption. The disease is very contagious, and is most common in the elderly, and in those suffering from chronic Bright's disease, alcoholism, or malnutrition. The existence of a wound or abraded surface (*e.g.* recent delivery) favours its development, but the abrasion may be so slight as to pass unperceived.

Describe the Morbid Changes.

The affected skin is inflamed, swollen, and œdematous, and the infection spreads by the lymphatics to the adjacent lymphatic glands. There may be visceral complications due to general pyæmia, and these take the form of

infarctions of lung, spleen, or kidney, or of inflammations of serous membranes or malignant endocarditis.

Describe a Typical Attack.

After an incubation period of two to seven days, the onset is sudden, generally accompanied by rigor, sometimes by vomiting, and the temperature rises sharply, reaching 104° F. or more on the second or third day. A red patch with sharply defined outlines appears on the skin, either where there is a wound, or upon the face at the inner canthus, the angle of the mouth, etc. It spreads rapidly the edges being raised, hard, and red, the centre paler and the surface œdematous. Vesicles or blebs appear upon the affected area, which extends until the whole face and often also the scalp, may be involved. The eyelids are swollen, and their swelling may close the eyes. The inflammation involves the neighbouring lymphatic glands, and may spread to the mouth, pharynx, or larynx. In most cases the temperature falls suddenly about the sixth day, but it may remain high, or rise again with the involvement of a new area. The general symptoms are those of fever, and death may be due to exhaustion or to complications.

What are the Chief Complications?

Septicæmia, œdema of the larynx, pneumonia, and ulcerative endocarditis, rarely meningitis, besides the conditions already mentioned. Alcoholism and advanced years influence the prognosis unfavourably.

What is the Treatment?

Isolation, confinement to bed, nourishing fluid food, and stimulants when necessary. Antistreptococcic serum (20 cc.) may be injected once daily, and Tr. Fer. Perchlor. given in large doses (℥ss-℥i every four hours). Locally protect the parts from air, and relieve pain by lead lotion or dusting powders (Zinci Oxidi, Pulv. Amyli, aa ℥i).

SEPTICÆMIA, SAPRÆMIA, and PYÆMIA.

To what Causes are these Conditions due?

To the introduction *into the circulation* of living micro-organisms or their toxins.

What are the Organisms chiefly concerned?

Usually the pyogenic organisms, streptococci and staphylococci; but some others (*e.g.* pneumococcus, gonococcus) may also set up a generalised sepsis.

How do the Three Conditions differ?

Septicæmia is due to the circulation in the blood of living organisms, which form toxins in the blood, and themselves multiply there. The depth of the intoxication, therefore, depends upon the capacity of the organism to propagate itself, and not upon the dose of toxin originally introduced.

Sapræmia is due to the circulation in the blood of toxins manufactured by organisms which have a *local* seat, and do not themselves enter the blood stream. The intoxication is proportionate to the quantity of toxin introduced.

Pyæmia is a condition in which fragments of septic matter containing micro-organisms are carried from a primary septic focus through the circulation, until they block the smaller vessels of the viscera or skin. These septic emboli cause the formation of local abscesses—metastatic abscesses.

How can they be distinguished clinically?

Septicæmia may arise from a gonococcus or pneumococcus infection, from an open wound, or from uterine infection. In some cases the local lesion is indiscoverable. The disease begins abruptly, with one or more rigors, the temperature rises rapidly, prostration is great, jaundice or diarrhœa may be present, and the appearance of the typhoid state heralds death, usually within a week or ten

days of the onset. The organism may be cultivable from the blood.

Sapræmia is always due to a local lesion, very frequently to post-partum uterine infection. Its symptoms are similar to those of septicæmia, though they may be less severe, but the organism is never to be found in the blood, and the intoxication ceases when its local cause is removed.

Pyæmia originates in connection with infected wounds, gonorrhœa, otitis media, ulcerative endocarditis, osteomyelitis, and intestinal or vesical ulceration. The streptococcus pyogenes is its most common cause. The onset is sudden, with repeated rigors and high fever of a remittent or intermittent type, and profuse sweating. Exhaustion and emaciation are rapid, vomiting and diarrhœa frequently appear, and there are local signs of visceral or subcutaneous abscess, or suppuration in the joints. Pleurisy or empyema, pericarditis, or bronchopneumonia may complicate the case; and jaundice may be present. Acute cases die within ten days, death being preceded by the typhoid state; in more chronic cases death may follow after weeks or months of exhausting suppuration, or there may be a slow recovery.

Leucocytosis may be present in all forms of sepsis.

What is the Treatment of these Conditions?

Any local focus of infection must be surgically treated. In both septicæmia and pyæmia nourishing fluid food and stimulants are needed, and quinine, gr. v-x every four hours, is of some use. In septicæmia, if the organism can be found, an autogenous vaccine may be tried; if not, a polyvalent antistreptococcic serum (20-30 cc. once daily). In pyæmia the treatment is similar, and abscesses must also be surgically dealt with.

RHEUMATIC FEVER (ACUTE RHEUMATISM).

What is Rheumatic Fever?

A specific infectious disease, characterised by polyarthritis, and a tendency to inflammatory affections of the pericardium and endocardium.

What are its Causes?

The specific organism has not been certainly identified, though the *micrococcus rheumaticus*, a short Gram-positive streptococcus found in the inflamed synovial membranes and in the cardiac lesions, is claimed by Poynton and Paine as the cause of the disease. Rheumatic fever is commonest in spring, and in damp climates; it attacks both sexes, and by preference young adults; it may be provoked by exposure to cold or damp; and one attack predisposes to a second.

What are the Morbid Changes?

Inflammatory affection of the synovial membranes and ligaments, the effused fluid being turbid and containing albumin, but never becoming purulent; in many cases pericardial, endocardial, or myocarditic changes of the ordinary inflammatory type, but with a special tendency to affect the valvular endocardium; and in some inflammation of other serous membranes, such as the pleura.

Describe the Chief Symptoms.

The attack, which is often preceded by tonsillitis, may begin either abruptly or after a few days' malaise. One of the larger joints is usually first affected (knee, ankle, or wrist), and becomes swollen, red, hot, and intensely painful. Fluid effusion can be recognised by palpation. The inflammation spreads to other joints, or flits from one to another, the first affected improving as the second is attacked. The smaller joints may be implicated later.

Fever accompanies the inflammation, but is not usually high (101°-103° F.), and though irregular in its course, generally disappears within ten days. In some cases *hyperpyrexia* forms a dangerous complication. There is free sweating, with a characteristic sourish smell. Anæmia is marked and develops rapidly. The tongue is heavily coated with a thick moist fur ("blanket" tongue). *Murmurs*, organic or due to anæmia, may at any time appear over the heart. Relapses are very common.

What are the Complications?

1. Implication of the heart is so common that the organ should be examined daily. Endocarditis, pericarditis, and myocarditis may all be present, or the endocardium or pericardium may chiefly suffer. To some slight extent the myocardium is probably always affected along with the membranes, and it may be profoundly damaged.
2. Pleurisy is common, and is often associated with pericarditis.
3. Cutaneous eruptions:—sudamina, purpura, erythema, and subcutaneous fibrous nodules in the neighbourhood of the joints.
4. Hyperpyrexia, accompanied by delirium or coma, a rare but very grave complication.
5. Chorea.
6. Tonsillitis.

Note that in children the joint affection and fever are slight, and the heart may be attacked with little to indicate its involvement. "Growing pains" should always call for attention.

How would you treat such a Case?

By absolute confinement to bed, the patient wearing a flannel gown, lying between blankets, and being kept on a fluid diet, consisting chiefly of milk. Salicylate of soda, salicin, and aspirin (acetyl-salicylic acid) are the drugs

principally used. In severe cases the salicylate should be given in doses of gr. xx. every two hours till pain is relieved, and afterwards less often and in smaller doses till it is entirely withdrawn. It may be combined with alkalies (Sodii Bicarb.) to avert the risk of salicylism, of which the symptoms are tinnitus, deafness, delirium, vomiting, and acetonuria. Should the salicylates fail, large doses of sodium bicarbonate should be used instead.

Locally, the joints should be wrapped in cotton wool, or if very painful, fomented and fixed on a splint. The compound methyl salicylate liniment is often useful, and morphia may be needed at first.

Hyperpyrexia is to be treated by cold baths or the cold pack.

GONORRHŒAL ARTHRITIS.

What are the possible After-effects of a Gonorrhœal Urethritis?

The infection may spread locally, involving other parts of the genito-urinary tract (cystitis, epididymitis, salpingitis, etc.); or a systemic infection may follow. This may cause a *gonorrhœal septicæmia or pyæmia*, recognisable by the history, the presence of urethral discharge, or the recovery of the organism from the blood; or, more commonly, a *gonorrhœal arthritis*, which may closely resemble the arthritis of acute rheumatism.

At what Stage is a Gonorrhœal Arthritis likely to appear?

Usually from a fortnight to a month after the gonorrhœa; sometimes while the urethral discharge is still acute, at other times in the stage of gleet, or even after discharge has ceased.

What are its Characteristics?

Both the joint itself and the periarticular tissues are affected. The effusion is usually serous, but may become

purulent; the cartilages may be eroded, and ankylosis is a not uncommon sequel. Only one joint may suffer, but generally several are involved, including both the knees, wrists, and ankles, and also joints *which do not usually suffer in acute rheumatism* (temporo-maxillary, sterno-clavicular, intervertebral, etc.). The joints are swollen, red, painful, and tender on movement or pressure; the fasciæ, especially the plantar, are apt to be involved at the same time; and there may be ocular complications (iritis, conjunctivitis). Fever is not usually high, *but does not yield to salicylates;* and when the acute symptoms have passed, obstinate stiffness or fibrous ankylosis may remain.

How is the Condition to be distinguished from Rheumatic Fever?

By the history or presence of urethral discharge; the implication of joints not often attacked by rheumatism; the absence of response to anti-rheumatic treatment; and the recovery of the organism from the urethral discharge or synovial fluid. *Note* that in many cases of gonorrhœal arthritis cultures from the synovial fluid remain sterile.

What is the Treatment?

Treat the discharge: paint the joints with iodine or with equal parts of ext. belladonnæ and glycerine, and fix them. Drugs, except tonics, are probably useless, although iodide of potassium is often recommended. Vaccines, autogenous if possible, are often useful, in doses of four to ten million organisms every four to eight days, and antigonococcic serum (25 cc. daily) may be tried if they fail. Later, massage and passive movement for the stiffened limbs are required.

SYPHILIS.

What is Syphilis?

A specific infective disease transmissible by inoculation (acquired syphilis) or by inheritance (congenital syphilis), due to the *Spirochaete pallida*, and characterised by the slow evolution and long duration of its symptoms.

Describe the Spirochaete.

The *Spirochaete pallida* or *spironema pallidum* is a spirally-shaped filamentous organism from 4 μ to 16 μ long, and ·25 μ broad, each spiral containing from 6 to 14 turns. The organism, which is highly mobile, is to be found in all syphilitic lesions, although in the tertiary stage only with difficulty, and in secondary syphilis it may be present in the blood. It is believed to be a protozoon.

How is Syphilis transmitted?

By direct infection, usually during the sexual act, or by mediate infection, through infected cups, glasses, or instruments. The chancre and all secondary lesions, also the blood in the secondary stage, and the lesions of congenital syphilis, are infective. In the tertiary stage infection is not so likely, but is not impossible.

Hereditary transmission may occur either through the father or mother. If the mother is infected during pregnancy, the child may be infected through the placenta, but often escapes when the maternal infection is late. The mother of a syphilitic child, even if she show no signs of the disease, acquires immunity to it and cannot be subsequently infected (*Colles' law*).

What are the Morbid Changes?

Both in the chancre of the primary stage and in the cutaneous lesions of the secondary, there is an accumula-

tion of round cells in the corium with infiltration of the connective tissue; the vascular epithelium is swollen, the walls of the vessels thickened, and their lumen narrowed; and the neighbouring lymphatic glands are infiltrated and enlarged. The secondary cutaneous lesions may be macular, papular, or pustular. Lesions on mucous membranes are papules modified by their situation, and on the genitalia or at the anus such papules take the form of moist vegetations known as condylomata.

The tertiary lesion, or *gumma*, is a hard, nodular mass, consisting of round cells with occasional epithelioid and giant cells, and surrounded by firm connective tissue. It tends to become necrotic in the centre, and when it breaks down and suppurates, may discharge through the skin or mucous membrane. Gummata are found in the most varied situations.

In this stage also arterial degenerations are common :— atheroma, endarteritis, aneurysm.

What are the Stages of a Typical Case ?

(1) The stage of incubation; (2) the primary sore; (3) the secondary, and (4) the tertiary stage.

Describe the Characters of these Stages.

The average *Incubation Period* is from three to five weeks; it may be as little as a fortnight or may extend to six weeks or more.

The *Primary Sore*, or *hard chancre*, appears on the glans or prepuce, the labia or vulva, as a papule with a base almost cartilaginous in hardness, and extending from the centre to form a flat coin-shaped induration. The papule may or may not ulcerate. The nearest lymphatic glands (in the groins) also become enlarged and hard, and can be rolled under the skin. In process of time the chancre disappears, and is replaced by a cicatrix.

The *Secondary Stage* appears from one to three months, usually about six weeks, after the appearance of the chancre. It is ushered in by transient and generally slight fever and malaise, by a fleeting roseolar rash, mainly on the abdomen, and by a sore throat, which at first may show merely faucial congestion. Somewhat later, the roseola is followed by other skin eruptions (*syphilides*), which are symmetrical in distribution, coppery or like raw ham in colour, circular or polycyclic in outline, and often polymorphous, *i.e.* presenting at the same time, in different situations, macules, papules, pustules, or scales. Still later, circular ulcerations of the skin may occur, which have a sharply-cut edge. If they are neglected the dried discharges form thick, adherent, conical greenish crusts (*rupia*). Such rupioid eruptions also occur in the tertiary stage.

The mouth also suffers, the tonsils being symmetrically ulcerated, and white "mucous patches"—*i.e.* papules modified by their situation—form on the buccal mucous membrane. Condylomata—moist, flattened, warty growths—appear around the anus or on the scrotum or labia.

The patient becomes anæmic; his hair may fall out (alopecia) although not permanently or completely; he suffers from headaches, chiefly at night, and from bone-pains; and the lymphatic glands are enlarged. Iritis or periostitis may occur.

In the *Tertiary Stage*, which may set in a year or so after the secondary, but often not for many years, *gummata* are the most characteristic lesions. They may occur subcutaneously, in the bones or muscles, or in any viscus. In the brain they may cause hemiplegia or Jacksonian epilepsy, in the liver syphilitic cirrhosis. Superficial gummata tend to ulcerate and discharge, deep gummata to be slowly absorbed, leaving cicatricial deformity of the viscera. Tertiary eruptions are asymmetrical, often rupioid, and leave permanent scars. "Nodes" on the

long bones or cranial convexity are common. The profound arterial changes have already been referred to.

In women miscarriages and still-births are common.

What is the Quaternary Stage of Syphilis?

A group of chronic diseases, not in themselves specific, but closely associated with syphilis, and due to degenerative changes set up by the syphilitic toxin. Locomotor ataxia, general paralysis, and amyloid disease are the chief examples, and are spoken of as *parasyphilitic* or *metasyphilitic* diseases, or sometimes as the quaternary stage. The recent discovery of spirochaetes in the lesions of general paralysis, however, proves it to be definitely specific.

What are the Features of Congenital Syphilis?

The child may be infected either from the father or the mother; in paternal infection the fœtus immunises the mother, who cannot thereafter be infected. The disease is transmissible in the primary and secondary stages, rarely in the tertiary.

With a few exceptions, the child is born apparently healthy. Bad cases may show at birth a pemphigoid eruption (pemphigus syphiliticus), snuffles, and general atrophy. Such cases do not long survive.

Usually, symptoms begin from the fourth week to the third month. "Snuffles" due to rhinitis appears, the nasal bones may become necrosed and *the root of the nose depressed*, fissures, or *rhagades*, appear at the angles of the mouth, and mucous patches within it, while condylomata are often present at the anus. A coppery erythema appears on the buttocks and inner sides of the thighs, or it may occupy the palms and soles. Onychia is common. The *spleen is enlarged*, and often the liver also, and there may be epiphysitis.

These early lesions may be fatal, or may pass off.

Symptoms recur about puberty; the upper central incisors of the permanent teeth are notched and peg-shaped ("Hutchinson teeth"); interstitial keratitis, iritis, or deafness may occur; periostitis and "nodes" on the long bones are common, and there is sometimes synovitis of both knees; there may be visceral gummata; and the child is ill-nourished, with a prominent brow and depression of the bridge of the nose.

How can the Diagnosis be aided?

The diagnosis is often evident, but at times difficult. Careful search for old cicatrices, or for a history of chancre, roseola, miscarriages, etc., must then be made. If doubt still exists, the blood must be examined, and a positive Wassermann reaction is conclusive. In the primary stage, the presence of spirochætes determines the specificity of the sore.

What is the Treatment of Syphilis?

There are two methods: (1) by mercury and iodide of potassium, (2) by salvarsan.

1. The chancre may be dusted with calomel or iodoform. If there is any doubt as to its nature, specific treatment should not be begun till secondaries appear; if the diagnosis is positive, such treatment may be begun at once. Mercury can be given in many forms:—(*a*) as Hutchinson's pill (Hydrarg. c. Creta, Pulv. Ipecac. Co. aa gr. i. One or two pills thrice daily). This can be given for long periods without salivating, and the treatment must be continued for two or three years. (*b*) Inunction of Ung. Hydrarg. ʒi daily, changing the area of skin anointed from day to day. This is used in repeated courses of six weeks' duration, and is more rapidly effective. (*c*) Intramuscular injection of corrosive sublimate (gr. $\frac{1}{8}$ every second day, or gr. $\frac{1}{3}$ once a week), or of calomel gr. i-ii in sterile oil, also once a week, or of mercurial creams. During all such

courses the mouth must be kept rigorously clean, and smoking should be forbidden.

In the tertiary stage, iodide of potassium gives better results than mercury, but the two are often combined. The dose is gr.v-gr.xx thrice daily, well diluted, in ordinary cases; cerebral syphilis calls for larger doses.

The general health must be carefully attended to; anæmia must be treated by iron or arsenic; and tonic or climatic treatment is of value.

2. Salvarsan (dioxydiamidoarsenobenzol), the recent discovery of Ehrlich, is best given by intravenous injection. Under its use symptoms in many cases rapidly disappear, and the Wassermann reaction becomes negative, at all events temporarily. But it is often necessary to repeat the injection, sometimes more than once, and it is best used in conjunction with a mercurial course. The dose for intravenous injection is 0·3 to 0·5 gramme, according to the weight and strength of the patient. It must not be given if cardiac or renal disease or grave nervous affection is present. Its use is not entirely free from danger, and should be confined to those who have experience of the technique.

TUBERCULOSIS.

Define the Disease.

A specific infective disease due to the *bacillus tuberculosis*, and characterised by the formation of "tubercles," which may be generalised or may have a local seat.

What is the Etiology?

The *bacillus tuberculosis* is a short Gram-positive bacillus, 3 to 4 μ in length, and 0·5 μ broad. When stained it is not decolorised by acids or by alcohol. In old cultures it forms long filaments, which sometimes branch, and is thus allied to the streptothriceæ. The organism is widely

distributed, and though it may attack even the robust, its entrance is favoured by conditions of lowered vitality, such as are induced by life in towns, sedentary occupations, malnutrition, absence of sunlight, and the insufficient ventilation of apartments laden with infected dust. In childhood, measles and whooping cough are predisposing causes, in the adult, diabetes and alcoholism ; in childhood, the bones, joints, glands, peritoneum, or meninges are most liable to attack, in the adult, the lungs. The child is liable to infection from infected parents, but though a tuberculous predisposition may be inherited, there is no direct inheritance of the disease.

How is the Infection conveyed ?

1. By inoculation upon abraded or injured surfaces (lupus, *post mortem* wounds) ; a local tuberculosis results.

2. By inhalation of the dried and powdered sputum in the form of *dust* (phthisis, laryngeal tuberculosis).

3. By the alimentary tract (tonsils, intestine). Infection through the tonsils may reach the cervical or bronchial glands, and cause phthisis or general tuberculosis ; infection through the intestine, principally due to infected milk, may cause intestinal or peritoneal tuberculosis, or may attack the lungs through a more remote route.

What are the Morbid Changes induced by the Bacillus ?

The organism responds to the attack of the bacillus by the formation of tubercles. The *grey miliary tubercle* is a small semitransparent body which at a later stage becomes opaque and yellowish. It is of such a size as usually to be visible to the naked eye, though in some cases a lens may be necessary for its recognition. Such tubercles are formed wherever the bacillus has found a lodgment. On microscopic examination, they are seen to consist of a central zone of giant cells, each with many nuclei at the periphery, a zone of epithelioid cells enclosing these, and

externally a ring of lymphoid cells surrounded by a network of fibrous tissue. The bacilli are found in the central strata.

Containing no blood-vessels, the tubercles are liable to degenerative softening (caseation), and thus become "yellow tubercles." The softened material is discharged, leaving behind tuberculous cavities or ulcers; but in some cases the tubercle becomes encapsuled by increase of the fibrous tissue, or is rendered obsolete by a process of calcification.

Tuberculosis may be local or general, and general tuberculosis results from the invasion of the wall of a blood-vessel by a local tuberculous process. The bacilli which thus enter the blood are carried by it to all parts of the body, and this condition constitutes acute miliary tuberculosis.

ACUTE MILIARY TUBERCULOSIS.

What are the Predisposing Causes?

Youth, the subjects being children or young adults (20-30); a localised chronic tuberculosis of internal glands or of bones; infective diseases such as measles, whooping-cough, or enteric, which may reduce the resistance or reactivate the local disease.

Mention the Chief Forms.

Acute generalised miliary tuberculosis, acute pulmonary tuberculosis, acute meningeal tuberculosis.

What are the Symptoms of Acute Miliary Tuberculosis?

The onset is indefinite, accompanied by weakness, loss of appetite and flesh, and irregular fever of variable type, but oftenest continuous, and sometimes higher in the morning than at night. The pulse is rapid and feeble, the tongue dry, and the cheeks flushed; there is often delirium of the typhoid type, passing into coma; cyanosis may be marked,

and the breathing is rapid, but the pulmonary signs indicate only a slight bronchitis; the spleen is often enlarged, and leucocytosis is usually present. From its resemblance to enteric fever this is often called the *typhoid* form.

How would you distinguish the Disease from Enteric Fever?

By the irregularity of the fever, the absence of rose-spots, of Widal's reaction, and of the enteric bacillus, and by the presence of leucocytosis, and, on lumbar puncture, of a lymphocytic cerebro-spinal fluid. Tubercles may occasionally be seen in the choroid.

What is the Outlook, and how would you treat such a Case?

Death occurs in from one to three months. The treatment is purely symptomatic.

For the pulmonary and meningeal forms, and for chronic tuberculosis, see sections on Lungs, Nervous System, Peritoneum, etc.

ACTINOMYCOSIS (STREPTOTRICHOSIS).

What is this Condition?

A chronic infective disease occurring in man and cattle, and due to the presence of the *Actinomyces* (*Streptothrix bovis communis*) or "ray fungus."

What is the Etiology?

The streptothrix, as found in the tissues, consists of colonies of which the centre is formed of long branching filaments, containing within them spores or *gonidia*, while at the periphery the free ends of the sheaths of the filaments enlarge into radially arranged pear-shaped *clubs*. The filaments and spores are Gram-positive, the clubs Gram-negative.

The fungus is common on grain, and infection is conveyed to man by chewing straw, eating raw grain, or inhaling grain dust during threshing or chaff-cutting. The mouth is most frequently infected.

Describe the Morbid Changes.

The jaw, neck, and tongue are most commonly affected, and are the seat of nodular masses resembling osteosarcoma, and due to connective tissue proliferation. Suppuration takes place around the colonies, and deep-seated abscesses are the result. Internal organs (lungs, liver, intestines, peritoneum) may also be infected, and in these situations suppuration is early, and the affected organ is honey-combed by pus, in which the minute yellowish granules formed by the colonies may be seen to lie.

What are the Symptoms?

They vary with the site of the infection. In the neck or lower jaw an indurated violaceous tumour, partly cicatrised, partly inflamed, and discharging from various sinuses a thin pus containing the yellow granules; in the lungs bronchitis, bronchopneumonia, or abscess, with fever and wasting; in the intestines ulceration with diarrhœa; secondary peritonitis, or hepatic abscesses; occasionally bossy tumours of the skin; these are the prominent features of the disease. It is to be recognised rather by the discovery of the sulphur-yellow granules in the discharges (sputum, stools) than by the symptoms.

How is Actinomycosis to be treated?

The only drug of material use is potassium iodide in large doses (ʒi-ʒiii daily). Abscesses must be treated surgically, and when the disease is accessible, partial or complete removal may be attempted.

ANTHRAX.

What is Anthrax?

An acute specific infection, common to cattle, sheep, and some other animals, and to man, and due to the *bacillus anthracis*. It is also known as malignant pustule, wool-sorters' disease, and splenic fever.

How is it produced?

The *bacillus anthracis* is a slender non-mobile rod, from 5 to 20 μ in length, and often forming chains of organisms joined end to end. It is Gram-positive, and in the presence of free oxygen forms highly resistant spores. It may enter the body through a wound or scratch, by inhalation, or by swallowing. The disease is found in slaughterers, and in those who handle hides or wool (tanners, wool-sorters, etc.).

What are its Forms?

The disease may be local or general. The local form is subdivided into *malignant pustule* and *anthrax œdema*, while the general may affect the alimentary or respiratory tract.

Describe a Case of Malignant Pustule.

The disease is due to local inoculation, and begins upon an exposed part (face, hands, arms, etc.) where a small papule first appears, and is surrounded by inflammatory induration. The papule then becomes a vesicle, and within 36 hours an eschar forms in its centre, often enclosed in a ring of vesicles. The lymphatics are inflamed and the adjacent glands swollen. The temperature, at first high, may fall below normal, and death, which occurs in a few days, is preceded by delirium and collapse. A slow recovery may follow sloughing out of the eschar.

What are the Characters of Anthrax-Œdema?

There is no local pustule, but an extensive and spreading œdema. The constitutional symptoms are severe, and death is the usual end.

Describe the Respiratory Form of the Disease.

This, which is known as "wool-sorters' disease," is characterised by œdema and ulceration of the trachea and larger bronchi, and enlargement of the mediastinal glands. The disease begins with rigor, fever, vomiting, headache, and diarrhœa. Respiration is rapid, and there are signs of bronchitis or bronchopneumonia, usually with cyanosis. Death occurs within a few days.

What are the Symptoms of the Alimentary Form?

Vomiting, diarrhœa, colicky pain, and sometimes blood in the stools, associated with enlargement of the spleen. There are hæmorrhages from the swollen intestinal mucosa, and enlargement of the mesenteric glands.

On what does the Diagnosis depend?

On the characteristics of the pustule, on the discovery of the bacillus in the blood, sputum, urine, or fæces, or on the results of animal inoculation. The recognition of a possible source of infection is important.

What is the Treatment?

(*a*) For malignant pustule: free excision and cauterisation of the wound with pure carbolic acid or zinc chloride, the surrounding tissues to be frequently injected with 2% carbolic acid, and the strength to be maintained by stimulants and quinine.

(*b*) For internal anthrax: quinine in large doses, stimulants.

(*c*) For all forms: Sclavo's anti-anthrax serum, 30-40 cc. daily, injected subcutaneously. Many cases have recovered under this treatment.

TETANUS.

Define Tetanus.

A specific infective disease caused by the *bacillus tetani*, and characterised by tonic convulsions affecting the general musculature and also the muscles of the jaw.

What are its Causes?

The *bacillus tetani* is a slender rod-like organism, anaerobic and Gram-positive, with a rounded spore at one end ("drum-stick bacillus"). It is found in the soil and in manure, and infection is conveyed through abrasions or wounds of the hands or feet. Gardeners and stablemen are thus liable. Tetanus may also follow small operations, and sometimes occurs after confinement, and, through infection of the umbilical cord, in new born children.

What are the Morbid Changes?

Congestion of the central nervous system, sometimes with small hæmorrhages. The bacilli remain localised in the wound, and death is due to their toxins, which travel to the central nervous system along the peripheral nerves.

Describe the Symptoms.

Within ten days of the injury stiffness of the neck and jaw is experienced, and increases till the jaw becomes fixed (*trismus*, lock-jaw) by the spasm of the muscles. The angles of the mouth are drawn outwards into a grin (*risus sardonicus*) and the eyebrows elevated; and soon afterwards paroxysmal tetanic spasms involve the general musculature. The body is arched backwards in a painful spasm (*opisthotonos*), and even in the intervals the muscles remain rigid. Occasionally the body may be bent forwards (*emprosthotonos*) or twisted to the side (*pleurosthotonos*). The temperature may be normal, but in other cases rises to hyperpyrexia, reaching even 110° F. before death, which

is due to spasm of the glottis, interference with respiration, or exhaustion.

How would you distinguish Tetanus from Strychnine Poisoning?

In strychnine poisoning the jaws are affected *late*, and they do not remain rigid between the spasms.

What is the Treatment?

Keep the patient in a darkened room, avoiding any cause of external irritation; feed by the rectum, or through a nasal tube. Antispasmodics such as chloral, bromide, extract of Calabar bean, or morphia should be given in large doses; and chloroform may also be used. Antitetanic serum should be given subcutaneously, intravenously, or intracerebrally, but is not much use in acute cases. Treat the wound by antiseptics.

The serum may be used preventively, by hypodermic injection, and should be applied locally to a dubious wound.

HYDROPHOBIA (RABIES).

What is Hydrophobia?

An acute specific disease transmitted to human beings by direct inoculation from the bite of a rabid animal.

How is it produced?

The specific organism is unknown, but is probably ultra-microscopic. The virus passes the coarser Berkefeld filters.

The animal transmitting the disease is usually a rabid dog, but rabies has also followed bites from the cat, wolf, and fox. The virus is contained in the saliva, and those bitten through clothing often escape, the saliva being wiped off the tooth before it penetrates. Bites on uncovered parts are much more dangerous.

What are the Morbid Changes?

There is nothing characteristic save the presence of Negri's bodies in the nerve-cells of the central nervous system. These are rounded eosinophil bodies from 0·5 to 25 μ in diameter; they may be protozoal, or may express a cellular reaction. The brain and spinal cord are hyperæmic.

Describe a Case of Hydrophobia.

The incubation period is long and variable, but averages from six weeks to two months. It may be as short as a fortnight, or as long as six months. Towards its end the scar of the wound may become red and painful, and soon after the patient begins to be sleepless, restless, and irritable, and suffers from a choking sensation in the throat. Tetanoid convulsions follow, involving especially the muscles of deglutition and respiration, and affecting the general musculature to a somewhat less degree. Opisthotonos may be present. Attempts to swallow bring on a spasm, and even the offer of water may do so; the saliva is not swallowed, and is therefore frequently ejected with a hawking or "barking" noise. The face is often terror-stricken, delirium or hallucinations may be present, and towards the end convulsions may cease, death being preceded by paralysis and coma. The temperature usually ranges between 100° and 103° F.

What are the Diagnostic Points?

1. The certainty that the animal inflicting the bite was rabid. It should be killed, and its brain should be examined for Negri's bodies. Intracerebral inoculation of the medullary substance should also be performed upon a rabbit.

2. The distinction from tetanus is made by the history, the absence of rigidity between the paroxysms, and the presence of mental disturbance.

What is the Treatment ?

The developed disease can be treated only by palliation of symptoms; morphia and chloroform. A fresh wound should be cauterised or excised, and Pasteur's preventive treatment must be carried out as soon as it is known that the dog was rabid. This consists of the injection of an emulsion of the spinal cords of rabbits which have died of artificially induced canine rabies, the cords having been previously dried in air for a varying period. The injections are begun with a cord which has been exposed for fourteen days, and is no longer toxic; then a less attenuated virus (shorter exposure) is used, and so on till the maximum of virulence is reached. Immunity is thus obtained before the disease has time to develop.

Rabies is now practically extinct in Great Britain.

II. SPECIFIC INFECTIVE DISEASES OF TROPICAL AND SUBTROPICAL CLIMATES.

CHOLERA.

What is Cholera?

A specific infectious disease due to the *comma bacillus*, characterised by violent purging and vomiting, cramps in the legs, and collapse, and occurring in epidemic form.

What are its Causes?

The *comma bacillus*, really a spirochaete, occurs as a curved rod about half the size of the tubercle bacillus. It may be S-shaped, or may assume a spiral form. It stains with the usual dyes, is Gram-negative, and grows freely on ordinary media.

The disease is not directly contagious, but is conveyed by the stools or by water contaminated by them, the organism having its habitat in the small intestine. Food-stuffs, particularly vegetables, washed in the infected water may also spread the disease; it may be carried by flies: and convalescents may act as "carriers." Cholera is endemic in India, whence epidemics spread to other countries. It is most frequent in summer and autumn.

Describe the Morbid Changes.

These are centred in the small intestine, which is usually congested and filled with a watery turbid fluid, while Peyer's patches and the other lymphoid follicles are swollen. The other organs show the usual febrile changes,

the tissues and serous membranes are dry, and the blood is thick and dark.

What are the Symptoms?

They are divided into three stages, those of evacuation, collapse, and reaction. After a brief *incubation period*—two to five days—there may be a prodromal stage of diarrhœa, with malaise, headache, and vertigo; but very often the onset is sudden, and the first symptoms are those of—

The Evacuative Stage. Diarrhœa is profuse and violent, the discharges being at first fæcal, afterwards bileless, watery, and greyish in colour ("rice-water stools"). They contain abundant comma bacilli, and sometimes blood. Vomiting follows in a few hours, the vomitus being similar to the stools. Vomiting and motions are both very frequent, and the loss of fluid causes extreme thirst. There is little abdominal pain, but cramps in the legs are severe and constant. Prostration rapidly increases, and in a few hours there follows—

The Stage of Collapse, or Algid Stage. In this stage the prostration is profound, the pulse small and flickering, the face shrunken and cyanosed, the eyeballs sunk, and the extremities and even the breath cold. The rectal temperature may at the same time be from 102° to 105° F. Purging often ceases, but vomiting continues, and the patient is often restless. Suppression of urine may be followed by coma and death; or, after lasting from 12 to 24 hours, the state of collapse may be followed by—

The Stage of Reaction. The temperature rises, the complexion improves, the urinary secretion reappears, and a gradual recovery sets in.

What are the Chief Varieties?

Cases of cholera may be slight (*cholerine*), or very severe, death occurring from collapse before purging has

begun (*cholera sicca*). In the reactive stage inflammatory complications (nephritis, enteritis) may ensue, there may be a fatal recurrence of symptoms, or death may be due to uræmic coma.

Mention the Complications.

Inflammatory affections—pneumonia, pleurisy, nephritis, arthritis, etc.; gangrene or diphtheritic inflammation of the genitalia; conjunctivitis, or corneal ulceration; bed-sore.

On what does the Diagnosis depend?

On the identification of the organism in the stools, and on its cultural characteristics. It is not, as in enteric fever, to be found in the blood.

What is the Treatment?

Check the preliminary diarrhœa, if possible, with astringents and opium or morphia. In the evacuative stage treat the vomiting by mustard externally, and ice to suck, the cramps by friction and morphia cautiously given. Food and stimulant must be withheld. Collapse is to be met by warmth, and, if it is very severe, by intravenous or subcutaneous injections of normal saline. In the reactive stage give light food often and in small quantities. Dry cupping and fomentations to the loins may be tried for suppression of urine.

What are the Preventive Measures?

Isolation, disinfection of excreta and fomites, attention to sanitation. Drinking-water and milk must be boiled, and attacks of diarrhœa promptly treated. Haffkine's preventive vaccine is extensively used in India, and with considerable success.

PLAGUE.

Define Plague.

A specific infectious disease due to the presence in the blood and tissues of *Bacillus pestis*, and accompanied either by buboes or by pulmonary inflammation.

What are the Characters of the Organism?

It is a very short (1 to 1·5 μ), thick bacillus, presenting polar staining. It is readily cultivable on ordinary media, stains with ordinary dyes, and is Gram-negative.

What are the other Etiological Factors?

The disease is usually conveyed from infected rats to man by means of fleas. In the bubonic form direct contagion is not common, although the organism is found in the fæces and urine, and may thus be conveyed by fomites. Houses may also be infected in this way. In the pulmonic form the disease is conveyed by the sputum, in which the organisms are abundant. Dirt, overcrowding, and bad sanitation are contributory factors. Asia is the headquarters of plague, and within modern times none but very small epidemics have spread to Europe.

Describe the Morbid Changes.

The characteristic buboes consist of swollen and inflamed lymphatic glands, surrounded by a hæmorrhagic exudation into the areolar tissue. In pneumonic plague there are patchy areas of consolidation in the lungs. The spleen in both forms is enlarged, there are wide-spread hæmorrhages into the various organs and tissues; and the other changes are those of the febrile state. The bacillus is found in the blood, the glands, and all the tissues and organs.

Describe a Case of Bubonic Plague.

The *incubation period* is short (three to five days), and the disease begins with a brief premonitory malaise, followed by a sharp rise of temperature (104° F. or more), with vomiting, sometimes rigor, and suffusion of the eyes. The expression is dazed or anxious, and the gait staggering. The pulse is rapid; the tongue furred, at first moist, but soon becoming brown and dry; the urine is scanty and may be suppressed; and the typhoid state may set in, with delirium or coma. From the second to the fifth day buboes appear in the groin, axilla, or neck. They are usually single; the swelling is large and tender, and the skin over it is inflamed. Death may take place in a few days, but in favourable cases the bubo softens and is discharged in the form of pus, the suppuration being often protracted.

What are the other Forms of the Disease?

Pneumonic plague, in which there are no buboes. The general symptoms are severe, and there is cough with a hæmorrhagic but not rusty expectoration. Moist râles at the bases, and patches of bronchopneumonic consolidation, may be found. The sputum yields the bacillus almost in pure culture. The mortality is very high, and death is early.

In *septicæmic plague*, without any conspicuous local symptoms in lungs or glands, the disease assumes a fulminating course, and the patient may be dead within twenty-four hours. *Pestis minor* or *ambulatory plague* is a mild type of the disease with slight fever and slight glandular swelling.

How is the Disease to be diagnosed?

The rapid onset, the extreme prostration, the dazed expression, and the suffusion of the eyes should suggest plague in the early stages; later, the bubo confirms the

suspicion; but in many cases certitude is attainable only by bacteriological examination of the blood, sputum, urine, or fæces.

What is the Treatment?

Nursing, stimulation, support of the strength, and relief of symptoms. Ice should be applied to the buboes, which must be opened when pus has formed. Of the various anti-plague sera Yersin's is the best; it should be given early, and at least in part intravenously. The dose is 60-150 cc.

Prophylaxis includes isolation, disinfection of excreta, clothes, bedding, and infected houses, and the use of Haffkine's prophylactic vaccine where the disease is epidemic. Rats must be hunted out and destroyed, their bodies being burnt.

DYSENTERY.

What is Dysentery?

It is the name for a condition characterised by the frequent passage of stools, either hæmorrhagic or containing much mucus, such passage being accompanied by griping or tenesmus.

What are its Varieties?

There are two forms, the one due to a bacillus, the other to an amœba, and named respectively bacillary and amœbic dysentery.

Bacillary Dysentery.

What is the Etiology of this Form?

The *bacillus dysenteriæ*, or bacillus of Shiga, resembles that of enteric fever morphologically and in its Gram-negative staining reaction, but it is non-mobile. The

disease is both endemic and epidemic in tropical climates, infection being conveyed by fæces, fomites, flies, infected water or soil, and "carriers." In temperate climates it may occur sporadically, and in conditions of defective sanitation (prisons, warfare) epidemically.

What are the Morbid Changes?

Their principal seat is in the large intestine, which, in acute bacillary dysentery, shows intense hyperæmia and swelling of its mucous membrane, sometimes going on to the formation of ulcers, which, beginning on the summit of the folds, extend both peripherally and into the substance of the bowel. In other cases necrosis may occur, with the formation of a pseudomembrane (diphtheritic dysentery). In chronic cases the whole mucosa is thickened, as are also the edges of the ulcers, which in healing cause much contraction of the lumen, and give rise to partial strictures of the bowel.

Describe the Symptoms.

The *incubation period* varies from two to eight days, but is usually from two to three. There may be some preliminary diarrhœa, or the onset may be sudden. The calls to stool are very frequent, and there is much abdominal pain and griping (tormina), with a sense of weight in the rectum (tenesmus). The stools are small, contain the bacillus, and consist of slimy mucus or of blood, occasionally with sloughs. The motion gives no relief to the griping, and the patient continues to strain. The general symptoms are moderate fever, thirst, a foul tongue, and rapid loss of strength. Death may be due to exhaustion, perforation, or pyæmia; in cases that recover, convalescence may occur in two or three weeks, but is often slow, and chronic dysentery may be left behind.

When the disease becomes *chronic*, the diarrhœa is less frequent, and the stools, still loose, are partly fæcal,

partly mucous, and sometimes resemble "frog's spawn." Progressive anæmia and emaciation lead to death by exhaustion.

Complications are comparatively rare in bacillary dysentery, and hepatic abscess does not occur unless the amœbic form is also present.

What is the Treatment?

In the acute disease, rest in bed, with a fluid diet; relief of pain by opium or morphia; and either a laxative treatment, or ipecacuan. Ipecacuan is thus given:—the stomach being empty, m. xx of laudanum are given, and soon afterwards gr. xx-xxx of ipecacuan, the patient being kept at rest on the back, and the dose repeated if vomiting recurs. This dose may be given thrice daily for two or three days. In the laxative treatment, a drachm of sodium sulphate is given every two hours till a feculent motion is produced, and blood and mucus have disappeared; bismuth and opium may then be given till the motions are solid.

Various antidysenteric sera, among them Shiga's, have been used, and good results are claimed from them. Vaccines have also been employed.

In chronic dysentery ipecacuan in smaller doses, dieting, and irrigation of the colon by astringent solutions (*e.g.* Argent. Nit. gr. x-xv to the pint) introduced in large quantities (two pints or more), are the chief measures.

Amœbic Dysentery.

Describe the Amœba Dysenteriæ.

It is a rounded unicellular organism with a clear ectoplasm, a granular endoplasm, and an eccentric nucleus, and is about five times the size of a red corpuscle. In the resting stage it becomes cystic, and resists drying; in the active stage it shows free amœboid movement. It chiefly

inhabits the rectum and colon, but may be found in the liver or stomach. From its power of penetrating the the tissues it is also called *A. histolytica*. It is the chief cause of endemic, as the bacillus is of epidemic dysentery.

What are the Contributory Causes?

Contaminated water, and uncooked vegetables (salads, etc.).

How do the Morbid Changes differ from those of Bacillary Dysentery?

The large intestine presents small gelatinous swellings of the mucosa, partially ulcerated, with necrosis and sloughing of the underlying tissues, so that the ulcers have *undermined edges*. The amœbæ are chiefly found beyond the ulcerated area, in the submucous and muscular coats, and sometimes they may be discovered in the portal capillaries. *Hepatic abscess* is frequent in this form of dysentery.

What are the Characteristic Symptoms?

The acute form closely resembles bacillary dysentery, save that amœbæ, not bacilli, are found in the stools. In the chronic form there is a tendency to alternating periods of constipation and diarrhœa, with marked emaciation.

What are the Chief Complications?

Hepatic abscess, usually single. It ends by rupture, most commonly into the lung, but in other cases into the pleura or peritoneum. Perforation of the bowel may take place, or partial stricture; and sometimes arthritis, or portal pyæmia.

How would you treat Amœbic Dysentery?

By the relief of symptoms as in bacillary dysentery; by irrigation of the bowel, especially with solutions of quinine (1 in 5000 to 1 in 1000); and by hypodermic injections of

emetine hydrochloride. This recently advocated drug is given in doses of gr. $\frac{1}{6}$ to gr. $\frac{1}{3}$, and as much as gr.iss may be injected daily. It has met with much success, and is effectual even in hepatic abscess, unless there is a microbic complication, in which case the treatment of the abscess becomes surgical.

MEDITERRANEAN (MALTA) FEVER.

What is this Disease?

A specific infection characterised by a prolonged course of intermittent fever with enlargement of the spleen, and due to the *micrococcus melitensis.*

How is it transmitted?

The organism is a small Gram-negative coccus, found in the spleen and often in the blood, and sometimes discharged in the urine and fæces. It is transmitted to man, in Malta at all events, by goats' milk, while in other regions it may possibly be transmitted by mosquitoes. The disease is endemic in Malta and on the Mediterranean coasts, and also occurs in South Africa, China, parts of America, India, and the West Indies.

What are the Symptoms?

The incubation period varies from six days to four weeks. The onset is insidious, and in the first week fever, headache, sleeplessness, anorexia, constipation, and profuse sweating, with enlargement of the spleen and liver, are the chief symptoms. The acute symptoms then disappear, but weakness and sweating persist, and the temperature remains high and irregular, often presenting an *undulatory* type with apyretic intervals. In all, the fever may extend over several weeks or months, and disappears very gradually. Arthritis, and sometimes orchitis, are the chief complications, and marked anæmia,

with leucopenia, accompanies the disease. Some few cases (*malignant* type) are fatal, but the great majority recover.

What is the Treatment?

Relief of symptoms, and the general treatment of fever. Vaccines are useful prophylactically, and infected goats must be removed from the flock.

MALARIAL FEVER.

Define Malaria.

It is a specific infective disease due to the *hæmamœba* or *plasmodium malariæ*, and characterised in its milder forms by periodic attacks of intermittent fever, in its graver forms by fever less regularly periodic, and either remittent or continuous.

What are its Causes?

It is a disease of tropical and subtropical climates, the organisms entering the blood by the bite of mosquitoes of the genus *anopheles*. Conditions favourable to the mosquito are therefore favourable to malaria—the warm season of the year, abundant vegetation, and stagnant surface water.

Describe the Life-History of the Parasite.

The parasite, which is a protozoon, occurs in man in three varieties, the organisms of tertian, quartan, and æstivo-autumnal fever, named respectively *hæmamœba* or *plasmodium vivax*, *h.* or *p. malariæ*, and *hæmomœnas* or *p. præcox*. The development of the tertian parasite is as follows. In its youngest stage it appears within the red corpuscles as a colourless amœboid body (*amœbula*, *trophozoite*), which grows rapidly, becomes pigmented, and when it has nearly filled the red corpuscle, undergoes

segmentation, dividing into a "rosette"-shaped body consisting of 15-20 *enhæmospores*. The corpuscle ruptures, and the liberation of the spores determines an attack of fever. The enhæmospores attack fresh corpuscles, and repeat the same cycle of development, which in the case of the tertian parasite lasts 48 hours.

The above describes the asexual cycle; but some of the amœbulæ do not become segmented, merely enlarging within the corpuscles to form rounded *gametocytes*, of which the male element is smaller than the female. When these are drawn into the stomach of the mosquito they burst the corpuscles, and the *microgametocyte* (male element) puts forth three or four flagella (*microgametes* or spermatozoa) which, becoming detached from the gametocyte, penetrate and fertilise the female cell. This now becomes an elongated pointed body (*zygote*, *oökinete*) which penetrates the mosquito's stomach wall, and developes on its outer surface into a capsule filled with thread-like *exotospores*. The capsule ruptures, and the exotospores are carried to the salivary gland and duct of the mosquito, entering the human blood at the insect's next bite, and there penetrating the red cells and becoming amœbulæ.

How does the History of the Quartan and Æstivo-autumnal Parasites differ from the above?

The quartan parasite undergoes segmentation into only 6-12 enhæmospores, and its asexual cycle lasts 72 hours. The æstivo-autumnal parasite undergoes segmentation into 6-20 enhæmospores, its asexual cycle lasts 24-48 hours, its amœbulæ are ring-shaped, and its sexual forms (gametocytes) are *crescentic*, not rounded.

How do these Differences affect the Clinical Forms of Malaria?

The fever coincides with the liberation of the enhæmospores; therefore in tertian ague there is a paroxysm every second day, and in quartan every third day. But in

tertian ague there may be two broods of spores, coming to maturity on alternate days, and so causing a daily paroxysm—double tertian: and in quartan ague there may be two broods—two daily paroxysms and a day's interval; or three, with a paroxysm every day—quotidian ague. Similar statements are true of the æstivo-autumnal parasite.

What are the Morbid Changes in Malaria?

During the paroxysms the spleen is enlarged and soft; the liver is also enlarged; and the bone-marrow, kidneys, and brain are congested. These organs are full of plasmodia; there is great destruction of red cells, but though there is a relative increase of lymphocytes, the total number of leucocytes is diminished (leucopenia). Chronic malarial conditions lead to a state of cachexia, in which, through repeated congestive attacks, the spleen becomes greatly enlarged, firm, and hard, with a thickened capsule and thickened trabeculæ. The liver is often enlarged, and there is great anæmia.

Describe an Attack of Intermittent Fever (Ague).

After an *incubation period* of from six to twenty days, there may be a day or two of malaise, followed by the sudden onset of the attack. This has three stages.

1. The *Cold Stage* is marked by violent shivering, coldness, and lividity of the skin, with elevation of the rectal temperature (104°-106° F.), smallness, hardness, and rapidity of the pulse, and an excessive output of clear, pale urine. This stage lasts from a few minutes to an hour or even two hours.

2. The *Hot Stage* directly succeeds it, the cold skin becoming flushed and burning, the pulse full and bounding, the temperature even further raised, the urine scanty and of high specific gravity, and the spleen palpably enlarged. This stage may last for several hours, and is followed by—

3. The *Sweating Stage*, in which, with the onset of free perspiration, the temperature gradually falls, and the patient is restored to the normal condition. This stage may occupy from two to four hours.

The parasites are present in the peripheral blood during the attack. (*See* Chart, p. 95.)

What are the Characteristics of the Remittent Type of the Disease?

It is due to the æstivo-autumnal parasite, and commonly occurs in the tropics, but may also be met with in temperate climates in late summer and autumn. The symptoms are furred tongue, pain and tenderness in the epigastrium, diarrhœa, vomiting, often jaundice, and delirium, which may pass into coma. The temperature may show marked remissions, but not intermissions, or there may be intermissions at irregular intervals. The duration of the paroxysm is longer than in ague, and the whole attack may last a few days or as long as three weeks, in which case it is often fatal. (*See* Chart, p. 95.)

What are the other Forms of Malaria?

The disease may assume the *pernicious* form, due, like the remittent, to the æstivo-autumnal parasite, occurring mainly in the tropics, and affecting those whose vitality is diminished, *e.g.* by alcohol or hardship. The attack begins suddenly, and may assume one of several types. *Hyperpyrexia* may be the characteristic feature (temperature 107°-110° F. or more); or *nervous* symptoms—comatose, convulsive, or paralytic; or an *algid* type may appear, in which the cold stage is followed not by fever but by collapse; and lastly a *dysenteric* type with intestinal hæmorrhages and jaundice. All these forms are of very grave prognosis.

Malarial cachexia is the result of repeated attacks of malaria, or of long residence in malarial countries

after an attack, and is characterised by marked anæmia, a tendency to hæmorrhages, occasional irregular fever, enlargement of the liver, and great enlargement of the spleen ("ague-cake").

How is an Attack of Ague to be treated?

By quinine, which is the specific remedy. During the attack it must be given in large doses (gr. x thrice daily), and the patient should be kept under its influence for a week after the parasites have disappeared from the blood. Thereafter it may be gradually reduced, but should not be entirely discontinued for at least three months.

What is the Treatment of the Graver Forms?

Intramuscular injection of quinine hydrochloride or hydrobromide in doses of from three to ten grains. The drug may be combined with the same amount of urethane.

What is the General Treatment of Malaria?

It includes the prophylactic use of small doses of quinine, drainage of malarial districts, covering the pools with a film of kerosene to destroy the larvæ of *Anopheles*, and the use of mosquito nets and screens. Besides the use of quinine, symptomatic treatment is necessary in the attacks. In chronic cases arsenic should be given, alone or with iron, and the subject should leave the infected district.

BLACKWATER FEVER.

What is Blackwater Fever?

A disease of obscure etiology, occurring in tropical countries in association with malaria, sometimes accompanied by the presence of scanty malarial parasites, and characterised by jaundice and hæmoglobinuria.

Describe the Symptoms.

They set in abruptly, usually, though not constantly, some hours after a large dose of quinine. An initial rigor is followed by breathlessness, "air-hunger," and vomiting. These symptoms rapidly pass off, and the next urine voided is found to be dark-red or brown from the presence of hæmoglobin, and to contain albumin, renal epithelium, and hyaline casts. The spleen and liver are enlarged, and jaundice is present. During the hæmoglobinuria the temperature is raised, and the pulse is rapid. The attack may last for about a week, or may continue, with intermittent hæmoglobinuria, for several weeks. In fatal cases death may be due to coma or collapse, and may be preceded by suppression of urine.

What is the Nature of the Disease?

The symptoms are indicative of an acute hæmolysis, of which the cause is not certainly known. Some attribute it to the abuse of quinine, but there is evidence in the direction of its being due to a special parasite. Whatever view is held, there is no doubt of its close relationship to malaria.

What is the Treatment?

Mainly symptomatic; it is to be directed chiefly against collapse and suppression of urine. Stimulants, therefore (ether, strychnine), and free flushing of the kidneys are indicated. If malarial parasites are present quinine may be *cautiously* given.

YELLOW FEVER.

Define this Disease.

An acute specific infection characterised by jaundice, albuminuria, and hæmorrhage from the stomach.

What are its Causes?

The specific organism is not known, and is probably ultramicroscopic. The disease, which has its principal seat in the West Indies and on the coasts of Central and South America and West Africa, is conveyed by the bite of a species of mosquito (*Stegomyia fasciata* or *calopus*), which is usually found at comparatively low levels and in crowded towns.

Describe the Morbid Changes.

They consist in fatty degeneration of the liver, into which small hæmorrhages may occur, acute inflammation of the kidneys, and engorgement of the stomach, which contains altered blood. The spleen is not enlarged.

What are the Symptoms?

The incubation period lasts from three to six days after the bite of the insect. The symptoms then set in abruptly, with rigor, backache, and fever rising rapidly to 104° F. or more. The pulse is slow in proportion to the temperature, and tends to became slower as the disease advances. Albuminuria is usually present from the outset, and there is also vomiting.

On the fourth day the temperature tends to fall, and in mild cases convalescence may begin. Usually the remission is followed by a rise of fever; jaundice, if not present before, now appears; vomiting is aggravated, and altered blood is brought up ("black vomit"); other hæmorrhages may occur; the urine is very scanty, and may be suppressed; and death may be due to collapse, coma, or the typhoid state. The mortality varies greatly in different epidemics.

How is such a Case to be treated?

Symptomatically; there is no specific drug. After an initial purge, fever must be relieved by sponging, vomiting

by ice or iced water, collapse by stimulants, suppression by cups to the loins, and so on. Prophylactic treatment deals with the mosquito and its breeding places, as in the case of malaria.

YAWS (FRAMBŒSIA).

What are the Main Features of the Disease?

It is a chronic specific infection due to the *Spirochæte pertenuis*, an organism which closely resembles the parasite of syphilis. The disease, which occurs in the tropics, is transmitted by the discharges of the cutaneous lesions, which may be inoculated direct, through the bites of insects, or through infected clothing. The contagion is not transmissible through unbroken skin.

Symptoms develop after an incubation period of from two to eight weeks. This is followed by malaise, and this in turn by an eruption, of which the primary lesion appears at the seat of inoculation, while the secondary eruption, similar to the primary, is widely distributed. The eruption consists of itchy subcuticular papules, at first very small, but rapidly enlarging until the epidermis over them gives way, and a prominent raw surface is left, covered with a sero-purulent exudate. This dries into a crust, under which the "yaw" usually heals in a few weeks, although ulceration sometimes occurs. Successive crops of the eruption may prolong the duration of the disease for many months.

How is Yaws to be treated?

By isolation, local applications, and strict cleanliness, while internally iodide of potassium, mercury, or arsenic may be given. The treatment is similar to that of syphilis, and good results have been obtained from salvarsan.

TRYPANOSOMIASIS (SLEEPING SICKNESS).

Briefly describe the Characteristics of Sleeping Sickness.

It is a disease endemic in Africa, due to a parasite which inhabits the blood of wild herbivora, and is transmitted to man and domestic animals by the bite of a species of tsetse fly (*glossina palpalis*), and also by the common tsetse fly (*glossina morsitans*). Different varieties of the parasite attack different animals. That of man (*trypanosoma gambiense*) is a spindle-shaped, actively mobile organism, measuring 17 to 28 μ in length, and 1·4 to 2 μ in breadth. Near its middle there is a large *macronucleus*, and near the posterior end a small *micronucleus* (*centrosome*) from which originates an *undulating membrane* which runs forward along one surface of the organism, and ends in a flagellum which projects beyond the anterior end.

The lesions of trypanosomiasis, which are due to the presence of the organisms in the cerebrospinal fluid, consist in chronic inflammatory changes in the cord, cerebral cortex, and membranes. The lymphatic glands are also enlarged.

The *incubation period* is prolonged, but not definitely known. The first symptoms—attacks of fever of malarial type, with enlargement of the glands—recur at irregular intervals, and may last for years before the nervous system is affected. When this at length happens, lassitude, anæmia, debility, and transient œdema, with slight fever, are succeeded by an increasing somnolence which ultimately passes into coma, and may be attended by paralysis or bedsores. Wasting is marked, and death is the invariable end. The diagnosis rests upon the recognition of the parasite in the blood, or in the juice of the enlarged glands.

Arsenic is the sole drug that is useful in treatment, and a few cures have been recorded from its use in the form of salvarsan.

KALA-AZAR.

(Tropical Splenomegaly).

What is the Nature of this Disease?

It is due, like sleeping sickness, to a protozoal parasite, occurs in India, Ceylon, China, and Egypt, and is characterised by progressive anæmia and wasting, fever, hæmorrhages, and enlargement of the spleen. The parasite, *Leishmania Donovani*, which is found in the spleen, liver, bone-marrow, and mesenteric glands, is a round or oval body measuring 2·5 to 3·5 μ, and containing a rounded macronucleus and a rod-shaped micronucleus. These Leishman-Donovan bodies are a stage in the development of a flagellate protozoon (*Herpetomonas*) which is transmitted to man by the bite of the bed-bug. The parasite causes enlargement of the liver and spleen, proliferation of the bone-marrow, and intestinal ulceration. The *symptoms* are those of irregular fever, with anæmia, hæmorrhages such as epistaxis or purpura, wasting, enlargement of the liver and spleen, diarrhœa, and transitory œdema. Leucopenia is constant. Untreated cases end fatally, usually in a few months.

The *diagnosis* depends upon the recognition of the parasite, obtained by splenic or preferably by hepatic puncture.

Quinine in large doses and arsenic (atoxyl) have been used in treatment, without much effect. Salvarsan may give better results.

LEPROSY.

Define this Disease.

A chronic specific infection due to the *bacillus leprœ*, and occurring in two forms, the one marked by the presence of granulomata in the skin and mucous mem-

branes, the other by affections of the peripheral nerves giving rise to anæsthesia, paralysis, and trophic changes.

What are its Causes?

The disease is wide-spread in tropical countries, but also occurs in parts of Europe (Norway, Greece, Turkey) and of America. If it is contagious, very prolonged contact is necessary for infection; and it has been recently suggested that it may be transmitted by the bed-bug, in which insect the parasite has been found in leper colonies. Both sexes and almost all ages are liable, although infants rarely suffer. The parasite, which is an acid-fast bacillus resembling that of tuberculosis, and like it allied to the *streptothriceæ*, is abundantly present in the skin and mucous membranes, nerve-trunks, lymphatic glands, and elsewhere.

Describe the Morbid Changes.

The *tubercular form* is characterised by the presence of nodules of granulation tissue in the skin and mucous membranes. These nodules, in which the bacilli are very numerous, may ulcerate and discharge freely. In the *anæsthetic form* there is a diffuse infiltration of the trunks of the peripheral nerves.

What are the Symptoms?

The tubercular form begins with a febrile attack, during which reddish or brownish erythematous patches appear upon the skin. These recede as the fever disappears, and are followed by slight pigmentation or induration. Successive attacks of fever and eruption lead to the appearance of brownish nodular elevations, causing thickening of the skin, particularly on the face. The nodules are widely distributed, and as they tend to break down, ulcers may appear upon the limbs. The mucous membranes suffer; the cornea ulcerates, and blindness is frequent; the interior of the nose may be destroyed; and the larynx may also

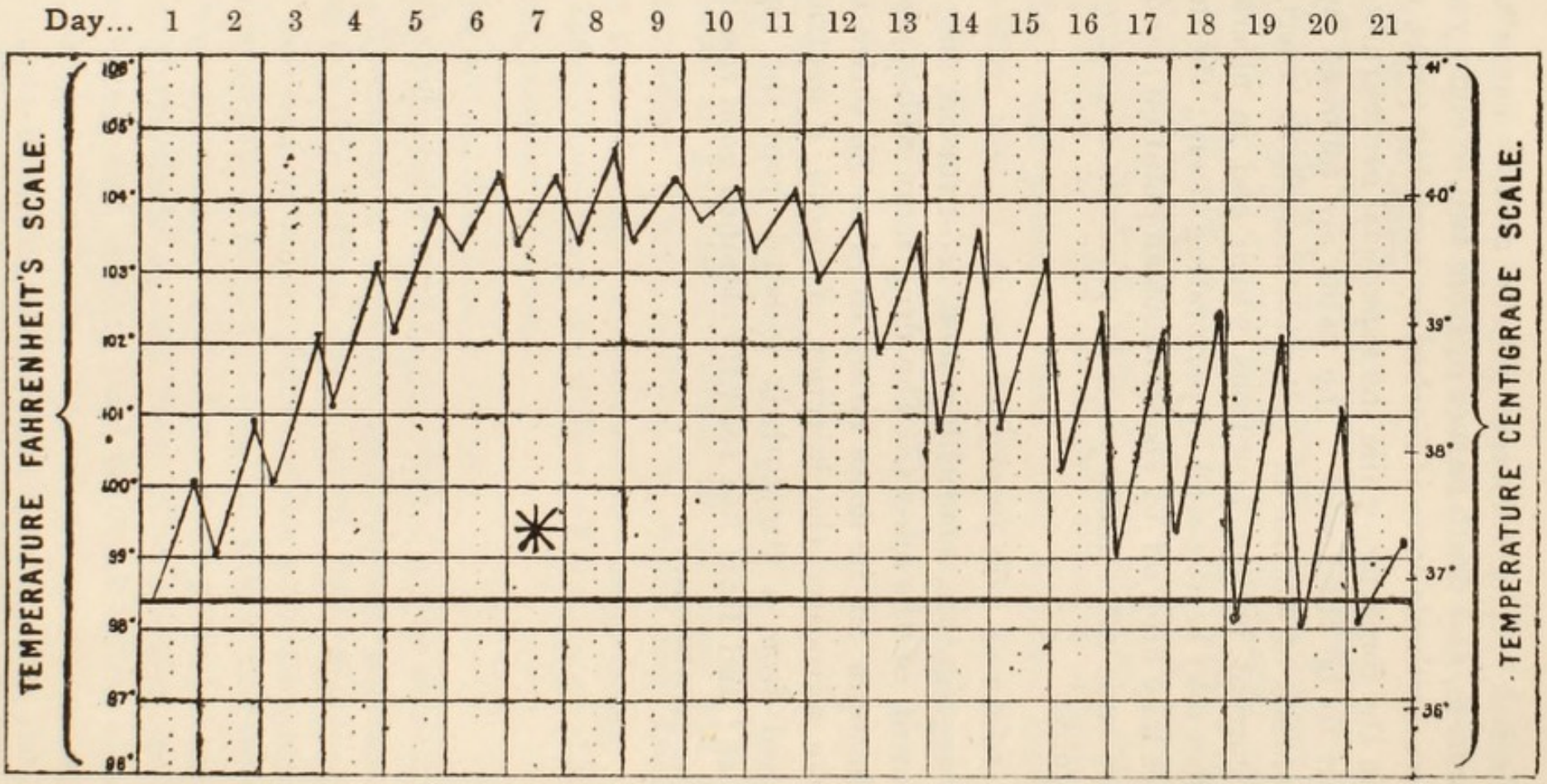
be affected. The recurrent febrile attacks cause much prostration, and death ensues in from two to eight years.

The anæsthetic form is marked by neuralgic pains, often first in the forearms, while the superficial nerve-trunks can be felt to be thickened. The pains are followed by the appearance of whitish or yellowish anæsthetic areas, and by recurrent bullous eruptions which end in ulceration. The feet may be attacked by perforating ulcers; the fingers or toes may drop off by spontaneous amputation; and the disease ends after many years in paralysis or gangrene of the limbs.

How is Leprosy to be treated?

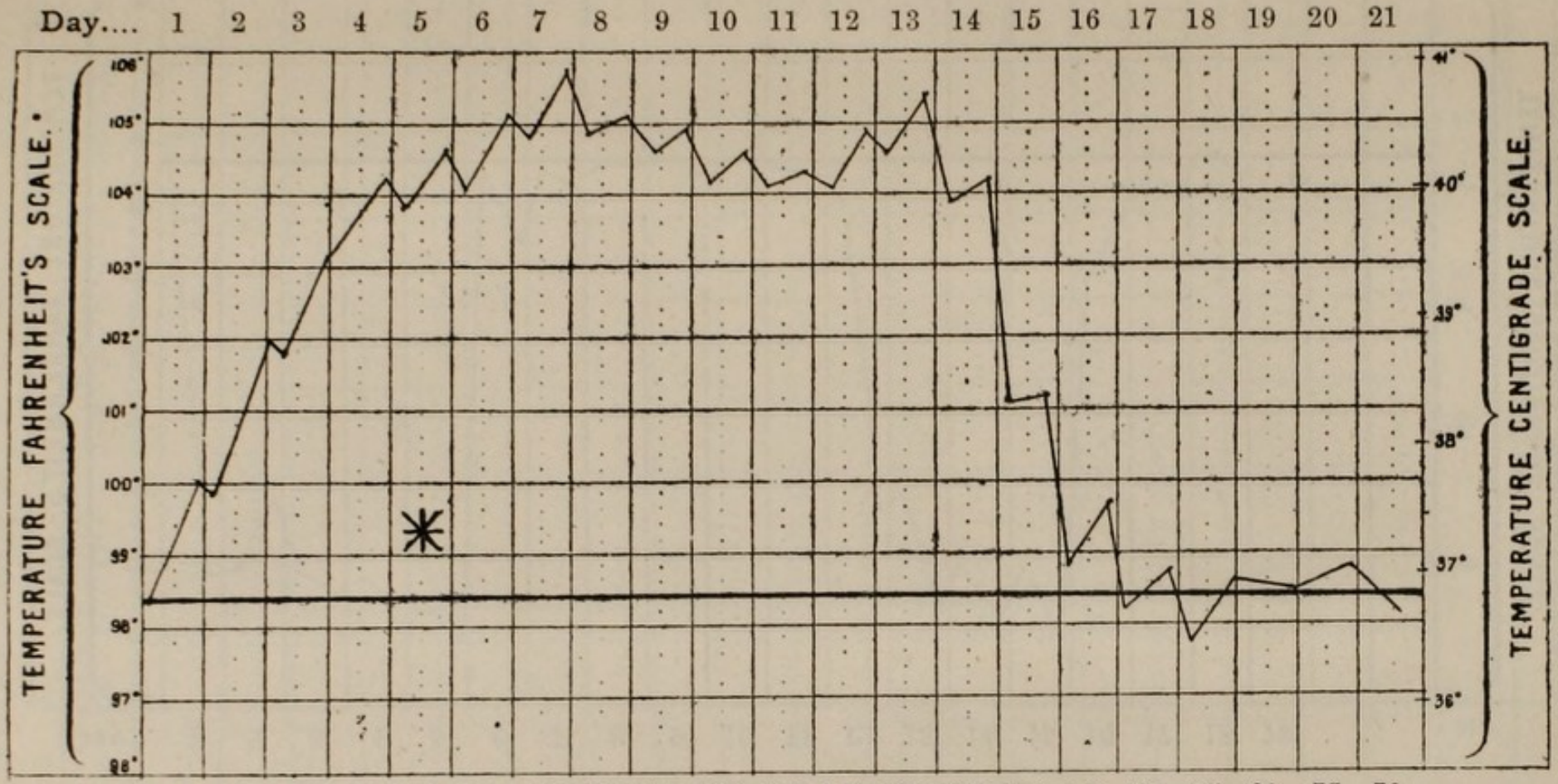
By segregation, nourishing food, cod-liver oil, and local treatment of the ulcers. The drug most widely used is chaulmoogra oil in doses rising to ℥i or ℥ii thrice daily. Deycke advocates the injection of *nastin*, a neutral fat obtained from a streptothrix isolated from the leprous nodules; and Rost has recently obtained good results by vaccine treatment.

TYPHOID FEVER.



The date of appearance of the rash is indicated by *

TYPHUS FEVER.



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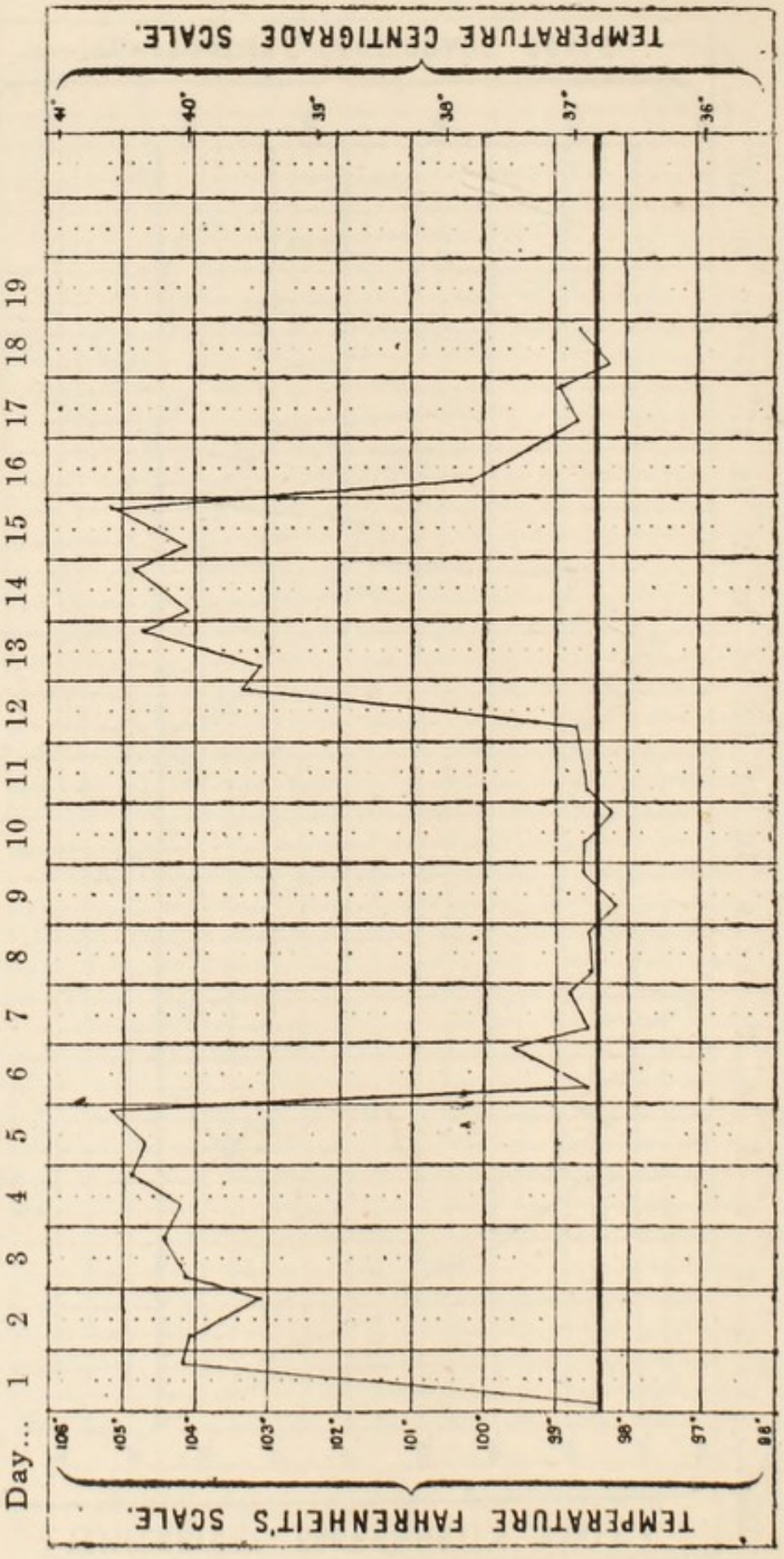
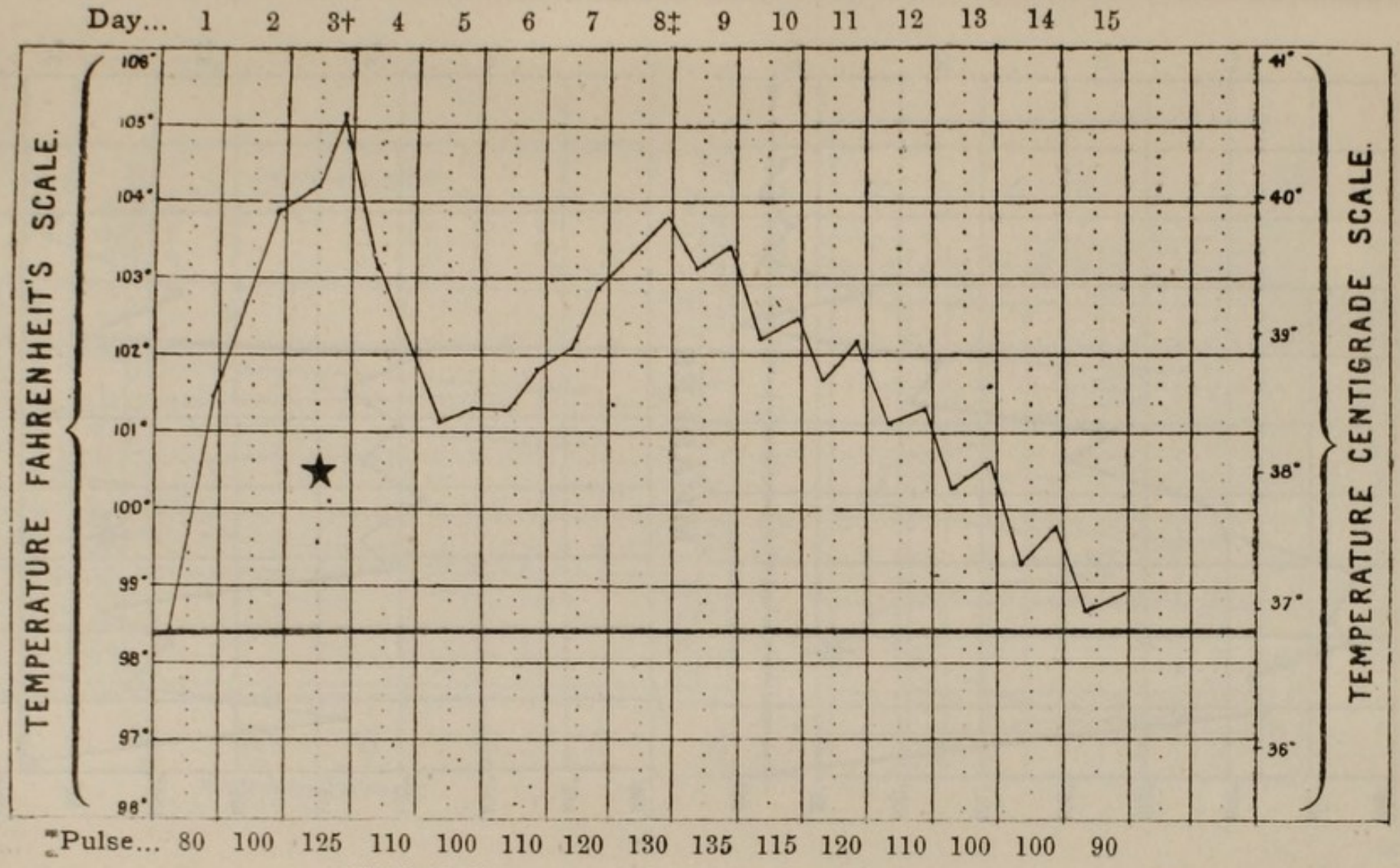
RELAPSING FEVER.

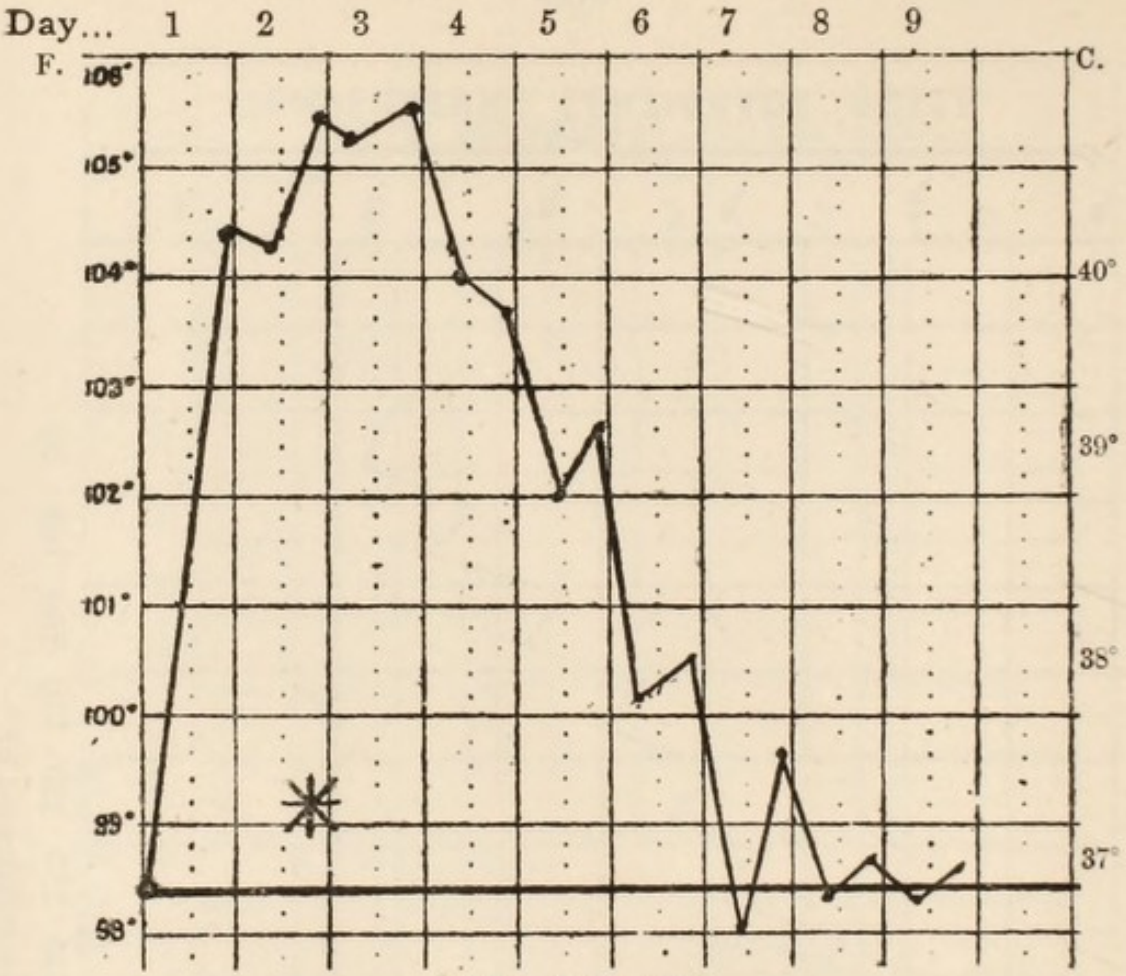


SMALL-POX.

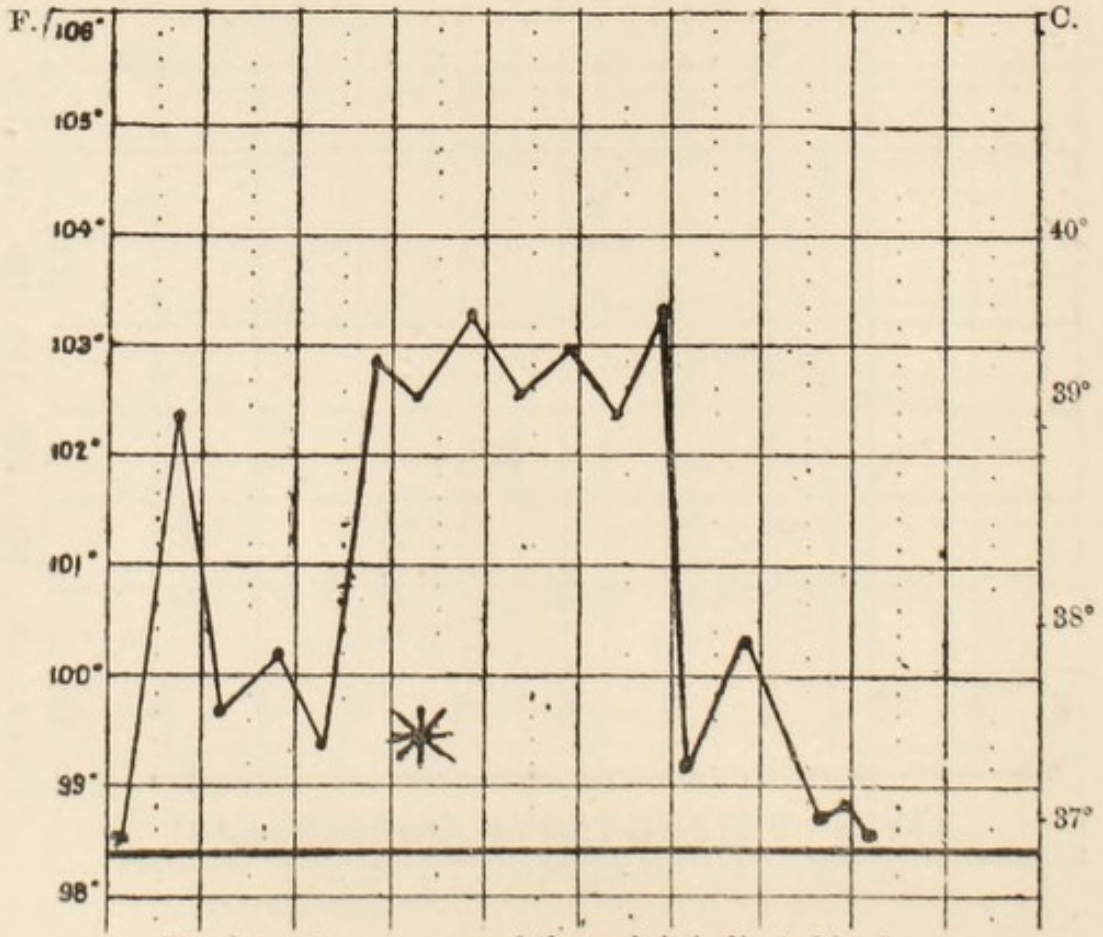


* The date of appearance of the rash is indicated by *
† Primary Fever.
‡ Secondary Fever.

SCARLATINA SIMPLEX.



MEASLES.



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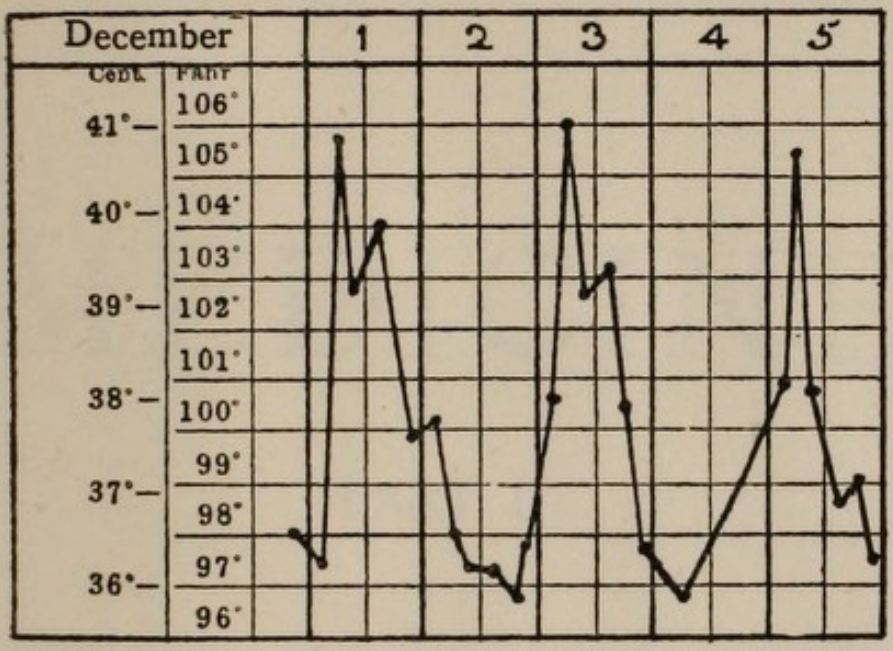
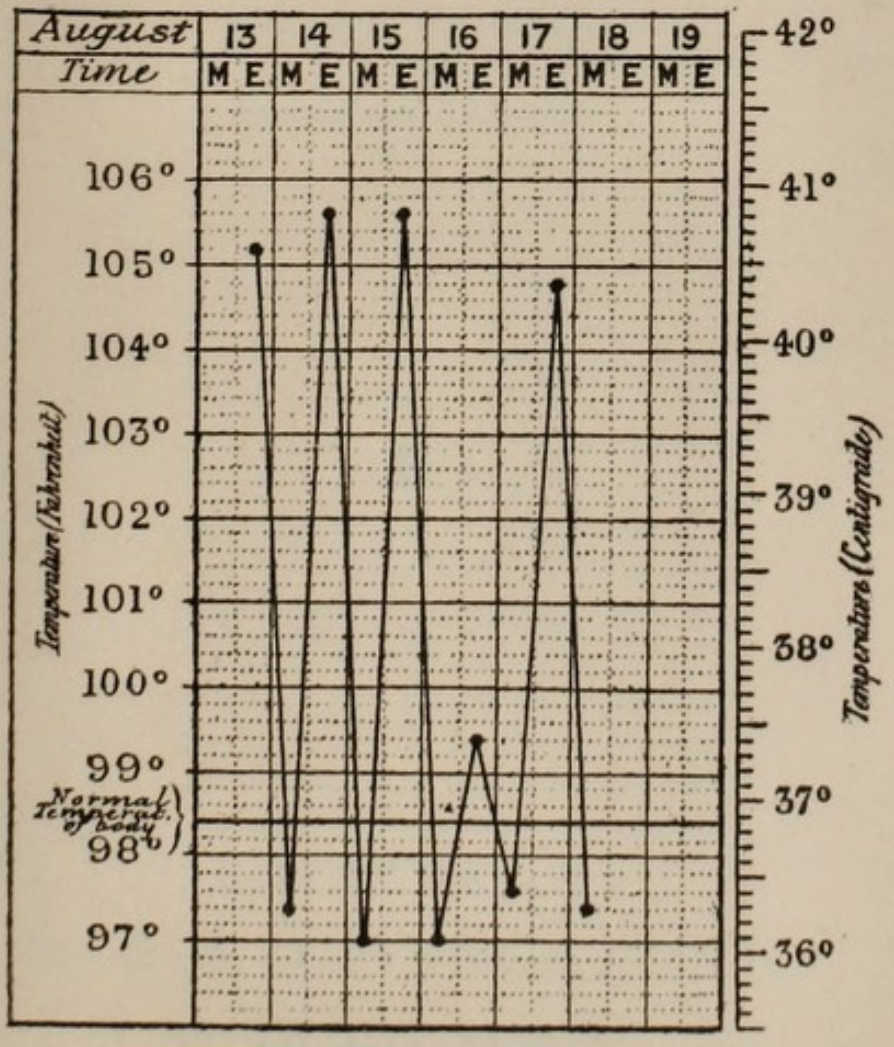
INTERMITTENT FEVER:

from a case of tertian ague.



QUOTIDIAN (Double Tertian) AGUE



CATECHISM SERIES

MEDICINE

PART II.

SECOND EDITION

Revised and Enlarged

EDINBURGH
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CONTENTS.

MEDICINE

PART II.

I. DISORDERS OF NUTRITION.

GOUT.

What is Gout?

A disease characterised by an excess of uric acid in the blood and by deposits of urate of soda in the joints, and manifesting itself by attacks of acute arthritis, leading to deformity of the joints.

What is its Etiology?

The disease is markedly hereditary. It principally affects men of middle age, although women and adolescents are not exempt, and its onset is favoured by habitual over-indulgence, whether in eating or drinking. Rich wines and beer are more provocative of it than spirits. It is commonest among the well-to-do, but "poor man's gout" is not unknown; and it is frequently seen in painters and others exposed to the influence of lead.

What is the Pathology of Gout?

The essential pathology of gout is still uncertain. A constant feature is the excess of uric acid in the blood, along with a deficient elimination of uric acid in the urine, particularly before an attack. But an excess of uric acid in the blood does not in itself produce gout, and is found in

conditions, such as pernicious anæmia, in which there are no gouty symptoms. Those who maintain the uric acid theory account in different ways for its constant excess in the blood.

How is Uric Acid formed?

It is one of the "purin bodies," which are oxidation products derived from the breaking down of cellular tissues (nucleins), and may therefore have two sources: (1) the cellular tissues of the body itself (endogenous purin bodies); (2) the cellular elements of the food (exogenous purin bodies). Of these the exogenous purin bodies may obviously be varied in amount by modification of the diet.

How is its Excess in the Blood accounted for?

In one of three ways:—increased formation, deficient elimination, or deficient destruction. There is no sufficient evidence for the first; on the second view it is supposed either that disease of the kidneys, so frequent in gout, interferes with elimination, or that the uric acid circulates in an abnormal combination which the kidneys cannot eliminate. On the third assumption it is maintained that the uric acid is not converted into urea in the normal way, owing to deficiency of the ferment (*oxidase*) by which it is destroyed in the liver.

Are there other Theories of Gout?

Yes; the occurrence of excess of uric acid in the blood in other conditions without gouty symptoms has led to further speculation. Thus Duckworth assumes that the disease has a nervous origin and that the changes in the joints are trophic, Ebstein, that local necrosis of the joints is due to altered nutrition of the tissues; and it is also held that the disease is dependent upon alterations in the digestive ferments, or

upon changes in the intestinal flora, causing a bacterial toxæmia.

What are the Morbid Changes?

In the affected joints there is, just below the surface of the cartilage, a deposition of acicular crystals of urate of soda, which causes a glistening white appearance. Beneath this the cartilage is more or less necrotic, and ultimately it may be eroded down to the bone. Urate of soda is also found in the synovial membranes and ligaments, in the structures around the joints, and in other situations, such as the lobe of the ear, when it forms subcutaneous chalky masses (*tophi*), which may ulcerate through the skin. In acute attacks the affected joint is much inflamed.

Chronic interstitial nephritis (granular kidney) is common, and is often associated with arteriosclerosis.

Describe an Acute Attack.

After certain premonitory symptoms—malaise, irritability, dyspepsia, scanty and high-coloured urine, etc.—which may last for a day or two, the patient is suddenly awakened in the early hours of the morning with severe pain, usually in the great toe. The pain increases and lasts for some hours, slight fever accompanies it, and the joint becomes red, hot, tender, and glazed. Swelling, at first moderate, later extends some distance from the joint, the skin at this stage having a more livid hue, and desquamating freely at the end of the attack. During the attack, which may last from five to twelve days, the pain is intermittent, and generally worse at night. At first uric acid is diminished in the urine, but later it is much increased, and returns to normal after the attack is over. Subsequent attacks may affect the same joint, or may extend to others, ultimately causing much deformity, especially of the fingers and toes (chronic gout).

What are the Changes of Chronic Gout?

The small joints of the fingers and toes are much swollen, stiff, and flexed or extended. They may be surrounded by subcutaneous tophi, which may ulcerate through the skin, exposing the chalky masses they contain. The larger joints may also be affected. Granular kidney and arteriosclerosis are frequent.

What are the other Forms of Gout?

(1) "Suppressed gout," in which internal symptoms appear as the acute joint affection improves, or after it has passed off. The condition may involve the heart, the gastro-intestinal system, or the cerebrum, and may end fatally. (2) Irregular gout, manifesting itself in gouty subjects who do not suffer from joint attacks by inflammatory affections of very varied character and seat, *e.g.* bronchitis, gastritis or enteritis, neuritis, conjunctivitis, phlebitis, eczema. Neuralgic affections such as migraine, cardialgia, and angina pectoris, are also common.

Mention the Complications.

Chronic renal disease, arteriosclerosis, cardiac hypertrophy, apoplexy, and inflammations of serous membranes.

How would you treat an Acute Attack?

The affected joint must be kept at rest and elevated, and should be wrapped in cotton-wool. Hot fomentations may be applied, or an anodyne liniment. The diet should be limited to milk and milk foods, and alcohol should be forbidden. After an initial mercurial purge, colchicum wine ℥xx-xxx should be given every four hours, along with the citrate or bicarbonate of potassium, until the pain is relieved. If it fails morphia may be necessary.

What is the Treatment of Chronic Gout?

It is mainly dietetic and hygienic. Foods rich in purin bodies, such as sweetbreads and liver, are to be forbidden; nitrogenous foods (meat) are to be cut down; and fish, milk foods, chicken, such vegetables as cabbage or lettuce, and fruit may be allowed. Abstinence from alcohol is better than moderation, but patients who will not abstain must use it sparingly, avoiding rich wines and beer. The bowels must be kept open. Among drugs guaiacum, potassium iodide, and sodium salicylate are recommended, and tonics are often of value.

ARTHRITIS DEFORMANS.

What is the Nature of this Disease?

It is a chronic inflammatory affection of the joints attended by changes in the synovial membrane and peri-articular tissues, and often by alterations in the bones. It has various forms, which occur at various ages. In childhood it may take the form of *Still's disease*, in which the swelling of the joints is accompanied by anæmia, glandular swelling, enlargement of the spleen, and fever; in early adult life (20-40) it appears as *rheumatoid arthritis*, in which the swelling is followed by much contraction and deformity; and in later life (40-60) *osteo-arthritis* is more common. In this there is little swelling, but much change in the cartilages and bones. It is still questionable whether these are to be regarded as distinct diseases.

What is the Etiology?

This is still uncertain. Cold and damp predispose to the disease, which is commoner in women than men. The ages of predilection have been mentioned above, and in women

the influence of the menopause must be noted. The chief theories of its origin are :—

1. That it is an infection. Various organisms have been found in the joints, but none specific ; and the disease sometimes begins acutely.

2. That it is an intoxication or chronic sapræmia, dependent upon the absorption of toxins from foci of local suppuration or inflammation (pyorrhœa alveolaris, uterine or ovarian disease, colitis, etc.).

3. That it is a tropho-neurosis. The joints are often symmetrically affected, and symmetrical neuritis and muscular atrophy may be present.

Describe the Morbid Changes.

The disease begins with inflammation and thickening of the synovial membranes, often accompanied by effusion, and with softening of the cartilages. The ligaments become thickened, and fibrous ankylosis may follow. The articular cartilage is eroded and at last destroyed ; the ends of the bones come into contact, and by friction become dense and highly polished (eburnated), while at the margins of the joints there are cartilaginous outgrowths which ultimately ossify, forming a rim or "lip" which may interfere with movement. Atrophy of the shaft of the bone may also be present.

In rheumatoid arthritis the soft tissues of the joints are principally affected, in osteo-arthritis the cartilages and bones.

Give an Account of the Symptoms.

The onset may be acute or more commonly gradual. In young women (acute rheumatoid arthritis), the disease sets in abruptly with fever and spindle-shaped enlargements of the joints, which are painful and tender. The finger-joints suffer first, and afterwards the wrists, ankles, knees, etc., and the vertebral and temporo-maxillary joints may be affected. When the attack has passed off, limitation of

movement remains, and is increased by subsequent attacks. Neuritis and muscular atrophy may occur.

In older people the onset is more gradual. The disease may attack only one joint, but generally several are involved, it may be unilaterally at first. Swelling and inflammation are less marked, and the chief signs are increasing stiffness and deformity. The shape of the hands is altered, the fingers being flexed at the metacarpo-phalangeal joints, extended at the first, and flexed at the second interphalangeal joints, and deviating towards the ulnar side of the hand. The joints creak on movement, and osteophytes may be felt at the sides of them. When these are confined to the fingers they are known as "Heberdens Nodes."

What is the Treatment?

This must be both local and general. In the acute cases rest in bed is necessary while fever lasts, and salicylate of soda, potassium iodide, or guaiacol carbonate may be given. Later, and in chronic cases from the outset, exercise and massage are advisable, with local blisters, applications of iodine, or hot-air baths. Tonics, especially arsenic, and good feeding are necessary, and all local discharges (pyorrhœa, leucorrhœa, etc.) must be treated. Vaccines prepared from the organisms of such discharges have been used, and sometimes with good effect.

MUSCULAR RHEUMATISM.

What is this Affection?

A painful condition of the voluntary muscles, dependent upon inflammatory changes in their fibrous tissues. It is known according to its seat as lumbago, pleurodynia, torticollis, etc.

What are its Causes?

Exposure to cold and damp and the gouty habit predispose; muscular overstrain is often an exciting cause.

What are the Symptoms?

In lumbago, pain across the small of the back, often intense, and worse on stooping or walking: in pleurodynia, a pain like that of pleurisy, with limitation of movement on the affected side, but without the physical signs of pleurisy; in torticollis, pain and stiffness over the sternomastoid muscle. Similar pains may occur in other situations.

How is it treated?

By rest and, if necessary, fixation, of the affected muscles (strapping), hot fomentations, anodyne liniments or plasters, and antirheumatic treatment. A saline purge should be given at the outset.

RICKETS.

(Rhachitis.)

Define this Disease.

A disorder of nutrition occurring in infants and young children, and characterised by softening of the bones.

What is its Etiology?

It occurs between the ages of six months and two years, and principally among the children of the poor, in whom improper feeding is combined with defective hygiene, want of sunlight, and defect of fresh air and exercise. The main cause is improper feeding, and it is therefore found in the children of unhealthy mothers, whose milk is not sufficiently nutritious, in children kept too long on the breast, and in

those who are brought up on artificial foods. In all these cases there is a deficiency of fat and proteid in the diet.

Describe the Morbid Changes.

These principally affect the bones, which are deficient in lime salts. The cartilaginous epiphyses are thickened, softened, and very vascular; and the line of ossification is irregular. Ossification from periosteum is imperfect, although the periosteum itself is thickened and proliferation of bone from it is active. The medullary cavity increases at the normal rate, and thus the shaft of the bone becomes softened and is liable to bend. The changes are most marked in the long bones, the ribs, and the skull.

Fibrotic changes occur in the spleen and lymphatic glands; the spleen is generally large, and the liver may be so. The musculature is flabby and wasted. Anæmia is sometimes, but not invariably, present.

What are the Symptoms of Rickets?

The aspect is characteristic; the face is small, and the skull, therefore, looks large; the forehead is square, and the head elongated; the fontanelles close late. The flat bones are thickened and bossy, but the occipital bone may be thinned. The ribs are "beaded" at their junction with the costal cartilages, and the soft bone yields to the atmospheric pressure, so that a shallow vertical groove forms on either side of the sternum (rickety chest). Pigeon-breast occurs when there is interference with inspiration. The epiphysial ends of the long bones (wrists, ankles, and knees), are enlarged and soft; and if the child has begun to walk, deformities of the limbs may occur (sickle-shaped tibia, etc.). Deformities of the pelvis are also common, and give rise in adult life to interference with labour.

The chief general symptoms are restlessness at night, nocturnal sweating of the head, and crying, due to general

tenderness, when the body is handled. The enlargement of the spleen and liver, coupled with flatulent distension, causes prominence of the abdomen, and bending outwards of the lower ribs, which a transverse depression (Harrison's groove), separates from the chest. There may be looseness of the bowels, with green and offensive motions, but this is not a necessary accompaniment of rickets. The musculature is soft, and the nervous system readily irritated; convulsions, laryngismus stridulus, and tetany are frequent complications. Respiratory affections are severe in rickety children, and may lead to a fatal issue.

What is the Treatment of Rickets?

Attention to diet and hygiene are the chief points. The child should be much in the fresh air. Where the maternal milk is of poor quality, a wet nurse should be employed, or cow's milk, diluted in proportion to the child's age, may be substituted. Starchy food must be avoided, and fat must be supplied (cream, cod-liver oil). Phosphorus and syrup of the phosphate or iodide of iron are also valuable remedies. Walking must not be allowed while the bones are soft, and splints extending beyond the feet may be used to prevent it.

BERI-BERI.

What is this Disease?

A nutritional disorder most common in Oriental countries, and characterised by the presence of dropsy and paralysis due to a multiple neuritis.

What are its Causes?

The disease is most common in Japan, the Malay Archipelago, and Burma, but it is not rare in Lascars who form

the crews of steamships plying from the East to this country. It attacks males more often than females, and is favoured by overcrowding.

It was for long supposed to be of bacterial origin, but it has recently been shown to depend upon the method of treating the rice, which is the chief food of the inhabitants of the countries referred to, Those who live upon whole rice do not develop beri-beri, those who use "polished" rice, from which the pericarp is removed by milling, are frequent sufferers. The pericarp contains an antineuritic substance allied to allantoin and named "vitamine" by Funk. It is found in association with phosphorus, the absence of which from the food indicates the absence of vitamine, to which the symptoms are apparently due.

What are the Morbid Changes?

Those of neuritis, which may affect not only the peripheral nerves, but also the phrenic and vagus. Muscular wasting, œdema, dropsy of the serous cavities, and wide-spread ecchymoses may also be present.

Describe the Symptoms.

In the *atrophic* or *dry* form of the disease weakness and pains in the limbs are succeeded by wasting, paralysis, loss of knee-jerks, foot-drop, and anæsthesia or hyperæsthesia. In the *dropsical* or *wet* form there is little wasting or loss of power, but there is marked œdema without albuminuria, and there is also fluid in the body cavities. In the *rudimentary* form paresis and paræsthesia replace paralysis and anæsthesia. Recovery is the rule, after weeks or months of illness; but in the *cardiac* form, which is characterised by dilatation of the heart, death from cardiac failure is the usual end.

What is the Treatment?

Changing the defective diet is the chief measure. The heart must be supported by strychnine and digitalis, and, in acute heart failure, purgatives or bleeding are required. The paralysis may be treated by massage and later by electricity, and dropsy may be relieved by diuretics or if necessary by tapping.

DIABETES MELLITUS.

Define this Disease.

A nutritional disorder characterised by the persistent passage of excessive quantities of urine containing sugar, and attended by progressive emaciation. A temporary glycosuria does not constitute diabetes.

In what Circumstances does it occur?

It is more common in males than females, and usually occurs about middle life. Old people less commonly suffer, and it is rare in childhood; but the younger the subject, the more acute is the disease. Of the different races the Jews are especially liable. The disease is sometimes hereditary. It frequently occurs in those of nervous temperament, and it may be associated with definite lesions of the nervous system. In certain instances the pancreas, in others the liver, thyroid, or suprarenal glands have been found diseased. Obesity is sometimes associated with it, and it may follow one of the specific infections or occur along with gout.

What is the Pathology of Diabetes?

Sugar appears in the urine when an excess of it is present in the blood, which normally contains about 0·05 to 0·1 per cent. of it. It is held that the carbohydrates of the food,

being converted in the intestine into sugar, are transformed by the liver into glycogen, and stored there. As sugar is required for the purposes of nutrition, the liver, by means of ferments, retransforms its glycogen into sugar, and passes it on to the blood and thus to the tissues, in which it is used up. Excess of sugar in the blood, and hence in the urine, might therefore be due to an excessive supply of carbohydrates, to defect in the hepatic function, or to defective oxidation in the muscles or respiratory system. A supply of carbohydrates beyond the assimilation limit will produce a temporary glycosuria even in the healthy, and in mild cases of diabetes, abstraction of carbohydrates from the food stops the glycosuria. In severe cases, however, the glycosuria continues even in the absence of carbohydrates from the diet, and in such instances the sugar is formed by the splitting up of proteids, and possibly also of fat. The proteid may be derived from the food, or, in the worst instances, from the body proteids.

It is not yet settled how the glycogenic function of the liver is regulated, but it appears that it is to a large extent under nervous control, and also that it is influenced by the secretions of the ductless glands. Hyperglycæmia, for example, may be produced by puncture of the floor of the fourth ventricle (the "diabetic puncture"), or by stimulation of the splanchnic or hepatic nerves, but this is only possible in the presence of adrenalin in the blood. The pancreas also has a marked effect upon glycogenesis, probably through an internal secretion which may be derived from the islets of Langerhans, and the parathyroids and pituitary body share in the control. There appears to be a balancing action between the secretion of the adrenals on the one hand and the pancreas on the other; but it is impossible as yet to say what is the precise nature of their action.

It is evident that in different cases different organs may be at fault in the production of diabetes.

What are the Morbid Changes?

No gross lesion may be discoverable. In some cases the *pancreas* is the seat of fibrotic or atrophic changes, or, it may, be of other diseases; the *liver* may be fatty or sclerosed (diabetic cirrhosis); the *kidneys* may be affected by interstitial nephritis; and occasionally lesions are found in the central nervous system. The *blood* may appear natural or unduly thick; its alkalinity is lessened; it sometimes contains fat, which, on standing, forms a creamy layer on the surface; and it always contains an excess of sugar.

Describe the Symptoms of a Typical Case.

The disease begins gradually, with increasing thirst, and sometimes increasing hunger, dryness of the skin, loss of flesh and weight, muscular weakness, and the passage of large quantities of pale urine. The tongue and mouth are dry, and the bowels costive. The urine, though pale, is of high specific gravity (1025 to 1045 or more); the quantity passed may be as much as ten or even twenty pints a day, and it contains sugar, often in large amount. In the late stages albuminuria may also be present, and, in severe cases, acetone, diacetic acid, and β oxybutyric acid are usually to be found. These bodies may be derived either from the breaking down of proteid, or from faulty oxidation of the fats.

What is the Course of the Disease?

It may be extremely acute, not yielding to treatment, and causing death in a few weeks or months. If the case is mild, treatment may remove the sugar from the urine, and it may remain absent for a considerable period. Relapses, however, occur, and coma or complications ultimately lead to death. The diabetes of young people is acute, that of the elderly may be very chronic.

Mention the Chief Complications.

1. *Skin* — Boils, carbuncles, pruritus (especially of the vulva in women), eczema, gangrene.

2. *Eyes*—Cataract, retinitis, optic atrophy. The first is common.

3. *Nervous System*—Peripheral neuritis (absent knee-jerks, cramps in the legs, neuralgia); trophic changes, such as perforating ulcer of the foot and brittleness of the nails. Coma is rather the natural end than a complication.

4. *Kidneys*—Albuminuria, later interstitial nephritis.

5. *Lungs*—Phthisis, pneumonia.

What is Diabetic Coma?

A condition which arises towards the end of severe cases of diabetes. It may set in suddenly or gradually, and its onset is favoured by strong emotion, excitement, or over-exertion. The condition is at first one of drowsiness, with subnormal temperatures, and prolonged, deep, and sighing respiration ("air-hunger"). The odour of the breath and urine is like that of apples, and the urine contains acetone, diacetic acid, and β oxybutyric acid. The coma deepens, and within a few days leads to death, which is not usually preceded by convulsions.

The coma is due to the "acidosis" produced by the presence of β oxybutyric acid. When this substance is present in the blood it forms a combination with the carbonate of soda which prevents its union with carbon dioxide to form the bicarbonate. It is as the bicarbonate that the carbon dioxide is normally carried to the lungs, where it is split off and exhaled. When this is prevented it accumulates in the blood and tissues, and so produces coma.

What is the Treatment of Diabetes?

Attention to the general health, avoidance of worry, daily gentle exercise, and precautions against chill are required in every case. The daily amount of sugar excreted must be ascertained, and when this is known, the patient is put upon a diet from which all starches and sugars are gradually removed, until the sugar disappears from the urine, or until a starch-and-sugar-free diet is reached. When the glycosuria has ceased, carbohydrates are once more carefully added to the food until the assimilation limit is ascertained, and the amount of carbohydrate allowed is then kept just below this limit. If the glycosuria does not entirely cease, its lowest point must be noted, and as much carbohydrate may be allowed as does not raise the excretion beyond this point. A diet exclusively nitrogenous, if long persevered in, is apt to lead to acidosis, and many patients are better in general health when a little carbohydrate is permitted.

From a strict diabetic diet the following articles are excluded: — All saccharine or farinaceous foods, *bread*, potatoes, rice, tapioca, sago, arrowroot, macaroni; turnips, carrots, parsnips, beans, and peas; liver (on account of its glycogen) and shell-fish with large livers (oysters, cockles, mussels); sweet fruits and sweet wines. The following are permitted:—Butcher's meat of all kinds (except liver), poultry, game, and fish; all green vegetables; fats (butter, cream, etc.) and oils; eggs in any form; meat soups; jellies made without sugar; dry wines or spirits; tea or coffee without sugar; mineral waters (not sweetened); and milk in very small quantity. Saxin may be given instead of sugar.

Opium and its alkaloids are the drugs which have most effect in controlling the output of sugar; of these codeia is most used, and may be given in doses increasing from gr. $\frac{1}{2}$ to as much as gr. x thrice daily. Alkalies are useful in acidosis, and should be given in large doses when diacetic acid is present. If coma appears, sodium bicarbonate ($\bar{3}$ i

in a pint and a half of water) may be injected subcutaneously or intravenously in divided doses, or sodium carbonate of the same strength, intravenously but not subcutaneously. Stimulants should also be given.

Many other drugs have been recommended, but their success is limited. Tonics such as arsenic and strychnine are beneficial to the general health; and complications must be treated as they arise.

DIABETES INSIPIDUS.

What are the Main Features of this Disease?

It is a condition characterised by a permanent polyuria, the urine containing neither sugar nor albumin, and accompanied by excessive thirst. Its etiology is obscure, but it is more frequent in the young than in later life, and in males than in females. It is sometimes hereditary, and has followed mental shock or blows upon the head. It is therefore probably of nervous origin.

There are no constant morbid changes, though hypertrophy of the bladder and dilatation of the ureters may be found. The symptoms are deducible from the definition. The urine is pale, clear, of very low specific gravity, and excessive in quantity, but not otherwise abnormal. Thirst is great, and there may be some emaciation and weakness, with insomnia. The disease is seldom fatal unless it is due to injury to the head, but death may follow intercurrent diseases. The onset is usually gradual, but the condition is persistent, and treatment is of little avail. Valerianates, strychnine, or galvanism to the cord may be tried, and where there is a history of syphilis, specific treatment.

II. CHRONIC INTOXICATIONS AND SUNSTROKE.

ALCOHOLISM.

What Causes contribute to the Development of the Alcoholic Habit?

Hereditary influences, and the influences of occupation. A nervous heredity is often apparent, and the child of an alcoholic father frequently also becomes alcoholic. Those engaged in the liquor trade are particularly liable; and unusually hot occupations, by producing thirst, predispose to the habit.

What are the Morbid Changes of Chronic Alcoholism?

Chronic catarrhal affections of the gastro-intestinal tract; fatty degeneration or cirrhosis of the liver, the latter especially in spirit drinkers; arteriosclerosis, and interstitial nephritis consecutive to it; and affections of the nervous system (*peripheral neuritis*, pachymeningitis, etc.).

What are its Symptoms?

Digestive disturbances such as morning sickness or vomiting, furred tongue, anorexia, and constipation or catarrh of the bowel; tremor of the hands, with partial paralysis of hands and feet, perverted sensations, loss of knee-jerks, and ultimately all the symptoms of peripheral neuritis; insomnia, loss of memory, defective moral sense, and untrustworthiness. Some cases end in dementia, others in organic disease of the liver, kidney, or heart (myocardium), or meningitis.

What is Delirium Tremens?

A condition common in habitual drinkers, following injury, shock, or exposure, or complicating acute disease. It sets in with insomnia, restlessness, and muscular *tremor*. At first the subject talks or mutters to himself, and this is followed by hallucinations of vision, usually in the shape of moving animals (rats, etc.) and by noisy delirium, during which he attempts to spring out of bed, and requires careful watching. There is slight or moderate fever, with a rapid soft pulse and free perspiration. Death is due to exhaustion, or the case may end in coma or convulsions. In cases that recover, sleep usually returns about the third day, but insomnia may persist until the fifth.

How is the Coma of Acute Alcoholism to be distinguished from other Forms of Coma?

In *uræmia* the coma usually follows upon convulsions, the pulse is slow, there is often evidence of arteriosclerosis and hypertrophy of the left ventricle, the urine contains albumin, the pupils may be dilated or contracted, and are sometimes unequal.

In *cerebral hæmorrhage*, along with the above signs, the respirations are stertorous, the sphincters may be relaxed, and there is evidence of unilateral paralysis. There is "conjugate deviation" of the eyes.

In *opium poisoning* the pupils are contracted to the size of "pinheads," the face is reddened or cyanosed, the pulse is slow, and the respiration very slow and deep. The patient, in the early stages, can usually be roused.

In *alcoholic coma* the face is at first congested, then pale; the eye reflexes are lost and the pupils usually dilated; the pulse is at first quick and strong, but becomes feeble; the skin, at first moist and warm, becomes afterwards cold. The coma is often profound, but the sphincters are not relaxed.

What is the Treatment of Alcoholism?

In alcoholic *coma*, wash out the stomach, and give hot coffee by enema or by the stomach tube.

In *delirium tremens*, after evacuation of the bowels by a mercurial purge, diaphoresis should be promoted by Liq. Ammon. Acetat., the patient must be fed frequently with small quantities of milk, and he must be prevented from injuring himself or others. The strait-jacket should, if possible, be avoided. Narcotics are often necessary, but must not be too frequently repeated. Bromide of potassium, chloral gr. xv-xx, or in bad cases hyoscine gr. $\frac{1}{120}$ may be given every six hours, or morphia, though the last is apt to be dangerous from its cumulative effect. Alcohol must be at once withdrawn, and in the ensuing prostration other stimulants (ammonia, etc.) must take its place.

In *chronic alcoholism* the treatment is largely psychical, and consists in aiding by appropriate suggestion any efforts that the patient may make to overcome his craving. He must stop alcohol at once, and must not be allowed to attempt a gradual reduction. Tonics may be given to meet the depression, nux vomica or quinine being among the most useful, and sleeplessness may require narcotics, though the danger of forming a drug-habit must be recognised. Institutional treatment is often needed at the outset, and he who has once been an alcoholic should become an entire abstainer for the rest of his life.

LEAD POISONING (Plumbism).

How is Chronic Lead-Poisoning induced?

The metal or its salts may be inhaled, swallowed, or absorbed by the skin. Chronic poisoning may thus follow upon inhalation of the fumes or dust, and is common among white-lead workers and smelters, painters and potters; it may

be due to swallowing water or tinned foods contaminated by lead; it may occur among workers in lead who eat with unwashed hands (compositors, painters); or it may result from the use of hair-dyes containing lead.

What Morbid Changes does it cause?

In the nervous system, peripheral neuritis, and sometimes meningo-encephalitis; in the kidneys, interstitial nephritis (granular kidney); in the vascular system, arteriosclerosis and cardiac hypertrophy; and in the blood, anæmia.

What are the Usual Symptoms?

They begin with digestive derangements (loss of appetite, constipation, headache), with which anæmia is soon associated. The complexion is pale and sallow, and a blue line develops on the margins of the gums, sulphide of lead being formed in this situation by contact with the sulphur contained in the tartar of the teeth. The line is therefore absent where teeth are missing. Attacks of intense abdominal pain (*lead colic*) are usually the next symptom. The pain, which may be preceded by vomiting, occurs in paroxysms, is seated in the umbilical region, which is often tender, and is accompanied by retraction of the abdominal wall. It is sometimes relieved by pressure. In the intervals between the paroxysms a dull, deep-seated pain persists. An attack of lead colic passes off in a few days, but during its continuance the pulse is slow and hard, there is obstinate constipation, and there may be vomiting. It is probably due to spasm of the small intestine, and may recur frequently.

What is Lead Palsy?

This is due to the neuritis produced by lead. It usually first attacks the upper limbs, and particularly the extensor muscles of the hands, so that the patient suffers from

bilateral "wrist-drop," and is unable to raise the hands from the position of extreme flexion. The paralysis is due to implication of the musculo-spiral nerve, but it does not affect the supinator longus or extensor carpi pollicis, which are innervated by the same nerve. The paralysed muscles show the reaction of degeneration.

The paralysis may extend to the legs (peroneal muscles), causing "foot-drop," and in some cases may begin there. It may also spread to the muscles of the upper arm. The affected muscles rapidly atrophy; but sensory disturbances are not common, and if they are present are usually slight.

Mention any other Symptoms of Lead-Poisoning.

Marked anæmia, many of the red cells containing basophil granules (*punctate basophilia*); albuminuria, due to the interstitial nephritis; the vascular changes referred to above; menorrhagia, amenorrhœa, or miscarriages; optic neuritis or atrophy; and sometimes lead encephalopathy. This last is a cerebral disturbance beginning with headache, leading to convulsions or coma, and often ending fatally in a few days.

How are such Cases to be treated?

The pains and constipation of lead colic are to be met by warmth to the abdomen, warm baths, and aperients; in some cases morphia is necessary. Elimination of the lead must be promoted by small doses of iodide of potassium (gr. v) in combination with saline aperients; larger doses of the iodide may throw a dangerous quantity of soluble iodide of lead into the circulation. Galvanism and massage are required for the paralysis, iron for the anæmia, and lead encephalopathy should be treated by nitrite of amyl inhalations, pilocarpine, and, if necessary, lumbar punctures. After recovery the causative occupation should be abandoned; and workers in lead or substances containing it should never eat with unwashed hands.

SUNSTROKE.

What Forms of Illness are due to Exposure to Heat?

They are three: heat-exhaustion, heat-apoplexy or sunstroke proper, and siriasis or thermic fever.

On what Causes do they depend?

They may be produced either by direct exposure to the rays of the sun or by the effects of artificial heat. The internal temperature rises in consequence of the action of excessive heat upon the heat-regulating centre in the medulla, as a result of which it may be that an autogenous toxæmia arises, as is suggested by some. Sambon regards siriasis, which occurs chiefly in tropical countries, as an infection due to an organism which, outside the body, can develop only at high temperatures. Contributory causes are fatigue, alcoholism, moisture of the atmosphere, and work in confined spaces.

The inconstant morbid changes are chiefly those of cerebral engorgement.

What are the Symptoms?

Heat-exhaustion, or the *syncopal* form, is attended by faintness or syncope, coldness and moisture of the skin, nausea or vomiting, dilatation of the pupils, and a rapid, feeble pulse. Recovery is the rule, but heart failure may be the cause of death.

Heat-apoplexy (the *asphyxial* form, sunstroke proper) is of sudden onset, and is due to direct exposure to the rays of the sun. The symptoms are unconsciousness, pallor, and failure of the heart *and respiration.* Death frequently occurs, and after recovery there may be permanent injury to the brain.

Thermic fever or *siriasis* may be of gradual onset, and may follow exposure to other than solar heat. Malaise, nausea, diarrhœa, thirst, giddiness, or dyspnœa may be the premonitory symptoms, which are followed by a sudden rise of temperature to 108° to 110° F. or more. The face becomes livid, the pulse rapid and feeble, the pupils contracted, and delirium is followed by coma and death.

How is Sunstroke to be treated?

The syncopal form by removal to a cool place, loosening the clothing, a brief use of the cold douche, and stimulation if the pulse indicates it. Removal to a cool place is necessary in all forms. Both in the asphyxial form and in thermic fever the head and body must be freely douched with cold water. In thermic fever cold baths may be needed, or the skin may be rubbed with ice until the temperature has fallen to 104° F., while blisters to the scalp may be tried if unconsciousness persists.

After recovery, life in the tropics should be abandoned.

II.—DISEASES OF THE KIDNEYS.

Before describing the inflammatory affections of the kidneys, it is desirable briefly to discuss one or two of the principal symptoms which may occur in all their forms.

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ALBUMINURIA.

What is Albuminuria?

The presence in the urine of serum-albumin and serum-globulin.

In what Circumstances does it appear?

Albumin is found in the urine in many forms of renal disease, especially congestion, active or passive, inflammation, acute and chronic, degenerations, such as amyloid disease, renal calculus, tuberculosis, etc. It may also occur in morbid conditions of the blood (anæmia, leukæmia, etc.); in fevers, when it may be either a febrile albuminuria or due to a complicating nephritis; in pregnancy; in poisoning by cantharides, turpentine, arsenic, and certain other drugs; in certain nervous diseases (apoplexy, concussion, epilepsy); and occasionally in healthy adolescents after exertion or mental overstrain (functional albuminuria). In all these cases the source of the albuminuria is in the kidney. It may also arise from disease of the urinary passages (pelvis, ureter, bladder, urethra), below the kidney, and is thus found in pyelitis, tuberculosis of the ureter, cystitis, and gonorrhœa.

How is Renal Albuminuria produced?

Two explanations are offered. That which is generally accepted and has much support in experimental evidence, is that it depends upon morbid alterations in the epithelial

cells of the glomeruli, which normally prevent the passage of albumin. Others hold that albumin may also escape by direct transudation into the renal tubules under conditions of increased blood-pressure.

What are the Usual Tests for Albumin?

1. Heat. When the upper part of an albuminous urine contained in a test-tube is boiled, a coagulum forms which varies in density with the amount of albumin present from a mere haze to a thick curd. The unboiled portion of the urine remains clear, and the slightest haze is perceptible by contrast. Turbid urines must first be cleared by filtration; gentle heat dissolves a turbidity due to urates. Phosphates may also be precipitated on boiling if the urine is neutral or feebly acid; the addition of a drop or two of acetic acid redissolves the precipitate, but does not dissolve an albuminous coagulum. If the urine is strongly alkaline, a drop or two of acetic acid should be added before boiling.

2. Nitric acid (Heller's test). A layer of nitric acid is put in the bottom of a test-tube, which is then inclined, and the suspected urine is allowed to flow slowly down the inside of the tube so as to form a layer on the surface of the acid. A ring of albumin, not soluble by heating, forms at the line of junction of the fluids. Urates and mucin form a haze *above* the line of junction, and are re-dissolved by heat. A precipitate also forms in urines containing copaiba, but heat re-dissolves it; and in concentrated urines a *crystalline* deposit of nitrate of urea may appear after some time. Albumoses are also precipitated, but re-dissolve upon heating.

3. Picric acid. When a saturated solution of picric acid is floated on the top of the urine, a ring of albumin forms at the line of junction, and is not dissolved by heat. Urates, alkaloids, and albumoses are also precipitated, but these precipitates are dissolved by heat.

4. A saturated solution of *salicyl-sulphonic acid* may be used in the same way as picric acid.

How is the Quantity of Albumin estimated?

For clinical purposes, Esbach's albuminimeter is used. This is a graduated tube into which urine is poured up to the mark V, and a watery solution containing 1 per cent. of picric acid and two per cent. of citric acid up to the mark R. The fluids are mixed by inverting the tube, which is then left to stand for twenty-four hours. The amount of precipitated albumin, which has settled to the bottom, is then read off upon the graduated lines which mark the tube, these lines representing grammes per litre of urine.

What is the Significance of Renal Albuminuria?

In the majority of instances it is indicative of disease of the kidney, and usually of either a congestive or an inflammatory state. In many cases, therefore, it signifies an incurable condition, and gravely affects the estimate of the probable duration of life. It is, however, often transitory (albuminuria of fevers, anæmia, poisoning, etc.), and in some cases (functional albuminuria of adolescents) it is apparently consistent with undisturbed health. This last form must be diagnosed only in the entire absence of renal or other symptoms, and after repeated examinations. It must also be recollected that in some forms of grave renal disease (chronic interstitial nephritis) albuminuria may be temporarily absent.

URAEMIA.

What is Uræmia?

A toxæmic condition frequently associated with Bright's disease in all its forms, and characterised by disturbances mainly of the central nervous system.

What are its Forms?

It may be acute, chronic, or latent.

How is Uræmia produced?

There is as yet no unanimity of view upon this subject, although it is evident that the defective elimination of urea and other urinary constituents by the damaged kidneys suggests that the toxic symptoms are due to retention of these products. But no single urinary constituent—urea, ammonia, or other ingredient, has been found to be solely responsible, and many regard uræmia as due to the combined effect of several of these. Others look upon it as produced by absence or defect of an internal secretion of the kidneys; and it has been recently suggested that it may be due to absorption of the decomposition-products of dead or dying renal cells.

Describe an Acute Uræmic Attack.

The seizure is usually convulsive. The convulsions, which may come on without warning or be preceded by headache or sickness, resemble those of epilepsy, being bilateral, and having a brief tonic stage, followed by clonic spasms which affect all the muscles of the body. The face is livid, the tongue may be bitten by the clenched teeth, the pupils are dilated, and unconsciousness is complete. The convulsion is succeeded by coma, which may be followed by a further convulsion, to be again followed by coma. In other instances there may be only a single fit. Recovery is common, but death may end even a first attack.

Are there any Deviations from the usual Type?

The convulsions may be unilateral, or coma may occur without convulsions. In some instances they are followed by temporary paralysis (apoplectiform type); in others they

may be replaced or succeeded by delirium or mania. Sudden blindness (*amaurosis*) without retinal lesions may follow them, or may occur independently, to pass off within a few days.

What are the symptoms of Chronic Uræmia?

They affect the nervous and digestive systems. Persistent headache is one of the most common. Vertigo, drowsiness, or stupor, and muscular twitchings also occur. Attacks of dyspnœa, sometimes asthmatic in character, are not uncommon, and in grave cases Cheyne-Stokes breathing may be present, and may precede death by weeks or even months. Itchiness of the skin (pruritus) is frequent. The digestive symptoms consist in nausea or vomiting, and diarrhœa, often intractable, and sometimes associated with chronic intestinal ulceration. Hiccough may also occur.

What is meant by Latent Uræmia?

A condition occurring in obstructive suppression of urine, as when both ureters are blocked by renal calculi. The suppression is accompanied by sleeplessness and lassitude, which last for about a week, and are followed by muscular twitchings, dyspnœa, and contraction of the pupils, without coma. The condition ends fatally a day or two after the onset of muscular twitchings.

How is Uræmia to be distinguished from other Comatose Conditions?

Uræmic coma is most liable to be confused with the coma due to apoplexy, alcohol, or opium poisoning. To avoid error, the urine must be drawn off by catheter and tested for albumin. The presence of albuminuria, accompanied by casts, speaks for renal disease, of which further evidence may be found in arterial changes and a hyper-

trophied left ventricle. In uræmia the pupils may vary in size, but are generally equal; in cerebral hæmorrhage they are usually unequal; in opium poisoning they are strongly contracted, and in alcoholic coma they are usually dilated. Cerebral hæmorrhage is frequently accompanied by conjugate deviation of the eyes, and one or other side is either completely flaccid or unduly rigid. It should be remembered that evidence of renal disease does not exclude cerebral hæmorrhage, which is a frequent complication of chronic nephritis. A smell of alcohol in the breath is no indication that a patient is suffering from alcoholic coma; he may have taken stimulant just before an attack of apoplexy.

For *Treatment* see Acute Nephritis.

BRIGHT'S DISEASE—NEPHRITIS.

What do you understand by Bright's Disease?

A non-suppurative inflammation of the kidneys.

What are its Varieties?

Non-suppurative nephritis may be acute or chronic. It may affect one or other of the anatomical elements of the kidney—the tubules, the glomeruli, or the interstitial tissue—and is therefore further divided into tubular or parenchymatous nephritis, glomerulo-nephritis, and interstitial nephritis. But it is to be remembered that in no case is the inflammation confined to one tissue only: the others, to some extent, share in it. In even the most acute tubular or glomerular nephritis there is some degree of interstitial inflammation, and in even the most chronic interstitial nephritis the tubules do not entirely escape. The distinguishing epithet refers, therefore, to the tissue predominantly, but not exclusively, affected, the inflammatory changes being diffused throughout the substance of the kidney.

Acute nephritis may principally affect the tubules, glomeruli, or interstitial tissue; chronic nephritis, which may follow the acute disease or arise independently, in one of its forms attacks chiefly the tubules (*large white kidney*), in the others chiefly the interstitial tissue.

ACUTE NEPHRITIS.

What are the Causes of this Disease?

Acute nephritis affects both sexes, but is rather more frequent in males, and it is more common before the age of 40 than after it. Its chief causes are:—exposure to cold; the organisms or toxins of the specific fevers, particularly scarlet fever, small-pox, and diphtheria; chemical poisons or irritating drugs (cantharides, turpentine, carbolic acid, etc.); septic absorption from extensive burns; and pregnancy.

Describe the Morbid Changes in the Kidney.

In tubular or parenchymatous nephritis of the catarrhal type the kidneys are enlarged, the capsule stripping easily, and the surface appearing pale or red. The cortex is red and wider than normal, the pyramids deeply congested; in extreme cases blood may drip from the cut surface. The Malpighian bodies are also injected.

The tubular epithelium is swollen and cloudy, its cells proliferate, and they are shed into the lumen of the tubules, which may be partly blocked by such granular or fatty cells and by blood corpuscles from the engorged vessels. Structureless transparent cylinders of an albuminoid character (hyaline "casts") fill up many of the tubules, of which they retain the shape. Fragments of these, carrying upon them the shed epithelium or corpuscles, appear in the urine, and are known as epithelial casts, blood casts, etc. The albuminoid material is probably exuded by the glomeruli.

The interstitial tissue is infiltrated by inflammatory exudation and also by round cells.

What is Glomerulo-Nephritis?

A form of acute nephritis in which effusion takes place into the glomeruli, the vessels of which may burst, causing hæmorrhage, while the capsular epithelium proliferates, and may later obliterate the tufts. The tubules and interstitial tissue also suffer, though less severely. The affection is most common in scarlatina, but is hardly to be classed as a distinct variety.

What are the Symptoms of Acute Nephritis?

The disease, as a rule, begins abruptly, either after exposure or in the course of a specific fever. A sense of chilliness, or even rigor, with some pain in the back and headache, and sometimes vomiting, are the first symptoms. There is sometimes slight fever at the outset. Œdema appears early, and affects first the feet and face, from which it may spread all over the body, and in severe cases may attack the serous cavities (ascites, hydrothorax). The urine is passed frequently, but in small quantity, and the amount passed in twenty-four hours is much less than normal. In very acute cases it may be temporarily suppressed. It is turbid, reddish-brown, or chocolate-coloured from admixture with blood, or when the blood is less abundant, "smoky" in appearance; it is of high specific gravity (1025-1030); it contains albumin in large quantity; and under the microscope it shows blood-casts, epithelial casts, blood corpuscles, and degenerated renal epithelium. Urea, phosphates, and chlorides are all much reduced in quantity.

The pulse is hard and not easily compressible, the blood-pressure is raised, and the second aortic sound may therefore

be accentuated. Dilatation of the heart may follow in severe cases. Anæmia is pronounced.

What is the Course of such a Case?

In favourable conditions, recovery may occur in a few weeks, the dropsy disappearing, and the urine increasing in amount and becoming normal. Much more often, although general symptoms may almost disappear, there is some permanent damage to the kidney; albuminuria persists, and chronic nephritis is in time established. Other cases end fatally in the acute stage, from uræmia or other complications.

What are the Chief Complications?

Uræmia: inflammation of the serous sacs (pericarditis, pleurisy, peritonitis), or of internal organs (pneumonia); œdema of the glottis or lungs; dilatation of the heart.

Are there any Difficulties in Diagnosis?

Rarely; the combination of dropsy with a highly albuminous urine containing blood and casts is distinctive. But when acute nephritis complicates a specific infection, dropsy may be slight or absent, and the diagnosis must then be made chiefly from the urinary characters. Scarlatina is an exception to this rule, dropsy being usually prominent in scarlatinal nephritis.

What is the Treatment?

Mild cases of acute nephritis are best treated by rest in bed, avoidance of nitrogenous foods with the exception of milk, diaphoretic measures, and occasional purgation. The diet should be of milk only, or of milk and soda-water; while thirst may be quenched by Potus Imperialis. Diaphoresis may be promoted by Liq. Ammon. Acetat.,

and as purgatives either Magnes. Sulph. or Pulv. Jalapæ Co. may be used. If the urine is much diminished or contains much blood, indicating extreme engorgement of the kidney, diuretics are not advisable. Before they are used, the congestion should be relieved by fomentations or dry cupping over the loins. If uræmia threatens, the patient should be freely purged, and sweating should be promoted by the hypodermic injection of pilocarpine gr. $\frac{1}{6} - \frac{1}{4}$. The same measures are applicable to excessive dropsy, and hot packs or hot air baths may be used in addition. Fluid may be removed from the legs by Southey's tubes.

In uræmic convulsions or coma, pilocarpine, jalap, and hot packs are also employed. If the convulsions do not yield, chloroform may be used, and in robust patients venesection up to $\bar{3}$ x is of much service.

The anæmia calls for the use of iron during convalescence.

CHRONIC BRIGHT'S DISEASE.

In what Forms may Chronic Bright's Disease occur?

There are two main forms, chronic parenchymatous (or tubal) nephritis, and chronic interstitial nephritis. Chronic parenchymatous nephritis most commonly presents the anatomical changes of "large white kidney," in which the tubules are chiefly affected, but in other instances ("small white kidney," secondary contracted kidney), the interstitial tissue is largely implicated, and by its contraction the size of the organ is reduced. This condition may follow upon large white kidney or arise independently of it.

Chronic interstitial nephritis ("small red kidney") is a chronic affection from the outset, and is found in middle-aged and elderly people, while chronic parenchymatous nephritis is often preceded by the acute disease, and is more common at an earlier age.

What Symptoms are common to the various Forms of Chronic Nephritis?

Anæmia; emaciation; cardio-vascular changes; a tendency to uræmia; and diminution of the daily excretion of urea and salts. The other urinary changes vary with the form of nephritis.

Describe the Cardio-vascular Changes.

They consist in arteriosclerosis (arterio-capillary fibrosis) and hypertrophy of the left ventricle. The blood-pressure is considerably and permanently raised, the pulse can be obliterated only with difficulty by digital compression, and the sphygmograph shows a high tidal wave and a poorly-marked dicrotic wave (pulse of high tension).

The arteriosclerosis affects chiefly the middle coat of the vessels, and particularly the smaller arteries, and hypertrophy of the media is the result. The intima is also thickened, and the changes extend to the vessels of the kidneys. In chronic tubular nephritis the arteriosclerosis is secondary to the renal condition; in chronic interstitial nephritis it may be primary.

The cardiac hypertrophy, which affects principally, but not exclusively, the left ventricle, may be consecutive to the arterial changes, or may be produced by the same causes which are responsible for these (circulation of impurities in the blood due to defective renal elimination).

Are there any Characteristic Ocular Changes?

In the later stages of chronic tubular nephritis, and still more commonly in chronic interstitial nephritis, *albuminuric retinitis* is liable to occur. In this condition ophthalmoscopic examination shows œdema of the retina, with white glistening spots or patches most numerous in the neighbourhood of the macula, and due to fatty change in the

nerve fibres and in Müller's fibres; hæmorrhages, often radially arranged and flame-shaped; and optic neuritis, the disc being swollen, with blurred edges, its arteries narrowed and often partially obliterated, and its veins large and tortuous.

Albuminuric retinitis does not occur in connection with acute nephritis, unless the acute attack be an aggravation of a previously existing chronic condition; nor is it often found in the stage of "large white kidney." Its presence is a grave prognostic indication.

CHRONIC PARENCHYMATOUS NEPHRITIS.

(Chronic Tubular Nephritis).

What are the Causes of Chronic Parenchymatous Nephritis?

It is frequently the sequel to an acute attack; but it sometimes arises independently of pre-existing acute disease. It may then follow insidiously upon acute infections, or occur in the course of tuberculosis, malaria, syphilis, or diabetes. Its onset is favoured by the prolonged influence of cold and damp.

Describe the Morbid Changes.

There are two forms of chronic parenchymatous nephritis. In the first (large white kidney), the kidneys are large, pale, and smooth on the surface, and the capsule strips easily. On section, the cortex is seen to be broad, and of a yellowish or mottled appearance, while the pyramids are injected, and in some instances there is hæmorrhage into the tubules. In the second (small white kidney or pale granular kidney), the organs are slightly or considerably smaller than normal, whitish or yellowish on the surface,

which is granular, and the capsule is adherent. The cortex is narrow and pale, and microscopically the changes are seen to be largely interstitial, the new-formed connective tissue causing by its contraction the diminished size of the kidney. The tubules are also affected, some being atrophied, some cystic, and others showing degenerated epithelium. The glomeruli are atrophied and the arteries thickened.

How are these two Forms related to each other?

This is still a subject of controversy. It is held on the one hand that both of them develop out of an acute nephritis, the large white kidney representing the first stage in the direction of chronicity, and the small white kidney being consecutive to it. On this view small white kidney presupposes an antecedent stage of large white kidney, the symptoms of which, however, are often absent, as a matter of clinical experience. A modification of this view represents the two conditions, while both arising out of acute nephritis, as independent of each other, but with intervening links; if in the acute attack the tubules are principally affected, the chronic nephritis will take the form of large white kidney; if the interstitial tissue has been much involved, contracted kidney will be the result; and there may be intermediate types according to the degree to which each tissue has been primarily involved. It must be remembered that the initial acute stage may be latent, or even altogether absent, and that, in practice, cases are not uncommon in which large white kidney or pale granular kidney arises without any evidence of a previous acute nephritis.

What are the Symptoms of Large White Kidney?

The disease may be a sequel of acute nephritis, the œdema, albuminuria, and other symptoms continuing. The

urine is somewhat scanty, turbid, high-coloured, and highly albuminous; it contains blood, although less than in the acute disease, and casts, which are granular, hyaline, sometimes epithelial, and *fatty*. Urea is defective. Cardiovascular changes, at first not pronounced, become gradually more marked, the left ventricle being hypertrophied and the arteries thickened, while the second aortic sound is accentuated. Œdema is general and extreme, and dropsy of the serous cavities is common. Inflammatory complications are frequent, and signs of uræmia may be found in sickness and headache, or in uræmic convulsions or amaurosis. Death after a protracted illness is the usual result, and is due, as a rule, to the complications mentioned, or to œdema of the lungs or glottis.

When it develops insidiously, the first symptoms of large white kidney are pallor, headache, anorexia, sickness, and frequent micturition. Œdema first appears in the feet and face, and gradually spreads.

What are the Symptoms of Pale Granular Kidney?

It may follow upon large white kidney if the patient survive, but the onset is usually insidious. Headache, sickness and vomiting, and anæmia are present, with dropsy, affecting chiefly the feet and face; the urine is turbid, somewhat pale, rather copious ($\bar{3}$ l.-$\bar{3}$ lxxx) and contains much albumin and little blood. Casts are chiefly hyaline and granular; urea is defective. Cardiovascular symptoms are prominent, and albuminuric retinitis may occur. Death is due to uræmia, cerebral hæmorrhage, inflammatory complications, or cardiac failure.

How are these Conditions to be treated?

In large white kidney the treatment is much the same as that for acute nephritis; but milk diet, diaphoresis, and

free purgation are the chief measures, and in the later stages iron must be given for the anæmia.

In the more chronic condition of pale granular kidney, confinement to bed is unnecessary except during acute exacerbations. The patient should guard against chill by warm underclothing, and when possible should live in a mild climate. Diet may be less restricted, and the daily loss of albumin may be to some extent compensated for by nitrogenous food. Meat, however, should not be taken more than once a day, and the diet, while nutritious, must be light and easily digestible. Fresh air and daily gentle exercise are advisable. Of medicaments, tonics are most useful, and iron to meet the anæmia, while excessively high blood-pressure may be reduced by saline laxatives, an occasional dose of calomel, iodide of potassium, or the vaso-dilators (spt. ætheris nitros., nitroglycerine, nitrite of sodium, erythrol tetranitrate). These last drugs, however, are of little use where the arteries have undergone fibrotic change.

For the treatment of uræmia, see p. 130, but remember that venesection is not so well borne in chronic as in acute nephritis, on account of the anæmic state of the blood.

CHRONIC INTERSTITIAL NEPHRITIS.

(Small Red Kidney, Granular, Cirrhotic, or Gouty Kidney.)

What are the Causes of Granular Kidney?

The disease is of independent origin, and is not connected with acute nephritis. It arises at or about middle life, and affects males more often than females. It is constantly associated with arteriosclerotic changes, and while in one of its forms these are probably secondary to the renal condition, in the other (*arteriosclerotic kidney*) the kidney disease

constitutes only a part of the general state. A hereditary tendency to the disease is sometimes observed, probably because arteriosclerosis is itself a hereditary affection. The chief exciting causes are long-standing chronic intoxications, the result, most commonly, of an excessively nitrogenous diet. The disease is thus often met with in gouty subjects. It is also frequent in lead-workers, and chronic alcoholism may contribute to it, although its influence is less than that of over-feeding.

Describe the Morbid Changes.

In typical cases the kidneys are much shrunken. The capsule is firmly adherent, and when it is torn off fragments of the cortex come with it. The surface is purplish-red, granular, and studded with small cysts, which are retention-cysts of the tubules produced by contraction of the interstitial tissue. The cortex is very narrow. Microscopically, the glomeruli are atrophied, and the capsules of Bowman much thickened, while throughout the kidney there is excess of fibrous tissue with atrophy of the tubules, the lumen of which often expands into microscopic or somewhat larger cysts.

What are the Symptoms?

The onset of the disease is very insidious. Pallor and œdema are absent in the early stages, and the first symptoms are often those of chronic uræmia. Thus headache, dyspepsia, anorexia, thirst, or in some cases failing vision may bring the patient for advice; and in all such cases occurring in the middle-aged the urine should be carefully examined. The urine remains clear, and its condition does not usually attract the patient's attention, although if he is questioned a history of *polyuria* may be obtainable. The symptom is often chiefly prominent at night. In other cases cardiac symptoms

may be the first in evidence—palpitation, dyspnœa, etc.—and in a few the first sign may be the sudden onset of acute uræmia, uræmic amaurosis, or an inflammatory complication (*e.g.*, pleurisy, pericarditis).

What are the Characters of the Urine?

It is passed in large quantities, and is pale in colour, *clear*, of low specific gravity in established cases (1005-1012), and contains little or no sediment on standing. Urea, chlorides, and phosphates are diminished in amount. Scanty granular or hyaline casts may be seen in the centrifugalised sediment, but blood is absent save for occasional intercurrent hæmaturia. Albumin, as a rule, is scanty, and it is often absent at certain periods of the day. A twenty-four hours' specimen should always be examined, but, even so, it may be found that albumin is absent in some cases for days at a time.

How is the Diagnosis to be made in such Cases?

The other characters of the urine, especially polyuria and defect of urea, will have awakened suspicion, along with the symptoms of chronic uræmia. Examination of the cardio-vascular system will then reveal sclerosis of the arteries, and evidence of hypertrophy of the left ventricle, usually considerable, and sometimes extreme. Albuminuric retinitis may also be present.

What is the Course of the Disease?

In the absence of complications, fair health may be maintained so long as the cardiac hypertrophy is adequate, and often for many years. Ultimately compensation fails, the left ventricle dilates, relative mitral insufficiency is established, and œdema of the *cardiac* type (*i.e.*, affecting the feet first, and extending gradually upwards) appears, and is followed by the other signs of cardiac failure. The disease may at any time be cut short by the occurrence of complica-

tions—uræmia, cerebral hæmorrhage, inflammations of the serous sacs or of internal organs (pneumonia, bronchitis, ulcerative colitis, etc.). Attacks of acute nephritis may also hasten its course.

How is Chronic Interstitial Nephritis to be distinguished from Pale Granular Kidney?

By its etiology and later age of onset; by the absence of pallor and œdema; and by the urinary characters (abundant secretion of clear pale urine containing little or no albumin and few casts, as contrasted with the less abundant turbid urine containing much albumin and more numerous casts in pale granular kidney).

What is the Treatment of Chronic Interstitial Nephritis?

It is on similar lines to that of pale granular kidney (see p. 135).

PYELITIS, PYELONEPHRITIS, AND PYONEPHROSIS.

Define the above-mentioned Terms.

Pyelitis is an inflammation of the pelvis of the kidney. Should the inflammatory change extend to the renal substance, we have the condition of pyelonephritis, a condition which, from its mode of origin, is usually suppurative. Pyelonephritis, therefore, is a suppurative inflammation of the renal substance and the renal pelvis. Pyonephrosis means an accumulation of pus in the renal pelvis consequent upon blocking of the ureter. The pus being unable to escape, the pelvis becomes distended, and the substance of the kidney is compressed, flattened, and atrophied.

What is the Origin of such Conditions?

In the great majority of cases, microbic infection. The bacillus coli is the commonest infecting organism, but others

may also be responsible (pyogenic organisms, gonococcus, tubercle bacillus, etc.). The routes by which the organisms gain access to the pelvis are various: through the blood-stream, or through the lower urinary passages, either along the lumen of the ureter or by the lymphatics. The chief predisposing causes are the following:—acute infections, constitutional diseases such as diabetes, severe anæmia, Bright's disease; extension of inflammation from below; the irritation of renal calculi, parasites, tumours or blood-clot; drugs such as cantharides; pressure upon the ureter, leading to dilatation and to decomposition of the retained urine; pregnancy; and injuries to the spinal cord.

Describe the Morbid Appearances.

In *pyelitis* the pelvic mucous membrane is opaque, thickened, and engorged, often with small hæmorrhages on its surface, or superficial erosions; and the pelvic cavity contains pus. In *pyelonephritis* there are in addition streaks of pus radiating through the pyramids into the cortex, in which small abscesses may be found. The kidney is large and soft, with a mottled surface. In *pyonephrosis* the pelvis is much distended by pus, and the kidney, flattened as above mentioned, is also the seat of inflammatory changes. It may be so much atrophied as to constitute merely a thin wall to the collection of pus, which is divided by septa formed of the interpyramidal connective tissue.

What are the Symptoms of Pyelitis?

They vary with the stage of the disease. In a simple pyelitis there may be dull pain in the loin, and perhaps some frequency of micturition. The urine contains a few pus cells, tailed epithelial cells from the pelvis of the kidney, and sometimes a little blood. Its reaction is acid, and there is only a trace of albumin. In later stages pus is more abundant, and on standing it forms a thick creamy deposit

at the bottom of the urine glass. Other symptoms are due to the causative disease.

What are the Tests for Pus in the Urine?

1. The recognition of leucocytes on microscopic examination of the sediment.

2. On the addition of Liq. Potassæ to the sediment it becomes viscid and glairy, and when it is poured from one vessel to another it falls in a ropy translucent string.

3. The addition of ozonic ether to purulent urine causes effervescence.

Urines containing pus are turbid, and in acid urine the deposit has the appearance mentioned above; if the urine is alkaline, the pus undergoes the change described under (2).

What are the Symptoms of Pyelonephritis?

Chills or rigors, with a high and swinging temperature, are superadded to the symptoms of the causative disease. Sweating and wasting are also present, the pulse is weak and rapid, and the supervention of the typhoid state is a forerunner of the end. The characters of the urine vary, but it may show, in addition to the pyuria probably already existent before the onset of pyelonephritis, the presence of casts, granular or hyaline, blood, and an amount of albumin in excess of that which the pyuria would account for.

How would you recognise a Pyonephrosis?

The distension of the renal pelvis causes a swelling in the flank, and leads to bulging both anteriorly and posteriorly. Pressure on the swelling elicits tenderness, and this is generally present in the lumbar region even when there is no palpable tumour. The tumour is dull to percussion except where it is crossed by the colon in front; it can be separated from the iliac crest below and the lower ribs in front; and it is crossed in front by a band of clear percussion, corresponding to the overlying colon.

If the obstruction causing the pyonephrosis is complete, the urine is clear and does not contain pus; if the obstruction is temporarily relieved, abundant pyuria appears, and the tumour subsides, to reappear as the sac once more fills up. Fever is generally present.

What is the Prognosis in these Conditions?

Mild pyelitis following the acute infections usually ends in recovery; calculous and other forms of pyelitis due to irritation may recover on removal of the cause. Pyelonephritis is fatal within a few weeks. In pyonephrosis the outlook, without treatment, is bad; the abscess may burst into the chest or peritoneum, or death may be due to exhaustion or amyloid disease.

How are they to be Treated?

Treatment must in the first place be directed to the cause, removal of which is indicated whenever possible. A simple pyelitis may be treated by rest in bed, light diet, diluent drinks in large quantity, and urotropin or other urinary antiseptics. Where there is much pus, in more chronic cases, astringents such as acetate of lead or the mineral acids are useful. Vaccines may be used in infections by the bacillus coli or streptococci. In pyelonephritis dry cupping and saline or other purgatives may be used, the skin should be kept active, and vaccines may possibly be of service. The treatment of pyonephrosis is surgical, nephrotomy and drainage of the sac being called for, and in some cases nephrectomy may be necessary.

HYDRONEPHROSIS.

What is meant by this Term?

Distension of the pelvis and calices of the kidney by retained urinary secretion, due usually to obstruction in the lower urinary passages. It may be congenital or acquired.

What are its Causes?

The congenital form, which is often double, is usually caused by an imperforate urethra, but abnormalities of the ureter may also cause a unilateral hydronephrosis. The kidneys may be so much distended as to impede delivery. The acquired form is frequently single, and is then due to narrowing of the ureter produced either by strictures or pressure from without (bands, kinks, floating kidney, etc.), or to obstruction of the ureter by an impacted calculus. The double form is the result of obstruction below the ureter, as from strictures of the urethra, enlarged prostrate, vesical tumours, or displacement of the uterus.

Describe the Morbid Changes.

The size attained by the tumour is greatest in the unilateral form. In bilateral hydronephrosis the function of both kidneys is interfered with, and uræmia follows while the dilatation is still comparatively small; in the unilateral form the other kidney carries on the renal function, and as there is no danger to life, the dilatation of the affected pelvis extends over a long time, and may become enormous.

The dilatation of the pelvis and calices leads to flattening of the pyramids, chronic interstitial changes, and ultimately atrophy of the kidney substance, which may even entirely disappear, the retained urinary secretion being then contained in a thin membranous sac, often divided into loculi by septa formed from the remains of the interpyramidal connective tissue. The ureter also is often enormously dilated. The retained fluid is pale and watery, and usually contains a trace of albumin. Urea and other urinary constituents are present only in minute quantities.

What are the Symptoms?

Incomplete bilateral obstruction leads to frequent micturition, with occasional partial suppression, lumbar pain, and,

later, symptoms of uræmia, death being preceded by the typhoid state. There may be little or no renal enlargement. Unilateral obstruction, when the tumour is large, leads to a sense of weight or pain in the loin, possibly accompanied with nausea or vomiting, and constipation from pressure on the colon. The signs of renal tumours are also present, namely :—bulging in the flank ; dulness to percussion over the affected area, the dull note extending into the loin, and being crossed in front by a band of clear percussion due to the presence of the colon ; and on palpation the recognition of a smooth or lobulated rounded tumour, which may in some cases even be fluctuant. In *intermittent* hydronephrosis, which may occur when the obstruction is due to a kink or floating kidney, the temporary removal of pressure on the ureter permits from time to time the escape of a large quantity of clear urine, the tumour at the same time diminishing or disappearing, and then slowly filling up again.

What is the Treatment ?

If symptoms are absent and the tumour is small, it should be left alone. Massage may succeed in emptying the sac, but aspiration is usually necessary. If the sac refills, it must be incised and drained, and should a discharging sinus remain, nephrectomy may be needed. This must not be done until it has been ascertained that the other kidney is healthy.

RENAL CALCULUS.

(Nephrolithiasis.)

What are the Common Forms of Renal Calculus ?

A renal calculus may be composed of uric acid (the most common), oxalate of lime, alternate layers of oxalate of lime and uric acid, or of phosphate of lime and triple phosphates.

The last form usually has a nucleus either of uric acid or oxalate of lime.

Mention the Rarer Forms.

Cystin, xanthin, calcium carbonate, urostealith, and indigo have all been found as the constituents of calculi.

What are the Characters of Renal Calculi?

They consist of a nucleus, which may differ in composition from the rest of the calculus, and of concentric laminæ surrounding it. The nucleus is most commonly composed of uric acid, but it may consist of oxalate of lime or of colloid bodies such as renal casts or blood-clot, or of a mass of micro-organisms. The surrounding laminæ may consist entirely of uric acid or oxalate of lime, or of the two substances in successive layers, and if the urine has undergone alkaline decomposition in the renal pelvis, phosphate of lime and triple phosphates may form the mass of the calculus. Uric acid calculi are smooth, reddish, or reddish-yellow in colour, and very hard. Where more than one is present, their adjacent surfaces are faceted. Oxalate calculi are also hard, but greyish in colour; they present a rough granular or tuberculated surface resembling that of a mulberry, and thus cause much irritation of the pelvis and ureter. Phosphatic calculi, which are white in colour, are softer, and readily crumble. They are often moulded to the shape of the pelvis and calices.

The size of calculi varies from that of coarse sand ("gravel") to a diameter of two or three inches.

What are the Causes of Renal Calculi?

Undue acidity of the urine, with excess either of uric acid or of oxalates. If at the same time the urinary salts (phosphates) or pigments are defective, precipitation of uric

acid is favoured. It is also necessary for the formation of calculi that some colloid material such as blood, casts, or mucus should be present in the pelvis, to form a matrix upon which the deposition of the uric acid or oxalate takes place.

What Morbid Changes may be caused by a Calculus?

Pyelitis from irritation of the renal pelvis; and blocking of the ureter leading to atrophy of the kidney, or to hydronephrosis or pyonephrosis. Continued pelvic irritation is said to have caused malignant renal tumours.

Describe the Symptoms.

They vary according as the stone is seated in the pelvis, or enters the ureter. In the former case there may be no symptoms, but more usually lumbar pain and tenderness, recurrent attacks of hæmaturia aggravated by exercise and relieved by rest, excessive acidity of the urine, which may contain uric acid or oxalate crystals, and the symptoms of pyelitis or hydronephrosis appear at some time in the course of the case.

When the stone enters the orifice of the ureter, an attempt at expulsion follows by contraction of the ureteral muscular fibres, and results either in impaction of the stone or in its passage downwards into the bladder. The symptoms thus produced are those of *renal colic.* The attack begins with intense pain seated in the loin, and radiating downwards to the testicle and inner side of the thigh, and inwards across the abdomen on the affected side. The testicle is retracted, tender, and swollen. The skin over the area of abdominal pain is hyperæsthetic, but the scrotal skin is not so. The pain, though constant, is subject to paroxysmal aggravations, and the attack of colic may last from a few hours to a few days. During its continuance the pulse is rapid and feeble, nausea and vomiting are present, with pallor, profuse sweating,

and collapse. Micturition is frequent, but only a few drops of urine, often blood-stained, are passed at each attempt, and the urine may be suppressed in severe attacks. Cessation of the attack is due to the passage of the stone into the bladder, to its impaction in the ureter, or to its slipping back into the pelvis.

What is meant by Obstructive Suppression?

A condition which may arise in renal calculus when, one ureter being already blocked by an impacted calculus, or one kidney being functionally inactive, the other ureter becomes blocked in the course of an attack of renal colic. It leads to the symptoms of *latent uræmia* (see p. 125), and to death in about ten days.

Does Renal Colic occur in other Conditions than Calculus?

Yes, similar but briefer attacks may be due to the passage of urinary sand or gravel (small concretions of uric acid), or to that of blood-clots or fragments of tumour-tissue. These conditions are to be distinguished by palpation of the kidney, the use of X-rays, and cystoscopic examination.

What is the Treatment of Renal Calculus?

Uric acid gravel may be dissolved by keeping the urine alkaline with large doses of citrate or acetate of potassium (℥ i-℥ ii thrice daily), the treatment being kept up for several months. The diet must be light and non-nitrogenous, mineral waters may be taken freely, and stimulants should be avoided. If a calculus has formed, operation is indicated. In the attack of renal colic, relief of pain is the first indication. Morphia should be injected at once, being antispasmodic as well as anodyne; hot fomentations may be applied locally, or a hot bath may be given; and in urgent cases chloroform may be inhaled.

TUBERCULOSIS OF THE KIDNEY.

In what Forms does this Disease occur?

It may be part of a general tuberculosis, and in this case the renal tubercles are miliary; or it may be primary, when it usually spreads down the ureter to the bladder. A local tuberculosis may also extend upwards from the bladder, in which case both kidneys may be infected.

Describe the Morbid Changes.

In primary renal tuberculosis the first step is the formation of tubercles in the medulla or cortex. These go on to caseation and softening, and cavities are then created, separated from each other by strands of intermedullary connective tissue. Almost the whole substance of the kidney may thus be destroyed. Along with this process, or consecutive to it, a tuberculous pyelitis occurs, and the disease spreads down the ureter and involves the bladder, at first in the region of the ureteral orifice. Ultimately it may extend to the prostate, and even sometimes to the urethra and testes.

What are the Symptoms?

Mainly those of pyelitis or pyelo-cystitis; dull pain in the loins, sometimes with tenderness on pressure, frequency of micturition, pyuria, the urine usually being acid, but sometimes alkaline from ammoniacal decomposition, and containing pelvic epithelium and tubercle bacilli. Albuminuria is proportionate to the amount of pus, and intermittent hæmaturia may be present. The general symptoms are fever, wasting, and sweating. Extension to other organs may occur, and death may be due to this cause, to exhaustion, or to uræmia if both kidneys are involved.

Indicate the Main Lines of Treatment.

The general treatment consists in supporting the strength, and the administration of tonics, as in other forms of tuberculosis. In early stages tuberculin may possibly be used, but if the diagnosis is certain, and the other kidney is unaffected, nephrectomy is the better course. In advanced disease, where the other kidney has also suffered, nephrectomy is inadvisable, but nephrotomy, with subsequent drainage, may relieve symptoms.

TUMOURS OF THE KIDNEY.

Of what Nature are Renal Tumours?

They are almost exclusively malignant, and may be sarcomata, epitheliomata, or adeno-carcinomata. The sarcomata occur in early life, and may be of congenital origin. They grow to a great size, and rapidly lead to death.

The other varieties occur in adult life, the epitheliomata taking origin in the renal pelvis, the adeno-carcinomata in the cortex. Many of the latter resemble in structure the suprarenal cortex, from remains of which, included in the substance of the kidney, they are held to originate, and are hence known as *hypernephromata.*

What are the Symptoms?

Aching pain in the lumbar region, which may alternate with attacks of renal colic if blood-clot enters the ureter; hæmaturia, moderate or profuse, and intermittent; cachexia; and the signs of renal tumour.

How is a Renal Tumour to be recognised?

It occupies the flank, and causes bulging laterally and in a forward direction. It is rounded in outline, and its long

axis is vertical. It is crossed by the ascending or descending colon which, *if empty*, gives a band of clear percussion breaking the dull note due to the tumour. The percussion dullness extends to the middle line behind; the fingers can be slipped under the ribs above it, and under the iliac crest below; and the tumour does not move on respiration.

What is the Course of such Cases?

The tumour may give rise to metastases in the lungs, liver, or lumbar glands; or direct extension to the retro-peritoneal tissue may occur. Pressure upon the spermatic vein may cause varicocele; upon the portal vein, ascites; upon the vena cava, œdema of the legs; upon the intestine, symptoms of obstruction. Death ensues in from six months to two years.

In very early cases removal may be attempted; later, palliation of symptoms is the only resource.

AMYLOID DISEASE.

Mention the Causes of this Affection.

They are those of amyloid disease in general: prolonged suppuration, phthisis, and old-standing syphilis. It occasionally occurs in cachectic states (cancer, malaria), and although not an inflammatory but a degenerative process, it is sometimes associated with chronic nephritis.

What are the Structural Changes?

The disease first affects the glomeruli, whence it spreads to the arterioles. It then attacks the basement membrane of the tubules. The affected structures stain of a dark brown colour with tincture of iodine. To the naked eye the kidney may at first appear unchanged, but iodine will show the glomerular affection. Later the kidney is enlarged, pale, and firm, and the capsule is easily detached. On

section the cortex is yellowish, with a waxy lustre, and the medulla is injected. If interstitial nephritis co-exists, the kidney may be smaller than normal.

Describe the Symptoms.

The urinary changes are variable. Where there is interstitial nephritis, the urine will present the characters of that disease (see p. 135), with perhaps more than the usual amount of albumin; where there is no nephritis, the urine is clear, of normal colour or slightly pale, and without appreciable sediment. The quantity varies; it may be normal, but is often somewhat excessive, and in that case the specific gravity is rather low. Albumin is usually present, but its quantity varies, being sometimes small, but often abundant, while occasionally it is entirely absent. Blood is not present, and the few casts that may be found in the scanty sediment are waxy or hyaline. Dropsy is considerable, but inflammatory complications are absent unless in the presence of nephritis.

The other symptoms are those either of amyloid disease in other organs—diarrhœa, enlargement of the liver and spleen—or of the causative disease.

Treatment must be directed to the cause — phthisis, syphilis, bone disease, etc.

MOVABLE KIDNEY.

What are the Causes of undue Mobility of the Kidney?

It may be congenital, the kidney having a mesonephron, but much more often it is acquired, as a result of relaxation of the subperitoneal fascia or loss of the perinephric fat. It may thus be part of a general enteroptosis, or may occur after repeated pregnancies. It is much commoner in women than men, and on the right side than on the left.

What are its Symptoms?

In many cases there are none, but a dragging pain in the loin is often complained of, and becomes worse on exertion. In some instances pain like that of renal colic, coming on in paroxysms, and accompanied by rigor and vomiting (*Dietl's crises*) may occur as a result of twisting of the renal vessels; and temporary kinking of the ureter may lead to intermittent hydronephrosis. Bimanual examination reveals the organ under the ribs, or in some instances low in the abdomen. It forms a smooth, firm tumour of the size of the kidney, and is freely mobile. If it lies under the ribs it comes further downward on deep inspiration. Grasping it causes a sensation of nausea; and on manipulation it can be returned to the loin.

What is the Treatment?

Non-interference if there are no symptoms; if the discovery is accidental the patient should not be told. But when symptoms are present, the organ must be supported in its normal position by a pad and bandage, and the tone of the abdominal muscles should be improved by massage. If these measures fail and severe symptoms recur frequently, it may be necessary to stitch the kidney to the posterior abdominal wall (*nephropexy*).

III.—DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

1. THE BLOOD.

ANÆMIA.

What is meant by the Term Anæmia?

It is a general term signifying defect of blood, and under it are included a number of different states to which particular names are applied. Thus deficiency in the quantity of blood is known as oligæmia, wateriness of the blood as hydræmia, deficiency of corpuscles as oligocythæmia, and deficiency of hæmoglobin as oligochromæmia. These conditions are prominent in different degrees in the different forms of anæmia.

How are the Anæmias divided?

Into those which are primary diseases of the blood or blood-forming organs—primary or essential anæmias; and those which are the consequence of constitutional states or disease of other organs—secondary anæmias. The two primary anæmias are chlorosis and pernicious anæmia.

SECONDARY OR SYMPTOMATIC ANÆMIA.

What Conditions may give rise to Secondary Anæmia?

Interference with nutrition either from *deficiency of food* or from defective assimilation; excessive discharges (sinuses, vaginal discharges, diarrhœa, etc.); cachexias (syphilis, malaria, cancer, etc.); toxic states (bacterial, or caused by

metallic poisons such as lead); hæmorrhages, external or internal; affections due to intestinal parasites, such as ankylostomiasis and bothriocephalus anæmia.

What are the General Symptoms of such Anæmias?

Pallor affecting both the skin and mucous membranes; lassitude and debility; headache and vertigo; dyspepsia and constipation; palpitation, shortness of breath, and a tendency to syncope. There may be slight œdema of the feet.

In the profound anæmia following severe hæmorrhages the pulse is rapid and soft, the tendency to syncope is marked, and the bloodlessness of the brain may lead to convulsions.

What are the Cardio-vascular Changes?

The so-called hæmic murmur is usually audible over the heart. It is systolic in rhythm, generally soft in character, and has its seat of maximum intensity in the pulmonic area, although it may also be heard in the other valvular areas, especially the mitral. There is generally some degree of dilatation of the heart, and in severe cases this may be sufficient to produce insufficiency of the auriculo-ventricular valves, and hence a systolic murmur at the mitral or tricuspid orifice. The more usual pulmonic murmur may depend either on alterations in the quality of the blood or on a localised dilatation of the base of the pulmonary artery.

On listening over the lower end of the jugular vein a continuous humming sound, known as the *bruit de diable*, may often be heard, but it also occurs in other conditions than anæmia.

From the malnutrition of the vascular walls, hæmorrhages may occur, particularly in the severer anæmias; and venous thrombosis is sometimes found.

What is the Condition of the Blood in Secondary Anæmias?

There is a deficiency both of hæmoglobin and corpuscles, and after hæmorrhages, also of the blood plasma. The *colour index* (*i.e.* the figure obtained by dividing the percentage of hæmoglobin by the percentage of corpuscles) is 1·0 or less than unity, hæmoglobin and corpuscles being either diminished in the same proportion, or the corpuscles less diminished than the hæmoglobin.

The *treatment* of the secondary anæmias is mainly that of the causative disease, but iron or arsenic is usually called for in addition.

CHLOROSIS.

What is Chlorosis?

A form of anæmia very common in young women, and characterised by a marked deficiency in hæmoglobin with a relatively small loss of corpuscles.

What is its Etiology?

It originates between the ages of fifteen and twenty-one; and its onset is favoured by defective nutrition and lack of fresh air and exercise. Its actual cause is not known, although many theories are advanced to explain it. Among these are (1) absorption of toxins from the gastro-intestinal tract, (2) conversion of the iron of the food into unassimilable compounds by decomposition products formed in the intestine, (3) repeated losses of blood at the time of puberty (irregular menstruation), (4) congenital narrowness of the aorta, leading to an insufficient blood supply. None of them is adequate to explain all the cases.

Describe the Symptoms.

They are those of anæmia in general, including languor, palpitation and dyspnœa on exertion, headache and vertigo.

Constipation is prominent, and dyspeptic symptoms are frequent. The hæmic murmur and *bruit de diable* may be present as in other anæmias. The menses are disordered, amenorrhœa being common, and irregularity often marked; in some cases the flow is profuse. The complexion presents in many cases the greenish pallor that gives the disease its name. The blood shows a marked diminution of hæmoglobin (50 per cent. or less) and a relatively small diminution of red cells, which are rarely below 60 per cent. Poikilocytosis is often present, but seldom nucleated red cells, and the leucocytes are normal. The volume of the blood is increased, and its specific gravity, like the colour index, is low.

Are there any Complications?

Gastric ulcer is not uncommon, and in severe cases there is a tendency to venous thrombosis, which may affect the veins of the limbs, or even, though rarely, the cerebral sinuses. Head symptoms are then present, and sometimes optic neuritis. If the anæmia is profound, the malnutrition of the heart may cause temporary dilatation, with the signs of relative mitral insufficiency.

What is the Treatment?

Iron is the principal remedy, and the form chosen should be one which does not irritate the stomach. Blaud's Pills, *freshly made*, in doses of two or three pills thrice daily, reduced iron (gr. ii-iii thrice daily), or the scale preparations may be used. Ferrous should be preferred to ferric salts when dyspepsia is present. The constipation must be met, and a laxative such as aloes or sulphate of magnesia should be regularly used. The diet should be nourishing, but when dyspepsia or gastric ulcer is present it must be suitably modified. Rest is necessary at the outset, and when the anæmia is marked the patient must be kept in bed. At a later

stage gentle exercise short of fatigue, and plenty of fresh air, are indicated. Should iron alone be unsuccessful, it may be combined with arsenic.

PERNICIOUS ANÆMIA.

What is this Disease?

A profound anæmia of a progressive character, ending fatally, and distinguished by great destruction of the red corpuscles.

What are its Causes?

The essential cause is not definitely known. It occurs in males rather oftener than in females, and it usually arises between the ages of twenty-five and forty. Pregnancy and the puerperal state are sometimes associated with its development. Its chief characteristic is a marked hæmolysis, and this is probably dependent on a toxæmic condition, which William Hunter believes to be due to bacterial toxins. These toxins are formed, in his opinion, by a specific organism inhabiting the gastro-intestinal tract, and causing a specific glossitis and also gastric disturbances and diarrhœa. The organism, however, is not yet isolated. Anæmias clinically similar have also been found in certain cases of cancer of the stomach or bones, and in ankylostomiasis.

Describe the Morbid Changes.

The tissues generally are pallid; the liver and kidneys show fatty degeneration, and this is also prominent in the heart and large vessels. The liver, spleen, and kidneys contain an excess of iron in the form of hæmosiderin, which gives a blue colouration with potassium ferrocyanide and dilute hydrochloric acid. Hæmorrhages are common in the retina, the serous and mucous membranes, and the lungs. The

bone-marrow is hyperplastic and unduly red, and shows large numbers of nucleated red cells, among which megaloblasts are numerous. In some cases, however, the bone-marrow fails to react to the toxin, and in these instances (*aplastic anæmia*) it may be atrophic. The spinal cord is sometimes affected, and then shows a combined sclerosis of the posterior and lateral columns. Oral sepsis (pyorrhœa alveolaris) is common.

What are the Symptoms?

Gradual loss of strength, with marked pallor of the mucous membranes and a *lemon yellow* tint of the skin, which is often velvety to the touch. Wasting is not at first apparent, as the subcutaneous fat is well preserved, but the muscular substance is wasted. The symptoms of anæmia already described are also present. In some instances there is slight jaundice (hæmolytic jaundice), and the urine is generally high-coloured from the presence of excess of urobilin. The tongue is often the seat of a glossitis which Hunter regards as specific, and gastric and intestinal disturbances (anorexia, nausea, vomiting, diarrhœa) are frequent. Pyorrhœa alveolaris is also common. Febrile attacks, during which the symptoms are aggravated, recur at irregular intervals, between which there may be quiescence or even temporary improvement under treatment. Hæmorrhages from mucous membranes are not uncommon, and retinal hæmorrhages are very frequent. The liver and spleen are sometimes moderately enlarged. In exceptional cases there may be symptoms of sclerosis of the spinal cord (ataxic paraplegia).

Describe the Condition of the Blood.

The red corpuscles are much reduced in number, and in the late stages they may be as few as 500,000 per cmm. The hæmoglobin is less reduced, and the colour index is therefore high. The red cells are altered in shape (*poikilo-*

cytosis) and in size, some being larger (*macrocytes*) and some smaller (*microcytes*) than normal. Nucleated red cells (*normoblasts* or *megaloblasts*, according to size) are also found. At certain stages of the disease there may be *blood-crises*, in which large numbers of nucleated red cells are present, and disappear again after a few days. The blood when shed is pale, and coagulates slowly.

What is the Treatment?

Arsenic is the chief remedy for the condition of the blood, iron alone being of little use. To be of any value arsenic must be pushed. The treatment is begun by small doses, which are very gradually increased until the patient is taking 12 to 15 minims of Liq. Arsenicalis thrice daily, and this dosage may be continued so long as there are no signs of arsenic intoxication. If these appear, the drug must be intermitted for a short time, and then resumed with a lower dose. Iron is sometimes used as an adjuvant to arsenic. Gastro-intestinal conditions and oral sepsis must receive attention; pyorrhœa alveolaris should be seen to, carious teeth removed, and salol, salicylate of soda, or divided doses of calomel given internally. Vomiting may sometimes yield only to lavage of the stomach. Antistreptococcic serum has been tried, but in most cases unsuccessfully.

Under the continued use of arsenic great improvement may occur, and the blood state may approach the normal; but sooner or later a relapse ensues, and a succession of such relapses leads ultimately to death.

LEUKÆMIA (Leucocythæmia).

What are the Characteristics of this Anæmia?

It is marked by a great increase in the number of white corpuscles, and also by changes in the spleen, lymphatic glands, and bone-marrow.

Two type forms of the disease are described, spleno-medullary or myelogenous and lymphatic or lymphocytic. In the former the spleen and bone-marrow are chiefly affected, in the latter the lymphatic glands; and the characters of the blood differ in typical cases of the two forms. But there are many transitional conditions, *e.g.*, the spleen and bone-marrow may be chiefly affected, and yet the blood may show the characters of lymphatic leukæmia.

How is Leukæmia caused?

Its essential cause is unknown, but there is an irritation of the bone-marrow leading to the production of an excess of leucocytes, and probably due to some form of toxæmia. The disease occurs for the most part in adult men, although the lymphatic form may attack younger people.

What are the Morbid Changes?

The *spleen* is much enlarged in the spleno-medullary form, less so in the lymphatic. In the former case it may weigh as much as twelve pounds. Its capsule is thickened, and may be adherent to adjacent organs or to the abdominal wall. On section it is pinkish or brownish, with a dark and hypertrophied pulp in which the Malpighian bodies are not easily seen; and it commonly shows hæmorrhagic infarctions.

The *lymphatic glands* may be somewhat enlarged in either form, but most prominently in lymphatic leukæmia. The *bone marrow* may be dark brown or yellowish red in colour, and shows in the spleno-medullary form nucleated red corpuscles, polymorphonuclear cells, and myelocytes, and in the lymphatic form lymphocytes, large or small. The *liver* is much enlarged, and infiltrated with leucocytes, and the kidneys may also show leukæmic changes. As in other severe anæmias there may be fatty degeneration of the heart.

Describe the Symptoms.

The development of symptoms is gradual, and usually those of anæmia, palpitation, dyspnœa, digestive derangement, etc., are first noticed. Pallor is obvious, and hæmorrhages, from the nose or, less commonly, from mucous membranes, may be early symptoms. Retinal and internal hæmorrhages also occur. In the one form of the disease the spleen, in the other the lymphatic glands, are notably enlarged. The spleen may attain an enormous size; it extends downwards and forwards into the iliac fossa, and across the abdomen to the umbilicus or even further, forms a firm, flattened, cake-like mass with rounded edges, and presents a recognisable splenic notch on its anterior border. The dulness due to splenic tumour is not interrupted by resonance due to the presence of the colon, as that of renal tumour is, for the colon never lies in front of it. The liver is also enlarged, though not greatly, and the bones are sometimes tender.

In the later stages hæmorrhages are more common than in the earlier; œdema of the feet occurs, and sometimes ascites; diarrhœa is frequent, and intermittent attacks of fever are common. The urine is of high specific gravity, and contains excess of uric acid. Death takes place in from six months to four years, the course being more rapid in the lymphatic type. Some cases of lymphatic leukæmia, indeed, end fatally in a few weeks or months (acute leukæmia).

What are the Changes in the Blood?

In spleno-medullary (or *myelocytic*) leukæmia the leucocytes are enormously increased, numbering 300,000 or more per cmm., the red cells are much reduced (2,000,000 or less), and the hæmoglobin even more so. The colour index is therefore low, and the ratio between white and red cells may be as 1 to 10, or even in severe cases as 1 to 2. Myelocytes are present in large numbers (30-50 per cent.),

polymorphonuclears are relatively decreased, but absolutely increased (40-50 per cent.), eosinophiles are proportionately increased (2-4 per cent.), and mast cells are often present. Lymphocytes are diminished. The red cells show the characters of a profound anæmia.

In lymphatic leukæmia the lymphocytes alone are increased, and may amount to over 90 per cent. of the white cells. They are in some cases small lymphocytes, in others large lymphocytes. The polymorphonuclear cells are few, and myelocytes and mast cells are rare.

What is the Treatment?

Arsenic, given in gradually increasing doses and continued for long periods, is the only reliable drug. Repeated applications of X-rays to the spleen have also produced temporary benefit. Of late benzol, in doses of ℥ xv thrice daily has been largely tried, and, it is said, with excellent results.

LYMPHADENOMA (Hodgkin's Disease).

What is meant by Hodgkin's Disease?

An affection characterised by enlargement of the lymphatic glands with secondary lymphoid growths in internal organs, and accompanied by anæmia.

What are its Causes?

The essential cause is not known, but many consider Hodgkin's disease to be an infection, and to bear some relation to leukæmia. It is more common in men than women, and chiefly affects children and young adults, though it may occur at any age. It sometimes follows injury, local lesions, or depressed general health, but there is often no obvious cause.

Describe the Morbid Changes.

The affected glands are enlarged and non-adherent. They vary in consistency, being sometimes very firm, at other times soft and elastic. There is, as a rule, no tendency to suppuration or caseation. On microscopic examination both connective tissue and endothelial cells are proliferated, and multinuclear giant cells (lymphadenoma cells) are also to be seen. The connective tissue forms a reticulum within which are abundant lymphocytes, and eosinophil cells are also present. The spleen is enlarged and firm, and on section shows a number of yellowish masses of lymphoid tissue enclosed in a fibrous reticulum, and originating in the Malpighian bodies. Similar changes may be found in the liver, kidneys, and lungs, and sometimes in the bone-marrow.

Describe the Symptoms.

Glandular enlargement is usually the first symptom, and generally begins in the cervical glands, at first unilaterally. The glands, though affected in groups, remain discrete and are not adherent to the skin. They vary in hardness, and are neither painful nor tender. The opposite side of the neck becomes affected, then the axillary and inguinal, and ultimately even the mediastinal and mesenteric glands. At a late stage the glands may become adherent, but they rarely suppurate. There is moderate enlargement of the spleen.

Anæmia is usually an early symptom, but may be slow to develop. The red cells are much reduced, and also the hæmoglobin, the colour index being unity or less than unity. The leucocytes are not increased.

Intercurrent attacks of fever are common. The fever may be malarial in type, or the attacks may last for a week or more, with apyretic intervals, or there may be continued fever.

Pressure symptoms may follow enlargement of the cervical and internal glands. The larynx, trachea, or

œsophagus in the neck; the great veins and the recurrent laryngeal nerves in the thorax; and the nerves of the solar plexus in the abdomen, may all be pressed upon. Pigmentation may follow pressure on the solar plexus.

Weakness, dyspnœa, œdema of the lower limbs, dropsy of the internal cavities, and hæmorrhages from mucous membranes are the later symptoms. Death takes place in from two to four years after the onset.

What is the Treatment?

Arsenic, given as in pernicious anæmia, is often of temporary benefit. If only one group of glands is affected, excision may be practised. The use of X-rays has also proved temporarily successful.

SCURVY (Scorbutus).

What is Scurvy?

A disease characterised by debility, anæmia, hæmorrhages, and a spongy condition of the gums.

How is it produced?

It is due to dietetic errors, the essential factor being the absence of fresh vegetables from the dietary. Defect either of the potassium salts or of the salts of vegetable acids (malates, citrates, tartrates), is supposed to be the cause. The disease is now rare, but was formerly very common in sailors during long voyages. An infective origin has also been assigned to scurvy, but upon slender evidence. Predisposing causes are fatigue, exposure, absence of sunlight, and depressing diseases.

The *morbid changes* are those of severe anæmia.

Describe the Symptoms.

The onset is insidious, with signs of increasing anæmia and languor. Subcutaneous petechial hæmorrhages surrounding the hair follicles, and large subcutaneous extravasations resembling bruises, appear on different parts of the body. Intramuscular hæmorrhages may cause brawny and tender swellings over the calves. The larger extravasations are common in the popliteal spaces, the bend of the elbow, and on the extensor surfaces of the limbs. At the same time the gums become swollen and spongy, and readily bleed on the slightest pressure. The breath is intensely offensive, and the teeth are loose. Epistaxis is common, and in severe cases there may be hæmorrhage from the lungs, stomach, or bowels. Marked anæmia is present (red cells 3,000,000, or less; colour index low), and there may be night blindness due to exhaustion of the retina. Death may be caused by exhaustion, syncope, or inflammatory complications.

What is the Treatment?

This is mainly dietetic. There should be a liberal supply of fresh vegetables and fresh fruit-juices. Milk should also be freely used, and as the condition of the mouth improves, fresh meat should be added. Rest in bed is necessary while there is a risk of syncope. Local measures are needed for the gums—chlorate of potash mouth washes, applications of nitrate of silver, etc. Pain in the limbs may require sedative liniments.

What are the Characteristics of Infantile Scurvy?

It occurs before the end of the second year, usually in children artifically reared. The lower limbs are tender, and the child does not willingly move them; there may be swellings of the shafts of the long bones from subperiosteal hæmorrhages; there may be separation of the epiphyses with

crepitus on manipulation; and if teeth are present there is sponginess of the gums. Fresh fruit juices and cow's milk should be given.

PURPURA.

What is Purpura?

A condition characterised by the presence of subcutaneous hæmorrhages, which give rise to red or purple blotches upon the skin. It is not a definite disease, but rather a name for a symptom of which the cause may vary.

What are its Main Causes?

A purpuric eruption may appear in the malignant type of specific fevers, the rash in such cases becoming hæmorrhagic—typhus, measles, small-pox, etc.; in such cachectic conditions as scurvy, chronic Bright's disease, malignant endocarditis, etc.; in poisoning by certain drugs—potassium iodide, antipyrin, benzol, etc., and by snake-venom; and in some nervous diseases—myelitis, locomotor ataxia, etc. There are also forms of unknown etiology, but probably, like the above, due to toxic causes. They are known as purpura simplex and purpura hæmorrhagica.

Describe the Symptoms.

In purpura simplex there is an eruption of deep-red spots, scattered over the body, and not disappearing on pressure. They are irregularly distributed and often more abundant on the legs than elsewhere. The general symptoms are slight, and the eruption disappears in about a fortnight.

Purpura hæmorrhagica, also known as *morbus maculosus Werlhoffii*, is marked by the presence of more extensive hæmorrhages, large bruise-like patches (*ecchymoses*) being

present in addition to the petechiæ, and blood-filled bullæ being also present. There are also hæmorrhages from the mucous membranes, and sometimes from the kidneys, lungs, or female genitalia. Fever is often present, anæmia is marked, and the severer cases end in death.

Henoch's purpura is a form which principally affects children, and in which there may be erythema or urticaria in addition to the purpuric eruption. There are also joint pains, attacks of pain in the abdomen, hæmorrhage from mucous membranes or from the kidney, and sometimes nephritis. The disease is often fatal.

Peliosis rheumatica is a term which should be restricted to the purpuric rashes sometimes occurring in acute rheumatism.

What is the Treatment?

In symptomatic purpura the cause must be treated. In the idiopathic forms the patient must be kept in bed, and tonics such as iron and arsenic may be given. When hæmorrhage is severe astringents must be used, and ergotin, turpentine (℥ x thrice daily), dilute sulphuric acid, adrenalin, and calcium lactate (gr. xv-xx every four hours), are all of use.

HÆMOPHILIA.

What is this Condition?

A tendency to uncontrollable bleeding, sometimes associated with effusion into the joints. It may be either traumatic or spontaneous, and is usually hereditary, the diathesis being transmitted through the female line, though the subjects of the disease are generally males.

Describe its Symptoms.

The condition is congenital, and symptoms usually begin in infancy. Uncontrollable bleeding may follow the infliction of a very trifling injury, or the performance of such small operations as the extraction of a tooth or the opening of a small abscess. Extensive subcutaneous hæmorrhages may also result from very slight violence. In more severe cases, spontaneous hæmorrhage may occur from the gums, nose, or other mucous membranes, and sometimes hæmorrhagic effusion occurs into the cavity of the joints. If the losses of blood are frequent and large, there may be severe anæmia; otherwise the health is good between the attacks. Most of the sufferers die in childhood.

What is the Pathology of Hæmophilia?

In these subjects the coagulation time of the blood is protracted, and Addis has shown that the fault lies, not in the capillary vessels, but in a congenital deficiency of prothrombin. Leucopenia is usually present, but there is no other obvious change in the blood.

How is it to be treated?

Such patients must lead a sequestered life, and avoid all chance of injury. Operations must be avoided unless urgently called for. The diet should be non-stimulating, and the bowels must be kept open. Even the slightest hæmorrhage must be treated by styptics, such as perchloride of iron, gallic acid, or calcium lactate (gr. xv. every four hours). Adrenalin may be used for hæmorrhages from the mouth or nose, and compression for external wounds. Gelatine may be injected, and horse serum, supplying the deficient prothrombin, may also be given hypodermically. Transfusion of blood has occasionally proved successful. Women of hæmophilic families should not marry.

HÆMOGLOBINURIA.

What is Hæmoglobinuria?

The presence of blood pigment in the urine, without blood corpuscles, the pigment being either oxyhæmoglobin, or, if the urine has had time to act upon the blood, methæmoglobin.

To what is it due?

To the breaking up of the blood corpuscles within the vessels with liberation of their hæmoglobin, which is excreted by the kidneys. It occurs in two forms, as the result of toxic action, and as a disease known as paroxysmal hæmoglobinuria.

What Poisons or Toxins may give rise to it?

Among drugs, chlorate of potassium, naphthol, nitrobenzol, pyrogallic acid, arseniuretted hydrogen, and carbon monoxide; among toxins, those of severe infectious, septic diseases, and extensive burns. It is the typical symptom of blackwater fever; it follows transfusion of the blood of one species of mammal into another species; and it occurs epidemically in infants.

How is Hæmoglobinuria recognised?

The urine presents the same appearances as in hæmaturia, and responds to the guaiac and spectroscopic tests; but on microscopic examination blood corpuscles are absent.

What is Paroxysmal Hæmoglobinuria?

A condition which occurs in young adults or adolescents, is closely associated with syphilis, and of which the attacks are determined by exposure to cold or over-exertion. The attack is preceded by languor, chilliness or rigor, lumbar or

abdominal pain, nausea, diarrhœa, or pains in the limbs. The passage of hæmoglobin-stained urine follows soon after those symptoms, and the whole attack may be over in twenty-four hours, the subject being in normal health in the intervals.

What is its Cause?

It is due to an immune-body contained in the serum, the hæmolytic properties of this body becoming active only after the blood has been cooled. The chilling takes place at the surface on exposure to cold, and there the red cells combine with the immune-body, this combination uniting with the normal complement when the blood returns to the warmer parts, and causing hæmolysis, hæmoglobinæmia, and ultimately hæmoglobinuria.

How would you treat it?

By the avoidance of exposure to cold, the use of warm clothing, and wintering in a mild climate. Hot drinks may be given during the chill. Specific treatment should be employed when the Wassermann reaction is positive, and tonics such as arsenic or quinine may be given for long periods.

II. THE DUCTLESS GLANDS.

(A) THE THYROID GLAND.

MYXŒDEMA AND CRETINISM.

What is Myxœdema, and what are its Causes?

It is a condition characterised by infiltration of the subcutaneous tissue, a dry, harsh skin, mental inactivity, and atrophy of the thyroid gland. It is due to defective thyroid secretion, the vesicular cells being atrophied and the gland as a whole fibrosed; and similar symptoms follow absence or atrophy of the gland either after its operative removal—*cachexia strumipriva*, or in *cretinism*, the congenital form of myxœdema. The disease is much more common in women than in men, and usually begins between the ages of thirty and fifty. It is sometimes preceded by goitre or exophthalmic goitre.

Describe the Morbid Changes.

The changes in the gland are those above noted. The skin shows nuclear proliferation and formation of connective tissue around the hair follicles and sweat-glands, with an increase of subcutaneous fat. There is sometimes a mucinoid or gelatinous substance in the subcutaneous connective tissue. Other changes are not constant, but interstitial nephritis may be present.

What are the Symptoms?

The onset is insidious, languor and apathy being among the early symptoms. When the disease is established, the face is broad, the lips thick, and there is a central patch of

red on the cheeks, the rest of the face being sallow and apathetic in expression. The speech is slow and monotonous, and the tongue thickened. The skin of the body and limbs is thick, dry, and harsh, but does not pit on pressure. The hands and feet are broadened, with thickened digits ("spade-like" hands), the nails are brittle, and the hair is dry, scanty, and falls out freely. The temperature is subnormal, and feelings of cold are complained of. The mind is dull, the memory defective, and later on there may be hallucinations or delusions. In the late stages interstitial nephritis may cause urinary changes and œdema which otherwise are not present. There is considerable anæmia, and the bowels are constipated. The course is prolonged, and death is due to intercurrent diseases.

What is the Treatment?

The administration of thyroid extract (B.P.) gr. ii-v thrice daily is the most important point. Symptoms rapidly disappear under its influence, but after apparent cure the drug must be constantly given in lessened doses throughout life. Signs of an overdose are headache, palpitation, tachycardia, feverishness, and gastro-intestinal disorders. The patient must be carefully protected from cold.

Describe a Case of Cretinism.

This, the infantile form of myxœdema, is endemic in parts of Central Europe, especially Switzerland, where it is often associated with goitre, the two conditions being sometimes present in the same individual. Sporadic cases occur all over Europe. In the endemic form the cretin is usually the child of a goitrous mother who is deficient in thyroid secretion, this defect exerting a toxic action upon the infant's thyroid gland. Symptoms begin in the first year of life, growth is arrested or stunted, the face is large, rounded, and baggy, the lips thick, and the tongue large, speech is defec-

tive or absent, and deaf-mutism may result; the limbs are bowed, and the gait is waddling; the belly is prominent and pendulous, and hernia is common; there are often subcutaneous fatty tumours above the clavicles; and the sexual organs are imperfectly developed in those cases which survive puberty.

The treatment, like that of myxœdema, is thyroid extract in appropriate doses. If begun early enough it leads to great improvement.

GOITRE (Bronchocele).

What is Goitre?

A persistent enlargement of the thyroid gland, due to hypertrophic or other changes exclusive of malignant disease. The morbid condition may be hypertrophic—parenchymatous goitre, cystic, or due to vascular dilatation.

What are its Causes?

It occurs endemically in deep mountain valleys, and particularly in limestone districts, being met with in Derbyshire and Devonshire, Central Europe, and the passes of Northern India. It most commonly attacks young women, and bad ventilation and overcrowding predispose to it. The essential cause is probably bacterial, and the work of M'Carrison and others shows that the organism may flourish both in the water and the soil of limestone districts, that it produces the goitre-forming toxins in the alimentary canal, and that a mixed vaccine prepared from the alimentary flora will relieve the symptoms of goitre. The organism is as yet unidentified, but the contagium can be separated from the water by filtration, and is destroyed by boiling.

Describe the Symptoms.

These consist in enlargement of the thyroid gland, uniform or unilateral, the tumour moving with deglutition, and in

evidence of pressure, in the case of marked enlargements, upon the trachea, œsophagus, or recurrent laryngeal nerve.

Treatment consists in removal from the affected district, or, if this is impossible, in drinking only boiled water, in the internal use of iodide of potassium, and the external application of iodine, in the use of intestinal antiseptics (thymol, gr. ii-v thrice daily, urotropin), or in the administration of vaccines prepared from the mixed flora of the bowel. Surgical interference may be needed if the tumour presses upon important structures.

EXOPHTHALMIC GOITRE.

Define this Disease.

It is characterised by enlargement of the thyroid gland, protrusion of the eyeballs (*exophthalmos*), rapid cardiac action, and frequently by muscular tremor.

What is its Etiology?

The disease is most frequent in women, in whom it begins between the ages of fifteen and thirty. It attacks men more seldom, and at a later age (thirty to forty-five). A neurotic tendency is often present in the patient, or a nervous heredity, and sometimes there is direct inheritance of the disease. There may be no obvious exciting cause, but sudden fright, grief, or shock, have frequently immediately preceded the onset.

What are the Morbid Changes and the Pathology of the Disease?

The main changes are found in the thyroid gland, which is enlarged and very vascular, with dilated and tortuous arteries. There is proliferation of the epithelium of the vesicles, which becomes columnar instead of cubical, and their contents are mucous rather than colloid. The thymus gland may persist, and other lymphatic glands may be

enlarged. The tachycardia causes cardiac hypertrophy; the fatty tissues of the orbit are increased; and minute hæmorrhages have sometimes been found in the central nervous system.

The pathology is obscure, but the symptoms are generally regarded as due to an excess of thyroid secretion, similar symptoms being found when large doses of thyroid are given experimentally, and operative removal of a portion of the gland being frequently followed by cure. But the cause of the *hyperthyrea* is not yet determined, although the etiology, the nervous heredity, and the ocular paralyses that are occasionally found would seem to indicate a starting point in the central nervous system.

Describe the Symptoms.

The onset may be abrupt, as after shock or fright, or more insidious. *Tachycardia* is often the first symptom, and the pulse rate may range between 100 and even 200 per minute. Palpitation and dyspnœa go along with it; anæmia is common; and dilatation of the heart may follow. Hence there may be hæmic murmurs or murmurs of relative insufficiency; and there is often visible pulsation of the large arteries. The other symptoms follow in irregular order. *Exophthalmos*, when prominent, causes a staring appearance, and is associated with imperfect closure of the eyelids. It may be unilateral, but is usually bilateral. The sclerotic is then visible all round the cornea from retraction of the upper lid (*Stellway's sign*); when the patient looks down, the upper lid only partially follows the movement of the eyeball (*von Græfe's sign*); there is imperfect convergence on near vision (*Möbius' sign*); and in some cases there are ocular paralyses (ptosis, strabismus). *Thyroid enlargement* takes place gradually, and may be bilateral, or may affect principally one or other lobe, or the isthmus. The gland feels elastic to the touch, and the increased vascularity causes a systolic thrill and murmur. *Tremor*

of a fine rhythmic character is most noticeable in the hands, but may affect the whole body.

Many other symptoms are occasionally present, most of them due to vasomotor disturbance. There may be cutaneous flushings or free perspiration; urinary changes—polyuria, glycosuria, albuminuria; gastro-intestinal symptoms—anorexia, diarrhœa, sometimes vomiting; attacks of fever; and mental changes which may tend to melancholia or mania. Many cases recover, others remain chronically affected, and about a fourth of the cases ends fatally after an illness of several years' duration.

What is the Treatment?

Rest is the first essential, and along with it fresh air, light nourishing diet, and avoidance of worry must be insisted on. Where the symptoms are marked confinement to bed is necessary. Stimulants must be forbidden, and tea, coffee, and tobacco much restricted. Belladonna or bromide of potassium may be given as sedatives, and if tachycardia is marked, digitalis, or ice to the thyroid, may be of use. Galvanism to the thyroid is often useful, the kathode being placed on the cervical spine. X-rays applied to the thyroid have given good results. Rodagen, the desiccated milk of thyroidectomised goats, and antithyroidin, the serum of thyroidectomised sheep or dogs, have not fulfilled expectations.

In cases which do not yield to treatment, and are rapidly progressive, operation may be considered, partial thyroidectomy or ligature of the superior thyroid arteries being the most successful. But such operations have still a high mortality, and it is questionable whether their percentage of permanent cures is higher than that attainable by medicinal means.

(B). THE SUPRARENAL GLAND.

ADDISON'S DISEASE.

Define this Condition.

It is a disease marked by debility and low blood-pressure, gastro-intestinal irritation, and pigmentation of the skin, and associated with morbid conditions of the suprarenal bodies.

What is its Etiology?

It is most often met with in males, and between the ages of twenty and forty, but other ages are not exempt. It is commonest among the poor; and for the most part the changes in the suprarenal bodies are tuberculous, whether they be primary or secondary to tuberculosis elsewhere. In some cases injury to the back or abdomen has been followed by Addison's disease.

What are the Morbid Changes and the Pathology?

The suprarenal capsules are usually tuberculous, fibrotic, and show caseation, which may lead to calcification or softening. Sometimes there may be simple atrophy, less commonly malignant growth. The semilunar ganglia are degenerated and pigmented, and their nerve fibres sclerosed. In some instances there is no obvious disease of the suprarenals.

The disease is due to the loss of the internal secretion of the suprarenals, the adrenalin constituent of which maintains the tone of the vascular musculature and of the heart, and inhibits peristalsis. Its absence therefore accounts for the asthenia, low blood-pressure, and cardiac weakness, and possibly also for the diarrhœa. The pigmentation has not yet found a satisfactory explanation, though it may possibly be accounted for by the close association not only of the adrenals but of the other chromaffin cells throughout the body with the sympathetic nervous system.

Describe a Case of Addison's Disease.

The onset is gradual, and is marked by feelings of languor and weakness, possibly with obscure pains in the back or epigastrium. The chief symptoms are a condition of *asthenia*, manifested in feeble action of the heart, low blood-pressure, muscular weakness, and a tendency to syncope; *gastro-intestinal attacks*, nausea, vomiting, and diarrhœa; and *pigmentation of the skin*. The temperature is often subnormal; there may be anæmia, but there is no marked wasting. Pigmentation begins in the parts which are normally darkest—areolæ, scrotum, axillæ, in parts subject to pressure (waistband, garters), and in those exposed to light. It remains deepest in those parts, but ultimately the whole surface may be affected. The mucous membranes are also pigmented, and dark patches may be seen upon the tongue and inside the cheeks.

The disease is fatal, death occurring sometimes in a few months, sometimes after several years, most commonly within a year or two. Asthenia is the most frequent cause of death.

What is the Treatment?

Avoidance of exposure to cold and of fatigue, along with the administration of iron, arsenic, or strychnine. Confinement to bed is called for in marked asthenia. Adrenalin may be given by the mouth or hypodermically, with temporary benefit; and one or two cases of cure under tuberculin have been reported.

(C). THE SPLEEN.

SPLENIC ANÆMIA (Banti's Disease).

What is this Disease?

A condition of anæmia, accompanied by great and progressive splenic enlargement, by characteristic changes in the blood, and in the late stages by cirrhosis of the liver.

What are the Morbid Changes ?

The enlargement of the spleen is due to increase of its fibrous tissue and proliferation of the endothelium of the blood sinuses, while the Malpighian corpuscles are atrophied. There may be perisplenitis following upon hæmorrhagic infarctions, and in the late stages hepatic cirrhosis is found. It is argued from the effects of splenectomy that the condition is a primary splenic disease, and is due to destruction of the blood corpuscles by the splenic endothelium.

Describe the Symptoms.

The onset is insidious, although the first appearance of symptoms may be sudden. The various signs of anæmia may be first apparent, but usually the patient suddenly discovers a "lump" in the side, or may be startled by a gastric hæmorrhage. On examination the spleen is found to be considerably enlarged, extending obliquely downwards from the left hypochondrium towards the umbilicus, and forming a tumour uniformly dull to percussion, with a smooth, flattened surface and rounded edges, the splenic notch being palpable in the lower epigastric region. It may be painful or tender on pressure if there is perisplenitis, in which case friction sounds may also be heard over it, but it is most commonly painless. The enlargement is progressive, and may reach a great size. The lymphatic glands are not enlarged.

The blood, when the disease is established, shows characteristic changes ; the red cells being reduced to 3,000,000 or less, the hæmoglobin to 50 per cent. or less, and the colour index low. The anæmia is thus chlorotic in type. There is also a persistent leucopenia. As the disease advances, hæmorrhages from mucous membranes may be added to the other symptoms of anæmia ; the liver becomes enlarged and cirrhotic, and ascites follows, with or without jaundice. The course is protracted, but death is the constant result.

What is the Treatment.

Improvement may be temporarily effected by iron and arsenic, used as in pernicious anæmia; and X-rays may be tried; but the operation of splenectomy is the only radical treatment. It is justified by a considerable number of successful results, and is best done in the early stages of the disease.

MOVABLE SPLEEN.

What are the Characteristics of this Condition?

It occasionally occurs as part of a general displacement of the abdominal viscera. The spleen, perhaps somewhat enlarged, descends into the abdominal cavity, and on palpation is found to be movable, and can be recognised by its shape and by the presence of the splenic notch. It may cause dragging pain in the left side. The only treatment needed is replacement by means of a suitable belt and pad.

Other diseases of the spleen—congestion, infarction, abscess, amyloid disease, syphilis, etc.—are secondary to disease of other organs and are referred to in their appropriate place.

(D). THE PITUITARY GLAND.

ACROMEGALY.

What is this Disease?

A chronic affection characterised by enlargement of the bones of the face and extremities, and by changes in the pituitary gland.

What are its Causes?

It begins in adolescence, or in the first half of adult life (up to the age of forty), and affects women somewhat oftener than men. It is due to changes in the anterior or glandular

portion of the pituitary body, one of the functions of which would seem to be regulation of skeletal growth. The changes found are either adenomatous or apparently sarcomatous, but many of the so-called sarcomata prove on closer examination to be adenomata, and acromegaly thus appears to be dependent upon hypersecretion of the anterior lobe. Changes in the thyroid gland, and persistence of the thymus, are sometimes associated with it.

Describe the Symptoms.

Languor, vague pains in the head and limbs, and in women amenorrhœa mark the onset. Headache usually becomes a prominent symptom, and there is some anæmia. The face, hands, and feet slowly enlarge, and when the disease is advanced the face is much broadened, especially in its lower part, the lower jaw-bone being particularly hypertrophied, so that the lower teeth may project beyond the upper. The teeth are separated from each other by the overgrowth of the jaws, the tongue is thickened and enlarged, the nose, the eyelids, and the lobes of the ears are thickened. The hands are very large and broad, the fingers thick, and the nails broad and coarse. The feet are similarly affected, but the long bones of the limbs do not suffer much change. The spine is generally involved, and marked kyphosis results.

Ocular changes (optic atrophy, bitemporal hemianopia) are common, there may be slow or defective cerebration, glycosuria and polyuria may occur, and there may be free sweating.

Medicinal treatment is unsuccessful, although either thyroid or pituitary extract may do temporary good. Partial removal of the pituitary body has been performed in a few cases, but no verdict can as yet be pronounced upon it.

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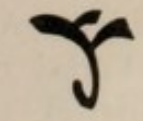

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CONTENTS

DISEASES OF THE ALIMENTARY TRACT.

MEDICINE

PART III.

DISEASES OF THE ALIMENTARY TRACT.

I.—THE MOUTH AND PHARYNX.

STOMATITIS.

What are the Causes of Inflammation of the Mouth?

It is sometimes a part of general diseases such as the specific infections; it occurs in scurvy and other disorders of the blood and in mercurial poisoning, and conditions of lowered vitality are predisposing causes. But most commonly stomatitis is of local origin, and is set up by organisms of various kinds—bacteria, moulds, or spirilla. The condition of *oral sepsis* which is set up by carious teeth or by pyorrhœa alveolaris is of much importance in its etiology.

What are its Forms?

Stomatitis may be catarrhal, aphthous or follicular, parasitic, ulcerative, or gangrenous.

Describe their Symptoms and Treatment.

Certain symptoms, namely, painful mastication, salivation, and fœtor of the breath are common to all forms of stomatitis, and may be slight or severe according to the particular form which is present.

Catarrhal stomatitis, due to local irritation (irritant poisons, bad teeth, etc.), to the spread of inflammation from other parts, to infective diseases (*e.g.* smallpox), or to

diseases of the blood, presents, besides the above symptoms, swelling and redness of the buccal mucosa, with enlargement and tenderness of the neighbouring lymphatic glands. There may be superficial ulceration. Mild antiseptic washes, and at a later stage astringents, with removal of the source of irritation, form the treatment.

Aphthous or follicular stomatitis is a trifling ailment, principally affecting children during dentition. It is characterized by the presence of small, circular, elevated gray patches due to a subepithelial exudation, and resembling vesicles in appearance. When the epithelium is shed, small greyish ulcers are left behind. A simple mouth-wash and the internal use of chlorate of potassium will remove the condition in a few days.

Parasitic stomatitis (*thrush*) is met with in weakly children and infants, and may occur in adults towards the end of exhausting diseases. It is associated with the presence of a fungus, the *oidium* or *saccharomyces albicans*, which is found in the epithelium of the affected patches. These are milk-white or greyish patches of irregular outline, which adhere to the mucous membranes of the mouth and pharynx, and are surrounded by a narrow zone of congestion. They consist of epithelial cells, fat globules, and the mycelium and spores of the fungus. In severe cases the patches may extend to the œsophagus and stomach. The mouth is dry and painful, and the general condition of debility is often associated with diarrhœa or gastric symptoms. In the later stages the patches become detachable, and leave superficial abrasions behind them. Treatment consists in attention to the general condition, local cleanliness, and the frequent application of a solution of borax (gr. x. to ℥i. – grm. 0·65 to 30 cc.).

Ulcerative stomatitis occurs in conditions of general ill-health, and most commonly attacks children after the second dentition. It also occurs in adults and may be locally

epidemic. The margins of the gums are swollen and ulcerated; and the ulcers, which are covered with a greyish membrane, may be deep and ragged, and may even expose the alveolus. The teeth become loose, there is profuse salivation, the submaxillary glands are enlarged, and there may be severe constitutional symptoms. The treatment consists in the internal use of chlorate of potassium (gr. x.-xx. – grm. 0·65-1·3 – , thrice daily according to age), and its local use as a mouth-wash (gr. x. to ℥i. – grm. 0·65 to 30 cc.). The strength must be maintained by nourishing fluid food.

Gangrenous stomatitis (*cancrum oris*, *noma*) is an uncommon affection met with in debilitated children, either as a consequence of bad hygiene, or as a sequela of infective diseases such as measles. It is of microbic origin, and probably due to a symbiosis of organisms in which the spirillum and bacillus of Vincent predominate. It begins in a patch of induration on the mucous membrane of the cheek, which goes on to necrosis, while the surrounding tissues become brawny and intensely inflamed. The slough separates, and the cheek may be perforated, or the gangrenous process may extend to the jaw or malar bone. There is little fever or pain, but prostration is rapid, and the child dies in the typhoid state. Treatment consists in excision of the gangrenous part, or, if this is not possible, free application of nitric acid or pure phenol, and in support of the patient's strength.

ACUTE TONSILLITIS.

What are the Causes of Acute Tonsillitis?

The disease may be a complication of many of the specific fevers, and it is also common as a primary affection. Primary acute tonsillitis is most common in adolescents and young adults, but may occur at any age, and is apt to be recurrent. It is frequently associated with acute rheumatism, of which it may be the first symptom. The organisms associated with

it are exceedingly various, but are most commonly the pyogenic cocci. The primary affection has two main forms, suppurative tonsillitis (*quinsy*) and follicular tonsillitis.

Describe the Symptoms.

The illness begins suddenly, with considerable constitutional disturbance and with sharp fever, often running up to 104° F. In the suppurative form there may be a rigor at the outset. Headache is common and severe, the tongue is furred, the breath offensive, and the glands at the angle of the jaw are enlarged and surrounded by diffuse tender swelling. The tonsils, one or both, are swollen and red, smooth and shining on the surface, which in the follicular form is dotted with yellowish spots of secretion protruding from the mouths of the follicles. In some cases the exudate is more extensive, and may have a whitish appearance like that of diphtheritic membrane. The fauces and soft palate are also swollen and congested, and swallowing and mastication are extremely painful. In suppurative tonsillitis an abscess forms in a few days, usually in one tonsil, sometimes in both, and fluctuation can be felt by the finger. If not evacuated it bursts into the throat, and recovery follows, although occasionally there may be a fatal issue from hæmorrhage or suffocation. The acute symptoms of follicular tonsillitis pass off in about a week.

How is Follicular Tonsillitis distinguished from Diphtheria?

In all doubtful cases cultures should be made at once. The presence of the *b. diphtheriæ* is the only certain criterion, and if there is doubt antitoxin should be given without waiting for the results of bacteriological examination. The exudate of tonsillitis never extends to the soft palate.

What is the Treatment of Acute Tonsillitis?

At the outset the bowels should be freely opened by a

mercurial purge followed by a saline. In follicular tonsillitis salicylate of soda (gr. x.-xv. – grm. 0·65-1·0) should be given internally every three or four hours, and astringent solutions such as glycerine of tannic acid may be used to paint the throat. Local application of a two per cent. solution of cocaine before food will relieve pain in swallowing, and external warmth often helps to alleviate pain. In quinsy the abscess, when it has formed, must be opened by a guarded knife. In both forms liquid diet is necessary, and quinine and iron in full doses may be given in the later stages to weakly individuals.

II.—THE ŒSOPHAGUS.

ŒSOPHAGITIS.

What are the Causes and Symptoms of Œsophagitis?

Acute œsophagitis may be due to the swallowing of hot fluids or corrosive poisons, may complicate the specific fevers, or may be caused by extension of inflammation from adjacent parts. Its symptoms are pain in the course of the œsophagus, aggravated by swelling, tenderness in its cervical part, and hawking of mucus. Rest to the inflamed part by giving a bland fluid diet, or, if necessary, by rectal feeding, is the main treatment.

Chronic œsophagitis may follow the acute form, or may be due to chronic alcoholism, cardiac valvular disease, or the pressure of a tumour. Its chief symptom, in chronic alcoholics, is the so-called "morning vomit," which consists mainly of the mucous secretion of the œsophagus.

STRICTURE OF THE ŒSOPHAGUS.

What are the Causes of this Condition?

It may be due to pressure from without, as by aneurysm or mediastinal tumour; to the cicatricial contraction following upon ulceration produced by corrosive poisons; to functional spasm; or to œsophageal tumours.

Describe the Symptoms of Cicatricial Stricture.

Dysphagia is the principal symptom, and it varies in degree with the amount of narrowing. When this is considerable solids cannot be swallowed, and even fluid food, if taken in quantity, may be regurgitated. If the stricture is high regurgitation occurs immediately after swallowing, if it is low there may be a brief interval. If the narrowing is extreme, so that even fluids pass only with difficulty, emaciation may ensue.

How do they differ from those of Œsophageal Spasm?

Spasmodic stricture, like cicatricial stricture, leads to dysphagia, but it occurs without obvious cause, and in young people, principally women, of a neurotic disposition. The onset is sudden, whereas in cicatricial stricture there is a gradually increasing difficulty in swallowing. In the spastic condition there is frequently burning pain in the chest, and the obstruction may suddenly disappear, to recur in a fresh attack.

What are the Symptoms of Tumour of the Œsophagus?

Œsophageal tumours, which are almost invariably cancerous, and of the type of squamous-celled epithelioma, form annular growths which are most common in the middle and lower thirds of the œsophagus, and especially opposite the bifurcation of the trachea. They infiltrate all the coats, and being constantly irritated by the passage of food, they are prone to rapid ulceration. They are most common in the male, between the ages of forty-five and sixty. The first symptom is an increasing difficulty of swallowing, and as the stricture becomes narrower, regurgitation of food sets in. If there is ulceration the food returned will be mixed with blood and mucus. Emaciation follows, and the patient assumes a cachectic appearance. The

cervical glands are often enlarged, and the growth may ultimately spread to the lung, causing broncho-pneumonia, may press upon the recurrent laryngeal nerves, or may cause metastases in other organs. Death occurs from starvation or from complications.

How are these Conditions to be Diagnosed?

The signs of dysphagia and regurgitation of food will, in the absence of evidence of aneurysm or other source of pressure on the œsophagus, point to the existence of stricture. Its site and its nature must be determined by further investigation. The passage of a bougie is contraindicated in the presence of aneurysm, mediastinal tumour, or cardiac valvular disease. Where these are present an X-ray examination after a bismuth meal will indicate the site of the obstruction, and the presence of a tumour can often be made out by the œsophagoscope, and sometimes even by the laryngoscope. If it is decided to use the bougie where cancer is suspected, it must be very gently done, as the softened tumour tissue may be perforated by undue force.

What is their Treatment?

A cicatricial stricture should be treated by the repeated passage of sounds increasing in size; if it is impermeable gastrostomy may be needed. A spasmodic stricture may be overcome by the passage of full-sized instruments, and by attention to the neurotic condition. A malignant stricture may be treated by the bougie, by intubation, or by gastrostomy, but all methods of treatment are merely palliative.

III.—THE STOMACH.

ACUTE GASTRITIS.

Mention the Forms of Acute Gastritis.

Acute gastritis may be catarrhal (the usual form), phlegmonous, toxic, or infective. In the phlegmonous form, which is rare, the inflammation goes on to suppuration, and the condition is usually fatal. Toxic gastritis is an intense inflammation due to the ingestion of irritant poisons or mineral acids. It is often fatal, and if recovery occurs it may leave a chronic catarrh behind. Infective gastritis occurs in the course of certain specific infections; in diphtheria, for example, a false membrane may form in the stomach, in smallpox a pustular eruption, while pneumococcal and other microbic infections of the stomach are also known. The symptoms are obscured by those of the causative disease.

What are the Causes of Acute Catarrhal Gastritis?

It is most commonly due to the use of over-rich or indigestible food, or of food which is not absolutely fresh. Unripe or over-ripe fruit, decomposed tinned meats, bad fish, shell-fish, and similar articles may therefore cause it; and as heat favours the bacterial decomposition of food, gastritis is commonest in warm weather. Excessive indulgence in strong spirits may also produce it, and infants unsuitably fed are very liable to it. It is predisposed to by a condition of chronic catarrh with fermentative changes and an excess of organic acids.

What are the Morbid Changes?

Those of catarrhal inflammation in general—hyperæmia and increased secretion of mucus, with cloudy swelling of the glandular epithelium. In severe cases there may be minute hæmorrhage from the engorged vessels.

Describe the Symptoms.

In the most intense cases the onset is sudden, with acute epigastric pain and diffuse epigastric tenderness, repeated vomiting of mucus, and symptoms of collapse. If due to irritant poisons, such cases may be fatal. In the usual form there is either a sense of oppression or pain in the epigastrium, with some tenderness, and nausea or vomiting. The vomited matter, after the food in the stomach has been got rid of, is watery or bile-stained; and hydrochloric acid is defective or absent. The tongue is furred, there is anorexia, thirst and headache are present, and there may be fever. Diarrhœa is common in children. The symptoms pass off in a week or more.

How would you treat it?

In severe attacks the stomach should be washed out at the start with a weak alkaline solution, and no food should be given for the first twenty-four hours. Thereafter small quantities of milk and soda-water may be given, and the return to solid food must be carefully graduated. The bowels should be opened by a dose of calomel, if it can be retained, and pain may be relieved by fomentations, or, if necessary, by morphia. If thirst is severe, ice may be sucked in small quantities.

CHRONIC GASTRITIS.

How does this Condition arise?

It may follow upon an acute attack or repeated acute attacks, but it is more often the result of continued irritation of the stomach. Constant indulgence in irritating foods or constant excess in the amount of food is a frequent cause, as is also excess in alcohol. It accompanies many conditions of organic disease of the stomach—carcinoma, chronic ulcer, dilatation, and the passive congestion due to cardiac or hepatic disease.

What are the Morbid Changes?

They vary with the duration of the disease. The glandular epithelium undergoes degeneration, and in late stages the mucosa may be completely atrophied. The connective tissue between the glands undergoes fibrosis, and its contraction causes the formation of intra-glandular cysts, which give rise to small projections upon the surface, especially in the pyloric region. The submucous and intermuscular tissue may also be fibrosed, and in old cases the gastric wall is thickened and the stomach as a whole may be shrunken (*cirrhosis*). Hæmorrhagic erosions of the mucous membrane may occur.

Describe the Symptoms.

The patient complains of defective or variable appetite, a feeling of weight or distension in the stomach, aggravated after meals and sometimes amounting to pain, nausea, and intermittent vomiting, which in the case of alcoholics is most common in the morning. There is some degree of diffuse tenderness in the epigastrium. The tongue is furred, thirst is often marked, and constipation and flatulence are common. The vomited matters contain much mucus, and are defective or lacking in hydrochloric acid, though organic acids may be in excess. The symptoms vary in intensity from time to time, and may be interrupted by acute attacks. Fever is seldom present, but some wasting is frequent.

What is the Treatment?

Mainly dietetic. It may be well to begin by washing out the stomach. Thereafter only the very simplest diet should be allowed, and at first only milk and milk foods. Alcohol, tea, and coffee, must be stopped. Fish, chicken, beef and mutton must be added very gradually to the diet. Constipation should be attended to, and pain may be relieved by

bismuth or dilute hydrocyanic acid. Mineral acids after meals along with nux vomica or strychnine replace the defective hydrochloric acid and increase the tone of the gastric musculature. The treatment is largely palliative, as the lesions of chronic gastric catarrh are permanent.

GASTRIC ULCER.

What are the Forms of Gastric Ulcer?

An ulcer of the stomach may be acute or chronic. Slight erosions (hæmorrhagic erosions) may occur in connection with gastric catarrh, or may be due to congestion of the gastric venous circulation. The typical ulcer is known as the peptic, round, or perforating ulcer, and it may be single or less commonly multiple.

What are its Causes?

Gastric ulcer is most common in young women, in whom it is often associated with anæmia. Sedentary occupations and lack of sunlight may possibly also predispose to it; at all events it is frequent in domestic servants. In men it is most common between the ages of thirty and fifty, in women between those of twenty and thirty-five. Multiple ulcers may be found in various septic conditions, and also, along with duodenal ulcers, after extensive burns.

The initial lesion preceding the formation of an acute ulcer may be, according to Bolton, a local necrosis of the mucous membrane, local hæmorrhage into it, or local inflammation of the gastric lymphatic follicles. Such conditions may be produced by bacterial infection, cutaneous burns (rarely), irritant substances introduced by the mouth (acute ulcer may follow gastritis), venous congestion, and endogenous poisons of metabolic origin (gastrotoxins). The gastric juice does not attack the healthy wall of the stomach, but acts upon such devitalised areas, and all the more rapidly if its

acidity is high. Hyperchlorhydria is therefore a predisposing cause. Some acute ulcers heal very rapidly, others extend either in depth or laterally, or in both directions. The more rapid the extension the less is the inflammatory thickening of its edges and base; the less rapid the greater is the thickening. Acute ulcers thus merge into chronic ulcers, and these latter arise out of the acute variety.

Describe the Morbid Appearances.

The ulcers vary both in their character and in their site. The most frequent sites of solitary ulcers are on the posterior wall, near the lesser curvature and near the pylorus. Ulcers on the anterior wall, though less common, are very apt to end in perforation. The acute ulcer is rounded, about half an inch or rather more in diameter, punched out, with sharp edges, which are soft, not thickened or undermined, and somewhat funnel-shaped. The base may consist of the submucous, muscular, or serous coat, according to the depth of the ulcer. The chronic ulcer, which is often much larger, has firm, thickened edges, and as the lateral extension is greatest at the surface, the edges have a terraced appearance, and the ulcer narrows towards the base, which may be formed of any of the coats. The serous coat is often thickened, and may adhere to adjacent organs or parts. Where no such adhesions exist perforation may occur, and this leads either to a generalised peritonitis or to a local *subphrenic abscess.* When adhesions have formed the ulcerative process extends into the adherent organ (liver, pancreas), and may largely destroy its substance. A subphrenic abscess may ultimately perforate into the pleura or pericardium, or into the duodenum or colon.

Should the ulcer heal there may be but little contraction if it has involved only the mucous membrane, but if the deeper structures are involved, the cicatricial contraction may lead to pyloric obstruction and hence to dilatation of

the stomach, or if the ulcer is remote from the pylorus, to hour-glass contraction of the stomach.

What are the Symptoms?

They are extremely variable. In a typical case of chronic ulcer the symptoms are as follows:—(*a*) *Pain* after meals, coming on at varying intervals and often increasing in severity until relieved by vomiting. The pain is seated in the epigastrium, sharply localised, and usually in the middle line, or slightly to the right or left of it. It is intense and often radiates to the back, about the level of the eighth dorsal to second lumbar vertebra. Occasionally, when the onset of pain is much delayed, it may seem to occur rather before the following meal than after the previous one. (*b*) *Tenderness* on pressure, generally in the same situation as the pain, and sometimes accompanied by hyperæsthesia of the skin over the tender area. (*c*) Contraction of the upper belly of the left rectus, if the pain is severe. (*d*) *Vomiting*, coming on after meals, and usually when pain has been present for some time. It therefore appears half an hour or more after food, and gives relief from the pain. The vomited matters consist of the food taken at the last meal, and generally contain an excess of hydrochloric acid, while blood may be present in small quantity. (*e*) *Hæmatemesis*, however, though a frequent but not a necessary accompaniment of ulcer, when it occurs is often due rather to the opening of a considerable vessel than to capillary oozing. Hence it is common to find large hæmorrhages occurring at irregular intervals, and causing profound or sometimes even fatal anæmia. Such a hæmorrhage may occur only once or may be repeated several times. Blood is often passed by the bowel (*melæna*), imparting a black or tarry appearance to the stools.

Appetite is not much interfered with and may be quite good, but the patient fears to eat because of the pain that

follows. The tongue is clean, unless in old-standing cases associated with gastric catarrh. In such cases the symptoms may be chiefly those of chronic gastritis.

In acute ulcer the symptoms sometimes resemble those of chronic ulcer, but in many instances there may be little indication of the presence of an ulcer until a severe hæmorrhage takes place, or until perforation occurs.

What are the Symptoms of Perforation?

They come on with extreme suddenness, usually after a meal or during the strain of exertion or vomiting. Violent pain in the epigastrium, radiating into the shoulders, rigidity of the abdominal wall, thoracic breathing, rapid wiry pulse, and a pinched anxious expression of face are the chief symptoms. The abdomen becomes rapidly distended, and the hepatic and splenic dulness may be obliterated by the presence of gas in its cavity. These are the early signs of perforative peritonitis; at a later stage there may be evidence of fluid in the flanks.

Mention the Main Complications.

Hæmorrhage, perforation, and subphrenic abscess. The last follows upon perforation into the lesser peritoneum, and leads to displacement of the adjacent organs (heart, liver), and to bulging of the epigastric region. It may be followed, as already mentioned, by pulmonary complications. Gastro-duodenal fistula may also occur, but rarely. Dilatation of the stomach often results from the cicatricial contraction of an ulcer at the pylorus.

How would you treat such a Case?

The first essential is rest in bed, which must be continued for at least a month. Diet must be very restricted, and if the symptoms have been at all severe, the stomach must be

left empty, and nourishment must be given by the rectum for the first few days. When oral feeding is permissible, milk, or milk and lime-water, should be given, at first in quantities of not more than two ounces every two hours. Only such quantities as produce no pain or vomiting are allowed, and they must be very gradually increased until the patient is taking two to three pints a day. Milk foods may then be added, and afterwards pounded fish or chicken, any return of pain demanding a return to more stringent diet. Vegetables should be avoided.

Pain may be relieved by bismuth, bicarbonate of soda, and powdered opium, given after food, or if necessary by morphia hypodermically, and heat may be applied over the epigastrium. Vomiting may be treated by large doses of bismuth; hæmorrhage by absolute rest and withdrawal of all food, and the application of ice to the epigastrium. If the hæmorrhage continues a full dose of morphia should be given hypodermically, whilst adrenalin (♏ xx-xxx, – 1·2 – 1·8 cc.), calcium lactate, or astringents may be orally administered. Horse serum (10 cc.) may also be given by the mouth, and continued for several days. Saline infusion, rectally or subcutaneously, may be needed to combat the asthenia following hæmorrhage.

Perforation and subphrenic abscess must be dealt with surgically, as also must dilatation of the stomach following cicatrisation. In the case of repeated hæmorrhages, or of old-standing ulcer which does not yield to treatment, gastro-enterostomy is advisable.

CANCER OF THE STOMACH.

Mention the Varieties of Cancer occurring in the Stomach.

1. Squamous-celled epithelioma; at the cardiac end and due to extension from the œsophageal epithelium. This form is comparatively rare.

2. Adenocarcinoma, spheroidal-celled or cylinder-celled, originating from the epithelium of the gastric glands. This variety may be either *medullary* or *scirrhous*; in the former case the cellular element is abundant, in the latter the stroma. Scirrhous cancer, often of an infiltrating type, is most common in the pyloric region.

3. Colloid, or rather mucoid, cancer, in which the cells undergo a mucinoid transformation and the alveoli are filled with inspissated mucin. This form, which is less common, has a markedly infiltrating tendency.

What are its Causes?

Those of cancer in general. The disease is most common between the ages of forty and sixty, and is somewhat more common in males than females. It affects all classes, and those engaged in the most various occupations. In some instances it is a sequela of ulcer of the stomach, but it has no relation to other gastric diseases, and often occurs in previously healthy subjects.

Describe the Morbid Changes.

The most frequent seat of cancer is at the pylorus, and after that the lesser curvature, but it may affect any part of the stomach. It begins in the glands of the mucous membrane, and spreads thence to the submucous and muscular coats, leading to considerable thickening, and at the pylorus to narrowing of the lumen. Thereafter the serous coat and the lymphatics are affected, and cancerous nodules may be seen upon the peritoneal surface. In the case of scirrhous and of colloid cancer, the tendency is to a lateral spread, the various gastric coats being widely infiltrated; in the medullary form the tumour protrudes more definitely into the lumen of the stomach, and is liable to ulceration from the passage of food over it. Such

ulceration frequently leads to hæmorrhage, and may give rise to perforation, although this is usually prevented by the formation of adhesions to adjacent organs. Dilatation of the stomach is very apt to follow the obstruction caused by a pyloric cancer, while a growth at the cardiac end may cause dilatation of the œsophagus. The contraction of a scirrhous cancer may lead to diminution in the size of the stomach as a whole (cirrhosis). Metastases in adjacent organs (liver) are frequent, and a chronic malignant peritonitis is not uncommon.

What are the Symptoms ?

The onset is gradual, and the symptoms are at first those of dyspepsia only. *Pain*, however, soon becomes persistent, and though it is aggravated by meals, it does not disappear in the intervals, nor does vomiting give much relief. *Vomiting* is less frequently repeated than in ulcer, and occurs at irregular intervals. The vomited matters often contain blood in small quantity, due to the erosion of small vessels, but large hæmorrhages are rare. As the blood has lain for some time in the stomach, it is altered by the gastric juice, and presents the dark brown appearance of "coffee-grounds." If the quantity is very minute it may be unrecognisable by the naked eye. The "occult blood" tests (guaiacum or benzidine) must then be used, with the precaution that no meat must be eaten for at least 48 hours previous to their employment. *Anorexia* is an important symptom, and usually appears early. *Wasting* is also rapid, and along with it goes an alteration of the complexion, which becomes anæmic, sallow and cachectic, the appearance sometimes resembling that of pernicious anæmia. Sooner or later a *tumour* is recognisable in the epigastrium in the majority of cases, but it may be absent in as many as 20 per cent. When present, it is firm, resistant, somewhat rounded, nodular, and tender; if adherent to the liver, it moves

with respiration. It lies usually slightly to the right of the middle line, and between the xiphoid cartilage and the umbilicus.

Analysis of the gastric juice withdrawn after a test meal reveals deficiency or absence of free hydrochloric acid and excess of organic acids, particularly lactic. When the patient has been kept for 24 hours upon a protein-free diet, the stomach contents still give a precipitate with Esbach's reagent for albumin, even in a dilution of 1 in 200 (Salomon's test). On miscroscopic examination, very long rod-shaped Gram-positive bacilli (Boas-Oppler bacilli), often forming long chains, are frequently to be found.

If there is marked pyloric obstruction, there may be evidence of dilatation of the stomach. If metastases have occurred, the liver may be enlarged, tender, and nodular; there may be indications of chronic peritonitis; or there may be enlargement of the cervical glands. The spleen is seldom implicated.

Death is usually due to exhaustion, sometimes to hæmorrhage; and it occurs within two years of the onset.

How would you treat such a Case?

In many cases treatment resolves itself into the relief of pain and the maintenance of strength. The same measures as in gastric ulcer may be taken for pain and vomiting; if the stomach is dilated, it should be washed out daily with a weak alkaline solution, and carbolic acid or creosote may be given to prevent bacterial fermentation. The food should be as nourishing as the state of the stomach will allow. Radical operation (pylorectomy) is seldom possible, as the disease is usually advanced before it is recognised; but gastro-enterostomy in cases with severe symptoms often gives temporary relief. The question in all cases is whether such operation will prolong life; many patients appear to sink more rapidly after operation than if they had been left alone.

DILATATION OF THE STOMACH.

How is this Condition produced?

It may be due to weakness of the muscular walls of the stomach—atonic dilatation; or to obstruction to the passage of food through the pyloric orifice.

Atonic dilatation is most commonly found in conditions of constitutional weakness (anæmia, febrile diseases), as the result of chronic gastritis, and in cases of habitual over-distension of the stomach by food.

Obstructive dilatation may be due to causes within the stomach—stenosis of the pylorus from cancer, cicatrising ulcer, or more rarely from muscular hypertrophy; or to causes external to the stomach—post-ulcerative stricture of the duodenum, pressure from without (displaced kidney, etc.), or peritoneal bands.

What are its Symptoms?

They are of gradual onset. Pain or discomfort in the epigastrium, occurring at variable intervals after meals, and increasing as the stomach becomes more distended with each successive meal, leads ultimately to vomiting, which may occur only at night, or only every two or three days. The pain, which is diffused over the epigastrium, is of a burning character (*pyrosis*); flatulence is frequently present, and regurgitation of the sour, watery, gastric contents into the mouth (*waterbrash*) is common. The vomited matter is larger in quantity than the normal content of the stomach, frothy, and exceedingly sour in taste from the presence of organic acids. Free hydrochloric acid is defective, and sarcina ventriculi, torula cerevisiæ (the yeast fungus), and bacillus acidi lactici may be found under the microscope. Fragments of partially digested food are also found.

The general symptoms are wasting, anæmia, debility, and constipation. They are most marked when the dilatation is due to malignant disease.

Describe the Physical Signs.

On inspection, the epigastrium may be seen to bulge, especially on the left side. The outline of the greater curvature can often be made out, at or below the umbilicus, and if the stomach is displaced downwards, the lesser curvature can also be seen. A sharp stroke of the finger nail often produces a peristaltic movement from left to right. Percussion or auscultatory percussion may show that the gastric note extends below the umbilicus. Its delimitation may be aided by distending the stomach with air passed in through a stomach tube, or fluid may be given and the outline (dull note) of the greater curvature determined while the patient sits up. Shaking the abdomen from side to side causes a splashing sound (*succussion*), due to the mixture of fluid and gas. This is only of value if it occurs when the stomach should normally be empty, *i.e.*, more than five hours after a meal. X-ray examination, after a meal of bismuth porridge, gives an accurate idea of the size and position of the stomach.

What is the Treatment?

The stomach should be washed out daily with a 1 or 2 per cent. solution of bicarbonate of soda, the washing being continued until the fluid returns clear. The diet must be carefully regulated, carbohydrates being avoided as they tend to induce fermentation, while the meals should be small and the amount of fluid limited. In atonic cases the general condition must be treated, iron, strychnine and other tonics being indicated. Constipation must also be relieved.

In obstructive dilatation these measures are preliminary to operation, previous to which the patient's health should be as far as possible improved. Pylorectomy or gastro-enterostomy may be done according to the condition present.

DYSPEPSIA.

What is Dyspepsia?

Disorder of the gastric functions without organic disease of the stomach.

What are its Causes?

In many instances so-called dyspepsia is merely a symptom of organic disease in other organs or parts of the body (*e.g.*, anæmia, tuberculosis, chronic nephritis, cardiac dilatation). But when it exists apart from these it may be caused (*a*) by the transitory irritation of an excessive or over-irritating meal (acute indigestion), when the symptoms are also transitory and akin to those of a mild gastritis; or (*b*) by more chronic irritation (chronic indigestion, chronic dyspepsia). Such irritation may be produced by habitual over-eating or over-drinking, whether the beverage be alcohol, tea or coffee; by habitual excess in condiments or the use of highly-spiced foods; by imperfect mastication resulting from bad teeth; by the abuse of tobacco; and by mental overstrain or worry. There is frequently a distinct nervous predisposition, and many of the anomalies of gastric secretion or motility are essentially of neurotic origin.

Mention the Forms of Dyspepsia.

There is no agreement in nomenclature. The older classification spoke of acid, atonic, and nervous dyspepsia, but many of the cases of the two former groups are now recognised as early stages of organic disease of the stomach, or as symptomatic of general diseases such as anæmia. The nervous dyspepsias are the best defined class, and they may cause disorder of motility, sensation, or secretion.

What are the Symptoms of these Disorders?

1. Motor neuroses may give rise to excessive motility, shown in increased peristalsis and gastric or intestinal gur-

gling, eructations, and sometimes painful spasm of the cardiac orifice ; or to atony of the muscular coat leading to moderate dilatation of the stomach.

2. Sensory neuroses are represented by *gastralgia* or *gastrodynia*. This is most frequent in women, and epigastric pain is the chief symptom. The pain is often severe and may radiate to the back, but it is relieved rather than aggravated by food, and firm pressure also relieves it. Constipation, anæmia, and a neurasthenic disposition are the accompanying symptoms.

3. Secretory neuroses may take the form of hyperacidity or excessive secretion on the one hand, and of diminished acidity or absent secretion on the other. The symptoms of hyperacidity (*hyperchlorhydria*) are pain of a burning character coming on an hour or two after meals, sour eructations, and occasional vomiting of food mixed with highly acid gastric juice. The appetite is usually unimpaired. Excessive secretion (gastrosuccorrhœa) which is not common, is usually manifested by severe paroxysms of gastric pain, occurring at night, and leading to the vomiting of large quantities of acid gastric juice unmixed with food.

Diminished acidity (*hypochlorhydria*) is often a symptom of organic disease of the stomach, or of constitutional disease. It is also met with towards the middle period of life in those of sedentary habits, and especially in women. A sense of weight after meals, due to flatulent distension, eructations, constipation, headache, and a broad, pale and flabby tongue are the chief symptoms. If muscular atony is also present dilatation of the stomach may follow.

Achylia gastrica is a rare condition. The gastric secretion may be absent as a result of neurosis, when digestion is carried out entirely in the intestine and there may be no symptoms, or in the late stages of gastric catarrh, of which the characteristic symptoms are present.

How would you treat a Case of Dyspepsia?

By attention, in the first place, to the causes. Any constitutional condition should be appropriately dealt with; defective teeth should be attended to; faults of hasty eating, insufficient mastication, over-eating, or over-drinking must be corrected; and a suitable dietary must be prescribed. The meals must be regular and moderate in quantity, and highly-seasoned or indigestible foods (pork, pastry, game, etc.), must be forbidden. Alcohol ought not to be taken, nor should coffee, and even tea should be very sparingly used. Tobacco must also be restricted or forbidden. Where there is severe pain the diet may at first consist of milk, or milk and fish; where there is much tendency to flatulence and atony farinaceous foods should be avoided. In cases dependent on nervous conditions change of air is useful, and complete rest is often necessary; in other cases moderate daily exercise is advisable. Abdominal massage is often of benefit in atonic cases, both for its effect on the gastric muscle and because it promotes the action of the bowels. Constipation must be carefully corrected. Medicinal treatment must be directed both to the general and the local condition. Tonics such as nux vomica or hæmatinics such as iron and arsenic are indicated in many cases. Gastric pain due to hyperacidity may be met by alkalies after meals, to which, if necessary, opium may be added. Opium or morphia is also frequently required in gastralgia. In diminished acidity alkalies may be given before meals to stimulate the flow of gastric juice, or mineral acids may be given after meals. Flatulence may be met by bismuth, carminatives, or bitter tonics.

IV.—THE INTESTINES.

ENTERITIS.

What is Enteritis?

Inflammation of the intestinal mucous membrane.

Under what Conditions may it occur?

It is frequent as a complication of the acute infections, and in the terminal stages of chronic constitutional diseases such as anæmia and Bright's disease. It may also complicate portal cirrhosis and chronic disease of the heart. The affection clinically known as enteritis, however, arises independently of these conditions, and is due to causes acting by direct irritation of the intestinal mucosa, such as indigestible food, or to general causes such as extremes of temperature.

What are its Forms?

Enteritis may be catarrhal, diphtheritic, phlegmonous, or ulcerative. The first is much the most common.

What are the Causes of Catarrhal Enteritis?

It is most frequent in summer, and this is principally due not to the heat itself but to the readiness with which food decomposes in hot weather, the organisms ingested along with it contributing to the enteritis. This is especially the case with milk, and therefore enteritis is very common in children in hot weather, milk being readily contaminated both by exposure to the air and by organisms conveyed by flies. A sudden change from heat to cold may also produce it. Further causes are the abuse of strong spirits, violent purgation, and mineral poisons such as arsenic and antimony.

Describe the Morbid Changes?

They are those of catarrhal inflammation in general, hyperæmia and round-celled infiltration of the mucous membrane, increased glandular secretion, cloudy swelling of the glandular epithelium, and enlargement of the solitary follicles. These changes are most marked in the small intestine in acute cases. In chronic enteritis the mucous membrane is thickened and pigmented, or in later stages it may be atrophic. Abrasions of the solitary follicles which may have existed in the acute stage, extend in chronic enteritis so as to form follicular ulcers. There may be polypoid outgrowths from the mucous membrane.

Describe the Symptoms.

Diarrhœa is the most prominent symptom, and is due both to increased secretion and to more vigorous peristalsis. The stools vary in character, being at first brownish, watery, and copious, while later they are paler, yellowish or greenish, and contain much slimy mucus. If the catarrhal secretion has blocked the orifice of the bile-duct the stools will be clay-coloured, and jaundice may follow. Under the microscope various organisms are to be seen along with fragments of undigested food, starch granules, fat, intestinal epithelium, and crystals of triple phosphates. The stools vary in number from three to ten or more per diem.

Abdominal *pain*, colicky and paroxysmal in character, is usually present, and its principal seat is in the neighbourhood of the umbilicus. In the severer cases there may be moderate *fever*. If the stomach is affected at the same time nausea, vomiting, and gastric pain are present. There is no tenderness on abdominal examination, but palpation may elicit gurgling noises in the course of the bowel. General symptoms may be slight or marked, the tongue is furred, the appetite poor or absent, thirst is often troublesome, and the urine may be scanty from loss of water by the bowel.

The duration of an acute attack is a few days, but the disease may become *chronic*, in which case there are several loose motions daily, often much mixed with mucus, and there are occasional attacks of griping pain. Wasting and anæmia are also present.

What is the Treatment?

Rest in bed, warmth to the abdomen, and milk diet. When the attack is due to irritating articles of food a laxative such as castor oil and laudanum, or calomel, may be given at the outset to clear the bowel; but if there has already been much diarrhœa this is unnecessary. Bismuth in large doses, bismuth and opium, or starch and laudanum enemata, may be given to check the diarrhœa; morphia hypodermically may be necessary for the pain. Similar measures are useful in chronic enteritis, with the addition of intestinal antiseptics such as salol, and in obstinate cases lavage of the bowel. The stools must be carefully examined to exclude dysentery.

What are the Special Features of Enteritis in Children?

The disease is caused principally by improper food, and when it occurs in breast-fed children is due to unduly frequent meals. It is most common in hot weather from contamination of the milk, and any debilitating disease (*e.g.* rickets), predisposes to it. Young children who, in the matter of food, are allowed "the run of the house" are also prone to suffer. Although in mild cases diarrhœa, characterised by the passage of frequent greenish and offensive stools, may be the chief symptom, in the severer forms the stomach is affected along with the intestine. Vomiting is common, and the milk is returned in large curds; there is abdominal pain, and the abdomen is distended by flatus and sometimes tender; the stools, still greenish and offensive,

are more liquid and much mixed with mucus; the child wastes rapidly, and there may be smart fever. Death may occur from collapse, though most cases recover. In the chronic form the stools are large, pale, semi-fluid, and offensive, the abdomen is distended, and death from exhaustion occurs after an illness of weeks or months.

Treatment consists mainly in regulation of the diet. For children on the breast the meals should be reduced in frequency, and the bowels should be cleared by a dose of castor oil. When the mother cannot suckle the child a wet-nurse should be obtained, if possible; in other cases, the child must be bottle-fed, the milk being suitably diluted in proportion to the age, with the addition of a proportionate amount of sugar. Lime-water is a better diluent than water, as it tends to prevent the formation of large curds. In many cases of acute enteritis milk is not tolerated, and for the first few days albumin-water or raw beef-juice must be given in place of it. Warm applications to the abdomen should be used, and the diarrhœa may be checked by divided doses of calomel, bismuth, or pulv. cretæ aromaticus. Dover's powder may help the action of bismuth, but should be given very carefully to children, and only in exceptional cases to those less than one year old. When there is much collapse alcohol may be needed.

What is meant by Cholera Nostras?

A form of catarrhal enteritis due to food-poisoning, and associated with the presence of such organisms as the bacillus enteritidis, b. proteus, and b. coli communis. It is commonest in hot weather, and is characterised by the sudden onset of choleraic diarrhœa, with rice-water stools and muscular cramps. The diagnosis depends upon bacteriological examination, and the treatment is similar to that of cholera. The term cholera nostras is now virtually obsolete.

What are the Characters of Diphtheritic and Phlegmonous Enteritis?

Diphtheritic or croupous enteritis is not an independent affection, but may complicate such diseases as pneumonia, enteric fever, cirrhosis of the liver, and the late stages of chronic Bright's disease. Patches of membrane are found on the free edges of the valvulæ conniventes, and the mucous membrane is intensely injected. The condition is usually fatal. *Phlegmonous* enteritis is the result of local interference with the circulation in the bowel (strangulated hernia, intussusception), or of intestinal obstruction. It is a local affection involving all the coats of the bowel, and its symptoms are chiefly those of localised peritonitis, plus the symptoms proper to the causative disease. The treatment is mainly surgical, and is that of the cause.

COLITIS.

What are the chief Varieties of Colitis?

Catarrhal, mucous or membranous, and ulcerative. Catarrhal colitis, most common in children, is usually associated with enteritis, and calls for similar treatment.

Describe a Case of Mucous Colitis.

This condition occurs chiefly in women of a nervous disposition, and particularly about middle age, but it is also seen in children. It is characterised by the intermittent passage of casts of the bowel, consisting chiefly of mucus. These evacuations are preceded by severe abdominal pain, while in the intervals the bowels are constipated, and the patients usually suffer from anæmia, emaciation, and dyspeptic symptoms. The casts, which are transparent, gelatinous, and of a yellow colour from fæcal staining, may be a few inches or even several feet long. The disease is not fatal *per se*, although it is extremely obstinate.

How would you treat such a Case?

By fresh air and regular exercise, a nutritious but non-irritating diet, occasional laxatives, and attention to the nervous predisposition. Lavage of the bowel is often useful, and in very obstinate cases appendicostomy with lavage through the opening has proved successful.

What are the Characters of Ulcerative Colitis?

It is a condition which may arise as a terminal event in cases of chronic nephritis or septic infection; but it may also occur without any obvious cause. It is most common in middle life. The colon as a whole is injected and inflamed, and its mucous membrane swollen, while numerous irregular ulcers with overhanging edges are to be found in it. Their surface may be covered by a slough; they may tend to run into one another; and here and there partial cicatrisation may be found.

The *symptoms* are diarrhœa with griping abdominal pain, and intestinal hæmorrhage. The stools are fluid and dark, without much mucus, and they are often mixed with bright red blood, and sometimes with shreds of sloughs. Emaciation occurs, the complexion is sallow, and there is irregular fever. Flatulence and abdominal distension are common, and death is due to exhaustion, hæmorrhage, or more rarely perforation.

Treatment includes rest in bed, warmth to the abdomen, bland fluid diet, and the use of astringents. Lavage of the bowel *per rectum* with astringent injections (nitrate of silver, argyrol, etc.) may be successful; if it fails, irrigation through the appendix may be tried.

APPENDICITIS.

What are the Causes of Appendicitis?

Inflammation of the appendix is of microbic origin, and is usually set up by the *bacillus coli*, although other organisms

inhabiting the bowel may also give rise to it. Unless these are exceptionally virulent, however, they do not cause inflammation to the undamaged appendix, but only in the presence of a contributory cause, such as blocking of the lumen by catarrhal secretion, and the retention of fæcal concretions or less commonly of foreign bodies such as grape-seeds. The onset is favoured by chill or overstrain, and appendicitis is an occasional complication of acute infections. It is commonest in young adults.

Mention its chief Varieties.

Appendicitis may be catarrhal or obliterative, ulcerative, or gangrenous.

What morbid Changes may they cause?

Catarrhal or obliterative appendicitis is attended by a merely local peritonitis, and passes off rapidly; but it is liable to recur or to become chronic, with the result that adhesions form between appendix, cœcum, and small intestine, or that the lumen becomes partially or entirely obliterated. Partial obliteration leads to the formation of cysts, due to retained secretion, and these may rupture into the abdominal cavity.

Ulcerative appendicitis, if the ulceration is deep, ends in perforation; and gangrenous appendicitis, resulting in sloughing, also leads to perforation near the tip.

The consequences of perforation depend mainly upon the presence or absence of adhesions. If these have formed before perforation takes place, the result is usually a localised peritonitis with intra-peritoneal abscess. The abscess may, however, be extra-peritoneal, if the perforation has occurred between the folds of the appendicular mesentery.

If adhesions have not formed, a general septic peritonitis follows perforation. This is commonest in gangrenous ap-

pendicitis, but even a localised appendicular abscess may at a later stage burst into the peritoneal cavity.

Describe the Symptoms of Appendicitis.

The onset is sudden, and is attended with severe abdominal pain, which may be diffuse at first but soon becomes localised to the right iliac fossa. There is marked tenderness, which is greatest at M'Burney's point (midway between the umbilicus and the anterior superior iliac spine), and is accompanied by rigidity of the right rectus, and often by slight flexion of the right thigh. Vomiting is often present at the outset. Along with these local symptoms there are fever, rapid pulse, furred tongue, anorexia, and constipation. After a day or two some resistance is usually perceptible in the iliac fossa.

In an attack of catarrhal appendicitis, after fever has persisted and resistance has increased for three or four days, the symptoms begin gradually to subside; fever diminishes, tenderness lessens, and in about a week the patient is convalescent.

In other cases tenderness increases, fever persists or increases and may become remittent in character, while the resistance in the iliac fossa becomes more extensive and harder, until a definite tumour may be felt. This may subside in about a fortnight or three weeks or may go on to suppuration, with rigors, sweating, and signs of septic absorption. Leucocytosis is present. The abscess may rupture into the bowel, with subsequent recovery, or into the peritoneum, causing generalised peritonitis.

If perforation takes place, as in ulcerative or gangrenous appendicitis, a rapidly fatal septic peritonitis is the result (for symptoms see *Peritonitis*).

The attacks may be often repeated, and each recurrence increases the density of the adhesions and favours obliteration of the appendix; but until obliteration has occurred abscess may form, or perforation may take place in any attack.

Are there any Difficulties in Diagnosis?

Almost any acute abdominal pain may be mistaken for appendicitis, but attention to the locality of the tenderness and resistance, and to the presence of fever, should avert mistakes in most instances, while the history and the presence of hepatic and urinary symptoms will help to distinguish biliary or renal colic. The referred pain of early pneumonia of the right side has often led to error, but in such cases there is no local tenderness or rigidity, and there is an altered pulse-respiration ratio with, it may be, rusty sputum or fine crepitus at the right base.

What is the Treatment of Appendicitis?

Operation should be performed as soon as the diagnosis is made. If the case has reached the stage of abscess, or if general peritonitis is present, operation is obviously necessary, although in the latter case the prospect of recovery is not great; but even in apparently simple cases, which in the majority of instances recover without interference, it is impossible to foresee that a perforation will not occur, and immediate operation avoids the risk of being compelled to operate at a later stage and in unfavourable circumstances. Should the patient decline, medicinal treatment consists in absolute rest in bed, avoidance of purgatives, the bowels being opened if necessary by enema, applications of hot fomentations, and morphia if the pain is of great severity. The diet should consist of milk and milk foods.

DUODENAL ULCER.

What is the Etiology of Duodenal Ulcer?

It is similar to that of gastric ulcer (*q.v.*). The peptic ulcer of the duodenum is found chiefly in the first part of the viscus, where the acid gastric juice is not yet neutralised by the pancreatic secretion. It may arise independently,

or may follow severe burns, possibly from the absorption of cytotoxins; it occurs as a complication of various infections; and it may be found in chronic uræmia. It may occur in infants, causing melæna neonatorum, and it is considerably more common in men than women (3 to 1).

The morbid changes are similar to those of gastric ulcer.

Describe the Symptoms.

These consist of pain, tenderness, and melæna. The *pain* comes on long after food, often three to four hours after, or just before the next meal, and is hence known as "hunger pain." It is not aggravated by food, but often relieved by it, and this is supposed to be due to the closure of the pylorus following the introduction of a meal, the acid chyme being thus prevented from flowing over the ulcerated surface. The pain is seated in the epigastrium, usually to the right of the middle line, or just beneath the right costal margin. Vomiting, when it occurs, does not relieve it. *Tenderness* is often present over the painful area, and the upper belly of the right rectus is often contracted. *Melæna*, due to erosion of a vessel in the wall or floor of the ulcer, is common, and when blood is not recognisable in the stools by naked-eye inspection, it may still be discoverable by chemical tests ("occult blood").

Vomiting is infrequent, but may occur, and occasionally hæmatemesis may also be present. Dilatation of the stomach may complicate the later stages, owing to cicatrisation of the ulcer, and will present its characteristic symptoms. Diarrhœa is more common than in gastric ulcer.

Perforation is common, and may lead to abscess in the right iliac fossa, subphrenic abscess, or general peritonitis.

What is the Treatment?

Medicinal treatment, similar to that of gastric ulcer, may be tried, though it is not constantly successful. On account

of the greater risk of perforation, operation is called for at an earlier stage.

OTHER ULCERS OF THE INTESTINE.

Mention the chief Forms of Ulcer of the Intestine.

These are: ulcers met with in the specific fevers (enteric, dysentery), which have been described in Part I.; those of chronic enteritis and ulcerative colitis (see p. 189); tuberculous and syphilitic ulceration; and malignant ulcers.

What are the Characteristics of Tuberculous Ulceration?

The ulcers affect Peyer's patches in the ileum, and also the solitary follicles of the ileum and colon. They are irregular in outline, with thickened and undermined edges, and their long diameter is transverse to the axis of the bowel. Their floor may consist of the submucous or muscular coat, and small tubercles may be found on the peritoneal surface of the affected patch. Perforation occasionally follows. If a tuberculous ulcer heals, stricture of the bowel is apt to follow. The symptoms are fever and diarrhœa, which is very resistant to treatment. The stools are semifluid, yellow, and abundant, and may resemble those of enteric fever. Treatment consists in regulation of the diet and the use of astringents.

Describe the Features of Syphilitic Ulceration.

It occurs occasionally in the tertiary stage, principally affects women, and takes the form of syphilitic stricture of the rectum, following upon ulceration of gummata in the submucous coat. The stricture, which is hard and fibrous, causes symptoms of chronic obstruction, and is recognisable on digital examination.

TUMOURS OF THE INTESTINE.

What is the Principal Form of Intestinal Tumour?

Carcinoma; simple tumours are rare. Cancer of the bowel is more common in the male than the female, occurs usually between the ages of forty and sixty, and is usually seated in the large intestine, where it may be found in any of the flexures or in the rectum.

What Morbid Changes does it induce?

The primary growth is a cylinder-celled sarcoma, which may surround the bowel in an annular fashion, or consist of hard irregular nodules. It tends to ulcerate early from the passage of fæces over it, and, though at first mobile, later contracts adhesions and becomes fixed. Metastases are common, and affect the intra-abdominal glands, the peritoneum, or the liver, which is implicated through the portal circulation.

Describe the Symptoms.

The general symptoms of malignant disease, emaciation, and cachexia, are accompanied by pain of a dull character over the site of the lesion, and also by attacks of colicky pain preceding defæcation. The bowels are constipated as a rule, but there may be intercurrent attacks of diarrhœa. The motions contain blood, which may be in considerable quantity, imparting a tarry appearance to the stool if it comes from high up, and streaking it with red if the lower bowel suffers, or may be only recognisable by the tests for "occult blood." Pus may also be present, and if the tumour is low down, the calibre of the stool may be lessened. On abdominal examination a tumour may be felt in many cases, but by no means in all. It is rounded or nodular; tender,

hard, and freely mobile unless it has contracted adhesions. If the rectum is affected the tumour can be felt by digital examination, when it presents similar characters. The examining finger is often smeared with blood. The pressure of the tumour on the sacral plexus may cause unilateral or bilateral sciatica.

Other symptoms are due to intestinal obstruction set up by the growth. The obstruction is at first partial, and may be indicated chiefly by meteorism and by the colicky pain referred to above. As it becomes more complete, visible peristalsis supervenes, and stercoraceous vomiting may occur. Reflex vomiting, without fœcal odour, may be present at an earlier stage.

If the case is complicated by peritoneal carcinosis, ascites will be present. If the liver is affected, it will be found enlarged, hard, irregular, and tender.

What is the Treatment?

Usually palliative. A radical operation may be possible while the tumour is still freely mobile; but in many cases operation can do no more than relieve symptoms of obstruction, for which purpose a colostomy may be done. Medicinal treatment is directed to maintenance of the strength and relief of pain.

INTESTINAL OBSTRUCTION.

Enumerate the Causes of Intestinal Obstruction.

They may be divided into those arising in the intestine, and those arising external to it. In the former class we have to do with obstruction due to stricture (healed ulcer, malignant disease); blockage of the lumen by gall-stones, foreign bodies, or hardened fæces; volvulus; and intussusception. In the latter class are strangulation by bands or through hernial or other apertures, traction on the intestine,

and pressure upon it, as by large tumours obliterating its lumen.

How is Strangulation produced?

This condition, which is the most frequent cause of acute obstruction, often arises in connection with bands or adhesions, the result of old peritonitis, under which a loop of bowel may pass. There are also other causes, such as slits in the mesentery or omentum, peritoneal pouches, and the band formed by an adherent Meckel's diverticulum. The small intestine is most commonly involved, and strangulation is most frequent in young adults.

What is Intussusception?

The invagination of one portion of bowel into another. This condition, which is most common in male children, may occur after strains or injuries or as the result of diarrhœa. As a rule the small intestine is invaginated into the large, and when such an accident occurs, three layers of bowel are produced at the invaginated part, the innermost or *entering* layer, the middle or *returning* layer, and the outermost or *receiving* layer or *sheath*. The intussuscepted bowel becomes engorged and swollen from pressure on its vessels, and is pressed onwards by the peristaltic action of the receiving layer. The intussusception thus increases in size, and as it advances, more and more of the receiving layer is dragged in along with it.

There are several varieties of intussusception, of which the *ileo-cæcal* is the most common. In this form the ileum and cæcum pass into the ascending colon, the ileo-cæcal valve forming the head of the intussusception. In the *enteric* form one part of the small intestine is invaginated into another; in the *colic* form the large intestine is alone concerned. In the *ileo-colic*, the rarest form, the ileum passes through the ileo-cæcal valve.

Although most common in children intussusception may occur at any age, in conditions which induce irregular peristalsis.

What is Volvulus?

This is the twisting of a loop of bowel upon itself, and occurs mainly in the sigmoid flexure and pelvic colon where the mesocolon is long. It is most common in males between forty and sixty years old.

Are there any other Causes of Acute Obstruction?

It may be caused by sudden blockage of the lumen, as by foreign bodies, or by a large gall-stone which has ulcerated through the gall-bladder into the duodenum. The other causes, such as stricture, traction, compression, are usually of gradual onset and cause chronic obstruction.

Describe the Changes produced in the Bowel.

The intestine in acute cases is distended above the obstruction, and its walls are congested. Below the obstruction it is contracted and empty.

In intussusception the opposed surfaces of peritoneum are inflamed and adhesions form between them. The intussuscepted bowel may slough.

In chronic cases there is hypertrophy of the muscular coat above the obstruction, and sometimes ulceration and even perforation of the bowel.

What are the Symptoms of Acute Obstruction?

They consist in pain, constipation, vomiting, and abdominal distension. The *pain* is of sudden onset, very severe, and at first paroxysmal, though later it may become continuous. It is seated in the middle line, either in the umbilical region when the small intestine is affected, or in

the hypogastric when the lesion is in the colon or rectum. *Constipation* is absolute; no fæces are passed, except those that may be lying in the bowel below the obstruction, and even flatus cannot pass. *Vomiting* comes on early, the gastric contents being at first rejected, then bile-stained matters, and ultimately brownish fluid smelling strongly of fæces (stercoraceous vomiting). The amount of *abdominal distension* depends on the position of the obstruction; if it is high up, only the upper abdomen may be distended; if low down, the distension is uniform. *Visible intestinal peristalsis*, varying in position with the seat of the lesion, is often to be seen. On palpation, if the obstruction is due to malignant disease, a tumour may sometimes be felt through the abdominal wall or discovered in the rectum.

Collapse occurs early, the face being pinched, the eyes sunken, the skin cold and clammy, the pulse small and quick, the temperature often subnormal, and the urine scanty. Asthenia or peritonitis is the usual cause of death, which occurs in from four to six days.

By what Signs is Intussusception to be distinguished?

Constipation is not at first absolute; thin fæces mixed with blood, or blood alone, may be passed by the bowel; tenesmus is often present; and a sausage-shaped tumour may be felt in the region of the cœcum or umbilicus, or in the line of the colon, according to the position of the lesion. In infants the head of the intussusception can sometimes be felt in the rectum.

How does Chronic Obstruction differ from the Acute Form?

The history is one of increasing difficulty in the passage of fæces, with some pain before the motions, and of attacks of constipation at first relieved by aperients. The constipation becomes more obstinate, the pain more severe, and the

abdomen distended, the symptoms being then relieved by an attack of diarrhœa. Such alternations may recur for a considerable time, but ultimately the constipation becomes complete, and the signs of acute obstruction develop. As the condition is usually due to malignant tumour, the symptoms of that affection will also be present. Fæcal impaction should be remembered as among the other causes.

What is the Treatment of Intestinal Obstruction?

In all cases of acute obstruction in which the diagnosis is definite, laparotomy should be immediately performed, and the cause should be removed whenever that is possible. If this cannot be done, the bowel must be opened above the obstruction. If operation is refused, or if the diagnosis is uncertain, symptomatic treatment must be employed. The bowels may be cleared by enema, not by purgatives; thereafter the patient should be fed by the rectum. Pain may be alleviated by hot fomentations or turpentine stupes; and morphia, as it obliterates the chief symptoms, should not be used while the diagnosis is uncertain. The vomiting should be treated by washing out the stomach.

A recent intussusception may be reduced by laparotomy and manipulation of the bowel; if adhesions make this impossible, the affected portion of the bowel must be excised. If operation is declined, the obstruction may sometimes be relieved by the injection of air or fluid by the rectum.

In chronic obstruction the diet must consist of easily digestible and non-constipating food. Enemata and occasional laxatives are needed to evacuate the bowels, and pain may be relieved by opium. Operation is ultimately necessary.

Fæcal accumulations may be removed by large enemata.

V.—INTESTINAL PARASITES.

Name the more common Intestinal Parasites.

Intestinal worms are of two classes — *cestoda*, or tape-worms, and *nematoda*, or round worms. Among the cestoda are *tænia solium*, *tænia mediocanellata*, *taenia echinococcus*, and *bothriocephalus latus*; among the nematoda, *ascaris lumbricoides*, *oxyuris vermicularis*, *trichocephalus dispar*, *ankylostoma duodenale*, and *trichina spiralis*.

What are the Characters of the Tape-worms?

The adult worm, which varies in length in the different species from a fraction of an inch to many feet, is a compound organism, consisting of a number of segments, each constituting a separate individual, arranged in a chain depending from a minute head which is attached to the wall of the intestine. The head is succeeded by a slender neck, and this by a series of segments or proglottides, of which the proximal segments are immature and have no sexual organs. The oldest segments—those farthest from the head—are sexually mature and are furnished with a water-vascular system consisting of parallel canals running the whole length of the body on either side of the segment. The male and female organs open by a common aperture—the genital pore. Sexual union takes place within the proglottides, which are hermaphrodite, and the numerous ova are partly developed within them. There is no digestive system. The entire adult worm is known as the *strobila*.

The fertilised proglottides at the caudal end of the worm break off, and are expelled from the intestine by the host. The ova, already containing embryos, are set free by the decomposition of the proglottides, and being ingested in water, on grass, or in refuse, reach the intestinal tract of the animal which acts as *intermediate host*. Here the embryos are liberated by the digestion of the surrounding

membrane, and take the form of a vesicle provided with six hooklets, known as the *proscolex*, which bores into the intestinal vessels and so reaches the liver, muscles, or other internal organ of the intermediate host. There it becomes encysted and develops into a small vesicle attached to a neck and hookleted head which are usually invaginated into the interior of the vesicle. In this state it is known as the *scolex*. It does not develop further unless the organ or tissue containing it is eaten by an appropriate host (man), when the head and neck are extruded from the vesicle, which is digested by the gastric juice of the host. The head then attaches itself to the intestinal mucous membrane, proglottides develop from the neck, a new adult worm is formed, and the cycle of development is repeated in other individuals.

TÆNIA SOLIUM.

Give a Description of the Distinctive Features of this Parasite.

The mature worm infests the small intestine of man. It is a flat, ribbon-shaped organism, varying in length from ten to twelve feet. The *head* is rounded, about the size of a pinhead, and ends in a *rostellum* carrying twenty-six hooklets, below which are four suckers. The *neck* is long and thread-like, and is followed by the immature segments, which are small and thin, and then by the sexually mature *proglottides*, which gradually become longer and broader towards the caudal end. A ripe proglottis is about half an inch long and a quarter of an inch broad. The genital pores of successive segments open upon opposite sides of the body of the worm. The uterus, mesially placed, runs the whole length of the segment and has on either side from seven to ten lateral branches, each of which branches freely in its turn. The ova, which are nearly spherical, measure about 0·03 mm. in

diameter, and are surrounded by a dense capsule inside of which the six-hooked embryo develops while the ovum is still in the uterus. When the proglottis passes from the bowel, the ova are liberated by its decomposition, and are swallowed by the pig, within the body of which the embryo becomes a scolex, which is also known as a *cysticercus*. The numerous scolices constitute the "measles" of pork, and if such pork is eaten imperfectly cooked, the mature worm re-develops in the human intestine. Cysticerci also are occasionally found in man.

TÆNIA MEDIOCANELLATA.

How does this Parasite differ from Tænia Solium?

The mature worm is longer, often measuring about fifteen to twenty feet. The head has four suckers but no hooklets. The mature segment is three-quarters of an inch long and one-third of an inch broad. The uterus runs the whole length of the segment, and has on each side twenty to thirty lateral branches, which bifurcate at their ends but do not branch freely like those of *T. solium*. The ova resemble those of T. solium. The scolices or cysticerci are found in cattle, not in the pig.

BOTHRIOCEPHALUS LATUS

What are the Characters of this Parasite?

It is the longest of the tape-worms, and may be from sixteen to thirty feet long. It is found in Switzerland, Central Europe, and Japan. The head is ovoid and has no hooklets, rostellum, or suckers, but has on either side a longitudinal groove. The segments are half an inch broad, and about the middle of the worm only one-seventh of an inch long, although lower down they are more nearly square. The genital pore opens in the middle of the segment. The

uterus is an unbranched coiled tube, and the ova, ovoid in shape, measure 0·7 mm. in length, and have an operculum at one end. The embryos, which are ciliated and have six hooklets, are not developed until the ova reach fresh water. There the embryo bursts the operculum and swims about until it is swallowed by such fish as the pike, in which it assumes the cysticercus form.

To what Symptoms do Tape-worms give rise?

There may be none, and there is none that is distinctive. Gastro-intestinal disturbances, such as vague abdominal pain, attacks of diarrhœa, or voracious appetite may be present. Itching of the nose or anus is common, and there may be melancholia or reflex convulsions. Reflex vomiting also occasionally occurs. The presence of bothriocephalus, but not of the other varieties, is marked by an anæmia closely resembling that of pernicious anæmia, and only distinguishable from it by discovery of the cause. In all the forms eosinophilia is occasionally present.

If such symptoms cause suspicion, the stools should be examined for segments, the detection of which is the only certain indication of the presence of tape-worm. Their recognition is easy in the case of bothriocephalus, as the segments are passed in chains; but in the case of the other parasites, where they are passed singly, the physician himself must see the stools.

What is the Treatment?

The object of treatment is first to kill the worm, and then to remove it by a purgative. Whatever anthelmintic is used it must come in contact with the worm, which is protected by the intestinal mucus, while its head may be covered by the valvulæ conniventes. The diet for twenty-four hours before the drug is given should therefore be fluid,

and no food should be taken after six on the evening before administration. The bowels should be emptied at the outset of treatment.

The best anthelmintic is liquid extract of male fern, which is given in the morning on an empty stomach. The dose for a child is ʒss, for an adult ʒi—ii, given in mucilage or in capsules containing ♏xv of the drug. A smart purge is given three or four hours afterwards, and brings away the dead worm. The motions must be carefully searched for the head; if it is not found, it is probable that the worm has been broken off at the neck, and that it will grow again from the head which remains in the bowel.

TÆNIA ECHINOCOCCUS.

What are the Characters of this Tape-worm?

The adult worm is very small, measuring only a quarter of an inch and consisting of four segments. The first segment or head has four suckers and a double circlet of hooks. The last, which is a mature proglottis, is larger than all the rest of the worm, and in it develop the ova containing six-hooked embryos. The worm inhabits the intestine of the dog, and the development of its cystic form from the embryo usually occurs in the sheep. If the ova are swallowed by man, the embryos are liberated in the intestine and thence are carried to the liver, or occasionally to other organs. There the embryo loses its hooklets and becomes transformed into a cyst, which, in contrast with the cysticercus of other tape-worms, may attain a great size, and on account of its watery contents is known as a hydatid cyst.

The cyst sets up irritation in the surrounding tissues, and becomes surrounded by a fibrous capsule, sometimes called the false cyst. The cyst proper is composed of an external tough elastic layer, made up of several laminæ—the ectocyst, and of an internal granular and cellular *parenchymatous*

layer—the endocyst. It contains a colourless watery fluid of low specific gravity—1005 to 1007—and non-albuminous. From the inner or parenchymatous layer there now develop a number of pedunculated vesicles projecting into the interior —the *brood-capsules*, and within these are formed scolices with six hooklets and four suckers. Secondary cysts, or daughter-cysts, are also formed either from the brood-capsules or by budding from the wall of the primary cyst, into the lumen of which the daughter-cysts grow and into which they are detached. Grand-daughter-cysts may form inside the daughter-cysts. This is *endogenous* cyst formation, but *exogenous* formation may also occur, the daughter-cysts being discharged externally into the liver. In rare cases hydatid cysts may be *multilocular*, when they consist of numerous separate alveoli surrounded by dense fibrous tissue.

Beyond the hydatid stage development does not go in man or in the sheep, the adult worm being once more developed in the intestine of the dog after ingestion of the scolices.

For symptoms see Hydatid Disease of the Liver.

What are the General Characteristics of the Nematoda?

They are simple organisms, not compound as are the tape-worms, in general form cylindrical or threadlike and resembling the common earthworm. They possess a distinct alimentary canal, a thick external cuticle, and a well-developed muscular system. The sexes are separate, and the female is the larger.

ASCARIS LUMBRICOIDES.

Describe this Parasite.

The worm closely resembles the earthworm in general appearance. It is of a brown or pinkish colour, and tapers towards either end. The male is about six to ten inches long, the female ten to sixteen inches. The mouth is tri-

lobate, and the tail of the male is curved. The ova, which are very numerous, measure about 0·07 mm. in length and are oval in shape. They are brownish in colour and nodulated on the surface. The embryo is swallowed in polluted drinking water, and becomes an adult worm in the intestinal canal. It is often solitary, but several may be present in the same host.

The worm inhabits the small intestine, from which it may be passed in the stools, or it may travel to the stomach and be vomited. It may pass to the common bile-duct or may wander to the larynx or nasal passages.

What Symptoms may it cause?

There may be no symptoms, or from reflex irritation it may give rise to symptoms similar of those of tape-worm. Eosinophilia is commonly present; and evidence of intestinal irritation is not uncommon. Obstruction of the bile-duct will cause jaundice, obstruction of the larynx suffocative dyspnœa, and intestinal obstruction has sometimes been produced by a knot of the worms. The diagnosis depends on the recognition of the worm or its ova in the stools.

What is the Treatment?

The administration of santonin, which should be given on three or four consecutive nights in doses varying from one grain for a young child to five grains for an adult, and should be followed by a purge such as Pulv. Rhei. Co. Temporary yellow vision (xanthopsy) may follow its use, and it always turns the urine yellow.

OXYURIS VERMICULARIS.

Give an Account of the Thread-worm.

This is a small worm of which the female is about three eighths and the male one eighth of an inch long. The female,

which is thread-like and has a tapering tail, produces very numerous ova measuring about 0·05 mm. in length, having an operculum, and containing a partly-formed embryo. The ova are passed in the fæces, and may thus infect water; but they also collect on the skin round the anus, which is itchy through the irritation produced by the adult worm. The child scratches, collects the ova on his finger-nails, and thus transfers them to his mouth. The embryos are liberated in the intestine, and become adult in the cæcum, where sexual union occurs. The impregnated female thus passes into the large bowel, and numbers of them collect in the rectum, whence they may pass by the anus and be found on the clothing.

The main *symptom* is irritation about the anus, often worse at night. Frequent micturition may also occur. When the parasites or ova are recognised in the stools or on the clothing, the *treatment* consists in the use of astringent injections after the rectum has been cleared by enema. Infusion of quassia, tincture of perchloride of iron, or salt solution may be used. The child must be prevented from scratching the anus, and the parts must be carefully cleaned by carbolic lotion.

What are the Characters of Trichocephalus Dispar?

This parasite, also called the whip-worm, is a small worm an inch to an inch and a half long, of which the anterior two-thirds is like a thin thread, while the rest of the worm is thicker. It is found in the cæcum, but rarely causes symptoms.

ANKYLOSTOMA DUODENALE.

Describe this Worm.

It is a small, white, cylindrical worm, the female being half an inch, and the male a third of an inch long. The

mouth is provided with four hooks and two conical teeth. The ova, which are very numerous, measure 0·05 to 0·06 mm. in length, and are oval in shape. They are to be found in the fæces. Outside the body they give rise to larvæ which penetrate the skin and pass by the lymphatics to the lungs. Being coughed up, they are swallowed with the sputum, and so reach the small intestine, where they develop into the adult worm. The parasite is very common in Egypt, but occurs also in Switzerland, Italy, and other parts of Europe, and it has been found in certain mines in this country.

What are its Symptoms, and how would you treat them?

Severe anæmia of the chlorotic type, with dyspnœa, lassitude, and digestive disorders are the chief symptoms. The anæmia may be in part due to abstraction of blood from the bowel, but is largely produced by the absorption of toxins. There is some leucocytosis, and eosinophilia is marked. If the skin is constantly exposed to the attacks of the larvæ there may be a papular or pustular eruption, chiefly on the forearms and hands. The disease may end fatally.

In these cases thymol is the best anthelmintic. No alcohol or oil should be taken during its administration, as they dissolve the drug, and may produce symptoms of poisoning. The bowels should be cleared by a saline at night, half a drachm of thymol should be given in cachet next morning, and a similar dose two hours later; and three hours afterwards a second dose of saline should be given. The treatment should be repeated once a week so long as ova are found in the fæces.

TRICHINA SPIRALIS.

Describe this Parasite.

The trichina or fleshworm is of very small size, the female being one eighth of an inch long, and the male a fifteenth

to a twentieth of an inch. The head is pointed, without hooklets; the body is thicker behind than in front, and in the male there are two small processes at the tail. The mature parasites inhabit the intestine, and after conjugation the females bore into the mucous membranes, where they deposit the embryos which have been hatched from the ova within the parent body. These ova measure about 0·15 mm. in length. The embryos, after liberation, are carried by the lymphatics to the voluntary muscles, where they penetrate the muscular fibres, growing within them to a length of about $\frac{1}{25}$ of an inch. After a week or two they become coiled up, and are surrounded by a capsule due to inflammatory irritation. In this state the embryos may remain quiescent for years, and the capsules may become calcareous; but when a piece of trichinised meat is eaten, the capsules are dissolved, and the trichinæ are set free in the intestine, where they become sexually mature, and the cycle is repeated. The parasite is conveyed to man from the infected flesh of the pig, eaten in an undercooked condition, and it is transmitted to the pig from the rat. The embryos are discharged within a week after ingestion of the infected food.

What Symptoms does it produce?

The first symptoms may be those of gastro-intestinal irritation—diarrhœa, abdominal pain, and sometimes vomiting. These, however, may be slight or absent, and there may be no symptoms until about a week after the infected pork has been eaten. A period of lassitude and malaise is then followed by fever of a remittent character, with soreness and stiffness of the voluntary muscles, which are tender and hard to palpation. All movements are painful, and mastication and even respiration are thus impeded. Œdema sets in a week later, beginning in the face and spreading to the neck and arms, and sometimes even to the legs. There

is marked leucocytosis, and eosinophilia is pronounced. In severe cases the patient becomes emaciated and exhausted, and death may be due to these causes, or to intercurrent pulmonary diseases. Mild cases recover, but convalescence is slow.

What is the Treatment?

With efficient inspection of meat and proper cooking the disease may be prevented. When it exists the parasites in the muscles cannot be attacked, but those that are in the bowel should be cleared out by a brisk dose of calomel or castor oil, which should be repeated every few days. Large doses of glycerin are also recommended. The pain and fever must be treated symptomatically.

VI.—DISEASES OF THE LIVER.

JAUNDICE.

What do you understand by this Term?

A yellow discolouration of the skin and mucous membranes arising from the circulation of bile-pigment in the blood.

Give an Account of the Symptoms.

The tint of the skin, in a case of moderate severity, is yellow, and the mucous membranes are at the same time pigmented. This is best seen in the conjunctiva, the yellow colour of which helps to distinguish jaundice from other diseases attended by pigmentation of the skin. In these the conjunctiva is unstained. When jaundice is persistent the tint of the skin becomes deeper, and may at last be a deep greenish-brown. The urine contains bile pigment, which imparts to it an orange or greenish tint, and in some cases

the urine may be dark brown. Most of the other secretions escape, but the sweat is sometimes pigmented.

The fæces are usually clay-coloured, since in most cases no bile enters the intestine. The absence of bile also leads to imperfect digestion of fat, which may be found in the stools, and to fœtidity of the excreta owing to increased rapidity of putrefaction. Constipation is usual, though diarrhœa may occur. Gastric disturbances such as nausea and anorexia are frequent. The pulse is sometimes slow; itchiness of the skin is frequent; and subcutaneous hæmorrhages, or hæmorrhage from mucous surfaces, may occur. Depression is common, and in grave cases delirium, convulsions, or coma may precede the end.

In non-obstructive jaundice the tint is usually lighter, the fæces are of normal colour, and bile-pigment may not be present in the urine.

What are the Tests for Bile-pigment in the Urine?

1. *Gmelin's test.*—Pour a little strong nitric acid into a test-tube, and float a little of the urine on its surface; a green ring develops at the junction of the fluids. Or let a few drops of urine fall on a porcelain plate, and a few drops of nitric acid beside them; tilt the plate so that the fluids run together, when a play of colours—green, blue, violet, red, yellow—is observed at the point of meeting. The green colouration is due to the oxidation of bilirubin into biliverdin.

2. *Maréchal's test.*—A dilute solution of iodine is floated on to the surface of the urine in a test-tube; a green ring forms at the line of junction.

Urine containing bile-pigments leaves a yellow or orange stain upon linen or blotting paper. When such urine is shaken, the froth upon its surface is not colourless, but greenish-yellow.

The tests for bile acids are unsatisfactory.

How is Jaundice produced?

It may be due to obvious obstruction to the larger bile-ducts, or to changes in the blood. In the former case (*obstructive* jaundice), the bile is prevented from entering the intestine, and consequently distends the bile-ducts and is forced into the lymphatics. Thence it passes into the blood, in which it circulates. In the latter case (*hæmatogenous*, *hæmolytic*, or *toxæmic* jaundice), poisons circulating in the blood and causing destruction of the red cells increase the amount of hæmoglobin brought to the liver, and there converted into bile-pigment. An excess of pigment in the bile increases its viscosity, and if at the same time, as is often the case, the toxins in the blood have set up a catarrh of the minute bile-ducts, the rate of biliary flow is much diminished, the pressure in the ducts is increased, and bile is forced into the lymphatics, and thence into the blood, even in the absence of complete obstruction. The poisons giving rise to such hæmolysis may be those of infective fevers (*e.g.* enteric, yellow fever, pneumonia); other microbic poisons; or such substances as toluylendiamine, snake-venom, phosphorus, etc.

What are the Causes of Jaundice?

Obstruction to the larger bile-ducts may be due to internal causes—gall-stones, hydatids, distomata, and foreign bodies from the intestines (*ascarides*); to inflammatory swelling of the mucosa of the duct or duodenum (catarrhal jaundice); to stricture or obliteration of the duct from congenital deficiency, perihepatitis, the cicatrisation of biliary or duodenal ulcers, or spasm of the duct; or to compression by tumours of the liver; enlarged glands in the portal fissure; tumours of the stomach, pancreas, kidney, omentum, ovaries, or uterus; abdominal aneurysm, fœcal accumulation, or the pregnant uterus.

Jaundice due to obstruction of the smaller ducts occurs in

the various forms of cirrhosis of the liver; catarrh of the minute bile-ducts and increased viscidity of the bile give rise to the jaundice of acute and chronic congestion; while toxæmic or hæmolytic jaundice occurs in poisoning by phosphorus, arsenic, mercury, antimony, and snake-venom, in acute infectious diseases such as typhus, malaria, pyæmia, and those mentioned above, in some anæmias, in Weil's disease, and acute yellow atrophy of the liver.

ASCITES.

What is Ascites?

The presence of serous fluid in the peritoneal cavity.

What are its Causes?

Obstruction to the portal circulation either in the portal vein or its branches in the liver; chronic peritonitis, whether simple, tuberculous, or malignant; and renal or cardiac dropsy.

How may Portal Obstruction be produced?

The trunk of the vein may be pressed upon by tumours of the liver or pancreas, or by tumours or enlarged glands in the portal fissure. Hepatic cirrhosis may cause obstruction within the liver. Thrombosis of the portal vein is an occasional cause.

What are the Physical Signs of Ascites?

Enlargement of the abdomen, which tends to bulge laterally when the patient lies upon his back; dilatation of the superficial veins; and obliteration of the umbilical depression. If the amount of fluid is large, the umbilicus may protrude. On percussion the umbilical and epigastric regions are resonant, while the flanks and hypogastrium are dull, the

fluid gravitating to the more dependent parts, and the intestines floating on its surface. If the patient be turned on one side, the upper flank becomes resonant, and the area of dulness in the lower flank is increased. If the intestines are so bound down by adhesions that they cannot float, the note may be dull all over; if adhesions limit the movement of the fluid, as sometimes in chronic peritonitis, the upper flank may not clear when the patient lies on his side.

When the hand is laid flat on one flank, and the opposite flank is tapped by a finger of the other hand, a wave of *fluctuation* is transmitted through the fluid and is felt by the examining hand. This may be simulated by vibrations transmitted through the abdominal wall in very fat people; to prevent mistake the hand of an assistant should be placed edgewise on the middle line of the abdomen for the purpose of stopping such vibrations.

What are the Characters of Ascitic Fluid?

It is a clear straw-coloured albuminous fluid, of a sp. gr. of 1010 or less. The fluid due to inflammatory exudation has a sp. gr. of 1014 or more. Occasionally the fluid is turbid and milky, and contains fat globules (*chylous* ascites due to ruptured lacteals or to pressure on the thoracic duct); and in the presence of malignant disease of the peritoneum it may be blood-stained.

ICTERUS NEONATCRUM.

Give an Account of this Condition.

The jaundice occurring in the new-born is usually a temporary condition, coming on about two or three days after birth and lasting for a week or a fortnight. The skin is yellow, the fæces are pale, and the urine may contain

bile, but there are few other symptoms. It is probably of hæmolytic origin. When the jaundice is due to congenital obstruction of the bile-duct, phlebitis of the umbilical vein, or congenital syphilitic hepatitis, the result is fatal.

CONGESTION OF THE LIVER.

Mention the Causes of Hepatic Congestion.

Congestion of the liver may be passive or active. The former is a consequence of cardiac disease in the stage of failure of compensation. The latter is present in the early stages of hepatitis, and in certain specific fevers as the result of toxæmia. It may also be brought about towards middle life by a prolonged course of luxurious living and over-indulgence in food and drink.

What are the Symptoms of Active Congestion?

Slight enlargement of the liver and tenderness to pressure, coupled with a sense of weight in the right hypochondrium, pain in the right shoulder, furred tongue, nausea or vomiting, constipation, and slight jaundice.

How would you treat it?

By rest, milk food, poultices to the liver, and small doses of a mercurial purgative.

ABSCESS OF THE LIVER.

What are the Varieties of Abscess of the Liver?

They may be multiple or solitary, and the infective material may reach the liver by the portal vein, the hepatic artery, or the bile-ducts. Multiple abscesses arise in the distribution of the hepatic artery in connection with cases of pyæmia, and in the distribution of the portal vein

(portal pyæmia), in connection with cases of suppurative pylephlebitis consecutive to such intestinal lesions as appendicitis, bacillary dysentery, etc. Solitary abscesses are most commonly the result of amœbic dysentery.

What are the Symptoms and Treatment of Multiple Abscesses?

The symptoms may be very obscure, but in general they are those of pyæmia. There is hectic fever, the pulse is rapid, the tongue dry and brown, and there is marked prostration. The liver is uniformly enlarged, painful, and tender to pressure. Jaundice is usually, but not always, present, and in its presence the urine contains bile-pigment. The disease ends fatally after a duration of a few weeks. The treatment is directed to the relief of pain and the maintenance of the general strength by stimulants and quinine.

How does a Solitary Abscess arise?

The condition is most frequent in the tropics, and alcoholic excess predisposes to it. It is usually produced by the amœba dysenteriæ, although in some cases the bacillus coli has been found. Most of the patients have had dysentery.

Describe the Morbid Appearances of a Tropical Abscess.

The abscess is usually single, but there may be more than one. Its contents are composed of liquefied necrotic tissue, and in the smaller abscesses are glairy and translucent, while in the larger they are thick, viscid, and chocolate-coloured and have a nauseous odour. The wall of a recent abscess is composed of ragged necrotic tissue without lining membrane; older abscesses have thicker walls and are surrounded by dense connective tissue. The abscess may open in various directions, very commonly into the right lung or

pleura, but often also into the abdominal cavity or the gastro-intestinal tract, while occasionally it opens externally. Abscesses due to the bacillus coli contain ordinary pus.

To what Symptoms does it give Rise?

At first the symptoms are those of active congestion or of hepatitis (which apart from hepatic abscess is an uncommon disease), *i.e.*, pain, tenderness, and a sense ef fullness in the right hypochondrium, usually with slight jaundice. Rigors and irregular fever follow, the pain becomes aggravated as the abscess nears the surface, and sets up perihepatitis; pain in the right shoulder may also be felt, there is a short, dry cough, and there may be some vomiting. The liver becomes much enlarged, the area of dulness being increased both upwards and downwards, and the enlargement is not uniform. There may be a definite prominence in the right hypochondrium. Friction or crepitus may be present over the base of the right lung, or the increased hepatic dulness, with diminished breath sounds, may simulate empyema. As the case progresses the patient becomes emaciated and prostrate.

The abscess may point or burst in any of the directions indicated above, and thus pleurisy, empyema, or pneumonia may occur, as may also pericarditis and localised or general peritonitis. Rupture into the lung is attended by the presence of a sputum resembling anchovy paste.

What is the Treatment?

Before there is evidence of pus, *i.e.*, in the stage of hepatitis, ipecacuanha in doses of gr. xx. (1·2 gm.) every six hours, or better emetine hydrochloride injected hypodermically in doses of gr. $\frac{1}{6}$ to $\frac{1}{3}$ (0·01—0·02 gm.) every four hours, may be given. The latter drug, in particular, may prevent suppuration. When pus has formed, the treatment

is surgical. Amœbic abscess without microbic complication may be treated by aspiration and injection of emetine into the cavity; if other organisms are present the abscess must be opened and drained.

ACUTE YELLOW ATROPHY OF THE LIVER.

Give an Account of this Disease.

This rare condition is characterised by rapid degeneration of the hepatic cells accompanied by marked diminution in the size of the liver. It is more common in women than men, and occurs chiefly between the ages of twenty and thirty. Predisposing causes are emotional overstrain, constitutional syphilis, and loose living. It is sometimes associated with pregnancy.

The liver is much diminished in size and soft in consistency, while the capsule, too large for the shrunken organ, is wrinkled. On section, the cut surface is yellow, mottled with bright red patches. In the red parts the hepatic cells are almost completely necrotic and destroyed; in the yellow parts, where destruction is less advanced, the cells are undergoing fatty degeneration. Crystals of leucin and tyrosin, pigment, and fat-granules are to be seen under the microscope. The bile-ducts are empty. Fatty degeneration is also present in other organs (heart, kidneys), and petechial hæmorrhages are found in the skin and mucous membranes.

The disease is probably of infective origin, the changes being due to a profound toxæmia, but no specific organism has been found.

The symptoms begin somewhat insidiously, with lassitude, gastro-intestinal disturbances, hepatic pain, and slight jaundice. After a week or two the jaundice becomes more intense, and headache, restlessness, and delirium appear, with rapidity of the pulse, a dry and brown tongue, and tenderness in the hepatic area. The liver dulness rapidly

decreases in size. The spleen is enlarged; the urine contains bile, and sometimes blood, while urea and salts are much diminished in amount, and leucin and tyrosin are present. Hæmorrhages into the skin or from the mucous membranes may occur. Convulsions or coma precede the end, which occurs within a week of the development of severe symptoms. Pregnant women usually abort.

Treatment consists in the relief of symptoms.

CIRRHOSIS OF THE LIVER.

What is this Disease?

It is a chronic inflammatory condition leading to an excessive growth of connective tissue, which by its contraction presses upon and destroys the hepatic cells and obstructs the portal circulation. It may take the form either of portal, multilobular, or atrophic cirrhosis, or of biliary, unilobular, or hypertrophic cirrhosis. A third variety is known as intralobular or pericellular cirrhosis. The portal and biliary types require separate description.

PORTAL OR ALCOHOLIC CIRRHOSIS.

What is the Etiology of this Affection?

The most prominent factor in the clinical history is generally alcoholic excess, and particularly excess in spirits, continued over a long period of time. The patients are therefore usually males, and, as a rule, middle-aged, although the disease occasionally occurs in childhood (congenital syphilis), and sometimes in patients in whom there is no alcoholic history. Alcohol, then, though the usual, is not the sole irritant which may produce it, and probably acts less by direct irritation than by lowering the resistance of the liver to toxins produced in the gastro-intestinal tract as the result of the catarrh which it sets up there, the toxins being conveyed to the liver by the portal circulation.

Describe the Morbid Changes.

The liver varies in size, being in some instances much enlarged, either from temporary congestion or from fatty change, in which case the enlargement is permanent. In the majority of instances it is temporary, and succeeded by an atrophic stage; and in very many cases there is no definite stage of hypertrophy, but atrophy is present from the first. The large livers are smooth or only slightly roughened; the atrophic livers, which may weigh as little as thirty ounces, have a rough and nodular surface. The changes are due to the proliferation and subsequent contraction of fibrous tissue surrounding and compressing the branches of the portal vein, the proliferation stage giving rise to enlargement of the liver. The capsule of the liver is also much thickened. The strands of fibrous tissue penetrating the organ separate the hepatic parenchyma into irregular masses, consisting, for the most part, of several lobules, while some of them may be smaller than a lobule. Each such area is surrounded by, or encapsulated in, dense fibrous tissue, which presses upon the enclosed hepatic cells and causes them to atrophy. The prominence of these irregular areas upon the surface of the organ causes the roughness already mentioned ("hob-nailed liver"). The bile-ducts are not pressed upon until a late stage of the disease.

What Secondary Changes result from this Condition?

Pressure upon the radicles of the portal vein causes obstruction to the portal circulation, and congestion in the area which it drains. Hence enlargement of the spleen follows, there is a tendency to hæmorrhoids, gastric and intestinal engorgement and catarrh are common, and ascites is a frequent symptom. Anastomoses between the portal and systemic veins result, and there is often visible evidence

of this in the present of a knot of varicose veins (*caput medusæ*) around the umbilicus (anastomosis with the epigastric veins through the accessory portal vein of Sappey).

What are the Symptoms of Portal Cirrhosis?

The onset is very insidious; in an alcoholic subject there may be evidences of gastric catarrh with morning vomiting and loss of appetite, and sometimes hepatic pain. The liver may even now be enlarged, but the patient's attention is often not aroused until a hæmorrhage from the nose or stomach occurs, or an attack of melæna. When the abdomen is now examined, besides the hepatic enlargement, the spleen is found to be increased in size, and hæmorrhoids may be present. The enlarged liver is firm and hard, and only slightly tender; the "hobnails" on the surface are only occasionally palpable. Emaciation follows, the abdomen becomes distended from meteorism, and at a later stage ascites makes its appearance. The liver may still be enlarged, and may be palpable through the abdominal fluid; but often it is atrophic and no longer palpable. The superficial abdominal veins become distended and varicose from the attempt at establishment of a collateral circulation; and pressure of the ascitic fluid on the inferior vena cava may cause œdema of the feet. The renal veins may also be compressed, the urine being high coloured, scanty, and containing albumin. Jaundice appears late, and is not marked. It is probably of hæmolytic origin. Fever is usually absent. Meantime the general health has suffered severely: the patient is weak and readily exhausted, and in the later stages nervous symptoms, due to toxæmia (drowsiness, delirium, coma), appear, and lead to the fatal end, which may also be due to cardiac failure, to asthenia, or, more rarely, to a severe hæmorrhage. The disease may be at least temporarily arrested in the early stages by an altered mode of life.

BILIARY CIRRHOSIS (Hanot's Disease).

What are the Causes of this Condition?

The essential cause is not known, but is probably a toxin circulating in the blood and reaching the liver by the systemic circulation. Hypertrophic cirrhosis is most common between the ages of twenty and thirty, though it may affect children. Several members of a family may be attacked, and males are more liable than females.

Compare the Morbid Changes with those of Portal Cirrhosis.

In biliary or unilobular cirrhosis the liver is constantly enlarged, often to a great extent (hence the name hypertrophic cirrhosis). It is firm, and either smooth or finely granular to the touch. The new-formed fibrous tissue, looser than that of portal cirrhosis, is diffused throughout the substance of the liver, and divides the parenchymatous cells into single lobules. The portal circulation is not pressed upon, but there is inflammation of the smaller bile-ducts (cholangitis), and hence jaundice occurs early. The capsule is not thickened. These changes contrast with the hypertrophy giving place to atrophy, the roughened surface, the multilobular distribution of the fibrous tissue, the involvement of the portal circulation, and the thickened capsule which are found in portal cirrhosis. The latter condition may, however, supervene in the late stages of the biliary form.

Describe the Symptoms.

The disease begins insidiously, and there is not necessarily an alcoholic history. Jaundice occurs early; at first slight, it progressively deepens, and may become intense. The urine contains bile pigment, but the stools retain their colour. The liver is found to be greatly enlarged, smooth, firm, and slightly tender. The spleen is also much increased in size, and the upper part of the abdomen is often

prominent. Attacks of abdominal pain may occur. The patient gradually loses flesh, and fever is an intercurrent or constant symptom. Ascites may be altogether absent, or if present, it occurs late, as a consequence of secondary portal cirrhosis. After a duration of two or three years, toxæmic symptoms become acute, hæmorrhages from the nose or under the skin appear, and delirium, coma, or the typhoid state precedes death.

How would you treat a Case of Cirrhosis of the Liver?

Whether the cirrhosis be portal or biliary, it is incurable, and in advanced cases nothing can be done beyond the relief of symptoms. In the early stages of portal cirrhosis it may be possible to arrest the disease. In all cases abstinence from alcohol must be enjoined; the diet must be light and nourishing, and, indeed, should consist chiefly of milk and milk foods so long as gastric symptoms are present; and the bowels must be kept active without excessive purgation. The saline purgatives are best, as they tend to deplete the engorged portal circulation. Moderate exercise, and tonics such as nux vomica, are useful when the disease is quiescent, and chloride of ammonium in five-grain (0·3 gm.) doses may sometimes do good as a hepatic stimulant. When ascites is present the patient must be kept in bed, and in the early stages purgatives such as jalap or the salines may be used. Diuretics (the potassium salts, diuretin, digitalis) should also be given, but often fail owing to the engorgement of the kidney produced by pressure of the ascitic fluid. When this becomes large in amount, embarrassing the breathing or the cardiac action, paracentesis is necessary, and may be repeated as the fluid reaccumulates. Operative attempts to improve the collateral circulation by suturing the great omentum to the abdominal wall have had only a limited success. For the treatment of hæmatemesis see p. 175.

SYPHILIS OF THE LIVER.

In what Forms may this Affection be met with?

It may be congenital or acquired. In congenital syphilis the liver is frequently involved, and the lesion is usually an interstitial hepatitis, less often gummatous. In interstitial hepatitis a fibroid induration surrounds the lobules, or even extends between the individual cells (intracellular cirrhosis). The liver is large, firm, and resistant, and the spleen may also be enlarged. Ascites is rare. Other signs of congenital syphilis are also present. The condition yields to prolonged antisyphilitic treatment, but may leave a liability to portal cirrhosis later in life.

In the adult, jaundice due to a specific hepatitis, and clearing up with appropriate treatment, may occur in the secondary stage. The tertiary lesions are commoner and more serious. They usually take the form of gummata or gummatous infiltration, producing irregular enlargements of the liver, and lobulation due to the presence of depressed cicatrices resulting from contraction of the fibrous tissue surrounding the gummata. Amyloid disease may develop in such livers. The symptoms are irregular enlargement of the liver, which is firm, and of which the edge shows palpable depressions; often coincident enlargement of the spleen; and sometimes ascites or jaundice. Intermittent or remittent fever may be present, and the Wassermann test is positive. Specific treatment may be curative in the early stages, while at a later period it can only prevent further change.

AMYLOID OR LARDACEOUS DISEASE OF THE LIVER.

What is Amyloid Degeneration?

It is a change affecting the tissues of the liver, spleen, kidneys, and sometimes the intestine, and resulting in the

laying down in these tissues of a firm, colourless, homogeneous material allied to the matrix of cartilage. This translucent material (lardacein or amyloid substance) is a glycoproteid. It is deposited first in the walls of the blood-vessels, next in connective tissues, and lastly it may attack the parenchymatous cells of the affected organs. Its presence is recognised by pouring tincture of iodine over the cut surface of the organ, when the amyloid areas turn a brownish red, which changes to purple on adding dilute sulphuric acid.

What are its Causes?

Prolonged suppuration such as occurs in phthisis or in caries of bone, old-standing syphilis, osteomyelitis, and occasionally the malarial cachexia or chronic renal disease.

How does it affect the Liver?

It causes an enormous increase in the size of the organ, the lower border of which may be felt below the umbilicus. The surface of the liver is smooth, firm, and not tender to pressure; its edge is hard and rounded; and its outline is regular. The only hepatic symptom is a sense of weight in the right hypochondrium; jaundice is absent. The disease may be diagnosed from the characters of the hepatic enlargement, the presence of an exciting cause, and the evidence of amyloid change in other organs (enlarged spleen, albuminuria, diarrhœa). Ascites may occur in the late stages, but is due to renal disease and not to portal obstruction. Amyloid change may coincide with syphilis or fatty disease of the liver. Death is its general result.

How would you treat it?

By attacking its cause, surgically or by other appropriate measures; and by maintenance of the strength. Cod-liver

oil, iron, or tonics may be prescribed, and iodide of potassium is often useful.

FATTY LIVER.

What Varieties of Fatty Change are met with in the Liver?

Fatty infiltration and fatty degeneration. The former of these, in which fat-globules accumulate in the liver cells to an excessive degree, is an exaggeration of the normal condition. In fatty degeneration the protoplasm of the cells is partly replaced by fat.

In what Circumstances may they occur?

Fatty infiltration is common in obesity, alcoholism, wasting diseases such as phthisis, and in the congested liver of chronic heart disease; fatty degeneration in acute yellow atrophy of the liver, and phosphorus poisoning.

What are the Characters of a Fatty Liver?

It is much enlarged, smooth, and has a rounded edge. Its consistency is sometimes soft, sometimes firm; and on section the cut surface is greasy. It gives rise to no symptoms except those of the condition causing it, but on physical examination the liver can be felt to be enlarged, smooth, and softish to palpation. It is not tender; there is no jaundice or ascites; and the spleen is not enlarged.

Treatment is that of the causative disease.

CANCER OF THE LIVER.

Mention the Forms of Tumour which may be found in the Liver.

Cancer, angioma, simple cysts, lymphoma (as in Hodgkin's disease), adenoma, and sarcoma. Cancer is the only one of these which is clinically important. Although sometimes

primary it is much more often secondary to growths in the gastro-intestinal tract, the female breast, the uterus, or the vertebræ. It may follow upon cancer of the gall-bladder consecutive to the long-standing irritation of gall-stones. It is most common between the ages of forty and sixty.

Describe the Morbid Appearances produced by Cancer of the Liver.

Primary carcinoma may be solitary, in which case it forms a greyish mass sharply separated from the normal hepatic tissue; nodular, the nodules being scattered throughout the organ; or diffuse, when it is often secondary to cirrhosis. The growth is usually epitheliomatous in type.

In secondary carcinoma the type of cell is determined by that of the primary tumour. The liver, which is greatly enlarged, is occupied by numerous yellowish-white nodular masses of varying size, which protrude upon the surface and the edge. As they increase in size they tend to break down in the centre, and thus their surface presents a central depression or *umbilication*. Section of the nodules shows that this central softening consists of granular and fatty debris, and shows also that hæmorrhages have taken place into their substance.

What are its Symptoms?

Pain in the hepatic region and the right shoulder is usually prominent. It may be slight at first, but tends to become severe. The liver is felt to be much enlarged and is *tender* on pressure. Its surface is irregular, being studded by large nodules which can be readily felt through the emaciated abdominal wall, and are intensely hard. A central depression can be made out in the larger nodules. The edge of the organ is also irregular. Jaundice is present when a nodule in the portal fissure presses on the common bile-duct,

i.e., in about half the cases. Ascites is somewhat less common. Attacks of local peritonitis occur in many cases; and irregular fever, due to these or to toxæmia, is often present. The general symptoms of cancer — emaciation, cachectic appearance, loss of strength — accompany the condition, and there is often evidence of a primary growth in the gastro-intestinal tract or elsewhere. In doubtful cases the rectum should always be examined.

The course is rapid, and no treatment beyond alleviation of symptoms can be attempted.

HYDATID DISEASE OF THE LIVER.

What is this Condition?

It is due to the formation of single or, less commonly, multiple cysts in the substance of the liver as a consequence of the presence of the embryo of *tæmia echinococcus*. The characters of the parasite and of the cysts have been described at p. 205.

What are its Symptoms?

In the early stages the general health is little disturbed, and there is no pain. The disease is therefore diagnosed mainly from physical signs. A hydatid cyst is usually seated in the right lobe of the liver, which is increased in size. If it grows from the anterior surface, it forms a tense globular tumour in the hypochondrium. The tumour may be elastic or fluctuant, and sometimes presents, on placing the fingers over it and tapping on them with a finger of the other hand, a vibratory sensation known as the "hydatid thrill." There is no tenderness. Cysts on the posterior surface cause an upward increase in the hepatic dulness; those in the substance of the liver cause bulging of the right side of the chest and elevation of the

lower ribs, with dulness at the base of the right lung. Eosinophilia is usually present. Pain may occur if the cyst causes a localised peritonitis, but it is generally due to suppuration of the contents. This is accompanied by rigors, fever, sweating, and loss of strength, *i.e.*, the symptoms of pyæmic abscess.

Should the parasite die, the cyst becomes obsolete; usually it increases in size until it ruptures into the lung, pleura, pericardium, gastro-intestinal tract, hepatic duct, or peritoneal cavity. Rupture into the serous sacs is generally fatal.

How is it to be treated?

By incision and removal of the cyst. Aspiration is often followed by the death of the parasite, but incision is the better treatment.

CHOLANGITIS.

What is meant by this Term?

Inflammation of the bile-ducts. It may be catarrhal or suppurative.

Give an Account of Catarrhal Cholangitis.

In this condition, also known as *catarrhal jaundice*, inflammation of the lower end of the common bile-duct is due to extension of a duodenal catarrh, and its usual causes are therefore those of gastritis and enteritis. Swelling of the biliary mucosa is the result, and leads to obstructive jaundice, of which catarrhal cholangitis is one of the commonest causes. The jaundice is often preceded, but not always, by gastro-intestinal symptoms, apart from which the patient suffers no pain. The skin and conjunctivæ are yellow, not bronzed; the urine contains bile-pigment; and

the stools are clay-coloured. The liver is neither tender nor enlarged; there is no fever; and the pulse is usually slow. Pruritus is often troublesome. The disease is most common in young adults, and the jaundice passes off in a few weeks. Treatment consists in light diet, abstinence from alcohol, the use of saline laxatives, and the administration of alkalies such as the bicarbonate of sodium.

What are the Features of Suppurative Cholangitis?

It is due to infection by the pyogenic organisms, the bacillus coli, or the typhoid bacillus, the determining cause being local conditions such as gall-stones, or general infections. The bile-ducts are inflamed, thickened, and dilated, and scattered foci of suppuration are found in their course. The general symptoms are rigors, fever, nausea, and vomiting. The liver is enlarged, painful, and tender, and there is often jaundice. The gall-bladder may be palpable. The disease may be fatal through general pyæmia. The treatment consists in drainage of the ducts, through the gall-bladder or otherwise.

CHOLECYSTITIS.

Give an Account of this Condition.

Inflammation of the gall-bladder may be catarrhal, suppurative, or gangrenous. It is the result of microbic infection, and may accompany cholangitis, may be due to gall-stones, or may be an uncommon complication of infectious diseases, especially enteric fever. The gall-bladder is distended, its walls thickened, and its mucous membrane swollen, often sufficiently to close the cystic duct. The symptoms are fever, pain, and tenderness, with increased resistance in the region of the gall-bladder or referred to the appendix, nausea, sometimes vomiting, and some degree of prostration. Jaundice is sometimes present. Treatment consists in rest in bed,

warm fomentations to the hypochondrium, and morphia hypodermically. In the presence of suppuration or of gall-stones, cholecystotomy or cholecystectomy is indicated.

GALL-STONES (Cholelithiasis).

What Causes lead to the Formation of Gall-stones?

Gall-stones, or biliary calculi, are formed from the bile in the gall-bladder, or more rarely in the smaller bile-ducts, as a result of chronic catarrh of the mucous membrane. This process is set up by microbic infection, the responsible organisms being *b. coli*, *b. typhosus*, and the less virulent strains of staphylococcus, and is favoured by conditions leading to the stagnation of bile in the gall-bladder, such as sedentary occupations, tight-lacing, enteroptosis, and especially pregnancy. These conditions are most common in women, who form the majority of the cases. Gall-stones are rare in youth or early adult life, when the habits are active.

What are the Characters of the Stones?

They are composed of cholesterin, bile-pigments, and sometimes of calcium carbonate. Pure cholesterin calculi are somewhat rare and are usually single. They are oval in shape, pale yellow, and have a waxy surface. Bilirubin calcium calculi without admixture of cholesterin appear as multiple blackish friable granules ("bile gravel"). Pure calcium carbonate calculi are decidedly rare. The common gall-stone is a mixture of all three ingredients in varying proportions. They are brown, green, or yellowish in colour, and though sometimes single are more often multiple, in which case the individual stones are faceted from contact with their neighbours. The size of the stones is in inverse ratio to their numbers.

With regard to the pure cholesterin calculus, Aschoff and

Bacmeister have of late maintained that it is the product simply of stagnation of bile in the gall-bladder, without any inflammatory condition or evidence of bacterial infection.

To what Symptoms may Gall-stones give Rise?

Their presence in the gall-bladder may give rise to no symptoms for many years, although in some cases there is dragging pain in the right hypochondrium. Sometimes, however, as a result of fresh infection, cholecystitis follows, and this may be either catarrhal or suppurative. Adhesions between the inflamed gall-bladder and neighbouring structures may occur, and this may be followed by a fistulous opening into the duodenum or colon, through which the stones pass and are voided *per anum*. A single large stone, entering the bowel in this way, may cause intestinal obstruction. If the gall-bladder adheres to the abdominal wall, the fistula may open externally.

Biliary colic is the result of the passage of a stone into the cystic duct, or the common bile-duct, and the consequent spasm of the duct in its effort at expulsion. The onset of an attack is sudden : there is excruciating pain in the right hypochondrium, radiating across the abdomen into the lower part of the chest and into the right shoulder. The pain is paroxysmal as a rule, lasting with great intensity for some time, and then becoming duller, without disappearing, until a fresh paroxysm arises. It is accompanied by tenderness over the right hypochondrium, and the gall-bladder may be enlarged. Rigors may occur, the pulse is feeble and rapid, the skin is covered with a cold sweat, there may be vomiting, and there are signs of collapse. If the stone is in the cystic duct there is no jaundice; if it is in the bile-duct, the signs of obstructive jaundice appear a few hours or a day or two after the onset. The attack may last for a few hours or for several days, and ends by the passage of the stone into the duodenum or by its slipping back into the

gall-bladder, after which the patient is left prostrate but free from pain, and the jaundice gradually clears up.

In other cases the stone becomes impacted in the cystic duct or common duct. If it is in the cystic duct, after the biliary colic due to the effort at expulsion has ceased, dropsy of the gall-bladder may follow, the organ becoming palpable as a movable ovoid tumour under the ribs; or suppurative cholecystitis may ensue; or, in long-standing cases, atrophy of the gall-bladder. If it is in the common duct, jaundice persists and deepens, the bile-ducts are distended, and the liver is at first enlarged, but later becomes somewhat atrophic.

It should be remembered that cancer of the gall-bladder, extending to the liver, may follow the long-continued presence of gall-stones.

How would you treat a Case of Cholelithiasis?

An attack of biliary colic is to be treated by hot fomentations to the hypochondrium, or by hot baths, hypodermic injections of morphia, and, as a temporary measure, chloroform inhalation. After its relief the diet must be regulated, starchy and fatty foods being limited in amount, regular exercise must be secured, and abdominal massage may be practised. Sulphate and phosphate of soda are supposed to have a solvent action on gall-stones; salicylate of soda is useful in thinning the bile and promoting its flow; and small repeated doses of calomel are also of benefit. Hot water or alkaline waters should be freely drunk. Complications or recurrent attacks of colic call for surgical treatment.

VII.—DISEASES OF THE PANCREAS.

PANCREATITIS.

Give an Account of this Disease.

It may be either acute or chronic. The acute form occasionally occurs in infective diseases, but is usually due to local infection extending along the duct from the intestine or from the biliary passages. It may be hæmorrhagic, suppurative, or gangrenous. The onset is usually sudden, but there may have been previous gastro-intestinal or hepatic symptoms. In *acute hæmorrhagic pancreatitis* the symptoms are sudden and "violent pain in the epigastrium, followed by vomiting and collapse, and in the course of twenty-four hours, by a circumscribed epigastric swelling, tympanitic or resistant, with slight rise of temperature." At a later stage there may be slight jaundice, and the extravasated blood may cause some dulness in the flanks. The suppurative form causes similar but less acute symptoms. Both, as a rule, end fatally. Medicinal treatment consists mainly in the relief of pain by morphia; but the urgency of the symptoms will usually call for surgical intervention, by which life has been saved in a certain proportion of the cases.

Chronic pancreatitis causes a general fibrosis of the gland, and in some instances is associated with diabetes, while in others it may lead to jaundice from implication of the common duct. It has no definite symptoms of its own, and is often discovered in the course of operations on the common bile-duct or gall-bladder.

TUMOURS AND CYSTS OF THE PANCREAS.

What are the Main Features of these Conditions?

Cancer is the only clinically important tumour. It may be either scirrhus or medullary, more commonly the former,

and its favourite seat is in the head of the gland. Metastases may affect the liver and spleen. From its situation it is apt to press upon the duodenum, causing dilatation of the stomach; or on the common bile-duct, causing jaundice. It may also involve the stomach, peritoneum, or vertebræ. The symptoms are severe and gnawing epigastric pain, often affected by food, and paroxysmal in character; wasting and malignant cachexia; and the subsequent development of a deep-seated epigastric tumour, immobile on respiration, and tender to palpation. Vomiting may occur; and the stools may contain excess of fat or undigested muscle fibres.

Pancreatic cysts are due to obstruction to the duct by a calculus or by the pressure of a tumour. They may attain a great size and form a globular swelling in the epigastrium, which often bulges visibly. Such a tumour is elastic but non-fluctuant. It may be covered by the stomach or may have pushed it aside, and the percussion note is therefore either tympanitic or dull. The fluid it contains is turbid, greenish, alkaline, of sp. gr. 1010—1020. It contains albumin and sugar, digests starch, and emulsifies fat. The treatment of such a cyst, after the diagnosis has been confirmed by aspiration and examination of the fluid, consists in incision and drainage.

VIII.—DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

What are the Causes of Acute Peritonitis?

It is almost invariably the consequence of bacterial infection, the usual organisms being the pyogenic cocci, those derived from the intestine (*b. coli*, *b. typhosus*), and the gonococcus. These organisms reach the peritoneum through local lesions of the gastro-intestinal tract, of the abdominal organs, or of the female genital tract. Peritonitis may thus

arise from extension of inflammation in cases of hepatic abscess, suppurative cholecystitis, splenic infarction; uterine or tubal affections such as metritis, parametritis, or salpingitis; and in cases of intestinal obstruction or strangulated hernia. Another large group of cases is due to perforation of some part of the gastro-intestinal tract, with the escape of food, pus, or fæces into the peritoneum. Perforating gastric or intestinal ulcers, acute appendicitis, or the rupture of abscesses, may thus cause it. Penetrating wounds of the abdomen are responsible for some cases. Occasionally infection would seem to be transmitted by the blood, as in pneumonia and septicæmia; and peritonitis sometimes arises as a complication of acute or chronic Bright's disease.

Describe the Morbid Changes.

There is first a stage of redness due to hyperæmia, most marked upon the free surfaces of the intestinal coils. This is followed by a fibrinous exudate causing a shaggy appearance of the bowel, and by an exudation of turbid fluid, which is at first serous, but tends more or less rapidly to become purulent. Acute peritonitis may be circumscribed (pelvic peritonitis, subphrenic abscess) or general.

What are the Symptoms of a Generalised Acute Peritonitis?

The disease begins with severe abdominal *pain*, sometimes at first localised, but soon spreading over the whole abdomen. It is accompanied by marked *tenderness*, the patient being often unable to bear the slightest touch, and it is increased by coughing, vomiting, or deep breathing. The breathing is therefore hurried, shallow, and thoracic in type; the pulse is quick, wiry, and incompressible; and there is moderate fever. The tongue is dry, small, and red, becoming brown in the later stages; vomiting sets in early; hiccough is frequent; and the bowels are constipated. The

face is pinched and anxious, the nose and ears contracted, and the eyes sunken. The legs are drawn up to avoid the pressure of the bedclothes, and the abdominal muscles are at first rigidly contracted, while at a later stage the abdomen is distended from accumulation of gas in the paralysed bowel (*meteorism* or *tympanites*). There is no visible peristalsis (distinction from intestinal obstruction). The percussion note is at first resonant all over; afterwards there may be dulness in the flanks from effusion of fluid.

In cases of perforative peritonitis gas escapes from the stomach or bowel into the abdominal cavity, and may intervene between the solid organs (liver, spleen) and the parietes. Hence in certain instances there is a complete disappearance of the hepatic and splenic dulness. Partial disappearance of the hepatic dulness is not uncommon when the bowel is much distended, even in the absence of perforation. The onset of perforative cases is sudden, and their course is rapid, collapse being severe and sometimes leading to death within twenty-four hours.

It is to be remembered that the symptoms of peritonitis may be masked by those of the disease it complicates. Thus in the later weeks of enteric fever there may be little pain, and diarrhœa may take the place of constipation. In other cases fever may be slight or absent. An untreated case of acute general peritonitis usually ends fatally within a week.

What are the Symptoms of Acute Local Peritonitis?

They are similar but somewhat less severe. The pain may be at first generalised, but soon becomes restricted to the area affected—appendix, pelvic organs, subphrenic area. Appendicitis and subphrenic abscess have been already discussed; but it should be recollected in connection with the latter that as it is often due to perforation of the stomach the abscess may contain air, and may be mistaken for a pyopneumothorax. The history of the patient's illness will

give the necessary clue. The fever in connection with a local peritoneal abscess is of the hectic type, and rigors and free sweating may be associated with it.

How is Acute Peritonitis to be treated?

By operation, the earlier the better. Particularly in perforative cases the prognosis depends upon the rapidity with which the abdomen is opened and the causative lesion dealt with. If operation is unavoidably delayed by distance from surgical help, refusal to accord permission, or such other cause, pain must be relieved by hot fomentations or ice applied locally, whichever is better borne. Morphia is usually needed, but should not be given to such an extent as to mask the symptoms. The bowels must be kept absolutely at rest, and nourishment must be given by the rectum, though the patient may be allowed to suck small pieces of ice for the relief of thirst. If there is much collapse intravenous or intracellular injections of normal saline are of benefit; and stimulants are often necessary.

CHRONIC PERITONITIS.

Give an Account of this Disease.

Simple chronic peritonitis may be local or general, like the acute disease. The local forms most commonly attack the capsule of either the liver or spleen (*perihepatitis, perisplenitis*), and sometimes the intestinal peritoneum. In either situation thickening is the result, and adhesions may form which, if they involve the bowel, may give rise to symptoms of intestinal obstruction. Perihepatitis and perisplenitis are to be recognised by pain over the affected organ, accompanied by friction sound or friction fremitus. In its later stages perihepatitis leads to ascites.

General chronic peritonitis, apart from tuberculosis and

malignant disease, is due most frequently to constitutional conditions such as chronic Bright's disease and to cirrhosis of the liver; or it may be part of a general infection of the serous membranes (*polyorromenitis*, *polyserositis*), in which the pericardium or pleura may suffer at the same time or subsequently. Thickening and opacity of the peritoneum are followed by shortening of the omentum and mesentery, and by the presence of adhesions binding the coils of intestine together. Fluid may lie in little pools separated from each other by such adhesions; or if adhesions are few and effusion of fluid is considerable, all the signs of ascites may be present. The *symptoms* vary accordingly. There is some abdominal pain, with anorexia and constipation. The abdominal surface is resistant to pressure, and the abdomen may be distended, with signs of free fluid in the flanks, or with a dull note all over if the intestines are prevented by adhesions from floating on the surface of the fluid. Localised areas of dulness may be found in a flat abdomen if there is little effusion. Treatment should be directed to care of the general health, the promotion of absorption by inunction of mercurial ointment or the oleate of mercury ointment, and the removal of fluid by tapping if its amount is sufficient to justify the step.

TUBERCULOUS PERITONITIS.

What are the Causes of Tuberculous Peritonitis?

The disease is most common in children, but also occurs in adult life. In children milk may be suspected as the source of infection, and in a considerable proportion of cases the bovine organism is present. It may gain admission to the peritoneum through the bowel as a consequence of tuberculous ulceration, or by way of caseous mesenteric glands. In other instances patients are the subjects of phthisis; in women a tuberculous salpingitis is frequently

present, and in males the testis or vesiculæ seminales are sometimes affected.

What are the Morbid Changes?

The surface of the peritoneum is studded with tubercles, most abundantly on the under surface of the diaphragm and in the flanks. There is an inflammatory exudate which may be fibrinous, sero-fibrinous, or serous. Where the fibrinous element predominates adhesions are numerous, the omentum becomes thickened and rolled into a sausage-shaped mass, the intestines adhere to it or to the abdominal wall, and there may be much caseous infiltration between their coils. Locular collections of serum or sero-purulent fluid may also be present. When the effusion is predominantly serous it may be considerable in quantity and free in the abdominal cavity. The mesenteric glands are often enlarged and caseous, especially in children.

Describe the Symptoms.

In some cases, and particularly in adults, the onset is acute, and the case at first resembles an ordinary acute peritonitis. The presence of tuberculosis elsewhere and the absence of the causes of peritonitis will, however, help towards a diagnosis. In another group of cases the onset is more like that of enteric fever, with diarrhœa and a gradually rising temperature, but the absence of Widal's reaction, the more irregular type of fever, and the later development of local signs should obviate mistakes.

Usually the disease develops insidiously. There is some abdominal pain; the patient wastes, while the abdomen gradually increases in size; and there is irregular fever of moderate degree. Anorexia and diarrhœa are often present. As time goes on the abdominal enlargement becomes more prominent in contrast to the general wasting; on palpation a

doughy resistance is felt over its surface, which is tender; and rounded masses can be felt in the abdominal cavity. There is often peri-umbilical thickening, and the skin in this region may be reddened; or the thickened omentum can be felt as a transverse band running across the upper part of the abdomen. Percussion gives a variable note, dull in some parts and tympanitic in others.

The above describes the *adhesive* type of case. In the *ascitic* type the prominent feature is effusion of fluid; the abdomen therefore becomes greatly distended, and the umbilicus may protrude. The central part of the abdominal surface is resonant, while the flanks and hypogastrium are dull; and the dulness changes with the changing positions of the child.

Tuberculous pleurisy is not an uncommon complication.

What is the Treatment?

It is essentially similar to that of chronic peritonitis. Mercurial inunction is of considerable value. General tonics, and, in the absence of diarrhœa, cod-liver oil, are indicated. When diarrhœa is present the diet must consist of milk, and astringents may be needed; when it has ceased, nutritious but easily digestible foods must be given. The child should be kept in bed while fever, pain, or diarrhœa is present, and there should be abundance of fresh air. If the treatment is unsuccessful laparotomy may be performed, and in the ascitic type of case it has often proved successful when medicinal treatment has failed.

CANCER OF THE PERITONEUM.

What are the Characteristics of this Disease?

It is secondary to cancer of the abdominal viscera, especially the stomach and ovaries, and is commoner in women than men. The small carcinomatous nodules are scattered

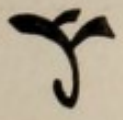
over the peritoneum, most thickly, as in tuberculosis, in the flanks and on the diaphragm; and as in tuberculosis the omentum may be thickened and drawn up. Besides the usual symptoms indicative of cachexia and the presence of a primary growth, ascites is the commonest symptom; if the intestine is involved there may be signs of obstruction. The fluid, if aspirated, is generally hæmorrhagic, and may contain tumour cells. Treatment is merely palliative.

CATECHISM SERIES

MEDICINE

PART IV

SECOND EDITION
Revised and Enlarged



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MEDICINE

PART IV.

I. DISEASES OF THE RESPIRATORY ORGANS

DISEASES OF THE UPPER AIR PASSAGES

CORYZA

What are the Characteristics of Acute Coryza?

A cold in the head is a microbic disease due to such organisms as are often normally found in the nasal cavities (pneumococcus, pneumobacillus, micrococcus catarrhalis, bacillus septus, etc.). But the determining cause is a lowering of the resistance of the subject, permitting the organisms to invade and set up inflammation of the nasal mucosa. Chill, whether due to damp feet, draughts, or sudden changes of temperature, thus usually precedes a cold. The symptoms are malaise and slight fever, followed by sneezing, headache, a watery discharge from the nose, interference with the senses of smell and taste, and obstruction to nasal breathing. In a few days the discharge becomes muco-purulent, and finally dries up, recovery taking place, as a rule, within a week. The inflammation may spread to the frontal sinuses, causing violent headache, or to the trachea and bronchi.

How should it be Treated?

In mild cases confinement to the house, a hot bath at night, and a dose of Dover's powder to produce sweating,

may be all that is necessary. In severer cases local applications such as bismuthi salicyl. ʒiv., pulv. camphoræ ʒiss., and cocainæ hydrochlor. gr. i., used as a snuff, or inhalations of adrenalin chloride (1 in 5000) are useful. Internally oil of cinnamon, or eucalyptus, may be given, and, if there is a tendency to recurrent colds, a vaccine should be prepared from the organisms of the nasal secretion.

Mention the Commoner Forms of Chronic Coryza.

Chronic coryza may lead to hypertrophic changes in the nasal mucosa and inferior turbinate bones (hypertrophic rhinitis), causing mouth-breathing and impairment of the sense of smell; or it may result in atrophy of the mucous membrane and a foul-smelling purulent discharge known as *ozœna*. It should be remembered that ozœna is still more commonly due to nasal syphilis. Antiseptic sprays or douches may be used in treatment, but these conditions are usually better left to the rhinologist.

HAY FEVER.

What is this Disease?

A severe nasal catarrh occurring paroxysmally in the spring or early summer, and set up by the floating pollen of certain flowering grasses. It is usually associated with a neurotic tendency, and may be accompanied by asthma. Chronic hypertrophic rhinitis may be present.

Treatment consists in avoidance of the country during the season of liability, the use of local applications such as cocaine or adrenalin sprays, and general tonics. Inoculations of pollen toxin have in some cases given good results.

EPISTAXIS.

In what Conditions may Bleeding from the Nose occur?

It is common, and not of serious consequence, in childhood and at puberty; and it may be due to local disease of the

nose (syphilitic or tubercular ulceration, etc.). It also occurs in many general diseases, as in heart lesions (aortic or mitral), chronic kidney disease, diabetes, cirrhosis of the liver and jaundice, severe anæmias and leukæmia, in sunstroke, as the result of sudden changes of temperature or atmospheric pressure, and at the outset of certain specific fevers (especially enteric fever), and influenza.

How is it to be Treated?

Slight cases may be met by keeping the head high and the arms raised, and applying cold to the back of the neck. In severer cases the bleeding point, usually on the septum, must be looked for, and should be touched with a plug soaked in adrenalin solution (1 in 1000). Plugging the anterior nares is often necessary, and sometimes the posterior nares must be plugged. This is done by passing a Belloc's sound or fine urethral bougie along the floor of the inferior meatus, seizing its end when it has reached the posterior pharyngeal wall by a forceps passed through the mouth, drawing it out and tying to it by a long thread a plug soaked in adrenalin, which is then pulled into place behind the soft palate by withdrawing the bougie from the nose. The end of the thread may be fixed to the cheek by adhesive plaster. Hæmostatics such as ergotin or calcium lactate may be needed, and after a severe hæmorrhage hæmatinics such as iron and arsenic. A smart purge is advisable at the outset.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

What are the Causes of this Disease?

Exposure of the larynx to cold air or to irritating vapours, extension of inflammation from other parts (coryza, bronchitis), the presence of foreign bodies, the poisons of

acute specific fevers, especially measles. Acute laryngitis may also follow chill.

Describe the Morbid Appearances.

The laryngeal mucosa is swollen and vascular; secretion is abundant, at first mucous and afterwards muco-purulent; the submucous coat is œdematous; and the inflammatory changes may cause paralysis of the thyro-arytenoid muscles.

What are the Symptoms?

Hoarseness or complete aphonia with a dry irritating cough and slight transitory fever. In adults there is no dyspnœa; in children it may be marked, and in some cases it assumes a spasmodic type, the attacks occurring during the night, and being marked by a croupy cough, stridulous inspiration, and some cyanosis (stridulous laryngitis or false croup). The attack passes off in half an hour or less, but may recur on the same or subsequent nights. The laryngoscope reveals swelling and redness of the vocal cords. Acute laryngitis tends to recovery usually within a week or ten days.

How would you treat it?

By rest, preferably in bed, and in a uniform temperature, with frequent inhalations of medicated steam. Demulcent drinks and a sedative cough mixture are useful, and the patient should speak as little as possible. In the spasmodic attacks of children hot sponges to the throat, repeated doses of ipecacuanha wine till vomiting is produced, and bromides in the intervals are the necessary measures.

ŒDEMA OF THE GLOTTIS (Œdematous Laryngitis).

Mention the Causes of Œdema of the Glottis.

It may be a complication of catarrhal laryngitis, but it is more commonly due to septic inflammations, such as

erysipelas or cellulitis extending from adjacent parts; it may complicate such acute infections as diphtheria and enteric; it may occur in laryngeal syphilis or tuberculosis; and it is not uncommon in Bright's disease. The mucous membrane and submucous coat are greatly swollen by an exudate, which may be either serous or inflammatory, according to the cause.

Describe the Symptoms.

They are chiefly those of urgent dyspnœa leading to asphyxiation. The dyspnœa may be preceded and accompanied by dysphagia from œdema of the uvula and epiglottis, the voice is hoarse and feeble, the respiration is stridulous, and the face is livid. The laryngoscope shows enormous swelling of the epiglottis, the aryepiglottidean folds, and the ventricular bands, by which the vocal cords, usually less affected, are almost hidden. The affection tends to be spasmodic, with periods in which the symptoms are somewhat less severe, and is fatal unless relieved.

What is the Treatment?

Apply leeches to the neck, or if these are not available, ice; inject pilocarpine hypodermically, and give ice to suck. If these measures fail, scarify the epiglottis with a guarded bistoury, and if this also fails intubate or perform tracheotomy. Even after relief of the œdema, death may result from the causative condition (sepsis, uræmia, etc.).

CHRONIC LARYNGITIS.

Give a brief Account of this Disease.

Chronic laryngitis may follow repeated attacks of the acute disease, or may be due to excessive use of the voice in singers and public speakers, to excessive smoking, to chronic alcoholism, or to mouth-breathing in cases of nasal obstruction. The symptoms are hoarsencss, irritation of the

throat without actual pain, frequent short cough, and hawking or expectoration of viscid mucus. The mucous membrane, as seen by the laryngoscope, is reddened and may be granular, while the cords are somewhat thickened, and may be slightly injected. *Treatment* consists in removal of the cause, the use of tonics such as iron and strychnine, rest to the voice, and avoidance of chill. Locally astringents are needed, and should be applied with the laryngeal brush; nitrate of silver (gr. x. to the ounce – grm. 0·6 to 30 c.c.) or chloride of zinc in the same strength are useful remedies. Antiseptic sprays may also be used.

TUBERCULOSIS OF THE LARYNX.

What are the main Features of this Disease?

Although very rarely primary, it is usually secondary to pulmonary phthisis. It attacks first, as a rule, the inter-arytenoid region, extending later to the arytenoid and cricoid cartilages, the ary-epiglottic folds, the ventricular bands, and the vocal cords. The affected parts are at first thickened and œdematous, and afterwards ulcerated. The ulceration may lead to necrosis of the cartilages.

Symptoms are at first merely those of chronic laryngitis, but even at an early stage a striking pallor of the palate, epiglottis, and pharynx may lead to suspicion. At a later stage dysphagia is marked, and swallowing may be extremely painful. Cough, dyspnœa, and wasting are largely due to the accompanying lung disease. Laryngoscopically the mucosa is at first pale, afterwards there is thickening of the structures mentioned above, and small ulcers may be present. Treatment includes the usual measures for the relief of phthisis, complete rest to the voice, abstinence from alcohol and tobacco, insufflation of morphia for the relief of pain, and the use in experienced hands of curettage or the galvano-cautery.

SYPHILIS OF THE LARYNX.

How does Syphilitic Laryngitis manifest itself?

In the secondary stage hoarseness or loss of voice may be due to erythema of the larynx or to superficial ulceration. The tertiary lesions take the form of gummata and deep ulceration of a phagedænic type. They usually first attack the epiglottis, where a gumma appears as a deep-red swelling, causing much thickening and deformity, and where ulcers of a serpiginous outline may extend rapidly and deeply, and lead to necrosis of cartilage, cicatricial contraction, and subsequent laryngeal stenosis. The process may extend to the cords and to the interarytenoid folds.

The symptoms are those of chronic laryngitis, with marked hoarseness; if laryngeal stenosis follows, stridor and dyspnœa may also be present. Œdema of the larynx is not uncommon. Laryngoscopically, either a gumma of the epiglottis or a more diffuse syphilitic ulceration may be found; there may be paralysis of a vocal cord; the palatal pallor of tuberculosis is absent. *Treatment* is that of syphilis in general, with appropriate local measures; laryngeal stenosis may require tracheotomy or intubation.

LARYNGISMUS STRIDULUS.

What is this Affection?

A paroxysmal form of spasm of the glottis, occurring in infants and young children, dependent upon a neurosis, and unassociated with laryngeal disease.

What are its Causes?

Any condition lowering the child's vitality, such as improper and inadequate feeding, and particularly *rickets*, which is present in three-fourths of the cases, and sometimes syphilis. The exciting cause is *reflex irritation* such as may be induced by teething, adenoids, gastric disturbances,

chill, or fright. In some instances there is a hereditary nervous tendency.

Describe the Symptoms.

The first attack usually occurs at night. The child has gone to bed apparently well, and wakes up in the night with inspiratory dyspnœa or arrest of respiration, the chest being immobile, the face cyanotic, and the heart's action rapid and tumultuous. In about a quarter of a minute or so relaxation of the spasm occurs, and air rapidly enters the lungs in a deep inspiration accompanied by a high-pitched crowing sound. The attacks may occur seldom, or may be frequently repeated at any hour of the day or night. In severe cases there may be *carpopedal spasms*, in which the hands and feet are rigid, the hands in a position of flexion and the feet bent on the extended legs. In the intervals between the attacks the child is well, but death may occur from asphyxia during the attack.

How would you treat it?

In the attack apply cold water to the head and face, or plunge the child in warm water, and apply cold water to the head. A few whiffs of chloroform may relax the spasm. In the intervals treat any constitutional disease, such as rickets or syphilis, attend to the nutrition, and give bromides in doses suitable to the age.

LARYNGEAL PARALYSIS.

What is the Nerve-Supply of the Laryngeal Muscles?

These muscles are supplied by the external laryngeal branch of the superior laryngeal, and by the recurrent laryngeal nerves, both of them branches of the vagus. The former supplies the crico-thyroid and the latter all the other muscles.

Mention the Causes of Laryngeal Paralysis.

1. Lesions of the laryngeal nerves, of the vagus, or of its nucleus in the medulla. The recurrent laryngeal may be pressed upon in the thorax (particularly the left recurrent laryngeal which curves round the arch of the aorta) by aneurysm, or by mediastinal tumour; the vagus may be pressed upon in the neck by tumours or enlarged glands, or may be attacked by neuritis, diphtheritic or influenzal, less commonly alcoholic; and the nucleus may be involved in bulbar paralysis, tumours of the medulla oblongata, and sometimes in syringomyelia and disseminated sclerosis.

2. Local lesions, the result of syphilitic, tuberculous, or malignant ulceration of the cords or of laryngeal catarrh.

3. Functional paralysis is not uncommon in hysteria.

Describe its Forms and Symptoms.

Total bilateral paralysis is very rare; it causes loss of voice and cough, and stridor on deep inspiration; the cords are fixed in the cadaveric position. *Total unilateral paralysis* causes loss or hoarseness of voice with a harsh, "brassy" type of cough; the affected cord lies in the cadaveric position, while the other is approximated to it during phonation by crossing the middle line. *Double abductor paralysis* causes marked dyspnœa and stridor, with little alteration of voice or cough; the cords lie nearly together, and are not separated during inspiration. *Unilateral abductor paralysis* causes dyspnœa only on exertion; the paralysed cord lies motionless in the middle line, and is met by the healthy cord on phonation. *Adductor paralysis* is usually hysterical; cough is perfect, there is no dyspnœa, but voice is lost, the patient speaking in a whisper; the glottis is widely open, and the cords are not approximated in phonation.

How are these Conditions to be treated?

Mainly by attention to the cause, syphilis calling for specific treatment, tuberculosis for the appropriate measures, and so on. Where dyspnœa is pronounced, tracheotomy may be required, and in hysterical paralysis faradism, either to the neck or endolaryngeal, is indicated. Local measures are the province of the laryngologist.

DISEASES OF THE BRONCHI.

ACUTE BRONCHITIS.

What are the Causes of Acute Bronchitis?

The disease is most common in winter and in damp climates; it selects by preference infants, children, and elderly people, although it may occur at any age; it is predisposed to both by insufficient nourishment and clothing and by excessive precautions against cold (warm rooms, over-abundant clothing); and diseases of the heart, Bright's disease, or previous attacks of bronchitis favour its onset. The exciting causes are exposure to cold or wet; the spread of catarrh of the upper air passages; certain infectious diseases such as measles, whooping-cough, enteric, and influenza; and the inhalation of irritating vapours. The organisms most frequently found are micrococcus catarrhalis, pneumococcus, and the pyogenic cocci.

What Morbid Changes does it induce?

The bronchial mucous membrane is hyperæmic and swollen, and desquamates slightly. At first dry, it afterwards exudes an abundant secretion, mucous at the outset, and later muco-purulent. The submucous coat may also be swollen and œdematous.

Describe the Symptoms and Signs.

The disease begins with slight fever, a feeling of rawness or oppression in the chest, usually substernal, and a cough

which is at first hard and dry and afterwards accompanied by a mucous or muco-purulent sputum. In severe cases there may be much dyspnœa with noisy wheezing respiration, but orthopnœa is seldom present unless emphysema or cardiac disease co-exists. The urine is scanty and high-coloured, the tongue furred, and the bowels costive. The acute stage passes off in a few days in favourable cases, but convalescence is somewhat slow. The disease may extend to the bronchioles or alveoli (capillary bronchitis, broncho-pneumonia), and, in elderly patients or in infants, it is often fatal from this cause. Marked dyspnœa and cyanosis, sucking-in of the lower intercostal spaces, and prostration are then the chief symptoms.

The *physical signs* are accelerated respiration with prolongation of the expiratory period, and, on auscultation, rhonchi and moist râles. The pitch of the rhonchus varies; it may be deep and *sonorous*, or high-pitched and piping (*sibilus*), according as it is produced in a large or small bronchus. It is heard both with expiration and inspiration, and, when it is very coarse, its vibrations may be transmitted to the chest wall (bronchial fremitus). The percussion note is not altered unless emphysema or collapse of the lung is present.

How would you treat a Case of Acute Bronchitis?

In mild cases keep the patient in bed in a warm room, use the bronchitis kettle, or inhalations of steam with Tr. Benzoin Co., and give a diaphoretic mixture containing Spt. Ætheris Nitros. and Vin. Ipecac. In severer cases the chest and back should be freely poulticed with linseed and mustard; antimonial wine, ipecacuan and squills should be given to promote secretion, and when this is established stimulant expectorants such as carbonate of ammonia and senega with small doses of iodide of potassium. In elderly patients stimulation is often needed, and digitalis to support

the weakened heart. In children an emetic may be of use in clearing the smaller bronchi. Inhalations of oxygen are valuable where there is much cyanosis.

CHRONIC BRONCHITIS.

How does this Disease originate?

It may follow repeated attacks of acute bronchitis, and cold damp weather predisposes to it. It is common in elderly people, and it may accompany other lung diseases, valvular affections of the heart, or disease of the kidneys. Gout, chronic alcoholism, and the constant inhalation of dust (millers, bakers, etc.), are also predisposing causes.

What are its Symptoms?

Mainly cough, expectoration, and dyspnœa, without febrile disturbance. The disease commonly recurs each winter, the intervals between attacks becoming steadily shorter, until throughout the year the patient may never be wholly free from cough. The sputum may be mucous or muco-purulent, and is often abundant. If the cough is very severe, the sputum may be streaked with blood. The *physical signs* are chiefly auscultatory-rhonchi and moist râles; but, if the disease is of long standing, signs of emphysema are present in addition.

To what Changes does it give rise?

The bronchial mucosa is often thinned, greyish or purple in colour, and its ciliated epithelium is lost. The mucous glands are dilated, the muscular coat thickened or atrophied, and the adventitia thickened by cellular infiltration. The cartilages are also atrophied or absent. Ulceration of the mucosa may occur, and this may be followed by dilatation of the bronchial tubes (*bronchiectasis*). In cases of any duration the constant coughing tends to weaken the alveolar walls, and produces atrophy of their elastic tissue and absorption of the interalveolar septa (*emphysema*). At the same time

the capillary vessels of the affected alveoli disappear, and the right ventricle has to force the blood through a lessened capillary area. Hypertrophy followed by *dilatation of the right side of the heart* is the natural consequence.

Mention the Varieties of Chronic Bronchitis.

Besides the ordinary form described above, there are:— (1) *Dry catarrh*, in which there is little or no secretion, but violent cough and much dyspnœa; (2) *Bronchorrhœa*, with very abundant secretion, either thin and watery, or like albumin-water. Symptoms are paroxysmal, and often worst on waking in the morning; (3) *Fœtid* or *putrid bronchitis*, often associated with bronchiectasis. The sputum is abundant, fluid, and very offensive; (4) *Plastic* or *fibrinous bronchitis*, a rare form, in which paroxysmal cough and dyspnœa are followed by the expectoration of fibrinous casts of the bronchial tree (tubes).

How would you treat it?

The patient's general health must be seen to; he should avoid chill or exposure, wear warm clothing, and when possible winter in a warm climate. He should be kept in a uniform temperature, and in cold weather he should have a fire in his bedroom. The diet should be light and nourishing, and the bowels must be kept regular. Any causative disease should be treated. Expectorants, such as those mentioned under acute bronchitis, are of value in the majority of cases; where the cough is dry and irritating, sedatives, such as heroin or liq. morphinae hydrochlor, may be given; and in fœtid bronchitis antiseptic inhalations such as the following:—

Menthol . . .	℥i	gm. 4
Creosoti pur. . . .	℥iij	cc. 12
Thymol	℥ss	gm. 2
Spt. Vini Rect. ad . .	℥iv	cc. 115

Morphia and other sedatives should not be given when

secretion is abundant, or when cyanosis is marked. Counter-irritation by means of poultices, mustard, or liniment of iodine is often useful. If the heart shows signs of failure digitalis is necessary. In many cases vaccines made from the organisms of the sputum are of considerable benefit.

BRONCHIECTASIS.

How is Dilatation of the Bronchi produced?

With the exception of the rare cases in which it is a congenital defect, it may be brought about either through softening of the bronchial walls in the course of inflammatory diseases, or by mechanical causes, the result of pressure or traction. In bronchitis, broncho-pneumonia, and sometimes in lobar pneumonia, the softened and often atrophied bronchial walls are liable to yield under the strain of excessive coughing, and the medium-sized and smaller tubes become the seat of a *cylindrical* or fusiform dilatation. When a large bronchus is pressed upon by an aneurysm or neoplasm, the bronchi below dilate owing to decomposition of the retained secretion, which leads to softening of their walls. In cases of phthisis pulmonalis, or chronic pleurisy accompanied by much fibrosis, the bronchial wall may be dragged upon by the contracting fibrous tissue. It tends to yield at the point of traction, and thus a *saccular* dilatation is produced. In such instances, one or more medium-sized cavities are found, usually in the lower lobes of the lungs. The walls of bronchiectatic cavities are smooth and thin, the cartilages and muscular tissue being atrophied; and at a later date ulceration of the walls is common; marked fibrosis exists around the cavities. Bronchiectasis is most common in the lower lobes.

Describe its Signs and Symptoms.

The symptoms, if the cavities are diffuse and fusiform, may be largely those of the causative disease (bronchitis,

phthisis, etc.) But when the cavities are large, and in the case of the saccular dilatation, there are usually cyanosis, dyspnœa, and clubbing of the finger tips, along with cough and expectoration. There may or may not be fever; in the late stages emaciation is common. Signs of consolidation, or of cavity formation, are present over the affected lobe. The cough is paroxysmal, often worst in the morning after the night's rest, or on change of position, and results in the expectoration of large quantities of fœtid sputum.

The sputum, if allowed to stand, separates into three layers, the lowermost composed largely of pus and containing fatty crystals, the second being greenish and serous, the uppermost brown and frothy. Its odour is highly offensive. Under the microscope pus cells, debris, crystals of fatty acids, and, if there is ulceration, elastic fibres may be seen.

The progress of the disease is slow, but complications such as septicæmia and abscess of the brain or liver may supervene.

What is the Treatment?

It is largely palliative. The strength must be supported by tonics, cod-liver oil, malt, etc. Fresh air treatment should be instituted. Locally antiseptic inhalations are beneficial, *e.g.* menthol ℥ij. (gm. 8), creosote ℥iij. (cc. 12), thymol ℥ss. (gm. 2·0), and rectified spirit to ℥iv. (cc. 115), used on a naso-oral respirator. Intratracheal injections of menthol or guaiacol are also given, and creosote may be tried internally, Surgical interference may be useful in a limited number of cases where there appears to be a solitary cavity with distinct physical signs.

ASTHMA.

Define this Disease.

It is a condition characterised by sudden attacks of dyspnœa of an expiratory type, recurring at irregular intervals, and most commonly during the night.

What are its Causes?

It occurs more frequently in men than in women. Many cases begin in childhood, but it may appear at any age. In children it often follows naso-pharyngeal disease (*e.g.* adenoids), measles, whooping-cough, or bronchitis. In adults nasal polypi, uterine troubles, gastric disorder, and an unsuitable climate may predispose to the disease. There is a marked herditary tendency, and it may be associated with such hereditary affections as gout, or still more commonly with a neurotic inheritance. Other neuroses may be present in the asthmatic individual. Exciting causes of an attack are sudden emotion, chill or draught, smoke or dust, certain odours varying in different cases, certain animal effluvia, and particular articles of diet.

What are the Main Views as to its Pathology?

There are three: (1) that it is due to a spasm of the bronchial muscles; (2) that it is caused by a hyperæmic swelling of the bronchial mucous membrane due to vaso-motor influences; (3) that there is an acute catarrh of the bronchioles (*bronchiolitis exudativa*). In any of these ways constriction of the lumen of the bronchioles would be produced, and dyspnœa would necessarily follow; but there is most evidence for the first of the three.

Describe an Attack of Asthma.

With or without premonitory symptoms, such as flatulence, sneezing, yawning, etc., the actual attack begins somewhat suddenly, and usually at night. The patient wakes distressed for want of air, with marked dyspnœa, expiration being especially laboured and wheezing in character, and respiration generally slow. The auxiliary respiratory muscles are called into play by fixation of the shoulder girdle, the patient sitting up and grasping the sides of the bed, or holding on to some article of furniture if he is on his feet.

The chest is barrel-shaped and in the position of full inspiration. The face, at first pale, may become somewhat cyanosed, and the expression is anxious. Fever is absent, and the pulse is small and rapid. This condition may last a few minutes or a few hours, and is relieved by a fit of coughing with expectoration of tenacious mucus. One paroxysm may constitute the whole attack, or successive paroxysms and remissions may prolong it over twenty-four hours.

The physical signs are as follows:—The thorax is barrel-shaped and moves as a whole by the aid of the auxiliary muscles; diaphragmatic breathing is also impeded. On percussion there is marked hyper-resonance over the whole chest, with diminution of the cardiac and hepatic dulness. On auscultation the breath sounds are feeble, but the expiratory murmur is prolonged and accompanied by loud sibilant rhonchi, which give place later on to moist râles. In the intervals between the attacks there may be signs of bronchitis, or the lungs may be apparently normal.

The sputum contains detached bronchial epithelium, spiral threads of mucus (Curschmann's spirals) twisted into little gelatinous masses, and octahedral crystals (Charcot-Leyden crystals) said to be phosphates of an undetermined organic base. There is an increase of eosinophil cells in the blood.

The asthmatic attack is not of itself fatal, but the frequent repetition of attacks sets up emphysema and aggravates bronchitis, and may thus lead to ultimate failure of the heart.

What is the Treatment?

During the attack relief may be obtained by burning powders composed of stramonium, lobelia, and hyoscyamus, or paper saturated with nitre. Anti-spasmodics such as nitroglycerin or nitrite of sodium may be given internally; nitrite of amyl or chloroform may be inhaled; or

morphia, or five minims of 1 in 1000 adrenalin solution may be injected hypodermically. In the intervals a suitable climate should be sought, some patients doing better in the country, others in town; diet should be light and easily digestible; and worry or excitement should be avoided. Of drugs iodide of potassium is the most generally useful, and should be given along with expectorants for a long time. Arsenic, cod-liver oil, and general tonics may also be tried. Any local cause (*e.g.* nasal polypi) must be suitably treated. In some cases vaccines have been of use.

DISEASES OF THE LUNGS.

EMPHYSEMA.

What is meant by Emphysema?

Undue distension of the lungs with air. The term includes two conditions: (*a*) *interstitial emphysema*, in which as the result of tracheotomy, a wound of the lung, or rupture of the air vesicles in violent cough, air escapes into the interstitial tissue of the lungs; and (*b*) *vesicular emphysema*, in which the lung alveoli are not ruptured but over-distended usually in consequence of chronic cough. The latter is by far the commoner condition.

Mention the Varieties of Vesicular Emphysema.

(1) *Compensatory* emphysema occurs when part of a lung is collapsed or consolidated, the surrounding parts expanding to fill the space left vacant.

(2) *Atrophic* or *small-lunged* emphysema results from a senile atrophy of the interalveolar septa, with coalescence of the alveoli. The lung as a whole is shrunken and pale.

(3) *Hypertrophic* emphysema, characterised by over-distension of the air-vesicles with atrophy of their walls

and consequent loss of elastic tissue and disappearance of capillary vessels. This is the common form.

What are the Causes of Hypertrophic Emphysema?

The mechanical theory of the disease is that, during the expiratory effort of coughing or expiration against resistance, there is an increase of intra-alveolar pressure. The chest wall, diaphragm, and glottis are fixed, and the tissues of the lung yield to the pressure, with the result of over-distension of the alveoli. Emphysema is therefore frequent in chronic bronchitis, in glass-blowers, players of wind instruments, and those who habitually lift heavy weights. In children it occurs as a consequence of whooping-cough, broncho-pneumonia, and similar diseases.

Another theory postulates a primary atrophy of the elastic tissue, and in support of this it is urged that in many cases emphysema is present where mechanical causes are absent. They are effective, when present, because of the change in the elastic tissue. It is in favour of this view that the affection is in many cases hereditary.

Both sexes are liable to the disease, and it is most common in the middle-aged and elderly, though it may occur in children.

Point out its Consequences.

Whether because of atrophy of the interalveolar septa due to increased pressure, or as the result of a primary change, loss of the elastic tissue is a constant feature. This leads to imperfect expiratory collapse of the lung, and to the use of the auxiliary muscles of respiration to evacuate the air. Their effort causes only an imperfect evacuation; the lung remains half-filled, and to get in sufficient air during inspiration, the auxiliary muscles are again used, tilting the ribs forwards, and increasing the antero-posterior diameter of the chest. This approximately circular shape comes to be permanently assumed, the lung is never completely

emptied, its mobility is much diminished, and attacks of bronchitis add to the respiratory difficulty.

The loss of the interalveolar septa also means loss of the capillary vessels which they carry, and consequent diminution of the area through which the blood circulates. To meet this, more forcible contraction of the right ventricle is needed, hypertrophy follows, and ultimately gives place to dilatation, leading to a relative insufficiency of the tricuspid valve. This occasions a backward flow of blood into the right auricle during systole, with consequent engorgement of the systemic veins, passive congestion of the internal organs, and œdema of the subcutaneous tissues. Heart failure is the ultimate consequence.

Give an Account of the Symptoms and Signs.

If emphysema is present without bronchitis, dyspnœa is the chief symptom, at first on exertion only, later permanently. When the heart is seriously implicated orthopnœa may be present. Cough and expectoration are due to an accompanying bronchitis, revealed by rhonchi and moist râles. On inspection the chest is seen to be circular or "barrel-shaped," and the auxiliary muscles of respiration are seen in vigorous action. The percussion note is hyper-resonant or tympanitic, and the increased volume of the lung causes it to be heard over areas normally dull. Thus the cardiac dulness is encroached on or even absent, and the upper border of hepatic dulness is lowered. The apex beat is feeble or absent, though epigastric pulsation is often conspicuous. In the later stages the heart is irregular, there are signs of dilatation of the right ventricle, and there is a systolic murmur in the tricuspid area. The liver is enlarged and tender, albuminuria is present, the face is cyanosed, and there is œdema of the feet and legs. The disease is chronic and usually progressive, but in the absence of complications life may be prolonged for a considerable number of years.

How would you treat such a Case?

By attention to the general health, regulation of diet and exercise, avoidance of chill, and when possible wintering in a warm climate. For treatment of the bronchitic attacks, see p. 257. It must be remembered that in the late stages the embarrassment of emphysema is as much cardiac as respiratory, and expectorant mixtures may be of little use without attention to the heart.

COLLAPSE OF THE LUNGS.

What are the Causes of Collapse of the Lungs?

It may be congenital (*congenital atelectasis*), when it occurs in weakly new-born children, whose respiratory movements are not strong enough to produce inspiratory expansion. It may be due to compression of the lung by pleural effusion, or by air in the pleural cavity (pneumothorax), intrathoracic tumours or aneurysms, pericardial effusion, or tumours or abscesses of the subdiaphragmatic viscera. It may be due to paralysis of the diaphragm or intercostal muscles (diphtheria, high lesions of the cord). Most commonly it is caused by obstruction to the entrance of air (laryngeal, tracheal, or bronchial obstruction; tough bronchial secretion in bronchitis; *bronchopneumonia*). Collapse may also follow a penetrating wound of the chest.

How is it produced?

In the case of compression, by simple squeezing of the spongy lung-tissue. In the case of obstruction, air which cannot escape from the air-vesicles is absorbed, and collapse follows; if the obstruction is incomplete, as from a plug of mucus in a bronchus acting as a ball-valve, air escapes during expiration, but cannot enter during inspiration, and thus the vesicles are rapidly emptied. In paralysis of the inspiratory muscles the contraction of the elastic tissue tends

to expel air, and the air which remains is absorbed by the capillary circulation.

Describe the Morbid Appearances.

The collapsed portion of the lung is dark red or purplish, airless, and dry, and lies rather below the level of the surrounding lung. On section it is firmer than the rest of the lung.

What are the Symptoms?

They usually occur in the course of some already existing pulmonary disease. If small areas are collapsed, feeble breathing and fine crepitation at the end of deep inspiration, usually at the bases, are the chief signs. If the area is larger, there may be dulness on percussion with feeble or absent breath-sounds and diminished vocal fremitus. Dyspnœa and sucking-in of the lower intercostal spaces on inspiration may also occur. In congenital atelectasis cyanosis is often present, the cry is weak, and the respiratory sounds are faint. The *treatment* is that of the cause; in atelectasis warmth and gentle friction of the chest are advisable.

ŒDEMA OF THE LUNGS.

Define this Condition and state its Causes.

Œdema of the lungs is an exudation of serous fluid into the air vesicles, terminal bronchi, and pulmonary interstitial tissue. It arises in course of valvular disease of the heart, diseases of the blood, and acute or chronic Bright's disease; also in prolonged fevers, and in paralytic conditions involving the dorsal decubitus (*hypostatic congestion*). It may occur locally in consequence of the pressure of a tumour or aneurysm, and it may be present on the side opposite to a pneumonia or pleuritic effusion. The affected lung is heavy and bulky, pits on pressure, and on section exudes a quantity of frothy blood-stained serum.

What are its Symptoms and Treatment?

Dyspnœa, sometimes orthopnœa, some degree of cyanosis, cough and expectoration of large quantities of frothy serous fluid. In *acute suffocative œdema*, which sometimes occurs in the course of chronic Bright's disease, the onset is sudden, the symptoms very urgent, and death may occur in a few hours. Physical signs are chiefly to be found at the bases, where the note may be resonant or slightly dull, the breath sounds are defective, and abundant fine râles are present.

Treatment should attack the cause; heart tonics and diffusible stimulants (strychnine, digitalis, ammonium carbonate) are necessary in cardiac cases, and in renal cases diaphoretics and purgatives. Pilocarpin is contra-indicated, as it stimulates the bronchial secretion as well as that of the skin. Atropine on the other hand, helps to check the secretion of the bronchi.

LOBAR PNEUMONIA.

Give a Definition of this Disease.

It is an acute specific infection characterised by consolidation of one or more lobes of one or both lungs, and by symptoms indicative of general toxæmia, abrupt in its onset and usually in its termination, and due in most, if not all, cases to the *pneumococcus* or *diplococcus lanceolatus* of Fränkel.

What is its Etiology?

It occurs in both sexes and at all ages, but attacks males more frequently than females, and is most common between the ages of twenty and fifty. The majority of cases occur in winter and spring. Sudden changes of temperature favour its onset, as also exposure to cold winds. Those occupations, which lead to exposure predispose to pneumonia. The liability is increased by indifferent health, and above all, by habits of intemperance. Exciting causes are exposure to

cold or draughts, or the inhalation of irritating gases. In some cases it has followed blows or injury to the chest. One attack confers no immunity from another, but rather increases the liability. Usually sporadic, the disease is occasionally epidemic, and sometimes spreads by direct contagion.

Describe the Specific Organism.

The *diplococcus* or *micrococcus lanceolatus* is a capsulated diplococcus, the free ends of the paired cocci being lancet-shaped. It sometimes occurs in chains. It is Gram-positive, and grows freely on blood-agar or blood-serum. It is found in the sputum, and often also in the blood; and it inhabits the upper respiratory passages of healthy people. Friedländer's *pneumobacillus*, a short capsulated rod, negative to Gram, and ordinary staphylococci or streptococci, may also be present.

Describe the Morbid Changes.

The affected part of the lung is usually the lower lobe, and the disease is unilateral; but the upper lobe is sometimes attacked, and both lungs may suffer. The alveolar inflammation passes through four stages :—

1. *Engorgement, congestion, or splenisation.*—The lung is heavy, dark-red in colour, pits on pressure, which causes the exudation of a frothy blood-stained serum, and breaks down readily. The alveolar vessels are injected, dilated, and tortuous.

2. *Red hepatisation.*—The lung is solid, friable, dull-red in appearance, with a granular surface resembling liver, quite airless; and it sinks in water. The alveoli are filled with a fibrinous exudation which contains red blood corpuscles, a few leucocytes, alveolar epithelium, and pneumococci.

3. *Grey hepatisation.*—The lung is still solid, but its colour is greyish, and it is less granular. Red corpuscles are few, but the alveoli and their walls are crowded with leucocytes. This with the obliteration of the capillary circulation by the pressure of the exudate, accounts for the alteration of colour. In some instances this stage may go on to *purulent infiltration*, which leads to a fatal issue; in this condition the lung is soft and yellowish, and on pressure a purulent fluid exudes from it.

4. *Resolution.*—The lung is soft, dirty grey in colour, and readily torn. The cells and fibrin undergo fatty change and are partly absorbed, partly got rid of, in the sputum. Resolution is in some cases delayed, and gangrene, chronic pneumonia, or bronchiectasis may follow.

In most cases there is a dry pleurisy over the affected areas; in some there may be considerable effusion of fluid (*pleuro-pneumonia*), and the fluid may be purulent (*empyema*) in a certain proportion of cases, particularly in children.

Other forms of pneumococcal inflammation (meningitis, endocarditis, nephritis, etc.) may occur along with the affection of the lungs. In such cases the term *pneumococcal septicæmia* is used.

In many instances there is a moderate enlargement of the spleen.

Describe the Symptoms of a Typical Case.

The onset of the disease is usually abrupt, but may be preceded for a day or two by indefinite malaise. Then follows a rigor, which may be repeated, and sometimes there is vomiting at the outset, often replaced in young children by a convulsion. The temperature rises rapidly to 103° or 104° F., or even higher, and sharp pain is felt in the affected side. The pain is aggravated by a deep breath or by

coughing. It may at first be referred to the abdominal wall, and such cases are often mistaken for appendicitis. Dyspnœa is considerable, and the respirations are rapid, their ratio to the pulse rate, which is also increased, being often as 1 to 3, or even as 1 to 2, instead of the normal 1 to 4. The pulse is full and soft, and the dicrotic wave can often be felt. Cough is at first dry and hacking, but soon becomes accompanied by expectoration of a characteristic blood-stained sputum, *rusty* in colour and extremely tenacious. The face is flushed, especially over the cheek bones (*malar flush*), and may be slightly livid; after the first few days a herpetic eruption often appears about the lips. The expression is anxious and the eyes are bright. The urine is concentrated, high-coloured, and precipitates urates abundantly; it is also deficient in chlorides, and albuminuria may be present. Leucocytosis is pronounced in favourable cases. The spleen is often enlarged. There may be some delirium in the later days of the illness, but delirium is prominent chiefly in alcoholic subjects. The condition of the patient changes little for several days. The fever is continued and the diurnal fluctuation small. Usually about the end of the first week, sometimes as early as the fifth day or as late as the ninth, the *crisis* occurs, in which the temperature, pulse, and respiration fall rapidly to normal, the symptoms abate and convalescence begins, the temperature often remaining sub-normal for several days. A *pseudo-crisis* may occur about the fourth or fifth day, the temperature falling to normal for a few hours, and returning to its former height; but in this case there is no corresponding fall of pulse and respirations.

In a considerable number of cases the termination is more gradual, and defervescence occurs by lysis, the fall of temperature being spread over several days. Diarrhœa or profuse sweating may occur about the time of the crisis.

A fatal result is usually due to heart failure or to œdema of the opposite lung. The pulse and respirations become more rapid ; cyanosis is present, and the pulse is small and weak ; the tongue is dry and brown ; the right heart becomes dilated, and muttering delirium ends in coma.

What are the Physical Signs ?

In the stage of *engorgement* the alterations are slight. The percussion note may be normal or very slightly dull. On auscultation there may be either diminution in the intensity of the respiratory murmur, or the characteristic *pneumonic crepitus*, a fine dry crepitation heard towards the end of inspiration, and produced by separation of the sticky alveolar walls.

In the stage of *consolidation* the affected lobe is definitely dull to percussion. On inspection the movements of the affected side are limited, and on palpation vocal fremitus is found to be increased. The normal vesicular murmur gives place to high-pitched tubular breathing. Crepitus may be entirely absent, or, if it persists, it has a more metallic quality than in the earlier stage Vocal resonance is increased to the point of bronchophony, and in some cases pectoriloquy is present when the patient whispers. Red hepatisation cannot be distinguished from grey hepatisation by physical examination, but in the later stages an extension of the area of consolidation is very frequently noticed.

The stage of *resolution* coincides more or less accurately with the period of crisis, the physical signs clearing up, usually rapidly, tubular breathing becoming less marked, and dulness diminishing. As the secretion becomes more fluid, crepitus returns ; it is louder and moister than the early crepitus, and is heard both on inspiration and expiration (*crepitus redux*).

The appearance of physical signs may be delayed particularly in *central* pneumonia, where the consolidation

is deeply seated. In such cases a diagnosis may often be made from the symptoms (mode of onset with rigor, rapid rise of temperature, altered pulse-respiration ratio, rusty sputum, malar flush, labial herpes), and from examination of the sputum for pneumococci. *Do not operate for appendicitis without examining the bases of the lungs.*

What are the Complications of Pneumonia?

Pleurisy, empyema, occasionally gangrene of the lung; pericarditis and endocarditis; nephritis; sometimes meningitis, but more often meningism, especially in children; in some cases jaundice. Neuritis, arthritis, and peritonitis are rare complications.

Indicate its Varieties.

It may affect both sides (double pneumonia), when it is very often fatal; it may implicate a new lobe as that first affected clears up (wandering pneumonia); it may be accompanied by early and violent delirium, particularly in alcoholic subjects; the onset may be insidious, and the fever slight in cases occurring in old age; and in many cases resolution is delayed.

How would you treat it?

The treatment is for the most part symptomatic, and though vaccine treatment is employed by some, it is as yet on its trial. The chief indications are to maintain the strength and support the heart, to relieve pain, and to procure sleep. The bowels should be opened at the outset by a mercurial purge. The diet should consist of milk, beef-tea, or broths, and food should be given often and in small quantities. Stimulant may be necessary, particularly in alcoholic patients, to whom it must be freely given; but young and previously healthy subjects do not usually need it. Should it increase the pulse rate alcohol should be stopped. Signs of heart failure should be met by the use

of digitalis or strychnine, or both, and many give digitalis in small doses from the outset. As the blood pressure is low, suprarenal extract in doses of $2\frac{1}{2}$ to 5 grains (grm. 0·15 – 0·3) may be given thrice daily. Pain is best relieved in some cases by heat in the form of poultices, in others by the application of an ice-bag. When the pain prevents sleep, morphia may be given in the early stages, but its depressant effect upon respiration makes it risky at a later period. Paraldehyde may then be substituted for it, but if milder hypnotics fail, the danger of continued sleeplessness may be greater than that of morphia. Expectoration should be promoted by stimulant expectorants such as carbonate of ammonia often given in combination with digitalis. If there is much cyanosis, oxygen may be inhaled, and, if the heart is dilated, leeches over the præcordia, or venesection may be tried.

BRONCHOPNEUMONIA (Catarrhal Pneumonia).

What are the Causes of this Disease?

It is most common in children under the age of five, and it is not uncommon in old age as a complication of exhausting diseases. It occurs as a primary affection, but is more often secondary. In children it complicates or follows the specific fevers, particularly measles, whooping-cough, and diphtheria; and it may be due to the extension of bronchitis to the air vesicles (*capillary bronchitis*). In adults it may be due to the inhalation of irritating gases, or to the aspiration of foreign bodies or septic matter from the throat into the lung (*inhalation* or *aspiration pneumonia*). It may be a frequent and serious complication in influenza. Diseases weakening the respiratory movements, such as rickets, predispose to it; so also does malnutrition, the result, for example, of infantile diarrhœa.

The organisms found are various, the most common being

Friedländer's pneumobacillus, and the pyogenic streptococci and staphylococci. The pneumococcus is also found, but is less common.

Describe the Morbid Changes.

These are usually bilateral, and they have a *lobular*, not a lobar distribution (*lobular pneumonia*). The disease begins in the terminal bronchioles, and spreads to the infundibula and alveoli. The bronchioles are inflamed, softened, infiltrated with small cells, and filled with a mucous secretion. The alveolar walls are thickened and congested, their epithelium is swollen, and it desquamates into the lumen of the alveolus, which is plugged by a mucous or mucopurulent exudate containing alveolar epithelia and abundant leucocytes. Neighbouring lobules may be emphysematous (compensatory emphysema), or if the bronchiole leading to them is plugged they may be collapsed. If many adjacent lobules are affected, the consolidated area may approach the extent of a lobe. On section the consolidated lobules present a rounded or conical form, the base of the cone being directed towards the pleural surface. They are reddish or reddish-grey in colour, solid to the touch, and separated by areas of crepitant lung tissue. Bluish-grey collapsed areas may also be seen towards the bases of the lungs.

Give an Account of the Symptoms and Course of the Disease.

The main symptoms are cough, dyspnœa, and fever, of either sudden, or gradual onset. When the disease complicates other respiratory affections or the specific fevers, the onset is often insidious; when it is primary the onset may be abrupt. The cough is short and painful, and the scanty mucous expectoration, may be streaked with blood, but is never rusty. Young children swallow the sputum. Dyspnœa is prominent, and the respirations are hurried. Cyanosis is present in bad cases, and the pulse is small, rapid, and possibly irregular. The physical signs are

variable. Dulness may be altogether absent if the consolidated areas are small, but often small patchy areas of dulness are to be found at one or both bases, with slight increase of vocal resonance; and where there is extensive consolidation, the dulness may be almost lobar in extent. Râles are usually present, moist or subcrepitant in character, and more consonating than those of simple bronchitis. Over large consolidations the breath sounds may be tubular. The lower intercostal spaces are often sucked-in during inspiration.

The disease ends by lysis, not crisis. The fever may last a week, but much oftener three weeks or more; it is remittent or intermittent in type, and relapses are common even after the normal temperature has been reached, indicating the implication of new foci of lung tissue. Emaciation is considerable, and death is often due to asthenia. Convalescence is usually slow. In old people and in the form due to aspiration the prognosis is grave.

What is the Treatment?

The child should be kept in a well-ventilated room at an equable temperature; the diet should be light and nourishing; and stimulant, in doses of 10 to 20 drops every three or four hours, according to the age or the child, is frequently required. Strychnine hypodermically is often of value. Stimulant expectorants such as ammonium carbonate, ipecacuan and senega are the drugs most commonly indicated; if the tubes are blocked by secretion an emetic dose of ipecacuan should be given. Poultices, though now out of fashion, are useful, especially in the form of jacket poultices, if not too frequently repeated; sinapisms may be applied to the chest and back; or, if breathing is much impeded, the child may be placed in a hot mustard bath for a few minutes, and respiration aided by kneading the chest. Oxygen should be given if there is much cyanosis. During

convalescence care should be exercised, the patient should be warmly clad to avoid chills, and tonics as cod liver oil, petroleum emulsion and hypophosphites given.

CHRONIC INTERSTITIAL PNEUMONIA.

(Cirrhosis or Fibrosis of Lung.)

What is meant by this Term ?

An induration of the lung tissue, arising from the gradual replacement of the normal by connective tissue. It may be localised or diffuse.

What are its Causes ?

The *local* form is common as an accompaniment of tubercle of the lung, but also occurs with tumour, abscess, or gumma of the lung, and in emphysema. The *diffuse* form follows lobar pneumonia (rarely), bronchopneumonia, acute or subacute (the fibrosis extends from the bronchi), chronic pleurisy (pleurogenic cirrhosis), or it may result from compression of a bronchus. A further form is that which is due to inhalation of various kinds of dust, and is termed *Pneumonokoniosis.* This includes *anthracosis* (coal miners), where the lung is black from carbonaceous matter, *silicosis* (stone-masons, grinders, potters), where the lung is greyish, and *siderosis*, due to oxide of iron or other metallic dusts. All these are associated with emphysema, and may be followed by phthisis.

Describe its Morbid Anatomy.

It is usually a unilateral disease. The affected lung is often enormously shrunken, hard, and airless, and the chest on that side is sunken and depressed. The sound lung is emphysematous. The heart is drawn over to the affected side, and its right ventricle is hypertrophied. In the lungs the over-growth of connective tissue causes gradual obliteration of air vesicles, changes in bronchial tubes, whose walls

become hard and yield to traction forming bronchiectatic cavities, and even obliteration of smaller blood vessels.

What are its Symptoms and Course?

It is essentially a chronic disease, and may extend over many years. Chronic cough and some dyspnœa are its chief symptoms for a time, during which health is fairly good. Later symptoms of bronchiectasis appear, and failure of right heart.

Give the Physical Signs.

Immobility, retraction and shrinking of the affected side (in striking contrast to the sound), and intercostal spaces are largely obliterated. The heart may be drawn to the affected side, and often upwards. Percussion varies with the condition of the bronchi, ranging from absolute dulness to dull tympanicity (boxy) or even to an amphoric note. On auscultation in the later stages the signs are those of extensive bronchitis, with small cavities.

What is the Treatment?

Though the condition cannot be cured, much may be done by maintaining the general health of the patient, regulation of life and work, preferably in a mild climate, and attention to bronchiectatic or cardiac conditions as they arise.

GANGRENE OF LUNG.

How may Gangrene arise?

It is not really a condition by itself, but may follow acute lobar pneumonia (rarely in healthy, more often in debilitated or diabetic cases), and more commonly aspiration pneumonia, The putrid contents of bronchiectatic or tuberculous cavities may excite gangrene in neighbouring tissues. It may arise from embolism of pulmonary artery, rarely from a simple plug, more commonly where it is septic.

Describe its Morbid Anatomy.

The condition may be circumscribed or diffuse, though the latter is rare, when it may involve the greater part of a lobe. The former is more common, when there is a well-defined area, usually in the lower lobe, and often near the periphery. Softening rapidly occurs with formation of a ragged cavity. The tissue around is inflamed and œdematous. A large vessel may be opened into with profuse hæmorrhage. The pleura may be perforated and pyopneumo-thorax result. There is usually an intense bronchitis.

What are its Symptoms and Course?

Characteristic symptoms are intense fœtor of the breath, and sputum. The sputum is profuse, contains shreddy fibres of elastic tissue, and on standing tends to separate into three layers—(1) lower greenish-brown, heavy sediment, (2) thin liquid of the same tint, (3) upper thick and frothy. It often contains blood. There is usually moderate fever with hectic tendency, rapid pulse and prostration. There are signs of bronchitis, and often of cavity. Death occurs from exhaustion or hæmorrhage.

What is its Treatment?

Treatment is unsatisfactory, though cases may recover. If a cavity can be localised, direct antiseptic injections may be tried, or surgical measures. In others, it consists mainly in keeping up the general strength, and the use of antiseptic inhalations or sprays (carbolic, creosote, or thymol).

ABSCESS OF LUNG.

What are its Causes?

It may follow inflammation of lung, lobar or lobular, but oftener it is due to aspiration in throat and nose operations, wounds of neck, and œsophageal cancer. Metastatic abcesses occur in pyæmia, and are usually small, and not localisable clinically. It may also follow perforation of

lung from without, as by foreign body, or on the right side, by hepatic abscess, or hydatid cyst, rupturing into it.

What are its Symptoms and Course?

Following pneumonia the existing symptoms are aggravated, fever becomes hectic, the sputum has an offensive odour, and contains abundant elastic tissue. There may be physical signs of a cavity. Pneumonic cases may recover, embolic are usually fatal.

What is the Treatment?

Practically as for gangrene. If the abscess is well defined and superficial, surgical measures are called for. Otherwise, keep up general strength and employ antiseptic inhalations or sprays. There is opportunity here for treatment by autogenous vaccine, prepared from the sputum.

TUBERCULOSIS OF THE LUNGS.

(Phthisis Pulmonalis.)

What Varieties are recognised?

There are three clinical groups—(1) acute, (2) chronic, (3) fibrous.

Describe the Characteristic Features of the Acute Form.

It is found in two forms, pneumonic and broncho-pneumonic. The *pneumonic* form is uncommon, and as the name implies simulates lobar pneumonia, though the lesion is usually in the upper lobe, which in whole, or in part, becomes solidified and airless. It shows miliary tubercles, few and scattered, or many closely aggregated. At times there is rapid softening with early cavity formation. Its onset is abrupt with chill, signs of consolidation, cough and sputum, which may be rusty. The alteration of the pulse-respiration ratio so characteristic of pneumonia is not marked. The fever is often more fluctuant than that of pneumonia, and crisis does not occur.

Later the sputum may show tubercle bacilli. Death may occur in two or three weeks, but more often signs of softening appear, and the case drags on for two or three months.

The *broncho-pneumonic* type ("galloping consumption") is the common variety, and is found specially in children, following measles or whooping cough. The onset may be more gradual with signs of diffuse bronchitis followed by evidence of localised dulness, usually apical, in one or both lungs. There is hectic fever, free sweating, and rapid emaciation. The sputum contains elastic tissue, and tubercle bacilli. A fatal issue is reached in a few months.

CHRONIC PHTHISIS.

What is its Etiology?

Generally speaking it is the same as for tuberculosis (*see* Part I. pp. 57-58), though here infection by inhalation is of greatest importance. Immunity is a relative condition enjoyed chiefly because of inherited tissue resistance, and is lessened by all circumstances which depress nutrition. This vulnerability of tissue, however brought about, is perhaps next to the bacillus itself, the most important factor.

Indicate the Morbid Changes induced in the Lungs.

The characteristic lesion is the formation of tubercles, whose structure and progress have been already described. (Part I. pp. 58 and 59). In most cases the primary site of the process is in the terminal bronchioles and the surrounding alveoli, which become filled with inflammatory exudate. The early changes are therefore those of peribronchitis and bronchopneumonia, and these are followed by caseation, softening, and cavity formation. A further stage may be found in more chronic cases, where around the mass is found a zone of sclerosis, in which calcium salts are deposited. Should this process become complete, the whole seat of lesion is represented by this fibrous calcified area, in which

there is no active disease. The lesion is then healed. When cavities occur they may show one of three forms (1) fresh and ulcerative, with no limiting membrane, (2) those with well-defined walls, the inner surface of which constantly produces pus, and (3) quiescent, usually small with dense fibrous tissue around them. There is involvement of the pleural sac in nearly every case, with the formation of adhesions. Effusion is common, and pneumothorax frequent. It should be remembered that the bronchial glands may be the primary focus, especially in children, but the enlargement or caseation of these is more often a secondary process.

Describe generally the Signs and Symptoms in a Typical Case.

These vary with the stage of the lesion in the lung, and are best considered in three stages, each of which has its associated train of clinical phenomena, though as there is marked variation in different cases, the classification must only be general. In the *first stage* that of *invasion*, the signs are diminished respiratory murmur, prolonged expiration, cog-wheel inspiration, with probably slight relative dulness at the affected apex. There may be no symptoms beyond persistent cough, anorexia and weakness. Fever may be present in the evening. In the *second stage*, *consolidation*, the signs are essentially those of the lesion, viz. : diminished movement, slight flattening, dull percussion, increase of vocal fremitus and resonance, and bronchial or tubular breathing. The symptoms are cough and increased debility, pyrexia especially towards evening, and commonly pain in the chest. In the *third stage*, that of *excavation*, the signs at the onset are those of consolidation with the addition of moist râles gradually merging into those of cavity, viz. : a boxy note on percussion, or the "cracked-pot" sound, whispering pectoriloquy, amphoric or cavernous breathing, and commonly large bubbling râles with metallic resonance. The symptoms

are those of the earlier stage aggravated, with great emaciation, night sweats, hectic temperature, characteristic sputum (nummular), often with hæmoptysis, and diarrhœa. Evidence of amyloid disease may be present in the liver. It should be remembered that the above typical physical signs may not all be present, or may be masked by the bronchial affection. The symptoms also vary with the mode of onset.

What is the Mode of Onset?

That of gradual invasion, but with very diverse symptoms, of which the most characteristic are—(1) anæmic and dyspeptic, (2) with chills and fever, (3) bronchitic (the "neglected cold"), (4) with hæmoptysis, (5) pleuritic, dry or with effusion, (6) laryngeal.

What is the Distribution of Lesions in the Lung?

The disease usually commences in the upper lobe, about 1-1½ inches below the apex, and nearer to the posterior and external borders. From there it spreads downwards by inhalation, commonly along the anterior borders. When the lower lobe becomes affected the seat of invasion is near its apex, opposite the fifth dorsal spine. It may extend to the other apex, but not usually before the lower parts on the same side are involved. Lesions at the base are rarely primary.

Describe the Sputum.

This varies much in amount and character at different stages. In the early stage it is muco-purulent and scanty. later it becomes more purulent and copious, with frequently greyish or greenish masses, which contain elastic tissue and tubercle bacilli. With excavation the sputum becomes nummular. The sputum may be tinged with blood or there may be a free hæmoptysis. It has a heavy sweet odour, but may be fœtid. Tubercle bacilli may be absent in the early

stages, or present only in very small numbers, but in the later stages they are present accompanied by pyogenic cocci, *i.e.* the primary pure tuberculosis ends as a mixed infection.

What are the Characteristics of the Fever?

In the early stages the temperature is usually only elevated in the evenings, but there may be almost continuous pyrexia with slight daily exacerbations. In the middle and later stages the fever is remittent with a considerable daily oscillation. With cavity formation the intermittent or hectic type may appear. The earlier fever is set up by tuberculous toxins, the later is due to sepsis from secondary infection and is commonly accompanied by hectic flush, dilated pupils, and severe night sweats.

Point out the Changes in the General Appearance of the Patient.

Anæmia is prominent from an early stage. There is a gradual and progressive emaciation, which is most pronounced in the later stages (the weight is a very good index of the progress of a case). Often there is marked muscular irritability (myoidema). The chest is frequently flattened, and the angles of the scapulæ may stand out from the ribs (alar chest). The epigastric angle is often acute, due to marked obliquity of the ribs. Later the fingers are markedly clubbed, and the nails curved.

What are the Common Complications?

Apart from the local conditions, pleurisy and pneumothorax, already referred to, these are largely due to extension of the disease to other parts of the body, viz.: laryngitis, peritonitis, meningitis, enteritis, and possibly nephritis, or a general tuberculosis. Amyloid disease in liver and spleen may occur late in the disease, and at times venous thrombosis or ischio-rectal abscess, with fistula-in-ano.

What is meant by Fibroid Phthisis?

It is a somewhat unusual form in which the interstitial lung tissue is more implicated than the parenchyma. The morbid changes, symptoms, and physical signs are almost identical with those already described under chronic interstitial pneumonia (which see). The essential difference is that in fibroid phthisis tubercle bacilli may be found in the sputum.

Where the Tubercle Bacillus is not found in the Sputum, what additional Means of Diagnosis are available?

(1) *X-ray examination* of the chest is often helpful in showing fixation, or diminished movement of the diaphragm on the affected side. It may also show "mottling" or blurring over the site of lesion.

(2) *The injection of old tuberculin* (T.O.A.), induces a febrile reaction in tuberculous subjects. The initial dose is 0·01 cc. and if no reaction follows 0·05 cc. should be injected three days later. If still there is no reaction 0·1 and 0·5 c.c., can be given at similar intervals. A negative result may then be taken as proof that the condition is not tuberculous. This test is not advisable in febrile cases, or where the diagnosis can be otherwise made.

(3) *Tuberculo-opsonic index.* In health this varies between ·8 and 1·2. Indices persistently above or below these limits point to tuberculosis.

(4) *Von Pirquet's cutaneous reaction.* This consists practically in vaccination of the patient with a drop of tuberculin. An inflammatory reaction follows in the tuberculous. A control inoculation through sterile saline solution should be made at the same time. This reaction is most valuable in children, as it occurs in healed as well as active tuberculous lesions.

(5) *Moro's percutaneous reaction*, a modification of the above. Here a tuberculin ointment is rubbed into the skin, and produces reaction or a small localised ulcer in the tuberculous. A control rubbing should also be carried out with a simple ointment.

(6) *Calmette's reaction.* This method is now largely in disuse, as it has been followed by very severe reaction and disastrous results. A solution of the precipitate obtained by treating tuberculin with absolute alcohol is used. One drop of this is instilled into the conjunctival sac, and sets up inflammatory reaction within 24 hours in tuberculous subjects.

Outline the Treatment of Phthisis Pulmonalis.

This may be considered in three parts, viz. preventive, general, and medicinal.

To *prevent infection* of others the patient must be educated in the disposal of the sputum. When indoors he should spit into paper or rags which should thereafter be burned, and for outdoor use he should carry a glass spittoon. This can readily be sterilised by boiling water, after its contents have been burned. The introduction of compulsory notification, and the institution of dispensary and other special departments for the observation and supervision of the tuberculous, form a great step in the prevention of infection.

General measures.—Fresh air and sunshine are the chief aims. The consumptive should be in the open air as much as possible by day, short of fatigue, and should sleep by night with the windows widely open. When there is fever he should be kept in bed, but if possible in the open, or in a large airy room. The open windows should be insisted on even in the presence of cough, fever, and sweats, though then with added care to avoid chill. *Sanatorium* treatment offers

the best chances of success along these lines, as general measures can be combined if required with those specially indicated by symptoms. Many early cases have thus been cured, while others, unless too far advanced, show marked improvement. *Climatic* treatment is not within the reach of all, and many places are available for selection. The essentials of a suitable climate are a pure atmosphere, an equable temperature, and a maximum of sunshine, with facilities for the open air life. Advanced cases should never be sent from home, and those with feeble hearts or tendency to hæmoptysis, should not be sent to high altitudes. Associated with open air treatment, suitable clothing should be worn, and proper diet chosen. Where digestion is good the *diet* should be nourishing, varied, and ample, with plenty of butter and fats. Systematic overfeeding has its advocates. Unfortunately difficulty often arises from anorexia, nausea, and dyspepsia, for which diet must be modified. Exercise and work, carefully regulated and graduated, are of value in promoting regular auto-inoculation.

Specific and medicinal treatment. — The former in incipient cases, used cautiously, has been frequently successful. T.R. or the bacillary emulsion should be used, and results should always be checked by opsonic estimations. The initial dose should be very small. The mixed infections of the later stage may be benefited by a suitable autogenous vaccine. Any specific treatment should be regarded as an adjuvant to the open air life, and must not supersede it. Of medicines the principal remedies in use are tonics and antiseptics. Cod liver oil, malt, hypophosphites, and arsenic, with creosote or guaiacol are those commonly employed.

Name the Symptoms which may call for Special Treatment.

Cough, if persistent and constant, may prevent sleep, or cause vomiting. For laryngeal and bronchial irritation, a spray of menthol and chloretone in paraffin, or inhalations of

tincture of benzoin or creosote may be valuable. A useful sedative combination is that of morphia, spirit of chloroform, and dilute hydrocyanic acid.

Night sweats are best controlled by a nightly pill, containing atropin gr. $\frac{1}{120}$ to $\frac{1}{60}$. Picrotoxin, zinc oxide, and aromatic sulphuric acid are also employed.

Fever should be treated by rest, fresh air, and tepid or cold sponging. Antipyretic drugs should be very cautiously used.

Diarrhœa.—For this troublesome symptom the astringent sedative combination of lead and opium in pill is most reliable treatment. Large doses of bismuth with Dover's powder may be useful. Starch and opium enemata may be of benefit in allaying local irritation.

Hæmoptysis—for treatment see under this heading.

What Recent Line of Treatment has been adopted in cases with Limited Lesion?

The establishment of an artificial pneumothorax on the affected side, nitrogen gas being introduced into the pleural sac, with the object of producing collapse of lung, and thus absolute rest to that side. As the gas at first is rapidly absorbed, it must be reintroduced at increasing intervals. In suitable cases, symptoms rapidly diminish, connective tissue proliferates, and the tuberculous lesion may thus heal. The pneumothorax must be kept up for one or two years.

TUMOUR OF THE LUNG.

Name the Varieties met with.

Carcinoma is the most common form, but sarcoma and endothelioma also occur. Primary tumours are rare, more often the condition is secondary to malignant growths elsewhere in the body. The common primary sources are in the bronchial glands or liver. A primary cancer or sarcoma

usually involves only one lung, while the secondary occurs in both. The former commonly occurs as one large mass, the secondary is disseminated in the form of dense hard nodules, or soft masses throughout the lung tissue. The cervical or supraclavicular glands are usually affected secondarily.

What are the Symptoms?

They are by no means distinctive, especially in the primary form. *Pain* in the chest is common, more marked when there is pleural involvement. Dyspnœa is variable, but apt to be paroxysmal where the trachea is compressed. There is often a dry and painful cough, with a blood-stained sputum resembling "red currant jelly." There may be various pressure symptoms, and the heart may be displaced to the opposite side. *Fever* is present in a number of cases. *Emaciation* is not necessarily extreme.

Indicate the Physical Signs.

Those of consolidation, or of fluid effusion. There may be diminished breathing from pressure on a bronchus. Clavicular or cervical glands are usually enlarged.

Mention the Important Points in Diagnosis.

In secondary growths diagnosis is not usually difficult. In the presence of, or following, a primary lesion, the onset of pulmonary symptoms is strongly suggestive. In the primary form its unilateral nature, the anomalous signs, the sputum, wasting, and involvement of cervical glands are important.

HÆMOPTYSIS.

What is meant by this Term?

It means literally the spitting of blood.

What Causes give rise to it?

It is a symptom of many diseases, of which the following are the chief:—phthisis pulmonalis, or other lung conditions

as pneumonia, cancer, gangrene, abscess, and bronchiectasis; heart affections, especially lesions of the mitral valve; ulceration of the respiratory tract, and aortic aneurysm. It may be due to injury, as from fractured ribs, or to severe coughing as in bronchitis occasionally. It may occur in purpura or malignant fevers consequent upon altered blood conditions. Rarely it is the expression of vicarious menstruation.

What are the Nature and Appearances of the Hæmorrhage?

The onset is usually sudden, the patient is aware of a warm saltish taste in the mouth, and coughs up blood, which is usually bright-red, aerated, and alkaline in reaction. It is mixed with the mucus of the sputum. The sputum may remain blood-tinged for a few days. There is no melæna, unless sufficient blood be swallowed.

It should be remembered that this condition may be simulated by hysterical patients or malingerers.

How would you distinguish this from Hæmatemesis?

Several points are valuable, but are not always conclusive. They can be tabulated thus :—

Hæmoptysis.	*Hæmatemesis.*
1. Previous history of pulmonary troubles.	1. Previous history of gastric disorders.
2. Blood is coughed up.	2. Blood is vomited.
3. Blood is frothy, bright-red, and alkaline in reaction.	3. Blood is not frothy, often dark, and clotted, and is acid in reaction.
4. Blood is usually mixed with sputum.	4. Blood is commonly mixed with food.
5. There is often dyspnœa, with pains in chest.	5. There is nausea, with epigastric fulness.
6. Usually no melæna.	6. Often followed by melæna.

What is the Treatment?

The bleeding usually ceases spontaneously, unless a large artery is opened, therefore the patient and friends should be reassured. Our aim is to reduce blood-pressure, and to favour the formation of a thrombus of sufficient strength to prevent further bleeding. Absolute rest and quiet should be insisted upon, the diet should be light and given cold. Ice is given to suck, or may be applied to the chest. Morphia should be given hypodermically, and beyond this drugs are rarely necessary. The inhalation of amyl nitrite is valuable as an immediate remedy. When the case is protracted, saline purgatives are employed to keep the pressure low, and aromatic sulphuric acid is given in small doses to allay thirst.

PULMONARY INFARCTION.

(Hæmorrhagic Infarction-Pulmonary Apoplexy.)

How is this produced?

It is in most cases due to embolism of a branch of the pulmonary artery, and is most common in chronic cardiac cases. Though the pulmonary vessels are end-arteries, blocking does not always cause infarction, as the wide capillaries provide anastomoses, and the bronchial arteries aid in keeping up the circulation.

Describe the Appearance of an Infarct.

It is a wedge-shaped mass at the periphery of the lung, with its base towards the pleura. When recent it is dark-red in colour, hard and airless, and the pleura over it is inflamed. Its size varies from that of a walnut upwards, and it may involve the greater part of a lobe, more commonly the lower lobe. Infarcts are usually multiple. Later, the infarct may possibly be absorbed, or more commonly becomes fibrosed with puckering. Occasionally gangrene ensues with cavity formation.

What are the Symptoms and Signs?

Dyspnœa, pleuritic pain, and hæmoptysis (the blood is dark). There may be pleuritic friction, and signs of a limited consolidation. The respiratory murmur may, however, be feeble.

When a main artery is blocked, sudden death ensues.

DISEASES OF THE PLEURA.

PLEURISY.

What is meant by this Term?

It is an inflammation of the pleural membranes, and may be primary or secondary, acute or chronic, dry or with effusion.

Give the Causes of this Condition.

The *primary*, or so-called idiopathic form may arise apparently from chill, but is more often tuberculous in origin, and may indeed be the first symptom of this disease.

The *secondary* form may arise from numerous causes, the chief of which is pneumonia. Other intra-pulmonary affections, as tubercle, cancer, etc., may cause it, or it may be set up by extra-pulmonary conditions as by injury, extension of abscesses (axillary, subdiaphragmatic, etc.) to pleura; general diseases (especially rheumatism and scarlet fever); or it may occur in the later stages of chronic diseases (Bright's, hepatic cirrhosis, cancer, etc.). Chill is a frequent determining cause.

What Organisms are most commonly found in Pleurisy?

The pneumococcus, streptococcus, and tubercle bacillus.

Describe its Morbid Anatomy.

Various stages may be recognised :—

(1) *Hyperæmia*, the membrane loses its lustre, and becomes dry and red.

(2) *Exudation of Fibrin*, which gives a shaggy appearance to the membranes. In dry pleurisy, these surfaces then adhere, and these adhesions permanently obliterate the cavity in whole or in part, though some cases appear to resolve completely.

(3) *Fluid Effusion.*—The usual exudate is sero-fibrinous, greenish-yellow in colour, with floating flakes of lymph. Its specific gravity is 1010 to 1020, and it is rich in albumin. In malignant or tuberculous pleurisy the fluid may be hæmorrhagic.

(4) *Resolution* with permanent adhesions of varying extent. Instead of resolving, the fluid may become purulent (empyema). Pneumococcal pleurisies are often purulent from the outset.

The effusion causes certain effects by pressure, if in quantity. The lung is collapsed, in part or in whole. The heart is often displaced to the right when there is left-sided effusion. In extreme cases the mediastinum is displaced to the opposite side. There is bulging of the affected side with downward displacement of liver or spleen, and diaphragmatic movement is much embarrassed.

The fluid is absorbed in from one to three weeks. If absorption is unusually slow, empyema should be suspected.

What are the Symptoms?

Its onset is ushered in by slight or repeated chills, and moderate pyrexia, with severe pain in the side, aggravated by respiration, or any movement or pressure. When effusion occurs the pain is replaced by dyspnœa. The patient tends to lie on the affected side where effusion is abundant. Respirations are hurried and shallow, the pulse is quickened, and may be irregular. There may be a short, hacking cough, with slight mucoid sputum, never rusty unless there

is associated pneumonia. Cyanosis may be present when effusion is abundant.

What are the Physical Signs?

These vary with the stage of the disease. While fibrinous, palpation may yield friction fremitus, and on auscultation the characteristic friction-rub is heard, synchronous with respiration, and of a leathery-creaking quality. In the stage of effusion, *over the affected area* movement is diminished, and the lower intercostal spaces bulge, percussion is absolutely dull and board-like, and there is absence of breath sounds, vocal fremitus, and vocal resonance. In the erect posture the line of dulness is curved, being higher behind and laterally than in front, but where effusion is abundant the upper level is horizontal. The area of dulness extends beyond the normal limits of the lung, and on the left side it encroaches on the crescentic space of Traube, this giving a useful distinction between pleurisy and pneumonia. At the *upper limit of effusion*, where the voice is transmitted through condensed lung and a thin layer of fluid, it has a peculiar quality termed ægophony (goat-voice). *Above the effusion* the signs vary with the amount of pressure. There are diminished movement, increased vocal fremitus and vocal resonance, Skodaic resonance, and weakened breath sounds (may be distinctly tubular). In such cases Grocco's sign is important, viz., the presence of a triangular area of dulness close to the spine at the base of the opposite lung. The apex points upwards. This area is due to the displacement of the mediastinum by fluid.

The heart is displaced to the unaffected side, and the liver or spleen downwards.

How may the Diagnosis be confirmed?

By exploratory puncture. Further the examination of the cellular elements of the fluid (cytodiagnosis) helps to

determine the character of the pleurisy. Excess of lymphocytes indicates tubercle, while the prevalence of polymorphonuclear cells indicates simple imflammations.

What is the Course and Prognosis of the Disease?

The fever subsides by lysis during one to three weeks, and slight effusions are rapidly absorbed, while those larger disappear slowly. Death is infrequent in uncomplicated pleurisy, though where effusion is copious sudden death from syncope may occur as the result of pressure. The fluid may become purulent (empyema) as indicated in typical cases by hectic fever, rigors, sweating, and leucocytosis. Exploratory puncture furnishes the only certain indication. The prognosis is then much more unfavourable. Where empyema remains untreated the pus may burrow anywhere, though it usually ruptures into the lung, or may open externally, commonly on the chest wall, in front between the 3rd and 6th space.

What are the Main Causes of Empyema?

It arises frequently from septic invasion of a simple effusion. An effusion in the course of infective fevers or pyæmia almost always becomes purulent. A pneumococcic pleurisy is usually purulent from the outset. It may arise from local causes as fracture of rib, or penetrating chest wounds, or from perforation from tuberculous cavities.

Describe the Treatment of Acute Pleurisy.

In dry pleurisy relieve the pain by poultice or blister, if need be by morphia. Fixation of the side by strapping often gives relief. In pleurisy with effusion, a fever diet with limitation of fluids should be adopted, and a free purge administered. With moderate exudates absorption should be encouraged by the external use of liniment of iodine, or mercurial ointment, and internally by a daily

saline purge of concentrated magnesium sulphate. The latter may prove rather exhausting to weakly patients, and diuretics may be given instead. If the fluid is not rapidly absorbed (in two to three weeks), aspiration or puncture is called for, and repeated if necessary. Remember that the longer aspiration or puncture is delayed, the lung is the less likely to expand satisfactorily. The fluid should be drawn off at once if there is interference with breathing, or evidence of serious pressure on the heart, and always if the dulness in front reaches as high as the second rib.

During convalescence, nourishing diet and tonics are necessary, and respiratory exercises to promote lung expansion should be employed early.

How is Empyema treated?

The treatment should be surgical and immediate to provide free drainage. Healing may be promoted by the use of its appropriate bacterial vaccine. Tonics and respiratory exercises should follow operation.

HYDROTHORAX.

What is meant by this Term?

It denotes the effusion of fluid into the pleural sac as the result of a passive exudation. It is a dropsy of the pleuræ, and a non-inflammatory condition.

What are its Causes?

Cardiac or renal disease, and sometimes grave anæmias.

How does it differ from an inflammatory Effusion.

There are no preceding symptoms of inflammation. It is usually bilateral. The fluid is always serous, and tends to gravitate with position. It is painless, but may cause greater dyspnœa. It is associated with dropsy elsewhere. Aspiration only temporarily relieves the pressure, and must usually be repeated.

What is the Prognosis, and how is it treated?

The prognosis and treatment are those of the causative disease.

PNEUMOTHORAX.

What does this Term denote?

It means the presence of air in the pleural sac.

What are its Causes?

It may arise from punctured wounds of chest wall, or from laceration by the end of a broken rib. From the lung side the commonest cause is perforation from a tuberculous cavity, or the breaking of a caseous focus. Similarly it may arise from gangrene or abscess of the lung. It may also be caused by rupture of emphysematous air vesicles, or of an empyema into a bronchus.

What are the Symptoms?

They depend largely on the cause, and the amount of air present. Usually there is sudden severe pain at time of rupture, with or without collapse, urgent dyspnœa, a rapid and feeble pulse, with slight lividity, and shallow breathing. In long-standing phthisis cases there may be no urgent symptoms.

Mention the Physical Signs.

These are usually distinctive. There is bulging and impaired movement on the affected side, and the cardiac impulse is often much displaced. Vocal fremitus is absent. Percussion gives a hyper-resonant or tympanitic note, or, if the pleural opening be patent, a cracked-pot sound. The breath sounds and vocal resonance are diminished or absent, and if present are distantly amphoric. There is metallic tinkling on coughing, and the characteristic bell or anvil-sound can be elicited with the aid of coins. Later, fluid may gather at the base, either serofibrinous (hydropneumo-

thorax) or purulent (pyopneumothorax) as the result of inflammatory changes, and an additional sign appears, that of hippocratic succussion (splashing sound on shaking the patient.)

What is the Prognosis?

This largely depends on the cause. In the healthy, recovery often occurs by healing of the opening, and absorption of the air. In phthisis death usually follows in a few weeks, though cases are on record where pneumothorax was followed by arrest of the symptoms.

Give the Treatment.

Relieve the pain by morphia hypodermically, or by hot poulticing. Combat the shock by stimulants. If pressure is great, perform paracentesis for its relief. In pyopneumothorax incision and drainage should be carried out, unless where there is advanced pulmonary disease.

DISEASES OF THE MEDIASTINUM

Mediastinal Tumours.

What is the Nature of these Growths?

They are most common in the anterior mediastinum, and originate in the bronchial or mediastinal glands, connective tissue, remains of the thymus, or in the pleura and lung. Carcinoma occurs but is always secondary. Sarcoma, lymphosarcoma, and lymphadenoma are the usual forms found, though gumma may occur. They are most common in males between the ages of thirty and forty.

Describe the Symptoms.

Generally there is pain under the sternum or posteriorly which may radiate, irregular fever and emaciation with

symptoms of pressure, viz. dyspnœa from pressure on trachea or bronchi, cyanosis and œdema from pressure on the large veins, which also gives rise to distension of the external jugulars and surface veins of chest, head, and arm. There may also be pressure on the recurrent laryngeal nerve (left cord paralysed), on sympathetic (pupils unequal), or on vagus (rapid pulse).

What are the Physical Signs?

Manubrial dulness is the chief, but pleural effusion may occur. Heart, lung, or liver may be displaced, and the cervical glands enlarged.

Give the Differential Diagnosis between such a Tumour and Aneurysm.

The absence of auscultatory phenomena and pulsation, with little pain, point to tumour, as also do enlarged cervical glands or new growths elsewhere. Tracheal tugging is rare with tumour, which also implicates veins more than arteries. A case with a prolonged course (over a year) is unlikely to be tumour.

What is the Treatment?

This can only be palliative for symptoms. The outlook is hopeless.

DISEASES OF THE CIRCULATORY SYSTEM

I. THE PERICARDIUM

PERICARDITIS

Define the Term.

Inflammation of the pericardium.

Under what Conditions does it occur?

It may arise as the result of infective processes, by direct extension of inflammation, or from constitutional diseases. It may also result from injury by penetrating wounds from without or within. It is never idiopathic.

Mention the Causes.

It is met with as a secondary process in various affections:—

(1) Rheumatic fever and chorea furnish the majority of cases—30 to 70 per cent.

(2) Septic processes—part of a general septicæmia or pyæmia, or in puerperal fever, or with acute necrosis of bone.

(3) Infective fevers, especially scarlet fever and measles, less often in pneumonia, influenza, variola, or enteric fever.

(4) Tuberculosis, usually part of a general involvement of serous sacs, or by extension from lung.

(5) Constitutional diseases: chronic Bright's, malignant disease, gout, scurvy, diabetes.

(6) Extension from contiguous organs—in pleurisy, pneumonia, and ulcerative endocarditis, or from bronchial glands, ribs, sternum, or vertebræ.

What are the Organisms most often present?

Apart from the rheumatic cases, the pyogenic cocci and pneumococcus are most often found.

Describe its Morbid Anatomy.

In the usual form—*fibrinous*—there are various stages:—

(1) Hyperæmia, with loss of lustre.

(2) Exudation of fibrin, in a thin coating, roughening the surfaces, or more markedly with shaggy, honeycomb appearance, ("bread-and-butter sandwich"). The process may go no further than this dry or plastic type, but often it goes on to—

(3) Effusion of fluid, serous or sero-fibrinous.

(4) Absorption with varying amount of adhesion.

The myocardium generally shares in the inflammatory change, and the mediastinal tissues may also partake (mediastino-pericarditis). In tuberculous or malignant disease the effusion may be hæmorrhagic, while in septic infections it is usually purulent from the outset.

What are its Symptoms?

These may be few, or may be masked by those of the causative disease. In a typical case there is fever of moderate degree, variable pain, præcordial or anginal, rapid pulse with some dyspnœa; later marked præcordial distress, dyspnœa, great restlessness, and feeble irregular pulse, as the fluid accumulates. Pressure symptoms on various organs may be present.

Give the Physical Signs.

During the dry stage the characteristic friction rub is heard. It is a double "to-and-fro" rub, often with a peculiar grating or creaking quality, and sounds superficial

and close to the ear. It is of variable intensity, and though it corresponds to systole and diastole it is not accurately synchronous with the heart sounds. It is often best heard over the right ventricle, though audible all over the præcordia, and, unlike murmurs, it has no definite line of transmission. When coarse it may be felt by hand (friction fremitus). It lessens *as effusion appears*, though it often persists at the base. With considerable effusion there is præcordial bulging, displacement of organs, and great increase in percussion dulness, the area being irregularly pear-shaped with base downwards. The heart sounds are weak and muffled (largely due to myocardial weakness). As the fluid absorbs the friction rub reappears.

What is its Course?

Mild cases quickly subside. Where effusion is present its limits should be marked out each day. Some reach the maximum in two days, others may gradually increase for a week or two. Absorption may be rapid, with formation of adhesions. Septic cases are often rapidly fatal, and those with effusion in renal disease or pneumonia. In serofibrinous cases recovery is the rule.

Distinguish between Pericarditis and Cardiac Dilatation.

This is often difficult, but the limits and shape of the dull area differ, and the cardiac sounds in the latter are not muffled, but clear and often sharp.

What are the Signs of Adherent Pericardium?

These may be indefinite, but characteristically several are present:—

1 Great hypertrophy, and dilatation of heart, with præcordial bulging.

2 Systolic retraction at the apex, or at lower sternum, followed by—

3 Diastolic rebound of the chest wall.

4 Collapse of the cervical veins during diastole (Friedreich's sign.)

5 Pulsus paradoxus, pulse small and feeble with inspiration.

None of these signs is pathognomonic by itself.

Describe the Treatment of Pericarditis.

The first essential is absolute rest in bed to reduce cardiac action. Ice bags to the præcordium may relieve pain and retard effusion. In robust cases local cupping or leeches may be beneficial. Morphia may be necessary. The cause should be treated, though depressant drugs should be avoided or used with care, *e.g.* in rheumatism. The diet should be light, dry and nutritious. When effusion is present promote absorption by local blister, saline purgation, and diuretics. If these measures fail, or if there is cardiac embarrassment, paracentesis should be performed, in the 4th or 5th left interspace one inch from sternal border. Should the fluid be purulent, incision and drainage is the treatment.

What other Effusions may occur in the Pericardial Sac?

Hydropericardium a simple dropsical effusion due to cardiac or renal disease, part of a general dropsy.

Hæmopericardium, in the course of tubercle, cancer or Bright's disease, or due to aneurysms of first part of aortic arch, heart, or coronary arteries. It may be due to wounds of heart.

Pneumopericardium, a rare condition due to penetrating wounds from without or within, and usually combined with acute purulent pericarditis ere long.

II. THE HEART.

ENDOCARDITIS.

What is meant by this Term?

Inflammation of the lining membrane of the interior of the heart, but usually confined to the valves.

In what Forms does it occur?

It may be acute or chronic. The acute may be simple and benign, or malignant and ulcerative.

Acute Endocarditis.

Give its Etiology.

It is rarely a primary disease, usually secondary to other affections. The causes are very similar to those of pericarditis. The *simple* form is closely associated with rheumatism and chorea, common in scarlet fever, though uncommon in other fevers, frequent in pneumonia, and may occur in debilitating diseases, *e.g.* phthisis, Bright's disease, diabetes, or gout. The *malignant* type may occur without discoverable cause, but is usually the result of some septic or infective condition as scarlet fever, gonorrhœa, puerperal sepsis, pneumonia, septicæmia, pyæmia, and influenza. It is also associated with the rheumatism-chorea group, though much less closely than the simple form.

What is the Morbid Anatomy?

In the simple form there is some œdematous thickening of the valvular lining, followed by erosions, and the formation of small granulations, upon which fibrin is deposited. Thus small warty vegetations are formed, not on the free margin of the valves, but along the lines of maximum contact. In course of time these become transformed into fibrous tissue. In the *malignant* type the initial changes are

similar, but ulcerations may completely replace the vegetations, which are larger and fungating especially in pneumococcal and gonococcal infections. The tissues beneath are necrotic and contain abundant micrococci (organisms are scanty in the simple form). The vegetations are more liable to be detached, forming septic emboli. There is great destruction of valves, which may show small aneurysms, ruptured or unruptured, in their cusps, or perforations. There may be mural infection with similar appearances in the walls of the heart. This type may affect both sides of the heart, though the left more severely (the simple attacks the left only, congenital endocarditis, the right). The myocardium always shares in the inflammation to a varying degree.

Mention the Symptoms of Simple Endocarditis.

These are not always characteristic or well marked, and are often masked by the primary disease. The condition may be discovered during routine examination. Increased rapidity of pulse, præcordial pain or discomfort, palpitation or dyspnœa may point to cardiac affection. A murmur may be present, the commonest being mitral systolic or presystolic. In most fevers cardiac dilatation with relative murmur occurs, not due to endocarditis, or there may be a hæmic murmur. In neither of them is there the sharp rise of temperature which often accompanies endocarditis. (An aortic diastolic or a mitral presystolic murmur is likely to be organic). Emboli may occur in various sites.

Distinguish between a Hæmic and an Organic Murmur.

Hæmic murmurs are always soft and systolic, best heard at the base and over pulmonic area, not definitely transmitted, and disappear under treatment. They are associated with anæmia and a venous hum in the neck, but never with cardiac hypertrophy.

Describe the Symptoms of the Malignant Form.

Various types are described:—(1) *Typhoid*, with irregular or intermittent temperature, diarrhœa, petechial rashes, and a rapid onset of the typhoid state, thus closely resembling typhoid fever, or perhaps meningitis; (2) *Septic*, characterised by symptoms of septic infection, *e.g.* rigors, sweats, oscillating pyrexia, emaciation, and metastatic abscesses. This type may be acute or prolonged for months. In these the cardiac symptoms are in the background, but in (3) *cardiac* group the cardiac features are pronounced. Here the symptoms of an acute infective process often appear in the course of a chronic valve lesion. In such cases death may be rapid, or the course prolonged. Recovery may occur after a protracted illness. These general symptoms are usually accompanied by a cardiac murmur or murmurs, irregularity, dilatation, etc., though these may be latent, rendering diagnosis difficult. Septic emboli or metastatic abscesses may occur.

What Features distinguish it from Enteric Fever?

In endocarditis the murmur or murmurs vary from day to day, the blood shows a leucocytosis, and blood culture may give a positive result. In enteric there is a leucopenia, and the Widal reaction is present.

Name the Organisms most commonly found in the Malignant Type.

It is pre-eminently a *streptococcal* disease, and if the *pneumococcus* be included, this group includes over 70 per cent. of all cases. The *influenza* bacillus has been found in quite a few cases. In primary surgical affections *staphylococci* (especially aureus) are found. The *streptococci* met with are commonly those of low virulence, viz.:—salivarius, anginosus, and fæcalis (Horder).

What is the Treatment of Acute Endocarditis?

All forms call for absolute rest, prolonged as required. The primary disease should be treated, and depressant remedies avoided or used with care. Milk or light diet should be given, the bowels regulated, and sleep secured. There is no need for direct treatment of the heart unless it shows signs of failure, then, and only then, should digitalis be employed. The malignant form should be treated like septicæmia. (It really is a septicæmia with a local endocardial lesion.) If the organism can be isolated from the blood an autogenous vaccine may be tried. Stock vaccines or sera are of little use. Whatever treatment be adopted the end is almost uniformly fatal.

CHRONIC ENDOCARDITIS.

Define this Disease.

It is a chronic inflammatory process resulting in sclerosis of the valve curtains. It may be primary, but is oftener secondary to acute form, and especially with rheumatism as the primary agent. Other infections, which cause acute, may also set up the chronic form. The primary form is really a *valvular degeneration*, the result of changes set up by syphilis, alcoholism, gout, chronic Bright's disease, and vascular overstrain. Most of those are associated with high blood pressure, and hence with arterio-sclerosis and atheroma. The valve lesion often follows degenerative changes in the aortic arch, and thus the aortic valve is the usual site.

How do Age Incidence, and Site of Lesion vary with the Causative Disease?

Rheumatic endocarditis most often attacks the mitral valve, and in early adult life. The degenerative types are most common in middle life, and affect the aortic oftener than the mitral valve. Syphilitic disease of valves may arise either in early or later life.

What is the Morbid Anatomy?

There is first thickening and stiffening of the cusps, often with small, firm, warty vegetations (nodular) at their margins. The whole valve may be affected. The fibrous tissue contracts, producing deformity of the segments which cannot be properly closed, and the valve becomes incompetent. This deformity, or adhesions between cusps, may cause obstruction. Incompetence and stenosis may thus coexist. In the mitral valve the cusps often adhere along the whole length of their margins, producing great narrowing of the orifice. The thickening and deformity may extend to the chordæ tendineæ and musculi papillares. Atheromatous changes or calcification may follow.

Describe the Effects of Chronic Valvular Disease on the Heart.

The normal cardiac mechanism is fully prepared to meet ordinary grades of sudden strain, by an inherent reserve of power. Where this strain is gradually applied, as with sclerotic changes, this reserve power gradually increases to meet the strain, but only for a time. More and more power is called for, and is supplied by muscular hypertrophy, thus preserving the equilibrium of the circulation, and the effects of the valve lesion are "compensated." A point is reached when the exhausted muscle yields to the strain, and dilatation becomes excessive, compensation is broken, and symptoms of cardiac failure appear. The duration of compensatory hypertrophy in individual cases varies considerably, and chiefly depends upon the extent to which the myocardium has been involved in the original endocarditis. With efficient muscle a serious valve lesion may be longer compensated than a slight one when the musculature is disabled. Age is also an important factor, young muscle possessing greater power of hypertrophy than that of the elderly.

Illustrate these Effects in detail in Aortic Stenosis.

The first effect of such an obstruction is felt on the chamber behind the valve, the left ventricle, which dilates slightly. Soon the reserve power comes into play, the ventricle contracts more forcibly to overcome the obstruction, and its walls begin to hypertrophy. When the stenosis is slight, a moderate hypertrophy may meet all demands, and no further trouble may ensue for a long time. Even with considerable and progressive stenosis, continued hypertrophy with stronger contractions may ward off serious symptoms. This is the *stage of compensation.* Finally, however, the ventricular reserve is exhausted and dilatation occurs, with consequent leakage at the mitral valve (relative insufficiency). There is regurgitation of blood into the left auricle, followed in course by pulmonary engorgement. To force the blood through the engorged lungs, the right ventricle is called upon for stronger contractions, and must hypertrophy. Its powers in that direction are more limited than those of the left, and dilatation occurs sooner, with a consequent relative tricuspid insufficiency and engorgement of the systemic veins. This is the stage of *failure of compensation*, and the aortic symptoms may be masked by mitral symptoms.

Describe in detail the Effects of Failure of Compensation.

Consequent on the establishment of relative tricuspid insufficiency, blood regurgitates into right auricle which becomes engorged, and obstructs the systemic venous return. There is thus passive congestion of internal organs, subcutaneous circulation, and later of serous cavities. The chief symptoms are cough, often with hæmoptysis, dyspnœa or orthopnœa, cyanosis, palpitation, insomnia, and cardiac irregularity. The physical signs form two classes :—

I. *Those directly of tricuspid regurgitation—*

(1) Tricuspid systolic murmur, (2) weakness of second pulmonic sound, (3) marked irregularity of pulse. The

heart is in the state of "delirium cordis" due to auricular fibrillation, (4) venous pulse in the neck, perhaps also in liver.

II. *Those of passive congestion*—

(1) Œdema first in feet and ankles (effect of gravity), later general.

(2) In severe cases ascites, hydrothorax, hydropericardium.

(3) Congestion of internal organs—

(*a*) Liver enlargement, tenderness, icteric tinge of skin.

(*b*) Kidneys:—scanty, high-coloured urine, abundant urates, varying albuminuria, few casts, and little blood, urea normal.

(*c*) Stomach and bowels:—catarrh, dyspepsia, enteritis, at times hæmatemesis and melæna.

(*d*) Lungs:—œdema and congestion, as shown by fine moist râles and basal dulness.

(*e*) Spleen:—enlargement and tenderness.

What are the Causes of Hypertrophy of the Heart?

Valvular lesions are the chief cause, but adherent pericardium, lung diseases, increased peripheral resistance (arterio-sclerosis, atheroma), aortic aneurysms, long continued exertion of a healthy heart (soldiers, athletes, etc.) and nervous causes may all give rise to it.

What are its Physical Signs?

In the *left ventricle* it may induce præcordial bulging, and the apex beat is altered in character and position. It is diffuse, slow and heaving, and may be felt in 6th, 7th, or even 8th space outside the nipple. Cardiac dulness is increased downwards, and to the left. The apical first sound is prolonged and of low pitch.

In the *right ventricle* it causes epigastric pulsation, and

moderate increase transversely to the right of the cardiac dulness. The second pulmonic sound is accentuated, and may be reduplicated.

Mention the Causes of Dilatation of the Heart.

It may be primary or secondary. The *primary* form is due to causes directly affecting the myocardium, bacterial or chemical (alcohol and tobacco), and also to emotion, shock, or over-exertion.

The *secondary* form has the same etiology as hypertrophy.

What are its Physical Signs?

Dilatation of the *left ventricle* causes displacement downwards and to the left of the apex beat, which is feeble and diffuse. Cardiac dulness is increased downwards and to the left. First apical sound is short, sharp, and loud, and there is altered rhythm (tic-tac). A systolic murmur of varying degree is usually present. The heart is rapid and irregular. A dilated *right ventricle* is evidenced by diffuse præcordial pulsation, feeble impulse (apex beat often absent), great increase of dulness to the right, a short and sharp first tricuspid sound (often a systolic murmur), and a weak second pulmonic. Venous pulsation is present in the neck. The heart is rapid and irregular.

INDIVIDUAL VALVE LESIONS.

How may the Valves of the Heart be affected?

The valvular orifice may be narrowed, impeding the flow of blood—obstruction or stenosis.

The valve may be incompetent—regurgitation or insufficiency. It may be obstructed and incompetent—a combined lesion.

How are these Conditions diagnosed?

By the position, rhythm, and conduction of their accompanying murmurs, and by the resultant general symptoms.

How are Murmurs propagated, and where are they best heard?

They are conducted in the direction of the blood stream, and are best heard over areas situated a short distance from their site of production. Aortic murmurs are best heard over the aortic area (second right costal cartilage). The aortic systolic murmur is conducted upwards into the vessels of the neck, while the regurgitant (diastolic) is carried best down the sternum. *Pulmonary* murmurs are best heard in the second left space, and are conducted obliquely upwards for about two inches. *Mitral* murmurs are loudest at the apex (mitral area). The systolic (regurgitant) are conducted outwards into the axilla and to the angle of the left scapula. The presystolic, and diastolic (obstructive) have little or no conduction. *Tricuspid* murmurs are best heard at the lower sternum.

With regard to a Murmur, what Points should be noted?

(1) Its point of maximum intensity; (2) Its rhythm; (3) Its character; (4) Its direction of propagation; (5) Any modification of the cardiac sounds which it induces; (6) Any accompaniments.

What are the Characteristic Features in Mitral Regurgitation?

A blowing systolic murmur heard best in the mitral area, conducted outwards to the axilla, and to left back. The first sound is modified, or may be replaced by the murmur, while the second pulmonic sound is accentuated. Systolic apical thrill is often felt. There is a varying degree of ventricular hypertrophy. With failure of compensation, the symptoms and signs already described appear.

Point out the Characteristic Features in Mitral Stenosis.

A rough, vibratory, presystolic murmur is heard at the mitral area, running up to a sharp first sound, in which it

abruptly ends. It has little propagation, and is often accompanied by a palpable presystolic thrill. When stenosis is considerable the murmur may be mid-diastolic, or be present throughout diastole. The second pulmonic sound is accentuated. There is hypertrophy chiefly of right ventricle, and evidences of failure of compensation appear later.

What Features characterise Aortic Regurgitation and Stenosis?

These are more often combined in the same case. Stenosis is indicated by a loud, rough, systolic murmur in the aortic area, conducted upwards, regurgitation by a soft blowing diastolic murmur at aortic area conducted downwards. In both, the left ventricle is hypertrophied, but much more so in regurgitation, with tendency to syncope (anæmia of circulation), and often anginoid pain. If compensation fails, mitral signs appear.

Indicate the Signs of Tricuspid Lesions.

Tricuspid *regurgitation* is indicated by a systolic murmur heard best at the lower sternum with slight propagation to each side, *stenosis* by a rough presystolic murmur in the same area. Those are congenital lesions, though with failure of compensation, or with lung diseases, the former may occur secondarily.

What are the Evidences of Pulmonic Lesion?

Both stenosis and regurgitation are usually congenital, the former is more frequent. It is indicated by a loud systolic murmur in the pulmonic area with propagation upwards. The right heart is enlarged and cyanosis is marked. Regurgitation is a rare lesion, and is indicated by a soft, blowing diastolic murmur at the pulmonic area.

Compare the Symptoms in Disease of Aortic and Mitral Valves.

Aortic lesions tend to produce arterial emptiness, while mitral cause venous congestion. The symptoms thus differ, but in the later stages all organic valve lesions tend to produce both these effects. The comparison is best shown in tabular form :—

Aortic Disease.	*Mitral Disease.*
1. Pallor.	1. Cyanosis.
2. Visible pulse in peripheral arteries.	2. Visible venous pulse in later stages.
3. Præcordial pain, often like that of angina pectoris.	3. Præcordial distress frequent, with palpitation; acute pain uncommon.
4. Dyspnœa frequent.	4. Dyspnœa frequent.
5. Gastric symptoms rare, or a late manifestation.	5. Gastric symptoms frequent.
6. Œdema late or slight.	6. Œdema frequent and marked.
7. Embolism rarely occurs.	7. Emboli common.
8. Cerebral symptoms and syncopal attacks common.	8. Pulmonary symptoms common.
9. Hypertrophy of left ventricle often considerable.	9. Chiefly hypertrophy of right ventricle.

What are the Common Sites of Embolism, and what are its Effects?

In the *spleen*, causing infarction and enlargement, frequently with pain and tenderness due to localised peritonitis.

In the *kidney*, when pain and hæmaturia result.

In the *brain*, the left middle cerebral artery is most often affected in its lenticulo-striate branch, with resultant hemiplegia and aphasia.

In limb arteries, causing pain, and disappearance of pulse on remote side. Rarely in the skin and mucous membranes, causing purpuric spots. In ulcerative endocarditis the emboli are septic, and give rise to metastatic abscesses.

How does the Pulse vary in Cardiac Lesions?

The variations are best shown by sphygmographic tracings, but in the main they can be appreciated by the finger.

In aortic stenosis as the blood is obstructed at its entrance into the aorta, the pulse is small, its time of ascent is slow, and often interrupted (anacrotic). The tension is usually high. The lesion is commonly combined with a varying degree of regurgitation, which modifies the nature of the pulse. In *aortic regurgitation*, as the left ventricle is filled more quickly by two streams, the normal and the regurgitant, the pulse beats more rapidly. It rises abruptly, and, owing to the regurgitation, it falls quickly. This gives it a peculiarly characteristic quality like a "water-hammer." It is otherwise termed the collapsing pulse, the pulse of unfilled arteries, or Corrigan's pulse (after the describer). Capillary pulsation is often well marked. In *mitral disease* the pulse is small, of low tension, and often irregular, especially in the later stages.

In the Prognosis of Heart Lesions, what Points should be taken into account?

Too much stress should not be placed on the mere presence of murmur, but it should be considered along with the state of cardiac nutrition, as indicated by the character of the sounds, and the degree of compensation, as well as the age and habits of the patient. The outlook varies somewhat with the heart lesion.

Indicate the Difference in Prognosis in Various Heart Lesions.

In *aortic stenosis* the outlook is more favourable when the lesion begins in the elderly, as its slow progress allows of sufficient compensation. The vessels are, however, often atheromatous and fragile, leading to rupture.

Aortic regurgitation is a very dangerous lesion, often fatal from sudden syncope, or from angina pectoris. The outlook largely depends on the degree of possible hypertrophy, and hence upon the age of the patient.

Either lesion may of course progress to implication of right heart, and failure of compensation. When both coexist, the stenosis is in a measure protective as it limits the regurgitation.

Mitral stenosis offers a graver outlook than regurgitation, as failure of compensation tends to arise earlier, and it is usually progressive. The prognosis in *regurgitation* largely depends on the cause, and the state of the heart muscle. Both may be compatible with long life.

In all lesions of the left heart the danger of embolism must never be forgotten.

What is the Treatment of Valvular Diseases of the Heart?

No routine treatment can be laid down. Our aim is to establish complete compensation and to maintain it. This is done by two lines of treatment, general and medicinal. The former comprises the regulation of skin, kidneys, and bowel, the avoidance of large bulky meals especially before bedtime, the regulation of work and exercise with avoidance of strain and extremes of temperature, and the administration of simple tonics. Carefully graduated exercise is often useful, as it imitates nature's method of producing hypertrophy of the chambers behind the lesion. Absolute rest on fluid diet should be enforced when compensation has failed.

Drug treatment includes general tonics, and those with special cardiac action. No drugs may be required when compensation is good, regulation as above may suffice, but arsenic, strychnine, or iron may be useful. Cardiac tonics are called for where compensation has failed, and of these digitalis is pre-eminent in slowing and strengthening the heart, and prolonging its diastole. It has also a diuretic action. Its effects are best seen in mitral disease, and it is particularly indicated in cases with auricular fibrillation. It has disadvantages in that, in toxic doses, it increases peripheral resistance, and thus causes greater strain, also, as it prolongs diastole, it is unsuitable in aortic regurgitation unless there are mitral symptoms. Its use should be stopped if the urine decreases, or if the pulse becomes more irregular, and to avoid its cumulative action, it should not be given for longer than three weeks without intermission. It is often combined advantageously with strychnine. When digitalis disagrees, or is intermitted, strophanthus may be employed. Strychnine is useful in regular doses, or as an immediate stimulant in sudden failure of compensation. Alcohol and ammonia (Spt. amm. arom.) are similarly useful, but perhaps the strongest emergency stimulant is camphor in oil (gr. 3 in ℥.—17) given intramuscularly. Cardiac pain is best relieved by morphia, or in degenerative cases by a course of potassium iodide. For the pain and distress of aortic cases amyl nitrite is often beneficial. With the œdema and venous congestion of the later stages diuretics and saline laxatives should be given. Tapping of chest or abdomen may be necessary, or drainage of the subcutaneous tissues by incisions or Southey's tubes. Venesection may be called for to ease the right heart, or leeches over the liver.

DISEASES OF THE MYOCARDIUM.

ACUTE MYOCARDITIS.

What is understood by this Term?

An acute interstitial inflammation usually associated with degeneration of heart muscle, and probably of toxic origin. It is usually associated with valve diseases due to rheumatism, or occurs in connection with specific fevers, especially diphtheria and influenza, in which there is liability to rapid fatty degeneration, and consequent acute dilatation. The symptoms are enlarged cardiac dulness, rapid and irregular action, a weak first sound, and tic-tac rhythm. There may be sudden death, but commonly the attack is mild, and passes off with convalescence. In septic diseases, *e.g.* malignant endocarditis, a suppurative form may occur with small abscesses, the result of septic emboli.

CHRONIC MYOCARDITIS.

(Hypertrophy and dilatation of the heart have already been dealt with under chronic endocarditis.)

Mention any further Myocardial Changes that may be found.

The chief are *fibroid* and *fatty* degenerations (one or both together), and that due to syphilitic disease.

What are the Causes of Fibroid and Fatty Changes?

They may follow the acute form, or result from coronary sclerosis, the effect of poisons, alcoholism, gout, anæmia, or arterio-sclerosis. Disease of the coronary arteries, which is the most important, may be primary, an arteritis, acute or chronic, or endarteritis obliterans (syphilis), from either of which coronary thrombosis may result. This may lead to an anæmic infarct, with aneurysm or rupture of the heart. Embolism of a coronary artery causes sudden death. Coronary disease may be secondary to chronic aortitis, or atheroma of aorta.

Give the Symptoms and Signs of Fibroid Degeneration.

Often there are none owing to compensatory hypertrophy, but usually there are dyspnœa, palpitation, præcordial pain (with anginal spasms), œdema of ankles, and cyanosis, all gradual in onset. On examination the heart is enlarged, and its first sound prolonged and muffled. Death may be sudden, or follow gradual cardiac failure.

What are the Symptoms and Signs of Fatty Heart?

These are often vague, but are those of general cardiac weakness with syncopal or epileptiform attacks. Heart failure may be sudden and unexpected, or gradual. The heart may be moderately enlarged, apex beat feeble or absent, and the sounds short and clear. The pulse is rapid and irregular, as a rule, though it may be strikingly slow.

How does Syphilis affect the Heart?

It may cause gummata, or a diffuse fibrosis of the myocardium, and produces symptoms and signs very similar to those of fibroid change.

What is meant by Heart-block?

It is a condition which is usually due to lesions of the auriculo-ventricular bundle (His), though it may follow vagal stimulation, or result from the action of digitalis. Lesions of the a.-v. bundle interfere in varying degree with the conduction of impulses from auricle to ventricle, and thus set up partial or complete block. In *partial block*, conduction may be only delayed, with increase of the interval between auricular and ventricular contractions, or auricle and ventricle may beat at different rates owing to failure of conduction of some impulses. The ratio of block is usually described in figures, thus "2 to 1 block" where every second impulse fails to pass the barrier, "3 to 1" with failure of every third impulse. The ratio of block may vary from time to time, giving a slow *and* irregular ventricular action. In *complete*

heart-block conduction is quite cut off, and there are two separate rhythms, the auricle beating regularly at its own rate about 72 per minute, while the ventricle beats independently at its own—idioventricular—rate about 30.

It is a condition which is not necessarily incompatible with a fair measure of health, but cases of complete block are frequently associated with a group of symptoms known as the Stokes-Adams Syndrome.

Describe the Stokes-Adams Syndrome.

It is a combination of symptoms, which include two which are characteristically associated, viz.: paroxysmal bradycardia, and syncopal or epileptiform attacks. It is commonest in men over fifty, and is usually caused by fibroid degeneration, consequent upon lesions of the coronary arteries, or by myocardial gummata affecting the a.-v. bundle.

Indicate the Symptoms and Signs of the Condition.

Often those of valvular disease, especially aortic, or merely those of dilatation and heart failure. There is usually arterio-sclerosis. The patient is commonly pale, with localised areas of cyanosis. Dyspnœa is common and is often paroxysmal. The pulse is persistently slow, and very markedly so during the paroxysms. These vary in frequency, character, and severity, and take the form of nervous disturbances such as giddiness, transient loss of memory, and syncopal or epileptiform attacks. Cheyne-Stokes breathing may be observed especially during the paroxysms. These seizures are due to cerebral anæmia consequent upon asystole.

Such cases end fatally, and often suddenly, though life may be prolonged for several years.

What is the Treatment of Myocardial Diseases?

The patient should live a quiet and peaceful life with carefully regulated diet and exercise. There should be moderation both in eating and drinking. In arterio-sclerotic

or specific cases potassium iodide is the drug indicated, and if arterial spasm be present, nitrites may be added. Cardiac stimulants, as strychnine and alcohol may be required for the syncopal attacks, but digitalis and strophanthus must be used with care, if at all. These have little or no action on degenerated muscle, and may be dangerous by increasing peripheral tension. When the breathing is laboured, or Cheyne-Stokes, the use of oxygen, or strychnine hypodermically may bolster up the failing muscle for a time.

ANGINA PECTORIS.

("Breast Pang" "Stenocardia.")

Indicate the Characteristic Features of this Condition.

Sudden attacks of severe pain in the præcordium, or radiating therefrom, with an accompanying sense of impending death.

What is its Etiology.

It occurs most often in males in late middle life. Many organic lesions predispose to it, but in general these all interfere with myocardial nutrition, *e.g.* coronary sclerosis, fatty and fibroid degenerations, aortic valvular disease, arterio-sclerosis or atheroma, and affections of the cardiac plexus or its ganglia. Thus it is associated with gout, syphilis, diabetes, etc. A neurotic type occurs associated with hysteria, and the abuse of alcohol or tobacco, and a reflex type from irritation in the stomach or elsewhere. The *exciting* causes are exertion, emotion, or a distended stomach.

Indicate its Morbid Anatomy.

Occasionally no lesions of the heart or vessels have been discovered, but usually one or more of the lesions enumerated under causation.

What Theories have been advanced to explain its Occurrence?

Spasm of the coronary arteries—*intermittent claudication*—is that most favoured. It is based upon the effects of spasm or partial obstruction of arteries elsewhere. The circulation becomes insufficient to allow of exertion, and muscular exhaustion occurs with cramping pain. High arterial tension probably participates in most cases, as it is often permanently present, and is usually so during an attack. However, in some cases the tension is unaltered or permanently low, and in these we may suppose that an exertion, which would be well borne by a healthy heart readily causes exhaustion to one with myocardial damage. Some explain the condition as a neuralgia of the cardiac nerves, others hold that it does not originate in the heart, but in the first part of the aorta (Allbutt). Mackenzie regards it as a protective reflex, accepting the obstructive theory, and compares the pain to that set up by the obstructed evacuation of other hollow viscera.

Describe the Features of an Anginal Attack.

The onset is sudden with acute pain over the heart, and a sense of constriction across the chest. Should the patient be walking, he stops rigid; if sitting, he leans forward and fixes the shoulder girdle by grasping some convenient support. The pain radiates from the lower sternum or aortic area down the left arm, it may be to the fingers, or up the left neck to the head, rarely down the right arm. He feels as if his chest were being gripped in a vice, and has a sensation of impending death. This is reflected by the anxious, terrified look, the pale face, and the cold sweating. Though there is a feeling of suffocation, and respirations are shallow and irregular, there is rarely dyspnœa, and no cyanosis. The heart varies, but may be rapid and irregular, and often the pulse shows increased tension. The attacks vary in length and severity, the first may be mild, lasting

only a few seconds, but usually they increase in severity and frequency, and are more easily excited. At times all the symptoms may be present without pain (*angina sine dolore*), or pain may be slight.

What is the Prognosis?

In the severe forms it is grave, and sudden death may occur. It is more hopeful in mild cases, and in the neurotic forms.

Differentiate between the Real and Pseudo-angina.

The latter is a purely neuralgic affection, and occurs at any age from six years, chiefly in women. The attack is spontaneous, without exertion or apparent exciting cause, often periodical and nocturnal. The pain is less severe, and is more a feeling of distension. The attack lasts longer, and is associated with restlessness, agitation, and other nervous symptoms. It is never fatal.

Give the Treatment of Angina Pectoris.

During an attack give amyl nitrite by inhalation, chloroform, or morphia. *In the intervals*, the habits should be regulated, and indiscretions in exercise, emotion, and diet avoided. Prohibit "flatulent" foods or bulky meals. Treat the cause, usually arterio-sclerosis or syphilitic endarteritis, by potassium iodide in increasing doses. With persistent high blood-pressure, give nitro-glycerin, or sodium nitrite. In *pseudo angina* treat the nervous causes.

What is meant by Angina Abdominis?

A condition in which sudden attacks of intense epigastric or umbilical pain occur from causes similar to angina pectoris. It is regarded by some as a referred pain of cardiac origin; others ascribe it to disease of the gastro-intestinal blood-vessels. *Treatment* as for angina pectoris.

DISORDERS OF THE CARDIAC RHYTHM.

Describe the Normal Order of Events in the Cardiac Rhythm.

The contraction of the heart originates at the sino-auricular node (at the mouth of the superior vena cava), and is conducted to the auricle, from which it passes through the a.-v. bundle to the ventricle.

How do Disorders arise?

Under abnormal stimuli, contraction may originate in at least three other points, with differing types of irregularity. These points are the a.-v. node (in the wall of the right auricle near the coronary sinus), the a.-v. bundle (His), and the tissues of the auricle or ventricle.

What Types of Irregularity are thus produced?

Indicate their Features.

(1) *Sinus arrhythmia.* Here contraction begins as usual at the sino-auricular node, but owing to depressed or increased excitability of tissue, the beats are irregular in rhythm, though equal in strength. It is a rhythmic irregularity, and may be found in young healthy hearts, or during convalescence from fevers. It does not call for special treatment.

(2) *Premature contractions* or *extra-systoles*, of auricles or ventricles, or both, independently of the sinus rhythm. Many patients show occasional and isolated missed beats. These may be due to incomplete heart block, or sudden vagal stimulation, but the most common cause is premature contraction. Such a contraction is followed by an abnormally long pause, diastole being prolonged till the succeeding sinus stimulus is due. The small beat may not be palpable at the wrist, but it is audible over the heart (differs from heart block). Extra systoles may be so frequent

as to replace each alternate normal beat. The pulse then assumes an intermittent or bigeminal character. Extra systole is commonly due to functional disturbances, *e.g.* dyspepsia, abuse of tobacco or tea, etc., ceasing when the cause is removed, but it occurs also in myocardial disease, associated with other evidences of cardiac trouble. Without other sign of cardiac disease, they are not of grave import.

(3) *Auricular flutter.* Here the normal auricular beats are replaced by a series of rapid rhythmical contractions originating from an abnormal source in the auricular wall. It is usually associated with chronic myocardial or valvular disease. Often some degree of heart-block coexists, causing cardiac irregularity, but if not, the pulse is persistently rapid and regular, and is unaffected by posture. Treatment is by rest and digitalis. It may merge into :—

(4) *Auricular fibrillation* which is the cause of the continued irregularity in force and rhythm found in cardiac failure from valvular or myocardial disease. There is no co-ordinate contraction of the muscular wall of the auricle, the normal systole being replaced by a hopeless collection of disconnected twitchings. Certain of these fibrillations get through in haphazard form to the ventricles, and give rise to rapidity and irregularity of its beat. Exercise accents this irregularity while it decreases or abolishes extra-systole and sinus arrhythmia. Treatment by rest and digitalis may produce very striking improvement.

(5) *Irregularities due to failure of conductivity of the a.-v. bundle.* Systole is omitted as the stimulus fails to reach the ventricle—the condition of heart-block (already described).

(6) *Irregularities due to depression of contractility,* of the ventricle causing a regular irregularity, the

alternate beats varying only in strength—*pulsus alterans.* (There is no prolongation of diastole as follows extra systole). The condition indicates imminent cardiac failure, and if it persists, is of grave import.

PALPITATION.

What is meant by this Term?

An unduly forcible, and sometimes irregular cardiac action which is perceived by the patient.

Point out its Causes.

It is usually functional or neurotic in origin, but may occur in organic cardiac disease. It is more common in women, associated with anæmias, gastro-intestinal disorders, and neurasthenia. In men it may result from excessive use of tobacco or alcohol. The attacks may be paroxysmal, and come on even during rest. Beyond the forcible, rapid, and often irregular cardiac action, the organ is otherwise normal.

What are the Prognosis and Treatment?

The former is favourable except when local disease is present. The treatment consists in removal of the cause, allaying anxiety, and the regulation of diet and habits.

TACHYCARDIA.

Define this Term.

. An increased rapidity of cardiac action, usually without subjective sensations. It may be paroxysmal in character.

What are its Causes?

General causes as for palpitation, but also found characteristically in exophthalmic goitre, or diseases causing pressure on vagus nucleus, and in vagal paralysis. It may be a climacteric manifestation.

Describe what is known as Paroxysmal Tachycardia.

It is a definite cardiac affection in which the attacks come on suddenly and end as suddenly. They are often accompanied by restlessness, discomfort in the chest, and exhaustion, vary much in duration, and are uninfluenced by posture. The pulse rate may rise to 200 per minute or even higher. The condition is due to a sudden abnormal rhythmic stimulation of a focus in the auricle, the a.-v. bundle, or node. When the stimulus originates in the a.-v. node, simultaneous contraction of auricles and ventricles occurs, with the establishment of *nodal rhythm.* Some cases progress to a fatal issue from dilatation, and cardiac failure. It is commonest in young adults, following rheumatism, and exertion, emotion or flatulence may excite an attack.

How is it treated ?

An attack can only be treated by rest, it is rarely influenced by drugs. Regulation of life, habits, and functions may prevent attacks.

BRADYCARDIA.

What is meant by this Term ?

Infrequency of the heart beat.

How is it caused ?

It may arise from vagal stimulation (many causes), or from fatigue, toxæmias, poisons (digitalis, alcohol, etc.) acting on the myocardium. It is often associated with jaundice, or in convalescence from acute fevers. A slow pulse may be normal and hereditary. The treatment is that of the cause.

III. THE ARTERIES.

ARTERIO-SCLEROSIS.

(Atheroma).

Define this Term.

A condition of increased rigidity in the arteries, consequent upon a thickening of their walls, especially of the intima, but extending to the other coats. The term atheroma is more correctly applied where the condition is patchy.

What is its Etiology?

There is a hereditary tendency to early arterio-sclerosis in some families, but the condition is one of the accompaniments physiologically of advancing years. Other causes are those which are associated with high arterial tension, as Bright's disease, diabetes, gout, lead poisoning, overeating or over-strain, also alcohol and syphilis.

Describe its Morbid Anatomy.

The condition may be *patchy* (nodose), or *diffuse.* The former affects chiefly the larger vessels, especially the aorta. The patches vary in size, are flat, raised, greyish-white or yellowish, and are scattered over the intimal surface. They may soften, forming atheromatous abscesses, which, breaking through the inner coat, cause ulcers, or they may become calcareous, and the hard plates may crack, lacerating the intima. The muscular coat atrophies, and the vessel becomes hard, thick and tortuous. In small vessels the lumen may be obliterated, especially in the diffuse form, where the thickening is general, and may affect also the capillaries and the veins.

What are the Symptoms and Signs?

Symptoms may be absent until the condition is well established, when those of cardiac hypertrophy, or dilatation with heart failure may appear. Where cerebral arteries are affected, there may be headache, giddiness, noises in the ears,

etc. The *signs* are high tension pulse, increased blood pressure, hypertrophy of left ventricle, accentuated second aortic sound, and thickening of peripheral arteries, which are rigid, visible and tortuous. Pulsation is visible in these vessels, and often irregular, or annular thickening can be felt in the radials (resembling to the touch a chicken's trachea).

Mention the Results that may follow such Changes.

These may be widespread and varied, depending on site; *cerebral*,—rupture (hæmorrhage), aneurysm, thrombosis; *coronary*,—angina pectoris, thrombosis; *aorta*,—dilatation of arch, aneurysm, implication of valve cusps, with stenosis, regurgitation or both; *limbs*,—thrombosis, gangrene (senile).

Indicate the Treatment.

Regulation of life, diet, and exercise, with avoidance of all excesses. High blood pressure can be effectively reduced by a periodic mercurial purge; nitrites or nitroglycerin should only be used in marked cases. A prolonged course of potassium iodide may be useful, especially in syphilitic cases.

ANEURYSMS.

Define the Term.

It is a localised and persistent dilatation of an artery.

How are Aneurysms classified?

1. *Fusiform* (spindle-shaped).—The vessel is affected in its whole circumference, and all its coats are involved.

2. *Sacculated.*—Only a limited part of the artery is dilated, the sac is formed by the outer coat, the inner and middle having ruptured.

3. *Dissecting.*—The inner coats having ruptured, the blood current forces its way between them, and rejoins the main current lower down.

What is its Etiology ?

The essential factors are *increased strain* on *damaged vessels.* Increased vascular strain is common in laborious occupations, and the vessels are damaged mainly by arteriosclerosis. Very rarely does a single overstrain, however violent, rupture the coats of a healthy artery. Aneurysm is most common in males about middle life.

Describe its Morbid Anatomy.

The inner coat is thickened by atheroma in the fusiform type, and in the sacculated it passes into the sac, where it disappears. The muscular coat atrophies, and may vanish. The outer coat is much fibrosed, and becomes adherent to surrounding structures.

What is the Course of a Sacculated Aneurysm ?

It tends to increase in size, and progress to rupture. Spontaneous cure is known in aneurysms with small orifices and sluggish circulation in the sac, by the formation of thrombi, and later fibrous tissue, or in larger sized by compression of the artery by the sac, cutting off its blood-supply.

Mention the Cardinal Signs common to Aneurysms.

Tumour, expansile pulsation, murmur, and pressure effects (including pain). These vary, depending on the size, and site of the aneurysm, and the nature of the surrounding structures. The tumour may not be recognisable. The pressure signs may be few, or form the only basis for a diagnosis.

What is the most frequent Site of Aneurysm and why ?

The aorta in its arch. The reasons for this are various, viz. : (1) Its great curving. (2) Its lack of support. (3) Its exposure to pressure during ventricular systole. (4) Its greater exposure to variations of cardiac pressure.

(5) Its walls are weakened by the number of branches given off within a small area. (6) It is the common site of aortitis and atheroma.

ANEURYSM OF THE THORACIC AORTA.

(Thoracic Aneurysm.)

What is its Etiology?

Its causes are those of aneurysm in general. Atheroma of aorta, and syphilis are the most constant. Traumatism may be a determining cause.

Describe its Symptoms.

These depend on the size and shape of aneurysm, and also on the part of the arch which is affected. The general symptoms have been already stated. In addition there are circulatory symptoms, and those due to pressure. Of the former, palpitation and anginoid pain are the chief, with symptoms of aortic regurgitation, and often difference in radial pulses (may be a pressure effect). There may be evidence of pressure on organs, nerves, vessels or bones:—

(1) *Œsophagus*:—difficulty in swallowing—*dysphagia.*

(2) *Respiratory tract*:—weakened breath sounds, dyspnœa, cough, and often hæmoptysis.

(3) *Nerves*:—with slight pressure *irritation*, with severe, *paralysis.* Those most often affected are the left recurrent laryngeal, and sympathetic.

	Irritation.	Paralysis.
Left recurrent laryngeal.	Spasm, causing stridor, altered voice, and cough (leopard growl and gander cough).	Aphonia. Paralysis of left vocal cord.
Sympathetic.	Pupils dilated—rapid heart—unilateral pallor (vaso-constriction) of face and head.	Pupils contracted, unilateral flushing and sweating.

The heart may be slowed or irregular owing to pressure on *vagus*, and hiccough or diaphragmatic paralysis may result from pressure on *phrenic* nerve. These are less common.

(4) *Veins.*—Œdema of chest, face, and arm, with venous dilatation on affected side.

(5) *Bones.*—Usually, with intense "boring" pain at seat of pressure.

(6) *Thoracic duct.*—Rarely, with starvation, emaciation, fatty stools, chylous ascites, etc.

What are the Physical Signs?

These vary with the part of the arch affected :—

(1) *Ascending portion.* — Expansile pulsation with or without swelling in second and third right spaces, systolic thrill and diastolic shock, dulness to right of sternum, rough systolic bruit (often also diastolic) with accentuated second sound, apex displaced downwards and outwards. Pressure on superior vena cava, right bronchus, ribs, and rarely on right recurrent laryngeal nerve. Right radial pulse weakened.

(2) *Transverse portion.*—Pulsation and systolic thrill in suprasternal notch, dulness and systolic murmur over manubrium, weakening of either radial pulse. Pressure on the œsophagus, trachea or left bronchus, left recurrent laryngeal and sympathetic nerves, left innominate vein.

(3) *Descending portion.*—Few or no signs, possibly dulness and tumour to left of spine. Pressure on spinal column or even the cord, left bronchus or lung, left recurrent laryngeal and sympathetic nerves, œsophagus and thoracic duct.

In all forms X-ray examination is important, revealing an abnormal pulsating shadow. *Tracheal tugging* (with

systole) may be felt wherever there is downward pressure on trachea or left bronchus. *Hæmorrhage* may occur at any time, small in quantity from granulations in trachea, or "weeping" from exposed sac, or profuse from rupture.

Indicate the Prognosis.

The outlook is very grave in every case, though occasionally, under treatment, growth may be arrested. Death occurs from asthenia, heart failure, asphyxia or rupture (rarely external).

What is the Treatment ?

The aims in treatment are—(1) to quieten the circulation, (2) to lower blood pressure, (3) to favour coagulation, (4) to lessen the enlargement, and (5) to avoid rupture of the sac. It is thus general, medicinal, and local. Absolute rest on a much restricted diet is the first essential. Tufnell's dietary is only suitable for early cases, and proves very irksome. Limitation of fluids is of the highest importance. Potassium iodide is almost always given, but is most efficacious in syphilitic cases, and in increasing doses. It often soothes the pain, but for this purpose morphia must be employed sooner or later. Locally, electricity to the sac, and the introduction of needles, fine wire, or horse hair have all been tried, at times with success in producing coagulation. Gelatine injections have been recommended with the same object. At first small hypodermic injections were employed, but larger intramuscular doses have been tried (6 to 8 ounces of a 2 per cent. solution in normal saline, repeated weekly). These are painful, and may be dangerous to life; they may more safely be given rectally.

Treatment may be called for to relieve urgent symptoms at any time.

ANEURYSM OF THE ABDOMINAL AORTA.

Describe the Special Features of this Condition.

It occurs most commonly near the cœliac axis, and may grow in any direction. The symptoms and signs are often indefinite. The essential point in diagnosis is the presence of a definite tumour with expansile pulsation. This often cannot be clearly demonstrated. The throbbing aorta of neurasthenics, and tumours over the aorta with communicated impulse therefrom, offer the greatest difficulties in differentiation. The absence of expansile pulsation, and examination in the knee-elbow position are very helpful in this regard. Pain may be present, neuralgic referred to the flanks, or typically "boring" behind.

Give the Prognosis.

Always grave. Death may result from compression paraplegia, embolism of the superior mesenteric artery, obliteration of the lumen by clots, or rupture (usually into the duodenum).

What is the Treatment?

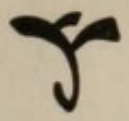
Generally as for thoracic variety. When the seat of aneurysm is low down compression under chloroform has been tried, but it must be sustained for many hours, and is not without risk.

CATECHISM SERIES

MEDICINE

SECOND EDITION

PART V



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(1) Facial Paralysis.
(2) Cerebral Tumours.
(3) " Hæmorrhage
(4) " abscess
(5) G.P.I.
(6) Cerebral Embolism
(7) " Haem.
8) Hemiplegia
9) Bulbar paral.
10) Spinal hemiplegia
11) Transverse myelitis
12) Loc. Muscular
13) Peripheral neuritis
14) Progressive bulbar
15) Acute Ant. poliomyelitis
16) Amyotrophic Lat. Sc.
17) Pseudohypertrophic paral.
18) Epilepsy
19) Chorea.
20) Paral Agitans
21) Rapeandrum
22) Hemiplegia
23) Sciatica.

CONTENTS

INTRODUCTORY NOTE.

In a series of this kind it must be presumed that the student embarks on the study of diseases of the nervous system with a general working knowledge of the anatomy and physiology of the brain and cord. He or she should be familiar with the primary elements which form the structure of the nervous system, the various tracts or columns which form the white matter of the cord, the motor and sensory paths and their relations with the great basal ganglia, and should have a general idea of the cerebral functions, with their localisation. Without such knowledge to work upon, lesions of the nervous system and their effects cannot properly be understood. Many of these necessary points will incidentally be referred to in the systematic review of the various diseases, but to consider them in detail is outwith the scope of the present volume.

MEDICINE

PART V.

DISEASES OF THE NERVOUS SYSTEM.

I. GENERAL SYMPTOMATOLOGY.

MOTOR DISTURBANCE.

What may this include?

(1) Diminution of movement of muscles—*paresis*—or complete loss—*paralysis*.

(2) Increase of muscular power—*spasm* and rigidity.

(3) Inco-ordination of movement—*ataxia*.

(4) Alterations in gait.

Name the Varieties of Paralysis which occur.

Its onset may be sudden or gradual, depending on the cause. Its distribution varies with the site of lesion, *e.g.* when due to a cerebral lesion it is unilateral (*hemiplegia*), when to a cord lesion bilateral (*paraplegia*). *Monoplegia* is the term applied when one limb, or one group of muscles only, is involved. Where paralysis is due to injury or disease of peripheral nerves, the loss of power is limited to the muscles supplied. Paralysis may be *spastic* or *flaccid*. In the former (an upper motor neurone lesion), there is rigidity of the affected limbs with exaggerated reflexes, and later contractures; in the latter (a lower motor neurone lesion), the muscles are flaccid with marked wasting, R.D. is present, and deep reflexes are lost. The former indicates a lesion in the lateral columns of the cord, the latter in the anterior cornua.

Describe the Varieties of Muscular Spasm which may be found.

Spasmodic contractions of muscle may be of two kinds (1) *tonic*, where they are continuous; (2) *clonic*, intermittent, with rapid and irregular movements. Tetanus and tetany are examples of exaggerated and spasmodic movements. *Tremor* (or, if marked, shaking), may be a symptom of disease. It may be volitional, as in disseminated sclerosis, or permanent, and occurring even during repose, as in paralysis agitans. It is also met with in senile decay, toxic and functional conditions. *Choreic* movements tend to affect groups of muscles, and cease during sleep. *Athetosis* is the term applied to peculiar, slow, wriggling movements of the fingers or toes, and is associated with mental deficiency, or organic cerebral disease. Exaggerated movements usually indicate irritative lesion in lateral columns, or contiguous grey matter.

Define the Term Ataxia.

Imperfect co-ordination of movement. Combined movements cannot be made with certitude, and the more complex movements are involved before the simple. The cerebellum and subsidiary centres control such movements, and incoordination may result from disease of these centres, rendering them powerless to emit the necessary influence, or from disease of afferent fibres cutting off the stimulation. There is no loss of muscular power. This is well seen in locomotor ataxia. Apart from the afferent impressions derived from sight and hearing, the co-ordinating fibres run in the posterior columns (Goll, Bürdach, and Flechsig).

What Characteristic Types of Gait are found in Spinal Disease?

Paretic—A shuffling gait, in which the fore part of the foot hangs, and the toes drag, while the foot is usually put down with the outer edge first and needs support. There is

little or no tottering, and the patient stands quietly. A *waddling* gait is rather characteristic of a paralysis below the knee.

Ataxic—A stamping gait. The movement of the foot is irregular and forcible, the toes being sent outwards and upwards, and the heel is brought down with a stamp, the knee is stiff, and the patient keeps his eyes fixed on the ground. There is some tottering or staggering, and movements are jerky, especially on turning.

Spastic—The legs are rigid, while the body is bent forwards, and is lifted stiffly at each step. The legs drag, and the toes are pointed from spasm of the calf muscles, thus tending to catch on any inequality of surface. There is often strong adductor spasm leading to cross-legged progression (*scissors gait*).

SENSORY DISTURBANCE.

What are the Principal Varieties of Cutaneous Sensation?

Those of touch, pain, temperature, and the muscular sense. In disease these may be diminished or increased in intensity. *Diminished* sensibility to touch is known as *anæsthesia*, varying in degree and distribution according to the causative lesion, which, generally speaking, is in the posterior columns and often involving the posterior cornua. Different sensations are conveyed upwards through the cord by different paths, hence one lesion may not abolish all. Pain and temperature travel together. When the sensation of pain is abolished the condition is called *analgesia*. The muscular sense is that which leads to the recognition of degree of muscular contractions, thus enabling us to recognise the position of the body in space. Certain sensations may be retarded in disease, especially pain, as in locomotor ataxia. Common sensibility may be *increased*. This is called *hyperæsthesia*, and is due to an irritative lesion in posterior roots, cornua,

or columns. *Hyperalgesia* denotes increased painful sensation. *Paræsthesia* means subjective sensations not present in health.

ELECTRICAL REACTION.

Describe what is meant by the Reaction of Degeneration.

Following on degenerative changes in the lower motor neurone, there are alterations in the electrical reactions of the muscles governed by the affected nerve area, and also in the nerves implicated. These are known as the *reaction of degeneration or R.D.* The changes are these:—(1) increased excitability to galvanic and faradic currents. This quickly passes off and is followed by (2) a decrease to faradic, though still increased to galvanic; and (3) a decrease to both currents. There is also a change in the polar reactions, *e.g.* A.C.C. may be greater than K.C.C.

What is the Significance of this R.D.?

It means that the nerve and muscles are cut off from their trophic centre, and hence it is present in all cases where there is degeneration of nerves (peripheral neuritis), disease of fibres in anterior root in the cord, or destruction of the centres in the anterior cornua (infantile paralysis, etc.).

REFLEX DISTUBANCES.

What Structures partake in the Formation of a Reflex Arc or Loop?

Any reflex act requires a continuity between an afferent and an efferent nerve. In this the parts concerned are (1) a receiving surface (skin or mucous membrane), (2) a sensory (afferent) nerve with its receiving station (cells in posterior ganglion) and its transmitting fibres (reflex collateral), (3) a discharging station (cells in anterior horn), (4) a motor (efferent) nerve with its end station in muscle. Loss of reflex must follow a break in any part of this loop.

How does the Brain control Reflexes?

It has an inhibitive action. If this be cut off, as by disease of upper motor neurone, reflexes must be exaggerated.

What is the Effect of a Transverse Cord Lesion on Reflexes?

At the point of lesion the reflexes are lost, owing to a break in the reflex loop. Above the lesion reflexes are normal. Below the lesion reflexes are exaggerated, due to cerebral inhibition being cut off. At the upper border of the lesion there is a band of hyperæsthesia, due to irritation of sensory fibres there. Thus a transverse spinal lesion can often be accurately localised.

In Nervous Diseases, what Reflexes are commonly tested?

These reflexes are of two kinds, superficial and deep. The *superficial* ones are elicited by irritation of the skin on any part of the body (by tickling or pinching, etc.), and are named according to the part contracting, *i.e.* plantar, cremasteric, abdominal, thoracic, etc. In disease these may be increased or diminished. Increase indicates excitability of the reflex loop, or that there is loss of inhibitory control. Diminution points to a reduction of the normal irritability of the factors in the reflex arc. The *deep* (or tendon) reflexes are muscular contractions produced by striking a muscle, or its tendon or aponeurosis. They are really not true reflexes, as they depend on the muscular tonus, but this in turn is dependent upon the integrity of the reflex loop. These, like the superficial, may be increased or diminished, and for the same reasons. In the leg the knee-jerk, and tendo-Achillis jerk are tested, and knee and ankle clonus tried for; the two former are present in health, but the latter pair only in disease. In the arms the most important are the triceps and supinator jerks, which may be absent or feeble even in health.

The following signs are often of value, and will frequently

be referred to, in considering the various nervous diseases :—

Babinski's Sign.—On stimulation of the sole of the foot, instead of the normal flexor response in the great toe, there is extension of the hallux, sometimes with a fan-like spreading out of the toes (*phénomène d'éventail*). This phenomenon is pathological except in young infants, and when constant it indicates organic lesion implicating the pyramidal tracts.

Kernig's Sign.—This consists of a reflex contraction of the hamstring muscles, and a wince of pain, when attempt is made to put the sacral nerve-roots on the stretch by flexing the hip to a right angle, and extending the knee.

What Trophic Changes may occur in Nervous Diseases?

Atrophy and *hypertrophy* of tissues may occur, without loss of power at first, though this supervenes later, *e.g.* progressive muscular atrophy, and in pseudo-hypertrophic muscular paralysis. Atrophy may be simple, where muscular fibres are reduced in size, or degenerative, where their structure is altered. The former is seen in late tabes dorsalis, and is due to disuse, the later is due to severance from trophic centres, as in infantile paralysis.

Changes may occur in the *skin* with thickening or atrophy (thin and glossy). There may be overgrowth or loss of hair, or it may change colour. Various eruptions may occur, as erythema, herpes, etc. Acute bed sore is a result of this trophic change, and may occur anywhere, but in unilateral lesions it occurs on the anæsthetic side.

There may be changes in *bones* and *joints*. The former may show arrest in growth, as in infantile paralysis. In tabes dorsalis there may be erosion of cartilages, and wasting of bones, with effusion into the affected joints (knee, hip, shoulder, elbow). The joint commonly becomes disorganised (*Charcot's joint*). General nutrition suffers little in spinal diseases.

Indicate the Changes which occur in the Brain and Cord?

The nutrition of the motor tract in brain and cord depends upon its continuity with the trophic centres at its upper end; that of the sensory, with trophic centres at its lower end. If the motor tract be interrupted by disease or injury, degeneration of the motor nerve fibres with new formation of connective tissue (*sclerosis*) extends downwards from the site of lesion (*descending degeneration*). From a similar lesion in the sensory tract, sclerosis extends upwards (*ascending degeneration*).

II. DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

What is meant by Spinal Meningitis?

An inflammation of the membranes (meninges) of the cord. It may be acute or chronic, diffuse or localised. In most acute cases all three membranes are involved, though one or other may be primarily affected. *Spinal pachymeningitis* (dura involved) and *arachnitis* only occur as the result of injury, or surgical spine diseases, and then are often accompanied by *leptomeningitis* (pia most involved).

ACUTE SPINAL MENINGITIS.

Describe the Nature of this Disease.

It may begin in the cellular tissue around the dura mater, or within its sheath, *i.e. external* or *internal* meningitis. The former is usually local, and can be located clinically by the painful or anæsthetic areas to which it gives rise, and by the altered reflexes. It may be diffuse, associated with caries or other vertebral disease.

Mention the Causes of Leptomeningitis (internal).

It is found as an extension from external or from cerebral meningitis (rarely), or resulting from exposure to cold and

wet. It may arise in connection with acute infections, especially septicæmia and pneumonia. It forms part of epidemic cerebro-spinal fever, and at times of tuberculous meningitis. The organisms found will thus be those of the causative disease.

What is its Morbid Anatomy?

There is active congestion of the pia mater, with effusion of turbid or even purulent exudate, thus bulging out the dura. In severe cases the cord itself is softened, and nerve-roots are often swollen. The inflammation generally involves an extensive area of the meninges.

Describe its Symptoms.

There is generally rigor at the outset, followed by high fever, and local symptoms due to irritation of the cord. These are severe spinal pains spreading to the trunk and limbs, increased by pressure, tonic spasm of muscles of back, with head retraction and often opisthotonos (the spasms may extend to the limbs), early hyperæsthesia of the skin, exaggerated reflexes, and at times Kernig's sign. Later these irritative symptoms give place to those of paralysis, with loss of reflexes, and anæsthesia (often patchy). Retention of urine and fæces may occur. Complete recovery is exceptional, partial is more common with degree of paralysis or anæsthesia, but usually the condition results fatally from asphyxia (paralysis of respiratory centre), asthenia with bed-sores, or from secondary kidney affection.

Indicate the Lines of Treatment.

Complete rest in a darkened room; sedatives (morphia or chloral and bromide) for pain and spasm; strong local counter-irritation to back by cupping and leeches; saline purgatives, antipyretics and diaphoretics may be useful. The inunction of oleate of mercury (even in non-syphilitic cases) is advocated (Gowers).

CHRONIC SPINAL MENINGITIS (Internal).

Mention its Causes.

It may follow the acute form, or be chronic from the outset. The latter is more common, usually in association with chronic cord diseases with sclerosis, or with syphilis.

Describe its Morbid Anatomy.

The membranes are thickened and opaque, and usually more or less adherent to each other by organisation of lymph exudate. Commonly there is increase of the cerebro-spinal fluid. The nerve-roots become fibrous and atrophied, and the spinal cord may suffer by compression. The changes are frequently limited in extent.

Compare its Symptoms with those of the Acute Form.

The onset is more gradual, without fever, and the progress is slower. Paroxysmal pains in the limbs resembling "rheumatic" pains, may be more marked than the spinal pain. The other symptoms follow the same course, viz. :—those of irritation succeeded gradually by those of paralysis with loss of reflexes, muscular wasting, R.D., and anæsthesia. In this form the intensity of the symptoms may vary considerably from time to time. Death is the usual termination with bed-sores, cystitis, or pulmonary complications.

What is the Treatment?

This is rarely of much avail. Rest in the prone position; sedatives and counter-irritation; mercurial inunction, especially in syphilitic cases. When the disease has begun outside the dura, laminectomy may relieve pressure.

MENINGEAL HÆMORRHAGE (Hæmatorrhachis).

How does this Condition occur?

It usually arises from injury and may be intradural, or extradural. Occasionally the latter results from rupture of an aortic aneurysm.

Describe its Symptoms.

The onset is abrupt with pain in the back, radiating into the limbs, muscular spasms and rigidity (irritation). There is no fever until secondary meningitis ensues. Symptoms of paralysis may soon follow, though not usually complete. Recovery is not infrequent.

Hæmorrhage into the substance of the cord (*hæmatomyelia*) has an abrupt onset, with little or no pain, but symptoms of sudden paralysis, with incontinence of urine and fæces. It is usually fatal, and should recovery occur it is never complete.

Indicate the Treatment.

Absolute rest with sedatives for the pain; depletion by venesection, leeches, or cupping; if evidence of pressure, laminectomy.

ACUTE MYELITIS (Softening of the Cord).

What is the Nature of this Lesion?

It is an inflammation of a portion of the cord substance, localised or more diffuse, affecting the cord as a rule in its whole thickness, and accompanied by motor and sensory paralysis, and trophic changes. The disease may be acute or subacute, though these differ mainly in their rate of onset. A chronic form, sometimes described, is really more degenerative than inflammatory.

Mention the Causes which give rise to it.

It is most common in males, frequently following acute infections, though exposure to cold is a common cause. It may be due to injury or pressure from vertebral disease. Sexual excesses or syphilis may set up a less acute form.

Describe its Morbid Anatomy.

The cord is swollen and softened, or even semi-fluid, at the affected part. The grey matter suffers most, and the

meninges occasionally share in the inflammation. The lesion is usually in the lower dorsal region and transverse.

What are the Symptoms?

These vary considerably, depending on the site of lesion, but generally come on rapidly with fever. They are at first irritative but later are those of paralysis (*paraplegia*).

Give the Symptoms in detail of Total Transverse Lesion in the Dorsal Region of the Cord.

Below the lesion there is complete paraplegia, the legs rapidly become rigid, with increased reflexes both superficial and deep. Sensibility is lost in legs and body, though the upper limit of the lesion is usually marked by a zone of hyperæsthesia and girdle sensation. There is paralysis of the bladder and rectum. There is no muscular wasting. Bed-sores and other trophic lesions may appear from pressure, anæsthesia, and difficulty in controlling discharges. Later, with descending degeneration of pyramidal tracts, there is greater spasm of the legs with spontaneous movements and twitchings.

How do the Symptoms differ when the Lesion is in the Lumbar or Cervical Regions?

Lumbar.—Here there is flaccid paralysis below the lesion, with rapid atrophy of muscles, loss of superficial and deep reflexes and pronounced paralysis of bladder and rectum.

Cervical.—With lesion in the lower cervical region there are atrophic paralysis of arms, spastic paralysis of legs, anæsthesia of arms, legs, and trunk, oculo-pupillary symptoms, and involvement of diaphragm and intercostal muscles with dyspnœa. From a high cervical lesion similar symptoms result, but there is spasticity of the arms also, without wasting.

Indicate the Course of the Disease.

The course is variable. Death may be due to septic absorption from bed-sores, cystitis, and nephritis, or, where disease extends upwards, to asphyxia following respiratory paralysis. Recovery may occur, sensation returning first, motor power much later, and is usually incomplete.

What is the Treatment?

Absolute rest, prone, or on the side, if possible; counter-irritation to the spine and mercurial inunction. To prevent bed-sores a water-bed should be used, and the discharges carefully attended to, so that the skin is kept free from irritation. Later, tonics, massage, and electricity may be employed with benefit. In definitely syphilitic cases, push specific treatment.

SYRINGOMYELIA.

Explain the Nature of this Disease.

It is a chronic disease due to a progressive gliomatous new growth around the central canal of the cord. By breaking down of tissue one, two, or more cavities are formed in the cord. These cavities usually occur in the grey matter (or press on it), are filled with fluid, and surrounded by a membrane formed from overgrowth of neuroglia. They usually occupy the cervical and dorsal regions, giving rise later to ascending and descending degenerations. The cause is unknown, but the condition is commonest in young male adults, and runs a very chronic course.

What are its Symptoms?

Its onset is gradual, the arms being first affected, and the symptoms fall into three groups:—(1) *Progressive muscular atrophy* (anterior cornua), with loss of power in the arms (later, owing to secondary changes, this may spread to the legs,

with spastic paraplegia or ataxia); (2) *sensory symptoms*, dissociated anæsthesia, where common sensation is perfect with loss of sensation to temperature and pain (the neck, upper trunk, and arms are most affected); (3) *trophic changes*, skin glossy or thickened, nails thickened, œdema and abscesses, necrosis of bones, and joint affections. (Later with upward spread there may be dysphagia, laryngeal paralysis, nystagmus, etc.)

Indicate the Treatment.

This is largely symptomatic, with attention to the general health. Electricity to the muscles may be beneficial.

TUMOURS OF THE CORD.

Mention the Common Varieties.

Sarcoma, myxoma, and gumma in the membranes, and solitary tubercle and gumma in the cord itself.

To what Symptoms do they give rise?

Those arising from irritation of nerve-roots at the site of lesion, and those from pressure within the cord. Persistent severe pain occurs in the course of the nerves implicated, and is at first usually unilateral. Rigidity may be present, but paralysis is constant. This is gradual in onset, paraplegic in character, but more marked on one side. The anæsthesia is at first partial, but later becomes complete. Muscular wasting occurs.

Indicate the Treatment.

It depends on the cause. For gummata specific treatment should be pushed: tuberculous cases call for suitable measures; a few of the others may be dealt with by operation.

SYSTEM DISEASES OF THE CORD.

ACUTE ANTERIOR POLIOMYELITIS.

(Infantile Paralysis.)

What is the Nature of this Disease?

It is an acute infection, producing destruction of cells in the anterior horns, followed by paralysis and muscular atrophy. It is a lower efferent neurone lesion, and occurs sporadically in children, though at times it is epidemic, and then may attack adults also.

Discuss its Etiology.

Sporadic cases occur in young children, usually under four years, and often attributed to chills or accidents. It is mostly a summer disease. The epidemic form is often fatal. The condition is now known to be caused by a virus, which is found in the brain, cord, mesenteric lymph glands, and the secretions from nasopharynx and gastro-intestinal tract. The nasal secretion can communicate the disease directly, and by carriers, and infection apparently enters by the nasal mucous membrane. The condition has been produced in monkeys by injections of cord emulsions. An organism has been described by Flexner and Noguchi, which grows anaërobically in the form of minute "globoid bodies" in pairs, chains, or clumps. The disease has been reproduced in monkeys by inoculation. The organism has more recently been shown to belong to the class of "filter-passers"; and it travels to the cord by the lymph channels, not by the blood. The stable-fly has been named as a possible carrier. The incubation period is not definitely known, but may be from one to fourteen days.

Describe its Morbid Anatomy.

Changes are first seen in the vessels of the anterior horn, followed by interstitial changes in the grey matter, and

destruction of nerve cells. The degenerative change extends to the anterior roots, and motor nerve trunks. With the increase of neuroglia, the anterior horn becomes sclerosed. The muscles affected are pale, flabby, and atrophied, as their trophic centre is destroyed. The posterior horn, and white matter may also be implicated in the epidemic form, as well as higher centres, but usually the ganglion cells of medulla, pons, and optic thalamus escape.

What are the Symptoms ?

The onset is sudden, often at night, with fever, pains in the limbs, and vomiting or convulsions. After a few days, paralysis of varying degree is manifest. It may be unilateral or bilateral, and may involve anything from part of a single muscle up to all the muscles of several limbs. Reflexes are lost, and there is rapid muscular atrophy with R.D. In a variable time the paralysis begins to improve, and, in the end, only a group or groups of muscles may remain paralysed. Complete recovery is rare. There is also arrest of bony growth, and permanent deformities may result. The symptoms may thus be described in four stages, viz. (1) constitutional disturbance, (2) paralytic rest, (3) regression (4) permanent paralysis.

Noteworthy points are that there is little or no pain, rigidity and spasms are absent, bladder and rectum are not involved, and cutaneous sensibility is unaffected.

The sporadic form is never fatal, but the epidemic type has an average death rate of about eight per cent.

Outline the Appropriate Treatment.

Rest, heat to the spine, low diet, and laxatives should be prescribed. In the early stages the patient should be isolated, and disinfection of secretions and excreta carried out. Urotropin should be employed in full doses for its bactericidal effect. Treatmcnt in the early stages has, so

far, had little success, but later, fresh air, massage, electricity, tonics, passive movements, and gentle exercise are all of value. To prevent deformities mechanical appliances may be necessary, or surgical procedures may be required for their correction. It should be remembered that recovery of muscle is possible up to twelve or even eighteen months.

CHRONIC ANTERIOR POLIOMYELITIS.

(Progressive Muscular Atrophy.)

Define this Disease.

It is a chronic degeneration of the lower efferent neurone, the anterior grey horn and motor roots being sclerosed. Its chief symptoms are therefore abolished reflexes, loss of power, and atrophy of muscle. In some cases as the lesion progresses the pyramidal tracts may be implicated (upper efferent neurone), with resultant spasm and rigidity. The disease then becomes *amyotrophic lateral sclerosis.*

What is its Etiology?

It is commonest in adult males, often associated with exposure to cold and wet, but frequently no cause can be found.

State shortly its Morbid Anatomy.

The anterior horns are sclerosed, the anterior roots, and, to a less extent, the nerve trunks are atrophied, while the affected muscles are pale and flabby, and may show degenerations. Often in the later stages there is sclerosis of pyramidal tracts.

Describe its Symptoms.

The onset is gradual, and the disease usually begins in the arms (one or both) with atrophy of thenar and hypothenar eminences, interossei and forearm muscles, and those of the shoulder girdle. In some the latter, and especially the deltoid, may be first affected. The paralysis of interossei

muscles produces a very characteristic deformity, the "claw-hand" (*main en griffe*). The leg muscles may escape for a time or altogether, though in rare cases the disease may begin there. The further extension is very gradual over a period of years. Fibrillary twitchings are seen in the affected muscles, or may be evoked by gentle tapping. The electrical reactions are often preserved, but, with marked atrophy, diminished irritability both to faradism and galvanism may occur. The disease may cease spontaneously, but commonly progresses slowly to a fatal issue from exhaustion, bulbar paralysis, or intercurrent disease.

In the *peroneal type* of muscular atrophy, which is a hereditary disease with onset in late childhood, the peronei and foot muscles are first affected, and club-foot may result. Knee-jerks are absent. The disease spreads slowly upwards, and the intrinsic muscles of the hands become affected usually after some years.

Indicate the Treatment.

As the disease is progressive and incurable our aim is to retard its onward march by the avoidance of fatigue, and the use of massage and electricity to the muscles. General tonics may be useful. Strychnine may be given hypodermically, gr. $\frac{1}{60}$th to $\frac{1}{30}$th (grm. 0·001 to 0·002) once daily. This is contra-indicated when spasm is present.

AMYOTROPHIC LATERAL SCLEROSIS.

Define this Disease.

It is a condition which, pathologically, is similar to that of progressive muscular atrophy, but in which the degeneration affects first, and chiefly, the upper neurone, and hence spastic symptoms are prominent in addition to muscular atrophy. It is thus a progressive muscular atrophy plus spasm, and, though typical examples of the two diseases offer marked contrast, intermediate cases are often met with.

State shortly its Etiology and Morbid Anatomy.

The former is similar to that of progressive muscular atrophy, though both sexes are equally liable. The essential lesion is a sclerosis of the crossed pyramidal tracts, extending upwards to the pons, medulla, or even cortex, and also of the anterior cornua, anterior nerve-roots, and motor nerve fibres.

Describe the Symptoms in a typical Case.

The onset is insidious with weakness and wasting of the arms, which are often painful on pressure. There is no anæsthesia. Electrical reactions are usually normal in the early stages, though excitability lessens as the lesion advances. The arms are rigid from tonic contraction, the reflexes are increased, and there is a characteristic deformity, viz. :—the arms are held close to the body, with forearms semiflexed and pronated, while the wrists are strongly flexed and the fingers bent into the palms. In the course of a year or so the legs are similarly attacked, though usually the spasm precedes the atrophy. The sphincters remain unaffected. Sooner or later bulbar symptoms supervene, and the patient dies from cardiac or respiratory paralysis, or septic pneumonia. The duration is commonly from one to three years. It should be noted that, once wasting is profound, reflexes may be abolished.

What is the Treatment?

As for progressive muscular atrophy, but strychnine is contra-indicated.

ACUTE ASCENDING PARALYSIS.

(Landry's.)

Describe this Condition.

It is an acute ascending motor paralysis, which begins in the legs, and *rapidly* extends to the trunk and arms.

Reflexes are abolished. There is no muscular wasting, electrical reactions are normal, sphincters are unaffected, and sensory symptoms are very slight. Finally bulbar symptoms appear, and a fatal issue is reached in two to seven days. The brain remains clear to the end. This rare condition is most common in middle-aged males, and is probably due to a toxic affection of the lower motor neurone. It has been noted as a sequel in acute fevers. There is no known efficient treatment.

LOCOMOTOR ATAXIA.

(Tabes Dorsalis.)

Define this Disease.

It is a degenerative condition of the lower afferent neurone, of insidious onset and slowly progressive course, with marked evidences of inco-ordination, trophic changes, and disturbances of special senses.

Discuss its Etiology.

The disease most often attacks men in middle life. Various conditions such as overwork, worry, exposure, excesses, etc., may act as determining causes, but syphilis is *the* essential factor. Formerly it was regarded as a para-syphilitic affection, but, now that spirochætes have been demonstrated in the diseased nerve tissues, locomotor ataxia must be classed as directly syphilitic, in almost all cases. It is a late manifestation, symptoms beginning five to fifteen years after the initial infection.

State shortly its Morbid Anatomy.

There is an extensive sclerosis affecting chiefly the posterior nerve-roots, root ganglia, and posterior columns of the cord. The lesion is, as a rule, most marked in the lumbar region and chiefly in the column of Bürdach, while in the higher reaches Goll's column shows greater implication.

Other tracts may be affected later. In the *brain*, the most common lesion is optic nerve atrophy, but the nuclei of other cranial nerves may suffer. The sensory fibres of peripheral nerves may also show degeneration.

Describe the Symptoms.

These vary much in individual cases, but come on slowly and insidiously. *For convenience* they are usually described under three stages, viz. pre-ataxic, ataxic, and paralytic :—

1. *Pre-ataxic stage.*—Lightning pains, usually in the legs. Girdle sensation—a feeling of constriction or "cord round the waist"—at the upper margin of sclerosis. Various paræsthesiæ may occur such as numbness and tingling. Loss of knee and Achilles tendon jerks (the latter may be lost first). Ocular symptoms :—optic atrophy, Argyll-Robertson pupil (reflex iridoplegia—reaction to light is lost while accommodation is unaffected), spinal myosis (extreme contraction of pupils), paralysis of ocular muscles causing squint and diplopia (double vision).

Priapism may be an early symptom, but later there is loss of sexual desire, and frequently there are urinary difficulties.

These are the symptoms of the early stages, but it is rare to find all present. The ocular paralyses may be transient. If optic atrophy appear early, the onset of ataxia is usually much delayed. Even without this, several years usually elapse before the onset of the ataxic stage.

2. *Ataxic stage*, or stage of inco-ordination, which comes on gradually. The motor symptoms are chiefly in the legs. Early signs are trembling when the eyes are closed, or stumbling in imperfect light. Then the ataxic gait (p. 337) develops. The patient cannot then walk along a straight line, or only with difficulty. When his eyes are closed, and his feet close together, he cannot stand without swaying, or falls if unsupported (*Romberg's sign*). The arms may be

similarly affected later, rendering the patient more helpless. There is no loss of muscular power. Pains, reflexes, and special sense symptoms may be present as in pre-ataxic, but now further sensory symptoms appear. Commonly there is anæsthesia of the soles of the feet with numbness or sensation of walking on "cotton wool." The anæsthesia may be tactile and thermal, and analgesia may also be present. There is loss of muscle sense, and sense of position (orientation). Sensation may be delayed, or there may be faulty localisation. Various visceral disturbances (*crises*) appear :— gastric (epigastric pain, sickness, and vomiting, often with evidences of collapse), vesical (resembling renal colic), rectal (heat and tenesmus), laryngeal (rare, with stridor, dyspnœa, and hoarse cough).

Various trophic changes may occur :—dry skin, hair falls out, nails crack, herpes, *perforating ulcer of the foot*, bones are brittle and thin, and there may be "spontaneous fractures;" Charcot's disease may appear in one or more joints.

3. *Paralytic stage.*—This is reached when the ataxia prevents walking or even standing, and the patient becomes bedridden, weak, and emaciated, with aggravation of the earlier symptoms, especially bladder troubles. Death commonly occurs from some intercurrent disease, *e.g.* pneumonia, or nephritis. Fatal mania may occur.

What is the Treatment?

So far the use of salvarsan has had little success, even when given intrathecally. Mercurial inunction, or with potassium iodide internally, may be of use. Cases in the early stages, or those where the ataxia has come on rapidly after the initial specific infection, are most likely to benefit. Other lines of treatment are general and symptomatic. Rest, or exercise short of fatigue, is indicated according to the stage. Tonics (iron and arsenic) should be given, and

massage, with or without electricity, may be useful. Lightning pains may require to be controlled by morphia, though at times aspirin gives relief. Sedatives, or mild counter-irritation may be beneficial for the crises. The ataxia is often greatly improved by systematic re-education of muscles under graduated exercises (Fränkel), and movements.

PRIMARY SPASTIC PARAPLEGIA.

(Primary Lateral Sclerosis.)

What are the Features of this Condition?

The lesion is in the upper efferent neurone, the pyramidal tracts are sclerosed, producing paralysis with spasm and rigidity. The crossed tracts are chiefly affected, and in the lower cord first. A congenital type is known, following birth injury (p. 376), also a hereditary type. Such sclerosis may occur as a secondary condition in myelitis. The sclerosis may become disseminated later. Its cause is unknown, and it occurs in early middle life. The predisposing causes are those mentioned under locomotor ataxia, but there is no connection with syphilis.

Describe its Symptoms.

The onset is gradual, with weakness and stiffness of the legs, followed by spasm and rigidity. The knee-jerk is increased, ankle and patellar clonus are present, and usually also Babinski's sign. Sensory symptoms and trophic lesions are absent until very late, sphincters are unaffected, and there is no muscular atrophy. A characteristic spastic gait (p. 337) may develop. The disease is very chronic, and may show periods of improvement apart from treatment.

Indicate the Treatment.

Give general tonics as required (strychnine is contra-indicated), warm baths, and massage to spine and muscles

help to allay spasm. Galvanism to spine may be beneficial, or combined with high frequency current. Exercise must be regulated according to the severity of the symptoms.

ATAXIC PARAPLEGIA.

Define this Condition.

It is a disease which presents features both of locomotor ataxia and spastic paraplegia, and is due to a combination of posterior and lateral sclerosis. The symptoms vary depending on which is more prominent, but the usual tendency is for the disease as it advances to become more spastic in type. It should be noted that the sclerosis spares the posterior nerve-roots. The essential cause is unknown. It is most common in men of middle age, and there appears to be no connection with syphilis.

Describe its Symptoms.

The onset is gradual, with progressive weakness and rigidity of the legs and inco-ordination, but without lightning pains or sensory disturbance. The knee-jerk is increased, and ankle clonus and Babinski's sign may be present. The condition may extend to the arms; its course is very chronic.

What is the Treatment?

It can only be general and tonic, or of intercurrent symptoms.

HEREDITARY ATAXIA.

(Friedreich's disease.)

State shortly the Nature and Etiology of this Disease.

Though described as hereditary, it is more often a family disease, affecting several children in the same household, and coming on usually about puberty. It affects both sexes, and its cause is unknown. It is a slowly progressive and incurable disease. Death occurs from intercurrent affections.

What is its Morbid Anatomy?

There is a wide spread sclerosis involving lateral and posterior columns of the cord, the nerve-roots, and also certain cranial nuclei.

Describe its Symptoms.

Inco-ordination of legs and arms, of a somewhat jerky type, is a marked feature. Speech is hesitating, the head is unsteady, and there is a jerky oscillation of the eyeballs (*nystagmus*). The knee jerk is lost, but Babinski's sign may be present. Anæsthesia is rarely marked. Club-foot and other deformities are frequently present.

III.—DISEASES OF THE MEDULLA OBLONGATA.

Give the Reasons which render Disease in this Region so grave.

The medulla contains the great decussations of motor and sensory fibres, and the deep origins of the cranial nerves from the fourth to the twelfth. It also contains the vital centres, respiratory, cardiac, and vaso-motor, as well as the centres concerned with swallowing, vomiting, etc., and centres for secretion, salivation, lachrymation, and sweating. Thus a lesion in this region, however small, may give rise to grave and extensive symptoms.

BULBAR PARALYSIS.

(Glosso-labio-pharyngeal Palsy.)

Define this Condition.

It is a progressive affection, characterised by paralysis of lips, tongue, palate, pharynx, and larynx, and is always fatal.

What is its Etiology?

It is most common in males, and is rare under forty. Various causes have been assigned, *e.g.* exposure, neck in-

juries, etc. It occurs also secondarily to progressive muscular atrophy, and other spinal diseases.

Give the Morbid Anatomy of this Disease.

There is sclerosis of the motor nerve nuclei in the medulla, and of the associated nerve-roots and fibres, with degenerative changes in the trunks and motor endings of the hypoglossal, glosso-pharyngeal, and spinal accessory nerves. Atrophy follows in the tongue, lips, and muscles supplied by these nerves.

Describe the Symptoms in a Typical Case.

The first symptom is usually difficulty in articulation (*dysarthria*), due to lack of precision in the movements of the tip of the tongue, and later to involvement of the lips. The former causes difficulty with letters like T and D, the latter with P, B, M, O, U, etc. There is also difficulty in swallowing, as food collects between the lips and gums. The soft palate becomes involved, and hangs loosely in the throat, giving a nasal twang to the voice, and allowing of regurgitation of fluids through the nose. The constrictors of the pharynx fail to grasp the food, and deglutition is difficult. For the same reason probably saliva dribbles from the mouth. The larynx becomes involved with alterations in voice, aphonia, cough, etc. Later, speech becomes unintelligible, the tongue is tremulous and atrophied, and lies almost motionless in the mouth. Mastication may also be disturbed (involvement of fifth nerve). Intellect is unaffected, but the patient is very emotional. The electrical behaviour of the affected muscles as a rule is little altered, but R.D. may be present. There may be symptoms of involvement of cardiac centre, viz. :—dyspnœa, irregular cardiac action or tachycardia. Further symptoms vary with the nuclei involved. The condition is ended by death from exhaustion, starvation, syncope, asphyxia, or intercurrent complication, *e.g.* bronchitis or pneumonia.

Indicate the Treatment.

It is on general and tonic lines, with great care in feeding.

Does this Condition ever occur acutely?

There is a sudden apoplectic type, which occurs as a sequel of thrombosis in atheromatous subjects, or those with syphilitic endarteritis. Acute in onset, at times with loss of consciousness, its further progress is as already detailed in the chronic form. The paralysis may here extend to the limbs, and is spastic in type. Respiratory and cardiac symptoms are usual, and there may be some pyrexia. Death may occur in a few days or weeks. There may be partial recovery in syphilitic cases, if vigorously treated.

IV.—DISEASES OF THE BRAIN AND ITS MEMBRANES.

GENERAL CONSIDERATIONS.

Describe shortly our present Knowledge of the Localisation of Cerebral Functions.

The frontal lobes are concerned in the higher psychical functions. The Rolandic area (anterior cerebral convolution) is the seat of motor functions, and contains the motor centres for the legs, arms, face, lips, and tongue, in order from above downwards. The motor centre for speech is in the left inferior frontal (Broca's) convolution. In a left-handed subject this centre is in the corresponding right convolution. Irritation of centres in the motor area gives rise to definite combined movements, while destructive lesions cause paralysis of corresponding groups of muscles. The effects of motor stimulation are seen on the opposite side of the body, though in the case of associated movements (eyes, mouth, and trunk, etc.), they may be bilateral.

The cortical centres for cutaneous, muscle, and joint sensation are situated in the ascending parietal (posterior

central) convolution. The visual centres are in the occipital lobes, and angular gyrus, the olfactory in the temporal lobes (uncinate gyrus), and the auditory in the superior temporal convolutions.

What are the Functions of the Basal Ganglia?

They are subordinate centres to the higher control of the cerebrum to which they convey, or from which they receive impulses, but they possess an inherent local power with regard to certain complex reflexes and co-ordination of movements.

Describe the Constitution of the Internal Capsule.

It consists of a great strand of nerve fibres passing between the lenticular nucleus externally, and the optic thalamus and caudate nucleus internally. It has an anterior and posterior limb, joining at an obtuse angle, thus forming the genu, or knee. The motor fibres for the leg and arm occupy the anterior two-thirds of the posterior limb, those for tongue and mouth are at the genu, and those for the face just in front. (The order of the various nerve fibres here differs from that at the cortex, as from above downwards they are shoulder, elbow, fingers). In the posterior part of the capsule the sensory fibres run, and behind these are the visual fibres. The sensory fibres partially mingle with some of the motor fibres for the leg.

Indicate the Effects of a Lesion in or near the Cerebral Cortex.

If moderate in size, such a lesion will produce, according to its site, a monoplegia of face, arm, or leg on the opposite side of the body. A larger lesion may produce a combined monoplegia, *e.g.* brachio-facial, or brachio-crural. To produce a complete hemiplegia a cortical lesion must be very extensive. The cortical centres for the leg lie very close to the middle line on each side, so that a mesial lesion

may implicate both, producing a diplegia, chiefly affecting the legs. The paralysis may be flaccid or spastic in type, usually the former. As the fibres converge passing through the *centrum ovale*, a lesion there will involve more fibres, and hence cause a more extensive paralysis on the opposite side.

What is the Effect of a Lesion in the Internal Capsule?

The fibres in this region are so closely packed that a moderate lesion can produce a complete hemiplegia. It is almost impossible to have a capsular lesion small enough to cause merely a monoplegia. With a capsular lesion in the genu, there is complete hemiplegia on the opposite side, with conjugate deviation of the head and eyes towards the lesion, owing to the unopposed action of muscles supplied from the side which is intact. If the lesion be further back in the posterior limb, the leg is more affected than the arm, and the face only slightly. The sensory fibres are now implicated, hence there is also hemianæsthesia. If at the extreme posterior end, visual fibres are also caught and hemianopia results. A capsular lesion usually produces a hemiplegia of a spastic type.

Mention the Effects of a Lesion (unilateral) in the Crus, and in the Pons.

The former produces a hemiplegia on the opposite side of the face, arm, and leg, but usually also implicates the third cranial nerve on the same side with ocular paralysis. This variety of crossed paralysis is known as *Weber's syndrome.*

A pons lesion at the level of exit of the facial nerve produces a crossed paralysis, viz. :—facial paralysis on same side, with hemiplegia of arm and leg on opposite side. The sixth nerve may also be implicated at its nucleus and paralysed on the side of lesion (convergent squint) This total combination is known as the *Millard-Gubler syndrome.*

Unilateral lesions of pons or medulla below the exit of the facial produce only a hemiplegia of arm and leg.

How is Motor Paralysis estimated ?

By observing the position of the limbs, the complete loss of resistance to passive movements, and the presence of stertor in breathing, or "puffing" of the lips and cheeks.

How is Sensory Paralysis recognised ?

By complete insensibility of the patient to external impressions, *e.g.* sound, light, pinching, or pricking.

CEREBRAL MENINGITIS.

Epidemic cerebro-spinal meningitis has already been dealt with in Part I. p. 38 *et seq.*

Mention the other Forms of Cerebral Meningitis.

The *simple* and *tubercular* types.

SIMPLE MENINGITIS.

What does this Form include ?

It includes inflammation of the dura, *pachymeningitis*, which may be internal or external, and *leptomeningitis*. External pachymeningitis is really a surgical condition, arising from disease or injury of the skull.

Describe the Morbid Anatomy of Pachymeningitis Hæmorrhagica Interna.

In this disease a delicate vascular membrane, of inflammatory origin, forms inside the dura, usually bilaterally, but it may be unilaterally. This membrane may be in layers, into which, from time to time, hæmorrhages occur, and from these a thin laminated clot results. The condition is thus really a *hæmatoma* of the dura.

What are its Symptoms?

These are variable, but, if the hæmorrhage is of some size, those of apoplexy occur. Headache, drowsiness, and optic neuritis may be found. As each hæmorrhage sets up a fresh train of symptoms, their repeated occurrence is suggestive. Death may result from apoplexy.

Indicate its Etiology and Treatment.

It is a rare affection, most commonly in elderly males, either alcoholic or lunatic. The treatment is that of cerebral hæmorrhage (*q.v.*)

Give the Etiology and Morbid Anatomy of Acute Lepto-meningitis.

It may be primary as in cerebro-spinal fever, or as a pneumococcic infection, but is usually secondary in pneumonia or other specific fevers, and in septic states. It may result from injury, or disease of the skull (otitis media).

The changes found are hyperæmia of the meninges, with fibrinous exudate and purulent effusion, affecting almost always the convexity of the brain.

Describe its Symptoms.

In primary cases these are usually described in four stages :—

1. *Premonitory* :—Headache, restlessness, and vomiting.

2. *Irritation* :—With febrile symptoms, delirium, marked restlessness, cerebral vomiting, hyperæsthesia of optic and auditory apparatus, severe headache, and cephalic cry. Reflexes are exaggerated, head is retracted, and Kernig's sign may be present. Convulsions may occur, with spasmodic squint and contracted pupils. The pulse, rapid at first, becomes slow and irregular.

3. *Depression.*—With variable temperature, and stupor (replacing irritation), pupils dilated, persistent squint, retraction of abdomen, and slow pulse.

4. *Paralysis*:—With coma, stertorous breathing, and relaxation of sphincters.

The outlook is always grave, and death may occur within a week of the onset of severe symptoms.

In secondary cases the onset may be gradual, and difficult to diagnose. Lumbar puncture is *the* great aid in diagnosis. The fluid is turbid, the polynuclear cells are increased, and the causative organism may be recognised.

What is the Treatment?

Rest and quiet must be secured; ice to the head; purgation by salines or calomel; chloral and bromides in the early stages; urotropin should be pushed. Tension may be relieved by venesection or by lumbar puncture. If there is a surgical origin, operation should be done.

TUBERCULAR MENINGITIS.

(Acute Hydrocephalus.)

Give the Etiology and Morbid Changes of this Disease.

It is commonest in children under five years, and may be secondary to tubercle elsewhere, or part of a miliary tuberculosis.

The disease attacks the basal membranes, with fibrinous exudate, and effusion of gelatinous, greenish, fibrino-purulent fluid. Tubercles are often abundant attached to the Sylvian and other vessels in their perivascular sheaths. The ventricles are often full of fluid, and the brain tissue is softened and flattened.

Describe the Symptoms.

These are largely as already given for the simple type, but the stages are usually more clearly defined. The prodromal stage is longer, and is marked by emaciation, anorexia, and other signs of the tubercular diathesis. After several weeks the irritative stage is entered upon. The temperature shows

greater oscillation than in the simple variety, head retraction and nuchal rigidity are well marked, and the abdomen is "scaphoid." Vaso-motor paralysis may be evidenced by the *tache cérébrale*. Otherwise the symptoms of this stage, which may last one week, are as already described in detail. In the compression stage optic neuritis is present, and stupor is marked, with the peculiarly shrill and characteristic hydrocephalic cry. The total duration in this variety from onset of severe symptoms is from two to four weeks. It is more rapid in adults than in children. Lumbar puncture here yields in most cases an almost clear fluid. The bacillus may be recognised, and the predominant cells are lymphocytes.

Indicate the Treatment.

In the premonitory stage general measures as for early tuberculosis, but, when symptoms have developed, treatment is similar to that of the simple variety. Mercurial inunction may produce temporary benefit.

HYDROCEPHALUS.

What is meant by this Term?

Apart from the acute form (*tubercular meningitis*) above described, and chronic forms due to mechanical causes (pressure of tumour, etc.), a congenital type occurs from no known cause. The ventricles are greatly distended by clear fluid, their inner lining is thickened and rough, the brain tissue is flattened and thinned, and there may be pressure on cranial nerves. The skull is characteristically deformed. Its sutures are un-united, and the fontanelles remain open. The brow overhangs, and the cranial circumference may be enormously increased, out of all proportion to the size of the face. The superficial veins are very distinct.

To what Symptoms does it give rise?

In addition to the increased size, and altered shape of the head, there is general arrest of development with feeble-

mindedness or imbecility. There are frequent convulsions, sometimes blindness or deafness, power of walking is slight or lost, and failure of nutrition of the body ensues, though the abdomen may be distended. The duration varies very much, but death often occurs from convulsions and coma, or from complication during the first two years, though it is occasionally delayed till much later.

How would you treat such a Condition?

Pressure by means of strapping, or elastic may be tried, but repeated lumbar paracentesis is often necessary, or direct drainage from the ventricles.

SINUS THROMBOSIS.

Define this Term.

It means the formation of blood clot in one, or more, of the cerebral sinuses. It may be *simple* or *infective.*

How do these Types differ?

The simple form affects mostly the superior longitudinal sinus, the infective more often the lateral; the former is non-inflammatory, the latter is inflammatory, and the clot breaks down with pus formation. Meningitis or cerebral abscess may result.

State shortly the Etiology.

The *simple* form occurs in marasmic children, and in adults with debilitating diseases (phthisis, cancer, fevers, etc.). The *infective* type occurs at any age following local injury or disease, especially otitis media, and erysipelas of the scalp.

To what Symptoms does it give rise?

In the *simple* type these may be indefinite or absent. Usually there are headache and vomiting, convulsions, and

drowsiness deepening to coma. The more characteristic signs are tension of the fontanelle, engorgement of nasal veins with epistaxis, or of those of forehead and face.

In thrombosis of *cavernous sinus* the orbital veins are engorged, the eyelids œdematous, the fundus oculi shows congestion and hæmorrhages, and ocular twitchings or palsy may occur. Where the *lateral sinus* is involved the œdema appears in the mastoid region which is tender; the jugular vein is affected by extension, and forms a hard tender cord in the neck. Local meningitis may occur.

In the *infective* type there are in addition septic symptoms, viz:—rigors and sweating, remittent fever, vomiting, and rapid pulse. There is often occipital pain and optic neuritis. Septic pneumonia may occur, and meningeal symptoms are present, followed by coma and death.

What is the Treatment?

For the simple form the cause should be treated, and free cerebral circulation encouraged.

For the infective type operation is required.

ENCEPHALITIS.

Define this Term.

It is an inflammation of brain substance, and may be either hæmorrhagic or suppurative (cerebral abscess). It is usually acute. The *hæmorrhagic* form is associated with infective fevers, though it may arise from cranial injury; the *suppurative* arises by extension from neighbouring parts (ear, orbit, nose, etc.).

ACUTE HÆMORRHAGIC ENCEPHALITIS.

(Polioencephalitis.)

Describe its Morbid Anatomy?

The affected parts are softened, vessels are engorged and thrombosed, hæmorrhages occur, and the nerve structures

are destroyed. These changes involve both grey and white matter, especially the former in the middle and posterior parts of the brain. Various cranial nuclei may be implicated. It is thus a localised condition, and resembles closely acute anterior poliomyelitis in the cord.

What are its Symptoms?

The onset is sudden, with symptoms of meningitis. There may be sudden ocular or bulbar paralysis.

A more diffuse form is at times found, more often in children, following infections. It is more gradual, and may have marked lethargy as an additional symptom (*encephalitis lethargica*).

These forms may be fatal, or end in partial recovery, with varying degree of paralysis.

ABSCESS OF THE BRAIN.

(Suppurative Encephalitis.)

Mention its Causes.

It arises from injury or disease of cranial bones, or by extension from suppuration in accessory cranial cavities, especially the middle ear. In these the abscess is solitary, but in pyæmia metastatic abscesses of small size occur in the brain tissue.

What is the Morbid Anatomy?

The abscess is usually in the white matter, and oftenest in the temporal lobe. In recent cases the abscess cavity is ill-defined, but in the more chronic, there is a definite capsule. There is usually a localised meningitis with dural adhesion over the abscess.

Describe its Symptoms.

In the acute form, resulting from otitis media, there is usually radiating pain from the ear over the head on same

side, fever, vomiting, and rigors. The aural discharge ceases. Following this stage of irritation is one of depression, with drowsiness, slow full pulse, slow respirations (may be *Cheyne-Stokes*), and normal or sub-normal temperatures. Optic neuritis or paralysis may appear later, and coma deepening to death, which may be sudden, from rupture into a ventricle, or gradual. Varying with the site of abscess, there may be localising symptoms :—

(*a*) *Temporal lobe.*—Hemiplegia on opposite side, most marked in the face, with possibly third nerve paralysis on same side.

(*b*) *Cerebellum.*—Giddiness and staggering; nuchal rigidity and head retraction ; hemiparesis on same side may occur, with deviation of eyes away from the lesion. The knee jerk may be exaggerated. Nystagmus is present.

(*c*) *Frontal lobe.*—This is known as the "silent area," and signs may be absent, though hemiplegia may occur on opposite side.

What is the Treatment ?

Operation should be performed when abscess is diagnosed. Prophylaxis should be carried out, by careful attention to any suppurative condition in, or injury to, scalp or cranium.

CEREBRAL HÆMORRHAGE.

What is its Etiology ?

Rupture of a cerebral vessel is most common in men, and after fifty, but may occur even in children. Many causes predispose to it, viz :—the stout, plethoric, "bull-necked" type of build, vascular degeneration (especially that due to chronic renal disease), certain occupations involving vascular strain (carters, etc.), or excessive nitrogenous and alcoholic diet (publicans, butchers, etc.), blood diseases (pernicious anæmia or leukæmia), cerebral softening, and others. The determining causes are vascular strain and

degeneration (arterio-sclerosis and atheroma), but it may occur from rupture of aneurysm.

Mention the usual Site of such a Hæmorrhage.

Usually into the brain substance, due to rupture of the lenticulo-striate branch of the middle cerebral artery, especially on the left side (most directly in touch with the forcible pulsations of the left ventricle). Meningeal hæmorrhage is more often associated with injury.

What Changes result in the Brain?

Some laceration of brain substance always occurs, varying with severity of hæmorrhage. Thereafter, if the patient survives the initial shock, the clot retracts and becomes discoloured, crystals of hæmatoidin form, and a serous cyst may result. Complete absorption is rare. Inflammation occurs around the clot, which becomes encapsulated by fibrous tissue. Secondary degeneration then occurs in the motor tracts (cut off from trophic cortical centres), on the affected side down to the decussation in medulla, thereafter on the opposite side.

Describe the Symptoms.

These may be classified into (1) *premonitory*, which may or may not occur; (2) *primary*, those of the *apoplectic fit*; and (3) *secondary*, those emerging from the primary. In a predisposed subject, headache and giddiness should be regarded as warnings, though the onset is commonly sudden, and may occur during rest or sleep, especially after a full meal, or following some severe exertion.

The apoplectic "stroke" is essentially characterised by coma, coming on suddenly. (An *ingravescent type* is known, where coma is more gradual in onset, becoming complete after some hours.) The face is usually flushed, the carotids may throb violently, the pulse is hard, full, and usually

slow, the breathing is laboured and stertorous, while the cheeks are puffed out at each expiration. The pupils, as a rule, are unequal or dilated, while the eyes and head are turned to the side opposite to the paralysis (*conjugate deviation*—see pp. 362, 384). The limbs at first are flaccid, with abolition of superficial and deep reflexes, but later these return on the sound side, and become exaggerated on the affected side. The patient is unable to swallow, and the urine and fæces are usually passed involuntarily. Thereafter, in some cases, the coma deepens, breathing becomes more laboured, and may be of Cheyne-Stokes type, and death ensues. More often the patient regains consciousness in a few hours or days, with symptoms of *reaction*, viz. :—pyrexia, rapid pulse, restlessness, and sweating. Delirium or convulsions may occur at this stage, and trophic disturbances, *e.g.* bed-sores on paralysed side. These are of unfavourable omen, and the case may end fatally. Recovery is more common, following the period of reaction, which may last for four or five days, but is rarely complete, a partial paralysis or a typical *hemiplegia* (*q.v.*) resulting.

The effects of lesions at different levels have already been indicated (see pp. 345, 361, 362).

Limited lesions may be followed by complete recovery. Basal hæmorrhages are rapidly fatal as a rule. The occurrence of marked febrile disturbance, or delay in regaining consciousness is unfavourable.

Hæmorrhage into the *pons* is associated with hyperpyrexia, and contracted pupils, and is usually quickly fatal.

What is the Treatment of Cerebral Hæmorrhage?

Absolute rest and quiet, with head slightly raised, and ice applied to scalp. Give one minim of croton oil (as bolus with butter), or move the bowels by enema. In strong full-blooded patients venesection often relieves the symptoms. The urine should be drawn off by catheter.

It may be necessary, where coma is prolonged, to employ rectal feeding. Febrile and other symptoms should have suitable attention. Preventive treatment for bed-sores should always be carried out.

EMBOLISM AND THROMBOSIS OF CEREBRAL VESSELS.

(Softening of the Brain.)

What is the Nature of these Conditions?

Embolism is due to blocking of an artery by an embolus (piece of valvular vegetation, or piece of clot from cardiac thrombi). *Thrombosis* is a local clotting of blood, and may follow on embolism. It usually occurs in a vein.

In both the consequent changes depend upon the size and site of the vessels affected, but in both degeneration and softening follow. The softening varies in colour with amount of blood effused into the area affected, and a cyst or cicatrix forms as in hæmorrhage.

Embolism occurs with valvular lesions (chiefly from mitral disease), thrombosis from degeneration of blood vessels, or pressure (by tumour or abscess). The former occurs chiefly in young adults, the latter in elderly persons. In either the condition may be septic.

Describe the Symptoms.

In *embolism* the onset is sudden, without premonitory symptoms. As the left middle cerebral artery is most often blocked, a sudden right hemiplegia occurs with aphasia. The coma may not be deep, and convulsions rarely occur. In *thrombosis* the onset is gradual, following headache, giddiness and numbness, or varying weakness on one side. Paralysis appears slowly, convulsions may occur (cortex often involved), aphasia is less common, as either side may suffer. There may or may not be loss of consciousness, which comes on slowly, following the paralysis.

Bulbar paralysis follows blocking of vertebral artery, while blocking of the basilar causes hyperpyrexia, and may cause bilateral palsy or sudden death from asphyxia.

What is the Treatment?

As for hæmorrhage, but remember that the heart and circulation are weak, and avoid too active measures (severe purgation or bleeding). Where syphilis is the known cause, begin vigorous treatment by potassium iodide.

HEMIPLEGIA.

Define this Term.

It is a paralysis of the face, arm and leg on one side of the body, and is produced by a lesion in the opposite side of the brain. Associated trunk movements (bilaterally innervated), *e.g.* respiration, etc., usually escape.

What is its Etiology?

Hæmorrhage, embolism, and thrombosis are the main causes. Tumour involving the course of the motor fibres to medulla produces a gradual hemiplegia. Injury over motor cortical area, as by depressed fracture, may cause it. A *functional form* may occur in hysteria.

Describe the usual Course of a Hemiplegia.

The mode of onset varies. Commonly it occurs suddenly with coma—"apoplectic fit"; at times, as from tumour, it develops slowly—the ingravescent type—and passes into coma. It may be found on awaking from sleep. There may be no coma, but simply a feeling of cloudiness or confusion, which passes off; or this may merge into coma. When developed there is loss of power on one side of the body, with difficulty of speech (*dysarthria*), or loss of speech (*anarthria*), or there may be *aphasia* (with right hemiplegia). The affected limbs are at first flaccid, but "early rigidity" may soon appear, varying in degree from time to time. This

may pass off, or merge into "late rigidity" some weeks later, which is a permanent condition with contractures, most marked in the arm and hand. Reflexes are lost at the onset, but with rigidity, the deep reflexes become exaggerated, and ankle clonus often appears. The superficial reflexes remain diminished, except the plantar, which is characteristically altered (Babinski). The sphincters are at first relaxed, but, as coma passes off, control is regained. Sensation is usually unaffected, or little, unless the lesion is in posterior part of internal capsule. There is no wasting of muscles until later from disuse. With recovery there is some restoration of movement, but the patient walks with a characteristic paretic gait (see p. 336). This movement is rendered possible by the preservation of the rubro-spinal tract, through which habitual motions of sitting, standing and walking are controlled. Tremors, and choreiform, or athetoid movements may occur in the affected limbs, in which also there are commonly vaso-motor disturbances.

Mention the General Rules which are recognised in a Hemiplegia.

The face is involved on the same side as the limbs, unless in crossed paralysis. The facial paralysis is most marked in its lower segment (compare Bell's paralysis). Paralysis of the arm is usually more complete than that of the leg, the face least of all. The face, leg and arm tend to recover power in the order named. Coarse movements (large joints) return before the finer movements (hand and fingers).

What is the Treatment?

This should be directed towards preventing rigidity, and avoiding contractures. Massage once or twice daily should be begun early to keep up muscular tone and nutrition. Passive movements should be carried out, and galvanism may be helpful, though, in the absence of rigidity, faradism is

better. Once contracture is established, electricity is useless, and friction with passive movements is the only hope.

Explain what is meant by Crossed or Alternate Paralysis.

There is paralysis of the limbs on one side, and of one or more cranial nerves on the other. It is liable to occur in lesions of the crus, pons, and medulla (see p. 362).

BIRTH AND INFANTILE PALSIES.

What Varieties occur, and how are they caused?

There may be *diplegia*, *paraplegia*, or *hemiplegia*. The first two are caused by tissue changes occurring *in utero*, or at birth, the last in early childhood. The chief determining causes are maternal injury or disease during pregnancy, birth injuries, or infectious diseases. Syphilis is a frequent cause. All are associated with some degree of weak mentality, and may later be followed by epilepsy.

Describe the Main Symptoms to which they give rise.

In the birth varieties there is spastic paresis or paralysis, chiefly of the legs, with contractures, and marked adductor spasm, producing "cross-legged progression" when the child attempts to walk. Talipes equinus is a frequent deformity. Tendon reflexes are exaggerated, sensation is unimpaired, and choreiform or athetoid movements may be seen in the arms or legs. In the hemiplegic type, the onset may be marked by convulsions, which are followed by rigidity and contractures on the affected side which atrophies, and may be the seat of purposeless movements.

What is the Treatment?

Should birth palsy be noted early, operation for the removal of the clot (usually a meningeal hæmorrhage) may be done. Specific treatment should be carried out vigorously where there is a syphilitic history, or where the Wassermann

reaction is positive. The affected child requires special education and physical training. Surgical measures may be necessary to correct deformities.

CEREBRAL SYPHILIS.

In what Forms does this occur?

A uniform narrowing may take place in the cerebral arteries (*endarteritis obliterans*), followed by thrombosis and occlusion, with cerebral softening. A *gumma*, or *gummata* may occur in or around the brain, or there may be *diffuse degenerative conditions* (as in general paralysis).

Give the Symptoms.

These may be similar to those of other cerebral lesions, but in syphilis there is often evidence of more than one lesion, the symptoms tend to vary in severity, and to improve strikingly under suitable treatment. They have a gradual onset, often with headache (nocturnal), vertigo, mental dulness, convulsions, etc. Endarteritis is liable to cause hemiplegia, with or without aphasia, of slow onset, and, it may be, temporary duration, followed by one or more similar attacks. Cortical gummata cause localised convulsions (Jacksonian epilepsy), and often slow and persistent monoplegia. Basal affections cause paralysis of cranial nerves. Optic neuritis is common in cortical, much less so in the basal variety. Diffuse general degenerations cause mental symptoms—syphilitic dementia.

Indicate the Treatment and Prognosis.

Mercury (by inunction or intramuscularly) or potassium iodide in large doses should be given. These may be combined. Salvarsan may be tried, but with care. Early cases may be cured, or greatly improved by vigorous treatment. Much improvement cannot be looked for in later cases.

CEREBRAL TUMOURS.

What Varieties occur?

The commonest are *tubercle*, *gummata*, and *glioma* in the order named. Tubercular tumours are often multiple in the cerebellum, or at the base, and occur most frequently during childhood. Gliomata are more frequent in adult life, and in the brain substance. They are usually innocent, but are closely related to sarcoma. Gummata have already been dealt with (*vide sup*), *sarcoma*, and *carcinoma* are more rare, and usually secondary. *Cysts* may arise from hæmorrhage, or faulty development, or may be of hydatid variety.

Describe generally the Symptoms of Cerebral Tumour.

These are very variable, depending on the nature, size, site, and rapidity of growth of the tumour. Because of their constancy certain *general* symptoms are regarded as classical, viz :—

1. *Headache.*—Usually intense, and persistent, though it may be paroxysmal. It may be localised, but affords no certain guide to the seat of tumour.

2. *Vertigo.*—Rarely absent, often early, and specially marked in the case of cerebellar tumour.

3. *Vomiting.*—Commonly persistent, painless, and without food relation.

4. *Double optic neuritis* (choked disc).—Very frequent, and almost pathognomonic.

Other symptoms—*localising*—may occur depending on the site of the tumour, and are those of irritation or destruction of the part affected :—

(*a*) *Prefrontal* area.—Often no motor or sensory symptoms, beyond perhaps unilateral anosmia; general symptoms marked.

(*b*) *Central motor* area (Rolandic).—Jacksonian convulsions, followed by monoplegia.

(*c*) *Parieto-occipital* lobe (angular region).—Visual aphasia, with or without hemianopia.

(*d*) *Basal ganglia* (internal capsule).—Hemiplegia, hemianæsthesia, and hemianopia.

(*e*) *Cerebellum.*—General symptoms severe, marked ataxia, "drunken" gait, with tendency to fall towards affected side. Pressure of tumour may cause cranial nerve palsies, or hydrocephalus. Reflexes may be variable.

What is the Treatment?

Only in syphilitic cases can anything in the way of curative drug treatment be attempted. In these, specific measures should be pushed. In the others, potassium iodide may cause temporary benefit. Palliative measures for the relief of pain, etc., should be employed. Surgically it may at times be possible to remove the tumour, and trephining is often performed for the relief of pressure.

APHASIA.

Define this Term.

Shortly it means the inability to understand spoken words, though hearing is unimpaired, or failure to translate ideas into words, though there is no muscular paralysis of the apparatus of speech. It is a symptom of many cerebral diseases, in which there is a cortical or subcortical lesion, interfering with the receptive, perceptive, or emissive centres of speech.

What forms of Aphasia occur?

To explain these it is necessary to review the apparatus concerned in speech. In acquiring language, impressions are taken up by the ear and eye, and stored in corresponding memory centres, while in another centre is stored the

memory of the movements of mouth and hand necessary to reproduce the impression in speech or writing. In a higher centre intellectual conceptions of word pictures are stored, and any of these centres, or their connecting fibres, may be disturbed, and *aphasia* will result. Thus there are two main types :—(1) *motor*, where the emissive centres are disturbed; and (2) *sensory*, where the interference is with the receptive centres. Each of these types can be subdivided.

What are the Varieties of Motor Aphasia?

The ordinary type occurs where the emissive centre for speech (*glosso-kinæsthetic*) in the third left frontal (Broca's) convolution is involved. The patient can understand what is said, or read to himself, but cannot express his ideas in words. Habitual expressions, like exclamations and oaths, may be retained, or the dumbness may be absolute. This is commonly combined with the further type, *agraphia*, where the centre for writing (*cheiro-kinæsthetic*) in the ascending frontal, or posterior part of second left frontal convolution, is disturbed. Here there is inability to express ideas in writing.

The patient is quite aware of the errors he makes, and there is little mental impairment.

Point out the Forms of Sensory Aphasia.

These are *visual* or *auditory*, dependent on the site of lesion, *i.e.* involvement of visual centre in the angular gyrus causes *word blindness* (*alexia*), while with disturbance of auditory centre in the temporo-sphenoidal lobe *word deafness* occurs. In the former there is loss of memory for printed or written characters, spoken words are understood and can be repeated, but he fails to recognise written or printed words. He can speak, may recognise letters, may or may not be able to write, and may write wrong words or interchange them. He is quite unaware of his errors, though

mentality is little impaired. In the latter, though he hears sounds and words, he fails to interpret them. He speaks, but often quite unintelligibly, and is not aware of his errors, while mentality is considerably impaired.

Though motor and sensory aphasia are thus divided for descriptive purposes it is obvious that pure forms are rare, and *mixed* types common.

It should be noted that in left-handed persons these centres are in the right hemisphere, and that in all, if these various centres are destroyed, the corresponding centres in the other hemisphere can, to a limited extent, take up their function gradually by education.

What Different Views with regard to Aphasia have more recently been advanced?

Those of Marie, who denies the existence of a separate motor centre for speech in Broca's lobe, and doubts the existence of a separate visual centre for words. He assumes one large sensory centre for speech, and holds that motor aphasia is produced by a lesion of the cortical speech centre, plus interference with the subcortical motor fibres.

Give the Causes and Treatment of Aphasia.

The causes involving organic cerebral changes are many, and have already been dealt with in reviewing the diseases. Transient aphasia may result from functional causes (fright, etc.), or from toxic conditions (fevers, uræmia, gout, etc.), and is not uncommon in those with cerebral atheroma.

The treatment is that of the causative condition.

GENERAL PARALYSIS OF THE INSANE.

(Paralytic Dementia.)

Describe the Nature of this Disease.

It is a chronic degenerative condition of the cortex and meninges, and is characterised by progressive paralysis, and

peculiar mental perversion. Its essential cause is now recognised to be syphilis, and spirochætes have been demonstrated in the brain tissues, while the Wassermann reaction is positive in the cerebro-spinal fluid in almost every case. The disease is found chiefly in males between thirty and forty years of age; excesses, mental worry and overstrain may predispose to it.

Give the Morbid Anatomy.

Thickening of the cerebral membranes with degeneration and atrophy of the cortical substance. The cerebral blood vessels show degeneration, and the ventricles contain fluid.

What are its Symptoms?

It begins insidiously some years after the initial infection. Headache, irritability, and excitement are followed by progressive loss of memory and intelligence, with moral perversion. Delusions of grandeur occur. Speech is slurring or blurred, and tremor of lips and tongue is very characteristic. The Argyll-Robertson pupil is present early, and pupils may be unequal. Ataxic or spastic symptoms may occur depending on implication of posterior or lateral columns respectively. Epileptiform convulsions are common, often with transient paralysis. Later, there is complete imbecility and general paralysis. The patient is bed-ridden, sphincters are relaxed, and bed-sores develop, or other complications occur. Occasionally remarkable remissions in the downward course are found.

Indicate the Treatment.

By care, strict diet, and hygiene its progress may be delayed. Asylum treatment is often necesssry. Where syphilis is recent, push specific measures. Symptoms, *e.g.* excitement or mania, may require suitable remedies. Measures should be adopted for the prevention of bed-sores.

DISSEMINATED SCLEROSIS.

(Multiple or Insular Sclerosis.)

Give the Morbid Anatomy of this Disease.

It is characterised by scattered, sharply-defined patches of sclerosis throughout the white matter of the brain and cord. There may be atrophy of nerve cells, but only partial degeneration of nerve fibres.

What is the Etiology?

It is a disease of young adults of both sexes. Various predisposing causes are ascribed, but the essential cause is still unknown. It is not syphilitic.

Describe the Symptoms.

The onset is insidious, and its course is very chronic. The symptoms naturally vary considerably owing to the diffuse lesions. Certain, however, are characteristic when in combination, viz:—*impaired speech* (scanning or staccato), *nystagmus* (jerking or oscillation of the eyeballs), and *intention tremor*, coarse and jerky, affecting chiefly the hands. There may be signs of spastic or ataxic paraplegia, but usually there is spasm with exaggerated reflexes in the legs (one or both). The abdominal reflex is often absent. Temporary remissions may occur, but commonly as the disease advances further parts are involved, cerebral symptoms become marked, and dementia is common. Apoplectiform or epileptiform attacks are liable to occur throughout the course of the disease. Sphincters are affected later, bed-sores commonly develop, and death may ensue from asthenia, or other complication.

What is the Treatment?

Rest and tonics are important. Relieve spasm by massage, warm baths, and galvanism to spine. Potassium iodide is useful.

V. DISEASES OF PERIPHERAL NERVES.

A. CRANIAL NERVES.

OLFACTORY—FIRST PAIR.

(Nerves of Smell.)

How may this (or other) Nerve of special sense be affected by a Lesion?

In (1) nerve endings, (2) nerve trunk, (3) nerve centre.

How may the Sense of Smell be tested?

The patient's eyes being closed, various aromatic substances, *e.g.* cloves, peppermint, etc., are held to each nostril in turn for recognition (ammonia, etc., should not be used, as pungency stimulates the fifth nerve).

What Disturbances of Smell may occur?

Total loss (*anosmia*) may arise from many causes, *e.g.* injuries, lesion of bulb or tract, etc., but also from local obstructive conditions, catarrh or polypi. *Partial loss* (*hyposmia*) may occur. *Perversion* of the sense of smell (*parosmia*) may accompany mental disease or hysteria, but typically it is always cortical in origin—lesions of uncinate gyrus.

SECOND OR OPTIC NERVE.

How may the Various Lesions of this Nerve be classified?

(1) Lesions of the nerve endings (retina); (2) those of the nerve trunk; (3) of the optic chiasma; (4) of optic tract and basal ganglia; (5) lesions of visual centre (occipital cortex.)

Mention some of the Important Retinal Affections which may occur.

Functional disturbances may arise in uræmia and hysteria (blindness); jaundice and santonin administration (yellow vision). Vision may be altered by day or by night, *e.g. nyctalopia*—clear vision by day, but not at dusk or in a dim light (night-blindness), and *hemeralopia*—bad vision by day or in bright light, much improved in the shade or a dim light. The size of the field of vision (measured by a perimeter) may be contracted, or perception of colours (tested by Holmgren's wools) may be disturbed. *Retinitis* may occur in chronic Bright's disease, diabetes mellitus, and in altered conditions of the blood, especially pernicious anæmia and leukæmia.

What Lesions of the Optic Nerve may occur?

Optic neuritis occurs in many diseases, especially in intracranial tumours and tuberculous meningitis, but also in those mentioned under retina.

Optic atrophy may occur primarily, as in locomotor ataxy and disseminated sclerosis, or it may be secondary to optic neuritis, as in chronic alcohol or tobacco poisoning. In optic neuritis there may be no impairment of vision, but with atrophy the visual field is much contracted, visual acuity is diminished, and ultimately there is blindness.

Mention the Effects of Lesions in the Optic Chiasma.

A lesion in the central part will interrupt the decussating optic fibres (belonging to the nasal half—inner—of each retina) and thus causes blindness in the outer half of each field, *temporal hemianopia*. This may occur in pituitary tumours, a lesion at one or other lateral extremity will only interrupt the non-decussating fibres of that side, and thus causes a *unilateral nasal hemianopia*. Two separate lesions, one at each end (very rare), would cause *bilateral nasal*

hemianopia. With more extensive lesions a combined blindness may occur, *e.g. bitemporal hemianopia plus unilateral nasal hemianopia*, which means total blindness of one eye, and temporal hemianopia of the other.

What is the Effect of a Lesion behind the Chiasma?

It must cause blindness of the temporal half on the same side, and of nasal on the opposite side (*homonymous hemianopia*). A cortical lesion (occipital) would have the same effect, but in addition, as the visual centre is involved there would be word blindness (see aphasia).

THE OCULAR NERVES, THIRD, FOURTH, AND SIXTH.

How are the Voluntary Muscles of the Eye innervated?

The *third* nerve (oculo-motor) supplies all the external ocular muscles except the superior oblique, and external rectus; the former is supplied by the *fourth*, the latter by the *sixth* nerve. The third also supplies the levator palpebræ superioris, the sphincter pupillæ, and ciliary muscle.

Indicate the Effects of a Lesion of the Third Nerve.

It may be diseased in its nucleus or along its course, and paralysis or spasm of the muscles supplied will result, depending on amount of disease or pressure. Paralysis of the whole nerve will thus cause drooping of the upper lid (*ptosis*), divergent squint with diplopia, dilated pupil (*mydriasis*), and loss of accommodation. The lesion may be partial, involving only the fibres to certain muscles, or the ciliary fibres alone may be implicated producing *iridoplegia*.

What Varieties of Paralysis of the Iris occur?

(1) *Accommodative*, (2) *Reflex*—failure of reaction to light, while response to accommodation is active (*Argyll-Robertson*

pupil), (3) *loss of skin reflex*—the pupil fails to dilate, when the skin of the neck is pinched (this is due to paralysis of cervical sympathetic.

What are the Causes of Ptosis?

It may occur as a congenital condition, or with hysteria, or it may be a symptom of cerebral disease. It occasionally is seen in locomotor ataxia.

What is meant by Ophthalmoplegia?

It is a paralysis due to a chronic degeneration of the nuclei of the oculo-motor nerves. It may be *internal* (of ciliary muscle and iris); *external* (muscles of eyeball, and upper lid); or *total*, when all the muscles are affected. It is met with in locomotor ataxia, or other syphilitic conditions.

Define the Term Nystagmus.

It is an involuntary rhythmic tremor or oscillation of the eyeballs, generally bilateral and symmetrical. It is usually horizontal, but may be vertical.

Under what Conditions does it occur?

It is common in albinos, and in miners. It may follow blindness, partial or total. It occurs in various organic diseases, especially in disseminated sclerosis, hereditary ataxy, and cerebellar disease.

Has this Symptom any special significance?

It occurs as above in very varying lesions, and is therefore valueless as a localising sign, but its presence aids in differentiating between functional and organic disease. It is not found in the former.

Mention some of the Conditions in which the Third Nerve is implicated.

Nuclear degeneration may be present in certain spinal affections, but the nerve is implicated in many intra-cranial

diseases, *e.g.* syphilis, tumours, and meningitis, the symptoms being those of irritation or paralysis, depending on the amount of pressure or disease.

What Effects are produced by a Lesion involving the Fourth Nerve?

There is defective downward and inward movement of the eye, with diplopia on looking downward. It may be affected by similar conditions to those of the third nerve.

Show the Effects resulting from a Lesion involving the Sixth Nerve.

A paralytic lesion causes internal squint, and double vision on looking to the paralysed side. It may be affected as in the third and fourth nerves.

FIFTH NERVE—TRIGEMINAL.

What is the Character of this Nerve?

It is almost entirely a sensory nerve, the ophthalmic and superior maxillary divisions are entirely so, while the inferior maxillary is "mixed."

Mention some of the Conditions which give rise to Paralysis of this Nerve.

Injury or disease at the base of the brain, medulla, or pons; pressure from tumours on its branches; neuritis (peripheral).

What are the Effects of Paralysis of the Fifth Nerve?

Paralysis may affect the whole nerve, or only certain branches. In the latter the results will only be manifest in the distribution of the affected branch. In the former there is *loss of sensation* throughout the very extensive distribution of the nerve. There is *diminished secretion* in lachrymal, buccal and salivary glands. Trophic lesions may

occur in these tissues. The *corneal reflex is abolished*, the cornea and conjunctiva being anæsthetic. The muscles supplied by the motor root (those of mastication) undergo *atrophic paralysis* and develop *R.D.* The jaw, when depressed, deviates to the paralysed side. There is loss of taste (*ageusia*) in the anterior two-thirds of the tongue, but it may not be total. Pungent odours can not be detected.

SEVENTH NERVE—FACIAL.

Describe the Nature of this Nerve.

It is largely motor in function, but it contains also certain secretory fibres and sensory fibres (taste) in association with the chorda tympani. It is more often paralysed than any other cranial nerve.

How are Lesions of this Nerve classified?

1. Those which implicate cortical fibres, producing *supranuclear paralysis.* 2. Those of the nucleus. 3. Those in the nerve trunk in its tortuous course (*peripheral*), or after its exit through the stylo-mastoid foramen (*Bell's paralysis*).

Mention the Common Causes of Seventh Nerve Paralysis.

The *supranuclear or cortical* form arises from similar causes to hemiplegia, with which it is associated; the *nuclear* from pressure by tumours, hæmorrhage, or softening; the *peripheral* from otitis media, cranial injury, exposure to colds, or pressure by tumour.

What are the Effects produced by Paralysis of the Seventh Nerve?

The affected side of the face is smooth and unwrinkled, immobile, and expressionless, and the face is drawn to the opposite side. There is thus asymmetry while at rest, which on voluntary movement is exaggerated. The brow cannot be wrinkled, and frowning is impossible. The eye cannot

be closed, and there is free lachrymation. The paralysed cheek puffs out with expiration, and food collects between the teeth and cheek. Whistling is impossible, and articulation of labial consonants is impaired. Voluntary movement of the integument by the platysma is abolished. These are the signs in *Bell's paralysis.* If the lesion be *within the Fallopian aqueduct*, it produces all the above signs, and in addition loss of taste in the anterior two-thirds of the tongue (*chorda tympani*), and painful sensitiveness to loud sounds—*hyperacousis*—if the nerve to the stapedius be involved.

If the nerve be affected *between the pons and geniculate ganglion* there are the signs of Bell's paralysis, but taste is preserved, and with auditory involvement *deafness* results.

If the lesion be *within the substance of the pons* taste and hearing are unaffected, but the sixth nerve is usually paralysed. When the lesion is *cortical* there is hemiplegia, the upper face escapes, voluntary movements are more affected than emotional, and the paralysis is not followed by atrophy or R.D. in the muscles involved (these occur in Bell's palsy).

Discuss the Prognosis in Facial Paralysis.

This depends on the cause, but slight cases may recover completely in a few weeks. More severe cases may persist for two to eight months ere recovery begins, or the palsy may remain permanent. In severe cases a *spastic or contractured condition* may appear, and *facial hemi-spasm* occur. *Secondary contractures* only occur where recovery is incomplete. Electrical reactions are important in prognosis. Where no polar changes occur the case recovers rapidly, but with R.D. the outlook is unfavourable.

What is the Treatment?

If possible, the cause should be removed, by surgical means if necessary. In syphilitic cases push specific remedies.

The muscular tone should be maintained by tonics, electricity. and massage. Counter-irritation may be beneficial over the exit of the nerve in the peripheral type, which is usually amenable to treatment.

EIGHTH OR AUDITORY NERVE.

Describe the Nature of this Nerve.

It comprises two quite different sets of fibres, viz.:— *cochlear* for the function of hearing, and *vestibular* for equilibration. The chief symptoms referable to this nerve are deafness, tinnitus, and vertigo.

How may Deafness due to Otitis Media be differentiated from that due to Nerve or Labyrinth Affections?

By tuning-fork tests. In middle ear disease there is loss of aerial conduction, but bone-conduction is preserved. In the others (*nerve deafness*) bone-conduction is lost. Further, in labyrinth disease there is often tinnitus or vertigo, and loss of perception for high-pitched tones (Galton's whistle).

What is meant by Tinnitus, and what is its significance?

It is understood to mean ringing in the ears, though the noises may be buzzing, hissing, or whistling in character. They may be *pulsating* or *continuous*. Broadly, tinnitus points to irritation of some part of the auditory apparatus. The pulsating variety (synchronous with pulse) is found in neurasthenia, temporary Eustachian obstruction (coryza), or at times in intra-cranial aneurysm. The continuous type, when of low pitch, is caused by venous hyperæmia (aggravated by recumbency) or anæmia (relieved by recumbency). When of high pitch it is due, as a rule, to labyrinthine stimulation from wax in external ear, Eustachian obstruction, or to disease of labyrinth. Certain drugs, *e.g.* quinine and salicylates may cause it.

Mention some of the Main Causes of Vertigo.

Vertigo or giddiness is produced in health by rapid rotation or change of position of the body. It is present in many general diseases, largely toxic as from alcohol or tobacco, or constipation and dyspepsia. It is associated with cerebral hyperæmia as in arterio-sclerosis, or anæmia as in aortic regurgitation. Intracranial tumours may cause it by pressure, but it is characteristically associated with cerebellar tumours. In the great majority of cases it is due to an ear disorder, external or internal. Labyrinthine vertigo is known as *Ménière's disease.*

What are the Causes of Ménière's Disease?

Its pathology is obscure, but it may be caused by hæmorrhage into the inner ear, or inflammation there setting up increased tension, or by pressure of tumours on the nerve.

Point out its Symptoms.

Those are of three classes, viz.:—(1) giddiness and reeling, due to affection of semicircular canals; (2) deafness and tinnitus, due to auditory nerve implication; (3) associated bulbar phenomena, *e.g.* nausea, vomiting, etc. The vertigo is sudden in onset, and is paroxysmal, the attacks varying much in duration. The patient commonly falls away from the side of the affected ear.

What is the Treatment of Ménière's Disease?

Rest and quiet during attacks; free purgation, and sedatives. Blisters over the mastoid region are beneficial, repeated once a week. Full doses of quinine with hydrobromic acid may be tried.

NINTH OR GLOSSO-PHARYNGEAL NERVE.

Mention the Effects which arise from Paralysis of this Nerve.

Anæsthesia of the back of the tongue and pharynx,

dysphagia, and loss of taste in the posterior third of the tongue. It is never involved alone.

TENTH NERVE—VAGUS OR PNEUMOGASTRIC.

What is the Nature of this Nerve?

It contains both motor and sensory fibres, and has a very extensive distribution, including, through its lowest roots of origin, motor fibres for the levator palati and larynx, and inhibitory fibres for the heart.

Mention some of the Conditions in which it is affected.

Intra-cranial lesions may affect it totally or partially. In the neck, tumour or aneurysm may implicate it, or it may be involved in injuries. In the thorax it may be compressed by mediastinal tumour or aneurysm. It may take part in peripheral neuritis as from alcohol, lead, or arsenic, post-diphtheritic or post-influenzal, or it may be paralysed in diseases of medulla, and disseminated sclerosis.

Describe the Symptoms of Vagal Paralysis.

These naturally vary with the site of the lesion. If the whole of one vagus trunk be affected there is unilateral paralysis of the palate and larynx, with unilateral anæsthesia of larynx. If both vagi be paralysed, the above effects will be bilateral, and in addition tachycardia and cardiac irregularity occur. Respiration is slow and irregular, and gastric symptoms are liable to occur, *e.g.* gastric dilatation, vomiting, pain, and abolition of hunger and thirst sensations.

Laryngeal paralysis has been considered in detail in Part IV. (p. 252).

ELEVENTH OR SPINAL ACCESSORY NERVE.

What are the Results of Paralysis of this Nerve?

It is exclusively a motor nerve, and it supplies the sterno-mastoid muscle, and part of the trapezius. Hence there is

weakness, or paralysis, and atrophy of these muscles with R.D. The scapula is displaced downwards and outwards.

TWELFTH OR HYPOGLOSSAL NERVE.

Mention the Effects of Hypoglossal Paralysis.

The corresponding half of the tongue is atrophied and wrinkled. It is more commonly affected by intra-cranial lesions, and other cranial nerves are commonly involved at the same time.

B. SPINAL NERVES.

PHRENIC NERVE.

What Lesions are liable to affect this Nerve?

It may be implicated in spinal injuries or tumours, by wounds or aneurysm in the neck, and in the thorax by tumours, aneurysm, or pleural affections. It may partake in a peripheral neuritis.

Give the Resultant Effects.

From affection of both phrenics, bilateral diaphragmatic paralysis results, and breathing becomes entirely thoracic. The paralysis is one-sided where one phrenic only is involved.

Mention the Features of Paralysis of Spinal Nerves.

A lesion of a *spinal nerve* after union of its anterior and posterior roots, but before it branches, is followed by combined motor and sensory paralysis according to root areas. Lesions of *peripheral mixed nerves* are followed by paralysis and atrophy of all the muscles they supply, together with anæsthesia in the area of their cutaneous distribution. Individual nerve palsies can be recognised by a knowledge of their anatomical distribution and function.

NEURITIS.

Define this Term.

It means strictly inflammation of a nerve, but the fibrous sheath and interstitial tissue are usually more affected than the nerve fibres. It may be *localised* or *multiple*.

What is the Etiology of the Simple or Localised Type?

It may be due to trauma, extension of neighbouring inflammation, or cold, and it occurs in gout, syphilis, diabetes mellitus, rheumatism, and in poisoning by arsenic or lead.

Mention the Symptoms.

These vary with the nerve affected. If *motor*, there is twitching with loss of power in the muscles supplied; if *sensory*, pain and numbness in its course. *Trophic* symptoms may occur, *e.g.* herpes, hyperæmia, and sweating.

Give the Treatment.

That of the cause. Blistering and fomentations often give relief. For paralysis, massage and electricity (galvanism).

MULTIPLE (PERIPHERAL) NEURITIS.

Define this Term.

An inflammation affecting several nerves simultaneously, or in rapid succession, throughout the body.

What is its Etiology?

It is due to the effect of *poisons* or *toxines* on the terminations of peripheral nerves, and the lesions produced are usually symmetrical. The former include *alcohol*, arsenic, lead, mercury, carbon bisulphide, and carbon monoxide. The toxines of specific fevers in general, but especially *diphtheria*, influenza, malaria, etc., may give rise to it. Diabetes, gout, syphilis, etc., may act as causes, but more often produce the more localised type.

Give the Morbid Anatomy.

Inflammatory changes occur in the interstitial tissue followed by marked atrophy and degeneration in nerve fibres. These are most evident at the peripheral terminations.

Describe the Symptoms.

The *alcoholic* variety may be taken as the type.

The onset is insidious, and is marked by pains, tingling, and numbness in the extremities. Muscular cramps are common, especially in the calves, and vaso-motor spasm may cause local syncope ("dead fingers or toes"). Muscular tenderness on compression, especially in the calves, is very characteristic. Later, progressive paralysis causes the patient to take to bed. This is specially marked in the extensors, producing foot and wrist drop. The face and sphincters are seldom affected; other muscles become involved, and death may result from implication of respiratory muscles. The affected muscles become soft, and atrophied, R.D. is present, and tendon reflexes are lost. The skin is commonly hyperæsthetic to pain. Mental symptoms may occur, *e.g.* delirium, hallucinations, loss of memory, etc. Muscular contractures may follow.

In the *post-diphtheritic* form, the paralysis may be confined to the *eyes*, *palate*, or *heart*.

What is the Treatment?

Complete rest in bed (water bed in bad cases). No alcohol should be allowed. For relief of pain antipyrin or morphia should be given, and hot soothing applications used locally. Sedatives may be necessary to procure sleep. For the paralysis the use of strychnine hypodermically is valuable, followed by massage and electricity. Nerve or general tonics may be employed, and suitable dieting should be prescribed. Passive movements may be necessary for contractures. In all cases of peripheral neuritis, the cause

should be at once removed, if possible, followed by general treatment as above.

VI. FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

NEURALGIA.

Define this Term.

It means paroxysmal pain in the course of a sensory nerve ; usually there is no structural change discoverable.

Give the Etiology.

It occurs chiefly in adults, as the result of exposure to cold and damp, from peripheral irritation (carious teeth), and is specially liable to occur in those who are nervous or debilitated.

What are the Symptoms ?

The pain may vary in character and intensity, but is always paroxysmal. "Tender points" are a very constant feature, on pressure over the exit of the nerve from a bony foramen, or at its passage through fascia or over bone. The pain is frequently associated with reflex muscular twitchings, secretory disturbances, and vaso-motor or trophic changes.

Special terms are employed to indicate implication of special nerves, *e.g.* trigeminal, sciatic, brachial, intercostal, etc.

Mention the Special Features of Trigeminal Neuralgia.

Trigeminal neuralgia or *tic douloureux* is a very severe form which occurs in the fifth nerve. It rarely attacks all three divisions ; two adjacent divisions may be affected, but usually it is confined to one, and most commonly the supra-orbital. It is very rarely bilateral. Paroxysms of agonising pain occur in the affected area, accompanied by involuntary muscular spasm, lachrymation, or salivation. Such attacks

often occur spontaneously, but may be induced by slight stimuli, *e.g.* chewing of food, washing the face, etc.

What is the Treatment?

Causes of irritation should be removed when possible. Improve the general health by generous diet, tonics, massage, and electricity (high frequency). For the relief of pain hot soothing applications, counter-irritation, etc. may be tried, and quinine, salicylates, or gelsemium (alone or combined with butyl-chloral hydrate) should be pushed. Morphia hypodermically may be necessary, or galvanism with positive pole over tender point may be helpful. Absolute alcohol may be injected into the affected nerve, or into the Gasserian ganglion in trigeminal type. Surgical measures may be necessary, either nerve stretching or excision. The corresponding ganglion may be excised—the Gasserian in trigeminal type.

MIGRAINE.

(Hemicrania—Sick-headache.)

Explain the Nature of this Affection.

It is characterised by paroxysmal headache with nausea, followed by vomiting, which gives relief. It may be preceded by visual disturbance.

It is largely a family disease, and begins in childhood, towards puberty, recurs throughout the working years, and tends to disappear in later life. The paroxysms may be induced by worry, overwork, eyestrain, and altered conditions of health. Migraine and epilepsy may alternate in the same patient.

Describe the Symptoms.

The classical symptoms have been referred to above. The *headache* is usually unilateral, and localised at the outset to one spot (temple, eyebrow, etc.), from which it later radiates all over one side of head, and neck. The scalp is tender to

pressure. There is increasing *nausea*, which culminates in *vomiting*, and this is followed by sleep and relief. The *visual aura* may take the form of blurred vision (blind spots), zig-zag coloured spectra, temporary hemianopia (partial or total). The headache is generally referred to the side opposite to that of the visual sensations, *i.e.* if these are on the left side of the visual fields, there is usually right-sided headache, and *vice versa*. An attack rarely lasts longer than twenty-four hours.

What is the Treatment?

For the attack.—Absolute rest and quiet, cooling lotions to the head, saline purge, and phenacetin or antipyrin with citrate of caffein. Strong coffee is also recommended.

In the intervals.—Regulate the general health by dieting and tonics, and remove any possible source of peripheral irritation.

EPILEPSY.

Define this Condition.

It is a chronic paroxysmal affection characterised by sudden attacks of unconsciousness, with convulsions (major or *grand-mal*), or without convulsions (minor or *petit-mal*).

What is its Etiology?

It usually begins between the ages of ten and twenty. It is often hereditary, either directly, or there is a family history of nervous disease, insanity, etc. Reflex or remote causes may determine the attacks, as fright, injury to head, phimosis, worms, etc. In women there may be a menstrual relation.

Give the Pathology.

No causative lesion has been found. The symptoms are due to a functional excitation of the cortical centres. The particular area which starts the epileptic explosion probably determines the nature of the *aura* or warning.

Describe the Symptoms.

A typical epileptic fit comprises three stages :—

(1) *Aura* or *warning.*—This may be of the most varied character, vertigo, epigastric, visual, auditory, twitchings or tingling, or it may be psychical. The *aura* stage is brief, or may be absent.

(2) *Fit.*—Usually following a piercing shriek or moan (epileptic cry), the patient suddenly falls. A *tonic* stage supervenes, in which all the voluntary muscles, including those of respiration, become suddenly rigid. The face and lips become cyanosed, the pupils dilate, and become insensitive to light. The conjunctival reflex is lost. This stage lasts from thirty to forty seconds, then merges into the *clonic* stage, in which violent jerking occurs in all the voluntary muscles, rapid at first, and gradually becoming slower. The eyes, which during the tonic stage have been drawn to the side of greatest spasm, now show rapid clonic jerks towards that side. The face loses its cyanosis, air re-enters the lungs, and is jerked out in short puffs, and a froth of saliva, usually blood-stained, appears at the mouth (due to biting of the tongue). At this stage urine and fæces may be passed. The convulsions last for two or three minutes, then the patient remains in a state of—

(3) *Coma* with stertorous breathing, flaccid limbs, and often profuse sweating, and contracted pupils. After about ten minutes the coma passes off, and the patient may vomit, or wake up with a headache, or may pass into a natural sleep.

One such attack may be rapidly followed by another, without recovery of consciousness, and this may continue for some hours (*status epilepticus*). Here there may be hyperpyrexia, and death from exhaustion.

Petit mal.—Attacks of this minor form may be overlooked, the loss of consciousness is so transient, and no convulsions occur. The patient may not even fall, but may only stop

speaking, look strange for a moment or two, then continue where he left off. The face pales during the attack, and pupils dilate. Flushing of face follows. Such minor attacks may be succeeded by grave post-epileptic manifestations.

What Post-epileptic Conditions occur?

(1) *Automatism* in which the patient performs some unusual and inappropriate act, of which later he has no recollection. Thus he may proceed to undress, or to empty his bladder, or he may commit some serious crime.

(2) *Mania* with homicidal impulses.

Indicate the Prognosis.

Some cases recover completely, the intervals between fits gradually lengthening. The outlook is more favourable when the intervals are long. In many cases mental weakness develops, and becomes permanent. In some, fatal injuries may occur during the attack.

What is the Treatment?

If any reflex cause exists it should be removed or remedied. The general health should be maintained, and dangerous occupations avoided. Bromides are the most active anti-epileptic drugs, and should be pushed till fits are controlled. Thereafter their use should be continued for a time, then gradually withdrawn. Signs of *bromism* may necessitate temporary rests from the drug. Zinc oxide (in pill), borax, and nerve tonics have been employed.

When the fits are *nocturnal*, a full dose of bromide should be given shortly before bed time.

Immediate treatment during fit.—If there is time at onset use amyl nitrite. Loosen all tight clothing, keep patient flat, and insert cork or gag between the teeth to protect the tongue. During the convulsion, guard the patient from injury. *In the status epilepticus* give full doses of bromides *per rectum*, while chloroform controls the convulsions.

JACKSONIAN EPILEPSY.

What is understood by this Term?

A condition set up by an irritative lesion in the cerebral cortex, and characterised by epileptiform convulsions of localised onset, without necessarily loss of consciousness.

State its Etiology.

It may arise from cortical injury, or from tumour, abscess, etc., over the motor area. Gumma is a frequent cause. (Although of organic origin, it is convenient to class it with other allied convulsive disorders.)

Describe its Symptoms.

A Jacksonian fit often begins with a subjective sensory aura, *e.g.* tingling, numbness, or twitching, localised to some particular part. Then follows tonic spasm of that part, and clonic jerking. This may remain confined to one muscle, or group, or may gradually spread to others. Thus a whole side may be involved. Consciousness is retained unless the convulsion becomes general (rare). Such fits are followed by localised and transitory paralysis in the parts convulsed.

Focal lesions of a sensory cortical area will produce analogous sensory fits. Many fits (50 to 100) may occur daily.

How is the Site of Lesion localised?

By the point of onset of successive fits. The motor centre for that part is the seat of greatest irritation.

What is the Treatment?

In syphilitic cases use potassium iodide, etc., vigorously. In other cases surgical treatment is necessary. Bromides may be valuable in limiting the fits.

INFANTILE CONVULSIONS.

Discuss the Nature of these Attacks.

They are epileptiform in character, and usually occur during the first two years of life. The symptoms are less violent than those of true epilepsy. Recurrence may be frequent, unless the cause is removed.

To what Causes are they due?

Rachitic and hereditarily neurotic children are specially liable to fits. Causes may be classed as reflex, toxic, or organic. *Reflex* causes include teething, worms, phimosis, gastro-intestinal irritation. *Toxic* conditions comprise the infectious fevers, in which, in children, convulsions may replace the initial rigor of the adult. Asphyxial convulsions may occur in all cases with dyspnœa, *e.g.* pneumonia, whooping cough, etc. Intra-cranial *organic* lesions may excite convulsions, as in polio-encephalitis, followed by hemiplegia or diplegia; or injuries to head, and meningitis.

What is the Treatment?

Remove any possible source of irritation, and treat the general condition. During a fit, give the child a warm bath, and afterwards a grain of calomel or grey powder, followed by castor oil. Potassium bromide in small doses may be useful, continued for several days.

TETANY.

Mention the Features of this Condition.

It is characterised by bilateral tonic spasm of the hands and feet, usually painful, with increased excitability of muscles and nerves.

What is the Etiology?

It is commonly associated with rickets in children, and in adults it may follow removal of parathyroids (in thyroi-

dectomy), or it occurs as a grave terminal symptom in gastric dilatation. It is known to occur at times during pregnancy or lactation.

Discuss its Pathology.

It is commonly accepted that the parathyroid glands produce an internal secretion which neutralises metabolic toxins; under certain conditions these may be produced in excess, and by their action on peripheral motor neurones set up tetany. If the glands be removed, the toxins exercise their action unopposed.

Describe the Spasm of Tetany.

The posture produced is very characteristic, and may persist during sleep. The hand becomes cone-shaped (*main d'accoucheur*), the fingers are extended at the interphalangeal joints, flexed at the metacarpo-phalangeal joints, and adducted with the thumb flexed into the palm, the hollow of which is deepened. In the feet the toes are flexed, the ankle extended, and the foot may be inverted. The spasm may be still more widespread involving the whole limb or the trunk, and lasts often for several days, passing off slowly. Pressure on a nerve trunk, or even compression of the limb sets up a typical spasm (*Trousseau's sign*). The electrical reactions are unduly active (*Erb's sign*). Percussion over a muscle or its motor nerve sets up a lively contraction (*Chvostek's sign*), best seen in the facial nerve.

What is the Treatment?

Rest, improvement of general condition, and removal of cause if possible, are the lines to follow. Chloral and bromide are helpful in lessening spasm, as also are warm baths or packs. In tetany following thyroid removal, the spasms may be arrested by the injection of solution of a calcium salt into a vein, or subcutaneously.

HYSTERIA.

Explain the Nature of this Condition.

It is a psychical disorder, characterised by defects or perversions of character and disposition, without failure of intellect, and by disturbances of nerve function not referable to any substantive disease. It is due to diminished inhibitory power, and to increased susceptibility to suggestion, or stimuli.

What is its Etiology?

Many cases have a nervous heredity. It is commoner in women than in men, and during adolescence or adult life. Nerve exhaustion, however produced, is a predisposing cause. Physical or emotional shocks, *e.g.* fright, grief, etc., may act as exciting causes, as also may disease or irritation of the genitals, and sexual excess.

Describe the Symptoms.

These are many and varied, and often closely simulate organic disease. (It should be remembered that this may coexist with hysteria.) The symptoms roughly fall into four groups, viz. psychical, sensory, motor, visceral and vasomotor;—

(1) *Psychical.*—Deficient inhibition is the main feature with increase of susceptibility to suggestion, leading to emotional changes, altered disposition, whims, and craving for sympathy.

(2) *Sensory.*—Pain and hyperæsthesia of the skin, especially over certain spots — "tender points" — *e.g.* vertebral, mammary, etc., on which at times pressure may cause a hysterical paroxysm, best seen in the left inguinal region in many cases. These are described as *hysterogenic areas.* A very common finding is *localised anæsthesia,* never limited to an individual nerve-area, but confined to a limb in part, or as a whole, or to one half of the body

(the most common distribution). Limited areas may thus take the form of shoe, sock, stocking, mitten, glove, sleeve ("segmented" type). Loss of sense of pain is even commoner than loss of touch. Special senses may all be affected similarly, and usually on one side. In the eye there is contraction of visual field, and of colour fields, or apparent blindness, again unilateral.

(3) *Motor.*—Irritative or paralytic phenomena may occur. Of the former, the *hysterical fit or paroxysm* is best known. It varies much in type and severity, and is usually associated with emotional outbursts, or with violent muscular spasms, but consciousness is never quite lost, the tongue is never bitten, and urine or fæces are never evacuated (*Hystero-epilepsy*). *Catalepsy* may occur, and a state of *trance* may follow a paroxysm, or may come on spontaneously. *Hysterical paralysis*, flaccid or spastic, may affect any of the voluntary muscles, but is rarely correct in its anatomical distribution, or the posture assumed, and is unaccompanied by R.D., or atrophy unless from disuse. The deep reflexes are never lost, and ankle-clonus does not occur, nor does Babinski's sign.

(4) *Visceral and vaso-motor.*—Bradycardia, tachycardia, or palpitation may occur. Digestive disturbances are common, *e g.* flatulence, dysphagia, *globus hystericus*, vomiting, anorexia. Phantom abdominal tumour may occur, or urinary disturbances, but rarely hæmorrhages.

What is the Treatment?

In a paroxysm.—Firmness and tact, assisted perhaps, in emergency, by a cold water douche, will usually rouse the patient. In severe cases an anæsthetic may be required.

In the intervals.—Remove the cause, if possible. Gain the confidence of the patient, and, by suggestion, strive to correct the disorder. Isolation on the Weir-Mitchell principle, in the hands of a firm but kindly nurse, is of great

value. Regulate and improve the general health. Bromides may at times be required. Massage and faradism are valuable adjuncts.

NEURASTHENIA.

Indicate the Nature and Etiology of this Condition.

It is rarely primary, commonly secondary to mental or physical over-strain. Drug habits or excesses, the toxins of infective diseases (influenza, enteric fever, etc.), organic general or nervous disease may be the determining cause. A *traumatic* form occurs following shock or injury, as in "railway spine."

Describe the Symptoms.

They are chiefly subjective. Undue fatigue, lack of concentration, depression, and introspection are marked features. Muscles are flabby, and may be wasted, but there is never true paralysis. Tremor of the hands or limbs is common as part of the asthenia. Knee jerks are often exaggerated, true ankle-clonus does not occur, and plantar reflexes are flexor, if present. Insomnia and gastro-intestinal atony are common, with anorexia, dyspepsia and constipation. There are usually vaso-motor disturbances.

What is the Treatment?

Complete change, rest and tonics, supplemented by massage, electrical and hydro-therapeutic measures. Isolation, on the Weir-Mitchell principle, may be required in severe cases.

CHOREA.

(St Vitus' Dance.)

Define the Nature of this Condition.

It is a functional disorder characterised by irregular, purposeless movements of limbs or body. It is most common

in childhood, and bears a very close relationship in symptoms and complications, to rheumatism.

What is the Etiology?

It is most frequent in girls between the ages of five and fifteen years. Neuropathic heredity and debility predispose to it. It is closely related to rheumatism in the patient or the parents. In the adult, it may occur during pregnancy (especially the first). Fright, shock or emotion may act as exciting causes. Excessive study or eye strain are also cited, but are more likely to act by aggravation.

Discuss its Pathology.

As no constant lesion has been found, various theories have been put forward. Punctiform cortical hæmorrhages have been observed, and embolic plugging of small capillaries in the basal ganglia. The latter led to Kirke's embolic theory, but endocarditis, though very common in chorea, is not constant. The *microbic* theory of origin is now largely accepted, and various organisms have been described, notably the isolation of a diplococcus (Poynton and Payne), which can reproduce the disease in animals, and can thereafter be recovered from their cortical vessels. The close relationship of chorea with rheumatism, and of both with endocarditis, strongly suggests a circulating toxin as the essential cause of both.

Describe the Symptoms.

Irregular, jerking, and purposeless muscular movements are the chief feature, affecting the limbs, trunk, face, soft palate and tongue, or confined to one side (*hemi-chorea*), and increased by voluntary movements or emotion. They cease during sleep. Muscular hypotonus is present. Respiration may be jerky and irregular. The heart is often rapid and irregular, and may show varying hæmic murmurs, or definite evidence of endocarditis. Associated rheumatic manifesta-

tions are common, and there may be mental weakness. Recovery is gradual, and cases are liable to relapse.

What is the Treatment?

Rest in bed and *isolation* are important, with arsenic given in increasing doses. Where movements are very violent, chloral is a valuable aid, alone or combined with bromides. Later, open air life and regulated exercise, with tonics.

PARALYSIS AGITANS.

(Parkinson's Disease.)

What is the Nature of this Condition?

It is characterised by progressive muscular weakness, constant rhythmical tremor while awake, and later a striking posture and gait. It is commonest in old men, without obvious cause or definite pathological changes, save those of senile degeneration.

Describe the Symptoms.

The onset is usually gradual with general muscular weakness. Tremor appears first in one or both hands, or in feet. At the outset this is transitory, and controllable by will and voluntary movement, but later, as more muscles are involved, it is persistent during both rest and movement, and is incontrollable. The tremor may completely affect one side before it involves the other. The head usually escapes. The tremor is short, rapid, and rhythmic, and includes joint movements, *e.g.* "pill-rolling" action of thumb and fingers, etc. There is distinct muscular rigidity producing a characteristic posture and gait. The body is bent forward when the patient stands, the face is expressionless, immobile, and mask-like (*Parkinsonian mask*), the forearms and hands are flexed, and the fingers bent as if holding a pen. The knees are also bent. When he walks his steps are at first slow and shuffling, but become quicker

as if "chasing his own centre of gravity." If gently pushed forward, or pulled backward, he proceeds by short, quick steps, and may be unable to stop (propulsion and retropulsion). The gait is described as "festinant." *Speech* may be slow and hesitating, or slow at first becoming quicker ("festinant" like the gait). The only *sensory symptoms* are feeling of muscular fatigue, flushes of heat, and tingling.

The course is prolonged, but after a time the faculties fail, there is wasting and weakness, and death occurs from asthenia, or some intercurrent disease.

What is the Treatment?

Results are unsatisfactory. Regulation of life, diet, and habits; arsenic may be useful, or sedatives. A combination of arsenic, cannabis indica, and opium has been advocated (Gowers). Massage, passive movements, and galvanism may be beneficial.

RAYNAUD'S DISEASE.

What is the Nature of this Disorder?

It arises from vasomotor spasm, with varying constriction of the peripheral arterioles, producing local changes of variable degree, viz. syncope, asphyxia, or gangrene. The distribution is usually symmetrical.

Discuss its Etiology and Pathology.

This disease is most frequent in females, between twenty and forty years of age. The usual exciting cause is cold, and in most cases there is a neurotic family or personal history. The symptoms arise from local spasm of the vessels, and resultant imperfect blood supply. The symmetrical distribution strongly suggests a circulating toxin as the cause.

Describe the Symptoms.

1. *Local syncope.*—The tips of one or more fingers or toes, ears, or nose, suddenly become cold, waxy, and "dead."

In these tingling and numbness are experienced. The spasm lasts from a few minutes to several hours, and may be followed by a painful and burning reaction in the parts affected.

2. *Local asphyxia.*—This more severe grade has a similar duration. The affected parts become suddenly congested, livid, swollen, and painful. As the attack passes off there is often free sweating of the parts affected. "Chilblains" come under this category.

3. *Local or symmetrical gangrene.*—This, the most severe variety, may follow either of the first two. There is a localised necrosis, with formation of blebs, which burst leaving ulcerating sores. The necrosis may be more extensive, especially in young children. The bones as a rule escape necrosis. Symmetry is a striking feature, and pain is often severe in the initial cyanosis. There are frequent recurrences, and the condition is sometimes associated with paroxysmal hæmoglobinuria.

What is the Treatment?

Improve general conditions, and often the affection gradually passes off. Warmth, gentle massage, and galvanism to the affected parts are beneficial. Morphia may be required where pain is severe. Vaso-dilators (nitrites, nitroglycerine, etc.), may be tried.

DISEASES OF THE MUSCLES.

THE MUSCULAR DYSTROPHIES.

(Myopathies.)

What does this Term include?

It comprises a group of cases of muscular weakness and atrophy localised in certain muscles, the fibres of which are primarily diseased, while the motor nerves and nuclei remain healthy. All those affections are due to congenital

defect, though the age of onset of noticeable symptoms varies considerably, but most cases begin in childhood or early adult life. In all there is a marked hereditary element or predisposition.

There are two main classes described.—(1) Those in which the affected muscles waste from the beginning; (2) those in which certain muscles show a preliminary false enlargement preceding the atrophy. In reality atrophy and enlargement may be combined in the same case.

Give the Morbid Anatomy.

The changes affect primarily and chiefly the trunk muscles and the larger muscles of the limbs. In some muscles there is atrophy of the fibres with marked increase of interstitial tissue. In others there is increase in connective tissue, which becomes loaded with fat cells, and as a result of pressure the muscular fibres waste and degenerate.

PSEUDO-HYPERTROPHIC PARALYSIS.

What are the Features of this Affection?

It is the most rapidly progressive form of myopathy, and usually begins in childhood, affecting chiefly boys, often several in the same family. The disease is transmitted through the female line. Enlargement is seen in the following muscles:—the calves, infraspinati, glutei, deltoids and triceps. Atrophic change is seen particularly in the latissimus dorsi, teres major, and the lower fibres of the pectoralis major.

Describe the Symptoms.

These are entirely *motor*. Owing to muscular weakness the child shows less activity than his comrades. This tends to be progressive. He stands and walks in a characteristic manner. On standing, the abdomen is prominent, and the back is hollowed (lordosis). The gait is wide-based and

waddling. The method of rising from the recumbent posture is still more characteristic. He rolls round, gets on his hands and knees, and gradually pushes himself up till his knees are straightened. Then he quickly transfers his hands to his knees, and by alternate movements of the hands up the thighs he gradually pushes himself into the erect posture. As described, he "climbs up his thighs." The knee jerks and electrical responses diminish progressively with the muscular wasting. As the disease advances, the shrinkage of muscles and contractures produce increasing weakness and impairment of locomotion, and finally the patient becomes helpless.

PRIMARY ATROPHIC MYOPATHY.

What is the Nature of this Affection?

It is a less common type, affecting either sex, and mostly of later onset. Three varieties are described, in each of which different muscles primarily atrophy :—

1. *Erb's juvenile type.*—The muscles of shoulder-girdles and upper limbs atrophy first, those of the lower limbs later. The deltoids, spinati, forearm, and hand muscles often escape.

2. *Facio-scapulo-humeral type* (Landouzy-Dejerine).—This may begin in infancy, the facial muscles being affected first. The orbiculares palpebrarum and oris are early involved, while the ocular muscles, and those of the tongue and jaw escape. The shoulder-girdle and upper-arm muscles become affected, and lastly the muscles of the spine and legs.

3. In this type the atrophy begins in the leg muscles, spreading later to the arms.

These all run a prolonged course, and the patients commonly die from intercurrent diseases to which, owing to muscular weakness, they are particularly liable.

Indicate the Treatment of Muscular Dystrophies.

General tonics, massage, exercises, and galvanism should be employed. Cod-liver oil and malt are specially indicated, with arsenic or strychnine (often best given hypodermically). The diet should be generous, and special precautions against chest troubles should be advised.

THOMSEN'S DISEASE.

(Myotonia Congenita.)

What is the Nature of this Affection?

It is a rare congenital condition of the voluntary muscles, in which attempts at voluntary movement set up tonic spasm. The disease tends to run in families. The muscular fibres are hypertrophied, but in this change the fibrillar element is lacking.

Describe the Symptoms.

On passing from rest to movement a muscle (or muscles) is thrown into tonic spasm, which passes off gradually, allowing the intended movement to be carried out. The muscles of the lower limbs are most often affected, and are usually enlarged. They respond readily to stimuli, and their electrical reactions are characteristically altered. Both to faradism and galvanism there is increased excitability, and the contraction set up is unduly persistent. A continuous galvanic current induces a series of contractions (*myotonic reaction*). Sensory functions and the reflexes are unaffected.

The spasm is decreased by warmth and continued movements, but increased by cold, excitement, or mental worry.

What is the Treatment?

No curative treatment exists, but measures to allay spasm may be carried out, *e.g.* warm baths, massage, and regulated exercises.