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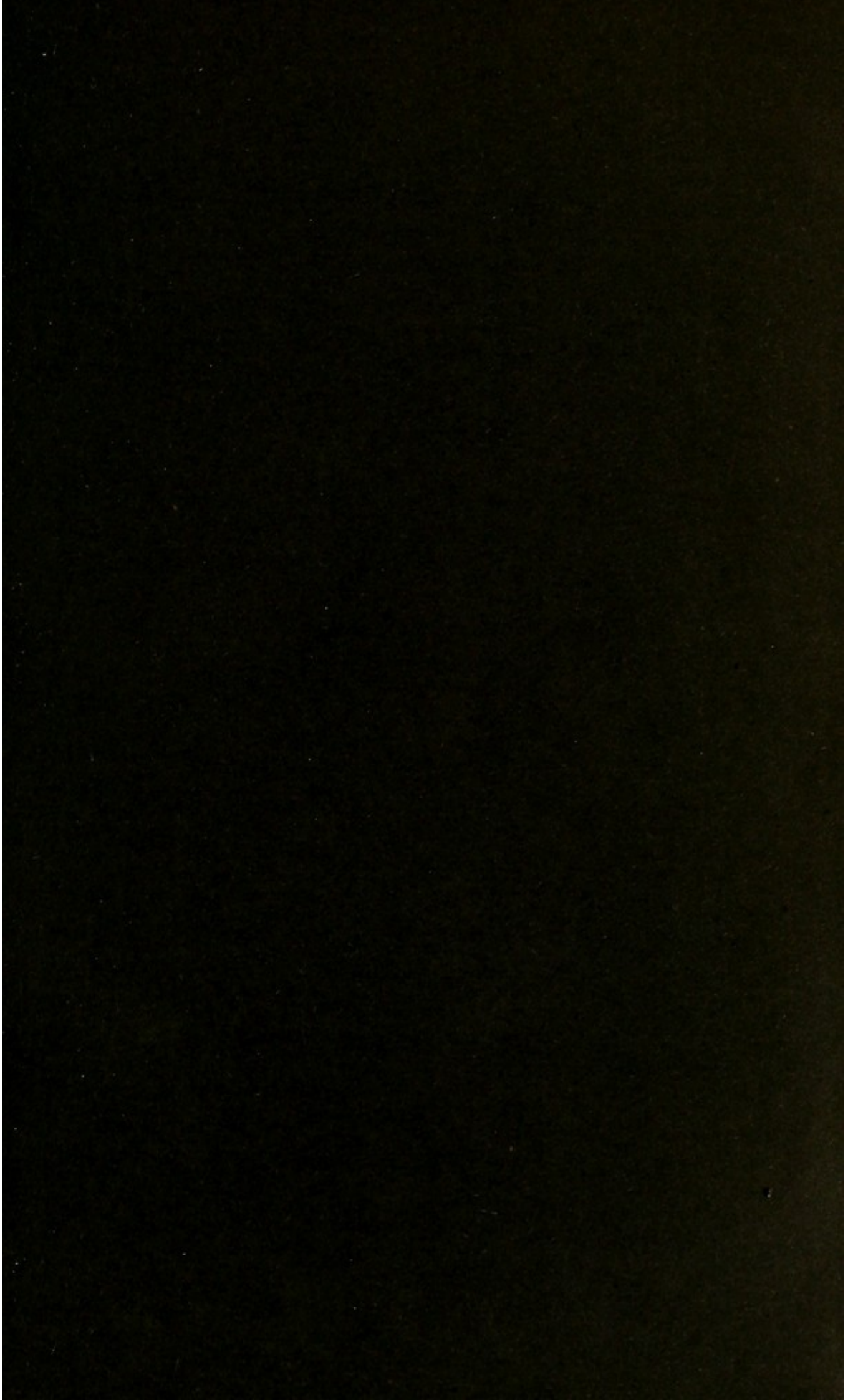

SURGICAL PATHOLOGY AND PRINCIPLES.

COLUMBIA UNIVERSITY

SURGICAL PATHOLOGY

AND PRINCIPLES

BY

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WITH ONE HUNDRED AND NINETY-FOUR ILLUSTRATIONS

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To

SIR WILLIAM HENRY BROADBENT, M.D., F.R.S.,

BARONET,

PHYSICIAN-IN-ORDINARY TO H.R.H. THE PRINCE OF WALES, AND
CONSULTING PHYSICIAN TO ST. MARY'S HOSPITAL,

THIS BOOK IS DEDICATED

AS A TOKEN OF GRATITUDE FOR HIS TEACHING AND FOR
MANY ACTS OF PERSONAL KINDNESS.

APR 9 " 1906 Dr V. O. Vaughan, U.

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PREFACE.

THE following pages are based on the surgical portion of a course of demonstrations given annually by me during the past seven years in the museum of St. Mary's Hospital. The arrangement of the matter is, to a considerable extent, influenced by experience gained in re-arranging and describing the collection of pathological specimens in that museum. It has been my endeavour in this book, as it was in my oral teaching, to put the material in as objective a form as possible, and to lay before the student a plan of the whole basis of surgery so arranged that he might readily be able to fill in further details from clinical experience and teaching, and from study. Since the work is intended chiefly for students, the pathology of common affections has been given more fully than that of rarer conditions; at the same time, in order to justify the existence of the book, it has been necessary to give something more than a bare description of appearances that can be studied in mortuary and museum. The only justification that need be urged in favour of the separate existence of works on surgical pathology as distinguished from pathology at large, is that in such works alone can the subject be dealt with so as to make the applications of pathological knowledge to surgical practice clear and prominent. Wherever I have ventured to deviate from the usual method of treating any topic, the step has been directed by a long experience of teaching. Thus, in the description of healing of wounds, the terms usually employed are modified, because I have found that the student is confused by reading of one class of wounds as "healing by granulation," when all injuries involving connec-

tive tissue are repaired by the formation of "granulation-tissue." It seemed to me that the phenomena of repair of wounds might be better explained by speaking of "normal repair" as equivalent to the process observed in aseptic wounds whether closed by sutures or open, as contrasted with conditions in which repair is delayed or prevented by suppuration, leucocythæmia, etc. Again, in dealing with intestinal obstruction, some text-books limit this term to the various forms of mechanical obstruction that occur within the abdominal cavity as opposed to external hernia. I have found that students, having once received the idea that there is something antithetical between the two conditions, are rendered for a long time insensible to the practical identity of the symptoms they occasion. On this ground strangulated hernia has been given as the type of acute intestinal obstruction, and for similar reasons acute peritonitis has been included among the causes of obstruction.

Many of the original illustrations are taken from drawings made in the post-mortem room, and used to illustrate my demonstrations in the museum, to which some of the specimens were subsequently added. Where numbers are added to the descriptions of figures, they refer to the numbers of the specimens of the printed catalogues. Some of the illustrations are taken from older specimens in the collection, and I have the pleasure of thanking Mr. G. P. Field, Dean of the Medical School, for permission to make drawings of these specimens for this book. I have also to thank Mr. Bland Sutton for the loan of one figure from his work on *Tumours*, and Mr. Roughton for two figures from his papers on *Oral Surgery*. Messrs. Longmans have kindly allowed me to borrow several illustrations from Prof. Coats's *Manual of Pathology*, Prof. Weichselbaum's *Pathological Histology*, and Mr. Warrington Haward's *Orthopædic Surgery*. Four illustrations are taken from Prof. Zuckerkandl's work on the *Anatomy of the Nasal Fossæ*.

9 Old Cavendish Street,
London, *September*, 1897.

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INTRODUCTION.

PATHOLOGY is defined as the science which treats of disease. The subject is thus a very wide one, and it may be considered desirable that some indication of the scope of this short treatise should be given. Among the sources of knowledge of any disease, three are of special importance. First, the group of phenomena presented by the patient during the continuance of the disease. These are due to alterations of nutrition, of physiological function, or of anatomical form and structure, and constitute the ground of clinical study of disease. Second, anatomical, chemical, and other changes produced in the tissues by the morbid process. These are studied as morbid anatomy, etc. Third, information elicited as to the immediate and remote causes of the disease, or, in other words, the Etiology (Gr. *aitia* = cause), which includes studies as far apart as bacteriology and the theory of evolution. Of these three sources the first is doubtless the most important. By losing sight of the fact that the knowledge gained by clinical study forms part of pathology, many have come to have a false idea of the meaning of the word, and to regard pathology as the art of making preparations for the microscope, or of cultivating bacteria; at the same time, they fail to recognize that they themselves are occupied in studying the most important (*i.e.* the clinical) part of pathology, and applying their pathological knowledge to the relief of suffering. But both the knowledge and its application must in many cases be imperfect, if other sources of information, such as morbid anatomy and bacteriology, are neglected. Malformations such as hare-lip present for treat-

ment little beyond simple anatomical abnormality, and though the general pathology of such conditions includes a consideration of their causation, histological or bacteriological investigation is unnecessary to their being understood. If a surgeon is responsible to society chiefly for the rectification of such anatomical irregularities, he may come to think too lightly of pathology. This feeling arises from the fact that the conditions with which he has to deal have a pathology that is obvious. Whilst clinical workers are apt to underrate the value of the other departments of pathology, a similar error is made by some who are occupied in histological and bacteriological research, who lose touch with clinical work, with the result that their efforts become aimless or misdirected. Thus experimental investigations require for their direction the experience of the sick-room.

In the contemplation and in the study of disease the normal physiological processes of the body must ever be present to the mind of the observer, for pathology is nothing more than physiology disturbed in one way or another. Having thus attempted to show what in the present volume is implied by the term "pathology," it becomes incumbent on the author to state what he intends by the title "Surgical Pathology." This may be taken to be equivalent to "The Pathology of Surgical Diseases." Even when so stated the scope of the term is hardly defined, since in different minds different interpretations of the word "surgical" will arise. Etymologically (Gr. *cheir* = hand; *ergon* = work) it signifies such affections as require manipulative or operative treatment. The German word for surgeon signifies wound-physician, and expresses the truth that the surgeon does not constitute a species distinct from the physician, but is only a variety of the latter. "Operative medicine" is a term used by some French writers to designate surgery. This term is a suggestive one, but it leaves out of view the fact that in some instances the highest surgical skill consists in abstaining from operation. At the present day, when the departmentation of medical practice is becoming more and more marked, there is especial call for emphasizing the unity of medicine. The physician should possess a sound knowledge of the principles which guide the surgeon, and conversely a surgeon should be a

physician in potential. A case of typhoid fever may be taken as an example of an affection which comes within the domain of the physician. Such a case may rapidly enter a surgical phase from perforation of the intestine or of the gall-bladder, or from sloughing of the mucous membrane of the larynx, and the physician must be able to recognize the surgical development at its very commencement. On the other hand, it may be as long as a year or more after the cessation of the general symptoms of an attack of typhoid that a swelling in the periosteum of one of the bones for the first time seriously attracts the patient's attention. Such a condition will probably come to the notice of the surgeon, and from the inflammatory character of the swelling he might be tempted to cut into it, a course which would not be taken if the nature of the case were recognized, because with rest and the administration of iodide of potassium the swelling will probably disappear.

In the present work the more important of the conditions that come to the notice of the surgeon will be considered. First, when necessary, an outline of what may be termed their natural history, that is, their average clinical course and natural termination; then their naked-eye and minute anatomy, and, where it exists, their bacteriology will be given, in so far as they bear on diagnosis, prognosis, and treatment.

The material is best arranged in two sections. Firstly, considerations of nutrition, infection, etc., which apply to the body generally; and secondly, a more detailed description of the pathology of special organs. The division of this work into parts has been to some extent deviated from in order that, by using the effects of injury of various structures as examples of the processes of repair, the real continuity that exists in the *Principles of Surgery* might be the better expressed.

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PART I.

GENERAL SURGICAL PATHOLOGY.

CHAPTER I.

GENERAL CONSIDERATIONS.

In the life-history of a complex organism distinct periods are to be recognized. First, a period of *development* in which the original fertilized ovum-cell by repeated subdivisions becomes converted into a cell-aggregate, and then definite re-arrangement of cells and the formation from them of differentiated tissues, such as fibres, muscles, nerves, etc., occur and result in the evolution of co-related organs until the structure of the organism is perfected. Second, a period of *growth* of the completed organism in which the organs of the body increase in size until the adult stage is reached and is maintained by healthy nutrition. Finally, a period of *degeneration*, in which different parts of the body undergo degenerative changes. In old age the skin becomes thin, wrinkled, and altered in colour from pigmentation, the bones tend to become more porous and hence more friable, the muscles, including the heart, and the organs generally diminish in size. The highest of all the organs, the brain, is the last to undergo marked senile changes, so that in normal conditions happy and useful life may be prolonged to advanced age. These periods are not absolutely separate in time, but to a certain extent overlap each other. The period of development may be taken as a repetition of the stages of evolution of higher or more complex organisms from simpler forms. At each successive

stage of evolution healthful growth and nutrition are requisite, in order for the next stage in the process of development to be completed. Pathological processes may occur at any period of life. During the evolution of the body malformations may result from either excess or defect of development, and certain diseases may be contracted by the embryo from the parents.

Nutrition. Healthy nutrition of the whole or any part of the body depends upon several factors, among which are: (1) A healthy state of the cells and tissues and a normal structure of the body or part; (2) a regular supply of blood of proper quality and in sufficient quantity; (3) a healthy condition of the nerves; (4) a proper formation of certain "internal secretions,"[1] such as that of the thyroid gland.

Without going too far into the consideration of questions that belong to general pathology, it will not be out of place to notice a few of the more common ways in which the factors that influence nutrition may be disturbed.

The cells of the body or of a part may be deficient in vitality from inherited defect or disease. In congenital syphilis an *infection* is transmitted from the parents to the fœtus. In many families an inadequacy of the cells of the body to resist the attacks of hostile organisms, such as the tubercle bacillus, is observed. Thus there may be a *hereditary* presence of or tendency to certain diseases.

The necessity for a proper circulation of blood to different parts of the body hardly needs emphasis. If the blood flow is completely arrested for sufficient length of time in any considerable part of the body the cells of the part perish, in other words, necrosis (Gr. *necros* = corpse) or gangrene (Gr. *gangraina* = eating sore) ensues.

The influence of a healthy state of the nerves in maintaining the nutrition of the body or of any part of it is very great. If a mixed nerve trunk is completely divided the muscle fibres and the skin and other tissues which it supplies undergo atrophy or other changes of nutrition. If the nerve trunk is the seat of chronic inflammation, as in certain cases of leprosy, the changes

[1] This term was introduced by Schäfer, see *British Medical Journal*, 1895, p. 341, Vol. II.

in the skin may be very pronounced. The importance of the central nervous structures in maintaining a healthy condition of the body may be illustrated by quoting as an example the hyperpyrexia which is observed in cases of injury to the dorsal and cervical part of the spinal cord and in many cases of cerebral hæmorrhage. Slighter deviations of the state of the nerves from the normal physiological state help to determine conditions such as chilblains.

Changes observed in portions of the body owing to altered innervation are partly direct, or due to the "trophic" influence of the nerves on the cells with which their terminal filaments are in close relationship; and partly indirect, *i.e.* due to alteration in the circulation owing to impairment of the regulating function of vaso-motor nerves, or to the absence of sensation in a part leading to increased exposure to injuries of various kinds.

Internal Secretions. The influence of ductless glands on the general nutrition of the body is best known in the case of the thyroid gland. After complete removal of all the thyroid tissue, either by operation or disease; anæmia, changes in the skin, and in the functions of the brain, are always observed. These symptoms disappear in a measure if either fresh thyroid gland, or an extract made from it, is administered to the patient. The constituent of the thyroid which has such a marked effect on nutrition is termed its internal secretion. The changes that occur in females after the removal of both ovaries, and in males after removal of both testes, may reasonably be attributed to the loss of the internal secretions of these glands.

Atrophy. (Gr. *a* = not; *trepho* = I nourish.) Atrophy of any part or organ of the body strictly signifies a diminution in size owing to lessened nutritive activity of its component elements. In surgery the term is frequently used to designate shrinkage of tissue from other causes, especially cicatricial shrinking.

In general emaciation, old age, certain mental states, etc., the tissues of the body generally tend to undergo atrophy, the chief causes of which are: (1) Deficient supply of blood; (2) prolonged disuse; and (3) certain nerve-lesions.

Thus the muscles of a limb, the use of which is restricted

by reason of the disease of a joint, become flabby or wasted. This is due in part to *disuse*, and is so constant a sequence of joint-disease that it becomes a valuable aid in distinguishing between real joint-disease and the simulation of joint-disease observed in some neurotic or so-called hysterical subjects.

Muscles, after loss of their nerve-supply, whether from destruction of the ganglion cells in the anterior cornua of the spinal cord, or loss of function of the nerve trunks whether from injury or disease, undergo atrophy. This may be accompanied by the deposit of fat in the connective-tissue cells between the muscular fibres, so that although atrophied the muscle may appear to be enlarged, *e.g.* as in pseudo-hypertrophic paralysis. On atrophy degeneration often supervenes; thus part of the protoplasm of the muscle fibres may become converted into fat (fatty degeneration), or pigment granules (pigmentary atrophy).

Long continued pressure produces atrophy chiefly by diminishing the blood supply.

Diminutive size of a part due to imperfect development may be distinguished from atrophy by using the term hypoplasia (Gr. *hypo* = under, *plasso* = I form), to designate a condition of original undersizedness, and "aplasia," to designate the total absence of a part from non-development.

Hypertrophy. (Gr. *hyper* = over, *trepho* = I nourish.) Hypertrophy means overgrowth of a part, and may result from increase in size of the individual cells or other elements of a part, or from a numerical increase in the cells. To the former kind of enlargement some writers would limit the term hypertrophy, using the word hyperplasia to designate cases of numerical increase.

The normal hypertrophy of the uterus during pregnancy, and that of muscles from healthful labour, are instances of natural hypertrophy.

The chief causes of hypertrophy are:

1. Increased labour.
2. Increased blood supply.

The first cause is operative in the production of *compensatory* hypertrophy. Thus if one kidney is removed the other, if healthy, will enlarge to nearly twice its original size. The

second determines overgrowth in many different conditions. Thus sometimes a bone which is the seat of chronic congestion from the presence within it of some source of irritation, *e.g.* a sequestrum, may become curiously elongated and thickened. Under this head, too, come cases of hypertrophy from friction or intermittent pressure. It will be observed that these causes act together in the production of hypertrophy. Thus increased use of a muscle causes an increased blood supply. If, however, the increase of function is excessive, leading to fatigue, the opposite result ensues—exhaustion of nerve-centres, producing atrophy of muscles, as in severe cases of writer's cramp and other spasmodic neuroses.

The Effects of Pressure. It has been observed above that in current surgical phraseology the terms hypertrophy and atrophy are frequently employed where strictly the words hyperplasia and cicatrization are more applicable. Thus the surgical aphorism—"Intermittent pressure causes hypertrophy, continuous pressure causes atrophy"—refers to processes which entail more than mere alterations in the nutrition of the cells of the part. As an example, that very common pathological condition—a corn—may be taken. The greater part of the hypertrophy or tissue-increase consists in an increase in the number of strata in the horny layer of the epidermis; but the mucous layer of the epidermis and the papillary layer of the corium also become thickened, or hyperplastic, from increase in the cells and other elements of the tissue. See Fig. 1, p. 10.

Beneath the corn the papillary layer of the corium becomes thinned after a time, and the subcutaneous fat undergoes more or less complete atrophy. The corn affords an illustration of both parts of the aphorism just quoted. A little reflection shows that both the increase and the loss of tissue are attributable to changes in the circulation. Friction and intermittent pressure produce the well-known effect of increasing the amount of blood circulating in a part, and improving the nutrition of its constituent elements. The production of a callosity, whether on a workman's hand from intermittent pressure of a tool or on the foot from pressure against the boot at each step, is the result of increased nutrition brought about by frequently repeated

periods of hyperæmia. The first stage of a corn is a *callosity* which serves the useful purpose of increasing the resisting power of the skin. The callosity is thickest at its centre, *i.e.* the point of the greatest friction. In the feet this will usually be over the heads of the metatarsal bones, on the outer surface of the little toe, the upper surface of the other toes, or some other part exposed to pressure and friction. The central part of the thickening is pressed from the outer side by the resistance of the boot, and it is prevented from expanding laterally by the

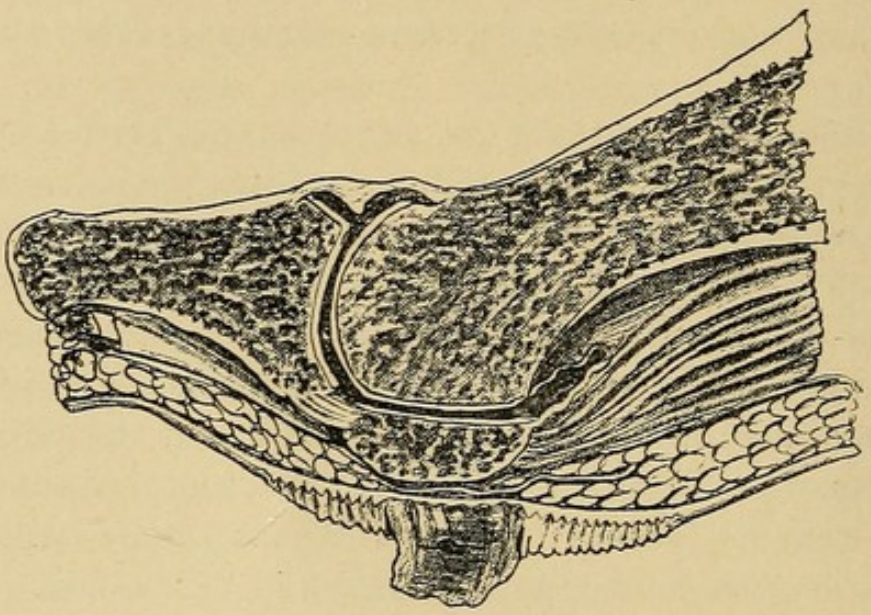


FIG. 1.—Vertical section through the first metatarso-phalangeal joint and the soft tissues beneath it. The skin bears a large corn. For a certain distance beyond the latter the epidermis is thickened and the ridges of the corium are increased in size, whilst immediately beneath the corn the ridges are flattened, and more deeply still the subcutaneous fat has entirely disappeared, leaving a small nerve almost unprotected between the corn and the sesamoid bone.

horny layer of the adjoining epidermis. This results in two events: firstly, the middle of the thickest part of the callosity becomes condensed, and secondly, its under surface becomes convex. It is now a typical corn, resembling, as its Latin name *clavus* suggests, a nail driven into the skin. In order to enable the corn to assume this convexity on its deep aspect certain atrophic changes must take place in the tissues beneath it. These atrophic changes are attributable to diminution of the blood in the parts of the corium and subcutaneous tissue which lie immediately beneath the corn, and hence are exposed to more continuous pressure. The fat-cells, deprived of adequate

blood supply, probably consume each its own store of fat, reverting to the type of the connective-tissue cell. The papillary ridges of the corium become flattened, first from compression of capillaries and lymphatic vessels, and afterwards, in large corns, from actual absorption of tissue.

These phenomena, *viz.* tissue-increase from intermittent, tissue-decrease from constant, pressure, are repeated in a variety of pathological conditions. Even bone may be extensively eroded and removed by constant pressure, *e.g.* that of an aneurysm. The bearing of the structure of a corn upon the symptoms to which it gives rise is easily traced. In the earlier stages removal of the cause, *i.e.* the pressure, is followed by disappearance of the lesion. The disappearance of more or less of the corium and of the subcutaneous fat leaves the nerves unprotected and exposed to pressure between the hard corn and the bone. Even when there is no active pressure, the resistant character of the corn causes the nerves to feel even slight variations in the amount of blood present in the part. Thus when the barometric pressure falls, the blood- and lymph-capillaries dilate from increase in the amount of blood in the part, and hence the increase in the tension on the nerves, and the pain. Such attacks of hyperæmia readily pass into inflammation.

The near kinship of pathological processes, which for convenience are classed under different designations, is also exemplified in the case of a corn. Had the initial friction and pressure which started the formation of the callosity been more pronounced, the condition would have become an inflammatory one and the increase of tissue would have been termed an inflammatory hyperplasia. Again, if the tissue-growth had been of more marked degree the term new-growth would have been applicable, and the condition would have been termed a papillomatous horn. Thus hypertrophy, inflammatory hyperplasia, and new-growth may represent different degrees of the same histological processes.

Gangrene. As has been mentioned above, extreme disturbance of nutrition results in death. Separate portions of the body may perish without death of the individual. When the life of relatively large portions of the body ceases, the process is termed gangrene; if death occurs in smaller portions of the body, different

terms are employed. Thus death in a portion of bone is termed necrosis; a small dead piece of soft tissue, such as skin, is termed a slough. When, as in a spreading sore, minute portions of tissue, too small to be separately recognized, undergo necrosis, the term ulceration is used. Among the causes of gangrene the most important are:

1. Arrest of the flow of blood through a part, as from the blocking of an artery, whether by embolism, atheroma, or other agency, or the anæmia produced by extreme pressure.
2. Extremes of temperature, as in frost-bite, scalds, and burns.
3. The action of caustic chemical agents.
4. Infections, such as the bacillus of emphysematous gangrene.
5. Disturbances of the vaso-motor nerves, as in symmetrical gangrene (Raynaud's disease) and in chronic ergot poisoning.
6. Nerve-lesions, as in acute bed-sore following fracture of the spine.

Gangrene from the Blocking of Arteries. This may occur in healthy persons from injury, as in the instance of subcutaneous laceration from which the artery represented in Fig. 2 was taken.

A portion of clot dislodged from the heart or elsewhere may lodge in and block an artery. If a large vessel, such as the common femoral, is occluded, or if the arteries are extensively affected by atheroma or calcification, the collateral circulation may fail to be completely established, and the distal part of the member below the seat of occlusion of the artery becomes gangrenous. In certain organs, such as the brain, kidney, and spleen, the occlusion of even small arteries leads to necrosis in the form of softening, infarction, etc. This occurs because in these organs the terminal arteries anastomose only by capillary branches which are incapable of forming a collateral circulation. When gangrene occurs in, say, a foot, from embolism, it is to be remembered that the embolus may be lodged as high as the external iliac artery, the remainder of the limb retaining its vitality through the collateral circulation. If

the seat of embolism can be ascertained by the finger there may be some tenderness observed. *Embolic gangrene* usually develops suddenly, and the limb becomes cold and numb. There may be agonizing pain, which is followed by loss of sensibility in the part. The skin changes in colour, becoming bluish, red, purple, and finally black. Where the dead joins the living tissue a red zone forms from inflammation which results in ulceration or the formation of the "line of demarcation," processes which will be explained in a later chapter. The general symptoms are fever followed by depression. The disease may prove fatal from shock or septic poisoning. Other causes of arterial obstruction which may be sufficient to occasion gangrene are atheroma and primary calcareous degeneration. These changes are more common in the aged, hence the term *senile gangrene* has been applied to a group of cases of gangrene.

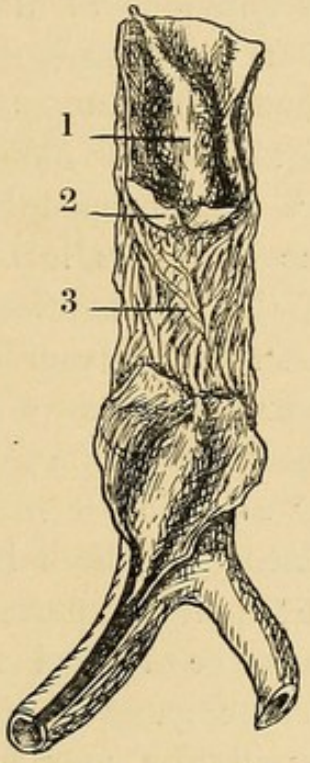


FIG. 2.—The lower part of a popliteal artery cut open and showing the inner and middle coats (2) torn through transversely and retracted, leaving about ¾ of an inch of the outer coat (3) exposed. Coagula, as at 1, occupy the lumen of the vessel above and below the seat of the injury. From a man over whose popliteal space passed the wheel of a cart weighing a ton. For an hour or so after the accident pulsation was felt in the tibial arteries at the ankle, but this soon ceased and gangrene set in, necessitating amputation (693). Nat. size.

Dry and Moist Gangrene. The appearances of a gangrenous part vary. If the tissues perish without there having been previous inflammatory changes the dead parts dry by evaporation and assume a black and shrivelled or, as it is termed, a *mummified* appearance. On the other hand, it frequently happens, that the failing circulation leads to malnutrition, and this to œdema and inflammation before actual gangrene ensues,—in such cases the tissues are soaked in fluid which undergoes decomposition, the epidermis becomes detached from liquefaction, and serum may collect under it, forming large blebs, which differ from inflammatory blisters in that the fluid they contain can by slight pressure be made to move in any direction beneath the detached epidermis.

Diabetic Gangrene. If gangrene occurs without obvious cause in a young subject, the first thing to be done is to examine the urine for sugar.[1] It has been shown that the production of gangrene in diabetics is similar to that of senile gangrene, that is to say, it is due to atheroma which, owing to the general disease, may develop at an earlier age than usual. The fact that the blood and tissues in cases of diabetic gangrene are handicapped by the excess of sugar they contain renders the prognosis less favourable than the relative youth of some of the patients would otherwise warrant.

In senile gangrene the indications for treatment are, firstly, general—to keep up the strength of the patient, to diminish shock by opium; and, secondly, local—to keep the limb warm by swathing it in cotton wool, and to help the circulation by placing the limb in the horizontal position; antiseptics should be applied to the dead parts. The question of amputation must be decided from the conditions met with in each case. Where gangrene follows ligature or other injury of an artery amputation may be performed as soon as there is unmistakable evidence of gangrene having occurred.

Burns and Scalds. The effects of heat on the tissues necessarily vary according to the degree of heat applied and the time during which it acts. There may result nothing more than a hyperæmia without any destruction of tissue, or the exposure may have been sufficient to cause a superficial inflammation of the skin (dermatitis), marked by the formation of blisters between the layers of cells of the epidermis; or again, the whole of the epidermis and part of the corium may be destroyed, and so on in successive degrees up to deep charing of the muscles and bones. When heat is suddenly applied to the tissues in such a way as to cause immediate death, the protoplasm of the cells becomes first coagulated and later undergoes disintegration. Where the epidermis is not destroyed complete repair will under favourable conditions result. But when the epidermis and the papillary layer of the corium have

[1] It is important to note that occasionally sugar appears in the urine as a result of gangrene. Such cases are not to be confused with instances of diabetic gangrene.

both been destroyed scarring will ensue, and in the case of extensive burns is apt to prove troublesome by the slowness of repair, the excessive formation of granulation tissue, and, when the scar-tissue contracts, in the production of deformity.

One feature in the general effects of burns, as in other severe injuries, is the production of *shock*. The degree of shock depends on the superficial extent[1] rather than on the depth of the burn. Severe shivering-fits may be experienced. The patient is cold from weakness of the heart's action brought about by reflex inhibition of the cardio-vascular centres. The temperature may fall to 95° F. or lower still. In some cases during the period of shock the patient feels no pain though consciousness is retained. Such cases often end fatally. When death occurs the internal organs are found to be congested. When recovery from shock takes place, this usually occurs within forty-eight hours and is frequently heralded by an attack of vomiting. The danger of shock past, the patient is exposed to the risk of septic infection, as will be explained in a future chapter.

Frost-bite. Under the influence of cold, exposed parts—nose, ears, fingers, and toes—become blanched from spasm of the arteries. In the slighter degrees of the affection a purple colour due to congestion supervenes. This may quickly pass away or a persistent dilatation of capillaries may remain. In cases where the part has been completely frozen for a considerable time, if it is too rapidly thawed, congestion from paralysis or inflammation due to changes in the vessel walls leads to *moist gangrene* of the part. In other cases where the frozen condition has lasted still longer, the blood-vessels are so changed that the blood cannot enter them and the part undergoes *dry gangrene*. That the tissues will bear a short period of freezing is proved every day by the use of the ether or ethyl-chloride spray.

Bed Sores. Compression long sustained and forcible enough to render a part completely anæmic will produce necrosis even

[1] This corresponds with what is observed in shock following amputations and other severe operations. After amputations the degree of shock is generally in direct proportion with the ratio borne by the ablated part to the mass of the body.

in healthy persons. If the skin is involved in the pressure it forms a slough which separates, leaving an ulcer. In persons whose heart's action is weak, as from long-continued fever, a slighter degree of pressure is effectual than in normal conditions. Thus old or weakly persons, when confined to bed, require constant attention to prevent the formation of bed sores over bony prominences, such as the heels, ischial tuberosities, etc. When the spinal cord has been injured by traumatism or inflammation, the lower limbs are paralyzed, and the skin insensitive and badly nourished, owing to disturbed innervation, —in such conditions extensive bed sores will form with great rapidity. Ulcers of similar causation frequently form in the larynx of bed-ridden and moribund persons from the mutual apposition of the mucous membrane covering the vocal processes of the arytenoid cartilages. The general indications for the prevention of bed sores are by the use of water beds, cushions, etc., to keep the skin protected from severe pressure over bony prominences, to maintain a state of cleanliness in the skin, and also of dryness by using spirit lotions and dusting-powders.

Gangrene from infective processes will be dealt with later.

Degeneration. Disturbances of nutrition, insufficient to cause death of tissue directly, result in altered vital processes, or metabolism, which is evidenced by the occurrence of degeneration in cells and intercellular substance.

The various retrogressive changes which the protoplasm and nuclei of cells and intercellular substance undergo in various circumstances can only be briefly noticed here. They affect both living and dead tissues.

Albuminous Degeneration, or cloudy swelling, is a change observed in the protoplasm of cells in conditions of fever, septic poisoning, etc. The protoplasm of the cell becomes finely granular. This change is associated with a swelling of the cell, which is due in part to imbibition of water. The granules are soluble in acetic acid. Albuminous degeneration readily passes into the next form of degeneration.

Fatty Degeneration consists in the conversion of part of the protoplasm of the cell into fat. What should be active nitrogenous material becomes a passive carbohydrate. The

commonest cause of the change is anæmia, general or local. In general anæmia the tissues of the whole body may be thus changed. Phosphorus, arsenic, and other poisons also produce fatty degeneration. Local anæmia produces the same result in a limited area. Thus in the heart, when branches of the coronary arteries are obstructed by atheroma, a patchy fatty degeneration ensues. The fat assumes the form of droplets, each one of which is isolated in an albuminous envelope. When nearly the whole cell has become fatty, it undergoes necrosis, as in atheroma of the arteries, and in the degenerated portions of tubercular, syphilitic, cancerous, and sarcomatous growths. Fatty degeneration may also follow necrosis, and then results in caseation. *Fatty infiltration* must be distinguished from *fatty degeneration.* In the latter, part of what should be active cell-protoplasm becomes fat; in the former, the active cell-protoplasm remains intact, but it forms fat, and stores it in the shape of one or more large drops. The normal adipose tissue of the body is an example of fatty infiltration of connective-tissue. When a muscle undergoes degeneration, whether from a nerve-lesion or from disuse, fatty infiltration of the connective tissue between the fibres occurs, so that the bulk of the muscle may appear to be maintained, or even increased, as in pseudo-hypertrophic paralysis.

Hyaline Degeneration. In various conditions cells, intercellular substance, and coagula of blood, lymph, or muscle substance assume a glassy appearance. The simplest form of this change is known as hyaline degeneration. It is observed in the internal walls of the arteries and capillaries of various parts of the body in renal disease, gout, etc. It is a common occurrence in the new-formed vessels of chronic inflammatory tissues, and in the cells and tissues of new growths. The capillaries of lymph-glands sometimes become greatly swollen and of vitreous appearance from this cause. The waxy degeneration of striped muscle-fibres that have been injured or exposed to the action of typhoid-bacilli appears to be due to hyaline degeneration of coagulated muscle substance. Hyaline material is deeply stained by carmine, acid fuchsine, etc.

Colloid degeneration has its type in the formation of colloid

(glue-like) material in the thyroid gland, where, in many elderly people, and in certain tumours, instead of the normal liquid secretion of early life, a jelly-like material is formed from the epithelial cells lining the acini. It must not be assumed that colloid matter formed by degeneration of cells in other situations is identical save in superficial physical characters with that which occurs in the thyroid. In pathological conditions colloid occurs most commonly as drops of clear jelly-like material formed by the degeneration of epithelial cells in certain cancers, such as those of the breast.

Mucoid degeneration results in the formation of clear, glassy substance, resembling in its optical characters hyaline and colloid matter, but differing from them in its chemical characters, being soluble in weak alkalies, and precipitated from solution by acids. It thus resembles normal mucus, whether formed from epithelial (goblet) cells or as an intercellular matrix, as in the mucous tissue of the umbilical cord. It is common in epithelial-cells of certain growths, such as ovarian cysts, and in the interstitial tissue of other growths, myxomata and myxosarcomata, and cancers.

Amyloid, lardaceous, or waxy degeneration is another condition in which a glassy material is produced in the tissues. It is distinguished by definite reactions, staining intensely with iodine, and giving a pink colour with methyl violet. This mode of degeneration has important relations in surgery, since its production depends on prolonged suppuration or late syphilis. The change is most serious when it affects the kidneys, the liver, and the intestine. These organs enlarge, becoming abnormally firm and waxy on section. Polyuria and albuminuria result from the affection of the kidneys, diarrhœa from that of the intestine. The change is first observed between the muscle-cells of the middle coat of small arteries. Later it appears in the walls of capillaries immediately external to the endothelial layer. If the cause of this disease can be removed before it is too far advanced, whether it be a source of suppuration such as a tubercular bone or active tertiary syphilis, the degenerated organs will recover their normal characters, the amyloid substance being absorbed.

Calcification, which occurs normally in the intercellular substance of bone, is met with in pathological conditions, chiefly

as an infiltration of necrotic matter, *e.g.* in atheroma, caseous glands, etc. It is a common occurrence in the walls of dead hydatids and other parasites, such as trichinæ. In calcified arteries the middle coat is the seat of the deposit, and after dissolving out the phosphate and carbonate of lime with acids, the muscle-fibres are seen to have undergone a form of hyaline degeneration as well as calcification.

Pigmentation is either extra- or intracellular. Extracellular pigmentation is most commonly the result of hæmorrhage. The hæmoglobin becomes changed into hæmatoidin and other substances, which may either remain in the intercellular spaces, or be taken up by the cells. This occurs also in chronic inflammatory processes. The normal pigmentation of the deeper cells of the epidermis is increased in tubercular disease of the suprarenal capsules, and at the edges of the depigmented areas in an affection of the skin named leucodermia. In leprosy, areas both of increase and loss of pigment also occur. The colouring matter of normal pigmented cells, and of the pigmented cells of melanotic sarcoma and cancer, is probably elaborated by the cell-protoplasm independently of blood-colouring matter.

CHAPTER II.

REPAIR.

The Regeneration of Fibrous Tissue. By the term "repair" in the tissues is meant the process by which breaches of continuity due either to injury or disease are closed. Of all kinds of repair that which results in the formation of scar-tissue is the most important to be understood. It is the commonest and the histological processes which lead up to it are repeated in the most varied conditions. As examples may be mentioned: the healing of wounds of the fibrous part of the skin, the healing of the divided inner and middle coats after the ligature of an artery, the organization of clot, the formation of adhesions, *e.g.* in the pleura, the repair of divided tendons, and the reunion of tubes such as the intestine and ureter after division. It may be studied in detail in the case of a tendon divided subcutaneously with antiseptic precautions.[1] If the tendo-Achillis of a rabbit is examined twenty-four hours after aseptic division the clean-cut ends are seen to have separated about one inch and to be coated with a thin layer of blood-clot. They are joined together, and, for a short distance, ensheathed by a jelly-like straw-coloured coagulum which is as large as the tendon, filling out the tendon-sheath. The puncture in the tendon-sheath is closed by a little blood-clot which is adherent to the tissues outside. This uniting medium or "plastic lymph" is formed by the coagulation of lymph which has escaped from the

[1] This description of repair of tendons is based on a series of observations made by the author and briefly recorded in *Transactions of the Pathological Society*, 1895, p. 218.

divided lymphatics of the tendon and from plasma which has escaped from the small blood-vessels injured by the operation. At the end of forty-eight hours the appearance to the naked

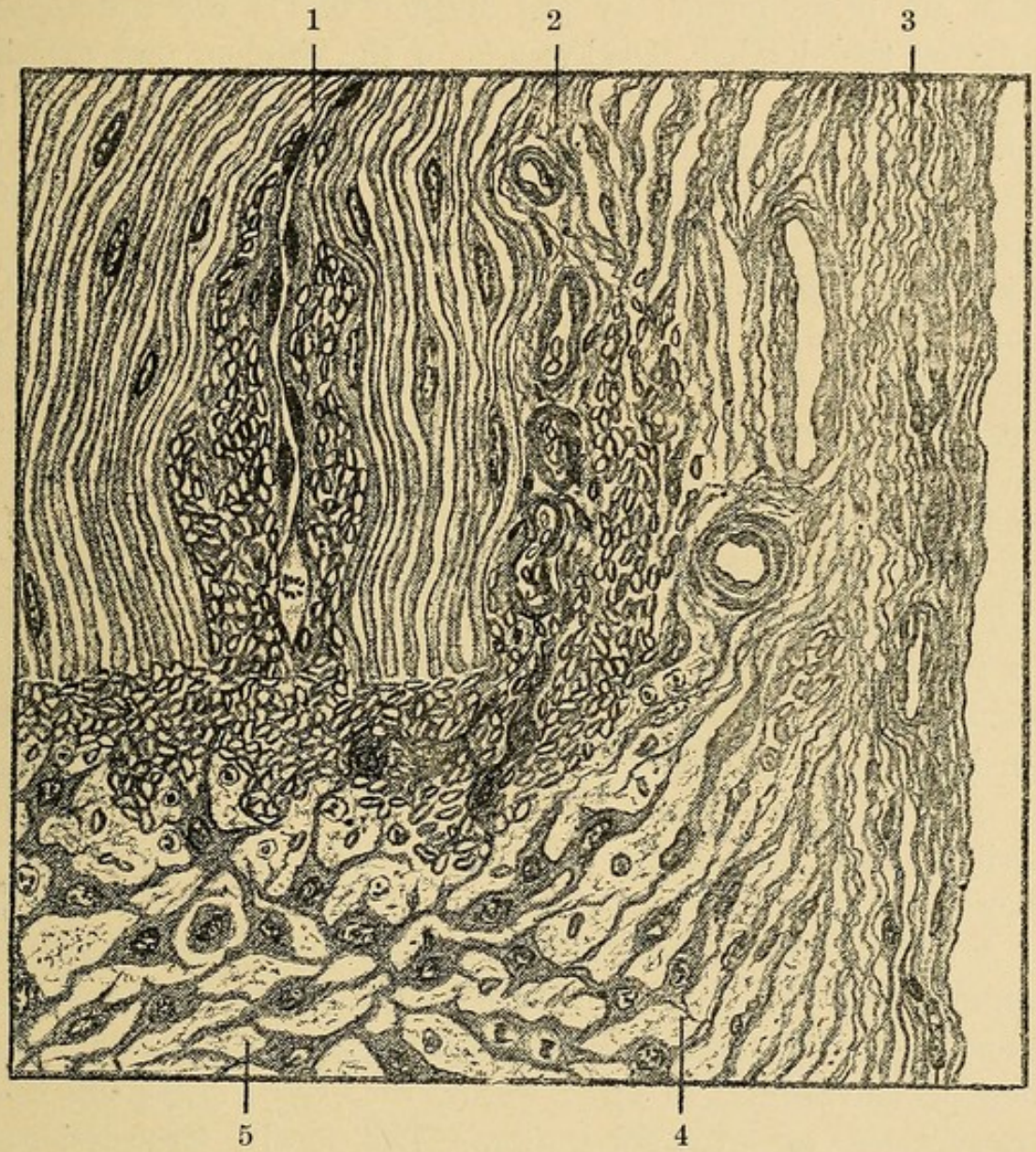


FIG. 3.—Part of a longitudinal section through one end of the tendo-Achillis of a rabbit, 48 hours after operation. 1, A capillary lying between two bundles of tendon fibres. The cut (lower) ends of tendon fibres show no change; they are surrounded by effused red blood-corpuscles which extend upwards between the tendon fibres, and also on the outer side of the tendon. 2, The inner part of the tendon-sheath where it closely invests the tendon and contains many small blood-vessels. 3, The outer part of the tendon sheath which consists of fibrous tissues with some blood-vessels. 4, The inner aspect of the sheath with proliferating cells. 5, Young connective-tissue cells. × 500.

eye is just the same as at the end of the first day, but if the tendon with the sheath and the uniting material be excised, hardened, and cut in sections, it is seen that active biological processes have occurred. The whole of the uniting bond is

traversed by branched young connective-tissue cells linked together, and careful examination reveals the fact that these cells are multiplying by indirect division,[1] and also that other cells are being formed by the division of (1) the endothelial cells of the divided blood-vessels of the tendon, and (2) the connective-tissue cells of the sheath and of the interfascicular spaces of the tendon. Besides the newly formed connective-tissue and endothelial cells are seen white blood-corpuscles, both the more numerous variety with a subdivided nucleus, and the less numerous uninuclear variety or lymphocytes. In the neighbourhood of the clot leucocytes of each variety may be found to have engulfed some of the red corpuscles. In the interior of some of the young connective-tissue cells both red and white blood-corpuscles are to be observed. This inception and subsequent digestion of cells and other extraneous particles by living cells is termed *phagocytosis.*[2]

The activity of growth and multiplication of cells[3] is caused in part by the presence of an unusual amount of food in the shape of serum, fibrin, and blood-corpuscles. The cells become predatory amœbæ. Their characters are shown in more detail in Fig. 4. From their destination, which is about to be described, they are termed "fibroblasts" and "vasoblasts" (Gr. *blastos* = bud).

A divided tendon examined on the fourth day presents a new appearance. The uniting medium, though still jelly-like, is now everywhere traversed by red streaks due to the formation of new blood-vessels. In other words, the *plastic lymph* has been transformed to *granulation tissue.* The newly formed blood-vessels arise as buds from pre-existing capillaries. In Figs. 3 and 4 examples of mitotic subdivision of the endothelial cells of capillaries in the vicinity of an injury are given.

[1] This is the principal mode of cell multiplication. Some of the stages of the process are shown in Fig. 4. It applies equally to epithelial and to connective-tissue cells. The process is also termed "mitosis" (Gr. *mitos* = thread), and "karyokinesis" (Gr. *karyon* = nucleus, *kinesis* = motion).

[2] Gr. *phagein* = eat, *kutos* = sac, vesicle, cell.

[3] An increase in the number of cells of any tissue is called "regeneration" when it serves the purpose of repairing a breach of continuity, and "hyperplasia" when it occurs in other conditions.

Examination of sections of the divided tendon on the fourth day affords evidence of the mode of formation of new blood-vessels and also of the formation of new connective-tissue fibres from the young connective-tissue cells or fibroblasts, as is shown in Fig. 5.

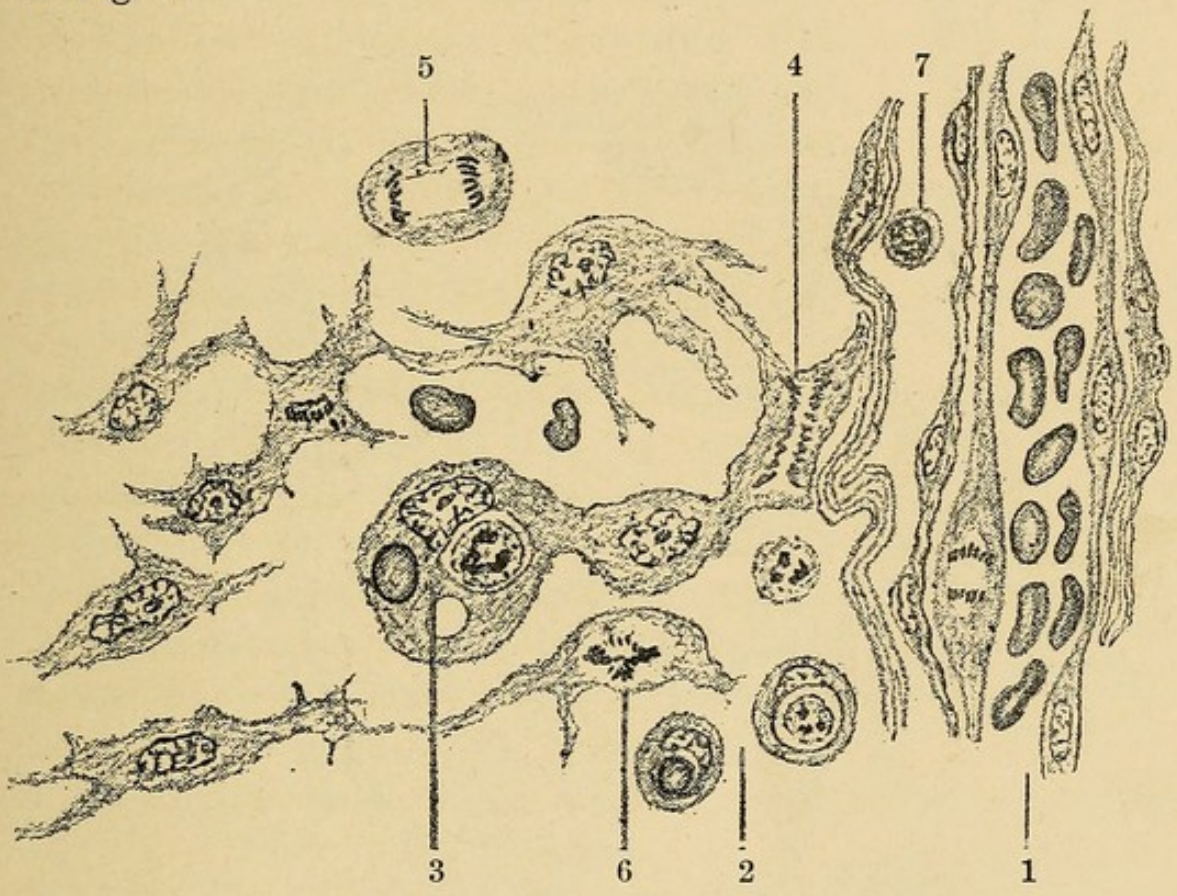


FIG. 4.—Part of the bond uniting the divided ends of the tendon after 48 hours. 1, A capillary of the tendon-sheath containing red blood-cells; one of its endothelial cells is undergoing subdivision. 2, Points between two lymphocytes, one of which contains an ordinary multinuclear leucocyte, the other a red corpuscle. 3, Indicates one of the fibroblasts which contains a leucocyte, a red corpuscle, and a vacuole. 4, Points to a connective-tissue cell which is undergoing division, and leaving the fibrous bundle which belongs to it. 5, Points to a rounded free connective-tissue cell undergoing subdivision. 6, Shows a fibroblast with an irregular mitosis. 7, A lymphocyte. Drawn to scale ×1000 diams. This figure is composed of several areas selected from a section.

Some other features in the structure of the new tissue at this stage are worthy of note. The cut ends of the tendon still present no change, and the blood-clot adhering to them has been to a large extent removed. Traces of digested red blood-corpuscles reduced to hæmatoidin granules are to be seen in some of the fibroblasts. The formation of fibrous tissue by the fibroblasts appears to occasion some shrinkage of the new tissue, for it contains in its central part clefts of relatively con-

siderable size filled with serous liquid. By the eighth day the new tissue, as seen after removal from the body, has a greyish pink appearance. It is now fairly firm, having lost its jelly-like appearance. The microscope shows it to be vascular

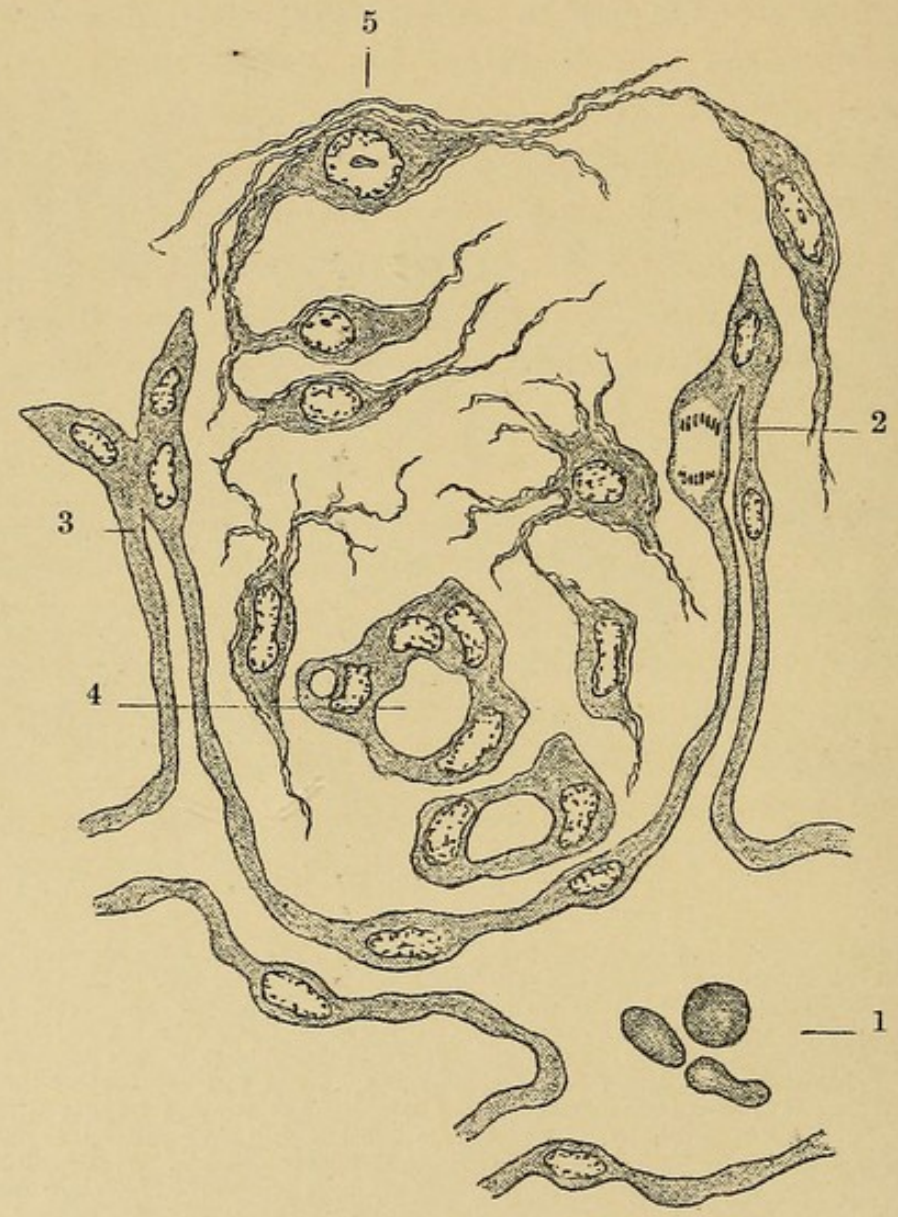


FIG. 5.—A section of a portion of the uniting medium on the 4th day. 1, A dilated capillary belonging to the tendon-sheath. 2 and 3, New capillaries branching off from 1. At the apex of 2, one of the cells (vasoblasts) is undergoing subdivision for the formation of a fresh branch. 4, Cross-section of a new capillary: one of the cells forming the vessel wall is vacuolated from the extension of the lumen of the vessel into it. 5, One of the young connective-tissue cells: the outer portion of its protoplasm and its branched processes are becoming fibrillated. × 1000 diams.

throughout and the fibrillation is still farther advanced; the fibroblasts are now closely packed, and, for the most part, are spindle-shaped. The cut ends of the tendon still present no marked change. On the fourteenth day the uniting bond consists of

well-formed scar-tissue (Fig. 7), and although to the naked eye the glistening white original tendon still contrasts sharply with the new material, as shown in Fig. 6. Under the microscope, in parts where the fibres of the new scar-tissue continue the direction of the tendon-fibres, it is somewhat difficult to distinguish where tendon ends and scar begins; but where the fibres of the scar cross the cut ends of tendon-fibres the distinction is easy. The junction of the scar with the tendon is made more intimate by the extension of the former into the interfascicular clefts as shown in Fig. 8. Moreover, fibroblasts find their way between the individual tendon fibres as at 4, Fig. 8.

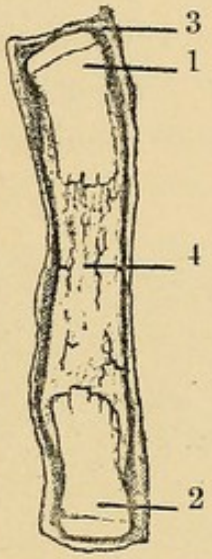


FIG. 6.—The severed ends of a rabbit's tendo-Achillis (1 and 2), with the sheath (3), and uniting scar-tissue (4), as seen after removal and vertical section on the 14th day after subcutaneous tenotomy. Nat. size.

After the second week a scar tends to contract owing to the progressive formation of fibres from the protoplasm of such fibroblasts as are not fully transformed. By this contraction the capillaries of the scar are narrowed and some of them are obliterated, hence scars on the

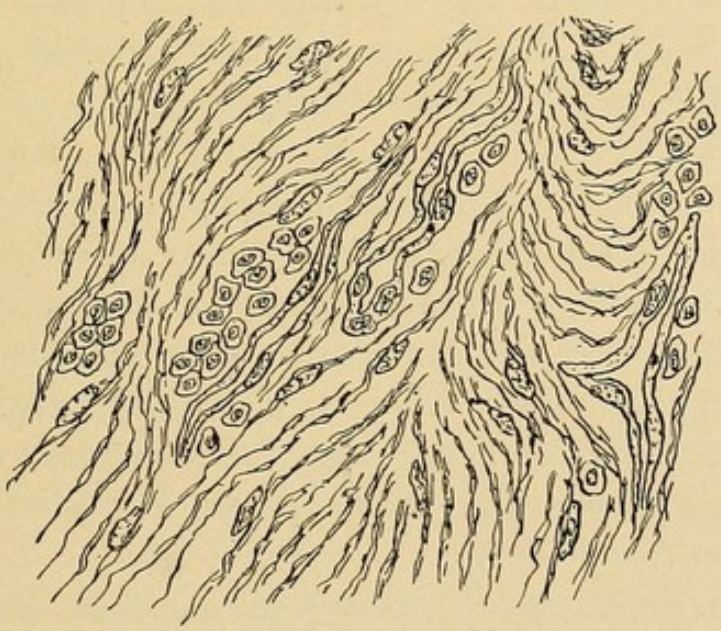


FIG. 7.—A portion of the scar-tissue in the middle of the uniting bond. There are several capillaries with clusters of cells around them. × 250.

skin which are at first of a red colour become gradually white. Before contraction begins the formation of new fibroblasts in

normal conditions comes to an end. The time occupied in the formation of scar-tissue is about the same in the rabbit as in man. To recapitulate the steps in the formation of a scar:

1. During the first few hours the effused blood and lymph collect between the severed tissues and coagulate, forming plastic lymph.

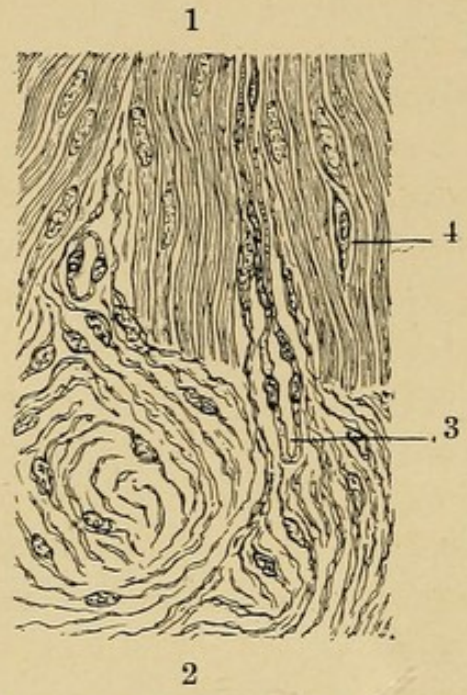


FIG. 8.—The junction of the divided end of the tendon (1), with the scar (2). At 3, a capillary of the scar extends into a cleft between the bundles, and a fibroblast (4) is seen between two tendon-fibres. The tendon fibres appear to be somewhat swollen. × 250.

2. The cells of the areolar tissue, and of structures such as a tendon sheath formed of connective tissue, begin to proliferate, and the new cells (fibroblasts) extend into the coagulum, and, at the same time, the endothelial cells of the neighbouring capillary blood-vessels also begin to subdivide. These processes are well advanced at the end of the second day.
3. The fibroblasts form bundles of fibres by the conversion of their protoplasm into fibrils (the latter yield gelatine on being boiled). The cells resulting from the subdivision of the endothelial cells of the capillaries (vasoblasts) extend into the coagulum like the fibroblasts. They remain in continuity with the parent vessel, the lumen of which extends into them, forming at first blind capillary diverticula. These become new capillary loops by coalescing with neighbouring diverticula. These processes are well advanced by the fourth day.
4. The development of fibroblasts and the formation of fibrous scar-tissue is so far advanced by the ninth day that the scar has some degree of resisting power, so that stitches may be safely removed from skin wounds at this period.

5. By the fourteenth day the growth of scar-tissue is fairly complete, and changes which lead to contraction commence.

It is often of importance to allow scar-tissue to attain its maximum strength before allowing a patient use of a part that has been operated on. It may be concluded that in aseptic wounds this stage has been reached at the end of five or six weeks.

Other modifications occur in the course of time induced chiefly by the physiological use of the repaired tendon. In the external part of the uniting bond, where it is attached to the sheath, from which it was largely generated, the fibres become drawn out by the alternate to and fro movement during the action of the muscle. At the same time the blood-vessels which pass from the sheath become elongated and attenuated, whilst those which pass into the uniting material from the interfascicular spaces of the tendon undergo compensatory enlargement. In some instances, where tendons have been divided and examined after death some years later, it has been found difficult to detect any difference between the old and the new material, although on closer scrutiny the parallel course of the fibres of the old tendon is seen to be interrupted where the scar-tissue joins together the two ends. The fibres of scars run in various directions, and the tissue which repairs the gap in a divided tendon is scar-tissue. These phenomena bear on surgical practice in regard to tenotomy, showing that though the ends of a tendon are separated for a considerable distance, good repair may ensue, and if the tendon-sheath is preserved intact, the lateral adhesions of the new piece of tendon will be limited to the sheath. It must be remembered that the new-tissue is merely scar-tissue, and will tend to contract and reproduce deformity unless this is prevented by suitable measures.

Repair of Wounds of the Skin and other Membranes covered by Epithelium.—*Normal Union.*[1] A clean surgical wound, the

[1] Before Lister taught the nature of wound-infection, surgeons described infective diseases of wounds as modes of repair. The commonest effect of infection, *i.e.* suppuration, was then so frequent, that thick pus was

edges of which are evenly adjusted under an aseptic dressing, is generally found about the ninth day to be united with sufficient firmness to allow of removal of stitches if they have been employed. When important cavities, such as the abdomen, have been opened, it is the rule to remove half the superficial stitches on the eighth, the rest on the sixteenth day. The firmness of the union depends chiefly on the new tissue, which joins the cut surfaces of the corium together. This new tissue is the *scar*, and it is formed in exactly the same way as that just described in the healing of tendons. By the eighth or ninth day the formation of fibrous tissue from the fibroblasts is fairly advanced. The epithelium is regenerated in the same way as the connective tissue. The epithelial cells, at and adjoining the cut surface, undergo mitotic subdivision, and grow over the subjacent granulation-tissue until the gap is closed.

When the edges of a skin-wound are not brought together, the plastic lymph coagulates on the surface, and into it grow the fibroblasts and vasoblasts. The new capillary loops are formed from the latter, and from the former the fibro-cellular tissue, which covers and surrounds the capillary loops; thus there is developed the red, slightly-granular surface, from which the name granulation-tissue has been derived. The new epithelium grows inwards over the granulations, and can be

called "laudable." Surgeons dreaded what seemed to be the vagaries of wounds: "traumatic erythema," suppuration, etc. John Hunter introduced the terms healing by first, second, or third "*intention*," as if a wound were a creature having a free will. The process of healing is always the same, whether it takes place between closely adjusted surfaces ("*primary union*"); on an open surface ("*by granulation*"); or by the *adhesion* of granulating surfaces which have been brought into apposition. An *immediate* union of wounds has been described. It is true that in certain protozoa, and other lowly organized animals, clean incisions are immediately repaired by the reunion of the parts of divided cells. A similar event is also possible in cellular tissues of man, and is sometimes observed in the epidermis; but the completion of the union by the formation of formed elements, such as the fibres of a scar, always occurs in the manner described above. And the cohesion of epithelia is so slight that complete repair cannot be said to have occurred in anything worthy of the name of a wound until these slower changes have been effected. Lister's first paper on antiseptic treatment appeared in 1867.

recognized as a smooth narrow zone at the margin of the wound. The advancing edge of the new epithelium is thinnest and shows the red of the granulations through it, its middle part being thicker, the colour of the granulations is dulled to a blue; and the thickest and more opaque outer part of the zone has a whitish appearance, owing to the refraction of light produced by the several layers of young epithelial cells.

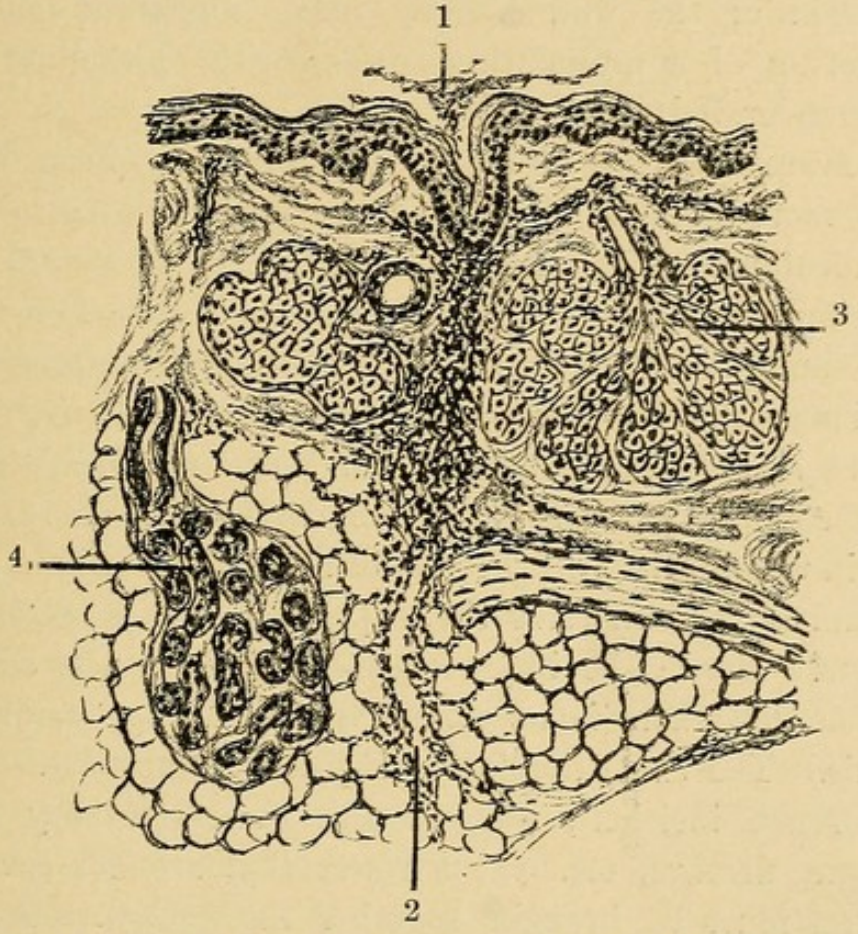


FIG. 9.—Section through a surgical wound of the scalp five days after operation. 1, Small scab covering the line of the incision. The continuity epidermis has been completely restored. The gap in the corium is sealed by plastic lymph which is infiltrated with fibroblasts which appear to have been derived chiefly from the loose connective tissue adjoining the capillaries. The weakest part of the union is that between the opposed surfaces of the subcutaneous fat (2), where cell-proliferation has taken place, but the plastic lymph does not completely fill the breach. There is a strand of unstriped muscular tissue on the right side above the fat. 3, Sebaceous-, 4, Sweat-gland. × 60.

As is suggested in Fig. 9, the subcutaneous fatty tissue is slow to undergo repair, probably because of the presence of liquid fat which prevents the fibrinous coagulum from adhering and binding together the opposed surfaces. The formation of scar-tissue from fibroblasts, etc., is the same as in the healing of fibrous tissue.

Skin Grafting. If small snippings of epidermis are made from a cleaned portion of healthy skin, and planted with the deep surface downwards on a healthy granulating surface, the deeper cells of the transplanted epidermis, nourished by the serum which exudes from the granulations, not only continue to live, but actively proliferate, forming foci from which new epidermis extends over the surface in precisely the same way as that at the margin of the wound. By using numerous skin-grafts, the covering of a granulating surface with epithelium may be greatly accelerated.

Skin Transplantation. Portions of clean skin, even a square inch or more in size, sliced off by a razor so as to be free from subcutaneous fat, and applied to a fresh wound or to a granulating surface, will, under favourable circumstances, retain vitality, and by the growth of vasoblasts acquire a vascular connection with the surface of the wound. Bone,[1] as well as fibrous and epithelial tissues, will bear transplantation under suitable conditions. In the above description of the healing of a skin-wound, it will be noticed that epithelium produces epithelium only, connective tissue produces dense connective or scar-tissue. Hence the importance of adjusting the edges of a wound, so that there is no undue tucking-in or overlapping of epidermis. The same applies to mucous membranes. Thus in closing rents, incisions, etc., in the bowel, urinary bladder, œsophagus, urethra, etc., the surfaces that are not covered by epithelium must be brought together throughout. A mucous surface must never be left exposed towards the peritoneum.

In *contused* and in *lacerated* wounds, such as are caused by blunt instruments, bullets, etc., a certain amount of tissue is devitalized by the mechanical injury. In such wounds healing is delayed by the separation of the dead parts, and, further, the

[1] The fact that skin, bone, and other tissues will bear transplantation may tempt a surgeon to refix, say, a finger-tip that has been smoothly sliced off. In such a case, although the parts are cleansed as well as may be with antiseptics, readjusted carefully, and placed under an antiseptic dressing, only the proximal one-eighth inch or so of grafts which contain skin, subcutaneous fat, and bone retains its vitality, the rest undergoes necrosis. So that except the part be a clean slice of skin devoid of fat, the attempt to save it is not worth making.

difficulty of rendering such wounds aseptic is very great, and repair is frequently delayed by suppuration.

Coagulation of Blood and Organization of Clot. The formation of fibrin, a solid elastic substance, from a previously liquid plasma, may be regarded as a degeneration of the intercellular substance of the blood, and the readiness with which fibrin undergoes hyaline degeneration suggests that its formation is akin to that process. But whilst coagulation is a retrograde process as far as the blood is concerned, it is a conservative process to the organism at large: for the coagulation of blood about the open ends of divided arteries is an important factor in natural arrest of hæmorrhage, and also in the repair of arteries after ligature or other artificial modes of occlusion. In certain individuals the tendency of blood to clot about the openings in divided blood-vessels is diminished. This condition constitutes a disease, and is known as **hæmophilia** (Gr. *haima* = blood; *philos* = beloved). Persons who suffer from the disease are called bleeders, and the condition is hereditary in the males of certain families. Bleeders are exposed to the risk of serious, or even fatal hæmorrhages from trivial wounds, such as result from the extraction of teeth, etc. Slight contusions which in ordinary persons would pass unnoticed, in them give rise to vast extravasations in the subcutaneous tissue or intermuscular spaces. They are liable to sudden extravasations into joints or bursæ and other parts. The effused blood certainly undergoes coagulation in bleeders as it does in other individuals, but it does not clot so readily about the openings in divided blood-vessels. The condition may be due to some defect either in the blood or in the walls of the vessels, or to both. Wright of Netley has suggested the administration of chloride of calcium as a drug likely to diminish the dangers and sufferings of bleeders by increasing the coagulability of the blood.

Blood-clot consists of a network of fibrin holding red and white corpuscles in its meshes. The fibrin may be regarded as dead material. The red and white corpuscles also die soon after coagulation has taken place. In the above description of healing of tendons it has been shown how the corpuscles are removed by phagocytes. If the clot is too large for speedy

removal by phagocytes, one of two events occurs: if there is abundance of serum around the red corpuscles, then hæmoglobin is dissolved out and absorbed by the lymphatics, and their stromata remain as part of the clot, or if the serum is not sufficient, the hæmoglobin becomes modified to brown translucent hæmatoidin. By the term *organization* as applied to blood-clot is meant its conversion into fibrous tissue. This conversion is brought about in precisely the same manner as that described above in the blood-clot and fibrinous coagulum formed between the ends of a divided tendon. It is effected by the ingrowing of fibroblasts and vasoblasts.

Repair in Arteries. When an artery is completely divided, the two ends separate from each other by the *retraction* which takes place by virtue of the elasticity of the elastic and longitudinal muscular tissue of the artery wall. The blood then flows out of the artery, and for a certain distance passes over the fibrous tissue of the sheath, which does not retract to the same extent as the artery. Blood, when no longer in contact with the normal endothelium, tends to clot and form a plug over the cut end of the artery. This is the *external coagulum*, which plays a most important part in bringing about the *natural arrest of hæmorrhage* which occurs when only small arteries have been divided. Another factor consists in the *contraction* which takes place in a divided artery owing to the contractile power of the circular fibres of the middle coat. The contraction takes place partly from the stimulus of the injury, partly from the local diminution of blood-pressure due to the opening of the artery. In "bleeders," as has been already explained, this coagulation, which leads to the *temporary* arrest of bleeding, fails, and the subjects of this disease may die from bleeding of even small wounds. It not infrequently happens that a clot formed about the mouth of a divided vessel is sufficient to arrest the hæmorrhage with the lowered blood-pressure that obtains during the "shock" which follows a severe operation; but when the patient recovers from shock, the higher blood-pressure dislodges the clot, giving rise to *recurrent hæmorrhage.* This occurrence takes place usually within twenty-four hours of the injury. After the temporary arrest of hæmorrhage the circulation of blood ceases in the part of the

artery between the divided end and the nearest branch. The stagnant blood in this section of the artery undergoes coagulation, forming the *internal coagulum*, which becomes converted into fibrous tissue in the same way as after the ligature of an artery. This leads to the *permanent arrest of hæmorrhage.* Where only capillaries and the smallest arteries (arterioles) have been opened, the coagula form in the tissues around their divided ends, and soon cause the temporary arrest. Where the structures around divided arteries are altered by the previous formation of fibrous tissue, the mouths of the small vessels are prevented from contraction; the vessels are then said to be "canalised," and the arrest of hæmorrhage is delayed owing to the loss of the contractile power of the arterioles.

The Artificial Arrest of Hæmorrhage. Styptics act either by increasing the tendency to coagulation or the contraction of the artery. Cold and heat act in the latter, perchloride of iron chiefly in the former, way. The cautery chars the open ends of the divided vessels, and causes them to be obliterated by cohesion of the coagulated substance of the artery wall. Ligatures applied in the ordinary way cause the division of the inner and middle coats, which look almost as though they had been cut through with a knife. If the ligature completely occludes the lumen, an internal coagulum forms as high as the nearest branch.

The frequency with which, in the ligation of an artery, the first loop or hitch of a knot yields before the knot is completed, has led Ballance and Edmunds (*Ligation in Continuity*, 1891, p. 396) to recommend that an artery, when tied in its continuity, should be surrounded by two single hitches placed side by side, and tied in the same manner, so that by taking up the two ends on each side, and tying them in pairs, a reef knot is completed, and constitutes what the authors name a "stay-knot." They also point out that the formation of the internal clot, and subsequent obliteration of the artery, takes place when the inner and middle coats are only drawn together and are not divided. The same kind of occlusion of an artery occurs when pressure is applied for a sufficient length of time, as in the old-fashioned procedure called "acupressure"—that is, when a needle is passed

through the skin under an artery, and out through the skin again. *Pressure forceps* crush the vessel and cause the opposed endothelial surfaces to cohere. *Torsion* of an artery results in the stripping up of the middle from the outer coat, which is twisted into a cone-shaped cap over the open end of the artery, while the inner and middle coats tend to curve inwards and meet, as shown in Fig. 2. The occlusion of the artery is completed when the blood clots within it. Slow division of arteries by the "écraseur" prevents hæmorrhage in a similar way to torsion, the inner and middle coats giving way long before the outer, which is drawn out as a cap over the other coats.

When an artery is only half divided, retraction is prevented, and the natural arrest of bleeding is slow, but usually bleeding soon ceases if the division of the artery be completed. A similar difficulty in the way of a natural arrest is observed when a branch is divided close to the parent trunk.

The repair of an artery as it occurs after the application of a ligature is shown diagrammatically in Fig. 10, in which it is imagined that the artery during repair had lain in the horizontal position. The upper part of the clot in such a condition adheres usually only to the under side of the lumen of the artery, whilst nearer the part of the vessel surrounded and injured by the ligature the clot adheres by its whole circumference. In the case of twisted or crushed arteries the process is practically the same. The most dangerous hindrance to repair (hence the most common cause of *secondary hæmorrhage*) is the occurrence of septic processes, as will be more fully detailed below under "Diseases of Arteries."

The importance of the complete sterilization of ligatures is nowhere more emphatic than when the ligature of a large artery is in question.

Contusion of Arteries. Severe contusion of an artery causes rupture of the inner and middle coats and their separation from the outer coat, and subsequent curling up, as shown in Fig. 2. Such severe injury to an artery is usually accompanied by injury to the veins, and also to the other arteries around the main artery, and so gangrene frequently results. Slight contusion of the wall of an artery may cause softening and the subsequent formation of an aneurysm. See "Diseases of Arteries."

Subcutaneous rupture of a large artery, e.g. the popliteal, causes a large extravasation, which distends the tissues, and by pressing on veins prevents the return of blood from the parts beyond the injury, and hence gangrene is a common result. Ruptured arteries should be cut down upon, and tied at the earliest possible moment.

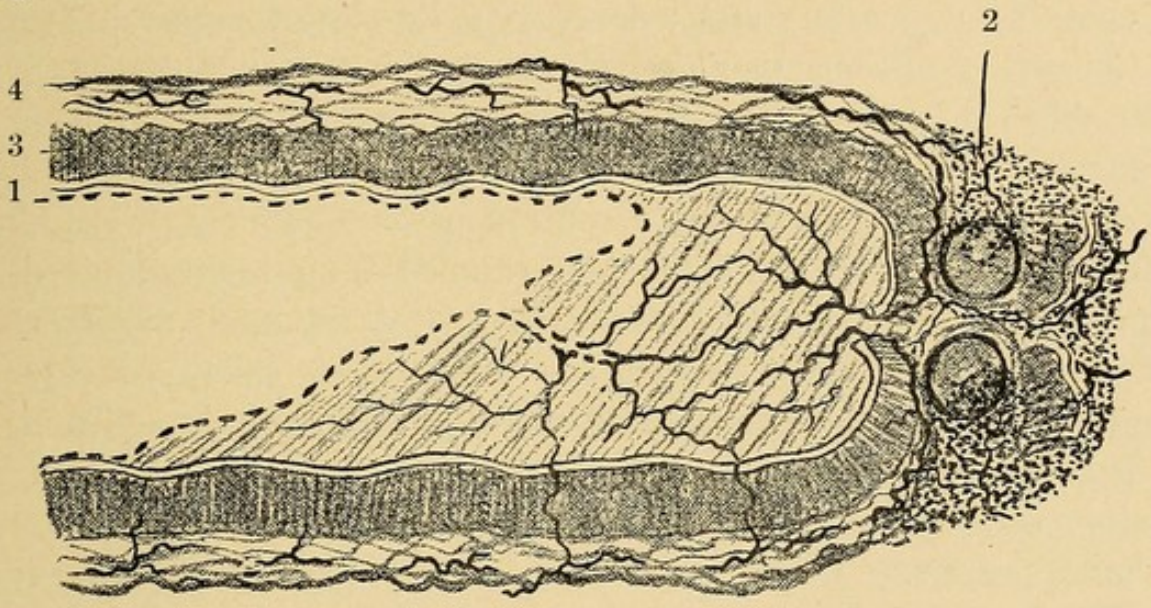


FIG. 10.—Diagram of an artery cut longitudinally in a vertical plane after ligature. Near the tied end the clot adheres to the entire circumference of the interior of the vessel, elsewhere the clot is adherent only at its lower aspect. The endothelium (1) has grown over the free surface of the clot and has projected into it, forming new blood-vessels which ramify in the clot and join branches of the vasa-vasorum of the outer coat (4) which enter the clot through the middle (3) and inner coats, and through the divided end of the artery. The clot is also traversed by a net-work of fibroblasts which are not represented in the diagram. A cat-gut ligature, surrounded by granulation tissue (2) is shown at the divided end of the vessel. Fibroblasts are invading the ligature from outer side.

Traumatic Aneurysm. When an artery is injured by a subcutaneous puncture, blood escapes into the surrounding tissues. The outermost part of this effusion may clot and undergo organization, forming the fibrous sac of an aneurysm, whilst the inner part remains fluid by its being kept moving in a stream, which first emerges from the opening and then re-enters it. Such a condition constitutes a *traumatic aneurysm.* Another form of traumatic aneurysm results from the gradual dilatation of a scar in the wall of an artery. Such aneurysms were common when bleeding from the superficial temporal arteries was practised for inflammation of the eye. The artery was partially divided, and when enough blood had escaped, a pad was placed over the wound. Under this pad

healing occurred, and as soon as the pad was removed the blood found its way along the artery, and dilated the still weak scar-tissue in the lateral opening.

Arterio-venous communications result from punctured wounds, implicating both an artery and a vein. They are commonest in situations such as the bend of the elbow and Scarpa's triangle, where a large artery and a large vein lie close together. Two different conditions may result: (1) A direct communication is established between the artery and the vein. The arterial blood passes into the vein, which becomes dilated and thickened, pulsating with a thrill and a continuous loud-humming "bruit." The arteries become dilated and thinned from lowered blood-pressure. This is an *aneurysmal varix.* (2) A traumatic aneurysm forms and communicates with the vein: this is called a *varicose aneurysm.* All the effects of aneurysmal varix, and, in addition, those of aneurysm follow. Thus the varicose aneurysm is the more serious condition. The nutrition and utility of the limb suffer greatly. If pressure fails, the only effective treatment is to secure the artery above and below the opening into the vein in "1" and the aneurysm in "2."

Hæmorrhage and Repair of the Blood. When blood escapes from the surface of the body, it does so by jets of a bright red colour if an artery is wounded, and in a dark uniform stream when it comes from a vein. When blood escapes into areolar tissue the occurrence is termed extravasation, when into an internal cavity it is called internal hæmorrhage.

If a very large quantity of blood be lost suddenly, as by injury to a large artery, the patient may die at once from heart-failure or syncope.

If the hæmorrhage is less considerable but still severe, the rapidity of the pulse is greatly increased, and the pulse-wave often seems large because the arteries are only partly filled between the beats. The surface of the body becomes cold and covered with sweat, the lips pale, there is deep sighing respiration, with a sense of "air-hunger." Thirst, faintness, nausea, restlessness, singing in the ears, failure of the power of vision, and loss of consciousness may all be noticed. If frequently repeated small hæmorrhages occur, marked anæmia,

œdema of the legs, weakness, and a tendency to syncope are observed.

Infants and aged persons bear loss of blood badly. In young persons, and especially women, the blood is rapidly reproduced: the corpuscular elements from the red marrow, lymph glands, etc.; the fluid part in a measure by absorption from the alimentary canal. Transfusion of saline solution, made by adding a teaspoonful of table-salt to a pint of sterilized water, may cause a patient to rally who would otherwise die of syncope.

Repair in Veins. Clean incised lateral wounds of veins, such as are made in phlebotomy, usually heal without entailing obliteration of the lumen of the vessel and, owing to the low blood-pressure, without any tendency to dilatation of the scar.

Entry of air into veins may occur in wounds about the root of the neck in which there is a negative pressure during inspiration. The air is sucked in with a sipping sound, and being frothed up in the right cavities of the heart, enters the pulmonary artery in bubbles which may cause death by blocking the branches of the pulmonary artery—air embolism; short of death, a severe attack of dyspnœa occurs.

Veins that have been ligatured heal in the same manner as arteries. To illustrate the events, the effects of thrombosis from a pathological process may be briefly described.

Thrombosis in Veins. The organization of the clot is much the same as in an artery. If the clotting is spontaneous, *i.e.* independent of ligature, etc., the exact result differs according to the condition of the endothelium of the vessel. If this has not been destroyed by inflammation or injury then the clot will probably adhere to the under aspect of the lumen, and the upper free surface of the clot will become covered by endothelium, so the lumen of the vessel will be restored in the form of a crescentic slit. If the endothelium has been destroyed the clot becomes adherent to the whole circumference of the vessel, at the extremities of the clot the endothelium of the vessel grows over it and sends in vasoblastic offshoots by which the clot becomes channelled, as is well shown in Fig. 11, from Coats's *Pathology*. If the clot is large and the circulation in the

neighbourhood sluggish, the organization may be incomplete. The outer part of the clot is then converted into fibrous tissue in the form of a capsule around the central portion of the clot, which then becomes the seat of deposit of carbonate and phosphate of lime and constitutes a "phlebolith." Phleboliths (Gr. *phleps* = vein, *lithos* = stone) are common in the dilated veins about the prostate and base of the bladder in elderly persons.

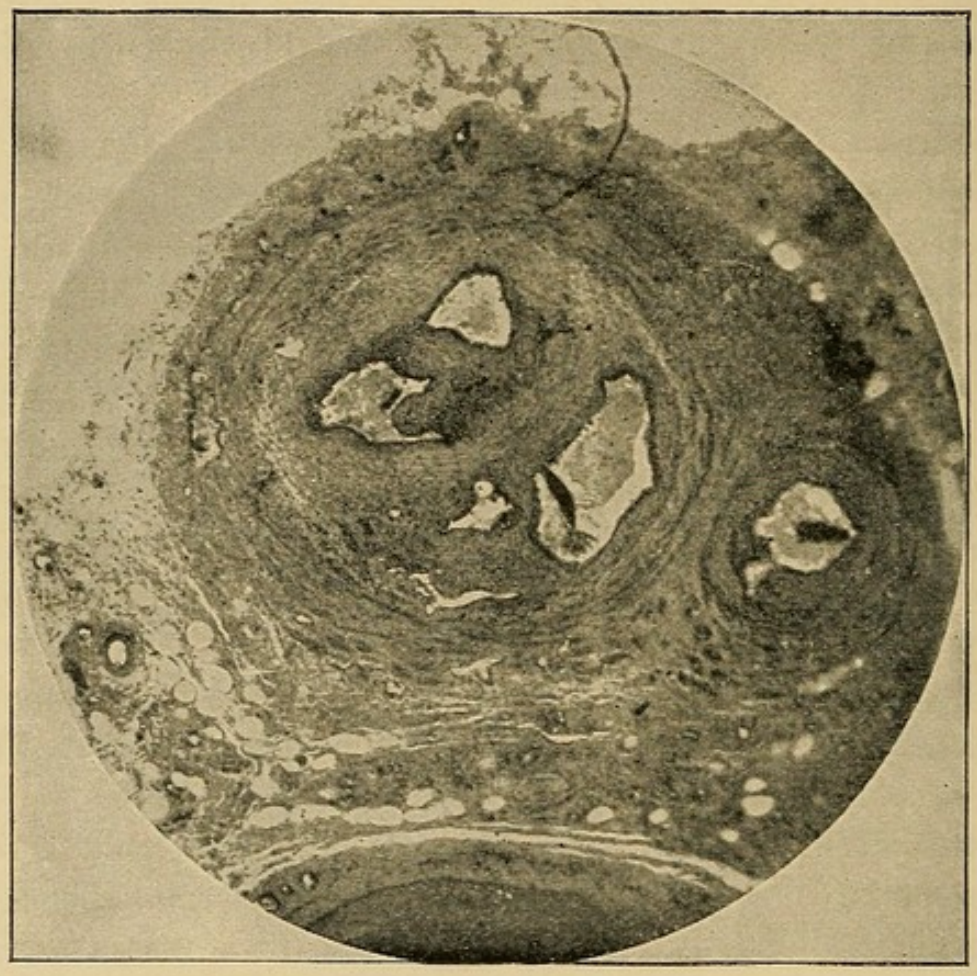


FIG. 11.—Two veins with organized thrombi in them. The thrombi contain large channels by which the circulation is restored. × 8. From Coats's *Manual of Pathology.*

Repair of Cartilage. Hyaline cartilage in most parts of the body, except that which covers the articular ends of bones, tends to be replaced by bone. Even that which is formed in the larynx ossifies, and the costal cartilages become calcified in middle age. Thus it might be anticipated that cartilage is a tissue which would have little tendency to reproduce itself after injury, and such indeed is the case. Wounds or fractures of hyaline cartilage are repaired by scar-tissue or by bone. The

behaviour of epiphyseal cartilage after traumatic (*i.e.* produced by physical violence, Gr. *trauma* = injury) separation of the epiphyses will be considered in the next section.

The Regeneration of Bone: Repair of Fractures. The process by which a fracture through a bone is repaired may be briefly summed up by saying that in the main it is the same as that described in the repair of tendons with the addition of ossification of the new fibro-cellular tissue. Taking a simple transverse fracture through the shaft of a long bone as an example, and presuming that the whole bone, including the periosteum, has been divided, the first event will be hæmorrhage from all three sections of the injured bone, *i.e.* from periosteum, compact tissue, and medulla, and from the injured tissues around the bone. The effused blood coagulates about the broken extremities of the bone. For a few hours additional plastic lymph will be effused and coagulate beyond the blood-clot as in the union of divided tendon. Next, connective-tissue cells and osteoblasts invade the coagulum from the injured muscles, etc., outside the bone, from the periosteum, the Haversian canals of the compact tissue, and from the medulla. The new tissue becomes fairly firm, like granulation tissue elsewhere, before the eighth day; it is then termed *callus* and may be distinguished by the fingers as a spindle-shaped swelling at the seat of fracture. The part of the callus that occupies the medullary cavity is called "internal," that beyond the surface of the bone "external," that between the fractured surfaces of compact tissue "intermediate" callus. Sometimes part of the new connective tissue consists of cartilage. This is especially the case in children and young animals generally, and where there is movement of the fragments. The formation of cartilage in the callus can often be recognized on inspection of the cut surface of the new tissue.

Under normal conditions the callus undergoes ossification. This process begins in the external callus. The study of macerated preparations is helpful to the gaining of a definite understanding of the process of ossification of callus. In the early stage of the process a series of little circular perforated projections of bone are seen to have grown out from the

FIG. 12.—Longitudinal section of a fractured rib, showing callus, etc.; the section is imperfect at the upper part. *a*, *a*, The broken extremities of the rib dove-tailing into each other; *b*, *b*, new-formed cartilage of the callus; *c*, *c*, new-formed bone in the outer part of the callus; *d*, *d*, fibrous tissue representing the internal and intermediate callus which have not yet undergone ossification. The new periosteum, which covers the callus, is not represented in the Figure. × 11. From Coats's *Manual of Pathology*.

surface of the compact tissue, showing that the first formation of bone occurs around blood-vessels which enter the bone from the periosteum.

The production of bone from granulation tissue connected with bone or periosteum is by no means limited to the repair of fractures. If an effusion of blood takes place beneath the periosteum, granulation tissue, which is subsequently ossified, forms on the deep surface of the periosteum. This process, which will be referred to again, is termed osteoplastic periostitis. The formation of callus in fractured bone might also be termed osteoplastic osteitis and periostitis. The bone salts are deposited around the osteoblasts of the periosteal portion of the callus in a manner similar to the normal formation of bone from periosteum. When callus forms at the site of the attachment of a muscle to the bone the formation of granulation tissue and of bone may extend for some distance into the muscle, constituting what is termed an osteophyte (Gr. *osteon* = bone, *phyton* = a shoot, a growth). The general plan of the formation of bone in the external callus is admirably shown in the accompanying Fig. 12, borrowed from Coats's work on *Pathology*.

The internal callus, when not prevented by movement of the parts, begins to ossify a little later than the external. The intermediate callus is the last to ossify, and the resulting bone uniting the fractured surfaces of the compact tissue is spongy at first, but it becomes gradually denser as time goes on. As soon as the intermediate callus is as strong as the original compact tissue, and able to transmit force and perform all the functions of compact tissue, the internal first, and later the external callus begin to be absorbed. This is effected by osteoclasts, as in the normal gradual widening of the medullary canal. The late ossification of the intermediate callus is probably due to its being affected by slighter movements than affect the rest of the callus. From its permanence the intermediate callus is also called the "definitive" callus. In some cases the formation of the definitive, and the absorption of the rest of the callus (provisional callus), is so complete that, after a year or two, it is impossible to find any trace of the fracture.

The rate of repair of bone is a matter of great practical

importance. As with repair of all kinds, the process is most rapid in the head and slowest in the lower limbs. These differences are due to the head being richer in blood-vessels, and to the mechanical difficulties which hamper the return of blood from the lower limbs, and by the richer nerve-supply, and the greater proximity to the brain and spinal cord enjoyed by the head as compared with the extremities. Ossification of the callus of ribs, even in old people, I have found to have begun by the ninth day, so that it is not surprising that a fractured nasal bone may be found to be repaired by firm osseous union as early as the seventh day—a fact that has its practical bearings. There is a skull in the museum of St. Mary's Hospital (No. 109), from a man who sustained an extensive comminuted fracture of the frontal bones and a fracture of the base, and who died of meningitis on the twenty-fourth day. In this case the whole of the fissures are solidly united by bone.

In the bones of the limbs the fracture is fairly firm as soon as the external callus is ossified. On an average this is about four or five weeks in the upper, seven or eight weeks in the lower extremity.

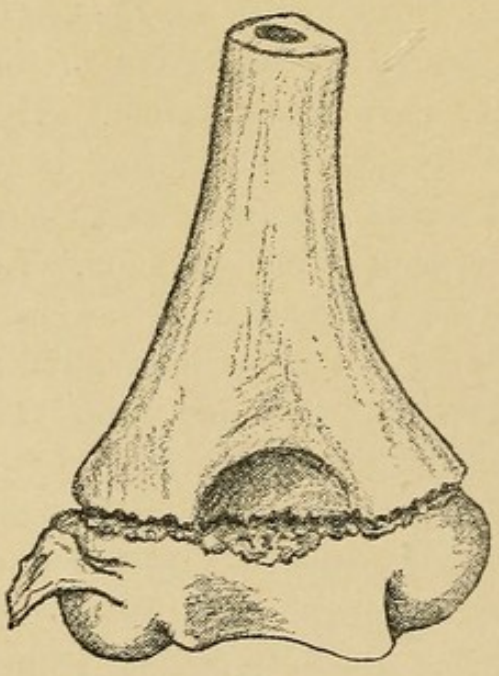


FIG. 13.—Separation of the lower epiphysis of a humerus. In this instance the separation occurred throughout at the junction of the epiphysis with the shaft.

Separation of Epiphyses. The growing tissue at the junction of the shaft and the epiphyseal cartilage, here weakened by the increased number of cartilage cells and the diminished amount of matrix, constitutes a narrow layer with parallel surfaces, and consists partly of cellular tissue and looped capillaries sprouting from the vascular bone of the diaphysis, and fitting in between the vertical pillar-like remains of the matrix of the cartilage, coated with a thin layer of new bone. This diaphysio-epiphyseal junction is the weakest part of a bone. Externally it is

clothed by the somewhat thickened periosteum, which in a slight degree atones for the weakness.

Repair occurs after separation of an epiphysis,[1] as after other forms of fracture, and the longitudinal growth of the bone is resumed; but when the epiphyseal ossification is present, and the cartilage is only a thin lamella which has been wholly destroyed by the mechanical violence of the injury or by subsequent suppuration, the growth of the bone in length is arrested at the damaged end, because there is no longer a continuous layer of cartilage to bud off cells towards the end of the diaphysis, from which new capillaries can be put out in the same manner as those of granulation tissue into the loose cellular tissue formed by the proliferating cartilage cells.

The accompanying figure (14), drawn from the skeleton of a hare, shows how much repair may be effected after separation of an epiphysis.

Failure of Ossification of the Callus. Many factors may, singly or in combination, prevent or impair the process of ossification in the callus. Two of these may be considered here. *Want of rest* may be first noticed, since usually it is in the power of the surgeon to avoid it. If the callus is continually stretched, and moved from side to side

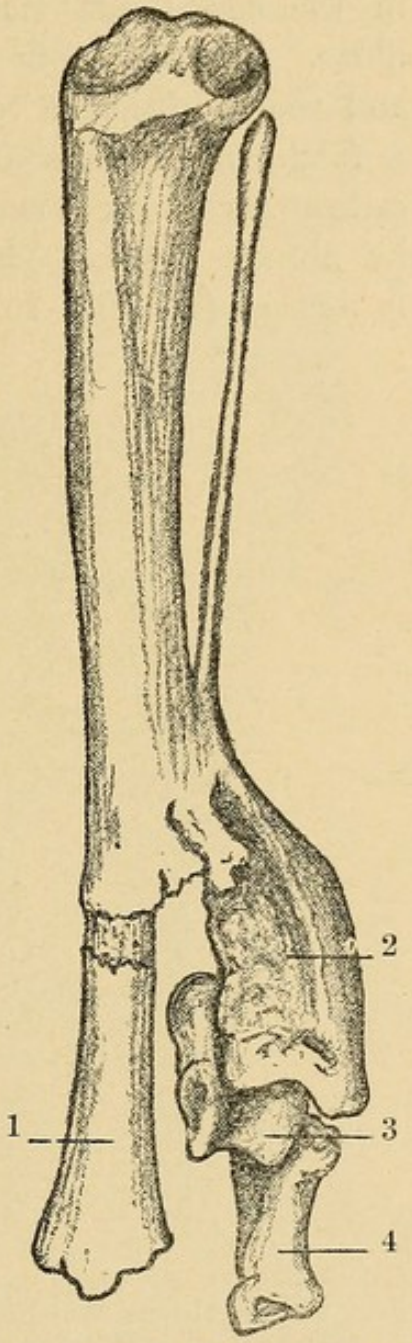


FIG. 14.—The bones of a right hind limb of a hare, showing the results of a compound fracture-separation of the lower epiphysis of the tibia. 1, Lower part of the shaft of the tibia which was protruded through the skin. It was necrosed and had begun to be separated from the rest of the bone. 2, A piece of bone, nearly one inch in length, which joins the separated lower epiphysis to the shaft. 3, Astragalus. 4, Os calcis. (40.) Nat. size.

[1] The line of separation of an epiphysis does not always keep to the diaphysio-epiphyseal junction. Very frequently portions of the shaft or epiphysis adhere to one or other of the fragments.

by the mobility of the fragments on each other, the changes in the new tissue may not proceed beyond the formation of fibres. The result is a false-joint of the synarthrodial type, and the condition is termed *ligamentous pseudarthrosis.*

If the movements are considerable, the central parts of the callus may break down, forming a cavity which is surrounded by fibrous tissue. Thus a false-joint of the diarthrodial type is formed (see Fig. 15).

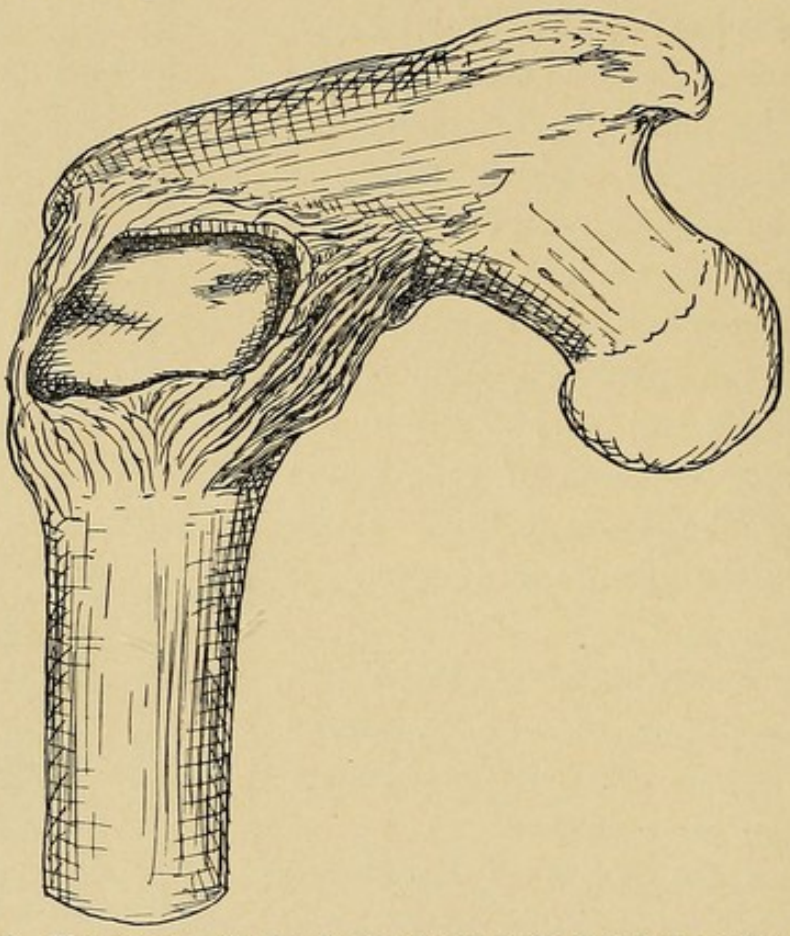


FIG. 15.—The upper part of a femur showing diarthrodial pseudarthrosis. There has been a fracture between the upper and second fourths of the shaft. There is a strong fibrous capsule formed from the external callus. The front of this capsule has been cut away in order to show the interior. The extremities of the fragments within this capsule are covered by fibro-cartilage. (91a.)

The commonest situations for such false-joints are the humerus and the femur. Bones in which nutrition is very active, such as the clavicle, are very rarely the seat of pseudarthrosis, even though they do not admit of perfect immobilization by splints.

A second condition in which fibrous union is apt to occur is when the fracture occurs within a joint. As examples, fractures of the olecranon, of the neck of the femur within the capsule (Fig. 16), and fracture of the patella may be given.

When, however, the fragments are driven one into the other (*impacted fracture*), they are likely to unite by bone in spite of their being within a joint cavity (see Fig. 17).

Compound fractures, that is, fractures in which the breach of continuity between the bones communicates with the exterior through a wound in the skin, heal in precisely the same manner as simple fractures, unless they become infected by pyogenic organisms and suppurate, a condition which will be described in a later chapter.

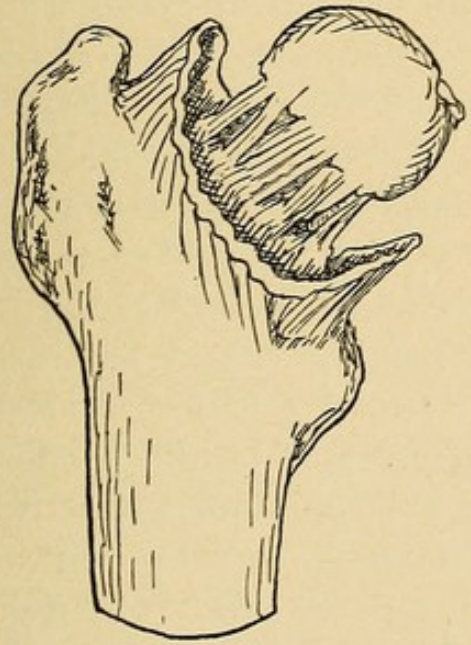


FIG. 16.—Intracapsular fracture of the neck of the femur, fibrous union.

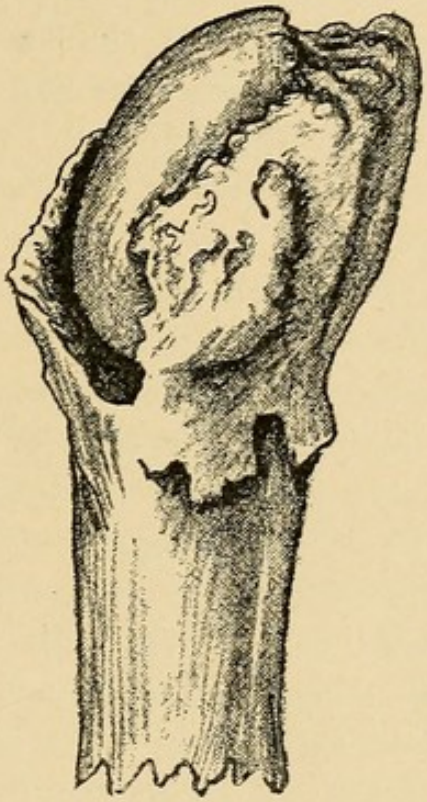


FIG. 17.—Impacted fracture at the anatomical neck of the humerus. The head is driven into the upper end of the shaft, and the greater tuberosity with the upper part of the shaft has been bent outwards and partly fractured. There is a considerable amount of bony union. From an old person who lived many years after receiving the injury.

Dislocation and Diastasis. Sprains. Dislocation means simply a displacement. In surgery, when used without qualification, it signifies the displacement of one bony surface from another with which, in normal conditions, it articulates.

When a marked recent dislocation is dissected, it is found that in addition to the displacement of the bones, the capsular ligament is torn, with the result that blood is effused both into and around the joint. Further, any tendons that are in close

relationship with the joint present effusions of blood in their tendon-sheaths, which may be ruptured, as also may the tendons and surrounding muscles. In some cases all these injuries are present yet the bones are not dislocated. This is termed a *diastasis.* (Gr. *dia,* apart; *stasis,* standing.) For instance, after an injury to the spinal column in the cervical region, I have found a torn intervertebral disc, and the anterior and posterior common ligaments, the capsular ligaments of the joints between the articular processes and those joining the laminæ and spines also torn through and the cord crushed, and yet the two vertebræ implicated were in their normal relative position. Here there was a diastasis of the spinal column, *i.e.* a dislocation which had righted itself.

When a dislocated bone is thrust through the skin, the dislocation is said to be *compound.* Dislocations may be *complicated* by fracture of the bone or serious injuries, such as the rupture of large arteries, veins, or nerves.

Repair after Dislocation. When the displacement is reduced immediately after its occurrence, the joint is fixed and the limb immobilized. After a few days, when the swelling of the joint has subsided, passive movements are begun, and are carried out in such a way that adhesions resulting from organization of clot, etc., within the damaged joint and in the sheaths of tendons in its neighbourhood are broken down, whilst repair of the laceration in the capsule is interfered with as little as possible. So-called "bone-setting" consists chiefly of breaking down adhesions in and about joints, adhesions which have become firm owing to the neglect of passive movements.

Unreduced Dislocations. When a dislocation is allowed to remain unreduced, adhesions form between the displaced bone and the surrounding structures, whilst the aperture in the capsule becomes closed by cicatrization. In this way a dislocation may become irreducible, or from firm adhesions having formed between the displaced bone and neighbouring blood-vessels or nerves, these may be injured in attempting reduction.

In cases of unreduced dislocation of a large joint, the patient is for a time unable to use the limb on account of pain; but in time, as repair becomes completed, the muscles, etc., accom-

modate themselves to new conditions, and, though impaired, a varying degree of function is regained. If the articular end of the displaced bone rests on another bone, it becomes flattened from pressure-atrophy, its articular cartilage is replaced by fibrous tissue, and the other bone on which it rests develops a more or less perfect new socket from the formation of a depression where the dislocated bone rests, and around the depression a raised lip of new bone grows, owing to the intermittent pressure and traction experienced during the movements of the limb. Meanwhile the original socket becomes more or less completely filled up by the formation in it of new fibrous tissue.

Sprains. Severe sprains imply stretching or tearing of ligamentous and muscular fibres. They are in some cases the clinical expression of diastasis. The extravasation of blood and long-lasting stiffness which follow sprains afford evidence that a considerable amount of injury has been done.

Repair in Striped Muscle. When a muscle like the biceps brachii is ruptured through its fleshy part, however much the torn surfaces are approximated by placing the limb in the position of greatest shortening of the muscle, *e.g.* by flexing the elbow after rupture of the biceps, a considerable amount of blood and plastic lymph is poured out, and after coagulation is "organized" and transformed into scar-tissue, which thus forms the chief part of the material of repair. But to a limited extent striped muscle is capable of regeneration. The injured fibres grow by a multiplication of the nuclei beneath the sarcolemma. These nuclei undergo division; they become constricted and separate into two parts. Tapering processes of protoplasm extend along with the new nuclei from the injured ends of the fibres, and become striated as they increase in size.

Unstriped muscle cells are regenerated by karyokinetic subdivision.

Regeneration of Nerves. In the brain and spinal cord injuries are repaired by fine scar-tissue generated from the neuroglia, and no effective regeneration of the nerve tissue occurs. In the peripheral nerves it is different. After a nerve trunk is cut through, complete restoration of function occurs

under favourable circumstances, usually after the part of the nerve beyond the point at which it is divided undergoes degeneration. This phenomenon is readily understood when it is remembered that the axis-cylinders of peripheral nerves are processes prolonged from nerve cells, the motor axis-cylinders being, in the case of spinal nerves, parts of the ganglion cells in the anterior cornua of the grey matter, the sensory axis-cylinders being prolonged from the cells of the ganglia of the posterior roots. The cranial nerves have similar origins. The nutrition of cells is governed by their nuclei, and when part of a cell not containing the nucleus is cut off from the rest of the cell, the part separated from the nucleus perishes. Thus when a mixed nerve trunk is divided, all the axis-cylinders below the division are cell-processes cut off from their nuclei, and consequently they perish. The myelin of the nerve fibres previously breaks up into drops which are taken up partly by the cells of the primitive sheath, and partly they escape into the surrounding lymph spaces. The axis-cylinders then become indistinct, and break up, leaving only a fibrous strand possessing more numerous nuclei than had the original primitive sheath.

The nutrition of striped muscle fibres depends on the nerves which enter them. They degenerate at the same time as the corresponding nerves. The fibres lose their transverse striation, becoming narrower and granular or undergoing a change termed hyaline degeneration (see p. 17).

The clinical signs of the degeneration of muscle are as follow: The reaction to the Faradaic current is lost in a few days, and to the galvanic current in from three to six weeks. Before the reaction to galvanism is lost it becomes altered in such a way that the reaction to the kathodic closing current is no longer, as in health, more marked than that to the anodic closing current, but the anodic closing contraction first becomes equal to, then greater than, the kathodic. The changes in the skin have already been alluded to, and will be discussed at length later.

The process of regeneration under favourable conditions begins in the axis-cylinders of the central divided end. Their divided

extremities enlarge and branch longitudinally, and the resulting fine fibres penetrate the granulation tissue, which is forming at the seat of the injury as it does in other conditions. If the distal end of the nerve is sufficiently near, the growing axis-cylinders penetrate into it, some of them finding their way into the old axis-cylinders. The regeneration of the sheaths and of myelin is the next event, and the process is completed by the new fibres acquiring connections with the muscular and sensory nerve-endings, events which are marked by a more or less complete recovery of the muscles and of the sensory function. Most important questions arise in cases where a nerve trunk has been divided. Will the functions of the nerve be restored? In many cases after the clean division of a nerve trunk an immediate return of sensation in the skin supplied by the nerve in question has been recorded. Discounting a good number of cases as errors of observation, and explaining others as due to anastomosis of nerves, and others again to abnormal nerve distribution, there remain cases in which there is immediate return of sensation followed by its disappearance, and in a goodly proportion of cases its return again later on when the divided ends have been sutured together. Thus one must admit that the conductive continuity may be *temporarily* restored. This is probably due to the substance of the divided axis-cylinders on each side of the division regaining contact, and for a time function, which is lost again very soon when degeneration begins. The phenomenon may be compared to what has been termed immediate repair of wounds (see p. 28).

When the ends of a nerve that has been completely divided are brought together at once by sutures,[1] the proceeding is termed *primary nerve-suture*. In more than one in three of such cases the operation has been completely successful, and in nearly another third of the cases partial return of function has been obtained. In such cases the sense of touch returns before that of pain and temperature, and these before the power of motion. On an average, function begins to return in eight weeks' time,

[1] Nerves are best sutured by round-pointed needles and with sterilized catgut. The sutures are passed through the substance of the nerve (direct sutures) and also through the sheath (paraneural sutures).

but it has occurred as early as a few days and has been delayed as long as eight months or longer (see Bowlby, *Injuries and Diseases of Nerves*). If the division of the nerve has occurred sufficiently long before the ends are reunited for degenerative changes to have been begun or completed in the distal end, the operation is termed *Secondary Suture*. The results of secondary suture have been even slightly more successful than those of primary suture. The return of function is slower than after primary suture, sensation returning on an average three and a half months after the operation and motion in eight months. Success has been obtained even where the injury occurred as long as fourteen years before the operation was done. The regenerative property in nerves is well shown by the return of sensation in grafts of skin that have been transplanted from one part of the body to another.

Nerve Grafting. In cases of secondary suture it is sometimes found that the ends are separated so widely that they cannot be approximated. In such circumstances portions of nerves excised from freshly amputated limbs, and in one case from a rabbit, have been used as grafts. Cases in which this proceeding has been successful have been recorded.

Amputation Stumps. In an amputation all the structures, the repair of which has been considered, are divided. If the wound is kept free from pyogenic germs, and there is no abnormal blood-condition present, healing takes place just as in other wounds. There are a few special features which require mention.

Muscle. The muscular tissue of an old (two years or more) amputation stump is found to have undergone complete atrophy, its place being taken by fibrous and fatty tissue. The atrophy is due to disuse, in some cases aided by pressure.

Nerves. The ends of the divided nerves are found to have undergone a bulb-like enlargement at their extremities (Fig. 18). This enlargement consists of medullated nerve fibres coursing in all directions; they must be regarded as an effect of attempted repair. When the growing axis-cylinders from the proximal end of a divided nerve fail to find the distal end to guide them to the terminal organs, whether sensory or motor, they and

the new sheaths around them form these swellings, which have been wrongly termed neuromata as if they were tumours. They are the result of the same regeneration which, under more favourable conditions, ends in restoring function.[1]

The Arteries are obliterated from their ligatured or twisted extremities up to the first branch (Fig. 18). The previous part of the artery undergoes diminution in size proportionate to the smaller amount of tissue it has to supply. Thus the axillary artery in Fig. 18 has dwindled to the size of the radial.

The Bone. When the divided end of the bone is used to bear the weight of the body, as after amputation through the lower extremity of the femur or the tibia, it retains its original size or even becomes thicker and more compact; on the contrary, when the end of

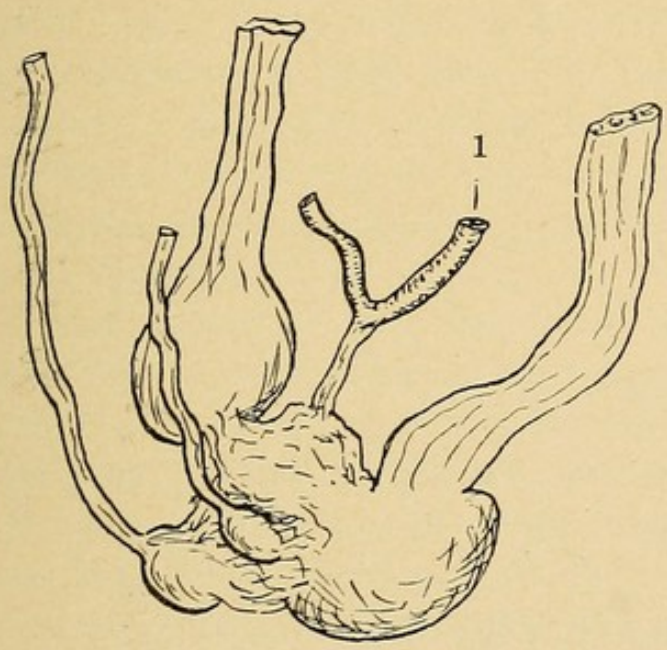


Fig. 18.—The nerves of the brachial plexus and the axillary artery (1). Removed from the stump of a shoulder amputation. The artery has dwindled to the size of a radial artery. (575.)

Fig. 19.—The upper half of a femur removed after death, sixteen years after an amputation done in childhood. The lower end of the bone is pointed and tapering from atrophy. (572.)

[1] These bulbous nerves of stumps as a rule are painless. When they are pressed on they give rise to "pins and needles," just as does the normal ulnar nerve at the back of the humerus. Occasionally troublesome neuralgia occurs. This is sometimes due to the nerve being involved in dense cicatricial tissue. In other cases it is only part of a general neuralgic tendency on the part of the patient.

the bone is not used in this way, like the muscles that clothe it, it undergoes atrophy, assuming the tapering appearance shown in Fig. 19.

Conical Stump. In young subjects the longitudinal growth of a bone in some cases takes place in excess of that of the soft parts. Thus it comes to project against the scar which is pushed forward and stretched over the end of the bone. This form of conical stump the surgeon is powerless to prevent: happily it is easily remedied by removing a portion of the bone. Other causes of conical stump are: (1) Cutting flaps too short; (2) excessive shrinking of flaps infiltrated by inflammatory exudation; (3) sloughing of flaps.

CHAPTER III.

FACTORS WHICH HINDER REPAIR. INFLAMMATION.

When repair fails or follows an abnormal course, this is usually due to the presence in the wound of pyogenic fungi. The recognition of this fact, and the adoption of practical measures to prevent the entry of living organisms into wounds, constitute the basis of Listerism, *i.e.* antiseptic or aseptic surgery. Whilst this factor should always be kept in mind, others must not be forgotten.

A good state of nutrition is as necessary to cells as to individuals. Doubtless many individuals inherit an inadequacy of vitality in certain tissues or in the body generally. Such inadequacy implies a lessened resistance to the attacks of pathogenic organisms, and a diminished activity of repair. More frequently the vigour of the tissues suffers from the disease of some important organ like the kidney, or from ill-chosen diet and insanitary surroundings, or to some disease such as syphilis. Formerly the subjects of lowered vitality were vaguely designated as "strumous." When the nutrient medium of the body—the blood—is deficient in amount, or in the number of red corpuscles or other constituents, repair is necessarily delayed. In other words, anæmia delays repair.

In *scurvy*, a disease marked by the occurrence of hæmorrhages into the tissues owing to a condition of malnutrition of the small blood-vessels, repair is greatly hindered, so that, for instance, a fractured bone may remain ununited as long as the disease lasts.

When the blood contains materials detrimental to cell-growth

the process of repair is slowed. In diabetes it is sugar, in gout uric acid; in kidney disease there is excess of urea and other substances, which are not excreted by the kidneys as rapidly as they should be. And in such conditions not only is repair delayed, but the defence against micro-organisms is weakened, because the phagocytic and other protective properties of the blood and tissues are impaired, and thus a second factor hostile to repair is added. In another condition, leucocythæmia, the blood is deficient in red, and loaded with excess of white corpuscles and abnormal cells, which accumulate in the injured part and prevent the formation of granulation tissue and other material of repair. These are a few *general conditions* which make against normal healing. *Local conditions*, such as the important one, asepsis, alluded to above, are often within the surgeon's control, and are of greatest practical importance. In considering fractures, the importance of proper rest to the injured parts has been already alluded to. Rest is as important to the proper union of wounds of the skin and other parts as it is to fractured bones. It is to be secured by careful stitching and bandaging, and in wounds about the anus, by stretching the sphincters in order to render them inactive for a time.

Effects of Venous Congestion. If the return of blood by the veins is obstructed, the small veins and capillaries become over-distended with blood. This is termed passive hyperæmia. When the condition is severe the altered walls of the vessels allow serum and some blood-cells to escape into the tissues: in other words, *œdema* (Gr. *oidema* = swelling) supervenes. In respiratory and cardiac affections such a condition is general, *i.e.* affects the whole of the body; but frequently in surgery it is a local state, as in varicose veins of the leg, or when bandages are too tightly placed about a limb above a fracture. In œdema the tissues are bathed in an excess of serum, and their condition is to some extent like that of the inner part of the sheath of a tendon after tenotomy (see p. 20). The connective-tissue cells are surrounded by an excessive amount of nutrient fluid; they enlarge and multiply, forming cicatricial tissue, the production of which continues so long as œdema lasts. In such a condition a superficial wound either increases

in extent or remains stationary, the continuous discharge of coagulable material, or of pus, preventing the epithelium from growing over the denuded surface. The ossification of the callus of a fractured bone is delayed, or even prevented, by the progressive formation of granulation tissue, fibrous union being the usual result.

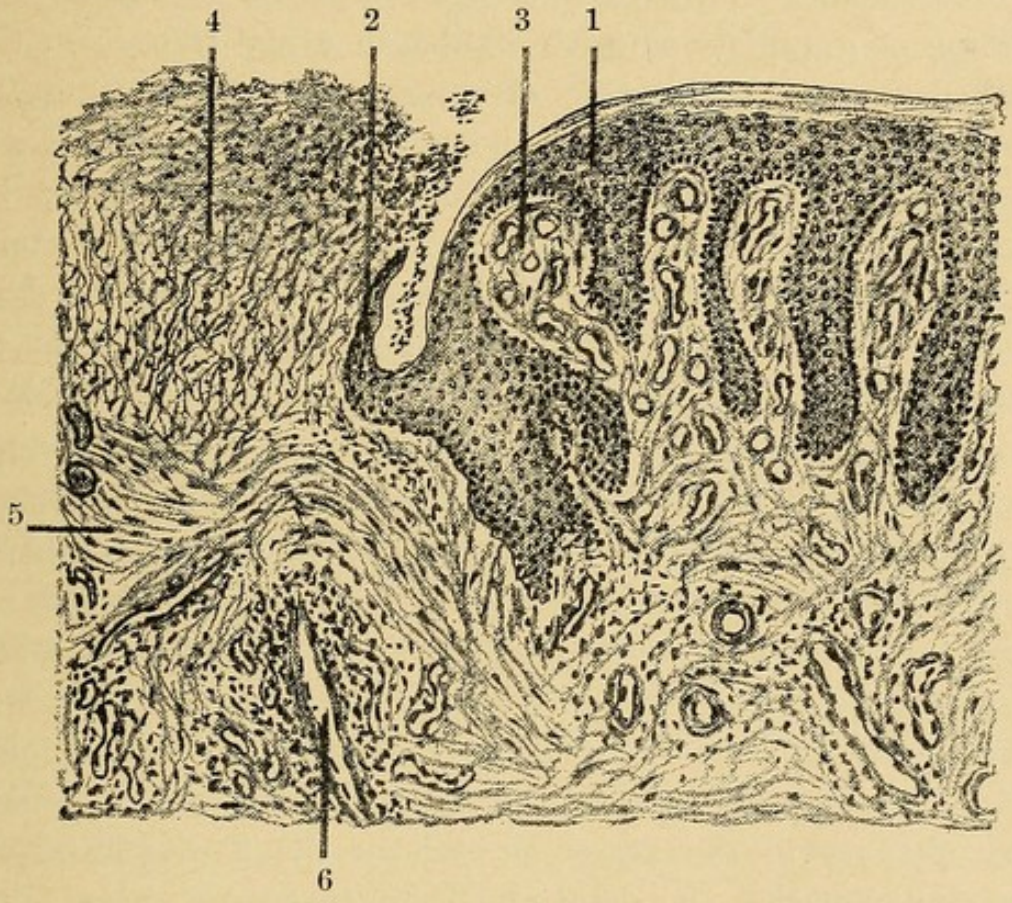


FIG. 20.—A section of the margin of a chronic (Gr. *chronos*=long-lasting, time) ulcer of the leg. 1, Epidermis; the interpapillary processes of which are greatly increased in size. 3, One of the papillæ of the corium also greatly enlarged from the formation of new capillaries and fibro-cellular tissue. 2, Thin edge of epidermis turned back by (4) the coagulated discharge which contains many pus cells. 5, Granulation and scar tissue forming the floor of the ulcer. 6, A dilated vein surrounded by leucocytes which have escaped through its walls. From the leg of an old woman who had varicose veins.

The practical indications to be gathered from the consequences of passive hyperæmia are, to assist the venous return by a horizontal position of the injured part, and in suitable cases by gentle massage.

A breach of an epithelium-covered surface, such as the skin or intestine, etc., which is not healing, is termed an ulcer, and, to illustrate the following remarks, the structure of a chronic ulcer of the leg, due to varicose veins, may be briefly con-

sidered, since it illustrates the various anatomical features of inflammation, as well as the effects of chronic venous congestion (see Fig. 20).

Inflammation.

In considering the effects of passive hyperæmia, two distinct sets of phenomena have been mentioned. One of these is the formation of granulation tissue similar in every respect to that described under the heading of "Regeneration of Connective Tissue." In the chronic ulcer, however, the formation of new tissue is not limited to what was required for repair, but extends to uninjured parts, and continues as long as the abnormality of the circulation. The second process included an altered condition of the circulation, and the escape from the blood-vessels of part of the liquid menstruum and of blood-corpuscles, or, as it is termed, diapedesis (Gr. *dia* = through, *pedesis* = leaping). In varying proportions these same phenomena —(1) changes in the blood-vessels, (2) exudation of liquid from them, (3) diapedesis of blood-corpuscles, and (4) hyperplasia of connective-tissue cells — are combined in the various grades of inflammation. The more acute the process, the more marked are the first three factors as compared with the last, which becomes prominent in the more chronic cases.

The changes in the vessel as seen in the frog's mesentery, when inflammation is determined by exposure to air and mechanical injury in manipulating the part, are: (1) Dilatation of the arteries, capillaries, and veins, and consequent increase of the rapidity of the circulation—active hyperæmia; (2) adhesion of the white blood-corpuscles to walls of the vessels and to each other, and consequent narrowing of the channel and retardation of the current, or even absolute arrest of the flow (stasis); (3) diapedesis. The latter may be inferred from the white corpuscles seen ranged outside the capillaries, and with patience the actual exit of a corpuscle can be followed. The liquid exudation is familiarly observed in the weeping of an acute eczema or the filling of the cavity of an inflamed joint. The classical symptoms—redness, swelling, heat, and pain are accounted for by these processes, pain being due to irritation

of the nerves by the altered pressure and other conditions. The sensation of heat is chiefly due to the increased flow of blood through the affected tissues. In some forms of inflammation, *e.g.* tubercular and syphilitic, these two of the cardinal symptoms may be but slightly marked, and at the present time it is impossible to restrict the term inflammation to conditions in which all the four signs are encountered.

Nor is it possible to regard inflammation as a disease. To a large extent this complex process is protective. In infective inflammation the multiplication of the connective-tissue cells and the escape of leucocytes, for instance, afford a supply of phagocytes which are very frequently able to destroy the active cause of the disturbance or to effect its elimination from the body by the formation and bursting of an abscess; and when the cause has ceased to act, the new-formed connective tissue serves also to repair any loss of substance that has taken place. For the surgeon the important points to be realized are the causes of inflammation and the pathological conditions to which the process gives rise. Among the causes pathogenic bacteria are by far the most important; among the consequences abscess, ulcer, and necrosis are familiar examples. Rheumatism, gout, rickets, and other conditions marked by inflammatory processes are not known to be caused by parasites, and in the case of the two latter diseases are probably not of parasitic origin, yet their effects are of importance to the surgeon as to the physician. It will be well to discuss here the surgical aspects of infection, and to consider the more important surgical effects of inflammatory processes not known to be infective in connection with the bones and joints which they chiefly concern.

Abscess.

In infective inflammation there are present toxic materials which in some cases determine such a degree of diapedesis that the leucocytes collect in great numbers in spaces in the tissues made partly by distension of natural lymph spaces, partly by digestion of the tissues. These accumulations of leucocytes with the fluid around them constitute *pus*, and the cavity in which

the pus lies is an *abscess*. The leucocytes of pus are for the most part dead and in a state of fatty degeneration. It will be seen that an abscess is practically the same as an ulcer, save that the former is closed in on all sides whilst an ulcer is exposed on a surface. The property of attracting leucocytes possessed by certain chemical substances, whether secreted by bacteria or, like croton oil, etc., of other origin, is termed *chemiotoxis*. This phenomenon has been observed in certain organisms such as the myxomycetes, which have been seen to be attracted by vegetable infusions and repelled by saline solutions. The latter phenomenon is termed *negative chemiotoxis*. The good effect of *drainage* and *irrigation* of abscesses, etc., is attributable to the removal of toxic materials from the tissues. To a certain extent pus may be absorbed, as is sometimes observed in the anterior chamber of the eye.

Abscesses, when caused by certain pyogenic bacteria, form and extend rapidly and are termed *acute*; when caused by slow-growing bacteria like the tubercle bacillus they form slowly and are termed *chronic*. The extension of an abscess is effected by removal and liquefaction of the abscess wall, which corresponds to the floor of an ulcer. Several factors co-operate in this process. The tissue cells and leucocytes digest the tissue, the tension of the contents of the abscess causes pressure-atrophy, and ferments secreted by the bacteria liquefy the tissues. If the latter perish too rapidly for liquefaction to keep pace with necrosis the abscess wall is *sloughy*. *Tension* in an abscess is produced by several factors, among which are: (1) The biological force of the emigrating leucocytes; (2) the osmotic force of the liquid exudation, and (3) the dilatation of blood-vessels in the abscess wall. The "pointing" of an abscess, *i.e.* its extension to a surface of the body, is indicated by œdema, redness, etc., and finally (when the tension is not too great) by "fluctuation"—a sensation given to the fingers like that of water in an india-rubber bag. At last the skin or mucous membrane covering the summit of the abscess gives way and some of the contents escape. The opening thus made is small and frequently the diminution of tension is not sufficient to allow healthy granulation tissue to form and bring about healing: pus

continues to escape from the cavity by a track or *sinus* in the tissues. The indications for obtaining healing of an abscess are to make a free opening, and to prevent its closing by the use of the drainage-tube or by packing with iodoform gauze; and to destroy micro-organisms by carbolic lotion, etc. Where a tubercular abscess, such as those met with in connection with the vertebræ, is of large extent, it is advisable after evacuating the contents to mop out the cavity with some strong antiseptic and then to close the external wound by sutures, the risk of re-formation of the abscess being less than that of adding pyogenic and putrefactive bacteria to the tubercular infection.

Ulceration, etc.

An ulcer is formed in exactly the same way as an abscess, *i.e.* suppurative inflammation causes a loss of tissue. Since, however, the loss is on a free surface, the result is not a cavity filled with pus, but an open breach of surface from which pus escapes. Loss of tissue caused by new growths invading a surface also forms an ulcer. When an ulcer begins to heal it is termed a *granulating wound*.

In order that healing by granulation tissue may progress in a normal manner, the various infections and other factors which hinder repair must be absent or kept in abeyance. Where it is impossible to render a cavity or surface aseptic, free drainage and, in suitable cases, the use of iodoform, preferably in the form of a glycerine suspension, often secures rapid healing. Glycerine is hygroscopic, and hence promotes exosmosis. That iodoform is not a powerful antiseptic is shown by the fact that, in a one per cent. suspension in nutrient gelatine it does not prevent the growth of anthrax bacilli. Iodoform acts on the toxic products of bacteria, rendering them harmless, and so enabling repair to go on.

In septic wounds granulations are apt to become over-developed—luxuriant, as it is termed, and the resulting scar is thick and often prominent, contrasting with the smooth scar that results in aseptic conditions.

Wounds may be attacked by tubercle, syphilis, etc., and these diseases cause a more serious obstacle to repair than do the ordinary pyogenic bacteria.

The various parts of an ulcer which are to be studied are: (1) the outline and size, (2) the floor and discharge, (3) the surrounding tissues. According to the characters of these and other features, ulcers may be variously designated. The edge may have a *sinuous* outline; if the peripheral extension of the ulcer is a marked feature, the term *serpiginous* is applied to it. Where the edge and base of the ulcer is thickened by the formation of much fibrous tissue, the ulcer is termed *callous*; if there is much pain from irritation of nerves in the ulcer, it is termed *irritable*. When the granulations are large and pale from œdema, the ulcer is said to be *weak*; and when the surrounding tissues are inflamed, the term *inflamed ulcer* may be used. In the following chapters many specifically different forms of ulceration will be described, *e.g.* leprous, cancerous, etc., etc.

Scars may be "weak," that is, liable to break down into ulcers at various points. This occurs especially in fat persons with weak circulation. The prominent scar resulting from septic wounds may assume a thickened appearance so marked that the terms "hypertrophic" or "warty" may be applied to it. In some cases, instead of the scar-tissue undergoing the usual atrophic shrinking, there may be a progressive formation of fibrous tissue—in other words, the formation of a new growth, which is termed *Keloid* (see the section on "Diseases of the Skin"). Chronic ulcers and scars are not infrequently the starting-point for cancerous growths.

Diffuse Suppuration.

When the infective organisms which give rise to suppuration are more than usually virulent, the effect is not limited to a definite area, but spreads in a diffuse manner, so that instead of an abscess or an ulcer the result is a diffuse suppuration.

CHAPTER IV.

INFECTIVE INFLAMMATION.

BACTERIA or fission fungi are the best-known agents of infective inflammation. The most certain proof of a micro-organism being the cause of a disease is obtained when it is found to answer to Koch's "postulates," which are: (1) That the organism is constantly present in every instance of the disease; (2) that it has been cultivated free from other organisms on nutrient media through several generations; (3) that when, after being thus cultivated, it is inoculated in animals it reproduces the original disease, and (4) that cultures of the organism are again obtained from the inoculated animals.

It would, however, be a mistake to think that this is the only mode of establishing the causal relationship between a micro-organism and a disease. Thus a protozoon has been proved to be the cause of malarial fever, though as yet no cultures of the parasite have been obtained. Its relation to the disease has been ascertained by observing the parasite in the blood of persons suffering from the disease, and noting the coincidence between the phases of the fever and those of the parasites in the blood. The conclusion has been arrived at on grounds similar to, if more refined than, those that enable us to conclude that the acarus scabiei is the cause of itch. The claims of any micro-organism to pathogenic properties must, however, be most critically examined in order to prevent false doctrine being accepted to the detriment of knowledge. The effects of bacteria are produced chiefly by poisonous substances formed by them. These substances vary in their chemical

characters. Many of the putrefactive bacteria produce *ptomaines* (Gr. *ptoma* = corpse), which have the characters of alkaloids. Ptomaines, when they are of a poisonous character, are termed *toxins* (Gr. *toxicon* = for the bow = arrow-poison). Others produce poisonous proteid bodies; when these are coagulable by heat they are termed *toxalbumins*, as, for instance, the poisonous product of the diphtheria bacillus; when not coagulable by heat the products are termed *proteins*. If the general symptoms of a disease are produced by the absorption into the blood of the poisonous products unaccompanied by the living organisms the condition is termed *septic intoxication* (Gr. *sepsis* = fermentation or decay). If living organisms (either by the lymphatics or the veins) gain an entry into the blood-vessels and multiply there and in the tissues, the condition is one of general infection, or septicæmia, one phase of which, marked by the formation of abscesses, is called pyæmia. Alcoholic intoxication affords an easy parallel with septic intoxication. An unicellular fungus, the saccharomyces, closely allied to the bacteria, produces by its vital action on sugar a toxic substance, ethylic alcohol. The latter, separated by various means from the fungus which generates it, is capable of producing the well-known effects on the central and peripheral nerves and other tissues and organs of the body. So in diphtheria, a bacillus growing on a surface where the *local* disease is in progress produces a toxalbumin which, absorbed into the blood, constitutes a septic intoxication, the evidences of which are fever, depression, albuminuria, paralysis of nerves, etc. Some bacteria produce ferments or enzymes, among which are several capable of splitting up proteids. Very minute quantities of such ferments absorbed into the blood may cause serious effects. As a marked example of septicæmia the effects of inoculating a guinea-pig with anthrax bacilli may be adduced. If living spores of this organism are placed beneath the skin in the track of a minute puncture, twenty-four hours later the animal is dead or dying. The blood in the heart and in the vessels of all parts of the body is crowded with bacilli.

From a practical point of view it is of the highest importance to prevent the occurrence of a local septic infection,

and, if local infection has occurred, to prevent it becoming a general infection or septicæmia. For instance, an acute abscess, say, in a cervical gland, is a local infection producing fever and other symptoms by septic intoxication. The abscess opened and drained, the symptoms rapidly disappear. But should septicæmia have occurred before the original abscess is treated the operation may produce but little effect; fever, rigors, etc., will continue because the organisms are distributed by the blood-stream to various parts of the body, or, in other words, the condition is one of general infection.

Immunity. The rapid generalization of anthrax bacilli in the guinea-pig has been alluded to above. We may compare it with what occurs in man when some of the same spores find their way into a scratch in the skin. Local inflammation is the first effect, showing itself as a red papule which itches violently, and on which a vesicle forms. A raised and indurated area forms around the vesicle, and by the end of the second day becomes black in the centre; meanwhile a circle of new vesicles arise around the black area, and the surrounding tissues become œdematous. There is some fever from septic intoxication. If the whole of the black (necrosed) area and that bearing the vesicles is freely excised the patient recovers, if this is not done the patient dies of septicæmia within a week of the commencement, and anthrax bacilli are to be found in the blood of all parts of the body. Allowing for the slightly different conditions of the two cases, it is seen that the anthrax bacilli are less rapidly destructive to man than to the guinea-pig. This may be explained by saying that in man the connective-tissue cells and the diapedesis of leucocytes, etc., hold back for a time the bacilli from entering the general circulation by the lymphatics and veins, whilst in the guinea-pig there is little or no power on the part of the connective-tissue cells and leucocytes to limit the invasion of the blood by the bacilli. As compared with the guinea pig, man is said to be better *protected* against the organisms. The amount of protection seems to be proportionate to the degree of inflammatory reaction set up. If any species of micro-organism is found to be incapable of growth in the tissues of an individual then the protection is said to be

complete, or the individual is said to enjoy *immunity* against the disease. Similarly, when an individual is not affected by the chemical product of any organism, he is immune to that substance, which may be toxic as regards other individuals. Immunity may be acquired, *e.g.* one attack of any of the specific fevers usually protects a person from that disease, or it may be inherited. The difference between inoculated anthrax in the guinea-pig and man respectively supports Metschnikoff's theory to the effect that immunity depends on the capacity of the phagocytes of an individual to destroy any given organism. The view is supported by various observations, *e.g.* under ordinary conditions a frog is immune against anthrax. If bacilli are injected into a frog's peritoneal cavity they are seized on by leucocytes and destroyed. If, however, the frog be kept at an elevated temperature or anæsthetized with chloroform and ether mixture, the bacilli multiply and little or no phagocytosis is observed. The altered condition of the frog in relation to anthrax bacilli shows that the same individual may be at one time immune against a pathogenic organism and not at another time. This variability of the host in relation to the parasite is complicated by varying virulence of the micro-organisms. Bacteria grown under favourable conditions of nutrient medium and temperature are more active (virulent) than those grown under adverse conditions, when the organisms are of diminished pathogenic potency and are then said to be *attenuated*. In thinking of micro-organisms in relation to disease, the modifications due to variations of protection in the host and of virulence in the parasite must not be lost sight of.

Animals may be made proof against the effects of the toxic products of bacteria. Thus a horse may be immunized against the diphtheria toxalbumin by administering gradually increasing doses by hypodermic injection. At first the animal suffers: local swelling, general fever, and malaise being observed, but afterwards even what in an unprotected animal would be a fatal dose is found to have no effect. When this stage is reached the serum of the blood of the artificially protected animal is found to neutralize the diphtheria toxalbumin when added to the latter outside the body, mixtures being inert when ad-

ministered to unprotected animals. This antitoxic property of the serum of animals rendered proof against diphtheria has been found to be effectual also when the poison has been injected alone into an unprotected animal and has given rise to symptoms. If then, after an interval, the antitoxic serum is injected, the symptoms of septic intoxication rapidly disappear. These facts have been used with some measure of success in the treatment of diphtheria. The blood-serum of protected horses, etc., injected into persons suffering from the disease has been found to diminish the symptoms of intoxication and to help in effecting a cure. A similar antitoxic serum has been found to annul the effects of snake-poison. In tetanus the results of a similar line of treatment have not been so successful. An antistreptococcic serum is now on its trial. These phenomena go to show that there are other factors besides phagocytosis in the production of immunity.

Fever.

The thermometer is of the greatest value in detecting the presence of septic inflammation. If the initial dose of poison is marked there is frequently a rigor, or short of this a sudden rise of temperature above the normal. The fever is probably the result of the action of the toxic material on the central nervous mechanism which regulates the temperature of the body, and on the tissues generally. There is an increased production of heat by the tissues of the body. After death the cells of the organs of the bodies of those who have died during this septic fever are found to be in a state called *cloudy-swelling* (see p. 16). The blood in suppurative fever is *hyperinotic*, that is, it clots with abnormal readiness. Thus on post-mortem examination of the bodies of those who have died from infective disease ante-mortem clots are usually present in the right ventricle of the heart.

Traumatic Fever. After severe injuries when the initial depression of the activity of the nerve centres or *shock* (see p. 15) has passed away, there is frequently observed a rise of temperature which may last for several days, and which is not necessarily

connected with sepsis. The two chief factors in the production of simple traumatic fever are reflex nerve irritation, and the reabsorption of the products of aseptic inflammation—leucocytes, serum, etc., into the blood. The sharp rise of temperature, which may be accompanied by a rigor, often observed after the passage of a catheter through a stricture of the urethra, is attributed to reflex nerve influence. This is one form of *Urethral fever.*

Suppuration.

Suppuration when not the result of artificial irritants like croton oil, etc., is due to infection by pyogenic organisms.

FIG. 21.—1 Staphycocci and marrow cells from a cover-glass preparation of inflamed marrow from a case of acute osteomyelitis. 2, Gonococci and leucocytes, from a cover-glass preparation of urethral discharge. 3, Streptococci and tubercle bacilli with pus cells, from a cover-glass preparation from the discharge in a case of tubercular inflammation of the middle ear. 4, A bunch of the clubs of actinomyces, from an unstained scraping from a nodule in the skin of the face of a woman. 5, Anthrax bacilli and red blood-cells. 6, A granular wandering cell simulating a collection of micrococci, from a case of erysipelas. The preparations were drawn under a $\frac{1}{12}$-in. oil-immersion lens. × 1000 diams.

Several different bacteria are pyogenic, *e.g.* streptococcus pyogenes, staphylococcus pyogenes aureus, diplococcus pneumoniæ,

gonococcus, bacterium coli, etc. The first two are the most commonly met with in pus. The streptococcus pyogenes as it occurs in ordinary acute abscesses is indistinguishable from the streptococcus erysipelatis in its morphological and biological properties. It is thus doubtful whether erysipelas is more than a special phase of the same infection that gives rise to cellulitis, etc.

Pyogenic organisms are frequently accompanied by the ordinary putrefactive or other bacteria, such as the bacillus which gives a blue or green colour to the matter in some abscesses.

Diseases caused by Streptococci. *Cutaneous Erysipelas* commonly starts from a small scratch or abrasion of the skin or mucous membrane, occasionally in a wound or ulcer that has been suppurating for some days, and frequently in chronic syphilitic lesions. Sometimes it begins in apparently sound skin. The rash begins as a bright red discoloration of a patch of skin bounded by a definite margin slightly raised from œdema, which is most marked in lax tissues such as the eyelids. The patch extends by advance of the margin, so that it may cover an extensive area of skin and extend to mucous membranes. Vesicles may form at first with clear then with turbid contents. There is smarting and burning pain. The corresponding lymphatic vessels and glands become tender. In children the skin may undergo necrosis. There is marked fever with chills, and sometimes a rigor. Headache, nausea with disturbed digestion, and in bad cases delirium are also among the general symptoms. The disease lasts from a few days to two or three weeks, and may relapse. During recovery the colour fades, the cuticle desquamates, and the hairs fall out of the area affected. In new-born infants the disease often starts at the navel and involves the whole body, and is usually fatal. Sections of the skin at the advancing margin show the lymphatic vessels to be crowded with streptococci. In the central part of the area the tissues are crowded with leucocytes, but there are no micrococci.

Cellulo-cutaneous Erysipelas begins in the same conditions as cutaneous erysipelas, but is marked by a greater amount of

œdema, causing the affected skin to be at first puffy and, later, tense. Vesications may appear. The condition may begin to subside at the end of the first week, or diffuse suppuration and sloughing of the subcutaneous tissue and general infection may result. The general symptoms are the same as those of cutaneous erysipelas. The microscope shows that streptococci are present in all parts of the affected area, and are not confined to the advancing edge as in cutaneous erysipelas.

Cellular Erysipelas or Cellulitis is due to the inoculation of the subcutaneous tissue, as in septic wounds, or from punctures such as the stings of insects. The condition differs from cellulo-cutaneous erysipelas only in that the tissue affected is more lax, the suppurative effusion being more free to travel, and the corium being only secondarily affected, so that the reddening of the skin is not so marked as it is in erysipelas, where the corium is primarily invaded by streptococci. The general condition of the patient is the same as in other streptococcic inflammations. Sloughing, pyæmia, etc., are common events.

Paronychia or Whitlow is a phase of streptococcic cellulitis of the skin at the side of the nail. The suppuration may be limited to a small area, matter collecting beneath the thick epidermis or the nail (sub-epithelial whitlow); or it may extend more widely in the subcutaneous tissue of the finger (subcutaneous whitlow); or it may extend to the periosteum of the terminal phalanx, causing the intense throbbing pain which is due to the tension caused by inflammatory exudation taking place in an inelastic tissue (periosteal whitlow). The suppuration may extend to the synovial cavity of the sheath of the flexor tendons, causing *thecal abscess*, which in the case of the thumb and little finger is likely to extend to the palmar bursa.

Some Diseases due to Staphylococci.—*Boils.* A boil begins as a small red papule formed round the root of a hair with itching and smarting. The papule increases in size, and its base extends into the subcutaneous tissue, causing much throbbing pain. The boil may abort at this stage, in which case it is termed blind. More usually a vesicle forms on its apex. The vesicle becomes a pustule, and suppuration extends

to the whole of the affected part, which includes the entire thickness of the corium with some of the subcutaneous tissue. The pustule ruptures, exposing a slough which becomes detached, leaving a small cavity which heals and leaves a scar. The neighbouring lymphatic glands are swollen, but rarely suppurate. Fresh boils are apt to appear owing to local infection from the original lesion.

Carbuncle. This lesion begins as a raised flat or slightly conical inflamed area of skin with a clearly defined border. The lesion varies in extent from that of a halfpenny piece to that of the palm. There is much burning and throbbing pain as the infiltration extends from the corium to the subcutaneous tissue and the swelling increases in size. The colour becomes purplish, and vesicles which are converted to pustules form and open, the discharging orifices, revealing a tough slough of skin and subcutaneous tissue which slowly separates. Carbuncles are commonest on the thick skin at the back of the neck and trunk. There is fever with depression, and fatal general infection is not an uncommon event, especially in diabetic subjects who are prone to this affection.

Contagious Impetigo is one of the commonest staphylococcic infections. Small superficial pustules form between the layers of the epidermis often at the site of punctures of the *pediculus capitis* in unhealthy children. Scabs are formed by the rupture of the pustules. Secondary inflammation of glands, accompanied by slight fever, is common; secondary glandular abscesses are not uncommon.

Other Suppurative Affections.

The Pneumococcus (diplococcus of Fränkel), the micro-organism which is the immediate cause of acute pneumonia, pleurisy, etc., sometimes determines acute suppuration in the middle ear, and also purulent meningitis, but in neither of these conditions is it as commonly the casual agent as is the staphylococcus or streptococcus. Pneumococci sometimes cause ulcerative endocarditis, which will be discussed farther under Septicæmia.

Gonorrhœa. The organism which causes the purulent discharge of gonorrhœa is a diplococcus which is found in clusters in the leucocytes and epithelial cells of the discharge, as well as free in the liquid portion of the discharge. Non-pathogenic diplococci exist in the normal urethra, but clusters of paired cocci within leucocytes and epithelial cells are characteristic of the gonococcus, which moreover is decolorized by Gram's method of staining. An attack of gonorrhœa in the male urethra if left to itself lasts about seven weeks if no complications arise. For two or three days after infection there are no symptoms, then for another day or two a little itching and slight excess of mucus is noticed about the meatus urinarius. The acute inflammatory stage follows, and lasts two weeks or so. It is marked by a profuse discharge accompanied by pain and swelling in the urethra, first at the anterior part, and extending afterwards to the bulbous part. Sometimes the prostatic portion becomes involved, deep-seated pain and frequent painful micturition being the result. There is usually a certain amount of fever and general disturbance during this stage, which passes gradually into the third or subacute stage in which the pain and discharge gradually diminish, and in favourable cases cease in about the seventh week from infection.

Septic Intoxication, due to the absorption of the poisonous products of bacteria from wounds or cavities such as the peritoneum, only differs from the effects of infection in the mode of entry of the poison into the blood and not in the nature of the poison, so that there is no reason why the symptoms of septic intoxication should differ essentially from those of septic infection. The term *acute septic intoxication* used to be reserved for a group of cases in which dangerous and usually rapidly fatal symptoms supervened about the third or fourth day after a surgical operation. High fever, flushed, hot skin, dry, brown tongue, vomiting, delirium, dyspnœa, and death, were among the symptoms, and doubtless by the time death occurred septic infection had in many cases supervened. Such cases have been all but abolished by antiseptics; they may still occur from neglected wounds or septic processes in the peritoneum. This is the only condition which is known

clinically as acute septic intoxication. Septic intoxication, in the broader sense of the word, occurs in every acute local suppuration, and a variety of other conditions. For instance, the fever and disturbance observed in a case of a local abscess, or of tonsillitis, etc., is due to the absorption of septic products into the lymphatics, and so into the blood.

General Septic Infection or Acute Septicæmia. A severe rigor marks the onset of fever, and is followed by the symptoms already enumerated under the head of acute septic intoxication. Death may occur in a day or two, usually before a week. After death, besides the changes usual in fever, small hæmorrhages (petechiæ) are found on the serous membranes, sometimes on the skin and in the substance of the solid organs; and intense broncho-pneumonic changes in the lungs. Bacteriological and microscopic examination generally shows streptococci or staphylococci in the blood, and blocking of the capillaries and the small veins with micrococci at the seat of the small hæmorrhages.

Pyæmia, originally signifying a disease due to the entry of pus into the blood, designates a phase of general septic infection of less virulence than that which leads to acute septicæmia as just described. The pyococci are distributed by the blood-stream (embolic distribution), which they enter *via* veins or lymphatics, but the duration of the disease allows time for the formation of abscesses at the points where they lodge. If there is an external suppurating wound, the condition which suggested the original idea of pus-infection is present. Since the exact nature of the infection has been known, the number of conditions which are classed under the head of pyæmia has been extended. Ulcerative endocarditis has been termed internal pyæmia. Here pyococci are present on the lesions in the heart, and are in the position which renders their embolic distribution most easy.

The Symptoms of Pyæmia. The infection of the blood is marked by a rigor which is generally repeated at intervals as new abscesses develop. There is continued fever, which is intensified as each rigor occurs, giving rise to a characteristic temperature chart. Sweating, wasting of muscles, pallor, diges-

tive disturbance, and other common effects of fever follow. There is frequently delirium at night, and the heart's action is accelerated. In a few days after the initial rigor, evidence of abscess formation in the joints, subcutaneous tissue, etc., is generally forthcoming.

The distribution of these abscesses, which are termed "secondary" or metastatic, is not difficult to follow. Since the organisms enter as a rule by the lymphatics or veins, they pass to the right heart and thence to the lungs, where many of them are arrested in the pulmonary capillaries, setting up intense inflammation and suppuration in the lobules in which they are arrested. Thus *lobular* or *broncho-pneumonia* is usually a pronounced feature in cases of pyæmia, just as it is in the more rapid infection, septicæmia. If the febrile condition or the presence of micro-organisms should cause clotting in the blood of the right heart, and fragments of this clot lodge in the branches of the pulmonary artery, the circulation ceases in the areas to which the occluded branches are distributed, because these branches form only capillary anastomoses, and the blood-pressure being removed from the branches of the occluded artery, blood flows into the area from the neighbouring capillaries until an equilibrium is attained; blood-stasis, and then, from the breaking down of the capillary walls, blood-extravasation follow. This is a *hæmorrhagic infarct*, and from the presence of micro-organisms it is soon converted into an abscess. Such abscesses frequently open into the pleural cavities, and there cause suppuration (empyæma). Many of the pyococci find their way by the pulmonary veins to the left side of the heart, whence they are distributed to all parts of the body. After the lungs the most common situations for secondary suppuration are the joints, serous cavities like the pericardium and peritoneum, liver, kidneys, spleen, subcutaneous and intermuscular areolar tissue; but no vascular part of the body is exempt. In the corium, patches of reddening (erythema) called "wandering erysipelas" may form and disappear. Streptococci and staphylococci are commonly the cause of the affection, but the other pyogenic organisms are capable, though to a slighter extent, of giving rise to secondary abscesses. Thus

after gonorrhœa has lasted for some three weeks or a month in the urethra, one or two large joints, especially the knee, or a fascia, may become painful and surrounded by œdema. This condition is termed gonorrhœal rheumatism. Sometimes the œdema (serous effusion) is followed by suppuration within the joint, constituting gonorrhœal pyæmia. This pus contains gonococci.

The general resemblance of ordinary pyæmia to acute rheumatism is striking. Fever, sweating, affections of the joints, subcutaneous tissue, and skin, occur in both, only in rheumatism the effusions are serous without any tendency to become purulent, in pyæmia they are purulent from the first. In general gonorrhœal infection as shown above, the affection may assume at first a serous, afterwards a purulent form. The general indications for *treatment* in suppurative conditions may be briefly referred to. The supreme importance of preventive, *i.e.* antiseptic, measures has already been dwelt upon. The value of drainage, irrigation, and continuous baths, where local suppuration has occurred, has also been mentioned. These measures act by removing toxic materials and in relieving tension, and so allowing healthy protecting granulations to form. Granulation tissue, consisting as it does of rapidly-multiplying cells and blood-vessels, is a tissue of considerable vitality. From its possessing no lymphatic vessels it has but little absorbent power. In the treatment of ulcers and other suppurating surfaces, the application should be of sufficient antiseptic power to repress bacterial growth and yet so mild that the growth of granulations is not interfered with. Thus, for ordinary chronic ulcers, an ointment made by dissolving carbolic acid crystals in a little olive oil, and mixing this solution well with vaseline, so that an ointment of 1 in 40 is obtained, is sufficiently strong. When general infection has occurred, the patient's strength must, as far as possible, be kept up by plenty of air, nourishing and stimulating liquid food, as well as by drainage of abscesses, etc., wherever they may form. Iron appears to be of value in many cases of erysipelas. In bad cases anti-streptococcic serum may be tried. Watson Cheyne[1] has used

[1] *Practitioner*, April, 1895.

it as a prophylactic before severe operations on the mouth and pharynx.

Other Infective Diseases. **Malignant pustule** due to infection of the skin by anthrax bacilli has already been described (p. 63); it is important for diagnosis to examine the exudation in the vesicles of a suspected case for bacilli, by making and staining a cover-glass preparation. The large size (5 to 10 × 1 to 1·5 μ) of the bacilli and their square or concave ends, together with their immobility, are characteristic and serve to distinguish them from the thinner, round-ended, motile bacilli of malignant œdema. Infection by anthrax occurs in the lungs (woolsorter's disease) and in the intestine as well as in the skin.

Tetanus is caused by a slender rod-shaped bacillus which occurs in garden earth, manure, etc. It forms terminal spores which give the sporing bacillus a drum-stick form. In persons suffering from wounds infected by the bacillus it has been found in the discharge and in the tissues in the immediate vicinity of the wound, but as yet not in the central nervous system or in the body generally. Tetanus has been found to follow contusions and other injuries which do not entail a breach of the integument. In some cases of tetanus no injury of any kind has been observed. These cases are called idiopathic, but since the infection of tetanus has been found in so slight a lesion as an acne pustule, it is easy to overlook such a source of infection, and thus it is probable that idiopathic is due to the same cause as traumatic tetanus.

Cultures of the bacillus can only be made in the absence of free oxygen, for the organism is anaërobic. The culture contains a poison, tetano-toxin, which, when freed from bacilli and injected into an animal, produces the symptoms of the disease. The symptoms set in from the fourth to the tenth day after infection, which is generally received in a septic wound or the breach of surface left at the umbilicus by separation of the cord in infants. The spasm like the contraction of rigor mortis begins in the muscles of mastication, whence the common name for the disease, "lock-jaw." Thence the spasm spreads to the face, trunk, and limbs, and differs from that caused by strychnia in that the continuous spasm is more severe whilst the paroxysmal

increase of spasm is, perhaps, not so marked. There is pain from the violent muscular contractions, sweating, and generally fever; acute cases end fatally within four or five days. If the patient survives the tenth day there is some chance of recovery.

An antitoxic serum obtained from horses and other animals rendered proof against the disease by gradually increased doses of pure culture of the bacillus has been found to neutralize tetano-toxin when added to it outside the body, and in a few cases the same serum has seemed to influence cases of tetanus for good when injected under the skin of persons suffering from the disease. From a practical point of view the important reflection is that vigorous antisepsis is the best preventive of the disease. It has indeed been found that apart from other organisms, *e.g.* pyococci, pure cultures of the bacillus have little effect when introduced under the skin of animals.

Diphtheria. The membrane characteristic of this disease is formed of coagulated fibrinous exudation infiltrated by leucocytes, and of the necrosed superficial layers of cells belonging to the surface on which it has formed. The specific bacilli are found in the superficial part of the membrane, whilst strepto-cocci are generally present in the deeper part. After tracheotomy has been performed for diphtheria it is often observed that the membrane spreads from the larynx and covers the surface of the wound. Occasionally a wound of the skin becomes the original seat of diphtheritic infection. Doubtless many of the cases described as diphtheria of wounds before the exact nature of the affection was made clear were only sloughy wounds, but in some cases of wound-diphtheria a typical attack of diphtheritic paralysis follows the appearance of membrane in the wound, and proves the nature of the affection. Diphtheria in wounds yields readily to strong antiseptics. For diagnosis, examination of cover-glass preparations and of cultures made on boiled white of egg or other suitable media is helpful.

The liquid filtrate from cultures of diphtheria bacilli contains a toxalbumin which, injected into animals, reproduces the general symptoms of the disease. The blood of horses and other animals rendered proof against this poison by repeated injections of

gradually increasing doses furnishes the antitoxic serum which, though it cannot be said to be curative, in many cases has been found to ameliorate the condition of the patient and help towards recovery.

Emphysematous or Spreading Gangrene begins in a wound very soon (second or third day) after its infliction. There is pain and swelling in the wound, around which the skin becomes first red then purple, and finally black, with bubbles of stinking gas in the meshes of the tissues which have undergone necrosis or gangrene. The inflammation and necrosis spread rapidly, so that a whole limb may soon become gangrenous. Speedy amputation gives the only chance of recovery, and after amputation the process usually recommences in the stump. The cause is a bacillus which occurs naturally in earth. It is motile and gives out a putrid smell and forms gas bubbles. It is distinguished from anthrax bacilli by having rounded or pointed ends and possessing flagella, whence its motility.

Malignant œdema is due to a bacillus which closely resembles the foregoing both in appearance and in pathological effects.

Hydrophobia or Rabies is an infective disease usually acquired by the bite of an animal. It follows in about 16 per cent. of bites received from dogs suffering from the disease. When the bite is made through clothing the risk of infection is not so great as when a naked part receives the injury.

Clinically the chief features of the disease are: (1) A remarkably long period of incubation, usually about six weeks, but sometimes longer. (2) A premonitory stage of two or three days, attended by chills, mental depression, and irritability with exaggerated reflexes. Final stage—(3) repeated violent reflex spasms of the muscles of respiration and deglutition. The spasms are started by noises, lights, etc., or by attempting to drink. There is a condition of extreme mental tension. There is an excessive secretion of viscid saliva.

Occasionally in man and dogs, and usually in rabbits, the disease assumes the form of an ascending paralysis. Dogs may be suspected of rabies when they show a marked change of temper, a sullen and unobservant bearing, an unusual snappishness, especially to other dogs, an alteration in the tone of the

voice, and a tendency to eat their own dung and other filth. The addition of a flow of viscid saliva, a dropping of the lower jaw, an inability to swallow, and repeated paroxysms of excitement with snappishness, makes the diagnosis certain.

The pathological changes are most marked in the medulla, where, though little is visible to the naked eye, the microscope shows changes due to acute inflammation: ante-mortem thrombosis in the small blood-vessels, and extravasation of leucocytes around them. There is also inflammatory infiltration of the salivary glands. The virus has not been isolated. Pasteur found that it is present in the spinal cord of animals that have died of the disease; and, further, that by drying such spinal cords under a bell-jar containing caustic potash the virus is attenuated and, after a fortnight or so, destroyed. By injecting triturations of dried cords of gradually increasing potency under the skin he rendered dogs immune to the disease, and by applying the same principle to human subjects who had been bitten by mad dogs he considerably diminished the rate of mortality. The treatment should be begun as soon as possible after the bite. If delayed after a fortnight it appears to have little effect.

Glanders occurs both as an acute and as a chronic affection in man and animals, and so constitutes a link between the acute infective diseases and the chronic or infective granulomata. In acute glanders malaise, headache, pains in the limbs, and diarrhœa are succeeded by an eruption of papules, which become flat vesicles and then pustules. Abscesses form in the muscles and subcutaneous tissues. Inflammation in the mucous membrane of the nasal fossæ, accompanied by swelling of lymphatic glands and vessels, usually follows, and the death occurs in the second or third week from extension to the lungs. In man the disease is usually acute, and is often mistaken for typhoid or rheumatism. The post-mortem appearances are very like those of pyæmia.

Chronic Glanders is marked by the formation of indolent ulcers with indurated edges like tertiary syphilitic lesions. The hands and face are common sites. The joints may be affected, giving rise to abscesses and fistulæ. The nasal fossæ may suffer, ulcers forming in them. Cough, with sanguineous expectoration and

hoarseness, may occur and are apt to be mistaken for syphilis. In horses the lymphatic vessels and glands of the head and neck, groin and axillæ, swell, become indurated and, frequently, ulcerate. Half the cases recover in about four months, the average duration of the disease. Diagnosis is best established by inoculating a guinea-pig, in which animal a local nodule forms and suppurates: glands then swell, and internal metastases form. Cultures are successful only at incubation-temperature. Stained with alkaline methyl blue or carbolic methyl blue, bacilli as long as and a little wider than those of tubercle are found. Chronic glanders, which is also called *Farcy*, is the form the disease usually takes in horses.

The Infective Granulomata.

This is the name given by Virchow to a group of infective diseases characterized by chronic inflammatory processes which lead to the formation of granulation tissue, and often tumour-like swellings. The group includes tubercle, leprosy, actinomycosis, and syphilis.

Tuberculosis is a disease widespread among birds and animals It is due to the bacillus discovered by R. Koch in 1880. The tubercle bacillus is a slightly curved rod about 4 μ (a little more than one-half the diameter of a red blood-corpuscle) in length. It is difficult to stain, and grows slowly on suitable media at warm temperatures. The commonest anatomical element produced by the growth of the bacilli in the tissues is familiar as the grey miliary tubercle seen in general tuberculosis. Each such element or lesion (=injury) comprises several "giant-celled systems." The bacilli enter the body in inhaled dust, in unboiled milk from tubercular cows, and by inoculation in openings in the skin, accidental or due to pediculi, or in abrasions, etc., of mucous membranes. Thus tuberculosis of the lungs, intestine, and skin is common. From the surface of the skin, etc., the bacilli pass along the lymphatics to the lymphatic glands, where tubercle is one of the most common affections.

From the lymphatic glands the bacilli frequently pass to the large veins, and thence to the heart, thence again to the lungs; such as are not arrested there return to the left heart to be distributed to any vascular parts of the body. If sufficient numbers of bacilli enter the circulation, general tuberculosis is the result. If they are but few they may remain in the lungs, which are thus seen to be exposed to a second mode of infection, or pass through to be sent from the left ventricle of the heart to set up local tuberculosis in various parts of the body. Doubtless in fairly resistant individuals many of the bacilli are destroyed by the phagocytic action of leucocytes or connective-tissue cells. In rare cases general tuberculosis is recovered from. But a local tuberculosis, which remains as the only survival of a general infection, may again become generalized by lymphatic infection, or by the escape of bacilli into a vein, the wall of which has become invaded by the micro-organisms.

To follow the process of tissue-change under the influence of tubercular infection a little more closely, we may imagine a living bacillus which has penetrated between two or into one of the endothelial cells of a capillary blood-vessel. If the subject of the infection is protected, the cells destroy the bacilli, and the process ends. If protection is wanting, the bacilli multiply, growing into the connective tissue beyond the capillary. The effect of the poisonous material (tuberculin) formed by the bacilli is to cause the connective-tissue cells to enlarge and become amœboid, surround some of the bacilli, and coalesce to greater or lesser extent, as at the beginning of the formation of granulation tissue (see p. 22). The enlarged connective-tissue cells are termed endo- or epi-thelioid on account of their size. At this stage each group of enlarged connective-tissue cells is said to constitute an *epithelioid tubercle.* It must be remembered that the smallest tubercular lesion visible to the naked eye, the "miliary tubercle," comprises several histological tubercles. An additional effect of the tuberculin is the attraction of leucocytes from the neighbouring blood-vessels. The leucocytes, which are chiefly of the uninucleated sort (lymphoid cells), collect outside the epithelioid cells, forming a zone of lymphoid cells. If the growth of bacilli is rapid, the lymphoid cells

constitute the chief part of the lesion, in these circumstances called a *lymphoid tubercle.* If the irritation is only of moderate degree, the central epithelioid cells fuse together, forming a multinucleated cell-aggregate or *giant cell,*[1] and the *giant-celled tubercle* is formed, which consists of the giant cell, outside this epithelioid cells, and again outside them the lymphoid cells.

Bacilli are found in the giant cell and in and between the endothelial cells.

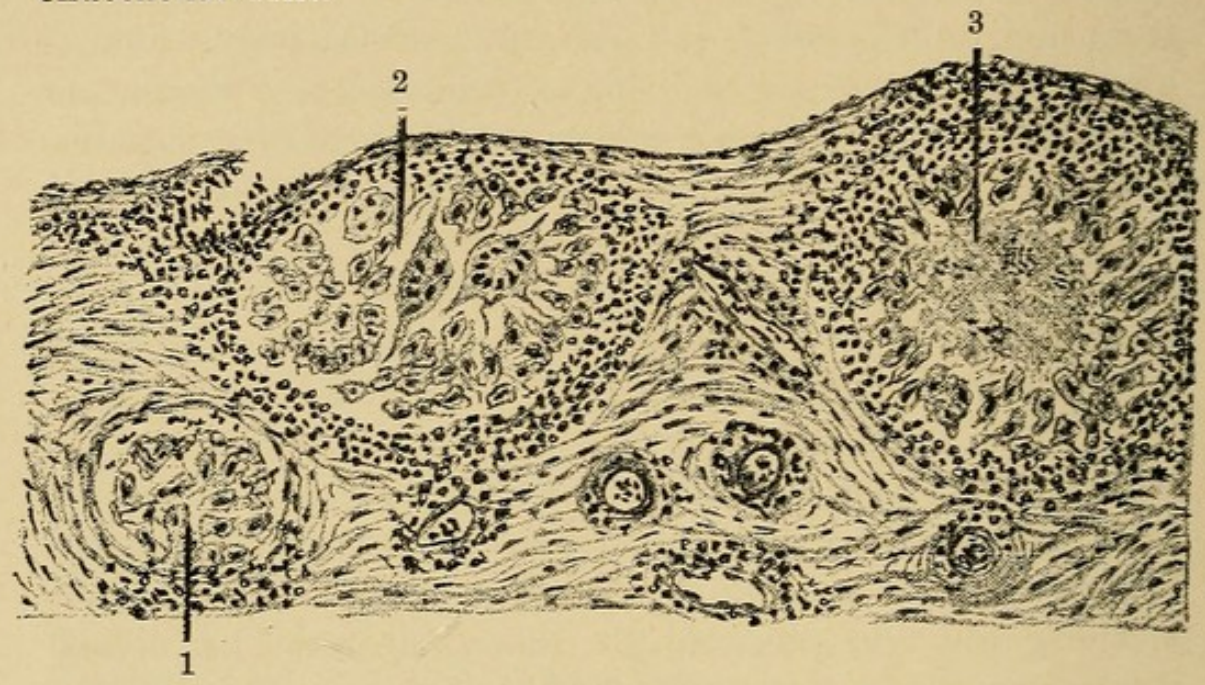


FIG. 22.—Section of a piece of synovial membrane, showing 1, tubercle commencing in the interior of a small vein; 2, a tubercle with two giant cells, endothelioid cells and leucocytes; 3, a tubercle with necrotic centre or caseous focus resulting from disintegration of the giant cell. From the knee-joint of a middle-aged man who suffered from tuberculosis of the suprarenal capsules (Addison's disease), and one of whose knee-joints rapidly swelled. The man died of heart failure. After death a considerable quantity of fluid was found in the joint, and every part of the synovial membrane was thickened and warty from the presence of recent tubercles. × 160.

The part of the giant cell in immediate contact with the bacilli undergoes necrosis, the protoplasm coagulating in fine granules. When the disease progresses, this necrotic change involves the whole of the giant cell, and afterwards the whole tubercle; new tubercles then form from the extension of bacilli from the caseous focus into the surrounding tissues. In this way large caseous masses may be formed.

[1] Giant cells are also formed by the subdivision of the nucleus of a connective-tissue cell. In sarcomata they are frequently multinucleated buds of capillaries. Giant cells occur in healthy granulation tissue as well as in many chronic inflammatory conditions.

When the growth of the bacilli is rapid, and the inflammatory reaction is the more marked, there is no time for the formation of giant cells and caseous matter, and the tubercular process then closely resembles suppuration, but is unattended with the heat and pain of an acute inflammatory process—hence the term "*cold abscess.*" Thus a gland may swell, rapidly undergo colliquative change (necrosis), and, on being incised, give exit to pus which on examination is found to contain bacilli. The earlier stages of tubercle often, and the later ones sometimes, are accompanied by the exudation of much serum. Thus at the early stage of tubercular peritonitis there may be much ascites, a condition which marks the right moment for surgical interference. In joints, too, there is sometimes a considerable degree of serous effusion. The thin pus found in many abscesses secondary to old spinal disease may be taken as an example of serous exudation in tuberculosis of old standing. Similarly, tuberculosis of the testis and other organs may be very rapid. On the other hand, when the bacilli multiply slowly, some of the enlarged connective-tissue cells of the tubercles are free to assume their fibroblastic function, and from the parts surrounding the tubercles ordinary granulation tissue is formed; hence scar-tissue is produced, which offers a barrier to rapid extension of the disease, and in many cases brings it to a close: the bacilli perish, and their remains, embedded in caseous material, are left surrounded by scar-tissue. The caseous material may then become calcified by the deposit of lime salts. When the reparative process is going on in bone affected by tubercle, new bone may be formed.

Wherever tuberculosis is going on in the body there is danger of its extension along lymphatics, or ulceration into veins, and hence of general infection, with the formation of metastatic foci of disease by embolism, just as in pyæmia, etc. Hence in any given case the surgeon has to balance the probability of a natural cure being obtained with the desirability of attempting to remove a possible source of general infection.

When there is a fair balance of power between the tissue elements and the bacilli, the protecting barrier assumes the form of granulation tissue. Thus a joint may be distended with

granulation tissue which covers the synovial membrane and the cartilage, which is gradually removed: multiplication of the cartilage cells having loosened its structure, granulation tissue springing from synovial membrane and from the bone replaces the cartilage at first in patches and, later, entirely, unless the cells of the granulation tissue defeat the bacilli, and the process is so brought to an end before all the cartilage has disappeared.

Syphilis. Syphilis is a disease caused by the introduction of a specific virus into the body. The nature of the virus is unknown. It is conveyed either by direct inoculation (acquired syphilis), or is contracted by the embryo before birth,[1]—congenital syphilis.

Acquired Syphilis. For a period of about twenty-five days after the inoculation there are no signs of the disease. This is termed the incubation period. Usually the abrasion or other slight injury through which the poison enters heals during this time, or it may be the seat of suppuration from the entry of other organisms along with the virus of syphilis.

Primary Syphilis. At the end of the period of incubation, a local thickening in the skin forms slowly at the seat of inoculation. The thickening is characterized by a definite induration. The surface over it may remain unbroken, a few scales only forming over a brownish red papule, but commonly an ulcer with an indurated base develops; the *primary sore* also known as the *hard* or *Hunterian Chancre.* It is usually painless. As the induration at the seat of inoculation is advancing, the lymphatic glands which first receive the lymph from the seat of inoculation become enlarged and hard. The

[1] If a woman, previously free from the disease, contracts it during pregnancy, the virus passes from the maternal to the fœtal blood by the placenta. More commonly the disease is transmitted to her offspring by a mother infected before conception. It is not impossible that the virus may pass with the spermatozoa, and so infect the ovum from the commencement. Colles observed that the mother of a syphilitic child never contracted a primary sore on her nipple, though she had shown no symptoms of the disease; whilst wet nurses who had not had syphilis were frequently affected by chancres of the nipple, due to infection from the mouth of syphilitic sucklings. Thus *Colles's law* is to the effec that the mother of a syphilitic infant is immune to the disease.

lymphatic vessels, which pass from the sore to the glands, may also be thickened. The sore is generally healed before the eighth week from the commencement of induration. At the margin of a primary sore the epidermis extends by elongation of its inter-papillary processes into the subjacent tissue. The latter is increased in amount by a multiplication of the connective-tissue and endothelial cells, and infiltration of leucocytes.

Induration, which is usually a marked feature of the primary manifestation of inoculated syphilis, recalls the firm character of some cancerous and other growths. To account for it there is, besides the cell-infiltration and cell-proliferation described above, evidence of proliferation of the lining cells of lymphatic vessels. Even when the induration is limited to the tissues immediately beneath the floor of the ulcer, it is still a marked feature, and is known as "parchment induration." When once the primary lesion has formed, the individual is insusceptible to inoculation with syphilis. This, together with the induration of the syphilitic sore, serves to distinguish the true chancre (Lat. *cancrum*) from the chancroid or soft sore.

Secondary Syphilis. This stage of the disease sets in from five to seven weeks after the commencement of induration in the primary sore. It is ushered in by slight fever, headache, and neuralgic pains, phenomena which correspond to what is termed the invasion of an ordinary exanthem such as measles, etc. The invasion marks the general infection or dissemination of the virus by the blood. To these symptoms soon follow an eruption of brownish-red spots on the trunk and the limbs, and sore throat with swelling of the tonsils, and (sometimes) of the lymphatic glands throughout the body. Some of the spots or *syphilides*, as the skin lesions are termed, may become raised above the surface (papules); on the papules scales of thickened horny epidermis often develop. The papules may become vesicles, and then change to pustules. In moist parts, as in the mouth, anus, and vulva, the papules become swollen, pale, or sodden from œdema, constituting *mucous tubercles*. The secondary stage may continue for eighteen months or more, different lesions appearing all the time; or the eruption may be slight and, after the first few weeks, no other symptoms

may be observed. During this period the hair usually falls out, and the tonsils may become ulcerated; there may be pains in the bones, and towards the end of the period, inflammation of the retina, of the internal ear, or of the iris. The secondary eruption is symmetrical, *i.e.* affects equally both sides of the body; it often exhibits a variety of different anatomical lesions —polymorphism. The great variety exhibited by syphilitic lesions often results in a close simulation of other eruptions; thus there are psoriasiform, lupoid, and other syphilides, as will be pointed out more fully under "Diseases of the Skin." In severe attacks the successive crops of secondary lesions become deeper, and the latest may undergo central necrosis and ulceration, having, in fact, the characters of gummata. When ulcerated secondary lesions become covered by crusts, and continue to enlarge, the crusts assume the appearance of limpet-shells, and the eruption is termed *rupia*. In these cases the secondary gradually merge into the tertiary lesions, and leave the impression that the latter are caused by the same virus as the earlier phenomena, though the virus is so altered that in the tertiary stage the disease is, as a rule, neither contagious nor transmitted to offspring.

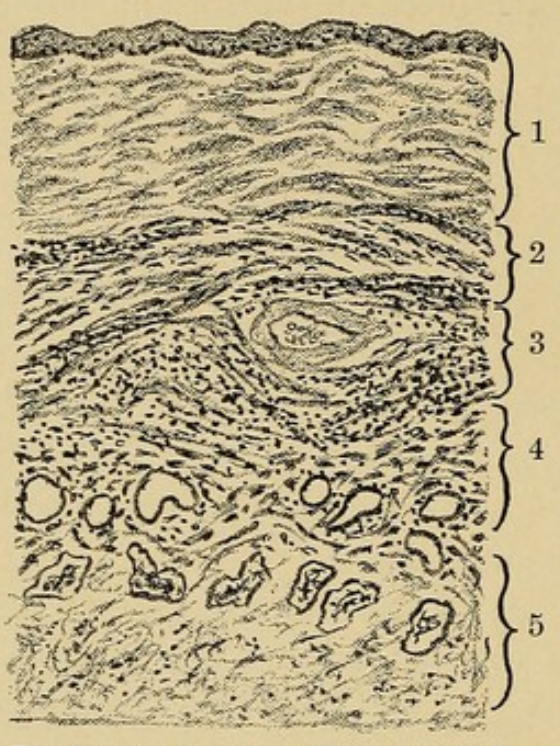


FIG. 23.—Section through the marginal part of a gumma of the subcutaneous tissue invading skin, seen under a low power. 1, Normal epidermis and papillary layer of corium. 2, Part of the corium showing enlargement of the nuclei of connective-tissue cells. 3, Deeper part of the corium altered by proliferation of connective-tissue cells and immigration of leucocytes. 4, Layer of granulation tissue. 5, Necrosed tissue with thrombosed capillaries.

Tertiary Stage.[1] After the first eighteen months or so, the

[1] Though the division of the periods of syphilis into stages is convenient and in harmony with the phenomena observed, it is to be remembered that symmetrical eruptions may appear as long as a year or more after the average time (eighteen months) for the secondary period to come to an end.

secondary lesions may gradually merge into the tertiary, *i.e.* they tend to acquire a more destructive character, shown by ulceration, etc., and to lose their symmetrical distribution. There may, however, be no tertiary symptoms at all, or years may separate the last secondary from the first tertiary symptoms. The most characteristic anatomical lesion in tertiary syphilis is the gumma, which is a focus of inflammatory change. If the process is rapid, colliquative necrosis occurs, and the somewhat sticky semi-transparent fluid from which the name "gumma" is derived is formed. The minute anatomy of rapid gummatous inflammation is shown in Fig. 23.

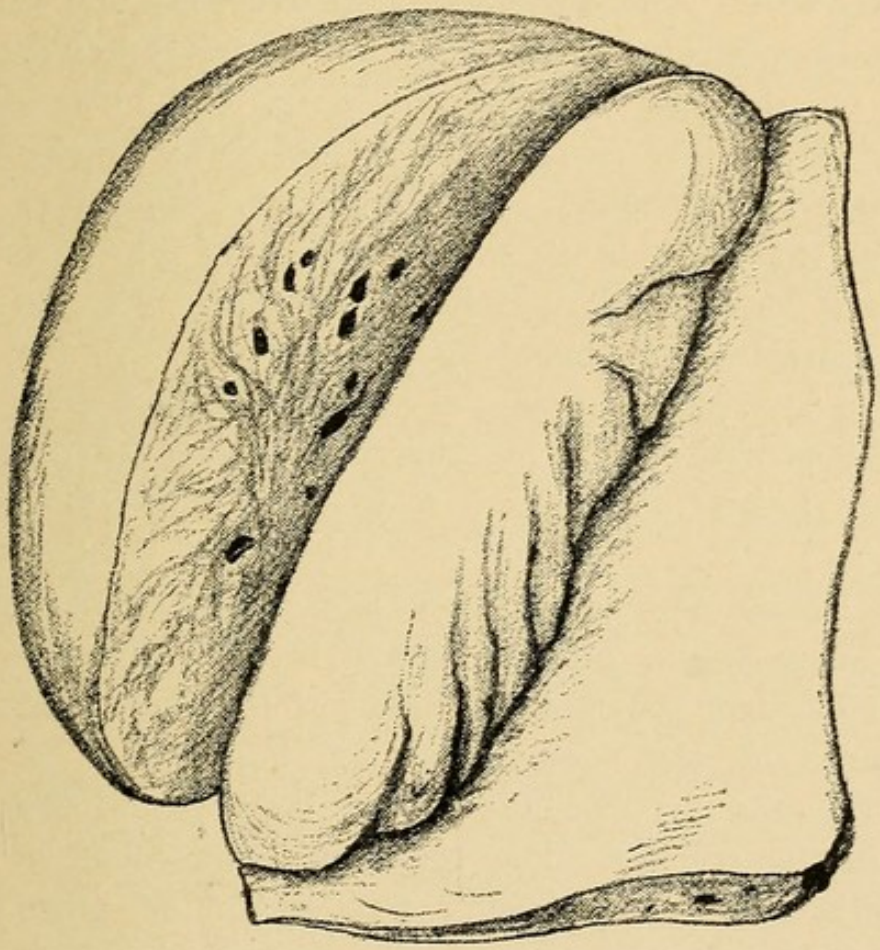


FIG. 24.

FIG. 25.

FIG. 24.—Part of a liver containing a large gumma cut open. The central portion of the tumour has become cicatrized. From a child aged six years, who died from the effects of congenital syphilis. Nodular swellings were felt in the liver during life. Nat. size.

FIG. 25.—The ulna of a child, vertical section. The shaft is greatly thickened by the formation of new bone (node) from the periosteum. Nat. size.

Gummatous inflammation of a degree of intensity, insufficient to cause liquefaction of the tissues results in the formation of

caseous foci surrounded by granulation tissue. A still less rapid process results in tumour-like growths which are most frequently observed in soft organs like muscles or the liver (Fig. 24).

When the inflammatory reaction to the virus of syphilis is least intense the process results in hyperplasia. The best examples of this are to be seen in the case of bones such as the one shown in Fig. 25.

The late or tertiary symptoms of syphilis differ from those of the secondary period only in being more local. The virus appears to have left the blood and to have lodged in various parts of the body where it is capable of local extension and is amenable to local treatment. Thus, whilst the secondary lesions appear equally on the two sides of the body, the tertiary phenomena tend to be more isolated or asymmetrical. Sometimes syphilis is a mild affection. The secondary stage may be very short, and there may be no tertiary lesions at all. The primary lesion may be so slight that it is overlooked. On the other hand, in individuals weakened by intemperance, etc., the disease may prove fatal, either by the supervention of lardaceous disease or by lesions of the brain or other important organs, or by the syphilitic lesions opening up a way for suppurative infection.

In *congenital syphilis* the early stages are passed through before birth. There is no primary stage, because infection occurs from the blood of the mother to that of the fœtus, and possibly sometimes by the spermatozoa at the moment of fertilization of the ovum. The fœtus is often destroyed by the disease, and abortion results. The secondary phenomena in congenital syphilis may be extremely severe and often prove fatal. Though essentially the same as the inoculated disease congenital syphilis has certain peculiarities owing to the period of life of the victims. In the bodies of new-born infants that have died of syphilis it is common to find small gummata in the liver and evidence of inflammation at the juxta-epiphyseal regions of long bones, etc.

Among the early eruptions are nasal catarrh ("snuffles"), and bullous or pemphigoid syphilides: large blebs filled with pus

which form in the skin and mucous membranes. Mucous tubercles are common at the angles of the mouth (where they leave radiating fissures) and about the anus and vulva.

The alteration in the shape of the second set of the upper incisor and canine teeth was first noticed by Hutchinson. These teeth may be notched, pegged, and inclined towards the middle line. This is probably the result of inflammation of the tooth-sacs at an early stage of the development of the teeth. Among the tertiary phenomena a chronic inflammation of the cornea (interstitial keratitis) is common. It results in a hazy opacity of the cornea, and if the newly formed tissue becomes vascular the opaque areas become pink (salmon patch).

Phagedæna is a rapid spreading of either a primary, secondary, or tertiary lesion accompanied by sloughing. It is due to the addition of pyogenic or other organisms to the syphilitic virus and is observed in non-syphilitic lesions also.

The nature of Syphilis. If syphilis be compared with inoculated small-pox, a close analogy is apparent. On the second day after inoculation with small-pox lymph discoloration of the site of inoculation was observed, and by the fifth day inflammatory infiltration and the formation of a vesicle occurred. This became pustular, and fever set in by the ninth day, and three or four days later the general eruption appeared. Thus there were "primary" and "secondary" stages. And farther, if a woman contracts small-pox during pregnancy abortion occurs and a dead fœtus, which may be covered with a typical small-pox eruption, is produced. Here is an approach to "congenital" small-pox. Moxon's aphorism to the effect that syphilis is specific fever "diluted by time" is very just. Second attacks of syphilis occasionally occur as in the other specific fevers. A vital question in syphilis is how soon after contracting the disease may a person marry without danger of transmitting the disease to his or her children. Though under suitable treatment a person appears as a rule to be free from symptoms six months after the appearance of the primary sore, Hutchinson's rule (*Syphilis*, 1887, p. 494) is that two full years should elapse between the beginning of the disease and marriage,

and that the patient should be under treatment the whole of this time.[1]

The good effect of mercury on early and iodide of potassium on late syphilis is very striking.

Soft Chancre or Chancroid. This is an ulcer due to a local infective inflammation which is marked by profuse suppuration. It commences in a small excoriation caused by coïtus. A small pustule forms and ruptures, so that in from three to five days the commencement of the ulcer is present. The ulcer is usually round in form, the edge is well-defined, its floor is uneven, and is covered with a grey sloughy coat; beyond the edge there is an areola bright red in colour; this is due to an inflammatory infiltration, which also causes a slight induration which fades off into the surrounding tissues and is not sharply defined as is the primary syphilitic sore. The discharge is purulent. If the discharge is pent up beneath a tight foreskin, more widespread sloughing may occur. Occasionally the ulcer may spread rapidly and widely with much sloughing, becoming "phagedænic" as it is termed. Soft sores are frequently followed by suppuration in the inguinal glands. The exact nature of the virus is not known. It differs from that of syphilis. The virus can be inoculated in the affected person and thus differs from that of syphilis, but it is to be remembered that the infection may be a double one, and what begins as a chancroid may after two or three weeks become a chancre.

Actinomycosis is due to the growth of a fungus which assumes in cultures the form of jointed branched filaments. Such forms are usually classed with the bacteria in a group

[1] The impress of syphilis on the offspring may be transmitted during a much longer period. Thus in a boy aged ten years, brought to hospital for pain and swelling in a knee-joint, I found a node on the corresponding tibia and radiating fissures at the angles of the mouth—positive signs of syphilis. The mother said she had "as good as" twelve children. The first born dead, the second and third died at the age of three months—one of "wasting," another of "swellings of bones and joints"—the fourth died at nine months, the patient was the fifth, the sixth, seventh, and eighth were living but had "snuffled" in infancy, ninth and tenth miscarried, eleventh and twelfth were alive and healthy.

termed cladotricheæ, from the club-like termination of some of the filaments. When growing parasitically in living tissues the clubbed ends of the filaments radiate from a common centre, forming a globular mass which in section has a radiated appearance, whence the name actinomyces (ray-fungus) of the parasite. The most common situation for the infection is on the tongue of calves. It is probably acquired from wheat and straw. In man it is met with in the jaw-bone (into which it finds its way through a hollow tooth), in the intestine and liver, and also in the skin either primarily or by extension. It forms indolent inflammatory tissue like that caused by tubercle. On the skin raised red nodules very like some forms of tubercular lesions are observed. They tend to soften centrally and to ulcerate. The diagnosis is made certain by the examination of a scraping under a moderately high power of the microscope (see Fig. 21, 4).

The administration of iodide of potassium has sometimes almost as marked an influence on actinomycosis as in late syphilis. The action is probably due to the drug altering the tissue cells or the fungus in such a way as to increase the power of the phagocytes in relation to the parasite.

Madura foot or Mycetoma, a disease met with in India and North Africa, is caused by a fungus which somewhat resembles actinomyces. The disease manifests itself by swelling of a hand or foot. Raised nodules appear in the skin and ulcerate, leaving suppurating apertures which communicate with large cavities in the soft tissues and the bones. These cavities are lined with granulation tissue containing pellets of the size of hemp-seed, composed of agglomerations of the fungus of which there are two varieties, one having a yellow the other a black colour. The disease is slowly progressive, and usually requires amputation.

Leprosy is a disease marked by a long period of incubation. This is always over two, and sometimes as long as ten years. A prodromal period follows, and is marked by slight illness, languor, headache, etc. At the end of this time there is a marked increase in the evidence of infection. This is termed the period of invasion. High fever with pains in the back and limbs characterize this period of the affection,

and have caused it to be mistaken for rheumatism. Some weeks or months after the commencement of invasion successive crops of red and slightly raised spots and patches appear on the face, trunk, and limbs. Sensation may be lost over the areas covered by the spots. The later phenomena of leprosy may affect the skin more pronouncedly than the other parts of the body, inflammatory nodules forming in the skin of the face, limbs, etc., and in the mucous membranes. It is then termed *skin leprosy*; or the effects of the disease may show principally by the formation of nodular enlargements of the large nerve trunks; in this case it is termed *nerve leprosy.* The result of leprous inflammation of nerve is pain and muscular paralysis together with inflammatory changes in the skin and wasting in the muscles supplied by the affected nerve. In other cases, termed *mixed leprosy*, the skin and nerve trunks suffer equally.

A section made through a leprous nodule of the skin and stained with carbol-fuchsin, decolorized and restained with methyl blue, shows the corium and more or less of the subcutaneous tissue to be crowded with bacilli which resemble those of tubercle save in that they stain much more readily. The bacilli lie for the most part in round or oval clumps, and careful examination shows that many of the larger or epithelioid cells of the inflammatory or granulation tissue in which they lie are packed with bacilli, and that the clumps of bacilli are formed within cells which afterwards undergo disintegration. In other words, the phagocytes are for the most part incapable of destroying the bacilli of leprosy. Examination of the body of a person who has died of leprosy reveals the fact that organs such as the liver, testes, etc., are crowded with bacilli lying in inflammatory foci. The thickenings in nerves affected by leprosy also contain bacilli in inflammatory tissue. Leprosy nodules differ from those of tubercle and syphilis in showing no tendency to undergo caseation. That the bacillus is the cause of the disease is highly probable, but it is hard to cultivate, and no animal has yet been found to be susceptible to leprosy; therefore the causal relationship of bacillus to the disease has not been proved by inoculation of pure cultures.

CHAPTER V.

NEW GROWTHS.

A NEW growth[1] or neoplasm is a formation of tissue that subserves no physiological function and obeys no physiological control. A fatty tumour (lipoma), such as is commonly seen in the subcutaneous tissue, may be taken as a familiar example of a simple new growth. It consists of cells similar to normal fat cells, and is surrounded by a thin capsule of fibrous tissue, which at one point is perforated by the blood-vessels of the growth. The chief feature which distinguishes it from normal fat is its coarsely lobulated form. Its independence of physiological control is seen when the person in whom it occurs becomes thin from illness or otherwise. The fatty growth remains undiminished in size, whilst the rest of the fat of the body is being absorbed. This independence of physiological control is a marked feature in new growths. A lipoma does not affect surrounding tissues, except by the pressure it exerts upon them, nor does it give rise to other similar growths in its neighbourhood or in distant parts of the body. In other words, it is an *innocent* growth. The innocent tumours are also termed "histioid" (Gr. *histos* = warp, web), because they resemble in structure certain fully differentiated normal tissues of the body, *e.g.* fat, fibrous tissue, etc.

[1] The word "tumour," which, strictly speaking, means nothing more definite than a swelling, has been applied to a number of conditions such as cysts, and errors of development, such as spina bifida, etc., so that it seemed better to use the term "new growths" as the title for the present chapter.

The new growths which in their habits most closely resemble the infective granulomata are the sarcomata and carcinomata. They differ from the innocent growths in that they infiltrate and destroy the tissues around them, and tend to form secondary growths in different parts of the body. Take for example a cancer of the tongue. If left to itself the growth invades the tissue of the tongue, and, when it comes to the surface, forms ulcers by its older parts breaking down from necrosis. By and by the glands which receive lymph from the tongue enlarge, and cancerous tissue exactly resembling the original growth in the tongue forms in them, and the same process of infiltration of surrounding tissues takes place around the cancerous glands as in the tongue itself. Thus, from invasion of the skin, cancerous ulcers may form under the jaw and at the sides of the neck. Finally, if the patient escapes death from septic broncho-pneumonia, other secondary growths may form in distant parts of the body. The dissemination of a sarcoma by secondary growths is similar to that observed in cancer, save that in the case of sarcoma the infective material is conveyed by veins more readily than by lymphatics, the reverse holding good for cancer. Thus cancer and sarcoma are malignant growths, and since in some particulars they resemble the infective granulomata, their chief characters may be considered a little more in detail. The sarcomata resemble inflammatory growths, such as syphilitic gummata, more closely than do the cancers, because the cells of sarcomata are of connective-tissue type, whereas the predominating element in cancers is epithelial.

The Sarcomata. These growths present a wide range of structure, and a correspondingly wide range of rapidity of growth and destructive power. Their common character is *malignancy*. The characters that are designated by this term will be made clear in the following description of sarcoma. The cells of some sarcomata (melanotic sarcomata—Gr. *melas* = black) are filled with granules of brown or black pigment. In such growths the pigmentation of the cells renders it an easy matter to follow in unstained sections under the microscope the first essential feature of malignancy, namely, the property of infiltrating and destroying the neighbouring normal tissues. As an example, a

melanotic sarcoma of the choroid coat of the eye may be taken. The choroid contains normally many pigmented connective-tissue cells, and the cells of the sarcoma may be regarded as being of the same descent as the normal cells of the part. If a section of such a growth is made where it abuts on the fibrous sclerotic coat of the eye, the manner in which the pigmented cells infiltrate the fibres of the healthy tissue is readily seen (Fig. 26).

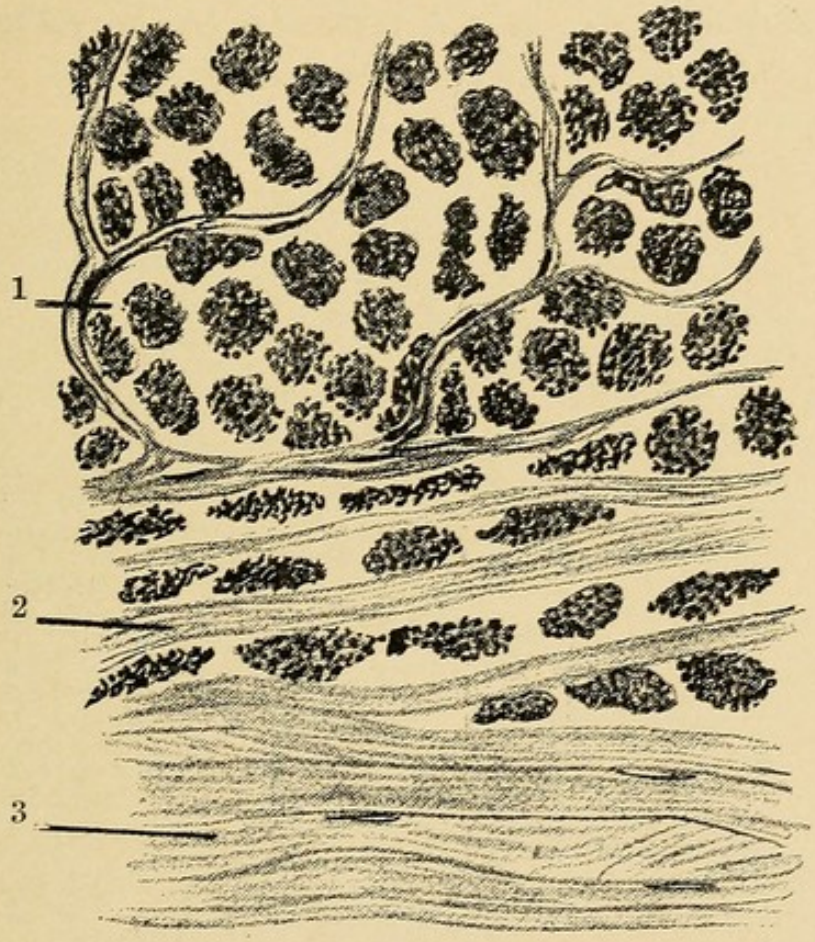


FIG. 26.—A section of a melanotic sarcoma of the choroid with part of the sclerotic. 1, Pigmented sarcoma cells which, at 2, have forced their way between the fibrous bundles of the sclerotic. 3, Normal sclerotic. The pigment obscures the nuclei of the cells of the sarcoma. × 200.

In this case of melanotic sarcoma it would appear that the cells which infiltrate and destroy the neighbouring tissues are derived from the original cells of the tumour. If, however, such a section is bleached and then stained in the usual way, the margin of the growth may show, besides the large cells of the growth, a number of leucocytes and connective-tissue cells similar to those seen in inflammatory conditions.

The close relation that exists between veins and sarcomata

is sometimes evidenced by the presence in large veins of ingrowths which project, often for a considerable distance, into the lumen of the vessel. In this way the inferior vena cava, in the whole of its course, has been found to be occupied by a plug of sarcoma. In persons who have died of melanotic sarcoma pigmented cells have been found in the blood-vessels of the lungs and other organs, having been conveyed along the veins from a distant growth. Hæmorrhage is a frequent accident in sarcoma, and depends on the tendency of the sarcoma cells to replace those of the blood-vessel (see Fig. 27).

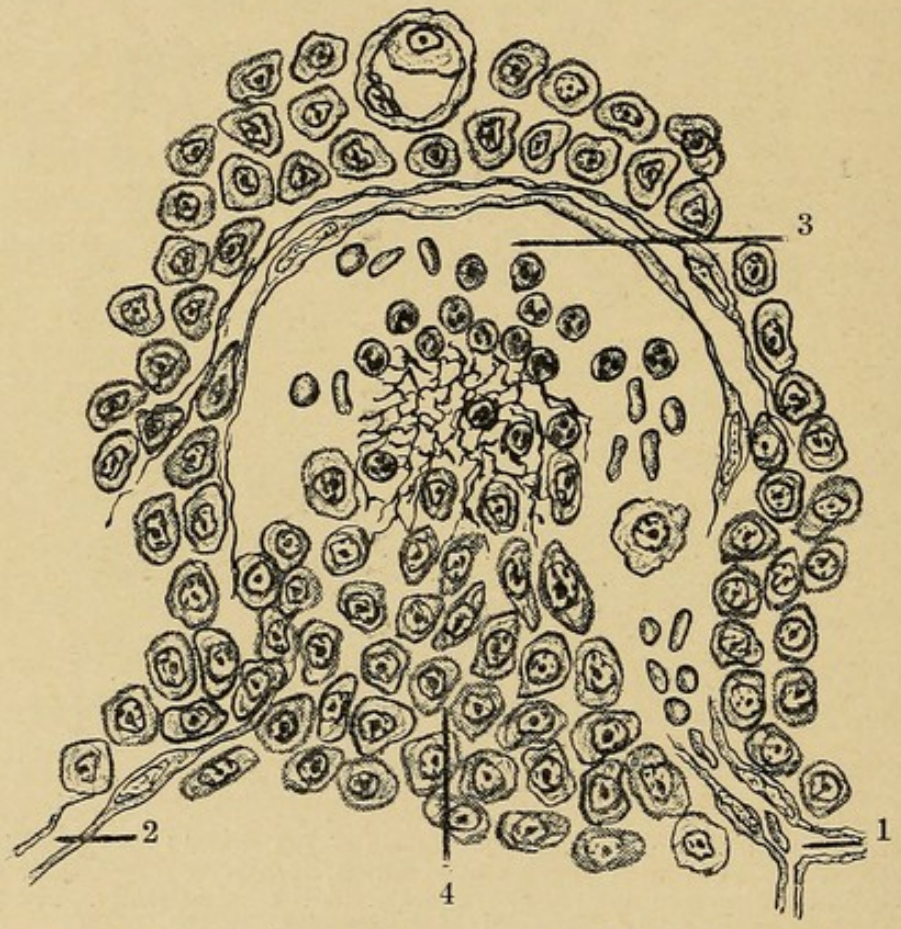


Fig. 27.—Section of an alveolar sarcoma of the breast. 1 and 2, Parts of a capillary which has been ruptured. 3, A space containing red and white blood cells which have escaped from the capillary. 4, A projection of sarcoma cells capped by fibrin towards the space (3). × 200.

If, as according to present knowledge is believed, it is the cells of the sarcoma that constitute the infection, there is an evident difference between the infiltration of a tissue by sarcoma and of an inflammatory formation such as tubercle. In the latter it is the bacilli and their products which, by acting on the connective-tissue cells and blood-vessels of the invaded tissue,

produce the lesions. Though in infective inflammatory lesions wandering leucocytes and connective-tissue cells invade neighbouring tissues, it is not these cells but the infective organisms which cause the spread of inflammation. It must be confessed, however, that when the margin of a sarcoma is examined under a high power, it is often impossible to say that some of the

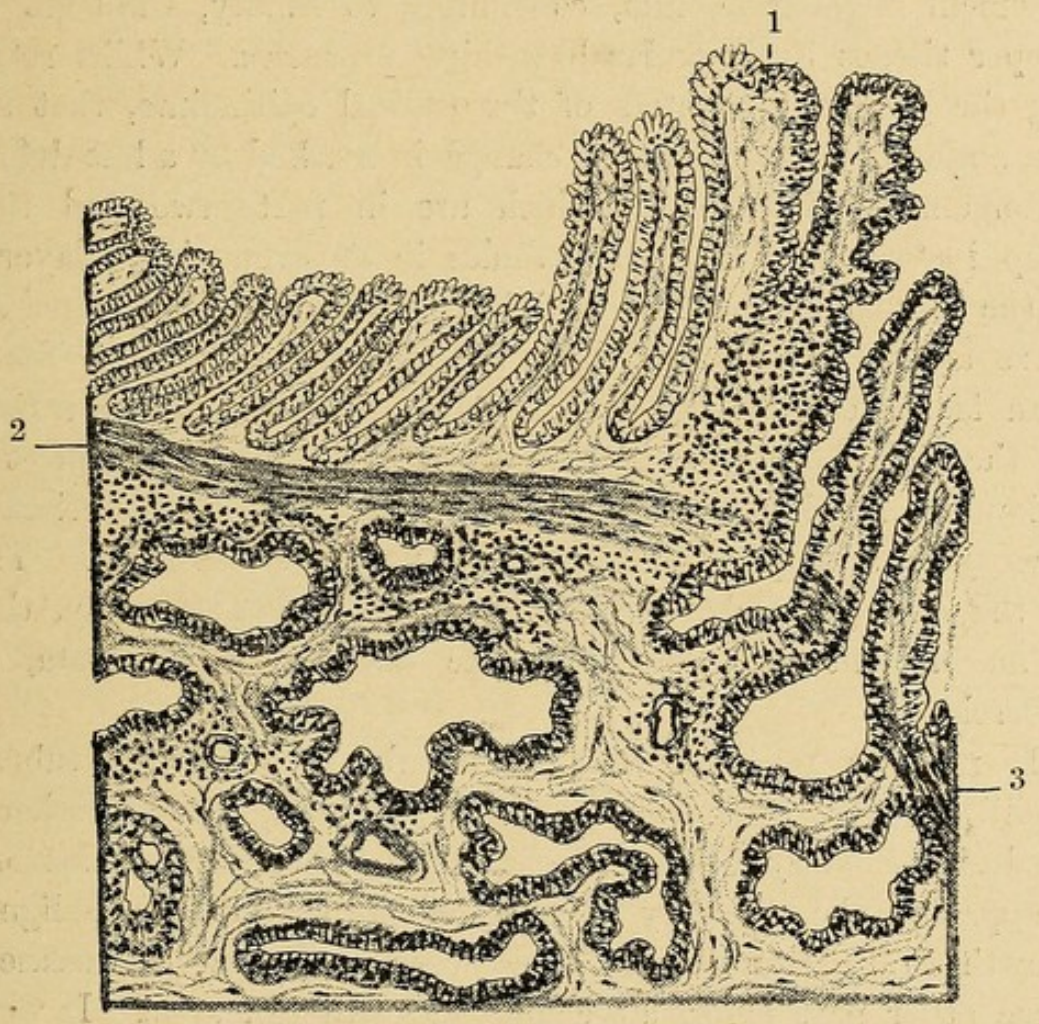


FIG. 28.—A section of a marginal part of a cancer of the colon. 1, Junction of the normal and the proliferating epithelium. 2, Muscularis mucosæ. 3, The cancerous down-growth piercing the muscularis mucosæ. There is small-celled inflammatory infiltration in various parts of the section. Near the edge of the growth (1) the adjoining mucous membrane is hyperplastic. The submucous tissue is everywhere infiltrated by tubular cancerous growth. × 30.

connective-tissue cells of the invaded part are not converted into sarcoma cells. The difference between the mode of extension of the infective granulomata and malignant growths is more easily made out in cancers than in sarcomas.

Cancer. In malignant epithelial growths or cancers the infiltration of surrounding structures is more easily traced because the invading tissue is epithelial. This can be well

seen at the edge of a cancerous growth on the surface of the skin or some mucous membrane. In Fig. 28, which represents a section made through the edge of a cancer of the large intestine, the normal mucous membrane is seen on the left, the cancerous on the right of the figure. The tubular glands, on becoming cancerous, grow unrestrainedly, pass through the basement membrane, and, continuing to ramify, infiltrate the deeper tissues by irregular branching processes. Whilst retaining the general characters of the original epithelium, that that has undergone the cancerous change is marked by a less definite arrangement of the cells, which are in parts two and three deep instead of lining the tubules in an even single layer as in the normal state. The individual cells also have larger and more active nuclei, and their protoplasm stains more deeply than is the case in the normal cells. In the connective tissue in the neighbourhood of the cancerous growth, evidences of inflammatory changes are present in the form of collections of leucocytes and proliferation of connective-tissue cells. Thus in the sarcoma probably, and in cancer almost certainly, there is an element not present in the infective granulomata, *e.g.* tubercle.

It may be repeated that it is only necessary for tubercle bacilli or other organisms to pass from one part to another to produce secondary lesions. In cancer, however, and in sarcoma, it appears to be necessary for some of the cells of the malignant growth to be transferred from the primary seat of disease to some other part before a secondary growth can arise. It would thus, according to present knowledge, appear that for the dissemination of malignant growth a transplantation of tissue cells is required, whilst in infective inflammation transplantation of virus is all that is necessary to produce the same effect. The growths secondary to a melanotic sarcoma, for instance, possess the same peculiarities of cell and pigment as does the primary growth.

In cancer the likeness of the secondary to the primary growth is still more striking. Thus a nodule of cancer in the liver, secondary to a cancer of the intestine, had broadly the same characters as the primary growth. In the same way a cancer in

bone secondary, say, to a growth in the breast is similar in character to the original growth.

Resemblances between Malignant Growths and the Infective Granulomata. At the margin of a cancerous surface, the conversion of previously healthy into cancerous cells may be observed. Thus in the cancer shown in Fig. 28, more and more of the epithelium at the margin of the growth becomes involved. Again, where a cancer involving a surface of skin lies against healthy skin, as in cancer of one of the labia majora, a second cancer may form exactly opposite to the first.

The gross aspect of secondary or metastatic growth, whether cancer or sarcoma, may strongly remind the observer of the infective granulomata. Thus a sarcoma of the vertebræ, secondary to a growth of some other part of the body, may present much the same appearance as the same part in tubercular caries. There may be absorption of one or more vertebral bodies by what might be termed "sarcomatous caries," and infiltration of the cancellous tissue in the neighbourhood of the area of destruction. In the sarcoma, however, there will be more extensive formation of soft tissue on the periosteal aspect of the bone, and probably less extensive caseation than in a case of tuberculosis. The dissemination of sarcoma more commonly occurs by way of the veins, but it also takes place by way of the lymphatics. In cancer lymphatic infection is the rule, and hence in cancer the likeness to tubercular infection is often very close. In many cases of tubercular ulcer of the small intestine the secondary tubercles may be seen following the course of the lymphatics beneath the serous coat of the bowel. The nearest mesenteric glands may be found to be infected, and also the whole of the peritoneum, which is studded with firm raised grey or caseous nodules. It frequently happens also in cancer of the stomach or intestine that the lymphatics and the whole of the peritoneum become studded with nodules of secondary growth. In such cases the close general resemblance of the cancerous peritoneum to cases of tubercular peritonitis has given rise to the term "cancerous peritonitis." In the pleura and the meninges a similar resemblance between cancerous and tubercular lesions is frequently

observed. And it may be added that certain sarcomatous infiltrations present also to the naked eye a close resemblance to tubercular infiltrations. Other features common to cancer and sarcoma and inflammatory formations may be mentioned. Thus the older parts of malignant growths may undergo necrosis and caseation as do the infective granulomata. When necrosis occurs in a growth involving skin or mucous membrane ulceration results. The edge of a cancerous ulcer is usually firm and raised, because the tissues beyond the edge of the ulcer are infiltrated with growth. On another point malignant growths may be compared with the infective granulomata. That is in the variations of rate of growth shown by the same kind of neoplasm in different individuals, or at different periods in the same individual. Taking the common hard cancer of the breast as an example two extremes may be noted: one of these growths may require years to attain the size of a walnut (atrophic scirrhus), whilst another cancer of the same kind may in a few months not only attain a large size, but also widely infiltrate the skin of a considerable part of the trunk (cuirass scirrhus).

Slow-growing cancers present a tendency to cicatrization, as is well seen in the atrophic hard cancer of the breast. The blood-vessels of malignant growths like those of granulation tissue are derived from the tissue in which the growth arises and the tissues which it invades.

Innocent Growths. The contrast between a simple fatty tumour and a sarcoma has already been made. Innocent tumours, such as lipomata, fibromata, myomata, etc., may attain a large size, and may even produce fatal symptoms by pressure on important organs. When they are superficially placed and have skin or mucous membrane stretched over them, they frequently cause ulceration by pressure, and thus open a way to pyogenic and other organisms, which may cause sloughing of the tumour, septic infection, etc. The tendency of a simple connective-tissue growth to become malignant will be noticed later.

Innocent Epithelial Growths—Papillomata and Adenomata. The difference between the histology of an adenoma and that of a

cancer may be illustrated by comparing a cancer, such as that shown in Fig. 28, with an adenoma of the rectum as shown diagrammatically in Fig 29.

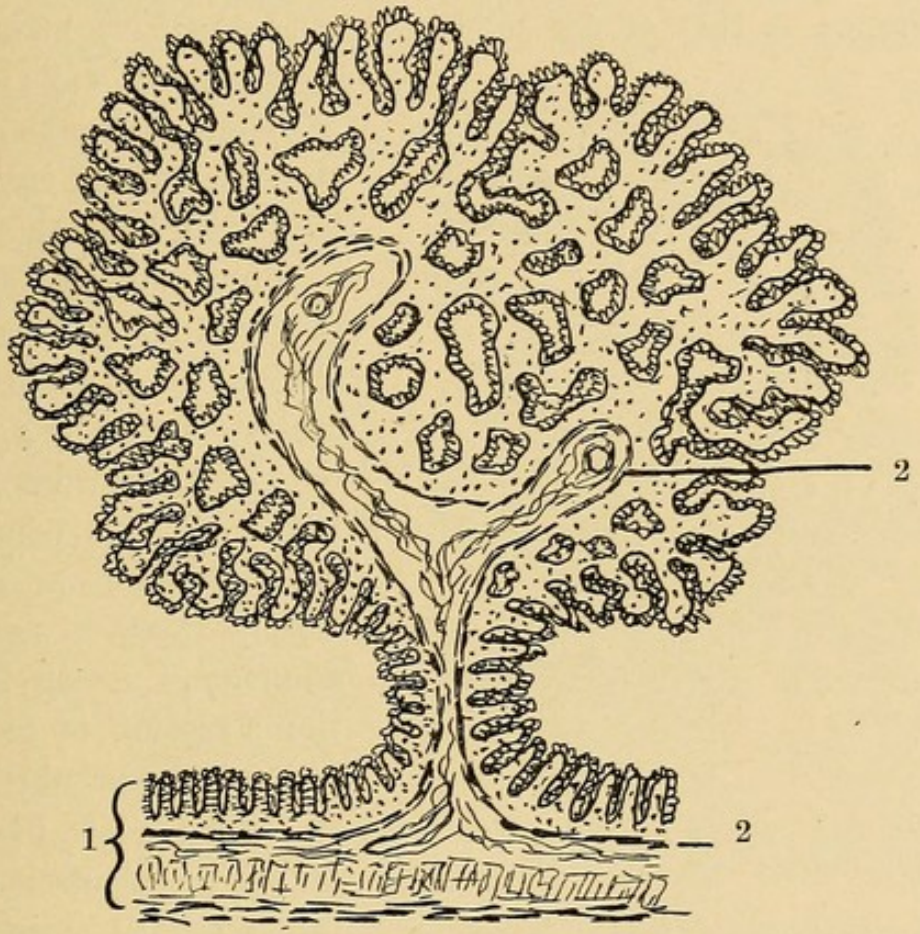


Fig. 29.—A diagram illustrating the nature of an adenoma of the rectum. 1, Normal bowel-wall. 2, 2, Muscularis mucosæ which is nowhere penetrated by the epithelial proliferation which constitutes the chief part of the growth. The pedicle consists of normal mucous membrane and submucous tissue.

In the adenoma, as in the cancer, there is an excessive proliferation of epithelial structures; but in the adenoma the latter are limited to the mucous membrane, at no point passing through the *muscularis mucosæ*. The histological structure of the adenoma shows a slighter deviation from the character of the normal epithelium of the part than is the case in cancer. Adenomata are devoid of the special characters of cancer, that is, they do not infiltrate and destroy neighbouring tissues, nor do they form metastases. Now and again an adenoma changes its character and becomes a cancer. When an adenoma develops in the interior of a gland, such as the breast or the thyroid, the process is similar to that which leads to the formation of

adenomata of mucous surfaces. For example, the margin of a commencing adenoma of the thyroid gland may be studied as shown in Fig. 30.

Papilloma. If in an area of the skin and of such mucous membranes as that of the mouth or of the urinary bladder, or in the fibrous walls of ducts of glands such as the breast, the small blood-vessels immediately beneath the epithelium may elongate and project from the surface as simple or branched processes covered by a greater or lesser thickness of epithelium. This constitutes a papilloma, a growth closely allied in nature to adenoma. As an illustration a section of an ordinary cutaneous wart[1] may be taken (see Fig. 31). Many papillomata are of inflammatory origin, for instance, gonorrhœal warts. An ordinary corn has many of the features of a papilloma. Comparing a section of a squamous-celled cancer (squamous epithelioma) with a papilloma, it is observed that in the former the most marked feature is a down-growth of the interpapillary processes of epidermis. Many of the down-growing processes contain "epithelial pearls" or "cell-nests," *i.e.* collections of flattened horny cells arranged concentrically (see Fig. 32). A section of part of a papilloma cut obliquely

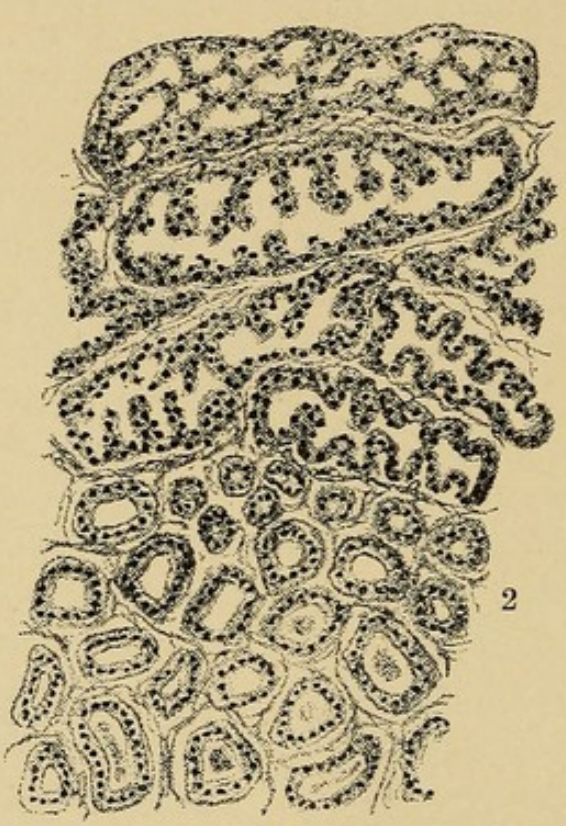


FIG. 30.—The margin of a commencing adenoma of the thyroid gland. 1, The adenoma consisting of acini, the mucous membrane of which is thrown into folds from overgrowth. 2, The adjoining normal gland-tissue. There is as yet no capsule to the growth, which was one of many similar tumours met with in a large goitre in a child. Various members of the same family were similarly affected. ×200.

[1] An ordinary cutaneous papilloma is sometimes called a "hard wart," to distinguish it from the so-called soft wart, which is a kind of mole (see below) and is due to overformation of the connective-tissue cells of the corium, the epidermis being thinned.

may very closely resemble a cancerous growth, and this fact necessitates extreme caution in basing a pathological diagnosis on the histological character of a growth. In every case the clinical characters should be carefully weighed before a diagnosis is made. The same may be said of small-celled sarcomatous growths which closely approach some inflammatory tissues. Granulation tissue will usually show formation of cicatricial tissue from fibroblasts, which will enable a practised eye to draw a distinction.

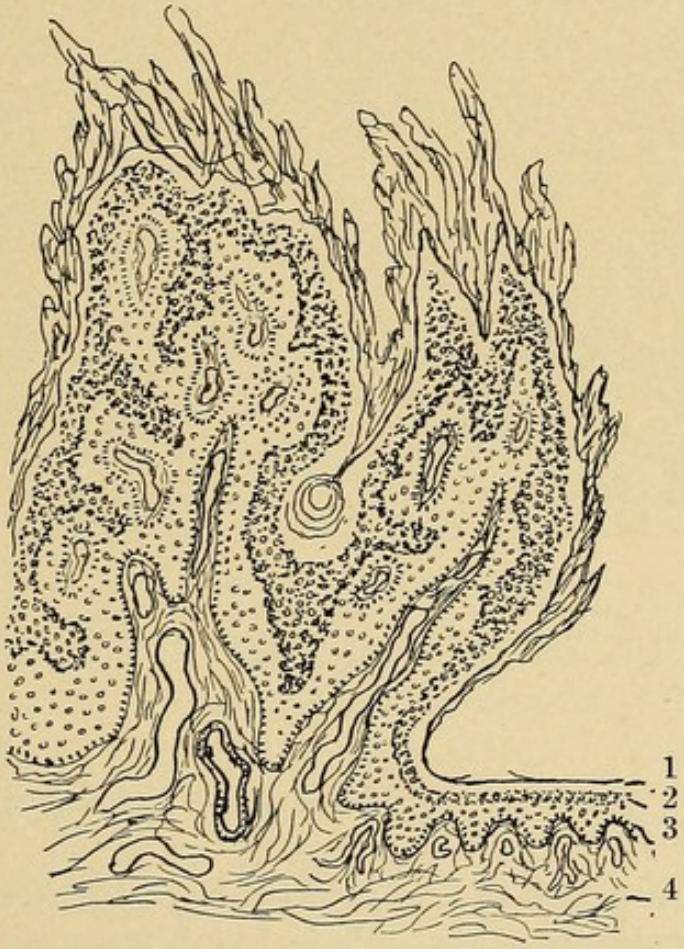


FIG. 31.—Vertical section through the margin of an ordinary cutaneous wart, with the adjoining part of normal skin, in which 1 = horny layer; 2, granular layer; 3, mucous layer of the epidermis; 4, papillary layer of the corium. All these layers are exaggerated in the papilloma, and the papillæ and their blood-vessels have undergone branching. ×25.

Malignant Metaplasia of Innocent Growths. Wherever connective tissue is present, whether in normal tissue or in neoplasms, there is a possibility of the development of sarcoma; and cancer may begin in any part where there is epithelium, whether this is in normal skin, mucous membrane, or glands, in embryonic 'rests' such as those of the Wollfian body in the broad liga-

ments, or the hilus of the testis, or in dermoid and other tumours. Adenomata may also undergo cancerous metaplasia.

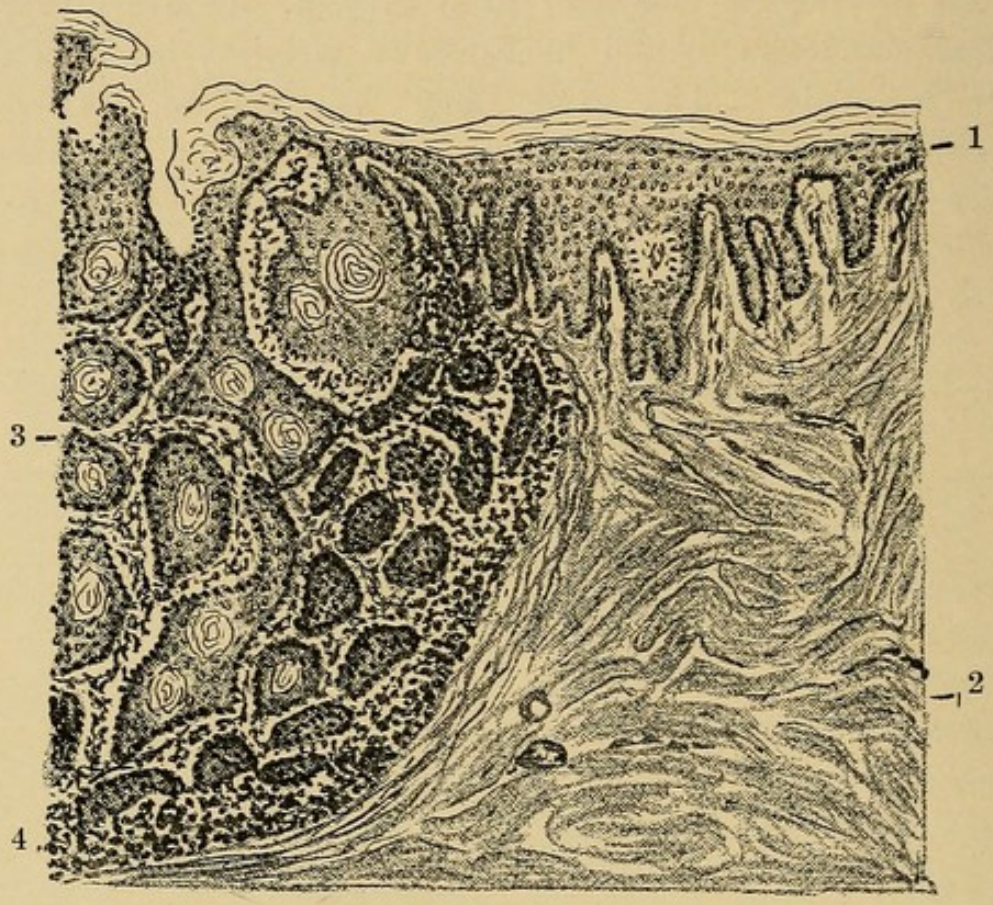


FIG. 32.—Section at the margin of a squamous-celled cancer. 1, Normal epidermis; 2, normal corium; 3, cancerous growth consisting of epithelial processes which contain cell-nests and are supported by cellular granulation-tissue; 4, advancing margin marked by inflammatory cell-infiltration. × 40 diams.

CLASSIFICATION OF NEW GROWTHS.

Since the causation of new growths is, in most cases, unknown, the only classification available is based upon their histological structure.[1] A large class of new growths are developed in tissues derived from the mesoblast of the embryo. This may be called Class I. It includes:

Fibroma, *Chondroma*, *Osteoma*, *Lipoma* resemble normal connective tissues.

Myxoma is a growth consisting of mucous tissues, *i.e.* resembling the connective tissue of the umbilical cord.

[1] This was first taken as a basis of classification by Johannes Müller in 1838.

Angioma consists of blood-vessels with supporting connective tissue.

Lymphangioma consists of lymph-vessels with supporting connective tissue.

Myoma. Tumours consisting of unstriped muscle (leiomyomata) are common. Rhabdomyomata, or tumours consisting wholly of striped muscle, are rare, only occurring as congenital tumours of the heart. Transversely-striped spindle cells (young muscle cells) or striped muscle cells also occur in some sarcomata of the kidney and testis.

Neuroma. This term is used somewhat loosely to designate not only growths consisting of nerve elements which are rare, but also the more common fibrous, myxomatous, and myxosarcomatous tumours that spring from nerve trunks.

Lymphoma is a term used vaguely to signify both inflammatory and neoplastic formations of which the tissue resembles lymphoid tissue, *i.e.* which consists of a reticulum of cells containing lymphocytes in its meshes.

Glioma is a growth resembling neuroglia.

Sarcoma consists chiefly of cells which tend to multiply without producing formed elements to any marked degree. The cells resemble more or less closely those of embryonic mesoblastic tissues. Frequently in sarcoma the cells are combined with more highly differentiated connective tissues, *e.g.* fibro-sarcoma, glio-sarcoma, etc.

Class II. A second large class is characterized by the presence of epithelial tissue as an integral part of the growth. Such are:

Papilloma, *Adenoma*, *Carcinoma.*

Class III. In a third group the new growths assume the form of cysts which are lined by epithelium which usually has the characters of the epidermis and produces hair and sebaceous secretion. These tumours may be grouped together as *dermoids.*

For surgical purposes it is more instructive to consider tumours in connection with the organs in which they most commonly

occur. Thus many adenomatous and cancerous growths will be noticed in dealing with the surgical pathology of the breast and other organs. It will, however, be convenient to give a brief summary of the chief varieties and favourite localities of the commoner forms of new growth.

Fibromata may arise in almost any part of the body. They are divisible into two classes according as they are dense and firm (hard fibromata) or soft and loose (soft fibromata). The tumours are composed of fibrous tissue and blood-vessels. Some growths, such as epulis and naso-pharyngeal fibroma, which are composed chiefly of fibrous tissue, show a marked tendency to recur locally after removal, and also to form the starting-point of sarcomatous growth. Many fibromata form in the connective tissue of nerves and are generally termed neuromata, under which heading they will be considered. This applies to the remarkable *multiple fibroma* of the skin, or *fibroma molluscum.* The qualification *molluscum* refers to the softness of the growth which may assume various forms. More commonly the skin is studded with projecting rounded pendulous growths varying in size from a walnut or larger to a pin's head. They show no tendency either to disappear or to cause metastasis, and may last forty years or more without undergoing much change. Careful microscopical examination of these growths reveals the presence of a small bundle of medullated nerve fibres in the centre of the pedicle, so that it is probable the growths arise in the sheaths of small nerves, and thus these fibromata may also be classified with neuromata.

Another phase of fibroma molluscum takes the form of a diffuse hypertrophy of some area of the skin which is thrown into loose folds, in which the sebaceous glands may be over active, producing an excessive secretion which undergoes decomposition, and causes a rancid odour. This second form is again allied to a more general hypertrophy of the subcutaneous tissue, or *dermatolysis*, in which the skin can be drawn up from the deeper tissues of any part of the body. In this condition there may be general dilatation of the subcutaneous veins and lymphatics as well as overgrowth of the nerve-sheaths. Persons affected in a marked degree by this

malformation of the skin have been shown at fairs as "elephant men." The condition is allied to congenital hypertrophy of limbs (see below).

Hard fibromata arise most commonly in periosteum (jaw, etc.), fasciæ, skin, ovary, etc. They do not form metastases: if they endanger life it is from their position and size.

Some growths, such as epulis and naso-pharyngeal tumour, which consist chiefly of fibrous tissue, show a marked tendency to recur locally after incomplete removal, and not infrequently they become sarcomatous (see "Fibro-sarcoma").

Œdematous Fibroma. Active or passive congestion of a fibrous tumour causes separation of fibrils and swelling of cells. Under the microscope such a condition may be indistinguishable from myxoma. It is sometimes difficult to distinguish between a fibroma and a myoma when the fusiform cells of the latter are very long and slender. The same applies in less degree to some spindle-celled sarcomata.

Myxomata, or tumours consisting of mucous tissue, are soft translucent growths. On section they exude a sticky colourless fluid which constitutes the chief part of their intercellular substance and contains mucin. Delicate connective tissue is usually also present supporting the branched cells of the growth. Mucous polypi of the nasal fossæ are the commonest examples of myxomata. Patches of mucous tissue are common in growths of other kinds, chondroma, sarcoma, etc.

Lipomata, or fatty tumours, are composed of fat which has usually a more markedly lobular character than normal fat. They are commonest in the subcutaneous tissue, especially that of the dorsal aspect of the trunk and limbs. The axillæ are also a common site. By drawing up the skin that covers them between the fingers, the lobulation is made apparent. Not infrequently subcutaneous lipomata become pedunculated, and pressure on the skin of such a pedunculated lipoma often causes ulceration. When a lipoma is composed of large lobules loosely joined together it may be mistaken for a cystic growth. Ordinary lipomata are surrounded by a fibrous capsule which is adherent to the surrounding tissues, and but slightly connected with the growth. This fact is important in practical

surgery, because for the removal of these growths it is only necessary to open the capsule freely and shell out the contents.

The subperitoneal tissue is not infrequently the seat of fatty tumours. Sometimes they lead to the formation of or complicate a hernia. Lipomata are also met with in the spermatic cord. Further, fatty growths may form wherever there is connective tissue. When a lipoma forms beneath a strong fascia it may occasion a tense swelling of the limb, and be mistaken for a malignant growth. Rarely, lipomata form in the submucous tissue of the bowel, and project into the lumen, where they may start an intussusception.

Parosteal lipoma has been observed in the periosteum of the long bones, pelvis, scapula, vertebræ, and skull. Besides fat such growths usually contain striped muscle. They are nearly always congenital. Fat is sometimes deposited in the synovial fringes in osteo-arthritis, giving rise to a condition known as "arborescent lipoma."

Mixed Fatty Tumours, such are fibro-lipoma and nævo-lipoma. In old-standing lipomata calcification is sometimes observed.

Diffuse Lipoma. Two distinct conditions have been described under this designation. Most frequently it is applied to an increase of fat in the occipital and submental regions of drunkards: a condition which differs from the growths described above, and is rather a hyperplastic than a neoplastic process. Some true lipomata are ill-defined and widespread. Such growths cause great deformity of the fingers when they occur in that situation.

Chondromata are composed either of hyaline or fibro-cartilage. Tumours of the former variety are the more common, and usually are lobulated from being intersected by fibrous tissue which carries blood-vessels. Hyaline chondromata are bluish on section and translucent, like rapidly proliferating cartilage at the epiphyseal line. Fibro-chondromata are white and glistening. Some cartilaginous tumours have soft patches of myxoma tissue alternating with the cartilage. Like mucous tissue, cartilage is a common constituent of mixed growths, especially some forms of sarcoma.

Chondromata occur most frequently in connection with bone.

Towards the ends of the diaphysis groups of ossified cartilage cells are present in normal subjects, and in young adults who have been rickety in childhood islands of hyaline cartilage are sometimes visible to the naked eye when a section of the bone is made. Such remnants of cartilage in the shafts of bones probably form the starting-points of chondromata, such as those that are not uncommon in the bones of the hand and foot, and which arise within the medullary cavity of the bones. In rickets and other conditions (*e.g.* syphilis), cartilage may be formed *de novo* from the periosteum; and the perichondromata, such as those of the femur and other long bones, probably arise in the periosteum. Small outgrowths of cartilage are not uncommon in the laryngeal and costal cartilages. They are usually termed ecchondroses, and are classed with the formation of cartilage at the margin of the articular cartilages in osteo-arthritis; that is to say, they are classed with inflammatory hyperplasiæ rather than with new growths. Here, as elsewhere, the boundary between inflammatory and neoplastic processes is hard to draw. Secondary changes in chondromata, such as ossification, etc., are common, and are more fully noticed elsewhere.

Osteomata. These tumours are fully described in the section on "Diseases of Bone." Some of them, the spongy osteomata, are closely related to the chondromata, since they are covered by a layer of hyaline cartilage. Hard osteomata, resembling the compact tissue of the long bones, or being of even greater hardness, are chiefly met with in connection with the skull bones, but they may occur in any bone. Both hard and cancellous osteomata occur in the bony part of the external auditory meatus. The formation of bone is sometimes observed in the lungs and muscles, and other tissues quite unconnected with the bones.

Odontomata are a varied group of tumours, etc., which arise in connection with the teeth and the embryonic structures of developing teeth.

Myomata are growths composed of long slender, spindle-shaped cells, resembling unstriped muscle, with more or less supporting fibrous tissue in which their blood-vessels lie. They

are most commonly observed in the uterus and broad ligament; they occur also in the ovary and vagina and in the skin. Uterine myomata may undergo calcification. Cells resembling striped muscle fibres occur in certain sarcomata connected with embryonic remains at the hilus of the kidney and in the testis. These sarcomata are called *Rhabdomyomata.*

Neuroma. New growths composed of nerve elements are very rare. The bulbous enlargements that form on divided nerves in amputation-stumps (see p. 51), and elsewhere, can hardly be termed new growths in the sense of the term adopted here. They are the results of the processes which constitute the normal repair of nerves (see p. 47).

Neuromata, consisting of non-medullated fibres, and sometimes of ganglion cells, have been observed in connection with the brain and the suprarenal capsules. The tumours connected with nerves that most frequently come to the surgeon's notice are fibromata, fibro-myxomata, or fibro-myxo-sarcomata that have their origin in the connective tissue of the sheath of the nerve trunks. Such growths may form at the peripheral extremity of the nerves. They are liable to be mistaken for rheumatic nodules and other subcutaneous formations. Occasionally all the branches of a large nerve may be thickened and nodular to such an extent that considerable deformity may result. This has been termed a *plexiform neuroma.* They are generally of congenital origin, and painless. The more common neuro-fibromata or neuro-myxomata usually form solitary growths, and may occasion much pain. When they occur on the roots of the nerves within the spinal canal they first cause pain, then paraplegia.

Angiomata, or **Hæmatangiomata**, are tumours, which consist of a new formation of blood-vessels. Three divisions of these tumours are made (Bland Sutton): *Capillary angioma* or *simple nævus vascularis*, *cavernous nævus*, and *plexiform angioma.* The last is also known as "cirsoid aneurysm," or "aneurysm by anastomosis."

Vascular Nævi (commonly but erroneously called nævi without the qualification—Lat. *nævus* = a mole) are due to a growth of capillary blood-vessels. When they occupy the corium alone

they are known as port-wine marks, such as are common on the face. If they affect the subcutaneous tissue alone, the skin is slightly raised and bluish. When both the corium and the subcutaneous tissue are occupied by the over-numerous, dilated, and often pouched capillaries, there is both elevation of the skin and discoloration. The exact tint of the nævus is probably determined by the degree of dilatation of the newly formed vessels. If they are wide the blood current is slow, and the blood becomes venous; hence the colour is darker than if they are narrow. The arteries which subdivide to form a capillary nævus are usually few in number. Present at birth, or developing soon after, nævi often increase considerably in size during the early years of life; sometimes they undergo involution, probably from thrombosis and organization of clot. The clots not infrequently calcify, forming phleboliths.

Care must be taken to distinguish *telangiectasis*, or acquired dilatation of blood-vessels, from congenital angiomata.

Cavernous angiomata, or venous nævi, are tumours composed of tissue resembling that of the corpora cavernosa and spongiosa of the penis: a fibrous meshwork containing cavities lined with endothelium. What appears to be a capillary nævus at birth may develop into a cavernous angioma, thus showing that there is no sharp boundary between these forms of vascular tumour. Cavernous angioma, like the capillary angioma, is most frequently formed in the skin. When large they may ulcerate, causing severe and even fatal hæmorrhage. They have also been found in the tongue. It is this form of nævus which is often combined with fatty growth—nævo-lipoma. Sometimes when their arterial supply is abundant these tumours pulsate, and so approach in character the plexiform angioma.

Plexiform angioma is a somewhat rare condition. It is also known as "cirsoid aneurysm" and "aneurysm by anastomosis." It is most frequently encountered in adolescents and young adults, though its beginnings are probably present at birth. The growth is either composed solely of arteries or of arteries and veins. In the former an artery and its main branches, or a set of contiguous arteries and their branches, become enlarged, thinned, and tortuous, forming a pulsating tumour. In the

arterio-venous tumour the veins as well as the arteries are enlarged. The danger of the condition lies in the liability of the dilated vessels to rupture. When they occur in the head and face they cause great disfigurement. Ligature of the main arteries which supply these tumours has little or no effect on them. Excision presents great difficulties.

Congenital Moles, or Nævi Pigmentosi, and "Soft Warts." Moles are congenital tumours of the skin. If they are not visible at birth they become so soon afterwards. They are usually pigmented, but occasionally the pigment is wanting; they are then termed "white moles." They may consist of a pigmented area not raised above the level of the skin; more commonly they are raised, and then are either devoid of hair *(nævus pigmentosus spilus)* or covered with coarse hair. Soft warts are of the same nature as moles, only they project above the surface and thus resemble warts, hence their name, "nævus verrucosus." It is important to remember, however, that they are essentially different from warts. In the latter the epithelium is increased in amount as well as the connective tissue and vessels; in the nævus verrucosus, the epidermis is thinned, the projections from the surface being due to accumulations of cells of embryonic connective-tissue type in the papillæ and corium. Hairy moles may be of vast extent, covering large portions of the surface of the body. Their proneness to become sarcomatous has already been alluded to. This same proneness to become sarcomatous is observed in soft warts. The latter, when they

FIG. 33.—Section through part of a congenital mole. The papillary processes of the corium are the seat of small-celled growth over which the epidermis is thinned and stretched. In the deeper part of the section the cells have proliferated into the subcutaneous tissue, showing that the growth had become sarcomatous. × 20.

assume malignant character, extend rapidly, maintaining, however, their warty type. Another very interesting feature of warty moles is their tendency to extend in one direction—thus are formed the "streaks" of dermatologists. One of these streaks may start in a patch of congenital warty moles, say, at the neck, and extend in a linear manner the whole length of the body, or along one of the limbs. Sometimes the direction they take is across the long axis of the body.

The only difference between a soft wart and a mole is that in the wart the accumulation of cells in the papillæ is sufficient to push up the epidermis into processes which project from the surface. In both conditions the epidermis is thinned. A nævoid condition of the lymphatics is sometimes present in papillated moles.

Lymphangiomata may be divided into three groups: Lymphatic nævi, cavernous lymphangioma, lymphatic cysts (Bland Sutton).

Lymphatic Nævi. The commonest example of this condition is to be found in the *sago-tongue*, which shows itself at or soon after birth as little bluish-grey smooth projections which are noticed in clusters on the dorsum of the tongue. Microscopically they are found to consist of dilated lymph capillaries with supporting fibrous tissue in which are a few blood capillaries.

Cavernous Lymphangioma bears a similar relation to the nævus lymphaticus that a capillary vascular nævus bears to a cavernous angioma. The lymph spaces are larger, and the condition is apt to be more widespread. In the condition known as *macroglossia* the anterior two-thirds of the tongue are greatly enlarged, the organ projecting from the mouth. On section the enlargement is found to be due to cavernous lymphangioma diffused through the affected part of the organ. A similar condition sometimes co-exists in the lips and is termed *macrocheilia*. Tissues affected by cavernous lymphangioma are peculiarly liable to inflammation. In cavernous and other lymphangiomata of the skin crops of tense vesicles (*Lymphangioma circumscriptum cutis*) appear during the attacks of inflammation, to which these growths are prone.

Lymphatic Cysts, or *Cystic Hygromata*, are analogous to the cavernous angiomata. They usually lie beneath the deep fascia of the neck or axillæ, but have been observed in other

regions. They are of congenital origin. They consist of one or more cysts surrounded by a wall of delicate fibrous tissue and lined by endothelium. They contain a clear serous fluid. The fibrous tissue round them is often œdematous and may resemble a myxoma in aspect. Clinically they form fluctuating swellings which vary in tension. They are subject to attacks of inflammation which are sometimes followed by spontaneous shrinking. They may or may not increase in size. They are peculiarly liable to become the seat of infective processes after operation for drainage or excision; the latter is usually difficult, often impossible.

Congenital Hypertrophy of the Limbs. The consideration of vascular nævi and lymphatic nævi brings to the mind a condition in which they are both sometimes found. The condition referred to is congenital hypertrophy of the limbs. Not infrequently one or two toes are the seat of a marked enlargement of all the constituent elements. Sometimes all the component elements of the parts are normal in structure save for their excessive development. At other times wide angiomatous tracts occur, or again cavernous lymphangioma may be present and, as in a case seen by the author, during inflammatory attacks may give rise to crops of vesicles on the surface. Or the skin of the part may be dermatolytic. In one case seen by the author there was an extensive hairy mole covering the thigh of a hypertrophied lower limb in a boy aged thirteen who died of sarcoma of the lumbar glands. The simpler forms of congenital hypertrophy may be termed localized giantism.

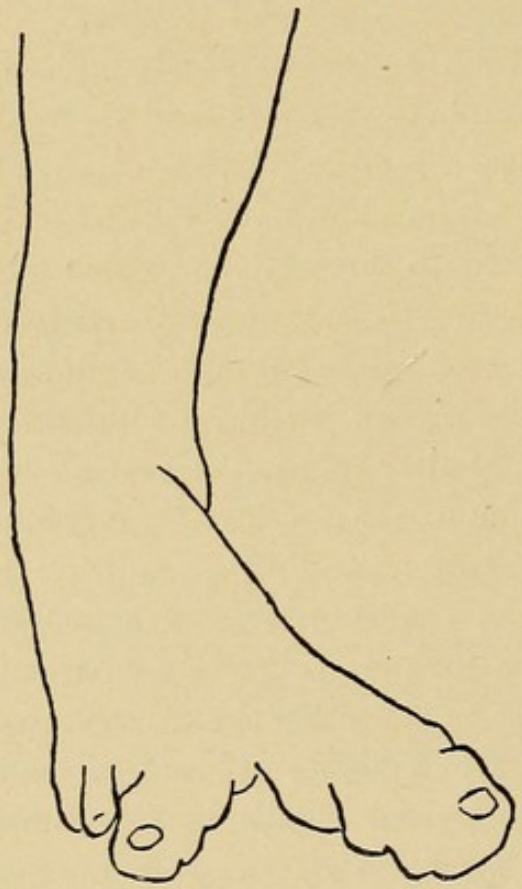


FIG. 34.—A foot, etc., showing congenital hypertrophy of the two inner toes and the inner half of the foot. After S. Hornstein

Sometimes one half of the body is affected; the toes are a favourite seat of the condition (see Fig. 34).

Sarcoma. The general features of sarcomata have already been dealt with. They present a wide range of structure. Both the cellular elements and the intercellular substance differ widely in character. In rapidly growing sarcomata the intercellular substance may be practically absent, what there is being structureless and granular. When the rate of growth is less rapid fibrous tissue may be formed. Sarcomata rarely show any cicatricial contraction and so differ from granulation tissue. In periosteal sarcoma the base of the growth may ossify and cartilage may be formed in the growth. The blood-vessels of sarcomata vary in their structure and numbers, etc. In the more slowly growing tumours they resemble the vessels of the surrounding parts from which they spring. In other cases the walls of the blood-vessels are not developed beyond the single layer of endothelium. In such sarcomata hæmorrhage is common. When the proportion of thin-walled blood-vessels in a sarcoma is very great the growth pulsates. Such a growth is termed an angiosarcoma. Clinically, sarcomata form rounded lobular tumours. In consistence they vary from extreme softness to cartilaginous hardness. Many of them appear to be encapsuled, but when examined by the microscope the tissues beyond the growth are seen to be the seat of some inflammatory and sarcomatous infiltration, and the capsule is found to be merely a condensation of the adjacent structures. The relation of the blood-vessels to the development of the growth has already been considered. Here a few words may be said of the chief types of sarcoma (see Fig. 35).

Round-celled Sarcoma. There are two varieties: Small round-celled and large round-celled sarcoma. The former is a rapidly growing tumour which consists of round cells a little larger than leucocytes, with well-defined nuclei and ill-defined protoplasm. These tumours have little or no intercellular substance, they rapidly infiltrate surrounding tissues, and may arise in almost any organ: brain, testis, mamma, skin, periosteum, etc. The small round-celled sarcoma is one of the commonest of malignant growths. Large round-celled sarcoma is also a common growth

and has frequently an alveolar structure. Its cells may be pigmented.

Spindle-celled Sarcoma. As in round-celled sarcoma so in the spindle-celled growth there is a wide range of dimensions exhibited by the constituent cells. The growths with long slender cells are common in skin, breast, brain, and many other tissues. On section to the naked eye they may be hard to distinguish from fibromata or myomata, and even under the microscope they may be indistinguishable from some myomata. Their cells tend to be arranged in intersecting bundles formed around the blood-vessels of the growth. This arrangement is particularly conspicuous in spindle-celled sarcoma of the pia mater, and justifies the designation of some of these growths as plexiform sarcomata. Large spindle-celled growths without being rare are not so common as the small-celled growth. In a few cases some of the large cells show cross-striation, in which case they are called *rhabdomyomata.*

Lymphadenoma and Lymphosarcoma. No sharp line can be drawn between these conditions. The growths appear primarily in lymphatic glands or in structures such as the tonsils which contain lymphoid tissue. In the former position the glands enlarge and, after remaining separate for a variable time, the growth passes beyond the limits of the glands and invades neighbouring structures. Large lobulated tumours are thus formed, in which areas of caseous degeneration are usually present. Sooner or later secondary growths appear in the lymph glands of the body generally, liver, spleen, kidneys, and bones. This condition is accompanied by great anæmia, and attacks of pyrexia, and is known clinically as Hodgkin's Disease. They may appear in any organ, including the brain and the skin. A common site of lymphosarcoma is in the bronchial glands. The structure of the growth recalls that of lymphoid tissue: small round cells supported by a fine cell-reticulum. When the growth is invading structures such as the bronchi, sections at the advancing margin show a very vascular small-celled sarcomatous growth passing between and replacing the tissues of the invaded part. Other forms of sarcoma may originate in lymph glands. The surgical indi-

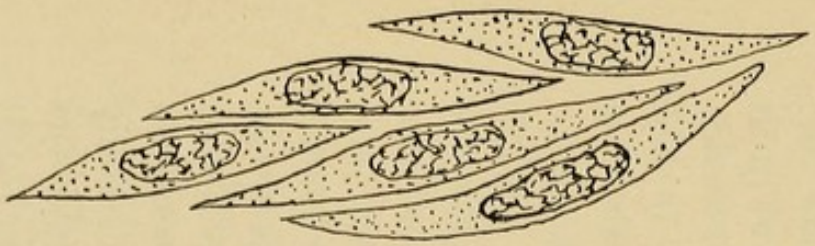
cation in lymphosarcoma is removal before the growth has become generalized.

Alveolar Sarcoma. Sarcomata when examined in sections present an appearance of subdivisions into cell groups by means of the blood-vessels and their supporting tissues. The term alveolar sarcoma is restricted to those in which this subdivision is a marked feature. The most typical alveolar sarcomata are large-celled tumours of which the dividing septa are capillary blood-vessels. An alveolar sarcoma may be melanotic (see Fig. 35, 5), or from the abundance of blood-vessels it may be pulsatile and so deserve the name of angiosarcoma.

Fibro-Sarcoma. A good example of this growth is an ordinary epulis. The superficial part of this growth is rich in small round cells supported by fibrous tissue and spindle cells, the deeper part is more fibrous in character.

Myeloid Sarcoma. This is one of the best characterized forms of sarcoma, and generally has its origin in the medulla of bones; some periosteal sarcomata have also the same character. The leading histological feature of these growths is the presence of numerous large multinucleated cells, which are surrounded by spindle cells and some fibrous tissue. The blood-vessels of these tumours are usually well formed. When they are confined within the medullary cavity of a bone their growth is usually slow and their tendency to form metastases small. Occasionally, however, they are widely disseminated in the various bones of the body. When cut into in the fresh state they have a fleshy appearance and a reddish-purple colour.

Endothelioma. Most tissues of the body contain lymphatic vessels and these contain endothelial cells. In some cases the structure of a sarcoma suggests that the cell-proliferation commenced in these endothelial cells. When these tumours form in fibrous membranes, such as the dura mater or the pleura, sections show under the microscope hollow or solid masses of cells surrounded by fairly thick septa of fibrous tissue, so that it is sometimes impossible to say whether the growth is sarcoma or cancer until its origin and distribution have been carefully considered. Probably it is in the endothelial cells of lymphatics that the cells of many different forms of sarcoma arise, but it



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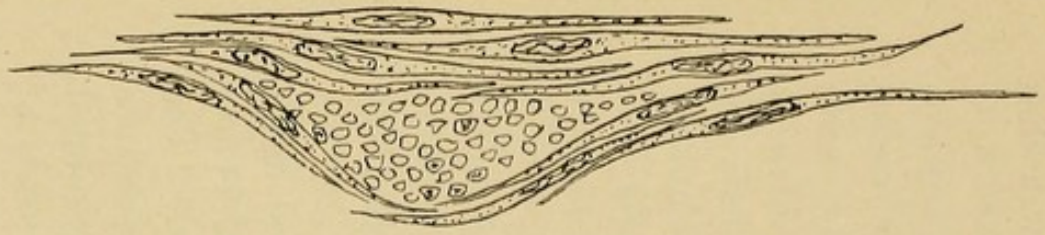

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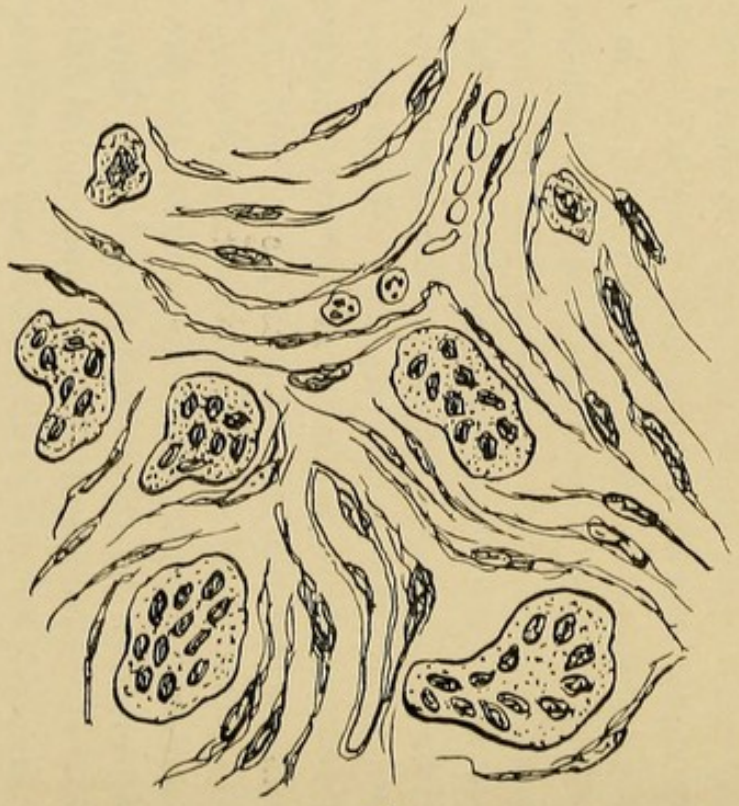

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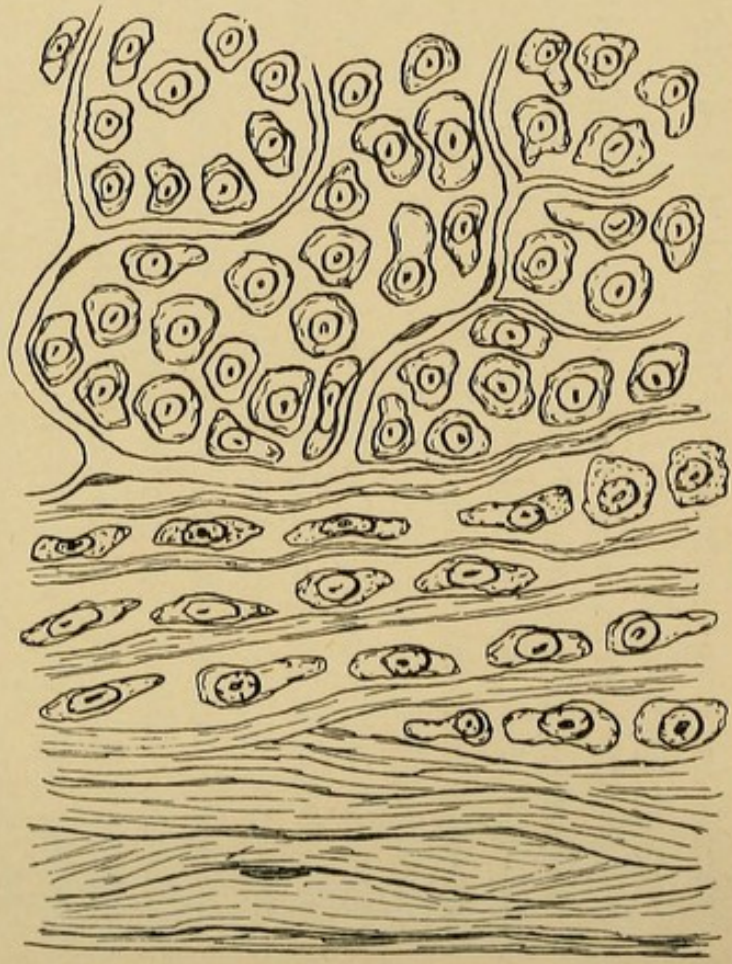

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FIG. 35.—Various types of sarcoma. 1, Large; 2, small spindle-celled; 3, small round-celled; 4, myeloid; 5, large round-celled, alveolar sarcoma. The last was taken from the same section as Fig. 26, p. 93, after bleaching with hypochlorite of lime and staining with picro-carmine. × about 250 diams.

is only when a structure such as that described above is met with that the term *endothelioma* is employed. In endothelioma of the dura mater the central cells of the groups form concentric whorls like the normal concentric cells of the thymus. In sarcoma of the dura mater these cell-clusters may become calcified. The growth is then termed a sand-grain sarcoma or *psammoma*.

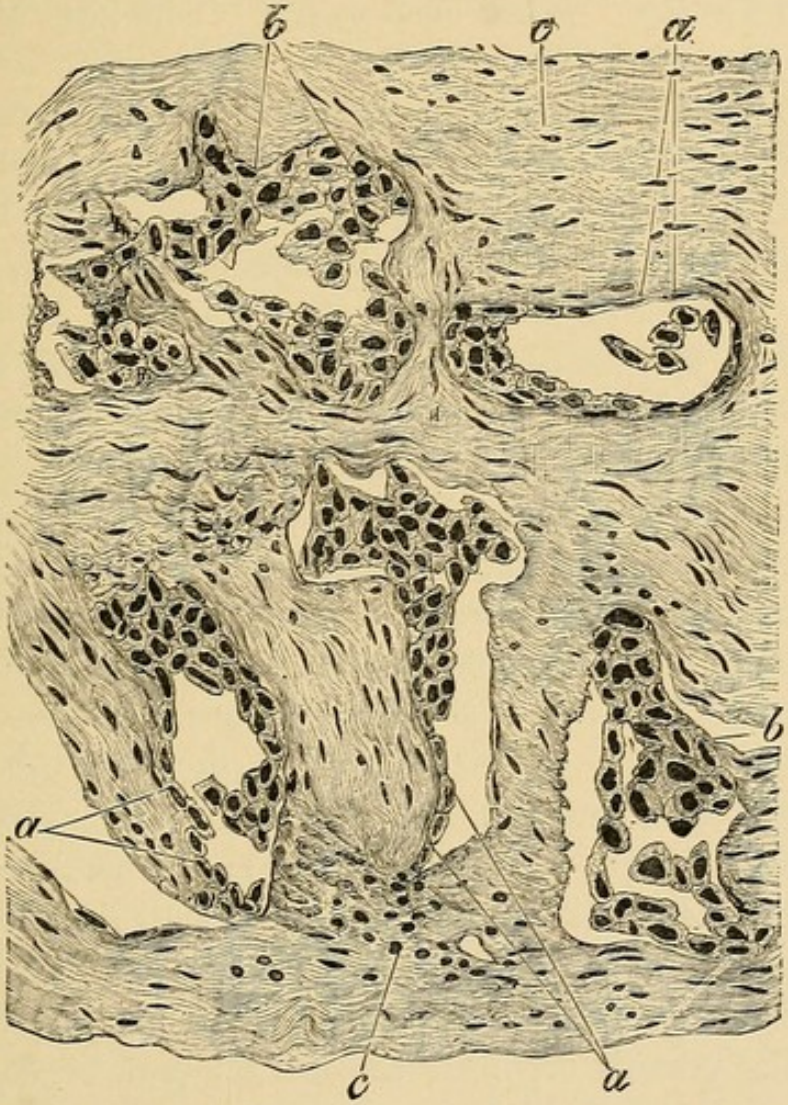


FIG. 36.—Alveolar endothelial sarcoma of the pleura, starting from the endothelium of the lymphatics. *a*, Endothelioid sarcoma cells, seen laterally; *b*, sarcoma cells, seen on the flat; *c*, connective-tissue stroma. × 285. Stained with alum cochineal. From Weichselbaum's *Pathological Histology*.

Cylindroma. The origin of sarcoma in the budding of the blood-vessels and proliferation of the cells which cover the new vascular offshoots has been observed. When the coats of the vessels of such tumours undergo hyaline degeneration the term cylindroma is applied to them.

Melanotic Sarcoma. Some of or all the cells of any kind of sarcoma may be pigmented from the presence of particles of black or brown pigment in the cell-protoplasm. Such growths are termed melanotic. They may occur in very various kinds of tissue, but their favourite sites of origin are the choroid coat of the eyes and the skin. In the latter situation they sometimes begin as a bluish streak at the side of the nail: "melanotic whitlow" it has been called. This form of sarcoma is rapidly fatal from dissemination.

Mixed Sarcomata. This term signifies two distinct conditions: (1) Where different kinds of cells, say, round- and spindle-celled, occur in the same growth; (2) where tissues like bone, cartilage, mucous tissue, etc., alternate with the less highly differentiated parts of a sarcoma.

Glioma is a tumour composed of tissue resembling neuroglia. It occurs in the brain, spinal cord, and the retina. Sometimes the growth proceeds slowly, and then its structure is much like that of normal neuroglia. In other cases the growth is more rapid and, histologically, the tissue is found to contain numerous round cells in the meshes of the finely reticular neuroglia-tissue. These cellular gliomata are designated glio-sarcomata. In the brain they may form large tumours replacing considerable tracts of brain tissue. Not infrequently their central parts break down, forming a cyst which is lined by gliomatous tissue. These tumours are generally of about the same consistence as the brain tissue into which they merge. They are not so common in the spinal cord as in the brain. In about 2000 autopsies I have met with one case of spinal glioma. The growth occupied the central part of the cervical cord, the tissue of which was stretched over the growth. The latter was nearly as thick as the original spinal cord and extended for four inches vertically. Glioma of the retina is not a very unusual occurrence.

EPITHELIAL NEW GROWTHS.

Papillomata are commonest in parts covered by squamous epithelium—skin, mouth, vocal cords, etc., etc. On the skin

they are familiar as warts. They consist of a core of fibrous tissue, composed of the hyperplastic papillæ of the part and their enlarged blood-vessels. This is covered with an equally increased amount of epithelium. In this category come many inflammatory formations to remind the student of the close proximity of new growths to inflammatory new formations, of which syphilitic condylomata, gonorrhœal warts, and the warty condition of the skin around some chronic ulcers will serve as illustrations. Sometimes numerous warts form on the vocal cords and cause hoarseness and other symptoms.

In mucous membranes, especially that of the urinary tract, papillomata also form—thus the villous tumours of the bladder belong to this category. Corns and some cutaneous horns also approach this group. Horns form on a base of hypertrophied papillæ. They are sometimes associated with cancer.

Adenomata are tumours composed of gland-like elements. Thus an adenoma of the rectum or uterus is composed of tubular glands; an adenoma of the breast, of ducts and acini which resemble broadly those of the normal breast. A greater or less amount of supporting connective-tissue always surrounds the epithelial parts of adenomata, and in some cases this supporting tissue assumes the character of one or other form of sarcoma. The growth is then termed an *adeno-sarcoma.*

Carcinoma. The characters of a malignant epithelial growth have been sketched on page 95. It has in this country become customary to speak of a few cancerous growths as epitheliomata and the rest as cancers. Thus the terms squamous epithelioma and cylindrical epithelioma refer to cancers of the squamous epithelial and the columnar-celled types. In this work these growths will be termed squamous-celled cancer and columnar-celled cancer respectively. This not only gives a more uniform designation to the cancerous growths, but will not clash with the terminology in use abroad, where all epithelial growths, benign and malignant alike, are termed epitheliomata.

The subdivision of cancer has yet to be made on a logical basis. At present no more can be done than describe the best known varieties, and leave the description of other forms to be considered with the organs in which they arise.

In general the epithelial parts of cancer tend to reproduce the type of epithelium in which they have their origin. The variations of hardness, softness, etc., have already been dealt with.

Squamous-celled Cancer. The lip, tongue, pharynx and gullet, larynx, skin, and glans penis are the commonest sites

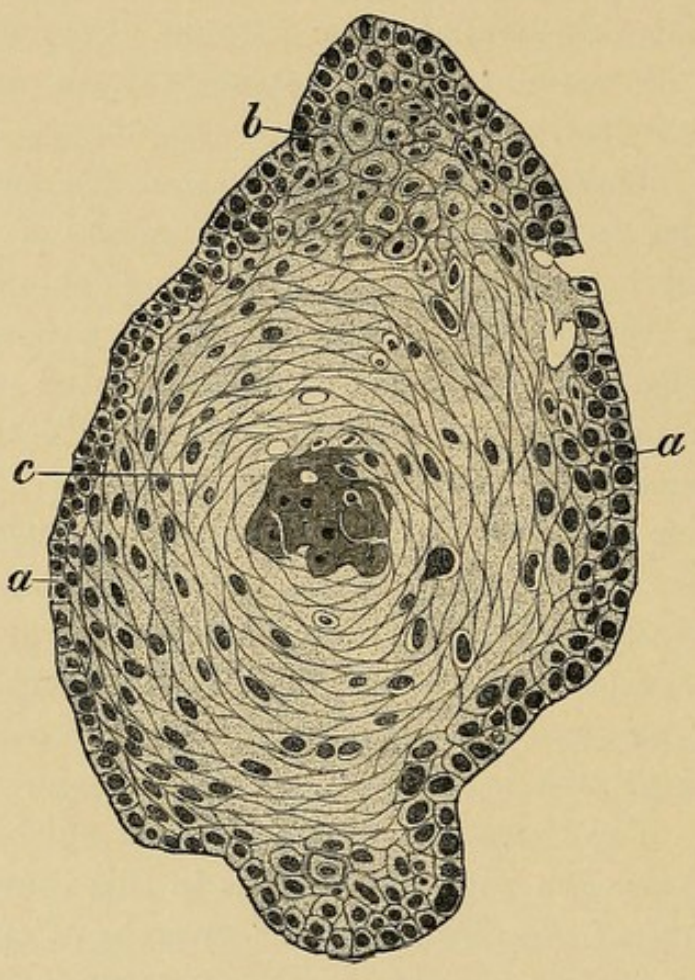


FIG. 37.—Cone of cancer cells in an epithelial carcinoma. *a*, Short cylindrical cancer cells at the circumference of the cone; *b*, spiny cells; *c*, concentrically stratified epidermic globule. × 200. Hæmatoxylin and eosin. Weichselbaum.

of this growth. Two clinical types are recognizable according as (1) the growth forms papillated projections from the surface—warty or proliferating type; or (2) the surface becomes excavated by ulceration—ulcerative type. The two types are frequently combined in the same growth. In both forms an indurated base and a border raised up by firm subjacent cancerous growth are present. Thus to the eye a glans penis thickly covered by gonorrhœal papillomata sometimes closely resembles the

same part when it is the seat of an epithelioma of the warty type. Palpation of the part will at once reveal the presence of induration in the case of cancer and the absence of it in the case of warts.

A section made through a squamous-celled cancer shows to the naked eye the solid white growth. Under the microscope sections show the epithelial down-growth in the shape of solid columns of cells separated by vascular connective-tissue. The larger of the epithelial columns present the cell-nests, which are composed of compressed epithelial cells (see Fig. 37). Scrapings show similar structures.

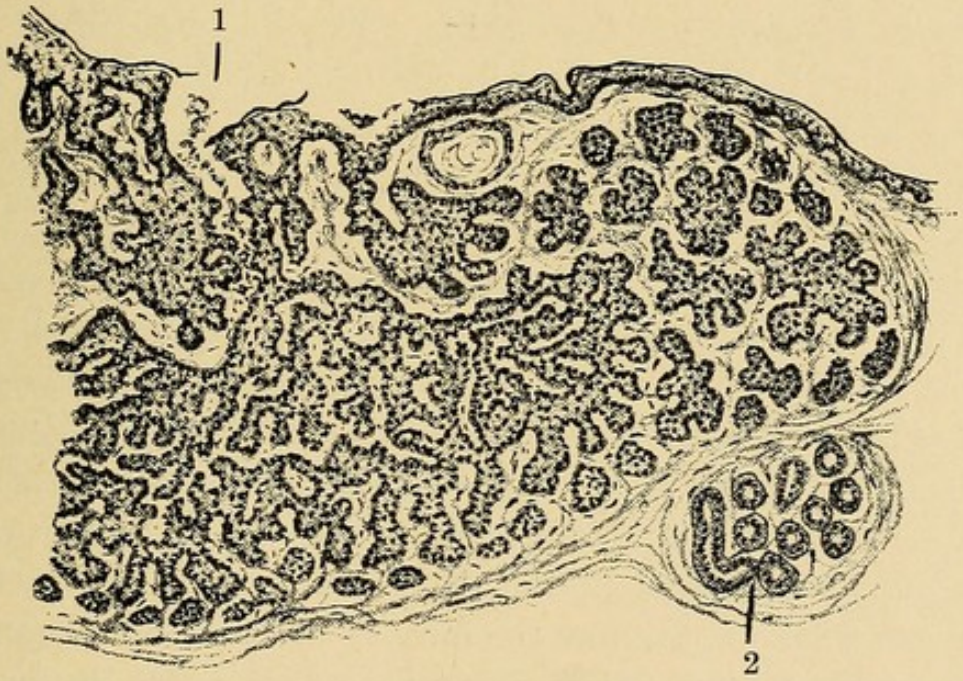


FIG. 38.—Section through a rodent cancer of the face, at an early stage. 1, ulcerated spot; 2, sweat-gland. × 30.

When a squamous-celled cancer is growing in succulent tissue, cystic spaces may take the place of these cell-nests. These cysts are due partly to the immigration of leucocytes, partly to changes in the epithelial cells themselves.

Squamous-celled cancers not infrequently originate in chronic ulcers, simple or syphilitic. They are also met with commencing in scars.

Rodent Cancer begins as a small nodule placed beneath the epidermis. Next it forms either a wart-like lesion or a shallow ulcer. It is most common on the side of the face, but may be

developed on any part of the skin. The cancerous nature of the growth is shown by its progressive and persistent, though slow, invasion and destruction of the parts on which it rests. The formation of new tissue is usually very slight, but the edge of the ulcer can usually be observed to be slightly raised by firm growth. Sometimes the formation of new tissue is more abundant, and the edge of the ulcer correspondingly elevated. This constitutes the "crateriform ulcer."

Histologically the ingrowing processes are composed of smaller cells than those of squamous-celled cancer, and cell-nests are usually absent (see Fig. 38). The lymph-glands may be affected, though this is not common.

Paget's Disease of the Skin. This disease was first described by Paget on the nipple. It occurs also on the scrotum and at the margin of the anus. Though regarded by some writers as an eczema, it has more associations with cancer. It begins as a red patch in the skin, generally at the summit of the nipple. This may become covered with scales. When the scales are absent, the bright rose colour and slightly irregular surface of the patch are noteworthy. It extends progressively in all directions, and is characterized by a definite, often slightly raised and indurated edge. Slight excoriations form from time to time. After lasting from two to twenty years on the nipple, epithelial proliferation extends down the ducts to the breast, where it assumes the form of ordinary breast cancer. Two cases have come under my notice. In the first the disease had existed seven years, and the patch was as large as the palm of the hand. There was no cancer of the breast. In the second the disease had existed for thirteen years, and the patch covered the nipple and areola, and a little of the skin beyond the latter. There was cancer in the breast and axillary glands. In the second patient there was also a rodent ulcer at the upper part of one thigh. Removal of the breast is the only satisfactory treatment of the disease. In the corium both the main features of inflammatory change—enlargement of connective-tissue cells and diapedesis of leucocytes—are present.

Columnar-celled Cancer. This kind of cancer arises in the stomach and intestine. Hard and soft varieties similar to

those described under squamous cancer occur. The ulcerative type is commonest, and is often combined with proliferation. Further particulars will be given below.

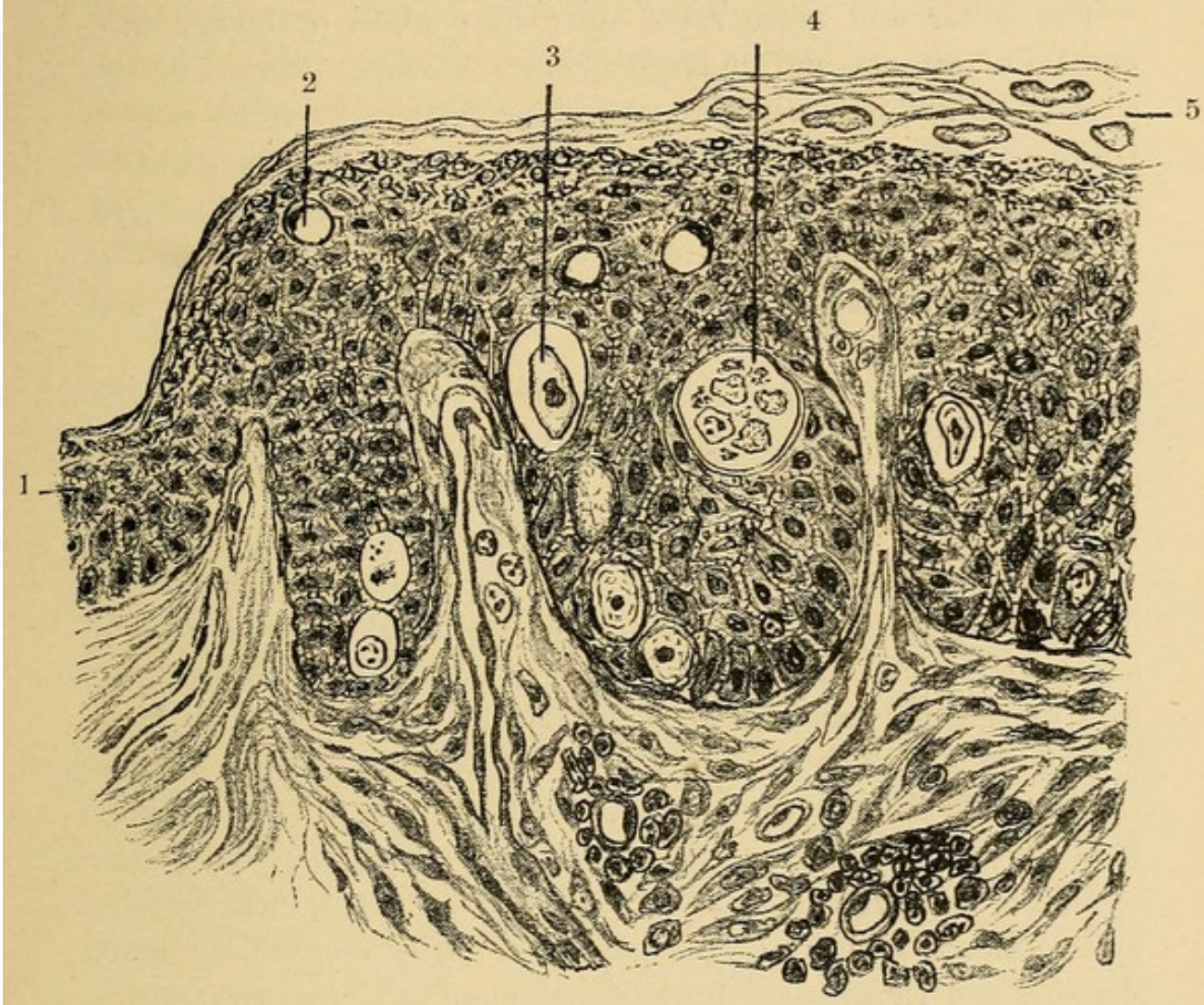


FIG. 39.—Section through the edge of a patch of Paget's disease. 1, Normal epidermis; 2, vacuolated epidermal cell; 3, swollen epidermal cell lying in a cavity; 4, a cavity containing a leucocyte and some granular bodies; 5, dense bodies in the horny epidermis. × 250.

Glandular Cancer. Cancer of organs such as the breast or testis only differ from those commencing on a surface in that the epithelial growth begins in the midst of a solid gland instead of on a mucous membrane. Taking cancer of the breast as an example, hard and soft[1] cancers may be distinguished. The former are the more common. If left to themselves they generally cause death from glandular and general infection in two or three years. But the more slowly-growing or atrophic

[1] The terms scirrhus (= lump of stucco) and encephaloid (= brain-like) only refer to the hardness or softness of the growth.

tumours may last for six or more years without producing ulceration. The hardness of these growths depends on the amount of fibrous (cicatricial) tissue present in them (see Fig. 40).

The softer and more rapid growths contain a smaller proportion of stroma, and undergo a central caseation instead of the cicatrization which occurs in the older parts of atrophic cancer. In one form of breast cancer—duct cancer—the microscope shows that the growth consists of papillated processes covered by cubical or cylindrical epithelium. Duct cancer may be taken as comparable with proliferous cancers of the skin and intestines.

Colloid Cancer. The epithelial cells in the middle of the lobules of the growth undergo colloid degeneration. On section such growths present gelatinous areas. See also "Diseases of the Breast."

Colloid cancer is also met with in the stomach and intestine, in the epididymis and spermatic cord, peritoneum, etc. A mucous degeneration of the stroma often accompanies colloid degeneration of the epithelial parts of cancers.

It is in glandular cancers, such as that shown in Fig. 40, that the alveolar character is most marked. This feature they hold in common with certain sarcomata. The character of the epithelial cells of cancer is more distinctive than their alveolar arrangement. The outlines of cancer cells are usually more definite and sharp than those of sarcomata. This observation is of value in diagnosis. Thus in cancer of the ovary, or peritoneum, accompanied by ascites, groups of large cells with definite outlines may be found in the fluid withdrawn by aspiration.

Cancer may arise in any part where epithelium exists, and may be carried to any vascular part of the body.

The Causes of New Growths. But little is known of the general etiology of new growths. Reviewing for a moment the varied array of new growths just described, they are found to present as wide a range of rate of increase and of habits as they do of histological structure. One of the features they have in common is "autonomy," *i.e.* their independence of physiological control. Even this is not absolute, as is shown by the fact that in women suffering from inoperable

cancer removal of both ovaries has been found to check the growth of the cancer, thus showing that the cells of the neoplasm are affected to some extent by the general nutrition of the body. Many growths such as nævi are congenital. Congenital moles form a striking group of new growths. They have the structure of sarcomata, yet in the majority of persons they do not assume the life-habit of a sarcoma. Some formations classed among

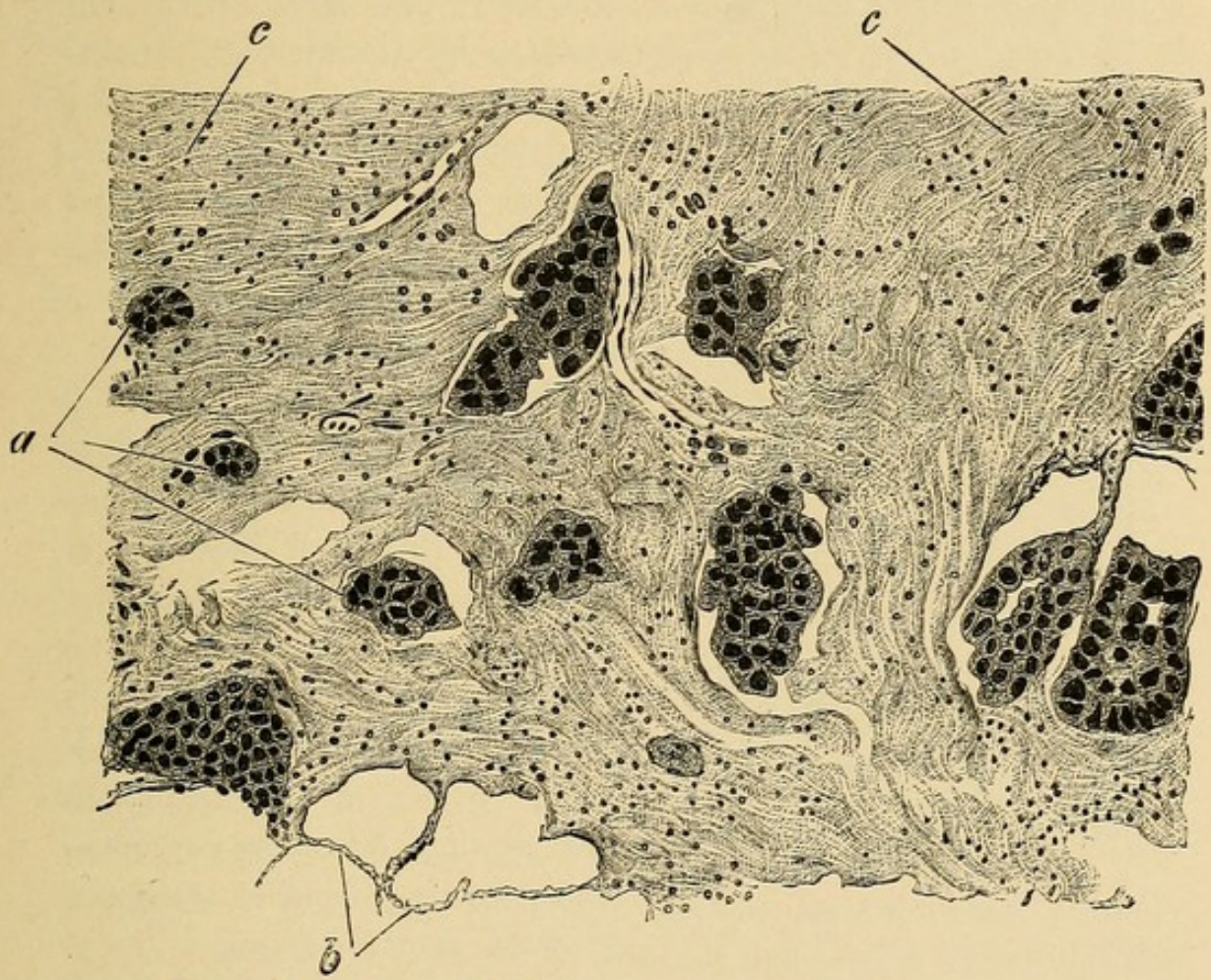


FIG. 40.—Section of a hard cancer of the breast. *a*, Small cancerous alveoli; *b*, empty alveoli; *c*, greatly developed connective-tissue stroma. × 240. Weichselbaum, *Pathological Histology*.

tumours are clearly simple deviations from normal development. Such are cervical auricles, etc. Certain congenital tumours, which occur most frequently at the back of the sacrum, are found on dissection to be really abortive embryos, which under more favourable conditions might have developed into separate individuals instead of forming a tumour-like mass (teratoma). In view of these teratomata, and of congenital tumours of other kinds, the "inclusion" theory formulated by Cohnheim, to the

effect that tumours arise from embryonic cells produced in excess of what is required for the building up of the body, appears to have a very great probability. Yet, as has been shown above, the malignant growths present close resemblances to the inflammatory processes, and many attempts have been made to prove a parasitic origin of cancer and sarcoma. One of the latest of these was based on the view, first advanced in this country by Ruffer, that parasitic protozoa were present in malignant growths. In this view I shared and went even further. The evidence was based on histological appearances, some of which are shown in Fig 41.

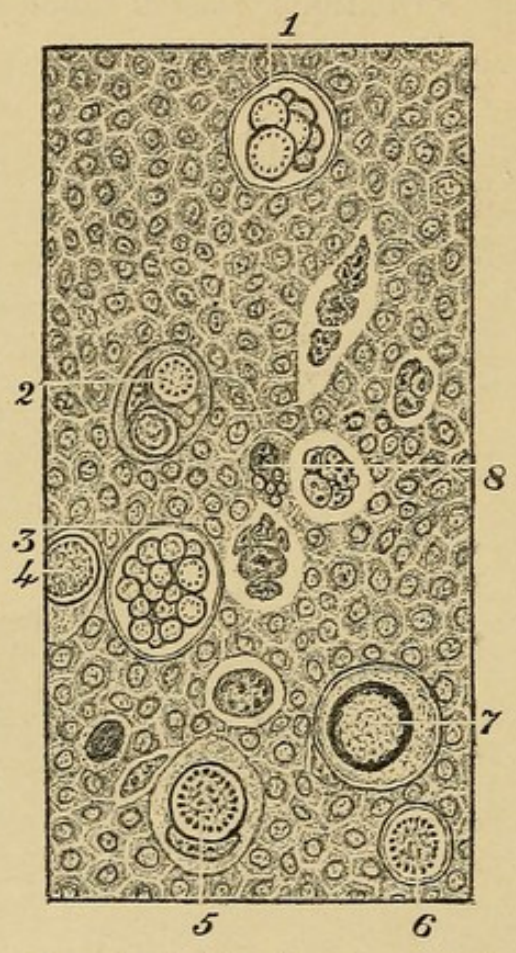


FIG. 41.—Section of a cancer of the penis. 1, Cluster of round slightly granular bodies; 2, an epithelial cell containing two similar granular bodies; 3, a large collection of small granular bodies within what may be a real capsule or only an optical appearance (see the Report of a Morbid Growths Committee, *Transactions of the Pathological Society*, 1894, p. 248); 4, 5, other granular bodies within cells; 6, granular body; 7, a dense body reticulated in the centre; opposite 8, in the middle of the section, an epithelial cell containing five small clear bodies. In the spaces between the cells are eucocytes. × about 400.

The cell-inclusions shown in this figure were described by Virchow as "cells of endogenous origin." Ruffer, D'Arcy Power, and others have pointed out that leucocytes and other bodies are often present in epithelial cells. I may at once admit that in many points I have modified the views I formerly expressed. The parasitic origin of malignant growths is not proven.

A *traumatic* or *irritative origin* has been suggested for some cancers and other growths. Thus, when a sarcoma appears at the site of a previous fracture of a bone, the growth has been attributed to the injury. In many such instances the growth was doubtless present before the fracture, which indeed it determined. Even when the events are really consecutive the evidence warrants no conclusion beyond that the injury may

be contributive, as it is in certain abscesses and tubercular lesions. Sweeps, workers in tar, petroleum, etc., are subject to epithelial growths, but the same objection may be taken against there being a causal relationship between the irritation and the growth as holds in the case of traumata just mentioned. Where papillomatous growths are evidently connected with gonorrhœal or other suppurations it is presumable that these growths are due to a slight degree of inflammatory action lasting for a long time. Hutchinson has shown that the long-continued internal administration of arsenic may be followed by horny and even cancerous growths of the skin. In exactly what way this is brought is not known. It will be seen that but little is established regarding the etiology of new growths. It must, however, be remembered that not many years ago tubercular and other now well-understood formations were included with new growths, and it is to be expected that with the increase of knowledge other members of this group of pathological formations will be explained.

Cysts.

A cyst (Gr. *kystis* = bladder) is a closed sac with fluid or semifluid contents. Cysts originate in various ways:—(1) By the distension of cavities or tubes. (2) By the formation of a fibrous capsule around effusions of blood or degenerated and liquefied tissue. This occurs frequently in new growths. (3) By the growth of parasites. Many cysts are of developmental origin, and will be described in the next section. Examples of glandular or "retention" cysts are the common sebaceous cysts of the skin, mucous cysts of the mouth and of the cervix uteri. The epithelial-lined cavities of new growths very commonly become converted into cysts. Of parasitic cysts, those due to the complex colonies or brood-capsules of the tænia echinococcus are best known.

CHAPTER VI.

DERMOID TUMOURS AND CYSTS, AND OTHER CONGENITAL PATHOLOGICAL FORMATIONS.

DERMOID tumours are generally in the form of cysts, which are lined with skin with the epidermis inwards. The epidermal lining produces sebaceous matter, and so sometimes a dermoid closely resembles a sebaceous cyst. Besides sebaceous matter, hairs, teeth, etc. are sometimes found in the larger and more complicated dermoids, such as those of the ovary and the testis. The tumours are of congenital origin, though frequently they do not show themselves till puberty or later. It is an easy matter to account for many dermoid cysts. They form from distension of relics of embryonic passages, such as the gill clefts, and of lines of coalescence, such as the lips of the medullary groove, etc. These have been termed "sequestration dermoids."[1] The position of such dermoids can be readily surmised from a consideration of the manner in which the body is built up from the three-layered blastoderm. Along the middle line of the back, perinæum, in the scrotum, the middle line of the abdomen, thorax, and neck up to the middle of the lower lip is such a line of coalescence. Dermoid cysts are not common in the dorsal part of this line, and when they occur there they are apt to be mistaken for spina bifida. In the scrotum they have been mistaken for dermoids of the testis (see p. 132). Cysts or dimples resulting from a still greater failure of coalescence are occasionally observed in the skin of the middle line of the thorax and neck. Rarely dermoids have been found within the

[1] J. Bland Sutton, *Tumours*, p. 279.

thorax. Sometimes they are connected with the lung, and then hairs may be coughed up from the cyst opening into a bronchus. Others have been found in connection with the pericardium.

On the face dermoid cysts are common in certain situations, and they may occur in the lines of any of the embryonic facial fissures. Thus about the angles of the eye where the orbito-nasal fissure lies in the embryo, dermoids are common whether beneath the skin or the conjunctiva. A defect of development greater than that which leaves an epithelium-lined cavity results in the persistence of these fissures or parts of them in conditions such as hare-lip, macrostoma, etc. A defect between these two in degree leads to the formation of a *congenital fistula.* The branchial fistulæ are the most important of these. Of the four branchial clefts of the embryo, the first is utilized to form the tympano-Eustachian passage; the other three normally disappear, but in certain families they tend to persist, either as passages blind internally and opening externally at the anterior border of the sterno-mastoid, or joining the pharynx with the surface. More rarely they open into the pharynx but are closed externally. Their external openings when present are, approximately: the second opposite the angle of the jaw, the third below the level of the hyoid bone, and the fourth near the sterno-clavicular articulation. Their internal orifices are in the neighbourhood of the tonsil. If the lower fissures remain and open into the pharynx the upper end of the passage is directed between the two carotid arteries and above the superior laryngeal nerve. When the passage is closed externally but open internally a *pharyngeal diverticulum*[1] is formed. Unless this malformation becomes the seat of abscess from the entry of food into it, it is not likely to be discovered during life.

It has been mentioned above that the first pharyngeal cleft is utilized as the tympano-Eustachian passage. In connection with its outer opening the pinna of the ear is developed by the fusion of six tubercles. In a similar manner abortive auricles are not infrequently developed at the points at which the remaining pharyngeal clefts open on the surface. Such

[1] See a case by M. Watson, *Journal of Anat. and Phys.*, 1874.

auricles consist of skin and cartilage. In the union of the six or more tubercles to form the pinna, fistulæ may be left or dermoid cysts arise in included portions of epiblast.

Dermoids of the Palate. These are solid tumours covered *externally* with skin. They form in the line of union of the two halves of the palate, and may project either into the mouth or naso-pharynx. They may be compared with rectal dermoids. Adenomata and epithelial pearls are met with in the same situation.

Dermoids of the Pituitary Gland. Dermoid tumours and adenoma have been found within the skull growing from the pituitary gland.

Dermoids of the Scalp are important because they may be attached to the dura mater by a pedicle which passes through a hole in the bone. They are commonly met with in the middle line, especially at the site of the anterior fontanelle or at the occiput. In infants they are liable to be mistaken for meningoceles, in adults for sebaceous cysts. The skin covering them may become ulcerated through and then suppuration may extend to the meninges along the pedicle of the cyst, causing fatal meningitis.

Cysts and tumours may develop in connection with the original duct of the thyroid gland (see below).

Dermoids of the Rectum and Congenital Sacro-Coccygeal Cysts.—*Post-rectal dermoids* may be formed between the hollow of the sacrum and the rectum. They are rare and may contain teeth. If they remain small they will probably give rise to no symptoms, and so be overlooked. They may attain a large size, bulging into the pelvis and pushing before them the peritoneum.

Rectal dermoids form pedunculated tumours within the rectum. Their presence may be made known by a wisp of hair which covers their surface protruding through the anus. They are covered externally with skin, like the palatine dermoids. They are to be distinguished from ovarian dermoids which have ulcerated into the rectum and discharged their contents per anum.

Sacro-coccygeal Cysts and Adenomata. These develop from

the post-anal gut or the neurenteric canal of the embryo. They consist of a fibrous wall connected with the lower part of the coccyx or sacrum. They resemble somewhat the thyroid gland in structure, and may contain cysts filled with ropy mucus. They usually project on the surface of the body, forming conspicuous tumours between the buttocks.

Dermoids of the Ovary and Testis. It is not difficult to understand the development of dermoid cysts at the site of embryonic lines of coalescence, etc., but it is less easy to understand how they come to form in the interior of the ovary and the testis. These organs are developed at the back of the abdomen near the middle line, and the explanation usually given of the formation of dermoids within them is that during development in some way portions of the outer layers of the blastoderm become included within their rudiments. Neither this nor other theories can be regarded as wholly satisfactory.

Ovarian Dermoids are among the commonest examples of the condition. They may remain of small size, and cause no symptoms during life. Frequently, however, they attain considerable dimensions, and then project beyond the organ in which they are developed. They may become pedunculated and their pedicles may then be twisted, causing them to undergo first congestion and then necrosis, causing pain and other symptoms. Sometimes they contract adhesions to the omentum, and in that case, by the intestinal movements, they may be pushed away from the ovary, and be found at some distance firmly connected with the omentum, their attachment to the ovary having first been stretched and then caused to give way. Anatomically they consist of a fibrous wall lined with skin. They often present a massive intracystic projection in which may be embedded complicated pieces of bone and cartilage. The skin which lines them frequently bears teeth, and sometimes even mammary glands. They usually contain hair and sebaceous matter. Now and again adenomata lined by mucous membrane are developed from their lining of skin.

Dermoids of the Testis must be distinguished from the somewhat similar tumours which arise in the raphe of the scrotum. They resemble the dermoids of the ovary, but they are much less

common. Since they offer a good example of the more complicated kind of dermoid tumour, an illustration of one of them is given in Fig. 42.

Between the tunica albuginea and the wall of the dermoid was a little fibrous tissue, but sections examined under the microscope failed to reveal any testicular tubules.

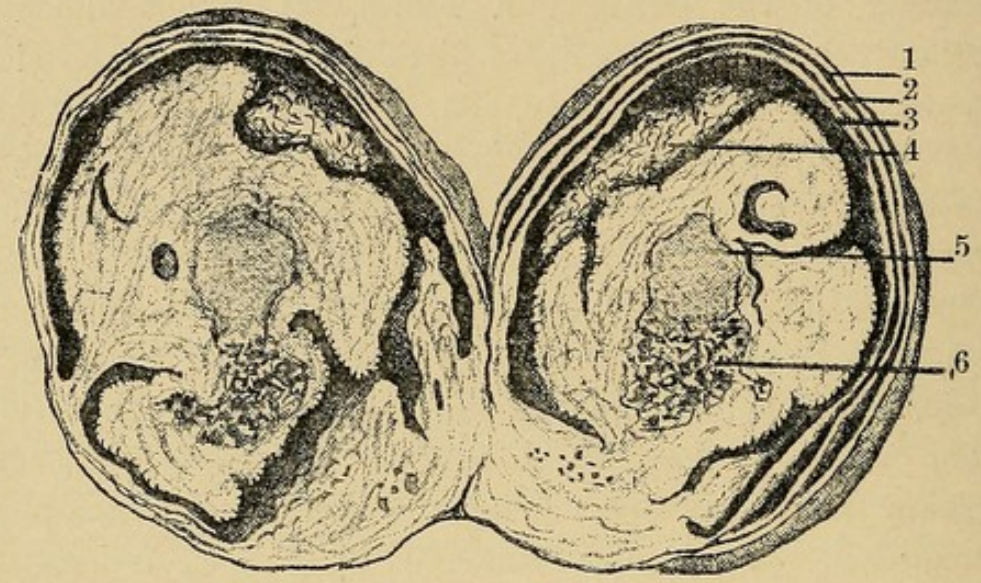


FIG. 42.—Section through a testis distended by a relatively large dermoid. From a one-year-old Hindu child. In the right half of the section the layers have been dissected. 1, Tunica vaginalis; 2, tunica albuginea; 3, wall of dermoid consisting of skin; 4, sebaceous matter and hair free in the cavity of the cyst; 5, cartilage; 6, bone in a large fibrous intracystic projection, which is covered by hairy skin. Nat. size.

Implantation Cysts. If a blunt instrument is pushed through the skin into the deeper tissues, it may carry with it a separated fragment of epidermis which may remain alive and produce a small mass of epidermis surrounded by vascular tissue. The epidermal cells then tend to form a cyst, those cells nearest the vascular tissue keeping to the type of the deeper layer of the epidermis, while those that lie in the centre become horny and are shed from time to time, and constitute the contents of what might be termed an artificial dermoid cyst. Similar implantation cysts have been observed in the cornea as the result of a portion of conjunctival epithelium being carried in by accidental injury or in operating.

Teratomata. A teratoma is a tumour which represents an abortive and suppressed fœtus attached to an otherwise normal individual. Though apparently a shapeless mass it proves on investigation to contain anatomical parts, skeletal,

visceral, etc., which show that it really belongs to the same category as double monsters. Their production is due to the same process that results in the formation of twins attached to a single placenta. The single ovum produces a blastoderm on which two embryonic areas appear. If the two embryos develop equally and fully, homologous twins result. If the two remain joined together at one part a twin monster is produced. If one fœtus develops vigorously, and in the other development is arrested at an early stage in the whole or part of the body, the weaker fœtus remains attached as an excrescence on the stronger. Teratomata are commonest at the back of the sacrum. They seldom admit of removal.

Tails. In the region of the coccyx tail-like appendages are sometimes observed. Many are dermoids, teratomata, etc. In one such caudal appendage I found a central core of muscle terminating in a tendon. The investment consisted of skin and subcutaneous tissue. It was caused by an adhesion of the amnion which had drawn out the superficial embryonic layers—an "amniotic band" in fact. True tails containing vertebræ have not been found in man (Sutton).

CHAPTER VII.

MALFORMATIONS.

CONGENITAL malformations are so varied in kind that Teratology, the part of pathology which deals with their origin, is a wide science in itself. Here only conditions of surgical importance will be dealt with. It may be observed that the same deformities which occur in man are met with in animals. In Fig. 43 is represented one of a litter of five kittens which were born all gravely deformed. It is still believed by many that the quickened emotions of women during pregnancy have the power of producing structural changes in the embryo. Though many strange coincidences occur no case that the author has examined has presented even a possibility of maternal mental impressions having any part in the production of deformity. Many deformities are due to simple mechanical conditions, others are readily understood in reference to the normal processes of development. Other malformations are the result of reduplication of normal parts, as in cases of supranumerary digits.

FIG. 43.—A kitten presenting a double hare-lip, club hands with some digits suppressed, and a condition of the lower extremities akin to talipes equino-varus of man. This kitten had also a cleft-palate. The left eye was open at birth. From a photograph.

In the consideration of the origin of dermoid cysts it is found that many can be traced to defective coalescence of parts that either arise in

the embryo as paired processes, which in the normal condition form single median structures by fusing one to the other, or by similar want of complete obliteration of fissures, ducts, or passages that are present at certain stages of embryonic life but which disappear in the course of development. It will be found also that most of the congenital deformities which are of surgical interest arise by a still greater defect in the same developmental processes. It will be less fatiguing to study the majority of these defects in the same chapter instead of placing them in the sections dealing with special organs. They should be studied with the aid of a simple account of the normal development of the body.

Congenital Defects of the Cerebro-spinal Axis.

Congenital Hydrocephalus is due to distension of the ventricles of the brain with fluid, and is frequently associated with spina bifida. The distension of the brain entails great enlargement of the skull. In pronounced cases the greater part of the skull is membranous at birth. In such cases the fœtus perishes during birth from compression and rupture of the brain. In more moderate cases the child may be born alive and then for years the head may continue to enlarge until it reaches a very great size, or, on the contrary, the condition may remain of moderate degree. If we compare congenital with acquired chronic hydrocephalus it might be expected that the former, like the latter, would be due to blockage of the foramen of Magendie, or of the iter, etc., that is, a stoppage in the drain-way from the lateral ventricles.

Cerebral Meningocele is a protrusion of the meninges of the brain through an aperture in the skull. This condition is a possibility, but on dissection of congenital cysts of the cranium that in life are diagnosed as meningoceles, it is nearly always found that their cavity is continuous with that of one or more of the cerebral ventricles, and that they are lined with ependyma resting on pia mater, with a little brain matter intervening near the aperture in the skull. They are thus for the most part

syringo-encephaloceles,[1] comparable to the syringo-myeloceles about to be described in the spinal region, and may be regarded as cases of partial hydrocephalus. They are covered externally with skin, which may be extremely thin over the convexity of the cyst, and under this by a layer of fibrous tissue continuous with the pericranium and dura mater. Internal to this, as stated above, is a lining of pia mater and ependyma. The favourite sites for these cysts are the foramen magnum, the posterior fontanelle, and the root of the nose. When they are small they are compatible with survival to adult life, when large they usually cause death from ulceration and meningitis. They are to be distinguished from cephalhæmatomata and from hydrencephalocele.

Spina Bifida is a congenital defect consisting in the failure of union of the laminæ of one or more vertebræ associated with defective formation of part of the spinal cord or its membranes. Six grades of the deformity may be recognized.

Myelocele. The medullary groove remains open in the region affected. What should be the epithelial lining of the central canal of the cord covers a more or less oval area which, in the living child, looks like a capillary nævus in the middle line of the back. The central canal opens at the upper end of the area, and from the opening the cerebro-spinal fluid drains away and thus causes death in a short time. This form of spina bifida is not so rare as once was thought.

Syringo-myelocele. The central canal is dilated in the affected region, the dilated cord is thinned and adherent to the skin in the middle line. This condition is not amenable to treatment. If the meninges are distended with fluid around a syringo-myelocele, the condition is termed *syringo-meningocele.*

Meningo-myelocele. The spinal cord is formed without marked dilatation of the central canal, but remains attached to the surface epiblast in the middle line. The meningeal space is dilated, and thus a tumour is formed. This variety of the spina bifida may often be diagnosed by a median depression which marks the adherence of the spinal cord. In meningo-myelocele some cures have been effected by injection of a fluid[2] containing

[1] Gr. *syrinx* = tube, *encephalon* = brain, *koilos* = hollow. [2] Morton's.

iodine (gr. x.), iodide of potassium (gr. xxx.), and glycerine (℥j.). Plastic operations have been successfully performed. Frequently there is congenital angular or lateral deformity of the spine associated with this and other forms of spina bifida. Patients who suffer from spina bifida are frequently partly or wholly paraplegic owing to inflammatory changes in, or defective structure of, the part of the cord or the nerves involved in a spina bifida.

Meningocele is a protrusion of the spinal membranes without the cord or nerves through the defective part of the spine. A meningocele may protrude through the normal aperture at the back of the sacrum. They are commonest in the sacral region. Sometimes they have been observed to form pedunculated cystic tumours.

Spina Bifida Occulta is the slightest degree of the malformation. The vertebral arches are defective, but there is no tumour because there is no protrusion of cord or meninges, but usually a small scar marks the site of the defect. On the skin around this scar a growth of hair like that of the head occurs, and often it is this striking phenomenon that causes parents to bring their child to the doctor. Even in spina bifida occulta changes in the cord and nerves may show themselves by the formation of a perforating ulcer of the foot, paralysis of the sphincters of the anus and bladder, spastic paraplegia, etc.

Congenital Defects of the Alimentary Tract.

Hare-lip and Cleft Palate. These deformities arise from arrest of development of parts of the embryo which go to form the face and buccal cavity. In order to understand them, a little study of development is required. It will be remembered that in the early stage of the folding of the embryo from the rest of the ovum, the head forms a blunt elevation (head-fold), and is without mouth or nose. The formation of the mouth begins before any of the parts of the face appear, as a depression (*stomodæum*) at the under aspect of the head-fold, and deepens until it meets the upper end of the intestine which constitutes the pharynx. About the eighth week the tissues at the line of

meeting disappear as if by pressure-absorption, in the form of a vertical slit, which constitutes the aperture of the fauces. The formation of the nasal fossæ begins about the same time as the mouth. The nasal fossæ are at first widely separated, and do not communicate with the mouth. The building up of the face begins by the formation of one median (fronto-nasal) and two lateral (maxillary) processes which grow out from the embryonic head around the stomodæum. The single median process arises on the dorsal aspect of the mouth, grows downwards and forwards, putting out two lateral processes which pass between the rudimentary eye and the outer side of the nasal pit on each side; meantime, from the outer angles of the median part of the fronto-nasal process grow out to form and deepen the inner part of the nasal pits, which are thus converted into deep grooves which open behind into the mouth. The middle part continues to grow and forms the upper lip, and more deeply, the premaxillary processes from which the upper central incisors are subsequently developed. At the same time that the nose and the middle part of the upper lip, etc., have been forming, the two maxillary processes grow forward from the sides of the mouth, closing in the floor of the nasal fossæ, and fusing with the lower end of the fronto-nasal process, thus completing the formation of the upper lip superficially and deeply, joining the premaxillary processes, and completing the rudimentary upper jaw. If the front of the maxillary process fails to unite with the middle part of the upper lip (derived from the fronto-nasal process), *hare-lip* is the result, and is single or double according as the failure is present on one or on both sides. If the deeper part of the maxillary process fails to unite with the premaxillary process, a complicated hare lip results, as described below.

The roof of the mouth, or palatine arch, which separates the mouth from the nasal passages, is derived from two horizontal processes which spring from the deeper and inner part of the maxillary process. If these palatine plates, from which the hard and soft palates and the uvula are formed, fail to unite, cleft palate results. The cleft may be partial, *e.g.* there may be only a bifid uvula, or a bifid uvula

and cleft of the soft palate, or the whole palate may be cleft. With complete cleft palate, maxillo-intermaxillary clefts and hare-lip on one or both sides frequently co-exist. The formation of the septum between the nasal fossæ occurs as an outgrowth from the premaxillary processes. In this outgrowth the vomer is developed, and hence in cleft palate with a double intermaxillary cleft, the central incisors appear at the extremity of the nasal septum.

Imperforate Pharynx. This term is applied to a condition in which the pharynx, instead of being continuous with the œsophagus, ends blindly about the level of the cricoid cartilage, and is connected with the œsophagus only by a solid fibrous cord. The œsophagus in such cases opens into the trachea. This defect is incompatible with life, though the infant has been known to live for thirteen days without any food entering the stomach.[1]

The origin of this deformity has some connection with the formation of the pulmonary diverticulum from the part of the alimentary canal that becomes the œsophagus. Instead of the pharynx being imperforate, there may be a narrowing opposite the cricoid. In such cases a pouch forms from the pharynx between the œsophagus and the spinal column. Such a pouch may fill with food, and cause difficulty in swallowing by pressing on the œsophagus. In some patients severe symptoms have not shown themselves until mature or even advanced age has been reached.

Meckel's Diverticulum. The formation of the alimentary canal takes place by the closing of the edges of a groove on the under surface of the embryo. The last part of this groove to close is at the umbilicus, where the vitello-intestinal duct joins the intestine. This duct normally disappears by absorption right up to the intestine. The atrophic process is sometimes excessive, extending to the bowel, and producing at the site of the junction of duct and intestine a narrowing or an obliteration of the gut. In the latter case there is a congenital intestinal obstruction. If the disappearance of the duct only occurs at the navel, there is produced a congenital pouch communicating

[1] C. Kessick Bower, *British Medical Journal*, March 6, 1897, p. 586.

with the intestine—this pouch is termed a *Meckel's Diverticulum.* It may either lose its connection with the navel or remain attached there. If it remains attached it may either be closed[1] or remain open on the surface as a congenital fæcal fistula. Or, again, the middle part of the duct may disappear, and the outer part remain open at the umbilicus as a blind fistula. From the latter adenomatous tumours may develop.

Fig. 44.

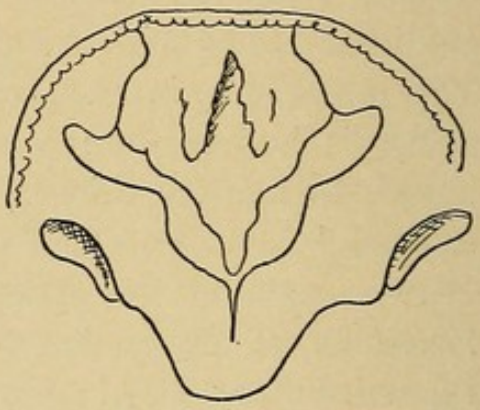


Fig. 45.

Fig. 44.—An average case of imperforate anus. The rectum ends blindly opposite the tip of the coccyx. There was no anal depression in this case.

Fig. 45.—The pelvic outlet from a case in which the rectum ceased opposite the upper end of the uterus to which it was attached. The ischial tuberosities are close to one another and to the tip of the coccyx.

Imperforate Anus. Just as the mouth becomes continuous with the anterior (or upper) extremity of the alimentary canal by the oval depression becoming continuous with the pharynx at the fauces, so the lower end of the canal at first closed is opened by its meeting a depression of the surface of the body, and the subsequent disappearance of the tissues which separate the depression of the surface from the lumen of the bowel. The depression (*proctodæum*) is at first common to the bowel and uro-genital passages, but later by the meeting of lateral processes across the middle line the perineum is formed.

Various degrees of the imperfection occur at the lower extremity of the bowel, and may be tabulated.

[1] For some possible effects of a Meckel's diverticulum, see the section on "Intestinal Obstruction."

1. The rectum and anus are continuous, but there is a narrowing at their junction.

2. The margins of the anal depression may become coherent, forming a membrane continuous with the surface of the skin.

3. In some cases only the anterior and posterior lips of the anal depression become coherent, so that the anus is divided into two lateral openings.

4. The anal depression and the lower end of the bowel are both developed, but the tissues between them (*proctodeal membrane*) fail to undergo absorption. The septum can be seen bulged downwards by pressure of meconium.

5. The anal depression may either be as in (4) or altogether absent, and then the lower part of the bowel may open in an abnormal direction—(*a*) into the vagina, (*b*) into the bladder,[1] (*c*) into the urethra, (*d*) at the raphe of the scrotum or elsewhere.

6. The anal depression may be present or absent, but the lower part of the rectum may be undeveloped, as shown in Figs. 45 and 46.

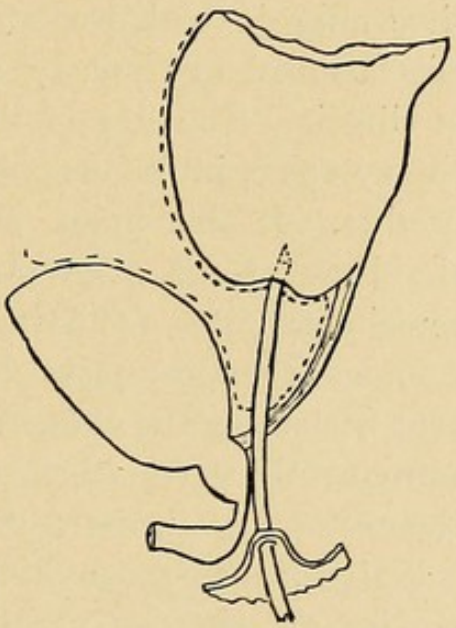


FIG. 46.—The rectum, bladder, etc. in a case in which there was no development of the lower part of the rectum. The anal depression was present and a trochar is represented to show what would have been the effect of exploring with this instrument.

Formerly it was the practice in these cases to seek for the blind end of the rectum by passing in a trochar in front of the coccyx. The effect in cases where the lower part of the rectum is undeveloped is shown in Fig. 46. The trochar traverses a part of the peritoneal cavity. When the trochar is removed meconium escapes into the peritoneum, and fatal peritonitis results.

There is an important indication by which the surgeon may surmise the graver defect of development, *viz.* in cases where

[1] In this condition cystitis occurs soon after birth, *i.e.* when bacteria have gained access to the intestine.

the lower part of the rectum is absent the parts at the pelvis outlet are crowded together as shown in Fig. 45.

In an average case of imperforate anus, such as that shown in Fig. 44, the ischial tuberosities and the pubic symphysis and coccyx have the normal degree of separation.

Congenital Defects of the Penis and Bladder.

Epispadias and Hypospadias. After the division of the common cloaca into two parts by the formation of the perineum the anterior division or uro-genital aperture is for a time alike in both sexes, and has the form of a narrow vertical slit bounded in front by a prominence, the rudimentary penis or clitoris. The lips of the slit which in the female remain as the nymphæ, in the male unite to complete the formation of the urethra. If this union fails completely the urethra opens at the back of the scrotum, and the two halves of the scrotum remain separate. This is complete *hypospadias.* Partial is more common than complete failure. Thus the part of the urethra that perforates the glans frequently remains open below, and not infrequently the urethra opens just in front of the scrotum, all the rest of the urethra remaining open.

Epispadias is a condition in which the urethra remains open on its anterior or ventral aspect (named "dorsum" of the penis in surgical works). The defect seems to be of similar origin to that about to be described.

Extroversion of the Bladder is a condition in which the bladder is open on the anterior aspect of the abdomen, the wall of which is deficient from the pubes to the umbilicus. The pressure of the intestines may push the bladder forwards so that its red mucous surface resembles a growth. The orifices of the ureters can be detected by their giving vent to jets of urine at intervals. In this condition the pubic bones and the insertions of the recti are separated by an interval. In the male epispadias is also present. The testes are usually retained. An explanation of the deformity has been suggested by Shattock, who believes it to arise from an excessive extension of the depression which forms the cloaca from which the anus and uro-genital sinus are

formed. If this depression extended up to the umbilicus and deeply into the base of the allantois which becomes the bladder, this grave defect would be accounted for.

Congenital Malformations of the Female Genital Organs.

Congenital Hypertrophy of the Clitoris. The clitoris may be as large as the corresponding organ in the male, and in such a case it may be difficult to decide whether a child is a female or a male with complete hypospadias. If in the latter condition the testes have descended into the halves of the split scrotum the distinction is readily made. If not, it may be necessary to make a closer examination to prove the presence or absence of the vagina and uterus.

Atresia of the Vulva. The depression in the surface of the embryo for the formation of the anus and vulva may fail to form, or the anal part may be formed whilst the anterior part (*uro-genital sinus*) is wanting. In the latter case the vulva will be absent. This condition has only been found in monsters.

Persistence of Cloaca. Among the forms of imperforate anus (see p. 141) is one in which the rectum opens into the vagina. This is really due to incompletion of the recto-vaginal septum.

Absence of the Urethra. This is due to a failure of formation of the urethra, so that the bladder leads directly into a long narrow vestibule which opens into the vagina. In other words, there is a persistence of the uro-genital sinus.

Imperforate Hymen. The hymen, which is placed at the entrance of the vagina in the normal virgin condition, may have the form of a crescentic fold with its free edge in front, or it may be a diaphragm attached all round just above the level of the meatus of the urethra. The latter condition is normal when there is a central perforation or slit, pathological when it is imperforate. Imperforate hymen results from the excessive formation of the hymen, which appears in the fifth month of intra-uterine life. An imperforate hymen is abnormally thick and tough. The condition may be overlooked until menstruation appears; after this event at each period menstrual blood accumulates in the vagina above the hymen. Sometimes

the latter gives way and the fluid escapes. More frequently the vagina becomes more and more dilated until it may fill the whole pelvis. Later, first the cervix then the body of the uterus becomes filled with blood. Rupture of the uterus or tube into the abdominal cavity, or septic inflammation following spontaneous rupture or operation may cause death.

Atresia of the Vagina, etc. The vagina may be occluded by adhesion of its opposed walls in intra-uterine life, or there may be a complete absence of the vagina owing to the non-formation of the whole or the lower parts of the Müllerian ducts from which the Fallopian tubes, uterus, and vagina are developed. The *vagina* may be divided throughout by a vertical antero-posterior septum, the remains of the division between the two Müllerian ducts which normally coalesce in their lower parts to form the uterus and vagina. In the same way the not infrequent occurrence of a double uterus, or of a two-horned uterus, is explained. In the former condition the parts of the Müllerian ducts which should fuse together to form the uterus remain separate. In such cases the vagina may be divided by a septum, or it may be normal. In the *uterus bicornis* the failure of fusion affects the parts of the ducts which go to form the upper part of the uterus.

Hermaphroditism.[1] True hermaphroditism means the co-existence of testis and ovary in the same individual. This condition is a very rare one. Only two authentic cases of bilateral hermaphroditism (ovary and testis present on each side) have been recorded. Unilateral hermaphroditism (ovary and testis present on one side, the other side having one form of the gland) has been mentioned in one case. Lateral hermaphroditism (ovary on one side and testicle on the other) has been more frequently met with.

Pseudo-hermaphroditism is a condition of the external organs so that they simulate those of the opposite sex. Congenital hypertrophy of the clitoris and complete hypospadias are examples.

Misplacement of the Testis. One or both testes may be found out of their proper place. If the organ is arrested at some

[1] See Hart and Barbour, *Manual of Gynæcology*, 1886, p. 513.

point between its origin at the side of the lumbar spine in the abdomen and its normal destination, the scrotum, the condition is termed *Retention* (abdominal, inguinal, etc.). If the organ is found in some abnormal position away from the normal line of descent the condition is termed *ectopia* (Gr. *ek* = out, *topos* = place). Various causes are given for retention and ectopia of the testis, and doubtless the condition may be brought about in several different ways. Thus arrest of growth of the spermatic blood-vessels or the peritoneum covering the testis would prevent its proper descent. The part attributed to the gubernaculum in drawing down the testis is problematical. In a deformed child, which died shortly after birth, I found the thighs so doubled on the abdomen that the left inguinal canal was practically closed; the left testis was retained within the abdomen and suspended by a long mesorchium. In the same subject the left thigh had been so tightly pressed against the upper part of the right that the right testis after emerging from the external ring had passed outwards in the superficial fascia of the abdomen and lay close to the right anterior superior spine. The most important form of retention is that in which the gland remains within the inguinal canal (*retentio inguinalis*). Here it is often associated with a congenital hernia. On examining such testes in young adults with the microscope they are found not to contain spermatozoa, and hence to be functionally sterile. A retained testis may become inflamed from forcible action of the abdominal muscles, or the cord may undergo *torsion or axial rotation*, and the testis then swells from congestion, œdema, and finally undergoes gangrene. The symptoms of this condition may closely resemble those of strangulated hernia in the inguinal canal. The more important forms of *ectopia testis* are constituted (*a*) by the gland passing through the crural ring into the femoral canal, or (*b*) after passing through the inguinal canal, instead of entering the scrotum, going into the perineum.

PART II.

DISEASES OF SPECIAL TISSUES AND ORGANS.

CHAPTER VIII.

DISEASES OF BONE.

Some of the peculiarities of pathological processes in bone depend upon its hardness. Bone may be regarded as a vascular fibrous tissue, having a calcified matrix in which the bone cells and fibres are embedded. The vascular channels contain arteries, veins, and lymphatics supported by connective tissue. The effect of this peculiarity of structure may be readily imagined when compact bony tissue becomes the seat of septic inflammation. The tissue cannot swell to accommodate the effused cells and fluid which serve to dilute the solution of septic matter in inflamed areas of more lax tissues. The *tension* thus becomes greater, and the inflammatory process is less likely to subside, and necrosis of tissue is likely to be more extensive than in soft parts. When necrosis has occurred, the dead tissue being hard and almost mineral in character, the process of separation of the dead part of the bone from the neighbouring living part is more difficult than when necrosed parts are composed of soft tissue. In the case of the long bones it is not only the histological structure, but also the mode of growth that influences pathological processes. The most active parts of a long bone during the period of growth are the deeper layer of the periosteum and the extremity of the shaft where it abuts

upon the epiphyseal cartilage, the "juxta-epiphyseal region" of Ollier. In both these situations new blood capillaries and new bone are being formed. We are therefore not surprised to find that these are the two parts of a long bone in which the structural alterations are most marked in rickets, and that these are the more common situations for pyogenic organisms to lodge and set up acute inflammation in bone. The vascular marrow of bone is also of great importance from a pathological as from a physiological point of view. Strictly speaking, the contents of all the Haversian canals, as well as the soft tissue in the central cavity and spongy parts of bones, come under this designation. The more active or red marrow is one of the sources of red-blood cells which are formed from its connective-tissue cells. The marrow of the older (central) parts of bones, and in later life, the whole of the marrow of the spongy tissue becomes the seat of fat formation by the deposit of oil globules within the connective-tissue cells. Absorption as well as growth, is active in bone. When the conditions are suitable, the cells (osteoblasts) which line the marrow spaces fuse together[1] forming multinuclear cells (*osteoclasts*), which digest the bone against which they lie, and form spaces with sinuous outlines. In this way bone becomes absorbed. The process occurs in chronic inflammatory processes of moderate intensity, which thus constitute rarefying ostitis, as it is termed. In old age, when all tissues of the body tend to atrophy, the absorption of bone often results in its becoming thin and porous. This condition is termed *senile osteoporosis.*

In the case of the jaw and some other bones certain

[1] This is Kölliker's view. It is also possible that single osteoblasts perform the same function (compare Fig. 53); just as in inflammation connective-tissue cells, whether they remain single or fuse together to form giant cells, may digest the pre-formed fibrous tissue, so bone cells, whether they form osteoclasts or remain single, may digest pre-formed bone. When a portion of bone has perished it is thus separated from the living bone, the part of the latter adjoining the dead bone being absorbed. Some of the dead bone itself, unless it is charged with septic matter, is also removed, just as are ligatures of catgut, etc., and in time even ivory pegs, though the latter have sometimes been found encapsuled in fibrous tissue long after insertion.

anatomical relations render them liable to affections which do not originate in the bones themselves. Thus the teeth, from their diseases and errors of development, lead to many abnormal conditions which demand separate consideration.

Acute Suppuration in Bones. In considering the repair of fractures the liability of the fragments to be forced through the skin (compound fracture), and so exposed to contamination with pyogenic organisms was mentioned. Besides delaying union, such infection may be the starting-point of septic inflammation. Before antiseptic surgery was established it was common for septic inflammation of bone to follow amputations. Both in a compound fracture and after an unclean amputation all parts of the bone are exposed to infection. If in an amputation a long bone, such as the femur, has been divided, say, in the middle of the shaft, and pyococci have gained access to the wound, suppuration may occur (*a*) in the medullary cavity (*osteomyelitis*), (*b*) in and beneath the periosteum, and (*c*) in the compact tissue which is attacked from below by the inflammation due to the original infection, and from within and without by inflammation spreading from the medulla and the periosteum respectively—the result being a more or less extensive necrosis, accompanied by the formation of new bone from the periosteum by "plastic periostitis." The patient is exposed to the risk of pyæmia, and suffers from pain in the limb during the months which are occupied in the separation of the *sequestrum*, as dead bone when shut in by living bone is termed.

Spontaneous Acute Suppuration in Bone. The frequency with which slight suppurative affections of the skin, throat, and other parts occur renders it highly probable that micrococci frequently find their way into the blood and to various parts of the body including the bones. That this occurrence usually produces no marked disturbance of the general health is owing to the fact that in persons of average vitality the organisms are destroyed by the white blood cells, and the endothelial and connective-tissue cells. In less resistant individuals they may set up suppuration. Suppurative processes are more readily set up in children than in adults, and in accordance with this is the fact that acute infective inflammation of

bones is more common in children than in adults, though by no means unknown in the latter. There is another important reason for the frequent occurrence of suppurative disease of bone in children. It is in early life that the common infective fevers or exanthemata usually occur. Whatever the unknown agents of these fevers may be they are probably living organisms. The inflammation of the throat which occurs in scarlet fever, measles, small-pox, etc., the vesicles, etc., of syphilis and chicken-pox, and the ulcers of typhoid fever may be given as examples of breaches of the surface observed in fevers which account for the entry of pyococci into lymphatics and so into the blood. The effect of the poisons of the specific fevers is to weaken the resistance of the cells of the tissues, and so to render it more likely that pyococci once in the blood should escape destruction and start suppurative processes. So common is acute suppuration in bones after specific fevers that such cases have been grouped together as examples of "exanthematous necrosis." Frequently only a single bone is affected, but too often the condition is but part of a general pyogenic infection—a pyæmia—which affects the body generally. Staphylococci or streptococci are commonly the offending organisms.

The Parts of the Bones Affected. As has been stated above, the most vulnerable parts of a growing bone are the extremities of the diaphysis and the deepest part of the periosteum. If suppuration starts in the former place it is usually termed acute epiphysitis, a term which is erroneous.[1] The process begins close to the epiphysis, and, if the latter is ossified, soon extends to it. The central part of the medulla or even the compact tissue may be the original seat of infection. In reality it is often impossible to learn from clinical evidence exactly where the inflammation began, so rapidly may inflammation beginning in one part of a bone spread to another. In the flat bones especially is this the case.

In Fig. 47 are represented the chief anatomical characters of a case of infective so-called epiphysitis.

A brief consideration of the illustration just given is sufficient to show that in acute epiphysitis prompt measures are called

[1] Ollier's term is "juxta-epiphysitis."

for if the infiltration of the whole shaft of a bone with pus, and consequent necrosis and possibly general pyæmic infection, are to be averted. With the trephine or gouge the medullary cavity of the bone must be freely opened.

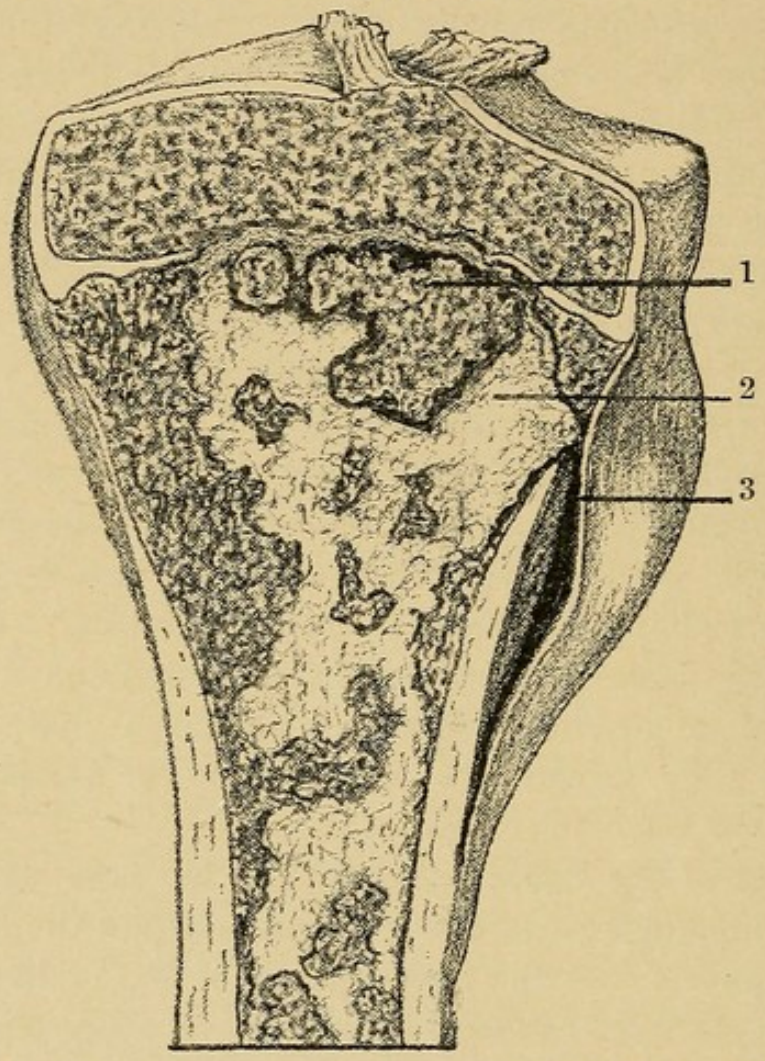


FIG. 47.—Vertical section through the upper end of a tibia. The central part of the epiphyseal cartilage has been destroyed by suppuration. 1, A sequestrum surrounded by (2) pus which infiltrates the spongy bone of the shaft and which has destroyed the middle part of the epiphyseal cartilage, and at (3) has extended to the periosteum and formed a subperiosteal abscess.

The most important Complication. When the epiphyseal line is within the cavity of a joint as at the upper end of the femur, the pus rapidly finds its way into the joint cavity and suppurative arthritis supervenes on epiphysitis. In such cases free opening of the joint and removal of the upper end of the femur can alone save the patient from death.

Joints are also frequently infected by pus escaping into them from the epiphysis, through a perforation in the articular cartilage.

In some cases the extremities of the diaphysis are perfectly sound, and it is evident that the process either began in the central part of the medulla and extended to the periosteum or *vice versâ*. In the case from which Fig. 48 was taken it is probable that the process began in the medulla.

The bone lesion is frequently one of many pyæmic lesions, and the case may be hopeless from the outset.

The Results of Septic Inflammation. The occurrence of necrosis has already been sketched. This event is so commonly observed in infective suppuration of bone that the older surgeons gave the disease the name of *acute necrosis*. Since necrosis is here an effect of septic infection, the terms infective ostitis or, when the distinction can be made, infective osteomyelitis or periostitis had better be adhered to. If the suppuration is limited to the deep layer of the periosteum, the process may end without necrosis, should the pus be promptly evacuated. If the infective inflammation spreads to the contiguous superficial part of the compact tissue and extends no farther, a superficial necrosis follows and the dead bone separates as a thin shell (see Fig. 49). This is *exfoliation*. When a portion of the shaft of a bone has undergone necrosis owing to acute osteomyelitis, new bone is formed from the

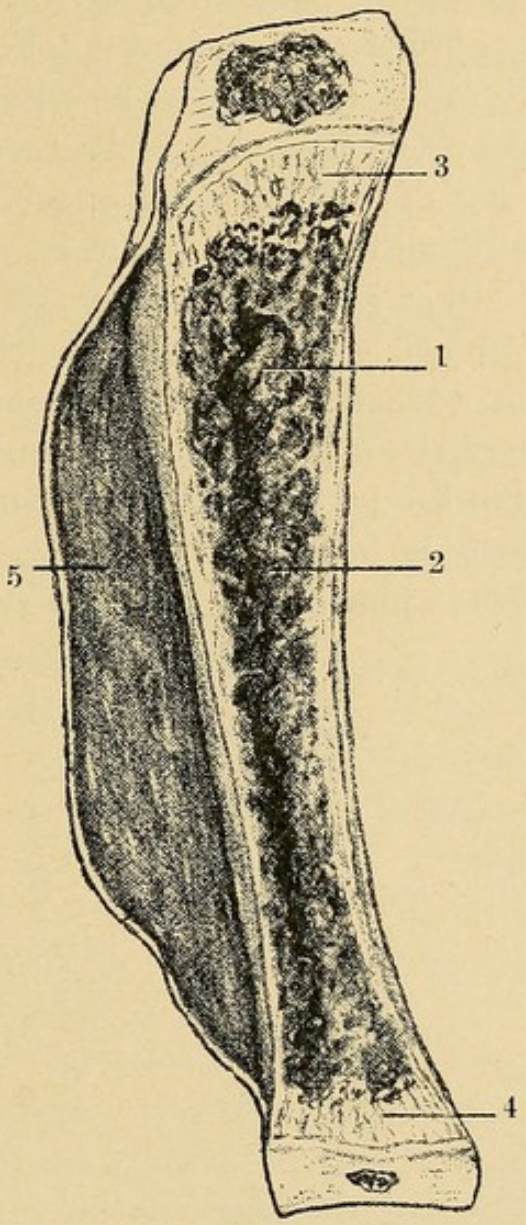


FIG. 48.—Vertical section through the left tibia of a child aged 10 months. 1, Upper end of; 2, middle of the medullary cavity which was full of pus; 3 and 4, extremities of the diaphysis which are normal; 5, cavity of a subperiosteal abscess. Note that the periosteum still remains attached at the posterior aspect of the bone. The patient died of pyæmic abscesses of the lungs. (229 B.)

periosteum, as described above under septic ostitis from traumatism. This results in the formation of a sheath of new bone (*involucrum*) perforated by apertures (*cloacæ* = drains), where pus has escaped, as shown in Fig. 50. The operation of chiselling away sufficient of the sheath to allow of the removal of a sequestrum either entire or after subdivision is termed *sequestrotomy*.

When the whole of the shaft of a bone rapidly undergoes necrosis from septic inflammation the periosteum is separated from the bone by pus save where strong muscles are attached and

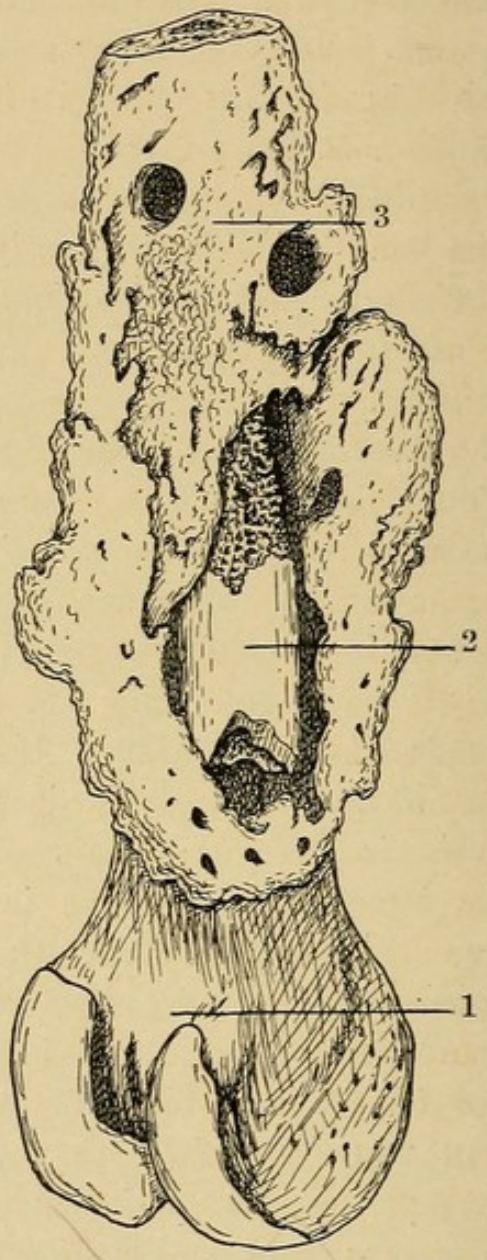


FIG. 50.—The lower end of the femur of an adult (1). 2, Sequestrum, the upper end of which is tapering and presents the "worm-eaten" appearance of dead bone after natural separation from living bone; the lower end includes the whole thickness of the original bone, and it has been chiselled away in an attempt to remove the sequestrum (probably before the introduction of anæsthetics). 3, Sheath of new bone formed from the periosteum, with two cloacæ. (226)

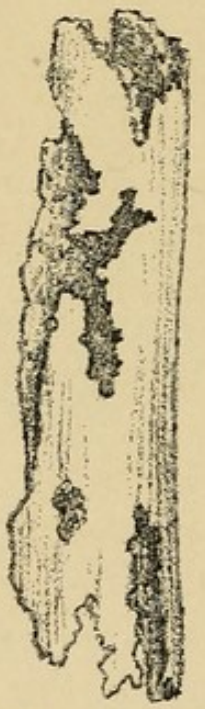


FIG. 49.—A thin superficial portion of the shaft of a femur exfoliated as the result of suppurative periostitis.

ligamentous fibres enter the bone as "perforating fibres." In such circumstances it may be necessary for the safety of the patient to remove the shaft of the bone at once before any

new bone has had time to form from the periosteum. This operation is termed "sub-periosteal resection." After both sequestrotomy and sub-periosteal resection the new bone formed from the periosteum has been found to be sufficiently strong to perform the functions of the original bone, and in some cases the new shaft has even been stronger than the original.

Acute septic inflammation of bone is by no means limited to the long bones. It is not infrequent in flat bones, such as the ileum and scapula, and it has been observed in the vertebræ. See also "Diseases of the Jaws."

Tuberculosis of Bone. Bone is frequently the seat of tubercular infection which usually occurs in the cancellous tissue. The histological changes are the same as those of tuberculosis in other tissues with the addition of the effect of the tubercular granulation tissue upon the special tissue. If the process is of moderate severity the bone is absorbed by osteoclasts as the tubercular infection extends peripherally on all sides from the original point of infection. Thus the bone is rarefied, and the process is termed rarefying ostitis or *caries* (L. = decay). The older part of the tubercular focus either breaks down into pus forming a chronic abscess or undergoes caseation. If the necrotic material is insignificant in amount, it may be removed by phagocytosis as fast as it is formed; in this case the process has been termed dry caries (*caries sicca*).

The deposit of tubercle may be either spread over a wide area of the medulla (*diffuse infiltration*), or it may be limited to a small area (*circumscribed infiltration*). Tuberculosis in the interior of a bone, apart from the destruction effected in the tissues occupied by the tubercles themselves, causes the formation of more or less vascular granulation tissue, which has the result of rarefying the bone. While this "rarefying ostitis" is in progress in the interior of the bone a new formation of bone may occur from the periosteum. In this way a swelling of the bone will arise, and may simulate the effects of a central sarcoma, etc. If the bacilli increase too rapidly for the bone to be absorbed *pari passu* with the extension of the tubercular tissue, then that part of the bone which is contained within the hollow sphere of tubercular tissue undergoes necrosis from thrombosis of

its vessels, the walls of which are attacked by the bacilli. If the disease, either from improved health on the part of the person affected or from diminished activity on the part of the bacilli, should become less rapid, then the portion of bone that has perished in the more rapid period of the disease may become separated by rarefaction taking place outside it when the disease has slowed down. It is then termed a tubercular sequestrum (see Figs. 51 and 52).

The condition shown in Fig. 51 has been termed "caries necrotica." The term is not a very good one since most tubercular processes are associated with more or less necrosis, but it serves to recall the fact that formerly the terms caries and necrosis were used in contrast, the former referring to the slow absorption of bone without the formation of sequestra, and the latter designating any process which led to the formation of sequestra of dimensions not readily overlooked by the naked eye. The importance of recognizing the presence of a sequestrum in tubercle of bone may be as great as it is in acute suppurative conditions: this is shown in Fig. 52, which also shows condensation or *sclerosis* of bone (Gr. *skleros* = hard). Sclerosis may result from any form of chronic ostitis that is too slight to cause rarefaction. In sclerosed bone the Haversian canals and other marrow-spaces are narrowed.

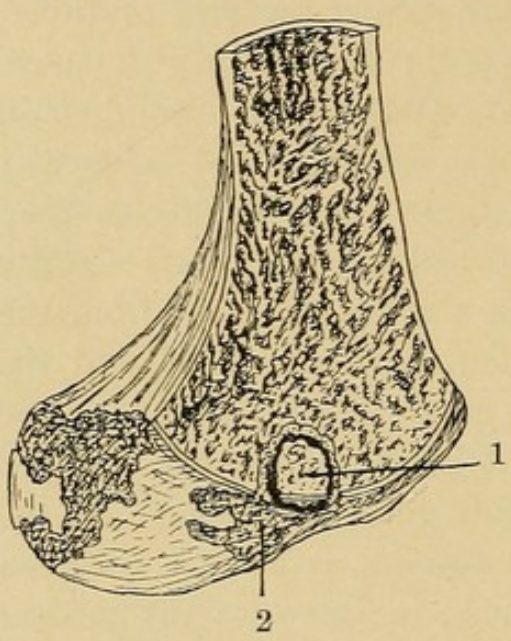


FIG. 51.—Vertical antero-posterior section through the lower end of a femur. 1, A tubercular sequestrum still covered by part of the articular cartilage and lying in a cavity lined by tubercular granulation tissue; 2, articular surface of the femur, from which much of the cartilage has been removed by granulation tissue which has invaded the joint cavity from the bone.

The extremity of the diaphysis has been indicated as a common situation for infection by ordinary pyogenic organisms, and it is also for tubercle bacilli. In the hip-joint and in other situations where the epiphyseal cartilage is situated within a joint-cavity this often leads to the infection of the synovial

membrane and the rest of the joint surfaces. Such cases will be referred to again in connection with the diseases of joints.

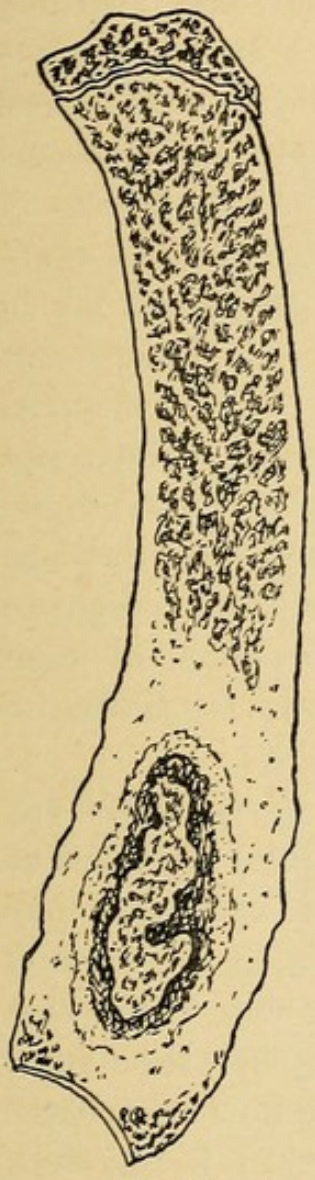


FIG. 52.

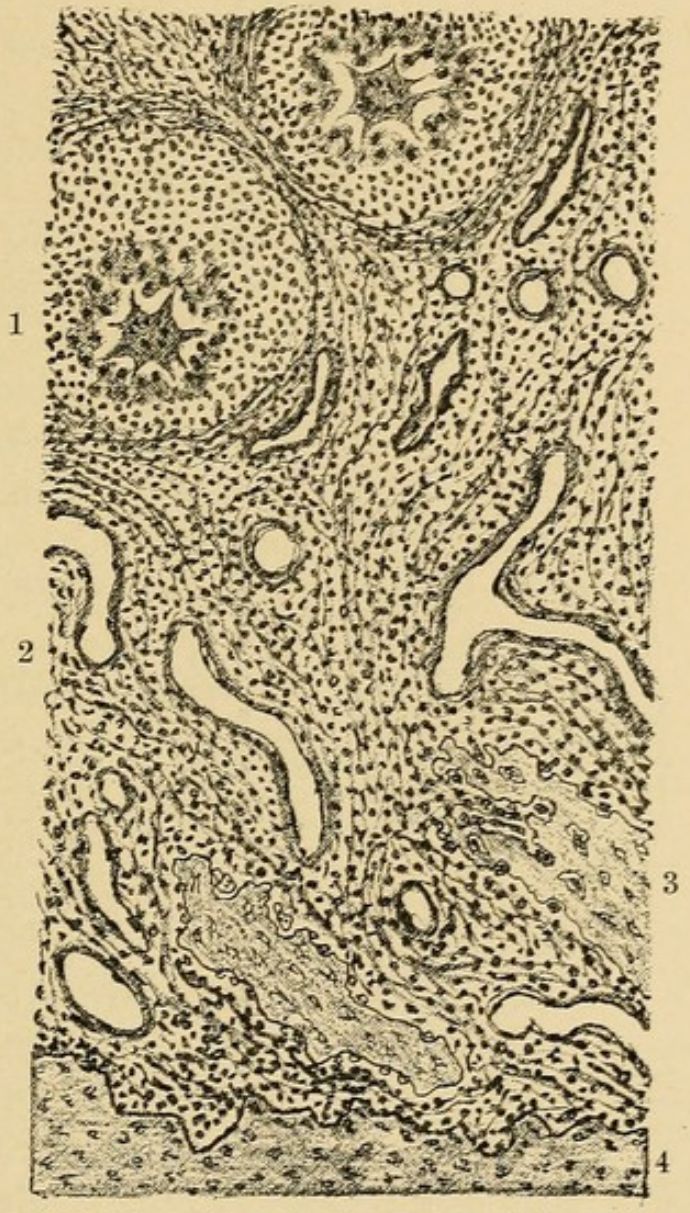


FIG. 53.

FIG. 52.—Section through a tibia showing a sequestrum surrounded by sclerosed bone. The cavity in which the sequestrum lies is lined by granulation tissue. It communicated by a sinus with the exterior. The shaft of the tibia is thickened in the lower half and elongated so that it is curved forwards and had forced the foot into the position of equino-varus. ¼ nat.

FIG. 53.—1, One of two tubercles; 2, surrounding granulation tissue; 3, one of two spicules of bone undergoing resorption. In this spicule the osteoblasts appear to be removing the bone; in the other below and to the left of it erosion by osteoclasts is in progress. 4, Part of the epiphyseal cartilage becoming absorbed by granulation tissue. × 150.

Cuboidal bones, such as the bodies of vertebræ, the carpal and tarsal bones, are all common situations for tuberculosis. Tuberculosis of the spine will be dealt with separately in the

section on "Diseases of the Spine." Fig 53 shows the histological structure of tubercular osteomyelitis.

Syphilis of Bones. Syphilis affects the periosteal aspect of bone more commonly than the spongy tissue, thus affording a contrast with tubercle. It is in the late period of the acquired disease that serious syphilitic bone lesions are apt to occur, though transient pains may be present in the secondary period. Bone-lesions are common in the early months of life in congenital syphilis, and late lesions also occur. In the earlier syphilitic inflammations of bone in congenital syphilis, not only the periosteum, but also the diaphysis in the region of the epiphyseal cartilage and this cartilage itself are prone to suffer. An admirable example of both periostitis and epiphysitis is shown in Fig. 54.

FIG. 54.—Vertical antero-posterior section through the humerus of an infant aged eight weeks. The lower half of the shaft is thickened by (1) a deposit of new bone (ossified node) from the periosteum. The proliferating zone of the epiphyseal cartilage has been destroyed by (2) granulation tissue which springs from the lower extremity of the diaphysis and extends through the synovial membrane into the elbow joint. (290 A.)

The chief symptoms of syphilitic epiphysitis (or osteo-chondritis) are helplessness and wasting of the limb, and they have gained for the affection the name of "pseudo-paralysis." The child from which the specimen shown in Fig. 54 was obtained by H. S. Collier, died from "marasmus" at the age of eight weeks; it had been treated with mercury, and the condition of the bones had somewhat improved. The histological appearances of the same humerus are shown in Fig. 55. The slighter degrees of syphilitic osteo-chondritis may occasion little pain or interference with movement, but are evidenced by the formation of ring-like thickenings at the epiphyseal regions of the femur, tibia, humerus, clavicles, ribs, and other long bones. The swellings are usually symmetrical, but are not so widespread as those of rickets.

In this case the periostitis led to bone hyperplasia, while the osteo-chondritis resulted in the destruction of bone and cartilage by granulation tissue, and extension of inflammation into

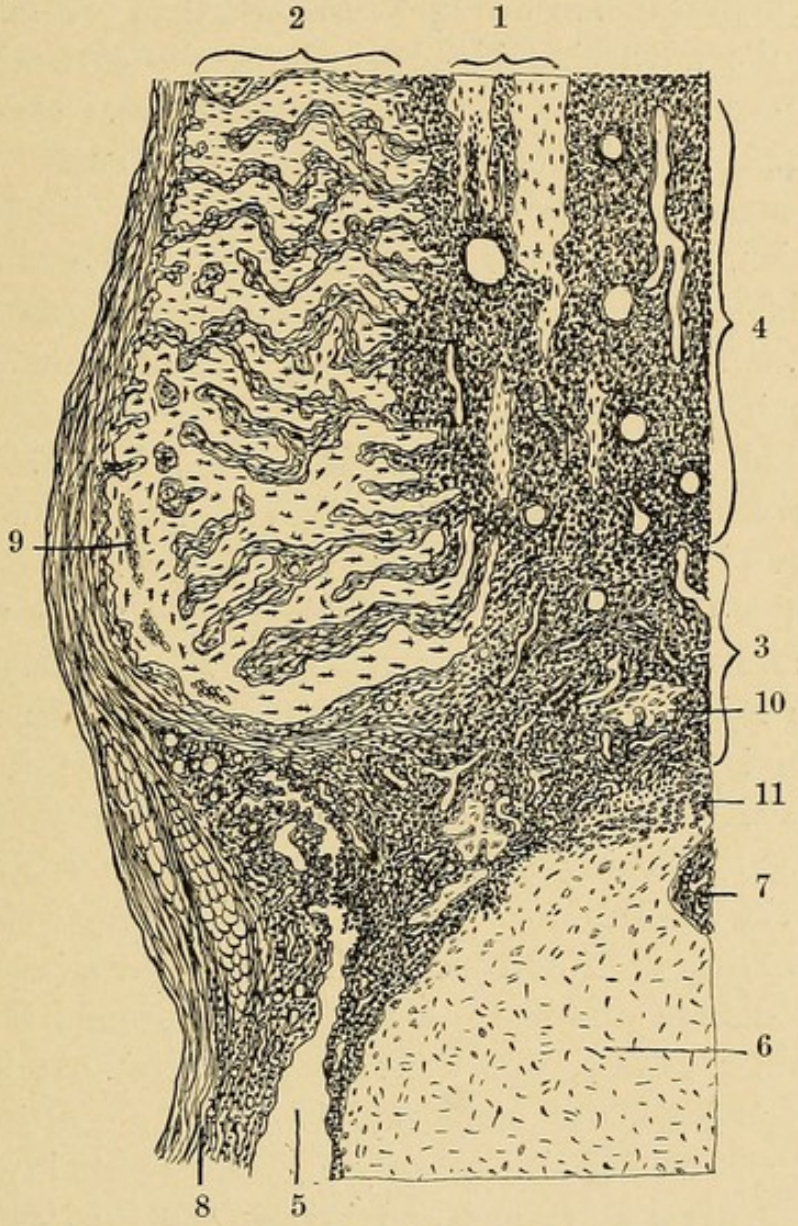


FIG. 55.—A part of a section of the bone shown in Fig. 53. 1, Remains of the old compact tissue of the shaft; 2, newly formed periosteal bone constituting the thickening or node on the shaft; 3, granulation tissue replacing the bone marrow and the proliferating layer of cartilage; 4, marrow with dilated blood-vessels and inflammatory infiltration; 5, cavity of the elbow joint; 6, cartilage of the epiphysis; 7, part of a newly formed marrow space in the cartilage; '8,' is placed between the fibrous capsule of the joint and the synovial membrane which is converted into granulation tissue; 9, one of three islands of cartilage newly formed from the periosteum; 10, island of epiphyseal cartilage that has escaped destruction by the granulation tissue; 11, cells of the cartilaginous epiphysis proliferating towards the shaft. × 30 diams.

the joint. Thus the changes are, like other syphilitic lesions, of varying degree. The slighter grades of syphilitic osteo-chondritis are very commonly met with in the bodies of syphilitic

infants who are either born dead or die soon after birth. The lower end of the femur is a frequent site for the disease. A section in a case of slight severity will reveal an irregularity at the epiphyseal line owing to irregular and excessive development of the advancing blood-vessels at the extremities of the shaft into the epiphyseal cartilage. The latter also shows an excessive proliferation of its cells towards the shaft. Both these changes occur in rickets, as will be more fully discussed in the next section. An increase and irregularity in the calcification of the proliferating layer of the epiphyseal cartilages is also observed. The most severe cases present foci of gummatous softening in the medulla.

The skull bones are apt to be affected by syphilis. If of moderate degree of intensity the disease causes diffuse nodes on the bones. In congenital syphilis this occurs most commonly in the neighbourhood of the anterior fontanelle where the sagittal, frontal, and coronal sutures make four right-angles, and when syphilitic nodes form in this situation the appearance of a hot-cross bun is roughly simulated. The condition is known as "Parrot's Nodes." If the process is more rapid in infants instead of thickening absorption of bone occurs; areas of the vault of the skull may become so thin that they can be pressed in by the fingers. This condition is termed *craniotabes*. In the acquired disease diffuse thickening of the frontal bones occurs on the inner surface, sometimes causing persistent headache.

Periosteal lesions of more rapid formation constitute at first fibrous nodes which either ossify or break down into gummata. Such gummata may extend and involve the skin, causing ulcers in the base of which bone is exposed to the ordinary pyogenic and putrefactive organisms. Thus a double pathological process is initiated, and suppuration supervenes on syphilitic ostitis. This double infection is of the highest importance in the case of the bones of the skull, since suppuration is liable to spread to the meninges and the brain. After syphilitic necrosis the process of separation of the dead bone is extremely tardy, years sometimes being required before the dead bone is completely free. This is partly accounted for by the progressive spreading of the disease to new areas before there is time for separation to occur.

Or again, whilst in some part of the area of bone which is the seat of the disease the process is sufficiently active to determine absorption by osteoclasts (*rarefying ostitis*), at another part there is less pathological activity, so that only osteoplastic changes occur. In this way an area of bone may be separated at one part, and at another remain attached by an isthmus of living bone which serves to transmit blood to, and to keep alive more or less of the partially separated segment, which is then termed a "living sequestrum." Thus syphilis, like tubercle, produces in bone anatomical changes which vary according to the intensity of the reaction of the tissues to the virus of the disease. Nowhere is this shown better than in the skull-bones.

Occasionally nearly the whole skull-cap is removed by what may be termed a dry syphilitic caries, and that without any implication of the skin. Any bone may become the seat of syphilitic inflammation. Destruction of the vomer and other bones of the nasal fossa leads to the sunken nose. Tubercular disease of bone frequently, as has been shown above, leads to joint disease; so occasionally does syphilis. An example of this has been given in Fig. 54. And now and again in adults gummata form in the bone and lead to syphilitic joint disease. This will be discussed more fully in treating of joints.

Rickets of Bone.

Before considering the changes produced in bone by rickets it is to be remembered that this disease affects the alimentary tract and the whole organism as well as the bones. The abdomen is distended, the liver and spleen enlarged, muscles are weak, and there is often marked nervous irritability. Before the bones become altered sufficiently for the most prominent sign of rickets—the enlargement about the epiphyseal regions—to be produced, there is a condition of hyperæmia or congestion which has been observed on post-mortem examination, and of which there is sometimes evidence during life in tenderness of bones. When the extremities of the bones have become enlarged the malady has existed for some time. To appreciate the kind of change that exists in rickets a normal bone may be com-

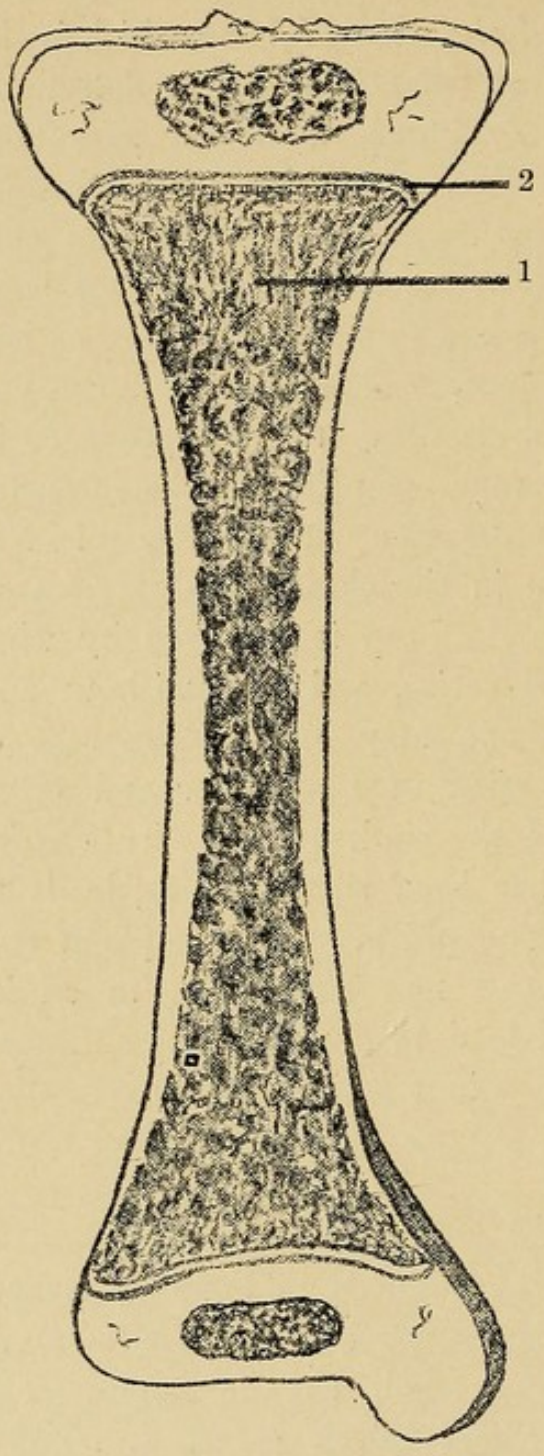


Fig. 56.

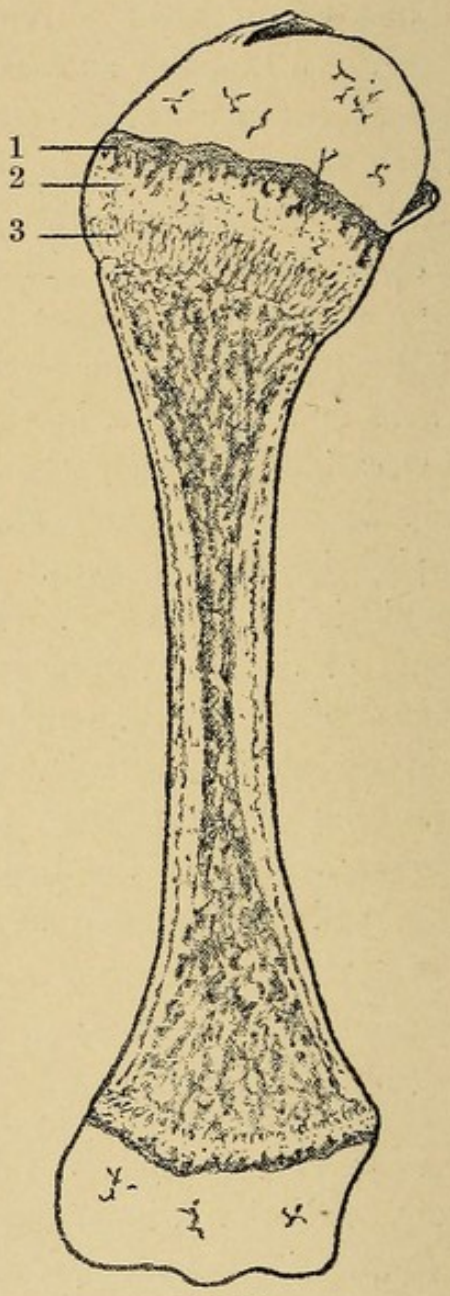


Fig. 57.

Fig. 56.—Vertical section of a normal tibia. 1, Diaphysis of the tibia; 2, translucent zone of proliferating cartilage—below it is a narrow white zone, that of calcification of cartilage matrix, and below this zone of calcification again is the uppermost part of the diaphyseal ossification. The three zones together make a layer not more than $\frac{1}{16}$ in. in depth.

Fig. 57.—Vertical section of a rickety humerus. 1, Zone of proliferating cartilage greatly increased in depth and prolonged below into processes, some of which have been cut off from the original layer to form islands of cartilage; 2, a layer corresponding to the layer of calcification of cartilage in normal bone and in rickets consisting of vascular marrow, uncalcified cartilage, and uncalcified bone or osteoid tissue; 3, the uppermost part of the diaphyseal ossification much increased in vertical depth. The surface of the shaft shows changes in the greater porosity and vascularity of the superficial part of the compact tissue. Macerated specimens of bones removed during the active period of rickets show a rough surface. When the disease has come to an end the smooth hard surface returns. Nat. size. (244 A.)

pared with one that presents marked rickety enlargement, as shown in Figs. 56 and 57 respectively. The enlargement at the ends of the long bones is fully accounted for by the increase in amount of tissue at the epiphyseal line, and in a severe case

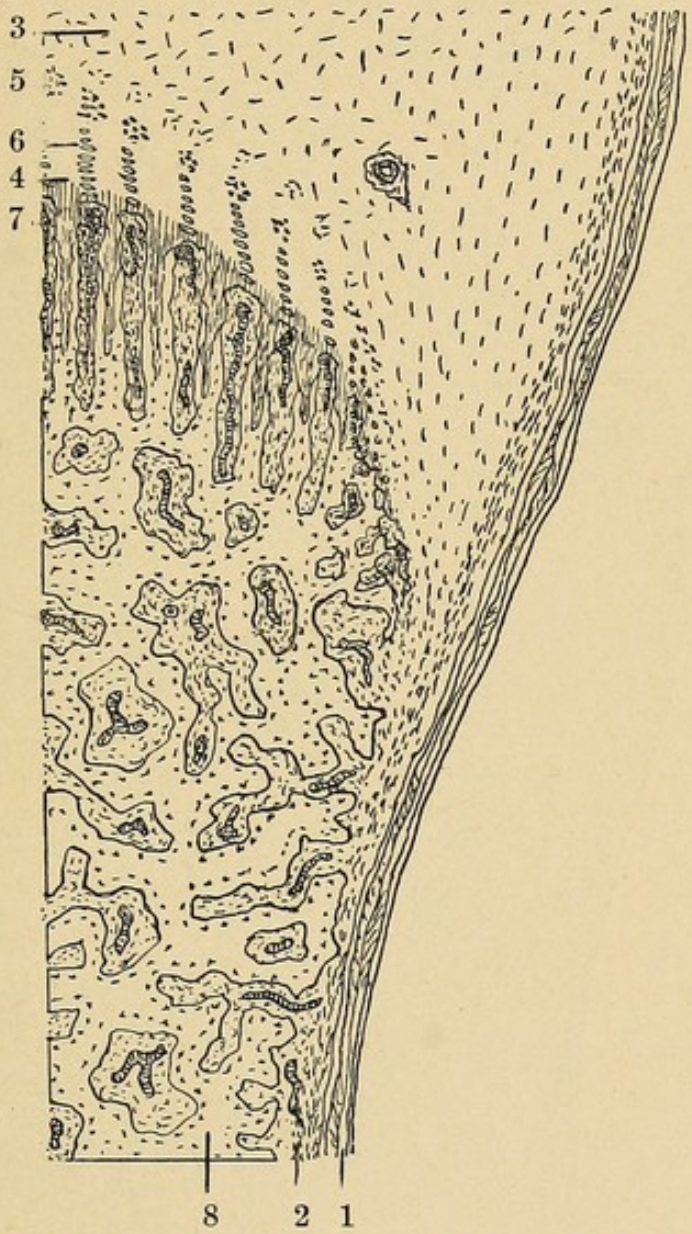


FIG. 58.—Vertical section of a normal humerus at the epiphyseal line. 1, Fibrous layer of periosteum; 2, cellular layer of the same; 3, cartilage of the epiphysis; 4, zone of calcification of the cartilage matrix; 5, cartilage cell-groups at the layer of commencing vertical proliferation; 6, scalariform groups of cartilage cells; 7, dilated capillary in one of the uppermost prolongations of the marrow-spaces of the diaphysis; 8, bone of the diaphysis. Undecalcified and unstained preparation. × 40.

like that from which the humerus (Fig. 57) was taken the greater part of the enlargement is found to consist of soft tissue; fine bony spicules can however be felt in the layer (3) by drawing the finger over the cut surface. On pressing upon

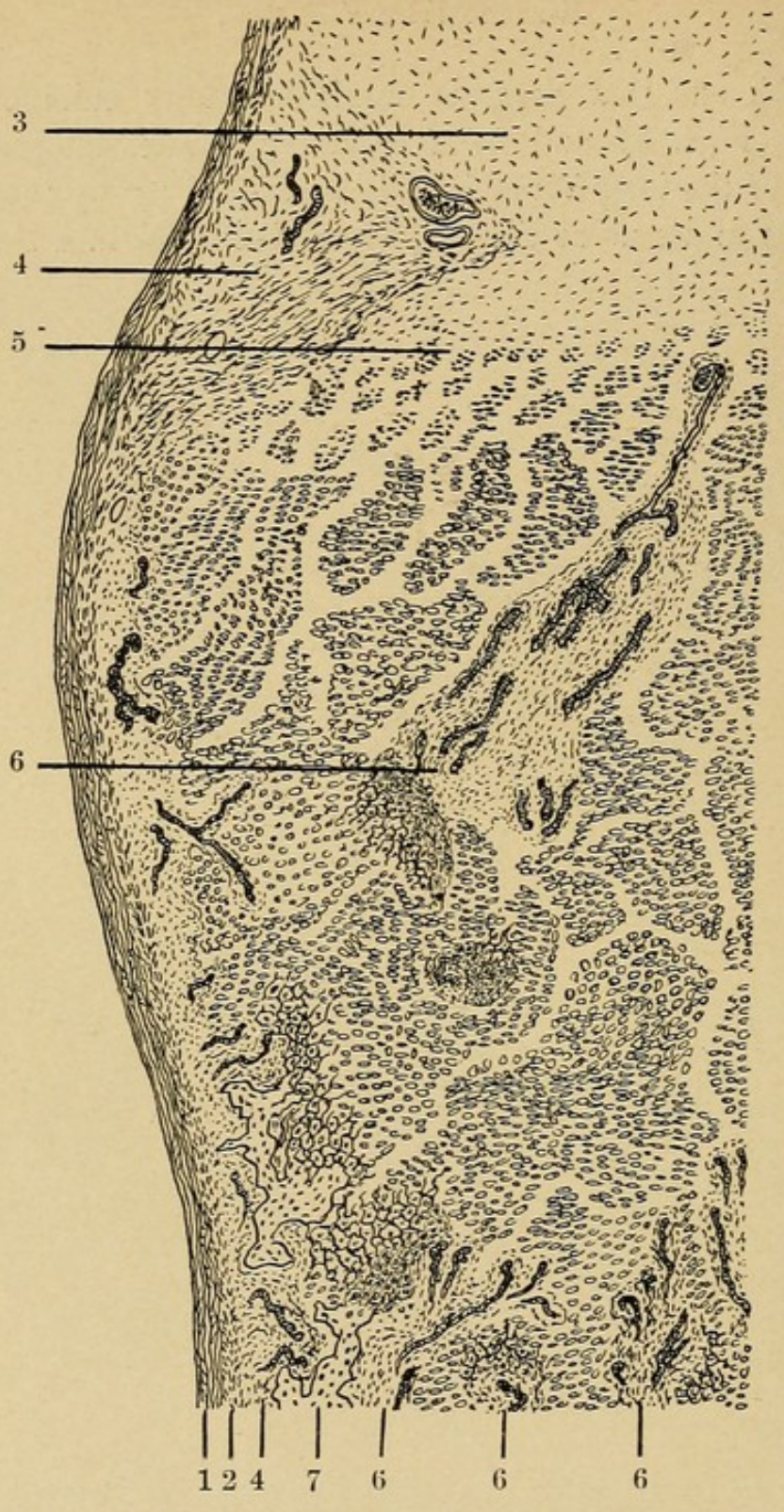


FIG. 59.—Vertical section of a rickety humerus at the upper epiphyseal line. 1, Fibrous layer of periosteum; 2, cellular layer of periosteum: 3, cartilage of epiphysis; 4, marrow formed from the cellular layer of periosteum, increased in amount and containing an increased number of blood-vessels which are dilated and full of blood (congestion); 5 (as in Fig 58), cartilage cell-groups at the layer of commencing vertical proliferation. From this layer to the lowest part of the figure irregular and large groups of cartilage cells are present. They represent the limited scalariform groups in the normal bone, and show that the whole of this Fig. below the line marked 5 represents the part of Fig. 58 between the lines 5 and 7. 6, Irregular marrow-spaces containing many tortuous vessels; 7, bone of the shaft corresponding to the uppermost part of zone 3 in Fig. 57. Above, 7 passes into osteoid tissue, and the latter again into calcified cartilage; the matrix of the cartilage is not represented save where it is calcified, and then it is shown by dark lines. Unstained section of an undecalcified bone. × 40.

the head of this humerus the soft tissue of the surface of the enlargement bulged outwards. Examined under the microscope the appearances of the normal and of the rickety bone are shown in Figs. 58 and 59. The histological changes which lead to the formation of swellings at the junction of the ribs with the rib-cartilages (Fig. 60) are precisely similar to those described above.

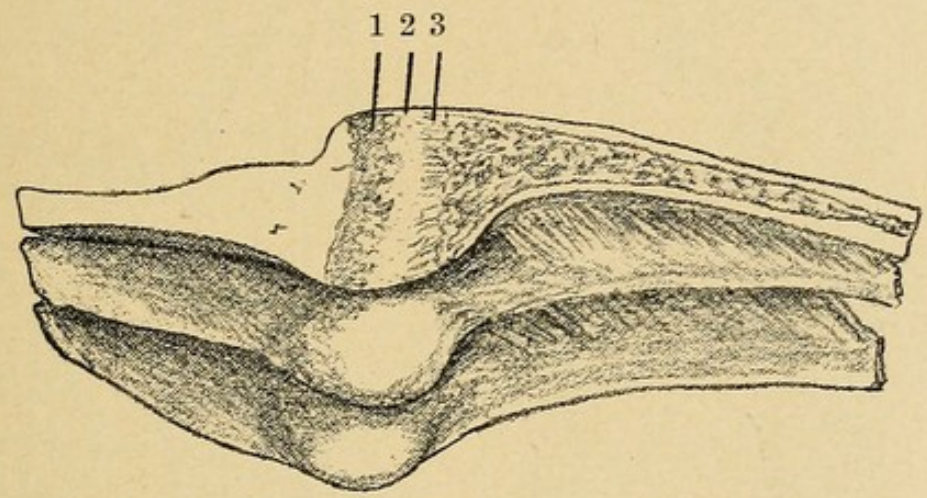


FIG. 60.—Portions of three ribs at the junction of shaft and costal cartilages. The "beading" is more marked on the pleural than on the superficial surface, owing to the sinking in at the junction of cartilage and bone. A section has been made through one of the ribs, and it shows zones 1, 2, 3 similar to those shown in the humerus in Fig. 57.

In severe rickets marked changes occur in the bone of periosteal formation. This is more porous than the normal bone in texture. In Fig. 57 the superficial layer of periosteal bone is somewhat more porous than is normal. Sometimes the change in the periosteal bone is more marked than at the epiphyseal line. When this is the case there is a great deficiency of lime in the periosteal bone. Much of it is osteoid tissue. Occasionally in rickets islands of hyaline cartilage are formed from the periosteum. In rickety animals the formation of subperiosteal osteoid tissue is often very marked (see Fig. 61).

In membrane bones, such as those of the skull, changes similar to those described above occur in periosteal bone. This is especially noticeable at the sutures of the skull, where distinct thickenings from the formation of porous bone and osteoid tissue may occur. When this formation of bone occurs over a considerable area of the skull, the latter has a resemblance to the "hot-cross bun" nodes of congenital syphilis.

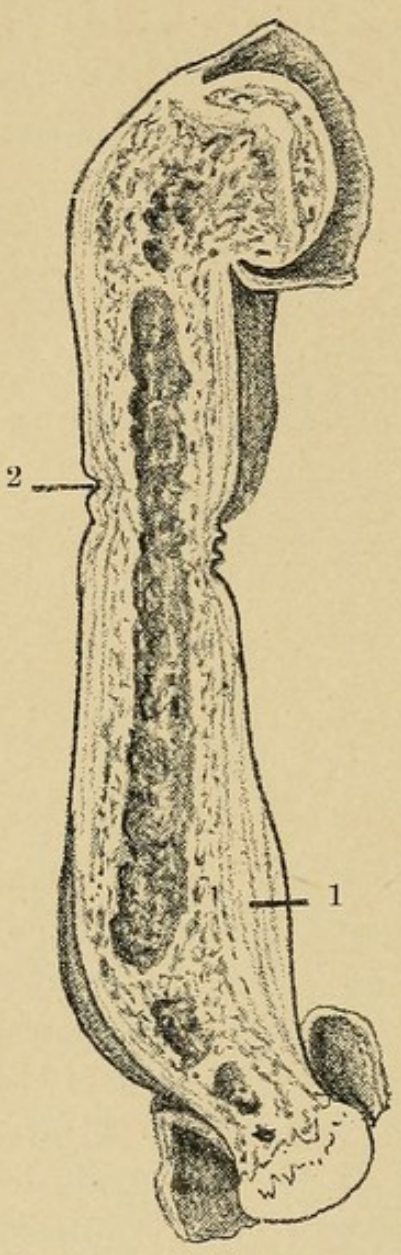


Fig. 61.

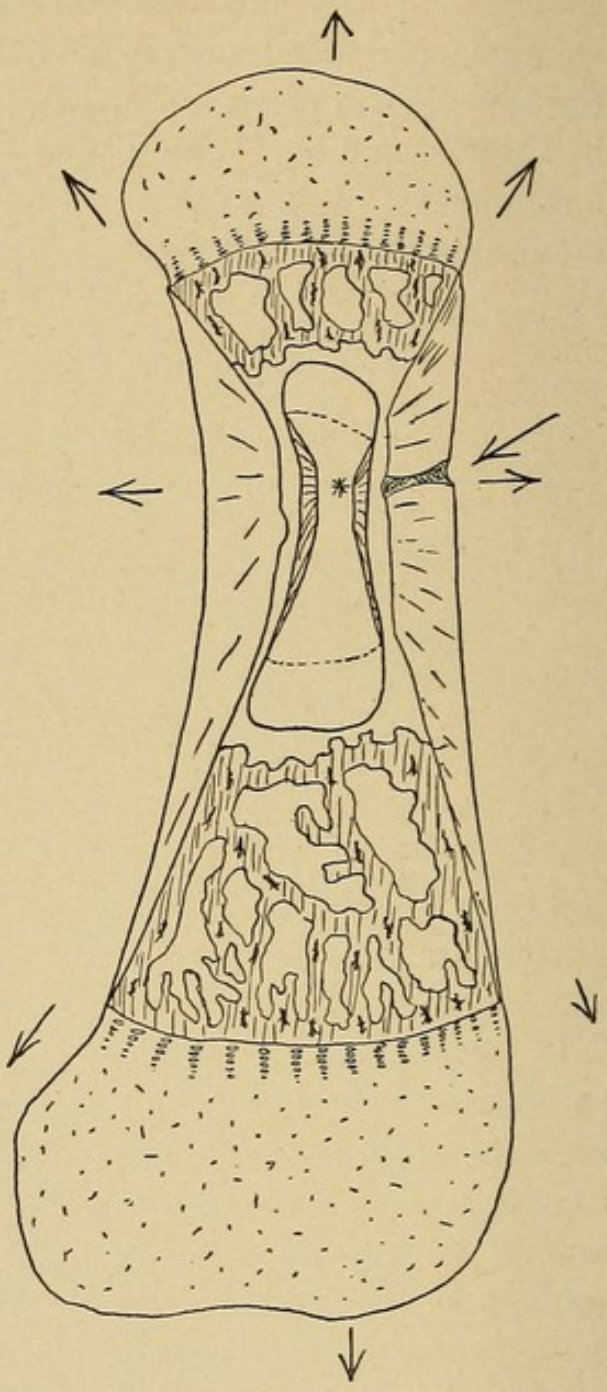


Fig. 62.

Fig. 61.—Section through the humerus of a rickety monkey. The periosteal osteoid tissue (1) is very abundant, and its yielding character is shown by the depressions (2) made where the bone had been surrounded by a loop of thread used to suspend the preparation. The head of the bone is bent on the shaft, and there is a sharp bend in the shaft. Nat. size.

Fig. 62.—Represents, on an enlarged scale, a metacarpal bone at birth. The part of the shaft of endochrondral formation is shaded. The bone formed from periosteum is white. The ingoing arrow points to the medullary canal which traverses the part of the shaft formed from periosteum, and is directed towards a star which marks the centre of growth in the bone. The outgoing arrows mark the directions of growth. The small figure within the larger one shows the same bone at an earlier period of life. It will be seen that any irregularities of outline in the smaller bone might readily be overcome by variations of the rate of growth in any particular direction. Modified from Kassowitz.

The practical bearings of the pathological anatomy of rickets are simple. Whilst the disease is in progress and the bones are soft, deformities are to be treated by splints. When the disease has ceased and the bones have become hard, osteotomy is necessary for the correction of deformity. Slight deformities have frequently been observed to disappear during the growth of a child. This at first puzzling phenomenon is readily understood when the nature of the growth of a long bone is remembered, as is shown in Fig. 62.

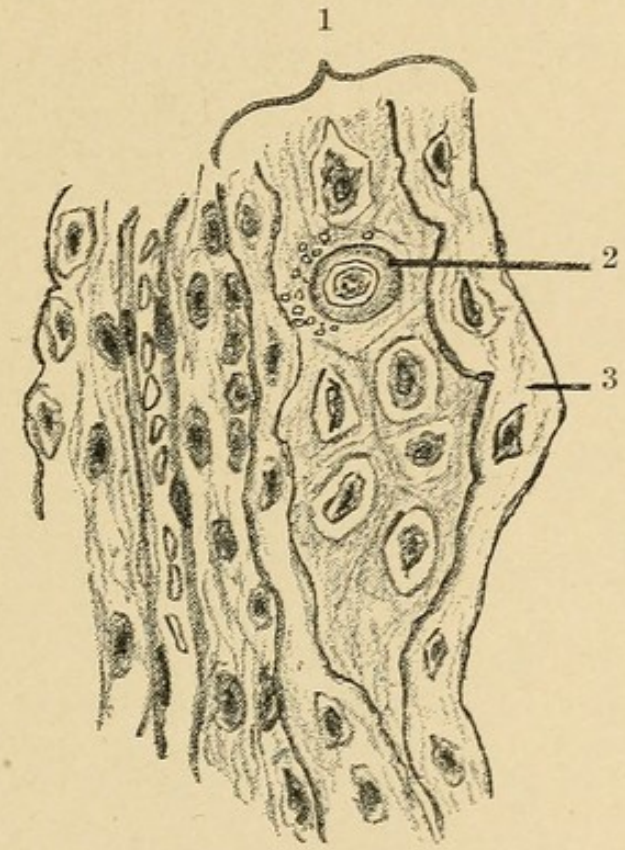


FIG. 63.—Represents on a larger scale the capillary marked 7 in Fig. 58, and at 1 the adjoining bone and remains of the cartilage. The latter contains seven cartilage cells, one of which (2) has formed around it a thick new capsule. This capsule becomes ossified by the deposit of granules of lime salts, similar to those in the cartilage matrix on its left, whilst the cartilage cell remains as a bone-corpuscle. Thus some of the cartilage becomes directly transformed into bone. This is termed metaplastic ossification. It has but little part in the formation of the shaft of the permanent bone. On the outer surface of the remains of the cartilage new bone (3) is deposited from the marrow. This has been termed neoplastic ossification. × 200.

In order to make clear the exact meaning of the term "osteoid tissue" it is necessary to refer to some points in the normal ossification. The processes which take place at the epiphyseal line in normal conditions may be considered first, as shown in Fig. 63, which shows a combination of metaplasia and neoplasia in the ossification of remains of the epiphyseal cartilage and in the formation of new bone on each side of them.

Metaplasia is the transformation of one kind of tissue into another by a modification of the cells and intercellular substance of the pre-existing tissue, *e.g.* the ossification of cartilage in Fig. 63. Neoplasia is the replacing of one tissue by another that invades and destroys the pre-existing tissue, as does the vascular marrow by sending processes of new blood capillaries and cells which

replace the greater part of the epiphyseal cartilage at its proliferating zone, in endochondral ossification. In rickets, instead of the regular scalariform groups of cartilage cells arranged in a definite zone, there are large collections of cartilage cells irregularly scattered over a wide area, and intersected by irregular marrow spaces. The formation of osteoid tissue from this hyperplastic cartilage is shown in Fig. 64.

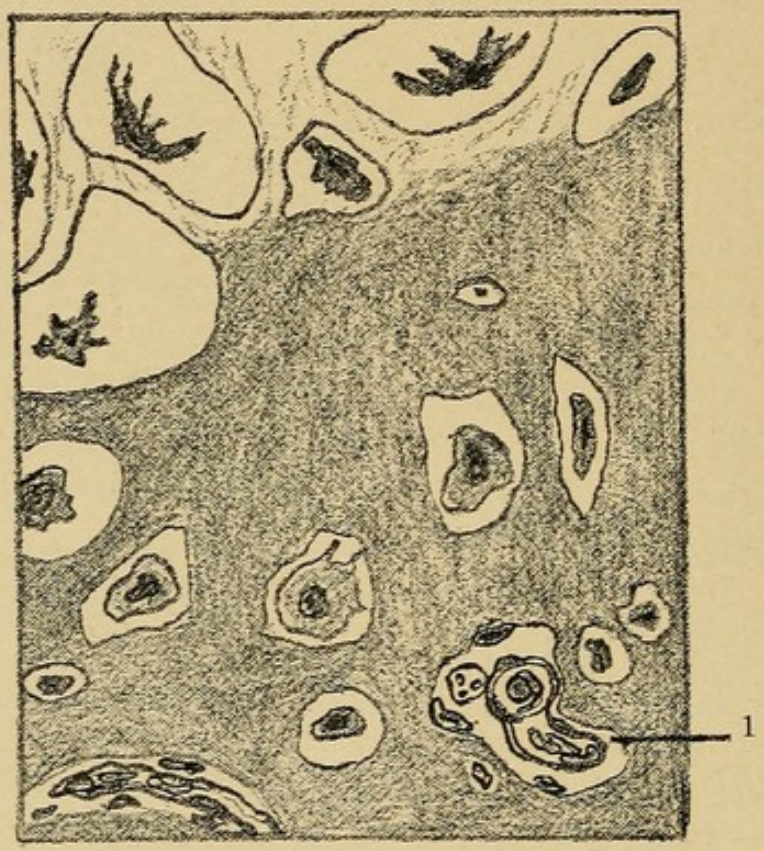


FIG. 64.—At the upper part of the section are distorted cartilage cells lying in large spaces of the matrix. Below these are cartilage cells surrounded by matrix altered in appearance from its taking the stain more deeply than the normal matrix of hyaline cartilage. This change would, in ordinary circumstances, be accompanied by the deposit of lime salts, and the cartilage cells it contains would be transformed to bone-corpuscles (metaplasia), as described in normal bone, and shown in Fig. 63. It is, in fact, unossified bone (osteoid tissue) of metaplastic origin. In the lower part of the figure at 1 is a space containing a blood capillary and some marrow cells. Some of the latter are embedded in the matrix that surrounds the space. This, in normal conditions, would be bone of neoplastic formation; here it is osteoid tissue. × 200.

Formation of osteoid tissue from the periosteum is not infrequently observed in rickets, as shown in Fig. 65.

When recovery from rickets takes place the osteoid tissue calcifies and becomes hard bone. Osteoid tissue then is simply uncalcified bone. It is found in the callus of fractures, in osteo-chondromata and other conditions as well as in rickets.

Excessive resorption of bone is observed in the active stages of rickets, rendering the bones unusually porous. When this rarefying process is pronounced the compact tissue may come to resemble the cancellous tissue. This also predisposes the bones to fracture.

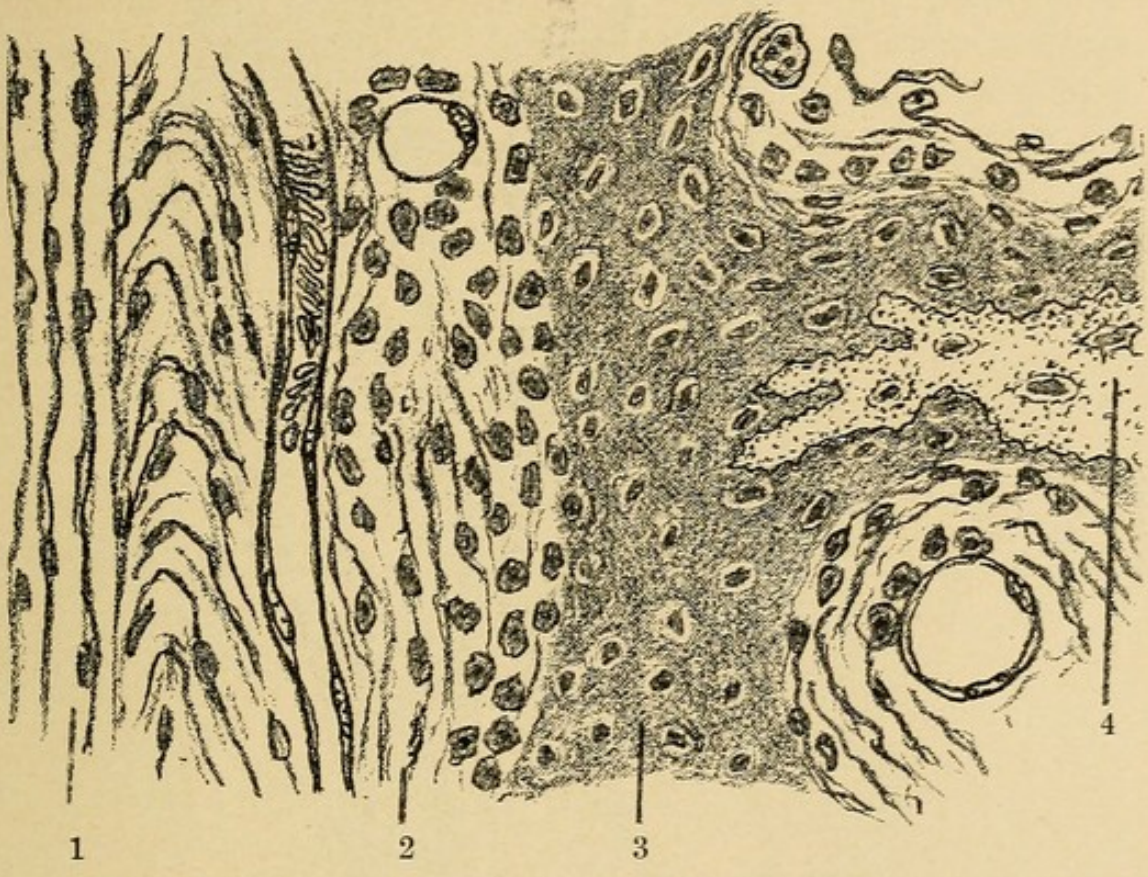


FIG. 65.—1, Fibrous layer of periosteum; 2, cellular layer of the same. 1 and 2 are, for convenience, represented as being much narrower than they are in reality. 3, Osteoid tissue formed from the periosteum; 4, bone formed from the periosteum. Figs. 63 and 64 are taken from sections of an undecalcified rickety bone, stained with picro-carmine. × 250.

The Gross Effects of Rickets are usually most marked in the bones of the lower extremity owing to their having to sustain the weight of the body when the child begins to walk. Some of the commoner curves are shown in Fig. 66.

Outward bending of the knee, genu-varum, is much less common than genu-valgum. In severe cases all the long bones may become curved. On making sections of such curved bones it is noticed that the shaft is thickest in the concavity of the bend (Fig. 67). This may be accounted for by remembering that the gradual bending of a bone may be regarded as the result of a series of minute green-stick fractures; and just as

the callus of a green-stick fracture is more marked in the receding angle (see Fig. 68), so in rickets, as the bone bends

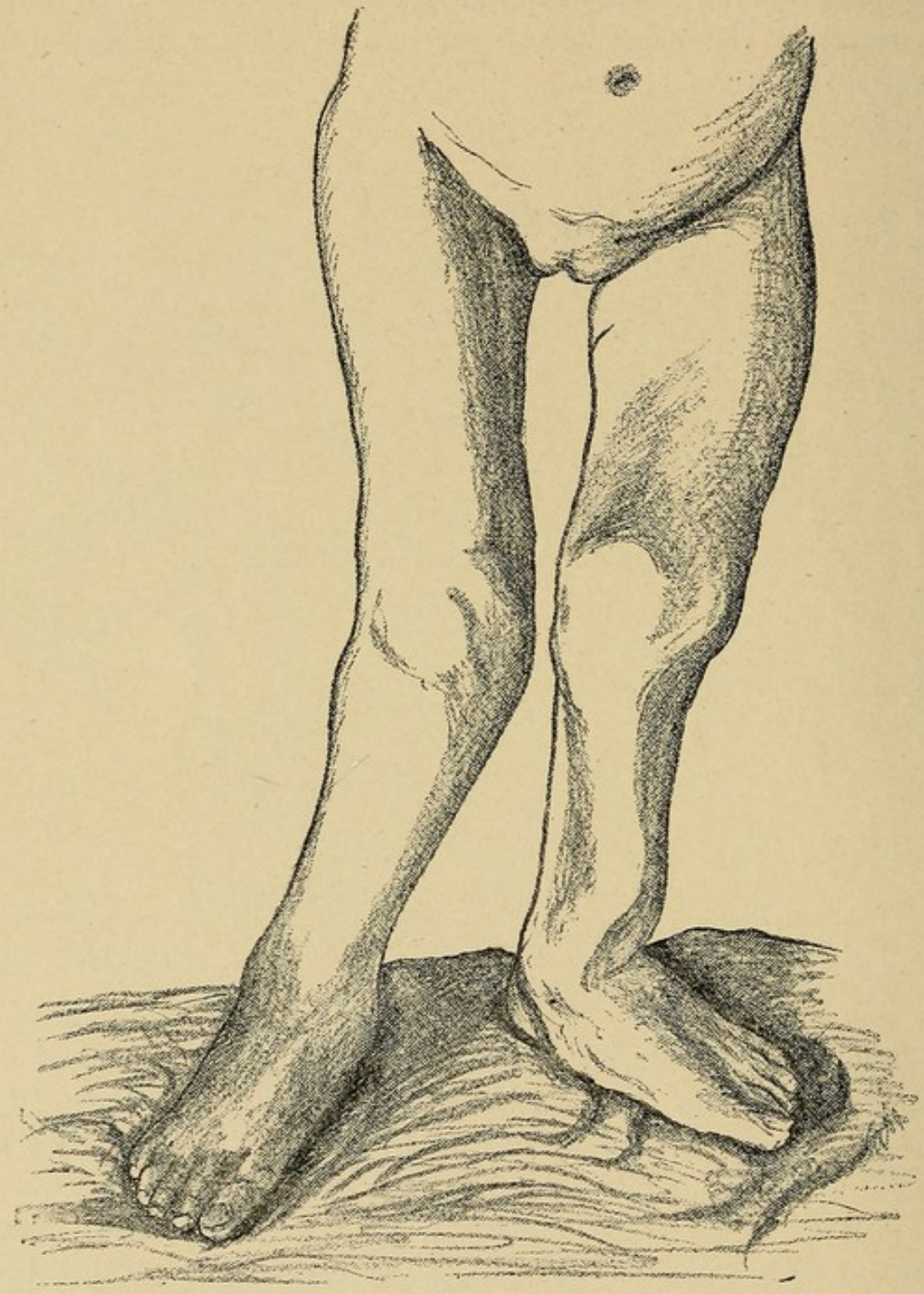


FIG. 66.—The lower limbs of a girl aged seven, showing, on the left side, marked curving of the tibia, flat-foot, and curving of the femur; and on the right the deformity known as knock-knee or "genu-valgum." From a photograph.

the periosteum becomes relaxed on the concave side, and thus the formation of new tissue is more abundant there; for the

blood-vessels are less stretched than on the convex side, and hence the nutrition is better.

Serious deformity is caused by the yielding of the soft bones in rickets. In this way rickety knock-knee and bow-legs are produced. Again the weight of the body may cause the soft tissue in the neck of the femur to give way, allowing the head of the femur to be depressed and giving rise to the deformity known as coxa vara (see Fig 67).

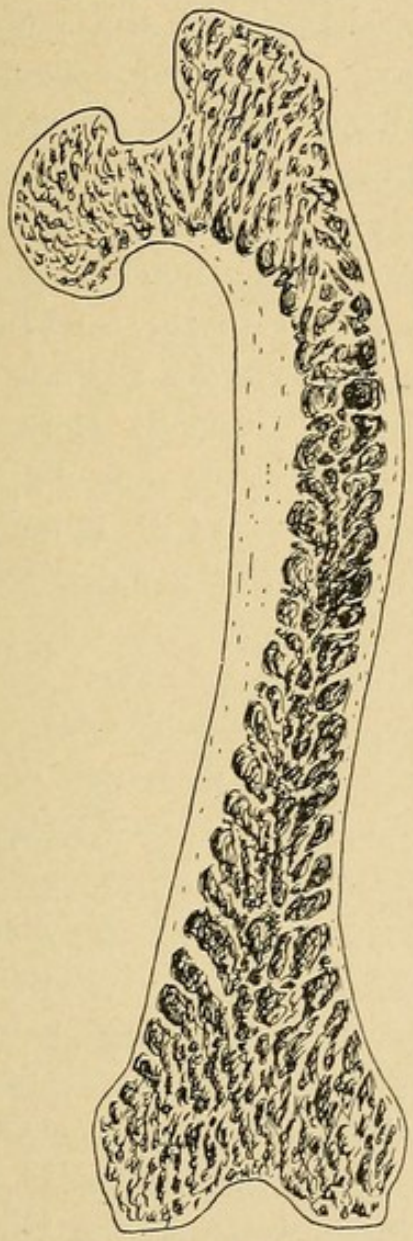


FIG. 67. — A section of a rickety femur. The head is depressed one inch and a half below the level of the top of the great trochanter, instead of being well above it. ¼ nat. (247.)

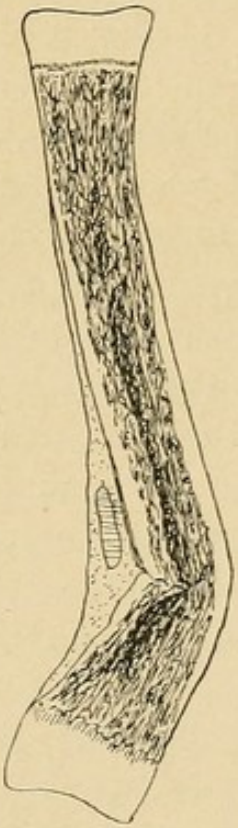


FIG. 68. — Section through the radius of an infant. There is a greenstick fracture. In the concavity of the bend there is a formation of callus, part of which is a translucent bar of hyaline cartilage. (83.)

The spine is often deformed. A C-shaped backward curve involving the whole spine is the commonest, but sometimes

lateral curvature occurs. The skull may become flattened on the crown and the frontal and parietal eminences bulge out. This gives the face a dwarfed appearance. The thorax sinks in at the junction of ribs and costal cartilages, thus producing pigeon-breast. The pelvis may become flattened.

The Nature of Rickets. The changes in the bones may be briefly summed up as follows: a stage of congestion is followed by hyperplasia of cartilage, vascular marrow, and often of periosteal bone. This hyperplasia is accompanied by defective calcification. These changes are best described as inflammatory in character. Comparing this hyperplastic osteo-chondritis, as the process which results in the thickenings at the epiphyseal regions may be called, with the slighter degrees of osteo-chondritis of congenital syphilis (see p. 156), a close histological resemblance is seen. There is the same hyperplasia of the proliferating layer of the epiphyseal cartilage and of the vascular marrow, but there is a difference in that the calcification of cartilage is increased in syphilis, whilst it is diminished in rickets. Another disease, osteo-arthritis, that affects bones and gives rise to inflammatory hyperplasia will be described in considering the joints. That the morbid processes of rickets is due to disorder of nutrition is hardly to be doubted. The symptoms of gastro-intestinal disturbance that accompany the disease, and the capacity of improper feeding—an insufficient supply of albuminous material and an excess of starch in the diet—to produce the disease both in animals and in children testify to this fact; as also do the good results of proper diet and hygiene combined with the administration of cod-liver oil and iron in the treatment. Whether the disease is brought about by some poison secreted by bacteria in the alimentary canal or by abnormal products of the cells of the body is not known.

The Differential Diagnosis of Syphilis and Rickets. The gross effects of syphilis may be compared with those of rickets by reference to Figs. 25 and 54 as examples of syphilis of bones, and Fig. 57 as a marked example of rickets. In rickets the changes affect both extremities of the bone, though, as in Fig. 57, one end may be more extensively altered than the other. If

the periosteal bone is affected the thickening occupies the whole of the shaft. In syphilitic bone shown in Fig. 54, the thickening of the bone and the changes at the epiphyseal line affect only one end of the bone. In rickets the whole of the skeleton may be affected whilst in syphilis, though the lesions of osteo-chondritis are often symmetrical, they are not as a rule so widespread as those of rickets. It is to be remembered, however, that syphilis and rickets often co-exist in the same individual. Kassowitz suggests that congenital syphilis, by disturbing the nutrition of the body generally, may cause rickets. In the skull there may be a very close resemblance between changes produced by these two affections. In a few cases rickets has been found by Barlow and Lees to cause "craniotabes."

The period of life at which rickets becomes pronounced is usually the first few months after birth, and not many cases develop after the age of two years. In a few instances the onset of the disease has appeared to be as late as seven or eight years. Another period of life at which skeletal deformities frequently arise is at the approach of puberty. These deformities are attributable in part to muscular weakness, but there may be also a weakness of bone, a condition termed *rickets of adolescence.*

Congenital Rickets. Belief in the occurrence of intra-uterine rickets has to a large extent been lost in this country, owing to the fact that at least two other conditions—cretinism and achondroplasia—quite distinct from rickets, had been mistaken for this disease. Yet Kassowitz, in one of the most recent (1881) and thorough investigations of the subject, says: "But still it is not, I believe, sufficiently known that in the great majority of cases rickets begins in a very early period of intra-uterine development. Most authors still make the error of believing congenital rickets to be a rare condition, whilst according to my experience this is by no means the case."[1] This opinion is based on the careful histological examination of new-born children, and hence is not to be disregarded.

Sporadic Cretinism, which has also been called "fœtal rickets," is described later with the diseases of the thyroid gland. The changes in the bones in this condition are due to a cessation

[1] Kassowitz, *Rickets*, 1881, p. 35.

of cartilage proliferation at the epiphyseal lines, where an ingrowth of fibrous tissue is found separating epiphysis from diaphysis. Hence the bones are greatly shortened. The condition is the converse of that of rickets, in which the epiphyseal cartilage proliferates in excess.

Fragilitas Ossium, or symptomatic osteopsathyrosis (Gr. *psathyros* = friable), is a state observed in infants, and is marked by fractures occurring in the bones from slight causes and without there being any obvious condition, such as scurvy, to account for the condition. An instance recently came under my notice in a male child aged ten months. When he was five days old the mother noticed that there was inability to move the right thigh and arm and the left ankle. Soon afterwards rings of callus about united fractures were found in the middle of the right femur, the lower third right humerus, and lower third of the left fibula. Fractures continued to occur from the slightest cause. The bones re-united readily. The child had congenital syphilis and craniotabes associated with rickets.

Achondroplasia, a condition in which the bones are stunted, but which is not associated with the atrophy of the thyroid gland that characterizes sporadic cretinism. To it the name "achondroplasia" has been applied. The bones of the limbs and other cartilage bones, such as the base of the skull, which begin to ossify early in fœtal life, are stunted and thickened. The spinal column and the bones of the hands and feet remain of normal size, as do the scapulæ and clavicles. The defects in the development of the bones, like those of sporadic cretinism, appear to depend upon an arrest of the proliferation of the epiphyseal cartilages. In most cases the fœtus are born dead, in others death occurs in infancy. The few subjects of the disease who have survived are dwarfs, but they show none of the mental and other defects that characterize sporadic cretinism.

Scurvy Rickets. Infantile scurvy is not uncommonly associated with rickets, and when this occurs Cheadle and Barlow have pointed out that sudden swellings about the bones are liable to occur from effusion of blood beneath the periosteum. Spontaneous fractures at the extremities of the shafts of the long bones are also met with. The condition occurs in the

children of both poor and rich: from neglect or want of proper food in the first class, and from rigid adherence to artificial and sterilized foods in the latter.

Mollities Ossium, or Osteomalacia. This disease is marked by a softening and bending of bones. It is commonest in middle-aged women, and frequently commences during pregnancy; but it occurs also in men, and has been observed in children. It is usually progressive and fatal, but in a few cases recovery has occurred. Extreme deformities of the ribs, pelvis, vertebræ, and limbs are produced. The deformity of the thorax is the usual cause of death. After death the bones may be readily cut with a knife, and they may be so light that they float in water.

The histological change in the bone is exactly like the effect of decalcification by acids. This process begins in the parts of the bony trabeculæ that border on the Haversian canals and other marrow spaces. The decalcified parts are progressively absorbed so that the marrow spaces enlarge. The spaces are filled with cellular fatty marrow, which varies in colour from yellow to deep crimson. It should be remembered that a diffuse, cancerous, or sarcomatous infiltration of bone sometimes produces a softening similar to that of osteomalacia.

Osteitis Deformans is a disease of old age, and is marked by a peculiar inflammatory affection of the bones. It occurs in both sexes. The bones of the limbs are thickened, softened, and curved. There may be tenderness over the bones. The bones of the skull are thickened so that a person's attention has been drawn to his condition by his requiring from time to time a larger sized hat than he had previously worn. The forehead becomes prominent, the clavicles thickened and curved. The femora curve outwards and forwards, the tibiæ curve forwards. The upper part of the spine also bends forwards. The thorax falls in from softening of the ribs, and the patients may die from the effects of impaired respiratory movements.

On examining a bone affected by osteitis deformans after removal from the body, the surface is found to be rough. The articular extremities of the bone are not enlarged. On making a section the compact tissue of the bone will be found to be thicker

on the convex than on the concave side, and to be more porous than normal. Comparing a curved bone of an old person who suffered from rickets in childhood with one deformed by osteitis deformans, it will be found that in the rickety bone there is a smooth surface, that the compact tissue is as hard as, or harder than, normal, and thicker on the concave than on the convex side.

Hyperostosis and Leontiasis Ossea. Diffuse thickening of the bones of the face, especially about the antrum of Highmore and the frontal sinuses, is not uncommon. Sometimes the process, starting in some of the bones of the face, slowly increases, and extends until all the bones of the face, and the supraciliary ridges and frontal bones are greatly thickened by the formation of hard coarse bone. Such cases usually commence in early life. The prominent eyebrows and cheek bones give a leonine aspect to the countenance, hence the term "Leontiasis Ossea," suggested by Virchow. In the course of years the orbits, the nasal fossæ, and the auditory meatus may be greatly narrowed, so that the functions of the chief sensory organs are destroyed. The disease appears to hold a place between inflammatory formations and new growths.

NEW GROWTHS OF BONE.

The complex structure of bone, comprising fibrous, osseous, cartilaginous, and other tissue, offers wide possibilities in the way of morbid growths. Those which occur most frequently are bony and cartilaginous among the non-malignant, and the sarcomata among the malignant tumours. Since bone contains no epithelial tissue primary cancer cannot occur, but secondary cancer is common. The more important growths of bone may now be considered in detail.

Osteoma. Adventitious processes of bone resulting from inflammation, such as may form from the callus of a fracture, or in a tendon, *e.g.* rider's bone in the tendon of the adductor longus, syphilitic or other ossifying inflammation may be distinguished as osteophytes, ossified nodes, etc., from new growths of bone or osteomata. The commonest form of growth is the

spongy osteoma which usually springs from the periosteal aspect of a cartilage-formed bone such as the long bones of the limbs, the scapula, and the os innominatum, etc. They most frequently arise from the shaft near the epiphyseal line, and are covered on the surface by a layer of cartilage which is usually thin, but may be as much as a quarter of an inch in thickness. Their cartilaginous covering suggests their origin in some of the isolated remains of the epiphyseal cartilage which are to be found even in normal and more frequently in rickety bones towards the ends of the diaphysis. They are sometimes multiple, and affect nearly all the bones of cartilage origin. In this case they may be hereditary in some members of a family. Fig. 69, from Coats's *Pathology*, shows bones which are the seat of multiple spongy osteomata.

Such tumours are not uncommonly solitary. Save for the inconvenience they may cause by producing deformity about joints, disturbing the attachments of muscles, or causing pain by pressure on nerves, they are not dangerous. When they are exposed to friction bursæ are sometimes developed over them. When it is desirable to remove them care must be taken to gouge freely the site of their attachment so that all trace of the investing cartilage is taken away. In one instance spongy

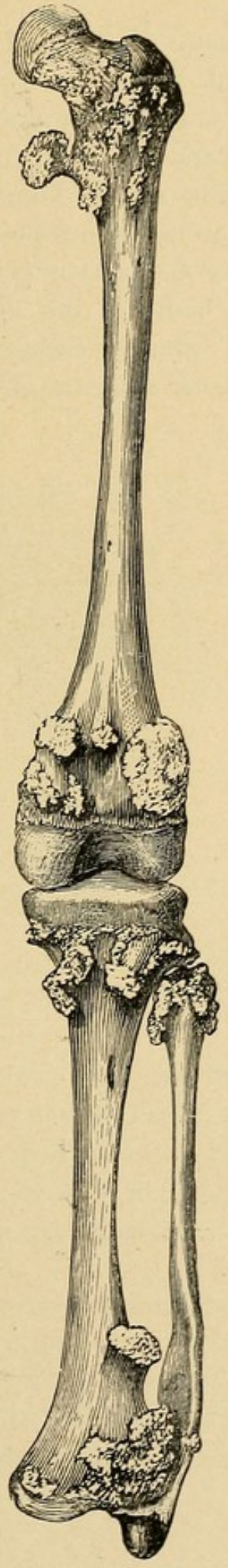


FIG. 69.—Multiple exostoses of femur, tibia, and fibula. These are grouped towards the ends of the diaphyses. At its lower end the fibula is flattened by the exostoses growing out from the tibia. From a boy ten years of age. One third the natural size. (VIRCHOW.) Coats's *Manual of Pathology*.

osteomata disappeared by absorption after being accidentally crushed.

Hard Osteomata are of two kinds: one variety has the characters of ordinary compact tissue, the other is of still denser structure, and is known by the name of ivory exostosis. The first variety occurs chiefly on the shafts of the long bones.

Ivory Osteomata (Fig. 70) occur upon either or both surfaces of the bones of the skull. Now and again they grow into the frontal sinuses, causing their gradual dilatation. They are of importance from the effects they produce upon the brain. Their

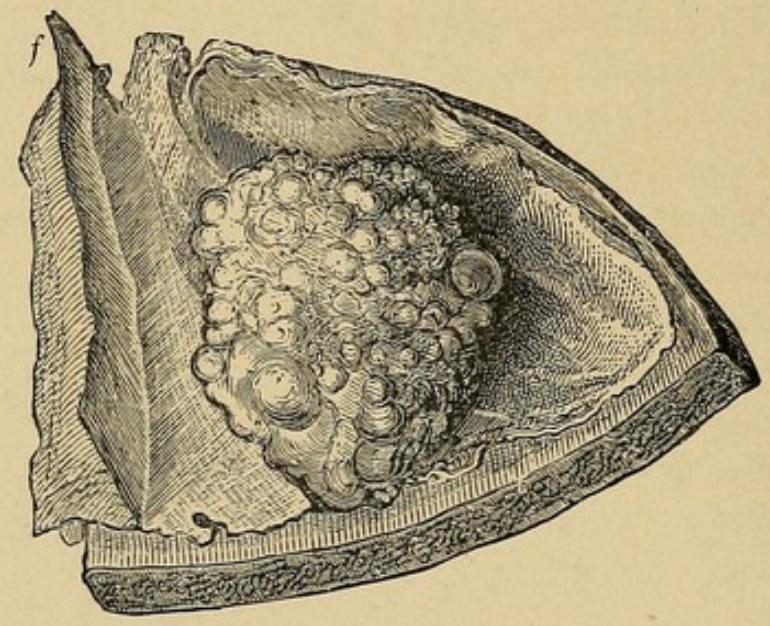


FIG. 70.—Internal ivory osteoma of the frontal bone. The tumour has a tuberculated surface and a narrow base. After Virchow. Coats's *Manual of Pathology*.

extreme hardness renders it unwise to attempt to chisel them away. When it is necessary to remove them this must be done by drilling them with a dental bur and sawing, or by dividing the normal bone beyond their attachment. Both spongy and ivory "exostoses" occur in the bony part of the external auditory meatus.

Chondroma is more frequently met with in bones that have been developed from cartilage, but it also occurs in membrane bones; a fact that is not surprising when it is remembered that hyaline cartilage sometimes appears in the callus of fractures, and has been observed to be formed from the periosteum in rickets;

nay more, cartilage is a frequent constituent of new growths of glands such as the parotid, and testes which in normal conditions have no cartilage about them. Cartilage is but one modification of connective tissue, and is of frequent occurrence in new growths. It will be mentioned again as a constituent of one form of malignant tumour of bone, the so-called osteo-chondroma.

In the long bones cartilaginous tumours may spring from either the medulla or the periosteum.

Central Chondromata are most frequently met with in the small bones of the hand and foot (see Fig. 71). When these central or en-chondromata are small an attempt may be made to rid the patient from them by removing some of the outer layers of the bone, and then gouging out the growth and its attachment. When the growths are large and numerous amputation is the only resource.

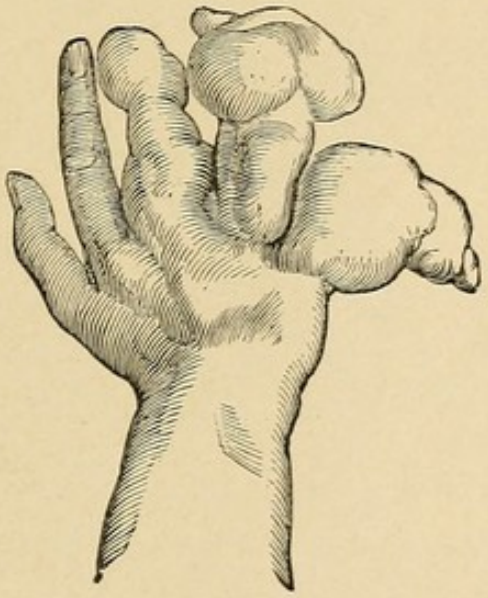


FIG. 71.—A hand showing the effects of multiple central chondromata of the phalanges. (CORNIL and RANVIER.) From Coats's *Pathology*.

In the larger long bones, and in the flat bones, chondroma usually arises from the periosteal surface. The main characters of such growths are readily appreciated on studying a well-marked specimen such as that represented in Fig. 72.

The *lobular* character of large chondroma is a necessity, for large masses of non vascular tissue like cartilage require supporting fibrous tissue to conduct blood-vessels into it. Moreover if the structure of inflammatory cartilage growths, such as those of osteo-arthritis, are examined, the mode of growth of large chondromata is found to be reproduced.

The *ossification* at the base of a peri-chrondroma is comparable to the ossification of callus and periosteal sarcomata, and depends on the power of bone and periosteum to convert into bone new connective tissue arising from them.

Areas of Calcification are of frequent occurrence in chondroma

and new growths of all kinds, especially in their degenerated portions, where the process may be compared with the formation of phleboliths, etc.

The formation of Cysts in chondroma is due to the occurrence of myxomatous metaplasia—that is to say, in certain areas of the growth the characters of the cells and matrix gradually change, the cells becoming branched and the matrix semifluid

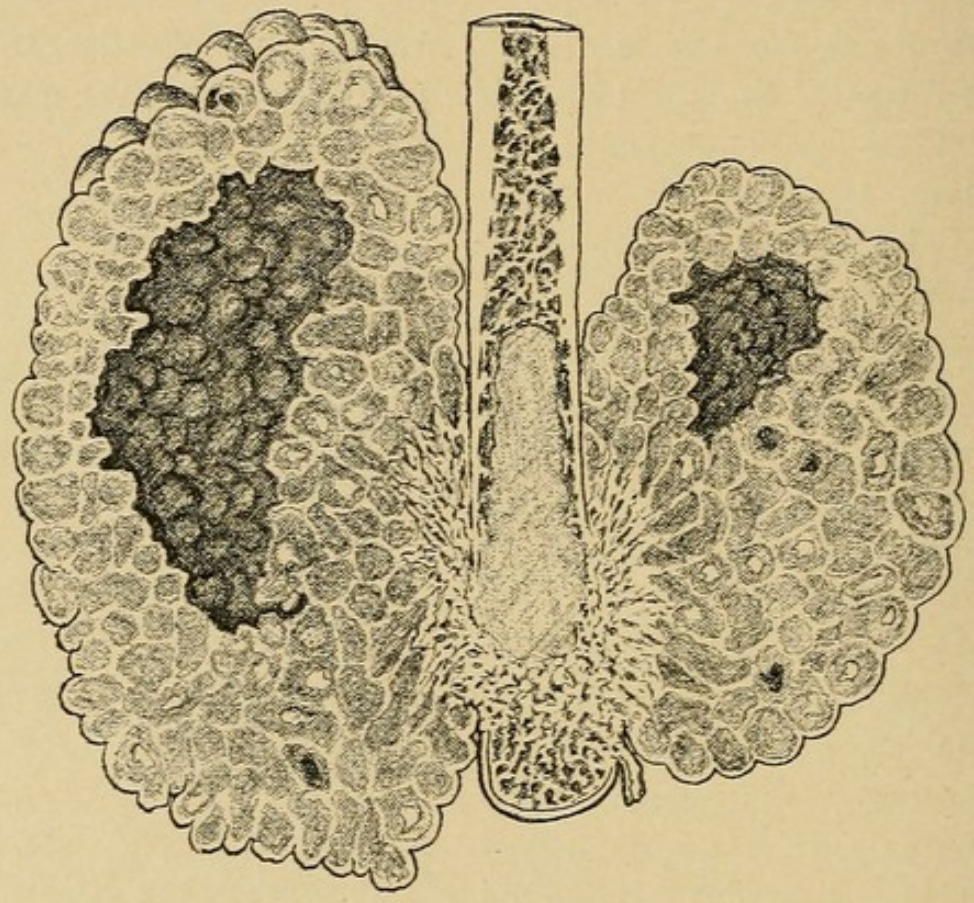


Fig. 72.—Vertical section of a femur from which springs a large peripheral chondroma. The lobular character of the tumour and the ossification of its base are recognizable. The growth has extended into the medullary cavity. The large cavities are due to portions of other lobes of the tumour having fallen out after the section was made. The smaller cavities are cysts caused by degeneration of myxomatous areas. (64.)

with the presence of a mucigenous substance. Cartilage and mucous tissue merge into one another :—chondroma and myxoma are formed as part of the same growth. Mucous tissue readily degenerates by complete liquefaction, thus forming cysts.

Sarcomatous Metaplasia occasionally occurs in chondroma; the tumour then loses its innocent character.

Fibroma of Bone. Considering the fibrous structure of the periosteum, fibromata are not so common in bones as might be

expected. They occur chiefly on the periosteal aspect of the bones of the base of the skull, where the growth constitutes the naso-pharyngeal fibroma. This growth is very tenacious of existence, tending to recur locally after removal, and hence, like the common fibrous epulis, may be regarded as a fibro-sarcoma. For details of the naso-pharyngeal fibromata, see "Diseases of the Respiratory Tract."

Malignant Disease of Bone.

Sarcoma is of common occurrence; indeed bone is of all tissues perhaps that most frequently affected by this disease. There are two well-defined types of sarcoma in bone—periosteal and central. The former shows a much greater tendency to local spreading and to general dissemination than does the latter.

Periosteal Sarcoma varies in histological structure. The spindle-celled variety is the one most frequently encountered, but round-celled, alveolar, and other forms of sarcoma are common. Now and then the blood-vessels of periosteal sarcoma are developed to such an extent that the growth pulsates, then in certain situations the conditions may be mistaken for aneurysm of a large artery. A form of sarcoma that has been mentioned previously is the "osteoid chondroma" of Virchow. This growth is less malignant than the other forms of periosteal sarcoma, but locally it may spread from end to end of a long bone. Periosteal sarcomata of the more common type, such as that depicted in Fig. 73, tend to ossify at their base. The ossification commonly takes the form of spicules of bone, which radiate into the growth from the original periosteal surface.

Occasionally the ossification of a periosteal sarcoma assumes the form of an irregular meshwork of bone traversing the tumour. Macerated specimens of this condition was formerly known by the quaint name, "Spina Ventosa."

Central Sarcoma is encountered in all kinds of bones. In the long bones it may begin in any part of the medullary cavity or cancellous tissue, but the articular extremities are perhaps more liable than the other regions of the bone.

The growth is of the myeloid or giant-celled type as a rule. A fresh section of the growth has a purplish appearance from the presence of blood-extravasation. Cystic spaces may result from hæmorrhage or colliquative necrosis, and, sometimes, from myxoma-metaplasia. The effects of the growth are first pressure-atrophy, and in time bulging of the outer part of the bone, which may become so thin that it yields to pressure with a

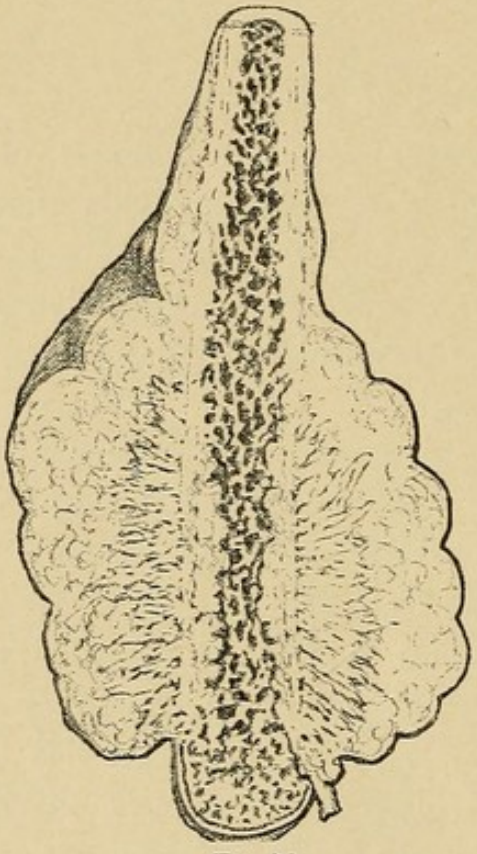


Fig. 73.

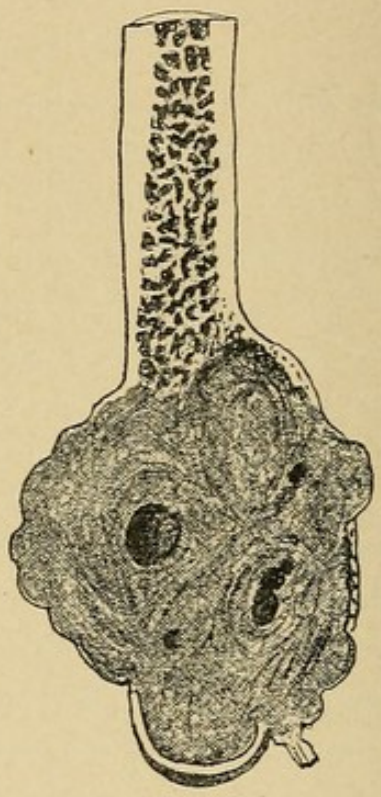


Fig. 74.

Fig. 73.—Vertical section through a periosteal sarcoma of the lower end of the shaft of the femur. The base of the growth is ossified. The growth is extending upwards in the periosteum and inwards into the medullary cavity.

Fig. 74.—Vertical section of the lower end of a femur which is the seat of a myeloid sarcoma. There are two cystic spaces in the growth: the surrounding bone has been thinned, dilated, and perforated by the growth.

crackling sound (egg-shell crackling). Finally the whole thickness of the bone may be destroyed, and the growth then projects from the surface of the bone. The process of absorption of the bone contrasts with the effects of the growing mass on the articular cartilages, which, though they may be pushed before the growth, are seldom or never perforated by it. The greater effect produced by the pressure of an aneurysm on bone as compared with the effect of the same pressure on cartilage

may be recalled as a parallel. The commonest situations for myeloid sarcoma are the lower jaw and lower end of the femur and the upper end of the tibia.

The weakness of the bone caused by the presence of a central sarcoma often determines what is termed a "spontaneous fracture." That is a fracture determined by some of the ordinary movements of the body, and without any unusual violence. Sometimes a central sarcoma spreads within the medullary space until the latter is quite filled. If, in these circumstances, the growth is rich in blood-vessels, arterial pulsations become perceptible externally wherever the compact tissue of the shaft has been absorbed. In some such cases the growth has broken down centrally, leaving a large cavity filled with blood. This condition has been erroneously termed "aneurysm of bone." The difference in malignity of periosteal as compared with central sarcoma may be illustrated by stating the fact that, for periosteal sarcoma of the lower end of the femur, amputation at the hip-joint is called for, whilst for central sarcoma of the same part, amputation through the middle of the shaft of the femur is sufficient.

CHAPTER IX.

DISEASES OF THE JAWS AND TEETH.

THE jaw bones are liable to the affections of bones in general, and in addition to others which the presence of the close investment of the alveolar process by the mucous membrane of the mouth, and in the upper jaw, the presence of the antrum of Highmore, and the proximity of the nasal fossæ entail. The chief morbid conditions of the antrum of Highmore will be considered with the diseases of the respiratory tract. A few special features connected with acute suppurative affections may be noticed here.

Acute periostitis of the jaws, as in bones generally, is common after the acute specific fevers, but the jaws are exposed to suppurative inflammation spreading from the exposed pulp of decayed teeth to the periosteum lining the alveoli, and there starting a periostitis which, though usually limited in extent and constituting a "gum-boil," in some cases spreads more widely and causes extensive necrosis. The slowly progressive and usually fatal necrosis which used to be observed in makers of lucifer matches, when common phosphorus was used for that purpose, is now rarely seen. Fractures of the body of the lower jaw are usually compound, communicating with the mouth, and hence necrosis of the exposed ends of the fragments is not uncommon. Necrosis of the jaws may have any of the terminations indicated under "Acute Inflammation of Bone," and there is in addition to the danger of pyæmia, that of septic bronchopneumonia, from the inhalation of foul discharges from the mouth, and in the case of the upper jaw, of extension of

suppuration to the base of the skull and the brain. If the part of a lower jaw that carries the teeth is destroyed by necrosis (see Fig. 75), a new piece of bone is usually generated from the periosteum, but the teeth are, of course, absent from it. After necrosis of the upper jaw, however, there is as a rule but little formation of new bone. In necrosis of the jaw the question as to whether the dead bone should be removed at once (sub-periosteal resection), or after waiting for the formation of a sheath of new bone (sequestrotomy) will arise. The latter course is usually followed.

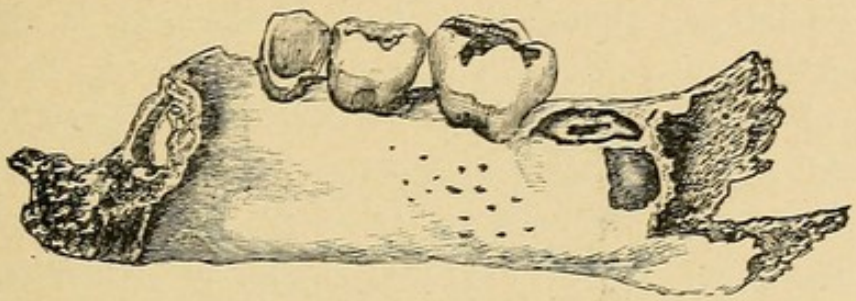


FIG. 75.—A sequestrum comprising the whole thickness of part of a lower jaw and the temporary and permanent teeth. From a girl aged six, in whom periostitis followed small-pox. From Roughton's *Oral Surgery*. (203.)

Tubercular Necrosis. In children who have tubercle of the lymphatic glands or other parts, the jaws are liable to undergo an insidious necrosis, which appears to be tubercular in character. The jaws are one of the sites of actinomycotic infection (see p. 88).

Tumours of the Jaws. In addition to the new growths that affect other bones, the jaws are exposed to those that arise from the teeth and the enamel organ: odontomes (see p. 184) and the upper jaw is affected by all the morbid conditions of the antrum (see the section on "Diseases of the Respiratory Tract.") Special note should be made of two growths which occur in the jaw: epulis and endosteal myeloid sarcoma.

Epulis (Gr. *epi* = upon, *oulis* = gum) is a firm fibrous tumour which springs from the periosteum lining an alveolus and from that of the alveolar process. In its growth it pushes aside the two teeth between which it lies. Histologically an epulis is chiefly formed of fibrous tissue, but immediately beneath the epithelium which covers the surface there are considerable

numbers of small round cells among the fibres. This fact would alone place the growth under suspicion as being of a slightly marked sarcomatous nature. And though in many persons it grows insensibly for years, yet now and again a rapid sarcomatous development occurs, and warrants the classification of the common epulis as fibro-sarcoma, and dealing with the growth by removing both the tooth from the socket of which it springs and the tooth next to it, and then cutting away the corresponding portion of the alveolus.

Diffuse Hypertrophy of the Gum is a condition in which a firm fibrous thickening occurs, causing the gum to grow around the teeth. Sometimes almost the whole of the gum is affected, at other times only a part. The condition, like epulis, requires removal of the alveolar process as well as the growth.

Myeloid Sarcoma not infrequently arises in the lower jaw bones, where it produces rarefaction as in other bones. The bone is first expanded and finally absorbed, and when the growth projects beneath the gum it sometimes causes rounded bluish swellings about the teeth. This appearance has been termed "Malignant Epulis." The disease is commonest in youth. If the segment of jaw containing the growth and some sound bone on each side of it is removed, the growth will probably not recur.

Odontomes.

Multilocular Cystic Tumours of the Jaws are growths of variable clinical characters, sometimes having an extremely slow, at others a more rapid, course. In structure they present one constant histological feature, *viz.* branched columns of epithelium. The central cells of the older columns break down something after the manner of the cells of the enamel organ in the formation of the tooth-sacs, and with the same result, namely, the formation of cysts. The connective-tissue part of these growths is variable; usually it is composed of fibrous tissue and bone, forming thin septa between the cysts. When this is the case the progress of the disease is slow, and six or ten years may pass before the patient applies for treatment, on account of a swelling of the jaw, produced by gradual expansion from within. In other instances

the tumour may reach a considerable size in a few months. In such cases the intercystic tissue is found to have the character of spindle-celled sarcoma. These tumours, as Coats has pointed out, might from their clinical characters be classed as adenomata and adeno-sarcomata. Their mode of development suggests their origin in unused relics of the common enamel organ, so that they are by Eve and Bland Sutton classed with the odontomes. When such tumours have reached a certain size they cause the teeth to fall out, and then some of the cysts may open on the surface, causing a discharge of sticky fluid.

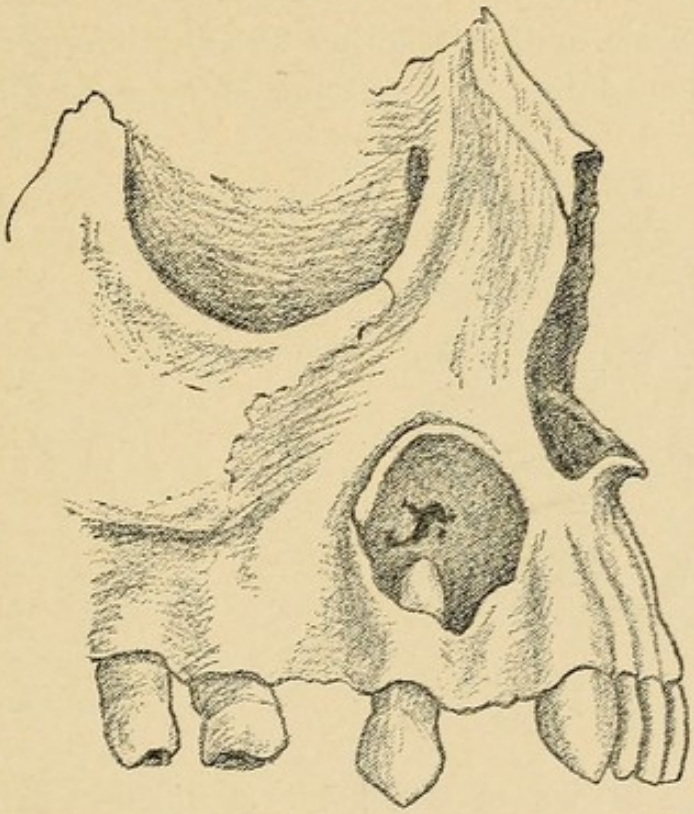


FIG. 76.—A cyst due to chronic abscess about the root of a tooth. There is an aperture in the posterior wall of the cyst leading into the antrum. After Zuckerkandl, *Anatomie der Nasenhöhle.*

Simple Cysts of the Jaw. The commonest is that which forms about the root of a carious tooth owing to the presence of a chronic abscess. The wall of the alveolus is gradually absorbed, and when the periosteum is reached it is lifted up and forms a new bony wall over the abscess. Such cysts may open into the antrum and cause antral empyæma (see Fig. 76).

Dentigerous Cyst of the Jaw. It sometimes happens that one of the permanent teeth fails to appear on the surface

owing to its developing in an abnormal direction. In such a case the sac of the tooth remains closed, and sometimes (not always) becomes distended with fluid forming a dentigerous cyst. Such a cyst may expand the jaw like a solid tumour causing bulging, thinning, and egg-shell crackling, etc. If such a cyst be opened the tooth is found in the cavity where it has most frequently come to lie free, having been loosened from its socket by the gradual expansion of the cyst. If the fang of the tooth remains fixed in the bone the tooth is found to have an abnormal position, *e.g.* to be directed horizontally or to lie vertically upside down. The tooth may be represented by a calcareous nodule or be altogether absent. The wall of the cyst may be thickened by the formation of bone or fibrous tissue.

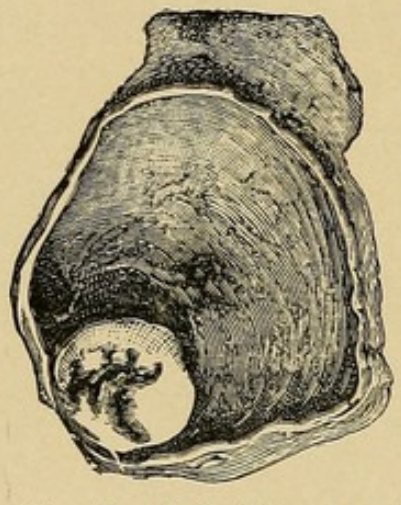


FIG. 77.—A dentiferous cyst. From Roughton's *Oral Surgery*.

FIG. 78.—A radicular odontome. After Salter.

Fibrous Thickening of the Sac of a developing tooth. This condition has been met with in animals and rickety children.

Osseous Thickening of a Tooth-Sac has also been observed. To this class of tumours Sutton has given the name of cementome. In some cases, both in man and animals, such tumours have been found to contain numerous ill-formed teeth, which represent the unerupted teeth which have formed from the sac in excessive numbers.

Tumours of the Roots of Teeth. If either the cement or the dentine of the root of a tooth is over-developed the condition is termed a radicular odontome. In Fig. 77 a typical example is depicted.

Composite Odontomes form craggy masses which have been often mistaken for osteomata. They have been found embedded in the jaws, and in one case the tumour was contained within the antrum of Highmore, whence it escaped through the cheek of a man aged 36. This tumour weighed nearly 15 ounces.[1]

Histologically such tumours are composed of dentine, enamel, and cement irregularly blended.

[1] Hilton's case, quoted by Bland Sutton, *Tumours*, p. 45.

CHAPTER X.

DISEASES OF JOINTS AND BURSÆ.

Synovial membrane and the supporting ligaments with the articular cartilage are the joint-structures proper. The former is a vascular membrane which is liable to infection by way of the blood stream, and is very commonly the seat of suppuration. The secretion of the synovial fluid is also sensitive to toxic as distinguished from infective states of the blood. The articular cartilage is non-vascular and is never the primary seat of infection. It is, however, usually the first tissue that is the seat of deposit of urate of soda in gout. When inflammatory disease spreads to the articular cartilage chondritis is the term employed. The ligaments, like the articular cartilages, are seldom the primary seat of disease. When inflammation spreads to the ligaments this is expressed by the term para-arthritis. When all the joint-structures are affected by inflammation the condition is termed pan-arthritis (Gr. *pan* = whole, *arthron* = joint). The bursæ, whether they are of the variety that form the synovial lining of tendon-sheaths, or are of the saccular variety which is found over bony prominences, are subject to the same diseases as the synovial membrane of diarthrodial joints. It has been seen in considering the diseases of bones that suppurative and tubercular arthritis very frequently arise by extension from the articular ends of the bones. Syphilitic joint-disease sometimes arises in the same way. When synarthrodial joints, like those between the bodies of the vertebræ, become the seat of disease, this usually begins in the neighbouring bones.

Traumatic Synovitis follows injuries such as a fall on the knee, sprains, etc. It is marked by a rapid filling of the joint with serous liquid effusion. If the violence has been sufficient to lacerate the parts blood is effused into the synovial cavity constituting *Hæmarthrosis.* In hæmophilia and scurvy hæmar throsis occurs spontaneously, *i.e.* from the ordinary use of the joint.

Chronic synovitis or hydrops articuli is usually a sequel of acute synovitis, and is most frequent in the young. It is commonest in the knee. Though not accompanied by much pain the distension of the joint gives a sense of weakness. It may be necessary to drain the joint.

Infective Suppurative Arthritis. After perforating wounds, in pyæmia, and sometimes in gonorrhœa, the synovial membrane becomes the seat of suppuration. Suppuration in a joint is classed as arthritis, because not only the synovial membrane but all the joint structures rapidly become involved. If a joint is opened during suppurative arthritis the synovial membrane is found to be injected and swollen, and if the condition has lasted for a sufficient time the articular cartilages are found to have been wholly or in part removed, exposing the bone beneath them. The appearance of the articular surfaces suggests that the cartilage is removed by a process akin to digestion: the cartilages look as if they had been dissolved away in patches.

The forms of arthritis that follow scarlet-fever and other exanthemata belong to this category, though they are sometimes unwisely referred to under such terms as "scarlatinal rheumatism."

If suppurative arthritis continues unrelieved the ligaments of the joint become inflamed, soften, and allow of displacement of the bones: pathological dislocation. In young children a form of primary suppurative arthritis is frequently observed, and must be distinguished from that which is secondary to infective osteomyelitis. The primary form yields readily to drainage, etc. In gonorrhœa it is probable that there are two distinct forms of joint affection: one, a synovitis due to absorption of toxic material, another, an arthritis due to gonorrhœal pyæmia. In

the latter condition pus is formed in the joint and gonococci are present in the pus. The synovitis of acute rheumatism so very rarely becomes purulent that suppuration in a joint may be taken as proof that the condition is not rheumatic.

Acute Rheumatism or Acute Rheumatic Polyarthritis is associated with the general constitutional symptoms, the full consideration of which does not come within the scope of this work. Several joints become affected one after the other. The synovial effusion is marked by great pain and tenderness, and is accompanied by some para-arthritis. The distribution of rheumatic joint-effusions recall those of pyæmia. And possibly the joint affections, which are not infrequently observed after the injection of antitoxic serum, throw some light on the nature of rheumatism, which may be the result of an intoxication. The tendency of acute rheumatism to simulate other affections and to become subacute or chronic, and lead to ankylosis, renders rheumatic affections of importance to the surgeon.

Chronic Ankylosing Arthritis,[1] **or Chronic Rheumatic Polyarthritis** (*arthritis pauperum*). This affection is marked by the joints of the body becoming one after another immovable from ankylosis. The disease may follow an attack of acute polyarticular rheumatism, or it may begin insidiously. Once begun it continues for the rest of the patient's life. The changes in the joint have some resemblance to those of osteo-arthritis, but differ in that the changes in the cartilage are accompanied by less proliferation of cells. The cartilages become villous and converted into fibroid tissue, which is replaced by vascular fibrous tissue, the blood-vessels of which are derived partly from the neighbouring synovial membrane, partly from the marrow-spaces of the underlying bone. Fibrous adhesions form between the articular surfaces. The remains of the articular cartilages become converted into osseous or osteoid tissue. The fibrous ankylosis once begun becomes more extensive, and finally the fibrous adhesions become converted into bone. In old-standing cases this remarkable disease may result

[1] This account is chiefly derived from MacAlister and Cattell's translation of Ziegler's *Special Pathological Anatomy*, chap. xxiii., p. 275, 1896.

in ankylosis of almost every joint from the toes and fingers to the jaws.

Osteo-arthritis or Chronic Arthritis Deformans, etc., is a disease chiefly of middle and advanced age, but occasionally it is seen in childhood. Sometimes only one joint, such as the hip or shoulder, is affected, but more commonly several joints, and sometimes nearly all the joints, including those of the fingers and toes and of the spine, are implicated. In infants the condition is accompanied by marked anæmia and weakness, and also by swelling of the epiphyses (*osteo-chondritis*). Pain, stiffness, and deformity of the joints, deformity from distension of bursæ about the affected joints and hernial protrusions of the synovial membrane, with reflex wasting of the limb, are among the chief local symptoms. They are often accompanied by neuralgic pains about the joints, by gastric disturbances and eczema and other cutaneous eruptions. The disease is frequently combined with gout, and it is commonest in those of gouty descent. It appears to have nothing in common with rheumatism.

The Chief Anatomical Changes. The synovial membrane secretes an excess of viscid synovia. When this is in very great quantity the membrane is bulged out, and where the capsule of the joint is weakest this bulging is greatest, and may take the form of hernial protrusions. In many cases there is hyperplasia of the connective tissue of the synovial membrane resulting in the formation of numberless pear-shaped projections (see Fig. 79), which remind the observer of the warty condition often seen in the skin about chronic ulcers of the leg.

These hyperplastic synovial villi sometimes become separated from the membrane from which they spring, and so constitute one variety of loose bodies which occur in joints. They may become cartilaginous and the cartilage in them may calcify. It occasionally happens that in dissecting a joint after death dozens of these calcified loose bodies fall out.

The articular cartilage, like the synovial membrane, undergoes inflammatory hyperplasia. The multiplication of the cartilage-cells loosens the fibres of the cartilage matrix, and where the cartilage is exposed to friction the surface of the cartilage

becomes fissured, and some of the cells escape into the joint-cavity. This gives the cartilage a velvety appearance (Fig. 79, 2). This velvety cartilage is readily worn away, exposing the bone, as at 4 (Fig. 79). The exposed bone, if the joint is much used,

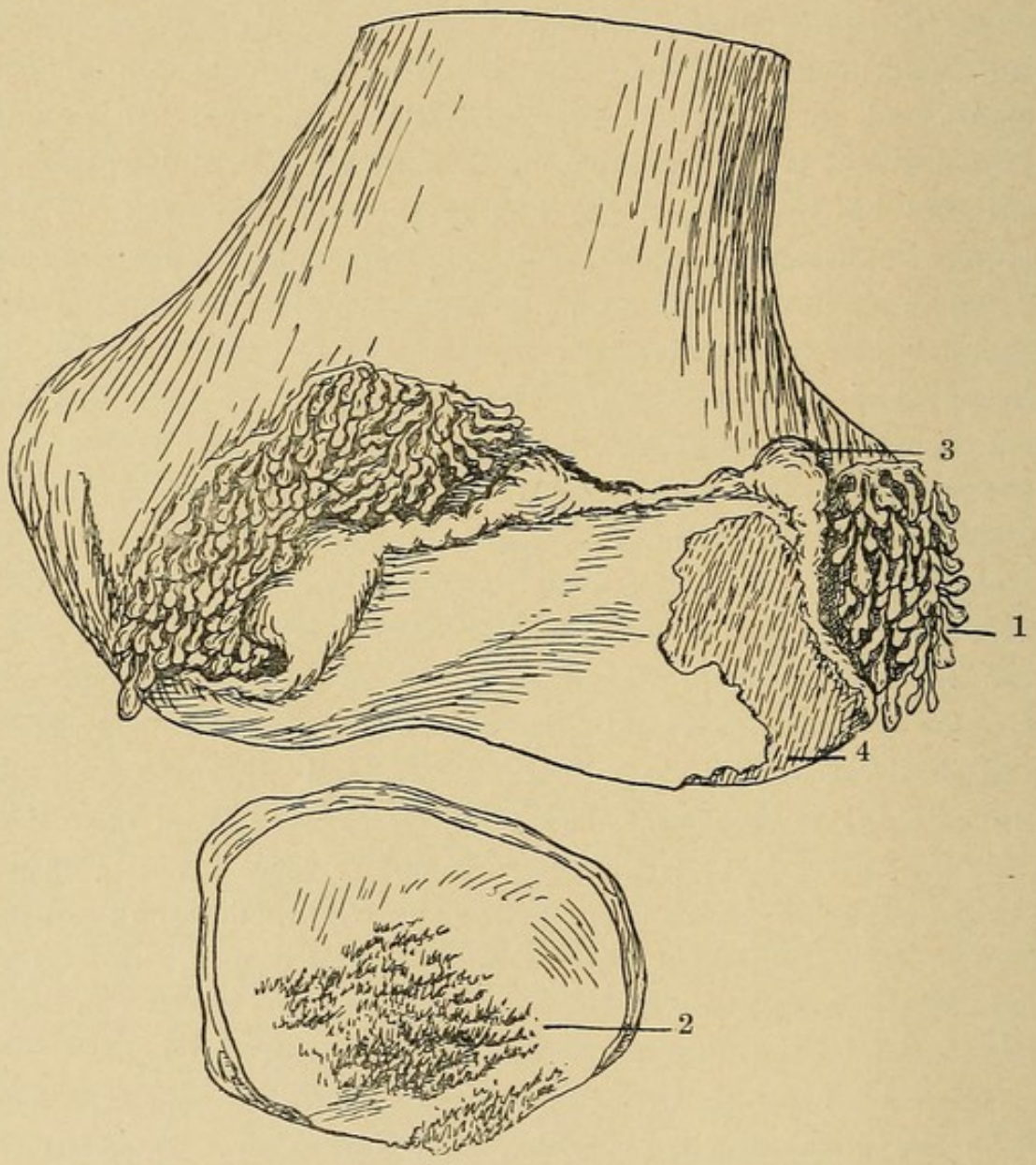


FIG. 79.—The lower end of a femur with the patella, showing changes due to osteo-arthritis. 1, Part of the synovial membrane with fibrous projections; 2, cartilage over the patella showing the velvety condition; 3, part of the collar of ecchondrosis and exostosis growing beyond the margin of the articular cartilage; 4, part of the articular surface of the femur denuded of cartilage by friction. (460.)

becomes grooved from friction, and assumes a peculiarly increased density, which gives it an appearance like the glaze on porcelain. At its margin, where it is not exposed to friction, the cartilage from the inflammatory overgrowth forms a collar

of ecchondrosis. Similar changes taking place in the bone beneath the cartilage forms a growth of bone (exostosis), on which that formed of cartilage rests. The histological structure in a case of osteo-arthritis combined with gout is shown in Fig. 80.

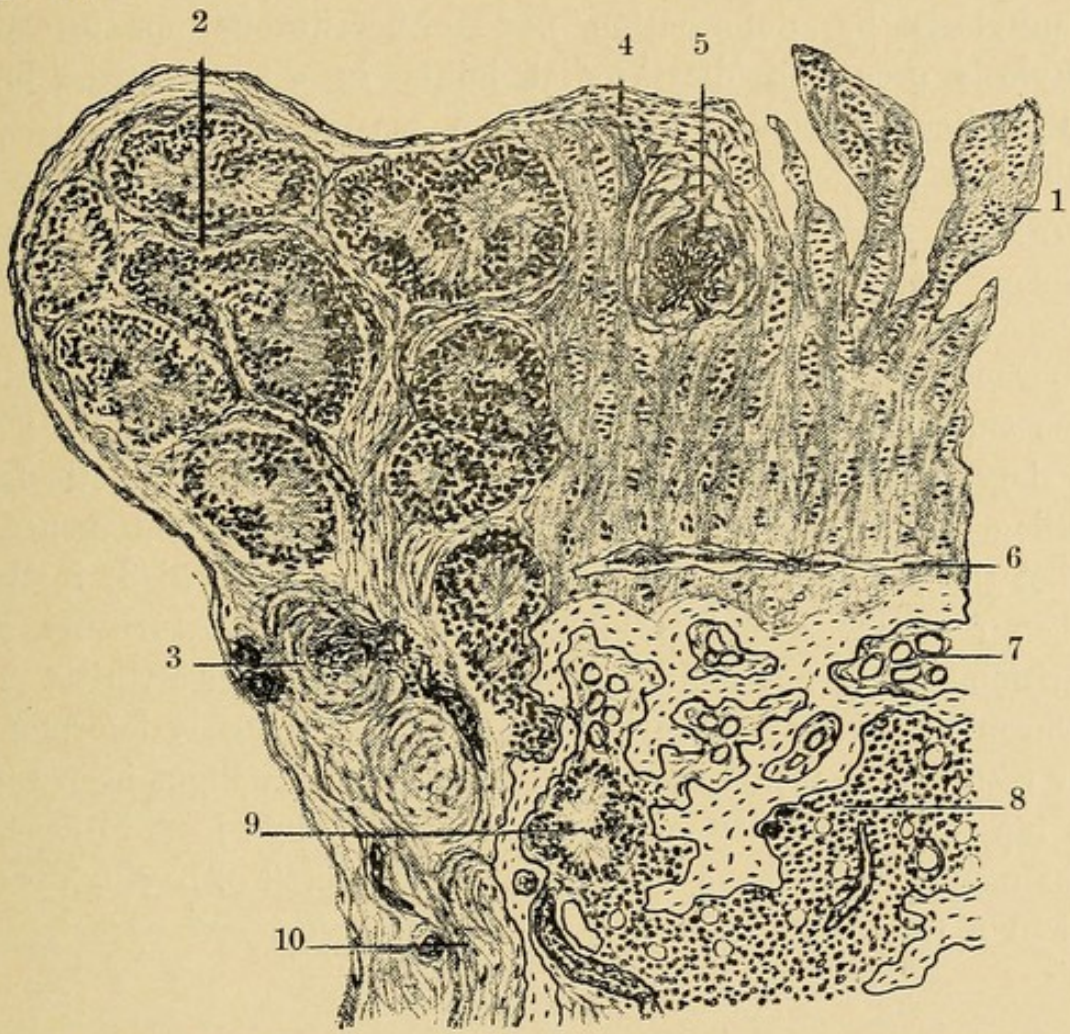


FIG. 80.—Section at the margin of the articular surface of a first metatarsal bone from a case of osteo-arthritis combined with gout. 1, Articular cartilage, the cells of which are proliferating, and the superficial layers of which are undergoing cleavage; 2, hyperplastic tissue of periosteal origin, and consisting of areas of dense fibroid tissue surrounded by cells, some of which are multinucleated. These cells are supported by connective tissue continuous with the periosteum and containing newly formed capillaries. 3, Commencing focus of inflammatory new tissue resembling those described under 2; 4, fibrous tissue derived from the synovial membrane growing for a short distance over the cartilage; 5, stellate cluster of urate of soda crystals in the articular cartilage; 6, cleft in the cartilage; 7, normal fatty marrow; 8, lymphoid marrow replacing the original fatty marrow; 9, (in the bone) focus of the newly formed fibro-cellular tissue resembling those forming "lip" at 2; 10, periosteum. In this instance the "lip" of new tissue consists neither of bone nor cartilage, but resembles the inflammatory foci seen around gouty "tophi" in the soft tissues.

The "lipping" of the cartilages, as the formation of ecchondro-exostes is commonly termed, is in some cases extreme; a large collar turned away from the articular surface, and consisting

chiefly of bone, then exists. Now and again, in osteo-arithitis, the denuded bony surfaces, instead of becoming indurated (porcellaneous), are rarefied and worn away progressively, so that in this way deformity arises either by allowing displacement ("dislocation by deformation"), or by interlocking ("ankylosis by deformation"). Neither fibrous nor osseous ankylosis occurs in diarthrodial joints in osteo-arthritis, but the bodies of contiguous vertebræ may become joined together by bridges of bone. The changes in the vertebræ are known as *spondylitis deformans.* The histological changes of osteo-arthritis of infants includes, besides the changes in the joints, excessive proliferation of the epiphyseal lines (*osteo-chondritis*).

Chronic Dry Ulcerative Arthritis is a term applied to conditions similar to osteo-arthritis, but marked anatomically rather by degenerative than proliferative processes in the articular cartilages, the matrix of which becomes fibrillated and fissured whilst the proliferation is wanting. It is met with chiefly in the *malum coxæ senile,* and is regarded as a senile disturbance of nutrition. It is not clinically separable from osteo-arthritis.

Chronic Traumatic Arthritis Deformans. Arthritis deformans may result from injury to a joint. Thus, after a slight injury to the hip joint deformity may gradually be produced, and in such cases medical men have been wrongly suspected of having overlooked a fracture.

Gout.

The general pathology of gout belongs to the medical aspect of pathology, but the surgical importance of the disease, and its frequent association with contraction of the kidneys, necessitate a careful consideration of certain of its aspects. As it affects the joints, it usually begins, according to Garrod, by the effusion of a clear liquid into the structures composing the joint, and from this a crystalline precipitate is deposited in the tissues. These events are accompanied by hyperæmia and swelling of the joint. The deposit generally occurs first in the articular cartilage, and on a joint affected by chronic gout being opened, the cartilages look as if they had been covered by a coat of white paint. On making sections the deposit is found to be

just beneath the surface of the cartilage. In pronounced cases of the disease the deposit is found in the periarticular and other soft tissues, where they constitute *Tophi* (Lat. *tophus* = tufa). These deposits in vascular tissues are accompanied by inflammatory changes[1] and the formation of new blood-vessels, similar to the condition shown in the hyperplastic tissue at the margin of the articular cartilage in Fig. 80. The central parts of the tophi are composed chiefly of crystals of urate of soda embedded in necrotic tissue. As the tophi enlarge extensive destruction of tissue may occur, so that cavities form which may open on the surface of the body, and so pave the way for pyogenic

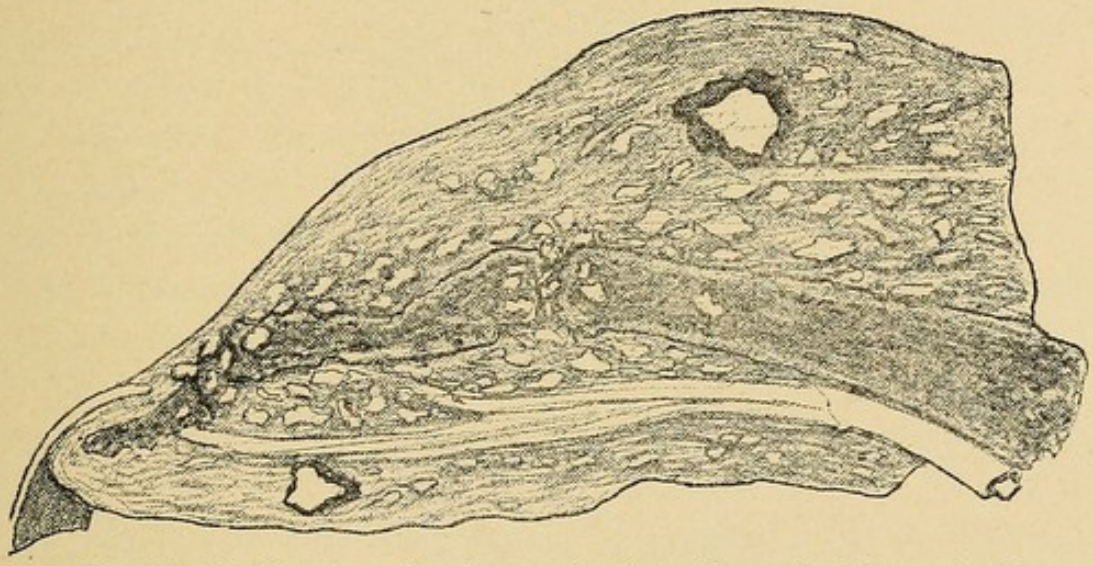


FIG. 81.—Section through a finger extensively disorganized by gout. The inter-phalangeal joints and the cartilages have been destroyed. Collections of urate of soda (tophi) are present in the ends of the bones, in the periarticular and subcutaneous tissues. (498 A.)

infection, which in old people may lead to extensive gangrene. The general disturbance of nutrition in gout is of great import to the surgeon. The hereditary gouty tendency may show itself even in young persons. The close relationship that may exist between gout and osteo-arthritis has already been pointed out. Gouty inflammation of the testis, urethra, and other organs will be mentioned in connection with these parts.

The widespread destruction that may be caused by gout is well shown in the specimen shown in Fig. 81, which represents a finger removed by Lunn from an old man on account of pain.

[1] See a paper by Berkart on "The Gouty Paroxysm," *British Medical Journal*, vol. i., 1895.

Charcot's Disease. In the course of the disease known as "Locomotor Ataxy" or "Tabes Dorsalis," the symptoms of which depend upon chronic inflammation of the postero-external columns of the spinal cord and the neighbouring parts, including the posterior nerve-roots, a joint affection of peculiar characters sometimes occurs. It begins with a sudden painless effusion of

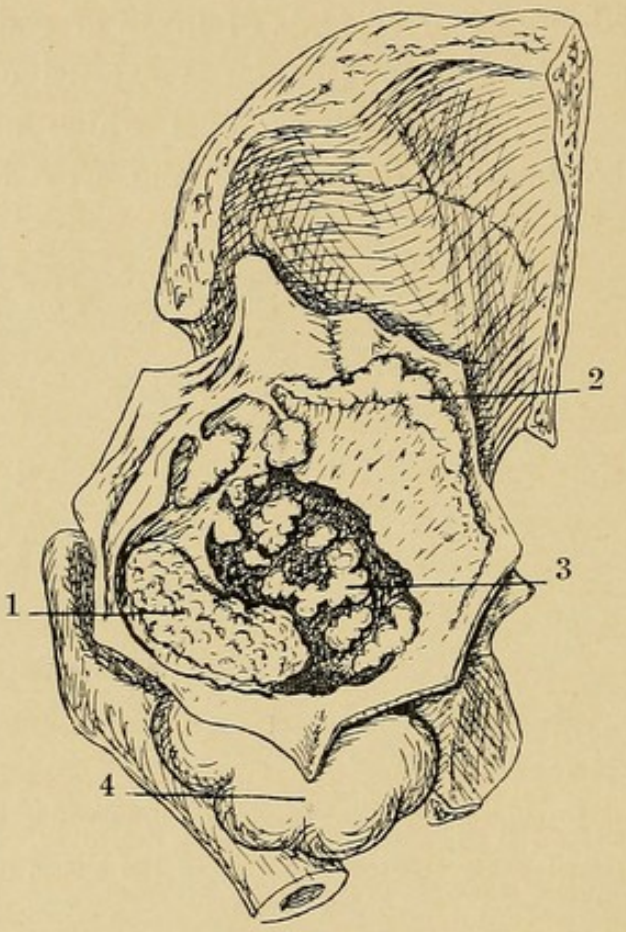


FIG. 82.—A hip-joint opened to show changes due to Charcot's disease. 1, remains of the neck of the femur, the head of which has been absorbed; 2, lip of new-formed bone on the dorsum ilii; 3, fibro-cartilaginous bodies formed from hyperplasia of the synovial membrane; 4, distended psoas bursa. From a man aged fifty who had had locomotor ataxy for fifteen years. (462 A.)

a large quantity of fluid into the synovial cavity. After a time this fluid may be absorbed. On opening such a joint the articular ends of the bones are found to have been extensively absorbed by rarefying ostitis, and the cartilages to have been for the most part removed. These and other changes are represented in Fig. 82.

It will be seen, on comparing the changes in this hip joint with those described above under "Osteo-arthritis," that some of the

features of the latter disease are present in Charcot's disease. This has given rise to the suggestion that the disease may be osteo-arthritis modified by the disease of the posterior spinal nerve-roots. However this may be, the alterations in both are of an inflammatory nature. In some cases of Charcot's disease the part of the bones adjoining the articular surface becomes greatly thickened.

Tubercular Arthritis. Now and again the infection leads to the rapid formation of tubercles in every part of the synovial membrane, accompanied by the effusion of much turbid serum and consequent rapid distension of the synovial sac. From such a case the portion of synovial membrane in the illustration given on p. 80 was taken because it affords a good example of tubercles unobscured by granulation tissue.

The more common form of tubercular arthritis is that in which abundant granulation tissue forms around the tubercles from the synovial membrane and grows over the articular cartilages from the edges, and erodes them by first causing in them hyperplasia, which loosens the texture of the cartilage and enables the granulation tissue to replace it and attack the bone. It is this form of tubercular disease which gives the appearance known as "white swelling" to joints, such as the knee, elbow, ankle, etc., or in synarthrodial joints like the sacro-iliac. The joint is swollen and pale; to the touch it has a doughy feeling. From the masses of granulation tissue in the joint this form of disease was formerly termed "fungous arthritis." Very frequently the infection of a joint is secondary to that of the bones which enter into its formation.

The effects of tubercular arthritis. When the process is rapid the tubercular granulations undergo caseous necrosis, and the infection may spread to the ligaments and skin. When the latter ulcerates sinuses form, leading into the joint and giving exit to tubercular pus.

Softening of the ligaments about a tubercular joint allows of gradual displacement of the bones (*pathological dislocation*). This is the more pronounced when the articular surfaces of the bones are extensively eroded. In a few instances tubercle in a joint has assumed the form of a circumscribed forma-

tion of tubercle on one part of the synovial membrane. But tubercular arthritis is due to extension of infection from the bones much more commonly than it is primarily located in the synovial membrane. Thus in the case of hip-joint disease, from which the accompanying illustration, Fig. 83, was taken, the disease evidently began in the neck of the femur near the epiphyseal line.

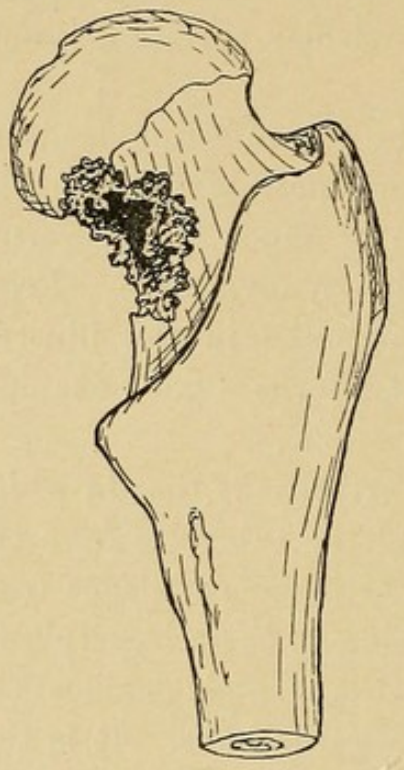


Fig. 83.—The head and neck of a femur from a case of hip-joint disease. There is a tubercular cavity at the posterior part of the neck of the bone.

Treatment. In the elbow and shoulder joints excision gives better results than the same operation in the hip or knee. In the two latter situations rest and extension in such a position as to prevent deformity is to be tried first. If the destructive process advances rapidly in spite of rest excision may be required. In cases such as the one referred to above, in which primary tuberculosis of the synovial membrane of the knee occurred, the membrane may be at once removed by erasion (*arthreotomy*). Each case must be studied individually. Every care should be taken to prevent the addition of pyogenic to tubercular infection, for suppuration from tubercular joints that have been opened is one of the common sources of amyloid disease and death.

An important secondary effect of all forms of serious joint inflammation is wasting of the muscles, and later atrophy of the bones of the limb. Disuse and reflex nerve-influence account for this phenomenon which is of the greatest importance in diagnosis.

Syphilitic Arthritis. In secondary syphilis a transient synovitis of large joints is sometimes observed, but its significance is not great. The important syphilitic disease of joints is that which occurs in the late period of the disease. As is the case with tubercle it may begin primarily in the synovial membrane,

or it may extend into the joint from gummatous inflammation of the ends of the bones. The former is the more common in syphilis. The synovial membrane becomes thickened and sometimes a nodular induration of the membrane can be felt during life. Unless the disease is checked by proper rest and iodide of potassium, the articular surfaces of the bones may be extensively destroyed.

Ankylosis.

Ankylosis (Gr. *ankylos* = crooked). From the frequent association of deformity with adhesion of articular surfaces one to the other this term has come to signify the fixation of a joint from the formation of fibrous or bony adhesions between different parts of the articular surfaces. Joints fixed in other ways, as by changes outside the joint, or by deformity of the articular surfaces, may be said to be subject to *virtual ankylosis*. Adhesions may form in joints from various causes, many of which have been already mentioned. In certain operations, as in excision of the knee, or again in arthrodesis of the ankle, the surgeon purposely produces a bony ankylosis. Adhesions that form after dislocations and other injuries have been mentioned. Continued or often repeated pressure may cause ankylosis as in the joints of the cervical vertebræ in dock-porters who carry heavy weights on the head. Hæmophilic extravasation into joints often leads to fibrous ankylosis. Pyæmic, chronic rheumatic, and other destructive diseases result in fibrous or osseous ankylosis. In tubercular disease a natural cure signifies a destruction of the tubercle bacilli by phagocytes of granulation tissue and the subsequent organization of the granulations into fibrous or osseous tissue. When repair takes place without the formation of sinuses, fibrous ankylosis usually occurs. Healthy joints may be fixed for a long time after fractures without real ankylosis taking place, though the joint may be fixed by the contraction of muscles around the joint. In the case of an ankle joint that had been fixed for four years on account of a compound fracture of the bones of the leg, in which ossification of the callus failed, I found the cartilages of the ankle and

tarsal joints thinned and perforated by granulations arising from the bone beneath the cartilages. Thus there was the beginning of ankylosis, but in this case the limb had been œdematous from obstruction in the veins in addition to the joints having been fixed.

Virtual Ankylosis is most commonly caused by the contraction of muscles about injured or diseased joints. It also occurs in Charcot's disease, a few cases of osteo-arthritis, in cases of scarring from extensive burns, and in cancerous growths, etc., in the neighbourhood of joints.

Diseases of Bursæ.

The morbid conditions of bursæ resemble those of the synovial membranes of joints, only a few special features require notice.

Acute Teno-synovitis from strain is a very common affection, especially in the tendon sheaths about the wrist. Effusion leads to swelling and proliferation of the cells lining the synovial membrane, causing a creaking friction during the action of the muscle. Fibrinous coagula ("melon-seed bodies") may form as in synovitis of joints, see p. 202.

Chronic Teno-synovitis is known as "compound ganglion." When it affects the large sheathing bursæ of the palmar tendons it gives rise to a characteristic deformity. **Housemaid's knee** is the result of chronic inflammation in the pre-patellar bursa. In the earlier stage the sac is simply distended; later, great fibrous thickening may occur in the wall of the bursa. Melon-seed bodies are frequently present.

Simple Ganglion, which forms a localized swelling in the vicinity of a tendon-sheath, also appears to arise in a chronic teno-synovitis restricted to a limited portion of the tendon-sheath; for sometimes on dissecting such a swelling it is found that the tendon lies exposed in the sac which is filled with thin fluid synovia. Usually, however, the cavity is found to be filled with jelly-like synovia, and it is completely shut off from the tendon sheath, though in close relation with it. Now and again a narrow communication passes from the ganglion to the interior of the synovial sheath of the tendon.

These facts point to the origin of a simple ganglion in a hernial protrusion of the synovial membrane through a weak part of the tendon-sheath.

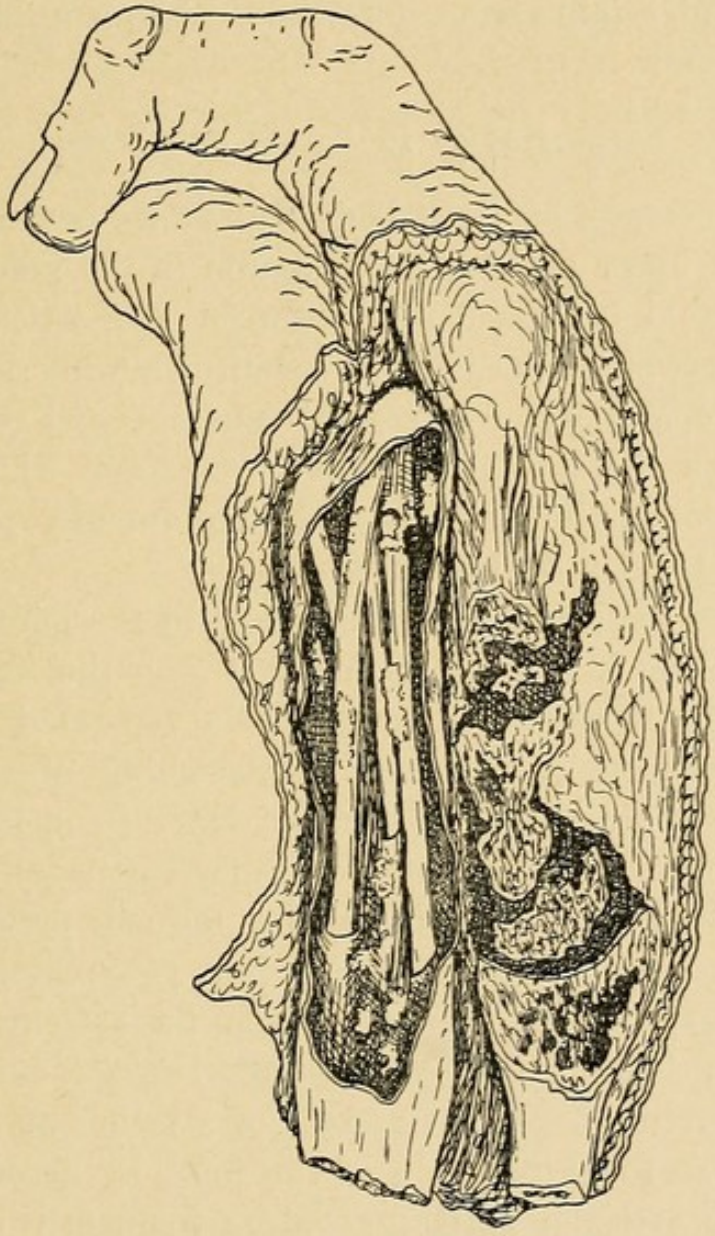


FIG. 84.—A section of a hand. There is extensive destruction of the carpal and metacarpal bones, and of the lower end of the radius. The sheath of the flexor tendons is much dilated, and its synovial lining presents patches of caseating tubercular tissue. In this case no direct connection between the distended bursa and the diseased bones could be made out.

Primary Suppuration occurs in bursæ as in joints. It is especially common as a sequel to traumatic inflammation in the bursa over the olecranon process. Often, especially in children, it arises without any previous injury.

Tubercular Inflammation of Bursæ is usually a complication,

secondary to tuberculosis of bones, especially those of the carpus, as shown in Fig. 84.

Syphilitic Bursitis is sometimes observed in the late period of the disease. Many of the larger bursæ may become the seat of nodular infiltration, forming hard tumour-like swellings.

Loose Bodies in Joints and Bursæ.

The importance of loose bodies in joints is twofold. They serve to set up or prolong inflammation in the joint, and if one of them should become locked between the articular surfaces during the action of the joint, it constitutes a fulcrum by means of which separation of the joint surfaces and stretching of ligaments on the side of the joint away from which action is taking place forcibly occurs, causing intense pain at the moment and inflammation subsequently.

Such loose bodies may be either free in the joint or attached to the synovial membrane at one part. In the knee joint one of the semilunar cartilages may become separated or loosened at its attachment by rupture or stretching of the coronary ligament to the capsular ligament. More frequently in osteo-arthritis, one or other of the semilunar cartilages, softened by chronic inflammation, yields to the normal pressure of the articular surfaces becoming divided into two parts, one or both of which may become loosened from its attachment and so constitute a loose body. *Hyperplastic synovial villi* have been already referred to. If their stalk of attachment is long they may give rise to acute strain of the joint by becoming nipped between the articular surfaces. More frequently they become completely detached forming free bodies in the joint.

Other examples of loose and free bodies in joints are:

Foreign bodies, such as portions of needles, may enter the cavity of a joint. Portions of *articular cartilage* with more or less of the subjacent bone may be separated by an injury such as a fall. They may either become free at the time or be slowly detached afterwards. Firm *blood-clots* which become rounded by the movements of the joints; firm fibrinous coagula rounded by the movements of the joint constituting *melon-seed*

bodies. A specially large form of these fibrinous bodies is met with in a condition known as "*synovitis fibrinosa.*"

A small lipoma in the sub-synovial tissue may project into the joint, and by stretching the synovial membrane beyond it acquire a pedicle and constitute a loose body. A more widespread deposit of fat in the synovial villi is observed in some cases of osteo-arthritis and is known as "arborescent lipoma." Similarly a small *fibrous growth* of some of the articular tissues may form a loose, or by detachment, a free, body in a joint.

CHAPTER XI.

DISEASES OF MUSCLE AND TENDON.

In the section on "Nutrition" it has already been pointed out that the effect of voluntary exercise is to improve the nutrition of the muscles, but if exercise is pushed to fatigue point the opposite effect—wasting of muscle—is produced.

The wasting of muscles observed about ankylosed joints in which active disease has ceased is to be explained as *atrophy from disuse.* Again, in severe congenital club-foot which is independent of any change in the spinal cord, the muscles of the calf and foot waste owing to impaired movement of the ankle and other joints, and as soon as the deformity is corrected the improvement in the circulation of the limb and the nutrition of the muscles is very marked.

The dependence of striped muscles on their connection with the ganglion cells of the spinal cord and medulla has also been mentioned. In surgery the effects of destruction of spinal ganglion-cells of the anterior cornua in infantile paralysis (*anterior polio-myelitis*) are encountered chiefly as deformities, due to the wasting of muscles. The degenerated muscles yield to the tonic contraction of the opposing active fibres, and so deformity arises.

After some injuries to nerve-trunks, especially where a large nerve like the median is involved in the callus of a fracture, the degeneration of the corresponding muscles is accompanied by a cicatricial shrinkage so that the resulting deformity is

produced by the contraction of the degenerated muscles and not by their elongation as in infantile paralysis.

In some forms of muscular degeneration no dependence upon nerve-lesions can be traced. Such conditions may be regarded as *primary myopathies*. Of these, *pseudo-hypertrophic muscular paralysis*, which often appears in several children, usually boys, of the same family, is one of the most striking. In this condition an increasing symmetrical paralysis of the muscles accompanied in most cases by an increase of size in the limbs from excessive deposit of fat between the muscular fibres is observed.

Myositis or Inflammation of Muscles. Pyæmic abscesses are not uncommon in muscle. To this category belong the inflammatory foci sometimes observed in typhoid fever, and the inflammation of muscles, tendons, and fasciæ in *gonorrhœal rheumatism*. In ordinary *acute rheumatism* a serous myositis may be observed. *Tubercular myositis* is usually secondary to tuberculosis of bone as in ordinary psoas abscess. Sometimes a primary tuberculosis of muscle is observed. *Syphilitic gummata*, sometimes of large size, and fibrosis, resulting from syphilitic infiltration are sometimes observed. The lesions of glanders and actino-mycosis have been met with in muscle, and the painful inflammation of muscles which is observed in *Trichinosis* is to be remembered.

Myositis Ossificans. The formation of bone in the substance of muscles is sometimes due to repeated slight injuries as in the *drill bone*, which forms in the deltoid of infantry soldiers from the impact of a heavy rifle against the shoulder, and in the *rider's bone* which forms in the adductors of the thigh. These formations consist of true bone formed from the fibrous tissue of the muscle.

Apart from these conditions there is a strange disease which shows itself by the formation of painful inflammatory swellings in various muscles, tendons and fasciæ in young persons. The swellings subside and the tissue involved undergoes ossification. The disease tends to be progressive with intervals during a number of years. The bony plates, spicules, etc., are first observed in the muscles of the thorax. Finally, almost every

muscle of the body may become involved with the result that the whole body becomes rigid.

New Growths of Muscle. Primary growths are not common. Four cases that have come to my notice have been instances of alveolar sarcoma. Fibroma, myxoma, and chondroma are all described. I have seen one instance of angioma (*nævus*) of muscle.

CHAPTER XII.

DISEASES OF NERVES.

THE morbid conditions of the nervous organs are extremely numerous; they admit subdivision into affections of the central axis and peripheral nerves. Only a brief notice of the chief conditions of surgical interest can be given: the peripheral nerves will be dealt with here, and the affections of the brain and cord later in the book.

Neuritis of *traumatic* origin commonly results from the bruising and crushing of nerves as in fractures and other injuries. Slight injury is completely recovered from, but in the more severe forms nerve-degeneration and its effects are seen.

The average clinical course of events when a nerve is involved in callus is usually as follows. (1) A period of hyperæsthesia; (2) a period of muscular paralysis; (3) a period of anæsthesia with "glossy skin," etc.; (4) a final period in which sensation is recovered whilst deformity from the atrophic shrinking of degenerated muscle occurs.

Neuritis from *lead*, *alcohol*, or the poisons of *diphtheria*, *influenza*, etc., lead to pain, weakness and sometimes wasting of muscles.

Syphilitic and Tubercular Neuritis is usually due to implication of the nerve-roots at their origin from the brain or spinal cord.

Leprous Neuritis, due to the growth of the lepra bacillus in the nerves, sets up a chronic inflammatory process which leads to proliferation of cells and destruction of nerve-fibres. Spindle-

shaped thickenings form on the nerve-trunks. The secondary effects on the skin afford an excellent example of the results of irritative lesions of mixed nerves.

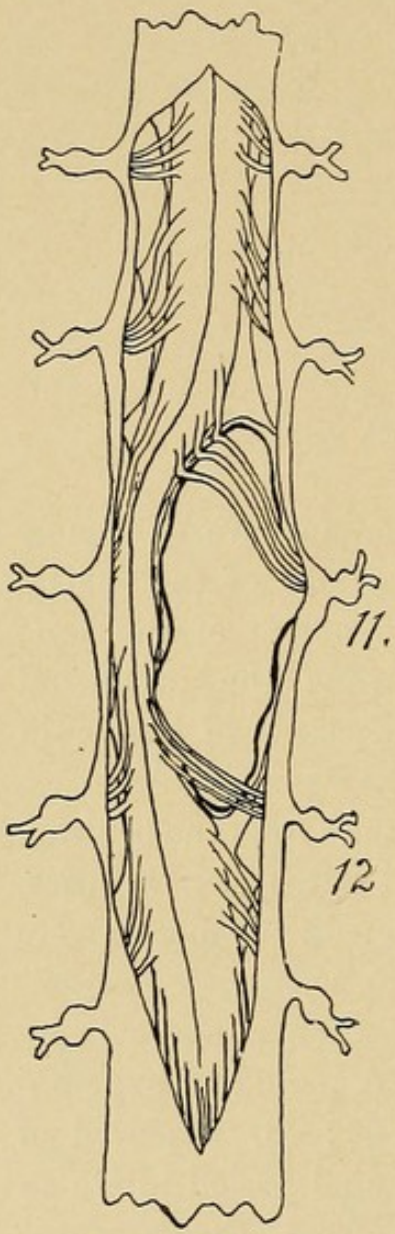


FIG. 85.—The lower part of a spinal cord in the dura mater; the latter is opened from behind to show an oval tumour, over which the posterior roots of the eleventh and twelfth right dorsal nerves are stretched. The cord is displaced to the left, and somewhat compressed. From a woman aged twenty-eight whose first symptom was pain in the back which was paroxysmal and so severe that it caused vomiting. There was continuous pain radiating from the right of the third and fourth lumbar spines, and passing along the twelfth rib to the anterior superior spine. There was ankle-clonus on the right side.

Neuralgic pains and hyperæsthesia are followed by the rapid formation of bullæ on the extremity corresponding to the affected nerve. Fresh bullæ appear as the older ones undergo retrogression. The skin of the area becomes first red, tense, and glossy. The redness becomes replaced by whiteness as cicatrization proceeds. Hyperæsthesia is replaced by anæsthesia. The muscles atrophy and deformities arise. Finally, interstitial absorption of the bones, ankylosis of joints, and the formation of perforating ulcers cause still farther deformity and mutilation.

The anatomical conditions that underlie the severe *neuralgias* that frequently demand surgical interference as in nerve-stretching, and in excision of nerves or ganglia are but little understood. Gout and malaria seem to have a part in the production of the conditions in some cases.

New Growths of Nerves. Neuromata. With the exception of the congenital ganglionic neuromata which are sometimes found in the suprarenal bodies, and the bulbous enlargements which form on the proximal ends of divided nerves, neuromata are fibrous, fibro-myxomatous, and fibro-myxosarcomatous tumours which arise in the supporting connective tissue of nerves. They affect (1) the nerve-

roots; (2) the large mixed nerve-trunks, and (3) the terminal branches of the nerves.

An important example of a fibro-myxoma affecting nerve-roots is shown in Fig. 85.

Similar spindle-shaped growths, such as the one represented diagrammatically in Fig. 86, affect nerve-trunks. They are usually single and tend to a sarcomatous structure. The growths found on terminal branches of nerve are most frequently multiple, as shown diagrammatically in Fig. 87.

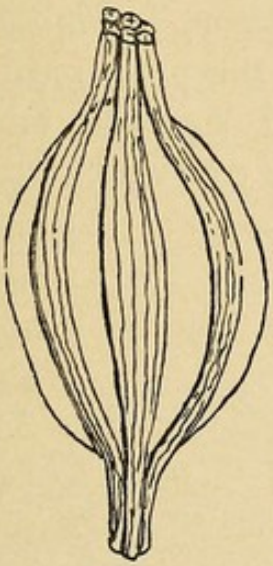


Fig. 86.

Fig. 87.

Fig. 86.—Diagram of a single neuroma of a nerve-trunk, the fasciculi of which are stretched over the tumour.

Fig. 87.—Diagram of multiple neuromata of the terminal branches of a nerve.

The multiplicity of fibrous growths on terminal nerve-plexuses is apt to be so great that they form a continuous tortuous plexus of contorted cords. This condition is termed a "plexiform neuroma," and clinically is recognizable by the presence of puffy ill-defined nodose growths. From such a condition it is but a step to the condition known as neuromatous elephantiasis, and to molluscum fibrosum (p. 104).

CHAPTER XIII.

DISEASES OF ARTERIES AND ANEURYSM.

To the surgeon the important diseases of arteries are such as lead (*a*) to clotting of blood within them, *i.e. thrombosis*; (*b*) to yielding of the wall of the vessel to the pressure of the blood, *i.e.* the formation of *aneurysm*; and such as interfere with the successful ligation of an artery, *e.g.* extensive *calcification of the coats.*

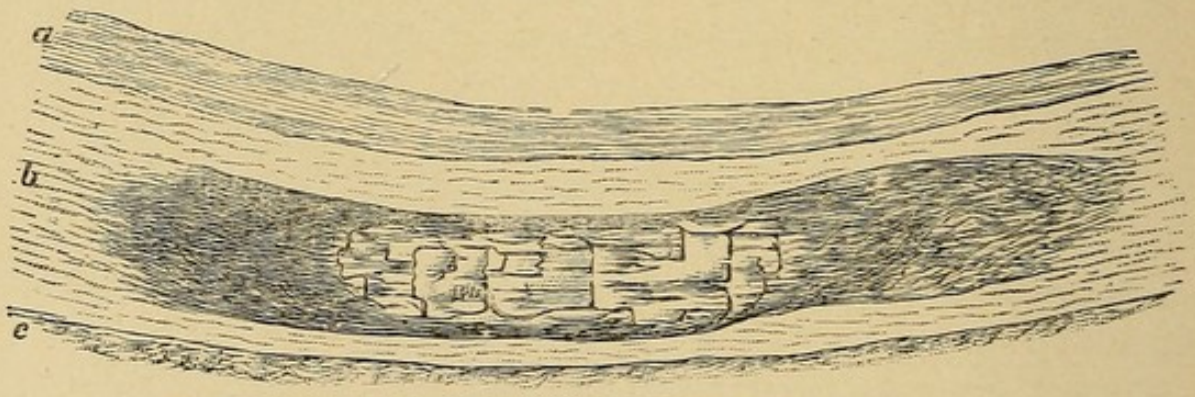


FIG. 88.—Calcareous infiltration of the middle coat of an artery. *a*, inner; *b*, middle; *c*, outer coat. ×22. Coats's *Pathology*.

Primary Calcareous Degeneration of the middle coat of an artery is an affection which may occur in otherwise healthy arteries. The vessel becomes brittle and rigid. When removed from the body it is seen to present calcareous rings which follow the course of the fibres of the middle coat. On examination under the microscope an artery at the early period of the disease shows the process to have begun in the middle coat (see Fig. 88).

The deposit of granules begins in the interior of the muscular fibres. When the disease is advanced the elasticity of the artery

is lost. This predisposes to the occurrence of thrombosis, and if this occurs in a large artery, say the femoral, gangrene is likely to occur. Indeed, it is usual to find calcareous degeneration (often combined with atheroma) in the arteries of limbs amputated for senile gangrene. Calcareous arteries are unsatisfactory to tie. The ligature may be found to cut through them several times before a part of the vessel sufficiently sound to hold a ligature is found.

Primary Fatty Degeneration of an Artery. In persons who have died from anæmia, or from infective or toxic diseases it is not uncommon to see small opaque specks in the intima of the arteries. They are quite superficial, slightly raised, and easily scraped off. They are due to fatty degeneration of the endothelial cells and subjacent subendothelial tissue. Similar changes may occur in the capillaries, and would account for the capillary hæmorrhages seen in purpura, etc.

Suppurative Arteritis. After ligature of an artery in a large wound, in which suppuration occurs, the part of the artery on either side of the ligature is liable to slough, and if at the same time the clot has become softened by suppuration the soft part of the artery is liable to give way, leading to *secondary hæmorrhage.* When this happens it is usually within the first fortnight, but it may be later. If the artery is tied in its continuity the hæmorrhage may come from an opening in the artery below the ligature, the collateral circulation having been established, and so the blood-pressure restored in the part of the artery below the ligature. Short of sloughing the artery wall may be softened by the inflammation, and, yielding to the blood-pressure, constitute an aneurysm. After ligature of very large arteries, such as the innominate, there may be secondary hæmorrhage apart from suppuration. Ballance and Edmunds attribute this to the division of the inner and middle coats that happens in the ordinary way of applying a ligature. They recommend the knot referred to on p. 33, applied so as to occlude the artery without dividing its coats.

Extensive calcification of the wall of an artery by diminishing its vitality may cause it to slough after artificial occlusion.

Suppuration in the neighbourhood of an artery may cause

perforation of its coats and hæmorrhage, especially if it is exposed to pressure of a drainage tube, or a portion of necrosed bone.

Acute and Subacute Arteritis are most commonly due to the lodgment of particles of fibrin displaced from the valves of the heart in endocarditis. If the endocarditis is rapid (*ulcerative endocarditis*) any such emboli which may lodge in the peripheral

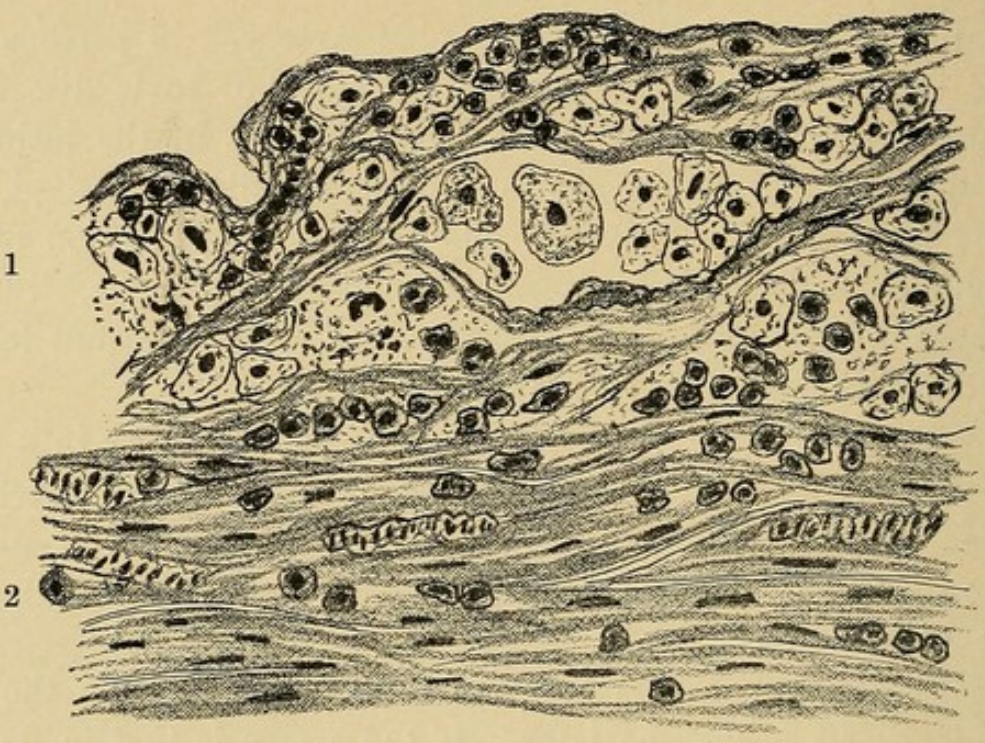


FIG. 89.—Subacute Arteritis. A section through a branch of a coronary artery near a commencing aneurysm. The intima, 1, is thickened, many of its cells are enlarged, and are undergoing fatty degeneration. Numerous leucocytes are present in the intima and in the inner part of the middle coat, 2. From a girl, aged nine years, who had recovered from a slight attack of rheumatic endocarditis, and who had many aneurysms on various minor arteries—one aneurysm ruptured in the right armpit, another in the right gluteus maximus, and a third into the pericardium—the last named was the cause of death. The leucocytes come from the branches of the vasa vasorum which enter the outer part of the middle coat. × 250. (664 A.)

arteries will carry with them the pyococci which cause the endocarditis, and set up rapid inflammation and softening of the wall of the artery in which they lie. This frequently leads to the formation of aneurysm (*embolic aneurysm*).

In some cases the endocarditis is less rapid, and may have disappeared as far as can be ascertained by clinical examination. Yet a subacute inflammatory process may go on in the walls of the arteries to which micrococci have been carried during the continuation of the cardiac disease. This subacute arteritis may

also result in the formation of aneurysms which are often very numerous, as in the case from which the artery shown in Fig. 89 was taken.

In another similar case examined by the author there were over twenty small aneurysms in the branches of the coronary arteries, and numerous ones in the kidneys and elsewhere. Death was immediately due to rupture of an aneurysm in the kidney. This form of arteritis is probably caused by pyococci.

Chronic Endarteritis—Syphilitic Endarteritis.

Chronic Endarteritis or Atheroma is one of the most familiar and at the same time one of the most important of pathological

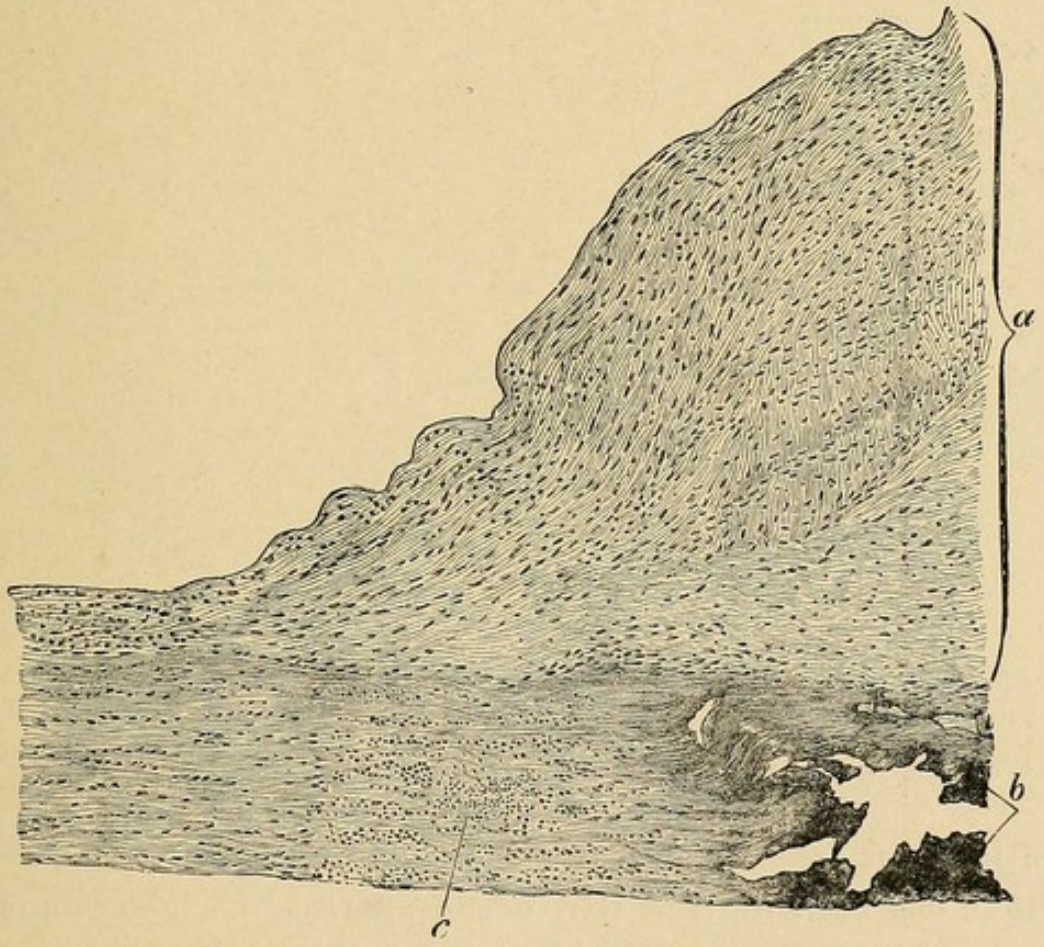


FIG. 90.—Atheroma of aorta. *a*, Thickened intima; *b*, deposit of lime in middle coat; *c*, cellular infiltration of middle coat. (Alum cochineal.) × 80. Weichselbaum.

changes. In the earlier stage it is seen to form raised pink or grey patches in the interior of the arteries. Microscopical

examination shows a marked increase in the number of the connective-tissue corpuscles of the subendothelial layer. A little later the thickenings become fibrous (see Fig. 90) or opaque and yellow in colour from the fatty degeneration of the proliferated connective-tissue cells which have become enlarged like those shown in Fig. 89 in subacute arteritis. The fatty cells may break

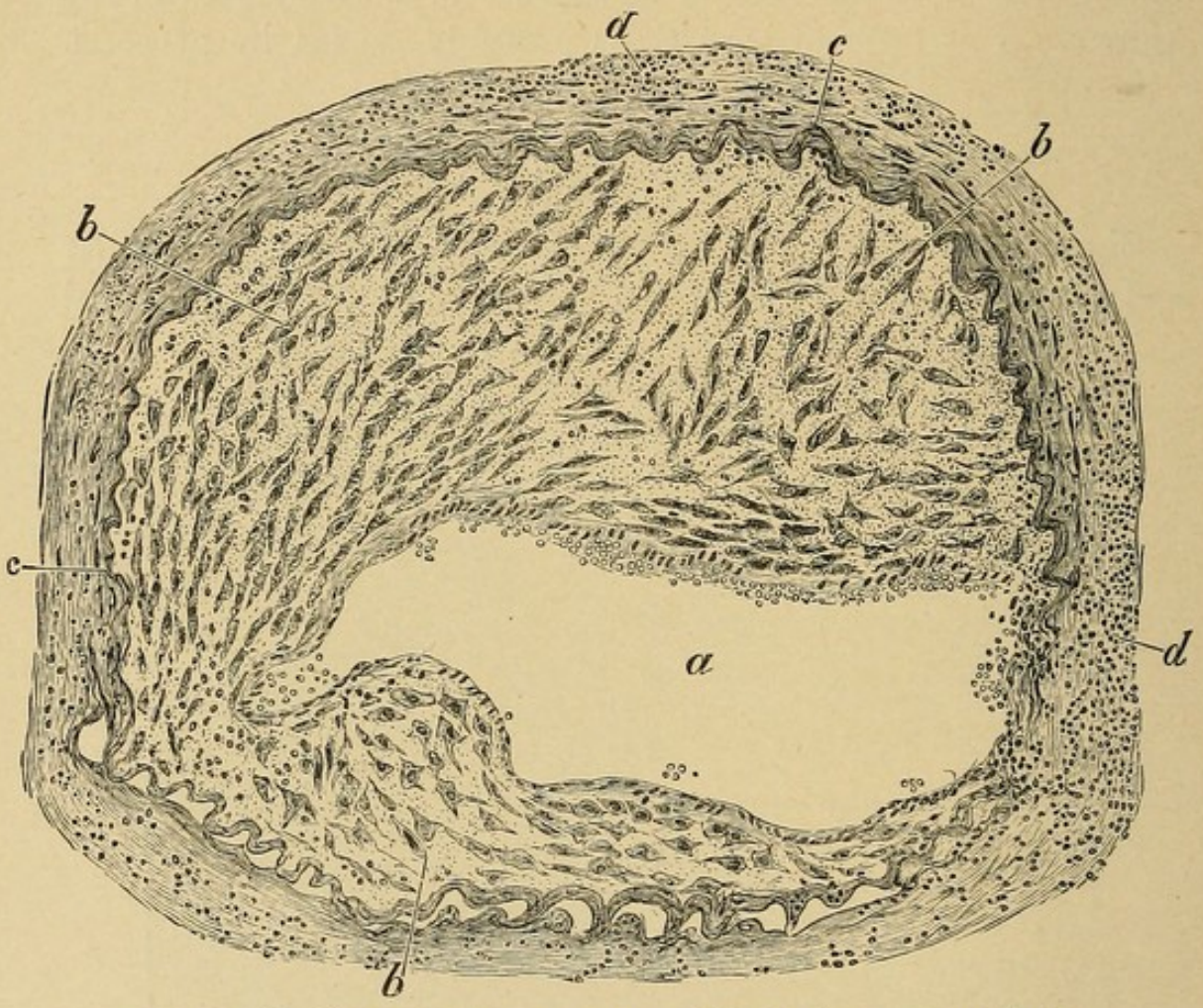


FIG. 91.—Syphilitic arteritis in a gumma of the brain. *a*, Eccentrically narrowed lumen of vessel; *b*, irregularly thickened intima, consisting of young connective tissue; *c*, internal elastic lamina; *d*, middle coat, infiltrated with round cells. (Hæmatoxylin and eosin.) Weichselbaum, *Pathological Histology*. × 160.

down into soft caseous matter, whence the common name of the affection has been derived (Gr. *athere* = porridge). The broken-down matter may either calcify forming thin, brittle *calcareous plates* in the lining of the artery, or it may escape into the blood leaving an *atheromatous ulcer*. In the floor of such an ulcer the middle coat of the artery is exposed to the action of the blood-pressure, and may gradually stretch and atrophy, forming a

localized dilatation or *aneurysm* of the artery. In some instances the blood is forced between the layers of the middle coat, forming a *dissecting aneurysm.* An artery that is lined by calcareous material is more brittle than normal, and any strain on the artery is likely to cause a rupture of the inner coat and subsequent formation of an aneurysm. Again the endothelial cells which cover degenerated atheromatous surfaces perish, leaving rough surfaces on which blood clot is likely to be deposited, leading to thrombosis of the artery.

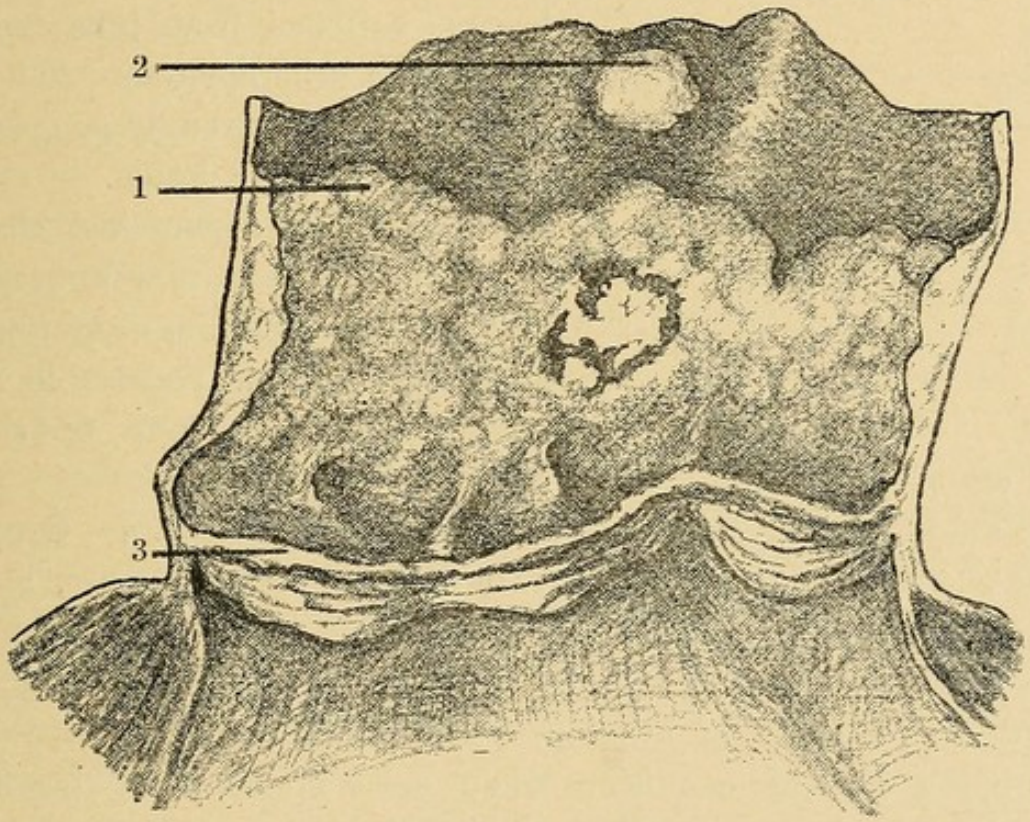


FIG. 92.—Atheroma of the aorta—probably syphilitic. 1, Advancing border of atheromatous patch ; 2, outlying gumma-like nodule ; 3, deformed aortic valves. In the middle of the larger patch is a calcified plate surrounded by an ulcer.

Syphilitic Arteritis. In the neighbourhood of gummata arteries are often found to have their inner coats greatly thickened, and on microscopic examination the thickening is found, like atheroma, to be due to a great increase in the number of the cells of the subendothelial layer (see Fig. 91).

Syphilitic thickening of the inner coat of small arteries may be so great that they are occluded, and in the case of the brain this commonly causes necrotic softening.

When arteries are taken from the immediate neighbourhood of an obviously syphilitic lesion there is no difficulty in coming to a conclusion as to the nature of any thickenings there may be in the arteries, but when there is a history of syphilis or many obvious syphilitic lesions are present in various parts of the body, it is often difficult to say whether atheromatous patches are of syphilitic origin or not. In Fig. 92 is depicted a not uncommon form of atheroma of the aorta. The chronic inflammation has caused thickening of the aortic valves, and also gradual yielding of their points of attachment which have become softened from inflammation. The atheromatous patches have characters strongly suggestive of a syphilitic lesion.

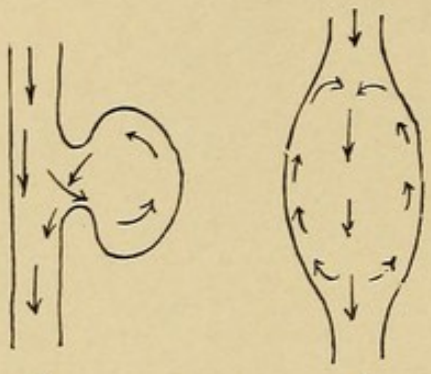


FIG. 93.—Diagram representing the two chief forms of aneurysm in the order of frequency of occurrence. 1, saccular; 2, fusiform. The arrows suggest the main currents, which however are in reality much more complicated. See Ballance and Edmunds, *loc cit.*

When it is remembered that syphilitic gummata may undergo caseation, calcification, ulceration, etc., it is, I think, reasonable to admit syphilis as one of the causes of atheroma.

Causes of Atheroma. Great variations of blood-pressure, and thus laborious occupations which entail them are by many surgeons regarded as a cause of atheroma. They are doubtless capable of keeping up the condition when once it has been begun. The high blood-pressure of renal disease is also to be regarded in the same light. Atheroma is of almost constant occurrence in Bright's disease. It is, however, to be remembered that it is associated with the presence of toxic materials in the blood. The abuse of alcohol would also appear to be a pregnant cause of atheroma. Here again there is the toxic product of the saccharomyces which may act directly on the arteries or indirectly by causing disease of the kidneys and liver. Gout, syphilis, rheumatism and other morbid conditions may also determine atheroma.

Aneurysm (Gr. *ana* = up or out, *eurys* = wide). An aneurysm is usually the effect of the blood-pressure acting on part of the

wall of an artery which has been weakened either by disease or injury. More commonly a patch which does not include the whole circumference gives way; this results in the formation of a pouch-like swelling or saccular aneurysm. If the whole circumference of the vessel for a certain distance the resulting swelling is spindle-shaped and is termed a "fusiform aneurysm."

A more wide-spread yielding of a section of an artery and of its main branches is termed a cirsoid (Gr. *kirsos* = varicose vein) aneurysm.

The production of aneurysm by injury has already been described (pp. 35, 36).

Even where atheroma is originally to blame, the immediate determining cause of the formation of an aneurysm is usually a strain which causes a rent of the inner coat.

The structure of the wall of an aneurysm varies in different cases. Where the whole thickness of the artery yields, all three coats of the artery are at first present though stretched out. If the aneurysm increases in size the original arterial wall becomes thinned out, and the sheath becomes adherent to the outer aspect of the swelling and incorporated with the walls or *sac* of the aneurysm. In course of time as dilatation proceeds the inner layers of the sac are absorbed whilst on the outer side, surrounding tissues are converted into fibrous tissue by inflammatory changes induced by pressure of the aneurysm, thus it happens that in the walls of large aneurysms nothing but fibrous tissue is to be found. When the structure of the artery wall has been altered by inflammatory changes the sac is composed of fibrous tissue from the first. The power of an aneurysm to cause absorption of tissue is nowhere shown so well as where it presses against a bone. First the intermittent pulsations are felt by the periosteum which becomes inflamed, and its fibrous tissue is thus converted into granulation tissue. As the aneurysm enlarges the effects of constant pressure tell on granulation tissue with the result that the latter is absorbed by new granulation tissue which is now formed in the bone. The latter in turn like the periosteum becomes gradually absorbed, forming part of the sac of the aneurysm, and like the rest of the sac gradually yields to pressure (see Fig. 94).

The Clinical Course of Aneurysm differs in the different forms of the disease. A fusiform aneurysm may remain with but slight change for a long time, then usually it becomes converted into a saccular aneurysm by its sac yielding more at one part than at another. Its course thenceforward is like that of the commoner and surgically more important variety. A general dilatation or fusiform aneurysm of the aorta may prove fatal from enlargement of the aortic orifice allowing of regurgitation into the left ventricle.

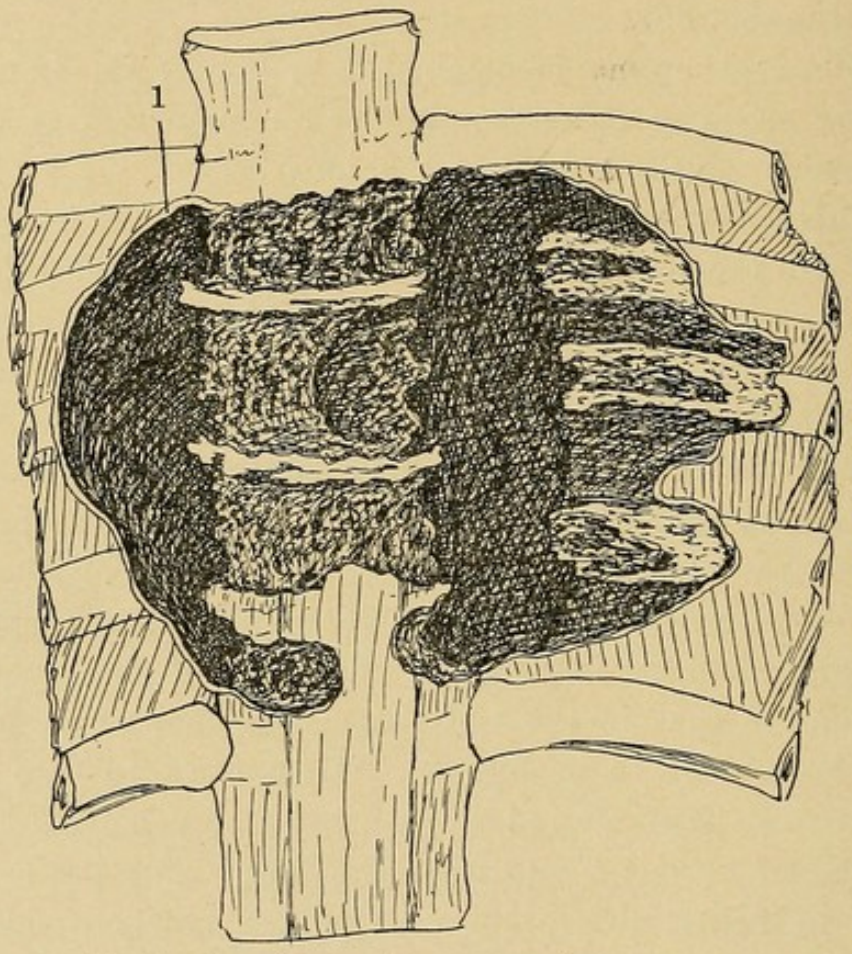


FIG. 94.—Six dorsal vertebræ with portions of the ribs, etc. Both ribs, vertebræ, and soft tissues have been extensively eroded by pressure of the aneurysm, part of the sac of which they constitute. At 1 the continuation of the fibrous part of the sac is seen. It is noteworthy that the non-vascular intervertebral cartilages have suffered less absorption than the bone.

Rupture of an Aneurysm. The manner in which an aneurysm causes absorption of tissues has been referred to. When the tumour reaches a surface, such as the skin, the mucous lining of the trachea or of a bronchus, or a cavity like the pericardium, the progressive absorption of tissue leads to rupture. When

the rupture occurs through a tough membrane like the skin or a thick mucous membrane the aperture is small and the bleeding likely to be rendered intermittent by the formation of clots which plug the orifice for a time to be dislodged by farther enlargement of the orifice. When rupture occurs into a serous cavity or into a joint the hæmorrhage is more rapid and the cavity quickly fills. Rupture may also occur into the soft tissues of a part from their yielding too rapidly to the pressure of the sac. If this occurs in the case of a popliteal aneurysm the pressure of the extravasated blood on the veins added to the already impeded arterial obstruction may result in gangrene.

Suppuration. The simple inflammatory changes which have been described above as usually occurring about an aneurysm may be transformed into suppuration if pyococci find their way to the part. By such suppuration the sac is likely to be softened or to be caused to slough with the result that rupture of the sac and death from hæmorrhage may occur, or the inflammation may be only sufficient to bring about clotting in the sac of the aneurysm, and so a spontaneous cure, see below.

The Formation of Clot in Aneurysms. Two conditions favourable to coagulation are present in a sacculated aneurysm; (1) the blood stream is slowed, (2) the internal endothelium of the artery is usually wanting. Thus it is that nearly all large aneurysms have more or less of the interior of the sac lined with blood-clot. Any clot that forms on the wall of the sac is deposited from moving blood, and hence resembles the clot that forms when blood is whipped with twigs out of the body; it is composed chiefly of fibrin and leucocytes, and is pale in colour. Successive layers of such clot formed one within another have a laminated arrangement because the rate of fibrin formation varies from time to time. This laminated clot formed during the circulation of the blood has been termed "active clot." If the circulation throughout an aneurysm should entirely cease for a sufficient length of time the whole of the blood it contains may undergo coagulation. In this case the clot contains the red corpuscles as well as fibrin, and has more the characters of ordinary post-mortem or "currant-jelly"

clot. In an aneurysm it has been called "passive." If the whole of the cavity of an aneurysm should be filled with either laminated or jelly-like clot, a *spontaneous cure* of the aneurysm is the result. The clot extends into the artery and occludes it for some way on each side of the aneurysm. The same result may ensue from the detachment of a portion of clot from an aneurysm and its lodgment in the artery below the aneurysm; in these circumstances, if there should be no arterial branch given off between the aneurysm and the point at which the detached clot has lodged, the whole of the blood from the point at which the last branch above the aneurysm to the point of occlusion below it undergoes coagulation.

Surgical Cure of an Aneurysm. The methods employed by the surgeon are usually designed to facilitate a natural cure of the disease. The presence of a dilatation in the course of an artery slows the current at the dilated part and also increases the resistance and hence diminishes the flow of blood through the artery to the parts beyond. The diminution of blood supply to the parts below an aneurysm is compensated for by the establishment of the *collateral circulation.* Branches given off from the artery above the obstruction dilate, and through their communications with other branches given off below the obstruction, convey to the main artery below the obstruction as much blood as may be required to make up the difference caused by the presence of the obstruction.

In some cases compression of the main artery above the aneurysm is sufficient to produce coagulation of the blood it contains. In other cases ligature of the main artery is required. The immediate effect of ligature of the main artery is the cessation of the pulsation in the aneurysm. If the collateral circulation is rapidly established the pulsation may return and again disappear, if under the altered conditions, *i.e.* with a slower blood stream, laminated clot is deposited till the sac is full. The dangers of a case of aneurysm are not over when the operation has apparently caused coagulation of the blood and cessation of pulsation. Secondary hæmorrhage at the seat of ligature, suppuration in the sac, and gangrene of the limb may occur. With antiseptic measures, in the case of arteries of the size of

the superficial femoral or smaller, secondary hæmorrhage may be practically set aside as extremely unlikely to occur. When the

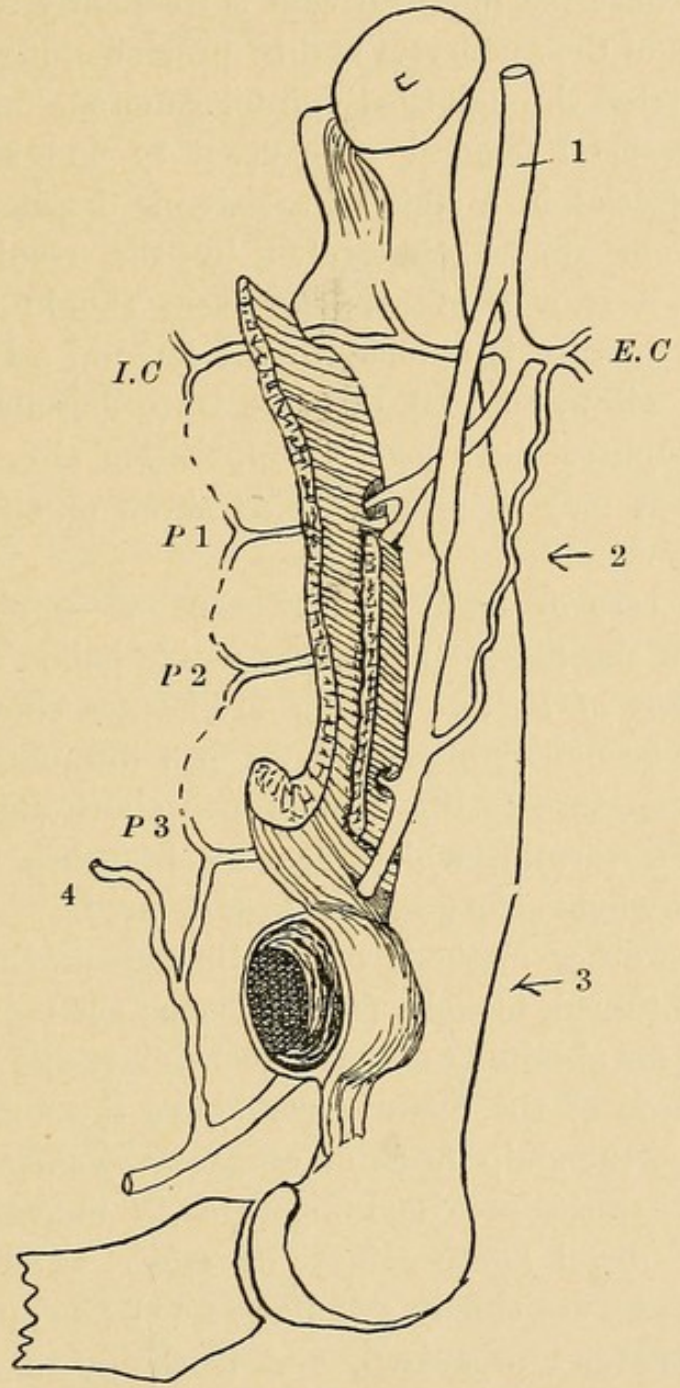


FIG. 95.—A dissection of a thigh showing an aneurysm on the upper part of the popliteal artery, and the results of ligation of the superficial femoral artery. 1, Common femoral; 2, points to a narrowed occluded part of the superficial femoral where a ligature had been applied two years previously; 3, points to the aneurysm on the uppermost part of the popliteal artery. The dark part was occupied by old dark clot which formed after ligature. The anterior part of the aneurysm contained laminated clot, and represents a recurrence of the condition. The collateral circulation is shown. *E.C*, External; *I.C*, internal circumflex; *P* 1, 2, 3, perforating branches of the profunda; 4, a branch which ran in the great sciatic nerve. (703.)

superficial femoral artery has been tied, if gangrene should supervene, it commences in the toes, and the extent to which it will

spread is determined by the amount of collateral circulation that has been established. The gangrene is usually of the moist variety, because the popliteal vein is frequently partially blocked by pressure of the aneurysm and by previous inflammation. In a limb amputated through the thigh for gangrene following ligature of the superficial femoral it is usual to find the femoral and popliteal arteries in a state of calcareous degeneration, and the popliteal vein almost obliterated by the results of previous thrombosis. If a popliteal aneurysm should have ruptured before the ligature is applied to the femoral artery gangrene is certain to follow, so that the rule is to amputate in a case of ruptured popliteal aneurysm. Suppuration after ligature is not common. It may occur as late as seven or eight weeks after the operation.

When a case of ligature has been successful the aneurysm may recur if the patient reassumes active habits too soon. This and *the effects of ligature* on the artery are shown in Fig. 95. The artery becomes obliterated by firm fibrous tissue from the seat of the ligature to the next branch above and below it. In the common carotid, which has no branches save the most minute, the whole artery becomes obliterated.

There are other methods of treating aneurysm besides those already mentioned, namely, (1) by placing the patient in the best conditions[1] for obtaining a spontaneous cure, and (2) by pressure on, or ligature of the main artery above the aneurysm. Small aneurysms of the scalp, forearm, etc., may be excised. In certain arteries in which proximal ligature is unavailable, *e.g.* the innominate, distal ligature may be tried. The changes in a cured aneurysm are chiefly as follows: (1) The outer part of the coagulum becomes organized, and if the aneurysm is one of moderate size it may in this way be wholly converted into scar tissue. When the clot is large the central part undergoes a granular disintegration and may become calcified. In the case from which the accompanying figure was taken the part of the clot formed after ligature two years previously was very dark in colour and horny in consistence.

[1] Rest in bed, diminution of liquids in the diet, and the administration of iodide of potassium are the most important of these.

CHAPTER XIV.

DISEASES OF VEINS. VARIX.

A VARIX is a dilatation of *a vein* beyond the normal.

Causes. Rapidly succeeding pregnancies frequently determine the formation of varicose veins in both lower extremities of women who had previously been free from any affection of the veins. This is especially observed in poor persons who are obliged to remain at work during the later months of pregnancy. The causation of the over-distension of the veins in such a case is obvious. The pregnant uterus pressing on the iliac veins obstructs the return of the blood to the heart, and an excessive quantity of blood accumulates in the veins of the lower limb. From one or two such temporary periods of distension the veins will probably recover in the intervals between the pregnancies, but if the latter follow each other with rapidity the dilatation becomes permanent, the valves of the veins cease to act when the dilatation has reached a certain degree, and the vessels become tortuous, because elongation accompanies dilatation. The tortuosity of a vein still farther impedes the flow of blood through it. Large sacculated dilatations recalling aneurysms may form at the bends of a vein, or where a superficial vein joins a deep one. In this instance it is seen that obstruction to the return of blood causes dilatation of the veins below it, and once established the dilatation tends to grow worse by reason of the loss of function in the valves, and of the tortuous course assumed by the varicose vessel. Later still, as will be farther discussed below, the vessel wall undergoes the familiar fibrotic changes which result from chronic venous congestion.

The causation of varicose veins will be found to be similar in other cases. Thus the impediment to the return of blood to the heart may consist in an extra long column of blood; so in persons with very long legs the internal saphena vein, which reaches from the groins to the ankle, may dilate in spite of its valves. Or again, in youths with a lax scrotum the testes may hang unusually low, and dilatation of the spermatic plexus (*varicocele*) may result. Dilatation of the prostatic plexus is common in elderly persons, and of the rectal veins (*hæmorrhoids*) in persons who have congestion of the liver from excessive eating and drinking, or who suffer from constipation, etc. Some persons seem to be predisposed to varicose veins from inherited weakness of the vessel wall. Chronic heart or lung disease by impeding the passage of blood through the heart causes a general tendency to over-distension of the veins, and the veins of the lower extremity, from being longest, and, in the erect position of body from having to work against the force of gravity, will feel the effects most. Though varix is visible only in the superficial veins it must not be forgotten that the deep veins may also be affected.

Histological Changes in Varicose Veins. It has already been mentioned that a limb, the veins of which are varicose, is in a state of chronic venous congestion and œdema. This applies to the walls of the vein itself equally with the other tissues. The effect of chronic venous congestion is hyperplasia of the connective-tissue elements of a part. Hence the fibrous tissue in the wall of the vein is increased. On opening such a vein the lining and valves are seen to be more opaque and thicker than normal. The wall of the vein is thicker than normal. In the early period this may be due to some hypertrophy of the muscular coat of the vein, but later the muscular fibres atrophy and the thickening is due to hyperplasia of fibrous tissue.

The Effects of Varices on the Surrounding Tissues. The subcutaneous tissue and skin immediately covering a large varix may undergo atrophy from pressure and becomes so thin that a slight injury is sufficient to rupture the vein. In some cases the skin, especially that over the veins, become pigmented from the lodgment in the tissues of hæmatoidin which remains after

small hæmorrhages in the neighbourhood. The skin in the vicinity of varicose veins, from its congested state, is prone to attacks of *eczema*, which is difficult to check, and which frequently leads to *ulceration*. Ulceration may also follow a scratch or other slight injury which opens a way for pyogenic organisms, which produce more serious effects in tissues of which the resistance is diminished by congestion and œdema than in normal conditions. Varicose ulcers in the working classes are certainly hard to heal, often, indeed, they increase to such an extent that amputation is resorted to. When, however, the patient can be kept at rest in bed, the ulcers heal readily under mild antiseptic applications.

Thrombosis and Phlebitis.

Clotting of blood within the heart, an artery, or a vein is termed thrombosis. The chief factors which lead to the clotting of blood are :

1. *Loss or alteration of the endothelial lining of the vessel wall*, from injury or inflammation. This acts by causing leucocytes to adhere to the vessel wall. Some of the leucocytes perish and liberate fibrin ferment, which, acting on the fibrinogen of the plasma, produces fibrin.

2. *Foreign bodies* in the lumen of a vessel, *e.g.* a ligature, which transfixes instead of surrounding the vessel. This acts in the same way as '1.'

3. *Retardation of the blood-stream.* The great frequency of thrombosis in varicose veins is chiefly due to this factor. It has been seen that in order to produce coagulation of blood in an aneurysm the stream is slowed by compressing or tying the artery above. This effect of slowness of the blood-stream is in part due to an alteration in the state of the endothelial cells, which are ill nourished when the blood that bathes them is not renewed with the usual rapidity. Slowness of the blood-stream is frequently due to weakness of the heart's action, as in phthisis and other wasting diseases.

4. *Toxic materials* or *parasites* in the blood-stream frequently determine thrombosis. Thus in gout excess of uric acid in the

blood acts as a provocative of coagulation. In pregnancy also there is excess of matters in the blood which tend to facilitate coagulation. After typhoid fever parasites, toxic materials in the blood, and weak heart action may all combine in producing thrombosis. In malaria the parasites actually plug the small vessels of the brain and spinal cord.

Phlebitis or Inflammation of a Vein has been mentioned as a cause of thrombosis. *Per contra* thrombosis may be said to be a cause of phlebitis, for the clot becomes organized by the formation of granulation tissue in the wall of the vessel to which it adheres; and since this process, though strictly one of repair, is accompanied by the clinical signs of inflammation, it is not distinguishable from phlebitis.

Other causes of phlebitis are: Injury such as bruising of a vein. More important is the extension of septic inflammation from without to the interior of a vein. Septic thrombosis of veins is a frequent precursor of pyæmic infection: of this, thrombosis of the lateral sinus forms a striking example. Tubercular infection also may attack the wall of a vein, and by ulcerating into its lumen cause a tubercular thrombosis, which may be the precursor of a general infection. Tertiary syphilis also is not infrequently a cause of phlebitis and thrombosis.

Later Changes in Thrombi. When the thrombus is aseptic organization proceeds, as in the formation of fibrous tissue in other parts, *e.g.* in the repair of tendon. One important feature is that during the shrinkage of the clot channels are often formed in it by which the lumen of the vein becomes partially restored (see p. 38). Indeed, on making a section through a large vein at a point where it had previously been occupied by clot it is usual to find several channels which perforate the scar-tissue left by organization of the thrombus. In many cases the clot almost entirely disappears and a slight thickening in the wall of the vessel is all that remains. If the clot is not organized throughout, the central part undergoes necrosis, becomes granular, and frequently is calcified, constituting a *phlebolith*. Phleboliths are commonest about the base of the bladder in varicosities of the prostatic plexus and in nævi in which thrombosis has occurred.

Embolism. Wherever there is recent thrombosis the possibility of part of the clot being detached, carried to the heart, and thence to the lungs must never be forgotten. One of the constantly-recurring tragedies of medical practice consists in the sudden death of a person previously quite well, save for a slightly painful swelling in a bunch of varicose veins in the leg. The medical man will probably have told the patient not to leave the bed on any account; this injunction is disobeyed with the tragic result just mentioned. What has happened? The fragment of clot detached, say, from the internal saphena vein, is borne to the right auricle. It has probably increased in size on its way to the heart by the addition of fresh layers of fibrin on its outer aspect. The clot passes from the right auricle to the right ventricle and thence to the pulmonary artery, in one of the divisions of which it lodges. Thus a greater or smaller segment of one of the lungs is suddenly deprived of circulating blood and hence of respiratory function. If the embolus is very large the patient experiences great pain and shock (*i.e.* reflex depression of the cardiac and respiratory centres) and sudden death results. If the embolus is smaller recovery may occur after a struggle against heart failure and dyspnœa. Then ensue signs of consolidation of the lung, followed after some days by the expectoration of blood, the result of infarction (see below).

Clinical Effects of Thrombosis. In small veins the difficulty of circulation caused by thrombosis is soon overcome by the blood finding its way by collateral veins, but when the main vein of the limb is thrombosed, as for instance the femoral, œdema of the limb is the result. The œdema generally begins about the ankle. If the block is permanent, so also will the œdema be, and with the œdema general thickening of the tissues of the limb due to fibrosis, weakness of muscles, heaviness and pain will be observed. One form of "white-leg," a condition which occurs generally two weeks or later after delivery of a child, is due to thrombosis of the femoral vein. After a long and dangerous illness under proper treatment the condition is usually recovered from, probably by the canalization of the thrombus mentioned above. The risk of pulmonary

embolism in such cases is very great. Such accidents have happened after operations on varicose veins, such as varicocele.

When a branch of the pulmonary artery is blocked, as has been stated above, all circulation ceases in the district supplied by that branch. This case is different from the blocking of an artery of the limb as by a ligature. In the latter case the collateral circulation is established because branches of the artery above the point of occlusion anastomose with other arterial branches below the same point. The branches of the pulmonary artery form only capillary anastomoses. There is a slight and *equal* pressure on all sides of area suddenly deprived of blood. From the pressure being equal on all sides there is no tendency for any flow of blood across the area, but only a slight regurgitant stream sets towards the plugged artery and ceases (*i.e.* stasis occurs) as soon as the vessels of the area have become completely charged with blood. The capillary walls give way allowing the blood to escape into the interstitial tissues and into the air vesicles. There the blood clots, giving rise to the signs of solidification of the lung. Later this clot disintegrates and is coughed up, escaping by the bronchi. The solid area or *infarct* in the lung does not undergo necrosis, because there is a second supply of blood to the tissues by way of the bronchial vessels.

Fat Embolism. In cases where persons have died soon after the fracture of a bone, globules of fat have been found filling many of the smaller branches of the pulmonary arteries. In some of these cases no other lesion has been found to explain why the person died, and it has been concluded that death was caused by the arrest of the pulmonary circulation owing to the obstruction offered to the passage of fat through the smaller pulmonary vessels. In order for sufficient fat to enter the blood to produce such a result the medulla of the fractured bone must be extensively crushed, or liquefied by inflammation in order to enable sufficient free liquid fat to be absorbed by the lymphatics, and so passed on to the blood-stream. Similarly the entry of air into a large vein near the root of the neck causes death by bubbles of air stopping up the pulmonary capillaries. This might be termed air embolism.

In organs that like the lung have a terminal circulation but no second blood-supply, such as the bronchial arteries in the lung, the whole of the infarct undergoes necrosis, and if it is aseptic it is gradually removed and replaced by scar-tissue. This occurs most frequently in the spleen and kidneys.

Septic Embolism. If pyogenic organisms are carried with the embolus then suppuration takes place as in ulcerative endocarditis. Indeed in any disease caused by micro-organisms which are distributed with the blood-stream the process may be rightly termed embolic, though the emboli are of microscopic size. In this way the abscesses of pyæmia may be said to have an embolic origin.

The brain also has a terminal circulation, and when one of its arteries becomes blocked the area deprived of blood undergoes necrosis and becomes soft; thus softening of the brain takes the place of infarction in other organs.

Though, as has been stated above, the collateral circulation in normal conditions is readily established in parts where arterial anastomoses are present, as is the case in the limbs, yet there are limits to the powers of adaptation. When the arteries are extensively calcified, either from primary calcification or from atheroma, their power of enlarging to compensate for the block of a main channel is greatly diminished. Thus in old persons an embolus that blocks a femoral artery usually causes gangrene of the foot. The collateral circulation, though it is able to supply a certain amount of the limb below the seat of embolism, is not sufficient to keep up circulation in the foot. Once begun the gangrene may extend considerably above the foot. It is usually of the dry variety, because the main vein of the limb is not affected.

Clots may form in the left side of the heart in either acute or chronic endocarditis. Of all kinds of chronic endocarditis the formation of clots is commonest in mitral stenosis. In this condition they form in the dilated left auricle.

CHAPTER XV.

DISEASES OF LYMPHATIC VESSELS AND GLANDS.

LYMPHANGITIS, or inflammation of lymphatic vessels, is due either to the absorption of irritant substances or to the infection of the vessels themselves by micro-organisms. The latter condition is encountered in erysipelas, where at the advancing edge of the affected area streptococci are formed within the vessel, the lining cells of which are increased in number. The clinical evidences of lymphangitis are well seen in that common occurrence, a septic wound of the hand, or, say, a whitlow. Extending from the wound, a rosy injection of the skin follows the path of the lymphatics, coursing upwards, towards, and sometimes to, the axilla, where the glands enlarge. The reddened skin is very tender. If incisions have been made (when necessary), and a wet carbolic dressing has been applied, in some cases the lymphangitis rapidly subsides, showing that it has been due to absorption of toxic products and not to the presence of organisms in the vessels. If, however, in spite of proper care the inflammation about the vessels does not subside, but an abscess forms in some part of their course, or in one of the glands, it proves that the organisms have passed into them. In gonorrhœa and syphilitic sores of the penis a subacute lymphangitis of the dorsal lymphatic of the penis is common.

Lymphangiectasis, or dilatation of lymphatic vessels, is observed wherever there is marked obstruction to the flow of lymph through the main lymphatics of a part. Thus in a case in which the thoracic duct was obstructed by a lymphosarcomatous growth in the abdominal glands, the superficial lymphatics

in the skin over the lower part of the abdomen became distended and tortuous, and were seen as white tortuous swellings covered only by thinned skin. In such cases the main lymph vessel of the lower part of the body being blocked, a collateral lymph circulation is established by dilatation of the superficial lymphatics which form connections above with the lymph vessels which enter the main trunks on each side of the neck. In other cases where there is chronic lymphangitis, as in the neighbourhood of chronic ulcers, the lymphatics may become dilated. In some tropical countries a parasite, the filaria sanguinis hominis, is introduced into the blood by mosquitos, and the full grown female worms choose the lymph vessels at the top of the thigh, or the superficial lymphatics at the root of the scrotum and penis, and in the female those of the labia; or, in other cases, they select the lymphatics of the kidney. The lymphatic vessels below the points where the large filaria lodge become dilated, and the tissues below them are subject to repeated attacks of inflammation, which results in the formation of an excess of fibrous tissue. In the skin this gives rise to *elephantiasis arabum.*

Lymphorrhœa. When dilated lymphatics below an obstruction are ruptured, whether spontaneously or by slight injury to the thinned skin covering them, lymph escapes as a milky fluid. In the kidney in cases of infection by filaria this gives rise to chylous urine.

Elephantiasis. Any lasting obstruction to the return of lymph from a part will give rise to great thickening of fibrous tissue. A common example of this is seen in some cases of chronic ulcer of the leg. The skin frequently becomes warty in the neighbourhood of the ulcers, and so greatly thickened that the elephantine appearance of the leg is very marked. Elephantiasis arabum, produced by the filaria sanguinis hominis, produces still more marked effects. The thighs may become as thick as the patient's waist, and the scrotum be so greatly enlarged that it comes to touch the ground in walking.

Now and again in this country a creeping form of lymphatic dilatation is observed, in which at first a patch of œdema appears in one or both lower limbs, and in the course of years slowly advances till a condition resembling elephantiasis is

produced. Treatment is of little service in such cases. In one case of this kind, under my colleague J. Poland, the disease affected a mother and her infant child. The cause of the condition is unknown.

Lymphangioma or Lymphatic Nævus has been described under morbid growths.

Lymphadenitis, or inflammation of lymphatic glands, is one of the commonest of surgical affections. From the frequency of slight injuries and septic wounds of the hands, the axillary glands are often inflamed, and when the pyococci obtain access to the gland, suppuration frequently follows. Thus the glands serve as a barrier against general infection. The deep cervical glands are frequently inflamed, from septic inflammation of the throat, and abscess beneath the sterno-mastoid is not uncommon. In children who suffer from superficial suppuration of the head and face (*contagious impetigo*), which frequently starts in the punctures of head-lice, the superficial glands of the scalp and those over the parotid are the first to inflame, the submaxillary and cervical glands also suffer. Under suitable antiseptic treatment the swelling and tenderness of the glands often subside, but not infrequently abscesses form. In other cases a chronic lymphadenitis is started, and is due to the entrance of tubercle bacilli into the glands. Inflammation of glands from bruises, etc., is described, but it is probably in most cases due to slight injuries of the skin, which serve to allow of the entrance of pyococci into the lymphatics, and heal up before the secondary inflammation occurs in the gland. The fact that such lymphadenitis not infrequently proceeds to abscess formation, supports the idea that it is probably due to inoculation.

A chronic lymphadenitis of the glands of the groins is sometimes caused by the pediculus pubis, which buries the upper part of its body in the skin.

Syphilitic Lymphadenitis in the secondary stage of the disease has been described under the account of Syphilis, p. 83. Gummata of lymphatic glands are of somewhat rare occurrence. The glands become hard and moderately enlarged, and have a somewhat angular outline. The affection appears independently of

any lesion of the skin, and usually subsides rapidly under treatment by iodide of potassium. In one case I examined after death, caseous gummata were present in the glands.

Tubercular Lymphadenitis is one of the most important of affections, both to physician and surgeon. The varying rate of tubercular disease has been alluded to in the general account of tuberculosis. It is well exemplified in the case of tubercular

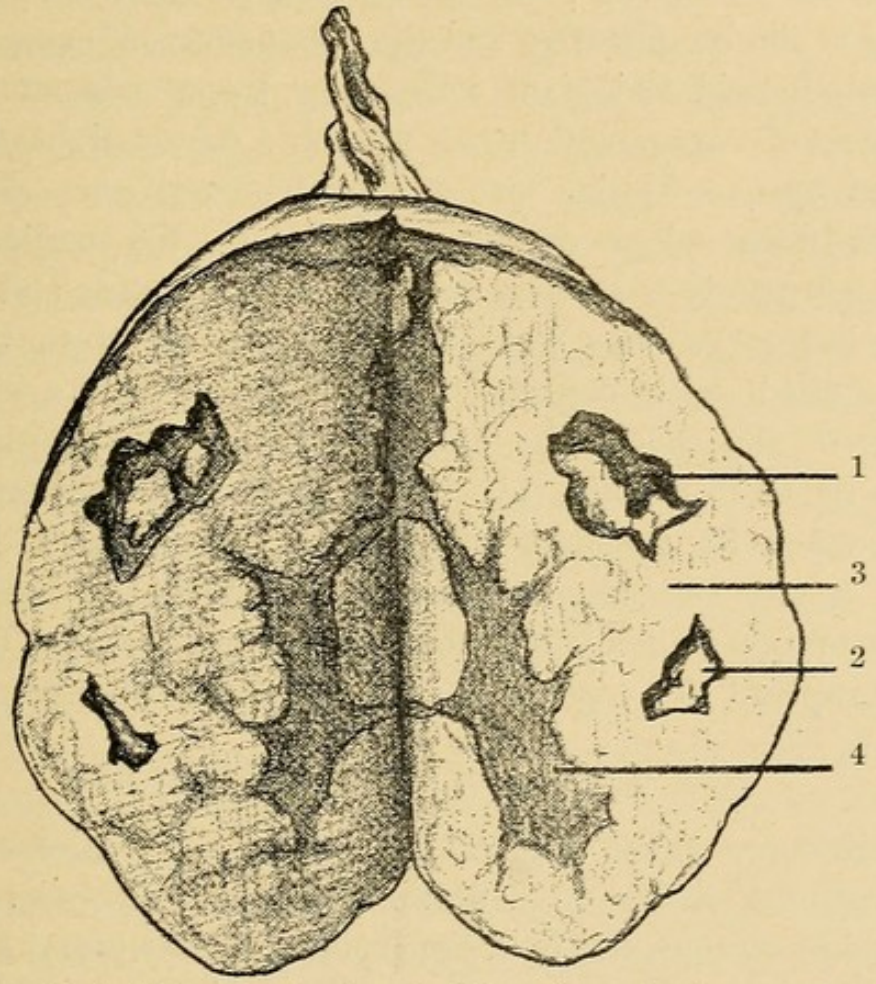


FIG. 96.—A tubercular gland excised from the neck of a girl. 1 and 2, Caseous masses which lie in cavities in the gland; 3, firm white tissue infiltrated by tubercle, but not yet caseous; 4, more normal part of the gland.

glands. Sometimes, after an indolent enlargement of the glands has been present for some months, they enlarge rapidly and break down into a tubercular abscess. In superficial glands the process then extends to the skin, and the pus escapes, forming sinuses which gradually close, leaving the familiar unsightly scars. In other cases the glands continue to enlarge to the size of pigeons' or bantams' eggs, and may give rise to a suspicion of commencing lymphosarcoma (see below). In other cases

again caseation and calcification go on slowly; the inflammatory process meanwhile passing beyond the capsule of the gland, causes the latter to become adherent to neighbouring glands and other parts, and so renders complete removal a matter of great difficulty.

On section an average tubercular gland (see Fig. 96) shows caseous areas, and the remaining gland substance is usually of a grey colour, *i.e.* much paler than normal, from the increase in number of the lymphocytes and the enlargement of many of them into endothelioid and giant cells. The larger œdematous cells, like those of cancer and other growths, refract light, and this gives an opaque whitish instead of the normal semi-translucent pink or fawn colour of the glands. In the earlier stages tubercular glands may only show increase in size and pallor; in the last stage they may consist of nothing more than the capsule filled with caseous matter. If in such a condition sections of the capsule be examined under the microscope, it will be found to contain tubercles. When tubercular glands lie within reach they should be removed as soon as a diagnosis can be made, *i.e.* before the infection has passed beyond the capsule of the glands. In operating great care must be taken not to rupture the capsule and so infect the wound.

New Growths of Lymphatic Glands.

Lymphoma. When a gland is enlarged, and on examination after removal shows no definite change, either tubercular or lymphosarcomatous, but only hyperplasia of the normal elements, it is termed a "Lymphoma," *i.e.* lymph-gland tumour. Such enlarged glands are probably not due to growth, but to hyperplasia from some source of irritation. The term is also applied vaguely to other conditions, *e.g.* adenoid foci in leucocythæmia.

Lymphosarcoma or Lymphadenoma is the commonest and at the same time one of the most formidable kinds of new growth that occur in lymphatic glands; in the writer's experience it is most common in the bronchial glands. In surgical cases the cervical, axillary, or other sets of lymphatic glands are the starting point (see p. 114). In situations accessible to the surgeon the glands should be excised as soon as possible. The examina-

tion of a freshly excised gland shows the more recent part of the growth to be of a pinkish-white colour. But caseation and even calcification frequently occur in lymphosarcomatous glands, and often renders the likeness to tubercle very close. In the neck lymphosarcoma may give rise to large lobulated masses of growth which quite obscure the normal contours. The vertebræ are often invaded by the malignant mass when it arises in the cervical, thoracic, or lumbar glands.

Leucocythæmia, or Leukæmia (white-cell-blood, or white-blood disease), is, again, somewhat akin to Hodgkin's disease. Here, however, there is not merely enlargement of lymphatic glands and spleen, accompanied by anæmia, but also a marked increase in the number of white cells in the blood. In the more common forms of the disease marked by great enlargement of the spleen, the ordinary multinuclear leucocytes are increased in number, and besides there are large cells with kidney-shaped nuclei, such as are normally found in bone marrow. In another form it is chiefly the mononucleated leucocytes (lymphocytes) that are increased in number. The disease is accompanied by increased growth of lymphoid tissue in the spleen, kidneys, liver, bones, etc. The surgical importance of leucocythæmia lies in the fact that the enlarged spleen may be mistaken for an ovarian or other tumour, and that in patients who suffer from the disease, surgical wounds heal slowly or not at all, owing to the accumulation in the tissues of leucocytes which, like the leucocytes in acute inflammation, impede repair and are not, as was formerly believed, of use in the formation of scar-tissue.

Sarcoma of Lymphatic Glands other than lymphosarcoma is rare as a primary, but not very uncommon as a secondary growth. Secondary *cancer* is common.

CHAPTER XVI.

SURGICAL AFFECTIONS OF THE MOUTH, TONGUE, AND ALIMENTARY TRACT.

APART from developmental errors, which have been considered elsewhere, there are a great many conditions of the mucous lining of the mouth which call for surgical consideration. A slight congenital shortness of the frenum of the tongue is common, and gives rise to the condition known as *tongue-tie.*

Ulcers of the Mouth and Tongue. The small, round, painful excoriations and ulcers not uncommon on the inner surfaces of the lips and on the tongue, appear to arise as vesicles similar to those which constitute herpes of the lips. They may be called herpetic ulcers. Other somewhat similar superficial lesions are described as arising from the suppuration of a mucous gland. They are termed follicular ulcers. The pathology of these minor ulcers seems to require reinvestigation. Surgically they are of importance from a diagnostic point of view, it being necessary to differentiate between them and syphilitic lesions.

Ulcerative Stomatitis. Is seen chiefly in badly-fed children. The inner surfaces of the lips, opposite to, and the gums around, the teeth, undergo a superficial suppuration, which leads to ulceration. This is accompanied by much fetor. This condition is one of those which are benefited by the internal administration of a drug, *viz.* chlorate of potassium. Similar ulceration occurs in mercurial poisoning, and, with bleeding, in scurvy. Ulcerative stomatitis may leave rigidity of the jaws by contraction of the scars left after healing of the ulcers.

Gangrenous Stomatitis or Noma is also observed in badly-fed

children, especially after the infective fevers. The disease first shows itself by tense red swelling of the cheek. The centre of the swelling is usually deeper in colour than the rest. On the inner side of the cheek at this stage there is an ulcer, which is small at first but rapidly increases in size, and, as this is taking place, the central part of the cheek becomes gangrenous and black. The gangrene spreads until death from septic intoxication or septic infection occurs.

Early thorough removal of all the gangrenous part and cauterization of the parts thus exposed with fuming nitric acid or the actual cautery is imperative. A similar affection sometimes occurs in the vulva.

Many other morbid conditions of the mouth are of importance to the surgeon, among them several claim special attention.

Ulcer of the Tongue from injury caused by a jagged tooth. The importance of the condition lies in the fact that it may be mistaken for cancer. Usually the induration of cancer is want ing, but since more or less induration is often present, it is best to remove the tooth, apply carbolic acid, and watch whether the ulcer begins to heal in a few days.

Acute Parenchymatous Glossitis—Acute Cellulitis of the Tongue, which now and again occurs, is sometimes the result of a bite or other injury; sometimes its origin cannot be traced. Rapid swelling of the tongue, which may become so large that it protrudes between the teeth and causes difficulty in respiration, pain, fever, etc., mark the affection. Abscess sometimes occurs. Bold incisions, one on either side of the middle line of the dorsum, are called for.

Urticarial Œdema of the tongue also causes rapid swelling, but without the fever, etc., which mark acute glossitis.

Ranula is a cyst which appears as a bluish swelling in the floor of the mouth at the side of the tongue. Ranula originates in the dilatation of one or more of the glands which lie in pairs on either side of the frenum of the tongue. They contain a thick, tenacious, clear fluid. In order to obtain a radical cure of ranula part of its wall must be cut away, the contents evacuated, and the cavity packed with lint wrung out of Tr. Iodi so that the cyst is closed by granulation tissue.

A Dermoid Cyst is sometimes found in the floor of the mouth between the genio-hyo-glossi muscles. Such cysts project into the mouth and below the jaw. They do not usually show themselves until the patient has passed puberty, though they are of congenital origin. They are lined with skin, and contain hair and sebaceous matter.

Adenoma of the Palate. This is an encapsuled tumour which sometimes forms in the soft palate, and is easily enucleated.

Macroglossia and nævus of the tongue have been described under diseases of the lymph-vessels.

Papillomata are not uncommon on the tongue.

Tubercular Ulceration of the Mouth. This may spread from the face to the gums, palate, etc., as part of a lupus vulgaris, or in consumptive persons superficial tubercular ulcers characterized by a greyish colour and great pain may occur. They are more frequent in the anterior part of the tongue, but sometimes they are found at the back of the organ (see Fig. 125).

Syphilitic Affections of the Mouth. Mucous tubercles are common, especially in the earlier period. Chronic inflammation of the mucous membrane of the cheek or of the tongue is not uncommon in the later period of the disease. Though smoking and other conditions produce similar inflammatory changes, syphilis is by far the commonest cause of the more severe cases. In the tongue the thickening is chiefly in the epidermal covering, whilst the fibrous part of the mucous membrane is infiltrated with small round cells; this gives the affected part a white opaque appearance, which, when it occurs on the tongue, is known as "Ichthyosis linguæ," or, when it is of more extensive degree, "Leucoplakia," This form of syphilitic glossitis is very prone to change into a cancerous process.

The tongue is one of the commonest sites for tertiary syphilitic ulceration. Superficial ulcers, resulting from the breaking down of a shallow gummatous infiltration, readily heal under treatment. The ulcer left by the breaking-down and opening of a larger gumma of the tongue presents sometimes some similarity to a cancerous ulcer, but the history and progress of the case enable its nature to be made out.

Many tertiary ulcers are extremely obstinate, and by the

contraction, due to cicatricial changes about them, they tend to assume the form of fissures.

Cancer arising primarily in the mouth is nearly always of the squamous-celled type. It may arise in any part: on the tongue, the floor of the mouth, the gums, etc. The most common appearance of a cancer of the tongue is shown in Fig. 97.

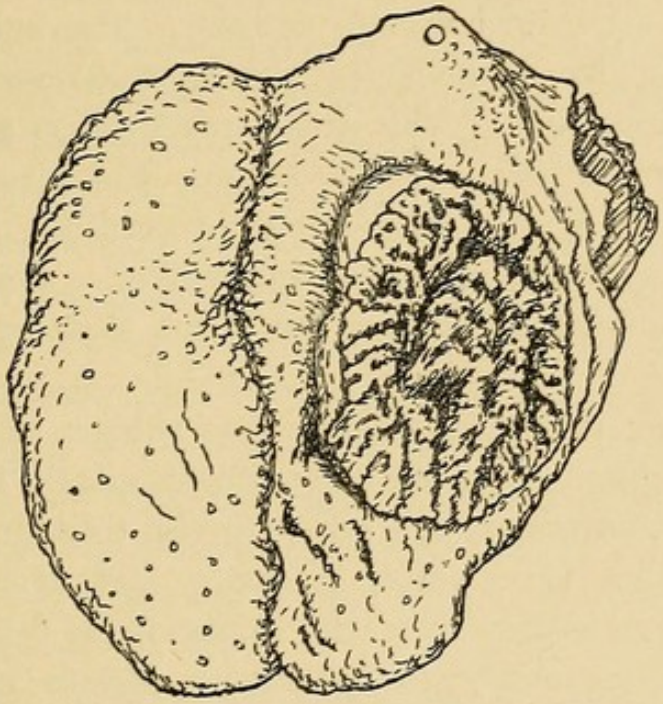


FIG. 97.—Part of a tongue amputated on account of the cancer, the ulcerated surface of which occupies the middle portion of the left half of the organ. (812 A.)

More rarely a squamous cancer grows widely in the interior of the tongue, whilst the original superficial part remains of limited extent.

Affections of the Tonsils.

The Tonsils are sensitive to infection both from without and within. Cold seems to act as an aid in producing infection in some cases. Local infection of the tonsils is common in diphtheria. In others general infection such as influenza, scarlet fever, or syphilis, affects the tonsils. In the majority of cases of acquired syphilis the tonsils are swollen and ulcerated in the secondary and sometimes in the tertiary stage.

Acute Tonsillitis whether in the form of a diffuse inflammation of the lymphoid tissue, which may lead to ulceration

or abscess, or in the form of follicular tonsillitis in which the mouths of the crypts fill with soft white catarrhal matter, is a common affection. There is pain on swallowing and sharp fever. Occasionally acute inflammation of the cervical glands of one or both sides ensues.

Chronic Tonsillitis leading to hyperplasia of the lymphoid tissue—the so-called hypertrophy of the tonsils—is usually marked by a series of subacute attacks. The enlarged tonsils if accompanied by adenoids in the naso-pharynx add to the difficulty of breathing. The usual treatment is slicing off the superficial part of the enlarged organ. Complete enucleation is in some cases a more satisfactory proceeding.

Malignant growths are not uncommon. Squamous-celled cancer and lymphosarcoma are the forms that most frequently occur.

The Pharynx is subject to affections similar to those of the tonsil. The lymphoid tissue and mucous glands when affected by *chronic pharyngitis* enlarge and give rise to granular pharynx, a condition that is common in gouty and anæmic patients. Retro-pharyngeal abscess may arise in one of the retro-pharyngeal glands or be secondary to tubercular disease of the upper cervical vertebræ.

Diseases of the Œsophagus. Surgical conditions of the œsophagus include the results of impaction of foreign bodies, such as sets of false teeth, etc., which may require the operation of œsophagotomy for their removal.

Fibrous Stricture of the Œsophagus results from the action of corrosives. The œsophagus above the stricture becomes dilated and hypertrophied, and when the narrowing is marked the dilatation may be so great that large quantities of fluid accumulate there to be regurgitated within an hour or so after food is taken. This condition is distinguished from gastric vomiting by the absence of gastric juice in the vomit.

Narrowing of the Œsophagus from other Causes. Cancerous stricture is the most important of these. The growth is of the squamous-celled kind. The usual sites for its commencement are: (1) the junction of the œsophagus and the pharynx; (2) opposite the aortic arch or the lower end of the trachea;

(3) where the stratified epithelium of the œsophagus abruptly joins the columnar epithelium of the stomach. The second is the most frequently observed. The first effects of the stricture are the same as those of simple stricture; later the destructive infiltration of cancer may lead to perforation of the trachea, leading to death from septic bronchopneumonia, gangrene of the lungs, etc. The dilatation of the tube above the stricture together with the friable nature of the cancerous growth necessitates the greatest caution in passing sounds. It is important also before passing a sound to exclude the possibility of the narrowing (or stenosis) being due to aneurysm of the aorta. Another condition often mistaken for cancer of the œsophagus is lymphosarcoma of the mediastinal glands; this, like aneurysm of the aorta, causes stenosis by pressure from without. The distinction between cancer of the œsophagus and lymphosarcoma of the mediastinum is also of importance since in the latter condition the surgeon would refrain from performing gastrostomy to enable the patient to be fed, whereas he would do that operation for cancer of the œsophagus. Another condition in which symptoms of occlusion of the œsophagus have led to the operation of gastrostomy being done is the presence of a small nodule of cancerous growth outside the œsophagus but adherent to it and to the vertebræ. Spasm in gouty œsophagitis and reflex spasm from some other morbid conditions may also simulate œsophageal stricture.

Diseases of surgical importance in the stomach and intestine other than those of the rectum will be considered under "Peritonitis and Intestinal Obstruction."

Diseases of the Rectum and Anus.

The lowest part of the large intestine is accessible to touch, and by means of specula to sight. The peculiarities of the vascular supply and muscular connections render it subject to affections not encountered in the higher part of the bowel in addition to most of those which are found in other parts of the intestine. In order to render the pathology of these affections more easy of description it will be well to recall, in the first

place, some aspects of the anatomy of the part not usually dealt with in ordinary text-books.

In Fig. 98 is represented the interior of a normal rectum of a boy aged nine years, opened from the mid-posterior line.

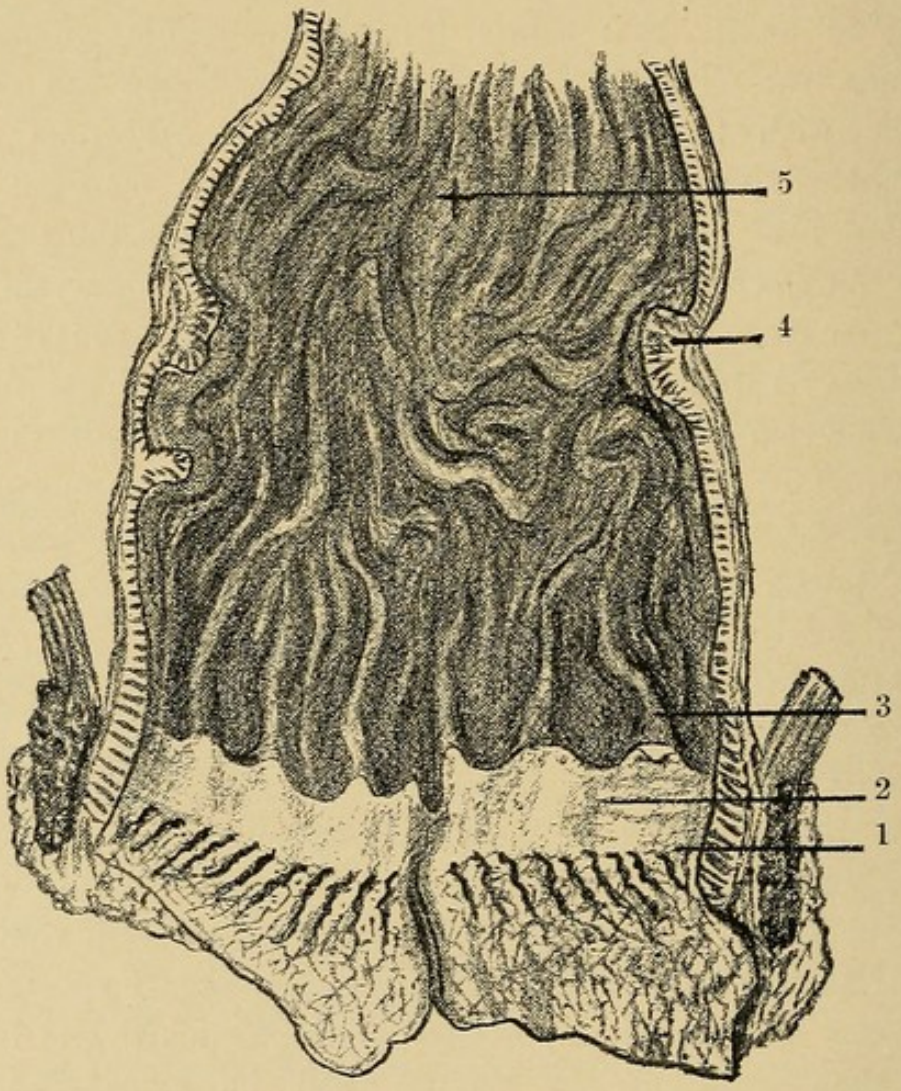


FIG. 98.—1, Muco-cutaneous junction; 2, pecten (comb) or mucous lining of the anal canal, ending above at the "linea dentata," the pointed processes of which are prolonged into the "columns of Morgagni; 3, the depressions between the columns—the saccules of Horner; 4, fold of Houston; 5, the lowest level of the peritoneum. Natural size.

The whole of the pecten is lined with squamous epithelium, and its upper limit represents the attachment of the proctodæal membrane, which, when it persists, constitutes one variety of imperforate anus.

Perhaps the commonest disease of the rectum is piles or varicosity of the veins of its lower inch or more. For this reason it is desirable to recall the arrangement of the blood-

vessels of the rectum. The superior hæmorrhoidal vessels divide each into two branches, and then each branch subdivides

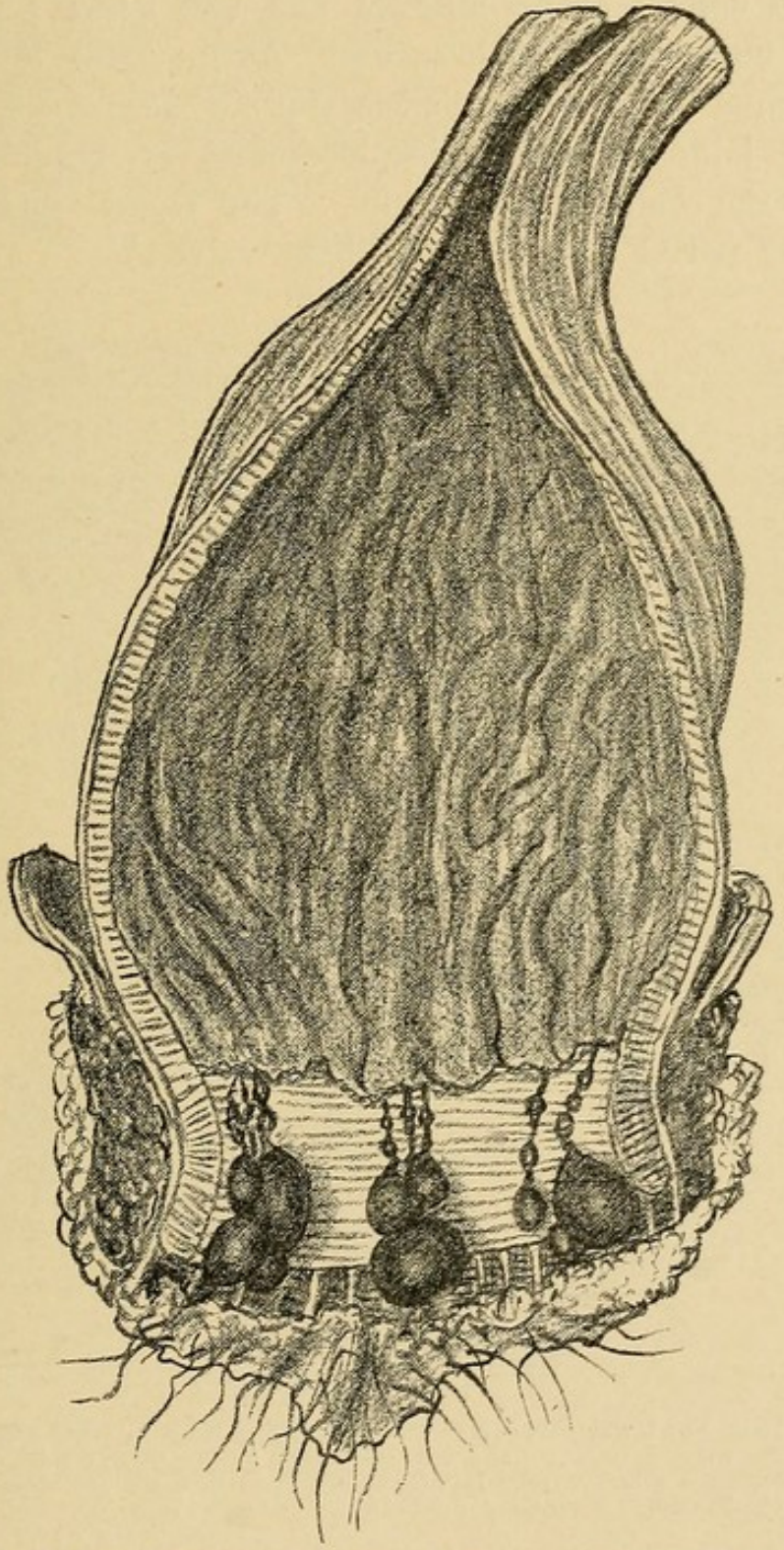


FIG. 99.—Thrombosed external piles. The thrombosed varices correspond to the three chief groups of veins. The white fibres of the longtitudinal muscular coat are seen emerging below the internal sphincter.

into two or more; the resulting vessels perforate the muscular coat in the adult four inches above the anus, and undergoing

further subdivisions in the submucous tissue, they pass downwards to the anus. On dissection it is found that the veins are grouped into three chief sets, an anterior and two lateral; there are, however, numerous cross branches. At the anus the superior hæmorrhoidal join the cutaneous (*inferior hæmorrhoidal*) veins, and they join the middle hæmorrhoidal by branches which perforate the wall of the rectum. The rectal veins like the rest of the portal system are without valves.

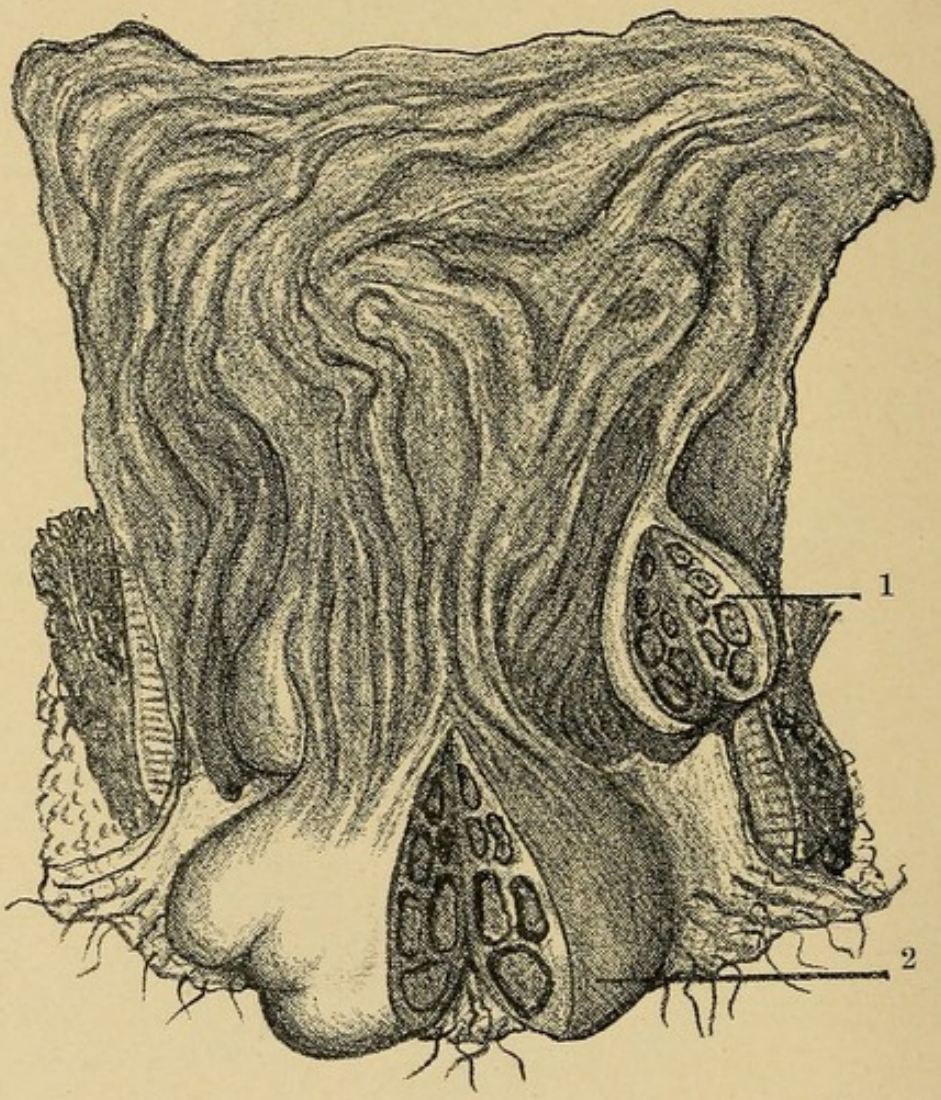


FIG. 100.—The lower part of a rectum presenting 1, an internal pile which has been incised to show the dilated venous spaces occupied by clot; 2, intero-external pile incised. It will be observed that the attachment of 2 reaches to the muco-cutaneous junction.

External Piles are varicosities at the junction of the hæmorrhoidal with the cutaneous veins. They are visible as soft compressible swellings. Frequently they become thrombosed. Then, if left to themselves, they become organized, the process

being accompanied by a dull pain lasting for seven or eight days. This results in the formation of a fibrous nodule. Sometimes the thrombosed pile suppurates. A dissection of thrombosed external piles is shown in Fig. 99. It will be noticed that in this case there are several varices in each external pile; the upper part of the piles extend as far upwards as the linea dentata. This is not uncommonly observed, but the dilated and thrombosed part is often single and altogether outside the anal orifice and beyond the grip of the sphincters, in which case, if the pain is severe, the clot can be readily turned out through an incision and relief obtained. **Internal piles** are varicosities of the rectal veins situated above the linea dentata (Fig. 100, 1).

Intero-external Piles are continuous varicosities of the veins from above the linea dentata to the skin (Fig. 100, 2).

Sometimes internal or intero-external piles form a continuous circle of varicosities around the lower inch or more of the rectum. Internal and intero-external piles are subject to thrombosis like external piles, and with the same results, *i.e.* formation of fibrous tabs (*rectal papillæ*), or abscess formation with subsequent ulceration. Varicose ulceration occurs in the rectum just as it does in the leg.

Arterial Piles. Rarely in internal piles, not only the veins but also the artery and its branches dilate, forming a combination of a varix with a cirsoid aneurysm. Such piles may bleed furiously as contrasted with the moderate bleeding of an ordinary venous pile. The main artery of an arterial pile has been observed to be as large as the radial.

Capillary Piles are small sessile papillated patches generally situated just within the internal sphincter. They bleed very readily since the dilated vessels are the capillaries immediately beneath the epithelium. They are commonly met with in children.

The causes of piles are local and general. Locally masses of fæces in constipation, by preventing the return of venous blood, help to produce them. Similarly pelvic tumours or cancer and other growths of the rectum, and in pregnancy the gravid uterus may have a similar effect. In the last instance,

since the cause of the disease will in course of time remove itself, there is no call for active surgical interference, at least during the pregnancy. Among general causes sedentary habits by producing passive portal congestion, or excessive eating and drinking by active portal congestion, may act as causes. Cirrhosis of the liver produces a congestion of the rectum, as it does of the whole of the intestine and stomach, and it may lead to the formation of piles.

Pruritus ani, popularly termed "itching piles," is an eczematous condition combined with œdema, resembling that of urticaria. It is often complicated by external piles. From scratching, excoriations and ulcers may form, and the condition is then one of intolerable itching. Simple cleanliness, with regulation of the bowels, etc., will arrest it in the earlier stages; in the later, cauterization, scraping, etc., may be required.

The skin about the anus is a favourite seat for *mucous tubercles*, usually termed piles by the laity. In infants other syphilitic cutaneous infiltrations are common about the anus. Ordinary, and in women gonorrhœal, warts are met with at the anus.

Ischio-rectal and Anal Abscess; Fistula in Ano.

Anal abscesses are small subcutaneous collections of pus which tend to open just within or without the muco-cutaneous junction at the anal orifice.

Ischio-rectal Abscesses (Periproctitis) form in the fat of the ischio-rectal fossa. Though they are accompanied by swelling and redness of the skin at the side of the skin, if not speedily opened, they more commonly rupture into the rectum above the internal sphincter, forming a "blind" internal fistula, which is closed externally (Fig. 101, 4). If it should rupture externally and not internally a blind external fistula is constituted (Fig. 101, 3). If it open both externally and internally it forms a complete fistula (Fig. 101, 1). If the pus forms channels with one or more openings into the rectum and two or more upon the skin, what is termed a horse-shoe fistula is the result (Fig. 101, 6).

The origin of ischio-rectal abscess is usually a lymphangitis starting either in an excoriation within the rectum or a similar lesion on the skin. Persons suffering from pulmonary consumption are especially liable to the formation of fistula, which begins in suppuration beneath a tubercular ulcer; when the pulmonary disease is acute this is a contra-indication to operation.

In all cases of fistula not only must the exact extent of the fistula be investigated, but also search should be made for cancerous syphilitic or tubercular ulcers which may have determined the formation of the fistula. It must be remembered that sinuses opening about the anus may have their origin in tubercular disease of the coccyx or some other condition quite independent of rectal disease.

Erysipelatous or Gangrenous Periproctitis in rare instances has followed operations on the rectum, and has proved a very fatal complication. Free incisions give the only chance of success.

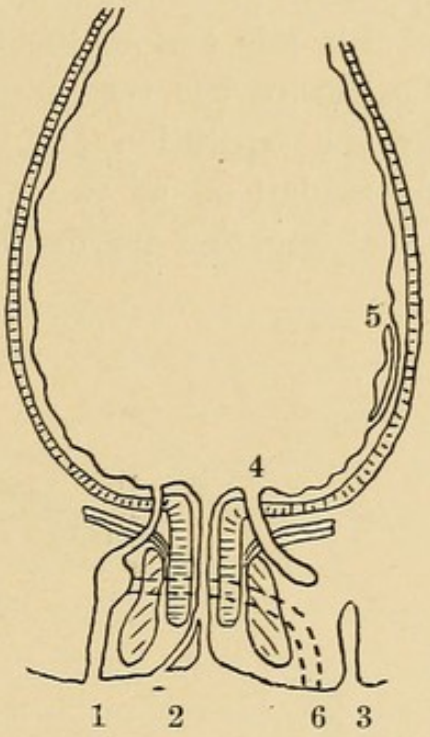


FIG. 101.—Diagram of a frontal vertical section of the rectum and ischio-rectal fossæ showing, 1, an ischio-rectal abscess opening into the rectum, forming a complete fistula; 2, complete fistula at the anal margin; 3, blind external fistula; 4, blind internal fistula; 5, complete internal fistula; 6, horse-shoe fistula, formed by a branch from a complete fistula, so that there are two external orifices.

Ulceration of the Rectum and Anus.

Fissure of the Anus is a chap or narrow superficial ulcer of the anal canal, sometimes extending to the skin. In some instances the fissure appears to begin externally at the muco-cutaneous junction, as stated previously. These fissures present but little tendency to spontaneous repair. They are generally accompanied by severe pain which lasts for an hour or so after defæcation, and is accompanied by spasm of the sphincter. On examining the anus this spasm of the sphincter is at once noticed, and the pursing up of the parts renders the fissure

invisible. In a certain proportion of cases the lower extremity of the fissure is overhung by a round tense pink swelling, the so-called "sentinel pile" (Fig. 102). Ball explains the origin of this pile and of the fissure in such cases by supposing that the fissure has been formed like a "hang-nail" on the finger by the separation and gradual forcing down of one of the denticulations of the pecten. The mid-posterior line is the usual situation for fissure.

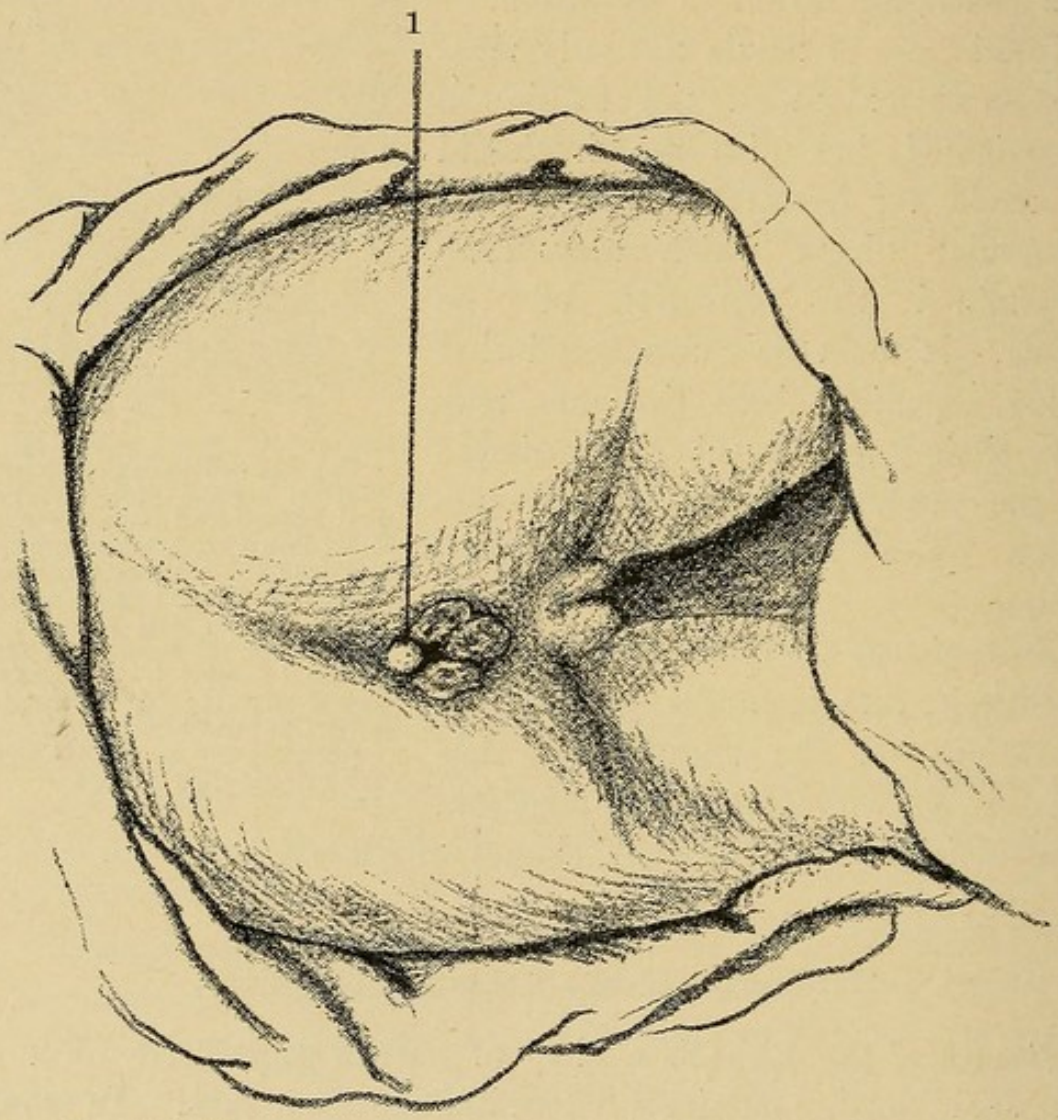


FIG. 102.—The anus and surrounding parts of a woman of twenty-five years. 1, The tense rounded "sentinel" pile of a fissure. There are also three ordinary external piles which have the same positions as those shown in Fig. 99.

The sentinal pile is by no means of constant occurrence in fissure of the anus; sometimes the upper end of the fissure is placed between two of the processes of the pecten.

Ulceration in connection with piles has already been mentioned.

Dysentery. Ulceration of the rectum is very common in this disease, which is chiefly one of tropical climates. It is attended with very severe pain, and is liable to be complicated by abscess of the liver, fistulæ, and stricture.

Ulcerative colitis is a severe affection which in its earlier phases is somewhat similar to acute dysentery. It is however a distinct disease. It begins generally in the rectum and ascends to the ileo-cœcal valve. Pain, diarrhœa, and fever are the most marked symptoms in the acute stage of the affection. At this period the mucous membrane is swollen and covered with patches of muco-pus; the mouths of the tubular glands are enlarged and show as minute pits. Ulcers begin to form by the deepening and fusion of thes pits. Recovery may take place without farther extension of the disease.

Whatever may be the initial agent in causing the disease, when once ulceration has commenced a rapid muco-cellulitis occurs in those cases which do not recover in the early stage. The mucous membrane becomes undermined, and by remaining attached at the points where the vessels pierce the muscular coat forms numberless bridges between the ulcers. Pellets of hard fæces may collect under these bridges and so simulate a cancerous or other growth by forming a lump perceptible on examining the abdominal wall by palpation.

Later still the bridges give way in the middle, and remain as pendent strips. If the patient does not succumb to the disease, healing may occur by growth of the epithelium from the polypus-like remains of the mucous membrane. It is probable that some of the so-called *glandular polypi* are of this character.

In some cases the remains of the mucous membrane increase in size by a chronic hyperplastic (inflammatory) process. Thus they come to form adenoma-like polypi which may project from the anus and be mistaken for piles.

Tubercular Ulceration has characters similar to those of other tubercular ulcers of the intestine. It has already been mentioned as one source of fistula.

Syphilitic Ulceration occurs in the tertiary stage of the disease, and is commoner in women than in men. It is also

a cause of fistula formation (see Fig. 103). The ulceration is often spread over an extensive area of the bowel. It is the commonest cause of *fibrous stricture* of the rectum. Another form of syphilitic disease of the rectum consists in extensive gummatous infiltration of the muscular and fibrous coats of the bowel.

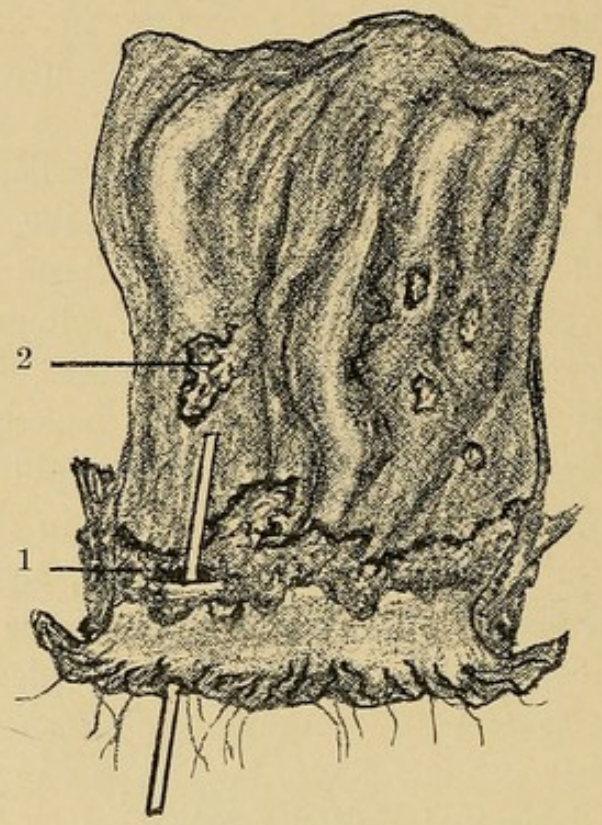


FIG. 103.—Syphilitic ulceration of the rectum. A large ulcer, 1, communicates with the exterior by a fistula which is marked by a glass rod and which commences just above the internal sphincter; 2, a small ulcer healing at its upper part.

Cancerous Ulceration and stricture of the rectum may occur in early adult life, but, as with cancer generally, it is generally met with in middle or advanced life. If the disease arises in the skin at the anus, or in the mucous membrane of the pecten, it is of the squamous-celled variety; if it arises higher in the bowel it has the same character as cancer of other parts of the intestine (see p. 95). The progressive character of the disease, and the deep infiltration and induration, together with its refractory behaviour under treatment, usually make diagnosis easy. When the disease is not too far advanced, any part of or the whole rectum may be removed. (See Figs. 104 and 105.)

Other New-Growths in the Rectum. Adenoma occurs in two forms—one of smooth rounded aspect, the other of a papillo-

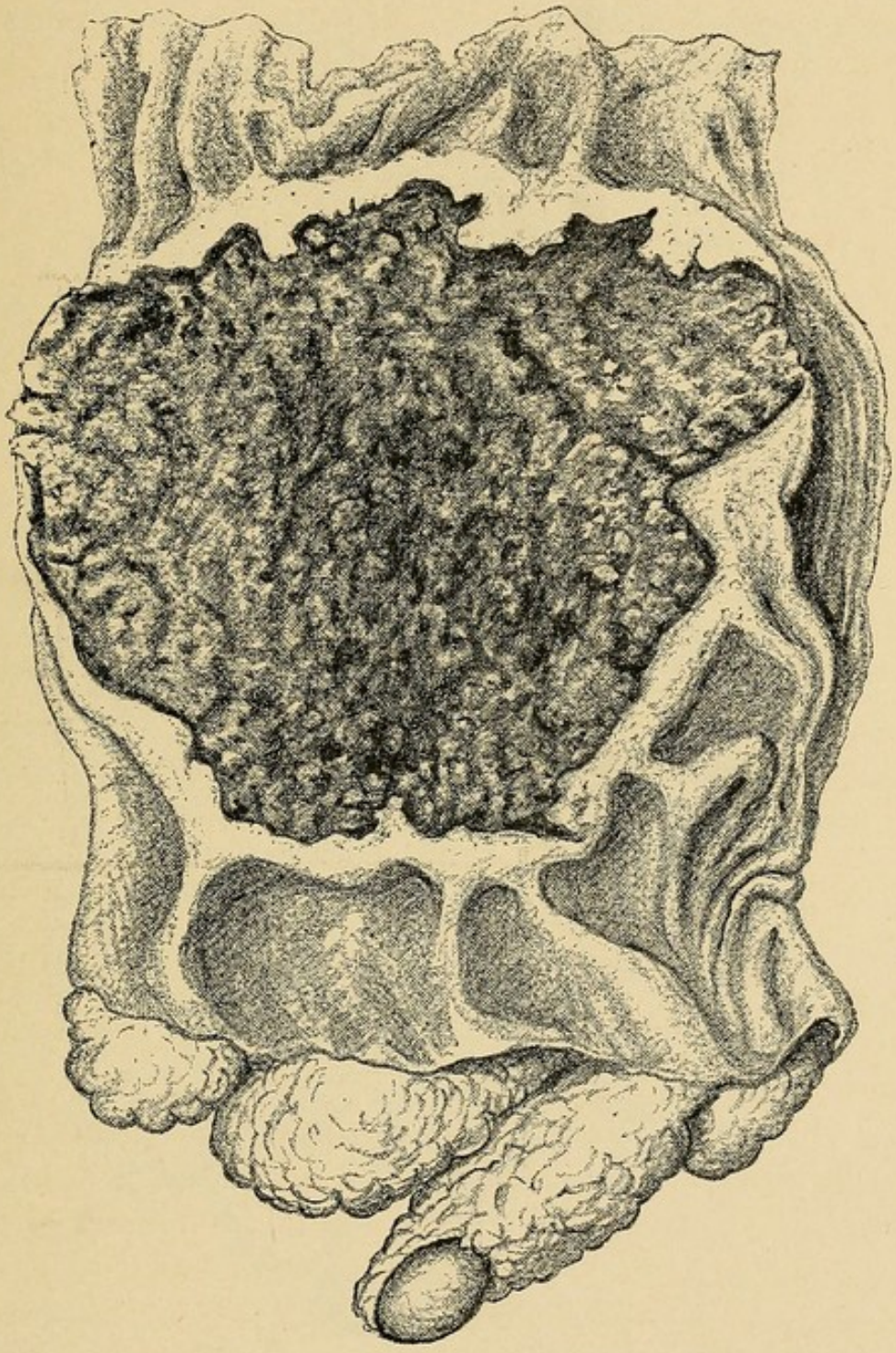


FIG. 104.—The upper four inches of the rectum. The bowel has been opened from the front, showing a typical cancer of proliferating type. Removed by Kraske's operation from a man aged 51. Nat. size.

matous character. They are usually pedunculated but sometimes are sessile. They are rather more frequent in children than in adults.

Prolapsus Ani and Procidentia Recti. Anal prolapse is an eversion of the mucous membrane at the anus which occurs

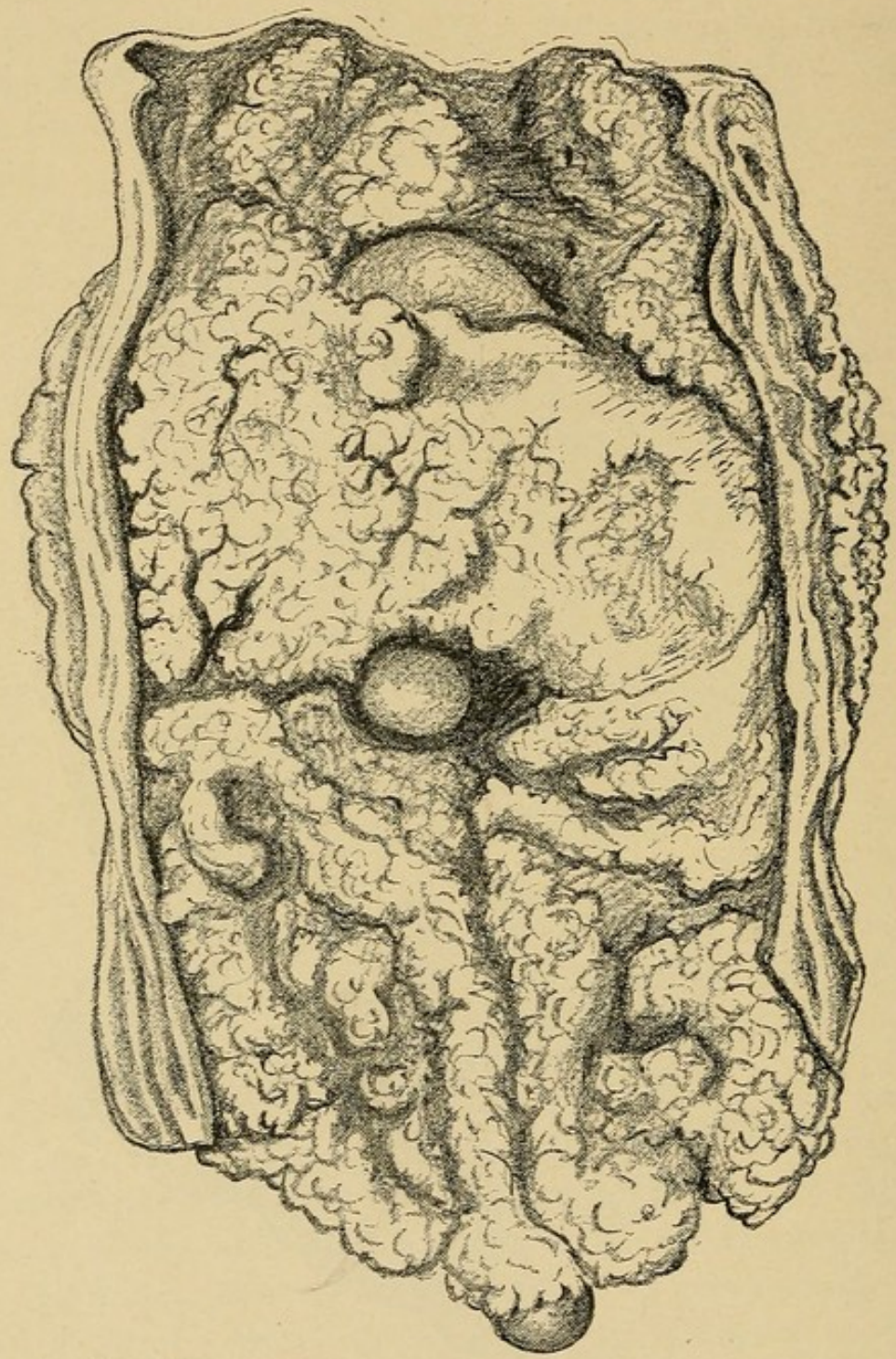


FIG. 105.—The same portion of rectum seen from behind. There is a considerable quantity of fat in which are embedded two enlarged lymphatic glands—one opposite the middle, the other at the lower end of the specimen. Nat. size.

both in adults and in ill-fed children. If the diet, etc., be corrected and the prolapsed portion returned after each stool, the condition usually disappears; but in neglected cases the

prolapsed portion becomes œdematous from inflammatory hyperplasia; it may ulcerate or even become gangrenous.

Procidentia recti is the descent of the whole thickness of the rectum through the anus. The condition is the equivalent

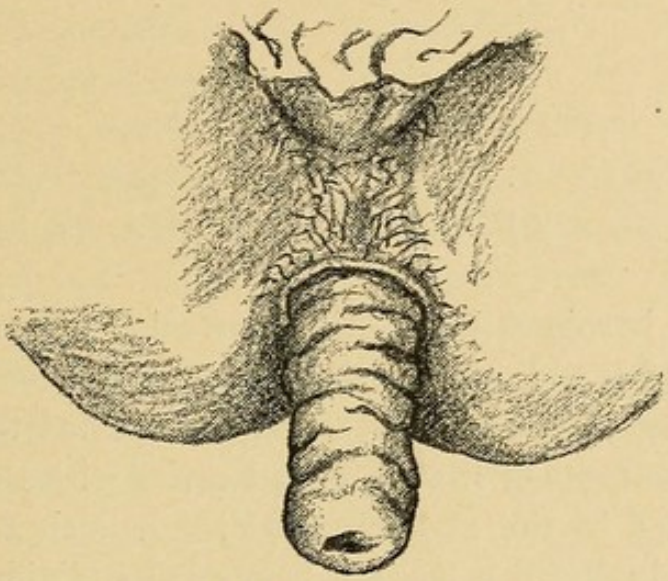


Fig. 106.—Procidentia Recti.

of intussusception (see below) in other parts of the bowel. In advanced cases there is a portion of the peritoneal space between the inner and outer part of the prolapsed bowel (see Fig. 106).

CHAPTER XVII.

ABDOMINAL HERNIA.

By the term hernia is meant the protrusion of a viscus or part of a viscus beyond the cavity that should contain it. In abdominal surgery the term is commonly applied to a bulging of some of the contents of the peritoneal cavity with the peritoneum at a weak spot in the abdominal wall. If such a protrusion is produced gradually in a previously normal individual, both the bulged peritoneum and the protruded abdominal contents are at first easily pressed back into their proper place or reduced. If the protrusion remains unreduced for a sufficient length of time the *sac*, or peritoneal part of the hernia, becomes adherent by the subperitoneal fat that covers it to surrounding parts, and so the sac becomes irreducible; and, farther, the part of the sac that passes through the weak spot in the abdominal wall, the *neck* of the sac as it is termed, becomes permanently narrowed by chronic inflammatory changes which cause thickening and adhesion of the folds into which the sac is thrown at the neck as more and more of the peritoneum is pushed through the aperture in the abdominal wall.

Sometimes the sac becomes greatly thickened in every part from chronic inflammatory changes which increase the amount of tissue in the sac, and cause the surrounding tissues to adhere to it.

The contents of a hernia are various: Omentum, intestine, ovary, bladder, etc., severally or together may be found. The two first named are the more common. A hernia containing omentum alone has been called an *epiplocele*; one containing bowel alone an *enterocele*.

Narrowing and thickening of the neck of the sac may render

it impossible to reduce the contents of the sac into the abdominal cavity. Another cause of *irreducibility* of the contents of a hernia is the formation of inflammatory adhesions between the sac and its contents, and between the different parts of the contents to one another. Increase in the amount of fat and fibrous tissue in the omental or other of the contents is another cause of irreducibility.

A hernia may become irreducible by reason of the large proportion of the abdominal contents being contained within it. In such cases the abdominal cavity becomes smaller, so that there is no longer room for the herniated viscera to be returned. These large herniæ serve to impress on the mind the fact that intestinal obstruction is, in most cases, merely an accidental complication of a hernia. During a muscular effort which increases the intra-abdominal pressure an additional portion of bowel may be forced into the sac, then the pressure of the neck of the sac or of the margin of the opening in the abdominal wall on the bowel may be so great as to cause obliteration of the lumen of the protruded part of the intestine. Such a hernia is said to be strangulated (see under "Intestinal Obstruction").

Sometimes when a hernia is suddenly developed during great muscular effort there is intestinal obstruction from the first.

The lumen of the herniated bowel may be blocked by firm fæcal matter, which is then said to be impacted, and the condition is known as "incarcerated hernia." Further, inflammation of the wall of the bowel in a hernia by weakening or paralyzing the muscular coat may give rise to more or less obstruction. Thus there may be intestinal obstruction from incarceration or inflammation of a hernia, and so these conditions may be mistaken for strangulation.

Congenital Hernia. During the early or embryonic part of fœtal life a portion of the small intestine lies in the base of the umbilical cord covered by thin amniotic membrane. In the normal course of development this part of intestine prolapsed into the cord is accommodated within the abdominal cavity by the growth of the abdominal parietes. Occasionally, however, this fails, like other developmental processes. If the child is

born before this process is completed the condition constitutes one form of congenital umbilical hernia.

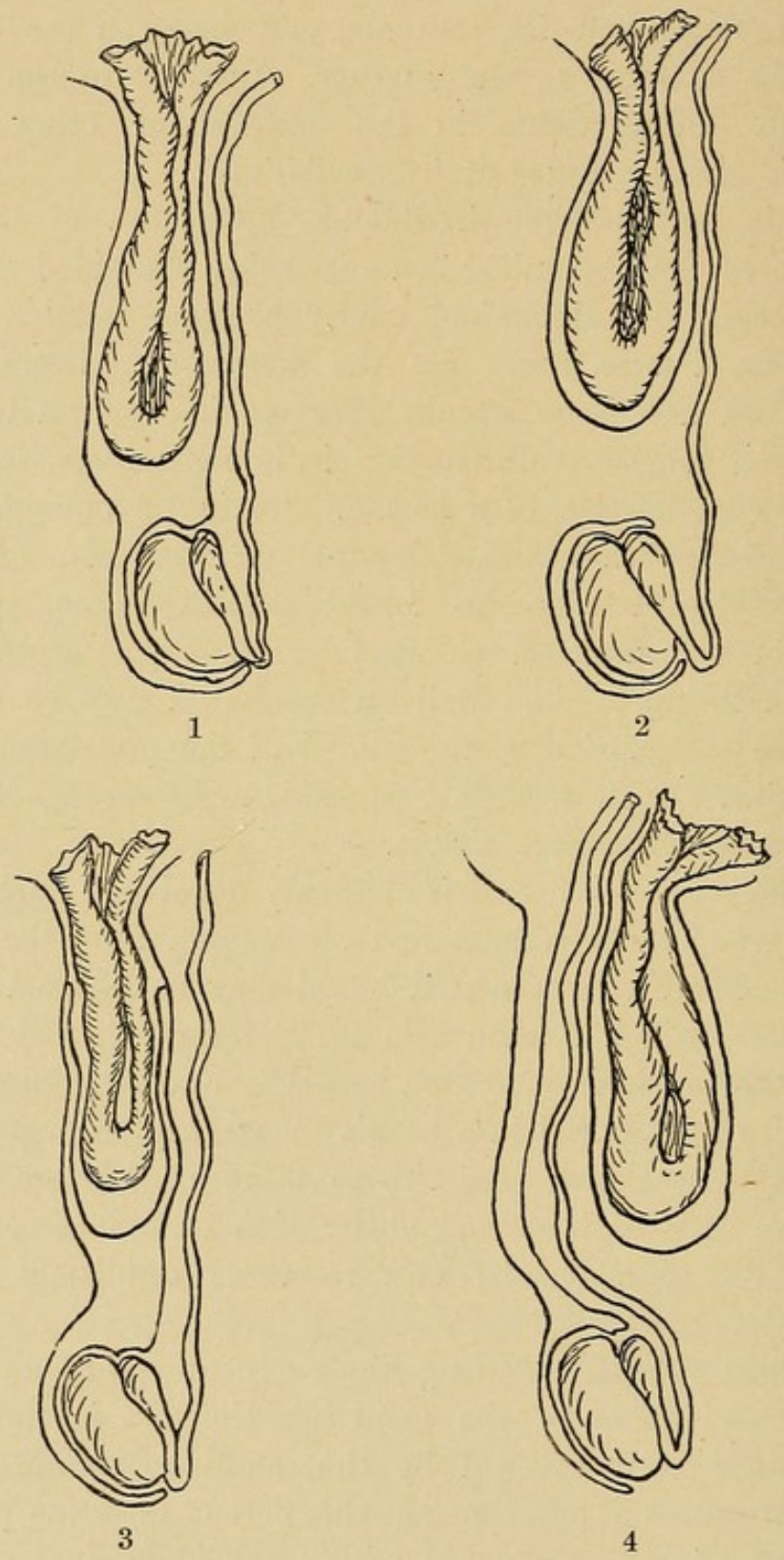


FIG. 107.—Diagrams of inguinal herniæ. 1, Congenital hernia; 2, funicular hernia. This diagram would serve equally well to illustrate the ordinary acquired inguinal hernia. 3, Infantile hernia, first variety; 4, infantile hernia, second variety.

The common umbilical hernia differs from the foregoing in that the portion of intestine after having re-entered the abdominal cavity is again prolapsed, but with a covering of skin and a sac of peritoneum. In the first variety if the quantity of bowel is not too great to allow of its being returned to the abdomen this should be done immediately after birth and the umbilical opening closed by sutures. When the hernia is covered by skin the urgency is not so great.

Congenital Inguinal Hernia. The vaginal processes of peritoneum, which usually pass with the testes through the internal abdominal ring, along the inguinal canal, through the external ring and on into the scrotum, under normal conditions, become obliterated from above down before birth, except their lowest parts, which remain as the tunicæ vaginales of the testes. If this obliteration fails to take place then there is extending from the peritoneum to the scrotum a passage, into which a loop of intestine may descend, either soon after birth, or at any subsequent period of life. This constitutes a congenital hernia.

Funicular Hernia. Sometimes the part of one or both vaginal processes of peritoneum becomes obliterated below, *i.e.* just above the tunica vaginalis, and remains open above. If bowel descends into the patent part of the funicular process the condition is termed a funicular hernia.

Infantile or Encysted Hernia. If the obliteration of the funicular[1] part of the vaginal process be begun but not completed, then the obliterated part may be stretched by the pressure of intestine from above and pushed into the funicular part of the process. This is one variety of infantile hernia.[2] Or

[1] That is the part that lies in front of the spermatic cord or funis.

[2] That this form of hernia is very rare has been shown by Lockwood. The possibility of its occurrence is shown by a hernial sac removed by E. Owen in doing the radical operation and examined by myself. In this instance the funicular process was closed at its upper and lower extremities, the intervening part was filled with fluid; in other words, there was an encysted hydrocele of the cord. The hernia had pushed down the thin central part of the septum between the peritoneum and the hydrocele into the cavity of the latter. Had the hydrocele not been shut off from the tunica vaginalis the condition would have been that shown in Fig. 108, 3.

a fresh hernia may form behind a patent vaginal process; this constitutes the second variety of infantile hernia.

When the testis is arrested in the inguinal canal it is accompanied by a vaginal process which usually remains patent and forms the foundation of a congenital hernia in the inguinal canal. The term "bubonocele" is applied to an inguinal hernia which does not pass beyond the inguinal canal.

In females there may be a congenital bubonocele from the patency of the process of peritoneum (canal of Nuck), which accompanies the round ligaments. Sometimes in such cases the ovary is prolapsed as a hernia into the inguinal canal.

Diaphragmatic Hernia. A third variety of congenital hernia is produced by the non-development of part of the left half of the diaphragm, through which there is a gap of communication between the abdomen and left pleura. The growth of the abdominal contents being more marked before birth than that of the lung the result is that the stomach is pushed up into the pleural cavity. There is no sac in this form of diaphragmatic hernia; this also holds good for traumatic diaphragmatic hernia in which forcible pressure on the abdomen causes the diaphragm to rupture and part of the stomach to prolapse through the rent.

Internal Hernia may be a congenital or an acquired condition. In the mesenteric retroperitoneal hernia the whole of the small intestine is behind the part of the mesentery which lies between the superior mesenteric vessels and the attachment of the mesentery to the posterior of the wall of the abdomen.

Another instance of the congenital internal hernia is the sigmoid variety of retroperitoneal hernia. In this condition the small intestine is packed into what is normally a minute fossa on the outer aspect of the sigmoid mesocolon. These conditions do not necessarily entail any obstruction of the bowel. Acquired or accidental internal hernia will be dealt with more fully under "Intestinal Obstruction."

Interstitial Hernia. Pouch-like diverticula are commonly formed from hernial sacs owing to the pressure of the intestine. When such pouches force their way between the layers of the abdominal wall the condition is known as "interstitial hernia," or *hernie en bissac.*

Classification of Herniæ. It will have been gathered from what has been said on the subject that there is no one method of classifying herniæ. The topographical classification is the most useful. Thus umbilical and ventral; inguinal (oblique or indirect and direct); femoral and obturator, are the commoner varieties. The first may be subdivided into congenital and acquired; so may the oblique inguinal.

Hernia of the Cæcum. When there is a long mesocæcum the cæcum may protrude freely in a sac like other parts of the intestine. But occasionally in operating on a right inguinal hernia it is found that the peritoneum does not cover the posterior part of the herniated cæcum. In operating on such a case the lax tissue behind the ascending colon, and also part of the sac, has to be pushed up into the abdominal cavity with the bowel.

Femoral Hernia is a protrusion through the crural ring, and along the short crural canal at the top of the thigh. The neck of the sac is thus placed internal to the femoral vessels behind Poupart's and external to Gimbernat's ligaments. Hernia in this situation is commoner in women than men. Rarely a hernial protrusion is observed under the outer part of Pouparts' ligaments, and external to the femoral vessels.

Obturator Hernia protrudes through the foramen, between the upper or free edge of the obturator membrane and the horizontal ramus of the pubes. The sac lies in front of the obturator externus, under cover of the pectineus and the femoral vein. The superficial branch of the obturator nerve is stretched over the inner side of the sac, and this fact explains the occurrence of pain along the inside of the thigh and knee in such cases.

Rarer Forms of Hernia.

Perineal Hernia occurs in men between the bladder and rectum, in women between the vagina and rectum.

Vaginal Hernia projects into the upper and posterior wall of the vagina.

Pudendal Hernia projects along the side of the vagina and into the lower part of a labium majus.

Sciatic Hernia occurs through the great sacro-sciatic foramen, at the lower border of the pyriformis muscle.

Causes of Hernia. Some of the causes have already been alluded to: *e.g.* congenital defects, traumatism, etc. Over-fatness is another potent factor. A fat omentum and mesentery, etc., increase the bulk of the abdominal contents, stretch the muscles, and weaken the hernial apertures, which then allow pouches of the peritoneum, pressed by the over-bulky adominal viscera, to pass through and beyond them. If a person from being very fat becomes thin, the formation of hernia is rendered still more easy. A chronic cough, chronic constipation, or any condition associated with frequently repeated forced expirations, by increasing the internal abdominal pressure, tends to the same end. The abdominal wall may be weakened by operations and the scars may yield, giving rise to hernia. One cause deserves particular mention. This is the formation of a local growth of fat, *i.e.* a lipoma in part of the subperitoneal tissue. The lipoma stretches and pushes before it the muscular or aponeurotic structures of the abdominal wall, the peritoneum bulges into the lipoma, and a tumour which looks like an ordinary lipoma, but which contains bowel, is thus formed.

CHAPTER XVIII.

INTESTINAL OBSTRUCTION AND PERITONITIS.

An ordinary case of strangulated hernia may be taken as the type of acute intestinal obstruction, and one of general purulent peritonitis from perforation of the bowel as the type of the worst form of surgical peritonitis. Every student will have an opportunity of studying such cases and of learning the dread import of the terms that head this chapter. Between the extremes presented by these types come a series of conditions which entail a more gradual clinical course. Such are cases of chronic intestinal obstruction, and cases of localized peritonitis: *e.g.* cases of stricture from cancerous or tubercular ulceration of the bowel, and cases of relapsing appendicitis.

Intestinal Obstruction, from what is called *strangulation* of a loop of bowel, is due to pressure occluding the passage through the two ends of the loop. The effects of strangulation may be now considered, since they include the changes that occur in a strangulated hernia, and also the effects common to all cases of acute intestinal obstruction.

The effects of strangulation of a loop of intestine, whether from nipping by the neck of the sac of an external or internal hernia, or the constriction of a peritonitic band, or that of the bowel itself in intussusception and volvulus, are as follows:

In the first place the veins feel the effects of pressure before the arteries, therefore the affected loop of bowel becomes *congested* and *swollen*. Owing to the congestion, exudation of serum occurs, as is seen in the collection of fluid in the sac in cases of strangulated hernia.

An excess of fluid is also poured out from the congested mucous membrane. If the constriction remains unrelieved *hæmorrhage* into the coats of the bowel ensues. Thus in a

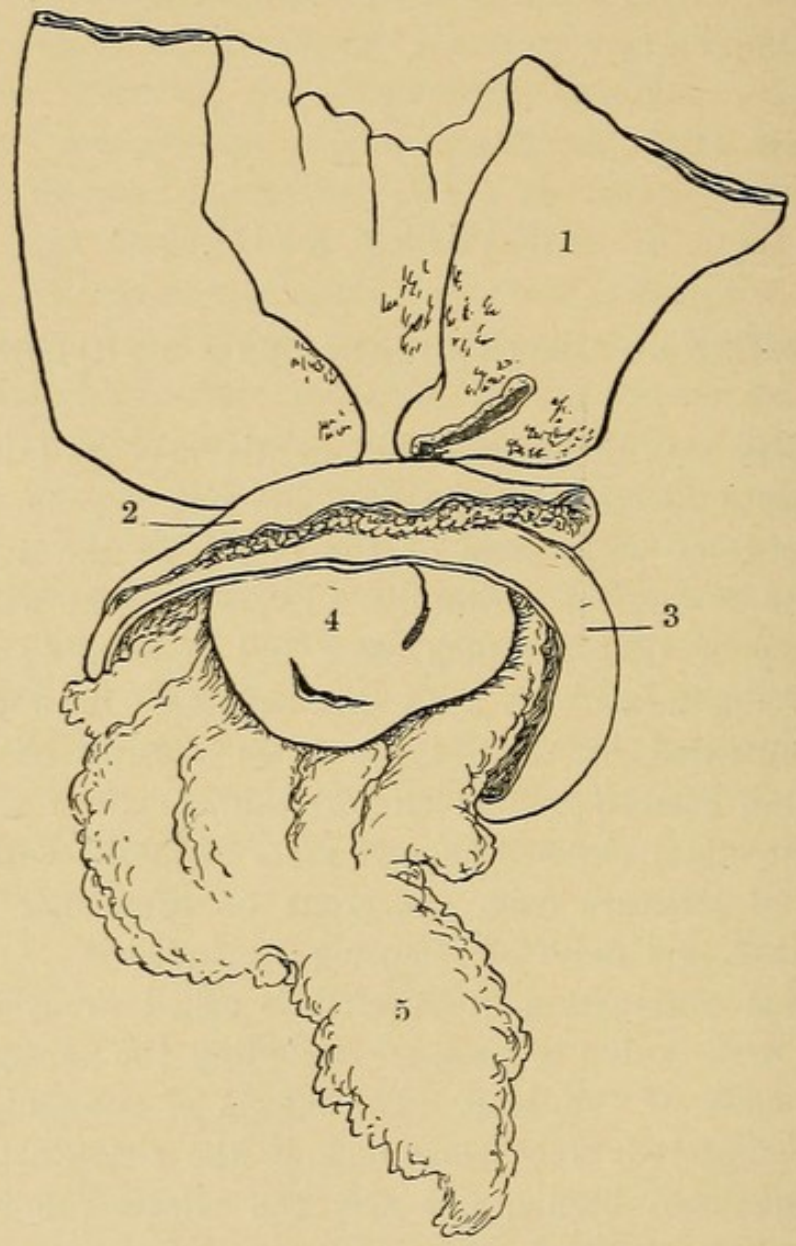


FIG. 108.—A strangulated hernia. 1, The upper portion of bowel above the protrusion. It has been ulcerated through its whole circumference from pressure of the constricting ring. 2, Portion of the peritoneum; 3, another portion of peritoneum forming the sac of the hernia; 4, protruded knuckle of bowel, ruptured (probably by taxis); 5, omentum in the hernia. From a man aged 73 who had constipation for nine, and stercoraceous vomiting for two days before death. (959.)

severely strangulated hernia the fluid in the sac becomes blood-stained. Later, the surface of the strangulated loop becomes *inflamed*, *i.e.* becomes covered by a layer of plastic lymph. Finally, if the pressure on the extremities of the loop continues, *necrosis* ensues. This is most commonly observed at the ex-

tremities of the loop,[1] where the tissues of the gut are directly pressed upon by the constricting ring (see Fig. 108). Finally, gangrene of the whole of the strangulated loop may occur, either from the arteries of the loop being occluded by pressure or from septic inflammation of the intensely congested loop of bowel. It cannot be too well known that gangrene from the latter cause may occur after the constricting ring has been divided and the loop of bowel returned into the abdominal cavity. The entire loop may become gangrenous.

Fig. 108 shows what may be termed a natural termination of a case of strangulated hernia, combined with an effect of injudicious *taxis*, *i.e.* an attempt to press the protruded loop back through the orifice into the abdominal cavity.

Besides rupture of the bowel, taxis may result in other disasters, *e.g.* (1) the sac may be reduced into the abdomen with the hernia, and if the constriction is caused, as is common, by the neck of the sac, symptoms of strangulation will persist; this is called *reduction in mass.* (2) The bowel may be reduced in a gangrenous or in an ulcerated condition and cause fatal peritonitis. (3) The manipulations may so damage the bowel that it becomes intensely inflamed or even gangrenous after reduction.

Taxis, if successful, may cause an opportunity of doing a radical operation to be missed.

Changes in the Bowel above the Seat of Constriction. The obstruction to the onward passage of fæces and gas is met by increased peristalsis, which results in an onward current at the sides of the intestine, and in the middle a reflux current which starts from the seat of obstruction, and constitutes the chief factor in producing vomiting.[2]

[1] Hence in operating on a strangulated hernia after the sac has been opened and the constricting tissues divided, it is necessary to draw down the loop to see whether there is any evidence of ulceration at its extremities. Frequently, though there is no actual perforation, a condition which is practically as serious is present: the mucous and muscular coats have been ulcerated through and only the necrotic fibrous coat remains at the seat of constriction.

[2] This is Brinton's explanation as opposed to the old idea of "reversed peristalsis."

The bowel above the seat of obstruction becomes weakened by fatigue and dilates under the pressure of fæces, gas, and fluid, secreted by the mucous membrane. Finally, a certain amount of peritonitis develops from the absorption of irritating soluble matter into the intestinal tissues. These pathological changes determine *subjective symptoms*, of which the chief are as follows:

1. *Pain.* The congestion, etc., of the strangulated loop causes pain which may in external hernia be referred by the patient to the hernia itself, or to the region of the umbilicus. The sac becomes more tense from swelling of the herniated structures. *Paroxysmal or colicky pain* is a marked feature in many cases of acute and subacute obstruction. It is due to irregular and violent peristalsis.

2. *Vomiting.* This is in part due to the regurgitant current mentioned above, and is in part reflex, as is shown by the presence of vomiting in cases of hernia where only part of the circumference of the bowel is protruded through the ring.[1] The profuse vomiting that ensues when the lower part of the duodenum or the upper part of the jejunum is constricted shows not only that reflex nerve action has an important share in the production of vomiting, but also that through the same influence an abundant secretion from the mucous membrane occurs.

It should also be borne in mind that reflex vomiting may be observed quite independently of intestinal obstruction in cases of crushed testicle and congestion of the testis from torsion of the cord, etc.

Constipation is also of dual causation, being due in part to the occlusion of the lumen of the bowel and in part reflex. Like the vomiting, complete constipation is observed in some cases of "partial enterocele."

Tympanites, or distension of the abdomen, is due to the dilatation of the bowel above the obstruction. It is accompanied by shock, *i.e.* weak and rapid heart-action, shallow respiration, and a drawn and anxious expression of countenance. The shock is partly reflex and partly due to the *upward displacementof the diaphragm*, which may be as high as the third

[1] This is termed "partial enterocele," and also "Richter's Hernia." Littré's hernia is strangulation of a Meckel's diverticulum.

intercostal spaces, and entails collapse of the lower parts of the lungs and displacement of the heart. The occurrence of *Gangrene* may be marked by hiccough and cessation of pain.

Chronic Intestinal Obstruction. When an obstruction of the bowels is incomplete, and the condition lasts for a long time, changes occur in the bowel above the block: the muscular coats hypertrophy, and the lumen of the bowel dilates. Since the patient wastes from the interference with nutrition caused by disorder, the movements of the hypertrophied and dilated bowel can be seen through the abdominal wall. This is termed *visible peristalsis.* Later, from the pressure and irritation of retained fæces, *ulceration* of the bowel is common.

Peritonitis.

In order to illustrate the chief practical features of suppurative peritonitis, the worst form of suppurative peritonitis that results from perforation of the vermiform process may be taken. A sudden pain is felt and may be correctly referred to the right iliac region; the pain increases and spreads over the abdomen, which becomes tender and tympanitic, causing the knees to be drawn up; vomiting and constipation set in. Death follows in a few hours or days. The autopsy reveals pus in every part of the peritoneum, and much distension of the bowel, especially of the small intestine. The diaphragm may be displaced up to the level of the third intercostal spaces; the pelvis is tightly filled with coils of small intestine, distended with gas. The cæcum and appendix are covered by large coils of intestine, and, on moving the latter aside and wiping away the pus, the swollen and injected appendix is found to present a sloughy aperture in which sometimes a fæcal concretion is found. Comparing the symptoms with those of a case of obstruction it is noted that the temperature is usually elevated, the abdominal tenderness is greater, the pain more continuous and less paroxysmal. The normal *diaphragmatic respiratory movements* are absent, and when the diaphragm is reached by the suppurative process and rendered paralytic, may be actually *reversed*—the abdomen receding in inspiration.

In the earlier part of the course of the affection the vomiting is purely reflex from irritation of the bowel and peritoneum, but when the suppurative process is advanced the bowel is paralyzed and unable to contract in peristalsis, hence unable to force along the contents of the bowel. This paralysis of the bowel causes obstruction as complete as that caused by strangulation and hence gives rise to the symptoms of obstruction, chief among which are absolute constipation and fæcal vomiting. That acute peritonitis is a cause of obstruction must never be left out of sight. When fever is not marked it is often a matter of the greatest difficulty to decide whether in a given case the primary disturbance is peritonitis or mechanical obstruction. Having thus briefly reviewed the more salient features of cases primarily of obstruction on the one hand, and cases primarily of peritonitis on the other hand, the causes of these conditions may be considered in detail.

The Chief Modes of Intestinal Obstruction.

CLASS I. By pressure from without.

1. By a loop of intestine passing through an orifice as in external and internal hernia.
2. By bands such as long adhesions left from peritonitis or an adherent Meckel's diverticulum.
3. By kinking, from the intestine dragging on a fixed point.
4. Intussusception.
5. Volvulus.
6. By compression of tumours, etc., outside the bowel.

CLASS II. By changes in the wall of the gut.

7. Acute inflammatory paralysis, peritonitis.
8. Contraction of scars, or tubercular ulcers.
9. Cancerous growth.

CLASS III. By free bodies within the bowel.

10. Impacted fæces.
11. Foreign bodies, *e.g.* sets of false teeth.
12. Gall-stones.
13. Concretions, *e.g.* of magnesia.

Combinations of these modes are not uncommon. Thus there may be a moderate narrowing of the bowel caused by the cicatrization of a tubercular ulcer, and yet no symptoms may arise until some hard body such as a plum stone lodges in and blocks the strictured part. Cases of obstruction from congenital defects have been dealt with on p. 140.

Strangulation by Passage through Apertures. The varieties of *external hernia* have been described at some length (see pp. 256-260). Some of them, such as the obturator variety, cause but little swelling externally, even a small femoral hernia in a fat woman may be overlooked. In some cases the marked symptoms of intestinal obstruction will lead to an abdominal section being made, and then on systematic examination of the intestines being carried out, the condition will be discovered.

Internal Hernia. Besides the two forms previously described, other displacements of bowel through normal or abnormal apertures within the abdomen occur. Thus intestine may pass through the foramen of Winslow, or into the fossa duodeno-jejunalis. More commonly the aperture is an abnormal one in the omentum or the mesentery.

Strangulation by Bands. Peritoneal bands are either fibrous adhesions resulting from previous peritonitis or parts of the intestine itself, such as the vermiform process or Meckel's diverticulum, which have become adherent at what should be their free extremities. Obstruction may be brought about in several ways. Sometimes the band is wrapped like a cord round a loop of bowel, sometimes it is simply stretched tightly over the loop. The constant motion of the intestines within the abdominal cavity has only to be remembered to explain the various ways in which bands may produce obstruction. A case in which an adherent Meckel's diverticulum caused fatal obstruction in a child aged two years is represented in Fig. 109.

Kinking is one way in which bands or other adhesions produce obstruction. A point, say, in the middle of the small intestine is adherent to the under surface of the transverse mesocolon. Then if the intestine above the adherent spot should fill with fluid more rapidly than usual and be dragged

by weight towards the pelvis, there may be such a sudden bend or kink at the adherent spot that the lumen is obliterated.

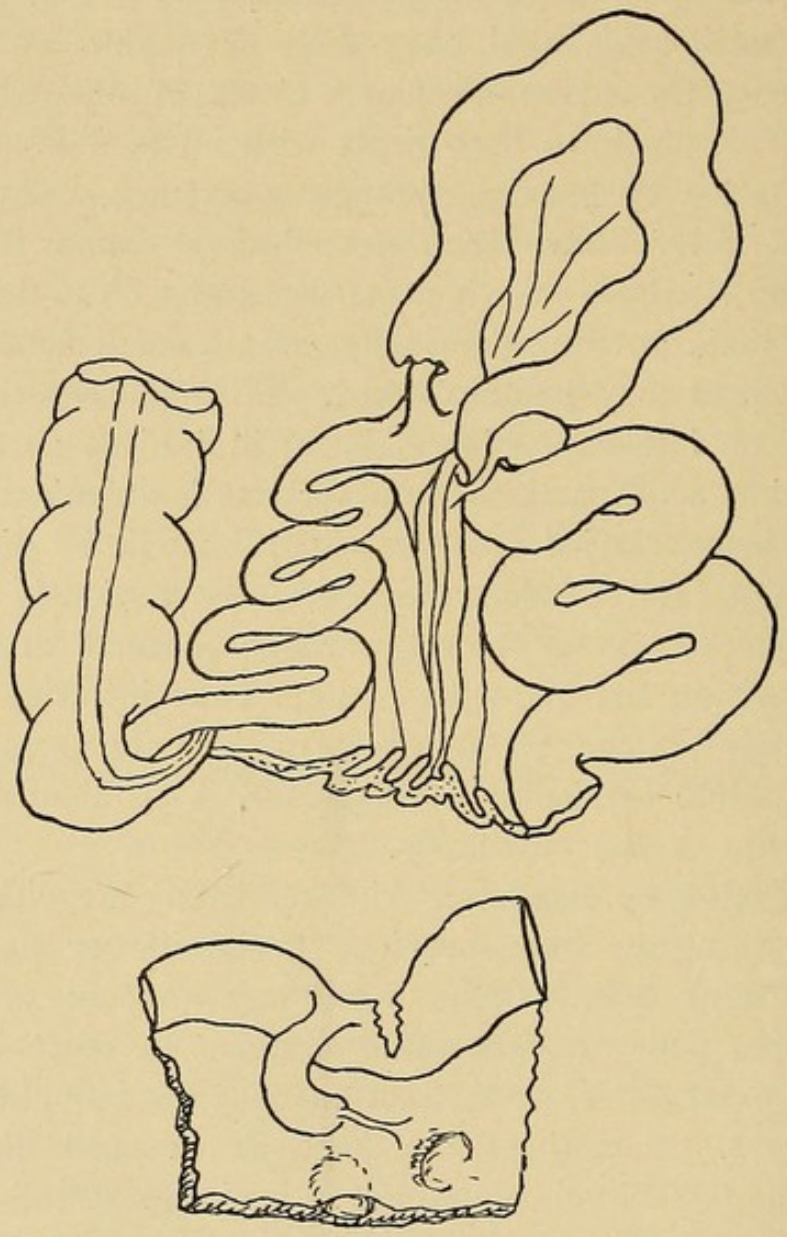


Fig. 109.—The cæcum, etc., and part of the small intestine as found on opening the abdominal cavity after death. A loop of ileum has been protruded through the arch formed by the adhesion of the diverticulum to the mesentery as shown in the lower sketch, which represents the bowel in the neighbourhood of the attachment of the diverticulum. In the mesentery are two tubercular glands which gave rise to peritonitis which caused the adhesion. The bowel is ulcerated half-way through owing to pressure of the constricting diverticulum. From a child aged two years, who died five days after symptoms set in. (935 A.)

Intussusception. This term signifies the prolapse of a part of the intestine into the interior of the portion next below or above it. The commonest situation for this displacement to occur is at the ileo-cæcal valve. In by far the greater majority of cases the valve forms the apex of the prolapsed portion

constituting the ileo-cæcal intussusception. When once begun the extent of the prolapse tends to be increased by the action of the colon grasping and forcing onward the prolapsed or intussuscepted part. An ileo-cæcal intussusception increases at the expense of the outer layer or *sheath*, the ileo-cæcal valve remaining as the apex of the intussusception of which the innermost part or *entering layer* is constituted by the last part of the ileum, and the outer part of the invaginated bowel (*returning layer*) is composed of the upper part of the colon. Between the entering and returning layers are placed the part of the mesentery of the implicated part of the ileum, the vermiform appendix, etc.

Intussusception may, however, occur at any part of the large or small intestine. The corresponding process at the lowest part of the rectum is prolapse (or *procidentia recti*) when the prolapsed portion escapes from the anus. In a few instances in the ileo-cæcal region, instead of the ileo-cæcal valve forming the apex of the intussusception, successive portions of the ileum are prolapsed through the valve. In this case the condition is termed an "ileo-colic intussusception." This variety forms an exception to the rule that an intussusception grows at the expense of its sheath.

In another condition, mixed ileo-colic and ileo-cæcal intussusception, the displacement begins as an ileo-cæcal, and is continued as an ileo-colic intussusception.

Figs. 110 and 111 are taken from a fatal case of ileo-cæcal intussusception.

The Causes of Intussusception. In some cases a definite cause can be found. For instance, a tumour growing from the mucous aspect of the bowel has with the part of the bowel to which it is attached been drawn into the bowel below it by peristalsis, or a part of the intestine narrowed, say by a cancerous stricture, is forced by the pressure of fæces a certain distance into the bowel below it, so forming the beginning of an intussusception. In the majority of cases no obvious cause can be observed. Some light may be found in the frequent occurrence of the intussusceptions which are found in the death-agony, and are so commonly seen in the post-

mortem room. They cause no symptoms and take no part in causing death, but they are due to irregular action of the bowel stimulated by the excess of CO_2 present in the blood when the respiration and circulation are failing in death. Indeed, Grieg Smith has suggested that the attack of colicky pain

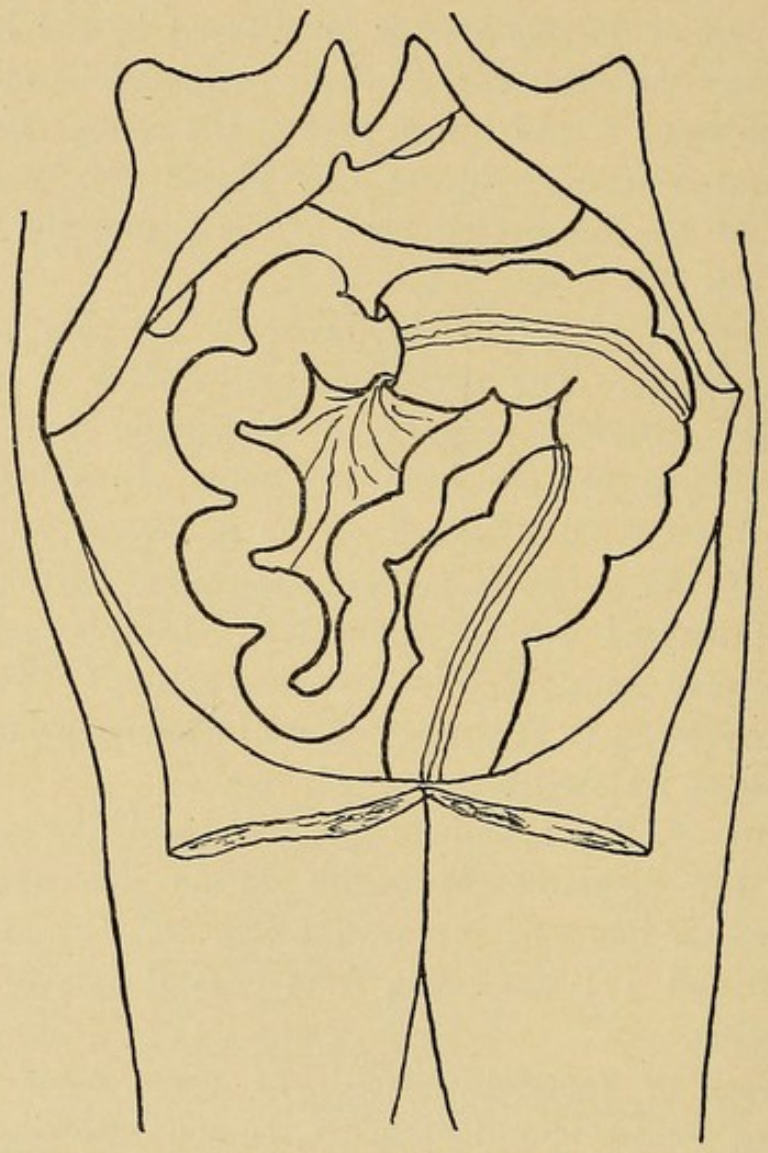


FIG. 110.—The abdominal cavity of a child opened. The cæcum, vermiform appendix, the ascending and half the transverse colon, with a corresponding length of the ileum, have disappeared, having been intussuscepted within the lower part of the colon. Diagrammatic.

experienced by children and others when indigestible substances, such as the rind of apples, have been swallowed, may be due to slight intussusceptions which right themselves.

The course of anatomically similar cases varies greatly, and is accounted for only by variations in nervous susceptibility in different patients. The fatal issue may occur within twenty-four

hours, or it may be delayed over a month. When the invagination is once begun it has the same effect on the bowel as an unusually large mass of undigested matter, *i.e.* it excites powerful intermittent peristalsis, which gives rise to the leading symptom of the first stage of the affection, *violent paroxysmal pain*, and produces pathological changes in the invaginated part of the bowel. To realize these changes it must be remembered, in the first place, that with the intestine a certain amount of mesentery is dragged in with the intussuscepted portion of the bowel. How great this traction may become is best realized by considering the condition of the mesentery in a case such as is depicted in Fig. 111, where between the entering and returning layers lie the mesentery corresponding to the invaginated portion of small intestine, and the stretched out peritoneal covering of the ascending and of half the transverse colon, together with the vermiform appendix, and some of the great omentum. The first result of this stretching of and pressure on the mesentery is compression of the mesenteric veins. This leads to congestion and œdema and swelling of the intussusception, together with excessive mucous secretion and oozing of blood and serum from its mucous aspect. These events mark the second stage of the case: *tenesmus*, with discharge of blood and mucus from the anus. To this succeeds the final stage, that of *complete obstruction*, in which the symptoms are the same as those seen in obstruction from other causes, constipation, vomiting, etc. In some instances the intussuscepted part has separated by sloughing, and in a few of these cases recovery has followed, the peritoneal surfaces of entering and returning layers having become united by lymph and granulation tissue at the base of the intussusception. The congestion has thus an important

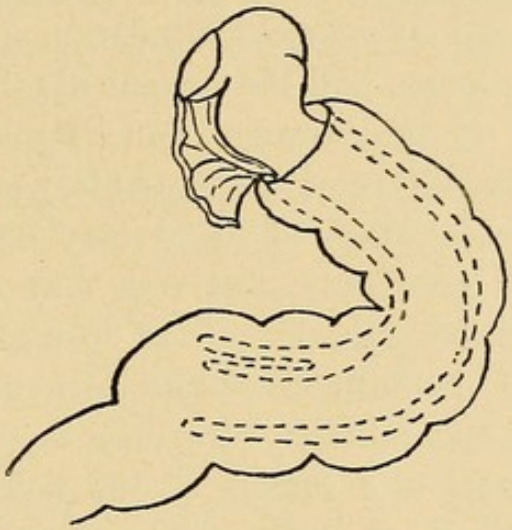


FIG. 111.—Shows by dotted lines the different layers of bowel in an ileo-cæcal intussusception.

result, that is, the formation of plastic lymph between the peritoneal surfaces of the two inner layers. This generally commences near the apex. Such a condition renders the last inch or so of an intussusception irreducible save by laparotomy, and accounts for cases in which the displacement, having apparently been overcome by inflation and manipulation, has immediately recurred and the patient has died unrelieved.

If the strangulation is severe, hæmorrhage occurs between the layers of the mesentery; and when it is still more marked, actual stasis of blood may occur in the intussusception, which, as previously observed, may slough off; and if in such a condition adhesions have formed between the peritoneal surfaces of the intussusception to a point above the line of separation, a spontaneous cure may be the result. When the swelling is great the lumen of the bowel becomes entirely blocked, and the third stage of the clinical aspect—the stage of obstruction—is established and is marked by absolute constipation and distension of the abdomen.

Looked at generally, the indications for treatment are some thing the same as those of strangulated hernia. In the earlier stages inflation (which may be compared with taxis as applied to hernia) from the rectum under anæsthesia, combined with manipulation, may succeed. Later it is better to do abdominal section so that the exact condition of the parts may be ascertained. It must be borne in mind that the displacement may appear to have been overcome when the last inch or so remains unreduced. The converse of this is also true, that is, it may appear from a tumour being felt in the left iliac region that reduction has failed when an abdominal section shows that the tumour is due to swelling and that complete reduction has been effected.

Volvulus. The commonest instance of volvulus is that of the sigmoid flexure. The C-shaped loop is twisted upon its mesenteric axis. Usually the twist occurs by the upper half of the loop falling in front of the lower half; sometimes the twist occurs in the opposite direction. In fatal cases the anatomical changes are striking. Thus, to take a typical example, in an autopsy made on the body of an old man who

was brought into St. Mary's Hospital in a dying state the abdomen was greatly distended (Fig. 112), the prominence of the front of the belly being even more marked than is the case when the distension is due to obstruction in the small intestine, When the usual vertical incision had been made an enormously distended piece of intestine, shown to be a part of the colon

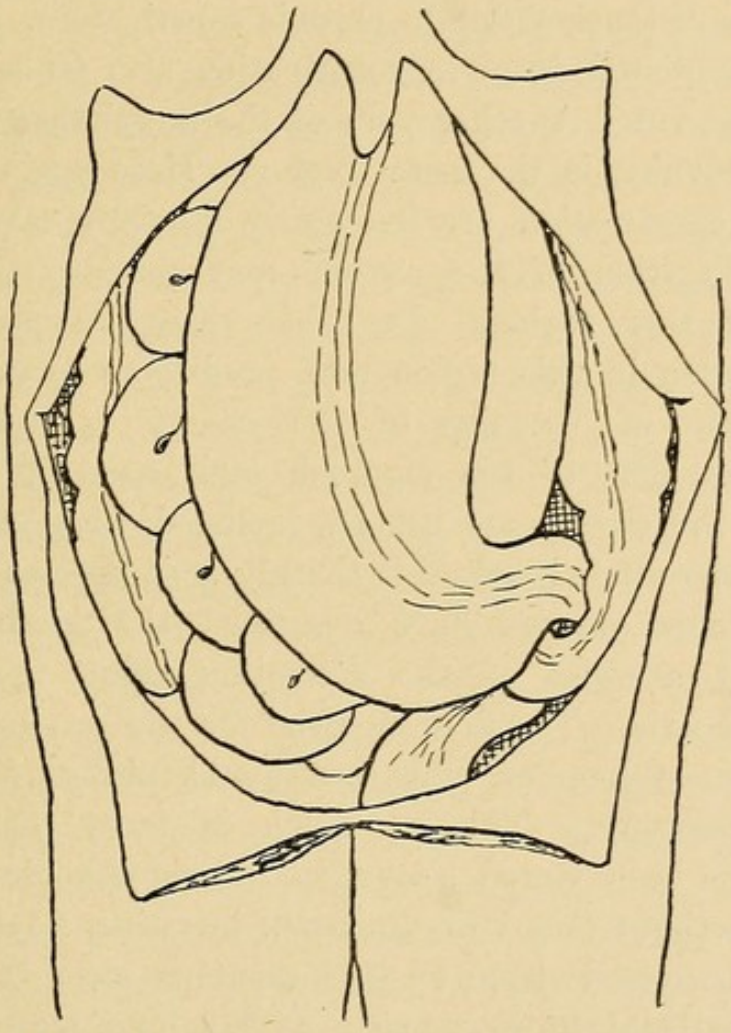


FIG. 112.—The abdomen of an old man opened to show a volvulus of the sigmoid flexure the extremities of which are twisted one round the other. The sigmoid flexure is greatly distended, forming a large loop, the upper end of which is behind the ensiform cartilage.

by a band of longitudinal fibres, was seen to occupy the whole of the front of the abdomen, its upper end lying under the ensiform cartilage and the diaphragm. On withdrawing the distended loop it was found to be the sigmoid flexure which was full of fluid,[1] congested and covered with patches of lymph.

[1] Sometimes solid fæces, and more frequently gas, are mingled with the liquid exudation in the dilated loop.

The strain on the upper extremity of the loop had been so great that a small perforation had formed there. The small intestine and the colon above the twist were also distended, so that the diaphragm was pushed up to the level of the fourth costal cartilages, the lower lobes of the lungs being collapsed to a considerable extent.

Causation. The predisposing cause of volvulus of the sigmoid is found in distension due to chronic constipation. The weight of the accumulated fæces in constipation also tends to produce the displacement. Another part of the bowel that is especially prone to volvulus is the cæcal region. Here again the effects of chronic constipation are frequently specially marked. Such marked elongation of the cæcum may prepare the way for volvulus of this region. In other cases the presence of a long mesocolon in this region may predispose to the condition. The anatomy of volvulus of the cæcum is not so clearly described as that of the sigmoid, and cases of *kinking* (see p. 267) of the cæcum are usually included with volvulus. In such cases the dilated cæcum is sharply bent upwards, producing a kink. Some authors have described the production of a volvulus by a twist of the ascending colon upon its own longitudinal axis. The first essential for the production of volvulus is that a loop of intestine should possess an unusually lengthy mesentery. This condition is most commonly met with, as has been stated above, in the sigmoid flexure, but it also is sometimes found in the small intestine, a loop of which may then become twisted on its mesenteric axis. When such a loop of small intestine co-exists with a long meso-sigmoid or meso-cæcum, a volvulus may be formed by the intertwining of the neck of a loop of small intestine with that of the sigmoid or the cæcum, or two loops of small intestine may be intertwined in a similar way.

Above all it must be remembered in practice that volvulus of the sigmoid is the commonest, and that in such cases the onset is usually as sudden and the symptoms as severe as they are in any case of intestinal obstruction. Some of the conditions ranked as volvulus of the cæcum have run an acute, others a subacute or chronic course. A similar variability has been

observed in cases of volvulus of the small intestine. Speaking of a chronic case Treves has observed: "The patient had no less than seven severe attacks of obstruction in a little more than twelve months. These attacks were somewhat sudden in their onset and associated with constipation, vomiting, and severe pain. They were relieved as a rule at the end of a few days by means of enemata, the patient recovering often very slowly. Here the volvulus was in the upper part of the jejunum. The case suggests the possibility of spontaneous cure in cases of volvulus, and it may be that several of the examples of acute intestinal obstruction that have either ceased spontaneously or yielded to some nostrum, have been instances of volvulus of the lesser bowel."[1] In this connection the effects of chronic constipation upon the large intestine must be remembered. In one post mortem I found the cæcum to be distended, thinned, and its length increased to 8 inches. The rest of the large intestine was covered with small pouch-like diverticula; the sigmoid flexure was also dilated.

Compression of the Bowel. Clinically closely resembling the effects of stricture are those which result from compression by tumours, etc., pressing on the bowel from without. The rectum is most commonly affected in this way owing to the frequency with which tumours arise in female pelvic viscera and the narrow space of the pelvis; any part of the intestine may however be similarly narrowed or occluded, and a variety of conditions, such as abdominal tumours or hydatids, tumours of the ovaries and uterus, may be found to have caused the pressure.

Stricture of the Intestine. Narrowing of the lumen of the bowel when due to changes in the wall of the gut is termed a stricture. Such a condition may be congenital or may arise from cicatricial contraction following dysenteric, tubercular, syphilitic or other ulceration; or from changes that have occurred in a loop of bowel that has been strangulated in a hernia: changes due either to *traction* and *pressure* of the neck of the sac on the extremities of the loop, or ulceration, etc., in the bowel itself. The spontaneous separation of an intussusception or an operation for the uniting of two portions

[1] *Intestinal Obstruction*, 1884, p. 163.

of bowel end to end, or the contraction of a tubercular ulcer, may leave a stricture. Perhaps the most important form of stricture is that which is due to the narrowing of the bowel by cancer (see Figs. 113 and 114).

With regard to cancer it must be borne in mind that, as in all forms of cancer, the degree of tumour-formation and the amount of stricture from cicatricial contraction due to the infiltration and cicatrization of the bowel wall vary within wide limits. If the latter processes predominate the growth is said to be of the *constricting* variety; if the former, of the *proliferating*. Other tumours of the bowel may cause obstruction: such are, *adenomata*, which are more commonly pedunculated and constitute the commonest form of "polypi"; but sometimes they are sessile, and then may spread laterally, and closely approach the carcinomata in character. Fibromata, myomata, lipomata, and sarcomata are all occasionally encountered: among the latter, multiple nodules of lymphosarcoma are commonest.

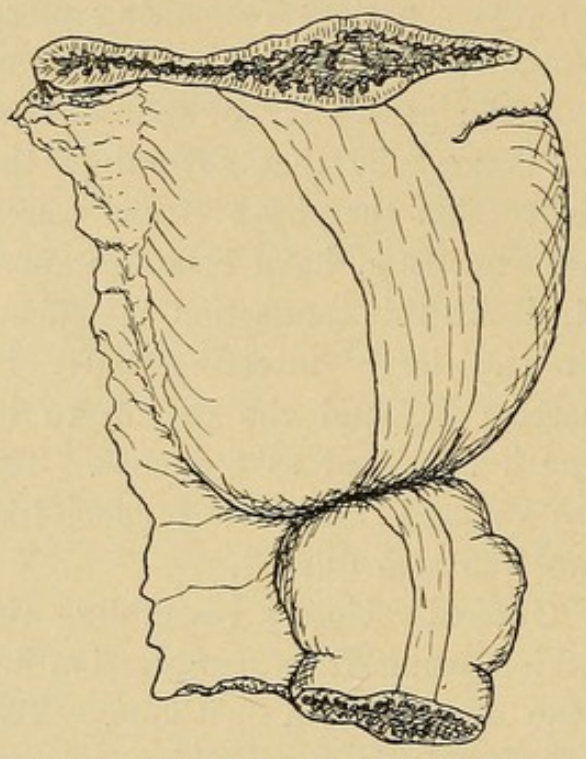


FIG. 113.—Cancer of the intestine. The outer aspect of the bowel showing the constriction caused by the growth.

Both in stricture and compression from without the symptoms may be acute, but especially in cases of stricture there is a chronic condition of obstruction which determines important pathological changes in the bowel, which becomes greatly dilated and hypertrophied above the narrowed part. These changes are often recognizable during life, for the emaciation of the patient consequent upon the interference with alimentation permits the movements of the hypertrophied bowel to be seen (*visible peristalsis*). When the partial gives place to complete obstruction the bowel rapidly dilates and the move-

ments of the bowel cease. Besides hypertrophy and dilatation, chronic obstruction may lead to ulceration of the bowel, owing to the pressure and irritation of retained fæces. This ulceration may occur at the upper limit of the obstructing lesion, but more frequently it takes place at a distance. Thus a cancer of the sigmoid often leads to a perforation of

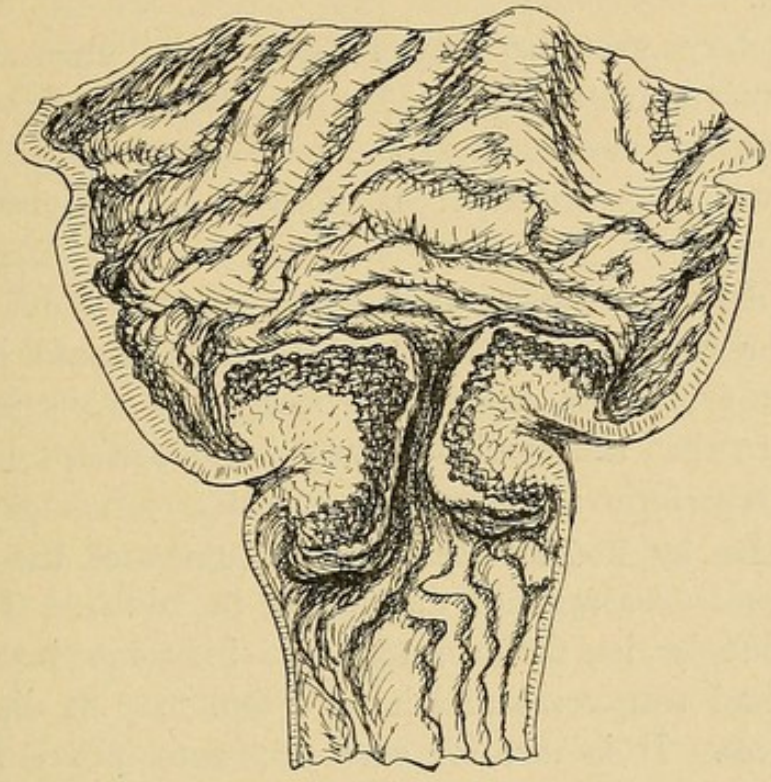


FIG. 114.—The bowel opened by cutting through the growth which has infiltrated the whole thickness of the bowel-wall.

the cæcum. In some cases, as ulceration is taking place adhesions form on the peritoneal aspect of the bowel. Some of *the natural modes of termination of a case of cancer of the intestine* are as follows:

1. Ulceration and perforation of the bowel above the stricture. The perforation may form either close above the growth or at a distance from it: thus in a case of cancer of the rectum the perforation may form in the cæcum. If the perforation is into the peritoneal cavity a large quantity of dammed up fæces and gas escape, causing rapid death. The perforation may be retroperitoneal, causing retroperitoneal fæcal abscess. In a few cases I have found the ulceration to be of the type of "ulcera-

tive colitis," *i.e.* innumerable small ulcers occupy the whole of the intestine above the stricture.

2. Acute may supervene on chronic obstruction and colotomy be required.

3. The growth may extend to other parts. Thus in cancer of the transverse colon a communication with the stomach may form, giving rise to fæcal vomiting which is independent of obstruction.

4. Secondary growths may form in the liver and other organs, causing death.

5. Gradual enfeeblement of the patient's strength may cause death either from intercurrent disease or by gradual heart failure.

Contemplation of a series of cases of cancer of the colon leads to the conclusion that great attention should be paid to the earlier symptoms, such as constipation alternating with diarrhœa, so that the condition may be recognized before it is too late to perform a radical operation.

Obstruction by Bodies Free in the Lumen of the Intestine. *Fæcal accumulations* are not uncommon in adults. They occur most commonly in the cæcum, but in some instances the ascending and transverse and lower colon may be distended by doughy fæces. It is rare for such cases to prove fatal from obstruction, but ulceration of the bowel, peritonitis, or perforation may occur from the presence of masses of stale fæces. In lunatics masses of hair, etc., may be found. Excessive doses of magnesia continued for a long time have been known to cause concretions in the intestine.

Large gall-stones which have found their way by ulceration from the gall-bladder into the duodenum are not infrequently the cause of fatal obstruction.

Obstruction by Foreign Bodies and Concretions. Large bodies, such as the plates of false teeth, are sometimes accidentally swallowed, and though remarkably large bodies often pass along the alimentary canal without causing pronounced symptoms, they may give rise to symptoms of obstruction which in some cases proves fatal. In children pieces of cockle-shell and similar objects are sometimes found fixed in the

rectum above the sphincters when an examination is made to ascertain the cause of obstinate constipation.

Peritonitis.

Inflammation of the peritoneum, which, like the pleuræ and other serous cavities may be regarded as a large lymph-space, may be of several kinds. The most important of these is *suppurative peritonitis.* Chronic plastic peritonitis may occur by extension of inflammation through the coats of the stomach or intestine in ulceration of these parts, or it may be due to inflammation about a gall-bladder, distended with stones, or to inflammation of the ovaries or other parts of the female generative organs, or to chronic cystitis, etc. Whenever the abdominal viscera are in a condition of chronic venous congestion, whether from cirrhosis of the liver, or disease of the heart or lungs, the peritoneum becomes thickened and the seat of formation of little tags and nodules of fibrous tissue. This form of chronic peritonitis is generally accompanied by ascites, or effusion of fluid into the cavity. *Tubercular infection* of the peritoneum is not uncommon, and it is important to remember that more or less ascites is usually present in the earlier stages of the disease, and it is during this stage that benefit is obtained from drainage.

Cancerous and Sarcomatous Infection of the Peritoneum is also not uncommon, and has been termed cancerous peritonitis. This affection is also accompanied by ascites, the fluid containing clusters of cells of the growth.

Suppurative Peritonitis is frequently due to perforation of an ulcer of the stomach or intestine, or to gangrene of the bowel after hernia, or other forms of direct spread of local septic infection. Septic thrombosis of the portal vein (*suppurative pylephlebitis*) may also cause suppurative peritonitis. Occasionally the peritoneum is infected by cocci contained in the blood, just as may happen in bones in acute osteomyelitis. Such cases of suppurative peritonitis are termed crypto genetic (hidden-origin). For practical purposes it is of the first importance to understand the chief forms of perforative peritonitis.

Appendicitis and Perforation of the Vermiform Appendix. One of the most common causes of rapid death is peritonitis from perforative ulceration of the vermiform appendix. In order to understand the frequency of ulceration in this part, compared with the rest of the intestine, and also to grasp the varying effects of perforation; the formation of concretions of fæcal

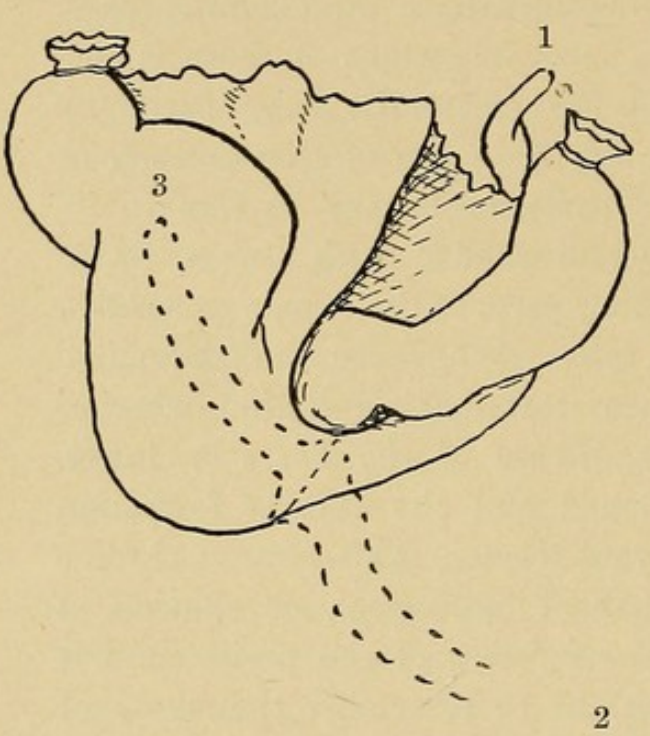


Fig. 115.

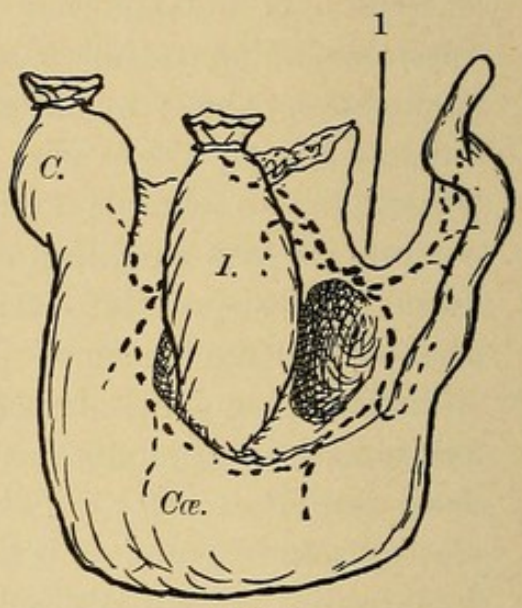


Fig. 116.

Fig. 115.—A front view of the cæcum and neighbouring parts of the intestine of an infant. 1, Marks the tip of the appendix which passes up behind the end of the ileum and the mesentery; 2, marks a common position in which the appendix may either be surrounded by peritoneum and suspended by a mesentery, or it may have the peritoneum stretched over its anterior surface, being then uncovered by peritoneum on three sides; 3, marks an occasional site when the appendix is behind and to the outer side of the ascending colon. In this position it may either be covered by peritoneum, or may be extra-peritoneal.

Fig. 116.—The same preparation seen from the inner (left) side. The dark recess behind the ileum is the inferior, the recess in front of the ileum being the superior ileo-cæcal fossa. The interrupted lines represent the termination of the ileo-colic artery (1) and its branches to the appendix, etc.

matter, or the lodgment of foreign bodies[1] which frequently cause ulceration, the variability in length (3-6 in.) and position must be borne in mind.

[1] These are not so common as once was thought. Fæcal concretions that have become the seat of a deposit of phosphate of magnesia having been mistaken for cherry stones, orange pips, etc. I have, however, found the appendix full of small shot, derived from eating game, and pins and other bodies have been found there.

Perhaps the commonest position is for the appendix to be directed upwards and inwards behind the end of the small intestine (see Fig. 115). It is usually surrounded by peritoneum and suspended by a short mesentery, in which runs its main artery derived from the ileo-colic (see Fig. 116).

Frequently, especially when it is long, the appendix hangs down, and its tip may reach over the pelvic brim (see Fig. 116, 2). In this position it may be surrounded by peritoneum, or simply have the peritoneum lining the abdominal wall stretched over it. Again, instead of being directed upwards and inwards, it may pass from its attachment in an upward and outward direction, lying to the outer side of, or behind, the colon (Fig. 115, 3). In the former case it is entirely surrounded by peritoneum, in the latter it may be entirely devoid of peritoneal covering. The usual shape of the appendix is spiral, or it may be sharply bent. If the appendix is the seat of frequent attacks of catarrhal inflammation, and the opening into the cæcum is blocked, it may undergo *cystic dilatation*. If a succession of non-perforating ulcers form, each attack will be accompanied by local non-suppurative peritonitis, which results in the formation of adhesions around this part of the bowel. In such cases, perforation during one of the attacks frequently leads to suppurative peritonitis, so that in practice the rule is to remove the appendix in a quiet interval following an attack of what is termed *relapsing appendicitis*.

When rapid ulceration and perforation occurs in an appendix wholly covered by peritoneum, local or general, suppurative peritonitis results. By *local suppurative peritonitis* is meant suppuration in a part of the peritoneal cavity, the abscess being shut off from the rest of the peritoneum by lymph and adhesions. Such a local peritoneal abscess from perforative peritonitis forms about the cæcum; frequently it is bounded above by inflamed omentum, and often it extends below into the pelvis, where, in Douglas's pouch in the female, and above the prostate on rectal examination in the male, firm inflammatory lymph may be felt by the finger. There is rigidity, swelling, and tenderness over the cæcum. The acute inflammation paralyzes the end of the ileum, giving rise to constipation and other symptoms of intestinal

obstruction. There is the presence of more or less fever to distinguish the condition from non-inflammatory acute obstruction. The suppuration, after being localized for a time, may extend to the whole peritoneum, or a careless exploratory operation may have the same effect, previously sound parts of the peritoneum being smeared with pus. Again, there may be at no period any local abscess. After the perforation, suppuration may rapidly spread from the neighbourhood of the cæcum to all parts of the abdomen, causing death in a few days or hours.

When perforation takes place in an appendix that has been shut off by adhesions from the general peritoneal cavity, or when the appendix has no peritoneal covering at the perforated spot, a retroperitoneal abscess results. This most frequently shows its presence by œdema in the right iliac region, but where an appendix passes upwards the suppuration may assume the form of a *perinephric abscess*, and this latter condition I have seen extend behind the liver and through the diaphragm, giving rise to an empyæma. When the appendix hangs low in the pelvis, it may become adherent to the uterus and cause symptoms similar to those of uterine or ovarian disease. When no adhesions to shut off the pus from the rest of the cavity form, *general peritonitis*, as described above, ensues.

The Nature of the Ulceration. In about one half of the cases examined by the author there has been no concretion present. Very often a large part of the appendix is gangrenous. This would point not only to intense infective inflammation, but also to imperfect blood-supply. As has already been suggested in conditions of distension of the cæcum, whether from fæces or gas, it is probable that the veins of the appendix are pressed on, and the congestive œdema thus produced would render the mucous membrane liable to invasion by pyococci. Perforation of the appendix is common in anæmic young women. Once this invasion has occurred, its liability to extend deeply is accounted for by the large collections of adenoid tissue with which the appendix is furnished. In a few cases the ulceration is tubercular.

Perforation of the Stomach or Duodenum from Ulceration. Simple ulceration of the stomach occurs in two different classes

of persons: in young anæmic subjects, chiefly girls, and in middle-aged drunkards. Duodenal ulcers resembling simple ulcers of the stomach are peculiar in occurring chiefly in middle-aged men apparently healthy in other respects. These ulcers are presumably due to some infection, since when one forms on the anterior wall of the stomach or duodenum there

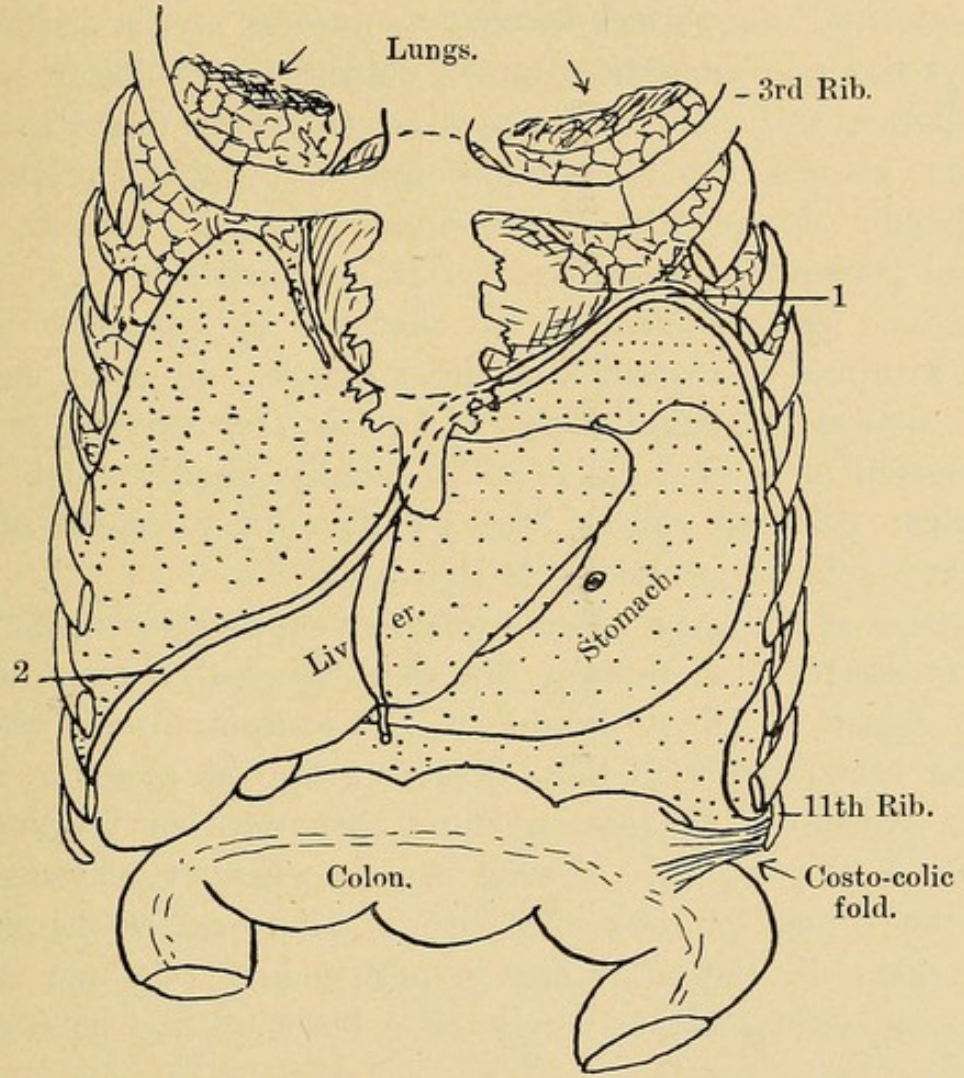


FIG. 117.—A diagram showing a subphrenic abscess from perforation of the stomach on the left side and an empyæma on the right side. 1, The diaphragm pushed up by the subphrenic abscess; 2, the right part of the diaphragm pushed down by the empyæma in the right pleura.

is usually another exactly opposite to it on the posterior wall. A favourite position for gastric ulcer is about the middle of the lesser curvature, but they may occur at any part. If perforation takes place through the posterior wall of the stomach the aperture is usually sealed by firm adhesion to the pancreas. Perforation of the anterior wall of the stomach may

give rise to three conditions: (1) There may be rapid general peritonitis, or (2) there may be suppurative peritonitis confined to the left hypogastrium and constituting a *subphrenic abscess.* Such an abscess is bounded above by the diaphragm, surrounds the spleen, on the left is bounded by the falciform ligament of the liver, below is bounded by the costo-colic fold and the meeting of the great omentum with the anterior wall of the abdomen. Subphrenic abscess, by pushing up the diaphragm, paralyzed by inflammation, causes dulness in the lower part of the thorax, and closely simulates an empyæma (see Fig. 117). If the abscess should contain gas a pyopneumothorax is simulated. Frequently a subphrenic abscess co-exists with general peritonitis in cases of perforated gastric ulcer.

(3) The perforation may be shut off by adhesions which form within a short radius of the aperture. In such cases a local abscess communicating with the stomach is formed. Perforation of a duodenal ulcer is usually rapidly fatal. The intestinal contents collect first on the under aspect of the liver and general peritonitis is the result.

Abscess of the Liver. Acute infective suppuration in the liver usually destroys life rapidly, but the "tropical abscess of the liver," which is due to dysentery, and contains in its wall the amœbæ associated with that disease as well as pyococci, has a slower course. Such abscesses may open into the peritoneum, causing fatal peritonitis, or they may be eliminated by opening into the colon, or they may form a *subphrenic abscess* on the right side. In the latter case empyæma and gangrene of the lung may ensue, and pus may enter a bronchus and be coughed up.

Perforation of the Intestine in Typhoid fever. This usually concerns the lower part of the ileum. Two very different modes of perforation occur. In one there is a single perforation, the formation of which is marked by sudden pain followed by signs of peritonitis. Such cases have been successfully dealt with by operation. In other cases a wide area of the bowel becomes softened and its coils adherent from peritonitis. Perforations of the bowel form and are closed in by lymph and soft adhesions.

Ulceration of the Gall-Bladder in Typhoid Fever also occurs occasionally. Perforation into the peritoneal cavity may then ensue. In one such case [1] a successful operation was performed.

Other Causes of Suppurative Peritonitis. Perforation of the bowel above a chronic obstruction, especially cancer, is common (see p. 275). Extension of septic inflammation from the umbilicus, from the uterus infected after delivery, etc., from perforation of the bowel, etc., after injuries, and many other modes of origin of suppurative peritonitis will occur to the reader.

[1] Marmaduke Sheild.

CHAPTER XIX.

SURGICAL AFFECTIONS OF THE SALIVARY GLANDS, LIVER, AND PANCREAS.

THESE glands are associated by their being accessory to the alimentary tract.

Of the *salivary glands* the parotid is the more frequently the seat of disease.

Salivary Fistula is a troublesome condition, arising most frequently from wounds of the parotid duct. It may be caused by suppurating glands, or necrosed bone. It sometimes arises from pathological ulceration extending both into the duct and through the skin. The opening in the skin may be very minute and yet discharge much saliva.

Salivary Calculi are met with most commonly in the ducts of the submaxillary and sublingual glands. They consist chiefly of phosphate of lime and usually have a somewhat elongated form corresponding to the shape of the duct in which they form. They may completely block the duct and so cause swelling of the gland, or they may cause ulceration of the duct and so project into the mouth. They are easily removed. If necessary, the duct may be slit for this purpose.

Blocking of the Ducts may occur apart from the formation of calculus. This occurs most commonly in the submaxillary gland, and is accompanied by rapid painful swelling.

Mumps or Infective Parotitis is one of the infective fevers the local effects of which usually commence in the parotid gland. Frequently the submaxillary glands also suffer. It is marked by pain on one side of the face, in mastication, etc.

First one parotid swells, then the submaxillary of the same side. The same glands on the other side of the face follow suit. In the male the testis, in the female the ovary or mamma may become inflamed. The affection is commonest in children, and it has to be distinguished from inflammatory swelling of lymphatic glands. The swelling usually begins to subside in about six days. The incubation period is given variously at three and at eighteen days.

New Growths are not uncommon in the parotid, whilst they are rare in the other salivary glands. The commoner growths are composed of varying proportions of adenomatous proliferation, with fibro-cartilaginous, myxomatous, and small-celled sarcomatous tissue. They are innocent or malignant according to the character of the connective-tissue part of the tumour. Thus fibro-chondro-adenomata form slow-growing lobulated tumours which can be removed, due care being taken of the facial nerve, the temporo-maxillary vein, and the external carotid artery. The adeno-sarcomata have a more rapid growth, and tend to infiltrate the surrounding tissues. Cancer is occasionally met with.

Diseases of the Liver.

Surgical affections of the liver are chiefly abscess, growths, hydatid cysts, and the effects of gall-stones.

Abscess of the Liver. Tropical abscess has been referred to above under "Peritonitis" (p. 284). Multiple abscesses occur in infants from septic absorption along the umbilical vein, due to neglectful dressing of the navel after birth. Other rapid infective inflammations of the liver are due to septic absorption from ulcers, etc., in the stomach and intestine. These conditions are much less amenable to surgical treatment than is the tropical abscess. In typhoid fever the wall of the *gall-bladder* may slough and lead to perforation into the peritoneum (see p. 285).

Hydatid Cysts. The liver is the commonest seat of hydatid disease, owing to the fact that the embryos of the parasite enter the portal venules in the wall of the intestine and pass

along the portal veins to the liver, where they lodge in the capillary subdivisions. Very exceptionally an embryo passes beyond the liver, as is evidenced by the occasional formation of hydatid cysts in the lung, kidneys, bones, or other organs. When hydatids form in the peritoneum it is usually due to an embryo having lodged in the vessels of the intestine.

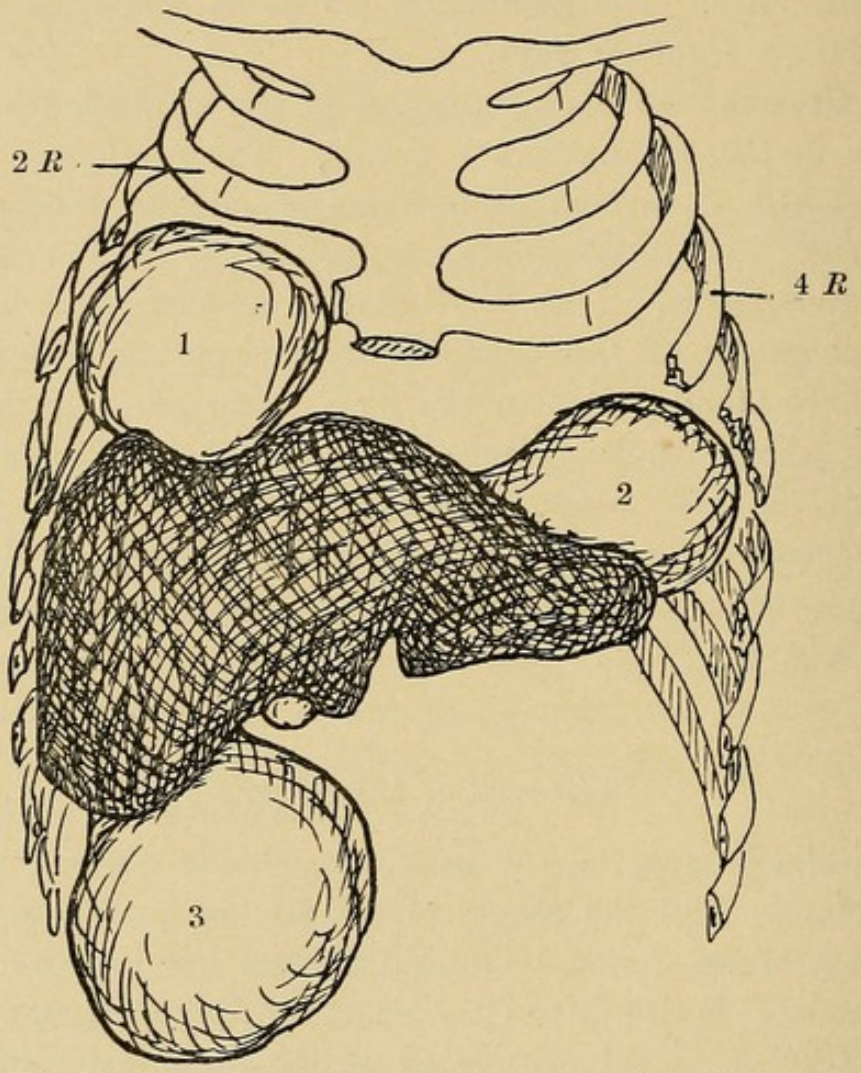


FIG. 118.—A diagram made from a fatal case of hydatid of the liver. 1, Hydatid cyst in the upper part of the right lobe: it pushed up the diaphragm to the level of the second rib (2 *R*); 2, hydatid cyst in the left lobe: it pushed up the diaphragm to the level of the fourth rib (4 *R*); 3, hydatid cyst which projected into the abdominal cavity from the lower surface of the right lobe of the liver. The heart was displaced, and the lower parts of the lungs were collapsed.

A hydatid cyst consists of two parts: (1) An outer fibrous cyst-wall which is produced by and is continuous with the tissues in which the parasite lodges; (2) a parasitic part which consists of an outer laminated chitinous coat, the cuticle of the colony of parasites; and an inner cellular coat from which (*a*) young parasites with hooklets, etc., and (*b*) daughter cysts are

formed. In operating on a hydatid cyst the outer fibrous coat of the parasite is opened, and then the parasitic part is removed and a drain inserted. When hydatid cysts increase rapidly in size they may cause death by displacing important organs, as shown in Fig. 118.

When a hydatid cyst pushes up the diaphragm it is operated on through the lower part of the thorax, below the posterior part of the ninth rib. When it projects below the costal margin it is operated on through the abdominal wall.

Multiple Hydatids. The liver may be thickly studded with small hydatid cysts. Such cases are unsuited for operative treatment.

Degenerative Changes. How long a hydatid colony can retain its vitality is not known. Frequently they perish after a time. Then the parasite and sometimes the fibrous capsule undergo calcareous infiltration, or the dead parasite may caseate, or it may set up suppuration, thus constituting one cause of abscess of the liver.

Gall-stones are usually composed chiefly of *cholesterin*. Often they are rather easily crushed. They have a somewhat waxy consistence, so that when moist they can be cut with a knife, the parts cut off readily breaking up in a soapy powder. Another kind of calculus is formed chiefly of inspissated bile pigment. Such calculi are usually of small size, and often resemble a collection of small shot. Much more rarely collections of calcium carbonate occur in the gall-bladder. Biliary calculi may form in any part of the ramifications of the bile-ducts, either before or after they have left the substance of the liver. They are commonest in the gall-bladder, and it is the passage of a stone along the cystic and common hepatic ducts that gives rise to a typical attack of hepatic colic. If a stone becomes permanently impacted in the common bile-duct, or in the common hepatic duct, persistent jaundice, and, it may be after some years of illness, death from cholæmia result.

It is surprising how common it is to find gall-stones on making a post-mortem examination. In many cases no symptoms have been present during life.

When the gall-bladder is distended with stones, and there

is much local pain, pointing to the possibility of ulceration, or where there are frequent attacks of renal colic, it is necessary to remove the calculi by *cholecystotomy, i.e.* cutting into the

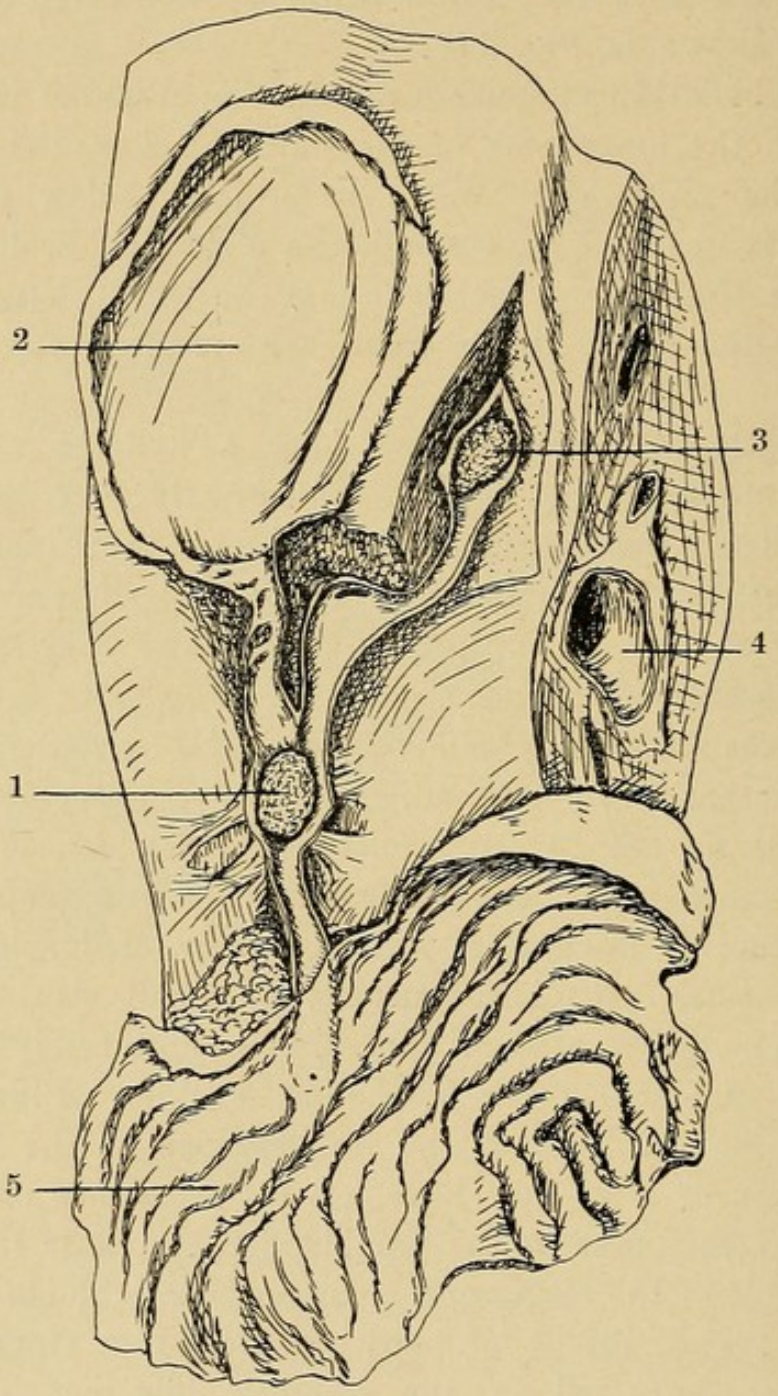


FIG. 119.—The biliary passages and part of the liver showing at 1, calculus impacted in the common bile duct; 2, the gall-bladder opened; 3, a gall-stone in an intrahepatic duct exposed by incising the liver; 4, an intra-hepatic duct which, like all the bile-ducts in the liver, was dilated and filled with thick bile; 5, duodenum opened.

gall-bladder. This operation is easy when the gall-bladder is distended and difficult when it is contracted.

Far more serious is the impaction of a stone in the common

bile-duct, as is shown in Fig. 119. If such a condition is left untreated, death must result. Sometimes a calculus may be safely *crushed* through the wall of the duct by the fingers or by padded forceps, aided, if necessary, by puncture of a *fine* needle:[1] this is *Cholelithotrity.* When the part of the duct in which the stone lies can be reached for stitching, the stone may be removed through an incision, a drain being made below the twelfth rib (R. Morison). Failing this, a communication may be made between the dilated gall-bladder and the duodenum (*Cholecystenterostomy*).

Growths of the Liver are chiefly adenoma and cancer. Sarcoma is not very rare. Usually these growths are not suited to operation, but adenomata, and cancers arising primarily in the gall-bladder, have been thus treated.

The Pancreas.

From its deep position behind the stomach, and its intimate connection with the duodenum, the pancreas is not well placed for surgical treatment; one condition, however, *pancreatic cyst*, has been successfully operated on. Such cysts are most frequently referred to traumatism. They have the stomach stretched over them, and form fluctuating swellings below the left costal margin. Jacobson[2] treated one such case successfully by drainage.

[1] See Mayo Robson, *Gall-Stones*, 1892.

[2] See Pitt and Jacobson, *Transactions of the Medico-Chirurgical Society*, 1891.

CHAPTER XX.

DISEASES OF THE RESPIRATORY TRACT.

THE nasal fossæ form the first portion of the respiratory tract. During sleep, and in quiet breathing, the nose is the proper air inlet. Hence the importance of all forms of nasal obstruction, which may be due to anatomical peculiarities, such as deviations of the septum nasi, or pathological conditions, such as adenoid vegetations of the naso-pharynx. In very young infants, in whom the nose-breathing instinct is strong, blocking of the nose from syphilitic coryza (*snuffles*) may be so serious that it becomes necessary to pass india-rubber tubes through the nostrils into the naso-pharynx in order to re-establish the passages. The importance of disease of the nasal fossæ has become more generally recognized of late years. In this place only a brief review of the more important features can be given.

Deviation of the Septum Nasi. These are either present at birth, or more commonly form by the septum growing in size out of proportion with its surroundings about the time of puberty, or again they may be of traumatic origin. A sharp bend of the cartilaginous portion may result in the complete blocking of one nostril (see Fig. 120). By a double or S-shaped bend of the septum both passages may be completely blocked.

Swelling of the Inferior Turbinated Bodies. This is a common cause of temporary nasal obstruction. The veins in the submucous tissue of the thick mucous membrane covering the inferior turbinated bones are large and are intersected by bands of muscular tissue, during contraction of which the veins

become distended with blood. This constitutes what is termed an erectile tissue. In some persons the irritation of dust, catarrh, and certain psychical states determine erection of the inferior turbinated bodies with resulting nasal obstruction. Spraying cocaine solution into the nostrils does away temporarily with both the erection and the obstruction.

Coryza. The milder forms of coryza—cold in the head—are familiar, needing no detailed description. Some of the more severe catarrhal conditions are of a serious nature.

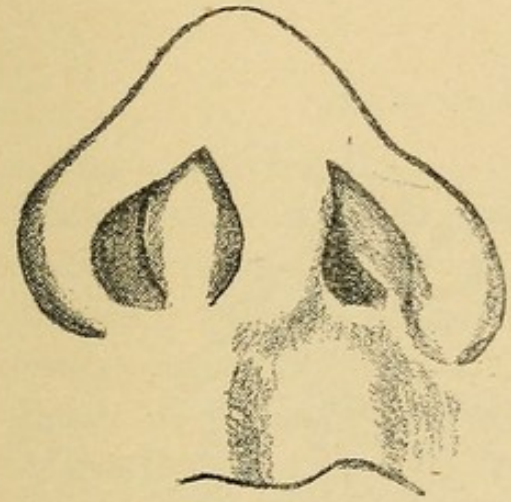


FIG. 120.

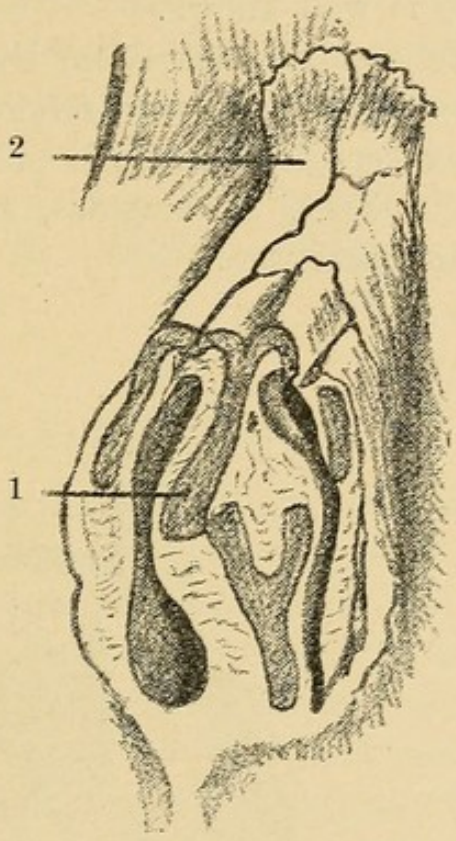


FIG. 121.

FIG. 120.—The anterior nares of a person, with deviation of the cartilaginous septum to the right. After Zuckerkandl.

FIG. 121.—Vertical section through the anterior part of the nose showing a fracture of the cartilaginous septum (1), with blocking of the nostrils; 2, right nasal bone. After Zuckerkandl.

Syphilitic Coryza is one of the earliest and most constant of the symptoms of congenital syphilis. Under treatment the catarrhal inflammation of the mucous membrane clears up without the occurrence of ulceration, which is more frequent in the later stages of the disease.

Atrophic Rhinitis usually begins early in life as a purulent catarrh. If it is not checked by prompt treatment the condition becomes chronic, the mucous membrane assumes a glazed red appearance, crusts and scabs form on the surface, and necrosis of the turbinate bones with the development of the

peculiarly objectionable smell which is known as *ozæna*. The atrophic process usually spreads to the pharynx and sometimes to the larynx.

Syphilitic Ulceration, with extensive destruction of the bones and ozæna, is a common late event in both the congenital and the acquired disease; the resulting sunken bridge of the nose is well known.

Tubercular Ulceration, both as lupus vulgaris, and as the more rapid so-called scrofulous ulceration, is common. In the latter condition ozæna is present.

Glandrous, sarcomatous, and cancerous ulcerations are sometimes encountered.

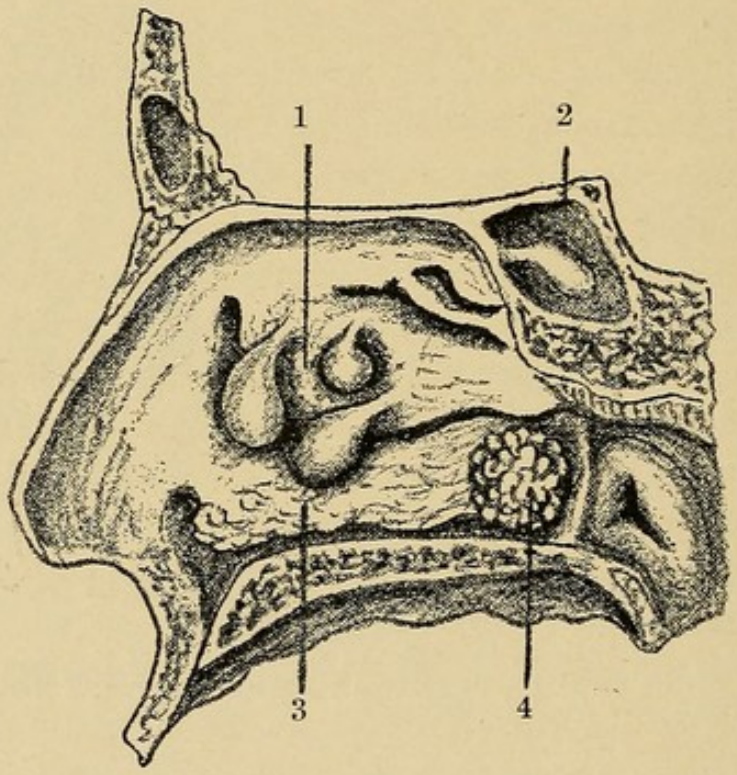


FIG. 122.—The outer wall of a right nasal fossa. 1, Mucous polypi growing from the middle turbinated bone; 2, sphenoidal sinus containing a mucous polyp; 3, hypertrophied inferior turbinate body; 4, mulberry growth. Diagrammatic.

Rhinoliths are calcareous formations most frequently formed around a foreign body in the inferior meatus. Bony growths from the bones of the nasal fossa are sometimes mistaken for rhinoliths.

Morbid Growths. The commonest of these are the "mucous polypi," which have a myxomatous structure and are covered

with ciliated nasal mucous membrane. Their commonest place of origin is the mucous membrane covering the middle turbinated bone (see Fig. 122).

Hypertrophy of the Inferior Turbinated Bodies (Fig. 122) is a common condition which results from chronic catarrh (*hypertrophic rhinitis*).

Mulberry Growths at the posterior extremity of the inferior turbinated body are not uncommon, and from their proximity to the Eustachian tube-orifices not infrequently cause deafness.

Fibrous and **Malignant** growths also occasionally arise from the roof or septum. In investigating any growth in the nasal cavities the possibility of its being a small meningocele must be borne in mind.

Diseases of the Air-Sinuses.

The air-sinuses, frontal, maxillary, ethmoidal, and sphenoidal, which open into the nasal fossæ, and which are lined with ciliated mucous membrane, are subject to some of the same diseases. The tympanic cavity and mastoid antrum constitute similar air-sinuses, but their diseases are so closely allied to disease of the brain that they will be left to a later section.

The frontal, ethmoidal, and sphenoidal sinuses appear during childhood, but remain of small size until the period of puberty, when they undergo a rapid enlargement. The maxillary sinuses begin to be formed early in fœtal life.

Suppuration in the frontal sinuses is apt to assume a chronic course, the pus finding its way by the infundibulum into the nose. If by reason of headache, inability to keep the head down in reading, redness of the skin over the sinus, or distension, the condition makes itself evident and the sinuses are opened, their inner surfaces are found to be studded with small papillated growths which give out a pungent smell. If the condition be neglected the suppuration may become acute during an attack of coryza, and so lead to meningeal or cerebral suppuration. The bones covering the frontal sinuses may undergo thickening (*hyperostosis*), or bony (see Fig. 70), cancerous, and other growths may arise within them.

The maxillary antrum, or antrum of Highmore, besides being subject to the same diseases as the rest of the respiratory tract, is exposed to the extension of inflammation from the teeth. The cavity forms a pyramid with its base at part of the outer wall of the nasal fossa, and perforated by the opening in the middle meatus. The three sides are facial, orbital, and zygomatic. The floor is in close relation with the outer fangs of the molar and sometimes of other teeth. This close relation with the teeth sometimes occasions acute or subacute suppuration in

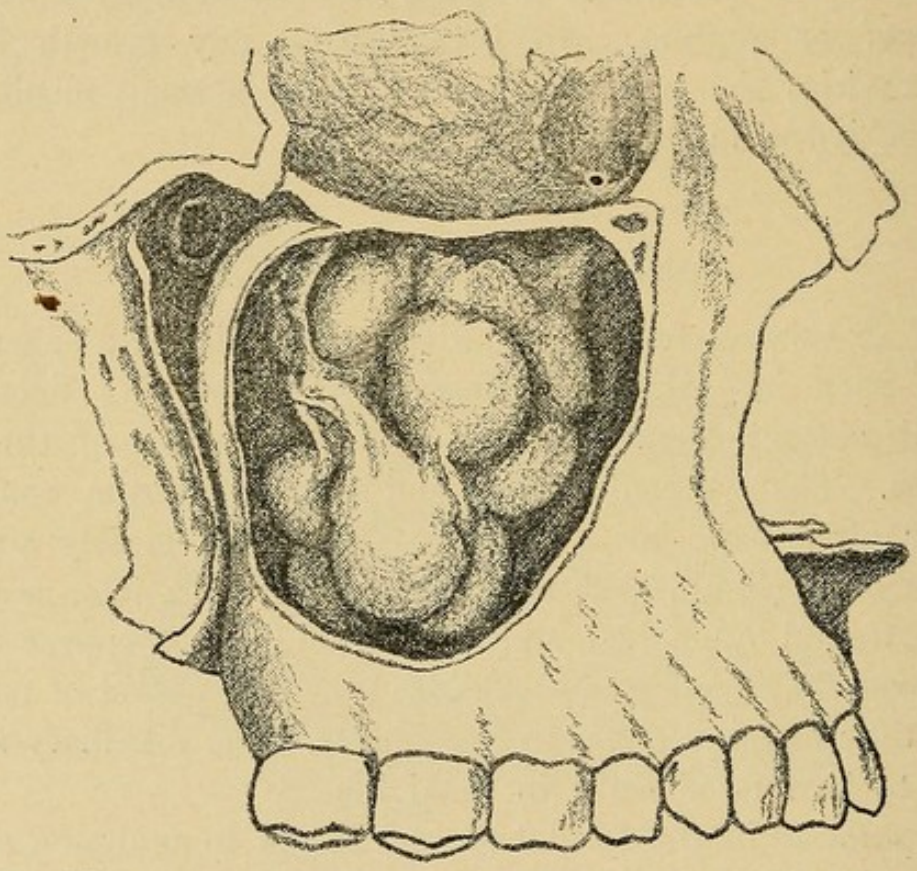


FIG. 123.—An antrum containing a large cluster of cystic polypi, one of which almost blocks the opening into the nose. After Zuckerkandl.

the antrum from extension of an alveolar abscess. Or independently of the teeth the suppuration or "empyæma" of the antrum may be due to purulent catarrh. In the latter condition there is often an intermittent flow of pus into the nose. This pus has a faint odour which, unlike the more repellent smell of ozæna, is perceptible to the patient, but not to those about him.

Cystic polypi are sometimes formed in the antrum (see Fig. 123).

When these cystic polypi are numerous the antrum may become dilated in the same manner as from other growths.

Other growths not infrequently met with in the antrum are cancer and sarcoma.

The ethmoidal sinuses are so close to the cribriform plate and the brain that suppuration in them spreads with great facility to the meninges.

Diseases of the Naso-Pharynx.

The naso-pharynx is lined with ciliated epithelium. The prominent Eusctachian cushions project into its anterior and lower lateral aspects. Between the Eustachian tubes, at the foremost part of the roof, on each side of the middle line, are two or more ridges of lymphoid tissue which constitute the pharyngeal tonsil.

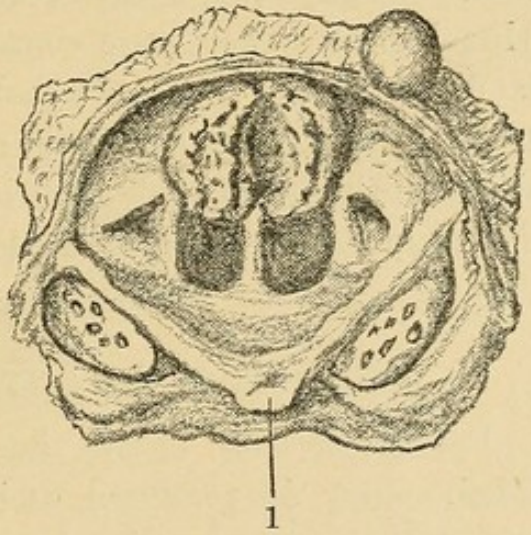


Fig. 124.—The naso-pharynx of a child. There is a mass of adenoids between the Eustachian tubes. 1, The uvula. A lymphatic gland, slightly enlarged, rests on the roof of the pharynx. (1057 A.)

Adenoid Vegetations, or, less classically, adenoids (Gr. *adēn*, *adĕnos* = gland, *eidos* = like), result from inflammatory hyperplasia of the pharyngeal tonsil, and are composed of tissue like that of lymph glands covered by epithelium. The latter in the specimen shown above consisted of several layers of flattened cells. Commonest in rachitic children, they are of very frequent occurrence and of serious import to the patient. The naso-pharynx of a child aged eight, dissected out, is represented in Fig. 124. There the adenoids form a mass as compact as a hypertrophied faucial tonsil, still the ridges from which the growths are developed are distinctly discernible.

More frequently the ridges throw out blunt processes as they enlarge, giving the mass a papillomatous look. During life, when turgid with blood, they are larger than after death, so that it is hard to gauge their importance from *post-mortem*

preparations. Their proximity to the orifices of the Eustachian tubes explains the deafness that accompanies adenoids, and by their proneness to attacks of inflammation the growths form a focus from which catarrhal suppuration spreads in all directions, including the Eustachian tube and the middle ear. The many other pathological effects of adenoids are described in clinical works.

Fibrous Tumours of the Naso-pharynx spring from the periosteum of the base of the skull. They are commoner in youth than in age. They sometimes grow to a large size, pushing down the soft palate, forcing their way into the nasal fossæ, and widening them they cause the deformity known as "frog face." They frequently recur locally after being removed. Histologically they have sometimes a purely fibrous, sometimes a fibro-cellular (fibro-sarcomatous) structure. In a few cases their blood-vessels are very markedly developed, so that they deserve the name of fibro-angiomata. Such growths bleed furiously when removed by the snare.

Affections of the Larynx, Trachea, and Bronchi.

Foreign bodies enter the air-passages during inspiration. Their entry is rendered easier by paralysis of the laryngeal muscles, *e.g.* after diphtheria, and during deep anæsthesia. The larynx may be occluded by a large bolus of food or other large body becoming impacted in the lower end of the pharynx.

In the larynx a foreign body sets up much reflex spasm, and, though the passage is not occluded completely by the foreign body, death may occur from spasm interfering with inspiration, unless speedy tracheotomy be done.

If the body enters the trachea it sets up an initial attack of cough and dyspnœa. This usually subsides. Fresh attacks of dyspnœa come on whenever the body is coughed up against the vocal cords. As the result of coughing, the body may be ejected; more frequently it becomes fixed in the larynx and causes death from spasm-suffocation. If the body should remain in the trachea in course of time it sets up ulceration

and suppuration: thence arises bronchitis and fatal broncho-pneumonia, if the foreign body be not removed through a tracheotomy wound.

Foreign bodies in the large bronchi occasion at first urgent dyspnœa. This may subside, leaving the patient, if not relieved by operation, finally to die of bronchitis or bronchiectasis.

Acute Laryngitis may be due to irritants such as chlorine gas or steam. More commonly it is part of an acute catarrh of the upper respiratory tract, such as is commonly ascribed to cold. The symptoms are usually severe constant cough, some dyspnœa from spasm, fever, etc. The important thing is to distinguish simple catarrhal from diphtheritic laryngitis.

Acute catarrhal laryngitis is sometimes observed in measles and other fevers: in small-pox pustules may form in the larynx.

Chronic Catarrhal Laryngitis is common in drunkards and in those who have to speak in badly ventilated rooms, or in cold foggy air. The mucous glands become enlarged, the voice husky. The slighter forms of syphilitic laryngitis may be mistaken for simple chronic catarrh.

Œdema of the Larynx. When the mucous membrane constituting the false cords becomes greatly swollen they meet across the middle line, and unless surgical relief is given death speedily ensues. Such swelling may occur after fracture of the hyoid bone or of the laryngeal cartilages; simple œdema from Bright's disease; swelling from inhaling steam, drinking hot water, etc.; and more commonly œdema from infective processes in the submucous tissue, *i.e.* cellulitis, in the larynx or its neighbourhood, also produce this condition. A varying amount of œdema is present in acute laryngeal catarrh.

Diphtheria of the Larynx owes its special importance to its interference with respiration. Symptoms of laryngitis mark the onset, and at this period the mucous membrane is red and slightly swollen. In a day or two the typical grey adherent membrane forms on the mucous membrane, causing a certain amount of obstruction which tends to increase so that when *stridor*, the symptom that indicates laryngeal obstruction, is present, all must be made ready for operative treatment either

by intubation or tracheotomy. In many cases the membrane is formed not only in the larynx but also in the trachea and larger bronchi. When the disease extends into the middle-sized or smaller bronchi the case is hopeless.

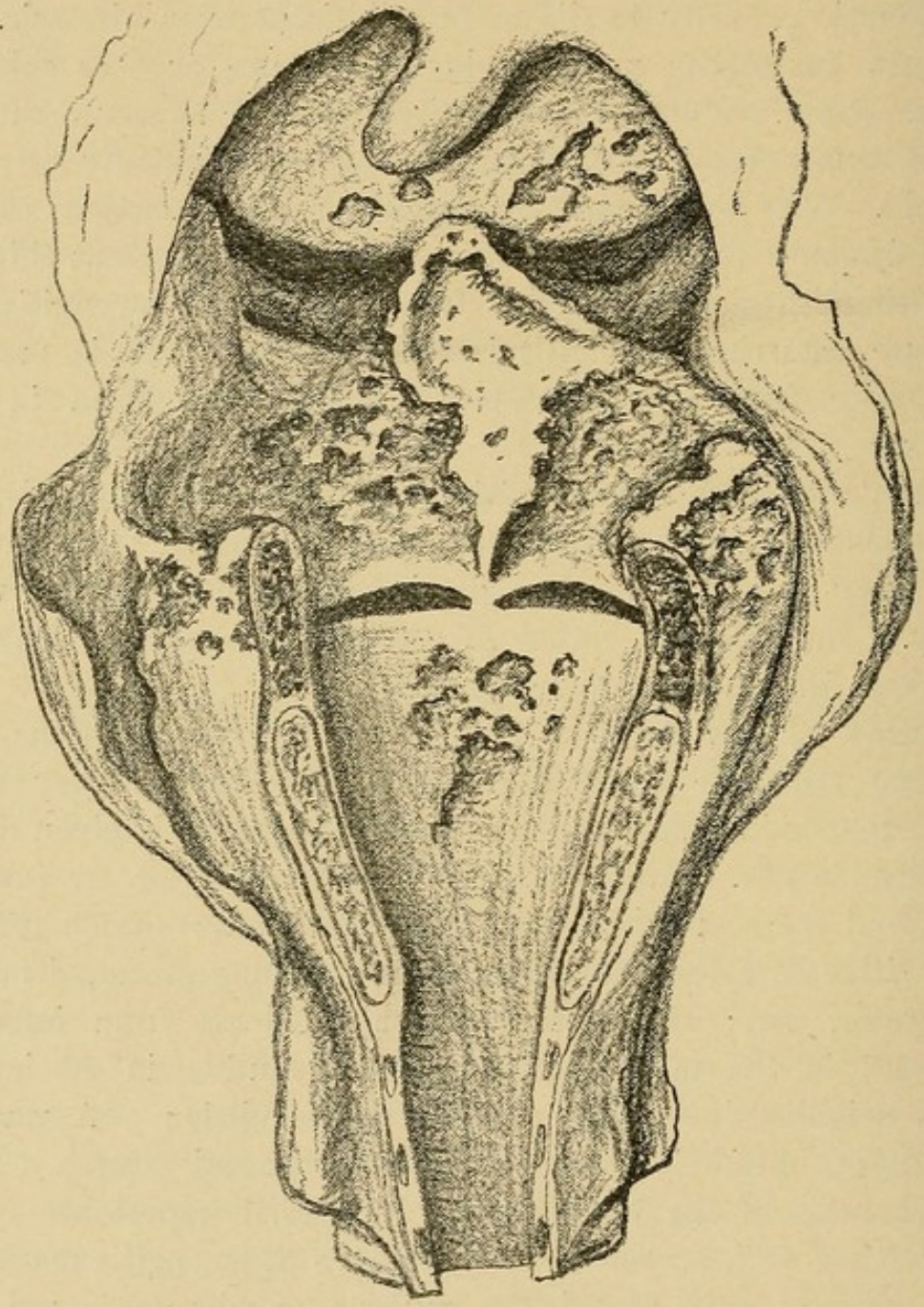


FIG. 125.—Advanced tubercular ulceration of the larynx, tongue, and trachea. From a man who died of phthisis. (1083.)

If recovery takes place the parts previously the seat of the membrane are covered by granulation tissue which may still block up the larynx, and during its disappearance leave per-

manent contraction. Death may occur either from the laryngeal obstruction, from diphtheria of the bronchi, from septic bronchopneumonia, or from subsequent diphtheritic paralysis. The results of operation vary according to the degree of virulence of the infection and the extent of the membrane, etc., as well as to the skill of the operator. . The low operation, as being likely to lead to emphysema of the tissues, is especially to be avoided.

Decubital Ulcers. In bed-ridden people with weak circulation ulcers may form in the mucous membrane, covering the vocal processes of the arytænoids. They are akin to bed-sores.

Typhoid Lesions of the Larynx. In severe cases of typhoid fever the entire mucous lining of the larynx, and sometimes of the trachea, undergoes necrosis. The swelling, etc., may necessitate an operation.

Tuberculosis of the Larynx. Pallor of the larynx and swelling of the mucous membrane forming the arytæno-epiglottidian folds, are usually the first objective signs. Small nodules which break down into ulcers form in the swollen mucous membrane. The ulceration often begins near the vocal processes of the arytænoids, and subsequently spreads and deepens. If the ulcers reach the perichondrium they open up a way for the entrance of ordinary pyogenic organisms, with the result that destructive suppurative perichondritis supervenes. Nearly all cases of laryngeal tuberculosis are secondary to tuberculosis of the lungs. Lupus vulgaris sometimes affects the larynx.

Leprosy. The larynx in nodular leprosy often becomes ulcerated and deformed.

Syphilitic Laryngitis. In secondary syphilis catarrh and mucous tubercles may occur. It is in the late period of the disease, however, that the more serious laryngeal affections arise. Many persons have for years a chronic syphilitic laryngitis which gives rise to a harsh voice but no other inconvenience. But at any time the infiltrated tissues may ulcerate, and this process may rapidly destroy the epiglottis; if the ulcers penetrate to the perichondrium, destructive and fatal suppuration may result, as is also the case in some tubercular and cancerous ulcerations.

Morbid Growths.

Papilloma of the Larynx is commonest at the anterior parts of the vocal cords (see Fig. 126). When they are abundant, as they are apt to be in children, they may cause serious and even fatal dyspnœa.

Other innocent tumours, such as mucous cysts, fibrous polypi, sometimes occur in the larynx.

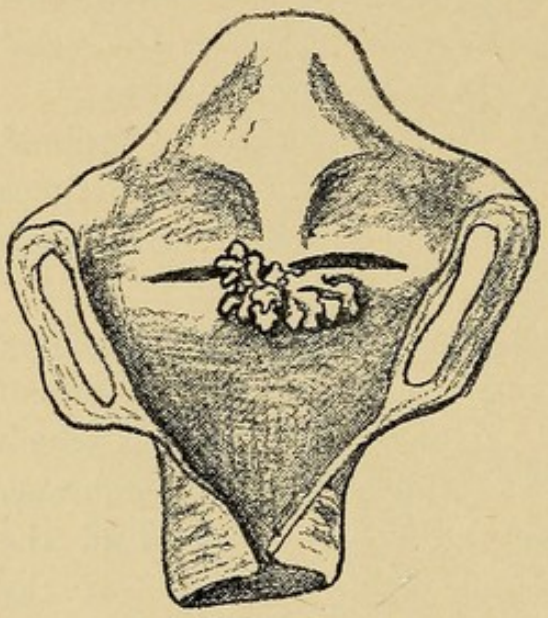


FIG. 126.—Papillomata in the larynx of a child. (1087.)

Cancer of the Larynx. This is either intrinsic, *i.e.* arising within the larynx itself, or extrinsic, *i.e.* extending into the larynx from the pharynx, or beginning on the epiglottis. The former condition is the only one in which operative removal can be contemplated, and it is of practical importance that in intrinsic cancer of the larynx the extension of the growth is slow and the infection of glands is late.

As soon as the growth ulcerates septic processes begin. The most common mode of death is from septic broncho-pneumonia.

Sarcoma of the Larynx. This growth sometimes originates within the larynx itself, more frequently it is an extension of lymphosarcoma from the glands of the neck. The course of the infection is much the same as in cancer.

Surgical affections of the trachea are few. Plastic operations for stenosis due to syphilitic ulceration have been proposed, but not as yet carried out. Stenosis of the trachea from the pressure of a goître will be dealt with elsewhere (p. 308). Stenosis of trachea or bronchi, from the pressure of tubercular bronchial glands, is a condition that is often beyond the power of surgery to remove.

Surgical Affections of the Lungs.

These are chiefly purulent pleural effusion or *empyæma* (see Fig. 118), gangrene of the lung, and a few other conditions such as hydatid cyst of the lung, tubercular, and bronchiectatic cavities. The first of these will be briefly noticed.

Empyæma, especially in children, is sometimes a primary affection due to infection of the pleura by pneumococci, sometimes it is secondary to perforating wounds or other injuries, to the perforation of the pleura by a tubercular lung-cavity, or perforation of the diaphragm by a subphrenic abscess such as that which results from perforation of a gastric ulcer or from an abscess of the liver. Empyæma is practically an abscess

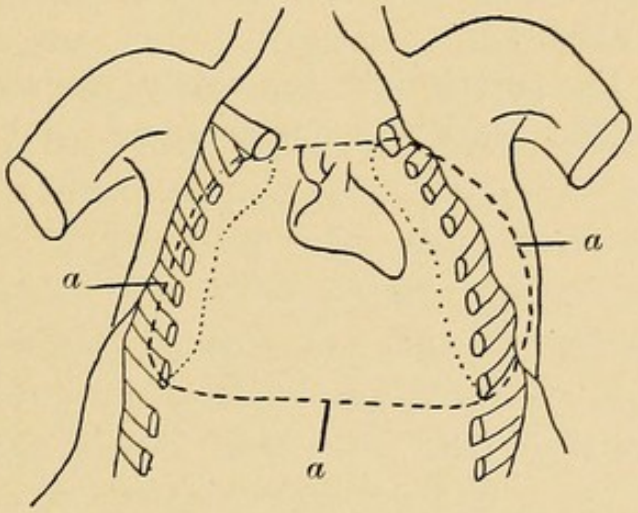


FIG. 127.—Pericardial effusion. The interrupted line *a* indicates the distended pericardium; the dotted lines indicate the pleuræ: the heart is drawn in outline.

which, whatever its position, must be opened in the most accessible position that allows of good drainage. Some empyæmata are complicated by adhesions of the lung to the chest wall, a condition which may cause the operation of draining it to be somewhat difficult.

Wounds of the Lung may lead to *prolapse* of part of the lung through the wound. If the wound is valvular air may escape from the lung into the areolar tissue of the chest wall and thence may widely distend the subcutaneous tissue of the body. This is *surgical emphysema*. It also occurs in some subcutaneous wounds of the lung, *e.g.* from fractured ribs. *Hæmothorax*

(blood in a pleural cavity), *pneumothorax* (air in a pleural cavity), and *pyopneumothorax* are among other possible consequences of wounds of the lung.

Pericardial Effusions. When they are of a purulent character, it is right to interfere surgically in cases of pericardial effusion. The chief difficulty in diagnosis is to distinguish between effusion and dilatation of the heart. As an example of the extreme pyopericardium the case from which Fig. 127 was taken may be given. Admitted in a dying state the patient, a girl aged nine years, lived only a few minutes after reaching the hospital. After death, on removing the front of the sternum and cutting back the ribs a little beyond the mid-clavicular lines, the sketch was made without further disturbing the parts. The pleuræ and lungs had been displaced outwards to an extraordinary extent.

On opening the pericardium the cavity was found to be full of thick pus. The heart was not enlarged.

CHAPTER XXI.

DISEASES OF THE THYROID GLAND: ACROMEGALY, Etc.

THE thyroid gland is developed from a diverticulum of the mouth; the *foramen cæcum* remains at the point of its origin. It then passes behind a mass of tissue, which afterwards becomes the body of hyoid bone; below that it bifurcates into two lobes, which become the lateral lobes of the thyroid, whilst the end of the duct just above it develops into the pyramid or middle lobe of the thyroid, above which the duct in normal conditions disappears. The diverticulum is sometimes continued in the middle line of the neck beyond the point of origin of the lateral lobes, and then it may open on the surface, forming a *median cervical fistula*.[1]

The lateral and median lobes develop into numberless acini, which under the microscope appear to be closed.

Accessory thyroids may form in any part of the thyroid duct. Thus solid tumours, resembling the thyroid gland in structure, may be found in the tongue, in the neighbourhood of the hyoid bone, or between the lateral lobes of the thyroid. From the lateral lobes also accessory thyroids may develop about the cornua of the hyoid bone.

The Functions of the Thyroid Gland are important. This importance is seen in cases where the whole of the gland has been removed by operation. A condition termed *cachexia strumipriva* then develops. It commences gradually from one to five months after the operation: weakness and heaviness of

[1] See Bland Sutton, *Tumours*, p, 314.

the limbs with dragging pains, coldness and weakness of the circulation, slow cerebration, loss of memory, etc. The eyelids and face swell, and all expression is lost. The skin becomes dry and scaly, the hair falls out, the abdomen enlarges, and in children growth is arrested. In cases where removal of the gland has not been followed by these symptoms it is supposed that an accessory thyroid, or some other thyroid tissue, has remained behind.

When the same symptoms as have been described above in cachexia strumipriva come on spontaneously in a previously healthy person, the disease is then called **myxœdema**. At an autopsy made by the author on a case of myxœdema, the thyroid gland was found to be transformed into tough scar-tissue by interstitial inflammation.

Sporadic Cretinism is the name given to the equivalent of myxœdema that commences either before birth or in infancy. Besides the symptoms enumerated above there are others due to arrest of growth. The limbs remain stunted, puberty is delayed or does not occur at all. A cretin, of whatever age, always looks like a child. In this condition the thyroid is often absent; it may appear to be normal in size, or even enlarged, but it is probably that, in all cases of sporadic cretinism, the gland is physiologically inactive.

Most striking is the effect of administering sheep's or other animals' thyroid glands to patients who suffer from any of the three conditions above mentioned. They improve mentally and physically, the improvement only lasting, however, as long as the treatment is kept up. The substance in the thyroid, the want of which causes myxœdema, etc., has been termed by Schäfer the "internal secretion" of the gland.

Inflammation of the Thyroid occurs from wounds and other injuries, and now and again acute thyroiditis has been observed as an epidemic. Acute inflammatory affections of the thyroid are very rare.

Chronic Thyroiditis may be assumed to account for the destruction of the gland and its replacement by scar-tissue that occurs in myxœdema.

Goître.

Goître, or **Bronchocele,** is the term applied to any enlargement of the thyroid gland that is not inflammatory or obviously malignant. It includes very different morbid conditions. In the normal gland the colloid secretion in the acini increases in amount in middle and advanced age.

Exophthalmic Goître. In this condition the thyroid gland undergoes a moderate degree of enlargement and becomes pulsatile from dilatation of its arteries. These phenomena are accompanied by proptosis and other symptoms, which are connected with a functional disturbance of the cervical sympathetic. The affection is more common in women than in men; it improves under suitable treatment.

New Growths of the Thyroid Gland.

Adenoma is the most frequent, and exhibits considerable variation of form. Colloid, cystic, fibroid, myxomatous, and other kinds of adenoma are distinguished.

Thus adenomata of the thyroid vary in their characters. The conditions shown in Fig. 128 are those most commonly occurring. The cysts in such tumours are of common occurrence. They are generally occupied by a dark serous fluid, some are the result of hæmorrhage. Papilliferous intracystic growths also may occur. Sometimes such a cyst appears to be the whole of the abnormality, but careful examination will usually reveal adenomatous tissue in the cyst wall.

The adenomatous tumours and cysts vary in one important particular, that is, in the presence or absence of a well-marked capsule. This is important, because if a well-marked capsule is present they can be shelled out during life without serious hæmorrhage.

The capsule when present may be composed solely of fibrous tissue, or more commonly it is merely an apparent capsule, *i.e.* a condensation of the outer part of the tumour. Adenomata may be single, but more commonly they are studded throughout the gland. In some cases the whole

gland assumes an adenomatous character; the condition has then been termed "diffuse" as opposed to "nodose" adenoma. Diffuse adenoma was formerly termed "hypertrophy of the gland." In the case of a child who with his brothers and sisters developed large nodose adenomata soon after birth, one of the smallest tumours had the histological appearance shown in Fig. 30, p. 100.

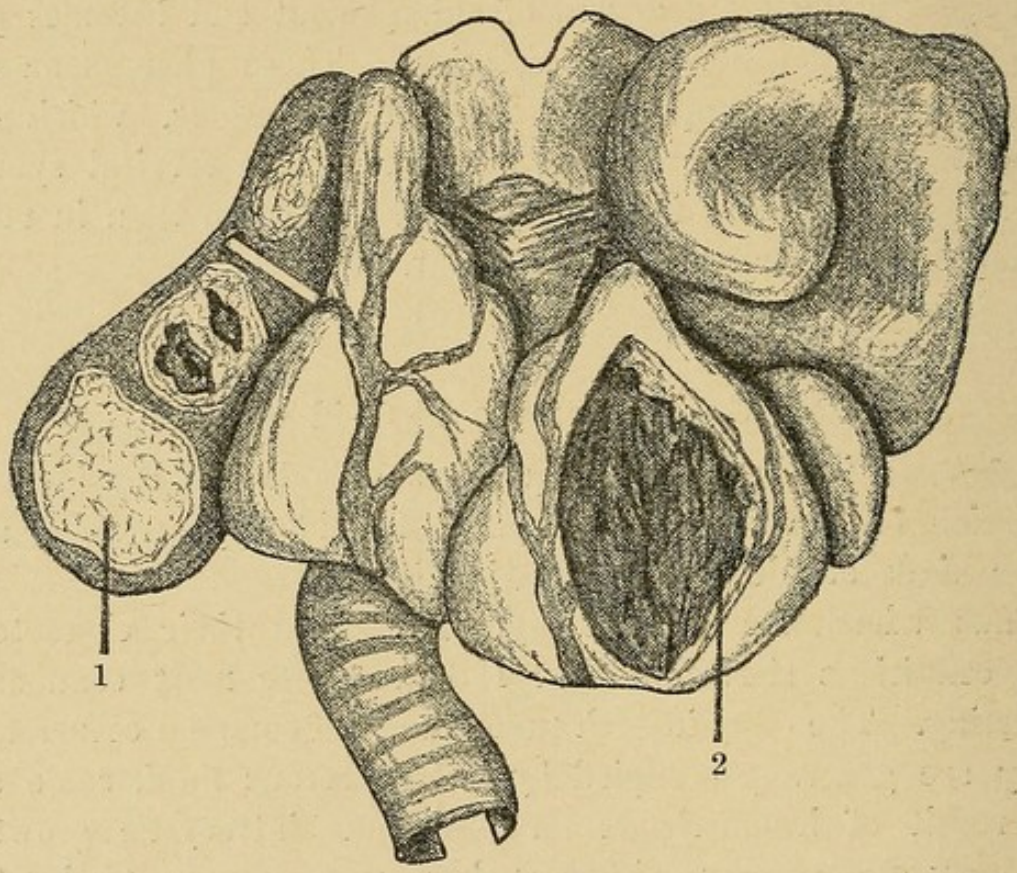


FIG. 128.—Adenomatous goître. The left lobe of the gland has been cut open to show, 1, a solid adenomatous tumour, and above this are two other similar tumours, one of which contains two cysts; 2, points to a large single cyst in the interior of another adenoma. To the right of and above the latter are three similar tumours. The trachea is displaced to the right. From an elderly woman who died in a street from dyspnœa.

In some cases of diffuse adenoma the follicles, instead of being empty as shown in Fig. 30, become more and more distended with colloid secretion. This constitutes the so-called "colloid goître." Or if the acini are very greatly distended the condition is termed "cystic goître."

The blood-vessels in some adenomata attain a considerable development and readily rupture, producing the hæmorrhagic variety of cyst. Hæmorrhages also occur in the connective

tissue of the growths, and the resulting clot undergoes organization, bringing about the condition known as "fibroid goître." In these fibrosed parts of a goître extensive calcareous deposits may occur. Or the interstitial tissue of the growths may undergo hyaline degeneration, leading to necrosis of the follicles from obstruction of the blood-vessels. This condition has been called "myxomatous goître." Goître sometimes increases very rapidly in size. It has then been termed "acute bronchocele." This is especially likely to occur at puberty.

Malignant Disease. Primary cancer is not of infrequent occurrence. By reason of its extending beyond the thyroid gland to the important surrounding organs, the results of removal of the thyroid for cancer are very discouraging. It should be remembered too that in some cases of growth of the thyroid not readily distinguishable from some of the forms of adenoma, secondary growths have appeared in the skull and other bones and have led to a fatal issue. Primary sarcoma is rare. Lymphosarcoma, secondary to lymphosarcoma of the cervical glands, has also been observed.

Effects of Goître. By pressure on the trachea the enlarged thyroid produces dyspnœa. Even though the gland does not appear to be very much enlarged there may be a considerable amount of pressure by the growth projecting on each side behind the trachea, or by the growth projecting behind the sternum and pressing the trachea against the vertebræ. The carotid arteries may be so much pressed on that anæmia of the brain and convulsions result. Pressure on the recurrent laryngeal nerve may produce spasm or paralysis of laryngeal muscles.

Acromegaly.

Acromegaly is a condition marked by overgrowth of all the tissues of the hands and feet and of the bones of the face, especially the lower jaw which projects forwards in front of the upper. The dorsal spine acquires a posterior and the lumbar spine an anterior curve (*lordosis*). The muscles are weak and there may be excessive sweating. The soft parts of the face, *e.g.* lips, nose, chin, ears, and other parts, may also

be increased in amount. The superciliary ridges are thickened, and the whole of the head may be enlarged, the skull becoming lengthened from before backwards. The tongue and the laryngeal cartilages enlarge and the voice becomes changed. The skin is generally thickened, especially that of the external genitals. The disease has been observed to begin as early as fifteen years of age and as late as sixty. Both sexes are liable to it. In females the catamenia cease. In some cases that have been examined after death the pituitary body has been found to be enlarged, thus accounting for optic atrophy with partial or complete blindness that has been observed during life, as also has loss of smell.

Hypertrophic Pulmonary Osteo-arthropathy.[1]

In some persons who suffer from chronic lung disease the clubbing of the fingers common in such affections becomes markedly increased. The bones of the hands and feet and the lower parts of the bones of the forearms and legs become thickened from osteo-plastic periostitis. Other bones, *e.g.* clavicles and spinous processes of the vertebræ, also become thickened. There may be effusion into some of the joints, especially the wrists and ankles, but this differs from that of osteo-arthritis in the absence of "lipping," and from that of Charcot's disease in the absence of marked absorption of bone. The bony thickenings differ from those of acromegaly in being confined to the periosteal surface and in being unaccompanied by extensive thickening of the soft parts.

[1] See a paper by Walters, *St. Thomas's Hospital Reports*, 1897.

CHAPTER XXII.

AFFECTIONS OF THE HEAD: BRAIN, EAR, AND EYE.

Contusions of the Scalp. These are common injuries owing to the highly vascular scalp lying upon a resisting surface. When an ordinary contusion is incised in the post-mortem room all the thickness of the scalp from skin to periosteum is found to be stained by extravasated blood. When blood is poured out in considerable quantity this usually occurs either beneath the epicranial aponeurosis or the periosteum and results in the formation of a *cephalhæmatoma*, that is, a swelling which has a soft fluctuating centre, and often a firm edge, which results from inflammatory reaction at the margin of the effusion and which disappears when steady pressure is made on it, and is thus distinguished from the edge of a fractured bone.

Cephalhæmatomata are usually divided into two classes, sub-aponeurotic and subperiosteal. The latter cannot extent beyond the sutures of the bone over which they form. Usually they do not extend so far. They are commonest in new-born infants from pressure of the forceps, etc. In a few cases of subperiosteal hæmatoma both of the skull and of other bones a shell of new bone is formed by the periosteum over the effused blood. Thus an osteoma-like tumour is produced. Usually there are gaps left in the bony shell from failure of ossification, and at these points fluctuation may be observed.

Fractures of the Skull can only be noticed briefly in this place. The commonest form of fracture of the cranial vault is a *fissure* which begins at the spot where the blow was received and extends in one or several directions therefrom. If several

fissures radiate from a point, the fracture is termed stellate. At the time of the occurrence of a fissure there is a certain amount of bending inwards of the bone, and this may remain after the force which caused the fracture has ceased to act. Thus a fissured fracture may also be a depressed fracture.

Fractures of the Base usually occur by the extension of a fissure from the vertex to the base. In such cases the fissure usually tends to reach first the nearest fossa of the base on the same side. Thus a fracture starting at a left parietal eminence would in all probability pass into the left middle fossa, and then either cross the base, or pass through the temporal bone, going across the tympanic cavity, into the occipital fossa. A fissure starting in the middle line of the forehead has been observed to keep to the middle line of the base, splitting it from the root of the nose to the foramen magnum.

A fracture of the base not infrequently complicates a fracture of a nasal bone. Some fractures of the base are caused by the impact of the atlas on the occipital condyles, others by foreign bodies entering the roof of the nose or orbit. Now and again a fissure of the skull is observed to have formed at a distance from the point at which the blow was applied to the skull.

Incised and Punctured Fractures resemble each other in being compound fractures, and in usually entailing septic infection of the meninges and brain, and also in both the inner table is depressed to a much greater extent than the outer. In short, both conditions call for immediate trephining.

Other Fractures of the Skull. Of the more severe injuries, perhaps a comminuted fracture is the commonest. It may be either compound or simple. It is no infrequent occurrence to find after death such a fracture, which in life has been obscured by blood effused into the scalp. Some of the fragments are usually more or less depressed.

Gutter-fracture. If an area of bone is depressed the above term is applied when an oval area of bone is concerned, and it is termed a "pond" fracture when the depressed area is of a round shape.

In a few cases a circumscribed area of the outer table is depressed or impacted into the diploe without the inner table being fractured at all. In other cases the inner table is depressed without any displacement of the outer table.

Very rarely a portion of the skull-cap is elevated. The fragment, usually quadrilateral, is separated from the rest of the skull at three of its sides, whilst at the fourth it remains attached.

Concussion and Contusion, etc., of the Brain.

Concussion of the brain is the name given to unconsciousness resulting immediately on the receipt of an injury to the head. This term involves the idea that the symptoms are due to a "shaking up" of the brain. Examination after death in fatal cases shows lesions to which more definite names apply; *e.g.* contusion of the part of the brain opposite to that which lay nearest the point of impact of the force. Thus, if a person falls violently on the occiput, the chief damage to the brain is at the tips of the frontal lobes. This is termed injury by counter-shock. The explanation is not simple. It may be remembered that the brain is surrounded by fluid, and that the momentum acquired by the fall in the rapid earthward movement, causes the brain to be pressed closely against the part of the dura that is downwards when the skull is arrested by contact with the ground; that liquid will be forced from between the brain and dura on the downward side, and will communicate the pressure by a wave which impinges on the opposite side of the brain, where before the wave breaks there will be a diminished pressure, which also may cause disintegration. However this may be, the fact remains that the damage is usually greater on the side opposite to that which receives the blow. Of course when there is a depressed fracture, the brain will be bruised or perhaps lacerated on the side of the injury, and, independently of a fracture, there is usually bruising here, but it is not so marked as that produced by "contrecoup." In severe concussion the effects on the brain vary from slight hæmorrhage beneath the arachnoid, to bruising and ecchymosis of the brain at the

points mentioned, and in the worst cases to extensive laceration of the brain, which may be accompanied by extensive hæmorrhage from the pia mater. Small hæmorrhages are sometimes found in the brain substance in cases of concussion. In some cases the hæmorrhage from the pia mater is diffused over a wide area of the brain, in the subarachnoid space. When the latter is torn by the injury, blood is found in the subdural space.

After severe injuries to the brain, persons sometimes remain unconscious for several weeks, and then recover more or less completely. These are usually cases of laceration of the brain with hæmorrhage. The blood either becomes absorbed or replaced by scar-tissue. In some instances only the outer part of the extravasated blood becomes organized, the central part remaining liquid, and being absorbed and replaced by serum, leads to the formation of a cyst.

Hæmorrhage from the Middle Meningeal Artery.

The more usual condition which gives rise to this complication is a fissured fracture, affecting one or other parietal bone and crossing the anterior branch of the artery, though the hæmorrhage may come from any part of either branch, or even the main artery before its division. The anterior branch is so much more commonly the source of the bleeding that the operation is based on the probability of its being injured. Blood gradually accumulates between the skull and the dura mater and clots. The condition corresponds to what, when it occurs in the limbs, is sometimes termed a traumatic aneurysm. The first effect on the brain is to render the part of the cortex which is directly pressed on by the clot, anæmic. And since the blood escapes more vigorously at each pulse, and the shock is communicated to the surface of the brain, there is some mechanical irritation of the cortex, and there may be some spasm in the muscles of the opposite side of the body. As the accumulation increases there is formed a depression in the surface of the brain. This pressure tells first on the part of the brain immediately beneath the clot, and since this is usually

close to the motor area, there is frequently some paralysis of the limbs on the opposite side of the body. As more and more blood is effused, the increased intracranial pressure communicated to all parts of the cortex by the subarachnoid fluid compresses the superficial blood-vessels and flattens the convolutions, producing unconsciousness (*coma*), dilatation of the pupils, etc. Frequently the clot works its way round to the base of the skull and reaches the sphenoidal fissure, and, pressing directly on the third nerve, causes an additional degree of paralysis of the pupil on the side of the hæmorrhage.

In many cases of middle meningeal hæmorrhage, the stunning caused by the injury is recovered from, and the patient may appear to be not much hurt, but after some minutes or an hour or more, effects of middle meningeal hæmorrhage develop.

It is a matter of importance to distinguish clinically between cases of middle meningeal hæmorrhage, and cases of hæmorrhage from the pia mater. The latter are accompanied by greater injury to the brain, and hence unconsciousness is usually more prolonged, and the circulation and respiration are more likely to be interfered with. Again, since laceration of the brain is generally due to contrecoup, if there is paralysis it will be on the same side of the body as the chief injury to the scalp, not on the opposite side as is the rule in middle meningeal hæmorrhage. The two conditions sometimes, however, closely simulate each other. In one case, although the three stages—concussion, recovery, and unconsciousness—were very like those of a typical case of middle meningeal hæmorrhage, yet the surgeon, A. T. Norton, was able to diagnose hæmorrhage from the pia mater by the irregularity of pulse and respiration.

Cerebral Abscess and Meningitis.

Abscess in the brain or purulent meningitis may follow contusion or laceration of the brain without there being any wound; in some such cases the formation of pus is very insidious and may occur many months after injury. Commoner is suppuration after compound fractures, and may occur where the skull necroses in scalp wounds that are not kept clean.

In the same way it may arise in cases of syphilitic necrosis of the skull. In some cases of hæmatoma or mere contusion of the scalp and skull suppuration ensues without there being any external wound. In the scalp, as in other situations, œdema of the skin (here called "Pott's puffy tumour") is one of the first signs of deep suppuration. In such a case the suppuration may spread to the meninges or the brain. Not infrequently abscess of the brain is pyæmic, *e.g.* in cases of ulcerative endocarditis. In suppuration of the frontal, ethmoidal, and other air-sinuses meningitis, cerebral abscess, etc., not infrequently ensue. This is most commonly observed in cases of middle ear disease, and will be described under that head.

Cerebral Hernia. After compound fractures in which the brain is exposed by a tear in the dura mater, or after trephining in conditions such as cerebral abscess where there is inflammation going on in the brain, a portion of the brain is frequently caused by the increased intracranial pressure to protrude through the opening in the skull. The "hernia," as it is termed, is swollen by inflammatory changes and becomes covered with granulation tissue. If the inflammatory condition of the brain continues the patient dies, if it is removed the hernia ceases to enlarge, shrinks from cicatrization which joins that in the skin, and the wound thus scars over, the scar being adherent to the brain.

Tumours of the Brain.

Various growths of the skull bones and dura mater affect the brain, but growths originating in the pia mater and brain substance are usually indicated by this term. The more common tumours are tubercular masses, gummatous foci, and gliomata.

Tubercular Tumours are attached to the pia mater and project into the brain substance. They shell out readily, but are usually unsuited for operation because of their being multiple.

Gummata are commonly placed at the surface of the brain and implicate the pia mater and subjacent brain matter.

Gliomata form lobulated masses in the brain substance or

projecting from the surface. They are frequently cystic and then temporary relief may be obtained by drainage.

Symptoms. In addition to headache, vomiting, and optic neuritis, Jacksonian epilepsy, that is, spasmodic contractions beginning in one limb or group of muscles, is a frequent symptom of cerebral growths.

Suppurative Middle Ear Disease.

Catarrhal inflammation frequently spreads from the naso-pharynx along the Eustachian tube to the tympanum. In

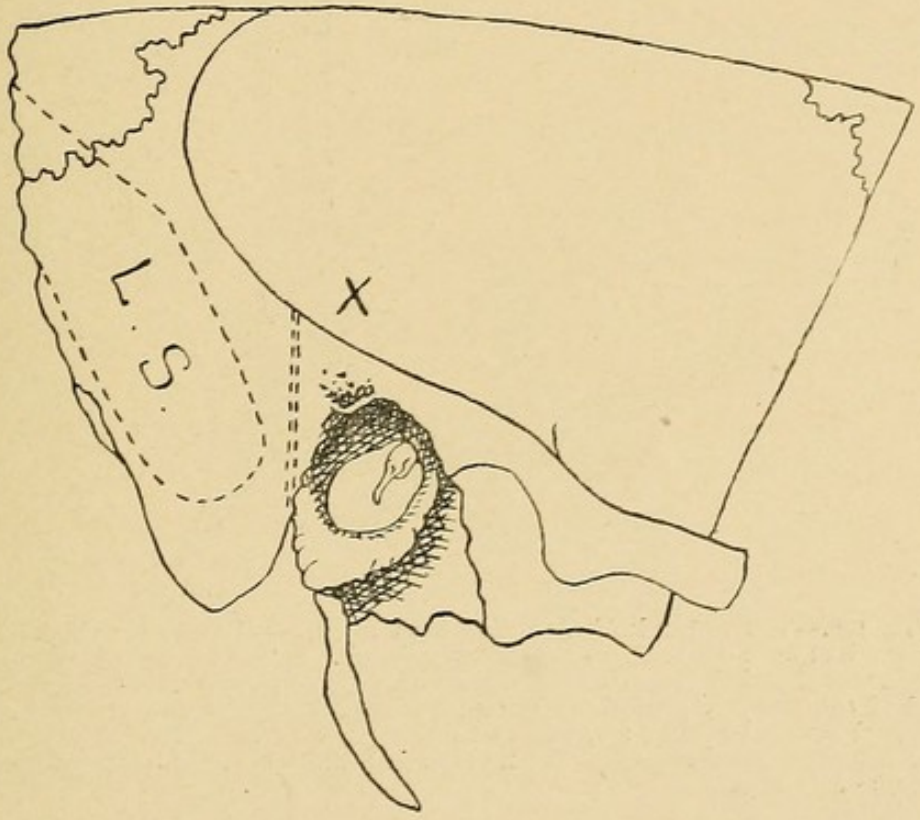


Fig. 129.—The outer surface of the right temporal bone of a boy, aged 16. In front of a perforated depression just behind the junction of the roof and posterior wall of the external meatus is the small suprameatal spine (the letter X is placed a little way above the spine). The double interrupted line shows the base of Macewen's suprameatal triangle; the single interrupted line corresponds with the position of the lateral sinus. Nat. size.

common cold in the head the effusion usually remains of a serous character and the inflammation subsides. In children of feeble resisting power, however, and especially those who suffer from adenoids, suppuration is liable to occur from the catarrh assuming a purulent character. In the infective fevers,

especially influenza, measles, scarlet fever, and diphtheria, suppurative catarrh of the middle ear is common. Any of the pyogenic organisms may be found in the pus. The tympanum becomes distended with pus which shows no tendency to escape into the pharynx by the Eustachian tube, but on the contrary causes perforation of the tympanic membrane and escapes by the external meatus. After this event the suppuration may cease and the perforation may heal, or granulation tissue may fill the tympanum, and in cicatrizing

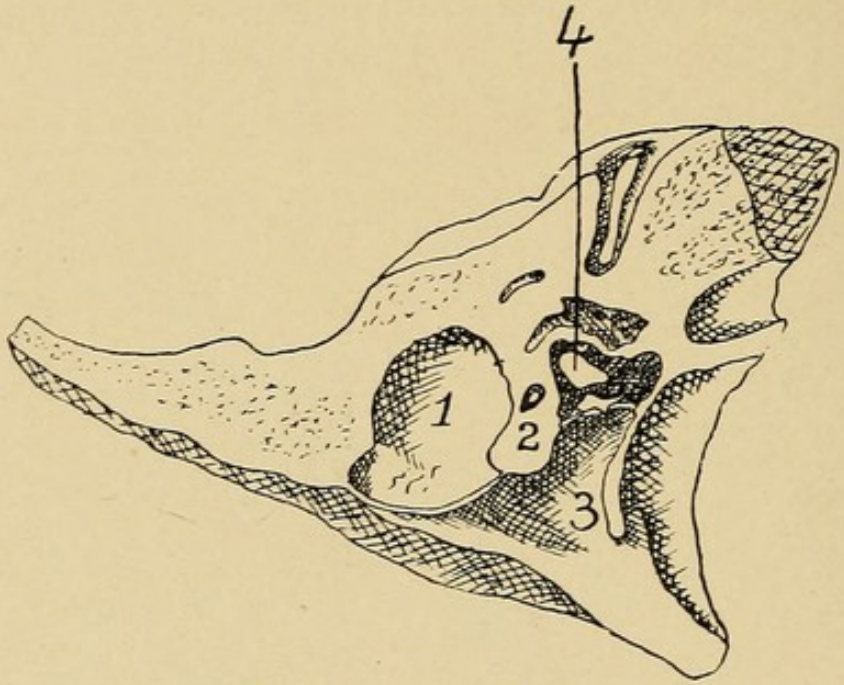


FIG. 130.—The upper half of a section of a temporal bone in which the antrum had been opened two years before death. 1, The excavated antrum, which was closed externally by a fibrous membrane (scar); 2, the posterior wall of the meatus, containing the aqueduct of Fallopius and (deeper) the external semicular canal; 3, marks the entrance of the external meatus; 4, shows the normal communication between the antrum and the tympanum. (801 c.) Nat. size.

may cause adhesion of the tympanic membrane to the inner wall of the tympanum; or the suppuration may continue. This may be accompanied by the formation of prominent granulation-tissue growths—the *polypi* which project through the tympanic membrane; or it may be due to the infection lingering in the mastoid antrum or its prolongations, the mastoid cells.

Syphilitic and Tubercular Disease of the Middle Ear. In cases of chronic suppuration which do not yield to regular

syringing combined with inflation from the nose (Politzerization), the pus should be examined for tubercle bacilli, since extensive tuberculosis of the mucous membrane and bones sometimes occurs with very little disturbance. Again in syphilis, especially if it is congenital, extensive necrosis, more especially of the petrous bone, is not uncommon, and after a fœtid discharge has lasted for some years a considerable portion of the bone of the internal ear may come away.

The Graver Complications of Middle Ear Disease.

If polypi, syphilis, tubercle and a few other conditions can be excluded in an obstinate case of aural suppuration, the most

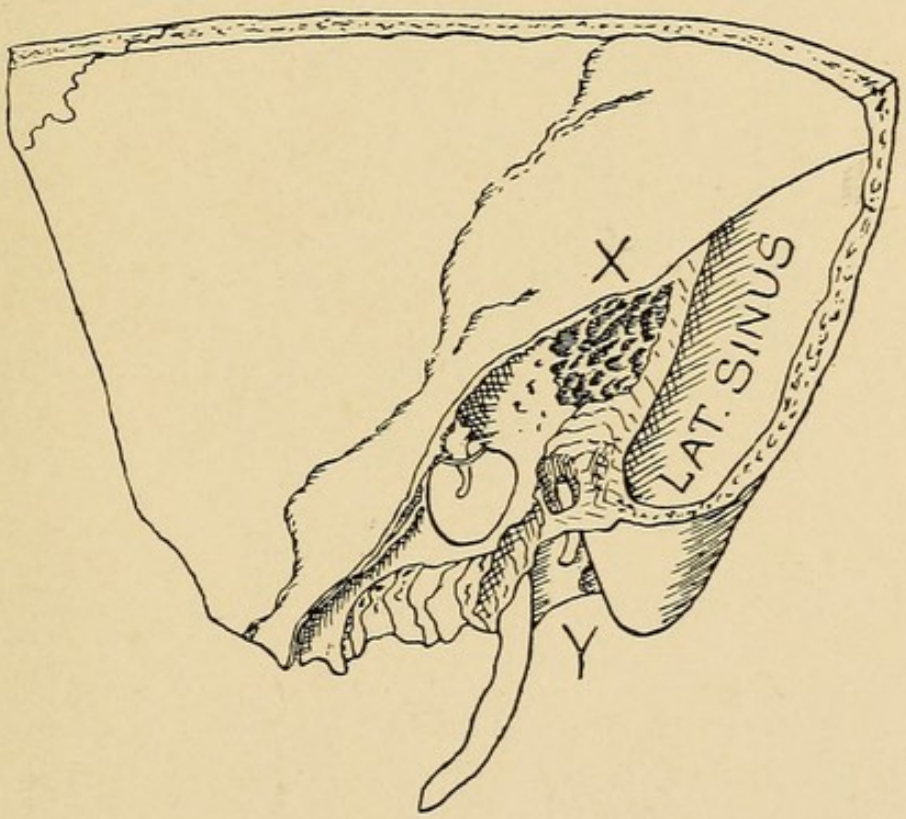


FIG. 131.—Inner aspect of a temporal bone from which the petrous part has been removed. Below X is the mastoid antrum, above Y is a portion of the aqueduct of Fallopius and the facial nerve. The tympanum, with the inner surface of the membrane, the malleus and chorda tympani, and the Eustachian tube, are also seen.

probable inference is that there is disease of the antrum, and this cavity should be opened from without to prevent still farther complications. The results of this operation are well shown in Fig. 130.

A brief examination of the anatomy of the middle ear and mastoid antrum will render the comprehension of the complications of middle ear disease more easy (see Figs. 129 and 131).

The time required for the production of these graver com-

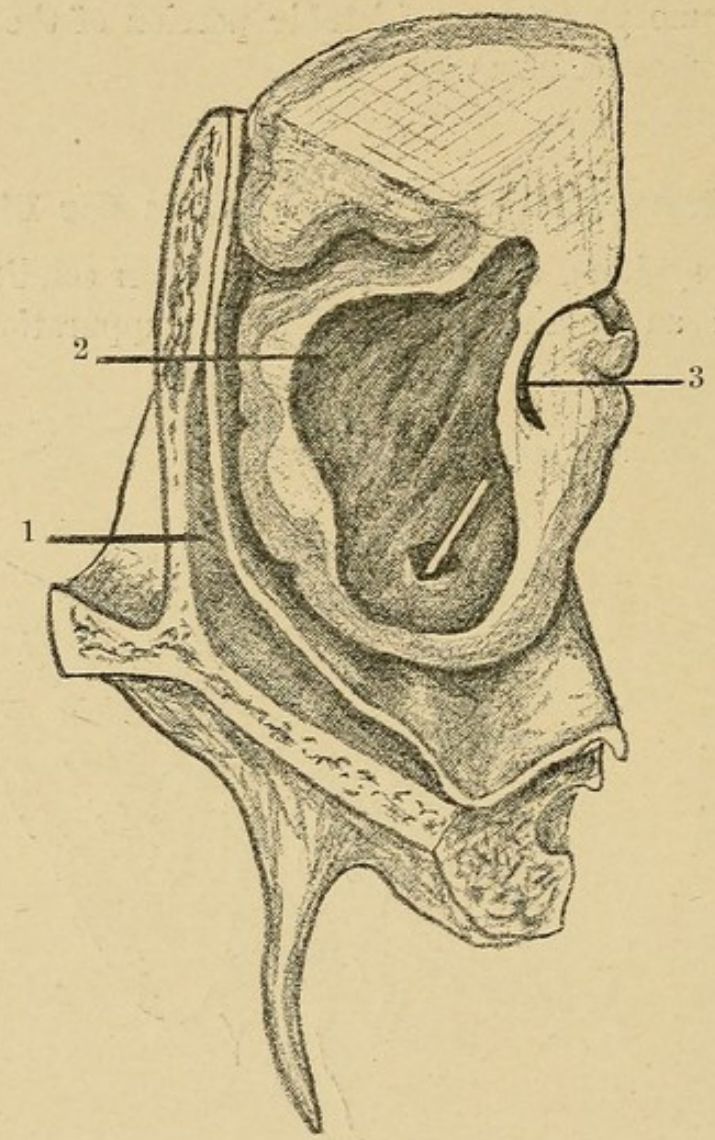


FIG. 132.—Vertical section of the fore part of a temporal bone, with the temporo-sphenoidal lobe of the brain. 1, Extra-dural abscess; 2, cerebral abscess; 3, descending cornu of the lateral ventricle. A glass rod has been passed through a communication between the cerebral and extra-dural abscesses. (746 c.)

plications of middle ear disease is usually considerable: on an average above two years. But in cases of scarlet fever the process is frequently more rapid.

The formation of air-cells in the mastoid process by extension of the mastoid antrum occurs at an early age; thus, at three

and a half years the author[1] has found the mastoid process to be wholly occupied by air-cells.

If the infection spreads through the posterior wall either an extradural abscess, meningitis, cerebellar abscess, or septic thrombosis of the lateral sinus, or a mixture of some of or all these conditions, will result.

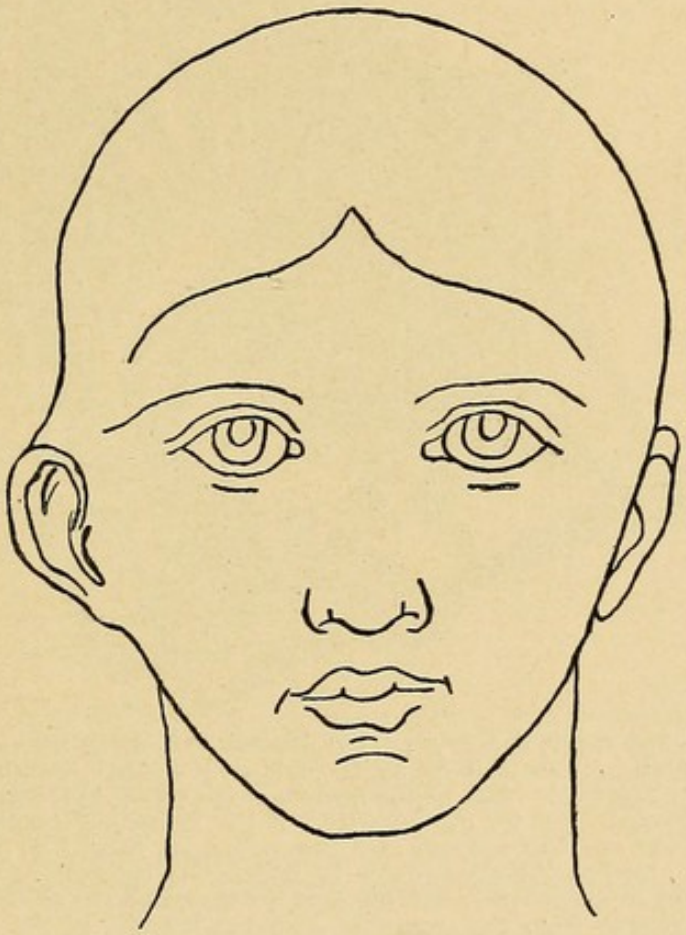


FIG. 133.—Outline of a face showing the displacement of the right ear in a case of subperiosteal mastoid abscess.

The study of an example will make this more clear. Fig. 134 represents a horizontal section of a temporal bone in a case of cerebellar abscess, with part of the cerebellum containing an abscess.

The formation of abscess of the temporo-sphenoidal lobe is similar to that of cerebellar abscess. It may occur either through the roof of the antrum or that of the tympanum; the bone may either be entirely removed by granulations or

[1] See a paper by the author on the "Anatomy of the Temporal Bone," in the *Journal of Anatomy and Physiology*, vol. xxvii., 1893.

it may perish rapidly and remain as a thin dead partition between the middle ear and the abscess (see Fig. 132).

If the suppuration with caries or necrosis extends through the outer wall of the antrum, a subperiosteal abscess and

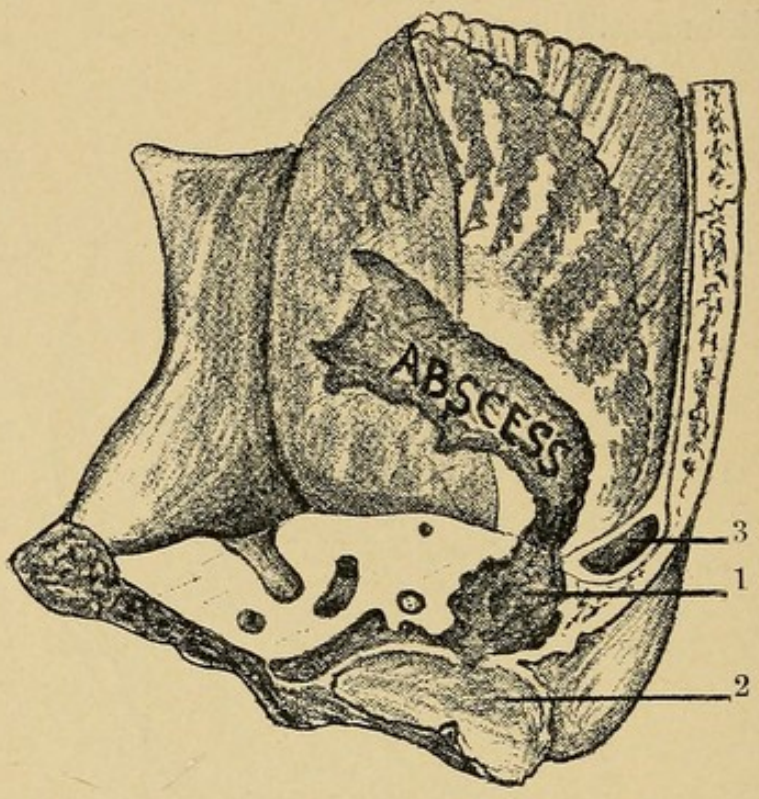


FIG. 134.—The mastoid antrum (1) is dilated and opens into the external auditory meatus (2) as well as by its enlarged normal opening into the tympanum. The white ridge at the bottom of the external meatus (2) marks the sulcus tympanicus, for the insertion of the tympanic membrane. The bony wall between the antrum and the cerebellar fossa has been eroded through, as also has the dura mater to which the cerebellum has become adherent, and suppuration extending into it has caused the abscess. In this case the discharge from the ear came from the interior of the cerebellum, until the passage became blocked by granulations. How readily the lateral sinus (3) may be infected may be readily seen. If the cerebellum had not become adherent to the dura mater, septic meningitis, instead of cerebral abscess, would have occurred. From a boy who had pain in the head, delirium, retraction of head and abdomen, irregular breathing, the hydrocephalic cry, and tâche cerebrale. He vomited twice. His temperature reached 102·5° and afterwards fell below normal. (746.)

subsequently a sinus forms over the root of the mastoid process. The swelling of such an abscess causes a peculiar displacement of the ear (see Fig. 133).

One feature brought out in Fig. 130, where the bone had been sawn in an oblique plane so as to pass through both antrum and tympanum, is the proximity of the facial nerve to both tympanum and antrum. This anatomical feature is still further illustrated in Fig. 135.

It is not surprising that facial paralysis sometimes supervenes in acute aural suppurations, and occasionally follows operations on the antrum.

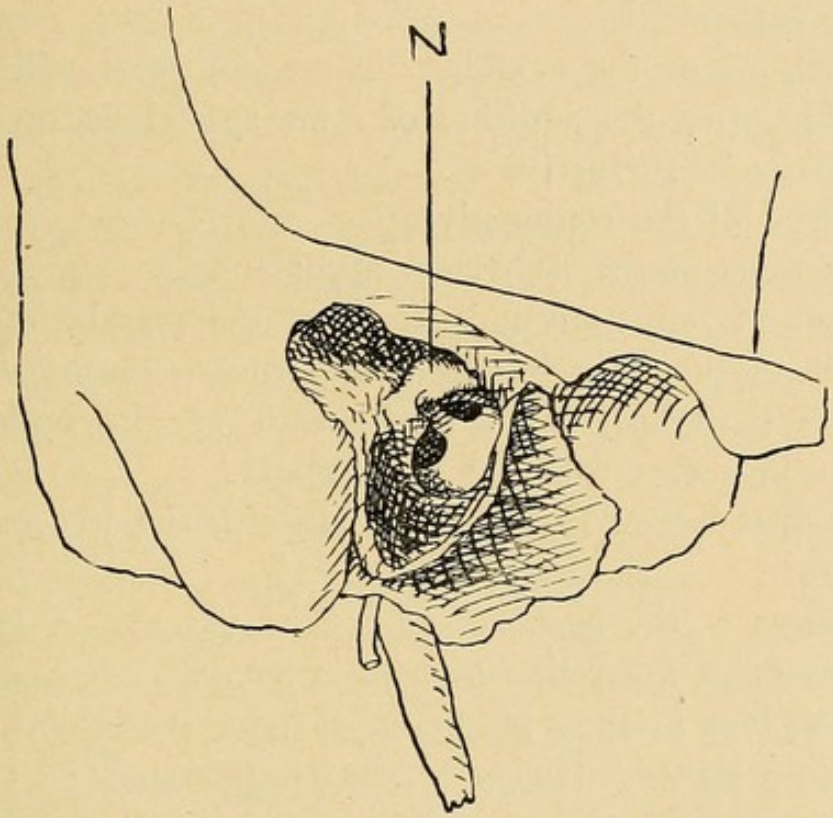


FIG. 135.—A temporal bone in which the antrum has been opened and thrown into the tympanum. The guide-line (N) marks the aqueduct of Fallopius. The posterior wall of the meatus has been cut away lower down than is done in operating.

To recapitulate the graver complications of middle ear suppuration:

1. Facial paralysis from inflammation within the aqueduct of Fallopius.
2. Suppuration in the mastoid antrum and cells.
3. Subperiosteal mastoid abscess or a sinus resulting therefrom.
4. Extradural abscess.
5. Septic thrombosis of the lateral sinus and pyæmia.
6. Suppurative meningitis.
7. Cerebral or cerebellar abscess.

Of these, 5 and 6 are the most fatal.

DISEASES OF THE EYE.

The affections of the eye may be considered in separate subdivisions, such as—

1. Affections of the eyelids. These are practically diseases of the skin with the addition of some special features due to peculiarities of structure.

2 Diseases of the conjunctiva.

3. Affections of the lachrymal apparatus.

4. Errors of refraction and defects of the muscles of the eye.

5. Diseases of the sclerotic and cornea—fibrous structures continuous posteriorly with the sheath of the optic nerve, which is continuous with the dura mater.

6. The disorders of the choroid coat with the iris and ciliary body.

7. Diseases of the lens.

8. Diseases of the retina and optic nerve.

The pathology of these groups is an extensive subject, and one of great importance. Owing to the transparency of the parts of the eye used for the transmission of light the pathology of many of the affections can, by means of the ophthalmoscope, be studied during life better than similar processes in any other part of the body. In this work only a few conditions of special importance to the general surgeon can be dealt with. They are: (1) One of the consequences of injury: sympathetic ophthalmitis; (2) gonorrhœal and syphilitic affections; and (3) glaucoma.

Sympathetic Irritation and Sympathetic Ophthalmitis. These conditions are induced in a previously sound eye by wounds of the other eye-ball in the "ciliary-region," that is, the part that comprises the iris and ciliary body.

The symptoms. The sympathizing eye in *sympathetic irritation* becomes *weak* and *irritable.* That is, after looking attentively at any object for a short time accommodation fails, the blood-vessels on the ciliary region of the sclerotic tend to be injected, and lachrymation is frequent and profuse. There is no plastic lymph on the iris or elsewhere. These symptoms tend to recur.

Sympathetic Ophthalmitis begins on an average about two months after injury to the sound eye, but it may set in any time after the first month, and may be observed as late as, or later than, the third month. Sometimes it begins as *irritation* or the onset may be insidious. The iris becomes altered in colour (muddy) from the effusion of lymph, the blood-vessels of the front part of the sclerotic (ciliary zone) are congested. The condition is an iridocyclitis, with a tendency to pass into panophthalmitis. Adhesions form between the iris and the lens (synechiæ), and flakes of lymph derived from the ciliary body and the choroid are discharged into the vitreous. The simplest explanation of the affection is that it is a lymphangitis which spreads from the injured eye by the communication that exists between the lymphatics of the two eyes within the sheath of the optic nerve by way of the optic chiasma. Irritation may be due to toxic products, ophthalmitis to pyogenic organisms. Preventive measures, such as antiseptic applications to the eye first injured (exciting eye), and, where the injury is such that there is no hope of a useful eye remaining, speedy removal are in the first place indicated.

If sympathetic irritation sets in, rest to both eyes by the use of atrophine and the exclusion of light is indicated, and if the exciting eye is beyond recovery, it should be removed. Excision of the exciting eye quickly removes the irritation.

If sympathetic ophthalmitis is present, the exciting eye in some cases retains a better vision than the sympathizing eye. In these circumstances the rule is not to excise the exciting eye unless all power of sight is lost.

Syphilitic Affections. Those that require the most prompt attention arise during the secondary period of the disease, *viz.* syphilitic retinitis and iritis.

Syphilitic Retinitis generally occurs between six and eighteen months after infection. Impairment of vision draws the attention of the patient to the affection. On ophthalmoscopic inspection the retina is discoloured grey, sometimes with yellow flecks; some of the latter may float in the vitreous.

Syphilitic Iritis possesses the characters common in cases of iritis—impaired mobility and altered colour of the iris; impaired

vision and congestion of the "ciliary zone." The special features of syphilitic iritis are the smaller degree of pain and the greater amount of plastic effusion that occurs in this as compared with other forms of the disease. Small gummata may form in the iris, and more or less adhesion of the iris to the capsule of the lens is usually observed. Both iritis and retinitis may be symmetrical, and the possibility of blindness resulting indicates the most prompt mercurial treatment.

Syphilitic Keratitis. In congenital syphilis inflammation of the cornea may show itself, usually between the ages of six and fifteen years. Some congestion of the ciliary zone and excessive flow of tears (lachrymation) are the first indication of the morbid process. Inflammatory infiltration shows itself by the cornea assuming a "ground glass" appearance. If the infiltration becomes vascularized by the ingrowth of blood-vessels from the sclerotic, it assumes a pink appearance, "salmon-patch." The keratitis may be accompanied by inflammation of the neighbouring part of the sclerotic and of the iris. Under suitable treatment long continued, great improvement may be obtained.

Syphilitic Choroiditis occurs in both acquired and inherited syphilis. It causes patches of infiltration in the choroid, and since the choroidal pigment is lost in the patches, the latter are seen on ophthalmoscopic examination as white or yellowish white areas. In severe cases sight may be greatly impaired.

Gonorrhœal Sclerotitis is an inflammation of the sclerotic tending to spread to the iris and neighbouring parts. It may be regarded as one of the evidences of gonorrhœal rheumatism. It is a very obstinate affection, and tends to take a chronic course. Exclusion of light and the use of atropine are amongst the measures that it is necessary to employ.

Gonorrhœal Conjunctivitis or Ophthalmia. In the conjunctiva the gonococcus produces an intense inflammatory reaction which begins with itching and injection of the conjunctiva, then œdema of the conjunctiva (chemosis) and of the lids follows. The cornea may become hazy and even slough, or it may escape altogether. Prompt and energetic treatment is required. The suppuration, if left untreated, lasts, like that of the urethra, about six weeks. Gonococci are present in the pus. Newly-born babies and young

adult men are the commoner victims. Washerwomen and others may contract it.

Glaucoma is an affection marked by increase of tension within the eye. It may be compared with hydrocephalus. In the latter condition a block in the ventricular passages, either at the foramen of Magendie or at the foramen of Monro, or at any intervening point, leads to the collection of fluid in the lateral ventricles, etc. So in glaucoma, if the spaces between the meshes of the *ligamentum pectinatum* (spaces of Fontana) are blocked, the escape of fluids from the eye into the lymphatics of the sclerotic is obstructed. Glaucoma may thus be produced by adhesion of the iris to the capsule of the lens, or by swelling of the lens as after "needling." This is called *secondary* glaucoma. When the affection begins without obvious cause it is termed *primary.* In the course of time the increased intraocular pressure causes the weakest part of the sclerotic (the lamina cribrosa) to bulge backwards, thus producing "cupping of the optic disc." Pressure on the retinal veins where they pierce the sclerotic at the lamina cribrosa causes them to dilate, and they pulsate owing to the communicated pressure at each arterial pulsation. The same pressure narrows the arteries, and so diminishes the supply of blood to the peripheral part of the retina that it becomes insensitive to light—hence the narrowing of the field of vision that is observed. The lens is usually pushed forwards, causing shallowness of the anterior chamber. The cornea and other media become less translucent than normal. The pupil is dilated and sluggish. When the symptoms are rapid (*acute glaucoma*) there are pain and flashes of light with congestion of the ciliary zone. Blindness may result in a week or two. Less rapid cases are termed "subacute" and "chronic." In all cases of glaucoma the use of atropine must be rigidly avoided since it increases the intraocular tension, and may change a chronic into an acute case. In acute cases iridectomy is usually required.

CHAPTER XXIII.

INJURIES AND DISEASES OF THE SPINAL COLUMN AND CORD.

FRACTURE, dislocation, and diastasis are observed in the spinal column. The injuries may be found combined in the same case. Thus in the dorsal region the body of a vertebra may be fractured whilst the articular processes are dislocated from each other.

The commonest cause of these injuries is indirect violence in the form of forcible overflexion of the spinal column. Thus, if a person falls on the head the cervical part of the column is bent forcibly forwards, and the spine may be broken. A fall on the shoulders may produce the same lesion in the dorsal region. In the lumbar region various accidents, such as alighting on the feet from a height, may give rise to fracture. In these cases, from the nature of the injury, the upper part of the column is usually displaced forwards, and, as is shown in Fig. 136, the body of one of the vertebra may be compressed into a wedge-shape, and sometimes it is forced backwards so that it presses upon the cord. In this figure the spinal cord is shown to be crushed between the body of the highest vertebra of the lower segment and the arch of the lowest vertebra of the upper segment. In some cases the fracture is produced by direct violence, and then one or more arches may be depressed upon the cord. In the cervical region, from the nature of the articulations, pure dislocations are not uncommon. When this happens between the atlas and the axis, the odontoid process is displaced backwards and presses on the medulla, causing instant death. In the dorsal region

fracture of a vertebral body with dislocation of an articular process is not uncommon. When a person lives long enough after a fracture of the spine for union of the bones to occur, callus is formed as after other fractures. Diastasis is commonest in the cervical region, and if the cord escapes injury at the time,

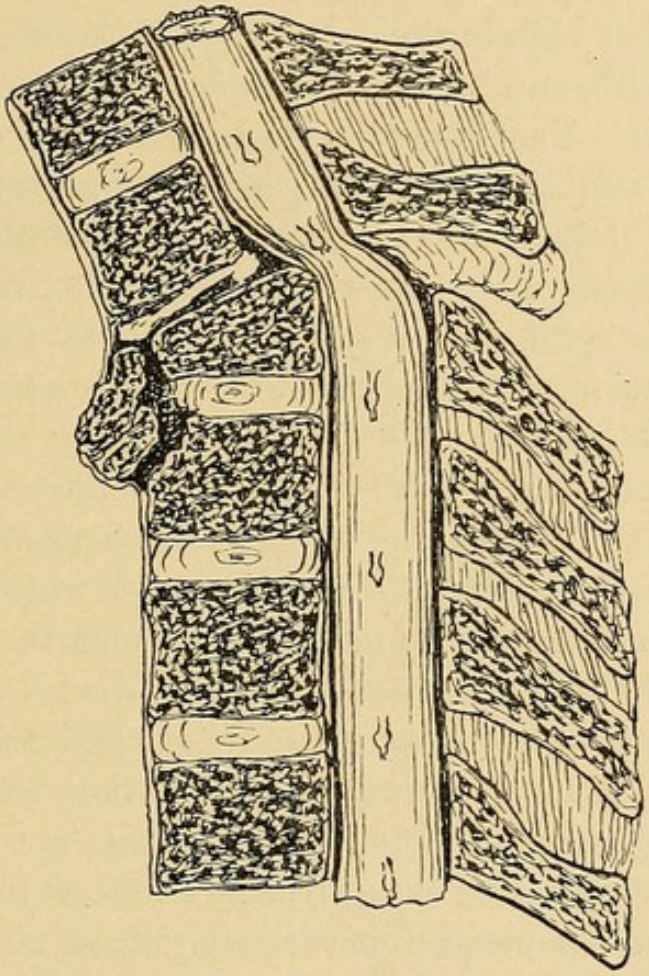


FIG. 136.—Vertical section through the dorsal region of a spinal column showing a fracture. The cord is shown in surface view, within the dura mater. The uppermost vertebral body of the lower segment of the column is compressed to a wedge-shape, and portions of the anterior parts of this and the next vertebra below have been chipped off. The cord is crushed between the arch of the lowest vertebra of the upper segment of the column and the posterior part of the highest body of the lower segment.

the weakness owing to rupture of ligaments may, at some time after the original accident, lead to a more extensive displacement, and fatal injury to the cord.

Surgical Affections of the Spinal Cord.

Traumatic Affections. The chief of these are caused by injuries of the spinal column. The importance of these injuries depends upon their being accompanied by injury to the cord.

If the latter is so crushed that its physiological continuity is completely abolished, no repair takes place. Complete motor and sensory paraplegia and abolition of deep reflexes is the result. If such a total transverse lesion occurs in the cervical spine above the origin of the phrenic nerves, rapid death ensues from arrest of respiration. If the injury is below the origin of the phrenics, but high in the dorsal region, the costal respiration is interfered with, and bronchitis and *broncho-pneumonia* are liable to cause death. The lower cervical and upper dorsal regions of the cord contain important parts of the temperature-regulating centres, injury to which is not infrequently followed by fatal *hyperpyrexia*. In complete transverse lesions of the cord paralysis of the sphincter ani and of the bladder form part of the paraplegia, the former showing itself by *incontinence of fæces*, the latter by *retention of urine*, necessitating the use of the catheter, by means of which bacterial contamination of the urine frequently occurs, setting up an intractable *cystitis* which may demand perineal drainage for its relief, and which is a common cause of death by spreading to the kidneys, or through the wall of the bladder to the peritoneum. *Bed sores* are of the commonest occurrence in paraplegia. When the lumbar centres are cut off from the controlling action of the brain, persistent *priapism* is observed. The deep reflexes are lost and the superficial reflexes are exaggerated below a total transverse lesion. When paraplegia immediately follows an injury to the back, and there is no deformity of the spine present, the condition has been attributed to concussion of the spine, comparable to concussion of the brain. In these cases, however, as H. W. Page has pointed out, there is probably always a definite pressure-lesion, and I have found on post-mortem examination in one such case, a complete crushing of the cord from diastasis. Besides the damage of the cord due to pressure of displaced bones, there may be pressure from hæmorrhage. This is seen most commonly on the outer surface of the dura mater, but occasionally it comes from the vessels of the cord.

Paraplegia from pressure of Pus is a common complication of tubercular disease of the vertebræ. The paralysis usually affects motor nerves, including those of the sphincters, whilst

the sensory function remains almost unimpaired. With rest alone this form of paraplegia is frequently recovered from, but it may require drainage.

Syphilitic Meningo-myelitis, resulting in thickening of the pia mater and compression of the cord, is a rare affection. Paraplegia may arise from growths of the cord itself (of these gliomata are the commonest); or of the nerve-roots, as shown in Fig. 85; or of the vertebræ, etc. Hydatid cysts in the spinal canal are an occasional cause of paraplegia.

Trephining the Spine, or Laminectomy. In fractures where there is evidence that the cord is completely crushed, there is no hope of restoring its functions; but when paraplegia is incomplete, there is a possibility that by removing the lamina of the vertebra above the fracture, and if necessary other laminæ, a better result will be obtained. If a fracture is due to direct violence and a lamina is displaced forwards upon the cord, the operation is also indicated. When paraplegia comes on gradually within an hour or two after an injury, there is a probability that it is due to hæmorrhage, and trephining should be performed as in cases of middle meningeal hæmorrhage. If improvement does not follow rest in cases of paraplegia from tuberculosis of the vertebræ, drainage may be effected by laminectomy; but the same end can be gained, and with less weakening of the spine, by working at the side of the vertebræ, and if necessary removing a transverse process and the neck of a rib (costo-transversectomy). In some growths, etc., within the spinal canal laminectomy is also indicated. An example of a favourable case is shown in Fig. 85.

After a sprain of the spine I have observed a subacute ascending spinal meningitis. *Railway spine*[1] is a condition of nervous weakness which is shown by weak pulse, profuse sweating, dilated pupils, and irritable weakness of the brain, and is not infrequently observed in those who have been in railway collisions.

Diseases and Deformities of the Spine.

Tuberculosis of the Spine. It is in the spongy parts of vertebræ, that is, the vertebral bodies, that the tubercular infec-

[1] See H. W. Page, *Injuries of the Spine and Spinal Cord.*

tion first shows itself. Moreover, it is in the anterior far more frequently than the posterior parts of the bodies that early tubercular changes are found when opportunities for post-mortem examination occur in suitable cases. This may be accounted for by the greater amount of wear and tear experienced by the anterior portions of the vertebral bodies as compared with the posterior, the movements of flexion being

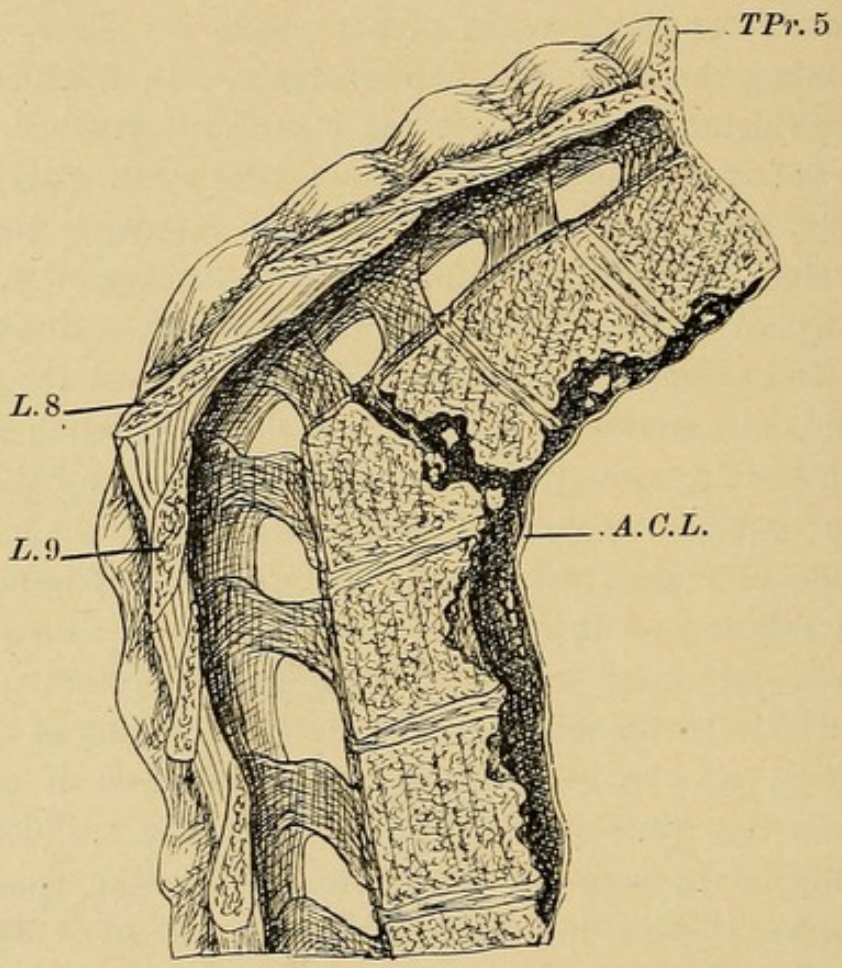


FIG. 137.—Section of a spinal column affected by tubercular caries. The section passes through the articular processes and the laminæ. Although the body of one vertebra (the eighth dorsal) has been almost entirely destroyed, there is no narrowing of the spinal canal. The anterior common ligament is raised up by caseous pus.

more extensive and more frequently called into play than those of extension. Though the disease may appear at any period of life, even in advanced age, it usually makes its appearance in childhood. The effects on the bones are the same as in tubercular osteitis elsewhere. The destruction of tissue extends from the vertebral body to the nearest intervertebral discs, and thence to adjoining vertebræ. The rarefaction and absorption

of bone causes the vertebral bodies to be compressed from above downwards by the weight of the parts above, and produces more or less sharp bend marked by a backward projection of the spines, or, as it is termed, *angular curvature*. The middle of the projection is opposite the centre of the affected area of the bodies. If the disease continues, the spines of the vertebræ tend to separate in proportion as the vertebral bodies give way; but the strong supraspinous and other ligaments and the attached muscles resist this tendency, and the result is that instead of opening out, the spines frequently bend one over the other, as shown in Fig. 140.

The articular processes remain in contact with one another and so prevent the posterior part of the spinal column being sharply bent. This prevents, in the vast majority of cases, any compression of the cord from narrowing of the spinal canal. By comparing a preparation of a spinal column affected by tubercle with one that has been fractured, it is seen how differently the canal is affected in average cases of the two conditions (compare Figs. 136 and 137).

Bony fusion of the arches and spines sometimes occurs from simple osteoplastic periostitis set up around the tubercular foci. This leads to bony fusion of the arches, and serves a useful purpose by strengthening of the arches and protecting the diseased parts of the spine from movement, *i.e.* serves as a splint to secure rest (Fig. 140).

If the tubercular process is of sufficient activity there is caseation, and the formation of thin pus and sequestra. The first pus is formed beneath the anterior common ligament, which may be separated from several vertebræ (see Fig. 137). In a few cases the ligament is stripped from nearly the whole vertebral column. More commonly the matter finds its way outward to the sides of the vertebræ, whence it passes in one of several directions. In the lumbar region the pus cannot easily escape entering the psoas muscle (see Fig. 138), which arises from the sides of the bodies and from the transverse processes of the lumbar vertebræ.

But it is not only in caries of those vertebræ from which the muscle arises that the psoas is converted into an abscess. Pus

from carious vertebræ in the lower dorsal region frequently enters the sheath of the psoas by gravitating downwards and passing beneath the internal arcuate ligament. When pus has once entered the psoas the muscle is gradually infiltrated with inflammatory matter, and to a considerable extent destroyed. Calcification of the walls of a psoas abscess is not uncommon.

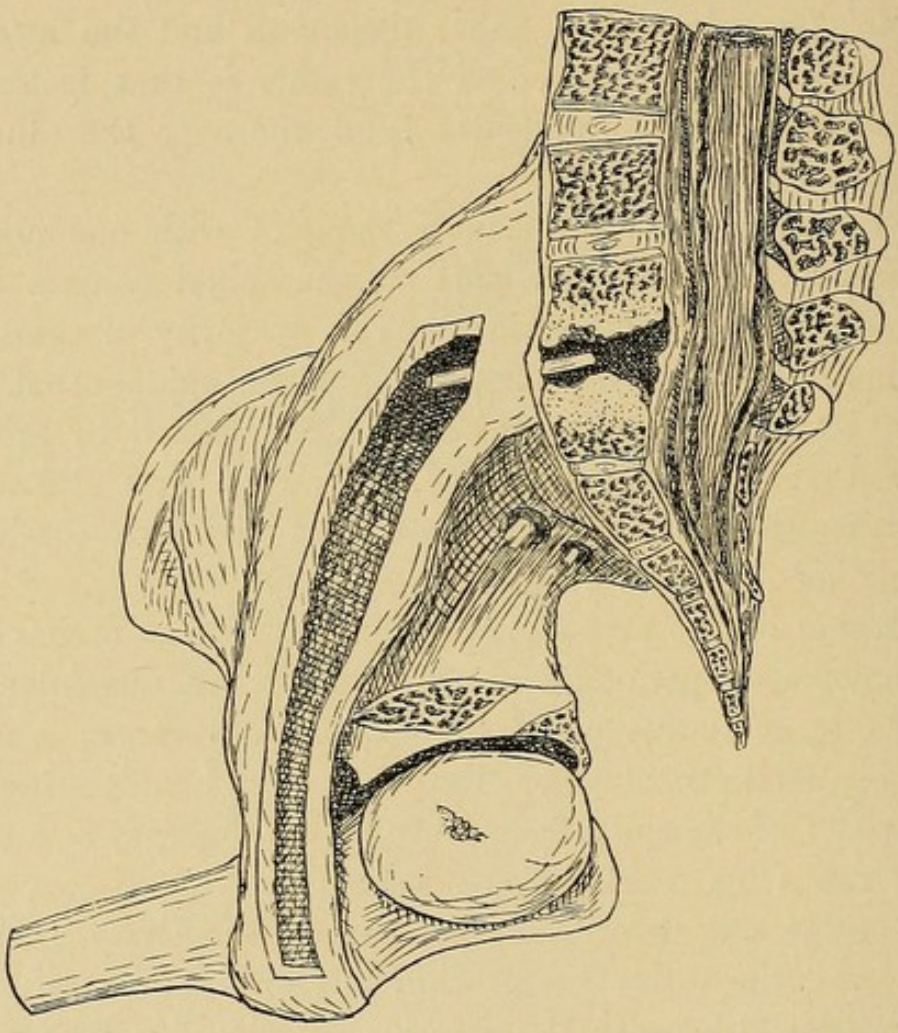


FIG. 138.—A psoas abscess. There is a cavity bounded above by the remains of the fourth lumbar vertebra, below by those of the first sacral. The fifth lumbar vertebra has been destroyed by the disease; in front and behind the cavity is closed by the common ligaments. This cavity, as is shown by a white glass rod, communicates with large psoas abscess. The bone on each side of the cavity in the spine is sclerosed. In this specimen the thickened sheath of the psoas and the remains of muscular tissue contained calcareous material. From a child aged five years. (266.)

When a psoas abscess has reached the point of insertion of the muscle the matter often passes beyond the psoas, either backwards or downwards. Cases have been known in which the abscess has reached as far as the foot.

Spinal Abscesses in Other Situations. In caries of the atlas

and axis pus frequently collects behind the pharynx, see Fig. 139 (*retro-pharyngeal abscess*), it may then open into the pharynx, or make its way between the muscles and point at the posterior border of the sterno-mastoid muscle. In caries of the lower cervical vertebræ pus may find its way into the axilla. A dorsal abscess sometimes forms, the pus following the course of the dorsal nerves. This is especially common in the case of the upper dorsal vertebræ. The iliacus muscle either together with or apart from the psoas may be converted into an abscess cavity by a spinal abscess tracking into it. From the lower lumbar and sacral vertebræ a pelvic abscess may result. The pus then usually follows the course of the sacral plexus, and thus leaves the pelvis by the great sacro-sciatic foramen and points as a gluteal abscess; or it may follow the course of the rectum into which it may open. Caries of the lower part of the sacrum or of the coccyx frequently forms on ischio-rectal abscess, and within the neighbourhood of the anus sinuses, which may be mistaken for rectal fistulæ, may result.

The Course of Spinal Tubercular Abscesses is usually slowly progressive until the abscess opens on a surface of the body. Then pyogenic and putrefactive bacteria obtain access to the abscess cavity, and more rapid suppuration with fever, and in the course of time lardaceous disease, are common results. Also, as soon as the abscess becomes septic, suppurative meningitis, etc., may occur (see Fig. 139). Hence the utmost care is taken to prevent septic infection when such abscesses are opened by the surgeon.

Healing of Tuberculosis of Vertebræ. Under proper management this occurs in a fair proportion of cases. Then reparative material is formed not only between the vertebral arches in the neighbourhood of the disease, but new bone may form from the remains of the diseased vertebral bodies themselves (see Fig. 140). Embedded in this new bone islands of caseous matter are usually to be found.

Effects of Spinal Tubercle on the Cord. As has been stated above, the gradual yielding of the vertebral arches usually prevents any pressure on the cord by the diseased vertebral bodies. When the process is rapid, however, and suitable

treatment is not applied, this occasionally occurs and leads to permanent paraplegia.

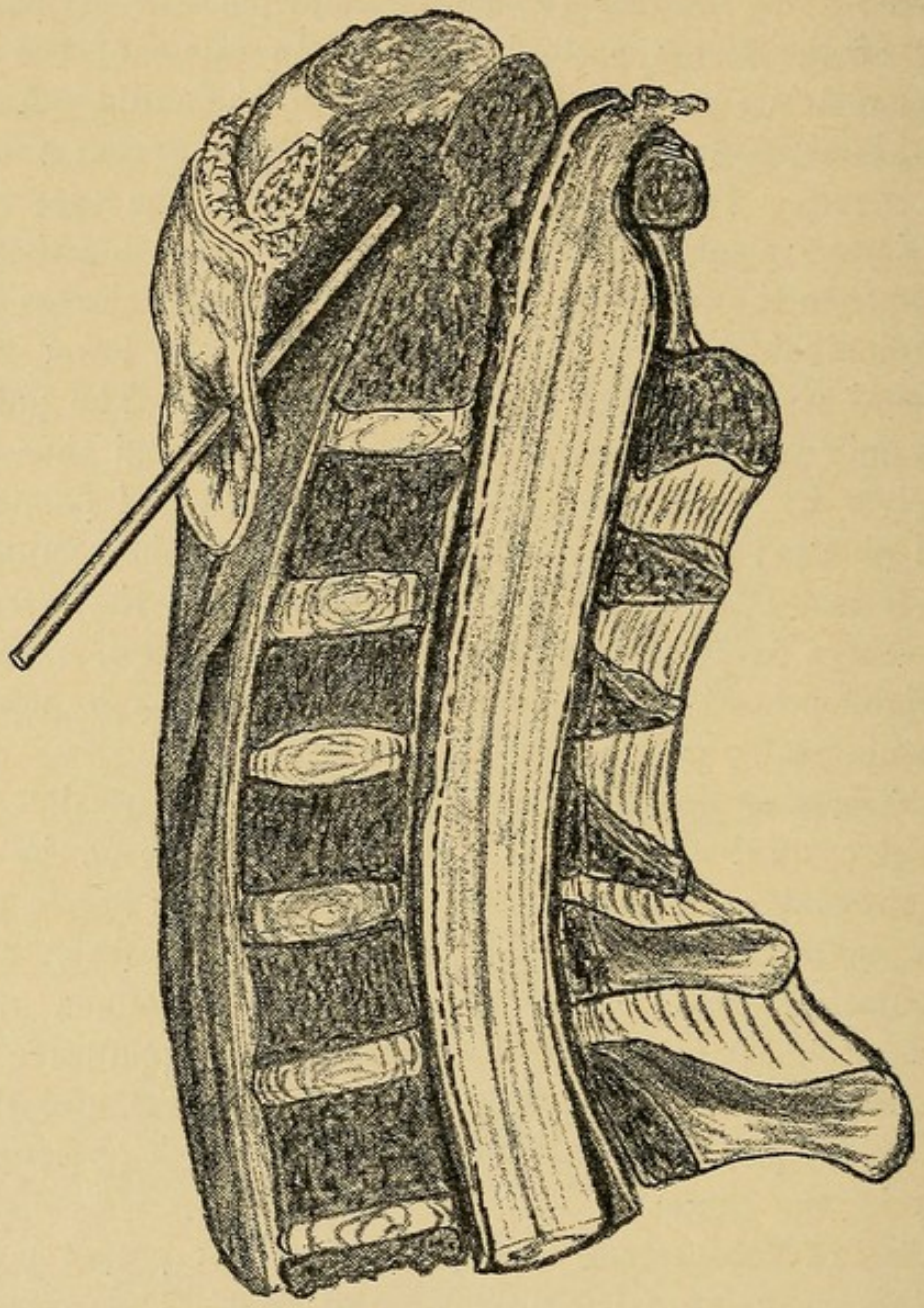


FIG. 139.—Atlo-axoid disease. The atlas is displaced ¾ inch forwards, and the uppermost part of the cord is compressed between the posterior arch of the atlas and the odontoid process. The glass rod marks a perforation in the posterior wall of the pharynx, where a retropharyngeal abscess opened. There was pus between the vertebræ and the dura mater, and the latter is adherent to the cord from meningitis. From a girl aged 19 who, from the age of 7, had complained of pain at the top of the head, and who had been treated for neuralgia. There was marked backward projection of the spine of the axis and a retropharyngeal abscess. Cheyne-Stokes respiration developed before death. (252 A.)

Paraplegia is more commonly the result of pressure by pus, etc., on the outer surface of the dura mater affecting the cord. In such cases, from the greater facility with which the motor

function of nerves is abolished, there is motor paralysis before the sensory function shows any change, and in many cases there is never any interference with sensation. Motor paraplegia is usually recovered from under suitable treatment. When the atlas and axis are the seat of tuberculosis the other cause of pressure on the cord frequently arises. The ligaments being softened the weight of the head carries the atlas forwards, and the cord may be pressed between the posterior arch of the atlas and the odontoid process of the axis (see Fig. 139).

The nerve trunks when surrounded by caseous matter become inflamed and cause pain, which is interpreted by the patient as coming from the skin to which the nerve is distributed. The irritation of the nerves in the immediate neighbourhood of the disease gives rise to pain and tenderness, and the effort of the muscles to prevent movement of the diseased parts causes the spine to be held rigid even in the early period of the disease. Pain, tenderness, and rigidity are the three first symptoms of disease, and frequently precede the formation of any marked angular projection.

Under suitable treatment a fair proportion of cases of tuberculosis of the spine recover, usually after some deformity has arisen. When recovery occurs the granulation tissue surrounding the tubercles and the caseous necrotic masses become converted into hard bone; in this way the remains of the caseous vertebral bodies, the articulations, and sometimes also the laminæ and spines become fused together by firm bony anchylosis (see Fig. 140).

Secondary Changes in the Skeleton. The changes in the thorax are partly shown in Fig. 140, there is also a flattening of the chest from side to side. Thus the position of the lungs and heart is altered, and their action and the respiratory movements are hampered. In cases of dorsal caries the lumbar part of the spine becomes over-arched (*lordosis*) in order to throw back the weight of the head and chest so that the centre of gravity is brought over the feet in walking. In young children one effect of walking in the position of lordosis is to cause narrowing of the pelvis from side to side.

Rheumatism of the Spine. Several affections of the spine

which may simulate tubercular disease, save that they are even more painful, are grouped together as "rheumatic." The commonest is part of a general osteo-arthritis and leads to the condition known as *spondylitis deformans*. In this disease a considerable backward curvature of the spine may occur, and

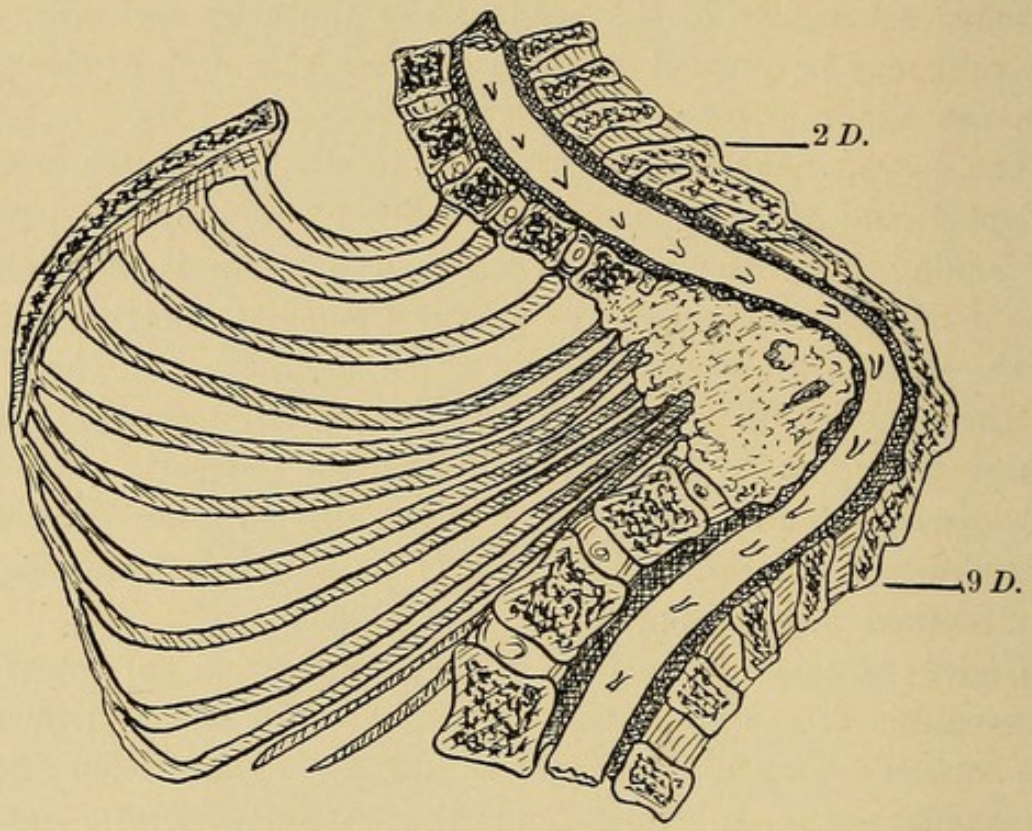


FIG. 140.—Healed tuberculosis of the spine. A schematised drawing of the right half of the thorax of a woman, aged 28, who had suffered from spinal caries in childhood, and who died of bronchitis. The fourth, fifth, sixth, seventh, eighth, and ninth dorsal vertebral bodies have been destroyed, and are represented by hard new bone, which contained several foci of caseous matter. The spines of the vertebræ, from the second to the ninth inclusive, are joined together by bone. The spinal canal is slightly narrowed, but there is no pressure on the cord. The new bone, depicted as having been formed from the vertebral bodies, is rather more than was actually present. Note the curved shape of the sternum, which is placed abnormally high, its upper end being opposite the seventh cervical instead of being opposite a point between the second and third dorsal vertebræ. There is great increase in depth in the thorax. The posterior ends of the ribs in the neighbourhood of the disease are crowded together. In the original there was a sharp lateral as well as an antero-posterior bend. (257 A.)

may become permanent by the formation of bridges of bone which join together the bodies of neighbouring vertebræ.

Sarcoma and Cancer of the Vertebræ may at first be difficult to distinguish from tubercular disease, especially when the latter occurs in elderly subjects.

Torticollis or Wry-Neck. Wry-neck arises from a great

variety of causes. The chief direct cause is contraction of one of the sterno-mastoid muscles; sometimes the trapezius and the deep muscles of the neck are also affected.

The affection sometimes appears to be congenital. Many such cases are due to contraction of a sterno-mastoid from cicatrization following rupture and hæmorrhage during parturition. Another simple and common cause is contraction of a sterno-mastoid which has for its object the protection of *inflamed cervical glands*. *Cervical caries* is sometimes accompanied by wry-neck. *Paralytic wry-neck* is rare and is due to partial or complete paralysis of one sterno-mastoid and contraction of its opponent. *Rheumatic wry-neck* is caused by rheumatic affections of the deeper cervical muscles. *Hysteria* is sometimes productive of wry-neck.

Spasmodic Wry-Neck is an affection which appears in adult life, and is caused by contraction of one sterno-mastoid and associated muscles. It appears to be akin to chorea, and is probably caused by some central irritation which affects the muscles through the spinal accessory nerve. The affection is extremely difficult to treat. Division of muscles and excision of part of the spinal-accessory, and the posterior branches of the first four cervical nerves has been practised with success.[1]

Lateral Curvature of the Spine, or Scoliosis. In a many-jointed bony column a slight deviation from the normal contour in each joint will produce great deformity in the whole column. When the manner in which the segments of the column are held together is complex, as is the case with the vertebræ, the deviation from the normal cannot fail to be complex if every part of the column is affected by a slight passive yielding to weight, as is the case in the commonest form of scoliosis. This deformity consists in the presence, not only of latero-flexion, but also of rotation in the various regions of the spine.

The degree of lateral flexion possible in the different regions of the healthy spine varies. In the cervical region of the spine, lateral flexion is always combined with a certain amount of rotation on account of obliquity of the articular processes. In the dorsal region a certain amount both of latero-flexion and

[1] See Noble Smith, *Spasmodic Wry-Neck*, 1891.

of rotation are allowed. In the lumbar region from the lower articular processes of each vertebra being received within the upper articular processes of the vertebra below it, whilst the interarticular discs hold the bodies of the vertebræ together in front, rotation would at first sight appear to be impossible; by a combination of latero-flexion with some antero-posterior flexion a certain amount of rotation is, however, allowed. The natural flexuosity of the spine is called into action during both exercise and rest. When the bones and muscles are healthy and exercise and rest are rightly balanced, there is no tendency for any one posture of the spine to become first accentuated, and subsequently fixed, *i.e.* converted into a permanent deformity. When lateral curvature has once become permanent, unless it is checked by treatment, structural changes, which render the affection incurable, occur in ligaments and bones. As an illustration of how scoliosis is most commonly produced an average example may be given. A rapidly growing and developing school-girl of 14 years is, from want of air, nourishment, and rest, anæmic. The muscles are weak from having insufficient oxygen and nourishment from the blood. The weak muscles are unable to hold the body upright for long periods at a time. In standing in class the "stand-at-ease" position is assumed, *i.e.* the weight of the body is thrown on the right leg, the hip and knee-joints being extended; the strain, as far as the lower limb is concerned, is on the strong ligaments in front of the former and behind the latter. The left hip and knee are bent, and the left foot rests so slightly on the ground that the weight of the whole

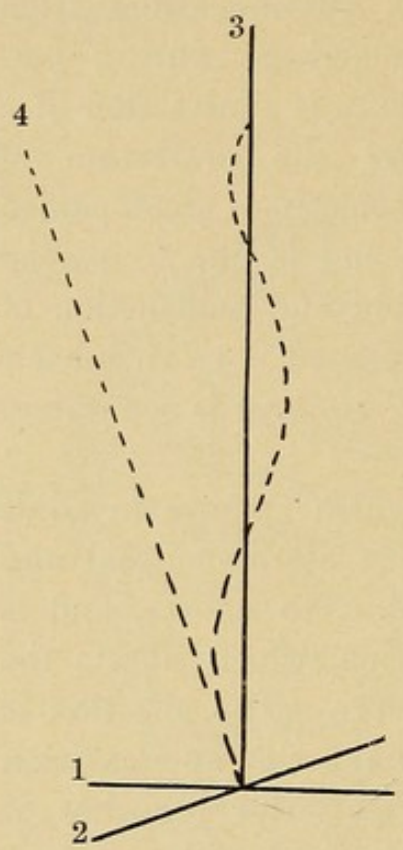


FIG. 141.—A diagram showing the production of lateral curvature from inclination of the pelvis. 1, The horizontal position of the pelvis; 3, the vertical position of the spine; 2, the oblique position of the pelvis; 4, the position the spine would assume when the pelvis is tilted to the left if a curvature of the spine to the right were not made to bring the centre of gravity over the supporting feet. The curved interrupted line shows the position of the spine assumed in lateral curvature of the usual type, *i.e.* with the lumbar curve convex to the left.

body is practically supported by the right leg, and the position of the left leg necessitates the pelvis being tilted downwards on that side (see Fig. 141).

The pelvis being inclined downwards on the left side the spine must be inclined to the right in order to bring the weight of the body over the supporting leg. Thus the lumbar spine curves over with a convexity to the left. If the back of a young person in the stand-at-ease position be examined, there will be found "a large sweeping curve, sometimes almost a semicircle, extending from the lower part of the dorsal region to the sacrum."[1] If this position is frequently adopted for long periods the lumbar

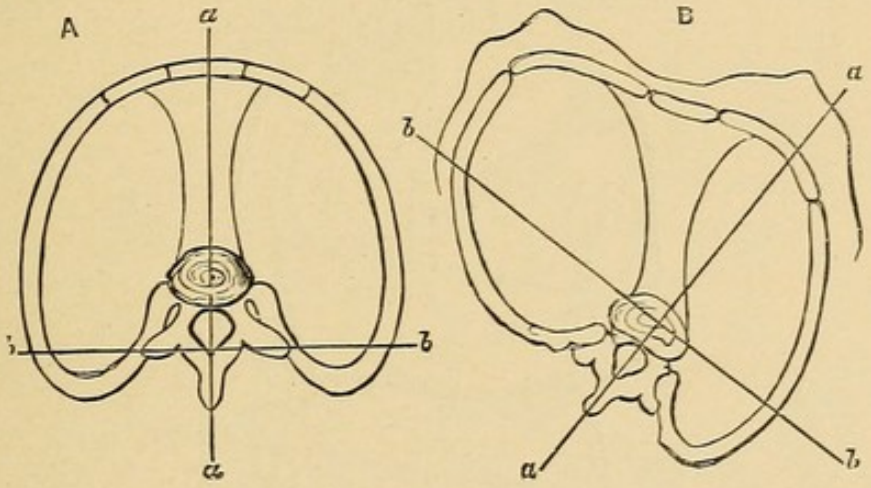


FIG. 142.—Diagrammatic section of normal chest and that with rotatory curvature. In the latter it is seen that the body of the vertebra is rotated into the right side of the chest, the rib on this side turning at an unduly sharp angle. The prominence of the transverse process on this side and of the breast on the opposite side is shown. (ADAMS.) Coats's *Manual of Pathology*.

curve becomes increased, so that the centre of gravity is thrown too far to the right. This necessitates a second curve in the dorsal region with the convexity to the right, as shown in Fig. 141. A third curve in the cervical region is also in time developed, and finally even the sacrum becomes deformed. Meanwhile, if proper treatment is not employed, the spinal curves become permanent from changes in the ligaments. Later still, the bones become altered in form (see Fig. 144).

Lateral curvature is always accompanied by marked rotation

[1] The quotation is from the article by W. J. Little in Holme's *System of Surgery*, vol. ii., p. 431.

of the vertebræ. The fronts of the bodies of the vertebræ rotate towards the convexity of the curve. Thus, the disease might well be termed latero-rotatory distortion of the spine. From this rotatory displacement it follows that the displacement of the spines does not fully indicate the amount of the deformity.

There are many other ways in which scoliosis may arise besides a lounging habit of standing; the same may result from long

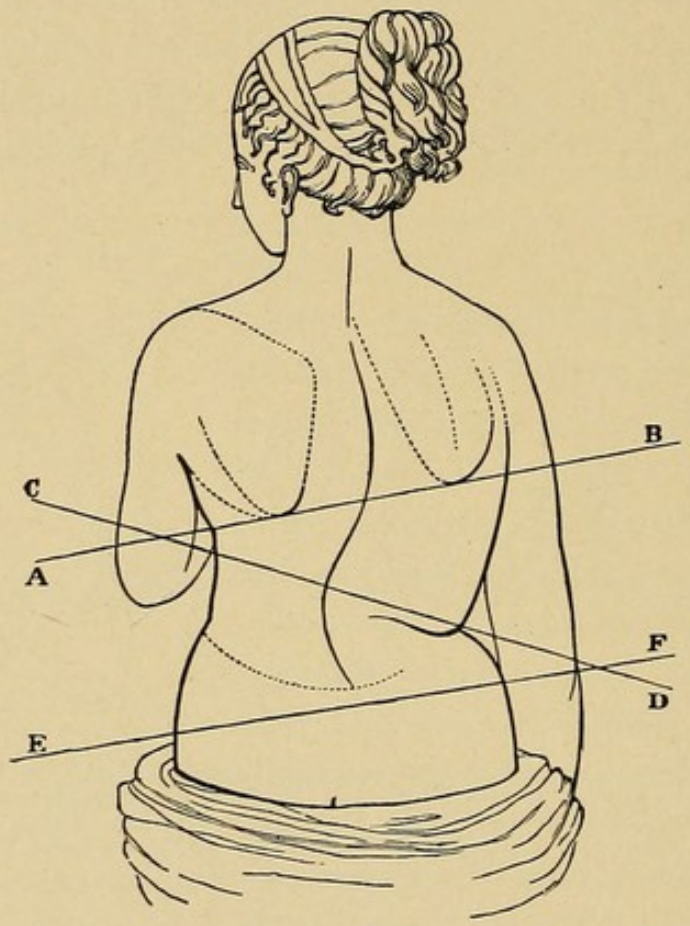


FIG. 143.—A diagram showing the altered position of the scapulæ (line A B), of the hollows of the waist (line C D), and of the iliac crests (line E F). Warrington Haward, *Orthopædic Surgery*.

sitting with the body twisted on account of bad sight, or bad light, or muscular weakness. Again, when from any cause one leg is shorter than the other, there is obliquity of the pelvis, and scoliosis is produced. In rickets, when the bones are softened and the muscles are weak, scoliosis sometimes occurs. Persons who use one arm much more than the other are apt to have a dorsal curve develop towards the more used side. After empyæma if the lung on the affected side does not expand sufficiently the chest becomes contracted, and a concavity of the

dorsal spine develops on that side. Again, some persons are born with a lateral curvature, owing to deformity of one or more vertebræ (*congenital lateral curvature*). A lateral frequently complicates an angular curve in tuberculosis of the spine. When scoliosis has become established, secondary changes occur in other parts of the skeleton. Those in the thorax and pelvis are the most important. In this place the former will be briefly discussed. The rotation of the dorsal vertebræ carries the necks

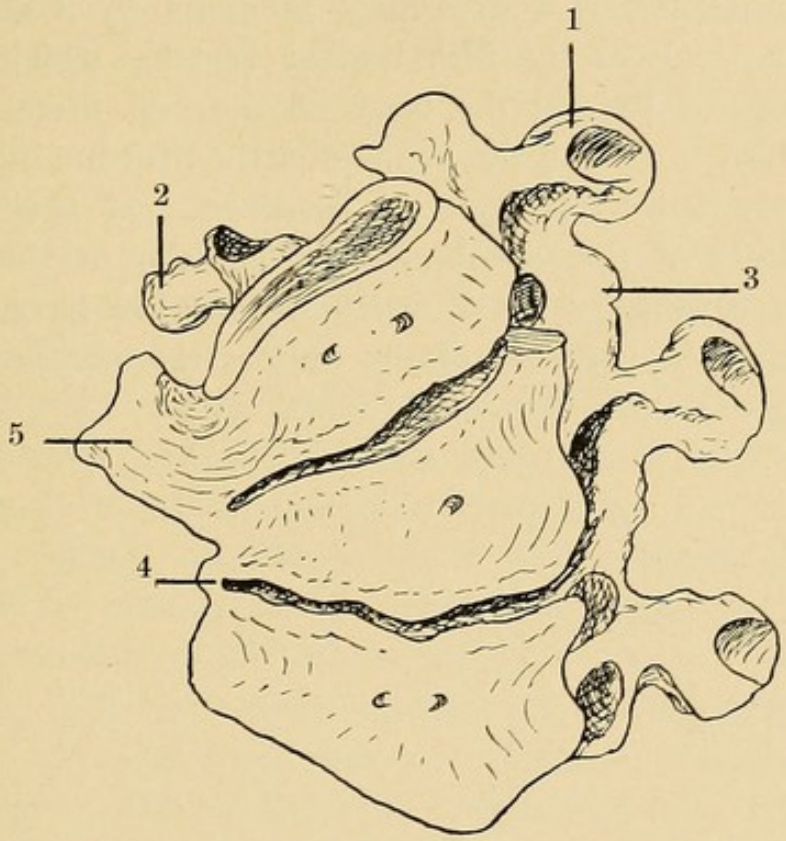


FIG. 144.—Three dorsal vertebræ from an old person who had scoliosis. The transverse articular processes are larger and the vertebral bodies are much deeper on the convex than on the concave side. 1 and 2, transverse processes; 3, articular processes ankylosed together; 4, a bridge of bone joining vertebral bodies; and 5, head of a rib ankylosed to two vertebræ. In this case osteo-arthritis probably supervened on scoliosis.

of ribs backwards on the convex side, forwards on the concave. Thus, on the convex side the ribs become sharply bent at their angles, on the concave side at the junction of the bone with the costal cartilage. In this way the scapula of the convex side of the curve and the mamma (see Fig. 142) on the concave side become more prominent.

Again, the ribs on the convex side tend to be separated, those on the concave side to be crowded, and sometimes fused together The heart and lungs are altered in position, and, in severe cases,

hampered in action. Fig. 143 shows diagrammatically the alteration in the appearance of the back.

The changes in the vertebræ that occur and render the disease incurable in its later stages are diminution of the depth of the vertebral bodies, and of the size of the half of the spinal arch and processes in the concavity of the bend, and, later, osseous ankylosis of the vertebræ (see Fig. 144), especially if osteo-arthritis should supervene.

Spondylolisthesis is a deformity produced by a sliding forwards of the body of the fifth lumbar vertebra which comes to project forwards beyond the body of the first sacral vertebra. The condition is due to the pedicles of the fifth lumbra vertebra being interrupted by a layer of cartilage near their junction with the body of the vertebra. The pedicles of the vertebra are elongated, but its lower articular processes have a normal relation to the upper ones of the first sacral vertebra.

CHAPTER XXIV.

CERTAIN DEFORMITIES OF THE EXTREMITIES: CLUB-FOOT, ETC.

THE conditions which determine deviation from the normal conformation and structure of the various parts of the body are extremely varied. Many of them have been dealt with in Chapter VII. Some, which from their frequency, are of special importance, and which from their dependence on pathological changes in nerve and muscle, or from the manner in which they affect locomotion, form well recognized clinical groups, will be noticed here. Such are talipes or club-foot, and congenital dislocation of the hip.

Club-foot. The two most important groups of cases are: (1) Congenital; (2) club-foot, due to infantile paralysis. Other conditions give rise to the same deformity, *viz.* spasm due to primary degeneration in the lateral columns or secondary to brain-injury, as in post-hemiplegic spasm; and among the latter is called the condition *spastic paralysis*, observed in children who are usually of weak intellect. The spasm of the muscles in some cases ceases during sleep, but when the patient is awake and likely to use the limb the spasm and deformity are present. The deformity that may arise from injury to nerves (see p. 207), and the hysterical or neuromimetic deformities of over self-conscious children, must not be forgotten.

Congenital Club-foot. In the opinion of the majority of observers[1] this condition is due to the limbs having been fixed in a certain position in the uterus instead of being free to

[1] See Parker and Shattock, *Congenital Club-Foot.*

enjoy the normal range of intrauterine exercise. The result of the examination of limbs deformed in this manner is certainly in support of this view, since no evidence of disease in nerve or muscle, save, in severe cases, pressure-atrophy, can be found. As an instance of how accidental mechanical conditions may produce club-foot the case from which Fig. 145 was taken may be cited.

FIG. 145.—A fœtus born with the cord wrapped round the feet which were both in a condition of equino-varus. There was marked flattening of the cord and grooving of the legs, showing that the cord and the feet had long been in the state shown here. There is some hydrocephalus.

The mechanical condition that prevents that member having the normal range of movement may be due to some peculiarity in the foot itself, as in the case shown in Fig. 146.

The occurrence of spina bifida, hydrocephalus, etc., with congenital club-foot, has been taken to prove that the latter is due to altered innervation; this view is not supported by the

results of examination of the limbs, and on the contrary it seems to the author more probable that the spina bifida and hydrocephalus, as well as the club-foot, may be due to increased intrauterine pressure at a period of intrauterine life previous to the closing of the medullary groove, and when the liquor amnii can be still forced into the canal of the spinal cord and so into the cerebral ventricles.

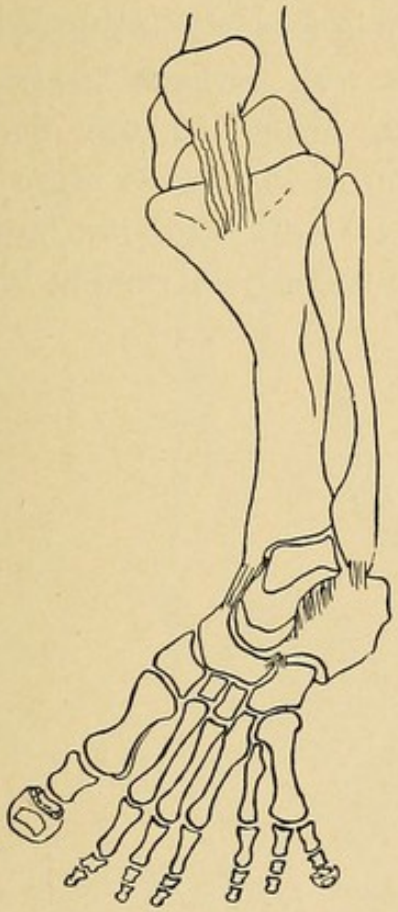


Fig. 146.

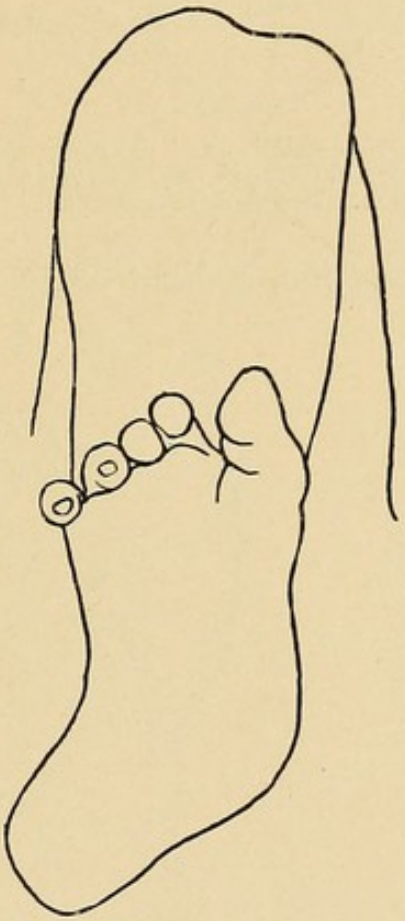


Fig. 147.

Fig. 146.—The bones of the left leg and foot of a still-born fœtus. There are six metacarpal bones and seven digits. The leg bones were somewhat thicker than normal, and the foot was in the position of the equino-varus form of club-foot.

Fig. 147.—Congenital talipes calcaneo-valgus.

Varieties.—*Talipes Calcaneus.* The deformity consists in the position of the foot being that of flexion (so-called) of the ankle-joint.

Talipes Valgus. The foot is in the position of eversion.

Talipes Equinus. The ankle-joint is extended without inversion of the foot. This is a very rare form in congenital talipes, whilst it is the commonest in paralytic talipes.

Talipes Varus. The foot is inverted.

Combinations of the first two and of the last two are more common than any of the varieties singly. They alone will be described in some detail.

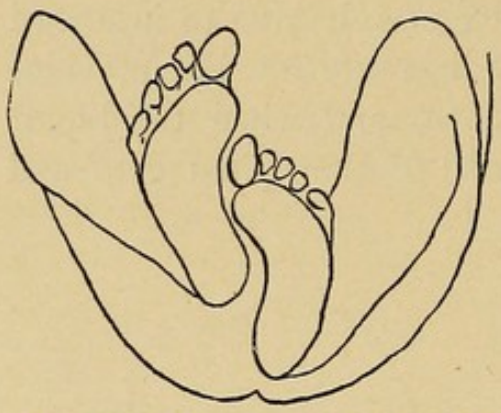


FIG. 148.—Attitude of the lower limbs of a normal fœtus in the later months of gestation. Modified from Braune.

Talipes Calcaneo-valgus (Fig. 147) is the commonest variety of congenital club-foot, but it is usually in such slight degree that it gets well without treatment. Indeed, owing to the normal position of the feet in utero (see Fig. 148) during the last months of gestation, a slight degree of calcaneo-valgus is normal at birth;

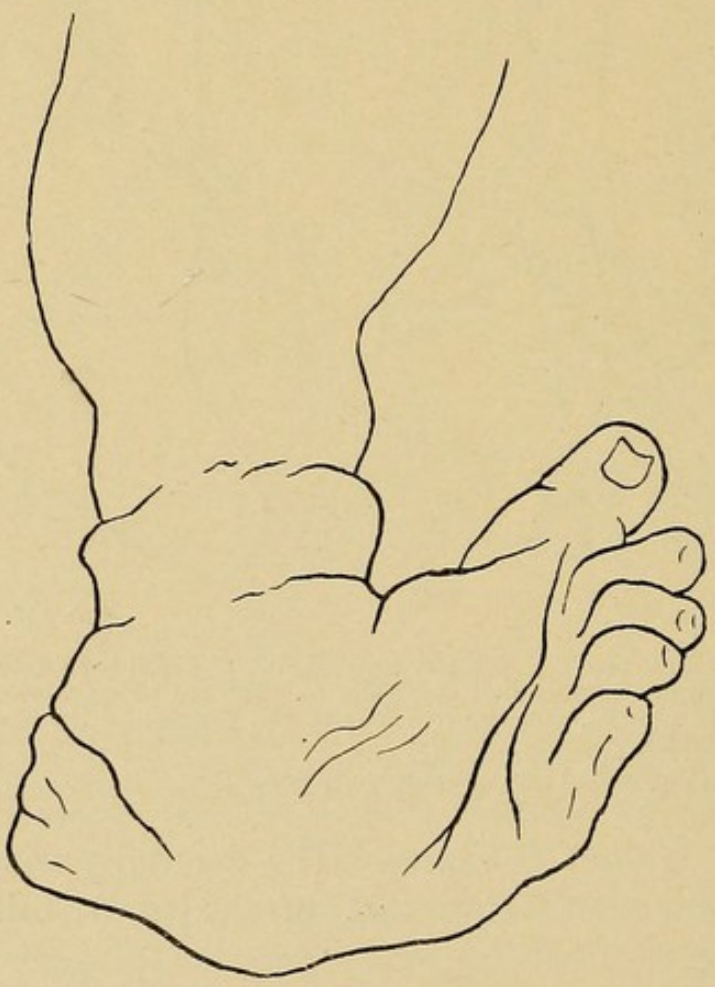


FIG. 149.—Front view of a foot affected by talipes equino-varus. The grooves on the leg were caused by pressure of the cord (see Fig. 145).

only more severe degrees, like that shown in Fig. 147, require surgical measures.

Congenital Equino-varus, though not so common as calcaneus, comes more frequently to the surgeon's notice, because it is not cured by the movements of the child in walking, etc. The condition is the converse of that just described. In a fairly severe case, such as that shown in Fig. 149, which is taken from the left limb of the fœtus, represented in Fig. 145, the great toe points more nearly upwards than forwards, and this might seem to be due to flexion of the ankle-joint, *i.e.* to calcaneus. On removing the soft parts, however, the ankle is seen to be really

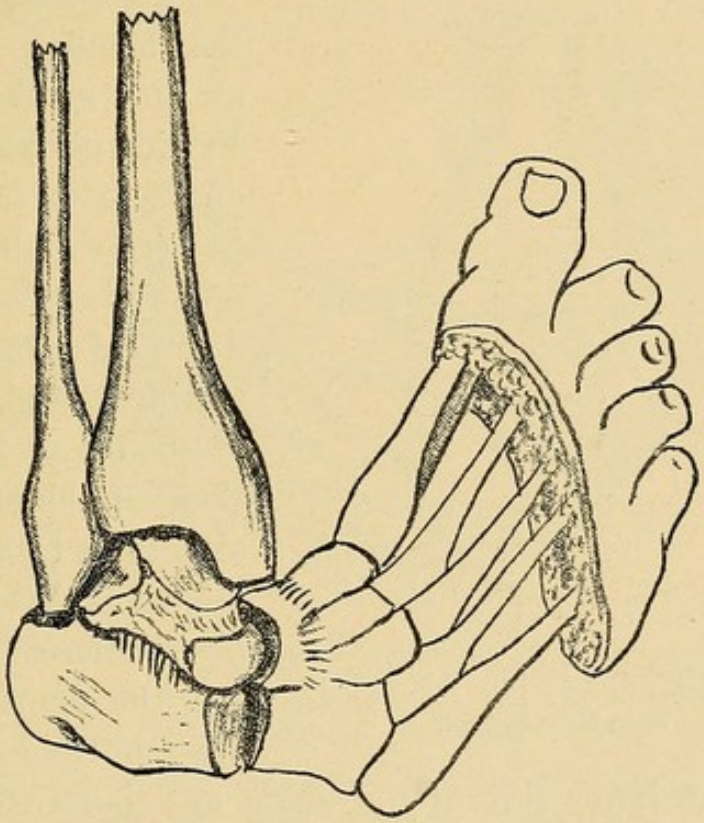


Fig. 150.—The same foot dissected to show that the ankle-joint is extended.

extended, as is shown in Fig. 150, which is taken from a dissection of the same foot as that shown in Fig. 149.

The alteration in the direction of the toes is due to inversion of the foot and a displacement of the anterior half of the foot inwards, and hence (in the inverted position), upwards at the medio-tarsal joint, where the outer half of the articular surface of the head of the astragalus is exposed by the inward displacement of the fore part of the foot. The metatarsus is also bent inwards on the tarsus. These alterations in the joints of the foot result, in extreme cases, in the great toe being

in contact with the inner border of the leg. Other features may be noticed. Thus in Fig. 150 two new articulations are present: the os calcis articulates with the external, and the scaphoid with the internal malleolus.

When the foot is viewed from the sole, which should be on the under but in varus is the postero-internal aspect, a deep crease is observed passing from the inner (now upper) border of the foot just behind its middle, outwards nearly to the opposite border (Fig. 151).

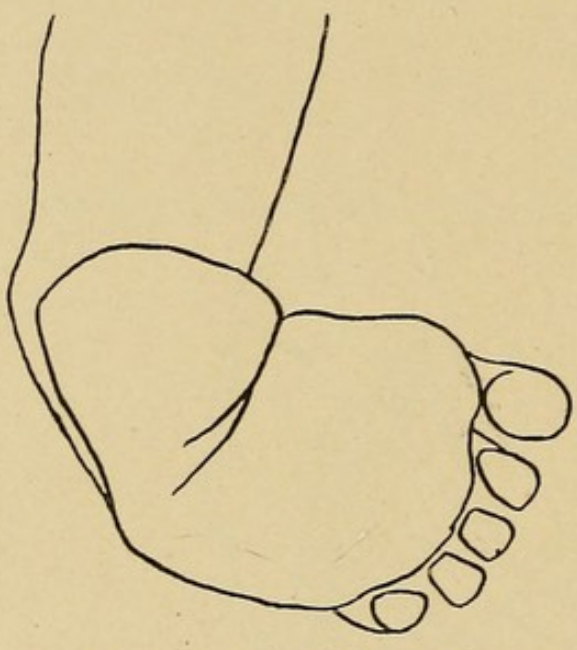


FIG. 151. — Congenital talipes equino-varus. The plantar surface of the foot showing the transverse crease. Seen from behind.

This crease is caused by the bending of the foot at the mediotarsal joint. Flexion at this joint entails an increase in the antero-posterior arch of the foot, or, in other words, a certain degree of *pes cavus*. On dissecting the plantar tissues of such a foot it is found that all the structures are altered in form to suit the altered skeleton. Thus the plantar fascia and muscles and the plantar ligaments are shortened, the ligaments on the inner side of the ankle are shortened, and those on the outer side are lengthened. From the hinder aspect the bones in a moderately severe case have the appearance shown in Fig. 152.

Among other changes may be mentioned the inward deviation of the neck of the astragalus and of the anterior part of the os calcis observed in severe cases.

The skin is shortened over the inner and stretched over the outer side of the foot. Over the prominences at the outer part of the ankle bursæ are often present at birth, and when the foot has been allowed to be used for walking large bursæ with thickened walls and large corns form on the feet.

The above description applies to severe and moderately severe

cases. In slighter cases similar but less pronounced anatomical deviations are found. These deformities are produced in intrauterine life when the tissues, including the cartilages, are readily moulded. Patient correction of the cavus and varus

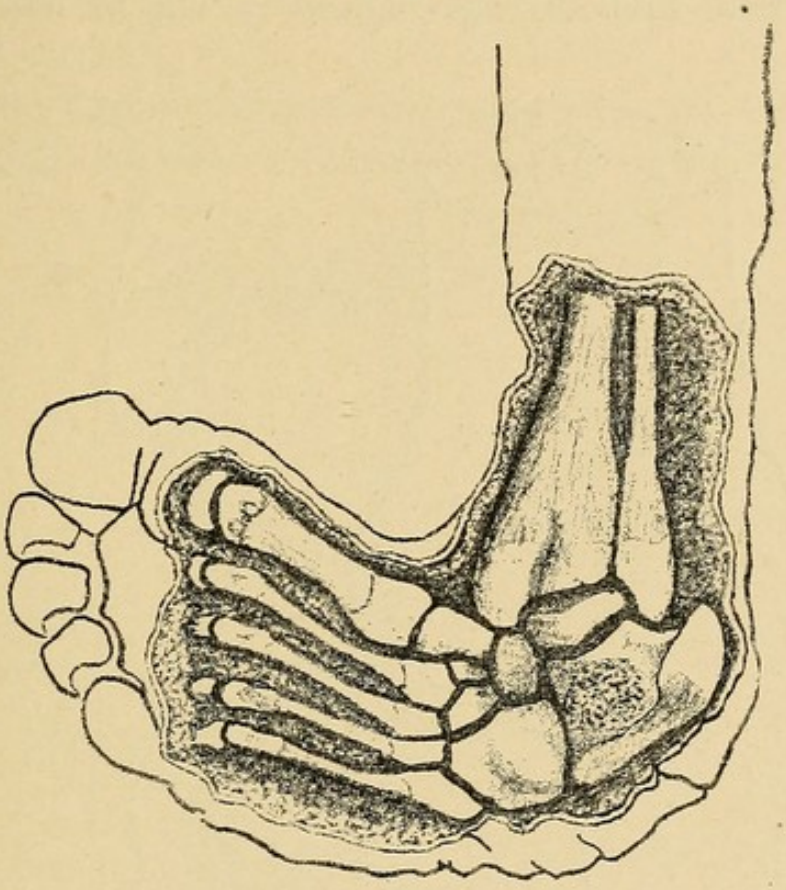


FIG. 152.—The bones of the foot in congenital equino-varus.

by careful splinting, continued for months it may be, is required before the limb is ready for any cutting operation.

Congenital Club-Hand is a deformity which corresponds to club-foot—over-flexion of the wrists is the more usual form of it.

Paralytic Talipes.

Talipes Equinus, which is the rarest form of congenital club-foot, is the commonest when the deformity results from infantile paralysis. It is due to the degeneration of part or of the whole of the fibres of the extensor muscles. If part of the extensor fibres escape then, as the tendo Achillis and other opposing muscles extend the ankle, the toes become over-extended at the metacarpo-phalangeal, as shown in Fig. 153, and may be over-flexed at the inter-phalangeal joints (claw-toes).

Sometimes nearly all the muscles, both flexor and extensor, are paralyzed, then from its weight and the pressure of the bed-clothes at night the foot assumes the position shown in Fig. 154. The limb is then cold, and chilblains and ulcers readily form. In both these figures it will be observed that

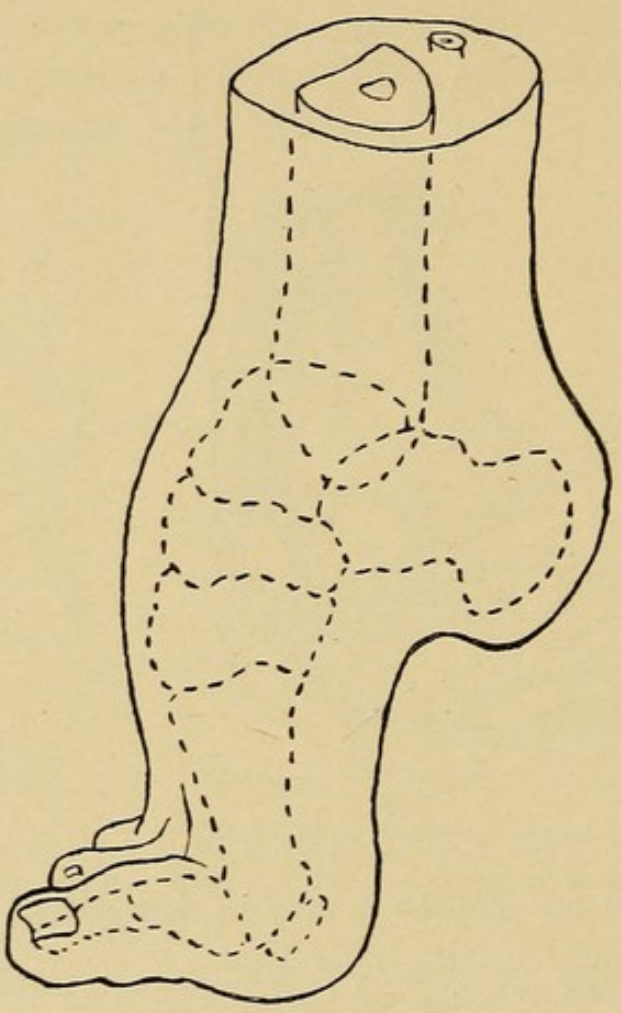


Fig. 153.

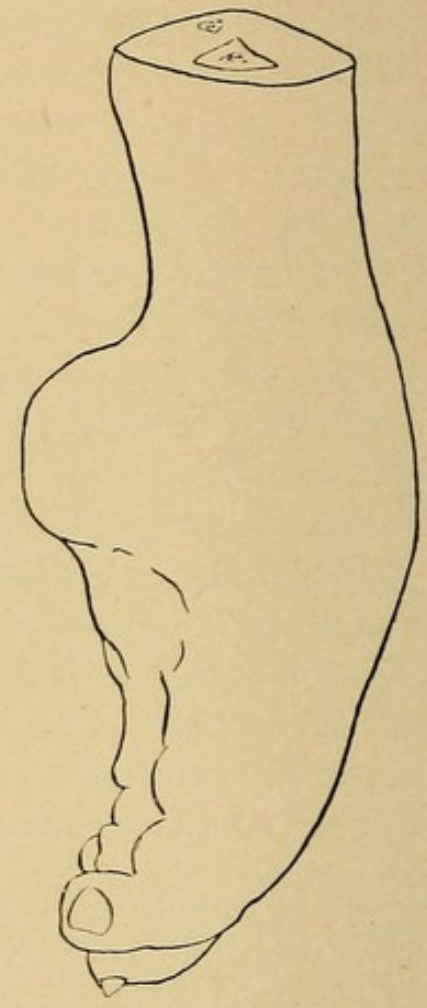


Fig. 154.

Fig. 153.—Paralytic talipes equinus from partial paralysis of extensors. After the late E. J. Chance.

Fig. 154.—Paralytic talipes equinus from total paralysis of extensors.

there is some *pes cavus* from flexion at the medio-tarsal joint. This is partly accounted for by the action of the flexor muscles, partly by the weight of the limb exerted at the ankle when the patient is walking.

Slight cases of equino-varus are called right-angled contraction of the ankle. When the knee is extended the ankle cannot be bent to less than a right angle. In such cases the *pes cavus*,

secondary to the equinus, may form the leading feature of the condition.

Paralytic Varus. When the everting muscles, the peronei, are paralyzed, the opposing muscles invert the foot, that is, produce talipes varus. The patient then walks on the outer border of the foot.

Paralytic Equino-Varus results when both the extensor and the everting muscles are paralyzed. The foot has a similar

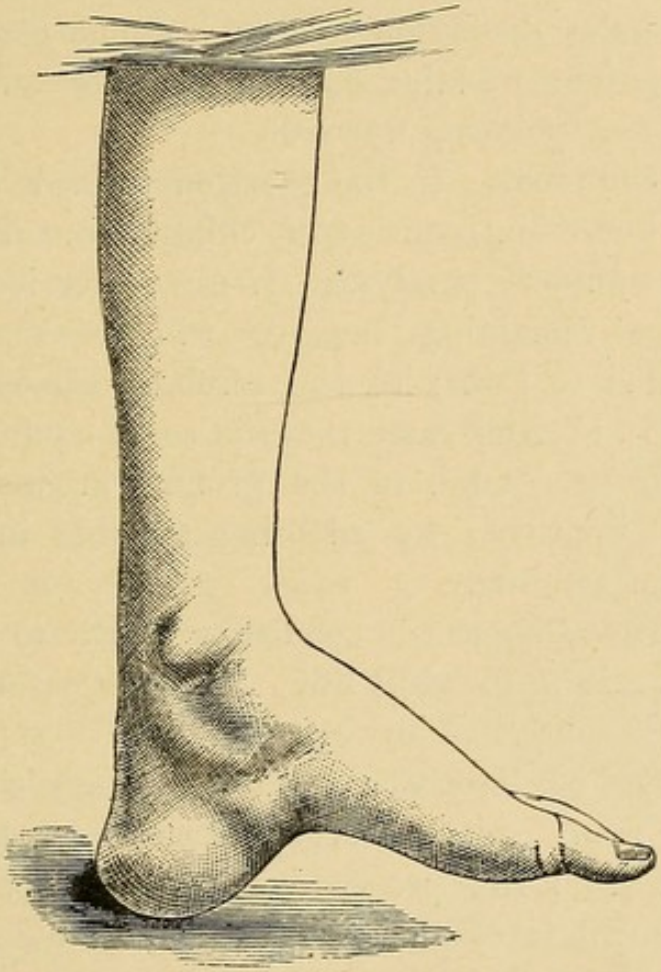


Fig. 155.—Paralytic talipes calcaneus. Warrington Haward, *Orthopædic Surgery.*

appearance to that figured in congenital equino-varus with, in some cases, the addition of a longitudinal furrow in the sole produced by walking on the outer border of the foot, and so increasing the transverse arch.

Paralytic Talipes Calcaneus differs from the congenital form of the disease in that it tends to grow worse instead of improving. The os calcis and astragalus may become nearly vertical in position, and at the same time the anterior part of the foot

drops by flexion of the transverse tarsal joint (see Fig. 155). It is due to paralysis and wasting of the calf muscles.

Other varieties of paralytic talipes result from the simultaneous occurrence of paralysis in various groups of muscles.

After a paralytic deformity has been cured instruments must as a rule be worn *in perpetuo* to prevent its recurrence.

Deformities of the Knee-joint akin to congenital and paralytic talipes are not uncommon. The commonest is genu recurvatum where the knee is bent, so that there is an angle salient backwards; rectangular distorsion is seen in some congenital cases, so that the patient waddles on the posterior surfaces of the leg with the feet pointing upwards.

Spasmodic Club-foot. In the affection termed "spastic paraplegia" the lower extremities of children are in a state of more or less complete paralysis. If the latter is not complete *rigidity* of the remaining muscles will be observed. The condition is due to injury to the cerebral convolutions during parturition. In slighter cases there is some equinus or equinovarus, and the toes catch in the ground in walking. These cases can be improved by adapting suitable instruments, if necessary, after tenotomy.

Post-hemiplegic Chorea is a condition similar to the foregoing in origin but arising in adult life. The spasms are usually too powerful to be controlled by instruments.

Other Forms of Paralysis such as progressive muscular atrophy and many other nervous diseases, may produce deformities, but from the progressive character of the diseases surgical treatment is of no use.

Pes Cavus, also called talipes plantaris or arcuatus, may arise in different ways. Pes cavus, secondary to talipes equinus, has already been referred to. Other cases are due to "rheumatic" contraction of the plantar fascia, others again to paralysis of the interosseous muscles. In addition to the over-arching of the foot, the toes are sometimes greatly over-extended at the metatarsophalangeal joints and over-flexed at the interphalangeal joints, a condition which resembles "hammer-toe," but is due to contraction of the unopposed lumbrical muscles, and not, like hammer-toe, to mechanical displacement.

Pes Valgus or Flat-foot is due to sinking of the plantar arch owing to stretching of ligaments, especially the inferior calcaneo-scaphoid. It is often accompanied by outward deviation of the ankle (*valgus ankle*), and this must be remembered in treatment. It occurs in rickets, as shown in Fig. 66, and in adolescents from weakness of muscles, especially the long flexor of the great toe, combined in some cases with softening of ligaments and bones; it also occurs in osteo-arthritis.

Hallux Rigidus is a condition in which the great toe is slightly flexed at the metatarso-phalangeal joint and cannot be extended. It is usually accompanied by some degree of flat-foot.

Hallux Valgus is due to outward displacement of the great toe from wearing boots with toes pointed towards the central line of the foot. There is a subluxation of the first phalanx and of the sesamoid bones outwards. Thus the head of the metatarsal bone becomes over-prominent and a bursa (or *bunion*) develops over its convexity. The second toe may be displaced upwards, as shown in Fig. 156, and doubled back (*hammer-toe*) behind the great toe.

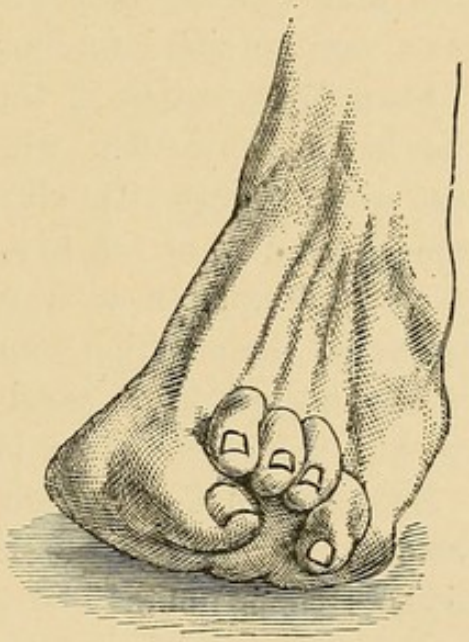


FIG. 156.—Hallux valgus and flexion of the four outer toes at the inter-phalangeal joints (hammer toe). Warrington Haward, *Orthopædic Surgery*.

Genu Valgum, or knock-knee, is a condition in which, when the knees are placed together, the internal malleoli are separated by a greater or less distance. It is in part referable to an outward concavity of the lowest part of the shaft of the femur, or, what is really the same thing, an elongation of the inner part of the shaft of the bone.[1] An outward bend at the upper end of the tibia contributes to the deformity. In the earlier stages proper instruments will cure the deformity (see Fig. 66).

[1] See a paper by Symington, *Transactions of the Royal Academy of Medicine, Ireland*, 1896, p. 408.

Genu Varum, or bow-leg, is the converse of genu valgum. When the internal tuberosities are together the knees are more or less widely separated. An outward convexity of the tibia is usually the chief factor, sometimes the femur is also bent.

Genu Recurvatum. This condition is marked by an over-extension of the knee. There are two classes of cases: (1) congenital, due to malposition of the fœtus in utero, and may be compared with congenital club-foot, and (2) acquired from paralysis of the hamstring muscles.

Coxa Vara. The deformity of the neck of the femur which leads to this condition has been shown in Fig. 67. Waddling gait and upward displacement of the great trochanters cause coxa vara to simulate congenital dislocation of the hip.

Some Deformities of the Fingers and Hand. Conditions of the hand comparable with the various kinds of club-foot are met with. Congenital club-hand is much less common than the same deformity of the foot. *Syndactyly* or fusion of the fingers is not uncommon as a congenital condition. The slightest degree of the affection constitutes webbed fingers. Contractions of the fingers from paralysis, spasm, or contraction of fascia are not uncommon; the last-named deserves special mention.

Dupuytren's Contraction affects most commonly the ring and little fingers, but all four inner fingers may be drawn down towards the palm. The cause of the deformity is thickening and contraction of the process of palmar fascia, which passes to the finger that is at fault. The thickened process stands out like a rigid band under the skin, which is drawn down and fixed to the band at various points. In most cases that have come to my notice, the subjects of the affection have suffered from lithæmia or gout. On dissection crystals of urate of soda are sometimes found in the thickened fascia. A slight injury occasionally seems to start the process in gouty persons. The contractions of scars in the palm of the hand may also produce similar deformities, but are distinguishable by their origin from Dupuytren's contraction.

Congenital Dislocation of the Hip. This deformity, which may be uni- or bi-lateral, is marked by an instability of the

hip-joint, so that when bearing the weight on one or other limb the patient inclines to that side in order that the os innominatum may rest on the femur; this produces a characteristic rolling gait. The limbs are shortened by the upward displacement of the femur. In standing the lumbar spine is arched forwards (*lordosis*) and the buttocks are very prominent. The results of the examination of still-born fœtuses in the hands of Lockwood and myself were similar and seem to me to point to malposition, etc., in utero being the cause of this deformity as is the case with club-foot. The condition in the author's case was as given below.

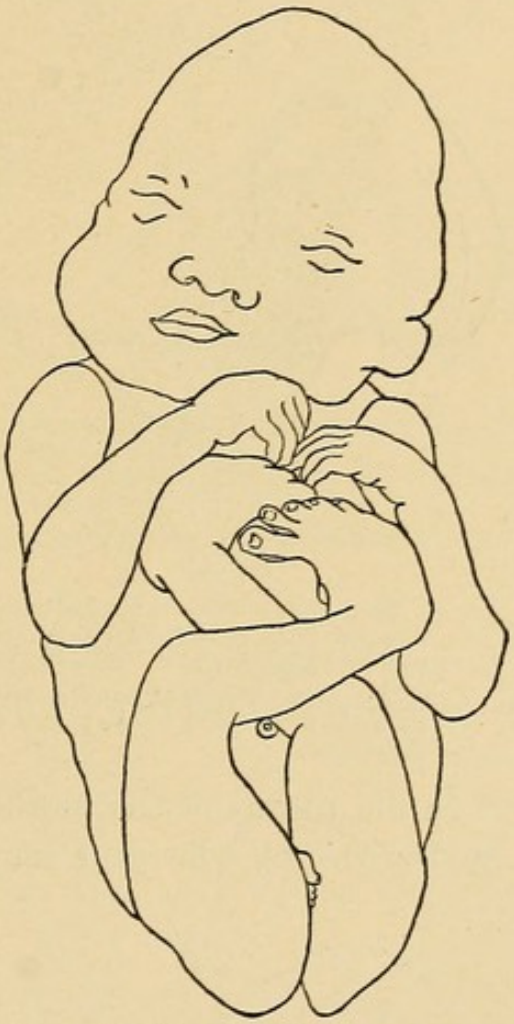


FIG. 157.—The attitude of the fœtus in utero. There is a projection of the chest-wall close to the inner border of the right foot.

The position of the fœtus before birth is shown in Fig. 157. This could be ascertained from the fact that the pressure of the feet had determined deformities in the chest-wall.

On dissection both hip joints before the capsules were opened had the appearance shown in Fig. 158. The femora were rotated out, and rested on the bone above the acetabulum. Inward rotation through 90° caused the head of the bone to slip into the socket, but the slightest upward pressure reproduced the dislocation, so proving that there was an abnormally shallow acetabulum. After the dislocation had been reduced, and the hip-joint extended, the front and back parts of the capsule were tightened (see Fig. 159).

On opening the joint, the head of the bone was seen to have the position shown in Fig. 160. The acetabulum was narrowed as shown in Fig. 161. The cartilage and cotyloid ligament were both present, but the former was diminished in amount,

and the latter flattened as if by pressure. The head of the femur was not much changed, but the pelvis was flattened from side to side (Figs. 163 and 164).

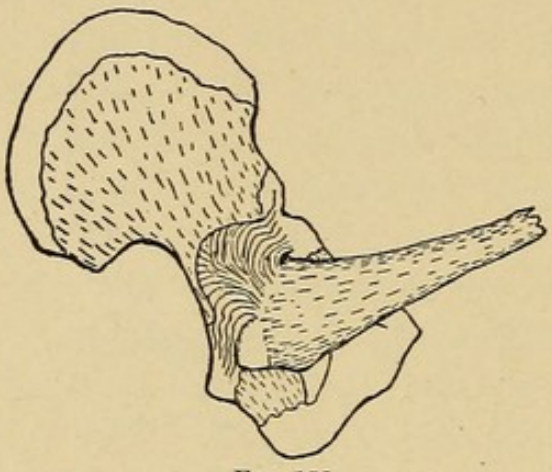


Fig. 158.

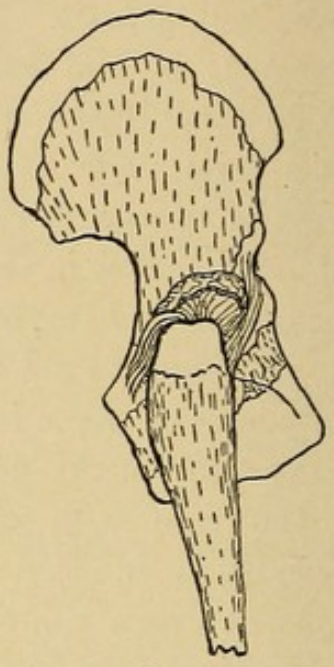


Fig. 159.

Fig. 158.—Shows the capsule of the hip-joint stretched over the dislocated femoral head which was separated from the ilium by the capsule and the cotyloid ligament.

Fig. 159.—Shows the parts with the dislocation reduced. The capsule is thrown into folds above; the ilio-femoral and ischio-capsular fibres are tightened.

In the uterus of the mother several fibroid tumours were felt by Dr. Déqué, who sent me the fœtus. These tumours would diminish the elasticity of the uterine walls, and the space in which the fœtus lay.

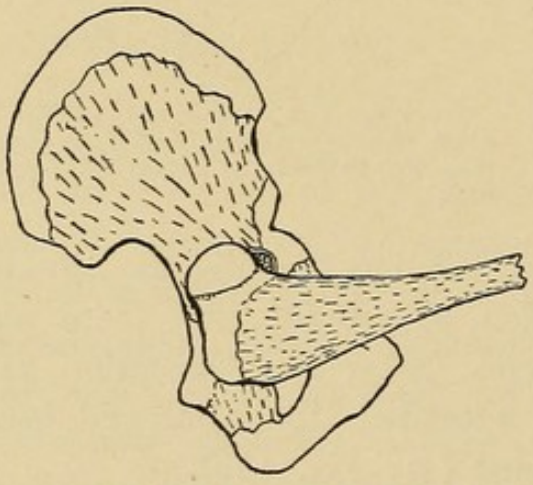


Fig. 160. – Shows the position of the femur in relation to the os innominatum.

The later changes in congenital hip dislocation may readily be inferred. In walking, the head of the femur is pressed upwards, the capsule of the joint is still further elongated till it assumes an hour-glass shape. The acetabulum, deprived of the head of the femur, tends to fill up entirely, whilst the head of the femur becomes atrophied from pressure on the ileum, where from the same cause a new

but imperfect socket is formed. The pelvis in time becomes flattened from before backwards.

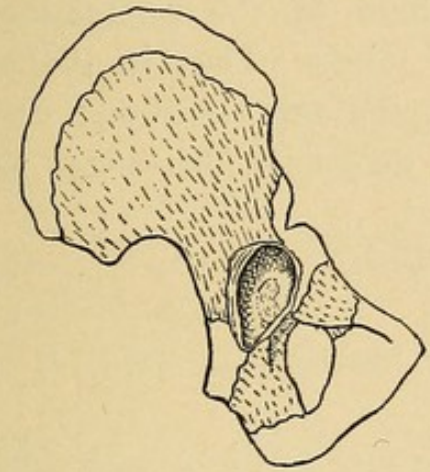


FIG. 161.

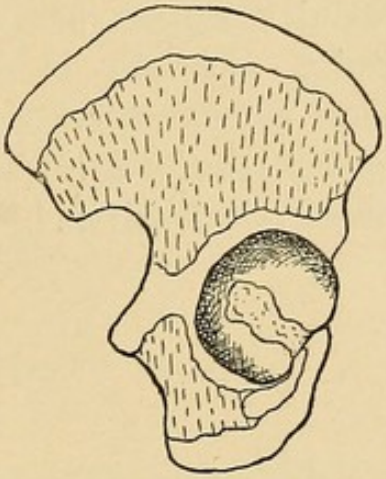


FIG. 162.

FIG. 161.—Side view of the right os innominatum of the fœtus shown in Fig. 157. The acetabulum is small, shallow, and misshapen. The cotyloid ligament is flattened out in the upper half of the acetabular rim. The os pubis is not directed as much inwards as in the normal bone.

FIG. 162.—The right os innominatum of a normal fœtus at full term for comparison.

Study of this case seems to warrant the hope that if in congenital dislocation of the hip suitable treatment is

FIG. 163.

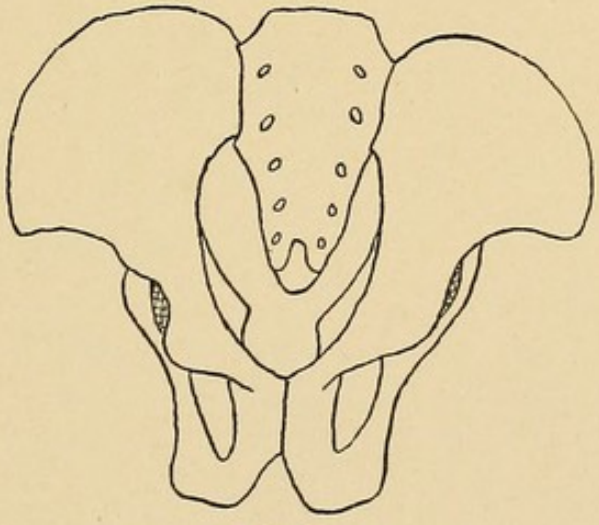


FIG. 164.

FIG. 163.—The head of the femur seen from behind. It is slightly flattened. The ligamentum teres is drawn out and flattened.

FIG. 164.—The pelvis considerably compressed from side to side

commenced immediately after birth, good results may be obtained.

Other Congenital Dislocations occur at the knee, wrist, shoulder, ankle, and elbow. They are sometimes associated with partial absence of bones, such as the radius or fibula.

Muscular Spasm has a large share in the production of deformity, especially in disease of joints. Thus in the early stages of angular curvature of the spine and tubercular disease of the hip, the greater part of the deformity disappears when an anæsthetic is given.

CHAPTER XXV.

DISEASES OF THE URINARY TRACT.

Affections of the Kidneys. Some anatomical peculiarities are apt to assume importance. Thus a person may be born with only one kidney, the other being either entirely absent or rudimentary; or may have only one functionally active, the other having been destroyed by disease. In either of these cases the

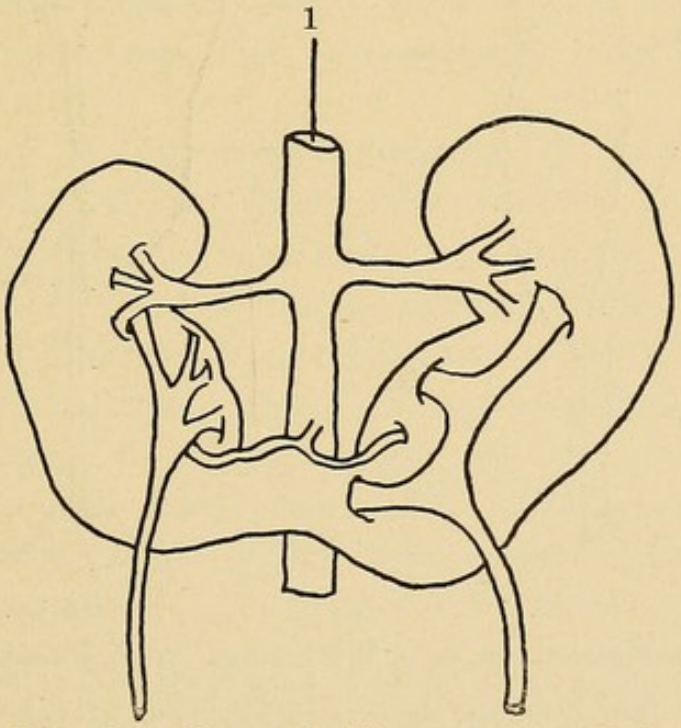


FIG. 165.—Horse-shoe kidney. The kidneys are joined by fusion of their lower ends. 1, Abdominal aorta. Front view.

kidney becomes hypertrophied to compensate for the absence of its fellow.

Fusion of the Kidneys. The commonest example of this condition is the horse-shoe kidney (Fig. 165, in which the lower

ends of the kidneys are joined together in front of the aorta and vena cava. Complete fusion is sometimes observed (Fig. 166); the fused kidneys then lie, as a rule, in the situation of the left kidney. Whenever the operation of removing a kidney is contemplated, the possibility of the patient having only one kidney, or of the kidneys being fused together into one, must be borne in mind.

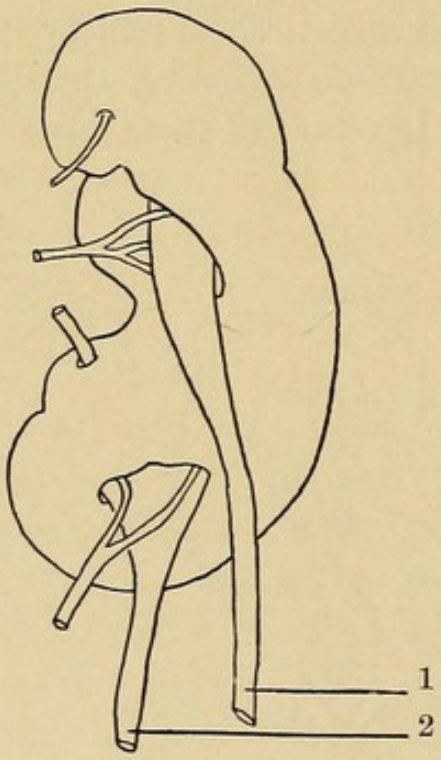


FIG. 166.

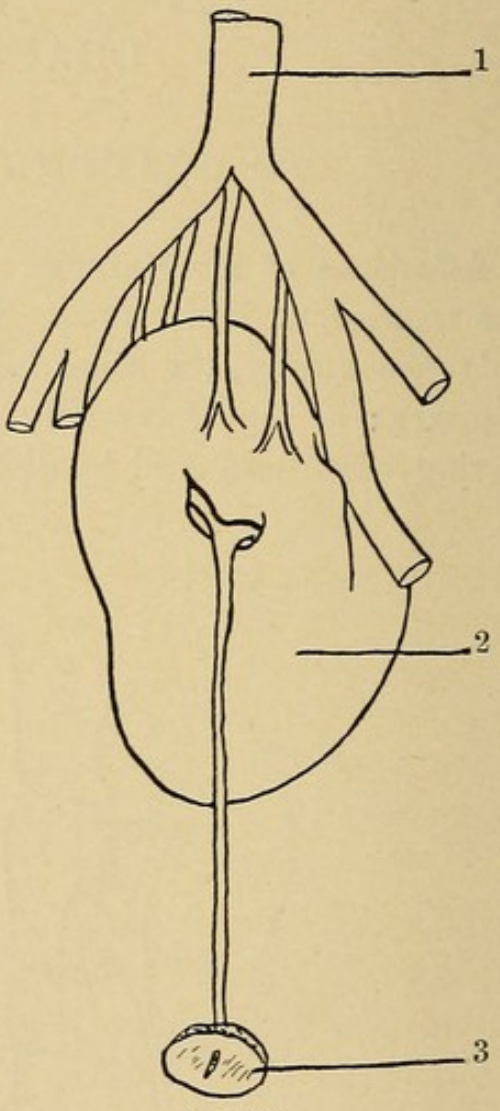


FIG. 167.

FIG. 166.—Complete fusion of the kidneys. The resulting "solitary" kidney was placed on the left side. 1 and 2, the ureters. Front view.

FIG. 167.—Solitary kidney, placed beneath the bifurcation of the abdominal aorta (1). 2, The kidney; 3, portion of the bladder wall. Front view.

Ectopia of a Kidney. As an example of congenital misplacement of a kidney the condition shown in Fig. 167 may be given. Such a kidney may be mistaken for a pelvic growth and removed; in which case, since the misplaced organ represents both kidneys, the patient would die.

Floating Kidney. This term is best reserved to describe a condition in which the kidney is suspended by a double fold of peritoneum or mesonephron. The position is probably congenital in the majority of cases. It allows the kidney a wide range of movement.

Movable Kidney. This term may be restricted to cases in which the kidney moves *behind* the peritoneum. It is usually seen in women who have borne several children, and is associated with a loose condition of the fat around the kidney. Both floating and movable kidneys may become hydronephrotic from kinking or twisting of the ureter.

Inflammatory affections of the kidneys, as far as they concern the surgeon, are chiefly secondary to conditions such as stricture of the urethra or enlarged prostate, which will be discussed below under the head of "surgical kidney." All diseases of the kidney are indirectly of importance to the surgeon, since those who suffer from Bright's disease are but little protected against septic processes, and recover badly from the shock of operations.

Tuberculosis is the most important of the inflammatory affections of the kidney. The ureter, bladder, prostate, testis, etc., frequently suffer with the kidney, so that the condition will be discussed with stone of the kidney and bladder at the end of this section. Though tubercular disease of the kidney is common, tertiary **syphilis** is rare. In one instance examined by the writer after death, the pelvis and caps of the pyramids presented ulcers and the renal vein was thrombosed. There had been much hæmaturia during life. There were typical gummata in the liver and elsewhere.

Cysts of the Kidney. Small cysts are very common in old age and in chronic interstitial nephritis. They usually cause no evidence of their presence, but occasionally some of them enlarge so that they become perceptible externally as a tumour. In one condition named *multilocular cystic disease of the kidney*, or *conglomerate cystic kidney*, the kidneys may attain a very large size, and the affection is marked by all the phenomena of chronic interstitial nephritis: high arterial tension, hyper-

trophy of the heart, etc. The condition may allow the patient to live to the age of twenty-five or thirty, then death usually ensues from cerebral hæmorrhage or uræmia. The surgeon's interest in the condition is purely negative. It is probably of congenital origin, the cysts increasing in size as time goes on. Similar cystic kidneys are occasionally present at birth; they may cause death of the fœtus and difficulties at birth.

Hydatid Cysts are sometimes found in the kidneys, and they may attain such a size that they fill the abdominal cavity. They are then difficult to distinguish from ovarian cystomata.

New Growths. Adenomata and fibromata of the kidneys are sometimes found after death, but they seldom give rise to symptoms. The growths more commonly observed are malignant.

Sarcoma of the Kidney is not infrequently a congenital condition, a tumour being observed soon after birth. The growth is then usually an adeno-sarcoma arising at the hilus of the kidney, and on histological examination presenting a series of tubules lined by epithelium and surrounded by sarcoma cells, among which may be new-formed striped muscular fibres. These growths are highly malignant and demand removal with the kidney as soon as possible after they are discovered. The position of such growths, at the hilus of the kidney, suggests an origin in some residual embryonic structure, such as part of the Wolffian body.

Sarcoma of the kidney is also encountered in adults. As soon as the growth ulcerates into the pelvis blood appears in the urine. In melanotic sarcoma the urine has been observed to be blackened. They grow rapidly and produce metastatic growths.

Cancer of the Kidney is rather less uncommon as a primary affection than sarcoma. The symptoms and rapidly fatal course of the disease is the same as in sarcoma. Removal can be effected only in the early period of the disease.

Diseases of the Urinary Bladder.

Cystitis, or inflammation of the bladder, occurs as a chronic and as an acute affection, but one form may pass into the

other. **Chronic cystitis** is usually secondary to stricture of the urethra, enlarged prostate, stone, or growths of the bladder. The urine contains an excess of stringy mucus, has an alkaline reaction, and a strongly ammoniacal character. It contains micrococci and bacteria; among the former are some which cause the ammoniacal fermentation of the urea. The latter are either introduced on a catheter or sound, either from want of proper sterilization or the use of a septic lubricant; or the instrument may become contaminated at its entrance into the meatus. Pain and frequent micturition are the leading symptoms. On examination of the bladder after death in such cases the mucous membrane is swollen, with enlarged veins, patches of congestion, and slaty pigmentation. There may be ulcers and phosphatic deposits. An attack of retention may at any time cause a chronic cystitis to become acute, pus being formed in large quantities, and usually the inflammation spreads along the ureters to the kidneys, causing death from suppurative pyelonephritis.

Acute Cystitis. Besides the supervention of acute on chronic cystitis the disease may be of the acute character from the first. This is especially the case after operations such as the removal of growths, and in paraplegia after fracture of the spine. The urine contains pus and blood, and is ammoniacal. The pain and frequency of micturition (except in paraplegic cases where the bladder is insensitive) are extremely marked, constituting the condition known as strangury. The changes in the bladder are more marked than in chronic cystitis. There may be extensive hæmorrhage into the mucous membrane; sometimes a sloughy condition of the mucous membrane has been observed. After difficult labour the whole of the mucous membrane, and most of the muscular wall of the bladder, has separated as a slough.

The chief danger of cystitis lies in the difficulty of completely cleansing the bladder, and in the advanced age of many of the patients. When other measures fail, free drainage of the bladder through a perineal or supra-pubic wound is the best resource, and if not left till too late, that is, till the kidneys have become affected, has a wonderfully good effect.

Tumours of the Bladder.

Malignant are rather more frequent than innocent growths. The latter are papillomata, fibro-papillomata, and myxomata.

Papilloma, or villous tumours, are typical papillomata. Each projection of the surface consists of a long, slender cone composed

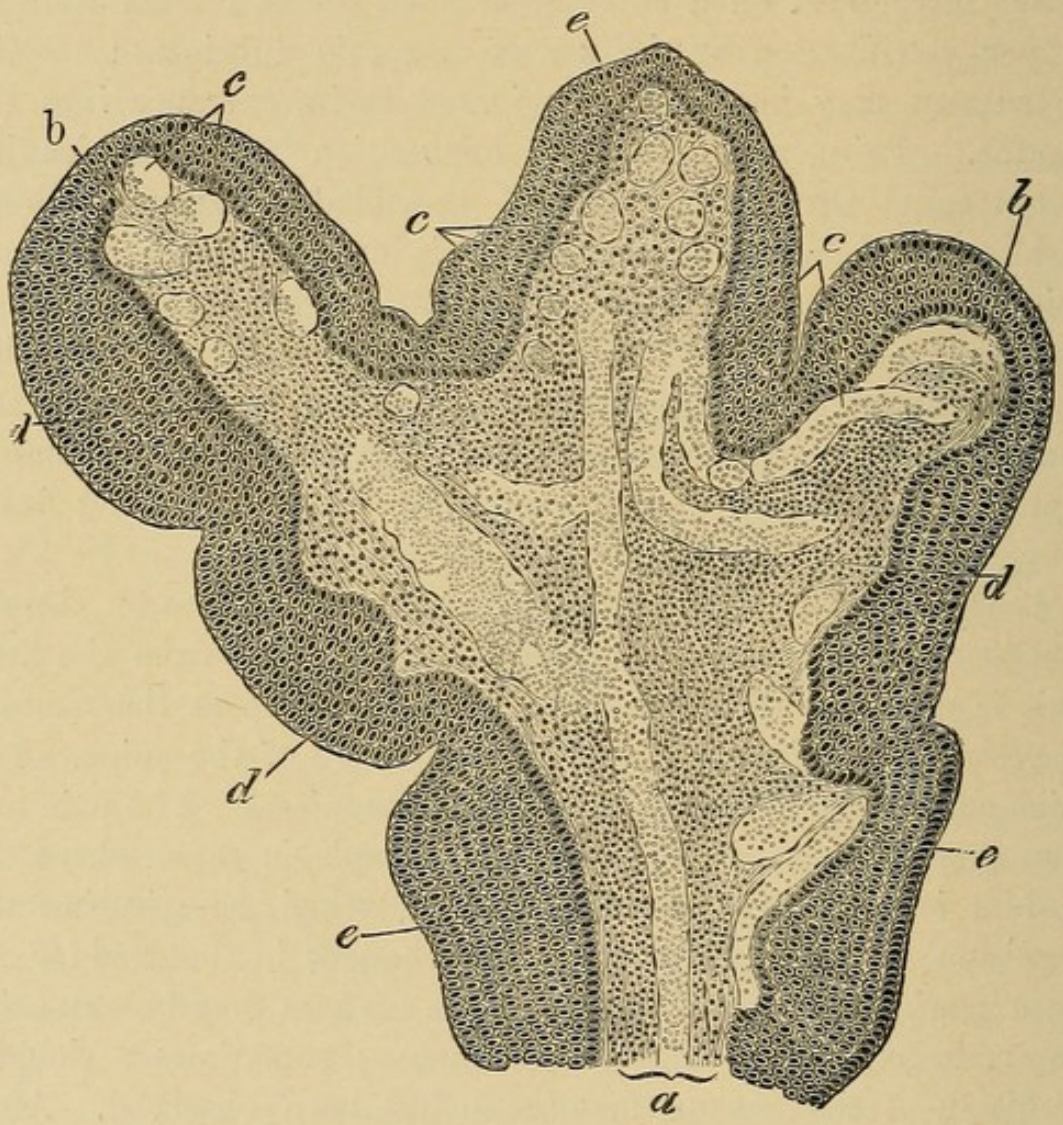


FIG. 168.—Portion of excrescence of a vesical papilloma, evacuated with the urine. *a*, Trunk of the papilla; *b*, branches of papilla; *c*, loops of blood-vessels cut longitudinally and transversely; *d*, stroma of the papilla infiltrated with small cells; *e*, stratified squamous epithelium. × 95. (Alum cochineal.) Weichselbaum, *Pathological Histology*.

of a central loop of blood-vessels surrounded by loose connective tissue, which is covered by an outer layer of epithelium, resembling the normal epithelium of the bladder. Sometimes, especially after sounding, these "villi" become detached, and the microscope reveals their presence in the urine and enables a

diagnosis to be made. They are commonest in middle age. They bleed readily. They are usually sessile, but sometimes pedunculated. In the latter case they may cause retention of urine

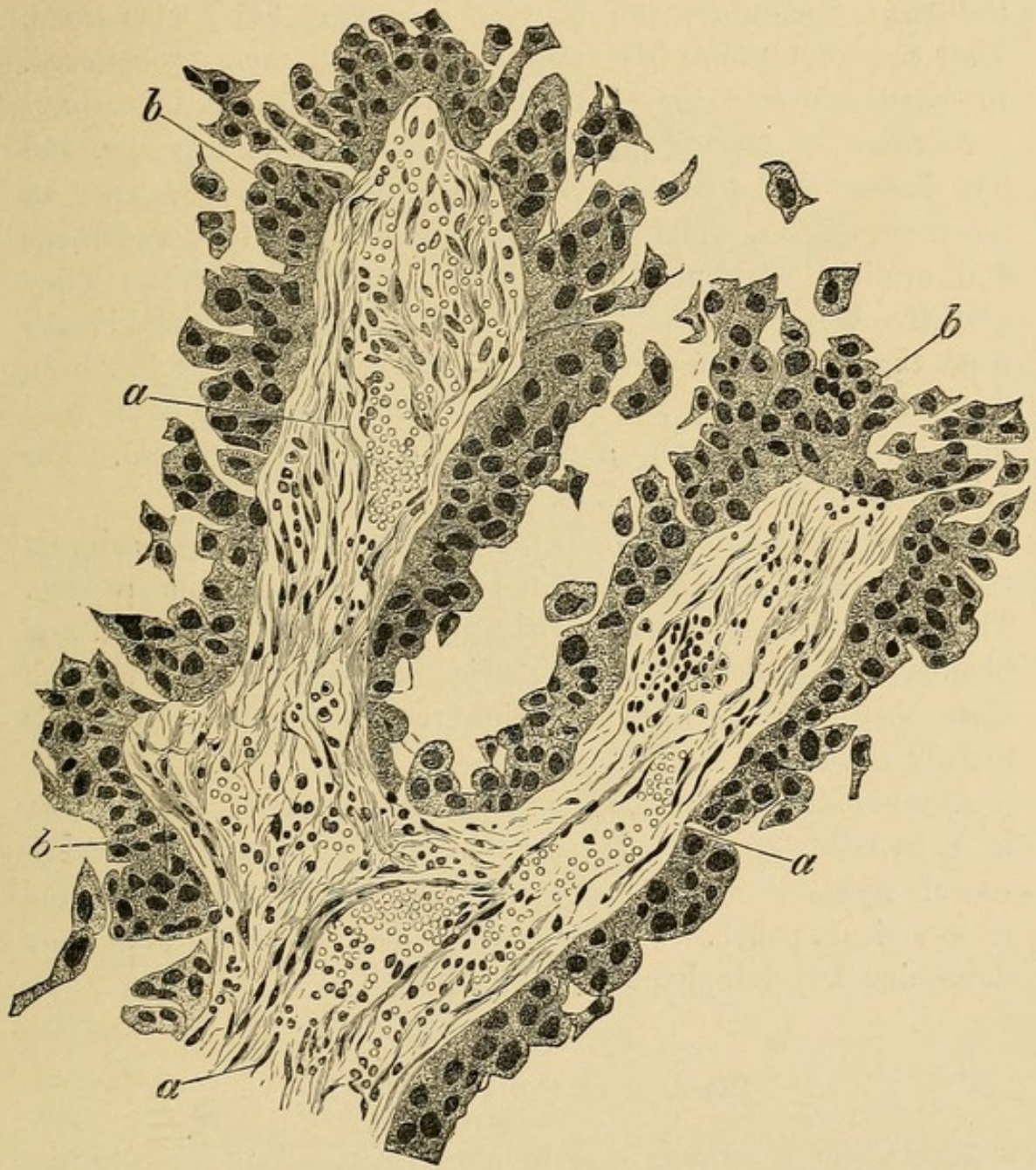


FIG. 169.—Branching villus-like structure from a cancer of the bladder. *a*, Connective-tissue stroma; *b*, polymorphous cancer cells. × 545. Weichselbaum, *Pathological Histology*.

by falling on the neck of the bladder and blocking it each time urine begins to flow. They may be very numerous.

Fibro-papilloma is merely a papilloma in the base of which much fibrous tissue is developed.

The histological characters of papilloma of the bladder are shown in Fig. 168.

Mucous Polypi of the bladder resemble in appearance those common in the nasal fossæ. They are rare, and occur in children. Sometimes the bladder is almost filled with them. They cause symptoms like those of stone, only more pronounced. In female children they may project at the meatus urinarius.

Sarcoma of the Bladder may be met with at any age, and like **Cancer of the Bladder** is in the present state of science an incurable disease. Malignant growths give rise to hæmorrhage and cystitis more rapidly than the innocent growths. They also frequently determine secondary changes in the kidneys from the obstruction they cause. After the bladder has been drained by making an external opening, the growth may invade the skin, causing a fungating mass in the wound. For Histology of Cancer, see Fig. 169.

Retro-vesical tumours. Large sarcomata sometimes arise in the connective tissue between the bladder and the rectum. Pushing their way upwards they may flatten the prostate and bladder and project into the pelvis. It is remarkable in such cases that little or no obstruction to the passage of urine is usually caused.

Another tumour which forms at the back of the bladder is a hydatid cyst. It is probable that at first it is a peritoneal hydatid which has gravitated to the bottom of the recto-vesical pouch. There it becomes encysted and may determine hypertrophy of the bladder.

Diseases of the Prostate, etc.

Prostatitis is usually due to gonorrhœa. Acute prostatitis, leading to abscess formation, is marked by fever and great pain and tenderness in the perineum. At first there is frequency of micturition; later, there may be retention from swelling. Abscess frequently results, and then redness from œdema is observed in the perineum. The pus may escape into the urethra, or into the rectum; rarely it extends into the tissue around the prostate, occasioning dangerous cellulitis.

Chronic Prostatitis is the commonest cause of "gleet," and is due to gonorrhœa. It may be the termination of acute prostatitis. It is marked by slight enlargement and tenderness of the gland, and is very obstinate to treatment.

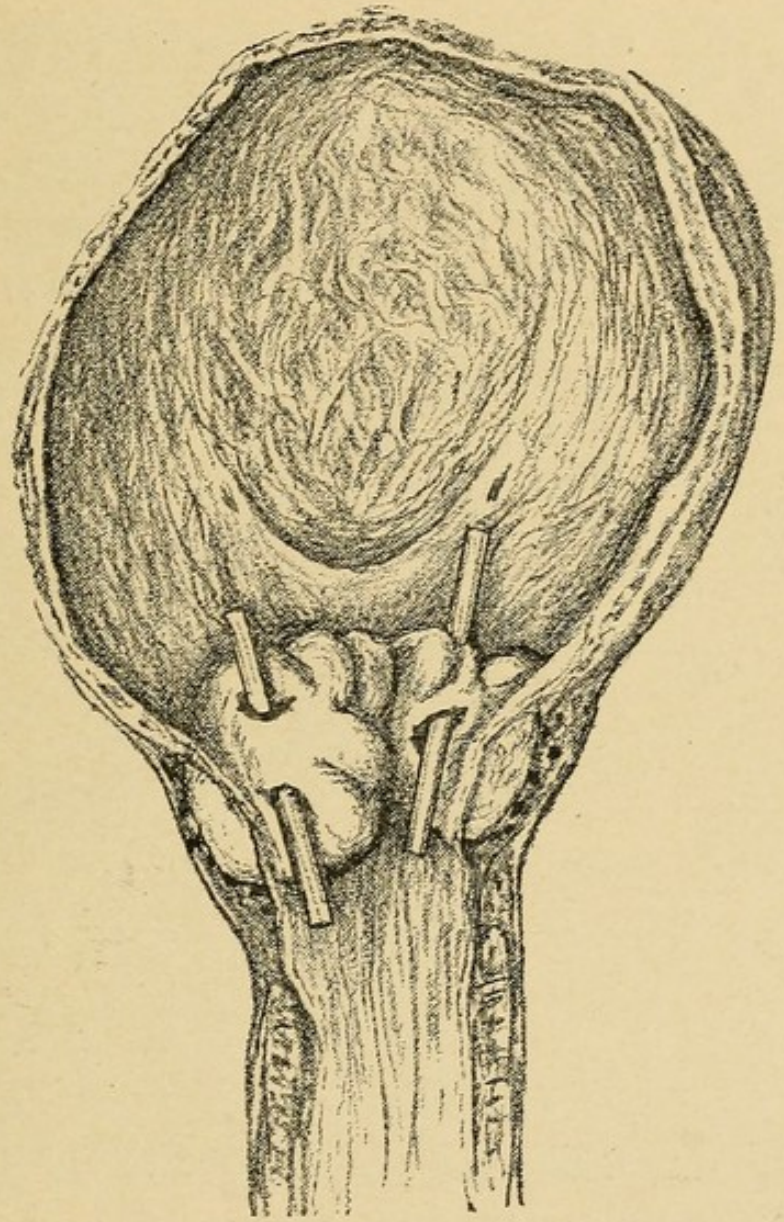


FIG. 170.—A bladder with the prostate and part of the urethra. Both the lateral and middle lobes of the prostate are enlarged. Two false passages, marked by white rods passed into them, were made during catheterization shortly before the patient, an old man, was admitted to hospital, where he died of cystitis and surgical kidneys.

Tubercle of the prostate and prostatic calculi will be described later.

Hypertrophy of the Prostate. After the age of fifty the prostate very commonly becomes enlarged. It has been estimated that "hypertrophy" of the prostate is present in as many as

30 per cent. of men over sixty years; but it is only in a relatively small proportion of these cases that the enlargement gives rise to symptoms of obstruction. This occurs chiefly when what is termed the middle lobe of the gland is enlarged so as to form

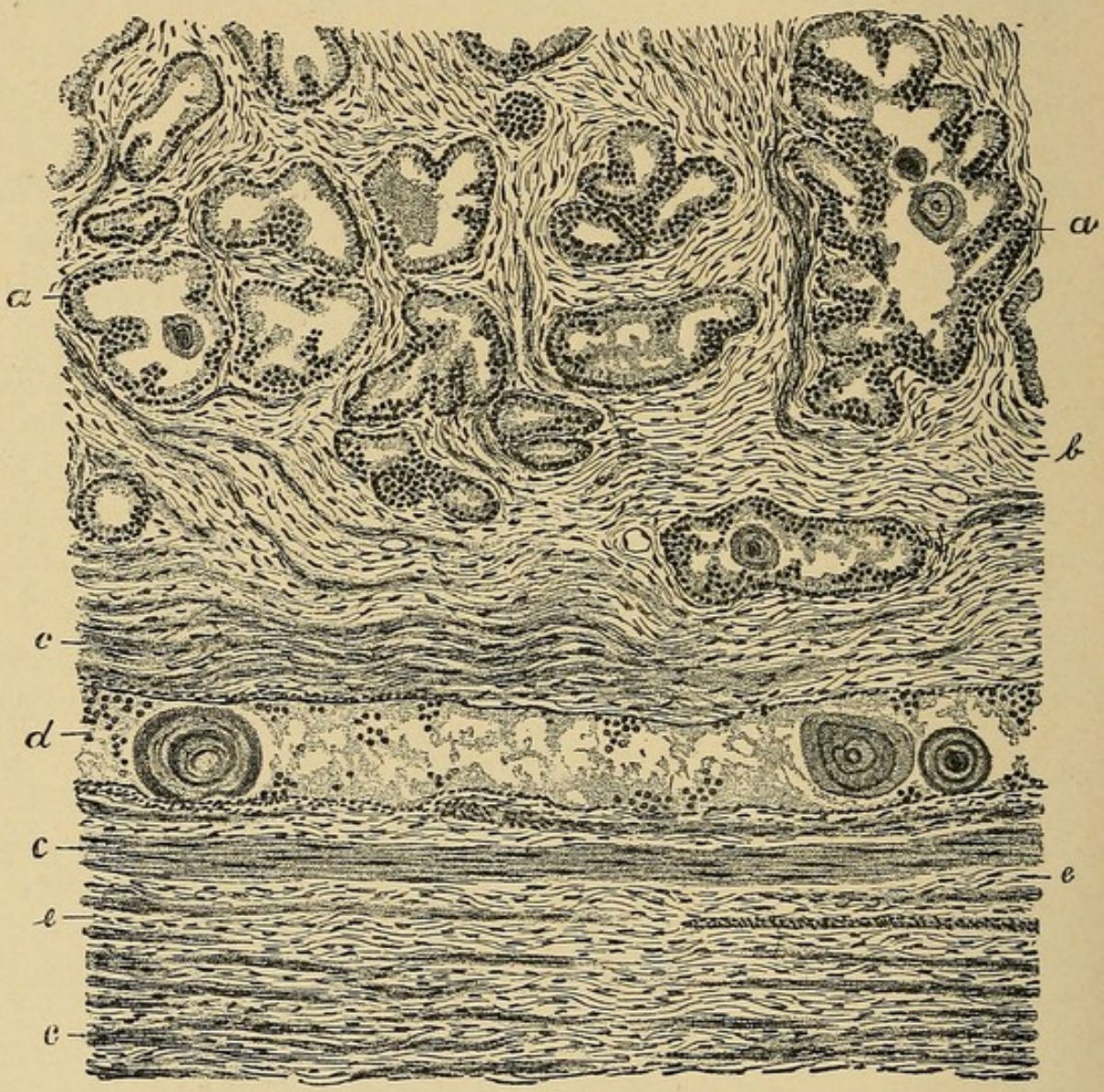


FIG. 171.—Hypertrophy of the prostate, adenomatous form. *a*, Acini, some of them dilated and containing small corpora amylacea; *b*, interacinous connective tissue; *c*, bundles of smooth muscle; *d*, gland duct with larger laminated corpora amylacea; *e*, intermuscular connective tissue. × 70. (Hæmatoxylin and eosin.) Weichselbaum, *Pathological Histology*.

a valve at the neck of the bladder. This lobe lies beneath the mucous membrane of the neck of the bladder, and a moderate degree of enlargement is sufficient to cause obstruction (see Fig. 174).

Enlargement of the lateral lobes in some cases also aids in

producing the same effect.[1] When the lateral as well as the middle lobes are markedly enlarged, the gland forms a collar-like projection at the neck of the bladder, as shown in Fig. 170.

The nature of the enlargement varies slightly in different cases, according as the muscular or the glandular constituents of the gland are the more concerned. In six hypertrophied prostates examined successively, the author found both the muscular and the glandular parts were about equally concerned. And although there were no separate encapsuled tumours, the increase of the epithelial parts could only be termed adenomatous in character. In all six cases small calculi (*corpora amylacea*) were present in the tubules. Sometimes distinctly encapsuled tumours are found on making a section through an enlarged prostate. Such tumours have a definitely adenomatous structure (see Fig. 171).

The concretions in the tubules give the amyloid reaction to iodine. They form the nuclei of the larger prostatic calculi.

The grave remote effects of prostatic enlargement will be noticed later on. The benefit obtained in a certain number of cases by castration, or removing portions of the vasa deferentia, goes to show that in some cases the hypertrophied prostate behaves to a certain extent like the normal gland, which also atrophies after castration.

Affections of the Urethra. Besides the consequences of gonorrhœa already dealt with, the urethra is subject to extension of cancer from the glans penis or prostate, or, more rarely, from the corpora cavernosa or bladder. In women frequently, and sometimes in men, a small painful fleshy growth, caruncle, is observed. It is readily removed by the cautery.

Urinary Calculi. In persons whose urine contains an excess of uric acid or of urate of ammonia, these bodies are apt to crystallize out, forming calculi. Ordinary "gravel" of the gouty consists of numerous minute uric acid calculi, composed of aggregations of coffin-shaped crystals, which form in the tubules of the kidney and pass with the urine along the ureter

[1] This is effected by the enlarged lateral lobes being pressed together across the middle line. There is no stricture of the urethra, which on the contrary increases in size as the gland enlarges, the increased length necessitating additional length in the curve of metal prostatic catheters.

into the bladder, causing a certain amount of irritation to the various parts, especially about the neck of the bladder. It would appear that a catarrhal condition of the tubules of the kidney is necessary to the formation of uric acid crystals. In other persons the amount of oxalate of lime contained in the urine is excessive and crystallizes out as octahedra, which, like uric acid crystals, may unite to form calculi. In many persons the urine, though otherwise healthy, becomes alkaline at certain periods of the day. In other persons the urine is persistently alkaline. In alkaline urine, crystals of the phosphate of ammonia and magnesia and an amorphous deposition of phosphate of lime may occur and a calculus result. Many calculi show alternation of uric acid or oxalate of lime with phosphate, or more commonly a deposit of phosphate forms about a uric acid or oxalatic calculus, when pyelitis or cystitis has supervened and the urine become alkaline. Some persons have the peculiarity of metabolism, which results in the formation of cystine in the kidney in the shape of crystals which have the form of flat hexagonal plates and give the urine a micescent appearance, and which, like other crystals, may form calculi. Cystine calculi are like bees-wax in texture, and contain much sulphur in combination.

If some gravel or other crystals becomes lodged either in a tubule or a calyx of the kidney its grains, united by freshly deposited crystals, form a calculus which may remain and enlarge in the kidney, or may pass along the ureter, causing an attack of renal colic. If it becomes impacted in the ureter it may lodge there and produce obstruction to the escape of urine. For further details, see p. 383.

Some Injuries to the Genito-Urinary Passages.

Rupture of the Urethra. By kicks or falls on the perineum a part of the wall of the urethra may be torn. Or when the pelvic rami are broken and pressed together the urethra may be torn across. In either case the first probable effect will be an escape of blood by the meatus independently of micturition, and if the injured person attempts to pass water, extravasation

of urine and its effects will ensue. If the urethra has been completely severed an intractable stricture results, and hence it is desirable in most cases to perform a perineal section, so that the state of the urethra can be ascertained and the rent in the mucous membrane sutured (see Fig. 172).

When the injury is caused by a fracture of the pelvis, the anatomical disorganization may be much more extensive. In such a

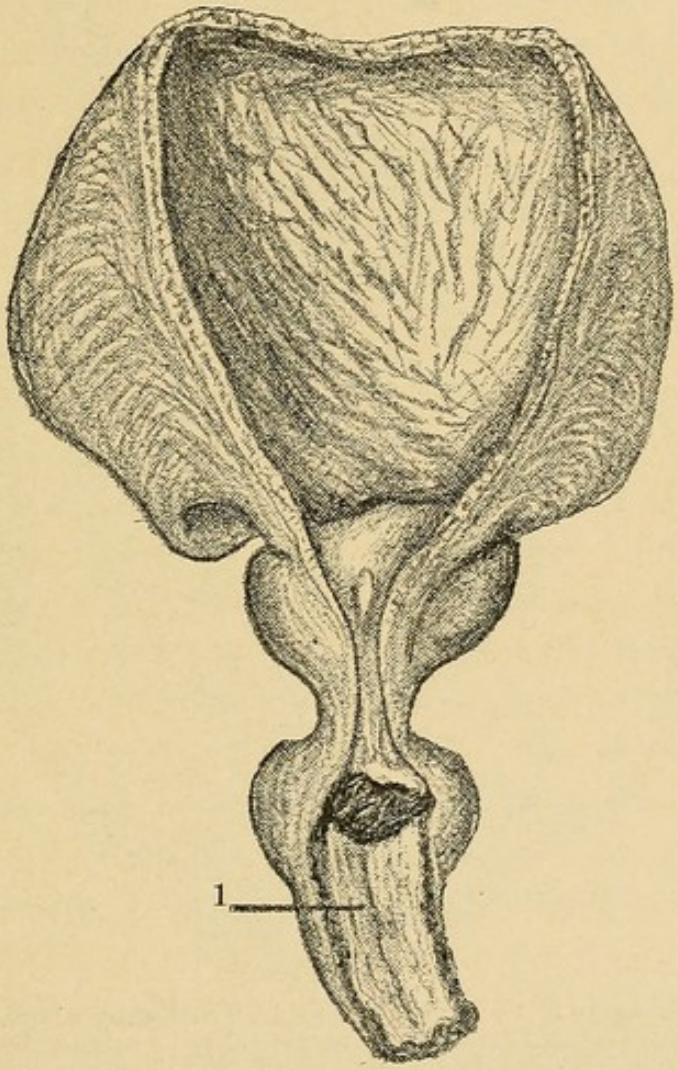


FIG. 172.—Ruptured urethra. There is a complete division of the mucous membrane (1) opposite the bulb; in the latter there is a cavity which opens into the urethra. From a cabman who, in getting down from his seat on a hansom-cab, fell astride the edge of a board. He bled to death from the artery of the bulb. (1242.)

case[1] it has been found that a catheter appeared to pass into the bladder when it only entered an artificial space in front of the bladder. Such an injury can only be produced by a very great force capable of breaking the pelvis and crushing together the two ossa innominata.

[1] H. W. Page, *British Medical Journal*, 1896.

Rupture of the Bladder. Traumatic ruptures may be classed as (1) intraperitoneal, and (2) extraperitoneal; a variety of the latter kind may be termed subperitoneal. There are also cases in which the bladder is spontaneously ruptured from over-

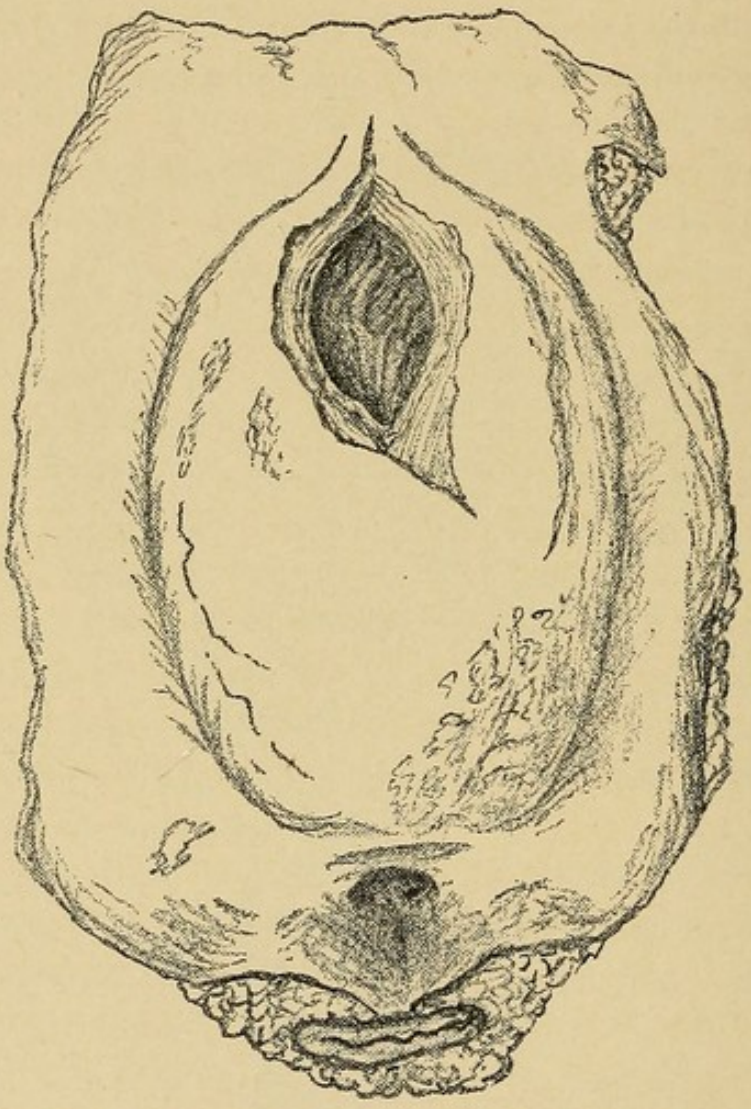


FIG. 173.—The posterior surface of a bladder with the peritoneum covering it, showing an intra-peritoneal rupture which begins behind the attachment of the urachus and extends for some two inches downwards. The rectum cut across is seen at the lower end of the preparation. From a man who was roughly handled when suffering from retention of urine. Ha passed a little blood by the urethra on the second, and died on the third day, having received no medical treatment. General purulent peritonitis was found. There was no stricture or other abnormality to account for the retention. (1202 A.)

distension or atrophy, or is perforated by ulceration. To this class of cases the term "spontaneous rupture" is applied.

Intraperitoneal Rupture of the Bladder. Violent pressure on the lower part of the abdomen when the bladder is distended produces this lesion. A typical example of the condition is shown

in Fig. 173. The injury causes great pain both from the rent in the bladder and from the contact of the peritoneum with urine. If the patient passes any urine it is small in quantity, flows without force, and is blood-stained. By these and other symptoms the lesion is diagnosed, and an immediate abdominal section for the purpose of suturing the rent in the bladder must be undertaken.

Subperitoneal Rupture. Only one example of this lesion has come to the author's notice. It was in a middle-aged man who had an impassable stricture of the urethra, and was knelt on in a brawl. The rent was in the posterior surface of the bladder, but the peritoneum remained intact.

The case was under the late W. Pye. There was great pain and dulness and prominence in the vesical region as high as the umbilicus. The position of the dulness did not change with that of the patient. No catheter could be made to pass the stricture. The case appeared to be one of retention merely. Suprapubic aspiration was done, and over a pint of what appeared to be blood was withdrawn. This, however, made but little difference to the area of dulness, so that a perineal section was performed. The patient died of suppurative pyelonephritis.

Extraperitoneal Rupture other than subperitoneal means a rent in the bladder at some part not covered by peritoneum. Thus it may be on the anterior surface. In cases of fracture of the pelvis the rent may be caused by a spicule of bone perforating the bladder, or by a sudden separation of the symphysis the wall of the viscus may be torn. Extravasation of urine and cellulitis result.

Spontaneous Rupture can hardly occur in a healthy bladder, but when the organ is distended and thinned from obstruction, *e.g.* that of enlarged prostate, retroversion of the gravid uterus, etc., the ordinary pressure of the abdominal muscles may be sufficient to cause rupture. In acute cystitis a portion of the bladder wall may slough; both are instances of spontaneous rupture. One such case was that of a young man who had a stricture of the urethra, and sustained a fracture of the dorsal spine. He lived six months. Acute cystitis supervened; the posterior wall of the bladder was ulcerated through into the peritoneum, and death from suppurative peritonitis followed.

Rupture of an Ureter may occur from great pressure on the abdomen. If the peritoneum covering the ureter is not torn, urine collects behind it, forming a swelling. In such cases the ureter has been successfully re-united. An ureter may be divided near the bladder in performing operations on the pelvic organs. Such an accident has been successfully met by implanting the upper end of the divided ureter into the bladder or the rectum.

Laceration and Rupture of the Kidney. By laceration is meant a tearing of the cortical substance from violent pressure. This gives rise to a transient hæmaturia which soon passes off and the patient recovers. By rupture is meant complete subdivision of the kidney into two or more portions. Large branches of the renal veins may be injured, and unless the kidney be removed the patient may bleed to death. In one such case a youth had been nipped between buffers. Blood, urine, and fæces were found in the clothes. Rupture of the bladder was excluded. The patient recovered from the shock and did well for three days, when a pleural effusion formed on the right side. On drawing this off it was found to consist of almost pure blood, and inferred that it was due to a laceration of the lung. Pleurisy of the left side then supervened, and the patient died a week after the accident. On examination after death it was found that the right kidney had been ruptured and herniated through the diaphragm, and the hæmorrhage into the right pleura had come from a torn renal vein. The pleurisy of the left side was unexplained.

Some Affections of the Urethra and their Effects.

The Complications and Sequels of Gonorrhœa. If the infiltration of the submucous tissue is extensive the corpus spongiosum may become firm and inelastic, and in this condition during erections causes the penis to curve downwards. This painful condition is termed *chordee*. If the infection spreads along one of Littré's glands there results a *follicular abscess* which may open internally and heal, or externally; in the latter case a *fistula* may form. The process sometimes spreads along the

ducts of Cowper's glands, giving rise to *Cowperitis*, which may result in the formation of a deep-seated perineal abscess. *Prostatitis* and *cystitis* are among the occasional complications of gonorrhœa. The cystitis is usually limited to the neck of the bladder and runs a favourable course, but occasionally it is more serious.

Epididymitis when it occurs is usually observed towards the end of a case, and with its onset the urethral discharge becomes suddenly lessened. From the epididymis inflammation may spread to the peritoneum. The germs of the infection may be transplanted to the conjunctiva, where a most acute and destructive inflammation results (see p. 326). A general pyæmia sometimes results, and may show itself by acute inflammation of joints (see p. 189).

Urethritis other than Gonorrhœal.

A catheter tied in the urethra sometimes determines a suppurative catarrh. The ordinary pyogenic and other bacteria have also been found in purulent urethritis without the gonococcus. Cases of membranous urethritis have been described.[1] Crystals of uric acid, oxalate of lime, or phosphates sometimes occasion urethritis. Among the results of urethritis stricture is the most important.

Stricture of the Urethra.

Thompson has defined stricture as "an organic or permanent change in the walls of the urethra which narrows them or prevents their opening or dilating to their natural calibre." Such a condition is most commonly due to the cicatrization of parts of the urethra most deeply infiltrated by gonorrhœa. The worst form of stricture follows traumatic rupture of the urethra, and an intraurethral chancre may also determine a stricture of the fore part of the canal. The average time required for the development of a stricture after gonorrhœa is two years. A narrow meatus, by preventing the free exit of

[1] W. H. Battle, *Lancet*, 1893, vol. ii., p. 303.

discharge, predisposes to stricture. The anatomical forms of stricture are very numerous since a scar of any shape will narrow the passage. The commonest kind is a general narrow-

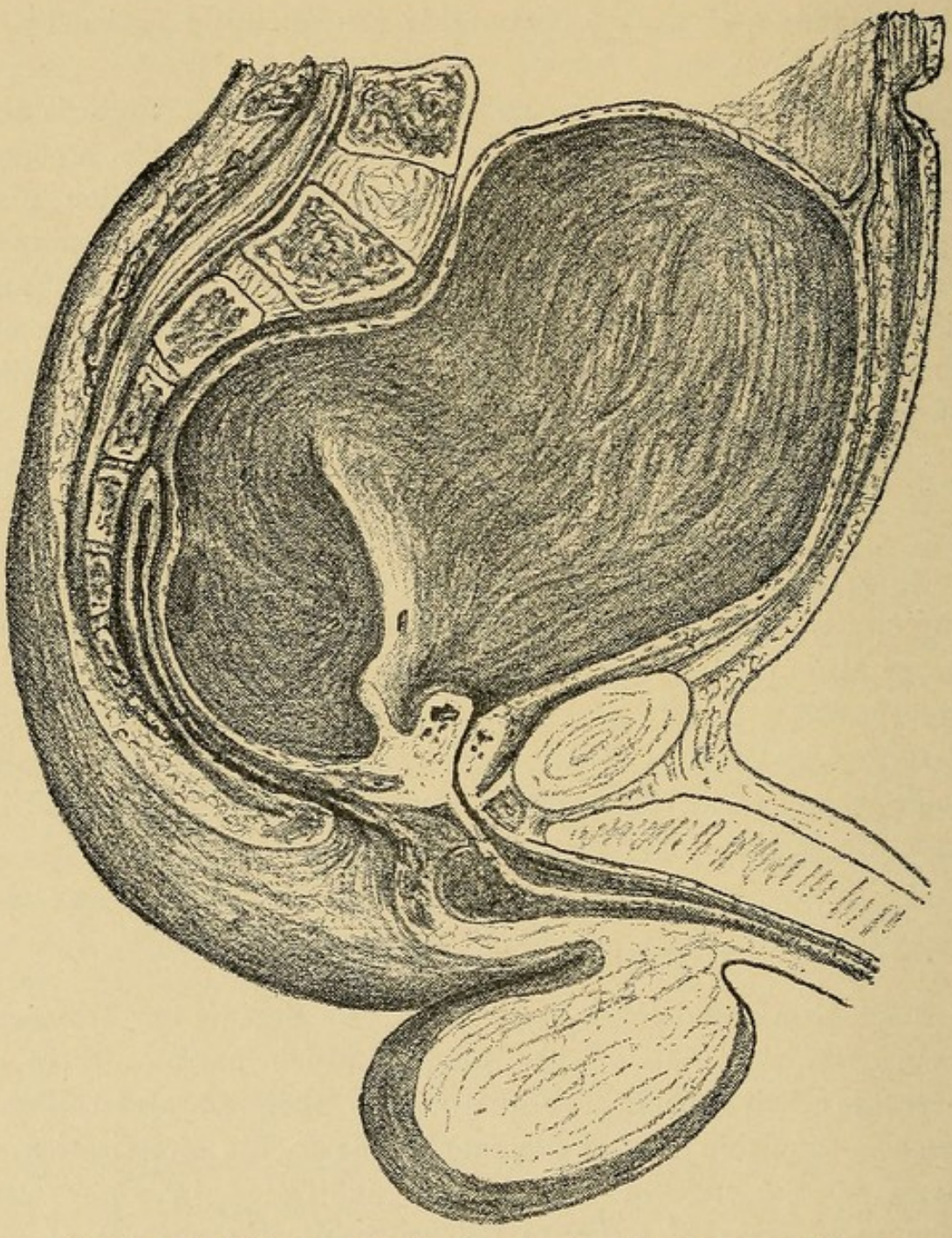


FIG. 174.—A bladder greatly dilated owing to obstruction caused by an enlarged middle lobe of the prostate. From an old man who was admitted into hospital in an unconscious state, dying from pneumonia. (1237 A.)

ing of a segment of the urethra, say an inch or more—the *linear* stricture. If the narrowed segment is very short the *annular* stricture is constituted. A *bridle* stricture is said to

exist when fibrous bands stretch across the lumen dividing it into two or more passages. Such bands are formed by the union of granulations on opposite sides of the lumen. A similar condition may possibly be formed by short false passages being kept open by the flow of urine through the artificial tunnel.

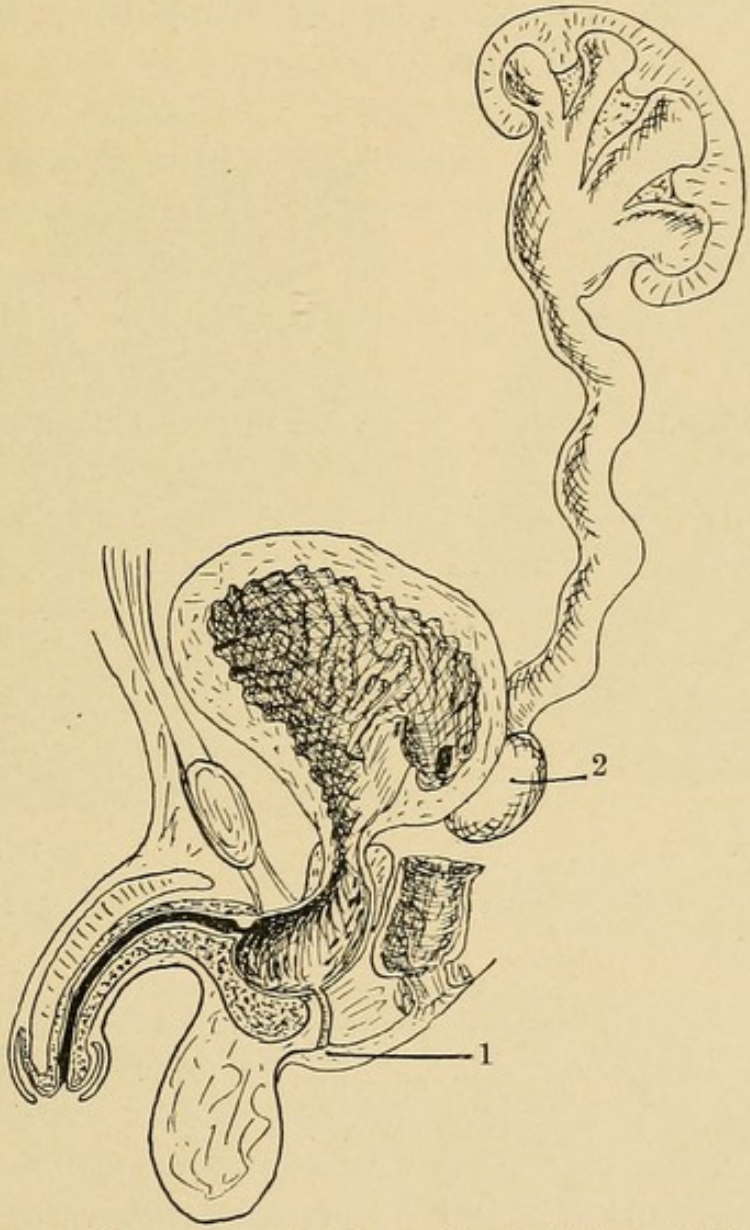


FIG. 175.—A diagram showing the results of stricture of the urethra. There is a thickening (annular stricture) in the wall of the urethra in front of the anterior layer of the triangular ligament. Behind this the urethra is dilated, and at 1 opens by a fistula in the perineum. The bladder is hypertrophied and dilated, and at 2 presents a sacculus. The ureter and pelvis of the kidney are dilated, and the kidney is atrophied—hydronephrosis.

The Effects of Stricture, etc. The part of the urethra behind the narrowing is exposed to greater pressure during the action of the bladder, and hence becomes dilated.

The ducts of the glands of this part of the urethra become dilated and may ulcerate, and thus lead to extravasation of urine, perineal abscess, and this opening on the surface to the formation of a fistula. The *bladder* having to overcome increased resistance, thus tends to be affected in two ways, (1) by hypertrophy; (2) by dilatation. In young subjects, in whom adaptive powers are active, the hypertrophy; in old subjects,

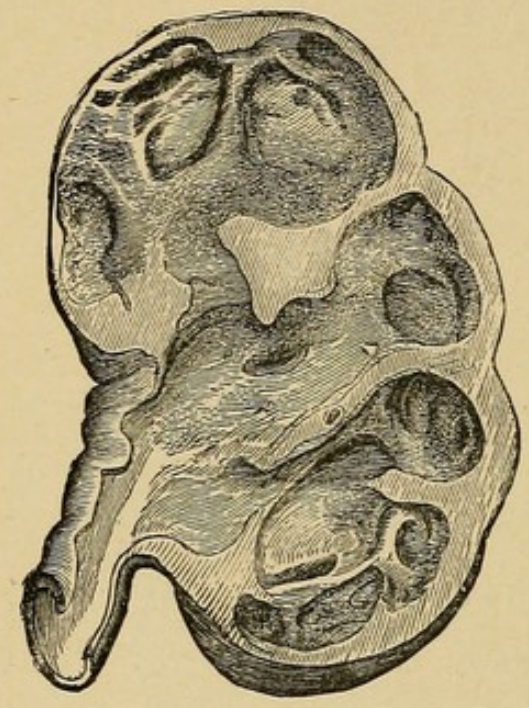


Fig. 176.

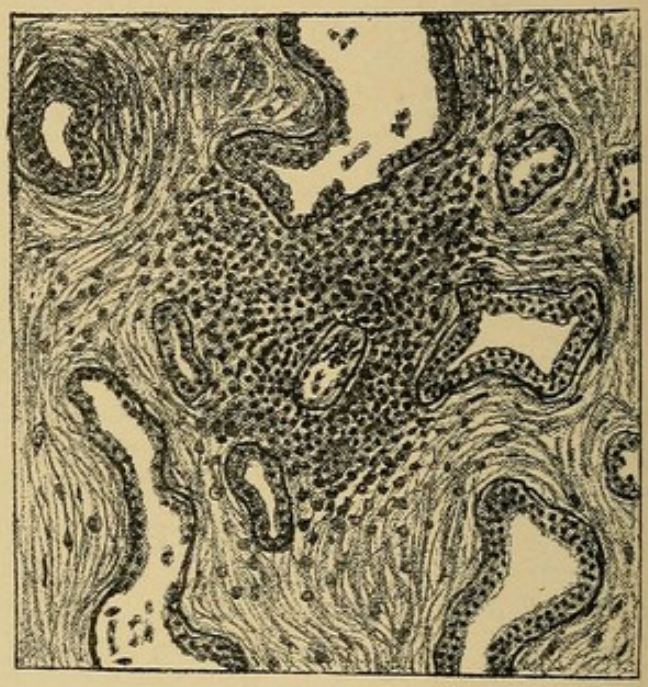


Fig. 177.

Fig. 176.—Hydronephrosis with granular atrophy of the kidney. The other kidney showed a marked compensatory hypertrophy. (Virchow.) Coats, *Manual of Pathology*.

Fig. 177.—Section of the kidney of a man who died of suppurative pyelonephritis, the result of hæmorrhage into the bladder following perforation of an enlarged prostate (case shown in Fig 170) by a catheter introduced for retention of urine. The tubules are dilated, and their epithelium is being shed; there is an excess of fibrous intertubular tissue. In the centre of the figure, around a small tubule, is an abscess.

who have diminished adaptive powers, the dilatation, will be the more marked. Thus in cases of stricture, which as a rule begin before thirty, the hypertrophy is the more marked; whilst, when the obstruction is caused by an enlarged prostate, which is more often met with in elderly persons, dilatation is usually the more pronounced feature (see Figs. 174, 175).

In hypertrophy, muscular fasciculi stand out in relief on the inner surface of the bladder, giving it a reticulated appearance.

The areas within the ridges caused by the prominent muscular bands yield more readily and give rise to sacculi (see Fig. 175).

Whether the obstruction be due to phimosis (congenital narrowing of the meatus), stricture, or enlarged prostate, these changes are produced in time, and further, the increased intravesical pressure offers an obstacle to the escape of urine from the ureters in which and in the pelvis of the kidney the same effects, dilatation and hypertrophy, are produced. In the kidney itself atrophy from pressure results. The pyramids become flattened. If the obstruction is in one ureter alone the whole kidney may be absorbed, leaving only the distended pelvis and capsule (see Figs. 175, 176).

Fig. 178.—Part of the tubule in the small abscess shown in Fig. 177. Numerous bacteria resembling those of decomposition are present in the tubule, and are invading the tissue beyond it. × 500.

Such kidneys show changes, known as those of interstitial nephritis, *i.e.* increase of connective tissue between the tubules, but in the general effects there is a marked difference between these cases of interstitial nephritis secondary to obstruction and the ordinary contracted kidney. In the former condition the increased arterial tension and hypertrophy of the heart and of the arteries are wanting.

Besides the mechanical effects the stagnating urine is readily infected with putrefactive and pyogenic bacteria, whether introduced by the catheter or by other means. In this way suppurative cystitis, pyelitis, and nephritis occur. All the abnormal conditions, such as hydronephrosis, etc., that result

from obstruction, are grouped together under the name of *Surgical Kidney*, but suppurative pyelonephritis is the surgical kidney *par excellence* (see Figs. 177, 178).

Tuberculosis of the Kidney and Bladder.

Tubercle attacks the kidney in three different forms. First, as part of a general tuberculosis, in which form it causes no special symptoms; second, as a local tuberculosis of the pelvis, and the surface of the projections of the pyramids into them—tubercular pyelonephritis; third, as a tubercular focus or abscess in one part of the kidney substance.

Tubercular Pyelonephritis gives rise to symptoms which may closely resemble those of stone in the kidney: pain in the loin radiating to the groin, and in the male to the penis, frequency of micturition, attacks of renal colic due to passage of shreds of tubercular slough separated from the kidney, hæmaturia, pus containing tubercle bacilli in the urine, may all be present. The affected kidney is usually distinctly enlarged, but occasionally cicatricial shrinking occurs. As the disease progresses the tubercular infiltration may perforate the capsule and infect the perinephric fat, causing an abscess which tends to point in the back. If only one kidney is affected, removal is the best treatment.

In tubercular abscess of the substance of the kidney the symptoms are not so marked as in tubercular pyelonephritis, and until either rupture into the pelvis of the kidney or extension to the perinephric fat occurs, the diagnosis may be impossible.

Tuberculosis of the Bladder seldom or never occurs as a primary affection, but as an extension, either from tubercle of the kidney or from tubercle of the testis, seminal vesicles, etc. The symptoms are those of cystitis and ulceration of the bladder (see Fig. 179).

Endemic Hæmaturia occurs in Egypt and South Africa, and is due to the presence of a fluke or distoma, the Bilharzia hæmatobia. The adult male is about half an inch, the female is nearly three quarters of an inch in length. The embryos enter the

body in drinking water, and develop in the vesical, portal, rectal, and other veins. The worms breed in the veins, and their ova penetrate the surrounding tissues, especially those of the bladder, kidney, and rectum, where they give rise to inflammation and

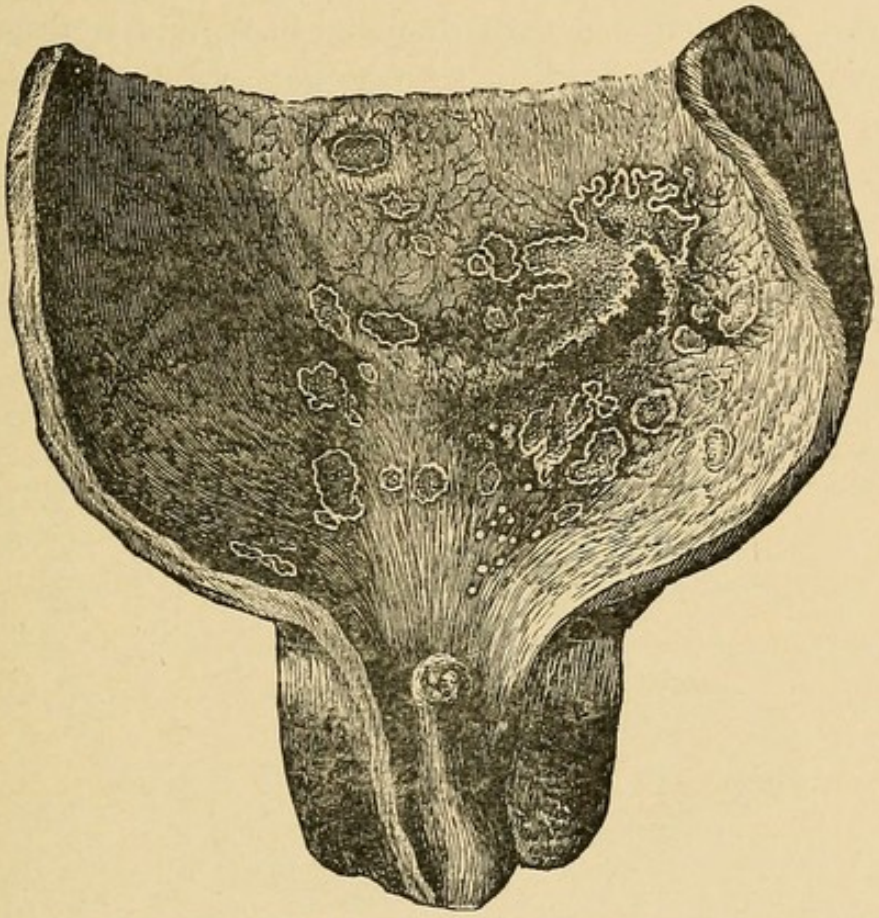


FIG. 179.—Tuberculosis of the bladder. Many round or serpiginous ulcers are shown. These are superficial, but are defined by their white edges which represent recent tubercles. There are several white spots, indicating tubercles which have not yet ulcerated. (VIRCHOW.) Coats, *Manual of Pathology.*

ulceration. In the bladder and kidney they cause recurrent attacks of hæmaturia, cystitis, etc. The ova are found in the urine. If the patient leaves the infected district the attacks usually cease in a year or two.

Urinary Calculi.

The Effects of Calculi in the Kidney. If the calculus remains embedded in the kidney substance it may become encapsuled and give rise to no symptoms. If, on the contrary, it is free in the pelvis or in a calyx, it causes pain, hæmorrhage, and

frequency of micturition, and more or less pyelitis. The latter may become purulent, calculous pyelitis. If the deposit continues a calculus may fill the pelvis and send growing branches into the calyces, where they produce pressure-atrophy of the pyramids. The same is the result of mere blocking of the ureter by a calculus. The urine is then dammed back, and hydro- or pyonephrosis results, according to whether suppuration has commenced or not.

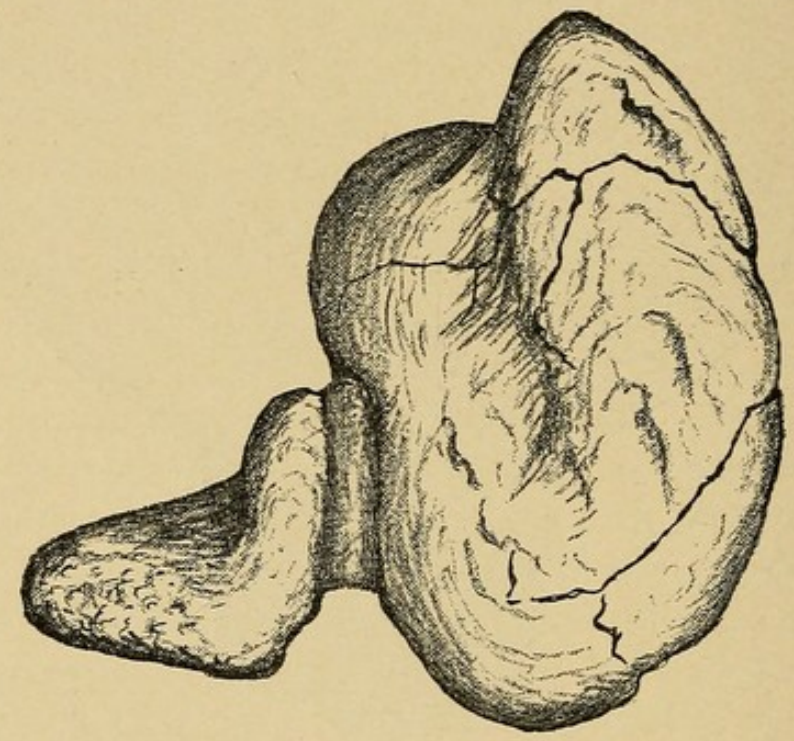


FIG. 180.—A calculus removed from the bladder of a man who had been paralyzed for six years. The stone shows traces of divisions which indicate that it resulted from the fusion of several separate calculi. The groove round the neck of the stone marks the position of the neck of the bladder; the tapering portion of the stone in front of this groove lay in the urethra. Nat. size.

When suppuration occurs the perinephric fat becomes matted together and adherent to the capsule of the kidney. And occasionally the perinephric tissues become infected with the result of the formation of a perinephric abscess.

If the stone passes into the bladder it may remain there and increase in size, or it may, whilst still small, pass into the urethra, where it may lodge, causing retention of urine, or ulceration with periurethral abscess and extravasation of urine; or it may escape from the meatus. Even then its possibilities are not exhausted. If there is a tight foreskin it may lodge between the glans penis and the orifice of the

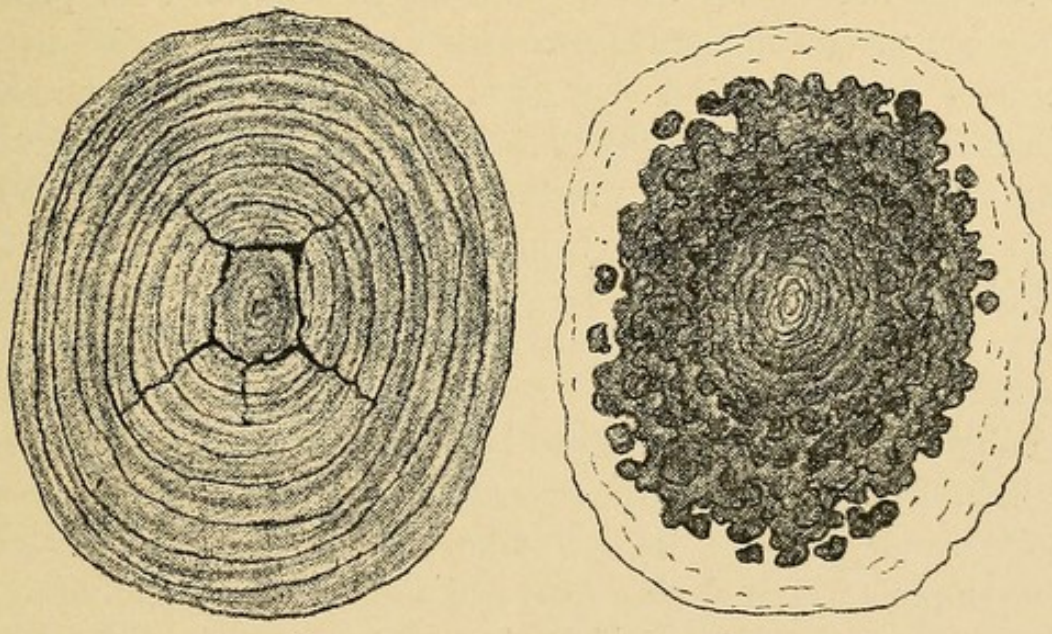
prepuce, and there acting as a ball-valve may produce all the evils of obstruction to the passage of urine.

In the Bladder a stone may cause but slight symptoms for a considerable time; as it increases in size and weight, pain and cystitis, the latter marked by the secretion of an excessive amount of mucus, results. Sometimes, especially in children, the calculus causes sudden stoppage in the flow of urine, save when the patient lies on the back. Hypertrophy of the bladder is produced by the frequency of micturition, the "ropy" quality of the urine making it more difficult to eject, etc. If calculi lodge in saccules they may be difficult to dislodge, and may readily cause ulceration and peritonitis. In the female a stone may ulcerate into the vagina.

Calculi may attain large dimensions by the continual addition of fresh layers (see Fig. 180).

Chief Characters of different Calculi.

Uric Acid Calculi. Yellow or buff in colour, moderately hard on section, the surface taking a polish and showing concentric



1 2

Fig. 181.—1, Section through an uric acid calculus; 2, section of calculus consisting of oxalate of lime with an outer coat of phosphates.

laminæ (their surface is somewhat rough). Uric acid being an entirely organic compound burns away before the flame of the blow-pipe (Fig. 181).

Urate of Ammonium Calculi occur chiefly in children. They have about the same hardness as uric acid stones, but on section they have a grey, mealy appearance, being non-laminated, and taking no polish. They are wholly combustible, and give off ammonia when heated with potash.

Oxalate of Lime Calculi are very hard and heavy. They are usually tuberculated on the surface, whence the name mulberry calculus, though now and then a renal oxalatic calculus is covered with needle-like crystals. On section they are usually dark, being blood-stained; they show a wavy lamination. On heating a fragment it leaves an ash of carbonate of lime (Fig. 181).

Phosphatic Calculi. The one most commonly occurring is the fusible calculus, portions of which in the flame of the blow-pipe fuse to a pearly bead. The calculi vary in consistence. Usually they are chalky and friable, sometimes harder, and on section are laminated.

The rarer calculi are phosphate of lime, carbonate of lime, cystine, and xanthine.

Prostatic calculi are formed by deposit of phosphate or carbonate of lime about the organic nuclei described on p. 371. They are more commonly met with as lentil-like bodies in the ducts of the gland, and they tend to project into the urethra. When they are more deeply placed they may be larger, and they may cause abscess in the prostate. Sometimes they can be felt from the rectum, and when multiple they give rise to a grating sensation. If the symptoms, pain and tenderness in the perineum, are marked, the calculi may call for removal either by the forceps from the urethra or by a median perineal incision.

Tuberculosis of the Prostate is not common, and is only met with in combination with tuberculosis of other parts of the genito-urinary tract. Its importance arises from the fact that the symptoms may closely resemble those of prostatic calculus. Abscess may form, and, if it does not empty itself into the urethra, may require perineal drainage.

CHAPTER XXVI.

DISEASES OF THE TESTIS AND NEIGHBOURING PARTS.

Torsion of the Spermatic Cord occurs in boys and young men, the subjects of retention of the testis in the inguinal canal. Pain and swelling in the inguinal region accompanied by reflex vomiting are the chief symptoms, so that the condition is very apt to be mistaken for strangulated hernia, more especially since a congenital hernia is usually present in the inguinal canal, complicating the condition. The torsion of the veins causes first intense congestion of the testis and epididymis; gangrene occurs if the testis is not removed. On cutting down and exposing the testis, the deep purple colour of the congested organ much resembles a piece of congested bowel, but palpation reveals the solid character of the swelling and clears up the diagnosis. The testis should be removed.

Rupture of the Vas Deferens is a rare injury, and results from severe pressure on the lower part of the abdomen. The duct usually gives way between the external abdominal ring and the urethra. Slight bleeding from the urethra and swelling of the testis are the chief signs of the injury. Sometimes an abscess forms within the pelvis.

Hydrocele. Almost any sac filled with liquid and connected with the testis is termed a hydrocele. The capacity for cyst formation in the neighbourhood of the testis is very great, as may be conceived from the diagram (Fig. 182).

Hydrocele of the Tunica Vaginalis. The commonest and most important form of hydrocele is due to distension of the tunica vaginalis with fluid which is effused passively. This is

called "chronic hydrocele." The condition is not uncommon in infants, but it is in middle-aged and elderly persons that it is most difficult to get rid of. The fluid is clear and of a yellow colour. It contains fibrinogen. In old standing cases the tunica vaginalis becomes thickened and may become calcified. The testis in time becomes atrophied from pressure: it lies at the lower and back part of the sac. The causation of hydrocele is by no means clear. In infants the condition frequently disappears after tapping and attention to the bowels, etc. In adults the condition is very obstinate. In most cases the best way of obtaining a radical cure is by opening the sac, mopping it out with 90 per cent. carbolic, and draining under an antiseptic dressing. When the sac is much thickened as much of it as possible should be excised.

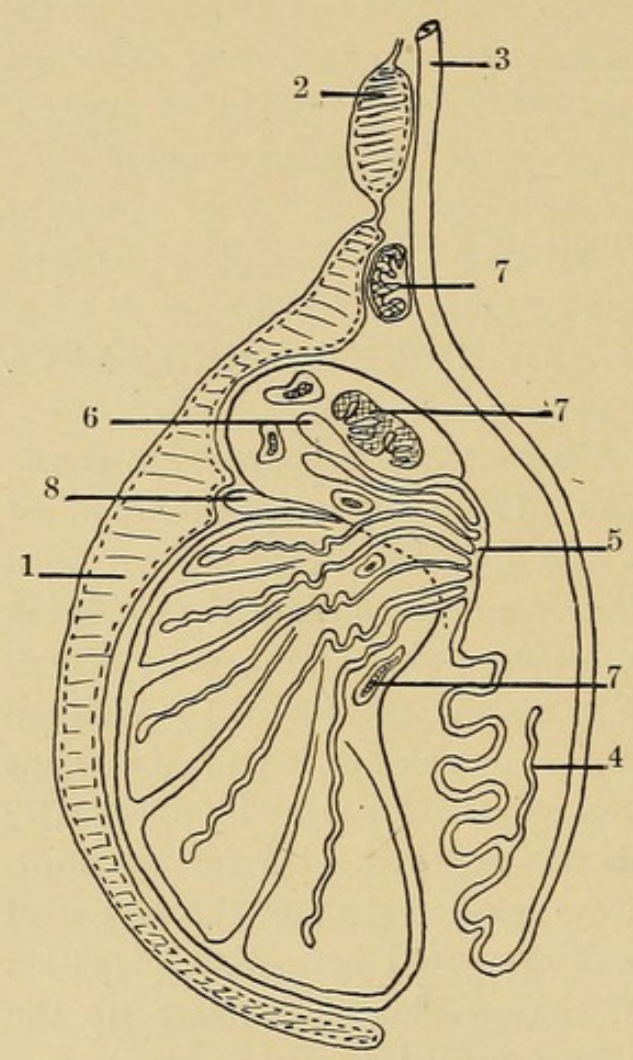


FIG. 182.—A diagram showing the possible sources of cysts in and about the testis. 1, Tunica vaginalis; 2, unobliterated funicular portion of the processus vaginalis; 3, vas deferens (originally Wolffian duct) continued into the tortuous canal of the epididymis, and near the junction of the two a vas aberrans, 4. Five tubules of the testis are represented in as many lobes separated by septa and prolonged by vasa efferentia (originally Wolffian tubes) to join the canal of the epididymis near 5; 6, one of two Kobelt's tubes, *i.e.* persistent Wolffian tubes which end blindly; 7, closed tubes constituting the paradidymis which represents remains of the Wolffian body; 8, the hydatid of Morgagni, representing the closed upper extremity of the Müllerian duct.

Congenital Hydrocele is an accumulation of fluid in a tunica vaginalis, which remains connected with the peritoneum. When the patient is placed on the back the fluid can by pressure be slowly emptied into the peritoneal cavity.

Hydrocele of the Canal of Nuck. In females a process of peritoneum corresponding to the processus vaginalis in the

male lies with the round ligament in the inguinal canal. This "canal of Nuck" normally becomes obliterated, but if part of it remains patent and fills with fluid, it constitutes a hydrocele which corresponds to an infantile hydrocele in the male.

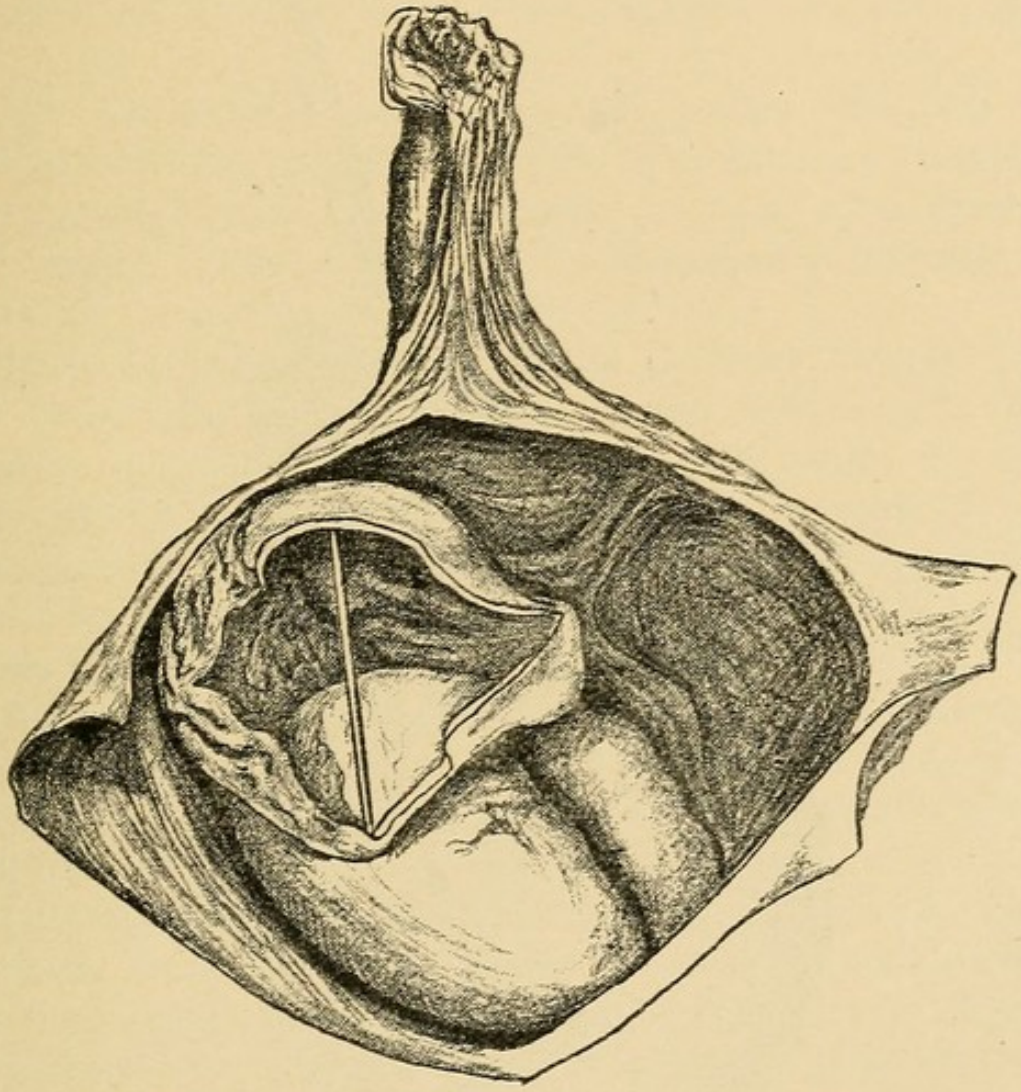


FIG. 183.—A cyst in the globus major of the epididymis projecting into the tunica vaginalis, hence called "encysted" hydrocele. The cyst has been incised and is propped open so that its wall is thickened at one part. The thickening is due to the epididymis with the vasa efferentia being stretched over the cyst. The fluid of the cyst was full of well-preserved spermatozoa. (1281.)

Infantile Hydrocele (*funicular hydrocele*) is a collection of fluid in a tunica vaginalis that remains in connection with an unobliterated funicular portion of the processus vaginalis, which is obliterated at its upper (peritoneal) end.

Encysted Hydrocele of the Cord is a collection of fluid in an unobliterated part of the funicular portion of the processus vaginalis as shown in Fig. 182, 2.

Encysted Hydrocele of the Epididymis is a cyst in the globus major of the epididymis, which projects into the tunica vaginalis, as shown in Fig. 183.

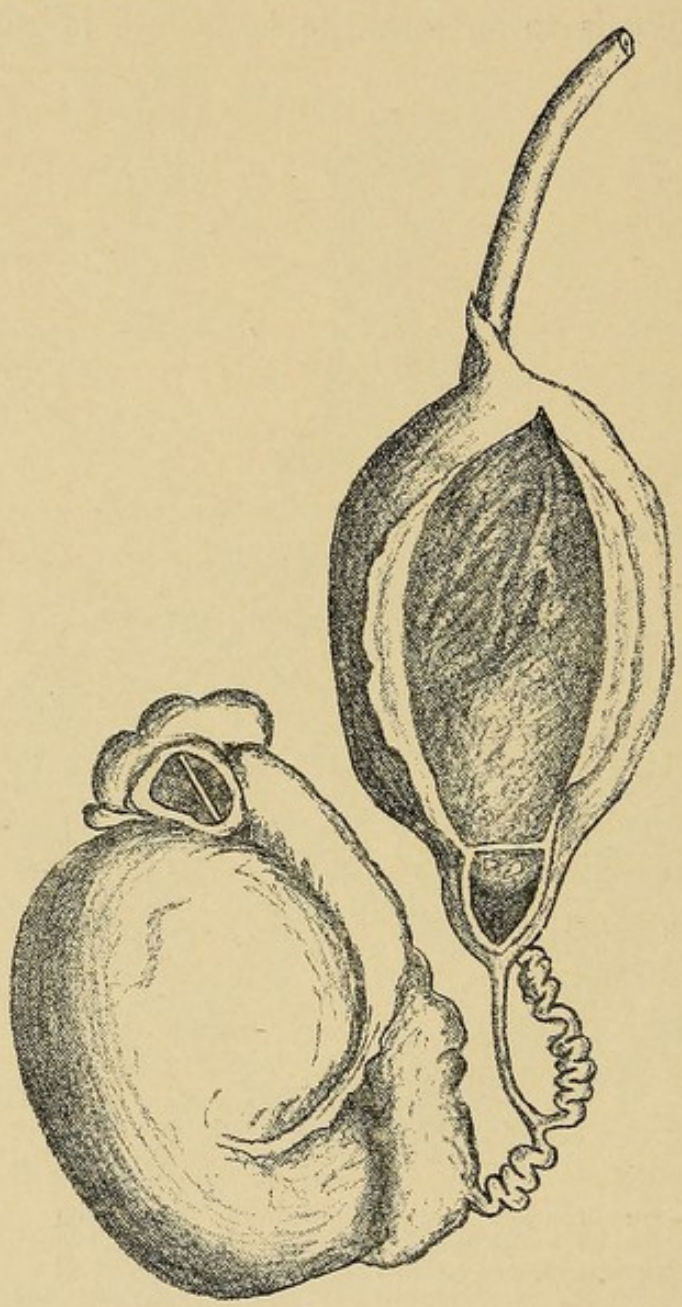


FIG. 184.—A hydrocele of the epididymis consisting of a dilated vas aberrans. From a middle-aged man who died of phthisis. The cyst had never caused any inconvenience, and the patient thought he had three testicles. In the original specimen there is a varicocele, but this has been omitted for the sake of clearness. The cyst contained spermatozoa. (1288.)

In two examples of this condition the author found the fluid of the cyst to be milky from the presence of spermatozoa. Hydroceles which contain spermatozoa are sometimes termed "spermatoceles." Such cysts sometimes arise in some of the remains of the Wolffian body (see Fig. 182).

Hydrocele of the Epididymis, not encysted. An example of this condition is shown in Fig. 183. The dissection of this specimen revealed the fact that the cyst was a dilated vas aberrans. The veins of the spermatic cord were varicose (*varicocele*). They are omitted in the figure. The fluid of the cyst was full of well preserved spermatozoa; see *Trans. Path. Soc.*, 1894.

Occasionally the hydatid of Morgagni becomes cystic, and may become detached, lying free in the cavity of the tunica vaginalis.

Encysted Hydrocele of the Testis is a cyst either between the tunica vaginalis and the tunica albuginea or between the layers of the tunica albuginea.

Cystic Degeneration of the Testis has been observed in a few cases quite apart from new growths. Such cysts have been attributed to obliteration of a spermatic cord.

Hydrocele of a Hernial Sac. When the aperture of communication between a hernial sac and the peritoneum becomes closed by adhesions, fluid may collect in the sac, and to such a condition the term "hydrocele of a hernial sac" has been applied. Œdema limited to the spermatic cord such as might be produced by the rupture of an encysted hydrocele of the cord seems to have been observed, and to it the term "diffuse hydrocele of the cord" has been applied.

Inflammatory or Acute Hydrocele occurs when the tunica vaginalis becomes inflamed from extension of inflammation from the testis in acute orchitis. Similar inflammatory effusions occur frequently in tertiary syphilitic inflammation of the testis, and sometimes in tuberculosis of the testis.

Suppuration in the Tunica Vaginalis has been observed after penetrating wounds, and in pyæmia.

Hæmatocele.

Hæmatocele is an effusion of blood into the tunica vaginalis. It is generally the result of injury, and often supervenes upon hydrocele either from injury of a vein in tapping or from some other injury. In some cases it has been attributed to the sudden diminution of pressure on the vessels

of the tunica vaginalis, resulting from withdrawing the fluid of a hydrocele by tapping. If small in quantity the blood may be absorbed. The red corpuscles may break up, forming a dark syrupy fluid. Usually clotting occurs, and the peripheral part of the resulting clot becomes adherent to the tunica vaginalis and organized; the remainder undergoes degeneration,

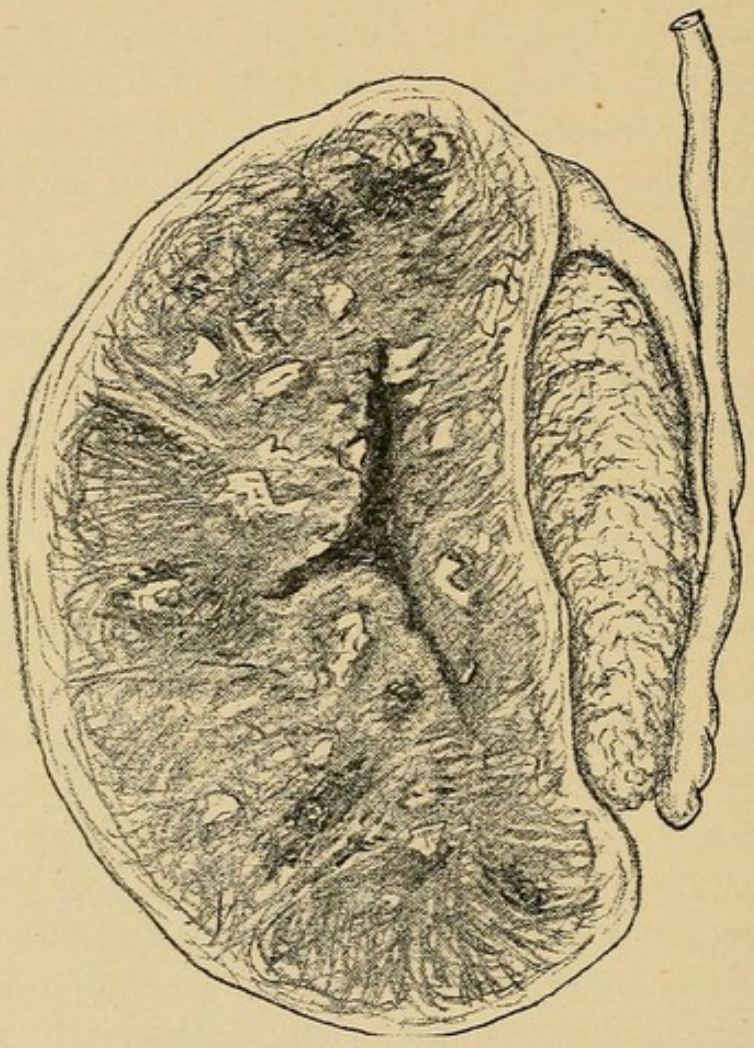


FIG. 185.—A section through an old hæmatocele and the testis. The contained clot has undergone organization of its outer part and centrally calcareous degeneration. Removed from a man on account of attacks of neuralgic pain. The swelling had been present for thirty-five years. The central cavity contained a brown liquid. The testis is compressed between the hæmatocele and the epididymis. (1285 A.)

sometimes calcareous (see Fig. 185). After the blood of a hæmatocele has clotted, the swelling is heavy and solid, and thus closely resembles some growths of the testis.

Injuries may cause effusion of blood beneath the tunica vaginalis constituting encysted hæmatocele. Similarly hæmatocele of the testis or of the cord may arise.

Varicocele.

A varicose condition of the spermatic veins is common in young subjects of lax fibre. The enlarged veins give to the hand the sensation described as being like a "bag of worms." It is more common on the left than on the right side; this is probably due to the slightly lower position of the left testis and to the fact that fæces in the sigmoid flexure press on the left spermatic vein. The testis affected by severe varicocele becomes rather softer and smaller than normal, but it is doubtful whether varicocele can render the testis sterile.

Inflammation of the Testis.

It is convenient to distinguish between inflammation limited to the body of the testis and that limited to the epididymis. The former is termed "orchitis," the latter "epididymitis." When the whole organ is involved the term "epididymo-orchitis" should be used.

Orchitis. Acute inflammation of the testis occurs in mumps and other specific fevers, *e.g.* typhoid, small-pox, scarlet fever, and influenza. Typhoid bacilli have been found in typhoid orchitis. It occurs also in malarial fever. In the latter condition the disease yields readily to quinine. Acute orchitis, owing to the unresisting character of the tunica vaginalis, is marked by great tension, and often by severe pain. Abscess is rare. The inflammatory process may rapidly clear up, but sometimes it becomes chronic. The chronic orchitis of syphilis will be separately considered. Orchitis not infrequently spreads to epididymis. Acute gouty orchitis occurs now and then.

Epididymitis. Acute epididymitis results almost solely from disease or injury of the prostatic urethra. Gonorrhœal inflammation, and operations which involve the prostatic urethra, are the commonest causes. Gouty and other forms of urethritis, prostatic calculi, strong urethral injections, stricture of the urethra, the use of a catheter, or the passage of gravel, have all been known to cause it.

Gonorrhœal epididymitis is now universally believed to be due to extension of inflammation along the ejaculatory duct, and

thence along the vas deferens, and this is also true of the majority of other forms of epididymitis. This is supported by the observation that the seminal vesicles at the base of the bladder can often be felt from the rectum to be swollen; and farther, that in some cases of epididymitis the inflammation is limited to the vas deferens and the cord. The older view of reflex nerve-influence, etc., inferred by the terms "sympathetic" and "metastatic," has not been supported by experience.

Symptoms. The vas deferens is swollen and tender; the epididymis swells from below upwards; effusion usually occurs into the tunica vaginalis (*acute hydrocele*). There is redness and œdema of the scrotum. Acute epididymitis usually subsides without the formation of pus; in but a small percentage of cases does the inflammation spread to the body of the testis. In broken-down individuals abscess and sloughing of the epididymis or testis may occur. After an abscess has opened a sloughy portion of the testis may project through the tunica albuginea, constituting *hernia testis.*

Chronic Epididymitis may remain as a sequel of an acute attack, and, if bilateral, may cause sterility. Primary chronic epididymitis is almost always tubercular, but occasionally syphilitic epididymitis is seen.

Syphilitic Orchitis.

Diffuse Interstitial Orchitis occurs in the late secondary period of the disease, and affects the intertubular and fibrous parts of the organ, which slowly becomes swollen and hard; there is but little pain. When persons have died during the early period of the disease the organ has been found to be redder and firmer than normal, and the tubules somewhat matted together. Later the testicle may atrophy if the condition is not treated. Under proper treatment the infiltration rapidly clears up.

Gummatous Orchitis. This is an event of later occurrence than the diffuse form of syphilitic inflammation just described. Gummatous foci form in various parts of the body of the testis, and the tunica albuginea becomes irregularly thickened, giving the organ a nodular feel. If not checked the original foci fuse

together and form large caseous masses; at the same time the tunica vaginalis becomes thickened and effusion takes place into it; the combination of the enlarged testis with inflammatory hydrocele has been called syphilitic hydro-sarcocele. Adhesions frequently form in the tunica vaginalis (see Fig. 186).

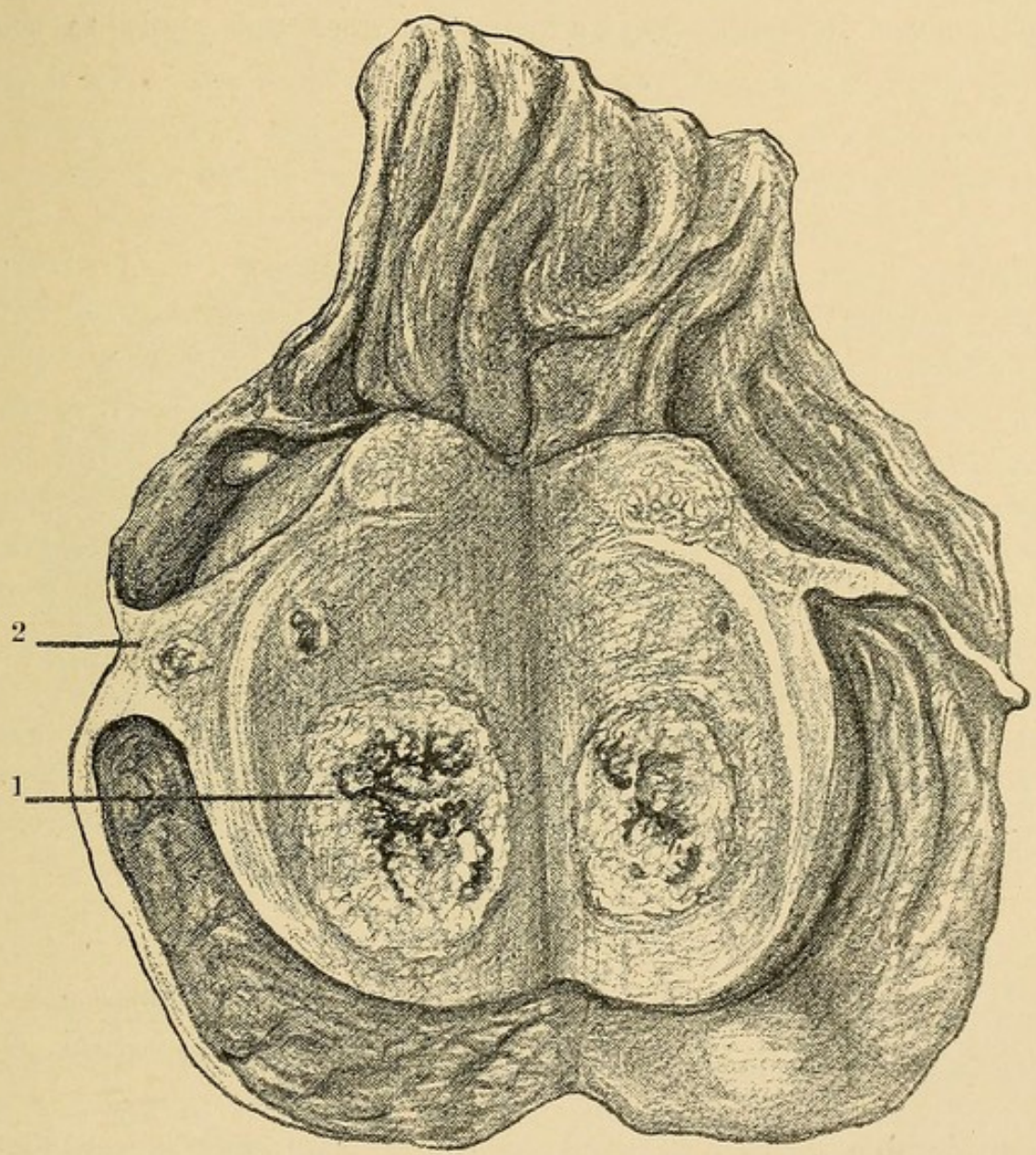


FIG. 186.—Gummatous orchitis with hydrocele. 1, Gumma; 2, adhesion in the tunica vaginalis, separating two loculi distended with fluid. The whole of the testis, epididymis, and tunica vaginalis are thickened and infiltrated. (1306.)

Both forms of syphilitic orchitis have been met with in congenital syphilis, but the condition is rare. In some instances epididymitis, independent of gonorrhœa, has been encountered in acquired syphilis.

Tuberculosis of the Testis.

Tubercular Epididymitis. Tubercle, when it affects the testis, nearly invariably begins in the epididymis. When the disease is secondary to tubercle of the kidney, prostate, etc., this is explained by supposing that the bacilli reach the organ by way of the vas deferens. This explanation does not apply to the

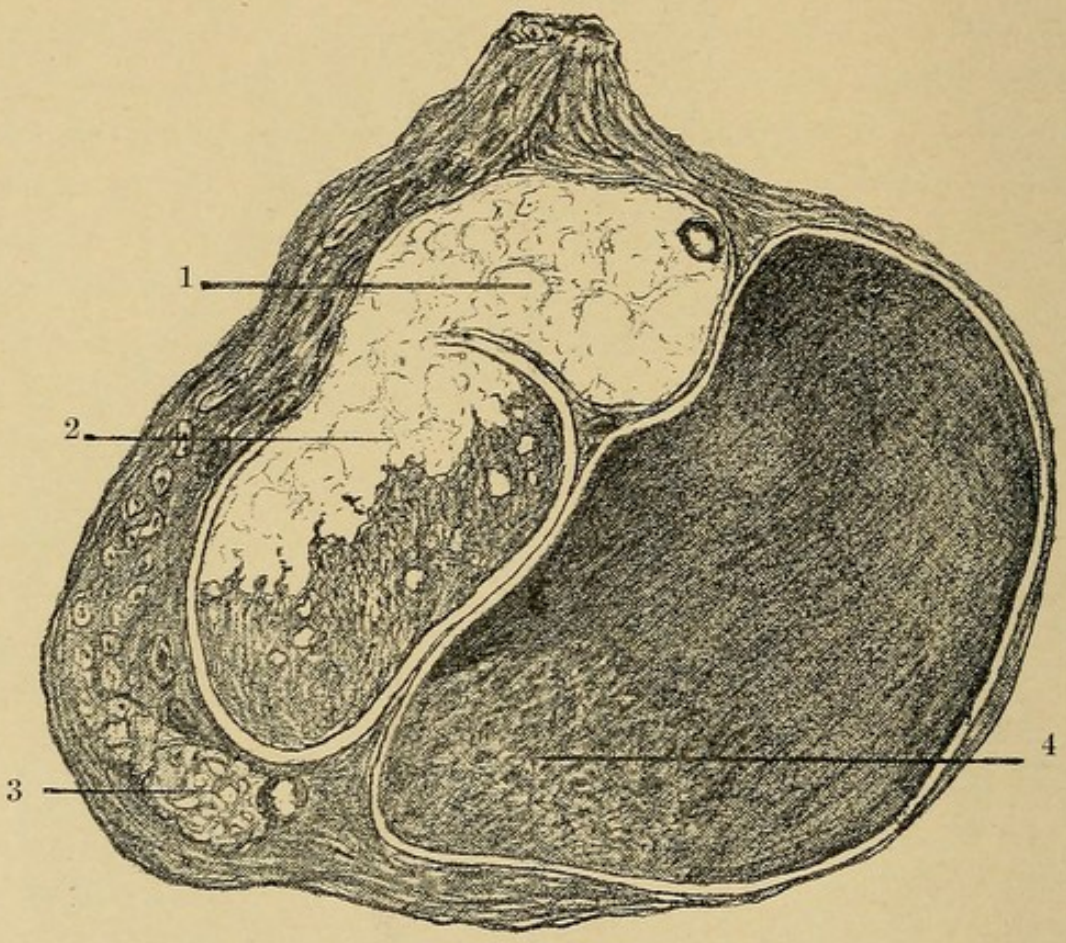


FIG. 187.—Section through a tubercular testis. 1, Epididymis containing caseous tubercular foci; 2, tubercles invading the body of the testis; 3, globus minor with commencing tubercular infiltration; 4, tunica vaginalis distended with fluid, part of which has undergone coagulation.

considerable number of cases in which the testis is the first organ to be affected, and hence the only way in which the bacilli can obtain access to the testis is by way of the spermatic artery. The disease shows itself first as a nodular swelling of the epididymis, which is hard and irregular to the touch. In some cases the inflammation rapidly involves the skin covering the epididymis, causes ulceration which allows of the escape of caseous matter. The vas deferens also becomes hard and

swollen. Sometimes the process spreads to the tunica vaginalis, causing hydrocele, as shown in Fig. 187.

When tubercle has appeared in one testicle it is likely to appear in the other also. The rapidity of the process varies as it does in tuberculosis of other organs. In a few cases it is very rapid, simulating gonorrhœal epididymitis; the organ rapidly softens with the formation of a large abscess. In other cases after the body of the organ has become infected the skin covering it becomes involved and breaks down with the escape of some tubercular pus. After this the testis substance tends to protrude through the opening of the skin, forming a "hernia." In this way the whole of the testis may protrude and slough away. In every case the seminal vesicles and prostate should be examined from the rectum to ascertain whether the disease has extended to them. Tuberculosis of the testis, both acute and chronic, has been observed in infants. The histological changes begin in the interstitial connective tissue and resemble those of tubercle elsewhere; the round-cell infiltration about the tubercles is very extensive. The duct of the epididymis is first affected by catarrhal inflammation; later the tubercles ulcerate through the walls of the duct, and tubercular granulations are seen in the cross sections of the duct. Similar changes are found in the vas deferens and in the tubules of the testis.

New Growths of the Testis.

The most important of these are sarcoma, cancer, and cystic adenoma. The two former more frequently arise in the body of the testis, the latter probably arises in the paradidymis (see Fig. 182, p. 388).

Innocent tumours arising either in the testis or its coverings are of less frequent occurrence. They include the dermoid tumours (see p. 131), chondroma and fibroma.

Sarcoma is of very frequent occurrence in children, and may even be of congenital origin. It is not uncommon in middle-aged men. There is a considerable variety of structure in different cases of sarcoma of the testis. Round-celled growths

predominate. These form soft rapidly growing tumours. Spindle-celled and mixed tumours are of slower growth and firmer consistence. The enlargement of the testis is as in gummatous infiltration usually painless. The cord sometimes becomes infiltrated, and not infrequently the lumbar and iliac glands. When the growth invades the tunica vaginalis hæmorrhage may lead to the formation of a hæmatocele which in some cases obscures the diagnosis for a time.

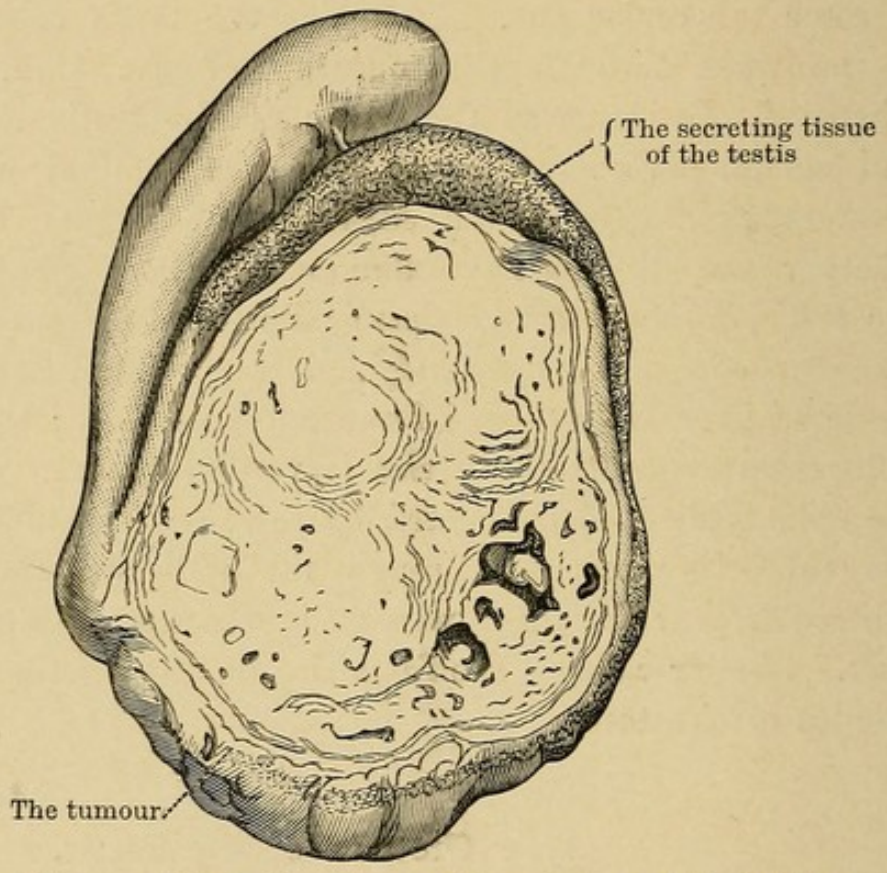


FIG. 188.—Adenoma of the testis. The secreting substance of the testis is stretched over the growth. Bland Sutton's *Tumours*. (1317.)

Cancer, like sarcoma, is usually soft and of rapid growth, but occasionally the firmer variety occurs. The glands frequently become involved, but since the same occurs in sarcoma it is often impossible to distinguish between the two by clinical examination. On section the alveolar structure distinguishes them from all except the alveolar sarcomata. In the latter alveoli are less definite in character than in the cancers. The soft translucent white of the more recent and the opaque white of the degenerated parts of cancerous growths are more marked

and less obscured by hæmorrhages, etc., than is the case in sarcomata.

Adenoma or Cystic Disease of the Testis. This neoplasm, according to the researches of Eve and Bland Sutton, arises, as stated above, in the paradidymis, and, in the course of growth, which is slow and painless, they push aside the body of the testis, flattening it, as shown in Fig. 187.

The cysts of these growths are lined with epithelium; the intercystic tissue varies in character. Very frequently it is fibrous and studded with islands of cartilage; in other cases it is myxomatous or sarcomatous. The rate of growth and danger of secondary tumours varies according to the character of the intercystic tissue. Metastasis occurs, however, in those of a fibro-chondromatous character, but not so frequently as in those of a sarcomatous character. They may appear at any age between birth and old age.

Fibromata have been noted arising in the tunica vaginalis, and since some of them have assumed sarcomatous characters their early removal is indicated. Chondroma, osteoma, and lipomata have been met with in connection with the testis.

New Growths of the Spermatic Cord are not infrequently met with beginning as oval elastic swellings lying in front of the cord. They may be either innocent or malignant.

CHAPTER XXVII.

DISEASES OF THE FEMALE GENERATIVE ORGANS.

Affections of the Vulva and Vagina.

Vulvitis. Severe suppuration of the vulva, though most frequently due to gonorrhœa, occurs also in weakly children from want of cleanliness. The presence or absence of gonococci should be ascertained.

Labial Hernia, corresponding to scrotal hernia in the male, is occasionally met with.

Hypertrophy of the Labia Majora. The most marked forms are seen in elephantiasis, but chronic eczema and chronic irritations from vaginal discharge will produce similar changes.

Rupture of the Perineum. During the passage of the fœtal head through the ostium vaginæ of primiparæ a rent always forms in the hymen, and this is most frequent at the mid-posterior line. This rent may extend to a variable depth into the perineum, which may be torn through into the rectum. In such a case, if an immediate operation is not successful, a carefully-planned plastic operation is required later.

Papilloma of the Labia Majora are commonly gonorrhœal. They may form large cauliflower-like excrescences.

Cancer of the Labia Majora. Squamous-celled cancer, beginning as a rule on the inner aspects of the labia majora, is not an uncommon occurrence.

The Vulvo-Vaginal, or **Bartholin's Glands,** correspond to Cowper's glands in the male, and lie at the sides of the lower end of the vagina towards the posterior aspect. They are internal to the sphincter vaginæ, and their ducts open about the

middle of the ostium vaginæ. They are subject to *abscess formation* in gonorrhœa, and also to *cystic distension*. The great vascularity of the part renders it advisable to use the thermo-cautery in operating on these glands.

Varix of the vulva and the erectile tissue of the bulb of the vagina sometimes forms rapidly during pregnancy, and may prove fatal from rupture and hæmorrhage.

Hæmatoma of the vulva is due either to parturition or traumatism. It arises from rupture of the veins of the bulb and forms a tumour which may be as large as an orange.

Urethral Caruncle. A small vascular growth, consisting of dilated capillaries and connective tissue covered by epithelium, which shows itself at the meatal orifice of the urethra. It is exquisitely tender, causing great pain on micturition, and very vascular, so that it should be removed by the cautery. On palpation it is softer than a carcinomatous growth.

Urethritis is part of gonorrhœa (see p. 70). It seldom leads to the formation of stricture in the female.

Prolapsus Uteri, as has been pointed out by Hart and Barbour, may be counted as a hernia. The pelvic peritoneum is displaced downwards, allowing of the eversion and protrusion from the vulva, first, of the anterior vaginal wall; second, of the cervix uteri; finally, of the posterior vaginal wall. The parts are easily replaced, and as easily returned unless the displacement is prevented by pressure, or the vagina is narrowed by a plastic operation, or the uterus fixed from above by stitching it to the abdominal wall. The bladder and the ureters are displaced downwards with the anterior vaginal wall, and pressure on the ureters against the pubis may cause retention of urine and uræmia. The cervix of the uterus becomes elongated in prolapse.

Cystocele is a bulging of the posterior wall of the bladder into the anterior vaginal wall.

Rectocele is a bulging of the anterior wall of the rectum into the vagina.

Affections of the Uterus.

Congenital Hypertrophy of the Cervix. The vaginal portion of the cervix is increased in length; the tissues of which it is

composed are normal. The elongation may be so great that the cervix may protrude into the vulva and simulate a uterine polypus. The cervix may become thickened from secondary inflammatory changes. Sterility results from the deformity, which is easily remedied by operation.

Laceration of the Cervix, to a greater or less extent, is caused by the passage of the fœtal head. It is important as forming a possible starting-point of pelvic cellulitis, and is a common factor in the production of the more chronic inflammatory conditions that give rise to subinvolution, *i.e.* failure of the uterus to return to its normal size after labour.

Septic Inflammation of the Uterus (*endometritis*) may follow labour, giving rise to pelvic peritonitis, etc., the so-called *puerperal fever*. In 1847, Semmelweis recognized the infective character of the disease, and recommended chlorine water as an antiseptic. Thus antiseptic midwifery foreshadowed antiseptic surgery.

Adenoma of the Cervix. A common condition, also known as "cervical endometritis," is really due to an overgrowth of the racemose glands which are abundant in the mucous membrane of the cervix, the epithelial lining of which consists of a single layer of columnar cells. The condition is marked by "chronic cervical catarrh," and on examination a red granular patch or a continuous red surface is seen at the os uteri and the neighbouring part of the vaginal cervix. On section, such patches show a proliferation of the glands, *i.e.* they have the characters of diffuse sessile adenomata.

Pedunculated Adenomata form small polypi which are frequently cystic. They may occasion slight hæmorrhages. They are common at middle age.

Compound Pedunculated Adenomata, or racemose adenomata, form tumours which may simulate a hydatidiform mole.

Cancer of the Cervix is of frequent occurrence. The growth most commonly makes its appearance in the lower part of the canal. Histologically it resembles the mucous membrane of the cervix. The infiltration is usually rapid and more frequently takes a horizontal course, invading the cervix and surrounding tissue, infiltrating the bladder, and often blocking the ureters.

It sometimes attacks the rectum, but this happens at a later period than invasion of the bladder. Now and again the growth creeps up into the body of the uterus. Dissemination by the peritoneum, liver, and lungs is not uncommon. When ulceration takes place hæmorrhage and fœtid discharge ensue. In slow-growing cancers of the cervix there may be but few symptoms, and the condition may be discovered accidentally after death; or the occurrence of hæmorrhage from the bladder, or spontaneous fracture of a bone the seat of a secondary deposit, may first call attention to the disease.

Adenoma of the Body of the Uterus is not so uncommon as has been supposed. Many uterine polypi are of this nature, and in examining the uterus after death I have frequently found flat sessile patches similar to the sessile adenoma of the cervix.

Uterine Polypi. Polypus is a vague anatomical term which simply signifies a pedunculated mass. Adenomata of both body and cervix, as mentioned above, may constitute polypi. The larger *fleshy polypi* of the body of the uterus consist of lax and œdematous mucous membrane covered by epithelium. The mucous membrane in these polypi frequently contains small myomata, or a single submucous myoma whether of body or cervix may become pedunculated and so constitute a polyp. *Fleshy moles*, the result of the formation of adherent clot on the placental site after abortion or child-birth, may also form polypi.

Cancer of the Body of the Uterus is not so common as that of the cervix. It forms a large mass of growth which projects into the uterine cavity. There may be considerable enlargement of the uterus. The recognition of a cancer of the body is not so easy as that of the cervix, hence cases are often overlooked until extension into the peritoneal cavity, etc., has occurred.

Myomata.

Tumours composed of unstriped muscle are very common in the uterus. They also occur in the broad, the ovarian, and the round ligament. On section they present, as a rule, to

the naked eye a firm whitish surface, because they contain few blood-vessels. Now and again, however, they are very vascular (*telangiectatic or cavernous myoma*). They are readily shelled out when they are surrounded by normal uterine muscle. The veins in the muscle adjoining the tumours are enlarged. Both subperitoneal and submucous uterine fibroids are apt to become pedunculated. Under the microscope the growths are found to consist of fasciculi of slender fibres. The fasciculi interlace in all directions, so that in sections some are cut transversely and may simulate collections of small round cells. The muscular fasciculi are supported by more or less fibrous tissue in which the vessels run.

Secondary Changes in Myomata. Calcification. Uterine myomata not infrequently undergo calcification. This change may affect the whole or only part of a myoma. If the calcified mass is small no additional symptoms are caused, but if large it may by its increased weight cause pain, and by its hardness cause injury to the intestine.

Cystic Change. Myxomatous metaplasia is not uncommon in myomata of the uterus, and results in the formation of cystic spaces in the tumour.

Suppuration. A submucous fibroid that projects into the uterine cavity, or still more into the vagina, may by ulceration of the mucous membrane become exposed to septic infection. The result of this is swelling of the tumour and fever. Sections show inflammatory infiltration, and, later, formation of granulation tissue. This can usually be distinguished from sarcoma by tracing the formation of cicatricial tissue.

Sarcomatous Metaplasia is not a very infrequent occurrence. A small myoma which has given rise to no symptoms may be found to have been the starting-point of a large sarcoma of the uterus. In such a case I have been able to trace the continuity of a myoma with a large myxosarcoma, which caused death, and which nearly filled the abdominal cavity.

The rate of growth of myomata varies greatly. In persons past the climacteric it is slower than in those in whom the menstrual function still continues. In pregnancy the growth is very rapid. The chief danger from larger myomata is their becoming impacted,

as it is termed, in the pelvis; that is to say, their filling the pelvis so tightly that they press upon the rectum, etc.

Diseases of the Fallopian Tubes.

These are similar to those of the uterus but are less common. The most important are *hydrosalpynx*, or distension of a tube with clear fluid; *pyosalpynx*, or distension of a tube with pus, as in some cases of gonorrhœa; *tubal gestation, and tuberculosis.*

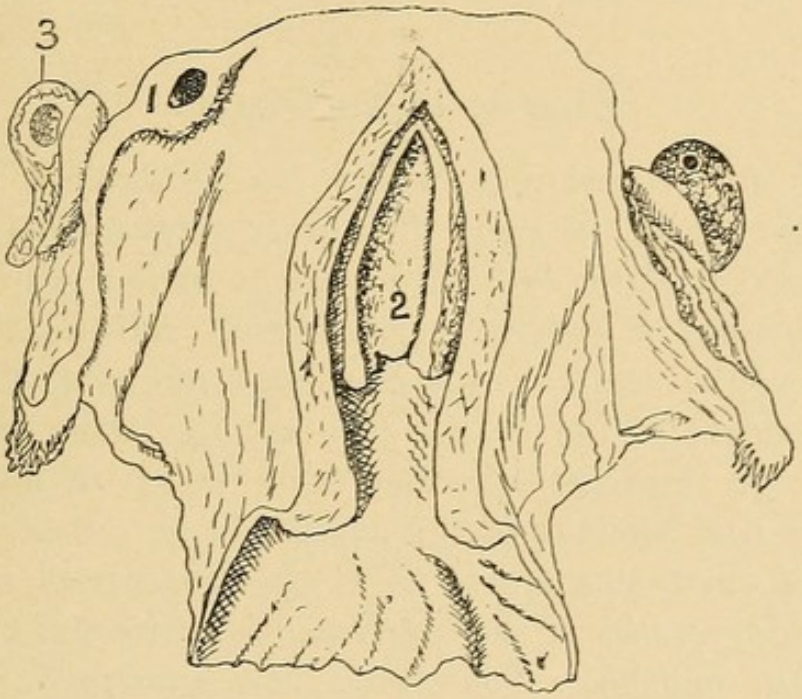


Fig. 189.—Ruptured tubal pregnancy. 1, A small dilatation in the right tube with a perforation from which the ovum escaped and fatal hæmorrhage occurred; 2, decidua in the body of the uterus; 3, the right ovary with a corpus luteum. From Jackson Clarke's *Post-mortems.* (1422.)

Tubal Gestation is a condition which may force itself painfully on the notice of any medical man. If the gestation goes on to the later months the condition will be recognized by the usual signs of pregnancy, and the discussion of the subject belongs to midwifery. It is when rupture of the tube and hæmorrhage occurs in the earlier months that the condition is apt to be misunderstood. Several typical examples have come to my notice. Such is the following: "A servant-girl, unmarried, and who denied the possibility of her being pregnant, was seized with sudden pain and became collapsed. She had had amenorrhœa for three months, and then a vaginal hæmorrhage which she took to

be a 'period.' The hymen was intact. When first seen she was already moribund. A lump could be felt in the recto-vaginal pouch. Transfusion was done, but she did not rally sufficiently to admit of operation." After death I found a rupture of the right tube in which was an ovum of about two months. There were three pints of blood in the peritoneum. An example of an earlier rupture (about the first month) is shown in Fig. 189.

Such cases demand immediate recognition and removal of the affected tube and broad ligament if life is to be saved.

Diseases of the Ovaries.

For a full description works on Gynæcology must be referred to, but at the same time every surgeon should have a fair knowledge of the morbid conditions of the ovaries. Thus an ovary may be present in a hernia. Adhesions resulting from chronic or acute ovaritis may cause intestinal obstruction. Cystic tumours of the ovaries may fill the abdomen and be mistaken for renal, hepatic, and other cysts. Here only the commoner new growths of the ovary will be considered.

Ovarian Cystomata are to be distinguished from *simple cysts* and *cystic degeneration* of the ovaries, which arise from distension of Graafian follicles. The cystic new growths are of two kinds: (1) the simple cystadenoma, and (2) the papilliferous cystadenoma.

Simple Cystadenomata form smooth lobulated growths composed of a stroma of connective tissue, which supports and separates the multiple cysts filled with mucus secreted by the lining epithelium, which in some parts of the growth is thrown into gland-like folds. These growths arise in the body of the gland. Such growths not infrequently attain very large dimensions.

Papilliferous Cystadenoma differs from the foregoing in that the cysts are filled by papillary growths which may perforate the surface of the growth and spread widely over the peritoneum, thus having a local malignancy or infective property. These growths arise at the hilus of the ovary, probably in some remains of the Wolffian bodies.

Solid Growths of the Ovary. Fibromata, fibro-myomata, sarcoma, and cancer are all met with from time to time in the ovaries.

Dermoid Tumours of the ovary have already been referred to (see p. 131).

Cysts and Myomatous Growths are also met with in the broad ligaments, the former arising from dilatation of the tubules of the parovarium, the latter from the unstriped muscle which the broad ligaments contain.

CHAPTER XXVIII.

DISEASES OF THE BREAST.

It will be found helpful in acquiring a knowledge of diseases of the breast to remember the more salient anatomical and physiological variations to which the organ is subject. Developmentally a collection of modified sweat-glands, anatomically the mamma is characterized as the gland that contains relatively more fibrous tissue than any other. The epithelial part of the gland is subject to wide variations. In the resting state, as in the virgin, after the climacteric, and in the intervals between the periods of lactation, the epithelial cells are scarcely larger than small cell-nuclei, and the gland-acini have scarcely any lumen. During lactation the cells and acini enlarge; active cell-formation occurs in the production of colostrom and milk. The three periods of life at which active changes occur in the breasts are: immediately after birth, when in both sexes there is sometimes a secretion of milk; at puberty when development of the breast occurs to a marked extent in the female, and also to a slight extent in the male; during pregnancy and lactation when there is great activity of the epithelial, and of the vascular and connective-tissue elements. This sympathy of the mamma with the generative organs is also shown to some extent in cases of disease of the ovaries.

Acute Mastitis is most commonly observed during lactation. The inflammation usually begins at the nipple in a crack or excoriation formed during suckling. It occurs also in newborn infants, especially if the breasts are injured. Sometimes the inflammatory process is limited to the nipple and areola,

which become œdematous and tender. The swelling of the areola may cause blocking of some of the milk ducts and consequent distension of one or more of the lobules, which then become knotty and tender—a condition which must be distinguished from abscess, though it may lead to abscess-formation by micro-organisms gaining access to the pent-up milk. A lymphangitis starting from a crack in the nipple may lead to abscess-formation in other parts of the gland. If the abscess forms in the subcutaneous fat which covers the gland it is termed a *supramammary abscess*, which gives rise to a superficial softening and fluctuation; if the lymphangitis spreads along one of the ducts to a lobule of the gland an *intramammary abscess* results, and shows itself by redness and tension over the corresponding area of the breast. Fluctuation is later in its appearance than is the case in a supramammary abscess. An intramammary abscess should be opened before fluctuation has become evident, and before the suppuration has spread to neighbouring lobules. In a few cases the pus forms in the deepest part of the gland, and the abscess is formed between the gland and the pectoral fascia; this constitutes an *inframammary abscess*, which shows itself by a general prominence of the affected breast, and may burrow widely before it points at the lower margin of the breast. The axillary glands may be inflamed.

Chronic Abscess. An acute suppuration in the breast may quiet down, the pus becoming surrounded by a thick wall of granulation tissue. If such a *chronic abscess* is situated at some depth in the breast it may give to the hand a sensation like that observed in solid growths. In some cases of this kind the breast has been removed on mistaken diagnosis. Hence the rule that before removing the breast for a growth an incision should be made to ascertain definitely whether the condition is really one of growth or a deep-seated cyst or chronic abscess.

Chronic Interstitial Mastitis. In elderly women who have passed the climacteric, one or more lobules of the breast become thickened and nodulated. If such areas are examined by the microscope it is found that the interstitial tissue is increased by the formation of new fibro-cellular tissue, and the ducts and acini are distended into small cysts with degeneration of the

epithelium. The condition is to be looked upon with suspicion, since in some cases it proves to be the commencement of cancer.

Tuberculosis of the Breast is a rare affection, which is marked by a nodular infiltration of one or more lobules. Caseation and abscess may result. Histologically typical tubercles in the interstitial tissue, ulcerating into the ducts and acini, have been found.

Galactocele. Some effects of blocking of milk-ducts during lactation are over-distension of the lobule with milk, and sometimes abscess. Another is the formation of a cystic cavity filled with milk. This is termed a galactocele. It is supposed to be due to rupture of a milk duct. Sometimes the milk is absorbed, at others it becomes inspissated and cheesy, and then may give rise to an abscess.

Hypertrophy of the Breast is an exaggeration of the general enlargement of the breasts observed in puberty. The enlargement may be accompanied by neuralgic pain. Though the condition may begin in one gland before the other it is usually symmetrical. The enlarged glands may be either hard or soft. Treatment by belladonna, etc., has, as a rule, but little effect. In extreme cases amputation may be required, but before removing the breast care should be taken to incise the gland to make sure that there is not placed behind the breast an adenoma or cyst, which has caused a simulation of hypertrophy by pushing the breast evenly forwards.

New Growths of the Breast.

Great variety is observed in tumours of the breast. The epithelial growths, adenoma and cancer, are the commonest, but sarcoma is by no means rare, whether occurring as pure sarcoma or forming the stroma of an adenoma, and thus constituting an adeno-sarcoma.

Adenoma of the Breast is a gland-like growth rather soft in consistence, yellowish in colour, and provided with a fibrous capsule. Histologically it resembles breast-tissue with certain differences; thus the connective tissue immediately beneath the epithelium is relaxed and there is a tendency to cyst-formation;

the epithelium is often arranged in a double or treble instead of a single layer.

Fibro-Adenoma. When the fibrous tissue of an adenoma is developed so that the fibrous preponderates over the glandular portion of the growth, the latter is firmer and whiter than the form of adenoma just described. The fibro-adenoma is one of the commonest of mammary tumours, and is usually met with in women between the age of twenty and thirty. It is freely movable and readily shells out (see Figs. 190 and 191).

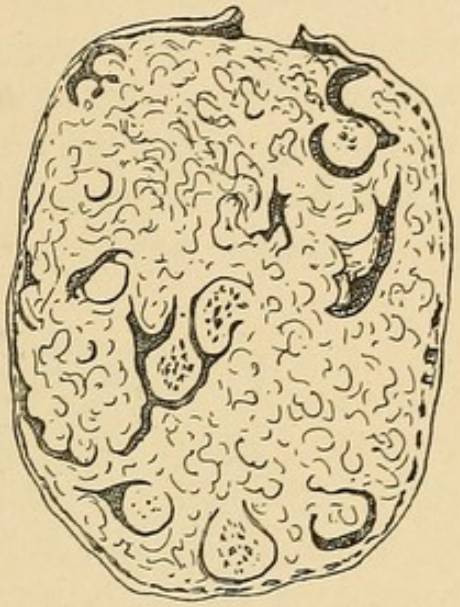


FIG. 190.—Section through a fibro-adenoma of the breast. There are numerous cystic clefts, and the tumour has a well-marked capsule. (1459.)

Other Forms of Adenoma. Together with fibrous tissue in mammary adenomata mucous tissue and cartilage are frequently combined. Thus fibro-myxo-adenomata, fibro-chondro-adenomata, etc., are constituted. They are similar in their general tendencies and course to the fibro-adenomata.

Duct-Papilloma. Closely allied to the adenoma is the duct-papilloma. Such growths sometimes arise in the ducts close to or within the nipple. In such cases they may ulcerate through the skin and form a pedunculated papilloma attached to the nipple.

Breasts containing these duct-papillomata often show that besides the tumours there is a change in the tissue of the whole gland. The microscope shows that not only the ducts but also the acini are dilated, and the epithelium undergoing proliferation.

It has been indicated already that in all adenomata there is a tendency to cyst-formation. In some cases they form a marked feature in the tumour. The contents of the cysts vary. In some cases they are filled with clear, in others with fatty, fluid. Where the blood-vessels of the intracystic projections are highly developed there is hæmorrhage into the cysts, and if the milk-ducts are not blocked the blood may escape at the nipple.

Cystic Degeneration of the Breast. This condition is chiefly met with in women soon after the menopause. Cysts develop in various parts of the breast, sometimes so rapidly that when the cyst is deeply placed and the nodular breast-tissue is stretched over it, it is easily mistaken for scirrhus. Sometimes on pressing such breasts a dirty cream-like substance escapes at the nipple. On incising any part of the breast that may appear to be unaffected by the cystic change it will be found that many small cysts are present. If in removing such a breast a portion be left in the body it has been found that cysts continue to develop therein.

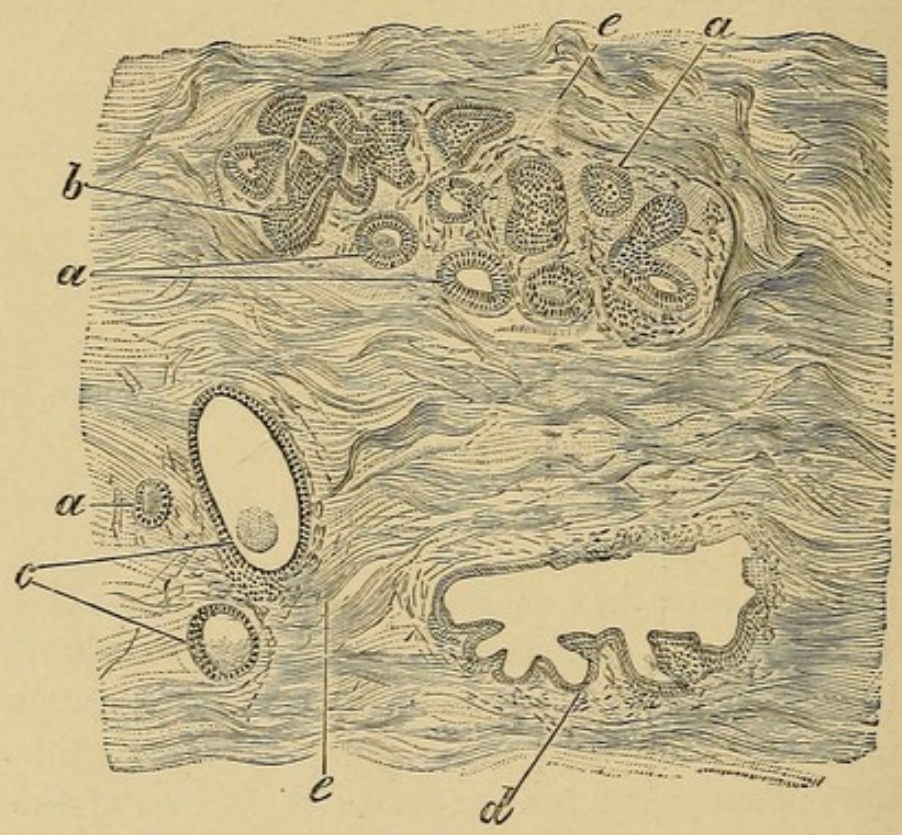


FIG. 191.—Cysto-adeno-fibroma of the mamma (acinous form). *a*, Acini, partly empty; *b*, branched tubule; *c*, smaller and larger cysts; *d*, cysts with papillary elevations of the walls; *e*, connective tissue. × 77. (Stained with alum cochineal.) Weichselbaum, *Pathological Histology*.

Adeno-Sarcoma of the Breast. These growths have a cellular instead of a fibrous stroma. The adenomatous ducts and acini are supported by round or spindle-cells. The tendency to cyst-formation is the same as in other adenomata, and most of the tumours which used to be termed cysto-sarcoma were of this kind. Masses of growth may project into the cysts in a manner similar to the intracystic projections of duct-papilloma. Indeed

the group of cases sometimes termed "proliferous cysts" of the breast included both adenomata and adeno-sarcomata. Clinically the adeno-sarcomata are marked by their more rapid growth and softer consistence as compared with the adenomata. They do not cause glandular infection, and but rarely do they invade the tissues outside the breast or cause secondary growths. Sometimes they grow to a very large size. They may cause ulceration of the skin, and then some of the growth projects through the aperture as a red mass, which bleeds readily. On section these growths have a duller appearance than the adenomata. Like sarcomata they form lobulated tumours.

Cancer of the Breast.

Cancer of the breast presents four well defined types: the hard or scirrhus (Gr. *skiros* = lump of stucco), the soft ("medullary" or "encephaloid"), the duct or villous, and the colloid varieties. Though intermediate or mixed forms are met with it is important to remember these anatomical phases of cancer, because they are associated with distinctive clinical features. Cancer commonly begins between the ages of forty and fifty, but it is not unknown as early as the thirtieth year. Though far more common in women it is met with now and then in men.

Hard Cancer is by far the most frequent. It first shows itself as a hard lump in the breast and is often discovered accidentally, there having been no pain to draw the patient's attention to it. As the growth extends it becomes irregular, and though at first the skin is freely movable over the tumour, the latter tends to spreads along the lymphatics to the corium, and then as the skin is moved from side to side over the tumour it is observed that it is held down at one or more points; this gives rise to *dimpling* of the skin. As the case progresses this subcutaneous attachment of the skin to the growth becomes more marked by cicatricial contraction taking place. Lancinating pain may be observed. What were dimples produced only when the skin was moved from side to side now become permanent depressions or puckerings. This process of shrinking is most likely to occur first in the skin

of the nipple by spreading of growth along the ducts. Thus *retraction* of the nipple is caused.

If the tumour is not removed the skin becomes infiltrated and the growth projects above the surface.

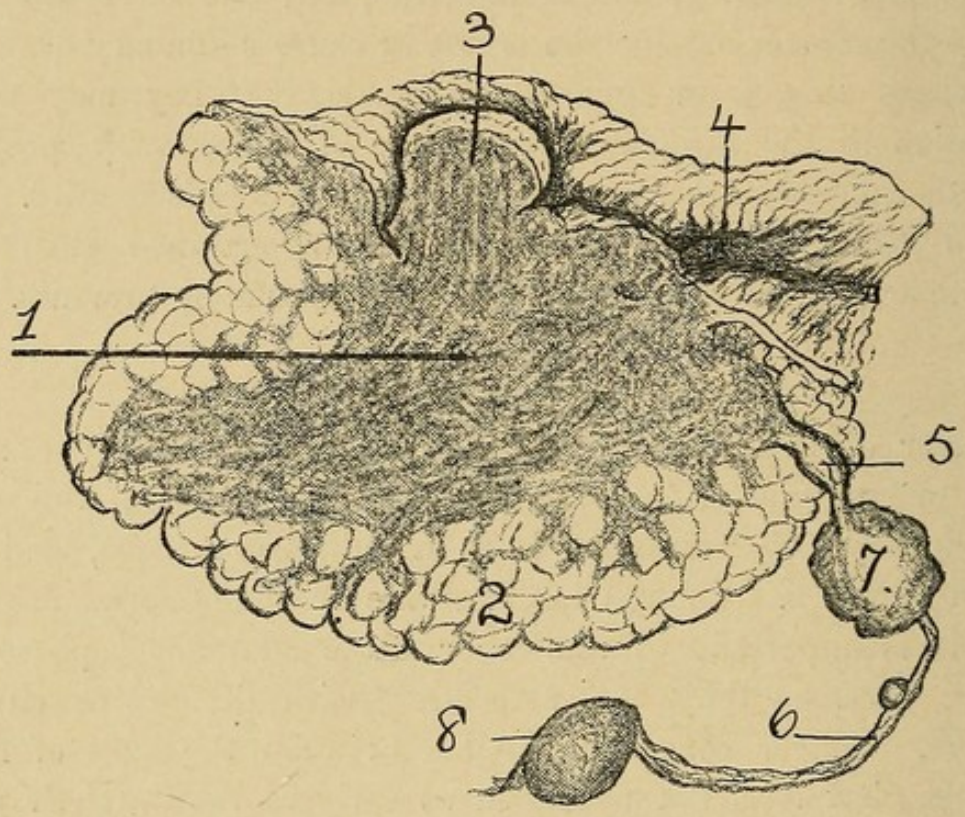


FIG. 192.—Section through a breast, the seat of hard cancer. The outline is that of the original gland. 1, Cancerous growth; 2, fat; 3, retracted nipple; 4, pucker in the skin; 5, 6, lymphatic vessels filled with cancerous growth; 7, one of the pectoral; 8, one of the axillary, set of glands. (1469.)

Later still the central part of the projecting mass undergoes necrosis, forming an ulcer which bleeds readily (*fungation*), and if not prevented becomes offensive by the development of putrefactive bacteria. While these changes are going on superficially exactly similar ones take place on the deep surface: first the pectoral fascia then the pectoral muscle become invaded by the growth, which may eventually reach the pleura, causing cancerous infection of that membrane and of the lung.

Generalization of the growth when it occurs is usually effected by way of the lymphatics, as in cancer generally. In many cases the axillary lymph-glands are the seat of secondary cancer. The pectoral set are usually the first to suffer, and later in the case the supra-clavicular glands may become involved. It is to be remembered that lymph passes from the inner part of the

breast to the anterior intercostal lymphatics, and these may be affected, though from their position they cannot be felt. Glands may appear to be normal in size and consistence, and be only slightly paler in colour than normal, yet on section they show firm white areas of cancerous infection. Sometimes (see Fig. 192) the lymphatic vessels passing from the breast to the glands are felt to be distended with firm cancerous matter.

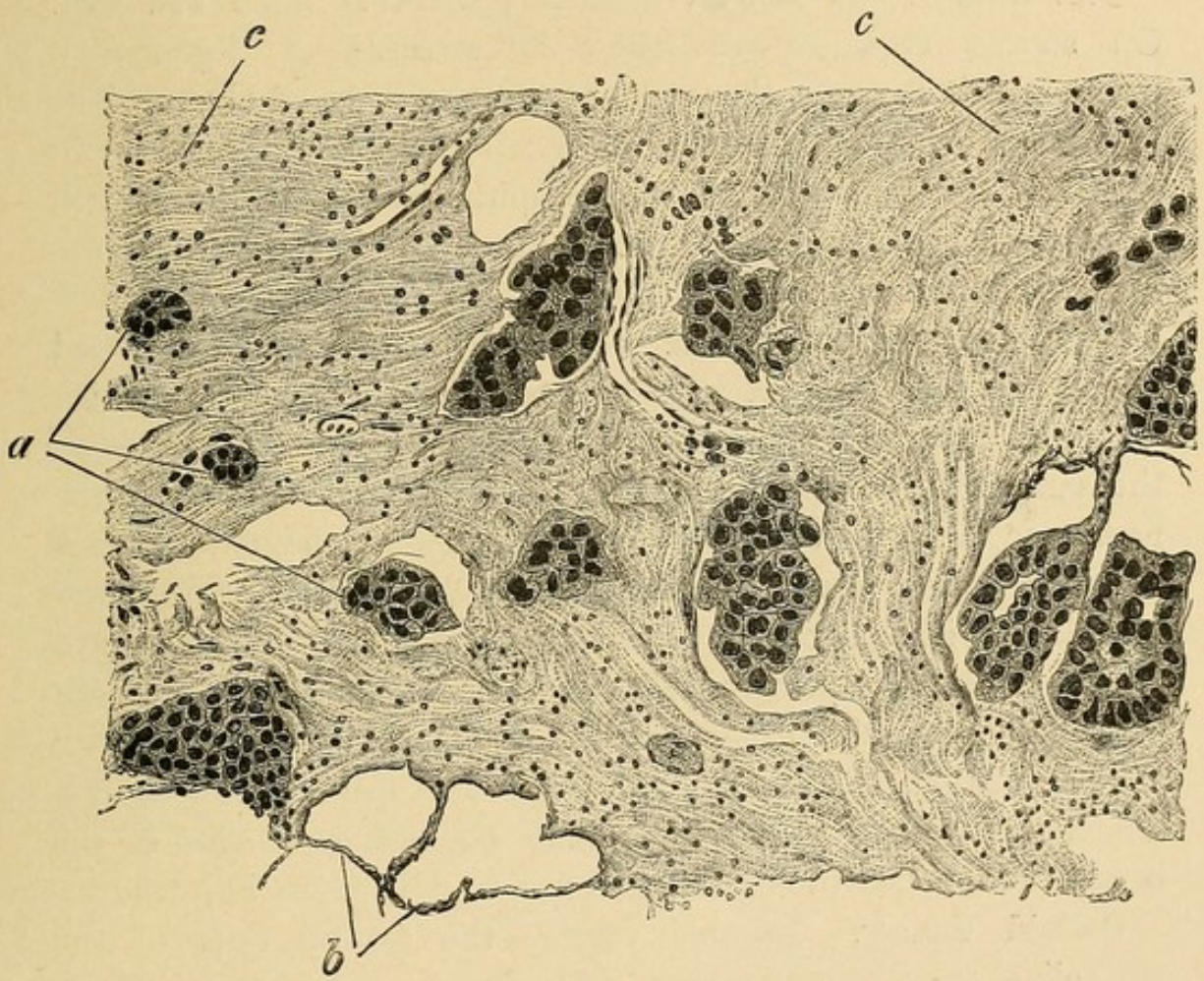


Fig. 193.—Scirrhus mammæ. *a*, Small cancerous alveoli; *b*, empty cancerous alveoli: *c*, greatly developed connective-tissue stroma, partly permeated by round cells. × 240. Weichselbaum, *Pathological Histology*.

On section, hard cancers of the breast present a surface which has been compared to that of an unripe pear. The grey translucent areas correspond to the collections of epithelial cells, while the paler meshes are the alveolar septa. In most hard cancers of the breast small cavities filled with creamy material resulting from fatty changes in dead cells can be found. In some cases the formation of these cavities is a marked feature; such tumours have been called "cystic scirrhus."

In many cases of cancer of the breast distinct nodules of growth are formed in various parts of the gland, and outlying nodules in the tissue beyond the gland.

Cuirass, or Hide-bound Cancer, is constituted by the skin becoming rapidly infected by the growth over a wide area. Sometimes the skin of a great part of the chest thus becomes the seat of a brawny induration, and assumes a purplish-red hue.

Soft Cancer. In many cases the growth is rapid and soft. On section such growths show large areas of degenerated tissue, which may be the seat of extravasations of blood. The microscope shows that the supporting fibrous tissue is in relatively small amount, whilst the epithelial part of the growth is composed of larger and more succulent cells than is the case with hard cancer.

Duct or Villous Cancer forms a rather soft lobulated tumour, which on section presents cystic clefts into which lobulated processes of the growth project. This growth bears the same relation to the ordinary forms of cancer that duct-papilloma bears to ordinary adenoma. The intracystic growths of duct cancer are covered by several layers of large and succulent epithelial cells. Hæmorrhage into the cysts is almost constant, and frequently leads to the discharge of a glairy pink fluid from the nipple. It is of importance to recognize the nature of this variety of growth since it shows less tendency to infect glands, etc., than the other cancers of the breast, and hence prognosis after removal is more hopeful.

Colloid Cancer. In this variety the central cells of the epithelial masses undergo colloid degeneration, so that on cutting into the tumour beads of a transparent jelly-like substance project from the cut surface. This form of growth is less rapid than the ordinary hard or the non-colloid soft growths.

The fact that cancer of the breast readily infects the axillary glands and extends to the fascia covering the pectoralis major renders it necessary in an average case to remove with the breast the pectoral fascia and the fat containing the lymphatic vessels and glands of the axilla.

Sarcoma of the Breast. Apart from adenoma-sarcoma, purely

connective tissue malignant growths occur not infrequently. The commonest form of sarcoma is the least malignant, *viz.* spindle-celled sarcoma. This growth was formerly termed "recurrent fibroid." Fibroid because, though it looks on section something like fibrous tissue, being fasciculated, it is less white and glistening. It is fibroid not fibrous. Recurrent because, after imperfect removal, it recurs locally.

Round-celled and other forms of sarcoma also occur.

Before the histological differences between cancer and sarcoma were well established, many cases of the softer kinds of sarcoma were doubtless classed as "Encephaloid cancer."

Cysts of the Breast. In a gland containing many acini and ducts, and subject to a wide range of physiological and pathological variation as is the breast, there is a wide range of cyst-formation. Cysts containing serous fluid are not infrequently observed, without the presence of any tumour-formation. Such cysts probably arise, as Bowlby has pointed out, in distended acini, and they may be compared to the simple cysts of organs like the kidney. In connection with "chronic interstitial mastitis," as has already been pointed out, small cysts sometimes form in elderly persons. In many cases such cysts form without there being any evident mastitis. Galactocele has been already described (p. 410), as also have cysts in mammary new growths of various kinds. In sarcomata cysts are usually due to degeneration or to hæmorrhage.

CHAPTER XXIX.

SURGICAL AFFECTIONS OF THE SKIN.

THE resistant part of the skin is the tough fibrous corium (*derma* or *cutis vera*) on which rests the stratified epithelium (*epidermis* or *cuticle*). The superficial or papillary layer of the corium contains the special nerve-endings and many capillary vessels. The appendages of the skin, the hair-follicles with the sebaceous glands that open into them (*pilo-sebaceous follicles*), and the sweat-glands offer a rich field for pathological processes. The nerves of common sensation break up into terminal filaments which pass between the cells of the deeper cell-layers of the epidermis (*rete Malpighii*), and in this close association of nerves with epithelial cells probably lies the explanation of many cutaneous affections.

In the corium, besides white and yellow connective-tissue fibres, nerves, blood-vessels, etc., are strands of unstriped muscle fibres connected with the hair-follicles.

The complexity of structure and function thus indicated would in itself account for a wide range of pathological phenomena, and the protective functions of the skin cause it to be exposed to injuries and infections of all kinds in a greater degree than any other structure of the body. Besides the pyococci and other organisms already mentioned in considering the pathology of infection, animal parasites, such as the pediculi, the itch-mite, moulds such as the fungi of ringworm and favus, may infect the skin. It is not within the scope of this work to discuss the pathology of the skin generally, but only such parts of it as apply directly to surgery. In speaking

of the cutaneous lesions of syphilis a variety of inflammatory lesions of the skin were mentioned, and it is necessary to grasp the fact that syphilis may imitate almost any skin disease.

Congenital Affections of the Skin.

Ichthyosis. The slightest degree of this affection is termed "xerodermia," and is marked by a dry scaly condition of the skin. The secretion of sebum and sweat is diminished. The extensor surfaces of the limbs are rough from the heaping up of horny epidermis at the mouths of the hair-follicles. In the more marked cases the horny layer of the epidermis is distinctly thickened. The skin in ichthyosis of all degrees is prone to attacks of eczema and the formation of "chaps" or fissures. The nails and hair are dry and brittle.

Ichthyosis Simplex. The scaliness of the skin and thickening of the epidermis is more marked than in xerodermia. The skin of the whole body is affected, but on the extensor surfaces of the limbs, especially about the elbows and knees, the thickening may be so great that the skin looks like that of a lizard or a crocodile.

In Ichthyosis Hystrix the skin generally resembles that in xerodermia or in ichthyosis simplex, but in parts of the skin of the limbs or trunk warty elevations form; they are generally arranged in longitudinal lines when they occur on the limbs, and in horizontal lines on the trunk.

Tylosis is a congenital thickening of the horny layer of the palms and soles; a similar condition is sometimes produced by the prolonged internal administration of arsenic, and by chronic eczema, psoriasis, syphilis, etc.

In order to be able to describe clearly such affections as come within the scope of this chapter it is necessary to define the elementary lesions of the skin. These lesions are called *primary* when they are the expression of a pathological process before or at the height of its evolution. They are called *secondary* when they result from the subsidence of the lesion. Thus a small-pox pustule is a primary, the scar it leaves is a secondary lesion.

Primary Lesions.

A macule is an area of skin altered in colour without marked elevation. Thus each element in a syphilitic roseola is a macule due to inflammation; a small capillary nævus is also a macule, though it is due to new growth and not to inflammation. In the same way there are pigmentary macules, *e.g.* freckles; macules from loss of pigment, as in leucodermia, and hæmorrhagic macules as in purpura.

A Papule is a solid elevation of the skin not larger than a pea. Syphilitic papules are inflammatory, being caused by exudation taking place into one or more papillæ of the corium. Other papules are non-inflammatory, *e.g.* the common comedones or "black heads" due to retained sebaceous secretion.

A Nodule is a solid elevation of the skin larger than a pea. The term tubercle used to be applied to such a lesion, but it is better to speak of small and large nodules now that tubercle applies to a specific disease.

A Wheal is a particular variety of papule (if small) or nodule (if large). Wheals are characteristic of nettle-rash (*urticaria*), and are due to changes in the superficial blood-vessels in an area of skin. They have a white centre (from œdema) and a red periphery due to hyperæmia.

Vesicles are hollow elevations of the skin not larger than a pea and filled with clear fluid, which may lie either between the layer of the epidermis, as in slight burns, or in the corium, as in lymphangioma.

Bulla is the same as a vesicle, only larger than a pea in size. In burns, pemphigus, and about fractures, for instance, bullæ are formed, also in moist gangrene. In the latter condition the bullæ differ from others in that they can be made to move from place to place by slight pressure. This shows that they are due to accumulation of fluid beneath an epidermis which has lost its attachment to the deeper tissues.

Pustules are hollow elevations containing pus instead of clear or opalescent fluid like vesicles and bullæ, which they otherwise resemble.

Secondary Lesions.

These may be of the same character as some of the primary lesions. Thus a pigment-macule may be primary, or it may be secondary to previous inflammation. The chief secondary lesions are as follows:

Scales or Squamæ, due to increased formation and separation of the superficial layers of the epidermis generally form after the subsidence of inflammatory lesions.

Excoriations are made by scratching, or remain after the rupture of vesicles, etc., which have not destroyed the whole thickness of the epidermis.

Ulcers are lesions caused by the destruction of the whole thickness of the epidermis by some inflammatory process. Thus any primary lesion may break down, leaving an ulcer.

Scabs or Crusts are due to the drying of liquid exudation on the surface of the skin. "They may be left after the cessation of hæmorrhage (*blood scab*), after the rupture of a vesicle (*serum scab*), or of a pustule (*pus scab*), or they may be formed of sebaceous matter, or be caused by a parasitic growth, as in favus. Scabs may consist of a commingling of these various dried exudations or growths."[1]

Thickening, or Hyperplasia, may result from long-lasting or frequently-repeated inflammation, *e.g.* chronic eczema or elephantiasis.

Scars are the result of the complete involution of granulation tissue formed in inflammation which has been of sufficient intensity to destroy part of the corium. Thus it may remain after almost any of the primary lesions.

Erythema and Eczema.

Under the designation of erythema come a great variety of affections of the skin, characterized at least at the beginning by hyperæmia of the corium. Some of these affections present only this feature, others are distinctly of inflammatory type.

[1] Malcolm Morris, *Diseases of Skin*, 1894, p. 13, to which work the reader is referred for further information on skin affections.

In some persons drinking a glass of beer will cause a dilatation of the capillaries of the face; in others various agents, such as the administration of an enema for constipation, will produce a redness of the whole body (*erythema scarlatiniforme*). This effect is doubtless due to the toxic effect on vaso-motor nerves of substances absorbed into the blood and so is akin to the *drug eruptions*, some of which are of especial importance.

Bromide Eruptions. The most characteristic and common is a pustular eruption—bromic acne. The pustules may be so large as to resemble boils or even carbuncles. Slighter degrees of reaction to bromides cause the appearance of patches of erythema, papules, or vesicles. By the rupture of pustules or vesicles scabs are formed. The lesions are commonest on the forehead, at the sides of the nose, and at the back of the shoulders, but they may appear on the trunk or elsewhere.

Iodic Eruptions. Any of the salts of iodine, even when given in moderate doses, may produce eruptions in certain susceptible individuals. The lesions generally appear as large or small erythematous patches scattered especially on the face, forearms, and chest. Papules, vesicles, and pustules may follow, and often closely resemble a small-pox eruption. Hæmorrhage may occur into the vesicles, or small hæmorrhages (*petechiæ*) may be present from the first. On the rupture of the vesicles or pustules scabs form, and on their separation scars may remain.

Copaiba and Cubebs cause various eruptions, the more common of which are the erythematous (resembling the rash of scarlet fever) or the papular, which is frequently mistaken for a syphilitic eruption.

Iodoform applied locally produces in some individuals an intense eczema-like eruption, the appearance of which is usually accompanied by some fever. Many other drugs, such as mercury and arsenic, may produce erythematous eruptions.

Antitoxin Eruption. The injection of diphtheria, tetanus, or other antitoxic serum is frequently followed by an erythematous eruption.

Erythema Multiforme. The study of the chief phases of erythema multiforme is of importance because of their close resemblance to syphilitic eruptions. The appearance of the

rash is accompanied by some fever. Patches of erythema, papules, vesicles, purpura, etc., may constitute the leading feature of the eruptions. By the peripheral spreading of lesions which clear up in the centre, gyrate and circinate figures may be formed. A bullous lesion spreading at its edge characterizes *erythema iris*. The dorsal aspects of the hand and foot are the favourite situations for its first appearance. The lesions tend to ascend the limbs, and sometimes spread over the trunk.

Erythema Nodosum appears in painful circumscribed swellings over the tibiæ. The forearms and thighs may also be affected. The condition is usually associated with rheumatism.

Purpura, or effusions of blood into the corium, is common to several affections in which there is anæmia. It is especially frequent in rheumatism.

Rheumatic Nodules are small firm formations which occur during the course of rheumatism in the subcutaneous tissue. They are of grave prognostic significance.

Acne Rosacea. A flush, like that which follows drinking a glass of beer in some persons, is almost constantly present in a few persons who are abstainers, especially in women, in whom it is worse at the menstrual periods. In those who habitually drink too much, the same flush may be constantly present. This hyperæmia or erythema leads to thickening of the skin of the nose and its neighbourhood. Also it causes blocking of the sebaceous ducts (necks of the hair-follicles), and consequent retention of sebum and sometimes pustulation, causing the affection to resemble acne vulgaris (see below). When the thickening of the skin is great it may form a lobulated growth resembling a lipoma, and has been misnamed *Lipoma nasi*. When the condition is pronounced, removal of the skin is justifiable, and the cosmetic effect of the operation is striking.

Lupus Erythematosus in its commonest form has a superficial resemblance to acne rosacea. That is when it takes the shape of a butterfly or bat's wing, appearing first as a patch of erythema on each cheek, the patches extending inwards, and meeting over the bridge of the nose. In other cases small

spots of erythema appear in different parts of the head and face, and even on the hands. The margin of the ears, the nape of the neck, the scalp, the lips, and the sides of the nose are all favourite sites for this the disseminated form of the disease. The margin of the patches is red, and slightly raised. The reddened skin is covered by a thin adherent crust which sends down processes (*comedones*) into dilated sebaceous orifices. In the central part of the lesions the corium is replaced by scar tissue. The microscope shows an inflammatory infiltration of the corium without any evidence of tubercle. The disease appears at or after puberty, and though there is scarring there is no ulceration, for the epidermis is not destroyed, only the corium is replaced by scar-tissue. The progress of the disease is very slow, and it may last twenty years before it ceases to extend. It leaves indelible scars, and on the scalp baldness. Sometimes the process is more rapid, so that it may closely resemble severe persistent erysipelas or some of the pyæmic skin eruptions. The chief differences between this affection and tubercular or common lupus are that the latter begins before, the former after, puberty. Lupus vulgaris tends to ulcerate. Lupus erythematosus never ulcerates.

Erythema Pernio, or Chilblain, is the result of constriction of cutaneous blood-vessels, by cold and subsequent paralytic dilatation of arterioles, which causes purple swollen areas in the skin. The usual positions are the fingers and the outer borders of the feet. The affection is commonest in children and old people. Inflammation and ulceration often supervene on congestion. Frost-bite, dealt with on p. 15, is a condition akin to chilblain.

Symmetrical Gangrene, or Raynaud's Disease, is akin to chilblains and frost-bite. Many persons, especially women, are subject to "dead fingers." That is, the finger becomes pale, numbed and insensitive, especially under the influence of cold. After this stage a period of congestion supervenes. A more severe degree of the same affection constitutes Raynaud's disease In cold weather the spasm of the arteries continues so long that the margins of the ears, the tips of the fingers, toes, heel,

or nose may undergo gangrene. The dead portions become black (*eschars*). The process is accompanied by shooting pain and itching. The sloughs separate slowly leaving intractable ulcers, or so much alteration of the part that amputation may become necessary.

Eczema.

Eczema may be defined as a superficial catarrhal dermatitis. The most characteristic lesions are vesicles, which on rupturing give rise to the characteristic serous discharge which dries on the skin, forming crusts. But, as in other skin affections, the stages of erythema and papules are passed through before vesiculation is reached, and the process may not go beyond the first or the second stage. Itching and burning generally occur before the eruption appears. The whole thickness of the epidermis is not destroyed by eczema, which does not leave scars. During the subsidence of the inflammation new surface epithelium is re-formed giving the "dry stage," and this new epithelium being formed in great abundance is shed as *scales* from the surface. In surgical practice the proneness of congested skin of the leg to eczema in cases of varicose veins is noteworthy. It is often accompanied by great thickening of the skin—elephantiasis. By the inoculation of eczematous surfaces with pyococci the whole thickness of the epidermis may be destroyed, in other words, ulceration may supervene. This is the commonest mode of origin of the ordinary varicose ulcer of the leg. Sections of eczematous skin show œdematous swelling of the remaining cells of the epidermis and inflammatory infiltration of the corium. Gouty conditions predispose to eczema.

Seborrhœic Eczema is a very common and important disease. It is probably due to a definite parasite. It is attended by hypersecretion of fatty matter from the sebaceous glands either in the form of seborrhœa oleosa or seborrhœa sicca (*dandruff*). In children it is commonest on the scalp where greasy patches of brown sebaceous secretion form over reddened skin, weeping and other features of eczema supervening. From the head the disease often extends to the face or neck. In adults the

disease is commonest in warm weather on the front of the chest (*flannel rash*), or it may appear, especially in women, about the climacteric as small patches covered with a dry, thin crust, and having a sharp margin. Such lesions may be mistaken for syphilis or psoriasis. Seborrhœa of the scalp is the commonest cause of baldness.

Eczema Intertrigo, or eczema from rubbing, is partly due to the irritation of retained sweat and sebum which accumulates in situations such as at the sides of or behind the scrotum, beneath the breasts of stout women, etc.

Cheiro-Pompholyx, or dysidiosis, is allied to eczema. It appears in hot weather as minute vesicles at the sides of the fingers and toes. The vesicles may run together, forming bullæ, the contents of which may become purulent and thus come to simulate a form of whitlow, differing, however, in this, that in whitlow the inflammation begins in the deep part of the corium, or in the subcutaneous tissue the matter formed piercing the corium and lifting up the epidermis. In cheiro-pompholyx the vesicles form between the layers of the epidermis.

Superficial Dermatitis from sunburn, Röntgen rays, washing soda, French polish, etc., though similar to eczema pathologically, differs in its causation, being definitely traceable to injurious agents. Certain varieties of this form of dermatitis are sometimes termed *trade-eczemas*.

Pemphigus is characterized by the formation of bullæ on previously healthy skin. It thus differs from conditions in which bullæ supervene on pre-existing inflammatory areas. Sometimes syphilitic lesions simulate pemphigus. It is better to speak of such lesions as bullous or pemphigoid syphilides, and not as syphilitic pemphigus.

Psoriasis is a superficial dermatitis characterized by the formation of dry, silvery, scaly patches, with a red margin; they usually make their first appearance on the backs of the elbows and the fronts of the knees; later they may appear in almost any part. Histologically they show increased thickness of the epidermis of the affected regions. Lesion for lesion they may be indistinguishable from some syphilitic eruptions. In psoriasis the lesions show a preference for the elbows or knees.

In syphilis the *polymorphism* of the eruption, that is, the co-existence of other lesions with the scaly ones, and the course of the disease, will suffice to make a distinction. Psoriasis may last a life-time: psoriasiform syphilides usually pass away in a few weeks or change their characters. The silverness of the scales is often wanting in syphilides. It is better to use the term "psoriasiform syphilide" than "syphilitic psoriasis."

Deep Dermatitis differs from eczema in that the lesions are placed more deeply, and tend to leave scars.

Dermatitis Herpetiformis. Met with in children and adults of both sexes, but especially in pregnant women. Any or every part of the body may be affected. The eruption is characterized by polymorphism; erythematous, papular, vesicular, and pustular lesions occurring. The vesicular and bullous lesions predominate. They leave pigmented scars. The disease shows a marked tendency to recur over a period of many years. The internal administration of arsenic is sometimes successful in checking the disease.

Herpes is characterized by clusters of vesicles on an erythematous area. Herpes of the lips, foreskin, etc., is common in children and young adults, especially during slight indisposition. *Herpes zoster* (shingles) is supposed to appear in the distribution of one or more of the posterior spinal nerve roots. It occurs also in the areas of distribution of branches of the fifth nerve. The lesions may leave scars.

Sclerodermia (*hard skin*) is a chronic disease which is marked by hardening of the skin from the formation of scar-tissue in the corium. The disease assumes two forms—diffuse sclerodermia and circumscribed sclerodermia or *morphœa.*

Diffuse Sclerodermia. In the more severe or atrophic form of the disease the affected skin becomes hard, tense, and rigid; also dry from the suppression of secretion. Before this cicatricial change occurs in the corium the skin is slightly œdematous. As the œdema disappears the skin shrinks and becomes white. Erythematous areas (*slight eczema*) may obscure the white colour. The affection may extend to the mucous membrane of the mouth, etc. In a second (infiltrated) form of the disease the œdematous stage lasts longer, and may end in resolution, the

skin again becoming soft, an event which rarely happens in the atrophic form. The skin of the whole body may be affected. The face, arms, and legs are the more common sites.

Morphœa resembles atrophic sclerodermia, only it occurs in the form of limited patches. The skin is parchment-like and white, save at the margin of the lesion, where it is purplish or pink. It may occur at any age, and usually gets well.

Lichen. This term is frequently vaguely applied to any papular eruption from eczema to syphilis. It is better to restrict it (Malcolm Morris) to one disease—lichen planus.

Lichen Planus, or *Lichen Ruber Planus.* A disease that shows itself in several modifications. It generally appears first as an eruption of small angular steep-edged, flat-topped papules arranged in wavy lines. The papules are covered by a thin scale, which gives them a lilac tint. The dorsal aspects of the hands, feet, and limbs are affected more frequently than the trunk, but the eruption may be universal on the skin, and it sometimes appears on the tongue. The rapidity of the spread of the eruption varies; in some cases it remains limited to one part, in other more acute cases it spreads over the whole body. In one variety of the disease the lesions are larger than usual. This is termed *Hypertrophic lichen planus.* The papules are as large as or larger than threepenny bits, and by spreading they may form much larger areas. The subjacent skin may become thickened, so that they form tumour-like elevations. The horny layer over these papules may become heaped up and warty (*Lichen verrucosus*). On the contrary the inflammatory infiltration of the corium may result in scarring (*Atrophic lichen planus.*) All forms of the disease show one common feature—chronicity. There is little tendency to spontaneous recovery. The affected parts itch violently. Pigment is deposited about them, causing them to simulate syphilitic lesions, from which the itching, however, distinguishes them. When the rash is abundant there is often great nervous depression, and this, together with exhaustion due to loss of sleep, etc., may lead to death.

Infective Skin Diseases.

Many of these have been discussed already, *e.g.* boil, carbuncle, etc. (see pp. 68 and 69). Other pustular affections are:

Acne is inflammation about a sebaceous gland. It first shows itself as a comedo, *i.e.* a plug of sebaceous matter in the neck of a pilo sebaceous follicle. Suppuration follows and scarring results. The face and back are the commonest sites of the disease.

Impetigo Contagiosa is a pustular affection commonest in children, and frequently arising in the punctures made by head lice. The pustules rupture and infect neighbouring areas. Scars and ulcers may form. The surgical importance of the affection lies in that it frequently leads to suppuration in lymphatic glands, and not uncommonly is the precursor of tubercular lymphadenitis.

Kerion is a variety of ringworm, in which small abscesses form at the bottom of the hair-follicles, so that a raised semi-fluctuant boggy area is present. The hairs fall out, and the pus escapes. This form of ringworm gets well in a much shorter time than the usual kind. Some permanent baldness may remain.

Tubercular Diseases of the Skin.

In the skin, as in other parts of the body, the gross effects of tubercle vary in rapidity according to the resisting power of the individual attacked. The same may be said of syphilitic, cancerous, and other lesions.

Lupus Vulgaris is simply cutaneous tuberculosis in an individual of a fairly high degree of resistance. **Scrophulodermia** is the more rapid tuberculosis in less resistant individuals. Each exhibits a gradation in rate of growth. In lupus the necrotic foci may form too rapidly for the process of absorption and cicatrization to keep pace with them; in other words, ulceration takes place. Hence two varieties—**Lupus non Exedens** and **Lupus Exedens**—are distinguishable.

Again, when the amber nodules of lupus are surrounded by a quantity of exudation and cell-infiltration, and the lupus

patch is raised above the surface; **Lupus Hypertrophicus** is constituted. Other varieties are constituted by slight peculiarities, such as a dilated state of the superficial blood-vessels, which gives rise to **Lupus Telangiectodes.**

What appears to be a patch of warts on a child's face may be found to have a reddened base, and if a piece of the skin is excised and examined under the microscope the giant cell-systems will be found, and the condition proved to be lupus This is **Lupus Verrucosus,** which affords a link between lupus and scrophulodermia.

Verruca Necrogenica is a localized raised red patch with a warty surface. It occurs in the hands of post-mortem porters and butchers, and is an inoculated tuberculosis which comes under the head of Scrophulodermia. Other examples of this condition are:

Simple Scrophulodermia, which may arise from the extension of tuberculosis from a lymphatic gland to the skin, or from a tubercular abscess (so-called tubercular gumma) in the subcutaneous tissue, or from tubercular bones, etc. I have seen tuberculosis arise in the wound left after a circumcision; more rarely the tuberculosis affects the corium primarily.

Lichen Scrophulosorum arises in weakly children in the form of flat, soft papules, reddish-brown or pale pink in colour, covered with scales at the top, and sometimes becoming pustular. The course is extremely slow. The disease is generally associated with tubercular glands, etc.

Acne Scrophulosorum is similar to lichen save that the lesions rapidly become pustular.

Erythema Induratum Scrophulosorum appears chiefly in the legs as round indurated areas resembling syphilitic gummata. The areas break down, leaving ulcers. Young girls with weak circulation form the majority of the subjects of this disease, which is regarded as tubercular.

Tubercular skin disease is of the greatest importance in surgery since, if a differentiation from syphilis is not made, an entirely wrong treatment may be adopted. Tubercular affections commonly commence before puberty.

Cysts and Growths of the Skin.

Dermoid cysts have already been described (p. 128).

Sebaceous Cysts are rightly regarded as retention cysts. They are common on the scalp, but may be met with wherever there are sebaceous glands. The duct of the gland may be patent or closed. In the former case the whole of the contents of the cyst may be expressed as a rancid pultaceous ill-smelling mass. On the scalp the duct is usually obliterated. With the distension of the cyst the skin over it may become gradually thinned and at last ulcerate; the cyst wall then also gives way and its lining is exposed as a reddish surface. This surface not infrequently assumes a cancerous character. A horn may also develop from a ruptured sebaceous cyst. Some persons are especially prone to the formation of sebaceous cysts, which appear to be caused by some agent which leads to slight inflammation and hypersecretion.

Adenomata, which may be cystic, sometimes arise in the sebaceous or in the sweat-glands.

Growths of the Skin. *Cheloid, or Keloid* (? Gr. *ch-el-e* = claw), is the result of an excessive formation of scar-tissue, and may start in even the most minute puncture, such as a pin-prick or the scar of an acne pustule. As commonly seen in surgical wounds it forms prominent ridges in the lines of the incisions, and if the latter radiate the growth has a claw-like aspect. At first red, the growth later becomes whitish like scar-tissue. It may increase slowly in size for years, assuming the character of a new growth which is composed of white fibrous tissue and spindle cells. In the course of time it tends to atrophy. Though it may be regarded as a form of new growth, it is really a variety of scar-tissue which in some individuals for some unknown cause does not undergo the normal atrophy. It is more common in the dark

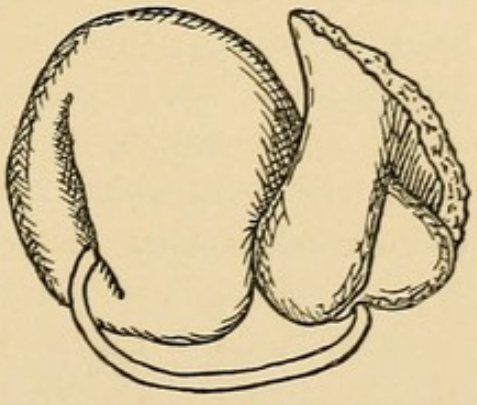


FIG. 194.—The lobule of the ear of a negro with a cheloid tumour growing on each side of the puncture made by the ear-ring. (499.)

races than in whites. In negroes the tumours are black, showing that they are covered with epidermis.

Xanthoma (*yellow-growth*) occurs in various forms; the commonest is *xanthoma planum*, which appears in the corium of the upper lids near the inner canthi; there it looks like a piece of wash-leather let into the lid. It is soft and smooth. Sometimes small cysts develop in it. Under the microscope it shows a structure very like atheroma of arteries. Removal is the only treatment, since the condition has no tendency to disappear, but after attaining a certain size it remains stationary for the rest of life. Sometimes it is hereditary, and it may be congenital.

Xanthoma Multiplex is usually associated with jaundice, but it may occur independently, especially in children. The eruption generally appears first in the eyelids, thence it becomes widespread over the body. The lesions may be flat, but they are more frequently nodular. They have been noticed in the mouth and pharynx. They are persistent. In *diabetes* an inflammatory form of xanthoma multiplex is occasionally observed. It improves under treatment.

Molluscum Contagiosum. Appears as little pearly bodies which project from the skin. They vary in size from a pin's head to a pea; they show a central depression, and spread locally from part to part. On section they are found to consist of an adenoma-like growth of epithelium, the centre of the nodule being occupied by bright oval bodies much larger than the epithelial cells. Sometimes the lesions suppurate.

Some Malignant Diseases of the Skin.

The chief forms of new growth of the skin have been described in an earlier part of this book. Here I would only mention three rare affections which are of pathological interest as being allied to malignant growths.

Mycosis Fungoides. Begins in erythematous patches which may be mistaken for eczema, etc. The patches vary in size from that of a sixpence to the palm of the hand. At first red, then scaly, they become moist, and later, after some

months or years, more and more infiltration occurs till they form red tomato-like projections which may be covered by scabs. Histologically the tumours resemble lymphosarcoma, and as in that condition the glands throughout the body may become involved.

What appears to be an obstinate eczema may thus be the beginning of a sarcomatous affection. These cases end in death practically always. In one case, however, recovery took place after an attack of erysipelas.

Xeroderma Pigmentosum. Begins as small pigmented spots resembling freckles, but rather darker, on the face, neck, arms and legs. These freckles are sometimes preceded by a rash resembling measles. Some of the freckles may disappear in winter, to return in summer. Later they become permanent and darker. They vary in size from a pin's head to a pea. Different lesions now appear—atrophic (scarred) spots which may run together; suppurating ulcers, the pus from which causes fresh ulcers by inoculation. Scabs form on the ulcers which heal. Finally, warty growths appear. From this time cancerous tumours appear and infiltrate and destroy surrounding tissues whilst they ulcerate on the surface in fungating masses. The spread of the disease is local, generalization rarely if ever taking place. The disease usually begins before the age of two years, but it has been known to begin in middle age. Histologically it has the characters of cancerous (epithelial) growth.

Acne Cornea, or **Darier's Disease,** is a chronic affection which begins as an indolent inflammatory induration of the pilo-sebaceous follicles of an area of the body. The follicles enlarge, become covered by an adherent crust and surrounded by a red infiltrated zone. This infiltration gradually increases, and in the course of several years assumes a cancerous character.

LITERATURE AND REFERENCES.

In the foregoing pages I have endeavoured to acknowledge the sources to which I am indebted for information. In many instances, however, it has not been practicable to trace directly obligations which none the less exist. I would mention, therefore, in this place, the general treatises which have most influenced my mind. They are:

Prof. Rud. Virchow's *Die Krankhaften Geschwülste.*
Sir James Paget's *Lectures on Surgical Pathology.*
The late Prof. Th. Billroth's *Surgical Pathology and Therapeutics.*
Prof. Joseph Coats's *Manual of Pathology.*
Prof. A. Weichselbaum's *Elements of Pathological Histology.*
Mr. A. J. Pepper's *Elements of Surgical Pathology.*
Mr. A. A. Bowlby's *Surgical Pathology.*
Mr. J. Bland Sutton's *Tumours.*

In acknowledging with gratitude these and other sources of information, it is only right to state that there is but little in the book that I have not confirmed by personal investigation. In some sections I have been aided by observations of my own previously published, *e.g.* "Illustrations of the Histology of Rickets," in Dr. Cheadle's article in Prof. Allbutt's *System of Medicine*; "Illustrations of the Pathology of Ear Disease," in Mr. G. P. Field's work on that subject; and "The Pathology of Diseases of the Skin," in Mr. Malcolm Morris's manual.

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