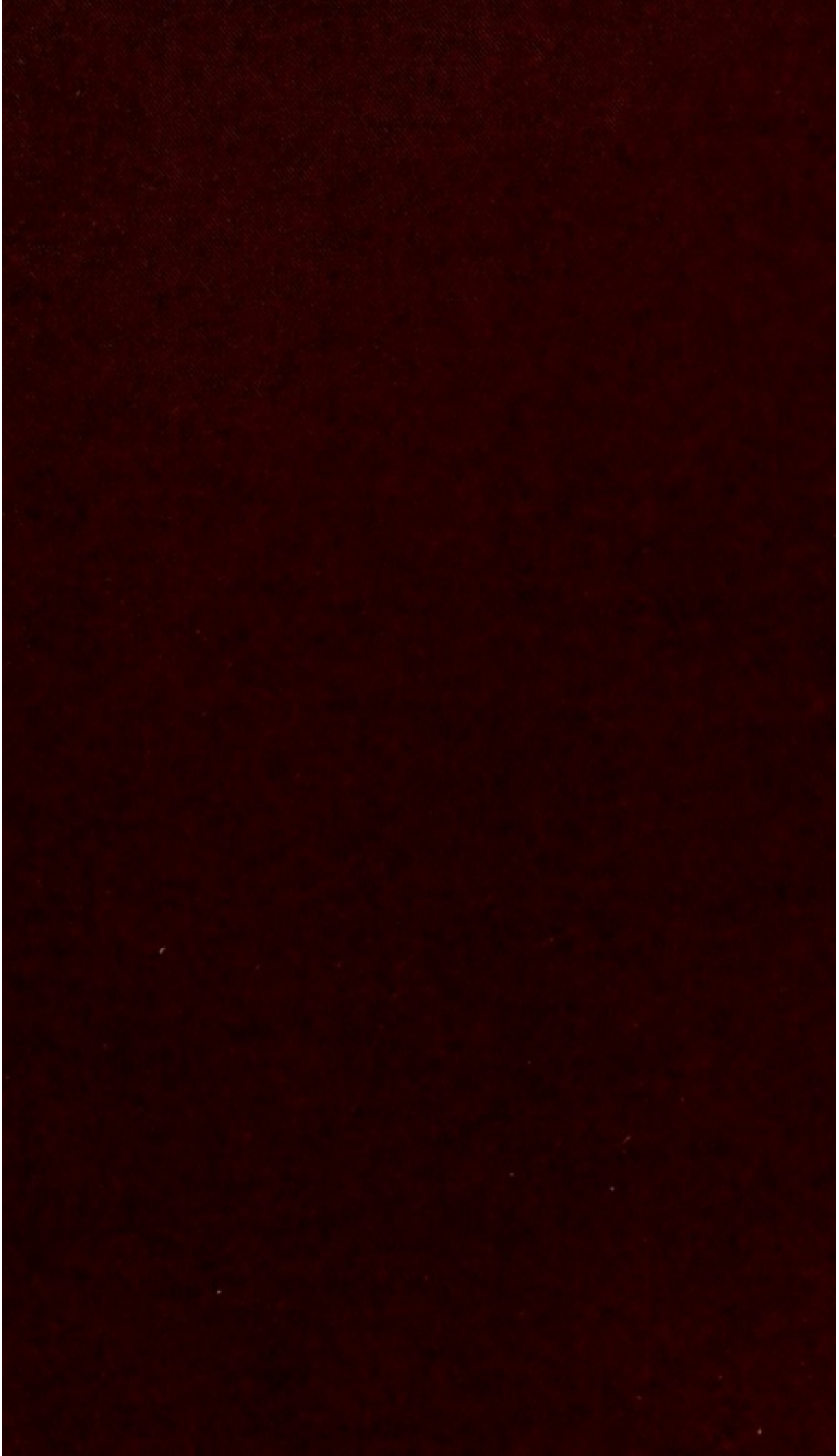
**Medical morbid anatomy and pathology / by Hugh Thursfield and William P.S. Branson.**



# MEDICAL MORBID ANATOMY AND PATHOLOGY

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# PREFACE

Our object has been to provide students with a concise account of the morbid anatomy of medical diseases and enough pathology to explain the lesions described.

For the convenience of members of our own school we have given marginal references to specimens in the Museum of St. Bartholomew's Hospital which illustrate the statements made in the text, and recommend that these specimens be studied with the aid of the descriptive catalogues to be found in that Museum.

We have written from our own experience when this has been adequate, but are much in debt to many friends and authors of text-books. These obligations we acknowledge gratefully, and hope that the result may be found of practical value.

H. T.

W. P. S. B.

*June*, 1909.

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# CHAPTER I.

## INTRODUCTORY.

I. Inflammation. II. Retrogressive Tissue Changes. III. Infective Granulomata. IV. The Nomenclature and Classification of Tumours. V. The Life-history of the Commoner Parasitic Worms.

*I.*

## INFLAMMATION.

"**Inflammation is the reaction of irritated and damaged tissues which still retain their vitality.**" This is the definition offered by Grawitz, and it is the most acceptable one. In medical pathology the irritant causing inflammation is generally bacterial; but it may be derived from ingested poisons or poisons elaborated by abnormal metabolic processes, or result from the defective excretion of normal metabolic products.

**The purpose served by inflammation is the repair of damage**, including under this head the neutralisation of poisons, chemical or bacterial: the removal of irritant substances (whether simple foreign bodies such as a piece of an aseptic ligature, or the dead products of the damage causing the reaction, or bacteria): and finally, the regeneration of tissue to replace that which has been destroyed.

The most prominent immediate results of inflammation are: (1) **An accumulation of cells.** (2) **An effusion of fluid, in and around the damaged area.** It is proposed in the following pages briefly to describe: I. The mechanisms by which these aims are achieved. II. The characters of, and the parts

played by, the cells and fluids thus accumulated. III. The sequelae of inflammation.

## I. The mechanisms by which cells and fluid are accumulated in inflamed areas.

### (*a*) The Cells.

1. **Chemiotaxis.**—There exists between certain living cells and various chemical substances, including dead tissues and foreign bodies such as bacteria, a relation evinced either by an attraction or a repulsion exerted by the chemical substance upon the cells. This relation is called "Chemiotaxis." When attractive, the relation is called "positive," when repellent, "negative" chemiotaxis. The phenomenon is well illustrated by the behaviour of a myxomycetes (which may be described as a multi-nucleated amoeba) found in tan pits. If such an organism be placed upon a moistened surface near a drop of an infusion of oak-bark, it moves actively towards and into the infusion. This is positive chemiotaxis. If, on the other hand, the infusion be replaced by a 0·5 per cent. solution of glucose, the organism moves with equal rapidity away from it. This is negative chemiotaxis.

A little higher in the scale of living things the bearing of chemiotaxis upon inflammation becomes pronounced. At one stage in development the larval forms of certain metazoa consist of an ectoderm, invaginated at one point to form the endoderm, while between the two lies the mesoderm, a semi-liquid mass containing amoeboid cells. This is the body-cavity of the larva. In such an organism the phenomena of inflammation are simplified by the absence of blood-vessels, and are limited to cell-accumulation. If a foreign body be thrust into the larva it becomes surrounded by wandering mesodermal cells attracted to it by positive chemiotaxis.

In vertebrates there exists a non-vascular tissue analogous to the body-cavity of the larval metazoon above described, the cornea, which in health contains no blood-vessels, but an ample supply of lymph-channels. If the cornea of a rabbit be cauterised, or if a piece of it be removed, a whitish opacity appears in the immediate neighbourhood of the injury. This opacity is due to an accumulation of small round cells, and

appears without any evidence of vascular dilatation around the cornea or of proliferation of the corneal corpuscles. The cells are wandering leucocytes attracted from the surrounding lymph-spaces by positive chemiotaxis.

When a more intense inflammation is excited, as for example by the injection of a pus-forming organism like the staphylococcus pyogenes aureus, there appears within twenty-four hours a dense packing of leucocytes around the damaged zone. But the accumulation is now attended by obvious changes in the veins at the periphery of the cornea. They are dilated, and it is apparent that leucocytes are passing from the blood-channels into the lymphatic spaces. This passage of leucocytes will be presently described in detail. It is enough, at present, to note that it is largely the result of positive chemiotaxis.

Under certain conditions the attraction of leucocytes fails to manifest itself, or may be exchanged for an active repulsion. Both these events are embraced by the term "negative chemiotaxis." Negative chemiotaxis attends irritants whose characters place them at one or other extreme in the scale of virulence, and results in an inflammatory exudate consisting chiefly of fluid. Thus infections due to bacteria of relatively low virulence, like the tubercle bacillus, or to bacteria of a high degree of virulence, such as those responsible for some forms of acute purulent peritonitis, are alike marked by an exudate composed largely of fluid and to a small degree only of cells, though the character of the fluid varies in the two cases.

2. **Vascular changes attending inflammation.**—It has been noted that some of the leucocytes accumulated in areas of inflammation are wandering cells from neighbouring lymphatic spaces. A second contingent, haematogenous leucocytes, is derived from the blood as a result of the following vascular changes. By these changes leucocytes are massed upon the outskirts of the scene of operations, whence they are drafted into the fighting line under the influence of chemiotaxis.

The first effect of an injury or irritation, as regards the blood-vessels of the injured part, is **dilatation** of the arterioles, venules and capillaries in succession, associated with an increase in the rate of the blood-flow through them. The corpuscles travel in the centre, or "axial" stream, while at the periphery is a zone of cell-less plasma. Presently the

pace of the blood-current abates, and the axial stream of corpuscles widens out. At its edge, now situated close to the wall of the vessel, leucocytes are seen, travelling more slowly than the axial current, adhering momentarily to the wall, and then advancing in a series of jerks as though they, or the wall, had become sticky. Finally the vessel becomes completely blocked with cells, and the blood-flow through it ceases. This is the stage of **Stasis**. With the gradual stoppage of the current the **Diapedesis** or migration of leucocytes commences, and is attended by an escape of fluid from the distended vessels. The leucocytes aggregated within the venules and capillaries penetrate the delicate walls of these channels by the exercise of amoeboid movements, and are shortly to be seen in large numbers in the lymph-spaces outside the vessels from which they are derived, actively making their way to the site of damage under the influence of chemiotaxis. This leucocytic migration is attended in its later stages by the escape of red-corpuscles in varying abundance. It is generally accepted that the escape of red-cells is a passive process, a means of exit being found, in part at least, in the orifices made by the migrating leucocytes.

3. **Changes in the fixed connective-tissue cells.**—There is a third important source of supply of leucocytes, namely the fixed connective-tissue cells of the injured area, especially the endothelial cells lining capillaries and lymph-spaces. It is characteristic of endothelial cells that under the influence of irritants they cease to be flat, and, becoming swollen, proliferate actively, giving rise to large hyaline leucocytes. This phenomenon has been studied most extensively in relation with the endothelial lining of the peritoneum, but is general. Further, connective-tissue corpuscles themselves, and even highly-specialised cells like muscle-fibres, are capable of a similar proliferation.

To sum up, the cells of the leucocytic accumulation comprise: (1) Wandering cells from the lymphatic spaces. (2) Haemal leucocytes, released from the blood by the series of changes which lead to diapedesis. (3) Leucocytes supplied by the proliferation of fixed connective tissue cells, particularly of the endothelial cells lining lymph-spaces and capillaries.

### (*b*) The Fluid of the Inflammatory Exudate.

Little is known of the mechanism which produces the fluid exudations of inflammation, but whenever injury or irritation leads to vascular dilatation an increased escape of fluid from the blood is a feature of the process. The characters of, and the services rendered by, the fluid exudate will be presently considered.

## II. The characters of, and the parts played by, the cells and fluid of the exudate.

### (*a*) The Cells of the Exudate.

We have seen that the leucocytes of inflammation own a variety of origins. They have also a variety of duties, though our knowledge on this point is incomplete.

The prime functions of the leucocytes, apart from their relations to immunity which are here omitted from consideration, appear to be the following: (1) To remove particulate irritants such as bacteria and dead tissues, and to counteract the poisons locally at work. (2) To regenerate destroyed tissues. The former of these functions is accomplished partly by the ingestion, and subsequent intra-cellular digestion, of the offending particles by certain types of leucocyte;[1] partly by the extra-cellular action of ferments either excreted by the leucocytes, or liberated from them upon their death in the inflamed zone. The leucocytes which evince the capacity for ingesting particulate bodies are called **Phagocytes.**

Of the leucocytes concerned with inflammation some are essentially blood-leucocytes (Haematogenous). They originate in the bone-marrow and thence enter the blood-stream, reaching the inflamed zone by diapedesis. Such are polymorphonuclear, eosinophil, and mast cells. Others are directly derived from tissue-cells (Histogenous). They originate in proliferated endothelial cells, connective-tissue corpuscles, or lymphoid tissues, and arrive direct upon the scene of action without passing through the blood-vessels. Such are a certain propor-

[1] Particles which resist digestion, such as granules of carbon, and occasionally living bacteria, may be ingested, carried to the lymph-glands and there deposited.

tion both of large hyaline leucocytes, and of lymphocytes. A third group is formed by the remaining fraction of these two last types. Though essentially histogenous they enter the blood-stream and reach the area of inflammation by the same route as the haematogenous forms. This group is therefore termed Histo-haematogenous.

1. **Polymorphonuclear cells.**—These are the most abundant leucocytes in foci of acute inflammation. They are actively phagocytic, especially as regards bacteria, and are the typical pus-cells. In addition they either excrete, or liberate upon their death, antidotes to bacterial poisons, and substances assisting in the digestion and solution of bacteria and dead tissues. They may die and be dissolved *in situ*, or may be ingested by large hyaline leucocytes. They play no part in tissue-regeneration.

2. **Eosinophil cells.**—These are seldom numerous, and are practically not phagocytic. It is presumed that they excrete some substance conducive to repair, but nothing definite is known of it. They play no part in tissue-regeneration.

3. **Mast-cells** (coarsely-granular basophil cells).—These are not abundant, and of their functions nothing is known.

4. **Large hyaline cells.**—These are not so common as the polymorphonuclears. They are phagocytic, and readily ingest other cells and cell-débris, but attack bacteria less actively than do the polymorphonuclears. In areas of chronic inflammation they may form giant-cells, either as a result of fusion of several individuals, or of nuclear multiplication in a single one. They are actively concerned in the regeneration of tissue.

5. **Small hyaline cells** (Lymphocytes).—These are the preponderating cells in areas of chronic inflammation, and form the "small round cell" infiltration found in such lesions. They are not phagocytic. It is a matter of doubt whether or not lymphocytes take part in tissue regeneration, but there is evidence that they may be transformed into "plasma-cells."

6. **Plasma cells** are uni-nuclear cells. The nucleus is small (relatively to the cell-body), oval or round; excentrically situated, and stains deeply. Plasma-cells are common in all areas of chronic inflammation, especially those due to the virus of syphilis. Their rôle and their fate are both uncertain.

### (*b*) The Fluid Exudate.

The fluid exudate of inflammation differs from ordinary lymph in being richer in proteids, approximating in this respect to blood-plasma. This proteid richness is in part attributable to the leucocytes and tissue-cells which become disintegrated within the exudate, in part to an increased transudation from the distended capillaries of the area concerned. In addition the fluid exudate contains digestive ferments and bactericidal substances. It is usually rich in fibrin-forming elements (except in the case of very virulent bacterial infections), and tends to coagulate, especially upon serous and mucous surfaces, but also within the tissues. Some varieties of bacterial infection excite fibrin-formation to a much more pronounced degree than others, the diphtheria bacillus and the pneumococcus being especially gifted in this respect.

The functions of the fluid exudate are (with others, which are in dispute) the following:

1. It flushes the injured area and dilutes the irritant.
2. The development of fibrin within it tends to limit the spread of inflammation.
3. It exercises a digestive function, by virtue of the cells disintegrated within it, and thus aids in the removal of dead products of irritation.
4. It probably contains substances, similarly derived from cells, capable of hindering the growth of bacteria.

## III. Sequelae of Inflammation.

1. Resolution.
2. Suppuration.
3. Gangrene.
4. Fibrosis.

**1. Resolution.**—The term "Resolution" has no strict pathological significance, but is commonly and conveniently used to describe a rapidly-established ascendancy of the defensive over the destructive forces at work. Both the irritant and the dead products of its activity are rapidly removed: there is at no time a gross accumulation of necrotic

material; and the inflamed part is restored to health with a minimum of structural alteration.

2. **Suppuration.**—When the irritant is virulent the destruction of leucocytes and tissue-cells outstrips, for the time at least, the reparative capacity of the organism. In consequence the destroyed leucocytes lie where they fall, suspended in a fluid exudate. Such an exudate containing dead leucocytes is called **pus**. Pus may be a thick and creamy, or a thin and turbid fluid, and in the latter case is often blood-stained. Thick pus, the "laudable pus" of old writers, indicates an irritant of moderate severity. The abundance of cells in it is evidence of active positive chemiotaxis. Thin turbid pus is evidence of negative chemiotaxis, and thus of a virulent irritant. When pus is formed one of two events occurs. In the one case, although the day goes against the leucocytes at the spot where the irritant is most active, the surrounding zones fare better. Here, remoter from the poison, leucocytic accumulation and fibrin-formation produce a barrier[1] by which the inflammatory agent is confined, and the result is a localised collection of pus—an **abscess**. In the other case the virulence of the irritant is such that no marked accumulation of leucocytes occurs: positive chemiotaxis is in abeyance, and no barrier is formed, and thus a large area of tissue may become the seat of a diffuse suppuration. (Phlegmonous inflammation.)

**The fate of an abscess.**—(1) "Pointing" and discharge. (2) Inspissation and encapsulation.

(1) "**Pointing.**"—Collections of pus acutely formed are commonly under pressure, and thus tend to advance in the direction of least resistance until they reach a surface. Here, under the combined influence of pressure and of the softening influence of the inflammation they excite in their immediate neighbourhood, they "point," perforate the surface, and discharge their contents.

(2) **Inspissation and encapsulation.**—Chronic abscesses, resulting from irritants of relatively low virulence, show less inclination to extend. Their fluid constituents tend to be absorbed in course of time, leaving a pultaceous mass of cell-débris, prone to infiltration by lime-salts. In this way

[1] The barrier of leucocytes and fibrin which forms the immediate wall of an abscess is called "granulation-tissue" (*v.* p. 9).

the inspissated material may be converted into a chalky plate or stone. Coincidently the chronic irritation of the mass produces tissue-regeneration about itself, as will presently be described, with the result that eventually the remains of the abscess are enclosed in a firm fibrous capsule.

3. **Gangrene** implies a widespread and rapid death of tissue in bulk. It may result from other causes than inflammation, for instance obstruction to blood-supply, but with such examples we are not here concerned. Inflammatory gangrene is the consequence of a wide-spread onslaught of virulent bacteria leading to the rapid death of large areas of tissue. In respect of medical pathology inflammatory gangrene most often affects the lung.

4. **Fibrosis.**—The extent of tissue-regeneration is proportionate rather to the duration than the severity of inflammation. Acute inflammations cause little of it, chronic inflammations much. Further, whereas no tissue is immune to inflammation, the regeneration of tissues lost as a result of inflammation is, in man, almost confined to the production of one type only, namely fibrous tissue. Thus highly specialised structures, nervous tissue, muscle, etc., when destroyed by inflammation, are in the main replaced by fibrous tissue. This replacement is termed **Fibrosis**, and is a fruitful source of ulterior disease owing to the inevitable tendency of inflammatory fibrous tissue to contract with time. Whether upon the skin, or within the lung, liver, kidney, or elsewhere, newly-formed fibrous tissue gradually cicatrises, deforming the organ affected and crippling its functional activity.

**Source of the new tissue.**—If the surface layers of the wall of an abscess be examined microscopically they are found to consist of delicate strands of fibrin, enclosing in their meshes leucocytes of various kinds. Somewhat deeper the stroma assumes a greater density, its fibrils are thicker and are interspersed with spindle-shaped cells and fine capillary blood-vessels. Such material is called "**granulation tissue**." The spindle-shaped cells are **fibroblasts**, derived from the histogenous leucocytes and from pre-existing connective-tissue cells at the margin of the inflamed area. They are presently converted into definite connective tissue corpuscles, their protoplasm becoming modified into parallel fibrils of young fibrous tissue, while their nuclei undergo a gradual shrinkage.

The capillary loops seen in granulation tissue are believed to be derived from buds, given off from pre-existing capillary loops at the periphery of the area, and subsequently channelled.

## CLINICAL VARIETIES OF INFLAMMATION NOT YET ENUMERATED.

**1. Catarrhal inflammation.**—This term is applied to inflammations of surfaces covered by mucous membrane. It is attended by swelling of the membrane and by an exaggeration of the normal secretion of mucus. When severe, catarrhal inflammation commonly leads to ulceration.

**2. Membranous or croupous inflammation.**—This signifies an inflammation of a surface, either mucous or cutaneous, attended by an abundant deposit of fibrin, which forms a membranous pellicle upon the affected part. The type of this variety is diphtheritic inflammation, though other organisms besides the diphtheria bacillus are capable of causing it.

**3. Serous inflammation.**—This implies an inflammation characterised by an abundant coagulable fluid exudate. Its simplest type is the inflammation produced by a blistering fluid; but certain bacteria of a low degree of virulence afford examples of it. This is particularly the case with the tubercle bacillus acting upon a serous membrane like the pleura. The term "serous inflammation" has a purely clinical significance.

**4. Ulceration.**—An ulcer is a surface which has lost its natural covering of mucous membrane, endothelium, or skin, either as a result of inflammation or of infiltration by malignant tumours. Inflammatory ulcers are produced by a chain of events analogous to those which produce an abscess, but the affected site being superficial, the dead products are removed as rapidly as they are formed, and do not accumulate. The floor of an inflammatory ulcer, like the wall of an abscess, is composed of granulation-tissue.

## II.

## RETROGRESSIVE TISSUE CHANGES.

The terminology of retrogressive changes is very confused, because the stages and manifestations of impaired vitality lying between health and death pass one into another by imperceptible gradations and often coexist in the same example. It is possible to define more or less exactly the following four groups, with the qualification that in practice no one of them is likely to occur alone:

1. Atrophy.
2. Necrosis.
3. Degenerations.
4. Infiltrations.

1. **Atrophy** indicates wasting of a tissue without any qualitative alteration in its constituent elements. Nevertheless it is a rule of pathology that highly-specialised structures such as nervous tissue, glands and muscles, are less resistant to an injurious environment than those less specialised, such as fibrous tissue: and in consequence the highly-specialised elements in an atrophic tissue suffer first, and most profoundly. Atrophy is usually associated with fatty degeneration.

**Causes of atrophy.**—The nutrition of a tissue depends very largely upon its functional activity, and thus the commonest cause of atrophy, in the sense defined, is disuse. Many atrophies are physiological, for example those affecting the ductus arteriosus and the ductus venosus at birth: the wasting of the thymus gland after the first two years of life, and the involution of the uterus after parturition. The commonest example of pathological atrophy is the wasting of muscles observed about a joint rendered immobile by disease.

2. **Necrosis** means tissue-death. Its causes are (*a*) Mechanical injury. (*b*) The action of poisons, whether chemical or bacterial. (*c*) Sudden arrest of nutrition, brought about generally by obstruction of the blood-supply (as is the case with infarction of the viscera and gangrene of the extremities), but sometimes by a failure in the nutritive function of the nervous system (as is the case with some acute forms of bed-sore).

Cell-death is evidenced by changes both in the nucleus and cell-body. The nucleus ceases to retain ordinary nuclear dyes, while the cell-body becomes distended by coagulated lymph, whence the term "**coagulation-necrosis.**" It is believed that the death of the cell permits the entrance of lymph, the latter being coagulated *in situ* by a substance liberated upon the death of the nucleus. An area of coagulation-necrosis may become liquefied by the ferment action of bacteria. On the other hand it may be liquefied almost from the beginning, as is the case with necrotic areas in the brain: for here the intercellular lymph (cerebrospinal fluid) is not coagulable. In either case the result is described as "**colliquative necrosis.**" The necrosis of tissues *en masse* (excepting the extensive necrosis of bone which results from inflammatory detachment of periosteum) is called Gangrene. Arrest of the blood-supply to an extremity, in the absence of putrefactive organisms, produces a gradual blackening and mummification of the starved area. (Dry Gangrene.) In soft tissues such as the lung, and in the extremities when putrefactive organisms have gained access to the dead tissue, Gangrene is "Moist."

The fate of necrotic tissues has been in part described in the section upon inflammation, where the extra-cellular digestion of them, their removal by phagocytosis, or by the pointing and discharge of abscesses, and their encapsulation by fibrous-tissue, have been briefly recounted. It remains to mention **Caseation,** a common termination of coagulation-necrosis. Caseation means the conversion of the necrotic material into a white or yellowish substance having the appearance and consistency of cheese. Caseation is a particular feature of necrosis produced by the tubercle bacillus, and by the virus of syphilis. As will be seen below, caseous accumulations are very prone to infiltration by lime-salts.

3. **Degenerations.**—Prolonged exposure of a cell to deleterious influences such as insufficient nourishment, or to a nutritive fluid containing poisonous elements, causes alterations in the constitution of the cell. The cell is sick and may die, but it dies slowly, undergoing in the meanwhile a gradual conversion into compounds foreign, whether as to quantity, or quality, or both—to its constitution in health. This retrogressive metamorphosis of the cell-tissue itself is the

commonly-accepted criterion of "degeneration" in the narrow sense which opposes it to "necrosis" on the one hand and to "infiltration" on the other; infiltration implying the deposit within a cell of some substance brought from without. The distinction is worth observing for purposes of description, but it cannot always be maintained, especially in the case of fatty changes.

**Degenerations.**—(*a*) Cloudy swelling; (*b*) Fatty degeneration; (*c*) Albuminoid degeneration.

(*a*) **Cloudy swelling.**—The organs of those who die rapidly after poisoning by antimony, phosphorus, and certain other poisons, or more slowly, after febrile diseases like typhoid fever or scarlet fever, commonly exhibit a generalised swelling and lack of lustre. Microscopic examination shows that the cells of the affected tissue are enlarged: their outline is obscured, and their protoplasm occupied by granules proved to be of an albuminous nature. This change is called "Cloudy swelling." It is most marked in the liver, kidneys, heart and voluntary muscles, and is often the first step towards fatty degeneration, notably when due to phosphorus-poisoning.

(*b*) **Fatty degeneration.**—Fatty degeneration was held by Virchow to be the result of conversion of cell-proteids into fat. It is now believed by many that the intra-cellular globules of fat, which are accepted as microscopic evidence of fatty degeneration, are due to the fusion, into visible droplets, of the fat which is a normal, though not visible, constituent of a healthy cell. The point is somewhat academic. It is sufficient for our purposes to observe that the appearance of fat globules within cells which do not normally contain visible fat is evidence of fatty degeneration.

Fatty degeneration may be a physiological process, as it is in the case of the lactating breast. For the fat of milk is supplied by degeneration of the central cells of the mammary acinus. On the other hand fatty degeneration plays an immense part in pathological processes. It occurs at some stage in nearly all lesions of any duration, and to some extent in acute inflammations. It is seen in the dead leucocytes of pus, and in the viscera of those who have died from wasting-diseases like diabetes mellitus, pulmonary tuberculosis and cancer. It attends continued suppuration, chronic heart-

disease, severe anaemias, and many kinds of poisoning. Indeed, there is hardly a chronic disease which proves fatal without entailing fatty degeneration in some organ.

Fatty degeneration gives to the tissue affected a yellowish pallor. In an advanced case an oily exudation may appear upon section.

Microscopic examination shows that the cells of the tissue are enlarged and their nuclei displaced by globules of fat. These globules are stained black by Osmic acid and yellow by Sudan III.

(*c*) **Albuminoid degenerations.**—(1) Mucoid degeneration; (2) Hyaline degeneration; (3) Colloid degeneration; (4) Amyloid degeneration.

In a variety of diseased conditions cell-protoplasm undergoes conversion into a homogeneous albuminoid material. The terminology of these degenerations is vague and confused, partly because the chemical constitution of the new bodies is varied in different cases and situations, partly because the descriptive terms in use denote characteristics which are sometimes chemical (*e.g.* "mucoid" and "amyloid"), sometimes physical (*e.g.* "hyaline" and "colloid").

(1) **Mucoid degeneration.**—Mucin is a compound proteid containing a carbo-hydrate element, and thus when boiled with an acid produces a substance capable of reducing Fehling's solution. It is precipitated by acetic acid and has an affinity for basic dyes. But there are a number of allied substances, pseudo-mucins, which are not precipitated by acetic acid and have an affinity for acid dyes. Degenerations resulting in the production of mucinous material in either of these forms are called mucoid degenerations. They fall into two groups, according as the structures attacked are epithelia or connective-tissues.

(*a*) Epithelial mucoid degeneration occurs particularly in tumours of parts which normally produce mucin, such as cancers of the stomach and intestine, but is met with also in the breast and elsewhere. The degeneration is often widespread, and then converts the tumour into a semi-transparent mass. Under the microscope the affected tissue appears structureless and homogeneous. Cells are scanty, or absent altogether. Such as survive have a bloated appearance owing to their distension by mucoid material. It is customary to speak of cancers which have undergone this degeneration as

"colloid cancers," a fact which emphasises the laxity of phrase current in this connection.

(*b*) Connective-tissue mucoid degeneration affects a great variety of connective-tissue tumours, fibromata, lipomata, chondromata, and sarcomata. The degeneration may be so extensive that no trace of the original structure persists (Myxoma). In certain tumours, especially those of the ovary, mucoid degeneration leads to the formation of cysts.

(2) **Hyaline degeneration.**—There is no one substance to which the term "hyalin" can be applied. "Hyaline degeneration" means the conversion of tissues into a pathological, homogeneous, clear substance with an affinity for acid dyes like eosin and fuchsin. This substance may appear in epithelial cells (epithelial hyalin) or in connective-tissues, (connective-tissue hyalin). Hyaline casts of renal tubules are a good example of epithelial hyalin. The conversion of connective-tissues into hyalin occurs in old scars, in the degenerate glomeruli of chronic interstitial nephritis, and in the walls of arteries. "Zenker's hyaline degeneration of muscle-fibres" is an analogous lesion common after death from typhoid fever.

(3) **Colloid degeneration.**—Properly speaking "Colloid degeneration" means the conversion of tissues into a substance resembling the normal iodine-containing secretion of the thyroid gland. In this limited sense colloid degeneration is rare except in tumours of the thyroid and, perhaps, pituitary glands. But in the commoner vague acceptation it embraces mucoid degeneration.

(4) **Amyloid degeneration** (lardaceous or waxy degeneration).—This is a well-differentiated form, characterised by the appearance in the tissues of a colourless, semi-transparent substance. This "amyloid material," when treated with a watery solution of iodine containing iodide of potash, becomes of a mahogany-red colour, changing as a rule, but not always, to blue upon the addition of sulphuric acid. Amyloid material is believed to be due to the interaction of damaged cell-substance and chemically-altered lymph.

This degeneration is generally caused by prolonged suppurative lesions, such as tuberculous caries of the spine, and is now a relative rarity owing to improved surgical treatment of the diseases most often responsible for it. At the present

time its commonest causes are tuberculous disease of bone and chronic pulmonary tuberculosis. But it occurs at times apart from suppurative lesions, in the course of syphilis, malaria, cancer, and leukaemia. The following is a list, in a descending scale of liability, of the organs most often attacked by it. Spleen, kidney, liver, large blood-vessels, mucous membrane of the intestines, lymphatic glands, supra-renal bodies, and heart. The lungs and genito-urinary system seldom suffer, and the central nervous system never.

The organ affected is enlarged and firm, and section of it leaves a sharp edge. The amyloid material has a glistening, translucent appearance.

**Microscopic Examination.**—Methyl aniline violet is a selective stain for amyloid material and colours it a rosy pink, while normal tissues are stained blue. The earliest changes are seen in the intima and media of small blood-vessels. The fixed connective-tissues suffer most; epithelial structures little if at all, although they may be the seat of other degenerative changes.

**4. Infiltrations.**—By "infiltration" is meant the deposit within the tissues of some foreign substance derived from without.

(*a*) Fatty infiltration.
(*b*) Calcareous infiltration.
(*c*) Pigmentary infiltration.

(*a*) **Fatty infiltration.**—Fatty infiltration is a physiological process in various situations, such as the subcutaneous tissues and the mammary glands of females. But in obese persons deposits of fat appear in situations where fat is not normally found, for instance, between the fibres of the heart's muscle, and between the liver-cells. Such deposits are pathological, and often, though not by any means always, coexist with fatty degeneration.

(*b*) **Calcareous infiltration.**—Calcareous infiltration is due to the precipitation within the tissues of lime-salts brought by the blood. It most often affects structures either dead (like pus, or caseous accumulations) or the seat of some form of degeneration. The chief salt deposited is calcium phosphate.

Calcareous deposits are white and chalky in appearance. In consistency they vary from a mortar-like material to concretions of almost stony hardness. This variation depends to a large extent upon the age of the deposit. Thus infiltrated

collections of pus or caseous matter pass through the softer to the harder stage as time goes on, the fluid constituents of the dead material undergoing absorption. But in some situations, notably the arterial walls, the deposits seem to be more or less hard throughout. Calcareous deposits are physiologically present in the pineal gland, and are commonly met with in a great variety of tumours, generally with, but sometimes without, gross evidences of degeneration or necrosis. The latter class is small: its chief representative being a variety of fibrous tumour of the dura mater called "Psammoma." The former class is large. Fibromyomata of the uterus, tumours of the thyroid gland, and large malignant tumours generally are particularly prone to extensive calcareous infiltration. Obsolete parasitic cysts such as hydatid cysts, and thrombi, particularly in veins, often undergo this kind of petrification.

(*c*) **Pigmentary infiltration**.—This term is reserved to express the deposit of pigmented particles within the tissues. Thus it would not be applied to the diffuse staining of the tissues produced by jaundice. These particles may be derived (*A*) from pigments already present in the body (intrinsic), or (*B*) from without (extrinsic).

*A*. The **intrinsic pigments** responsible for pigmentary infiltration are: (1) Haematogenous. (2) Non-haematogenous.

(1) Haematogenous pigmentary infiltration is due to altered blood-pigments. It is met with therefore most often in the course of diseases which involve an extensive destruction of red blood corpuscles, such as pernicious anaemia and malaria, but occurs also in other diseases, and in a peculiar form of degeneration of cardiac muscle. (Brown atrophy of the heart.) The resulting pigments are of two kinds: (*a*) Haemosiderin, containing iron. (*b*) Haematoidin, iron-free. Haemosiderin, when treated with ferro-cyanide of potash acidulated with hydrochloric acid, gives the Prussian-blue reaction. It occurs in the form of granules, golden-brown in colour, both within the cells and in the intercellular substance. Haematoidin occurs particularly as a result of blood-extravasation. In areas of haemorrhage granules and rhombic plates of haematoidin may be seen with the microscope.

(2) Non-haematogenous pigmentary infiltration. The most important example of this variety is melanotic sarcoma. The

deposits of melanin, though black in the mass, appear as brownish granules under the microscope. Melanin is an iron-free pigment and is not derived from the blood.

*B.* **Extrinsic pigmentary infiltration.**—The pigments in this case reach the tissues by way of the respiratory and alimentary systems. The commonest is carbon. The bronchial lymphatic glands and lungs of adults are invariably blackened by deposits of carbon-granules. This infiltration is seen at its maximum in the case of those exposed to an atmosphere laden with carbon dust, as are coal-miners, and is then termed Anthracosis. The lungs of workers in stone and iron are liable to analogous infiltrations by mineral particles. (Silicosis, Siderosis.) The blue line along the edges of the gums which is characteristic of lead poisoning is due to an infiltration by sulphide of lead.

## *III.*

## INFECTIVE GRANULOMATA.

A granuloma is a tumour composed of granulation-tissue; that is, an inflammatory tumour. An infective granuloma, in the strict sense, is such a tumour produced as a result of any bacterial irritation: but in practice the term is reserved for the lesions of a limited group of organisms which have the following points in common. (1) The lesions they respectively produce bear a broad general resemblance to one another from the histological point of view. (2) The inflammation they excite is essentially chronic, relatively to other bacterial lesions. (3) The products of this inflammation show a pronounced tendency to degeneration.[1]

Of the diseases characterised by infective granulomata two, namely Tuberculosis and Syphilis, are so common and important that their lesions will be described at some length. Others, such as Glanders and Actinomycosis, are relatively

[1] According to current usage the term "Infective granuloma" expresses not only the lesion but the disease causing it. Thus it is common to hear Tuberculosis and Syphilis described as infective granulomata.

infrequent, and will be dealt with briefly in connection with the organs most subject to attack by them. A third group is mainly tropical, *e.g.* Leprosy and Mycetoma (Madura disease), and is omitted from consideration.

**Tuberculosis.**—The anatomical unit of tuberculous lesions is the **Tubercle**, a small nodule of granulation-tissue developed around a tubercle bacillus or a collection of them. The earliest macroscopic stage of the inflammation is the "**Gray Tubercle**," a minute, semi-translucent, gray nodule.

Under the microscope it is seen, in a typical case, to consist of three more or less distinct zones. The central one consists of a caseous ill-stained débris. This is surrounded by a zone of cells with large nuclei and granular protoplasm, the so-called "epithelioid cells," interspersed with giant-cells. Giant-cells are cells containing many nuclei. They are not confined to tuberculous lesions, being found in a variety of chronic inflammations, but the tuberculous giant-cell has this peculiarity that in its typical form the nuclei are arranged peripherally around the hyaline protoplasm which forms the cell-body, while in other forms the nuclei are more indiscriminately placed. The third and outermost zone is formed by a dense collection of small round cells (lymphocytes). The above is the characteristic arrangement in a tubercle, but it is not always to be seen owing to secondary changes.

**Secondary changes in the Tubercle.**—(1) Caseation. (2) Fibrosis. (3) Calcification.

(1) **Caseation.**—Caseation is due to coagulation-necrosis and fatty degeneration. It occurs first in the central zone and is due in part to the directly poisonous action of the bacilli, in part perhaps to a defective blood-supply. With the enlargement of the central caseous area the whole tubercle increases in bulk and becomes of a yellowish colour (**Yellow tubercle**). In rare instances such a tubercle may remain isolated and reach a considerable size, but, since in a vast majority of cases tubercles do not occur singly, it is more usual for the extending tubercle to meet and fuse with neighbouring ones. By repeated coalescences of this kind large areas of tissues become involved. In certain situations caseous accumulations become liquefied and form tuberculous or "cold" abscesses. A tuberculous abscess differs in important respects from an acute abscess due to pyogenic organisms. It is essentially chronic. Its solid contents are a granular and caseous detritus, and a few degenerate leucocytes. Such abscesses occur particularly in connection with tuberculous disease of lymphatic glands (notably those in the neck), and bone. But an analogous

process is responsible for the excavation of the lung in pulmonary tuberculosis, though here the liquefied material is generally discharged by expectoration as it is formed, instead of accumulating to form a definite abscess. It is rare to find liquefied tuberculous deposits in the brain. In this situation they maintain a cheesy consistency and tend to assume a greenish colour.

(2) **Fibrosis.**—The spread of tuberculous inflammation in a tissue is limited in the ordinary course by the formation of fibrous tissue in and around the infected site, the extent of this formation being a measure of the resisting capacity of the individual concerned. Thus when resistance is low fibrosis is little in evidence, while caseation and spread of inflammation proceed with rapidity: in the contrary case the infected focus becomes surrounded and isolated by a firm fibrous capsule. Unless caseation has been extensive, the subsequent contraction of the newly-formed fibrous-tissue may leave no legacy of the lesion beyond a puckered scar.

(3) **Calcification** has already been described (*v.* p. 16).

**Paths of infection.**—Direct inheritance of the tubercle bacillus itself, *i.e.* congenital tuberculosis, is met with as a pathological curiosity. It is extremely rare. But there is little doubt that certain families exhibit a peculiar susceptibility to the disease. The disease itself is not hereditary, but a low degree of specific resistance towards the tubercle bacillus appears to be transmissible to offspring.

The two commonest avenues of infection are the respiratory and alimentary tracts. The relative importance of these two channels is a matter of dispute. It was held at one time that pulmonary tuberculosis was always derived from the inhalation of tubercle bacilli, but recent work tends to the conclusion that bacilli which have been ingested may traverse the intact mucous membrane of the intestine and, reaching the right heart by way of the thoracic duct, may be deposited in the lung and there set up an inflammation without infecting the paths by which they have travelled.

**Dissemination of the disease within the body.**—This may be effected by four routes. (*a*) By direct extension from a primary focus. (*b*) By lymphatic paths, whether with or without infection of the lymphatic vessels themselves. (*c*) By way of the blood-vessels. (*d*) By way of the air-tubes.

Acute general tuberculosis is due to distribution of bacilli by the blood-stream. In the lungs (the commonest situation of tuberculosis) infection spreads both by direct extension, by lymphatic paths, and sometimes by way of the air-tubes. The last event is commonly due to the rupture into a bronchus of a caseous accumulation situated in the immediate neighbourhood. The condition is not infrequent, and the lymphatic glands at the bifurcation of the trachea are the usual source of the infecting material.

**Syphilis.**—The lesions of syphilis which exhibit best the characteristic features of infective granulomata are the Hunterian chancre of the primary stage, and the **gumma** of the tertiary stage. It is with the latter only that we are here concerned.

Anatomically a gumma is a firm yellowish-white (or, in its early stages, reddish) nodule. It may be quite minute, or may reach a diameter of two or three inches. It is more or less definitely encapsuled. Even in its early stages the central portions of the nodule show evidences of caseous change, while interspersed with the necrotic material are strands of fibrous tissue. The latter is irregularly distributed, but broadly speaking, tends to shut off the diseased area, and at the same time sends prolongations into the surrounding districts. The subsequent contraction of this new fibrous tissue results in the scarring which is a prominent feature in gummata of long standing. Local deposits of amyloid material are occasionally seen in the neighbourhood.

Like tubercles, gummata may become liquefied. When this occurs in a gumma situated near a surface such as mucous membrane or skin, sloughing of the surface commonly results, and permits the discharge of the softened material. The cavity thus left is a "gummatous ulcer," the characteristic features of which are steep sides and a "punched-out" appearance.

The commonest sites of gummata are periosteum, the liver, the testicle, the brain, the spleen, the tongue, tonsil, pharynx and larynx, fasciae and subcutaneous tissues.

**Microscopic examination.**—In its early stages a gumma consists of an abundant cell-exudation which obscures the structure of the affected tissue, and is well supplied with blood-vessels. At a later stage necrosis and fibrous-tissue formation become prominent. Giant-cells occur at

the edges of the lesion, but they are scanty and seldom exhibit the regular peripheral arrangement of nuclei characteristic of tuberculous giant-cells. The smaller blood-vessels in the vicinity of a gumma are generally the seat of an obliterative endarteritis. This last is commonly received as one of the best histological criteria of a gumma as opposed to other infective granulomata.

## *IV.*

## CLASSIFICATION AND NOMENCLATURE OF TUMOURS.

Used in a clinical sense the word Tumour implies a localised swelling of any kind, but in pathological phraseology it is restricted to express a new formation of tissue which is physiologically independent, and obeys its own laws of growth and decay rather than those of the structures amidst which it lies.

Tumours have been subjected to a great variety of classifications, but the two most commonly received are: (1) Clinical; (2) Morphological.

1. **Clinical classification.**—(*a*) Innocent; (*b*) Malignant.

(*a*) **Innocent tumours.**—Innocent tumours possess as their chief clinical characteristic the quality of being circumscribed and well delimited, often by a definite fibrous capsule. They evince no tendency to infiltrate the neighbouring tissues, but merely displace them. Such disorganisation as they excite is due to the accident of their situation and their bulk, not to any actively-destructive capacity inherent in them. They may be multiple, but in this event each individual tumour is, in origin, independent of the others.

(*b*) **Malignant tumours.**—The clinical criteria of malignancy in a tumour are: (1) Lack of circumscription; (2) Secondary growths (Metastases).

Malignant tumours are ill-defined against their surroundings. They infiltrate, destroy, and replace adjoining tissues. Moreover, detached particles of the growth are commonly swept away in the lymph or blood stream, and, being deposited in lymphatic glands or other distant organs, give rise to a

second generation of tumours. These secondary or metastatic growths breed true to the original stock, uninfluenced by the nature of the tissue in which they chance to be situated.

Although this clinical distinction is in the main a sound and serviceable one, it must not be supposed that any sharp line of demarcation separates the innocent from the malignant groups: for all gradations between the two are encountered from time to time. Nor is it necessary that both the above attributes of malignancy should be present in any given example. Thus Rodent Ulcer exhibits a local malignancy in that it infiltrates and destroys adjacent tissues, yet it does not produce metastases.

Histology affords important corroboration of the clinical innocence or malignancy of any tumour. A prominent histological characteristic of normal adult tissues is their pecialisation for different functions, while embryonic tissues are specialised less or not at all. In the case of a tumour it is found that the more nearly it reproduces the features of normal adult tissue, the more innocent it is. Conversely, malignancy is linked with, and measured by, the degree to which such specialisation is lacking. Thus a hard fibroma, one of the most innocent of tumours, reproduces exactly the characteristics of normal adult fibrous tissue, while a small round-celled sarcoma, one of the most malignant varieties, is composed of a dense collection of totally undifferentiated cells, massed together at random and without arrangement. These are extremes, but between them occur all grades of reversion to the undifferentiated embryonic type (anaplasia), the degree of this reversion measuring the malignancy of the growth.

2. **Morphological classification** (after Dr. Andrewes).—Tumours are grouped morphologically according to the type of tissue from which they have originated. There are five such groups:

(*A*) Connective tissue tumours.
(*B*) Epithelial tumours.
(*C*) Endothelial tumours.
(*D*) Nerve-tissue tumours.
(*E*) Mixed tumours, belonging to two or more of the above groups.

### A.—Connective tissue tumours.

1. Adult type. Innocent.
   - (*a*) Fibroma (Fibrous tissue).
   - (*b*) Lipoma (Adipose tissue).
   - (*c*) Chondroma (Cartilage).
   - (*d*) Osteoma (Bone).
   - (*e*) Angeioma (Vessels. Haemangeioma = Blood-vessels. Lymphangeioma = Lymphatic vessels).
   - (*f*) Myoma (Muscle).
   - (*g*) Glioma (Neuroglia).
2. With reversion to less specialised type. Malignant.
   - (*a*) Sarcoma.

### B.—Epithelial tumours.

1. Adult type. Innocent.
   - (*a*) Papilloma (Squamous epithelium).
   - (*b*) Adenoma (Columnar, cubical, and spheroidal epithelium).
2. With reversion to less specialised type. Malignant.
   - (*a*) Rodent ulcer (Locally malignant).
   - (*b*) Carcinoma. { Squamous-celled. Epithelioma. / Columnar, cubical, spheroidal-celled.

### C.—Endothelial tumours.

Endothelioma. Generally innocent, sometimes malignant.

### D.—Nerve-tissue tumours.

Neuroma (True). Extremely rare.

### E.—Mixed tumours.

(*a*) True teratoma or embryoma (representing the remains of another individual).

(*b*) Sequestration-dermoid (representing a misplaced part of the individual in whom it exists).

A tumour may exhibit the features of two or more groups, and is then denoted by a compound name, *e.g.* Fibro-lipoma, Osteo-chondroma. Connective-tissue tumours which, though partially specialised, yet fail to reach the standard of normal

adult tissues, form the sub-groups of Sarcoma, *e.g.* Osteo-chondro-sarcoma, Glio-sarcoma. The true sarcomata are differentiated according to the shape of the cells composing them, into four classes, Round-celled, Spindle celled, Giant-celled, Mixed-celled.

Endotheliomata form a group both histologically and clinically ill-defined. Histologically they partake of the characters of carcinoma and of sarcoma, yet on the whole are clinically innocent.

Many tumours are liable to undergo mucoid degeneration. When this degeneration is extensive enough to destroy altogether the original characters of a growth, the resulting gelatinous mass is termed a Myxoma. When the change is less pronounced its existence is indicated by the prefix "Myxo-," *e.g.* Myxo-lipoma, Myxo-sarcoma. This is the general rule, but Carcinomata which have undergone mucoid degeneration are conventionally termed "Colloid carcinomata."

## *V.*

## LIFE HISTORY OF THE COMMONER PARASITIC WORMS.

### A. Cestodes or Tapeworms.

(1) Taenia saginata or mediocanellata.
(2) Taenia solium.
(3) Taenia bothriocephalus latus.
(4) Taenia echinococcus.

### B. Nematodes or Round Worms.

(1) Ascaris lumbricoides.
(2) Oxyuris vermicularis.
(3) Ankylostoma duodenale.
(4) Trichocephalus dispar.
(5) Trichina (trichinella) spiralis.
(6) Filaridae { *a.* Filaria sanguinis hominis. *b.* Filaria medinensis.

### C. Trematodes or Flukes.

Bilharzia haematobia (Schistosomum haematobium).

**A. Cestodes or Tapeworms.**—Tapeworms are long flat worms consisting of a head and neck, together called the **Scolex**, attached to a series of segments or proglottides. The head is minute and surmounts the tapering anterior extremity of the worm. The segments nearest to the head are also minute, but the succeeding ones exhibit a progressive enlargement, reaching their maximum towards the middle of worm's length and thence becoming gradually smaller. When fully developed each proglottis may be considered a separate hermaphrodite individual, for each contains both male and female organs of generation. When sexually mature the uterus of the proglottis becomes filled with eggs which develop into embryos, each armed with six hooklets at one of its poles. At this stage the ripe proglottis is detached and discharged with the faeces of the host. The life-cycle of the embryo includes an intermediate, resting phase which is commonly, but not invariably, passed in the tissues of an animal differing in species from that which harbours the adult worm. This animal is called the "*intermediate host*," the species of animal being more or less strictly defined for each variety of worm. The host of the mature worm is called the *definitive host*. If the ripe proglottis, or the embryos which have escaped from it, be ingested by an appropriate animal, the embryo bores its way through the intestine and finally becomes arrested in some viscus or structure suitable to its further development. The hooklets now disappear, and from the opposite pole there arises a scolex precisely similar to that of the original worm, while the body of the embryo is converted into a watery bladder within which the scolex lies invaginated. The embryo in this resting stage is called a "cysticercus." The details of the cystic stage vary with different species of cestode, and some varieties form no cyst, the scolex simply increasing in size, but remaining sexually immature. If the definitive host devours flesh containing these larval forms still alive, the cyst-capsule is destroyed by digestion, and the scolex, affixing itself to the mucous membrane of the intestine, rapidly becomes developed into a mature worm.

There are three tapeworms commonly found mature in the intestine of man, Taenia Saginata, Taenia Solium, and Taenia Bothriocephalus latus. A fourth, Taenia Echinococcus, is a parasite of man in its cystic stage only (hydatid cyst).

(1) **Taenia Saginata** (or Mediocanellata, or Inermis), the beef tapeworm, so called because its intermediate host is the ox, is the only common tapeworm of man in these islands. It varies from four to eight yards in length and is composed of twelve or thirteen hundred proglottides. The uterus of each segment exhibits 20–30 branchings. The head is pear shaped and carries four pigmented suckers. The "cysticercus bovis" is the cystic larva of this variety and the meat infected with these parasites is known as "measly beef." Beef thus affected exhibits between its fibres oblong watery cysts containing the scolices of the worm, and if eaten by man, uncooked, gives rise to the mature intestinal parasite. The cysticerci in very rare instances have been found in man.

Series of Invertebrata. Top gallery. 1472 1473*a*

(2) **Taenia solium**, the pork tapeworm. The intermediate host in this case is the pig, while the mature worm is a parasite of man exclusively. Taenia Solium is rare in this country, but common upon the continent, particularly in parts of Germany. It is shorter than Taenia Saginata, measuring from two to four yards, and is composed of some eight hundred proglottides. The uterus exhibits 8–12 branchings. The head is spherical and terminates in a circular "*rostellum*" or beak, round which are arranged a double circle of *hooklets* numbering from 20–30. The presence of these structures, and the fewer uterine branchings in the segments, afford an easy distinction between Taenia Solium and Taenia Saginata. Behind the rostellum are four prominent suckers. The cystic stage is passed for the most part in the muscles of the pig, producing "measly pork." Each cyst is called a "cysticercus cellulosae." It measures about a third of an inch in length and contains an exact replica of the scolex of the mature worm. Whereas the cystic stage of Taenia Saginata is very rarely encountered in the human body, cysticercus cellulosae is a common cause of human disease in countries where the worm abounds. Occasionally the cysts are widely distributed throughout the body, and may number many hundreds, but more often they affect the cortex of the brain or the eye.

Series of Invertebrata. Top gallery. 1474

Cysticercus cellulosae in muscle. Ground floor. 1175

(3) **Taenia bothriocephalus latus**, the fish tape-worm. This worm is never met with in this country but is common in certain parts of the world, particularly Russia. The pike and the trout are its usual intermediate hosts. It is very long, measuring upwards of ten yards in length, and consists of

Series of Invertebrata. Top gallery. 1479 1479*A*

three to four thousand segments, which are broader than they are long, and contain a rosette-shaped uterus. The head is flattened and carries *two laterally-placed suctorial grooves, but no rostellum, nor hooklets.* The embryos are not destroyed by salting, pickling, or smoking, and thus remain long capable of infecting those who eat of the fish which harbour them.

(4) **Taenia echinococcus** in its adult phase is an intestinal parasite of the Canidae. Its cystic phase may be passed in man, and gives rise to "hydatid disease."

The worm is a small one measuring $\frac{1}{6}$ of an inch, and is composed of not more than four segments of which the terminal one is the largest and contains fully developed sexual organs. The head carries four suckers and a *rostellum surrounded by two rows of hooklets.* The terminal segment when mature discharges its ova into the intestine, whence they are distributed abroad with the faeces. Access to the human subject is obtained by way of contaminated uncooked foods such as lettuce and water-cress. Within the human stomach the chitinous envelope of the ovum is dissolved, setting free by its solution an oval embryo with six spines at one of its poles. These spines are directed backward and serve as a barb making the path of the embryo one of constant progression. By the aid of this mechanism it penetrates the coats of the bowel and may then be arrested in any part of the body; but the most frequent site of arrest is the liver. The cystic stage of development now commences. The hooklets disappear and the embryo enlarges into a cyst. The wall of this cyst comprises two layers, an outer chitinous one, the ectocyst, and an inner, soft, granular one, the endocyst. The primary cyst may be sterile, or may undergo reproductive changes. In the latter case there appear minute opaque buddings of the endocyst, named "*brood-capsules.*" These appear to be cystic invaginations of the primary cyst from without, for their outer layer is formed by the endocyst and the inner layer by the ectocyst, the condition seen in the original formation being thus reversed. Upon the outer granular wall of the brood-capsules develop the "scolices" or immature head segments of future worms. These may become detached and float free in the cavity of the original cyst, or may be invaginated in bulk within the brood-capsule and subsequently liberated by its rupture. A scolex is

1st gallery. 2235*B*

about 3 mm. long and consists of two segments. By one of these it is attached to the wall of the brood-capsule; the other is free, and is armed with four suckers and a ring of hooklets, each shaped after the fashion of a shark's tooth. Should a scolex gain entrance to the intestine of one of the Canidae it developes into the adult worm.

Hydatid cysts may be single or multiple. The parent cysts, when proliferative, may contain an indefinite number of daughter and grand-daughter cysts. These successive reproductions are accomplished either by invaginations of the endocyst which become vesicular, or by a cystic development of the scolices and brood capsules. The ectocyst is tough and elastic, and presents under the microscope a laminated appearance. The endocyst is of a pearl-white colour and gelatinous in consistency. Hydatid fluid from such cysts is clear and of a specific gravity ranging from 1002 to 1010. During the lifetime of the parasite the fluid contains sodium chloride in abundance, but no albumin. After death of the cyst its fluid becomes albuminous and generally contains detached hooklets, scolices, and brood capsules. The tissues in the immediate neighbourhood of the cyst undergo a slow fibrosis by which means an adventitious capsule appears around the ectocyst.

1st gallery. 2232*A*

1st gallery. 2233*A*

**Fate of the cysts.**—It is probable that hydatid cysts may live for years unaltered. They may rupture during the lifetime of the parasite, producing secondary cysts along the path of the escaping fluid, unless the primary cyst be sterile, in which case there may be no resultant lesion. Frequently they undergo spontaneous death from causes little understood. In this case one of two events ensues.

(1) **Absorption.**—The contained fluid of both the parent and daughter cysts becomes albuminous and subsequently inspissated by the absorption of its fluid ingredients, forming ultimately a putty-like mass. This shrinkage is accompanied by collapse of the walls of all the cysts concerned. They become folded upon themselves and their diminishing contents, and give to the cut surface of the dead cysts an appearance of gelatinous stratification. In course of time calcareous salts are deposited in the adventitious capsule and in the dead remains of the parasite.

1st gallery. 2230*B* 2230*C*

1st gallery. 2233*B*

(2) **Suppuration.**—Bacterial infection may convert the cyst-

fluid into pus. The abscess thus formed, like every abscess, tends to "point" along the line of least resistance, and may discharge its contents into any neighbouring cavity or tube.

1st gallery. 2235*B*

**B. Nematodes or Round-worms.**—The round-worms are slender cylindrical animals tapering towards both ends, at one of which is situated the mouth, at the other the anus. Most of the nematodes are not hermaphrodite, and the males are smaller than the females.

(1) **Ascaris lumbricoides,** the common round-worm.—This worm is very frequently met. It is of a brownish or yellowish colour. The female may reach a length of fifteen inches, the male being much smaller, and measuring from six to nine inches. The worm may occur singly or in large numbers, and inhabits the small intestine for choice; but it may wander upwards or downwards, occasionally entering the stomach, or oesophagus, or even the glottis. The uterine tubes contain millions of ova. These are stained brown by the bile of the intestinal contents, and are capable of developing into the mature worm *in situ*, without the intervention of an intermediate host.

Series of Invertebrata. Top gallery. 1488 1488*A*

(2) **Oxyuris vermicularis,** the thread-worm.—This is the commonest of all the intestinal worms, and affects children more often than adults. The female measures one-third of an inch in length. It has a tapering, pointed tail and resembles a piece of fine thread. The male measures one-sixth of an inch in length and has a blunt hinder end. The female thread-worms inhabit the caecum and large intestine, often in enormous numbers. They are prone to nocturnal migrations in the course of which they may escape from the anus and cause much irritation in the neighbouring parts.

Series of Invertebrata. Top gallery. 1488*B*

**Symptoms.**—Both the Ascaris lumbricoides and Oxyuris vermicularis may inhabit the intestine without producing symptoms. But occasionally, especially among children, they give rise to obscure nervous manifestations. This is particularly the case with Ascaris lumbricoides, which appears capable of causing convulsions, squints, nystagmus and delirium. The effects of Oxyuris vermicularis are limited to local irritation about the rectum and anus.

(3) **Ankylostoma duodenale,** the tunnel worm.—This worm is the cause of ankylostomiasis (syn.: Egyptian anaemia,

Miner's anaemia), a disease endemic in many parts of the tropics and common among miners in certain parts of Europe. The male worm measures about one-fifth of an inch in length, the female about three fifths. The worm tapers towards its anterior extremity, which ends in a prominent circular mouth-cavity furnished with four claw-like hooks at its ventral edges, two on either side of the middle line. From the dorsal aspect or roof of the mouth-cavity there project two conical teeth, one on either side of the median line. The tail of the male worm is expanded into a three-lobed, umbrella-like bursa with eleven ribs. Into the cavity of this bursa opens the cloaca, from which project delicate spicules. The female is pointed at the hinder end, which terminates in a single spicule. The female produces an enormous number of oval eggs, which are discharged with the faeces of the host and then develop into embryos capable of living for weeks or months in muddy water or damp earth. When these larvae come into contact with the human skin they bore through the epidermis giving rise to irritation at the site of entrance, the "coolie itch" of tropical countries, the "bunches" of Cornish mines. From the skin they pass by way of the venous system to the lungs: thence along the bronchi to the trachea, which they ascend; turning back at the glottis they traverse the oesophagus to the stomach and finally reach the duodenum and jejunum, where they become mature. The habitat of the adult form is the jejunum and, less often, the duodenum. Here it affixes itself to the mucous membrane and sucks blood from it. When the worm is present in large numbers the loss of blood is extreme enough to give rise to serious anaemia, known as "Tunnel," "Plantation," "Miners'" or "Egyptian" anaemia, according to the occupation or country of the persons attacked.

1st gallery. 1956*A*

(4) **Trichocephalus dispar**, the whip-worm.—This worm measures from one-and a-half to two inches in length, the female being but slightly larger than the male. The anterior three-fifths of the body are very slender, the posterior two-fifths much thicker, while the whole has a distant resemblance to the stock and lash of a miniature whip. The eggs are oval and covered by a thick brown ectoderm. They are distinguished from all other intestinal ova by two transparent blunt projections, one at either pole of the oval.

This worm is not very important pathologically. It inhabits the caecum of man and a few related species. No intermediate host is required, but the eggs must undergo a long sojourn in water before the embryo is developed and ready to complete its cycle upon transference to the human stomach.

(5) **Trichina Spiralis.**—This worm in its mature form occurs as an intestinal parasite of man, but its pathological importance depends upon its capacity to pass its larval phase embedded in human voluntary muscle.

The adult impregnated female measures about one-eighth of an inch in length. It is believed that the gravid worm perforates the wall of the intestine and, reaching a lymph-space, there discharges her embryos. These finally come to rest in the voluntary muscles, where they become encysted. Examined with a lens each cyst is found to be oval, with a prolongation at either pole, and contains the coiled embryo worm. The parasite can live for a long time in this larval state, but finally dies and becomes infiltrated with lime-salts. These dead and calcified cysts appear as a profusion of minute opaque white dots among the fibres of infected muscle.

Ground floor.
1176*A*
1176*B*

Man becomes infected by eating the insufficiently cooked flesh of infected pigs. But since the pig is only carnivorous under conditions of domestication, it is believed that the pig is not the natural host of the parasite. Rats are very subject to infection by Trichina spiralis and are held to be its natural host. But the dissemination of the infection among pigs is probably effected by feeding them upon tainted offal. The encysted Trichina is very resistant to heat, being able to withstand temperatures up to 80° C. The centre of a large ham does not reach 85° C. until it has been boiled for ten hours. The destruction of the parasite in pork therefore involves more prolonged cooking than is commonly thought necessary.

The disease produced by the larvae is called Trichinosis. It is marked by diarrhoea and fever, accompanied by severe muscular pain, and has been mistaken for typhoid fever.

(6) **Filaridae.**—These are long slender worms of uniform diameter. Two tropical representatives of the group are important: (*a*) Filaria sanguinis hominis; (*b*) Filaria medinensis.

(*a*) **Filaria sanguinis hominis.**—This name is applied to the embryos of certain Filariae, the embryos being found in the blood, while the adults lie in the tissues or lymphatics. There are several varieties of Filaria sanguinis hominis, but the most important is Filaria Nocturna, the embryo of Filaria Bancrofti, the cause of Elephantiasis. The name Filaria Nocturna has been given to the larval form because it appears in the blood-stream only at night. The mature worm lives in the lymphatics of the trunk and extremities, the female reaching a length of three inches. The embryos traverse the lymph-channels and reach the blood-stream by way of the thoracic duct. They measure 0·3 mm. and are actively motile. Although they may be present in enormous numbers the embryos produce no symptoms, but the adult worms, by blocking lymph-channels, give rise to immense enlargements of subcutaneous tissues (Elephantiasis) and to chylous effusions. 1st gallery. 2695*B*

Several varieties of mosquito play the part of intermediate host to Filariae, imbibing the embryo with the blood they suck. Leaving the stomach of the mosquito the embryo comes to rest in the thoracic muscles of the insect and here undergoes development. Finally it reaches the mouth-parts and enters man when the mosquito bites.

(*b*) **Filaria medinensis**, the Guinea Worm.—This is common in tropical countries. The female only is well known. It reaches a length of from three to six feet and lives in the adult state in the subcutaneous connective-tissues of man. The uterus runs from head to tail and is packed with free embryos. When the worm approaches maturity she passes slowly through the tissues towards the foot. In this neighbourhood the head drills a hole through the dermis leaving the epidermis intact. Presently an epidermal bulla, the result of irritation, is formed over the site of this hole. Upon the rupture of the bulla the embryo worms are gradually discharged. The embryos swim actively in water, and pass the next phase of their existence within the bodies of certain fresh-water crustaceans (Cyclops). The infection reaches man again through the drinking of water containing these infected organisms. Series of Invertebrata. Top gallery. 1493 1493*C*

**C. Trematodes or flukes.**—The trematodes are usually short, flat, and leaf-shaped, and are for the most part hermaphrodite. But the only one to be described here, Bilharzia Haematobia, is not hermaphrodite. Series of Invertebrata. Top gallery. 1469 1470

**Bilharzia haematobia** (Schistosomum haematobium).

The male is rather less than half-an-inch long. Its ventral edges are folded inwards to form a "gynaecophoric canal," within which lies the female. The latter is thinner and longer than the male, measuring about four-fifths of an inch. Nevertheless it can be accommodated in the gynaecophoric canal. The principal habitat of the mature worms is the portal vein, from which they pass, as maturity approaches, to reach the submucous tissues of the bladder and rectum. Here the female deposits her eggs in the veins. Each egg is elliptical, about 0·1 mm. in length, and furnished at its posterior extremity with a small pointed spine. These eggs penetrate the mucous membrane of the bladder and rectum, and are discharged with the excreta. The irritation caused by their presence in the mucous membrane of the bladder or rectum gives rise to papillomatous outgrowths, which bleed readily. The Haematuria thus excited is one of the prominent symptoms of Bilharziasis, and to it the disease owes its alternative names "Endemic" or "Egyptian" Haematuria. The eggs thus passed with the urine and faeces contain a ciliated embryo, the hatching of which is not effected in urine, but rapidly takes place in water. How the embryo enters man is not known, but it is believed to enter by way of the skin.

1st gallery. 2065*C* 2065*D* 2393*B*

Bilharziasis is endemic in many parts of Africa. It is characterised by haematuria, cystitis, and pelvic irritation, due to the presence of numberless eggs within the tissues of the pelvis.

# CHAPTER II.

## DISEASES OF BONE.

1. Syphilis. 2. Tuberculosis. 3. Rickets. 4. Infantile Scurvy. 5. Osteitis Deformans. 6. Mollities Ossium. 7. Acromegaly. 8. Hypertrophic (Pulmonary) Osteoarthropathy. 9. Achondroplasia. 10. Cretinism. 11. Hydrocephalus. 12. Pathological Changes in the Bone-marrow.

### 1. SYPHILIS.

The bone lesions of acquired syphilis are dealt with at length in works devoted to surgical pathology, but there is one lesion of congenital syphilis which demands notice here.

**Epiphysitis.**—Acute epiphysitis is a not uncommon manifestation of inherited syphilis during the first six months of life. The epiphyses of the humerus and radius suffer most frequently, those of the femur and tibia occasionally, and other bones seldom.

The inflammation is generally confined to one limb, but occasionally is multiple, and, by reason of the softening which attends it, renders the affected epiphysis liable to become detached by slight degrees of violence. Clinically the disease gives rise to an apparent paralysis of the affected limbs which are held motionless on account of the pain attending movement.

**Parrot's Nodes and Craniotabes.**—It is convenient to consider here two lesions of the cranial vault commonly, but

on insufficient evidence, considered peculiar to congenital syphilis: (*A*) Parrot's nodes; (*B*) Craniotabes.

(*A*) **Parrot's nodes** (bossing of the skull: natiform skull).—This is a condition in which exaggerated bosses appear upon the normal eminences of the cranial vault, particularly those
350*A* of the frontal and parietal bones. These bosses are formed by deposits of periosteal new bone. They are undoubtedly met with in subjects of congenital syphilis, but there is equally no doubt that they occur in the absence of all evidence, actual or presumptive, of a syphilitic taint. The balance of authority inclines to regard them as of rachitic rather than syphilitic origin.

(*B*) **Craniotabes.**—The significance of this term is ill-defined. In general it implies the existence of areas of defective ossification in the cranial vault. Sometimes the whole vault is pitted by rounded indentations over which the bone is reduced
268*A* to a thin shell having the appearance of parchment. This
268*B* variety is not by any means peculiarly syphilitic, but occurs in infants the subjects of protrusions of the brain or its membranes (cranial encephalocele or meningocele). In a second variety the bones of the frontal and parietal eminences are unduly thin. This form is to be regarded as a manifesta-
268 tion of rickets. Finally, hydrocephalus occurring before or shortly after birth produces soft areas in the skull owing to wide separation of the cranial bones under the influence of the contained accumulation of cerebro-spinal fluid.

## 2. TUBERCULOSIS.

Most tuberculous lesions of bone are of surgical rather than medical interest. But *tuberculous disease of the spine* has important medical bearings. The dorsal vertebrae are those most often attacked, but no part of the spine is exempt. The stress of the disease falls upon the bodies of the vertebrae, which become caseous and are destroyed by ulceration, the arches remaining intact. With the gradual disappearance of the affected bodies, those of the healthy vertebrae above and below the site of damage come into contact, while the arches of the diseased bones form an angular projection on the back. The condition is known as "*angular curvature*" and is characteristic of tuberculous disease of the spine. The medical importance of the

disease lies in the liability of the spinal cord to be damaged as it passes through the deformed zone. This damage is generally of the nature of compression, exerted partly by the deformed bony canal, but more particularly by caseous masses developing on or in the spinal dura mater. The result of such compression is a partial or complete paralysis of the lower extremities. The relation between paralysis of the lower extremities and caries of the vertebrae was first noted by Percival Pott. 1097 (Pott's original specimen.) 1100

## 3. RICKETS.

Rickets is a chronic constitutional disease of which bone-lesions form a prominent manifestation. Its precise cause is unknown, but errors of diet, especially a *deficiency of fat with or without an excess of unaltered starch*, play an important part in its causation.

The disease begins usually in the second half of the first year of life, is common throughout the two succeeding years, is occasionally seen in older children between six and twelve years of age, and, more rarely still, in young adults (late rickets).

Changes in the growing bones are constant and characteristic features of rickets. In order to appreciate these changes it must be remembered that the increase in length of long bones is chiefly secured by growth and subsequent ossification of the layer of cartilage situated between the epiphysis and the shaft, while enlargement of the marrow-cavity is effected by absorption of the innermost layers of the shaft. Increase in the thickness of bones is due to deposition of bone by the inner layers of the periosteum. Rickets disorders all these three processes. The growth of epiphyseal cartilage is exaggerated while its ossification is defective and uneven. In this way the epiphyseal extremities of bones become enlarged while the epiphyseal line, as seen in section, instead of being sharply defined is obscured by the irregular progress of ossification. The bone laid down by the periosteum is imperfectly ossified, while the absorption of bone within the marrow-cavity is more rapid than usual. The result of these anomalies is a generalised enlargement of the epiphyses of long bones and undue softness of the whole bony skeleton. 270*A* 270*B*

The chief effects of rickets upon the skeleton may be tabulated as follows:

(*A*) The forehead is prominent: craniotabes may be present, and the fontanelles are late in closing.

(*B*) The epiphyseal extremities of the long bones are enlarged, and the shafts are liable to become bowed.

(*C*) The line of junction between the ribs and their cartilages is marked by a heaping-up of cartilage, forming the series
271 of knobs known as the "rickety rosary."

(*D*) Owing to the unnatural softness of the ribs they tend to yield to the pressure of the atmosphere during inspiration. This yielding produces a variety of deformities of the chest, the following being the commonest:

Series of casts. 13*B* Drawing. 25*C* (*a*) Lateral flattening of the chest, with undue prominence of the sternum, the "*pigeon-breast*." This depends upon diminution in the normal curvature of the ribs beyond their angles, while the straighter they become, the more is the sternum protruded.

(*b*) A groove, more or less transversely disposed, running across the anterior aspect of the chest just above the line of attachment of the diaphragm to the ribs (*Harrison's sulcus*). This groove may be prolonged upwards on one or both sides of the sternum along the line of articulation between the ribs and their cartilages.

(*c*) Lateral curvature of the spine due to loss of strength in
272 its ligaments and muscles. Such lateral curvature produces
(Case B.) compensatory deformities of the ribs.

## 4. INFANTILE SCURVY.

This disease depends upon the absence from the diet of certain unknown elements, vital to health, which are present in uncooked food-stuffs but are destroyed by the application of high temperatures. Thus the boiling of milk deprives it of its antiscorbutic powers. The disease is met with in infants towards the end of the first year of life, and is commoner among the well-to-do than among the poor, since infants of the better class are more often confined to a strict dietary of boiled milk and sterilised milk-foods.

The chief bone lesions are *sub-periosteal haemorrhages*, sometimes of large extent, situated generally beneath the periosteum

of the femora and tibiae towards their lower extremities. Here the haemorrhages form large rounded tumours accompanied by an extreme degree of tenderness, and liability of the bones to fracture. Haemorrhages into the gums are common ("*spongy gums*"), the affected areas becoming swollen and plum-coloured. But the typical appearance of the gums is only seen in the neighbourhood of erupted teeth, bare areas of gum not sharing in the lesion.

365*A*
Drawing. 26

## 5. OSTEITIS DEFORMANS.

This is a form of chronic osteitis and periostitis affecting elderly people and first described by Sir James Paget. Its cause is unknown. The bones are simultaneously thickened and softened, with the result that though the skeleton as a whole becomes abnormally massive, the diseased bones undergo a variety of deformities owing to their loss of strength. The forehead becomes prominent and the ribs tend to fall in: the femora are bowed outwards, the tibiae forwards and outwards, the radius and ulna backwards. The clavicles are enormously thickened, and their natural curves exaggerated. The spinal column becomes curved anteroposteriorly, and the head and shoulders fall forwards, diminishing the patient's height by some inches.

72*B* (Case E.) 72*K* 72*F* 74*F* (Case E.)
Drawings. 16*D* 16*E* 72$E_1$

The course of the disease is slow. In a large proportion of cases one or other of the affected bones becomes the seat of **sarcoma.**

## 6. MOLLITIES OSSIUM (OSTEOMALAKIA).

This is a disease of unknown origin. It is characterised by destruction and decalcification of bones, particularly the long bones and bones of the pelvis. In consequence the medullary cavities of the long bones are enlarged, while the whole osseous system becomes extremely yielding and fragile.

Two clinical forms of the disease are recognised. One, affecting the pelvic bones chiefly or solely, is limited to the female sex and generally associated with pregnancy. The other affects middle-aged individuals of both sexes, and involves the whole skeleton more or less. This form has definite geographical limits, the valley of the Rhine being particularly notorious for it.

291 (Case E.) 292 (Case E.) 289 293

## 7. ACROMEGALY.

This disease is marked by an increase in size of certain parts of the skeleton, particularly the face, hands, and feet, together with the soft parts belonging to them, including the ears and tongue. It affects men more often than women, commences as a rule at or about middle age, and is associated with an adenomatous overgrowth of the pituitary body.

The bones of the hands and feet are greatly increased in size and exhibit a pronounced prominence of the ridges marking the attachments of muscles. The whole head is enlarged, but the increase in size is most evident in the bones of the face, particularly the lower jaw, which comes to project beyond the upper, giving a great appearance of heaviness to the face. The dorsal spine is curved backwards, and the thorax flattened from side to side. In the neighbourhood of the sella turcica is a deep depression, made by the *enlarged pituitary body* which appears in some obscure way to be the cause of the disease.

74*H* (Case E.) Drawings. 3*B* 3*C* 2504*C*

## 8. HYPERTROPHIC OSTEO-ARTHROPATHY (HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY).

This is a chronic symmetrical disease of the bones, joints, and soft parts of the extremities. It is always a secondary manifestation. The primary disease may be one of several, but the commonest is some suppurating disorder within the thorax, particularly sacculated *bronchiectasis*, *empyema*, *or chronic pulmonary tuberculosis*. Other rarer associations are malignant disease within the thorax, congenital heart disease, syphilis, and chronic diarrhoea.

The joints most often affected are the wrists and ankles. These become intermittently swollen by effusions, and finally are permanently thickened. The articular cartilages are eroded, but there is no gross destruction, no eburnation, and no osteophyte-formation such as occurs with osteo-arthritis.

The lesion of the bones consists of a deposit of periosteal new bone upon the shafts of the radius, ulna, tibia, and fibula towards their distal extremities, upon the shafts of the metacarpal and metatarsal bones, upon the proximal phalanges, and occasionally upon the carpal and tarsal bones.

The fingers are always clubbed to a pronounced degree, while the nails are large and much curved longitudinally. This enlargement of the distal phalanges is due to overgrowth of the soft parts. The toes, especially the first and second, may be similarly affected. The precise causation of the disease is in doubt, but it is believed to depend upon the absorption of poisons from the site of the primary disease. If this can be relieved the osteo-arthropathy gradually disappears. Drawing. 4*G*

## 9. ACHONDROPLASIA (CHONDRODYSTROPHIA FOETALIS).

This is a congenital disease of unknown origin characterised by *disproportionate shortness of the arms and legs as compared with the trunk, and of the basis cranii as compared with the vault of the skull.* It has at times been known as "foetal rickets" or "sporadic cretinism," but has no real relation to either of these diseases. 288*F* 288*G* Drawing. 4*N*

The shortness of the long bones is due to an anomalous development of their epiphyseal cartilages. There are two varieties of this anomaly. One, the "hyperplastic" form, reproduces many of the anatomical features of rickets. Growth of the epiphyseal cartilage is excessive and disordered. In place of the normal columnar arrangement of cartilage cells at the growing ends of the bones, these cells, though unduly numerous, are indiscriminately placed. The result is a heaping up of imperfect cartilage along the line of the epiphysis. The other, "hypoplastic" form, exhibits a similar lack of orderly columnar arrangement of the cartilage-cells, but these are here deficient in quantity also, the cartilage being composed mainly of a hyaline ground-substance. In both cases ossification of the abnormal cartilage is practically absent. The shafts of the long bones are stunted and often imperfectly ossified, while union of the epiphyses with the shafts is long delayed and may never take place. The shortening of the basis cranii is due to premature synostosis of its component bones. 3492*A* 3492*C* 3492*D*

The achondroplasic foetus is generally still-born, but occasionally survives to grow up a dwarf with a well-developed trunk but extraordinarily short limbs. The scapulae, clavicles, hands and feet are proportionate to the size of the trunk, but

the vault of the skull appears unduly large owing to the imperfect development of its base. The mental powers of these dwarfs are fully up to the average standard, in contrast with the intellectual dulness characteristic of cretins.

## 10. CRETINISM.

Drawing. 570*A*

This is an abnormality of development due to *deficiency or absence of a secretion of the thyroid gland.* The changes in the skeleton present many points of resemblance to those of achondroplasia, in that arrest of development is a pronounced feature; but whereas this arrest is selective in the case of the latter disease, the skeleton of a cretin shows a *uniform immaturity.* In both cases the basis cranii undergoes premature synostosis, but the vault of the adult achondroplasic skull is more or less normal, while that of the adult cretin exhibits persistence of the anterior fontanelle, and often a defective union of the cranial sutures. The hands and feet of an achondroplasic dwarf are well-proportioned, but in the case of the cretin the general stunting of the bones leaves the fingers, like those of an infant, nearly of equal length.

286$B_1$ (Case F.)

## 11. HYDROCEPHALUS.

Hydrocephalus signifies an excess of fluid within the skull. The accumulation is usually situated within the brain (internal hydrocephalus) and appears in consequence of some interference with the normal drainage of the ventricles the effect of past meningitis at the base of the brain. The cerebrospinal fluid therefore accumulates within the ventricles and the passages uniting them, distending the brain and with it the skull-cap, since the sutures of the skull are not yet ossified. By these means the cranial cavity may be enormously enlarged, and assumes a globular shape, the bones of the skull being widely separated from each other and only united by areas of membrane containing sesamoid bones here and there. The orbital plates of the frontal bones are depressed, causing protusion of the eyeballs, and the thinned scalp is covered by large, tortuous, distended veins. The disease, when congenital, may give rise to obstructed labour.

When not congenital its commonest cause is meningo-coccal meningitis (posterior basal meningitis). When hydrocephalus is extreme the skin over the enlarged skull is liable to become deeply ulcerated, with the result that fatal meningitis supervenes. Hydrocephalus of any considerable degree gives rise to a proportionate physical and mental impairment. The globular shape of the skull distinguishes this disease from all other kinds of cranial enlargement.

2520 (Case D.)

2521*B* (Case D.)

3489 (Case D.)

## 12. PATHOLOGICAL CHANGES IN THE BONE-MARROW.

The bone-marrow of infants and children is a soft reddish tissue, which in colour and consistency resembles splenic pulp. It consists of a very delicate fibrillar stroma, which carries in its meshes cells of various types, myelocytes, mono-nuclear and polymorphonuclear cells, basophil or mast-cells, and also nucleated and non-nucleated red corpuscles; the lymphocytes of the peripheral blood are normally present in scanty numbers. With advancing years these cells give place in the marrow of the shafts of the long bones to fat-cells, and thus in adult life the spleen-like marrow is replaced by a yellowish fatty tissue, which contains but few marrow cells; these however persist in the spongy bone of the epiphyses and ends of the shafts, giving to them a reddish tint. In old age the fat is absorbed, and the marrow cavity then contains only a gelatinous tissue, which microscopically contains few cells of any description.

Marrow tissue undergoes profound changes in the course of several diseases, which may be grouped as follows:

1. **The wasting diseases of infancy** deprive the marrow of its essential cells, and leave only the supporting reticulum with a few nucleated red cells, and a few myelocytes; to the naked eye the marrow is gelatinous and semi-transparent, as in old age. The most extreme examples of this change are seen in infantile scurvy.

365*A*

2. **The wasting diseases of adult life**, for instance pulmonary tuberculosis, and the cachexia of malignant disease, tend to reconvert the normal yellow fatty marrow into a red splenoid pulp, but this is by no means a constant change.

3. **Acute infective diseases** also change yellow into reddish marrow.

4. In diseases which are accompanied by great destruction
295*F* of the blood-corpuscles, **pernicious malaria, pernicious anaemia,**
295*G* and other similar affections, the marrow again is usually but not invariably of a deep red colour; the change being due partly to increased production of nucleated red corpuscles, partly to disappearance of fat, and partly to hyperaemia of the marrow-vessels.

5. **Acute and chronic lymphocytic leukaemia** cause the
295*D* marrow to assume a deep red colour. The fat is replaced
295*E* by an accumulation of cells of a lymphocytic type, both
295*A* large and small; a type which normally is present in very
295*C* scanty numbers. **Myelocytic leukaemia** produces a grayish-red, or gray colour, due to the accumulation of myelocytes. In both these cases extravasations of blood are apt to take place within the marrow cavity, and have a considerable share in the changes of colour which occur.

6. Similar changes are apt to occur, but are not constant, in such diseases as **round-celled sarcoma** when it is widely diffused.

7. Lastly there is a group of rare diseases in which the marrow of the bones is the seat of a malignant growth; here the cells of the growth vary in type; now they are principally myelocytes, now principally mononuclear non-granular cells, and sometimes frankly lymphocytes. These tumours are
289*F* classed together as **myelomata**; but in one case where the new tissue assumes a greenish tint, due to an unknown pigment,
437*G* the growth is called **Chloroma**. This peculiar growth is most closely related to sarcoma of lymphatic structures, and is probably not primary in the marrow of the bones, but rather in their periosteum.

# CHAPTER III.

## DISEASES OF THE JOINTS.

1. Rheumatic Fever. 2. Gout. 3. Arthritis Deformans. 4. Charcot's Disease. 5. Haemophilia. 6. Infective Arthritis. 7. Syphilitic Synovitis.

**For malformations, injuries and tuberculous disease the student is referred to surgical pathology.**

The structures which enter into the formation of a joint are (1) the ends of the bones, (2) the articular cartilages, (3) the synovial membrane lining the joint-cavity, (4) the ligaments, (5) the thick connective-tissue capsule. Disease of the joint may involve the synovial membrane alone, in which case the term *synovitis* is employed to designate it: when the lesion is not thus limited the more general term *arthritis* is employed.

1. **Rheumatic fever** is undoubtedly the expression of a bacterial infection: but it is a moot point whether the infecting organism is specific, that is to say always of the same variety, or whether various organisms are capable of exciting an apparently identical series of lesions. The disease is characterised by a synovitis which involves several joints. The inflammation, though primarily of the synovial membrane, affects also the capsule, tendon-sheaths, and muscles in the neighbourhood of the joints. The capsule is distended by an effusion of serous fluid, containing flakes of lymph, while the synovial membrane is reddened, swollen and lustreless. The inflammation as a rule subsides, and the effusion is absorbed, leaving the joint unharmed; but in a few instances when the inflammation is chronic or frequently recurrent, changes occur in the cartilages and capsule which produce permanent

Drawing. 65

disability of the joint. Such changes include shortening of the tendons and ligaments about the joint, the result partly of prolonged flexion induced by pain, partly of cicatricial contraction after inflammation. Occasionally the articular surfaces are united by fibrous tissue (fibrous ankylosis).

**Chronic rheumatism** is a term loosely applied to any painful crippling disease of the joints. In the strict sense it
650*C* should be limited to the lesions which attend long-continued or repeated attacks of rheumatic fever.

**2. Gout** is a constitutional disease of which the familiar "gouty arthritis" is merely a local manifestation. The essential pathological change is the deposition in the tissues of crystals of sodium urate. These appear first in the articular
709*B* cartilages of the affected joint, as white specks, or, more often,
710 as a diffuse deposit giving the articular cartilage a gleaming
711 white appearance, like that of fresh paint. The crystals are deposited in the substance of the cartilage, not upon its surface. In long-standing cases the deposits are found not
707 only in the articular cartilages, but in the tendon-sheaths,
707*B* bursae, ligaments, periosteum, and even in the muscles in the
1216*F* neighbourhood of the joint. The deposition of these crystals is attended by a violent inflammatory reaction in the synovial membrane and the tissues round the joint which gives its character to the acute attack of articular gout. After repeated attacks the cartilages are eroded, the periosteum of the bones proliferates with the formation of bony excrescences, and the accumulated uratic deposits protrude the skin, forming the
Cast 23. protuberances known as **tophi** or chalk-stones. The skin over these tophi is reddened and shiny, and may ulcerate, leading to the discharge of inspissated uratic deposit ("gouty
Drawings. abscess"). Tophi are especially frequent over the bony
1161 prominences of the hands and fingers; they occur also in the
1161*A* cartilages of the ear and nose, in bursae over joints, and
Drawings. occasionally in the skin of other parts. The joint most
1160 often attacked by gout is the metatarso-phalangeal of the
1160*A* great toe, but any joint of the fingers or toes may be attacked, while the larger joints, the knee, the elbow, the ankle, and the wrist, do not always escape.

711*E* Suppuration in a gouty joint is a rare complication, and must be regarded as evidence of a secondary infection by micro-organisms.

**3. Arthritis deformans** (osteo-arthritis) is the name given to an affection of the joints characterised by a combination of degenerative and proliferative changes. It must not be supposed that the term denotes any well-defined disease. In the present state of our knowledge it expresses little more than certain anatomical appearances of diseased joints, found under widely differing circumstances and probably to be attributed to a variety of causes.

**The lesions.**—The primary changes consist in degeneration of articular cartilage and destruction of the underlying bone, proliferative new formation of bone and cartilage occurring as later manifestations. In the cartilage the earliest changes are fibrillation and splitting of the columns of cartilage cells, with the result that the articular surface assumes a velvety or shaggy appearance. The fibrillated cartilage wears slowly away and in places exposes the underlying bone, particularly at the points of greatest pressure, while about the periphery of the articular surface the cartilage-cells proliferate, forming nodular outgrowths (ecchondroses). Subsequent ossification of these may produce a fringe of bony projections around the joint (osteophytes). (Case G.)

While these changes are occurring in the cartilage the synovial membrane becomes hyperaemic, and thickened; its fringes are elongated and increased in size owing to an overgrowth of fibrous tissue and a deposit of fat: they project more and more into the cavity of the joint and become subdivided and branched, producing the appearance known as "arborescent lipoma." More rarely nodules of cartilage, which may become calcified or ossified, appear in the enlarged synovial fringes. The synovial fluid is generally increased in quantity, and is rendered turbid owing to the presence in it of oil-droplets and flakes of lymph. The "loose bodies" found in the joints of osteo-arthritic patients are nodules of this kind which have been detached from the synovial membrane.

The bone exposed by the erosion of the articular cartilage in its turn becomes subject to attrition. The surface-layer becomes smooth, compact and "eburnated," while the cancellous bone beneath is absorbed. The friction of opposing surfaces not infrequently produces marked grooving of the eburnated bone. As the central portions of the articular surfaces yield, the peripheral parts come in time to overlap the (Case G.)

696*B* joint. This overlapping together with the osteophyte-formation above described is the cause of the "lipping" of the joint
673*D* characteristic of the disease. The ligaments and tendons about
692*A* the joint may share in the destruction, and in many instances disappear completely; the long tendon of the biceps, after
702 destruction of its intra-capsular portion, may find a new point
703 of attachment below the joint.

Outside the joint the muscles, tendon-sheaths and other structures are affected; the muscles waste, the tendon-sheaths become swollen and inflamed, and the skin shiny and oedematous. Ankylosis (*i.e.* actual union of the articular surfaces) of the affected joints is not common. True bony ankylosis is probably unknown except when the disease attacks the spinal column (spondylitis deformans) and fibrous ankylosis is rare; but a false ankylosis, *i e.* immobility without union, due to the altered position of the articular ends of the bones or to the position of the osteophytes, is not infrequent.

The joints in which the changes above described occur most characteristically are those of the knee, hip, and shoulder; but any joint in the body may be affected, and in certain clinical types of the disease the smaller joints appear to be more susceptible than the larger.

Of the clinical varieties of the disease it must be sufficient here to note that it may occur as:

(*a*) An acute progressive multi-articular type.

(*b*) A chronic multi-articular type.

690 (*c*) A uni-articular type.

1085 (*d*) A type in which the spinal column is principally or
1085*B* alone affected, Spondylitis deformans.

(Case H.) (*e*) A type in which the small joints of the fingers and toes
645*A* are principally or alone affected (Heberden's nodes).

(*f*) An acute multi-articular type peculiar to children: this has some anatomical peculiarities, in that the cartilage-degeneration is as a rule slight and local, and often especially marked at the periphery, the central portions remaining smooth and white. Bony changes are almost absent, the thickening of the joint being due almost entirely to thickening of the capsule and synovial membrane.

Drawings
67
67*B*

4. "**Charcot's disease**" implies an articular affection exhibiting the lesions of arthritis deformans, but occurring in the

course of certain diseases of the nervous system, particularly Tabes dorsalis and Syringomyelia. Its peculiarity is two-fold; first that unlike true arthritis deformans it is almost painless, and secondly that, great as are the proliferative changes, they are far surpassed by the destructive processes; hence in "Charcot's disease," dislocation and sub-luxation of the joint are frequent. All the larger joints are liable to this disease but particularly the knee-joint.

Elbow.—691*H*.
Knee.—691*B*. 691*D*. 691*F*. 691*G*. (Case G.)
Ankle.—691*E*. (Case G.)
Drawings 88, 89, 94.
Photographs 96*A*. 96*B*.

5. **Haemophilia.**—In the joints of haemophilic patients a condition resembling that produced by arthritis deformans sometimes occurs. The changes in the cartilages and the tendency to the formation of ecchondroses and osteophytes are reproduced exactly: but the bones rarely suffer at all, while fibrous adhesions between the opposing surfaces, rare in arthritis deformans, are common in haemophilic joints. The changes only occur after repeated effusions of blood into the joint, and affect most commonly the knee and ankle-joints. The cartilages and synovial membranes usually retain a rusty pigmentation due to the presence of altered haemoglobin deposited from the effused blood.

740*A* 740*B* 740*C* 740*D*

Drawings 83*A* 83*B*

6. **Infective arthritis.**—Infection of a joint may take place (1) by way of an external wound; (2) owing to the involvement of a joint-cavity in an inflammation already existing in its neighbourhood; (3) by way of the blood-stream, either from a distant focus of disease, or in consequence of a general blood-infection by bacteria. The two first of these groups are surgical in character and do not fall within the province of this book. The third or haematogenous group includes tuberculosis of the joints; this again is omitted from consideration here as being within the sphere of surgery rather than of medicine. But this group numbers also a variety of lesions of medical importance.

We have said that infective arthritis, haematogenous in origin, implies a primary focus of infection elsewhere or a general infection of the blood. It is therefore a secondary

manifestation. It is convenient to divide cases of this kind into two categories. In the first category the infective arthritis is due to the same organism as is responsible for the primary disease. Of such are the infective joint-lesions of pyaemia, and those due to the pneumococcus, the gonococcus and the meningococcus. Medically speaking, the most important of these is gonorrhoeal arthritis, the so-called "gonorrhoeal rheumatism."

In the second category, as in the first, the joint affection supervenes in the course of disease elsewhere, but is actually excited by organisms distinct from those responsible for the primary disease. Thus the joint affections associated with dysentery are not excited by the amoeba or bacillus of dysentery, but by streptococci or staphylococci. The same is probably true of the joint complications of scarlet fever and typhoid fever.

**The lesions.**—The early lesions are thickening and hyperaemia of the synovial membrane, together with an effusion of a turbid fluid into the cavity of the joint. At this stage the inflammation may cease, and progress to complete recovery after absorption of the inflammatory exudation. In more
567*D* virulent examples the joint becomes distended with pus; the cartilages and ligaments are softened, and the former often become ulcerated. Recovery after such severe inflammations is commonly attended by some degree of fixation of the joint, the result of ankylosis, fibrous or bony according to the severity of the lesion.

Drawings. **69, 70, 71.**
Bony ankylosis. **630.**
Fibrous ankylosis. **629, 631.**

7. **Syphilitic affections of the joints** occur in two varieties. (1) As a sub-acute synovitis in the secondary stage of the acquired disease; or in children between the sixth and sixteenth years, who are the subjects of inherited syphilis. In these cases the synovitis is usually of the knee-joints, and symmetrical.

(2) As a gummatous synovitis, occurring in the tertiary
567*A* stages of the disease, the synovial membrane being thickened by gummatous deposits. There is an accompanying increase of synovial fluid, and a general thickening of the capsule of the joint, but the bones and cartilages escape.

# CHAPTER IV.

## DISEASES OF VOLUNTARY MUSCLES.

1. Congenital Defects. 2. Atrophy.—(*a*) Primary (Myopathy), (*b*) Secondary (*A*) to overuse, (*B*) to disuse, (*C*) to interference with the trophic influence of nerves. 3. Degenerations.—(*a*) Fatty, (*b*) Zenker's. 4. Inflammation (Myositis). — (*a*) Tuberculous, (*b*) Syphilitic. 5. Ossification. 6. Tumours. 7. Parasites.

### 1. CONGENITAL DEFECTS.

Congenital absence of certain muscles, or portions of them, is not common. The muscles of the shoulder girdle are those most often affected in this way, particularly the pectoralis major.

### 2. ATROPHY

Muscular atrophy whatever its cause is usually associated with degeneration, especially fatty degeneration.

(*a*) **Primary atrophy** (Myopathy, muscular dystrophy).—The group of diseases to which the above names are applied is characterised by atrophy of muscles without any known cause. The atrophy commences in certain more or less defined groups of muscles, but in course of time becomes general. It may be preceded by a phase of real or apparent hypertrophy of the muscles affected (pseudo-hypertrophic muscular paralysis). In this case the enlargement of the muscle is mainly due to an increase in its connective tissue elements and to the appearance of fat-cells between the muscle-bundles. True hypertrophy is rare, though it is said to occur. As the disease

progresses the muscle-fibres disappear in the surrounding connective-tissue and fat.

(*b*) **Secondary atrophy.**—(*A*) **From overuse.**—It is said that excessive use of individual muscles is capable of causing atrophy in them. This certainly appears to occur in the small muscles of the hand as a result of constant repetition of the same movement in certain crafts.

(*B*) **From disuse.**—A muscle rendered functionless by injury, or by chronic disease of the joint it serves, undergoes a slow atrophy, ending in course of time in the replacement of its fibres by fat and connective-tissues.

(*C*) **From interference with the trophic influence of nerves.**—The motor nerve-trunks contain fibres which exercise a trophic influence upon the muscles to which they are distributed, and the removal of this influence by disease or injury leads to muscular atrophy. In the diseases known as Progressive muscular atrophy, Acute anterior polio-myelitis, and Bulbar paralysis, the lesion is situated in the anterior horn cells of the central nervous system, and the resulting atrophy is spoken of as "central muscular atrophy." But a similar result attends lesions of the peripheral motor system of nerves. Thus division of a nerve, and all forms of peripheral neuritis, are liable to be attended by atrophy of the muscles concerned. The atrophies of this group are distinguished as "neural muscular atrophies."

Drawing 138*A*

Of late years numerous cases have been reported in which atrophy of the muscles of the hand and forearm has been associated with the presence of cervical ribs. It is still a matter of dispute in what way the rib causes the atrophy.

1168*B*

Whatever the cause of muscular atrophy the final result is the same, namely, disappearance of muscle-fibres and replacement of them by fat and connective-tissue. The inter-muscular septa survive and outline the sites of the vanished muscles.

## 3. DEGENERATIONS.

(*a*) **Fatty degeneration of muscle** is characterised by the appearance of droplets of fat in the substance of the muscle-fibres. It is a constant feature of severe anaemias, wasting diseases, and acute infections.

1168

(*b*) **Zenker's hyaline degeneration.**—This degeneration is

characterised by the conversion of the contractile substance of muscle into a homogeneous material giving the muscle a transparent appearance. It is common in connection with acute febrile diseases, especially typhoid fever.

## 4. INFLAMMATION.

Myositis, or inflammation of voluntary muscles, is rare, with the exception of the tuberculous and syphilitic forms.

(*a*) **Tuberculous myositis.**—Tuberculous myositis is generally secondary to tuberculosis of a neighbouring bone or joint. The commonest example of the lesion is the affection of the psoas muscle which often attends tuberculous caries of the lumbar vertebrae, the infection spreading directly from the diseased bone. In this case a large part of the psoas muscle 1171
may be destroyed and replaced by caseous material (**psoas** 1171*B*
**abscess**). But occasionally tuberculous abscesses are met with in muscles remote from any obvious seat of disease, the infection having apparently reached the muscle by the blood-stream. On the whole the voluntary muscles are remarkably immune to tuberculosis.

(*b*) **Syphilitic myositis.**—Gummata not uncommonly appear in voluntary muscles, especially those of the arm and neck, and may form tumours of considerable size.

**Microscopic examination.**—In the earlier stages of the lesion the muscle-fibres are separated from each other by an abundant exudation of round-cells and plasma-cells. This exudation shows a tendency to degenerate rapidly, leading to the appearance of caseous areas intersected by bands of cellular fibrous tissue. At the margins of the caseous areas it is not uncommon to find a few giant-cells, together with obliterative endarteritis of the smaller blood-vessels.

## 5. OSSIFICATION.

Bony deposits in muscle are by no means uncommon. They appear to result most often from constant exertion of, or repeated slight injuries to, certain muscles, especially the deltoids and the adductor muscles of the thighs. The bony deposit in the latter case is known as "rider's bone" from the frequency of its occurrence in men who ride.

A second variety of muscular ossification constitutes the disease known as Progressive Myositis Ossificans. The affec-

tion generally begins in the muscles of the neck, shoulders and
Casts. thorax, and involves one muscle after another until the affected
66*C* individual is more or less completely encased in a bony
66*D* cuirasse. In the early stages the damaged muscles are painful, and exhibit an inelastic enlargement, which subsides as the development of bone progresses. The formation of bone occurs in the interstitial connective-tissue, not in the muscle-fibres themselves.

## 6. TUMOURS.

(*a*) **Innocent tumours** are rare, but fibromata, lipomata, and lymphangiomata are met with.

(*b*) **Malignant tumours**.—Primary malignant tumours of
1174$C_2$ muscle are invariably sarcomata, usually round-celled. Sometimes bone or cartilage occurs in these growths (osteo-sarcoma,
1174$C_1$ chondro-sarcoma), and mucoid degeneration of them is common
1174$D_1$ (myxo-sarcoma).

Secondary malignant tumours in muscle may be either
1174*G* sarcomata or carcinomata.

## 7. PARASITES.

(For the life-history of these parasites *v.* Chap. I.)

(*a*) **Hydatid cysts** are occasionally found in voluntary
1175*A* muscles, but present no features special to the locality.

(*b*) **Cysticercus cellulosae.**—This represents the cystic stage of Taenia solium. It gives rise to the development between the muscle-bundles of elliptical cysts about half-an-inch long. Each cyst comprises a capsule formed by the condensed tissues
1175 of the host, within which lies the parasitic cyst containing the
1176 embryo.

(*c*) **Trichinosis** (**Trichinelliasis**).—This disease is caused by the presence in voluntary muscles of Trichina Spiralis in its larval, cystic stage. To the naked eye the affected muscle appears profusely dotted with minute specks, often of a dead-white colour owing to calcification of the cysts. Examination
1176*A* with a lens shows that each cyst is oval in form with a pro-
1176*B* longation at either pole. Within the cyst lies the coiled worm.

# CHAPTER V.

## DISEASES OF THE PERICARDIUM AND HEART.

I. Congenital Heart Disease. II. The Pericardium.—1. Malformations. 2 Pericarditis. 3. Tuberculosis. 4. Milk-spots. 5. Haemo-pericardium. 6. Pneumo-pericardium. 7. Tumours. III. The Myocardium.—(*A*) Alterations in size of the heart, (*B*) Alterations in the myocardium (Myocarditis), (*C*) Cardiac aneurysms and ruptures, (*D*) Parasites of the heart-muscle, (*E*) Tumours. IV. The Endocardium and Valves.—(*A*) Endocarditis and valvular disease, (*B*) Compensation, (*C*) Valvular aneurysms. V. The Coronary Arteries. VI. Intra-cardiac Thrombi. VII. Embolism and Infarction.

### I. CONGENITAL HEART DISEASE.

Congenital affections of the heart result (*A*) **from anomalies of development**; (*B*) **from intra-uterine endocarditis.** These two varieties may occur in the same specimen, deformed structures being especially liable to inflammations.

(*A*) **1. Developmental anomalies affecting the heart as a whole.**

(*a*) Acardia, or absence of the heart: an anatomical curiosity.

(*b*) Dextro-cardia or transposition of the heart. This condition is compatible with perfect health. The transposition may affect the heart alone, or may involve the whole of the viscera.

(*c*) Ectopia cordis.

**2. Anomalies affecting the cardiac septa.**

3606 (*a*) Absence of the auricular septum.

(*b*) Persistence of the foramen ovale. This lesion is commonly found by accident in individuals who
3593 during life presented no evidences of it. In these
3594 cases the orifice is often a valvular slit.

3615 (*c*) Absence of the ventricular septum.

(*d*) Patency of the "undefended space," an area at the upper extremity of the ventricular septum, which is
3609 normally closed not by muscle but by fibrous tissue. Congenital pulmonary stenosis is usually accompanied by this deformity, but the latter may occur alone.

**3. Anomalies of the valves.**

(*a*) Variation in the number of valve-cusps is common in the case of the sigmoid valves: rare in the case of the mitral and tri-cuspid. Supernumerary cusps are of little importance, but fusion of cusps appears to render the valve more liable to inflammation. This fusion is sometimes due to faulty development, sometimes probably to intra-uterine endocarditis.

Pulmonary valves. **3584. 3587. 3588.**

Aortic valves. **3586*A*. 3589. 3590.**

3600 (*b*) There may be no trace at all of an auriculo-ventri-
3606 cular orifice. This is commonest on the right side of
3612*B* the heart, and is then associated with a rudimentary condition of the right ventricle.

3604 **4. Anomalies of the great vessels.**—(*a*) Persistence of the
3618 right aortic arch; (*b*) transposition of pulmonary artery and aorta.

(*B*) **Foetal endocarditis.** (1) **Congenital pulmonary stenosis.**—This lesion, the commonest of all congenital errors, is often purely developmental in origin. In this case the pulmonary conus arteriosus is narrowed as well as the valvular orifice. When due to foetal endocarditis the stenosis is situated at the pulmonary valve itself, the valve-cusps being fused even to total obliteration of the channel. As a rule, however, a narrow orifice remains. Pulmonary stenosis is
3609 often accompanied by a partial deficiency of the interventri-
3611 cular septum, and by a persistent patency of the ductus
3612 arteriosus. Clinically it gives rise to dyspnoea, cyanosis,

clubbing of the fingers, and a harsh systolic murmur, often 3612*B*
attended by a thrill, of maximum intensity over the second left costal cartilage.

(2) When the aortic orifice is affected the endocarditis is more often of the warty type, and is very often assigned to syphilis, but without sufficient ground.

(3) It is rare to find lesions of this class at the mitral or tricuspid orifices.

## II. PERICARDIUM.

1. **Malformations.**—Absence of the pericardial sac. This is a rare event, and when it occurs is generally associated with other gross deformities such as ectopia cordis. 3621

2. **Pericarditis. General considerations.**—Inflammation of the pericardium may be primary or secondary to disease elsewhere. Clinically considered, cases of pericarditis may be divided into two groups, one marked by scanty, highly-fibrinous exudate—*dry pericarditis*—the other by a more copious and more liquid exudate—*pericarditis with effusion.* The latter cases are further divisible, according to the character of the effusion, into the examples of *serous* and of *purulent* pericarditis. But from the point of view of morbid anatomy the line of demarcation between these groups is not easily drawn: for inflammatory exudates present in different cases all gradations of consistency from the purely fibrinous to the definitely fluid, while the same cause may produce in one part of the pericardial sac a dry pericarditis, and in another a pericarditis with effusion. But the classification given above accents the common clinical types, and will be followed in the ensuing description. It is only necessary for the student to bear in mind that a dry pericarditis may pass into a pericarditis with effusion, or may co-exist with it. Haemorrhagic affections of the pericardium do not form a well-defined group: the chief conditions under which they occur are briefly described below.

**Acute pericarditis.** (*a*) **Fibrinous or dry.**—The common causes of acute fibrinous pericarditis, in a descending scale of importance, are rheumatic fever, chorea, Bright's disease, tuberculosis (this variety will be separately described), and occasionally some of the specific fevers.

The earliest change in the membrane is a dilatation of its

capillaries especially towards the base of the heart, followed presently by an effusion of lymph by which the surface of the pericardium is roughened. As the effusion increases the opposed surfaces of the pericardium become covered by a thick gelatinous coat. Under the influence of the cardiac movements
1225 this viscid covering is drawn into strands which, upon separa-
1226 tion of the pericardial layers after death, gives to the opposed
1310 surfaces a honey-combed or shaggy appearance (cor villosum).

**Microscopic examination** shows that the exudate consists of fibrin and leucocytes. The heart-muscle immediately beneath the visceral pericardium is inflamed and often degenerate.

(*b*) **Pericarditis with effusion.**—(1) Serous; (2) purulent.

(1) The causes of a **serous pericardial effusion** are identical with those of the dry variety. It should be mentioned, however, that in cases of general anasarca the pericardium, like the other serous sacs, may be distended by a serous effusion which is a mere transudation and not inflammatory at all.

The serous membrane in this condition resembles the appearances given above, that is to say some roughening and injection occur, but coagulable lymph is much less in evidence. The quantity of the effusion is seldom great, but may in exceptional
1231*D* instances amount to several pints.

(2) **Purulent pericarditis.**—Purulent pericarditis occurs in association with empyemata, in which case the organism responsible for the lesion is usually the pneumococcus; also
1233*B* with the acute infectious fevers, particularly scarlet fever;
1233*C* with ulcerative endocarditis; with pneumonia, and with
1233*E* pyaemia.
1233*F* The pus is seldom present in large quantity. It may be
1238*A* thin or thick, according to the virulence or otherwise of the infection. In very chronic cases calcareous deposits may appear in the inspissated pus.
1238*L* Among the rarer causes of pericarditis in general may be
1238*M* mentioned an extension of inflammation from a mediastinal or hepatic abscess, and erosion by a malignant growth or the pressure of an aneurysm. Also, pericarditis, both dry and (more often) with effusion, is a common outcome of a terminal infection in chronic illnesses.

**Haemorrhagic affections of the pericardium.**—Ecchymoses
1296 of the pericardium occur in cases of death by suffocation (Tardieu's spots): in scurvy, purpura, leukaemia, in infective

diseases such as septico-pyaemia and ulcerative endocarditis, and in the haemorrhagic forms of infectious fevers like smallpox. 1233*D* 1302*C*

Haemorrhagic effusions which are not pure blood occur in the course of Bright's disease and haemorrhagic diseases generally. Effusion of pure blood into the pericardial sac is described under the title "Haemopericardium."

**Sequelae of pericarditis.** (Chronic pericarditis. Adherent pericardium).—The usual effect of an attack of pericarditis is more or less adhesion between the visceral and parietal layers of the membrane: for though complete resolution is conceivable it is probable that in the case of the pericardium it never occurs. At times the adhesions are slight, being represented by thin fibrous filaments: but in most fatal cases the adherent layers of the membrane are at least $\frac{1}{8}$ inch in thickness. 1229 Occasionally when organisation has not progressed very far, a 1231 fresh exudation converts the pericardial sac into a series of 1232 cavities lined by a gelatinous material and containing serous fluid

Both the pleura and the mediastinal connective-tissues may share in a pericardial inflammation. In extreme cases of this kind the contents of the thorax form a solid mass, encased in fibrous tissue and firmly adherent to the sternum, the ribs, and the diaphragm (mediastinitis).

*All forms of pericardial adhesion lead to a generalised hypertrophy of the heart.*

3. **Tuberculosis of the pericardium.**—This presents three varieties.

(*a*) **Miliary tubercles** may occur either without much evidence of inflammation, or on the other hand accompanied by a large serous effusion. 1231*C* 1231*D*

(*b*) **Solitary tumours** of caseous material are sometimes found, chiefly towards the base of the heart, and situated upon the parietal pericardium.

(*c*) **Caseous masses** may form in the exudations, and involve both layers of the sac. 1231*B*

4. **Milk spots.**—These are most probably the result of a slow inflammation comparable to the formation of corns in the skin. As a rule they are found only upon the visceral layer, but occasionally the process which causes them produces adhesion between the two layers at the affected spot. The commonest

situation for a milk-spot is upon the anterior wall of the left
1240 ventricle at a distance of one or two inches from the apex of the heart.

The spot may be of considerable size and varies in shape. It is white, opaque, and slightly raised above the surface, but retains the glistening appearance of the normal membrane.

5. **Haemopericardium.**—Effusion of pure blood into the pericardial sac occurs:

(1) as the result of a wound which effects a communication between the sac on the one hand and the cavity of the heart or great vessels on the other;

(2) from rupture of the heart;

1480*A* (3) from rupture of an aneurysm into the sac.

6. **Pneumo-pericardium.**- The presence of gas or air within the pericardial sac is a rare phenomenon. It occurs:

(1) In infections of the pericardium by gas forming organisms.

(2) When a communication is established between the interior of the sac and some hollow viscus, as for example by a malignant ulcer of the oesophagus or stomach.

In any case pneumo-pericardium is generally associated with a purulent effusion into the sac—pyo-pneumo-pericardium.

7. **Tumours of the pericardium.**—Primary new growths of
1239 the pericardium are extremely rare and are always sarcomata
1285 or endotheliomata. Secondary growths may invade the pericardium from any of the adjacent structures.

## III. (*A*) THE HEART.

**Alterations in size.**—(1) Decrease; (2) Increase.

1. **Decrease.**—Atrophy of the heart as a whole is met with chiefly in old people, or in those who have suffered from chronic starvation owing to such diseases as oesophageal or gastric cancer.

The organ is smaller than the normal heart: the epicardial fat has entirely disappeared: the pericardium has a wrinkled and shrunken surface, while the coronary vessels and their
1242 branches are unusually tortuous and prominent. In a well-
1243 marked case the heart-muscle has a brown appearance (brown
1243*A* atrophy).

**Microscopic examination** shows that the brown appearance depends upon the deposition within the muscle-fibre of granules of a golden-brown pigment. This deposit is most in evidence near the nuclei of the fibres, but may be distributed throughout their length. The atrophy is in all cases due to an actual diminution in the size of the muscle-fibres, and not in their number.

**2. Increase in the size of the heart** is due to (*a*) Hypertrophy; (*b*) Dilatation; (*c*) Lipomatosis.

(*a*) **Hypertrophy.**—This is always symptomatic of increased work and therefore appears first in that portion of the heart which is most exposed to the increased strain. Hence the left ventricle is hypertrophied in disease of the aortic valve, in mitral regurgitation, in renal disease and arteriosclerosis and after prolonged muscular exertion, such as is involved in the pursuit of athletics. 1241 1241*A* 1241*B* 3214

The right ventricle suffers most in pulmonary diseases and in diseases of the pulmonary and tricuspid valves. 1271*C* 1316*A*

In the case of the auricles hypertrophy is always associated with dilatation. The left auricle suffers in mitral stenosis; the right whenever there is an increased pressure in the pulmonary circulation, whether this is due to disease of the mitral orifice or of the lung. Tricuspid stenosis is rare, but its prime effect is exercised upon the right auricle. 1321*A* 1321*B* 1259*B* 1327*A* 1321*B*

Hypertrophy of the ventricles is usually divided for descriptive purposes into two types—(1) Concentric hypertrophy; (2) Eccentric hypertrophy.

(1) **Concentric hypertrophy** indicates a hypertrophy of the ventricular wall without any increase in the size of the cavity. It is seldom seen except in connection with well-marked cases of pure aortic stenosis. 1353*A*

(2) **Eccentric hypertrophy** is the common form. It implies that there is also dilatation of the ventricular cavity. 1257

**Hypertrophy of the whole heart** occurs—(1) When there is complete adhesion of the pericardial layers to each other. (2) In obese muscular men. To the latter class belong the enormous hearts to which the name "bullock hearts" has been given. The heaviest heart on record weighed 53 oz. as against the normal weight of 9 oz. 3214

Hypertrophy alters the shape of the heart as well as its size. The whole organ tends to become more square than elongated, while the apex is rounded, and, in extreme cases, almost lost in the general outline.

(*b*) **Dilatation.**—Dilatation of the heart is commonly met
1259*B* with in conjunction with hypertrophy in cases of chronic
1334 valvular disease and of chronic lung disease, especially emphy-
1271*C* sema. Apart from hypertrophy it occurs in various acute diseases as a result of myocardial weakening due to degeneration or inflammation. The commonest causes of this acute dilatation are rheumatic fever, diphtheria, and severe anaemias.

(*c*) **Lipomatosis.**—Lipomatosis of the heart occurs as an incident of general obesity. The heart is considerably enlarged, but there is neither hypertrophy nor dilatation. The enlargement is due to a great increase of the epicardial fat, which appears between the muscle-bundles, and may even penetrate the thickness of the heart-wall and be visible
1245 beneath the endocardium.

(*B*) **Alterations in the myocardium.**—Myocarditis—(1) Acute; (2) Chronic.

(1) **Acute myocarditis.**—This is always the result of an infection, the degree of damage depending upon the nature and virulence of the poisons at work. This damage may be inflammatory in the main, or chiefly degenerative. In most examples of pericarditis and endocarditis the myocardium in the neighbourhood of the lesion shares in the inflammation. But certain bacterial poisons, notably the poison of diphtheria, are capable of producing a myocardial degeneration without inflammation in the ordinary sense.

1233*C* Suppurative myocarditis occurs in the course of pyaemia.
1279 The lesion consists of minute metastatic abscesses in the muscle, often visible to the naked eye.

When myocarditis, originally acute, becomes chronic, the affected portions of the heart become the seat of fibrosis (*v.* chronic myocarditis).

(2) **Chronic myocarditis** —This heading embraces a variety of lesions of the myocardium, some degenerative, some inflammatory.

1243*A* (*a*) **Brown atrophy** has already been described.

(*b*) **Zenker's hyaline degeneration** is a rare affection occurring in the course of some of the acute specific fevers, particularly typhoid fever. It can seldom be detected by the naked eye.

**Microscopic examination** shows that the muscle fibres lose their striation and are converted into transparent hyaline bands.

(*c*) **Fatty degeneration** and infiltration. This is the result of infections and intoxications such as those of diphtheria and tuberculosis, grave anaemias and diabetes. It occurs also as a sequel of acute myocarditis and pericarditis: as a result of sclerosis of the coronary arteries; and in connection with the lipomatosis of the heart described above.

A heart thus affected becomes stippled with white or yellowish dots and stripes, the appearance being most marked in the papillary muscles of the left ventricle ("Tabby-cat" or "tiger" striation). Extreme fatty degeneration and infiltration of the heart may result in spontaneous rupture. 1256 1299*H*

(*d*) **Fibroid degeneration** or induration of the heart-muscle is always associated with some degree of fatty degeneration. It occurs:

(*a*) As a sequel of acute myocarditis. 1246

(*b*) As the result of narrowing of the coronary arteries due to arterio-sclerosis.

(*c*) As a result of syphilitic inflammation, including both gummata of the heart-wall, and syphilitic endarteritis of the coronary arteries. 1261*B* 1280

(*d*) As part of a general arteriosclerosis, being then associated with hypertrophy of the heart.

(*e*) As a result of embolism or thrombosis of the coronary arteries (Myomalacia cordis).

The muscle of a heart affected with fibroid degeneration is less ruddy than normal muscle, firmer to the touch, less friable, and often exhibits white patches of fibrous tissue interspersed among the muscle-bundles. These changes are most frequently seen in the musculi papillares of hearts affected by chronic valvular disease, but often appear upon the ventricular walls.

**Myomalacia cordis** deserves separate consideration. It implies an infarction of the heart-wall by blockage of a coronary artery or of some important branch of it. The infarcted area appears as a patch in the heart-wall, dull-yellow or pearly-white according to its age, or, very rarely, haemorrhagic. Its usual situation is in the wall of the left ventricle or upon the septum ventriculorum. 1249

**Microscopic examination** of the infarct shows in the early stages necrosis and fatty degeneration, and later, fibrosis.

The results of such a lesion vary with the extent and

situation of the infarct. Should it occupy the whole thickness of the wall the softened area may yield to the blood-pressure and become an aneurysm of the heart, or may rupture completely. Should it be superficial it will give rise to a localised pericarditis; if immediately beneath the endocardium the result is
1284 likely to be thrombosis within the ventricle.

(*e*) **Tuberculosis** of the myocardium is rare. It occurs chiefly
1281 in the course of general miliary tuberculosis. The tubercles
1690*E* may be miliary, or may exist in the form of caseous masses of considerable size.

(*f*) **Syphilitic myocarditis**—(*a*) Gummatous; (*b*) Endarterial.

(*a*) Gummata appear in the myocardium in the form of white or yellowish caseous masses, commonly associated with a wide-spread interstitial myocarditis, the latter being cellular in the acute phase, but later fibrotic. Gumma of the heart-muscle is an occasional cause of sudden death.

1280*A* (*b*) Syphilitic endarteritis of the coronary arteries with an
1280*B* attendant fibroid induration of the myocardium is a commoner event than gumma. This variety cannot be distinguished by the eye from other kinds of fibroid induration.

(*C*) **Cardiac aneurysms and ruptures.**—Cardiac aneurysms are pouches formed in the heart-muscle itself. They may be (*a*) acute; (*b*) chronic.

(*a*) Acute cardiac aneurysms result from the weakening of
1365*B* some part of the heart-wall by acute inflammations. They thus occur either as a consequence of ulcerative endocarditis or of infective embolism of a branch of a coronary artery.

(*b*) Chronic cardiac aneurysms depend upon the yielding of
1261*B* heart-muscle under the influence of one or other of the chronic
1263*A* degenerations described above. They vary in size from that of
1264 a pea to that of an orange, and are situated most often in the
1264*B* walls of the ventricles. They may be empty after death, or lined by laminated clot. The muscle in the neighbourhood always shows signs of degeneration, usually of the fibroid variety.

Cardiac aneurysms in general occur most often near the apex of the left ventricle. But they may attack the septum ventriculorum or the auricles.

1250 **Spontaneous ruptures** of the heart are due either to
1249 infective foci in the muscle, or to chronic degenerations of
1251 it, especially those depending upon sclerosis of the coronary
1253 arteries which entails faulty nutrition of the heart.

The rupture is usually situated on the anterior aspect of the 1248*B*
left ventricle, occasionally on that of the right ventricle, or in 1254
the septum ventriculorum.

(*D*) **Parasites of the heart-muscle.**—The myocardium may be 1295
the seat of parasitic cysts, both the cysticercus cellulosae and 1295*A*
the hydatid varieties being met with. Rupture of such cysts 1295*B*
into the chambers of the heart leads to parasitic embolisms.

(*E*) **Tumours of the myocardium.**—(*a*) Primary; (*b*) secondary.

(*a*) Primary growths are rare, but a few examples of primary sarcoma have been recorded, and some innocent growths of congenital origin,—fibromata and leio-myomata.

(*b*) Secondary growths are more common. They may be 1292
either carcinomatous or sarcomatous. Secondary carcinoma is the less frequent.

Secondary sarcoma may attack the myocardium by direct 1285
extension from a neighbouring growth. More often myocar- 1285*C*
dial sarcomata represent metastatic deposits. 1285*D*
1289*A*

## IV. THE ENDOCARDIUM AND VALVES.

(*A*) **Endocarditis.**—(1) Acute; (2) Chronic.

### 1. Acute Endocarditis.

Acute endocarditis attacks most often the endocardium of the valve curtains, that is, it is a valvular endocarditis. Its commonest causes are **rheumatic fever** and **chorea**, but any infective disease is capable of causing it, while in a number of examples met with post-mortem the endocarditis depends upon a terminal infection superimposed upon some grave constitutional disease. Formerly a clear distinction was attempted between "acute simple endocarditis" on the one hand, and "ulcerative" or "malignant endocarditis" on the other. The distinction remains useful both in morbid anatomy and in clinical medicine, but pathologically speaking no strict line of demarcation separates the two varieties since both depend upon the action of micro-organisms. The most benign inflammations and the most destructive must therefore be regarded as

the extreme poles of one pathological process: and it is common to encounter lesions of every degree between them.

The valves are attacked in a descending scale of frequency as follows: mitral, aortic, tricuspid, pulmonary. That is to say, the left side of the heart suffers more than the right. Of intra-uterine endocarditis, however, the reverse is true, the right side suffering most (*v.* Foetal endocarditis, p. 56). It must further be said that in the case of children with advanced valvular disease the tricupsid valve often shares in the inflammation of the endocardium, while certain of the more virulent infections, *e.g.* pneumococcal endocarditis, attack the pulmonary and tricuspid valves before the mitral and aortic.

**The lesions.**—The lesion of the valve-cusps commences, not at their free margins, but along the line of their maximum
1330*A* contact during closure. Along this line the endocardium
1331 undergoes a superficial inflammatory necrosis, the surface thus roughened becoming then the seat of a deposit of fibrin, derived from the circulating blood. These minute thrombi are called "**Vegetations.**" At first of a delicate pink colour and almost transparent, they become, as time goes on, organised and fibrotic, appearing as white, opaque, glistening nodules to which may be adherent small recent thrombi. In chronic cases the vegetations have the aspect of warty outgrowths often capped by thrombi of large dimensions.

When the inflammation is intense, and the proliferation of connective-tissue not active, ulceration occurs, and may progress to the destruction of the whole or a part of the valve-curtain, sometimes involving also the muscular tissue of
1297 the heart-wall or the chordae tendineae. In such a case the
1298 endocarditis is said to be "ulcerative" or "malignant": if the
1302*C* heart-wall is attacked the term "mural" endocarditis is applied
1302*D* to the lesion.

**Remote effects of acute endocarditis.**—Detached portions of vegetations formed upon the valves are liable to be swept along by the blood-stream until, reaching a vessel of calibre too small to permit of their passage, they become impacted. Such impacted particles are called "**emboli.**" When the impaction affects a terminal arteriole, *i.e.* one possessing only insignificant anastomoses with its neighbours, the area of tissue which is deprived of its blood supply passes into a condition of necrosis termed "**infarction,**" the necrotic area itself being

called an "**infarct.**" Embolism and infarction are described on p. 72.

### 2. Chronic Endocarditis.

(*A*) **Secondary to acute endocarditis.**—It is possible that acute endocarditis may be of a degree so slight as to permit of perfect resolution, the affected areas returning to a normal condition. But such an event, if it occurs, is rare. Usually the inflammatory infiltration becomes permeated by newly-formed capillaries and replaced by fibrous tissue, the shrinkage of which, by deforming the shape of the valve-curtains and impairing their mobility produces the common lesions embraced by the term "chronic valvular disease."

(*B*) **Primary degenerative endocarditis.**—Although chronic valvular disease is most often a sequel of acute endocarditis, the same effects may result from a slow degeneration without the intervention of an acute stage. This sclerosing endocarditis is generally found as a part of a wide-spread degeneration of the vascular system, affecting for the most part elderly people, and in particular persons tainted by gout, syphilis, and alcoholic or lead-poisoning, or those whose arterial system has been subjected to continued high pressure. 1353*A*
Primary sclerosing endocarditis attacks the aortic valves most 1353*C*
often: but in the case of middle-aged women the mitral valve is 1303*A*
frequently affected, without any recognised predisposing cause.

**The mechanical effects of chronic valvular disease.**—These are exercised in one or both of two directions: (1) the deformity and abnormal rigidity of the valve-curtains prevents their accurate apposition; the valve becomes incompetent and permits the regurgitation of blood through it; (2) the valvular orifice becomes narrowed (stenosis), thus offering an actual obstruction to the passage through it of blood in normal quantity.

**Stenosis and regurgitation.**—A valve which is damaged to the extent of being stenosed must be incompetent also as a general rule. But clinically a valve may be stenosed yet competent. This is not uncommonly the case with the mitral valve, the physical signs indicating stenosis unaccompanied by regurgitation. The explanation is probably this: (1) that the blood-pressure within the left auricle is maintained at an abnormally high pitch by the stenosis of the valve; (2) that the quantity of blood within the left ventricle at each systole

is abnormally small, by reason of the narrowed orifice. Both these factors will tend to prevent reflux of blood through the mitral orifice during the systole of the ventricle. The term "pure stenosis," when applied to an orifice of the heart bears therefore a clinical and not a literal significance. But in a vast majority of cases a valve which is stenosed is also regurgitant, and many of the specimens cited exemplify both conditions.

As regards regurgitation or incompetence of a valve, it is necessary to note that this may be absolute or relative. When the curtains of a valve are so stiffened or deformed by disease that they do not meet properly although the orifice they should guard remains unaltered in size, the incompetence of the valve is said to be *absolute*. But occasionally, owing to dilatation of the heart, the orifice itself becomes enlarged, in which case a valve may be incompetent though not diseased. This form is called *relative* incompetence. It is observed most often at the tricuspid orifice.

**Disease of the mitral valve** —(1) Stenosis; (2) regurgitation; (3) stenosis with regurgitation.

1298 (1) **Mitral stenosis** may be acute or chronic.

1299*H* 1. In acute cases the lesion is an ulcerative endocarditis,
1301 the orifice being blocked to a greater or less extent by projec-
1302*B* ting vegetations.

1302*C* 2. In chronic cases the orifice of the valve may be reduced
1327 to a mere slit in the sclerotic tissues replacing the valve—the "button-hole" form—or to a small rounded or funnel-shaped opening. In less extreme cases the valve-curtains show puckering and increased opacity.

As regards the musculature of the heart the stress of mitral
1327*A* stenosis falls upon the left auricle in the first instance, causing
1259 it to become both dilated and hypertrophied. In the dilated
1274 auricle thrombi are frequently deposited during life. These
1303*A* thrombi may be merely flat clots adherent to the wall, but
1304 occasionally spherical free clots—the "ball" or "bullet"
1328 thrombi may be encountered. These are peculiar to mitral
1329 stenosis.

(2) **Mitral regurgitation** may be acute or chronic.

Mitral regurgitation of recent origin is due to dilatation of
1310 the mitral ring, accompanied by vegetations which prevent
1330*A* the accurate apposition of the valve-curtains.

In lesions of old standing sclerosis of the valve prevents its complete closure, while narrowing the orifice. Hence in such instances stenosis and incompetence commonly coexist. 1298

**Disease of the aortic valve.**—(1) Stenosis; (2) regurgitation; (3) stenosis with regurgitation.

(1) **Aortic stenosis.**—Pure stenosis of the aortic valve, in the clinical sense defined above, is rare, far rarer than so-called pure mitral stenosis. In acute cases it depends upon the blocking of the orifice by vegetations. 1299*B* 1299*D* 1299*G* 1340

More often it depends upon a slow sclerosis of the valve, occurring in old age. This so-called pure stenosis of the aortic valve gives rise to that form of hypertrophy of the left ventricle which is called "concentric," implying hypertrophy without dilatation. It should be remembered that stenosis of the aortic valve is not common, the murmur suggesting it being more often due to roughening of the valve-cusps, or of the aorta beyond them, than to actual narrowing of the orifice. 1353*A* 1347 1350 1353

(2) **Aortic regurgitation.**—This also may be acute or chronic. In either case the left ventricle suffers most and may attain an extreme grade of hypertrophy. 1319 1353 1353*C*

**Disease of the tricuspid valve.**—(1) Stenosis; (2) regurgitation.

(1) **Tricuspid stenosis** is rare and when it occurs is invariably associated with a similar lesion of the mitral valve. It requires no special description beyond the statement that the effect of the lesion is felt in the first instance by the right auricle and the systemic veins. 1304*A* 1311 1314 1312

(2) **Tricuspid regurgitation.**—This may result from disease of the valve, but is more often secondary to valvular disease elsewhere or to diseases of the lung which obstruct the pulmonary circulation, such as emphysema and chronic bronchitis. This is to say that the incompetence of the valve is more often relative than absolute, resulting rather from dilatation of the whole orifice than from deformities of the valve-curtains. The right auricle suffers most. 1321*A*

**Disease of the pulmonary valve.**—Stenosis of this valve is usually congenital. It is described under the heading "Congenital pulmonary stenosis," p. 56. 1321*B* 1321*C* 1316*A*

Regurgitation is rare and when it occurs is almost always due to ulcerative endocarditis. 1317 1322

(*B*) **Compensation.**—Mechanical deficiency of any valve implies additional work for the heart if the integrity of the circulation is to be maintained. Extra calls thus made upon the heart are met by muscular hypertrophy of the chamber or chambers most concerned. This responsive hypertrophy is called "compensatory hypertrophy" or "compensation." When the recuperative power of the heart is no longer equal to the growing demands upon it, dilatation occurs and compensation is said to have failed. It is clear that compensation is dependent upon the vitality of the myocardium: hence in youth, when the coronary arteries are healthy and recuperative power is at its height, valvular lesions are more readily compensated and compensation is more efficiently maintained than in later life. But at any age a sudden extraordinary demand upon a compensated heart, such as the strain of pregnancy or of constitutional disease, is likely to produce dilatation.

**Failure of compensation.**—When for any reason compensation fails, there commences a series of events which if continued ends in the mechanical disorganisation of the whole heart, whichever the orifice primarily at fault. For descriptive purposes we may select the commonest case, a compensated heart affected with mitral regurgitation, and trace the changes consequent upon the failure of compensation. *Mutatis mutandis* this description may be applied to all other valvular lesions.

In the case of mitral regurgitation compensation implies an hypertrophy of the left auricle and ventricle sufficient to meet the mechanical disability of the damaged heart. A time comes when this hypertrophy ceases to be sufficient, whether from intrinsic failure of the heart-muscle or on account of some urgent call upon it such as excessive muscular exertion. The ventricle thereupon dilates, the auricle following suit. Congestion is established in the left chambers of the heart, causing an obstacle to the free discharge of blood from the pulmonary circuit. The lungs then become congested, throwing increased labour upon the right ventricle. This in its turn dilates. The right auricle suffers next; the return of blood from the systemic circulation is delayed; the systemic veins become engorged, the liver is distended by blood and

enlarged, and the whole body becomes the seat of a generalised dropsy.

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(*C*) **Valvular aneurysms.**—Aneurysms of the valves are due to ulcerative endocarditis, the inflamed valve yielding to the blood-pressure against it, and becoming permanently pouched. Occasionally when ulceration is extensive the pouched and weakened valve-cusp may rupture.

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1357*A*

## V. LESIONS OF THE CORONARY ARTERIES.

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The coronary arteries may be partially or completely occluded, by atheromatous degeneration in the first case, or by an embolus or thrombosis in the second. The effects of such occlusion have already been mentioned (*v.* pp. 63, 64). The disease known as "*angina pectoris*" is always associated with coronary lesions, usually atheroma.

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1374*A*

## VI. INTRA-CARDIAC THROMBI.

Ante-mortem clots are readily distinguished from those formed post-mortem or at the time of death (agonal clots) by their whitish colour and by their relatively firm adhesion to the walls of the heart. The following are the chief varieties.

1. **Flat thrombi.**—These may be local or general. They form a smooth lining to the chamber in which they occur, which is commonly a ventricle.

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1271*C*

2. **Thrombi in the auricular appendices.**—These are common in association with chronic dilatation of the auricles.

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3. **"Ball" or "bullet" thrombi.**—These have already been mentioned in connection with the auricular dilatation of mitral stenosis. The thrombus is perfectly round and smooth on the surface. It is but little laminated and usually contains an irregular central cavity.

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4. **Thrombi forming vegetations on diseased valve.**—These assume considerable proportions in ulcerative endocarditis. They may infect those portions of the auricular or ventricular walls against which they impinge during the movements of the heart.

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### Changes undergone by Thrombi.

1278 1. Simple softening.

1278*C* 2. Septic softening or suppuration.

3. Organisation, by which means they become an integral part of the structure to which they are attached.

1278*B* 4. Calcification. A loose thrombus may undergo calcification forming a cardiac stone or cardiolith.

1374*A* 5. Detached fragments of a thrombus may pass into the
1559*B* circulation causing embolism and infarction.
1564*A*

## VII. EMBOLISM AND INFARCTION.

Embolism is the impaction of a foreign substance in a vessel too small to allow of its passage. The obstructing body is the *embolus*. The chief sources of emboli are thrombi in the chambers of the heart or in the veins; but they may be derived from vegetations on the valves, or consist of calcareous or fibrinous particles from a diseased aorta, fragments of malignant growths, parasites, or fat cells. The area robbed of its blood-supply by an embolus is called an *infarct*.

The effects of embolism are: (1) mechanical (simple infarction); (2) specific, depending, that is to say, upon the quality of the embolus (septic infarction).

(1) **Mechanical effects of embolism.**—When an embolus suddenly occludes the main branch of a vital artery, death anticipates the appearance of secondary results. This is the case with embolism of the main branches of the pulmonary or
1564*A* coronary arteries.

When the branch is smaller or less vital the effect varies with the capacity of the part to develop a collateral circulation. When anastomoses are abundant, secondary thrombosis occurs on the proximal side of the embolus as far back as the nearest
1571*F* branch, the anastomosing channels become dilated and a
1571*H* collateral circulation is established. The occluded vessel may remain permanently obliterated, but occasionally the clot and embolus within it become channelled and the lumen restored.

When, however, the blocked vessel has no efficient anastomoses with its neighbours—a "terminal arteriole"—the area which it supplies becomes drained of blood; for the elasticity

of the arterial walls suffices to drive on the blood which is within them at the moment of occlusion. The anaemic area soon becomes necrotic, and is known as a "*white*" or "*anaemic*" *infarct.* Infarcts are roughly triangular in shape, the base being directed towards the periphery of the organ, and the site of occlusion forming the apex. The infarct is surrounded in the recent state by a hyperaemic zone.

In some viscera poorly supplied with anastomosing arterioles there is, nevertheless, a capillary anastomosis between neighbouring terminal arterioles. Dilatation of this capillary anastomosis may, upon the occurrence of embolism, allow the previously anaemic area to become flooded with blood which has behind it no arterial pressure to drive it onward into the veins. The infarct, therefore, becomes congested, swollen and purple in colour, and is known as a "*Haemorrhagic infarct.*"

**Microscopic examination** of an anaemic infarct shows that the cells composing it are necrotic and refuse to take the ordinary stains.

**Microscopic examination** of a haemorrhagic infarct shows a similar necrosis in conjunction with much extravasation of blood.

**Sites of infarcts.**—Primary white infarcts are seen in the brain and heart; haemorrhagic infarcts in the lungs, kidneys, spleen and intestine.

**Later changes in infarcts.**—All simple infarcts, whether originally haemorrhagic or not, in course of time lose their pigmentation and become white triangular areas of necrotic material surrounded by a fibrous capsule. Ultimately the site of a simple infarct may be marked by nothing more than a depressed cicatrix or a deposit of calcareous salts.

**Microscopic examination** of an infarct in the later stages shows a structureless hyaline substance lying in a capsule of fibrous tissue.

(2) **Specific effects of embolism.** (*a*) **Purulent infarction.**—When the embolus is infective, contains, that is to say, pathogenic organisms, as in the case of a particle of thrombus derived from an ulcerated valve, the infarcted area softens and becomes purulent. Such an area is called a purulent, septic, or pyaemic infarct. Purulent infarcts are associated most often with ulcerative endocarditis or with septico-pyaemia without endocardial lesions. In addition to its effect upon the infarcted area a septic embolus may produce an acute infective aneurysm of the artery at the point of its impaction.

Heart. 1295*B* Lung from the same case. 1746*C*

(*b*) **Other specific infarctions.**—When the embolus consists of a fragment detached from a malignant growth which has invaded an artery, or of living parasites, such as hydatid scolices, which have gained an entry into the arterial lumen, the result, in addition to mechanical infarction, is a metastatic development of the tumour or parasite at the points of impaction.

# CHAPTER VI.

## DISEASES OF THE BLOOD-VESSELS.

**A. Arteries.**—1. Normal Structure. 2. Hypertrophy and Atrophy. 3. Fatty Degeneration. 4. Inflammation, (*a*) Acute, (*b*) Chronic. 5. Primary Calcification. 6. Syphilis. 7. Tuberculosis. 8. Aneurysm. **B. Veins.**—1. Phlebitis. 2. Tuberculosis. 3. Varicose Veins. 4. Tumours.

## A. ARTERIES.

**1. Normal Structure.**—The wall of an artery consists of three layers, the Intima, the Media, and the Adventitia. In the smaller arteries and arterioles the Intima consists of a single layer of flattened endothelial cells; the Media wholly of muscle-fibres, while the Adventitia is reduced to a few connective-tissue and elastic fibres. In the aorta and the larger arteries the Intima consists of a fine connective-tissue with a few elastic fibres, lined by a layer of flattened endothelial cells, and separated from the Media by a relatively thick strand of elastic tissue, the fenestrated membrane of Henle, which in microscopic sections usually appears as an undulating line The Media in the aorta and larger arteries forms by far the largest part of the wall; it is composed of muscle-fibres and elastic fibres in varying proportions, the elastic fibres predominating in the largest vessels. The Adventitia consists of connective-tissue and elastic fibrils. It contains blood-vessels (Vasa Vasorum), a few muscle-fibres, and nerves. The blood-vessels penetrate

a short distance into the middle coat, but do not normally reach the Intima.

**2. Hypertrophy and Atrophy.**—Abnormal thickness of the walls of arteries occurs sometimes without known cause; with the advance of age there is often some thickening of the Intima; and with increased pressure in the arterial system, arising from any cause, there is often an hypertrophy of the muscular layer.

Abnormal thinning of the walls is found in persons dying of wasting diseases, such as pulmonary tuberculosis, and, it is said, in haemophilia also.

**3. Fatty degeneration** of the Intima of the aorta and larger arteries is one of the most frequent phenomena, and has little or no intrinsic importance. It shows itself in the form of streaks and spots of a yellowish colour, usually level with the general surface, but occasionally slightly raised above it. Such streaks are found in the bodies of quite young persons, and occur commonly in those dying of anaemia, marasmus, and cachexias of varying kinds. Fatty degeneration of the sub-endothelial layer of the Intima occurs also as an item of chronic arteritis.

Fatty degeneration of the Media occurs in association with that of the Intima, and under the same conditions.

**4. Inflammation.**—(*a*) Acute; (*b*) chronic.

(*a*) **Acute inflammation of Arteries** is generally due to one of two causes. (1) Some injury, wound, or ulcerative process invading the arterial walls from without. (2) The invasion and destruction of the arterial wall by an infective agent brought by the blood-stream.

(1) The best instances of the first variety are those due to the involvement of an artery in a spreading inflammation. In such an instance the usual phenomena of an acute inflammation are found; hyperaemia, exudation of lymph and leucocytes, and disintegration of the normal structures. As a result the
1439 arterial wall yields to the pressure of the blood within it, and
1439*A* sooner or later allows the blood to leak, or to pour out
1444 in a profuse haemorrhage. Secondary haemorrhages from suppurating wounds are due to such an acute arteritis.

(2) The second variety is most often occasioned by the
1538*C* embolism of infected particles derived from a distant focus,
1538*D* such as an ulcerated heart-valve, the result being an infective

aneurysm; but in rare instances the arteries, especially the 1505*A*
aorta, appear to be the primary seat of an infection.

(*b*) **Chronic arteritis.**—The variety of names which have been used to describe the chronic diseases of the arterial wall has added obscurity to a difficult subject. The following account includes the lesions which have been described from time to time under the names Arteriosclerosis, Arteriocapillary fibrosis, Atheroma, Chronic endarteritis, Obliterative endarteritis, Endarteritis deformans. These names have not all an identical significance, but all denote one or another phase of chronic disease of the arterial wall.

**Causes of chronic arteritis.**—The causes which give rise to the lesions of chronic arteritis are much disputed. It is agreed that gout, syphilis, chronic lead poisoning, chronic nephritis and the abuse of alcohol are predisposing causes, and to these many authorities add a permanent increase of blood-pressure in the arterial system, from whatever cause arising. Further, it is admitted that people of advanced age may exhibit the lesion in the absence of all these causes. But of the exciting cause nothing is certainly known. The fact that similar lesions occur in children and young adults who have died of acute infective diseases or nephritis, and the occurrence of chronic arteritis in association with prolonged anaemias and cachexias, lend support to the theory that the essential cause is some toxin which interferes with the nutrition of the vessel wall. But the influence of inheritance must not be forgotten. It is certain that in some families arterial disease is especially frequent, while in others the predisposing causes seem to be powerless. Hence Osler lays stress on the "bad material used for the tubing" as a predominant cause in many instances.

The factors concerned in the production of chronic arteritis may, therefore, be tabulated as follows:

(1) Inherited weakness of the vessel-walls.
(2) Chronic intoxications by such tissue-poisons as alcohol, lead, and syphilis.
(3) The poisons of some of the acute infections, notably that of pneumonia.
(4) The existence of prolonged high blood-pressure in the arterial system, whether from disease or laborious muscular exertion.

(5) Renal disease, which may act either by the production of a poison, or by the high blood-pressure which often accompanies it, or in both ways.

**The lesions.**—The lesions of chronic arteritis vary considerably with their situation, and to this variation is due the bewildering multiplicity of names.

In the aorta the disease is often diffuse, affecting especially
1446 the arch, but not sparing the descending, thoracic, and abdominal
1470 aorta. It is sometimes curiously partial; in some instances the aorta above the diaphragm is alone affected; in others the lesions are practically confined to the abdominal aorta; but as a general rule if the aorta is diseased in any portion of its walls the arch will show the most severe and wide-spread injury.

In smaller arteries the process is more often circumscribed, affecting especially those portions of the vessels where a branch is given off, or where a vessel divides. This type of arteritis
1529 is especially well seen in the arteries of the brain and in the
1529*A* coronary vessels of the heart, where the greater portion of a
1374 vessel may be apparently healthy, but at certain situations the wall is thickened by opaque yellow or white plaques or rings.

These varieties of appearance have given rise to the division into Diffuse and Circumscribed or Nodose Arteritis, names which do not connote any essential difference in the lesion.

The diseased areas, whether diffuse, or circumscribed, are in
1418*A* the earliest stages transparent and of a gelatinous consistency, but later become white or yellow in colour, firm to the touch, and slightly raised above the surface of the inner wall of the vessel. In the smaller arteries they may project so far as to occupy half or more of the lumen. In the aorta these areas may occupy the entire surface, but more frequently appear in strips and patches with healthy tissue intervening. In some of the patches the degenerate tissue is softened, and these bear a resemblance to small abscesses, which has given them the
1426 name of "*Atheromatous abscesses.*" Where the softened Intima has given way, and allowed the escape of the fluid débris, the muscular wall is exposed at the base of a shallow breach,
1432 to which a thrombus often adheres. Such a breach is styled
1426*A* an "*Atheromatous ulcer.*" These ulcers often form the
1428 starting point of a sacculated, or a dissecting, aneurysm. In addition to these lesions the Intima of the vessels often shows
1503*A* scarring, due to the formation of fibrous tissue in the degenerate

areas, and numerous calcareous plaques due to the infiltration 1420
of the degenerate tissue with lime salts. 1421

**Microscopic examination.**—The changes in the Intima consist of two elements. (1) Fatty degeneration, and (2) proliferation of connective-tissue. To these two essential processes are added infiltration of the tissues by inflammatory exudates, together with swelling and hyaline degeneration of the connective-tissue fibrillae. The earliest recognisable change takes place in the connective-tissue which lies immediately below the intimal endothelium. Here the two processes of fatty degeneration and connective-tissue proliferation go on separately or together, with the result that the thickness of the inner wall is considerably increased. The new formation consists either of necrotic and fatty detritus with crystals of cholesterin and of fatty acids, or of proliferated cells, arranged in layers, some or all of which have undergone hyaline degeneration. Where the fatty degeneration predominates a small pocket is formed containing a creamy fluid (Atheromatous abscess), the evacuation of which leaves an Atheromatous ulcer. When proliferation predominates there is in addition to the thickening of the sub-endothelial layers, a new formation of elastic fibres, and an infiltration of the new tissue with leucocytes and round cells. In either case the changes are not confined to the Intima; the Media and Adventitia suffer as well; the Media by the destruction and twisting of its elastic fibres and an exudation of inflammatory fluid and cells; the Adventitia by the massing of inflammatory cells, especially around the Vasa Vasorum.

In the later stages fibrous changes leading to contraction and scarring, and deposition of calcium salts leading to the formation of calcareous plates, occur in the diseased areas. There is a difference of opinion as to the coat which is first affected. Some authorities hold that the initial lesion is an arteritis of the Vasa Vasorum leading to a deficient supply of blood to the Media and consequent weakening of this coat. According to this view the intimal thickening is secondary, and is to be considered an effort to give additional strength to the weakened spot. Other authors believe that the Intima suffers first owing to direct exposure to a poison circulating with the blood.

So far then as concerns the aorta and arteries of moderate size, the series of changes may be set out thus:

(1) Thickening of the Intima with the formation of transparent gelatinous elevations, generally circumscribed.
(2) The areas of intimal thickening become opaque, yellowish in colour, and tend to spread and coalesce; they may be flush with the normal Intima, but are more often slightly raised.
(3) Atheromatous abscesses.

(4) Atheromatous ulcers.
(5) Fibrous scars.
(6) Calcareous plaques.

In the arterioles and smaller arteries of the limbs and viscera, chronic arteritis often produces somewhat different results; especially when the arteritis is associated with renal disease. Here the changes are seated principally in the middle coat, though the Intima often shows proliferation of its endothelium. The middle coat undergoes hypertrophy, with which is associated an increase in the connective-tissue of the arterial wall in general. An affected arteriole in the kidney, for example, will possess a muscular wall twice or three times its normal thickness, and showing, both intermingled with the muscle fibres and also surrounding the muscular layer, a number of circular strands of fibrous tissue.

This is the change originally described by Gull and Sutton as "Arterio-capillary fibrosis." Since its most prominent consequence is a narrowing of the arterial lumen, even to complete obliteration, it is sometimes spoken of as an "obliterative arteritis." The thickened muscular wall is apt to undergo atrophy and hyaline degeneration.

**Effects of chronic arteritis.**—The sequelae of chronic endarteritis are important. In the first place owing to the weakening of the walls aneurysms frequently occur. Secondly, owing to the narrowing of the lumen of the arteries the nutrition of the tissues is likely to suffer; and if, in addition, thrombosis occurs within the narrowed lumen, anaemic infarction or gangrene may follow. Thrombosis of the cerebral arteries in such cases leads to cerebral softening; of the coronary arteries to myomalacia cordis.

1434-1438 **5. Primary calcification of the Media.**—In addition to the calcification which occurs as a secondary phenomenon in chronic endarteritis, there is a somewhat rare disease, in which the Media becomes infiltrated with calcium salts. It is found principally in the femoral and tibial arteries of old people, but occurs also in the arteries of certain viscera, especially the uterus. An artery thus affected is hard, like a clay-pipe stem. The calcareous deposit is arranged in rings round the entire circumference of the vessel, the rings resembling in miniature the arrangement of the cartilage-rings of the trachea. In most cases the Intima is unaffected, but in some

there is evidence of a proliferative thickening. The calcification appears to commence in the Media, which has undergone fibrous and hyaline degeneration, but in some instances the elastic fibres of the Adventitia are the first to suffer. Senile gangrene of the feet is occasionally due to this primary calcification of the tibial arteries.

6. **Syphilis.**—Syphilitic arteritis of the smaller arterioles occurs either as a widely-spread affection, or as a local phenomenon in the neighbourhood of gummata. In the first case the lesion is most often observed in the arteries of the brain and meninges. The Intima Media, and Adventitia are all involved, but the most characteristic change is intimal—a **proliferative endarteritis**. The Intima is converted into many layers of round or spindle-shaped cells, which narrow and at times obliterate the lumen of the vessel. In arteries affected for a long time this active cell-proliferation gives place to a more fibrous thickening. The changes in the media and adventitia are similar to those in the intima. It is to be observed that there is nothing specific in these changes. It is not possible to diagnose that an arteritis is of syphilitic origin from the appearances. At the same time it is unusual in other conditions to meet with so high a degree of proliferation either in the intima or adventitia. Further, in syphilis there appears to be little or no tendency to fatty degeneration such as is common in other forms of chronic arteritis. Syphilitic arteritis of the aorta leads to the formation in the Intima of gelatinous semi-transparent nodules of hyaline degeneration, 1418*A*
which have a special tendency to become fibrous, thus causing contraction and giving a scarred appearance to the surface. The media and adventitia are greatly altered; the media by invasion of new vessels and inflammatory cells, which cause destruction of the elastic fibres and are in time replaced by fibrous tissue; the adventitia similarly by the formation of fibrous tissue, and by obliterative endarteritis of the vasa vasorum.

7. **Tuberculous arteritis.**—Tuberculosis seldom attacks arteries unless as a result of extension of the disease into their walls from the surrounding tissues. In this case one finds a periarteritis in which definite anatomical tubercles can be detected, accompanied by a proliferative endarteritis 2458*E*
identical in appearance with that seen in syphilis. In the

lung the arteries resist the destructive inflammation excited by the tuberculous process longer than the other tissues, so
1724*A* that a cavity is frequently traversed by an artery; in
1718$C_2$ many cases, fortunately, thrombosis takes place in the artery before its wall is completely destroyed, but where this is not so the blood-pressure often leads to rupture and profuse haemorrhage.

In acute miliary tuberculosis it is possible to find small miliary tubercles situated in the intima of the larger arteries, with little or no evidence of periarteritis or mesarteritis, but such appearances are rare and of little pathological importance.

8. **Aneurysm.**—An aneurysm is a circumscribed dilatation of an artery. There are three main anatomical varieties: (1) fusiform, *i.e.* involving the whole circumference of the vessel; (2) sacculated, *i.e.* a bulging of the vessel wall in some particular spot; (3) dissecting, *i.e.* the sac is formed by the separation of the coats of the artery, blood being driven between them. Besides these well-marked varieties there occur instances in which the whole extent of a vessel, or one definite
1446 portion of it, is dilated; for example, the whole thoracic, or
1447 abdominal aorta may be dilated, the circumference of the vessel being nearly twice its natural dimensions. Such diffuse dilatations occur nearly always in the aorta, or in one of the larger arteries; in the iliac arteries such a dilatation is often accompanied by tortuous curves in the course of the artery. In some cases this diffuse aneurysmal dilatation is not uniform, but is rather a succession of fusiform aneurysms occurring at
1531*A* frequent intervals, so that the vessel comes to resemble a string of large beads. The intima of the affected vessel may be quite natural, or may exhibit all the changes of chronic endarteritis, but the essential lesion in such cases appears to be always in the media, and to consist of fibrous degeneration of the muscular and elastic fibres.

**Constitution of the Sac.**—The sac of an aneurysm may consist of the whole of the wall of the artery; of the media and adventitia; of the adventitia only; or, lastly, of the surrounding tissues, condensed by the pressure exercised upon them by the aneurysm. The sac always contains blood, which may be still fluid, or in various stages of coagulation. In a fusiform aneurysm there may be little or no clotting, for the blood-

flow through it is always rapid; but in a sacculated aneurysm the whole or a portion of the sac may be filled with a clot, which usually presents a laminated arrangement due to 1548*A* the varying age of the deposits, the outermost layers being 1548*B* firm, dry, and white in colour, while those more recently deposited are red, soft, and moist. In the older decolourised layers it is not unusual to find deposits of calcareous matter.

**Varieties of aneurysm. Fusiform aneurysm.**—A fusiform aneurysm is a dilatation of the whole circumference of the vessel; its walls therefore consist of all three coats, and its 1450*A* cavity is continuous at either end with the normal vessel. Besides the widening there is always some elongation of the dilated part of the artery. Aneurysms originally fusiform often become sacculated; the wall of the fusiform dilatation 1484*B* yields unequally, and localised diverticula (sacculated aneurysms) result. Fusiform aneurysms are often large, their cavities seldom contain laminated clot, and their most frequent situation is in the aorta, and its primary branches.

**Sacculated aneurysm.**—A sacculated aneurysm occurs where a part of the circumference of the artery yields to the blood-pressure. The sac sometimes consists of all three coats, but 1454 more often the intima is entirely destroyed, and the sac-wall is formed by the media and adventitia, by the adventitia only, or when this too has perished, by the condensed tissues in the neighbourhood. Not infrequently secondary sacculi are developed in the wall of the original aneurysm, thus giving rise to loculated aneurysms. 1484*A*

Sacculated aneurysms of the aorta and large arteries are of very various sizes, from tumours not much bigger than a pea, to those as large as a foetal head. In the arteries of the brain such aneurysms are often of microscopic size and multiple; in 1527 this situation they are known as "miliary aneurysms."

The sac of a sacculated aneurysm, communicating with the artery by a narrow opening, as is often the case, usually contains a certain amount of laminated clot. When, as some- 1548 times happens, this clot has completely filled the cavity, the 1548*A* aneurysm is said to have undergone spontaneous cure.

**Dissecting aneurysm.**—A dissecting aneurysm is formed by the separation of the coats of an artery, usually the inner and middle ones, under the pressure of blood extravasated between them through a rupture of the intima. A dissecting aneurysm 1464*A*

therefore, has not a sac in the ordinary sense of the word. The blood which escapes into the division between the coats may
1505*A* leave the vessel altogether, may coagulate in its new position,
1464*E* or may return to the normal channel at a point further along the vessel.

Dissecting aneurysms are chiefly formed in the aorta and its largest branches, but are also met with occasionally in much smaller vessels, for example in the arteries of the brain.

**Aneurysmal varix and varicose aneurysm.**—*Aneurysmal varix* is the name given to a direct communication between an
1462 artery and a vein; *varicose aneurysm* indicates a communication between an artery and a vein through the medium of an aneurysmal sac. Both lesions are most commonly found as the result of punctured wounds affecting the arteries of the limbs.

**Causes of aneurysms.**—The causes of aneurysm are: (1) chronic arteritis; (2) infective processes, whether attacking the artery from a focus of inflammation situated in the neighbourhood (*e.g.* pulmonary tuberculosis), or originating within the lumen (*e.g.* infective embolism); (3) trauma.

**Racemose aneurysm.**—The so-called "racemose aneurysm" has in reality no claim to the title of aneurysm. It consists of a number of dilated and tortuous vessels, and is more properly
2466*C* entitled a "racemose angeioma."

**Spontaneous cure of aneurysm.**—The spontaneous cure of an aneurysm may take place as a result of obliteration of the
1548 sac by laminated clot, or through the coagulation in bulk of
1548*A* the blood within the sac, *i.e.* by the formation of "active" or of "passive" clot. In such circumstances the aneurysm becomes converted into a solid mass, which in process of time is organised into fibrous tissue. Spontaneous cure is rare in the case of the large arteries, but is seen occasionally in
1454 sacculated aneurysms of the arch of the aorta. In large fusiform aneurysms of the aorta it never occurs.

Rupture of an aneurysm is the most common fatal termination of the disease.

**Aneurysms of the ascending aorta rupture—**

*Most commonly—*

(1) Into the pericardium. **1480***A*. **1464***C*.
(2) Into the pleural cavity. **1487**.

(3) Into the vena cava superior. **1490.**
(4) Externally. **1454***B*. **1487***B*. **1496.**

*Occasionally—*
(5) Into the right ventricle. **1473***A*.
(6) Into the right auricle. **1472.**
(7) Into the pulmonary artery. **1476***A*. **1485.**

**Aneurysms of the arch rupture—**
(1) Into the trachea. **1500.**
(2) Into a bronchus, usually the left. **1499.**
(3) Into the oesophagus. **1502***A*. **1502***B*.
(4) Into the pleura.
(5) Into the lung.
(6) Externally.
(7) Into the pulmonary artery.

**Aneurysms of the descending thoracic aorta rupture—**
(1) Into the pleura.
(2) Into the oesophagus.
(3) Into the stomach. **1505***A*.
(4) Into the mediastinum. **1505.**

**Pressure effects of aneurysm.**—The gradual increase in size of an aneurysm, especially of an aneurysm within the thoracic cavity, leads to constant pressure upon the surrounding parts. The effects of such pressure vary with the character of the tissues which lie adjacent to the sac.

(1) Bones and cartilage undergo erosion. The cartilages of the ribs, the bony ribs, and the sternum are thus frequently pressed upon and perforated by aortic aneurysms. The vertebrae also not infrequently suffer in this way, the gradual disappearance of the vertebral bodies, which suffer more than do the intervertebral discs, sometimes exposing the spinal cord to pressure and giving rise to paresis of the lower limbs. 1484*B* 1454*B* 1487 1502*A* 1532*B*

(2) Pressure upon blood-vessels leads to constriction of their lumen, stagnation of the blood-stream, and subsequent thrombosis. An aneurysm may lead to its own spontaneous cure by the pressure it exercises upon the artery serving it, a phenomenon most often observed in aneurysms of the femoral or popliteal arteries. 1472

(3) Pressure upon nerves often gives rise to much pain, and

sometimes to paralysis. An aneurysm of the arch of the aorta is peculiarly likely to press upon the left recurrent laryngeal
1454*C* nerve, causing abductor paralysis of the larynx.

(4) Pressure upon the trachea and bronchi may lead to
1454*A* difficulty of respiration in the case of the trachea, or in the
1455 case of the left bronchus to collapse of the lung with subsequent
1483*A* bronchiectasis. Pressure upon the lung itself may lead to condensation of the portion affected, and, rarely, to gangrene.

1484*B* (5) Pressure upon the oesophagus or thoracic duct is occasionally fatal, from inability to swallow food in the one case, and from obstruction to the entry of chyle into the blood-stream in the other.

## B. DISEASES OF THE VEINS.

The veins are liable to inflammations and degenerations analogous to those met with in the arteries. Thus fatty degeneration, fibrosis and calcification are not uncommon, but are not as a rule important. The lesions of moment are:

(1) Acute inflammation (Phlebitis).
(2) Tuberculosis.
(3) Dilatation (varicose veins).
(4) Tumours.

1. **Phlebitis** may be primary or secondary.

(*a*) An apparently **primary phlebitis** is not uncommon among middle-aged and elderly persons, especially those who are subject to gout, and in persons of all ages who are in
1589 a debilitated condition. It usually attacks the veins of the
1599 lower extremity.

(*b*) **Secondary phlebitis** depends upon (1) direct injury to the vein, (2) involvement of a vein in an inflammation existing in its neighbourhood, (3) infection of a vein by blood derived from a distant focus of inflammation. The commonest examples of the second class are the phlebitis of the lateral
1593 sinus which attends otitis media, and that of the pelvic veins
1594 associated with puerperal sepsis. Of the third class, pylephlebitis (*i.e.* phlebitis of the portal vein), occurring as a consequence of active inflammation in the intestines or appendix vermiformis may be taken as a type.

**Effects of phlebitis.**—The effects of phlebitis are in part mechanical and in part depend upon the nature of its cause. In every case blood tends to clot upon the inflamed wall of the vein (thrombosis), until the lumen is occluded. In consequence 1578*A*
the venous return is obstructed to a degree proportional to the 1578*C*
importance of the vessel affected, and the result is a corre- 1588*A*
sponding congestion and oedema of the district served by the occluded vessel. As is the case with occluded arteries, a "consecutive thrombosis" occurs in the affected vein on either side of the lesion, between the latter and the nearest important tributary. It may happen that a portion of this thrombus becomes detached and entering the blood-stream produces an embolism of some distant arteriole. Emboli of this order, when derived from the systemic veins will traverse the right side of the heart and produce embolism of branches of the pulmonary artery and infarction of the lung. Emboli derived from the veins of the portal system will produce a similar result in the liver.

So much for the purely mechanical effects of phlebitis and of the thrombosis which is its almost constant accompaniment. But if the phlebitis is due to pyogenic organisms the thrombus in the vein is an "infective thrombus" and tends to become 1583
softened and purulent. The consequences in this case depend upon the extent to which consecutive thrombosis on the proximal side of the lesion serves to limit the spread of the infective material. If the path from the latter to the general blood-stream is well guarded by a considerable and firm "consecutive thrombus," the purulent material is confined, and forms a local abscess. Otherwise portions of it are swept into the general blood-stream giving rise to purulent infarction, and metastatic abscesses at the site of their lodgment.

2. **Tuberculosis.**—The importance of tuberculosis of the veins lies in the ease with which tubercle bacilli may gain entrance to the blood-stream as a result of rupture of a caseous tubercle into the lumen of a vein.

3. **Varicose veins.**—Varicose dilatation of veins is met with in a variety of situations. As regards the systemic system, the long veins of the lower extremities and those of the pampiniform plexus (varicocele) are the most liable to this affection. Varicosity of the veins of the haemorrhoidal plexus (which forms a link between the systemic and the portal veins

contributing to both) gives rise to piles. Varicosity in the portal system is commonest in the stomach and lower part of the oesophagus.

The causes of varicosity are not entirely understood, but in general terms any obstruction to the venous return is productive of it. Thus prolonged standing, the pregnant uterus and all abdominal tumours of any size are common causes of varicosity of the veins of the lower extremities. Cancer of the colon or rectum, and cirrhosis of the liver are often respectively attended by piles and by varicose dilatations in the stomach or oesophagus. But commonly enough varicose veins in the lower extremities, piles, and varicocele appear in the absence of an identifiable cause, seeming then to be due to an inherent weakness in the vein-walls.

1575 Varicose veins are tortuous and thickened. In situations
1576*D* where they are exposed to injury they often become inflamed
1576*E* and occluded by thrombi, the subsequent organisation of which converts them into fibrous cords. Ulceration may attack them and penetrate their walls, or the latter may be absorbed by the pressure of the blood within. In either case destruction of the wall will lead to haemorrhage.

**4. Tumours.**—Malignant tumours penetrate the walls of veins with some difficulty, but having done so, tend to grow
1606*A* along the lumen with great rapidity. The detachment of
1606*C* portions of such intra-venous growths with their subsequent lodgment as emboli in distant parts, is a fruitful source of metastatic deposits of malignant disease.

# CHAPTER VII.

## DISEASES OF THE LARYNX.

I. **Laryngitis.**—(*A*) Acute: 1. Simple. 2. Membranous 3. Typhoid. 4. Septic. (*B*) Sub-acute or Chronic: (*a*) Infective: (1) Tuberculous; (2) Syphilitic; (3) Leprous. (*b*) Non-infective: (1) Pachydermia Laryngis; (2) Chorditis Tuberosa; (3) Gouty Laryngitis. II. **Tumours of the Larynx.**—(*A*) Innocent. (*B*) Malignant. III. **Oedema of the Larynx.**

### I. LARYNGITIS.

(*A*) **1. Acute simple laryngitis.**—(Syn. Spasmodic laryngitis, spasmodic croup, catarrhal croup, laryngitis stridulosa.)—Acute simple laryngitis is a common associate of acute coryza. In adult life the condition is of little moment, but during infancy assumes a greater prominence owing to the minute size of the rima glottidis at this time of life, and the frequency with which spasm of the glottis complicates the laryngeal catarrh. The lesion is an inflammation of the mucous membrane above the vocal cords, producing in the first instance dryness, hyperaemia and swelling, and followed by an increased secretion of mucus. This variety of laryngitis is a common symptom of the invasion-period of measles.

**2. Membranous laryngitis.**—In a vast majority of instances membranous laryngitis is caused by the diphtheria bacillus, though other organisms are very occasionally responsible. For present purposes we may consider membranous laryngitis as synonymous with diphtheritic laryngitis.

**Diphtheria** may attack the larynx in the first instance, or, as happens far more often, involve the larynx by extension from

the fauces. The characteristic pathological feature of the disease is the formation of a membrane on the affected mucous surface. In uncomplicated cases this membrane is white or grey, but may present all degrees of discolouration when the infection is complicated by other organisms, such as those of the streptococcus group. The typical membrane is a tough pellicle firmly attached to the subjacent tissues, which bleed upon its removal. It is composed of degenerated epithelial cells, leucocytes and fibrin in varying proportions, and contains within its substance quantities of diphtheria bacilli, and often of other organisms. Its consistency is dictated by the bulk of its fibrin element. When this is abundant the membrane forms a perfect cast of the surface to which it is applied; otherwise the affected area has a granular necrotic aspect, and
1616 though still technically "membranous" presents no coherent
1616*D* pellicle. In all cases the deeper layers of the mucosa become
1616*E* infiltrated and swollen by inflammatory exudations of lymph
1616*F* and leucocytes.

It is a rare thing to find the larynx involved without evidence of membrane upon the tonsils; but the lesion may be limited to any part of the air-passage from the larynx upwards, or, on the other hand, may involve it all, from the nose above to the
1616*A* smallest bronchi below. In the latter event complete mem-
1616*B* branous casts of the bronchial tree may be recovered after
1616*C* death, or more rarely, discharged through a tracheotomy wound
1616*G* during life. When the glottis or trachea are invaded, the presence of membrane combined with the associated inflammatory swelling of the underlying tissues, results in obstruction of the air-way, and a corresponding degree of dyspnoea. In young subjects this is often extreme enough to call for tracheotomy or intubation.

**3. Typhoid laryngitis.**—The larynx suffers with moderate frequency in the course of typhoid fever. There are two varieties of the lesion, either of which may appear alone. These
1641$A_2$ are: (1) superficial ulceration, most often appearing on the
1641*D* epiglottis or on the aryteno-epiglottic folds; and (2) perichondritis of the laryngeal cartilages, attacking the cricoid most frequently. The ulceration is superficial and leaves no scarring. The perichondritis, if complicated by infection with pyogenic
1641*B* organisms, may end in necrosis of the affected cartilage, which
1641*C* then lies loose in an abscess-cavity formed by the detached

perichondrium. This event leads to much cicatricial contraction.

**4. Acute septic laryngitis.**—Under this title may be considered a number of acute infections of the larynx, sometimes occurring alone, sometimes in association with exanthematous fevers, particularly the malignant types of scarlet fever and variola. The condition is not an isolated affection of the larynx, the latter merely sharing in a general invasion of the upper air and food passages. The appearance of the larynx under these 1615*B*
conditions is one of diffuse and vivid injection, accompanied by 1837*D*
oedema of the glottis, often extreme, and by ecchymoses of 1837*E*
varying number and extent. When the haemorrhages are extensive the mucous membrane assumes a deep purple colour. Acute perichondritis, with resulting necrosis of the laryngeal 1643
cartilages, occasionally attends these infections as it does that 1644
of typhoid fever. The organisms most often responsible for this condition are those of the streptococcus group.

(*B*) **Sub-acute or chronic laryngitis.** (*a*) **Infective.**—(1) **Tuberculous laryngitis.**—Tuberculosis of the larynx may be either primarily laryngeal, or secondary to pulmonary tuberculosis. Of these varieties the second is by far the more usual.

**Primary laryngeal tuberculosis** occurs in the form of circumscribed ulcers upon the surface of the epiglottis, and tends to spread slowly downwards towards the larynx. In course of time the epiglottis may be entirely destroyed, but elsewhere the ulceration is commonly superficial.

**Secondary laryngeal tuberculosis** presents the following lesions occurring alone or in combination: (1) ulceration; (2) submucous infiltration; (3) local tuberculous tumour.

(1) Tuberculous ulceration may be either superficial or deep. Superficial ulcers are generally unilateral and situated on a 1631*C*
vocal cord or ventricular band, but they may occur on the 1631*D*
epiglottis and aryteno-epiglottic folds. Deep ulceration follows 1631*F*
the disintegration of local tuberculous tumours, or of the sub-mucous infiltrations to be next described. It is often impossible to distinguish, by the naked eye, between deep ulceration due to tuberculosis and that due to syphilis.

(2) Submucous tuberculous infiltration affects most often the extrinsic parts of the larynx, viz. the arytenoids, the aryteno- 1632*A*
epiglottic folds and the epiglottis. These parts become in 1633*B*

consequence oedematous and swollen, often without any evidence of ulceration. In course of time, if the disease is not arrested, the infiltrated areas may undergo softening, which results in deep and extensive ulceration as described above. In both cases pronounced cicatrisation is common.

(3) Local tuberculous masses appear most commonly as pale rounded projections from the posterior surface of the larynx at the inter-arytenoid fold. They may remain unchanged for years and finally disintegrate, or, more rarely, disappear spontaneously.

(2) **Syphilitic laryngitis.**—Primary syphilis of the larynx is unknown. In the secondary phase the larynx shares in the faucial erythema and may be the seat of mucous patches. The tertiary lesions are common and important.

Tertiary syphilitic laryngitis usually appears at first as a diffuse swelling of the epiglottis and arytenoids, due to gummatous infiltration. The tendency of this deposit is towards disintegration. The gumma in consequence softens, and after eroding its mucous covering and discharging its
1627*A* contents, becomes a gummatous ulcer. Such an ulcer in its
1628 typical form has a "punched-out" appearance, with sheer edges and a grey and sloughy base.

The laryngeal cartilages are occasionally attacked by a gummatous perichondritis which may end in the necrosis of the cartilage concerned. These gummatous lesions, if extensive and untreated, leave much deformity. The epiglottis is often completely destroyed and the laryngeal chamber narrowed to a dangerous extent by the gradual shrinkage of cicatricial tissues in its neighbourhood.

Congenital syphilis seldom causes ulceration of the larynx.
1630*A* When it does so the lesions are anatomically similar to the tertiary lesions of the acquired disease. On the other hand a catarrhal laryngitis is a frequent accompaniment of the earlier manifestations of congenital syphilitic infection.

(3) **Leprous larnygitis.**—In the course of leprosy the larynx may be attacked. The epiglottis and aryteno-epiglottic folds then become the seat of a diffuse nodular infiltration. All the laryngeal tissues become greatly thickened and finally involved in a slow ulceration. Extensive destruction is uncommon since the death of the patient from the general affection usually
1624*A* anticipates it.

(*b*) **Non-infective laryngitis.** (1) **Pachydermia laryngis.**—Syn. Alcoholic laryngitis.

Habitual over-use of the voice combined with faulty production of it often results in a thickening of the epithelium of the vocal processes and inter-arytenoid space. These over-growths form greyish-pink elevations which in cases of long-standing become almost horny in appearance and consistency. The synonym of the disease, viz. alcoholic laryngitis, is not always deserved, but the lesion is particularly prone to attack those addicted to a free use of alcohol, such, for example, as the market porters of Smithfield. 1634*A* 1634*B*

(2) **Chorditis tuberosa.**—Syn. Singer's nodes. This variety of laryngitis is found particularly among singers, or others who use their voices much, as school-mistresses. The lesion consists of small nodules occupying the free margins of the vocal cords at the junction of their anterior and middle thirds. Both cords are usually attacked, and the growths appear as small white excrescences. 1634*C*

Histologically the nodules consist of heaped-up keratinised epithelium with some hyperplasia of the underlying connective-tissues.

(3) **Gouty laryngitis.**—Gouty subjects sometimes present a chronic form of laryngitis characterised by redness, thickening and dryness of the cords, ventricular bands, and inter-arytenoid space. Gouty deposits of sodium salts are occasionally to be observed about the vocal cords and in the crico-arytenoid joints. 1611*A*

## II. TUMOURS OF THE LARYNX.

(*A*) **Innocent.**—Almost any variety of innocent tumour may be found occasionally attacking the larynx, but the commonest are papillomata, fibromata, and cysts. 1645*B* 1646 1647

(1) Papillomata may be single or multiple, and may attack any part of the larynx. 1648 1650

(2) Fibromata usually occur as single sessile projections from one vocal cord. 1650*A*

(3) Simple cysts containing a mucous fluid are occasionally met with on the epiglottis, particularly on its lingual surface.

(*B*) **Malignant.**—The larynx may be the seat either of carcinoma or sarcoma, but the commonest variety of malignant new-growth in this situation is squamous-celled carcinoma. It is usual to sub-divide the malignant neo-plasms of the larynx

according to their position, into (1) the *Intrinsic*, and (2) the *Extrinsic* groups.

(1) Intrinsic growths are those which commence in the vocal cords, ventricular bands or ventricles. This group is characterised by a relatively slow progression and deferred involvement of lymphatic glands. In early stages the growth is unilateral and may appear either as a small indolent ulcer, or as a papillomatous projection, or merely as a thickening of the cord, either localised or diffuse. As the disease advances the
1656*F* lesion assumes the characteristics of an epitheliomatous ulcer,
1656*G* *i.e.* an ulcer with thick and everted edges and a grey foul floor.

1653 (2) Extrinsic growths affect the epiglottis, aryteno-epiglottic
1655*A* folds and arytenoid cartilages. In this class the involvement
1656$C_2$ of lymphatic glands is an early feature and the disease advances rapidly, causing much infiltration and destructive ulceration of neighbouring parts. Death, when not due to suffocation, results most often from septic broncho-pneumonia brought about by aspiration of discharges from the ulcerated surface.

## III. OEDEMA OF THE LARYNX.

Generalised oedema of the larnyx may complicate any severe inflammation of the glottis or its neighbourhood: it may thus
1615 be associated with any variety of laryngeal ulceration, with destructive injuries, such as the inhalation of steam or the
1615*A* swallowing of corrosive poisons, and with any form of exten-
1615*B* sive cellulitis in the neck, such as that known as Ludwig's angina. In addition to these local causes oedema of the larynx may be attributable to certain constitutional disorders of which the chief are nephritis, and the vasomotor disturbance called angeio-neurotic oedema. It may also complicate heart-failure from valvular disease, and the advanced stages of grave anaemias.

Whatever its cause oedema of the larynx presents the same general appearances. It appears first in the extrinsic portions of the larnyx and subsequently involves the whole. The mucous membrane becomes tumid, pale and semi-translucent, and may in extreme cases reach a stage of general swelling which entirely occludes the airway

# CHAPTER VIII.

## DISEASES OF THE TRACHEA.

(For malformations, see section on Oesophagus.)

A. **Ulcerations.**—1. Tuberculous. 2. Syphilitic. 3. Malignant. B. **Stenosis.**—1. From external pressure. 2. Cicatricial. C. **Foreign Bodies in Trachea and Bronchi.**

### A. ULCERATIONS.

1. **Tuberculous ulceration** of the trachea may complicate pulmonary tuberculosis. The lesion consists of multiple superficial ulcers on the mucous surface, the edges of the ulcers being undermined, as is the rule with tuberculous ulcerations. 1633*F* 1633$F_2$

2. **Tertiary syphilitic ulceration** may attack the trachea as well as the larynx, and produces similar lesions in either situation, viz. much destruction during its activity, and subsequent cicatricial deformity to a proportionate extent. The latter may lead to dangerous stenosis of the trachea.

3. **Primary malignant disease** of the trachea is rare enough to be negligible: but it is not unusual to find a cancerous ulcer of the trachea due to the spread of squamous-celled carcinoma from the oesophagus. In such a case the trachea and oesophagus become united by the growth, and the tracheal wall is replaced by a fungating ulcer. Ultimately perforation may occur, resulting in the passage of food-stuffs through the fistula into the trachea, and consequent septic bronchopneumonia.

4. Any cause of *prolonged pressure* upon the tracheal wall may lead to the formation of an ulcer; *e.g.* aneurysm, enlarged glands, or new growth. 1844*D*

## B. STENOSIS OF THE TRACHEA.

1. The trachea may be narrowed by pressure from without. The most frequent causes of this lesion are :

(*a*) Aneurysms. **1507.**

(*b*) Enlarged glands, tuberculous, lymphadenomatous or malignant.

(*c*) Massive goitres. **2310.**

(*d*) Malignant growths. **2309***B*.

2. As mentioned above tertiary syphilitic ulceration occasionally results in cicatricial stenosis of the trachea.

## C. FOREIGN BODIES IN THE TRACHEA AND BRONCHI.

1662 Foreign bodies of small size occasionally traverse the rima
1662*A* glottidis. Of these the most common are, in the case of
1662*B* children, such objects as glass beads and dried peas, and in the case of adults, teeth extracted during anaesthesia. The effect of such an accident varies with the size, shape, and nature of the foreign body. A pointed article may become impacted in the trachea, but otherwise foreign bodies generally fall into the right bronchus since it is larger than its fellow and more directly in the line of the trachea. Spherical bodies soon become impacted in a tube of suitable size, especially when, as in the case of a pea, they are hygroscopic and swell amid their moist surroundings. Bodies of irregular shape and of unabsorbent material may remain loose for some time, and
1681 displaceable by the act of coughing. Ultimately, however,
1681*A* impaction occurs, with resulting lesions of two kinds; firstly,
1681*B* ulceration at the site of lodgement; secondly, septic broncho-
1681*C* pneumonia in the area of lung below. When occlusion is complete there results in the first instance collapse of the area of lung served by the occluded tube, and presently ulceration at the point of impaction. This ulceration may loosen the impacted body and permit of its displacement by coughing. More commonly such displacement does not occur, but the resulting inflammatory exudations gravitate into the finer tubes, producing septic broncho-pneumonia in their course. The naked-eye appearances of a lung so affected may be very similar to those of tuberculous pneumonia, the vesicular tissue

being entirely replaced by whitish cheesy material, dotted with cavities, some representing bronchiectatic dilatations of the air-tubes, others situated in the lung-tissue and produced by gaseous products of bacterial fermentation. When the impacted body does not completely fill the tube in which it lies the chain of processes remains substantially the same, except that the initial phase of collapse is absent, and the secondary broncho-pneumonic consolidation is not so massive.

# CHAPTER IX.

## DISEASES OF THE PLEURA.

I. Pleurisy. II. Pneumothorax. III. Haemothorax. IV. Chylothorax. V. Tumours.

### I. PLEURISY.

**General considerations.**—Inflammation of the pleura may be a primary lesion, or secondary to disease elsewhere, whether of the thoracic contents or of some distant portion of the body. Clinically considered, pleurisies fall into two large groups, one of them marked by an inflammatory exudate small in bulk and rich in fibrin, the "dry pleurisies"; the other by a more or less copious liquid exudate—the "pleurisies with effusion." The latter are further divisible according to the character of the effusion into the "serous pleurisies," and the "purulent pleurisies" or "empyemata." But the line of demarcation between these groups is not so easily drawn in the light of morbid anatomy, for inflammatory exudates present in different cases all gradations of consistency from the purely fibrinous to the definitely fluid, while the same cause may produce in one part of the pleural sac a dry pleurisy, and in another a pleurisy with effusion. None the less the classification mentioned above satisfactorily emphasises the various common types of lesion, and will be followed here. It is only necessary for the student to bear in mind that a dry pleurisy may pass into a pleurisy with effusion, or may co-exist with it. Haemorrhagic pleurisies do not form a well-defined group: the chief conditions with which they are associated are briefly described at the end of this section.

**Fibrinous or dry pleurisy.**—The pleura in this condition loses

its surface lustre and becomes roughened by a granular deposit of fibrin. Adhesion takes place between the two opposed serous layers at the site of inflammation, and is followed by the entry of newly-formed blood-vessels into the inflammatory exudate. In this way the inflammatory deposit becomes gradually organised, with the result that the visceral and parietal pleurae are united by fibrous adhesions presenting all degrees of density according to the duration of the inflammation. In the slightest cases these adhesions appear after death as thin transparent strands, stretched and attenuated by the continual movements of respiration. At the other extreme one 1702*A*
meets with dense masses of yellowish cicatricial tissue, occupy- 1719*B*
ing the position of the serous membranes and uniting the lung and chest-wall inseparably together. 1669*A*

**The causes.**—Dry pleurisy may be primary, At least, clinically one meets with such instances although they can seldom be demonstrated to the eye, inasmuch as the lesion is never, *per se*, a fatal one. Far more often such pleurisy is secondary to some inflammation of the thorax or its contents, or to some constitutional affection.

Every inflammation of the lung, or pericardium, or thoracic parietes, which reaches the pleura is competent to produce a fibrinous pleurisy at the site of its invasion. Of such inflammations the following are the most frequently encountered.

**Lesions of the lung producing fibrinous pleurisy. Lobar pneumonia.**—This disease involves the pleura with such con- 1697*A*
stancy as to have earned the synonym *pleuro-pneumonia*. As 1698
will be seen below, the pleural inflammation in this case leads 1700
not infrequently to a purulent effusion. **Broncho-pneumonia** $1696A_3$
is often attended by dry pleurisy, but not so constantly as is $1696A_4$
lobar pneumonia.

**Tuberculosis of the lung**, especially when the disease is chronic, is generally attended by a localised pleurisy in the area of the lesion, the pleural thickening often reaching an extreme degree. 1699*A*
But a lung may be studded with miliary tubercles and yet be 1719*C*
free from fibrinous deposits on the pleura, showing that the conjunction is not an essential feature of a purely tuberculous infection. Tuberculous lesions of the pleura show a peculiar 1717
liability to the production of serous effusions.

**Infarctions of the lung**, both simple and purulent, and **septic conditions**, such as gangrene, and abscess, are almost invariably 1709*A*

1709*B* attended by a fibrinous pleurisy at least, and not seldom by subsequent pus-formation.

**Lesions outside the lung causing fibrinous pleurisy.**—Although most secondary dry pleurisies depend upon a lung-lesion, any inflammation of adjacent parts is competent to produce it by direct extension. Thus pericarditis, hepatic or subdiaphragmatic abscess, or any inflammation which involves the diaphragm, may produce a pleurisy. Lastly, localised pleurisy is associated with any infective lesion of the ribs or sternum or vertebrae which is extensive enough to reach the pleural sac.

**Constitutional affections attended by fibrinous pleurisy.**—The constitutional affections most prone to be attended by a dry pleurisy are: (1) acute rheumatism, and (2) chronic nephritis. But almost any zymotic disease may on occasion have dry pleurisy for an accompaniment.

**Pleurisy with effusion.** *(A)* **Serous pleurisy.**—Effusions of serum into the pleural sac fall into two distinct groups. In the one the effusion accompanies a definite lesion of the pleura, and is an inflammatory exudate. Strictly speaking this variety of effusion is the only one to which the term "serous pleurisy" is properly applicable. The other is provided by effusions occurring in the course of grave constitutional diseases such as chronic failure of the heart, diseases in which the fluid is not an exudation but a transudation and part of a general anasarca. But though the distinction is sound in the main it cannot always be drawn, for terminal inflammations of the pleura or lung are common events in the final stages of all constitutional disorders, the effusions met with in such instances being thus in part inflammatory and in part oedematous. The following description of serous pleurisy divides these groups of serous effusion, as they are divided from the clinical stand-point, into the primary and secondary.

**Primary serous pleurisy** is marked by a more or less copious effusion of serum into the pleural sac, the quantity varying from a few ounces to five pints or even more. This fluid closely resembles blood-serum in composition. It is opalescent, highly albuminous, and will often clot spontaneously on removal from the chest.

Microscopically it contains a few lymphocytes and desquamated epithelial cells, and is sterile to ordinary methods of cultivation though

tubercle bacilli may occasionally be demonstrated by appropriate methods.

The lesion of the pleura is in a large proportion of cases, if not indeed always, a tuberculous one. This conclusion has been established by the discovery of tubercle bacilli in the effusion, by the results of animal experiment, and by observation of the subsequent history of patients who have suffered from primary serous pleurisy. But actual demonstration of the lesion is seldom possible, for the disease is rarely fatal. On the analogy of tuberculous ascites, which is commonly associated with a miliary tuberculosis of the peritoneum, one may assume with a probability of accuracy that primary serous pleurisy indicates miliary tuberculosis of the pleura. But whatever the precise lesion the point to be remembered is that *such pleurisies generally have a tuberculous basis.*

Serous effusions generally gravitate to the base of the chest, but may be loculated in any situation. Their effect depends upon their size and duration. If large they displace the displaceable contents of the thorax, the heart and mediastinal contents moving towards the opposite side under pressure of the effusion. This pressure also produces collapse of the lung on the affected side, the viscus appearing in an extreme case as a dark, airless, almost unrecognisable mass lying along the spinal aspect of the pleural cavity (carnification). If the effusion be persistent the expansion of the collapsed lung becomes permanently diminished, and the chest-wall correspondingly shrunken even after complete removal or absorption of the fluid. The ultimate effect of serous effusions upon the pleura is to produce adhesions between its visceral and parietal layers, with a degree of thickening determined principally by the duration of the inflammatory process.

**Secondary serous pleurisy.**—(1) An inflammatory serous effusion may complicate pulmonary tuberculosis.

(2) Apart from pulmonary tuberculosis the diseases most often complicated by an inflammatory serous effusion into the pleural sac are chronic renal disease in its later stages and malignant disease of mediastinum or lung. The only point of special note in connection with the effusion in such cases is that it is often blood-stained.

(3) As has been observed above, serous effusions often occur,

either with or without evidences of pleural inflammation, as a terminal complication of all chronic diseases.

(*B*) **Purulent pleurisy or empyema.**—An empyema is a collection of pus situated between two layers of pleura. The effusion generally gravitates to the base of the chest, but may form a loculated abscess anywhere on the surface of the lobes or between them. In the latter case the empyema is said to be *interlobar*.

1704$A_2$ Confusion sometimes arises as to whether a given exudate is to be considered purulent or not, for certain effusions have the appearance of a turbid serum. An abiding distinction lies in the microscopic characters of the exudate. When polymorphonuclear leucocytes are abundant and micro-organisms are demonstrable in any film of the effusion, the latter is to be considered purulent, not serous. This distinction is of great significance, for whereas a serous effusion is of comparatively little immediate moment, the thin pus with which it is liable to be confounded is often an indication of a virulent general infection—that is to say, a septicaemia—and of grave import.

The belief that effusions originally serous may subsequently become purulent is probably based on the above-mentioned confusion, for there is good evidence that such a transformation is a very rare event; if, indeed, it ever occurs except as a result of accidental infection from without, as by contamination with a dirty exploring needle.

Empyemata fall into two classes, the acute and the chronic. **Acute empyemata** are characterised by the thin turbid effusions above described. They are seldom loculated, and often show the virulence of the infection causing them by being slightly blood-stained. The organisms most often responsible for this variety of empyema are the pneumococcus and the pyogenic cocci. When the pneumococcus is the agent the disease is often merely the local expression of a general septicaemia, the primary focus being sometimes in the lung, but sometimes not discoverable. Cases depending upon the pyogenic cocci are usually secondary to some septic focus, either in the lung, such as a purulent infarct, or, more often, elsewhere, such as an infective periostitis or osteo-myelitis.

**Chronic empyema**, the commoner variety, is caused most often by the pneumococcus and appears as a sequel of lobar pneumonia or some less definite inflammatory lesion of the lungs.

It may complicate pulmonary tuberculosis, but is then generally associated with pneumo-thorax (*v.* p. 104). Chronic empyemata present all grades of bulk. The pus forming the common pneumococcal empyema is thick and of a greenish-yellowish colour which is almost distinctive of this infection. There is a marked tendency to fibrin-formation, which often leads to the appearance of gelatinous masses of clot containing pus-cells within its meshes. The pus is inodorous if the infection be purely pneumococcal, but when contaminated by other organisms, it may be exceedingly offensive.

Microscopically the effusion contains pus-cells in varying stages of disintegration. In a case of long standing there may be few distinguishable cells, and much amorphous granular débris.

The mechanical effects of a purulent effusion are identical with those of a serous pleurisy, that is to say, displacement of thoracic viscera, and compression of the adjoining lung. Left to itself an empyema of the chronic type progresses to one or two terminations. If small in bulk it becomes gradually inspissated by the absorption of its fluid constituents, and ultimately forms a condensed layer of yellowish material between the layers of pleura. Calcareous deposits occasionally 1672
appear in this inspissated pus, and in extreme cases form 1674
definite plates. When the quantity of pus is large the abscess may evacuate itself spontaneously, either by perforating the 1675
thoracic parietes and discharging itself externally, or by $1704A_2$
eroding a bronchus, in which case the pus is voided by coughing.

The consequence of long-deferred drainage of an empyema is more or less permanent carnification of the lung submitted to its pressure. As a result the cavity of the abscess, when the $1704A$
pus is at length evacuated, is very slowly obliterated, and that chiefly by a falling in of the chest-wall on the affected side. In order to expedite this obliteration it is usual in such protracted cases to remove large portions of many ribs, a proceeding known as Estlander's operation. $1675A$

(*C*) **Haemorrhagic pleurisy.**—This is a vague group, for bleeding into or from the pleural surface may result from a great variety of conditions. These conditions may be summarised under the following three heads, which embrace the most important associated conditions.

(1) Acute infections.—Acute septicaemias, irrespective of the variety of organism at work, evince a haemorrhagic tendency.

In fulminating cases it is usual to find the pleurae dotted with petechial haemorrhages, death having anticipated any inflammatory reaction. In less acute but still virulent infections, pleurisy when it occurs is of the purulent type, the effusion being thin and blood-stained.

(2) Constitutional affections associated with a tendency towards bleeding. It has already been mentioned that effusions occurring in the course of chronic renal disease are often blood-stained. The same phenomenon may be observed occasionally in the course of lymphadenoma and the grave anaemias.

(3) Malignant tumours of the lung or mediastinum may be associated with haemorrhagic effusions.

## II. PNEUMOTHORAX.

In health, the term "pleural cavity" is a misnomer, for each pleura forms a closed and empty sac, whose visceral and parietal walls are maintained in close opposition. Opinions vary as to the nature of the force maintaining this opposition, some authors assuming it to be the atmospheric pressure alone, others upholding a positive cohesion between the two layers of membrane. Whatever be the truth, it seems certain that during each effort of inspiration there is established, between the two layers, a negative pressure due to the elasticity of the expanding lung. Any lesion, therefore, which exposes the interior of the pleural sac to the pressure of the atmosphere, tends to destroy this negative pressure, and to allow the elasticity of the lung full play, with the result that the lung collapses towards its root, and the pleural cavity, ceasing to
1704$A_3$ be potential only, becomes actual and is occupied by air. This condition is called pneumothorax. The physical consequences of a pneumothorax, which is in effect a gaseous effusion, are precisely similar to those of a large liquid effusion. The displaceable contents of the thorax are pushed towards the opposite side, and the affected side of the chest assumes its maximum capacity.

1704*A* Although many lesions are competent to produce a pneumothorax, its most frequent cause is the rupture into the pleural sac of a bronchus or air-vesicles weakened by tuberculous infiltration. *The* cause of pneumothorax is chronic pulmonary tuberculosis. By such a rupture a communication is established

between the atmosphere and the interior of the pleural sac, by way of the bronchi. With each effort of inspiration air enters the sac, and, since the orifice is often valvular owing to the direction of the perforation, the effort of expiration frequently fails to expel it again. By successive inspirations, the layers of the pleura are progressively stripped from each other by the growing quantity of imprisoned air, and finally complete collapse of the lung ensues, the air-pressure within the pneumothorax often exceeding that of the atmosphere, especially during expiration.

Pneumothorax signifies the presence within the pleural sac of air only. But the conditions which produce pneumothorax generally promote an accompanying pleurisy with effusion, which may be serous, but is more often purulent. To these combined lesions the terms hydro-pneumothorax and pyo-pneumothorax are respectively applied. Of causes, other than tuberculous disease of the lung, the following are the most important: 1704*A*

1. Perforating wounds of the chest.
2. Rupture of air-vesicles in an apparently healthy lung under the influence of strain.
3. Perforation of the pleura through the diaphragm, establishing a communication between an air-containing viscus and the pleural sac. For example, malignant disease of the stomach may erode the pleura and cause pyo-pneumothorax.
4. Perforation of the lung from the pleura, as by an empyema which has ruptured into a bronchus.
5. Rupture into the pleural cavity of purulent infarcts, or pyaemic abscesses in the lung, such as follow septic thrombosis of the jugular vein due to otitis media.
6. Perforation of the pleura by cancer of the oesophagus.
7. Spontaneous gas-formation in an empyema, due to the presence of gas-forming organisms.

## III. HAEMOTHORAX.

Effusions of pure blood into the pleural sac are rare. They occur most often as a result of rupture of aneurysms of the thoracic aorta, or from perforating wounds which happen

to lay open a considerable artery. Haemothorax occasionally complicates aspiration of the chest, as the result of damage to an intercostal artery by the aspirating needle.

## IV. CHYLOTHORAX.

Occasionally there are met within the pleural cavity effusions having a milky appearance due to the presence of fat globules in suspension. The condition is known as "chylothorax," the effusion containing an admixture of chyle. This type of effusion has no constant associations, but generally depends upon obstruction to the thoracic duct, as, for example, by a mediastinal tumour. The term "chyliform" is applied to effusions which are milky in appearance, yet contain no fat. These are rare, and little is known of them.

## V. TUMOURS OF THE PLEURA.

These may be primary, but are in a vast majority of instances secondary. The only primary growth in this situation which requires mention is endothelioma, and this is rare.

1678*A* Secondary tumours of the pleura may be either carcinomatous
1678*B* or sarcomatous. The deposits are usually multiple, and appear
1678*E* as raised opaque areas, scattered at random about the pleural
1678*H* surface. There is, however, a variety of pleural malignant disease which is rather an extension from the primary growth than a secondary deposit in the current sense of the term. It
1728*K* is encountered in connection with malignant growths at the
1729*C* root of the lung, and gives the lungs the appearance of being enveloped in a thick cuirasse of yellow growth. Malignant disease of the pleura is often associated with effusions of serum, stained with blood and sometimes containing detached particles of growth.

# CHAPTER X.

## DISEASES OF THE BRONCHI AND LUNGS.

I. Bronchitis. II. Collapse of Lung. III. Dilatation of Air-Tubes. IV. Emphysema. V. Chronic Pneumonia. VI. Vascular Disturbances. VII. Lobar Pneumonia. VIII. Broncho-Pneumonia. IX. Pulmonary Tuberculosis. X. Syphilis (Gummata). XI. Glanders. XII. Actinomycosis. XIII. Gangrene. XIV. Abscess. XV. Tumours. XVI. Parasites.

### I. BRONCHITIS.

(A) Acute. (B) Chronic (See Emphysema).

(A) **1. Simple; 2. Fibrinous.**

**1. Simple or catarrhal bronchitis.**—Catarrhal inflammation of the large air-tubes is a common incident of many zymotic diseases, particularly the common "cold," measles, whooping-cough, influenza, and typhoid fever. But it may occur apart from these affections, as a consequence of exposure to cold, or to irritating vapours. The lesion comprises an initial stage of dryness and hyperaemia of the mucous membrane, passing on to a phase of pronounced activity of the mucous glands.

Microscopically, a section of the bronchi shows hyperaemia of the mucous and submucous coats, infiltration of the whole wall with polymorphonuclear leucocytes and round cells, and proliferation and desquamation of the epithelium. The glands secrete abundant mucus. The lumina of the bronchi contain an exudation, consisting of leucocytes, mucus, and cast-off epithelial cells. The picture is typical of "catarrhal" inflammation.

**2. Fibrinous or plastic bronchitis** is a rare disease. It is characterised by a recurrence of attacks of acute bronchitis, during which the bronchi secrete a highly-fibrinous material. The adhesive nature of this secretion obstructs its expulsion, with the result that the affected tubes presently become completely filled by concentrically-laminated plugs of fibrin. The effect of this occlusion is to produce collapse of the area of
1683 lung served by the obstructed tube. From time to time a
1685*B* violent effort of coughing dislodges these collections, which are
1685*C* then expectorated, and supply perfect casts of the bronchial tree. Re-accumulations are frequent, and it is not uncommon for the same tubes to yield two or three complete casts in the course of a single attack of the disease. Apart from this definite disease, fibrinous casts of the bronchi are occasionally met with in the course of fibrinous pneumonia and of diphtheria.

## II. COLLAPSE OF THE LUNG.

Collapse of the lung, or pulmonary atelectasis, may be congenital or acquired.

1702*D* **Congenital atelectasis** is rare except in cases of still-birth, but is sometimes produced by the pressure of congenital intra-thoracic tumours. Congenital diaphragmatic hernia is the commonest cause of this condition, for such herniae are often bulky and may contain large viscera such as the liver and stomach. The adjoining lung in these cases preserves its foetal
1702 characters, that is to say it is solid and small.

**Acquired atelectasis.**—Even in health certain parts of the lung are often in a condition of collapse during periods of quiet breathing, especially the thin anterior edges of the upper lobes. But in many diseased states of the lungs (especially in early childhood), and in many lesions of neighbouring parts, collapse of air vesicles forms an important feature. It is caused either by obstruction of the bronchi from within, or by compression of the bronchi or lung from without. Every enduring obstruction of a bronchus results in a gradual collapse of the area of lung served thereby. Thus
1681*A* foreign bodies within the bronchial tree, on the one hand,
1681*B* and on the other, aneurysms and intra-thoracic tumours which exert pressure upon the larger air-tubes, are efficient

causes of pulmonary collapse; as are pleural effusions of any magnitude, whether liquid or gaseous (pneumothorax).

But of all causes of pulmonary collapse the most frequent is obstruction of the smaller air-tubes by catarrhal secretions. The characteristic feature of bronchial exudates is a stickiness and coherence due to the abundance of mucus which they contain. Such exudates are normally discharged by the combined action of the ciliated epithelium of the bronchi and the sudden expiratory effort of coughing. Everything, therefore, which diminishes the efficiency of cough diminishes equally the capacity to eject the bronchial plugs of exudation, and increases the liability to pulmonary collapse. These considerations serve to explain the fact noted above, that early childhood is the epoch at which pulmonary collapse is most prominent; for at this age catarrhal lesions of the bronchi are extremely frequent, while the efficiency of cough is hampered by the yielding character of the thoracic skeleton.

Areas of pulmonary collapse are depressed and of a dark colour. On the post-mortem table almost every lung exhibits a longitudinal band of collapse along its vertebral border—a post-mortem collapse due to the dorsal decubitus of the body. This apart, collapse of the lung is seen most often in association with broncho-pneumonia, and, in early infancy, with acute bronchitis or extreme general debility, especially in rickety subjects. Broncho-pneumonic lungs show, on section, grey raised areas of consolidation interspersed by depressed purple patches of collapse The true nature of the lesion is best 1696$A_4$
appreciated if the lung be artificially inflated before section, for such a proceeding facilitates discrimination between the collapsed and the consolidated portions of the viscus. In early infancy large portions of the lungs may be collapsed without any evidence of broncho-pneumonia, but the characters of the collapsed areas correspond to the description given above.

Lungs subjected for a length of time to pressure from without, especially in the case of purulent pleural effusions, pass through a stage of mere mechanical collapse into a condition of subacute inflammation which ultimately reduces them to shrunken masses of fibrous tissue (*Carnification*). 1704$A_1$

## III. DILATATIONS OF AIR TUBES.

### 1. Bronchiectasis. 2. Bronchiolectasis.

Any chronic inflammation of the air-tubes, or of the alveolar tissues in their neighbourhood, results in a weakening of the tube-walls; and this in turn inclines them to yield before the pressure of the cough and the traction of the forced inspirations with which such inflammations are always associated. Such bronchial dilatation is called *bronchiectasis* or *bronchiolectasis* according to the size of the tubes which sustain the stress of the affection.

**1. Bronchiectasis** is a chronic disease. It may involve the bronchi more or less evenly throughout their length—"tubular" or "cylindrical" bronchiectasis—or may more particularly attack certain areas, with the formation of localised pouchings—"sacculated" bronchiectasis. This is a useful descriptive distinction, but must not be too rigorously received since most specimens show a combination of the two varieties. Bronchiectasis is always associated with fibrous hyperplasia of the tube-walls, in obedience to the rule that chronic inflammations result in fibrous over-growth. It is not uncommon to find a limited tuberculosis apparently implanted upon an already existing bronchiectasis.

**Tubular bronchiectasis.**—The commonest causes of tubular bronchiectasis are chronic, or frequently repeated attacks of bronchitis, or of any form of chronic lung inflammation whether pneumonic, broncho-pneumonic, or tuberculous. Lungs so affected vary in appearance with the cause of the condition, but commonly exhibit a diminution of the vesicular tissue combined with extreme prominence of the air-tubes. In an advanced case the lung appears on section to be mainly composed of immensely thickened, rigid and dilated
1679*B* tubes, filled with a thick purulent secretion and traversing a
1679*C* hardly-recognisable remnant of cirrhotic lung-tissue.

**Sacculated bronchiectasis** is met with in two groups of cases: (1) in chronic lung inflammations which have resulted in much cicatrisation, the bronchial diverticula owing their origin in part to the pull of the contracting fibrous-tissue outside the tube, and in part to the weight and softening
1680*A* influence of accumulated secretions within it.

(2) In cases of continued obstruction of the bronchi, irrespective of the nature of such obstruction. The sacculation is in this form due to the softening influence of retained secretions, as noted above. The commonest causes of bronchial obstruction are:

(*a*) *Within the tube.* Foreign bodies. 1681

(*b*) *Without the tube.* The pressure of aneurysms, or malignant growths at the root of the lung, or of enlarged caseous or lymphadenomatous glands at the tracheal bifurcation.

All these lesions are liable to be associated with a sacculated bronchiectasis. The lung is riddled with cavities lined by a smooth membrane and filled with foul purulent secretion. To 1679*D* the naked eye these cavities may, on section of the lung, appear more of the nature of multiple pulmonary abscesses than bronchial dilatations. But their true character may be demonstrated by proof of their direct communication with air-tubes of considerable size.

2. **Bronchiolectasis.**—Bronchiolectasis indicates a permanent dilatation of the smaller bronchi. It is an acute condition more nearly allied to emphysema than to bronchiectasis. It is particularly common in childhood, and occurs in the course of acute or subacute lung inflammations, especially when violent cough is a feature of the disease. Thus broncho-pneumonia complicating whooping-cough is its most frequent associate. The dilatations are probably produced in this instance, as is believed to be the case with emphysema, chiefly by the violence of forced inspirations; the pull acting upon a lung whose tubes are weakened by inflammation, and whose alveolar tissues are in large part inexpansible as a result of broncho-pneumonic consolidation.

A lung affected by bronchiolectasis has on section a honeycombed aspect produced by innumerable dilatations affecting the smaller air-tubes. The intervening lung-tissue is solid in 1690*B* the main, but emphysematous bullae may often be seen on 1690*C* the pleural surface. The lower lobes are those most fre- 1690*D* quently involved.

## IV. EMPHYSEMA.

The term pulmonary emphysema indicates a distension of the vesicular tissues of the lung exceeding that which can be produced by the maximum effort of normal inspiration. The

1689 lung in consequence is increased in bulk and exhibits, especi-
1689*A* ally upon its pleural aspect and along the anterior edges of the
1689*B* upper lobes, air-containing bullae, often of large size, produced
1690*A* by the coalescence of permanently over-distended air-vesicles.

Emphysema appears in two forms, acute and chronic, the pathology varying in the two groups of cases, although the physical conditions governing the appearance of the lesions are in general terms the same in both.

The mechanism by which emphysema is produced has been the subject of much controversy, some authorities upholding an inspiratory origin for the pulmonary over-distension, others an expiratory one, while a third school has assumed a degeneration of the lung-tissues as the responsible cause. In the appended account we have followed the teaching of Dr. Gee, which is that now most commonly received. As will be seen it accepts the associated operation of all the three causes mentioned above, but places them in a descending scale of importance.

**Acute emphysema.**—Acute emphysema is often found in the lungs of children who have been attacked by suffocative dyspnoea such as attends membranous laryngitis. A few hours of severe laryngeal obstruction suffice to produce extensive emphysema. In such cases the pathology is uncomplicated; cough is absent or trivial, and the lungs as a rule free from pre-existing disease. The only symptom of importance is inspiratory dyspnoea. It is, therefore, necessary to conclude that at least in the acute type of the lesion the prime cause of emphysema is a repetition of forced inspirations.

**Chronic emphysema.**—Chronic emphysema presents a more complex problem, for the condition is but slowly established and the possible contributory causes are numerous. The lesion is a rarefaction and destruction of lung-tissue, associated with an increase in the size of the lung.

Now increase in the size of the lung as a whole can only be produced by the aid of a prior expansion of the thorax, and this can only result from forced inspirations (inspiratory theory).

Secondly, as regards the part played by intrinsic debility of the lung-tissues, repeated over-expansion connotes a repeated anaemia of the over-stretched alveoli, an anaemia likely to result in faulty nutrition and loss of natural resilience.

The third factor is the influence of forced expiration (expiratory theory). The muscles concerned in forced expiration are abdominal and exert pressure upon the lower parts of the lung. When, however, the glottis is closed preparatory to the act of coughing, and the muscles of forced expiration come into play, the imprisoned air leaves the lower lobes where pressure is considerable, and, failing an immediate exit by way of the glottis, temporarily over-distends that part of the lung where pressure is least; namely, the upper and anterior margins. Forced expiration must therefore be considered a contributory cause of emphysema, being made efficient for this purpose by the uneven distribution of the pressure it excites. But the area of lung under notice is not only at the mercy of expiratory over distension, but of inspiratory over-distension also. For the muscles of forced inspiration act upon the upper part of the chest, and by fixing the clavicles and upper ribs allow the muscles of normal inspiration to act to the best advantage. Thus both forced inspiration and forced expiration combine to over-distend that portion of the lung in which emphysema is, as a rule, most in evidence.

Chronic emphysema is always associated with chronic bronchitis; and chronic bronchitis, whether dry or attended by much secretion, implies a frequent partial closure of the larger air-tubes by exudations. The sense of dyspnoea induced by this partial closure is to be regarded as the fundamental cause of chronic emphysema. It excites exaggerated efforts at inspiration, and these in turn involve a corresponding energy of expiration, the excess in both directions serving to maintain parts of the lung in a condition of unnatural expansion by which its inherent resilience is in the course of time permanently impaired. Emphysema once established is itself a cause of chronic bronchial catarrh; and in this way a vicious circle is established, which increases progressively the extent both of the emphysema and bronchitis.

## V. CHRONIC PNEUMONIA.

### (Chronic Interstitial Pneumonia; Fibrosis or Cirrhosis of the Lung.)

The central feature of chronic pneumonia is an increase in the fibrous elements of the lung, the outcome of a continued

inflammation. The distribution of the fibrosis is entirely dictated by the extent of the inflammation of which it is an adjunct; but, wherever it appears, the newly-formed fibrous tissue tends to contract in course of time, producing a degree of deformity and shrinkage proportional to the bulk of lung-tissue which it has replaced. A prominent item in this deformity is bronchiectasis (*q.v.*). Although the localised fibrosis which attends apical tuberculosis, gummata, hydatid cysts and similar limited lesions is strictly a chronic pneumonia, the term is usually reserved for diffuse fibrosis affecting large areas of one or both lungs. Such lesions may attend the following diseases.

1. **Chronic fibrinous pneumonia.**—Occasionally the inflam-
1697 matory exudate of this disease fails to undergo resolution, but becomes organised, with the result that large parts of the lung are in course of time replaced by fibrous tissue. In an extreme case the alveolar structure disappears, the lung being converted into a shrunken grey mass traversed by dilated and thickened air-tubes.

2. **Chronic bronchopneumonia.**—By an analogous process chronic lobular inflammations, whether simple, septic, or tuberculous, may lead to a permanent excess of fibrous tissue. But since in the case of bronchopneumonia both lungs are commonly affected, the resulting fibrosis is here more widely spread. In all cases the anatomical features are roughly identical. Much of the lung is replaced by cicatricial tissue, whose contraction produces on the one hand diminution in the bulk of the lung, and on the other dilatation of the bronchi.

3. **Chronic bronchitis.**—Chronic bronchitis of long standing is often associated with fibrosis distributed along the course of the bronchi.

4. **Chronic pleurisy.**—It is not uncommon to find radiating bands of fibrous tissue passing into the lung from an area of
1696*F* thickened pleura which marks the site of a chronic pleurisy. This variety of fibrosis is met with at or about the bases of the lungs, and may destroy large portions of one of the lower lobes. The carnification of lung resulting from prolonged exposure to the influence of pleural effusions, especially empyemata, is an analogous variety.

5. **Pneumonokoniosis.**—In the ordinary course insoluble particles suspended in the inspired air and adhering to the

mucosa of the bronchi, are swept upwards by the ciliated epithelial cells and expectorated, or are ingested and finally deposited in the lympathic glands at the root of the lung. These glands are in consequence generally blackened by the accumulation of carbon granules within them. But when such particles are very abundant they are carried along by the lymphatic channels to the tissues of the lungs and, being arrested there, excite a chronic inflammation. The disease so caused is called generically "pneumonokoniosis" It is met 1745*B*
with among people exposed for long periods to an atmosphere 1696*E*
heavily charged with insoluble particles, particularly coal-miners 1696*F*
(anthracosis), knife-grinders, workers in silicious and other 1696*G*
stones, and occasionally among workers in cotton, flax and grain.

Lungs affected by pneumonokoniosis present certain features in common. They are bulky, tough and dark-coloured. On section they are seen to be composed in large part of fibrous tissue having a pearl-grey translucent appearance, often disposed in circumscribed whorls or nodules, but at other times diffuse. The colour of the lung varies with that of the particles responsible for the disease, and is consequently darker in the case of the coal-miner than in that of the stone-worker. Pneumonokoniosis predisposes a lung to the attack of the 1696*F*
tubercle bacillus. 1696*G*

**6. Congenital syphilis.**—A diffuse fibrosis is an occasional lesion of foetal syphilis. The whole of both lungs may be converted into a solid whitish tissue—syphilitic pneumonia. 1726

**Microscopic examination** shows a diffuse interstitial fibrosis leading to extreme thickening of the alveolar walls. The arteries exhibit well-marked adventitial thickening.

## VI. VASCULAR DISTURBANCES.

(*A*) Congestion. (*B*) Oedema. (*C*) Infarction.

(*A*) **Congestion.**—This may be active or passive. Active congestion is an early incident in all acute inflammatory lung-lesions, and is particularly well-marked at the commencement of lobar pneumonia. Considered alone, however, active congestion of the lungs has far less pathological significance than the passive variety. The latter is met with whenever circu-

lation through the heart is obstructed to a serious degree. Its chief association therefore is with chronic valvular disease of the heart in the stage of failing compensation. A lung the seat of chronic venous congestion is tough and of a deep brown colour, characteristics denoted by the synonym "*Brown induration.*" On section, the cut surface rapidly becomes bright red owing to oxidation of the contained haemoglobin, and discharges a large quantity of dark blood.

**Microscopic examination** shows a desquamation of the endothelial cells lining the alveoli, extreme dilatation of the capillaries, increase in the inter-alveolar connective-tissue, and in many places deposits of a golden-brown pigment, a product of the destruction of red blood-corpuscles.

A similar but partial congestion is met with, apart from gross lesions of the heart, in subjects enfeebled by age or disease who maintain a supine position for a length of time. In this case, the posterior portions of the lungs are most affected, and the lesion is termed "*hypostatic congestion.*"

(*B*) **Oedema.**—Little is known of the intimate pathology of oedema of the lung. It is present to some extent whenever chronic venous congestion is pronounced, and occurs as an acute condition in some cases of intra-cranial haemorrhage, and chronic nephritis, and occasionally after aspiration of fluid from the pleural cavity. An oedematous lung is bulky, has a somewhat translucent appearance, and pits on pressure. The fluid with which it is charged fills the air tubes. It is watery in consistency, often blood-stained, and flows freely from the
1702*A* cut surface of the lung.

(*C*) **Infarction.**—When an area of tissue supplied with blood by a "terminal artery," *i.e.* an artery with few or no anastomoses, is suddenly deprived of its normal blood supply, it undergoes a series of changes, which are collectively termed infarction. The common cause of infarction is occlusion of a terminal arteriole by an embolus or thrombus. Pulmonary emboli are generally derived from thrombi in the systemic veins, or from thrombi or vegetations in the right side of the heart. Their impaction produces in the lung infarcts of two kinds, the variety depending upon the presence or absence of pyogenic cocci. When the embolus is sterile, the infarction is said to be simple: when pyogenic cocci are present in it, the resulting infarct is a purulent infarct. Simple infarction occurs

only in lungs which are already congested, and is always haemorrhagic in character.

Haemorrhagic infarcts appear as deep-red solid areas, triangular in shape (if the plane of section passes through their length), with the base of the triangle at the periphery of the lung and the apex pointing towards the root. They occur most often in the course of mitral valvular disease, as a result of emboli derived from clots formed in the 1708*A*
dilated right auricle or ventricle; but they may be due to 1708*B*
thrombosis in the pulmonary artery. Such infarcts in course of 1750*B*
time become vascularised by advancing loops from neighbouring vessels. The dead lung-tissue is gradually absorbed and replaced by granulation-tissue which in turn follows its normal evolution into fibrous tissue and leaves nothing but a puckered scar to mark the site of the infarct.

A purulent infarct implies that the embolus responsible for the lesion is derived from an infective thrombus. An infective embolus, in addition to its physical effects, viz. infarction, introduces pyogenic organisms into the damaged area, and the outcome is a metastatic abscess involving not only the infarct, but the neighbouring lung as well. Purulent infarcts, therefore, often lack the regularity of outline which marks the simple variety, and appear as grey and often disintegrating foci of necrosis. The diseases with which such infarcts are 1711
most often associated are septic conditions of the uterus during 1711*A*
the puerperium, osteomyelitis, and purulent otitis media com- 1711*D*
plicated by infective thrombosis of the lateral sinus: but any lesion producing an infective thrombosis in the systemic venous circulation is equally competent under favourable circumstances to produce a purulent infarction of the lung.

## VII. LOBAR, CROUPOUS, OR FIBRINOUS PNEUMONIA.

This disease is an inflammation produced by the diplococcus pneumoniae, or pneumococcus. Its characteristic feature is the fibrinous nature of the inflammatory exudate. Although one or more whole lobes are generally affected, partial involvement is sufficiently common to make the term lobar pneumonia technically incorrect.

It is usual to describe the progress of the lesion as consisting

of three stages. The first is that of *engorgement*. The lung is actively congested, but its vesicles still contain air. Presently the second stage, that of *red hepatisation*, is announced by the escape of blood from the dilated capillaries into the air-vesicles, where it clots. The lung is thus converted into a deep-red airless tissue, unnaturally friable and not unlike the liver in consistency. The pleura covering it is invariably roughened, injected and coated by a layer of inflammatory lymph, while the cut surface has a finely granular aspect produced by the innumerable fibrinous plugs which occupy the alveoli.

Microscopically these plugs are found to consist of a fibrinous network holding in its meshes many blood-cells, both white and red.

The third stage, that of *grey hepatisation*, is the first step towards resolution of the inflammatory exudate. It is marked by a copious influx of leucocytes into the air-vesicles. The red cells are destroyed and the fibrinous network liquefied. As a result of this leucocytic invasion the inflamed lung assumes a grey colour from which this stage of the process takes its name, while the air-cells are filled with leucocytes and
1697*A* a granular débris. Under the combined influence of absorp-
1700*A* tion and expectoration the liquefied alveolar contents are gradually removed, the inflammation is finally resolved, and the lung regains its function.

The above is the normal sequence of events in a case ending in recovery. But occasionally the sequence is interrupted by complications. Of these the commonest are *empyema*, *gangrene*, *abscess*, and *delayed resolution*, forming one of the varieties of *chronic pneumonia*. A description of these conditions, all of which occur in other associations, will be found under their respective headings.

## VIII. BRONCHOPNEUMONIA.

Bronchopneumonia is an inflammatory consolidation of the lung produced by an infection which has travelled by way of the air-tubes. The characteristic feature of the lesions is therefore a lobular distribution of the consolidated areas, dictated by the distribution of the air-tubes which have formed the path of the infection. In an early case the patchy nature of the inflammation is easily detected, but at a later period the

coalescence of many contiguous areas of consolidation often produces a massive solidity of the lung which obscures the essentially lobular nature of the lesion.

Bronchopneumonia is not a specific disease like fibrinous pneumonia, but may be caused by a large variety of organisms, whether acting alone or in combination. It is usual for purposes of description to divide the class into three groups, as follows:

1. Tuberculous bronchopneumonia.
2. Septic bronchopneumonia.
3. Simple bronchopneumonia.

**1. Tuberculous bronchopneumonia** is generally caused by the tubercle bacillus acting alone. It results from the passage along the bronchi of tuberculous discharges derived from some disintegrating tuberculous focus which has access to the bronchial lumina. This focus is commonly a tuberculous deposit at the apex of the lung, but may be a caseous lymphatic gland at the tracheal bifurcation or at the root of the lung which has eroded the wall of an adjacent bronchus and has thus established a communication with the lumen of the bronchial tree. The varieties of tuberculous bronchopneumonia are dealt with in the section devoted to pulmonary tuberculosis.

**2. Septic bronchopneumonia** is the name applied to the variety of lung inflammation which follows the entry into the bronchial lumen of grossly contaminated material. Its common causes are the following:

(*a*) Inhalation of matter vomited during anaesthesia.
(*b*) Inhalation and gravitation of discharges from ulcerative lesions of the upper air-passages, such as malignant disease, or diphtheria.
(*c*) The rupture of an abscess into the air-passages.
(*d*) Fistulous connections established between the oesophagus and trachea whereby food is enabled to reach the bronchi. Such fistulae generally depend upon injury, or the ulceration consequent upon malignant disease of the oesophagus or the impaction in it of a foreign body.
(*e*) The inhalation of inflammatory exudates produced by the presence of foreign bodies within the air-tubes.

**The lesions.**—A lung affected by septic bronchopneumonia is often extensively consolidated by the time death occurs, owing, as has been said, to the coalescence of adjacent areas of consolidation. The cut surface is mottled and may be dotted with
1702*B* cavities of varying size. Of these some are abscesses filled
1702*C* with inspissated pus, while others are empty, being merely bullae produced by the action of gas forming organisms. The bronchi leading to the affected areas are always filled with muco-pus, and some degree of pleurisy is an almost invariable accompaniment.

3. **Simple bronchopneumonia.**—In the two preceding groups the course of the infection by the bronchi and bronchioles can usually be demonstrated: in simple bronchopneumonia such demonstration except in early acute cases is not often possible, but the lobular distribution of the lesions, and the characters of the exudation distinguish the disease from lobar pneumonia.

As regards causation two classes must be recognised: primary and secondary bronchopneumonia. **Primary bronchopneumonia** is due in the majority of instances to infection by the pneumococcus, but in a few instances the infective agent is the influenza bacillus or a streptococcus: the infective agent, whatever it may be, is usually unmixed. In **secondary bronchopneumonia**, on the other hand, mixed infections are much more common, the pneumococcus being found in association with a streptococcus, with staphylococci and with Friedländer's bacillus. The diseases which are particularly apt to give rise to bronchopneumonia as a complication are measles, whooping-cough, diphtheria, and the other infective fevers.

**The lesions.**—The naked eye appearances differ remarkably with the extent and duration of the disease. In the most acute form the lung is a deep-red colour with areas of purple consolidation, which on microscopic examination are found to consist of alveoli crowded with the products of inflammation together with extravasated blood. These appearances are met with most often in bronchopneumonia of a fulminant character. In less acute cases the lung presents a mottled grey and red appearance: the grey areas of consolidation standing out from
1696$A_1$ the redder air-containing portions. The areas of consolidation
1696$A_2$ vary in size from nodules which closely simulate small grey
1696$A_4$ tubercles, to patches of irregular shape and half an inch or more in diameter: in rare instances the original areas of consolidation

have coalesced until the whole of a lobe is involved (pseudo-lobar bronchopneumonia). 1696$A_3$

Lastly, in chronic bronchopneumonia the consolidated lung tissue is of a uniform grey or yellowish-white colour; the bronchioles are dilated, and exude pus. Occasionally liquefaction of the foci of inflammation gives rise to numerous minute abscesses. 1696$A_3$

**Histology.**—The characteristic feature of bronchopneumonia is the catarrhal nature of the exudation, which consists largely of the desquamated cells of the alveolar epithelium intermixed with leucocytes. There is little or no fibrin-formation. In the haemorrhagic lesions extravasated blood is abundant, while in the chronic grey bronchopneumonias the original features are obscured by an infiltration of the affected tissues with leucocytes and round-cells. The bronchi and bronchioles show proliferation of their epithelium, and their lumina are filled with leucocytes, mucus and epithelial débris. The alveoli nearest to the bronchioles are packed with leucocytes, but further from the bronchioles the characteristic desquamative features of the exudation are evident. In the inflamed areas the alveolar walls are thickened by dilated capillaries, and by infiltration with leucocytes. In the mottled grey and red form the irregularity of the inflammation is even more obvious under the microscope than to the naked eye, normal and inflamed alveoli lying side by side.

## IX. PULMONARY TUBERCULOSIS.

**General considerations.**—Of all organs the lungs are the most liable to tuberculosis. The anatomical unit of the disease here as elsewhere, is the "grey tubercle," which represents the effects of tissue-reaction against the products of the tubercle bacillus. A grey tubercle is in its earlier stages a rounded body about the size of a pin's head, hard, and of a pearl-grey colour. Histologically it consists in a typical instance of three fairly well-defined zones; a central one immediately surrounding the offending organisms, often containing giant-cells, and prone to caseous degeneration; a middle one consisting for the most part of large epithelioid cells and an outer one of small round-celled infiltration. As the caseous degeneration of the central zone progresses its cellular character disappears, the constituent cells being converted into débris of necrotic material, commonly called pus on account of its macroscopic appearance but containing no recognisable pus-cells. Tubercles which have advanced to such a pronounced stage of caseation assume a yellow colour and are spoken of as "yellow

tubercles." When many such yellow tubercles lie side by side they tend to coalesce, with the formation of tuberculous cavities varying in extent, and occupied by collections of caseous material ranging in consistency from liquid to semi-solid.

In the lung the ultimate fate of these collections depends upon the activity of the infection. In the acutest cases caseation and subsequent coalescence of caseous areas may
1718*B* destroy large tracts of lung, and, since the blood-vessels and air-tubes are more resistant than the lung tissue, the resulting cavities in the lung are often found to be crossed by stripped and unsupported tubes. In such a case the affected arteries, being weakened by the ulceration and necrosis going
1718$C_2$ on around them, not uncommonly undergo aneurysmal dilata-
1725*B* tion within the cavity; or, giving way entirely, permit of
1758*C* copious arterial effusions and the haemoptysis so common in the disease. At the opposite extreme lie the lesions indicative of pronounced tissue-resistance and capacity for spontaneous limitation of the disease. Either the original focus of infection has never progressed to the stage of caseation, in which case the site of the mischief is represented by nothing more than a puckered fibrous area, a mere scar in the lung; or, more often, caseation having occurred, the accumulation
1718$A_1$ becomes confined by a tough envelope of fibrous tissue, and
1718$A_2$ calcareous salts are deposited in it converting it into a chalky concretion. But the great majority of examples of pulmonary tuberculosis fall between these extremes and exhibit, in all degrees of gradation and proportion, combinations of the destructive and reparative elements in the natural history of the lesion.

The lymphatic glands situated at the bifurcation of the trachea and at the root of the lung almost invariably share in tuberculous infections of the lung. They become enlarged and
1664*M* often caseous, or calcareous, and may play an important part
1717 in the evolution of tuberculous lesions elsewhere in the body by acting as a focus from which a general infection takes it source.

In this connection we may mark three important points of difference between the types of lesion met with in early and in later life respectively.

(1) In the adult, pulmonary tuberculosis generally makes its first assault upon the apical region of the lung, where the

process may remain more or less localised for years, while in early childhood we find lesions scattered at random throughout all the lobes.

(2) During the first lustrum the bronchial glands bulk large in the pathology of tuberculosis, for it is common at this age to find caseation of these glands even without evidence of a lung lesion, while it is never absent when the lung is actively involved. These caseous bronchial glands often in early childhood serve as foci from which the disease becomes generalised throughout the body.

(3) During early life tuberculous lesions show a greater virulence than characterises them later. The infection is more often diffuse, caseation is rapid, and repair much less in evidence.

**Paths of infection.**—Much conjecture and little certainty surround the question of the avenues by which the lung becomes infected. There are five possible routes, all of which are probably utilised on occasion.

(1) Infection may attack the lung by direct extension from a contiguous focus of the disease. This is particularly the case in early life, the commonest focus being in the glands at the bifurcation of the trachea and about the root of the lung. 1664*M* 1724*F*

(2) Since in many cases a large part of the mischief is bronchopneumonic in distribution it is certain that aspiration of infected material, assisted probably by gravitation, disseminates the disease by way of the air-tubes.

(3) Miliary tuberculosis of the lung represents a diffusion of the bacillus by way of the blood-stream.

(4) The early apical involvement which characterises the ordinary adult type of pulmonary tuberculosis has been held to indicate an infection travelling along the lymphatics of the neck from the tonsils and upper air passages; a likely but unproved hypothesis. There is no doubt that within the lung itself the infection spreads by lymphatic paths: for it is common to see around a focus of tuberculosis a radial arrangement of grey tubercles marking the infected lymphatic channels.

(5) It has lately been shown that insoluble particles, ingested, are rapidly deposited in the lung, having entered the lymphatic vessels and reached the lung by way of the thoracic duct and pulmonary artery. According to this view the

swallowing of infected material is a fruitful source of pulmonary tuberculosis.

**Pulmonary tuberculosis.** (*a*) **Acute** ; (*b*) **chronic.**—(*a*) Acute pulmonary tuberculosis appears in two forms : (1) acute miliary tuberculosis ; (2) acute caseating tuberculosis.

(1) **Acute miliary tuberculosis** of the lung is generally bilateral and indicates an infection by way of the blood-stream. A lung so affected is dotted with innumerable grey or yellow
1719*A* tubercles giving to the tissues a feeling as though they had
1717 been riddled with small shot. Such miliary tuberculosis may attack a previously undamaged lung, or may appear as a terminal event in cases of long-standing pulmonary disease. In the latter case the lung will present both the acute and the chronic types of lesion.

(2) **Acute caseating tuberculosis** of the lung presents itself in two forms. Of these the commonest is obviously broncho-
1719*D* pneumonic, the lesions having a lobular arrangement deter-
1721*A* mined by the distribution of the air-tubes. The oldest foci are generally to be found near the apex of the upper lobe but occasionally towards the apex of the lower lobe. Cavitation is common in these situations. A cavity or "vomica" of long standing is generally enclosed in a fibrous envelope and lined
1723*A* by a smooth pyogenic membrane, while recent vomicae appear
1723*B* as ragged ulcerous excavations of the lung-tissue lined by necrotic material. Elsewhere the affected lung shows abundant tuberculous deposits in all stages of caseous degeneration, the intervening areas presenting a solid appearance due to secondary catarrhal inflammation.

The second variety of acute caseating tuberculosis of the lung is sometimes called "**caseous pneumonia**" or "**pneumonic phthisis**" because the affected lung bears a general resemblance to a lung in the stage of grey hepatisation produced by lobar
1718*A* pneumonia. The lesion is generally unilateral and involves a
1718$C_1$ whole lung. The lung is solid and of a pale grey colour indicative of wide-spread caseation. If life be sufficiently prolonged, large tracts of the lung may disintegrate with the
1718*B* formation of an intra-pulmonary caseous abscess, into the
1718*E* cavity of which projects a bunch of stripped blood-vessels and air-tubes. These cases are really examples of confluent tuberculous bronchopneumonia, and it is sometimes possible to demonstrate an ulcerous communication between a bronchus

on the affected side and a liquefied caseous gland at the tracheal bifurcation, which has thus been enabled to discharge its infective contents into the lumen of the bronchial tree. 1724*G*

**Chronic pulmonary tuberculosis.**—Within this category falls a vast majority of instances of pulmonary tuberculosis. The characteristic features of the lesions are, firstly, a remarkable constancy in their general distribution, and, secondly, an abundant and often diffuse fibrosis indicative of effort at repair. The seat of election for the oldest lesions is an area in the upper lobes situated about one and a half inches below the summit. Here one finds evidences of a chronic infection, from a puckered 1719*C* scar enclosing fibro-caseous deposits, to cavities lined by a smooth membrane and encapsuled by fibrous tissue. These cavities, 1721 though primarily tuberculous, often, in a chronic case, become 1723*A* infected by pyogenic organisms. This mixed infection plays an important part in the clinical course of the disease, but does not produce any typical alteration in its morbid anatomy. Elsewhere about the lungs it is usual to find areas of disease evincing all grades of activity, groups of grey or slate-coloured fibrotic tubercles, caseous or calcareous collections of varying size, bronchiectatic dilatations of air-tubes leading to old areas 1719*D* of disintegration, often a diffuse fibrosis of the lung, and, surrounding the zones of chronic tuberculosis, areas of simple consolidation.

## X. SYPHILIS.

In the tertiary stage of syphilis gummata occasionally appear in the lung. They form yellowish tumours, generally multiple and each surrounded by a fibrous envelope.

**Microscopic examination** shows them to consist of connective-tissue interspersed with aggregations of small round cells. The arteries in the infected zone exhibit thickening of the tunica intima, while the central portions of the gumma are prone to undergo a hyaline degeneration.

Congenital syphilitic disease of the lung is described in the section devoted to chronic pneumonia. 1726

## XI. GLANDERS.

The bacillus mallei is capable of producing a specific broncho-pneumonia in man. It is characterised by multiple areas of

consolidation closely resembling septic infarcts, for they are of a yellowish colour and liable to undergo disintegration at the
1712*A* centre.

## XII. ACTINOMYCOSIS.

The lung-lesions produced by the streptothrix actinomyces are not characteristic like those seen in the liver. They consist of grey nodules enclosing small collections of pus. This pus contains the yellow granules of the streptothrix distinctive of actinomycosis. Occasionally there is a diffuse fibrosis in
1727*E* the neighbourhood of the purulent accumulations.

## XIII. GANGRENE.

When any inflammation of the lung is intense enough to cause necrosis on a large scale, subsequent invasion of the dead tissue by putrefactive organisms may result in gangrene. Gangrene, therefore, is an occasional complication of various diseases. The most active in this direction are fibrinous pneumonia, septic pneumonia and septic embolism. But it is not always possible to trace an antecedent cause for pulmonary gangrene.

A gangrenous lung is generally consolidated throughout. The dead area is of a greenish-black colour, and intensely
1704$C_1$ foetid. Liquefaction of the necrotic focus ensues, converting
1704$C_2$ it into one or more ragged cavities filled with greenish fluid
1704$C_3$ and shreds of putrid lung-tissue.

## XIV. ABSCESS.

Abscess-formation within the lung is not a disease *sui generis*, but, like gangrene, occurs as an occasional incident in various diseases. Of the latter the following are the chief.

**Fibrinous pneumonia.**—Although a recognised possibility, abscess in the course of this malady is rare.

**Bronchopneumonia.**—Both the simple and the septic varieties commonly lead to multiple abscesses. The lung of simple bronchopneumonia is often riddled with minute collections of pus, each representing a focus of bronchopneumonic
1696$A_3$ infiltration which has softened and disintegrated. In septic cases the abscesses tend to be much larger.

**Septic embolism.**—This is a common cause of multiple abscesses in the course of pyaemia. Such embolic abscesses 1711*D*
are roughly triangular in outline and situated near the surface, as is the rule with infarcted areas.

**Tuberculosis.**—Gross caseation and subsequent liquefaction may produce large intrapulmonary abscesses. Thus as a result of pneumonic phthisis the whole of a lobe or lung may be destroyed, the visceral pleura forming the wall of a large abscess into which projects a collection of stripped air-tubes and blood-vessels. Exploratory puncture of the chest under 1718*B*
such circumstances may lead to the faulty conclusion that the lesion is an empyema.

**Hydatid disease.**—Suppuration of an intrapulmonary hydatid cyst is an occasional cause of pulmonary abscess.

**Injuries**, such as stabs and bullet wounds, may lead to pulmonary abscess.

## XV. TUMOURS.

Innocent tumours of the lung are rare and unimportant.

Malignant tumours may be primary or secondary. Primary growths are generally carcinomata originating in the bronchial epithelium, or round-celled sarcomata. In the latter form the growth appears to originate in the lymphatic glands at the pulmonary root, whence it invades the lung by direct extension. In such a case, section of the lung shows a large 1728*F*
white mass enveloping and often occluding the primary 1728*K*
bronchi, and throwing out processes which pass in all direc- 1728*I*
tions into the lung tissue. In an extreme example but little of the lung remains. The bronchi are in places obliterated by the malignant infiltration, but those which escape are dilated into bronchiectatic cavities by the pent-up secretions within them. The pleura is generally involved, but to a varying extent. At times the growth appears to have advanced primarily along this membrane, converting it into a thick cuirasse of new growth. As a rule all distinction between the 1729*C*
visceral and parietal layers is lost, but occasionally there are cystic collections in the situation of the pleural cavity, representative of loculated pleural effusions. In the lung, as elsewhere, large neoplasms tend to become necrotic towards their centre.

1728*H* **Secondary growths.**—Pulmonary deposits of secondary
1728*H*$_1$ growth, whether carcinoma or sarcoma, are commonly encoun-
1729*B* tered. Such growths are multiple and distributed at random,
1729*B*$_1$ giving the lungs a marbled appearance. Histologically they reproduce the cell-type of the parent tumour.

## XVI. PARASITES.

**Hydatid disease.**—The life-history of the taenia echinococcus is related in Chap. I. In its cystic stage the parasite may attack either the pleurae or lungs.

In each case the lesion is substantially the same, and com-
1746 prises a cyst enclosed in a fibrous capsule. As the cyst grows the pressure exerted by it produces mechanical effects identical with those of a pleural effusion, namely compression of the neighbouring lung and displacement of the heart. These cysts may suppurate and discharge their contents externally. More often the point of rupture is connected with the pleural cavity or a bronchus. In the former of these events the result is empyema, in the latter the cyst-contents are voided by the
1746*A* mouth. Multiple hydatid embolism of the lungs may be produced by the rupture into the right chambers of the heart
1746*C* of a hydatid cyst situated in the cardiac wall.

# CHAPTER XI.

## DISEASES OF THE MOUTH AND TONGUE.

1. Aphthous Stomatitis. 2. Ulcerative Stomatitis. 3. Gangrenous Stomatitis. 4. Parasitic Stomatitis or Thrush. 5. Mercurial Stomatitis. 6. Pyorrhoea Alveolaris. 7. Scurvy. 8. Chronic Lead-Poisoning. 9. Syphilis. 10. Tuberculosis. 11. Leukoplakia. 12. Tumours.

Under normal conditions the mouth harbours bacteria, moulds, and spirochaetes in large numbers. The majority of these are not pathogenic, though some of them may under certain circumstances become so, and themselves excite inflammations of varying degree, or modify the course of inflammations already existing. Apart from new growths, the lesions of the mouth are mostly transitory.

**1. Aphthous stomatitis.**—In this condition, commonly seen in children who are out of health from any cause, the mucous membrane of the mouth is generally injected, while here and there, especially upon the inner aspect of the cheeks, are small areas in which the superficial layers of the mucous membrane have a grey, macerated appearance. At a later stage, removal of the damaged epithelium by the movements of mastication reveals shallow ulcers known as "*aphthous ulcers.*"

**2. Ulcerative stomatitis** is the name applied to an ulceration differing from the last only in being more extensive and severe. It may result from a gross disregard of oral hygiene in healthy persons, but is met with more often in people debilitated by various grave diseases.

**3. Gangrenous stomatitis** (cancrum oris, or noma) is the severest form of inflammatory ulceration of the mouth. The process generally commences on the inside of the cheek on one side. Once established, it tends to spread with great rapidity, producing extensive sloughing of all the tissues in the neighbourhood, including the bones and skin. It is almost limited to children under the age of five years.

**4. Parasitic stomatitis or thrush** is a common disease of infants. It is due to the growth of an organism developing in milk, the oïdium albicans, which appears as a white fungous deposit upon the affected mucous membranes. It may attack any part of the oral or pharyngeal surface, including the tongue, and when removed leaves a superficial ulceration of the area upon which it has been growing.

Microscopically, the oïdium albicans consists of mycelial filaments sometimes branched and broken up, especially at the ends, into a series of short rod-shaped or oval segments which behave like spores.

**5. Mercurial stomatitis.**—The administration of mercury may lead to a spongy condition of the gums, and loosening of the teeth. Occasionally also to more or less severe ulceration within the mouth.

**6. Pyorrhoea alveolaris** is the name applied to a diffuse inflammation of the gums and roots of the teeth, for the most part excited by streptococci. It occurs in those who are careless of oral hygiene, and leads to a perpetual, though slight, discharge of pus from the sockets of the teeth. The importance of the condition lies in the facility thus provided for the entrance of organisms into the blood-stream.

**7. Scurvy** produces a swelling of the gums, "*spongy gums*," often associated with an extravasation of blood into the swollen tissues, which gives them a dark-blue or blackened appearance. A somewhat similar lesion is met with in leukaemia and haemophilia.

**8. Chronic lead-poisoning** is attended by a deposit of granules of sulphide of lead beneath the mucous membrane of the gums at the margins of the tooth-sockets. This deposit gives rise to the characteristic "*blue-line.*" It is only found in the neighbourhood of teeth, being absent in edentulous areas.

Drawings 1163 1164

**9. Syphilis.**—All stages of the disease are at times represented in the mouth. Primary chancres are found most

Drawing 1121

often either on the lips or on the tongue. Secondary syphilis affects the mouth in two forms. The more constant is an erythema on the inside of the cheeks and lips, and on the palate and fauces. The pillars of the fauces, the tonsils and the soft palate are swollen, reddened, and often superficially ulcerated. The second form consists of mucous patches upon the inner aspects of the lips and upon the sides of the tongue. They are flat-topped sessile projections, covered by a macerated epithelium, and surrounded by a zone of redness. These oral lesions are a fairly constant phenomenon in the development of secondary syphilis, and usually occur at the same time as the general roseola of the skin. Gummata in the tertiary stage of syphilis affect most frequently the tongue, the soft palate, and anterior pillars of the fauces. They usually degenerate rapidly, and form sharply defined, deep ulcers, which destroy the tissues widely and lead to marked scarring and contraction when healed.

Drawing 1126 1127

Paintings, Plates v. viii.

In congenital syphilis lesions of the mouth are not uncommon. The most frequent lesion is a fissuring of the angles of the mouth, sometimes so deep and persistent as to leave marked linear scars (rhagades). The rarer lesions are small ulcers, especially at the junction of the hard and soft palates, and gummata of the tongue or fauces.

In late hereditary syphilis, the upper central incisors have a characteristic sickle-shaped notch at their lower margins, and are wider at the margins than at the base (Hutchinson's teeth). The canine teeth have what has been described as a "peg-top" shape, produced by a breaking away of the enamel at their free edges, by which means a portion of the dentine protrudes like the peg of a peg top.

Drawings 347 348

**10. Tuberculosis of the tongue** is not infrequent in the form of an indolent superficial ulcer with undermined edges. Similar ulceration may attack the soft palate, fauces and tonsils.

Paintings. Plates iv. ix. 1781 1781*C*

**11. Leukoplakia buccalis.**—Any chronic irritation of the mucous membrane of the mouth may produce a proliferation of the horny epithelium, leading to the appearance of opaque white streaks and patches on the tongue, and sometimes on the cheeks. Such leukoplakia is most frequently found in the subjects of past syphilis, but any source of chronic irritation, such as that of a broken tooth, may produce it even in the

absence of past syphilitic infection. The importance of leukoplakia is due to the fact that the patches are not infrequently the starting point of a squamous-celled carcinoma.

Paintings. Plates vi. vii.

**Microscopic examination** shows a proliferation of the epidermal layers of the skin and small-celled infiltration of the underlying tissues.

**12. Tumours of the mouth.** (*a*) **Macro-glossia and macrocheilia.**—This is a rare disease, the pathology of which is obscure. The lips and tongue, either alone or together, form large shapeless tumours. The condition is usually congenital, but not always. The tongue is too large to be contained within the mouth and remains protruded from the thickened lips, the protruded portion becoming dry, fissured, and inflamed.

1785*I*

**Microscopic examination** shows that the overgrowth consists, as a rule, of a soft fibrous tissue containing lymphatic spaces: it is, therefore, a lymphangeioma, and not a true hypertrophy of the tongue. Cases have been described in which the overgrowth was due (1) to naevoid, (2) to a neuro-fibromatous tissue. The overgrowth may affect the palate and fauces, and may be confined to one side of the mouth, or involve both.

1779*B*

1796*B* 1798*D* (*b*) Any solid tumour of the gums is called **epulis**. The commonest variety consists of fibrous tissue, and is called a "fibrous epulis," but sarcomatous forms occur. These usually exhibit a large number of giant cells in the sarcomatous tissue. Their origin is generally in the periosteum of the teeth-alveoli.

461

1785$A_5$ (*c*) **Papillomata** of the tongue are common.

1788$H_1$ 1788$H_2$ (*d*) The lips, tongue, and floor of the mouth are often attacked by **squamous-celled carcinoma**.

1786*A* **Sarcoma** in these situations is rare.

(*e*) **Cysts.**—The thyroglossal duct which terminates in the foramen caecum at the back of the tongue is sometimes dilated in this situation, forming a cyst (thyroglossal cyst) lined by cubical or flattened epithelium, and occasionally containing in its wall traces of thyroid tissue. When the solid tissue is considerable, the tumour is called a *thyroglossal adenoma*.

1785$D_1$

**Ranula** is the name given to cyst-formation between the tongue and the floor of the mouth. These cysts are formed by the secretion of certain mucous glands (glands of Nuhn) accumulated owing to blocking of their ducts.

Painting. Plate xi.

# CHAPTER XII.

## DISEASES OF THE PALATE, TONSILS, PHARYNX, AND SALIVARY GLANDS.

**A. Varieties of "Acute Sore Throat."**—(*a*) Catarrhal Inflammation. (*b*) Follicular Tonsilitis. (*c*) Quinsy. (*d*) Membranous Inflammations.—($d_1$) Diphtheria; ($d_2$) Vincent's Angina. (*e*) Scarlet Fever. **B. Other Lesions.**—1. Chronic Tonsilitis and Adenoids. 2. Ludwig's Angina. 3. Pharyngo-mycosis. 4. Retro-pharyngeal Abscess. 5. Syphilis. 6. Tuberculosis. 7. Tumours. **Diseases of the Salivary Glands.**—1 Epidemic Parotitis. 2. Acute Parotitis not Epidemic. 3. Salivary Fistulae. 4. Salivary Cysts and Calculi. 5. Tumours.

These structures share in a variety of inflammations, many of which are acute, and give rise to the different kinds of "acute sore throat." It may be remarked in passing that apart from diphtheria most acute sore throats are due to infection by streptococci. But the clinical and pathological features of throat-lesions of all kinds are often modified by secondary infection of the damaged tissues by others of the organisms with which the region abounds.

### A. VARIETIES OF ACUTE SORE THROAT.

(*a*) **Catarrhal inflammation**, evidenced by diffuse injection of the mucous membrane. This occurs in association with measles, with the common "cold"—a term probably compris-

ing a variety of distinct infections—and with other ill-defined maladies.

(*b*) **Follicular tonsilitis.**—In this disease the whole fauces are inflamed but the stress of the process falls upon the tonsils. These are swollen and exhibit upon the surface a number of white specks, discrete at first but tending to coalesce and form a membranous deposit. The white specks represent plugs of inflammatory exudation which occupy the crypts of the tonsil. Follicular tonsilitis may be an independent disease, but it is well to remember on the one hand that the sore throat of scarlet fever may assume the characters of a follicular tonsilitis, and on the other that coalescence of several contiguous plugs of exudation may lead to a simulation of the membranous exudate of diphtheria.

(*c*) **Suppurative tonsilitis or quinsy.**—This is a deep-seated inflammation of one or both tonsils which often progresses to the formation of an abscess. It evinces itself by great swelling of the fauces and soft palate in the neighbourhood of the affected tonsil. When an abscess forms it may point in any direction and not uncommonly discharges itself through the soft palate.

(*d*) **Membranous tonsilitis** is generally due to infection by the diphtheria bacillus. But other bacteria may produce a membranous exudation, especially streptococci, and the bacillus of "Vincent's angina," to be presently described.

($d_1$) **Diphtheria** generally attacks the tonsil primarily, but shows a great tendency to spread and involve the fauces, palate, pharynx, and larynx. It is characterised by injection and swelling of the fauces, and by a membranous exudation appearing in the first instance upon the tonsils. The membrane is a tough, yellowish or grey layer so closely adherent to the underlying tissue that bleeding follows its forcible removal.

1837*B*
1837*C*
Drawing 226

Microscopic examination shows that the membrane consists of a fine network of fibrin containing a few leucocytes. In its surface layers lie masses of diphtheria bacilli. Fibrin filaments are continued from the under surface of the membrane into the degenerating upper layer of the mucosa, and account for the close adhesion between the deposit and the tissues beneath it.

($d_2$) **Vincent's angina** is a peculiar affection of the tonsils and pharynx, which occurs in two clinical forms. The first and commonest is an ulcerative process attacking the tonsils, the

lateral walls of the pharynx, or the pillars of the fauces; the ulceration being associated usually with the formation of a membrane closely resembling that of diphtheria. The second and rarer form consists in the formation of a grey or white membrane, loosely attached, on the tonsils or pharynx, without any attendant or subsequent ulceration. These lesions are characterised by the presence of a large fusiform bacillus decolourised by Gram's method of staining, and associated with a fine spirochaete in considerable numbers.

(*e*) **Scarlet fever.**—Early in the course of this disease the throat is the seat of an acute inflammation varying much in severity in different instances. The mildest lesion is simple congestion producing redness of the parts. In severer cases the palate and fauces are vividly red—perhaps the most distinctive feature of the sore throat in this disease—while white or yellowish plaques appear upon the tonsils. These plaques may simulate, as has been said, those attending both follicular tonsilitis and diphtheria, but they rarely, if ever, involve the fauces and palate in the way familiar in the case of diphtheria. In the gravest instances the tissues of the neck become the seat of a diffuse cellulitis. (*v. B. 2. infra.*)

## B. OTHER LESIONS.

Apart from the "acute sore throats" the following diseases deserve consideration here.

**1. Chronic tonsilitis and adenoid vegetations.**—Frequent recurrence of acute or subacute tonsilitis leads to a permanent enlargement of the tonsils, sometimes extreme enough to bring 1806
the tonsils together in the middle line. This enlargement is 1806*A*
common among children who have been subject to chronic naso-pharyngeal catarrh, and is often associated with a similar enlargement of the lymphoid tissue of the naso-pharynx and upper part of the pharynx—"adenoid vegetations." These 1806*C*
vegetations may reach a great size and are a frequent source of 1806*B*
nasal obstruction, of deafness due to blocking of the orifices of the Eustachian tubes, and of otitis media excited by the spread of micro-organisms along the Eustachian tubes from the inflamed and unhealthy naso-pharynx. Occlusion of the latter by adenoid vegetations, occurring as it does during the period of growth, produces a more or less distinctive facial aspect. The

Drawing 1373 expression of the face is heavy and foolish because the mouth is held constantly open for breathing purposes. The nostrils are small, being undeveloped through want of use.

**Microscopic examination** of enlarged tonsils and of adenoids shows that the increase in size depends partly upon hyperplasia of the lymphoid tissues, but also, and largely, upon fibrous over-growth. Occasionally there is evidence of a tuberculous origin of the chronic inflammation.

2. **Ludwig's angina.**—This term is applied to a diffuse cellulitis of the front of the neck. It has no specific relations, but may complicate any virulent infection of the throat, and is 1615*A* generally due to streptococci.

3. **Pharyngo-mycosis.**—This is a chronic affection of the tonsils, the base of the tongue, or the posterior wall of the pharynx. It is characterised by the appearance of small white specks upon the mucous membrane. These specks project slightly from the surface and are pointed at their free extremities; in some cases the specks agglomerate, and form islets or patches of a tough membranous consistency, with a number of projecting spicules.

Microscopically these appearances are associated with the presence, among other organisms, of long jointed leptothrix filaments which are not branched. The affection is not a serious one, and its importance is due to the fact that in the conglomerate form it may be confused with diphtheria.

4. **Retropharyngeal abscess** is met with in various situations in the pharyngeal wall. The commonest site is the lateral wall of the pharynx, the abscess lying immediately behind one or other tonsil, and being due to an extension of acute inflammation from the tonsils. In other cases the abscess lies in the middle line or slightly to one side, and may be at any level from high up behind the veil of the soft palate to low down opposite the cricoid cartilage. These abscesses are sometimes due to tuberculous caries of the cervical vertebrae, and may be the first indication of serious disease, but in a vast majority of instances the disease is due to an acute infection of the cellular tissue. In themselves they are of importance chiefly on account of the pressure they exert upon 1841*A* the air-passages.

5. **Syphilis.**—The lesions of syphilis affect rather the fauces 1839 than the pharynx, but the latter sometimes suffers. The 1839*A* tonsils are occasionally the seat of primary chancres, and the

palate both hard and soft, may show tertiary ulceration. Necrosis of the hard palate and perforations of the soft palate both depend as a rule upon gummatous ulceration. 14 (Case E.)

6. **Tuberculosis** may occur on the palate, tonsils and pharyngeal walls, in the form of shallow chronic ulcers with undermined edges. In some cases adenoid vegetations are tuberculous in character. 1801*A*

7. **Tumours of the tonsils** apart from the chronic enlargement mentioned above are comparatively rare; they may be innocent or malignant. 1805 1807*A* 1807$B_1$

The uvula is occasionally the seat of papillomata. Carcinoma is rarely found in this region, but in a few instances palatal tumours are of this kind. For the most part palatal tumours contain embryonic tissue with islets of cartilage and even of bone; these are analogous to the "mixed" tumours of the parotid gland, and are variously described as endotheliomata or teratomata. 1803*B* 1800*C* 1800*E*

## DISEASES OF THE SALIVARY GLANDS.

1. **Epidemic parotitis or mumps** is an inflammatory swelling of one or both parotid glands, the submaxillary and sublingual glands occasionally suffering at the same time. The inflammation never progresses to the formation of pus, and nothing definite is known of its aetiology or pathology, except that it is sometimes attended by an acute orchitis or oophoritis, which may lead to atrophy of the affected organs.

2. **Acute parotitis**, often ending in suppuration, is an occasional complication of certain disorders of the digestive tract; *e.g.* typhoid fever, cholera, gastric ulceration, etc.

3. **Salivary fistulae** occur as the consequence of extensive inflammation in the neighbourhood of the various ducts.

4. **Salivary cysts**, due to the retention of the gland secretion, are occasionally formed in the salivary glands as a result of blocking of their ducts by salivary **calculi**. These consist of calcium phosphate and carbonate. 1826*B*. Series of calculi 245*F*

5. Of **tumours** of the parotid gland the most common is that termed a "mixed" tumour. It contains embryonic tissue, cartilage, fibrous tissue, and gland tubules. It is probably to be classed among the endotheliomata. 1824 1827

Sarcomata and carcinomata are rare. 1832*D*

# CHAPTER XIII.

## DISEASES OF THE OESOPHAGUS.

1. Malformations. 2. Inflammations. 3. Strictures. 4. Dilatations. 5. Spontaneous Rupture. 6. Post-mortem Digestion. 7. Foreign Bodies. 8. Varicose Veins. 9. Tumours.

**Normal structure.**—The wall of the oesophagus is composed of a mucous membrane lined by stratified squamous epithelium; of a submucosa containing numerous glands; and of a muscular coat in two layers, the inner transverse, the outer longitudinal.

The glands of the submucosa are often lined by columnar epithelial cells resembling those of the cardiac end of the stomach, and are hence sometimes known as the "cardiac" glands of the oesophagus.

**1. Malformations.**—The only common malformation is that in which the upper third of the tube terminates as a blind sac at or about the level of the cricoid cartilage, while the lower
3626*A* part forms a fistulous communication with the trachea.
3626*B* The two portions are usually united by a band of fibrous tissue.

**2. Inflammations.**—These are met with:

(*a*) As the result of injuries produced by foreign bodies or corrosive poisons. The former are described later in this section. The results of corrosive poisons upon the alimentary mucous membranes are given in the section devoted to diseases
1870 of the stomach.
1838*B* (*b*) In the course of certain acute infections. Diphtheria may lead to a membranous inflammation of the whole oeso-

phagus. Virulent scarlet fever, small-pox, and some other inflammations of the pharynx may cause a vivid injection, and sometimes sloughing, of the oesophageal mucosa.

(*c*) As a result of involvement of the oesophagus in inflammations originating in adjacent structures.

**3. Stricture of the oesophagus** may be (*a*) functional; (*b*) organic.

(*a*) **Functional stricture,** due to spasm of its muscular wall, occurs in association with hysteria and hydrophobia.

(*b*) **Organic strictures** may be due to congenital narrowing of the tube. Otherwise they depend upon:

(*A*) Cicatricial contraction the result of past ulceration. 1834
The commonest antecedents of this condition are corrosive poisoning and ulceration due to the impaction of foreign bodies. 1834*A*

(*B*) The growth of cancer within the tube. 1845*A*

(*C*) The pressure of tumours situated without the tube, *e.g.* aneurysms, enlarged lymphatic glands and malignant growths.

**4. Dilatation of the oesophagus** may be (*a*) circumscribed; (*b*) diffuse.

(*a*) **Circumscribed dilatations** are called *diverticula*, and are of two kinds, called respectively traction-diverticula and pressure-diverticula.

**Traction-diverticula** are due to a pull from without exerted upon a portion of the tube. The commonest cause of this variety is the adhesion of an inflamed lymphatic gland to the wall of the oesophagus. As the inflammation subsides the 1833*C*
gland undergoes cicatricial contraction drawing with it the area of the tube to which it is adherent, and thus producing a shallow depression, or a definite pouch, covered by intact mucous membrane. The glands most often responsible for this lesion are those situated at the bifurcation of the trachea. In consequence traction-diverticula are usually found at this level and in the anterior wall of the oesophagus.

**Pressure-diverticula** are usually of obscure origin, but take their name from the presumption that they depend upon pressure from within. They occur in the dorsal or lateral wall of the oesophagus in its upper part, and form sacs varying in size from that of a pea to that of an orange, lined by normal mucous membrane. The muscular wall is sometimes deficient 1833*B*

1833$B_1$ at the site of the diverticulum, but may appear to be split longitudinally permitting the mucous membrane to become herniated between its fibres.

Diverticula are sometimes artificially produced in the
3391*L* pharynx in order to form places of concealment for coins
3391*M* and other valuables.

(*b*) **Diffuse dilatation** of the oesophagus is generally the
1834 sequel of a stricture. But it occurs sometimes in the absence
1834*A* of obvious obstruction. These apparently primary dilatations
1833 have not received a satisfactory explanation. It has been suggested that they depend upon long-continued spasmodic stricture of the cardiac orifice of the stomach.

**5. Spontaneous rupture** of the oesophagus is a rare com-
1863 plication of the act of vomiting.

**6. Post-mortem digestion** of the oesophagus by the gastric
1861 juice is not uncommon. The affected area is disintegrated and
1862 shredded, but free from any trace of inflammatory reaction.

**7. Foreign bodies.**—A great variety of foreign bodies have been found impacted in the oesophagus, the commonest being coins, bones, and artificial tooth-plates. The effects of such impaction vary with the characters of the impacted object and the site of the impaction. Sharp objects may rapidly lead to
1866 ulceration and perforation of the oesophageal wall and the subsequent formation of abscesses in the neighbourhood. The trachea may be eroded and a fistulous communication established between it and the oesophagus, in which case septic bronchopneumonia is almost certain to be excited by the passage of food-stuffs into the air-tubes. Implication of the
1867 pericardium or pleura will result in purulent pericarditis or
1868*A* empyema: perforation of the aorta, in haematemesis. Occasionally a foreign body, even though sharp and irregular in outline, may remain impacted for a long period without producing grave complications. In these circumstances it may be gradually loosened from its position by the ulceration it
1869 excites and be expelled by the mouth. It is in such instances of long-continued ulceration that fibrous structure is likely to ensue as the ulcerated area becomes gradually cicatrised.

**8. Varicose veins.**—The veins draining the lower portion of the oesophagus form a part of the portal system. In consequence lesions like cirrhosis of the liver, which are attended by obstruction in that system, are liable to produce dilatation

of the lower oesophageal veins. Occasionally these become definitely varicose and form the protrusions sometimes called "oesophageal piles." Rupture of such varicosities is one of the causes of haematemesis. 1857*B*

9. **Tumours of the oesophagus.—Innocent tumours** are rare in man; the least uncommon are small fibromata, and papillomata; leio-myomata, occurring beneath the mucous membrane; and small cysts originating in dilatation of the submucous glands. 1857*C* 1857*D* 1855*A*

Of **malignant tumours** sarcomata are very uncommon, but carcinomata frequent. 1842*A*

**Carcinoma of the oesophagus.**—The growth commences as a rule in one of three situations; (1) at or just below the level of the cricoid cartilage; (2) at the level of the bifurcation of the trachea; (3) at the lower end of the tube. Occasionally the lower end of the oesophagus is involved in a cancer which has originated in the cardiac portion of the stomach. 1844 1844*D* 1845*A* 1848 1849 1847

**Microscopic examination.**—All carcinomata which commence in the oesophagus are squamous-celled. Those which involve the oesophagus by extending into it from the stomach are either columnar or spheroidal-celled.

A cancer of the oesophagus is usually single; but sometimes discrete nodules of the neoplasm appear at some distance from the main mass. The growth infiltrates the wall of the oesophagus and narrows the lumen of the tube; it is prone to extensive ulceration. Above the stricture dilatation takes place together with hypertrophy of the muscular wall. With enlargement of the growth neighbouring structures become infiltrated, and, if hollow, may be perforated as a result of ulceration. In this way fistulous communications are established with the oesophagus. Perforation of the trachea or bronchi will set up septic bronchopneumonia; of the pleura, a pyo-pneumothorax; of the mediastinum, a mediastinal abscess or empyema; of the aorta, a fatal haematemesis; of the lungs, abscess or gangrene. 1844*D* 1844*D*$_1$ 1844*C* 1844*C*$_1$ 1844*F*

# CHAPTER XIV.

## DISEASES OF THE PERITONEUM.

1. **Peritonitis.**—(*A*) Acute. (*B*) Chronic. (*C*) Tuberculous. (*D*) Sequelae of Peritonitis. 2. **Ascites.** 3. **Tumours.**

### 1. PERITONITIS.

Following the practice adopted with regard to inflammations of the pleura and of the pericardium, the inflammations of the peritoneum are here dealt with in relation rather to their clinical than their pathological grouping. Tuberculous peritonitis, and malignant disease of the peritoneum, sometimes called "malignant peritonitis," are considered apart from the other forms.

(*A*) **Acute peritonitis.**—Whether local or general, acute peritonitis produces identical changes in the peritoneum. The earliest sign of inflammation is reddening, due to injection of the blood-vessels, accompanied by a loss of the lustre which characterises the membrane in the healthy state. Presently a sero-fibrinous exudation appears, either coating the damaged surface with a flaky coagulum, or, if its fluid constituents be abundant, accumulating in the most dependent parts of the sac as a free effusion. The next event is adhesion between the inflamed peritoneal surface and that which lies against it. If this adhesion be pronounced the area of inflammation, when originally local, becomes definitely circumscribed, and the security of the general peritoneal cavity is for the time being assured. Inflammation of the peritoneum may cease at this stage, or, if severe, may proceed to the formation of pus. In the former case the inflammatory material between the

adherent surfaces becomes organised and stretched into fibrous bands by the movements of the intestines. In the latter 1875
case the result is a localised abscess if the protective adhesions suffice to confine the purulent effusion; otherwise a general purulent peritonitis. In the latter event the whole peritoneum becomes injected, softened, and swollen, and is easily separated from the underlying viscera. The intestines are distended with gas owing to paralysis of their muscular 1990*B*
coats, and the cavity of the peritoneum contains a purulent fluid whose character varies with the nature and virulence of the infecting organism.

(*a*) **Acute general peritonitis** is invariably the result of bacterial infection.

It may be (1) primary; (2) secondary to a local lesion within the belly.

(1) **Primary acute general peritonitis.**—This is a rare variety. The commonest example of it is that due to infection by the pneumococcus. Pneumococcal peritonitis is met with most often among children. It may be an item in the course of a pneumococcal septicaemia, and occurs in association with pneumococcal infections of other serous sacs, especially the pleura and pericardium; but the peritoneum may be affected alone. If the infection is a virulent one the effusion has the appearance of a turbid serum containing flocculi of lymph, and is found under the microscope to be a watery pus teeming with pneumococci. In the less virulent cases the pus is thick and of a yellowish-green colour; fibrinous coagula are abundant in it and pneumococci relatively scanty.

In the final stages of grave diseases like cancer and Bright's disease the peritoneum may become infected by various organisms. These "terminal infections" are of little clinical moment for they are overshadowed by the gravity of the constitutional diseases of which they are incidents.

(2) **Secondary acute general peritonitis** is common. It is due to an extension to the peritoneum of an inflammation already existing within the belly and is particularly associated with the following organisms, Streptococcus pyogenes, B. coli communis, B. pyocyaneus and, when attending pelvic inflammations, the Gonococcus.

The common primary lesions are as follows:

(*a*) Virulent inflammations of abdominal viscera. Here the

acuteness of the process permits the general cavity of the peritoneum to become infected before sufficient time has elapsed to allow of the establishment of protective adhesions around the damaged zone. By far the commonest cause in this category is acute gangrenous appendicitis, but others of tolerable frequency are acute septic endometritis following childbirth, and virulent inflammations of the gall-bladder, generally associated with the presence of biliary calculi.

(*b*) Perforations of the hollow viscera. In this class come perforations of the alimentary canal owing to peptic ulcers of the stomach, to the analogous duodenal ulcers, and to typhoid ulcers of the intestine. The peritonitis depends rather upon the escape of the intestinal or gastric contents than upon the inflammation which produces the perforation.

(*c*) Rupture into the general peritoneal cavity of abscesses existing in the belly. A great variety of abscesses may produce this result, the commonest being appendix abscesses, abscesses in and about the Fallopian tubes and uterus, abscesses in or about the stomach and liver.

(*d*) Gangrene of the intestine from whatever cause. The commonest item here is gangrene from intestinal strangulation.

(*e*) Perforating wounds of the abdominal wall.

(*f*) Cancer of the bowel in its more advanced phases.

(*b*) **Acute local peritonitis.**—This is generally secondary to an inflammation of some abdominal viscus, the inflammation being limited by the establishment of peritoneal adhesions. The
1872*A* commonest causes are inflammations of the appendix, of the pelvic genitalia of the female, and of the gall-bladder; and the rupture of chronic ulcers of the alimentary canal, when such rupture has been anticipated by the establishment of adhesions.

(*B*) **Chronic peritonitis** may be (*a*) general; (*b*) local.

(*a*) **Chronic general peritonitis.**—There are three varieties: (1) "Simple," (2) Tuberculosis (*v. infra*, section C.), (3) Malignant (*v. infra*, section D.).

(1) The condition known as "**simple chronic peritonitis**" is found most often in the subjects of chronic alcoholism, but in some instances no explanation whatever is forthcoming to account for the lesions. It is marked by the deposit upon the peritoneum of a thick white opaque membrane which

can be readily detached from the underlying peritoneum. The deposit is most abundant upon the liver and spleen, and often presents pits and indentations upon its surface. The peritoneal cavity usually contains an excess of fluid, but exhibits no adhesions. 2193*A* 2193*B* 2193*D* 2297*B*

(*b*) **Chronic local peritonitis.**—This occurs in the form of a thickening of the peritoneum in the neighbourhood of the liver and spleen, with adhesions in this situation. The relations of the condition are obscure and probably varied, but it is constantly present when the liver is the seat of gummata. 2202*A* 2202*D*

(*C*) **Tuberculous peritonitis** may be (*a*) general; (*b*) local.

(*a*) **General tuberculous peritonitis** may be (1) acute; (2) chronic.

(1) **Acute general tuberculous peritonitis** implies a miliary tuberculosis of the whole peritoneum. The infection may reach the serous membrane (1) by the blood-stream—the haematogenous variety, or (2) by the lymph-channels—the lymphogenous variety. In the former case the peritoneal lesion is part of a general tuberculous septicaemia, and often produces no symptoms since the constitutional disturbance overshadows the local damage. In the latter case the peritoneum is infected along lymphatic paths from foci of tuberculosis already existent within the belly, particularly caseous mesenteric glands, tuberculous ulcers of the intestine, or tuberculosis of the uterine appendages. Such miliary tuberculosis of the peritoneum is usually attended by ascites, and some degree of adhesion between the peritoneal surfaces remains after absorption of the exudation. The lymphogenous variety of miliary tuberculous peritonitis constitutes the most favourable form of the disease. But not uncommonly, upon absorption of the fluid, the lesions assume the chronic adhesive character to be presently described. 1879 1880 1883*D*

(2) **Chronic general tuberculous peritonitis.**—The anatomical characteristic of chronic general tuberculous peritonitis is a *fibrocaseous thickening of the peritoneum with much adhesion.* The adhesion may be universal or may be broken by loculi containing a turbid fluid. It is convenient to divide cases of this class into three groups. 1874

(*a*) A group whose prominent feature is caseous infiltration most marked in the visceral peritoneum and produced by the coalescence of innumerable caseating tubercles. Infiltration of

1883*B* the great omentum is commonly pronounced and produces the
1883$B_1$ transverse mass often to be felt during life.

(*β*) A group marked by diffuse fibrocaseous infiltration affecting the parietal peritoneum principally. In this group it may be impossible to detect individual tubercles.

(*γ*) A group in which caseation of the mesenteric glands is an outstanding feature, the general involvement of the peritoneum
2281 being apparently secondary to the lesions of the glands. It is to this variety that the term "*Tabes mesenterica*" applies.

Whatever the precise type assumed by the lesion, general chronic tuberculous peritonitis leads to an inextricable matting
1883 of the intestines. Intestinal ulceration is a common adjunct, and, if severe, may produce fistulous communications between
1960 adherent coils of intestine, or faecal abscesses, or faecal fistulae, particularly in the neighbourhood of the umbilicus. Even in the early stages of this adhesive form a loop of gut may become kinked by its adhesion and produce the symptoms of acute intestinal obstruction.

(*b*) **Localised tuberculous peritonitis** may be acute or chronic, but in either case is not of much intrinsic moment. Isolated areas of the peritoneum may be the seat of miliary tubercles, apart from any general affection of the abdominal cavity or viscera. This is particularly the case with the peritoneum
2196$A_2$ covering the liver, spleen, and uterus, while large tuberculous masses are sometimes found between the liver and the diaphragm. A more chronic type of lesion, consisting of miliary tubercles often associated with fibrinous exudation and some adhesion, is commonly met with on the peritoneal surface
2012*F* of tuberculous ulcers in the intestine.

(*D*) **Sequelae of peritonitis.**—Adhesions between various viscera, or between viscera and the abdominal wall, tend to stretch in course of time and persist as fibrous bands of varying thickness and strength. Such bands are a fruitful source of internal strangulation of the intestine.

## 2. ASCITES.

**Ascites** signifies an accumulation of free fluid within the peritoneal sac. The causes of it may be (*a*) mechanical, (*b*) inflammatory and other.

(*a*) Ascites is *mechanically produced* by failure of the heart,

especially as a result of valvular disease, and is then merely a part of a general dropsy. It is also produced mechanically by all lesions which cause obstruction of the portal vein, particularly cirrhosis of the liver. The fluid in these cases is a mere transudate. It is opalescent, albuminous, occasionally (when associated with cirrhosis of the liver) blood-stained, and of a specific gravity ranging from 1010 to 1015.

(*b*) Ascites occurs as an *inflammatory exudate* in connection with tuberculous, malignant, and "simple" chronic peritonitis. The fluid from such examples is generally more turbid and more cellular than the transudate described above.

Malignant tumours of the abdominal organs, and Bright's disease in its later stages, are sometimes attended by ascites. Obstruction of the thoracic duct by the pressure of a tumour may result in a milky effusion, "Chylous ascites," the milky appearance depending upon the presence of minute globules of fat.

## 3. TUMOURS.

(*a*) **Innocent.**—Fibromata and lipomata are met with, though rarely. 1884 1884*A*

The great omentum is occasionally the seat of hydatid cysts, and of multiple cysts, apparently the result of mucoid degeneration of the omental tissue. 1893 1893*A* 1885*D* 1885*E*

(*b*) **Malignant.**—Primary malignant tumours of the peritoneum are rare. Secondary carcinomatous and sarcomatous deposits are fairly common, especially in connection with primary growths of the abdominal viscera. 1886 1886$C_2$ 1886*E* 1887*A*

# CHAPTER XV.

## DISEASES OF THE STOMACH.

1. Malformations. 2. Abnormal Position. 3. Dilatation: (*a*) Acute; (*b*) Chronic. 4. Contractions: (*a*) Local; (*b*) Diffuse. 5. Rupture. 6. Perforation. 7. Foreign Bodies. 8. Inflammations. 9. Atrophy. 10. Post-mortem Digestion. 11. Haemorrhages. 12. Gastric Ulceration: (*a*) Haemorrhagic Erosions; (*b*) Simple or Peptic Ulcer; (*c*) Malignant Ulcer. 13. Stricture of the Pylorus: (*a*) Malignant; (*b*) Simple or Fibrous; (*c*) Mechanical; (*d*) Congenital Hypertrophic Stenosis. 14. Tumours.

**1. Malformations** are rare. Absence of the organ, abnormally small size, and complete occlusion of the pylorus have been observed, usually in association with other congenital defects.

**2. Abnormal position** occurs (*a*) when there is a complete transposition of the viscera, in which case the fundus of the stomach lies in the right hypochondrium, the long axis of the viscus passing from right to left.

(*b*) When a congenital deficiency of the diaphragm gives access to the cavity of the thorax (congenital diaphragmatic hernia). In this condition, the stomach, with or without other
2163*F* abdominal viscera, may lie within the chest.

**3. Dilatation.**—(*a*) **Acute dilatation** is the result either of overloading of the stomach, or of paralysis of the gastric
1906*A* muscle. The latter sometimes attends acute toxaemias, *e.g.* pneumonia, but may occur without any known cause.

(*b*) **Chronic dilatation** in its lesser degrees is a constant

accompaniment of "nervous dyspepsia." It is then a local expression of the general loss of nervous "tone" which characterises the subjects of this disease, and is in its essence a functional disorder. But if it be long continued, organic inflammatory changes may appear owing to the prolonged retention of food within the stomach. The more pronounced grades of dilatation depend upon obstruction of the pylorus. 1923*A*

The cavity of a dilated stomach in an extreme case may accommodate as much as ten pints. In such instances, the lesser curvature is straightened out, and lies parallel to the vertebral column in direct prolongation of the line of the oesophagus. A short distance above the pylorus, a sharp turn directs the lumen upwards and to the right. The greater curvature occupies the whole of the left hypochondrium and flank, and may even reach the symphysis pubis. 1906*A*

4. **Contractions** of the stomach may be localised or diffuse. The localised contractions are described in the sections devoted to gastric ulcers and their effects. The commonest cause of diffuse contraction is infiltration of the stomach by cancer, which produces the appearance known as the "leather-bottle stomach" (*q.v.*).

5. **Rupture** of the stomach is generally due to direct violence exerted upon the stomach when it is loaded with food. 1900

6. **Perforation** may attend gastric ulceration, both simple and malignant, more often the former.

7. **Foreign bodies.**—In this connection it is only necessary to describe the "hair-balls" occasionally found in the stomachs of hysterical women who are in the habit of eating their own hair. These "hair-balls" are oval masses of hair bound together by mucus and particles of food. Series of calculi. 285*A* 285*B*

8. **Inflammation of the stomach.**—Although much is heard in medicine of "gastritis," little is really known of inflammations of the stomach, and it is probable that a large proportion of the symptoms embraced by the term "gastritis" depend upon purely functional derangements. The demonstrable inflammations of any frequency are those depending upon the following causes: (1) the ingestion of irritant poisons; (2) microbic infection; (3) the extension to the stomach of an inflammation existing in some adjoining structure. It is necessary to add to these simple gastric ulcer, but this stands

in a category by itself and will receive detailed consideration presently.

(1) **Irritant poisons.**—Mild irritants produce a catarrhal inflammation evidenced by congestion and swelling of the gastric mucosa, and by a copious discharge of mucus. At the other extreme come the lesions due to powerful corrosive poisons. These lesions vary in appearance with the nature of the poison, but all exhibit sloughing of the gastric mucosa.
1944*A* The slough produced by strong sulphuric acid is hard, dry,
1946*B* and of a deep-red or black colour; that produced by hydro-
1946*C* chloric and nitric acids is yellowish or black; that produced
1947 by oxalic acid is ashen-grey or brown. Carbolic acid whitens
1948*A* and corrugates the mucous membrane, while caustic alkalies
1948*B* reduce it to a pulp. The mucous membrane in the neighbour-
1949*D* hood of the lesions is the seat of capillary haemorrhages and
1949*E* of active inflammatory changes.

*After-effects of corrosive poisoning.*—When the injury produced by strong corrosive poisons is not immediately fatal, cicatricial contractions may lead to gross deformities which vary with the distribution and extent of the tissue-
1907*C* destruction.

(2) **Microbic infections.**—Occasionally, but rarely, the infection of diphtheria travels down the oesophagus and involves
1918*B* the stomach. A similar membranous inflammation may attend
1918$B_1$ other infections, such as scarlet fever, but is then extremely rare. Tuberculous ulcers of the stomach are occasionally encountered, but typhoid ulcers practically never. Stomachs dilated as a consequence either of pyloric obstruction or of functional weakness are liable to chronic inflammation, the result of bacterial decomposition of their retained contents.

(3) It is obvious that the stomach may share in inflammations existing in its neighbourhood, such as cholecystitis. But the lesion of the stomach is then so subordinate to the primary mischief as to make it almost negligible.

9. **Atrophy** of the stomach. In certain ill defined conditions the stomach undergoes a generalised atrophy affecting both the glands and muscular wall.

10. **Post-mortem digestion** of the stomach is commonly seen. It leads to disintegration and sometimes perforation of the stomach wall, and occasionally to involvement of neighbour-
1895 ing structures. The lesions are easily identified by their

shredded appearance and by the absence of all inflammatory reaction.

**11. Haemorrhages** in the stomach may be (*a*) arterial, (*b*) venous, (*c*) capillary.

(*a*) Arterial bleeding is generally caused by the erosion of an artery owing to simple gastric ulceration.

(*b*) Venous bleeding depends most often upon the rupture of varicose veins dilated in consequence of some obstruction to the portal circulation such as that induced by cirrhosis of the liver. 1905

(*c*) Capillary haemorrhages into the submucous tissue commonly attend congestion of the stomach such as occurs in cirrhosis of the liver or chronic valvular disease of the heart. They are met with also in those who have died of 1918*C* acute infections, and sometimes occur without any known 1918*E* associations. They may be punctate or diffuse, and are the 1918*D* antecedents of the "haemorrhagic erosions" to be presently described. *Vomiting of blood* (haematemesis) may be due, as far as the stomach is concerned, to *gastric ulcer*, or to the *rupture of varicose veins*, or to *oozing from submucous haemorrhages*.

**12. Gastric ulceration.**—(*a*) Haemorrhagic erosions, (*b*) simple or peptic ulcer, (*c*) malignant ulcer.

(*a*) **Haemorrhagic erosions.**—When a haemorrhage occurs into the gastric submucous tissues the damaged epithelium covering the site of the extravasation is likely to be destroyed by the digestive juices. In this way are formed small superficial ulcers situated in the haemorrhagic zone, and called 1908*C* "Haemorrhagic erosions." 1918*D*

(*b*) **Simple or peptic ulcer.**—This variety of ulceration is more frequent in females than in males and is particularly associated with anaemia in young women. Males are affected at a later age, as a rule, namely between the fortieth and sixtieth years. The ulcers may be acute or chronic. In the case of young women they are usually more or less acute in their course, while middle-aged or elderly men may exhibit post-mortem a chronic ulcer unsuspected during life. The origin of these ulcers is not known, but it is believed that some local lesion, such as a submucous haemorrhage, embolism or thrombosis of a small vessel, or a bacterial infection, damages the mucous membrane and exposes it to the action

of the gastric juice. Sometimes acute peptic ulcer of the duodenum, and less often, of the stomach, follows severe
1908*A* burns.
1911*A* The simple ulcer is usually single but may be multiple.
1913*B* In its typical form it is oval or circular in outline and diminishes in size from the surface downwards. The shelving
1912 walls are often terraced, that is to say marked by a series of ledges. A chronic ulcer is attended by thickening and induration of the affected site, while the base of the ulcer
1914*B* is hard and smooth, and formed, according to the extent of
1911 tissue destruction, by the muscular coat, the peritoneal coat,
1913*B* or adjacent structures, such as the pancreas. Peptic ulcers
1914*C* are found only in the stomach and duodenum. In the stomach the favourite site is the neighbourhood of the lesser curvature towards its pyloric extremity.

**Effects of peptic ulcer.**—Many simple ulcers of the stomach become healed, leaving only a puckered scar in the mucous
1918 membrane and subjacent tissues, while if the ulceration has involved the peritoneum it is usual to find adhesions established between the damaged site and the neighbouring organs. In cases of longer standing the process of healing is often attended by a pronounced degree of cicatricial contraction and gross deformity of the viscus. If the ulcer has been situated near the pylorus the result may be stricture of the
1909 pyloric orifice and dilatation of the stomach : if it has involved the stomach at or about the middle of its length the viscus may be divided by the resulting scar into two sacs communicating by a narrow passage. From its resemblance to an
1907*B*$_1$ hour-glass this deformity has been termed "hour-glass con-
1918*A* striction of the stomach."

The **complications** which may attend gastric ulcers in the phase of activity are the following :

(1) **Perforation into the general peritoneal cavity.**—This accident attends both the acute and the chronic forms of ulcer, but especially the acuter ulcers met with in young women. It is particularly liable to happen when the ulcer involves the anterior wall, because protective adhesions with neighbouring structures are not readily established in this
1912 situation. The result of such a perforation, if untreated, is
1913*B* general peritonitis.

(2) **Perforation occurring subsequently to adhesion** of the

stomach to neighbouring parts leads to the formation of a localised abscess, generally containing gas—"sub-diaphragmatic abscess." For the reasons given these abscesses are generally due to perforation of the posterior wall. They may lie above or below the liver and often excite a basal pleurisy owing to extension of inflammation along the lymphatics of the diaphragm. On the other hand they may track downwards, producing abscesses in other parts of the belly or in the pelvis. 1907*C* 1909

(3) **Haemorrhage.**—The commonest cause of arterial bleeding in the stomach is the erosion of an artery by a simple gastric ulcer. The vessel most frequently attacked is one or other artery of the lesser curvature, but branches of the splenic artery, and occasionally the latter itself, may be the site of the haemorrhage. The erosion of the vessel is sometimes preceded by an aneurysmal dilatation of the weakened area of its wall, such as is seen in tuberculous cavities in the lung. 1907*A* 1907$A_1$

(4) **Thrombosis of the splenic artery** is an occasional result of ulcers upon the posterior surface of the stomach which have excited adhesion between the stomach and the pancreas.

(5) A simple gastric ulcer, or the scar left by one, may be the starting point of *carcinoma.*

(*c*) **Malignant ulcer,** *v. infra* "Tumours of Stomach."

**13. Stricture of the pylorus** is due to the following causes:

(1) Malignant growths.

(2) The scarring which attends (*a*) simple chronic ulcer situated near the pylorus, (*b*) ulceration due to corrosive poisons. 1908*B* 1914*B*

(3) Kinking, the result of adhesions excited by gastric ulcers in the past. 1914*A*

(4) **Congenital hypertrophic stenosis of the pylorus.**—This disease may date from birth and be truly congenital, but more often attacks infants during the second month of life. The chief symptom is perpetual vomiting leading to an exhaustion which often proves fatal. The stomach is sometimes dilated as a whole, sometimes contracted, but in any case the pyloric channel is narrowed and surrounded by a greatly thickened muscular coat. The cause of this hypertrophy is in doubt, but it depends in all probability upon long-continued or frequently-recurrent spasm of the muscle. 1914*A* 1914*C*

**Microscopical examination** shows no abnormality of the pyloric region except pronounced thickening of the sphincter.

**14. Tumours of the stomach.**—(*a*) Innocent; (*b*) malignant.

(*a*) **Innocent tumours** are rare. The commonest are polypoid outgrowths of the mucous membrane. These occur singly or in numbers, and in the latter case may by coalescence produce irregular elevations of considerable size upon the inner aspect of the stomach.

**Microscopical examination.**—Such tumours are generally papillomatous outgrowths of the mucous membrane, and resemble it in structure.

(*b*) **Malignant tumours** are common. The large majority are carcinomata, but round-celled sarcomata are met with in children and young people, usually occurring as diffuse infiltrations of large areas of the gastric wall.

A gastric carcinoma is in about 60 per cent. of cases, situated in the pyloric region, either actually in the canal, or on the anterior or posterior wall in its immediate neighbourhood; but no portion of the organ is exempt. The naked eye appearances vary considerably, partly with the histological character, partly with the distribution of the cancerous tissue. In the region of the pylorus the commonest varieties are two: (1) a roughly circular projection surrounding a depressed carcinomatous ulcer, readily distinguished from a simple ulcer by its irregular fungating edges; the margin of the projecting tumour slopes gradually towards the stomach, but is often steep and abrupt towards the duodenum; (2) an annular infiltration of the walls of the pyloric canal rendering them three or four times their normal thickness. At some part of this infiltrated area there is often either a small ulcer or a warty outgrowth.

Elsewhere than in the pyloric region the growths appear either as sprouting cauliflower masses, or as crateriform ulcers with widely infiltrated margins.

A peculiar form of gastric carcinoma has received the name of "*leather-bottle*" stomach, from the extreme thickness and rigidity of the infiltrated walls, and the wide, and sometimes universal, distribution of the cancerous tissue. Such "leather-bottle" carcinomata are of the so-called "colloid" variety, *i.e.* they have undergone mucoid degeneration. The cavity of the stomach is much reduced in size. Ulceration of the infiltrated walls is usual but not invariable.

(*N.B.*—Owing to the varieties of naked eye appearances the old descriptive names "encaphaloid" or "medullary," and "scirrhous" carcinoma are still in use to denote respectively the soft fungating tumours, and the hard fibrous infiltrations. These names indicate only the naked eye characteristics of the growth.)

**Microscopic examination.**—From the histological standpoint there are three varieties of gastric carcinoma. (1) Adenocarcinoma, distinguished by an abundance of atypical gland-tubules, set in a scanty framework of connective-tissue. (2) A variety in which the alveoli are filled with closely packed masses of cells for the most part spheroidal in shape, but here and there showing tracing of a columnar form; the connective-tissue again is scanty. (3) Scirrhus, *i.e.* a carcinoma in which there is a great overgrowth of connective-tissue, with comparatively few cells. The cells occur in small groups and chains, separated from each other by bands of stroma, and are spheroidal or polygonal in shape.

**Colloid carcinoma.**—Any of these histological types may undergo "colloid" degeneration, but it is less frequent in scirrhous cancer than in the other two varieties. A "colloid" cancer has a gelatinous semi-transparent look due to the 1935*A* presence of masses of "colloid" (mucoid) material. 1936*A*

Microscopically the growth consists of a structureless hyaline substance in which lie a few cancer cells which have not yet degenerated.

Growths in the neighbourhood of the pylorus are usually of the scirrhous variety; adenocarcinomata, and the richly cellular carcinomata form the cauliflower excrescences and crateriform ulcers of the body of the stomach.

Lastly, squamous-celled carcinomata are occasionally found at the cardiac orifice. These invariably originate in the mucous membrane of the oesophagus, and involve the stomach by a 1849 process of direct extension. 1850

**Complications of cancer of the stomach.**—(1) The lymphatic glands along the lesser curvature are implicated early, and occasionally form large masses of growth, far in excess of the original tumour. (2) Metastatic deposits, especially in the liver, are common. (3) Stricture of the pylorus, with sub- 1923*A* sequent dilatation of the stomach, is common. (4) Perforation 1923*B* of a cancerous ulcer may lead to general peritonitis, to the 1934*B* formation of a sub-diaphragmatic abscess, to a pyo-pneumo- 1931*B* thorax, or a pyo-pneumopericardium, or to a gastro-colic, or 1931 gastro-duodenal fistula. 1931*A*

# CHAPTER XVI.

## DISEASES OF THE INTESTINES AND OF THE APPENDIX VERMIFORMIS.

1. Malformations. 2. Inflammations. (*A*) Non-specific: (*a*) Duodenal Ulcer, (*b*) Irritant Poisoning. (*B*) Specific: (*a*) Catarrhal Enteritis and Colitis, (*b*) Typhoid Fever, (*c*) Cholera Asiatica, (*d*) Tuberculosis, (*e*) Syphilis, (*f*) Actinomycosis, (*g*) Dysentery. 3. Vascular Lesions: (*a*) Infarction, (*b*) Submucous Haemorrhages, (*c*) Haemorrhoids. 4. Degeneration. 5. Foreign Bodies. 6. Intestinal Sand. 7. Intestinal Parasites. 8. Tumours. **Diseases of the Appendix Vermiformis.**—1. Foreign Bodies and Concretions. 2. Inflammation. 3. Tumours.

### 1. MALFORMATIONS.

Congenital malformations are not rare; those most commonly met with are:

(1) **Stenosis** or actual occlusion of the lumen of the small intestine. Occlusion may be due to a simple failure of development by which the gut is divided into two totally disconnected
3635*B* lengths, both blind at their adjacent extremities; or the patent
3635*C* portions may be united by a fibrous band. Occasionally, especially in the duodenum, the lumen is completely or partially closed by a diaphragm passing inwards from the whole circumference of the gut. This diaphragm consists of a fold of the submucosa lined on both sides by normal epithelium. The duodenum above the obstruction is dilated and gives the
3635*D* appearance of a second stomach. The most usual sites of
3635*E* developmental anomaly are the duodenum, and a spot near

the ileocaecal junction; but in some cases the lumen is stenosed or absent in many places throughout the length of the gut. Some authorities attribute certain of these deformities to inflammatory lesions of the peritoneum, strangulation by bands, and other similar accidents occurring during foetal life.

(2) **Meckel's diverticulum.**—This is due to the persistence, more or less complete, of the omphalo-meseraic duct, and occurs in about 2 per cent. of all bodies examined after death. It is a diverticulum of the ileum and is identical with this portion of the gut in structure. It takes its origin from a point within three feet of the ileocaecal junction and varies considerably in length and capacity. In some cases it is small and short, with a blind distal extremity either unattached or 3637
connected with the umbilicus by a fibrous cord. In others it 3638*B*
extends up to and opens at the umbilicus, being fully patent throughout its length. When the distal extremity of the diverticulum is attached to the umbilicus a band is formed by which a loop of intestine is occasionally imprisoned and strangulated. In this way a Meckel's diverticulum may cause intestinal obstruction. The umbilical extremity of a patent diverticulum may form a polypoid excrescence at the navel, owing to eversion of the mucous membrane. Such an excre-
scence is called an "umbilical polyp, or adenoma," and may 3638*G*
discharge faecal material. Where the duct is obliterated both at the ileum and at the surface or at some point short of this, being nevertheless patent in part of its length, the patent portion may be distended to form a cyst, lined with intestinal mucous membrane, and situated either in the wall of the abdomen in the region of the umbilicus, or within the peritoneal cavity (enterocystoma).

(3) **Displacement of the large intestine.**—The colon, instead of observing its usual arrangement, may lie obliquely across the abdominal cavity, or may be so disposed that its ascending and descending portions lie parallel to one another on the right side of the cavity. The caecum, owing to undue length of its mesentery attachment may have an undue mobility and lie in unusual situations.

(4) **Congenital dilatation of the colon** with hypertrophy. This may occur without any obvious obstruction to account for it, and is probably, in essence, a developmental anomaly, though some of the changes observed in the coats of the viscus, such 1952*C*

as the hypertrophy, may be secondary to the long continuance of faecal accumulations within the dilated gut. The dilatation may reach an enormous degree.

3640*A* (5) **Opening of the rectum into the urethra or vagina.**

(6) **Imperforate anus** (atresia ani).—The invagination at the anus which normally establishes communication with the gut in the process of development may fail to make the connection. In this case the lower end of the bowel is blind and much distended by meconium. The anal depression may be
3640 well-formed, and half-an-inch deep or more, the interval between
3642 its extremity and the gut being occupied by fibrous tissue.

(7) **Atresia recti.**—The rectum may be absent, the gut ending blindly at the brim of the pelvis.

## 2. INFLAMMATIONS.

(*A*) Non-Specific. (*B*) Specific.

(*A*) **Non-specific inflammations.** (*a*) **Duodenal ulcer.**—A variety of ulceration precisely analogous to the simple or peptic
1965 of the stomach is met with in the duodenum. The scarring consequent upon the healing of such an ulcer may produce
1965 stricture of the duodenum. Perforation of the intestinal wall
1966 may take place, leading to a localised abscess or general
1967*A* peritonitis, and erosion of an artery may lead to intestinal
1969 haemorrhage. Duodenal ulcer is an occasional sequel of severe
1969*A* burns, but the nature of the association is not known.

(*b*) **Irritant poisoning.**—Swallowed irritants which reach the intestine produce an inflammation proportionate to the activity
2042*B* of the irritant. In the milder cases the inflammation is catar-
2043 rhal, evidenced by swelling and injection of the mucosa, and an
2042*A* increased secretion of mucus. At the other extreme comes
2044*A* the extensive destruction produced by corrosive poisons. The lesions produced in the latter case are similar in kind to those already described in the section upon corrosive poisoning of the stomach.

(*B*) **Specific inflammations.** (*a*) **Catarrhal enteritis and colitis.**—A lesion similar to that described above in connection with mild irritant poisoning may be produced by the action of micro-organisms. The best example is the lesion associated with the epidemic diarrhoea of infants, which is so common

and so fatal a disease in a hot summer. The precise organisms responsible for the disease have not yet been fully identified, but its bacterial nature is unquestioned. In addition to the general catarrhal swelling of the mucous membrane, there is often a pronounced affection of the solitary follicles. These are swollen out of proportion with the remainder of the mucosa, and appear as innumerable circular elevations. To this variety is given the name "follicular enteritis." Such inflammations as these may become chronic, and lead to ulceration or atrophy of the mucous membrane. 1962*B* 1962*C* 1962*D*

(*b*) **Typhoid fever.**—Typhoid fever is due to the bacillus typhosus. The lesions consist essentially of an inflammation of the lymphatic tissues of the gut, though the neighbouring mucous membrane suffers to a variable extent. The stress of the disease falls upon the lower part of the ileum and the upper part of the colon together with the caecum and, sometimes, the appendix. For descriptive purposes it is usual to divide the course of the inflammation into three stages, as follows :

(1) **Hyperplasia.**—In this stage the Peyer's patches and solitary follicles are congested and swollen. They project from the mucous membrane, forming flat-topped elevations, greyish-white in colour, and often furrowed or pitted upon the surface. This stage of the lesion is reached at or about the tenth day of the disease. Resolution may now occur bringing the inflammation to an end, but if the latter is severe it proceeds to the next stage, namely necrosis. 1988 1990*A*

(2) **Necrosis and ulceration.**—The stage of necrosis is marked by the death, in bulk or in part, of the damaged areas. Their surface becomes frayed and ragged, and often discoloured by bile. Presently the dead tissue is exfoliated, sometimes piecemeal, sometimes *en masse*. This "sloughing" commonly commences towards the end of the second week. The "slough" may be quite superficial or involve the entire thickness of the gut-wall, and, though generally limited to the Peyer's patches and solitary follicles, may implicate the neighbouring mucous membrane. The exfoliation of sloughs is accomplished by a process of ulceration. Beneath each slough the inflammatory reaction produces a layer of granulation tissue, which, by phagocytosis and digestion, effects the separation of the dead material. The result of such separation is an ulcer, with soft, irregular, and often undermined edges, and a floor generally 1989 1997 1998 1999

2004 supplied by the muscular coat. These ulcers, representing as
2004*B* they do for the most part the sites of Peyer's patches, are *disposed longitudinally*, differing in this respect from the common form of tuberculous ulcer of the intestine which lies
2012 transversely to the long axis of the gut. Ulceration appears towards the end of the second week, continues during the third and fourth weeks, and throughout this period may give rise to haemorrhage, the result of arterial erosion. If the ulcer be deep enough to implicate the peritoneum, the latter
1990*B* often develops a layer of inflammatory lymph upon its external
2006 surface. Later it may give way completely, the result of such
2006*C* perforation, if untreated, being general peritonitis.

(3) **Healing.**—Repair may begin at any stage of the inflammation, it being no rarity to see all grades of damage and
1990*B* repair existing simultaneously in different parts of the intestine.
2006 Areas which have never reached the stage of necrosis are often left pigmented. Ulcers are healed by a gradual in-
2006*A* growth of the epithelium from their edges, and finally present a smooth, depressed surface of a slate-grey colour. Gross scarring and strictures, such as follow tuberculous and dysenteric lesions, are unknown in connection with typhoid fever.

(*c*) **Cholera asiatica.**—This disease is marked by an intense inflammation of the intestinal mucosa. The latter is congested, and may show points of submucous haemorrhage. The Peyer's patches and solitary follicles show a pronounced degree of swelling, and project above the general level of the mucous membrane. The disease is often so rapidly fatal as to antici-
1957 pate the appearance of ulcers, but these may attend the more
1958 chronic examples of the disease.

(*d*) **Tuberculosis** of the intestine is most often an accompaniment of pulmonary tuberculosis, the infection being derived from infected sputa which have been swallowed. But not uncommonly, especially in childhood, intestinal lesions appear in the absence of infection within the thorax or elsewhere. These cases are by most authorities attributed to the ingestion of tuberculous milk, and are regarded as infections by the bacillus of bovine tuberculosis. The region of the intestine most often affected by tuberculosis is the lower part of the ileum, but the caecum, colon, and rectum are far from being immune. The lesions commence in the lymphatic structures, the Peyer's patches and solitary follicles. A preliminary phase of swelling

is succeeded by a localised degeneration, whose subsequent spread produces an ulcer with undermined edges and a tendency to *extend transversely*, involving an ever-increasing segment of the circumference of the gut. But occasionally 2011
tuberculous ulcers are longitudinally disposed. The peritoneal 2012
surface of such an ulcer usually has a somewhat puckered 2012*A*
appearance, and exhibits a number of miliary tubercles visible 2012*B*
to the naked eye. The lymph-channels are affected, and the 2012*F*
lymphatic glands serving the affected district commonly become 2012$F_1$
caseous. The path of infection between these glands and the 2012*C*
ulcer is often clearly indicated by a row of miliary tubercles. 2012$C_1$
With the involvement of the peritoneal surface of an ulcer, adhesions appear between the affected coil of gut and its neighbours. For this reason, even though an ulcer perforates the gut, as it often does, general peritonitis seldom ensues, the site of perforation being shut off from the general cavity of the peritoneum. But ulcerous fistulae between contiguous and adherent coils of gut are quite common, as are localised faecal 1968
abscesses communicating with the lumen of the gut. Such abscesses may discharge their contents through the abdominal wall and produce faecal fistulae. Although tuberculous ulcer- 1960
ation of the intestines is a grave disease, healing may take 2016
place. It is very liable to be attended by cicatricial contraction 2016*B*
of the affected zone. 2018*A*

(*e*) **Syphilis** of the intestine is most frequently a tertiary manifestation. It appears in the form of gummatous ulcera-
tion of the rectum and anus. The slow healing of these ulcers 2059
is peculiarly apt to lead to stricture, the usual site being from 2061
one to three inches above the anal margin. 2057*B*

Hereditary syphilis is often attended by condylomata at the anus, but these affect the skin rather more than the mucous surface.

Primary chancres are occasionally observed in the rectum.

(*f*) **Actinomycosis of the intestine.** — The Ray-fungus (streptothrix actinomyces) most often finds its entry into the body with the food or with air, and hence its most frequent situations are either the cavity of the mouth, or the stomach, intestines, or lungs. In the intestines the caecum and appendix 2035*C*
are the portions of gut most often affected in the first instance. Subsequent dissemination may lead to the involvement of other organs, especially the liver.

The process commences with the appearance of raised whitish nodules, prone to ulceration and disintegration. It is attended by much inflammatory thickening and the formation of abscesses. The pus from such abscesses contains the yellow granules of fungus which characterise the disease.

(*g*) **Dysentery.**—The term "dysentery" is somewhat loosely applied to various ulcerative inflammations of the large intestine. It is certain that the disease clinically recognised as dysentery is due to several different agents, two of which have been identified, namely the amoeba dysenteriae, and the bacillus dysenteriae of Shiga and Flexner, both of these being common
1970*B* causes of tropical dysentery. In England epidemics of ulcerative colitis sometimes appear in institutions for the sick,
1970*A* especially asylums for the insane (asylum dysentery). Ulcerative colitis appears also as an occasional complication of grave constitutional maladies such as cirrhosis of the liver.

There is no precise distinction to be drawn between the lesions produced by the amoeba and bacillus of dysentery on the one hand, and ulcerative colitis of unknown cause on the other. The morbid appearances vary with the intensity and extent of the infection. At times the rectum and sigmoid flexure are affected alone, at others the whole of the large intestine and even some portions of the ileum. Where the infection is recent and not very severe, the mucous membrane
1973 shows intense congestion and often minute ulcers. It is much swollen and may be dotted with small haemorrhages, while its
1982 surface is covered with an exudation of blood-streaked mucus.
1970*B* In a short period, however, portions of the affected mucous
1987*B* membrane become necrotic and begin to slough, exposing the
1987*D* muscular coat. The resulting ulcers are widely distributed and
1987*E* are separated from each other by islets and patches of surviv-
2019*A* ing mucous membrane. These remnants may form prominent velvety excrescences (colitis polyposa). The irregular distribution of the necrosis, and its superficial character, are marked features of amoebic dysentery.

In severe cases the process does not stop with the destruction of the mucous membrane, but involves the muscular coat, and leads to deep and persistent ulceration spreading even to the peritoneum. It is said that ulceration is comparatively rare in the bacillary form of the disease, and common in amoebic dysentery.

**Sequelae of "dysentery."**—In slight cases the mucous membrane is probably regenerated to a considerable extent, although some degree of replacement by fibrous tissue is always present; when the deeper layers of the wall have been affected the resulting fibrosis may lead to cicatricial stricture of 1984
the gut. Stricture is said to be an uncommon sequel of the 1986
amoebic form.

A large solitary abscess in the liver is a frequent sequel of the amoebic variety, the amoebae being found in the walls of the abscess: in the bacillary form the large liver abscess is rare, but pylephlebitis and suppuration in the portal system are common.

## 3. VASCULAR LESIONS.

(*a*) **Infarction.**—Infarction due to embolism, such as occurs so commonly in the lungs, spleen, and kidneys, is not a prominent lesion of the intestine. But the appearance of a large haemorrhagic infarction is sometimes seen as a result of thrombosis of the superior mesenteric vein or its branches. In this case a portion of the gut, varying in length with the importance of the vessel affected, is the seat of a copious extravasation of blood within its tissues. The affected portion has a deep-red, purple, or black appearance, while the veins serving it are filled 1956*G*
with clotted blood. 1956*H*

(*b*) **Haemorrhages into the mucous membrane.**—Small mucous haemorrhages attend many acute inflammations of the intestine. They occur also in the course of pyaemia, probably 1956$E_1$
as a result of minute emboli (haemorrhagic erosion), and in 1956$E_2$
purpuric diseases. 1987*I*

(*c*) **Haemorrhoids** or piles are varicose dilations of the veins of the anus and lower part of the rectum. They are commonly attributed to obstruction somewhere in the portal system such as that produced by carcinoma of the rectum, cirrhosis of the liver, or the pressure of a pregnant uterus or of a rectum habitually loaded with faeces. Nevertheless they occur quite frequently in persons who present no evidence of such obstruction. According to their situation without or within the anal orifice, piles are spoken of as external or internal. *External piles* are covered by skin, and when not inflamed form shrivelled projections of skin about the anal orifice. *Internal piles* may 2076
be capillary, venous, or rarely, arterial. Capillary piles form 2076*B*

small flat projections with a papillated surface which bleeds
2075 easily. Venous piles, the commonest of the internal varieties, are larger than the preceding, and often pedunculated. They do not bleed so readily as the capillary form. Arterial piles are rare.

**Complications of piles.**—(*a*) Haemorrhage, due either to oozing from capillary piles or rupture of the other varieties.

(*b*) **Inflammation.**—An inflamed pile forms a tense, purple, and extremely tender tumour. A common sequel of such
2077 inflammation is thrombosis of the blood within the dilated
2075 vein, and occasionally ulceration of the mucous membrane covering it.

## 4. DEGENERATION.

**Amyloid degeneration** is the only form requiring special mention here. It is associated with a similar degeneration in other organs, and is most marked in the duodenum and jejunum, giving the mucous membrane a more transparent and whiter appearance than is normal. The amyloid deposit is most marked in the immediate neighbourhood of the blood-vessels of the villi.

## 5. FOREIGN BODIES.

A variety of foreign bodies may be impacted in the intestine and produce intestinal obstruction, inflammation, or ulceration. Foreign bodies which are swallowed and are small enough to escape from the stomach are generally passed per anum. But concretions of different kinds may be produced in the intestine or reach it from neighbouring organs such as the gall-bladder. Such concretions are called *enteroliths* and exist in four main varieties:

Series of calculi. 282*A* 282*B* 282*C*
(*a*) Heavy hard concretions concentrically laminated, and of a yellow, brown, or white colour. These are composed of magnesium and other phosphates with an admixture of mucus and faecal material; sometimes the faecal element is predominant.

285*A* 285*B*
(*b*) Light porous masses consisting of hair or vegetable fibres united by faecal material.

$282B_1$
(*c*) Concretions consisting of insoluble drugs administered by the mouth over a long period, *e.g.* bismuth, magnesia, or chalk.

(*d*) Gall-stones. 264*A*

Small stones may reach the gut by way of the common bile duct. Large stones sometimes gain access to the gut directly from the gall-bladder by a process of ulceration. They may cause intestinal obstruction. 274*B* 2030 2030*A*

## 6. INTESTINAL SAND.

Gritty particles are occasionally passed from the intestine in large quantities. They are of two varieties, known respectively as "False" and "True" intestinal sand.

"*False*" sand consists of woody particles of certain fruits, especially pears. "*True*" sand is composed of salts of calcium phosphorus, magnesium or iron. These particles are sometimes brilliantly coloured. Series of calculi. 285 285*C*

## 7. INTESTINAL PARASITES.

(*A*) **Cestodes or tapeworms.**—(1) Taenia saginata, *v.* p. 27. (2) Taenia solium, *v.* p. 27.

(*B*) **Nematode or round worms.**—(1) Ascaris lumbricoides, *v.* p. 30. (2) Oxyuris vermicularis, *v.* p. 30. (3) Ankylostoma duodenale, *v.* p. 31. (4) Trichina spiralis, *v.* p. 32. 1956*A*

## 8. TUMOURS OF THE INTESTINE.

(*A*) INNOCENT. (*B*) MALIGNANT.

(*A*) **Innocent tumours.**—Innocent tumours of the intestine are usually pedunculated and form "*polypi*"[1] projecting into the lumen of the gut. Such polypi may be single or multiple. The small intestine, especially the upper part of it, is occasionally the seat of innumerable minute polypoid excrescences fringing the valvulae conniventes. Coarser polypi appear in equal abundance in the large intestine. 2019*F* 2019*G* 2019*E* 2019$F_1$ 2019$B_1$

**Varieties of polypi.**—(1) Circumscribed tumours covered by normal intestinal mucosa. There are three main varieties, viz.:

(*a*) Fibroma; (*b*) lipoma; (*c*) adenoma. Adenomatous polypi are found most often in the rectum. 2019 2019*D*

Histologically rectal polypi consist of closely-set glandular tubules lined by columnar epithelium. 2019$D_1$ 2062*A*

[1] A "polypus" is a stalked tumour of any kind, whether innocent or malignant. But most polypoid growths are innocent.

2019$B_1$ (2) Pedunculated hypertrophied folds of mucous membrane
2019$E$ or "*simple papillomata.*"

2063$A$ (3) Cauliflower-like shaggy growths consisting of interlacing
2063$B$ villous proliferations of the mucous membrane. These affect the rectum as a rule and are called "*compound papillomata.*"

A papillomatous condition of the intestine may be produced
2065$C$ by chronic irritation such as that resulting from the presence
2065$D$ in the intestinal wall of the eggs of Bilharzia haematobia.

Innocent tumours are sometimes found in association with
2065$A$ carcinoma. The relation between the two is not fully under-
2065$E$ stood, but it is known that tumours originally innocent may assume malignant characters.

($B$) **Malignant tumours.**—($a$) Sarcoma; ($b$) carcinoma.

($a$) **Sarcoma** of the intestine may be primary or secondary, more often the former. It is met with chiefly in the lower part of the ileum as a whitish tumour infiltrating and thicken-
2027$L$ ing the gut-wall and often thus producing stenosis of the
2027$M$ lumen, with a corresponding degree of intestinal obstruction.

($b$) **Carcinoma** of the intestine is almost invariably primary. The seats of election are the lower three inches of the rectum, and the three flexures of the large intestine, but no part of the gut is immune.

For descriptive purposes these growths may be divided into two groups: ($a$) the fungating tumours; ($b$) the infiltrating, scirrhous tumours.

2069 ($a$) **Fungating tumours.**—In this case the growth forms a
2069$A$ fungating ulcerated projection into the lumen, the latter
2027$F$ becoming blocked by the mere bulk of the growth.

2027$F_1$ ($b$) **Scirrhous tumours.**—Here the gut-wall is narrowed by
2027$K$ an annular constriction of cicatricial aspect, the whole circum-
2027$K_1$ ference of the gut being infiltrated and shrunken.

In the intestine, as in the stomach, carcinomata show a
2027$G$ pronounced tendency to undergo "colloid" degeneration. In this case the affected area has on section a peculiarly translucent appearance. A growth thus degenerated is called a "colloid cancer."

Microscopically intestinal cancers are generally columnar-celled, and of the glandular type—adenocarcinomata.

Secondary deposits occur in the peritoneum, liver and mesenteric glands.

## DISEASES OF THE APPENDIX VERMIFORMIS.

The appendix vermiformis varies considerably in its position in different individuals. As a general rule it points obliquely upwards towards the spleen, but it may lie entirely behind the caecum, or point downwards and overhang the brim of the pelvis. The results of appendicitis are materially affected by the position of the viscus.

### 1. FOREIGN BODIES AND CONCRETIONS.

From its situation at the head of the caecum the appendix is peculiarly liable to become the resting place of foreign 2032
bodies, such as pins, nails, shot, pieces of bone, and the seeds 2033
of various fruits. Normal appendices are often full of soft 2034
faecal material, but true faecal concretions are common asso- 2031*B*
ciates of appendicitis. These are hard, rounded or oval, bodies
of the size of a small hazel-nut. They consist of faecal 2032*F*
material and inspissated mucus infiltrated by lime-salts, and 2032*H*
on section present a concentric lamination. The importance of foreign bodies and concretions in this viscus lies in the frequency with which they appear to predispose it to inflammation.

### 2. INFLAMMATION.

Micro-organisms are the exciting cause of appendicitis, particularly the bacillus coli communis, the streptococcus pyogenes, and the staphylococcus pyogenes aureus. The effects of inflammation vary from a slight swelling and hyperaemia of the mucous membrane to the most acute gangrene. It is convenient to divide the lesions into three groups: (1) catarrhal; (2) ulcerative; (3) gangrenous.

(1) **Catarrhal appendicitis.**—In the acute catarrhal form the
appendix is thickened, its mucous membrane swollen, velvety, 2034*A*
hyperaemic, and sometimes dotted with petechial haemorrhages. The peritoneum covering it is inflamed and becomes adherent to its neighbourhood. In these mild cases the inflammation

may become chronic, or be recurrent at intervals of a few months, leading in time to stenosis or obliteration of the lumen. When obliteration is complete the appendix is con-
2036*C* verted into a fibrous cord. More often obliteration occurs at
2036*D* one point only, in which case the distal extremity becomes
2036*E* distended by accumulated secretion into a pear-shaped cyst
2036*F* filled with a mucous fluid (mucocele).

(2) **Ulcerative appendicitis.**—Ulceration of the appendix may be general or local. In the latter case it commonly appears at the site of impaction of a foreign body or con-
2034*C* cretion. Ulceration may be superficial only, or may involve the whole thickness of the wall and produce a perforation.
2035*A* Typhoid and tuberculous ulcers are sometimes found in the
2035*B* appendix.

(3) **Gangrenous appendicitis.**—Gangrene of the appendix may be a final stage of the preceding varieties, but sometimes is established so acutely as to merit the term "primary"
2034*B* gangrene. The gangrenous viscus is swollen, deeply-injected,
2034*C* blackened or greenish, and tears easily.

**Complications of appendicitis.** (*A*) **Abscess.**—Severe inflammations of the appendix, whether attended by perforation or not, commonly result in localised abscesses in the neigh-
2033 bourhood, the infected area being shut off from the general peritoneal cavity by adhesions established before the formation of pus. These abscesses may be intra- or extra-peritoneal, and in either case may point in remote situations. Thus an intra-peritoneal abscess may point in the left iliac fossa, or rupture into the rectum, while an extra-peritoneal one may form a perinephric tumour, or point below Poupart's ligament and simulate a psoas abscess.

(*B*) **Perforation.**—In the case of acute inflammation perforation may anticipate the appearance of the peritoneal adhesions which commonly localise the infective process. When this occurs the result is a general peritonitis.

(*C*) **Suppurative pylephlebitis.**—Appendicitis is the commonest cause of suppurative thrombosis of the portal vein.

(*D*) **Bands.**—The adhesions left as a result of past appendicitis may become stretched in course of time and form fibrous
2164*A* bands capable of causing intestinal obstruction.

## 3. TUMOURS OF THE APPENDIX.

These are uncommon. A peculiar form of growth is occasionally met with. It is slow in its progress and shows no tendency to become disseminated, but nevertheless infiltrates the wall of the appendix and forms a bulbous swelling towards its extremity. 2029*D* 2029*E*

Histologically these growths bear some resemblance to carcinoma, but are generally held to be endotheliomata.

# CHAPTER XVII.

## DISEASES OF THE LIVER.

I. Anatomical Variations. II. Perihepatitis. III. Circulatory Disturbances.—(*a*) Venous Congestion, (*b*) Thrombosis of the Portal Vein, (*c*) Infarction. IV. Inflammations.—(*a*) Hepatic Abscess, (*b*) Chronic Hepatitis (Cirrhosis of the Liver), (*c*) Syphilis, (*d*) Tuberculosis, (*e*) Actinomycosis. V. Degenerations.—(*a*) Acute, (*b*) Chronic. VI. Hydatid Disease. VII. Lymphadenoma. VIII. Leukaemic Infiltration. IX. Pernicious Anaemia. X. Tumours.—(*a*) Innocent, (*b*) Malignant.

### I. ANATOMICAL VARIATIONS.

With one exception the simple anatomical variations which affect the liver, as distinct from the gall-bladder and bile-ducts, are not important. This exception is a tongue-like projection from the anterior edge of the right lobe, known as "Riedel's lobe." This projection is sometimes a developmental anomaly,
2241*C* but may result from tight-lacing, and occasionally from the traction of adhesions upon an inflamed gall-bladder.

**"Tight-lacer's" liver.**—The tight-lacer's liver is marked by a transverse furrow across the anterior superior surface. The peritoneum covering this furrow is thickened and opaque, and the liver beneath it thinned by pressure. This thinning may be so extreme as to produce the appearance of an adventitious
2204*D* lobe attached to the anterior border of the right side of the
2241*C* liver by little more than a double layer of peritoneum.

## II. PERIHEPATITIS.

Inflammations of the peritoneum covering the liver may be (1) localised, (2) diffuse.

(1) Any inflammation of the liver which reaches the surface will produce a **local perihepatitis**, as will inflammatory conditions of neighbouring viscera. Thus hepatic abscesses, gastric and duodenal ulcers, pleural or pulmonary inflammations which traverse the diaphragm, and inflammations of the gall-bladder, with many other lesions of a like nature, will induce a localised perihepatitis. In an early stage the affected area loses its lustre, becomes coated with inflammatory lymph and tends to adhere to any serous surface with which it is in contact. This adhesive layer subsequently becomes organised by the entry of blood-vessels, and converted into adhesions whose density is proportional to the persistence of the inflammation. A localised chronic perihepatitis occurs as a result of 2202*C*
tight lacing.

(2) **Diffuse perihepatitis** is part of a generalised lesion of the peritoneum, and, with the exception of one group of cases, does not require to be distinguished from peritonitis. The exception is the condition known as "diffuse chronic perihepatitis." This, like other forms of diffuse perihepatitis, is merely the local expression of a general peritoneal lesion, but deserves special mention because the lesion of the peritoneum covering the liver is disproportionately prominent. It is associated with a generalised chronic peritonitis found among the subjects of arteriosclerosis and granular kidney. The lesion consists of a white membranous deposit, sometimes as much as $\frac{1}{8}$ inch in thickness, upon the surface of the liver. This layer is often pitted by circular indentations, and may be stripped from the underlying peritoneum leaving the latter more or less intact. The liver is generally reduced in size, but 2193*A*
not cirrhotic. The spleen and intestines may be similarly 2193*B*
affected.

Microscopically the deposit consists of laminated fibrous tissue.

## III. CIRCULAR DISTURBANCES.

(*a*) **Venous congestion.**—Passive congestion of the liver follows any considerable interference with the flow of blood

through the heart. Its commonest cause, therefore, is chronic valvular disease of the heart in the stage of failing compensation, especially disease of the mitral orifice. But the cardiac embarrassment consequent upon myocardial degeneration, emphysema, or any extensive lung-destruction is competent to produce the lesion. The impediment thus offered to the discharge of blood from the inferior vena cava speedily distends the efferent vessels of the liver—the hepatic, sub-lobular, and intra-lobular veins. Prolonged congestion of this kind produces a very typical alteration in the liver. It is enlarged, firm but unduly friable, and deep-red in colour. On section it exhibits a fine mottling, to which is due its common name—"the **nutmeg liver.**"

It will be remembered that the portal vein is distributed, in the form of inter-lobular veins, about the periphery of the lobules. Here the arterial function of the portal vein ceases. Its blood is distributed from this point by a network of capillaries which traverse the lobule to unite at its centre in the intra-lobular vein. Hence it passes in turn through the sub-lobular and hepatic veins to reach the inferior vena cava. Thus the arterial district of the portal service is comprised by the inter-lobular veins and the periphery of the lobules, while the venous district includes the central portions of the lobules, with the hepatic veins and their branches. Backward pressure, therefore, produces in the first instance congestion of the venous district, together with some extravasation of blood in the central zone of each lobule, leaving the arterial district at the periphery relatively anaemic. When such congestion is prolonged the liver-cells of the innermost zone undergo fatty degeneration. The stasis in the circulation results also in an extensive destruction of red blood corpuscles and an accumulation of dark granules of haematoidin derived from them. If these considerations be borne in mind the explanation of the nutmeg appearance is easily understood. Close inspection of the cut surface of a nutmeg liver shows a multitude of minute intra-lobular veins, transversely divided. Each of these is surrounded by a narrow zone, generally of a yellowish colour, but occasionally green from bile-staining. Outside this central zone is one of a chocolate colour. It represents the intermediate zone of the lobule and owes its colour primarily to the abundant deposit of haematoidin

granules mentioned above, but in part to a dense accumulation of red blood-cells. Outside the chocolate-coloured zone is a second yellowish area merging with similar adjacent districts. It is usually taught that the intra-lobular vein is immediately surrounded by a dark zone of congestion, but whenever the nutmeg character is well defined the above description will be found correct. 2203*B* 2203*C*

**Microscopic examination** shows that the central pale zone is composed of fatty and often necrotic liver cells. The pigment deposits of the intermediate chocolate-coloured area are best seen in an unstained fresh section of the gland. In a section prepared in the ordinary way this zone seems to be composed for the most part of accumulated red-cells distending the capillaries and extravasated outside them. The liver cells of the outer yellowish zone present an approximately normal appearance.

(*b*) **Portal thrombosis.**—(*A*) simple; (*B*) infective (suppurative pylephlebitis).

(*A*) **Simple thrombosis of the portal vein** occurs under a variety of circumstances, but most often in the course either of hepatic cirrhosis or of abdominal malignant disease.

The vein itself is generally thickened by chronic phlebosclerosis and may be calcareous. It is distended by a firm clot whose character varies with the duration of the thrombosis. In recent cases the clot is red and not firmly adherent to the vein wall, while in those of long standing it is decolourised and inseparably attached to the vessel. The liver undergoes no constant changes as a result of simple portal thrombosis, but some degree of fatty change is the rule. 2205 2205$A_1$ 2205$A_2$ 2218 2219

(*B*) **Infective thrombosis or suppurative pylephlebitis.**—This may be produced by any suppuration or ulceration in connection with the abdominal viscera, but its commonest cause is appendicitis.

The portal vein is acutely inflamed, thickened, and filled by purulent blood-clot in all stages of disintegration. The liver is almost invariably affected. It is enlarged and is the seat of multiple metastatic abscesses, the result of lodgment of infective particles derived from the portal vein. These abscesses are often stained a green colour by bile. They may perforate the capsule of the liver and cause a sub diaphragmatic abscess. 2206 2206*C* 2206*G* 2206*B*

(*c*) **Infarction.**—Infarction is not a prominent lesion in the liver, but haemorrhagic infarcts, due to obliteration of branches

2205*B* of the portal vein, are occasionally observed. The secondary abscesses in the liver which attend suppurative pylephlebitis are sometimes due, not so much to a direct spread of the infection along the portal vein, as to embolism by particles of purulent thrombus. In such instances the abscesses are of the nature of purulent infarcts.

## IV. INFLAMMATIONS.

(*a*) **Hepatic abscess.**—Hepatic abscesses fall into two broad divisions. In one the abscess is large and single; in the other, many small abscesses occur throughout the liver.

**Single abscess.**—This may depend upon suppuration in a hydatid cyst. Otherwise its commonest cause is tropical
2195 dysentery, particularly the amoebic variety. The abscess is
2196 generally situated in the right lobe and varies greatly in size. It is surrounded by a fibrous capsule whose density increases with the age of the abscess. The contents of a pure amoebic abscess are described as being translucent, glairy and viscid, and composed of amoebae, necrotic liver cells and red blood-corpuscles, with very few leucocytes. But pyogenic organisms often invade such amoebic abscesses, in which case the contents assume the ordinary characters of pus.

If untreated, hepatic abscesses of this order may rupture in any direction. Most often they take an upward path and penetrating the diaphragm and eroding the lung discharge themselves by way of the bronchi of the right lower lobe.

**Multiple abscess.**—The infection responsible for this lesion may reach the liver by one of three paths.

2206*E* (1) By the hepatic artery in the course of pyaemia.

(2) By the portal vein, from foci of suppuration anywhere within the portal district. The condition then is one of suppurative pylephlebitis.

(3) By the bile-ducts, most often as an ascending cholangitis from a suppurating gall-bladder.

(*b*) **Cirrhosis of the liver** (chronic hepatitis).—By this term is meant a diffuse fibrous hyperplasia of the liver. The essential cause of the lesion is a poisonous condition of the blood circulating within the gland, but the poisons capable of producing cirrhosis are very various.

Cirrhotic livers may be divided into three fairly distinct

groups, though a rigid line cannot always be drawn between them. This grouping is dictated by anatomical considerations, and is as follows:

(1) Multilobular or portal cirrhosis.
(2) Unilobular or biliary cirrhosis.
(3) Intercellular or syphilitic cirrhosis.

(1) **Multilobular or portal cirrhosis** is by far the most common and important. The poison in this case comes to the liver by way of the portal vein, though the precise nature of it is doubtful. The lesion is generally found among excessive drinkers of alcohol, but many such people escape it, while experimental attempts to produce the disease by feeding animals with alcohol have generally failed. In view of these facts it has been assumed, either that alcohol produces cirrhosis of the liver only in those whose liver-cells are inherently deficient in resistance, whether from previous syphilis or some other infection, or that the lesion is due to poisons resulting from the chronic gastro-intestinal catarrh common in alcoholic subjects. But alcohol is not by any means the only cause, for the lesion occurs in a typical form under circumstances which exclude the possibility of alcoholism. 2198*E*

The liver of multilobular cirrhosis may be either larger or smaller than the normal. The smaller, atrophic, variety (gin-drinker's liver) is much shrunken. Its capsule is thickened, its edges sharp and uneven, and its surface tuberculated or coarsely granular, whence the name "hob-nail liver." The larger or "hypertrophic" variety (the beer-drinker's liver) may reach an enormous size. It is very fatty and often of a bright yellow colour. The edges are rounded and the surface finely granular, so finely as to be almost smooth. On section 2199*A*
the cirrhotic liver is very resistant to the knife. The appearance of the cut surface is variable in different specimens, but *all exhibit grey bands of translucent fibrous tissue dividing the parenchyma into islets of unequal size.* These islets are of a 2198*A*
yellow colour, due to fatty changes. 2198*E*

**Microscopic examination** shows that the invading bands of fibrous tissue for the most part enclose many lobules together; the cirrhosis, that is to say, is multilobular. The intersections consist of fibrous tissue, highly-cellular or fully-formed according to the activity or chronicity of the process. They often contain in abundance double columns of deeply-stained cells having the appearance of tubules.

These structures are commonly described as newly-formed bile-ducts, to which they bear a remarkable resemblance, but their nature is doubtful. It is likely that they are columns of liver-cells actively dividing in an attempt to compensate for the destruction in process elsewhere (Rolleston). The liver-cells generally are compressed and fatty.

The gradual shrinkage of the newly-formed fibrous tissue results in the indentations and granularity of surface characteristic of the lesion, and, by obliterating the interlobular branches of the portal vein, gives rise to portal obstruction. The blood vessels of the new fibrous tissue belong to the hepatic, and not the portal, system.

(2) **Unilobular cirrhosis** (biliary cirrhosis).—This variety permits of two sub-divisions: (*a*) hypertrophic biliary, or Hanot's cirrhosis; (*b*) obstructive biliary cirrhosis. Both of these are rare.

(*a*) **Hypertrophic biliary cirrhosis** is characterised by chronic jaundice with fever and enlargement of the liver and spleen. The liver, in the absence of secondary portal cirrhosis is smooth on the surface. It is firm and of a dark-green colour. Its cut surface exhibits a finely-marbled appearance due to closely-set intersections of fibrous tissue.

**Microscopic examination** of an early example of the lesion shows that the fibrous intersections isolate individual lobules, but at a later stage secondary portal cirrhosis is liable to supervene and obscure the unilobular character of the lesion.

(*b*) **Obstructive biliary cirrhosis.**—Whereas acquired obstruction of the bile-ducts very rarely leads to cirrhosis, congenital obliteration of these ducts is almost invariably associated with a unilobular or mixed cirrhosis. But it is a debatable question whether the cirrhosis in this case is due to the biliary obstruction, or whether both lesions depend upon an identical poison borne by the maternal blood.

(3) **Intercellular cirrhosis.**—This is the commonest hepatic lesion of congenital syphilis, the poison reaching the liver by the umbilical vein. The typical lesions are met with, as a rule,
2198*D* only in very early life, for during later childhood the damaged
2202*E* liver frequently becomes the seat of a secondary portal cirrhosis which renders it hob-nailed.

The liver in such an early instance is lighter in colour and firmer on section than the normal gland. Its cut surface is

mottled with yellowish areas, and often picked out with minute grey dots resembling miliary tubercles. 2202*F*

**Microscopic examination** shows a diffuse intercellular infiltration with small round cells, and occasional aggregations of these cells representing the grey dots mentioned above. At a later stage, this cellular infiltration is converted into fully-formed fibrous tissue. The liver cells are generally compressed and degenerate.

(*c*) **Hepatic lesions of tertiary syphilis.**—Gummata, the localised inflammatory infiltrations met with in cases of tertiary syphilis (and in some of the later manifestations of the congenital disease,) are not uncommonly met with in the liver. 2202*A* They may progress to resolution, in which case the result is a trifling scar; or they may caseate at the centre. The caseous matter in turn may undergo a slow absorption, and result in an extensive scar, often enclosing the calcified residue of the necrotic accumulation. Commonly enough cicatrices and active gummata co-exist in the same specimen.

Syphilitic cicatrices appear as puckers or deep indentations furrowing the surface in various directions. Gummata of the 2202*C* liver are generally multiple. They are yellowish masses surrounded by a zone of fibrosis, and prone to disintegrate at the centre with the formation of a cheesy material. 2202*D*

Microscopically, a typical gumma consists of three zones. The outermost is fibrous; the innermost, hyaline, ill-stained, and often definitely necrotic. Between these lies an ill-defined transitional zone partaking of the characters of both. It is formed by granulation tissue showing some degree of hyaline degeneration, and occasionally containing giant-cells. These giant-cells are not so large as those found in tubercles, nor are the nuclei so regularly arranged around their periphery. The arteries in the neighbourhood of a gumma invariably show evidence of a proliferating endarteritis, while extensions from the fibrous capsule often produce a localised intercellular cirrhosis.

(*d*) **Tuberculosis** of the liver is almost always a multiple lesion. The three common varieties are: (1) miliary tuberculosis, (2) tuberculous cholangitis, (3) gross tuberculosis of the capsule.

(1) **Miliary tuberculosis** of the liver is the result of a tuberculous septicaemia, the bacilli reaching the gland by way of the hepatic artery. The liver is plentifully studded by minute grey tubercles which tend to enlarge in course of time, and become yellowish from commencing caseation. In the 2196$A_3$ course of acute general tuberculosis the liver is invariably

affected, but the tubercles are sometimes not visible except
2196*A* under the microscope. They are generally more apparent on the surface than within the substance of the gland. The histological features of a tubercle are given on page 19.

(2) **Tuberculous cholangitis.**—If the infection is carried by the portal vein, as is the case when the intestine is the seat of tuberculous ulcers, the bacilli are deposited in the portal spaces, and give rise to tubercles in this situation. Subsequent enlargement and caseation of these tubercles frequently ends in their rupture into neighbouring bile-ducts. The result is the so-called "tuberculous cholangitis." It is not a cholangitis strictly speaking, for the infection of the bile-ducts results merely from the accident of their propinquity to the portal tubercles.

The liver is fatty, and on section marked by numerous
2196$A_4$ small cavities with a lining of bile-stained caseous material.
2196$A_5$ Caseation of the lymphatic glands at the hilum of the liver is common.

(3) **Gross tuberculosis of the capsule.**—Large caseous tubercles are occasionally found, situated in the capsule, and
2196$A_2$ infiltrating the superficial parts of the liver beneath them.

(*e*) **Actinomycosis.**—The ray-fungus "streptothrix actinomyces," a parasite of many kinds of corn, may affect the liver either by direct extension from a neighbouring lesion, or by the blood-stream. The primary focus is generally in the intestine.

The liver is enlarged, and often adherent to its surroundings. On section, the affected district presents a characteristic appear-
2239*A* ance It is a localised, but often large, area of softened tissue,
2239*E* surrounded by a zone of fibrosis, and of a honey-combed
2239*F* aspect. The spaces enclosed by the intersections forming the honey-comb are filled with pus which contains the yellow granules pathognomonic of the disease.

**Microscopic examination** shows that these granules are colonies of the fungus. They are composed of a mass of interwoven mycelial threads, some of which terminate in bulbous extremities, the so-called "clubs." The formation of clubs takes place with greater frequency in the bovine than in the human type of the malady.

## V. DEGENERATIONS.

(*a*) **Acute degenerations.**—A number of poisons, both chemical and bacterial, are capable of producing acute degenerations

of the liver cells. Some of these poisons have been identified, but more remain unknown, the resulting diseases being roughly grouped under the clinical title "icterus gravis," on 2202*H* the score of the jaundice which is their prominent characteristic. There are, however, three more or less well-defined varieties of acute degeneration.

1. **Focal necrosis.**—After death from certain acute infections, notably typhoid fever, the liver may be dotted with minute areas of necrosis. These areas are as a rule microscopic only, and are identified by the fact that the cells composing them are ill-stained and often fatty.

2. **Acute yellow atrophy.**—The poisons producing this disease are not known. The liver is greatly reduced in size, and unnaturally flaccid, while its capsule is often wrinkled owing to the shrinkage of the gland. The cut surface is of a 2202*G* bright yellow colour, mottled by reddish patches.

**Microscopic examination** shows that the red areas represent an advanced stage of degeneration, the necrotic cells having here been replaced by fibrous tissue and blood-containing capillaries. Elsewhere there is extensive necrosis of liver-cells, with occasional signs of inflammatory reaction. A scraping taken from the cut surface of such a liver shows, under the microscope, necrotic liver cells with crystals of leucin and tyrosin.

3. **Acute phosphorus poisoning.**—The liver in this condition is generally enlarged, smooth, yellow, and obviously the seat of extreme fatty changes. 2238*A*

(*b*) **Chronic degenerations.** (1) **Fatty liver.**—The cells of the liver may contain fat in conditions of health, especially in the young and the obese, but many diseases increase this fat-content to a pathological degree. Attempts have been made to differentiate fatty infiltration—a mere exaggeration of the physiological storage alluded to above, from fatty degeneration—the actual conversion of cell-protoplasm into fat. But the distinction is too fine to be drawn with conviction, and it is better to consider under the generic title "fatty liver" all varieties of gross excess in the fat-content.

The essential cause of fatty liver is a poison in the circulating blood. The poison concerned may be chemical, *e.g.* alcohol and phosphorus, or microbic, or metabolic. Most microbic infections lead to fatty changes in the liver, especially those of tuberculosis, typhoid fever, diphtheria, cellulitis and suppurative lesions generally. The fatty changes seen in the nutmeg

liver probably depend upon an accumulation of metabolic poisons, the result of stasis in the circulation.

2193 The fatty liver is increased in bulk, and often very large.
2238*A* It is smooth, soft, friable and yellow. On section it is obviously greasy and will sometimes float in water.

**Microscopic examination** shows that the liver cells, especially those towards the periphery of the lobules, are distended by refractive globules of fat which are stained black by osmic acid and orange with Sudan III.

(2) **Amyloid degeneration** (waxy or lardaceous degenera-
2194 tion).—A general description of this degeneration is given at
2194*A* p. 15. The amyloid liver is enlarged, often to an extreme
2194*C* degree ; its edges are rounded and its consistency is unnaturally
2194*D* firm. On section it has an anaemic, translucent appearance produced by diffuse deposits of amyloid material.

**Microscopic examination** shows that the first deposits appear in the middle coats of the small arterioles of the hepatic artery. But the bulk of the deposit in an established case is found in the capillary walls of the intermediate zone. In a section treated with a watery solution of methyl-aniline violet the infiltrated walls of the capillaries appear as radiating streaks of a red or pink colour while the healthy tissues are stained violet. As the lesion advances the whole liver becomes involved, the surviving liver-cells showing sign of compression and often of fatty change.

## VI. HYDATID CYSTS.

Hydatid cysts represent the cystic stage of taenia echinococcus, a small tapeworm which in its adult phase inhabits the intestine of the canidae. The life history of the parasite is given on p. 28.

The liver is by far the commonest site of hydatid cysts.
2235*A* These are usually situated in the right lobe and are for the most
2235*B* part solitary, though two or more may co-exist in the same specimen. The parasitic cyst itself, while alive, consists of two well-defined layers, an outer chitinous ectocyst, an inner soft, granular and gelatinous endocyst. By a process of budding there are formed upon the inner layer minute white elevations,
2235*B* "brood-capsules" upon which grow scolices of the worm. Cystic dilatations of the blood-capsules, and invaginations of the inner layer produce "daughter-cysts," either free or attached, and
2232*A* often bile-stained. These vary in abundance and size, and are capable of originating a third generation, "grand-daughter

cysts," within them. The fluid generally contains both scolices, and detached hooklets derived from scolices which have become disintegrated. The whole parasitic cyst is enclosed by a false capsule supplied by the condensed liver-tissue in its immediate neighbourhood. 2233*A*

## Fate of the Cysts.

(*a*) **Rupture during the life of the parasite.**—A cyst which becomes ruptured during the life of the parasite discharges its contents, including living scolices, through the resulting aperture. The consequences vary with the nature of the cavity into which the discharge takes place. If a serous sac is 2235*B* invaded, multiple hydatid cysts may develop in it. If the alimentary canal, the contents of the sac may be voided per 2237*A* anum and the disease undergoes spontaneous cure. Rupture 2237*B* into the common bile duct may occur and produce biliary 2235*D* obstruction.

(*b*) **Death of the parasite.**—A hydatid cyst of the liver may die *in situ* (1) spontaneously, (2) as a consequence of invasion by pyogenic organisms.

(1) Spontaneous death results in gradual absorption of the contained fluid. The cyst-wall becomes folded upon itself as it collapses, compressing the daughter-cysts lying within it. Ultimately the cyst is converted into a solid mass, the interior 2230*B* of which is composed of fold on fold of cyst-wall, closely com- 2230*C* pressed and more or less infiltrated by lime-salts. 2233*B*

(2) Hydatid cysts may suppurate in consequence of the invasion of pyogenic organisms. In this event the parasite dies and the cyst becomes an abscess containing remnants of hydatid membrane. The abscess thus formed may point and discharge its contents in a variety of directions, the following being the commonest.

(*a*) Externally, through the abdominal wall. 2235

(*b*) Into the peritoneal cavity, giving rise to general purulent peritonitis. 2235*B*

(*c*) Into the pleural sac, or, after penetration of the lung, into a bronchus.

## VII. LYMPHADENOMA.

2223*A* 2223$A_1$ Lymphadenomatous deposits appear in the liver as numerous small white areas, varying in size from minute specks to the dimensions of a hazel nut, and not unlike caseous tubercles; but occasionally large masses may be found. The growth commences in the portal spaces and thence invades the lobules.

Histologically the growth consists of a connective tissue reticulum supporting leucocytes and large multinucleated endothelial cells, the so-called "lymphadenoma cells."

## VIII. LEUKAEMIC INFILTRATION.

Leukaemic infiltrations of the liver occur both in the myelogenous and lymphatic varieties of the disease. The liver is enlarged. It is soft, pale, and smooth, and, on section, may exhibit minute white dots indicative of leucocytic accumulations.

Microscopically the portal spaces and capillaries are the seat of dense aggregations of leucocytes.

## IX. PERNICIOUS ANAEMIA.

3220*A* In the course of pernicious anaemia the liver becomes fatty and has deposited in it an iron-containing product of the destruction of red blood cells. This substance is haemosiderin. It is identified by the blue colour resulting from the treatment of the cut surface with hydrochloric acid and a solution of potassium ferrocyanide. The reaction is not confined to pernicious anaemia, but is seldom absent in this disease.

## X. TUMOURS.

(*a*) **Innocent.** (1) **Simple cysts.**—The liver may be the seat of simple cysts, either single or multiple, the condition in the latter case being often associated with a similar state of the 2204*D* kidneys. During infancy the cysts are minute, but in adult life they may attain a large size. Each cyst is surrounded by a fibrous capsule, is thin-walled, and contains a clear fluid.

**Microscopic examination** in the infantile cases shows in the portal spaces dilated tubes, lined by columnar epithelium and having the appearance of expanded bile-ducts, but containing no bile. In the adult the cells lining the cysts vary from a columnar to a flattened type according as the cysts are small or large.

The solitary simple cyst is usually regarded as a retention 2204*B*
cyst the result of obstruction to a bile-duct, the retained bile 2204*C*
being gradually replaced by a clear fluid as is the case with 2204*D*
the gall-bladder when the cystic duct is obstructed. The origin of multiple cysts is not known. Some authorities maintain that they represent dilatations of bile-ducts produced by the fibrosis following an antecedent cholangitis; a process comparable with that which produces cysts in granular kidneys. Others hold them to be developmental anomalies due to the irregular development of parts of the original duodenal diverticulum which forms the liver.

(2) **Cavernous naevus.**—Cavernous naevi are occasionally 2224
seen in the liver. They appear as well-defined dark-red areas 2224*A*
exhibiting on section a finely honey-combed aspect. 2224*B*
2225

Microscopically they consist of a fibrous reticulum marking out communicating blood-spaces lined by endothelial cells. 2225*A*

(3) **Adenoma.**—Solitary adenomata of the liver are rare. They form yellowish encapsuled tumours projecting from the surface of the liver, and are liable to degeneration. 2223*B*

Microscopically these adenomata consist of convoluted columns of cells, varying in size but bearing a general resemblance to liver-cells though not grouped into typical lobules.

Multiple adenomata generally occur in cirrhotic livers, and probably represent a compensatory hyperplasia. A liver occupied by these tumours has the appearance of a hob-nail liver in which the size of the hob-nails is greatly exaggerated. On section the liver suggests the presence of multiple secondary growths or tubercles, for the adenomatous masses are of a whitish colour and may be softened.

**Microscopic examination** shows that the hyperplastic nodules consist of liver-cells actively proliferating. Degeneration may occur, rendering the cells of the adenomata ill-stained and fatty.

(*b*) **Malignant tumours.**—(1) Carcinoma; (2) sarcoma.

(1) **Carcinoma.**—Carcinoma of the liver may be either primary or secondary, but the latter is by far the more frequent.

Primary carcinoma of the liver appears in two principal
2216 forms, and is often associated with cirrhosis. In one the growth is solitary and forms a large white mass, distending the viscus with or without the production of marked irregularities of the surface. These large growths are very prone to disinte-
2218 gration at the centre. In the other and more common form
2218*A* the growths are multiple, yet appear to be primary, no focus of disease elsewhere being capable of detection. A third variety is supplied by a diffuse growth tending to spread along the portal canals.

**Microscopic examination** shows a great variety in the cell-types of these growths. Sometimes they accurately reproduce the appearance of liver cells; at others the cells are columnar, as though having originated in connection with intra-hepatic bile-ducts; at others again the cells are spheroidal.

**Secondary carcinoma** of the liver is common. The primary focus is generally in the alimentary canal or pancreas, the metastases depending upon the lodgment in the liver of
2216*F* particles of growth carried along the portal vein. Livers thus
2216*H* affected are always enlarged and may reach an enormous size;
2216*I* the surface is tuberculated by white projections of growth, often umbilicated as a result of central necrosis. On section the numerous deposits give the cut surface a marbled appearance, the secondary growths being either white, or greenish, or blood-stained. The remainder of the liver is commonly jaundiced.

Histologically the secondary deposits resemble the parent tumour.

(2) **Sarcoma.**—As with carcinoma so with sarcoma the growth may be primary but is more often secondary.

**Primary sarcoma** cannot be distinguished by the naked eye from primary carcinoma, and like it, may appear as a solitary
2215*B* tumour or as a collection of growths. All varieties of
2215*D* sarcoma may be met with as primary growths in the liver
2215*D* except the melanotic form. Cases of the latter kind have
2215$D_2$ been reported, but their authenticity is doubtful.

**Secondary sarcoma.**—All the histological types of sarcoma,
2209*B* including the melanotic form, may be encountered in the liver.
2209*C* The growths are multiple, and, with the exception of the last
2209*D* variety, cannot be distinguished from secondary carcinoma
2215*C* without the aid of the microscope.

# CHAPTER XVIII.

## DISEASES OF THE GALL-BLADDER AND BILE-DUCTS.

I. Malformations. II. Cholelithiasis. III. Cholecystitis. IV. Cholangitis. V. Tumours. VI. Mechanical Causes of Chronic Jaundice.

### I. MALFORMATIONS.

**Congenital obliteration of the bile-ducts.**—The common and cystic ducts are occasionally found obliterated at birth, and may be absent altogether. The origin of the condition is a matter of dispute. In some instances it is probable that it is the result of a true failure of development; in others, and these the majority, it is the result of foetal disease, and is invariably associated with a coarse hepatic cirrhosis of the multilobular type.

The liver is large, its edges rounded, its surface nodular, and its colour a deep olive-green due to accumulation of bile-pigments. The common and cystic ducts are sometimes visible as fibrous cords; sometimes the most careful dissection cannot detect them. In a few cases they are pervious for a portion of their course, but obliterated before their junction, or between their junction and the duodenum. The liver is resistant to the knife owing to the grey fibrous bands which traverse it in all directions, and give to the cut surface a mottled green and grey appearance.

**Microscopic examination** shows that the new fibrous tissue is distributed erratically throughout the liver, and is accompanied by a considerable exudation of small round cells. The structures described as new bile-ducts are numerous.

Congenital absence of the gall-bladder, with or without absence of the common duct, is an occasional anomaly. 3623*A* 3623*A*$_1$

## II. CHOLELITHIASIS (GALL-STONES).

(The numbers in the first paragraph of this section refer to the series of calculi on the ground floor.)

Gall-stones are concretions formed in the biliary passages or in the gall-bladder; exceptionally, and in a special form, they are found in the intra-hepatic ducts. The colour, consistency, and shape of the stones vary with their situation, their number, and their composition. In the intra-hepatic ducts the stones are minute black particles of irregular shape and smooth surface, consisting of pure *bilirubin-calcium*. In the gall-bladder
274*D* their size varies from that of a pea or less, to that of a hen's
264 egg. When small they are multiple, often amounting to
274*B* several hundreds: when large they are usually single. When multiple their surfaces are usually facetted by mutual
267 pressure, and their shape consequently variable; when single they are oval or round The colour also varies considerably from a pearly-white to a dull brown or black, the variation being due to the varying proportion of their chemical constituents. *With the exception of the intra-hepatic calculi already mentioned, the chief constituent of all gall-stones is cholesterin.* In a few cases pure cholesterin stones are found, usually single or few in number, round or oval, of a greyish-
267*A* brown colour, and slightly translucent. More commonly the
276 stones are of various shades of yellow, brown, black, or deep olive-green; and not infrequently of different colours in their different layers. On section a stone of moderate size is found to consist of a hard, and often laminated rind, enclosing a much softer core, at the centre of which there is a dark nucleus or sometimes a small cavity; the rind is commonly composed of calcium and magnesium salts; the bulk of the stone of cholesterin, and the nucleus at the centre of bile-
266 pigment. The colour of the rind and of the core varies with
266*A* the proportion of bile-pigment, and of calcium and magnesium salts. Cholesterin stones are very light, and float easily in water. Stones consisting of pure calcium carbonate are rare. Gall-stones are found at all ages, even in infancy, but are most common after middle life, and in the female sex. Three-fourths of all cases occur in women who have borne children.

Chronic catarrh of the bile-passages is the essential factor in the production of gall-stones; and since it is sometimes possible to demonstrate bacteria within the stones, it is believed that the catarrh is due to bacterial infection. The catarrh is accompanied by a desquamation of epithelium, and an albuminous exudation; the next stage is the precipitation, especially in stagnant bile, of a combination of bilirubin and calcium, which forms a nucleus upon which is deposited cholesterin derived from the degenerate epithelium. With the cholesterin is associated an albuminous material, which forms a framework for the crystalline substance, and can be demonstrated by dissolving out the cholesterin. In the majority of stones bile-pigments, bile-salts, and compounds of calcium, magnesium, and sodium are deposited together with the cholesterin.

**Effects of gall-stones.**—In a large number of cases stones in the gall-bladder give rise to no symptoms, and are only discovered at an operation or after death. But the presence of gall-stones predisposes the gall-bladder to inflammation **(cholecystitis)**, *q.v.* 2259*A*

Owing to the muscular contractions of the wall of the gall-bladder a stone may be forced into the orifice of the cystic duct; if small enough it will pass along this, and thence by the common duct to the intestine, and be discharged with the faeces. In its passage there are two points at which it is especially likely to become impacted owing to the narrowness of the channel; these are (1) the commencement of the cystic 2248*C* duct and (2) the ampulla of Vater at the entrance of the 2248*D* common duct into the duodenum. If the stone be so large as to be unable to pass easily, the muscular walls of the duct contract upon it, and its passage is accompanied by acute pain, known as *biliary colic*. The passage of a stone along the common duct is often attended by transient jaundice, due to temporary blocking of the bile flow.

(1) **Impaction in the cystic duct.**—If impaction take place in the cystic duct, the common sequel is dilatation of the gall-bladder, sometimes to an enormous degree. Owing to the 2248*A* blocking of the duct no bile reaches the gall-bladder, while 2248*D* that present at the time of the impaction is absorbed, and replaced by clear mucus, the product of catarrhal inflammation of the epithelium (chronic catarrhal cholecystitis).

In some cases this collection of fluid becomes infected by pyogenic organisms, and suppuration takes place (suppurative cholecystitis). Occasionally, instead of undergoing hypertrophy and dilatation, the gall-bladder shrinks, becomes atrophied, and finally calcified.

(2) **Impaction in the common bile-duct.**—If impaction takes place in the common duct and is complete, deep and persistent jaundice is the result, with dilatation of the larger, and occasionally even of the intrahepatic, ducts. The common duct under these circumstances, may be as large as the normal duodenum, but the gall-bladder as a rule is not dilated. If
2243 the obstruction be incomplete there is intermittent jaundice
2246 with some dilatation of the ducts. Infection of the retained secretion by pyogenic organisms may occur, causing suppurative cholangitis, and multiple abscesses in the liver. Cholangitis due to obstruction of the ducts by a stone may be accompanied by pylephlebitis, either simple or suppurative, but more often the latter.

(3) **Ulceration of gall-bladder.**—Stones too large to enter upon the passage of the ducts may escape from the gall-bladder in consequence of ulcerous perforation of its wall. But a gall-stone is rarely extruded into the general peritoneal cavity, because in most cases the inflamed gall-bladder becomes united to some neighbouring structure by peritoneal adhesions. More commonly there is a localised abscess cavity; or a fistulous communication with one of the hollow viscera. The most common fistulae are those between
2261 the gall-bladder and the duodenum, jejunum, or colon; but
2262 cases are on record of fistulous passages between the gall-
2261*A* bladder on the one hand, and the stomach, lungs, urinary
2262*A* passages, and the exterior of the body on the other. In one case, at least, a gall stone was found to have passed through the wall of the portal vein.

(4) **Intestinal obstruction.**—When a gall-stone has reached the intestine either by passage along the ducts, or by ulcera-
2261*A* tion, it may give rise to intestinal obstruction; in the colon the obstruction usually occurs either at the flexures or at the anal orifice; in the small intestine, at the ileocaecal valve.

(5) **Carcinoma.**—Carcinoma of the gall-bladder may be regarded as one of the remote results of the irritation of gall-

stones: it is at least certain that stones are invariably found in association with it. 2264*E* 2264*F*

## III. CHOLECYSTITIS (INFLAMMATION OF THE GALL-BLADDER).

Inflammation of the gall-bladder is the result of microbic infection; the organisms most often responsible being the bacillus coli communis, the bacillus typhosus, and the pyogenic cocci. A predisposing cause is the presence of gall-stones. 2242*A* The nature of the inflammation may be catarrhal or suppurative, and in either case acute or chronic. In all cases the walls of the bladder are thickened and hypertrophied. Unless the 2242 inflammation is recent, the gall-bladder contains no bile, but either clear mucus or pus.

A rare sequela of cholecystitis is atrophy of the gall-bladder, and calcification of its walls.

Suppurative cholecystitis may involve the bile-ducts and lead to a suppurative cholangitis, but as a rule the obliteration of the cystic duct prevents this complication.

## IV. CHOLANGITIS (INFLAMMATION OF THE BILE-DUCTS).

(1) **Acute catarrhal cholangitis** (catarrhal jaundice) is thought to be due to an extension of a gastroduodenal catarrh along the common duct. The mucous membrane of the common duct is swollen, and its orifice plugged with inspissated detritus. This lesion occurs as an independent affection, but also in the course of some of the acute fevers, *e.g.* pneumonia and typhoid fever. It is probably due to microbic invasion of the lower bile-passages.

(2) **Chronic catarrhal cholangitis** is not known as an independent affection, but accompanies obstruction in the *common duct*. The bile-ducts are dilated, and filled with thickened bile; when the obstruction depends upon an impacted stone the gall-bladder as a rule is not dilated, but when the obstruction is due to other causes, such as carcinoma of the head of the pancreas, the gall-bladder is often much enlarged (Courvoisier's law).

(3) **Suppurative cholangitis** is often associated with suppurative cholecystitis and the presence of gall-stones, but any

suppurative lesion of the liver may infect the ducts. The striking feature of the disease is the presence throughout the liver of dilated ducts filled with pus.

## V. TUMOURS OF THE GALL-BLADDER AND BILE-DUCTS.

**Simple tumours** are very rare; fibromata and myxomata have been described Adenomata liable to cystic degeneration occur both in the intra- and extra-hepatic ducts.

Of **malignant tumours,** carcinoma is not infrequent; it usually attacks the fundus of the gall-bladder, but may appear
2254 at any point in the bladder or bile-ducts. Almost without
2264*A* exception, gall-stones are found in the carcinomatous bladder.
2265 The growth, as a rule, is either fungous or villous; in rare cases it forms a solid mass infiltrating and thickening the bladder-
2264*F* walls. The carcinomatous growth usually extends into the adjoining liver-substance, and may spread along the ducts to the duodenum. Secondary suppurative cholecystitis and cholangitis are not uncommon complications.

Carcinoma occasionally occurs at the ampulla of Vater; it is seldom possible to determine whether the growth has originated in the intestinal or in the biliary mucous membrane.

**Microscopic examination.**—Primary carcinoma of the gall-bladder and bile-ducts is usually columnar-celled or spheroidal-celled. But there is a rare form of carcinoma affecting the bile-ducts, which resembles in structure the adenocarcinomata of the large intestine. It is characterised by the formation of tubes, thickly set, and lined by one or more layers of cubical cells.

## VI. MECHANICAL CAUSES OF CHRONIC JAUNDICE.

2247 (1) Impaction of gall-stones in the common duct.
2235*D* (2) Impaction of hydatid cysts in the common duct.
2252 (3) Tumours of the bile-passages especially carcinoma.
2254 (4) Tumours pressing on the ducts from without.
2254*B* (*a*) Carcinoma of the head of the pancreas.
2275*A* (*b*) Carcinoma of the pylorus.
(*c*) Carcinoma of the duodenum.
(*d*) Enlargement of the glands at the hilum of the liver in tuberculosis, cancer, lymphadenoma.
(*e*) Pressure of hydatid cysts.
(*f*) Pressure of an aneurysm of the coeliac axis artery.

# CHAPTER XIX.

## DISEASES OF THE PANCREAS.

I. Malformations. II. Inflammation. III. Fatty Degeneration. IV. Fat-Necrosis. V. Atrophy. VI. Tuberculosis. VII. Tumours. VIII. Calculi.

### I. MALFORMATIONS.

(1) The pancreas may be divided into two portions united only by the duct.

(2) Accessory organs (pancreatic rests) may be found embedded in the walls of the stomach, duodenum, or jejunum.

(3) The head of the pancreas may form a complete ring round the duodenum and constrict the gut (pancreas annulare).

(4) The duct may open into the stomach or in some other abnormal situation.

### II. INFLAMMATION.

**Acute pancreatitis** is a rare affection, due to an invasion of bacteria. It is associated usually with the presence of pancreatic calculi; but may originate in disease of the neighbouring organs, *e.g.* ulcer of the stomach. In severe cases, the pancreas is riddled with minute abscesses.

**Haemorrhagic pancreatitis** is the name given to an acute affection, in which the whole or part of the organ is infiltrated with extravasated blood. In many cases there is little or no evidence of inflammation. The cause of the disease is not known, but it occurs usually in middle age, and is apt to attack

persons of alcoholic tendencies. The symptoms resemble those of acute intestinal obstruction. Post-mortem, the pancreas and the neighbouring structures are found infiltrated with blood; the omentum and the lesser peritoneal cavity contain a bloody effusion, and there is usually also marked fat-necrosis
2276*E* (*v. infra*).

**Chronic inflammation** of the pancreas is also an uncommon event. It manifests itself in a sclerosis of the organ, with or without an atrophy of the gland-substance, and is one of the lesions associated with diabetes mellitus (*v.* Atrophy, *infra*).

## III. FATTY DEGENERATION.

Fatty degeneration and infiltration of the pancreas both occur, the first in the course of the acute specific fevers, and other acute toxaemias; the second in association with general
2269 obesity.

## IV. FAT NECROSIS.

In connection with various affections of the gland, especially haemorrhagic pancreatitis, small yellowish-white areas of necrosis appear in the interstitial tissue of the pancreas, and
2269*B* in the mesentery, omentum, and sub-peritoneal fat. These
2276*D* are usually the size of a pin's head, but may be much larger,
2276*E* and are occasionally surrounded by a zone of hyperaemia. On section they are soft and tallowy in consistence, or may be infiltrated with lime salts. The necrosis is due to the action of a fat-splitting ferment which converts the fats into fatty acids. Similar lesions have been experimentally produced by the injection of bile into the pancreatic duct. The softening and liquefaction of such areas is said to be one cause of the formation of pancreatic cysts.

## V. ATROPHY.

**Atrophy of the pancreas** occurs as a result of a chronic pancreatitis, and also as a simple senile change. It is not infrequently associated with diabetes, in which case the atrophy more particularly concerns small groups of polygonal cells, interspersed in the interstitial tissue of the gland, and known

as the islands of Langerhans. Their function is not known, but it is surmised that they are connected with the internal secretion which the pancreas is known to produce.

An atrophied pancreas is small, firm, and fibrotic, or fatty. 2269*A*

Microscopically, the connective tissue is greatly increased, and the gland-substance poorly-stained owing to degeneration. It is to be observed in this connection that the cells of the pancreas often become rapidly necrotic after death owing to post-mortem digestion. Caution is therefore necessary in accepting degenerative changes in it as evidence of disease.

## VI. TUBERCULOSIS.

The pancreas is rarely attacked by tuberculosis; when it is involved, the lesion is usually the result of direct extension from diseased lymphatic glands; in a few instances of generalised tuberculosis, miliary nodules are found in its substance; but there is no organ in the body which is so generally free from tuberculous disease. 2272

## VII. TUMOURS OF THE PANCREAS.

**Innocent tumours** are extremely rare, with the exception of **pancreatic cysts.** These are often of mysterious origin. They are said to result from traumatism, from chronic inflammatory processes, and from the degeneration of adenomatous nodules, but there is very little evidence to substantiate these assertions. The only cysts which appear to have a well-ascertained causation, are the small retention cysts found in atrophied fibrotic glands and due to cicatricial occlusion of the ducts, and those due to obstruction of the duct of Wirsung itself by pancreatic calculi or new growths. 2271

The cysts, whatever their origin, often form considerable tumours in the abdomen, and contain a reddish-brown or 2271*A*
blood-stained fluid, with a specific gravity varying from 1010 2271*B*
to 1020. This fluid usually contains one or more of the pancreatic ferments; the presence of trypsin, however, is the only sign on which the least reliance can be placed in diagnosis, since fat-splitting and diastatic ferments are found frequently in exudations elsewhere in the body.

In the neighbourhood of the pancreas, but lacking connection with it, are found cysts of similar size and appearance, which

3372*A* 3372*C* are either (1) cysts of the lesser sac of the peritoneum, or (2) retroperitoneal cysts, whose origin is probably congenital.

**Malignant tumours.**—Carcinoma of the head of the pancreas is not uncommon, though the remainder of the gland is seldom the seat of malignant disease. Carcinoma in this situation is one of the causes of persistent obstructive jaundice, since it 2275*A* commonly exerts pressure upon, or actually involves, the bile 2276*A* ducts.

Microscopically pancreatic carcinoma consists of spheroidal cells imbedded in dense fibrous tissue.

Secondary carcinomatous deposits are rare.

2276*C* Primary sarcoma of the pancreas has been met with, but it 2276*B* is a pathological curiosity, sarcomatous tumours in this situation being almost invariably secondary to disease in other organs.

## VIII. CALCULI.

**Pancreatic calculi** are usually multiple, and are found in the 2270 ducts. They are smooth or spinous, round or oval in shape, Series of and of an opaque white colour. Chemically, they consist of calculi. calcium, carbonate, or phosphate. They are usually found in 281 association with obstruction and dilatation of the duct; or 281*A* with chronic inflammation; or with carcinomatous deposits.

# CHAPTER XX

## DISEASES OF THE LYMPHATIC VESSELS AND GLANDS

I. Lymphangitis. II. Dilatation of Lymphatic Channels. Lymphangeiectasis and Lymphangeioma.—(1) Congenital; (2) Acquired. **Diseases of Lymphatic Glands.** I. Lymphadenitis. II. Tumours.—(1) Sarcoma; (2) Carcinoma; (3) Lymphadenoma. III. Pigmentation. IV. Leukaemia.

### I. LYMPHANGITIS.

(1) **Simple acute lymphangitis.**—Acute inflammation of lymphatic vessels is never a primary affection but is always due to the presence of an infected focus in the area drained by the affected vessels. In the case of the superficial vessels the inflammation is recognised by the appearance of red lines running from the infected focus to the nearest lymph gland. Occasionally the inflammation is of such severity as to lead to diffuse cellulitis.

(2) **Tuberculous lymphangitis.**—The lymphatic vessels draining a tuberculous focus may under suitable circumstances be seen to be studded with small tubercles. This is a common 2012$F_1$
event with the intestine. But even without becoming infected themselves the lymphatic vessels form a common and important means whereby the bacillus is disseminated within the body. This applies more particularly to the lungs.

(3) **Malignant lymphangitis.**—Malignant disease is spread to a large extent by lymphatic paths. For example, when the 3316
breast is the seat of cancer it is not infrequent to find a cord of new growth passing from the breast towards the axillary glands, this cord representing infiltrated lymphatic vessels.

But the cells of the growth may invade the lymphatic vessels, and reach the nearest lymphatic glands without further involving the vessels along which they pass.

## II. DILATATION OF LYMPHATIC CHANNELS.

Diffuse dilatation of these channels is termed **lymphangeiectasis**; circumscribed dilatations, forming tumours composed chiefly of dilated lymph-spaces, are called **lymphangeiomata.**

Lymphangeiectasis may be (1) congenital; (2) acquired.

(1) **Congenital lymphangeiectasis** produces a permanent enlargement of the district in which it is situated. Two of
1785*H* the commonest situations for these diffuse lymphatic enlarge-
1785*I* ments are the tongue and the lip (macroglossia, macrocheilia). But the whole of an extremity may be affected, and occasion-
3366*C* ally other parts of the body. The effect of the lesion is a great thickening of the subcutaneous tissues of the part concerned. The condition when it affects the skin is often called *spurious elephantiasis* to distinguish it from *true elephantiasis*, due to the filaria Bancrofti and presently to be described.

**Microscopic examination** shows a multitude of dilated lymphatic spaces lined by flattened endothelial cells.

(2) **Acquired lymphangeiectasis.**—The dilatation in this case may be due

(*a*) To the blocking of the lymphatic channels by parasitic worms;

Series of drawings. 519 (*b*) To the pressure of inflammatory exudations or tumours upon lymphatic trunks.

(*a*) This variety constitutes the tropical disease called *true elephantiasis.* The worm in question is "Filaria Bancrofti," a round worm some three or four inches long and one-hundredth of an inch thick (for fuller description, *v.* Chap. I.). It occupies the lymphatic channels in large numbers and there discharges its embryos. Each embryo is about one-ninetieth of an inch in length and of the diameter of a red blood-corpuscle. It lies within a transparent sheath and in fresh blood may be seen to move about with the greatest rapidity. These embryos enter the blood-stream by the thoracic duct. The intermediate host is a mosquito which sucks up the embryos with the blood of the affected person. The infected

mosquito presently dies after depositing its eggs in some collection of water, and from its dead body the embryos escape. With the water thus contaminated they gain access to the human stomach, and boring through the tissues, reach the lymphatic vessels. Here they become sexually mature and discharge a fresh generation of embryos.

**The lesions.**—The skin and subcutaneous tissues are immensely thickened and the bulk of the affected part correspondingly increased, whence the name elephantiasis. The cut surface shows wide lymphatic spaces, separated from each other by thick fibrous trabeculae and containing turbid lymph. The condition is seen most often in the lower extremities and the scrotum; but occlusion of the lymph-channels nearer the heart may lead to chylous effusions in the pleural and peritoneal cavities, and occasionally to the presence of chyle in the urine (chyluria). It should, however, be observed that chyluria is sometimes met with in temperate climates apart from any suggestion of elephantiasis. The cause of the condition in these cases is not understood. 2695*A* 2695*B*

(*b*) Acquired lymphangeiectasis depending upon the pressure of inflammatory effusions or tumours is not a condition of great importance.

## DISEASES OF THE LYMPHATIC GLANDS.

### I. INFLAMMATORY.

(1) **Lymphadenitis.**—Lymphadenitis is caused by bacteria or their toxins, and is most often seen as a local process in the glands serving an infected focus, *e.g.* the inguinal lymphadenitis which attends infections of the genitals. But some diseases are attended by a generalised inflammation of the lymphatic glands, particularly tuberculosis, syphilis in its secondary stage, and bubonic plague. Rubella or German measles is commonly attended by a swelling of the occipital glands, but the precise connection of this lesion with the disease in general is obscure. For descriptive purposes the tuberculous and syphilitic forms will be considered individually. The remaining varieties permit of inclusion in a general account.

Glands which are the seat of lymphadenitis are swollen, soft, and if the infection attacking them be virulent, may be
3382*L* dotted with areas of extravasated blood, this haemorrhagic
3382*M* tendency being a particular feature of bubonic plague. The inflammation may end in resolution or suppuration on the one hand, or in a chronic lymphadenitis. In the latter event the glands remain permanently enlarged owing to the gradual development of fibrous tissue in them. From the standpoint of medical practice this chronic lymphadenitis occurs most often in the glands at the angle of the jaw among children the subjects of chronic tonsilitis and chronic naso-pharyngeal irritation. In course of time the enlargement disappears owing to the gradual cicatricial shrinkage of the newly-formed fibrous tissue.

(2) **Tuberculous lymphadenitis.**—This is extremely common, and may be (*a*) primary; (*b*) secondary.

(*a*) **Primary tuberculous lymphadenitis.**—There is no doubt that the tubercle bacillus can pass through an intact mucous membrane and produce its lesions in the nearest lymphatic gland without having previously infected any part of the path which it has followed. The glandular systems most subject to such a primary invasion are the cervical, bronchial, and mesenteric. Tuberculosis of the cervical glands is the modern
2283*A* synonym of the old term "scrofula." Tuberculosis of the glands at the bifurcation of the trachea is a common incident of the post-mortem room in the bodies of children who present no evidences of tuberculosis in the lungs or elsewhere in the body, and the same is true of the mesenteric glands. The importance of this primary glandular tuberculosis lies in the frequency with which such latent foci appear to originate a general infection by the tubercle bacillus, and death from tuberculous meningitis.

(*b*) **Secondary tuberculous lymphadenitis.** — Although primary glandular tuberculosis is common, especially in childhood, tuberculous lymphadenitis is generally secondary to a tuberculous focus in the area drained by the glands in question. This secondary lesion is therefore generally local, but occasionally in people already the subjects of tuberculosis the whole lymphatic glandular system becomes rapidly involved out of proportion with the spread of the disease elsewhere.

**The lesions.**—In an early stage the glands are enlarged and on section show grey or yellowish points, proved by the microscope to be tubercles. Later these individual foci coalesce and caseate, while the capsule of the gland is thickened and becomes adherent to the surrounding tissues. The caseous accumulation thus formed may liquefy and form a tuberculous abscess, or, if the fibrous tissue reaction around it be well marked, may become entirely isolated by a strong fibrous capsule, and converted into a chalky concretion by the gradual deposit of lime salts within it. A glandular abscess will point in the direction of least resistance, and if deep-seated may cause grave results from the discharge of its contents. Thus a glandular abscess at the bifurcation of the trachea may erode a bronchus, and discharging its infected contents into the lumen of the air-tube, set up an acute tuberculous bronchopneumonia. 2281 2283*A* 2285 2286

(3) **Syphilitic lymphadenitis.**—The lymphatic glands serving an area which is the site of a primary chancre are always enlarged and hard. A more or less general enlargement of the lymphatic glands is also a common feature of the secondary stage of the disease. Syphilitic lymphadenitis never ends in suppuration.

**Microscopic examination.**—The prominent histological feature is the abundance of plasma-cells (*v.* p. 6).

## II. TUMOURS.

(1) **Sarcoma.**—The lymphatic glands may be the seat of primary round-celled sarcoma, and to such a growth the term *lymphosarcoma*, if used at all, should be restricted. The glands of the mediastinal regions are particularly liable to this kind of growth. Spindle-celled sarcomata have also been described. Secondary sarcomatous deposits are commonly met with. 2309$B_1$ 2309$B_2$ 2294*B* 2294*C*

(2) **Carcinoma.**—The lymphatic glands are the commonest sites of secondary carcinoma and are liable to early implication. The lymphatic channels leading from the affected organ to the nearest lymph glands are also frequently involved. 2287*B* 2287*C*

(3) **Lymphadenoma** or Hodgkin's disease is an affection of uncertain etiology, characterised by enlargement of one or more groups of lymphatic glands, spleen, and liver. The Series of drawings. 516*A*

glands are enlarged by a diffuse overgrowth of their framework which obscures the distinction between cortex and medulla. They vary in consistency according to the amount of fibrous tissue present, being sometimes of almost rocky hardness, at others quite soft. They are usually free from adhesions, at least in the early stages, and never caseate or
2278*B* suppurate unless as a sequel of a secondary infection. On
2278*C* section they are reddish or yellow in colour, and homogeneous. The white deposits met with in the liver, spleen, kidneys, bone, etc., possess the same microscopical characters as the glands.

**Microscopic examination** of a lymphadenomatus gland shows that the lymphocytes are reduced in numbers, while the fibrous trabeculae are increased in thickness. Endothelial cells are much more numerous than in a normal gland: they often attain a large size and contain several large, deeply-stained nuclei (lymphadenoma cells). The abundance of these cells is a distinguishing feature of lymphadenoma.

## III. PIGMENTATION.

The bronchial glands of adults, especially those whose occupations have been dusty, *e.g.* coal-miners, are always
2284*B* blackened by deposited particles of carbon and dust. Glands in which the deposit is abundant are generally enlarged and show an excess of fibrous tissue. Pigmentation occurs also in consequence of *chronic malaria*, the pigment being derived from the red blood-cells destroyed by the parasite: in *Addison's disease:* and as a sequel of *haemorrhagic affections* of the glands.

## IV. LEUKAEMIA.

Lymphatic leukaemia is characterised by a generalised enlargement of the lymphatic glands, especially those of the neck, axillae, and groins. On section they are of a deep red colour and resemble the tissue of the spleen. In consistency they are soft, and remain discrete and freely movable. Widespread glandular enlargement is an occasional feature of spleno-
2277*A* medullary leukaemia.

# CHAPTER XXI.

## DISEASES OF THE SPLEEN.

I. Anatomical Variations. II. Perisplenitis. III. Circulatory Disturbances. IV. Inflammation. V. Degeneration. VI. Pigmentation. VII. Lymphadenoma. VIII. Spleno-medullary Leukaemia. IX. Anaemia Pseudo-leukaemica Infantum. X. Hydatid Cysts. XI. Tumours.

### I. ANATOMICAL VARIATIONS.

(*a*) Small supernumerary spleens (splenculi), are common, and are usually situated in the immediate neighbourhood of the main organ. 2301*B*

(*b*) Marked lobulation is occasionally seen. 3382*O*

(*c*) Transposition of the spleen to the right side occurs in a majority of the cases in which other viscera are transposed. 3401*A*

(*d*) Abnormal mobility is common. It is generally attended by a similar affection of other intestinal viscera, such as the kidneys and stomach, but may occur alone.

(*e*) Atrophy of the spleen occurs in old people, and in those who have died from wasting diseases such as diabetes mellitus. The spleen in these instances is shrunken, its capsule wrinkled, and its cut surface abnormally dry. 2295

### II. PERISPLENITIS.

Inflammation of the peritoneum covering the spleen is common. It may depend upon lesions of various kinds within the spleen, but is more often an incident in a general peritoneal

inflammation. The most marked examples are those which attend the chronic forms of peritonitis found among the subjects of arteriosclerosis, cirrhosis of the liver, and granular kidney. The whole surface of the spleen is covered by a white membranous deposit, often pitted by circular indentations. The capsule of the gland may be much thickened and form a dense fibrous cuirasse, while the splenic tissue itself is unduly
2297 firm owing to an increase in its fibrous elements. The liver
2297*B* commonly and the intestines sometimes are similarly affected.

## III. CIRCULATORY DISTURBANCES.

(*a*) **Congestion.**—Any lesion which obstructs the return of blood from the spleen produces a venous engorgement. Thus cirrhosis of the liver, owing to the portal obstruction attending it, and chronic valvular disease of the heart in the stage of failing compensation, both produce congestion of the spleen. In the former case the gland is enlarged, often
2296*A* greatly, and unduly firm. In the latter case it may be enlarged, but is always extremely tough and dark-coloured (cardiac spleen).

(*b*) **Infarction.**

Infarction of the spleen is as a rule due to embolism of a branch or branches of the splenic artery by emboli derived
2295*K* from an endocarditis of the valves or chambers of the left side of the heart. But on rare occasions it is due to thrombosis of the splenic artery or vein at the instance of some adjacent inflammation which involves the vessels, such as a gastric
1914*C* ulcer.

The enlarged spleens of spleno-medullary leukaemia often
2295$G_2$ show multiple areas of infarction.

Infarctions of the spleen may be single or multiple, and lead to an enlargement of the gland. They are irregularly pyramidal in outline, the base being on the periphery, the apex directed towards the hilum. They may be yellowish in colour
2295*C* (anaemic infarcts), or deep-red (haemorrhagic infarcts), more often the former; but occasionally an infarct, anaemic in its
2295$F_1$ deeper parts, is haemorrhagic at its edges. Recent infarcts usually project slightly above the surrounding surface. The peritoneum over them is commonly thickened and covered
2295*K* with an exudation of lymph. On section the infarcted area is

seen to be surrounded by a reddish zone produced by dilatation of the neighbouring capillaries, while at the apex the occluded vessel responsible for the lesion may sometimes be detected.

**Fate of infarcts.**—When due to an non-infective embolus an infarct slowly contracts and is finally reduced to a mass of fibrous tissue sometimes infiltrated by lime-salts. Such scarring often leads to marked deformity of the gland. If the 2295*E*
embolus is infective the infarct commonly suppurates, and 2295$F_2$
is converted into an abscess. Large portions of the spleen 2295*O*
may be destroyed in this way. In favourable cases the suppurating area becomes enclosed in a fibrous envelope produced by the reaction of the adjoining tissues; its contents become inspissated and impregnated with lime-salts, the whole finally appearing as a deep cicatrix enclosing a concretion. In less favourable instances the abscesses may rupture either into the peritoneal cavity or intestine. 2295$F_2$

(*c*) Haemorrhages into the substance of the spleen, in the form of small capillary extravasations, are common in many infective conditions, and are frequently met with in the bodies of infants who have died of marasmus or diarrhoea.

## IV. INFLAMMATION.

(1) **Acute inflammation.**—The spleen becomes inflamed and enlarged in the course of a number of infective diseases, par- 2296*B*
ticularly typhoid fever. Such inflammatory enlargements are attended by softening, the splenic pulp being sometimes almost diffluent.

**Microscopic examination** in these cases shows numerous areas of necrosis, the splenic pulp being here replaced by a degenerate débris of fibrin, necrotic cells and fat-globules. The periphery of these areas is crowded with polymorphonuclear leucocytes.

(2) **Tuberculosis.** (*a*) **Miliary.**—In the course of acute miliary tuberculosis, especially during childhood, the spleen is almost invariably affected. It is studded with closely-set grey or yellow nodules which are conspicuous objects against the red or scarlet background of the gland, and almost invari- 2301*B*
ably involve the capsule. 2301*D*

(*b*) **Caseous.**—Caseous tubercles are of common occurrence 2303
when tuberculosis is chronic and generalised. They vary 2303*A*
greatly in abundance and size, and may lead to the develop-

2299*B* ment of calcareous masses. Tubercles in the spleen, whether miliary or caseous, are often confined to the capsule and the tissue immediately beneath it.

(3) **Syphilis.** (*a*) **Gummata.**—These are not common in the spleen. When they do occur they form either discrete,
2299 firm, caseous masses; or, more rarely, soft tumours involving
2299*C* large areas of the gland. In either case they tend to produce
2299*D* in course of time much cicatricial contraction and deformity.

(*b*) Congenital syphilis produces an enlargement of the spleen. This enlargement is seldom due to gummata but depends rather upon a diffuse fibrosis which renders the spleen unnaturally hard. Amyloid degeneration is a common accompaniment of this lesion.

2306*C* (4) **Actinomycosis** occasionally affects the spleen.

## V. DEGENERATION.

**Amyloid degeneration** of the spleen appears in two forms.

(1) The "**Sago-spleen.**"—In this variety the amyloid
2298*C* deposits are more or less confined to the Malpighian bodies,
2298*D* and convert them into translucent granules bearing some
2298*E* resemblance to grains of sago.

(2) The "**Bacon-spleen.**"—In this variety the deposit is diffuse.

In both cases the spleen is enlarged and hard. (For an account of amyloid degeneration in general, *vide* p. 15.)

## VI. PIGMENTATION.

Pigmentation, affecting chiefly the capsule and superficial parts, is met with in some cases of cirrhosis of the liver, and in the enlarged spleens associated with chronic malarial
2303*A* cachexia.

## VII. LYMPHADENOMA.

Lymphadenomatous deposits in the spleen are a constant feature of this disease. In the common form the gland is much
2305*C* enlarged, hard, and studded with nodules about the size of a
2305*D* pea. (The "**hard-bake** spleen.") These nodules present some resemblance to tubercles but are firmer than the latter, and

more sharply defined. In anomalous cases lymphadenomatous deposits in the spleen may assume the form of large white infiltrating tumours. 2305*E* 2305*F*

(For the histology of lymphadenoma, *vide* p. 199.)

## VIII. SPLENO-MEDULLARY LEUKAEMIA.

In this disease the spleen is always enlarged, and sometimes to such a degree that it may reach the pelvis. It is hard, and often adherent to its surroundings, but maintains its normal outline. On section it is extremely tough, and often mottled by yellow necrotic areas shown by the microscope to be anaemic infarctions. 2295*G* 2295$G_2$

**Microscopic examination.**—The splenic tissue is practically destroyed and replaced by an abundant fibrous tissue containing few cells, and these more or less necrotic. The infarcted areas are completely degenerate.

## IX. ANAEMIA PSEUDO-LEUKAEMICA INFANTUM.

This disease is characterised by profound anaemia and the presence of myelocytes in the blood, but differs in important respects from spleno-medullary leukaemia. Enlargement of the spleen is a constant feature. 2295$G_1$

## X. HYDATID CYSTS.

2306*B*

Hydatid cysts are occasionally found in the spleen. 2306$B_1$

## XI. TUMOURS.

Both carcinoma and sarcoma may attack the spleen, though rarely. Carcinoma in this situation is invariably secondary to the disease elsewhere, and sarcoma also as a general rule. But primary sarcoma is occasionally met with here. 2304*C* 2304*B* 2304*E* 2304*D*

# CHAPTER XXII.

## DISEASES OF THE THYMUS GLAND.

1. Hypertrophy and Lymphatism. 2. Tumours (Mediastinal Tumours).

The thymus gland consists of two elongated fleshy lobes lying upon the anterior aspect of the pericardium in its upper part. The function of the gland is not known. It reaches its maximum size during the second year of life and thereafter undergoes atrophy, though at all stages traces of glandular tissues may be discovered by microscopic examination of its atrophied remains.

The thymus is occasionally the seat of abscess-formation, and of tuberculous deposits, but with two exceptions the lesions affecting it are not of much importance. These exceptions are hypertrophy, and malignant tumours.

2314*T* 2314*U* 1. **Hypertrophy of the thymus** occurs in many cases of Graves' disease, and of myasthenia gravis, but here it has no known significance. It is also met with in a good many instances of sudden death affecting infants, and more rarely, adults. It is usually stated that the cause of death in these cases is asphyxia due to compression of the trachea by the enlarged gland, but the accuracy of the statement is extremely doubtful. A variety of paroxysmal dyspnoea, *thymic asthma*, has also been credited to the pressure of an enlarged thymus gland, but here too the connection between the pathological and clinical phenomena is obscure.

Paltauf holds that such enlargements of the thymus are a part of a general disease affecting the lymphatic structures throughout the body—the lymphatic glands, the tonsils, the

lymphatic follicles of the pharynx, stomach and intestine—and that the disease damages the heart either by alteration of its muscular tissue or by the upsetting of the nervous regulation of cardiac rhythm. To this generalised lymphatic overgrowth the term **lymphatism** is applied. 2309 2309*C*

2. **Malignant tumours.**—The thymus gland appears to be the seat of origin of a considerable number of mediastinal new-growths.

Histologically the types met with are:

(1) Small round-celled sarcoma. 2309*B*. 2309$B_1$.
(2) Endothelioma. 2309$A_1$.
(3) Carcinoma (rare). 2309*A*.

# CHAPTER XXIII.

## DISEASES OF THE THYROID GLAND.

1. Hypertrophy.—(*a*) Parenchymatous Goitre; (*b*) Exophthalmic Goitre. 2. Atrophy.—(*a*) Congenital (Cretinism); (*b*) Acquired (Myxoedema and Cachexia Strumipriva). 3. Inflammations. 4. Tumours. 5. Pressure Effects of Thyroid Enlargements in General.

The thyroid gland is developed from a diverticulum which, originating in the floor of the pharynx, unites with two lateral diverticula from the fourth bronchial cleft. Traces of this origin are found in the occasional presence of a median pyramidal lobe, and in the persistence, complete or partial, of the thyroglossal duct. The latter structure is the point of origin of many of the cysts which develop in the middle line of the neck.

The parathyroid glands are four small glands situated, in man, upon the posterior surface of the thyroid gland. The upper, or internal, pair, may be imbedded in the substance of the thyroid. They are composed of solid columns of epithelial cells, and contain no colloid. The function of the parathyroid glands is not known, but removal of them, in the case of animals at least, causes convulsions (tetany).

Accessory thyroid glands may occur in many situations from the base of the tongue to the arch of the aorta, and are occasionally the seat of disease.

*N.B.*—**Goitre** is a generic term for all enlargements of the thyroid gland. The term thus used includes hypertrophy, adenomata, cysts, and malignant growths, the variety being signified by a qualifying adjective, *e.g.* cystic or malignant goitre.

## 1. HYPERTROPHY.

(*a*) **Parenchymatous goitre.**—Parenchymatous goitre indicates a general and more or less uniform enlargement of the thyroid gland. It occurs sporadically everywhere, but in certain hilly districts is endemic. The special liability of such districts has been variously credited to the practice of drinking water derived from melted snow or ice, and to the presence in the water of lime-salts in large quantities, but the causal relations of the disease are still in doubt. The hypertrophied gland maintains its normal outlines as a rule, but may be deformed. Fibrosis and partial calcification are occasional late events. 2311*C* 2311*D* 2312*C* Drawings. 568 561

**Microscopic examination** shows that the hypertrophied tissue does not differ materially from what is normal.

(*b*) **Exophthalmic goitre.**—(Graves' disease. Basedow's disease). This disease in its typical form is associated with a pulsating enlargement of the thyroid gland. The enlargement is uniform, not extreme, and depends largely upon the dilatation of the blood-vessels within the gland, though there is also some true hypertrophy. Drawings. 569

**Microscopic examination** shows that the gland is abnormally cellular, and approximates to the foetal type. The alveoli are irregular in size and shape, and exhibit a deficiency of colloid material.

Hypertrophy of the thymus gland is a constant accompaniment of the severe forms of exophthalmic goitre. 2314*T* 2314*U*

## 2. ATROPHY (CRETINISM AND MYXOEDEMA).

Atrophy of the thyroid gland may be (*a*) congenital; (*b*) acquired.

(*a*) **Congenital atrophy** is the cause of a form of imbecility called **Cretinism**. A cretin is undersized and mentally deficient. The physiognomy is striking. A short broad skull; a marked depression of the nasal bones associated with widening of the alae nasi; tumid eyelids and thick protuberant lips. The expression is stupid; the body short; the abdomen protuberant; the limbs short and thick. The epiphyses are large, while the shafts of the bones, though short and thick, are normally formed. The skin is dry and harsh; the hair scanty, and the genital organs remain infantile. A Drawings. 570 570*A*

common feature is the development of thick pads of fat above the clavicles. Cretinism in this country is sporadic, but in
2317*C* certain places, especially where endemic goitre prevails, it
Drawing, also may be endemic. The thyroid gland is usually atrophied,
572 but may be absent altogether or diseased.
2317*A* (*b*) **Acquired atrophy** is the cause of **myxoedema**, a disease of
2317*B* adult life and characterised by a wide-spread thickening of the
Drawing, subcutaneous tissues owing to an increase both of their fibrous
585 and fatty components. The name was given to the disease under the belief that this thickening was due to a deposit of mucin, a belief now shown to be in most cases erroneous. The physical features of cretinism are reproduced, while the mental powers show a gradual decline. A peculiar slowness both of speech and action is a pronounced feature of the disease.

Myxoedema is far more common among women than men. Although for the most part associated with unaccountable atrophy of the thyroid gland, it may follow an attack of exophthalmic goitre, or appear in a person the subject of ordinary parenchymatous goitre. A condition very similar to
Drawings. myxoedema results from surgical removal of the thyroid
576 gland. It is usual to distinguish this form by the name
577 *Cachexia strumipriva.*

## 3. INFLAMMATIONS.

Inflammations of the thyroid gland are rare, but occasionally occur in connection with bacterial infections elsewhere, and
2319*E* may proceed to suppuration. Tuberculous and gummatous deposits are met with from time to time.

## 4. TUMOURS.

2310*C* (*A*) **Innocent tumours.** (1) **Adenomata.**—Thyroid adeno-
2310*D* mata form encapsulated tumours embedded in the tissue of the
2314*O* gland. They are generally unilateral, may be multiple, and
2314*Q* often become the seat of cystic degeneration. In appearance
2314*R* they bear a general resemblance to normal thyroid tissue.

**Microscopic examination** shows that there are two types of thyroid adenoma. The one resembles the normal gland, the other conforms to the pattern of the foetal thyroid, viz. closely packed columns of cells and an absence of colloid material.

(2) **Cysts.**—Multilocular cysts are met with both in otherwise normal and in hypertrophied thyroid glands, as a result of distension of the thyroid follicles by colloid secretion. Unilocular cysts are usually derived from the degeneration of thyroid adenomata, and are therefore commonly unilateral. Thyroid cysts contain a clear serum or blood-stained débris, for haemorrhage into the cysts is common. Drawings 546 551 2314*M* 2314*L* 2314*P*

(*B*) **Malignant tumours.**—Both carcinoma and sarcoma occur, the latter being the more common. 2318*B* 2318*C*

## 5. PRESSURE EFFECTS OF THYROID ENLARGEMENTS IN GENERAL.

These are exercised chiefly upon the trachea, which may be displaced by a unilateral goitre, flattened from side to side by enlarged lateral lobes, or flattened from before backward by an enlarged isthmus or substernal goitre. The resulting diminution in the calibre of the trachea is a frequent cause of dyspnoea. The degree of dyspnoea in any given case depends upon (1) the extent of the narrowing; (2) the rapidity with which the narrowing is established. Thus the severest dyspnoea is caused by goitres which have grown quickly or which have undergone a rapid accession of bulk owing to haemorrhage into them. Occasionally thyroid tumours cause abductor paralysis of the larynx by pressing upon and paralysing the recurrent laryngeal nerve in the neck. 2310

# CHAPTER XXIV.

## DISEASES OF THE ADRENAL BODIES.

1. Accessory Adrenals. 2. Atrophy. 3. Amyloid Disease. 4. Haemorrhage. 5. Tuberculosis. 6. Addison's Disease. 7. Tumours.

**1. Accessory adrenals.**—These are common. They form small solid tumours, usually situated in the capsule of the kidney or immediately beneath it; but they have been observed in the broad ligaments, in the uterus, in the liver, and in the connective tissue of various parts of the abdomen. Their chief importance lies in their liability to originate malignant tumours, especially in the kidney. Microscopically, they may reproduce the structure of the true gland perfectly, but more often they depart from the normal arrangement of the glandular elements and assume the histological features of adenomata.

**2. Atrophy** occurs (i) as a senile change, (ii) without any obvious cause, but associated with the symptoms of Addison's disease.

**3. Amyloid disease** occurs with some degree of frequency; it is found, in connection with similar changes in other organs, in cases of prolonged cachexia due to tuberculosis, syphilis,
2320 or suppuration.

**4. Haemorrhage into the adrenals** is not common, but occurs as follows:

2320*A* (i) In cases of epilepsy and pertussis, diseases associated with the paroxysmal elevation of the blood-pressure

(ii) In new-born infants delivered after a difficult labour.

2320*B* (iii) As an unexplained event in fat and apparently

healthy infants. The clinical features are symptoms of acute disease within the belly and death within a few hours. In some of these cases the adrenal haemorrhage is accompanied by a profuse purpura. 2320*C* 2320*D*

(iv) In septicaemic diseases. 2320*E*

5. **Tuberculosis.**—(i) Miliary tuberculosis is of no great moment. (ii) *Fibro-caseous tuberculosis* of the adrenals is the lesion most frequently associated with the symptoms of *Addison's disease.* The adrenal bodies are much enlarged, and consist of fibrous tissue enclosing caseous material. The neighbouring organs are often affected by an extension of the process, particularly the semi-lunar ganglia and their connections. The lesion is usually bilateral, and secondary to tuberculosis elsewhere. Calcification of portions of the tuberculous deposits is not uncommon. 2325*A* 2325*B* 2323

6. **Addison's disease.**—This is a chronic fatal malady, characterised by progressive emaciation, asthenia, marked pigmentation of the skin, and persistent vomiting. It is associated in the vast majority of cases with a lesion of the adrenal bodies. In from 70 % to 80 % of all cases, this lesion is a chronic fibro-caseous tuberculosis; other recorded lesions are simple atrophy, malignant tumours, and haemorrhagic extravasations. On the other hand, Addison's disease does undoubtedly occur without recognisable alterations in the adrenal bodies, and further, marked changes may be present in the adrenal bodies without the supervention of Addison's disease.

7. **Tumours.**—Tumours of the adrenals themselves, as opposed to accessory adrenals, are so rare as not to be of much significance, but carcinoma, sarcoma, adenoma, and cysts are occasionally encountered. 2327*B* 2330$B_1$ 2330*C*

# CHAPTER XXV.

## DISEASES OF THE KIDNEYS AND URETERS.

I. Malformations. II. Malpositions. III. Hypertrophy. IV. Atrophy. V. Circulatory Disturbances. VI. Nephritis. VII. Degenerations. VIII. Tuberculosis. IX. Syphilis. X. Uratic Deposits and Uric Acid Infarcts. XI. Nephrolithiasis. XII. Pyelitis. XIII. Hydronephrosis. XIV. Pyonephrosis. XV. Tumours. XVI. Parasites.

### I. MALFORMATIONS.

Malformations of the kidney are common. The most frequent are :

(i) Entire absence of one kidney, usually the left, often accompanied by a misplacement of the single kidney, *e.g.* into the hollow of the sacrum.

2331*G* (ii) Smallness or atrophy of one kidney, usually the left,
2331*H* with a corresponding hypertrophy of the other. In some cases the atrophy appears to be developmental and due to a
3666*A* deficient blood supply produced by a vascular fault, such as abnormal smallness (hypoplasia) of the renal artery ; but in
2361 others it is due to intra-uterine inflammation, or to atresia of the ureter.

(iii) Fusion of the two kidneys across the middle line (“*horseshoe kidney*”). This is the most common of all mal-
3651*B* formations. The fusion is usually situated at the lower pole,
3652*A* and is effected by a fibrous band, or a bridge of true renal substance ; it is usually accompanied by abnormalities in the number or arrangement of the vessels and ureters.

(iv) Supernumerary kidneys have been observed, though rarely.

(v) Doubling of the ureters is common; the pelvis may be common to the two, or each ureter may arise from a separate and distinct pelvis; they may run independently, entering the bladder by separate orifices, or may coalesce in any portion of their course. 2368*A* 2338*F* 3661*A*

## II. MALPOSITIONS.

Malposition of the kidney is more common in women than in men, and affects the right kidney by preference. The malposition may be: (*a*) congenital, (*b*) acquired.

(*a*) **Congenital malposition.**—In this form the affected kidney is usually found fixed at an abnormally low level and may lie in the pelvis or over the sacrum, while in rare instances both kidneys lie on the same side, one below the other. These deformities are attended by abnormal arrangements of the ureters and blood-vessels of the misplaced organ. 3660*C*

(*b*) **Acquired malposition**, "movable" or "floating kidney," is more frequent in women than in men, and most often affects the right kidney only; but both kidneys may suffer, or the left alone, the last event being the rarest of all. It may occur at any age, but as a rule is observed in women between the ages of 20 and 45. The reputed causes of the condition are manifold: relaxation of the abdominal walls due to repeated pregnancies, shallowness of the praevertebral niche, tight-lacing, traumatism, the lifting of heavy weights, and rapid emaciation, have all been cited. The most important of these are probably the first two, but there is no reasonable explanation of the greater liability of the right kidney to displacement. The kidney may vary in mobility from the slightest degree to such an extent that it can be readily moved to any part of the abdominal cavity. Such "floating kidneys" may become fixed in an abnormal position.

**Effects of movable kidney.**—Torsion or kinking of the ureter, accompanied by paroxysmal pain (Dietl's crises), and leading to intermittent or permanent hydronephrosis, may result from the abnormal mobility. But it is important to observe that a high degree of mobility of the kidney is compatible with complete freedom from all untoward symptoms.

## III. HYPERTROPHY.

2331*F* 2331*G* Hypertrophy of one kidney is usually caused by absence of function in, or removal of, the opposite organ. The hypertrophy affects all parts of the kidney, cortex as well as medulla, and, in growing subjects, is "numerical," *i.e.* the elements are multiplied as well as enlarged.

In the very obese, in beer-drinkers, and occasionally in the subjects of diabetes mellitus and insipidus, both kidneys may be enormously hypertrophied without showing any pathological change in their tissues.

## IV. ATROPHY.

3666*A* 2361 2331*K* 2331 2335*A* 2331$E_2$ 2331*L* The kidney may be congenitally small. It may also become atrophied by disuse, as is the case when the ureter is blocked, or the kidney itself, or its blood supply, interfered with by a new growth. Atrophy may follow extensive embolism, or the contraction of gummatous or other inflammatory exudations in the perinephric tissue. Lastly, in the subjects of arteriosclerosis and chronic interstitial nephritis both organs are often very small as a result of cicatricial shrinkage of newly formed fibrous tissue within them. Localised atrophy is usually due to embolic or other interference with the blood supply.

## V. CIRCULATORY DISTURBANCES.

(*a*) **Anaemia** of the kidney is found in cachectic conditions, in profound general anaemias, and in the case of thrombosis or embolism of the renal artery. It occurs also in association with strychnine-poisoning, lead-poisoning and eclampsia, as a result, it is said, of spasm of the arterioles. Pernicious anaemia causes a deposit in the kidneys, and some other organs, of an iron-containing pigment (haemosiderin) derived from the 3220$C_1$ destruction of red blood-cells. Haemosiderin when treated with hydrochloric acid and potassium ferro-cyanide produces the Prussian-blue reaction.

2320*E* (*b*) **Hyperaemia.—Active congestion** occurs in fevers and intoxications, and in acute inflammatory conditions. The kidney is large, soft, and dark red on section.

**Passive congestion** is most often due to chronic heart

disease, but sometimes to thrombosis of the renal vein. The 2331*L*
kidney is large and firm; the stellate veins are engorged; on 2331*M*
section the pyramids are dark purple in colour, the cortex 2331*I*
being slightly paler and often marked with venous striae; the capsule is usually—especially in cases of long standing—adherent to the kidney.

**Microscopic examination** shows marked engorgement of all vessels and often an increase in the connective tissues.

(*c*) **Haemorrhages** into the kidneys are usually due to lesions of the capillaries, to injury, or to infarction. Capillary haemorrhages are found as the result of minute infective 2389*C*
emboli in cases of malignant endocarditis. Haemorrhage into the pelvis of the kidney may occur in the course of purpura, 2389
scurvy, and in the haemorrhagic forms of the infectious fevers. 2389*D*

(*d*) **Thrombosis.**—Thrombosis of the renal vein leads to congestion of the kidney and sometimes to extravasation of blood within it. It occurs occasionally in infants born after a 2331*I*
difficult labour, or dead as a result of wasting diseases. The left vein, perhaps on account of its longer course, is affected more frequently than the right. Thrombosis of the renal artery is a rare affection.

(*e*) **Embolism and infarction.**—Embolism of the arterioles of the cortex of the kidney is a frequent occurrence, and, as elsewhere in the case of "terminal" vessels, leads to the formation of infarcts.

Infarcts are divided according to the character of the embolus which causes them into (1) non-infective or simple infarcts, and (2) infective infarcts; and according to the appearances they present into (i) anaemic, (ii) haemorrhagic, (iii) purulent infarcts.

(i) Simple infarcts are usually anaemic; they are conical in 2331$E_3$
shape, with the apex towards the medulla; of a dull white or yellowish colour; of a firm consistency; and project slightly above the level of the normal surface. When recent they are surrounded by a red areola of dilated vessels. Their size varies but is seldom less than $\frac{1}{4}$ inch in diameter.

(ii) Haemorrhagic infarcts may belong to the non-infective group, but more usually are due to infective emboli; they are comparatively rare in the kidney. In shape they resemble the anaemic variety, but are purple or dark red in colour, of a softer consistency, and often considerably smaller.

Non-infective infarcts are gradually replaced by fibrous tissue the shrinkage of which leads to the formation of
2331*L* puckered scars. When these are numerous the kidney is often extraordinarily deformed, even the non-infarcted areas undergoing atrophy and degeneration under the pressure exerted by the contracting scars.

(iii) Purulent infarcts belong solely to the infective group. In their earliest stage they are mottled red and white areas,
2331$E_5$ obviously purulent to the naked eye; but eventually the affected tissue is transformed into an abscess. In shape they are not so often conical as the other varieties of infarct, nor is their margin so accurately defined; and in size they vary from that of a split pea or less, to abscesses involving a third of the kidney substance.

## VI. NEPHRITIS.

Many classifications of nephritis have been proposed, but none is wholly satisfactory. That here adopted is based partly upon anatomical, partly upon clinical grounds, and is used only for purposes of description.

(The term "Bright's Disease" was originally given to any diseased condition of the kidneys associated with dropsy and albuminuria; but was gradually extended to cover any form of inflammatory renal disease. Its significance is therefore extremely vague.)

### (*A*) Acute Nephritis.

(i) Acute diffuse nephritis (acute parenchymatous or glomerulo-tubular nephritis).

(ii) Acute interstitial nephritis.

### (*B*) Chronic Nephritis.

(i) Chronic diffuse nephritis without induration (large white kidney; chronic parenchymatous nephritis).

(ii) Chronic diffuse nephritis with induration (contracted white kidney, secondary chronic interstitial nephritis. Small white kidney).

(iii) Primary chronic interstitial nephritis (gouty kidney. Small red kidney. Granular kidney). *Sub-variety.* Arteriosclerotic kidney.

### (*C*) Suppurative Nephritis.

*N.B.*—The term *chronic interstitial nephritis* is used to cover all forms of kidney disease in which the organ is smaller and harder than is natural. Similarly the terms "cirrhotic" and "granular" are not limited to any one variety.

(*A*) (i) **Acute diffuse nephritis** (acute parenchymatous nephritis).—The appearance of the kidney varies with the duration and intensity of the disease, and the amount of blood which is present in the organ. It is usually considerably enlarged, soft, and varies in colour from a deep purple to a greyish red. The capsule is tightly stretched, and retracts 2332
when incised, allowing the renal tissue to bulge and overlap the retracted edges. The capsule is easily detached, leaving a smooth surface marked by engorged venae stellatae. On section of the kidney the cortex is seen to be wider than usual; its striations are abnormally far apart, and are sometimes indistinct, sometimes prominent by reason of the vascular engorgement. It is often mottled by minute haemorrhagic points, which represent the engorged and enlarged glomeruli. The junction of the cortex and medulla is dark red, as are the pyramids, all the blood-vessels being so congested that in pronounced cases blood will drip from the organ after section.

Histologically, the glomeruli, the tubes, and the interstitial tissue all share in the inflammatory changes. The chief features are a fluid exudation into Bowman's capsule, together with proliferation and degeneration of the cells which line it; fatty degeneration of the cells which line the convoluted tubules, and an accumulation of inflammatory products in their lumina, forming "casts"; such casts may consist of red blood cells, of leucocytes, of desquamated epithelial cells, or of a granular detritus undergoing mucoid degeneration. Lastly, the interstitial tissue, especially in the neighbourhood of blood-vessels, is infiltrated by a fluid exudation containing many small round cells. These changes may be combined in all proportions in different cases. Hence a case in which the brunt of the disease has fallen on the glomeruli is spoken of as a glomerular nephritis; where the tubes are chiefly involved it is a tubular nephritis; when the exudation and desquamation are marked it is a catarrhal or desquamative nephritis; and if capillary haemorrhages be a prominent feature it is a haemorrhagic nephritis.

(*A*) (ii) **Acute interstitial nephritis.**—In the majority of cases of acute nephritis the lesions are most marked in the tubes and glomeruli, while the interstitial changes are slighter. But occasionally, and this is particularly the case with the forms of acute nephritis associated with infectious diseases in childhood, the changes are almost confined

to the interstitial tissue, the glomeruli and tubes escaping serious damage. To the naked eye there is but little difference between this form and acute diffuse nephritis.

Histologically there is a well-marked round-cell infiltration of the interstitial tissue, especially round the glomeruli, beneath the capsule, and in the intermediate zone. The glomeruli and the tubes are little, if at all, affected.

The causes of these two forms of acute nephritis are:

(i) Microbic diseases, especially scarlet fever, diphtheria, small-pox, infective endocarditis, and pneumonia.

(ii) Poisons of various kinds, carbolic acid, turpentine, cantharides, oxalic acid and some others.

(iii) Exposure to cold; clinically this is an undoubted predisposing cause, but whether it suffices alone to produce nephritis is doubtful. Probably exposure acts by lowering the resistance of the tissues and rendering them susceptible to the action of toxins which they would in ordinary circumstances excrete without suffering harm.

(iv) Poisons elaborated within the body by defective or abnormal metabolic processes. None of these hypothetical bodies have been as yet detected; but their existence affords the only reasonable explanation of certain obscure forms of nephritis such as nephritis occurring during pregnancy.

(*B*) (i) **Chronic diffuse nephritis without induration** (chronic parenchymatous nephritis. Large white kidney).

It is not possible to draw a sharp line of demarcation between acute and chronic forms of diffuse nephritis. The acute and sub-acute pass by imperceptible gradations into the chronic varieties. Of the latter the type is the "*large white*
2332*B* *kidney.*" The organ is enlarged, sometimes to twice its normal
2332*C* size; it is usually pale grey, yellow, or white in colour, with but little differentiation between the cortex and medulla, though on occasion the pyramids are deeply congested, and afford a marked contrast to the white cortex. The latter is increased in depth; its colour is white, yellow, or a mottled red and yellow, and the striations upon it are indistinct. On section the tightly-stretched capsule retracts, and the cut surface of the cortex bulges, becoming convex. The kidney is soft and sometimes friable; it may exude an oily fluid when incised, owing to fatty degeneration of its elements. The capsule is usually adherent to the cortex and when

detached brings away small pieces of the kidney surface with it.

Although the "large white kidney" is usually accepted as the typical form of chronic parenchymatous nephritis, in practice the more common variety is the mottled red and yellow form. In this the red colour predominates, inter- 2332*B*
spersed with yellow and opaque creamy patches. The surface in these cases is more granular than in the case of the large white kidney, and the capsule as a rule is more adherent.

Histologically, tubes, glomeruli, and interstitial substance are alike the seat of degenerative and proliferative changes, the degenerative predominating. In the glomeruli the exudation and proliferation seen in acute nephritis are present, but are more marked, and more universal; few glomeruli escape altogether. The tubules are dilated, their epithelium is the seat of extensive fatty degeneration, and their lumina are filled with a granular detritus, and desquamated cells. Casts are nearly always present in the straight tubules. The interstitial substance is oedematous and in places shows areas of round-cell infiltration. In the "large white" variety there is no marked increase of the interstitial connective tissue, but in the mottled red and yellow kidneys the connective tissue elements are increased in quantity.

(*B*) (ii) **Chronic diffuse nephritis with induration** (secondary chronic interstitial nephritis: contracted or small white kidney).—When chronic diffuse nephritis is of some duration, the proliferated connective tissue tends to undergo cicatricial contraction, the whole kidney becoming smaller. In its extreme forms this is known as the "*small*" or "*contracted white kidney.*" The small white kidney is firm, tough, shrunken, 2334*B*
and nodular upon the surface; the capsule is firmly adherent 2334*D*
to the cortex, and tears it during removal; the cortex is diminished in depth, and often contains small retention cysts due to cicatricial occlusion of tubules and the accumulation of secretion behind the sites of occlusion.

Histologically the glomeruli are especially affected, being transformed in many instances into spherical hyaline areas showing no trace of their original structure. The connective tissue is everywhere increased, but notably round the glomeruli. The tubules are in places denuded of epithelium, sometimes dilated by retained secretions, and in places entirely replaced by new connective tissue.

(*B*) (iii) **Primary chronic interstitial nephritis** (the *small red kidney*, the gouty kidney, the *granular kidney*).—This form of nephritis develops insidiously without any clinical evidence of inflammation of the kidney. The organ is small, hard, and

red; the capsule is abnormally adherent, and the surface left by
2335*A* its removal is rough and granular. It is embedded in a mass of perinephric fat which is often firmly adherent to the capsule. The granular projections upon the surface are of a somewhat lighter colour than the surrounding tissue and are formed by the surviving but degenerate parenchyma, while the depressions represent the contracted fibrous tissue. Cortical cysts are nearly always present, and are filled with urine or a colloid material. On section the cortex is greatly reduced
2335*A* in width, and is usually mottled in colour, red predominating; the pyramids are also reduced in size. The fat in the pelvis of the kidney is increased in quantity and encroaches on the
2335*C* kidney substance; the blood-vessels are enlarged and thickened, and gape on section.

Histologically the changes are chiefly in the direction of connective tissue overgrowth. The subsequent shrinkage of this new formation compresses the tubules and glomeruli, causing degeneration of the tubular epithelium and the conversion of the glomerular tufts into hyaline structureless masses.

The middle coat of the large arteries is enormously hypertrophied while the intima of the smaller vessels becomes hyaline and thickened.

**Sub-variety. Arteriosclerotic kidney.**—This name is given to a variety of "granular kidney" whose salient feature is a thickening of the renal arterioles. Otherwise there is no marked difference between this form and the last.

(*C*) **Suppurative nephritis** is invariably secondary to pyogenic processes elsewhere in the body, except in the rare cases when suppuration follows traumatism. The infecting agent may reach the kidney by two routes: (1) the blood stream, (2) the urinary passages (ascending nephritis: surgical kidney).

(1) In this, the **haematogenous** class, the suppuration depends upon septic embolisms occurring in the course of pyaemia, infective endocarditis, osteo-myelitis, scarlet fever, puerperal infection, and certain other conditions. Both kidneys are usually affected; they are enlarged, soft, and present upon the
2331$E_5$ surface yellowish-white projections, more or less numerous, each of which is a metastatic abscess. The abscesses are surrounded by a red areola, and adjacent haemorrhages are common. The cortex is the region most frequently affected, but areas of suppuration are also present in the medulla and pyramids.

(2) **Ascending nephritis: surgical kidney.**—When the infection ascends along the urinary passages, its source is usually a cystitis secondary to prostatic enlargement, urethral stricture, vesical calculus, or new growth of the bladder. The pelvis of the kidney is always affected, but the ureter may escape although the infective agent ascends along it. The lesion may affect one kidney only. From the pelvis of the kidney the inflammation extends to the apices of the pyramids, and ascends the tubules to the cortex, which as a rule is less 2338*C*
affected than the medulla. But the whole kidney is more or 2338*D*
less involved and exhibits a multitude of small abscesses. The 2338*E*
principal bacterium associated with these cases is the B. coli communis.

## VII. DEGENERATIONS.

(1) **Fatty degeneration** of the kidney occurs in the course of various diseases, especially chronic parenchymatous nephritis, profound wasting diseases like pulmonary tuberculosis, and acute infections. The macroscopic appearances depend on the extent and intensity of the fatty change. When this is local the kidneys present a mottled yellow and red appearance; when it is general they are uniformly white or yellow, 2353
enlarged, soft, and exhibit little or no distinction between cortex and medulla; the striations of the cortex disappear, and an oily exudation follows incision of the organ.

(2) **Amyloid degeneration** is found in the kidneys, usually in association with a similar change in the liver and spleen, in the course of syphilis or of prolonged suppuration; but it occurs independently in cases of nephritis of long standing, whether parenchymatous or interstitial. The amyloid kidney is enlarged, firm, usually pale-grey in colour, and, if the change is pronounced, may be dotted with translucent areas. The striations of the cortex are ill-marked. A watery 2331*C*
solution of iodine containing iodide of potash makes the amyloid deposits assume a mahogany-brown colour. Microscopically the glomerular capillaries are the first to suffer, their walls becoming infiltrated by hyaline deposits of amyloid material. At a later stage, the other blood-vessels and the walls of the tubules share in the infiltration. A tubular nephritis is a constant accompaniment, or possibly, precursor, of the amyloid change.

(3) **Cloudy swelling** of the kidney is the name given to a condition in which the structural markings are obscured, and the cut surface has an opaque appearance. It is met with as a result of infections of many kinds, and in association with poisoning by phosphorus, mineral acids, and a number of other substances.

Microscopically the cells of the tubules are swollen and, to some extent, desquamated. Their nuclei are stained with difficulty, and the protoplasm has a markedly granular appearance.

## VIII. TUBERCULOSIS.

Tuberculosis of the kidney is met with in two forms: (i) miliary, (ii) fibrocaseous.

(i) **Miliary tuberculosis** of the kidney is not of clinical importance. It is always secondary to the disease elsewhere in the body, and is due to infection by the blood-stream In consequence, the tubercles are found in greatest abundance in
2341*H* the cortical region. They appear as small grey or yellow dots, seldom numerous and generally surrounded by red areolae.

Microscopically the tubercle is somewhat less defined from the surrounding healthy tissue than the miliary tubercle in other organs, and the centre of the nodule is usually degenerated.

(ii) **Fibrocaseous tuberculosis.**—This variety forms a well-defined disease. The infection of the kidney is almost invariably secondary to a tuberculous focus situated somewhere outside the genito-urinary system. But it is believed that tubercle bacilli may occasionally reach the kidney by way of the urethra. The disease may seem, clinically, to be unilateral: but when one kidney is seriously damaged the other is seldom intact.

The lesions are usually in the early stages circumscribed and
2341*G* limited to one pole of the kidney, and consist of small caseous foci scattered through the cortex and pyramids. These coalesce and extend, and presently infect the pelvis of the kidney, whence the remaining calices are attacked. When the disease has advanced, there is usually but little healthy kidney substance remaining. The organ is enlarged, but retains its natural outline; on section the renal substance, with the exception of a narrow strip of cortex, is found to be destroyed and replaced by softened caseous masses,

which are separated from each other by the connective-tissue trabeculae of the gland. If the ureter is blocked by an extension of the disease to it, the whole organ may be converted into soft fluctuating rounded tumour containing a mixture of urine and caseous material and closely adherent to the perinephric fat (tuberculous pyonephrosis). When the inflammation becomes quiescent at this stage the capsule contracts upon the softened contents; the latter are gradually inspissated and infiltrated by limesalts, the whole organ being ultimately reduced to a cheesy or calcareous mass, weighing only a few grammes. 2340 2341 2341*C* 2358 2341*L*

In other instances the brunt of the disease falls upon the pelvis and the apices of the pyramids (tuberculous pyelitis), which are extensively ulcerated. The continual washing away of the softened tissue by the urine makes ulceration a predominating feature of this type. In consequence, haemorrhage from eroded vessels is a frequent event. 2341$A_1$ 2341*K* 2341*I*

The ureter, and eventually the bladder, are nearly always involved with the kidney. 2341*A* 2341$A_1$

## IX. SYPHILIS.

Syphilis of the kidney is rare, though the disease is one of the causes of amyloid degeneration in this organ. The following varieties are described:

(i) *Gummata*, occurring as grey nodules in the cortex or pyramids; in rare instances the kidney is the seat of diffuse gummatous infiltration. 2343*A*

(ii) A sclerosing *perinephritis* leading to atrophy of the kidney. 2331

(iii) Occasionally in young subjects the kidneys are the seat of extensive *cicatricial deformity due to an obliterating endarteritis* of the renal arterioles. This lesion is commonly regarded as "parasyphilitic"—a late manifestation of hereditary syphilis.

## X. URATIC DEPOSITS.

Uratic deposits are met with in gouty subjects, and occasionally in aged persons who are not gouty, in the form of white striae composed of urates, situated in the cortex and pyramids.

The so called *uric-acid infarcts* are golden striations of the

apices of the pyramids, found in the kidneys of new-born children, in older children who have died of acute diarrhoea, and occasionally in the kidneys of adults who have died of spleno-medullary leukaemia. They have no pathological significance.

## XI. NEPHROLITHIASIS.

Although precipitations of salts may occur in the substance itself of the kidney, as described in the preceding section, the term *nephrolithiasis* is generally reserved to express calculus-formation in the renal pelvis or calices. Chemically, renal calculi usually consist of the following substances, alone, or in various combinations. Uric acid, urates, calcium oxalate, and the phosphates of ammonio-magnesium and calcium.

(1) **Uric acid calculi.**—These often contain urates as well as uric acid. They form hard, smooth stones of a brownish-red colour. In size they vary from solitary crystals of uric acid,
2346*B* or small agglomerations of such crystals (*renal sand*), to large
2345 dendritic stones filling the pelvis and calices. The larger
2345*C* stones are generally stratified.

(2) **Calcium oxalate calculi** are dark and hard. Their surface is very irregular and covered with projecting nodules or
2358*C* spines. This variety forms the "*mulberry*" calculus. It is composed of crystals of calcium oxalate surrounding a nucleus of uric acid or urates.

(3) **Phosphatic calculi** imply a pathological alkalinity of the
Series of urine. In consequence a purely phosphatic renal stone is rare,
calculi. though a smooth white coating of phosphates may be deposited
212 upon either of the above-mentioned varieties of calculi when, as often happens, they have set up a pyelitis.

166-169 (4) **Rare calculi.**—Xanthin, cystin and indigo.

**Effects of renal calculi.** (*a*) **Within the kidney.**—(1) The irritation of a calculus may set up inflammation of the pelvis of
2345*B* the kidney (*pyelitis*). This inflammation is often suppurative.
2354*B* (2) When a calculus obstructs the orifice of the ureter
2357*A* the pelvis and calyces become distended with urine (*hydro-*
2358*C* *nephrosis*). The urine so accumulated is very liable to
2358*D* secondary infection and conversion into pus (*pyonephrosis*).

(3) *Haematuria.* Ulceration of the renal pelvis by a calculus may lead to the presence of blood in the urine.

(*b*) **Without the kidney.**—(1) The passage of a stone of any size along the ureter is attended by acute lumbar pain (*renal colic*). In its passage the stone may wound the ureter and lead to the appearance of blood in the urine; or it may become impacted at any point in the length of the ureter causing dilatation above the point of impaction, and *hydronephrosis*. The commonest sites of impaction are the two extremities of the ureter.

(2) A renal stone, or part of one, may reach the bladder 2354*B*
and cause *cystitis* or haematuria; or it may become impacted 2354*C*
in the urethra and cause obstruction, and even extravasation of urine.

## XII. PYELITIS.

This indicates an inflammation of the pelvis of the kidney. But it is seldom that the renal pelvis is affected to the exclusion of the neighbouring parts of the urinary tract. Thus in the case of ascending nephritis there is a pyelitis, but it is merely an incident in an infection of the whole urinary tract from the bladder upwards. There is one variety of lesion which may be said to belong to the renal pelvis in that it originates there, viz. calculous pyelitis. But even here the lesions are seldom limited to the pelvis. They are described under the heading *pyonephrosis*.

## XIII. HYDRONEPHROSIS.

This signifies an accumulation of urine in the renal pelvis, leading to dilatation of the main ducts of the kidney, generalised enlargement of the organ, and ultimately atrophy of its glandular tissue. It is due to some obstruction to the flow of urine. The chief causes of such obstruction are the following:

(1) Congenital stenosis or obliteration of the ureter. 2366

(2) Blocking of the ureter from within by calculi, tuber- 2362
culous deposits, or malignant growths. 2354*B*

(3) Kinking of the ureter when the kidney is abnormally movable.

(4) Pressure upon the ureter from without by abdominal and pelvic tumours, especially tumours of the female generative organs.

2369*A* (5) Urethral stricture, and the pressure of an enlarged
2361*B* prostate gland.
2370*B* (6) Extreme phimosis.

The hydronephrotic kidney is met with in all degrees of distension. It may reach an enormous size, especially when the obstruction causing it is intermittent. In such extreme cases the kidney is converted into a thin-walled sac, partially loculated on its inner aspect by the fibrous remains of the calices, and containing in its walls the merest remnants of renal parenchyma. The fluid contents are usually clear, and contain urea, uric acid, and a small quantity of albumin.

## XIV. PYONEPHROSIS.

Pyonephrosis signifies a dilatation of the kidney similar to that above described, but one in which the contained fluid is pus. As a general rule pyonephrosis is due to the infection by pyogenic organisms—notably the bacillus coli communis,—of a kidney already hydronephrotic. But occasionally, especially when a calculous pyelitis precedes occlusion of the ureter, the distending fluid is purulent from the commencement.

The progress of pyonephrosis may advance in one of two directions. (1) The inflammation may become quiescent, in which case the pus is slowly inspissated and converted into an accumulation of mortar-like consistency. (2) The abscess may
2355 point and discharge itself externally, or into a hollow viscus,
2351*A* or into the pleural cavity.

## XV. TUMOURS.

**Tumours of the kidney.**

(*a*) **Innocent tumours.**—(1) **Fibromata** and **adenomata** are occasionally met with as small, white, rounded tumours, the former situated in the pyramids, the latter in the cortex of the
2392$G_2$ organ.

**Microscopic examination.**—Renal adenomata exhibit two histological varieties. (*a*) Tubular adenomata consist of closely-packed tubules resembling in structure those of the normal kidney, but arranged in disorderly fashion; (*b*) papillary adenomata consist of branched and interlaced connective tissue processes covered with cubical or cylindrical epithelium.

Papillary adenomata, though sometimes innocent, commonly assume malignant characters, grow rapidly, and become the seat of gross haemorrhages or cystic degeneration. (*N.B.*— The term "adenoma," as used to describe these tumours which have become malignant, is a traditional misnomer.) 2392*G* 3392$G_1$ 2392*F*

(2) Encapsulated aberrant adrenal tissue—**adrenal "rests"**—occur in the cortex of the kidney, and may reach a considerable size. They are believed to be frequently the starting point of carcinomatous growths. 2390*G* 2390$G_1$ 2390$G_2$ 2390$H_2$

(3) **Cysts.**— (i) Small cortical retention cysts are common in chronic renal disease. 2335*G* 2336*A*

(ii) A single large cyst, or more than one, may be found in a kidney otherwise healthy. 2378$A_1$ 2378*C*

(iii) "*Congenital cystic disease of the kidney*" is the term applied to a condition in which the whole kidney is converted into a collection of cysts of various sizes. Commonly both kidneys are involved; they are greatly enlarged, and may weigh together several pounds. Little or no renal tissue may be visible to the naked eye, but remnants of it can always be found with the microscope. The cysts contain a clear, turbid, or blood-stained fluid. In a rarer type of the lesion one kidney only is affected, while the cystic change is much less pronounced. 2383 2383*A* 2379*B* 2379*D*

Microscopically the cysts are lined by a flattened epithelium.

Cystic kidneys when occurring as large tumours in the foetus are occasionally a cause of obstructed labour; but they seldom reach a large size at this early age. As a rule the enlargement is a slow process, but it may be rapid. The origin of the lesion is obscure; it has been variously attributed to a papillitis obstructing the outflow of the secretion of the tubules (Virchow); to a mal-development of the foetal kidney; and to a cysto-adenomatous new growth of the renal substance. In rare instances cystic kidneys are associated with a similar development of cysts in other organs, *e.g.* the liver, pancreas, and thyroid gland. 2385*A* Liver. 2204*D*

(*b*) **Malignant tumours.**—Both sarcomata and carcinomata occur in the kidney as primary or secondary tumours.

Primary sarcomata are: (1) round celled; (2) spindle celled; (3) adeno-sarcomata, *i.e.* connective tissue tumours, in which appear glandular columns and tubes; (4) rhabdo-myosarcomata, *i.e.* sarcomata which contain striped muscle fibres. 2391*C* 2391*E* 2391*K* 2391*F*

2392$H_1$ Primary carcinomata vary in type. There is a form which requires special notice. It is a yellowish growth, mottled with light chocolate-coloured areas of blood-extravasation,
2390$H_5$ usually encapsulated, but in places breaking through the capsule. It attains a very considerable size, and leads to wide dissemination of secondary growths in skin, mucous surfaces,
2390$G_2$ viscera, and muscles. This form of carcinoma is specially
2390$H_2$ malignant.

Microscopically its tissue resembles closely that of the cortex of the suprarenal body. These growths are believed to originate in the adrenal "rests" mentioned above.

## XVI. PARASITES.

2393$I$ Hydatid cysts are found in the kidney, though rarely.

Bilharzia haematobia (*v.* p. 34) is a small filiform worm
2393$D$ about $\frac{2}{3}$ of an inch long, found in Egypt and South Africa. It inhabits the portal vein and the veins of the genito-urinary system. The eggs, which are oval in shape with a sharp spike at one pole, find their way into the urine in large quantities,
2393$B$ and give rise to haematuria, pyelitis, and cystitis.

# CHAPTER XXVI.

## DISEASES OF THE NERVOUS SYSTEM.

I. Developmental Anomalies.—(1) Protrusions; (2) Macrencephaly; (3) Micrencephaly; (4) Anencephaly; (5) Porencephaly. II. Diseases of the Meninges.—(*A*) Pachymeningitis; (*B*) Leptomeningitis; (*C*) Meningeal Haemorrhage; (*D*) Chronic Diffuse Meningo-encephalitis; (*E*) Tumours of the Meninges. III. Diseases of Blood vessels of Central Nervous System.—(*A*) Arteries; (*B*) Veins and Sinuses. IV. Hydrocephalus. V. Cerebral and Cerebellar Abscess. VI. Tumours of the Brain. VII. Diseases of Spinal Meninges. VIII. Myelitis. IX. Limited Scleroses of the Spinal Cord. X. Syringomyelia. XI. Tumours of the Spinal Cord. XII. Nuclear Affections of the Cord. XIII. Diseases of Nerves.—(*A*) Neuritis; (*B*) Tumours of Nerves.

## I. DEVELOPMENTAL ANOMALIES OF THE MENINGES AND CENTRAL NERVOUS SYSTEM.

### 1. Protrusions.

(*A*) **Cranial**—(*a*) meningocele, (*b*) encephalocele, (*c*) hydrencephalocele.

(*B*) **Spinal**—(*a*) meningocele, (*b*) meningo-myelocele, (*c*) syringo-myelocele.

(*A*) **Cranial protrusions.**—Whatever their precise character, these tend to appear at one of two situations, the occipital and

the naso-frontal regions. Occipital tumours are the commonest, the aperture of communication with the cranial cavity being situated at the posterior fontanelle, or in the median line below it. Naso-frontal tumours appear at the root of the nose, slightly to one side or other of the middle line. The aperture of communication lies in this case between the cribriform plate
3472*B* of the ethmoid and the frontal bone.

(*a*) A **meningocele,** whether cranial or spinal, is a cystic tumour whose cavity communicates directly with the sub-arachnoid space. It therefore contains cerebrospinal fluid. The sac is composed from without inwards, of skin, dura mater, and arachnoid mater. The tumour is rounded, generally pedunculated, and may reach a large size.

(*b*) An **encephalocele** is a solid tumour containing brain-tissue with the meninges covering it.

(*c*) A **hydrencephalocele** is partly solid and partly cystic. The name implies that the central canal of the nervous system is prolonged into the tumour, thus providing a cystic centre occupied by cerebrospinal fluid. The lesion is generally associated with internal hydrocephalus, and probably repre-
3471*A* sents a pouching of the lateral ventricle under pressure of its
3472*A* fluid contents.

(*B*) **Spinal protrusions.**—(*a*) **Spinal meningoceles** do not differ in structure from the cranial type. They are met with most often in the cervical region and are not necessarily associated with "spina bifida" (*v.* infra) as are the other two
3481 varieties of spinal protrusion.

(*b*) A **meningo-myelocele** is the commonest variety of spinal protrusion. It forms a sessile cystic tumour in the lumbo-sacral region, and is invariably associated with a deficiency of the spinal arches in the affected district, a condition known as "*spina bifida.*" The sac is covered by skin at its periphery, but towards the centre the epidermal covering gives place to a thin membrane sometimes called the "central scar." The whole or a part of the spinal cord at the affected level passes into the sac, where it becomes flattened and incorporated with the wall. It may be traced to the central scar and disappears in it, occasionally however emerging from the lower extremity
3478 of the scar and pursuing its way back into the spinal canal.
3479 The tumour contains cerebrospinal fluid, its cavity being con-
3479*B* tinuous with the sub-arachnoid space.

(*c*) **Syringo-myelocele** is the spinal counterpart of hydrencephalocele, for it implies a tumour produced by fluid distension of the central canal of the spinal cord. The tumour is sessile and appears most frequently in the dorsi-lumbar region, and like hydrencephalocele is associated with hydrocephalus. The wall of the sac is composed in part of the stretched and flattened walls of the central canal, but in its general features the tumour resembles a meningo-myelocele. Various forms of club-foot are common accompaniments of meningo- and syringo-myelocele.

**Fate of the protrusions.**— Meningoceles (whether cranial or spinal) and encephaloceles may persist indefinitely without much alteration. But the remaining varieties are prone to undergo spontaneous rupture partly as a result of internal pressure, partly with the help of external ulceration, to which their imperfectly-developed coverings are very liable. The common result of such rupture is septic meningitis. 3479*A*

## 2. Macrencephaly.

Increase in the size of the brain, beyond the wide limits of normal variation, is a rare event. When it occurs the brain has a natural aspect apart from its bulk. The increase in size is due to a uniform enlargement of the gyri by an overgrowth of neuroglial tissue, giving to the brain an abnormally firm consistency. The skull, though enlarged, maintains a natural outline, thus contrasting with the globular shape of the skull produced by hydrocephalus. 2483*C*

## 3. Micrencephaly.

Abnormal smallness of the brain is much commoner than the foregoing. The diminution in size may be limited to certain areas of the brain or may affect the whole of it. Sometimes one hemisphere is undeveloped, while the remainder of the brain is approximately normal. Sometimes the convolutions may be perfect in form and distribution, but unduly small 2483*B*
(microgyria). Sometimes the gyri in addition to being small, are unusually arranged. Even when the whole brain is affected it is common to find the frontal convolutions more undeveloped than the remainder of the organ.

In rare cases a small brain may co-exist with a skull of natural dimensions, the interval between the dura mater and the skull being occupied by fluid (external hydrocephalus). As a rule the skull is developed proportionately to the brain, and misshapen like it. The commonest deformity is a very retreating forehead.

Both macrencephaly and micrencephaly, if at all extreme are associated with mental deficiency.

### 4. Anencephaly.

3459 Absence of the brain is common among foetal monstrosities.
3466*A* It is often associated with absence of the heart.

### 5. Porencephaly.

This term has a variety of meanings. Strictly it denotes a defect in the brain-substance as a result of which the lateral ventricle is brought into direct communication with the sub-arachnoid space. Where the brain substance is deficient the
2483*D* pia-arachnoid is thickened and often contains cystic spaces.
2483*E* This lesion is commonly situated in the neighbourhood of the Sylvian fissure, and is one of those associated with spastic diplegia in children.

In a looser sense porencephalus signifies a hollowing out of the brain by extensions of one or other lateral ventricle which nevertheless do not reach the surface. In such cases the brain may appear normal externally, or may exhibit an area of thinning of the cortex, with some flattening of the gyri and meningeal adhesion. Lesions of the above two varieties are generally attributed to foetal haemorrhages or encephalitis. In a sense still more remote from its original meaning porencephalus is sometimes used to indicate acquired deficiencies of brain substance, particularly a cystic condition of the cortex resulting from haemorrhages in the past.

## II. DISEASES OF THE MENINGES.

(*A*) Of the Dura Mater, Pachymeningitis.
(*B*) Of the Pia-arachnoid, Leptomeningitis.

(*A*) **Pachymeningitis.**—(*a*) External, (*b*) internal.

(*a*) **External pachymeningitis.**—The outer aspect of the

dura shares in all inflammations of the bone enclosing it, the commonest being the tuberculous, gummatous, and septic varieties.

The *tuberculous* form generally attacks the spinal dura mater, owing to the frequency with which the spine is the seat of tuberculous caries. The result is a thickening of the membrane and an accumulation of caseous material between it and the bone. Pressure of such caseous masses upon the spinal cord plays a large part in the pressure-paresis of the 2534
lower extremities which is so common a feature of spinal 2535
caries. 2458*B*

The *gummatous* form generally attacks the vertical dura mater from a gummatous focus in the vault of the skull. The lesion conforms to the ordinary type of gummatous inflammation (see p. 21). 2457*A*

The *septic* variety is in a majority of cases an associate of suppurative otitis media, the infection traversing the petrous portion of the temporal bone and involving the dura mater covering it. The inflammation in this case may merely excite a thickening and injection of the dura, or may proceed to pus-formation. In the latter event an *extra-dural abscess* appears between the bone and the dura mater. Such abscesses are localised and single, and are often complicated by an infective thrombosis of the lateral sinus owing to its proximity at this point to the cavity of the middle ear.

(*b*) **Internal pachymeningitis.**—The internal aspect of the dura may be involved in any of inflammations attacking its 2458
outer aspect, and often shares in those of the pia-arachnoid 2458*A*
beneath it. But with one exception the lesions of internal pachymeningitis are not individualised. This exception is haemorrhagic pachymeningitis.

**Haemorrhagic pachymeningitis** attends virulent infections, being then merely a local expression of the general tendency to extravasations of blood. It occurs also in many ill-defined marasmic states during early life. But among the insane it is 2447
especially common. 2447*A*

In a typical example the inner aspect of the dura is of a bright red colour, and has the appearance of a vascular membrane. Sometimes the extravasation is gross, forming a 2449*B*
visible layer of clot on section of the membrane, or even 2449*C*
penetrating its inner surface and becoming diffused upon the 2449*D*

2450 pia-arachnoid beneath. At other times the lesion is limited to splashes of extravasation, scattered at random about the dura mater, but purely interstitial. A condition probably to be considered as a late stage in the course of haemorrhagic pachymeningitis is the cystic formation within the layers of the
2449*E* dura mater, which is met with among the insane.

The spinal dura mater, especially its cervical portion, may also be the seat of haemorrhagic pachymeningitis.

(*B*) **Leptomeningitis.**—Inflammation of the pia-arachnoid may be excited by a great variety of organisms. Of these the tubercle bacillus, the diplococcus intra-cellularis meningitidis (commonly called the meningococcus), and the pneumococcus, produce more or less specialised lesions. The pyogenic infections are described under the title "septic meningitis."

*Note.*—The term "cerebrospinal meningitis" has long been reserved to signify the disease produced by the meningococcus, especially the adult form known also as "epidemic cerebrospinal fever" or "spotted fever." But the monopoly is not deserved, for diffuse meningitis of whatever kind generally affects both the cerebral and the spinal meninges, and is thus a "cerebrospinal meningitis." It would be better, therefore, if the latter phrase were to be discarded from clinical nomenclature and reserved to express only the pathological fact it implies. The lesions produced by the meningococcus will be found described under the title "meningococcal meningitis."

*Varieties of Leptomeningitis.*

(1) Tuberculous meningitis.
(2) Meningococcal meningitis.
(3) Pneumococcal meningitis.
(4) Septic meningitis.

(1) **Tuberculous meningitis.**—Tuberculous meningitis is always secondary, and generally occurs as a part of a tuberculous septicaemia. The primary focus from which diffusion of the infection takes place may be situated anywhere, but lies for the most part in the lungs or, during childhood particularly, in bronchial or mesenteric lymphatic glands which have become caseous.

The typical meningeal lesion is a miliary tuberculosis affecting

*the basal region of the brain by preference.* Here the convolutions are flattened; the pia-arachnoid is thickened, adherent both to the brain and dura mater, and rendered opaque by inflammatory exudates. Close inspection shows an abundance of minute white, grey, or yellowish projections from the pia-arachnoid. These are miliary tubercles. They are found in the greatest plenty upon the walls of the Sylvian fissures and upon the meningeal sheaths of the minute arteries passing to the anterior perforated space, but are by no means limited to this distribution. The brain is often unnaturally diffluent owing to co-existent encephalitis, and the cerebrospinal fluid increased in quantity, producing internal hydrocephalus. 2458*C* 2458*D* 2458*E*

As a rare event other regions than the base may show the most advanced lesions, for example the vertex or the median aspects of the hemispheres. When this is the case the lesions commonly show signs of chronicity which are absent from the ordinary type, the tubercles being larger and often definitely caseous.

The histological features of tuberculous lesions are described on page 19.

(2) **Meningococcal meningitis.**—Syn.: Epidemic cerebrospinal meningitis (adult form). Posterior basal meningitis (infantile form).

This is the only common form of primary meningitis. The organism responsible for it is now generally known as the meningococcus. It is a diplococcus resembling in shape the two halves of a chestnut with the flat sides in apposition, and is decolourised by Gram's method of staining. From the fact that in film-preparations the organism is often seen within the pus-cells it is also known as the *diplococcus intra-cellularis meningitidis.*

Although the clinical features of the adult and the infantile forms present many features of contrast, the meningeal lesions of the two are identical. *The inflammation is pre-eminently basal*, and often, in a chronic case, defined by an oval area limited in front by the level of the optic chiasma and behind by the posterior extremity of the cerebellum. Sometimes purulent deposits are also found near the anterior extremities of the middle fossa. The meninges of the spinal cord are invariably involved. In the oval district above mentioned is a purulent meningitis. Both here and throughout the spinal 2453*B*

canal the sub-arachnoid space is distended by yellow pus, while the ventricular fluid is increased in quantity and turbid even to the point of being purulent to the eye.

In fulminating examples of the disease, when death has anticipated the appearance of an inflammatory reaction, there is no evidence of meningitis, but a haemorrhagic condition of
2470*C* the membranes associated with similar extravasations elsewhere. This haemorrhagic tendency is a notable feature of the adult form, and has resulted in the name "spotted fever."

When the disease is chronic the purulent exudate becomes inspissated and gradually absorbed, leaving the damaged membranes thickened and opaque. A common sequel in this case is progressive internal hydrocephalus, usually credited to blocking of the foramen of Majendie in the roof of the fourth ventricle by post-inflammatory sclerosis; but thrombosis of emissary veins like those of Galen probably plays an important part in the production of this form of hydrocephalus.

(3) **Pneumococcal meningitis.**—The pneumococcus can produce a purulent meningitis, either as a sequel of fibrinous pneumonia or in association with a similar inflammation of one or more of the other serous membranes. The lesion is a purulent exudation into the sub-arachnoid space, and in a typical case *affects the vertex rather than the base* of the brain, giving the former the appearance of having been bathed in greenish-yellow pus. The ventricular fluid is increased in
2453$A_2$ quantity; and turbid from the presence of pus-cells and
2453$A_3$ pneumococci.

(4) "**Septic**" **meningitis.**—Besides the meningococcus and the pneumococcus already mentioned, a variety of organisms is from time to time concerned with the production of "septic" or suppurative meningitis, especially the streptococcus pyogenes, the staphylococcus pyogenes aureus and the staphylococcus pyogenes albus. The chief associations of septic meningitis are the following:

(*a*) Perforating wounds and ulcers of the skull.

(*b*) A septic condition of the frontal sinus or of the middle ear. *Of all the causes of septic meningitis, suppurative otitis media is by far the commonest.*

(*c*) Cellulitis of the face or scalp, particularly that excited by carbuncles in these situations.

(*d*) The terminal phases of grave constitutional disorders

like chronic nephritis. This is the only variety in which the meningeal lesion has the appearance of being primary. When it occurs it is common to find a similar inflammation of the pericardium or pleurae.

(*e*) Rupture of a cerebral abscess into the sub-arachnoid or sub-dural spaces.

(*f*) Scarlet fever, and occasionally influenza and typhoid fever.

(*g*) Septic infarction of the brain, the result of infective endocarditis of the left chambers or valves of the heart.

(*h*) Acute infections of distant parts, such as acute infective periostitis and osteomyelitis, and pyaemic conditions generally.

Septic meningitis may be localised or diffuse. In the former case the brain or spinal cord in the neighbourhood of the infecting focus is covered by a layer of pus lying in the sub-arachnoid space, while the adjacent cerebral veins and sinuses are often the seat of a purulent thrombosis which fills them with blood stained pus. The same description holds good for the diffuse variety, but the suppurative thrombosis of the veins is here less advanced owing to the rapidity with which the disease progresses to a fatal issue. 2532 2453*A* 2453$A_1$

(*C*) **Meningeal haemorrhage.**—Haemorrhagic pachymeningitis is described on p. 235. Two other varieties of haemorrhagic lesion require to be mentioned: (1) traumatic, (2) infective.

(1) **Traumatic meningeal haemorrhage.**—This is a common complication of severe blows upon the skull, both with and without fracture. The usual source of the haemorrhage is a rupture of the middle meningeal artery within its groove upon the inner aspect of the temporal bone, resulting in an effusion of blood between the dura mater and the skull. 2446 2446*B* 2446*C*

(2) **Infective meningeal haemorrhage.**—The pia-arachnoid, both cranial and spinal, is liable to share in the generalised haemorrhages which attend fulminating infections. 2470*A* 2470*C* 2532*A*

(*D*) **Chronic diffuse meningo-encephalitis.**—This is the lesion responsible for general paralysis of the insane. Its causation is not fully known, but there is little doubt that the infection of syphilis plays an essential part either directly or indirectly in the production of the disease.

The dura mater is thickened and may be the seat of haemorrhagic pachymeningitis. The pia-arachnoid is also diffusely 2447

2511*B* thickened and unnaturally adherent to the cortex cerebri, while the brain itself is reduced in size by shrinkage of its convolutions. The frontal region commonly suffers most.

**Microscopic examination** shows an infiltration of the pia mater and of the perivascular tissues by round cells: also a neuroglial hyperplasia with atrophy and degeneration of the nervous elements. The lymph-channels are packed with leucocytes and the arteries universally thickened in all their coats.

### (*E*) Tumours of the meninges.

(1) Infective granulomata.—(*a*) Tuberculous masses; (*b*) Gummata.
(2) Calcareous deposits.
(3) Cysts.
(4) Innocent tumours.
(5) Malignant tumours.—(*a*) Carcinoma; (*b*) Sarcoma; (*c*) Endothelioma; (*d*) Cholesteatoma.

2456*A* 2458 2458*A* (1) **Infective granulomata.**—Both tuberculous masses and gummata form yellowish tumours projecting from the inner surface of the dura mater. Distinction between them cannot be established with certainty except by microscopic examination (*v.* pp. 19 and 21).

(2) **Calcareous deposits** occur on the inner aspect of the dura mater, particularly about the falx cerebri. These 2463 2464 deposits may produce definite tumours or diffuse calcareous plaques. Their pathology is unknown.

(3) **Cysts** of the membranes are generally either parasitic or the result of antecedent haemorrhages. The latter variety is described in the sections upon haemorrhagic pachymeningitis and porencephalus the former among the tumours of the brain. In addition, a cystic condition of the choroid 2511*B* plexus is occasionally met with. It has no definite associa- 2511*C* tions.

2465 2465*A* (4) **Innocent tumours** are rare, but fibromata are occasionally met with on the inner aspect of the dura mater.

(5) **Malignant tumours.**—(*a*) **Carcinoma** of the membranes is always secondary, and even then is generally due to direct invasion from a secondary deposit in the bones of the skull.

(*b*) **Sarcoma** of the meninges may be primary or secondary.

2465*C* **Primary sarcoma.**—All the histological varieties except the

melanotic may occur as primary growths of the dura mater, 2465*D*
infiltrating both the bone above and the brain beneath them. 2466*A*

**Secondary sarcoma.**—All varieties of sarcoma, without exception, may attack the membranes as secondary growths. But there is one variety requiring special description, namely *chloroma*, or green sarcoma. The tumour originates in the orbit and gives rise to extensive secondary deposits in the dura 2468*C*
mater. These tend to spread along the lines of the cranial 2468*D*
sutures, which are seen, after removal of the skull-cap, to be outlined by continuous stripes of greenish growth. On the inner aspect of the dura the invasion is more diffuse, but definite tumours may form in this situation, infiltrating the pia-arachnoid and brain beneath them.

Histologically chloroma is a round-celled sarcoma.

(*c*) **Endothelioma.**—This type of tumour is of little or no 2465*B*
malignancy and of slow growth. A form peculiar to the dura 2466$B_1$
mater is the *Psammoma*, which contains foci of calcification. 2466$B_2$

(*d*) **Cholesteatoma.**—This is a tumour of little or no malignancy, found chiefly in the pia mater or brain. It is characterised by a pearly or satin-like appearance and consists of flat cells, concentrically arranged and sometimes enclosing cholesterin. Its precise place in the catalogue of tumours is uncertain. Ziegler classes it with dermoid tumours. It is certainly of epidermal origin.[1] 2501*C*

## III. DISEASES OF THE BLOOD-VESSELS OF THE CENTRAL NERVOUS SYSTEM.

A. Arteries. B. Veins and Sinuses.

(*A*) **Arteries.**—(1) Atheroma. (2) Gummatous arteritis. *Results of arterial disease*: (*a*) thrombosis; (*b*) haemorrhage. (3) Embolism.

(*B*) **Veins and sinuses.**—Thrombosis: (1) simple; (2) septic.

(*A*) **Diseases of arteries.** (1) **Atheroma.**—Whenever atheroma is at all generalised the cerebral arteries are certain to

[1] The so-called cholesteatomata found in the external auditory meatus belong to a different category and consist of heaped up keratinised epidermal cells.

be affected. The most advanced changes are usually to be found in the branches of the middle cerebral artery, but may occur anywhere. The lesions include proliferative endarteritis, atheromatous plaques aud ulcers, calcification and aneurysm-formation. In a well-marked example the larger arteries appear as opaque rigid tubes often exhibiting minute dilatations—the so-called "miliary aneurysms." The smaller
2474$B_1$ branches are mottled with opaque patches and may also be the seat of aneurysms.

The histological changes produced by atheroma will be found described on p. 79.

(2) **Gummatous arteritis.**—During the tertiary stage of syphilis the cerebral arteries may be the seat of gummatous infiltrations, the result being localised thickenings of the arterial coats. This variety of syphilitic arteritis is not to be confounded with the diffuse form of proliferative endarteritis which, though often attacking syphilitic subjects, is not by any means peculiar to them, and is justly included among the lesions of atheroma.

**Microscopic examination** shews that gummatous arteritis commences in the tunica adventitia as a small-celled infiltration prone to undergo hyaline degeneration. In course of time the tunica intima is invaded, while the subendothelial tissues of the tunica intima proliferate and obstruct the arterial channel at the seat of damage.

**Results of arterial disease.**—The effects of arterial disease in the brain, as elsewhere, are determined by two factors, viz. the roughening of the channel and the weakening of the tube. The prime result of the former is *arterial thrombosis* on the roughened site, while the latter leads to localised protrusions of the damaged wall—*aneurysms*.

(1) **Cerebral thrombosis.**—The cerebral arterioles being poorly supplied with anastomoses, the obstruction of one of them by a clot produces an anaemic necrosis of the area robbed of its blood-supply, an event similar in mechanism to anaemic infarctions of the spleen or kidney. But whereas in the latter situations the infarcted area never softens unless the embolus is infective, in the brain subsequent softening is the rule, even when the thrombosis is simple. So constant is this that the result of cerebral thrombosis is spoken of as "cerebral softening." In most cases the affected area is converted into a pulpy semi-liquid mass of a yellowish colour (yellow softening);

but occasionally blood is extravasated into the necrotic district after the fashion of a haemorrhagic infarct, the condition being then termed "red softening." 2474*E*

(2) **Cerebral aneurysms.**—The importance of cerebral aneurysms lies in their tendency to undergo rupture. The result of such rupture is a gross extravasation of blood into the brain-tissue—"**cerebral haemorrhage.**" Extravasations of this kind may occur anywhere, but the seats of election are three.

(*a*) *By far the commonest situation for a cerebral haemorrhage is the region of the internal capsule at its anterior extremity*, the artery at fault being one of the lenticulo-striate branches of the middle cerebral arteries. The consequence is an effusion of blood at a point where a large number of fibres from the motor cortex converge to traverse the internal capsule. The laceration and destruction of tissue which ensues commonly involves all the motor fibres of the affected hemisphere, producing hemiplegia 2474$A_1$ of the opposite side. If the extravasation be copious it may 2474$A_2$ erode the wall of the ventricle and fill its cavity with blood— 2474$B_1$ *ventricular haemorrhage.* 2476

(*b*) Rupture may occur in the pons Varolii—*pontine haemor-* 2481*B* *rhage*, or, 2481*C*

(*c*) In the cerebellum—*cerebellar haemorrhage.* Spinal haemorrhages are very rare.

(3) **Cerebral embolism.**—Cerebral emboli are derived from the left side of the heart. They consist of detached fragments either of antemortem thrombi formed within the cardiac chambers – a common incident of mitral stenosis – or of vegetations upon the valvular or mural endocardium, the result of infective endocarditis.

The general effect of an embolism is identical with that of thrombosis, namely cerebral softening. But if the embolus be derived from an infective vegetation the process becomes a 2470*C* purulent infarction and the infarcted area is converted into pus. 2470*D* At times the rapid disintegration of a septic embolus results in 2474*C* erosion of the artery and subsequent haemorrhage—pyaemic 2474*D* cerebral haemorrhage. 2474*F*

(*B*) **Thrombosis of the cerebral veins and sinuses.**—Thrombosis may be primary or secondary.

(*a*) Primary thrombosis is rare. It occurs in infants dying of wasting diseases; in anaemic conditions, for instance, severe

chlorosis; in the terminal stages of cachexias; and as a sequel to scarlet fever, typhoid, or influenza.

(*b*) Secondary thrombosis is more common. The chief causes are *disease of the middle ear*, fracture of the skull, compression of the sinuses by new growths or aneurysms, and suppurative diseases of the face and skull. It is also seen in patients who die of acute infections, for instance, pneumonia.

Whether the thrombosis is simple or suppurative depends upon the primary lesion; if this is suppurative, as is the case with middle-ear disease, the clot is purulent; if the thrombosis on the other hand is caused by pressure, or occurs in the course of a tuberculous meningitis, the clot is not purulent, but fibrinous.

The sinus most often involved is the lateral, and since the primary lesion is usually suppuration connected with the middle ear, the thrombus is usually, though not always, infected, and becomes purulent. Non-purulent thrombosis is most common in the longitudinal sinus and its tributaries, and
2470*B* in the cavernous sinus.

## IV. HYDROCEPHALUS.

(*A*) External. (*B*) Internal.

(*A*) **External hydrocephalus** signifies an accumulation of serous fluid between the dura mater and the arachnoid. It is a rare condition occasionally attending atrophy of the brain, pachymeningitis, and meningeal haemorrhage.

(*B*) **Internal hydrocephalus**, an excessive accumulation of serous fluid within the cavities of the brain, is common. It presents two varieties: (1) acute; (2) chronic.

(1) **Acute internal hydrocephalus.**—Some increase in the quantity of cerebrospinal fluid, with corresponding dilatation of the cerebral chambers, is an incident of all forms of acute meningitis; but the excess is seldom gross. The fluid is generally turbid to a varying degree owing to the presence of pus-cells and micro-organisms. The term "acute hydrocephalus" used to be employed as a synonym for tuberculous meningitis, but the latter disease enjoys no monopoly in the production of the lesion.

(2) **Chronic internal hydrocephalus** may be congenital or acquired.

The *congenital form* is sometimes due to occlusion of the foramen of Majendie, presumably as a result of foetal meningitis of some kind. At other times the aqueduct of Sylvius is found to be obliterated. The distension of the ventricles is extreme, and is attended by a corresponding enlargement of the skull. The bones of the vault are widely separated and the head assumes a globular shape, while the veins of the scalp are engorged and prominent. As the ventricular fluid increases in bulk the septum lucidum gives way, thus converting the two lateral ventricles into one large cavity. The brain-tissues covering the hemispheres are stretched and attenuated and in an advanced case become a mere membrane enclosing the accumulated fluid. The latter may amount to as much as five pints, and has the characters of cerebrospinal fluid. 2516*E* 2514 2516*A* 2521*C*

The *acquired form* occurs as a sequel of basal meningitis, particularly the meningococcal variety, the foramen of Majendie having been obliterated by adhesions. It may also depend upon occlusion of the central canal, especially the aqueduct of Sylvius, by new growths, tuberculous masses or gummata. The resultant lesions are not to be distinguished from those of the primary variety if the patient be an infant. But at a later age distension of the skull may be precluded by ossification of its sutures, in which case internal hydrocephalus, however extreme, produces no alteration in the outline of the skull. 2453*B* 2616*D*

## V. CEREBRAL AND CEREBELLAR ABSCESS.

Cerebral abscesses are generally secondary to an obvious lesion elsewhere, but at times no antecedent can be detected. If these apparently primary examples of the disease be grouped together cerebral abscesses fall into three main categories, as follows:

(1) Those due to suppurative otitis media.
(2) Those due to septic infarction of a cerebral vessel.
(3) Those of unknown origin.

(1) **Suppurative otitis media is by far the commonest cause of cerebral abscess.**—The abscess is usually solitary and of considerable size. It lies most often in the temporo-sphenoidal lobe, the cerebellum taking second place. Septic meningitis in the neighbourhood of the inflamed petrous bone may be present to mark the path of the infection, but the latter is capable of travelling by the lymphatic channels without infecting them. Abscesses of this class are sub-acute or chronic, and are lined by a pyogenic membrane enclosing greenish pus. The infection is a mixed one. Rupture of such an abscess is an occasional cause of diffuse septic meningitis.

2486*E* 2486*E*$_1$ 2486*G* 2486*G*$_1$

(2) Septic infarctions of the brain, consequent upon infective endocarditis of the left side of the heart or septic conditions of the lung, particularly bronchiectasis, become disintegrated and converted into abscesses. These abscesses are usually multiple and not encapsulated. Their contents comprise necrotic brain-tissue and pus, and are often rendered reddish-brown in colour by altered blood.

(3) Large solitary abscesses are occasionally found for which no explanation is forthcoming. They are chronic, and lined by a pyogenic membrane.

2484*A* 2485*A*

## VI. TUMOURS OF THE BRAIN.

(1) Infective granulomata.—(*a*) Tuberculous masses, (*b*) gummata.

(2) Cysts.—(*a*) Post-haemorrhagic, (*b*) parasitic—(1) hydatid, (2) cysticercus cellulosae.

(3) Simple new growths.—Glioma.

(4) Malignant new growths.—(*a*) Glio-sarcoma, (*b*) sarcoma, (*c*) carcinoma.

(5) Tumours of the cerebral appendages.—(*a*) Pineal gland, (*b*) pituitary body.

### 1. Infective Granulomata.

(*a*) **Tuberculous masses.**—These are generally multiple and originate either in the meninges or in the adventitial coats of arteries. They occur most often in the cerebellum and about the region of the pons Varolii, but may be met with anywhere. On section they have a smooth, homogeneous surface and are

2488 2491 2492*A*

of a greenish colour. Unlike caseous accumulations elsewhere they seldom become liquefied. The microscopic characters of a caseous tubercle are given on p. 19.

(*b*) **Gummata** of the brain may be single or multiple and vary in size, sometimes assuming minute proportions, at others reaching the size of a pigeon's egg. They are situated most often on the surface of the hemispheres and may be either circumscribed or diffuse. The commonest seat of origin is the sub-arachnoid space, the brain becoming subsequently involved. To the naked eye gummata are greyish-pink in colour and upon section yellow and caseous towards the centre. 2490

## 2. Cysts.

(*a*) **Post-haemorrhagic.**—As in the membranes, so in the brain a cyst may remain as the legacy of a haemorrhage, the wall of the cyst being generally stained by altered blood-pigments.

(*b*) **Parasitic.**

(1) *Hydatid Cysts*, representing the cystic stage in the development of Taenia echinococcus, may occur in the brain. The life-history of this parasite and an account of the cysts will 2507
be found in Chap. I. 2508

(2) *Cysticercus cellulosae.*—This, the cystic, larval form of Taenia solium, the pork tapeworm, may occur in the meninges and cortex. The life-history of the worm is given in Chap. I.

## 3. Innocent Tumours.

**Innocent tumours** of the brain, as opposed to the membranes, are very rare, with the exception of the pure *gliomata*. The latter are connective tissue tumours analogous to fibromata in other parts of the body. They may originate in any part of the brain and form soft pinkish encapsulated masses. Gliomata, however, generally contain sarcomatous elements which then exclude them from the class of innocent tumours, though gliosarcomata are often loosely described as gliomata.

Histologically the pure gliomata reproduce the characters of neuroglial tissue. They consist of a loose reticulum scantily supplied with neuroglial cells.

## 4. Malignant Tumours.

**Malignant tumours** of the brain may be (*a*) primary, (*b*) secondary.

(*a*) **Primary.**—*The commonest primary malignant tumour of the brain is the glio-sarcoma.* This is a sarcoma arising in connection with the neuroglia. It is found most frequently in the cerebral and cerebellar hemispheres, but may occur in any
2497*C* part of the central nervous system. It appears as a grey
2497*C*$_1$ or whitish growth, ill-defined and exhibiting the soft con-
2497*D* sistency of the normal brain. It gives rise to little gross deformity as a rule, but some bulging of the brain in the neighbourhood can generally be detected. Tumours of this kind are liable to the following changes :

(1) Mucoid degeneration, giving the mass a gelatinous semi-
2497*C* transparent aspect.

(2) Extravasation of blood. Glio-sarcomata are richly supplied with blood-vessels. In consequence haemorrhage frequently transforms them into bright or dark red areas, or
2497*C* streaks them with red splashes and dots. The extravasation
2497*D* of blood is occasionally so gross as to leave no trace of the original tumour.

2497*B* (3) Cystic degeneration.

Histologically glio-sarcomata consist of branched neuroglia-cells, round connective-tissue cells representing the sarcomatous element, and numerous thin-walled blood-vessels.

(i) **Sarcomata** other than glio-sarcomata may occur in the brain, arising in connection with the pia mater and the adventitial coats of cerebral arteries. They are more defined than the glio-sarcomata, round or oval in shape, and irregular
2500*B* upon the surface. They are very liable to extravasations of
2500*E* blood, the latter sometimes escaping from the confines of the tumour and producing an area of brownish softening in the
2500*D* neighbouring brain-tissue.

Histologically these sarcomata may be round-celled, spindle-celled or mixed-celled.

(ii) **Primary carcinoma** of the brain is unknown except in connection with the choroid plexus.

(*b*) **Secondary malignant tumours of the brain.**—All
2502 varieties of malignant tumour, carcinoma, sarcoma, and

syncytioma malignum, may give rise to metastases in the brain. 2499*C* 2499*E*

Histologically these secondary deposits conform to the type of the original tumour.

### 5. Tumours of the Cerebral Appendages.

(*a*) **The pineal gland.**—Simple cysts of this gland, reaching the size of a cherry, are occasionally met with. They appear to have no pathological significance. 2512*A*

(*b*) **The pituitary body.**—Adenomata of this organ bear a fairly constant relation to *acromegaly*. They appear as solid tumours, showing no tendency to infiltrate the tissues in their neighbourhood, but, by the pressure they exert, indenting both the sella turcica and the brain above them. In rare cases a tumour associated with acromegaly may be sarcomatous; still more rarely tumours are found in this situation in patients who have shown no signs of acromegaly. 2504*B* 2504*C* 2504*D* 2504*E*

## VII. DISEASES OF THE SPINAL MENINGES.

Most of the common forms of spinal meningitis have been described in the section devoted to diseases of the meninges, for the spinal membranes share to some extent most diffuse forms of meningitis; but they may undergo local lesions as a result of extension of inflammations from neighbouring foci of disease. In all acute inflammations of the meninges the fluid in the sub-arachnoid space is increased in quantity and becomes turbid and cellular. Of the cells contained in the cerebrospinal fluid under such circumstances it may be said that when the infection is tuberculous lymphocytes preponderate. Most other infection produces a poly-morphonuclear leucocytosis.

## VIII. MYELITIS.

(*A*) *Acute*: (1) diffuse, (2) transverse. (*B*) *Chronic*.

(*A*) (1) **Acute diffuse myelitis** is a rare disease. The term indicates a spreading or widely disseminated inflammation of the substance of the spinal cord. The causes of this condition are but vaguely known, though a bacterial origin has been demonstrated in some cases and is presumed in most.

The affected regions of the cord are injected and unnaturally soft, while the distinction between white and grey matter is
2543 lost.

(2) **Acute transverse myelitis** results from the extension into the cord of an inflammation already existing in some contiguous structure, e.g. spinal caries; from the local injury
2547*A* attending fracture of the spine; or from the pressure of
2547*B* tumours situated within the spinal canal.

Microscopically acute myelitis presents the following characters. The vessels are congested and their lymphatic sheaths filled with red and white cells. The nerve-cells become swollen and subseqently disappear. The axis-cylinders are irregularly swollen, while their myelin sheaths break up into fatty droplets.

(*B*) **Chronic myelitis** is for the most part a sequel of the acute form, but may appear insidiously without passing through any such obvious phase of activity. Chronic transverse myelitis is the only variety which needs mention here, for the chronic diffuse form, whatever its origin, becomes indistinguishable from disseminated sclerosis, to be presently described.

Chronic transverse myelitis results in a hardening of the
2544 substance of the cord at the level of the lesion. This is due
2545 to an excess of neuroglial elements, which give to the white areas of the cord a pinkish-grey appearance resembling that of healthy grey matter.

**Microscopical examination** shows neuroglial overgrowth and atrophy of nerve fibres.

## IX. LIMITED SCLEROSES OF THE SPINAL CORD.

(1) Disseminated sclerosis.
(2) Tabes dorsalis (posterior columns).
(3) Lateral sclerosis (lateral columns).
(4) Combined scleroses.

(1) **Disseminated sclerosis.**—Although this disease is usually described among the diseases of the spinal cord its lesions appear in the brain as well. Practically nothing is known of its aetiology. It results in the appearance of grey translucent islets of sclerosis scattered at random throughout the central nervous system. Both the grey matter and the white matter are affected, and there seems to be no selection of site with the

exception that the cerebellum is less affected than the rest of the central nervous system.

**Microscopic examination** shows that the myelin sheaths of the nerve fibres in the affected districts are atrophied, while the nerve-cells and axis-cylinders escape. There is also a marked proliferation of neuroglia.

(2) **Tabes dorsalis.**—Naked eye examination of the spinal cord in this disease shows a thickening of the pia-arachnoid over the posterior surface of the cord, with atrophy of the posterior columns and posterior nerve-roots. On section the atrophied columns are grey or greyish-red in colour, and contrast strongly with the white appearance of the antero-lateral columns. The degeneration is usually more obvious in the lumbo-sacral region than elsewhere.

The subjects of this disease are liable to the following gross lesions:

(i) A painless destructive arthritis. Charcot's disease (*v.* Diseases of Joints).

(ii) Atrophy of the optic nerves. 2552

(iii) Perforating ulcers of the sole of the foot, leading to destruction of bone. 3233*C*

**Microscopic examination** of the cord at different levels shows an ascending degeneration of the fibres derived from the posterior root. In the lower parts of the cord the degenerated fibres run for the most part in the column of Burdach, but at a higher level the column of Goll is also degenerated.

(3) **Sclerosis of the lateral columns** is the lesion which produces the symptoms comprehended under the term "spastic paraplegia." Clinically the condition may be primary, or secondary to a known lesion such as caries of, or injury to, the spine, disseminated sclerosis, tumour, or general paralysis of the insane. Authorities differ as to the cases which appear to be primary and limited to the lateral columns, some avouching the possibility of such a limited primary sclerosis of the lateral columns, others denying it. There is, however, a consensus of opinion that if such a condition exists it is very rare, and that as a rule the lesion is not limited to the crossed pyramidal tracts.

(4) **Combined scleroses.**—There are some well-defined conditions in which sclerosis of the lateral columns is combined with degeneration of other parts of the cord. Thus:

*Amyotrophic lateral sclerosis* indicates a combination of

lateral sclerosis with an atrophy of anterior-horn cells, which results in muscular wasting.

*Ataxic paraplegia* indicates a combination of lateral sclerosis with sclerosis of the posterior columns such as is seen in tabes dorsalis.

Subject to minute differences the lesions of *Friedreich's ataxia* are similar to those of ataxic paraplegia, namely, a sclerosis of the lateral and of the posterior columns.

## X. SYRINGOMYELIA.

The cardinal lesion of syringomyelia is an overgrowth of neuroglia in the central parts of the cord, and the presence of a longitudinal cavity within the overgrowth. The regions of the cord most often affected are the lower cervical and upper dorsal. Here the cord is enlarged, but the membranes covering it are normal. On section a cavity is exposed, lying either in the situation of the central canal or behind it, in which case the two cavities may communicate or may be independent. The abnormal cavity is filled with a clear serum, and varies much in length and size and shape. The pathology of the disease is very obscure. Some authorities maintain that it depends upon a developmental anomaly; others that it represents a diffuse gliomatosis of the central parts of the cord, the cavity being formed by the liquefaction
2542 of the overgrowth in its deeper parts.

**Microscopic examination** shows that the cavity is limited by a thick, dense wall of neuroglia not lined by epithelium.

In one variety of the disease the hands and feet are liable to
3233$C_1$ be attacked by painless whitlows and trophic ulcers (Morvan's disease).

## XI. TUMOURS OF THE SPINAL CORD.

Tumours of the spinal cord and its membranes are rare.

| | | |
|---|---|---|
| Of spinal cord. | Sarcoma. | **2541***a*. |
| | Glio-sarcoma. | **2541***b*. |
| Of membranes. | Endothelioma. | **2541***c*. |
| | Carcinoma. | **2541.** |
| | Tuberculous mass. | **2534. 2535.** |

## XII. NUCLEAR AFFECTIONS OF THE CORD.

### (1) Acute Anterior Poliomyelitis. (2) Chronic Anterior Poliomyelitis.

(1) **Acute anterior poliomyelitis.**—Although the causation of this disease is uncertain, there is a good deal of evidence that it is an infective process. Thus it has a seasonal variation of frequency, the hottest months producing the majority of examples of it. The disease also occasionally occurs in epidemic form, and commonly has an acute onset, accompanied by fever and very suggestive of an acute infectious process. The morbid anatomy, when the lesion is recent, is as follows. There is some injection of the pia-arachnoid. On section of the cord the neighbourhood of the anterior-horn cells in the affected district of the cord is unnaturally red, swollen, and soft. A similar lesion may attack the ganglion cells of the motor cortex (acute polio-encephalitis).

**Microscopic examination** shows vascular engorgement, leucocytic infiltration, and degeneration of ganglion cells in the anterior horn. At a later stage the congestion and cellular infiltration disappear, leaving a neuroglial sclerosis of the damaged area.

(2) **Chronic anterior poliomyelitis.**—This disease has no acute phase, but appears to be a primary degeneration of the anterior-horn cells. The cervical enlargement is most affected. To the naked eye the only noticeable change is the small size of the anterior roots in this area.

**Microscopic examination** shows wasting of the grey matter of the anterior horns, with shrinkage and atrophy of the large ganglion cells within it.

As has been observed above, this lesion may be associated with degeneration of the lateral columns in the disease known as "amyotrophic lateral sclerosis."

## XIII. DISEASES OF NERVES.

### (*A*) Neuritis.

Although in medical nomenclature the termination "itis" implies an inflammatory process, yet the lesions embraced under the name "neuritis" include many which seem to be pure degenerations of the nervous parenchyma, unattended by the vascular congestion and cellular infiltration which mark

ordinary inflammatory processes. In many examples of neuritis both parenchymatous degeneration and interstitial inflammation are present in varying proportions, the former being often secondary to the latter. As regards these proportions it may be said that local neuritis—that is, a lesion limited to one nerve or part of it—depends chiefly upon an interstitial inflammation, while multiple neuritis exhibits a preponderance of degenerative changes in the nerve fibres themselves.

**Local neuritis.**—This lesion may depend upon an obvious local cause, such as the extension of inflammation from an
2566*A* adjacent focus, injury, or pressure; or upon vaguer circumstances, such as cold and wet acting upon rheumatic or gouty subjects. The most typical example of local neuritis is neuritis of the sciatic nerve, the commonest cause of sciatica. In this condition the nerve trunk is injected and swollen.

**Microscopic examination** shows interstitial exudation and leucocytic infiltration. In severe and protracted cases there is in addition degeneration of the nerve fibres to a varying extent.

**Multiple neuritis.**—Multiple neuritis is due to a poison in the circulating blood. This poison may be any one of a great variety, but the commonest actually encountered are the following:

**Metallic.**—Lead, arsenic.

**Organic.**—Alcohol.

**Microbic.**—The toxins of diphtheria, influenza, typhoid fever, and beri-beri.

**Metabolic.**—The poisons of diabetes mellitus.

The lesions of multiple neuritis are generally symmetrical. Although the stress of the mischief may fall upon the peripheral nervous system, the central nervous system often suffers as well, witness the encepalopathy which may attend lead-palsy.

Certain of the poisons above mentioned exhibit a selective action.

**Lead.**—Lead affects the nerves of the upper extremity by preference, and particularly the musculo-spiral nerve and its branches. It attacks the motor fibres in the mixed nerve-trunks, but leaves the sensory fibres intact.

**Arsenic** usually involves the nerves of the lower extremities, as does **alcohol.**

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