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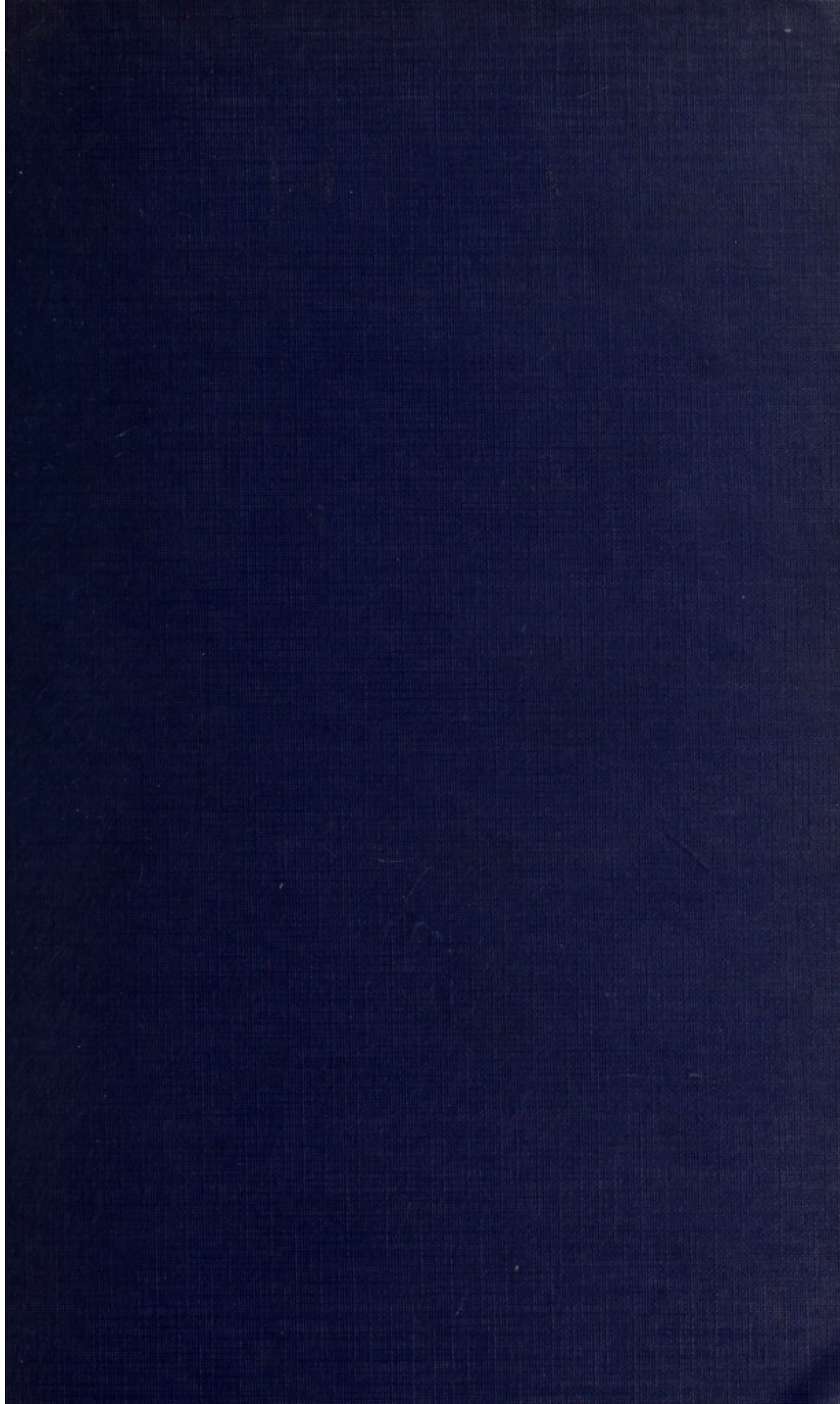
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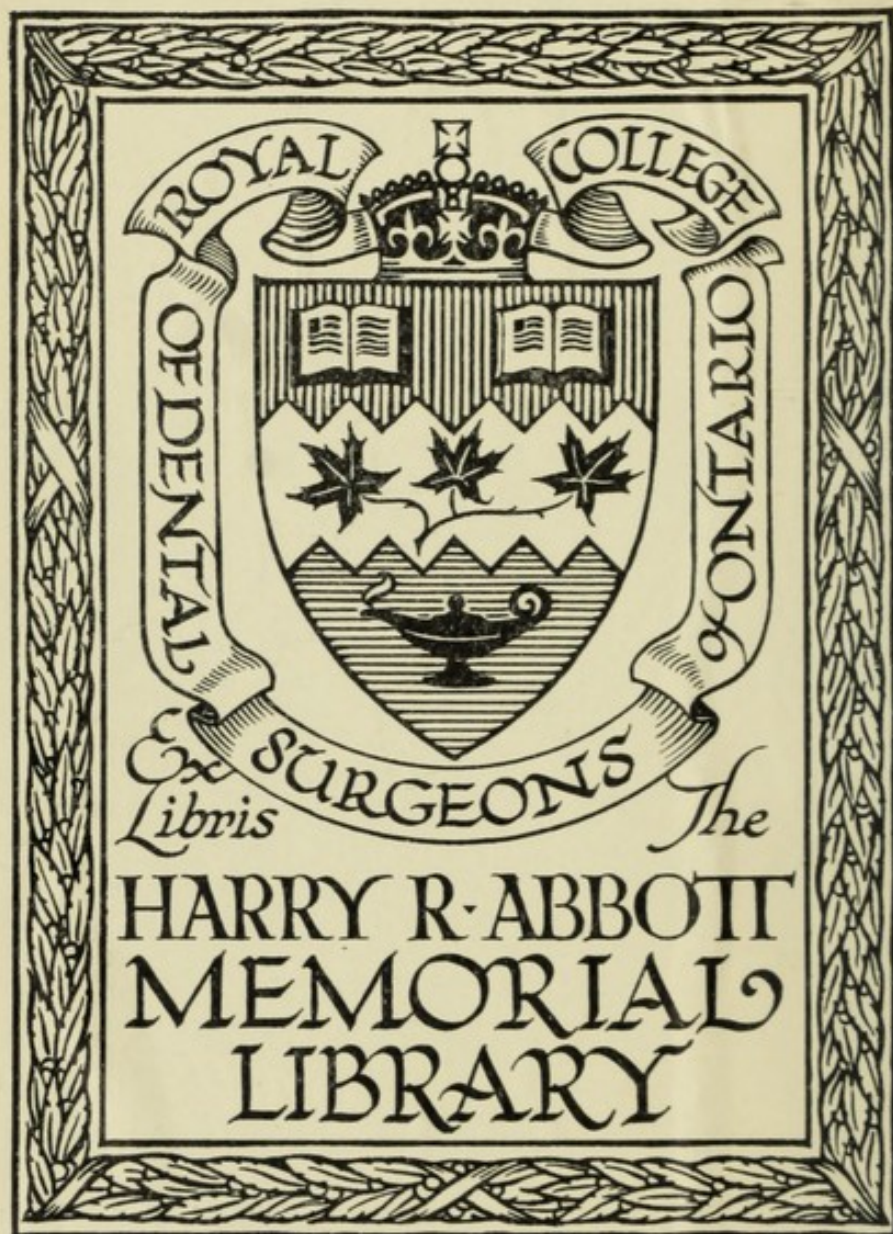
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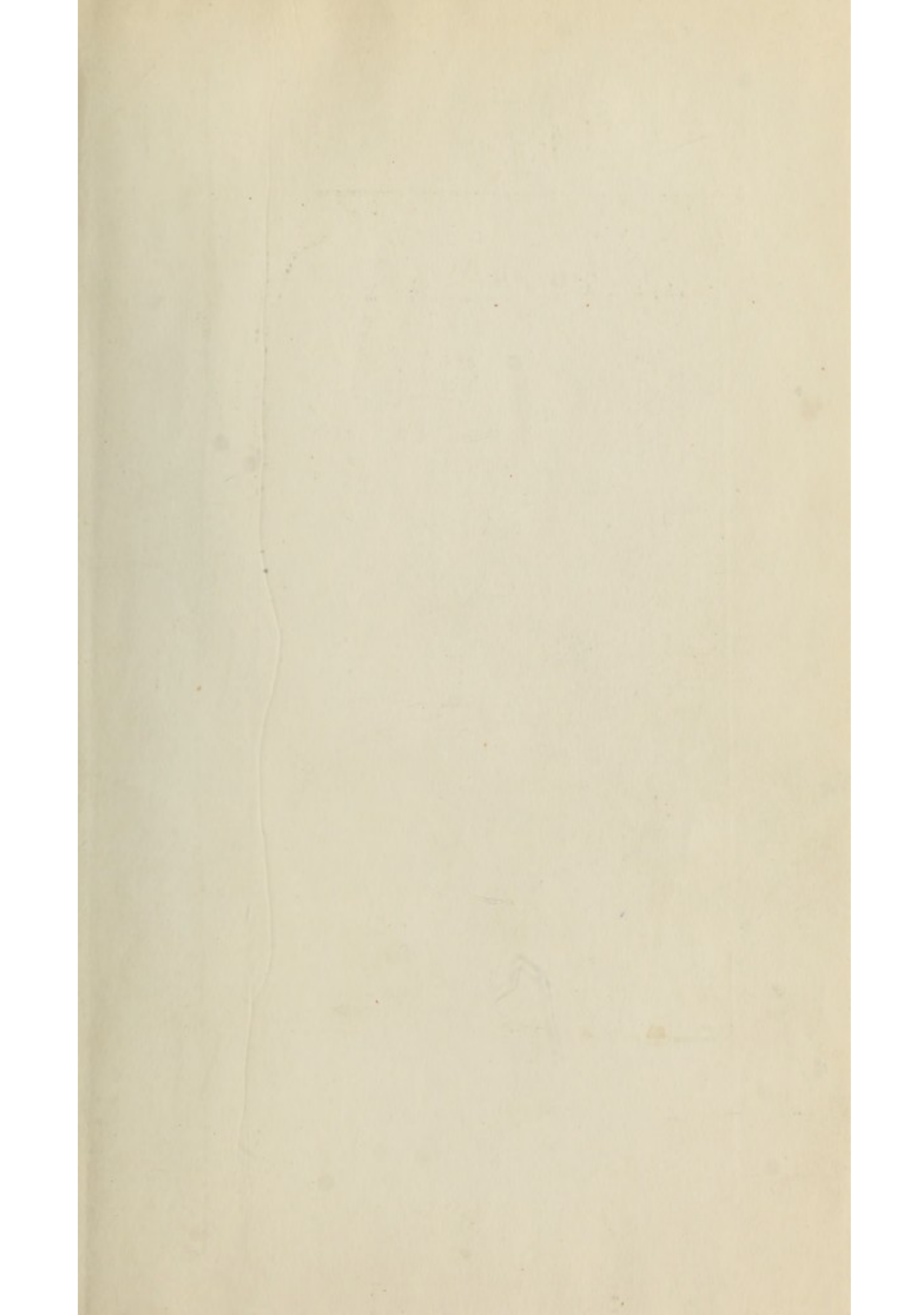


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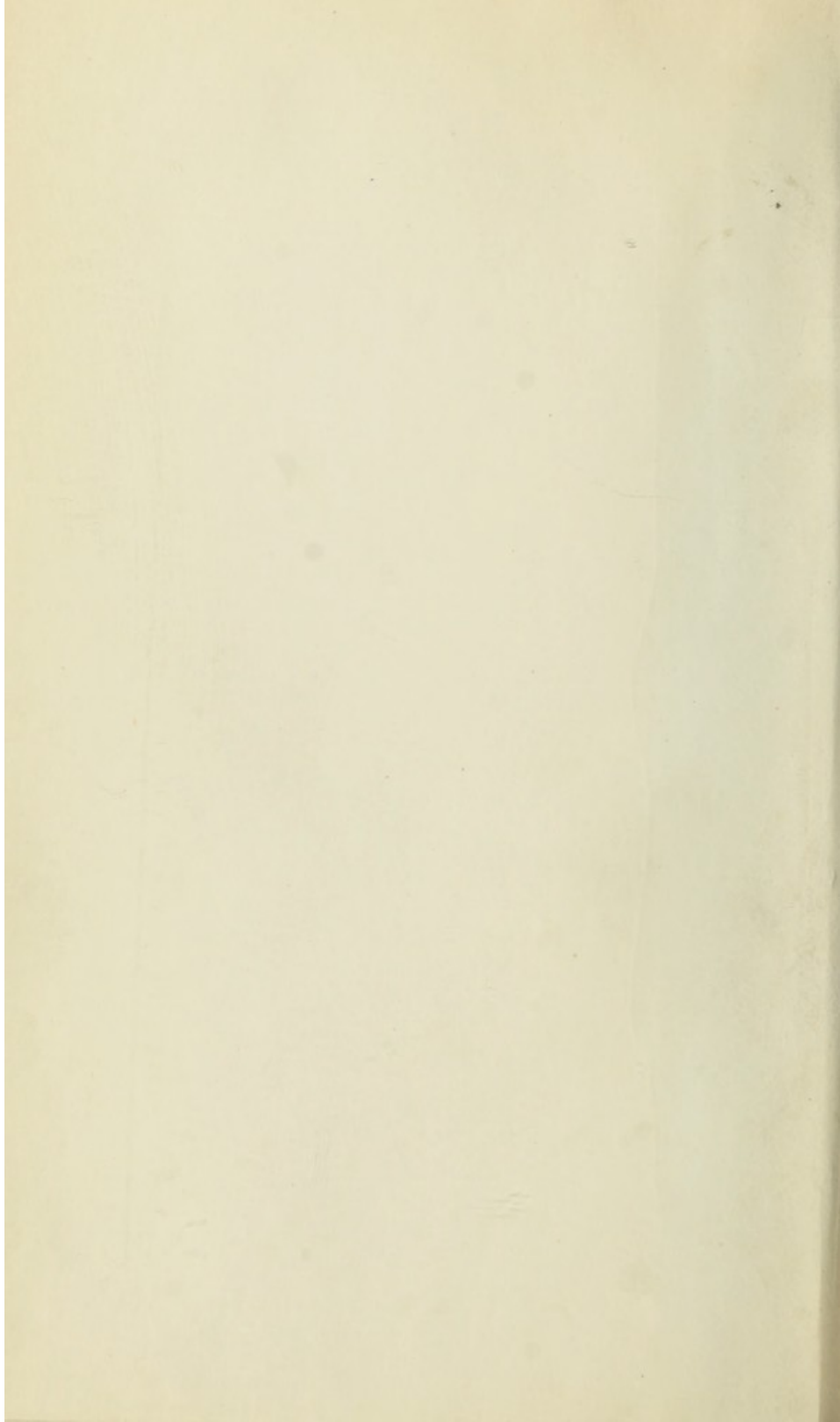


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GENERAL  
SURGERY AND PATHOLOGY  
FOR DENTISTS.

BY

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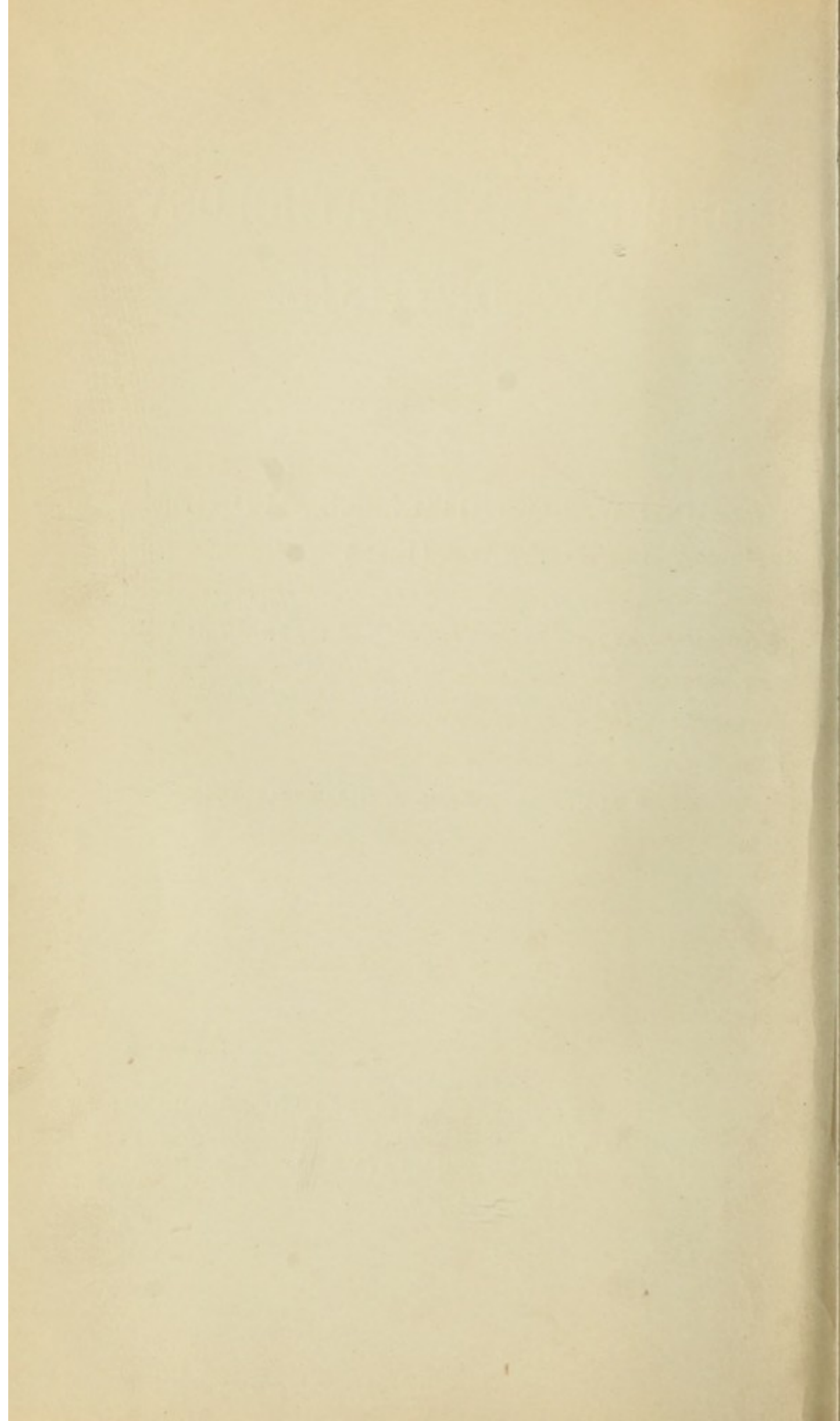
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## P R E F A C E.

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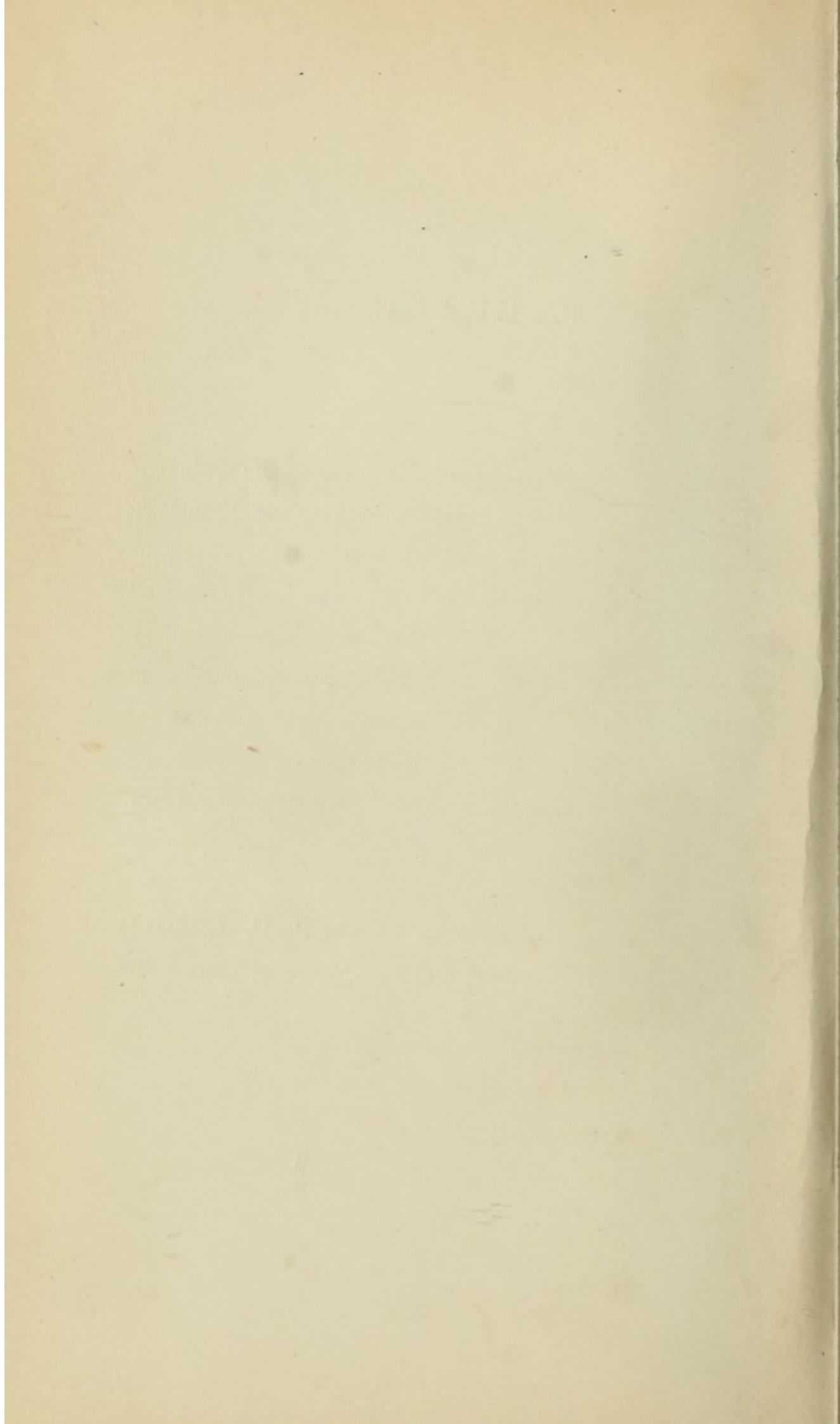
THE object of the following pages is to supply the student of Dentistry with an account of General Surgery and Pathology sufficiently comprehensive to enable him to practise his profession intelligently, yet concise enough to be easily mastered whilst preparing for his examination. It is hoped that they may save him the expense of purchasing a large work on Surgery, and the difficulty and uncertainty of choosing for himself what to read, and what to leave alone. Scarcely any reference has been made to the special surgery of the mouth, I hope to deal with that subject in another volume.

My best thanks are due to my friends, Dr. C. H. COSENS and Dr. KNYVETT GORDON for much valuable help in the preparation of the illustrations.

33, WESTBOURNE TERRACE.

*March, 1895.*

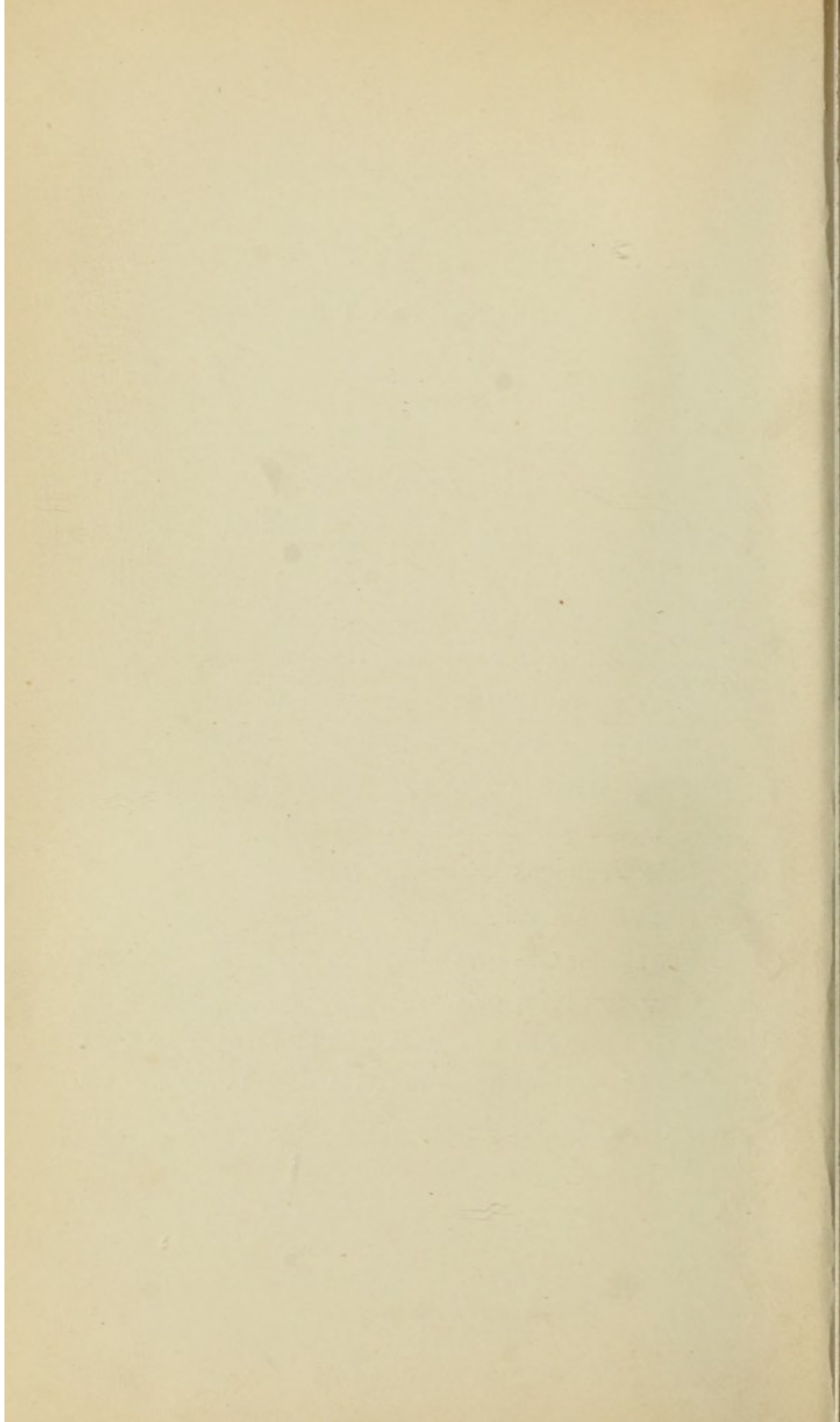




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GENERAL  
PATHOLOGY AND SURGERY  
FOR DENTISTS.

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INTRODUCTION.

---

EVERY practitioner of the healing art, no matter what his particular branch may be, cannot do his work with satisfaction to himself, and advantage to his patients, unless he possesses an adequate knowledge of the structure and working of the complicated human machine with which he has to deal, both in health and in disease.

Anatomy and Physiology deal with the structure and functions of the body in health, and Pathology with the same in disease. To the student of Dentistry it is not necessary to be acquainted with the whole of these extensive subjects. In the following pages an endeavour will be made to set forth, in as clear and concise a manner as possible, just so much Surgery and Surgical Pathology as the student in Dentistry will find necessary for his examinations, and for the intelligent and efficient practice of his profession.



## CHAPTER I.

## INFLAMMATION.

This important process underlies nearly the whole of Pathology, and must be thoroughly understood at the outset.

If an irritant be applied to a living tissue, a series of changes (collectively called inflammation) will ensue. These

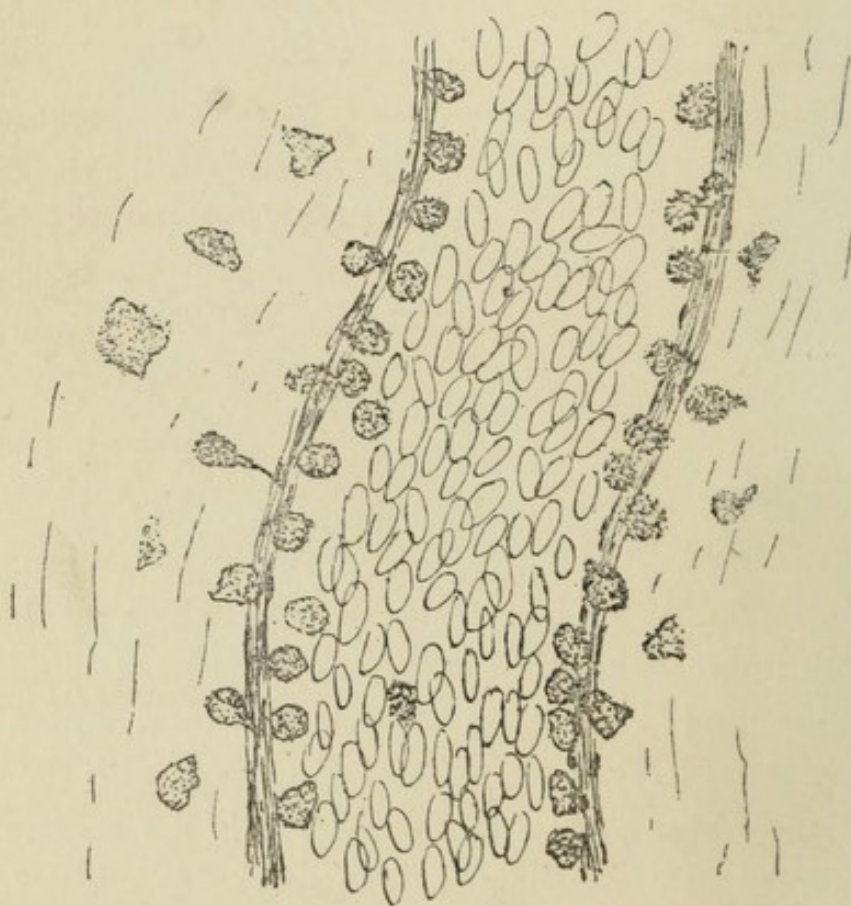


FIG. I.

Small vessel from the inflamed mesentery of a frog, showing migration of leucocytes.

changes can be best studied in a transparent tissue such as the mesentery of the frog. Suppose then that this tissue be spread out under the microscope, and be punctured with a

hot needle. The first effect noticed is a momentary contraction of the vessels in the neighbourhood of the injury. This is soon followed by *dilatation*, and an increased flow of blood to the part. Very soon the flow of blood gets slower and the white corpuscles (leucocytes) collect in quantities at the sides of the vessel and adhere to its wall, whilst the red corpuscles still pass on. Before long the stream becomes very slow indeed, the corpuscles passing onwards during systole, and back again during diastole. This is called *oscillation*, and is soon followed by *stasis* or complete stoppage. Whilst these changes are occurring, the liquor sanguinis oozes out into the tissues through the vessel wall, and the white corpuscles also escape by a process called *migration* or *diapedesis*; they work their way through the thin vessel wall by their own amoeboid movement, leaving no aperture whence they came out. (See Fig. 1.) When the inflammation is very intense a few red corpuscles escape too, but they are simply forced out by the increased pressure inside the vessel, not by any active movement on their own part, indeed their presence in the exudation is accidental.

*The Exudation.* In health, the fluid which filters through the vessel walls contains no fibrin-forming substances and is not coagulable. In inflammation, the damaged vessel walls allow the plasma to pass out almost unaltered. With the aid of the leucocytes which contain the fibrin ferment, the exudation clots. This clotted plasma with its entangled leucocytes is called *inflammatory lymph*, or *plastic exudation*. In other words lymph is clotted blood minus all or nearly all its red corpuscles. In the immediate neighbourhood of an inflamed part there is nearly always an excess of serous exudation as well.



*Changes in the tissues.* The inflamed tissue becomes swollen and softened. The swelling is due to the excess of fluid and leucocytes, and the softening is due partly to the soaking with exudation, and partly to the digestive action of the leucocytes, with which the tissue becomes crowded. Opinion has differed from time to time as to whether the new cells in inflamed tissue were derived partly from the connective-tissue corpuscles, or entirely from migrated leucocytes. It is now taught that some of these cells at least are the result of proliferation of the connective-tissue corpuscles. They are larger than the leucocytes and are called *fibro-blasts*.

TERMINATIONS OF INFLAMMATION.—(1) At any stage of the process, the inflammation may stop and the part may return to a condition of more or less perfect health. This is called *resolution*. If resolution occurs early, the crowding of the vessels diminishes and the circulation becomes restored; if it occur later after stasis has taken place, the obstruction melts away; if serum has been exuded, it drains away by the lymphatics; if plastic exudation has been poured out, the coagulated fibrin disintegrates and is removed by the leucocytes, which in turn either break up or disappear along the lymphatics.

(2) When the inflammatory process is severe the tissues may be so damaged that they liquefy under the combined influence of the exudation and the digestive action of the leucocytes. The latter which are in enormous numbers die for want of sufficient nutrition. The result is dead leucocytes floating in serum, or in other words *pus*. The process of formation of pus is called *suppuration*. When the pus forms a localized collection it is called an *abscess*.



(3) When the inflammation is near the surface of the body and causes a breach of continuity, the pus escaping externally, the process (essentially the same as suppuration) is called *ulceration*.

(4) Sometimes the inflammation is so intense that the whole or large part of the inflamed area dies en masse, before sufficient time has elapsed for it to become liquefied ; this is known as *mortification, gangrene, sloughing, or necrosis*.

(5) Sometimes the newly formed cells become converted into fibrous tissue, the result being *fibrous induration* or *scarring*.

(6) Sometimes the newly formed cells degenerate owing to deficient nutrition and form a caseous mass. The more liquid part of the caseous material may then become absorbed leaving only the calcareous constituents. These changes are called *caseation* and *calcification* respectively.

CAUSES OF INFLAMMATION. The immediate cause of inflammation is supposed to be some damage to the vessel walls, preventing them from performing their normal functions in controlling the osmosis of fluids between the tissues and the blood, which is the essential part of nutrition. This may be the result of either predisposing or exciting causes.

(1) *Predisposing causes* may be *constitutional* such as feeble circulation, anæmia, or impure blood the result of Bright's disease, gout, alcoholism, poisoning by mercury, lead, phosphorus, etc., or *local* such as congestion, loss of trophic nerve influence, loss of function, previous disease, etc.

(2) *Exciting Causes* are all some form of irritant, such as mechanical violence, heat, cold, electricity, chemical substances, the products of micro-organisms, or the presence of these organisms themselves.



THE SYMPTOMS OF INFLAMMATION may be divided into (a) *Local Symptoms*. These are :

*Pain* due to pressure upon or stretching of nerves. As a rule the pain is proportional to the amount of tension present.

*Heat* due to the increased amount of blood in the inflamed part.

*Redness* due to the increased amount of blood and dilatation of the vessels.

*Swelling* due to the increased amount of blood in the part and to the pressure of inflammatory exudation.

*Disturbance of function* due to impaired nutrition.

(b) *Constitutional symptoms*. The chief is fever, which subject will be dealt with subsequently : it is sufficient at present to point out that the fever in inflammation may assume three chief types (i) the *sthenic* in which the symptoms are acute, the pulse full, strong, and rapid, and the temperature high, (ii) the *asthenic* in which the strength fails, the pulse gets rapid and feeble and so called typhoid symptoms supervene, and (iii) the *irritative* in which delirium is a prominent symptom.

VARIETIES OF INFLAMMATION. Inflammation may be divided according to its intensity and duration into *acute* and *chronic*, or according to its cause into *traumatic*, *septic*, *infective*, *gouty*, *syphilitic*, *strumous*, etc., or according to its effects into *adhesive*, *suppurative*, *ulcerative*, *sloughing*, etc.

TREATMENT OF INFLAMMATION. The treatment may be divided into preventive and curative.

*Preventive Treatment* consists in avoiding or combating all the known causes of inflammation. Preventive treatment

in inflammation assumes its greatest importance in dealing with wounds, either accidental or operative, where our object is to avoid the occurrence of any more inflammation than that necessary for the healing process. This subject will be more fully dealt with when we consider the subject of wounds and their treatment.

*The curative treatment* must be both local and constitutional.

*The local treatment* may be summarised as follows :—

When possible remove the cause, such as a foreign body, a carious tooth, etc., or any source of irritation, such as micro-organisms and their products, by the employment of antiseptics ; tension by incisions or by drainage ; irritating applications by substituting less irritating dressings, etc. As far as possible the inflamed part should have perfect rest, both mechanical and physiological.

The circulation through the inflamed part should be restored to the normal condition by means of elevation of the part, application of cold in the early stages, or heat and moisture where suppuration is likely to occur ; astringents to diminish the calibre of the vessels under suitable circumstances ; local blood-letting by incisions, scarification, wet cupping or the application of leeches to relieve the congested vessels.

*Constitutional treatment.* When the inflammation is due to a constitutional cause such as syphilis, the appropriate remedies for that particular disease must be employed. In ordinary simple inflammations no constitutional treatment is necessary beyond opening the bowels with a saline purge. In young strong subjects when the fever is high and of the sthenic type, antiphlogistic or lowering treatment, such as



liquid diet, depressing drugs, (antimony, aconite, mercury), and sometimes, though rarely, general blood-letting by venesection, may be used. When the fever assumes the asthenic type, the treatment should be stimulating and supporting, the diet should be light, nutritious, and easily digestible, and combined with a suitable amount of brandy, or other stimulant, and drugs such as ammonia, quinine and iron should be administered.

Pain should be relieved by opiates or other anodynes.

### CHRONIC INFLAMMATION.

Chronic inflammation differs from acute inflammation in that the process is less severe but of much longer duration; otherwise the pathological changes are essentially the same. Constitutional causes are of greater frequency and importance and local causes of less import, and often difficult or impossible to discover. The inflammatory exudation contains less fibrin-forming elements and is more prone to develop into fibrous tissue causing induration, but, as in acute inflammation, suppuration, ulceration, or caseation may result.

*Symptoms.* The pain is usually less severe, heat is often absent or slightly marked. The redness is more livid in hue or may be absent, but swelling is usually well marked. The constitutional symptoms are unimportant, being more often due to the general dyscrasia of which the inflammation is the result. Fever is mostly absent.

*Treatment.* Means should be adopted to improve the general health, especially when any definite constitutional disease such as syphilis or struma is present. Rest should



be provided, and every discoverable source of irritation removed. The absorption of inflammatory products should be promoted by counter-irritation, by blisters, or iodine, friction, massage, shampooing ; the rubbing in of absorbent ointment, like oleate of mercury, Scott's dressing, etc., pressure by strappings or bandages, or application of the actual cautery.

### CATARRHAL INFLAMMATION.

Catarrhal Inflammation, or catarrh, affects surfaces covered by epithelium. It most often affects mucous membranes, but also affects the skin in the disease known as eczema.

The inflammatory changes already described take place beneath the epithelial covering, and the products of inflammation escape through or between the epithelial cells upon the surface. The epithelial cells multiply rapidly, dividing into smaller and rounder cells, some of which are carried away in the exudation. At the same time an abundance of thin mucus is secreted and this mixed with the exudation derived from the blood forms the sticky watery discharge characteristic of mucous catarrh. Sometimes the number of small round cells in the exudation is so large that it becomes purulent in character. When the inflammation is sufficiently severe and prolonged the epithelial cells are destroyed and the result is an ulcer.

### SUPPURATION.

As already stated, when the inflammation is severe the tissue may become so crowded with inflammatory cells



(leucocytes and proliferated connective-tissue corpuscles) that it melts away and disappears, its place being occupied by these cells floating in serum, in fact by pus. (Fig 2.)

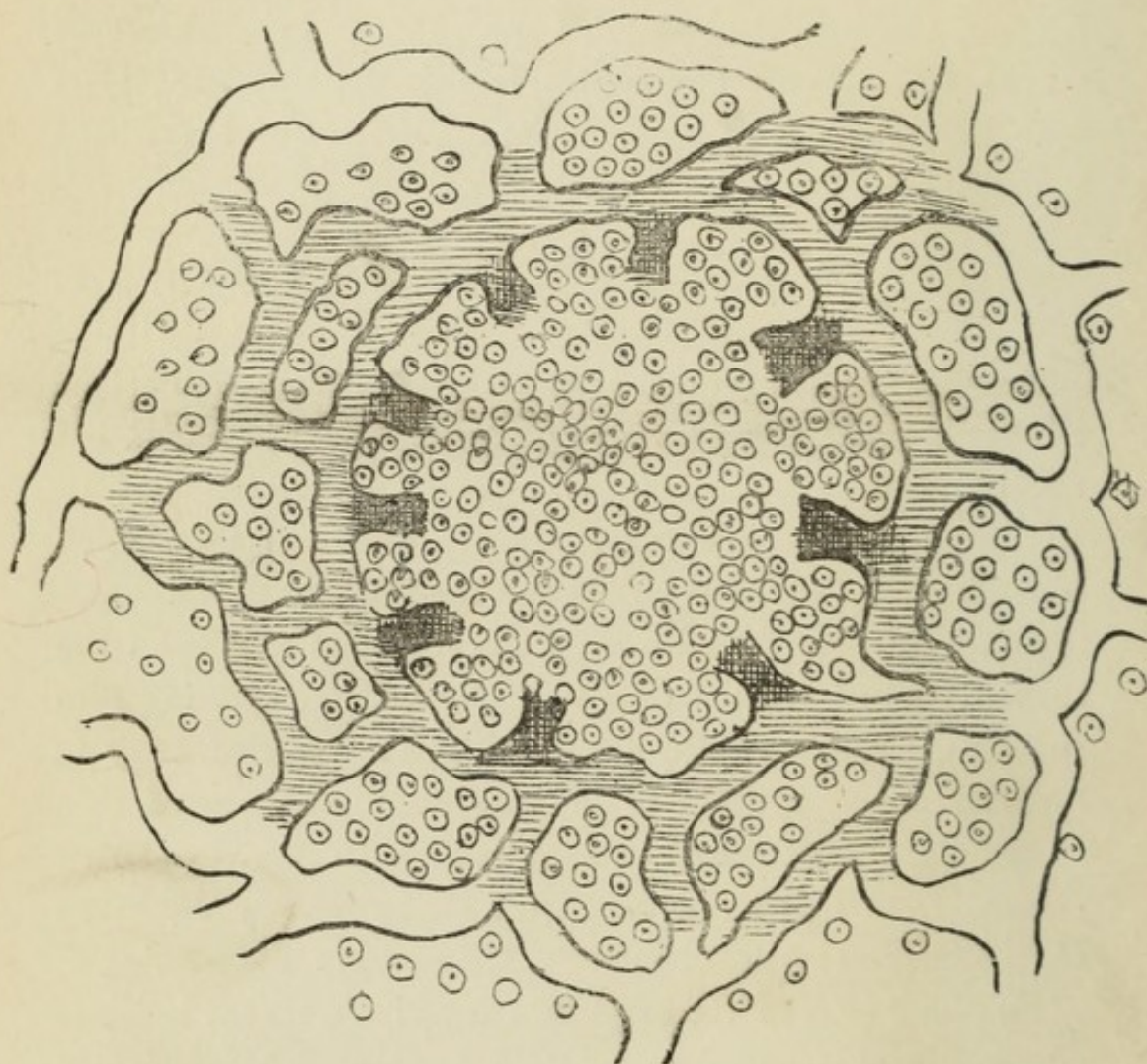


FIG. II.

Diagrammatic sketch of the formation of a small abscess. In the centre (abscess cavity) the tissues have melted away and nothing but pus is visible. The wall of the abscess is formed by congested and occluded capillaries and inflammatory cells.

*Characters of Pus.* Pus is a creamy, yellowish white opaque fluid of slightly alkaline reaction and a specific gravity of about 1030. It varies considerably in appearance under



different circumstances, being sometimes thin and watery (ichorous), sometimes blood-stained (sanious), sometimes mixed with mucus (muco-pus), sometimes containing cheesy looking flakes (curdy). Under the microscope it is seen to consist of corpuscles floating in a fluid. The corpuscles are globular, granular, about 1-2500th of an inch in diameter, and contain two or three nuclei. They are dead cells and are derived from the migrating leucocytes and the proliferated connective-tissue corpuscles. The fluid portion is called *liquor puris* and differs but slightly from blood serum.

Pus may form in different situations in the body. When a definite collection forms in the interior of a solid tissue, the result is an *abscess*. Fig 2 represents diagrammatically the way in which this takes place. At the periphery are represented a number of congested capillaries from which small round cells are exuding in large quantities; nearer the centre the congestion is greater and many of the capillaries are thrombosed. In the centre of the diagram the tissues and capillaries have disappeared and only round cells are visible.

In some cases of acute inflammation the pus instead of forming a definite collection infiltrates the tissues; this is called *diffuse suppuration*.

In other cases the pus runs off from a surface, as in ulceration or purulent catarrh.

*Causes of Suppuration.* These are of course very much the same as those of inflammation. It is, however, very important to understand that a single irritant once applied produces simple inflammation which soon undergoes resolution, but if the irritation be kept up the inflammation continues and the excessive number of exuded leucocytes and proliferated connective-tissue corpuscles die from want of nourishment and



form pus. In the great majority of cases (according to some authorities in all cases) the prolonged irritation which results in suppuration is due to the presence of micro-organisms. It should be added that any local or constitutional condition which lowers the vitality of the inflamed part renders suppuration more likely to occur.

*The symptoms* of acute suppuration are fever, usually accompanied by shivering (rigors), and certain changes in the inflamed part; the swelling increases and the skin over it becomes red, glazed and œdematous, so that it pits on pressure and in the midst of the inflammatory induration a soft spot can usually be detected. When the pus has formed in sufficient quantity, and near enough to the surface, an elastic feeling known as *fluctuation* is detected. If left alone the abscess becomes prominent at one place (*pointing*) and bursts.

*Treatment.* Endeavours should be made to prevent the occurrence of suppuration by removing every source of continued irritation, especially micro-organisms. When suppuration actually threatens, it may sometimes be prevented by the use of cold evaporating lotions. When the formation of pus becomes inevitable, it may be hastened by the application of warm, moist, antiseptic dressings. When pus has actually formed, the indications are to evacuate it with as little damage as possible to the tissues, to provide efficient drainage, and to place the part at rest and free from all sources of irritation.

CHRONIC SUPPURATION may occur either from a free surface, as in chronic purulent catarrh, and chronic ulceration, or in the form of a localized collection (chronic abscess). The process differs from acute suppuration only in being



slower and less severe. A chronic abscess is usually due to diseased bone, joint disease or tubercular gland disease. It usually forms a fluctuating swelling with no active inflammatory characters, and no constitutional disturbance, unless it has been opened and decomposition of the pus has occurred. A chronic abscess may remain in *statu quo* for a long time or it may increase and burst, or the liquor puris may become absorbed leaving a caseous mass which may ultimately calcify.

### MODE OF HEALING OF AN ABSCESS.

The bursting of an abscess is immediately followed by considerable diminution in the size of its cavity, owing to the pressure of the surrounding tissues. The inflammatory cells lining the abscess cavity arrange themselves into little pyramidal heaps called granulations, and the neighbouring blood vessels send minute capillary offshoots into their bases. The cells at the apices of the granulations float off in the serum, which is constantly exuding, and become pus cells, whilst those in the deeper parts, with the coagulated plasma by which they are held together, become converted into fibrous tissue. This newly formed tissue shrinks, causing still further diminution in the size of the cavity. The granulations go on growing and becoming gradually converted into fibrous tissue until the cavity is quite filled up, and its former site occupied by a scar which becomes covered over by epithelium derived from the neighbouring skin or mucous membrane. The more the neighbouring parts will allow the new fibrous tissue to contract, the quicker the process of repair is; thus in abscess of bone the healing is slow as the cavity has



to be entirely filled by the granulating process, no shrinking being possible. Sometimes an abscess does not heal completely, but contracts into a narrow suppurating track called a *fistula* or *sinus*. The walls of a sinus are usually thickened by fibrous tissue and lined by ill-formed granula-

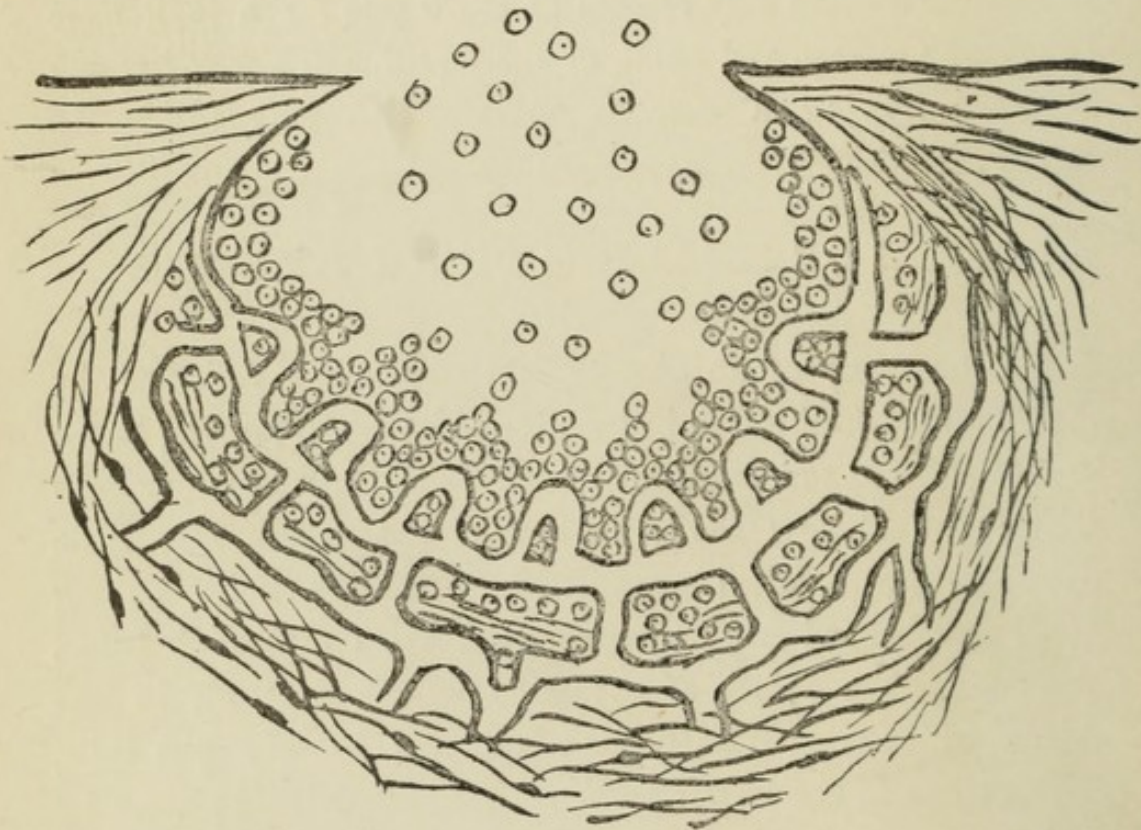


FIG. III.

Diagrammatic sketch of the healing of an abscess. The inflammatory cells have arranged themselves into granulations, each containing a capillary loop. Pus cells are being exuded into the abscess cavity and fibrous tissue is being formed outside the wall.

tions, and the orifice is usually surmounted by a crop of protruberant granulations. The degeneration of an abscess into a sinus is usually due to some foreign body such as a piece of dead bone or the root of a carious tooth, keeping up the irritation.

## ULCERATION.

Ulceration is essentially the same process as suppuration. In both there is molecular death and disintegration of tissue following upon, and due to inflammation, and death of the exuded leucocytes and proliferated tissue-cells producing pus. In an ulcer the process takes place on the surface of skin or mucous membrane. An ulcer may be said to be an open abscess, and conversely an abscess may be said to be a closed ulcer.

An *ulcer* is an open sore or loss of substance of skin or mucous membrane produced by ulceration or sloughing. The floor of an ulcer is composed of granulation tissue and exactly resembles in microscopic structure the wall of an abscess. Healing takes place in exactly the same way. Many different varieties of ulcer are described ; these we need not consider at present, but it will be useful to summarize the various points which must be noted in describing the appearance of an ulcer for purposes of diagnosis or record. They are as follows :—

1. Number, size, and shape.
2. Situation.
3. The surface and the discharge—character of the granulations.
4. The edges, whether raised, undermined, sloping, sharply cut, etc.
5. The base. This means the part immediately under the surface of the ulcer. The base therefore cannot be seen. Its character must be determined by feeling, the important thing to ascertain, being whether it is indurated or not.
6. The condition of the surrounding parts.



## GANGRENE.

Gangrene is death of a part *en masse*. Various conditions, such as Bright's disease, diabetes, heart disease, old age, etc., may act as predisposing causes by lowering the vitality of the tissues, thus rendering them less able to withstand injurious influences. The immediate or exciting causes of gangrene act either by cutting off the blood supply, or by destroying the vitality of the tissue.

The blood supply of a part may be cut off by obstruction of the main artery due to ligature, injury, thrombosis, and embolism, by obstruction of the arterioles by spasm, as in Raynaud's disease and ergotism, or by obstruction of the capillaries from pressure, as in bed sores. Obstruction of venous circulation may be a cause of gangrene by indirectly interfering with the arterial or capillary circulation. The vitality of a part may be destroyed by physical or chemical causes such as severe crushing, burns or scalds, frost bite, strong acids and alkalies, electrolysis, etc., or as already mentioned as a result of inflammation. It is especially in tissues which cannot yield or expand that inflammation is apt to lead to gangrene, for in these the exudation may lead to such tension, that the circulation through the capillaries becomes arrested, and the nutrition of the part stopped.

When gangrene has taken place the dead part becomes cast off by a process of ulceration in that portion of the living tissue in immediate contact with the dead. This process is called the formation of the *line of demarcation*.

The general principles of the treatment of gangrene are (i) To remove the cause when possible, (ii) To aid the removal



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of the dead part, (iii) To prevent decomposition as much as possible by the use of antiseptics, (iv) To support the patient's strength, and (v) to relieve pain.

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## CHAPTER II.

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### BACTERIA IN RELATION TO DISEASE.

Bacteria are minute specks of vegetable protoplasm. They are widely distributed in nature, being found practically everywhere, except in the middle of the ocean, at very high altitudes, and deep in the earth. They form a considerable portion of the dust which can be seen floating in the air in bright sunlight, and which deposits itself everywhere, especially in large towns. These minute organisms in their proper places perform useful functions, such as the conversion of starch into sugar, of sugar into alcohol, of alcohol into vinegar, &c. ; processes which are essential in various commercial operations. They also perform the function of general scavengers. Were it not for their almost universal presence, dead animal and vegetable matter would remain practically unchanged for indefinite periods, but by their agency dead organic matter undergoes decomposition whereby

complicated organic chemical compounds are split up into simpler inorganic compounds which serve as nourishment for vegetable life.

Unfortunately for humanity at large, (perhaps fortunately for the practitioner of dentistry) these organisms sometimes exceed their duty and attempt, too often with success, the decomposition of portions of our body which we would rather retain intact. Decay of teeth is nothing more or less than decomposition or devouring of dental tissues by different sorts of bacteria. It is essential therefore that the student of dentistry should be familiar with these organisms, which in a sense may be termed the authors of his existence. The following are some of the most important forms met with.

*Bacilli* are minute rod-shaped organisms averaging about  $\frac{1}{6000}$  inch long and  $\frac{1}{25000}$  inch broad. It is only with the highest powers of the microscope that their structure and characters can be accurately observed. They consist of a very thin wall containing living protoplasm (myco-protein). They grow in two ways, either by fission, i.e., one bacillus divides into two, or by the formation of spores or seed-like bodies. (Fig. 4 *a* and *b*.)

*Micrococci* are minute spherical cells, about  $\frac{1}{25000}$  inch in diameter, having the same structure as bacilli, but only multiplying by fission. They are often grouped in pairs (diplococci), or in clusters (staphylococci), or in chains (streptococci). (Fig. 4 *c*.)

*Spirilla* or *vibrios* are twisted screw-shape organisms. (Fig. 4 *d*.) They multiply by fission.

Certain conditions influence the growth and development of bacteria. Most of them grow readily at the temperature of the body, 37° C. Some of them will grow at temper-



## DIFFERENT FORMS OF BACTERIA.



Fig. 4.

A.—Bacilli.

B.—Spore-bearing bacilli.

C.—Micrococci, diplococci, streptococci and staphylococci.

D.—Different forms of spirilla.

atures as low as  $20^{\circ}$  C. Growth is almost or completely stopped by temperatures above  $40^{\circ}$  C. or below  $5^{\circ}$  C. All bacteria are killed by boiling, but their spores will stand even this amount of heat, being extremely resistant and durable. Some bacteria (aërobic) will only grow when there is a sufficient supply of oxygen; others will not grow in the presence of oxygen (anaërobic); others again are indifferent to this gas, doing equally well with it or without it. Food is necessary for all living things. In the body bacteria live on such things as blood, serum, lymph, saliva, débris of food in the mouth, etc. In the laboratory artificial culture media are used as food materials. They consist mostly of peptonised beef extract, either in the fluid state or solidified by the addition of gelatine or agar-agar. Moisture is essential for bacterial growth, most forms being killed by drying. Bacteria are also killed by certain chemical substances called *antiseptics*.

#### EFFECTS OF BACTERIA ON THE BODY.

Bacteria may effect an entrance into any part of the body either directly, by means of a wound or breach of continuity of surface, or indirectly, by means of the alimentary or respiratory tracts. According to the effects they produce they are divided into two important classes:—

(a) *Saprophytic, septic, or non-pathogenic* organisms can only live on dead tissue, consequently they are only found in the secretions of a wound, or on some mucous or cutaneous surface; if they should find their way into the circulation they very soon die, being incapable of splitting up living organic matter for purposes of their own nutrition. They are, however, capable of chemically decomposing dead organic matter such as serum, the result being that certain



poisonous substances called *ptomaines* are formed, which act as irritants to the living tissues producing inflammation, and even suppuration if their action is sufficiently severe and prolonged. Sometimes, but by no means always, the process of fermentation, whereby the ptomaines are produced, is accompanied by the evolution of foul-smelling gases—*putrefaction*. The ptomaines may become absorbed into the blood, and, acting as poisons on the heat regulating apparatus, produce fever.

(b) *Parasitic, infective, or pathogenic* organisms, although able to live on dead organic matter like the saprophytes, are distinguished from the latter by their ability to decompose living organic material, and thus maintain an existence in the blood and tissues, producing grave alterations of nutrition, in as much as they appropriate to their own use various nutritive materials intended for the benefit of the tissues, and at the same time contaminate the blood with their poisonous secretions. A few pathogenic organisms such as the anthrax bacillus, seem to be able to flourish in the blood of a healthy animal, provided they have once been successfully inoculated, but the majority of them are only capable of successfully attacking the tissues when the vitality of the latter has been lowered by some local or constitutional predisposing cause, and this is because living blood has a certain power of destroying bacteria. It is at present uncertain how the bacteria are killed, but it seems pretty certain that leucocytes are capable of swallowing and digesting them (*phagocytosis*). If the vitality of the leucocyte (or phagocyte) is lowered, it is apt to find the bacterium too tough for its digestive powers, and itself dies like a soldier fighting for his country.



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THE BACTERIA OF THE MOUTH.

The mouth forms an excellent incubator or warm chamber for the growth of bacteria ; its temperature is pretty constantly about 37° C. which is, as already stated, the optimum temperature for the growth of most organisms ; sufficient access of air is afforded for those germs which require oxygen or are indifferent to its presence. Food material for them is present in abundance ; dead and cast-off epithelium affords nourishment for at least some germs, for it is not uncommon to find squamous epithelial cells covered and partly disintegrated by chains of bacteria. Particles of food remaining between the teeth after a meal are utilised by bacteria ; this may easily be proved by chewing up some bread and placing it in a stoppered test tube in the incubator ; after two or three days, examination under the microscope will show that many different sorts of bacilli and micrococci are present in abundance. Even such a hard substance as dentine is, when decalcified, readily devoured by many of the mouth bacteria. When we add to the above food materials the exposed pulps of teeth, buccal mucus, and inflammatory exudations from the gums, we shall see that there is no lack of nourishment for germs resident in the mouth. Wherever there is something worth having and nothing to pay for it, it is only natural to expect a crowd ; and so it is in the mouth, for here germs simply abound. Whenever we open our mouths to take in food, drink or air, we admit at the same time a great many micro-organisms of different sorts, and it is only reasonable to expect that they, finding such comfortable quarters, should settle down and propagate. These considerations might lead one to suppose that the human mouth would contain every single germ in existence ; and so it would were



it not for "the struggle for existence" which is constantly going on everywhere, causing the stronger to prevail and the weaker to perish ; thus it is that although over a hundred different sorts have been discovered, the number of regular tenants of the mouth is very small, viz., about six. It seems that the conditions in the mouth exactly suit these few bacteria, enabling them to crowd out all others ; moreover, it is a strange fact that they absolutely refuse to grow outside the mouth on any of the artificial nutrient media of which we know.

If a small portion of the soft white matter, which collects upon the teeth, be spread out in an extremely thin layer upon a cover glass and stained with an aniline dye, such as gentian-violet, a specimen of great beauty and interest, from a bacteriological point of view, will be produced. If we examine it with a one-twelfth oil immersion lens we shall see swarms of micro-organisms of different sorts and sizes, some being rod or thread-shaped, some round or egg-shaped, and others spiral or screw-shaped.

(a) *The rod-shaped organisms.*—Of these there is a great variety, both with regard to length and breadth, some of them being jointed transversely and others jointless. The name leptothrix is commonly given to the jointless thread-like forms ; the name, however, is not a good one, as authors are not agreed amongst themselves what to include under it, and what to exclude. Most commonly leptothrix filaments appear as a jumbled-up heap, like an entangled piece of string ; sometimes the threads are much larger and wavy in outline, many threads being attached to a sort of common root of structureless material.

The jointed rod-shaped organisms (bacilli) also vary greatly



in size ; notable amongst them on account of its dimensions is the bacillus buccalis maximus, which is found in long thick jointed rods about three to ten  $\mu$  long, sometime singly, sometime in tufts. It is stained violet by iodine, whereas the leptothrix remains unaffected. Many other bacilli will usually be seen in different specimens and in different mouths; some of these will be referred to presently.

(b) *Spherical Organisms or Micrococci*.—The micrococci, although perhaps as numerous, are on account of their smaller size less conspicuous than the bacilli and threads. They are sometimes in clusters, sometimes in chains, or tetrads, or pairs, or scattered singly. The pairs, tetrads and chains, when short, are sometimes enclosed in a transparent glassy-looking capsule.

(c) *Spiral Organisms*.—The occurrence in the mouth of curved organisms resembling a comma has long been recognised. Professor Lewis drew especial attention to them, and he and Klein at one time regarded them as identical with Koch's cholera bacillus, but subsequent investigation has shown that although much alike in morphological character, they differ in this marked respect, viz., that whereas the cholera vibrio grows readily on gelatine, the comma bacillus of the mouth (*spirillum sputigenum*) refuses to grow anywhere but in the mouth. In an ordinary clean and healthy mouth it is found in small numbers, but under the margins of slightly reddened and inflamed gums it is found in large numbers. When examined in its natural live state, it is seen to be highly motile ; it is by means of flagella or whip-like processes that these motile organisms move about. The number and mode of attachment of the flagella varies a good deal ; in some of them only one flagellum is to be seen.



attached either to one end, or to the middle of either the concave or convex side of the comma, whilst in others a bunch of four or five flagella is not infrequently seen. They may exceed the length of the comma itself by several times ; they cannot be seen in the unstained condition, nor indeed by the ordinary method of staining. They require to be treated with a mordant, and then stained after the method of Loeffler. Sometimes two or more comma bacilli may be seen in a row, end to end, forming a short spiral ; this is due to incomplete fission.

Another spiral organism often found under inflamed gums is the spirochæte dentium. It occurs in the form of long wavy spirals of varying thickness and size of twists.

The organisms just described which occur in nearly every mouth, are nearly all of them non-pathogenic, *i.e.*, they are incapable of producing any morbid condition when injected into the blood or tissues of an animal. The fact that they are uncultivable has rendered it probable that they are also uncultivable pathogenic organisms inhabiting the mouth ; indeed such organisms have been described by Kreibohm and by Miller. But passing these by, there are others which have been cultivated and very fully studied.

It has long been known (even to the ancients) that human saliva was poisonous. This toxic property has been, from time to time, attributed to various causes, but it was first shown by Raynaud and Lannelongue in 1881 that it was really due to the presence of micro-organisms. About the same time Pasteur discovered in the mouth of a child suffering from hydrophobia an encapsuled diplococcus resembling the figure 8. Saliva containing these germs, when injected into rabbits, produced a rapidly fatal result. This he con-



sidered to be the specific germ of hydrophobia, but Vulpian soon afterwards produced the same symptoms and fatal result by injecting healthy saliva, and the same capsule cocci were found in the blood of the dead animals. A. Fraenkel discovered the same organism in cases of croupous pneumonia, and others have since found it in peritonitis, pleurisy, meningitis, otitis media, and other inflammatory processes. In the causation of these various diseases it plays a most important part, if indeed it is not solely responsible for them. This important organism is now designated the micrococcus of sputum septicæmia. It occurs frequently in the mouths of healthy persons in the form of encapsuled diplococci, which are not always easy to find under the microscope, but whose presence may be demonstrated by injecting some of the saliva into mice. Out of one hundred and eleven mice killed in this way, Miller found the micrococci of sputum septicæmia in the blood and tissues in sixty-one. The saliva had been taken from a different person in each case.

This micrococcus will grow on culture media, but not readily. It requires a temperature somewhere between 24, and 42° C., and a faintly alkaline medium. The colonies resemble dewdrops in appearance. They will not live much longer than five or six days, after which time, if it is desired to further propagate the species, it must be done by passing it through the body of a susceptible animal, and from its blood inoculating fresh culture media.

#### EFFECTS OF MOUTH BACTERIA.

As already stated, dental caries is nothing more or less than the devouring of the teeth by the bacteria of the mouth ; the only conditions that the germs exact are that the dentine shall be exposed by removal of its enamel covering, and decalcified



by acids. We are not concerned at present with the numerous causes which bring about these conditions, but it should be clearly understood, that in the absence of bacteria, caries cannot occur. If properly prepared sections of decayed dentine be examined under a high power it will be found that the tubes are stuffed with bacteria of different sorts, in some places being bulged out into the so-called liquefaction foci. Some tubes are found to contain only micrococci, others bacilli only, others spiral organisms, while frequently the infection is "mixed." Sooner or later, if the decay is unchecked, the bacteria gain admission to the pulp cavity, setting up acute inflammation which may lead to gangrene of the pulp. From the pulp the bacteria may extend to the apical foramen, producing an alveolar abscess with its possible results, such as disfigurement of the face, necrosis of the jaw, spreading suppuration, and even sometimes septicæmia, pyæmia, and death.

The various inflammatory and catarrhal conditions and ulcers about the mouth are to a large extent due to the action of bacteria. Some local or constitutional condition diminishes the vital resistance of a part of the mucous membrane, and so permits of its invasion and destruction by bacteria.

After all large operations on the mouth one of the greatest dangers to be feared is some form of septicæmia, for by such proceedings we bring about conditions very favourable for the entrance of germs into the system—in fact, we imitate very closely the experimental injection of septic saliva into animals, the fatal results of which have been already alluded to. Perhaps at the time of the operation when we make our incisions, germs may enter directly into the circulatory



system. The abundant oozing of serum from the large raw surfaces left by the operation affords additional culture medium for the growth of micro-organisms; the injury inflicted directly on the tissues diminishes their vitality, and thereby their power of resistance to septic infection, and the loss of blood (always serious in a large mouth operation) weakens the whole system and still further renders it a prey to the ravages of invading micro-organisms. Not only after large operations, but after the extraction of teeth and lancing of gums, fatal septicaemia has occurred. Not only may a dirty mouth serve as a source of infection for its owner, but also for other patients operated on with the same instruments imperfectly cleaned. It is well known that syphilis has been inoculated in this way, and, perhaps, other diseases too. It must also be remembered that there is risk to the operator as well as to the patient in the performance of an operation in an unclean mouth. Both local and general infection may follow injuries to the fingers by ragged teeth or dental instruments. Miller has recorded a case of chronic pyaemia in a dentist who injured his finger with a bur whilst cleaning out a decayed tooth. The result was suppuration in the wounded finger, the axilla, the lungs and elsewhere, no less than 135 abscesses forming in the course of two years. The pus contained the *bacillus buccalis septicus*, an organism frequently found in the mouth.

An unclean mouth, coupled with nasal obstruction and a weak state of the general health, are the conditions most fruitful in producing inflammatory conditions of the throat, and (by extension along the Eustachian tube) of the middle ear. The pneumococcus of Fraenkel and the pneumo-bacillus of Friedlander (both of them frequent residents in the mouth)



have been found in otitis media and in cerebral abscesses consequent upon it.

Allusion has already been made to septicæmia following an operation upon the mouth. One of the commonest forms of it is septic pneumonia, produced by the inhalation of septic matter from the mouth. The fact that the pneumococcus of Fraenkel and the micrococcus of sputum septicæmia are almost certainly one and the same, leads one to suppose that many cases of acute lobar pneumonia may be the result of direct infection from the mouth. At first sight this does not seem to harmonise with the well-known fact that pneumonia often follows directly upon exposure to cold winds, immersion in cold water, or some other form of "catching cold." But the depressing influence of cold simply diminishes the vital resistance of the lungs, and so places them at the mercy of any germs which may be inspired from the mouth or outside air.

When we swallow food or saliva we take into our stomachs hosts of germs, many of which are capable of setting up diverse forms of fermentation and so disordering digestion. This fact is sometimes lost sight of by physicians, some of whom treat dyspepsia without inquiring into the condition of the mouth and teeth.

The chief practical lesson to be learnt from a study of the bacteria of the mouth is that the greatest possible care should be taken to thoroughly cleanse all dental instruments *both before and after use.*

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## CHAPTER III.

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WOUNDS.

A wound is a solution of continuity due to mechanical violence. In an *open* wound the skin (or mucous membrane) is divided ; in a *subcutaneous* wound the damaged tissue is not exposed to the air.

Open wounds present the following varieties :—

*Incised wounds* are those made by sharp, cutting, instruments. A minimum of damage is inflicted on the wounded part, and therefore healing is likely to occur without suppuration. Their chief danger is hæmorrhage.

*Contused* and *lacerated wounds* are those inflicted by blunt instruments, machinery, etc. The tissues are more or less bruised and torn, and therefore healing is apt to be attended by suppuration.

*Punctured wounds* are those made by pointed instruments such as needles, bayonets, daggers, etc. A punctured wound is of necessity more or less contused, and being deep and unable to drain efficiently is apt to heal slowly and to be attended by suppuration.

*Poisoned wounds* are those which have been inoculated with some form of virus. The virus may be a chemical one



as in the sting of an insect or a snake bite, or a living one (bacteria) as in a post mortem wound.

### THE HEALING OF WOUNDS.

It was at one time thought that "immediate union" might occur between two wounded surfaces if they were accurately brought into apposition. It is now known that such a process does not occur, and that in whatever way a wound heals, the process of repair is always the result of inflammation. The following are the ways in which a wound may heal.

(a) *By first intention.* The injury causing the wound acts as an irritant and causes just sufficient inflammation for the process of repair, and no more. It is essential that no other source of irritation should keep up the inflammation, for then suppuration would occur. Let us suppose that a deep incision be made in any part of the body and the wound spread open so that we can see what is going on. The immediate result would be hæmorrhage, which would cease after a time owing to the wounded vessels becoming occluded by blood-clot, and by other means which will be more fully described subsequently. In a few hours a moderate degree of inflammation would ensue, the neighbouring vessels would dilate and plastic lymph (liquor sanguinis and white corpuscles) would be exuded both into the tissue itself and upon the wounded surface. As the lymph was exuded so it would coagulate, thus the surface of the wound would become glazed over. Presently serum would begin to ooze out in beads on the glazed surface and run down in little streams to the most dependent part of the wound, like drops of rain on a window. Now let us suppose that instead of our wound being spread open, the

sides of it have been brought into accurate apposition, so that no space is left between them, the result would be that they would be stuck together precisely as two sheets of gummed paper would be. Thus the wounded surfaces are temporarily united, or gummed together by lymph(see Fig.5).

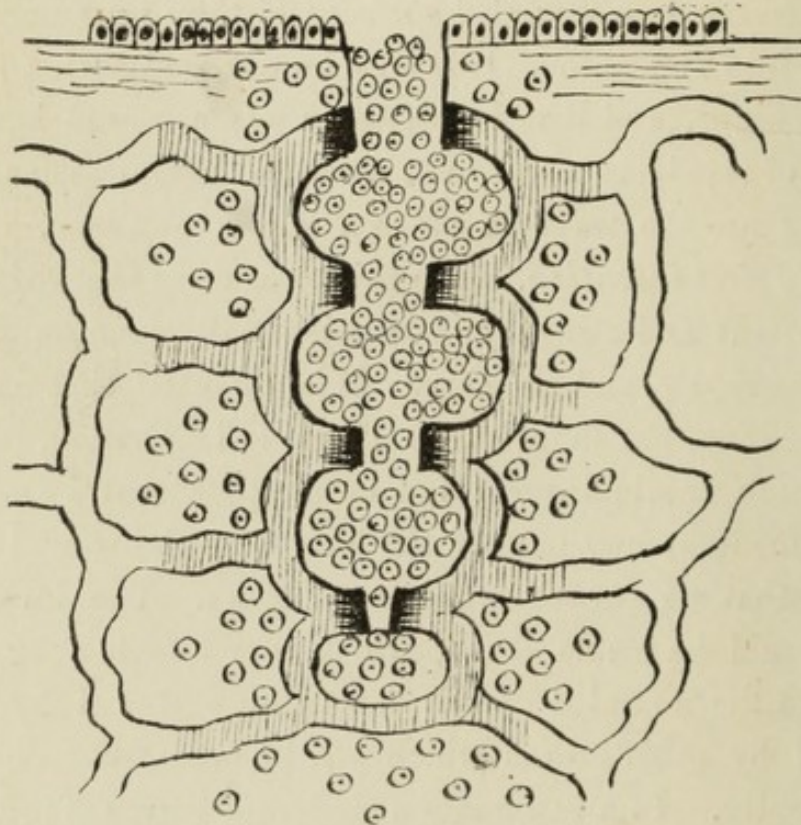


Fig. 5.

#### UNION BY FIRST INTENTION.

The Surfaces of the wound are gummed together by plastic lymph. The divided ends of the capillaries are occluded by blood clot, and the neighbouring tissue is inflamed.

After a few days the neighbouring capillaries send out little buds into the lymph which thus becomes vascular. The lymph is then gradually converted into fibrous tissue. The epithelium from the neighbouring skin or mucous membrane



grows over the surface of the wound, and the healing is complete (see Fig. 6). The resulting scar is at first reddish in colour, but as it contracts the new vessels which it contains become obliterated leaving it pale and non-vascular.

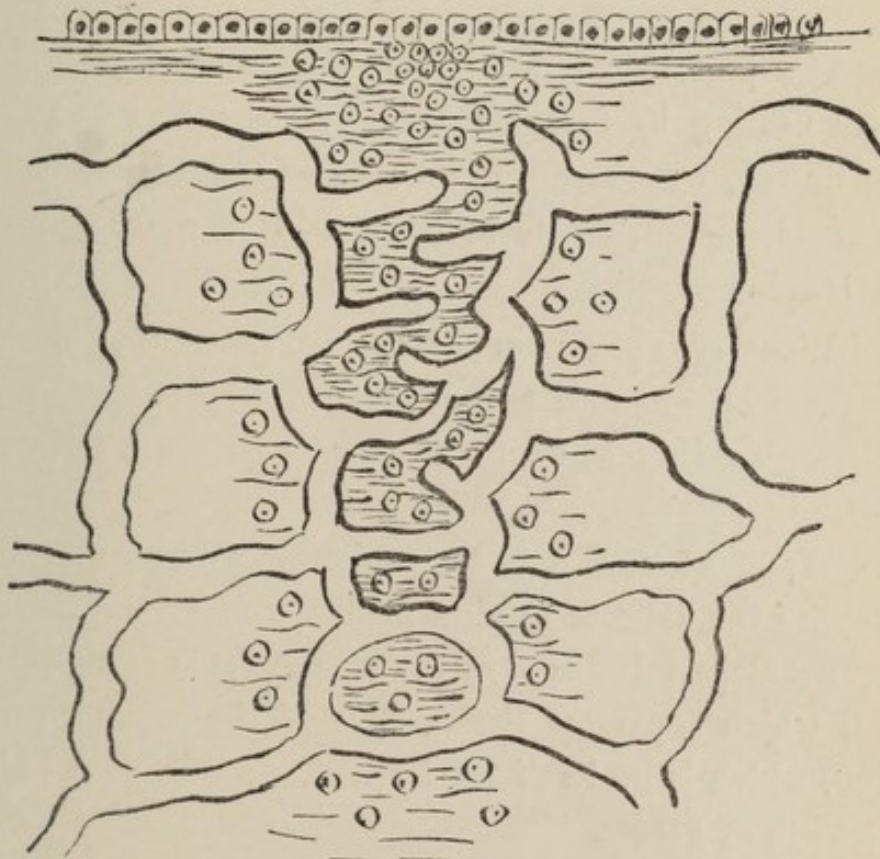


Fig. 6.

#### UNION BY FIRST INTENTION.

The Surfaces of the wound are joined together by newly formed fibrous tissue which has become permeated with capillaries. The epithelium has grown over.

A wound may be prevented from healing by the first intention by the following conditions :—

1. Bruising of the edges of the wound, leading to death

of the adjacent tissue. Dead tissue cannot undergo repair ; it must be cast off as a slough or become absorbed.

2. Constitutional or local conditions producing a state of lowered vitality of the part wounded. A healthy tissue will stand more irritation without suppurating than a diseased one will.

3. The presence of foreign bodies in the wound may prevent healing by first intention by separating the surfaces to be united, by causing irritation, or sometimes, as in the case of blood-clot, by forming a nidus for the growth of bacteria.

4. Want of apposition of the surfaces of the wound. Two pieces of gummed paper will not stick together unless they be placed in contact ; neither will the surfaces of a wound.

5. Want of rest acts by prolonging irritation, and by destroying the delicate uniting medium.

6. Inefficient drainage of the wound leads to retention of serum, which separates the surfaces of the wound, produces tension, which is in itself an irritant, and forms a nidus for the growth of germs.

7. Inoculation of the wound by septic or infective bacteria, the presence of which may prolong the irritation.

(b) *By second intention or by granulation.* As the result of any of the conditions just mentioned, the inflammation may be prolonged, and so many inflammatory cells formed that they die for want of nourishment and form pus. The surface of the wound then comes to resemble precisely the wall of an abscess or the floor of an ulcer, and healing takes place in the manner already described. (See Healing of an Abscess.)



(c) *By third intention or union of granulating surfaces.* When two healthy granulating surfaces are brought into contact they fuse with one another. This often takes place when a large abscess has been opened and its walls allowed to approximate.

(d) *Healing under a Scab* is not a distinct process of repair. The scab simply forms a natural dressing for the wound, and if it happens to be aseptic and non-irritating, the wound underneath it heals by the first intention. If it becomes septic (i.e., invaded by bacteria) suppuration takes place underneath it, the pus lifts it up and escapes at one side, and healing takes place by granulation.

#### TREATMENT OF WOUNDS.

Our object is always to obtain union by first intention when that is possible, therefore our treatment consists in avoiding or combating all those conditions which we know prevent this process from taking place. The chief indications are :—

1. To arrest hæmorrhage. (This subject will be considered subsequently.)
2. To remove all foreign substances by gentle sponging or washing.
3. To accurately approximate the *surfaces* of the wound by means of sutures, strapping or bandages. It is not sufficient to approximate the edges only.
4. To keep the wound at rest.
5. To provide efficient drainage. In most wounds it is only necessary to keep the surfaces in contact and so obliterate the wound cavity by carefully applied pressure. When this cannot be done a drainage tube must be inserted to allow the serum to run off.

6. To exclude bacteria of all sorts. This brief sentence really comprises the whole of antiseptic surgery. Here it is only possible to point out a few general principles. When a wound has already become septic, we must kill the germs it contains by means of antiseptics such as perchloride of mercury and carbolic acid. When we intend to make a wound, as in the performance of an operation, we must so arrange matters as to prevent as far as possible any germs from getting into it. Thus it will be seen that *our antiseptic treatment is directed not to the wound itself, but to the different sources of infection by which it may become contaminated*. Bacteria may be conveyed to a wound by the atmosphere, by the hands of the surgeon or his assistants, by instruments, sponges, dressings, etc., and from the patient's own skin or mucous membrane. Therefore in planning and conducting an operation the following points must be attended to :—

(a.) The room in which the operation is to be performed must be as clean and free from dust as possible. Formerly the carbolic spray was invariably used, it has now been discarded because it has many drawbacks, and because it is now recognized that the air is one of the least important sources of infection. When the operation is concluded antiseptic dressings are applied, one great object of this is to filter and so purify any air which subsequently reaches the wound.

(b.) The hands of the surgeon and of those who assist him must be scrubbed with soap and water and dipped in carbolic acid (1 in 40) or perchloride of mercury (1 in 1000). During the operation the surgeon's hands must not touch anything which has not been purified.

(c.) All instruments must be carefully cleansed before



being put away and again immediately before use. Boiling in water is the most efficient means of purifying them ; after being boiled they should be placed in a solution of carbolic acid (1 in 40). Instruments should not be put in perchloride solution or they will soon lose their polish.

(d.) The skin covering the part to be operated on should be well washed with soap and water, shaved if necessary, and then covered with an antiseptic dressing for some hours before the operation. When the wound is to be made in a mucous membrane, the soap and water must of course be dispensed with, but it should nevertheless be well mopped over with cotton wool soaked in perchloride solution (1 in 1000).

*It should be clearly understood that antiseptic applications do not possess any healing properties, on the contrary they are all irritants and therefore the less they are applied to the surface of the wound itself the better. An ideal antiseptic would kill bacteria without doing any harm to animal tissues; unfortunately no such thing exists.*

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CHAPTER IV.

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## SURGICAL FEVER.

In health the temperature of the body is about 98.6° F. being slightly higher in the evening, and lower in the morning. Heat is generated in the body chiefly by oxidation of the tissues, and is lost chiefly by conduction and radiation from the general surface, and by evaporation. These processes are under the direct control of the nervous system and are constantly being regulated and balanced to suit surrounding circumstances. In many forms of disease the heat-regulating apparatus is disturbed with the result that the temperature rises above the normal, producing *fever* or *pyrexia*.

## THE GENERAL SYMPTOMS OF FEVER

are malaise, shivering, thirst, loss of appetite, constipation, furred tongue, hot dry skin, flushed face, high-coloured scanty urine, rapid pulse and respiration, headache, sleeplessness, delirium, etc. The amount of fever is indicated by the use of the clinical thermometer. It may vary considerably in degree and duration. A temperature of 102° or 103° is usually regarded as "moderate" from 104° to 105° as "high,"



above  $105^{\circ}$  as excessive or hyperpyretic. In rare cases the temperature may rise as high as  $110^{\circ}$  or  $112^{\circ}$ . When the temperature goes up and remains at about the same level it is called "continued," when it falls at intervals (but not to normal) it is called "remittent," when it falls at intervals to the normal or below it, it is called "intermittent."

### THE CAUSES OF FEVER.

At one time it was thought that fever symptomatic of inflammation, was due to the blood becoming heated in the inflamed part; it is now known that the blood in a vein returning from an inflamed part is not hotter than in the corresponding artery. The fever of inflammation is due to two causes:—

(a) The absorption into the blood of some pyrogenous (heat-producing) substance from the inflamed area, which acts as a poison to the heat-regulating apparatus and causes increased production of heat throughout the body.

(b) Disturbance of the heat-regulating apparatus by stimuli passing from the inflamed area through the nervous system.

### VARIETIES OF FEVER.

Several different varieties of fever are met with; they may be most conveniently grouped as follows:—

(i.) *Aseptic Traumatic Fever.* When any severe injury has been inflicted, the inflammation necessary for the process of repair is always accompanied by slight febrile disturbance. The temperature goes up for about 24 or 48 hours, reaching its maximum, (rarely exceeding  $101^{\circ}$ ) on the evening of the second day. It then gradually falls and becomes normal by



the fourth or fifth day, never lasting beyond a week. This is the variety of fever which occurs in wounds which are aseptic, i.e. free from bacteria or their products. The fever is due in part to reflex nervous disturbance, but chiefly to absorption of fibrin ferment from the leucocytes which disintegrate in the process of inflammation. It has been proved by experiment that fibrin ferment is a pyrogenous material and is capable of producing fever by poisoning the heat-regulating centres. No treatment is necessary.

(ii.) *Septic Traumatic Fever* is the result of absorption of chemical poisons produced in a wound by bacteria. The fever does not run a definite course, but remains just so long as the wound is septic. The amount of fever depends partly on the violence of the poisons produced by the bacteria, but also on the condition of the wound itself. Thus if the latter be well drained, the poisonous materials will tend to run out of the wound instead of into the blood stream; if, on the contrary, there be inefficient drainage, the poison being pent up under pressure, is readily absorbed. Again, if the wound be lined by a layer of granulations the absorption is much less, as the granulations offer a considerable barrier to the passage of poisonous substances into the circulation. Hundreds of people are walking about every day with foul suppurating ulcers of the leg, yet very few of them have any increase of temperature. Inasmuch as a septic wound always suppurates, septic traumatic fever is often known as *suppurative fever*. When the quantity of poison absorbed is very large, the patient becomes very ill, the temperature rises to  $103^{\circ}$  or  $104^{\circ}$  and is often accompanied by a severe rigor. In addition to the usual febrile symptoms, nausea and vomiting, diarrhoea, delirium, collapse and death may occur. This severe form



of septic traumatic fever is known as *sapraemia* or *septic intoxication*.

The treatment consists in rendering the wound aseptic by means of antiseptic applications and supporting the patient's strength by fluid nourishment and stimulants.

*Hectic Fever* is of the same nature as septic traumatic fever. It occurs in cases of prolonged suppuration when the pus has undergone decomposition and drains away imperfectly. Small quantities of poisonous chemical substances are continually being absorbed into the system. The result is, that the temperature remains almost constantly above the normal, but with well-marked evening exacerbations and morning remissions. The patient gradually wastes away, and dies unless the suppuration is checked by removing the cause.

(iii.) *Infective Fevers* are the result of the absorption of various sorts of parasitic bacteria into the tissues or blood. In some diseases, such as erysipelas, phagedæna, hospital gangrene, actinomycosis, etc., the infection is local, i.e. the surrounding tissues and lymphatic vessels are infected, but not the general blood stream. In others, such as septicæmia, pyæmia, glanders, hydrophobia, tetanus, etc., the infection is general, the bacteria being found in the blood in parts of the body remote from the original site of infection. We must now consider some of these infective diseases in more detail.

### ERYSIPELAS.

Erysipelas is a diffuse, spreading inflammation of the skin and subcutaneous tissues. Sometimes it affects mucous membranes. Three varieties are described.

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1.—CUTANEOUS ERYSIPELAS

always starts from a wound, although sometimes the latter is so small as to escape detection. It appears as a vivid red blush, which spreads very rapidly. The spreading edge is raised and sharply defined, whilst the receding border fades off gradually into the normal skin. The affected skin is oedematous and shining; when the subcutaneous tissue is loose as in the eyelids and scrotum, there may be considerable swelling. When the blush starts from an obvious wound the latter usually dries up, ceases to heal, and assumes an unhealthy appearance. The neighbouring lymphatic glands usually become tender and enlarged. These local signs are accompanied by a rigor, a rise of temperature to  $103^{\circ}$  or  $104^{\circ}$ , general febrile symptoms and gastro-intestinal disturbance.

*Pathology.*—The disease is caused by a micrococcus called the *streptococcus erysipelatosus*. This organism may be conveyed to the wound by air or water, by the hands of the surgeon or nurse, by instruments, dressings, etc. Having gained admission to the wound, it infects the neighbouring skin by travelling along the lymphatics. The organisms are not found in the blood. Strong healthy people seldom suffer from erysipelas; it occurs most frequently in those who are broken down in health, from Bright's disease, alcoholism, and bad hygienic surroundings.

*Treatment.*—The patient must be isolated lest he infect others. The bowels should be opened by a brisk purge, and perchloride of iron administered in doses of 40 minims every three or four hours. The diet must be liquid but nutritious.



Usually, stimulants are indicated, as the febrile symptoms are apt to assume the asthenic type. The affected part should be dusted over with a powder composed of equal parts of oxide of zinc and starch, and covered over with cotton wool.

## 2.—CELLULO-CUTANEOUS OR PHLEGMONOUS ERYSIPELAS

affects the subcutaneous tissue as well as the skin, resulting in much more swelling. Suppuration is very prone to occur, and large pieces of skin may slough. The constitutional symptoms are apt to assume a very asthenic type, and the patient may die from sapræmia, exhaustion, or hectic.

*Pathology.*—This variety is not nearly so contagious as the cutaneous; it is caused by local bacterial infection, although, as far as is known, there is no one specific germ producing the disease.

*Treatment.*—In addition to that already mentioned under the cutaneous variety, numerous incisions should be made into the swollen parts, and a warm, moist, unirritating antiseptic dressing applied.

## 3.—CELLULAR ERYSIPELAS, OR DIFFUSE CELLULITIS

is a diffuse inflammation of the cellular tissue. It may occur in any situation where there is much cellular tissue. The symptoms, pathology and treatment closely resemble the preceeding variety.

## PHAGEDÆNA.

This is a very rapid form of spreading ulceration, due to local infection with micrococci. It is usually seen complicating venereal ulcers.

When the phagedænic ulceration is accompanied by sloughing it is called *hospital gangrene*, a condition common in former days when hospitals were overcrowded and badly ventilated, but scarcely ever seen in the present day.

### SEPTICÆMIA AND PYÆMIA.

In both these diseases the blood is infected with pathogenic micro-organisms derived from a wound. These two diseases are often indistinguishable clinically, indeed they may occur at the same time in the same patient, and may be complicated by sapræmia or septic intoxication. The chief distinction between them is that in septicæmia the post-mortem appearances are not characteristic, there being only softening and congestion of the internal organs, whereas in pyæmia numerous small collections of pus (*metastatic abscesses*) are found scattered about the body. It is probable that several different kinds of bacteria are capable of producing septicæmia and pyæmia.

*The symptoms of septicæmia* closely resemble those of sapræmia; the temperature rises suddenly to 103° or 104° and is accompanied by a rigor. The general symptoms of fever are well marked, especially the headache, vomiting, delirium and diarrhœa. Collapse and death frequently occurs, but sometimes the symptoms are less severe and recovery takes place. Septicæmia may be distinguished from sapræmia by the discovery of bacteria in the blood, by appropriate bacteriological methods.

*The symptoms of pyæmia* may closely resemble those of septicæmia, but in a typical case the rigors are repeated, and the temperature instead of remaining high, undergoes great



fluctuations. The temperature goes up to  $104^{\circ}$  or higher when the rigors occur, and falls again to near the normal, the fall being accompanied by profuse sweating. The febrile symptoms assume the asthenic type and the patient gets into a low typhoid state. When metastatic abscesses occur they produce symptoms referable to the parts affected such as the liver, lungs, joints, etc. Death nearly always occurs.

Pyæmia is especially prone to complicate septic wounds involving the cancellous tissue of bone, the veins of which do not collapse on section, and so offer a suitable portal for the entrance of bacteria into the blood.

The metastatic abscesses of pyæmia are formed in the following way :—The cut ends of the veins in the septic wound become plugged with clot, and the clot becomes impregnated with bacteria, which grow into it from the wound. The clots usually extend as far as the next largest vein in which circulation is still being carried on, hence small portions of clot are apt to become detached and carried along in the blood stream ; eventually they get impacted in some small artery and there the contained bacteria set up inflammation and suppuration.

*The curative treatment* of septicæmia and pyæmia is very unsatisfactory. Beyond supporting the patient's strength and administering drugs like quinine, salicylic acid, etc., nothing can be done.

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## CHAPTER V.

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SHOCK.

Shock is a condition of depressed vitality due to some profound impression on the nervous system.

The causes of shock are mechanical injuries such as blows upon the abdomen or testicle, extensive burns or scalds, severe and prolonged surgical operations, and powerful mental emotions, such as fright, grief, etc.

In fatal cases of shock the right side of the heart and the large veins, especially those in the abdomen, are found distended with blood, whilst the rest of the body, including the nervous system, is very anæmic. The patient may be said to have bled to death into his own veins. His condition is due partly to vaso-motor dilatation of the abdominal veins, through the splanchnic nerves, and partly to inhibition of the heart through the pneumogastriacs.

The symptoms of shock vary in severity. In the worst cases, the symptoms closely resemble those of severe hæmorrhage. The pulse is rapid and feeble, perhaps imperceptible at the wrist, the respiration feeble, the temperature low, the surface of the body and the extremities cold, the face pale, the lips white, the muscles relaxed, and the skin clammy. The patient may become unconscious and die. When death does not occur, reaction sets in, the temperature



rises to normal or a little higher, the surface becomes warm and the pulse full. If no complications are present the fever of reaction is slight, and soon passes away.

#### TREATMENT.

In severe cases of shock the patient should be put into a bed warmed with hot water bottles. Small doses of brandy should be frequently given, the effect on the pulse being watched. Opium or morphia may also be given with advantage when there is much pain. In some cases transfusion and artificial respiration may be required.

### TRAUMATIC DELIRIUM.

Traumatic delirium is the disturbed mental state that some times follows on severe accidents and surgical operations. Three varieties are usually described.

1. *Inflammatory Traumatic Delirium* is only a symptom of septic traumatic fever (which see).

2. *Alcoholic delirium, or delirium tremens* frequently occurs in persons addicted to alcohol when they have sustained an accident, or have been subjected to an operation. Its onset may be apprehended if the patient cannot sleep and refuses his food. The delirium is of the low muttering kind ; the patient talks away incessantly to imaginary persons, and fancies he sees loathsome animals on his bed and about his room. Sometimes the delirium is of a more violent nature. The temperature is usually slightly raised, the skin moist, the pulse full and soft, the hand shaky, the tongue furred and tremulous, and the bowels confined. The treatment consists in getting the patient to take fluid nourishment, and in procuring sleep by means of chloral and bromide of potassium.

3. *Nervous Traumatic delirium* occurs in highly nervous over-worked people. It closely resembles the preceding variety, but is not due to alcohol.

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## CHAPTER VI.

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### HÆMORRHAGE.

Hæmorrhage is the escape of blood from the blood vessels. The hæmorrhage may take place on the surface of the body, (*external hæmorrhage*), or into some cavity (*internal hæmorrhage*), or into the tissues (*ecchymosis, extravasation*). The blood may come from an artery, from a vein, or from capillaries. In arterial hæmorrhage the blood is bright in colour and escapes per saltum with each beat of the heart; in venous hæmorrhage the blood is dark in colour and escapes in a continuous stream; in capillary hæmorrhage the blood oozes out from a raw surface.

*The constitutional effects* of hæmorrhage vary with the amount of blood lost. When a large quantity of blood is suddenly lost, death from syncope may occur in a few minutes. When the loss is less rapid or smaller in amount, the surface of the body becomes pale and cold, the pulse rapid and feeble, the respiration shallow, and the skin bathed in perspiration, the mind deranged and the sight dim; syncope supervenes, and sometimes ends in convulsions and death.

#### THE NATURAL ARREST OF ARTERIAL HÆMORRHAGE

The means by which nature arrests hæmorrhage may be divided into general and local.



A. *General means.* The greater the loss of blood, the more feeble is the action of the heart, and therefore the less the force pumping the blood out of the wounded vessel. Another result of hæmorrhage is that the blood becomes more coagulable and therefore more apt to occlude the wounded vessel.

B. *Local means.* These are subdivided into temporary and permanent.

(a) The wounded vessel becomes *temporarily* occluded by *contraction* and *retraction* of the cut end, and by the *formation of clot*. The contraction, or narrowing of the cut end is due to the action of the circular muscular fibres in the middle coat of the artery. Retraction takes place because an artery in its normal condition is elastic and slightly on the stretch. When a vessel has been partially divided, retraction and contraction cannot occur, consequently the bleeding may be prolonged until syncope occurs. In a lacerated wound the end of the artery becomes so twisted and curled up that its lumen may be completely occluded. It is not uncommon to see a limb torn off in a machinery accident with practically no loss of blood.

Blood in a healthy vessel does not clot, because the fibrin ferment (which is essential for the process of clotting), is locked up in white corpuscles. When the blood is spilt or comes in contact with the air or any rough surface, the white corpuscles break up and allow the fibrin ferment to mix with the plasma and produce fibrin. When a vessel is wounded, a clot forms inside the cut end, and in the tissues immediately around it, plugging up the opening like a piece of putty used to stop a leak in a damaged box. In the condition known as *hæmophilia* the blood refuses to clot, consequently a very

small wound may go on bleeding for an indefinite period, and may cause death. Persons affected in this way are called *bleeders*.

(b) A wounded artery is permanently sealed by adhesive inflammation of the cut end, in precisely the same way as a wound of any other soft tissue. Plastic lymph is poured out by the vasa vasorum of the wounded artery, infiltrates the base of the internal clot, and gradually develops into fibrous tissue. The internal clot is often said to become organised, this is not strictly correct; in reality it becomes infiltrated and destroyed by leucocytes, the original clot, which is only a temporary plug, taking no part in the formation of the fibrous tissue which permanently seals the artery.

Nature's means of arrest of hæmorrhage may be thus summarized:—

Temporary	{	General	{ Diminished force of heart beat Increased coagulability of blood
		Local	{ Contraction Retraction Coagulation

Permanent—Plastic Inflammation and obliteration of wounded vessel by fibrous tissue.

The natural arrest of venous and capillary hæmorrhage differs from arterial only in that contraction and retraction do not take place.

#### ARTIFICIAL MEANS OF ARRESTING HÆMORRHAGE.

It must be clearly understood that hæmorrhage can be only temporarily arrested by artificial means. A wounded vessel can only be permanently occluded by the process of plastic



inflammation as already described. The following are the various means used to arrest hæmorrhage.

1. *Pressure* may be applied by means of the finger, Spencer Wells forceps, the tourniquet, a plug of lint, or graduated compress. The pressure should be applied on the bleeding point ; when this is not possible it must be applied between the wound and the heart when the bleeding is arterial, and on the distal side of the wound when it is venous. The object of pressure is to close the wound whilst clotting takes place; it should not be maintained for much longer than twelve hours, lest sloughing result. Pressure is relied upon chiefly when the bleeding vessel can be effectually compressed against bone, as in cases of bleeding from the socket of a tooth, or from a wounded vessel in the scalp.

2. *Heat* is often used in the form of hot water (a little hotter than the hand can bear). It causes contraction of the muscular fibres of the small arteries.

3. *Cold* also causes contraction, and is often used in hæmorrhage from mucous membranes.

4. *Styptics* are drugs such as perchloride of iron, tannin, matico, etc., which cause coagulation. They should be avoided as much as possible.

5. The *Cautery* acts by contracting the vessels, by causing coagulation, or by charring the tissues. It may be used in the shape of an ordinary red hot iron, the Pacquelin benzoline cautery, or the galvano-cautery.

6. The *Ligature* is the means most frequently used for securing vessels which are too large to be stopped by pressure. The ligature is applied by seizing the bleeding point in a suitable pair of forceps and tying a reef knot round it. The material generally used for ligatures is catgut or silk, which



has been rendered aseptic by special means of preparation. When a ligature is firmly applied around a vessel, it divides the internal and middle coats without affecting the outer coat. The divided coats curl up, clot forms on them, and plastic inflammation ensuing, the vessel becomes obliterated. If the ligature is composed of aseptic catgut it soon becomes absorbed ; if of aseptic silk it becomes at first encapsuled in fibrous tissue and then very slowly absorbed.

7. *Torsion* consists in seizing the end of the vessel and twisting it until the coats curl up sufficiently to occlude the orifice.

#### CONSTITUTIONAL TREATMENT OF HÆMORRHAGE.

In cases of slight hæmorrhage, no constitutional treatment is needed, but when the bleeding is furious and sudden, we must be on the look-out to prevent the occurrence of fatal syncope, by keeping the patient's head low and raising the limbs to ensure a sufficient supply of blood to the brain ; if this is not followed by the desired result alcohol must be administered by the mouth, or rectum, or ether by hypodermic injection. In cases of internal hæmorrhage where no direct means can be used to stop the bleeding, stimulants are inadmissible unless death actually threatens, because the increased action of the heart may displace clots which are occluding the bleeding vessels. The intravenous injection of saline solution (salt ʒi warm water Oi) is sometimes of service by increasing the blood-pressure sufficiently to allow the patient to rally. The remote effects of hæmorrhage must be treated by good nourishment, tonics, especially iron, and sea-air.



## RECURRENT OR REACTIONARY HÆMORRHAGE

takes place during the period of reaction and is due to failure of the temporary means (natural or artificial) of arrest. It sometimes happens that during an operation the action of the heart becomes so weak that many of the smaller wounded vessels do not bleed and consequently are not ligatured. When the patient gets warm in bed and begins to rally from the shock of the operation, the heart beats more strongly, the contraction of the muscular coat of the small vessels passes off and recurrent hæmorrhage is the result. This usually occurs within twenty-four hours of the operation.

## SECONDARY HÆMORRHAGE

may occur at any period until the wound is healed. It is due to failure of the permanent means of arrest of hæmorrhage, or in other words anything preventing the efficient sealing of the vessel by plastic inflammation may cause secondary hæmorrhage. The causes of secondary hæmorrhage may therefore be included under the following heads :—

(1). *Any constitutional condition*, such as Bright's disease, diabetes, septicæmia, etc., which lowers the vitality of the tissues and prevents them from healing properly.

(2). *Suppuration and Sloughing in the Wound*. It is obvious that if these processes involve the wounded artery, healing will not take place, and hæmorrhage will result.

(3). *Disease of the Vessel*. Secondary hæmorrhage is very apt to occur when there is extensive calcareous degeneration of the vessel wall, the latter being very little more than a

tube of chalk. Under such conditions plastic inflammation will not take place.

(4). *Defect in the ligature.* The ligature may become absorbed before the artery is permanently sealed, or it may be septic and set up suppuration instead of adhesive inflammation, or it may have caused so much damage to the vessel wall as to lead to its death, or it may be applied so near to a large branch that the wounded vessel is deprived of the rest necessary for its repair.

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## CHAPTER VII.

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### FRACTURES.

A fracture is a sudden solution of continuity of a bone.

THE CAUSES OF FRACTURE are

1. *Predisposing*, such as atrophy, degeneration, syphilitic or tubercular inflammation, new growth, or any other cause rendering the bone more fragile than usual.

2. *Exciting Causes*, which include external violence and muscular action.

(a) External violence may be either direct or indirect, in



the former the bone being broken at the place where the violence is applied, and in the latter at some remote spot, *e.g.* the neck of the thigh bone being broken by a twist of the foot.

(b) Muscular action, when sudden and very forcible, may cause fracture. This is most commonly seen in the patella. When a bone is much weakened by disease, an ordinary muscular effort may suffice to break it, such fractures are called *spontaneous*.

#### VARIETIES OF FRACTURE.

*A simple fracture* is one in which the soft parts covering the bone are not torn through.

*A compound fracture* is one in which there is a wound in the soft parts *which permits of air reaching the broken ends*.

*A complicated fracture* is one which is associated with damage to some adjacent part, such as a joint, or large artery. Fractures are also divided,

1. According to their extent into :—

- (a.) Complete,
- (b.) Incomplete,
- (c.) Comminuted—broken in several small pieces.
- (d.) Multiple—two or more fractures in the same bone or in different bones.

2. According to the direction of the line of fracture into,

- (a) Transverse,
- (b) Oblique,
- (c) Longitudinal,
- (d) Spiral,
- (e) Stellate.

3. According to the condition of the fragments into,
  - (a) Impacted—one fragment driven into the other,
  - (b) Fissured—a mere crack without displacement,
  - (c) Depressed—the broken part depressed below the surrounding bone.
  - (d) Punctured,—made by pointed weapons,
  - (e) Splintered—a small piece chipped off.

#### THE SYMPTOMS OF FRACTURE.

1. The patient feels, and sometimes hears, a sudden snap.
2. Pain.
3. Inability to use the injured part.
4. Deformity.—This is due to displacement of the fragments, which may be brought about by (a) the direction of the force producing the fracture, (b) muscular action, and (c) the weight of the part. When the fracture occurs in a limb the displacement of the fragments often produces shortening.
5. Preternatural mobility.
6. Crepitus is a grating sensation detected by the surgeon gently moving the fragments against one another.

#### DIAGNOSIS OF FRACTURE.

The only sure sign of fracture is preternatural mobility, but it is not always present, *e.g.*, in impacted fracture. Each of the other signs may be at times absent, or when present, may be due to some other cause, thus pain, sensation of snapping and inability to use the injured part, may be due to damage of muscles or ligaments; deformity may be due to dislocation; and crepitus



may be detected in joint disease, extravasation of blood or air into the subcutaneous tissues, and in inflammation of the sheaths of tendons.

### THE UNION OF FRACTURES.

The union of a simple fracture is essentially the same process as the healing of a wound by first intention. The immediate result of the fracture is extravasation of blood

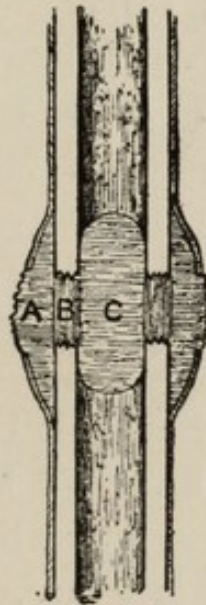


Fig. 7.

Diagrammatic sketch of repair of simple Fracture.

- A. External or Ensheathing Callus. B. Permanent or Definite Callus.  
C. Internal Callus.

around the broken ends ; this gets gradually absorbed, and takes no part in the healing process. Very soon after the injury traumatic inflammation supervenes, and plastic lymph exudes from the inflamed bone, medulla, periosteum and neighbouring tissues. The lymph, becoming vascular, forms a mass of granulation tissue surrounding the broken ends of

the bone, and is known as *callus*. That portion of it outside the broken ends is called external or ensheathing callus, that inside the medullary cavity internal callus, and that actually between the broken ends permanent or definitive callus. The internal and external callus is ultimately absorbed, and is therefore often called temporary, (see Fig. 7.) The callus becomes converted into fibrous tissue. The outer layer of the ensheathing callus forms the new periosteum, and from its deeper aspect, and from the angle between the separated periosteum and the bone, the process of ossification invades the fibrous callus, converting it into a mass of bone, fixing the broken ends together. In animals, and in some fractures in man, the callus is converted into fibro-cartilage before being ossified.

The union of a compound fracture may take place in two ways, (a), the wound over the fracture may heal by first intention, thus converting it into a simple fracture, or (b), suppuration may occur, granulation tissue being formed around the ends of the bone and healing taking place by granulation, or by the second intention. The fibrous tissue thus formed is converted into bone.

#### NON-UNION.

It sometimes happens that the process of union of a fracture remains incomplete or fails entirely. In certain fractures, chiefly those involving joints (such as fracture of the patella), and those where a bony prominence is knocked off, and dragged by muscular action some distance from its proper place union by fibrous tissue only is the rule, and bony union the exception. When the un-united fragments remain in contact, their ends may become rounded off and enclosed in a sort of



fibrous capsule forming the condition known as *false joint*, or *pseudarthrosis*.

The causes of non-union are both constitutional and local.

The constitutional causes are conditions such as scurvy, nephritis, syphilis, gout, old age, which diminish the vitality of the tissues or impede the process of repair.

The local causes are (a) want of rest, (b) want of apposition from muscular contraction, the loss of a large piece of bone in a compound fracture, or from the interposition of a piece of muscle, tendon, or other foreign substance, (c) necrosis of the broken ends, (d) deficient blood supply.

#### TREATMENT OF FRACTURES.

A. Treatment of simple fractures. The following are the indications:—

1. To place the fragments in accurate apposition so that they may have a fair chance of uniting, and may be, when united, the same shape as the original bone. This is called reducing or setting the fracture.

2. To keep the fragments in accurate apposition by means of splints or other devices, until firm union has occurred.

3. When union has occurred to restore the functions of the damaged part.

B. Treatment of compound fractures. When the wound is small and aseptic, it should be closed as soon as possible, so as to convert the fracture into a simple one. When the wound is large it must be treated on the general principles already laid down in speaking of the treatment of wound

and the fracture must be fixed in some form of apparatus which will allow of the necessary dressings being applied to the wound with the least possible disturbance of the fragments. In the case of a severe compound fracture of a limb, amputation may be required, when (a) there is great damage to the soft parts, (b) when the bone is extremely comminuted, (c) when the main vessels and nerves are torn, (d) when a large joint is involved, and (e) when the patient is old or broken down in health.

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## CHAPTER VIII.

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### SYPHILIS.

Syphilis is a constitutional disease which may be inherited by children from their parents, or may be acquired by local inoculation from another person afflicted with the disease.

#### ACQUIRED SYPHILIS.

The site of inoculation is usually the genital organs. The disease first makes its appearance about three to five weeks after the exposure to contagion. In the male the *primary sore* is most commonly found just behind



the corona glandis, in the female inside the labium. The most typical variety of primary sore is the *Hunterian chancre*; this begins as a small papule which develops into a definite raised nodule with a flattened, glazed and sometimes excoriated surface, and a densely indurated base, so that when grasped between the fingers it feels like a piece of gristle. The development of the primary sore is the result of implantation of the syphilitic virus. The nature of this virus is not at present exactly known, but it is almost certainly some form of micro-organism. Other kinds of sores frequently occur on the genital organs as the result of impure connection; they are called *chancreoids* or soft sores, and have nothing whatever to do with syphilis.

The primary sore of syphilis is not by any means always found on the genital organs. There is scarcely a spot on the body on which such a sore has not been occasionally seen, but the commonest situations for these so-called *erratic chancres* are the lips, the nipple, and the finger of the accoucheur. They are also met with on the tongue and tonsils. A chancre has been known to occur on the gum after extraction of a tooth with infected forceps.

The primary sore will not have lasted long before the poison extends along the lymphatic vessels to the nearest lymphatic glands (usually, of course, those in the groin). These become enlarged and indurated, but remain separate from one another, and free from pain or tenderness. They are usually described as feeling amygdaloid or shotty. So far, the poison has not gained admission to the general circulation, and the infection is local; within, however, about six weeks to two or three months, the infection becomes general,



and results in the appearance of so-called secondary manifestations.

*Secondary Syphilis.* The virus is now at work in the blood, and produces various affections of the skin, mucous membranes, and other structures. The earliest skin affection is a *roseola* over the chest and abdomen; this eruption is scarcely more than a mottling of the skin, it is due to congestion of the surface capillaries and fades on pressure. Roseola is often succeeded by a scaly eruption called syphilitic *psoriasis*; this occurs on the face, chest, and flexor aspects of the limbs; it must be distinguished from ordinary psoriasis, which is much more scaly, and occurs chiefly on the extensor aspects. *Condylomata* occur in situations where the skin is moist, as about the mouth and anus. They are small flattened elevations of a pale rose, or whitish colour, and moist shining aspect, about the size of a split pea, or larger; they are very contagious and infect neighbouring portions of skin with which they come in contact. When situated between the toes they are prone to ulcerate, producing *rhagades*. In some cases of secondary syphilis one sees *papular* eruptions (small pimples), and *vesicular* eruptions (tiny blisters). The contents of the vesicles may become purulent; they are then called *pustules*. The larger pustules may dry up, and form scabs, beneath which ulceration may occur; as the ulceration extends, larger and larger scabs are formed, producing a graduated crust resembling a limpet shell. This condition, (which is called *rupia*) occurs in the later stage of secondary syphilis in those who are broken down in health.

All secondary eruptions have the following characters in common, viz., they are more or less the colour



of copper or lean ham ; they are arranged symmetrically on the two halves of the body, and they are often polymorphic, *i.e.*, several varieties co-exist. The commonest secondary syphilitic affection of mucous membrane is the *mucous tubercle*. These tubercles appear as slightly elevated whitish patches, and are most commonly found on the tongue, cheek, lips, palate and tonsils. They consist of sodden epithe-

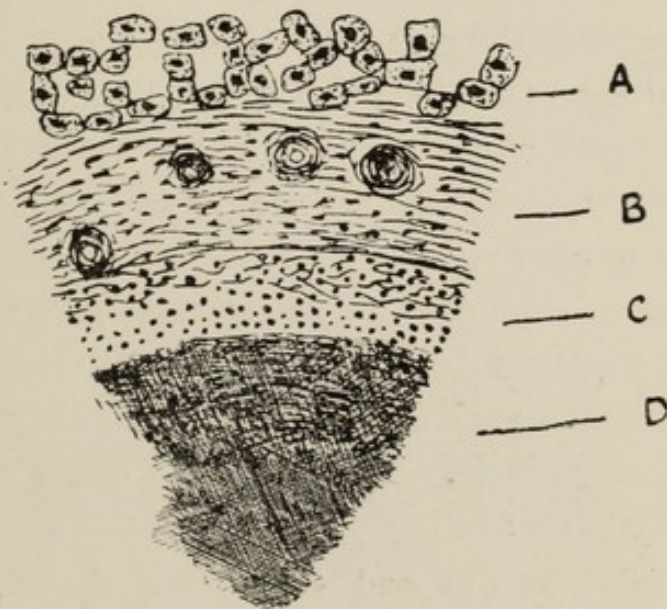


Fig. 8.

#### GUMMA OF LIVER.

A.—Normal Liver Tissue. B.—Leucocytes containing small vessels with thickened walls and narrowed lumen. C.—Leucocytes in reticulated matrix. D.—Caseous structureless material. Mag. 350 diam.  
(Drawn from nature by A. K. Gordon).

lium infiltrated with inflammatory cells. Portions of the patches are prone to die, producing superficial ulcers.

During the later stages of secondary syphilis, other parts are not unfrequently affected ; the lymphatic glands all over the body, especially those at the back of the neck, become

enlarged, and the bones, joints, eye and testicle may become inflamed.

*Tertiary Syphilis.* Under this title are included a number of lesions which may occur at any time after the end of the secondary stage. Usually they do not appear within a year of the primary infection, but sometimes may occur early enough to overlap the secondary stage. They may not occur until many years have elapsed. The essential lesion of tertiary syphilis is the *gumma*. Gummata are composed of granulation tissue (*i.e.*, inflammatory cells). The cells in the centre of the gumma undergo fatty degeneration from want of blood supply, and may become converted into a caseous mass (see Fig. 8). Frequently the whole gumma dies, forming a slough resembling a piece of wet wash-leather, and which on separation leaves a deep ulcer. Gummata may vary in size from a pea to an orange or even larger, and may be situated in any part of the body. They are most commonly found in the following parts :—

In the subcutaneous and submucous tissues, producing ill-defined, roughly spherical lumps, which have a great tendency to break down and form deep circular ulcers.

In the periosteum, producing *nodes*.

In bones, leading to caries and necrosis.

In muscles, producing lumps, which are apt to be mistaken for tumours.

In the skin, producing little lumps (*tubercular syphilide*), and terminating in a creeping (*serpiginous*) form of ulceration.

In the tongue, producing scars and furrows, sometimes terminating in cancer.



In the brain, spinal cord, and nerves, producing paralysis and other nervous phenomena.

In the arteries, causing aneurism, etc.

In the viscera (liver, lungs, etc.), producing many different symptoms.

All gummatous affections have the following characters in common:—they are chronic in their course; they have no tendency to heal; they are prone to break down; they are not contagious; they are usually not arranged symmetrically.

*Treatment.* The primary sore should be kept clean by an application of black wash, and protected from irritation. No other local treatment is of any use. During the primary and secondary stages mercury must be administered either by the mouth, by inunction, by fumigation or by subcutaneous injection. In most cases the best way to give mercury is in the form of pil. hydrarg. cum creta, in doses of  $2\frac{1}{2}$  grains three times a day. The course of mercury should extend over six months, or longer if the secondary manifestations have not disappeared in that time. When a patient is fully under the influence of mercury, the edges of the gums are slightly reddened and there is slight tenderness on biting. If the drug be pushed further, profuse salivation and even necrosis of the jaw may ensue; should salivation occur, the drug must be withheld, a slight purge administered and the mouth rinsed out frequently with a 1 per cent. solution of chlorate of potash. Condylomata should be dusted over with a powder composed of equal parts of calomel and oxide of zinc. Mucous tubercles about the mouth may be treated with black wash or a 2 per cent. solution of chromic acid. In the tertiary stage, iodide of potassium in doses of from 5 to 15 grains three times a day



should be administered. Gummatous ulcers should be dressed with an antiseptic application such as iodoform, red oxide of mercury ointment, &c.

#### CONGENITAL SYPHILIS.

Syphilis may be inherited either from the mother or from the father. In the former case, the ovum may be syphilitic when impregnation occurs, or may become infected subsequently through the placental circulation ; in the latter case the ovum becomes syphilised by the spermatozoon at the time of impregnation. Infants may acquire syphilis by coming in contact with infecting sores during birth, but syphilis acquired in this way is not strictly speaking congenital. The child usually appears quite healthy when born, and remains so until it is four or five weeks old. The symptoms which occur then are a catarrhal condition of the nose (*snuffles*), a rash over the body and mucous tubercles about the mouth and anus. The rash is best marked about the nates and genital organs, where it is usually erythematous in character. It may, however, closely resemble any of the rashes of acquired secondary syphilis. In most children the general nutrition suffers very much, so that they become wizened and shrunken, looking more like monkeys than human beings. The wasting (*marasmus*) often ends in death. Under treatment all secondary phenomena usually disappear within a year. In some cases no further symptoms occur, but in many of them after a longer or shorter time, tertiary affections appear ; the most important of these are interstitial keratitis, inflammatory affections of bone and periosteum, chronic synovitis of joints, gummata, ulceration of the palate, and deafness. In a marked case of congenital syphilis some, or all, of the following appearances may be



present: 1. The bridge of the nose is depressed and broad, the result of the infantile catarrh. 2. There are radiating scars about the angle of the mouth, the result of previous ulceration. 3. The skull is large and either surmounted by natiform or hot-cross-bun-like swellings (*Parrot's nodes*) or extremely thinned in places (*craniotabes*); the forehead is high and square. 4. The permanent upper central incisors are dwarfed and narrowed towards the cutting edge, which presents a well-marked crescentic notch. 5. The stature is stunted.

*Treatment.* In the early stages, mercury should be administered by inunction, half a drachm of unguentum hydrargyri being put on a flannel binder and fastened around the child's waist every day. The feeding must be carefully attended to, and if the mother cannot suckle the child, it must be brought up by hand. The services of a wet nurse are inadmissible, as she would run great risk of acquiring a chancre on the nipple. It sometimes happens that a mother who has given birth to a syphilitic child shows no signs of syphilis herself; such a one may suckle her child without risk of becoming infected. This is known as Colles' Law. The results of treatment of congenital syphilis by mercury are usually most satisfactory. In many cases no tertiary phenomena ensue. When these do occur they should be treated in the same way as in the acquired disease.

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CHAPTER IX.

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## TUBERCULOSIS.

Tuberculosis results from infection by a micro-organism called the tubercle bacillus. The essential lesion in tuberculosis is the development of tubercles either in a single organ or tissue (local tuberculosis) or throughout the body (general tuberculosis). The bacillus of tuberculosis may enter the body by means of (a) *the digestive tract*; by eating imperfectly cooked tuberculous meat, by drinking milk from tuberculous cows; and by direct infection from the sputum of phthisical persons. (b) *By the respiratory tract*; by inhaling dust impregnated with dried phthisical sputum. (c) *By the skin* by direct inoculation. It appears that tubercle is only rarely inoculated in this way and that even when inoculated it remains local and does not affect the general system. It is quite possible that the bacillus may affect the glands of the neck by having gained admission through a carious tooth.

In tuberculosis, as in all other microparasitic diseases, the vitality of the host must be lowered before the bacillus can gain a footing. The chief *predisposing causes* producing the necessary condition of lowered vitality are :—

1. *The strumous diathesis.* This name is used to designate a general state of nutrition or constitution which is peculiarly suitable for the development of tuberculosis. Two



distinct types are usually described; in the first known as the *sanguineous* or *serous*, the skin is fair and thin, the hair blonde or auburn, the features delicate, the eyes blue, the lashes long and silken, the intellect active and often precocious; in the second known as the *melancholic* or *phlegmatic* the general aspect is dull and heavy, the skin is thick, muddy and harsh, and the mind sluggish. In strumous subjects slight injuries are prone to produce inflammatory conditions characterized by great chronicity, feeble power of repair and tendency to caseation of the inflammatory products. By some pathologists these "strumous" inflammations are thought to be the result of actual deposit of tubercle and not of the general state of nutrition. Be this as it may, it is quite certain that some people (usually called strumous) although not actually tubercular are very prone to become so. It should be added that some authorities use the terms "struma" and "tuberculosis" as identical. This custom is to be regretted as it leads to confusion. It is better to apply the term "tubercular" to those who are actually afflicted with tuberculosis, and "strumous" to those who, although not actually afflicted, are in a condition of general health which predisposes to it. The term "scrofulous" largely used by the older writers is now used synonymously with "strumous" and had better be entirely discarded. The manner in which the strumous diathesis is produced is not fully understood; but it appears that it is often inherited from tubercular parents, and is apt to occur in the children of very young or very old, or dyspeptic or perhaps syphilitic parents.

2. *Bad hygienic surroundings.* Such as poor food, damp, imperfect ventilation, etc.

3. *Youth.* Tuberculosis, although occasionally met with



in old people, is almost entirely a disease of children and young adults.

4. *Injury.* The development of tubercular disease in a bone or joint is often determined by some slight injury which in a healthy person would produce no permanent evil result.

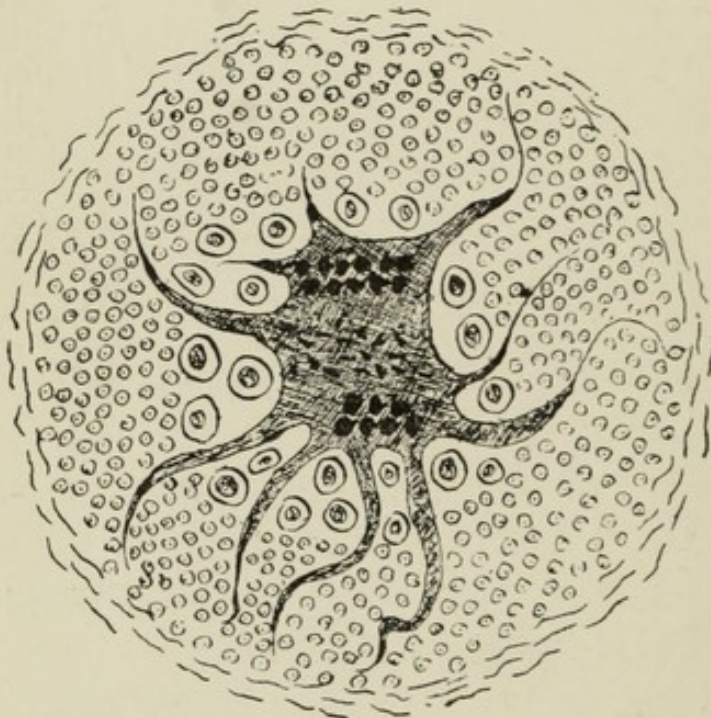


Fig. 9.

DIAGRAM OF A TUBERCLE.

In the centre is a giant cell containing tubercle bacilli and many nuclei. Around this are several endotheioid cells, outside which again are numerous leucocytes.

*Structure of a Tubercle.* Two varieties of tubercle are usually described, viz., the *grey miliary tubercle* and the *yellow or caseous tubercle*, the latter, however, is not a distinct variety, but is simply a degenerated caseated mass of miliary tubercle.



The miliary tubercle is globular in shape, varying in size from a pin's head to millet or hemp seed, and closely resembling a tiny nodule of cartilage in appearance, being hard and semi-translucent. When examined under the microscope it is found to consist of a central giant cell containing many nuclei; from the giant cell processes radiate out forming a reticulum or network in which are enclosed a number of cells. Those in the immediate vicinity of the giant cell resemble endothelial cells and are called "endothelioid"; the others are simply leucocytes, (see Fig. 9). A tubercle contains no blood vessels, and is consequently very prone to undergo caseous degeneration. By appropriate methods of staining, the tubercle bacilli can be demonstrated in the giant cell and the surrounding endothelioid cells. Various explanations of the structure of tubercle have been given by different authorities at different times. It seems probable that the cluster of cells which constitute a tubercle are really phagocytic in nature, their aggregation constituting an attack upon the invading bacilli. The giant cells and endothelioid cells are probably of connective tissue origin, and the small round cells are derived from the blood.

There is scarcely any part of the body which is not at times affected with tuberculosis, but there are certain localities which are especially prone to be affected; these are the lungs, bones, joints, serous membranes and lymphatic glands.

The presence of tubercle in a tissue always sets up a certain amount of inflammatory reaction, in some cases, with the fortunate result that the tubercular deposit becomes encysted in a fibrous capsule, and is so rendered practically inert; it may then undergo caseation, and subsequent calcification. In other cases, the caseated tubercle may soften and

form the focus of a chronic abscess, the necessary result being severe damage to the tissues ; thus in bone disease caries usually results, in phthisis (pulmonary tuberculosis) the lungs become hollowed out with large suppurating cavities, etc.

*Symptoms.* The symptoms of tuberculosis are necessarily extremely varied, as any organ or tissue in the body may be affected. It may be stated generally, that in the early stage of a tubercular lesion, the symptoms are chiefly those of irritation, and that in the later stages they are the result of destruction of tissue.

*Treatment.* The general principles of treatment of tubercular lesions are (*a*) to improve the general health in every possible way, so as to increase the resisting power of the tissues, (*b*) to provide mechanical and physiological rest for the affected part, and (*c*) to remove the diseased parts, when they are beyond the reach of natural repair, by excision, amputation, evacuation of abscesses, scraping, etc.

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CHAPTER X.

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## TUMOURS.

The term "tumour" in its widest sense denotes a swelling, no matter what the cause or nature of it may be : thus an abscess, a gumma, a caseous mass, an effusion of blood, a distended bladder, a displaced bone, are all tumours in this sense. When speaking pathologically, it is customary to limit the term tumour to designate only those swellings which are the result of abnormal growth of tissue and which have no tendency to undergo spontaneous cure or to dwindle under the influence of drugs.

The elements of which a tumour is composed resemble those found in the healthy tissues either in their fully developed condition or in their immature stage of development. Tumours are supposed to arise by the multiplication of pre-existing cells, and to retain the type of the cells from which they sprang. Very little is known of the causes which give rise to the growth of tumours ; they not unfrequently follow upon prolonged irritation, chronic inflammation, or injury, but the exact relation of these conditions to tumour formation is not fully understood. In former years, tumours, especially cancer, were supposed to be hereditary, but now, the idea has been almost entirely given up and a local cause is favoured. During the last few years some pathologists

have endeavoured to prove that many tumours, especially the more malignant varieties are the result of the action of a microscopic animal parasite to which the name *psorosperm* has been given. This view is not at present generally accepted.

*Clinical Course.* The clinical characters which different kinds of tumours present have led to their being divided into *Innocent* and *Malignant*. <sup>*Benign*</sup> Innocent tumours grow slowly ; they resemble in structure fully formed tissues such as fat, cartilage, bone, etc., and usually are composed of the same kind of cells as the tissue in which they occur (*homologous*). They are enclosed, as a rule, in a fibrous capsule, they are well defined and circumscribed, and simply push neighbouring parts on one side without infiltrating them. They do not involve the lymphatic glands, they do not become disseminated in distant organs, and they do not grow again after they have been completely removed. An innocent tumour may nevertheless cause death by pressing upon some vital organ. Malignant tumours grow rapidly ; they do not resemble the tissue in which they grow (*heterologous*), being usually composed of immature cells like those of the embryo; they tend to infiltrate neighbouring structures rather than to push them aside ; they often lead to secondary deposits in the neighbouring lymphatic glands, and become disseminated throughout the body by means of the blood or lymph stream. <sup>*Sarcomata*</sup> They often recur after removal, either in the scar, the lymphatic glands, or the internal organs. They cause death by exhaustion due to pain, hæmorrhage, general interference with nutrition from the rapid growth of the tumour and its dissemination throughout the body, or by direct interference with a vital part. <sup>*Carcinoma*</sup>



*Classification.* Tumours may be classified in different ways ; it is most convenient to classify them according to their structure, thus :—

### TUMOURS.

#### A. CONNECTIVE TISSUE TUMOURS.

I. Those composed of fully-formed connective tissue. (They are clinically innocent.)

1. Fibroma.
2. Lipoma.
3. Enchondroma.
4. Osteoma.
5. Myxoma.
6. Myoma.
7. Neuroma.
8. Angelioma.
9. Lymphangioma.
10. Lymphoma.

II. Those composed of embryonic connective tissue  
(They are clinically malignant.)

*Sarcoma.*

#### B. EPITHELIAL TUMOURS.

I. Those composed of fully formed epithelial tissue.  
(They are clinically innocent).

1. Papilloma.
2. Adenoma.

II. Those composed of embryonic epithelial tissue.  
(They are clinically malignant.)

*Carcinoma.*

1. Squamous-celled.
2. Spheroidal-celled.
3. Cylindrical-celled.

## FIBROMA.

Fibromata are composed of fibrous tissue ; some of them are hard, and are composed of dense bundles of fibres closely interwoven (see Fig. 10), others are soft, and are composed of looser fibres containing a number of cells and spaces.



Fig. 10.—FIBROMA.

The soft fibromata grow from the subcutaneous connective tissue and form pedunculated tumours ; the hard fibromata grow from the periosteum of bone, especially the jaws. They grow slowly, and are painless, unless they happen to involve a nerve ; they are globular, smooth, and, as a rule, freely moveable. They should be removed by operation when practicable. They do not recur.

## LIPOMA.

Lipomata are composed of fat (see Fig. 11) ; they occur in two forms—the circumscribed and the diffuse ; the former being quite common, and the latter decidedly rare.

The circumscribed lipomata are enclosed in a fibrous capsule which is usually adherent to the skin and but loosely



attached to the tumour. They grow slowly, are lobulated, freely moveable on the deeper parts, but cause a dimpling of the skin when the latter is pinched up over them. They are most often seen in the subcutaneous fat in the region of the shoulder and buttock.

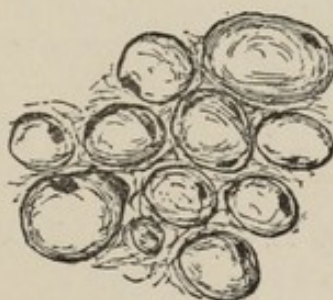


Fig. 11.—LIPOMA.

The diffuse lipomata are not surrounded by a capsule, but are directly continuous with the fat in which they grow. They are usually found at the back of the neck and under the chin in stout middle-aged men.

The circumscribed variety should be removed; they shell out of their capsules readily. The diffuse variety is best left alone.

#### ENCHONDROMA.

Enchondromata are composed of cartilage. They are innocent tumours, and grow slowly; cartilage is, however, sometimes found in malignant tumours (chondrifying sarcoma). They are composed of cartilage, the matrix of which may be either hyaline or fibrous (see Fig. 12), they are usually encapsuled and lobulated. They may undergo calcification or ossification, or they may soften, forming cysts.

They are most commonly found growing from the phalanges of the fingers, but are sometimes also found in the parotid

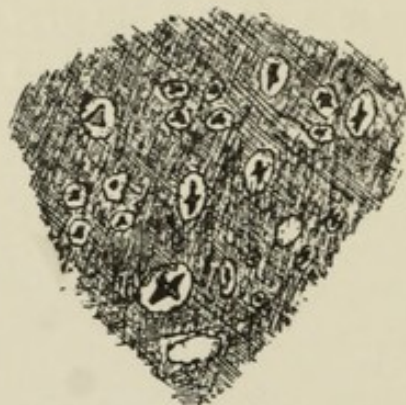


Fig. 12.—ENCHONDROMA.

gland and testis. They should be removed when possible ; the pure enchondroma does not recur after removal.

#### OSTEOMA.

Osteomata are composed of bone. They will be dealt with under *Diseases of Bone*.

#### MYXOMA.

Myxomata are composed of tissue resembling the Whar-

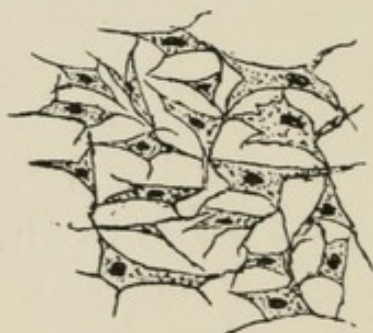


Fig. 13.—MYXOMA.

tonian jelly of the umbilical cord ; they are really soft fibrous tumours in which nearly all the intercellular substance has



become replaced by mucin in which the branched connective tissue corpuscles are suspended, forming a kind of loose network. (See Fig. 13.) Myxomata form soft semi-translucent gelatinous tumours, often pedunculated, and yielding a tenacious glairy fluid on section. Their commonest situation is the mucous membrane of the nose, where they constitute the ordinary nasal polypus. They should be removed; they do not recur.

### MYOMA.

Myomata are composed of muscle tissue, nearly always of the unstriped variety. They are most commonly seen in the uterus and prostate gland. They are composed of long spindle-shaped nucleated cells held together by a reticulum of fibrous tissue. They are prone to undergo calcareous degeneration, especially when situated in the uterus. They are innocent tumours, and do not recur after removal. Their common situation renders them only occasionally amenable to surgical treatment.

Tumours containing striped muscle are very rare, are usually composed chiefly of sarcoma tissue, and have been most often found in the kidney.

### NEUROMA.

Neuromata are tumours situated on nerve trunks. Pure neuromata are very rare; they are composed of medullated or non-medullated nerve fibres. False neuromata are composed of fibrous, myxomatous or sarcomatous tissue

## ANGEIOMA.

Angeiomata are composed of blood vessels. They occur in two chief forms, the *capillary*, and the *venous*. Capillary angeiomata (*nævi*), are composed of a fibrous stroma containing many large thin walled capillary vessels with frequent irregular pouches and anastomoses. They occur in the skin, forming the so-called "port wine stains," "mother's marks," etc. The venous *nævus* or cavernous angeioma consists of large cavernous spaces lined with endothelium. They are usually found in the subcutaneous connective tissue, forming soft swellings which can be emptied by pressure. *Nævi* are always congenital. They may be treated by

- (1) Excision with the knife.
- (2) Ligature.
- (3) Coagulating injections.
- (4) Electrolysis.

## LYMPHANGIOMA.

Lymphangiomata are tumours composed of dilated lymphatic vessels.

## LYMPHOMA.

Lymphomata are tumours composed of lymphatic gland tissue. Except for their large size they closely resemble normal lymphatic glands, both in naked eye appearance and in their microscopical characters.



## SARCOMA.

Sarcomata are tumours composed of embryonic connective tissue cells like those seen in developing portions of the embryo.

Four chief varieties are described.

1. *The Round-celled Sarcoma* is composed of round cells which may closely resemble leucocytes in size and shape or may be considerably larger. The cells are embedded in a homogeneous or granular matrix, the whole mass being held together by a delicate fibrous meshwork. Running between the cells are a large number of blood vessels with very thin walls; some of them indeed have no walls, but are simply spaces between the cells. (See Fig. 14.)

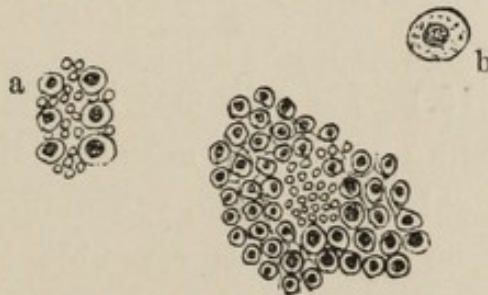


Fig. 14.

## ROUND-CELLED SARCOMA.

a.—Blood corpuscles in spaces between the cells.

b.—A single cell more highly magnified, showing nucleus and granules.

Round-celled sarcomata may grow from any part of the body where connective tissue exists; their most common situations are the skin and subcutaneous tissues, the bones, periosteum, and the lymphatic glands. They form large rapidly-growing tumours of a soft brain-like consistency, and of a dirty-whitish colour, mottled in places by extravasation

of blood. In some cases they are enclosed in an imperfect capsule, but usually they infiltrate the neighbouring tissues without having any very definite limit.

2. *Spindle-celled Sarcomata* are composed of cells which are elongated in one diameter. The cells may be oval or oat-shaped, or they may be much longer, having delicate tapering



Fig 15.—SPINDLE-CELLED SARCOMA.

ends. (See Fig. 15.) The cells may be arranged in bundles somewhat resembling unstriped muscular tissue. They are found most commonly in the skin, subcutaneous tissue, bones and periosteum. They closely resemble the round-celled sarcomata in naked eye appearance, but are apt to be of somewhat firmer consistence.

3. *Mixed-celled Sarcomata* are composed partly of round and partly of spindle-shaped cells; they closely resemble the preceding varieties.

4. *Myeloid Sarcomata* are characterized by the presence of large multinucleated cells resembling those found in the red marrow of bones. (See Fig. 16.) These myeloid cells consist of masses of protoplasm containing from ten to fifty nuclei. They are surrounded by round, oval, or spindle-shaped cells. Myeloid sarcomata grow chiefly from the



interior of bones, especially the lower end of the femur, the upper end of the tibia, and the lower jaw. On section they present a deep red or maroon colour, and are often stained by extravasation of blood.

Other varieties of sarcoma are sometimes met with. These are (a) *Melanotic Sarcoma*, which is characterized by the pre-



Fig. 16.  
MYELOID SARCOMA.

sence of pigment, and which grows from situations where pigment is normally present, viz., the skin and the pigmented tunics of the eye. The cells may be either round or spindle-shaped. (b) The *Alveolar Sarcoma*, in which the cells are arranged in spaces or alveoli. (c) *Glioma* or *Glio-sarcoma* which grows from the connective tissue of nerve substance (neuroglia). (d) *Psammoma*, which is characterized by the presence of concentric calcareous masses, and (e) *Lympho-sarcoma*, which resembles lymphoid tissue.

*Secondary changes in Sarcoma.*—The most common are—

(a) *Mucoid degeneration.* A clear substance collects in the interior of the cells which gradually distend and liquefy, thus producing cysts.

(b) *Hæmorrhagic softening.* The blood vessels, as already



stated, have extremely thin walls ; consequently their contents are apt to become extravasated amongst the tissues. In this way blood cysts are formed.

(c) *Ossification* may take place in those sarcomata which grow from bone.

(d) *Chondrification*, or the formation of cartilage is sometimes met with, especially in sarcomata of the testicle.

(e) *Fibrification*, or development into fibrous tissue, is seen in the less malignant varieties growing from the subcutaneous or intermuscular connective tissue.

*Clinical characters of Sarcomata.* All sarcomata are more or less malignant, but the degree of malignancy depends upon the situation of the tumour, and the nature of the cells of which it is composed. The nearer the elements of the tumour approach to the character of fully formed connective tissue, the less malignant is it. Thus the round-celled sarcoma is extremely malignant, whilst some of the spindle-celled sarcomata, in which the cells are very elongated, almost forming fibrous tissue, are scarcely malignant at all. Sarcomata are commoner in young than in old people ; their rate of growth may vary greatly, the more malignant growing more rapidly than the more benign tumours. The extreme thinness of the walls of their blood vessels renders them very prone to disseminate throughout the body by means of the blood current. They possess no lymphatics, and consequently do not affect the neighbouring lymphatic glands, with the notable exception of sarcoma of the tonsil, testis, and lymphatic glands and melanotic sarcoma of the skin, in all of which glandular affection is a prominent feature. Those sarcomata which grow from the subcutaneous and intermuscular tissues are nearly always enclosed in



distinct capsules, whereas those occurring in the tonsil, testicle, and lymphatic glands speedily occupy the whole organ, and are only encapsuled by its tunic. Even in encapsuled sarcomata the neighbouring tissue is always infiltrated to some extent. When sarcomata approach the skin, they tend to infiltrate it, and eventually ulcerate, forming soft fungating masses discharging a mixture of blood and pus. Recurrence *in situ* is very common after removal. In some cases the recurrent growth is more cellular, and consequently more malignant than the original one. In other cases, the malignancy gradually diminishes, so that recurrence takes place at longer intervals after each removal, and eventually ceases.

*The diagnosis* of sarcoma is often very difficult, especially when growing in connection with bone, and must be made by a careful consideration of all the circumstances of the case. In some instances the diagnosis cannot be effected until an exploratory incision has been made into the tumour, and even then mistakes are sometimes made.

*The prognosis* is usually bad, but varies with the seat of the disease. Thus a sarcoma of the tonsil is always rapidly fatal, whilst a myeloid sarcoma of the jaw, or a spindle-celled sarcoma of the subcutaneous tissue is often permanently cured by operation.

*Treatment of Sarcoma.* Every sarcoma should be removed when possible, and the sooner the operation is done the better the chance of success. It is always advisable to remove a considerable area of apparently healthy tissue as well as the actual tumour, especially in the more malignant varieties. In myeloid sarcomata of bone it may suffice in some cases simply to scoop out the tumour, but in round-



celled sarcoma of bone it is advisable to remove the whole bone. Some sarcomata, such as those of the tonsil, are so malignant that it is only rarely that an operation can be of any use.

### PAPILLOMA.

Papillomata (warts) are composed of papillæ resembling somewhat those found in normal skin and mucous membrane. They consist of a stroma of fibrous tissue containing blood vessels and lymphatics. The surface is covered by epithelium, the nature of which varies according to the tissue from which the growth springs ; thus papillomata of the skin are covered by several layers of squamous epithelium, (see Fig. 17), whilst papillomata of the bladder are covered by columnar



Fig. 17.

### PAPILLOMA OF SKIN

epithelium. In simple papillomata the stroma is single and undivided, but in compound papillomata it gives off numerous cauliflower-like branches which subdivide again and again.

Papillomata are innocent growths, but it should be remembered that in some situations, such as the tongue and lower



lip, they are prone to become epitheliomatous, especially in old people. This change may be recognized by the base of the wart becoming indurated.

*Treatment.* Warts on the skin may be destroyed with nitric acid. From mucous membranes they may usually be snapped off with scissors. When they are at all indurated they should be freely removed with the knife or scissors.

### ADENOMA.

Adenomata are tumours composed of gland tissue. A pure adenoma (i.e., one containing no more connective tissue

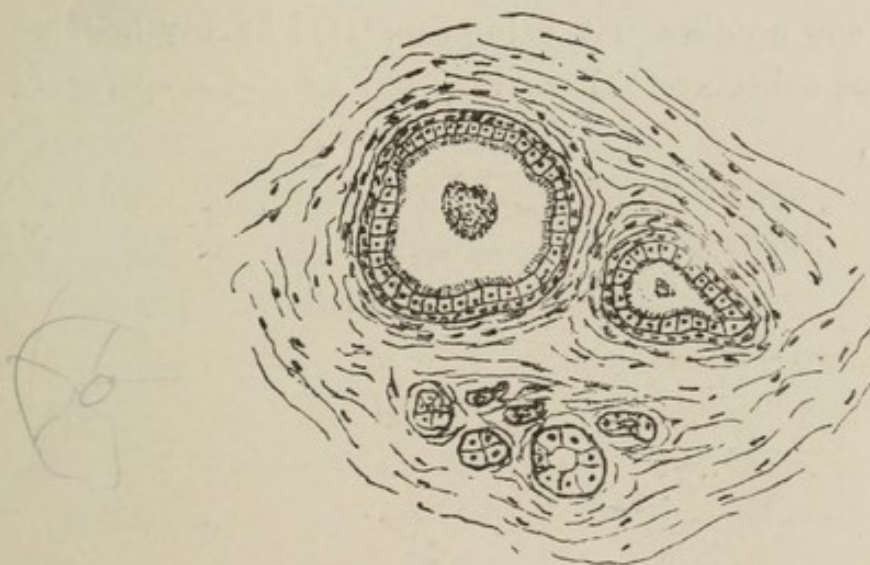


Fig. 18.

FIBRO-ADENOMA OF THE BREAST,  
Showing Ducts and Dilated Acini.

than a normal gland would), is very rare. There is nearly always an excess of connective tissue which may be either fibrous (*adeno-fibroma*), mucous (*adeno-myxoma*), or sarcomatous (*adeno-sarcoma*). The gland tissue itself may be

either *acinous* in structure like that of the breast (see Fig. 18) or *tubular* like the follicles of the rectum.

Adenomata are innocent tumours, unless their connective tissue is sarcomatous, in which case they are malignant, the malignancy depending not on the epithelial but upon the connective tissue elements.

*Treatment.* Removal by operation.

### CARCINOMA.

Carcinomata are tumours composed of epithelial cells arranged in clusters within spaces or alveoli formed by connective tissue. The individual epithelial cells lie in immediate contact with one another, not being separated by any matrix or intercellular substance. They vary in size and shape, and

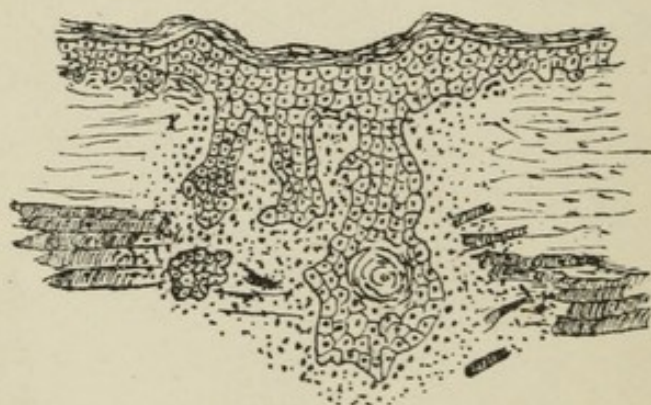


Fig. 19.

#### SQUAMOUS-CELLED CARCINOMA (EPITHELIOMA.)

Processes of epithelium are growing down into the subjacent connective tissue and muscle. The largest one contains a cell nest. The neighbouring tissues contain an excess of small round cells.

usually resemble the natural epithelium from which they arise. They are said to multiply both by fission and by endogenous cell formation. The fibrous tissue of which the



alveolar walls are composed contains blood vessels and lymphatics. There are three chief varieties of carcinoma, and they differ from each other chiefly in the character of the cells which are found in the alveoli.

1. SQUAMOUS-CELLED CARCINOMA. This variety is usually called *epithelioma*; it is composed of masses or columns of squamous epithelial cells resembling those seen in the skin. These columns grow from the interpapillary processes of skin or mucous membrane, and extend into the subjacent tissues, where they throw out lateral branches which, uniting with similar offshoots from neighbouring columns form a sort of epithelial network which infiltrates everything with which it comes in contact. At the same time an outgrowth takes place upon the surface which forms a sort of cauliflower excrescence, unless, as not unfrequently happens, the outgrowth ulcerates as rapidly as it is formed. The cells of epithelioma are usually large and irregular in shape; they are frequently grouped together into concentric masses called *cell nests*. In a cell nest the central cells are rounded, whilst those towards the periphery are flattened out into a crescentic shape. The tissue in the immediate vicinity of an epithelioma is usually infiltrated with small round cells (inflammatory exudation).

Epitheliomata are most commonly situated at the junction of skin and mucous membrane, e. g., on the lower lip, the tongue, the anus, the penis and vulva. They occur in people past middle life, and more often in men than in women. The disease is rare before the age of forty.

Epithelioma may commence as a warty growth, as a fissure, or as a tiny nodule or lump; ulceration occurs at a very early date and proceeds so rapidly that there is usually more



ulcer than tumour. The base and edges of the ulcer are always indurated. The rapidity of growth varies greatly; thus on the lower lip the disease may take three or four years to reach the size of a nut, whereas in the tongue a much larger mass may be produced in a few months. The lymphatic glands become involved early in the course of the disease; they become enlarged and very hard. In epithelioma of the antrum and of the interior of the larynx involvement of the glands does not take place until late in the disease.

*The diagnosis* of epithelioma is usually quite easy when attention is directed to the small size of the tumour, the early ulceration, the induration of the base, the enlargement of lymphatic glands, the situation of the tumour, and the age of the patient. In doubtful cases, a scraping from the surface of the ulcer may be placed on a glass slide in a drop of water, and examined under the microscope. The scrapings from syphilitic and tubercular ulcers are composed of debris of food, micro-organisms, pus, and blood. In scrapings from an epithelomatous ulcer there are numerous epithelial cells of varying size and shape, and sometimes cell nests.

RODENT ULCER is a variety of epithelioma, which possesses sufficiently distinguishing characters to merit a separate description. It is seldom seen in people younger than forty-five, and is nearly always found on some part of the face, especially the side of the nose near the inner angle of the orbit. It commences as a small wart or papule, which soon ulcerates. The ulceration proceeds almost as rapidly as the growth, so that at no time is there any marked tumour. The ulcer may heal in one place whilst it is extending in another, but the scar is very prone to break down and ulcerate afresh. The progress of the disease is so slow that death usually occurs from some



other disease or from old age, before the ulceration has extended to any vital part. The lymphatics are never affected, nor do secondary deposits ever occur. There is no cancerous cachexia, and the disease, if thoroughly removed does not recur. Microscopic examination of a rodent ulcer shows that the epithelial growth is developed mainly in connection with the sebaceous and sweat glands of the skin, not the surface epithelium. Usually there are no cell nests, and the cells are smaller and rounder than those of epithelioma.

2. SPHEROIDAL-CELLED CARCINOMA grows in connection with glands which are lined with spheroidal (glandular) epithelium. There are two chief varieties, the *hard* and *soft*.

(a) *Hard Carcinoma (scirrhus)* is most often met with in the female breast, where it forms an indurated, irregular nodular mass which cannot be separated from the gland itself. If left alone it soon infiltrates adjoining parts, thus becoming adherent to the skin, muscles, fasciæ and even to the ribs. The glands in the axilla soon become infiltrated, and deposits occur in the internal organs. When the disease has become extensive and has involved the skin or internal organs a general condition of impaired nutrition, known as "cancerous cachexia" is produced. Recurrence after removal is very common. Under the microscope the disease is found to consist of spheroidal cells enclosed within alveoli of dense fibrous tissue (see Fig. 20). To the naked eye the growth presents on section the appearance of an unripe pear or turnip. It is very hard, creaking under the knife, and has a concave section from which a peculiar juicy fluid can be expressed. It has no defined edge or capsule.

(b) *Soft Carcinoma (encephaloid)* differs from scirrhus in that it contains more cells and less fibrous tissue, and is

consequently softer and more brain-like in consistence. It is even more malignant than scirrhus.

*Treatment of Carcinoma.* Whenever it is possible the disease must be completely removed by some *radical* measure such as the knife or caustic. When an organ such as the

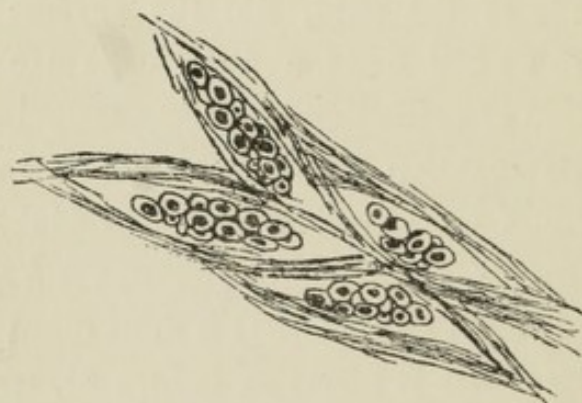


Fig 20.

SPHEROIDAL-CELLED CARCINOMA (SCIRRHUS).

breast, eye, or testis is the seat of disease the entire organ must be removed. Not only must all visible disease be removed but a certain amount of surrounding apparently healthy tissue must be included in the operation, for the microscope demonstrates that infiltration extends beyond the limits of the disease actually obvious to sight and touch. The complete removal of the disease, of course, includes the thorough extirpation of all affected lymphatic glands. It is obvious that when secondary deposits have occurred in internal organs no radical cure can be attempted. Before proceeding to operate on any given case, the surgeon must assure himself, as far as possible, that he will be able to completely remove the disease. If this cannot be done, he must content



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himself with *palliative* treatment, which consists in relieving pain by administering opiates, keeping the part clean by means of antiseptics, and supporting the patient's general health as much as possible by food, tonics, stimulants, etc. It not infrequently happens that an operation becomes necessary when some canal, such as the intestine, becomes occluded by the growth. In a few cases it may be justifiable to remove the bulk of a cancerous growth, when a radical operation is impossible, with the object of relieving pain or diminishing fœtor and hæmorrhage.

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## CHAPTER XI.

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### CYSTS.

A cyst is a closed sac with liquid or semi-solid contents. Cysts are usually divided into

1. Those formed by distension of pre-existing tubes or cavities.
2. Those of new formation.
3. Those of developmental origin.

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DISTENSION CYSTS.

The most common are those that result from obstruction of a duct ; such as (a) *mucous cysts* which result from obstruction of the duct of a mucous gland. Such cysts are often seen on the lips, forming little semi-translucent swellings which contain a watery or glairy fluid. They are treated by snipping off a portion of the cyst-wall with scissors ; (b) *sebaceous cysts*, which result from obstruction of the duct of a sebaceous gland by dirt, etc. They are most often met with on the scalp (wens) and face, forming tumours varying in size from a pea to a hen's egg. They are situated in the substance of the skin, which consequently cannot be pinched up over them. On the summit of the tumour a black speck may sometimes be seen ; this is the orifice of the obstructed duct. They contain semi-solid sebaceous material. Sometimes the patency of the obstructed duct can be re-established, and the contents squeezed out : when this cannot be done the cyst should be removed. In some cases sebaceous cysts become inflamed and suppurate producing an abscess, which requires to be opened in the ordinary way. They often heal slowly.

Other distension cysts are the result of distension of a cavity which has no duct or outlet, *e.g.*, the tunica vaginalis of the testis, bursæ, etc.

## CYST OF NEW FORMATION.

Under this head are included :—

(a) *Blood cysts.* These are most often met with in sarcomata the vessels of which are, as already stated, very thin and apt to burst. The extravasated blood becomes enclosed in a thin fibrous capsule. It may remain fluid for



an indefinite period, or it may clot and liquify again, forming a kind of treacly fluid. In some myeloid sarcomata of bone, blood-cysts may form to such an extent that there is hardly any trace of the sarcoma tissue left. Blood-cysts may also result from extravasation of blood into a healthy tissue or organ, such as the brain (*cerebral hæmorrhage—apoplexy*) or into a serous cavity such as the tunica vaginalis (*hæmatocoele*), the blood becoming enclosed by inflammatory exudation.

(b) *Degeneration Cysts* may form in the interior of solid tumours, such as sarcomata, enchondromata, or soft fibromata, as the result of the cells of the tumour undergoing mucoid or colloid degeneration.

(c) *Serous Cysts* result from the distension and coalescence of lymphatic spaces in connective tissue; in this way the so-called adventitious bursæ are formed.

(d) *Parasitic cysts* result from the growth of some form of parasite in a tissue. The best example is the hydatid cyst of the liver.

(e) *Implantation cysts* are caused by the accidental transplantation of portions of skin, surface epithelium or hair bulbs into the underlying connective tissue. They are most often found on the fingers of persons who gain their living by sewing, carpentering, shoe-making, etc. They are lined by skin and contain sebaceous matter and other skin products.

#### CYSTS OF DEVELOPMENTAL ORIGIN.

These are the result of some error or imperfection in the process of development. They may be divided into two chief classes.

(a) *Tubulo-dermoids*. In the foetus there are certain

canals and cavities which become obliterated at or before birth. In some cases the obliteration is imperfect, and the persistent portion becomes distended with fluid and forms a cyst.

(b) *Sequestration dermoids.* During embryonic life certain portions of the body covered with skin come in contact with one another and coalesce. During this process of coalescence it may happen that a piece of skin gets nipped between the two coalescing portions, and becomes buried beneath the surface ; in its new situation it may continue to grow and form a cyst lined with skin, and containing hair, teeth, sebaceous material, etc. Sequestration dermoids are found only in those situations where coalescence takes place during embryonic life. Thus on the trunk they occur in the middle line of the body ; on the face they occur at the angles of the orbit, along the naso-facial sulcus, near the angle of the mouth, in the middle line of the chin, and on the nose. They form small rounded or oval tumours situated beneath the skin (not in it like a sebaceous cyst); they may extend somewhat deeply, and when situated over one of the cranial bones, the latter may be indented, or even perforated, a fact to be borne in mind in dissecting them out.

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CHAPTER XII.

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## DISEASES OF ARTERIES.

**CALCAREOUS DEGENERATION.** This occurs in two forms (a) as a primary affection and (b) secondary to atheroma. Primary calcareous degeneration occurs in old people, and affects vessels of medium size, especially the tibials and the arteries of the brain. The muscular fibres of the middle coat of the vessel, become infiltrated with salts, the result being that a number of rings are formed extending round the artery; after a time, the inner and outer coats become affected as well, so that the artery becomes converted into a rigid and brittle tube. The circulation through a calcareous artery may be so much impeded, as to lead to gangrene. Secondary calcareous degeneration will be described under Atheroma.

**ATHEROMA.** This is a condition which results from chronic arteritis. It is met with most frequently in the larger arteries, such as the aorta. The inflammation affects chiefly the deeper layers of the inner coat. It commences by an exudation of small round cells which form broad, flat, slightly raised patches of a whitish-yellow colour on the inner surface of the artery. After a longer or shorter time these cells, having an insufficient blood supply, undergo fatty degeneration and form a liquid mass which is still separated from the cavity of the vessel by the superficial layers of the inner coat.



This condition is sometimes called an *atheromatous abscess*. Ultimately the abscess bursts and discharges its contents into the blood-vessel, leaving a rough surface sometimes called an *atheromatous ulcer*. In many instances the atheromatous abscess does not burst, but the more liquid part of the degenerate mass becomes absorbed, leaving only an irregular calcareous plate in the site of the original exudation. This secondary calcareous degeneration is readily distinguished by its irregularity from the regular circular arrangement of primary calcareous degeneration. The inflammatory process is not limited to the inner coat but extends also to the middle and outer coats, but here the inflammatory products being better supplied with blood, become converted into fibrous tissue, thus thickening and strengthening the artery at the spot weakened by the degeneration of the inner coat.

The most important causes of atheroma are chronic renal disease and hypertrophy of the heart causing increased blood pressure in the arteries. It is most often met with in men past middle life who have been addicted to alcohol and who have followed laborious occupations.

The chief results of atheroma are aneurysm, thrombosis, and embolism.

**ANEURYSM.** An aneurysm is a tumour containing blood and communicating with the cavity of an artery. It is usually the result of distension of a portion of the wall of an artery which has been weakened by atheroma. The sac of an aneurysm is formed by the coats of the artery together with the sheath and surrounding cellular tissue. An aneurysm is described as *fusiform* when it involves the whole circumference of the artery, as *sacculated* when it involves only a part, (say one side) of the arterial circumference, thus producing



a projecting sac ; as *true* when all the arterial coats are distended and as *false* when some of them have given way. An aneurysm contains blood which is usually partly fluid and partly clotted. The clot may be of the ordinary gelatinous consistency, or may be laminated, i.e., deposited in layers. If left alone an aneurysm tends in most cases to go on increasing in size until it bursts either into some internal cavity or upon the surface of the body. In some cases the laminated clot increases in quantity until it fills up the aneurysm and affects a spontaneous cure. The tumour or swelling formed by an aneurysm presents the following characters in a typical case; it is in the line of an artery, it pulsates, the pulsation being synchronous with the action of the heart, expansile in character, and arrested by compression of the artery above the tumour ; by pressure the sac may be partially emptied and the size of the swelling reduced ; on listening with a stethoscope placed on the tumour a *bruit* is heard.

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## CHAPTER XIII.

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### DISEASES OF VEINS.

VARIX. A vein is said to be varicose when it is more dilated than natural. Varix is most commonly the result of mechanical obstruction to the return of venous blood to the

heart. Varicose veins are most common in the legs, lower part of rectum and anus (hæmorrhoids) and spermatic cord (varicocele). The affected veins become distended, and elongated, and consequently tortuous. Owing to the dilatation the valves cease to act. At first the walls of the vein become thickened to withstand the increased pressure, but later they get thinned in places, producing little bulging pouches. Varicose veins are very prone to become inflamed and thrombosed (*vide infra*).

PHLEBITIS is inflammation of a vein, and may be either *plastic* or *suppurative*.

*Plastic phlebitis* may be due to mechanical injuries, to extension of inflammation from neighbouring parts, or to the presence of a thrombus. The changes that take place in the inflamed vein are similar to those that occur in plastic inflammation of other parts, with this addition, viz., that phlebitis is nearly always associated with thrombosis either as cause or effect. The symptoms and treatment are identical with those of thrombosis to be presently described.

*Suppurative phlebitis* is most frequently seen in the veins of a bone which is the seat of septic inflammation. The septic organisms invade the clot which occludes the inflamed vein and cause it to soften and break down, so that it no longer forms an efficient barrier between the suppurating area and the fluid blood inside the healthy portion of the vessel. The result is that the phlebitis spreads along the vein. Moreover portions of the softened clot are apt to become detached and carried to distant parts producing metastatic abscesses (see Pyæmia). It may be inferred from the above account that the local symptoms of suppurative phlebitis are usually obscured by the septic affection of which it



forms a part, whilst the constitutional symptoms are those of pyæmia. The treatment consists in endeavouring to render the part aseptic, and in preventing the infected clot from entering the general circulation. In some cases, e.g., septic thrombosis of the lateral sinus, this has been successfully done by placing a ligature on the vessel, between the inflamed portion and the heart.

THROMBOSIS is the clotting of blood inside a blood vessel. It occurs in arteries as well as in veins, but is much more common in the latter on account of their superficial position exposing them to injury, and on account of the relative slowness of the venous circulation. In considering the causes of thrombosis it must be borne in mind that the reason the blood does not clot inside the vessels of healthy persons is that the fibrin ferment (which is essential for the process of coagulation) is locked up within the white corpuscles; any cause which by damaging the white corpuscles sets the ferment free, so that it can act on the fibrin-forming elements of the blood, produces thrombosis. These causes may be arranged under two heads, viz., (a) those in the blood, and (b) those in the wall of the vessel. The first includes (1) various constitutional affections, such as gout, rheumatism, anæmia, typhoid fever, etc., (2) Foreign substances in the blood stream, such as pre-existing clot, an embolus, micro-organisms, a ligature transfixing a vein, etc., and (3) Slowing of the blood stream, whether due to local or general conditions. The second set of causes includes (4) Mechanical injury of the vessel wall, and (5) Roughening of the vessel wall from phlebitis or atheroma.

The clot which forms in the thrombosed vessel is called thrombus. When the clot forms quickly the red corpuscles



become entangled and give it the usual red colour ; when on the other hand the blood continues in rapid motion, and when the focus of coagulation is small, as in the case of a thread transfixing a vein, the leucocytes adhere to the rough surface, fibrin forms around them and a white thrombus is produced ; sometimes the thrombus is mottled, consisting partly of red and partly of white clot.

The following changes may occur in a thrombus in different cases.

1. *Contraction.* A clot in a vessel has the same tendency to contract, as a clot formed outside the body.

2. So-called *Organisation.* This means that the clot becomes infiltrated with leucocytes which develop into fibrous tissue.

3. *Calcification.* The clot becomes gradually converted into a calcareous mass called a phlebolith.

4. *Softening.* An aseptic clot may soften and form a greyish-red pulp which gets gradually washed away. Softening begins in the centre of the clot. A septic clot often undergoes yellow or puriform softening ; this is always the result of the action of bacteria and occurs in cases of suppurative phlebitis.

5. *Tunneling.* Occasionally the lumen of the vessel becomes restored by a narrow channel or tunnel being formed through the clot or between it and the vessel walls. The symptoms of thrombosis are the formation of a cord-like swelling in the course of a vein, the skin over which is usually tender, reddened and œdematous. The parts normally drained by the blocked vein become congested and œdematous, unless there is a free collateral circulation. The treatment consists in keeping the part at rest, and allaying



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the concomitant inflammation by hot fomentations, or by smearing a mixture of belladonna and glycerine over the affected part.

EMBOLISM is the plugging of a vessel by some foreign substance brought from a distance by the blood stream.

An embolus may consist of a vegetation from the valves of the heart, calcareous matter from an atheromatous artery, blood clot from a thrombosed vein, a portion of tumour which has invaded a vessel, a colony of bacteria, etc.

An embolus may lodge in any of the systemic arteries, the pulmonary artery or the portal vein. At the site of impaction a *secondary thrombus* usually forms around the embolus and completes the occlusion of the affected vessel. The parts supplied by the blocked artery become deprived of blood until collateral circulation is established. Embolism of the pulmonary artery may be at once fatal. When the embolus is derived from a septic source and contains bacteria it sets up inflammation and suppuration (see suppurative phlebitis and pyæmia).

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CHAPTER XIV.

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## INJURIES AND DISEASES OF NERVES.

WOUNDS. The following are the effects of complete division of a mixed (motor and sensory) nerve such as the ulnar nerve.

1. *Loss of Sensation.* The area of anæsthesia corresponds roughly but not accurately to the anatomical distribution of the nerve below the point of section. It sometimes happens that a part of the area supplied by the wounded nerve retains a feeble power of sensation; this is due to the fact that the nerve below the seat of injury may receive small communicating branches from a neighbouring uninjured nerve.

2. *Loss of Motor power.* The muscles supplied by the nerve below the point of section are immediately paralysed and therefore cannot be contracted at will.

3. *Changes in Temperature.* Immediately after the section of the nerve the parts below become suffused with blood and consequently hotter by a degree or a degree and a half Fahrenheit. The increase of temperature may last a fortnight or longer. Subsequently the affected parts become colder than natural.

4. *Trophic Changes.* The nutrition of the tissues is



under the direct control of the nervous system, consequently after section of a nerve the parts supplied by it suffer from want of trophic nervous influence and undergo various degenerative changes.

(a) *The nerve* itself on the distal side of the section undergoes a series of changes known as *Wallerian* degeneration; the myelin breaks up into globules and the axis cylinders disappear so that the sheaths of Schwann are more or less empty. The connective tissue (neurilemma) of the nerve proliferates and eventually shrinks completing the destruction of the proper nerve elements.

(b) *The muscles* undergo similar changes, the connective tissue enclosing the fibres proliferates whilst the muscle fibres themselves break up and disappear; the consequence is that the affected muscles shrink and cease to contract when electric currents are passed through them; Faradic contractibility disappears at once, whilst galvanic contractibility after a temporary increase fades away gradually, only ceasing when all the muscular fibres have been destroyed (*reaction of degeneration.*)

(c). *The Skin* of the affected area becomes glossy or shiny, or may be subject to various eruptions, or may ulcerate from trivial injuries, or may even slough.

(d). *The joints* may become ankylosed or may undergo degenerative changes. There is a well-marked form of joint disease (*Charcot's disease*) which is dependent upon an affection of the spinal cord called *locomotor ataxy*.

*Treatment.* A divided nerve should be sutured as soon as possible after the injury. If done at once and primary union is secured the functions of the nerve may be restored without



any degeneration taking place, but such a fortunate result is very rare. If the case is not seen until some time has elapsed the divided ends must be exposed, a fresh section made, and the ends united by suture. Regeneration will occur in the nerve and muscles unless they have already completely wasted. A year or more may elapse before motion and sensation are completely restored.

**NEURITIS.** Signifies inflammation of a nerve. It may be due to injury, to cold, or to extension of inflammation from neighbouring parts. It may also occur as a complication of syphilis, lead poisoning, chronic alcoholism, gout, rheumatism, and the specific fevers, especially diphtheria. The inflammatory process occurs mostly in the connective tissue; the proper nerve elements undergoing degeneration similar to that described under wounds of nerves.

Neuritis may affect a single nerve when it is due to a local cause, or may affect many nerves when the cause is a general one, such as alcoholism.

The early symptoms are those of irritation, such as pain and tingling in the parts supplied by the affected nerves. The later symptoms are due to destruction of the nerve and consequent inability to conduct impressions; they closely resemble those due to section of a nerve.

**TUMOURS OF NERVES.** See Neuromata.

**NEURALGIA.** When a person suffers from severe pain in the course of a sensory nerve, and the cause of the pain cannot be discovered, it is usual to call the condition neuralgia. It must be clearly understood that neuralgia is not in itself a disease, but it is only a symptom of an undiscovered cause; to make the diagnosis of neuralgia is but to confess one's inability to find out the cause of the pain. Patients (espe-



cially females) suffering from toothache, not unfrequently try to persuade themselves that their disease is "neuralgia" with the object of avoiding the unpleasantness of treatment.

*The causes* of neuralgic pain are (a) impaired general health due to anæmia, debility, malaria, rheumatism, syphilis, gout, chronic lead poisoning, pregnancy, and lactation, and inherited neurotic temperament; and (b) some local condition irritating a sensory nerve, such as neuritis, a carious tooth, pressure on a nerve by a bony tumour or other form of growth, or by inflammatory exudation, such as a gumma or periosteal node, or by a piece of dead bone.

*The pain* in neuralgia may be slight or extremely severe; in the latter case it usually comes on suddenly with a kind of shock, and is of a darting, cutting, or tearing character, extending along the course of the affected nerve. The pain is sometimes accompanied by spasm of muscles, heat and redness, a vesicular eruption, or increase of secretion over the affected area. The points where the affected nerve emerges from a bony canal or passes through an opening in the deep fascia are often tender on pressure.

*Treatment.* Every endeavour must be made to discover the cause of the disease and to remove it by appropriate remedies; thus if the patient be anæmic, iron must be given; if rheumatic, salicylate of soda; if gouty, potassium iodide, colchicum or salicylate of soda; if syphilitic, potassium iodide; if he has suffered from ague, quinine or arsenic, etc. Every possible local cause of pain must be sought for, and it must be remembered that pain in one branch of a nerve may be due to some cause irritating another branch of it, thus a carious or impacted lower wisdom tooth may cause neuralgia of the whole of the fifth nerve, or of a part of it, e.g., earache.

When the cause of the pain cannot be discovered, attempts must be made to alleviate it by local and general analgesics. Among the former may be mentioned belladonna, aconite, veratria, chloroform, the galvanic current, etc., and among the latter opium, morphia, chloral, and bromide of potassium. Great caution should be exercised in the administration of opium, morphia or chloral lest the patient become addicted to the drug. In severe cases of neuralgia, which do not yield to local or general remedies, it is sometimes justifiable to resort to nerve-stretching or to section or excision of a part of the affected nerve.

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## CHAPTER XV.

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### DISEASES OF LYMPHATICS.

LYMPHANGITIS is an inflammation of the lymphatic vessels. It is nearly always due to irritation caused by the absorption of septic matter from some portion of the tissues from which the affected vessels derive their lymph. Thus in poisoned wounds of the fingers it is very common to find the lymphatic vessels of the forearm inflamed. The disease may be recog-



nized by the presence of fine red lines running from an inflamed or suppurating sore to the nearest lymphatic glands, which are also inflamed. These red lines are slightly raised above the surface, and are hard and tender to the touch ; the surrounding skin is often œdematous, the body temperature may be raised several degrees, and marked febrile symptoms may occur. The disease usually ends in resolution in about a week or ten days, but it may go on to suppuration. In some cases the poison gets into the general circulation and leads to septicæmia, pyæmia and death. The treatment is the same as that of phlebitis.

OBSTRUCTION OF LYMPHATIC VESSELS may result from their involvement in cicatricial tissue, or from blocking by a parasite called the *filaria sanguinis*. The result is that the parts which should be drained by the blocked vessels become swollen and engorged with lymph. When the obstruction has lasted a long time the connective tissue of the part becomes greatly hypertrophied, producing the condition known as *elephantiasis*.

LYMPHADENITIS is inflammation of lymphatic glands ; it may be acute or chronic.

*Acute inflammation* of lymphatic glands is nearly always the result of the irritation of some poison conveyed to them by the lymphatic vessels. The lymphatic vessels may also be inflamed, but they more often escape, as they allow poisonous particles (bacteria) to pass through them more readily than the lymphatic glands. The secondary inflammation in the glands usually resembles in character that of the primary lesion, from which the poison has been conveyed, thus if the latter be suppurating, an abscess is likely to form in and around the inflamed glands ; if,



on the other hand, the primary affection be simply an acute inflammation, as in cutaneous erysipelas, the glands may be expected to undergo resolution without suppurating. When suppuration occurs it may begin either in the gland itself, or in the cellular tissue surrounding it.

*The symptoms* are simply those of acute inflammation occurring in the situation of a lymphatic gland. The student must avoid making the mistake of diagnosing a swelling as an inflamed gland in a situation where no gland normally exists.

*The treatment* must be directed to the original source of infection as well as to the inflamed gland itself. In the early stage an attempt must be made to prevent suppuration by applying cold evaporating lotions ; if suppuration becomes inevitable it should be hastened by the application of a hot boracic fomentation. When pus has actually formed it should be evacuated by incision. Glandular abscesses should not be allowed to burst spontaneously, as they are often slow in healing, and are apt to degenerate into sinuses.

*Chronic Inflammation* of lymphatic glands is usually due to some constitutional taint in addition to a local source of irritation ; the latter is often so trivial as to escape detection. The constitutional taint in nearly all cases is the strumous diathesis. The commencement of the glandular disease is usually a simple inflammation, which in a healthy person would subside without leaving any traces. In a strumous subject the inflamed gland soon becomes the seat of a tubercular deposit which is extremely prone to undergo caseous degeneration. The caseous mass may liquify and be expelled by suppuration, or it may dry up and form a calcareous nodule. The enlarged glands are almost painless, and at first



are hard and freely movable. Usually several glands are affected in the same region forming a chain ; this is often seen in the neck. After a variable time the glands become adherent to one another and to the deeper parts, so that they are no longer freely movable ; the skin over them becomes adherent, and of a dusky red or bluish tinge, and finally bursts, giving exit to broken down caseous matter and thin flaky pus. Such abscesses may go on discharging for many months, and when they do heal, are prone to leave ugly puckered scars.

*Treatment.* This must be both local and constitutional. The latter comprises every means calculated to improve the general health, especially good food, sea air, cod liver oil, iron, and iodine. In the early stages of the disease, local treatment aims at causing absorption of the inflammatory products without suppuration. With this object, tincture of iodine may be painted on the skin, or iodide of lead ointment may be thickly spread on it every night, but these remedies are more often useless than not. When the glands have remained enlarged and hard for a long time, and have refused to yield to local and constitutional remedies it is justifiable to remove them by operation. When suppuration has occurred the pus must be freely evacuated, and the affected gland or glands dissected out if possible ; failing this, all the caseous matter should be scraped away with a sharp spoon ; if any of it is left behind, suppuration will go on until it has been evacuated. The scarring that follows operation is much less unsightly than that which ensues when abscesses are allowed to burst spontaneously.

CHAPTER XVI.  
  
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## DISEASES OF BONE

Under this heading we must include diseases not only of bone itself, but also of the periosteum and medullary membrane.

## PERIOSTITIS.

Inflammation of the periosteum of a bone may be due to local causes, such as injury, the irritation of a neighbouring tumour, the pulsations of an aneurysm, the presence of inflammation in the subjacent bone or neighbouring joint, or to general causes such as struma, syphilis and rheumatism, or to the action of specific micro-organisms.

The pathological changes which take place in the beginning are simply those of inflammation; the outer fibrous layer swells up and becomes redder than natural, whilst the cells of the osteogenetic layer proliferate and loosen the attachment of the membrane to the bone. The inflammation may lead to the following *results* or *terminations*.

(a) *Resolution*. When the cause has been a local one and has been removed or has ceased to act, the inflammatory process may subside and the parts return to their normal condition.



(b) *Suppuration.* Acute periostitis, especially when of septic origin, may lead to the formation of an abscess between the periosteum and the bone. Chronic periostitis may produce the same result in persons whose tissues are reduced to a low condition of vitality by syphilis or tuberculosis. Periosteal suppuration is often followed by death of a portion of the bone (see necrosis).

(c) *Formation of new bone.* In chronic periostitis the activity of the osteogenetic layer is increased, so that an excessive quantity of new bone is deposited. The new bone may take the shape of a smooth rounded elevation (*node*), or of sharp pointed stalactytic processes (*osteophytes*).

*Symptoms.* In acute periostitis there is very severe throbbing pain. The affected part is swollen and tender on pressure. If suppuration occurs the skin becomes red and œdematous, and a soft spot develops in the midst of the inflammatory hardness. In chronic periostitis the pain is less severe, but the swelling is very hard and unyielding owing to the formation of new bone; the skin is of normal colour, unless a chronic abscess forms, in which case it becomes dusky and mottled. In making a diagnosis in a case of periostitis, the cause of the disease must be discovered; in syphilis the pain is nearly always much worse at night; in rheumatism it varies in time, severity, and situation; in struma it is comparatively slight. Confirmatory symptoms must be looked for in other parts of the body.

*Treatment.* The cause of the disease must be dealt with whenever this is possible. When the periostitis is of syphilitic origin a course of iodide of potassium should be given; when rheumatic, salicylates or the iodide of potassium; and when strumous, cod-liver oil and iron. Any local source of



irritation must be removed. The local treatment varies with the condition of the inflamed part. Rest is always desirable, and sometimes essential. If the inflammation is acute it must be subdued by cooling lotions or local blood-letting by means of leeches. If there is any chance of suppuration occurring, and *a fortiori* if it has already occurred, an incision must be made through the soft parts right down to the bone so as to give free exit to all inflammatory exudation, for any such accumulation, by lifting the periosteum from the bone, deprives the latter of a part of its blood supply and endangers its vitality. In chronic osteoplastic periostitis the most useful local remedy is a succession of blisters.

#### OSTEITIS.

Inflammation of bone is essentially the same process as inflammation of soft parts, but the hard and unyielding nature of bone modifies to a certain extent the symptoms and effects of the disease.

*Causes.* The constitutional causes of osteitis include all conditions of impaired general health, especially the strumous diathesis. The local causes include injury, extension of inflammation from neighbouring parts, the deposit of tubercle, the formation of gummata, and the action of various micro-organisms.

*Morbid anatomy.* The first change is *congestion* ; this is soon followed by *exudation* of inflammatory lymph, the small round cells of which find their way between the lamellæ of compact bone and into the spaces of cancellous bone. The inflammatory process thus started may terminate in one of the following ways :—



1. *Resolution.* The inflammatory exudation becomes absorbed before it has had time to do any material damage to the bone, and the latter returns to its normal condition.

2. *Rarefaction.* The inflammatory exudation separates the bone lamellæ from one another and expands the cancellous spaces (see Fig. 21), so that the bone becomes more porous or spongy. This change is produced in two ways, partly by the mechanical pressure of the exudation, and

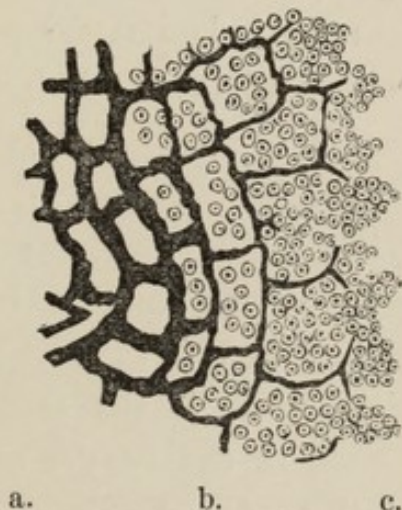


Fig. 21.—RAREFYING OSTEITIS AND CARIES OF CANCELLOUS TISSUE.

(a) Normal Cancelli.

(b) Rarefying Osteitis.

(c) Caries.

partly by erosion or eating away of the bone lamellæ and trabeculæ. The erosion is effected chiefly by large multinucleated cells called *osteoclasts*, which seem to have the power of dissolving out the bone salts, thus eating out little crescentic spaces in the bone called *Howship's lacunæ*. It is not certain whether the osteoclasts are derived from the blood or from the connective-tissue corpuscles of the bone.

3. *Caries.* The process of rarefaction may go on until some of the trabeculæ or lamellæ are completely eaten away; this constitutes caries (vide infra).

4. *Osteo-Sclerosis.* The inflammatory exudation becomes converted into bone, the result being that the bone is eventually much denser, harder, and heavier than it originally was. This change is analogous to the fibroid thickening that follows chronic inflammation of soft parts.

5. *Suppuration.* The inflammatory exudation may break down and form pus (see abscess of bone).

6. *Necrosis.* When the inflammatory process is severe, it may lead to death either of the whole bone affected or of some considerable portion of it. This is called necrosis (vide infra).

*Symptoms of Osteitis.* The symptoms closely resemble those of periostitis, and the two conditions are frequently, indeed usually, present together.

*Treatment of Osteitis.* This is the same as for that of periostitis. The treatment of the results of osteitis will be given with the description of caries, suppuration of bone, and necrosis (which see).

#### CARIES.

Caries of bone is essentially the same process as ulceration of soft parts. It is one of the terminations or results of osteitis, and is prone to occur when the osteitis depends on struma, tuberculosis or syphilis, or occurs in a person who is in a low state of general health from any other cause. It is more common in cancellous than in compact bone, affecting especially the bodies of the vertebræ, the carpus and tarsus and the ends of the long bones.



The nature of the process will be best understood by referring to Fig. 21. The thick black lines to the left of the diagram represent normal bone trabeculæ, and between them are the spaces or cancelli which are occupied by marrow (the marrow is not represented in the diagram). Passing to the right, the spaces are seen to grow larger and to contain inflammatory exudation, whilst the trabeculæ become thinner

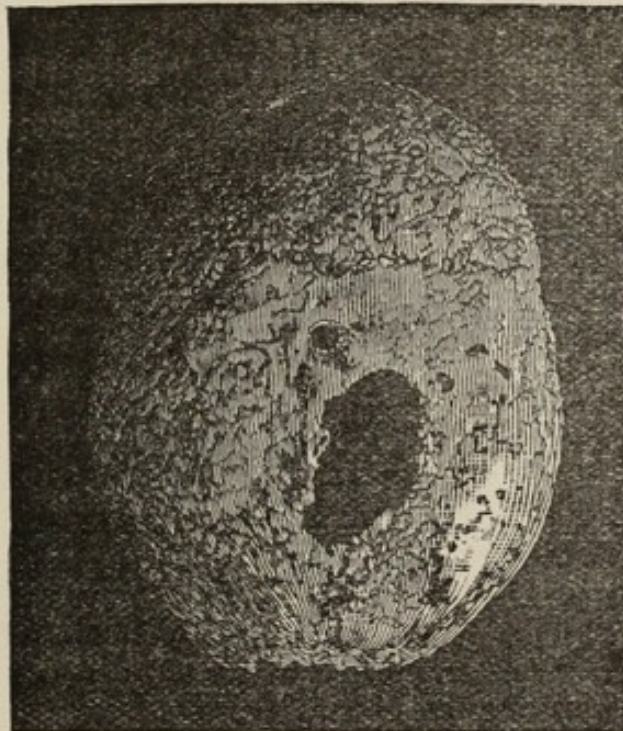


Fig. 22.

SYPHILITIC CRIES AND NECROSIS OF SKULL.

(Museum of St. Mary's Hospital.)

from absorption (*rarefying osteitis*). To the right of the diagram the trabeculæ have completely disappeared, and there is nothing but granulation tissue (*caries*). Under favourable conditions the granulation tissue may ossify, and sclerosis result. In the great majority of cases, however, the inflammatory material undergoes caseous degeneration or forms pus, which may find its way either to the surface



of the body or into a neighbouring joint. Sometimes caries continues for a long time without either leading to ossification or to suppuration, this condition is called *caries sicca*. Sometimes a small piece of cancellous tissue dies *en masse*, instead of molecule by molecule; when caries and necrosis are thus associated the condition is called *caries necrotica*, (see Fig. 22). In caries the result of tuberculosis the inflammatory exudation is especially prone to caseate, the bone is soft, greasy, crumbling, and extensively destroyed, and there is little or no tendency to repair. Giant cells and tubercle bacilli may be discovered in the inflammatory exudation.

*Symptoms.* An abscess forms in connection with a bone which has been for some time inflamed. The abscess instead of healing, dwindles down into a sinus, the orifice of which is usually surmounted by a crop of œdematous granulations, and gives exit to thin watery pus. A probe passed down the sinus impinges on soft friable bone.

*Treatment.* Remove the cause when possible and improve the general health. When the caries is in an accessible part, the diseased bone must be scraped or gouged away, and the cavity thus made allowed to granulate up. When there is extensive caries of a long bone, or of the foot or hand, excision or amputation may be required.

#### ABSCESS OF BONE.

Abscess of bone is usually chronic and situated in the cancellous ends of the long bones. It commences as a rarefying osteitis leading to caries of a circumscribed patch of cancellous tissue. The broken-down bone trabeculae become replaced by feeble granulation-tissue which degenerates into



pus. The granulation tissue lining the abscess cavity is sometimes partly organized forming a sort of membrane to which the name "pyogenic membrane" is sometimes applied. The surrounding bone may be sclerosed. When the abscess comes near the surface of the bone, it gives rise to osteoplastic periostitis, and the new bone thus formed may prevent its bursting on the surface ; this is not the case, however, when it comes near the articular surface of a neighbouring joint, for here there is no periosteum, and consequently no osteoplastic process ; hence the tendency of these abscesses to burst into the joint rather than on to the surface. When an abscess in bone has formed quickly, it often contains a small crumb of necrosed cancellous tissue.

The symptoms are usually chronic and obscure ; there is a dull boring pain in the affected part ; the pain is intermittent and worse at night. The skin over the affected spot may be œdematous, and there is tenderness on pressure. The neighbouring joint may be inflamed from time to time.

The treatment consists in opening the abscess with a trephine or gouge, and scraping the granulation-tissue away, rendering the cavity aseptic and allowing it to granulate up.

#### NECROSIS.

Necrosis is death of a piece of bone *en masse*. It corresponds to gangrene or sloughing of soft parts. Inflammation is more apt to be followed by death of the affected part when it occurs in bone than when it occurs in soft parts, because in the former, owing to the unyielding nature of the tissue, the blood vessels are compressed by the inflammatory exudation. For the same reason necrosis is more frequent in



compact than in cancellous bone. Necrosis is more apt to follow acute inflammation than chronic.

The immediate *cause* of necrosis is deprivation of blood supply ; a bone receives blood from two sources, from its nutrient artery, and from the periosteum covering it, hence when the periosteum is separated by injury or disease, the outer or superficial layers of the bone are apt to die (*peripheral necrosis*), and when the nutrient vessels in its interior are blocked or compressed, the central portion of the bone suffers, (*central necrosis*). When both periosteal and endosteal vessels are occluded the result is death of the whole thickness of the bone, (*total necrosis*). The commonest conditions interfering with the blood supply of bone, and producing necrosis, are injury (such as compound fracture, stripping off of periosteum, etc.), syphilitic osteitis, and periostitis, the specific fevers, and poisoning by mercury or phosphorus. The bones most commonly necrosed are the tibia, femur, lower jaw and skull.

The piece of bone which dies is called the *sequestrum* ; it is hard, dry, bloodless, and, unless exposed to the air, white ; its free surface is smooth, its margins ragged, its previously attached surface rough and worm-eaten.

The separation of the sequestrum from the living bone is effected by the process of ulceration. The irritation produced by the presence of the sequestrum sets up inflammation in the adjacent living bone ; a layer of granulation tissue is formed at the line of demarcation between the living and the dead bone ; the leucocytes and osteoclasts slowly dissolve or eat away the opposed surfaces of both the living and the dead tissue, so that when the process is complete, the dead bone is loosened from its attachments. When the sequestrum is



on the surface of the bone, it comes away spontaneously when the process of separation is complete (*exfoliation*), but when the sequestrum is central or involves the whole thickness of the bone, it is usually imprisoned in a dense irregular case of

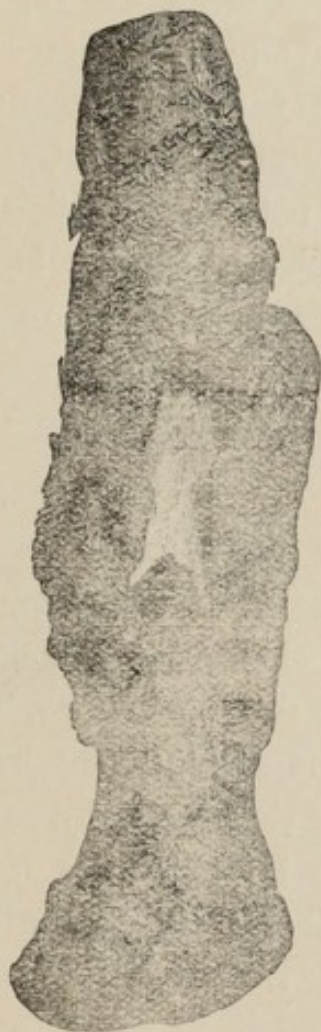


Fig. 23.

NECROSIS OF FEMUR.

The sequestrum is enclosed in a thick involucrum of periosteal new bone perforated by cloacæ.

(Museum of St. Mary's Hospital.)

new bone (*involucrum*) formed by the periosteum. The involucrum is perforated by several large apertures (*cloacæ*)

through which pus escapes (see Fig. 23). When the sequestrum is thus *invaginated* its release is practically impossible without surgical interference. After the escape or removal of the sequestrum the cavity which is left is slowly filled up by granulations, which ossify and replace the lost bone.

*Symptoms.* The symptoms of necrosis are always preceded by those of the injury or inflammation of which it is the result. When suppuration has occurred and the pus has been evacuated either by natural or artificial means, there remains a sinus through which a probe can be passed, and the dead bone examined, when it is found to present the characters already described ; the process of separation of the sequestrum may be known to be complete when the latter can be moved by the probe.

*Treatment.* The sequestrum must be removed as soon as the process of separation is complete. When the necrosis is superficial, removal of the sequestrum can generally be easily effected by means of suitable forceps, but when the dead bone is invaginated to any great extent by new bone, it may be necessary to enlarge the cloacæ by saws or cutting forceps, a proceeding which is sometimes tedious and difficult, so much so indeed that not unfrequently amputation must be resorted to. After removing the sequestrum the cavity remaining must be rendered aseptic and allowed to granulate up. The general condition of the patient must also receive careful attention.

#### OSTEO-MYELITIS.

Osteo-myelitis is inflammation of the marrow of bone. It may be acute or chronic ; the acute variety may be localized or diffuse.



*Acute localized Osteo-myelitis* is usually the result of an injury exposing the medullary cavity of a bone, such as compound fracture or amputation. The inflammation may terminate either in resolution, or in localized central necrosis.

*Acute diffuse osteo-myelitis* is dependent on the action of bacteria. There are two chief varieties. (a) The *septic* variety ; this occurs after compound fractures and operations on bones when the parts have not been kept aseptic, i.e. they have become inoculated with saprophytic bacteria from without. (b) The *infective* variety ; this is of the nature of a specific infective fever, and is due to the presence of bacteria (streptococci and staphylococci) in the blood, which have a special tendency to attack bones the vitality of which have been lowered by any local or constitutional cause; the periosteum and the bone itself are also inflamed at the same time (see Infective panosteitis). In both the septic and the infective variety, the inflammation spreads along the whole length and thickness of the bone, and leads to diffuse suppuration ; the veins in the interior of the bone participate in the process, and become blocked by infective or septic thrombi, which may be carried to distant parts, giving rise to septicæmia and pyæmia.

*Symptoms.* The symptoms of infective osteo-myelitis are the same as those of infective panosteitis (which see). In the septic variety there is high fever and swelling and œdema of the limb ; the wound looks unhealthy, and the periosteum recedes, leaving the bone bare ; from the open medullary cavity a fungating mass of granulations protrudes.

*Treatment.* In the septic variety the choice lies between amputation and scraping out the inflamed medulla. For the treatment of the infective variety, see infective panosteitis.



*Chronic osteo-myelitis* is hardly to be distinguished from osteitis, with which it is practically always associated.

#### INFECTIVE PANOSTEITIS.

This disease has received various names, such as diffuse periostitis, acute necrosis, etc. As the disease affects not only the bone, but also the periosteum and medulla, I have called it "panosteitis" (inflammation of all the parts of bone); the adjective "infective" is added to the name because the disease is due to the action of infective bacteria.

It usually occurs in debilitated children, and affects a bone the vitality of which has been lowered by exposure to cold, or some slight injury. Authorities differ as to whether the disease begins in the medulla and spreads to the periosteum, or *vice versa*, but all agree that both are soon affected. The periosteum becomes stripped from the bone by an accumulation of pus beneath it, whilst the nutrient vessels in the medulla are compressed by the inflammatory exudation inside the bone. The result is that a large portion, sometimes the whole, of the diaphysis dies. The epiphyses usually escape, because they have a separate set of nutrient arteries and the periosteum covering them is more adherent and therefore less easily stripped off; sometimes, however, the disease spreads to the neighbouring joints. Owing to the involvement of the veins of the medulla, pyæmia may occur and kill the patient (see infective osteo-myelitis). Should recovery occur, the necrosed shaft becomes encased in periosteal new bone (see necrosis.)

*Symptoms.* The disease begins by high fever, often accompanied by rigors and delirium. The local signs may at first be obscure, so that it is not uncommon for the disease



to be mistaken for acute rheumatism or some other febrile disease. Such a mistake is a serious calamity, for the proper treatment is delayed, and the effects of the disease are consequently more extensive and apt to prove fatal. The local signs, however, become sooner or later sufficiently obvious; the affected part becomes intensely painful, and swollen and oedematous; the signs of blood-poisoning supervene, and death ensues from pyæmia, unless the proper treatment can be successfully carried out.

*Treatment.* As soon as the nature of the disease has been discovered, a free incision must be made right down to the bone. Usually it will be found that pus has stripped up the periosteum to a considerable extent. If the incision has anticipated this condition, so much the better for the chance of the patient's recovery. Every means must be adopted to get the parts aseptic and to keep them so. If the patient escapes the dangers of pyæmia, the dead bone must be removed as soon as it is loose. Throughout the whole course of the disease, the patient's strength must be maintained by suitable nourishment, stimulants and tonics.

#### SUMMARY OF INFLAMMATION OF BONE.

Before dismissing the subject it will be well to remind the student that although osteitis, osteo-myelitis and periostitis have been separately considered for convenience of description, yet they often co-exist in the same bone at the same time.

The stages and results of osteitis may be summarized thus:

Congestion and Exudation	{ Resolution	
	{ Osteo-Sclerosis	
	{ Rarefaction—Caries	
	{ Necrosis	
	{ Osteo-Sclerosis	
	{ Suppuration	



## TUMOURS OF BONE.

Any of the connective-tissue tumours may occur as primary growths in bone. Epithelial tumours of bone are rare, and can only occur as secondary deposits.

### OSTEOMATA.

Osteomata are tumours composed of bone ; they may be circumscribed or diffuse ; the circumscribed osteomata (exostoses) may be composed of compact or cancellous bone.

*Compact Exostoses* grow from the bones of the skull and face. They are composed of compact bone, and are sometimes as hard and dense as ivory ; they have no definite Haversian systems ; they are usually sessile or broadly pedunculated. They grow very slowly and are painless unless they happen to press upon a nerve. No attempt should be made to remove them unless they cause pain or deformity. They are best attacked by means of drills and saws worked by the surgical engine ; the ordinary saw is powerless with the hardest exostoses.

*Cancellous Exostoses* grow from the long bones near the epiphysial cartilages, or at the insertion of tendons ; they are also frequently found on the ungual phalanx of the great toe. They are composed of cancellous tissue, limited by a layer of compact bone, which is either encrusted by cartilage or covered by periosteum. They grow either by ossification of the encrusting cartilage or by deposit of new bone from the periosteal covering. Their growth is slow and tends to cease when the ossification of the bone from which they spring is completed. They occur in young people and are usually single ; sometimes, however, they are multiple, and may affect nearly every bone in



the body. A cancellous exostosis should be left alone, unless it is large and unsightly, or inconvenient, or is growing quickly, or continues growing after complete ossification of the bone from which it springs. Removal may be accomplished by the saw, gouge or chisel. Multiple exostoses are not amenable to treatment.

*Diffuse Osteomata* affect the bones of the face. They consist of finely cancellated bone ; they have no definite limits, but gradually encroach upon the cavities of the orbit, nose, antrum, &c., until they are nearly filled up. The pathology of this condition is not understood, and no treatment is of any avail.

#### ENCHONDROMATA.

Cartilaginous tumours of bone are most commonly found growing from the interior of the metacarpal bones and phalanges of the fingers, and expanding the compact tissue. They are usually multiple and often congenital ; they grow slowly and sometimes calcify, but only rarely become ossified. The smaller ones may be enucleated from the interior of the affected bones ; the larger ones require amputation of the fingers or hand.

#### FIBROMATA.

Fibrous tumours of bone are almost limited to the jaws (*fibrous epulis*) and base of the skull (*naso-pharyngeal polypus*.)

#### SARCOMATA.

Sarcomata of bone are divided into periosteal and endosteal. *Periosteal Sarcomata* may be composed of round, spindle,



or mixed cells, and may grow from the deeper (*subperiosteal*) or superficial layers of the periosteum. They are most often found in the vicinity of large joints affecting the ends of the long bones. They form large rapidly-growing tumours which often invade the interior of the bone as well as the structures towards the surface of the limb. They are very malignant and are prone to disseminate throughout the body, but they do not often affect the lymphatic glands. They may calcify or ossify; fig. 24 represents a periosteal sarcoma of the upper end of the fibula which has become extensively ossified. The diagnosis is very difficult in some cases; it is often impossible to distinguish between a periosteal sarcoma and a mass of inflammatory exudation in connection with osteitis and periostitis until an incision has been made into the swelling. Periosteal sarcomata sometimes are so vascular that they pulsate; they may then closely resemble an aneurysm. The treatment consists in free and early removal; this usually necessitates amputation.

*Endosteal Sarcomata* are most often found in the upper end of the tibia, the lower end of the femur and the lower jaw. A few endosteal sarcomata are of the small round-celled variety; they are of rapid growth, involve the whole length of the interior of the bone without definite limitation or capsule, and are extremely malignant; but by far the commonest variety of endosteal sarcoma is the myeloid. This is of slower growth and less malignant than the periosteal sarcoma. It is usually limited to the articular extremity of the bone and distinctly circumscribed; it expands the bone equally in all directions (see Fig 25). The expansion may be so great as to reduce the layer of compact tissue to a mere shell, which can readily be indented by the finger, producing



a crackling sensation usually called "egg-shell crackling," but much more closely resembling the sensation obtained by buckling-in a hard felt hat. It is rare for the tumour to invade the neighbouring joint, in this way differing markedly



Fig. 24.

PERIOSTEAL SARCOMA.

A large ossifying Periosteal Sarcoma of the upper end of the fibula. The soft parts have been removed by maceration. The shafts of the tibia and fibula are roughened and joined together by osteoplastic periostitis. (From a specimen in the Museum of St. Mary's Hospital.)

from abscess of the articular ends ; it appears that cartilage does not offer a favourable soil for the growth of tumours, and is not readily absorbed under simple pressure. The thinned bone may fracture "spontaneously." On section



Fig. 25.

MYELOID SARCOMA OF TIBIA.

The upper extremity of the tibia is expanded by a growth containing numerous cysts. (Museum of St. Mary's Hospital.)

the tumour usually presents a dark red tint and contains several cystic spaces occupied by a blood-stained fluid. The symptoms are usually obscure ; a diagnosis is not often possible until the bone has been sufficiently expanded to crackle under pressure, or sufficiently weakened to lead to spontaneous fracture.

Sometimes it is possible to enucleate a small myeloid sarcoma, but in the majority of cases it is necessary to remove the diseased bone by excision or amputation.



## RICKETS.

Rickets is a general state of malnutrition and consequent arrest of development in which the bones become soft and bent. It occurs most commonly in children from eighteen months to three years old. It may be due to debility of the mother during pregnancy and lactation, improper feeding, and bad hygienic surroundings. The bones are not the only parts affected; the liver, spleen, and lymphatic glands become enlarged and contain an excess of fibrous tissue, and the muscles become soft, flabby and wasted.

In studying the changes which occur in rickety bones it is necessary to bear in mind the mode of growth of a healthy bone. A bone increases in length by formation of bone at the epiphysial cartilages, and in thickness by deposition of bone from the deeper layers of the periosteum; as the new bone is deposited at the periphery from the periosteum, so the older central portion becomes absorbed and the due proportion in size between the medullary cavity and the thickness of the shaft maintained. In rickets the line of ossification between the epiphysis and diaphysis is greatly increased in thickness and very irregular, small islands of cartilage frequently escaping ossification; the neighbouring medullary substance is abnormally vascular, and the new bone formed is deficient in earthy salts. The osteogenetic layer of the periosteum is thickened and produces soft spongy bone deficient in earthy material, and unable to bear the weight it has to carry.

*Symptoms.* The child is restless and fretful, especially at night, when he throws off the bedclothes. There may be

tenderness about the limbs and a great tendency to sweat, especially about the head ; the abdomen is large and tumid ;

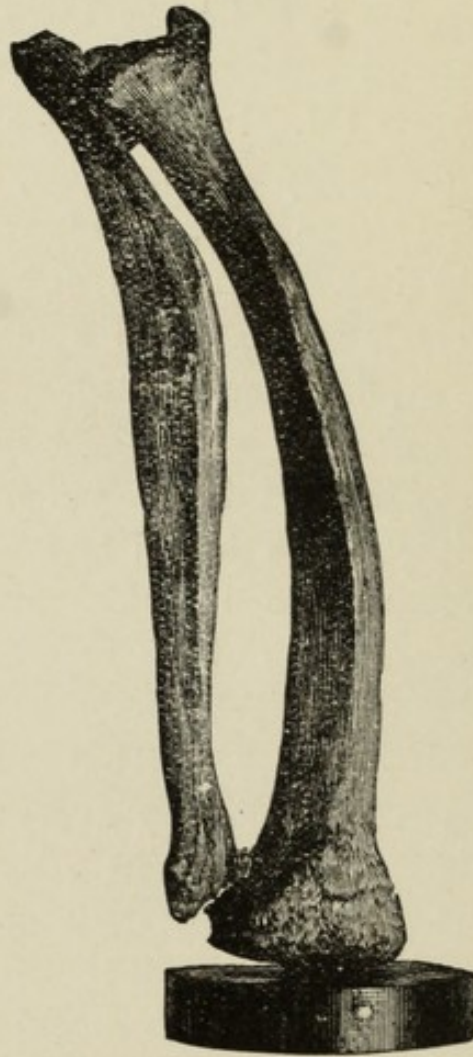


Fig. 26.

RICKETS.

The tibia and fibula of an adult who had suffered from rickets in childhood. Both bones are bent. The fibula is much thickened.

(Museum of St. Mary's Hospital.)

the dentition is delayed and the teeth ill-formed and prone to decay ; sometimes large quantities of phosphates are excreted



in the urine ; bronchitis, diarrhœa, convulsions, etc., are more common than in healthy children.

The ends of the long bones become thickened, so that the ribs are "beaded" at their junctions with the costal cartilages, and the wrists and ankles enlarged. Sooner or later the long bones bend, leading to knock-knee, bow-legs, etc. The ribs yield to the atmospheric pressure, the chest becomes narrowed from side to side, and the sternum unduly prominent (pigeon-breast). The spine becomes bent, and the pelvis flattened. The head is square and the frontal and parietal eminences unduly prominent ; the fontanelles are late in closing ; in places, especially at the back of the skull, ossification may be so imperfect as to leave thin places which can be indented by the finger (*craniotabes*). Rickety children frequently succumb to some of the various intercurrent affections to which they are liable ; but when recovery takes place the bones become consolidated in their deformed shapes and are often harder and denser than natural, their concavities being strengthened by a buttress-like ridge of bone.

*Treatment.* Proper food and healthy hygienic surroundings are the most important remedies. The general nutrition should be improved by the administration of cod-liver oil, phosphate of iron, etc. Deformity must be prevented by suitable splints or remedied by operative proceedings as occasion may demand.

### MOLLITIES OSSIUM.

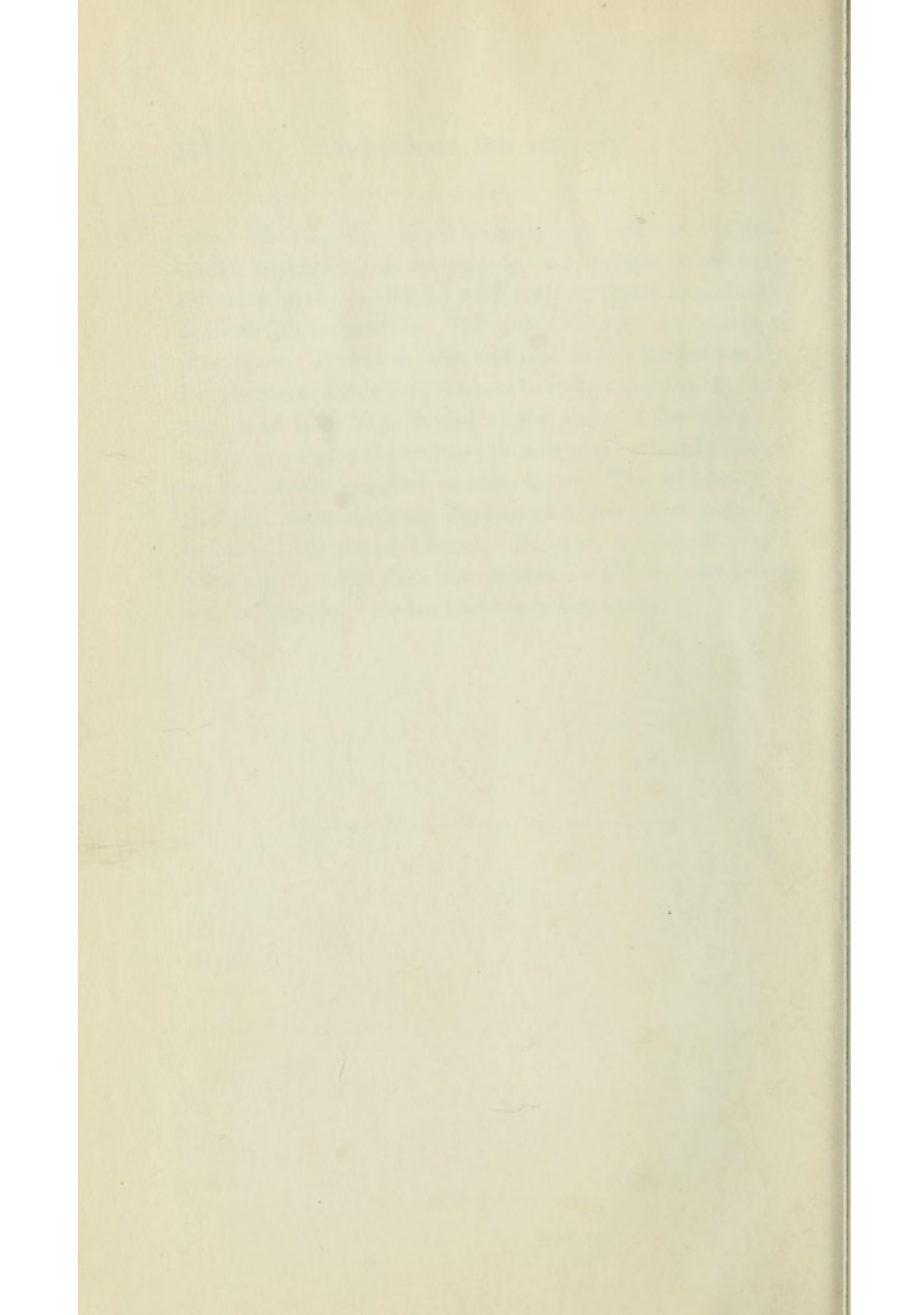
This disease occurs in adults, especially in females during the child-bearing period. Its causation is unknown. The bones become softened through re-absorption of their earthy

salts. The medullary tissue becomes soft, dark red and gelatinous, resembling the spleen-pulp, and replaces all the bony substance except a thin layer of compact tissue immediately beneath the periosteum. The urine contains an excess of phosphates. It will be observed that in this disease healthy bone becomes decalcified, whereas in rickets any healthy bone that may have been formed at the onset of the disease is simply removed in the ordinary process of growth and replaced by imperfectly calcified osseous tissue. The deformity in mollities ossium is usually extreme and often accompanied by fracture of the softened bones. Recovery is extremely rare ; death usually takes place from exhaustion or from interference with respiration. No treatment is of any avail.

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