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A MANUAL

OF THE

INJURIES AND SURGICAL DISEASES

OF THE

FACE, MOUTH, AND JAWS.

BY

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PHILADELPHIA:
THE S. S. WHITE DENTAL MFG. COMPANY.
1897.

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To my friends and confrères,

JONATHAN TAFT, M.D., D.D.S.,

AND

S. B. PALMER, M.D.S.,

In remembrance of the kindly sympathy and encouragement
so often extended in the earlier years of my professional
life, this book is affectionately dedicated by

THE AUTHOR.

PREFACE.

THE plan of this volume is the outgrowth of several years' experience as a teacher of Oral Surgery, in Medical and Dental Colleges. During these years the author has been more and more impressed with the disadvantages under which teachers and students have labored in the old system of teaching by didactic lectures. The same feeling has been growing, year by year, among many of the teachers in the American Medical and Dental Colleges, and many of them have expressed themselves as anxious to adopt a recitation system of teaching in their special departments. The greatest objection which has been raised to the inauguration of such a system of teaching has been the lack of text-books arranged upon a suitable plan for teaching by this method.

The author has endeavored to fill this requirement in the department of Oral Surgery by the preparation of this volume. In the selection and presentation of the various subjects comprising the work, he has constantly kept in mind the particular needs of the medical and dental student. In Part First will be found those subjects which belong to the General Principles of Surgery, while Part Second is devoted to the more common Injuries and Surgical Diseases which are associated with the Face, the Mouth, and the Jaws.

These subjects have been divided into short chapters, suitable to class recitation work, and each chapter is followed by a series of review questions covering the most important facts presented upon each topic. These questions can be used by the teacher as a basis for class quizzes; they will also enable the student to quiz himself upon every subject presented.

How well the author has succeeded in the undertaking he must leave to the criticism of his colleagues; but he trusts that the general plan will meet with the approval of all interested in a better system of teaching.

The very excellent illustrations upon Bacteriologic and Pathologic subjects have been made from photo-micrographs especially prepared for this volume by the author's friend and confrère, Dr. Vida A. Latham, of the Bacteriologic Laboratory of the Woman's Medical Department of the Northwestern University, and he takes very great pleasure in making this acknowledgment. To Dr. William H. Knapp, of Chicago, he is also indebted for valuable services rendered in photographing some of these slides and for the photo-micrographs of the karyokinetic figures; and to T. Charters White, of London, England, for several interesting photo-micrographs.

Especial thanks are due to the various authors quoted in the work, for much valuable information gathered from their labors, and also to Dr. N. Senn, Mr. Christopher Heath, Mr. J. Bland Sutton, Dr. W. D. Miller, and to their publishers, W. B. Saunders, Churchill & Co., Cassell Publishing Company, The S. S. White Dental Manufacturing Company, and F. A. Davis, for courtesies extended in permission to use many valuable illustrations.

JOHN SAYRE MARSHALL.

36 WASHINGTON ST., CHICAGO.

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INJURIES AND SURGICAL DISEASES OF THE FACE, MOUTH, AND JAWS.

PART I.

CHAPTER I.

SURGICAL BACTERIOLOGY.

THE promulgation of the Germ Theory of disease was the beginning of a great revolution in the practice of both medicine and surgery; a revolution which at the present time is still going on with increasing success, recording victory after victory, and constantly invading new territory heretofore occupied by dread pestilence and epidemic disease, and tearing from their grasp trophies in the form of the discovery of the causation of these dreaded maladies, and of the means wherewith to successfully combat them. The application of the principle of the germ theory of disease has had its greatest successes in the department of surgery; in fact, it has placed modern surgery upon the exalted pinnacle which it occupies to-day. Without the discovery of the pyogenic bacteria and of the other pathogenic forms now known to science, and without a knowledge of the principles of modern antiseptics, much of the success which has been achieved in surgery during the last two decades would still be an impossibility.

"In the light of the germ theory, disease may be considered to be a battle between the organism and an invading army of parasites, while the treatment of diseases resolves itself into the question of how best to assist the organism in overcoming the enemy which has entered its territory." (Gradle.)

Parasites.—*Definition.* Parasites are plants or animals which live upon other plants or animals.

Parasites may belong to either the animal or the vegetable kingdom. In the early history of the germ theory of disease, there was

great difficulty in classing them. In most instances the parasites which enter the animal organism are microscopic in size; hence they are referred to as micro-organisms, microbes, or bacteria. Bacteria are now generally considered as belonging to the vegetable kingdom. The bacteria belong to the smallest and simplest of all plants, the *Protophytes*. "Many of them are so small as to approach the limits of visibility, even when the highest powers of the microscope are used." When located in the animal tissues they are demonstrated with great difficulty, and only by the aid of special staining agents can they be differentiated from the cellular elements of the tissues; even then doubt sometimes shadows the certainty of the demonstration, and it becomes necessary to make experimental cultivations of the products of tissue disintegration in the case before a positive diagnosis can be reached. The *sphero bacteria,* or *micrococci,* are the smallest of all the bacterial forms. Fig. 1 represents some of the common forms of bacteria.

These organisms, the bacteria, are classed by Pasteur under two general heads, namely: *Aërobes* and *Anaërobes*.

The aërobic microbes require the oxygen of the atmosphere in order to maintain life, and therefore live upon the surfaces of substances. The yeast fungi are examples of aërobic microbes (Fig. 2).

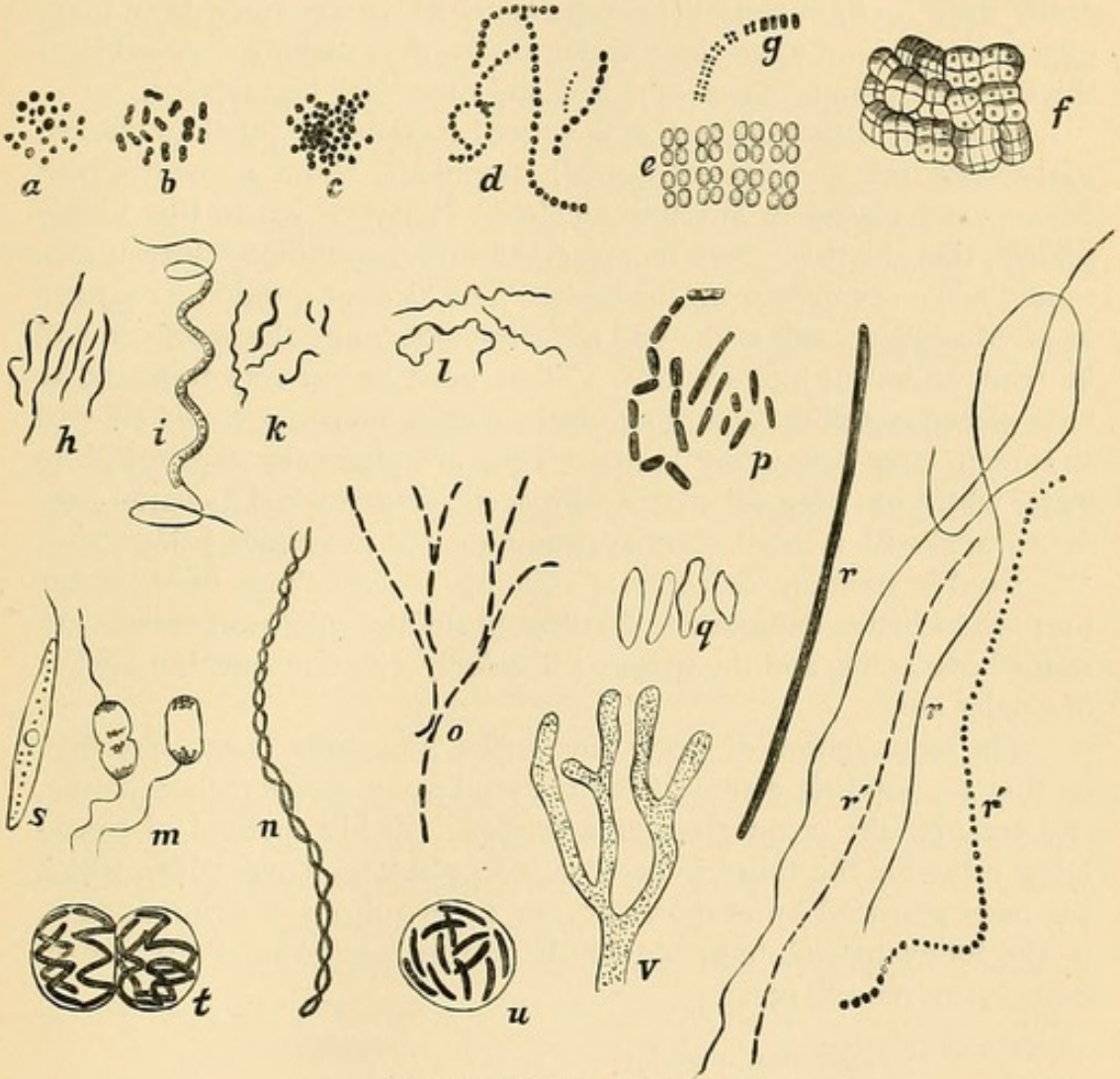
The anaërobic microbes do not require oxygen to maintain life, and therefore live beneath the surfaces of liquids and inside of living bodies. The Bacterium tetani is an example of the anaërobic microbes.

The greater portion of the bacteria are aërobic. Some of them are so dependent upon oxygen that the slightest diminution in the supply is sufficient to arrest or completely prevent their development. These have been called *obligate* aërobic bacteria. Others grow in media rich in oxygen, and also where there is no oxygen. These have been termed *facultative* aërobic bacteria. Nearly all the pathogenic forms of bacteria belong to the facultative variety. The tissues of the body contain a certain amount of oxygen, but this is soon consumed by the micro-organisms in their growth; consequently they would die if they did not have the faculty of living without oxygen under certain conditions. The anaërobic bacteria are exceedingly rare among the pathogenic forms.

Pasteur discovered that when artificial cultures of certain pathogenic bacteria were exposed for a considerable time to oxygen, generally from three to eight months, the virus became very much attenuated. His first experiments were with the microbe of chicken cholera and the bacillus of anthrax. He found that a chicken inoculated with the weak cultures of the chicken cholera bacillus was rendered immune to the action of the virulent virus, and that the attenuated culture of the anthrax bacillus, prepared in the same manner, rendered sheep

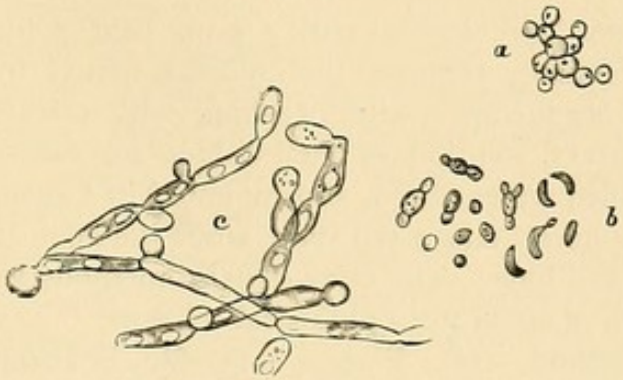
immune to anthrax, or if they contracted the disease after inoculation, it appeared in only a very mild form.

FIG. 1.



VARIOUS FORMS OF BACTERIA.

FIG. 2.



VARIOUS FORMS OF YEAST FUNGI.

Paul Bert has shown that oxygen, under a pressure of from 20 to 40 centimeters, destroys the vitality of the anthrax bacillus.

The Microscopic Study of Bacteria.—In order to successfully study bacteria, a good quality of microscope, with oil immersion lenses and Abbé condenser, is an absolute necessity. Anilin dyes are generally used to stain the micro-organisms, which are often very tenacious in their hold upon the staining fluids, thus making it possible to discover the minute forms which would otherwise be invisible.

Portions of tissue which it is desired to examine for the presence of bacteria are first cut into small fragments, about a quarter-inch square, and placed in absolute alcohol. It is best to do this immediately, that the tissue may be preserved in the condition which it presented when removed from the body. The alcohol should be changed as often as twice, and at the end of forty-eight hours the specimen will be ready to be cut into sections. These must be cut very thin, and at once placed in a dilute solution of fuchsin or gentian violet, and allowed to remain from one to six hours. They are afterward decolorized in water which has been acidulated with acetic acid, washed in water, then dehydrated with alcohol, clarified, and mounted in Canada balsam.

Double staining, or contrast staining, is sometimes used for the purpose of better definition. By this means the micro-organisms are stained one color, and the tissues a decidedly different one, but always of a paler hue.

The examination of blood, pus, urine, and sputa is accomplished by first evaporating a film of the material upon a cover-glass, fixing, and treating the cover-glass as a section. Ziehl's method of examining urine for the tubercle bacillus is to place the cover-glass, which has been prepared by evaporating one or two drops of urine upon it, in the following solution, previously warmed, allowing it to remain from five to ten minutes:

Fuchsin, 1 gram;
Carbolic acid solution (5 per cent.), 80 c.c.;
Alcohol (95 per cent.), 20 c.c.

It is afterward decolorized with a 5 per cent. solution of sulfuric acid, which effectually removes the coloring matter from everything but the micro-organisms. After washing with distilled water, it is placed in a watery solution of methyl-blue for five minutes, again washed in distilled water, dried, and mounted in Canada balsam. By this method the bacilli take a red stain, while the deposit in which they are held is colored blue. The same general methods are applicable in the examination of sputa from phthisical patients.

Gram's Method.—(*a*) Place a cover-film in absolute alcohol for one or two minutes.

(*b*) Stain in anilin gentian violet for one or two minutes.

(*c*) Remove superfluous stain by draining.

(*d*) Now place in Gram's solution of iodin for one-half to one minute,—until the specimen turns black.

(*e*) Soak up the superfluous iodin solution.

(*f*) Wash in alcohol until the film is almost colorless,—until no more stain comes away. Dry and mount in xylol balsam.

(*g*) If to double stain, pass quickly through a dilute alcoholic solution of eosin. The leucocytes and ground substance will be colored pink, the gonococcus and also the chromilin violet.

(*h*) Wash in water; examine if deep enough. Dry thoroughly and mount in xylol balsam. Examine with oil immersion.

Formulæ.—*Gentian Anilin Water.* 1. Mix 4 c.c. anilin oil with 100 c.c. distilled water. Shake for one or two minutes.

2. Filter resulting emulsion through filter-paper moistened with distilled water.

3. To 100 c.c. of anilin water add 11 c.c. of a concentrated alcoholic solution of gentian violet. Shake. Mix thoroughly. Always filter before using. This does not keep well, consequently only small quantities should be prepared at a time.

Gram's Iodin Solution.
Iodin crystals, 1 gram;
Iodid potassium, 2 grams;
Distilled water, 300 c.c.

Functions of Bacteria.—Certain species of bacteria are disease producing, or *pathogenic;* others are color producing, or *chromogenic.* Another species is ferment producing, or *zymogenic;* while still others have as yet no discoverable function.

When arranging bacteria according to their relation to disease, it is customary to class them under two general heads:

First. *Non-pathogenic,* or those which do not as a direct cause produce disease.

Second. *Pathogenic,* or those which are the direct cause of disease.

Fermentation and putrefaction are the results of the growth of micro-organisms in the substances which ferment or putrefy.

Among the non-pathogenic micro-organisms are included the *saprophytic* germs. These organisms, which may become indirect causes of disease, can live and grow only in dead and dying tissues. Organisms of this character entering a wound in which there are pent-up discharges and dying tissues, increase with great rapidity, and produce certain substances of a poisonous and irritating character, called *ptomaines,* the absorption of which by the system gives rise to symptoms which are denominated as *septic intoxication, ptomaine fever,* or *septicemia.*

Pathogenic micro-organisms grow and flourish in dead and dying

matter, and invade the living tissues and destroy them. They also enter the circulation by direct inoculation through wounds and abrasions, and are carried to all parts of the body, and wherever deposited increase in numbers with amazing rapidity, forming fresh foci for the production of poisonous and irritating substances. The chief difference therefore between the *saprophytes* and *pathogenic* germs is that the former act as *indirect* causes of disease by the production of poisonous substances, but they have no power to penetrate the tissues or enter the circulation; while the latter possess this power, and act as *direct* disease-producing agents.

The pathogenic micro-organisms may be divided again into two general classes:

First. *Micrococci.*

Second. *Bacilli.*

Each of these classes has been divided and subdivided by the bacteriologist into an almost endless variety. This classification is the result of a thorough and careful study as to their size, form, and length, their growth, groupings, and action in the various culture-media, their chemical reaction, the color imparted to the culture-media, their susceptibility to the various staining agents, and their action upon fermentable substances and living organisms.

New forms of bacteria are constantly being discovered, and further research into the life and habits of old forms develops new features and modes of action which a little while before had not been dreamed of, while the etiology of certain diseases which were before considered as obscure are one by one being cleared up by the discovery of a specific microbe, which, when introduced into the system in sufficient quantities, will produce the disease. It has been recently announced that Kitasato has discovered the bacillus of the plague, and that it resembles the micro-organism of chicken cholera.

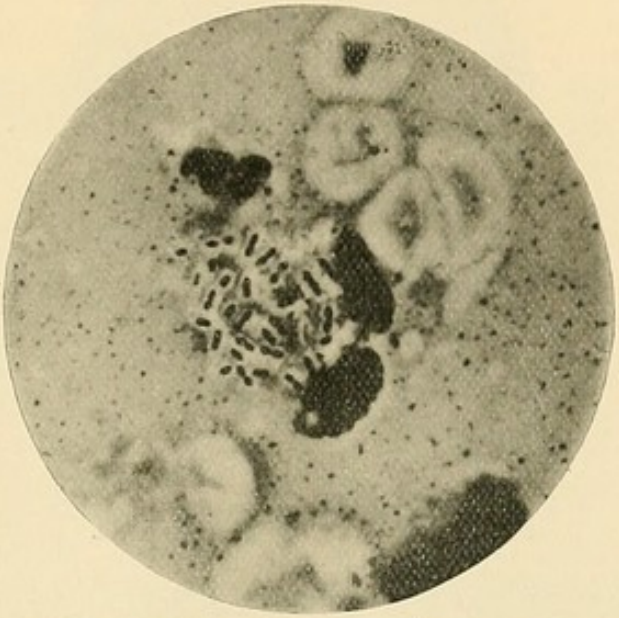
The *Micrococcus* is an individual bacterium, the smallest of all the bacterial forms, having spheric elements—tiny, globe-like masses of matter—in some cases isolated, in others united in twos or in larger numbers, or disposed in chains or chaplets, or deposited in masses of zooglea,—a gelatinous matrix secreted by the bacteria themselves. (*a*, Fig. 1.) When united in twos they are called *Diplococci* (*b*, Fig. 1). Sometimes they are united in such a way as to resemble a bunch of grapes. They are then termed *Staphylococci* (*c*, Fig. 1). If arranged in chains or chaplets they are denominated *Streptococci* (*d*, Fig. 1).

The pneumococcus or diplococcus of pneumonia (Fig. 3) is a good representation of the diplococci.

The bacillus of tetanus (Fig. 4) would seem from its form, which is like a drum-stick, to be a combination of a micrococcus and a bacillus, but in reality it is a bacillus with a spore at one end.

The bacillus of cholera (Fig. 5) seems to be a combination of the same character, but having a curved stem resembling a comma; hence it has been named the comma bacillus, or Koch bacillus, after the

FIG. 3.



DIPLOCOCCUS PNEUMONIA FROM LUNG. × 1000.

name of its discoverer. Fig. 6 represents various micro-organisms of the mouth.

The Bacterium lactis, which is an active agent in the production

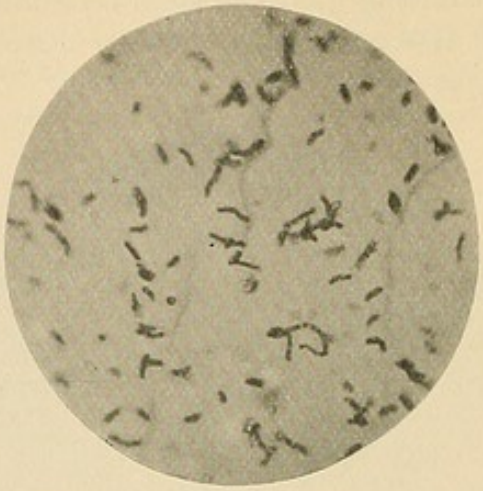
FIG. 4.



TETANUS BACILLUS.

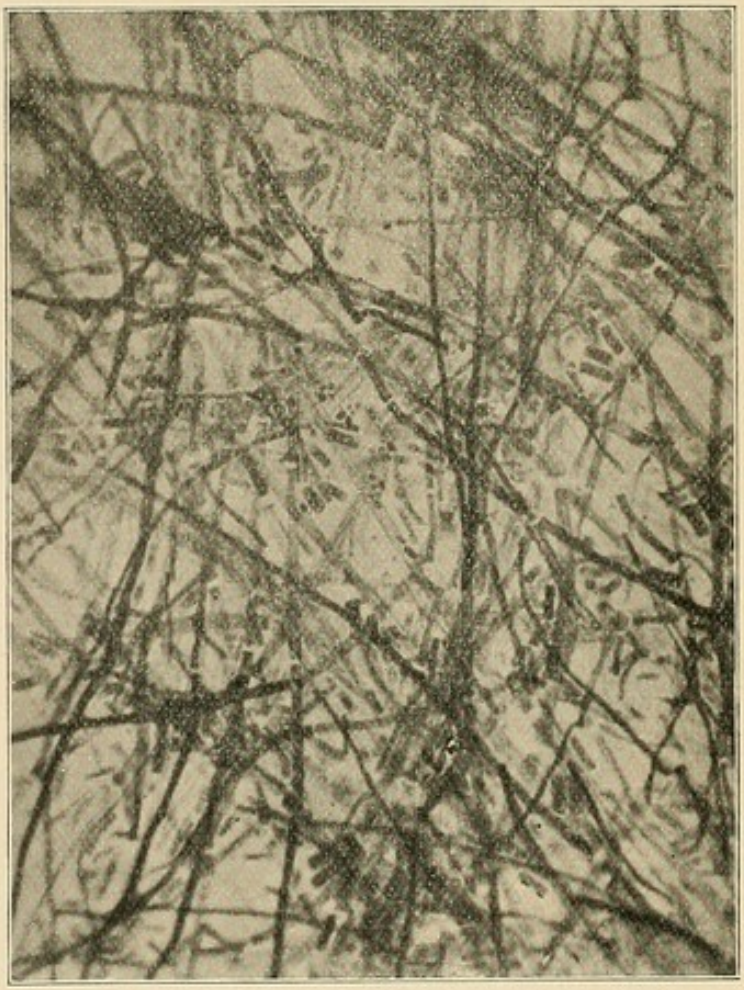
of dental caries, is a short, straight, rod-like bacillus. Its function is that of forming lactic acid, and it is the organism that causes the souring of milk. The Oidium lactis (Fig. 7) is common in the mouth.

FIG. 5.



BACILLUS ASIATIC CHOLERA. × 1200.

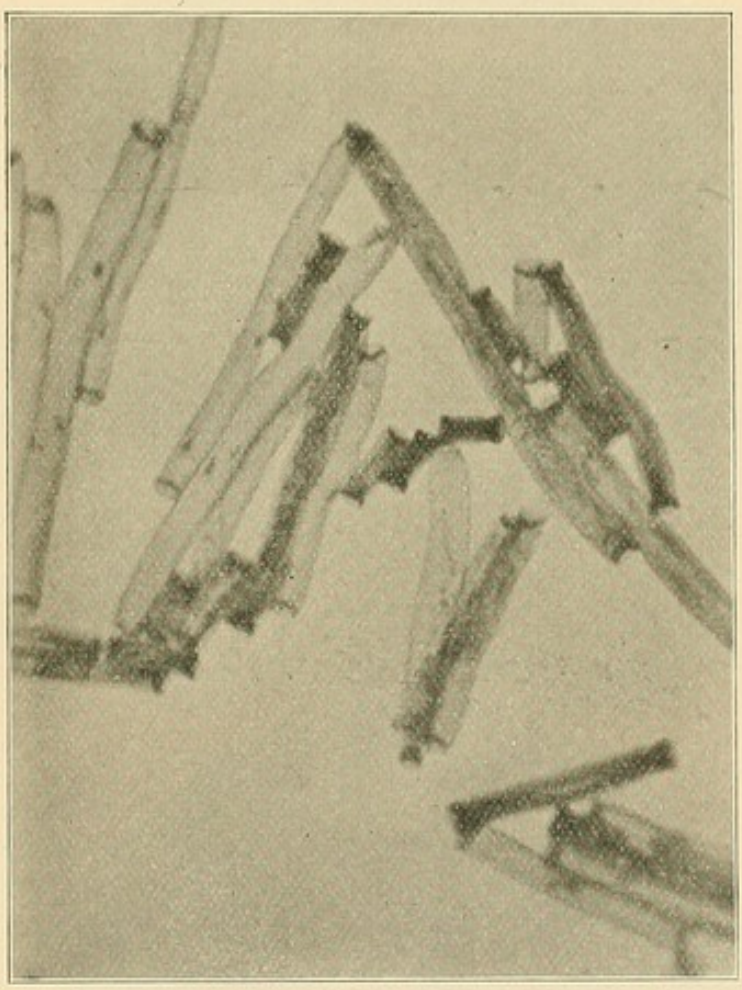
FIG. 6.



VARIOUS MICRO-ORGANISMS OF THE MOUTH. LEPTOTHRIX BUCCALIS, MICROCOCCI AND BACILLI, ETC. × 1200.

The Leptothrix buccalis (Fig. 8), another bacterium frequently found in the mouth, and associated with dental caries, is a long, slender, thread-like bacillus, usually found grouped in masses. Fig. 9 shows the Leptothrix gigantiæ.

FIG. 7.



Active.

Old or Matured.

OIDIUM LACTIS. (MILK MOLD.) × 1200.

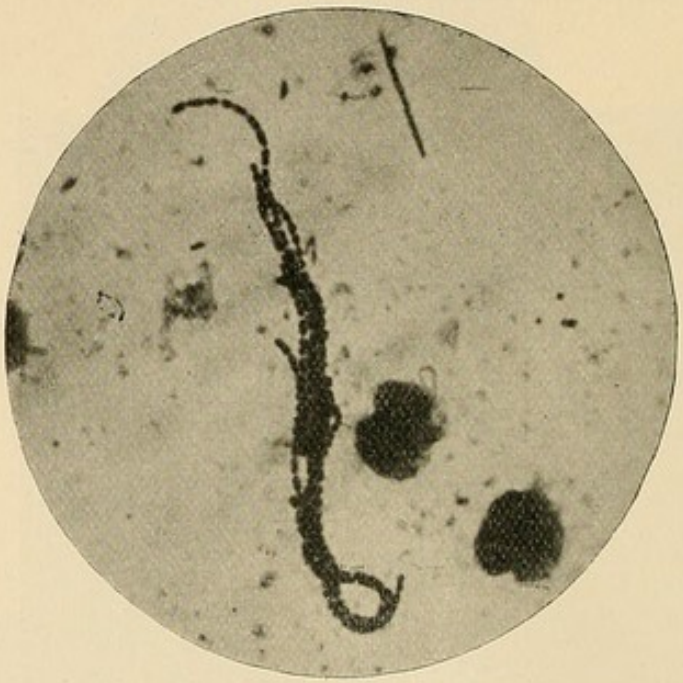
One of the difficulties which the bacteriologist and the pathologist have to meet is the seeming identity of certain forms of bacteria in diseases presenting dissimilar characteristics; for instance, the Streptococcus pyogenes (Fig. 10) seems to be identical with the streptococcus of erysipelas, the only difference being one of size, the coccus of erysipelas being the larger.

The cocci multiply only by fission or division, a process similar to karyokinesis (Fig. 11). The cell elongates prior to its segmentation, when a constriction appears in the center, which becomes deeper and deeper until complete division of the cell into two equal parts takes place. These new cells soon attain the size of the parent cell.

The staphylococci, diplococci, and streptococci are generally

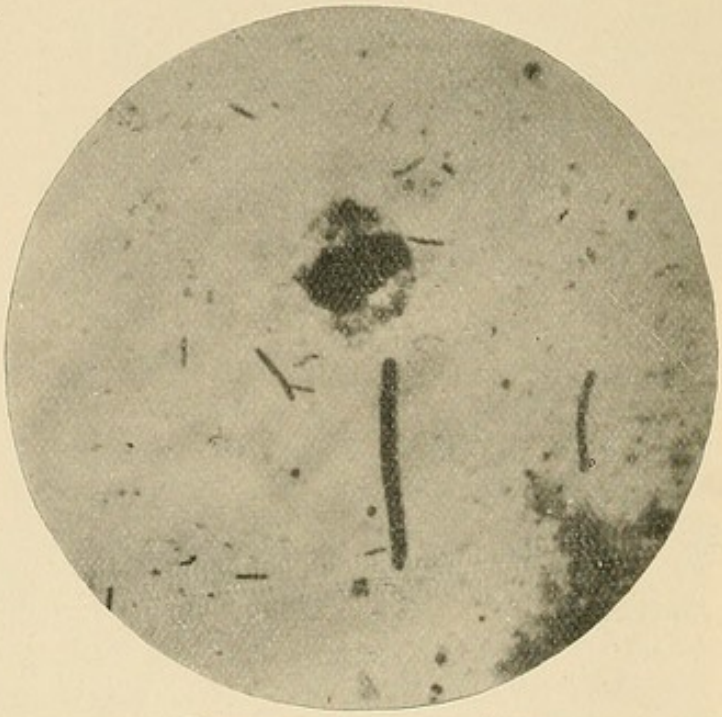
found in the broken-down tissue and discharges which result from inflammatory action, particularly in pus formations.

FIG. 8.



LEPTOTHRIX BUCCALIS. × 1200.

FIG. 9.

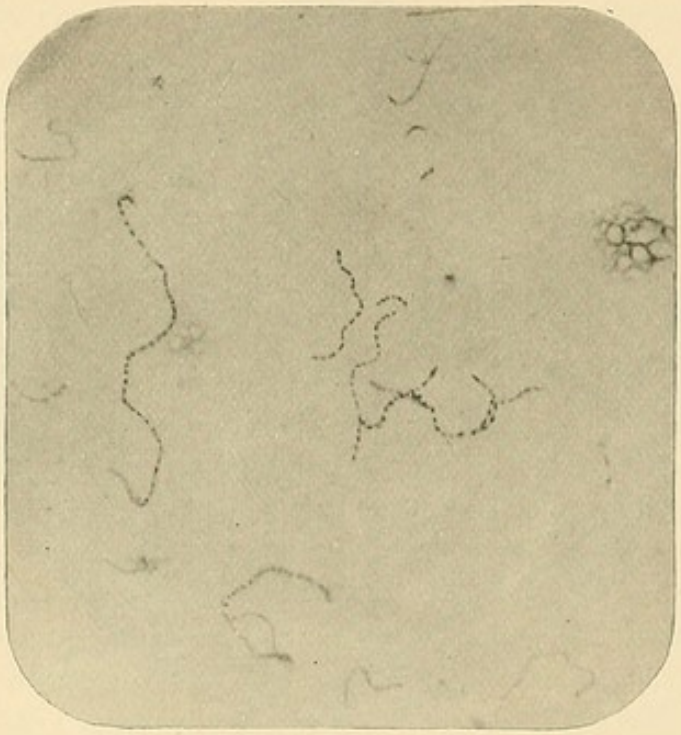


LEPTOTHRIX GIGANTIÆ. × 1200.

The *Bacillus* is an individual bacterium of rod-like form, and includes all the elongated forms of bacteria, except such as are spiral and have a gyratory motion, which are classed with the genus Spirillum.

A *Spirillum* is an individual bacterium whose elements are curved, often forming a spiral of several turns. The comma bacillus of Koch, found in the discharges of cholera patients, is a spirillum, and represents the simple curved variety; while the spirillum of Miller, found in

FIG. 10.

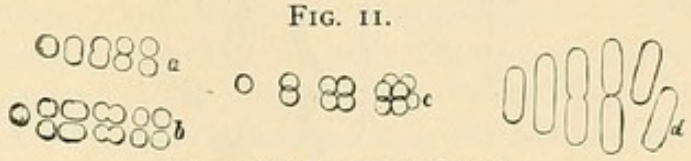


STREPTOCOCCUS PYOGENES. × 1200.

carious teeth, represents the spiral form. Some of the spirilli have cilia attached (Fig. 12).

Bacilli are rigid or flexible, motile or non-motile, and reproduce their kind either by direct fission or by endogenous spore-formation,—the formation of a cell within the body of the parent cell (Fig. 13). This process is a very rapid one. Flügge observed the process of segmentation in a coccus to occur in twenty minutes. Cohn has made the calculation that if it should take one hour to complete the process of

FIG. 11.

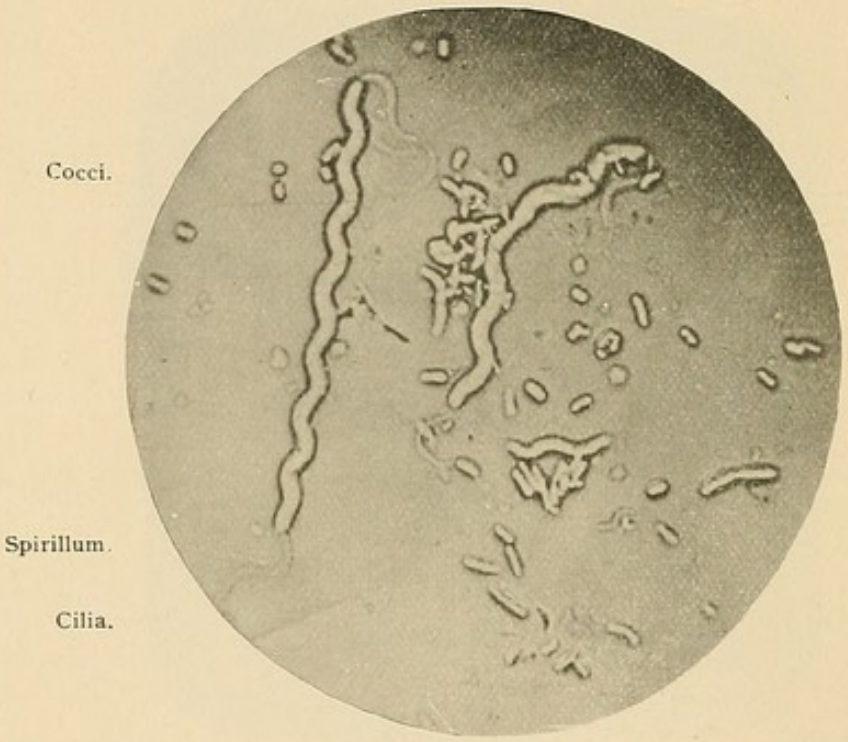


FISSION OF BACTERIA (COCCI).

segmentation, and for the new cell to attain the size of the parent cell, one coccus, multiplying by this process, would in one day produce 16,000,000 cocci; at the end of two days 281,000,000,000, while at the end of the third day it would have reached the enormous number of 46,000,000,000,000.

The spore possesses an exceedingly dense enveloping membrane, which protects it from deleterious external influences until such time as it finds a soil favorable to its growth and development. The cell is usually enlarged in the center or at one end by the presence of the

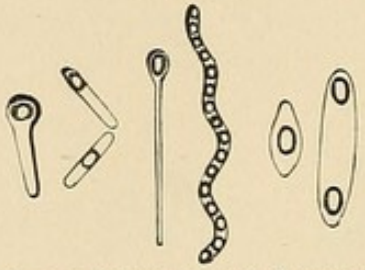
FIG. 12.



SPIRILLI CILIA. × 1250.

spore, and when the latter reaches its full development, gelatinous softening of the cell-membrane takes place, the cell breaks up, and the spore is set free. During the process of development of the spore into a bacillus it loses its tough enveloping membrane and is therefore more readily destroyed. The majority of bacteria grow at a temperature

FIG. 13.



SPORE FORMATIONS IN BACILLI.

of 37° C.=98° F. Spores resist the action of germicidal agents to a much greater degree than the bacilli which produce them. Mature bacteria cannot resist a temperature of 77° C., 170° F. Most of them are destroyed when exposed to 55° C., 131° F., while spores have been known to resist a temperature of 100° to 120° C., 212° to 236° F. A

temperature of 100° C., 212° F., if maintained for ten to fifteen minutes, will effectually destroy the most persistent of spores.

Spores which have gained an entrance to the body may remain dormant for years, and give rise to no untoward symptoms until aroused to activity by conditions which favor their growth and development. Two conditions are necessary for the germination of bacteria, viz: a certain amount of heat and moisture. Both must be present. The requisite amount of heat minus the moisture, or the moisture without the heat, are in neither case favorable to their development. This is eminently true in the treatment of dental caries and devitalized teeth; with thorough desiccation of the cavity of decay or of the root-canal, and the prevention of the ingress of moisture, caries will be arrested in the one case, and suppuration prevented in the other. In no department of surgery is thorough antisepsis more important than in operations upon the teeth.

The Pyogenic or Pus Microbes.—The micro-organisms with which the surgeon has most frequently to contend are those which cause suppuration. Their effect upon the inflammatory exudates, leucocytes, and cellular elements of the tissues, is one of specific action by which they convert them into pus-corpuscles. They are therefore called pyogenic or pus-microbes. Of these there are several varieties, most of them of the globular or coccus form. The number of bacterial forms which have been found in connection with surgical diseases, and fully identified as their exciting cause, is not large; yet the statement may be safely made upon the basis of our present knowledge, that all traumatic infective diseases can be traced to the action of micro-organisms which have gained an entrance to the tissues.

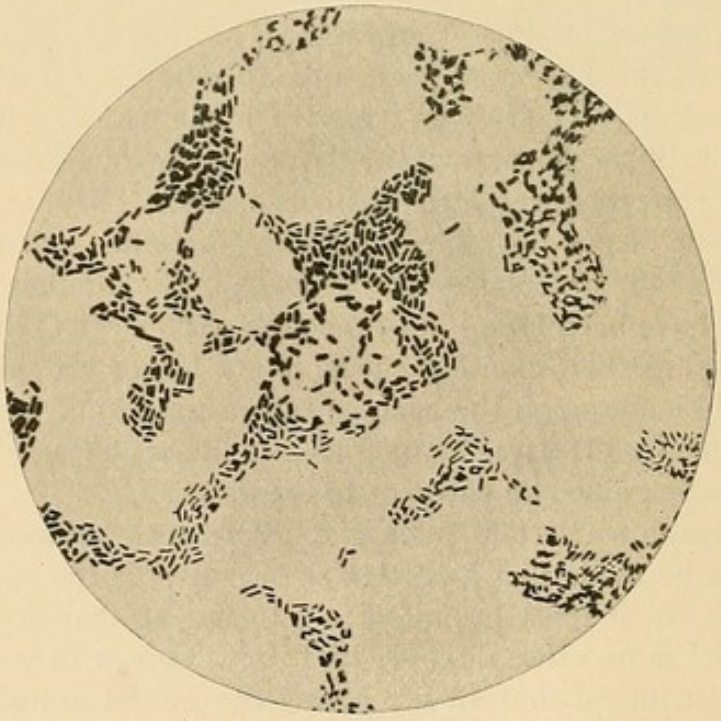
Koch lays down certain rules as a crucial test, before the positive assertion can be made that a particular organism is the specific cause of a disease: 1st, it must be found in all cases of that disease; 2d, it must be found in no other disease; 3d, it must appear in such quantity and be so distributed that all symptoms can be accounted for by its presence; 4th, the organism must be capable of being isolated from the diseased tissues, and be grown upon some of the artificial culture-media; 5th, when injected into an animal it must be capable of reproducing the disease. All of these conditions can rarely be fulfilled in many cases, yet when a certain form or variety of bacteria is constantly present in a particular disease, it is pretty good evidence that it is the cause of that disease.

Infection.—The effect of infection with the pyogenic cocci will always vary with the number of the microbes entering the tissues, the favorable or unfavorable conditions of the tissues for their growth, and the general susceptibility of the organism. These facts have been abundantly proved by repeated inoculation experiments upon

animals. Watson Cheyne found that the number of the bacteria injected makes a very great difference in the intensity of the symptoms and the character of the disease. He arrived at a general idea of the number of bacteria in a given quantity of fluid by means of the plate culture, the fluid having been diluted for the purpose; a definite quantity of this fluid was injected into an animal, and at the same time plates were made from an equal quantity. The number of organisms in the fluid injected was thus quite accurately determined.

In the case of the *Proteus vulgaris* of Hauser, Fig. 14 (a bacterium commonly associated with putrefaction), he found that a dose of 1-10 c.c. of an undiluted culture contained about 250,000,000 bacteria, and when injected into the muscular tissue of a rabbit quickly proved fatal; while a dose of 1-40 c.c., containing about 56,000,000,

FIG. 14.



BACILLUS PROTEUS VULGARIS OF HAUSER. × 1000.

caused very extensive abscess and resulted in the death of the animal in from six to eight weeks. Doses which contained less than 18,000,000 very rarely produced any effect.

He also demonstrated that with cultures of the *Staphylococcus pyogenes aureus* it was necessary to inject a dose sufficient to include at least 1,000,000,000 cocci into the muscle of the rabbit to procure a speedy fatal effect; while a dose of 250,000,000 caused the formation only of a small circumscribed abscess. The *Staphylococcus pyogenes albus* was found to produce the same results, but with somewhat larger doses.

Another interesting fact discovered by the same investigator was,

that concentration of the bacterial material in a certain locality was necessary to produce the most marked results. Dividing the dose and injecting it at different times or in different locations at the same time, did not produce the same results as when it was all injected into a single locality.

The susceptibility of the human organism to the action of the pyogenic cocci is not very great, and the results produced by them will vary according to the numbers introduced and the conditions of the tissues at the time. The introduction of small numbers of these microbes, if accompanied with the toxic substances which are present in the virulent cultures, is more liable to cause suppuration than when not so accompanied, and the extent of the inflammatory process will bear a close relation to the quantity and quality of these substances. (Warren.)

The entrance of a few pus-microbes into a wound may be entirely harmless unless the conditions are favorable for their growth and multiplication. Such conditions would be represented by retained exudations, a blood-clot, or irritation of the wound from sutures or dressings.

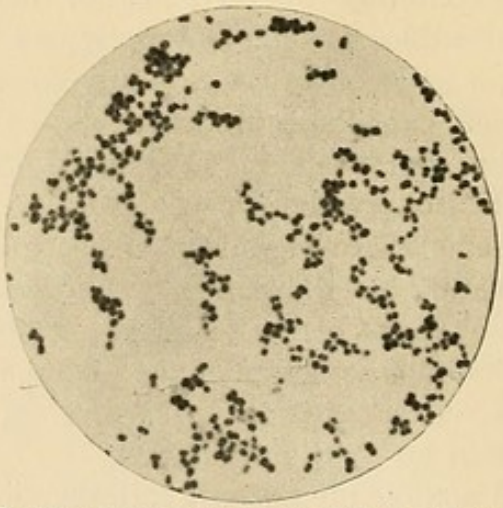
Pathogenic micro-organisms are frequently found in the blood of healthy living persons, but it remains a disputed question as to whether they can exist in the body without causing disease. Experiment has proved that pathogenic micro-organisms are harmless so long as they remain in the circulating blood, but if they become localized then their specific pathogenic action becomes manifest. Pathogenic spores may remain in the healthy body for an indefinite period, in a quiescent state, or until some pathologic change takes place in the tissues, furnishing the soil and conditions for their germination.

Fodor injected pathogenic bacteria into the circulation of rabbits, for the purpose of studying their effect upon the tissues and the mode of elimination. In a majority of instances he found that they had entirely disappeared from the blood at the end of twenty-four hours, and he is of the opinion that they were destroyed by the blood-corpuscles. Metschnikoff advanced the theory that the leucocytes, which are always present in large numbers in acute inflammatory processes, and which he denominated phagocytes, have the power of appropriating and destroying the invading bacteria.

The relative frequency of the presence of pyogenic cocci in cases of suppuration in the human species is shown by Steinhaus to be as follows: Out of 330 cases reported by different observers, the *staphylococci* were present in 66.5 per cent.; the *streptococci* in 20.4 per cent., and a mixture of these two forms in 9.5 per cent., while the *tenuis* was present in only 1 per cent., and the other forms even more rarely. In other words, pus micro-organisms were present in 97.4 per cent. of cases.

The *Staphylococcus pyogenes aureus* (Fig. 15), yellow cocci,—so called from the fact that it is arranged in clusters, and gives a yellow color when cultivated in beef-gelatin,—is the most common of all the pus-microbes. It is globular in shape, and its diameter ranges from 0.7 to 0.87 micro-millimeter. The size depends upon the age of the coccus and the soil in which it grows. It multiplies by fission, but the line of division is difficult to make out. It grows readily upon beef-gelatin at the house temperature, but is more active when grown in a temperature like that of the body, and does not require a large amount of oxygen to maintain a vigorous growth. It has the power of liquefying gelatin by virtue of its peptonizing action, and it receives the color of nearly all the staining agents very readily, and is well adapted to the Gram method. It is also very tenacious of life, and requires to be subjected to the boiling temperature for several minutes in order to

FIG. 15.



STAPHYLOCOCCUS PYOGENES AUREUS. × 1200.

destroy its vitality. Cultures of this coccus have a peculiar and disagreeable odor like that of sour paste. It is found abundantly outside of the body. Its most common seat is the superficial layers of the skin, particularly in those parts of the body which are kept moist, like the axillæ, between the buttocks, etc.; also under the free ends of the finger-nails; in the mucus of the nasal passages, pharynx, mouth, and digestive tract. It has likewise been found in the air, especially of hospital wards which were in an unsanitary condition; in garden soil, in the dirt of the streets, in dirty dish-water, and in fact almost everywhere.

The *Staphylococcus pyogenes albus*—white coccus—cannot be distinguished from the *aureus*, except that it does not develop the yellow or golden-colored pigment. It would seem to be a variety of the aureus but for the fact that it cannot be so cultivated as to give the color of the yellow coccus. It always maintains its white color in any

culture-medium upon which it will grow. It has the power of liquefying gelatin. It is found less often than the aureus, is not so virulent, and the disturbances in the tissues caused by its presence are less pronounced.

The *Staphylococcus viridis flavescens*,—greenish-yellow coccus,—found in the vesicles of varicella, is an exceedingly rare variety. According to Babes it occupies an intermediate position between the aureus and albus. The cocci are irregular in shape, and larger than the aureus. When cultivated upon agar-agar, it forms a delicate film. Its characteristic color is a greenish-yellow pigment.

The *Staphylococcus pyogenes citreus*—lemon-colored coccus. This variety seems to be in all respects like the aureus and albus in its behavior, with the exception that it develops a pale-yellow or lemon-yellow pigment when cultivated in beef gelatin. It liquefies gelatin more slowly than the aureus or albus.

The *Staphylococcus cereus albus* et *flavus*—white and yellow cocci. These are two rare and unimportant forms. The *albus* is found in the pus of acute abscesses, and Tils discovered it in hydrant water. The *flavus* was also found in acute abscess. Passet, the discoverer, has only found them in two cases of abscess; other investigators have been unable to find them. Under the microscope they cannot be distinguished from the other varieties. When cultivated in artificial media, they each develop their characteristic pigment, and produce a dull, waxy growth when cultivated upon the surface of gelatin plates.

The *Micrococcus pyogenes tenuis*. This coccus obtained its name from the great delicacy of its growth. It was discovered by Rosenbach in the pus of an abscess, and is another rare form of pyogenic coccus. It is more than probable that like the cereus it may have had only an accidental presence in an abscess, and not be in any sense a pus-microbe. Under the microscope it presents a somewhat irregular shape, and is larger than the aureus. It produces a thin, transparent, varnish-like film upon the agar culture.

The *Streptococcus pyogenes* (Fig. 16) is a very important variety of the pyogenic cocci. It is usually found alone, but occasionally it is associated with the staphylococci. The arrangement of the organism is in chains or rows, usually from six to ten being attached together. They measure in diameter about one micro-millimeter. Microscopically they are identical with the streptococcus of erysipelas, the only discoverable difference being one of size, the erysipelas organism being the larger. The streptococcus grows at house temperature, but is more active at a temperature of from 30° to 37° C. On culture-media the coccus reaches its full development in from four to five days. It is not particularly sensitive to the absence of oxygen, but nevertheless grows best upon the surface of the gelatin. At first it has

a transparent whitish appearance, but later this color changes to a faint brown. It grows most readily in bouillon, multiplying with great rapidity. The organism is found under normal conditions in the saliva and mucous secretions of the mouth and nasal passages, in vaginal mucus, and in the urethra; it is also found as a "mixed infection" associated with the pathogenic organisms of typhoid fever, pneumonia, tuberculosis, scarlet fever, and diphtheria, and may therefore be an important agent in causing the various complications of these affections.

FIG. 16.



STREPTOCOCCUS PYOGENES. × 1200.

The *Bacillus pyocyaneus* is a widely distributed form, but not necessarily a pus-producing microbe. It is the organism of blue or green pus, and is found in wounds with purulent or serous discharges, in the perspiration, and in the viscera of human cadavers. It is a small, slender rod with distinctively rounded ends, and may occur in chains or rows, usually arranged five or six in a row. It has active motility, and

produces upon gelatin a beautiful green fluorescent pigment. The pigment is formed when the organism is in contact with the oxygen, and this may be seen upon the edges of dressings and bandages. It grows readily at house temperature, and belongs to that class which will grow and multiply with a scant amount of oxygen. Spores have not been seen to form. The coloring matter produced by this organism has been termed "pyocyanine." It was discovered by Bouchard that cultures of the Bacillus pyocyaneus will prevent the development of anthrax or splenic fever, if injected into the tissues of animals already infected with virulent cultures of the anthrax bacillus; and that the disease could be cured by the same means even after it had developed.

The *Bacillus pyogenes fetidus* is a rare and unimportant organism found in ischiorectal abscesses. Upon gelatin cultures it forms upon the surface a white or grayish film of delicate growth. When grown upon agar or potato, it produces a light brown color and has an offensive odor.

The *Micrococcus tetragenus* is also a somewhat rare form, and was first found by Gaffky in a tuberculous cavity of a lung; it is also occasionally seen in both morbid and healthy expectorations. Steinhaus found it in an acute abscess near the angle of the jaw. Iakowski also found it in two cases of acute abscess, one of the finger, the other in the palm of the hand. This organism is characterized by being grouped in fours and involved in a capsule, hence its name, tetragenus. In culture-media the coccus does not grow in any regular order, but when found in the tissues it is always in groups of four imbedded in a gelatinous envelope. It takes the stain of all anilin dyes, and also of the Gram method. It is an aërobic organism, and when grown upon gelatin appears as thick, globular, whitish masses with a somewhat glistening surface, but does not cause liquefaction of the gelatin.

Steinhaus and others have proved by experimental research that pus may be produced without the aid of the pus-producing micro-organisms, by the introduction into the tissues of certain irritating chemical substances, but that the pus so produced was aseptic, and that inoculation with it would not produce an infectious inflammation. It was also found that injection of the chemical substances elaborated by the pyogenic cocci when separated from them would produce pus of a non-bacterial character. Practically, however, this knowledge is of little value from the clinical standpoint; as acute suppuration without the presence of the pyogenic micro-organisms in the system is never observed. Mechanical irritation or the presence of foreign bodies in the tissues cannot produce an infectious pus without the aid of micro-organisms. The power of the pyogenic cocci to produce pus lies in their ability to liquefy the fibrinous elements of the tissues and the inflammatory exudates. (Senn.)

The presence of the pyogenic cocci is exceedingly rare in cold abscesses, and for this reason it has generally been supposed that they were only produced by the *Bacillus tuberculosis,* but Ernst and several other observers have found the Staphylococcus aureus and albus in cases of psoas abscess. The failure to obtain cultures of the pyogenic organisms in cold abscess may possibly be due to the death of the microbes as a result of the age of the abscess. Acute inflammation often immediately follows the opening of such abscesses, either from infection from the outside, or growth of latent spores previously deposited, and which have taken on active growth and multiplication as a result of the changed surroundings which have furnished a favorable soil for their development.

REVIEW.

CHAPTER I.

What has the Germ Theory of disease accomplished for Medicine and Surgery?

In the light of this theory, what may disease be considered?

What are parasites?

Are bacteria classed as animal or vegetable organisms?

To what order of plants do they belong?

What can be said of their size?

How can they be demonstrated in the tissues?

What other methods are sometimes necessary to demonstrate their presence?

What forms of bacteria are the smallest?

What other terms are applied to these organisms?

How are these organisms classed?

What is the peculiarity of an aërobic microbe?

Give an example of an aërobic microbe.

What is the peculiarity of the anaërobic microbe?

Name an organism of this class.

To which of these classes do the majority of microbes belong?

What term is applied to those aërobes in which a diminution of the amount of oxygen arrests their development?

What term is applied to those aërobes which can live with or without oxygen?

Are the anaërobes common among the pathogenic bacteria?

What is the effect of long exposure to oxygen upon certain pathogenic organisms?

Under what conditions will oxygen destroy the vitality of the anthrax bacilli?

What dyes are used to stain bacteria?

What is the object of using staining fluids?

How are tissues prepared for examination?

What is the value of double staining?

Give the formula for Ziehl's staining fluid.

What are the various functions of bacteria?

How is it customary to class them in relation to disease?

What are non-pathogenic bacteria?

What are pathogenic bacteria?

What is the function of the saprophytic germs?

How do pathogenic micro-organisms produce these effects?

Into what two classes are pathogenic micro-organisms divided?

What is the basis upon which further subdivision is made?

What is a micrococcus?

How are these organisms arranged?

When are they termed staphylococci?

When are they termed diplococci?

When are they termed streptococci?

What is the form of the bacillus of tetanus?

What is the form of the bacillus of cholera?

What is the form of the Bacterium lactis?

What is the form of the Leptothrix buccalis?

What is the difference between the streptococcus of pus and the streptococcus of erysipelas?

By what process do the cocci multiply?

Where are the cocci generally found?

What is a bacillus?

What forms of bacteria are not classed as bacilli?

What is a spirillum?

Give examples of this form of bacteria.

What are the characteristics of bacilli?

How do they multiply?

What is endogenous spore formation?

How long does it take for segmentation to take place in a coccus?

What structure protects the spores from deleterious external influences?

Describe the process of liberating the spore.

What becomes of the enveloping membrane during the development of the spore into a bacillus?

Which are more resistant to germicidal agents, spores or bacilli?

What is the temperature which cannot be resisted by a mature bacillus?

At what degree are most of them destroyed?

What degree will spores resist?

What sometimes becomes of spores which have entered the body and do not immediately germinate?

What two essentials are necessary for the germination of bacteria?

What forms of bacteria does the surgeon have to contend with most frequently?

How do they cause suppuration?

What are the causes of traumatic infectious diseases?

Name the conditions which Koch applied as a test before positive assertion could be made that an organism was the specific cause of a disease.

What conditions will govern infection with the pus-microbes?

How does quantity affect the result?

What dose is necessary to produce extensive abscesses?

What is the minimum dose to produce an effect?

How large a dose of the Staphylococcus pyogenes aureus is necessary to produce death in a rabbit?

How large a dose to produce a circumscribed abscess?

What is the size of the dose and the effect when the albus is injected?

What condition is necessary to produce the most marked results?

What is the effect of divided doses?

What is the effect when the toxic substances are introduced with the organisms?

What conditions favor the growth of microbes in a wound?

Are pathogenic organisms ever found in the blood of healthy persons?

Is their presence harmful, and if so, under what conditions?

What may become of spores which remain in a healthy body?

What part may the phagocytes play in the destruction of bacteria?

In what per cent. of cases did Steinhaus find the pus cocci in suppuration?

Describe the Staphylococcus pyogenes aureus.

Where is it most frequently found upon the outside of the body?

Where else is it found?

Describe the Staphylococcus pyogenes albus.

What are its effects upon the tissues as compared with the aureus?

Describe the Staphylococcus viridis flavescens.

How does it differ from the aureus and albus?

Describe the Staphylococcus pyogenes citreus.

How does this variety differ from the aureus and albus?

Describe the Staphylococcus cereus, albus et flavus.

Describe the Micrococcus pyogenes tenuis.

Describe the Streptococcus pyogenes.

How does it resemble the streptococcus of erysipelas?

Where is it found under normal conditions?

With what pathologic conditions is it sometimes found?

Describe the Bacillus pyocyaneus.

Is it motile or non-motile?

What term is applied to the coloring matter produced by this organism?

What effect has injection of cultures of this organism over anthrax?

Describe the Bacillus pyogenes fetidus.

Describe the Micrococcus tetragenus.

How is it distinguished from other micrococci?

How may pus be produced without the presence of pyogenic bacteria?

What is the effect of the injection into the tissues of the chemical substances elaborated by the pyogenic cocci?

Does acute suppuration occur without the presence of the pyogenic cocci?

How do the pyogenic cocci produce pus?

Are they found in cold abscesses?

How is the fact that they are rarely so found accounted for?

CHAPTER II.

SURGICAL BACTERIOLOGY (Continued).

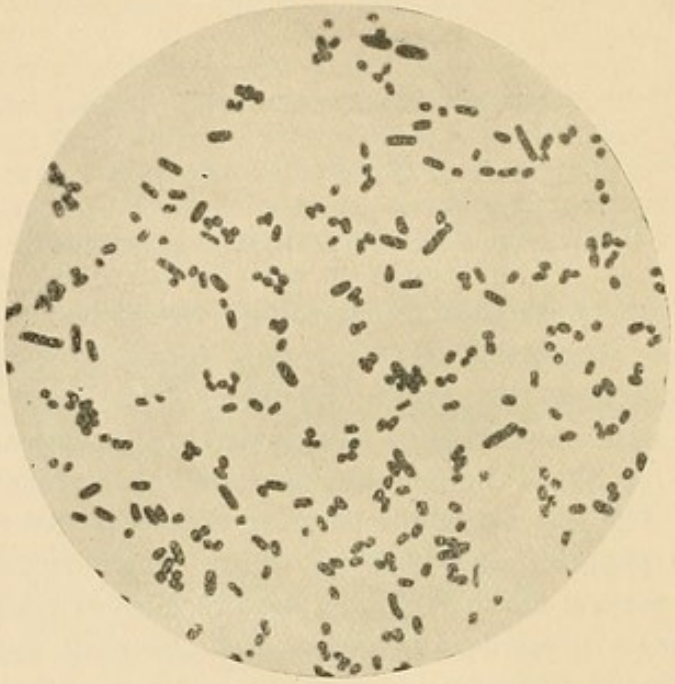
THE subject of Surgical Bacteriology would be very incomplete if the pyogenic organisms were the only forms considered in these pages. Prominent as they are in all surgical practice, they are nevertheless much more amenable to treatment by antiseptics, and far less dangerous to the life of the patient than some of the forms to be mentioned later. The interest of the surgeon is largely taken up with the prevention of suppuration following injury and surgical operations, but there are several diseases due to the presence of micro-organisms which sometimes present very grave and alarming symptoms, and which the surgeon must be prepared to combat. There can be no better preparation for such a battle than a knowledge of the character, number, and fighting qualities of the enemy, his base of supplies, the strong and weak points in his line of battle, and the number of his efficient reserves. The science of bacteriology furnishes this knowledge to the surgeon.

The *Bacillus coli communis* (Fig. 17) is constantly found in the discharges of healthy and unhealthy persons. It was first found in the discharges of cholera patients at Naples. It is also found outside the body in the air, in water, and in putrefying fluids. The importance of this organism has recently been augmented by its recognition as a cause of septic and suppurative processes in the peritoneal cavity.

This bacillus is usually seen as a short rod with rounded ends; it most frequently forms in pairs, but it may be combined in chains of from four to six filaments. Occasionally both of these forms are associated together, which may lead to a mistake, as the appearance is that of a mixed culture. The organism possesses numerous and peculiar cilia. Spores have not been demonstrated. It stains readily with the anilin dyes, but is decolorized with iodin. It grows freely upon acid or alkaline media, with or without the presence of oxygen; the products of its growth are acid, and it does not liquefy gelatin. On gelatin plates it has two forms of growth: One is an irregular film, rapidly spreading over the plate, and having an opalescent color, while the other is an ivory-white, heaped-up colony, which shows no tendency to spread. Under favoring circumstances other bacteria like

the typhoid bacillus and the pneumococcus may assume pyogenic qualities.

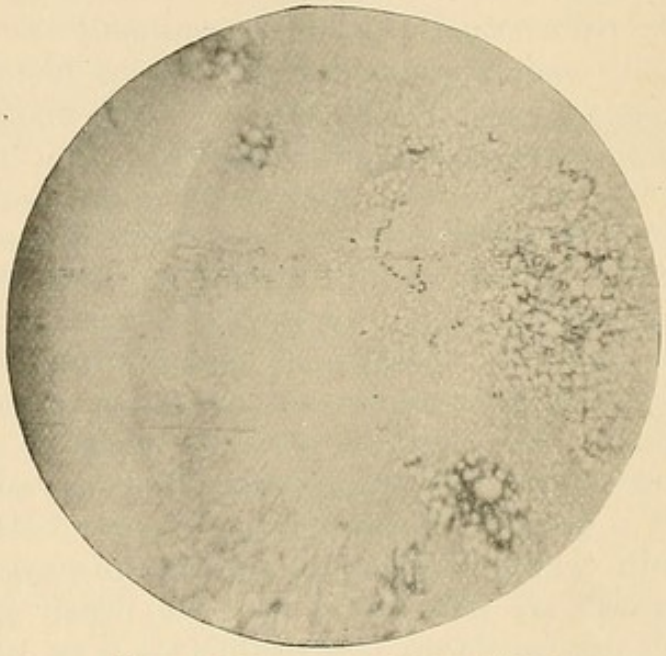
FIG. 17.



BACILLUS COLI COMMUNIS. × 1200.

The *Streptococcus erysipelatus* (Fig. 18) so closely resembles the Streptococcus pyogenes that it is almost impossible to distinguish be-

FIG. 18.



STREPTOCOCCUS ERYSIPELATUS. × 1200.

tween them, the only physical difference discoverable being one of size, the erysipelatus coccus being the larger. (Rosenbach.) The consensus

of opinion, however, is in favor of their identity; for the reason that the majority of bacteriologists are unable to detect any differences which are constant between them.

The description of this organism is the same as for the Streptococcus pyogenes. It is without doubt the cause of erysipelas, and perhaps of puerperal fever, for there is a very close relationship existing between these conditions, as many medical men have learned by sad experience. Experimental research has discovered that cultivations of the streptococcus from puerperal cases injected into rabbits produced erysipelas.

FIG. 19.



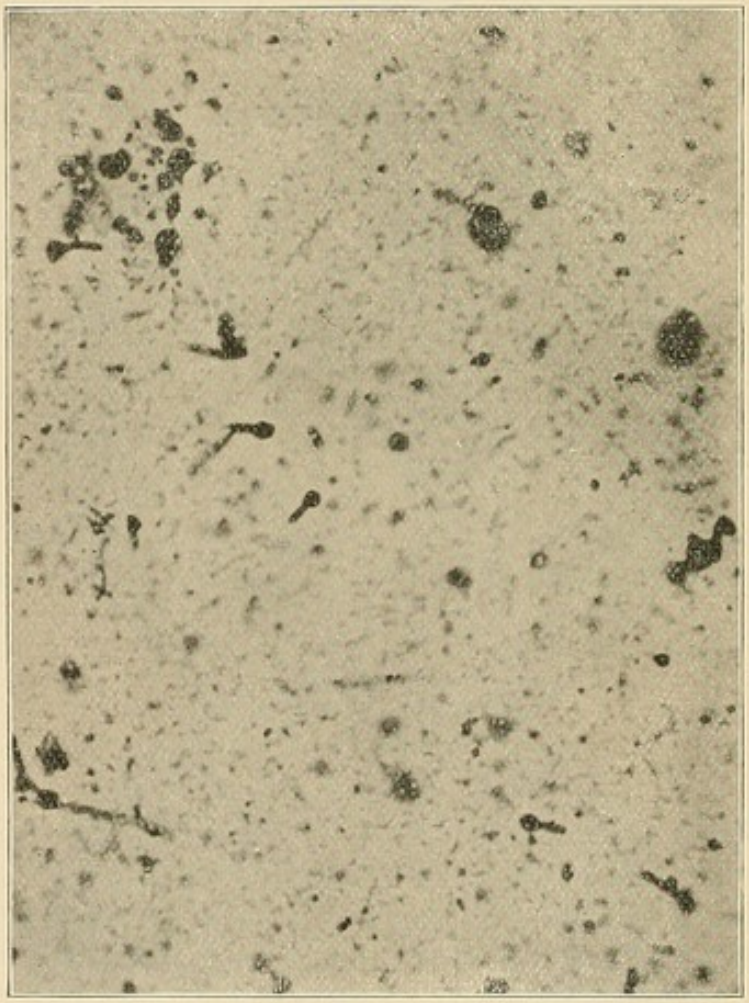
GONOCOCCUS. × 1200.

The infection of old ulcers with the virus of erysipelas has often proved curative, and cases are on record in which tumors have disappeared during an attack of erysipelas. Cultures of the organism have been used frequently of late in the treatment of inoperable neoplasms, particularly sarcomas, with varying success.

The *Gonococcus* (Fig. 19) is the specific micro-organism which produces gonorrhea. It is found in gonorrheal pus, generally in the form of a diplococcus, and measures 1.25 micro-millimeters in diameter. It is exceedingly difficult to cultivate, only growing upon blood-serum at a temperature of 33° to 37° C., and not in company with other organisms. One of its marked peculiarities is the power which it possesses to enter other cells, and grow and multiply within them. This peculiarity differentiates it from nearly all other forms of micrococci. It is rare to find a gonococcus outside of a pus-cell. Sometimes the cell becomes so filled with them as to lose all of its characteristics and assume the appearance of a cluster of diplococci. The

gonococcus stains well with methyl blue, but not with the Gram method. Neisser recommends double staining with eosin and methyl blue. Its growth upon blood-serum produces a thin, varnish-like film with irregular but sharply defined edges; later it becomes grayish white, and afterward a slightly brownish hue. The gonococcus is only found associated with certain mucous membranes,—those which possess a cylinder epithelium, or one closely allied to it, like the mem-

FIG. 20.



Bacillus.

TETANUS (Shows Smear of Culture-Medium) × 1200.

branes of the male and female urethra, vagina, uterus, and conjunctiva. Why it should grow in these particular membranes and only in their superficial layers, and nowhere else in the body, is still unexplained.

The *Bacillus tetani* (Fig. 20) is a large slender rod with rounded ends,—one end being enlarged, giving it the appearance of a round-headed pin or a drum-stick. It is motile, and belongs to the anaërobic class of micro-organisms. Exposure to the air quickly destroys its vitality. It sometimes grows in long chains, the divisions being imperfectly seen. The enlargement at the end is due to the formation of

a spore. The spore germinates in thirty hours if kept at a temperature of 37.5° C., while if kept at the house temperature it requires about one week. It is readily colored by methyl blue and fuchsin, the Gram method being especially adapted to bring it out to perfection. It can be grown in cultures of gelatin mixed with grape sugar; the latter aids its rapid development. Being a strictly anaërobic microbe, it does not grow when exposed to the atmosphere, and this accounts for the fact that the surface of the gelatin inoculated with the bacillus remains sterile; while at the bottom of the culture there is active growth, sending out innumerable slender prolongations, and producing in the gelatin the appearance of an inverted fir tree. At the end of a week the

FIG. 21.



BACILLUS TUBERCULOSIS IN SPUTUM. × 1200.

gelatin begins to liquefy, and soon the whole mass is changed into a light gray, tenacious, shining substance.

The spores have been found in garden soil, in masonry, in the dust of the streets, in decomposing liquids, and in stable manure. Hence the frequency of tetanus among gardeners and stable men. Tetanus is thought to be produced by the elaboration of certain toxic substances from the bacillus which are taken up by the circulation, and cause irritation of the nerve-centers. Brieger obtained three toxic substances from cultures of the bacillus, which he named "tetanin," "tetanotoxin," and "spasmotoxin." All of these, when injected into animals, caused spasms or convulsive movements, and finally paralysis.

The *Bacillus tuberculosis* (Fig. 21) is a small, slender rod, varying in length from one-fourth to three-fourths the diameter of a red blood-corpuscle. It has rounded ends, and is somewhat bent or curved near

the center; is usually single, sometimes arranged in pairs, and often in the form of a letter V, or strung together. The longest rods are usually seen in the sputa of phthisical patients. This bacillus does not possess motile power. It is thought by Baumgarten that it multiplies by endogenous spore formation, from the fact that the cheesy material of a tubercle in which no bacilli can be discovered by any method of staining, if injected into animals, produces the disease. The bacilli have never been seen in the process of spore formation, and free spores have never been discovered. These bacilli are very tenacious of life; sputum charged with them may be kept for months and even years in a dried state without endangering their vitality. They also possess great resistive power to the acids of the stomach and to the products of decomposition; they may even pass through the entire alimentary tract without in any way impairing their vitality. This resistance is due to the unusually tough enveloping membrane or cell-wall possessed by the bacillus. It requires the boiling temperature for twenty minutes to destroy the organisms in tuberculous sputa. The tubercle bacillus is a facultative anaërobic microbe,—grows with or without oxygen, but flourishes best in the atmosphere. It is among the most difficult of the bacteria to stain with the anilin dyes, and like the bacillus of leprosy, which it greatly resembles, does not readily yield to the action of bleaching agents. Nearly all other bacterial forms are readily decolorized. The bacilli are found in the tubercles, between the leucocytes in the epithelioid cells, and in the giant cells.

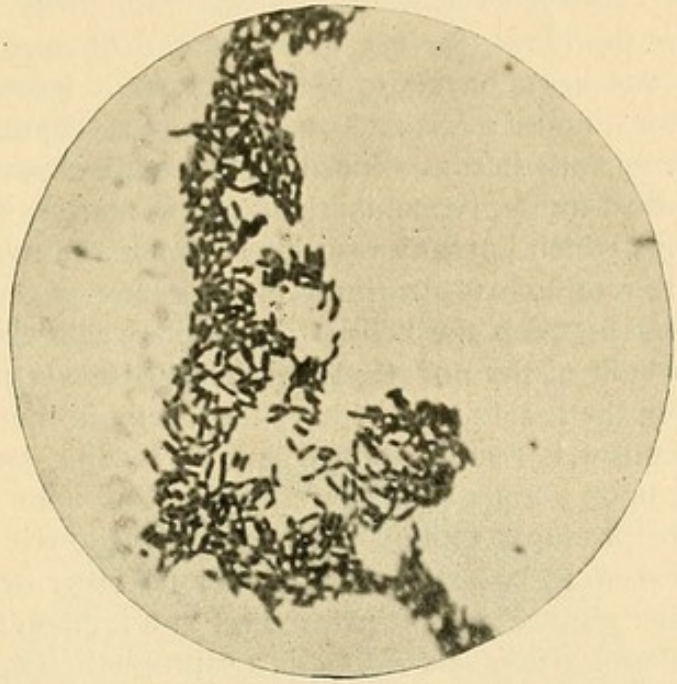
This organism is exceedingly difficult to cultivate, though it grows upon the hardened blood-serum of Koch, or upon a combination of agar and glycerol, but it requires from twenty-one to twenty-eight days when hardened blood-serum is used, which indicates a predilection of the bacillus for glycerol. At the end of this time the culture appears as thick, dull, grayish-white scales, very dry and brittle. Material for cultures is obtained by inoculating guinea-pigs with tuberculous sputa. When the disease is established the animal is killed, and fragments of tubercle from the lungs are placed upon the culture-medium.

The tubercle bacilli are never found growing outside of the living tissues of man and animals, as the proper conditions of nutrition and temperature can nowhere else be found; they are therefore true animal parasites. Inoculation may occur through abrasions of the skin, through the mucous membrane of the respiratory tract or digestive system. Ernst has shown that six drops of milk from a tuberculous cow injected under the skin of a guinea-pig may develop tuberculosis.

The *Bacillus mallei*, Figs. 22, 23 (bacillus of glanders), is a short, straight rod; in length about two-thirds the diameter of a red blood-corpuscle,—that is, somewhat shorter and a trifle thicker than the

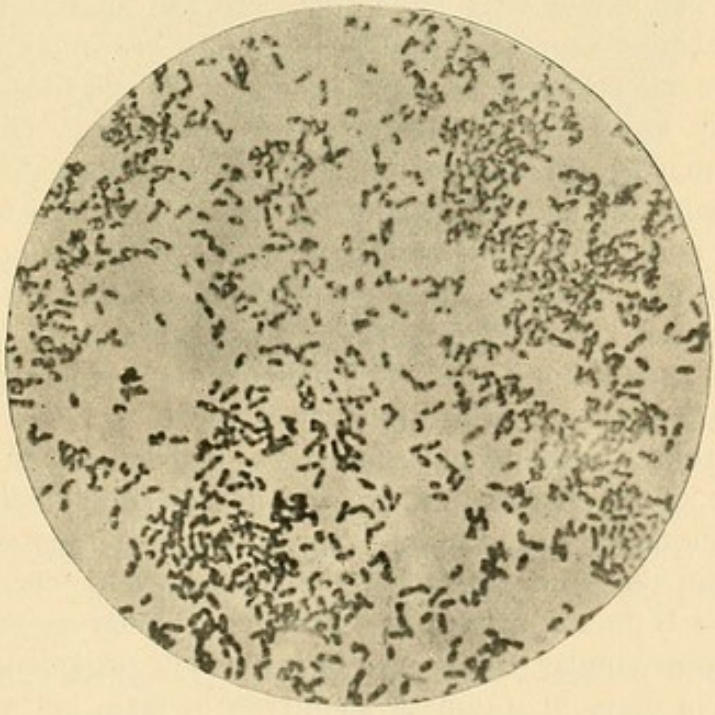
tubercle bacillus. They are generally found single, but are sometimes arranged in couples, side by side. In the tissues they are grouped in

FIG. 22.



BACILLUS MALLEI (GLANDERS). × 1200.

FIG. 23.



BACILLUS MALLEI (GLANDERS). × 1000.

clusters, either parallel with one another or at various angles. In culture-media several of them may be joined together in chains. They are non-motile, and belong to the facultative anaërobic organ-

isms. This bacillus was discovered by Löffler and Schütz, who succeeded in demonstrating its presence in the tissues, cultivated it outside the living organism, and produced the disease by inoculation of animals with the culture. The animals in which the virus can be readily inoculated are the horse, the ass, goats, cats, field mice, and guinea-pigs. Lions and tigers have also been successfully inoculated with it. Pigs, white mice, house mice, and oxen are not susceptible to the disease. Man frequently becomes inoculated through abrasions upon the hands, etc. Post-mortem examinations reveal nodules in the spleen, liver, and lungs which in many respects resemble the tubercle nodule. The bacilli are found most plentifully in the center of the nodule, and generally lying between the cells. Epithelioid cells and leucocytes make up the bulk of the nodule; giant cells are never present. The secretions from the nasal passages have few bacilli in them.

The organism is readily stained and as readily decolorized. It grows readily upon a 4 per cent. glycerol-agar, and upon potato. The potato culture produces around the border a yellowish-green color, which would seem to be pathognomonic, as no other organism gives this color under cultivation. It grows best at a temperature of 37° C.

The *Bacillus of Malignant Edema* is a saprophytic organism. It is a slender rod, narrower than the anthrax bacillus, for which it is sometimes mistaken; frequently arranged in bands, which are often bent or curved. They are strictly anaërobic, and are endowed with active motility. Motion soon ceases on their coming in contact with oxygen, as they are exceedingly sensitive to even the slightest trace of it. Spores are formed in a temperature above 20° C.; these are large, and may be situated at the center or the end of the rod. They stain well with the anilin dyes, but not by the Gram method. When stained the ends of the rods appear pointed, which distinguishes them from the anthrax bacillus. They grow best in gelatin cultures to which from 1 to 2 per cent. of grape-sugar has been added. They liquefy gelatin, and usually form gas which distends the needle tract and gives off an offensive odor peculiar to putrefaction.

The bacillus is found in decomposing substances, in dirty water, in the dust of the streets, and in rich garden-mold; being found in greatest abundance in the latter. Injections of garden soil are more virulent than the pure culture of the bacillus, and when introduced subcutaneously in the guinea-pig, produce a progressive emphysematous gangrene similar to that seen in man. The organism is occasionally found in cases of traumatic gangrene in man, but always in the superficial tissues and never in the blood-vessels, in marked contrast to the habit of the anthrax bacillus. According to Chauveau, animals which have recovered from an attack of malignant edema are rendered immune to the disease ever afterward.

The *"pseudo-edema"* bacillus is distinguished from the bacillus of malignant edema by its thicker form, the possession of a very bright border, and by the formation of two spores in each rod. It is strictly an aërobic organism, and not so infectious as the true bacillus. In cultures it is accompanied by an abundant gas-formation which has the odor of old cheese. It is sometimes found in traumatic gangrene of man. Noma, a malignant gangrenous inflammation of the mouth and genitals in young children, is probably caused in many cases by the bacteria of this class, which with the pyogenic cocci are always present and ready to attack wounds located in uncleanly parts of the body, and in the tissues whose vitality has been impaired or destroyed by injury.

The *Bacillus of Leprosy.* This bacillus in appearance is almost identical with the tubercle bacillus and the glanders bacillus. It is a

FIG. 24.



BACILLUS OF LEPROSY. × 1000.

long, slender rod, with sharpened ends, is non-motile, and is the only organism which reacts to the coloring reagents in the same manner as the tubercle bacilli. (Fig. 24.) The readiness with which it takes the anilin dyes, and also stains by the Gram method, distinguishes it from the tubercle bacilli.

Bacteriologists have not as yet settled the question as to its being the actual cause of leprosy. The bacilli of leprosy are usually found in the skin and the tissues immediately surrounding the nerves; in the lymphatic glands, the spleen and liver, but rarely in the blood. They grow in clusters, usually inside of the cells—leucocytes and epithelioid cells, which in consequence have been called *lepra cells.*

The *Bacillus of Syphilis.* The microbic origin of syphilis has attracted the attention of bacteriologists generally, but so far the specific organism of the affection has not been conceded. The clinical features of the disease make its microbic origin almost certain, and that the organism is a bacillus, but it has not been satisfactorily demonstrated. Lustgarten announced in 1884 the discovery of an "S" shaped bacillus in the tissues and discharges of syphilitic ulcers which closely resembled the tubercle bacillus, but he was unable to successfully cultivate it. It was distinguishable from other forms by the peculiar methods necessary to stain it. Gelatin made from the bladders of Russian sturgeon is said to be a medium upon which it may be successfully cultivated. Doubt has been thrown upon Lustgarten's discovery through the simultaneous finding by two different observers of a similar bacillus in the preputial and vulvar smegma. Eve and Lingard cultivated a bacillus which they found in the blood and tissues of syphilitic patients, and describe it as resembling the tubercle bacillus. It was readily stained by the ordinary anilin dyes and by the Gram method, but would not take the stain by Lustgarten's method. Pure cultures were obtained, but inoculation of monkeys with these gave negative results. Disse and Taguchi found an almost constant "coccus" in patients suffering from secondary syphilis. The organism was found isolated or in groups between the corpuscles. Inoculation with pure cultures, in rabbits, dogs, and sheep, produced a chronic infectious disease, which was transmitted to the offspring.

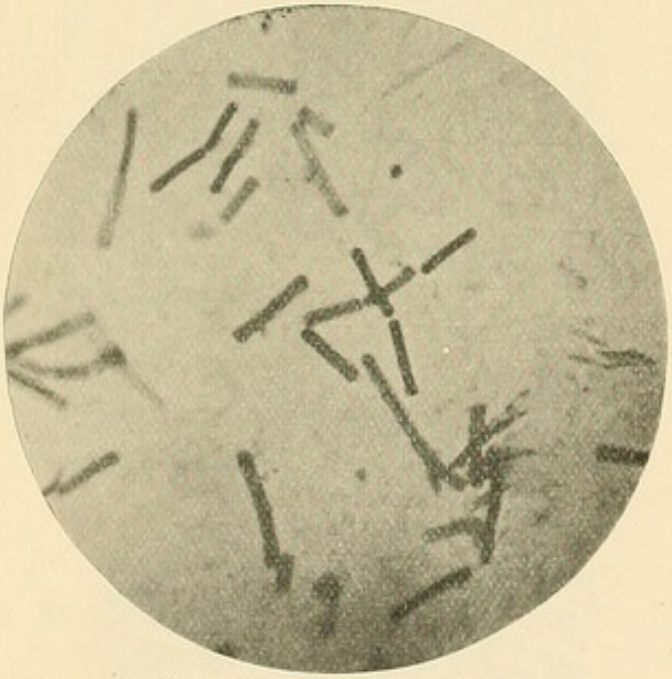
Martineau and Hammic succeeded in producing eruptions in monkeys resembling those of syphilis, and nodules which simulated indurated chancre, by inoculating these animals with a culture bouillon in which fragments of chancres had been placed, and in which the growth of bacilli had been demonstrated. Secondary symptoms were also developed. Klebs successfully inoculated monkeys in the same manner. Mucous patches were developed upon the buccal mucous membrane, and caseous deposits were found in the dura mater which resembled gummata. Implantation of a fragment of chancre under the skin resulted in caseous deposits, resembling the deposits of tubercle. The evidence seems therefore to favor Lustgarten's bacillus as the specific cause of the disease.

The *Bacillus Anthracis* is the organism which produces anthrax, or splenic fever in animals. It is classed among the surgical bacteria, because inoculation with the virus in man produces malignant pustule, and this frequently happens to those who have to deal with animals suffering from the malady, or who handle the hides or wool of such animals; hence the name "wool-sorters' disease." It also deserves to be so classed on account of its position historically, for it was the first of the bacterial forms discovered in the blood and tissues, and the first to

be demonstrated, experimentally, as the specific cause of a disease. Upon the investigations which proved this microbe to be the real and only cause of splenic fever, the whole science of bacteriology has since been reared.

The bacillus of anthrax (Fig. 25) is a large rod, from three to six micro-millimeters in length, and 1.5 micro-millimeters in thickness. When grown upon culture-media they appear as bright, transparent rods, with slightly rounded ends, and are entirely devoid of motion. The bacilli taken from the blood of animals which have recently died of anthrax appear somewhat larger at each end than in the center, and are joined together in the form of a chain, simulating the articulation of the phalangeal bones. This form of enlarged ends and articulation of the

FIG. 25.



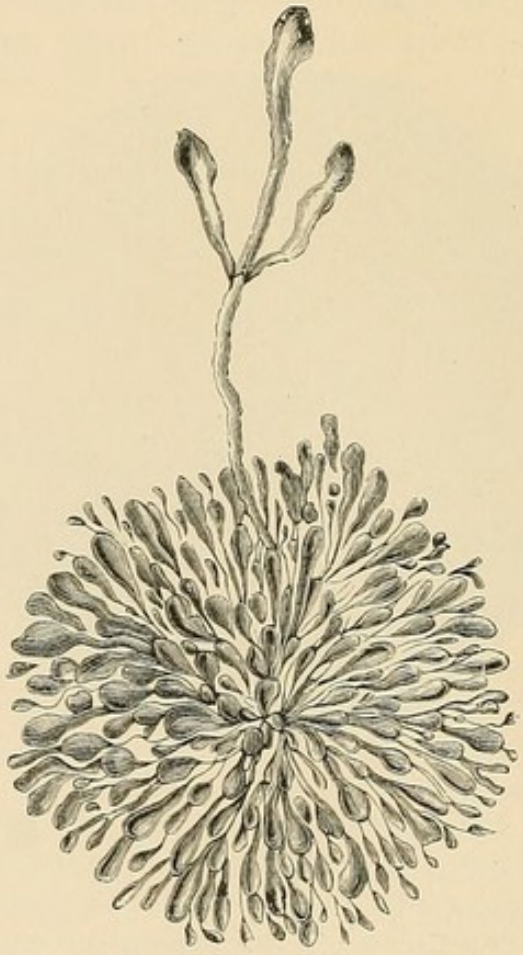
BACILLUS OF ANTHRAX. × 1200.

rods is peculiar to the anthrax bacillus, and differentiates it from all other forms of bacteria. The spores when forming are seen as bright, glistening spots in the center of the bacilli. The developed organisms are rather delicate, but the spores are among the most resistant to external influences, and are therefore commonly used as a test of the value of germicides. The bacilli can be cultivated in bouillon, agar, gelatin, or potato, and in human urine freely exposed to the air. Oxygen is necessary to their growth, and they grow best at a temperature of 37° C. They will not grow at a lower temperature than 16° C., or at a higher temperature than 45° C.; the spores will not germinate without a large supply of oxygen or below a temperature of 24° C. Pasteur succeeded in producing an attenuated virus by long cultivation or cultivation carried on at high temperature, which

by repeated injections rendered animals immune to the disease for a certain time—about a year—through all avenues of infection except the intestinal canal. The spores are taken into the stomach with food that has been contaminated by other diseased cattle, from nasal discharges, urine, and feces.

Oxen and sheep are particularly susceptible to infection through the alimentary mucous membrane, and as this is the most common avenue of infection in cattle, further experiment will be necessary to determine the possibility of devising a practical method of protection from the disease by "vaccination."

FIG. 26.



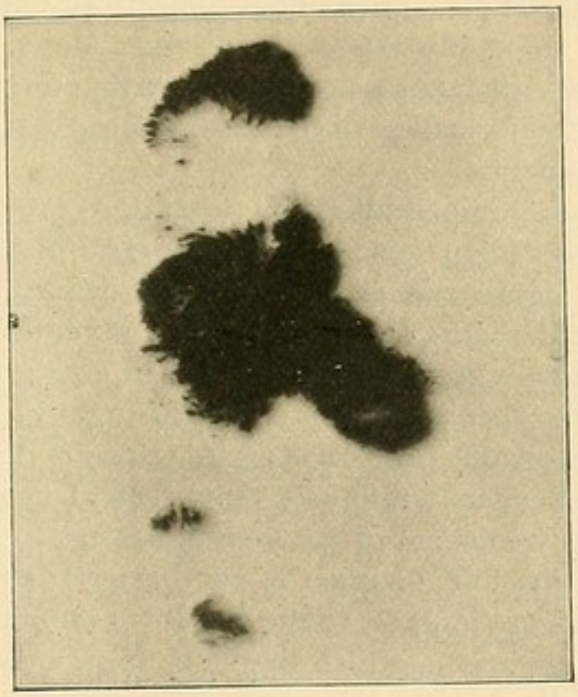
THE RAY-FUNGUS. (ACTINOMYCES.) (After Ponfick.)

Actinomyces is a ray-bacterium or a ray-fungus, which produces a disease in cattle, pigs, etc., known as Actinomycosis, or *lumpy-jaw* (Fig. 26). This bacterium is classed with the surgical bacteria because the disease occasionally occurs in man as a result of infection. The organism occurs in the form of spirally curved branching threads, radiating from a common center, and terminating in bulbed extremities. These prolongations are so arranged as to look something like a sunflower. The bacterium macroscopically appears about the size of

a millet-seed, yellowish in color, and of a tallowy consistence. It can be cultivated upon blood-serum, agar, or gelatin, and the temperatures in which its growth is most active are from 33° to 37° C. Development is completed in from five to six days. (Fig. 27.)

Actinomycosis in man is an exceedingly rare affection, but one that can be diagnosed with great readiness on account of the peculiar character of the micro-organism.

FIG. 27.



ACTINOMYCOSIS (RAY-FUNGUS) OF GLANDS. × 500.

Action of Bacteria.—The question of *how bacteria act upon the living tissues of the body* is not yet fully decided. Some are of the opinion that the symptoms of infectious disease are the result of the formation by the bacteria of chemical substances of an irritating or poisonous nature, a sort of specific excreta. Others suppose the phenomena, both local and constitutional, to be due to changes brought about in the tissues by the organisms themselves, during their development, and that it is not necessary to assume the formation of a specific poison or virus.

The action of the pyogenic bacteria locally is to produce irritation or inflammation; while the chemical substances elaborated are disseminated throughout the body, which by virtue of a peculiar action—thought to be ferment-like—augments tissue-metamorphosis, stimulates the "thermic centers," and thereby increases the body temperature, producing fever or systemic disturbance. This condition is known as *septic infection*.

The absorption of ptomaines without the presence of bacteria will produce grave systemic disturbances. This condition would be termed *septic intoxication,* or *toxic infection.*

Ptomaines, which are powerful animal poisons, are developed by the process of decomposition of animal tissue in the presence of saprophytic bacteria. In their physiological action they resemble the alkaloids, and when received into the circulation by the process of absorption, they produce more or less severe constitutional symptoms. The "toxines" probably belong to this class of substances. The development of these substances—ptomaines—appears to exert a controlling inhibitory effect upon the micro-organisms. Many of the artificial cultures of the bacteria, after a period of growth, cease to develop, and it is by virtue of the formation of these substances that this controlling effect is brought about.

Leucomaines are animal alkaloids, which are produced in the tissues by metabolism (tissue changes), independent of micro-organisms. Their pathologic significance is as yet not well defined.

The effect of the virus of certain bacteria upon the vital fluids and the tissues of the body in certain diseases is to give protection against future attacks,—in other words, to render the organism *immune.*

Pasteur thought this protection or immunity to be due to the exhaustion of the chemical substances (supposedly) necessary to maintain the life and development of the specific bacteria. Fraenkel was of the opinion that the first invasion of the bacteria left behind certain substances which were inimical to the further development of the same species of micro-organism, which might at some other time gain an entrance to the system.

The direct transmission of bacterial diseases from parents to fetus is a question which has not yet been satisfactorily proved, and is hardly susceptible of a ready demonstration, though the best authorities have admitted for a long time, from clinical proofs, that many infectious surgical diseases are hereditary. The avenues through which hereditary diseases may be communicated are the semen, and the placenta during intra-uterine life. It is well known that syphilis may be transmitted both through the semen and the placenta; although the bacteriologic proofs are lacking as to the existence of a specific syphilis bacillus. Infection through the placenta has been frequently observed in animals; "glanders has been transmitted from mare to foal, and the bacilli of anthrax, glanders, and malignant edema have been shown by experiment to pass through the placenta to the fetus." (American Text-Book of Surgery.)

The best of clinical evidence exists that diseases like smallpox, typhoid fever, intermittent fever, erysipelas, measles, and scarlatina are directly transmissible from mother to fetus. Several well-authenticated

cases are on record in which these diseases occurred in new-born children, and the lack of an incubation period for the disease can only be explained upon the hypothesis of pre-natal infection.

The tubercle bacillus has been found in the testicles and in the prostate gland, and "it requires no stretch of the imagination to understand how the spermatozoön in the testicle or on its way to the vesiculæ seminalis can be contaminated with bacilli and the disease thus transmitted from father to fetus." (Senn.)

The Fallopian tubes are often the seat of tuberculous disease, which makes it more than probable that the ovum in its passage to the uterus may become infected with the bacillus.

The General Principles of Antiseptic Treatment.—Inasmuch as all suppurative processes are the result of the action of micro-organisms, which enter the system through some break in the continuity of the surface, and cause putrefaction of the tissues and exudates, the first duty of the surgeon or dentist is to prevent putrefaction, and if it has already been established, to arrest its further progress.

This may be accomplished: First, *By excluding all organisms from the wound*, by strict attention to the details of surgical cleanliness; Second, *By removing the organisms which may already have gained an entrance to the wound*, by thorough irrigation before they can produce their harmful effects; Third, *By destroying the organisms which may remain*, with solutions of bichlorid of mercury, or other germicides; Fourth, *By removing dead and dying tissue, and establishing free drainage*, for the escape of the discharges. Disorganized tissue is the soil in which micro-organisms best grow and flourish; Fifth, *By preventing the formation of a favorable soil in which they can grow.* This can best be accomplished by avoiding unnecessary manipulation of the wound, guarding against tension from stitches or bandages, and by careful, dry antiseptic dressings.

Dry dressings are not applicable in wounds within the mouth, vagina, or anus. In these cases dependence must be placed upon free irrigation, with antiseptic solutions.

A wound is called aseptic when it is free from pathogenic or septic micro-organisms, and septic when it is the seat of infection.

The term Antiseptic means germ-destroying. Asepsis can only be secured by antisepsis. An aseptic wound is therefore one which is free from germs, or has been rendered germ free by antiseptic treatment.

An antiseptic dressing is one which has been rendered sterile, and contains germ-destroying substances.

An aseptic dressing is one which has been made germ free by sterilizing with heat.

The common antiseptic solutions are carbolic acid, 3 to 5 per cent.;

bichlorid of mercury, 1 part to 5000, or 1 to 2000, 1 to 1000, 1 to 500 of water; a saturated solution of boric acid; Thiersch's solution 12 parts boric acid, four parts salicylic acid to 1000 of water. Listerine and borolyptol are also valuable antiseptics, especially for use in the mouth, but are too expensive for common use.

Pulverized boric acid and iodoform are the remedies generally used in dry dressing of wounds. On account of the disagreeable odor of iodoform, boric acid has the preference with many surgeons. Various other antiseptics might be mentioned, but these are the ones in most common use.

REVIEW.

CHAPTER II.

Describe the Bacillus coli communis.
Where is it commonly found?
What is the importance of this organism?
Describe the Streptococcus erysipelatus.
Is there any doubt as to the specific action of the organism?
Has the organism any curative value in other diseases?
Describe the Gonococcus.
What is a marked characteristic of this organism?
Where is the organism found?
Why does it select this form of mucous membrane?
Describe the Bacillus tetani.
How does oxygen affect it?
Where are the spores found?
What class of individuals are most likely to be affected by the organism?
How is tetanus produced?
Describe the Bacillus tuberculosis.
How does it multiply?
How long may it live in the dried state?
What other resistive powers does it possess?
What temperature will it resist, and for how long?
Where are the bacilli found in the tissues?
Are they ever found growing outside the tissues?
Through what avenues do the organisms gain access to the tissues?
Describe the Bacillus mallei.
What animals are susceptible to glanders by inoculation?
What animals are not susceptible to the disease by inoculation?
How may man become inoculated?
How do the nodules found in glanders resemble those of tuberculosis?
Where are the bacilli of glanders found?
Under what conditions does infection occur in man?
Describe the bacillus of malignant edema.

How is it distinguished from the anthrax bacillus?
Where is the bacillus of malignant edema found?
Under what conditions is it found in man?
Does one attack give immunity?
How may the "pseudo-edema" bacillus be distinguished from the real organism?
Under what circumstances is it found in man?
What is noma?
What is the probable cause?
Describe the bacillus of leprosy.
What characteristic distinguishes it from the tubercle bacillus?
Is the bacillus the actual cause of the disease?
Where is it usually found?
Has the bacillus of syphilis been found?
What would the clinical features of the disease indicate?
Who discovered the "S" shaped bacillus, and in what year?
What has thrown doubt upon Lustgarten's discovery?
What well-known form does the S-shaped bacillus resemble?
What result followed inoculation of monkeys with cultures made from pieces of chancre in which the bacilli were demonstrated to be present?
What would these results seem to indicate?
What is the Bacillus anthracis?
Why is it classed as a surgical bacterium?
What is its historic position?
Describe the bacillus of anthrax.
How did Pasteur render animals immune to the disease?
How long did this immunity last?
Through what avenue of infection did it not protect the animal?
What avenue of infection is most common in cattle?
What is Actinomycosis?
What common term is applied to this disease?
Why is this bacterium classed with the surgical bacteria?
Describe the organism.
How does it appear macroscopically?
Is there any difficulty in its diagnosis?
What are the opinions as to how bacteria act upon the bony tissues?
How do the pyogenic bacteria produce their effects?
What form of infection is this termed?
What is the condition produced by the absorption of ptomaines, without the presence of bacteria?
What are ptomaines?
How are they elaborated?
What do they resemble in their physiological action?
How are the "toxines" classed?
What effect do the ptomaines have upon the bacteria?
What are Leucomaines?
How are they elaborated?
What is their pathologic significance?
What is the effect of the virus of certain bacteria upon the fluids and tissues of the body?
How did Pasteur explain this?
What was Fraenkel's opinion?

What are the opinions as to the hereditary transmission of infectious surgical diseases?

Through what avenues does infection take place?

Are there any bacteriologic proofs that syphilis is so transmitted?

Are there clinical proofs that such diseases are so transmitted?

What evidence is there that other infectious diseases are so transmitted?

What are the causes of all suppurative processes?

How may putrefaction be prevented? First?—Second?—Third?—Fourth?—Fifth?

When is a wound called aseptic?

When is it called septic?

What does the term antiseptic mean?

By what means can asepsis be secured?

What is an antiseptic dressing?

What is an aseptic dressing?

Name the common antiseptic solutions.

What is the formula of the Thiersch solution?

Why is boric acid preferred to iodoform?

CHAPTER III.

INFLAMMATION.

A KNOWLEDGE of surgical pathology presupposes an understanding of the process of inflammation; in fact, a correct appreciation of the various phenomena of inflammation is absolutely essential as a foundation upon which to build a correct knowledge of surgical pathology. It is therefore of the utmost importance that the student make himself thoroughly familiar with the entire subject of inflammation, for upon this knowledge, and the ability to apply it, will depend in very large measure the success or failure of the practitioner in any department of surgery.

To Cohnheim belongs the honor of first placing before the world a scientific explanation of the phenomena of inflammation, by the publication in 1867 of the results of his study and experiments upon the circulation of the blood and the action of the white blood-corpuscles, as observed in inflammation. These labors added very greatly to the then existing knowledge of the inflammatory process in the tissues, and have since formed the basis for all further research in this direction. Cohnheim's views, however, have not been adopted in their entirety, as various modifications have been suggested by other investigators, but in the main they form the prevailing theory of to-day.

Inflammation.—Synonym, Lat. *inflammatio* (from *inflammare*, to inflame.—*Inflammo*, I set on fire).

Definition.—Inflammation is a condition of nutritive disturbance, characterized by hyperemia, with proliferation of the cells of a tissue or organ, and attended by one or more of the symptoms of pain, heat, swelling, discoloration, and disordered function.

Inflammation is a series of changes in a part, identical with those which are produced in the same part by injury from a chemical or physical irritant.

Physical Signs.—The physical signs or cardinal symptoms of inflammation, as described by Celsus, are four in number, viz: Rubor, Tumor, Calor, and Dolor,—redness, swelling, heat, pain; a fifth symptom has since been added, viz: *Functio Læsa*, disturbance of function in the part.

All forms of inflammation are the result of the action of certain

irritants. Irritants are classed under the four following heads: mechanical, chemical, septic, and nervous.

A *mechanical irritant* produces irritation through its mechanical action. Examples: Foreign bodies, scarification, puncture, acupressure, etc.

A *chemical irritant* is a substance which irritates by virtue of its chemical reaction upon the tissue elements to which it is applied. Examples: Alkalies, acids, croton oil, cantharides. Other examples are found in drugs which have a predilection for certain organs, and which, if administered in poisonous doses, will cause inflammation. Mercury acting upon the tissues of the mouth and salivary glands, produces stomatitis, and salivation. Cantharides will cause irritation of the urinary organs, ergot of the uterus, and uric acid will cause gouty inflammations. The toxic inflammation caused by the poison of serpents and insects, the poisonous action of certain plants like ivy, and the ptomaines, are examples of other groups of chemical irritants.

A *septic irritant* is a living organism,—a parasite, a micro-organism,—causing irritation by its presence, and the formation of poisons, —ptomaines,—as waste products, and their introduction into the system. The pus-microbes and the saprophytic germs belong to this class.

A *nervous irritant* is one which produces irritation through the medium of the nervous system. The influence exerted by the nervous system over the functions of nutrition, both generally and locally, and in the production of inflammatory symptoms, has long been recognized. Impairment of the nutrition of the skin is sometimes observed to follow injury of the nerves supplying the part. Reflex conditions are also recognized as productive of inflammation. An instance in point is irritation of the dental pulp, which sometimes occurs in pregnant women. Herpes zoster is an example of a pustular eruption following the course of a nerve, and is accompanied with infiltration of leucocytes, both around the terminal branches and the trunk of the nerve. (Warren.)

Irritation.—Definition: To excite, to stir up, to inflame. Irritation is the state of a tissue or an organ in which there is an excess of vital movement, commonly manifested by increase of the circulation and the sensibility. Irritation in some form always precedes inflammation, or, in other words, inflammation is always caused by some form of irritation. When the irritation is confined to a particular portion of the body, it is termed *local irritation.* Examples would be, an injury upon the surface of the body; infection from a devitalized tooth-pulp, etc. When the irritation affects the whole system, it is termed *general* or *constitutional irritation.* Examples would be the presence of malarial poison in the blood, or pyemia.

Inflammation is a process which may affect any tissue of the body having a vascular circulation, or which is connected with blood-vessels. It begins usually with the phenomena of hyperemia, and progresses to exudation or suppuration, sometimes healing, sometimes leading to the production of new formations, or to metamorphoses of various kinds, or to death and destruction of tissue, and creating a more or less serious disturbance of the functions of the parts. The disease may vary very greatly in its character and in its location. The histologic character of inflammations depends upon two factors: the nature of the exudation and the changes in the tissues. Both are used to classify the various forms of inflammation, according as the one or the other seems to be the most pronounced.

Hyperemia.—The condition known as hyperemia is one of the most elementary physical disturbances in the realm of surgical pathology, and occupies a position, both physiologic and pathologic, that is difficult to define. A hyperemia may be a physiologic expression of an emotion, or a local stimulation, causing an accelerated movement of the blood in a given part, or it may be the beginning of a pathologic condition induced by slight injury, or the entrance into the circulation of certain noxæ; but just at what point the process ceases to be physiologic and becomes a pathologic expression cannot be demonstrated. Fig. 28 shows capillary blood-vessels in a normal condition, with the blood-corpuscles passing through them.

Definition: Hyperemia is a condition in which there is an increased amount of blood in a part. When there is an increased amount of blood in all the vessels of the body, it is termed *plethora*. The opposite conditions to these are, *ischemia*, a decreased flow of blood to a part, and *anemia*, when there is a less quantity of blood in the body than is usual. In a medical sense, the latter term is used to indicate certain pathologic conditions in the character of the blood.

Hyperemia may be a physiologic or a pathologic condition. Flushing of the cheeks, as the result of mental excitement produced by joy, shame, or anger, is an illustration of the physiologic hyperemia; while the redness following a local irritation would more nearly express a pathologic hyperemia.

Hyperemia may be divided into two forms, generally denominated *active* and *passive;* the difference between these forms being that in active hyperemia there is an increase in the amount of arterial blood flowing into the part, while in passive hyperemia there is a slowing of the blood-current,—a partial or complete stagnation of the movement of the blood through the vessels. Local active hyperemia may be produced through various forms of stimulation applied to the surface of the body, acting through the vaso-motor system of nerves, like heat, cold, mechanical pressure, rapid blows sufficiently light not to cause

pain,—for instance, the blows of the hand, mechanical or electric mallet in filling teeth, and the action of the faradic current. The stimulation by the faradic current, when applied to the surface of the body, may be likened to the mechanical stimulation of that form of massage known as *hacken*,—rapid and light blows made with the edges of the hands.

FIG. 28.



Connective-tissue corpuscles pigmented.

NORMAL CAPILLARY VESSELS; BLOOD-CORPUSCLES IN THEM. BRANCHED CONNECTIVE-TISSUE CORPUSCLES WITH PIGMENT.

Vulpian demonstrated the vaso-motor effects upon the circulation by faradic stimulation of the peripheral segment of the lingual nerve in a dog. He found as a result of this stimulation a considerable dilatation of the vessels in the region of that half of the tongue to which this nerve has its distribution. The mucous membrane on the corresponding side of the frenum linguæ of this half of the tongue also became bright red, while the principal vein became turgescent, and the blood, both in it and in its tributaries, was of bright color, like arterial blood. There was also a perceptible rise in the temperature of the part.

From the experiments of Claude Bernard and others upon the

physiologic action of the vaso-motor system, it was demonstrated that section of the sympathetic nerve would produce a hyperemia of the part, through paralysis of the vaso-constrictor nerves. Stimulation of the chorda-tympani nerve resulted in dilatation of the blood-vessels of the submaxillary gland. This served to prove the existence of two sets of nerves, one vaso-constrictors, and the other vaso-dilators, which placed the vascular system under the control of the vaso-motor centers. There is also a system of periphero-vaso-motor nerves, under the control of what is known as the perivascular ganglia. The continuous action of these ganglia and of the constrictors secures a state of chronic contraction of the muscular walls of the blood-vessels. The dilators come into action only under exceptional circumstances. Stricker is of the opinion that most of the physiologic and pathologic hyperemias are the result of irritation of the dilators. The existence of such a system of nerves and ganglia has never been positively demonstrated, either anatomically or microscopically, although microscopic clusters of ganglia have been discovered upon the arteries of the submaxillary glands, and in other locations.

As hyperemia may be caused by paralysis of the constrictors, or by irritation of the dilators, two forms of active hyperemia may be recognized. When caused by paralysis of the constrictors, it is known as *hyperemia of paralysis*. When caused by irritation of the dilators, it is known as *hyperemia of irritation*. (Warren.) These elements, which combine to form the vaso-motor system, are so nicely adjusted that they counteract each other, and when disturbances arise in the circulation in one direction, a reaction in the opposite way may soon occur to restore the normal condition.

Injuries of various forms which affect the sympathetic nerves are the most common cause of the hyperemia of paralysis. Warren mentions a case in his hospital service in which injury to the cervical sympathetic was immediately followed by changes in the pupil, and hyperidrosis of the injured half of the face and neck. Syncope following blows upon the chest and abdomen, and resulting in death or recovery, are the result, no doubt, of reflex paralysis of the heart and abdominal vessels.

Blushing and the redness of the face following the use of tea, coffee, and alcoholic stimulants are considered *hyperemias of dilatation*. This form of hyperemia also accompanies the pain of facial neuralgia, causing flushing of the forehead and face, redness of the conjunctiva, and secretion of tears. It is also associated with hemicrania, and with certain forms of peripheral disturbances of the nervous system, the production of herpes zoster being a marked example. Recklinghausen is of the opinion that all reflex hyperemias are *hyperemias of dilatation*.

The hyperemia observed in parts separated from the nerve-centers,

like transplantation flaps, and in other portions of the body which have been separated by division of the nerves, is caused by *paralysis of the perivascular ganglia.*

Exudation.—Exudation is the process by which the corpuscular elements of the blood and the liquor sanguinis pass through the walls of the blood-vessels into the tissue spaces beyond. Exudation is the result of changes in the vessel-walls, which permit the passage or leakage of the circulating fluid through their walls. Ziegler says, "It may be accepted as an established fact that in inflammation the vessel-wall is affected, but it is still questioned by some whether the affection is of the nature of a chemical alteration, or a mere widening of pre-existing intercellular spaces." Burdon Sanderson believes that "it is due to the loss of the power by the vessels of resistance to dilatation, and the loss of vital power, in consequence of which leakage takes place."

Inflammation is divided into two forms, viz: acute and chronic; and these again into many varieties, according to the anatomical location of the disease, as taught by Virchow, such as catarrhal, fibrinous, parenchymatous, phlegmonous, indurative, degenerative, scrofulous, and infective.

In acute inflammation the disease runs a more or less rapid course, and the symptoms are marked, while in the chronic form the symptoms are all less prominent, and any or all of the cardinal signs may be so slight as to escape notice altogether. A form between these two conditions has been denominated subacute inflammation.

Catarrhal inflammation is an inflammatory condition of the mucous membrane in particular, wherever found in the body.

Fibrinous inflammation may be regarded as the usual form found in serous membranes and the connective tissue. Fibrinous exudates, however, often form upon mucous surfaces, as in diphtheria and membranous croup.

Parenchymatous inflammation is a term applied to those changes which take place in the special tissues of organs, independent of the connective-tissue frame-work,—affecting the elementary components of a tissue,—the cellular elements,—and expressed by a tendency to effusion of plastic material from the blood-vessels.

Phlegmonous inflammation is principally confined to the connective tissue in the form of abscesses.

Indurative inflammation is that variety which is productive of new-tissue formations in the interior of organs.

Degenerative inflammation may attack any of the tissues, and produce a retrograde metamorphosis in their structure. Examples are, fatty degeneration, caseation, calcareous degeneration, etc.

Scrofulous inflammation—the use of this term is now questioned by good authorities—is a type of inflammation occurring in cachectic

individuals, whose tissues injure easily and heal slowly, and are prone to degenerative changes.

Infective inflammation is produced by the introduction into the blood of infective materials, usually derived from the entrance of bacteria, or from some pre-existing inflammation. Examples are erysipelas, pyemia, tuberculosis, syphilis.

The terms *idiopathic* and *traumatic*, as applied to inflammation, are now considered as obsolete.

General Inflammation is the condition known as fever. Fever involves a rise in the body temperature, weakening and acceleration of heart-action, disturbances of the nervous system, and interruption in all of the processes of nutrition.

Temperature.—The normal temperature of the body is 98.5° Fahrenheit. When it rises above this it indicates fever; when it falls below it indicates shock or collapse. A temperature of 105° is generally considered dangerous, while 107° is usually fatal. It rarely occurs that the temperature reaches a higher degree than this, though exceptional cases are on record which reached 110° to 112°. Richet recently called the attention of the Biological Society of Paris to a remarkable case of hyperpyrexia in a woman suffering from intermittent fever. While the temperature in the morning was no higher than 102° Fahrenheit, in the evening it ascended to 113°. Upon two different occasions it rose to 114.8°. The utmost care was used to avoid deception. Moreover, under the influence of quinin, it fell to 96.8°, and when the quinin was suspended, the temperature rose again to 118.8°. After a fresh exacerbation of longer continuance, the temperature fell to normal, and the patient recovered.

In fevers generally the temperature is highest in the evening and lowest in the morning. In the fever accompanying difficult dentition, it is generally the reverse of this, viz, highest in the morning and lowest in the evening.

Pulse.—The normal pulse in an adult is about 75 beats per minute. In infants it ranges from 120 to 140 per minute. Very old persons have a much higher pulse-rate than people in middle life. A full, rapid, bounding pulse is indicative of high fever, and bears a relative ratio to the increase in bodily temperature. For instance, a temperature of 103° would be accompanied with a pulse-rate of about 100. A soft, rapid, small, flowing pulse is indicative of great weakness, and of collapse if occurring in fevers. The pulse in severe cases of pneumonia may run from 120 to 130 beats per minute. An irregular or intermittent pulse is indicative of functional or organic disease of the heart.

Symptoms of Acute Local Inflammation.

Redness.—This symptom is persistent, and is due to hyperemia. By digital pressure the capillaries can be emptied, but on removing the pressure the redness immediately returns. The shade of color depends upon the freedom from obstruction in the vessels, and the rapidity of the circulation. When the color is dark or purplish it denotes stasis; rose-red streaks along the track of the lymph-vessels indicate lymphangitis; a dark red track along the course of the veins would point to phlebitis; while a copper-red color would denote syphilitic inflammation.

Swelling.—This symptom is due to the engorgement of the blood-vessels of the part, to exudation from the blood-vessels, and to proliferation of cells. In acute inflammations the swelling is soft; in the chronic forms it is hard. Swelling is especially marked in loose connective tissue.

Heat.—This symptom is most marked at the center or focus of the inflamed area. It is thought to be produced by the increased rapidity of the circulation, and the volume of blood in the part. Hunter taught that the heat of the part was never above the heat of the internal organs. Hunter's Law reads as follows: "In inflammation the heat of the part is increased above the normal temperature of the part, but not beyond the temperature of the internal organs."

Pain.—This symptom is persistent, and is increased by pressure, by motion of the part, or by general exercise. Exercise increases arterial tension, and thus augments the pain. The pain is most intense in dense structures, and is mainly due to mechanical pressure upon the nerve-filaments, and is sometimes reflected to regions remote from the seat of the inflammation. Examples are, knee pain in hip-joint disease, shoulder pain in hepatitis, otalgia in pulpitis.

Disturbance of Function.—This symptom is marked in its action upon the secretions, which often become perverted or suppressed. The reflexes are generally exaggerated. Examples are the tenesmus of dysentery, the strangury of cystitis, the convulsions of teething. Non-sensitive parts become hypersensitive, examples being the pain of pleurisy, peritonitis, teething, or decayed dentine in vital teeth.

Description of the Inflammatory Process in the Vascular Tissues.

The phenomena of inflammation in the vascular tissues are best studied in the tongue, the mesentery, or the web of the frog's foot. A good microscope is indispensable.

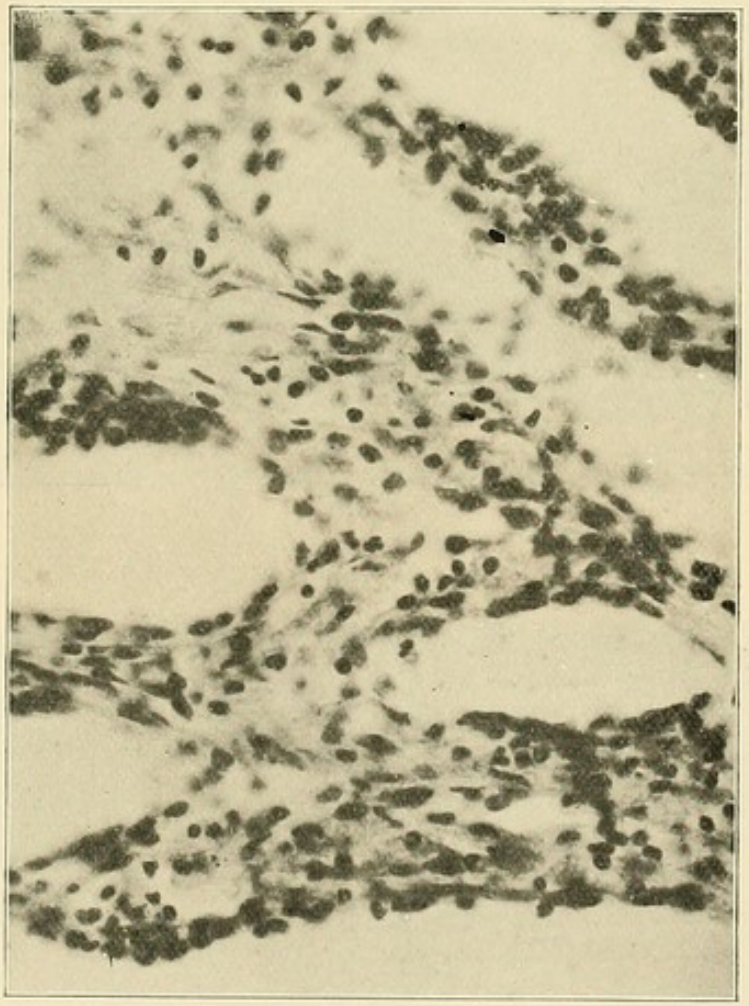
The process may be divided and briefly described in the following order: Irritation, Hyperemia, Expansion of Blood-vessels, Retarded Flow of Blood, Migration of Leucocytes, Exudation of Liquor

Sanguinis, Partial Stasis, Resolution, Complete Stasis, Suppuration, Ulceration, Gangrene, Necrosis, Somatic Death.

Irritation, in some of its forms,—traumatism, heat, cold, acids, alkalies, vesicants, micro-organisms, and their products,—is always necessary to establish the phenomena of inflammation in the vascular tissues.

Hyperemia is caused by an increased flow of blood to the part, as a result of the stimulation of irritation.

FIG. 29.



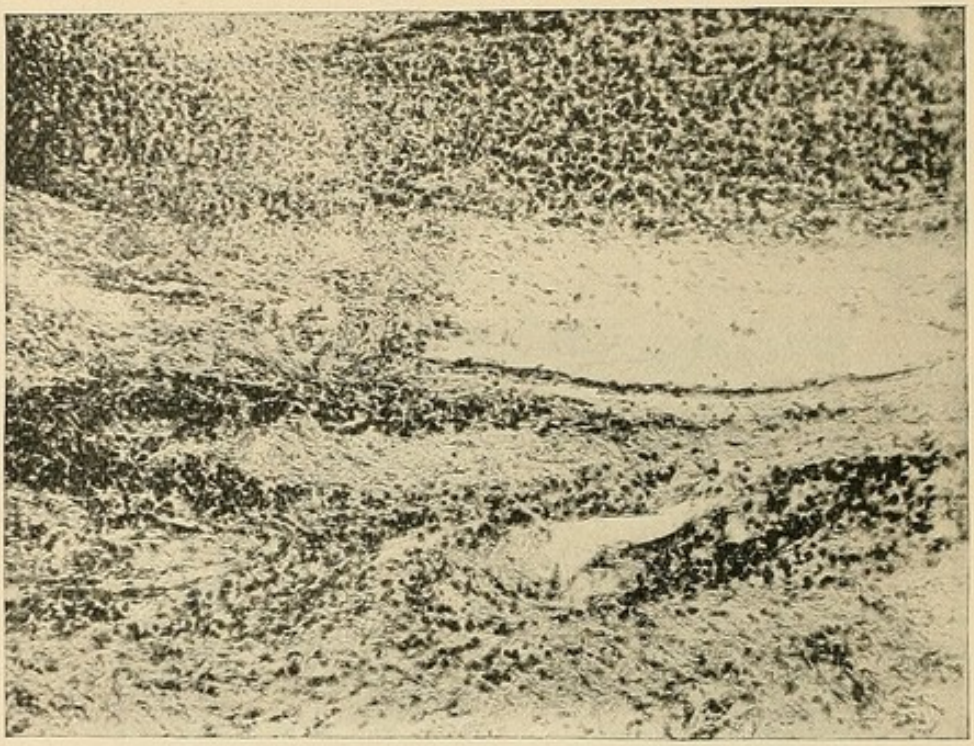
INFLAMMATION OF THE PERICEMENTUM, SHOWING THE LEUCOCYTES.

Expansion of blood-vessels results from the increased flow or volume of blood entering them, and paralysis of their muscles.

Retarded flow of blood is caused by the continued expansion of the blood-vessels, and adhesion of the leucocytes—colorless blood-corpuscles—to the sides of the vessels. This stage in the process constitutes acute congestion.

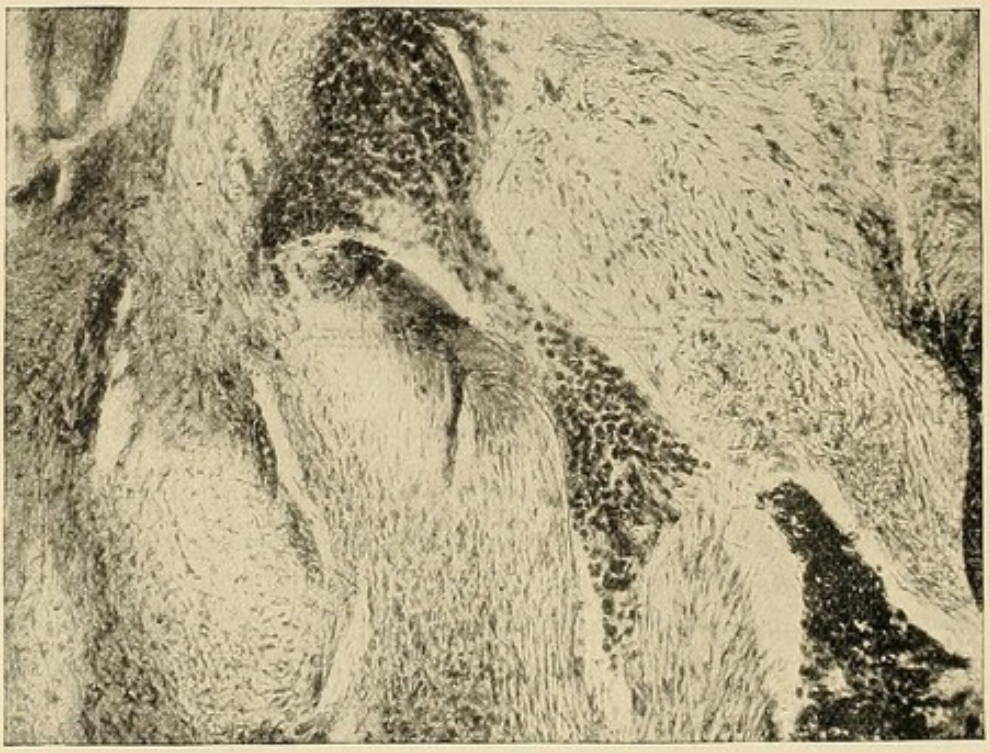
Migration of leucocytes. These corpuscles escape through the walls of the vessels by an ameboid movement, and collect in the

FIG. 30.



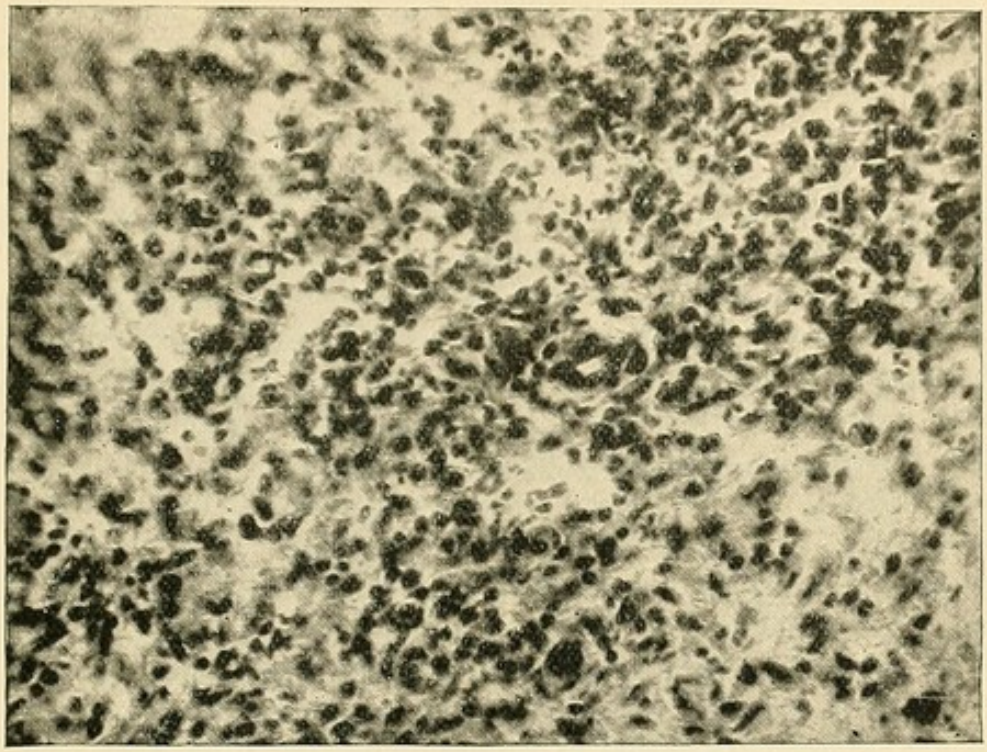
INFLAMMATION OF THE PERICEMENTUM, SHOWING INFLAMMATORY EXUDATES. (After Talbot.) × 480.

FIG. 31.



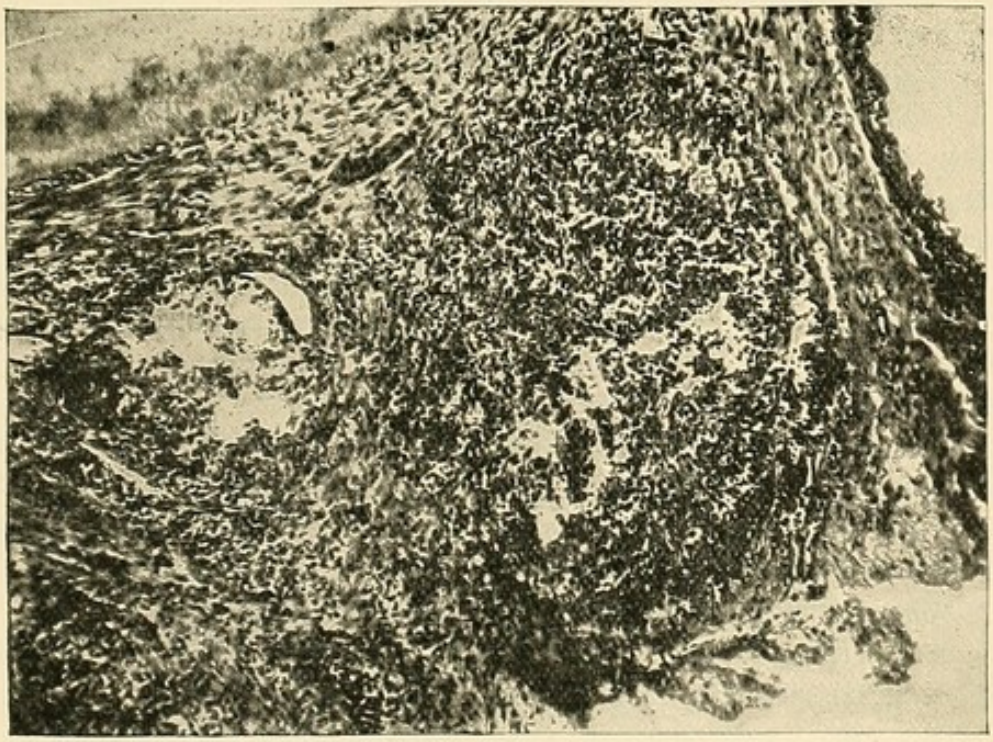
INFLAMMATION OF THE PERICEMENTUM, SHOWING MIGRATED BLOOD-CELLS, AND COAGULUM OF PLASMA. (After Talbot.) × 480.

FIG. 32.



INFLAMMATION OF THE PERICEMENTUM, SHOWING CONNECTIVE-TISSUE SPACES CROWDED WITH INFLAMMATORY EXUDATES PRECEDING STASIS. (After Talbot.) × 480.

FIG. 33.



INFLAMMATION OF THE PERICEMENTUM, SHOWING STAGE OF DEGENERATION AND COMMENCING LIQUEFACTION OF TISSUE. (After Talbot.) × 480.

meshes of the connective tissues (Fig. 29). The red corpuscles also, in some instances, pass through the vessel-walls, but, not having the power of migration, they are consequently found in the immediate neighborhood of the blood-vessels.

Exudation of liquor sanguinis causes induration and swelling, accompanied by coagulation of the fibrinous elements. The coagulation of this plasma incloses the migrated corpuscles, and thereby prevents their further movement. (Figs. 30, 31.)

Partial stasis. The leucocytes adhere to the walls of the vessels. The liquor sanguinis separates and flows in a current next to the walls (plasma layer), while the red corpuscles move in a stream through the center (axial stream), occasionally stopping for a moment, and then slowly moving on again in an irregular manner.

Resolution. At this stage, resolution—a return to normal function—may take place under favorable conditions. The blood-current gradually resumes its natural flow; resorption of the exudation takes place, the induration and swelling disappear, the pain ceases, and the tissues regain their normal color, or complete stasis takes place.

Complete stasis is that condition in which the circulation of the blood is completely arrested in a more or less extensive portion of the inflamed tissue. (Fig. 32.) When this occurs, gangrene and necrosis are the result, and it may be followed by suppuration.

Suppuration (molecular death). In suppuration the exuded blood-corpuscles or leucocytes lose their vitality. Originally they were tissue-builders, thrown out to repair damages; failing in this, degeneration and liquefaction take place (Fig. 33); they form with the exuded liquor sanguinis a thick creamy substance known as pus.

Ulceration (molecular death of tissue). This process causes a break in the continuity of the tissues, and is accompanied by a discharge. The surfaces become vascularized, and are converted into granulations; or

Gangrene (slough—death *en masse* of soft tissue). This condition is the result of complete stasis. The soft tissues are cut off from their nutrient supply, lose their vitality, and slough away in portions corresponding to the area thus involved.

Necrosis (exfoliation—death *en masse* of bony tissue). The hard tissues, when deprived of their blood-supply, also die *en masse*, separation finally takes place between the vital and the non-vital parts, and the necrosed portion is exfoliated.

Somatic death is death of the whole body.

In the further study of this subject the term necrosis will be applied to death *en masse* of bone-tissue only.

NOTE.—The above division of the various processes in inflammation is somewhat arbitrary, but the writer believes that by this plan the student will better understand and retain the subject.

REVIEW.

CHAPTER III.

What is the definition of Inflammation?
Name the physical signs of inflammation.
What is Irritation?
What are the general forms of irritation?
What is a Mechanical irritant? Chemical? Septic? Nervous?
Describe the general features of inflammation.
How may it terminate?
What two factors determine the classification of inflammation?
Describe the condition known as hyperemia.
What position does it occupy physiologically and pathologically?
What is the definition of hyperemia?
What is the definition of plethora?
What is the definition of ischemia?
What is the definition of anemia?
Into what forms is hyperemia divided?
What is the difference between the active and passive forms?
How may active hyperemia be produced?
How does section of the sympathetic nerve produce hyperemia?
What are the nerves called which preside over the physiologic functions of the blood-vessels?
How many sets of nerves are found in these vessels?
What are they called?
Name the forms or varieties of *active* hyperemia.
Describe hyperemia of paralysis.
Describe hyperemia of irritation.
What forms of hyperemia are considered hyperemias of dilatation?
Under what conditions is paralysis of the perivascular ganglia observed?
What is Exudation?
What is the cause of exudation?
What are the theories in relation to the causes of the changes in the vessel-wall?
Describe acute local inflammation.
Describe chronic local inflammation.
What is the definition of Catarrhal inflammation?
What is the definition of Fibrinous inflammation?
In what diseases may fibrinous exudates appear upon mucous membrane?
What is the definition of Parenchymatous inflammation?
How is it expressed?
What is the definition of Phlegmonous inflammation?
How is it expressed?
What is the definition of Indurative inflammation?
What is the definition of Degenerative inflammation?
Name some of the forms of degeneration.
What is the definition of Scrofulous inflammation?
What is the definition of Infective inflammation?
Name examples of this form of inflammation.
What is general inflammation?
What is the normal temperature of the body?

What does a sub-normal temperature indicate?
What is the normal pulse-rate of an adult?
What is the cause of redness in inflammation?
What is the cause of swelling?
What is the cause of heat?
What is the cause of pain?
Describe the inflammatory process in vascular tissues.
What is the cause of hyperemia?
What is the cause of the expansion of blood-vessels?
What is the cause of the retarded flow of blood?
What do you understand by the migration of the leucocytes?
What do you understand by the exudation of liquor sanguinis?
What do you understand by partial stasis?
What is resolution?
What is suppuration?
What is ulceration?
What is gangrene?
What is necrosis?
What is somatic death?

CHAPTER IV.

INFLAMMATION (Continued).

Suppuration.

Definition.—Suppuration. (Lat. *suppuratio*, from *suppurare*, to form pus.) The formation of pus.

Suppuration is the most common or frequent termination of acute inflammation. An inflammation which terminates in this manner is termed Suppurative Inflammation.

Suppuration is a process by which the morphologic elements produced by the inflammation—the leucocytes and the embryonic cell formed from the fixed tissue-cells—are converted into pus-corpuscles, and the intercellular substance of the tissues is liquefied.

"Suppuration takes place in the tissues by virtue of the peculiar peptonizing or digestive action which the bacteria exert upon them." (Warren.)

When pus collects and forms an abscess it exerts a solvent action upon the tissues, as evidenced by the presence of broken-down tissue-cells and remains of tissue, mixed with pus-corpuscles. There must therefore be some chemical substance in the pus which sets up this solvent or digestive action.

The *direct cause* of suppuration is the action of certain specific micro-organisms—the pus-microbes—upon the tissues, the leucocytes, and the embryonal cells.

Pus may be produced, however, under given circumstances, without the presence or intervention of micro-organisms; as, for instance, by the introduction under the skin of certain chemical irritants.

Heuter and his school took a radical position in reference to the power of micro-organisms to cause the production of pus, and stated that there could be no pus without bacteria. This statement met with strong dissent on the part of many close observers, who were not ready to grant such complete control to the ubiquitous microbe.

Billroth believed that bacteria were not the cause of suppuration, but only an accompaniment, and that the active cause was a chemical ferment. Pasteur and others claimed to prove this position, by producing suppuration with pus which had been subjected to a sufficient degree of heat to destroy the bacteria contained in it. It was also

found by experiment that certain chemical substances inserted under the skin would produce pus. Councilman first proved the fact that croton oil when injected under the skin in rabbits would produce suppuration without the action of micro-organisms. The early experiments in this line were very conflicting; some observers succeeded in producing an aseptic pus,—others a septic product, with the same chemical agent. Christmas could not produce suppuration in rabbits with turpentine or mercury, but succeeded with dogs. These conflicting results were due in some cases to imperfect asepsis, in others to the fact that the same chemical substance would produce suppuration in one species of animal, and not in another. Warren says, "It must be conceded that it is possible to produce suppuration without the direct intervention of bacteria, but all (authorities) are agreed that mechanical irritation or foreign bodies are unable to produce suppuration without the aid of bacteria."

Senn believes all inflammatory wound complications, including suppuration, are caused by the introduction into the tissues of pathogenic micro-organisms, the clinical varieties being mostly determined by the intensity of the infection, the manner of localization, and the degree of resistance possessed by the tissues. The same streptococcus which produces a simple abscess is likewise the most frequent cause of progressive gangrene, and of that most grave form of suppuration known as pyemia.

In suppurative inflammation two forms of leucocytes are found; one is mononucleated, the other polynucleated. (Warren.) The former is found only in limited degree in the early stages of suppuration in acute inflammation, but in its later stages, and in the chronic forms of inflammation, it is present in greater numbers. They are derived from the blood, and from the fixed tissue-cells. The latter are the wandering cells, described by Ziegler. They are distinguished from the pus-cells by the larger size of the nucleus. The polynucleated cells possess two or three nuclei, or the nucleus is peculiarly deformed. This change in the shape of the nucleus does not seem to be the beginning of the karyokinetic process—cell division—but rather one of degeneration preceding the final breaking down of the corpuscles. A few tissue-cells are also found mixed with the pus-corpuscles. The polynucleated cells may be considered as a type of the pus-corpuscle.

In addition to the cells already mentioned, there are found larger cells, with a single large, bright, oval nucleus, known as "fibroblasts." These in the later stages of suppuration increase in numbers until they become more numerous than the pus-corpuscles, and are the active agents in the process of repair.

The *indirect causes* of suppuration are the inflammatory phenomena

of exudation, the crowding of the connective-tissue spaces with the corpuscular elements of the blood and the consequent pressure upon the capillary blood-vesels, resulting in complete stasis and death of the tissues involved. All conditions which impair cell-nutrition favor the suppurative process. (Senn.) Death of the tissues is not always necessary to produce suppuration, but the changes which take place in the affected parts are those which are expected to follow intense irritation, viz: hyperemia, exudation, coagulation of fibrinous elements, and partial stasis.

Suppuration produced by chemical pyogenic substances, like metallic mercury, turpentine, concentrated ammonia, and croton oil introduced under the skin hypodermically—with strict antiseptic precautions—produce a sterile aseptic pus, which is very different from clinical pus, while the inflammation and suppuration do not follow the progressive course of an infectious inflammation.

In suppuration due to infectious inflammations, the direct cause which produces it multiplies in the tissues. Consequently it has a tendency to become progressive, while from the pus which is produced the pathogenic micro-organism—pus-microbe—can be cultivated, and if introduced into another organism, will produce inflammation and suppuration. (Senn.)

The power of pyogenic micro-organisms to produce pus seems to lie in their ability to liquefy the fibrinous exudates; but in order to accomplish this it is necessary that they be present in large numbers. In certain forms they exert a chemical action which results in the death of the tissues.

Pus.—Pus is a thick, creamy liquid of specific gravity 1030, a product of suppuration, consisting of degenerated leucocytes, living and dead, liquor sanguinis, granular detritus from broken-down cellular structures, fat-globules, and sometimes flakes of coagulated fibrin. Micro-organisms are plentiful. Those which produce suppuration are the Staphylococcus pyogenes aureus, Staphylococcus pyogenes albus, Micrococcus pyogenes tenuis, and the Streptococcus pyogenes. The staphylococci and the streptococci are the most common forms which produce suppuration.

Varieties of Pus.—Thick, cream-like pus, without odor, coming from acute inflammation in healthy subjects, is termed *good* or *laudable*.

Thin reddish pus, mixed with blood, comes from chronic ulcers and malignant disease, and is termed *sanious*.

Thin, watery, irritating pus comes from chronic ulcers, bone-disease, etc., and is termed *ichorous*.

A sanious pus, containing flakes of coagulated fibrin, coming from chronic abscesses associated with bone-disease, is termed *curdy* or *cheesy*.

Thick, ropy pus comes from syphilitic abscesses, and is termed *gummy*.

Thin, watery pus coming from inflamed mucous surfaces, is termed *muco-pus*,—mucus mixed with pus. Sometimes it is very offensive, having an odor of hydrogen sulfid, when retained in cavities like the antrum of Highmore, or the frontal sinus. The odor is the result of putrefaction.

Thin, watery pus, containing considerable fibrin, coming from serous membranes like the pleura and peritoneum, is termed *sero-pus*.

When pus is not discharged it may undergo disintegration and be absorbed, or its more liquid portions, together with the surrounding affected tissues, may undergo fatty degeneration or form a cheesy mass, which is termed caseation.

Constitutional Symptoms of Acute Inflammation.—The most common constitutional symptom of acute inflammation is fever. Fever may be sthenic or asthenic in form. *Sthenic fever*—sthenic means strength—is characterized by full, strong, rapid pulse, flushed face, injected conjunctivæ, increased temperature,—100° to 103° F.,—headache, lumbar pains, troubled sleep; special senses often hyperesthetic; secretions diminished; urine dark colored, irritating, and of high specific gravity; thirst, tongue coated (white or yellowish), and bowels constipated. This form of fever is characteristic of strong and robust individuals.

Asthenic fever—asthenic means feeble. This form of fever is common in infants, old persons, and the very feeble. The general symptoms are the same as in the sthenic form, except that in place of overaction of the circulation there is a profound depression, followed by a typhoid condition. The pulse is feeble. The temperature fluctuates between 99°, 100°, to 103° F., or even as high as 105°. The mental condition is dull and torpid, at times delirious. The tongue is dry and coated, brown or black.

Predisposing Causes.—Among the most prominent of the predisposing causes of inflammation may be mentioned that of age. Nutritional changes in growing children readily lead to inflammatory conditions of the mucous membranes, and of the bones, which are not likely to occur in the adult from the same cause. In old age there is a lowering of the vital powers, resistance to the encroachment of pathogenic bacteria is less vigorous, and as a consequence many catarrhal affections are present at this period. Certain morbid conditions of the blood also predispose to inflammatory conditions, like the presence of an excess of uric acid, or of sugar. Individuals with the uric-acid diathesis are prone to rheumatic and gouty affections and to inflammations of the pericementum. There is a well-known tendency in individuals suffering from diabetes to the formation of carbuncles.

The weakened condition of the system, however, may be responsible for this tendency, rather than the presence of the sugar. Infection also more readily takes place in anemic subjects from the injection of the Pyogenes aureus, as proved by Gärtner, thus explaining the reason for the frequency of boils in persons of debilitated health. The excessive use of alcoholic stimulants is also a predisposing cause of inflammatory affections. Climatic influences are very potent factors in predisposing to inflammations,—in cold climates to affections of the throat and lungs, in hot climates to affections of the abdominal viscera.

Symptoms and Diagnosis.—The diagnosis of a typical case of acute inflammation occurring upon the surface of the body, with all the symptoms well marked, would be an easy matter to any one of limited experience. The local symptoms of redness, heat, swelling, and pain are always present in a typical acute inflammation, while the constitutional symptoms of elevation of temperature, etc., are more or less marked. Its character, course, and termination will depend upon the primary cause of the affection, the condition of the patient, and the environment. According to Senn, the nature of the primary cause determines the character and course of the inflammation. The microbes of suppuration, erysipelas, anthrax, glanders, tetanus, and gonorrhea cause acute affections, while the micro-organisms of tuberculosis, lepra, and actimomycosis cause affections which are marked by the chronicity of their course and development.

Acute inflammation may become subacute, and finally chronic. Dental pulpitis and pericementitis occasionally pass through all three of these stages. In acute inflammations, where the diagnosis is clear, it only remains to decide upon the character of the infection.

The fever which attends the inflammation is only a symptom, and is indicative of the introduction into the system of poisonous substances resulting from the pathologic changes which have taken place in the exudates or the fixed tissue-cells, by reason of the action of specific micro-organisms. The micro-organisms which produce acute inflammation differ very greatly in their power to cause elevation of temperature from the substance which they produce in the inflamed tissues. The changes in the tissues caused by the Micrococcus pyogenes tenuis of suppuration do not produce so high a temperature as when caused by the Staphylococcus pyogenes aureus or albus, or the Streptococcus pyogenes. (Senn.)

The general disturbances, such as headache, lumbar pains, loss of appetite, vomiting, constipation, feeling of lassitude, etc., which attend acute inflammations, are caused by the elevated temperature, and the presence of specific ptomaines in the blood.

Just how micro-organisms cause febrile irritations, whether by the production of chemical substances which enter the blood by absorp-

tion, or by their simple presence in the tissues, cannot be definitely stated. It is a well-known fact, however, that many surgical fevers are due to the presence in the blood and tissues of a ptomaine produced by chemical changes occurring in putrefying wounds in the presence of micro-organisms. Febrile disturbance following injury is not always dependent upon the presence of bacteria. Genuine fever may develop in cases where perfect asepsis has been secured, and the wounds have healed by first intention. Simple fractures and other subcutaneous injuries are often followed by elevation of temperature, probably the result of shock. Warren says, "In general it may be said fever is due to the presence in the blood of a pyogenous substance of an organic nature, that may have been produced by bacteria, or have been due to the presence of bacteria, or finally, to some ferment-like substance which has resulted from cell-disintegration;" while Senn says, "The nature of the inflammatory product always answers to the specific action of the microbe."

The inflammation caused by pus-microbes results in the formation of pus, while the microbes which cause chronic inflammation as a rule only convert the pre-existing mature tissue by degenerative metamorphosis into an embryonal form, a granulation-tissue. Those micro-organisms whose existence in the tissues is short-lived, as for instance the streptococcus of erysipelas, may give rise only to an intense hyperemia, with moderate exudation and migration of the blood-corpuscles. Genuine uncomplicated erysipelas is of such short duration that the inflammatory symptoms rapidly subside, and perfect restoration of the parts is accomplished in a few days.

Prognosis.—Resolution is the most favorable termination of inflammation. The inflammatory process is arrested "as soon as the blood which circulates through the vessels restores their walls to a normal condition." When this condition is brought about, resolution is immediately established. The exudation ceases as soon as the vessels are restored to functional activity, and the process of resorption of the exuded plasma and leucocytes begins immediately. The simple serous exudates are those which are most readily resorbed. As soon as the normal nutrition of the part is readjusted, the constituent cells of the tissues which have been injured in the course of the inflammation take on new vigor, and soon recover their normal condition.

Resolution is only possible in the milder forms of inflammation, where the migration of the leucocytes has been moderate in quantity, and where the exudates and the cellular elements have not been converted into pus-corpuscles. In resolution, many of the escaped leucocytes which have retained their vitality return through the vessel-walls into the general circulation, or through the lymphatic system. The blood-corpuscles which remain, both white and red, undergo degen-

eration and liquefaction, and are removed by absorption. The coagulated fibrin of the exudates is transformed into a granular mass, and is then removed in like manner. The embryonal cells which have lost their vitality by reason of the inflammation are also converted into granular masses, and absorbed.

The loss of tissue caused by the inflammation, if not too extensive, and if the remaining parts are healthy, will soon be replaced by the process of regeneration, accomplished through the karyokinetic function of the fixed tissue-cells. The epithelial cells produce epithelium, the muscle-cells generate new muscular tissue, the periosteum forms new bone, the fibroblasts new fibrous tissue, new blood-vessels are formed by capillary offshoots from existing vessels, and new nerves from nerve-cells, etc.

Inflammatory exudates become a source of danger, when, by reason of their amount, they cause mechanical pressure that interferes with the performance of function in important organs, like the heart, the lungs, or the brain. (Senn.) A moderate amount of inflammatory exudation occurring in any of the meninges of the brain may cause death from compression. Effusion into the pericardium of sufficient quantity to interfere with the action of the heart would cause death by syncope. A copious effusion into the pleural cavity, especially if it accumulates rapidly, may so interfere with respiration as to cause death by apnœa, while a slight edema of the glottis or diphtheritic exudation upon the vocal cords may destroy life by causing mechanical obstruction to the entrance of sufficient air to the lungs.

The modifying influences which control the effects of inflammation are the age and general condition of the individual. Infants and elderly persons have little resistive power; consequently, when attacked with inflammation, the disease is prone to lead to serious results. The same is true of persons debilitated from disease or excesses, particularly intemperance in the use of alcoholic drinks.

In tuberculosis there is always danger of extension of the disease to other organs through the specific bacilli which are carried by the circulation and lymphatic channels.

"Chronic suppuration eventually causes amyloid degeneration of the important organs, and death ensues as a result." (Senn.)

REVIEW.

CHAPTER IV.

What is the definition of Suppuration?
What is Suppuration?
What is its direct cause?

How do micro-organisms cause the formation of pus?
What was Heuter's position in reference to the formation of pus?
What was Billroth's opinion?
How many forms of leucocytes are found in suppurative inflammation?
Under what conditions are the mononucleated cells present?
From what are they derived?
How are they distinguished from the pus-cells?
Describe the polynucleated cells.
What is the cause of the change of the nuclei?
What other kind of cells are found mixed with the pus-corpuscles?
Which of these cells is a type of the pus-corpuscle?
Describe the fibroblasts.
What are their functions?
What is the difference between the infectious inflammatory product and that produced by chemical pyogenic substances?
What is pus?
How many forms of pus have been mentioned? Name them.
What is laudable pus?
What is sanious pus?
What is ichorous pus?
What is curdy or cheesy pus?
What is gummy pus?
What is muco-pus?
What is sero-pus?
What becomes of pus when not discharged?
What are the most common constitutional symptoms of acute inflammation?
What are the characteristics of sthenic fever?
What are the characteristics of asthenic fever?
Name the prominent predisposing causes of inflammation.
How does old age predispose to inflammation?
What conditions of the blood are predisposing causes?
What forms of inflammation will climatic changes produce?
What are the diagnostic symptoms of acute inflammation?
Upon what do its character, course, and termination depend?
What forms of microbes cause acute affections?
What forms produce chronic affections?
Of what is the fever attending inflammation indicative?
How do micro-organisms cause the fever?
What are the theories upon the matter?
Is fever following injury ever produced without the presence of bacteria?
What is the difference in the results produced by the pus-microbes of acute inflammation, and those which cause chronic inflammation?
When is the inflammatory process arrested?
When do the exudates cease to be formed?
At what period does resorption begin?
When do the injured tissue-cells begin to recover?
What becomes of the vital leucocytes in the process of resolution?
What becomes of those which have lost their vitality?
What becomes of the fibrin and dead embryonal cells which remain?
By what process is lost tissue restored?
When do the inflammatory exudates become a source of danger?
What modifying influences control the effects of inflammation?

CHAPTER V.

TREATMENT OF INFLAMMATION.

SENN says, inflammation is not a disease, but a symptom; an effort upon the part of the system to eliminate, or render inert or harmless the primary cause; the treatment, therefore, must be directed in each individual case to the symptoms presented.

The *nature* and *tendencies* of inflammation must be thoroughly understood in order to arrive at a rational method of treatment.

Van Buren defines inflammation as "a condition located in the apparatus of nutrition, affecting a limited area, and consisting in temporary perversion of nutrition from its natural and regular order."

The treatment of septic inflammation, as of all other affections, comprehends the questions of prevention and cure. The subject may therefore be divided into *Prophylactic treatment* and *Curative treatment.*

Prophylactic treatment is of first and greatest importance, and the old adage that "an ounce of prevention is worth a pound of cure" never had greater force, nor was ever more strongly indorsed or more firmly believed by the profession and the general public than it is to-day. The evidence of this is constantly before us in the efforts of medical and surgical science to discover means for rendering the animal economy immune to disease, and of methods of combating the spread of contagious and epidemic diseases. At no time in the history of the world has such great advancement been made in this direction as during the last decade. The study of the science of bacteriology has done more to awaken interest and stir enthusiasm in the subject of preventive medicine than all other subjects combined; in fact, it is the key to the whole situation.

Prophylactic treatment in acute septic inflammation by antiseptic methods is of first importance, and is usually very satisfactory in its results. Preventive antiseptic precautions have made modern surgery what it is to-day. The surgeon or the dentist who fails to properly appreciate the value of prophylactic treatment from the standpoint of antisepsis is behind his day and generation, and cannot hope for the same degree of success in the prevention and cure of disease as those who follow the antiseptic method. The specific action of the pus-microbes is no longer doubted by even the most skeptical.

Lister, the apostle of antiseptic surgery, and his early disciples, by their work and teaching, have been the means of saving thousands upon thousands of human lives that otherwise would have been lost.

The mortality of even the most desperate operations, when antiseptic methods can be used, has been so reduced that many have been encouraged to suggest and to attempt operations which at any previous time would have been considered the vaporings of a diseased mind (Senn), or stigmatized as criminal.

The abdominal cavity is now opened with impunity, and operations made upon the inclosed viscera that a few years ago would have been considered impossible. The chest is explored, and portions of the lung removed. The cranium is perforated, and tumors extracted from portions of the brain, and a large percentage of the individuals operated upon recover, and are restored to health and usefulness.

By the introduction of antisepsis the surgeon has it in his power to prevent the almost innumerable complications surrounding traumatic injuries, and their too often fatal terminations.

The preventive treatment of inflammation in relation to wounds and other avenues of infection, consists in so protecting the locality which has been deprived of its natural barrier against the entrance of pathogenic micro-organisms—the skin and mucous membrane—by first securing an aseptic condition of the parts, and maintaining this by bringing in contact with it only such things as are in themselves antiseptic, or as have been rendered aseptic by thorough sterilization. In inflammations where there is no external traumatism through which infection could have entered, it must be taken for granted that the micro-organisms have found ingress to the circulation through some slight break in the continuity of the external tissues, which has left no mark, and has escaped the notice of the patient; or the infection may have entered through some of the various appendages of the skin or mucous membrane, and later located in some organ or part which by its abnormal condition is prepared to foster their growth, a location which has at the time a lowered vital resistive power. (Senn.)

Prevent the infection of a wound by pus-forming microbes, and inflammation is prevented. Both in theory and clinical experience this axiom is eminently true.

Curative Treatment.—It has already been intimated that irritation and inflammation sustain to each other the relationship of cause and effect. It is therefore readily seen that curative treatment, to be effective, must reach beyond the use of palliative measures, and destroy, eliminate, or render inert the active or exciting cause, by means adapted to the nature, course, and progress of the inflammation and the condition of the patient. These methods may be radical or conservative, but in either case they should be directed to the removal of

the cause of irritation at the earliest practicable moment, compatible with the surrounding conditions. When the disturbing cause is purely local its removal may sometimes be easily accomplished by resorting to a surgical operation, as, for instance, the removal of a fragment of bone, splinters, bullets, or other foreign substances; the resection of diseased joints (Senn), the extraction of diseased teeth, the evacuation of pent-up secretions, releasing strangulated tissues, rendering innocuous infective or acrid discharges, or promptly displacing the contents of abscesses, and establishing drainage to prevent further accumulation.

Local Treatment.—Senn says, recognizing the fact that acute inflammation, wherever it occurs, is the result of the action of certain specific micro-organisms upon the vessel-walls and the tissues outside of them, the rational treatment would seem to be to destroy the microbes in the tissues as soon as their presence is discovered, by the saturation of the tissues with some solution having germicidal powers.

Heuter advised and extensively practiced this method long before it was known that certain microbes and definite forms of inflammation had any relationship with one another. He also claimed that all inflammations were caused by certain noxæ (harmful, hurtful, baneful substances) introduced from without the body, and which he tried to combat by saturating the tissues with antiseptic solutions. His favorite remedy was a 3 to 5 per cent. solution of carbolic acid. This he introduced into the tissues by means of a Pravaz syringe, armed with a long needle having several lateral openings.

In adults he often injected as much as ten grammes. In treating large, open, granulating surfaces, or tubercular foci, he used an infuser in place of the syringe. This was a graduated glass cylinder, having a rubber tube attached, and this joined to the needle; and he depended upon gravitation to diffuse the solution through the soft granular tissue. This method has never been generally practiced, for the reason that except in inflammatory conditions of very limited area, there would be too great danger of causing the death of the patient by a toxic dose of the germicidal agent.

Corrosive sublimate, permanganate of potassa, nitrate of silver, iodin, and other germicidal agents, have all been used in the same way, but with no degree of satisfaction.

If this method is practiced, it should be under strict antiseptic precautions, the amount of the agent introduced should never exceed the dose given internally, and the danger from a toxic dose should be remembered if the injection is repeated.

Depletion.—For generations it has been the practice to deplete the system by venesection, and the internal use of emetics and cathartics, to reduce the arterial tension in inflammation, and thereby prevent, or

cut short, the more serious symptoms. Blood-letting is rarely ever resorted to in these days in the treatment of any form of inflammation. General depletion, if carried far enough to weaken the vital forces, diminishes the prospect of a favorable termination of the inflammation. General depletion favors stasis; local depletion often proves of great value by relieving the engorged capillary vessels, and thereby preventing stasis.

Leeches should never be used, as they often carry infection which may prove disastrous. Scarification, as generally practiced for cupping, is unsafe, for the reason that it is difficult to keep the instruments aseptic, and the number and depth of the incisions are not under the control of the surgeon.

Local depletion is best obtained by incisions made with a scalpel, and free bleeding promoted by the use of warm water.

Vomiting and diarrhea sometimes occur in the early stages of acute inflammation. This is an attempt upon the part of the system to eliminate through the gastro-intestinal mucous membrane the toxic elements which have been introduced into the circulation by the presence of the micro-organisms.

This process of elimination can be promoted by administering a few doses of hydrargyri chloridum mite, followed by a saline cathartic, and will also many times control the vomiting and diarrhea more promptly than the remedies usually administered for that purpose. (Senn.)

Rest. —Rest of an inflamed organ or part is one of the most valuable adjuncts to the methods of treatment, and should approach, as nearly as possible, physiological rest. Examples, the exclusion of light from an inflamed eye; fixation of a limb with inflamed joint; prevention of occlusion of a tooth that has become sore from pericementitis; interdiction of the use of the voice in inflammatory conditions of the tonsils, pharynx, or larynx. (Senn.) Such rest often affords marked relief from the severity of the pain.

The advantages of elevation of an inflamed part cannot be overestimated, and are most manifest in inflammatory affections of the extremities. The throbbing pain is greatly aggravated when the limb is kept in a dependent position. Elevation not only relieves the pain, but greatly assists in removing the edematous condition. It is often necessary to secure complete rest of the entire body in severe cases of acute inflammation. The recumbent position is usually the best; this relieves arterial tension, prevents unnecessary strain upon the blood-vessels, and reduces the dangers of embolism from the detachment of a thrombus. Persons suffering from alveolar abscesses and pulpitis find a sitting or recumbent position most comfortable; exercise in any form increases the pain by increasing arterial tension.

Cold.—Senn believes the application of cold has been resorted to indiscriminately in the treatment of inflammation. No agent is more potent for good or harm, according to the stage of the inflammatory process to which it is applied. In the early stages of inflammation, before exudation has progressed beyond its first beginnings, and the capillary vessels are dilated and only partially obstructed, it is of great advantage by producing contraction of the blood-vessels, and retarding the growth and multiplication of the micro-organisms.

If stasis has become established, the application of cold will prove harmful, by preventing the establishment of collateral circulation, and increasing the dangers of complete stasis and death of the part.

Cold is most effective in a superficial inflammation, but often proves of benefit in inflammations of the deeper structures when its use is prolonged. (Examples, the pleura, peritoneum, bones, joints, teeth, and the meninges of the brain.)

The best method of applying cold is by means of the ice bag, the part being protected from the danger of freezing by being covered with a wet towel, folded several times. It is most beneficial in those cases in which congestion of the vessels is a prominent symptom, and where redness and heat are pronounced. (Warren.)

Heat.—Heat is most beneficial in the later stages of inflammation, and is best applied by means of compresses wrung out of hot antiseptic solutions, covered with rubber sheeting, and reapplied as often as they become cooled. Hot fomentations favor collateral circulation, stimulate the absorption of the exudates, and relieve the pain. The surface to which the fomentations are to be applied should be thoroughly cleansed with soap and water.

Care must be exercised in the selection of the antiseptic drug; the age and condition of the patient, and the area to be covered, must be taken into consideration, to avoid producing toxic symptoms. Carbolic acid and sublimate solutions must be used with care with aged persons and little children, or with persons suffering from affections of the kidneys. A 1 per cent. solution of acetate of aluminum, a saturated solution of boric acid, or the Thiersch solutions, are entirely safe, and quite as efficacious as the more poisonous drugs. All antiseptic solutions should be made from sterilized water.

Senn thinks poultices of every name and nature should be relegated to the dead past, and never used by any enlightened surgeon, as they are simply hot-beds of pollution and infection.

Warren says heat acts differently according to the degree used. Mild heat favors an increase of hyperemia; greater heat causes constriction of the blood-vessels.

Constitutional Treatment.—Drugs are of little value for reducing the temperature in inflammation. The coal tar derivatives, salicylate

of soda, salol, quinin, and other antipyretics, when employed in large doses to reduce the temperature, accomplish this result at the expense of the vital forces, which are already being taxed very greatly in the effort to eliminate the poisonous elements which have been introduced by the action of the micro-organisms.

Sponging the surface of the body with tepid water, or the use of warm baths, is far more effective in reducing the temperature; and besides being grateful to the patient, promotes elimination (Senn), and the dangers from disarranging the stomach and weakening the heart by large doses of antipyretics are obviated.

Cathartics are very valuable in many cases, and have probably a wider range of usefulness in inflammation than any other class of constitutional remedies. Their chief value lies first in removing the unwholesome ingesta and acrid fecal accumulations from the stomach and bowels. Second, by stimulating the secretions of the gastric and intestinal glands, the liver, the pancreas, etc., they assist the system to eliminate through these channels the toxic elements which have been introduced into the blood by the presence of micro-organisms. Third, by their revulsive action, which, operating upon such an extensive surface in immediate sympathy with the whole nervous system, exerts a powerful influence in withdrawing nervous action, or over-action, from the inflamed part.

Diaphoretics and diuretics are both valuable aids to other means of treatment, as they promote elimination of toxic substances. The kidneys are avenues through which are eliminated micro-organisms that reach them through the general circulation.

The development of symptoms of sepsis in the course of an inflammation calls for the administration of diffusible stimulants, which should be used freely to ward off the dangers from approaching heart-failure. Brandy, cognac, and whiskey are the best for the purpose.

Diet.—The diet should be nutritious, and well selected; meat-broths, beef-tea, and milk should be given freely from the beginning, and if the stomach will bear it, more substantial food. The days of starvation treatment have passed away, and the aim of the surgeon to-day is to sustain the vital powers of the patient, that he may make a more successful fight against disease. If the stomach will not retain food, then the strength of the individual must be sustained by nutritive rectal enemata of peptonized milk and beef-tea, in quantities of two to four ounces, alternately, every six to eight hours, as the condition of the patient suggests. Tonic doses of quinin are sometimes indicated where the inflammatory symptoms are protracted. When the appetite is defective, some of the bitter tonics will be found useful, and after the acute symptoms have subsided, the tincture of chlorid of iron will be found of great advantage.

To relieve pain, chloral and phenacetin are to be preferred to opium. There is great danger in painful chronic conditions of forming the habit of taking anodynes. Caution should therefore be exercised, and it would be better to seek for the cause of the pain, and remove it by local measures, if possible, than to depend upon drugs to give relief.

REVIEW.

CHAPTER V.

Is Inflammation a disease or a symptom?
What is the system attempting to do by the process of inflammation?
What does treatment comprehend?
What is prophylactic treatment?
What is preventive antiseptic treatment?
What have antiseptic methods done for modern surgery?
What does preventive antiseptic treatment consist of?
What are the active agents that produce inflammation?
How will the exclusion of pus-forming microbes prevent inflammation?
What does curative treatment comprehend?
What was Heuter's theory as to the cause of inflammation?
How did he try to prevent it?
What were the dangers of this method?
What other drugs have been used for the same purpose?
What is the value, if any, of general depletion in the treatment of inflammation?
What are the benefits of local depletion?
How may this best be obtained?
How does nature sometimes try to eliminate the toxic substances?
How may the elimination of the toxic substances be promoted?
What is the value of rest in inflammatory conditions?
How does elevation of an inflamed limb relieve pain?
When should cold be applied?
How does it control inflammation?
Where is it most beneficial?
When is its application harmful?
When apply heat?
How is it best applied?
What are its benefits?
What is the effect of different degrees of heat?
Should poultices ever be used?
Why not?
What value, if any, have antipyretics in inflammation?
How may the temperature be reduced without the use of antipyretic drugs?
What is the value of cathartics in inflammation?
How do diaphoretics and diuretics prove beneficial in inflammation?

What should be the treatment when symptoms of sepsis have developed?
What should be the diet in inflammation?
How administered if the stomach will not retain food?
What should be the treatment in cases with defective appetite?
What drugs may be used to relieve pain?
Should anodynes be administered in all cases?

CHAPTER VI.

CHRONIC INFLAMMATION.

Chronic Inflammation is generally preceded by the acute form, which has not passed beyond the stages of partial stasis and suppuration, or of ulceration.

Its Causes are long-continued local irritation, or functional activity, constitutional dyscrasia, or diathesis.

Its most common terminations are Induration, Hypertrophy, Tumefaction, Suppuration, Ulceration, and Fatty Degeneration of the infiltrated tissues, the formation of *cold abscesses* from the breaking down of this degenerative tissue, and Caseation.

Induration (Lat. *induro*, I harden). A process of hardening the tissues from coagulation of the fibrinous elements of the exudates, and new formations in the connective tissues.

Hypertrophy (Lat. *hyper*, excess; Gr. τροφή, nourishment). Enlargement of a part due to constant irritation and congestion, resulting in the formation of new tissue elements of the same character.

Tumefaction (Lat. *tumere*, to swell, *facere*, to make) (a tumor, a swelling). A circumscribed enlargement, caused by proliferation of cells and their organization into new tissue, often of a different character, resulting in the formation of tumors of various kinds, benign and malignant.

Fatty Degeneration. A process or retrograde change, by which the albuminoid elements of the tissues and the exudates are converted into granular fatty matter.

Caseation is a process of degeneration in pus, tubercles, etc., by which they are converted into a soft, cheese-like mass.

The presence of chronic inflammation implies either a continued existence of the original cause, some abnormal condition of the general system, or both. Coucilman thinks "the condition of the individual has often much to do with the chronicity of inflammation." A feeble condition of the circulation results in a tardy and incomplete resorption of the exudates.

The local manifestations of chronic inflammation differ from the acute form only in the degree of severity. The cardinal symptoms are all less marked, or may be so slight as to escape notice altogether.

The vascular changes in the tissues which have been already described come on more slowly, and never assume the degree of severity which is characteristic of the acute form, and for this reason the migration of the leucocytes takes place much less rapidly, and in some instances does not occur at all.

According to Senn, the inflammatory product is composed largely of effused plastic lymph, which undergoes partial organization and causes *induration*, and of embryonal cells derived from the fixed tissue-cell. Sometimes the inflammatory product is largely or entirely composed of these embryonal cells. This is explained by the action of the noxious elements which cause chronic inflammation exerting their baneful effects more directly upon the tissue-cells than upon the capillary blood-vessels.

The effect upon the tissues is to convert the mature tissue-cells into an embryonal form, and to increase their proliferation, thus forming *granulation-tissue* which remains in this condition so long as the primary cause of the inflammation retains its pathogenic qualities, or until *degenerative changes* take place in the new cells. Warren says, "If the exudation goes on to such an extent that the part is completely infiltrated with leucocytes, the structure of the tissue will be seriously impaired and the fibers and cells of the part disappear. The fixed cells undergo proliferation and become indistinguishable from the migratory cells, the intercellular substance is gradually changed into a more or less homogeneous granular material in which the new cells are imbedded, which constitutes granulation-tissue." The degenerative change which most frequently takes place in this temporary tissue is *fatty degeneration*, and this may break down and liquefy, forming *cold abscesses*.

If degenerative changes do not take place in the embryonal cells, and the primary cause ceases to exist, the new cells are either resorbed or converted into mature tissue, and the inflammation results in a *hyperplasia* of tissue.

Chronic inflammation includes all that class of affections known as "inflammatory swellings," or *granulomata*. All of the inflammatory swellings are composed of granulation-tissue irrespective of the nature of the primary cause. The granulomata have been classed by some eminent pathologists with the neoplasms or tumors, because their development is usually attended with none of the characteristic symptoms of inflammation; and on account of their tendency to spread to adjacent tissues and involve remote parts by dissemination through the lymphatic system, they have a close resemblance to the malignant growths.

The granulomata are true inflammatory products.

Under the microscope they exhibit all the characteristic appear-

ances of inflammation, and histologically are composed of embryonal cells, representing the type of tissue from which they had their origin.

Tuberculosis, syphilis, and actinomycosis are examples of inflammatory swellings or granulomata.

Treatment.—*Local treatment* in chronic inflammation would be the same as that followed in the acute form of the affection, as far as the removal of the cause is concerned, the same means being used in both. Another method of treatment sometimes resorted to by surgeons is to convert a chronic inflammation into the acute form of the affection, as this is nature's way of establishing a cure. This may be accomplished by various drugs, such as iodin, nitrate of silver, etc., or the actual cautery or the electro-thermal cautery.

Stimulating applications like the ammonia liniment, tincture of iodin, etc., are useful as aids in promoting resorption. Counter-irritants are sometimes serviceable, but if applied indiscriminately are likely to do more harm than good.

Massage is an exceedingly important and efficient means of treatment in chronic inflammatory affections, when scientifically practiced. Its value lies in the fact that it stimulates the vessels to increased action, assists in restoring a normal circulation through the injured capillaries, and greatly promotes the process of resorption.

The application of hot and cold douches, rest, active or passive movements, etc., adapted to the individual case, will often prove of great benefit and materially hasten a cure.

Constitutional treatment is of the utmost importance in many forms of chronic inflammation, from the fact that there is associated with the local condition a constitutional or systemic dyscrasia, often more alarming than the local manifestation.

Fortunately, however, many of these cases, though they differ widely as to the pathologic characters of the systemic dyscrasia, may be classified clinically as examples of enfeebled constitutions, and as demanding nutritious food, tonics, and perhaps stimulants. (Hamilton.) Such cases are greatly benefited by improved hygienic surroundings, fresh air, sunlight, exercise, nutritious food, tonics, etc., a sea voyage, a trip to the mountains or the seashore.

In tubercular inflammation cod-liver oil and iodin are demanded. In syphilitic inflammations, represented by the gummata, vigorous antisyphilitic treatment soon causes the local lesion to disappear, and greatly improves the general health.

The other specific causes of general enfeebled health are diseases of the digestive organs, the liver and the kidneys, the rheumatic and gouty diathesis, septicemia, pyemia, or other blood-poisoning. These conditions must be combated and the general health improved before a subsidence of the inflammatory symptoms can be reasonably

expected. Gouty and rheumatic conditions may be relieved by a carefully-selected diet and the administration of the salicylic compounds; while inflammations depending upon blood-poisoning will demand other special agents adapted to the elimination of specific microorganisms and their products, such as the compounds of mercury and iodin.

REVIEW.

CHAPTER VI.

What are the causes of chronic inflammation?
What are its most common terminations?
What is induration?
What is hypertrophy?
What is tumefaction?
What is fatty degeneration?
What is caseation?
What does the presence of chronic inflammation imply?
How does the condition of the individual favor chronic inflammation?
How do the local manifestations of chronic inflammation differ from the acute forms?
How do the vascular changes differ?
Of what is the inflammatory product largely composed?
What causes the induration?
From what are the embryonal cells derived?
What part do they form of the exudate?
What is the effect of the poisonous elements upon the mature tissue?
How is the granulation-tissue formed?
What degenerative changes may take place?
What becomes of the embryonal cells after the primary cause of the inflammation has been removed?
What class of affections does chronic inflammation include?
How have the granulomata been classed?
Why should they not be classed as neoplasms?
By what appearances are they classed with the inflammatory swellings?
What is the local treatment of chronic inflammation?
What method is sometimes practiced to change the character of the inflammation?
What is the value of local stimulation?
Are counter-irritants serviceable?—to what extent?
How does massage benefit?
What is the condition of the general system in chronic inflammation?
How should such cases be treated constitutionally?
What remedies are demanded in tubercular inflammation?
How should syphilitic inflammation be treated?
What other specific causes engender enfeebled health?
How may gouty and rheumatic conditions be relieved?
How may noxious elements in blood-poisoning be eliminated?

CHAPTER VII.

ABSCESS.

Definition.—*Abscess* (Lat. *abscedere*, to depart).

An abscess is an accumulation of pus in the tissues surrounded by a wall of *lymph* (formerly termed the pyogenic membrane, from the erroneous notion that it secreted pus). An abscess may be termed a hollow ulcer.

When a collection of pus occurs in such locations as the pleura, pericardium, Fallopian tubes, pelves of the kidneys, peritoneum, etc., the prefix *pyo*, added to the anatomical name of the locality, indicates the presence of pus; thus: Pyo-thorax, pyo-pericardium, pyo-salpinx, pyo-nephrosis, pyo-peritonitis, pyo-ulitis.

Causes.—Suppurative inflammation always precedes the formation of an abscess. Excessive and continued irritation causes so copious an exudation that the lymph-channels are blocked, complete blood-stasis occurs, and coagulation follows. The leucocytes lose their vitality, pressure upon the connective-tissue cells involved in the inflamed area produces a like result in them, and by the action of the micro-organisms which have gained an entrance through the circulation or by other avenues, the tissues and exudates are converted into pus.

When the bacteria accumulate in a mass, as they frequently do, at some particular point in the system, the concentrated action of the bacteria, or of the chemical product, causes coagulation of the serum and of the contiguous tissue,—"coagulation necrosis,"—thus forming a nidus or central point for the development of suppuration, and the formation of an abscess. Around this central point, composed of dead tissue, and containing a nest of micro-organisms, the leucocytes accumulate in great numbers, completely inclosing it by forming a kind of wall. The central mass of dead tissue, and the immediately surrounding intercellular substance, soon begin to liquefy. This liberates the leucocytes, which were entangled in the meshes of the intercellular substance, and they become mixed with the pus. This process continues, and the fluid contents of the abscess gradually increase in amount; tension of the tissues results, and they eventually give way at the location offering the least resistance. This is termed *"pointing,"* and through this opening the contents are discharged. Active cell

proliferation in the fixed tissue-cells is going on at the same time in the outer portion of the wall of leucocytes. Lining the abscess cavity is a tissue known as *"granulation-tissue,"* which by its growth repairs the damage caused by the destruction of tissue. This tissue is composed chiefly of small, round cells, with scanty intercellular substance, but very rich in capillary blood-vessels.

Classification.—Abscesses may be classed as *superficial* and *deep-seated, diffuse* (phlegmonous) and *circumscribed, acute* and *chronic.*

Superficial or subcutaneous abscesses have a tendency to spread laterally. This is explained by the fact that the pus moves in the direction of the least resistance. The loose subcutaneous connective tissue favors this route of extension, and offers but little resistance to the pressure caused by the accumulated pus.

Deep-seated or sub-fascial abscesses burrow along the sheaths of muscles and blood-vessels in the connective tissue, and may even dissect the periosteum from the bone. Such cases are not infrequently met with in connection with abscesses of the neck resulting from abscessed teeth, or abscesses resulting from the irritation of unerupted lower third molars, and in compound fractures of the lower jaw followed by septic inflammation. Inflammation of the lymphatic glands situated in the upper triangle of the neck, or in the submaxillary triangle, frequently results in the formation of deep-seated abscesses, which burrow downward to the anterior mediastinum. This is caused by their inability to penetrate the deeper layer of the cervical fascia, the action of the law of gravitation which carries the pus downward, and the slight resistance offered by the intermuscular connective tissue.

The retropharyngeal abscess is an example of a still deeper variety. This abscess is situated in the space between the œsophagus and the spine. The anatomical relations of the part prevent the pointing of the abscess at its primary seat; the œsophagus being in front, the spine behind, and the sheaths of the blood-vessels—which are quite unyielding in this location—on either side, while the space between is composed of loose connective tissue, which favors its downward course into the posterior mediastinum. In the early stages the symptoms are often not well marked. Such abscesses occasionally point at or near the jaw, but more often they follow the downward course already indicated. Retropharyngeal abscesses usually originate in a tubercular nodule located in the body of a cervical vertebra.

Diffuse (spreading,—phlegmonous) abscess is a term applied to that form of abscess which spreads in various directions. Its location may be in the subcutaneous connective tissue, submucous tissue, intermuscular connective tissue, or subperiosteal tissue. This form of abscess is caused by the infection of the tissues with the pus-producing streptococci, and the favorite routes through which it spreads are the

connective tissues and the lymphatic glands. The term *diffuse purulent infiltration* is sometimes applied to this form of abscess. In this type of the affection, all the symptoms of acute inflammation are present, and the extent of the involved tissue is often considerable. The swelling is usually great, and the surface of the distended integument may develop vesicles filled with serum. The constitutional symptoms are also well marked, the disturbance sometimes being profound. Familiar examples of this form of abscess are those associated with infectious inflammations of compound fractures, and other injuries. Phlegmonous erysipelas is a more severe form of this same type of inflammation. The diffuse suppurative inflammation following the infection of dissecting wounds, *"malignant edema,"* is the gravest form of the affection. In this type of inflammation the process is very rapid and intense, the tissues are quickly overwhelmed by the action of the poison, resulting in extensive death of the tissues, and sometimes causing a fatal termination from acute septicemia before the suppurative process has become established.

Circumscribed abscess is a term used to designate a form of abscess possessing defined limits. This is the most common form of abscess. It is the result of a suppurative inflammation, having limited or circumscribed boundaries, and is an opposite condition to diffuse abscess or purulent infiltration. A furuncle (boil) or an alveolar abscess are common examples of this form of the affection. Circumscribed abscess may be located in any part of the body, in any vascular tissue. It is also occasionally found in the ivory of the elephant's tusk (a non-vascular tissue) at considerable distances from the pulp, and completely encapsuled. Such conditions must have been the result of injury to the pulp of the tusk, as suppuration in a tissue without a vascular system is an impossibility. Professor Busch, of Berlin, exhibited several specimens of this character at the Ninth International Medical Congress, held at Washington, D. C., in 1887. He believed them to be the result of injury to the pulp, causing suppurative inflammation. The tusk of the elephant grows continually during the life of the animal; the odontoblasts must, therefore, be in a state of constant functional activity, which would explain the fact of the abscess cavities being removed to such distances from the pulp by growth of the tissue from its base. This would also explain the encapsulation of the pus by the formation of secondary dentine. In all of these specimens the pus was dried up, leaving the cavity empty.

The micro-organisms most commonly found in acute abscesses are the staphylococci and the streptococci. The pus found in cold abscesses often contains but few micro-organisms, and sometimes it seems to be entirely free from them.

The staphylococcus is more frequently found in circumscribed

abscesses, while the streptococcus is more prone to give rise to diffuse purulent infiltration. (Senn.)

The size of the abscess will be determined by the character of the primary cause of the inflammation, its location, the age, habit of life, and diathesis of the patient, and the condition of the tissues involved.

Tissues which have been debilitated by a previous inflammation, a contusion or other injury, have not the same resistive powers as healthy tissues; consequently abscesses developing in these locations reach much greater dimensions. In individuals whose vital powers have been impaired by old age, improper or insufficient food, the drink habit, mental anxiety, or some previous acute or chronic ailment, acute suppurative inflammation has a greater tendency to rapid extension than in healthy persons.

Acute Abscesses.—An acute abscess or hot abscess is the usual termination of an acute circumscribed, suppurative inflammation. Its most common location is the connective tissue. Its direct cause is infection from micro-organisms,—the staphylococcus most frequently. Its contents are the characteristic yellowish, cream-like pus and shreds of devitalized connective tissue. It runs a rapid course, reaching its maximum size in a few days after the first signs of inflammation have appeared.

The opening of the pulp-chamber of a devitalized tooth, which before had been impervious, or the plugging with a dressing or a filling of an open pulp-canal, either with or without a sinus, through the alveolus, is sometimes immediately followed by an acute phlegmonous inflammation, when strict antiseptic precautions have not been taken, or the caliber of the canals is so small as to preclude the possibility of rendering them aseptic. In such cases the inflammation rapidly extends to the surrounding connective tissue, producing septic cellulitis, which follows along the intermuscular septa, fascia, etc., with great swelling and tension, and accompanied with lymphangitis. The constitutional symptoms are marked by high temperature, rigors, followed by profuse sweating, and other symptoms of grave disturbance. Occasionally it may result in gangrene, and finally death of the patient. One case of this character, associated with a lower third molar as the starting point of the inflammation, came under the observation of the writer at Mercy Hospital.

Symptoms.—The local diagnostic signs or symptoms of acute abscess are, throbbing pain, increasing swelling, surface reddened, and sometimes glazed; fluctuation discovered by palpation, percussion, and pressure; tendency to point. Pointing always occurs at the location of least resistance. Constitutional symptoms are rigors, fever, loss of appetite, general malaise, and thirst. Under ordinary circumstances the diagnosis is simple, but occasionally it becomes more diffi-

cult on account of the modification of the symptoms, depending upon the primary cause of the suppurative inflammation, its location, and the character of the tissues involved.

Grave blunders have sometimes occurred through relying too implicitly upon one, or even all, of the symptoms, when so modified. Aneurisms have been opened under the belief that they were abscesses. An angeioma may likewise be mistaken for an abscess. To avoid such accidents, the exploring needle, or exploring syringe, should always be used in doubtful cases. Too much care cannot be exercised, as some of the ablest and most careful surgeons have had these unfortunate experiences.

Treatment.—*"Ubi pus ibi evacuo."* This rule is as wise to-day in the treatment of acute abscesses as it was centuries ago. Many surgeons have abandoned expectant treatment, and now cut down upon the abscess as soon as a sufficient quantity of pus has been formed to make the diagnosis clear. (Senn.) Much suffering may be saved by the adoption of this method of treatment.

In opening an abscess, the surface should always be first carefully cleansed, and other antiseptic precautions preserved, while the incision should be made at the most dependent part. In large abscesses, several small incisions, not over an inch in length, are better than one large one which lays open the entire cavity. Evacuate the pus and irrigate the cavity with some bland antiseptic solution,—boric acid or Thiersch solution,—until the fluid runs clear, after which insert one or more drainage-tubes, as the case may require, and dress with antiseptic gauze, oakum, or other sterilized material.

Antiseptic solutions.—The trend of treatment by antiseptic solutions to-day is toward those which do not coagulate the proteid elements. Corrosive sublimate has been considered as the most valuable of all the drugs for use in antiseptic surgery, and this idea has been most thoroughly instilled into the minds of medical and dental students. Koch, through whose experiments and statements the mercuric chlorid solutions received such a boom, exaggerated its antiseptic value, or overlooked the differences which must always be reckoned upon between laboratory experiment and clinical experience.

The coagulating property of the mercuric chlorid is undoubtedly a great hindrance to its practical usefulness, as it reduces its powers of penetration.

Sir Joseph Lister has recently announced that he has entirely abandoned the use of the sublimate solution in favor of carbolic acid. He says, "A 5 per cent. solution of carbolic acid is more trustworthy as a germicide, for surgical purposes, than corrosive sublimate, and in other respects greatly to be preferred.

"A great advantage of phénol seems to be that it has a powerful

affinity for the epidermis, penetrating deeply into its substances, and mixing with fatty materials in any proportion."

Some of our best surgeons have to-day discarded all antiseptic drugs, relying upon soap and sterilized water for cleansing the surfaces, sterilized water alone for all other purposes, and simple sterilized materials for dressings, and they claim as good results as when they depended upon antiseptic drugs.

This emphasizes the fact that it is better to prevent the ingress of pathogenic micro-organisms than to attempt to destroy them after they have gained entrance to the tissues; also that surgical cleanliness is more valuable than drugs.

In wounds, and in suppurative conditions of the oral cavity, antiseptic solutions are indispensable, but solutions of mercuric chlorid and carbolic acid have no advantages over the boric acid or the Thiersch solutions, while they have the disadvantage of being irritating to the mucous membrane if used of sufficient strength to be of real value as germicides, as well as poisonous if by accident they enter the stomach.

Methods of Opening Abscesses.—Abscesses as large as a hen's egg will not generally require more than one incision.

In deep-seated abscesses, it is best to incise the skin and fascia, and then with a pair of sharp-pointed hemostatic forceps tunnel the tissue until the abscess is reached; then unlock the handles of the forceps and separate the blades sufficiently, so that on withdrawing the forceps an opening will be made large enough to admit a drainage-tube of the proper diameter. This method is especially valuable when the abscess is located in the neighborhood of important vessels and nerves, as it thereby greatly reduces the dangers of wounding them. It is the safest method to follow in opening large, deep-seated abscesses in the neck.

In the treatment of alveolar abscesses, the disease may be cut short by trephining the external alveolar plate at the point of suppuration. To prevent a recurrence, the pulp-canals must be rendered aseptic, and maintained in that condition by proper dressings, temporary or permanent fillings.

Dressings.—In the application of dressings to external abscesses, care should be taken to insure equable compression, that the surfaces of the abscess cavity may be kept in apposition. Where drainage-tubes are used, these should be shortened from time to time, and entirely removed as soon as suppuration has ceased.

Healing of Abscesses.—Abscess cavities, when aseptic, heal by granulation. Absolute rest of the part is a valuable adjunct to the treatment. Patients with large abscesses should be kept in bed.

The principal causes of retarded healing are imperfect drainage, non-apposition of granulation surfaces, hemorrhage, rupture of the

limiting walls, permitting infiltration of pus into the surrounding connective tissues, indolent granulations, or constitutional dyscrasia. In the latter conditions, general tonic treatment is indicated.

Chronic Abscess.—Chronic abscess differs from the acute form in that its course is slow, the signs or symptoms are greatly modified, or wanting altogether. It is usually painless, not tender to the touch, and causes little or no febrile disturbances. The tendency to point is less marked, and pus accumulates often to an extraordinary amount before the skin shows any sign of yielding. Generally the pus-corpuscles are considerably disintegrated, the abscess walls are greatly thickened, and show signs of organization into connective tissue, and with very little tendency toward healthy granulation.

The condition is one of passive congestion, with slight development of inflammatory heat; hence the terms, congestive abscess and cold abscess.

Causes.—Chronic abscess may generally be traced to some specific chronic inflammation, most often of a tubercular nature. The forms most frequently coming under the observation of the oral surgeon are alveolar abscesses, caused by specific infection from devitalized teeth, abscesses of the face and neck from chronic inflammation of unerupted third molars, or portions of necrosed bone, tubercular or syphilitic inflammation of the jaws, tubercular inflammation of the cervical glands, and retropharyngeal abscess. Abscesses in connection with unerupted third molars and the cervical glands often burrow downward, following the septa between the muscles, and point as low down, sometimes, as the clavicle and mammæ.

When large chronic abscesses rupture spontaneously, or are opened with the bistoury, profuse suppuration and hectic fever quickly develop, frequently preceded by rigors, and followed by profuse sweating. Occasionally, under such conditions, emaciation is rapid and continuous, and the patient dies from septic infection.

Symptoms.—The diagnosis of chronic abscess depends more upon the careful consideration of the symptoms of the local lesion from which it started than upon the location, size, and special features of the swelling. (Senn.) Tubercular affections are usually accompanied by such well-marked symptoms at the stage when abscesses form, that there is very little difficulty in locating the primary lesion. The same is true of chronic abscesses originating from unerupted third molars.

An exploratory puncture, and a microscopic examination of the contents of a chronic abscess, will many times be necessary to a positive diagnosis as to its character.

In tuberculosis, the product of tissue proliferation coagulates, dies, and disintegrates into a granular mass, which when mixed with serum in sufficient quantity forms an emulsion that to the unaided eye closely

resembles pus, but which the microscope proves to contain none of the histologic elements found in pus.

Secondary infection of tubercular, actinomycotic, or syphilitic lesions may take place from the localization of the pus-microbe, and true chronic abscess result, or occasionally be followed by an acute phlegmonous inflammation. Generally, however, no acute symptoms develop.

Treatment.—Surgical interference in the treatment of chronic abscess is never so urgent as in the acute form, on account of its slower development and slight constitutional disturbance.

The abscesses appear months, and sometimes years, after the first development of the primary cause.

It has already been stated that acute abscess should never be opened without antiseptic precautions. This principle needs to be doubly emphasized in the treatment of chronic abscess, especially that form known as cold abscess.

The antiseptic precautions in the latter form should be of the most rigid and elaborate character, in order to guard against the dangers from septic infection, and a possible fatal result.

To avoid these dangers, the German surgeons advocate evacuation by aspiration, and iodoform injections, in preference to incision and drainage. Aspiration in tubercular abscess is generally unsatisfactory, for the reason that the needle or trocar soon becomes clogged by the shreds of dead tissue, and renders complete evacuation impossible.

In those cases where the seat of the primary lesion can be reached by an incision of the abscess, this is the proper method, as it gives opportunity to remove the infected tissue or the cause of infection. The abscess cavity should then be thoroughly scraped out (curetted), and all infected tissue removed, cleansed with antiseptic solutions, dried, covered with iodoform or boric acid, and treated as a recent wound, by suturing, drainage, and antiseptic dressing.

In those cases caused by devitalized teeth, unerupted teeth, or necrosed bone, rational treatment would demand the immediate removal of the cause of irritation as soon as the diagnosis could be made clear. Constitutional treatment comprehends a generous diet, stimulants, and tonics,—iron, cod-liver oil, etc.

REVIEW.

CHAPTER VII.

What is an abscess?
What does the prefix pyo denote when coupled with pleura, etc.?
What form of inflammation always precedes the formation of an abscess?
How is an abscess formed?
What term is applied to the natural opening of an abscess?
What process is going on at the same time in the outer portion of the wall of leucocytes?
What lines the abscess cavity, and what is the function of this tissue?
Of what is this tissue composed?
How are abscesses classed?
What is the tendency of superficial abscess?
In what kind of tissue is it usually found?
How can the tendency to spread be explained?
What is the tendency of deep-seated abscess?
What are some of the causes associated with the mouth that are productive of this form of abscess?
Under what other conditions are these abscesses found in the neck?
How far down do they sometimes burrow?
What causes operate to produce this condition?
What form of abscess represents a deeper location?
Where is retropharyngeal abscess located?
What are the anatomical relations of the parts?
What is the character of the early symptoms?
In what location do these abscesses point?
What is the cause of retropharyngeal abscess?
Name the terms applied to diffuse abscess.
What is its tendency?
In what tissues is it usually located?
What is the cause of this form of abscess?
By what routes does it usually spread?
What is the character of the local and constitutional symptoms?
What are the affections in which it is most frequently seen?
Give an example of a more severe form of this type of inflammation.
What is the gravest form of this type of inflammation?
From what is it caused?
What is the character of the symptoms?
What are its tendencies?
What is a circumscribed abscess?
Give an example.
Where may this form of abscess be located?
Is it ever found in non-vascular tissues?
What is the explanation of its presence in the elephant's tusk?
What species of micro-organisms is most frequently found in circumscribed abscesses?
Which species is most prone to give rise to diffuse purulent infiltration?
What conditions determine the size of the abscess?
Describe an acute abscess.
What is its most common location?

What is the character of its contents?

What are the diagnostic signs of acute abscess?

What precautions should be taken in diagnosing doubtful cases?

What general principle should be followed in the treatment of acute abscess?

What precautions should be observed in opening acute abscess?

Where should the incision be made in opening the abscess, and why?

What treatment should follow the evacuation of the pus?

What antiseptic solutions are considered best?

What are the objections to corrosive sublimate?

What advantage has phénol over sublimate solutions?

Are antiseptic drugs used by all surgeons?

What does the discarding of antiseptics imply?

What class of antiseptics are most desirable to use in the mouth?

Are antiseptics necessary in wounds of the mouth, and why?

What operation will shorten the duration of an alveolar abscess?

How prevent a recurrence of the abscess?

How does a chronic abscess differ from the acute form?

What are the causes of chronic abscesses?

What forms are most commonly found in the region of the face and mouth?

What are the methods of diagnosis?

How does the surgical treatment differ from that followed in acute abscesses?

What should be the constitutional treatment?

CHAPTER VIII.

ULCERATION.

Definition.—*Ulceration.* (Lat. *ulcus*, a sore.)

An ulcer is an open sore; a destructive loss or solution of continuity upon any of the free surfaces of the body, which will not permit of repair by primary union; a molecular death of tissue.

It owes its existence to an excess in action of the retrograde changes over those of repair.

The difference between an open granulating wound and an ulcer is that the wound shows a tendency to heal, while the ulcer shows no such tendency, but on the contrary is often inclined to spread. The explanation is that in the granulating wound the primary cause has ceased to exist, while in the ulcer it is still persistent, or infection has been introduced. An open granulating wound may become an ulcer at any time if the granulation-tissue takes on a retrogressive change. This change may be induced by infection with the pus-microbe, from mechanical or chemical irritation, from dressings, the presence of a foreign substance, or the chemical action of drugs applied to the wound.

Ulceration and gangrene are closely allied to each other, the difference being that ulceration is the death of cells, the fixed tissue-cells and the embryonic or new-formed cells ("cell necrosis"), while gangrene is death *en masse* of tissue (formed tissue).

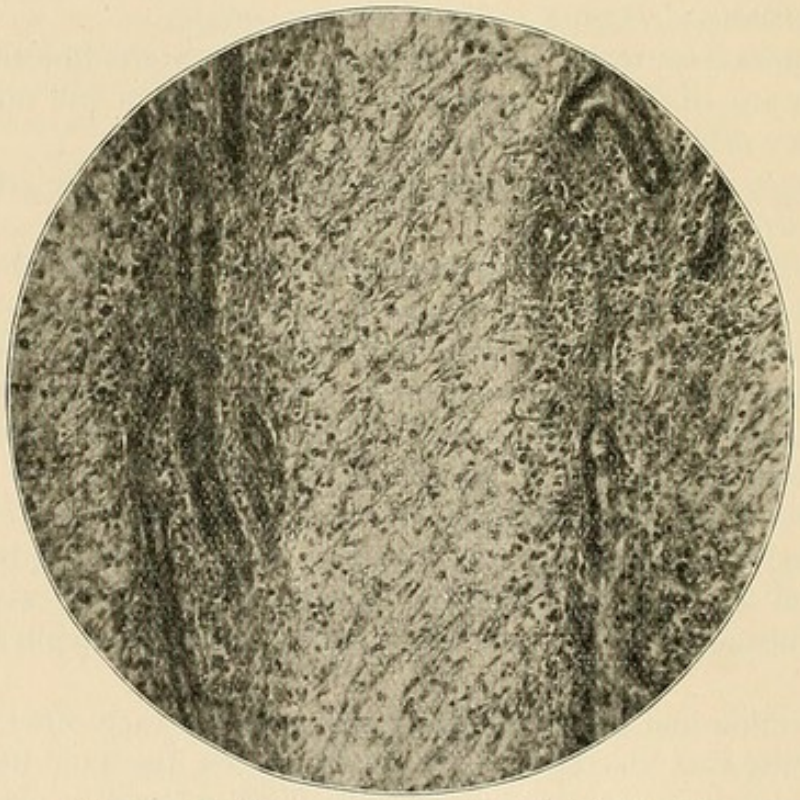
All wounds, of whatever nature, which do not heal by primary union or "first intention," heal by the process of granulation. (Fig. 34.) A granulation is composed of a capillary loop about which are clustered a number of living leucocytes, held together by a delicate intercellular material. Healthy granulations are cherry red in color, non-sensitive, elastic, and discharge a laudable pus.

An ulcer is quite indefinite as to its size, and variable as to its shape. It is usually round, but may be reniform, irregular, or serpiginous. It may be deep or shallow, with abrupt or with sloping sides, and a smooth or an irregular base. Its edges may be sharp or round, everted or undermined. The surface is covered with coarse granulations, dark red in color, which bleed readily. The surface may be

clean or sloughy, and covered with pus or serum. On healing, it always leaves a scar.

A vertical section of an ulcer examined microscopically reveals the following conditions: First, a layer of pus upon the surface; projecting into the pus fine capillary loops, surrounded by living leucocytes, constituting granulations. Beneath this is a zone of thickened inflammatory tissue, consisting mainly of fine fibrous tissue, and underneath this again a zone of hyperemia, where the capillaries are very numerous, and the leucocytes are in excess. Beyond this are healthy tissues.

FIG. 34.



GRANULATION-TISSUE—BLOOD-VESSELS AND MATRIX. × 75.

The ulcerative process is so intimately associated with inflammation, suppuration, gangrene, phagedena, granulation, and cicatrization, that it is impossible to detach it from any one of these and call it a separate and definite process.

Causes.—The causes of ulceration may be divided into *constitutional* and *local*, *predisposing* and *exciting*.

The predisposing causes are those which operate through the general system, and comprehend changes in nutrition, in the quantity and quality of the blood, peculiar dyscrasiæ and diatheses, and the freedom and rapidity of the circulation. Familiar examples are seen in persons who are badly nourished or debilitated by disease, such as tubercular affections, intestinal disorders accompanied by exhausting

discharges, typhoid fever, diabetes, scurvy, syphilis, mercurial poisoning, and dropsy.

Age.—The influence of age is often stated to be an important factor in the etiology of ulcers. Old age is without doubt a period marked by many retrogressive tissue-changes, and by diminished physical power and vital resistance, which predispose to and favor the ulcerative process, and yet statistics show only a very slight increase in the percentage of this affection among the aged.

Sex.—Sex seems to be a potent factor in the determination of ulcers. Statistics show that ulcers are three times as prevalent among men as among women. This may be explained, however, by the fact that men are subject to much greater exposure to injuries, and are more liable to contract syphilis or the habit of intemperance.

Occupation.—Occupation owes its influence to the degree of exposure to traumatism. The greatest number of individuals suffering from ulcers come from the laboring classes. Among the most prominent predisposing causes of ulcers are neglect and filth. It therefore happens that a greater number of cases of ulceration are found among that class of individuals whose habits of personal cleanliness are not good, whose means or lack of means prevents the proper care of the lesion in its earlier stages when it might be easily cured.

Traumatism.—Traumatisms are the most frequent cause of ulcers of the skin and mucous membrane, at least of the acute variety. The degree of injury necessary to produce an ulcer will depend upon the individual peculiarities of the constitution. The young and the robust adult will resist an injury which in the aged might result in extensive death of tissue. In the feeble and those afflicted with some constitutional dyscrasia like tuberculosis, syphilis, gout, diabetes, etc., slight injury often causes death of tissue, with sloughing and the formation of troublesome ulcers.

Classification.—Ulcers are usually classed according to their mode of origin, and are divided into two groups, the *non-infectious* and the *infectious*.

Among the non-infectious ulcers may be classed all those which are caused by friction, pressure, and other mechanical injuries, and those which arise from chemical irritation or from trophic changes due to enervation, general faulty nutrition, and impeded local circulation. The great majority of ulcers are the result of the action of various forms of infectious micro-organisms, such as the pyogenic bacteria, those of tuberculosis, syphilis, leprosy, glanders, and perhaps cancer. Ulcers which are not caused primarily by the action of pathogenic bacteria usually become infected as soon as an open wound is formed, by the bacteria invading the exposed surfaces, and establishing the inflammatory process.

Ulcers are also classified according to certain changes, complications or modifications which may occur in them. These changes, etc., are indicated in the terms applied in the classification, such as *inflamed, erethistic* (irritable), *fungous, hemorrhagic, torpid, callous, corroding, perforating, phagedenic*, and *malignant* ulcers.

An *inflamed* ulcer is one having its base and surrounding parts in a state of more or less acute inflammation; the surface is very red; it bleeds easily, and the formation of pus is plentiful. The edges of the ulcer are swollen and raised, the surrounding skin is exceedingly tender, dense and shining. Ulcers of this character are often very painful. The causes which produce these conditions are neglect, contact with acrid secretions, or the application of substances of an irritating character.

The *erethistic* or irritable ulcer possesses extreme sensitiveness which is exceedingly difficult to relieve. It is most often located in parts which are highly sensitive, like the anus. These ulcers have the appearance of granulating surfaces in which the active process of repair has been arrested. The edges are abrupt, and show no evidence of a tendency to cicatrize; they are exceedingly tender, the slightest touch causing intense pain. The cause of the exceeding sensitiveness has been thought to be the thinness of the granulation-tissue. It is more often associated with nutritional changes due to anemia resulting from the loss of blood or from severe disease. Upon a re-establishment of the normal conditions of nutrition, the exalted sensibility passes away.

The *fungous* ulcer is the result of an exuberant growth of granulation-tissue. It is caused by an over-supply of blood to the part, which results in the rapid growth of capillary loops from the pre-existing blood-vessels, while the epithelial cells present a sluggish kinetic function, which retards the process of repair. Fungous granulations in a wound, or an ulcerating surface, are popularly known as "proud flesh." Such a condition is an obstacle to the process of repair. Fungous granulations are frequently seen protruding from the external opening of fistulous tracts, especially those leading to tubercular abscesses, necrosed bone, or other foreign body. Occasionally the granulations will be so large as to protrude beyond the surrounding surface of the skin, sometimes presenting a mushroom appearance, with a narrow pedicle. This condition is due to the growth of the epidermis into the granulation, and the presence of a large arteriole at the base of the granulation-tissue. If the granulations are cut off, they are reproduced before the sluggish epithelium incloses the wound. Conditions of this character are frequently associated with chronic alveolar abscesses associated with pulpless teeth.

The *hemorrhagic* ulcer is one which bleeds upon the least provocation. It is most frequently seen in scurvy. The ulcerating surface has a characteristic livid blue color, and the granulations possess an active tendency toward disintegration. Vicarious hemorrhage has been observed in cases in which there has been an arrest of the bleeding from hemorrhoids, and following a suppression of the menses.

Torpid ulcers are those which show no active tendencies in any direction. They are seen most often in individuals suffering from the debilitating effects of acute or chronic disease, resulting in defective nutrition and impaired or diminished blood-supply to the affected part. The characteristic color of the granulation is pale red, and the pus which is formed is thin and watery.

The *callous* ulcer presents a dirty, granulating surface, with thin, muco-purulent pus, and edges raised considerably above the surface. The skin is indurated and fixed for some distance around the ulcer. This form of ulcer exists without material change in size for a long time, and it is most often seen in connection with old varicose ulcers of the legs.

Corroding ulcer is one which causes a progressive destruction of the soft tissues, usually starting in the form of a cutaneous affection which assumes the form of a boil—Delhi boil—and afterward ulcerates, causing considerable loss of tissue. Ulcers associated with lupus may also be classed with this form, as it shows a slow but constant tendency to spread to adjacent cutaneous tissue.

Perforating ulcer (round ulcer) is an ulcerative condition of the stomach, usually dependent upon local obstruction of the blood-vessels of that organ. Its most common location is the posterior wall of the pyloric portion of the stomach. The perforation through the serous coat of the stomach wall has the appearance of having been punched out or cut out, and is usually round.

Phagedenic ulcer is one which spreads rapidly, causing considerable loss of tissue, and accompanied by great local irritation. It usually occurs in persons who are broken down by disease, lack of proper nourishment, or debauchery, and is seen most often in epidemics of hospital gangrene, in ulcers which have been treated by irritating or escharotic substances, and in mercurial ptyalism. The primary syphilitic lesion sometimes takes on a phagedenic form, when it becomes very obstinate to the effect of remedial agents.

Malignant ulcers are those which run a rapid course, spread in all directions, perforating the soft parts, and causing extensive gangrene and sloughing, with necrosis of bone. The constitutional symptoms which accompany this type of ulcer are often profound, and not infrequently terminate fatally. The class of individuals in whom this form of ulcer is most commonly found are children who have suffered from

long and exhausting illness, or whose surroundings are unhealthy, and the food scanty and unwholesome. The parts most frequently attacked are the lips, cheeks, and gums. Noma or gangrena oris is an example of this type of ulcer. It is also associated with epithelioma and with carcinoma in other portions of the body.

The *exciting* causes are irritations of a physical, chemical, or septic nature.

The forms of ulcers most interesting to the oral surgeon, and which most frequently come under his observation, are simple follicular ulceration of the mucous membrane, aphthæ, syphilitic mucous patches, deep syphilitic ulcerations, sloughing phagedena following mercurial ptyalism and scurvy, cancrum oris, gangrena oris, ulceration associated with cancerous growths, and lupus.

Healing.—The process of healing in ulceration is by granulation and cicatrization. During the process of healing the dead parts of the ulcer come away as a thin, ichorous discharge, the exudates beneath and around become vascularized, and capillary loops shoot up toward the surface. Large numbers of leucocytes cluster around these, forming a surface of healthy granulation, which then discharge a thin, creamy pus, the laudable pus of the old writers.

Cicatrization is the process of covering or skinning over the new tissue formed by granulation. During this process the surrounding surface of the skin sinks to the level of the granulation, the epithelial cells at the edge of the ulcer undergo segmentation or karyokinesis, and grow toward the center of the ulcer. This is denoted by a blue film, and while this is extending the new tissue of the ulcer is contracting from the conversion of the leucocytes into fibrous tissue. Contraction does not stop with the healing of the ulcer, but continues for a considerable period afterward, and sometimes causes very great deformity. A cicatrix has neither nerves, glands, lymphatics, nor hair, and when injured it does not heal readily, and is prone to ulceration.

Prognosis.—The prognosis of an ulcer depends upon several factors, viz: The nature and primary cause, the situation, the age and constitution of the patient, and the complications. The complication most likely to occur is septic infection. Like open wounds, ulcers are constantly exposed to this danger, and when the fact of neglect and filthiness, as often seen in the out-patients who visit the infirmaries in our large cities, is taken into consideration, it seems wonderful that more cases are not complicated with some form of septic poisoning. The erysipelatous streptococcus is a frequent cause of septic infection of ulcers, and is most commonly seen among ignorant individuals who are filthy in their habits. Cases of this character may have progressed to such an extent before they are seen by the surgeon as to render treatment of no avail, and a fatal termination is quickly reached.

Adenitis resulting in extensive suppuration of the glands from sepsis is another form of complication which may terminate in death from hectic fever, exhaustion, and amyloid degeneration of the liver and kidneys. (Minot.)

The most important and dangerous complication is perforation of some important internal viscus, like the stomach, intestines, or bladder, which establishes septic inflammation of the peritoneal cavity; or malignant and syphilitic ulcers, which perforate the cheek or palate and nasal septum. Peritonitis is the inevitable consequence of perforation of an internal viscus in those cases in which the ulcerative process has been rapid. When the ulceration progresses more slowly, nature attempts to prevent perforation by establishing a plastic inflammation and thickening of the outer wall of the viscus at the base of the ulcer, and also of the peritoneum lining the abdominal walls, or covering some adjacent organ, resulting in adhesion of the surface in contact with it, and perforation of the wall of the adherent organ without entering the peritoneal cavity.

Hemorrhage is sometimes a serious complication, calling for ligation of the arterial trunk supplying the locality of the ulcer. This is most likely to occur in malignant ulcers, ulceration associated with malignant tumors, and in ulceration of varicose veins of the leg. The severity of the hemorrhage may be such as to endanger life. Hemorrhages occurring in ulcers upon the surface of internal organs are often fatal from their inaccessibility to surgical treatment.

Ordinary ulcers of the skin and mucous membrane of traumatic or idiopathic origin are never dangerous to life except through septic infection. The curability of an ulcer will depend largely upon the nutrition of the part, the character of the treatment, and the amount of care exercised in carrying out its details.

Treatment. —The treatment of ulcers in general must be directed both to the local and constitutional conditions. The local treatment consists primarily in improving the circulation of the part. Passive hyperemia usually exists, and it is necessary to relieve this condition in order that the parts may regain their normal function, and thus be enabled to carry out the process of repair. (Warren.)

Ulcers as they come under the observation of the surgeon are usually more or less in a state of inflammation from the presence of micro-organisms or other irritating substances which prevent the establishment of the healing process. Rational treatment would therefore be directed first to the removal of the cause of irritation, and the adoption of means which would relieve the inflammatory symptoms. These ends may be accomplished by rest of the part, cleanliness, antiseptic compresses saturated with hot antiseptic solutions, antiseptic dressings, and, if the disease is located in an extremity, ele-

vation of the limb to a higher level than the rest of the body, as this favors relief of the hyperemic condition of the part. Rest in bed is always advisable in any case of serious ulceration. Care must be exercised, when using moist compresses and dressings, that the tissues are not injured by too long an application at one time; twenty-four to thirty-six hours is as long as the tissues should be subjected to this kind of treatment without intermission. In addition to the above treatment, accumulations of pus in the cellular tissue or adjacent glands should be evacuated by free incisions and the establishment of ample drainage. If erysipelas is present it is best to treat this condition by the use of wet compresses wrung out of hot bichlorid solution of the strength of one to one thousand, or one to two thousand, in water.

In a majority of cases an ordinary ulcer will begin to heal as soon as the inflammatory symptoms have subsided. Indolent ulcers may be stimulated by the application of balsam of Peru, nitrate of silver, alum, permanganate of potash, sulfate of copper or zinc, chromic acid, iodin, boric acid, and ichthyol. Numerous drugs have been recommended for their stimulating qualities, but the above mentioned are sufficient to indicate this class of remedies.

Balsam of Peru is an excellent remedy and one of the oldest for stimulating the growth of granulations in that class of external chronic ulcers and granulating wounds in poorly nourished individuals in which the process of healing has been established, but progresses very slowly.

Nitrate of silver is used in solution of 5 to 10 grains to the fluidounce of water, as a stimulating application to ulcers of the mucous membrane. It is also used in stick form, freely applied, for the purpose of destroying unhealthy granulations. To stimulate the base of the ulcer it may be lightly applied so as to leave only the slightest evidence of an eschar.

Alum applied in powder or solution acts through its irritating and astringent properties.

Permanganate of potash, in solution of from 5 to 10 grains to the fluidounce, is an excellent stimulant to chronic ulcers, but sometimes these strengths will prove painful, and weaker solutions must be used. Solutions of copper and zinc sulfate of about the same strength often prove themselves to be valuable remedies in the same class of cases.

Chromic acid is of value in secondary syphilitic ulcers of the mucous membrane, especially of the mouth, and in noma. A solution of 10 grains to the fluidounce of water will sometimes cause the former to heal without the aid of constitutional treatment. In noma it is claimed to be beneficial by destroying the diseased tissue, and thus giving opportunity for the formation of healthy granulations, but in the writer's experience nothing has proved of permanent benefit in cases of true noma.

Iodin and iodoform are the best remedies in tubercular ulcers.

Boric acid has a wide range of usefulness in the treatment of all forms of ulcers, being antiseptic as well as stimulating.

Ichthyol was introduced to the profession by Unna as a stimulant to the growth of epithelium, but it is also a valuable aid to the growth of granulations. It may be employed in a 10 per cent. solution, but it is most commonly applied in the form of an ointment of a 25 per cent. strength.

Operative Treatment.—Skin-grafting and plastic flap operations are sometimes employed to close extensive open wounds and ulcers, caused by loss of tissue from surgical operations and other traumatisms, particularly those following burns and scalds. The Thiersch method of skin-grafting is generally considered the best, and consists of cutting thin shavings of healthy skin from an arm or a leg, and applying them to the surface of the ulcer or granulating wound, which has been previously prepared to receive them by removing the granulations and arresting the hemorrhage. The grafts are then gently pressed down and held in position by cotton dressing saturated with a salt solution, and protected by rubber tissue and a bandage. The dressing is changed in twenty-four hours, and renewed as often as the case requires. Antiseptic solutions are not used, but the dressings are kept moist with a sterilized salt solution. Large surfaces are frequently covered at a single sitting by this method.

Plastic flap operations are sometimes utilized to cover granulations of moderate size, and to fill gaps caused by the loss of tissue in the removal of tumors. These flaps are taken from adjacent tissue, but always in such a way as to leave a broad pedicle through which the flap is nourished until such time as union takes place between the flap and the granulating surface. The flap is stitched to the edges of the ulcer after they have been freshened, and it is then treated as a recent wound.

Sponge-grafting is another method of hastening the process of healing of ulcers and granulating wounds. The sponge is prepared by first decalcifying it by soaking in nitro-hydrochloric acid, and then washing it in an alkaline solution to remove the acid and preserving it in a one to twenty solution of carbolic acid. When applied to the granulating surface it is first cut into a thin piece of the exact shape of the opening to be filled, and after careful disinfection of the ulcer and its surroundings it is placed in position, and covered with antiseptic dressings, which will require frequent renewal on account of the decomposition which usually occurs. After a few days the granulations will spring up and fill the interstices of the sponge, finally reaching the surface, and the sponge will be buried out of sight; the process of covering with epidermis will then begin. The sponge is

afterward absorbed. The late Dr. William H. Atkinson, of New York, Brock, of St. Louis, and others, made extensive use of this method of healing ulcers of the mouth, and reproducing tissue lost by accident or surgical operations.

Constitutional Treatment.—The constitutional treatment of ulcers must be directed to the systemic condition which operated as the predisposing cause of the affection. It may not be possible in all cases to determine or discover the evidence of a general disorder, or taint of the system, but, when such a taint can be detected, appropriate remedies must be administered for its eradication. Syphilis will require appropriate specific treatment accompanied with tonics. Tuberculosis will require rest, change of climate, and remedies which will build up and support the strength of the patient. Anemia and diabetes are frequently predisposing causes of ulcer, and should not be overlooked when searching for a constitutional cause for the ulceration. Gouty affections are often associated with vascular changes and ulceration of the skin and pericemental tissue. This condition should be sought for, and, if present, measures must be taken to improve the general condition, such as vegetable diet, abstinence from wines and malt liquors, etc. In many cases of chronic ulcers, no constitutional predisposing or other cause can be discovered, and nothing remains in the line of constitutional treatment beyond the observance of simple hygienic rules of life, the administration of tonics, and change of surroundings.

REVIEW.

CHAPTER VIII.

What is an ulcer?

To what does it owe its existence?

What is the difference between a granulating wound and an ulcer?

How is this difference explained?

Under what conditions may a granulating wound become an ulcer?

What is the difference between ulceration and gangrene?

By what process do wounds heal which have failed to unite by first intention?

Of what is a granulation composed?

What are the characteristics of healthy granulations?

What are the characteristics of an ulcer as to its size, its edges, its base, and its surface?

What is the character of the tissue left by healing?

What are the pathologic conditions of an ulcer as revealed by the microscope?

With what other conditions is the ulcerative process intimately connected?

How are the causes divided?

What are the predisposing causes?
What are the exciting or active causes?
What influence does age exercise in the etiology of ulcers?
How does old age predispose to ulceration?
What influence does sex exercise in their production?
What is the difference in ratio?
How is this difference explained?
What is the influence of occupation in the production of ulceration?
What influence have neglect and filth in their production?
Name the most frequent cause of acute ulceration.
What constitutional conditions predispose to ulceration?
How are ulcers classified?
Into what general forms are they divided?
What varieties are classed as non-infectious?
What as infectious?
Which form is the most common?
What is the usual condition of exposed ulcerated surfaces?
How is the inflammatory process established?
What other conditions in the character of ulcers form a basis of classification?
Name the varieties.
What is an inflamed ulcer?
Describe its appearance.
What is an erethistic ulcer?
Where is it most frequently located?
Describe its appearance.
What causes the hyper-sensitiveness?
With what constitutional conditions is it often associated?
What is a fungous ulcer?
How are the fungous granulations produced?
What retards the process of healing?
Of what are fungous granulations an evidence when found protruding from a fistulous tract?
From what is the mushroom-like granulation due?
What is hemorrhagic ulcer?
How may it be distinguished?
Under what conditions has it been observed to possess a vicarious function?
Describe a torpid ulcer.
In what class of individuals is this form most common?
How may a callous ulcer be distinguished?
In what connection is it most often seen?
Describe a corroding ulcer.
With what other condition is it sometimes associated?
What is a perforating ulcer?
Where is it most commonly located?
What is the appearance of the ulcer?
What is a phagedenic ulcer?
In what class of individuals, and under what circumstances, is it most commonly seen?
What class of syphilitic lesions sometimes take on this form of ulceration?
What is a malignant ulcer?
What is the character of the constitutional symptoms?

How does it frequently terminate?
In whom is it most commonly found?
Under what conditions?
Where is it most frequently located?
Give an example of this form of ulcer.
With what form of tumors is it sometimes associated?
Name the forms of ulceration most common in the mouth.
How do ulcers heal?
What term is applied to the tissue formed by granulation?
What anatomical elements are not found in cicatricial tissue?
Upon what does the prognosis of ulcers depend?
What is the most common complication?
What forms of septic infection sometimes prove fatal?
What is the most important and dangerous complication?
In perforation of an internal viscus, what is the result?
How does nature sometimes prevent perforation into the peritoneal cavity?
In what form of ulceration may dangerous hemorrhage occur?
Under what circumstances might it prove fatal?
What is the prognosis of ordinary ulcers of the skin and mucous membrane?
Upon what will the curability of an ulcer largely depend?
To what conditions must the treatment be directed?
What special condition must first be relieved in order to establish the process of repair?
What is the usual condition of ulcers, and what are the causes?
How may these conditions be removed?
What is the danger of long-continued moist dressings?
When suppuration is present, what is the treatment?
When complicated with erysipelas, what is the treatment?
Name the remedies used to stimulate the healing process of ulcers.
In what class of cases is balsam of Peru advantageous?
In what form of ulcer is nitrate of silver most valuable, and how is it applied?
Through what properties does alum produce beneficial effects?
In what form of ulcers is the permanganate of potash useful, and in what strength?
Sulfates of copper and of zinc are useful in what class of cases, and in what strength may they be applied?
Under what circumstances and in what strength is chromic acid used?
In what form of ulcer are iodin and iodoform used?
When is boric acid useful?
What is the value of ichthyol?
How may it be employed?
What operative measures are used to cover ulcerated surfaces?
Describe the Thiersch method of skin-grafting.
How is the flap operation made?
What is the after-treatment?
Describe the method of sponge-grafting.
What is the line of constitutional treatment in ulcers?
How should a syphilitic condition be treated?
What is the treatment in tubercular disease?
When no constitutional taint can be discovered, what treatment should be followed to build up the system?

CHAPTER IX.

NECROSIS, CARIES, AND GANGRENE.

Necrosis, Caries, and Gangrene are conditions, not diseases.

These forms of death occurring in tissues and organs, and in cells and cell groups, are termed local death, or necrosis, in contradistinction to somatic death or death of the whole organism. The causes which lead to local death of tissues may be divided into three groups. The first includes those which destroy the tissues by mechanical or chemical action; for instance, external violence, which may crush a finger or a toe; sulfuric acid or caustic potash, which may destroy a patch of skin; or micro-organisms, which may disorganize the structure of a gland to which they have gained access. The second group of injuries may be classed as thermal, and are dependent upon high or low degrees of temperature. If the temperature of the tissue be raised to 130° F., or 140° F., and maintained at that degree for any length of time, death of the tissue is the inevitable result. Higher temperatures act still more rapidly. The lower limit within which the life of tissues may be maintained for any considerable period is 60° to 64° F. (Ziegler.) The third group are those which arise from arrestation of the circulation and nutrient functions. All conditions which seriously interfere with the circulation so as to bring about stasis, such as inflammation, hemorrhage, extravasation, pressure upon the tissues, thrombosis, embolism, or closure of the vessels by disease or ligation, may lead to death of the affected tissue. It is possible for all three of these groups to act together or successively.

The effect upon the tissues of a given injury in producing local death will depend upon the condition of the tissues at the time of the injury, their power of resistance, and the condition of the general organism. Tissues which have been the seat of a pre-existing inflammation, or any other condition which has lowered their powers of vital resistance, succumb more readily to injuries which produce necrosis, caries, and gangrene than normal tissues. When through disease the vital powers of the general system have been reduced, slight injuries will often cause serious consequences. In persons suffering from uncompensated valvular disease of the heart, slight injury may induce gangrene of the limbs. In patients emaciated from

typhoid fever, slight pressure upon the skin over the trochanter, elbow, sacrum, or heels may induce mortification of the skin and subcutaneous tissues.

It is interesting to note the time required to produce death in the various tissues of the body by the arrestation of the blood-current. The period varies in different tissues. Brain-tissue, renal epithelium, and intestinal epithelium die in two hours. Skin, bone, and connective tissue continue to live over twelve hours. (Cohnheim.) Tissues which exercise special functions die more quickly than those which do not exercise such functions. These facts should govern all operations for the transplantation or replantation of tissues. Success is more likely to crown the effort in transplanting and replanting of teeth if the operation is completed within an hour or two after the extraction of the tooth.

Necrosis.

Definition.—*Necrosis* (from the Greek νεκρός, dead).

Necrosis is death *en masse* of bone-tissue.

The term necrosis signifies the condition of death. When used in its technical sense, it refers to the process of death, or "the sum of the actions which terminate in the death of a portion of the skeleton, osseous or cartilaginous." (Markoe.) It is evident, therefore, that the term does not stand for a particular disease, but for a condition or result following many forms of disease associated with the bone. It has become common practice, however, with surgeons and pathologists to use the name applied to this condition or symptom as the one most convenient and best suited to designate the affection of which it is at most only an accidental consequence, and yet one that is very prone to occur as a result of certain injuries and diseased conditions affecting the bones or the tissues immediately connected with them. The degree of injury, whether traumatic or idiopathic, necessary to cause necrosis of bone, is much less than is required to produce death of the soft tissues. This may be explained by the fact that the external or superficial layers of the bone are supplied with blood through numberless small vessels given off from the deeper portions of the periosteum. Separation of the periosteum from the bone is very liable to occur, and may be brought about in various ways; for example, it may be stripped from the bone by violence, either accidental or surgical; it may be lifted from its attachment by effusion of blood, resulting from injury, or from the effusion of serum or other inflammatory product, especially pus. Another reason which may be advanced to account for the readiness with which bone-tissue succumbs to inflammatory processes is found in the compact tissue of the bone itself. "The Haversian canals are filled completely by the vessels which run

into them, and by the cell structures which are packed tightly around these vessels, so that when the inflammatory stimulus calls for larger blood-currents and more blood-cells, there is no room for the enlargement of vessels thus imperatively demanded, and the consequence is that the vessels, not having the power to accommodate themselves to this new and sudden demand, become choked, stasis occurs, and, as a consequence, those portions of the bone most deeply involved die." (Markoe.) Death of dentine occurs in the same manner by strangulation of the dental pulp. In speaking of local death of tissue, the devitalized portion of bone is called a *sequestrum*, the dead portion of soft tissue is called a *slough*. These considerations very naturally lead up to and suggest the causes of necrosis.

Causes.—Necrosis or death of bone-tissue is the result of conditions which have impaired or entirely arrested the supply of blood to the part, like traumatisms, inflammatory conditions resulting from syphilis, mercurial ptyalism, poisoning from phosphorus or other toxic substances, extensive inflammation from neighboring parts, or any other cause which produces a lowered vitality of the tissues. Death of the bone may be confined to small portions, or it may involve an entire bone. Fragments of bone which have lost their vascular connection may also become the seat of necrosis. Fractures, and periostitis the result of specific infection, or the constitutional effects of certain drugs, are the most frequent causes of necrosis.

The dead portion of bone, when not already separated from the living tissues, later becomes detached in consequence of the formation of granulation-tissue between the dead and living portions, and eventually the disintegration of this tissue leaves the necrosed portion separated. The process of separation is generally slow and tedious, and if the necrosed part is deeply situated its exfoliation may be long delayed. The structure of the dead portion is still preserved, so that there is no difficulty in its recognition. Suppuration is always present, and around the necrosed bone there is an accumulation of a fetid, purulent fluid, containing broken-down tissue, débris the result of decomposition and of the inflammatory process, which by degrees burrows the tissues, and eventually reaches the surface. A sequestrum of necrosed bone is porous, and somewhat lighter than living bone, owing to the fact that the organic elements have been removed from it by the process of decomposition and the action of the granulation-tissues which surrounded it.

The treatment of necrosis and caries will be considered in the chapter on Necrosis of the Jaws.

Caries.

Definition.—*Caries* (Lat. *caries*, rottenness).

A chronic inflammation of bone, with rarefaction or absorption of bone-tissue, followed usually by pus formation; molecular death of bone, with the accompanying process; sometimes termed ulceration of the bone.

The term caries is applied to a molecular death and disintegration of bone-tissue. The two forms of death of bone—necrosis and caries—are to the osseous tissue what gangrene and ulceration are to the soft tissues. Necrosis and caries of bone may be distinguished clinically from each other by the difference in the sound given off when percussed (the percussion note), and by the difference in the degree of density. Upon probing the opening in the external tissues of necrosed bone, it will be recognized by its hardness, and the sharp percussion note when struck with an instrument, while caries of bone will give a dull sound, and permit the penetration of the instrument into its structure.

Causes.—Caries of bone is a chronic inflammatory condition, induced almost always by tubercular or syphilitic infection. The tubercular form occurs most frequently at the epiphyseal ends of the long bones, in the bodies of the vertebræ, and occasionally in the bones of the face, the lower jaw most frequently. The syphilitic form is most commonly seen in the bones of the nose and palate. In the tubercular form the affection usually causes the formation of abscesses, which may burrow and open at some distance from the seat of a tubercular disease. Upon exposing the diseased portion of the bone it will be found to be softened, disintegrated, while portions have been removed by liquefaction or absorption, leaving a cavity of greater or less extent, the surface of which will be covered by granulation-tissue and exuding pus. If the process has been rapid, small spiculæ of necrosed bone will be found entangled in the granulations or mixed with the pus. In syphilitic caries, especially of the bones of the nose and palate, the external covering of the bone will usually have disappeared from ulceration, leaving a granulating surface, discharging the characteristic gummy pus.

Gangrene.

Definition.—*Gangrene* (Gr. *γάγγραινα*, a sore, and *γραίνειν*, to gnaw).

Mortification or death of a part of the body from failure in nutrition, death *en masse* of soft tissue. The putrefactive fermentation of a dead limb or tissue.

Gangrene presents itself in two forms. The first is designated as *dry* or *senile gangrene;* the other as *moist gangrene.*

One of the first changes which takes place in the tissues after the death of the part is the disappearance of the nuclei of the cells. In some cases the nuclei become granular, probably from the breaking up of the chromatin—the delicate reticular structure of the nuclei—or the material which is most susceptible to staining agents, and passes from the nucleus into the protoplasm or body of the cells, where it is dissolved and disappears. In other cases the nucleus itself seems to lose its susceptibility to the staining fluids, is dissolved, and disappears. These changes take place after closure of a vessel by embolism, and are readily seen in the epithelium of the kidneys under such circumstances. The color of the tissue of the organ is also changed to a pale, cloudy, yellowish-white appearance. (Warren.)

"Coagulation-necrosis" (Weigert) may sometimes take place, and the cellular elements be transformed into granular or hyaline masses, with loss of their nuclei. Hyaline degeneration (a conversion of tissue to a clear, transparent, jelly-like material) often attacks the intercellular structure of the tissues. This change is seen in muscle fiber when death has taken place from toxic infection, a burn, or other trauma.

Death of tissue accompanied by coagulation occurs in two ways. One kind occurs in certain of the vital fluids like the blood and lymph, and in fluids which have escaped from the vessels, by the formation of granular, fibrous, or homogeneous coagula. In the other, cells and intercellular structures, as they die, become solid and firm, and form by coalescence peculiar homogeneous or hyaline masses. (Ziegler.)

The coagulation of blood or of lymph, according to current opinion, occurs when the white blood-corpuscles die and are dissolved in the plasma, and the granular, fibrous, and hyaline masses which appear are albuminoid in character, and are designated in general terms as fibrin. To bring about coagulation,—in other words, the formation of fibrin,—the presence of fibrinoplastin (a native proteid obtained from the blood-serum) and a ferment is necessary. Both of these substances are supplied by the white blood-cells as they die and are dissolved in the plasma. Inflammatory exudates and effusions may coagulate in the same manner, forming masses which are rich in fibrin. False membranes are formed in this way upon the surfaces of inflamed tissue.

In the second form of death of tissues, the circumstances and appearances are essentially different from those of the first. Coagulation of the albuminoids has taken place in this form as in the other, but with this difference, that the coagulation has occurred in the substance of the formed tissue elements, the cells and intercellular substance, instead of in a fluid. It is necessary, however, in order to produce this form of coagulation, that a moderate amount of lymph shall flow through the dead portion of tissue. The lymph con-

tains fibrinogen, the cells fibrino-plastin, and by a combination of these substances fibrin is produced, resulting in coagulation-necrosis. Coagulation of the dead cells is not so likely to occur in tissues in which the process of death has been protracted, for the reason that degenerative changes may be established, like fatty degeneration, and thus render the cells non-coagulative. Similar cell changes to those which occur in coagulation-necrosis, and particularly the loss of the nuclei, may result from the process of putrefaction.

Causes.—Gangrene is so intimately associated with certain changes in the arterial system that it will be necessary to mention some of the conditions which are liable to produce it. Among the most frequent of these conditions is inflammation of the walls of the arteries. Arteritis is almost always followed by the production of new tissue within the walls of the vessel, and this holds an important relationship to the degree of freedom with which the blood circulates through the diseased vessels. In the aorta and large arteries, this new formation assumes the form of warty or pediculated projections, which in the smaller arteries may involve them for a considerable distance, and be so extensive as to more or less completely close the lumen or caliber of the vessel, and which is termed *"obliterating endarteritis"* (Warren), and thus greatly impede the flow of blood or arrest it altogether.

Atheromatous degeneration is a not infrequent termination of many cases of inflammation. It begins in this particular form by the formation upon the inner wall of the vessel of soft, gelatinous nodules, which after a time become quite hard, but later show signs of degeneration, and upon cutting them open they are found to contain masses of whitish or yellowish material. These masses finally soften, resulting in atheromatous ulcers, or, according to Orth, if the mass be seated deeper in the wall of the vessel, a cavity may be formed containing fatty granules, fragments of tissue, etc., and forming an *"atheromatous abscess,"* which may eventually discharge its contents into the interior of the vessel. Thrombi sometimes form at the location of these abscess cavities or open ulcers. In the smaller vessels, these ulcers may result in a complete obliteration of the lumen of the vessel. Calcification sometimes takes place in the atheromatous foci, and these masses, covered with epithelium, may be present in large numbers in the aorta and large arteries, or they may be found projecting from atheromatous ulcers. Syphilis and tubercle also produce changes in the walls of the arteries which impair their function of nutrition. The effect of these changes coming late in life must be very great upon the arterial circulation of the extremities, as there is naturally at this time an enfeeblement of the circulation in these localities. If the disease has been confined to the smaller vessels, the circulation has been gradually diminished, and finally ceases, as the result of the formation of a

small thrombus, or of some insignificant traumatism. The obliteration of the lumen of the artery cuts off all fluid from the dead part, and, as the disease has not been connected with the veins, they have been unobstructed, and have carried off the venous blood in the part; consequently the dead tissue becomes dried from evaporation, and the condition is produced which is known as dry or senile gangrene.

When death of a portion of the soft tissue takes place, there is always, as a constant result, a more or less severe inflammation in the surrounding part, which is most severe when decomposition has taken place in the dead portion. Decomposition is due to the action of the saprophytic germs. By means of this surrounding inflammation—the inflammatory zone—the dead portion is differentiated and isolated from the rest. The inflammation is therefore described as *definitive*, and the zone as the line of *demarkation*.

The final terminations of death of soft tissues are generally classed under four main types. In the first class, the dead tissue is thrown off or absorbed, and replaced by newly-formed tissue of the same character—normal tissue: this is termed healing by regeneration.

In the second the dead tissue is likewise thrown off or absorbed, but is not replaced by tissue of the same character, but by a fibrous tissue, which fills up the gap in part or in whole. This is termed healing by scar or cicatrix.

In the third, the dead tissue is only partially absorbed, a portion remaining as a caseous mass, which later may become calcified, and inclosed in a capsule of connective tissue. This is termed caseation and calcification.

In the fourth, the dead tissues are also absorbed, and in their place there is developed over the boundary of the vacated space a small amount of fibrous tissue. In other instances this space becomes filled with fluid, which is thus encysted. This is termed encystment. (Ziegler.) Examples of this not infrequently occur in connection with severe pericementitis and alveolar abscesses.

Dry Gangrene or Mummification.—This condition is usually the result of death of soft tissue in parts which are exposed to the air, and is caused by defective blood-supply from the general feebleness of the circulation, and local changes in the vessels themselves. Examples are senile gangrene of the toes and the feet. The affected part becomes engorged with blood before its death takes place, and the coloring matter transudes the tissues, and gives rise to a dark red or purple appearance. At the same time, the tissues begin to dry from evaporation. The part first becomes leathery, then perfectly hard, brittle, and black. Between the dead and the sound tissues there is always the inflammatory zone, or line of demarkation. Dry gangrene is often an aseptic gangrene. This form of gangrene is not usually attended with

any general constitutional symptoms. Attempts to remove the dead tissue should be postponed until separation takes place.

Moist Gangrene.—This is death of soft tissue, followed by decomposition and putrefaction, which is brought about by the presence of micro-organisms. These organisms may reach the part either through the air or through the circulation. The putrefying tissue takes on the characteristic odor of putrid animal matter, disintegrates, and liquefies. Not infrequently gases are formed in the tissues during the process of disintegration, causing puffiness of the part, which crepitates under pressure. This is termed *emphysematous gangrene.* The condition is sometimes associated with alveolar abscess or severe crushing injuries of the soft tissues of the face, or it may result from ligating the facial artery, or by septic poisoning. Moist gangrene is necessarily a progressive gangrene.

Symptoms.—The local symptoms are at first acute inflammation, with great congestion, and pain of an intense burning character. Later the pain ceases, followed by loss of sensation and of power to move the part. The local temperature falls below normal, and pulsation in the arteries cannot be detected. The color, which at first was dusky red, gradually assumes a blue, purple, dirty brown, or black. Occasionally blebs are formed, and the superficial vessels are marked by lines of dark discoloration. Vitality, even at this stage, is not absolutely destroyed, and may still be restored; but if the cuticle separates from the derma, and can be removed by lightly rubbing the part, and if there are crepitation, emphysema, and foul odor, there can no longer be doubt of the absolute death of the part.

The constitutional symptoms of gangrene are those of inflammatory fever of low type, with rapid, feeble pulse, and low delirium.

Prognosis.—The prognosis must be based upon the etiology, the location, the extent of the disease which caused the gangrene, and the general condition of the patient.

An acute, rapidly-spreading gangrene, especially of the face and head, should always be considered as an exceedingly grave condition. The danger arises from the introduction into the general circulation of soluble toxic substances or ptomaines.

When the gangrene is of mycotic origin, and rapidly progressive in its character,—with or without emphysema,—it almost without exception proves fatal, unless early and heroic treatment has been instituted. Death results from septicemia through the introduction of pus-microbes or putrefactive bacteria.

Noma (*cancer aquaticus*), or gangrenous ulceration of the cheek, is characterized by an exceedingly rapid destruction of the tissues. Very little is known of its etiology. Its favorite primary seat is the mucous membrane of the cheek, generally at some distance from the

lips, and most often at the junction of the cheek with the gums; more frequently at the inferior gingivo-buccal fold than at the superior fold. It is generally confined to one side of the face. On this account, some have thought it to be caused by a disturbance of the nervous system. It is more likely to be of mycotic origin. No specific micro-organism has yet been found in noma. Lingard found long bacilli, and Ranke has found streptococci. Various other forms have been discovered, but their relations have not been determined.

It attacks exclusively little children between the ages of two and eight years, who have been poorly nourished, or are suffering from one of the eruptive fevers, typhus, or are of cachectic habit. It is rarely seen in this country except in districts the most unsanitary, but it is quite common in the large cities of Europe among the very poor.

The disease often spreads very rapidly, quickly destroying the entire cheek. It is not, however, confined to the soft tissues, but attacks the maxillary bones, often causing extensive necrosis and loss of the teeth. The disease generally proves fatal in a few days. In one case which came under the notice of the writer at St. Luke's Hospital, the patient lived but five days; another just one week.

Dry gangrene does not therefore present the serious conditions of the moist varieties, as it is not attended with the same dangers of septic intoxication.

Hospital gangrene only occurs as an infection of wounds, and is seldom found outside of unsanitary and overcrowded hospitals. Before the introduction of antiseptic surgery it was quite common, especially in the military hospitals of Europe, and in our own during the War of the Rebellion.

Billroth believed the disease was due to a specific micro-organism, which is only produced under certain atmospheric conditions; hence its epidemic form. There is no doubt that the disease is often carried from one patient to another by the sponges, instruments, the hands of the operators and nurses, and in the atmosphere.

The fact that this terrible disease has been stamped out of the oldest and most unsanitary hospitals by the strict antiseptic treatment of wounds, would seem to be conclusive evidence that it is of mycotic origin.

Treatment.—All patients suffering from gangrene are in a debilitated condition, either from antecedent or concomitant causes, and are consequently unfavorably affected by the so-called antiphlogistic or sedative treatment.

Fever is always the result of septic elements which have gained an entrance into the system; antipyretics are therefore not indicated, but effort should be made to remove the primary cause of the infection. This is the most important point in the whole line of treatment.

The strength of the patient must be supported from the very beginning, by a generous diet and the use of stimulants. In case of feeble heart-action, digitalis is administered with benefit. The bitter tonics are often beneficial in improving the appetite.

In all cases of gangrene of the face and oral cavity, the removal of the dead tissues should be accomplished just as soon as the line of demarkation has been established, not waiting for complete separation to take place. The partially separated tissue may be removed by the scissors and the curette, after which the wound resulting from such treatment may be cared for upon antiseptic principles.

In cases of emphysematous gangrene, the gases and fluid should be evacuated just as soon as their presence is established, by numerous small incisions made over the affected area, care being taken not to injure important blood-vessels. Good drainage must be established and maintained so long as discharges are present.

The most favorable symptom after operation in cases with septic intoxication is a reduction of the temperature within a few hours to the normal point. The removal of the tissue which had caused the septic poisoning, and the elimination of these toxic substances from the system through the excretory organs, produces a subsidence of the constitutional symptoms, and if the patient has sufficient strength to carry him through the shock of the operation, he has good prospects of an ultimate recovery.

In noma and hospital gangrene, after the removal of the dead tissue with the scissors and curette, and thorough irrigation, the surfaces of the wound should be seared with the electro-thermal cautery. Antiseptic conditions should be maintained in the after-treatment. This will be found to be somewhat difficult of accomplishment when the disease is associated with the oral cavity; but with care and persistent effort much may be done in this direction. Thiersch solution and boric acid solution are valuable antiseptics, and may be used with impunity in the oral cavity, without fear of toxic symptoms.

REVIEW.

CHAPTER IX.

Are necrosis, caries, and gangrene diseases?
Give the definition of each of these conditions.
What is the distinction between this form of death and somatic death?
What are the causes of necrosis, caries, and gangrene?
Into how many groups are these causes divided?

What does the first group include?

Name the classes of injuries of the second group.

What is the effect of high and low temperature upon tissues?

What conditions are included in the third group?

Do these causes ever act together?

What effect does the lowering of the vital powers have upon tissues when only slightly injured?

How long will various tissues live after being cut off from their blood-supply?

What is the effect upon tissues having special functions?

In transplanting or replanting teeth, how long a time may a tooth be out of the mouth with safety to the operation?

When the term necrosis is used technically, to what does it refer?

What is the difference in the degree of injury necessary to cause death in bone over that in soft tissue?

How is this explained?

What is another reason found in the bone itself?

How does death of dentine occur?

What terms are applied to the dead portions of bone and of soft tissues?

What are the *causes* of death of bone-tissue?

What are the variations in the extent of necrosis?

What are the most common causes of necrosis?

By what means is the process of separation accomplished between the living and dead tissue?

Is it a slow or a rapid process?

Is the structure of the dead bone changed?

What inflammatory condition is always present?

What is the character of the pus?

Name the differences in the appearance between a sequestrum and living bone.

How are these differences explained?

What is caries?

How does it differ from necrosis?

By what means may they be distinguished from each other clinically?

What are the causes of caries?

Where are each of these forms commonly located?

What are the local conditions of tubercular caries?

What are the local appearances of syphilitic caries?

What is gangrene?

Into how many forms is gangrene divided?

Name them.

What changes take place in the tissue-cells soon after the death of a soft part?

What other conditions may take place?

What degenerative change may take place in the coagulated cellular elements?

In what kind of tissue is it seen?

How does coagulation of blood or lymph occur?

How does it occur in formed tissue elements and intercellular substance?

Under what conditions may coagulation not occur in the dead cells?

What other process may result in the loss of the nuclei in the dead cells?

What diseased condition of the arteries is intimately associated with gangrene?

How does arteritis cause gangrene?
How may atheromatous degeneration cause gangrene?
What other diseases may produce changes in the walls of the arteries?
How is "dry or senile" gangrene produced?
What follows as a constant result of gangrene?
When is this inflammation most severe?
What is the cause of the decomposition?
What is a definitive inflammation?
What is the line of demarkation?
The fixed terminations of dead soft tissue are classed under how many types?
Describe these four conditions.
What is dry gangrene?
How is it caused?
What is its appearance?
Is this form of gangrene attended with constitutional symptoms?
What is moist gangrene?
How is it caused?
What is emphysematous gangrene?
What are the constitutional symptoms of gangrene?
Describe the local symptoms.
Upon what is the prognosis based?
Is rapidly spreading gangrene dangerous?
What is the usual termination of rapidly progressive gangrene?
What is noma?
What is its probable origin?
Whom does it attack exclusively?
Under what conditions is it commonly seen?
What is the course and termination of the disease?
How does hospital gangrene occur?
What is its probable cause?
How is the disease transmitted?
What proves its mycotic origin?
What is the usual condition of patients suffering from gangrene?
How does an antiseptic or sedative treatment affect such cases?
What is the cause of the fever?
What is the most important step in the treatment?
Of what should the constitutional treatment consist?
What should be the line of treatment in gangrene of the oral cavity?
How should cases of emphysematous gangrene be treated?
What is the most favorable symptom after operation?
How has the reduction of temperature been brought about?
What is the treatment for noma?

CHAPTER X.

TRAUMATIC INFLAMMATORY FEVER.

Definition.—*Traumatic fever* is a reactive fever following shock from injury or operation.

In shock following injury the temperature falls below the normal; when reaction sets in the temperature rises above the normal. As a general rule, the greater the shock the lower the temperature, and when reaction sets in the reactive temperature will be correspondingly high. The fever usually develops a few hours after the injury, and generally subsides in from twenty-four to forty-eight hours.

Traumatic fever may be aseptic or septic, the character depending upon the condition which prevailed at the time of the injury.

Aseptic fever is a condition which accompanies the healing of wounds by first intention. An elevation of temperature from two to three degrees frequently accompanies the healing of wounds which have been treated antiseptically, but in which there is the formation of a considerable blood-clot between the lips of the wound, or in those cases where the bruising and manipulation of the tissues has been extensive. Attention has already been called to the fact that certain chemical substances of a non-pyogenic nature were capable of causing a rise in the temperature when introduced into the circulation. Among these substances is a peculiar ferment, obtained from defibrinated blood, known as *"fibrin-ferment."* This substance, when injected into animals, causes extensive coagulation of the blood, and death. The animal alkaloids or *leucomaines* produced by the *constructive metabolism* of the tissues which occurs in the healing of wounds are not unlikely active factors in the production of this form of fever. On the other hand, the leucomaines produced by the *destructive metabolism*, which takes place in minute portions of tissue, and in blood-clots remaining between the lips of wounds, may produce a like result.

In aseptic fever, the only symptom of marked character is the elevation of the body temperature. This may reach 102° or even 104°, and not entirely disappear for several days. Patients suffering from this form of fever are rarely conscious of feeling unwell, and may be able to sit up in bed, or even move about the room. (Warren.) The conditions under which this form of fever is most commonly seen

are in deep and extensive wounds which are healing by first intention; in wounds closed without drainage; in simple fractures of the bones, and other subcutaneous injuries.

Septic fever was supposed, before the introduction of antiseptic surgery, to be a natural consequence of the *process of repair*, as all wounds healed with more or less inflammation, even though suppuration was not established. The constitutional symptom of fever, which was sometimes very considerable, was called surgical or traumatic fever. We now know that these conditions were due to the presence of micro-organisms in the exudations of such wounds, which produced fermentative changes or decomposition, with the elaboration of ptomaines, the absorption of which caused an elevation of body temperature or fever, by their poisonous effects upon the system.

This type of fever is often seen to-day following wounds which have not been treated antiseptically, and in those injuries which have been exposed to infection, or in which infective material has been introduced. Examination of the blood taken from patients suffering from this type of fever reveals but few bacteria in this fluid, and when they are present rapid elimination or destruction takes place, so that they may entirely disappear in the course of two or three days. Progressive development of micro-organisms in the blood never takes place in this form of fever as it does in septicemia. A rigor followed by a sudden elevation of temperature, occurring on the first or second day after the injury, is indicative of septic poisoning and the formation of pus. A sudden fall of temperature, with a weak pulse, occurring at the same period, would indicate shock from internal hemorrhage, or collapse.

Septic fever occurring in these days of antiseptic surgery is an evidence of either a slovenly operation; of deep-seated, penetrating or tortuous wounds, impossible to cleanse; wounds involving the peritoneum, and of compound fractures of the bone in locations impossible to keep thoroughly clean; as, for instance, in fractures of the maxillary bone of individuals who give no thought to the cleanliness of their mouths, or of infection of wounds by septic instruments, especially in the extraction of teeth. This form of fever generally appears on the second or third day after the injury or the infection, and lasts from two to six days.

The constitutional symptoms are a sudden rise of temperature, 100° to 102° F., skin hot and dry, the pulse rapid, and the tongue coated, accompanied with delirium or with digestive disturbances, heat, restlessness, and thirst. The urine is scanty and high colored, the bowels often constipated. The character of the evening temperature is progressive. On the evening of the second day it may be 101°, the next morning drop one or two degrees, to again rise in the evening

to 102°, while on the fourth day it may reach 103°. These signs of constitutional irritation would certainly point to the presence of decomposition in the exuded material, the establishment of the suppurative process, or some form of infective inflammation. If suppuration takes place, pus is formed on the third or fourth day. The wound then cleans off, the ptomaines are washed away with the discharges, granulations spring up, and the system regains its normal temperature.

Traumatic fever, suppurative fever, septicemia, and pyemia are all dependent upon the same causes, and are of the same nature, the only difference between them being one of degree. The symptoms, however, are somewhat different.

Suppurative fever, or "secondary fever," as it is sometimes called, is dependent upon the suppurative process, and is therefore a septic fever. It is, however, different from traumatic or surgical fever, for in this form the temperature falls as soon as pus is formed. Suppurative fever appears two or three days after the injury, and generally subsides at the end of the first week; but if the fever continues beyond this period, or if at the beginning of the second week there should be a sudden rise of temperature, with or without a rigor, this would be a good reason for suspecting the presence of pus at the point of injury. If the injury is at a point inaccessible to drainage, or difficult to reach with antiseptics, the high temperature will correspond in degree with the severity of the suppurative process; while on the other hand, if the pus can be reached and evacuated, and the wound treated antiseptically, the temperature will soon subside and the general febrile symptoms disappear.

It more commonly happens, however, when the infective inflammation is of an acute form, that the character of the fever will be of the *continued type*, with frequent exacerbations. The local symptoms, under proper treatment, become less acute, and eventually, as in joint and bone diseases, numerous sinuses are formed, communicating with the seat of suppuration, and from which pus freely discharges, thus establishing *chronic suppuration*. From this time forward the fever assumes the *remittent type* of suppurative fever, which consists of a normal or subnormal temperature in the morning, followed by a rise of from two to as high as six degrees in the afternoon, and accompanied by the hectic flush and other signs of febrile disturbances. If the suppurative process is not checked, there will be considerable loss of flesh, great prostration, diarrhea, and profuse perspiration at night. Emaciation is progressive, bed-sores are developed, and the strength of the patient gradually fails, until it becomes only a question of how long the physical endurance will hold out. (Warren.)

In chronic suppuration resulting from tubercular disease this form of fever may continue for months, with increasing but more gradual

emaciation; and when death occurs, post-mortem examination of the internal organs shows extensive amyloid degeneration. These facts prove conclusively that the temperature is due to the continued absorption of chemical substances elaborated in the wound by a destructive metabolism, or by the presence of the pus-producing micro-organisms. The clinical evidence of this lies in the fact already indicated, namely: That as soon as the supply of the pyogenic material is cut off by thorough drainage and antiseptic treatment, the febrile symptoms disappear.

Suppurative fever of long continuance, if accompanied with great emaciation and prostration, would be a *contra indication* for a severe surgical operation, as in all probability there would exist extensive amyloid degeneration of the internal organs.

Treatment.—The local treatment of traumatic inflammatory fever consists of freeing the wound of all tension, providing against the possibility of the retention of the inflammatory discharges, irrigating thoroughly with germicidal solutions, covering the wound with boric acid or iodoform, and the use of sterilized dressings, changed every day or twice a day if necessary.

The constitutional treatment would be to clear the bowels by means of a saline cathartic, control the temperature with aconite or the antipyretic drugs; the pain by the use of morphia, and nervous excitement with bromid of potassium.

The treatment of suppurative fever of either the acute or chronic type consists in the establishment of thorough drainage, and of disinfection of the entire suppurating surface. To accomplish this the pus cavity must be laid open by free incisions, and its walls thoroughly scraped with the curette. After the infective material has been scraped away the wound may be cleansed with antiseptic solutions, followed by peroxid of hydrogen, and again irrigated with the carbolic or bichlorid solution. When joints are diseased, amputation frequently offers a better chance of saving life than by resecting the joint.

The constitutional treatment should consist of free stimulation, a nutritious diet, and tonics. Out-of-door life, if practicable, will many times bring about a decided improvement in the conditions. If the affection is the result of tuberculosis, the prognosis is most unfavorable.

REVIEW.

CHAPTER X.

What is traumatic fever?
How does shock affect the temperature of the body?
When reaction sets in, how is the temperature affected?

What relation does the degree of shock bear to the reactive temperature?

When does the reactive fever develop?

How long is its duration?

What is aseptic fever?

What degree of temperature often accompanies the healing of wounds?

What is the explanation of the elevation of temperature in the healing of wounds?

What is the effect of the "fibrin ferment" when injected subcutaneously in animals?

What other substances produced in the tissues may cause the elevation of temperature?

By what two processes are these leucomaines produced?

Are the leucomaines produced by the *constructive* and *destructive metabolism*, pyogenic or non-pyogenic products?

What symptom is generally the only marked one in aseptic fever?

What is the condition of the patient at such time?

Under what circumstances is this form of fever most commonly seen?

What is septic fever?

What are the causes of septic fever?

Under what conditions is this form of fever usually seen to-day?

Are micro-organisms found in the blood of such patients?

What becomes of the micro-organisms?

Are they ever found progressive in their development as in septicemia?

Of what is a sudden rise of temperature indicative if it occurs upon the first or second day after an injury?

Of what is a sudden fall of temperature associated with a weak pulse indicative?

Of what is septic fever an evidence?

How may infection occur in the extraction of teeth?

When does this form of fever generally appear, and how long does it last?

What are the constitutional symptoms?

What is the character of the evening temperature?

What degree of temperature may be reached upon the fourth day?

Of what are these signs of constitutional irritation an evidence?

What occurs upon the establishment of suppuration?

What is suppurative fever?

How does it differ from traumatic or surgical fever?

What is the character of the fever in the chronic form?

How is this form of fever expressed?

Name the other symptoms of this form of fever.

How does this form of suppurative fever differ from that caused by tubercular disease?

What do these facts prove as to the septic nature of the disease?

What are the clinical proofs of the same facts?

Why should suppurative fever be a contra-indication for severe surgical operations?

What is the local treatment of traumatic inflammatory fever?

What is the constitutional treatment?

How should suppurative fever be treated locally?

What is the local treatment in joint-disease?

What should be the constitutional treatment?

CHAPTER XI.

SEPTICEMIA.

Definition.—Septicemia (from the Greek σηπτός, putrid, and αιμα, blood).

Septicemia is a disease or condition induced by the absorption of septic products; a form of blood-poisoning.

Two forms of septic fever are still to be considered, viz: Septicemia and pyemia. Both of these conditions have been recognized ever since their description by Hippocrates, and on account of their dangerous and often fatal character have always been the subjects of anxious thought and investigation. Since the promulgation of the germ theory of disease, investigation and experimental research into the cause of these diseases have been greatly stimulated, and though it may not be said with positive assurance what the actual cause may be, yet such advancement has been made as to make it almost certain that they are due to the action of the micro-organisms of putrefaction, the saprophytic germs. Antiseptic surgery has here won another signal victory by preventing the development of these grave conditions, thereby saving thousands of lives that would otherwise have perished. Both of these conditions, however, are still occasionally seen in hospital wards, where thorough antisepsis has been neglected or has failed on account of the peculiar character of the wound, which has rendered it impossible to carry out the most approved methods; or by reason of the severity of the injury to the tissues, which has resulted in gangrene and sloughing.

Billroth said, in speaking of these diseases, that septicemia bore the same relationship to surgical or traumatic fever that pyemia bears to suppurative fever, each being the malignant type of the corresponding milder affection.

Surgical fever, as already stated, is developed in the earlier stages of the process of repair in wounds, before the establishment of the suppurative process, by the absorption of ptomaines elaborated in the wounded tissues as a result of putrefaction; the fever subsides upon the formation of pus, which cleanses the wound and prevents further absorption by washing away the pyogenic substances. (Warren.)

Septicemia is also dependent upon septic infection of the wound,

the absorption of poisonous substances resulting from decomposition, which produces a profound impression upon the system, and often terminates fatally. The disease is therefore a *malignant* form of putrid infection, and is a sequel of a grave type of surgical or traumatic fever. It is seen most frequently as a complication of amputation wounds, severe crushing injuries, compound fractures of the long bones and of the lower jaw, acute osteomyelitis, deep tortuous wounds which it is impossible to treat by thorough antisepsis, gangrenous conditions of the tissues, wounds of the peritoneum, and in obstetrical cases from the retention and decomposition of blood-clots or placental débris within the uterus. It most often occurs in wounds in which the discharges are abundant; the septic micro-organisms have had free access, and the process of putrefaction has been established.

The disease is characterized by serious constitutional disturbances, such as high temperature, great prostration, disorders of the nervous system, inflammatory conditions of certain internal organs, accompanied by typhoid symptoms and a tendency to heart-failure.

Causes.—The nature of the toxic element which produces the disease is not yet fully demonstrated, but it is generally accepted to be a product of the process of putrefaction, either in the form of ptomaines which are absorbed by the system, the introduction into the body and tissues of certain forms of micro-organisms which rapidly grow and multiply, or possibly of the introduction of some "ferment-like substances" having the power of reproduction, and acting within the organism like the poison of serpents, the virus of tetanus or of diphtheria. (Warren.)

By experimentation upon animals it has been demonstrated that there are *two varieties* of septicemia,—one the result of poisoning by a chemical substance, and the other by the presence of bacteria in the blood. The first is termed *sapremia, toxemia,* or *septic intoxication.* The second is termed *mycosis* or *septic infection.* In the first the symptoms begin immediately upon the injection of the poison into the tissues, their intensity being governed by the size and the virulence of the dose, while in the second form the disease is developed after an interval, probably dependent upon the rapidity of the development and the multiplication of the bacteria in the blood, but progressing uninterruptedly to a fatal termination.

Koch could not discover a constant bacterium in septicemia, and therefore concluded that those which were found in the blood were not the specific organism of the disease. The forms of bacteria usually found in septicemia in man are the staphylococci and the streptococci. Micro-organisms are, however, by no means always present in the blood in septicemia. Rosenbach found that blood-cultures taken from septicemia in man proved sterile. In these cases,

however, he found the staphylococci present in the blood. He therefore comes to the conclusion that the disease in man is usually the result of the absorption of ptomaines or ferments, and not of an invasion of the blood by a specific micro-organism. Besser takes just the opposite view, and believes the disease is produced solely by the streptococcus.

Clinically, the two forms of the disease just mentioned are also found in the human species. In the former there is an early absorption of the products of putrefaction, but the symptoms, as soon as further absorption of the poisonous substances is prevented by draining and cleansing the wound, rapidly disappear. In the latter the symptoms develop more slowly, and there are progressive changes established which often continue to a fatal termination in spite of all efforts by antiseptic treatment of the wound. This form is doubtless due to the presence of micro-organisms in the blood, although their demonstration is not always possible. Gussenbauer thinks that there is frequently a mixture of these two types of the disease.

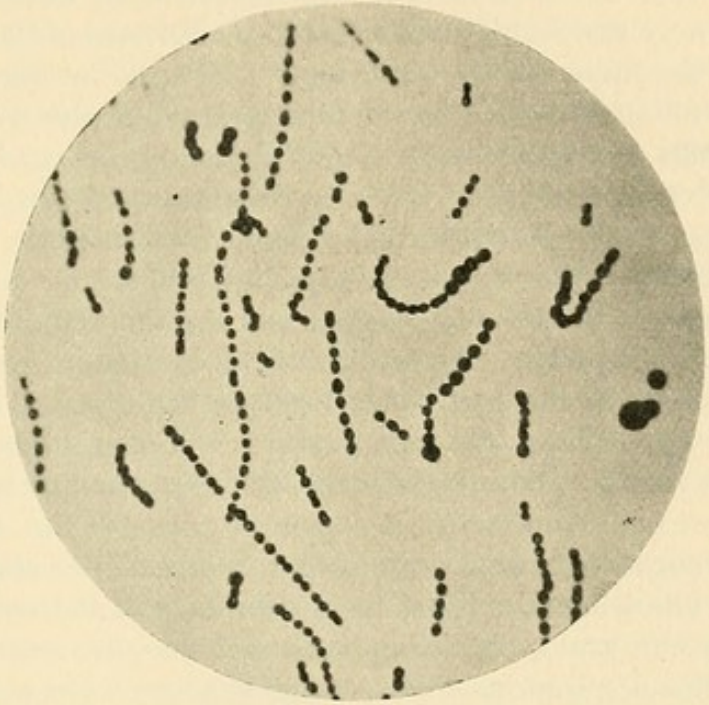
Avenues of Infection.—*The avenues through which infection may enter the system* are: septic wounds, accompanied by gangrene and sloughing; this is the most common mode of infection, though the disease may appear in its malignant form from even a very slight and insignificant wound, in which the putrefactive process has been established, or bacteria have multiplied and been diffused through the system. Septic infection sometimes accompanies other traumatic infective diseases, like erysipelas and hospital gangrene. Infection may also take place through the mucous membrane of the intestinal tract. The intestinal canal is always loaded with the various micro-organisms of disease, and under favorable conditions represented by enfeebled health they may gain access to the tissues and blood, and, as pointed out by Cheyne, a local injury or inflammation may furnish the proper soil for the development and multiplication of these wandering bacteria, and thus an infective inflammation will be established, followed by general infection of the system.

The genito-urinary tract, when in a normal condition, is rarely an avenue through which infection takes place. The uterus immediately following parturition is a quite common avenue of infection, and many lives have heretofore been lost through ignorance of or an imperfect knowledge of this fact. The respiratory mucous membrane is generally considered to be proof against the entrance of putrefactive infection, as is also the skin, when it is in a normal condition. Fig. 35 is a culture from "Sputum Septicemia," *Bronchitis putrida*, or fetid bronchitis resulting from gangrene of portions of the lung.

Sapremia.—Sapremia, or the toxic form of septicemia, is most frequently observed in obstetrical wards and lying-in hospitals, and is

due to putrefaction of blood-clots and fragments of placenta retained in the uterus. Infection takes place by absorption of the poison through the mucous membrane of the vagina or the uterus; through

FIG. 35.



MICROCOCCUS OF SPUTUM SEPTICEMIA. × 1000.

abrasions or lacerations of the vagina or cervix uteri; at the point of attachment of the placenta with the uterus, or directly into the circulation through the uterine sinuses.

Symptoms.—The disease is ushered in by a sharp and sudden rise of temperature, 101° to 103° F., but rarely higher. The chill which usually precedes the onset of other acute affections is commonly absent. The parts which are chiefly attacked by the poison are the blood, the nervous system, and the intestinal canal. The changes in the blood are marked, causing anemia, while the inflammatory condition of the intestinal canal is doubtless due, in part at least, to the efforts of nature to eliminate the poison. The temperature remains high, and is accompanied later by delirium. The skin is cold and clammy, and coma supervenes in the fatal cases.

In surgery it rarely happens that conditions favorable to the development of this form of blood-poisoning occur. It is seen occasionally, however, in large wounds which have not been provided with proper means of drainage for the escape of the blood and serum; in the presence of gangrenous and sloughing tissues; in psoas or other deep-seated abscesses from putrefaction of their contents, or in wounds of the peritoneum followed by the formation of blood-clots within the abdominal cavity and their decomposition. The results of treatment

in this type of the disease are usually very striking and satisfactory, as the prompt removal of the decomposing material contained in the uterus, under antiseptic conditions, is followed in a few hours by a lowering of the temperature and a disappearance of the alarming symptoms. The same is true of the treatment of those conditions which are purely surgical in their nature; the removal of the putrefying substances, accompanied by thorough antiseptic treatment of the wound, is generally followed by the most gratifying results.

Symptoms of Septicemia.—Septic infection, or true septicemia, does not differ materially, in its early constitutional symptoms, from those just described. The principal difference exists in the more gradual development of the disease, and the interval which occurs before the multiplication of the micro-organisms, or the elaboration of the poison is made manifest. The fever is of the continuous type, like that in sapremia, but in the malignant form of the disease, as the fatal termination approaches, the temperature will run higher, ranging from 103° to 106° F., and may suddenly fall to normal or below.

A sub-normal temperature is sometimes observed in cases resulting from strangulated hernia, gunshot wounds of the abdomen, and occasionally following operations for the removal of abdominal tumors. The pulse is soft, rapid, becoming weak and thready, and easily compressible. The respirations are rapid and shallow. Great prostration, accompanied by headache, loss of appetite, and thirst, are the usual symptoms. Later the patient becomes apathetic and indifferent to surroundings; diarrhea and vomiting are frequently developed. Enlargement of the spleen and lymphatic glands throughout the body is a characteristic of the disease. The skin takes on a pale, dusky hue, and the conjunctivæ are tinged a faint yellow, though this condition is not so marked as in pyemia. Examinations of the blood during life show a breaking down of the red blood-corpuscles, an increase in the number of leucocytes, and the presence of micrococci. Scarlet eruptions sometimes occur, resembling the rash of scarlet fever. The skin, which in the earlier stages of the disease was hot and dry, later is bathed in perspiration, and finally becomes cold and clammy. The tongue is dry, hard, and coated brown; the teeth are covered with sordes. The expression becomes listless and indifferent. The diarrhea increases, the stools are offensive, and the urine becomes concentrated and scanty in amount. The feces and urine may be passed involuntarily. Delirium follows the stupor, coma is developed, and death takes place in collapse.

Diagnosis.—The diagnostic signs of the disease would be the high continued fever, with absence of chills, the listlessness and indifference of the patient, and the general disturbance of the alimentary canal, as constitutional symptoms. An increased area of dullness in the region

of the spleen, the presence of albumin and micro-organisms in the urine, would be further aids to diagnosis, as would also a septic condition of the wound. There are no very marked or characteristic signs in this disease to guide in the diagnosis, and the surgeon must therefore depend upon the general appearance of the patient, and his ability to reach a diagnosis by the process of exclusion.

Prognosis.—The prognosis of this disease is always grave. When the disease is caused by septic intoxication (*sapremia*), it is much more favorable than when the result of septic infection *(mycosis)*. In the former, when of a pure type, the removal of the decomposing material is usually followed by a subsidence of the constitutional symptoms, but inasmuch as the surgeon can never be quite sure that the disease is not of a mixed type, the opinion must be a guarded one.

When the disease is the result of acute septic infection, the prognosis is exceedingly unfavorable, as it is one of the most fatal of diseases. The chronic form of the disease gives a little more hope of a favorable termination, although a majority of this class of cases finally end in death.

Treatment.—The treatment must be both local and constitutional. Prophylactic treatment consists principally in establishing aseptic conditions of the wound and the surroundings, by a strict application of the principles of antiseptic surgery. As soon as the diagnosis of septicemia is made, the attention of the surgeon should be at once directed to the condition of the wound, and the most thorough measures of disinfection inaugurated, compatible with the strength of the patient. The wound must be opened by removing the stitches, and all stitch-sinuses fully exposed, and free drainage established for the putrefying discharges. Irrigation of the wound with bichlorid of mercury solution, 1 to 1000; carbolic acid solution, 1 to 20, or peroxid of hydrogen in moderate quantity for the purpose of disinfection, is of prime importance before applying the dressings. In certain cases it will be necessary to curette the surface of the wound in order that the disinfecting fluid may reach the deeper portions of the tissues, and thus hasten the expulsion of the invading micro-organisms. The wound may then be packed with iodoform or boric acid gauze, or hot antiseptic fomentations may be applied to stimulate a free drainage.

In sapremia, free irrigation of the wound with antiseptic solutions is frequently followed by an immediate relief of the symptoms. Washing of the uterus in puerperal fever gives a like result.

The constitutional treatment should be directed to the elimination of the ptomaines taken up by the circulation, which may be accomplished by a free saline purgative; and to sustaining the strength of the patient and his resistance to the effects of the poison. Reliance must therefore be placed upon nourishment and alcoholic stimulants. The

form of nourishment must be suited to the condition of the digestive system, and is best administered in small quantities and frequently. Large quantities of alcohol can be consumed by a patient suffering from this disease without producing alcoholism.

The use of antipyretic drugs for the control of the fever does not give very satisfactory results, and is therefore not recommended by many of our best surgeons.

Heart-failure should be guarded against by the use of heart-tonics. The tincture of digitalis, in doses of 5 to 10 drops, is of great value in this direction. Strychnia in doses of gr. 1-40 to 1-30 every two to six hours, is one of the best. The diarrhea can be treated by opium, or bismuth and tannin.

REVIEW.

CHAPTER XI.

Give the definition of septicemia.
What is the character of the disease?
What is the effect of antiseptic surgery upon the disease?
Under what conditions is it seen to-day?
What does Billroth say of the relationship of septicemia to surgical fever?—and pyemia to suppurative fever?
How is surgical fever developed?
Upon what condition is the development of septicemia dependent?
What may the disease therefore be termed?
Under what conditions is it most frequently seen?
By what constitutional disturbances is the disease characterized?
What is the nature of the toxic element which produces the disease?
Name the three ways by which the toxic element might be introduced into the system.
How many varieties of septicemia are recognized?
What are they termed?
What is the cause of sapremia?
What is the cause of mycosis?
When do the symptoms develop in sapremia?
When do they develop in mycosis, and how does it progress?
What was the result of Koch's researches?
What forms of bacteria are usually found in septicemia in man?
Are micro-organisms always found in the blood in septicemia?
What is the opinion of Rosenbach as to the cause of this disease?
What is the opinion of Besser?
How many forms of the disease are seen in man?
What are they termed?
How do sapremia and septicemia differ?
Through what avenues may the infection enter the system?

Which is the most common?
Where is sapremia most frequently seen?
What are its causes?
How does the infection take place?
Describe the symptoms.
Under what conditions is this form of the disease seen in surgical cases?
What is the result of antiseptic treatment in these cases?
Describe the symptoms of septicemia.
In what particulars are the symptoms like sapremia?
What are the diagnostic signs of septicemia?
What is the prognosis of septicemia?
What is it in acute septicemia?
What is it in chronic septicemia?
Of what does prophylactic treatment consist?
What should be the local treatment?
How should sapremia be treated locally?
What should be the constitutional treatment?
How may heart-failure be guarded against?
What remedies are suggested to control the diarrhea?

CHAPTER XII.

PYEMIA.

Definition.—*Pyemia* (from the Greek πύον, pus, and αἷμα, blood).

Pyemia is a phlebitic or sanguiferous septicemia, with the presence of pyogenic micro-organisms in the blood, and the formation of metastatic abscesses as a result of the localization of the micro-organisms in remote portions of the body.

Pyemia or *septic fever* is an infectious disease, termed by Velpeau a purulent infection, developed during the process of suppuration in wounds or the formation of abscesses, and is due to the presence in the blood of pyogenic bacteria. It is attended with the formation of *multiple, metastatic,* or *secondary abscesses* in various portions of the body, as a result of the lodgment of the micro-organisms and their development into colonies, thus establishing independent foci of suppuration. The same form of micro-organism is found in the secondary abscesses that are present in the wound secretions. (Senn.) The disease is further characterized by recurring chills and fever, of an intermittent type.

The disease is always associated with suppuration, and is in reality a complication or a grave constitutional expression of that affection. It is never seen before suppuration has been established.

The disease was well known and correctly described by the ancient medical writers. The old view that the pus found in the wound was absorbed or entered the blood-current direct, was abandoned long ago. We know, however, that certain elements of the pus, and micro-organisms, do gain access to the circulation, and by their lodgment cause the formation of thrombi, which finally, on breaking up, float away in the blood-current, and produce embolism by plugging a small vessel in some distant location of the body, and thus cause the formation of septic infarcts and metastatic abscesses.

Predisposing Causes.—All suppurating wounds, especially of bone, are predisposing causes of pyemia. Among those which predispose to metastatic inflammations are contused wounds, wounds of joints, compound fractures, particularly of the head, abscesses associated with the jaws, osteomyelitis, injuries of the veins, wounds received by individuals in a low state of vitality, and wounds received

in war, on account of the severe shock and the difficulties in the way of immediate care of the injured, and of thorough antiseptic treatment upon the field. The disease usually makes its appearance about ten days after the injury, when the suppurative process is at its height, though it may begin at any time after the suppurative process has been established.

Climate.—Certain seasons of the year, especially the early spring, are thought to predispose to the disease, as the conditions of the atmosphere at this time seem to favor the more rapid growth and development of the pus-microbes.

Age and Sex.—The influence of age and sex as predisposing causes is of considerable importance. Men in the prime of life are much oftener affected with the disease than children, old men, or women at any time of life. The greater prevalence of the disease among men in the prime of life is doubtless due to the greater exposure to traumatism to which they are subjected; to the diseases incident to advancing years; and also to certain conditions of the system, such as diabetes and alcoholism, which render the system peculiarly susceptible to the influence of traumatisms.

Active Causes.—The active causes of the disease are the pus-microbes. It is only during the last decade that the bacterial nature of the disease has been satisfactorily demonstrated by the discovery of the pus-microbes—the staphylococcus and streptococcus—in the blood of patients suffering from pyemia, and the production of the disease by inoculating animals with infected tissue and blood. Koch produced the disease in rabbits by injecting into the subcutaneous tissue fluid obtained from decomposing flesh. From the heart of this animal he abstracted blood and injected it into another rabbit, and again produced the characteristic metastatic deposits. In the blood-vessels he found chain-like micrococci which were adherent to the walls in little masses; each mass inclosing several blood-corpuscles. The micro-organisms seemed to possess the power of causing adhesion of the blood-corpuscles and the formation of thrombi. He was not able to find the micrococci in the lymphatics.

Besser examined the blood, pus, and parenchymatous fluids of 23 cases of pyemia, and found the staphylococcus present in 8 cases, the streptococcus in 14, and both kinds in 1. He also collected the records of 46 cases, and out of these he found the staphylococcus present 22 times, the streptococcus 21 times, and both in 3 cases. He thinks the cocci of pus and the cocci of pyemia are identical.

Pawlowsky is of the opinion that the staphylococcus is the usual cause of the disease. The data presented up to the present time are not sufficiently conclusive to settle the question of the particular pyogenic organism which causes the formation of the metastatic abscesses,

for both the staphylococcus and the streptococcus have been found in the disease in man, but the proof is sufficient to establish the pus-microbes as the active cause of disease.

Infection of the general system always takes place from a suppurating wound or an abscess, and the avenues through which the infection enters the system are, almost without exception, the blood-vessels. Occasionally, however, it may gain an entrance through the lymphatic system.

The effect of the micro-organisms when they have developed in such quantity as to overwhelm the tissues in the immediate neighborhood of the wound or abscess, is to cause an infective inflammation of the walls of the veins with which they come in contact, with the result of causing a *thrombo-phlebitis*. Nutritional changes take place in the endothelium as a result of the inflammation, and rough places are formed upon the inner surface of the vein. Leucocytes now congregate at the roughened places and become adherent, finally forming homogeneous masses, and thus become the starting point of a thrombus, which may increase in size, and more or less completely fill the lumen or caliber of the vessel. The red corpuscles also collect about these rough places, and materially aid the process of forming a thrombus. Eventually the thrombus softens and breaks down as a result of its infection with the micro-organisms of the wound, and masses are floated away in the blood-current as emboli, or a zoöglea mass of micrococci may accumulate upon the inner wall of the vein, and finally become dislodged and carried away by the circulation, to be deposited in the lungs, where they form fresh foci of infection, which may result in the formation of pulmonary *metastatic abscesses*.

A *thrombus* is a clot of blood formed within the heart or blood-vessels during life, and is due usually to some impediment to the circulation, or to alteration in the blood or vessel-walls. *Thrombosis* is the process by which the clot is formed.

An *embolus* is a detached thrombus, or part of a thrombus, a clot of blood, or other foreign substance, brought by the blood-current from a distant artery, and forming an obstruction at its place of lodgment. The act or process by which this is brought about is termed *embolism*. The process of closing or plugging an artery or capillary by an embolus is called *infarction*, and the area of tissue cut off from its supply of blood is called an *infarct*. The shape of the *infarct* is always that of a wedge, with the apex at the point of lodgment of the embolus, and it corresponds in size to the caliber and distribution of the vessel obstructed. The infarct may be discovered by the ischemia of the part which has been deprived of its supply of blood.

Thrombo-arteritis also occurs from penetration of the walls of the arteries by the micrococci, but on account of the greater density of the

walls and rapidity of the blood-current, thrombosis rarely occurs. When thrombi are formed in the arteries they are not of such serious consequence as when developed in the veins, as they are not carried to distant organs, but when detached the emboli are simply deposited in adjacent capillary regions. Minute emboli and micrococci may pass through the capillaries of the lungs, enter the pulmonary vein, the left side of the heart, and thus be carried through the arterial system to remote organs, where they become arrested in the capillaries and favor the development of metastatic abscesses; or zoöglea masses of micrococci may form in the pulmonary vein and result in the development of a thrombus, which later breaks up by puriform softening induced by the micrococci, and fragments are floated away to cause embolism in the capillaries of distant organs.

The internal organs which are most often the seat of metastatic abscesses are the lungs, liver, kidneys, spleen, brain, and heart. The lungs are the most common location of these abscesses. Among the important complications of this character are the formation of suppurative conditions of the joints, as a result of secondary infection, and phlegmonous inflammations, which frequently accompany puerperal pyemia. The parotid gland is not an infrequent seat of metastatic abscess.

Pus, as a rare occurrence, may form an abscess and find its way into the circulation through absorption or necrosis of the walls of the vein. The so-called spontaneous pyemia occurs by means of an intervascular infection. Micrococci are frequently found circulating in the blood of individuals of a more or less enfeebled condition of health, and under such circumstances a cold or trifling wound, a bruise or an injury occurring to a bone without an external wound, as, for instance, to the medulla, by a fall, may result in acute symptoms with the development of an osteomyelitis, and the patient eventually dies of pyemia.

Single organisms would be very unlikely to inaugurate the suppurative process, as the resistance of the tissues is such that their pathogenic action would soon be neutralized, and they are moreover quickly eliminated from the blood. (Warren.) It only seems necessary, however, in certain individuals, that the tone of the general system should be somewhat lowered, or that there shall exist at some point a lack of normal resistance induced by disease or injury, for the rapid development and localization of these organisms in the circulation to take place, thus overcoming the barriers afforded by the tissues against their growth and multiplication. Such localization may become the starting point of an extensive infection of the system with the formation of metastatic abscesses, and all the other conditions of pyemia. Poore has reported several cases of chronic pyemia resulting from alveolar abscesses, which illustrate the danger of the retention of

small accumulations of pus under certain conditions of the general health.

Symptoms.—The most prominent of the symptoms of pyemia are the chills, which are severe and prolonged. This symptom is first made manifest usually in the second week of the healing process, when suppuration has been well established. It is usually associated with a high temperature, 104° to 106° F., soon terminating in a drenching perspiration, the temperature quickly falling to the normal or below. The fever is therefore of the intermittent type. The chills, however, may not accompany the first onset of the fever, while in other cases the chills may be the only symptom which at first arrests the attention of the surgeon, as the febrile disturbance has been but slight. The occurrence of a rigor during the healing of a wound should always arouse the suspicion of the surgeon, and cause a careful inspection of the wound, which generally reveals a local infection, and symptoms of an infective inflammation. The lips of the wound are red and swollen, and the interior will present a characteristic grayish, sloughy appearance. The fever is of an irregular, intermittent type, and may vary with the frequency of the chills or possess an hourly variation of its own. Several chills may occur during twenty-four hours, followed by profuse perspiration and fall of temperature. The recurrences, however, are very irregular. The fever differs from all other forms in not having a regular evening rise and morning fall of the temperature, and is characterized by a series of sharp rises and falls during the twenty-four hours.

The development of secondary abscesses is indicated by febrile exacerbations and local inflammatory symptoms. The formation of an abscess in the lung, or a septic pleuritic effusion, will be indicated by pain in the side and respiratory disturbance. Metastatic abscesses in the liver cannot be readily recognized unless occurring near the surface, when a peritonitis will be developed, and its presence indicated by sharp pain at that location. Inflammatory conditions of the joints resulting from secondary deposits are a frequent symptom of the disease, and are accompanied by considerable swelling of the surrounding soft parts. Swelling of the parotid gland, with the formation of metastatic abscesses, is not an infrequent symptom of the disease. The skin becomes *icteric,* or tinged a deep yellow hue. With this discoloration of the skin there is always a progressive emaciation, which in chronic cases becomes extreme. The breath has a sweetish, hay-like, purulent odor. The pulse becomes weak, rapid, and thready; the strength rapidly fails. The tongue is dry and coated brown, the gums and teeth are covered with sordes. A scarlet rash, or erythematous patches appear, which have a tendency to form pustules, doubtless from localization of the micrococci, which later may coalesce and dis-

charge an offensive puriform fluid. The mental condition of the individual is generally unaffected, but there is developed as a marked symptom a general hyperesthesia, and the patient complains of sharp pains in various localities of the body, which may be due in some cases to metastatic abscesses, and in others to hypersensitiveness of the nerves. The presence of an infective endocarditis would be revealed by the heart-sounds. The spleen is not so often affected with enlargement in pyemia as it is in septicemia. The kidneys are occasionally affected, but the urine affords but little positive information. Brain symptoms are rarely present, though metastatic inflammations may occur and hemiplegia result from emboli due to the formation of a thrombus in the left side of the heart. The duration of acute pyemia is from ten to fifteen days. The chronic form of the disease may run for weeks and months. In the later stages of the disease the mind fails, delirium sets in, followed by coma and death.

Diagnosis.—The diagnostic signs of the disease are repeated chills, the intermittent type of the fever, excessive diaphoresis, emaciation, hyperesthesia, and great prostration. The presence of metastatic abscesses and inflammatory conditions of the joints would confirm the diagnosis.

Prognosis.—The prognosis, as a rule, is exceedingly unfavorable in the acute cases. It has generally been considered to be a fatal disease, though undoubted cases of recovery are on record, with a relatively higher percentage of recoveries from puerperal pyemia than from surgical pyemia. The chronic form of the disease is more likely to terminate favorably than the acute form. Guerin thinks these patients do not long survive their recovery from the disease.

Treatment.—The treatment of pyemia is for the most part prophylactic, and this consists in the prevention of suppuration. No more brilliant achievement has ever been obtained in any department of medicine or surgery than that recorded in the treatment of wounds by antiseptic methods. The antiseptic treatment of wounds has almost entirely abolished pyemia and kindred diseases from hospitals, where, before its introduction, they were of common occurrence. The first step to be taken upon the discovery of septic disturbance is thorough and complete disinfection of the wound, after which the surfaces should be curetted for a sufficient depth to remove all sloughing and putrefying material, not only of the wound itself, but also of the interior of the vein through which the infection is being introduced into the system. This method has been successfully carried out by Macewen in thrombosis of the lateral and sigmoid sinuses following suppuration of the middle ear and of the mastoid cells. He recommends the opening of the vein and the removal of the disintegrating clot with a small spoon, and, if antiseptic solutions are used to wash out the débris, care must

be taken to avoid the admission of air into the vein. Tying of the vein is recommended by others, before the clot is removed, as a further precaution against this danger, and also to prevent the further passage of septic material into the circulation. The ligature must be placed at a point between the wound, the point of primary suppuration, or the puriform thrombus and the heart. Amputation of a limb when the point of infection is located in that region has been recommended. Warren made the attempt to save a patient by this method, but without success. Several cases have been reported, however, of favorable results. To be successful, the operation must be done early, and the surgeon must be reasonably sure that the point selected for the operation is above the location of the thrombus. Metastatic abscesses, where accessible, should be immediately opened and disinfected. The same active measures should be employed in the treatment of suppurating joints. In chronic cases, which are more amenable, such treatment may be effective in saving the life of the patient.

The constitutional treatment should consist mainly of easily-digested and nutritious food, given in as large quantities as the patient can appropriate, and alcoholic stimulants, administered as freely as the patient can bear. The use of antipyretic drugs—the coal-tar derivatives—is not advisable, as most of them have a depressing effect upon the heart. Quinin in large doses is sometimes advised. Carbonate of ammonium and digitalis are of service during the stage of prostration. Hygienic conditions of the surroundings of the patient are not to be overlooked; good ventilation or out-of-door air, if the patient can be moved with safety, are of great value in chronic cases. Patients in hospitals, with pyemia, should be isolated, and a strict quarantine maintained over the nurses in attendance, as a safeguard against the spread of the disease.

Pyemia dependent upon alveolar abscesses demands the immediate extraction of the offending teeth, and thorough curetting of the abscess cavity. After irrigation with antiseptic solutions, the cavity should be packed with antiseptic gauze, or iodoform gauze, and the dressing changed twice or three times during the day, until a healthy granulating surface is established, after which frequent irrigation with antiseptic solutions will be sufficient.

The constitutional symptoms should be treated upon the lines already indicated.

REVIEW.

CHAPTER XII.

Give the definition of pyemia.
What other term is applied to it?
When does it develop?
To what is it due?
With what is it attended?
What produces the metastatic abscess?
By what other symptoms is the disease characterized?
With what is it always associated?
Of what is it a grave complication?
What were the old views of the disease?
How are the metastatic abscesses formed?
What are the predisposing causes?
When does the disease usually make its appearance?
What seasons of the year predispose to the disease?
What conditions of the atmosphere seem to predispose to the disease?
How does this favor the disease?
At what age is the disease most often developed?
Which sex is most liable to the disease?
What are the reasons for this?
Is it yet positively settled which of the pus-microbes causes the disease?
What are the active causes?
How long has this fact been demonstrated?
How did Koch demonstrate it?
What were Besser's conclusions?
How and by what avenues does infection of the general system take place?
What is the effect of the micro-organisms upon the blood-vessels?
How may a thrombo-phlebitis cause metastatic abscesses?
What is a thrombus?
What is thrombosis?
What is an embolus?
What is embolism?
What term is applied to the process of plugging an artery or a capillary?
What term is applied to the area of tissue thus deprived of its blood-supply?
What is the shape of the infarct?
Which way does the apex point?
How is the area of the infarct discovered?
How does thrombo-arteritis occur?
Is it of common occurrence?
What becomes of the thrombi thus formed?
Why are they not so dangerous as thrombi formed in the veins?
How may minute emboli and micro-organisms located in the veins enter the arterial system?
What internal organs are most commonly the seat of metastatic abscesses?
Which of these organs is the most often affected?
Name the other important locations of secondary abscesses.
How may pus enter the circulation?
How does the so-called spontaneous pyemia occur?

Under what circumstances may a trifling injury cause pyemia?
Why are single organisms not likely to cause the disease?
Under what conditions may the disease develop rapidly?
What disease associated with the teeth may cause pyemia?
What is the most prominent symptom of pyemia?
At what period is this symptom made manifest?
What degree of temperature is usually associated with it?
What is the character of the fever?
Are the chills always the prominent symptom?
What is the significance of a rigor during the healing of a wound?
What is the appearance of the wound at such time?
What is the type of the fever?
How often may the chills occur?
Do they occur at regular or irregular intervals?
How does the fever differ from other forms?
What indicates the formation of secondary abscesses?
How is their formation in the lung indicated?
How is their formation in the liver indicated?
What are the indications when located in the joints?
Which of the salivary glands is sometimes affected?
What other symptoms are common to the disease?
How would an infective endocarditis be revealed?
How is infection of the spleen discovered?
Is there any means of diagnosing the disease in the kidneys?
Are metastatic abscesses of the liver common?
What is the duration of acute pyemia?
What is the duration of chronic pyemia?
What is the condition of the wound in the later stages of this disease?
What are the diagnostic signs of pyemia?
What is the prognosis of acute pyemia?
Which form of the disease has the highest rate of recoveries?
What is the prognosis of the chronic form?
What is the most important consideration in the treatment of the disease?
How may the disease be prevented?
What is the first step in the local treatment?
What is Macewen's method of treatment?
What is the value of tying the vein before operating?
Where should the ligature be placed?
What operation has been recommended when the wound is located in an extremity?
When must amputation be practiced to be successful?
How should accessible metastatic abscesses be treated?
How treat these abscesses when located in a joint?
What should be the treatment in chronic cases?
Describe the constitutional treatment.
Why are the antipyretic drugs not admissible?
What remedies are advised in the stage of prostration?
What other treatment is of value in chronic cases?
Should hospital cases be isolated, and why?
How should pyemia dependent upon alveolar abscess be treated?

CHAPTER XIII.

ERYSIPELAS.

Definition.—*Erysipelas* (from two Greek words, ἐρυθρός, red, and πέλλα, hide or skin). Sometimes called St. Anthony's fire.

Erysipelas is an infectious inflammation which primarily affects the skin.

Erysipelas is one of the most common traumatic infective diseases. It is a constitutional, specific, infective acute inflammation of rapidly-spreading type, affecting the skin, and occasionally the mucous and serous membranes. It is attended with redness, as its name indicates, swelling, often with considerable serous exudation, sometimes of a purulent character, and manifested in the form of edema, vesicles, bullæ, pustules, or by diffuse suppuration, and occasionally, in the severer types, it may be followed by gangrene and the formation of gas in the cellular tissues, from the action of the micro-organisms of putrefaction.

The origin of the disease is a micro-organism, the *Streptococcus erysipelatus*, a bacterium similar to the pus streptococcus.

The disease is characterized by a marked tendency to spread at the periphery. There is no longer any doubt of its infectious nature, as the experimental proofs upon this point are abundant, while some authorities consider it to be not only infectious but decidedly contagious. Clinical proof is not wanting to establish this fact also. The disease being of microbic origin, the infection is readily carried upon the hands of the surgeon and nurses, upon the instruments, dressings, towels, bedding, etc. This accounts for its spread in the surgical wards of hospitals, and for the infection of obstetrical cases by physicians or nurses who have been in recent contact with either of the various forms of the disease, or with suppurating wounds, and thus inducing puerperal fever. It is one of those diseases which have not been entirely eradicated from our hospitals by the introduction of antiseptic methods of treatment, although it has been rendered much less prevalent than formerly.

Erysipelas has been recognized from the earliest times, but reliable data are not obtainable farther back than the latter part of the eighteenth century. The disease has frequently appeared as an epi-

demic affection. Thus, in 1750 it was epidemic in France; in 1777 and 1800 in Great Britain, and again in the same country in 1821 and 1832. In 1842-3 it prevailed as an epidemic disease in certain portions of the United States. During 1843 it spread over Scotland, Denmark, and Germany. In these epidemics the affection was marked by a severity which does not prevail to-day. From the accounts given of the outbreak in America, it is evident that it assumed in some cases a very malignant character, while in others it resulted in extensive suppurative cellulitis, the inflammation many times involving the muscles and the bone. The epidemics of 1842-3 seem to have been the last supreme effort of the affection to maintain itself in this particular form, for there are no records since these dates of any such extensive spread of the disease, or of the prevalence of such a severe type as then occurred. Erysipelas, however, sometimes becomes epidemic in certain localities to-day, where antiseptic methods are neglected, and occasionally from vaccination, particularly with the humanized virus of kine-pox.

Causes.—That the disease is caused by a specific microbe there can no longer be any question, and this fact has been abundantly proven by experiments on animals and man. The microbe of erysipelas, as just stated, is a streptococcus which was discovered by Fehleisen, and is composed of serpentine chains made up of cocci of minute size, three to four micromillimeters in diameter. The organism multiplies by fission or division. The pus streptococcus and that of erysipelas are almost identical, the only discoverable difference being that the cocci of erysipelas are somewhat larger, while they are smaller than the staphylococci. Fehleisen succeeded in transmitting the disease from man to man by inoculation with the streptococcus which he had isolated. These inoculations were justified by being used as a therapeutic measure for the cure of lupus, rodent ulcer, and inoperable sarcomas, though the inoculations failed to cure the malignant growths. The period of incubation of erysipelas he found to be from fifteen to sixty-one hours, and when the culture with which the disease was produced was pure, it never caused suppuration. The very smallest dose did not fail to produce the disease, differing in this respect from the staphylococcus, which must be introduced in large doses. The investigations of Koch, Rosenbach, and others, in the same line, have abundantly proved the conclusions of Fehleisen as to the specific nature of the organism.

The very close relationship existing between erysipelas and puerperal fever, if not their absolute identity, was suspected long before the science of bacteriology made it possible to identify the specific cause of the disease, and of antisepsis to prevent its spread. Infection of a parturient woman with the erysipelas streptococci will result in the

establishment of puerperal fever, while cultivations from the micro-organisms of puerperal fever injected into rabbits will produce erysipelas. Dr. Oliver Wendell Holmes believed in and taught this theory long before it was possible to bring bacteriologic proof of the fact.

The most common location of the streptococci of erysipelas is in the capillary lymphatics of the skin, but they are also occasionally seen in the capillary blood-vessels and smaller veins. They are found most active and in greatest number near the margin of the red border, or "erysipelatous blush," where the lymphatics are so crowded with them as to make it difficult to discover the leucocytes. They may be found outside of the red border, or "lines of inflammation," in tissues as yet unchanged. The organisms do not spread to other parts of the body through the circulation, though they are occasionally found in the vascular current, at some distance from the seat of the inflammation, but are carried through the lymphatics of the skin. (Warren.)

The constitutional symptoms which are developed are in all probability due to the presence of ptomaines in the blood.

The disease may be acquired in two ways, viz: by direct contact or infection, and by contagion through the air, etc., as in epidemics. Billroth claims that it is most likely to occur in wounds which are chiefly discharging decomposed material mixed with blood. The micro-organism usually obtains an entrance to the system through a wound or an abrasion. In so-called idiopathic erysipelas, where no wound can be discovered, it is more than possible that the streptococci have gained an entrance through an abrasion or wound which has escaped notice, or it may be from internal infection, the organism having entered the respiratory or alimentary tract, lodged in some break of continuity in the mucous membrane, and been absorbed.

The clinical proofs of the *contagiousness of erysipelas* are so abundant that doubt can no longer exist upon this point in the mind of the observant surgeon. The disease has frequently become epidemic in certain localities as a result, or rather as a complication, of vaccination, which had for the time being to be abandoned. The relationship of erysipelas and puerperal fever has already been referred to, and that both are caused by the same type of micro-organism, if not an identical bacterium. The contagiousness of the disease has become so thoroughly recognized at the present time that if it should develop in the surgical wards of one of our best hospitals, the case would be immediately isolated, and the patients removed from the ward until it had been thoroughly cleaned and fumigated, while the nurse and the surgeon attending the isolated patient would not be permitted to come in contact with those of the surgical or obstetrical wards.

The season of the year and the state of the atmosphere are thought to have an influence upon the development of the specific organism.

Epidemics are more likely to occur during the winter and early spring months than at other seasons of the year. Bad hygienic surroundings, imperfect sewerage, and the presence of decomposing substances favor the growth and development of the organism, and may therefore be considered as predisposing causes of the affection.

It has already been stated that the most frequent point at which the virus enters the system is through wounds, and from the primary seat of the infection it spreads rapidly to the surrounding skin through the capillary lymphatics. The streptococci are chiefly found in the lymph spaces, but they are occasionally found in the capillary blood-vessels and the small veins. The appearance of the disease at distant points from the primary seat of the infection is thought by some to be clinical proof that the virus may be transmitted through the vascular circulation. The writer is of the opinion that many times this condition is the result of auto-infection through the fingers of the patient which have been contaminated with the virus; the inoculation taking place by abrading the skin in the act of scratching.

Idiopathic erysipelas was supposed to develop itself independent of any traumatic lesion, but, as already stated, it is extremely doubtful that the virus could gain an entrance to the system except through some lesion of the skin or mucous membrane. The lesion may be so slight as to have escaped notice, and yet offer an entrance sufficient for the inoculation of the system with the specific microbe.

The general symptoms which accompany idiopathic erysipelatous inflammation are of such a character as to suggest the presence in the circulation of a specific micro-organism, or of ptomaines developed by these organisms.

Symptoms.—The symptoms are an initial chill or rigor, malaise, high temperature, 103° to 104° F., nausea, vomiting, which is preceded by heavily-coated tongue, and oppression in the epigastrium; sometimes albumin in the urine, and the characteristic rash with a well-defined margin upon the skin, rose-red hue, with glazed, smooth, edematous surface, which is slightly raised, stiffness of the parts, itching or burning pain, with frequently the formation of vesicles or blebs, and enlargement of the lymphatic glands in the vicinity. The most marked characteristic of the disease is the inflammation of the skin, and its sudden disappearance from one part and its reappearance in another. As soon as the local inflammation has developed, it immediately shows signs of spreading in various directions. The center of a true erysipelatous inflammation shows more or less swelling, with diffuse redness, but its edges are marked by an irregular outline. The red hue is mingled with a yellowish tinge, which becomes more evident upon pressure (Warren); the yellow staining of the skin being very noticeable for the brief period intervening before the return of the

blood to the capillary vessels. The vesicles, which are minute in size, often coalesce to form blebs and bullæ of considerable size; these are filled with a clear, yellowish serum, which may become turbid, and not infrequently discharge a purulent material. Yellowish or brown scabs are formed by the drying up of the smaller vesicles.

The irregular outline of an advancing erysipelatous inflammation is due to the anatomical relations of the lymph-channels, through which the micro-organisms of the disease extend to the surrounding tissue.

The duration of the local inflammation or eruption is about four days. At the end of the attack desquamation takes place. The development of the disease is greatly favored by filthy surroundings, overcrowding, and defective ventilation. In severe cases of the affection the subsidence of the fever may be followed by a subnormal temperature, sometimes lasting for two or three weeks. This condition is due, probably, to the enfeebled state of the patient, as this is the only symptom of collapse.

Diagnosis.—In well-marked erysipelatous inflammation of the skin, there is no difficulty in making a diagnosis. In the early stages of the disease, however, previous to the development of the local manifestations, considerable difficulty may be experienced. The nausea, vomiting, and febrile symptoms, with enlargement of the lymphatic glands in the immediate neighborhood of the wound, if not explainable, should suggest an approaching attack of erysipelas. The most reliable diagnostic signs are the local rose-red appearance, the peculiar doughy swelling of the skin, the irregular and slightly-raised outline of the advancing inflammation, and the yellowish infiltration. Bullæ sometimes appear which are dark in color. This is the result of hemorrhage, and indicates a grave attack. (Senn.)

Prognosis.—Uncomplicated erysipelas is not a fatal disease, except when it attacks very young children, the aged, and those who have been debilitated by previous diseased conditions. The affection may, however, prove fatal, but this is usually the result of some form of complication, the most common of which are suppurative inflammation, metastatic abscesses from secondary infection with the pus-producing micro-organisms, or the extension of the disease to the meninges of the brain, as sometimes occurs when it is located upon the face or head, or the formation of secondary erysipelas in vital organs through the processes of embolism. The prognosis, therefore, must be based upon the surrounding conditions, the presence or absence of complications, and their nature and gravity. One attack does not give immunity, but seems rather to predispose to other attacks, while relapses are frequent.

Varieties.—The other varieties of erysipelas which are usually

mentioned are phlegmonous, facial, and a rare variety attacking newborn children, erysipelas neonatorum.

Phlegmonous erysipelas usually begins as the ordinary form in the skin, but later extends to the subcutaneous tissues. The extension of the disease to the deeper tissues is indicated by increased swelling of the skin, which becomes tense and hard. Blisters form, which may be filled with a bloody fluid. The swelling extends to the surrounding tissues, which may become edematous. The constitutional symptoms are more marked, and the fever assumes a typhoid character. Under these circumstances, suppuration, which is an exceedingly rare complication in the ordinary form, is quite likely to occur. When suppuration occurs it is not circumscribed, but rather assumes the character of a purulent infiltration of the subcutaneous connective tissue. The formation of pus is quite likely to be announced by rigors. The pus, when discharged, is a foul, thin, watery, discolored fluid, in which are found shreds of sloughing connective tissue. Large areas may become infiltrated, and considerable masses of connective tissue destroyed and thrown off. Numerous incisions are sometimes necessary for the proper drainage of the tissues, and for the removal of the sloughing masses. Sometimes the disease takes on a malignant type, and extends to the muscles, periosteum, and bone, causing gangrene and necrosis.

Phlegmonous cellulitis may be distinguished from *phlegmonous erysipelas* by the absence of the erysipelatous blush.

Facial erysipelas was originally considered to be idiopathic, but it is now believed to be the result of infection, like the other varieties of the disease. In all probability, the infection has occurred through some slight wound or abrasion upon the face, but which has been forgotten or unnoticed. The first manifestations of the disease are a slight redness or blush near the root of the nose, or the lachrymal duct, spreading laterally toward the ear. It rarely attacks the tip of the nose, and it is said to have a predilection for the right cheek. The attack is generally ushered in by a severe chill. The presence of enlarged lymphatic glands is considered to be characteristic. The color of the skin is scarlet red, shading to a more livid hue toward the ears. The swelling is frequently great, and edema about the eyelids is common, sometimes completely closing them and obliterating all facial expression. The spread of the disease over the face is by the characteristic irregular outline. The swelling of the nose is often so great as to close the nostrils, making all but mouth-breathing an impossibility. The swelling sometimes extends to the ears, causing impairment of hearing. The inflammation rarely affects the chin, but it may extend downward, and involve the submaxillary region. Its more frequent course is to spread toward the scalp, where, if the hair is thick, it may

escape notice. There is often, however, a considerable swelling of the neck, with enlargement of the glands, which are sensitive and painful to pressure. In the severe form of the disease the temperature will run high, ranging from 103° to 104° F., and the fever will be of the continued type, with only a slight evening increase.

The disease usually reaches its height in from four to five days, when there is a rapid decrease in the temperature. Exacerbations may occur, one or more, before the fever entirely subsides, which are due to secondary local outbreaks of the disease. Delirium is a frequent accompaniment of this form of erysipelas, and is thought to be caused either by reflex nerve action, disturbance of the vaso-motor system, or by septic intoxication. The cerebral symptoms are always aggravated in those cases where the disease involves the scalp, but no evidence of pathologic changes can be discovered in the meninges of the brain, or in the brain itself, to account for them. Delirium in and of itself is not a dangerous symptom, and it usually subsides with the fever. Occasionally suppurative meningitis may occur, but it is always caused by direct extension of the disease through the orbital fissure or the cribriform plate, when the nasal mucous membrane is involved. Abscess, when it occurs, is usually located in the orbit. There is a marked tendency of this form of the disease to involve the tissues located in this cavity. Gangrene sometimes follows the edema and extension of the eyelids. Conjunctivitis, congestion of the sclerotic, and cloudiness of the cornea are not infrequent complications, causing more or less disturbance of vision. These symptoms disappear with the subsidence of the fever and the local inflammation, though blindness sometimes results from atrophic degenerative changes.

Erysipelas neonatorum is a fatal form of the disease, occurring in newly-born children, and is very rarely seen outside the walls of lying-in hospitals. The affection bears a close connection to puerperal fever. It makes its appearance at about the time of the separation of the umbilical cord, its seat of origin being in the granulating surface of the stump of the cord. The first manifestations of the disease are a slight rise in the temperature, and a faint redness of the skin about the navel or the pubes. The inflammatory symptoms rapidly increase, the redness becomes brighter, and the subcutaneous connective tissue is swollen and indurated. By the next day the inflammation may have extended over the abdomen and to the thighs. The temperature now runs high, prostration is great; the child frets and cries, and is very restless, and finally, on the fifth or sixth day, falls into collapse and dies. In the later stages of the disease phlegmonous inflammation or gangrene may occur. The arteries and veins sometimes become involved in the inflammation, resulting in periarteritis and phlebitis.

Erysipelas of the Mucous Membrane.—Erysipelas very rarely has its primary seat in the mucous membrane. The principal anatomical location of the disease is the skin; but it not infrequently spreads to contiguous mucous membranes. Erysipelas of the face frequently spreads to the nasal mucous membrane, the pharynx, and glottis. The extension of the infection to the Schneiderian membrane may be foretold, according to Raynaud, by swelling of the lachrymal duct, through which the disease passes to this membrane. Marked enlargement of the submaxillary and cervical glands are indications of the spread of the disease to the pharynx. The patient will complain of a burning sensation in the throat, dryness, and difficulty in breathing. The color of the mucous membrane will be dark red, sometimes showing in patches, at others covering the entire throat; the swelling, which may involve the tonsils, is of a marked character. Vesicles form, and later break, and discharge a sero-purulent secretion, leaving behind little yellowish-white patches. The duration of the disease is from five to six days. It is sometimes complicated with gangrenous inflammation, and abscess which simulates retro-pharyngeal abscess. The disease may pass downward to the glottis, causing edema of this organ. It may also extend to the mouth, involving the buccal mucous membrane and the tongue, causing dark or livid redness, with extensive swelling, and sometimes the formation of vesicles. The tongue is the part which is the most frequently and most extensively involved. The swelling, which is often considerable, is due to the infiltration of the mucous membrane and inter-muscular connective tissue with liquid and migratory cells. (Ziegler.) The disease often follows other mucous tracts, like the Eustachian tubes, and passes to the auditory canal, and thence to the head and scalp. The beneficial and sometimes curative influence of erysipelas upon diseases like lupus, rodent ulcer, and inoperable sarcomas is a remarkable fact in therapeutics.

Treatment.—The treatment of erysipelas may be divided into local and constitutional, but from the nature and course of the disease it must be largely that of prevention and palliation. Isolation of the patient is the first step in the treatment of the affection, as this is the best means of preventing its spread to other persons. The disease is one of short duration, and when uncomplicated is rarely a dangerous condition. The value of specifics, either in local or constitutional treatment, is to be seriously questioned, for what has seemed in one case to be successful has in another proved a signal failure. From the nature and cause of the disease, it would seem probable that the local application of germicidal solutions, like the bichlorid of mercury, or carbolic acid, would be most efficacious, but this does not always prove to be the case; while the danger exists, in the use of bichlorid of mercury, of producing toxic conditions if the area to be

treated is large, and the solution of sufficient strength to destroy the streptococcus; while carbolic acid of sufficient strength to accomplish the same purpose would be likely to cause local irritation, and perhaps death of the tissue.

The local symptoms, particularly the burning, smarting sensations, are sometimes greatly relieved by the application of hot cloths wrung out of heated solutions of bichlorid of mercury, 1 to 3000; when larger surfaces are to be covered, a weaker solution would be advisable.

Erysipelas of the mouth, nose, and fauces may be treated with the various forms of antiseptics already mentioned, by spraying them upon the parts. Used in this manner they may control the active development of the specific micro-organism, and assist in preventing complications arising from edema of the mucous membrane.

An ointment of carbolic acid and vaselin, 1 to 100, is an excellent application to the skin in facial erysipelas. It may be applied with a soft brush, and covered with thin rubber tissue, oiled silk, or oiled paper. Zinc ointment is sometimes employed in the same manner. An ointment of ichthyol in vaselin is considered by some surgeons as almost a specific in this disease, and relieves the burning and smarting sensation.

In the phlegmonous form of the disease, free incisions for the escape of the discharges are always indicated, and should be followed by thorough irrigation. Sloughing tissue may be removed, and abscess cavities should be disinfected.

Isolation of the patient should be maintained until desquamation has ceased, as there is great danger from infection through this source during the period of convalescence. Warren thinks the tendency to relapse, which is so frequent in this disease, may find its explanation in a reinfection of the wound from the patient's own surroundings.

A favorite constitutional treatment with many is tincture fer. chlor., ♏ xx every two hours, supplemented later by quinin in tonic doses, gr. v to x daily. Milk diet and stimulants are indicated; the diet should be as full as the stomach will bear after the first symptoms have subsided.

When the temperature runs high, antipyretic drugs are demanded. Quinin seems to exert a beneficial influence over the local inflammatory process, and the general condition of the patient. When restlessness is marked, a full dose of Dover's powder may be given at bedtime. Symptoms of prostration should be early combated by some form of diffusible stimulant, alcoholic stimulants having the preference. When collapse is threatened, camphor administered every hour in doses of gr. 1½ (Senn), until symptoms of intoxication and reduction of pulse-rate to 50 or 55 are produced, may prevent a fatal termination.

REVIEW.

CHAPTER XIII.

Give the definition of erysipelas.

What is the nature of the disease?

What serious condition may follow the severer type of the disease?

Give the name of the micro-organism which causes the disease.

What is a marked characteristic of the disease?

Is it infectious or contagious, or both?

How may the disease be spread through hospitals and communities?

What influence has antiseptic treatment exercised over the prevalence of the disease?

In what form did the disease most commonly appear before the days of antiseptics?

When was the last great epidemic in America and certain European countries?

What was the character of the disease at that time?

Under what circumstances does it become locally epidemic to-day?

What are the proofs that erysipelas is caused by a specific microbe?

Describe the Streptococcus erysipelas.

Who discovered the organism?

What is the difference between this organism and the pus streptococcus?

What is the incubation period of the disease?

Does a pure culture of the Streptococcus erysipelatus ever cause suppuration?

By whom have Fehleisen's observations been confirmed?

What relationship exists between erysipelas and puerperal fever?

Where are the streptococci of erysipelas most commonly located?

Where are they found most active and in greatest numbers?

In what other location are they sometimes found?

By what channels do they spread to other parts of the body?

What is the cause of the constitutional symptoms of the disease?

In what ways may the disease be acquired?

In what class of wounds is the disease most likely to occur?

By what means do the micro-organisms usually obtain an entrance to the system?

How is the so-called idiopathic erysipelas accounted for?

By what means is contagion prevented in the surgical wards of hospitals?

At what seasons of the year, etc., is the disease most likely to appear?

What other conditions predispose to the disease?

What is thought to be a clinical proof that the disease is spread by the vascular circulation?

What other explanation is offered upon this point?

What do the constitutional symptoms of idiopathic erysipelas indicate?

Describe the symptoms of erysipelas.

What is the most marked feature of the disease?

How does the local inflammation extend?

By what signs may an erysipelatous swelling be distinguished?

Describe the vesicles, the character of the discharge, etc.

What controls the irregular outline of the advancing erysipelatous inflammation?

What is the duration of the local inflammation?

What takes place at the end of that time?

How is the temperature affected after the fever subsides in some severe cases?

In what stage of the disease is diagnosis somewhat difficult?

What are the diagnostic signs of the disease?

Is uncomplicated erysipelas a fatal disease?

Under what conditions may the disease prove fatal?

Upon what should the prognosis be based?

Does a previous attack give immunity or predispose to other attacks?

What is the tendency to relapse?

Name the other varieties of erysipelas.

How does phlegmonous erysipelas differ from the common form?

What is the character of the constitutional symptoms?

What type of suppuration accompanies this form of the disease?

What is the character of the pus?

When the disease is of a malignant type, to what other tissues does it extend?

How may phlegmonous cellulitis be distinguished from the phlegmonous form of erysipelas?

What is the origin of facial erysipelas?

How does the infection occur?

What are the first manifestations of the affection?

What condition is considered characteristic of the disease?

Describe the symptoms of the disease and its manner of extension.

What is the temperature in the severe form?

How long does the disease run before reaching its height?

What causes the exacerbations in the fever?

What is thought to be the cause of the delirium?

Under what circumstances is it aggravated?

Is delirium a dangerous symptom?

When suppurative meningitis occurs, what is the cause?

Where are abscesses most often located?

What other complications may arise in the tissues of the orbital cavity?

Are these conditions permanent?

What changes may take place resulting in blindness?

Describe erysipelas neonatorum.

Where is it located?

What are the first manifestations of the disease?

What is the character of the inflammation?

How does the disease terminate?

What are the complications in the later stages of the disease?

Does erysipelas ever develop primarily in the mucous membrane?

How does the mucous membrane usually become affected?

What is the most frequent location in the mucous membrane?

By what symptoms may the extension of the disease to the Schneiderian membrane be foretold?

What symptom indicates its extension to the pharynx?

What other symptoms are indicative of the extension to the throat?

What is the duration of the disease?

With what conditions is it sometimes complicated?

Does the disease ever extend below the pharynx?
What are the manifestations in the mouth?
How is the tongue affected?
What other mucous tracts does it sometimes follow?
What is the curative value of erysipelas?
How is the treatment of erysipelas divided?
From the nature of the disease, what must the treatment largely consist of?
What is the first step in the treatment?
Are specifics of universal value in the disease?
What is the danger in the use of germicidal agents?
How are the local symptoms to be treated?
What line of treatment is to be adopted when the disease is located in the mouth, nose, and fauces?
What applications are recommended for facial erysipelas?
How should the phlegmonous variety be treated?
How long should isolation be maintained?
How may relapses be accounted for?
What is a favorite constitutional treatment?
What diet should be prescribed?
How may the high temperature be controlled?
How control the restlessness?
By what means combat symptoms of prostration?
What treatment is recommended in threatened collapse?

CHAPTER XIV.

TETANUS.

Definition.—*Tetanus* (from the Greek τέτανος, τείνειν,—to stretch). Tetanus is a disease causing spasmodic and tonic contraction of muscles, producing rigidity in the parts to which they are attached. It is an infectious disease, in which a specific micro-organism exerts a pathogenic action upon the central nervous system, probably through the production of a chemical poison or ferment.

The wound-infective diseases in which micro-organisms or their ptomaines act upon the central nervous system, are represented by tetanus and hydrophobia. (Senn.) These diseases are caused by specific microbes, and while they produce no pathological changes of a gross character in the brain or spinal cord, minute tissue-changes, like hyperemia, take place, causing a central irritation, which is made manifest by tonic spasm of certain definite groups of muscles.

The infectious nature of the disease had long been suspected, but it is only very recently that its true nature has been demonstrated by the discovery of the *Bacillus tetani*. The honor of the discovery belongs jointly to Rosenbach and Nicolaier. Nicolaier found a bacillus in earth which, when injected into the tissues of animals, produced tetanus. Rosenbach found the same bacillus in pus taken from animals suffering from traumatic tetanus, and on April 10, 1887, it was demonstrated by Koch that the bacillus of Nicolaier and that of Rosenbach were one and the same.

Rosenbach describes the *Bacillus tetani* as anaërobic and in form a bristle-like organism, with a spore at one end of it, which gives it the appearance of a drum-stick.

This micro-organism is rapid in its growth, producing spores in thirty hours in cultures which are kept at a temperature equaling that of the human body. They possess great resistance to heat, as they have been found active after having been subjected to a moist heat of 80° C.=176° F. for one hour. When subjected to a heat of 100° C.=212° F., for five minutes, it was found that they were destroyed.

This bacillus is so common that it has been found in many kinds of garden and top soils, in street dust, sweepings of dwellings, in old masonry, in putrefying fluids and manure.

Causes.—The causes may be divided into predisposing and exciting.

The *Predisposing* causes are hot climate, exposure to a cold and damp atmosphere, sudden changes of temperature, lacerated and punctured wounds, burns, frost-bites, and all septic wounds. Wounds of the extremities are more frequently followed by tetanus than those of other parts of the body. The negro race is much more susceptible to the disease than the white.

The *Exciting* cause is a micro-organism of marked anaërobic type, the *Bacillus tetani*, which acts upon the central nervous system through the elaboration of ptomaines and their poisonous effects.

Period of Incubation.—The period of incubation is exceedingly variable, both in man and in animals. The symptoms of the disease may develop in twenty-four hours, or not for several weeks. Out of 367 cases reported in the "Surgical History of the War," 287 occurred during the first two weeks after the injury. Yandell found that of 415 cases, the histories of which he had gathered, the disease developed in 196 cases during the first two weeks. This variation in the period of incubation may depend upon the number of the bacilli introduced at the time of the infection; the character of the tissues at the point of infection, which may be favorable or unfavorable to their development; or to the vital resistance of the individual to the action of the ptomaines produced by the bacilli.

Forms of the Disease.—The disease develops in two forms, namely: acute and chronic, and they are always of traumatic origin. The old idea that tetanus in some cases might be of idiopathic origin is now no longer tenable, as the latest investigations have proved conclusively that the disease is caused by a specific micro-organism which gains access to the circulation, and produces its toxic effects upon the central nervous system through the formation of ptomaines or a chemical ferment. The development of so-called idiopathic tetanus can be easily explained by the fact that a slight wound or abrasion upon the surface of the body is sufficient to permit the entrance of the micro-organisms; but this may be so trivial as to escape the notice of the individual. It is also not improbable that infection may occur through the respiratory mucous membrane, or the alimentary tract, from the inspiration of dust, or the swallowing of food contaminated with the organisms.

Wounds of the hands and feet are much more liable to result in tetanus than wounds in other locations, because the instruments or substances inflicting these wounds are very liable to be contaminated with infected earth or dust. They are also, from neglect, frequently subjected to subsequent infection, or infected foreign bodies are allowed to remain in the wound. The disease is said to have followed

a simple fracture. Under such circumstances the infection must have gained an entrance through some unnoticed abrasion or wound, or by internal infection.

Tetanus may follow the extraction of teeth, the infection coming from without,—or it may follow from the infection of the wound by uncleanly instruments; it may also follow the transplantation of teeth, where the operation is not performed under antiseptic precautions. John Hunter, who, more than a hundred years ago, invented the operation of the transplantation of teeth, finally discarded it for the reason that tetanus was liable to follow operations of this character. The use of antiseptics has largely removed this danger, and when performed under the strictest methods obtainable in the mouth, it is now considered a safe operation. Hunter's difficulty lay in two facts: first, that he knew nothing of the use of antiseptics; and second, that he did not appreciate or did not know the necessity of removing the pulp of the tooth, and hermetically sealing the pulp-canal with some indestructible filling-material. Had he done this, he would have had fewer cases of alveolar abscess and less danger of tetanus following the operation.

Acute Tetanus.—This form of the disease usually appears during the first and second weeks after the injury, and reaches its height on the third or fourth day. The affection is characterized, in even the most acute cases, by its insidious approach, and by the absence of all recognizable constitutional symptoms in its early stages. The first manifestation of the disease is usually a sense of having taken cold, followed by stiffness and soreness of the muscles of the neck and jaws. The development of the disease is very rapid from this time, and it may terminate fatally in from four to five days. In tropical climates it develops much more rapidly, and cases have been reported in which death followed in a few hours after the first symptoms were discovered. When the symptoms are prolonged beyond the fifth day, there is hope that the disease will assume the chronic or milder form, and that recovery will ultimately take place.

One of the most common forms of the disease is that known as trismus, or lock-jaw. Trismus (from the Greek τρισμός, τρίζειν—to gnash) is a tonic spasm of the muscles of mastication, which firmly fixes the jaws, making it impossible for the sufferer to open the mouth.

A very fatal form of tetanus occasionally occurs in infants during the first five or six days of life. It is marked by frequent convulsions, occurring in paroxysms, with rigidity of the muscles of the body, and special involvement of the muscles of the jaws, mouth, and throat, producing inability to swallow. This form of the disease is, in all probability, caused by infection at the time of birth, or immediately afterward, through the wound at the umbilicus.

The muscles of mastication are the first group to be affected by the action of the ptomaines of the bacillus upon the central nervous system. They are also, usually, the most markedly affected, and for this reason the term lock-jaw has become synonymous for tetanus.

Another condition known as *Opisthotonos* (from the Greek ὀπισθεν, *behind*, and τόνος, *tone* or *tension*) is a tetanic condition of the muscles, especially of the back, which causes an arching of the trunk backward, so that the only point of contact of the body would be upon the head and heels; or, in other words, when lying upon the back, the body rests upon the head and heels during the spasm. The same condition is characteristic of strychnia poisoning, hydrophobia, hysteria, and other tetanic affections. Occasionally the trunk is arched forward. This condition is termed *Emprosthotonos* (Greek ἐμπροσθεν, forward, and τόνος, tension). At other times the body may be curved or bent upon one side—curved laterally—in a tetanic convulsion. This is termed *Pleurosthotonos* (Greek πλευροθεν, from the side, and γεινειν, to bend).

Symptoms.—The commencement of the disease is marked by chilliness, slight stiffness of the muscles and jaws, accompanied by gradually increasing pain, and the appearance of a sanious or ichorous discharge at the wound or the point of primary infection. The muscles of deglutition become affected, and the individual experiences more or less difficulty in swallowing. The countenance presents a peculiar grinning expression (*risus sardonicus*). Later, all of the voluntary muscles, including those of respiration, may become involved. The affected muscles are rigid from tonic spasm, but this condition is increased by frequently recurring clonic spasms.

In severe acute cases the temperature soon rises to 102° or 104° F., with a corresponding acceleration of the pulse. The difference between the morning and evening temperatures is very slight. The mental faculties remain clear during the entire course of the disease. The salivary secretions are increased, and owing to the inability of the patient to swallow the fluid, or expectorate, it escapes from the mouth. Respiration is affected in proportion to the extent that the respiratory muscles are involved. In severe cases dyspnea and cyanosis are early manifestations. The special senses are not affected. The pain is usually excruciating, and extends along the track of the nerves supplying special groups of muscles, and it is increased by the tonic spasms, or by external irritants. A draft of cold air, an unexpected noise, or an effort to swallow, is in some cases sufficient to produce a paroxysm. On account of the difficulty of taking sufficient food, the loss of sleep, and the intense pain, emaciation and loss of strength are early manifestations. As the condition of exhaustion becomes more marked there is profuse, clammy perspiration, coldness of extremities, and

feeble, rapid pulse. Finally the intercostal muscles are affected, and the diaphragm is thrown into tonic spasm, the respirations and pulse cease, and death supervenes. The temperature just before death may rise to 108°, to 110°, or even 113° F.

Diagnosis.—The diagnostic signs of tetanus are the absence of fever in the early stages of the disease, the tonic character of the spasm, the early manifestations of spasmodic contraction of the muscles of mastication and of the neck, the convulsive character of the attacks, and the clearness of the intellectual faculties to the last.

The *differential diagnosis* would take into consideration the stiffness of the jaws arising from inflammatory conditions of the mouth, abscesses associated with devitalized teeth, impacted third molars, suppurative inflammation of the cervical gland, inflammation of the parotid gland, rheumatic conditions of the temporo-maxillary articulations, and reflex muscular contractions due to peripheral irritation of the nervous system. Many of the symptoms of strychnia poisoning and of hydrophobia simulate those of tetanus, and might mislead one who was not thoroughly familiar with all the symptoms of each of these affections.

Chronic Tetanus.—This form of the disease usually appears at a later date than the acute form, generally two to three weeks after the injury, and although the symptoms may be very severe and the development of the disease rapid, there will be periods when the patient will be free from the spasmodic muscular contractions, sometimes for an entire day. During these periods the patient can take nourishment, and his strength is correspondingly sustained. The intervals between the convulsive seizures gradually become longer, and the paroxysms less and less severe. After convalescence sets in, relapses may occur and thus prolong the date of recovery.

Prognosis.—The prognosis depends largely upon the character of the disease. The more acute and intense the symptoms, the greater is the danger of a fatal termination. Tetanus following wounds received in battle is much more fatal than that following other wounds. Out of 505 cases recorded during the Civil War, 451 died, or 89.3 per cent. In those cases which are prolonged beyond two weeks, the chances for recovery are good. The general mortality of the disease is about seventy-five in every one hundred. The chronic cases, as a rule, recover after an illness ranging from six to ten weeks.

Treatment.—The antiseptic treatment of all wounds, no matter how small and insignificant they may appear, is imperative as a prophylaxis.

Tetanus follows more frequently upon injuries of an insignificant nature than extensive wounds, or amputations, for the reason that the minute lesions are overlooked, or considered of little or no importance;

while on the contrary, these lesions should receive the greatest care, and be treated in accordance with the strictest methods of antisepsis.

Old wounds should be opened, and diligent search made for foreign substances; and recent traumatisms should be carefully examined, and treated in the most thorough antiseptic manner.

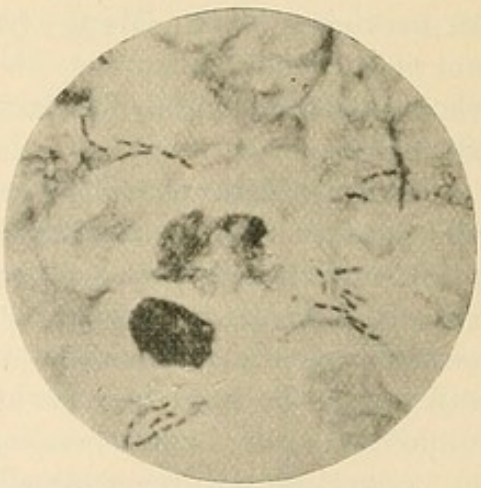
In severe cases palliation is the only treatment that can be instituted, as the immediate symptoms are always urgent. The excruciating pain and spasm of the muscles can generally be relieved only by the inhalation of chloroform. Its administration should be conducted by a competent person, and carried only to the point of muscular relaxation. Morphia, one-quarter to one-half grain, combined with one two-hundredth grain of atropia, is sometimes of benefit; it should be given hypodermically.

FIG. 36.



DIPHTHERIA OR KLEBS-LŒFFLER BACILLUS. × 1200.

FIG. 37.



PSEUDO-DIPHTHERIA. × 1200.

In milder cases chloral hydrate and potassic bromid, fifteen to twenty grains, may be given by the stomach with good effect.

It is imperative that all patients suffering from tetanus should be kept in a quiet and dark room, and free from all forms of excitement, as absolute quiet of mind and body is an important element in the treatment.

In severe cases in which swallowing is impossible, food in a liquid form can be introduced into the stomach by means of a small elastic tube passed through one of the nostrils into the œsophagus; the food should be given at regular intervals. Surgical treatment, by means of nerve-section and nerve-stretching, gives no better results than internal medication.

Some startling reports have recently appeared in the home and foreign medical journals in reference to the blood-serum and antitoxic treatment in tetanus and diphtheria.

The mortality of tetanus, according to the recent statistics of

Richter and Forgues-Reclus, is about 88 per cent.,—this is about 13 per cent. higher than the generally accepted mortality,—but it is claimed that by the new treatment this frightful mortality has been reduced to 20 per cent., with the prospect of a still further reduction.

In diphtheria it is more difficult to arrive at a fair estimate of the value of the treatment, on account of the great variance in the virulence of the various epidemics of the disease. Kossel reported 233 cases treated by this method, with 179 recoveries, which is equal to 77 per cent. This number included 72 cases upon whom tracheotomy had been performed, 41 of whom recovered, making a percentage of 57 as against 25 in former years.

Diphtheria, like tetanus, is dependent upon the action of a specific micro-organism which has gained access to the body through infection or inoculation, and the production and absorption by the system of a toxic substance elaborated by the micro-organisms. But there is a possibility that pseudo-diphtheria may be mistaken for the more serious form of the malady, and thus render the statistics of little value. Figs. 36 and 37 represent the difference in the size and grouping between the Klebs-Lœffler bacillus (true diphtheria) and the bacillus of pseudo-diphtheria. A positive diagnosis cannot be made except by a bacteriologic examination.

The principle upon which the new treatment is founded dates back to the discovery of Jenner, that vaccination with kine-pox would render the human subject immune to the virus of smallpox. Jenner, however, had little or no conception of the great scientific principle which he discovered; it remained for Pasteur, Chauveau, Behring, and others to discover why and how inoculation with the vaccine virus—the introduction, in specified quantities, of the products of certain specific micro-organisms into the system of healthy individuals—would protect them from the disease caused by the particular microbe; and in those already suffering from the disease in its earlier stages, would shorten the duration of the attack and greatly reduce its virulence.

If confidence can be placed in these reports, it would seem that the science of medicine was upon the threshold of the greatest revolution in practice that the world has ever seen, for the possibilities of this line of treatment seem to be almost limitless.

With the marvelous achievements of vaccination before us,—the mortality from smallpox in England, before the introduction of vaccination, was 21.2 per cent., while now under the law which requires that every child born in England shall be vaccinated before it is three months old, the mortality has been reduced to 0.67 per cent.,—what may we not hope for in the future in relation to the terrible scourges of diphtheria, phthisis, cholera, etc., which annually destroy so many lives?

REVIEW.

CHAPTER XIV.

What is the definition of tetanus?
By what is it produced?
What effect do the micro-organisms have upon the central nervous system?
Who discovered the bacillus tetani?
Describe its appearance.
How long does it take to mature?
How is it affected by high degrees of heat?
What temperature is necessary to destroy it?
How long must it be subjected to this temperature?
Where is the bacillus most commonly found?
How are the causes classed?
Name the predisposing causes.
What portions of the body are most liable to be the seat of infection?
Which race is most susceptible to the disease?
What is the exciting cause?
What can be said of the period of incubation?
State the usual period of incubation.
To what may this difference be due?
In what forms may the disease appear?
Is the disease of traumatic or idiopathic origin?
What is the proof that the disease is never of idiopathic origin?
How can the so-called idiopathic tetanus be produced?
Through what other avenues may the micro-organisms enter the system?
Why are wounds of the hands and feet more liable to result in tetanus than wounds in other locations?
By what means may infection take place in the extraction of teeth?
How may the disease be guarded against in the transplantation of teeth and other like operations?
At what period after an injury does acute tetanus develop?
How soon does it reach its height?
What are the characteristics of the onset of the disease?
How is the disease usually first manifested?
At what period may a fatal termination be expected?
What is the prognosis if the disease is prolonged beyond the fifth day?
What form may it take after this period?
What is trismus?
How are infants affected by it?
How are infants infected?
What group of muscles is first to be affected?
What is opisthotonos?
In what other diseases is this condition frequently noticed?
What is emprosthotonos?
What is pleurosthotonos?
What injuries are most likely to result in tetanus, and why?
What are the symptoms of tetanus?
Give the diagnostic signs of the disease.
What facts are to be considered in the differential diagnosis?

When does chronic tetanus appear?

How does it differ from the acute form?

What is the prognosis?

What class of injuries gives the largest per cent. of fatalities from tetanus?

What is the mortality?

How long do chronic cases usually run?

What is the local treatment?

How are the immediate constitutional symptoms to be controlled?

What should be the surroundings of such patients?

In case swallowing is impossible, how should the patient be fed?

Is there any advantage in surgical treatment?

What is the latest treatment for tetanus and diphtheria?

What is the cause of diphtheria?

Upon what discovery is the blood-serum therapy or the antitoxin treatment based?

What seem to be the possibilities of this form of treatment?

CHAPTER XV.

SHOCK AND COLLAPSE.

Shock.

Definition.—*Shock* (comes from the Saxon, meaning to shake—to shake with violence).

"Shock is a lowering of the vital powers, the depression or grave effects produced by severe injuries, operations, or profound mental impressions."

Shock is "a relaxation or abolition of the sustaining and controlling influences which the nervous system exercises over the vital organic functions of the body, and is the result of a profound impression made upon the cerebro-spinal axis, either directly, through the agency of an afferent nerve, or through the circulatory medium. It is a vaso-motor paralysis, affecting also the heart, and chiefly the abdominal vessels. It may also be described as a "sudden or instantaneous depression of organic, nervous, or vital power, often with more or less perturbation of body and mind, passing either into reaction or into fatal sinking,—collapse,—and occasioned by the nature, severity, or extent of an injury, or by overwhelming moral calamity.

"A condition of sudden depression of the whole functions of the body, due to powerful impressions upon the system by physical injury or mental emotion. Its more obvious manifestations are signs of lowered activity of the cardiac, respiratory, and sensorial functions, and reduction of surface temperature." (Sir William MacCormac.)

Collapse.

Collapse (comes from the Latin *collapsus*, fallen together,—to fall in ruins;—"Medically, a general prostration or failure of the vital powers,—a complete prostration of strength, either at the commencement or in the progress of a disease").

"Collapse is a state of nervous prostration. When it is extreme, the vital functions are in a condition of partial, and sometimes nearly complete, abeyance. It may terminate in death, or be followed by general reaction and complete recovery.

"Collapse and shock have usually been classed together, but it is not accurate to do so. It is true that the ganglionic centers of the

medulla oblongata are more or less profoundly involved in both, and that both possess many symptoms in common, dependent upon the derangement of function of one or more of these centers. Some confusion is attributable to the fact that shock is a term applied not only to a state or morbid condition, but the cause which most frequently produces that condition,—a violent impression or shock to the nervous centers. Collapse arises from many different causes, shock being one, of which collapse may be regarded as a final and extreme degree, and into which it often imperceptibly passes. Collapse, on the other hand, may occur under conditions where there has been no antecedent state of shock. Collapse presupposes previous nervous exhaustion, while shock may instantly appear in a healthy individual." (Sir William MacCormac.)

Shock, therefore, may appear instantaneously in healthy individuals, from fright, extreme mental emotion, traumatic injury, or surgical operation; the depression of vital force corresponding to the severity of the impression made upon the nerve-centers, the tendency being generally toward reaction, which may take place in a few minutes, or be delayed for several hours, while in severe cases it may terminate fatally.

Collapse, on the other hand, generally manifests itself in persons who have undergone previous prolonged nervous strain, which has caused exhaustion. The system is therefore at a disadvantage, if called upon to sustain the debilitating effects of disease, or any of the emotional, traumatic, or surgical conditions just mentioned. The depression of vital force is more prolonged and extreme, depending upon the strength of the individual and the character and degree of the exciting cause. Reaction sets in less quickly, and complete recovery may require weeks or months for its accomplishment.

Collapse, in its most extreme form, following cholera, yellow fever, or shock from severe accident or surgical injury, generally proves fatal. Shock with profuse hemorrhage often causes fatal collapse.

In the normal condition of the system there is a very great difference in the nervous excitability and physical endurance of individuals, while in the same person, under abnormal conditions, the individual peculiarities are accentuated. All are less able to endure the shock of accident or surgical injury, the extremes of joy or grief, if the physical or mental conditions are below the normal. Women, taken as a class, are less susceptible to shock than men; temperament also exerts a modifying influence in shock. The phlegmatic and lymphatic temperaments do not possess the same susceptibility to shock as is found in the sanguine and nervous. Age is also a strong modifying factor in shock. Children bear injuries well, and quickly rally from shock,

when the injury has not been accompanied with much loss of blood. In the aged, on account of the presence of organic diseases, or debility, shock is often of a severe and prolonged character.

Pathology.—Very little is known of the pathology of shock, as it is one of those conditions which cannot be demonstrated by the ordinary methods of research. In persons who have died from shock, it is impossible to discover any pathologic changes to have taken place in the tissues. The principal phenomena of shock are those which occur or manifest themselves through the agency of the nervous system. Goltz discovered a marked distention of the intra-abdominal veins in certain cases of shock. Experimental physiology has demonstrated beyond a doubt that in shock there is a *reflex paralysis* of the heart and abdominal vessels, brought about through the medium of the vaso-motor system. All the symptoms of shock are of such a nature as would indicate a more or less severe paralysis of the heart and vessels, with the accompanying impression upon the nervous system and the brain.

Symptoms.—The symptoms of shock comprehend those of depression of the vital forces, which may be slight or grave, according to the extent of the injury or the force of the mental impression, and the nervous susceptibility of the individual. The most striking symptoms of shock are the contracted, pinched expression of the features; the sickly-white hue of the skin; the thin, pale lips; the sunken appearance of the eyes; the cold, clammy feeling of the surface of the body; the feeble, rapid pulse; the shallow and irregular respiration; the subnormal temperature; the general relaxation of the muscular system, and loss of control of the sphincters, causing involuntary evacuations, while consciousness and the special senses are often considerably blunted; the sensation of pain is more or less diminished, and nausea and vomiting frequently occur. Reaction may take place in a few hours, or not for twenty-four hours, or the condition of shock may terminate in collapse and the patient die from syncope or asthenia.

Normal reaction is characterized by an increase in the strength of the heart-action, with diminution in the rapidity of its pulsation, a rise in the body temperature, return of the natural color of the skin, and a re-establishment of full, deep inspirations. Another form of reaction—abnormal—sometimes takes place, in which acute fever is developed, with flushed face, bloodshot eyes, high temperature, extreme restlessness, rapid, throbbing pulse, and delirium. The pulse, however, does not possess the characteristics of high fever, as it is soft and compressible. The tongue is dry, and the general condition one of great weakness and prostration. This form of shock is very liable to lapse into profound shock and fatal collapse.

Prognosis.—The prognosis of shock will depend very largely

upon the severity of the injury, or extent of the mental impression, and the reactive powers of the individual; but the outcome is always uncertain and doubtful. Shock may prove fatal in the brief space of a few seconds, or the individual may not succumb for twenty-four to forty-eight hours. The prognosis of shock is considered unfavorable if reaction does not set in within eighteen to twenty-four hours, or if the temperature falls below 96° F. Such a condition contra-indicates any surgical operation. Another particularly unfavorable symptom is the loss of the power of swallowing. Loss of sensation in the conjunctiva, persistent vomiting, and relaxation of the sphincter muscles are also considered as fatal symptoms.

Treatment.—In those cases of shock which may be classed as mild, very little in the line of treatment is required beyond that of keeping the patient warm and quiet, and allowing nature to restore the equilibrium of the circulation and the forces of the nervous system. Occasionally a little stimulation may be advantageously employed, but generally the administration of drugs under these circumstances is more harmful than beneficial. In the severer cases of shock, the measures employed should be of such a nature as will tend to stimulate the vital forces, which at such a time are at an exceedingly low ebb. The principal indications or requirements in the treatment of shock are the preservation and supply of heat to the body; to stimulate the feeble action of the heart, to allay restlessness, to control the hemorrhage and other complications as they may arise. If shock supervenes during a surgical operation, the operation should be terminated as speedily as possible, or it may even be better to suspend it altogether. Better this than that the life of the patient should be sacrificed to a prolongation of the operation.

Persons suffering from shock should be placed in a comfortable position upon the back; the foot of the bed elevated so as to bring the head lowest, that the weak heart may be called upon to expend the least possible amount of energy in forcing the blood to the exhausted vital nerve-centers. Rest and quiet are of the utmost importance, and, after the patient has been placed in bed, he should remain there without disturbance until reaction has set in. Next of importance to undisturbed rest is the application of heat to the body. This may be accomplished by placing about the extremities and side of the body of the patient bottles filled with hot water; hot bricks or other suitable means can be used for the same purpose, care being taken to protect the patient from being burned with the hot appliances. The artificial heat should be confined by covering the patient with blankets, and over these a rubber sheet.

In severe cases of shock resulting from accident, the patient should be moved as little as possible, while the clothing should not be

removed unless it is wet or some other cause imperatively demands it. Unnecessary disturbance under such circumstances, even to clean up the patient, is productive of more harm than good, while the examination of the medical attendant should be as gentle and quiet as possible, for the same reason. The necessary manipulations for the control of the hemorrhage, and the care of the wounds, should be done in the most expeditious manner possible consistent with the best interests of the patient at the time, and for his future well-being.

In shock and collapse resulting from severe hemorrhage, transfusion of blood was at one time extensively practiced. This treatment, of late years, has been practically abandoned, on account of the difficulties and dangers which surrounded it. Intravenous infusion or injection of warm salt solutions is now practiced to a considerable extent, and with quite as good, if not better, results. The chief advantages of a saline solution are that it can always be procured in a few minutes, and that it can be introduced with the ordinary fountain, or other good syringe which will carry a continuous stream, and permit of being charged while in position.

Mikulicz suggested the following formula:

Sodii chlorid., ʒjss;
Sodii bicarb., gr. xv;
Aq. dest., Oij.—M.

The temperature of the solution should be 100° F.

It may be introduced into the median cephalic vein, or into the subcutaneous connective tissue of the abdominal wall. The injection should be made slowly, and care taken that air is not introduced with it. The quantity which may be safely injected will vary from one to four pints. The pulse and respirations are to be carefully noted during the injection.

Patients suffering from shock from hemorrhage are inclined to drink large quantities of water. This is nature's method of supplying the deficiency of fluid at the vital centers. There can be no objection, therefore, to its use, provided the stomach is in a condition to retain it. In irritable conditions of the stomach, where the water is not borne well, recourse may be had to rectal injections of weak salt solutions at a temperature of 100° F. A quart or more may be injected at a time, and repeated in half an hour, for absorption goes on with amazing rapidity in the lower bowel of persons whose systems have been depleted by the loss of large quantities of blood.

Diffusible stimulants are of great value in cases of shock, and may be administered as the indications suggest, either by the stomach or hypodermically. Whiskey or brandy are the best for this purpose, and may be injected in doses from fl ʒss to fl ʒj, and repeated every

ten or fifteen minutes until a decided stimulant effect is produced upon the heart-action, as manifested in the improvement of the pulse. The heart's action may be further strengthened and the nausea relieved by the administration of black coffee in small and frequent doses, either alone or in combination with brandy. The aromatic spirit of ammonia is also a valuable diffusive stimulant, and is generally well borne by the stomach, dose gtt. xxv to fl ʒj, in water.

Opium in small doses, administered by hypodermic injections or by rectal injection, is often recommended for its stimulating action, and for the quieting influence which it exerts at a time when repose is of so much importance. Digitalis, alone or combined with atropin, is often administered in those cases in which collapse is threatened. Digitalis is a powerful cardiac restorative, but it is not well borne by the stomach, hence it is best administered by hypodermic injection. The dose of the tincture of digitalis is gtt. xv. This may be repeated every fifteen or twenty minutes until the pulse shows decided improvement, but not more than three or four consecutive doses should be administered. The first dose only should be combined with atropin; one one-hundredth of a grain is the usual dose.

Nitro-glycerin in doses of one two-hundredths of a grain is often of service in those cases where digitalis fails to stimulate the flagging action of the heart.

Strychnia in doses of one-fiftieth to one-thirtieth of a grain is highly recommended by some authorities in such emergencies.

As soon as reaction begins the stimulant should be gradually withdrawn, but the pulse should be carefully watched in the mean time, and, if signs of relapse appear, the stimulant should be immediately renewed, and, as soon as indications warrant, again gradually withdrawn. The means which have been employed to restore the normal temperature of the body may be removed as soon as the patient complains of discomfort from their use.

Shock from Dental Operations.—Prolonged and painful dental operations commonly produce considerable depression of the vital powers. In individuals already in a more or less exhausted condition from disease, overwork of mind or body, or emotional causes, and occasionally in chlorotic girls, pregnant women, or those suffering from uterine or ovarian diseases, and in frail, delicate children, they may produce alarming symptoms of shock. It is therefore, in view of the foregoing facts, as much the duty of the dental surgeon to look carefully into the general condition of health of those who present themselves for dental operations, as it is for the general surgeon to do so before commencing any surgical procedure. Only in extreme cases where immediate operation seems necessary, as the only chance of preserving life, will the intelligent surgeon permit himself to operate upon

individuals whose physical condition, as in shock and collapse resulting from hemorrhage, strangulated hernia, perforation of the bowel from traumatism, certain brain injuries, etc., or where the general health is greatly impaired, without first making the attempt to stimulate the flagging energies of the heart or to recuperate the depressed vitality, and place the patient in the best possible condition for the operation.

Failure to properly appreciate the necessity of such treatment tends to disappointment or disaster, of greater or less moment, either in the success of the operation, the after-effects upon the system, or the life of the patient.

There are but few *strictly* dental diseases which, *per se*, are dangerous to life in the otherwise healthy individual, and few operations upon the teeth, or immediately associated tissues, which can in any sense be considered dangerous to the same class of persons; and yet, the presence of an alveolar abscess, or the extraction of a tooth, occasionally proves fatal, either from septic infection, a peculiarity in diathesis,—hemorrhagic,—or some pre-existent morbid tendency which predisposes to a fatal termination.

On the other hand, painful or prolonged operations upon the teeth, like the preparation of a series of exceedingly sensitive cavities of decay, or restoring the contour of the teeth with gold, is very depressing and irritating to the nervous system, and fatal results may follow such operation in consequence of reflex impressions made through the fifth nerve upon vital ganglia.

The dentist, unfortunately, is usually obliged to perform these operations without the benefit of any of the anesthetics. The nature of the operations, the necessarily upright position of the patient, the considerable amount of time required to complete them, and the difficulties encountered in the maintenance of anesthesia preclude the use of these agents; consequently shock and collapse, in various degrees, are often the sequel of the severe strain upon the nervous system to which the individual has been subjected. It is not at all uncommon for strong, robust men to develop various symptoms of shock after sitting for two or three hours under such manipulations.

Children especially should not be compelled to submit to the fatigue and nervous strain of long and painful operations, particularly during the period of rapid growth, puberty, and just preceding it; while engaged in severe study, or just after a prolonged illness. At these periods the nervous system is generally taxed to its utmost limit of endurance. When crowded beyond this point, the health gives way, and often, when too late, it is discovered that the foundation has been laid for a train of nervous affections, chorea, etc., which persist to the end of life.

The rapid movement of the teeth by the various methods employed in orthodontia is often a serious and dangerous operation, on account of the severe and constant irritating influences upon the nerve-centers. It would be much better to forego the attempt to improve the personal appearance of the child than to destroy the health.

Chlorotic girls and women suffering from uterine and ovarian diseases in their manifold forms, or during gestation and the functional changes which establish the menopause, are usually in a more or less exalted state of nervous irritability, and the nerve-centers seem to be more easily impressed, while reflex disturbances are common. As a consequence, dental operations of any kind are not well borne by such individuals.

When the operations are of a prolonged and painful character, they should not be undertaken unless they can be performed with the help of anodynes or of anesthetics. In most cases it would be far better to adopt palliative and temporary measures until such time as the health is restored, rather than by heroic treatment to run the risk of nervous exhaustion or more serious consequences. Miscarriage and other serious affections are not unheard of as *sequelæ* of heroic dental operations.

Shock from the extraction of teeth is often severe, sometimes inducing alarming symptoms; syncope is the most common, but this is frequently followed by a decrease in the surface temperature, cold perspiration, feeble pulse, muscular tremor, nausea, and vomiting. In the more severe cases, the depression may be so great as to cause a fatal termination.

Cases of death following the extraction of teeth under the influence of anesthetics, and sometimes erroneously attributed to heart-disease, are no doubt often the result of shock, induced by the progress of the operation before the sensory nerves were completely paralyzed by the action of the anesthetics.

The fifth nerve is considered to be the most sensitive nerve of the body, and it is also among the last to yield to the influences of anesthetic drugs. The necessity, therefore, of complete anesthesia in tooth-extraction, or in any other operation in which this nerve is involved, is self-evident.

The object of anesthesia is to annihilate pain and prevent shock from surgical injury; but if it is not carried to the point of profound impression, or complete insensibility, the desired result is not attained. The loss of mental consciousness is not anesthesia, but this condition takes place some time before the sensory function of the fifth nerve is paralyzed. There is much less danger to life from shock in surgical operations under profound anesthesia than when it is incomplete. Partial anesthesia invites shock, while profound anesthesia prevents it.

Cases are on record, in considerable number, of persons who have died from shock superinduced by mental excitement, fright, sudden joy, or great grief, or the dread of a surgical operation, sometimes of a trivial nature. In many of these cases there was evidence of abnormal conditions of the system, often associated with diseases of the heart.

Lauder Brunton relates a remarkable case of sudden death from shock, the result of mental emotion. Some medical students seized the janitor, who had displeased them, and made him believe they intended to behead him. They blindfolded him, placed him upon a block, then struck him a sharp blow upon the back of the neck with a wet towel. On removing the bandage they were horrified to find that he was dead.

Several years ago, in a city in central New York, a lady past fifty years of age called upon a dentist for the extraction of an aching tooth. She was placed in a chair preparatory to the operation, when almost immediately she expired. The post-mortem examination revealed fatty degeneration of the heart. The coroner's verdict was death from heart-disease, caused by fright. This is an exceptional case, but it illustrates the fact that shock is sometimes superinduced by mental excitement, and that it may, under certain weakened conditions of the heart, terminate in instant death.

A lawyer, aged fifty years, strong and robust, and of great physical endurance, applied for dental treatment. Five years before he was injured by his horse falling upon him, breaking the right femur and fracturing several ribs. The nervous shock was very great, and he never fully recovered from it. Before the accident he had always prided himself upon being able to endure any amount of physical suffering incident to dental operations without flinching. After the accident he was never able to endure a sitting of more than fifteen or twenty minutes long, while during the whole time cold perspiration would stand in great drops upon his forehead, and he would complain of suffering the most intense agony. It was only possible to insert temporary fillings from this time until his death, which occurred about two years later. The fatigue and prostration following even these short sittings were very considerable, emphasizing the fact that the character of the operations must be tempered to the physical condition of each individual.

The various symptoms and conditions of surgical shock and collapse occasionally following dental operations to the healthy and robust individual may be of trivial moment, but in those of exalted nervous susceptibility, the physically or mentally overworked or depressed, and those in whom vitality is low from chronic ailments, or because just recovering from acute disease, may result in serious and sometimes fatal consequences.

The prevention of such untoward results will depend very largely upon the ability of the surgeon or dentist to correctly diagnosticate the physical conditions of individuals presenting themselves for treatment, to correctly judge as to the amount of fatigue and nervous strain each can endure without detriment to health, and the proper use of drugs and other means at his command to mitigate the suffering from such operations.

The following formulæ may be used with benefit in allaying nervous irritability and in preventing shock from dental operations:

℞—Croton chloral hydrate, gr. x;
Bourbon whisky, fl℥ j.
Sig.—Twenty minutes before operation.
For an adult.

℞—Morphia sulf., gr. ¼;
Bourbon whisky, fl℥ j.
Sig.—Thirty minutes before operation.
For an adult.

℞—Potassium bromid, gr. xx to xxx;
Cinnamon water, fl℥ j.
Sig.—Thirty minutes before operation.
For an adult.

℞—Potassium bromid, gr. xv to xx;
Croton chloral hydrate, gr. x;
Cinnamon water, fl℥ j.
Sig.—Thirty minutes before operation.
For an adult.

REVIEW.

CHAPTER XV.

Give the definition of shock.
How is shock produced?
How does it cause death?
What are the most obvious symptoms?
Give the definition of collapse.
What are the manifestations in extreme collapse?
How do shock and collapse differ in relation to the causes which produce them?
In what class of individuals is shock generally manifested?
What are its causes?
How does it terminate?
In what class of individuals is collapse most likely to appear?

How does exhaustion predispose to collapse?

What are the differences in the period of reaction between shock and collapse?

How does extreme collapse terminate?

What is the result of shock complicated with profuse hemorrhage?

What influence do abnormal conditions of the system exert upon nervous excitability and physical endurance?

How do temperaments modify the influence of shock?

What influence does age exert?

What is known of the pathology of shock?

Through what agency are the phenomena of shock made manifest?

What has experimental physiology demonstrated in this direction?

What do the symptoms indicate?

What do the symptoms of shock comprehend?

What are the most striking symptoms?

When may reaction take place?

What are the signs of normal reaction?

What are the characteristics of abnormal reaction?

How does this form of reaction usually terminate?

Upon what conditions does the prognosis of shock largely depend?

In fatal shock, how soon does death supervene?

What conditions are considered unfavorable?

Are surgical operations contra-indicated under such circumstances?

What other conditions are considered as fatal symptoms?

What line of treatment is indicated in mild cases of shock?

What is the line of treatment for the severer cases?

What are the principal requirements in the treatment?

In shock occurring during an operation, what course should be followed?

Describe the care and treatment to be employed in a severe case of shock.

What treatment is recommended in severe cases of shock and collapse resulting from hemorrhage?

Give the formula for the salt solution.

At what temperature should the solution be injected?

How is it to be introduced into the circulation?

What precaution should be observed?

In what quantity may the solution be safely introduced into the system?

Should patients suffering from shock caused by hemorrhage be permitted to gratify their desire to drink large quantities of water?

What other means can be employed to replenish the lost fluid?

In what quantity and how often may rectal injections be administered?

What form of stimulants is recommended?

Give the dose of each.

How may opium be administered, and what is its value?

Of what value is digitalis?

With what other drug may it be combined?

What is the dose of each of these?

In what cases is nitro-glycerin recommended?

What is the dose?

In what dose is strychnia administered in such cases?

When and how should the stimulants be withdrawn?

When should the means for restoring the normal temperature be discontinued?

What class of individuals are unfavorably affected by painful and long-continued dental operations?

Why is it not wise to submit delicate children to severe dental operations?

What is the usual result of such painful and prolonged operations?

How might fatal consequences result?

At what particular periods is it unsafe to submit children to such operations if prolonged and painful?

What are some of the nervous conditions which may be developed as a result of such operations?

Under what conditions are the methods of orthodontia dangerous?

Under what circumstances are dental operations not well borne by girls and women?

What then should be the character of the operation?

What serious conditions sometimes result from some dental operations?

What symptoms of shock may sometimes follow the extraction of teeth?

How does partial anesthesia invite shock?

How does complete anesthesia prevent it?

What other means may be employed to relieve pain in dental operations?

What would be the dose of croton chloral hydrate?

How is it best administered for this purpose?

What would be the dose of morphia sulfate?—and with what may it be combined?

What is the dose of potassium bromid?

Give the formula for the bromid mixture.

Give the formula for the bromid and chloral mixture.

CHAPTER XVI.

LIGATURES, SUTURES, AND SUTURING.

Ligatures.

Definition.—*Ligature* (Lat. *ligare*, to tie). A ligature is a cord or thread of any material that is used for tying arteries, or ligating abnormal growths, etc.

Silk, catgut, silkworm-gut, and horse-hair are the materials most commonly used for the purposes of ligation.

The animal ligatures are made from the sheep's gut, the sinews, tendons, and the skins of various animals.

All animal ligatures are capable of being absorbed.

When a ligature is to be applied to a deep-seated vessel and the wound closed over the ligature,—buried,—absorbable materials in most cases should have the preference.

Catgut is most frequently used for buried ligatures. This material is made from the middle coat of the intestines of the sheep previously treated to render it tough and strong, and cut into threads of various sizes to suit the demand.

The first absorbable ligature was made and used by Dr. Physick, who utilized the untanned buckskin for this purpose.

Horse-hair makes an excellent ligature when formed by braiding several strands together, and it becomes exceedingly strong and pliable when immersed in oil for a considerable period. It is capable of slow absorption.

In selecting horse-hair for ligatures and sutures, it is always best to take those from the top of the tail, where they have not been contaminated so much with the excrementitious matter of the body.

Kangaroo Ligatures are made from the smaller tendons of the kangaroo's tail. They are stronger than catgut, but not so rapidly absorbed.

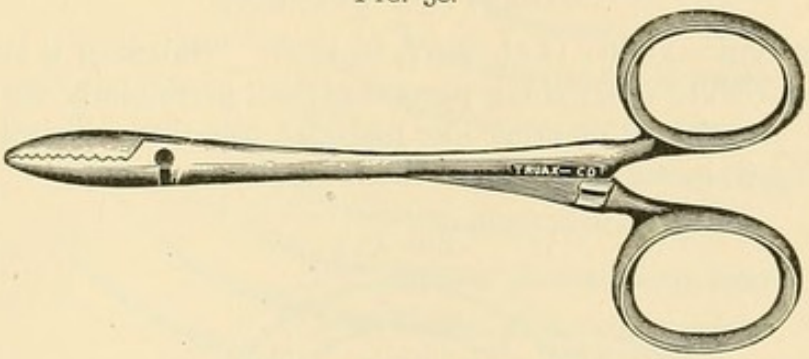
Silkworm-gut is made from the thin, thread-like material drawn from the silkworm which has been killed when ready to spin its cocoon. This material and silk thread are, under favorable circumstances, sometimes absorbed, but the process is much slower than with catgut and other animal ligatures.

Silver wire is occasionally used for the ligation of arteries; when used for this purpose the ends are cut short, and the wire is allowed to remain and become encysted.

Ligation of Vessels.—Ligation of bleeding vessels is the most important of all the measures employed for the arrest of hemorrhage.

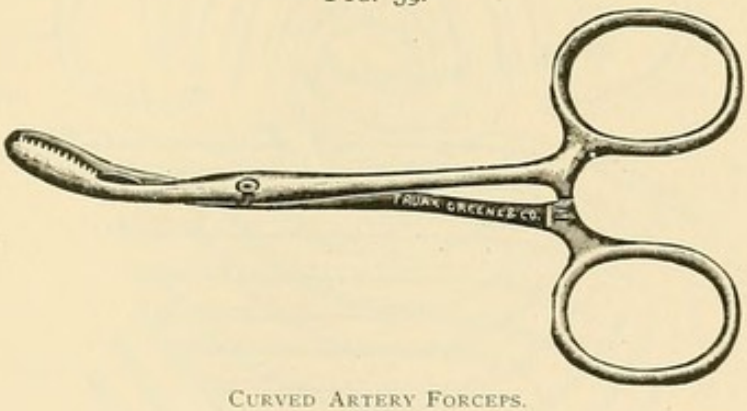
This operation consists of seizing the end of the bleeding vessel with the hemostatic forceps (Figs. 38, 39), lifting it from its bed and passing the ligature around the vessel at the point of the forceps, and tying it tightly with a square or surgeon's knot (Fig. 40).

FIG. 38.



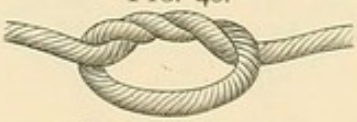
STRAIGHT ARTERY FORCEPS.

FIG. 39.



CURVED ARTERY FORCEPS.

FIG. 40.



SURGEON'S KNOT.

If the ligature is to be buried, the ends should be cut short; if it is to be removed at a later period after it has sloughed off, one end should be left long enough to reach outside the lips of the wound.

The effect of a ligature tied about an artery is to divide the middle and inner coats, which curl up within the vessel,—invaginate; the blood-current is arrested, and clotting takes place, which extends backward to the first lateral branch.

Plastic lymph is then deposited about the ligature, the clot becomes infiltrated with leucocytes and is organized into fibrous tissue, and the artery is converted into a fibrous cord. The ligature, if of animal tissue and aseptic, is absorbed; otherwise it is encysted.

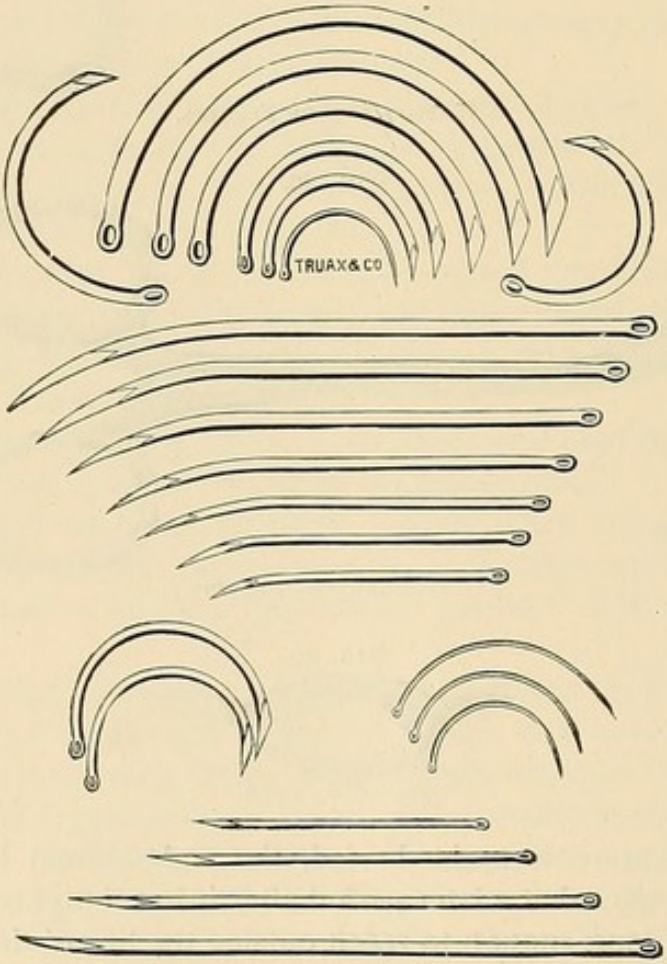
If the ligature is septic, it causes irritation; inflammation with suppuration and ulceration takes place, followed by sloughing, and may be accompanied by secondary hemorrhage.

All ligatures, of every description, should be rendered aseptic by treatment with antiseptic solutions, or by boiling.

Sutures and Suturing.

Definition.—*Suture* (Lat. *suere*, to sew). "Suturing is the operation or procedure of stitching parts together, particularly the lips of a wound. The thread or other like material, together with the method of inserting it, in the operation of stitching parts together."

Fig. 41.



Hagedorn Needles.

Numerous forms of needles are used by surgeons, each adapted to its special purpose, or to conform to the fancy of the operator. For accessible wounds, a straight needle is most convenient; but

where they are less accessible, it is necessary to employ needles with more or less curvature at the point, or even crescent-shaped (Figs. 41, 42, 43).

A good needle-holder is very essential in the suturing of wounds, and it must be adapted to the form of needle to be used. Figs. 44, 45, are forms in common use; Fig. 46 is a new instrument devised by Dr. Belknap.

FIG. 42.

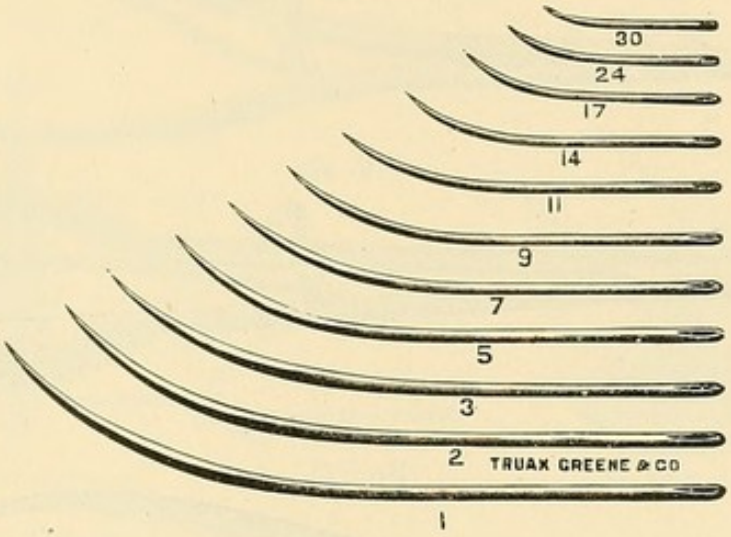
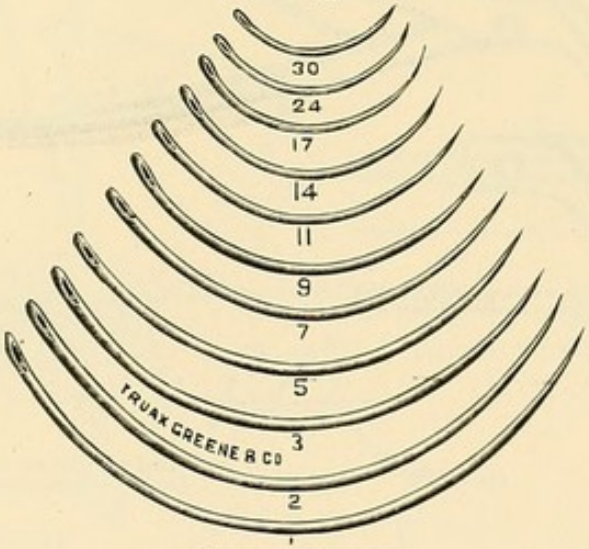


FIG. 43.



CURVED NEEDLES.

The common methods of suturing for closing wounds or stitching divided soft structures together, are—

1st. The Continuous or Glover's Suture.—By this method the wound is closed with one continuous thread, after the manner of the glover's stitch or the tailor's whip-stitch, or overhand. The needle being armed with a single or double thread, as desired, is passed through both lips of the wound from side to side, and the first stitch

tied; then the needle is passed in the same manner as at first, always entering in the same side, and tying the last stitch when completed (Fig. 47).

2d. The Interrupted Suture.—In approximating divided surfaces by this method, the needle is passed through both lips of the wound,

FIG. 44.

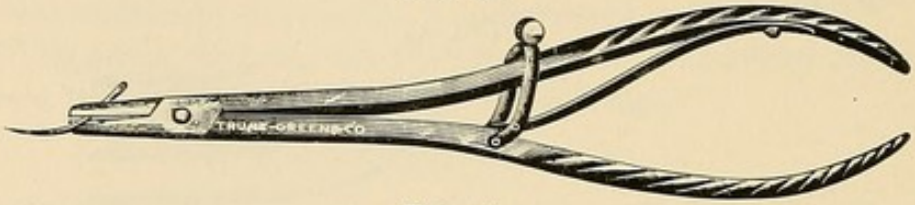
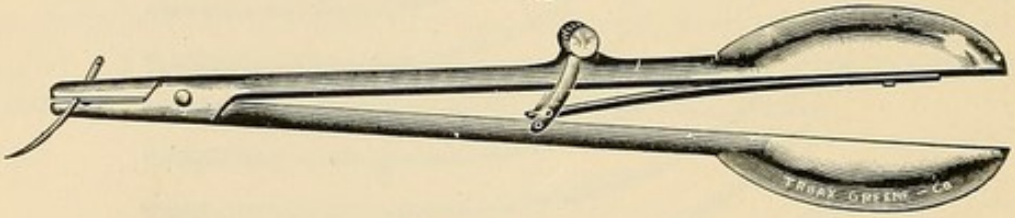
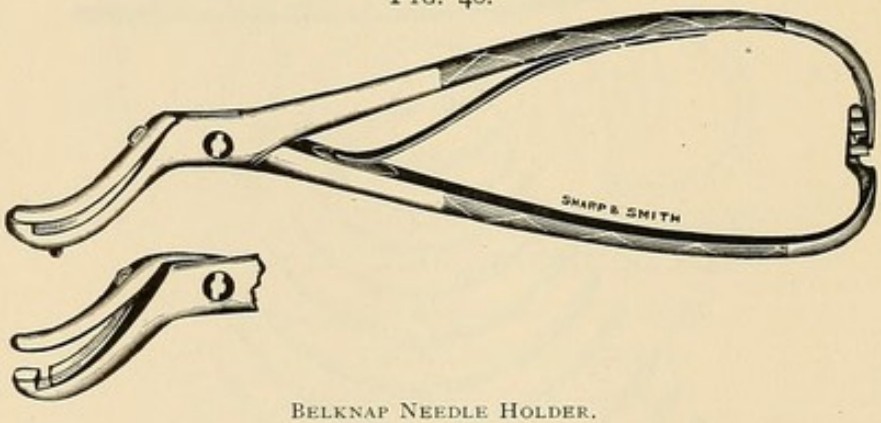


FIG. 45.



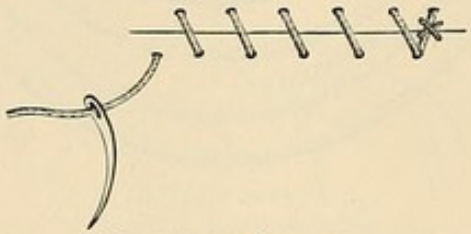
NEEDLE HOLDERS.

FIG. 46.



BELKNAP NEEDLE HOLDER.

FIG. 47.



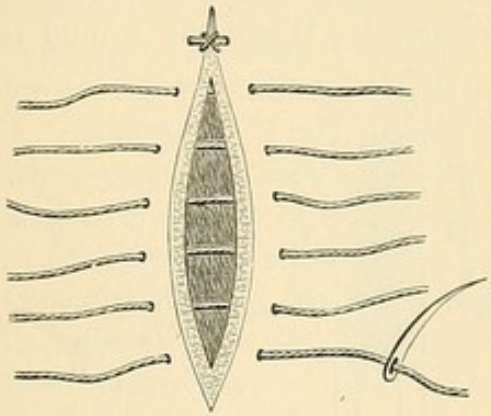
CONTINUOUS SUTURE.

and each stitch tied before another is placed (Fig. 48). This is the form of suture most commonly employed with all suture materials except metallic wires.

3d. The Pin Suture.—This form of suture is better known as the hare-lip or twisted suture, as it is more frequently used in closing hare-lip than in any other operation. It consists of passing long, slender

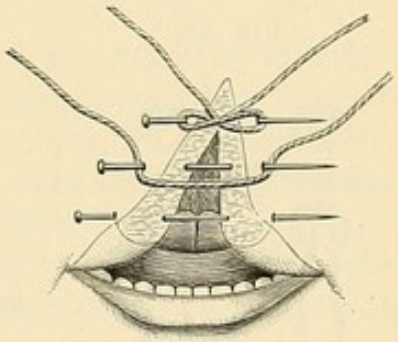
metallic pins through the lips of the wound, and securing apposition by winding a silk thread under the exposed and opposite ends of the pin (Fig. 49). The thread may be carried in the form of an ellipse, or the figure 8, and finally, in either case, from one pin to the other, in order the more completely to cover the edges of the wound. The points of the pins must be cut off with a pair of nippers, after the suture is tied. Rubber bands are sometimes used instead of the thread.

FIG. 48.



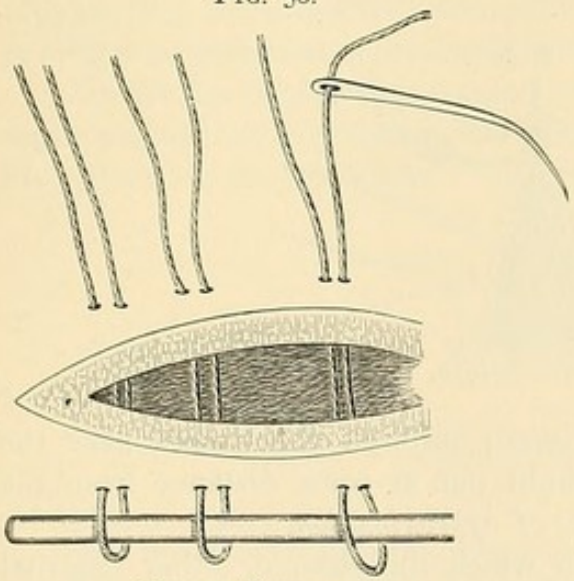
INTERRUPTED SUTURE.

FIG. 49.



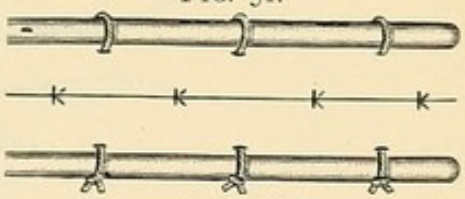
PIN SUTURE.

FIG. 50.



QUILLED SUTURE.

FIG. 51.



QUILLED SUTURE COMPLETED.

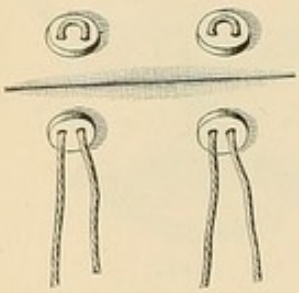
4th. The Quilled Suture.—Double threads are passed across the wound at some little distance from the edges, and quite deeply, including the skin and other tissues. One end of the thread is looped over a quill, or a section of soft, elastic catheter, on the one side, and the other free end drawn tightly and tied over the quill upon the opposite side (Figs. 50, 51). Superficial stitches are usually inserted between the quilled sutures.

5th. The Clamp Suture.—This suture is a modification of the

quilled suture. Strips of lead, having holes punched through them at the proper intervals, are used instead of the quills, the thread being passed through the holes in the lead, and tied upon opposite sides.

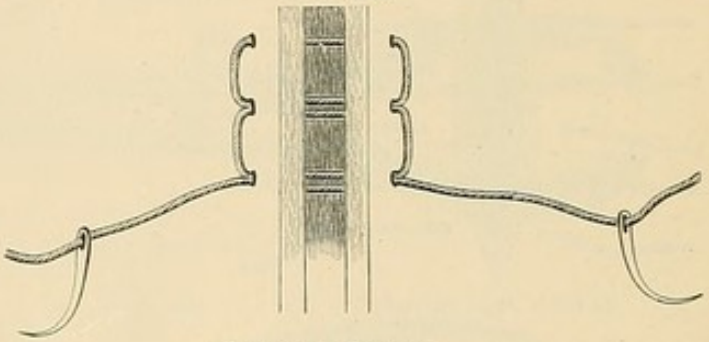
6th. The Button Suture.—This is also a modification of the quilled or the clamp suture. Wire is passed across the bottom of the wound, brought out at some distance from the edges, and secured by fastening to leaden buttons, having holes punched through them (Fig. 52). The quill, clamp, and button sutures are employed where the loss of tissue has been considerable, to prevent tension upon the superficial stitches, and are termed *sutures of relaxation.* When the wound is

FIG. 52.



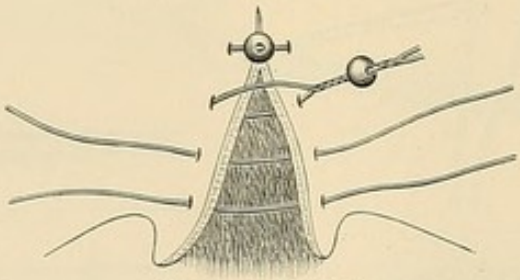
BUTTON SUTURE.

FIG. 53.



COBBLER'S SUTURE.

FIG. 54.



SHOTTED SUTURE.

deep, interrupted sutures are passed through the tissues near the bottom of the wound, and brought out at some distance from the edges. These are termed *sutures of approximation.*

The superficial sutures with which the skin, or other external surfaces, are accurately joined, are termed *sutures of coaptation.*

7th. The Buried Suture.—This form of suture is employed to approximate structures in the deep portions of wounds. The suture is usually passed horizontally through the sides of a wound, and tied, the superficial tissues being brought together over it. Buried sutures are always completely covered by the skin, and do not enter this structure at all.

8th. The Cobbler's Suture.—This is a continuous suture; armed with two needles, the lips of the wound or denuded surface being

brought together by a back-and-forth stitch, with first one needle and then the other (Fig. 53). This is a favorite suture with the writer for uniting the denuded surfaces of the subperiosteal flaps in urano-plasties.

9th. The Shotted Suture.—This suture is of silver wire, which is passed through both lips of the wound, or the edges to be united and twisted; a perforated leaden shot is then threaded upon both ends of the wire, and carried downward until the lips of the wound are approximated, and then it is tightly compressed upon the wire, and the wire cut close to the shot (Fig. 54). This form of suture is frequently used for uniting the edges of a cleft palate.

Sutures, as a rule, should be removed on the fourth or fifth day, when employed about the face, except in those cases where there is danger of gaping of the wound; and even in these, by a judicious use of adhesive plaster, or gauze and collodion, this danger may be successfully guarded against. A safe rule to follow is always to remove the sutures upon the first observable tendency to irritation. Suppuration must always be guarded against, as this process invariably produces a cicatrix.

In operations for cleft palate where silver wire sutures are employed, they are sometimes allowed to remain from eight to ten days, provided no irritation is produced by their presence.

By the use of antiseptic sutures, dressings and solutions, and antiseptic pre-operative and after-treatment, the dangers from suppuration are reduced to the minimum.

REVIEW.

CHAPTER XVI.

What is a ligature?
What materials are used for ligatures?
What materials should have the preference for buried ligatures?
Give the reason why.
To whom belongs the credit of first having used an absorbable ligature?
From what is catgut made?
Are silk and silkworm-gut absorbable materials?
What is the method of applying a ligature?
What is the effect upon the structures of an artery of tying a ligature around it?
How should ligatures be prepared to prevent suppuration?
Describe the knot used in tying ligatures.
What is a suture?
How should the thread be prepared for suturing a wound?

Describe the forms of surgeon's needles.
Describe the continuous suture.
Describe the interrupted suture.
Describe the pin suture.
Describe the quilled suture.
Describe the clamp suture.
Describe the button suture.
Describe the buried suture.
Describe the cobbler's suture.
Describe the shotted suture.
At what time may sutures generally be removed?
What should indicate their immediate removal?
How long may silver sutures remain in cleft-palate cases?

PART II.

CHAPTER XVII.

WOUNDS.

WOUND (A. S. *wund*, a wound).

Definition.—A *wound* is a break or division of continuity of the soft parts, caused by mechanical violence,—a trauma.

"Solutions of continuity may occur in any of the tissues of the body from slowly-acting causes operating within the body itself, like the process of ulceration, or the gradual wasting of atrophy." Such a breach in the continuity of the tissues, however, would not be classed as a wound. In the idea of wounds there is involved the action of some force outside of the body which has caused a division or break in the tissues by violence. But even with this somewhat restricted definition of a wound, the term has yet a very wide range of applicability. All solutions of continuity, therefore, the result of violence from external forces, such as contusions, sprains, fractures, punctures, lacerations, incisions, etc., of any of the external or internal tissues or organs of the body, would be termed wounds. All traumatic injuries are essentially identical in that the same process of repair is involved in the healing of all of them, and they are subject to the same general principles of treatment, the application of which is regulated by the structure of the tissue, its means of nutrition, its particular function, its relationship to surrounding tissues, and the character and extent of the injury.

Classification.—All wounds may be divided into two general classes, viz: *open* and *subcutaneous* wounds. To the first class belong all wounds which are accompanied by a break in the external tissues, while under the second may be grouped all those injuries to the subcutaneous tissues which are caused without a solution of the continuity of the skin or mucous membrane.

Wounds are also classified as *superficial* and *deep, simple* and *complicated*. They are *superficial* when the skin or mucous membrane only

are involved; *deep* when the underlying tissues are implicated; *simple* when the soft parts are divided without complication of any kind; *complicated* when a foreign body or substance is present, or when there is considerable hemorrhage or pain. Wounds are termed *incised* when caused by a cutting instrument; *lacerated* when the tissues are torn; *contused* when bruised or crushed with a blunt instrument or heavy weight; *punctured* when made with a pointed instrument; *poisoned* when some toxic substance has been introduced, like dissecting wounds, the bite of a rabid dog or of a venomous snake.

A *gunshot* wound is one which has been made by a projectile. *Penetrating* wounds are those in which the missile enters a cavity without emerging. *Perforating* wounds are those in which the missile both enters into and emerges from the cavity.

A *surgical* wound is one which has been made in the performance of a surgical operation.

A *flesh* wound is one in which the skin and muscular tissues only are involved.

An *open* wound is one having a free external opening or breach of the skin or mucous membrane.

A *closed* wound is one having no external opening, or when the wound has been brought together with sutures.

A *subcutaneous* wound is one in which the tissues beneath the skin or mucous membrane have been torn or lacerated, and the external tissues have remained intact. Subcutaneous wounds generally heal without suppurative inflammation, as the unbroken external tissues protect them from outside forms of irritation and from infection.

Wounds may be again classed according to their septic or aseptic condition. Septic wounds include all those traumatic injuries which have become infected with the micro-organisms which cause fermentation or putrefaction, like the pus-microbes or the saprophytic germs. Such wounds are always accompanied by a considerable degree of inflammation, suppuration, and sloughing of dead tissue; the amount of dead tissue thus thrown off depending upon the extent and severity of the primary injury and the character of the inflammation.

Aseptic wounds include all traumatic injuries which have escaped infection by micro-organisms or their products. The aseptic condition may be the result of the resistance of the tissues to the growth and development of the micro-organisms which may have gained access to the wound; or of the protection which the wound received at the time of its infliction, against the entrance of septic agents; or of the application of agents which removed or destroyed them and afterward prevented their ingress.

Healing of Wounds.—The process of repair or regeneration of tissues, constantly going on in the living human body, is a physiolog-

ical function, intended to replace the normal waste of tissue, and to restore tissues which have been lost by injury or disease. In the full-grown body the waste incident to a healthy activity is balanced by the reparative process, but during the period of the development of the body the material appropriated by the tissues is largely in excess of the physiological waste. This increase in the material appropriated by the tissues constitutes growth or physical development. When waste of tissue is in excess of cell-production, atrophy is the result; *e.g.*, general emaciation on the one hand, and atrophy of certain groups of muscles on the other, the result of neurotic disturbance, are marked illustrations.

When the process of regeneration is completely suspended, death must soon follow as a natural consequence.

The blood is the first tissue to be affected by any marked change in the equilibrium between waste and repair. The other tissues soon suffer in a degree proportionate to the diminution in the function of nutrition, and finally death results from marasmus.

Before the introduction of antiseptic treatment of wounds it was supposed that inflammation was necessary for the regeneration of injuries or lost tissues, but since it has been discovered that wounds in a state of asepsis heal without the usual phenomena of inflammation, the processes of repair and that of inflammation have been regarded as entirely distinct from each other.

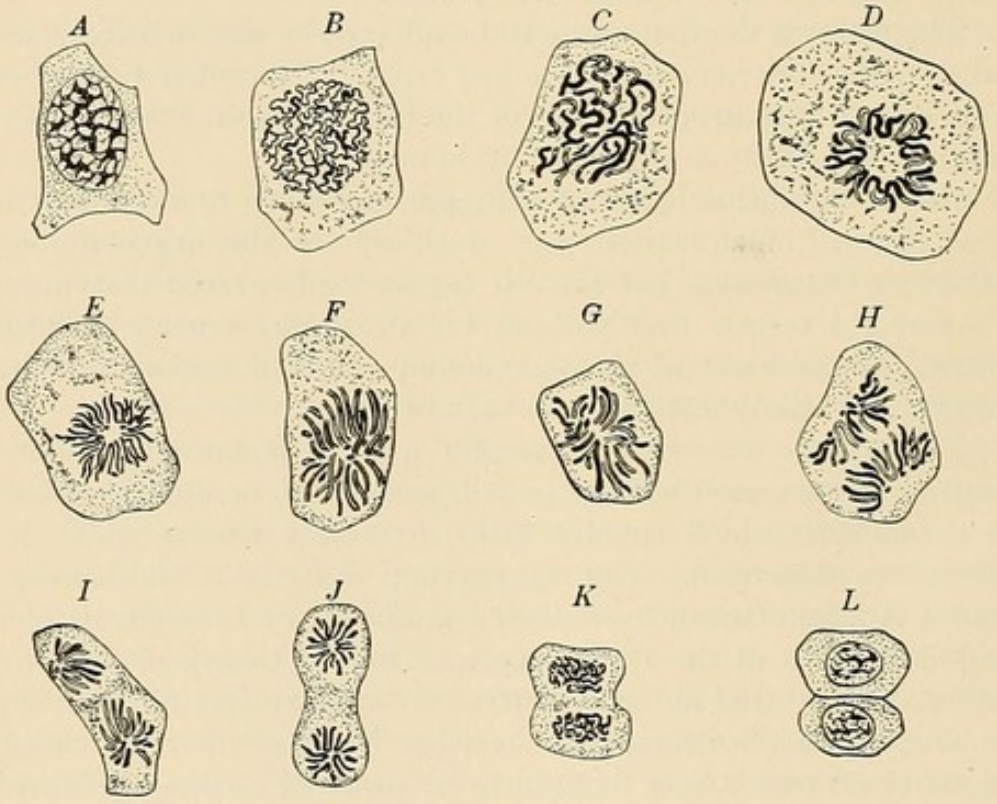
The process of repair in aseptic wounds is brought about by means of an increased activity in cell-production, or multiplication of the tissue-cells, which enables them to replace tissues which have been lost or destroyed. The regeneration of tissue in the healing of wounds is accompanied by *indirect cell-division* or *karyokinesis* of the fixed-tissue cells of the injured part. The old theory of cell-proliferation assumed that all cells underwent segmentation of the nucleus and division of the protoplasm by what is called *direct cell-division*. The latter process is now thought to be confined to those cells which do not possess the power of forming new tissues; as, for instance, the leucocytes, which take no prominent part in the process of repair, but rather serve as nutrient media for the forming tissues.

According to the most recent investigations into the process of cell-multiplication, or karyokinesis, it has been shown (Ziegler) that the process is attended with peculiar changes in the cell and its nucleus. The first signs which are made manifest that cell-division is about to take place are certain changes in the nucleus which finally result in subdivision. Later the protoplasm takes on active changes, influenced, seemingly, by the activity of the nucleus, which eventuates in the complete subdivision of the cell into two equal portions, each complete in itself.

These changes, according to Flemming (Ziegler), take place in the following order: The first stage in the process of division of the nucleus is the disappearance of the nuclei, while the substance of the nucleus assumes the form of a mass of sinuous filaments or a raveled coil, which is termed the coil-form of the *mother nucleus*. From this stage in the process the nuclear substance alone is susceptible to the action of staining reagents, and for this reason the nuclear substance has been termed chromatin (Fig. 55).

The filaments now become thicker and the coil looser; breaks occur in its continuity, and it gradually passes into a wreath-like form. The arrangement of the filaments is in a series of loose central and

FIG. 55.

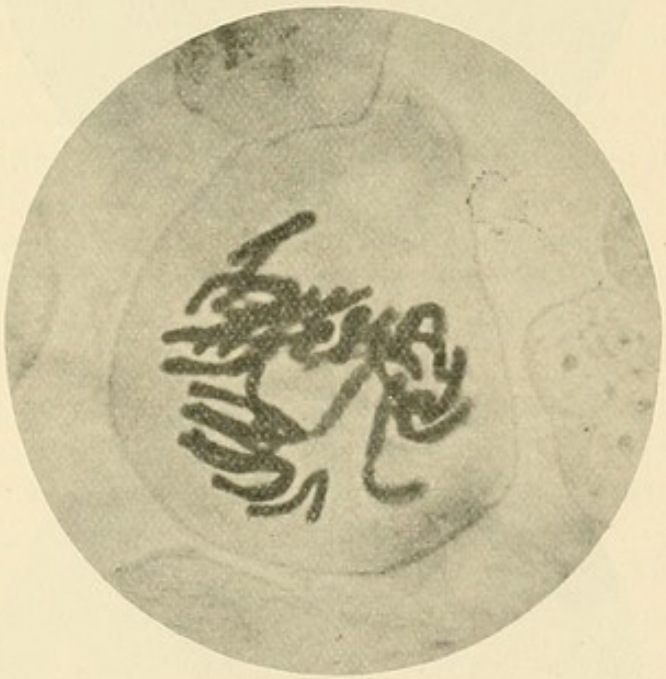


CELLS FROM THE EPIDERMIS OF VERY YOUNG LARVA OF NEWT. (After Piersol.)
A, resting nucleus; *B*, close skeins; *C*, loose skeins; *D* and *E*, mother stars, seen from the polar field and appearing as the wreath stage; *F*, mother star from the side; *G*, migration of segments; *H*, daughter stars; *I* and *J*, segments grouped about new polar fields (in *J* this protoplasm exhibits constriction); *K*, daughter skeins (division of nucleus complete, with slight constriction of cell-body); *L*, complete division of nucleus and protoplasm.

peripheral loopings with the center of the *mother nucleus* unoccupied. The next change of the nucleus is from the wreath-like arrangement of the filaments to that of a star-form or asterisk, with double rays, and the peripheral loops later divided at their free extremities. Following this the double rays divide longitudinally, and a considerable contraction of the whole star-form takes place. The single-rayed star thus formed next divides through the equator into two equal polar seg-

ments. This division is accompanied by the formation of a transparent equatorial plate (Strasburger's cell-plate), which is often marked by a line of five points. Later the polar segments move asunder toward opposite poles, and assume an appearance resembling a "half-barrel" or a "basket form." These now represent the *daughter nuclei*, which soon pass into the star-form, and this into the wreath-form, by a fusion of the ends of the star-rays. At the same time a constriction of the cell protoplasm commences. The wreath-form of the daughter nuclei now shrinks, and its filaments become more and more sinuous, until it assumes the coil-form. The constriction of the cell-protoplasm has also by this time been completed. In the last stage the coil-form becomes more loose and regular, and finally develops into the nuclear net-work or reticulum, like that of the mother nucleus.

FIG. 56.



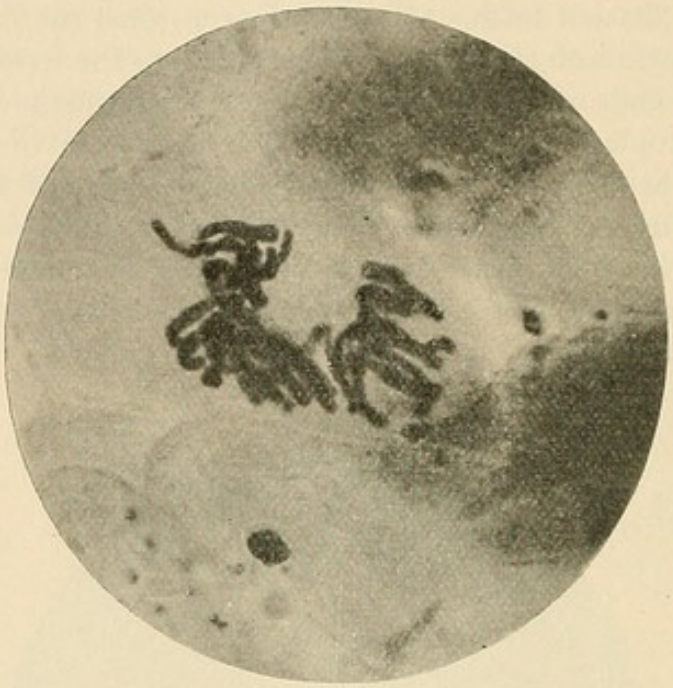
KARYOKINESIS—LOOSE SKEIN. × 1500.

The severance of the cell-protoplasm completes the process of karyokinesis, and the new-formed nuclei enter the resting state corresponding to the resting state of the mother nucleus. During the process of subdivision the nucleus is surrounded by a clear intermediate substance. This substance during the active stages of subdivision is not susceptible to staining reagents, but in the resting state it is readily stained. Figs. 56, 57, 58, are photo-micrographs of the three principal stages in the process of karyokinesis,—the formation of the loose skein, the equatorial plate, and the separation into daughter asters or stars; the outline of the cell-protoplasm being distinctly seen.

Sometimes segmentation of the nucleus takes place without sub-

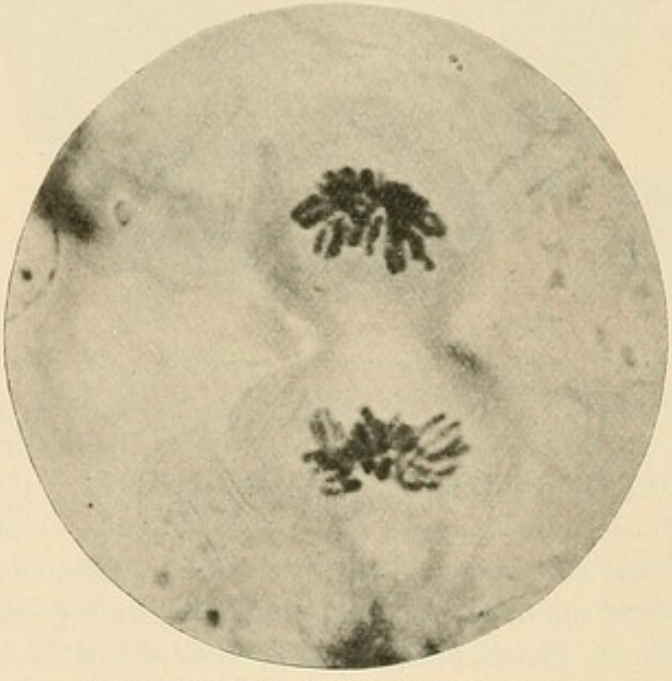
division of the cell-protoplasm, thus forming a binucleated cell; or if the process of segmentation goes on, a multi-nucleated or *giant cell* is formed. The giant cells, however, may later break up into uninu-

FIG. 57.



KARYOKINESIS—EQUATORIAL PLATE. × 1500.

FIG. 58.



KARYOKINESIS—DAUGHTER ASTERS. × 1500.

cleated cells, the protoplasm gathering around the individual nuclei and dividing along the boundaries so defined.

Regeneration of tissue includes the reparative process which takes place in the healing of traumatic wounds, and the reproduction of tissue lost from inflammatory processes.

All reparative processes are brought about by cell-proliferation, "each after its kind." This is a histogenetic law which has been abundantly demonstrated, and generally accepted. Every normal cell is endowed with an inherent tendency to reproduce itself, and to transmit its own peculiar function of tissue-building. This function is never perverted to the production of a tissue with a materially different histologic structure, but always reproduces a tissue which is anatomically and physiologically like the tissue of which it formed a part. Nerve-cells produce nerve-cells; epithelial cells are only produced by epithelial cells; bone-cells by bone-cells; enamel-cells by enamel-cells, etc.

All wounds, whatever their anatomical structure, heal by cell-proliferation,—by the production of new material from the fixed-tissue cells of the immediate neighborhood.

The fixed-tissue cells being endowed with the power of adaptation to conditions which surround them, begin, immediately after the injury has been received, to inaugurate the process of repair. This consists of rapid segmentation of the pre-existing tissue-cells, thus forming embryonal cells; these cells gradually assume the character of mature cells, as the process of healing progresses, until finally they represent the tissues from which they had their origin.

Methods of Healing.—The processes by which wounds heal are generally classed under three heads: First, union by primary adhesion, or first intention; second, adhesion by granulation, or second intention; third, secondary adhesion, or third intention.

Healing by Primary Adhesion, or First Intention.—To secure union by this process the surfaces of the wound must be accurately approximated, and shielded from all forms of irritation. Under such circumstances the exudate will be of minimum quantity, and absorption of the red blood-corpuscles will occur; at the expiration of twenty-four hours adhesion between the surfaces has taken place, and at the end of two or three days the new-formed material (plastic lymph) which binds them together will be traversed by blood-vessels.

John Hunter believed that the process of primary union might be accomplished in a few hours in incised wounds where perfect coaptation of the parts could be secured, without the interposition of new material. He has been supported in these views by Macartney and Sir James Paget, and later, among the more modern pathologists and surgeons, we find Thiersch holding the same views.

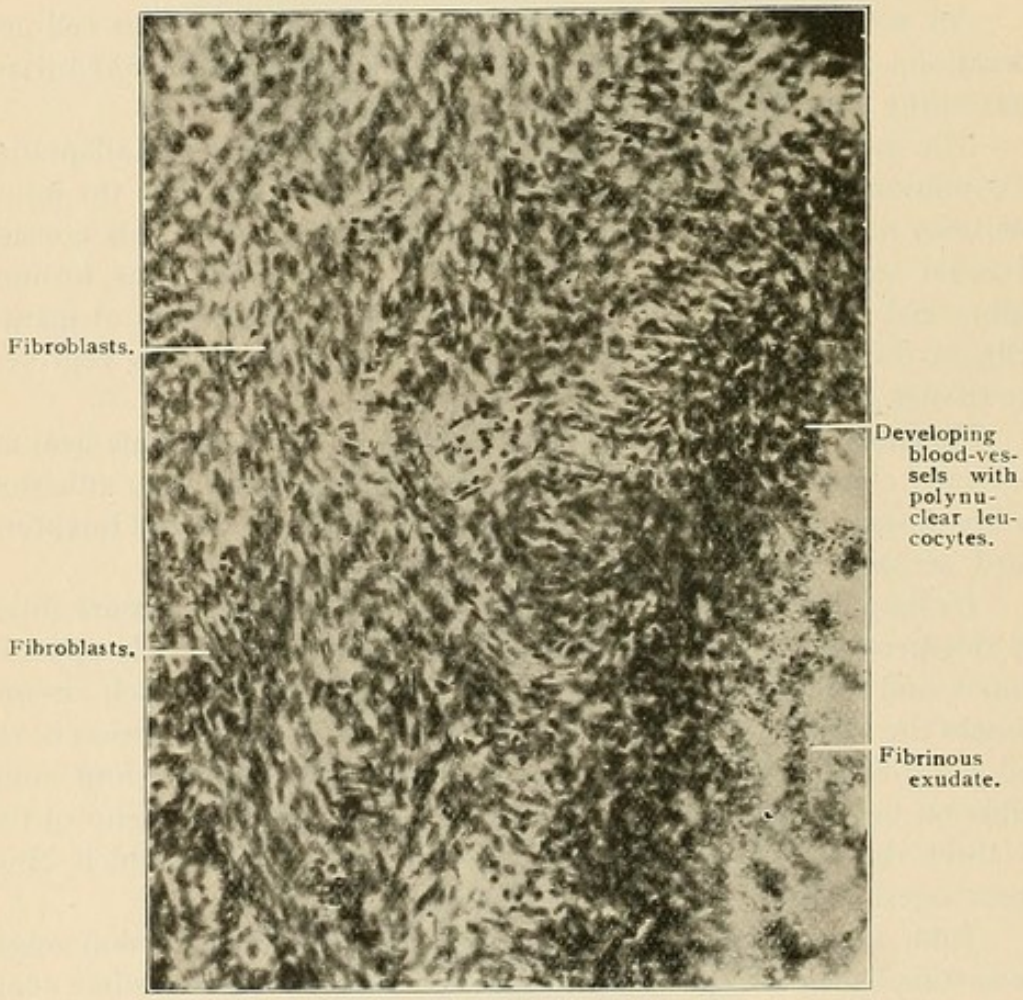
The process of *primary* or *immediate union* is usually accomplished with only the slightest manifestations of inflammation, rarely pro-

gressing beyond a little hyperemia, puffiness, and tenderness about the edges of the wound, and leaves little or no scar.

In primary union there is restoration of continuity, a coaptation of divided tissues, part with part,—an organic union, vessel with vessel, and nerve with nerve. In deep wounds, where important nerve-trunks are severed, it is necessary to bring the divided ends of the nerve together by suturing, and some surgeons maintain that nerve function is occasionally restored in a few hours after this operation.

Restoration of function in the circulation and nerve-supply, even under the most favorable circumstances that can possibly surround an incised wound, rarely occurs under six to eight days. Wounds which heal by first intention are always aseptic.

FIG. 59.



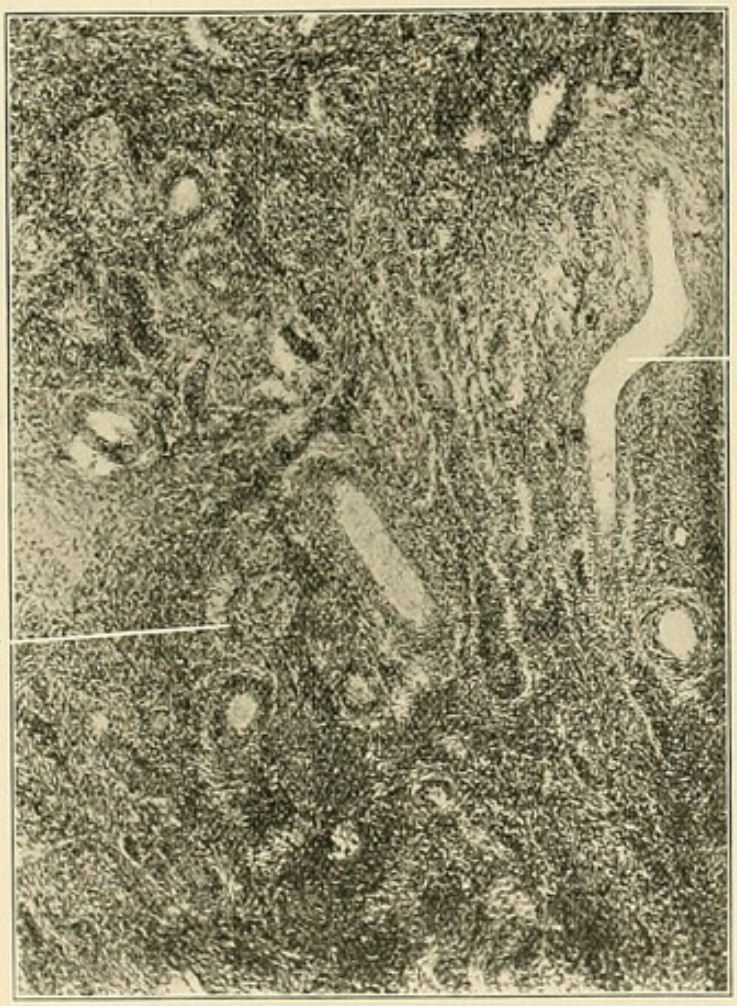
INFLAMMATION—SHOWING FIBROBLASTS IN REGENERATION OF TISSUE. × 50.

Healing by Second Intention, or Adhesion by Granulation.—This process takes place in those wounds in which the surfaces have not been accurately brought together, or have been subjected to irritation of a mechanical, chemical, or septic nature. Under these circumstances the exuded material becomes excessive, death of the blood-

cells and the newly-formed embryonal cells takes place, resulting in suppuration. On the removal of the source of irritation, the inflammatory process quickly subsides, and is immediately followed by cell-proliferation, which soon fills the gap with granulations, and later covers the surface with a modified epithelial structure which forms a scar. During the formation of the granulation-tissue, the fixed-tissue cells are in an active state of multiplication by subdivision, and from these embryonic cells the new tissue is formed to replace that which has been lost (Fig. 59).

Ziegler defines *granulation-tissue* as "a structure fashioned out of

FIG. 60.



INFLAMMATION—SHOWING FIBROBLASTS AND VASCULARIZATION IN GRANULATION-TISSUE. × 50.

the cellular material gathered by the blood from the system in general, and utilized to make good a defect which the fixed-tissue cells of the injured region are unable to repair." The tissue formed by the process of granulation is cicatricial tissue, and is devoid of all specialized structures except blood-vessels. (Fig. 60.) All specialized tissues like epithelium, muscles, bones, nerves, and blood-vessels can be repro-

duced only by a regenerative cell-proliferation of identical, pre-existing tissues. None of these can be reproduced from granulation-tissue.

Healing by Third Intention, or Adhesion of Granulating Surface.—This process takes place in amputation flaps which have failed to unite by first intention, in large abscess-cavities where it is impossible to bring the walls together, and in those cases where large portions of tissue have been lost by gangrene or traumatisms. In this form of healing, the granulation-tissue reaches out from all surfaces of the wound, gradually encroaching upon the space until they meet and unite. The process of healing may extend over weeks and months, depending upon the character and extent of the tissue to be restored and the condition of the wound. Under a strict antiseptic *régime* such wounds heal much more readily than when these precautions are omitted. The extent of the scar will depend upon the character of the tissue which has been lost and the amount of tissue to be restored.

Senn classes all forms of healing under two heads,—*Union by Primary Intention*, and *Union by Secondary Intention*. The first he would have include all wounds which heal without septic manifestations; in other words, all aseptic wounds, no matter whether they heal in three or four days, or require as many months; while in the other class he would place all wounds which have given evidence of septic inflammation. He does not believe it is correct, from a pathologic or from a practical standpoint, to class aseptic wounds, which, on account of failure of approximation or loss of tissue, must heal by granulation, with infective wounds, in which the reparative process has been disturbed and retarded by suppuration.

Surgical Cleanliness.—Absolute cleanliness in surgical operations is of such great importance in the successful treatment and final issue of all surgical cases, that too much stress cannot be laid upon its strict observance in every detail. The student particularly needs to be impressed at the very outset of his studies in surgery, not only with the fact that wounds do better when such precautions have been taken, but that the dangers to life from complications arising from septic infection are thereby reduced to the minimum. The question of the value of asepsis and antisepsis in all surgical operations, and in the treatment of all surgical diseases, is no longer one of controversy. The statistics of surgical operations of all kinds before the days of Lister, contrasted with those of the last two decades, are the best proof of the value of asepsis and antisepsis in surgery. The best surgeons the world over admit their value, and attempt as far as possible to carry out the principles of this method of treatment. The laboratory proofs that suppuration, septicemia, pyemia, erysipelas, and kindred dangers arise from the pyogenic micro-organisms are now considered so complete that no really unbiased mind can doubt them, while the clinical

proofs are equally convincing. Success does not always perch upon the banners of the most brilliant surgeons, but rather upon the standard of him who is the most careful and painstaking, and whose technique is most perfect in all its details. Carelessness in some apparently minor matter may ruin an otherwise successful operation, or endanger the life of the patient. Cleanliness, above all things, is necessary to a successful treatment of wounds. Not ordinary cleanliness, in its general acceptation, but surgical cleanliness, and there is a vast difference between them. To be surgically clean means to be germ free.

The part to be operated upon must be freed from micro-organisms by thorough washing with soap and water, and irrigating with antiseptic solutions. The hands of the operator, assistants, and nurses must be cleansed in a like manner, and the accumulations under the finger-nails removed. This should be done before bathing them in the antiseptic solutions. All instruments must be first scrubbed with soap and water, and then boiled in water. The ligatures and the drainage-tubes should be kept in antiseptic solutions, and the sponges and dressings of every kind sterilized by heat and antiseptics.

By following such a *régime* as this, all wounds capable of being protected against the entrance of micro-organisms or other foreign elements will heal without inflammatory symptoms, provided they are shielded from mechanical irritation from the sutures and dressings, and the general health of the individual is fairly good.

Wounds sometimes do badly from defective nutrition, either local or general. The former is usually caused by tension or a bad position. These can easily be remedied. But it may occur as a result of the necessary ligation or injury of some important artery, and failure in the establishment of an adequate collateral circulation.

General defective nutrition is the result of a debilitated condition of the system, arising from illness, insufficient or improper food, or excesses, particularly the drink habit. As a rule, fractures of the bones do not unite as readily in the confirmed drunkard as in other persons, and injuries to the tissues are much more liable to result in gangrene and necrosis.

REVIEW.

CHAPTER XVII.

Give the definition of the term wound.
What forms of solution of continuity would not be classed as wounds?
What is involved in the idea of wounds?
What class of injuries are termed wounds?

Is there any difference in the process of repair in the various traumatic injuries?

What regulates the general principles of treatment?

Into what two general classes are wounds divided?

What forms are comprehended in the first class?

What forms comprise the second class?

Into what other classes are they divided?

Describe superficial, deep, simple, and complicated wounds.

What are incised, lacerated, contused, punctured, and poisoned wounds?

What are gunshot, penetrating, and perforating wounds?

Describe surgical, flesh, open, closed, and subcutaneous wounds.

What other classification of wounds may be used?

What are septic wounds?

What are aseptic wounds?

What form of wounds usually remain aseptic?

What is the process of physiologic regeneration?

Which of the tissues is first affected by a change in the equilibrium between waste and repair?

What is the evidence that inflammation is not necessary to the process of repair in wounds?

How is the reparative process brought about?

What other term is applied to *indirect cell-division?*

Describe the process of karyokinesis.

How are giant cells formed?

What does regeneration of tissue include?

What law prevails in the regeneration of the various tissues?

From what form of cells is a new tissue developed?

How is it accomplished?

Name the methods of healing.

Describe the process of healing by first intention.

Is this process accompanied by inflammation?

What is the result of primary union of tissue?

How soon may these functions be restored?

Describe the process of healing by second intention.

What is granulation-tissue?

What specialized structures are found in granulation-tissue?

Can epithelium, muscle, bone, etc., be reproduced by granulation-tissue?

Describe healing by third intention.

What is the value of antiseptic treatment in this form of healing?

How does Senn classify the methods of healing?

What are his reasons for this?

What is the value of surgical cleanliness?

Is there a difference between surgical cleanliness and ordinary cleanliness?

What is the meaning of *surgically clean?*

How may surgical cleanliness be obtained?

What effect does local or general defective nutrition have upon the process of healing?

What are the local causes?

By what conditions is general defective nutrition established?

CHAPTER XVIII.

TREATMENT OF WOUNDS.

THE *Treatment of Wounds* consists of—

First. Asepsis.

Second. The arrest of hemorrhage.

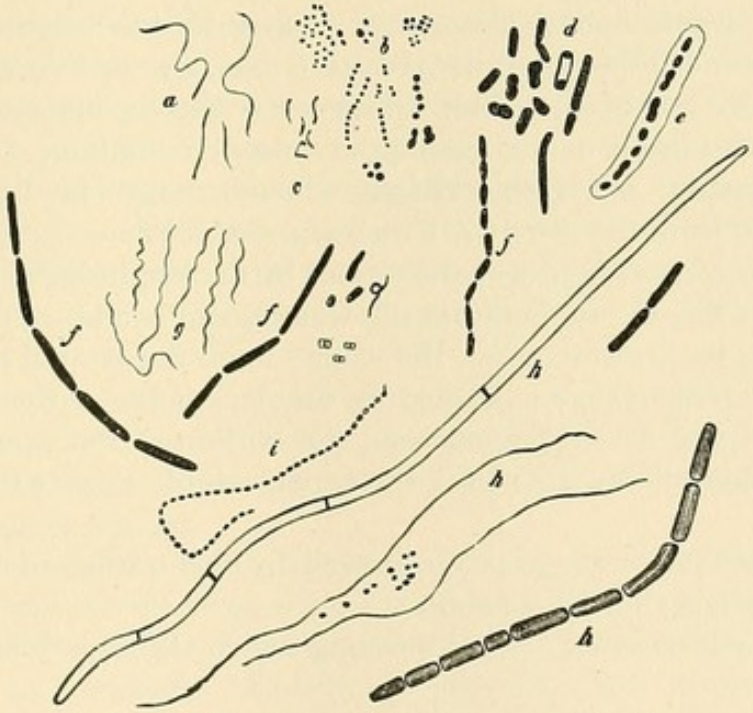
Third. Accurate approximation of the divided surfaces.

Fourth. Providing for drainage.

Fifth. Physiological rest.

Sixth. Proper dressings.

FIG. 61.



VARIOUS FORMS OF BACTERIA FROM THE MOUTH.

a, *c*, *g*, screw-forms; *b*, cocci; *d*, rods; *e*, coccus-chain with sheath; *i*, coccus-chain (streptococci); *f*, rod-chain; *h*, various thread forms. (After Miller.)

Asepsis.—The first consideration in the treatment of all wounds is that of establishing aseptic conditions. To accomplish this end, the wound must be freed from all extraneous substances and foreign bodies, and thoroughly irrigated with antiseptic solutions. When the wound is upon the external surface of the body, the skin about the injured part should be carefully cleansed with soap and water; if hair

be present, it should be removed with a razor, and the wound again carefully irrigated.

When the wound is upon a mucous surface, the same precautions as to asepsis should be rigidly carried out; and when associated with the oral cavity, a determined effort should be made to place the mouth and teeth, as far as possible, in an aseptic condition; but this is not an easy task in those cases where the words *oral hygiene*, or their equivalent, have formed no part of the vocabulary of the individual.

The removal of the salivary calculus and deposits of food upon the teeth, and a thorough irrigation of the mouth with suitable antiseptic solutions, is the only proper method of rendering this cavity approximately germ free; approximately, because absolute sterilization would be impossible of accomplishment with solutions that would not cause serious irritation to the mucous membrane, and though, metaphorically speaking, rivers of these solutions were turned through it, still it would not be germ free. Fig. 61 shows some of the various forms of bacteria found in the human mouth.

Wounds, however, of the soft tissues of the mouth, in healthy persons of cleanly habits, very rarely suppurate; but when associated with compound fractures of the jaws, the cases are rare in which suppuration does not occur. This difference is largely due to the great difficulty in securing or maintaining an aseptic condition.

Arrestation of Hemorrhage.—Hemorrhage is from three sources, viz: from the *Arteries*, *Veins*, and *Capillaries*.

Arterial Hemorrhage is characterized by the bright red color of the blood, by its flowing in jets from the wound, and by the arrestation of the bleeding by pressure above the wound upon the arterial trunk.

Venous Hemorrhage is known by the dark color of the blood, its steady flow, and its welling up from the bottom of the wound, while pressure below the wound, upon the venous trunk, arrests the hemorrhage.

Capillary Hemorrhage is recognized by the oozing of the blood from the surfaces of the wound.

Primary Hemorrhage is the bleeding which takes place at the time of the injury.

Secondary Hemorrhage is the bleeding which comes on after reaction is established, or later, from the sloughing of ligated vessels and other tissues.

Internal Hemorrhage is the bleeding which takes place in the various cavities of the body.

Extravasation is the escape of blood into the connective tissue.

Severe hemorrhage produces marked constitutional effects, characterized by a feeble, fluttering, rapid pulse, which is in the later stages only recognized in the large arteries. The lips are colorless, and the

surface of the body is blanched, cold, and clammy. The respiration is slow and sighing. Faintness and nausea are prominent symptoms, accompanied by great restlessness, roaring in the ears, and darkness before the eyes. A fatal syncope may follow, or the patient may recover. Fever usually follows recovery from a severe hemorrhage.

Sudden and severe hemorrhage is more likely to prove fatal than one that is slow and continuous. Infants are much more seriously affected by the loss of blood than older persons.

The complete arrest of all hemorrhage is an important factor in the healing of wounds. Blood-clots between the lips of a wound are objectionable, for the reasons: First, that they prevent perfect apposition of the wounded tissues; second, they are excellent soil for the growth of micro-organisms; third, by their presence they cause tension and pain.

There are various methods of arresting hemorrhage, viz: By ligation, torsion, pressure, cautery, heat, cold, position, acupressure, forcipressure, styptics, and constitutional treatment. The most common method of arresting arterial or venous hemorrhage is by *ligation.* This is accomplished by picking up the end of the bleeding vessel with the hemostatic forceps, and tying a ligature around it. Small arteries may be controlled by *forcipressure*—crushing the end of the artery by grasping it with the hemostatic forceps—or by *torsion;* this is accomplished by twisting the vessel while in the grasp of the forceps. *Acupressure* consists of passing a pin or needle under the vessel, bringing the ends of the instrument above the external tissues, and causing pressure by passing a ligature around the ends in the form of the figure 8.

Severe hemorrhage should be checked, when possible, by pressure upon the arterial trunk, above the wound, by a tourniquet, or an Esmarch bandage (rubber bandage), until the severed arteries in the wound can be ligated.

In wounds of the face, appliances of this character have no place; consequently the bleeding vessels must be gathered up and secured as quickly as possible by ligatures. If, however, the vessels cannot be reached, as sometimes happens in compound and comminuted fractures, or following operations for the removal of malignant tumors, etc., they may be controlled by ligating the external carotid artery. Some surgeons prefer to take this precaution prior to performing any capital operation upon the maxillary bones. To the writer the latter procedure does not seem necessary, for in his personal experience the simpler methods have always proved sufficient.

Capillary hemorrhage may be controlled by the application of a jet of cold water thrown upon the bleeding surfaces, or by hot water applied with sponges or compresses. When the wound is in an ex-

tremity, the elevation of the limb will often control this form of hemorrhage. If the foregoing measures fail, then recourse must be had to the actual cautery, electro-thermal cautery, or styptics. The latter, however, are to be avoided when possible, as they increase the dangers of secondary hemorrhage, by causing sloughing of the cauterized tissues, and preclude the possibility of immediate union.

When capillary hemorrhage or oozing is persistent, the administration of ergot will often prove useful in checking it. The fluid extract may be given in doses of 20 to 30 drops every twenty minutes until three doses are taken, or the wine of ergot may be substituted, dose fl ʒ j to ij. Care must be taken in administering ergot to pregnant women not to bring on contractions of the uterus.

Coaptation.—*Accurate approximation of the surfaces is of prime importance in the healing of wounds*, and the surgeon should be prepared not only to bring the lips of the wound together, but to unite tissue to tissue. If a nerve-trunk or tendon is divided, it should be brought together by means of a sterilized animal suture. In deep wounds, the lower portions should be brought together by means of deep sutures,—sutures of approximation. The buried suture of sterilized catgut or silk is also used to bring together the deeper parts of the wound. The edges of the wound are to be closed by superficial sutures of the same material.

Superficial wounds are closed by adhesive plaster or collodion dressings.

Drainage.—In all deep wounds *drainage must be provided for*, as the exuded serum is usually considerable in amount, and if it has no way of escape, produces tension and pain, and offers a fertile soil for the growth of micro-organisms.

Drainage is secured by sterilized rubber tubing, decalcified chicken-bone, glass tubes, strands of catgut, silk, horse-hair, or strips of gauze.

Drainage-tubes are rarely ever necessary in the region of the face. The use of drainage-tubes increases the liability to the formation of scars, hence they should be dispensed with as far as possible in operations upon the face. In capital operations, traumatic wounds, and operative wounds involving the antrum of Highmore, drainage-tubes often become necessary.

Physiological Rest.—Rest of the injured part, and of the entire body, is imperatively demanded in the treatment of all serious wounds, and is of great value in all classes of wounds.

Rest in great measure prevents inflammation, as taught by Hilton.

The position of the injured part should be such as to favor a normal blood-supply, while immobility adds greatly to the rapidity of the healing process.

Wounds in parts which on account of their particular function it is difficult to keep at perfect physiological rest,—like surgical wounds of the lip, following operations for hare-lip and cleft palate in nursing infants,—do not always give as perfect results as do wounds in other locations that can be controlled. Anything like perfect rest of the lips or palate in such children is an impossibility; feeding and crying are among the principal occupations of the normal baby when not asleep. The motions incident to these functions more or less disturb the process of healing, and as a consequence the best results are not always obtainable.

Dressings.—The character of the dressings and their adjustment are matters of considerable consequence in the healing of wounds. In these days of antiseptic surgery, no intelligent surgeon would think of using other than aseptic dressings.

The main objects of dressings are, to support the wounded tissues during the process of healing, to absorb the discharges, and to prevent the ingress of all substances to the injured tissues which cause or favor septic infection. Antiseptic dressings, like the plug of cotton inserted into the mouth of the test-tube filled with sterilized bouillon, obstruct the entrance of the septic micro-organisms found in the dust and atmosphere almost everywhere, and thus prevent the establishment of putrefactive fermentation.

Wounds, like the unprotected bouillon when exposed to the atmosphere, soon become the seat of colonies of septic bacteria. If the resistance of the tissues is impaired, or the environment of the wound is such as to favor their rapid development, the tissues are soon overwhelmed, the reparative process is impeded or entirely suspended, the leucocytes and embryonal cells lose their vitality, and suppuration is the result; while, on the other hand, these conditions do not obtain if the wound has been dressed according to the best aseptic methods.

The adjustment of the dressings must always be governed by the extent, location, character, surroundings, and seriousness of the wound. Thicker dressings are required in cases with copious discharges than in those with little or no discharge. The covering, however, should always be sufficiently thick to protect the wound from infection from the outside.

In applying the dressings, care must be exercised not to produce uncomfortable pressure, as this soon becomes painful, and may establish inflammatory symptoms from mechanical irritation, and thus jeopardize the healing of the wound.

In operations upon the lips and face impervious dressings are the best; the collodion dressing has the preference.

REVIEW.

CHAPTER XVIII.

What does the treatment of wounds consist of?

What is the first consideration in the treatment of wounds?

How are the surfaces of the body about the wound to be treated to render them aseptic?

How are wounds of the mouth to be treated to obtain the same condition?

Can the human mouth be made germ free?

Do all wounds of the soft tissues of the mouth in healthy persons suppurate?

What is the result when associated with fractures of the jaw?

What are the three sources of hemorrhage?

How can you determine arterial hemorrhage?

How determine venous hemorrhage?

How determine capillary hemorrhage?

What is primary hemorrhage?

What is secondary hemorrhage?

What is internal hemorrhage?

What is extravasation?

What are the constitutional effects of severe hemorrhage?

What may be its termination?

What class of individuals are most affected by severe hemorrhage?

Why are blood-clots between the lips of wounds objectionable?

By what methods is hemorrhage arrested?

What is ligation?

What is forcipressure?

What is torsion?

What is acupressure?

By what means is hemorrhage arrested during operations upon the extremities?

Are these means applicable in wounds of the face?

What means are used as a last resort to control hemorrhage?

What are the means usually adopted to control capillary hemorrhage?

How are wounded surfaces to be approximated?

What is the object of providing drainage?

What substances are used for drainage?

Are drainage-tubes ever required in wounds of the face?

What is the value of physiological rest in the treatment of wounds?

What should be the character of all dressings?

What are the main objects of dressings?

How do dressings prevent the infection of wounds?

What conditions of the tissues favor the rapid development of septic bacteria?

What should govern in the adjustment of dressings?

What conditions must be guarded against in the application of dressings?

What dressings are generally preferred in wounds of the lips and face?

CHAPTER XIX.

GUNSHOT WOUNDS.

UNDER the term Gunshot are included all those wounds which are caused by projectiles that have been propelled by the elastic or explosive power of gunpowder, dynamite, nitro-glycerin, etc. To the injuries inflicted by these missiles may be added the wounds caused through the concussions of the explosives themselves by fragments of wood, iron, or stone, by portions of the body of a comrade,—pieces of bone or teeth,—or portions of accoutrements or clothing. The projectiles used by civilized nations in modern warfare are buckshot; bullets, round and conical; shrapnel, grape and canister, chain or bar-shot; solid cannon-balls, shells, slugs, explosive musket-balls, hand-grenades, and torpedoes.

These missiles are projected with great velocity and force. The wounds, therefore, which are inflicted by them are classed as contusions, lacerations, penetrations, perforations, simple fractures, partial fractures, complete fractures, with various degrees of comminution and destruction of substance.

The great majority of gunshot wounds are produced by the ball or bullet, either the round or conical. At the commencement of the War of the Rebellion, both opposing armies were obliged to employ the round ball to a greater or less extent, on account of the construction of the firearms. Later, through the introduction of the rifle arm, the conical bullet superseded the round ball. The rifled arms most commonly used were the Enfield, the Austrian, and the Springfield. The Enfield carried a shot weighing 450 grains; the Austrian, one weighing 460 grains, and the Springfield, one of 500 grains.

Hamilton says, "Some idea of the velocity and power of the conical shot can be obtained from the following statement: When fired from a Springfield rifle, with a charge of 60 grains of powder, at 200 yards, it will penetrate eleven one-inch pine planks, separated by intervals of one and one-half inches; while at a distance of 1000 yards it will penetrate one such plank, and enter the second to the depth of one-quarter of an inch."

The preponderance of gunshot injuries over other wounds during the late war is shown by the report of the Surgeon-General of the

army. There were treated in hospitals during that period 246,712 cases of wounds caused by weapons of war. Of these, 245,790 were gunshot wounds and 992 were bayonet and sabre wounds. And from the same report it is found that by far the greater number of gunshot injuries were inflicted by projectiles from small arms, the rifle or pistol.

The number of cases in which the nature of the missiles was with certainty ascertained was 141,961. Of these, 127,929, or 90.1 per cent., were caused by shot from small arms.

Among the casualties of the late war, there were recorded as occurring from explosive bullets 130 cases of wounds. Injuries from this form of projectile are much more serious than from the ordinary round or conical bullet. The destruction of the soft parts is greater, the bony parts are more extensively shattered, hemorrhage, both primary and secondary, is much more common, sloughing of an extensive character is more likely to occur, and the process of repair is slow and unsatisfactory.

Diagnosis.—Small fragments of exploded shell, case and canister shot, produce effects which do not materially differ from the injuries caused by missiles from small arms.

Large fragments of shell produce great laceration and destruction of tissues. When of sufficient size and velocity, they may carry away a portion of the trunk, or an extremity. Under such circumstances the laceration and contusion may be very great.

Wounds made by conical bullets are frequently of irregular shape at the points of entrance and exit. Sometimes the entrance is indicated by a mere slit, or it may be irregularly round or oval, or broadly lacerated; while the wound of exit is always larger and still more irregular. The track of the wound also gradually increases in size as it reaches the point of exit, thus giving it a somewhat conical form.

Wounds caused by round balls are quite generally round, the wound not larger than the missile; the surface depressed at the point of entrance, and the edges discolored. The wound of exit is irregularly round, and somewhat larger, while the surface is elevated and the edges everted. The round ball is not so destructive to tissue as the conical shot, which, on account of its increased weight and velocity, meets with little resistance from any of the structures of the body at ordinary range, and is seldom deflected from a straight line, crushing, tearing, and comminuting everything in its path. The wounds of entrance and exit will correspond in most cases with the line of projection. Hamilton mentions a peculiar exception to this rule, to be found in the Army Medical Museum at Washington: "A conical ball, marked 4622, entered the thoracic parietes on one side, made a semi-circuit of the body, and emerged at a point corresponding to the place of entrance on the opposite side."

Round balls, from their shape and decreased velocity, are more liable to be deflected from the line of projection at the point of contact with the surface of the body, or of their passage through the various tissues.

Leaden bullets, when they come in contact with bony tissue, often split into fragments, the pieces tearing through the tissues at a tangent from the line of projection, causing several wounds of exit. When the momentum is sufficient to cause penetration of bony tissue, they are usually badly comminuted or more or less splintered.

Experiments made with steel bullets by the Army Medical Department, and exhibited at the World's Fair at Chicago, proved conclusively that projectiles made of metal which retained its form, in passing through bone, would in a majority of cases, under the average momentum, penetrate without fracture, and rarely fissure or comminute this tissue.

It is to be hoped, for the sake of humanity, that this form of small-arm projectile may be universally adopted by civilized nations. By the adoption of such a missile a great amount of suffering could be prevented, and limbs and lives saved that are now sacrificed as a result of the terrible destruction wrought by the lead projectile.

Gunshot Wounds of the Face.

Many of the bones of the face are so thin and shell-like, and the parts so exceedingly vascular, that gunshot injuries are likely to cause great comminution of the bones, and serious hemorrhage, both of a primary and secondary nature.

Laceration of the soft tissues, with hemorrhage of a more or less serious character, is a constant accompanying feature of gunshot injuries of the face, while the mortality is exceedingly high, both from the immediate effects of the injury and from secondary hemorrhage.

According to the report of the Surgeon-General of the Union Army, the principal cause of fatalities from injuries of this class was secondary hemorrhage, and this seems to be the general opinion of all surgeons who have had any considerable experience in military surgery.

The fact that secondary hemorrhage is so common in gunshot injuries of the face is explained by the inaccessibility of the vessels to ligature, while ligation of the carotid does not always prove successful in preventing it on account of the free anastomosis with the vessels of the opposite side, and of the same side.

During the War of the Rebellion, there were reported 9416 gunshot injuries of the face. Of these, 4914 were flesh wounds, with 3706 recoveries, 58 deaths, 1150 undetermined results, and 1.5 percentage

of fatalities. The remaining 4502 were complicated with fractures of the bones; 3700 recovered; 404 died; 398 results undetermined, and percentage of fatalities, 9.8.

The mortality is always very much higher in all cases of gunshot injuries complicated with fractures of the bones. The effects of the injuries from the modern firearm, with its heavier missile, greater velocity, and surer aim, contrast very unfavorably, from this standpoint, with the round bullet and smooth-bore musket.

During the Crimean War there were 533 wounds of the face; 107 were complicated with bone injury; 445 recovered, and reported for duty; 74 were invalided, and 14 died; percentage of fatality, 3.8.

Wounds of the face from gunshot injuries often cause great disfigurement from the loss of tissue; but as a rule, laying aside the fatalities from the immediate injury and secondary hemorrhage, they generally do well; the soft tissues heal kindly, and it rarely happens that there is any extensive necrosis of the bones.

Solid shot and fragments of shell striking the face usually prove fatal, but occasionally an individual will survive after having a considerable portion of the face carried away. Heath mentions some very interesting cases, which it will pay the student who is specially interested in such matters to carefully read.

Bullets penetrating the face sometimes lodge in the antrum or nose, and remain there for a considerable time, and may finally become encysted.

Gunshot wounds of the superior maxilla, occurring through the mouth, are usually the result of a suicidal intention, though such injuries do occasionally occur from accidental causes, and are usually immediately fatal. Sometimes, however, the injury is not very severe, and the patient recovers. Fig. 62 is the cast of a case from the writer's collection, showing result of gunshot injury of the upper jaw from the accidental discharge of a shot-gun.

Missiles striking the lower jaw invariably produce fracture, and often considerable comminution and loss of tissue, while hemorrhage is quite likely to be severe, especially when the facial artery is involved in the wound.

Symptoms.—The immediate symptoms of gunshot injuries are pain, shock, and hemorrhage.

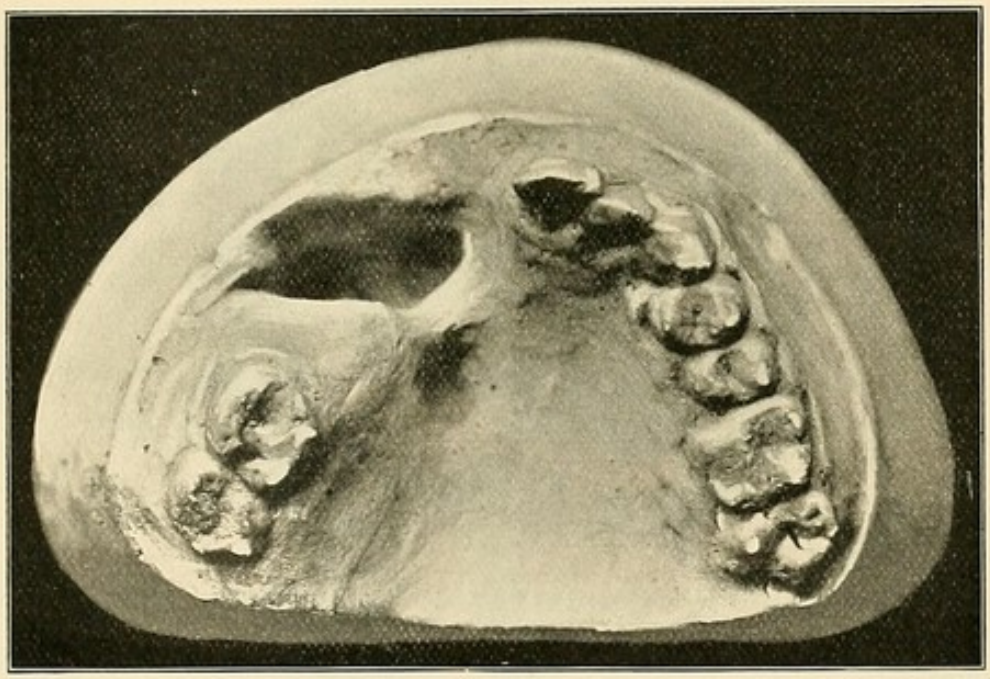
Pain as a first symptom is rarely absent. The degree of pain will depend upon the location of the wound, its nature, the tissues involved, and the mental condition of the individual. Nerves when injured or contused are productive of severe pain, which is referred generally to the region supplied by the injured nerve. When a nerve is completely divided, there is a total loss of sensation in the part supplied, and more or less complete paralysis of motion.

The degree of shock depends upon the temperament of the individual, the physical condition at the time of the injury, and the region of the body in which the wound has been received.

In wounds of the abdomen, shock is more profound and persistent than in wounds in any other part of the body. Injuries which produce considerable splintering and comminution of the long bones are usually followed by shock of severe degree.

Hemorrhage will be slight or severe, according to the extent of the injury to large blood-vessels and the vascularity of the soft tissues involved. Wounds of large arterial trunks speedily terminate fatally.

FIG. 62.



RESULT OF GUNSHOT INJURY OF THE UPPER JAW.

Treatment.—The first thing to be done in gunshot wounds of the face is to arrest the hemorrhage if it is in any way alarming.

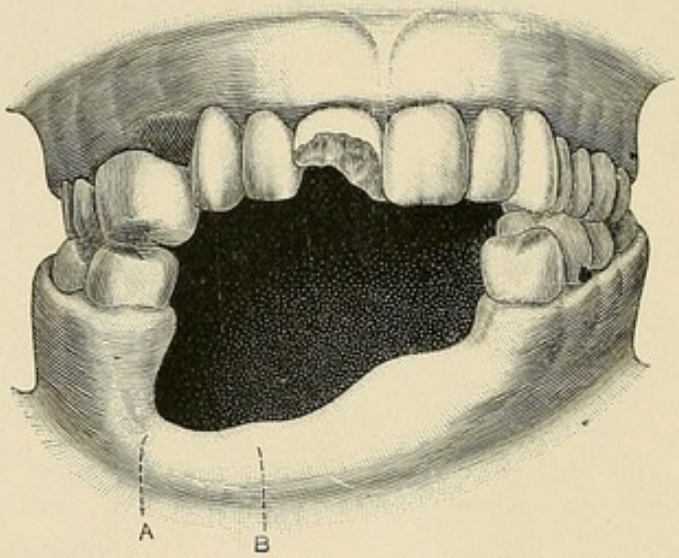
The hemorrhage may be controlled by tying the bleeding arteries where possible to reach them, and, when inaccessible, by packing the wound with antiseptic gauze; where packing cannot be utilized, as in the case of large surface wounds, or where portions of the face have been carried away by fragments of shell, stone, etc., recourse must be had to styptics. Compresses, wrung out of hot antiseptic solutions preferably, should be applied as hot as can be borne. Persulfate of iron is frequently used in these cases, but it has the disadvantage of occasionally causing extensive sloughs, and is sometimes followed by secondary hemorrhage.

The missile should be searched for and removed as soon as pos-

sible, together with all detached fragments of bone, and foreign substances, which may have entered the wound. Loose fragments of bone which are still attached to the soft tissues should under no circumstances be removed, but placed as nearly as possible in their normal positions, and retained by means of sterilized packing or other suitable support.

Bullet injuries of the upper part of the face, which at first seem likely to result in extensive deformity, often recover with so little disfigurement as to be a surprise to all concerned, provided care has been taken to preserve every fragment of bone which, by reason of attachment to the soft tissue, gives hope of sustaining its vitality and making a union with its fellows.

FIG. 63.



GUNSHOT FRACTURE OF LOWER JAW WITH LOSS OF BONE FROM A TO B. TREATED BY INTERDENTAL SPLINT BRIDGE. (After Patterson.)

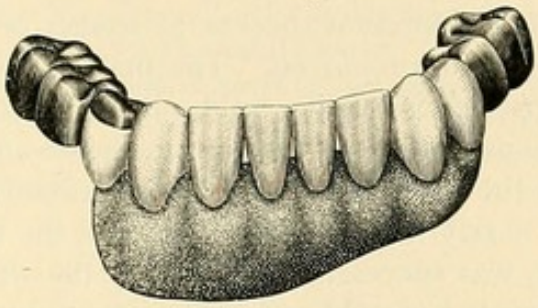
Gunshot injuries involving the lower jaw are usually complicated with multiple fractures, great comminution, and many times with extensive loss of bone-tissue, which was carried away. The successful management of these cases will depend largely upon the skill and the inventive genius of the surgeon. A wise conservatism, however, is nowhere of greater value than in the treatment of this class of injuries.

In those cases where there has been a considerable loss of bone-tissue, it is important that some kind of a support be applied to prevent the free ends of the jaw from falling together, which would otherwise cause a serious disfigurement, and destroy the occlusion of the remaining teeth.

When teeth remain on either side of the gap, gold or platinum bands can be fitted to them, and extension and immobility secured by soldering a gold wire to the approximal surfaces of the bands, and the appliance cemented to the teeth with the ordinary oxyphosphate cement. To provide for further extension or approximation, the wire

can be divided in the center, and be fitted with a double screw nut, and the appliance lengthened or shortened at the will of the operator. Figs. 63, 64, illustrate a case of this character reported by Patterson, in which a large fragment of bone was lost by a gunshot wound, and the contour of the jaw restored and the remaining fragments held in a normal position by an interdental splint bridge cemented to the remaining teeth.

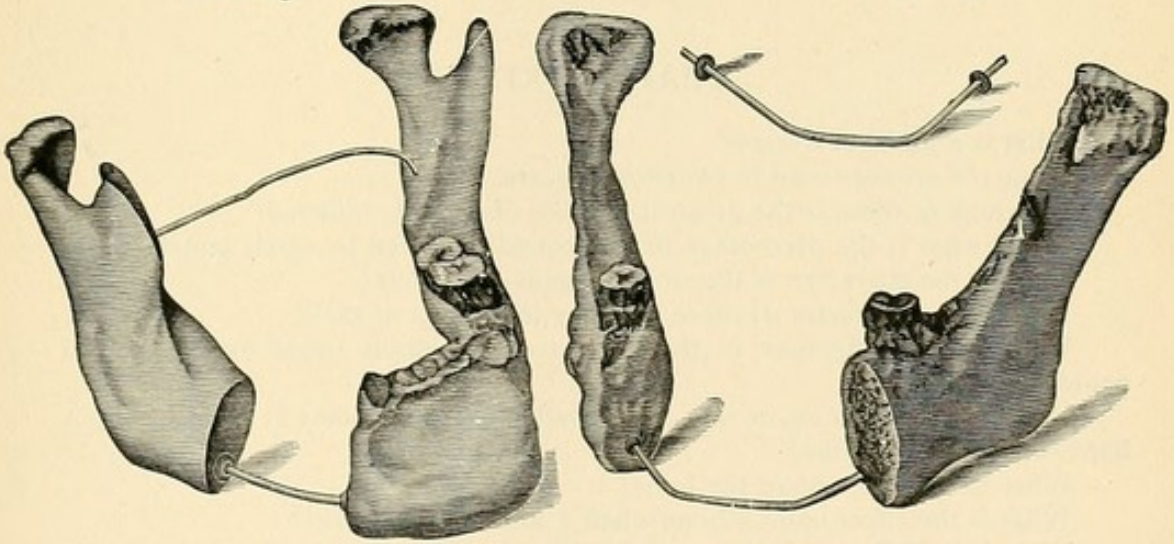
FIG. 64.



INTERDENTAL SPLINT BRIDGE. (After Patterson.)

If the jaw is edentulous, the same result may be obtained by introducing a gold wire of the proper length, having a collar of the same material attached one-eighth of an inch from each end, and this inserted between the free ends of the jaw, holes having been made in them for the reception of the wire, and the tissues closed over it, as

FIG. 65. FIG. 66.



PROSTHETIC WIRE ARCH FOR PARTIAL RESECTION OF LOWER JAW. (After Cervera.)

suggested by Cervera, Figs. 65, 66. Such an appliance will generally become encysted, and is then not likely to give future inconvenience.

After the wound has healed and the cicatrix hardened, the gap may be filled with a suitable piece of bridge-work, or a removable denture.

Loss of extensive portions of the superior maxilla is a not infre-

quent result of gunshot injuries upon the field of battle, from accidents while hunting, and from attempts at suicide.

Restoration of the contour of the face can many times be accomplished—when the soft parts are intact, or can be made so by a plastic operation—by the construction of supports to the soft tissues representing the portions of lost bone, and attaching them to the remaining teeth.

Attempts have been made to bury such appliances in the soft tissues, but with only indifferent success, as sooner or later they cause ulceration, and have to be removed. The materials which have been used for this purpose are vulcanite, gold, platinum, and aluminum.

One such case as first described, in which the superior maxillary bone was lost from the median line back to the second molar, involving the palate process nearly to the median line, and the body of the bone to the orbital plate, was successfully treated by the writer by means of an appliance constructed of gold and vulcanite combined, and retained in position by clasps attached to the remaining teeth.

The discussion of fractures of the maxillary bones and their treatment will be reserved for a later chapter.

REVIEW.

CHAPTER XIX.

What is a gunshot wound?

Name the missiles used in common warfare.

By which of these is the greatest number of wounds inflicted?

About what is the percentage of the wounds inflicted by small arms?

What is the character of the wounds made by bullets?

What is the character of those made by fragments of shell?

What is the difference in the character of wounds made by round and conical bullets?

What is likely to occur to a leaden bullet when it comes in contact with a hard substance like bone?

What is the effect upon the bone?

What is the effect upon a bone when a steel bullet is used?

What is the effect of bullet wounds of the face?

What is the principal cause of fatality?

Why does not tying the carotid artery always prove successful in arresting secondary hemorrhage?

What complication in gunshot injuries increases the mortality?

What may become of bullets that lodge in locations where they cannot be found or removed?

What are the immediate symptoms of gunshot injuries?

In what class of wounds is shock most severe?

What is the line of treatment in gunshot injuries of the face?

What means can be used to arrest the hemorrhage?

What is the danger from the use of the persulfate of iron?

What should be done with loose fragments of bone?

What is a frequent result of gunshot injuries of the upper part of the face?

How may this result be obtained?

What is a usual result in gunshot injuries of the lower jaw?

What line of treatment should be followed in these cases?

What are some of the methods that may be used to maintain the normal position of the lower jaw when a fragment has been carried away?

What becomes of the inclosed metal support?

What methods can be used to close the gap after the tissues have healed?

How may restoration of contour be secured artificially?

What success has attended the effort to restore the contour of the superior maxilla by buried metal appliances?

CHAPTER XX.

FRACTURES OF THE INFERIOR MAXILLA.

Definition.—*Fracture* (Lat. *fractura,* a break). The breaking of a bone, either by external force or by the action of the muscles of the body.

Fractures of the jaws are of quite common occurrence, and are generally the result of blows upon the face from the fist, kicks of large animals, the impact of some heavy missile propelled with considerable velocity; gunshot injuries; the extraction of teeth; a fall from a bicycle, a horse, a building, or other considerable height; passage of a wheel over the face; injuries from passenger or freight elevators, or other crushing force.

Fractures of the superior maxilla are much less frequent than fractures of the inferior maxilla, on account of the fact that the superior maxillary bones are, by reason of their location and shape, less exposed to injury. When fractures of these bones do occur, they are generally the result of a severe traumatism.

The inferior maxilla, from its size, shape, and location, is more often fractured than any other bone of the face. In size, it is the largest of all the bones of the face. Its shape makes it liable to fracture in the anterior portion or at the angle, when blows are received upon the side of the face, and through the ramus or at the neck of the condyle when the blow is received upon the chin.

The weakest point of the lower jaw is just anterior to the mental foramen, through the alveolus of the cuspid tooth. In edentulous jaws this weak point would be through the mental foramen (Fig. 67).

Its location is exposed, and it is therefore more liable to receive an injury than those parts which are better protected. Fractures of the lower jaw are ten times more frequent in males than in females. This is largely due to the difference in the occupations and the degree of exposure to accident between the sexes.

Fractures of the maxillary bones are classed under two general forms, viz: *Simple* and *Complicated.*

Simple fractures are those in which there is a single fracture of the bone, without injury or break in the continuity of the external tissues.

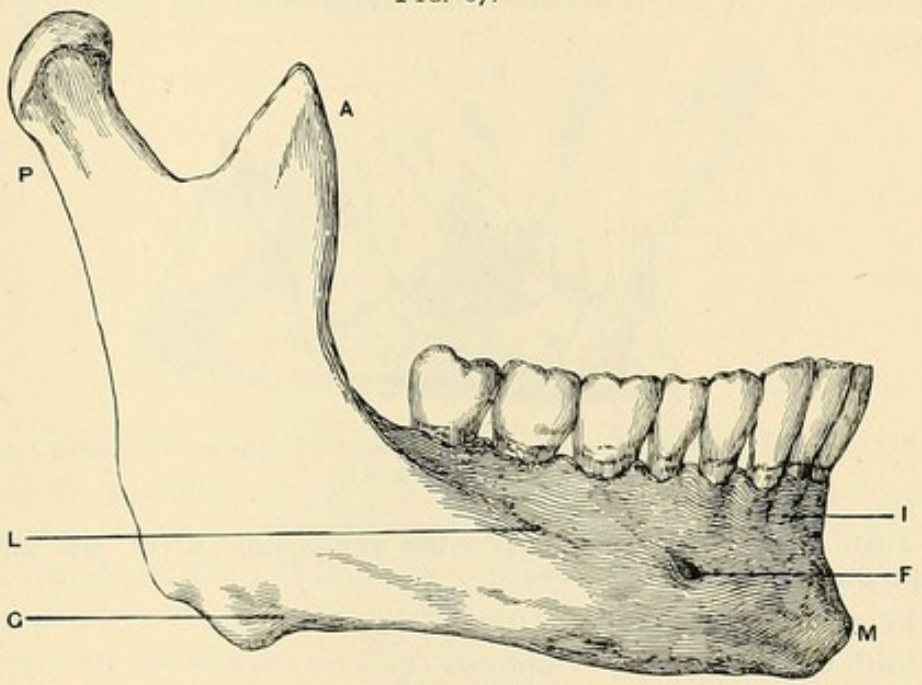
Complicated fractures include all other conditions associated with a fracture of the bone, such as injuries to the external tissues, to vessels, to nerves, to teeth, to a comminuted condition of the bone itself, or any other condition which complicates a simple fracture.

Complicated fractures may be divided into *Multiple, Comminuted,* and *Compound.*

Multiple fractures include those in which there is more than one break in the continuity of the bone. Double and triple fractures would be classed under this head.

Comminuted fractures are the result of crushing injuries or gunshot wounds, which cause splintering and crushing of the bone into small fragments.

FIG. 67.



THE INFERIOR MAXILLARY BONE, EXTERNAL SURFACE OF THE RIGHT SIDE.

M, Mental process; I, Incisive fossa; F, Mental foramen; L, External oblique line; G, Groove for facial artery; A, Anterior or coronoid process; P, Posterior or condyloid process.

Compound fractures are those which have associated with them injuries of the soft tissues, causing exposure of the fractured ends of the bone.

Fractures of the Alveolar Process.—The most common fractures of the jaw are those of the alveolar process, and they are generally associated with the extraction of teeth. These fractures rarely involve more of the process than the external plate lying immediately over the roots of the tooth extracted, and perhaps a small portion extending over those adjacent to it.

Fractures of the alveolar process frequently occur as a result of falls or of blows upon the chin which have an upward direction, driving the teeth into their sockets, and splitting the process on a line with the

alveoli. These accidents most frequently occur among men engaged in the building trades, as the result of falls from buildings and scaffolding.

When this accident occurs to the upper jaw, the external plate of the alveolar process is usually the part to give way; the internal plate is supported by the palate process, hence its greater power of resistance. When the same accident occurs to the lower jaw, the external and internal plates of the process are usually both fractured and separated.

Fractures of the Body of the Lower Jaw.—Fractures of the body of the bone most frequently occur as follows:

First. In the region of the cuspid tooth (Fig. 68, 1).

Second. At points between the cuspid tooth and the angle of the jaw (2).

FIG. 68.



FRACTURE OF THE BODY, CONDYLES, AND CORONOID PROCESS OF THE LOWER JAW. (After Fergusson.)

Third. In locations between the symphysis of the jaw and the cuspid tooth (3).

Fourth. At the angle of the jaw (4).

Fifth. Through the symphysis (5).

Sixth. At points through the ascending ramus (6).

Seventh. At the neck of the condyle (7).

Eighth. Through the coronoid process (8).

Fractures located above the angle are exceedingly rare. Out of fifty-five cases of fracture of the lower jaw reported by Hamilton, only three were above the angle.

Fractures of the body of the jaw, through the cuspid, bicuspid, or molar regions, the angle or symphysis, are usually the result of injuries received upon the side of the face, while fractures of the ascending ramus and the neck of the condyle are usually caused by injuries received upon the chin,—as blows or falls. Out of the fifty-five cases just mentioned as reported by Hamilton, four only were through the symphysis. The great majority of the fractures of the lower jaw are compounded, generally into the mouth.

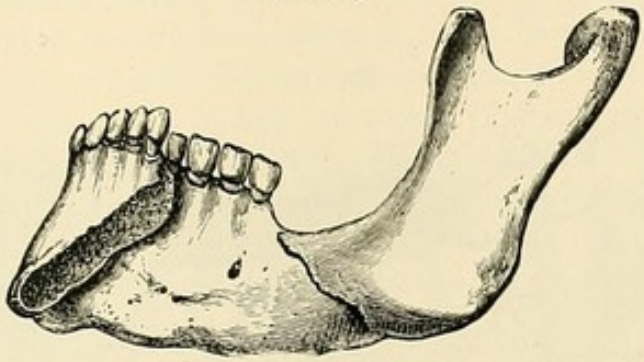
Displacements.—In fractures through the symphysis, displace-

ment is usually very slight, on account of the attachment of the muscles of the lateral halves of the jaw, and their equalized action. Such a fracture may occur and not be recognized by the patient, except from the crepitation produced when attempting to masticate food.

In fractures at the neck of the condyle, the displacement, as a rule, is not very great. The direction of the displacement when it does occur will be forward, on account of the action of the external pterygoideus, which drags the body in this direction.

Fractures at the symphysis, through the ascending ramus, or the neck of the condyle, are rarely compounded into the mouth; fractures at these locations are, however, sometimes compounded externally from laceration of the covering tissues at the point where the injury was received.

FIG. 69.



FRACTURES OF THE LOWER JAW WITH DISPLACEMENT. (After Malgaigne).

Simple fractures are not subject to the same amount of displacement as are multiple and compound fractures.

A simple fracture through the cuspid regions will not cause so great a displacement as when the same character of fracture is compounded into the mouth, for the covering tissues combat to a certain extent the tendency of the muscles to draw the ends of the fractured bone out of position. When the continuity of the covering tissues is broken, the muscles have full play, and cause a displacement commensurate with the location and the character of the injury.

Multiple fractures are subject to the greatest amount of displacement. Fractures occurring upon both sides of the jaw at the same time always present the greatest degree of displacement.

In fractures occurring in the anterior portion of the jaw, and upon both sides, through the cuspid or bicuspid region, the central portion would be dragged downward and backward by the action of the genio-hyoid, genio-hypoglossus, and digastric muscles.

When the fracture occurs at the cuspid region, and through the body or ascending ramus, the intermediate fragment will be displaced inward by the action of the mylo-hyoideus, upward by the masseter, and forward by the action of the external pterygoideus (Fig. 69).

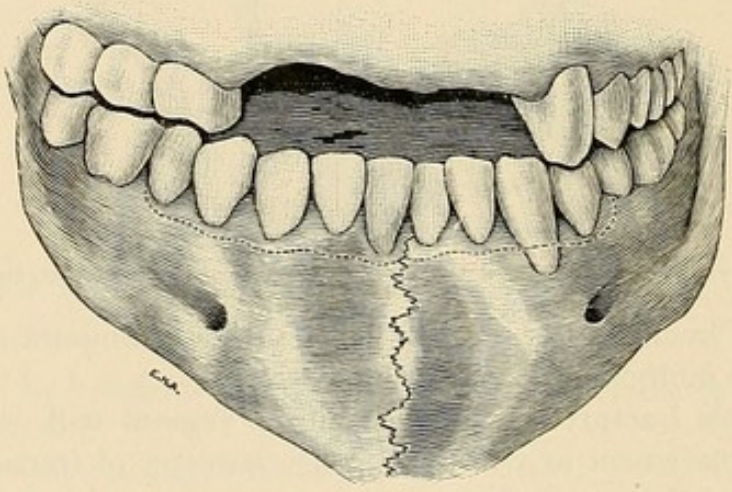
In single compound fractures occurring in the region of the cuspid tooth, or just anterior or posterior to it, the displacement is sometimes considerable.

Lines of Fracture.—The lines of fracture may be vertical, oblique, or horizontal. It sometimes occurs, however, that the lines of fracture may be combined, as, for instance, vertical and oblique, vertical and horizontal, double oblique, etc.

When the fracture is through the symphysis, it is almost always vertical (Fig. 70). In the four cases of this class reported by Hamilton, the fractures were all vertical. Two cases seen by the writer, occurring in elderly people, were both vertical.

Fractures of the alveolar process are generally vertical and oblique combined; as, for instance, when the external plate of the process is split off in the extraction of teeth, or as the result of upward blows upon the chin.

FIG. 70.



FRACTURE THROUGH THE SYMPHYSIS. (After Angle.)

The great majority of the fractures of the body of the bone, however, are oblique. According to Malgaigne, the thickness of the bone is also divided obliquely, so that generally the fracture occurs at the expense of the internal plate of the anterior fragment and the external plate of the posterior fragment. When the fracture is very oblique, there is usually considerable overlapping and locking, making reduction sometimes very difficult, as in Fig. 69.

Out of forty cases of fracture of the body of the bone reported by Hamilton, eighteen were demonstrated to be single oblique, and thirteen double and triple fractures.

Nearly all fractures of the body of the bone have a perpendicular direction through the alveolar process. The direction of the line of fracture outside of this may be obliquely forward or backward. There is considerable difficulty, however, in positively demonstrating this fact

in the living subject, and as the mortality from this class of injury is very low, the opportunities for post-mortem examinations are rare.

The specimens preserved in the various museums would seem, however, to corroborate the general opinion that the majority of the fractures through the body of the bone are oblique. Heath mentions a case in the museum of King's College, London, as being obliquely forward and backward.

Symptoms.—The symptoms of fracture of the lower jaw are generally well marked, except in simple fractures through the symphysis. The special diagnostic signs of fracture are crepitus, more or less deformity in the contour of the lower part of the face, and unnatural or excessive mobility. Pain is always present, and is increased by the movements of the jaw. In the majority of the cases, the mucous membrane is lacerated, giving rise to more or less hemorrhage. The saliva is secreted in excessive quantity, and, being mixed with discharges from the wound, decomposes and causes a fetid odor of the breath. Changes in the normal occlusion of the teeth are generally well marked at the point of injury. The teeth upon either side of the fracture are commonly loosened, and sometimes entirely luxated. Erichsen mentions a case in which the tooth had been detached from its alveolus and become lodged between the fragments of the jaw, preventing adjustment of the fracture until it was found and removed.

Considerable inflammation, as a rule, follows fracture of the jaw, accompanied by swelling and infiltration of the face and neck, and followed not infrequently by troublesome abscesses and necrosis of splintered portions of the bone. This is explained by the fact that such fractures are generally compounded and often comminuted, making infection certain.

Among the possible complications of fractures of the lower jaw are hemorrhage from wounding the inferior dental artery, paralysis of the lower lip and chin from injury of the inferior dental nerve, salivary fistula, abscess, necrosis, septicemia, and pyemia.

Diagnosis.—The diagnosis of fractures of the lower jaw is generally a simple matter, but occasionally difficulty is experienced in locating the exact seat of the fracture, especially if it is a simple one, without displacement. When doubt exists, seat the patient upon a low chair, taking a position behind him; then grasp the jaw with both hands, the thumbs upon the ends of the teeth and the fingers underneath the chin, and test, by alternately depressing and elevating first one side, then the other. If fracture exists, crepitation will be discovered at the point of injury. If the fracture is through the coronoid process only, the diagnosis will be made from the inability of the patient to properly close the jaw upon this side.

Prognosis.—The prognosis of fracture of the lower jaw is, as a general rule, very favorable. The mortality from this injury is exceedingly low; a fatal termination would be due, in all probability, to other conditions arising as complications, of which septic poisoning would be an example.

Simple fractures of the lower jaw unite in from four to six weeks. Compound, multiple, and comminuted fractures are often retarded considerably beyond this period, two to three months not infrequently being required for a good union to be formed.

The callus formed about the fractured ends of the bone frequently causes considerable deformity; this, however, is only temporary, for it is eventually removed by absorption, and the contour of the face is restored to its original lines.

REVIEW.

CHAPTER XX.

What is the definition of fracture?

Do fractures of the maxillary bones often occur?

What are the common causes of fractures of the jaws?

Which of the jaws is most frequently fractured?

What reasons can you give for the greater liability of the lower jaw to receive such injuries?

What must be the character of an injury which will produce fracture of the superior maxilla?

Blows upon the side of the lower jaw would cause fractures in what locations?

What would be the location of a fracture caused by a blow or fall upon the chin?

Where is the weakest point in the lower jaw?

In which sex are fractures of the lower jaw most frequent?

What is the difference in frequency?

What are the causes of this difference?

Under what two general designations are fractures classed?

What is a simple fracture?

What is a complicated fracture?

How are complicated fractures classed?

What is a multiple fracture?

What is a comminuted fracture?

What portions of the maxillary bones are most often fractured?

What are the causes of fractures of the alveolar process?

Give the relative order of frequency of fractures in the various localities of the lower jaw.

What is the cause of the displacement of the fractured ends of the bone?

What form of fracture has the least displacement?

Which form has the greatest displacement?
What is the direction of displacement in fracture of the neck of the condyle?
What muscle produces this displacement?
What is the direction of displacement in fracture through the cuspid region?
What muscles produce this displacement?
What are the general lines of fracture?
What fractures are vertical?
What fractures are horizontal?
What fractures are oblique?
What are the symptoms of fracture of the lower jaw?
Name the possible complications of these injuries.
How can obscure cases of fracture be diagnosed?
What is the prognosis?
How long does it take for simple fractures to unite?
How long for the other varieties to unite?
What becomes of a callus that is formed in the process of union?

CHAPTER XXI.

FRACTURES OF THE INFERIOR MAXILLA (Continued).

Treatment.

Two conditions are absolutely necessary to the successful treatment of fractures, no matter where located:

First. Accurate adjustment of the fractured portions of the bone.

Second. Complete immobility of the parts until union has taken place.

Fig. 71.



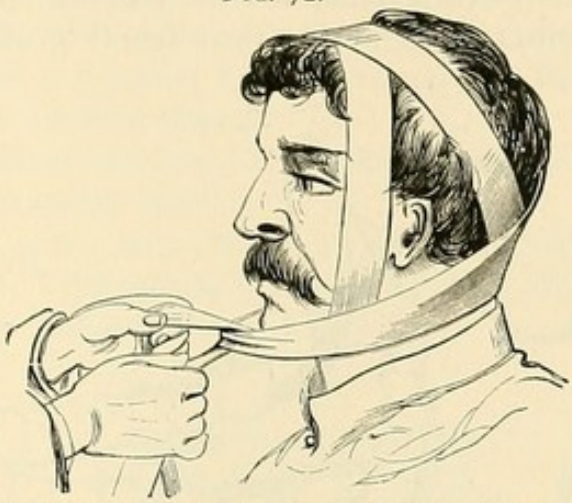
Four-tailed Bandage for Fracture of the Lower Jaw. (After Heath.)

Various methods and appliances have been introduced for the purpose of fixation of fractures in the lower jaw, from the simple four-tailed bandage to the most elaborate interdental splint. The particular method to be adopted in each individual case must be determined by the location and extent of the fracture, and the complications attending it. Fractures complicated with laceration of the soft tissues, or with hemorrhage, must be treated upon the common ground of wounds in general, viz: to arrest the hemorrhage, render the wound aseptic, and close the soft tissues; after which the fracture may be reduced and the

appliance adjusted which has been selected to maintain the immobility of the parts.

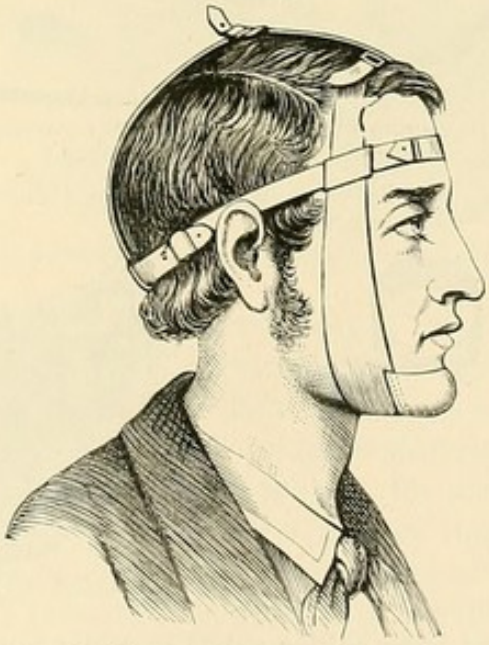
Simple fractures, with only slight displacement, may be reduced and usually maintained in position by a simple four-tailed bandage (Fig. 71), or the Barton or Hamilton bandages (Figs. 72, 73), some-

FIG. 72.



GARRETSON'S MODIFICATION OF THE BARTON BANDAGE. (After Garretson.)

FIG. 73.



HAMILTON BANDAGE FOR FRACTURE OF THE LOWER JAW. (After Hamilton.)

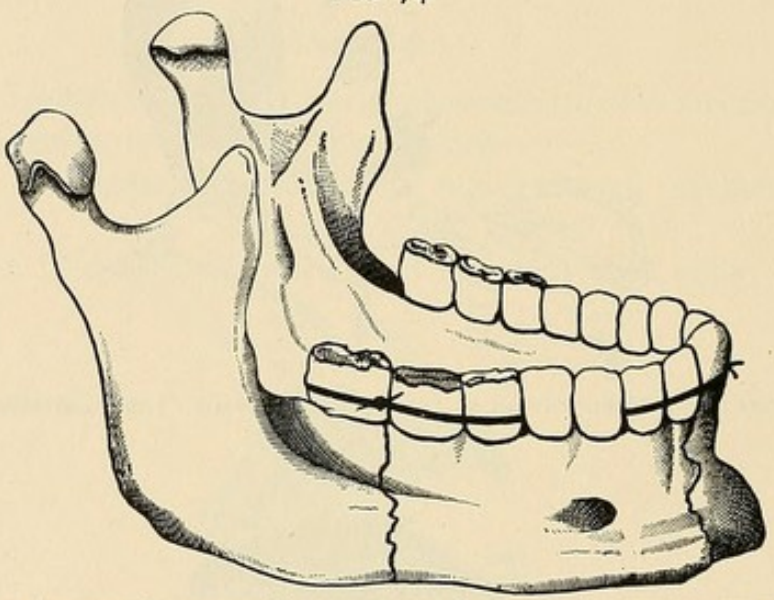
times combined with an external splint molded to the chin; or wires may be twisted around the firm teeth upon either side of the fracture (Fig. 74), the wires to be passed through the approximal spaces at the margin of the gums; but, better still, the Angle fracture bands and screws or wire.

The Angle appliance consists of platinum or German silver bands,

which pass around the teeth in the form of a loop and are retained in position by means of a set-screw, which passes through the tubes soldered to the ends of the band, and which are drawn together by the screw until it securely grips the tooth (Fig. 75).

One of these bands is fastened to a firm tooth on either side of the fracture; a screw is then passed through tubes prepared for its reception upon the side of each band, and the fracture approximated and maintained in position by tightening the screw (Fig. 76).

FIG. 74.



METHOD OF HOLDING DETACHED PIECES OF THE RAMUS IN APPOSITION WITH OTHER FRAGMENTS OF THE JAW. (After Vinke.)

FIG. 75.

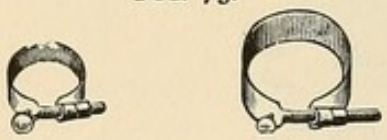
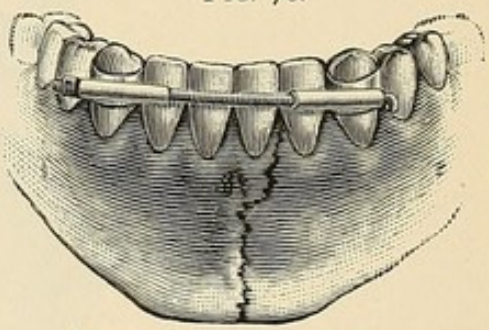


FIG. 76.



ANGLE APPARATUS APPLIED.

Another method is to solder small metallic buttons upon the side of the bands, and approximate the fractured ends of the bone by passing binding wire around the buttons in the form of the figure 8 (Fig. 77).

This method is applied to single fractures in all locations of the jaw, but in comminuted or multiple fractures, where the displacement is considerable, and difficult of reduction, there is constant danger of displacement, while the strain upon the teeth to which the bands are

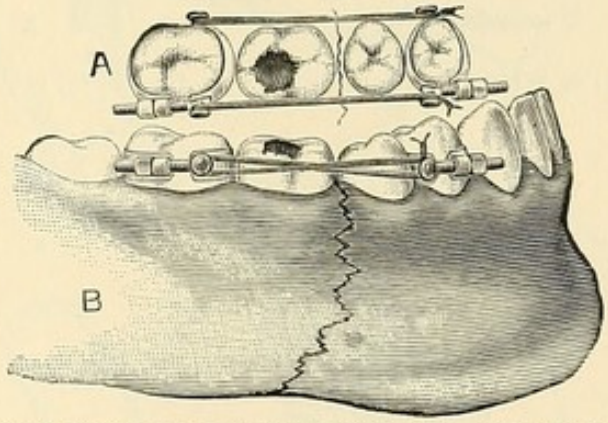
fastened soon loosens them in their alveoli, and they become worthless as points of anchorage.

In those cases where the teeth have been loosened or dislodged, or the jaw is edentulous, the above method of wiring and the Angle appliance cannot be used. Drilling the maxillary bone and wiring the fractured ends together is the best method in these cases. Wiring the fractured jaw at any point anterior to the first molar can be accomplished inside of the mouth, but in locations posterior to this, or in the ascending ramus, it becomes necessary to operate from the outside, by laying open the external tissues.

Two considerations, however, should be constantly borne in mind in all operations on the jaws:

First. To avoid any cutting operations upon the external tissues of the face, if the operation can be done through the mouth, that there may be no disfiguring scar left behind.

FIG. 77.



ANGLE APPARATUS, SHOWING ADJUSTMENT. (After Angle.)

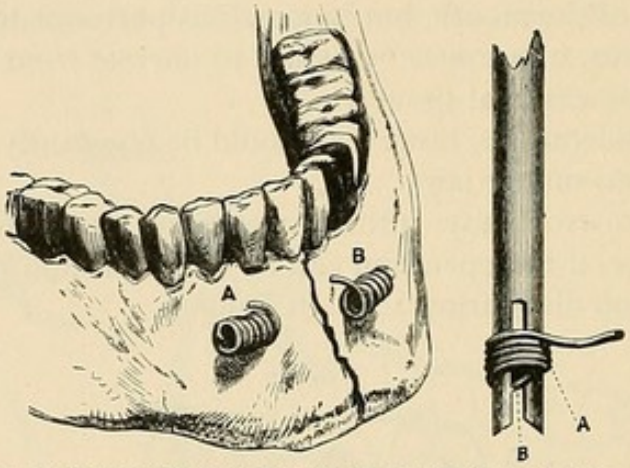
Second. If it becomes necessary—and this often occurs—to operate through incisions in the external tissues, care should be taken that the lines of incision follow the natural lines of the face, and when operating upon the lower maxilla to, as far as possible, keep the line of incision under the lower border of the jaw, for the same reason.

In wiring a fracture of the lower maxilla through the mouth, the lip or cheek should be dissected from the bone at the point of fracture to a depth that will permit of the passage of a drill between the roots of the adjacent teeth without injuring them. The size of the drill should be three-thirty-seconds to one-eighth of an inch in diameter. Holes should be drilled through the bone, one upon each side of the fracture, between the roots of the teeth, at points sufficiently remote to insure solid osseous structure. A single or double silver wire, or silver-plated copper wire, is then passed through the hole upon one side, back through the hole upon the opposite side, the ends brought together, and twisted until the fractured ends of the bone are approxi-

mated. The wire is then cut at a little distance from the jaw, and bent down, or turned in between the teeth.

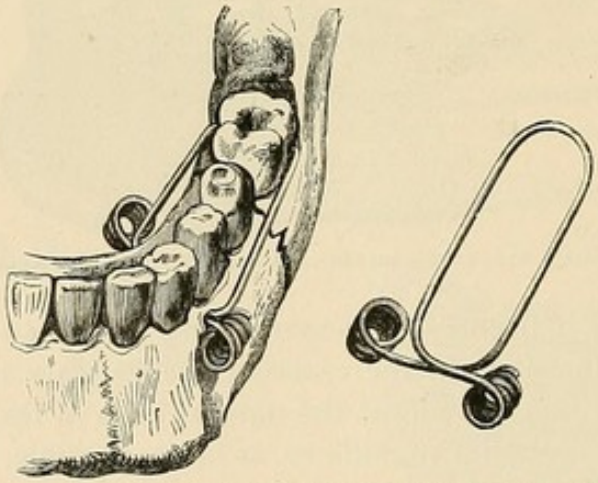
Another method of securing the ends of the wire is by twisting each end separately in the form of a spiral, as practiced by Thomas, of Liverpool, England (Fig. 78).

FIG. 78.



THOMAS'S METHOD OF WIRING MEDIAN FRACTURE OF THE LOWER JAW. (After Erichsen.)

FIG. 79.



THOMAS'S METHOD OF WIRING FRACTURE OF THE LOWER JAW. (After Erichsen.)

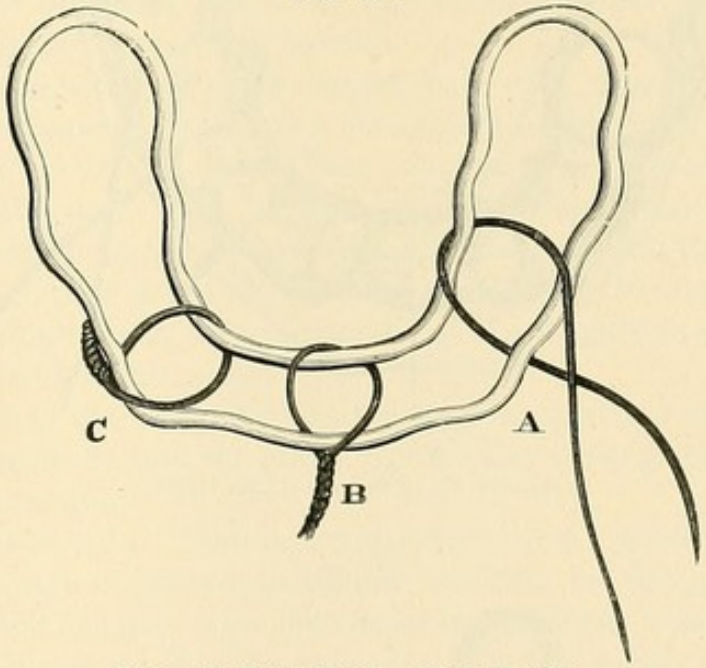
Fig. 79 illustrates a method of wire suturing in cases where the fracture is anterior to the third molar and posterior to the bicuspids, by passing the suture around a molar tooth and then through the jaw at some point at the anterior side of the fracture.

The writer has adopted a method which he likes better than either of these, viz: that of passing the ends of the wire through lead buttons, and, where the dangers of displacement are considerable, using a lead clamp which reaches across the fracture, having two holes in the ends to correspond with the holes which have been drilled in the jaw. The wire is first passed through the holes in one end of the clamp, then

through the jaw on one side, back through the other, through the holes in the opposite end of the clamp, and the free ends of the wire twisted until the fracture is brought into position. He has also found it to be an advantage, in those cases presenting considerable displacement with a tendency of the fractured ends of the bone to slide upon each other and thus prevent a perfect occlusion of the teeth, to insert *two* wire sutures about half an inch apart, one as near the lower border of the jaw as possible without impinging upon the contents of the inferior dental canal, and the other through the alveolar process.

When the operation is made through the external tissues of the face, it is better to twist the wires, cutting them as short as possible,

FIG. 80.



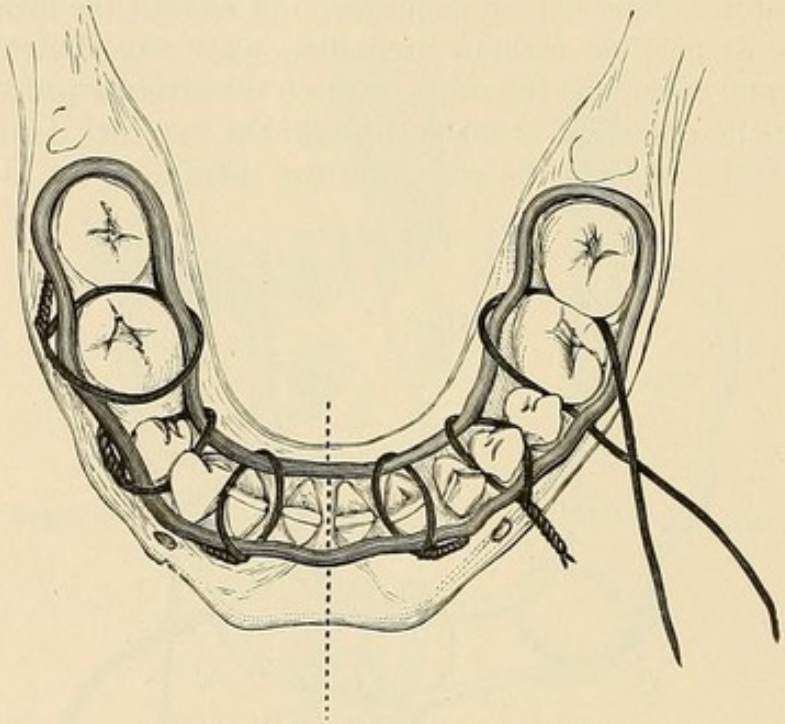
HAMMOND WIRE SPLINT. (After Heath.)

without endangering their strength, turn the points down, and after thorough irrigation with antiseptic solutions, to close the wound except at the point directly over the ends of the wire, treating it antiseptically. The wires may be removed in from four to six weeks.

Hammond's wire splint is a very useful appliance, exceedingly effective and simple to construct (Fig. 80). An impression of the jaw is taken, and a cast made from this. Upon the cast a strong iron or German-silver wire is fitted, following the outlines of the teeth, at the margin of the gums, upon the lingual and buccal surfaces, and the ends soldered or brazed. This is then slipped over the teeth, being held in position by means of fine wire carried between the teeth, and over the splint (see Fig. 81). To prevent the iron wire from rusting, the appliance should be tinned; the German-silver appliance can be plated with gold.

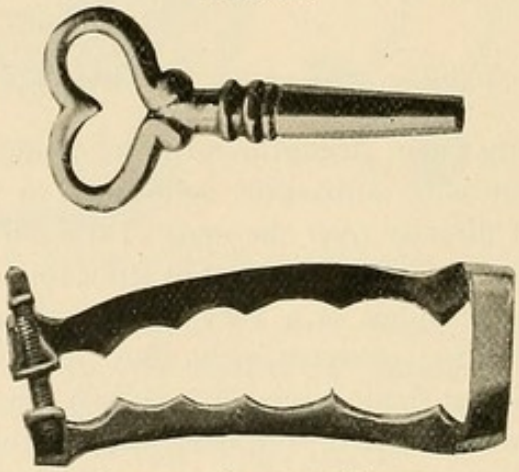
The Shotwell fracture clamp is an ingenious adaptation of the principle of the rubber-dam clamp to the treatment of fractures of the jaws (Fig. 82). It is susceptible of being applied to all forms of fractures situated in front of the angle. This clamp may be made of steel or German silver.

FIG. 81.



HAMMOND WIRE SPLINT. (After Heath.)

FIG. 82.



SHOTWELL FRACTURE CLAMP.

Various forms of interdental splints have been devised, and made of many materials,—gutta-percha, vulcanite, and the various metals; the principal feature of all being that they were molded to fit the teeth, and extended to some distance upon each side of the fracture. They are either intended to act as an internal support while the jaw is

firmly held against the upper teeth, or they are secured to the teeth or bone by screws or metal wire, or by external supports in the form of rods fastened to the side of the splint, and coming out at the angles of the mouth.

Gunning, Hayward, Kingsley, Bean, Moore, Lonsdale, and Hill have each devised interdental splints, having attachments for outside supports.

The Kingsley splint is the one most commonly used, and the description of it will be given in Dr. Kingsley's own words:

"Restore to position displaced fragments, as far as can be done without much effort, the only object being that it makes it a little easier to take an impression. I have always used plaster for such an impression, and see no reason for using any other substance, and, indeed, know of no other substance as good.

"The impression of the deranged fragments may be taken as a whole in an impression-cup, or, if convenient to do so, it can be taken in sections without any cup. Either course, in my practice, has answered equally well. The only object is to obtain casts of all the fragments, either together or separately. Take also an impression in plaster of the upper jaw, and make a cast from it. No dentist should be at all in doubt as to the relation which the fragments of the lower jaw should hold to the upper."

The cast of the lower jaw must be separated with a saw upon the line of the fractures, and the pieces readjusted by using the cast of the upper jaw as a guide.

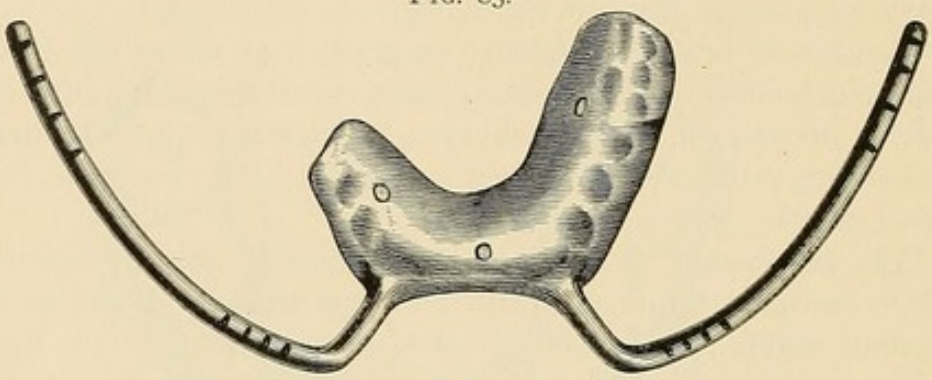
He further says, "There are invariably, even if there are but few teeth in the mouth, certain marks of abrasion on the antagonizing surfaces which identify with exactness the position which the fragment formerly sustained to the upper jaw, and like means of identification I have never failed to find, even in the mouths of children, when they were shedding and erupting teeth; therefore, there is no excuse for failing to reconstruct the model of the lower jaw, and make it identical with the original in its normal condition.

"Upon such a model the construction of a splint of vulcanite involves no manipulations which are not common. Sheet wax, a single line in thickness, carefully pressed over the teeth, and to a little extent encroaching on the gums, gives the form required. If the fracture is in front, the splint need not cover all the back teeth; but if it be at the sides, it is better to cover all the teeth of that side. It is also better to set the casts of the upper and lower jaws in an articulator, and thus make prints of the upper teeth in the wax, to be retained in the splint.

"One of the easiest things of which to make the arms is a couple of discarded excavators, flattening the ends which are to be imbedded, and curving them with much care around the corners of the mouth;

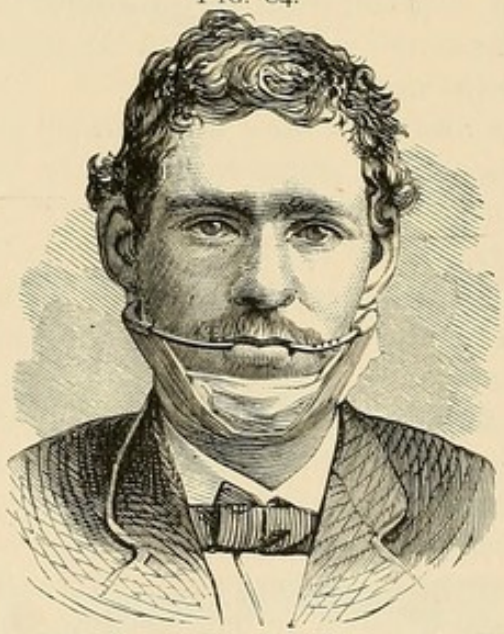
they should terminate at the angle of the jaw. The flattened ends should be made quite broad, and thoroughly imbedded in the splint, as much strain comes upon them. The subsequent steps are familiar to every dentist, viz: investing, packing, vulcanizing, and finishing. In finishing, it is better to enlarge the sockets for the teeth a little, so that there will be no impigning upon the crowns when the splint is introduced, and also to make openings through the top or side, against each

FIG. 83.



KINGSLEY'S INTERDENTAL SPLINT. (After Kingsley.)

FIG. 84.



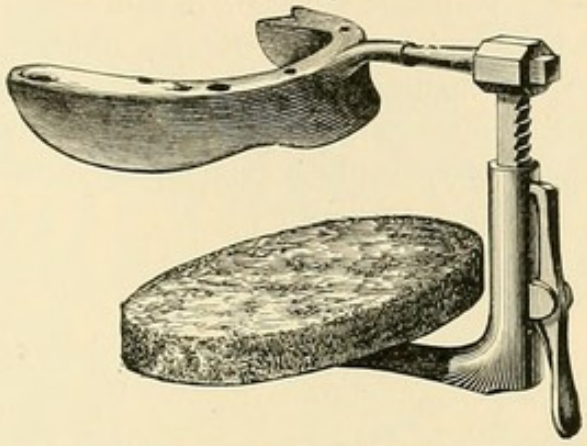
KINGSLEY'S INTERDENTAL SPLINT APPLIED. (After Kingsley.)

tooth adjoining the fracture, so that it can be determined when the fragments are fully in their place (Fig. 83). The latter holes will be convenient to use in cleansing the apparatus by inserting in them the nozzle of a syringe. If the splint is properly made, the teeth of each fragment will follow into the indentations prepared for them, without severe pressure; if they do not, it is quite as well to bind the splint in position, and wait events. It will probably be found, a few hours later, that they have gained their place without further aid."

The splint is retained in position by passing a narrow bandage over the arm of the splint and under the chin, back and forth, until it is firmly fixed (Fig. 84). The after-treatment is that of wounds of the mouth in general.

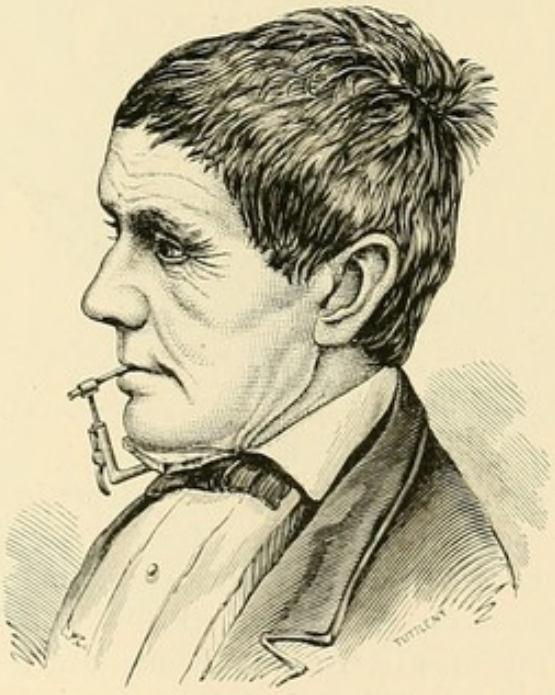
The ordinary duct-compressor may be utilized for the same pur-

FIG. 85.



INTERDENTAL SPLINT ATTACHED TO DUCT DEPRESSOR. (After Kingsley.)

FIG. 86.



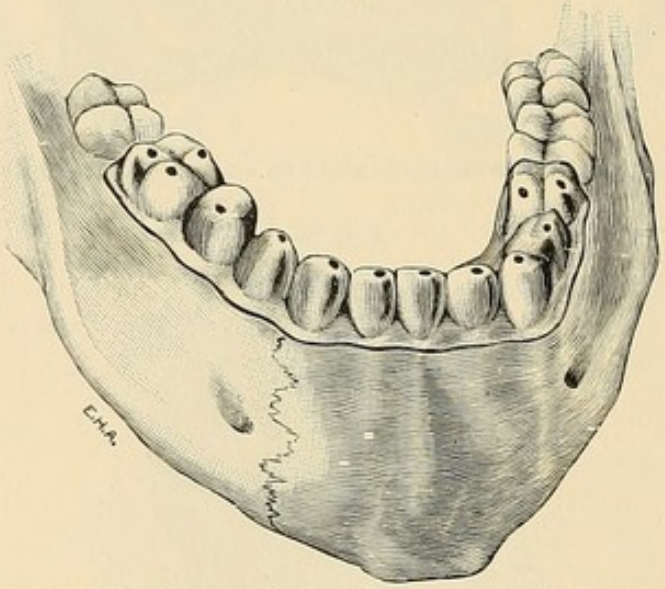
SAME IN POSITION. (After Kingsley.)

pose by attaching the splint to the upper arm of the compressor (Fig. 85), and securing it in place by the ratchet device for causing compression. Fig. 86 shows splint in position.

Another admirable method of securing the ordinary interdental splint in position is by the use of oxyphosphate of zinc cement. The writer has used this method many times during the past twelve years—

in cases where there was moderate displacement—to his entire satisfaction (Fig. 87). The cement should be mixed a little thicker than cream, and the sockets in the splints for the reception of the crowns of the teeth lined with it. The teeth must be thoroughly cleansed beforehand; then dry the surface with bibulous paper, and press the splint into place with the thumb and fingers, holding it in that position until the cement has set. To insure adhesion of this cement, the apparatus must be kept free from moisture while the cement is setting. This may be accomplished with bibulous paper or napkins and the saliva ejector. This method, which was first suggested by Heath, who used gutta-percha instead of the oxyphosphate cement, is certainly preferable to the use of retaining screws or wires.

FIG. 87.



INTERDENTAL METAL SPLINT CEMENTED INTO POSITION.

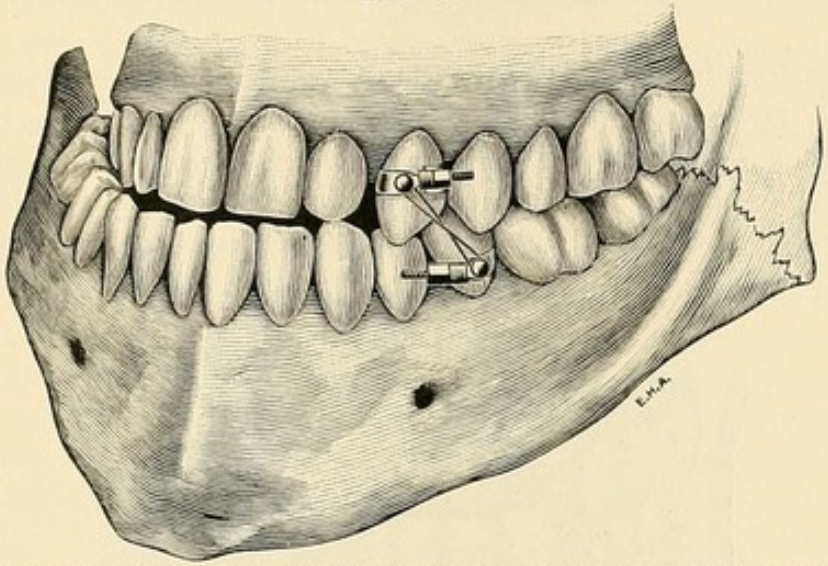
The Kingsley splint may be made of cast metal,—tin or Weston's metal,—or of silver swaged over metal dies, or by the electro-deposit method. The latter method gives the best adaptation, but has the disadvantage of requiring a longer time for its construction. It has, however, in the practice of the writer, given the utmost satisfaction.

In the selection of the particular method that shall be employed in the treatment of fractures of the body of the jaw, that one should be chosen which will be most likely to secure absolute immobility of the fracture, and at the same time give free use of the jaw. The Kingsley interdental splint, the Angle apparatus, and the bone-wiring operation will all give these results in individual cases.

In charity work, the latter operation is generally chosen as least expensive in the consumption of time and money. It has the disadvantage, however, of rendering a simple fracture a compound one; but simple fractures are not often found among hospital cases, as

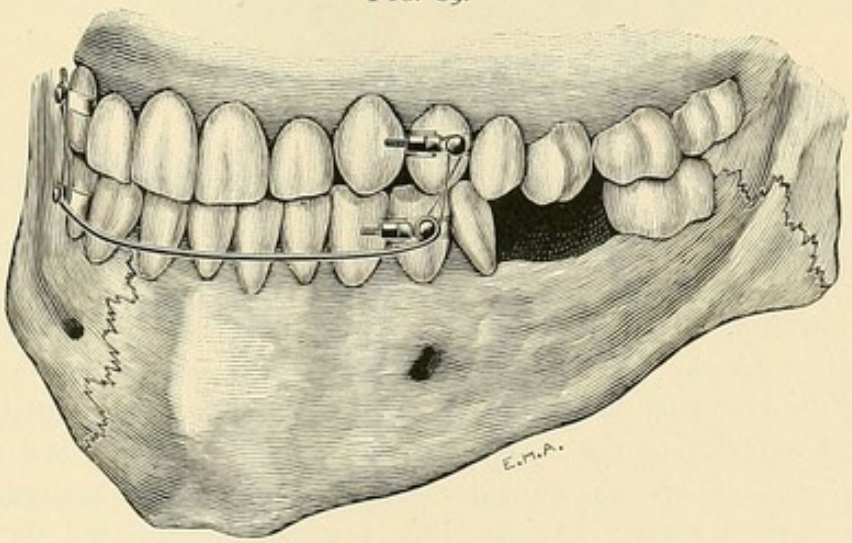
nearly all of them are the result of severe injuries, and consequently are usually compound fractures. It has been the fortune of the writer many times to treat this class of injury by wiring the bone, and experience teaches, taking all things into consideration, that it is the most satisfactory method.

FIG. 88.



ANGLE'S APPLIANCE FOR FRACTURE THROUGH THE ANGLE. (After Angle.)

FIG. 89.



ANGLE'S APPLIANCE FOR FRACTURE THROUGH THE ANGLE AND CUSPID REGION. (After Angle.)

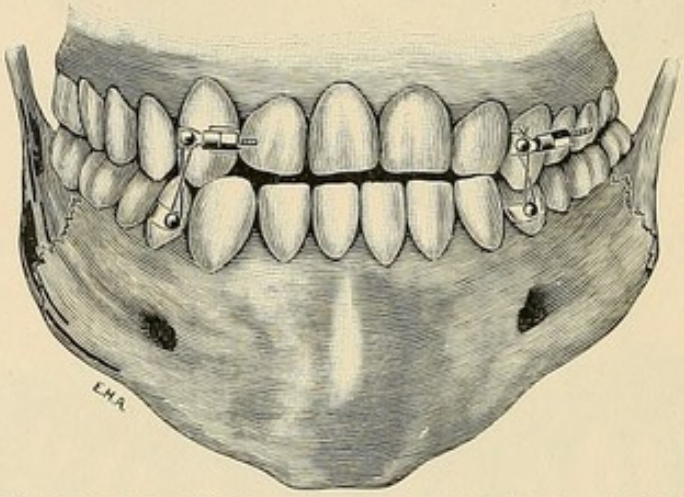
The hygienic conditions of the mouth are important factors in the treatment of fractures of the jaw, and that method will be most successful, other things being equal, which will permit of the most perfect cleansing of the mouth without disturbing the appliance.

Interdental splints are of no value in the treatment of fractures of the angle, ramus, coronoid or condyloid processes, if uncomplicated with fractures of the body of the bone. In such cases the various forms of bandages, wiring the upper and lower teeth together after the

suggestion of Heath, or the Angle appliance, are the best means of treatment. By the Angle method the jaws would be firmly bound together by applying bands to the upper and lower teeth at points opposite to each other, and the use of the wire ligature in the form of a figure 8, as described before in the treatment of simple fractures. Figs. 88, 89, 90, illustrate the Angle method applied to this class of cases.

Occasionally the surgeon will be called upon to treat a fracture of the lower jaw in which all the known methods of retaining the fractured bones in their proper apposition will fail, or in which an appliance must be devised upon short notice which will be applicable to the peculiar conditions existing in an individual case. As an illustration,

FIG. 90.



ANGLE'S APPLIANCE FOR FRACTURE THROUGH BOTH ANGLES. (After Angle.)

a case in point will be briefly described. Mr. G. G., a furnace man, employed at the Iroquois Furnace Company, South Chicago, was blown, by the bursting of the furnace door, twenty-six yards, striking broadside against a wall. His face and head especially seemed to receive the force of the concussion. The lower jaw was fractured upon the right side, just anterior to the angle, and between the cuspid and first bicuspid teeth. There was considerable displacement on account of the location of the fractures and the contraction of the muscles, the middle fragment overriding the others upon the outside. For several days reduction was not attempted, as the man was suffering from a severe concussion of the brain, and gave little hope of recovery. Four days after his admission, the brain symptoms having somewhat improved, a careful examination, under ether, was instituted. Efforts were made to replace and secure the middle fragment in its normal position by the various methods of external splints, bandages, and wiring of the teeth, but without avail. An interdental splint could have been made for the case if the patient had been sufficiently recovered from the injury to the

head to exercise self-control. This being out of the question, it therefore became necessary to devise some other means of maintaining the fractured bone in normal position. This was accomplished by making two incisions in the soft tissues down to the bone, one just behind the angle of the jaw, about half an inch above the lower border, and the other beneath the roots of the cuspid and lateral incisor teeth, at about the same distance above the lower border of the jaw. Holes were then drilled in the bone at the points of incision, and long nickel-plated picture screws set into the holes. Extension was then made upon the anterior fragment, aided by ligatures passed around the incisor teeth, until the middle fragment could be forced into place. Extension was maintained by placing a wooden brace, made from the side of a cigar-box, between the screws. Ligatures were then passed from screw to screw, and under the ends of the brace, in the form of the figure 8, thus making a rigid appliance, which held the fractures immovably in their normal position. Dressings were applied to the external wounds, and the mouth kept as clean as the circumstances would permit. Four weeks later the screws were removed, and union was found to have taken place, while the perfect occlusion of the teeth proved that the fractured bones had been placed in normal apposition.

Fractures of the alveolar process following the extraction of teeth need no other treatment, as a rule, than forcing the separated parts into position with the thumb and fingers.

The after-treatment is comprehended in the term "surgical cleanliness."

Abscess of the Jaws.—Abscess of the jaws frequently follows compound fractures. This is the result of local infection, generally from a filthy condition of the oral cavity or from necrosed bone.

It is often stated by the opponents of the germ theory of disease that pathogenic micro-organisms are constantly found in the blood and tissues of healthy individuals. This statement has not been substantiated, either by the most careful microscopic research or by clinical observation. There is no doubt whatever that such organisms do sometimes gain access to the tissues of the healthy living body, but they are at once, and almost invariably, destroyed by the action of the living cells and fluids of the body, or are eliminated by the excretory organs.

A simple fracture will heal without suppuration in a healthy individual, even though it had been demonstrated that micro-organisms were already in the blood; but if a certain quantity of the pus-producing microbes were introduced, sufficient to overcome the resistance of the tissues and fluids of the body, suppuration was the result. Vital resistance, therefore, plays an important part in preventing suppuration.

As soon as fluctuation can be detected, the abscess should be opened, preferably through the mouth, so as to avoid a scar; but sometimes it is necessary to open it through the external tissues, especially where there is a tendency to phlegmonous inflammation in the submaxillary region and the neck. After the pus has been evacuated, search should be made for necrosed bone, and if found, it should be immediately removed, provided separation has taken place, and the wound irrigated and dressed antiseptically. If separation of the dead bone from the living has not taken place, it is better to wait for nature to complete the process, as surgical interference under such circumstances is not indicated.

Preceding the discovery of an abscess, there will always be an elevation of the body temperature from 2° to 6° or 7° Fahrenheit, which is always indicative of the formation of pus, and the presence in the circulation of septic ptomaines. Such a condition should at once lead to a critical examination of the wound and the surrounding tissues. If septicemia should develop, treat the case as indicated in a previous chapter upon Septicemia.

REVIEW.

CHAPTER XXI.

What two conditions are necessary to successful treatment of fractures?

What appliances are used for the treatment of fractures of the lower jaw?

What should determine the selection of the appliance?

What are the common complications of fractures of the lower jaw, and how should they be treated?

Describe the method of treating fractures by wiring the teeth.

Describe the Angle appliances.

What class of fractures is generally treated by these methods?

What method is thought to be best in treating fractures with considerable displacement?

Under what circumstances is it possible to operate by this method within the mouth?

Under what circumstances is it necessary to operate by incisions through the external tissues of the face?

What two considerations are to be recognized in all operations upon the jaws?

Describe the operation for wiring the bone.

What are the methods for securing the ends of the wire?

In what class of cases are two wire sutures of advantage?

At what locations should they be placed?

How soon may the wires be removed?

Describe the Hammond wire splint.

How is it constructed?

How is it secured in position?

What is the Shotwell fracture clamp?

To what class of cases is it adapted?

From what materials are interdental splints usually made?

What is their principal feature?

By what means are they usually secured in position?

What form of interdental splint is most commonly used?

How may the impressions be taken?

How is the cast to be readjusted to represent the normal jaw?

What is the value of a cast of the upper jaw?

What is the difference between the Kingsley or Hayward splint and other forms?

How are the wire arms secured?

How is the splint retained in the mouth?

What other method is sometimes used to secure the ordinary interdental splint?

What should be the after-treatment in fractures of the jaws?

What is the cause of abscess following fractures of the jaws?

Under what circumstanecs might the presence of pus-microbes in the blood cause suppuration?

What form of fracture heals without suppuration?

How should abscesses following fractures be treated?

When should necrosed portions of bone be removed?

What marked constitutional symptom is present in septic infection?

How high does the temperature rise?

What does this condition demand?

CHAPTER XXII.

FRACTURES OF THE SUPERIOR MAXILLÆ AND UPPER BONES OF THE FACE.

FRACTURES of the superior maxillary bones are, from their protected location, quite rarely met with except in the alveolar process. The causes of such injuries in this location are usually the extraction of teeth or blows or falls upon the chin, which separate and split open the walls of the alveoli. This is accomplished in the one case by the lateral force applied breaking up the attachments of the roots of the teeth, and in the other by driving the teeth upward and through their alveoli.

Such injuries, however, are never very serious, and rarely require special apparatus to maintain the fractured bones in their normal position.

Injuries of the upper bones of the face, which cause comminuted fractures and separation from the bones of the cranium, are always the result of great violence,—like the passage of the wheel of a carriage over the face, falling from a great height, the kick of a horse, a blow in the face by some heavy missile thrown with great force, a gunshot wound, the overturning of a carriage upon the occupant, crushing of the head between a moving elevator and the floor, or other heavy, crushing force.

Since the general introduction of passenger and freight elevators into hotels, office buildings, and large manufacturing establishments, "elevator" accidents are much more likely to occur, and will doubtless be found more and more common. The experience of the writer, at least, leads to this conclusion, for during the last ten years about twenty-five per cent. of all the cases of fracture of the upper bones of the face which have come under his care have been caused by this class of accidents.

The class of injuries which forms the subject of the present chapter is one which has received but little attention from either the general surgeon or the oral specialist. Several of the leading works on surgery make no mention whatever of them, which is due no doubt to the fact that the accidents which cause them have been in the past of rare occurrence.

During the last few years five cases have come under the personal care of the writer at St. Luke's Hospital. The first led to a somewhat careful examination of the text-books and periodical literature bearing upon the subject. So far he has been able to gather together but nineteen cases which can be fairly classed as similar to those which form the subject of this chapter.

These injuries are always serious, and often prove fatal, either from shock, hemorrhage, direct injury to the brain, or from secondary complication.

In those cases which survive the shock of injury and escape immediately serious complications of the brain, a favorable termination may be looked for, and in many cases, if properly treated, with very little deformity. This, however, will depend very much upon the character and location of the particular injury, and the success obtained in readjusting the fractured and dislocated bones, and maintaining them in their proper positions.

For the purpose of reference, the various published cases are here grouped together, only brief mention being made of the extent of the injury and the percentage of mortality.

In speaking of this class of injuries, Erichsen says, "In some cases all the bones of the face appear to have been smashed and separated from the skull by the infliction of great violence." He mentions four cases of this form of injury,—one reported by South, one by Vidal, and two which came under his own notice.

The injury in South's case was caused by a man being "struck in the face by the handle of a rapidly revolving crank." All the bones of the face were "separated and loosened," and so comminuted as to feel "like beans in a bag."

Vidal's case, also a man, was injured by a "fall from a building and striking upon the face, which fractured and separated all the facial bones."

Erichsen's cases were both the result of falls from a considerable elevation, and striking upon the face. The two former recovered; the two latter died in a few hours.

Packard mentions three cases, one by Cotting, in which the face was crushed by a cart-wheel passing over it; another, brought to the Pennsylvania Hospital, in which the injury was received by the head being caught between the platform of a steam hoisting machine and the floor; the third, a case reported by Heath in his "Injuries and Diseases of the Jaws," which was under the care of Dr. Fyffe. The first and last cases recovered; in the other, death resulted in a few hours.

Heath describes two cases. The first came under his personal notice, and was "caused by the passage of a wagon-wheel over the face. The bones were completely crushed and separated one from

another, and death was instantaneous." The second one is that reported by Dr. Fyffe, the same before referred to as mentioned by Packard, and which will be described later.

Tiffany mentions a single case, which was reported by Professor Christopher Johnston. The patient, a man, was struck in the face by the walking beam of a steamboat. All the bones of the face were crushed, and "seemed literally to consist of a bag of bones, moving freely with inspiration and expiration, so extensive was the comminution." This case made a good recovery, and an excellent result was obtained by supporting the superior maxillæ by means of a silver wire passed through the cheeks and under the teeth, and uniting the ends of the wire over the head by a rubber band.

Richard Wiseman published the report of the first case on record, and described the method of treatment. The patient was a little boy, eight years of age, who was kicked by a horse, the whole upper jaw being driven in so that the finger could not be passed behind the palate. A flattened hook was constructed which could be inserted behind the palate, and by extension, constantly maintained by the patient and assistants, the bones were held in place and a good recovery followed.

In the case reported by Dr. Fyffe, of Westminster Hospital, London, the patient was thrown from a cab, the vehicle turning over upon him. The superior and inferior maxillæ were fractured, and the bones of the face detached from the skull, so that the former "moved up and down in the act of swallowing." This patient also recovered.

Holmes describes a single case, in which the bones of the face were crushed and dislocated by a carriage-wheel passing over the face, and in which, after recovery, there "was a disagreeable lengthening of the face," as the result of the injury. It would seem more likely, however, that this condition was the result of the treatment. Among the methods of treatment suggested by Holmes are gutta-percha molds, cork disks placed between the teeth, wiring of fragments, and carefully adjusted pressure by the Hanesby truss.

Hamilton refers to one case which came under his own care, in which the upper bones of the face were fractured and torn from their attachments to the cranium, and had to be supported to keep them in place. The patient died on the twelfth day after the injury.

Mason reports a case which was under the care of Mr. Bickersteth, of Liverpool. A man, standing upon the deck of a steamer, was struck upon the side of the face by an iron hook attached to the hawser, which had parted under a heavy strain. On examination, "immediately after the accident, the mouth seemed to be filled by a piece of bloody meat; but upon a more thorough examination this proved to be the muscles attached to the upper jaw; the orbital plate

of the superior maxilla of the injured side was found beneath the cheek, while the palate process, with the alveolar ridge and teeth, were, for the time, situated in the upper part of the pharynx, looking toward the bodies of the upper cervical vertebræ. The facial surface of the bone took the place of the roof of the mouth, jamming the jaws open. The soft palate was not torn, but considerably injured. The superior maxilla of the injured side was turned completely upon its axis. The detached mass was replaced, the lower jaw firmly closed upon it for support, and the whole rapidly united with scarcely any deformity."

Salter reported a case in which the superior maxillæ and malar bones were separated from their attachments with the skull, and so crushed as to feel like a mass of "loose bones."

Harris, of New York, also reported a case of a little child, only two years of age, who fell a distance of fifty feet to the pavement, striking upon the face and sustaining fractures and separation upon the median lines of both superior maxillæ and palate bones. "Union had not taken place six weeks after the injury."

Houghton describes a case in which the "superior maxillæ were so fractured and displaced as to make it impossible for the patient to protrude the tongue until after the bones had been adjusted to their normal position."

Bryant mentions one case in which "the superior maxillary bones were completely detached from the skull, and could be moved about in any direction, yet a good recovery ensued."

Agnew cites the cases of Wiseman, Fyffe, and Packard, but describes no new cases.

Garretson reports two cases. The history of the first was furnished him by Professor Agnew, in which a lad was crushed between the bumpers of two railroad cars, and sustained diastasis of all the bones of the face from the skull, comminuted fracture of the superior maxilla, and four fractures of the inferior maxilla. The patient recovered, but with considerable deformity. The second case, a painter, came under his own care. The injury was caused by falling from the roof of a house, and striking upon the pavement below. He sustained severe comminuted fractures of the superior and inferior maxillæ, and fracture of both arms and legs. Several splinters of bone were removed from the anterior portion of the lower jaw. This allowed the parts to fall together, "the symphysis of junction being midway between its former position and the hyoid bone." The patient made a good recovery, but with considerable deformity.

With this brief summary of the history and results of the various cases found on record, the writer will present those cases which have come under his own observation and treatment.

Case No. 1. Mr. Conrad A., Swede, age 28 years; occupation, sawyer; employed at the Pullman Palace Car Works, Pullman, Illinois; was brought into St. Luke's Hospital on March 15, 1887, in an unconscious condition, with an incised wound of the right cheek, starting from a point just below the inner canthus of the eye, and extending obliquely backward and downward a distance of four inches, exposing the superior maxillary bone and completely penetrating the cheek, leaving a considerable external opening into the mouth. This wound had been sewed up by the surgeon at the Works. Further examination revealed that the right malar was crushed, both nasal bones were fractured and separated at the naso-frontal suture. The left zygomatic process was fractured near its union with the malar bone, both superior maxillæ were torn loose from the bones of the cranium, so that the whole mass was loose and freely movable in any direction, and suspended by soft tissues. When the mouth was open to its fullest extent, the teeth of the upper jaw rested upon those of the lower. Both superior maxillary bones were also fractured on a nearly perpendicular line, on the right side between the first and second molar teeth, on the left between the second bicuspid and first molar teeth. Openings existed in both antra. The right could be entered with the probe through the wound in the cheek, and also through the alveolar process on the buccal aspect between the roots of the first and second molars, while the left could be penetrated through the buccal surface of the alveolar process on the line of the fracture between the second bicuspid and first molar teeth.

The palate process and palate bones were also badly crushed, forming a compound comminuted fracture, with loss of bone-tissue, leaving an opening in the hard palate on the right side near its posterior edge and the median line, through which the index finger could be freely passed. The bones of the internal nose were badly comminuted, and several pieces which were loose were removed.

The left side of the face was completely anesthetic over the whole region supplied by the infraorbital nerve, while upon the right side the upper lip and wing of the nose only had lost sensation. The inferior maxilla was not injured, and none of the teeth had been lost by the injury in either jaw.

The accident occurred by the patient being struck in the face by a piece of oak timber, twelve inches in length and eight by nine inches in diameter, which was thrown by a circular saw eighteen inches in diameter, and running at the rate of about three thousand revolutions per minute.

When the patient was admitted to the hospital, the chances for recovery seemed very small. Cold applications were ordered over the face, and stimulants hypodermically, if the temperature should fall

below normal and the pulse below sixty; nourishment to be given if possible, and one-fourth of a grain of morphia to allay pain, if found necessary upon regaining consciousness.

March 16. Swelling of parts very great; both eyes closed and nasal passages completely plugged. Pulse 84. Temperature 101.8°. Would arouse when spoken to.

March 17. Patient has rallied. Pulse 74. Temperature 100°. Seems to be conscious. Swelling less.

On the 18th his temperature was normal, and he rapidly improved in general condition from this date.

The treatment of the fractured jaws was begun on the 17th, and consisted of first wiring the posterior fragments of both superior maxillæ to the anterior or middle portion, by means of silver wire passed around the teeth upon either side of the fracture. The fractured palate bones and the palate processes were then molded into place as nearly as possible by the fingers, and the nasal bones lifted into position by means of the handle of a small instrument. The lower jaw was next closed upon the superior teeth, care being taken to get a correct occlusion, and then held in position by means of an occipito-frontal and occipito-mental bandage.

The following night the patient tore off the bandage several times. The nasal passages being closed from the results of the injury, he had great difficulty in breathing when the jaws were held tightly together. The bandages were therefore reapplied more loosely, but this allowed the injured bones to fall out of place and defeat the object in view. The foregoing is the plan of treatment usually recommended by such authors as mention this class of injuries; but in the hands of the writer it has proved a signal failure, from the fact that several times the nose has been so injured and the parts so badly swollen as to close the nasal passages for several days, therefore making it impossible to breathe with any degree of comfort except through the open mouth.

There must always be considerable difficulty in any severe case in maintaining the position of the fractured and dislocated bones when this plan is adopted, and it is nearly impossible in those cases in which all the teeth were lost prior to the accident, for it is a well-known fact that, as a rule, edentulous jaws do not come in contact, and if they should, a normal occlusion could not be obtained, nor the injured parts be prevented from slipping out of position. The plan of treatment adopted by Professor Johnston is also objectionable by reason of the wounds made in the cheeks.

In this case the writer was compelled to devise other means, which would maintain the position of the fractured bones, and at the same time leave the lower jaw free, so that the mouth could be open for the purpose of breathing. This was accomplished by adapting the

principle of the Hayward or Kingsley interdental splint to the upper jaw, and supporting it from the cranium. Impressions of the upper and lower teeth were taken with the modeling compound, by first molding it upon the upper teeth, and while it was yet soft forcing the lower jaw upward till a correct occlusion of the teeth was obtained. This impression was trimmed to the desired shape, a one-eighth inch steel wire was imbedded in the sides upon a line with the ends of the teeth, then bent backward upon itself opposite the cuspid teeth, and allowed to extend outside the cheek nearly to the lower border of the ear. From this was constructed a hard rubber splint, with the wires attached. The splint can be made from silver swaged over metal dies; but if a metal plate is desired the most perfect adaptation can be secured by the electro-deposit plate, the wires being attached with solder. The splint is held in position by means of double elastic straps, attached to the wire upon each side, and buckled to a close-fitting leather or net cap, which is reinforced with leather and laced firmly upon the head. This proved to be a very successful appliance, as it held the fractured bones in their proper position, permitted comfortable breathing and free movement of the lower jaw, which enabled him to talk, and, after a few days, to masticate soft food. Deep indentations were made in the under side of the splint, in which the lower teeth fitted accurately when the mouth was closed. (Fig. 91.)

The object of this was to furnish a sure guide to the normal position of the superior maxillæ. Without this, the correctness of the adjustment of the bones could not have been verified. Its importance, therefore, cannot be over-estimated. The only other treatment was good feeding, thorough irrigation of the wound, antrum and mouth, with a 2 per cent. solution of carbolic acid every two or three hours until the discharges ceased, and the removal of a few spiculæ of bone from the nose and the wall of the right antrum.

The patient was discharged on May 14, with small fistulous openings through the gums, leading into both antra at the points of fracture, and a small opening in the hard palate, which was gradually growing less. There was also a slight deflection to the left of the nasal septum, and a deep cicatrix in the right cheek (Fig. 92). The opening into the right antrum soon closed, as did that of the hard palate.

June 22. The patient returned for the removal of the cicatrix in the right cheek, which was adherent to the maxillary bone.

June 25. Stitches removed and adhesive strips applied, and the patient discharged three days later.

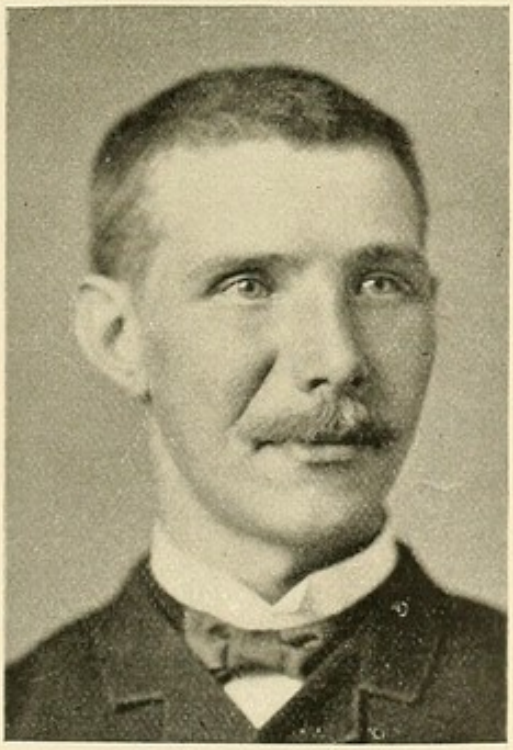
The opening into the left antrum remained patulous for some months, with slight discharge into the mouth, but finally closed. Sensation has been entirely restored in both sides of the face. The occlusion of the teeth is nearly, if not quite, normal, and the only deformity

FIG. 91.



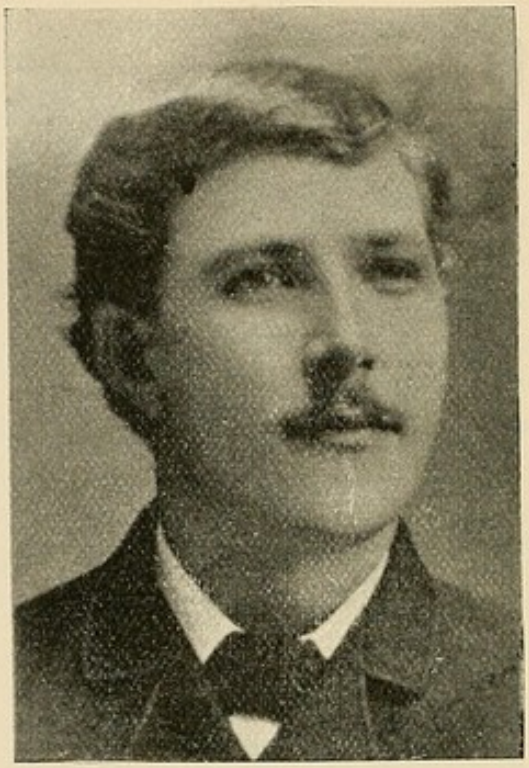
Case 1. SPLINT IN POSITION AND SUPPORTED BY A LEATHER CAP.

FIG. 92.



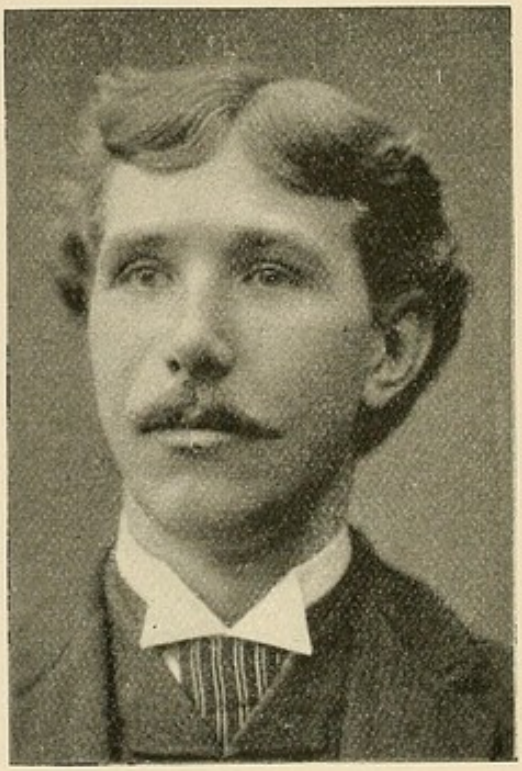
Case 1. AFTER THE SPLINT WAS REMOVED.

FIG. 93.



Case I. BEFORE THE ACCIDENT.

FIG. 94.



Case I. FINAL RESULT OF TREATMENT AFTER OPERATION UPON THE CHEEK.

visible is a slight flattening of the right superior maxilla, and a faint line of the cicatrix resulting from the incised wound in the cheek. Fig. 93 shows the patient as he appeared one year before the accident; Fig. 94 shows the final result of the treatment.

Case No. II. Henry S., German, age 35; occupation, laborer; employed at Armour's packing house. Was admitted to the hospital October 7, 1887, one hour after the accident, which was caused by being struck across the bridge of the nose by a descending elevator, while he was in the act of looking up the shaft. When admitted he was suffering from concussion of the brain. Examination disclosed a lacerated wound over the left eye, extending across the nose to the right eye. The finger could be introduced and readily passed down into each orbit, and against the fractured edges of the nasal and sphenoid bones. By taking hold of the upper teeth, all the bones of the upper face were found to be movable; when the mouth was open the upper teeth rested upon the lower, and there was a peculiar and disagreeable elongation of the face. A later examination revealed the fact that the frontal sinus was crushed in, the nasal and lachrymal bones comminuted, all the bones of the face torn loose from the skull on a line passing through the orbits; and that the superior maxillæ were separated from the other bones of the face. The inferior maxilla was not injured. Several loose pieces of bone were removed from the region of the inner canthus of each eye by the house surgeon. The wound was treated antiseptically, stitched, drainage-tubes inserted, the lower jaw bandaged tightly against the upper teeth, and iced cloths ordered over the face and head. Pulse 60; temperature 97.4°. Stimulants administered freely. Profuse hemorrhage occurred during the night, and vomiting of blood every few hours until two o'clock on the following day. Patient became conscious during the night, but the other symptoms seemed to give little hope of a final recovery.

The temperature reached 102.2° on the 8th, at 4 P.M., and fell to normal on the 9th. No brain symptoms developed, and the patient rapidly improved in general condition from this time. Swelling and suppuration were so excessive for several days as to render the adjustment of a splint impossible. The treatment consisted of stimulants and thorough irrigation of the wounds. The old method of bandaging was resorted to, but proved a failure, for the reason that the lower jaw could not be held sufficiently closed to support the fractured bones in their proper position without obstructing breathing by the mouth. This was necessary, as nasal breathing was at the time impossible.

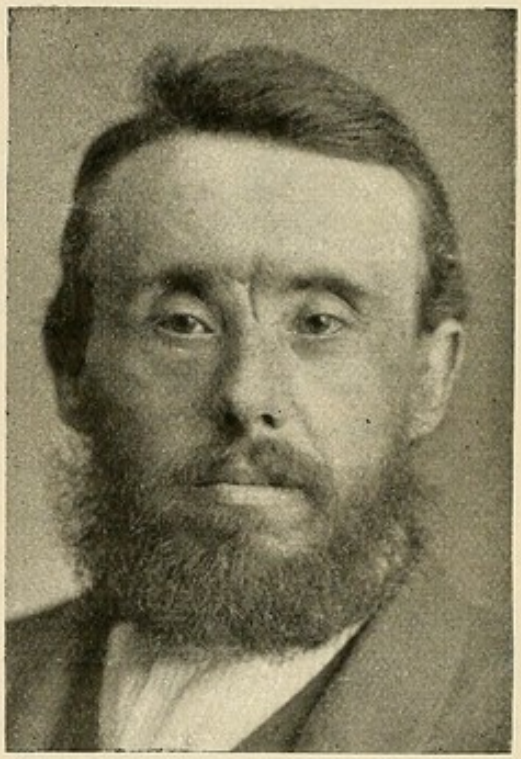
Oct. 24. Inserted an interdental splint, constructed after the plan of the one used in the preceding case and supported from the head by the same means. The case progressed favorably and was discharged December 3, the bones having all united.

Dec. 24. Patient returned complaining of double vision. The lens of the left eye looked cloudy, and traumatic cataract was feared.

April 1, 1888. Patient seems entirely well. The double vision complained of has passed away and the lens has cleared up.

The occlusion of the jaws is equally good with that in Case No. I, but the deformity of the face is greater. This was the result of the loss of portions of the nasal bones and of the external wall of the frontal sinus, and from adhesion and contraction of the cicatricial tissue over these locations (Fig. 95).

FIG. 95.



Case 2. FINAL RESULT OF THE TREATMENT.

Case No. III. Joseph J., Pole, age 17 years; employed in the printing office of the Chicago *Evening Journal*. Was admitted to St. Luke's Hospital at 4 P.M., May 9, 1889, in an unconscious condition, and within an hour after the occurrence of the accident,—a blow across the left orbit from a descending passenger elevator while he was in the act of looking up the shaft.

The external tissues about the left eye and right side of the chin were badly lacerated and bruised; the superior maxillæ were fractured upon a line with the orbits and at the articulation of the sphenoid bone, and so displaced that the posterior portions of the superior maxillæ hung down and prevented the closing of the mouth, causing a separation of the anterior teeth by the space of one-half an inch. Upward pressure applied to the region of the superior molar teeth carried the bones into position, but upon removal of the force they immediately

fell back again. The bones of the face were not comminuted as in the before-mentioned cases, but they were freely movable upon slight pressure.

Swelling was very great from effusion, and there was also a considerable protrusion of the left eye, with effusion of blood into the anterior chamber. The face and eye were immediately dressed with cold applications, and ice applied to the head. Hemorrhage was slight. Later in the evening consciousness returned, and at no time afterward did any brain complication arise. Pulse 60; temperature normal. Morphia sulf., gr. ¼, was administered to allay the pain.

An attempt was made also in this case to reduce the diastasis of the bones, and maintain them in position by bandaging the jaws together, but this could not be borne on account of the injury to the nasal passages, causing inability to breathe except through the mouth, while the swelling was so great as to make it impossible at that time to take impressions of the jaws.

May 10. The temperature rose to 100° F. On the 12th it fell to normal, and did not again go above that point.

The general treatment consisted of liquid diet, thorough irrigation of the external wound and of the mouth every hour with Thiersch's antiseptic solution. The treatment of the eye was placed in the hands of the attending ophthalmologist.

May 14. The patient was up and doing well. Impressions were taken of both jaws, and an interdental splint constructed upon the same principles as in the other cases.

May 16. Splint inserted and supported from the head, bringing the bones into their normal position. The appliance was worn with comfort, and at the end of seven weeks the patient was discharged cured.

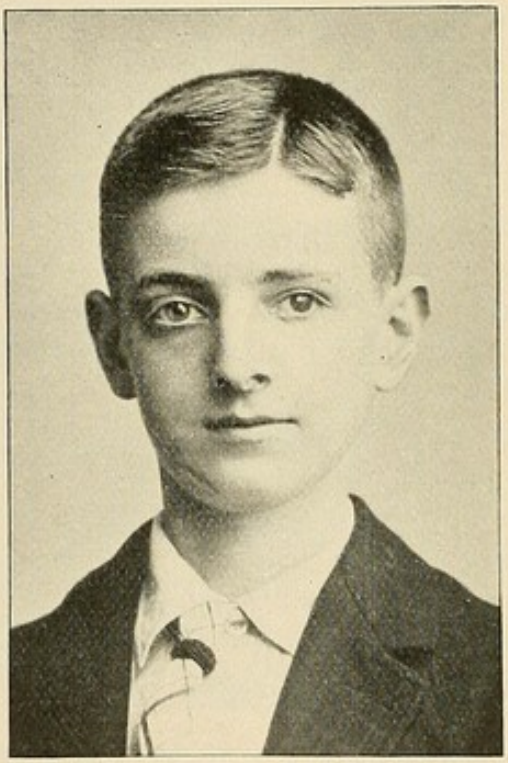
The occlusion of the teeth is normal. There is no deformity of the face, and but for the loss of sight in the left eye, and a small scar upon the right of the chin, no evidence would be left to tell of so serious an injury (Fig. 96).

Case No. IV. Mr. A. C., German, aged 40, machinist. Employed at Armour's packing house. Was admitted to St. Luke's Hospital September 23, 1891, very soon after the accident occurred, in a semi-unconscious condition, with laceration and contusion of the left side of the face, comminuted fracture of the left malar and zygomatic process and nasal bones, fracture of the superior maxillæ through the orbits, with separation of the bones of the face from the skull. The upper bones of the face were slightly movable in all directions. On opening the mouth the bones sagged down, and upon closing the teeth together the bones were again driven upward. The displacement, however, was not so marked as in Case No. I or No. II. The injuries

were caused by the patient being struck upon the left side of the face by the corner of a descending elevator while he was working in the bottom of the shaft. The same treatment was adopted in this case as in the preceding one, and with entire satisfaction. The case was discharged at the end of ten weeks, with perfect occlusion of the teeth and no deformity, saving a little flattening of the face in the region of the left malar.

Case No. V. Mr. P. C., Irish, aged 42, roofer. Admitted to St. Luke's Hospital a few minutes after the accident, on October 7, 1893;

FIG. 96.



Case 3. FINAL RESULT OF THE TREATMENT.

the patient having fallen from the roof of the new Twelfth street Illinois Central Depot, Chicago, a distance of at least sixty feet, striking upon the scaffolding in the descent, and sustaining a fracture of the left humerus, contusion of the left side of the face, a simple fracture of the lower jaw between the second bicuspid and first molar, and fracture of the superior maxillæ, apparently through the lines of the sutures. Crepitation and mobility were distinctly discernible, but displacement was very slight.

The fracture of the lower jaw was treated by wiring the teeth together upon either side of the fracture, and the application of the four-tailed bandage. The support of the lower jaw was sufficient to maintain the proper position of the fractures of the superior maxillæ; consequently a splint for this purpose was not necessary.

The case did well from the commencement. The patient was discharged at the end of seven weeks, cured, and with no deformity of any kind.

In constructing the interdental splint for this class of cases, metal has the advantage over vulcanite, in that it is not so clumsy, does not take up so much room in the mouth, and is less liable to break.

When metal is used the upper and lower sections are to be swaged separately, and afterward soldered together, and the arms attached in the same manner. A better adaptation may be secured by making the sections by the electro-deposit method, then soldering them together and attaching the arms in the same way. If a more elegant-looking appliance is desired, it may then be electro-plated with gold.

REVIEW.

CHAPTER XXII.

What are the causes of fracture and diastasis of the superior maxillæ and upper bones of the face?

What are the complications that may cause a fatal termination?

What is the prognosis?

What should be done with loose pieces of bone?

Should other than completely detached pieces be removed?

Why not?

What general treatment is important in dressing the external wounds?

What means can be used to control the swelling of the soft tissues?

What should be the treatment in severe hemorrhage?

If the injured blood-vessels cannot be reached, what other local treatment would be admissible?

What drug administered internally would assist in controlling the hemorrhage?

What drug may be administered to relieve severe pain, and in what dose?

Would operation be advisable while the patient was suffering from shock?

What are the methods of treatment which have been suggested to retain the fractured and displaced bones in their normal position?

Is bandaging the jaws together practicable in those cases where the injury is very great?

How soon after the injury can impressions of the jaws be taken?

What is the best material to use for impressions in these cases?

Why is it necessary to take impressions of both jaws?

What are the best materials of which to make the splint?

Describe the apparatus used by the writer.

What is the after-treatment?

What advantages have interdental splints made from metal over those made from vulcanite?

What metal is used?

How are the metal splints constructed?

What form of metal plate gives the best adaptation?

CHAPTER XXIII.

DELAYED UNION AND UNUNITED FRACTURES.

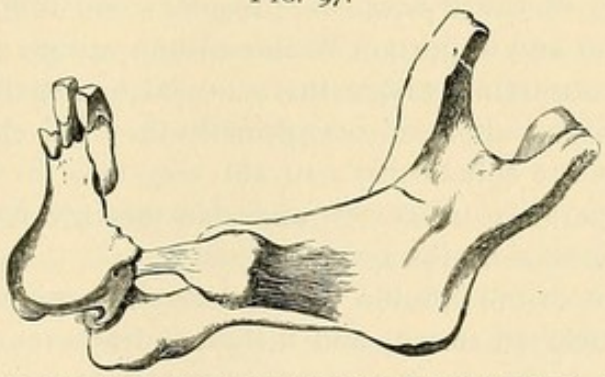
Union of fractured bones is sometimes delayed beyond the time usually occupied by the system in this process of repair, and occasionally there is complete failure in the process, resulting in what is known as "*ununited fracture or false joint.*" Delayed union is by no means an uncommon condition, but complete failure of union is quite rare. These conditions occur most often in the long bones of the extremities and in the lower jaw, the causes which operate to bring about such conditions being equally applicable to fractures in both locations. Delayed union and ununited fractures are the result, in a large majority of cases, of arrestation of the process of callus formation before it has reached the stage of calcification or ossification. In others it is the result of the loss of osseous tissue, or such a displacement of the fragments as to leave a gap too large to be bridged by a continuous callus. The conditions, however, which combine to cause delayed union and ununited fractures do not seem to be very well understood. In some cases it appears to be an inability on the part of the bone-producing structures to form new bone. (Warren.) The material thrown out by the inflammatory process is absorbed, but no new bone-forming material takes its place, and, as a consequence, the fractured ends of the bone fail to unite, and there is, as a result, an ununited fracture. Examination of the bone at this time reveals the fact that absorption has been going on at the fractured ends, as evidenced by the loss of the sharp edges, and their more or less pointed form. The ends of the bone in these cases are usually bound together with a ligamentous band (Fig. 97). In some cases nature attempts to develop a false joint by forming a capsule which invests the fractured ends of the bone, and contains a clear serum. The ends of the bone may also be covered with cartilage, more or less perfectly representing the hyaline cartilage. Occasionally complete absorption of the bone takes place, but this result is exceedingly rare. Such a case is to be found in the Warren Museum, Boston, in the arm of a man (Warren) whose humerus was entirely absorbed after fracture.

As a rule, fractures of the lower jaw unite with surprisingly good results, notwithstanding the difficulty frequently experienced in main-

taining the ends of the fractured bone in proper apposition. The rapidity with which the process is accomplished in some cases is also a great surprise. Hamilton refers to a case occurring in an adult, in which consolidation was completed in seventeen days; he also states that he had never observed a case in which union did not eventually take place, although the process had been delayed until the eleventh week. Cases are on record, however, at the present time, in considerable number, in which union was delayed from ten to twelve weeks, and of ununited fractures with false joint, which had been successfully treated after a lapse of eighteen months, as in one case reported by Dr. Physick, and in another treated by Dupuytren, four years after the receipt of the injury, which was caused by a gunshot wound.

The majority of the cases of delayed union and ununited fractures occur during middle life. Old age is not so potent a predisposing cause of these conditions as is generally supposed.

FIG. 97.



FRACTURE OF THE LOWER JAW WITH IRREGULAR AND FIBROUS UNION. (After Malgaigne.)

The frequency of delayed union and ununited fractures may be estimated from a study of Sommers upon fractures seen at the Zurich Clinic during seven years. There were 489 cases of recent fractures admitted to the Clinic during this time. Out of this number there were developed sixteen cases of delayed union, and six cases of ununited fracture.

Causes.—The causes which operate to produce delayed union and ununited fractures are twofold: Predisposing and Exciting. The former are Constitutional, the latter Local.

The Predisposing or Constitutional Causes are morbid conditions of the general system, which impair nutrition or are manifested in some peculiar dyscrasia or cachexia. Among these conditions may be mentioned extreme old age, long illness from continued fevers immediately following the injury, a debilitated condition of the system resulting from improper or insufficient food, profuse hemorrhage, pregnancy and prolonged lactation, the withdrawal of an accustomed stimulant, scorbutic, tuberculous, or the syphilitic diatheses, cancer, rachitis, and diabetes.

In fractures occurring during gestation, union is often delayed for a considerable period, sometimes until after delivery. Padieu describes a case of fractures of the tibia and fibula occurring nine days after the suppression of the menses, and in which union was delayed until the end of gestation. The process of union began ten days after delivery, and was completed at the end of a month.

The Exciting or Local Causes of delayed union or non-union of fractures of the lower jaw are the interposition of foreign substances between the fractured ends of the bone, such as loose fragments of bone, a luxated tooth, portions of muscle, ligaments, or other soft tissue of the immediate neighborhood, a large blood-clot, or arrest of circulation by tight bandages, long-continued applications of cold and moist dressings, suppuration, caries or necrosis, incomplete reduction, and mobility at the point of the fracture. Gunshot injuries are more prone to cause non-union than any other form of fracture, doubtless on account of the loss of bone-tissue at the time of the injury, and their greater liability to cause necrosis. Neglect and improper treatment are also frequent and important causes of non-union.

Fractures occurring within the synovial or articular surfaces are often very slow to unite, and occasionally they fail altogether, owing to the fact that the soft tissues can aid very little in the accomplishment of the reparative processes, and also that the fractured surfaces are constantly bathed by the synovial fluid.

Hamilton is of the opinion that too much stress is laid on motion as a cause of delayed union, and instances fractures of the ribs and of the clavicle, which are never at complete rest, to support his opinion. Motion, however, in these locations is very slight, and not to be compared with the extent of motion that may occur in an extremity or in the lower jaw. If motion is sufficient to cause a sliding of the fractured ends of the bone over each other in any direction, union is delayed or repair may be prevented altogether.

The formation of the callus takes place at many points upon the circumference of the bone, as well as between the fractured ends, the process appearing when completed somewhat like that employed by the plumber in uniting two ends of a lead pipe. This may account for the fact that slight motion does not materially interfere with the process of union.

If, however, motion is considerable, the development of the callus may be arrested, the fractured ends of the bone become rounded from absorption, and a so-called false joint be formed.

In those cases where there is complete failure in the process of repair, necrosis and suppuration sooner or later supervene, and union will not take place until after the dead bone has been exfoliated and suppuration has ceased. If there has been an extensive loss of bone-

tissue by this process, union cannot take place through the ordinary process of repair, but nature may eventually provide, in some cases, an adventitious tissue, in the form of fibrous bands or fibro-cartilage, which bind the fractured ends together. Such union, however, is not very strong, yet it serves in a measure the purpose of bone. This kind of union is sometimes formed after the operation of resection in the long bones, and in the lower jaw after the removal of necrosed bone and tumors involving the body of the jaw.

Treatment of Delayed Union.—Examples of delayed union are quite frequent, and are often met with in fractures of the lower jaw, consolidation of the callus having been delayed for various reasons. This condition in hospital practice is often associated with the drink habit. Fractures as a rule do not unite so speedily in the confirmed drunkard as in other individuals. In those cases dependent upon impaired health and long confinement in bed, consolidation will eventually, by a gradual process, take place as the health and the surroundings improve. When it is the result of imperfect reduction or mobility at the seat of the injury, a proper reduction of the fracture and the application of suitable supports for maintaining a correct apposition of the fractured ends of the bone in rigid immobility will finally result in union.

Treatment of Ununited Fractures.—The only real difference between delayed union and an ununited fracture, is one of time. When union is not complete within the usual period, thirty to sixty days after the injury, the case is termed one of *delayed union*, even though consolidation has not taken place after the lapse of several months, but gives hope of final union without a surgical operation. If, however, the case gives no hope of union without surgical interference, after the lapse of several months, it is termed an *ununited fracture*.

Various methods have been suggested from time to time for stimulating the delayed process of repair. Wilson, in 1787, suggested a seton passed through the tissues. Physick, in 1802, used a silk ribbon passed through the ends of the fractured bone. Sommé used a silver wire loop. Dieffenbach perforated the ends of the bone with a drill, and fastened the overlapping fragments together with ivory pegs. Gaillard suggested the perforation of both ends of the fracture with a metallic trocar and canula. Malgaigne perforated the tissues with the acupuncture needle. Miller penetrated the tissues subcutaneously with a tenotome knife. Detmold used a gimlet for the same purpose, and Brainard a spear-pointed drill. M. Mayor used the actual cautery, and others have used caustics.

The most satisfactory method for the treatment of this condition in the lower jaw is that of Professor Brainard, which consists in making several perforations in both ends of the bone, causing the drill to

pass freely through at points near the fracture, and then firmly fixing the fractured bones by means of the interdental splint or external splint, or by wiring the bone in the manner described in a preceding chapter.

In case the first treatment should fail, after a period of several weeks, it should be repeated. The writer has, however, never found it necessary in operations upon the lower jaw to repeat the treatment, and prefers the operation of suturing the jaw with wire to the use of the interdental splint, as the presence of the wire seems to act as an added stimulus to the tardy process of repair.

Another method of treatment of ununited fractures is by the implantation of bone from man and the lower animals. Several successful cases are to be found reported in the periodical surgical literature of the past eight or nine years. Poncet, in January, 1887, grafted a portion of the first phalanx of the great toe measuring about one inch in length and half an inch in width, taken from a recently amputated limb of a healthy man forty-five years of age, into the middle of the shaft of the tibia of a patient nineteen years of age, who had sustained a compound fracture thirteen months before, which from necrosis had failed to unite. The graft did well, and united finely with the lower fragment, but failed by about a quarter of an inch with the upper fragment, to which it was attached by a weak fibrous band.

McGill reported a compound fracture of both bones of the forearm in a young man of twenty years. The ulna united, but the radius failed, and three months later was wired unsuccessfully. One year after the original injury (1889) McGill, after freshening the ends of the bone, which left a gap of three-quarters of an inch, grafted into the space thirteen pieces of bone chiseled from the femur of a freshly killed rabbit. Five weeks later there was firm union, and at the end of four months the limb was as useful as its fellow.

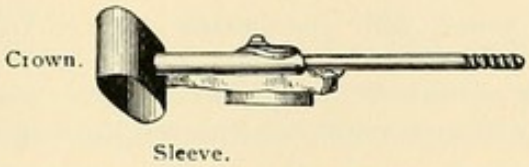
A similar case was reported by W. H. Sherwood. In this case both bones of the forearm had failed to unite, but union was secured by grafting nine segments of the femur of a rabbit, each of which was one-quarter of an inch thick. Four of the pieces were afterward removed through one of the wounds, which had failed to close. Six months after this operation union was completed and the man was able to use the arm at his usual occupation.

The writer reported to the Ninth International Medical Congress a partially successful case of bone-grafting in the lower jaw of a woman. The gap to be filled was one and a half inches in length, and had been caused by a resection for the removal of an osteo-sarcoma of the right side about eight years before.

The operation was performed in January, 1887, by grafting twelve small pieces of bone taken from the lower epiphysis of the femur of a

young rabbit. Union took place with the ramus, but not with the anterior fragment of the bone, leaving a gap of half an inch yet to be filled. A second operation was undertaken in May of the same year to fill this gap by transplanting a piece of bone, also from a young rabbit, sufficiently large to fill the space, but this failed from necrosis after sixteen days, and further attempts were abandoned. Fig. 98 shows the apparatus used to maintain the ends of the jaw-bone in their proper relations. The screw was set into the ramus, and the crown, to which a tube was soldered of proper size to receive the smooth end of the screw, was cemented to the bicuspid tooth. Extension was maintained by a set-screw fitted in the tube. The whole was constructed of 23-carat gold.

FIG. 98.



APPLIANCE FOR RETAINING THE RAMUS IN ITS NORMAL POSITION, DURING TREATMENT BY BONE-GRAFTING.

Another method of treatment is that of interstitial injections of 10 per cent. solutions of chlorid of zinc. Twenty minims of the solution is injected between and around the ends of the fragments. Menard reports a case of the successful issue of this treatment in an ununited fracture of the leg of five months' standing, in which union took place in one month after the commencement of the treatment. Glacial acetic acid has also been used for the same purpose, injected in six-minim doses between the ends of the fragments. Massage of the fractured bones and overlying tissues has been highly recommended by some authorities as an efficient method of promoting the reparative process.

REVIEW.

CHAPTER XXIII.

Are the conditions known as delayed union and ununited fractures common?

In what portions of the body are they most often found?

How are delayed union and ununited fractures produced?

What is the condition of the ends of the bone in ununited fracture?

What is the condition of false joint?

Does complete absorption of the fractured bone ever occur?

What, as a rule, are the results of fracture of the lower jaw?

How long may union sometimes be delayed?

At what period of life do these conditions most often occur?

What is the relative frequency of delayed union and ununited fractures?

How are the causes classed?

What are the predisposing causes?

What are the exciting causes?

In what locations may fractures fail altogether to unite?

What are the reasons for this?

Is slight motion at the point of fracture detrimental to the consolidation of the callus?

What degree of motion would be detrimental?

Where does the formation of the callus take place, and how?

When motion is considerable, what occurs at the point of fracture?

What result usually follows a complete failure in the process of repair?

Will the reparative process be established while exfoliation and suppuration are going on?

How are fractured bones, with considerable loss of tissue, sometimes united?

What is the character of this form of union?

Does this form of union ever follow resections of the long bones or of the lower jaw?

What is the treatment for delayed union of fracture of the lower jaw?

What are the constitutional conditions which must be established in these cases in order to promote the process of repair?

What is the difference between delayed union of a fracture and an ununited fracture?

What feature, besides time, determines the difference?

What are some of the methods which have been suggested for the treatment of ununited fractures?

Which method is generally considered best for ununited fractures of the lower jaw?

If the first attempt fails, how soon after may the operation be repeated?

What is the writer's preference between wiring the jaw and the use of interdental splints in these cases?

What are the reasons for this preference?

Describe the method of treatment by the implantation of bone.

Has this treatment proved successful?

Describe the interstitial injection method.

What drugs are used for the purposes of injection?

State the strength of the solutions and the dose of each.

What is claimed for the massage treatment?

CHAPTER XXIV.

DISLOCATION OF THE INFERIOR MAXILLA.

Definition.—*Dislocation* (Lat. *dislocatus*—to put out of place). *Luxation* (Lat. *luxatus*—to put out of joint).

A dislocation or luxation is a displacement of a part from its proper situation.

Two or more bones whose articular surfaces have lost, wholly or in part, their natural connection would be said to be dislocated or luxated, and the condition would be termed a dislocation or a luxation.

Dislocations in general are classed as simple, compound, and complicated; partial and complete; recent and ancient; primitive and consecutive; spontaneous, traumatic, and relapsing; pathologic and congenital; unilateral and bilateral; single and double.

A Simple Dislocation is one without other important injury of the joint, and with no communicating wound.

A Compound Dislocation is one having an external wound which communicates with the joint.

A Complicated Dislocation is one having any serious lesion not comprehended under the term compound, especially fracture of the misplaced bones at their articular surfaces.

Partial Dislocations are those in which some portions of the disturbed articulating surfaces continue to remain in contact.

Complete Dislocations are those in which the articular surfaces of the bone override each other.

The terms Recent and Ancient explain themselves.

A Primitive Dislocation is one in which the dislocated surfaces remain in the position in which they were thrown at the time of the luxation.

A Consecutive Dislocation is one in which the dislocated surfaces have assumed a new position. This is usually the result in luxations of long standing.

Spontaneous Dislocations are those which are not caused by external violence.

Traumatic Dislocations are those which occur as the direct result of external violence.

A Relapsing Dislocation is one which is prone to recur on very slight provocation, on account of the relaxed state of the ligaments, or on account of active movement of the joint having been permitted before repair of the articular capsule was completed after a previous luxation.

A Pathologic Dislocation is one produced by a diseased state of the structures of the joint.

Congenital Dislocations are those which are present at the time of birth.

When bones like the inferior maxilla, the hyoid and sternum, having a median position in the body, are dislocated upon one side only, it is termed Unilateral; when both sides are luxated, it is called Bilateral.

When two bones corresponding to each other, in opposite sides of the body, are luxated at the same point, it is termed a Double Dislocation; but when only one is luxated, it is called a Single Dislocation.

Dislocations occur most frequently in middle life, less often in old age, and still more rarely in childhood.

The action of the muscles alone sometimes causes a dislocation, and in all dislocations caused by external violence the muscles play an important part. It is almost impossible to produce a dislocation upon the cadaver when the muscles can no longer take part in producing the displacement.

As a rule, the force which causes the dislocation of a long bone is applied at the opposite extremity, rarely at the point of dislocation.

Dislocations of the Lower Jaw.—This accident is rarely met with in the extremes of age, but is most common during middle life. It occurs more frequently in women than in men.

The forms of luxation of the inferior maxilla are four in number—complete, incomplete, unilateral, and bilateral.

A Complete Dislocation of the lower jaw is that condition in which one or both condyles have been forced entirely out of the glenoid cavity. (Fig. 99.)

An Incomplete Dislocation is one in which the condyle of one or both sides rests upon the interarticular cartilage, directly over the articular eminence.

A Unilateral Dislocation, as the term implies, relates to a displacement of one side only.

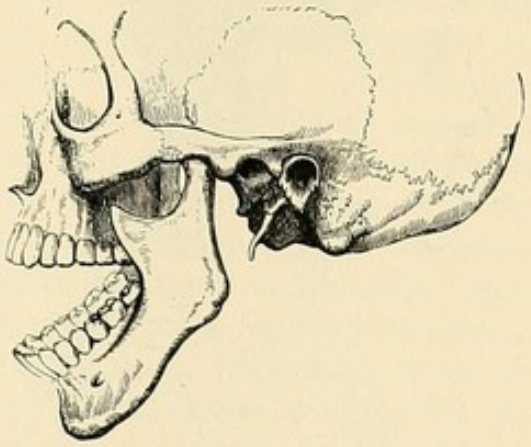
A Bilateral Dislocation is one in which a displacement involving both sides has occurred.

The great majority of dislocations of the temporo-maxillary joint are bilateral. Out of twenty-eight cases mentioned by Giraldés, fifteen were bilateral; of seventy-six cases reported by Malgaigne, fifty-four were bilateral, and thirty-one of these were in women.

On account of the anatomy of the temporo-maxillary joint, luxation cannot occur backward, upward, or outward; consequently the direction of the dislocation, when uncomplicated, is forward. When complicated with fracture of the rim of the glenoid fossa a backward or outward dislocation may take place, but without fracture of the bone the dislocation must always be forward.

The temporo-maxillary joint is an arthrodial joint, and provided with an interarticular fibro-cartilage, oval in form, and thicker at the margins than at the center. A pouch of synovial membrane is interposed between the fibro-cartilage and the glenoid cavity, and between the articular surface of the condyle and the fibro-cartilage is a smaller synovial pouch. Occasionally the pouches are united through a deficiency in the center of the fibro-cartilage. The fibro-cartilage acts as a buffer to prevent shocks from a violent closing of the jaw,

FIG. 99.



COMPLETE DISLOCATION OF THE LOWER JAW, SHOWING THE ANATOMICAL RELATIONS.
(After Sir Astley Cooper.)

and to guard against injury to the brain through the thin bony plate of the glenoid cavity. The joint is furnished with two lateral ligaments, a short *external ligament*, extending from the tubercle of the zygoma to the outer and posterior borders of the neck of the condyle, and a long, flat *internal ligament*, which extends from the spinous process of the sphenoid bone to the inner border of the dental foramen on the inner side of the ramus of the jaw.

The *Capsular Ligament* consists of a few fibers which arise from the margin of the glenoid cavity, and blend below with the lateral ligaments.

The form of this articulation admits of free motion of the jaw from side to side, forward and backward, upward and downward, thus enabling the teeth to thoroughly triturate the food.

The muscles which elevate the lower jaw are the temporal, masseter, and internal pterygoid. The fibro-cartilage maintains its proper relation to the condyle in all of the ordinary movements of the jaw,

but if depression of the jaw is carried beyond a certain limit, a forward dislocation may result.

The articular eminence at the inner side of the zygoma is coated with cartilage, and is in contact with the fibro-cartilage of the joint in front. Ordinarily the condyle never reaches the top of the eminence, although it glides forward when the mouth is wide open. When the depression of the jaw is strained in this position, the external pterygoid muscle forcibly contracts and draws the condyle forward and over the eminence into the zygomatic fossa, leaving the fibro-cartilage behind, while the condyle becomes more or less fixed in its new position by the contraction of the elevator muscles.

FIG. 100.



DISLOCATION OF THE LOWER JAW, SHOWING POSITION OF THE MOUTH AND JAW AND THE DEPRESSION IN FRONT OF THE EAR. (After Fergusson.)

Causes.—The Predisposing Causes of dislocation of the lower jaw are relaxation of the articular ligaments, and pathologic changes in the structures of the joint, from rheumatoid arthritis.

The Exciting or Immediate Causes are muscular contraction and violence applied, either within the mouth or externally upon the chin; yawning, vomiting, shouting, immoderate laughter, or anything which opens the mouth widely, may induce a dislocation. Epileptic convulsions have also been known to cause it.

Among the forms of violence which may cause the accident are operations within the mouth, like taking impressions, plugging the lower teeth, the use of gags, and the extraction of teeth. The forms of external violence which might produce the luxation are falls and blows upon the chin, having a downward direction, and occurring when the mouth was widely extended.

Symptoms.—The diagnosis of a recent luxation is an exceedingly simple matter.

The symptoms are most marked when the dislocation is bilateral. Under this condition the mouth is extended to its widest limit and firmly fixed in that position, while speech and deglutition are very greatly impaired. The jaw is thrust forward so that the lower teeth project beyond the upper, and on account of the impaired ability to swallow, the saliva constantly dribbles from the mouth. Immediately in front of the tragus of the ear is a marked depression over the now empty glenoid fossa, while by passing the finger under the lower border of the malar bone the coronoid process can be distinctly felt. There is also a considerable prominence above the zygoma, the result of the contraction of the temporal muscle, and of the presence of the condyle in its new position. (Fig. 100.) When the dislocation is unilateral the chin will be carried to the right or left, according to the location of the displacement. If the left side is dislocated, the chin is carried to the right, and *vice versa*. The depression in front of the ear will be found only on the injured side. The other symptoms are generally the same, though not always so well marked; occasionally they may be so imperfectly defined as to escape notice altogether.

Treatment.—The reduction of dislocations of the jaw, if of recent date, is a matter of easy accomplishment; but when the luxated jaw has remained unreduced for any considerable time, adhesions take place, and it then becomes a much more difficult task.

In reducing dislocations of the jaw, the force is to be applied in such a way as to depress the angle, while at the same time the chin is carried toward the superior maxillæ. The effect of this application of mechanical force is to depress the condyle to a level with the articular eminence, when the contraction of the internal pterygoid muscle carries it back into the glenoid fossa.

The method usually employed for reducing dislocations of the jaw is for the patient to be seated in a low chair, the surgeon taking his position directly in front.

The thumbs of the operator having been wrapped with a few turns of bandage, or other suitable protection, are placed upon the ends of the posterior inferior molars of either side, as near the ramus as possible, the fingers of each hand grasping the jaw upon the outside; forcible downward and backward pressure is then made upon the molar teeth, while at the same time the chin is elevated with the fingers. This liberates the condyle from its false position, and allows it to slip over the articular eminence and into the glenoid fossa.

Roth has recently suggested a new method of reducing dislocations of the jaws, as follows: The patient is seated in an ordinary chair, the surgeon standing in front of him in the usual way. He then flexes

himself at the hips, and causes the patient to lean forward and place his forehead on the breast of the operator. The neck of the operator is now flexed so that the chin comes in contact with the head of the patient at the upper portion of the occipital bone, thus securing a firm hold of the head between the chin and the breast. The protected thumbs are now inserted into the patient's mouth, and placed upon the inferior molar teeth, while the fingers of both hands grasp the lower border of the jaw, and force is applied in the usual manner.

In exceptional cases it may be necessary to reduce one side at a time, but care must be exercised to prevent a redislocation of the condyle of one side while the other is being reduced.

In unilateral dislocations, efforts at reduction should be applied only to the injured side.

Another method, which was first suggested by Ambrose Paré, is often valuable in those cases in which the methods just described have failed; this is to place wedges made of cork, wood, or soft rubber, between the molar teeth, and then gradually but forcibly carry the chin upward toward the superior maxillæ. The position of the operator in this method is behind the patient, whose head rests upon the breast of the surgeon or upon the back of the chair, while the hands of the operator are placed under the chin. This position gives perfect control of the patient's head, a matter of considerable moment if the reduction proves difficult.

By using the wedges in this manner, the principle of the lever and fulcrum is introduced; the jaw acts as a lever, and the wedges as the fulcra. The condyles are thus forced downward until they pass the *eminentia articularis*, when they readily slip into the glenoid cavity.

Nélaton's method was to stand in front of the patient and place the thumbs upon the coronoid processes, either within or without the mouth, and grasp the mastoid processes with the fingers, gradually forcing the jaw into position; or sitting behind the patient, place the thumbs upon the nape of the neck, and with the fingers over the ascending ramus endeavor to draw the jaw backward into position.

Dislocations of long standing are often difficult to manage, on account of the inflammatory adhesions or pathologic changes which may have taken place in the structures of the joint. Heath mentions the case of a woman for whom Pollock successfully reduced a dislocation of four months' standing, by inserting wedges between the molar teeth and elevating the chin by means of a strap tourniquet passed over the head. Several other cases are on record in which successful reduction was accomplished after periods ranging from thirty-five to ninety-eight days after the accident. In these cases the reduction is greatly facilitated by the use of anesthetics, as muscular contraction is thereby overcome, and the inflammatory adhesions are more readily broken up.

Occasionally dislocations of the jaw may be reduced spontaneously, or with no other assistance than that which the injured individual has been able to apply.

Recurrent dislocation of the jaw is a condition dependent upon the existence of a ruptured capsule, great relaxation of the articular ligaments, or some change in the form of the articular eminence. Dislocations of this character are exceedingly rare.

After reduction of a dislocated jaw, it is necessary to limit its movements for a week or ten days in order to guard against the possibility of the condyle again slipping out of place, and also to give rest to the injured parts. This may be accomplished by the application of a four-tailed bandage, and prohibiting talking or the mastication of food. The diet should, of course, be limited for several days to liquid food.

In cases of recurrent dislocations of the jaw, the tendency may be overcome, as suggested by Goodwillie, by the adjustment of an apparatus composed of a netted silk cap covering the chin, and attached by elastic bands to a close-fitting skull-cap of the same material, reinforced with leather. This permits the ordinary movements of the jaw, but at the same time prevents the opening of the mouth to such an extent as to allow the condyles to slip out of position.

Subluxation of the Jaw.—Subluxation of the jaw is a condition in which the condyle partially slips from the glenoid fossa in a forward direction, and in front of the interarticular cartilage, when the mouth is being opened, and on closing it the condyle goes back again to its place, with a clicking, snapping sound. Sometimes it catches for a moment when the mouth is widely extended, and causes anxiety on the part of the patient for fear that the jaw is dislocated.

This condition is usually seen in young women of delicate health and physique, and in certain cases of malocclusion of the teeth, in young subjects, especially in those having a short or underhung jaw.

Causes.—It is due in some cases to a relaxed condition of the articular ligaments; in others to strain upon the muscles incident to an effort to obtain a comfortable occlusion of the teeth in mastication; and in still others, in all probability, to changes in the joint as a result of rheumatoid arthritis. The exciting causes are yawning, biting hard substances, and fatigue from operations upon the teeth which necessitate a long-continued open position of the jaws.

Treatment.—Reduction is generally accomplished by the subject of the accident making a lateral movement of the jaw, or by upward pressure upon the chin. External support may sometimes become necessary to overcome the tendency to the accident, as already described for recurrent dislocations.

REVIEW.

CHAPTER XXIV.

What is the meaning of the terms dislocation and luxation?
How are dislocations classed?
What is a simple dislocation?
What is a compound dislocation?
What is a complicated dislocation?
What is a partial dislocation?
What is a complete dislocation?
What is a primitive dislocation?
What is a consecutive dislocation?
What is a spontaneous dislocation?
What is a traumatic dislocation?
What is a relapsing dislocation?
What is a pathologic dislocation?
What is a congenital dislocation?
To what bones are the terms unilateral and bilateral dislocations applied?
What terms are applied to dislocations of similar bones in opposite sides of the body?
At what period of life are dislocations most likely to occur?
What part do the muscles play in the production of dislocations?
At what point is the force usually applied to cause a dislocation of a long bone?
At what period of life is dislocation of the lower jaw most likely to occur?
In which sex does it most frequently occur?
Name the forms of luxation of the lower jaw.
What is a complete dislocation of the lower jaw?
What is the position in incomplete discoloration of the lower jaw?
What is understood by complete unilateral and bilateral dislocations?
What is understood by incomplete unilateral and bilateral dislocations?
What is always the direction of uncomplicated dislocations of the lower jaw?
Under what conditions may backward and outward dislocations occur?
What group of muscles fix the jaw in its abnormal position?
What are the predisposing causes of dislocations?
What are the exciting causes?
What are the symptoms of dislocation of the jaw?
Describe the common method of reducing a dislocation of the jaw.
Describe Roth's method.
Describe the Paré or wedge method.
What is Nélaton's method?
Describe Pollock's method for reducing dislocations of long standing.
What is the after-treatment for dislocations of the jaw?
What is the treatment for recurrent dislocation?
What is subluxation of the jaw?
In what class of individuals does it occur?
What are its causes?
What form of treatment sometimes becomes necessary?

CHAPTER XXV.

ANCHYLOSIS OF THE JAWS.

Definition.—*Anchylosis* (Greek ἀγκύλος, meaning a stiffening of the joints or of the eyelids).

The coalescence of two bones, so as to prevent motion between them.

Immobility of a joint, from any cause, is termed Anchylosis.

The temporo-maxillary articulation, being a synovial joint, is subject to the same class of diseases that affect joints of like structure in other portions of the body, and with corresponding results.

Anchylosis of the jaws may therefore be classed as Temporary and Permanent, Incomplete or False, Complete or Bony, Fibrous and Osseous, Unilateral and Bilateral.

Anchylosis in any of its forms is the result of injury, accidental or surgical, or of disease.

Temporary Anchylosis.—Temporary closure of the jaws is a condition of tonic spasm of the muscles of mastication, especially of the masseter and internal pterygoid, resulting from prolonged and severe irritation of the third division of the trifacial nerve, and inflammatory conditions of the premaxillary soft tissues. This condition must not be confounded with trismus, the result of tetanic spasm of the muscles of mastication.

Causes.—The most common causes of this affection are the difficulties which often attend the eruption of the inferior third molars, or wisdom-teeth. It often occurs that these teeth are misplaced in the jaw, or, by reason of the shortness of the horizontal ramus, there is not room between the second molar and the ascending ramus for the third molar to make its exit. As a consequence of these conditions, the eruption of these teeth is slow and difficult, or altogether impossible. The effort, therefore, of the tooth to gain its normal position in the dental arch superinduces irritation, which may be made manifest either in spasm of the muscles, or in a suppurative inflammation of the alveolar and gingival tissues, or an extensive periostitis of the jaw and cellulitis involving the whole side of the face.

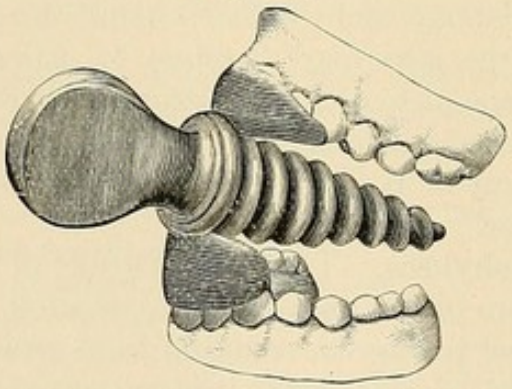
Among the other conditions which may cause a temporary closure of the jaws may be mentioned alveolar abscess, associated with

the molar teeth (usually the third molars), inflammation of the parotid glands, suppurative tonsillitis, and necrosis of the jaw.

Treatment.—In those cases dependent upon the difficult eruption or the non-eruption of the third molars, the first effort at curing the affection should be directed to the removal of the cause. This cannot be accomplished, in many instances, without great suffering to the patient, except by the administration of a general anesthetic, on account of the rigidity of the muscles and the hypersensitive condition of the tissues involved.

It is often recommended that the jaws should be separated by slow degrees, by the use of wedges, the screw-gag, etc., in the hands of the patient. This procedure, in the experience of the writer, has never been other than a complete failure, for the reasons that the patient will not persist in the effort on account of the severe pain often inflicted; and when the attempt has been made by the surgeon, he has been obliged to desist for the same reason.

FIG. 101.



METHOD OF APPLYING THE SCREW FOR OPENING THE JAWS. (After Grady.)

After the patient is profoundly anesthetized (complete anesthesia is always necessary) the jaws should be gradually separated by means of a screw-gag (Fig. 101), lever, or other device, and the offending tooth extracted. This can best be accomplished in those cases where the tooth is partially erupted by the aid of the Physick forceps. The writer has rarely found it necessary to extract the second molar, so often advised in many of the text-books, as a necessary operation, in some cases, to permit the extraction of the third molar. With proper instruments and ordinary skill, there is no difficulty in the way of extracting the third molar which cannot be surmounted.

In those cases in which the tooth is impacted the gum-tissue can be excised, the covering bone cut away with chisel or surgical bur, and the tooth lifted from its crypt with elevator or forceps.

In exceptional cases, where the third molar is in a horizontal position in the jaw, and has already impinged upon the roots of the second molar, causing absorption at the point of contact, it becomes

necessary for this reason to extract the second molar. The third molar should not be permitted to remain, however, even under these circumstances, if there has been any suppurative inflammation in connection with it, on account of the liability to frequent recurrence of the inflammatory symptoms.

The irritation rapidly subsides upon the removal of its cause, and the jaw soon regains its normal function.

In those cases associated with periostitis of the jaw and cellulitis it will be necessary to evacuate any accumulations of pus within the mouth, if possible, and irrigate the mouth and abscess-cavities with antiseptic solutions.

When the closure of the jaws is the result of an alveolar abscess, the offending tooth can better be sacrificed than that chances be taken of a recurrence.

Cases dependent upon inflammation of the parotid glands and suppurative tonsillitis are usually of short duration, the function of the jaw being completely restored upon the subsidence of the inflammatory symptoms.

When it is the result of necrosis of the jaw, a restoration of the normal function of the joint is sometimes delayed for a considerable time, depending upon the location and the extent of the necrosis and the inflammation and swelling of the adjacent tissues.

Permanent Anchylosis.—Permanent closure of the jaws is a much more serious condition than that just described. In this form of the affection articulation is imperfect, the mastication of food impossible, and when the teeth are tightly closed together, as frequently occurs, deglutition is more or less difficult, and in some cases the saliva constantly dribbles from the mouth, making the conditon of these patients really deplorable in the extreme.

This form of the affection may be divided conveniently into two classes, viz: Incomplete or False Anchylosis, and Complete or True Anchylosis. A fibrous or cicatricial anchylosis (Fig. 102) would be classed as incomplete or false, while a bony anchylosis would be classed as true.

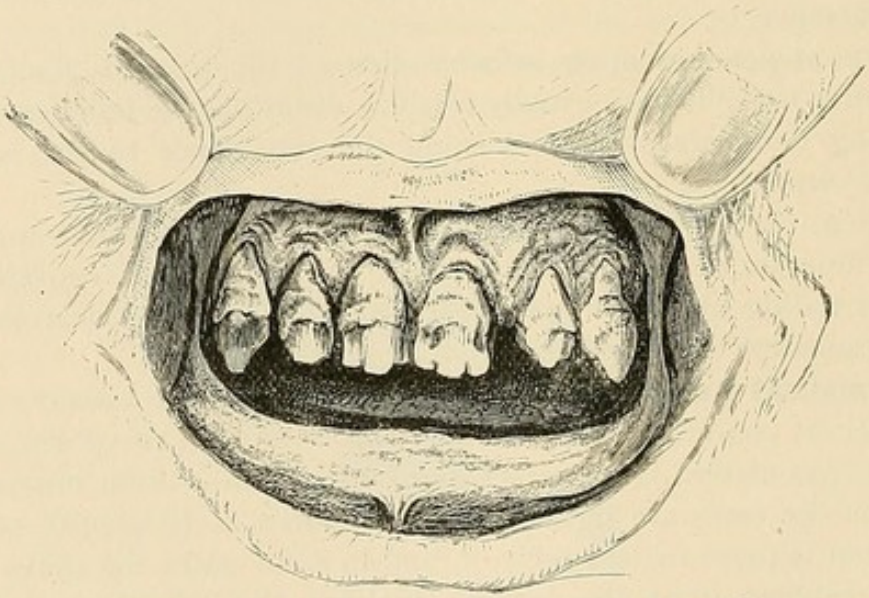
Causes.—An incomplete or false anchylosis may be either intra-articular or extra-articular, and is due to the formation of fibrous adhesions within the joint, between the opposing surfaces, or by the deposition of fibrous exudates within the tissues surrounding the joint; cicatrization and contraction of the articular ligaments of muscles and integument, following injuries of an accidental or surgical nature; syphilitic ulceration of the oral mucous membrane, ulcerative mercurial stomatitis, and gangræna oris in children.

The formation of cicatricial bands is the usual result of extensive ulceration and loss of tissue; similar lesions are followed by a like result

in the oral mucous membrane and cheeks. The cheeks and mucous membrane of the mouth are exceedingly elastic in their normal condition, permitting a very considerable expansion without injury to their structure; but when the character of these tissues has been changed or replaced by a mass of cicatricial tissue, elasticity is lost, and if the cicatrix involves the entire cheek from one jaw to the other, even if but one side is affected, it will as completely and effectually bind the jaws together as would a bony anchylosis of the articular surfaces.

Professor Gross says bone is frequently formed in the new tissue, and occasionally assumes the form of an osseous bridge, extending from one jaw to the other, and thus serves to more firmly bind them together.

FIG. 102.



CICATRICIAL ANCHYLOSIS. THE LIPS ONLY COULD BE OPENED; LOWER JAW EDENTULOUS. (After Weiss.)

Complete or true anchylosis is usually the result of disease or injuries affecting the joint, the most common being acute arthritis following the exanthematous fevers, in connection with suppurative conditions of the middle ear and traumatic injuries, like blows upon the side of the face, dislocations, or fractures into the joint. In elderly persons, chronic rheumatoid arthritis may sometimes produce the affection.

Diagnosis.—The diagnosis between a fibrous and an osseous anchylosis can only be determined, in many cases, by placing the patient under an anesthetic. If the anchylosis is fibrous, slight motion will be discovered under the relaxation produced by the anesthetic, and by the aid of a screw-gag, wedges, or levers, the jaws may be forcibly separated and the adhesions broken up; while on the other hand, if the anchylosis is bony, the jaws will be found firmly set, and all efforts to separate them will be unavailing. In this latter form there is a coales-

cence of the opposing articular surfaces, producing absolute immobility.

In permanent closure of the jaws the causative lesion is usually found upon one side only, though occasionally both sides are affected. In bilateral anchylosis the original cause of the difficulty can generally be traced to disease or injury affecting but one side, the opposite side becoming affected later, as a consequence of disuse.

When a joint in any part of the body is kept motionless for a considerable period of time, the bones which constitute the joint take on a passive inflammation, with the exudation of plastic lymph, which later becomes organized, resulting in a fibrous or an osseous anchylosis. The temporo-maxillary joint has the same tendencies, and when rendered motionless through cicatricial contraction of the cheek and mucous membrane will sooner or later become completely anchylosed, unless relieved by a surgical operation.

Treatment.—The treatment of false anchylosis caused by contraction of the mucous membrane and muscles of the cheek is by no means an easy task. The method generally suggested, of cutting through the cicatrix and separating it from the bones, is frequently worse than useless, as it quickly reunites, leaving an augmented area of cicatricial tissue, which, upon secondary contraction, increases the difficulty instead of relieving it.

Heath mentions a successful case described by Holt, in which, by the application of silver shields, adapted to the teeth of the upper and lower jaws, extending into the incisions made by the separation of the cheek from the alveolar walls, and the removal of the cicatrix in the mucous membrane, reunion was prevented, while eventually the denuded inner surface of the cheek was covered with a normal mucous membrane, which permitted the mouth to be opened to its fullest extent (Fig. 103).

Occasionally the transplantation of tissues, as suggested by Dieffenbach, will in a measure restore the normal elasticity of the cheek and permit the jaws to be opened; but unfortunately it rarely happens that a sufficient amount of healthy mucous membrane remains in the locality of the cicatrix that could be utilized for the purpose of covering the area occupied by the cicatricial tissue.

A flap of skin taken from the immediate neighborhood was transplanted upon the inner side of the cheek by Jaesche, with good results.

Grafting, after the Thiersch method, would be still more troublesome to accomplish, on account of the difficulties of maintaining the grafts in position until adhesion had taken place; yet this method would be worthy of a trial in this location if a suitable case presented.

Dieffenbach recommends the formation of a false joint upon the affected side, by dividing the jaw, section of the bone to be made be-

hind the seat of the cicatrix in the ascending ramus; but this proves of no real value, as the impediment to the mobility of the jaw lies forward of this, through the cicatrix.

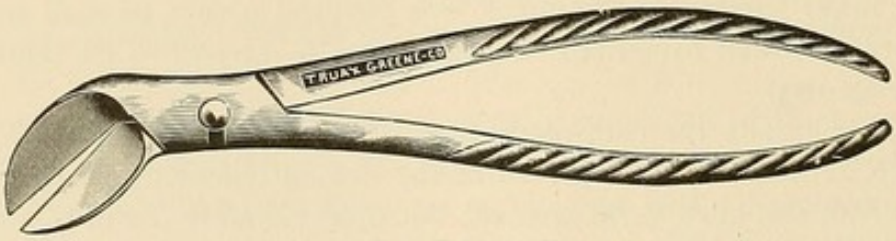
Rizzoli's operation for forming an artificial joint is made in front of the cicatrix, within the mouth, the object in operating within the mouth being to avoid a scar upon the external tissues of the face. The operation consists in first dividing the soft tissues covering the jaw, and then, with powerful cutting forceps (Fig. 104), dividing the bone. The disadvantages of this operation are the risks of splintering the

FIG. 103.



CICATRICIAL ANCHYLOSIS. THE RESULT OBTAINED BY DIVISION OF THE CICATRICES AND PREVENTING REUNION BY PROTECTING SILVER SHIELDS FITTED TO THE TEETH, AND GRADUAL SEPARATION BY THE USE OF WEDGES. (Holt, after Heath.)

FIG. 104.



ANGULAR BONE-CUTTING FORCEPS.

bone, and the tendency of a mere fracture of the jaw—for this is nothing more—in healthy individuals to reunite. He claimed, however, to prevent this by placing a piece of gutta-percha between the ends of the bone.

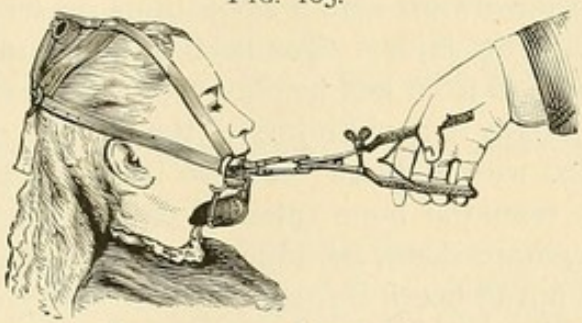
Esmarch's operation for forming a false joint is also made in front of the cicatrix, and consists in removing an inverted V-shaped section of bone, through an external incision made at the lower border of the jaw. The section can be made very neatly and quickly by a small circular saw, revolved by the surgical engine. In bilateral anchylosis, both sides of the jaw are to be treated in the same manner. Esmarch's

operation has given the most satisfactory results, and is more generally practiced than any other. Daily motion of the jaw, however, is necessary after an operation, to prevent the formation of rigid adhesions.

The treatment of false anchylosis of the jaws, dependent upon fibrous adhesions between the opposing articular surfaces and in the tissues about the joint, presents considerable difficulty. The object of any form of treatment must be the restoration of the mobility of the joint, or to form an artificial joint, which will, in a measure at least, restore the function of the jaws.

Mechanical Treatment.—In exceptional cases, in which the adhesions are of recent formation, mobility of the joint may be secured by forcible extension of the jaws, under an anesthetic, by means of a screw-gag, or wedges, introduced between the teeth. The adhesions by this procedure are broken up, and mobility obtained, but on ac-

FIG. 105.



GOODWILLIE'S APPLIANCE FOR TREATMENT OF FIBROUS ANCHYLOSIS OF THE TEMPORO-MAXILLARY ARTICULATION. (After Goodwillie.)

count of the tendency of the adhesions to reform, it becomes necessary to repeat the extension of the jaws daily, for weeks and months, and even then it is not always successful.

Goodwillie's apparatus (Fig. 105) is the most reliable for the daily extension of the jaws which is so necessary after any of the operations for cicatricial anchylosis, and following the mechanical method of treatment.

In cases of long standing the mechanical treatment is rarely of any permanent value, even when carried out in the most persistent manner, and with the heartiest co-operation of the patient. Most surgeons have, therefore, abandoned this line of treatment as offering very little inducement from the curative standpoint.

Surgical Treatment.—Subcutaneous division of the fibrous bands by a tenotome introduced into the joint through the mouth has been frequently tried, and with somewhat better results. The constant tendency, however, to a reunion of the divided bands is a serious drawback to the permanent success of this operation.

The operation which promises the best results, in both fibrous

and osseous anchylosis, is excision of the head of the condyle and a portion of the neck of the jaw, thus forming an artificial joint. Less formidable operations for establishing an artificial joint would be that of Dieffenbach, which has already been mentioned as dividing the ascending ramus, or that of Esmarch, which removes a wedge-shaped segment from the body of the jaw near the angle.

The method of procedure usually followed in excision of the condyle is that described in Stimson's "Manual of Operative Surgery," viz:

"An incision is begun at the lower margin of the zygoma, close in front of the temporal artery, where it adjoins the ear, and carried forward along the zygoma 1¼ inch, the tissues being divided, layer by layer, until the bone is reached. A second incision, involving only the skin, is then carried from the center of the first, directly downward for about an inch. The soft parts are carefully separated with elevator and knife from the margin of the zygoma and the outer surface of the joint, and drawn downward with a hook, thus preserving the parotid nerves and vessels from injury. The neck of the condyle is then freed by working around, in front and behind, with a small elevator, keeping close to the bone, so as to avoid injury to the internal maxillary artery, and finally divided with a chisel. If there is bony union between the condyle and the temporal bone (glenoid cavity), the chisel must be again used to separate them, its edge being kept directed somewhat downward, so as not to break through into the cavity of the cranium." Antiseptic precautions should be rigidly carried out through the operation and in the after-treatment. Drainage for the wound should be provided; but this may be dispensed with after forty-eight hours if there are no signs of inflammation. Movements of the jaw may be commenced on the second or third day after the operation, to prevent the formation of adhesions.

REVIEW.

CHAPTER XXV.

What is anchylosis of a joint?

How are the various forms of anchylosis of the temporo-maxillary joint classed?

What is temporary anchylosis?

What is permanent anchylosis?

What is false anchylosis?

What is true anchylosis?

What are the causes of anchylosis of the jaws?

What is the term applied to anchylosis of one side?

What is the term applied when both sides are affected?

What are the causes of temporary closure of the jaws?

What muscles are affected in this condition?

Is this the same as trismus resulting from tetanus?

How are the muscles of mastication affected by the local irritation induced by an impacted third molar?

What is the treatment for trismus resulting from an impacted third molar?

What is the objection to attempting this operation without an anesthetic?

What style of forceps is indispensable for removing partially erupted third molars?

Under what conditions may it become necessary to remove the second molar?

What should be the method employed for removing an unerupted third molar?

Describe the conditions as to speech, mastication, etc., in permanent closure of the jaws.

What are the causes of incomplete or false anchylosis?

How may cicatrices of the oral mucous membrane and of the cheek cause false anchylosis?

What is the condition of the joint in fibrous anchylosis?

What is the condition of the joint in osseous anchylosis?

How may the diagnosis between false and true anchylosis be determined?

What is likely to occur if a joint is kept motionless for any considerable length of time?

What are the methods of treatment for false anchylosis caused by cicatrices of the cheeks?

What was Holt's method?

Describe Dieffenbach's method for transplanting tissue.

What are the difficulties in the way of using Thiersch grafts?

Describe Dieffenbach's method of forming a false joint.

What was Rizzoli's method?

How does Esmarch's operation differ from this?

Which of these operations gives the best results?

What are the methods of treatment adopted in fibrous and osseous anchylosis?

In what class of cases is the mechanical treatment sometimes useful?

Describe the operation for excision of the condyle.

How soon may movement of the jaw be commenced?

What is the value of such movements?

CHAPTER XXVI.

PERIOSTITIS OF THE JAWS.

Definition.—*Periostitis* (from the Greek περί—around, ὀστέον—bone, and ιτις—inflammation). Inflammation of the periosteum.

The periosteum is a fibrous membrane which invests or covers the external surfaces of bones, except at the articular surfaces and at the points of insertion of tendons and ligaments. It is composed of two layers—an outer or fibrous, and an inner or osteogenetic. The periosteum serves to give attachment to the surrounding tissues, and as a means of nourishment, growth, and regeneration of bone.

Periostitis of the Jaws.—The jaws, like bones in other parts of the body, are subject to inflammatory conditions of their periosteal covering, which may be made manifest in either an Acute or a Chronic form.

Acute periostitis of the jaws may be either a simple local inflammation, which may become suppurative, forming subperiosteal abscesses, or it may be diffuse and infective, depending upon the cause of the disease, the severity of the attack, and the diathesis of the patient. In an individual of good habit, the disease would in all probability not progress beyond a simple local inflammation, with suppuration; while on the other hand, if the individual were possessed of a strumous or tubercular diathesis, or was anemic, or recovering from a protracted illness, etc., the inflammation would be more likely to take on a diffuse or infective character.

The disease may terminate either in resolution, suppuration, or necrosis.

Simple local periostitis is the most common form of the disease. It occurs more frequently in the inferior than in the superior maxilla, runs a more rapid course when located in the lower jaw, and almost invariably terminates in necrosis of the bone unless the inflammatory symptoms are promptly arrested. Occasionally the disease will attack opposite sides of the jaw at the same time, and gradually extend until the entire jaw is involved; when beginning upon one side only, it may cross the median line and involve the jaw of the opposite side to a greater or less extent.

Causes.—The exciting causes of acute local periostitis are the diffi-

cult eruption of the deciduous or permanent teeth, irritation from devitalized teeth, traumatic injuries, the effects of the eruptive fevers, typhoid conditions, prolonged anemia in young children, syphilis, scorbutus, and long exposure to cold; chemical poisons, such as mercury, carried to salivation, and the vapor of phosphorus. In children of tubercular diathesis the disease may occur with no other evidence of the cause than the constitutional taint, as is frequently observed in periostitis of other portions of the body.

The predisposing causes are the scrofulous, tubercular, and syphilitic diatheses, and all other conditions which produce a lowered vitality.

Symptoms.—The symptoms of the disease are elevation of temperature, with general constitutional disturbance; swelling and congestion of the gum and of the affected side of the face; severe, tense, bursting pains, generally worse at night; the teeth become loose and raised from their alveoli; pressure or percussion upon the teeth causes excruciating pain. The swelling often extends down the neck, and when pus forms it may point beneath the jaw, or burrow downward between the muscles, following the connective tissue, and point at various locations above or even below the clavicle. Spasmodic closure of the jaws is not an infrequent accompaniment of the disease.

In the milder cases of simple acute periostitis, the disease may be very insidious in its approach. The pain is intermittent, usually occurring at night; the swelling of the gum and side of the face is less marked, and may be overlooked altogether, while the teeth may not give evidence of being sore, unless sharply percussed. For these reasons advice may not be sought and the disease not recognized until considerable mischief has been done by the formation of periosteal abscesses and death of the bone.

The general tendency of acute periostitis is to end in suppuration. When suppuration occurs the periosteum is dissected from the bone, and necrosis is induced.

Acute Diffuse Periostitis.—This form of the disease occurs most frequently at the age of puberty, in children of strumous habit, and those suffering from impoverished conditions of the blood. It runs a more rapid course than simple acute periostitis, the general and local symptoms are more aggravated, pus accumulates in greater quantity, and the disease invariably ends in necrosis of the bone.

Causes.—The direct or exciting causes of the disease are external injuries, septic infection, eruptive fevers, exposure to cold and dampness, etc. Occasionally, in young children of tubercular or strumous diathesis, a considerable portion of the jaw may become necrosed without any previous history of injury, exposure, derangement of health, or other discoverable cause which seems adequate to account for the disease.

Treatment.—The treatment of acute simple, and of diffuse periostitis, if recognized in their earlier stages, should consist of energetic measures: the extraction of devitalized teeth, local depletion by free scarification of the gum, cold applications,—or if these prove painful, hot fomentations, or hot water held in the mouth,—and opiates to relieve the pain.

When the inflammatory process has reached the suppurative stage, the sooner the evacuation of the pus is secured the better. Free incisions down to the bone, made from within the mouth, ought to be insisted upon as soon as pus is discovered, with the view of giving exit to the discharges and relieving the periosteal tension. Such a procedure affords great relief to the patient.

When the abscess points toward the external surfaces beneath the jaw, as frequently happens, or burrows down the neck, external incisions become necessary, as evacuation and drainage are thus better accomplished. Irrigation with antiseptic solutions should be frequently employed, and if necrosis results the dead bone should not be removed until separation has taken place. The constitutional treatment must be sustaining throughout, viz: good foods, tonics,—iron, cod-liver oil, etc.,—and stimulants if indicated.

Mercurial Periostitis.—This form of the disease is due to the constitutional impression of mercury, and has been so common in the past as to come under the notice of almost every middle-aged practitioner of medicine.

The effects which are produced by mercury upon the general system, and locally in the mouth, depend upon the quantity administered and the susceptibility of the individual to the action of the drug. Children from five to ten years of age are peculiarly susceptible. There is, however, a very great variety in the susceptibility of different individuals; in one, an ordinary dose of blue pill or of calomel will produce severe salivation and swollen tongue, while another seems to be almost proof against its action, even in large and repeated doses.

Garretson mentions a case under his care, a child seven years of age, in whom necrosis of the left half of the body of the lower jaw was produced by the administration of three grains of calomel.

Symptoms.—The symptoms of the disease, as presented in stages, are a coppery or metallic taste in the mouth, speedily followed by an increase in the flow of saliva and swelling of the tongue. The tongue in its swollen condition presses upon the teeth, causing indentations upon its edges. The gums next become swollen and puffy, commencing generally in the neighborhood of the inferior incisor teeth. A congested condition of the oral mucous membrane also appears, extending over the entire mouth, and sometimes associated with a sense of dryness or of burning. Tumefaction of the gums now becomes general;

they assume a livid color, and bleed easily. The salivary glands are swollen and tender, and the secretion of saliva is greatly augmented in amount; so much so, that the patient is obliged to constantly expectorate. In some cases the secretion is so profuse that it runs from the mouth. The quantity in severe cases may reach several pints per day. The teeth frequently become loose, and can be picked out with the fingers; the breath and secretions have a very disagreeable fetid odor. The disease, if unchecked, may be complicated with necrosis of more or less extensive portions of the alveolar process, or of the entire jaw, or with sloughing of the gums and cheeks.

Treatment.—The treatment of mercurial periostitis consists of eliminating the mercury from the system by the aid of the iodid of potassium in doses of from five to ten grains in solution, after meals, which forms soluble compounds with the mercury retained in the economy; or by the administration of chlorate of potassium in ten- to fifteen-grain doses every few hours, for its oxidizing effects in contaminated conditions of the blood. Saline cathartics are also useful in promoting elimination. The general health should be built up by change of air, generous diet, and tonics. The local conditions are to be corrected by scarifying the gums and painting them with the tinctures of aconite and iodin, equal parts, or tincture of iodin and glycerin, equal parts. Solutions of chlorate of potassium, one drachm to an ounce of water, used as a mouth-wash, will be found very efficacious in relieving the local inflammatory conditions, and can be used *ad libitum*. Permanganate of potassium, two to ten grains in an ounce of water, or cinnamon water, are very useful in correcting the fetid odor of the breath. Solutions of boric acid and the Thiersch solution may be used freely as antiseptic washes.

Chronic Periostitis of the Jaws.—This form of the disease is usually the result of syphilis. It is generally painless, causes but little swelling of the soft tissues, and manifests itself in the formation of nodes, as in syphilitic periostitis of other parts of the body. The palate and alveolar borders are particularly liable to those enlargements, which are due to exudations between the periosteum and the bone, and unless constitutional treatment is instituted for their removal, necrosis will sooner or later supervene. Fortunately, the disease will usually yield to large doses of the iodid of potassium, twenty grains three times per day, in the compound syrup of sarsaparilla, rapidly increased to drachm doses. By this treatment in a few weeks the swelling will disappear, and the periosteum be restored to its normal condition. Mercury is generally considered to be inadmissible in this form or stage of syphilitic disease.

REVIEW.

CHAPTER XXVI.

What is the definition of periostitis?

Describe the periosteum.

Of how many layers is it composed, and how are they designated?

What are the functions of the periosteum?

Into what forms is periostitis divided?

Describe acute periostitis.

Upon what conditions does the character of the inflammation depend?

How may simple acute periostitis terminate?

Which is the most common form of the disease?

What is the difference in the character of the disease when affecting the upper and lower jaws?

How does the disease extend?

What are the exciting causes?

What are the predisposing causes?

Describe the symptoms of the disease.

What condition of the jaws is a frequent accompaniment of the disease?

What is the difference in the symptoms of mild cases?

On this account what may be the result?

What is the general tendency of the disease?

How does suppuration produce necrosis?

At what age does acute diffuse periostitis most frequently occur?

In what class of children is it most often seen?

What are the differences in the symptoms between *simple* and *diffuse* periostitis?

What are the exciting causes?

What is the treatment in the early stages of the disease?

What is the treatment in the suppurative stage?

What should be the constitutional treatment?

What do you understand by the term *mercurial* periostitis?

Upon what conditions do the effects of mercury upon the general system and in the mouth depend?

What are the symptoms of mercurial periostitis?

What are the effects of mercurial poisoning upon the salivary glands and the flow of saliva?

What are the complications of the disease?

Of what does the constitutional treatment consist?

Describe the local treatment.

What is generally the cause of chronic periostitis?

How is it made manifest in the jaws?

In what locations are these manifestations found?

What is the constitutional treatment of this form of the disease?

CHAPTER XXVII.

NECROSIS OF THE JAWS.

Definition.—Necrosis (from the Greek νεκρός, dead). Death of the bone *en masse*.

Necrosis is a condition, not a disease. It is rather a symptom, representing a local condition, which may be brought about by various causes.

Necrosis of the Jaws.—Necrosis is much more common in the lower than in the upper jaw. This is no doubt due mainly to the greater vascularity of the tissues of the upper jaw, and the free anastomosis of its vessels, which augments its recuperative power; and, in comparison with the lower jaw, its better protected position, which renders it less liable to injury.

The superior maxillæ are supplied with numerous branches of the internal maxillary arteries, which freely inosculate with one another and with those from the opposite side, while the inferior maxilla is supplied with only one small branch upon each side, and these do not so fully anastomose one with the other.

According to Stanley ("Diseases of the Bones"), the lower jaw stands fifth among the bones of the skeleton in its liability to necrosis, while the upper jaw occupies the twelfth place. Taking this statement as our authority, the lower jaw is therefore nearly two and a half times more liable to necrosis than the upper.

Necrosis of the jaws may be complete or partial. It is *complete* when the entire thickness of the bone is involved, and *partial* when it is confined to the alveolar process.

Causes.—The causes which produce necrosis are identical with those of periostitis, viz: traumatisms, the eruptive fevers, scorbutus, syphilis, inflammatory conditions of the periosteum and periodonteum, mercurial and phosphorus poisoning, local arsenical poisoning, gangræna oris, and other ulcerative affections of the soft tissues of the mouth, which establish inflammatory conditions, causing death of the bone by strangulation of its blood-vessels. Necrosis in this respect resembles gangrene of the soft tissues.

Necrosis is therefore the result of unchecked periostitis, or suppurative inflammation, induced by any of these conditions, and the

separation of the periosteum from the bone by the accumulation of the pus.

In the upper jaw, which is composed of thin plates of bone, covered upon both sides with periosteum, and the whole exceedingly vascular; and in the long bones, where there is medullary tissue abundantly supplied with blood-vessels, the resistance and recuperative powers are much greater than in the lower jaw, and it frequently occurs that after an extensive subperiosteal abscess has formed, and the bone has been denuded of its periosteum, recovery has taken place without death of the bone; but when the same conditions are associated with the lower jaw, it rarely happens that recovery takes place without more or less extensive necrosis, while death of the bone is not infrequently accomplished in a few hours after the formation of pus between the periosteum and the bone. The necrotic process does not, however, necessarily extend to the entire thickness of the bone, but may be confined to the outer surface only,—which it usually attacks first,—if proper treatment is instituted to remove the accumulated pus and control the inflammation. In such cases the internal plate of the alveolar process remains intact, and gives support to the teeth, which otherwise would loosen and fall out, as generally occurs when the entire thickness of the jaws is necrosed. If, however, the disease involves the entire thickness of the bone, it may not extend beyond the alveolar process, the base of the jaw being left intact.

Separation finally takes place between the living and the necrosed portions of the bone. The dead portion is termed the *sequestrum*. It frequently happens in the lower jaw, but rarely in the upper, that new bone is formed from the periosteum over and around the sequestrum. This shell of new bone is termed the *involucrum*. The new shell of bone often has openings in it, which are termed *cloacæ*.

In the more serious cases affecting the inferior maxilla, large sections of the entire thickness of the jaw, or even the entire jaw, from the articulation of one side to that of the other, may become necrosed. Similar conditions may prevail in the upper jaw, but it is exceedingly rare that the necrosis is so extensive as in the lower jaw.

Several cases of an interesting character have come under the observation of the writer, which will serve to illustrate the above statements,—one in which the trouble followed tropical fever in a man thirty years of age, and which began upon the right side of the jaw, in the region of the first molar tooth. Soon afterward it attacked the opposite side, and extended forward to the median line on both sides, and backward to the angles. The necrosis was principally confined to the external plate of the alveolar process, which came away in sections and spiculæ to the number of over fifty, and involved the loss of three anterior teeth. Another was the result of suppurative inflam-

mation of the left lower third molar in a man fifty years of age, suffering from anemia. The necrosis extended rapidly until the entire alveolar process was involved from the left angle to the right second molar. All of the teeth, and the entire alveolar process, were lost between these points. A third was the result of scarlet fever in a boy five years of age, in whom the body of the jaw, from the right lower first deciduous molar backward, and the entire ramus, including the condyle and the coronoid process, were lost. A fourth (Fig. 106) was the result of a suppurative inflammation of the left inferior third molar in a woman thirty-five years of age, suffering from general debility, the

FIG. 106.



NECROSIS OF LOWER JAW AND SLOUGHING OF SOFT TISSUES OF CHIN AND NECK.

result of frequent gestations, with short intervals, and overwork. The necrosis extended very rapidly until it involved the entire jaw, which was finally lost from the articulation of the left side to the upper third of the ascending ramus of the right side. This case terminated fatally from exhaustion complicated with la grippe, sixteen days after the removal of the necrosed maxillary bone. The illustration shows extensive loss of the soft tissues of the chin and neck by sloughing, which had occurred before the patient was admitted to the clinic at Mercy Hospital. Another was in a boy of seven years of age, the result of a severe attack of measles, in which the alveolar process and body of the right superior maxilla from the canine fossa backward to the tuberosity, and upward to the orbital plate, was lost, the palate process and

the orbital plate remaining intact. Others of an equally grave nature might be mentioned, but these are sufficient to illustrate the subject.

Symptoms.—The early symptoms of necrosis are usually those of periostitis, which have already been mentioned under that head. After necrosis has been established the pus finds an outlet by the side of the loosened teeth, or burrows through the gums. Later the gums become loosened from the bone, and the pus oozes from between them. This is the usual course of necrosis in the upper jaw. When associated with the lower jaw it often burrows through the tissues covering the body of the bone, and points upon the under side of the jaw, or follows the inter-muscular connective tissue of the neck downward, pointing at various locations, even as low down as the clavicle or mammæ. The discharges have the peculiar fetid odor which is characteristic of dead bone, and in those cases in which the pus is discharged into the mouth in considerable quantity, nausea and vomiting may ensue, digestion soon becomes deranged by reason of the entrance into the stomach of the foul discharges, general emaciation frequently takes place, and septicemia is not an uncommon sequence.

Necrosis of the jaws has been known to extend to adjacent bones of the face and head, and so involve the brain, causing a fatal termination.

Treatment.—The treafment of necrosis in general should be that of non-interference, except the opening of the subperiosteal abscesses, and disinfection, until such time as separation of the sequestrum has taken place. Nothing is to be gained by surgical operations for the removal of necrosed portions of bone before separation occurs, as it is usually impossible to previously determine to what extent the necrosis will involve the bone. Furthermore, such attempts at removing the dead bone would be worse than useless, as they would be likely to aggravate the inflammatory symptoms, and make a secondary operation necessary. The treatment must therefore be one of expectation and conservatism.

The establishment of free openings for the discharge of the accumulated pus, and frequent irrigation of the suppurating surfaces with antiseptic solutions, is about all that can be done until the sequestrum is loosened. The fetid odor of the breath and of the discharges may be corrected by the free use of solutions of the permanganate of potassium and cinnamon water. The peroxid of hydrogen and the medicinal pyrozone are also useful in the same direction, but they should not be used in those cases where there are not free openings for the escape of the liberated gas, since it may occur that the pressure of the gas will be so great as to dissect a considerable area of periosteum from the bone, beyond the original lesion, and by that much increase the extent of the necrosis. One such case occurred in the practice of the writer,

with the result of making him thereafter extremely cautious in the use of these remedies. The constitutional treatment should be supporting throughout,—good food, milk, and concentrated liquid foods are best in these cases, on account of the inability of the patient to use the jaws for mastication. Tonics are also indicated,—iron, quinin, malt extracts, cod-liver oil, etc., and change of air.

REVIEW.

CHAPTER XXVII.

What is necrosis?

In which of the jaws is necrosis most common?

What reasons are given for this?

What is the relative liability to necrosis in the lower and upper jaws as compared with the other bones of the skeleton?

What is the difference in liability to necrosis between the upper and lower jaws?

How is necrosis divided?

When is necrosis termed complete?

When is necrosis termed incomplete?

What are the causes of necrosis?

What is the sequestrum?

What is the involucrum?

What are the cloacæ?

In which jaw is the necrotic process usually most extensive?

Describe the symptoms of necrosis.

What are the complications of necrosis?

Does it ever prove fatal, and under what conditions?

What general lines should be followed in the local treatment?

At what time is it proper to attempt to remove the sequestrum?

Why is it better to wait for separation to take place than to attempt its removal before?

What should be the treatment before separation of the sequestrum has taken place?

How may the fetid odor of the breath and discharges be corrected?

Under what circumstances would it not be wise to inject peroxid of hydrogen and pyrozone?

What should constitute the systemic or constitutional treatment?

CHAPTER XXVIII.

NECROSIS OF THE JAWS (Continued).

Exanthematous Necrosis.—The eruptive fevers are productive of a large percentage of the cases of necrosis of the jaws in children. The age limit in which it is most likely to occur is between the third and eighth years. It is exceedingly rare that a case of necrosis is developed as one of the sequelæ of the exanthems outside of this limit.

It is interesting to note the correspondence of the age limit with the period of the greatest activity in the developmental processes of the jaws and the teeth. The teeth are dermal appendages, developed from the layers of the mucous membrane; consequently these tissues are more or less susceptible during their development to the same influences which affect other portions of the dermal skeleton. The peculiar toxic conditions of the system which exist during attacks of scarlet fever, measles, and smallpox would seem to be the exciting cause of necrosis, while the exceeding activity of the vascular system of the parts would predispose to congestion and inflammation.

Necrosis is more liable to follow scarlet fever than measles or smallpox. Salter places the ratio as between scarlet fever and measles at about three of the former to one of the latter; between scarlet fever and smallpox, as about four to one; and between measles and smallpox, six to four.

The severity of the attack seems to bear no relation to the liability to cause necrosis. Mild cases develop necrosis as frequently as severe ones, and *vice versa*. Occasionally the necrosis is associated with other secondary symptoms, but in the majority of instances this is the only one, and it would seem that it had a predilection for otherwise healthy children.

Symptoms.—This form of necrosis first shows itself a few weeks after the attack of the fever; the approach of the affection is marked by aching and soreness of the teeth, swelling, tenderness, and turgescence of the gums, and fetid breath, quickly followed by suppuration and all the symptoms of necrosis.

Treatment.—The treatment is substantially the same as that indicated for necrosis of the jaws in general. The condition of the general health, however, should be carefully watched, and every effort

made to improve it, by good foods, tonics, fresh air, and stimulants when required.

Mercurial Necrosis.—Mercurial periostitis and necrosis of the jaws used to be quite common forty or fifty years ago, especially in the southern portions of our country, when mercury was used so extensively and in such large doses. Happily, in these days the more intelligent use of the drug has made such cases much less common.

The loss of osseous tissue from mercurial poisoning varies very greatly in its extent. It may be confined to the alveolar process surrounding one or two teeth, or extend throughout the entire alveolar process of one or both jaws, or an entire jaw may be lost. Sometimes the constitutional effects may be so overwhelming as to endanger the life of the sufferer, occasionally proving fatal.

When the death of the bone is extensive there is often associated with it, as a complication, sloughing of the gums and of the cheeks, causing perforation of the buccal walls and consequent disfigurement of the face. In other cases the sloughing is confined to the mucous membrane, or it may invade the muscular tissue of the cheek or lips without perforating the integument. Under these circumstances large masses of cicatricial tissue are formed by the process of healing, which upon contraction may give rise to permanent closure of the jaws.

The writer has seen but few cases of necrosis of the jaws that could be fairly attributed to mercurial poisoning, and two of these proved fatal. The first was a railroad engineer, forty years of age. Large doses of mercury were administered by his physician during an attack of malarial fever, with the result of producing severe ptyalism, with a profuse flow of saliva, three to four pints per day, and extensive stomatitis, periostitis, and necrosis of both the upper and lower jaws. All of the teeth in both jaws became so loose that most of them were removed with the fingers; the rest were extracted with the forceps. Suppuration was very extensive in both jaws, pus discharging into the mouth in large quantities, and through several sinuses under the lower jaw. The patient rapidly failed, and died before separation of the sequestra had taken place. This was an exceptionally severe case, and fortunately an uncommon one.

The second fatal case was quite recently under observation. The patient was an Italian woman about thirty years of age, who was suffering from mercurial periostitis and necrosis of the superior and inferior maxillæ, and gave a history of having taken only fifteen grains of calomel in three-grain doses "at bedtime." The necrosis was accompanied with extensive sloughing of the gums of the inferior maxilla and the soft tissues covering the hard palate, swollen tongue, fetid breath, excessive salivary secretion, loosened teeth, and an uncontrollable diarrhea. Death was from exhaustion.

Treatment.—The treatment has been already described under the head of mercurial periostitis. The rules governing the removal of the sequestra are the same as those for necrosis in general.

Arsenical Necrosis.—Necrosis of the alveolar process is not an uncommon result of the careless application of arsenious acid for the devitalization of the tooth-pulp; or from accidental causes, like the penetration of the drug beyond the apical foramen; or through the minute canals which sometimes exist in the sides of the roots of the teeth, and which communicate with the root-canal and the pericementum. Accidents more often occur in the treatment of the teeth of children, for the deciduous teeth have large apical foramina; consequently, the use of arsenic for pulp-devitalization in these cases is dangerous in the extreme. The same is true of all the permanent teeth during the development of their roots, which are not completed until some considerable time after the eruption of the crown. The first permanent molars most often require the devitalization of the pulp as a result of caries, but as they are not fully developed until about the end of the tenth year, it would be dangerous to apply arsenic for this purpose before that time. This rule should apply to all the teeth of young people during the development of these organs.

Arsenious acid is a powerful escharotic, but at the same time a valuable remedy when carefully used. The faults in applying arsenic to the tooth-pulp for the purpose of devitalizing it, lie in two directions:

First, too large a quantity is generally used; and,

Second, it is not properly sealed in the cavity. The one-hundredth of a grain of arsenic is just as effective in destroying a pulp as a larger quantity would be. This amount may be safely left in the adult tooth from two to three days, when properly sealed in the cavity; in fact, it will require about this length of time to effectually destroy the vitality. The only safe method of sealing a cavity in which arsenic has been placed is with the oxyphosphate cement. Gutta-percha, the temporary stoppings, and cotton and sandarac varnish cannot hermetically seal a cavity, and anything less than this is dangerous.

In arsenical necrosis the disease rarely, if ever, extends beyond a fragment of the alveolar process involving one or two teeth, with the possible loss of the teeth involved in the death of this portion of the bone. It oftener occurs, however, that the necrosis does not extend beyond a portion of the alveolar process upon that side of the tooth on which the arsenic came in contact with the soft tissue. These accidents occur more frequently in applying the drug to teeth having cavities upon their approximal and buccal surfaces near the gingival borders; consequently, the alveolar septi and the outer plate of the alveolar process are the most common locations of necrosis from this

cause. The soft tissue with which the drug comes in contact is always devitalized and sloughs away.

Treatment.—The treatment of necrosis from the effects of arsenious acid does not materially differ from that of necrosis from other causes. The application of the hydrated oxid of iron—sesquioxid—to the injured tissues is advocated very strongly by some authorities. To the writer the local application seems of little value, except to serve as an antidote for that which still remains in the tissues of the tooth.

Phosphorus Necrosis.—Maxillary necrosis, the result of poisoning from the fumes of phosphorus, was at one time a very common affection among the operatives in match factories. It was so terrible in its results that it became the subject several times of legislative inquiry in England and other European countries, with the desire of discovering some means of preventing or mitigating its ravages. Scientific investigation was also instituted by medical men as to the cause of the disease, and to discover, if possible, some means of prevention, with the result of establishing beyond a reasonable doubt these facts: First, That the affection was caused by the fumes of phosphorus, but that it must gain access to the periosteum of the bone in order to establish the disease. Second, That in every case of necrosis of the jaws from this cause, the disease originated in connection with a carious tooth, and, on the other hand, operatives with sound teeth were entirely exempt from it. This seemed to indicate that the phosphorus gained access to the periosteum through the tooth-pulp, and thus established the affection. The disease appeared, therefore, to be one of local poisoning, and the fact that the other bones of the body escaped the disease added strength to the argument.

Opposed to this view are Langenbeck and others, who maintain that the effects of the poison were produced through the system, the same as with mercury. There seems to be no fact in pathology better established than that the disease is the result of local poisoning, produced through some break in the continuity of the structures of the mouth, which permits the poisonous fumes to come in contact with the periosteum.

Precautionary measures were therefore adopted by the manufacturers, as a result of the scientific investigation, for the protection of their employes, which have proved so efficacious that now the disease is rarely seen. These means consisted of thorough ventilation of the dipping rooms, cleanliness of the factories, with a proper care and treatment of the teeth of the operators, and teaching them habits of personal cleanliness. The disease has occasionally been contracted by children from the chewing of the dipped ends of matches.

Symptoms.—Phosphorus necrosis is very insidious in its first approach, the symptoms being so mild as scarcely to be noticed, usually

beginning in a supposed toothache. As the disease progresses, the symptoms become marked and aggravated; the pain in the jaw is excruciating in the extreme, the swelling very great, often extending over the entire side of the face and head. Abscesses form and open upon the external surfaces, and also into the mouth, forming sinuses through which the dead bone can be felt with a probe. The opening of the abscesses usually affords great relief; the pus is very offensive, and usually profuse. The health of the patient often deteriorates very rapidly from inability to take food, and from the disturbance of the stomach induced by the unavoidable swallowing of considerable quantities of the fetid discharges. When the necrosis is extensive, the constitutional disturbance is correspondingly great. Death frequently occurs from exhaustion. A peculiar and characteristic feature of phosphorus necrosis is the pumice-like deposit upon the sequestrum.

Treatment.—The treatment consists of sustaining the vital powers of the patient by the administration of concentrated liquid food, tonics, and stimulants. The local treatment is that of necrosis in general.

Syphilitic Necrosis.—The manifestations of syphilis in the jaws are generally associated with the tertiary stage of the disease, the earlier lesions being rarely met with in this region. The upper jaw is much oftener affected than the lower. When the disease attacks the lower maxilla, it is usually confined to the alveolar process, though it sometimes extends to the body of the bone. In the upper maxilla it attacks the alveolar and palatine processes and the palate bones most frequently; its most common site is the central part of the dome of the hard palate (Fig. 107), but it is not always confined to these locations. The writer recently saw a case in an old soldier in which the palate bones, the bones of the nose, and nearly the entire upper jaw, had been destroyed by the disease; the only portions of the jaw that remained were the orbital plate and malar processes, and a portion of the outer walls of the antra. The soft palate was intact. The opening into the nose was bounded posteriorly by the soft palate, laterally and anteriorly by the cheeks and lips only, except that portion of the outer wall of the antra just referred to. The opening measured antero-posteriorly one and three-fourths inch, and from side to side one and one-half inch. The destruction of the osseous tissue of the nose and jaws is rarely so appalling as in this case.

The tertiary symptoms of syphilis frequently present themselves long after the primary and secondary symptoms have disappeared, while the natural reticence of the patient to admit early indiscretion often makes the diagnosis extremely difficult, so that for lack of positive evidence many cases are excluded from the category which no doubt properly belong there.

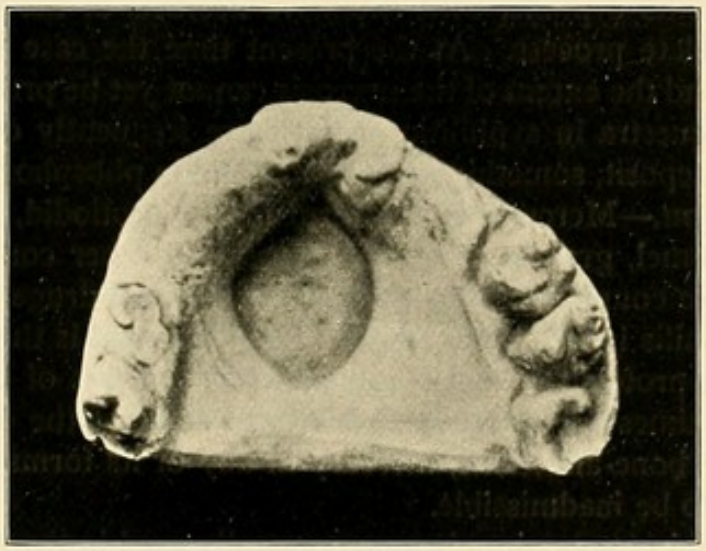
The syphilitic virus has a predilection for the compact tissue of

the bone, and most often attacks those portions of the bone which have soft, thin coverings, like the bones of the skull, the palate process, the palate bones, and the alveolar processes. A marked exception to this is the necrosis of the spongy bones of the nose, following syphilitic ulceration of the nasal mucous membrane.

Symptoms.—Syphilitic periostitis of a marked type, or ulceration, always precedes death of the bone in the region of the mouth and nose. This form of the disease is, however, much less rapid in its work of destruction than that form caused by the toxic influence of the eruptive fevers.

In syphilitic necrosis, on account of the slower progress or chronic condition of the disease, and frequent exacerbations in the inflammatory process, death of the bone frequently occurs in such a way as to

FIG. 107.



SYPHILITIC PERFORATION OF THE HARD OR BONY PALATE.

form numerous sequestra. Surgical interference before active inflammation has entirely subsided is often responsible for a renewal of the inflammatory symptoms, and extension of the necrotic process.

It has been the fortune of the writer in his hospital service to encounter a goodly number of cases of syphilitic necrosis of the jaws, the great majority of which have been associated with the vault of the mouth and the alveolar process of the upper jaw. Among these might be mentioned, by way of illustration, the case of a young lawyer, thirty years of age, who had contracted syphilis seven years before, and for whom was removed a sequestrum of bone involving the median half of the alveoli of the central incisor teeth, and the floor of the nasal fossa, to the extent of about half an inch in diameter, leaving an opening under the lip into the nasal fossa. This opening was successfully closed, and the teeth became reattached by the aid of sponge-grafts.

This operation was made eight years ago, and the teeth are still firm and, to all appearances, in a normal condition.

Another case, a man forty-five years of age, contracted syphilis at the age of twenty, had been married sixteen years, and had three healthy children. Necrosis of the right superior maxilla developed three months before his first visit. A sequestrum of bone was later removed for him, involving the alveolar process from the lateral incisor to the tuberosity, including the entire floor of the antrum.

Another, a young man twenty-six years old, had contracted the disease about five years before. His present trouble had developed about four months previous to his admission to the hospital. In this case there was extensive necrosis of the palate process and palate bones. Several sequestra were removed at various times, which left an opening in the hard palate the size of a silver half-dollar.

A very recent case is that of a little girl, nine years old, suffering from congenital syphilis, with extensive necrosis of the bones of the nose and palate process. At the present time the case is still under treatment, and the extent of the necrosis cannot yet be predicted.

The sequestra in syphilitic necrosis are frequently coated with a gray-black deposit, something like that found in phosphorus necrosis.

Treatment.—Mercury in the form of the protiodid, biniodid, bichlorid, calomel, gray powder, blue mass, and other combinations of the metal are counted among the most efficacious drugs in the treatment of syphilis in its earlier stages. The protiodid is the most popular with the profession, and is administered in doses of one-fifth of a grain three times per day. In the tertiary stage of the disease, especially in the bone-affections, mercury in any of its forms is generally considered to be inadmissible.

The iodid of potassium is more generally used in the tertiary stage of the disease than any other drug. It is administered in doses of from three grains to twenty, dissolved in distilled water, milk, cinnamon water, syrup of sarsaparilla, or any of the various syrups used as vehicles by the druggists. The iodid of potassium gives the happiest results in the treatment of syphilitic bone-diseases. The larger doses of the drug, viz: two drachms to one-half ounce, in twenty-four hours, are sometimes administered, but are not generally indicated in this form of the affection.

In those cases in which the necrosis is very extensive and the vital powers are much depressed, it is better to withdraw the drug altogether, and substitute tonics.

Especial attention should be given in all cases to the general building up of the vital forces. The diet should be plentiful and nutritious. Alcohol should be administered in moderation, if at all, for its tonic, not for its stimulating effects. Tobacco should be excluded in all its

forms. The body should be kept scrupulously clean by daily sponge baths with tepid water, and covered with warm clothing.

The mind and body should be kept employed with the customary duties, care being taken not to overtax the mental and physical powers.

In the severer cases, change of climate, such as a sea voyage or a few months' residence at the seashore in the summer season, or a trip South in the cold months, is to be advised.

The tonics which may be administered with benefit are iron, cod-liver oil, quinin; the bitter tonics, coca wine, etc.

Reproduction of Bone.—The bones in general have a very marked power of regeneration. The seat of this power lies in great measure in the periosteum, and in a less degree in the medullary tissue. In many cases the periosteum alone performs the office of reproducing the bone.

The knowledge of these facts has brought about the most conservative treatment upon the part of the surgeon in all operations upon the bones, that no more of the periosteum be sacrificed than is necessary to insure the success of the treatment for which the operative procedure was instituted.

Malgaigne was the first to recognize the importance of conserving the periosteum for the purpose of establishing the process of regeneration of bone.

Ollier demonstrated the possibility of transplanting the periosteum for the purpose of reproducing bone in locations where the original bone and periosteum had been lost.

Reproduction of osseous tissue is a frequent occurrence following fractures, gunshot wounds, and amputations; more rarely after trephining and resections, after extirpation of bones and following necrosis. In all of these conditions there is a more or less incomplete or a complete reproduction of the bone. Regeneration of entire bones is rare. Wagner mentions the case of a woman in whom an entire new clavicle was formed following necrosis. Several cases of reproduction of nearly the entire lower jaw have been recorded, and numerous cases of regeneration of considerable portions.

The writer has reported two cases of regeneration of the ascending ramus with perfect mobility of the temporo-maxillary joint. The first was in a girl sixteen years of age, suffering from dentigerous cyst of the lower jaw, the result of an inverted third molar tooth, and necrosis of the ramus, including the head of the condyle, which was removed (Fig. 108). One year afterward the restoration was so complete in all respects as to make it seem impossible that so extensive a loss of tissue had occurred. The other was in a boy five years of age—already referred to—who lost the ramus and body of the jaw behind the second deciduous molar from scarlet fever. In this case also there

was a complete restoration of the lost bone and perfect mobility of the joint.

The process of regeneration or repair after loss of the jaw from necrosis differs greatly in the upper and the lower maxillæ. In the upper jaw it is very rarely that true bone is reproduced, but instead there is developed a hard, fibrous tissue, which fills the gap and serves the purpose of bone. This often occurs in children who have suffered from exanthematous necrosis, but rarely in adults, except by the aid of sponge-grafting.

In the lower jaw regeneration of bone after necrosis is the general result, but it is claimed by some authors that resorption of the new bone sometimes takes place—occasionally after a considerable period

FIG. 108.



CONDYLE OF INFERIOR MAXILLA WITH MISPLACED AND INVERTED THIRD MOLARS.

—in those cases where the entire body of the jaw has been reproduced, so that finally there is scarcely enough bone left to keep out the lower lip and the chin, and this feature of the face is completely obliterated.

A case of this character, a young lady, is now under the care of the writer, for whom he has attempted the restoration of the contour of the face by a plastic operation and the construction of an appliance to represent the body of the jaw, upon which are mounted artificial teeth. This appliance is being worn with considerable comfort; the effort has greatly improved the contour of the lower part of the face, and bids fair to be a tolerable success.

The cause of this resorption does not seem to be understood. Salter suggests that the resorption of the new jaw might possibly be prevented by inserting a plate of artificial teeth, and thus supply it with a definite function.

REVIEW.

CHAPTER XXVIII.

What forms of fever are productive of necrosis of the jaws?

What is the age limit during which it is most likely to occur from this cause?

What are the conditions of development of the jaws and teeth at this time?

What are the causes of exanthematous necrosis?

Which of the exanthems is most liable to cause necrosis?

What is the ratio between scarlet fever and measles?

What is the ratio between scarlet fever and smallpox?

What is the ratio between measles and smallpox?

What relationship does the severity of exanthems bear to the liability to cause necrosis?

What are the symptoms of exanthematous necrosis?

Does the treatment differ from that of necrosis in general?

What is the cause of mercurial necrosis?

Is necrosis from mercurial poisoning common?

What are the local complications of mercurial necrosis?

What is the effect upon the cheek and mucous membrane of the sloughing of these tissues?

How may the contraction of cicatricial tissue affect the movement of the jaws?

What is the cause of arsenical necrosis?

In what class of teeth is it unsafe to apply arsenic for pulp-devitalization?

What are the usual faults in applying arsenic for this purpose?

Is arsenical necrosis ever as extensive as in other forms?

To what portion of the jaw is it usually confined?

What is the effect of the arsenic upon the soft tissues?

Describe the treatment.

What is the cause of phosphorus necrosis?

What class of individuals are liable to be affected by it?

Are the effects produced through the constitution or by local poisoning?

How does the poison gain access to the tissues of the jaw?

What means are now adopted to prevent it?

What is Langenbeck's view upon this point?

Describe the local symptoms of phosphorus necrosis.

Describe the constitutional symptoms.

What is frequently a cause of death in these cases?

What is the peculiar character of the deposit found on the sequestrum?

Describe the treatment.

In which stage of syphilis is maxillary necrosis most common?

Are the earlier lesions often manifested in this region?

Which jaw is most often affected by syphilitic necrosis?

Which portions of the upper jaw are generally affected?

Which portions of the lower jaw?

Is it always confined to these portions?

What conditions sometimes render the diagnosis difficult?

Which portion of the bone-tissue is most frequently attacked, and in what bones?

Name a marked exception to this rule.

Does syphilitic necrosis progress as rapidly as other forms of necrosis? If not, why?

What is the result of these exacerbations?

Describe the constitutional treatment.

Is mercury admissible in this form of syphilitic disease?

How should the iodid of potassium be administered?

When the vital powers are greatly depressed, what should be the treatment?

Is tobacco admissible in such cases?

How should the severer cases be treated in regard to climate, food, etc.?

Under what circumstances may regeneration of bone take place?

From what tissue is it formed?

Can the periosteum be successfully transplanted?

Are bones ever completely regenerated?

In which jaw is regeneration of bone most likely to occur?

What is the general result after necrosis in the lower jaw?

What sometimes becomes of the new-formed bone?

What is Salter's suggestion in reference to preventing this resorption?

CHAPTER XXIX

STOMATITIS.

Definition.—Stomatitis (Gr. στομα, mouth, and ιτις, inflammation).

Stomatitis is an inflammation of the mucous membrane of the cavity of the mouth. All inflammatory conditions which involve the gums, the inner surface of the cheeks, the lips, and the tongue are included under the term stomatitis.

The affections which are thus included are with few exceptions confined to the period of infancy and childhood. Adults seldom suffer from these affections except as a manifestation of some other morbid condition.

A clinical study of the inflammatory affections of the mucous membrane of the mouth will reveal a close resemblance in certain features to the inflammatory affections as they appear in the skin; while in other points they will present features which are common to inflammatory conditions of the mucous membrane in general. It is frequently noticed that "in measles a spotty or macular eruption appears upon the oral mucous membrane, and in scarlatina a punctate or diffuse scarlet eruption," while "in smallpox, chicken-pox, herpes, pemphigus, and in foot-and-mouth disease,"—an infection from cattle,—"there are eruptions of vesicles and pustules, which pass through the same stages as those of the skin" (Ziegler).

Erysipelas of the face not infrequently presents an inflamed condition of the oral and nasal mucous membrane, while syphilis and scurvy are accompanied by characteristic mouth-affections.

Diphtheria is not always confined to the tonsils, pharynx, uvula, and velum palati, but may likewise involve the mouth. Certain drugs, also, such as mercury and iodin, and the mineral acids, often produce inflammatory conditions of the oral mucous membrane.

The forms of inflammation of the mouth which are most common are *stomatitis simplex, stomatitis catarrhalis, stomatitis aphthosa, stomatitis parasitica,* and *stomatitis ulcerosa.*

From the time of Hippocrates to the present day, it has been the custom of some authorities to class all forms of inflammation of the mouth that are characterized by white patches, as aphthæ; while others have included all those forms which present ulcerated patches not

specific, and the more serious phagedenic conditions, as different degrees of the same affection.

At the present time the distinctions are more sharply drawn, and the classification based upon the etiology and pathology of these affections. Stomatitis with white patches is now divided into two distinct forms: *stomatitis aphthosa* and *stomatitis parasitica*, the former being due to a follicular inflammation with exudation or false membrane, and the latter to the action of a specific fungus which grows into the squamous layer of the mucous membrane. These, to the unaided eye, are readily mistaken one for the other, the only observable difference being the smaller size of the patches in stomatitis parasitica, and the tendency of the patches in stomatitis aphthosa to spread, and in some instances to become confluent. The microscope and bacteriologic cultures are necessary to arrive at a positive diagnosis.

Stomatitis Simplex.—This form of the disease is the mildest of the inflammatory affections of the mouth, and is generally expressed in a "more or less intense redness of the surface" of the mucous membrane of the cheeks, the lips, and the gums, which is due to localized hyperemia. It is usually found in infants and young children, and associated with some form of gastric or intestinal derangement. As a rule it is of short duration, and rapidly disappears, but occasionally it persists and gradually passes into a severe type of the disease known as stomatitis catarrhalis.

Symptoms.—The disease is sometimes designated as stomatitis erythema, from its resemblance to erythema of the skin. The affection is characterized by the appearance of rose-red, elevated patches upon the surface of the mucous membrane of the mouth, usually upon the cheeks and the lips, but occasionally also upon the gums, the palate, and the velum. Like erythema simplex of the skin, it appears suddenly, lasts for a few hours or two or three days, and as rapidly subsides. The bright color of the patches may be made to disappear by pressure upon them, but the color immediately returns upon removing the pressure. Heat and dryness of the mouth are often prominent symptoms, while in other cases the salivary glands are sometimes very active and the saliva dribbles from the mouth. (Day.)

There may be, and often is a rise in the body temperature, and other febrile symptoms accompanying the appearance of the erythema, but this in all probability is due to the constitutional disorder of which it is symptomatic. Restlessness, flatulency, and diarrhea are often prominent symptoms.

Treatment.—The treatment of this affection is to be directed to the constitutional condition upon which it is dependent, and of which it is but a local expression. The erythema usually subsides as soon as the constitutional disorder is corrected. This in many instances may

be accomplished by the administration of a mild cathartic and the regulation of the diet.

Stomatitis Catarrhalis.—This affection is often a symptomatic expression of a more grave constitutional malady, though it may be an entirely local disease induced by irritation from erupting teeth, or the taking into the mouth of irritating substances. The disease is often preceded by the simple erythematous condition just described, and it appears as a generally uniform diffuse inflammation, spreading over the cheeks, lips, and gums, and upon the hard palate as "streaks and patches." The papillæ of the tongue are most affected (Ziegler), many of them appearing as prominent tubercles. The mucous glands become swollen and prominent, so that they can be readily felt by passing the finger over the surface of the membrane. As the inflammation progresses, the mucous follicles become enlarged, "giving rise to grayish or grayish-red elevations of the surface surrounded by a reddened areola" (Ziegler). Occasionally tiny cysts are developed as a result of the plugging of the excretory ducts of the follicles with mucoid cells and the retention of the secretions. Sometimes cracks and fissures will appear upon the lips and at the angles of the mouth, with exudation and the formation of crusts.

The disease is most common among the children of the very poor, during the first year of infantile life, and is usually associated with bad food and unsanitary surroundings. It is rarely seen among the infants of the better classes of society, "unless the nipples of the nurse are sore or the milk is faulty" (Day), or in the bottle-fed children when the nurse allows the bottles and tubes to become foul.

Causes.—The causes are gastro-intestinal disorders, unwholesome food, uncleanliness, and the nervous irritation induced by the eruption of the deciduous teeth. It is occasionally "the result of taking cold, the inflammation being an extension of the inflammatory conditions of the mucous membrane of the respiratory tract" (Swift).

Symptoms.—The local symptoms are redness and capillary congestion of the mucous membrane of the mouth, accompanied by engorgement and swelling of the mucous follicles; swelling of the tongue, lips, cheeks, and gums; fetor of the breath; heat and dryness of the mouth, followed in some cases by an excessive secretion of the saliva and mucus, especially in children who are teething. In the latter cases the gums are often soft and spongy, and bleed under the slightest provocation. Vesicles and blebs are sometimes found upon the tongue, lips, and cheeks, which when ruptured leave minute ulcers "having a yellowish patch of lymph in the center, with a red margin" (Day). Fissures form at the angles of the mouth, and upon the lips, with exudation and the formation of crusts. Pain in this form of the disease is seldom very great, and the ulcerations are small and rarely troublesome.

The constitutional symptoms are febrile disturbances, diarrhea, thirst, loss of appetite, and sleeplessness. The prognosis is favorable.

Treatment.—The treatment consists in correcting the gastro-intestinal derangement by appropriate remedies, such as a dose of castor oil, or a powder of rhubarb combined with carbonate of soda (Day). The milk should be inspected, and if found unwholesome changed for that which is good. The breasts of the nurse should be examined, and if the nipples are found sore the child should be fed with the spoon or bottle. In the bottle-fed children the feeding utensils should be critically inspected, for many times the disease may be traced to unclean bottles, tubes, and nipples. The irritation to the gums from an advancing tooth is often relieved by lancing. The mouth should be cleansed after each feeding, by means of a piece of gauze or absorbent cotton wrapped about the finger or a probe, and dipped in a mild antiseptic solution, followed by washing the mouth with a chlorate of potassium solution, or "an astringent lotion in the form of a weak solution of the sulfate of zinc or of copper" (Swift). Lime-water should be added to the milk, and every effort made to keep the food and the surroundings of the child in a hygienic condition.

Stomatitis Aphthosa.—(Gr. ἀφθα, an eruption).

This disease—known as Canker sore-mouth—is considered by Ziegler to be a peculiar form of catarrhal stomatitis, for the reason that the aphthous patches occur upon the oral mucous membrane while in a catarrhal condition. Aphthous stomatitis may be considered as a severer form of catarrhal stomatitis. The disease is most common among sickly children during first dentition, but it is frequently seen during second dentition, and occasionally later in life in those who are debilitated from illness or debauchery, and "in women during menstruation, in pregnancy, and during the puerperal period." (Ziegler.) It is sometimes associated with pneumonia, gastric and intestinal catarrh, bronchitis, diphtheria, exanthematous diseases, ague, whooping cough, and tonsillitis.

Bohn has compared aphthous stomatitis with impetiginous eczema of the skin, which appears in the form of "an eruption consisting of pustules of the size of a pea, and drying into scabs without rupturing."

Aphthous stomatitis is characterized by the appearance upon the mucous membrane of small whitish or slightly yellowish patches from the size of a hemp-seed to that of a split pea (Ziegler). These patches may appear singly or in groups, and are most abundant upon the edges of the tongue and at the gingivo-buccal fold of the lips and the cheeks. Each patch is surrounded by a more or less inflamed zone; they are inclined to spread and coalesce, forming larger patches or streaks, though they never reach any considerable size. "The eruption occurs in successive crops, and may thus be kept up for weeks."

Bohn found the aphthous patches "to consist of a solid fibrinous exudate lying between the fibrous tissue and the epithelium." Sometimes the inflammation does not extend beyond the formation of the exudate; resorption then takes place, and the aphthous patches disappear by resolution. The more common termination is for the epithelial covering to be lost, thus exposing the fibrinous exudate, which is gradually separated from its base and thrown off by the regeneration of the epithelium which advances from the margins and extends beneath the exudate. As a result of the simultaneous reproduction of the epithelium with the extension of the exudate there is rarely the formation of what might be termed a true ulcerated surface. The exudate is easily removed and comes away in the form of a thin, dirty-yellow slough, leaving behind a livid base. Occasionally the inflamed zone which surrounds the aphthous patch becomes infected with the pus-microbes, and suppuration results.

Causes.—The causes of aphthous stomatitis are usually those which produce catarrhal stomatitis, viz: gastro-intestinal disorders, extension of inflammatory conditions of the respiratory tract to the mucous membrane of the mouth, unwholesome food, uncleanness of the feeding apparatus, and the nervous irritation induced by the eruption of the deciduous teeth; to which may be added mechanical irritation from the roughened surfaces and sharp edges of carious teeth, and the irritating effect of certain chemical substances which have been taken into the mouth. The acids of certain fruits, strawberries and tomatoes particularly, are not infrequently the cause of aphthous patches.

Symptoms.—The local symptoms are the presence upon the lingual, buccal, and labial mucous membrane of numerous small white or yellowish-white patches, slightly elevated above the surrounding membrane, and which are exceedingly sensitive to the touch, to hot or highly seasoned food, and to acids. The most prominent symptoms are the pain and the soreness of the mouth which prevents the child from taking food. The eruption passes away under appropriate treatment, but it is prone to frequent recurrence, especially in debilitated children and adults who are suffering from indigestion and other derangements of the digestive functions.

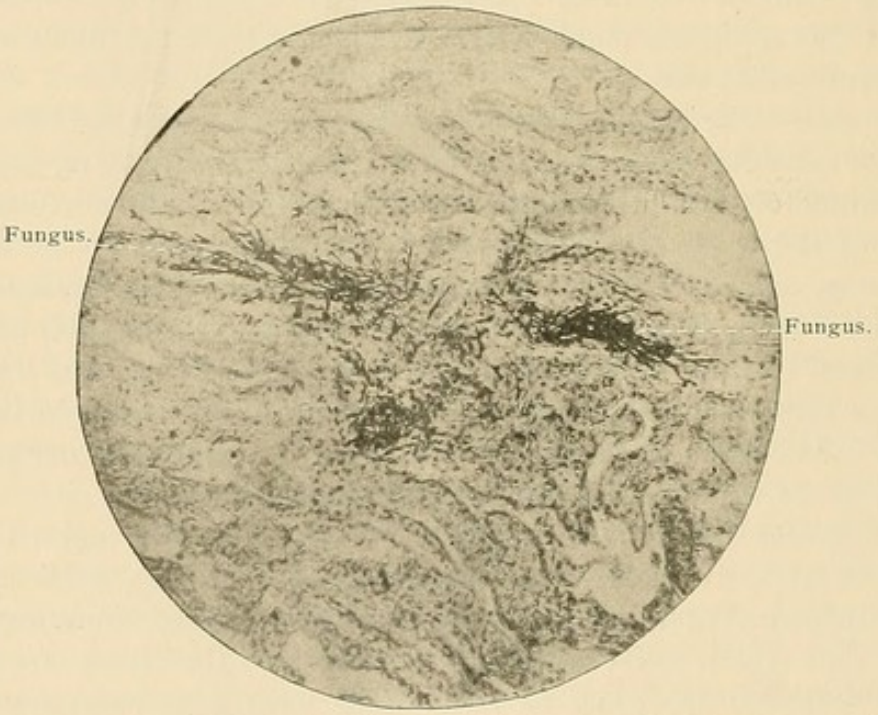
The constitutional symptoms rarely exceed a slight feverishness, loss of appetite, thirst, and irritability (Day). When the temperature rises more than one or two degrees above the normal, this is good evidence that the child is suffering from a more serious disorder of which the oral affection may be only a symptomatic expression.

Treatment.—There is no special treatment for this affection other than that already indicated in the treatment of catarrhal stomatitis, except that the inflamed parts of the mucous membrane may be lightly brushed over with a camel's-hair pencil which has been dipped in a

solution of boric acid in glycerol. In the more obstinate cases the patches may be touched with a solution of silver nitrate (gr. v to fl℥j of water).

Stomatitis Parasitica.—This disease is popularly known as *thrush* or *white mouth,* and is a parasitic or mycotic affection, generally found in the mouths of infants and little children. The parasite is a confervoid plant or fungus (Fig. 109), the thrush fungus or *oïdium albicans,* which grows upon and between the layers of the epithelium, but develops most rapidly upon the squamous type of this tissue. The disease is most commonly seen in the mouths of artificially-fed children, and is due to imperfect cleansing of the feeding apparatus. Being of

FIG. 109.



OÏDIUM ALBICANS (THRUSH FUNGUS) IN KIDNEY OF MOUSE AFTER SUBCUTANEOUS INOCULATION. × 1000.

mycotic origin, it is readily conveyed from the mouth of one child to another. It is most frequently seen in foundling and maternity hospitals. To prevent its spreading constant care should be exercised in the examination of the children's mouths, and under no circumstances should the promiscuous use of nursing bottles be allowed. When the disease occurs in adult life it is always associated with a debilitated condition of the system, and is considered as an unfavorable omen. The growth of the organism is favored by an abnormal acidity of the oral secretions, a debilitated condition of the system, and bad sanitary and hygienic surroundings.

Symptoms.—The disease is characterized by a dry, feverish mouth, and scanty salivary secretion. Small, white, elevated patches are found

upon the inside of the lips, cheeks, angles of the mouth, and sides of the tongue (Day).

These elevated patches, after two or three days, assume a curdy or soft cheesy appearance. This "thrush film" can be removed as a false membrane, leaving a denuded surface which bleeds easily, until it is again covered by another parasitic growth. The denuded or excoriated surfaces are exceedingly sensitive, and render nursing and swallowing very painful. These patches sometimes extend to the pharynx, tonsils, and hard palate, and may even develop in the œsophagus and air-passages.

The constitutional symptoms are disorders of the stomach and intestines, with vomiting and diarrhea. The excreta from the bowels are greenish in color, mixed with curdy masses of milk, and are often acrid, causing excoriation of the anus, buttock, perineum, and in the male sex of the scrotum. Elevation of temperature and acceleration of the pulse are not uncommon symptoms. The disease sometimes terminates fatally in debilitated children from exhaustion and inanition.

Treatment.—The treatment consists of clearing the alimentary tract with a dose of castor oil or calomel, and a proper regulation of the diet. Day recommends the use of chlorate of potash administered in small dose of a few grains, three times per diem, as he considers it a specific.

The local treatment consists of washing the mouth after each meal with some bland antiseptic solution, and the application of boric acid in glycerol or honey. In the more severe cases the patches may be lightly touched with a solution of silver nitrate, gr. v to water fl℥j. Occasionally it may be necessary when there is a tendency to ulceration at the bottom of the patches to touch them with the stick nitrate. Sir William Jenner recommended a solution of soda sulfite ℥j to fl℥j of water.

Stomatitis Ulcerosa.—Ulcerative stomatitis is a much more serious affection than any of the forms previously described, although it is not considered to be a dangerous malady, like the other forms of the affection, is in general a disease of childhood, and is most frequently observed between the fifth and tenth years. The disease is rarely seen outside of hospital wards and public clinics, as it seldom attacks the children of the better class of society. Individuals who are "badly nourished or debilitated from disease, such as scrofulous disorders, intestinal complaints accompanied by exhausting discharges, typhoid fever, diabetes or scurvy" (Ziegler), the exanthems, pneumonia, or irritation from diseased teeth, are most susceptible to the affection. Convalescence from exhausting acute disease predisposes to an attack. There seems to be good evidence that the disease is sometimes epidemic, as several cases are usually seen at about the same period, while on the other hand, long periods may elapse without the appearance of

a single case (Swift). Unsanitary surroundings, like cold, damp, impure air, seem to favor its appearance and its dissemination.

The disease is generally acute in type, rarely chronic, and "always starts from the alveolar margin of the gums" (Bohn).

Causes.—The causes are, principally, bad hygienic surroundings, unwholesome food, insufficient nourishment, debility from acute and exhausting diseases. Local injuries and irritations from diseased teeth, and the chronic poisoning by mercury, phosphorus, lead, and copper (Ziegler) may also be causative factors in the production of the disease.

Symptoms.—The disease begins in the margins of the gums by redness, swelling, pain or sense of discomfort, tenderness and loosening of the gums from around the teeth. The loosened margins and festoons of the gums become swollen, congested, and partially cover the teeth, which now become loosened, and hemorrhage is easily provoked. Later the swollen and congested gum becomes discolored, softens, and sloughs away as a yellowish mass, leaving an irregular, ulcerating surface. The ulcers thus formed present an angry red surface and thickened borders, the center of the ulcer being sometimes partially covered with shreds of necrotic soft tissue. The progress of the ulceration is rapid, extending to the deeper tissues and to the surrounding parts of the cheeks and the lips. It occasionally involves the periosteum and the bone, causing necrosis and exfoliation of considerable portions of the alveolar process and the neighboring teeth.

In the milder cases involving a limited area of ulceration, there is a slight rise in the temperature, accompanied by other mild febrile symptoms, which may last for a few days and subside upon the healing of the ulcerations. In the more severe cases in which the ulceration is extensive and involves the periosteum and the bone, the temperature may run high and not subside for several days, dependent upon the extent of the ulceration and the tissues involved. In those cases in which the ulcerations are deep, foul, and extensive (Swift), the breath is offensive and the saliva, which is increased in quantity, is discolored with pus and blood, and emits a foul odor; the salivary glands become enlarged and tender, and the jaws swollen and stiff. Sometimes the disease resembles a gangrenous stomatitis, and there is always danger that it may assume such form. (See NOMA, chapter on Gangrene.) The prognosis of ulcerative stomatitis is good even in the severe form, provided it does not become gangrenous.

Treatment.—This consists in the first place of correcting the bad condition of the health which may have been the cause of the affection. The regulation of the diet to the individual needs of the patient is a matter of prime importance, and should receive immediate attention. If the surroundings are unhealthy the child should be removed as

quickly as possible to a more healthful environment, preferably to the country or the seashore. Stimulants and tonics may also be necessary to assist the enfeebled system to regain its normal tone, but change of air and good food are the most valuable means at our disposal in the cure of this affection. In the local treatment the application of a solution of the chlorate of potassium every two or three hours is a valuable remedy. Swift recommends a solution of the sulfate of copper gr. v to fl℥j of water, or boric acid in glycerol or honey. Day advocates the use of the silver nitrate solution gr. v to fl℥j of water, used in conjunction with the chlorate of potassium solution; also the painting of the ulcers with the tincture of iron and glycerol.

REVIEW.

CHAPTER XXIX.

Give the definition of stomatitis.

What affections are included in the term stomatitis?

To what period of life is stomatitis usually confined?

What resemblance is there between inflammatory conditions of the oral mucous membrane and of the skin?

Is diphtheria always confined to the fauces and palate?

What drugs may cause a stomatitis?

Name the various forms of stomatitis.

How have the various forms of stomatitis been classed?

How are they classed at the present time?

How many varieties of stomatitis are there with white patches?

By what means may they be distinguished from one another?

What is necessary to establish a positive diagnosis?

Describe stomatitis simplex.

In what class of children is it usually found?

With what other conditions is it associated?

What is the cause of the disease?

What other term is sometimes applied to this form of the affection, and why?

Describe the local symptoms.

What are the causes of stomatitis catarrhalis?

By what condition is it often preceded?

Describe the appearance of the affection.

How are the mucous cysts developed?

What other condition is sometimes found upon the lips and angles of the mouth?

In what class of children is the disease most common?

With what is it usually associated?

What are the causes of the affection?

Describe the local symptoms.

What are the constitutional symptoms?

What is the prognosis?
What should be the constitutional treatment?
To what may the affection be many times traced?
How may dental irritation be relieved?
What should be the local treatment?
What is the popular term applied to stomatitis aphthosa?
What is Ziegler's opinion as to the character of the disease?
In what classes of individuals is it most frequently observed?
With what diseases is it sometimes associated?
With what disease of the skin has it been compared?
What is the characteristic feature of stomatitis aphthosa?
Where are the patches most abundant?
How are they characterized?
Of what do the aphthous patches consist?
How may the disease terminate?
Are true ulcers developed?
How may suppuration develop?
Name the causes of the affection.
Describe the local symptoms.
What is the character of the constitutional symptoms?
What does a temperature of one or two degrees above the normal indicate?
Give an outline of the treatment.
What is the popular term applied to aphthosa parasitica?
What is the character of the disease?
Name the organism which produces the disease.
Upon what form of epithelium does it develop most readily?
In what classes of individuals is it most frequently observed?
How is the disease carried to other children?
How may its spreading be prevented?
Under what circumstances is the disease observed in the adult?
What conditions favor the growth of the organism?
Describe the local symptoms.
What constitutional symptoms are developed?
Under what conditions may the disease terminate fatally?
Give an outline of the constitutional treatment.
What should be the local treatment?
What is the nature of stomatitis ulcerosa?
At what age is it most likely to appear?
Where is it most commonly seen?
What conditions render individuals most susceptible to the disease?
What circumstances point to the epidemic character of the disease?
What conditions favor its appearance and dissemination?
In what form does the disease usually appear?
Name the causes of the disease.
Describe the local symptoms of the disease.
What are the constitutional symptoms?
Does the disease ever become gangrenous?
What is the prognosis?
What constitutes the first step in the treatment?
What should be done about the diet and the surroundings?
Upon what means is the greatest reliance to be placed for the cure of the disease?
Describe the local treatment.

CHAPTER XXX.

SURGICAL TUBERCULOSIS.

Definition.—*Tuberculosis* (Lat. from *tuberculum,* dim. of tuber, a little swelling. An infectious disease caused by the *Bacillus tuberculosis.*

Tuberculosis has a widespread, almost universal distribution among the human race, and it has been estimated that more than one-seventh of the entire population of the civilized world die from its effects. Warren places the mortality as about one to every five deaths, and when the fact is taken into consideration that a considerable portion of those who contract the disease finally recover their health, it will need no other demonstration to prove that the disease is one of the most serious and widespread of all the afflictions of mankind. The active etiological factor in the disease is the *Bacillus tuberculosis,* which was discovered by Koch in 1882. The discovery of this bacillus, and the demonstration, also by Koch, that by it only could the various phenomena of the disease be produced, have wrought great changes in the views held as to the pathology of the disease, revolutionizing a large and interesting department of surgery.

Senn says, "Tubercular lesions furnish a most excellent illustration of the origin, force, termination, and tissue-changes of what is known as chronic inflammation, and a description of the histology of a tubercular nodule is a description of the pathology of chronic inflammation.

"Of all the diseases which are produced by micro-organisms, next to that of suppuration, tuberculosis is of the greatest interest to the surgeon; of greatest interest, because it is better understood, from the bacteriological standpoint, than are most other surgical diseases which come under his care, and of no less great importance on account of its frequency."

"The discovery of Koch has also done away with that vague and indefinite term *scrofula,* which has been used so long to indicate a large and ill-defined class of diseases, for later experiments have proved conclusively that they were identical in all respects with recognized forms of tuberculosis."

Avenues of infection.—The virus of tuberculosis gains an entrance into the body by various channels,—through the inspired air, with the

food, and by direct inoculation. It has been recognized since 1826 that the disease was transmissible by inoculation through the case of Laënnec, who injured his finger with a saw while making an autopsy upon a subject affected with tubercular disease of the vertebræ, and thus contracted the affection. He finally died, some years afterward, of tubercular disease of the lungs.

Through experimentation, it was found that tubercular peritonitis could be produced by the injection of infected sputa into the peritoneum of guinea-pigs; infected food produced tubercular ulceration of the mesenteric glands; and the dried sputum when inhaled produced tubercular inflammation of the lungs.

Senn, in speaking of the frequency of the disease and the dangers of infection, says, "At least one person out of every seven dies of some form of tuberculosis. Most of our large hospitals contain from twenty-five to fifty per cent. of patients afflicted with the disease. . . Health resorts frequented for years by tubercular patients have become infected to such an extent that there is great danger of the whole population becoming exterminated by this disease. The sources of infection in such places have become so numerous that it is unsafe to breathe the air, to drink the water, or to eat the food prepared in houses which for years have been hot-beds for the *Bacillus tuberculosis*, and by persons carrying the microbes upon every square inch of their surface. That whole communities and nations where this disease has been prevalent for centuries have not been completely depopulated long ago is owing to the fact that many persons possess, from the time of their birth, such a degree of resistance to infection that even direct infection by inoculation would prove harmless."

The question of hereditary transmission of the disease from the mother to the child through the placenta is one which has been earnestly discussed, Baumgarten and others maintaining that the virus may be implanted during fetal life, and yet not show itself until perhaps many years later.

Experiments, however, upon pregnant guinea-pigs by inoculation failed to produce the disease in their offspring. According to Warren, tuberculosis in new-born children is exceedingly rare, and in those cases reported with early manifestations of the disease it is an open question as to whether the disease may not have been contracted from the milk of the mother, or in various other ways. Most authorities, however, maintain that a predisposition may be inherited through a peculiar weakened condition of the tissues and fluids of the body, which makes them a favorable soil for the growth and propagation of the bacillus.

In substantiation of this statement, it is generally found that tubercular subjects have a family history of tuberculosis.

The *pulmonary tissues* are probably the most common avenue through which the tubercular virus enters the system. The *Bacillus tuberculosis* is very tenacious of life, and retains its vitality for a considerable period even in the dried state. These attributes make it a constant menace to the health of those persons who come in contact with tubercular subjects, unless the greatest care is exercised to destroy the sputa and discharges while in a moist state. The bacillus when in a dried state is capable of being floated in the atmosphere and introduced into the lungs by inspiration, and therefore becomes a source of great danger, as has been frequently proved by experiments upon animals. It has also been shown by Cornil that the dust of rooms occupied by such patients contains large numbers of bacilli; while Prudden and others have found it in the dust of the streets. The linen, carpets, and dishes used by tubercular subjects are also a source of danger, as is also the communion cup; while the habit of expectorating upon floors of public halls, street and railroad cars, indulged in by so many consumptives, is an added danger to the public health which should be prohibited.

Certain cases of pulmonary tuberculosis, according to Bollinger, are not due to the inhalation of the bacilli, but to metastasis from disease in other parts of the body. Tuberculosis of the upper extremities is most liable to produce secondary infection of the lungs. Tubercular disease of the wrist is a well-known cause of pulmonary consumption.

The alimentary tract is also a channel through which tubercular infection may take place. It has been frequently demonstrated that the intestinal tract of animals can readily be infected by feeding them with tuberculous food. In the human subject primary tuberculosis of the intestines produced from infected food is not an uncommon occurrence. Milk from tuberculous cows, and water infected with the virus, have long been recognized as sources of danger. The viability of the bacillus is somewhat remarkable, Cornil having demonstrated that it could live in sterilized water at the ordinary temperature for seventy days.

Roasted meat, if infected with tubercle, may be a source of considerable danger on account of the central portions not being subjected to the same high degree of temperature during the process of cooking. The *Bacillus tuberculosis* is destroyed if subjected to a temperature of 212° F. for four minutes, but the spores will resist a much higher temperature, and for a longer time.

Secondary tuberculosis of the intestines is due to auto-infection, as the sputum is frequently swallowed. The investing membrane or capsule of the bacillus is not readily acted upon by the gastric juice, consequently it arrives in the intestinal tract in an uninjured condition, where it attacks the Peyer's patches, or the solitary glands.

Later, the mesenteric glands become infected, and also the peritoneum. In women, the infection frequently takes place through the genital tract.

The mucous membranes of the mouth, nose, and pharynx are also channels through which infection may take place. The disease may be transmitted from one individual to another by kissing, or by the drinking-vessel or spoon used by a person suffering from pulmonary tuberculosis. The tongue spatula, or the instruments and hands of the dentist, may become a source of considerable danger, unless they are carefully washed and sterilized after being used upon such a patient. As much care should be taken in this direction with instruments, etc., as would be given to them after having been used upon a syphilitic subject.

The skin is sometimes the avenue for the introduction of the tubercular virus. Bollinger thinks this channel of infection is underestimated. Direct inoculation, however, through the skin does not play a very important rôle in the causation of the disease. All cases of primary tuberculosis of the skin, however, are the result of inoculation. The bacillus does not seem to have the power to enter the skin like the pus-producing cocci. Infection, however, may occur through superficial wounds and slight abrasions of the cutis. A considerable number of cases have been reported during the last few years, —enough, it would seem, to establish the fact that tubercular infection may take place in man by absorption of the virus through slight abrasions and superficial wounds of the skin.

Tubercular infection occasionally takes place in those whose duty it is to perform autopsies upon bodies of persons who have died of tuberculosis, as in the case of Laënnec, just mentioned.

Watson Cheyne also reports such a case in a student who injured the finger at the base of the nail. A wart formed, which remained as an ulcer after three years of treatment. Later an abscess formed upon the back of the hand, and finally the finger was amputated. Six years after the injury he died of tubercular meningitis.

All portions of the body do not appear to be equally open to infection. Certain tissues and organs seem to have a predisposition for the disease, viz: the lungs, the lymphatic glands, and the bones. The face and head are peculiarly liable to infection. There is, however, hardly a tissue of the body which under favorable conditions may not become the seat of primary tubercular infection, or escape secondary infection when the virus is disseminated through the general infection.

The lymphatic system is often the avenue through which remote parts of the body may become infected. The lymphatic glands, however, on the other hand, exert a protective influence against the dis-

semination of the disease, by retarding the progress of the bacilli or indirectly accomplishing their destruction.

The bacilli may gain an entrance to the general circulation either through the thoracic duct, after having traversed the last chain of glands of the lymphatic system, or by the breaking down of tubercles in the immediate vicinity of blood-vessels, the contents being discharged directly into the blood-current. Eventually the bacilli are conveyed to some arteriole or capillary where they become lodged, and the conditions are established which favor the development of miliary tubercle.

Pathology.—The lesion which is produced by the growth of the *Bacillus tuberculosis* in the tissues is known as *tubercle.* It is variously designated as the miliary or gray tubercle or nodule.

In appearance it is a grayish, translucent mass from 1-10 to 2 mm. in diameter, and firmly imbedded in the surrounding tissues. It is made up of an aggregation of cells which are microscopic in size, and is the product of a minute point of inflammation established by the presence of the tubercular bacillus. Larger masses are formed by the coalescence of neighboring tubercles, producing the so-called *tuberculous infiltrations.*

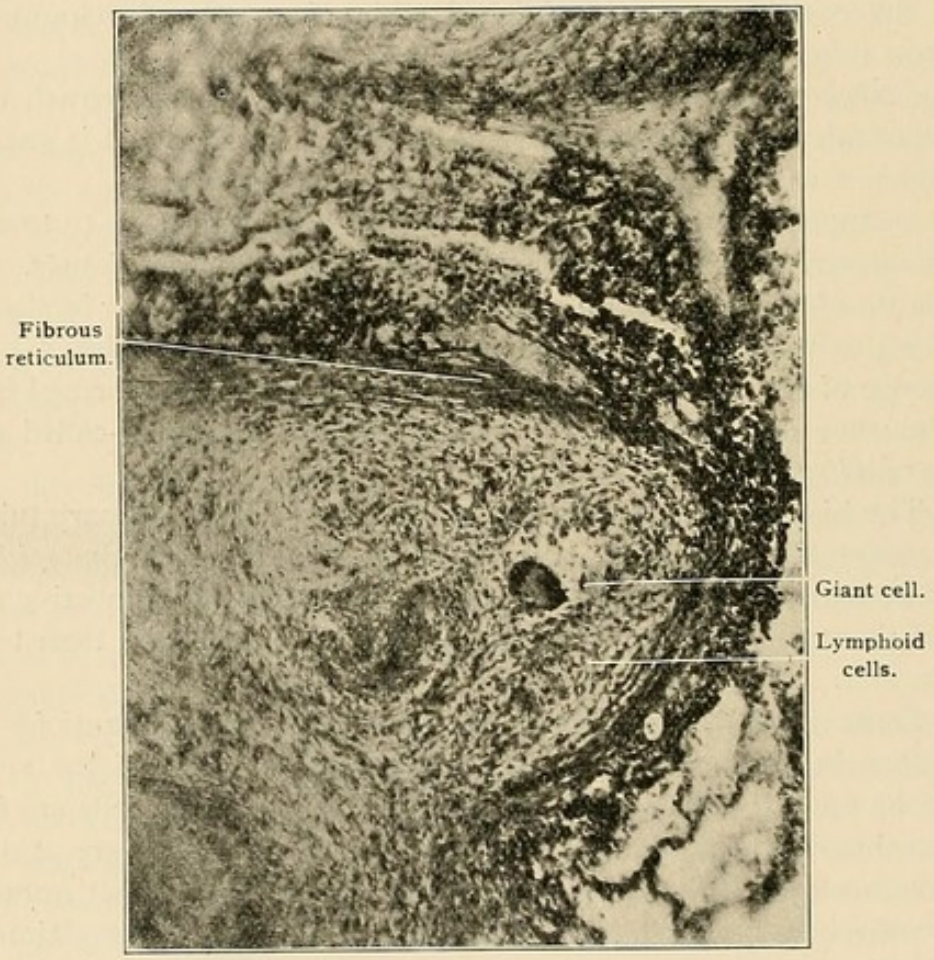
The histologic elements which make up a typical primary tubercle are three groups of cells,—the round or *lymphoid cells,* the *epithelioid cells,* and the *giant cells,* and a delicate reticulum of connective tissue which is more dense at the outer surface of the tubercle than toward the center. (Fig. 110.)

Senn classes the *lymphoid cells* as *leucocytes,* and accounts for their presence in the tubercle by the inflammatory action of the specific microbe upon the walls of the capillary vessels. These cells are found most abundantly at the periphery of the tubercle, but are scattered about through all the cellular elements, and they are most numerous when the inflammatory process is acute. These facts, he claims, are convincing proofs of the inflammatory nature of tuberculosis.

The *epithelioid cells* are so designated by their resemblance to epithelial cells. These cells were first described by Rindfleisch. Klebs calls them platycytes. Cheyne is authority for the statement that the epithelioid cells are the most characteristic cellular elements of tubercle, and are more constant than the giant cells. They are probably derived from the epithelial tissue (epithelium and endothelium), and are about two or three times as large as the white blood-corpuscles; in shape they are round or elongated; in structure finely granular, and they contain one large or several smaller nuclei. They are scattered all through the tubercles, but are found in the greatest numbers grouped around the giant cells and at the periphery of the nodule.

The *giant cells,* according to Senn, are hyperplastic epithelial cells, and consequently are derived from the same kind of tissue. They are a characteristic feature of tubercular nodules, one or more being found in the center of each. (Fig. 111.) This feature enables the microscopist to make an almost positive diagnosis, even though the tubercle bacilli cannot be found in a nodule.

FIG. 110.



TUBERCULOSIS—RETICULAR FORM—LUNG, SHOWING FIBROUS RETICULUM, LYMPHOID AND GIANT CELLS. × 50.

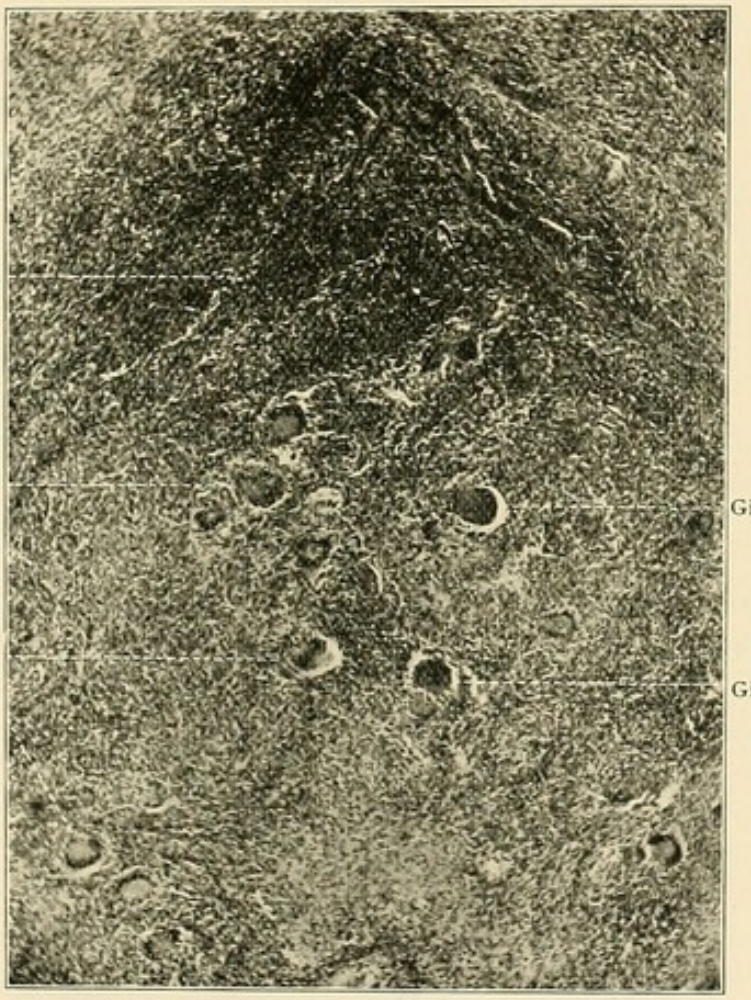
The giant cell in structure is finely granular, and contains multiple nuclei. These nuclei occupy chiefly a position at the periphery of the cell, and are arranged with their long diameters radiating from the center. Occasionally they are arranged in the form of a crescent at one end. During the progress of the disease the giant cells become progressively fibrous at their periphery, which gradually encroaches upon the protoplasmic central portion. The bacilli are found in the giant cells; also between and in the epithelioid cells, and in the later stages in the round cells.

Degeneration of the nodule begins first in the center of the giant

cells, and as this central degeneration progresses the bacilli disappear in this portion of the cell, though they may still be found at the periphery.

The giant cells of tubercular tissue are similar to the cells found in normal tissue (particularly in bone and the medullary tissue). They are also found in the tissues surrounding foreign bodies which are undergoing the process of encystment. Giant cells have the power of

FIG. 111.



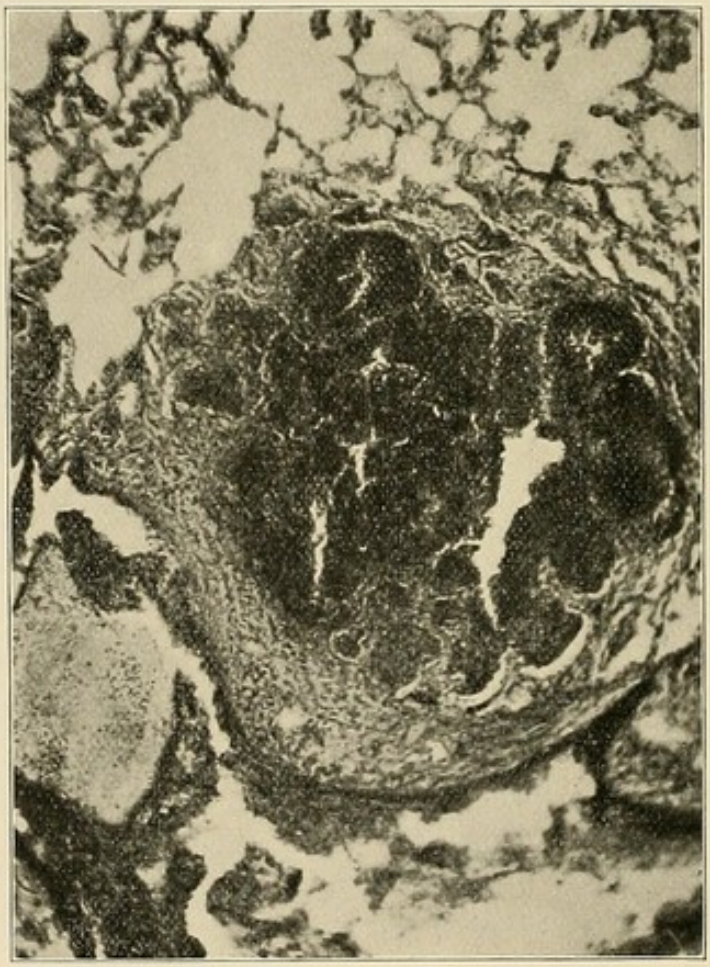
ACUTE TUBERCULOSIS—SMALL NODULE, SHOWING MANY GIANT CELLS. LUNG. × 50.

amœboid movement, which enables them to take up into their protoplasm small bodies, such as micro-organisms, disintegrated blood-corpuscles, etc.

The *reticulum*, according to Warren, is not usually a new formation, but is composed of the pre-existing intercellular connective tissue. Cell-growth being most active in the center, a certain amount of pressure is exerted from within outward, causing a thickening of the fibrous elements, which sometimes amounts almost to the formation of a capsule. Sometimes the reticulum seems to be formed, to a large

extent, by the processes of the epithelial cells. As the vascular supply is generally very slight, the smaller vessels soon disappear altogether. In consequence of this, the vitality of the nodule is soon greatly lowered, resulting in the death of the cellular elements, or coagulation necrosis, followed by granular disintegration and fatty degeneration of the cells, producing the condition known as *caseous degeneration.* The tubercular bacillus, by its specific action, or through its ptomaines, also appears to exert an influence which brings about a chemical change in the cells.

FIG. 112.



Tubercle.

TUBERCULOSIS—CASEOUS TUBERCLE. LUNG. × 50.

The process of caseation (Fig. 112) begins in the center of the nodule, and may gradually extend so as to affect the entire mass. When caseous degeneration is extensive, ulceration may take place, or abscesses may form. Sometimes calcareous materials are deposited in the mass, resulting in what is known as *calcification.*

Arrest of the disease frequently takes place in the lungs, and occasionally in the lymphatic glands, by the processes of caseation and calcification.

Calcification is nature's method of preventing the local extension of the disease, and guarding against the infection of surrounding tissues or of the general system.

Liquefaction of the caseous material, and the formation of abscesses, is the more common termination of the cheesy degeneration. The material thus formed has always heretofore been regarded as pus; recent investigations, however, have established beyond a doubt that it is the product of a retrograde metamorphosis of tissue, and not true pus.

Tubercular, or the so-called cold abscess, contains a fluid which to the unaided eye resembles pus, but when subjected to examination by the microscope it presents none of the characteristic histologic elements of pus.

The effects produced by the presence of the tubercular bacillus in the tissues are always those of chronic inflammation, and this invariably results in the production of granulation-tissue. The embryonal cells of which the granulation-tissue is composed seem under certain conditions to act as a wall of protection against the encroachment of the disease upon the surrounding tissues.

The secondary infection of tubercular abscesses with the pus-microbes causes a breaking down of this wall of protection, and the patient incurs the dangers of local septic infection, and a general dissemination of the tubercular condition.

Garré is of the opinion that many cases of tubercular ulcerations and abscesses are the result of a mixed infection. The examination of the contents of cold abscesses, and of the liquefied caseous material of tuberculous cavities in bone, revealed no pus-microbes, not even in those which pursue a rapid course. He therefore believes it is possible in many cases of suppuration following the tubercular process, that the pus-microbes had ceased to exist before examination for their presence was instituted.

When the specific bacillus meets with a sufficient resistance from the tissues surrounding the nodule, it eventually exhausts the nutrient material found in the granulation, and either dies or assumes a latent condition. The granulation-tissue is then converted into cicatricial tissue, and the local manifestations of the disease disappear.

If, on the other hand, the bacilli are present in sufficient numbers to cause destruction of the embryonal cells with coagulation, caseation, and liquefaction of the infected tissue, a spontaneous cure may still be possible, by absorption of the fluid portion and the encystment of the solid débris. If bacilli or spores remain behind, there will always be great danger of a relapse in this disease.

REVIEW.

CHAPTER XXX.

What is tuberculosis?

Is it a common disease?

What is the estimated mortality?

What is the active cause of the disease?

When and by whom was the Bacillus tuberculosis discovered?

What effect has this discovery had upon the views held in reference to the pathology of the disease?

How has it affected the surgery of tubercular diseases?

What has the discovery of Koch proved in reference to those diseases classed as scrofulous?

When was the inoculability of the disease first recognized?

How was the infective nature of the disease established?

Is the disease transmitted by heredity?

What facts seem to prove that it is not?

Is a predisposition to the disease inherited?

Of what does this predisposition consist?

Which is the most common channel through which the bacillus enters the system?

What attributes of the bacillus make it a menace to public health?

How does the bacillus generally gain entrance to the pulmonary tissues?

By what other means may it gain a lodgment in these tissues?

By what means may the alimentary tract become infected?

How long did Cornil find the bacillus could live in sterilized water?

What degree of heat will destroy the bacillus?

Will this degree destroy the spores?

How is secondary infection of the intestines produced?

What is the effect of the gastric juice upon the capsule of the bacillus?

What intestinal glands are affected by the disease?

How may infection take place through the mouth, nose, and pharynx?

What means can be used to prevent infection through instruments, etc.?

Is the genital tract ever an avenue of entrance through which tubercular infection may take place?

How may infection take place through the skin?

Does direct inoculation play an important part in causing the disease?

What persons are not liable to infection through this channel?

Are all parts of the body equally liable to infection?

What tissues and organs are most susceptible to the disease?

Are any tissues exempt from the primary and secondary forms of the disease?

Through what channels does secondary infection usually take place?

How do the lymphatic glands exert a protective influence over the disease?

How may the bacilli gain an entrance to the general circulation?

What term is applied to the lesion produced by the growth of the Bacillus tuberculosis in the tissues?

What are its various designations?

Describe miliary tubercle.

Describe tuberculous infiltration.

What are the histologic elements of primary tubercle?
What is the character of the lymphoid cells?
How are they distributed in the tubercles?
Describe the epithelioid cells.
How are they distributed?
Describe the giant cells.
Are they always present in tubercular nodules?
What location do they occupy in the nodule?
What changes take place in them during the progress of the disease?
How are the bacilli distributed in the nodules?
In which cells does degeneration of the tubercle begin?
What occurs in reference to the bacilli as degeneration progresses?
Describe the reticulum.
Of what is it composed?
What two factors are supposed to bring about caseous degeneration?
Where does this process first begin?
What conditions may take place when caseous degeneration is extensive?
What other form of degeneration sometimes takes place?
What is nature's method of preventing the extension of the disease?
What is the more common termination of caseous degeneration?
Is this material pus?
Do cold abscesses contain pus?
What is the effect upon the tissues of the presence of the tubercle bacillus?
What seems to be the office of the granulation-tissue?
What is the result of secondary infection of tubercular abscesses with the pus-microbe?
What becomes of the bacilli when the tissues offer a successful resistance?
What becomes of the granulation-tissue?
If the bacilli destroy the granulation-tissue and produce caseation, liquefaction, etc., how may nature still establish a cure?
If bacilli or spores remain, what is to be apprehended?

CHAPTER XXXI.

SURGICAL TUBERCULOSIS (Continued).

Tuberculosis of Bone.

With the definite statement of the proposition that tuberculosis is an infectious disease caused by the *Bacillus tuberculosis;* that it is characterized by the production of a peculiar tissue designated as tuberculous, and of certain inflammatory products which appear in the form of nodules or miliary tubercles, and as a diffuse infiltration, and which rapidly undergo caseous degeneration; we may now turn to the consideration of those tubercular conditions which are found affecting the bones, the skin, and the mucous membrane, as of greatest interest from the standpoint of the oral surgeon and the dentist.

Tuberculosis of the bone is one of the most common of tubercular affections, those of the lungs and lymphatic glands only being more frequent. Tubercular disease of the bones occurs very often in children and youth,—in fact, the great majority of cases occur before adult life. Dollinger reported, as a result of investigation into the family history of two hundred and fifty cases of tubercular disease of the bones, that in more than one-third of them, one or more of the immediate ancestors had suffered from pulmonary tuberculosis, usually the grandparents. He therefore comes to the conclusion that the influence of the tubercular virus must be exerted through several generations before the normal resistance of osseous structures is so far weakened that they become a suitable field for the lodgment and development of the tubercular bacillus, and that in the inherited (?) form, or predisposition to tuberculosis, the lungs are attacked in the first generation, and the bones in the second.

The most common location of the disease is in the epiphyseal ends of the long bones, but it is, however, frequently found in the short bones of the hand and the foot, and occasionally in the flat and irregular bones, as the vertebræ, the ribs, the scapula, the ilium, the bones of the cranium, of the nose, and of the face. The disease is most frequently found in the cancellated structure of the bone, but it may occur in the compact tissue, and in any portion of the bone.

The disease may be *primary* or *secondary* in its origin.

It is probable, however, that only a very small portion of the cases of tubercular nodules of the bones are primary in their origin; the great majority of them are secondary to disease of the lymphatic glands, of the bronchial or mesenteric group, infection having taken place through the mucous membrane of the respiratory or alimentary tract, the bacilli being transported through the circulation, and deposited in the bone. Landerer, according to Warren, examined *post mortem* one hundred and fifty cases of tubercular and bone diseases, and with one or two exceptions found tubercular disease of the bronchial glands that evidently antedated the bone-affection.

Primary tuberculosis of bone when it does occur is doubtless the result of inoculation, through wounds and abrasions,—in other words, of traumatic origin.

Authorities differ as to the influence of traumatic injuries in producing tubercular disease of the bones. Senn quotes Volkmann as saying that traumatisms which produce tubercular disease of the bones are always slight, and often insignificant. Senn himself believes that only in a small per cent. of tubercular disease of bones can the disease be traced to a traumatic origin. Warren says the great majority follow slight contusions and sprains. Experience teaches that tuberculosis of bone rarely if ever, even in tubercular subjects, follows a severe injury or fracture of the bone, doubtless on account, as Senn expresses it, of the active cell-proliferation going on about such an injury that neutralizes the pathogenic action of the bacilli which might reach the seat of the injury with the extravasated blood. But in injuries less severe, the same cell-activity does not exist, the tissues are disabled for a brief period by the damage which they have sustained, and during this time they are in a less resistant state through the deleterious action of the bacterial ptomaines. They then become a favorable soil for the development of the bacillus, and, as already shown, in individuals predisposed to tuberculosis the seeds of the disease may already be present in the lymphatic glands, and only waiting for favorable conditions to begin their active growth. Such a point of injury might therefore establish a focus for the development of the bacilli, the formation of nodules, and later a more or less extensive tubercular osteitis.

It very rarely happens that tuberculosis of bone occurs during the progress of tubercular disease of the lungs, but pulmonary tuberculosis and diffuse miliary tuberculosis can frequently be traced to tubercular disease of the bone. The frequency with which this occurs is explained by the intimate relationship existing between the tubercular nodule in bone and the blood-vessels, thus rendering systemic infection almost certain. The tendency of tubercular disease of the glands, if allowed to take its course, is toward bone tuberculosis, and later, to pulmonary or diffuse miliary tuberculosis.

Volkmann says that a child suffering from glandular tuberculosis has a good chance of becoming the subject of osseous tuberculosis during adolescence, and to die of pulmonary tuberculosis before reaching the age of thirty.

A peculiarity of tubercular disease of the bone is, that it generally begins at that point where the growth is the most rapid and greatest in amount. In the long bones this is at the epiphyseal ends; in the flat and irregular bones at the outer borders. When infection takes place it is practically never direct, and when the disease makes its appearance it is only an evidence that the germs of the disease were already present in some other organ. When the bacilli are present in the blood-current they sometimes become localized in the terminal branches of an artery, particularly in partially developed bone, by being arrested in their progress, and the lumen of the vessel is obliterated by the presence of a minute embolus of granulation-tissue containing bacilli, or the caliber of the vessel may be gradually decreased until it is finally obliterated by the formation of a mural thrombus around bacilli which have found a lodgment upon the inner walls of the vessel. In young persons the new vessels which are forming in partially developed bone are, by their imperfect structure, and the irregularity of their contour, the most favorable in location and conditions for the arrest of floating granular particles and bacilli. These conditions would therefore seem to be a strong predisposing cause, and an explanation of the frequency with which tuberculosis of bone occurs at the point of greatest growth.

The relative frequency with which tuberculosis occurs in the bones of the head and face, as compared with other bones of the body, is one of considerable interest. Quoting from the tables of Schmallfuss, in Senn's "Principles of Surgery," we find Billroth places the bones of the cranium and the face third in the list, with a percentage of thirteen. Jaffe gives the bones of the cranium the eighth place, with a percentage of three; but no reference is made to cases affecting the bones of the face. Schmallfuss also places the bones of the cranium in the eighth place, with a percentage of four, but also mentions no cases affecting the bones of the face. If Billroth's cases of tubercular disease of the bones of the cranium did not average higher than those of the other authorities named, it would be fair to say that the ratio of cases of the disease in the bones of the face would be nine per cent.

Tuberculosis of the bones of the cranium is found most frequently in the bones of the ear and the mastoid process. After these, it is found chiefly in the frontal and temporal bones.

The bones of the face which are most often the seat of the disease are those of the nose, the superior maxillæ, the malar, and the palate. The inferior maxilla is rarely affected by the disease.

Tuberculosis of Bone, or *Caries of Bone,* is an ulcerative process; a

molecular death of bone, due, as we have already learned, to the presence of tubercular nodules formed by the action of the specific bacillus upon the bone-tissue in which it has been deposited.

The disease always begins as an intercellular osteitis, of low, chronic type, and Volkmann says these chronic tuberculous inflammations of bone have a tendency to form in the ends of the long bones near the joints, just as pulmonary tuberculosis does in the apex of the lung.

The clinical history of tuberculosis of bone, as well as the appearance of the tissues, with the unaided power of the eye, and also microscopically, is the same as that found in typical cases in other organs of the body. Warren describes these appearances as follows: "On making a section of the bone the tubercular nodule appears as a well-defined mass of reddish gray, yellowish white, or pure yellow color. The surrounding bony tissue is usually red and hyperemic, and the trabeculæ may be somewhat thickened. The cancellous spaces are devoid of fat-cells, and they contain a swollen semi-fibrous material. With a microscope the miliary tubercles are seen at the periphery of the nodule, its center being composed of broken-down cheesy material. The size of these nodules varies greatly. As they grow, the tubercular virus attacks the trabeculæ, leading to their absorption, the bone becomes softened, and breaks up into a mass of greasy, cheesy material, containing crumbling fragments of bone-tissue. When complete softening has taken place, the material of which the nodule is composed becomes puriform, and it may be washed away, leaving a cavity lined with granulation-tissue. In case the trabeculæ are not completely destroyed in the infected part, the cancelli between them become filled with cheesy débris; as the vitality of the part is destroyed, granulation-tissue is formed around the diseased mass, and absorption of the connecting trabeculæ occurs; the spongy sequestrum which is thus formed separating from the living bone. These sequestra are quite small, and are more or less globular in form. The surrounding bone becomes somewhat thickened, and the interstices are filled with gray fibrous tissue, or eburnation of the bone may in some cases take place. When the nobule has softened completely into pus—liquefied—the surrounding bone is covered by a tubercular membrane, or its surface is infiltrated with granulation-tissue, which usually contains miliary tubercles on its inner aspect, affording, nevertheless, protection to the adjacent bone. These small sequestra—spiculæ—lie firmly imbedded in a thick layer of blue-gray, transparent granulation-tissue, dotted with yellow spots. Large amounts of pus rarely accumulate around these nodules."

The crucial test, however, of the tubercular character of all chronic inflammatory bone affections, is the presence of the specific bacillus.

In many cases there is great difficulty in finding the bacillus, but Cheyne attributes this to the fact that they are more numerous in the first stage of the disease, and that their numbers decrease in the later stages, or rapidly pass into the spore formation. There are also difficulties in staining them, which are not understood, for sometimes in double staining some will take red and others blue, which he thinks is probably due to the different stages of their development.

Tubercular abscesses are formed by the breaking down into caseous material of confluent masses of tubercle in the center of a nodule. This material becomes infiltrated with fluids and leucocytes. A cavity is thus formed which contains fluid, fatty material, fragments of disorganized cells, and leucocytes, surrounded by granulation-tissue filled with tubercles.

Cold abscess is the result of the burrowing of pus, forming large cavities, into which tuberculous cavities at neighboring points may have discharged after liquefaction has taken place. The pus which these abscesses contain is so characteristic that it would never be mistaken after it had once been seen. It is of a pale white color, much thinner than the pus of acute abscesses; it frequently contains masses of cheesy material resembling coagulated casein, and for this reason is termed grumous. Sometimes it is mingled with blood, when it will be a dirty brown color. Small particles of bone are not infrequently present, which feel to the fingers like grains of sand. The presence of bacilli can rarely be demonstrated with the microscope in such pus. Cultures, however, yield the characteristic microbe.

The tubercular membrane, as it is termed, was first described by Volkmann. It is an opaque membrane, several millimeters in thickness, of violet gray or yellowish brown color, scantily supplied with blood-vessels on its inner aspect, containing innumerable clusters of miliary tubercles, which are supported by a matrix of coagulated fibrin. It is easily scraped off with the finger, or removed by washing with a stream of water from the irrigator, or peels off during an operation. This membrane is considered by Volkmann as an absolutely certain diagnostic sign of the tuberculous character of the abscess.

Burrowing of the pus is a much more rare condition in tubercular disease of the bones of the cranium and of the face than it is in the long bones and in the vertebræ, as the former are not so deeply covered by soft tissues.

Symptoms and Diagnosis.—In this disease the general symptoms are often of little value as an indication of the presence or the extent of the local affection, as it frequently occurs that patients with quite extensive tuberculosis of the bones may give every indication of robust health. König, who is authority on all matters relating to tuberculosis, has called attention to the fact that in nearly all cases of

even limited local tuberculosis there will be found a slight evening rise of temperature. Senn says that an evening rise, if not more than one-half of a degree F., if continued for weeks, should indicate a careful search for a local tubercular focus.

Anemia, if progressive, is always an unfavorable symptom, and is the result either of the extension of the disease to other important organs, or of exhausting discharges, growing out of secondary infection with the pus-producing micro-organisms. Such infection is always announced by a sudden and high temperature, with the accompanying signs of septic infection.

Pain, of a more or less mild character, is an almost constant symptom. It is rarely, however, so intense as in acute suppurative osteomyelitis, where the tension from the accumulated pus is sometimes very great. In tubercular inflammation, the primary exudation is always scanty, and the product of the inflammation is principally granulation-tissue formed from the pre-existing cells,—fixed tissue-cells; the bone in the immediate neighborhood becomes porous, thus allowing the pus to penetrate the bone, and relieving the tension that would otherwise exist, and mitigating the pain to a greater or less extent. When the pain is severe, it indicates an acute inflammatory condition. The pain is also intermittent, and always more severe at night. Another peculiarity of the pain is, that it is often referred to some remote part, as, for instance, in hip-joint disease, the pain is referred to the knee, and in tuberculosis of the vertebræ the suffering is usually experienced in the pit of the stomach, or some part of the abdomen supplied by nerves having their exit from the spinal canal near the diseased vertebræ.

Tenderness is usually present over a tubercular focus in the interior of the bone, which can be readily located by palpation. *Swelling* is usually absent in the early stage of the disease, or until the external compact tissue yields to the pressure from within or is perforated and forms a soft, boggy, circumscribed swelling beneath the periosteum. This condition is not always indicative of the presence of pus. The swelling may seem to fluctuate, but is misleading on account of the character of the granulation-tissue beneath, which gives it a pseudo-fluctuation. Such granulating foci have many times been incised under the belief that they were abscesses. When caseation takes place in the tubercular focus before perforation of the periosteum occurs, the surrounding tissues become rapidly infected, and a tubercular abscess is the result.

The color of the skin is not changed over a tubercular focus in bone, or over a tubercular abscess, until the granulations have permeated the deeper portions of the skin, or until the liquefied caseous material has so far reached the surface as to have only the skin for a covering, when it presents a dusky red hue. This is due to an impaired

circulation; the skin becomes thinner and thinner from atrophic changes induced by pressure, destruction occurring in the deeper portions, until finally it ruptures spontaneously, and the contents are discharged.

Differential Diagnosis.—As a means of differential diagnosis, a doubtful swelling may be explored (antiseptically) by a strong, spear-pointed steel needle,—or, if such an instrument is not at hand, a heavy hypodermic needle will serve the purpose. Such an instrument will usually enter the bone, which has been reduced in density by the action of the chronic inflammation, provided osteo-sclerosis has not taken place. In the active stage of tuberculosis of bone, the osseous tissue becomes softened and porous, so that sometimes the needle readily penetrates it. If the needle meets with any considerable resistance, it may be rotated as it advances; when it reaches the granulating focus or caseous mass, resistance is suddenly lost, and the needle may be passed through to the opposite side. The size of the cavity may be approximately determined by this method.

In tubercular disease of the bones of the face, it is also necessary to differentiate between syphilis, sarcoma, cysts, and chronic indurations located in the alveolar processes. The great majority of chronic inflammations of bones are due to tuberculosis. Senn claims that 95 out of every 100 cases are due to this cause. This, as a general statement, is quite correct, but it would need to be somewhat modified in applying it to the bones of the face. A bacteriologic examination is necessary to establish a positive diagnosis.

Prognosis.—The prognosis in cases of tuberculosis of bone is more favorable than if it were located in a joint, or in the skin, the lymphatic glands, or any of the internal organs. Tuberculosis of bone is sometimes spontaneously arrested, and a complete cure takes place, just as occurs in certain cases of pulmonary tuberculosis. This is brought about by the establishment of favorable conditions of the health, which give the system control of the disease, and limitations are defined, sometimes before caseation has taken place. If, however, caseous material has been formed, and it can be removed surgically, the prognosis is still favorable. As already stated in preceding pages, an individual who has suffered from osteo-tuberculosis in childhood or youth is always liable, under favoring conditions, to reinfection with the disease from the spores of the bacilli which may remain indefinitely in the tissues where they have been deposited.

In *osteo-tuberculosis of the face,* the part most liable to be affected is the infraorbital ridge. Tubercular inflammation occurring in this region is most common in children. Warren has seen it in adults. The disease progresses very slowly, being marked by swelling or fullness of the region; suppuration finally occurs, the skin may rupture

spontaneously, one or more sinuses are formed, the discharge becomes chronic, and may continue for months, terminating in unsightly scars and ectropion of the lower eyelid. A case of this character in a boy of seven years came under the care of the writer five years ago, in which the entire orbital plate of the maxillary bone and a portion of the body of the bone were destroyed, producing ectropion and closure of the nasal duct.

The malar bone is occasionally the seat of the disease. Warren mentions a case in which the disease in this location caused an extensive suppuration, and finally terminated in anchylosis of the jaw. Osteotomy was performed a year after the old sinuses had closed.

The bones of the nose are not infrequently the location of the disease. The infection may be primary, or it may be secondary to tuberculosis of the skin (lupus), or to the disease in the mucous membrane. In this way, also, the floor of the nasal fossa may become involved, and tubercular caries of the hard palate, with perforation, occur; or the disease may have its origin in the hard palate, and upon perforating the floor of the nares, extend to the bones of the nose, resulting in loss of tissue, and sometimes considerable deformity, by reason of the removal of the support to the soft tissues. In this form of the disease there is danger of confounding it with syphilitic manifestations in the same locality. It is therefore better where doubt exists as to the diagnosis to place the patient upon anti-syphilitic treatment for two or three weeks, when the diagnosis will most likely be made clear.

The alveolar process is also occasionally the seat of the affection, the disease generally being located through the chronic inflammatory process established by devitalized teeth. Garretson mentions a case of this character having its origin at the seat of a chronic abscess caused by a pulpless superior lateral incisor, which resulted in caries of nearly the entire upper jaw, and required an extensive operation for the removal of the diseased bone.

The writer recently operated upon a little boy, five years of age, for extensive caries of the alveolar process of the right upper jaw caused by the irritation of two badly decayed and devitalized deciduous molars. The bone was so soft as to be easily scraped away with the curette, while the pus was filled with cheesy masses. Fig. 113 shows a giant cell, etc., from tubercular disease of the angle of the lower jaw.

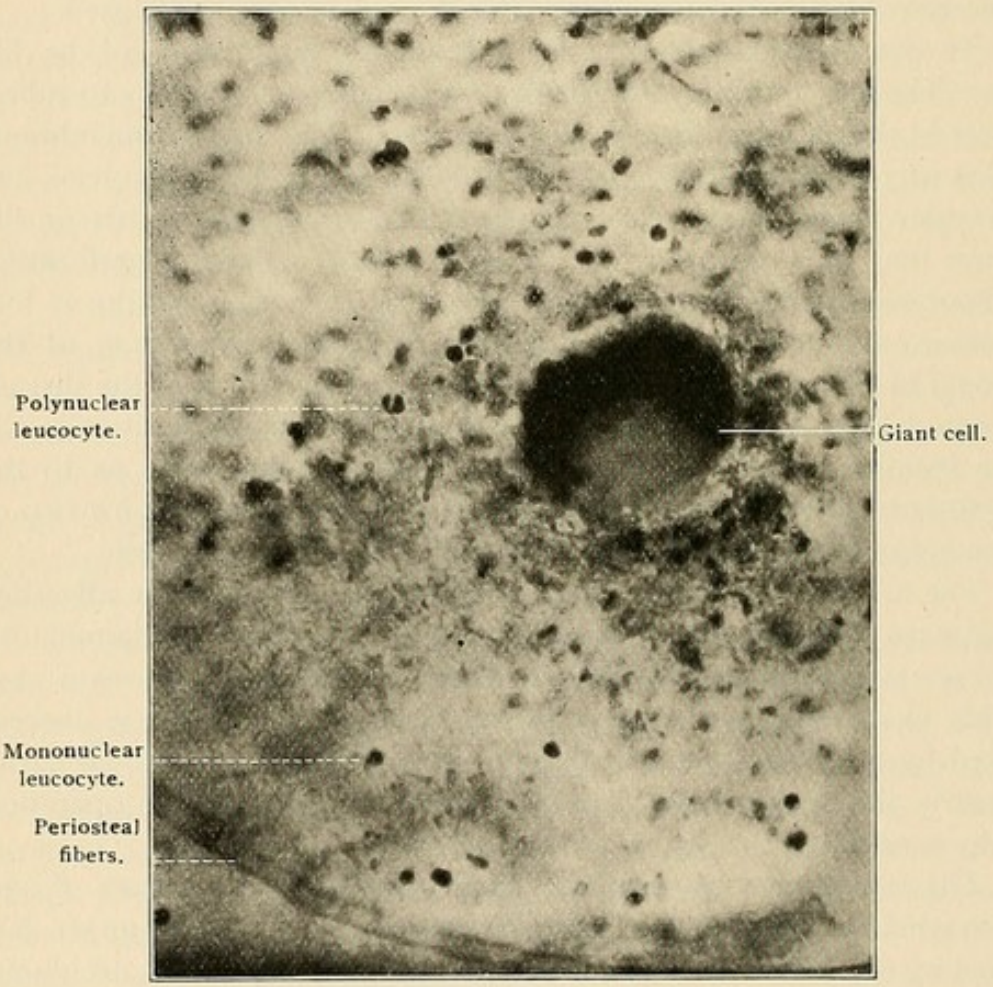
Treatment.—Patients who are suffering from tubercular disease of the bones need, first of all, tonic and supporting treatment, nourishing food, out-door air, and moderate exercise; change of climate, sea-bathing, or a sea-voyage are often of more real value than drugs.

The *local treatment* of tuberculosis of the bones of the face comprehends mainly a radical operation. This consists of removing, under antiseptic precautions, the entire focus of infected tissue, and the

measure of success will depend largely upon the stage of the disease when the operation is made. Success is more likely to be assured in the granulating form of the disease if caseation has not taken place. Operations should not be delayed after a positive diagnosis has been established, that adjacent tissues may not become involved, and that general infection may be prevented.

In operations upon the bones of the face and jaws, the writer has found the surgical engine and round burs the most satisfactory instru-

FIG. 113.

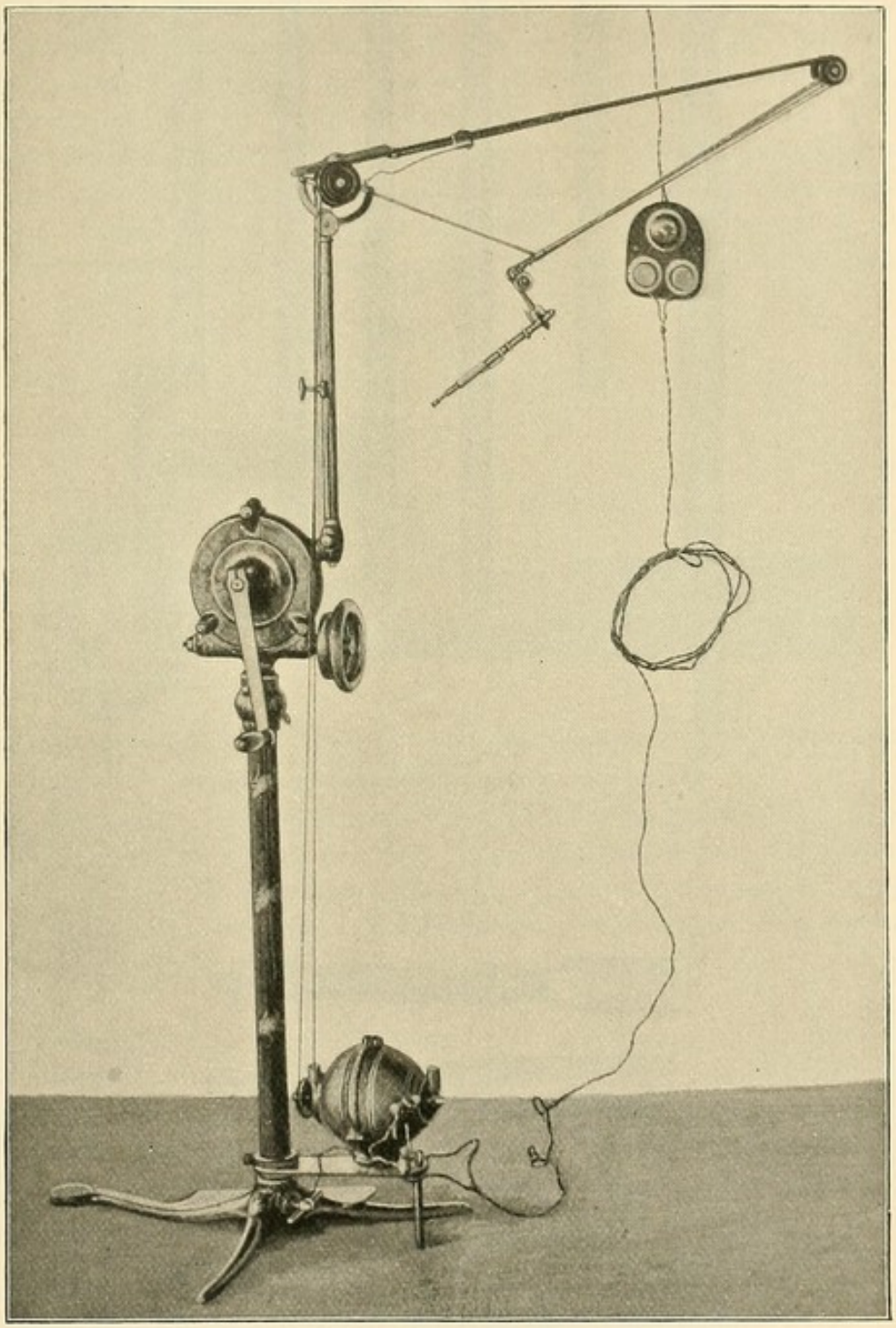


TUBERCULOSIS OF ANGLE OF INFERIOR MAXILLA, SHOWING GIANT CELL. × 800.

ments for the removal of carious bone. Figs. 114, 115, 116, 117 illustrate the improved surgical engine, trephine, burs and oseotome, etc. The curette and chisel, however, answer a good purpose, if the engine is not to be obtained. The delicacy and speed with which the bone can be removed with the engine and burs places these instruments in the front rank of this kind of bone surgery.

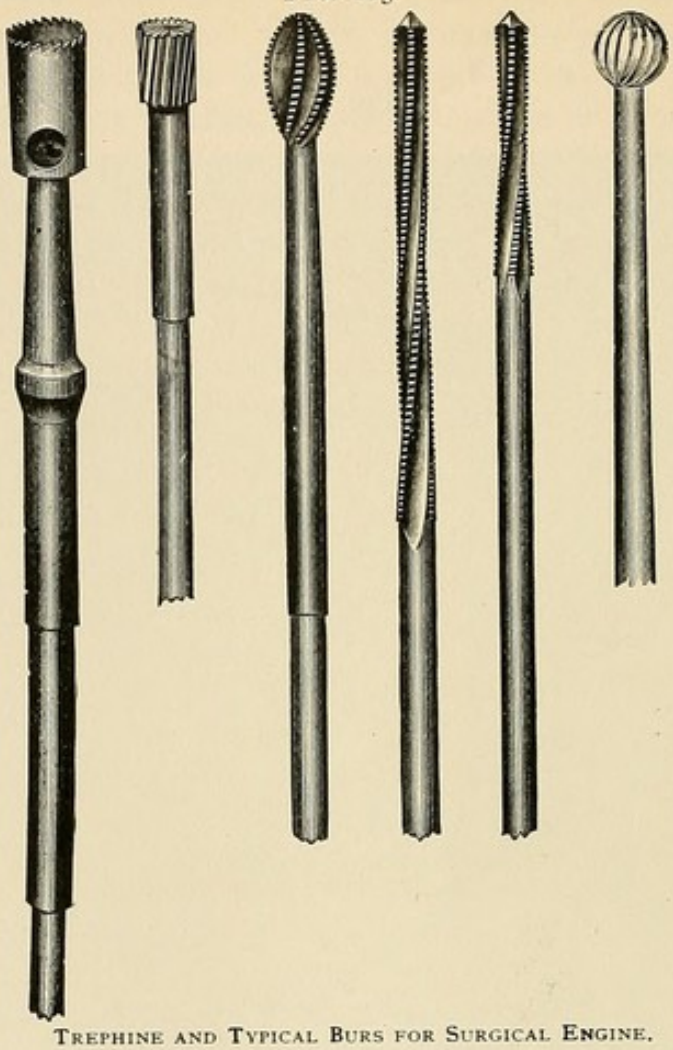
The softened or osteoporotic bone must be thoroughly removed, and the surface carefully examined, to see that healthy tissue has been

FIG. 114.



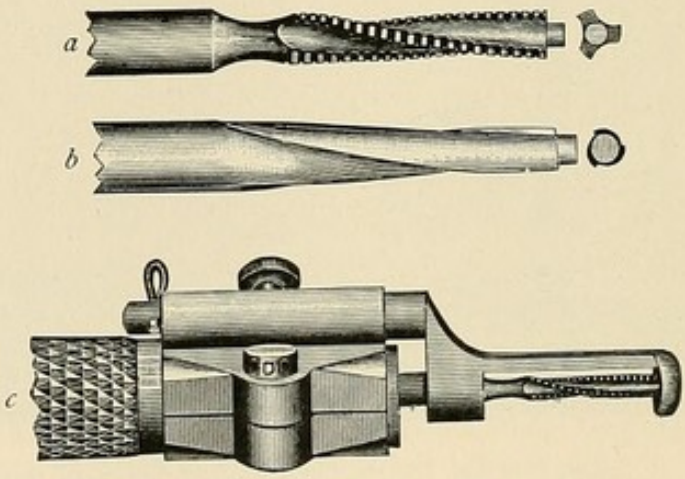
THE SURGICAL ENGINE IN ITS LATEST FORM.

FIG. 115.



TREPHINE AND TYPICAL BURS FOR SURGICAL ENGINE.

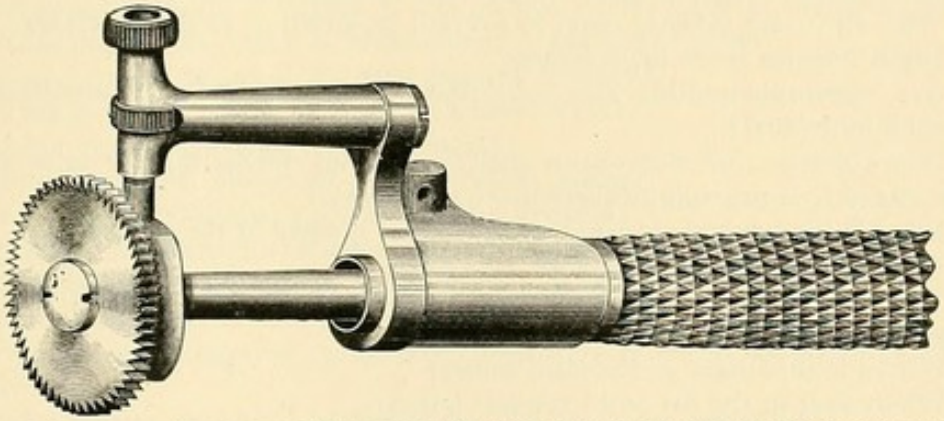
FIG. 116.



Two forms of spiral osteotome, one (*a*) with dentate, and one (*b*) with plain cutting-edges. *c* shows the osteotome mounted, with its button-like guard for cutting fenestra, etc., in the brain-case without injury to the subjacent membranes. *a* and *b* are twice the size of cutting-tool; *c* is full size.

reached; as an added precaution, the surface may be punctured at suspicious places with a sharp-pointed steel probe, as occasionally this instrument will reveal a concealed focus of infection that can then be removed. Senn recommends that the surface be punctured with a sharp-pointed Paquelin cautery to the depth of a few lines, as this procedure will destroy some of the bacilli that might remain, and also incite a plastic inflammation that would effectually resist the pathogenic action of such bacilli as were still present. Too much stress cannot be laid upon this part of the operation. The cavity is then to be dried, dusted with iodoform, packed with iodoform gauze; the edges of the wound sutured, except at the lower angle, where space is left for the removal of the packing and for drainage. Senn fills the cavity

FIG. 117.



CIRCULAR SAW WITH ADJUSTABLE GUARD FOR CUTTING THE BRAIN-CASE TO ANY PREDETERMINED DEPTH.

with antiseptic, decalcified bone-chips, suturing the periosteum separately in operations upon the long bones, and claims excellent results. Such a procedure ought to be available in the treatment of certain cases of tuberculosis of the superior maxillary and malar bones having external openings, and where loss of bone-tissue would cause a serious deformity.

Tubercular abscesses are treated by incision, and the removal of the tubercular membrane and granulation, and irrigation with an aqueous solution of iodin. The primary lesion must also be found and removed. Aspiration and injections of solutions of iodoform and glycerol are rarely practiced in connection with tubercular abscesses in the region of the face, for the very good reason that abscesses requiring such treatment are exceedingly rare in connection with the bones of this region. Most cases of tubercular abscess associated with the face can be treated more successfully by radical operation.

REVIEW.

CHAPTER XXXI.

At what period of life is tuberculosis of bone most common?

What is the usual family history of children suffering from bone tuberculosis?

Which of the bones are most often the seat of the disease?

What tissue of the bone is generally affected?

In what location of the bone is the disease most often found?

Is the disease primary or secondary in its origin?

How does primary infection occur?

Through what channels does secondary infection take place?

What influence do traumatic injuries exert in producing the disease?

What class of injuries seem to exert the greatest influence?

Why do severe injuries seem to prevent localization of the tubercular virus and slight injuries favor localization?

Are osteo-tuberculosis and pulmonary tuberculosis found associated in the same individual?

Is it common for pulmonary tuberculosis and diffuse miliary tuberculosis to be traced to a preceding tubercular bone-disease?

What is the general tendency of tubercular disease of the glands?

What does Volkmann say upon this point?

What peculiarity of osteo-tuberculosis in reference to the point of attack is almost universal?

Where is this point in the long bones?

Where is it in the flat and irregular bones?

What reasons are given for this localization in growing bone?

What is the relative frequency of tuberculosis of the bones of the head and face compared with the other bones of the body?

What is the estimated frequency of the disease of the bones of the face, as figured from Billroth's table?

Which bones of the cranium are most often affected?

Which bones of the face are most often affected?

What is tuberculosis of bone, or caries of bone?

Does it differ in clinical history or appearances from the disease in other organs?

What are these appearances?

What only can be considered the crucial test of the disease?

Are the bacilli always demonstrated with readiness?

What are the difficulties encountered in demonstrating them?

How are tubercular abscesses formed?

What is the character of the pus found in such abscesses?

Can the bacilli be demonstrated with the microscope in such pus?

How then may their presence be determined?

Describe the tubercular membrane which lines tubercular abscesses.

What does Volkmann say of its diagnostic value?

What is often the condition of the general health of patients in the early stages of tubercular bone-disease?

How is the temperature affected?

What would a constant low evening temperature indicate?

What would progressive anemia indicate?
What is the character of the pain?
What would severe pain indicate?
Is the pain always referred to the seat of the disease?
What symptom would locate a tubercular focus?
When is swelling present?
Does fluctuation always indicate the presence of pus?
At what stage of the disease is the color of the skin changed?
What causes this dusky-red hue?
What means can be used to diagnose a doubtful swelling?
What diseases of the face and mouth may simulate tubercular swellings?
What is the prognosis of osteo-tuberculosis?
Does a spontaneous cure ever occur, and, if so, under what circumstances?
Under such circumstances, is the patient liable to reinfection?
What portion of the bones of the face is most frequently affected by the disease?
What is the result of disease in this portion of the superior maxillary bone?
What may be the result of tuberculosis in the malar bone?
How may the bones of the nose become infected?
What is the result of the disease in these bones?
Is the disease found in the hard palate?
How may it be diagnosed from syphilitic disease?
How may the alveolar process become infected?
What is the general treatment of osteo-tuberculosis?
What is the local treatment of the disease in the bones of the face?
Upon what does success largely depend?
What is the danger of delaying an operation after the diagnosis is established?
What instruments are considered the best for these operations?
What precaution should not be neglected as a final part of the operation?
How should the cavity be treated?
How does Senn treat such cavities in the long bones?
What is the suggestion as to its applicability in cavities in the bones of the face?
How should a tubercular abscess of the face be treated?

CHAPTER XXXII.

SURGICAL TUBERCULOSIS (Continued).

Tuberculosis of the Skin and Mucous Membrane.

Tuberculosis of the Skin, or Lupus Vulgaris.—Until the positive demonstration by Koch that lupus was a form of tuberculosis of the skin, there was a very wide difference of opinion among the various authorities as to the real nature and origin of the disease. The French and English authors were quite generally agreed that it was one of the manifestations of scrofula, and that it was composed of granulation-tissue. The German authorities differed very greatly as to the causation of the affection. Virchow did not believe it to be a manifestation of scrofula, and classed it with the granulomata. Heuter considered it to be a *fungous* inflammation, and that the specific cause was capable of producing miliary tuberculosis when introduced into the tissues. Volkmann classed it with those diseases which are characterized by the production of granulation-tissue. Baumgarten affirmed that the absence of caseous material in lupus was an evidence of its non-tuberculous character, while Friedlander stoutly maintained that lupus was a tubercular disease of the skin, identical in its histologic structure with other forms of the affection, and presenting the same characteristic miliary tubercles.

At the present time there seems to be no doubt that lupus, and many other forms of skin-disease, are tubercular in their nature, and directly caused by the presence in the tissues of the *Bacillus tuberculosis*.

Koch not only demonstrated the presence of the bacillus in lupus nodules, but he succeeded in producing a pure culture of the microbe from lupus tissue, which in every respect resembled that produced from recognized tubercular tissue, while with the fifteenth generation of the bacillus from this culture he successfully inoculated five guinea-pigs by subcutaneous injection, producing typical tuberculosis in each of them.

Before this time, however, clinical observation and the accumulation of anatomical proofs had demonstrated that in all probability lupoid affections were of tubercular origin, or that there was a very close relation between them and tuberculosis, but the positive proofs

were lacking until the discovery of Koch. The experiments of Koch have since been repeated many times by other scientists to prove the identity of the bacillus found in lupus tissue and tuberculosis, with almost uniformly positive results.

Tuberculosis of the skin is often associated with other forms of tuberculosis. Brock found 79 per cent. of the cases examined by him were complicated with other forms of tuberculosis. Rassdnitz found that 30 per cent. out of two hundred and nine cases were associated with other manifestations of tubercular disease. Bessnier reported that 21 per cent. of the cases of lupus that came under his observation eventually died of phthisis. Pontoppindan said that 50 to 75 per cent. of his patients suffering from lupus gave additional evidence of other forms of tuberculosis.

Tuberculosis of the skin may be *primary* or *secondary* in its origin.

All forms of primary tubercular disease of the skin are doubtless the result of inoculation with the *Bacillus tuberculosis*. It is somewhat remarkable, however, that taking into account the frequency with which abrasions and slight wounds occur upon the exposed portions of the skin, and the many ways in which the dangers of infection with the tubercular virus are presented, the primary form of the disease does not occur with much greater frequency.

It is a well-known fact that lupus occurs most frequently upon those parts of the body which are most constantly exposed to injury and infection. Lupus is found most frequently in the skin of the nose, face, eyelids, ears, and hands, locations which are not afforded protection of either the hair or clothing, and which are constantly exposed to slight injuries, to the lodgment of bacilli floating in the atmosphere, and to direct inoculation with the virus from almost innumerable sources. The secondary form of the disease is usually found in patients suffering from advanced tuberculosis, and is a manifestation of a general diffusion of the affection to the skin and mucous membrane, or of auto-infection in persons suffering from primary tuberculosis of the lungs.

Pathology.—As primary tuberculosis of the skin is always the direct result of inoculation, the pathologic changes are therefore always first made manifest at the point of infection. These manifestations consist of the formation of nodules which contain all of the histologic elements of true tubercular nodules, viz: giant cells, epithelioid cells, leucocytes, and the *Bacillus tuberculosis;* caseous material is, however, rarely found. This is accounted for, in all probability, as suggested by Senn, from the location of the tubercular product so near to the surface of the skin, and also because the granulation-tissue soon becomes the seat of suppuration, due to secondary infection from the pus-microbes. By the aggregation of these nodules, and the infiltration

of the surrounding cellular tissues, the lesion gradually spreads, and by the coalescence of the infiltrated portions there is established a more or less extensive area of tubercular tissue.

In those cases where the break in the continuity of the tissue at the point of the infection has been restored, the cell proliferation may be so abundant as to cause a swelling resembling a papillomatous growth and covered with a scaly epidermis, the result of excessive formation and exfoliation of epidermal tissue. Whenever the underlying granulation-tissue becomes exposed, septic infection immediately takes place from the introduction of the pus-microbes, and the process of destruction of the granulation-tissues is hastened by the action of the septic organisms and their ptomaines. Ulceration immediately takes place, the break in the continuity of the skin increases in size and rapidity, commensurate with the formation of granulation-tissue by the action of the *Bacillus tuberculosis* and the development of new nodules in the immediate vicinity of the ulceration.

In some forms of lupus the infection remains superficial, and only the outer layers of the skin become involved; in others the destructive process strikes deeper and deeper, involving the muscles, fascia, periosteum, and bone, simulating very closely the clinical features of malignant neoplasms. This form of the disease not infrequently attacks the face, destroying the nose, eyelids, lips, and a greater portion of the cheeks, leaving the face much like that of a skeleton.

Symptoms and Diagnosis.—Tuberculosis of the skin is found most frequently in middle life; no age, however, is exempt from it. It is occasionally found in little children and persons of advanced age.

The disease is usually described as a neoplastic affection of the skin, or of the contiguous mucous membrane, of highly chronic character and type, manifested in the form of slowly developing small reddish-brown or yellowish-red nodules, or centers of infiltration. These nodules, when further developed, tend to rise above the surface, and form papules or tubercles. The spread of the disease is by peripheral extension and the formation of new centers, while the older ones disappear by gradual resorption, or ulceration takes place, resulting in the formation of disfiguring scars.

The varying degree to which the corium and the papillary layers of the skin are involved gives rise to the differences in the clinical appearances of the disease, and the terms applied to them. The ordinary classification is as follows:

Lupus Maculosus.
Lupus Exfoliativus.
Lupus Exulcerans.
Lupus Serpiginosus.
Lupus Hypertrophicus.

Another classification of the various forms of the disease, also based upon the clinical appearances, is as follows:

Non-ulcerative, or *lupus non-excedens;*

Ulcerative, or *lupus excedens;*

Exfoliative, or *lupus exfoliativus;*

Hypertrophic, or *lupus hypertrophicus* (Wagner).

Lupus Maculosus is characterized by the formation in the skin of minute yellow-brown nodules or patches, usually of pin-head size, more or less transparent, and covered with epidermis. The color is changed to a lighter shade under pressure. The nodules appear to lie just beneath the surface, their outline being well defined. The epidermis covering the patch is usually smooth, but it is occasionally scaly or shiny. The papules commonly appear in clusters, and as they grow they approach one another, finally becoming confluent and forming nodules of considerable size.

The most characteristic feature of the lupus patch is its soft consistence. It is much less firm than the surrounding skin, and offers little resistance to the end of a blunt probe when pressed upon it. The normal skin will entirely resist such pressure, while the lupus patch gives way and the probe is buried in the mass. Lupus maculosus is the simple form of the affection, and is always the first stage of the disease. It also appears at the periphery of old patches, and is often the first indication of a relapse in old cicatrices. This constitutes lupus non-excedens, or the non-ulcerative variety. When ulceration does not take place, the nodules may remain stationary for an indefinite period, or a spontaneous cure may take place by cicatrization.

Lupus Exfoliativus is a later stage in the progress of the disease, characterized by central degeneration of the matured nodule, caseous change, and cicatrization. The skin becomes rough, scaly, and fissured; exfoliation takes place, leaving the skin considerably thinned or atrophied, which thus easily becomes folded or wrinkled.

Lupus Exulcerans. Occasionally the lupus process terminates by a sort of subcutaneous cicatrization. Usually, however, the disease progresses to ulceration. Before ulceration takes place the surface is usually covered with thickened epidermis, which can be scraped off in white scales. Ulceration begins over the center of the nodule, and extends toward the periphery, attacking the new nodules almost as rapidly as they are formed. The ulcerative process is hastened by the secondary infection with the pus-producing micro-organisms, which enter the granulation-tissue at the border of the ulcer. Repair by cicatrization and the ulcerative process often go on at the same time in a lupus patch. Repair is more likely to occur if the tubercular process has been confined to the skin, than when it has progressed beyond this tissue. This constitutes the ulcerative variety, or *lupus excedens.*

Lupus Serpiginosus is but another form of *lupus excedens*, in which the process of repair by cicatrization and epidermization progresses in an irregular form. Healing may take place in the center of a lupus patch, or in a segment of the periphery, while at other points the morbid process continues, and the disease creeps on, followed by the scar, and giving rise to irregular gyrate forms. When the ulcerative process accomplishes its work of destruction with greatest rapidity, penetrating to muscle and bone, and destroying them, it is termed *lupus vorax*.

Lupus Hypertrophicus is a form of the disease in which there is an exuberant formation of tissue which produces a papillary growth. These papillary growths are probably derived from the granulation-tissue which has been covered by epithelium in the process of healing, and may remain as permanent warty growths, or at other times become soft and fungous, with a tendency to bleed. When this form of the disease is located in the lower extremities, the formation of hypertrophied tissue is sometimes so excessive as to cause a very considerable enlargement of the limbs, producing a species of elephantiasis. This latter form never remains as a permanent condition, but sooner or later, sometimes after years, the hypertrophied tissue breaks down, followed by ulceration and cicatrization.

Tuberculosis of the Skin of the Face.—The first manifestations of lupus in the face are the so-called primary efflorescences found upon one or both cheeks, upon the nose, or upon the cheek and nose, in the form of a dull-colored maculation upon the skin, often unnoticed for a long time; or it may appear in the form of a minute nodule; or a thickened purplish patch, the size of the finger-nail. The disease spreads, as already described, by extension from a single patch, or by multiple lesions. The contraction of the cicatrices formed by the process of healing often results in great disfigurement of the face, in some cases causing ectropion of the eyelid or lip. The nose often becomes very much reduced in size after the ravages of the disease have subsided, the point being markedly sharpened, though occasionally, according to Hyde, the point becomes bulbous, flattened, livid, and knobbed, with a thickened septum and distorted alæ.

The upper lip is frequently involved when the disease is situated upon the nose, marked at first by considerable swelling, followed by fissures which are prone to bleed and the formation of crusts on the granulating surface. Considerable deformity usually follows the healing of tubercular ulceration of the upper lip, the mouth being sometimes reduced to a mere slit or hole in the face, with little power to open or close it.

Tuberculosis of the Mucous Membrane of the Mouth.—Tubercular disease of the mucous membrane of the mouth is generally

found as an extension of the affection from the neighboring infected integument. In the great majority of cases it is secondary to tuberculosis of the skin, proceeding from this tissue to the mouth, extending to the mucous lining of the lip, the gum, the hard palate and the velum palati, or the pharynx, or the conjunctiva. Primary tuberculosis of the mucous membrane is comparatively of rare occurrence. When it is remembered that the oral cavity is often the seat of superficial injuries and pathologic changes which form excellent points for infection with the bacillus, it seems a wonder that the primary form of the disease is not much more prevalent in this locality than it is.

The changes which take place in the mucous membrane are the same as when the disease is located in the skin.

The lupus nodules, as found in the mucous membrane, are minute white points, set in the livid red and slightly thickened membrane; they may assume the form of a papillary outgrowth or a granulating patch, which may ulcerate and cicatrize. Ulceration is an earlier and more frequent symptom in tuberculosis of the oral cavity than in other locations, on account of the constant maceration of the newly-formed abnormal tissue by the fluids of the mouth. The ulcerating patch has well-defined borders, and is usually covered by a whitish film or false membrane, produced by the death of the superficial layers of the mucous membrane. On removal of this pseudo-membrane the characteristic granulating surface is exposed. Caseation is seldom seen. Ulceration and cicatrization sometimes cause serious deformities which interfere with the proper function of the parts.

The most characteristic feature of tubercular ulcer of the mucous membrane of the mouth and the tongue is the presence of minute tubercular nodules in the margins and underneath the layer of granulations, and if the infection has extended to some distance, in the surrounding mucous membrane also. (Senn.)

The disease is most often seen in persons from forty to fifty years of age, and rarely attacks the very young.

Tuberculosis of the Tongue and Pharynx may be seen independently of the disease in the skin, and may be primary or secondary in its origin.

Tubercular ulcers of the tongue are exceedingly rare. Butlin says the disease is so uncommon that at intervals it excites an entirely new interest, and is described almost as if it were a new disorder. These ulcers are most often situated upon or near the tip of the tongue, though they are found in all locations, especially upon the dorsum. Men are more prone to the disease than women, and adults more than children. The ulcer (or ulcers, for there may be more than one) is at first indolent, not painful or very tender, but later, as the disease takes on a more active progression, it becomes more and more painful, ex-

ceedingly sensitive, and salivation becomes a marked symptom. Fig. 118 represents a typical tubercular ulceration of the tongue. In some cases the tongue is rapidly destroyed, the lymphatic glands becoming infected, and as the sore extends the strength of the patient fails, death resulting in a few months, or at the end of a year or two. Fig. 119 shows a tubercular nodule from the same case with giant cells and beginning caseation.

FIG. 118.

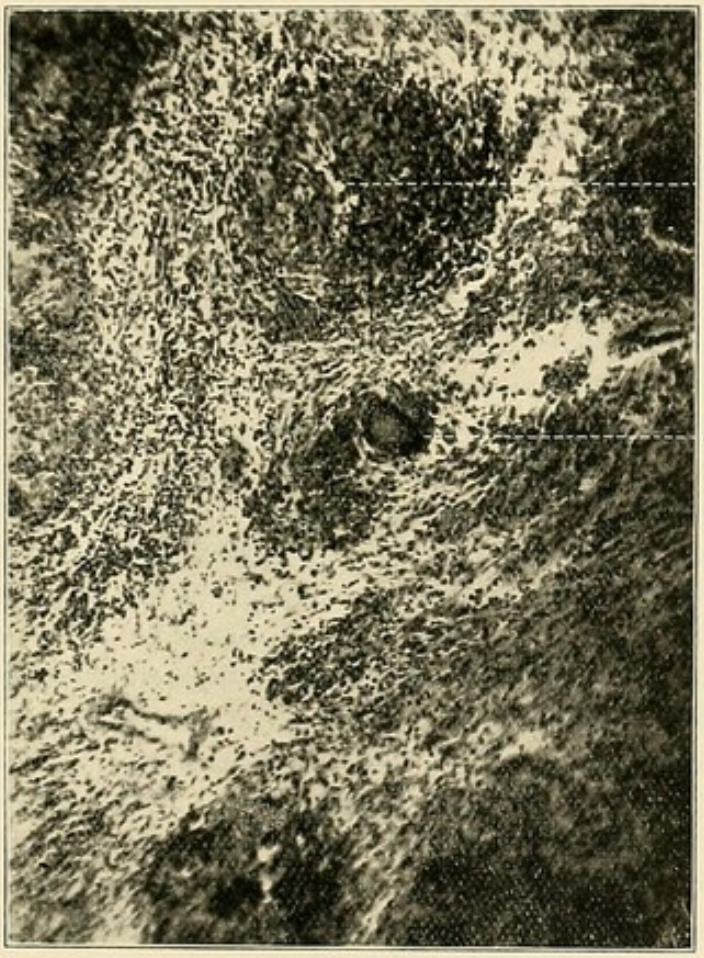


TUBERCULOUS ULCER OF THE TONGUE. × 50.

Tubercular disease of the pharynx may extend to the tonsils and velum palati, destroying these organs, or if cicatrization takes place the posterior nares may become more or less contracted, and the exercise of function become greatly hindered, speech also being rendered imperfect. The larynx may also become involved from extension of the disease, and aphonia result from implication of the epiglottis and the vocal cords.

Differential Diagnosis.—In the diagnosis of lupus of the face and mucous membrane, it must be borne in mind that the clinical features of certain forms of tertiary syphilis and epithelioma closely simulate those of tuberculosis of the skin and mucous membrane, and to differentiate them is sometimes very difficult or well-nigh impossible. Even the microscope in the hands of an expert cannot always be relied upon as an exclusive means of diagnosis, on account of the great similarity in the histologic elements of a tubercular nodule and a gumma.

FIG. 119.



TUBERCULOSIS OF TONGUE, SHOWING GIANT CELLS AND CASEATION. × 50.

A careful examination into the history of the case is of the greatest importance when trying to differentiate between tuberculosis and syphilis, for even though the patient may be sure that syphilis has not been acquired, still it is possible that the disease may have been inherited; while on the other hand, although a positive history of primary and secondary syphilis may have been established, it is not improbable that the manifestations may be those of tuberculosis.

When doubt exists as to the true nature of the disease, the matter may be cleared up in the course of a month or six weeks by prescrib-

ing anti-syphilitic treatment, and at the same time inoculating several guinea-pigs or rabbits, after the method of Koch, by implanting subcutaneously small fragments from the diseased area, as these animals are very susceptible to tuberculosis.

The doubt is removed either by the improvement manifest in the ulceration, after two or three weeks, as a result of the anti-syphilitic treatment, or by the production of tuberculosis in the inoculated animals, and their death at the end of five or six weeks. If the fragments which have been implanted are from syphilitic ulcers, it will have no effect upon these animals, as they cannot be inoculated with syphilis. In differentiating between tuberculosis and epithelioma, the microscope is the only reliable means of diagnosis. A section of a tubercular nodule shows a fine, delicate reticulum, the meshes of which are occupied by granulation-cells; in epithelioma there is a well-marked reticulum, the alveolar spaces of which are filled with embryonal epithelial cells, arranged concentrically. Blood-vessels are also abundant in epithelioma, and absent in the tubercle nodule.

Glandular infection is an early manifestation in epithelioma, while in tubercular ulcerations of the mucous membrane it is a late manifestation or may not occur at all.

Simple ulcers of the cheek and tongue sometimes occur from the mechanical irritation of a sharp or jagged tooth, resulting from a carious cavity, or from masses of salivary calculus, or from a misplaced tooth. Such ulcerations are easily recognized from their location and appearance, and only a careless observer would be misled. It should be borne in mind, however, that they may become the focus of infection of tuberculosis, or the starting point of a carcinomatous growth.

Prognosis.—Although lupus is usually confined to the skin, it may attack deeper parts, involving the muscles and periosteum, causing necrosis of the bone. Primary tuberculosis of the skin may lead to infection of the lymphatic glands nearest to the seat of the disease, and eventually to general miliary tuberculosis. Pulmonary tuberculosis often develops as a secondary complication. It occasionally happens that a lupus patch is the cause which locates the formation of a carcinoma. The tendency to local extension varies greatly. In some cases the disease may begin in early life, remain stationary for a number of years, then suddenly become very active and not confined to the skin, but attacking the deeper tissues and destroying them with the greatest rapidity, regardless of their structure. In tuberculosis of the face, the tendency is toward rapid extension; in some cases the soft tissues and the superficial bones may be completely destroyed in a few months. On the other hand, the process of repair by cicatrization follows closely upon the destruction of tissue, and extensive scars are formed, causing frightful deformity.

The prognosis, so far as the life of the patient is concerned, is favorable so long as the disease remains local, or does not progress more rapidly than the process of repair. Regional infection of the lymphatic glands is always considered as a menace to life, as sooner or later important internal organs are affected, or miliary tuberculosis becomes general. A spontaneous cure is sometimes effected. The disease, however, is prone to recurrence in the scar tissue.

The prognosis of tubercular ulceration of the mucous membrane of the mouth is usually favorable, except when located in the tongue. When associated with generalized tuberculosis, or cachexia, as occasionally happens, the prognosis is exceedingly unfavorable. The destruction of the membrane is sometimes very extensive, and the resulting cicatrix, by its contraction and adhesions, causes unsightly deformity, often greatly interfering with the function of the parts.

The prognosis of tubercular ulcer of the tongue is almost as bad as in carcinoma. The disease is not only fatal, but the lease of life is usually short, the end coming in a few months, or in a year or two at the longest. The pain and distress which accompany the downward course of the disease is very great, while the patient is considered as fortunate if the end is hastened by the presence of a rapidly-progressive tuberculosis of some important internal organ, which produces a fatal termination before the ulcer of the tongue becomes large and painful. Occasionally tubercular ulcers of the tongue heal, but the cure is usually only a temporary affair, for sooner or later the disease returns, the second outbreak being more rapid in its course, and all efforts for its cure are unavailing. (Butlin.)

Treatment.—The internal treatment of tubercular disease of the skin and the mucous membrane should be governed by the indications of the patient. There is no known remedy that has any specific action in curing the disease, or, according to Hyde, that is capable of relieving the victim of his local ailment. Recent authors think the only remedy that deserves any confidence is arsenic, in the form of Fowler's solution. It is administered in doses of from three to ten drops after meals, diluted with water, beginning with the smallest dose and gradually increasing until the maximum dose is reached or the physiologic effect is produced, and then gradually diminishing. To be of any real value its use must be continued for several weeks or months.

Cod-liver oil, the tincture of chlorid of iron, the bitter tonics, combined with nutritious diet, out-door exercise and sea-bathing, are the most useful agents in sustaining the general health, assisting nature to limit the spread of the disease and favor the process of repair.

The local treatment consists of the removal of the diseased tissue by surgical operation, under anesthetics; to be efficient it must be thorough; half-way measures are of no more real value here than they

would be in the treatment of malignant neoplasms, for the disease is almost sure to recur unless every particle of infected tissue is removed.

The use of caustics is generaliy of no real value, often positively harmful.

Since the nature of the disease has been recognized, antiseptic agents have been recommended for iocal treatment. White uses the bichlorid of mercury, one to two grains to an ounce of water, applied for half an hour morning and evening on compresses kept wet with this solution, or an ointment made from the same drug, two grains to the ounce, applied continuously, and changed morning and evening. Care must be taken that salivation is not produced by absorption of the drug. He also obtained satisfactory results by the application of a 2 to 4 per cent. solution of salicylic acid in castor oil. Iodoform in the form of the powder, ointment, or emulsion in glycerol is recommended as one of the very best antiseptics in all forms of tubercular disease.

Balsam of Peru is also of benefit as a local application.

Dr. Thomas S. K. Morton, of Philadelphia, recommends acetanilid as an antiseptic dressing in all surgical wounds, tuberculous ulcerations, and bone-disease, either in substance or as gauze, or ointment (1 in 8), or dissolved in alcohol or oil (as an injection). A 10 per cent. solution in water seems to answer every purpose in preventing suppuration in all surgical cases, while it seems to act better than iodoform in the treatment of tuberculous lesions. Care must be exercised in the use of the drug in substance to prevent toxic symptoms.

Antiseptic agents, however, can never become efficient means of treatment in this affection, for the reason that they cannot be brought into direct contact—except at the surface and for a little distance beneath—with the bacilli and those parts in an active state of disease. In those cases in which a radical operation is declined by the patient or friends, the above treatment is the next best means at the disposal of the surgeon.

Excision is the most effectual form of radical treatment, but unfortunately it cannot be performed in all cases.

This operation is not admissible when the disease is upon the nose or some other prominent part of the face where the resultant deformity would be nearly as unwelcome to the sufferer as the disease, nor where the disease is very extensive.

When excision is practiced, the lines of incision should be made at some distance from the margins of the visible diseased area, in order that all infected tissue may be included; while great care should be exercised in removing the deeper portions of the infiltration, as this may send out projections at various points, which must be extirpated in order to insure non-recurrence. If it becomes necessary to remove

extensive portions of the skin, the gap may be filled by the Thiersch method of skin-grafting.

Another method of radical operation is by curetting. This operation consists of scraping out the diseased tissue by means of sharp spoons or curettes. Lupus tissue is much softer than the healthy skin, consequently the curette easily penetrates the former, while the latter offers sufficient resistance to guide the operator in removing the diseased tissue.

Bessnier recommends the use of the galvano-cautery for removing the diseased tissue, and he has devised special cautery points for this purpose.

The writer believes the most effectual plan of treatment is a combination of the last two methods, the bulk of the diseased tissue being first removed by the curette and then followed by the galvano-cautery. The storage battery makes it possible for every surgeon to use Bessnier's cautery point and knives. No more successful method can be used to follow up and remove the more minute points of the disease which have been left behind by the other operations of excision and curetting.

Antiseptic after-treatment is very desirable, and the patient should be kept under observation for some time after the healing of the wound. The site of the operation should be protected from injury for several months, as a precaution against the pathogenic action of remaining latent bacilli or reinfection from the outside.

The treatment of the disease located in the mucous membrane of the mouth should be upon the same general principles as when located in the integument. The curette and galvano-cautery are most applicable for radical treatment.

Excision, if practiced early, is the most effectual treatment in tubercular ulceration of the tongue. This may be accomplished by removing a wedge-shaped piece with the knife, and stitching the surface together, or it may be removed with the écraseur or the galvano-cautery. Methods of operating will be found described in the chapter which deals with carcinoma of the tongue. Recurrence, however, is the rule after operations for this disease when located in the tongue.

REVIEW.

CHAPTER XXXII.

What did Koch demonstrate in reference to the nature of lupus vulgaris?
What were the previous opinions as to its nature?
What is the cause of tuberculosis of the skin?
How did Koch demonstrate this?
Is lupus associated with other forms of tubercular disease?
How does the disease originate?
How does primary lupus originate?
How does secondary lupus originate?
Does lupus differ in its histologic pathology from other forms of tuberculosis?
Does caseation take place?
Why is caseation so seldom found?
How does the disease spread?
How are the warty growths of lupus formed?
What occurs when the granulation-tissue becomes exposed?
What governs the rapidity of the extension in the ulcerative process?
What characterizes the more severe or malignant type of lupus?
At what period of life is lupus most common?
Does it ever occur at the opposite extremes of age?
What are the symptoms and diagnostic features of the disease?
What gives rise to the various terms applied to lupus?
What is the ordinary classification of the disease?
Describe lupus maculosus.
What is the most characteristic feature of lupus?
How may this softness be demonstrated?
Is this form always the first stage of the disease?
What does it indicate when found in old cicatrices?
What other name is applied to this form of the disease?
Describe lupus exfoliativus.
Describe lupus exulcerans.
What form of secondary infection hastens the ulcerative process?
What two processes sometimes go on together in the same lupus patch?
Under what conditions is repair most likely to occur?
What other name is applied to this form of the disease?
Describe lupus serpiginosus.
What term is applied to the disease when the ulceration is rapid and penetrates to the deeper tissues?
Describe lupus hypertrophicus.
From what are the papillary growths derived?
What becomes of these growths?
How is the disease manifested in the lower extremities?
What are the manifestations of lupus of the face?
What is the result of lupus of the nose?
What are the manifestations of the disease in the upper lip?
What is sometimes the result of the disease in the lip?
What is the usual cause of tuberculosis of the mucous membrane of the mouth?

Is the primary form of the disease common or rare?

Is there any difference in the pathology of the disease as found in the mucous membrane and the skin?

What are the diagnostic signs of the disease in the mouth?

Is ulceration an early or a late symptom?

What is the character of the borders of the ulcerating patch?

Is caseation a feature in this location?

What frequently results from the formation of large cicatrices?

What is the most characteristic feature of lupus of this membrane?

At what age is the disease most often seen?

Is the disease ever found in the tongue and pharynx independently of the disease in the skin?

Is the disease in the tongue common?

Where is it usually located?

In which sex does it most often occur?

Does it occur most often in adults or children?

Describe the course of the disease in the tongue.

What is the result of the disease in the pharynx?

What diseases simulate lupus in the skin and mucous membrane?

Is there any difficulty in differentiating them?

What are the difficulties?

How may the diagnosis be established between lupus and syphilis?

How may the diagnosis be established between lupus and epithelioma?

What are the histologic differences?

How are the lymphatics affected in each?

What other form of ulceration may be mistaken for these diseases by a careless observer?

What is the prognosis in lupus of the skin?

What is the tendency in lupus of the face?

What is the result when repair takes place?

What is the prognosis in tubercular disease of the mucous membrane?

Under what conditions would it be unfavorable?

What is the prognosis when the disease is located in the tongue?

Does a permanent cure ever take place?

What is the internal treatment for the disease?

What drugs, if any, are valuable in this disease?

What are the methods of local treatment?

Detail the antiseptic treatment.

What are the methods of radical treatment?

Which method is the most effectual?

Where is this method inadmissible?

Describe the method of excision.

How may the gap be filled?

Describe the method of curetting.

What other means may be used to accomplish the same purpose?

What is the writer's recommendation?

What should be the after-treatment?

Which method is most applicable in lupus of the mucous membrane?

Which method will give the best results in treating the disease in the tongue?

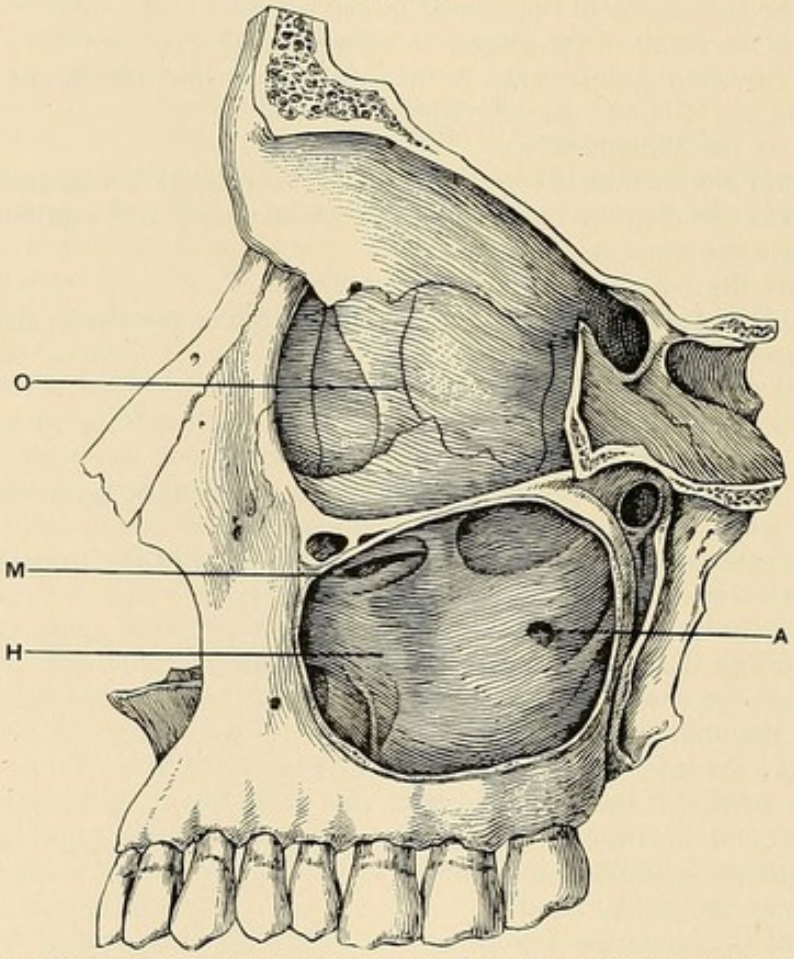
What other methods can be utilized?

CHAPTER XXXIII.

DISEASES OF THE MAXILLARY SINUS.

THE *Maxillary Sinus,* or *Antrum of Highmore,* is a cavity in the body of the superior maxillary bone, somewhat pyramidal in form, having its base at the nasal fossa, and its apex directed toward the maxil-

FIG. 120.



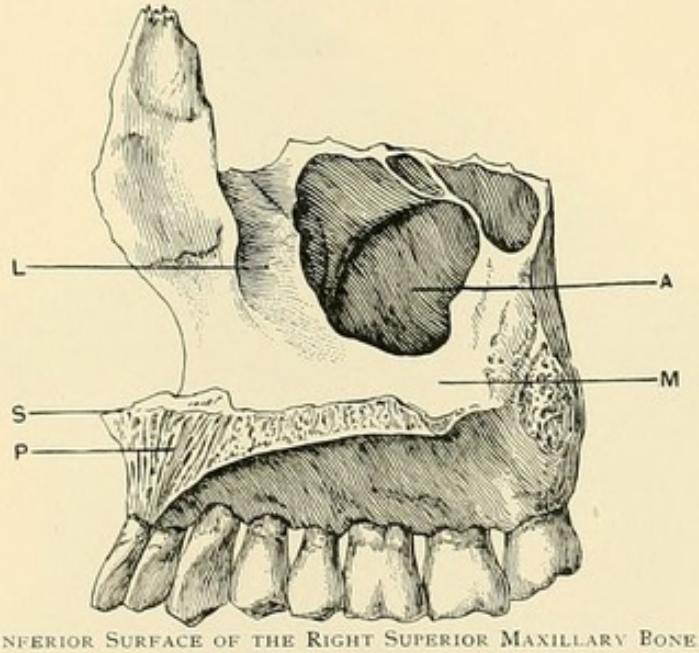
LEFT SUPERIOR MAXILLARY BONE, EXHIBITING THE COMMUNICATIONS BETWEEN ANTRUM AND NASAL CAVITY. (After Zuckerkandl.)

O, orbital cavity; H, maxillary cavity of antrum of Highmore; M, slit-like opening osteum maxillare; A, accessory opening between antrum and nasal cavity.

lary tuberosity. (Fig. 120.) It communicates with the nasal cavity by an irregular opening in the external wall of the middle meatus. Through this opening the mucous membrane of the nasal cavity, the Schneiderian membrane, passes to line the sinus.

The maxillary sinus has five walls; an internal, which is the lateral wall of the nasal cavity and forms the base of the pyramid (Fig. 121); an antero-external, which is the antero-external portion of the body of the superior maxillary bone; a superior, which is the floor of the orbit; a posterior, which is that portion of the superior maxillary bone that articulates with the pterygoid process of the sphenoid bone; and an inferior, which is that part of the superior maxillary bone from which arises the alveolar process. The thinnest and most inferior, or dependent portion of this wall, is opposite the alveoli of the second molar tooth. The floor of the antrum has generally an uneven surface, the eminences corresponding to the roots of the teeth. Occasionally the roots of the first and second molars penetrate the floor of the sinus.

FIG. 121.



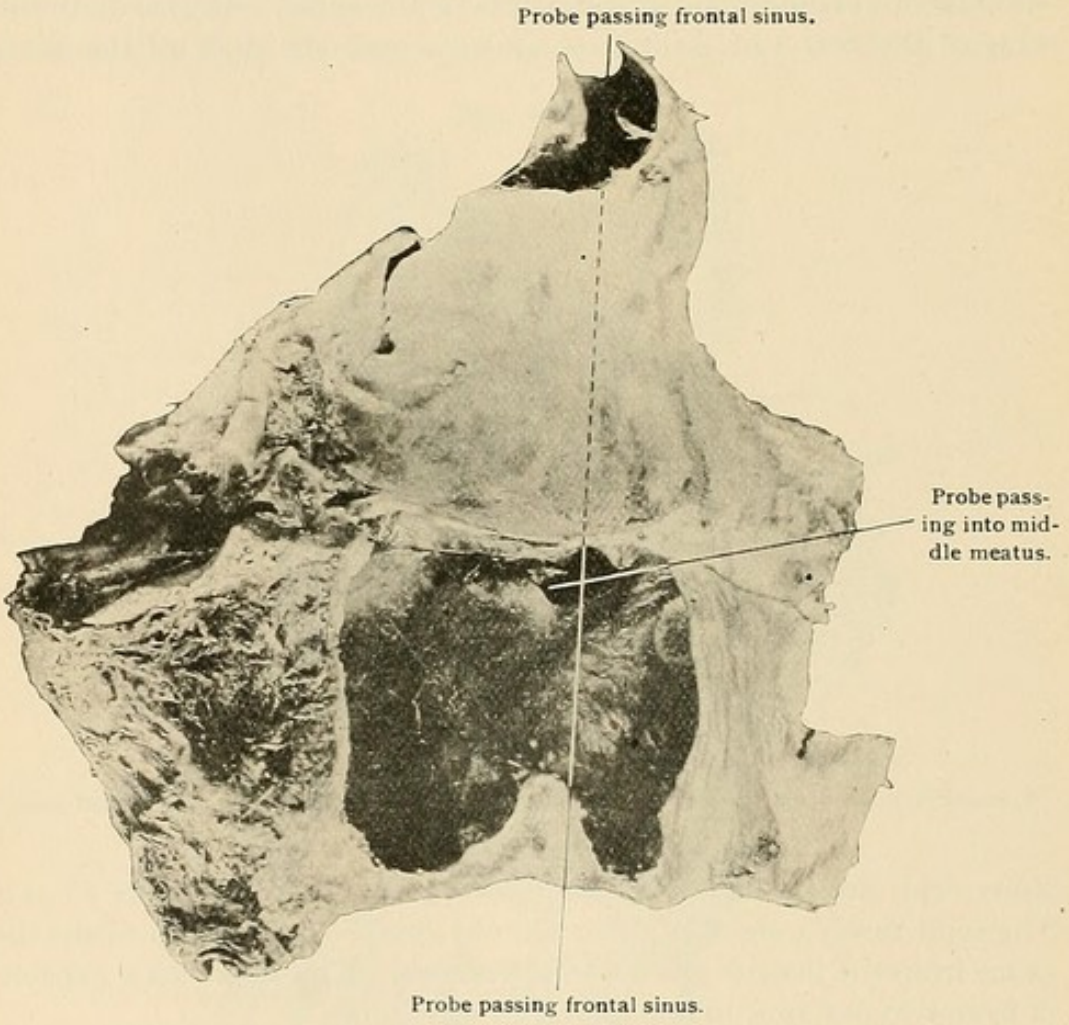
INFERIOR SURFACE OF THE RIGHT SUPERIOR MAXILLARY BONE.
P, palate process; S, anterior nasal spine; M, lower meatus of nasal cavity; L, lachrymal groove; A, antrum of Highmore.

Bony septi are frequently found crossing the floor from side to side. The septi rarely extend higher than one-fourth to one-third of the distance from the floor to the roof of the sinus. The sinus has a capacity of from two drachms to one ounce, or even more.

The other accessory cavities which open into the nasal passages are the frontal and sphenoidal sinuses. The frontal sinuses are two irregular cavities situated between the plates of the frontal bone, on either side of the median line. They are not present in childhood, but are fully developed in adult life. They communicate with the nares by a rounded canal, which opens into the middle meatus, and is called the infundibulum. Cryer has shown, in a paper read before the American Dental Association in 1895, illustrated by the stereopticon and published in the *Dental Cosmos* for January, 1896, that the infundibulum

often discharges directly into the antrum, and in others so near to the osteum maxillare that it might discharge into it. This was a new discovery, and goes far toward an explanation of the difficulties often encountered in the treatment of antral inflammation. In describing this newly-discovered relationship and in explanation of Figs. 122 and 123, he says this "is a sagittal section made near the inner wall of the orbit. The frontal sinus is seen at the top, and below this is the inner wall of the orbit, os planum of the ethmoid, including the edge of the

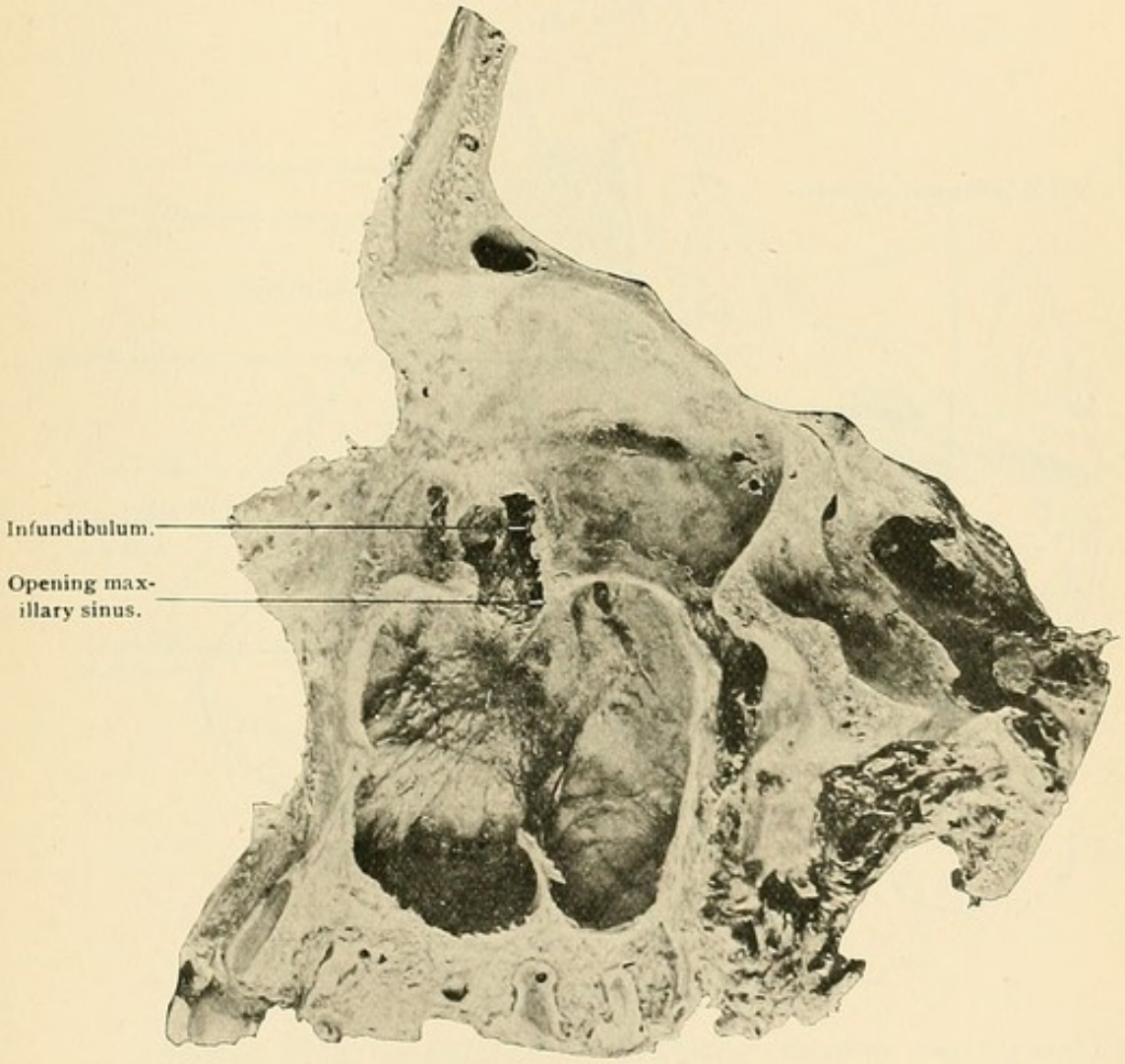
FIG. 122.



inner portion of the floor, below which is the internal wall of the maxillary sinus. . . . To locate the opening and the direction in which the excess of fluid would pass from the sinus, a wire has been passed backward into the hiatus semilunaris. A perpendicular probe passes through the upper portion of the opening of the sinus, which in this case is partly in its roof or at the angle of the internal wall and the roof; as the straight probe passes out of the antrum, it passes through the infundibulum into the frontal sinus, showing that fluids could pass

directly downward from the frontal into the maxillary sinus." These specimens also show "where the anterior ethmoidal cells open just at the maxillary sinus. If the hiatus semilunaris should be closed by pressure of the septum or inflammation of the mucous membrane, the fluïds from the frontal and ethmoidal cells would pass into the antrum." Fillebrown, in a paper read before the American Dental Association in 1896, and published in the *Dental Cosmos* for November, 1896, states

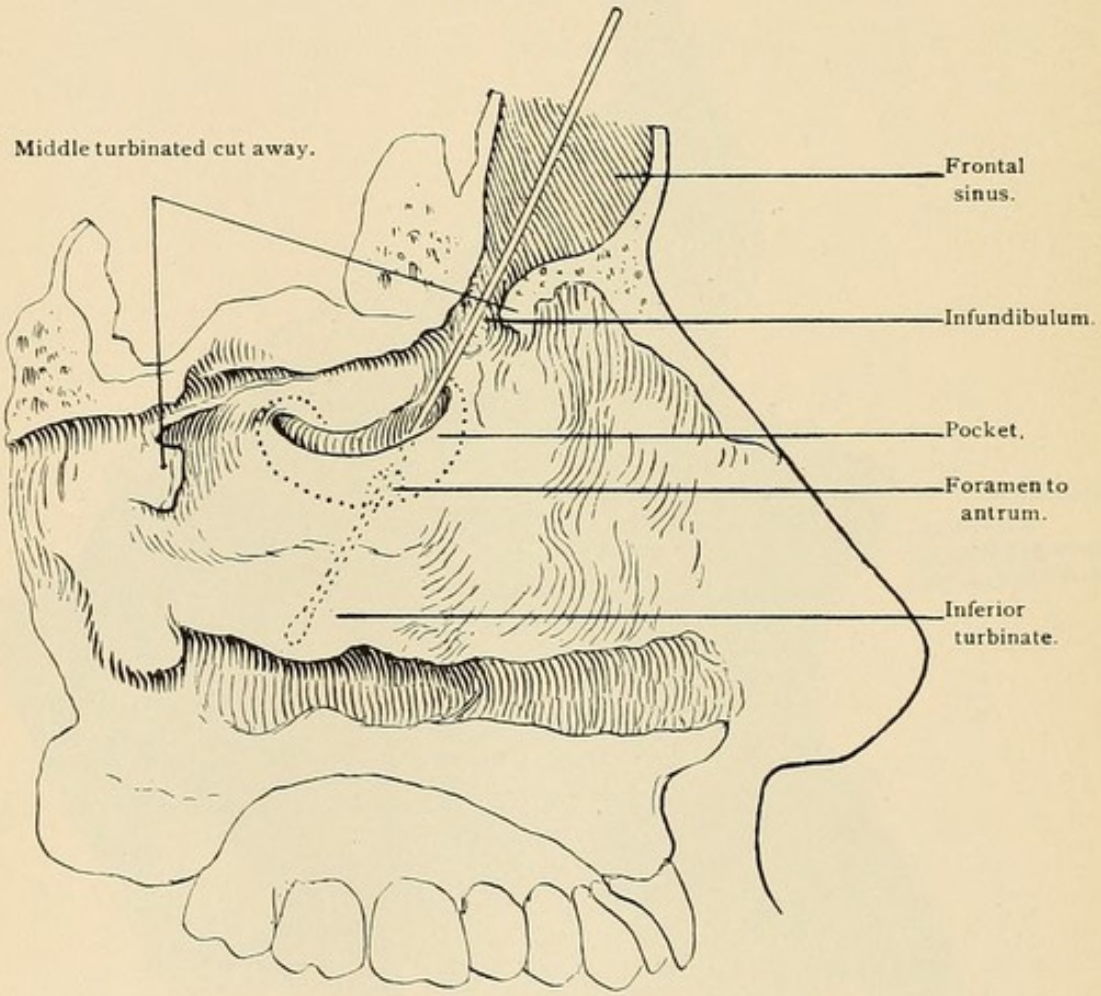
FIG. 123.



it as his opinion that in certain obstinate chronic cases of empyema of the antrum, the frontal sinuses are also affected, and that the inflammatory secretions from these sinuses drain into the antra, and thus keep up the inflammatory conditions of the latter sinuses. But inasmuch as such a supposition was contrary to the accepted opinion upon the subject, and also not in accord with the generally accepted teaching as to the anatomical relations of the parts, he instituted a line of investigation in order to demonstrate the correctness or the possible error

as to the relations of the infundibulum and the point at which it discharged its secretion. The text-books on anatomy state that the infundibulum terminates in the middle meatus of the nose, but Fillebrown's investigations do not bear out the correctness of this statement, for he has found in eight subjects, taken at random, that in every one the frontal sinuses communicated directly with the antra, thus corroborating the investigations of Cryer. He says, "The infundibulum, instead of terminating directly in the middle meatus, continues as a

FIG. 124.



(After Fillebrown.)

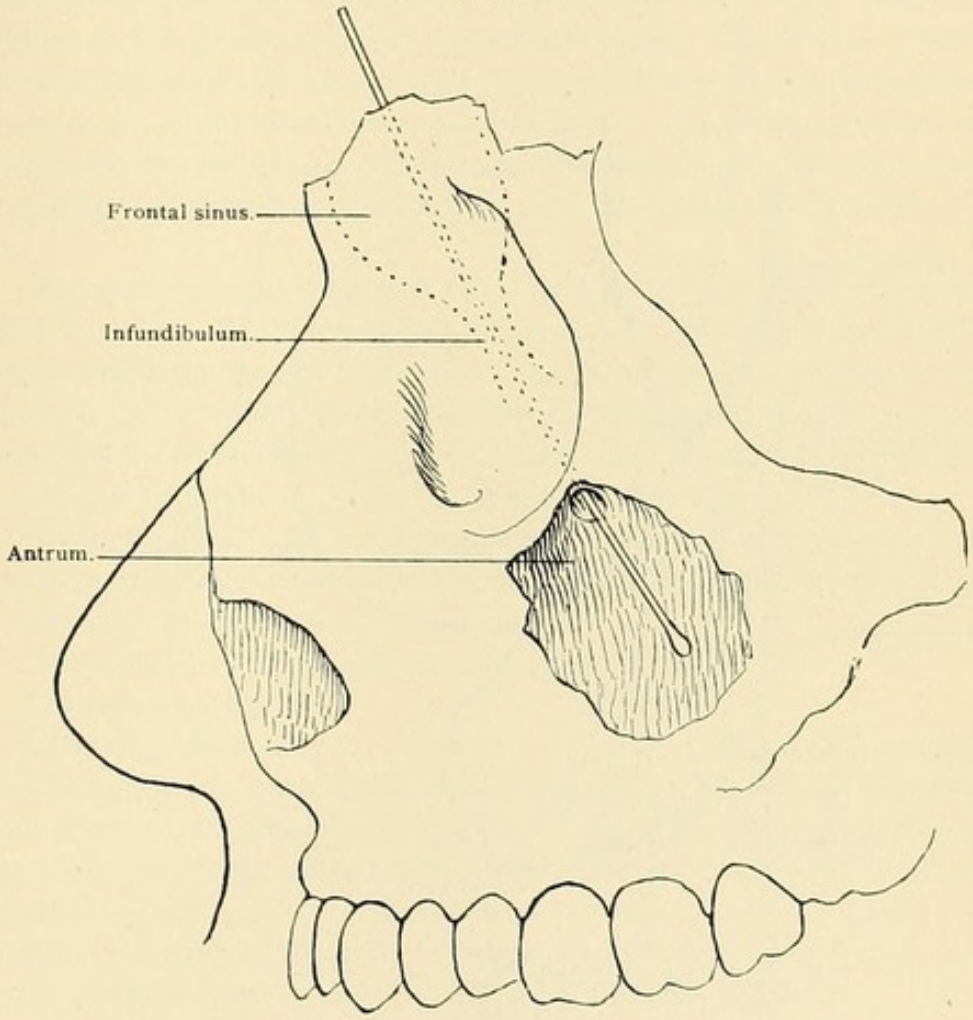
half-tube, this half-tube terminating directly in the foramen of the maxillary sinus. In seven of the specimens there was a fold of mucous membrane which served as a continuation of the unciform process and reached upward, covering the foramen and forming a pocket which effectually prevented any secretion from the frontal sinus getting into the meatus until the antrum and pocket were full to overflowing."

Dr. Fillebrown further thinks that this pocket cannot be an anomaly, as thought by some other observers, as it was found in seven out

of eight subjects, but that its absence might be considered anomalous, rather than the usual type. Figs. 124, 125, 126 illustrate the points made by the author of the paper.

The sphenoidal sinuses are two cavities hollowed out in the body of the sphenoid bone, and are separated from each other upon the median line by a thin lamella of bone. These sinuses are also lined with mucous membrane.

FIG. 125.



(After Fillebrown.)

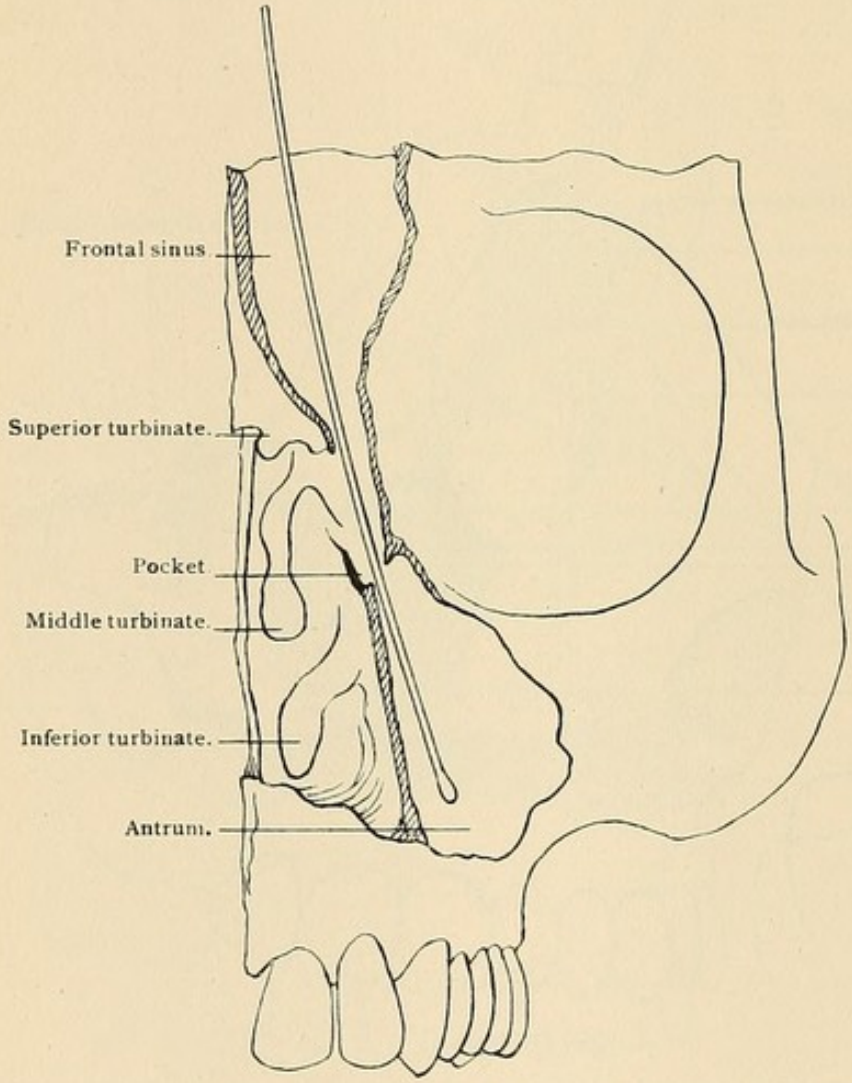
Diseases of the maxillary sinus are quite common, much more so than is generally supposed. These diseases are more common among the lower classes of society, especially those who give no attention to the care of their teeth. Abundant proof of this statement can be found in any of the surgical clinics in the free dispensaries and hospitals of our large cities.

Climatic influences are important factors also in the production of certain forms of antral disease. Mucous engorgements, and empyema, are much more prevalent in damp and changeable climates, where catarrhal conditions abound, than in dry or equable climates.

The diseased conditions most commonly found affecting the maxillary sinus are:

1st. Suppurative inflammation, or purulent empyema.
2d. Mucous engorgements.
3d. Syphilitic ulceration.
4th. Necrosis of the bony walls.
5th. Tumors.

FIG. 126.



(After Fillebrown.)

Suppurative Inflammation of the Maxillary Sinus.—Suppurative inflammation of the antrum is the most common of all diseases affecting this sinus. It is not an idiopathic affection. It may be acute, subacute, or chronic. In the acute form there is rarely any difficulty in making a correct diagnosis, but in the chronic very great difficulty is sometimes encountered, owing to the fact that the symptoms are rarely well marked, and so closely simulate chronic nasal catarrh as to be easily mistaken for that affection. The disease may

be unilateral or bilateral. It is extremely rare that both antra are found affected at the same time. Two cases only of bilateral disease have ever come under the personal observation of the writer. The first was a German woman about forty years of age, who presented herself at the clinic of the Post-Graduate Medical School of Chicago. Both antra were engorged with purulent secretions, the face much swollen in the infraorbital region, the left somewhat more than the right; parchment-like crepitation was present over both antra, and bulging of the palate process upon both sides. There was considerable protrusion of the left eye, but this condition was not so marked in the right; she complained of impaired vision. The superior teeth were all decayed to the gums. This case was due to the infection of the antra with pus from several alveolar abscesses upon both sides of the jaw.

The second was a recent case, in the person of a professional friend, in which the disease followed an attack of *la grippe*, producing a mucous engorgement of both antra, and loss of the voice as a complication. This at least was the opinion of the patient, and it proved to be correct, for immediately after draining the antra the laryngeal symptoms began to improve. This case made a quick recovery.

Etiology.—The causes of suppurative inflammation of the maxillary sinus generally arise from one of the following local conditions:

Diseases of the teeth,

Presence of foreign bodies in the sinus,

Traumatic injuries,

Catarrhal affections.

The diseases of the teeth which may give rise to suppurative inflammation of the antrum are often of a more or less obscure nature, frequently requiring considerable skill in special diagnosis to arrive at a correct solution of the difficulties presented in certain cases. It is therefore no wonder that the general practitioner of medicine or of dentistry should sometimes fail to make a correct diagnosis.

The lesions of the teeth which may be classed as active causes in the production of suppurative inflammation of this sinus are,—

(a) Devitalized pulps.

(b) Alveolar abscesses.

(c) Malposed teeth.

Devitalized Pulps.—A devitalized pulp in the root of a tooth which penetrates the floor of the antrum may give rise to a septic inflammation of the lining membrane of this cavity from the escape of the liquefied and putrescent pulp-tissue and mephitic gases, without giving the least evidence of the real cause of the trouble other than a slight discoloration of the tooth.

Cases of this character are by no means uncommon, while the

obscurity of the cause of the difficulty makes it doubly interesting from the diagnostic point of view. Cases of this obscure nature have frequently come under the observation of the writer, which had been examined by some of the very best general practitioners, without finding the cause. The difficulty in the diagnosis is greatly augmented if there are several devitalized teeth and roots in the affected side of the jaw. The location of the offending tooth then becomes a matter of conjecture, and there is no certainty of making a cure except by the extraction of all teeth which have lost their vitality, that may be associated with the diseased antrum.

Sometimes the offending tooth will be of such good color as to appear to the eye like a living tooth, and thereby be overlooked. There are three methods of diagnosing pulpless or devitalized teeth; one is to reduce the temperature by the application of a piece of ice. If the tooth be vital, the great change in temperature will cause pain; or the temperature may be reduced by throwing upon the suspected tooth a spray of ether. A second is to illuminate the mouth with the electric lamp, the patient being seated in a dark room. The living teeth will transmit the light very readily, the devitalized teeth will not, the difference in the translucency being very marked. A third is to apply the Faradic current. The devitalized teeth will give no response, while the living teeth will be very sensitive to the shock. This latter method has been used by the writer for many years, and experience teaches that it is the most reliable, as he has never failed to make a correct diagnosis by this method.

Alveolar Abscesses.—Devitalized pulps, under ordinary circumstances, if not interfered with surgically, usually result, sooner or later, in the development of alveolar abscesses. A devitalized pulp in the roots of a superior bicuspid or molar, which results in the formation of an alveolar abscess, may on account of the thinness of the floor of the antrum at these locations point into the sinus, and thus establish a suppurative inflammation.

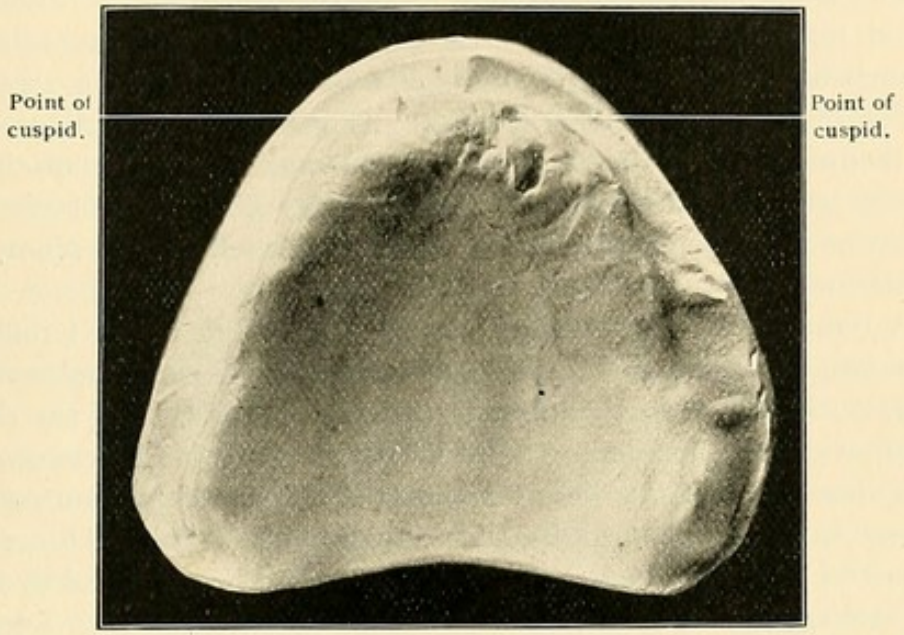
An alveolar abscess which discharges into the antrum of Highmore is the most common factor in the production of suppurative conditions of this sinus. The association between the alveolar abscess and the inflammatory conditions of the sinus is sometimes quite obscure. The patient will frequently give a history of an abscessed tooth which was troublesome for a week or ten days, and then the symptoms subsided. Later, whenever a cold is taken, the tooth is a little tender. Sometimes there is a bad-smelling discharge from the nose as the only symptom. Another case will present all of the characteristic symptoms of the disease, and the offending tooth will be readily recognized.

Devitalized pulps and alveolar abscesses cause suppurative inflammation of the antrum by septic infection, and often produce marked

symptoms during the acute stage of general septic intoxication. When the latter condition is present, it calls for speedy and heroic treatment.

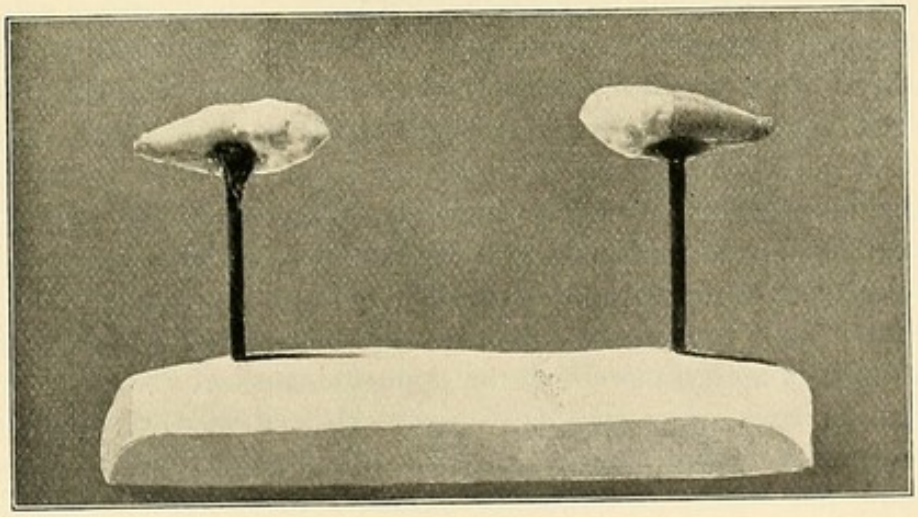
Malposed Teeth.—Malposed teeth are often found in locations where it is impossible for them to take their normal position in the

FIG. 127.



MALPOSITION OF THE CUSPID TEETH IN WOMAN 45 YEARS OF AGE.

FIG. 128.



POSITIONS OCCUPIED BY THE CUSPID TEETH IN THE JAW.

alveolar arch; in fact, they are found in almost every conceivable position, and with every line of inclination. Occasionally they are found lying near the floor of the antrum, in a longitudinal direction, or even

with an upward inclination. Fig. 127 is from a cast showing the malposition of the superior cuspids, which were lying close to the floor of the antrum. Fig. 128 indicates the relative position which they occupied to each other. More rarely they have been found completely inverted. Under these conditions a suppurative inflammation may be established as a direct cause of the irritation produced by the effort on the part of nature to complete the development of the tooth, and force it from its bony crypt. As the pus increases in quantity, the surrounding bony walls grow thinner and eventually give way upon the side offering the least resistance. When this side of least resistance happens to be the floor of the antrum, the pus is discharged into this sinus, establishing an inflammatory condition of its lining mucous membrane, which may go on indefinitely if not relieved by surgical treatment.

A remarkable case of this character, occurring in the family of a professional acquaintance, and coming under the knowledge of the writer, is of interest in this direction. Mrs. G., mother of the doctor, had for sixteen years been troubled with an offensive discharge from the left nostril. Prior to this she had suffered intensely from pain and swelling in the region of the left antrum, which lasted for several weeks, and then subsided. This condition was accompanied by a profuse discharge of offensive secretions from the left nostril. Later the swelling disappeared, but the discharge never wholly ceased. Six years after the first attack, the face again became very painful and much swollen, this time extending farther backward toward the ear, finally developing what was thought to be abscess of the middle ear. Large quantities of pus were discharged from the ear, and at one time it was feared that the suppurative process would extend to the meninges of the brain. After several weeks of intense suffering the symptoms again subsided, and finally the discharge entirely ceased from the ear. After this, however, the discharge from the nose seemed to be increased, and the symptoms were always aggravated by taking cold. The teeth of the left side of the upper jaw had all been extracted as a possible cause of the trouble, except the third molar, which had never erupted. This procedure, however, produced no abatement of the symptoms. Later, the teeth of the opposite side were removed for the purpose of inserting a complete upper artificial denture. About ten years after the last-mentioned attack, while leaning over a washbowl, brushing her teeth, she suddenly noticed that the left nostril was plugged up with some movable body. On throwing the head back, in an attempt to dislodge it, the mass fell into the fauces, and was ejected from the mouth into the bowl. Upon examination, it proved to be a well-developed left superior third molar, more or less covered with hard concretions of a dark brown color. The discharge from the nose

from this time on gradually grew less, and finally, after a few months, ceased altogether.

The probable explanation of the peculiar features of this case are, briefly: First, the third molar was developed in an inverted position, and very near to the floor of the antrum; second, suppurative inflammation was established in the crypt of the tooth-germ from irritation induced by its development in an abnormal position; third, the pus-cavity ruptured into the antrum; fourth, the abscess which ruptured into the middle ear may have been caused by the tooth, though there is some doubt as to this, as the anatomy of the parts would not favor such a supposition. The tooth probably became dislodged at this time, and escaped into the antrum; fifth, at a later period it must have become lodged against the nasal wall of the antrum, causing ulceration, which finally permitted it to pass into the nasal cavity, and thus be expelled.

Foreign Bodies.—Foreign bodies of various materials are occasionally met with in the antrum as a cause of disease. Those most commonly found are the roots of teeth which have slipped from the forceps and escaped into the sinus through an enlarged alveolus in attempting to extract them; malposed teeth which have erupted into it, or have found entrance through pathologic conditions; fragments of bone or of teeth which have been forced into it by some crushing injury of the walls of the antrum; portions of lead or other metal which have lodged there as a result of gunshot injuries; and particles of food which have found entrance through some artificial opening that has been made for the treatment of a pre-existing diseased condition.

The presence of any foreign substance is usually productive of inflammatory conditions of the lining mucous membrane, resulting in subacute or chronic inflammation, with purulent discharge, or of engorgement and protrusion of the walls, with possibly graver consequences.

When there is no positive history of the entrance of such foreign bodies, the diagnosis sometimes becomes exceedingly difficult, and can only be reached by a critical examination of every feature and symptom of the disease, one by one excluding those causes which do not give a marked history, then from the remaining possible etiologic factors, by a still closer analysis and exclusion, arrive at a correct diagnosis.

The diagnosis being assured, operative measures are necessary. These measures comprehend the making of an opening into the antrum, the search for the foreign body, and its removal.

The selection of the point at which to open the antrum should be governed by the surrounding conditions. The unnecessary sacrifice of sound teeth in order to gain easy access to it does not commend

itself to a wise conservatism. In the extraction of teeth for this purpose those should be selected which from their condition and location are of the least value to the individual, provided only that they are in close relation to the floor of the antrum. If a bicuspid or a molar has already been lost, it is preferable to enter the antrum from this point rather than sacrifice remaining sound teeth.

Traumatic Injuries.—Traumatic injuries involving the maxillary sinus are of rare occurrence, and are the result, generally, of gunshot wounds or crushing injuries of the face, causing fracture and comminution of the superior maxillary bones. Cases of this character almost invariably terminate in suppurative inflammation,—at least this has been the observation of the writer. This is explained by the fact that such injuries always produce compound fractures of the bone, and when associated with the oral cavity are always infected from the secretions and alimentary débris, consequently septic inflammation is prone to follow, as is the case with compound fractures of the lower jaw which communicate with the mouth.

Disease of this character is much more amenable to treatment, as a rule, than are those conditions which result from disease of the teeth, for the reason that in the first the inflammation is generally of acute type, while in the latter it is usually subacute or chronic; the character of the infection may also be a modifying factor.

Catarrhal Affections.—Catarrhal affections of the nasal mucous membrane often result in the extension of these conditions to the frontal, sphenoidal, and maxillary sinuses. The mucous membrane lining the accessory cavities is a reflection of the membrane which lines the nasal passages; consequently the inflammatory conditions which affect the mucous tissue of these passages are quite likely to extend to the accessory cavities which open into them through the continuity and functional identity of their lining membrane.

Mucous Engorgements.—Among the most common of the catarrhal inflammations of the nasal passages which may extend to the accessory sinuses and produce mucous engorgements are acute and chronic coryza, and *la grippe* or influenza.

Mucous engorgements, in the opinion of the writer, are much more likely to follow acute catarrhal inflammations than the chronic form.

Acute coryza, or cold in the head, is usually the result of exposure to cold, though it is occasionally due to the irritating effects of acrid vapors, or other irritating substances. It also occurs at the commencement of certain of the eruptive fevers, as measles, scarlet fever, etc., and in rare cases, the breathing of the vapor of iodin, or of particles of ipecacuanha in those possessing peculiar idiosyncrasies will bring on an attack.

The onset of an attack of acute coryza is ushered in by varying degrees of chill, from a slight chilly sensation to a pronounced rigor, a feeling of lassitude and general malaise, followed by a slight increase in body temperature, with myalgia and loss of appetite. The mucous membrane of the nasal passages becomes congested, accompanied by a sense of burning and prickling, with a feeling of dryness and heat. This may last for a few hours, or even a couple of days, when a watery, acrid discharge sets in, gradually changing in the course of a few days to a thick yellow mucus, more or less copious in amount, and finally to a free discharge of a purulent character. The duration of the attack may be from three or four days to two or three weeks.

These symptoms are the result first of congestion of the mucous membrane and the arrest of the secretions producing the characteristic dryness. This is followed by a free transudation of the liquor sanguinis from the engorged blood-vessels, causing swelling of the membrane, and supplying the main portion of the earliest secretion, which is of a serous character. Later, the glandular structures are stimulated to an abnormal activity, and a profuse discharge is established, consisting of mucus, epithelial cells, and leucocytes, with a slight admixture of red blood-corpuscles. As the disease progresses, the embryonic cells increase in numbers, and the secretion assumes a purulent character. When the nasal mucous membrane is alone affected, the discomfort is referable to the nose only, and consists of an increased secretion, sense of fullness, or complete occlusion, due to swelling of the membrane, and repeated and distressing attacks of sneezing. If the disease extends to the frontal sinuses there is often, as a marked symptom, a severe frontal headache; when the antrum of Highmore is involved, there is usually a more or less severe neuralgia, referred to the infraorbital or malar region. If the inflammatory symptoms are severe, causing considerable swelling and thickening of the mucous membrane, it is likely to result in the closure of the openings into the nasal passages of the accessory sinuses, and consequent retention of the secretions, followed by the symptoms described under the head of suppurative inflammation of the antrum.

La grippe is frequently manifested in an acute inflammation of the mucous membrane of the nasal passages, which often extends to the accessory sinuses, leading to mucous engorgement and other more serious involvement, especially of the antrum of Highmore.

It was the experience of the writer in the last epidemic of *la grippe* in Chicago, during the winter of 1891-92, to treat a larger proportion of cases of engorgement of the maxillary sinuses as a direct result of attacks of this disease which were principally confined to the upper air-passages, than from any other or all other causes combined.

The prognosis in these cases, as in those arising from acute

coryza, is much better than when the cause is some chronic inflammatory condition of the mucous membrane.

Chronic coryza is the result of repeated attacks of the acute disease, or it may occasionally be of a chronic type from the beginning. The most prominent symptom is an increased secretion of mucus, or of muco-pus, which is discharged through the nose, or through the pharynx into the mouth. The discharge is semi-fluid, having somewhat of a purulent character, on account of the copious admixture of embryonic cells and of epithelial scales with the mucus. There is no marked thickening of the mucous membrane, and its surface is at all times soft and moist. Its color is abnormal, showing a reddened, congested appearance, sometimes turgid or purplish. The disease is aggravated by changes in the weather, and is more prominent in the chilly spring and autumn days. Such conditions, when affecting the antrum, rarely cause stenosis of the nasal opening, consequently the discharges escape from time to time into the nose, when the body assumes a favorable position for the drainage of the cavity. The secretions, as a rule, however, are not discharged with sufficient freedom to prevent their decomposition and consequent fetid odor, though sufficiently so to prevent the sense of fullness or the expansion of the antrum, so common in those cases having retained secretions.

REVIEW.

CHAPTER XXXIII.

What is the location of the maxillary sinus?
What is its shape?
In what direction does the apex point?
Has it any communication with the nasal passages, and where?
With what is the antrum lined?
How many walls has the antrum?
Give the name and location of these walls.
Where is the thinnest, most dependent point of the antrum?
What is the character of the floor of the antrum?
What is the capacity of the antrum?
Name the accessory cavities.
By what canal may the frontal sinuses discharge into the antrum?
What do Cryer's investigations prove as to the anatomy of the parts?
Do Fillebrown's investigations corroborate these discoveries?
Are diseases of the antrum common or rare?
In what class of society are they most prevalent?
What are some of the conditions which predispose to antral disease?
What are the most common diseased conditions affecting it?

Which of these is most frequently met with?

What are the causes of suppurative inflammation of this sinus?

Name the lesions of the teeth that may produce this affection.

What are the forms of inflammation?

How may a dead pulp cause it?

What are the methods employed for testing the vitality of suspected teeth?

How may an alveolar abscess cause suppuration of the antrum?

Does septic intoxication ever accompany acute suppurative inflammation?

How may a malposed tooth produce the disease?

How did the third molar in the case described produce the disease?

Name the foreign bodies that are sometimes found in the antrum.

How do such substances produce suppurative inflammation?

What is the character of the inflammation under these circumstances?

Are such conditions easily diagnosed?

How may the diagnosis be made sure?

What are the traumatic injuries that may result in this disease?

What is the usual result of such injuries when associated with the oral cavity?

Why is suppuration more likely to supervene under such circumstances?

How may catarrhal affections of the nasal mucous membrane produce engorgement of the antrum of Highmore?

What catarrhal affections are the most common causes?

Are acute or chronic inflammations of the lining membrane more likely to cause the disease?

Describe the symptoms of acute coryza.

Describe the pathologic changes which take place in the mucous membrane.

What is the characteristic symptom of extension of the disease to the frontal sinus?

What symptom marks its extension to the maxillary sinus?

What is the result of severe inflammation of the lining membrane of the accessory cavities?

May this swelling, etc., cause stenosis of the nasal opening, and what would be the result?

How may *la grippe* cause engorgement of the antrum?

Is it a prolific cause of this condition during epidemics?

What is the prognosis in those cases caused by acute inflammations?

What are the symptoms of chronic coryza?

Does the disease cause stenosis of the maxillary sinuses?

How and when are the secretions discharged?

Does decomposition occur, and is fetor present?

Why are these conditions present?

CHAPTER XXXIV.

DISEASES OF THE MAXILLARY SINUS (Continued).

Suppuration of the Antrum of Highmore.

Symptoms.—The symptoms of suppuration of the antrum are pain, which is at first dull and deep-seated, later becoming more intense, shooting over the face and forehead, sometimes including the ear. Occasionally the pain is very acute, and of a sharp, stabbing character. The cheek becomes swollen and tender; the walls of the antrum are thinned, and later give forth, under pressure, a crackling sound, like that of crushing an egg-shell, or of crumpling parchment. Frequently there is protrusion of the eyeball, sometimes accompanied by amaurosis. This feature is due to the thinning of the floor of the orbit, or roof of the antrum, and protrusion into the orbital cavity, which forces the eyeball outward, and causes paralysis of the optic nerve from pressure upon it. In acute cases, the formation of pus is ushered in with a rigor, followed by elevation of temperature and general systemic disturbance. In the subacute and chronic forms, the constitutional symptoms may be entirely absent. Generally there is an offensive, purulent discharge from the nostril of the affected side; this symptom, however, may not be present, on account of the closure of the normal opening into the nasal passages, the osteum maxillare, from induration of the lining mucous membrane. In the latter condition the protrusion of the antral walls is usually much greater, and the suffering induced by the pressure of the pent-up inflammatory products is often very severe. Sooner or later the walls of the antrum are absorbed, the soft tissues are penetrated, and the fluid escapes. The location at which the rupture of the wall takes place is generally that point which offers the least resistance. This varies greatly in different individuals. The most common locations are the nasal wall, the palate process, and the infraorbital plate, or roof of the antrum. Occasionally the buccal wall will give way, or the fluid may follow the root of a tooth which penetrates the floor of the antrum, and discharges into the mouth; more rarely it may burrow backward, after having penetrated the floor of the orbit, and enter the brain through the sphenoidal fissure or the optic foramen. Rupture of the walls of the antrum usually

causes considerable ulceration and necrosis, which may involve the entire maxillary bone. If the discharges enter the cranial cavity, epileptiform convulsions are likely to be induced, with other brain-symptoms, closely followed by a fatal termination.

In those cases in which the discharges escape into the nose, the breath is exceedingly offensive, having the odor of a rotten egg, which is due to the presence of hydrogen sulfid gas, liberated by the decomposition of the albuminates contained in the discharges. Blowing the nose will sometimes relieve the sense of fullness by causing a flow of the discharges. The more common course is for the discharge to be quite constant, the amount varying from a slight quantity to a considerable amount, and flowing into the nose and throat when lying upon the unaffected side. During sleep the discharges are often swallowed, producing nausea and sometimes vomiting on rising in the morning. The movements of mastication may also cause a discharge of the pent-up secretions, while taking food, and thereby induce nausea and vomiting.

A case of this character came under the care of the writer some years ago, and the gentleman related that he had not taken a meal with his family, for this reason, and on account of the fetid odor of his breath, for more than six years.

The teeth of the affected side are often sore and painful to percussion, even though they may not be factors in the production of the disease. This is a point that should be borne in mind when conducting an examination in a case of this character. When the disease is due to lesions of the teeth, the affected ones are usually more sensitive and painful to the percussion test than are those adjacent to them; yet in exceptional cases, the most careful examination may fail to detect a special tenderness in any of the teeth.

Diagnosis.—The diagnostic signs are, the location and the character of the pain, unilateral discharge from the nose (except where both antra are affected, when it would be in all probability bilateral), swelling of the face, bulging of the diseased side of the vault of the mouth, tenderness of the affected side of the face, crepitation over the thinned walls of the antrum, frequently the presence of diseased teeth, soreness of the teeth to percussion, protrusion of the eyeball, fetor of the breath, nausea or vomiting on rising in the morning.

These conditions are not always well marked in every case, but a sufficient number will be present in a majority of them to establish a diagnosis.

Differential Diagnosis.—The diseases of the maxillary sinus which may be confounded with abscess or suppuration of this sinus are angiomata, malignant neoplasms, and bony tumors. Tumors of the antrum can be very positively diagnosed from suppuration and mucous

engorgements by means of the electric mouth-lamp or stomatoscope. The patient being seated in a dark room, the lamp is placed in the mouth, and the lips closed over it, when, if the tissues are in a normal condition, the light will be quite readily transmitted through the cheeks and lower eyelids. If the antrum is filled with fluid, like pus or heavy mucus, the light is somewhat impeded, while if it be filled with a solid tumor, it is entirely obstructed. An exploratory puncture or incision with the exploring needle or small scalpel will often immediately clear up the diagnosis.

Prognosis.—The prognosis is usually favorable, except in those cases in which the discharges have burrowed into the cranial cavity. In a majority of cases a cure is effected by slow degrees, sometimes requiring months, and even one or two years, to accomplish it. The general condition of the patient is an important factor in the prognosis. A tubercular or scrofulous diathesis, or a generally debilitated condition, militates against a rapid cure. In persons of otherwise good health a cure is sometimes effected in a few weeks; the majority, however, extend over a much longer period.

Treatment.—For the successful treatment of suppuration or abscess of the maxillary sinus, three conditions must always be secured, viz: First, the removal of the exciting cause; second, the complete evacuation of the contents of the sinus; and, third, the establishment of perfect drainage.

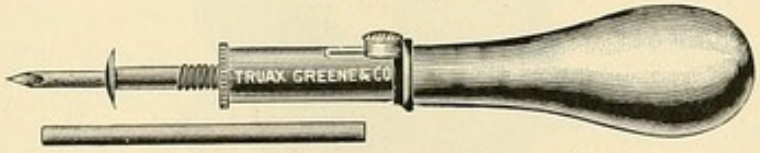
Failure to secure these conditions is among the principal reasons why the treatment of this disease is many times so unsatisfactory. In order to secure these conditions the sinus should be opened at its most dependent point, which is at the floor of the cavity opposite the alveolus of the second molar tooth, and if bony septi are present, these should be broken down with the curette or surgical bur. The simplest way of entering the antrum is by extracting the first or second molar tooth, and enlarging and deepening the alveolus of the anterior buccal root. This alveolus is selected because it carries a larger root than the posterior one, hence is more available for this purpose. If these teeth are sound, it is preferable to make an opening between the posterior buccal root of the first molar and the anterior root of the second, as suggested by Heath, rather than to needlessly sacrifice sound teeth. On the other hand, all diseased teeth and roots upon the affected side should be extracted at once, as they are more than likely to be the primary cause of the disease; and if they should not be directly responsible, they are usually a source of irritation, and may therefore become a secondary cause of the lesion.

In opening the antrum, the ordinary trocar fitted with a canula is a very satisfactory instrument if properly handled. There is danger, however, when the bone is thick and hard, requiring a considerable

amount of pressure to penetrate it, that it may suddenly break through, and the point of the trocar penetrate the floor of the orbit. This accident may be guarded against by so holding the instrument that it can only penetrate to the desired depth, or an adjustable metal guard can be placed upon the instrument, which will effectually prevent its going beyond the depth to which it is desired to penetrate. The trocar, Fig. 129, was devised by the writer expressly for this purpose. The guard is fitted into the handle by means of a screw, which makes it possible to penetrate the antrum at any desired depth, while the handle is fitted with a device operated by the thumb which carries the canula forward to the tip or the trocar, and allows the trocar to be withdrawn, leaving the canula in position.

The most satisfactory method of opening the antrum is by the use of a spear-pointed drill, revolved by the surgical engine. With this instrument the surgeon can feel his way through the bone so delicately

FIG. 129.



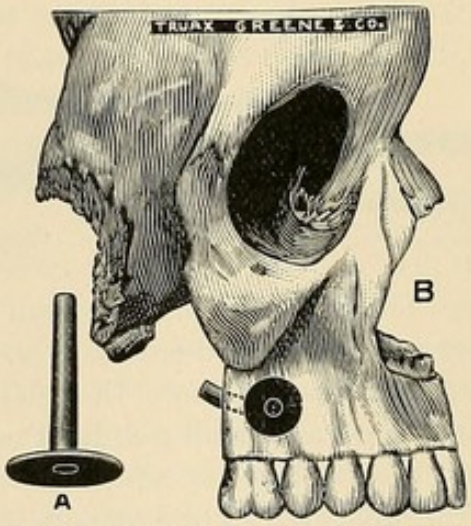
AUTHOR'S ANTRUM TROCAR WITH GUARD. (Reduced.)

and surely that he can tell when the drill is about to enter the cavity, and even when it pierces the mucous membrane, so that by this method accidents are reduced to the minimum. In order to secure free discharge, the drill should leave an opening at least one-fourth of an inch in diameter. This is about as large an opening as can be secured between the roots of the first and second molars without injuring the roots of these teeth; but when the opening is made through the alveolus, there is no reason why it may not with advantage be made larger. When the probe reveals the presence of bony septi, it becomes necessary to make the opening somewhat larger. Under such circumstances, the septum of bone between the anterior and posterior buccal alveoli can be cut through with a long side-cutting bur, which is an enlargement in shape of the dentist's fissure bur. (See Fig. 115.) This will give entrance to a small spoon curette, or the round surgical bur, with which to break down the bony septi.

An opening which is made through the external wall of the antrum between the roots of the first and second molars is preferable to one made through the alveolus of a tooth, for the reason that in the former case the cheek, falling over the opening made through the external wall, is a protection against the entrance of food, while in the latter, unless it is kept plugged, foreign substances constantly enter, which, acting as irritants, tend to keep up the inflammatory symptoms.

Plugs and drainage-tubes are a source of great annoyance and inconvenience to the patient, and, in the opinion of the writer, are many times a source of irritation, thus retarding the progress of the cure. When the opening is through the alveolus of a tooth, plugs or tubes with stoppers are necessary to prevent the ingress of food; at the same time they prevent the free discharge of the secretions. This condition, in relation to an abscess in any other location of the body, would not be tolerated by an enlightened surgeon for a moment, as it would defeat the very object for which drainage was established. The preference, therefore, should be given, when circumstances will permit, to that operation for opening the antrum which will not require the use of plugs or tubes in the after-treatment. When a drainage-tube is employed, no better form can be chosen than that suggested by Talbot, Fig. 130.

FIG. 130.



TALBOT'S ANTRUM TUBE. (After Talbot.)

Some surgeons prefer the method of opening the antrum through the nose, as near to the natural entrance as possible, one purpose of which is to render it impossible for infection to occur through the mouth, as is feared if an opening is made into the antrum through this cavity. This method seems to be open to several important objections, on account of the difficulties in the way if it becomes necessary to break down bony septi, or to curette the surfaces of the sinus. This method, however, has its advantages in the treatment of mucous engorgements, for all that is necessary in these cases is to re-establish the discharge of the secretions into the nose; but in those more serious conditions, like abscess of the antrum, from various causes, entrance through the mouth is to be preferred, because an opening can be made as large as the circumstances require,—large enough to admit the index finger, or larger, as sometimes becomes necessary, especially where septi are to be broken down, or search made for foreign bodies

or malposed teeth. Under circumstances like these, the tactile sensation of the finger is a much surer guide to a correct understanding of existing conditions than a probe or a sound, which are the only means of detecting these conditions when entering the antrum through the nose.

General anesthetics are frequently necessary in operations for opening the antrum, especially in those cases requiring the extraction of teeth, the cutting of bone to any considerable extent, or curetting the mucous membrane. In those cases requiring only the puncture of a thin wall of bone, the local application of cocain by hypodermic injection will answer a good purpose. The writer has made several quite extensive operations upon the floor of the antrum with no other anesthetic than cocain; yet he feels safer with ether or chloroform than with cocain, and therefore does not recommend its use when repeated injections would be necessary to maintain the condition of local insensibility, for fear of establishing the toxic symptoms of the drug. Solutions of cocain of a greater strength than 2 to 4 per cent. are never required in these operations.

After an opening has been made into the antrum, it should be thoroughly irrigated with some *bland, non-irritating* antiseptic solution. The writer prefers the Thiersch solution, or the boric acid solution. Irrigation should be continued until the fluid runs clear. The ordinary irrigating bag, with rubber tubing and a glass nozzle, is preferable to any of the syringes recommended for this purpose.

Solutions of bichlorid of mercury and carbolic acid have certain disadvantages which should cause them to be discarded in all diseases of the mucous membrane. The bichlorid of mercury solutions are more or less irritating to all mucous surfaces, if of a strength sufficient to be of real value as a germicide, and have the added disadvantage of being readily absorbed in sufficient quantities to produce toxic symptoms, if by chance the fluid should be retained in the sinus. Carbolic acid is also irritating to mucous surfaces when of a strength to be valuable for antiseptic purposes. A solution of less than 5 per cent. would be of little value as a germicide, while one of that strength would be irritating.

The employment of irritating solutions is, in the mind of the writer, another reason why the inflammatory conditions of the maxillary sinus are so tedious and difficult to cure. This opinion has grown out of a considerable experience in the treatment of this class of diseases, and experimenting with the various methods of treatment with drugs suggested by the recognized authorities on this subject.

The peroxid of hydrogen, and medicinal pyrozone, are remedies which the writer has lost confidence in for this purpose,—not that they are not good scavengers or good disinfectants, but that they frequently

cause great pain and irritation, even when diluted, from the rapid evolution of oxygen gas, and the consequent pressure upon a highly sensitive membrane when they are injected into the antrum, especially in those cases where the opening made for evacuation and drainage is necessarily small.

In the after-treatment the same bland solutions, or sterilized water alone, are to be preferred to solutions which are in the least irritating or over-stimulating. Irrigation should be performed three or four times *per diem* for the first few days after the operation, preferably after meals, and on rising in the morning. As the symptoms subside, irrigation may be gradually decreased to once *per diem*, and finally withdrawn altogether.

Insufflation with powders is not to be recommended, for the reason that there is no assurance that they are invariably dissolved in the secretions. Materials of this character, if left undissolved in the antrum, would be likely to act as foreign bodies, thus continuing the irritation and preventing a cure.

No anxiety need be felt in reference to the final closing of the opening made into the antrum. There is more difficulty experienced, as a rule, in keeping it open for a sufficient length of time for proper treatment, except in those cases where plugs or tubes are used. In exceptional cases, where large openings have been made, or plugs and tubes have been used, it may become necessary to stimulate granulation by touching the edges of the opening with nitrate of silver or the galvano-cautery, followed by repeated applications of tincture of iodin, or it may be closed by a plastic operation.

The treatment of mucous engorgements is less difficult, from the curative standpoint, than suppurative conditions of the antrum, except in those cases where the secretions have been retained for a period sufficiently long for decomposition to have taken place, when the inflammatory condition assumes a chronic type, rendering the treatment much more difficult and tedious.

In the ordinary cases of mucous engorgement of the antrum resulting from acute coryza, or *la grippe*, drainage can be secured by expanding the natural opening into the nose. This may be accomplished by passing probes or sounds of gradually-increasing diameter. To avoid the paroxysms of sneezing, and the pain induced by the introduction of the probe, the mucous membrane should be sprayed with a 10 per cent. solution of cocain. When the natural opening cannot be found,—and it is not always an easy matter to find it and introduce the probe,—the sinus can be entered at one of those points already indicated for draining this cavity, preferably between the roots of the first and second molar teeth.

In the experience of the writer, this simple form of antral disease

is the most amenable to treatment; thorough drainage alone being all that has been required in many cases to effect a complete and permanent cure in from two to three weeks. In the more persistent cases, daily irrigation with the nasal douche, charged with sterilized water or some of the bland antiseptic solutions, will be necessary. If a purulent condition of the secretions should follow the opening of the antrum, it is certain that the condition first mentioned has not been secured; upon a more careful examination it will be discovered that either complete evacuation has not been secured on account of dividing septi, or not having punctured the cavity at its most dependent point; or that the drainage is imperfect, either from the closure of the opening in the mucous membrane, the formation of a clot in the wound, or the entrance of foreign substances or septic bacteria. It is often difficult to maintain a free opening in the mucous lining of the cavity by the ordinary methods, as the tendency of wounds in this tissue is to heal very quickly. To obviate this difficulty, the sides of the opening may be cauterized with the electro-thermal cautery; two or three applications may be necessary to secure an opening that will remain patulous for a sufficiently long period to accomplish a cure.

REVIEW.

CHAPTER XXXIV.

What is the character of the pain in suppuration of the antrum?

What is the cause of the parchment-like crepitation?

What is the cause of the protrusion of the eyeball?

What constitutional symptoms accompany acute cases?

What is the character of the discharge?

When the discharges do not escape into the nose, what result is likely to follow?

At what point does rupture take place?

Which walls offer the least resistance?

What complications usually follow rupture of the antrum?

What is the result when the discharges enter the brain?

What characteristic odor always accompanies purulent discharges from the antrum?

If this characteristic discharge is present, but no swelling to indicate which antrum is affected, what other symptoms will indicate it?

Give the diagnostic signs.

What conditions of the antrum might be mistaken for suppuration of this sinus?

How may they be differentiated from suppuration of the antrum?

Is artificial light transmitted through the normal tissues of the superior maxillary bone when the light is placed in the mouth?

Would the light be transmitted if the antrum was filled with fluid?

If filled with an abnormal growth?

What is the prognosis?

What constitutional conditions militate against a rapid cure?

What three conditions must be secured to successfully treat this disease?

Where is the best point to enter the antrum?

What other points have been suggested?

If the teeth are all in a healthy condition would it be advisable to extract any of them for the purpose of entering the antrum, and why?

Under these circumstances what location should be chosen for this purpose?

What advantage has this location over opening through the alveolus?

What instruments are necessary for opening the antrum?

How are bony septi to be broken down?

What are the disadvantages of plugs and drainage-tubes?

What are the disadvantages of making an opening through the nasal wall?

What is the treatment after the antrum has been evacuated?

What solutions are best for these cases?

What are the objections to carbolic acid and bichlorid solutions?

What are the objections to peroxid of hydrogen solutions?

How often should irrigation be performed?

What are the dangers from the insufflation of powders?

By what means may a large opening be finally closed?

What method of draining the antrum for the relief of mucous engorgements is recommended?

How can this expansion be accomplished?

Where should the antrum be penetrated if the natural opening cannot be found?

What other means beside drainage is sometimes necessary to restore the cavity to a normal condition?

What are the probable causes of a purulent discharge following the opening of the antrum?

By what means may the mucous lining be prevented from closing the opening made into the antrum?

CHAPTER XXXV.

DISEASES OF THE MAXILLARY SINUS (Continued).

Syphilitic Ulceration of the Antrum of Highmore.—This is a condition of rare occurrence. The antrum, however, sometimes becomes involved when the roof of the mouth is the seat of the destructive syphilitic process, or when the turbinated bones and the nasal wall of the antrum are affected.

This manifestation of a syphilitic infection is generally the result of the tertiary form of the disease, and is usually an extension of the affection from the nasal fossa. The writer is of the opinion that syphilitic ulceration of the mucous lining of the antrum is never found except as a tertiary manifestation and an extension of the disease already located in contiguous parts, although he is fully aware that in giving expression to this opinion he places himself in opposition to the views of some excellent observers. In support of this position, reference is made to Morrow, who says, "Familiar sequelæ of tertiary syphilis are perforation of the cartilaginous and bony septum, and the palatine roof; caries and necrosis of bone, ozena, and *extension of the disease to the antrum*, and other accessory cavities, and to the bones of the face and skull."

Garretson says, after a careful examination of the syphilitic patients in Blockley Hospital, Philadelphia, extending over a whole year, he could not find a single case of syphilitic disease of the antrum in which the disease had its origin in this sinus.

The manifestations of syphilis are quite common in the nasal cavity, but they belong essentially to the tertiary stage, the earlier symptoms being very rarely manifested in this region.

Bosworth does not believe the secondary stage of the disease, in the form of mucous patches, ever appears in the nose.

It is more than likely that such secondary manifestations of the disease are never found in the antrum of Highmore; though positive or negative demonstration of this statement could not be furnished except by a long and careful search for the proof upon persons who had died while suffering from secondary lesions of the disease. Such examinations may have been made and the results published, but the writer does not recall any published account of such a line of investigation.

It is fair, however, under the circumstances, to reason from analogy, that inasmuch as the same type of mucous membrane lines the maxillary sinus that lines the nasal passages, and that the secondary lesions of the disease are very rarely if ever found in the nasal passages, the same immunity in all probability is possessed by the antral lining membrane. The tertiary manifestations of syphilis in the nose and antrum usually develop in from five to fifteen years after the initial lesion, or the primary stage of the disease, in the formation of deep-seated ulcerations of a grave and destructive character, pursuing a rapid course, causing a more or less extensive destruction of tissues, and involving the cartilages and the nasal and turbinated bones to such an extent as to cause most horrible deformities. The disease is characterized by suppuration and necrosis, accompanied by the discharge of masses of pus, blood, and necrotic tissue, and a most intolerably fetid and penetrating odor which makes the patient an offense to himself and to every one who comes near him.

There are two varieties of syphilitic ulceration of the nose in the tertiary form of the disease, viz: superficial ulcer, and deep-seated ulcer.

The *superficial ulcer* is usually found upon the mucous membrane covering the cartilaginous septum. It may destroy the cartilaginous septum and then attack the bony septum, resulting in a more or less complete destruction of this portion of the nose, though it rarely extends so far, being confined generally to the cartilaginous septum.

The *deep-seated* ulcer is by far the most serious and destructive form of tertiary syphilis found in the nasal cavity. It is due to the formation of gummata in the deeper layers of the mucous membrane, which later become softened and break down, developing an ulcerative action of a rapid and destructive type.

This form of ulceration is usually found upon the turbinated bones, and is characterized by deep excavating ulcers with ragged, overhanging edges; the surface of the ulcer is bathed with yellow pus mingled with fragments of blackened gangrenous tissue; and the surrounding mucous membrane is highly congested, often turgid and purplish in color.

The ulcerative process rapidly penetrates to the periosteum and the bone, also extending laterally with equal rapidity, often causing extensive necrosis and exfoliation of the osseous structures of the nose, and sometimes involves the accessory sinuses.

There is always an offensive discharge, giving the odor of decomposing pus and dead bone, accompanying the disease, which makes the presence of these poor unfortunates almost intolerable. The tendency to the formation of crusts or masses of dried pus, mucus, blood, and gangrenous tissue, which adhere closely to the surface, and are exceed-

ingly difficult to dislodge, is another characteristic of the disease. These masses when expelled are in odor and appearance most disgusting and nauseating. As the disease progresses, the nasal wall of the antrum may become involved in the destructive process, which may finally extend to the mucous membrane lining this cavity. When the antrum becomes involved there is usually as an indication a swelling at the angle of the nose and cheek, and in those cases where the nasal wall of the sinus has been lost by necrosis, this fact may be ascertained by exploration with a probe.

Diagnosis.—There are no characteristic symptoms of the disease located in the antrum, other than the swelling at the angle of the nose and cheek, that are distinguishable from those found in the nose.

The more common location of the disease in the superior maxillary bones is the floor of the nasal fossa, which may be destroyed, leaving a more or less extensive opening between the nose and mouth, making the swallowing of food and liquids difficult, and greatly impairing the voice.

The tertiary form of the disease rarely involves the velum palati, the septum alæ narium, or the cutaneous surfaces; but is usually confined to the cartilaginous septum, the inner nasal bones, the turbinated bones, and the superior maxilla.

The deformities which result are the falling in of the nose caused by the loss of its bony supports, and the perforation of the hard palate just referred to.

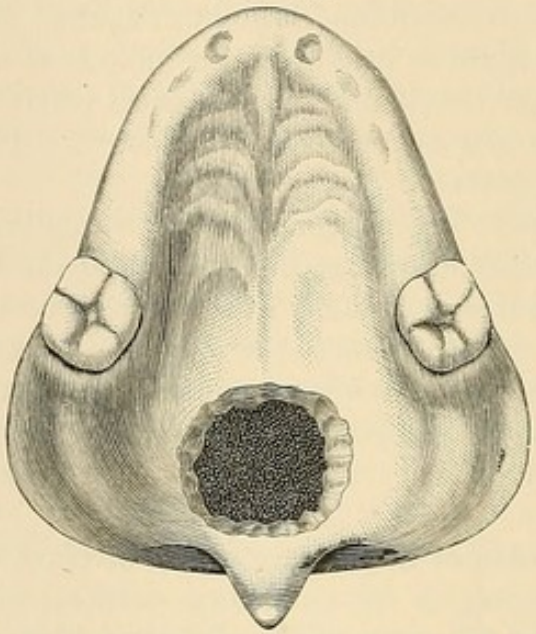
One case only has the writer seen of syphilitic ulceration of the antrum, which did not have its starting point in the nasal fossa. The patient was a man who had been inoculated twenty years before. The disease began as an ulceration of the mucous membrane covering the hard palate opposite the right second molar tooth, resulting in perforation of the hard palate and antrum, with loss of the entire floor of the sinus, together with the teeth, from necrosis. Figs. 131 and 132 are illustrations of the secondary form of the disease which had their commencement in the velum palati in the form of ulcers, and later involved the palate bones. In Fig. 131 the opening originally extended considerably farther forward, but this has been gradually filled up by the process of granulation.

Differential Diagnosis.—Syphilis of the nose and antrum is often diagnosed as fetid catarrh, ozena, and other forms of disease which are accompanied by foul-smelling odor and discharges. The diagnosis, however, is rendered quite simple if a good view of the nasal cavity can be obtained through the anterior nares, as the parts most likely to be affected are generally within the range of vision through these openings.

In order to obtain a good view of the parts, the crusts and dis-

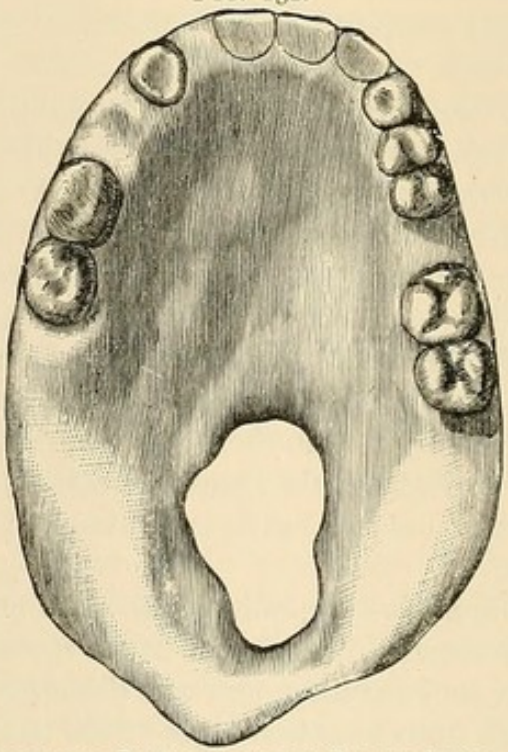
charges must first be removed, as these cover and conceal the conditions of the tissues beneath. If the disease is fetid catarrh or ozena, the mucous membrane, the septum, and the walls of the nasal cavity

FIG. 131.



SYPHILITIC ULCERATION OF THE VELUM PALATI INVOLVING THE POSTERIOR BORDER OF THE PALATE BONES.

FIG. 132.



SYPHILITIC ULCERATION INVOLVING PORTIONS OF THE PALATE BONES AND THE VELUM PALATI.

will be found intact; while on the other hand, if it is syphilis, ulcerations upon the septum or turbinated bones or necrosis of bone of greater or less extent, will be discovered.

This fact, however, must be borne in mind in making a diagnosis, viz: that ulceration never occurs except as a result of some general dyscrasia like syphilis, tuberculosis, cancer, the exanthemata, etc.; there is, however, no danger of making an error in the diagnosis, for differentiation is made comparatively simple by the presence of symptoms which are characteristic of one or the other of the diseases. It is safe therefore to say if ulceration is found without any of the general symptoms of impaired health which accompany tuberculosis, cancer, or the exanthemata, that the disease is in all probability due to syphilis, even though no positive history of infection with the syphilitic virus can be established, for it is often difficult after the lapse of so long a period to arrive at a definite history of the presence of the primary lesion or of secondary manifestations.

Treatment.—The systemic treatment of tertiary syphilis is confined to the iodid of potassium in gradually increasing doses, beginning with 10 to 15 grain doses three times per day, adding three grains each day until the toxic manifestations of the drug appear, when a marked improvement in the symptoms is usually observed. Mercury is contra-indicated in this form of the disease. It is well occasionally to withdraw all systemic medication. Tonics, sea air, a generous diet with wine or malt liquors, are always indicated, and occasionally this is all that is necessary.

The local treatment of syphilitic ulceration of the nose and antrum should be directed to securing cleanliness of the parts, by removing secretions, crusts, and pieces of dead bone which are sources of irritation, stimulating ulceration and retarding the reparative process.

The removal of the crusts is greatly facilitated by the use of the post-nasal syringe and the douche charged with antiseptic solutions,—the milder forms, like the Thiersch and the boric acid solutions, being preferable.

When the antrum is involved, this cavity can usually be reached through the nose by means of a curved nozzle attached to the syringe or douche.

Boric acid solution and cinnamon water, equal parts, is a good disinfectant and deodorizer, and may be used to best advantage with the atomizer.

The removal of dead bone should be accomplished at as early a period as possible, but the writer does not believe it is the part of a wise conservatism to attempt its removal until separation has taken place.

Necrosis of the Walls of the Maxillary Sinus.—Necrosis of the walls of the maxillary sinus is a diseased condition of somewhat common occurrence, for the reason that there are so many lesions, either of an idiopathic, traumatic, or specific origin, which affect this part of

the face and have a tendency to result in inflammatory conditions of the bone, and finally in necrosis.

Necrosis of the walls of the antrum may occur as the result of periostitis, induced by certain diseases of the teeth, such as septic pericementitis, or alveolar abscess; local arsenical poisoning caused by the escape of the drug into the surrounding tissues when used for the purpose of devitalizing the tooth-pulp; fractures resulting from the extraction of teeth contiguous to the antrum; rupture of the walls of the antrum from the accumulation of pus or other fluids in the sinus; gunshot wounds and other injuries causing crushing and comminution of the walls of the antrum; tuberculosis, syphilis, the exanthemata, and mercurial and phosphorus poisoning.

The walls of the antrum most often found necrosed are the nasal wall, caused by specific disease extending from the nasal fossa; the superior wall or orbital plate and the inferior wall or floor of the sinus, from rupture induced by accumulated fluid. The floor of the antrum is also quite frequently the seat of necrosis as a result of the diseased conditions of the teeth already mentioned, and from surgical violence in extracting the teeth of this locality. More rarely the anterior and posterior walls become necrosed from inflammatory conditions of the sinus and from traumatic injuries.

Symptoms.—The symptoms of necrosis of the walls of the maxillary sinus are the same as in necrosis located in other portions of the body; briefly: a history of a previous acute inflammation, with great pain, swelling, and discharge of thick pus. Present conditions, pain slight, swelling of the overlying tissues, discharge of offensive smelling, purulent pus, numerous sinuses, and denuded bone, which may be felt with a probe passed through the sinuses. Sinuses which lead to dead bone always present a granular appearance at the surface, and have a tendency to bleed on being touched.

The most frequent locations of the sinuses in necrosis of the antrum are the roof of the mouth, alveolar ridge and external integument in the regions of the inner canthus of the eye, and the infraorbital foramen. Ugly scars often result from the healing of these sinuses when located in the latter regions, and not infrequently cause ectropion of the lower eyelid.

Treatment.—The operative treatment does not differ essentially from that for the same conditions in other locations, except that every means should be employed to prevent the formation of unsightly scars upon the face. A word of caution may not be amiss to the young practitioner in relation to the proper time to operate for the removal of dead bone in the region of the face. A safe rule to follow is not to attempt to remove the necrosed bone until separation has taken place between the living and dead portions. Less deformity, in the judgment

of the writer, follows such procedure than when an operation is performed before this process of exfoliation has been completed. It is wise to frequently examine the sequestrum to ascertain if separation has taken place, and as soon as this can be demonstrated it should be removed. The constitutional treatment must be directed to the building up of the health of the patient and controlling the tendencies of peculiar dyscrasia or constiutional vice.

A case at present under treatment, which gave the writer considerable trouble from its rapid extension until the real nature of the disease was discovered, may be used as an illustration upon this point. The patient, a man forty years old, was referred to the writer by a professional friend. At the time he was first seen there was considerable swelling of the right side of the face below the malar bone, and the teeth and external plate of the alveolar process had been removed between the right central incisor and the first molar of the same side. From general appearances, tertiary syphilis was suspected, but he denied ever having contracted the disease, and his word was taken upon that point, and only general tonics administered. At the end of ten days the disease had extended upward to the inferior border of the orbit and to the nasal bone, and backward, involving the floor of the antrum and the palate process to the median line. He was then placed upon the iodid of potassium, 15 grs. three times *per diem*, increasing the dose each day 3 grains; at the end of two weeks there was a very marked improvement in the conditions, the swelling was less, the disease had not extended beyond the limits just mentioned, exfoliation had already begun, and the case bade fair to make a good recovery, but with the loss of a considerable portion of the superior maxillary bone. It is possible that this might have been prevented had the iodid been administered at first, as would have been done but for the positive assurances of the patient. The wiser plan therefore, in all doubtful cases, regardless of the statements of the patient, be they never so positive, is to immediately begin a course of anti-syphilitic treatment.

REVIEW.

CHAPTER XXXV.

Is syphilitic ulceration of the antrum rare or common?
How does the antrum become involved?
During which stage of syphilis does the antrum become involved?
Are secondary symptoms of the disease ever found in the nose?
Is it likely that the symptoms are ever found in the antrum?

What reasons are given for this opinion?

How soon after inoculation does the tertiary stage of syphilis develop in the nose and antrum?

How is it manifested in these locations?

What is the character of these ulcerations?

By what are they always accompanied?

Name the number and varieties of syphilitic ulceration of the nose and antrum.

Where is the superficial ulcer usually located?

How does it affect the cartilaginous septum?

Does it attack other tissues?

To what portion of the nose is it usually confined?

What is the difference in character between the superficial and deep-seated ulcer?

What is the origin of the deep-seated ulcer?

What is the character of its progress?

Where is it most frequently located?

Describe the clinical appearance of the deep-seated ulcer.

What is the character of the discharges?

How does the antrum become affected?

What are the symptoms when the antrum is involved?

Which portion of the superior maxillary bone is most frequently the seat of the disease?

What portions of the nose are most often involved?

What other diseases may simulate syphilis of the nose and antrum?

What is the differential diagnosis in fetid catarrh?

What is it in tuberculosis, cancer, and the exanthemata?

What is the systemic treatment for tertiary syphilis?

What is the local treatment for the disease in the nose and antrum?

Through what channel is the antrum reached?

What solution is recommended as a good disinfectant and deodorizer?

When should the necrosed bone be removed?

Is necrosis of the walls of the antrum common?

What reasons are given for this frequency?

What are the causes of necrosis of the walls of the antrum?

Which walls of the antrum are most often necrosed?

What are the most common causes of each?

What are the symptoms of necrosis of the antrum?

Where are the sinuses most frequently located?

What is the peculiarity of a sinus which leads to necrosed bone?

What is the result of the healing of sinuses opening through the integument?

What is the surgical treatment?

When is the proper time to remove dead bone, located in this region?

What is the line of general treatment?

CHAPTER XXXVI.

CYSTIC TUMORS OF THE MAXILLARY SINUS.

THE maxillary sinus is not infrequently the seat of various forms of cystic and solid tumors, but their presence is rarely discovered until they have obtained a considerable size, filling the sinus and expanding its walls. Attention is drawn to the presence of solid tumors by the swelling, and the pain which usually accompanies the formation of these growths and certain forms of malignant neoplasms. Among the cystomata or cystic tumors found in the antrum may be mentioned Mucous Cysts and Polypi.

Mucous Cysts of the Antrum, the *hydrops antri* or dropsy of the antrum of the old writers, is a disease resulting from the cystic degeneration of the glandular follicles which are very numerous over the entire mucous membrane lining the cavity. The disease is characterized by the presence of a dark straw-colored, glairy fluid, sometimes gelatinous, and of the consistence of egg albumen, frequently containing considerable quantities of cholesterin which appear in the form of small flakes, floating in the fluid. The accumulation of the fluid is slow, which causes a painless enlargement of the face upon the affected side, with expansion of the antrum and thinning of its walls.

The disease was formerly thought to be caused by the retention of the natural secretions, but modern research has proved this view to be incorrect. The retention of the natural secretions is generally the result of acute inflammatory conditions of the mucous membrane of the nose which have extended to the sinus, and produced occlusion of the osteum maxillare.

In cystic degeneration of the mucous membrane of the antrum there is frequently an aggregation of small cysts which makes the treatment of the case a more difficult one than when the sinus is filled with a single cyst. In some cases the cyst seems to be developed within the bony wall of the antrum, or between the bone and the periosteum, and by lateral extension fills the cavity and makes it possible under such circumstances to mistake the cavity of the cyst when it is opened for the sinus itself.

Occasionally serous cysts develop at the roots of devitalized teeth as a result of chronic inflammation. Heath mentions a case reported

by Fischer in which he was able by post-mortem examination to clearly trace such a cyst which occupied the entire antrum, but had no connection whatever with its walls, and was attached only to the roots of a molar tooth by its pericementum.

Cysts of this character, though not so extensive in size, are frequently found in both the upper and lower jaws; at least this has been the observation and experience of the writer. One somewhat similar case to that mentioned by Heath, occurring in his private practice, may be used as an example of the difficulties sometimes experienced in making a correct diagnosis. Mrs. O., thirty-five years of age, was referred for treatment of an extensive enlargement of the right side of the face in the region of the antrum, and extending forward to the ala of the nose. There was some difficulty in breathing through the right nostril, due to bulging of the nasal wall of the antrum. The contour of the palate was normal. On examination of the teeth, it was found that the lateral incisor and the first bicuspid were both devitalized, the pulp-canals of each having been filled some years before. Percussion of these teeth elicited tenderness in the first bicuspid, but not in the lateral. The swelling of the face had been noticed for more than a year, and it was slowly increasing in size. There was no discharge from the nose, and no sinus leading to the enlargement. The tumor was firm and unyielding. These symptoms all pointed to the presence of a solid tumor of the antrum. As a more positive means of diagnosis an exploratory puncture was decided upon, and inasmuch as the first bicuspid tooth was badly decayed, this was extracted with the intention of puncturing the floor of the antrum through its alveolus, but upon removal of the tooth an ounce or more of a thick, tenacious, straw-colored fluid, filled with flakes of cholesterin, escaped into the mouth. The opening was therefore enlarged with a surgical bur, and the surface of the antrum curetted under the local anesthetic effect of cocain. The wound was afterward kept open for several weeks, the antrum irrigated twice *per diem,* when the fullness of the jaw subsided and the opening in the antrum was permitted to close.

Six months later the patient returned with a recurrence of the disease, and the extraction of the lateral incisor was advised on account of the location of the swelling, which was greatest under the ala of the nose. This she declined to have done, but as a compromise, submitted to the opening of the root-canal. In this way the cyst was reached, and a considerable discharge followed of a fluid similar to that evacuated from the antrum. The wall of the cyst was then punctured near the apex of the root of the tooth, and a further discharge followed.

Injections of a 10 per cent. solution of iodin and glycerol were used twice a week for a month, with marked improvement, after which the opening was allowed to close and the root of the tooth was refilled.

A few weeks later the patient returned again with the face much swollen. At no time did she complain of pain except during the treatments. This time the lateral incisor was extracted, and it was found that a large cyst had been formed at the apex of the root, communicating with the antrum. A counter-opening was made into the sinus at a point near the apex of the alveolus of the lost first bicuspid tooth, and the cyst and antrum were curetted and then irrigated with Thiersch solution. Following the operation there was a slight discharge of the typical secretion of cystic tumors for a few days, when it ceased altogether, and the openings were allowed to close. At the end of five years there had been no return of the disease.

Symptoms and Diagnosis.—The disease is of slow and painless growth, the very antithesis of suppuration or empyema of the antrum, which is rapid and painful in its development. Sooner or later the cheek becomes prominent and rounded, sometimes considerably enlarged; protrusion of the eye may occur, the nose is forced to the opposite side, the nasal fossa becomes occluded from bulging of the nasal wall of the antrum, and sometimes the palate is depressed to such an extent as to interfere with deglutition. The tumor may be soft and elastic in some places, and hard and resisting at others.

Pressure over the elastic portions gives the parchment-like crepitation which accompanies bone that is greatly thinned and expanded. There is usually no discharge from the nose. In many respects it closely resembles in appearance solid tumors of the jaws. Errors in diagnosis on this account have been frequently made, through which even excision of an entire jaw has been performed, and the mistake not discovered until it was too late to rectify it. The conservative surgeon will not fail to take the precaution of an exploratory puncture or incision when making the diagnosis of a doubtful tumor, as this will give him positive data upon which to base his opinion so far as the general character of the growth is concerned.

Prognosis.—The disease is prone to recurrence unless the treatment is thorough and heroic. The deformity of the face which has been occasioned by the expansion of the bone will gradually but finally disappear after the disease has been cured.

Treatment.—The treatment consists of the evacuation of the contents of the cyst by free incision at its most dependent portion, curetting its inner surface, irrigation with some antiseptic solution, and stimulating injections to promote granulation. In those cases where the distention of the bone has been considerable, a portion of the external wall should be removed, or crushed in.

Irrigation and the stimulating injections should not be discontinued until all tendency to the formation of the characteristic discharge has ceased.

Polypus of the Antrum.—A polypus is a small pedunculated cystic tumor growing from a mucous surface.

Polypi of the antrum are similar in structure to those found associated with the mucous membrane of the nasal passages. There are two varieties, the fleshy and the cystic. They are the result of hypertrophies—inflammatory hyperplasia—of the submucous connective tissue and of the mucous membrane.

When the tumor arises from the submucous layer the connective-tissue elements will predominate and result in the formation of a fleshy polypus; when it arises from the mucous membrane proper, the glandular structures will be in excess and a cystic form will be produced. An intermediate form is sometimes developed, having a loose fibrous structure with glandular elements, resulting in the formation of a semi-gelatinous polypus which very closely resembles the common form of polypus of the nose. Polypi of the antrum are usually very vascular, and cause considerable hemorrhage when they are surgically interfered with.

The disease is not a common one, and yet far more frequent than is generally supposed. Luschka in his investigations found that out of sixty subjects examined by him, five had polypi of the antrum, or one in twelve.

Symptoms and Diagnosis.—The symptoms do not differ materially from those of mucous cysts of the antrum.

Polypi may be present in the antrum for years, and the patient remain entirely unconscious of the fact until by their size they cause absorption of the nasal wall of the sinus or expansion of the bone, with external deformity of the face. The thin nasal wall of the antrum is the one which most frequently gives way from absorption, induced by the pressure of the cysts which occupy this cavity, and for this reason it is sometimes difficult to determine when the polypus occupies the nasal fossa also, whether it originated in the nose or in the antrum. John Bell, Syme, Vidal de Cassis, and others, have maintained that polypus never originated in the antrum, but was always an intrusion from the nose, while Paget, Fergusson, and others hold the opposite view, their opinions being based upon practical demonstration of the fact.

It would seem, therefore, that in most individuals who may perhaps have polypi in the antrum, the growths never reach a size to cause any inconvenience or deformity, and consequently their presence remains unsuspected.

When the polypus is of sufficient size to have intruded into the nasal passages, the nostril will be more or less completely occluded. Damp weather seems to cause them to swell and more completely close the nasal passages.

Prognosis.—The prognosis of polypi of the antrum is generally

considered very good, but according to Heath in some instances they seem to have a malignant character, or at least are the forerunners of malignant disease in the antrum and jaw. The writer's experience has been so limited in this particular direction that he does not feel qualified to hazard an opinion upon the matter, therefore accepts the teaching of the eminent authority just mentioned, especially as polypus of the nose in certain cases seems to possess a tendency to malignant degeneration.

Treatment.—The treatment of polypus of the antrum, if it succeeds, must be somewhat in the nature of a radical operation. Thorough extirpation of the growth, either through the nasal wall, the external wall, or the floor of the antrum, is indicated.

The first is to be preferred when it can be accomplished in the thorough manner necessary for a cure, as it does not require an incision through the external tissue of the face nor the loss of several teeth, as would be the case if the sinus was to be reached, on the one hand through the cheek, or on the other through its floor. Considerable difficulty is sometimes experienced in trying to remove such growth in the antrum through the nostril, but with polypus forceps properly curved, and a goodly allowance of skill and patience, the antrum may be reached through a previously made opening in the nasal wall, and explored to its farthest extremity. There is, however, an element of uncertainty always present in this operation, as to whether the growth has been entirely removed or not.

If the antrum is opened through the external wall the superior maxillary bone must be laid bare, by laying back the lip and cheek as in the operation for exsection of this bone, and the wall of the antrum penetrated with a trephine, chisel, or the surgical saw or bur.

If the opening is made through the floor of the antrum, two or three teeth must first be extracted, and afterward the bone can be cut away with the surgical burs. A sufficiently large opening must be made to permit exploration with the index finger.

The after-treatment consists of the usual irrigation of the sinus with non-irritating antiseptic solutions, until the tissues of the antrum have healed and the external wound has closed.

REVIEW.

CHAPTER XXXVI.

What varieties of tumors are found in the antrum?
Name the common cystic tumors found in this cavity.
What is the cause of mucous cyst of the antrum?
What are the peculiarities of its formation?

What is the character of its contents?

Are cysts of the antrum always single?

Would such an aggregation render the treatment more difficult?

Are cysts of the antrum always developed from the mucous membrane?

Describe the other forms of cysts which do not arise from the mucous membrane.

Give the symptoms of cyst of the antrum.

What are the diagnostic symptoms?

How may errors in diagnosis be avoided?

What is the prognosis?

Describe the surgical treatment.

What is a polypus?

How many varieties are there?

How are they designated?

What is the cause of their formation?

Which variety is developed from the submucous layer?

From what is the cystic variety developed?

What is the character of the intermediate variety?

All being usually very vascular, what may be expected when removing them surgically?

Is the disease common?

What was the result of Luschka's investigation upon this point?

What are the symptoms?

Is the presence of polypi in the antrum always recognized?

Which wall of the antrum in these cases most frequently gives way under the process of absorption?

May the polypus be located in the nose and enter the antrum by intrusion?

Give the opinion of John Bell and others upon this point.

What is the opinion of Paget and Fergusson?

What takes place in the nasal passages from intrusion of a polypus of the antrum?

How are the polypi affected by damp weather?

What is the prognosis?

Do polypi ever have malignant tendencies?

What should be the character of the surgical treatment?

Of what should it consist?

How may extirpation be accomplished?

Which method is to be preferred?

Describe the operation for opening the external wall of the antrum.

Describe the operation for opening the floor of the antrum.

What is the after-treatment?

CHAPTER XXXVII.

DISEASES OF THE SALIVARY GLANDS.

Inflammation of the Parotid Gland, parotitis, or mumps, is a specific infectious disease, which affects one or both parotid glands. It occurs most frequently in young males, and most commonly during the period of adolescence. A diphtheritic form of the disease is sometimes observed, but its most common form is the simple inflammation of the gland known as mumps. The disease often assumes an epidemic character, and spreads throughout schools and communities.

Symptoms and Diagnosis.—The affection may be ushered in by a rigor, nausea, elevation of temperature, and a general feeling of lassitude. In a day or two there is dull pain at the back of the jaw, and considerable local swelling, which may interfere with mastication and deglutition. In the milder forms of the disease the patient only complains of slight stiffness of the jaws, and pain when masticating food or upon taking acids into the mouth; while the constitutional disturbances may be so slight as to escape observation. The disease may appear first upon one side, then upon the other, or both sides may be affected simultaneously. One attack gives immunity for the future. The pathology of the disease is still in obscurity. The chief danger to be apprehended in this disease is metastasis to the testes, mammæ, and ovaries. Happily these complications are rare. In about three per cent. of the cases metastasis of the testes occurs, producing a true orchitis, which is rarely found as a primary affection under any other circumstances. The duration of the disease is commonly from eight to twelve days; the affection usually terminates by resolution, and rarely ends in suppuration.

Inflammation and suppuration of the glands is sometimes a sequel of typhoid fever, puerperal fever, and erysipelas, and of scarlet fever and variola in children; or it may be associated with pyemia. Involvement of the neighboring lymphatics in these cases is to be expected. Under such circumstances the disease is attended with great prostration, high temperature, and delirium. The pus coming from such abscesses is of fetid odor.

Prognosis.—The prognosis in the latter cases is very grave indeed, for if the disease is left to itself the abscess may open into the auditory

meatus, or, as an exceptional complication, it may pass downward to the chest, or extend in an upward direction along the sheath of the carotid artery to the skull, or behind the pharynx, or upward to the temporo-maxillary joint.

Treatment.—In the milder form of the disease the only treatment that is required is protection from taking cold. In the more ordinary form, absolute rest and protection from sudden change of temperature are demanded. If the temperature runs high, this should be controlled by appropriate remedies. Dry heat applied to the side of the face in the form of heated flannels, a hot brick wrapped in flannel, or a rubber bag filled with hot water, will mitigate the pain. Liquid food will be required for a few days. Medicines beyond a saline cathartic and anodynes are rarely needed. Occasionally the attack will leave the patient in a debilitated condition demanding tonics. In the more serious form of the disease, involving suppuration of the glands, the pus should be evacuated as soon as fluctuation can be discovered, and the cavity irrigated with bichlorid of mercury solution, 1 to 2000, drainage provided for, and the wound dressed antiseptically. When the pus is superficially located, it is best to make the incision in a line with the course of the facial nerve, so as to avoid the possibility of producing paralysis of the facial muscles by severing this nerve. If a deep incision is necessary, it should be made upon the line and in front of the external carotid artery. The exhibition of quinin and iron, with nourishing food, should constitute the systemic treatment, and in cases accompanied with great prostration stimulants should be freely used.

Salivary Calculi.—A salivary calculus is a calcareous deposit in a salivary gland or its duct.

These concretions occasionally form within the salivary ducts, and much more rarely within the gland itself. Salivary calculi are seldom found within the parotid gland or its duct. The most common location of these concretions is in the duct of Wharton; more rarely in the ducts of the sublingual gland. Calculi of the submaxillary and sublingual glands are somewhat rare, but the greater number of the reported cases have been found associated with the submaxillary glands. Fütterer found one hundred and fifty-eight cases of salivary calculi in the submaxillary and sublingual glands, mentioned in medical and surgical literature, but he could only secure access to the full reports of sixty-seven of this number. Out of the sixty-seven cases, he found nine were located in the submaxillary gland itself; six were found in the sublingual gland or its ducts, and the remainder—fifty-two—were located in the duct of Wharton. Six cases only of calculi in the parotid gland or its duct were found in this search. To recapitulate: Out of seventy-three cases of salivary calculi, affecting the parotid, submaxillary, and sublingual glands and their ducts, six were associated with the parotid

or the duct of Stenson; nine with the submaxillary gland alone; six with the sublingual gland or its ducts, and fifty-two with the duct of Wharton.

Salivary calculi are seen most frequently between the ages of twenty and forty years. The youngest person reported in the cases gathered by Fütterer in which a calculus was found, was twelve years of age, and the oldest was seventy years. Burdel reported a case to the French Academy, in which a concretion was found in the sublingual duct of an infant only three weeks old. This calculus was about the size of a grain of wheat, yellow in color, its surface granular, wrinkled, and apparently formed of minute cones cemented together at their base. The analysis showed it to be composed almost entirely of calcium phosphate, and a small proportion of nitrogenous organic matter. This no doubt was a concretion of pre-natal formation.

Causes.—The causes of salivary calculus may be the presence of a foreign substance lodged in the duct, or bacteria, most likely the *Leptothrix buccalis.* Fütterer was unable to demonstrate leptothrix, but he still argues that they are the most likely nidus around which the deposit is formed. The presence of a foreign substance in the center of the calculus has been demonstrated in a few cases; but in the great majority no such evidence could be found. There seems to be a close connection between the formation of calculi in the salivary glands and the formation of calculus upon the teeth, as persons with salivary calculi of the glands usually have considerable calcareous deposits upon the teeth. Richet first called attention to this fact. It has been the general impression among English surgeons that these deposits in the salivary glands were closely connected with the gouty diathesis.

It has been generally stated that these calculi were composed principally of calcium carbonate and phosphate, and magnesium phosphate. Fütterer has examined several specimens, and finds calcium phosphate largely in excess of the carbonate. Garretson reports the examination of a calculus taken from the duct of Wharton, which showed the same composition. Ptyalin, xanthin, and uric acid were also found in them by Fütterer, which would seem to prove the connection of the formation of these concretions with the gouty diathesis. The specific gravity of the calculi in the reported cases varies so greatly that no reliance can be placed upon the result of this part of the examination, from the fact that some were weighed in the dry, and others in the fresh state. Sections of the calculi all show a lamellar arrangement, beginning at the center, which indicates the manner of their increase in size. These calculi form very slowly; perhaps years elapse before their presence gives rise to any serious complications.

Symptoms.—The symptoms are acute inflammation, accompanied by extensive swelling in the floor of the mouth, at the side of and

beneath the tongue. This organ is sometimes lifted up and pushed back into the fauces. Fullness of the submaxillary triangle of the neck is also observed. The pain is frequently very severe. The constitutional symptoms are elevation of temperature, nausea, dizziness, and a general feeling of prostration. Upon an examination of the swelling by palpation, the index finger of one hand within the mouth, over the swelling, and the other upon the neck beneath the gland, the form of the swollen gland and duct may be readily outlined. Occasionally the calculus can be found as a hard mass within the duct. This, however, would not be possible when the swelling was at all extensive, or the calculus was located within the gland. Inflammation and suppuration are commonly associated at various intervals with the presence of these formations in the gland or its duct. These calculi are usually oblong or spindle-shaped in form. In size they have varied from one grain to two hundred and seventy, the latter being the largest recorded calculus found in a human subject. The largest calculus in measurement was six centimeters in length, and five and one-half in

FIG. 133.



SALIVARY CALCULUS FROM THE SUBMAXILLARY GLAND OF A HORSE. (Reduced one-half.)

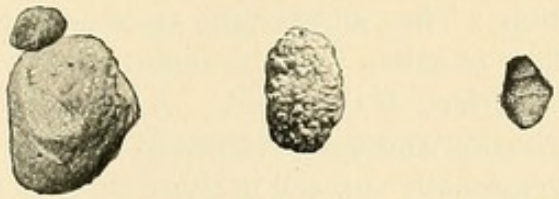
width. Usually there is but one calculus found in the duct or the gland, but occasionally two or more are found lying together, facets having formed at the points of contact. Garretson mentions a case in which both of the ducts of Wharton were filled with small calculi. Salivary calculi are quite common in the larger animals, like the horse and the ox. The accompanying illustration (Fig. 133) is a calculus taken from the submaxillary gland of a horse; it weighed eleven and one-half ounces in the dried state, and measured six inches in length and two inches in width, having a circumference at its largest part of six inches. Although inflammation and suppuration are commonly recurring conditions, an external fistula as a result is an exceedingly rare occurrence. Fistula following operation through the external tissues has occasionally occurred, and it is somewhat difficult to heal. Relapses after complete removal of the calculi are very rare, and it is more than likely that in most of those cases reported as relapsing, all of the calculi were not removed at the first operation.

Diagnosis.—The diagnosis is usually a simple matter. If the duct is still patulous, a small probe—preferably made of untempered steel,

as this metal conducts sound, and the vibrations produced by contact are much better than the ordinary silver probe—may be passed into it until it reaches the calculus, which will be distinguished by the rough, grating sensation imparted to the fingers upon bringing the probe in contact with the stone. If the duct is not open, an exploring needle may be thrust into the swelling at various points, when if a calculus is present it will most likely be found. Cases with extensive swelling of a chronic character have been mistaken for malignant growths. The difficulty in diagnosis is much greater when the calculus is situated in the gland than when located in the duct.

Treatment.—The usual method employed for removing a salivary calculus from either of the glands or their ducts is by an incision made within the mouth over the point at which the calculus lies imbedded, or by dilating the orifice of the duct, and expressing the calculus from its bed; or by lifting it out with the forceps. Occasionally the swelling will be so great that none of these methods will avail, and this is especially true of those cases in which the calculus is lodged within the gland. Fütterer reports a case of this character which was seen by the writer in consultation, in which two calculi (Fig. 134) were found imbedded

FIG. 134.



SALIVARY CALCULUS FROM THE SUBMAXILLARY GLAND. (After Fütterer.)

in the submaxillary gland, one of which weighed one grain, and the other twenty-three grains. In this case there was extensive suppuration and great swelling which had lasted for several days. The presence of the calculi was demonstrated by passing a fine probe into the duct of Wharton. After making an extensive incision down to the calculi, and repeated unsuccessful trials to grasp and remove them, the effort was abandoned, and the wound packed with gauze. On the next day the packing was removed, which was followed by a profuse discharge of pus. The calculi were then easily grasped and removed with a pair of long slender forceps.

Operations through the external tissues for the removal of calculi are to be deprecated, on account of the dangers of forming salivary fistulæ. The after-treatment in these cases is simple. Thorough cleanliness of the mouth and wound, maintained by the use of antiseptic solutions, is all that is required.

Salivary Fistulæ.—Salivary fistula is a rare, but nevertheless a very troublesome affection. It is usually associated with the duct of

the parotid gland, and is very rarely met with in the submaxillary or the sublingual glands.

Causes.—It may be caused by traumatic or surgical injuries, or by inflammatory conditions resulting in abscess, or by ulceration. Lacerated and gunshot wounds of the cheek, and surgical operations requiring incisions of the cheek, are the most frequent causes of the affection. The inflammatory conditions which may result in fistulæ of the parotid gland are suppurative parotitis, suppurative inflammation from injury and the presence of calculi, ulceration following mercurial ptyalism, and gangrenous stomatitis. Fistula of the parotid gland has sometimes occurred as the result of operations upon this organ for the removal of tumors.

Diagnosis.—The affection consists of an outward opening of the duct of Stenson upon the external surface of the cheek, through which the saliva flows over the cheek instead of into the mouth. During the stimulation of the gland induced by the act of mastication or the odors of appetizing foods, the saliva pours out over the cheek, and becomes very annoying. Sometimes the cheek is excoriated by the fluid, and unhealthy looking granulations spring up about the orifice of the fistula.

Treatment.—A variety of operations have been proposed for the cure of this affection. They all have the same end in view, namely: to re-establish the flow of saliva into the mouth, and to close the fistula upon the external surface of the cheek. A common method of treatment is by cauterization and compression of the fistula. This form of treatment may occasionally succeed in those cases in which the natural orifice in the mouth still remains patulous. In the greater number of cases, however, the orifice in the mouth has been obliterated and calls for an operation to re-establish it.

Agnew's method consists of passing a curved needle, armed with silk thread, around the duct, posteriorly to the fistula, from within the mouth. The needle should be entered and emerged at as nearly as possible the same point, care being taken to include the duct, but not the skin, within the loop. The thread is then to be tightly knotted, and the ends cut off. The effect of this operation is to produce ulceration within the cheek, while the ligature cutting its way through the confined tissue, separates after a few days, and leaves a new and artificial duct through which the saliva may find a free passage into the mouth. If the external fistula does not close immediately, the edges may be pared and brought together with sutures, and covered with a collodion dressing.

Deguise's method consists of first making a puncture through the fistulous opening in the cheek obliquely backward to the inner surface of the cheek, and passing one end of a leaden wire through it.

Second, through the same opening another puncture is made, which is directed obliquely forward, and through which the other end of the wire is passed. The ends of the wire are then brought together and tied. The loop passing through the fistula conducts the saliva into the mouth, and the fistula closes in a few days. If it does not, it should be closed by the method just described.

Van Buren succeeded in closing a salivary fistula, the result of a gunshot wound, by transferring the fistulous orifice from the outer to the inner surface of the cheek. This may be accomplished by first passing a fine silver wire through the skin at opposite points on the edge of the fistulous orifice. The next step in the operation is to loosen the fistulous orifice and the duct from the surrounding tissue, for the distance of about half an inch backward, then make an incision through the wound to the inner side of the cheek, drawing the fistulous orifice through it, and retaining it in its new position by means of the wire. The external opening is to be closed with silver wire sutures. The wound should be treated antiseptically.

REVIEW.

CHAPTER XXXVII.

What terms are applied to inflammation of the parotid gland?
What is parotitis?
In what forms does the disease appear?
What character does the disease sometimes assume?
What are the symptoms of the disease?
How does the milder form differ from this?
Does it affect the glands simultaneously?
Is immunity obtained by a single attack?
Is the pathology known?
What are the chief dangers of the disease?
In about what per cent. does metastatis occur in the testes?
What is the usual duration of the disease?
How does it generally terminate?
With what other diseases is parotitis sometimes associated?
Does lymphatic involvement take place?
What are the constitutional conditions in this form of the disease?
What is the character of the pus?
Give the prognosis.
In what directions may the pus burrow?
How should the milder cases be treated?
What should be the treatment in the common form?
What should be the treatment in the severer form?

Where should the line of incision be made in operating upon suppurating glands?

How should the constitutional condition be treated?

What are salivary calculi?

Are they common or rare?

In which glands are they most frequently found?

What do Fütterer's statistics prove?

At what period of life do salivary calculi most frequently occur?

What was the age of the youngest case found by Fütterer?

What was the age of the oldest?

What was the age in Burdel's case?

How is the presence of a calculus in so young a child explained?

What are the causes of the disease?

With what local and constitutional conditions are these formations closely connected?

What is the composition of salivary calculi?

What is the appearance on section?

Do such calculi form rapidly or slowly?

Describe the symptoms.

What is the usual shape of these calculi?

What are the variations in weight?

What are the measurements of the largest calculus removed from the human subject?

Do they occur singly, or how?

Are they found in animals?

Is external fistula common?

Do relapses occur?

How may the diagnosis be made?

In what kind of cases is the diagnosis most difficult?

How may the calculi be removed?

What is the after-treatment?

Is salivary fistula a rare affection?

With which glands is it most commonly associated?

What are its causes?

What are considered as the most frequent causes?

What are the inflammatory conditions occurring in this gland which may result in fistula?

Describe the appearance of salivary fistula.

What condition is caused by the flowing of the secretion over the cheek?

What are the main objects of an operation?

Describe Agnew's operation for the cure of salivary fistula.

What is the effect of this method of operation?

How should the external fistula be closed?

Describe Deguise's operation.

How did Van Buren succeed in closing a salivary fistula resulting from a gunshot wound?

CHAPTER XXXVIII.

NEURALGIA.

Definition.—Neuralgia (Greek νευρον, nerve; ἀλγος, pain).

Neuralgia is a severe paroxysmal pain in the area of distribution of a nerve, or along its course.

It has become customary to designate all pains which occur in paroxysms, unattended with local or general elevation of temperature, and distributed along the course of nerve-trunks or nerve-branches, for which no adequate cause can be assigned, as neuralgia. (Putnam.)

Neuralgia is not a disease, nor a morbid condition in the sense of its having an individuality, but is a phenomenon, or an expression of a disease or of a morbid general or local condition. Neuralgia has been called "the prayer of the nerves for blood," and "the cry of the hungry nerves for food," but these suggestions as to the etiology of neuralgia do not cover all the causes which produce this most distressing and painful phenomenon.

Neuralgia is a medical rather than a surgical affection, and rarely comes under the observation of the surgeon except as the result of injury, or the implication of the nerves in the healing of wounds or cicatrices, or when medical treatment has failed to relieve the pain and surgical operation is sought as a last resource.

The conditions which are productive of neuralgia are many and varied, and consist chiefly of diseases which lower the vital powers of the system, such as anemia, or those which interfere with such functions as the circulation, respiration, digestion, assimilation, secretion, and elimination; the presence in the system of abnormal substances, as in gout, rheumatism, diabetes, malaria, nephritis, chronic pyemia, syphilis, and metallic poisoning; local conditions which cause reflex peripheral irritation, such as diseases of the teeth, eyes, ears, stomach, uterus, and ovaries; chronic inflammation of the nerve or its sheath; pressure from abnormal growths within the bony canal through which the nerve-trunk passes, or pressure from tumors, and localized anemia or congestion of nerves or nerve-centers.

Neuralgia may therefore be the result of an actual diseased condition of the nerve, as for instance in a neuritis, or it may exist with no discernible structural change in the nerve-tissue or the nerve-centers.

The changes which may take place in the nerve-tissue under such conditions may be simply molecular, and these, with our present means of examination, are not capable of being demonstrated; but the way in which certain forms of neuralgia behave, as for instance their sudden disappearance from one part to reappear in another and perhaps remote location, or their complete disappearance after a short period, confirms the general opinion that these forms are not due to any organic change in the nerve itself.

On this account it has become customary to divide all neuralgias into two distinct classes, placing those in which there are appreciable changes in the nerve-tissue under the head of *symptomatic neuralgia*, and all others under the head of *idiopathic neuralgia*.

In symptomatic neuralgia the pain is dependent upon the neuritis or other structural changes in the nerve-tissue or its sheath, while in idiopathic neuralgia the pain does not depend upon any discoverable change or alteration in the nerve-tissue. Of the pathologic anatomy of such a condition there is absolutely nothing known.

In the character of the pain in these two varieties of neuralgia, there is very little difference; but the symptoms which accompany the pain are not alike. The principal difference in the symptoms lies in the greater degree of pain manifested in a neuritis, and the sensitiveness which exists over the nerve-trunk. (Sinkler.)

All neuralgias have one common tendency, which is manifested in a greater or less degree, viz: periodic recurrences, but the degree of periodicity varies greatly. These recurrences are most regular and best marked in the malarial neuralgias, and in those dependent upon neurotic conditions like migraine and the periodic headaches.

Neuralgia affecting the viscera occurs with less regularity. One variety of neuralgia affecting the ophthalmic division of the trifacial nerve evinces a tendency to daily recurrences at the same hour (usually about nine A.M.) for a certain period. This is particularly marked in those cases dependent upon malarial influences, and catarrhal affections of the frontal sinuses. The writer had under observation recently a case of this character which was due to catarrhal inflammation of the frontal sinuses. The pain was intense for an hour to an hour and a half, the paroxysms being from three to five minutes apart. The patient complained of a stopped-up feeling in the nasal passages and a sense of fullness in the frontal region on rising in the morning. This lasted until the paroxysms ceased, when the sense of fullness also disappeared. The patient made constant effort during the period of the paroxysm to clear the nasal passages, and he believed that as soon as this effort was successful the pain ceased.

The neuralgias are also again divided according to their location and their symptomatology, into,—

1. Superficial.
2. Visceral.
3. Migraine and the migrainoid headaches.

The *superficial* variety of neuralgia is limited to the course and area of distribution of a single superficial nerve or group of nerves, like the sciatic and the trifacial.

The *visceral* forms of neuralgia are less definitely localized by the sensations of the patient than in the superficial variety, and as these nerves are deep-seated it is difficult to indicate which are at fault.

Migraine is a complex sensory neurosis characterized by pain in various locations of the cranium; the occipital region, the vertex, the frontal, or the temporal region.

Causes.—The causes of neuralgia may be divided into two forms, predisposing and exciting.

The predisposing causes are,—

1. Hereditary tendencies.
2. Periods of life at which certain critical changes take place.
3. Influences associated with sex.
4. The effects of constitutional diseases, such as anemia, gout, rheumatism, phthisis, diabetes, nephritis, malaria, syphilis, and metallic poisoning.

The exciting causes are,—

1. Atmospheric conditions, as indicated by a low barometer; and the local action of heat and cold.
2. Injuries and direct irritation of the nerves.
3. Indirect irritation of the nerves (reflex).
4. Acute febrile diseases.

Predisposing Causes.—*Hereditary Tendencies:* The fact of the inheritance of neuralgic tendencies by certain families who give other signs of a neuropathic taint, is so well established that it does not admit of question. The tendency is most marked in the case of migraine, and other periodic headaches. It is also noticed in visceral neuralgias, and particularly so in facial neuralgias, though less marked in the other forms of superficial neuralgias. (Putnam.)

Age: Age is an important factor in the predisposition to neuralgic affections, which are most common in middle life, and at those periods which mark the growth and the decline of the sexual functions. According to Anstie, these conditions when once established are inclined to continue into advanced age, but fortunately cases beginning at this period are relatively rare, though exceedingly intractable.

Childhood and youth are usually exempt from superficial neuralgias, though migraine and periodic headaches may develop in children of neurotic temperament. These conditions may later in life give way to more serious neuroses.

Sex: Women, as a rule, are more liable to certain forms of neuralgia than are men. This is particularly noticeable in neuralgias of the trifacial, occipital, and intercostal nerves; while men suffer most frequently from the brachial, crural, and sciatic neuralgias. Putnam thinks this is due to the stronger neurosal element in women, and the neuritic element in men.

Constitutional Diseases: Any and all constitutional disorders which by virtue of their action produce an impoverished condition of the blood or retrograde tissue-changes, or the disturbance of vital or other important functions of the body, undoubtedly predispose to neuralgia and other neurotic affections. Among these predisposing constitutional causes may be mentioned phthisis, anemia, gout, rheumatism, diabetes, malaria, nephritis, chronic pyemia, syphilis, indigestion, malassimilation, perversions in secretion and elimination, conditions of the vaso-motor system which produce local congestion or anemia, and the presence within the system of certain toxic substances such as the metallic poisons. Ehrmann has published an account of four cases in which small doses of potassium iodid produced trigeminal neuralgia.

Lead, arsenic, antimony, and mercury are all capable under favoring circumstances of so impairing the nutrition of all nerve-tissue as to seriously predispose it to neuralgic attacks.

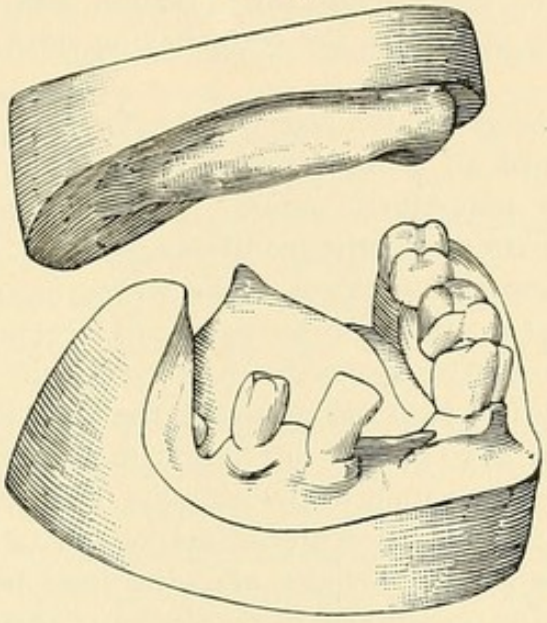
Exciting Causes.—*Atmospheric conditions, and Thermal influences:* It has long been known that certain atmospheric conditions, especially those preceding a storm, were likely to excite attacks of neuralgia, and that the various forms of neuralgia were more prevalent in the cold and damp seasons of the year, in cold and damp localities, and in persons whose occupations compelled them to work in a cold and moist atmosphere, or who were subject to frequent and extreme changes of temperature, than under different circumstances.

In a series of observations conducted by a military officer under the direction of S. Weir Mitchell, in relation to the conditions of the atmosphere preceding a storm and the coincident attacks of neuralgia in the stump of an amputated limb, from which the officer suffered most intensely, it was found that the attacks of pain were accompanied by a falling barometer, though the severity of the pain did not necessarily bear a proportionate increase with the rapidity or the extent of the fall. The moisture of the atmosphere seemed to have a certain effect, but the attacks occurred even when the storm center was so far removed that no local rain-fall took place. The electrical disturbances of the atmosphere could not be studied with accuracy, but there seemed to be a certain relationship between the attacks of pain and the appearance of the aurora borealis. (Putnam.)

Injuries and Direct Irritation of the Nerves.—Among the principal exciting causes of this class may be named wounds and injuries to

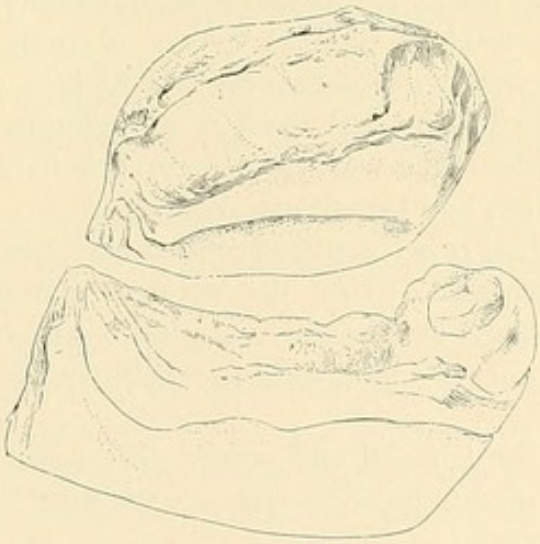
the nerves; impingement of nerves within cicatricial tissue; pressure from neoplasms and certain inflammatory swellings (gumma, etc.); narrowing of the bony canals and foramina, and aneurisms.

FIG. 135.



MALPOSITION OF THE RIGHT RAMUS, THE RESULT OF PARTIAL EXSECTION OF THE JAW FOR SARCOMA, WHICH CAUSED PERSISTENT TEMPORO-MAXILLARY NEURALGIA.

FIG. 136.



FINAL RESULT, ONE YEAR AFTER OPERATION. TEETH WERE LOST BY REASON OF PYORRHŒA ALVEOLARIS.

The writer reported a case at the Ninth International Medical Congress, of persistent neuralgia of the temporo-maxillary articulation of eight years' standing, which was due to the malposition of the right ramus, caused by exsection of a portion of the jaw,—from the angle to the first bicuspid tooth,—and contraction of the cicatricial tissue, which

was entirely cured by an operation which replaced the ramus in its normal position and thereby relieved the tension upon the articular ligaments of the joint. The case has already been referred to in another chapter as an example of bone-grafting. Fig. 135 is an illustration of the position of the ramus before the operation, and Fig. 136 shows it one year after the operation, the teeth having been lost by pyorrhea alveolaris.

Indirect Irritation of the Nerves.—Under this class of exciting causes are grouped all those disorders which produce these effects through reflex or sympathetic action. As an illustration, diseases of the uterus and ovaries not infrequently cause reflex facial, mammary, intercostal, and gastric neuralgia. The writer for several years had under observation a sufferer from dyspepsia accompanied by severe gastralgia and reflected neuralgic paroxysms in the left brachial plexus. The reflected pain was always in the same location, and often of so severe a type as to greatly interfere with the use of the arm upon the following day on account of the soreness of the muscles.

The eye is an important center of nervous irritation, and errors of refraction, even when quite slight, are sometimes productive of migraine. Inflammatory conditions of an acute and chronic nature affecting the maxillary and frontal sinuses are also productive of neuralgic attacks.

Acute Febrile Diseases.—Acute fevers are occasionally the exciting cause of neuralgia. Thus, Nothnagel describes neuralgias which came on during the first week of typhoid fevers. Putnam mentions a case which came under his own observation, in which "a severe facial neuralgia appeared during the first week of an insidious attack of pneumonia in an individual who was not of neuralgic habit, and before the fever or inflammation had become at all severe."

It is possible that other acute affections may have a similar effect.

Trifacial Neuralgia.—From the surgical standpoint the superficial forms of neuralgia are the only varieties of especial interest to the surgeon, as these only are amenable to treatment by surgical procedures.

Neuralgia of the trifacial and sciatic nerves, the brachial plexus, and the neuralgia of stumps and scars, are the most common forms of the superficial affection.

Neuralgia of the trifacial nerve (tic douloureux) is the form which most frequently comes under the observation of the dentist and the oral surgeon.

Trifacial or trigeminal neuralgia appears in two forms, viz: *acute* and *chronic*.

The *acute* form of the affection is frequently associated with or dependent upon acute inflammatory conditions of the teeth and alveolar

processes, acute affections of the eye and ear, and of the maxillary and frontal sinuses. This form of neuralgia is usually of short duration, and generally disappears upon the subsidence or the removal of the exciting cause.

The *chronic* form is often persistent in its character; the exciting cause difficult to find; and the affection does not always disappear upon the removal of the supposed cause on account of the structural changes which may have taken place in the nerve-tissue or the sheath of the affected branches or of their blood-vessels. It not infrequently happens that more than one abnormal local or constitutional factor may be involved in the causation of the various forms of neuralgia; failure in one direction should therefore lead to renewed search in some other, with the hope of finally discovering the other factors in the case, and removing them if possible.

Neuralgia may exist in any of the nerves as a result of neuritis, but it is most frequently observed in the sciatic and trifacial nerves.

The frequency with which the various branches of the trifacial nerve are the seat of neuralgia may be stated to occur in the following order: the superior maxillary division, the inferior maxillary division, and lastly, the ophthalmic division.

In neuralgia of the superior maxillary division, the pain is sometimes located in the dental branches, being referred to the upper teeth, gums, and maxillary bone; at other times it affects the infraorbital branch, the pain being referred to the integument of the cheek, the side of the nose, and the upper lip.

Infraorbital neuralgia is frequently associated with neuralgia of one of the other branches of the fifth nerve, usually with the first division.

When affecting the inferior division, the pain is most frequently located in the lower teeth, the gums, and the integument of the lower lip and the chin.

When located in the ophthalmic division it most often affects the supraorbital branch, the pain spreading out over the forehead, the eyebrow, and the upper eyelid. It is a common occurrence for the patient to refer the pain in the beginning of a paroxysm to the point of exit of the nerve from its bony canal, for instance at the infraorbital, mental, and supraorbital foramina.

Symptoms.—Neuralgia of the fifth nerve rarely appears until after middle life, and in old persons it frequently resists the most intelligent treatment. The fact that the affection does not appear as a rule until after middle life, when the senile changes are beginning to take place, would seem to indicate a connection between these changes and the appearance of this form of neuralgia.

The pain is of the most excruciating character. There is no other

disease, with possibly the exception of tetanus, which from the severity of the suffering is so calculated to arouse the sympathies and commiseration of the surgeon and those in attendance upon the case, as the severe forms of tic douloureux.

In tic douloureux, or "epileptiform neuralgia," as Trousseau termed it, the pain comes on suddenly, sometimes preceded as in epilepsy by an aura. The character of the pain is acute, occurring in distinct paroxysms, with longer or shorter intervals, sharp, stinging, or lancinating, gradually increasing in intensity for a few moments, until it reaches a climax, and as gradually and quickly subsiding, to be again followed by another paroxysm of equal degree of intensity. The pain is so fearfully severe in some cases as to cause the patient to moan or cry aloud with every paroxysm, and after the paroxysm is past to sit in terror waiting the onset of the next attack. This may continue for hours without cessation, or the paroxysms may last but for an hour or two, the remainder of the day being quite free from pain; or it may be excited at any time by talking, laughing, mastication, or even passive movements of the muscles of mastication, speech, or expression. A slight noise or a light touch may precipitate a paroxysm.

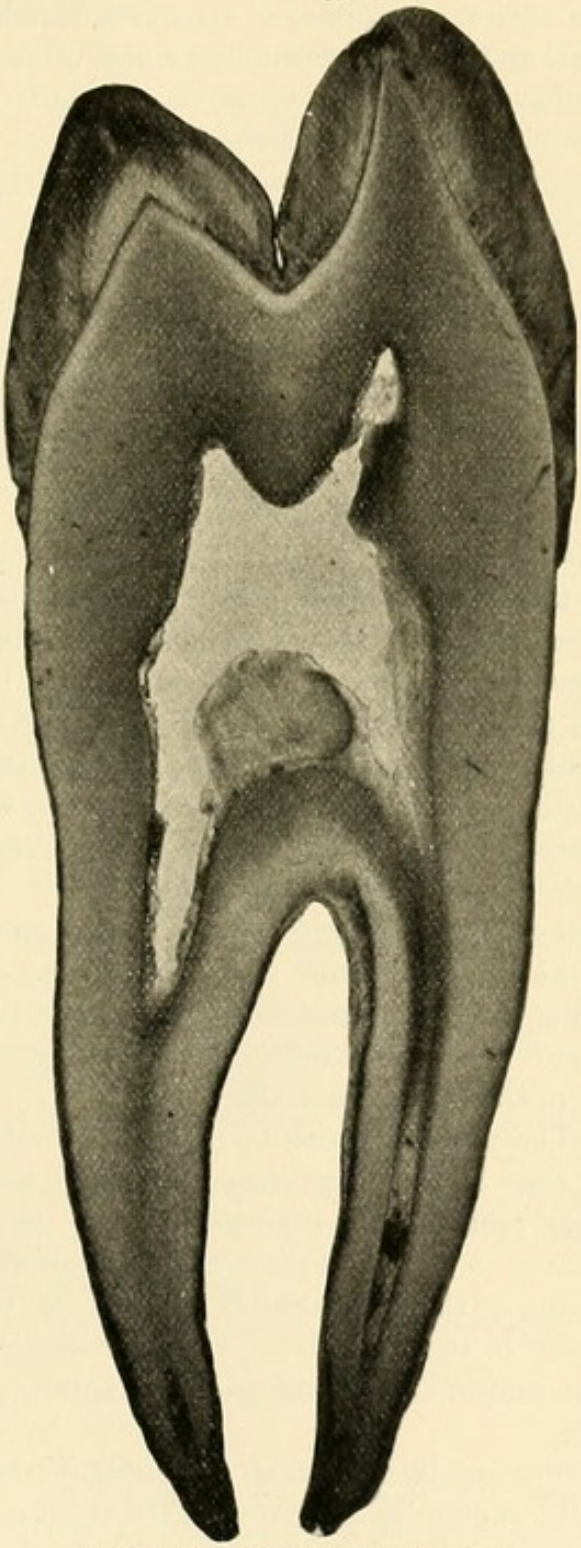
In some cases the patient will be entirely free from pain during the night, but upon awakening in the morning the slightest movement of the muscles of the face precipitates the paroxysms of pain. Occasionally the pain will be severe during the night, and the patient comparatively free during the day, and able to go about his daily vocation; while in others the pain may be induced at any time by the movement of the muscles, so that the question of taking food becomes for several days at a time one of great dread; in fact, patients frequently abstain from the taking of aliment except in a liquid form for days together. The pain and the dread of the returning paroxysms make life a burden.

Besides the pain, other symptoms of lesser note occur in facial neuralgia. Increased secretion of the lachrymal, salivary, and mucous glands is a frequent occurrence. The hair of the face or side of the head becomes dry and brittle, and is inclined to fall out, or it may lose its color rapidly, regaining it after the attack has passed. (Putnam.) There is increased secretion of urine. Anstie noticed unilateral furring of the tongue. The muscles to which the pain is referred may sometimes become paretic. Temporary amaurosis, and sometimes loss of the eye of the affected side, may take place in tic douloureux. The senses of hearing, taste, and smell may likewise be temporarily lost.

Causes.—Among the more common exciting causes of trifacial neuralgia expressed in the various divisions of the fifth nerve, may be mentioned the following conditions: In the ophthalmic division inflammatory affections of the conjunctivæ, diseases of the globe of the eye and iritis, and catarrhal conditions of the frontal sinuses. In the

superior and inferior maxillary divisions, inflammatory conditions of the teeth and jaws, particularly pulpitis, pericementitis, and periostitis

FIG. 137.



INFERIOR MOLAR—VERTICAL SECTION.
Showing Pulp-nodule (enlarged).

of the alveolar processes; structural changes in the teeth, like interstitial calcification of the pulp, pulp-nodules, and exostosis of the root.

Fig. 137 is from a case of neuralgia of the third division of the trifacial nerve induced by the formation of a pulp-nodule. Difficult eruption of the teeth, particularly of the lower third molars; exposed sensitive dentine from caries, abrasions, or fractures which have caused a loss of the enamel; and lastly, inflammatory and other diseased conditions of the maxillary sinuses, which are particularly associated with neuralgia of the superior maxillary division.

Syphilitic manifestations of a secondary and tertiary nature may produce inflammatory infiltration of the nerve-sheath, or neuritis, and cause neuralgia in either or all of the branches of the fifth nerve. Osseous growths within the bony canals or the foramina through which these nerves pass is not an infrequent exciting cause of neuralgia. These growths may be in the form of tubercles arising from the walls of the canals, or in the form of a diffuse exostosis; in either case a narrowing of the canal or its foramen is the result, pressure upon the nerve is induced, and as the patient grows older there is caused a progressive neuralgia.

The various forms of irritation, both direct and indirect, not infrequently set up a neuritis in the various branches of the nerve, as demonstrated by Putnam and others. Dana found striking evidences of arterial disease in three cases of typical trifacial neuralgia, but no noteworthy changes in the nerves. He believes the cause of neuralgia to be obliterating arteritis, and gives as his reasons for this opinion that "the disease occurs at an age when degenerative changes begin in the arteries and follow a certain fixed distribution."

Tuffier found positive evidence of neuritis in one case examined by him of neuralgia of the inferior maxillary nerve. In this case the nerve was "swollen and reddened both within the dental canal and before its entrance." Sinkler also found like evidences of neuritis in a case of neuralgia of the inferior maxillary nerve.

Diagnosis.—The manifestations of trifacial neuralgia are so characteristic that an error in the diagnosis could hardly be made. Valliex discovered certain points of tenderness in cases of facial neuralgia which are designated as "points douleureux." These are located in the ophthalmic division, at the supraorbital foramen, on the upper eyelid, at the line of union of the nasal bone with the cartilage, at the inner angle of the orbit, and in the eyeball itself. Another point is near the parietal eminence.

In the superior maxillary branch the painful points are situated at the infraorbital foramen; at a point over the most prominent part of the malar bone; an uncertain point on the gum of the superior maxilla; a similar point upon the upper lip, and another upon the palate.

In the inferior maxillary division the painful points are found over the auriculo-temporal branch just in front of the ear; another over the

inferior dental foramen, and still another over the mental foramen. The presence or absence of these "points douleureux" are not positive diagnostic signs, though as a general rule tenderness will be found over the foramina named.

REVIEW.

CHAPTER XXXVIII.

Give the definition of neuralgia.

What forms of pain are termed neuralgia?

Is neuralgia a disease?

What has this phenomenon been called?

Is neuralgia a medical or surgical affection?

What are some of the principal conditions that are productive of neuralgia?

What changes may some of these conditions produce in the nerve?

Is it possible to demonstrate the molecular changes that may take place in the nerves?

What peculiar phenomena in the history of neuralgia would seem to indicate that the affection was not always due to changes in the nerve-tissue?

What is the difference between symptomatic and idiopathic neuralgia?

To what is the pain in symptomatic neuralgia due?

What does the pain depend upon in idiopathic neuralgia?

Are there any known pathologic changes in the nerve in idiopathic neuralgia?

What are the differences between these varieties of neuralgia in the character of the pain and symptoms?

What common tendency belongs to all neuralgias?

In what forms of neuralgia are these periodic recurrences most marked?

Do visceral neuralgias recur with the same regularity?

What forms of neuralgia sometimes have daily recurrences at the same hour?

What is the character and duration of the pain in these cases?

Upon what other basis may neuralgias be divided?

Name these forms.

Describe the superficial variety.

How does the visceral form differ from the superficial?

What is migraine?

How may the causes of neuralgia be divided?

Name the predisposing causes.

Name the exciting causes.

In what forms of neuralgia are the hereditary forms most marked?

How does age affect the different forms of neuralgia?

At what period of life is neuralgia most common?

What period of life is most exempt from neuralgia?

How does sex influence the predisposition to neuralgia?

How are these tendencies accounted for?

How do constitutional tendencies predispose to neuralgia?

Name some of the constitutional conditions which predispose to neuralgia.

Name some of the toxic substances that may be productive of neuralgia.

What are the atmospheric and thermal conditions which excite attacks of neuralgia?

What are the forms of injury and direct irritation that cause neuralgia?

What class of disorders produce neuralgia by indirect irritation?

What forms of acute febrile disease have been known to produce neuralgia?

What are the most common forms of superficial neuralgia?

What other term is applied to trifacial neuralgia?

In what forms does trifacial neuralgia appear?

What are the causes of the acute form of neuralgia?

What is the character of its course?

What is the character of the chronic form?

Is it possible that more than one abnormal factor may be present in the causation of chronic neuralgia?

In what superficial nerves is neuritis most frequently observed?

In which branches of the trifacial nerve is neuralgia most commonly observed?

Where is the pain located in the superior maxillary division?

Neuralgia of what other division may be associated with neuralgia of the infraorbital?

Where is the pain most frequently located when the inferior maxillary division is affected?

What is the location of the pain in neuralgia of the ophthalmic division?

To what point does the patient usually refer the pain at the beginning of a paroxysm?

At what period of life does neuralgia of the fifth nerve usually appear?

What would this seem to indicate as a cause of the affection?

What is the character of the pain?

By what condition is the pain sometimes preceded?

Describe a neuralgic paroxysm.

What are some of the conditions which excite the paroxysms?

What are some of the exciting causes of neuralgia of the ophthalmic division?

Name some of the more common exciting causes of the superior maxillary division.

How may syphilis become an exciting cause?

How may osseous growths produce neuralgia?

What sometimes results from direct and indirect irritation to a nerve?

Has neuritis been demonstrated in trifacial neuralgia?

To what does Dana think neuralgia may be due?

Is the diagnosis of trifacial neuralgia difficult?

Name Valliex's "points douleureux" of the ophthalmic division.

Where are they located in the superior maxillary division?

What is their location in the inferior division?

CHAPTER XXXIX.

TREATMENT OF TRIFACIAL NEURALGIA.

Trifacial neuralgia is sometimes amenable to treatment by certain drugs. Their therapeutic effect is, however, often very disappointing, and one after another may be tried with little or perhaps no benefit. Success in the direction of treatment by drugs will depend upon the age and the general condition of the patient, and the causative agents which are responsible for the affection. It must be regarded, however, as one of the most intractable of diseases. The long list of remedies which have been recommended from time to time attest the difficulties that are met with in attempts to eradicate the disease.

It is especially necessary in the treatment of facial neuralgia to look beyond the relief of the particular attack under observation and search for the cause or causes which have provoked the attack. These conditions have already been referred to, and should receive that treatment which is appropriate to the individual ailment.

It may be assumed, however, that in a majority of the cases of protracted neuralgia, neuritis is present, and this condition should receive appropriate treatment by local applications and galvanism.

As many cases of trifacial neuralgia are due to the impairment of the general health, remedies which are directed to the building-up of the system are sometimes successful in curing the affection.

Quinin, arsenic, and *iron* have been found useful in this direction; the precipitated subcarbonate of iron administered in large doses has been found exceedingly beneficial. *Gelsemium* has been found by many authorities to be one of the most potent remedies in the treatment of neuralgias of the fifth nerve. It gives its best results, however, in those cases which are dependent upon diseased conditions of the teeth. Sinkler has found it useful in neuralgia of all the branches of the fifth nerve. The writer has had a like experience in most of the cases in which it has had a fair trial. In one case, however, recently under his care, this drug at first gave complete relief administered in the form of *sulfate gelseminine,* gr. 1-30 every two hours until the constitutional effect was produced. In the next attack, which came on seven days afterward, it had no appreciable effect when carried to the point of drooping eyelids and dimness of vision. The fluid extract is

generally considered the best preparation, but because of its unreliability in strength the writer prefers the *sulfate gelseminine.*

Valerianate of zinc was at one time highly recommended, but it is not at the present time held in much esteem.

Cannabis indica is a remedy of value in some cases. To obtain the best results it should be given in full doses, and repeated as often as the patient can tolerate.

Seguin and others have highly recommended the use of *aconite.* This drug, to be of value, should be administered until numbness and tingling are felt in the lips and face. It should, however, be administered with great caution. Its value no doubt lies in its power to diminish arterial tension.

Belladonna, though highly recommended, does not appear to receive the confidence of the profession as a valuable remedy in this connection.

Thompson has recommended *phosphorus* in large doses for its curative effects. Gowers reports a case that was entirely relieved by a three months' treatment with phosphorus. Others, however, have not succeeded in obtaining the same results. It has the objection of being irritating to the stomach.

Cimicifuga combined with *cannabis indica* has been extolled as valuable in those cases dependent upon or connected with rheumatism.

Ringer, Hare, and others have highly recommended *croton chloral*—butyl chloral—in the treatment of tic douloureux. Hare administers it in five-grain doses every two hours, and finds that its influence is not only palliative but curative. *Antipyrin, phenacetin,* and *salol* have all been recommended as valuable agents, particularly in the rheumatic forms of the affection.

Opium has no curative value, but it is often necessary to administer *morphin* hypodermically to control the severe paroxysms of pain.

Cocain is sometimes administered in the same manner, and for the same purpose.

In cases presenting a syphilitic history, iodid of potassium in twenty-grain doses, increased to thirty or forty grains three times per day, has sometimes proved curative.

Electricity, when judiciously applied, is of great value. The galvanic current is the most beneficial. Authorities differ as to which pole should be applied to the painful spot. Sinkler recommends the negative pole. Gowers thinks the direction of the current is of no great importance. The writer uses the positive pole at the painful spot, and a current of from one to three milliamperes. The current may be applied for from two to five minutes.

As local applications, *menthol* and the *oil of peppermint* often afford temporary relief.

Surgical Treatment.—Various surgical operations have been recommended for the relief of superficial neuralgia. These operations are, subcutaneous division of the trunk of the nerve, resection of the trunk of the nerve, nerve-stretching, evulsion, and the tying of arteries leading to nerve-trunks and nerve-centers.

Subcutaneous division of nerves is productive of immediate relief from the pain for a short time, but eventually, after a few weeks or months, the pain returns as a result of the reunion of the divided nerve.

Excision gives somewhat better results, in that the interval between the operation and the return of the pain is much longer; usually there is complete relief for six or seven months and occasionally for two or three years. As a rule, the more extensive the piece removed, the longer will relief be afforded. It is customary to remove at least half an inch, and as an added safeguard against reunion, to bend back the peripheral end of the segment upon itself.

Nerve-stretching has been followed by complete relief for a time, but so far the reports upon this method do not seem to indicate any better results than are obtained by simple section of the nerve. Andrews reports a case in which stretching of the stump and tearing away of the cicatrix in a case of neuralgia of the inferior maxillary division upon which he had operated one and a half years before by excision with complete relief, but in which the pain had again become severe, was completely successful in abating the trouble.

Evulsion is sometimes productive of better results than excision, as many times a longer section of the nerve can be removed than by cutting it with a knife or neurotome. This method consists of grasping the nerve—after it has been separated from its vessels—as far back as possible, with hemostatic forceps, and forcibly tearing it away.

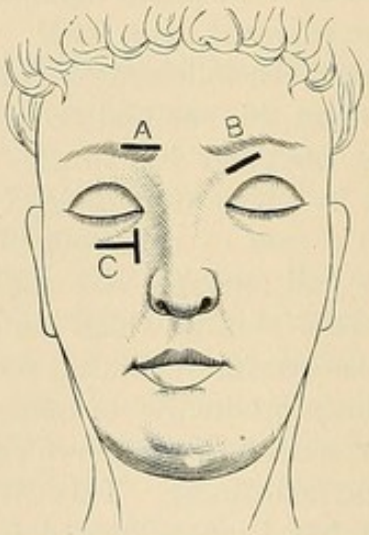
Subcutaneous Division of the Supraorbital Nerve is accomplished by entering a tenotome knife between the eyebrows midway between the nerve and the median line, and passing horizontally beneath the skin until its point is beyond the nerve; its edge is then turned backward and pressed against the bone, and the nerve, lying between it and the bone, is divided by withdrawing the knife. Or, the knife may be entered at the same point, but passed close to the bone instead of just under the skin, its edge turned downward toward the margin of the orbit, and the nerve divided by sweeping the knife downward across the mouth of the supraorbital foramen. (Stimson.)

Excision of the Supraorbital Nerve may be made through incisions above or below the eyebrow. When made above, the incision may be an inch long and parallel to the eyebrow, with its center directly over the supraorbital notch or foramen. (Fig. 138, A.) The incision is carried down to the bone, the distal end of the nerve seized with forceps, dissected out, and excised.

The incision made below the eyebrow requires the eyebrow to be drawn up, and the eyelid down, so as to make the tissues tense. An incision is then made close to the lower edge of the supraorbital arch, an inch long, through the skin, orbicularis muscle, and tarsal ligament. (Fig. 138, B.) The nerve is then traced backward from the notch as far as necessary by depressing the eyeball and the levator palpebræ with a spatula, and dividing the nerve with curved scissors.

Excision of the Superior Maxillary Nerve is made by a curved incision about an inch and a half long following the lower border of the orbit; a second incision at right angles to the first, one inch in length, is next made upon a line drawn from the supraorbital notch to the mental foramen, which will intersect the infraorbital foramen and

FIG. 138.



A, B, Incisions for Excision of the Supraorbital Nerve; C, Incision for Excision of the Infraorbital Nerve, after the method of Tillaux.

expose the nerve. (Fig. 138, C.) A silk thread is now passed beneath the nerve and tied for the purposes of identification and traction. The orbital tissues are then lifted by dissecting up the periosteum from the floor of the orbit, and elevated with a spatula; the infraorbital canal found and broken through, the nerve isolated and lifted from its bed with a curved hook, and divided with curved scissors at a point as far back in the orbit as possible. Traction upon the ligature will draw the nerve from its canal, when it may be severed below the ligature. Hemorrhage is rarely troublesome, though occasionally it may be necessary to use a little packing to control the hemorrhage within the orbit. Fig. 139 shows scar resulting from two different operations for infraorbital neuralgia; A, Lücke's operation; B, Tillaux's operation.

Removal of Meckel's Ganglion.—This operation is sometimes undertaken after the removal of the superior maxillary nerve. This is done by an operation devised by Carnochan, which consists of a T-

shaped incision below the orbit, the horizontal line reaching from canthus to canthus, and the vertical one nearly to the mouth; the tissues are dissected from the facial surface of the bone, and the infraorbital nerve found and secured with a ligature. The outer wall of the antrum is next perforated with trephine or chisel, the infraorbital foramen

FIG. 139.



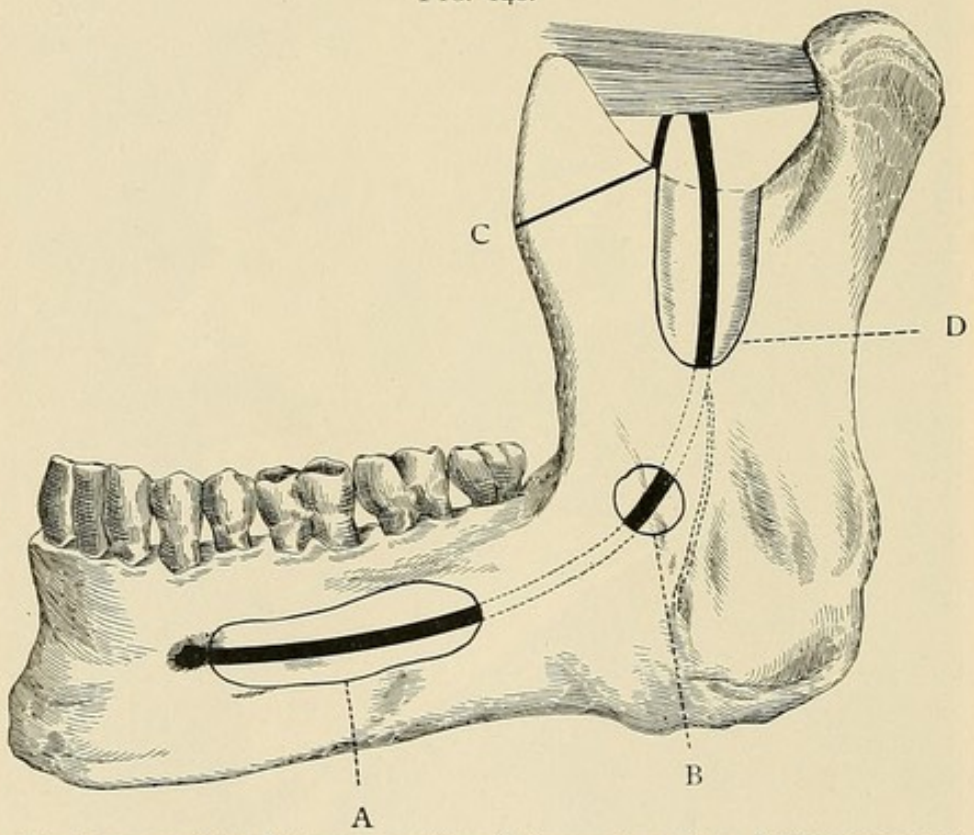
A, Incision for Lücke's operation for Excision of the Infraorbital Nerve; B, Incision for Tillaux operation.

being included. The posterior wall of the antrum is also perforated in the same manner, care being taken not to wound the internal maxillary artery, which lies immediately behind and in close relation to the bone. The groove in the floor of the orbit is next broken through, and after dividing the nerve upon the cheek it is drawn down and through the perforation in the posterior wall of the antrum. Tension upon the

nerve offers a sure guide to the ganglion, by tracing it back into the spheno-maxillary fossa, and to the foramen rotundum, where it may be divided by long, slender, curved scissors. Hemorrhage may be controlled with gauze or sponges fastened to sponge-holders. For the purpose of illuminating the deeper portions of the wound an electric light or a head mirror are absolutely necessary.

Excision of the Inferior Maxillary Nerve.—This nerve may be divided in three locations, at its exit from the mental foramen, in the canal, and before its entrance into the canal.

FIG. 140.



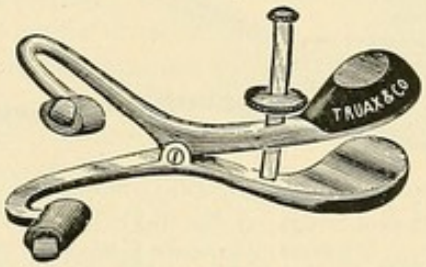
A, Garretson operation; B, Agnew operation; C, Pancoast operation; D, Cryer operation. (After Cryer.)

Excision at the Mental Foramen.—This is accomplished within the mouth by an incision in the gingivo-labial fold above the foramen, which is located just behind the root of the first bicuspid tooth. The soft parts are dissected from the bone with an elevator or periosteotome, until the nerve is reached, which is usually about an inch or an inch and a quarter below the tip of the cuspid tooth. The nerve may then be seized with forceps and drawn as far from the canal as possible, and divided with scissors close to the bone, and upon the peripheral side close to the soft tissues.

Excision within the Canal.—This can be most successfully made by the Garretson operation (Fig. 140, A), which consists of making an in-

cision about two inches long from the angle of the jaw forward. The incision should, for cosmetic reasons, be kept well under the lower border of the jaw. This incision will divide the facial artery, which must be secured. The tissues are now lifted from the outer surface of the bone by a periosteotome, for the entire length of the incision. The external plate of the jaw is next trephined at the opposite ends of the incision, and about a quarter of an inch above the lower border of the jaw; the perforations made by the trephine are next united by two parallel incisions in the bone by a small circular saw, revolved by the surgical engine,—the trephine is driven by the same power,—when with an elevator the section of bone can be lifted from its place, thus exposing the inferior dental nerve and vessels lying in the canal. The nerve is then isolated and lifted from its bed with a blunt hook, and a section removed. Care should be taken not to wound the artery, as hemorrhage is sometimes troublesome. Agnew's operation consists of trephining the jaw at the angle directly over the canal (Fig. 140, B), and removing a section of the nerve.

FIG. 141.



MASON GAG.

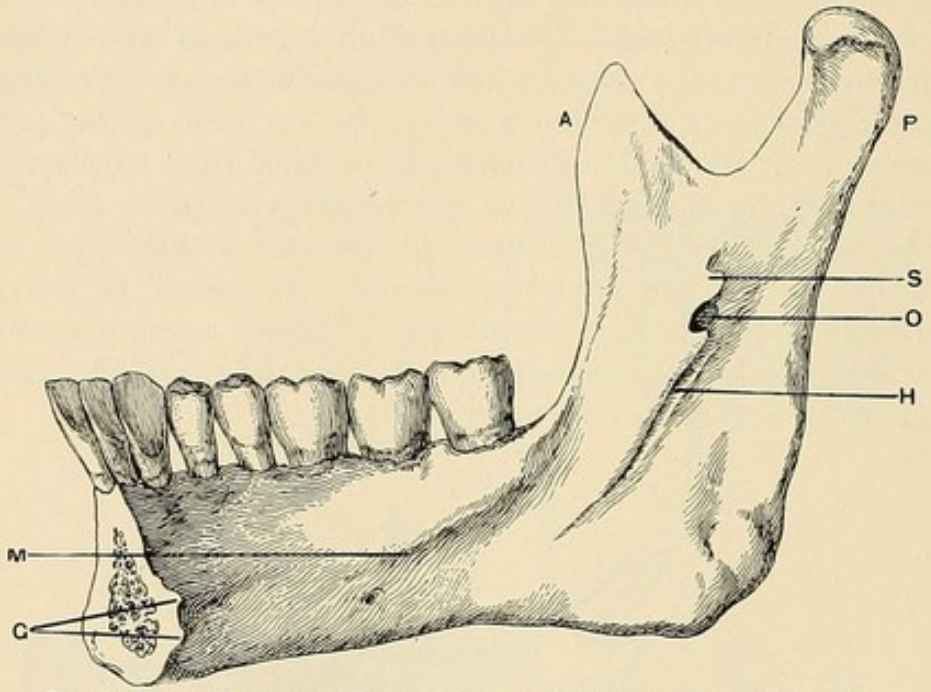
Excision before its entry into the canal may be made either through the mouth or through the cheek.

In operating through the mouth, the jaws must be extended as far as possible with a mouth-gag placed upon the opposite side (Fig. 141). The mucous membrane is first incised at a point on the anterior border of the ascending ramus, midway between the crowns of the upper and lower second or third molar teeth, while the jaws are in this extended position. The finger is next inserted between the internal pterygoid muscle and the ramus. The tubercle situated at the opening of the foramen is now felt (Fig. 142, S), and the nerve brought to the surface by means of a blunt hook, grasped with hemostatic forceps, and a section removed. Hemorrhage is sometimes profuse. Care must be exercised not to mistake the long internal lateral ligament for the nerve.

Pancoast performed excision of the nerve by first removing the coronoid process by an incision through the cheek (Fig. 140, C). There is a serious objection to this operation, for the reason that it destroys the use of the temporal muscle.

In operating through the cheek by an external incision, after the method of Cryer, an incision is made over the center of the ramus, beginning at the zygomatic arch and extending downward an inch and a half; the semilunar notch is now exposed and deepened with surgical burs to the depth of about one inch, when the opening thus made

FIG. 142.



THE INFERIOR MAXILLARY BONE—INTERNAL SURFACE OF THE RIGHT SIDE.

G, Genial tubercles; M, Mylo-hyoid ridge; O, Opening of the inferior dental canal; H, Mylo-hyoid groove; S, Pterygoid tubercle; A, Anterior or coronoid process; P, Posterior or condyloid process.

through the bone exposes the nerve. (Fig. 140, D.) It is now picked up and a section removed. The wounds are to be sutured, and if treated with antiseptic precautions, will commonly unite by first intention.

REVIEW.

CHAPTER XXXIX.

The systemic treatment of neuralgia depends upon what conditions?

Does the affection respond to drugs?

What general conditions should be sought for?

On what affection may it be assumed that the majority of cases of facial neuralgia depend?

What should be the treatment of those cases dependent upon the impairment of the general health?

What remedies are usually prescribed in such cases?

What is the value of gelsemium in trifacial neuralgia?

In what form of the drug and in what doses may it be administered?

What is the value of valerianate of zinc?

How is cannabis indica administered to obtain the best results?

How should aconite be administered, and how does it produce its effect?

Is belladonna useful in the treatment of this affection?

What are the opinions in regard to the use of phosphorus in this affection?

In what form of neuralgia is cimicifuga combined with cannabis indica recommended?

What are the opinions as to the value of croton chloral in the treatment of neuralgia?

In what doses may it be administered?

In what forms of neuralgia are antipyrin, phenacetin, and salol recommended?

For what purpose are opium and morphia used in neuralgia?

How may cocain be beneficially used?

In what cases is the iodid of potassium recommended?

What is the value of electricity, and which current gives the best results?

What strength of current is most beneficial?

What local applications may sometimes afford relief?

What operations have been recommended for the surgical treatment of trifacial neuralgia?

What is the value of subcutaneous section of the nerve?

About how long is relief obtained by this operation?

Does excision give better results?

How long may relief be obtained by this operation?

What have been the results of nerve-stretching?

What is the value of evulsion?

How is evulsion practiced?

Describe the operation for the subcutaneous division of the supraorbital nerve.

Describe the operation for the excision of the supraorbital nerve.

Describe the operation for the excision of the superior maxillary nerve.

Under what circumstances is the removal of Meckel's ganglion sometimes undertaken?

Describe the operation for the removal of this ganglion.

In what particular locations may the inferior maxillary nerve be divided or excised?

Describe excision of the inferior dental nerve at the mental foramen.

Describe the steps in Garretson's operation for excision of the inferior dental nerve within the canal.

What are the various steps in the operation for excision of the inferior maxillary nerve at the inferior dental foramen through the mouth?

Describe the Pancoast operation and its objections.

Describe the various steps of Cryer's operation for excision of the inferior maxillary nerve at the inferior dental foramen through the cheek.

CHAPTER XL.

CONGENITAL FISSURES OF THE LIP AND THE VAULT OF THE MOUTH.

FISSURES of the upper lip, superior maxillary bones, and soft palate are the result of arrested development of the parts involved, and consequent failure of these parts to form a junction and coalesce. The fissure may be of any degree from a slight notch in the lip or a bifurcation of the uvula, to a complete cleft of the lip, alveolar process, palate bones, and velum palati; or a double cleft of the lip and bony palate and almost entire absence of the velum.

The slightest degree of fissure is represented by a superficial notch or scar in the upper lip, and by a mere suggestion of a bifurcation of the uvula. The most common forms are fissures of the lip and the velum. Fissures of the lip often occur without cleft of the velum or maxillary bones, while on the other hand cleft of the velum frequently occurs without fissure of the lip; the cleft in the palate may even extend forward to the alveolar process, and still not be associated with a fissure of the lip; but where the fissure extends through the alveolar process the writer has always found it associated with a fissure of the lip. "In some rare instances, however, the alveolar process alone may be fissured."

Fissures of the lip and palate may be *unilateral* or *bilateral;* but are most frequently unilateral, and most commonly upon the left side.

Figs. 143 and 144 are photographs of cases which have come under the care of the writer, and are inserted for the purpose of illustrating some of the extremes in unilateral fissures of the lip and the vault of the mouth.

In the child (male) represented by Fig. 143, there was complete cleft of the hard palate, velum palati, and *right* side of the lip, with marked protrusion and eversion of the intermaxillary bone; in all other respects the child was perfectly formed. No history of hereditary tendency or of maternal impressions could be deduced.

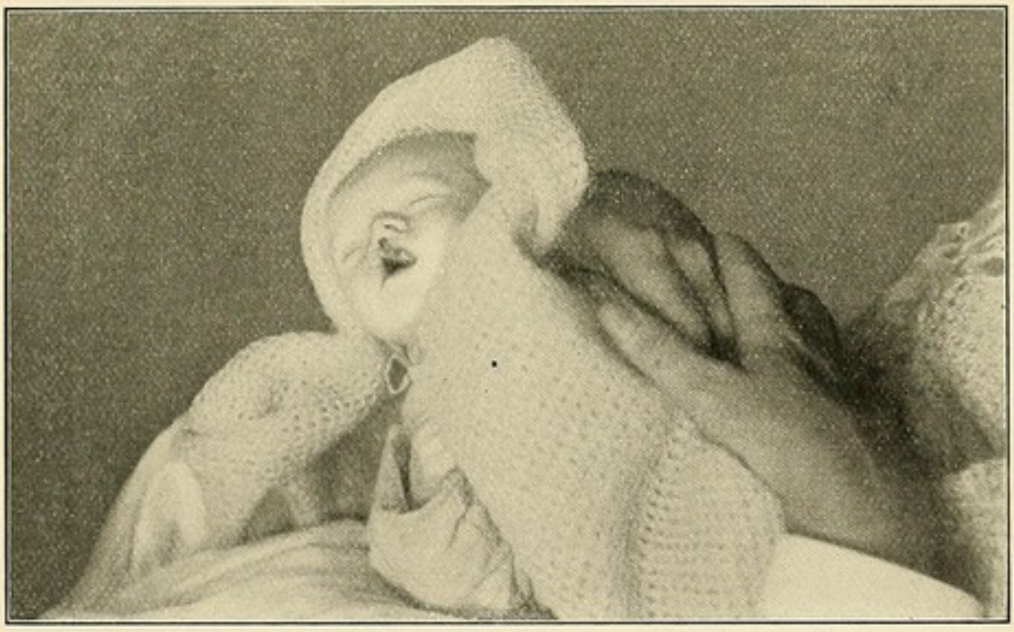
Fig. 144, also a male child, has complete cleft of the hard palate, velum palati, and *left* side of the lip, with marked protrusion of the intermaxillary bone. The deformity of the face in this case is much greater than in the preceding one, while in other respects the child was

defective in development, having an immense congenital scrotal hernia, —larger than a goose-egg,—and the fourth and fifth toes of the left foot united.

Bilateral fissure of the lip and maxillary bones with protrusion of the intermaxillary bones and median cleft of the soft palate occasionally occurs. (Fig. 145.) The writer has operated upon several cases of this character associated with extensive protrusion of the intermaxillary bones. In each of these cases there was no union of the palate process with the vomer on either side.

In exceptional cases the fissures may extend upward on either side of the nose, or backward, involving the base of the skull. Median fissure is very rare. Salter mentions three cases, one described by

FIG. 143.



COMPLETE CLEFT OF THE HARD PALATE, VELUM PALATI, AND RIGHT SIDE OF THE LIP, WITH MARKED PROTRUSION OF THE INTERMAXILLARY PORTION OF THE JAW. CHILD 9 WEEKS OLD.

Rokitansky, one met in his own practice, and a specimen in the Museum of the Royal College of Surgeons, London. Occasionally there is an entire absence of the maxillary bones, and consequently of the incisor teeth.

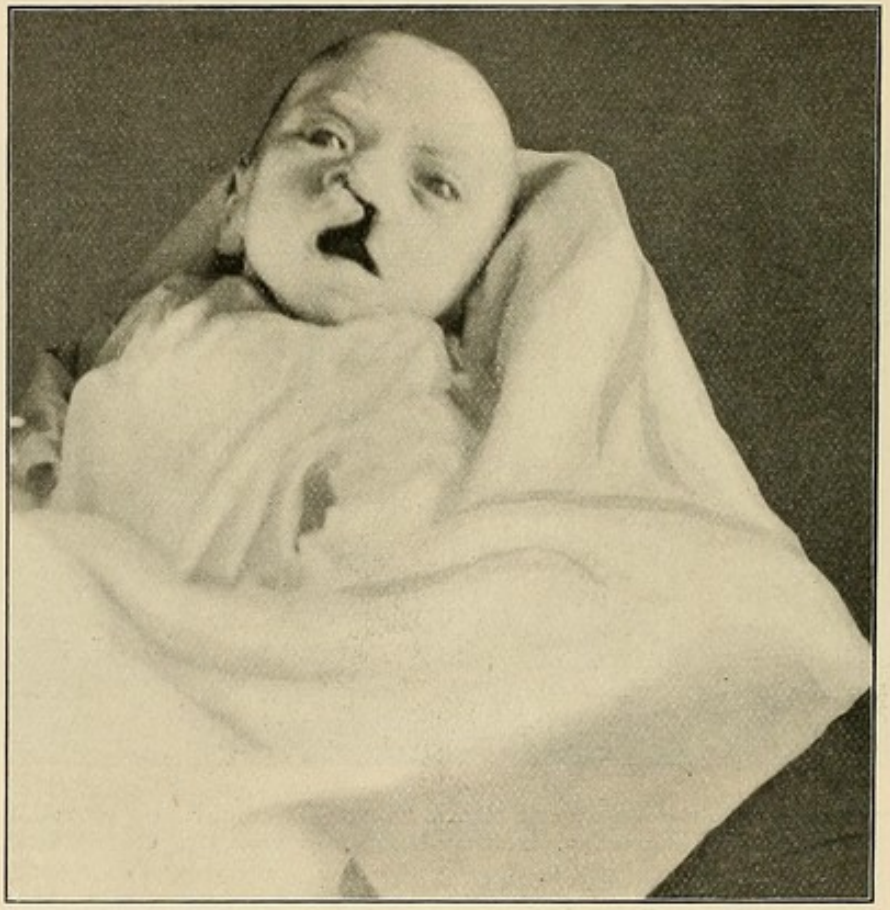
A case of this character recently came under the observation of the writer in a female child six weeks old, in which there was a complete cleft of the hard and soft palates and fissure of the lip on the left side, with entire absence of the intermaxillary bone and the vomer. The cleft through the alveolar process measured five-eighths of an inch in width. The tip of the nose was greatly depressed, being but very slightly elevated above the level of the cheeks. The cartilaginous

nasal septum was also absent. The whole condition caused one of the most ugly deformities imaginable. There was no history of heredity or of maternal impression.

In Salter's case of median fissure, the right intermaxillary bone was slightly deficient, with absence of the central incisor; the lateral, however, was in position standing close to the cuspid and separated from the left central by a deep fissure.

Broca has reported quite recently a case of complete fissure of the upper lip with absence of the median tubercle.

FIG. 144.



COMPLETE CLEFT OF HARD PALATE, VELUM PALATI, AND LEFT SIDE OF THE LIP, WITH MARKED PROTRUSION OF THE INTERMAXILLARY PORTION OF THE JAW. CHILD 8 MONTHS OLD.

Median fissure of the face is more often associated with the lower lip and inferior maxilla than with the upper portion of the face. A remarkable case of this character (Fig. 146) is reported by A. Wölfler, as occurring in an infant that came under his notice when it was twenty-three days old, in which the lower lip was cleft, the inferior maxilla separated upon the median line, but held together by a cicatricial band; the fissure extending downward into the neck to the supra-sternal fossa; the anterior portion of the tongue was likewise

divided into two halves upon the median line. These deformities were successfully corrected by surgical operations.

Sometimes there are other defects of development associated in the individual with fissure of the palate, due to the same general causes. These defects are occasionally of an extreme character and serious nature, and interfere with the performance of the natural functions of the body.

FIG. 145.



Double Hare-Lip, with protrusion of the intermaxillary bone, and non-union of the maxillary bone with the vomer on both sides, making what might be termed a double cleft in the hard palate. The cleft in the soft palate was exceedingly wide, showing very imperfect development.

Origin.—The origin and causes of hare-lip and cleft palate are to be sought for among the pre-natal influences, and are generally conceded to be faults in the developmental process. These influences to be operative must occur prior to the tenth week after conception. The formation of the maxilla begins at a very early period of intra-uterine life, viz: at about the twenty-eighth day by the development of four tiny buds, tubercles, or processes near the central portion of that sur-

face of the rudimentary head which is destined to form the face (Fig. 147), which are denominated the superior or frontal processes or tubercles, and the lateral or oblique maxillary processes or tubercles. The superior processes elongate downward, and at the same time approach each other toward the median line, where they finally coalesce to form the intermaxillary bones and the central portion of the upper lip. The latter processes likewise elongate and approach each other toward the median line, where they finally meet the superior processes and unite with them, thus forming the lateral halves of the superior maxillary bone, palate bones, the cheeks, and lateral portions of the upper lip and the velum palati.

FIG. 146.



MEDIAN FISSURE OF THE LOWER LIP AND CHIN. (After Wölfler.)

Non-Union of Superior and Lateral Processes.—The frontal processes rarely fail to unite upon the median line; but it is not uncommon for the oblique maxillary processes of either side to fail to unite with the frontal processes, and occasionally both sides fail; thus, in the former case producing a fissure upon the right or left side, and in the latter a double fissure.

Fissures of the lip are the result of the same causes, viz: failure of the central portion of the lip to unite with the lateral portions, while

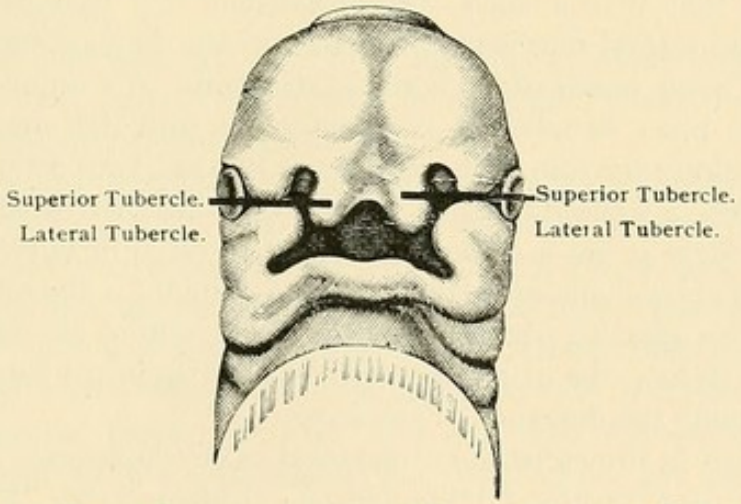
clefts in the velum palati are the result of a non-union of the lateral half with its fellow upon the median line.

Arrested Development.—In many cases there is a deficiency in the various tissues which go to make up the complete superior maxillary bone, palate bones, lip, and soft palate; hence the primary cause would seem to be an arrestment of the developmental process in these particular parts. The writer has seen several cases in which the velum palati especially was very deficient and only rudimentary in character, while in others there was a marked deficiency in all of the tissues of these parts, leaving a broad, yawning aperture.

The extent of the fissure will depend very largely, if not entirely, upon the time at which the arrestation of development occurred; the earlier the period the more extensive the cleft, and *vice versa*.

At about the fortieth day after conception the superior and lateral processes have united, and by the end of the tenth week the vault of the mouth has been completed by the union of the velum palati and uvula

FIG. 147.



HEAD OF AN EARLY HUMAN EMBRYO, SHOWING THE DISPOSITION OF THE FACIAL FISSURES AND OF THE SUPERIOR AND LATERAL TUBERCLES. (After His.)

through their entire length; this process begins at the anterior aspect, and progresses backward, the uvula being the last portion to unite.

In certain cases of hare-lip and cleft palate there is a sufficiency of tissues, the only fault seemingly being a failure of union of the parts at the proper time. These cases when treated surgically in the early months of infancy, generally secure normal position of the parts and a complete restoration of function.

The fundamental influences, however, which underlie the causation of these defects in development have not yet been reached, and all theories which have so far been advanced to account for them belong to the realm of speculation and conjecture.

Faulty Nutrition.—Some have thought the trouble to be caused by a fault in the diet, through the exclusion of meat as an article of

food, or the introduction of an insufficient quantity of calcium phosphates into the system of the mother during gestation.

As an argument in favor of this view it might be stated that the lions in the Zoological Gardens of London were fed for several years upon meat from large animals having bones too large for them to crush and swallow; this was followed by the birth of cubs with cleft palate,—99 per cent.,—which lived but a short time on account of their inability to suckle. The lions were then occasionally given a small animal, like a goat or sheep, the bones of which were readily crushed by their teeth, and the young afterward born had perfectly formed palates.

At the Zoological Garden of Dublin a like experience was encountered, and was counteracted by feeding the pregnant lions with ground bones and foods containing calcium phosphates. Dr. J. Ewing Mears reports the same condition prevailing among the offspring of the lions at the Philadelphia Zoological Garden.

Neither of these arguments is entitled to very much weight, for the reason that it is a fairly well-established fact that union of the superior and lateral maxillary processes in the human subject is not dependent upon ossification of these structures, for union or coalescence takes place in advance of ossification, and this process is not completed along the line of the sutures until some time after birth.

It is also a well-established physiologic law that in the pregnant woman, if there is not a sufficient amount of calcium salts ingested to support the extra demands made upon the system for the proper development of the osseous framework of the fetus, and to recoup the waste in her own tissues, the material already stored up in her body is drawn upon to supply the demands of the fetus.

Changes in the constituent elements of the bones are of common occurrence as a result of malnutrition. Dalton says, "Next to the chlorid of sodium, the phosphate of calcium is considered the most important ingredient of the body. It is met with universally in every tissue and every fluid," and "whenever the nutrition of the bone during life is interfered with from any pathologic cause, so that its phosphate of calcium becomes deficient in amount, a softening of the osseous tissue is the consequence, by which the bone yields to external pressure and becomes more or less distorted."

In fractures occurring during gestation, union is often delayed, sometimes until after delivery. Padieu describes a case in which fractures of the tibia and fibula occurred nine days after the suppression of the menses, and in which union was delayed until the end of gestation. The process of union began ten days after delivery, and was completed at the end of a month.

The pelvis, though looked upon at all other times as a compara-

tively solid framework, frequently becomes relaxed in its articulations during pregnancy, so that the sacro-iliac and pubic joints become movable.

Many women are in the habit of discarding from their aliment during pregnancy all those foods which contain an abundance of calcium salts, and restrict themselves, as nearly as possible, to a fruit diet, believing that by such practice the bones of the child will be imperfectly calcified, and thus parturition be robbed of much of its suffering. There is, however, no scientific evidence that such a result is obtained, while on the other hand, as in those cases affected with hyperemesis, though the child when born may be small and much emaciated, it has the appearance of being properly formed, and its bones as dense as in the majority of normal pregnancies.

Heredity.—The question of hereditary influence is one that calls for more than a passing notice, for *a priori* it would strike one as likely to be an important factor in the production of defects of development. The facts, however, which have been deduced, are not of sufficient strength to establish it.

A few isolated instances have been reported in which there seemed to be an indirect inherited tendency in a certain family to produce offspring with hare-lip and cleft palate.

Oakley Coles mentions two families in which there was a marked tendency in this direction. In the first family there were three with cleft palate, one seventeen years of age, another thirty, and the third thirty-five; the first and last were women. The man, who is married, has a family without a single instance of the father's deformity. The second family was composed of five children, two of whom had fissure of the lip and palate; the first child was born perfect, the second had single hare-lip and cleft palate, the third child was perfect, the fourth had double hare-lip and cleft palate, and the last child was perfect. The maternal grandmother also had cleft palate.

Lawson Tait believes that heredity is a strong factor in the production of this deformity, and says he has known it to skip three generations and then appear in a hereditary form.

E. F. Plicque reports a case of hare-lip in a female, in which he thinks the deformity is undoubtedly inherited. The family history is as follows: Both parents of the patient were entirely free from any congenital defect. One of her father's brothers had supernumerary fingers. A brother of her mother was born with hare-lip, but both of his children were free from the deformity. Her sister, the mother of the patient, has given birth to nine children, five of whom had hare-lip but no palatal defect. Another sister of the patient's mother, who was free from congenital defect, gave birth to two children with hare-lip and cleft palate of an uncommonly severe type. A brother and a

sister of the patient, both of whom had congenital hare-lip, had married, but neither of their five children had any sign of the defect.

Manley says in all of his cases there was either a history of heredity or of maternal impression.

The writer recently operated upon a child six months old, with double fissure of the upper lip and hard palate, with protrusion of the intermaxillary bones and only rudimentary velum, this being the second child born of the same parents, in whom the tendency was marked. The first child was born with fissure of the left side of the upper lip; the second child was born perfect, and the third child with the defect first described. There was no history of similar defects of development in the family of either of the parents.

Maternal Impressions.—It is interesting to note in this connection that most women who are so unfortunate as to give birth to deformed children, especially those with deformities of the face and mouth, feel very confident that it is the result of maternal impressions induced by fright, the sight or knowledge of a like deformity, etc. How much, if any, there may be of scientific truth in this popular notion the writer is not prepared to say, more than that in all popular notions there is generally somewhere hidden away a kernel of truth. When we know more about the influence which the nervous system exerts over cell-life, the effects of the physical and mental conditions of the parents at the time of conception, and of the female parent during gestation, we shall be better able to consider the question from a scientific standpoint; till then it would be mere speculation.

Prognosis.—Most of the fissures and perforations of the hard palate are susceptible of radical cure by plastic surgical operations; the exceptions being those cases where there is marked deficiency in development, or loss of tissue, and even in these there is reason to hope that the operation of transplanting new tissue from some adjacent locality will be so perfected as to become not only feasible as an operation, but successful in re-establishing the functions of the parts. In clefts of the velum palati where the fissure is very wide and the deficiency in tissue is considerable, it is better to depend upon the artificial velum, rather than to attempt a cure by surgical measures; for unless the velum can be restored to its normal length so as to perfectly close the naso-pharyngeal opening, the operation would be a failure, from the practical standpoint, for restoration of function is the main object in view.

The enthusiasm of the surgeon has many times carried him beyond the limits of a wise conservatism in the treatment of these cases, especially in operations upon the velum, with the natural result, failure. Consequently there are those who decry all attempts at cure by a surgical procedure, and maintain that in all cases mechanical appliances

accomplish the best results in restoring the functions of the parts. A wise conservatism in both directions will, in the writer's opinion, give the best results in the individual case, for in no department of surgery is good judgment of more value to the patient.

The mortality of operations for fissure of the lip and palate, according to the investigations of Hoffa, is greatly influenced by the deformity itself. From the records of 114 cases of hare-lip, twenty-seven deaths occurred, giving a mortality of 23.7 per cent.; while out of 111 cases of complete fissure of the lip and palate operated upon, there were forty-three deaths, making the mortality 38.73 per cent.

Bolling maintains that although the mortality rate is high in operations for the correction of these deformities, it does not very much exceed that of children of the same age.

Although the writer has not statistics at hand to substantiate his opinion upon this question, he yet feels sure that this rate of mortality is considerably higher than for such operations among American surgeons.

REVIEW.

CHAPTER XL.

What do hare-lip and cleft palate result from?
What are the slightest degrees of fissure of the lip and palate?
What are the more severe degrees of fissure?
Does cleft of the lip occur without fissure of the vault of the mouth?
May fissure of the vault occur without cleft of the lip?
If the fissure extends through the alveolar process, is there cleft of the lip?
Which form of fissure is most common, unilateral or bilateral?
Which side is most commonly fissured?
Are the intermaxillary bones sometimes undeveloped?
Do the lateral fissures ever extend beyond the lip and vault of the mouth?
Do median fissures ever occur?
With what part of the face are they most frequently associated?
What are the causes of fissures of the lip and vault of the mouth?
At what period of intra-uterine life do the jaws begin to develop?
What are the first evidences of this development?
How do these tubercles unite to form the upper jaw, soft palate, and lip?
How are simple fissures produced?
How are double fissures produced?
What is the cause of failure of union?
At what period of intra-uterine life is the process of union completed in the vault of the mouth?
At what part of the vault does this process begin, and which is the last part to unite?
May faults in nutrition be a factor in causing non-union of the parts?

May faults in diet be a cause?

What physiologic law is opposed to this?

Is heredity an important factor in the causation?

May maternal impressions have an influence in causing the deformity?

In what class of cases is the prognosis favorable for cure by surgical operation?

What are the main objects to be attained in radical cure by a surgical operation?

What condition must be obtained in the velum palati to restore the functions of deglutition and speech?

Are all cases of cleft palate susceptible of cure by a radical operation?

What should govern the surgeon in these questions?

What class of cases are not amenable to surgical cure?

What is the best treatment in these cases?

What is the stated mortality of operations for fissure of the lip?

What for operations of fissure of the palate?

CHAPTER XLI.

CONGENITAL FISSURES OF THE LIP AND THE VAULT OF THE MOUTH (Continued).

Surgical Treatment.

The operations which are practiced for closing the fissures of the palate are designated as *uranorrhaphy* and *staphylorrhaphy*. Uranoplasty or uranorrhaphy is the operation for closing a fissure in the hard or bony palate, while staphyloplasty or staphylorrhaphy is the term applied to the operation for closing a cleft in the soft palate or velum palati. Chiloplasty or chilorrhaphy is the operation for closing a fissure in the lip.

Lemonnier, a French dentist, is credited with having been the first to suggest and to successfully operate for the closure of fissures of the palate by surgical operation, the record having been published in 1766. Lemonnier succeeded in closing a fissure in both the hard and soft palates, by paring the edges of the cleft with a knife, and approximating them by the use of sutures. Perforations in the hard palate he successfully closed by exciting granulation of their borders.

Eustache, a physician of Beziers, in 1799 recommended the same procedure to a patient for whom the day before he had split the soft palate for the purpose of removing a polypus of the pharynx. The operation, however, was declined by the patient. In 1800 he presented a paper upon the subject of closing congenital fissures of the soft palate to the Académie Royale de Chirurgie at Paris, asking their approval of the operation, but this they declined to grant.

Von Graefe revived the operation in 1816, and reported to the Medico-Chirurgical Society of Berlin that after many unsuccessful efforts to close fissures of the soft palate he had at last obtained success by freshening the edges by the application of muriatic acid and the tincture of cantharides, and then approximating them with sutures.

The operation was modified by Roux, in 1819, who closed a fissure of the palate by paring the edges and applying sutures.

Warren, of Boston, in 1820, being ignorant of the efforts of the other surgeons, performed successfully a similar operation. After this time the operation became generally known and practiced.

To Sir William Fergusson, of England, however, belongs the credit, more than to any other surgeon, of first demonstrating and giving to the world a scientific basis for the requirements of the operation of staphylorrhaphy.

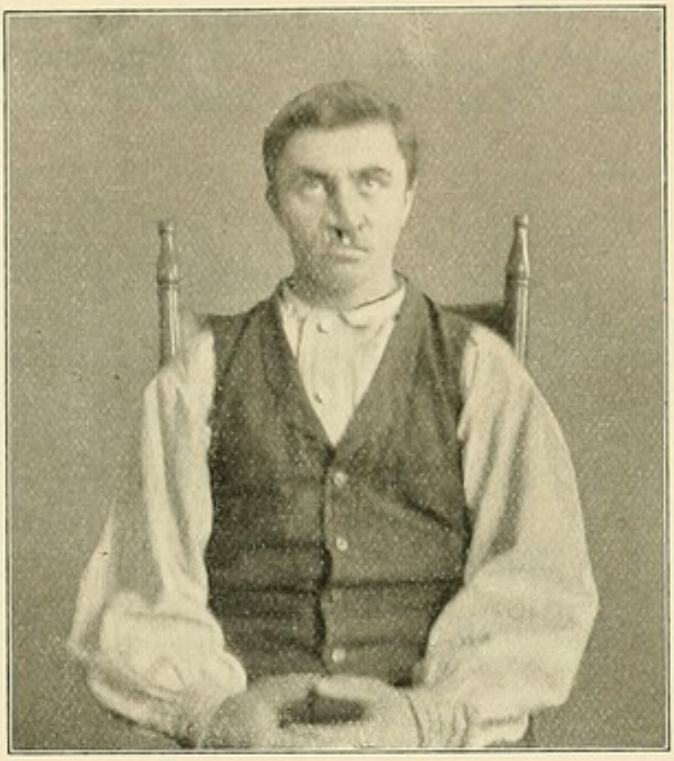
The first important question in relation to the surgical treatment of cleft palate is that of the age of the child which gives the best prospect of a successful issue of the operation, and the restoration of the parts to normal function.

Experience has taught the writer that skillful operations for closure of fissures of the palate when performed during the early months of infancy are more successful in restoring the functions of deglutition and articulation than when postponed, as is generally advised, until after the eruption of the deciduous teeth, or even to as late a period as the fifteenth year. In order to obtain the best results, the operation should be completed before the child begins the first attempt at articulate speech. When delayed until after speech has been acquired, it is much more difficult to overcome the peculiar nasal tone that always accompanies the voice in persons with perforations, or clefts, of the bony palate or velum. Another argument for early operation is the facility and comparative safety with which infants can be brought under the control of anesthetics, and the ease with which anesthesia can be maintained, and this is a great *desideratum* in all operations upon the mouth. Chloroform has the preference, with the writer, for operations upon little children. In complete clefts of the upper lip and maxilla there is a noticeable broadening of the face upon the affected side; the distance from the median line of the apex of the nose to the antero-inferior angle of the malar bone is greater than upon the perfect side, and there is also accompanying this a decided spreading out and flattening of the ala of the nose. (Fig. 148.) When the lip has not been closed this broadening of the face and flattening of the ala of the nose increase with the growth of the individual. On the other hand, in those cases where the lip has been closed early, this widening is not only prevented, but there seems to be a slight narrowing of the cleft, due no doubt to the muscular contraction of the united lip. An early operation should therefore be recommended for the closure of the lip and, where the strength of the child will permit, of the fissure in the bony palate and velum as well, provided the condition of the velum gives promise of successful restoration of function. A second operation for the closure of the velum can be done a few months later if the condition of the child is not favorable for such procedure at the time of closing the fissure in the hard palate.

Operations.—In operations about the mouth the choice of anesthetics must be governed by the age of the patient and the general conditions of health. Chloroform is the pleasantest for all mouth-surgery,

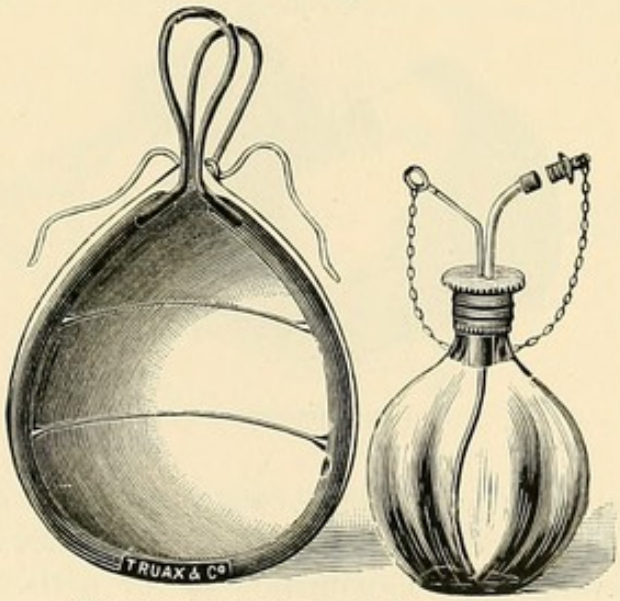
especially in operations upon the velum, on account of the fact that it is not so liable to cause vomiting or irritation of the bronchial

FIG. 148.



HARE-LIP AND CLEFT PALATE.

FIG. 149.



CHLOROFORM INHALER AND DROP-BOTTLE.

mucous membrane as is ether, though it has the disadvantage of being much more dangerous to life. Little children, however, as a rule, bear chloroform much better than adults, and it may therefore be adminis-

tered with comparative safety, and also much better after-effects. A most convenient method of administering chloroform is by use of the inhaler and drop-bottle (Fig. 149), though it may be administered upon a handkerchief or a napkin. When administering ether, the inhaler, Figs. 150 and 151, will be found most convenient and useful.

The position of the patient in operations upon the vault of the mouth is one that needs careful consideration, on account of hemor-

FIG. 150.



ETHER INHALER.

FIG. 151.



ETHER INHALER.

rhage, which is often quite profuse when operating upon the hard palate by the Langenbeck method, and the difficulties sometimes experienced in getting good illumination of the parts. The position shown in Fig. 152 is the best under nearly all circumstances, as it permits the blood to escape by the nostrils instead of into the throat, and at the same time gives a good view of the parts if the operator stands at the head of the patient.

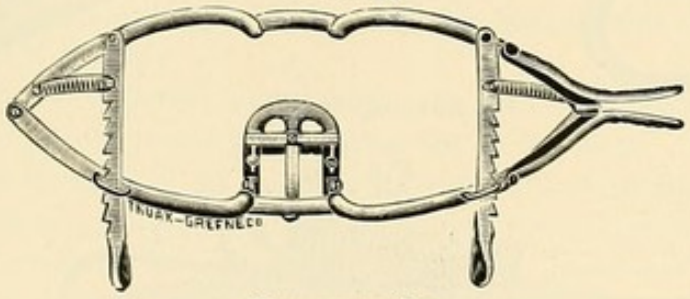
The instruments needed in performing a staphylorrhaphy are: a mouth-gag, a Mason's or a Whitehead's (Fig. 153), a sharp-pointed curved bistoury (Fig. 154), a pair of mouse-tooth tissue-forceps (Fig. 155), a pair of curved needles—right and left—with handles (Fig. 156), a suture pick-up (Fig. 157) and a wire-twister (Fig. 158), a pair of small, long-handled scissors, silk and silver wire sutures, perforated shot, and a shot compressor.

FIG. 152.



POSITION OF HEAD DURING OPERATIONS ON THE MOUTH. (After Murphy.)

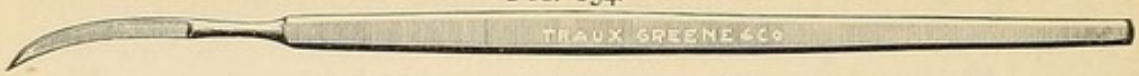
FIG. 153.



WHITEHEAD GAG.

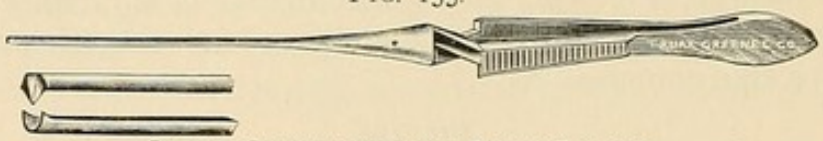
In operating upon a simple hare-lip, a bistoury or the hare-lip scissors may be used for paring the edges of the cleft. (Fig. 159.) In a flap operation upon the lip the bistoury or a small scalpel is the best for this purpose. A pair of lip-compressors (Fig. 160) will also be found serviceable for controlling the hemorrhage. The lip may be united either with the hare-lip pins and the figure of 8 suture, or with the interrupted suture of silk or catgut.

FIG. 154.



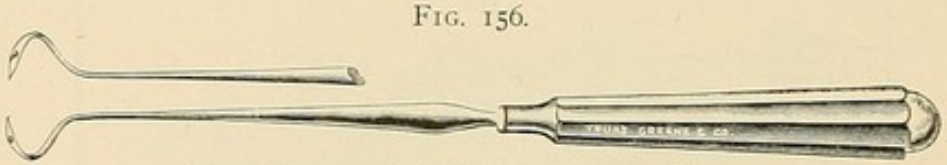
CURVE-POINTED STAPHYLORRHAPHY BISTOURY.

FIG. 155.



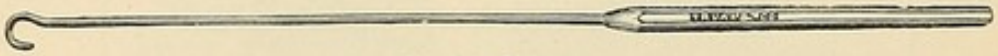
SPECIAL STAPHYLORRHAPHY SCREW FORCEPS.

FIG. 156.



AUTHOR'S JACKSON-EYE STAPHYLORRHAPHY NEEDLES.

FIG. 157.



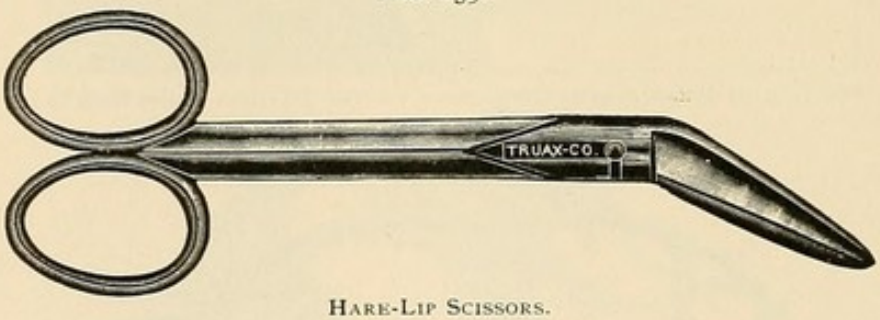
SUTURE PICK-UP.

FIG. 158.



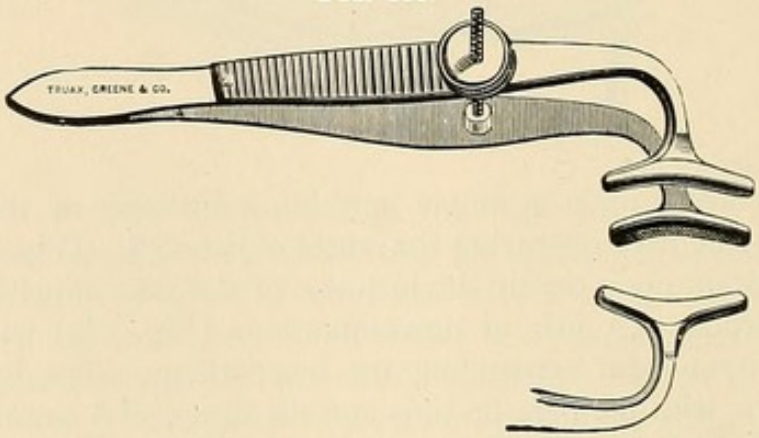
SPECIAL WIRE-TWISTER.

FIG. 159.



HARE-LIP SCISSORS.

FIG. 160.



LIP COMPRESSOR.

Hare-Lip.—In uniting fissures of the lip it is important to conserve as much of the tissue as possible, in order that the lip may not be unnecessarily contracted. Various methods have been devised to give a normal shape to the free border of the lip and to prevent the notched condition which so often follows hare-lip operations.

In all operations for closing fissures of the lip it is important to dissect the lip from the alveolar process upon either side of the cleft for a considerable distance backward, in order to gain as much tissue as possible and to prevent strain upon the freshly-united edges of the cleft.

The common method of uniting a cleft in the lip is simply to pare the edges with a curved bistoury, inserting it at the angle of the cleft upon one side and then upon the other, and carrying it through the lip to the vermilion border, removing a paring the full thickness of the lip, or obtaining the same result by trimming the edges with the hare-lip scissors. The edges are then brought together with sutures of silk, catgut, or hare-lip pins. The lip-compressor may be necessary to control the hemorrhage from the coronary arteries until the sutures are ready to be placed. Tying of these vessels is rarely if ever necessary.

Fenger, of Chicago, has lately devised a new operation for hare-lip, which consists essentially of utilizing the parings of the fissure to lengthen the border of the lip, splitting the edges of the flaps and uniting the edges of the mucous membrane and the skin by separate lines of sutures, this part being similar to the operation of Tait for uniting the lacerated perineum, and also that of Marcy for closing the velum palati.

Fillebrown, of Boston, recommends the following operation, which is similar to that devised by Nélaton, for relieving the notched condition of the lip so frequently seen in the border after operation for single fissure of the lip:

"A male, aged thirty, a patient in the Harvard Dental Hospital, came to have an obturator constructed for cleft palate. The cleft involved the hard as well as the soft palate, and originally a hare-lip. The lip had been operated on, and of course much improved, but the characteristic notch was present. (Fig. 161.)

"It had long been my belief that this deformity could be remedied, and I hailed with pleasure the opportunity to apply the remedy.

"The operation performed for it is shown in the illustration here presented. It was the result of study, experiment, advice, and accident, and its success entirely fulfilled my expectations and hopes.

"By comparing the two cuts and noting the position of the letters, the operation will be fully understood.

"The line a-b, Fig. 162, represents the cicatrix left by the former

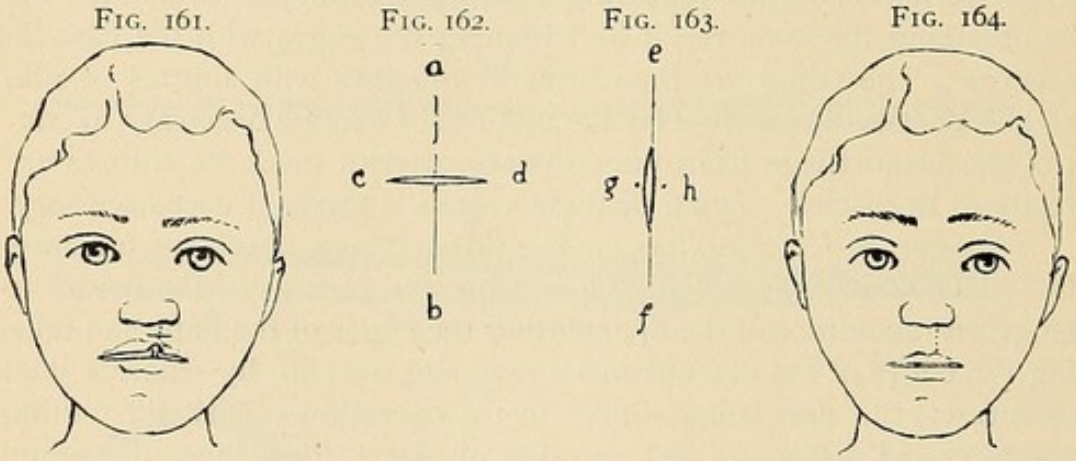
operation. The line c-d shows the line of the incision transversely across the lip.

"Fig. 163 shows the incision made vertical by drawing the lip down and inserting a suture and drawing the points g-h, representing c-d of Fig. 162, close together. This converted the horizontal incision into a vertical one, and lengthened the lip at that point by just the length of the cut.

"Fig. 161 shows the case as photographed just previous to the operation. The notch was so considerable as to show the patient's lateral incisor tooth continually.

"Fig. 164 shows the case as photographed after the lip had healed.

"The approximation of the lips was perfect, and but very little narrowing of the red border was perceptible.



Fillebrown's Operation.

"The excessive size of the nostril was reduced by a V-shaped incision, taking out a piece of the wall of the nostril and drawing the edges together. This was entirely independent of the lengthening of the lip.

"The operation proved an entire success."

In cases of single cleft of the lip and palate the writer advises the closing of the lip as soon after birth as the condition of the child will permit, and the operation upon the bony palate and velum from the sixth to the twelfth month.

In operating for single hare-lip, preference is given to the Mirault method as most likely to produce a lip of normal length and width. This consists of bringing down a flap from one side, sliding it across the cleft, and attaching it to the pared opposite side. Fig. 165 is the result of a Mirault operation upon the child, Fig. 143, photographed ten days after the operation. The intermaxillary bone was brought into position by fracturing the bone upon the left side and uniting the edges of the cleft in the maxilla by a wire suture. Owen's operation, Figs. 166 and 167, is quite similar, although the incision for making the

flap is carried into the lip somewhat deeper. This incision gives the fullness to the lip where most needed. In closing the hard palate in these cases the writer prefers the Langenbeck operation—muco-periosteal flap—from the fact that in a majority of instances it succeeds in filling the gap with osseous tissue.

In cases of double cleft of the lip and palate with protrusion of the intermaxillary tubercle, operation should be advised at the earliest

FIG. 165.



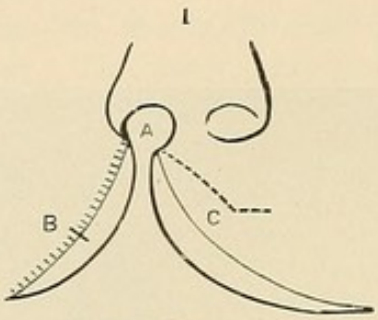
RESULT OF OPERATION FOR DEPRESSION OF THE INTERMAXILLARY PORTION OF THE JAW AND CLOSURE OF THE HARE-LIP. TEN DAYS AFTER OPERATION.

possible day, as these children are prevented from taking the breast or even the bottle; feeding by the spoon is therefore the only method that can be used, and on account of the difficulty in swallowing, they as a rule do not obtain sufficient nourishment to properly sustain the functions of life, and as a result many dwindle away and die. Early correction of the deformity in the anterior portion of the mouth is therefore imperative.

It is customary with most surgeons in operating upon these cases to cut away the protruding intermaxillary tubercle and close the lip upon the median line.

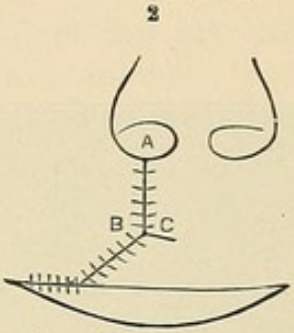
This certainly is the easier method, but it is open to serious objections; first, because this portion of the maxilla contains the incisor

FIG. 166.



OWEN'S OPERATION.

FIG. 167.



OWEN'S OPERATION.

teeth; second, because it removes the bony column upon which the nose rests; and third, because it produces an extreme narrowing of the face in the incisor and cuspid regions, with more or less complete stenosis of the nostrils, a deformity which can never afterward be remedied.

The preferable method is to replace the intermaxillary tubercle by removing an inverted V-shaped section from the vomer, carrying the tubercle into position and retaining it there by a wire passed through holes drilled in the vomer anteriorly and posteriorly, to the point of section, and allowed to remain until union has taken place.

FIG. 168.



HANESBY TRUSS.

If the lateral surfaces of the tubercle now come in contact with the lateral halves of the maxilla, the occluding surfaces should be freshened, and one or more fine silk sutures passed through the gum-tissue upon either side. When the surfaces do not occlude and the separation to be overcome is not very great, forcible pressure can be made upon the malar bones by the hands of the operator until the

surfaces meet, and afterward held in position by means of the Hanesby truss (Fig. 168) or rubber bandage, as suggested by Garretson.

In closing the lip, it is preferable to utilize the central portion if it is of sufficient width to admit the passage of sutures, rather than to cut it away, and unite the lip upon the median line.

The Golding-Bird operation is the one usually practiced by the writer in these cases; it consists in removing the vermilion border on all sides, leaving the prolabium with straight edges. The lateral halves of the lip are then dissected from their attachment to the bone, and pared to fit the trimmed prolabium. In order to obtain a sufficient amount of tissue to form a good lip, it sometimes becomes necessary to carry an incision around the ala of the nose and into the cheek. A deep suture is passed near the border of the lip, just above the edge of the mucous membrane, and another at the lower border of the ala of the nose to give proper shape to the nostrils,—it is often impossible to pass more than these,—and the edges of the skin and mucous membrane brought into nice apposition with fine interrupted silk sutures. To relieve tension and protect the wound it is covered with a collodion dressing, and over this an adhesive strip.

The writer prefers to replace the maxillary tubercle and close the lip at the same time. Operation upon the fissured palate should be deferred till a later period. This operation he has performed several times with uniformly good results. One child was but six days old, but the operation was borne well, and he took the breast three hours afterward. The others were between two and six months old.

Uranorrhaphy.—In operations upon the bony palate—uranoplasty—the writer usually prefers the Langenbeck method, which consists of first paring the edges of the cleft; second, making an incision through the soft tissues covering the hard palate close to the teeth, and lifting these tissues from the bone with a curved periosteotome, sliding them over the fissure, and uniting the periosteal surfaces together with the cobbler's stitch or suture. This method of stitching is a modification suggested by the writer, and gives more satisfactory results than the old method of uniting the edges by the interrupted suture.

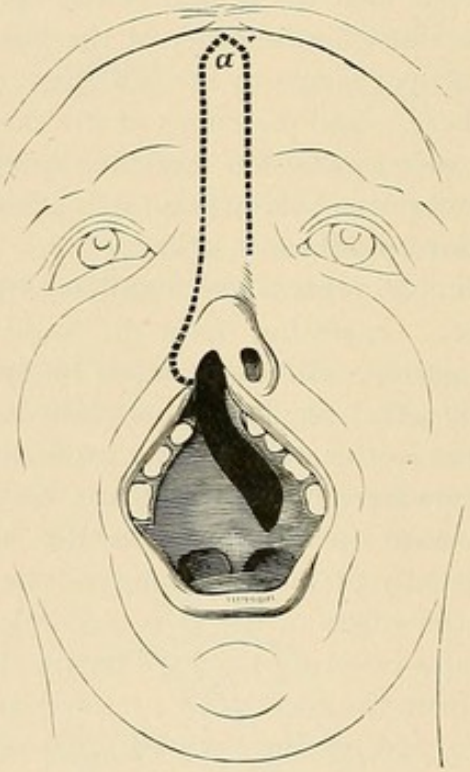
Fergusson's operation consisted of drilling the bony palate at short intervals from before backward, and then splitting it with a chisel. The edges of the cleft having been previously pared, are then united by silver wire sutures.

Billroth has recently modified the Langenbeck operation for closing fissure of the hard palate and velum, by approximating the internal wings of the sphenoid. This he accomplishes by dividing the mucous membrane at the sides of the velum, and fracturing the bone with a chisel. The mucous membrane is then utilized to close the cleft. His object is to do away with the necessity of dividing the muscles of the

velum, particularly the circular or sphincter muscle which surrounds the naso-pharyngeal opening, and which has its anterior fibers in the velum palati.

Rotter, of Munich, describes an interesting case of a child six years old, upon whom he had operated previously by the Langenbeck method for an uncommonly wide fissure; the muco-periosteal flaps had united upon the median line by first intention through their entire length, but left an opening upon the left side near the teeth, about four-tenths of an inch in width, giving free communication between the mouth and nasal cavity. To close this opening he raised a flap of skin from the forehead (Fig. 169), having a long pedicle attached, and the

FIG. 169.



ROTTER'S OPERATION FOR CLEFT PALATE. (After Rotter.)

gap in the forehead was immediately closed with sutures (Fig. 170). He then placed upon the raw surface of the flap numerous epidermal grafts after the method of Thiersch, placed the flap against the forehead with the raw surface undermost, and held it in position by a bandage.

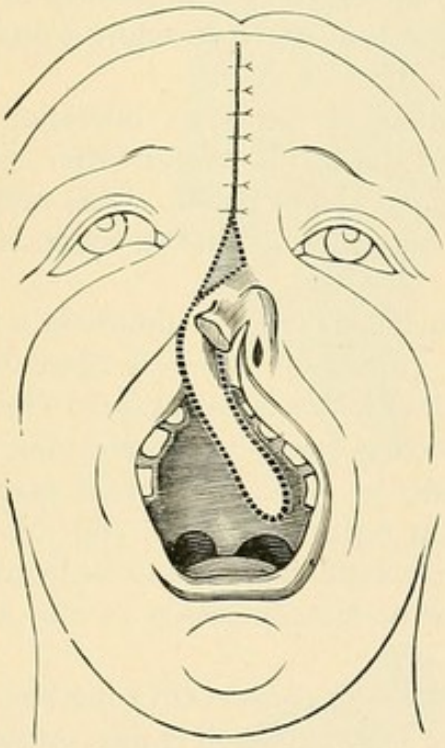
At the end of eight days the grafts had taken, and the flap was covered with skin upon both sides. The next steps were to lengthen the incision along the right side of the nose to the cleft in the lip, raise the right ala of the nose, freshen the edges of the opening in the palate, stitch the flap into position, close the fissure in the lip, and replace the ala of the nose. (Fig. 171.)

The case was successful, and Rotter exhibited the child two years later to the Congress of German Surgeons. One of the remarkable facts in the case was that though the flap was covered upon both sides with skin, the moisture in which it was constantly bathed seemed to have no deleterious effect upon it.

Rotter states that only two other cases are on record in which tissues had been transplanted for a like purpose from other locations than the palate itself, the first by Blaisus, the second by Thiersch.

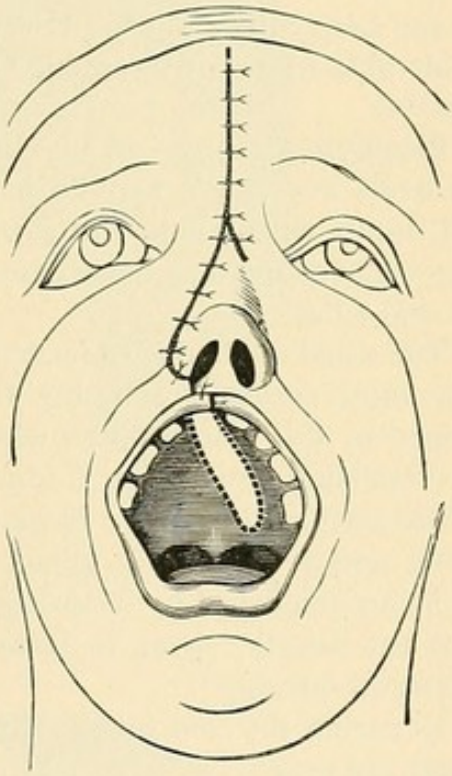
Brophy, of Chicago, has recently devised a new method of approximating the edges of fissure of the palate which is unique and original, and for which he claims a decided advantage over other operations.

FIG. 170.



ROTTER'S OPERATION FOR CLEFT PALATE.
(After Rotter.)

FIG. 171.



ROTTER'S OPERATION FOR CLEFT PALATE.
(After Rotter.)

The method consists substantially in passing two double silver wire sutures through the superior maxillary bone, within the mouth; the posterior one inserted just *behind* the malar process, and high enough to pass over the palate plate of the bone, emerging at the same point upon the opposite side; the anterior one is passed through the bone just in *front* of the malar process. A lead button having two eye-holes is threaded upon the wire, and the ends twisted together. The edges of the cleft having been previously freshened, the wires are twisted until the edges of the cleft are brought together. If the resistance is such that the edges do not readily approximate, the malar

process is divided on either side by the aid of a heavy scalpel. The edges of the cleft are united by sutures in the usual way.

This operation would seem to be valuable if performed during the early months of infancy, while the bones are still imperfectly calcified, in those cases where the deficiency of tissue is slight and the edges of the fissure but moderately separated. Where the cleft is wide and the deficiency of bony tissue considerable, it might succeed in closing the defect in the palate; though it would establish another deformity equally grave in character, viz: partial or complete stenosis of the nasal passage of the affected side. It is to be presumed, therefore, that Brophy would not advise this operation, only in selected cases where this condition could not result.

In the cases first mentioned it would seem to have an advantage over the Langenbeck muco-periosteal operation; but in fissures of any considerable width, or in double fissure, the latter procedure would be preferable.

Staphylorrhaphy.—A modification of Nélaton's operation for bifid uvula and single hare-lip has, in the hands of the writer, given good results when applied to closing the velum palati. This method is especially adapted to those cases where the cleft in the velum is the only oral defect.

The usual method is to freshen the edges of the velum with a bold hand—most surgeons claiming that successful union is more often obtained by this method than when less tissue is cut away. This, so far as obtaining good union is concerned, is a self-evident fact; but why sacrifice tissue when there is generally so little in the first place to operate upon? The main object is to restore function, and this can only be accomplished by restoring the velum and uvula to their proper width and length, so as to insure a perfect occlusion of the naso-pharyngeal opening.

In paring the cleft by either the bold or conservative method, the parings are usually sacrificed. By the method which is now presented the parings are utilized to broaden and lengthen the velum and form a new uvula.

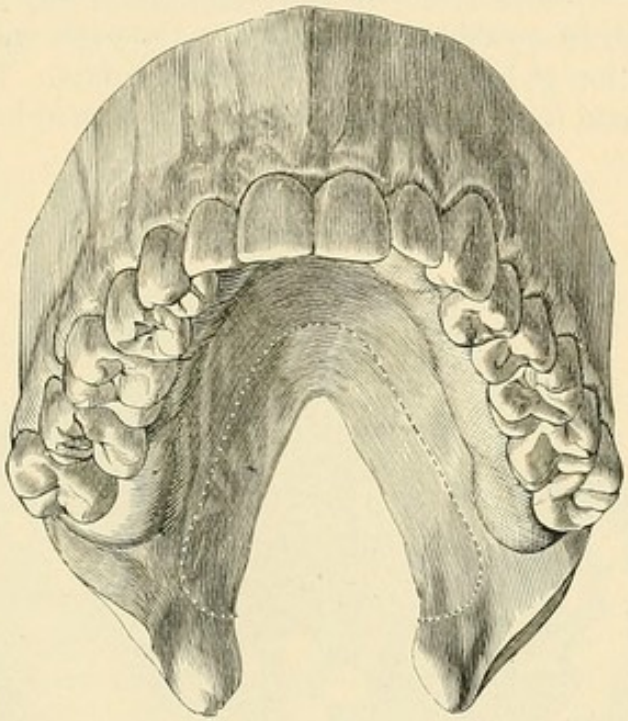
In the act of deglutition the velum palati is drawn up against the walls of the pharynx, the base of the uvula filling the depression upon the median line formed by the approximation of the two sides of the pharynx, thus perfectly closing the naso-pharyngeal opening and preventing the escape of food into the nasal passages during the act of swallowing, and materially assisting in the production of articulate speech by preventing the escape of certain sounds through the nasal passages.

By the modified Nélaton operation every particle of tissue is saved

and carried to that part of the velum where it is most needed to improve the shape, the width, and the length.

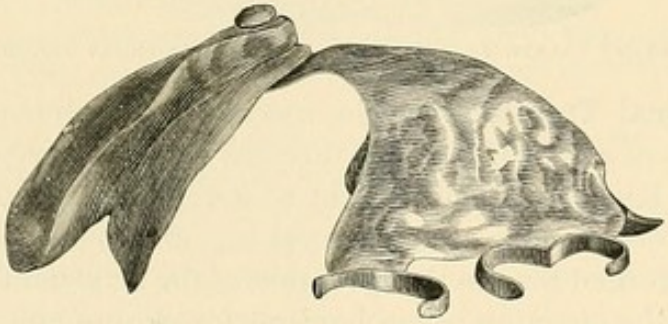
The method is similar to that often practiced upon single harelip, viz: a curved, pointed bistoury is inserted just above the apex of the cleft, and carried downward toward the uvula of one side, about one line from the cleft at the apex, gradually increasing the distance as

FIG. 172.



CLEFT OF THE SOFT PALATE.

FIG. 173.



KINGSLEY'S ARTIFICIAL VELUM. (After Kingsley.)

the bistoury approaches the uvula to about two lines from the edge of the cleft and three lines from the posterior border of the velum. This incision is repeated upon the opposite side.

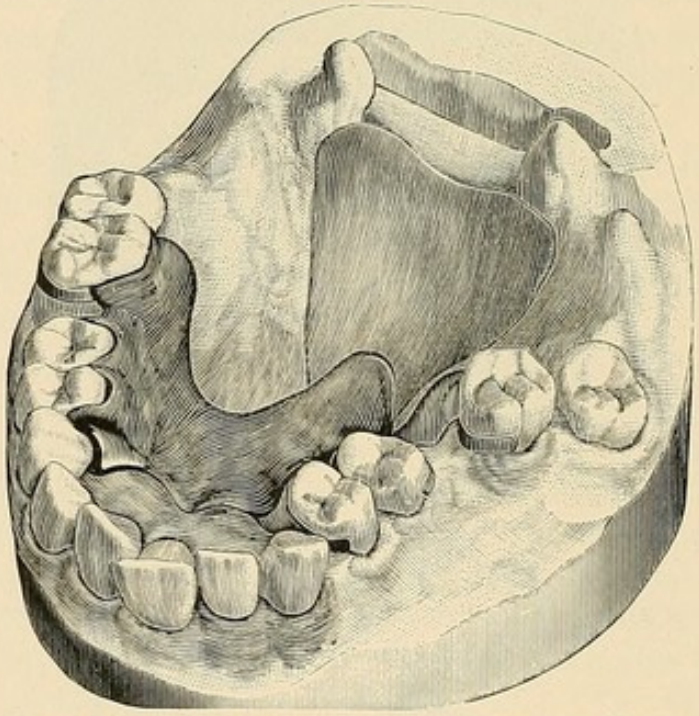
The apex of the paring is then carried backward, and the freshened edges of the palate, after having been split to the depth of about one-fourth of an inch, are approximated and sutured. The object in

splitting the palate is to gain a broader surface of tissue at the edges of the cleft, and thus increase the chances of primary union.

Three or four sutures are usually inserted, silver wire being given the preference as less likely to induce suppuration or ulceration about the sutures. Tension is relieved when necessary by dividing the tensor palati muscle upon either side, but this procedure is by no means always indicated.

The after-treatment consists in keeping the wound and sutures as clean as possible by swabbing and spraying the parts with the Thiersch antiseptic solution at least every two or three hours. The sutures are allowed to remain from four to ten days, according to indications.

FIG. 174.



CLEFT PALATE TREATED BY KINGSLEY'S ARTIFICIAL VELUM.

Mechanical Treatment.—The mechanical treatment of cleft palate by means of artificial vela has through the inventive genius of Dr. Norman W. Kingsley been brought to a very high degree of perfection. In no department of mechanical surgery has a greater achievement been recorded than in the invention of the artificial flexible velum. All efforts in the direction of mechanisms for closing congenital defects in the palate which preceded the apparatus of Kingsley were crude, clumsy, rigid affairs, and of but little practical utility to the patient. The discovery by Goodyear of the process of vulcanizing rubber made it possible for Kingsley to construct an appliance from a material which by its adaptability could be readily formed in moulds made from accurate impressions of the defective parts, and adjusted to them in such a manner as to be free from irritation. The form of the appli-

ance is such that it is under complete control of the muscles of the parts, rising and falling with the contraction and relaxation of the levator palati muscles, thus opening and closing the naso-pharyngeal space and preventing the regurgitation of fluids into the nose and making it possible with proper training of the vocal organs to attain comparatively perfect speech. Fig. 172 represents a typical case of congenital cleft of the velum palati. Fig. 173 shows the mechanical construction of the velum and plate to retain it in its position in the mouth. In Fig. 174 the velum is shown in its position.

REVIEW.

CHAPTER XLI.

Who first performed the operation of staphylorrhaphy, and at what date?

Who revived the operation, and at what date?

What was the date of Roux's first operation?

When did Warren make his first operation?

To whom belongs the credit of first demonstrating and giving to the world a scientific basis for the operation of staphylorrhaphy?

What is the first important question in relation to the surgical treatment of cleft palate?

At what age is the best time to operate for cleft palate?

Why are operations more successful at this age?

What abnormal conditions are prevented by an early operation?

What anesthetic is best in mouth operations?

What is the best position of the patient for the operation of staphylorrhaphy?

Why is this position valuable in this operation?

What important fact should be borne in mind in uniting fissures of the lip?

How may tissue be joined and strain be prevented upon the freshly united edges?

Describe the common method of closing fissures of the lip.

Describe Fenger's operation for hare-lip.

What age is the best time to operate for hare-lip?

Describe Mirault's operation for hare-lip.

What is the surgical procedure in double hare-lip with protrusion of the intermaxillary bone?

Why is an early operation advisable?

What is the usual procedure in reference to the intermaxillary bone?

What is the object in conserving this portion of the maxilla?

What is the preferable method?

How may this be done?

What means can be used to unite the intermaxillary with the lateral halves of the maxilla?

What should be done with the central portion of the lip in these cases?

Describe the Golding-Bird operation for double hare-lip.

Describe the Langenbeck operation for closing the hard palate.

Describe Fergusson's operation.

What is Billroth's modification of the Langenbeck operation?

Who made the first operation for transplanting tissue to close a cleft in the palate?

How many operations of this character are on record, and who were the operators?

Describe the Brophy operation.

Has this operation any advantage over the Langenbeck operation?

What are the dangers from the Brophy operation?

What is the value of the Langenbeck operation over the others?

Describe the Nélaton operation, as modified by the writer, for closing the soft palate.

What are the advantages of this operation?

What is the after-treatment of operations upon the palate?

CHAPTER XLII.

TUMORS.

Before entering upon a description of the tumors of the face, mouth, and jaws, it will be of advantage to consider briefly the origin, structure, growth, character, and classification of tumors in general.

Definition.—Tumor (Latin *Tumere,* to swell).

A tumor is an enlargement or swelling of a part. A better definition, and one more in accord with the later and stricter use of the term, is "any new growth not the result of inflammation or hyperplasia."

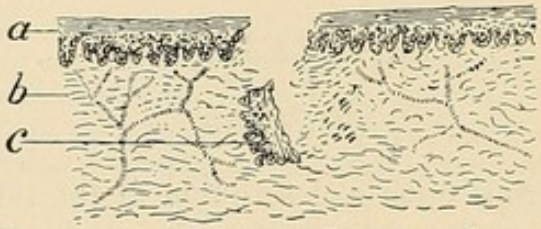
In the later classification of tumors by pathologists, a sharp distinction is drawn between tumors, inflammatory swellings, and retention cysts.

Inflammatory hyperplasia of tissue due to infection by micro-organisms is sometimes mistaken for a neoplasm, and *vice versa.* The difference between them, however, may be recognized by several important features in their history. In inflammatory swellings, the growth and extension are often very rapid and progressive, but not continuous nor permanent. There is lack of definite outline; they are amenable to agents which promote absorption by neutralizing or removing the primary cause, and they are subject to early and acute degenerative changes. An acute suppurative inflammation, on account of its violent local and general symptoms, is rarely mistaken for a malignant neoplasm; while, on the other hand, new growths are usually characterized by their definite outline, slow but progressive growth, permanency of the new-formed tissue, and their resistance to internal medication.

In certain forms of innocent tumors, like neuroma and osteoma, growth becomes spontaneously arrested when they have reached a definite size. The nearer the new growth resembles normal tissue, the greater the probability that it will be spontaneously arrested in its growth. Occasionally, in rapid-growing malignant neoplasms, such inflammatory symptoms as enlargement of the superficial veins and edema may be present. Senn emphasizes the fact that the nearer a malignant tumor resembles an inflammatory swelling the greater is its malignancy.

Origin.—Nearly all new growths originate from misplaced embryonic cells, and this constitutes the matrix from which the tumor is developed. Cohnheim was of the opinion that all tumors were of congenital origin, and were developed from a matrix of embryonic tissue,

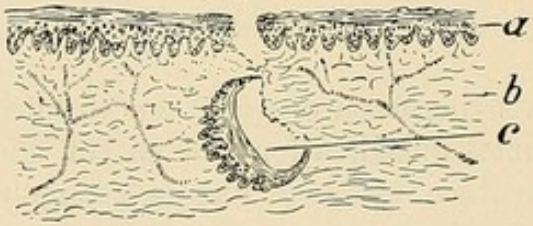
FIG. 175.



MANNER OF PRODUCTION OF TRAUMATIC EPITHELIAL CYST. (After Garré.)
a, skin; *b*, subcutaneous tissue; *c*, dislocated fragment of skin.

while Garré, Senn, and others believe they may be occasionally of post-natal origin, and independent of causes arising from the action of micro-organisms,—that they may be derived from pre-existing *mature* cells, which in consequence of injury or disease fall short of complete

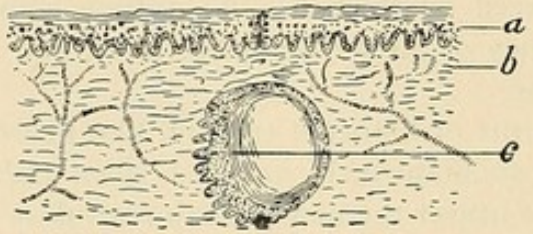
FIG. 176.



BEGINNING OF HEALING OF THE SKIN-DEFECT AND COMMENCING PROLIFERATION FROM THE MARGINS OF THE IMPLANTED SKIN. (After Garré.)

differentiation, thus forming a tumor-matrix, from which a neoplasm may be developed in the same manner as from embryonic cells which have been misplaced during fetal life. Figs. 175, 176, and 177 illustrate the origin of a post-natal epithelial tumor.

FIG. 177.



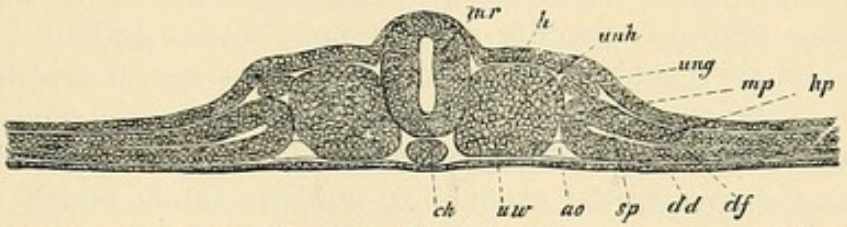
WOUND ENTIRELY HEALED, AND THE BURIED SKIN-GRAFT ENLARGED BY PROLIFERATION FROM THE SURFACE AND MARGINS OF THE GRAFT. (After Garré.)

Cohnheim was the first to teach that all tumors were developed from embryonic tissue, and to trace their origin to the various layers of the germinal disk. Ziegler, however, looks upon the theory of Cohnheim as more or less hypothetical, and the present knowledge of

the etiology of tumors in general as still very defective. He considers the histologic evidence of the existence of embryonal germinal tissue in the fully developed organism as very inadequate, and that it is a "bold step to ascribe an embryonic origin to all forms of tumors." The two latest and most reliable authorities upon the subject of tumors—Sutton, "Tumors, Innocent and Benign," 1893, and Senn, "Pathology and Surgical Treatment of Tumors," 1895—both accept the teaching of Cohnheim in relation to the congenital origin of nearly all forms of neoplasms and their development from misplaced embryonic tissue.

Germinal Layers.—Pander discovered, in 1847, that in the embryo of the chick the germinal disk was composed of three layers,—the *external* he denominated the serosa; the *internal*, the mucosa; and the *middle*, a muscular layer. They are now usually designated as the external layer or *epiblast*, the internal or *hypoblast*, and the middle layer or *mesoblast*. The layers can be plainly distinguished and their complicated arrangement readily traced in the embryo of the chick on the second day of incubation. (Fig. 178.)

FIG. 178.



TRANSVERSE SECTION THROUGH EMBRYO OF CHICK TWO DAYS OLD. × 100. (After Kölliker.)

dd, hypoblast; *ch*, cord; *uw*, primitive vertebra; *unh*, primitive vertebral canal; *ao*, primitive aorta; *ung*, primitive urinary canal; *sp*, cleft in lateral plates (first indication of pleuro-peritoneal cavity), which though lost in the *hp* and intestinal connective-tissue plates *df*, are connected through the mesoblast *mp*; *mr*, medullary tube; *h*, epiblast thickened at some points. The embryo at the time is composed of two epithelial layers, the outer the *epiblast*, the inner the *hypoblast*, connected by the middle layer, the *mesoblast*.

Embryologists trace the origin of all the tissues and organs of the vertebrate animals, including the human species, to these three general layers, which may be distinguished in the various embryos during the first few days after conception. From the *epiblast* are developed all those tissues and organs of epithelial structure, the skin and its glandular appendages, the hair, the nails, the lens of the eye, the brain and spinal cord, the epithelial lining of the mouth, the nasal passages, the labyrinth of the ear, and the teeth. From the *hypoblast* there are developed the mucous membrane of the entire alimentary tract, with all its glandular appendages, the urinary organs, the liver, the lungs, the thyroids, and the kidneys. From the *mesoblast* is formed the great bulk of the body, viz: The bones, the connective tissues, the muscles, the nerves, the serous membrane and its glands, the vascular organs, the lymphatics, the ductless glands, the thymus, and the spleen.

In the differentiation of the cells which takes place in the embryo, each cell is endowed with its own particular genetic function of forming other cells like itself, but farther than this it cannot go. Cells may be arrested in their growth, and their character thereby somewhat changed, but there is never a transition from one variety to another, and this law remains in force during the entire life of the organism.

When incomplete differentiation has taken place in some misplaced portion of one of these germinal layers, it often remains buried in the tissues in its embryonic state for an indefinite period, but it may take on active growth at any time, with the result of invariably producing a tumor corresponding in its structure to the variety and stage of development of the cells from which it had its origin.

Structure.—"A tumor-matrix of congenital origin always represents normal tissue-elements in an abnormal place." (Senn.) The histologic structure of the tissue which composes a new growth is governed by the inherent genetic function of the embryonic cells which form the tumor-matrix. A matrix derived from the epiblast or the hypoblast would invariably produce a tumor of the epithelial type, while if derived from the mesoblast the result would be the formation of a tumor of the connective-tissue type. Senn maintains that the character of the neoplasm "depends upon the stage of arrested cell-growth" in the tumor-matrix. The nearer the tissue composing the tumor-matrix approaches the completion of the process of cell-differentiation, the greater the probability that the tumor which may be developed from it will be benign in character, while, upon the other hand, the nearer it resembles embryonic tissue—the more immature the cells—the greater the liability that the tumor will be of a malignant type.

A tumor-matrix derived from the epiblast or the hypoblast, and composed of cells in which the process of differentiation has been almost completed, would give rise to a benign tumor of the epithelial type, viz: a papilloma, or an adenoma, while if composed of cells in which this process was arrested in its earlier stages, it would result in the formation of a malignant tumor of the same type, an epithelioma or a carcinoma. Senn makes no distinction in malignant tumors of the epithelial type, but classes them all as carcinomata.

In tumors arising from the mesoblastic layer, the same conditions are manifest; a tumor-matrix composed of connective-tissue cells of high differentiation will produce an innocent tumor of the connective-tissue type, viz: A fibroma, chondroma, or an osteoma, while if of low differentiation it will be likely to result in the development of a sarcoma.

The structure of benign tumors so closely resembles normal tissue, both macroscopically and microscopically, that it is many times

exceedingly difficult to distinguish between them. The same is true of their cellular elements. The cells retain the original form and type of the tissues from which they had their origin, and as they reach their highest degree of development it becomes almost impossible to differentiate between the tumor-cells and the normal cells representing the tissue from which they originated.

The structure of malignant tumors is distinguished by the embryonic or immature character of the tumor-cells, which closely resemble the fixed-tissue cells in their early stage of development. "The striking difference between a sarcoma cell and an immature connective-tissue cell is in the size and number of their nuclei." (Senn.) In the sarcoma cells the nucleus is large and often multiple, while in the connective-tissue cells it is single and much smaller in comparison with the nucleus of the sarcoma cells. Another distinguishing feature of the sarcoma cells is their lack of uniformity in size, form, and color. The chief varieties of the sarcoma cells are the round, fusiform, myeloid, and pigmented.

In malignant tumors of the epiblast and hypoblast the cells are characterized by their immature or embryonic development, and they bear a very close resemblance to the cellular elements which are found in these layers of the germinal disk. Here, also, the cells lack uniformity in size and shape. They vary in size from 1-600 to 1-1500 of an inch in diameter, and are polygonal, round, oval, caudate, and fusiform in shape, while the nuclei of the epithelioma and carcinoma cells, as in the sarcoma cells, are multiple. At one time the polymorphic character of the cells in epithelial tumors was supposed to be diagnostic of carcinoma, but it is now thought to be the result of rapid cell-proliferation and pressure. Nevertheless, the polymorphic character of the cells and the large multiple nuclei, though they cannot be considered as positively diagnostic of a malignant growth, yet they cast suspicion of malignancy upon all neoplasms in which they are found.

Growth.—Tumor-cells multiply, like their prototypes, the normal cells, by a process of indirect division, or segmentation, known as *karyokinesis.* Nearly all the fixed-tissue cells of the higher type multiply in this manner. Growth in benign tumors is, as a rule, much slower than in the malignant form. A fibroma may require years to attain the size of a man's fist, but a malignant growth may reach the same dimensions in a few months. Cell-proliferation is very active in the malignant tumors, which results in rapid growth and defective development of the cellular elements. The *kinetic* process, as described by Strassburger, consists of three stages. During the first stage, or *Prophase,* the nuclear chromatin arranges itself in the form of an oval mass. In the second stage, or *Metaphase,* the nucleus elon-

gates or becomes barrel-form, with a suggestion of equatorial division. During the last stage, or *Anaphase,* the nucleus and protoplasm are divided into two symmetrical halves, and separate, thus completing the process of segmentation. (See Figs. 55, 56, 57, 58.) Segmentation of the nucleus without division of the protoplasm results in the formation of multinuclear and giant cells. Such imperfect karyokinesis is frequently found in the cellular elements of malignant neoplasms. In the study of the kinetic process in malignant tumors, pathologists have discovered that the same phases are passed through as in the formation of normal tissues, with the exception that complete cell-differentiation is seldom reached in this form of tumor.

Character.—Clinically all neoplasms are divided into three classes, viz: *Benign, malignant,* and *suspicious.* The class to which each belongs is determined by the character of the tissue which composes them, and the stage of development of the cellular elements. The more nearly a tumor approaches to normal tissue of the class from which it originated, the greater are the chances that it will prove benign; while, on the other hand, the more nearly the tissue and cells simulate an embryonic development, the greater is the liability that the tumor will be malignant. The suspicious tumors are those which do not belong strictly to the benign or malignant forms, but contain elements of both—the mixed tumors—or which are liable to take on active cell-proliferation of an embryonic character later in life, as the result of irritation.

Certain forms of tumors may remain benign in character for years, when suddenly they take on active growth or cell-proliferation, and assume a malignant type. This is particularly true of papilloma and adenoma. Senn believes that this sudden change in the clinical behavior of tumors is not an evidence of the semi-malignant nature of the growth, but that it was either malignant from its incipiency, or that it has undergone changes which give it a malignant character.

The most marked clinical features which distinguish the benign from the malignant tumors are: First, a benign tumor never extends to other tissue than that from which it had its origin, while all malignant tumors extend to and involve other tissues than those from which its matrix was derived. For instance, a benign epithelial growth is always confined to the epithelial structures of the epiblast or the hypoblast, while a malignant epithelial tumor would involve the tissues in its neighborhood irrespective of their character or their origin. Second, a malignant tumor has a tendency, which is made manifest early in its history, to involve the lymphatic glands, beginning with those which are most closely associated with the particular location of the tumor, and extending through the lymph-channels to others farther removed. These two features have been regarded for a

long time as the most reliable clinical evidence of the malignant character of tumors. The manifestation of these evidences, however, particularly the latter, comes too late to prove of value from the standpoint of effecting a radical cure. When extension to and involvement of surrounding tissues has become very marked, or the lymphatics are implicated, there is little hope of a radical cure of the disease.

The character of the tumor can be diagnosed in most cases by the microscope, and yet, as we have already seen, there are certain malignant tumors which have a cellular structure in their early stages, so closely resembling the embryonic cells of the various germinal layers from which they spring, that it is difficult to distinguish between them. Consequently, every tumor which cannot be satisfactorily diagnosed to be a benign growth, should be classed as malignant, and immediately extirpated as the only safe method of dealing with it. Waiting for positive evidence of malignant character before recommending a radical operation is foolhardy in the extreme, for when positive evidence is at hand, it may be too late to avert a fatal termination of the disease.

Classification.—Tumors are now usually classified from two standpoints: First, from their origin and histologic structure, and second, from the stage of development of the cells composing the tumor-matrix. The germinal layers from which the neoplasms have their origin indicate the type of the growth, while the stage of arrested development of the cellular elements of the matrix will indicate the character of the tumor. Tumors of highly organized tissue represent the benign growths. Those approaching embryonic conditions of tissue represent the malignant growths. The first systematic classification of tumors was that made by Virchow, in which he made the attempt to arrange all tumors according to their histologic structure.

Virchow's Classification of Tumors.

1. Histioid;
2. Organoid;
3. Granulomata;
4. Teratoid;
5. Combination tumors;
6. Extravasation and exudation tumors.
7. Retention cysts.

This classification, in view of our present knowledge of the origin and cause of certain tumors, seems very imperfect indeed.

All tumors composed of one kind of cells Virchow classed as "histioid tumors." Klebs maintains that a pure histioid tumor is only found in very small epitheliomas, sarcomas, and in angeiomas. As "organoid tumors" he classed all those growths which are composed

of several kinds of cells arranged in definite and typical forms, representing the structure of organs. Senn thinks this term is incorrect and misleading, for the reason that even the most perfectly developed adenoma, or any other form of tumor, for that matter, is devoid of physiologic function. Among the "granulomata" which are infective inflammatory swellings, he includes carcinomata and sarcomata, but until it can be proved that these tumors are the result of microbic infection, they have no place with the granulomata. The "teratoid" tumors include all those growths which are composed of a system of organs, like dermoid cysts, an extra digit or limb, or a fetus within a fetus. "Combination tumors" are those which are composed of two or more kinds of tumor tissues. The "extravasation and exudation tumors" are the result of traumatism or inflammation, and should be classed with the granulomata and inflammatory swellings. "Retention cysts" should also be classed by themselves, as they are in no sense neoplasms.

Virchow also divided tumors into two general groups. One he denominated Homologous, and the other Heterologous. In the first group he included all tumors whose histologic structure closely resembles normal tissue, and reproduces the type of tissue of the organ or part in which they are located. In the second group he placed all growths which deviate in their histologic structure from the type of tissue from which the tumor had its origin. From the clinical standpoint all homologous tumors may be said to be benign, and, in a general way, all heterologous tumors may be classed as malignant. There are exceptions, however, to this rule. A chondroma is a heterologous tumor, but it is benign; while on the other hand malignant tumors sometimes, in their early stages, present a homologous structure. Malignant tumors, however, are always heterologous in their structure.

Cohnheim, as already stated, was the first to classify tumors according to their embryonic origin. The classification is, however, somewhat imperfect, as the myomata and neuromata should be classed with the tumors of the connective-tissue type.

Cohnheim's Classification of Tumors.

I. Connective-tissue Type.	Fibroma (Fibrous-tissue tumor).
	Lipoma (Fatty tumor).
	Myxoma (Mucoid tumor).
	Chondroma (Cartilaginous tumor).
	Osteoma (Bony tumor).
	Angeioma (Vascular tumor).
	Lymphangeioma (Lymphatic-vessel tumor).
	Lymphoma (Lymphatic-gland tumor).
	Sarcoma (Embryonic connective-tissue tumor).

II. Epithelial Type.	Epithelioma (Epithelial tumor of skin or mucous membrane). Onychoma (Horny tumor). Struma (Scrofulous tumor). Cystoma (Cystic tumor). Adenoma (Glandular tumor). Carcinoma (Embryonic epithelial-tissue tumor).
III. Myomata.	Myoma lævi-cellulare (Involuntary-muscle tumor). Myoma stri-cellulare (Voluntary-muscle tumor).
IV. Neuromata.	Neuroma (Nerve-tissue tumor). Glioma (Klebs). (Connective-tissue tumor of nerve and brain.)
V. Teratomata.	Teratoma (Virchow). (Tumors composed of various tissues or organs. Monstrosities.)

Sutton, in his classification of tumors, makes the attempt to bring order out of confusion by following lines of classification similar to those employed in biology, and divides tumors into four groups, viz: First, connective-tissue tumors; second, epithelial tumors; third, dermoids; fourth, cysts. These he again subdivides into genera, species, and varieties, according to their histologic structure and character.

This classification is a great improvement over those of Virchow or Cohnheim, by reason of the fact that it is based upon the origin and the character of the histologic structures which compose the tumors.

Sutton's Classification of Tumors.

I. Group, Connective Tissue.	Lipomata. Chondromata. Osteomata. Odontomata (tooth tumors). Fibromata. Myxomata. Gliomata (neuroglia tumors). Sarcomata. Myomata (muscle tumors). Neuromata (tumors on nerves). Angeiomata. Lymphangeiomata.

II. Group, Epithelial Tissue.
- Papillomata
  - Skin warts.
  - Villous papillomata.
  - Intra-cystic warts.
  - Psammomata (Meningeal tumors).
- Epithelioma (Epithelioma).
- Adenoma
  - Mammary. Renal.
  - Sebaceous. Ovarian.
  - Thyroid. Testicular.
  - Pituitary. Gastric.
  - Prostatic. Intestinal.
  - Parotid. Fallopian.
  - Hepatic. Uterine.
- Carcinoma (cancer)
  - Mammary. Renal.
  - Sebaceous. Ovarian.
  - Thyroid. Testicular.
  - Prostatic. Gastric.
  - Parotid. Intestinal.
  - Pancreatic. Fallopian.
  - Hepatic. Uterine.

III. Group, Dermoids.
- Sequestration dermoids; Tumors from hidden surface epithelium.
- Tubulo-dermoids; Tumors from obsolete fetal canals.
- Ovarian dermoids.
- Dermoid patches.

IV. Group, Cysts.
- Retention cysts.
- Tubulo-cysts.
- Hydroceles.
- Gland cysts.

Sub-group, Pseudo-cysts.
- Diverticula.
- Bursæ.
- Neural cysts.
- Parasites.

Senn classifies tumors with special reference to their relationship to the various germinal layers, and the stage of arrested cell-development in the elements composing the tumor-matrix.

Senn's Classification of Tumors.

I. Epiblastic and Hypoblastic Tumors.
- Papilloma.
- Adenoma.
- Cystoma.
- Carcinoma.

II. Mesoblastic Tumors.	Fibroma.	
	Lipoma.	
	Myxoma.	
	Chondroma.	
	Osteoma.	
	Angeioma.	
	Lymphangeioma.	
	Lymphoma.	
	Myomata	(Lævi-cellulare; Stri-cellulare).
	Neuromata	(Neuroma; Myelinic; Amyelinic).
		Glioma (Klebs).
	Sarcoma.	
III. Epiblastic, Hypoblastic, and Mesoblastic Tumors.	Teratomata.	
IV. Swellings caused by retention of physiologic secretions.	Retention cysts.	

REVIEW.

CHAPTER XLII.

Give the definitions of *tumor*.
What conditions are sometimes mistaken for neoplasms?
How may inflammatory swellings be recognized?
How recognize new growths?
In what form of tumor is growth spontaneously arrested?
Do malignant tumors ever resemble inflammatory swellings?
What does this indicate?
From what do neoplasms originate?
What were Cohnheim's views?
What are Senn's modifications?
Name the germinal layers of the disk of Pander.
What tissues and organs are developed from the epiblast?
What tissues and organs are developed from the hypoblast?
What tissues and organs are developed from the mesoblast?

With what peculiar function are cells endowed?

Are cells of one variety ever changed into another variety?

What becomes of the misplaced embryonic tissue?

What does a tumor-matrix of congenital origin always represent?

What governs the histologic structure of new growths?

A tumor-matrix derived from the epiblast or hypoblast would produce what form of tumor?

What form of tumor would be produced if derived from the mesoblast?

The character of the tumor depends upon what condition in the tumor-matrix.

What condition of cell-growth favors the development of benign tumors?

What condition favors the development of malignant growths?

What type of tumor would be developed from a matrix derived from the epiblast or hypoblast with cell-differentiation almost completed?

What type, if cell-differentiation was arrested in its early stages?

What type of tumors is developed from the mesoblast under like conditions?

What resemblance is found between benign tumors and normal tissue?

How are malignant tumors distinguished?

What is the difference between a sarcoma cell and an immature connective-tissue cell?

What other conditions distinguish them?

What are the distinguishing features of malignant tumors of the epiblast and hypoblast?

What is the cause of the polymorphic character of the cells?

What do they indicate as to the character of the tumors?

How do tumor-cells multiply?

By what term is the process known?

What character of tumor is slowest in growth?

In which is cell-proliferation most active?

Name the stages of the *kinetic* process or cell-segmentation.

Describe the process in the first stage—Prophase.

Describe the process in the second stage—Metaphase.

Describe the process in the third stage—Anaphase.

How are giant cells formed?

Is the kinetic process the same in the growth of malignant tumors as in normal tissue?

How are tumors divided clinically?

By what is their character determined?

What are suspicious tumors?

What forms of benign tumors sometimes take on a malignant character late in life?

How is this explained?

What are the clinical features which distinguish benign from malignant tumors?

What are the chances of cure after the lymphatics have become involved?

How may diagnosis be made of the character of the tumor?

What course should be pursued when the tumor cannot be proved to be benign?

From what two standpoints are tumors now classified?

What form of tissue represents the benign growths?

What form of tissue represents the malignant growths?

How did Virchow classify them?

Name some of the objections to this method of classification.

Into how many groups did Virchow divide tumors?

Name them.

What is the character of all homologous tumors viewed from the clinical standpoint?

What is the character of the heterologous tumors from the same standpoint?

What are the objections to Cohnheim's classification?

What is the method of classification adopted by Sutton?

Into how many groups does he divide them?

Name these groups.

Upon what does he base the subdivision?

How does Senn classify tumors?

CHAPTER XLIII.

TUMORS OF THE FACE, MOUTH, AND JAWS.

Epithelial Tumors.

The various forms of tumors which are most commonly found associated with the face, mouth, and jaws are:

Epithelial Group, or *Epiblastic* and *Hypoblastic Tumors.*	Papillomata.
	Adenomata.
	Cystomata.
	Carcinomata.
Connective-Tissue Group, or *Mesoblastic Tumors.*	Fibromata.
	Chondromata.
	Osteomata.
	Angeiomata.
	Sarcomata.
Composite Group, or *Epiblastic, Hypoblastic,* and *Mesoblastic Tumors.*	Odontomata.
Swellings resulting from retention of normal secretions.	Retention Cysts.

In the consideration of the subject of tumors located in the region of the face, the foregoing order will be followed, for the reason that the student, it is hoped, will gain a better knowledge of the character and tendencies of such growths if they are studied in separate groups, and according to their classification, from the standpoint of their histogenesis and morphology.

The *Epithelial group* comprises all of those neoplasms which have their origin in the *epiblast* or *hypoblast*. In these tumors the epithelial elements predominate; in fact, they constitute the essential and distinguishing morphologic forms. The functions of the epithelium are exceedingly varied in man and in animals of complex organization; in certain locations it acts as a protection; as, for instance, the epidermis; it also becomes modified into hair, nail, horn, or enamel; in others the epithelial cells extend into the connective tissue beneath, in the form of processes, to form secreting glands, some of which are

simple; as, for instance, the tubular glands of the intestines; others are complex, such as the liver, kidneys, pancreas, mammæ, and the salivary glands. But whether the gland is simple or complex, the underlying principle of construction is the same (Sutton), and is characterized by narrow canals lined with epithelial cells arranged in a definite order, the canals resting upon a connective-tissue groundwork, which is ramified with blood-vessels, lymphatics, and nerves. The canals, or "epithelial recesses," of a gland are known as *acini*. Each acinus either directly communicates with a free surface of the body by means of its own duct, as in the simple mucous and sebaceous glands; or through several main ducts, as in the complex structure of the mammæ; or by a common duct, as in the equally complex structures of the parotid gland and the pancreas. Sutton calls attention to three notable exceptions to this rule, viz: the thyroid glands, the pituitary bodies, and the ovaries. All other secreting glands possess means of direct communication with free surfaces of the body, and are therefore subject to infection from all forms of micro-organisms.

All epithelial tumors are composed of two kinds of tissue,—epithelial cells and vascular connective tissue, the latter forming the stroma or framework in which the epithelial cells are imbedded (Ziegler); while the number, character, and arrangement of the cells indicate the variety of tumor and its benign or malignant tendencies. The general plan of construction of epithelial tumors is that of a simple gland, and this form is maintained throughout many phases of their development. The degree of resemblance, however, differs greatly in the various forms. The *adenomata* most nearly resemble the structure of some particular glandular type. The tumors which are farthest removed from this regularity in structure are the *epitheliomata* and the *carcinomata*. In these the epithelial cells are arranged in "compact, irregular masses,"—"cell nests,"—while in the *adenomata* the tendency is to a regular arrangement, the cells lining the inner wall of the alveoli and leaving an open space or lumen which corresponds to the saccule or acinus of a gland. It has already been intimated, in the preceding chapter, that tumors which in their structure closely resemble the normal tissues of the part in which they are located are benign, and that tumors of an opposite character are malignant, or, to state it more correctly, the nearer the cells which give special character to the tumor approach complete differentiation the more certain are they to be innocent; while, on the other hand, the more nearly these cells approach an embryonic condition of development the greater the certainty that they are malignant.

Epithelial tumors are developed by a multiplication of the epithelial cells. These are imbedded in the connective tissue, which is also undergoing multiplication of its cellular elements. This multi-

plication of cells may be, in certain forms of tumors, in the line, apparently, of a normal development of tissue, but in abnormal numbers, as when located in the papillary layer of the skin or mucous membrane, and resulting in the formation of *papillomata;* or the new formation may exhibit a structure resembling that of glandular tissue, and result in the formation of *adenomata;* in others the structural arrangement may very imperfectly represent glandular tissue, exhibiting only the earliest stages of gland formation; or the epithelial cells may be gathered into "irregular, compact masses," with a tendency to unlimited growth, as in carcinoma.

In the epithelial type of tumor, especially in carcinoma, the epithelial cells lie in close contact with one another, and are seemingly united by a cement substance, or are continuous with one another (Warren). These "cell nests" are not directly supplied with blood-vessels. The connective-tissue framework or stroma, which is arranged in the form of alveoli, contains the blood-vessels. The epithelial cells lie within the alveoli, and are sometimes so arranged as to give the appearance of a "bird's nest." The absence of tissue between the cells is characteristic of epithelial tumors (Warren), and forms a diagnostic sign by which doubtful cases of carcinoma may be differentiated from alveolar sarcoma. In alveolar sarcoma the cells resemble those of carcinoma, but a close inspection reveals the fact that they are separated by a delicate connective-tissue framework or reticulum.

Papillomata.

Definition.—Papilloma (Lat. *Papilla,* a nipple; Gr. *ωμα,* a tumor).

A term employed to include corns, warts, horns, and certain nevi. A papilloma is a growth on the skin or mucous membrane, based upon or resembling a normal papilla.

The papillomata are epithelial growths occurring upon the cutaneous or mucous surfaces of the body, and are benign in character. These formations are generally considered as occupying a position midway between inflammatory swellings and true tumors, though some authors class them among the fibromata.

Morphologically, the papillomata belong to the class of tumors which arise from the epiblast and hypoblast,—the *epithelial group.* They are essentially composed of epithelial cells, but the framework or stroma is furnished by the underlying connective tissue, consequently they contain elements which are derived from the mesoblast. All epithelial tumors contain more or less connective tissue as stroma, and, strictly speaking, are therefore mixed tumors, though they are not generally classed as such. The new tissue developed from the germinal layers in combination is never uniform in quantity; one or the other element predominates, thus giving different histologic characters to

the neoplasms. This has made it somewhat difficult to determine to which group they belong, and has therefore caused the confusion in the classification.

Papillary formations are frequently found in tumors which do not belong to this class of new growths; hence Virchow, Rokitansky, and others have objected to classifying the papillomata as a distinct type of tumor. Virchow named them *"fibroma papillare."* Warren places them among the epithelial neoplasms. Sutton and Senn both class them as epiblastic and hypoblastic tumors.

In a majority of those tumors which can be classed, morphologically, as papillomata, the epithelial elements predominate and give character to the growth; while in others in which the fibrous elements are in excess this circumstance may be considered as an accidental feature, due to the close relationship existing between the epithelial structures and the underlying connective tissue.

In papilloma the essential part of the tumor is composed of epithelial cells, while the stroma or fibrous element is derived from the connective tissue, and contains the vascular supply. (Fig. 179.) The type of papilloma found upon the skin and in connection with the mucous membrane of the mouth, tongue, palate, etc., consists of a papilla, with a vascular connective-tissue base, covered with epithelial cells. They may be defined as excrescences from the epithelium of the skin and mucous surfaces. Their blood-supply varies greatly, but in certain forms associated with the mucous membrane it is often very considerable. Usually the epithelium covers a single papilla or villus, but occasionally it extends over several, forming smooth plaques.

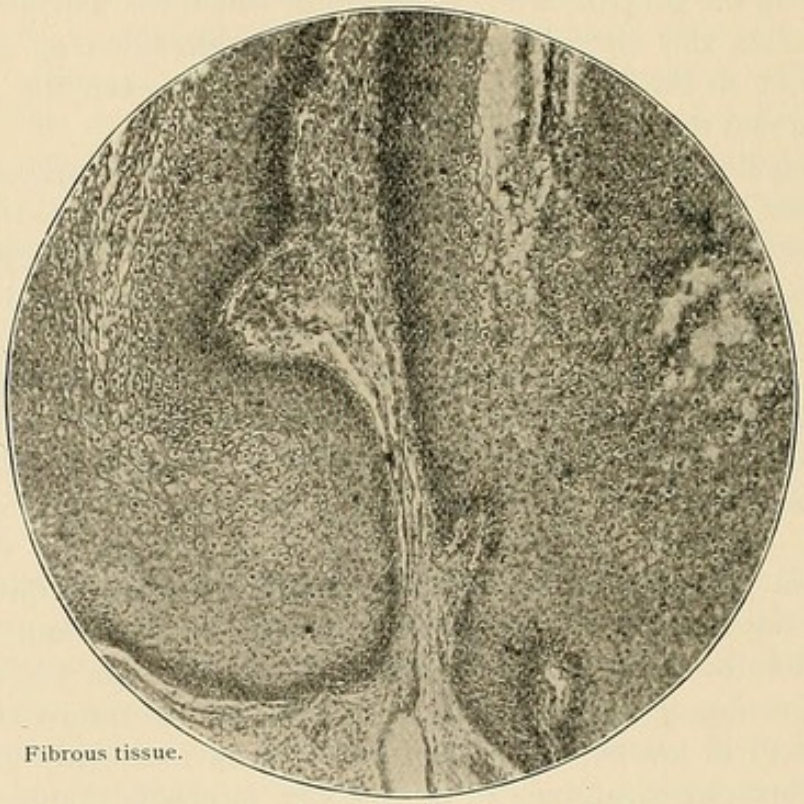
The papillomata appear in two forms,—the *hard* and the *soft*. The hard form is the variety commonly located upon the skin and mucous membrane, and generally designated by the term *Verruca* (an excrescence) or warts. The soft form is most frequently associated with the mucous membrane of the bladder, stomach, duodenum, and colon (Birch-Hirschfeld), and also of the uterus (Warren). Growths of this character are sometimes found springing from the meninges of the brain, and may grow into the venous sinuses (Klebs).

Sutton divides the papillomata into four species: warts, villous papillomata, intracystic warts, and psammomata. The difference in this classification from that generally followed is that of adding intracystic warts or papillomata, a rare variety sometimes found in mammary cysts, and of making separate species of those soft villous papillomata found in connection with the pelvis of the kidneys and the bladder, and of the epithelial bodies found in the membranes of the brain and the spinal cord, the psammomata.

Senn classes warts with the condylomata and molluscum contagiosum, which are inflammatory swellings of infective origin.

Hard papillomata of the skin, or skin warts, are the most common variety, and they are simply overgrown or hypertrophied papillæ. They may occur singly or in groups, and unless irritated are rarely painful. The most common locations are the hands, feet, face, scalp, neck, and genitals. (Hyde.) They may be congenital or acquired. They are exceedingly erratic in their development and disappearance, their growth being sometimes slow, sometimes rapid; they may persist for years, or disappear suddenly, without apparent cause. In form they may be sessile or pedunculated, flat or pointed, smooth or covered with secondary processes presenting a mulberry- or cauliflower-like

FIG. 179.



PAPILLOMA OF THE SKIN (WART). TRANSVERSE SECTION. × 60.

appearance. In color they may be like the skin or pigmented; in some cases mottled with black. In size they may vary from a pin-head to a walnut, or even attain the size of the closed fist. They sometimes bleed and ulcerate when irritated, and occasionally give off a very offensive odor.

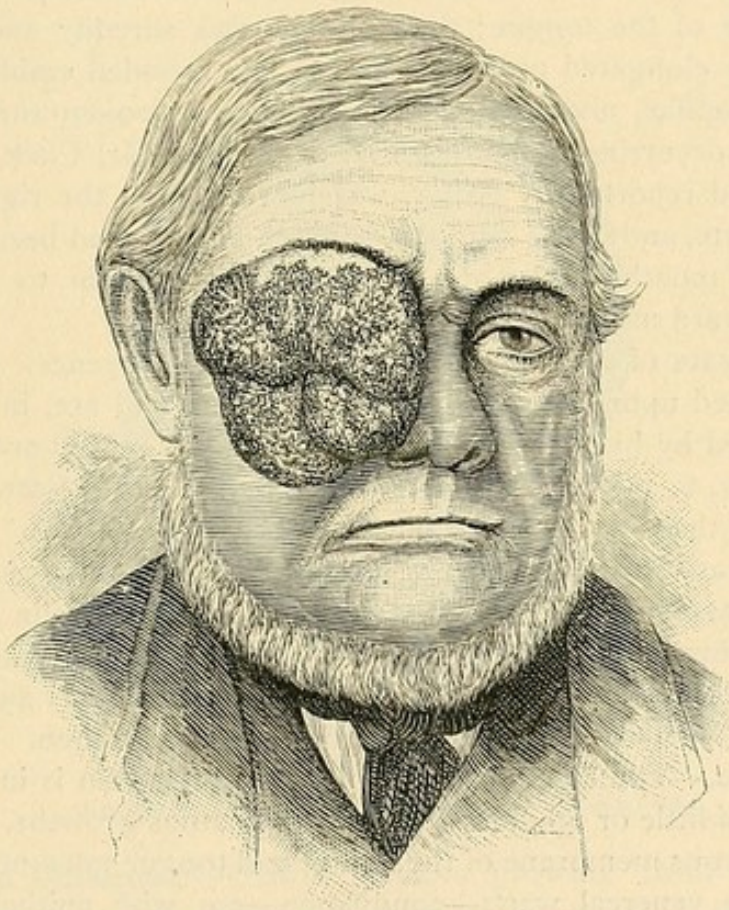
McCarthy reported a case (Sutton) of a man seventy-six years old having a tumor which sprang from the cheek and attained the size of half an orange, and completely covered the right eye. (Fig. 180.) Sutton reports a similar case of a woman forty years of age, with the tumor growing from the skin of the left temple, and another of a man thirty-six years of age, in whom the tumor was situated in the center of the pubic arch. This was as large as the closed fist, of the color of a

cock's comb, and emitted an abominable odor. The inguinal glands were enlarged upon both sides, but after the removal of the tumor the enlargement disappeared.

Senn thinks papillomata of such size are never individual growths, but that they are produced by a confluence of several tumors.

The hard form of papilloma is also found in connection with the mucous membrane of the lips, cheeks, tongue, hard and soft palates, uvula, pharynx, larynx, and nasal cavity, and also of the urethra, labia, vagina, cervix uteri, and bladder.

FIG. 180.



PAPILLOMA—WART—GROWING FROM THE SKIN OF THE CHEEK AND OBSCURING THE EYE. (McCarthy, after Sutton.)

Papilloma of the mucous membrane of the mouth bears a close resemblance to the ordinary seed wart, and is of more frequent occurrence than is commonly supposed; at least this is the observation of the writer, who has had a large field for clinical observation of the diseases of the mouth, and has found many opportunities for removing such growths. The most common locations are the lower lip, the soft palate, and tongue, and they appear in both the sessile and pedunculated forms. Papillomata of the larynx are the most common of all the tumors of this region, comprising about 75 per cent. (Bos-

worth.) Butlin says they "are among the more common of the innocent tumors which affect the tongue."

Another form of papilloma is occasionally observed upon the gums, the hard palate, and the tongue, in which the papillæ are greatly elongated, and the base of the tumor is of a dirty-white hue. Sir William Fergusson first described the disease in the London *Lancet,* September 6, 1862, the case occurring in the lower jaw of a man of eighty years.

Salter describes the same case, and says the tumor was "a curious white mass, consisting of coarse detached fibers; in fact, it was a mass of papillæ, many of them an inch long, and similar in shape to the filiform papillæ of the tongue; their surface was shreddy and broken; among these elongated processes were a few rounded eminences like fungiform papillæ, and these had a smooth, unbroken surface." A similar case occurring in the hospital practice of Mr. Cock, of Guy's Hospital, and reported by Salter, was located upon the right side of the hard palate, and of the size of a "split chestnut;" had been growing about eight months. Tumors of this character seem to possess a tendency toward malignancy.

Papillomata of the tongue are of frequent occurrence. They are usually located upon the dorsum of that organ, and are, in all probability, caused by hypertrophy of natural papillæ. They are not limited, however, to the papillary area, but may have their seat upon the under side of the tongue, where the surface is quite smooth. (Butlin.) They appear as "small white tufts," usually upon a sessile base.

Papillomata may appear at any time of life. Butlin mentions a case occurring in an infant ten months old. The disease occurs most frequently, however, in the later years of life, between sixty and seventy years of age (Watson), and oftener in men than in women.

Diagnosis.—The diagnosis of papilloma of the skin is in most instances one of little or no difficulty. Papillomatous growths, however, upon the mucous membrane of the mouth and tongue must not be confounded with venereal warts—condyloma—nor with epithelioma, as errors of this character might be productive of serious consequences. In children and youths, condyloma is the only affection with which it can be confounded, as epithelioma is not a disease of early life. In adults, especially men, there is danger that the more serious form of epithelial tumor may be diagnosed as papilloma. This would be an exceedingly grave error, endangering the life of the patient.

The diagnosis in the latter period of life becomes still more difficult, on account of the tendency at this period of the simple form of the disease being transformed into the malignant type.

Treatment.—The treatment of papillomata is ablation with the knife or scissors and thorough cauterization of the base, either with

stick nitrate of silver, chlorid of zinc, or the galvano-cautery. Various other methods are recommended for their removal, such as the application of Vienna paste, ligation, and galvano-puncture.

The treatment of the disease when occurring late in life should be more heroic. A considerable portion of the surrounding tissue should be removed with the tumor, as by such treatment the dangers of a recurrence, should the growth prove to possess malignant tendencies, would be much less than though a minimum amount of tissue had been scarified. Besides this, the surgeon would have the satisfaction of feeling that if he had made an error in his diagnosis, it was upon the side of the best interests of the patient.

FIG. 181.



CUTANEOUS HORN. MADAM DIMANCHE. (After Sutton.)

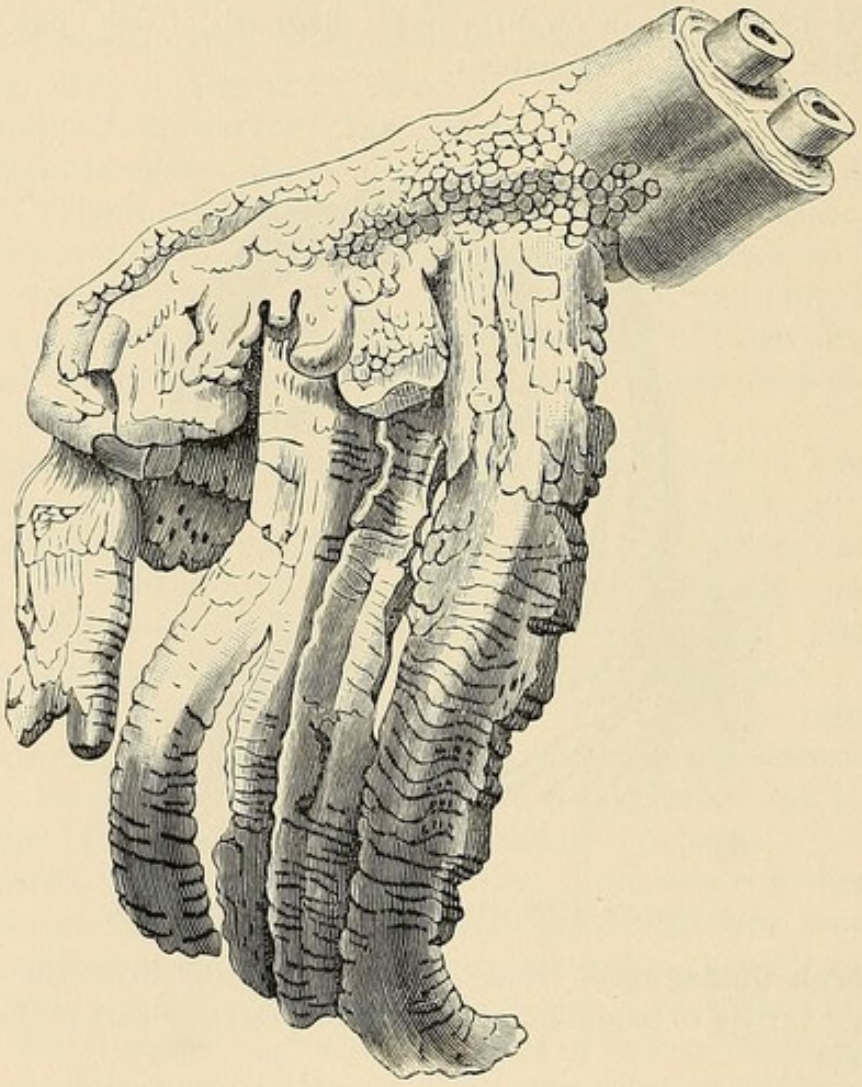
Cornu Cutaneum—(Horny tumors growing from the skin).—Another variety of papillomata, though one rarely seen, are the *cutaneous horns.*

In this form of papilloma the tumor is composed almost exclusively of epithelial cells from the horny layer of the skin, which ordinarily, after serving their purpose, are desquamated, but which for some reason remain attached to the tumor-matrix, gradually increasing until they form projections or horns of various lengths, from ½ inch to 12 or more inches. It is possible that the matrix of the cutaneous horn furnishes a cement substance (Senn) which fixes the epithelial cells, and thus prevents their removal by desquamation.

These tumors have their origin in sebaceous glands, warts, cicatri-

cial tissue, and nails. They are most frequently located upon the scalp, temple, forehead, eyelid, nose, lip, cheek, and shoulder, and upon various other portions of the body. One of the most remarkable cases of *cutaneous horns*—sebaceous—is that of Madam Dimanche, who had a long horn growing from the forehead, and another smaller one from the right cheek (Sutton); a wax cast of whose face is preserved in the

FIG. 182.



HORNS GROWING FROM THE SCAR OF A BURN. (Cruvielhier, after Sutton.)

museum of the Royal College of Surgeons, England. (Fig. 181.) A very remarkable *cicatricial horn* arising from the palmar surface of the hand following a severe burn, and which reached such an enormous size that amputation became necessary, was reported by Cruvielhier. (Fig. 182.) Mr. Edwards, of London, also reported a similar one originating in a burn which occurred sixty-five years previously (Sutton). The tumors in both of the latter cases were multiple. Wart

horns are prone to degeneration at their base, and in elderly persons they may not infrequently terminate in epithelioma.

To determine the origin of cutaneous horns, Sutton divides them longitudinally. The existence of a cyst at the base proves them to be of sebaceous origin; its absence, of warty origin. The *nail horns* need no description in these pages, as they are only met with in connection with the fingers and toes.

Treatment.—Cutaneous horns, as a rule, are not very firmly attached to the skin, and are usually easily detached with the fingers. When too firmly adherent for removal by this means, they can be excised. Occasionally amputation of the member upon which it grows may be necessary. After removal of the growth, the base should be thoroughly cauterized to destroy any remnants of the tumor which might remain, and thus prevent a recurrence. When epithelioma attacks the base of the horn, early and heroic treatment is demanded.

REVIEW.

CHAPTER XLIII.

Name the tumors of the Epithelial group most often associated with the face, mouth, and jaws.

Name those of the Connective-tissue group.

Name those of the Composite group.

What are those tumors called which result from the retention of normal secretions?

What is the origin of the neoplasms comprised in the epithelial group?

What form of cells predominate in these tumors?

Describe the functions of the epithelium.

What three glands do not have direct communication with free surfaces of the body?

What does this direct communication with the surface of the body of the other glands subject them to?

Of what two kinds of tissue are epithelial tumors composed?

How do these tissues indicate the benign or malignant character of the neoplasm?

What is the general plan of construction of the epithelial tumors?

Which tumors most nearly resemble glandular structures?

Which have the least resemblance?

What is the character of the arrangement of the cells in the epitheliomata and carcinomata?

How do the adenomata differ from this?

What are the stages of cell differentiation which indicate the innocent or malignant character of these neoplasms?

How are the epithelial tumors developed?

What are the conditions of cell-multiplication which result in the formation of these various forms of epithelial tumors?

What is the arrangement of the cells and the stroma in carcinoma?

How may carcinoma be distinguished from alveolar sarcoma?

Give the definition of Papilloma.

Where are these tumors found, and what is their character?

How do some authorities class the papillomata?

Where do they belong, morphologically?

Of what are they composed?

What are some of the conditions which have led to confusion in the classification of the epithelial tumors?

Are papillary formations found in other locations than upon the skin and mucous membrane?

What cell elements predominate in the true papillomata?

Of what does the typical papilloma of the mucous membrane of the mouth, tongue, palate, etc., consist?

In what *forms* do the papillomata appear?

Where are the *hard* forms commonly located?

What term is applied to them?

Where is the *soft* form generally located?

What is Sutton's classification of the papillomata?

How does Senn class the warts?

Which are the most common forms of papilloma, the *hard* or the *soft* varieties?

What is the most common location of warts?

Describe their peculiar characteristics.

Describe their form, color, etc.

Upon what portions of the mucous membrane are they found?

What is their appearance?

Where are they most commonly located?

In what other locations connected with the mouth are they found?

What other form of papilloma is occasionally found upon the gums, hard palate, and tongue?

What seems to be the tendency of tumors of this type?

Are papillomata of the tongue frequent?

What are the most common locations?

What is the appearance?

At what age do papillomata most frequently occur?

In which sex are they most common?

Is there difficulty in the diagnosis?

What affections of the mucous membrane should not be confounded with papilloma?

When is the diagnosis most difficult?

Describe the usual treatment.

What should be the character of the treatment in cases occurring late in life?

What are the cornu cutaneum?

Describe these growths.

Where do these tumors have their origin?

What is the most common location?

What is the tendency of wart horns?

How may their origin be determined?

What is the treatment usually adopted?

When epithelioma attacks the base, what should be the treatment?

CHAPTER XLIV.

EPITHELIAL TUMORS (Continued).

Adenomata.

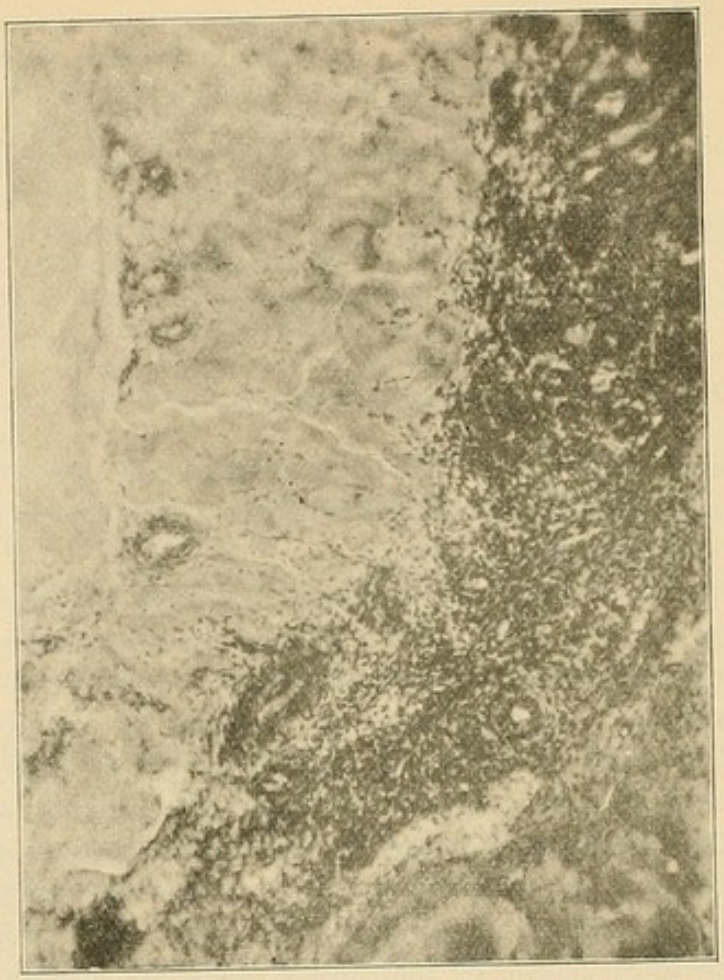
Definition.—Adenoma (Gr. ἀδήν, gland, and ωμα, tumor). An adenoma is a tumor that has developed from a gland, or is constructed after the type of a secreting gland.

An adenoma is a true neoplasm, and should not be confounded with retention cysts, or with glands enlarged by overgrowth, overwork, or chronic inflammation, for these conditions are in no sense new formations. Although we might on first thought be inclined from the definition of the term adenoma, to class all glandular enlargements in which there was an abnormal multiplication of the glandular elements, as adenomata, yet upon a consideration of the evident physiologic impotence of new growths to produce a normal gland-secretion (Ziegler), and the lack of anatomic relations with surrounding tissues, such a classification would be manifestly incorrect. Senn says, "In the strictest etiologic and pathologic sense, the term should be limited to glandular tumors containing adenomatous tissue produced from a tumor-matrix independently of the pre-existing glandular tissue."

The adenomata are less distinctly defined in structure than most of the other neoplasms. The transitions between glandular hyperplasias and glandular tumors are manifold. The former are many times distinguished with difficulty from certain inflammatory swellings, while the latter not infrequently present transitional forms which are almost identical with epithelioma. The anatomic and histologic structures of these growths are sometimes most difficult to define microscopically, on account not only of the transitional changes just referred to, but also because of other changes occurring within them and in the surrounding tissues, such as hemorrhage, edema, and various degenerative metamorphoses, like the fatty mucoid, colloid, fibroid, and hyaline (Figs. 183, 184); the formation of papillary and villous growths, and the development of cysts.

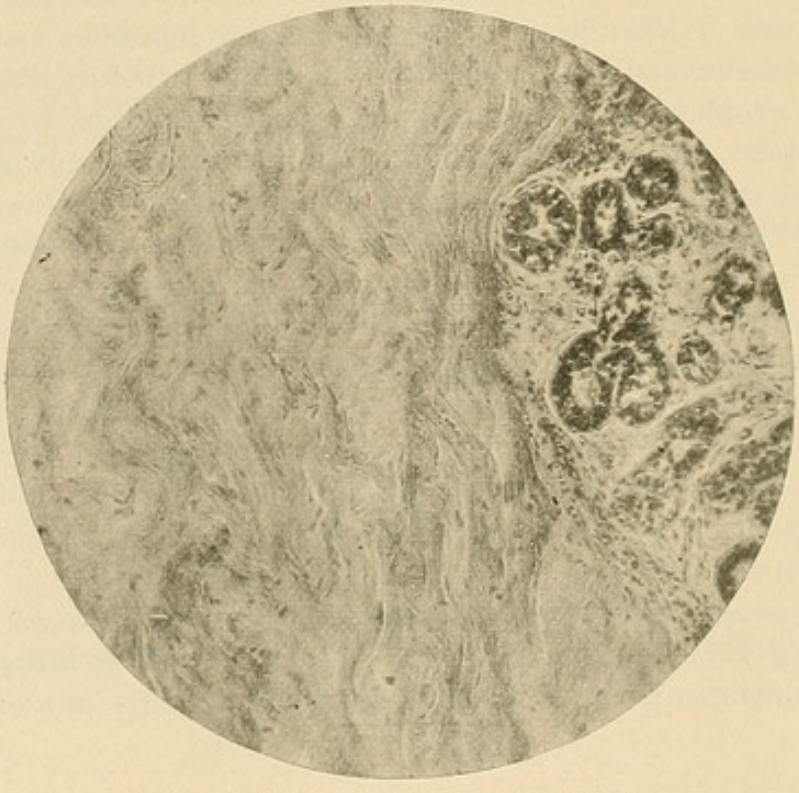
Adenomata of the intestinal tract are very prone to present a malignant degeneration, or at least have the properties of malignant tumors (Councilman), viz: of infecting surrounding parts, and producing

FIG. 183.



HYALINE DEGENERATION FROM INFLAMMATION OF LYMPHATIC GLAND OF THE NECK.

FIG. 184.



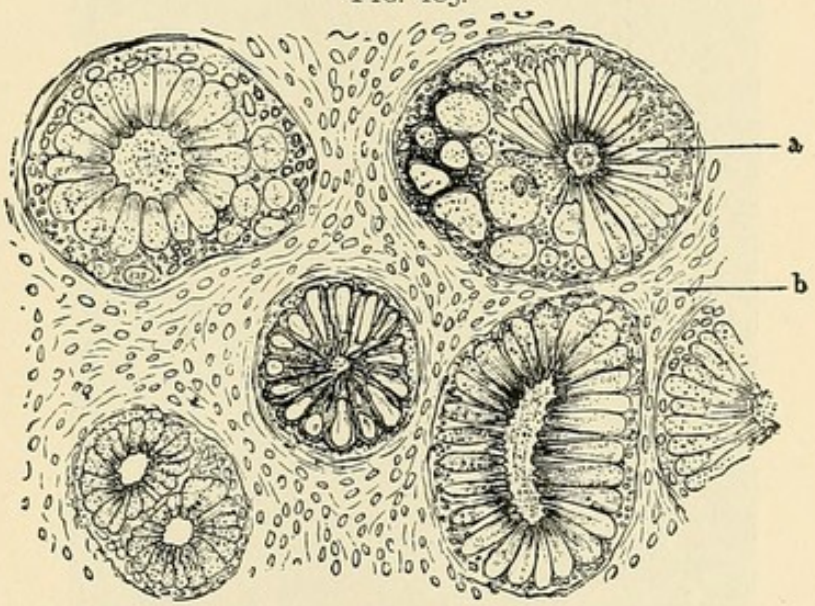
FIBROID DEGENERATION—MAMMÆ. × 50.

metastases. When the disease is located in the stomach or intestines, the several coats are successively attacked, often resulting in perforation. Ziegler applied the term *adenoma destruens* to this variety of adenomata.

The *true* adenomata can generally be distinguished from the glandular hyperplasias by certain well-marked signs of consistence, color, and structure, as compared with the surrounding tissue. These tumors are of themselves benign growths, but the tendency to pass into cancerous formation must be constantly borne in mind. Pure adenoma is a rare affection.

The structural peculiarity of an adenoma is the presence of epithelial cell-elements which are arranged after the order of secreting glands, and supported by a connective-tissue stroma. (Fig. 185.) In

FIG. 185.



ADENOMA (POLYPUS) OF THE RECTUM, SHOWING THE GLANDS OF THE TUMOR. × 350.
(After J. D. Hamilton.)

a, gland lined by columnar epithelium; *b*, stroma of the tumor.

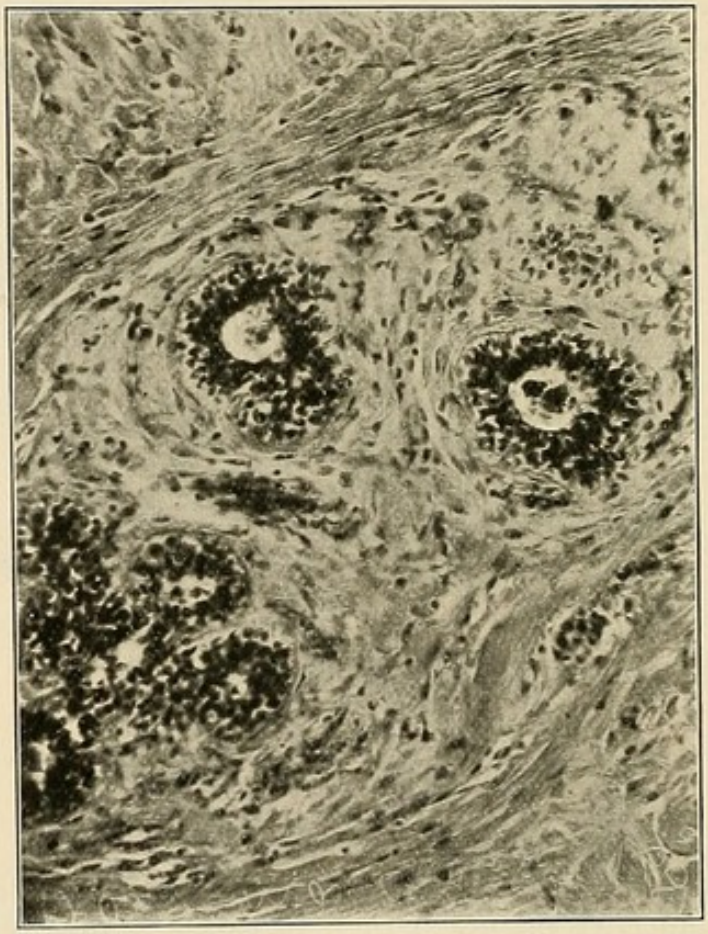
some forms of adenoma the epithelial element predominates, while in others the connective tissue is largely in excess. Most of these neoplasms are mixed tumors, having other elements,—fibrous, myxomatous, sarcomatous, carcinomatous, etc.,—in combination with the glandular structure. Tumors formed in this manner are designated as fibro-adenoma, myxo-adenoma, etc. Fig. 186 shows the histologic structure of fibro-adenoma.

Adenomata occur as congenital tumors; as developments of early life, and they occasionally occur during any period of adult life. They are found in such tissues as the breast, the skin, the mucous membrane, the kidneys, the liver, the thyroid and parotid glands. Although adenoma is not a common form of tumor, it has a wide distribution, being

found in nearly all portions of the body which have had their origin in the epiblastic and hypoblastic layers of the germinal disk. Inasmuch as adenomata are found in all of the glandular structures of the body, it should be noticed, as a marked peculiarity, that the cells which compose these neoplasms closely resemble the individual structure of the gland or duct with which they are associated.

The adenomata usually appear as knot-like growths in the substance of glands, in glandular epithelium, or epidermic tissues (Ziegler); their most common seat being such glands as the mamma, the

FIG. 186.



FIBRO-ADENOMA. × 50.

ovary, the parotid, the thyroid, and the liver; also in the glands of the mucous membrane of the rectum, the intestines, and the uterus (Sutton). They may be sessile or pedunculated, the form being governed by their character and location.

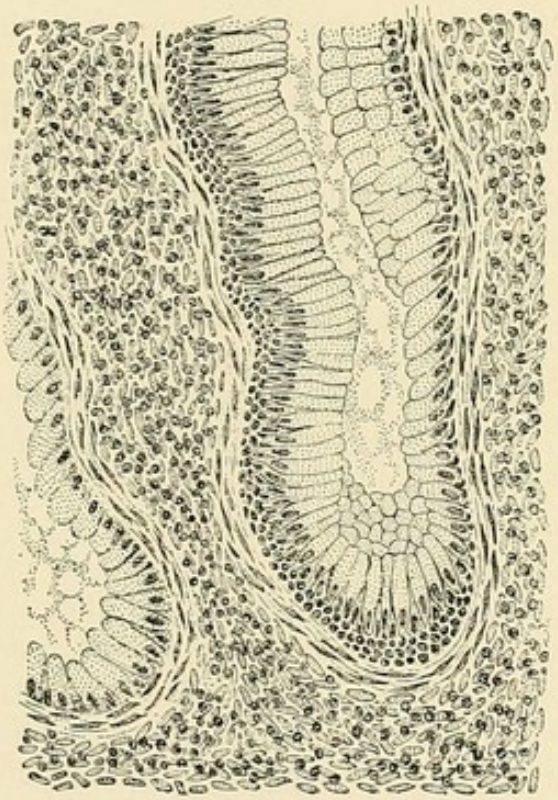
The development of adenoma is not necessarily confined to pre-existing glandular structures, as they may be formed in locations where glands do not normally exist. The explanation of the origin of the tumors in such locations is to be found in the displacement and isolation of embryonic cells during fetal life, or in the formation of a "tumor

matrix" of embryonic cells within a supernumerary or accessory gland. (Senn.)

The glandular tumors vary considerably in size, ranging from a pea-like nodule to as large as a man's head, in locations like the female breast; while cysto-adenoma of the ovaries weighing thirty to forty pounds are not uncommon.

The adenomata are developed in two forms: the *acinous* and the *tubular*. Acinous adenoma simulates the structure of the conglomerate glands. The stroma varies in amount. If the tumor is hard, the

FIG. 187.



SECTION OF AN ADENOMA FROM A CHILD'S RECTUM. HIGHLY MAGNIFIED. (After Sutton.)

stroma is abundant; if soft, it is scanty. The blood-vessels are located in the stroma, and each lobule or tubule is supplied from these with a capillary net-work of vessels, while the lobules or tubules are lined with flat epithelial cells. These tumors are usually associated with the conglomerate glands.

Tubular adenoma closely resembles the simple tubular glands. The epithelial cells are arranged in single or stratified layers within the tubule, while an open space is left in the center. This form springs from mucous membranes which have glands of the tubular form. It is most commonly associated with glands of this construction located in the intestinal canal, and especially in the rectum. (Fig. 187.)

Causes.—The causes may be divided into predisposing and exciting.

The *predisposing* or *essential* cause is the presence of a tumor-matrix of embryonic cells which have been misplaced, and isolated during fetal development. The tumor-matrix, however, may remain indefinitely in a quiescent and undeveloped state, until stimulated to activity by some abnormal condition of its surroundings, or by a direct injury.

The *exciting* or *active* causes of their development are traumatic injuries, irritation of various forms, and acute or chronic inflammations. Organs which are the seat of periodic congestions, like the mamma, the ovary, the uterus, and the prostate gland, are the most common location of these tumors; while in the mucous membranes which are most liable to catarrhal affections, like the nasal passages and the rectum, these neoplasms are of common occurrence.

Prognosis.—The prognosis of true adenoma depends principally upon the location in which it is developed. It is benign in its character, and when completely removed does not return. It differs from the malignant tumors in that it does not infect the lymph-glands in its neighborhood, neither does it cause metastatic deposits. It frequently attains an enormous size, and when located in the ovaries, or in the thyroid glands, sometimes causes a fatal termination. Death in these cases is caused by mechanical complications, usually of pressure upon important viscera or vital organs. Adenoma is inclined to undergo degenerative changes, particularly hyaline, colloid, cystic, and fatty degeneration. It has also a tendency in the later years of life to take on malignant transformations.

Adenoma of the Skin.—In adenomata of the skin, the tumors are found associated with the *sweat* or *sudoriparous glands* and the *sebaceous glands.* (Fig. 188.) Adenoma of the sweat-glands is seen in various parts of the body, but most frequently upon the face. These tumors may vary in size from a pea to a walnut, or may even be as large as the fist. In appearance they are commonly small, soft tumors, with nodular surface and a dirty, grayish-white color. Generally they are circumscribed, but occasionally they form diffuse or ill-defined growths. The skin over them is at first but little changed, but later it is often ulcerated (Wagner), and they have been mistaken for angeiomata. They are slow of growth, and are a somewhat rare form of neoplasm. A cut section of such a growth discloses coils of dilated ducts, from which can be pressed degenerated epithelium (Warren).

The growth of the tumor seems in some instances to take place from the deeper portion of the tubule of existing sweat-glands, while in others it seems to be quite independent of any glandular origin. Thierfelder observed a case in which the tumor had its origin in the diploë of the cranial bones, but communicated with the skin. This connection would point to the probability that it originated in the skin.

Adenoma of the sebaceous glands appears upon the face in the form of papules, which are usually of congenital origin (Warren), and form little, roundish, convex tumors, the size varying from that of a pin-head to that of a pea, often bright crimson in color. The small tumors assume the form of a sebaceous gland, but in the larger ones the glandular tubule (Senn) forms a convoluted mass. (Fig. 189.)

FIG. 188.



HORIZONTAL SECTION OF HUMAN SCALP, SHOWING GLANDULAR STRUCTURE AND HAIR. × 50. (T. Charters White.)

The nose is a favorite seat for the development of this form of tumor. Tumors found in this location were formerly called lipomata, but they are now known in many instances (Sutton) to be sebaceous adenomata. This form of adenoma is quite prone to ulcerate, and occasionally to calcify (Eve).

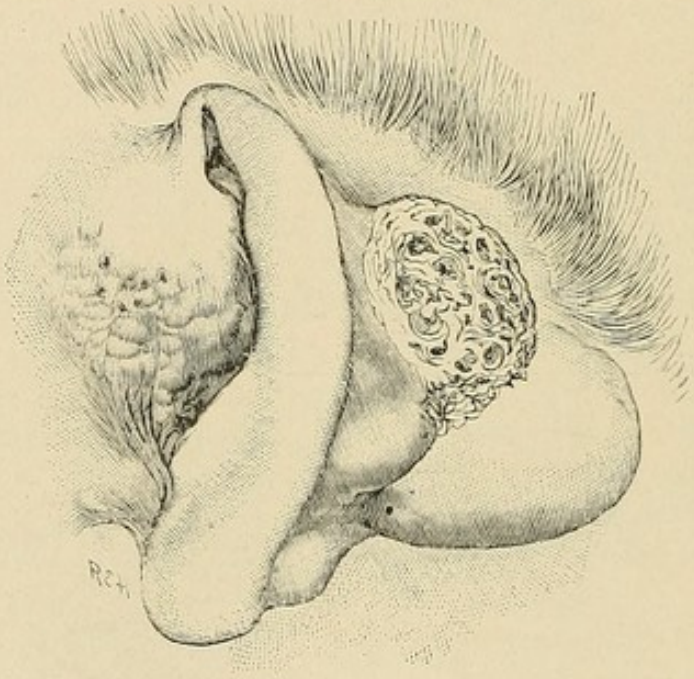
Diagnosis.—In diagnosing adenoma of the skin, there is as a rule very little difficulty, for the especial features of the disease are well defined, and the growth always occurs in regions occupied by sudoripar-

ous or sebaceous glands. The diseases which may be mistaken for adenoma of the skin are epithelioma, molluscum epitheliale, and lipoma.

Prognosis.—The prognosis in uncomplicated adenoma of the skin is always favorable; recurrence after extirpation is rare.

Treatment.—The treatment is surgical, and consists of the removal of the tumor. This is accomplished in most cases with ease, as these tumors are usually encapsulated, and are readily shelled out or enucleated.

FIG. 189.



LARGE SEBACEOUS ADENOMA INVOLVING THE PINNA. (After Sutton.)

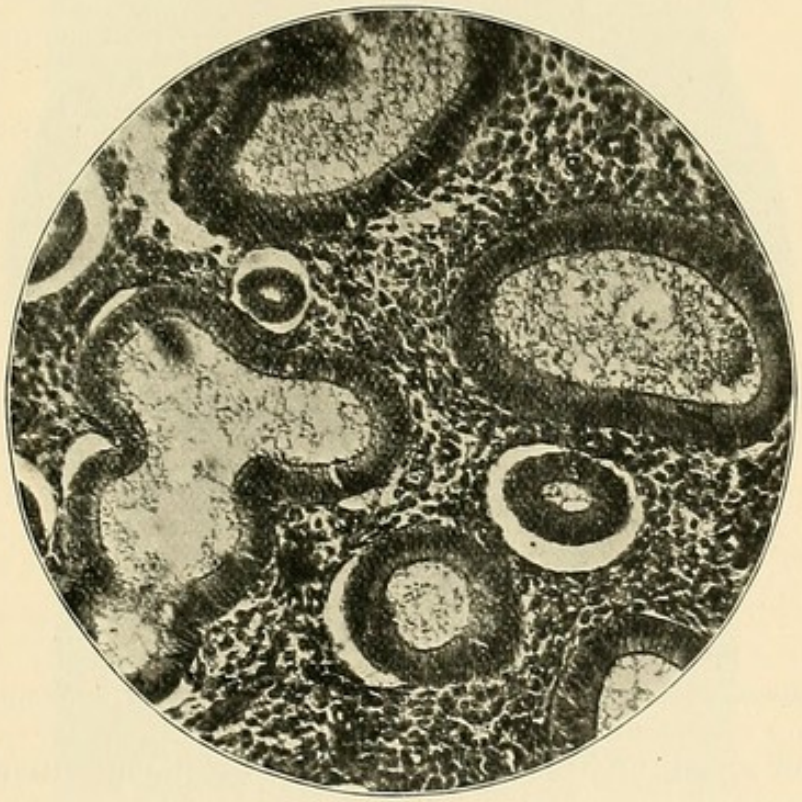
Adenoma of the Mucous Membrane.—Adenoma of the mucous membrane appears in two forms. One is confined to the *mucosa* or mucous membrane proper; the other involves the *sub-mucosa*, or the submucous connective tissue, and from this it extends to adjacent tissues and organs.

The first variety is expressed in the form of polypoid growths, the structure of which closely simulates the glandular arrangement of the membrane from which it springs, the difference being that the glandular elements are larger, more numerous, and less regular (Ziegler) than in the normal tissue. (Fig. 190.) This form Ziegler classes as *glandular hyperplasias*. The second variety he designates as *adenoma destruens*. This latter form also simulates the glandular structure of the mucous membrane, but differs from the other variety in its mode of development and its tendencies to involve surrounding tissues and cause metastases.

The first variety is frequently associated with the Schneiderian mucous membrane in the form of pedunculated growths in the nasal

passages and vault of the pharynx. These growths may be in the form of mucous or gelatinous polypi, or they may be true adenomata. (Fig. 191.) The majority of polypi are not true adenoid growths, but inflammatory enlargements or hyperplasias of the glandular elements of the mucous membrane. The adenoid vegetations described by Meyer, Cohn, and others, are not of very frequent occurrence, but a condition of hyperplasia of the glandular elements of the pharyngeal tonsil, which is expressed in nodular enlargements or polypoid excrescences, is quite common. The true adenoid growths sometimes attain to considerable size. In the nasal passages they have been known to fill these passages and the adjacent sinuses, causing distention and facial deformity.

FIG. 190.



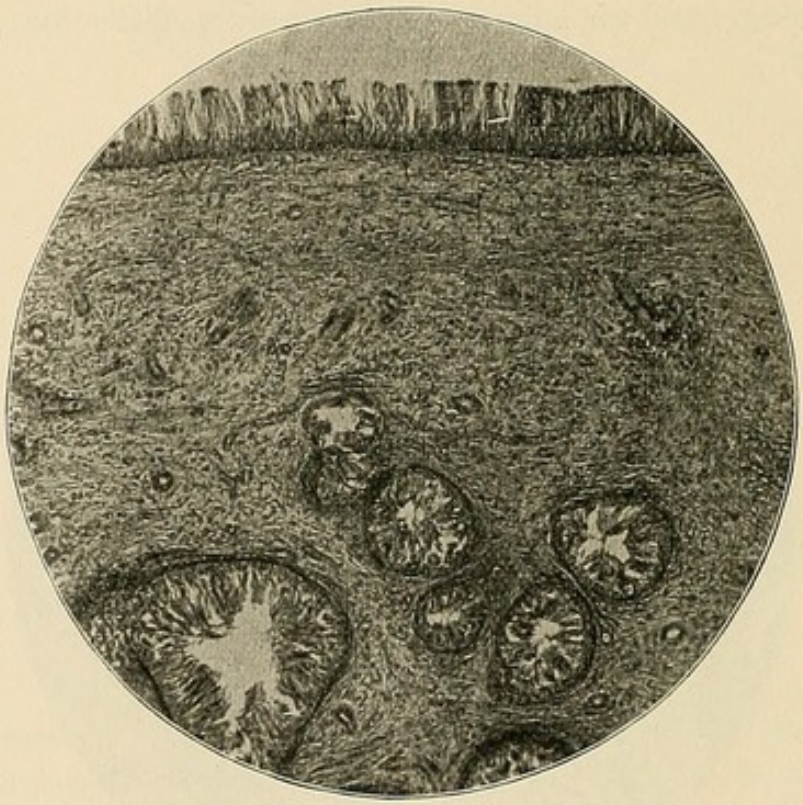
ADENOMA—BENIGN. × 50.

Cystic degeneration of the mucous glands, of the lining membrane of the nasal passages, and of the antrum of Highmore, is occasionally seen. This form of the disease, however, is more common in the antrum than in the nose. Occasionally the glands are enlarged and multiplied as in adenoma, resulting in *adeno-myxoma*. The adenoid growths of the antrum not infrequently manifest malignant tendencies, ending in *adeno-carcinoma*. The malignancy of this form of tumor is equal to that of the most malignant type of carcinoma, and it is the opinion of the writer that many of the malignant growths of the antrum that are classed as carcinoma and epithelioma have their origin in

adenoid growths, associated with the lining mucous membrane of this sinus.

The destructive adenoma ("adenoma destruens") is a soft, marrowy tumor, taking the form of capillary or fungous outgrowths (Ziegler), or of an extensive thickening, and a slightly raised surface of the affected portion of the mucous membrane. The new-formed tissue shows a marked tendency toward degeneration in the formation of ulcerated surfaces. The ulcers appear with raised, "rampart-like"

FIG. 191.



NASAL POLYPUS—MYXOMATOUS TISSUE—SHOWING CILIATED EPITHELIAL CELLS. × 50.

edges, and a soft, infiltrated base, and the surrounding tissue is frequently studded with nodular growths. This form is most frequently seen in the stomach and intestinal mucous membrane.

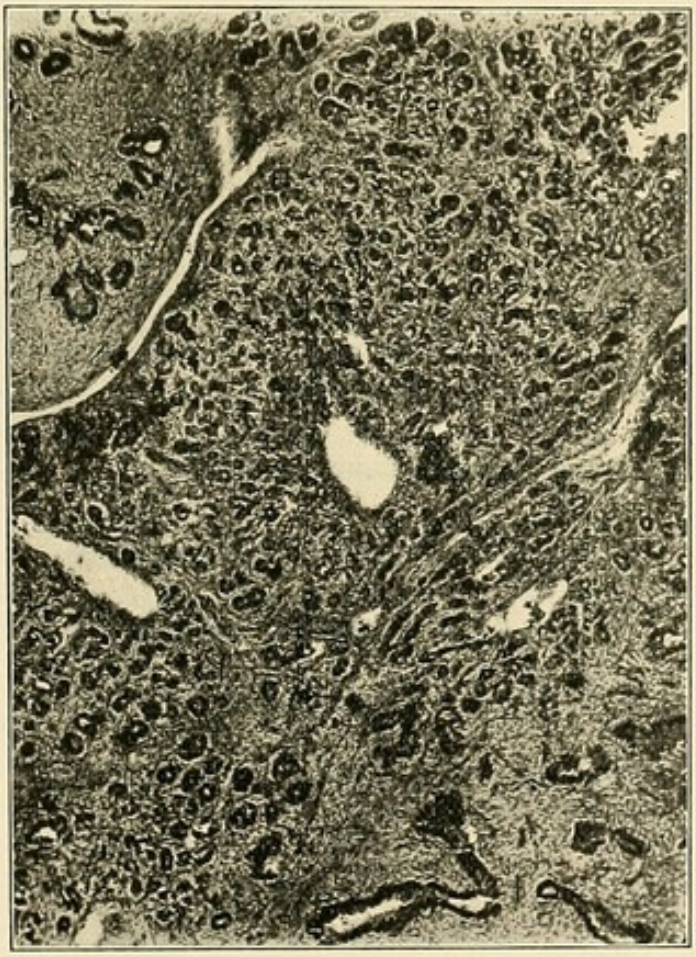
Adenoma of the buccal mucous membrane rarely attains dimensions larger than a pea or a small bean, though occasionally it may reach a much larger size. They are generally true adenomata, and are most often seen upon the lower lip and cheeks,—rarely upon the upper lip. These tumors are of slow growth, and several years may elapse before they attain a size sufficient to cause inconvenience.

Diagnosis and Symptoms.—Adenoma of the buccal mucous membrane appears as single, smooth or nodulated enlargements beneath the mucous membrane, firm and non-elastic to the touch, usually sessile, and but slightly adherent to the overlying membrane or to the tis-

sues beneath, and generally encapsulated. They are not painful unless ulceration or cancerous degeneration takes place. The only prominent symptom in the benign form of the disease is one of inconvenience, occasioned by its size or location, or both.

Adenoma of the Palate.—Adenoma is more frequent in the palate than in other portions of the oral cavity. The growths appear as smooth or nodulated tumors beneath the mucous membrane; in other

FIG. 192.



ADENOMA—PURE—OF PALATE. × 40.

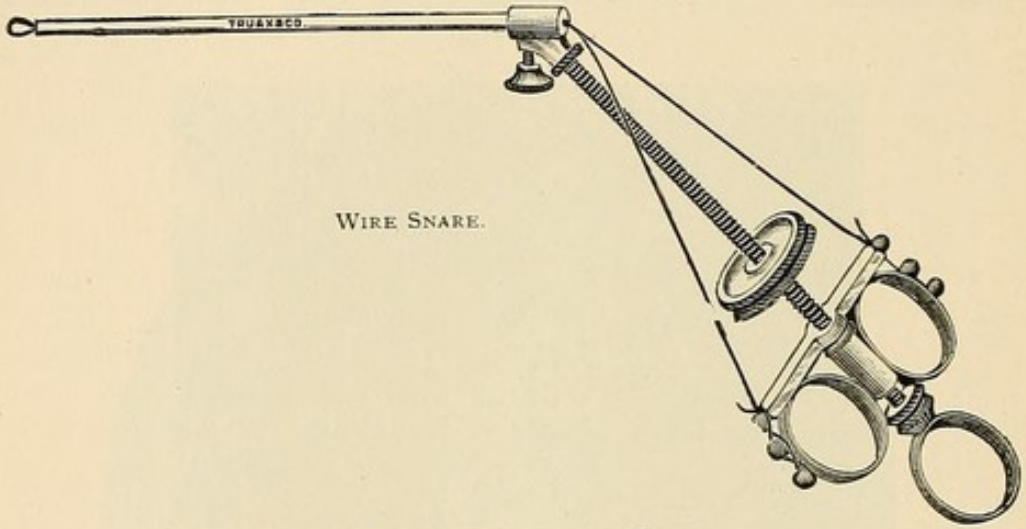
respects they are like those found upon the lips and cheeks. They are located upon either side of the median line, and when they are of large size cause, as a first evidence of their presence, a slight nasal twang of the voice (Cohen). Later there is a gradually increasing mechanical difficulty in swallowing, at first of liquids only; later of all ingesta. Fig. 192 shows the histologic structure of a pure adenoma of the palate.

Diagnosis and Symptoms.—The development of neoplasms and inflammatory swellings in the *velum palati* gives rise to certain definite symptoms, viz: dysphasia, cough, difficulty of breathing, changes in the resonance of the voice, which acquires a peculiar nasal twang due to

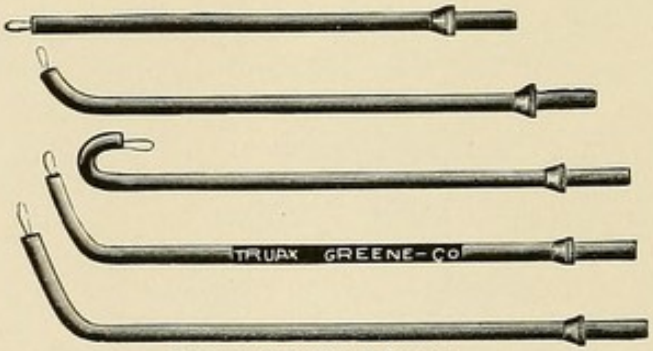
the imperfect occlusion of the soft palate with the vault of the pharynx. Pain is rarely associated with the growth of these tumors, or with the dysphasia incident to their presence.

Prognosis.—The prognosis of adenoma of the oral mucous membrane and of the palate is usually favorable. In the destructive variety of the affection the prognosis would be unfavorable, as metastases and

FIG. 193.



WIRE SNARE.



POINTS FOR WIRE SNARE.

FIG. 194.



WIRE ÉCRASEUR.

recurrence are likely to follow. In the benign form of the disease, operation gives good results, as the growths can be removed in their entirety.

Treatment.—The treatment of adenoma of the palate consists of excision, evulsion, or constriction. The character of the operation should be governed by the pathologic tendencies of the growth, its shape, and its accessibility. Polypi of the nasal passages may be removed by the wire snare or écraseur. (Figs. 193, 194.) The ma-

jority of these tumors are encapsulated, and are easily removed by a single or double incision through the mucous membrane. A single incision only is required for growths of small size; the double incisions which are made at right angles to each other, are necessary for the removal of the larger growths. The existing adhesions are then torn or dissected away, the tumor turned out of its capsule, and the wound closed with sutures. In the region of the palate hemorrhage from the posterior palatine artery may sometimes prove troublesome, but it can be controlled by a tampon, ice, the various hemostatics, or by the electro-thermal cautery. The after-treatment should consist of frequent irrigation of the mouth with antiseptic solutions.

Adenoma of the Tongue is an exceedingly rare affection. Butlin in his work on "Diseases of the Tongue" considers it "so rare, that no general account can be written of it." Of the four cases which he mentions, two of them occurred in persons near middle life, the third in a girl of sixteen, and the fourth in a new-born babe which lived only sixteen hours; death being due to pressure of the growth upon the larynx, the tumor having developed in the base of the tongue. In one the tumor was situated well back upon the dorsum of the tongue; in another it was located upon the under side of the tip of the tongue, but in the case of the girl the location of the growth was not mentioned.

Rosenberg thinks adenoma of the tongue is very rare. According to Thaon and Larabie, these tumors are really mixed epitheliomas. The microscope reveals a connective-tissue net-work or stroma which embraces the epithelial infiltration. In the early history of these growths the epithelial elements are in excess; later the connective-tissue elements predominate, which in all probability accounts for the relative innocence of these neoplasms. Larabie advises, on account of the possibilities of these growths developing true carcinomatous elements, early and free excision of the tumor.

Diagnosis and Symptoms.—There are no diagnostic signs or symptoms which are distinctive of adenoma of the tongue, and no means whereby an exact diagnosis can be made, except by removal of a portion of the tumor and subjecting it to a microscopic examination. Adenoma, fibroma, and lipoma are not readily distinguished from one another while *in situ*, and may therefore be easily mistaken one for the other.

The symptoms which are common to enlarged glands and the various tumors which may be located upon the dorsum of the tongue are a sensation of a foreign body in the throat, irritation, cough, dyspnea, dysphagia, fatigue of the voice, and painful deglutition in those cases associated with inflammatory conditions of the glands or of the neoplasms.

Prognosis.—The prognosis of adenoma of the tongue is good.

Early extirpation is demanded as a precaution against the development of malignant disease, and to relieve the local symptoms.

Treatment.—The treatment consists of enucleation or excision of the tumor. The removal of a sufficient amount of the surrounding tissue to insure a complete extirpation should always be practiced in those tumors which arouse suspicion as to their malignant character. Adenoid tumors of the tongue are usually found situated well back upon the dorsum of the organ, which makes the operation somewhat difficult to perform. The removal of these growths is not essentially different from the operation in other locations of the oral cavity, as they are always found encapsulated and are easily enucleated. Before commencing the operation, the tongue should be secured by passing a strong ligature through the tip, the tongue being drawn well forward and held in that position during the operation. The jaws should be held apart by means of a mouth gag. Hemorrhage is sometimes troublesome from venous and capillary oozing, but this can usually be controlled by pressure over the bleeding surfaces with a piece of gauze and the finger. When the loss of tissue has not been too great to permit of this, the edges of the wound should be brought together with sutures. Antiseptic treatment of the wound and of the mouth should be carefully followed to prevent suppuration and secondary septic infection.

Adenoma of the Salivary Glands.—Adenoma of the salivary glands is not a common affection, but when it does exist it is associated with the parotid gland. The submaxillary and the sublingual glands seem to be quite exempt from this form of tumor. Weber is of the opinion that even the parotid gland is very rarely the seat of true adenoma. Billroth does not think that true adenoma ever exists in the parotid gland, but that all adenoid growths located in this gland are mixed tumors, adeno-cystoma, adeno-chondroma, adeno-carcinoma, etc. Pure adenoma, however, has been found in the parotid gland. Warren describes a perfectly formed adenoma in the parotid gland of the size of a hen's egg, and quite soft in structure. In this respect it differed from the ordinary tumors found in this gland.

Parotid adenoma most frequently occurs in youth and early adult life,—between the fifteenth and thirtieth years of age (Sutton).

Diagnosis and Symptoms.—Adenoma of the parotid occurs as distinctly encapsulated tumors. They are not painful, may arise in any part of the gland, are of slow growth, and rarely exceed a pigeon's egg in size. Positive diagnosis is difficult to make except by a microscopic examination after the operation.

Cystic degeneration is common, and papillary excrescences often develop upon the cyst-walls and project into the tumor. Microscopic examination of these excrescences shows them to be composed of the

same elements as the secreting tissue of the gland. These tumors have a strong resemblance to the adenomata found in the thyroid gland. Usually they are encapsulated, and can be readily enucleated with little or no permanent damage to the gland.

Prognosis.—The prognosis is favorable in uncomplicated adenoma of the salivary glands. In adenoma with malignant tendencies it would be very unfavorable. In the latter case, early and complete extirpation of the entire gland is the only means of saving the life of the patient.

Treatment.—In operating for the removal of adenoma of the parotid, the incisions should be made with especial reference to the preservation of Stenson's duct without mutilation, and with due regard to the location of the branches of the facial nerve. The posterior edge of the gland lies in close relation to the external carotid, superficial temporal, transverse facial, internal maxillary, and internal carotid arteries, the external jugular vein and its anastomosing submaxillary branch, and the internal jugular vein.

In cutting down upon the tumor, a thin portion of the gland may be incised, and as a result salivary fistula may follow the operation. Usually, however, if Stenson's duct has not been injured, the discharge of saliva through the wound is only a temporary matter.

The extirpation of the gland for the removal of malignant growths may require the ligation of the external carotid artery and of the external jugular vein.

REVIEW.

CHAPTER XLIV.

Give the definition of adenoma.

What is an adenoma?

With what conditions should it not be confounded?

What characteristics mark the difference between adenoma and these conditions?

What is the distinction made by Senn?

State the conditions which make it difficult to distinguish glandular hyperplasias from glandular tumors.

What is the tendency of adenoma of the intestinal tract?

What term does Ziegler apply to this form of the disease?

How may *true* adenoma be distinguished from glandular hyperplasias?

What is the character of a true adenoma?

Is this form common or rare?

What is the structural peculiarity of an adenoma?

What is the character of most of these neoplasms?

Name the most frequent tumor-elements with which they are combined.

At what periods of life do the adenomata occur?

In what tissues are they located?

From which embryonic layers do they originate?

What is a marked peculiarity in the structure of adenomata of the various glands of the body?

How do the adenomata usually appear?

In what glands are they most frequently found?

What is their form?

Are the adenomata confined to pre-existing glandular structures?

If not, what is the explanation of their origin?

What is the variation in the size of these tumors?

State the number and names of the forms of adenomata.

Describe the acinous form.

With what form of glands are they usually associated?

Describe the tubular form.

With what form of glands are they most often associated?

How are the causes divided?

What are the predisposing causes?

What are the exciting causes?

In what organs and tissues are the exciting causes most liable to produce the development of adenoma?

Upon what condition does the prognosis of true adenomata depend?

Do the true adenomata recur if completely removed?

In what respects do they differ from the malignant tumors?

How may death occur from true adenomata?

To what forms of degeneration are the adenomata subject?

With what glands is adenoma of the skin associated?

Describe adenoma of the sweat-glands.

Are they always associated with the glands?

Describe adenoma of the sebaceous glands.

What feature of the face is a favorite location for their growth?

What is the tendency of this form of tumor?

In the diagnosis of adenoma of the skin, what disease may be mistaken for it?

What is the prognosis?

What is the character of the treatment?

In how many forms does adenoma of the mucous membrane appear, and in what portions of the membrane are they located?

How is the first variety expressed?

How does the second variety differ from the first?

With what special mucous membrane is the first variety often associated?

What form do these growths assume?

What is the character of the majority of the nasal polypi?

What condition of the pharyngeal tonsil is common?

Do adenomata of the nasal passages ever attain large dimensions?

What form of degeneration sometimes occurs in the glands of the lining membrane of the antrum of Highmore?

What other form of degeneration may take place in adenoma of the antrum?

Describe the appearance of the "destructive adenomata."

What is the usual character of adenomata of the buccal mucous membrane?

Where are they generally located?
What is the character of their growth?
Give the diagnosis and symptoms.
Describe adenoma of the palate, and its location.
What is the first evidence of a large tumor in this location?
Give the diagnosis and symptoms.
What is the prognosis?
Describe the surgical treatment.
How may hemorrhage be controlled?
What should be the after-treatment?
Is adenoma of the tongue a common or a rare disease?
What is the character of these growths?
What is the explanation of the relative innocency of these tumors?
Give the diagnosis and symptoms.
What is the prognosis?
Describe the operation for their removal.
When hemorrhage is troublesome, how may it be controlled?
With which of the salivary glands is adenoma most frequently associated?
Is the disease common?
Which forms are most frequently associated with the parotid gland?
Are pure adenomata found in the parotid gland?
At what age do they most often occur?
Give the diagnosis and symptoms.
What form of degeneration is common in adenoma of the parotid?
Of what are the papillary excrescences composed?
What is the prognosis?
In operations for the removal of adenoma of the parotid gland, what special anatomical parts are involved?
With what reference should the incisions be made?
What form of fistula may result from the operation?
Is this likely to be permanent?
In extirpation of the gland for malignant growths, what artery may it be necessary to ligate?

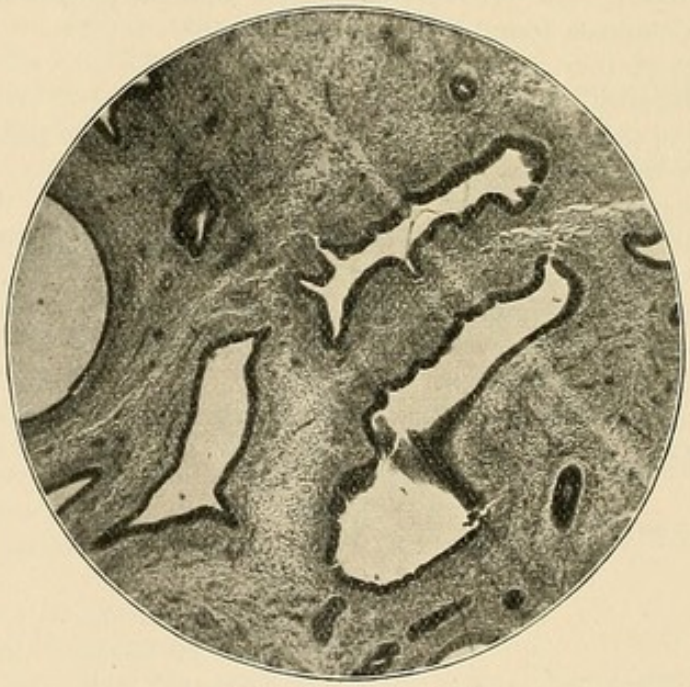
CHAPTER XLV.

CYSTOMATA.

Definition.—Cyst (Gr. κυστις, a pouch), a cavity containing fluid, and surrounded by a capsule.

"A cyst is a cavity, either natural or newly-formed, filled with a material more or less fluid, or pulpaceous, and surrounded by an investing membrane or capsule, which separates it from the surrounding tissues."

FIG. 195.



SIMPLE CYSTOMA. × 50.

Cysts may be divided into two groups: First, those which are formed by the dilatation of cavities already in existence—the natural cavities of the body—or which are formed by a tissue already present in the body, by softening or degeneration; and, second, those which are the result of new formations—neoplasms—which press apart the normal tissues and form cavities in locations where they did not previously exist. (Fig. 195.)

Virchow makes three divisions of the first group, according to the manner in which the filling of these cavities takes place, viz: Extravasation cysts, Exudation cysts, Retention cysts.

Senn would restrict the use of the term "cystoma" to those cystic tumors whose cyst-wall is produced from a matrix of misplaced embryonic cells, and whose contents are the product of tissue-proliferation of the cells which line the cyst-wall. This excludes all other forms of cysts except those which have originated independently of pre-existing cavities or glandular structures, and which are in the strictest sense new formations.

The cystomata are usually classed as formations belonging to the epiblastic and hypoblastic group of tumors. In a strictly histologic sense, only the cystic neoplasms should be placed in this group, but from a clinical standpoint all forms of cysts might be included in it. An examination of the cyst-wall shows the inner surface to be lined with epithelium or endothelium, according to its mode of origin.

The epithelial cysts arise from distention of epithelial cavities already in existence (Ziegler). A glandular cyst caused by the closure of its duct is an instance in point. The secretion which collects behind the obstruction dilates the gland, forming a cyst, which is filled with an altered secretion. These are termed *cysts of retention,* and are found associated in the oral cavity with the salivary and mucous glands. They are, however, more commonly seen in connection with the mammæ, kidneys, uterus, intestines, and the skin.

Endothelial cysts arise from distention of cavities in the connective tissue which had a previous existence, like tendon-sheaths, synovial sheaths, obstructed lymphatics, and old hernial sacs. These are *exudation cysts.* The contents consist usually of lymph.

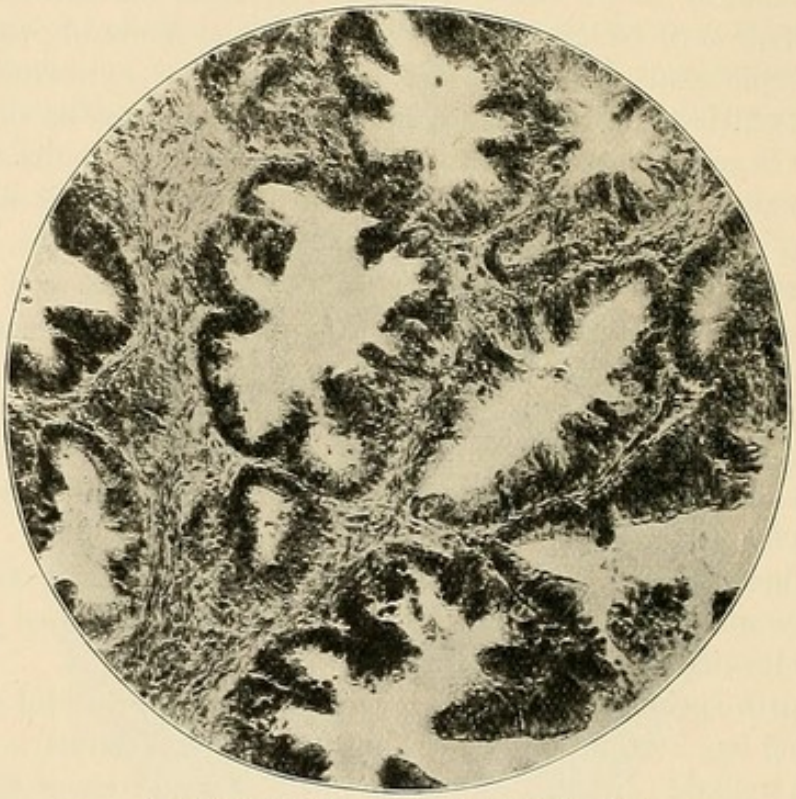
Another form of cyst is that which occurs in the substance of solid organs, by softening and disintegration (Ziegler) of defined portions. These are termed *cysts of disintegration.*

A fourth species of cyst is that which is formed around a foreign body which has become lodged in the tissues, like a bullet, or a parasite, like a hydatid (Ziegler), and is the result of a new-tissue formation.

The classification generally used by surgeons is one based upon the character of the contents of the cyst, viz: Serous cysts, fat cysts, blood cysts, mucous cysts, grumous cysts, etc. The extravasation cysts contain blood; the exudation cysts, serum; while the contents of the retention cysts would vary with the physiologic function of the glands with which they were associated. Cysts found in connection with the teeth are usually exudation cysts; those associated with the mucous membrane and salivary glands are retention cysts; while those found in the bone are the results of new formations. The contents of a cyst are always inclosed in a capsule or investing membrane, the capsule being of the same structure in all essential particulars as that lining the original cavity from which the cyst had its origin; consequently it would vary with the character of the anatomical structure

of the tissue in which it was located. In cysts associated with glandular structures there is usually a well-defined epithelial lining, and it is a generally accepted fact that in all cysts with a well-defined epithelial lining the cyst is not the result of a new formation. (Fig. 196.) This epithelial lining is subject to considerable change, as, for instance, when greatly distended, a glandular epithelium may resemble the lining of a serous cavity. It may also be lost, in great part, by fatty degeneration. The cyst membrane is made up of firm, fibrous connective tissue, which is always more dense than the surrounding soft tissue, but in some cases it is much better defined than in others. The firmness and density

FIG. 196.



MULTILOCULAR CYSTOMA—GLANDULAR. × 50.

of the capsule renders it possible in some cases to enucleate the cyst in its entirety. Inflammatory adhesions, however, often take place between the cyst-wall and the surrounding tissue, as a result of trauma or medication, which renders it difficult or impossible to enucleate it. In cysts formed in the bones an inner connective-tissue membrane is usually present, and may sometimes be dissected out.

Cysts may be classed as either *simple* or *compound*. A simple cyst is one consisting of a single cavity. A compound cyst is composed of an aggregation of simple cysts, or of many cavities which communicate more or less freely with one another.

A single cyst is spoken of as a *unilocular cyst*, and a compound cyst as a *multilocular cyst*. The small cysts which occur upon the mucous

membrane of the cheeks or upon the tongue, and in connection with the ducts of the salivary glands, are examples of unilocular retention cysts, while those which occur in the body of the glands where the acini of the glands become distended and communicate with one another are illustrations of multilocular retention cysts. The simple and compound cysts are frequently found in connection with bones. They are occasionally met with in the jaws, and sometimes are associated with the teeth. The most common forms of cysts are those associated with some glandular organ in which a tumor has formed, cutting off a portion of the glandular acini in which the secretion accumulates, distending the individual sacculi or lobuli.

Age seems to have little or no effect upon the formation of cysts. They may develop at any age from infancy to senility, and they are sometimes congenital. It has been thought by certain authorities that puberty exerted an unfavorable influence over those organs connected with the genital apparatus in which a predisposition existed to cystic formation, on account of the increase in the circulation and growth of the parts at this period.

Cysts, as a rule, are slow in growth, but retention cysts and extravasation cysts are sometimes very rapid in their formation, often attaining an enormous size in a short period of time. The size of cysts is exceedingly variable, ranging all the way from microscopic smallness to gigantic dimensions. The material forming the contents of a cyst is also subject to a variety of changes. It seldom happens in a cyst of long standing that the character of the contents remains in an unchanged condition. In extravasation cysts the blood undergoes the various changes incident to blood extravasations. It is first converted into a coagulum, which gradually grows more firm and dense, while the coloring matter is deposited in the form of amorphous coloring materials and crystals. Occasionally the blood will remain fluid for a considerable length of time, and apparently unchanged in other respects. In serous cysts the liability to undergo change is very considerable, the most frequent form being that of colloid degeneration, by which the contents are converted into a more or less thick, honey-like fluid. Cholesterin and fatty crystals of great variety are frequently found in profusion, while calcareous formations are not uncommon. Calcareous formations may arise from calcification of clots of fibrin, or they may be the result of direct precipitation from the retained glandular secretions, as sometimes occurs in occluded salivary ducts. Fatty degeneration is frequently found in slight degree in the epithelium of cysts, but it is of no particular consequence. In the higher degrees of metamorphosis the epithelium is thrown off, and the contents of the cyst become streaked or uniformly grayish-yellow or yellow. The walls of the cyst may also suffer fatty degeneration.

The capsule or investing membrane is liable to considerable change in the course of time. The most common is that of an increase of the thickness of the membrane, though the reverse of this may occur under exceptional circumstances. Calcification and, according to some authorities, ossification even may take place in the cyst-walls. When calcification takes place it usually forms in isolated places which have no connection with one another, though they occasionally unite and convert the entire cyst-wall into a firm, calcareous capsule. Under such circumstances the blood-supply is cut off by the occlusion of the vessels, all further growth of the tumor is arrested, and it assumes the position of a foreign substance in the tissues.

Destruction of the cyst membrane may also take place from inflammatory processes arising from traumatic injuries or inflammation extending from surrounding tissues. An injury which caused an extravasation of blood into a cyst containing serous fluid might cause coagulation of the entire mass and result in its obliteration. Inflammation sometimes leads to suppuration, and the cyst would then be converted into a pus-sac, which would destroy the epithelial lining and eventually close the cavity by granulation. Upon this fact is based the treatment often adopted by surgeons of inducing an inflammation by injecting irritating substances like iodin, etc., into the cyst.

Cysts may exist throughout a lifetime without producing any ill effects, yet their location and size may sometimes become a menace to life, as, for instance, when occurring in the neck, they may seriously interfere with the act of swallowing and with respiration. Cysts of the internal organs, when of large size, are the most dangerous to life, usually from the secondary disturbances which they induce in the organs of the abdomen and thorax, and by inflammatory processes ending in suppuration or other changes in the cyst itself; or rupture of the cyst and discharge of its contents into the peritoneal cavity. Peritonitis with a fatal termination has often resulted from the latter condition, on account of the highly irritating effect often possessed by the contents of such cysts.

Spontaneous involution—a shrinking or shriveling—of the cyst, whereby the capsule becomes hard, dense, and rigid, accompanied by degeneration of the papillary growths of the internal surface, has been observed in the ovarian cysts of old women.

Cysts of the Jaws and Teeth.—Cysts of the jaws which are found associated with the teeth may be classed under two heads: First, those which are connected with the roots of fully-developed teeth; second, those associated with malposed teeth or those of abnormal development.

Single cysts in the form of accumulations of serum or degenerated pus are frequently found in connection with the roots of devitalized

teeth which, from septic conditions, have been the subjects of inflammatory processes, usually of a subacute or chronic form, and which has resulted in necrosis or erosion of these apices. These cysts are always adherent to the apex of the root, the necrosed or eroded portion being inclosed in a sac, and when small enough to pass through the alveolus they are frequently found attached to the apex upon the extraction of the offending tooth. The size of these cysts varies considerably, and they are found associated with both jaws, but they never assume the character, and rarely the proportions, of the ordinary dentigerous or tooth-bearing cyst.

Diagnosis.—This class of cystic tumor is always found located beneath the periosteum, hence Magitot denominates them *periosteal cysts.* They vary in size from that of a pea to a marble, though they occasionally attain much larger dimensions. When of large size, they cause extensive resorption of the bone, considerable swelling and deformity, and if located in the superior maxilla they may simulate empyema of the maxillary sinus, and be mistaken for that disease, while in exceptional cases the cyst may occupy the antrum and give rise to a true empyema. Heath says that in his experience large cysts, which cause more or less absorption of the outer wall of the maxilla, are very common consequences of the retention of diseased teeth, but that they seem to give very little inconvenience to the patients, even though they may be so large as to produce a considerable deformity of the face.

A case of this character is described by Heath in a woman forty years of age. The tumor was of two years' standing, and situated immediately above the incisor teeth, which were decayed to the margin of the gum. The maxillary sinus had become secondarily involved, as was proved by passing a probe through the incision made above the incisor teeth for the evacuation of the fluid. A case somewhat similar, which came under the observation of the writer, was associated with a superior lateral incisor and involved the antrum. Fischer, as quoted by Heath, reported a case of a large cyst associated with the root of a superior posterior molar, in which he had the opportunity of making a post-mortem examination. After removing the external wall of the antrum, the cyst was found to be connected with the pericementum at the apex of one of the roots, and it filled the whole of the antrum without being connected with the lining mucous membrane. The cyst was composed of a perfectly closed serous sac, with a smooth inner surface, and contained a yellowish serous fluid.

An example of a common form of cysts in the inferior maxilla associated with devitalized teeth occurred recently in the private practice of the writer. Miss G., aged thirty-five, housekeeper, had a painless swelling of the right side of the lower jaw in the region of the bicuspid teeth. The swelling was confined to the outer surface of the

jaw, and extended from the cuspid tooth to the first molar; the gum over the tumor was purple in color and hard to the touch, except at one point near the center, where there was evidence of fluctuation. Both bicuspid teeth were devitalized, with a history of acute abscess occurring two or three years previously. Puncture of the tumor revealed a cyst containing a thick, straw-colored, tenacious fluid, in which were found glistening flakes of cholesterin. The cyst was evacuated and explored, when it was discovered that the roots of both of the bicuspids were involved in the cyst, and that they were considerably roughened near their apices. On extracting the teeth, it was found that erosion had taken place at the apex in both of them.

Another case, in which the cyst was located in the superior maxilla, may be mentioned as a rather uncommon type. Mrs. W., aged twenty-eight, a private patient, came in September, 1891, with a large swelling under the left ala of the nose, involving the lip and side of the face. The swelling was painless, and had been of rapid growth, the first evidence having been noticed only five days before. On raising the lip, the gum was found to be considerably swollen, very red, the disturbance extending from the cuspid tooth forward to the median line. Fluctuation was discernible over the center of the swelling. The incisor and bicuspid teeth had all been removed some years before, on account of extensive caries and abscesses, with a previous history of swelling and discharge of a watery fluid at frequent intervals in the location of the left lateral incisor, twice repeated at short intervals after the extraction of the teeth. The case was treated by incision and a 10 per cent. solution of iodin in glycerol, with seemingly a speedy cure. About six months later she returned with a recurrence of the disease. This time a careful search was made for an unerupted tooth or a fragment of the root of a devitalized tooth, but nothing of the kind could be found. The cavity was then thoroughly curetted, under the belief that the original cyst-wall had not been destroyed when the tooth had been extracted, and that it still retained the power of exciting a serous exudation. After washing out the cavity, it was packed from day to day with borated gauze. At the end of a month the cavity had closed by granulations, and no further inconvenience had occurred after a period of nearly four years.

The character of the fluid contained in cysts associated with the diseased roots of teeth is generally a dark straw-colored, gluey liquid, often containing flakes of cholesterin. Occasionally it is thick and ropy, or if inflammation has been recently present, the contents may be purulent. Tomes suggests that cystic disease of the lower jaw may not infrequently be associated as the initial cause of irritation with the roots of diseased teeth. When the disease is located in the lower jaw, it is generally the external plate of the alveolus which gives way under

the pressure of the accumulating fluid. As resorption of the bone progresses, crepitation over the swelling is discernible, and if sufficient time elapses the bone will become completely resorbed, while fluctuation may be detected through the membranous covering of the cyst. Spontaneous rupture of the tumor sometimes occurs, but this does not effect a cure, for as soon as the opening in the cyst-wall has healed the fluid reaccumulates.

The history of this form of cysts, from a clinical standpoint, is one of painless expansion of the alveolus and surrounding bone, resorption of the bony wall, crepitation under pressure, evidence of fluctuation, bluish appearance of the gum immediately overlying the cyst. It may be located in the alveolar process of either jaw, but most commonly in the superior. Wedl is of the opinion that the growths are more commonly found anterior to the bicuspid teeth than behind them.

Prognosis.—The prognosis is one of recurrence, unless a radical operation is performed. Beyond the deformity caused by the swelling and its progressive enlargement, it need give no anxiety as to its issue, for fatalities from this cause are unheard-of results.

Treatment.—The treatment, to be effective, must be radical. The diseased tooth must be first extracted, after which the cyst should be opened and the thin external wall cut away with scissors or the bone forceps, and the cavity thoroughly curetted. The surgical engine and bone-cutting burs will serve an admirable purpose in this class of operations. Curetting the cavity without removing the tooth, or removing the tooth without curetting the cavity, are only half-way measures, and will be followed, sooner or later, by a recurrence of the disease. Simple draining of the cavity is likewise a futile proceeding, as are all attempts to evacuate the cyst through the pulp-canal. Failure to cure this form of cyst has many times been caused by lack of proper appreciation of these simple facts. The cavity, after it has been curetted, should be dressed by packing it with bichlorid or boric acid gauze, and the dressings changed once or twice each day until the process of healing by granulation at the bottom of the cavity has been established, when the gauze may be dispensed with, and the wound kept clean by frequent irrigation. The expanded condition of the bone will gradually disappear as the healing process goes on, and eventually the deformity will entirely pass away.

REVIEW.

CHAPTER XLV.

What is the definition of a cyst?
How are cysts divided?
What are comprehended in the first group?
What in the second?
How did Virchow divide the first group?
Upon what is the classification usually based by surgeons?
What is an extravasation cyst?
What is an exudation cyst?
What is a retention cyst?
What form of cysts is usually connected with the teeth?
What are they called when associated with glands?
What are they called when associated with bone?
By what are the contents of a cyst inclosed?
What is the character of this investing membrane?
What is the character when associated with glandular structures?
What does a well-defined epithelial lining indicate?
What forms of degeneration may attack the investing membrane?
Of what is the cyst membrane composed?
Is it possible to enucleate the capsule?
What conditions may render it impossible to enucleate it?
How are cysts classed?
What is a simple cyst?
How does a compound cyst differ from a simple cyst?
What other terms are applied to these forms of cysts?
Give examples of unilocular retention cysts.
Give illustration of multilocular retention cysts.
Are simple and compound cysts found in connection with bone?
Are they commonly associated with the jaws and teeth?
What is the most common form of cyst?
What effect does age have upon the formation of cysts?
Are they ever congenital?
Does puberty exert an unfavorable influence or predisposition to the formation of cysts?
What is the character of their growth?
Are retention and extravasation cysts slow or rapid in their formation?
What can you say of the size of cysts?
What are the changes to which the cyst contents are liable?
What change takes place in extravasation cysts?
What in serous cysts?
How do calcareous formations arise?
How do they arise in retained glandular secretions?
What changes may take place in the capsule of the cyst?
Does complete calcification ever occur, and how?
How may an injury cause obliteration of a cyst?
How may inflammation produce the same result?
What are the means sometimes adopted by surgeons to bring about this result?

Under what conditions may cysts prove a menace to life?
How may cysts of internal organs prove fatal?
How are cysts of the jaws classed?
What are included under the first head?
What under the second?
What forms of cysts are found in connection with devitalized teeth?
What are the causes?
What is the condition of the apices of the teeth under these conditions?
To what is the cyst always adherent?
What can you say of their size?
Where are they located?
What are the diagnostic signs?
Do they ever involve the antrum?
What is the character of the fluid contained in cysts associated with devitalized teeth?
When the disease is located in the lower jaw, which plate of the alveolus usually gives way first?
What is the clinical history of this form of cysts?
Which jaw is the most frequent location of these cysts?
What is Wedl's opinion as to their location?
What is the prognosis?
Do fatalities ever occur from this form of cysts?
What must be the character of the treatment to be effective?
Of what should this radical treatment consist?
Are half-measures of any value?
What will be the result of such measures?
What should be the character of the dressings?
How often should they be changed?
When should the packing be dispensed with?
What should be the treatment after granulation has been established?
Will the deformity remain or gradually pass away?

CHAPTER XLVI.

CYSTOMATA (Continued).

Multilocular Cysts of the Jaws.

Definition.—A multilocular cyst is a congeries of small cysts; a polycyst; a variety of cyst having many cavities, or a cyst containing many similar smaller cysts attached to the inner wall of the original cavity.

True cystoma of the bone is an exceedingly rare affection, only a very few cases ever having been reported. The most interesting one is that described by Engle, which occurred in a woman fifty-five years of age, the mother of six healthy children, and who during her life gave no evidence of any bone-affection. Death occurred from an acute disease, and at the post-mortem examination it was discovered that the entire skeleton was occupied by innumerable cysts, ranging in size from a pea to three inches in diameter. The cyst-walls were composed of a layer of connective tissue, and the cysts contained in some instances a clear, in others a bloody serum.

In a few instances single cysts of considerable size have been found in various bones.

Multiple bone-cysts resulting from "embryonic inclusion of a matrix of epithelial cells" (Senn) are most commonly associated with the maxillary bones. Tumors of this character have been observed much more frequently in the lower than in the upper maxilla.

Multilocular cysts of the jaws, also known as *proliferating follicular cystomata*, and designated by Sutton as *epithelial odontomes*, are a type of cystomata that is very rare.

Becker recently described two cases which were seen at the clinic at Bonn, and he has been able to find but sixteen additional cases reported in surgical literature.

Cysts which are formed from the beginning with separate compartments, or which are produced later by coalescence with other cysts, or by proliferation from the original cyst, are termed multilocular cysts.

Causes.—Multilocular cysts of the jaws are in most cases found associated with the teeth or with the roots of teeth which have lost their crowns from caries, and which have, through their septic influ-

ence, been the cause of long-continued irritation and inflammation. Occasionally their development has been known to follow an injury to the jaw.

The etiology of this affection has always been somewhat obscure, and even at the present time there still remains considerable doubt as to the real cause of the disease.

Eve maintained that multilocular cysts of the jaws were not of dental origin, but that they were caused by an ingrowth or inclusion of the epithelial layer of the gum, and that they followed injury and long-continued irritation from diseased teeth and inflammation. He applied the term *multilocular cystic epithelial tumor* to these growths.

Senn believes that they are developed from an embryonic inclusion (matrix) of epithelial cells, and that it is evident that misplaced dental germs are not the cause of the affection, from the fact that the lower jaw is most frequently the seat of the disease. This latter fact may find its explanation in the greater liability of the lower jaw, from its exposed position, to receive injuries, while the upper, from its location, is comparatively free from traumas.

Sutton thinks that multilocular cysts or *"epithelial odontomes* arise probably from the presence of persistent portions of the epithelium of the enamel-organs."

The origin of supernumerary teeth and small, malformed teeth or denticles often found in the dentigerous cysts may also be explained in the same manner. These persistent portions of the epithelium of the enamel-organ are derived from the epithelial cord of the tooth-germ, which has been cut off from the enamel-organ by the closing of the dental follicle. After the cord has been separated from the enamel-organ by this process, it breaks up into minute globules (Magitot), which are absorbed; but if for any reason they are not removed by absorption, these globules may develop into supernumerary teeth or denticles, or they may induce the formation of multilocular cysts, the "epithelial odontomes" of Sutton.

Audry is of the opinion that multilocular cysts have a positive connection with the enamel-organs; he also succeeded in demonstrating the epithelial origin of these growths. Their origin, therefore, would invest them with a certain degree of malignancy. These observations most positively confirm the first researches of Falkson and Malassez as to the origin of many of the maxillary tumors.

Kruse also confirms these investigations, and considers the origin of these cysts to be the paradental epithelial *débris* of Malassez.

Multilocular cysts are sometimes termed *proliferating follicular cystoma*, from the nature and character of their development, which is generally thought to be due to the gland-like arrangement of the "tumor matrix" and the proliferation of the epithelial cells. The can-

cellated structure of the bone may favor the multiple character and growth of this variety of cyst, by the rupture or absorption of the thin septi or partition walls which exist between the vacuoles or loculi, as the cysts grow and the fluid increases in quantity.

The recent anatomical researches of Cryer upon the inferior maxilla also favor the supposition that the multilocular character of these cysts may be influenced by the peculiarities of the structure in which they are formed. Cryer has demonstrated the inferior dental canal to be a cribriform structure; that an abundant communication exists between the vacuoles or loculi of the cancellated tissue of the bone; that the alveoli of the teeth are not only in communication with the inferior dental canal, but with the loculi of the cancellated tissue in all directions, and with one another through the same channels, thus furnishing, in the opinion of the writer, a possible explanation of the multilocular character of those cysts of the lower jaw which appear to be induced by the irritation of diseased teeth and traumatic injuries.

If, therefore, this supposition is correct, a single cyst of epithelial origin, located in any portion of the alveolar process, the ramus, or body of the maxilla, might readily become multiple by the growth following the communicating canals and occupying the loculi of the cancellated tissue in its immediate neighborhood, expansion of the loculi taking place as the fluid accumulated.

Inflammation alone, according to Senn, is never productive of tumor-formations,—neoplasms,—but that inflammation occurring in the immediate neighborhood of a tumor-matrix, whether of pre-natal or post-natal origin, causes an increase or augmentation of its blood-supply, which arouses the embryonic tissue from its dormant condition and stimulates it to active cell-proliferation.

Multilocular cysts of the jaws (Fig. 197), according to the best authorities upon this subject, may be stated to be caused by the presence within the jaws of embryonic *"inclusions"* or *"nests"* of epithelial tissue—probably derived from the epithelial cords of the enamel-organs during the development of the teeth, and which have been stimulated to active cell-proliferation by injuries to the jaws, inducing an increase in their blood-supply through inflammatory conditions.

The causes, therefore, may be divided into Predisposing or Essential, and Active or Exciting.

The *predisposing* or *essential* cause is the presence within the jaw of an embryonic epithelial tumor-matrix.

The *active* or *exciting* causes are inflammatory conditions and traumatic injuries.

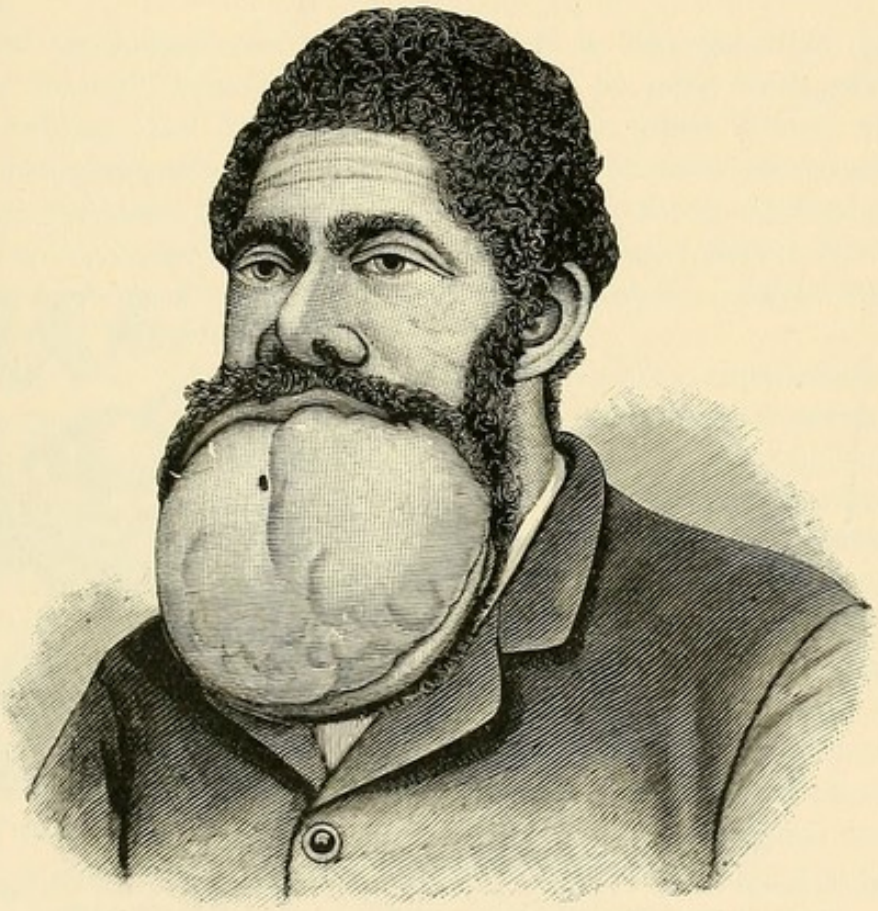
The disease is essentially one of early life, the majority of cases occurring under thirty years of age. Of the published cases, the youngest was an infant six months old, the cyst being congenital

(Heath), and the oldest an individual of seventy-five years of age. The development of these cysts usually begins in childhood or at puberty, and they are of slow growth, though they may attain a very considerable size.

One case described by Falkson and Bryk reached an enormous size, the tumor weighing one and one-half kilograms, and extending from the zygomatic arch to the sternum (Senn).

They are located most frequently in the region of the bicuspid and molar teeth. When associated with the upper jaw they may rupture into the antrum of Highmore.

FIG. 197.



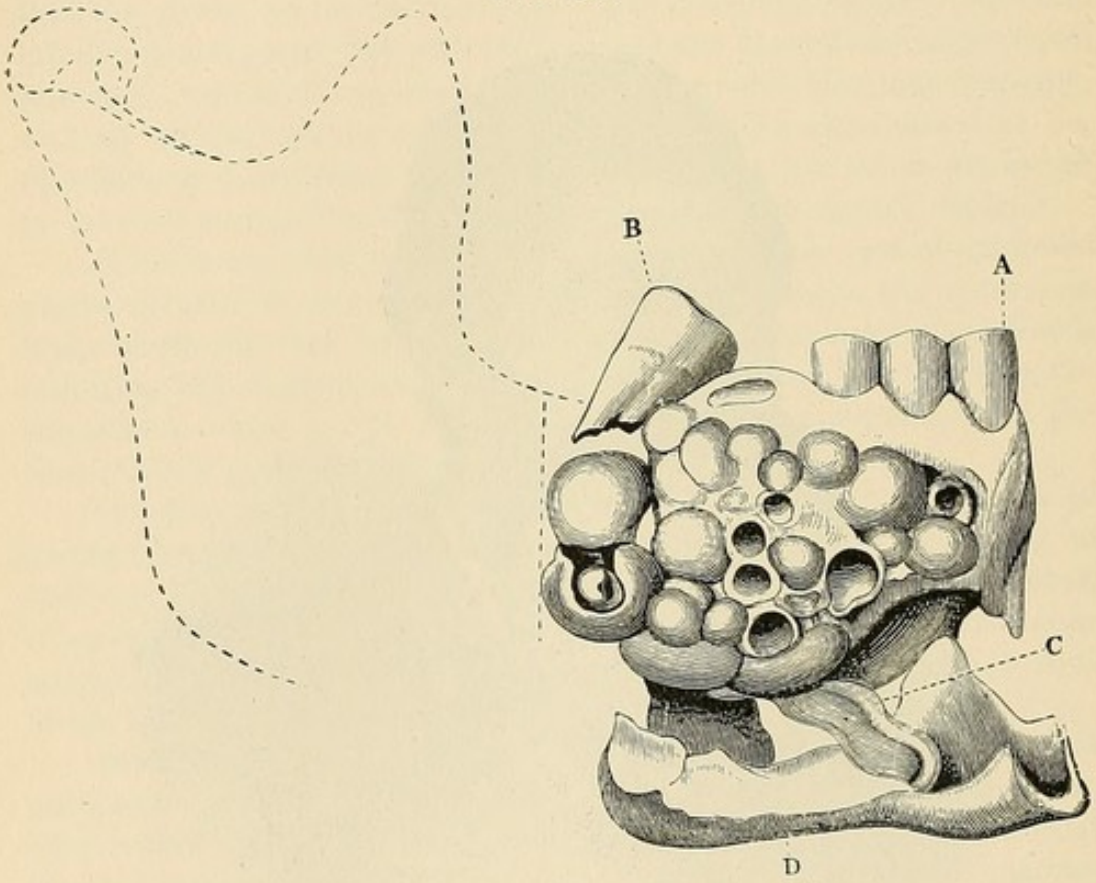
MUTILOCULAR CYST OF THE LOWER JAW. (After Rogers.)

Pure multilocular cysts are commonly considered to be benign growths. Degenerative changes, however, are liable to occur in those cysts which are present in the middle and later periods of life, the tendency being toward sarcomatous and carcinomatous transformation. Authorities, however, differ as to the original character of malignant multilocular cysts of the jaws. Some believe them to be a form of cystic degeneration of sarcomatous and carcinomatous growths, while others are of the opinion that they are malignant degenerations of multiple cysts which were originally benign.

The histologic character of multilocular cyst of the jaws is that of an epithelial tumor, and consists of branching and anastomosing columns of epithelium, portions of which form alveoli. The cells which occupy the alveoli vary; the outer layer may be columnar, while those in the center degenerate and give rise to tissue resembling the stratum intermedium of an enamel-organ (Sutton).

Clinically these cysts have a firm capsule, and are composed of a great number of smaller cysts grouped together and occupying the cancellated structure of the bone. The individual cysts vary in shape

FIG. 198.



MUTILOCULAR CYST OF THE LOWER JAW. (After Adams.)

A, cuspid; B, second molar; C, anterior portion of dental nerve; D, remains of the base of the horizontal ramus excavated on its upper surface, on which lay the tumor.

and size. They are usually of the dimensions of a small pea to that of an almond; exceptionally they are much larger. These cavities are separated by thin fibrous or bony septi; when the disease is of long standing, they often communicate freely with each other. (Fig. 198.) The contents of the cysts may be a clear, limpid, mucoid fluid; in other cases, thick and almost gelatinous, and of brown or dark-brown color.

The cysts in the portions of the tumor which are growing are lined with a very red, pulpy, vascular membrane, resembling a myeloid sarcoma.

Diagnosis and Symptoms.—The cystic character of these tumors may be easily ascertained by the introduction of a trocar or an exploring needle, but the multiple character of the cyst cannot be positively demonstrated except by an incision, or by the extraction of the teeth and diseased roots involved in the cyst, which may sometimes demonstrate its character by the escape of the fluid.

In making a differential diagnosis this form of cyst must not be confounded with the simple cysts which follow suppurative alveolar inflammation of devitalized teeth, nor with dentigerous cysts, nor with cysto-adenoma, cysto-sarcoma, or cysto-carcinoma. The character of the three latter forms can only be determined by microscopic examination.

The symptoms are the presence of a slow-growing, painless tumor, situated in the region of the bicuspid or molar teeth, usually in the lower jaw, but occasionally in the upper, when it may simulate mucous engorgement, or cyst of the antrum. It rarely produces ulceration of the gums or infiltration or ulceration of the external tissues, but when such conditions have occurred they have always been associated with a malignant degeneration of the growth. Heath mentions three cases of this character.

Prognosis.—Multilocular cysts of the jaws are comparatively innocent growths; they show very little tendency to implicate surrounding tissues, to involve the neighboring lymphatic glands, or to cause metastatic deposits. When thoroughly removed by surgical operation, they rarely recur. Their comparatively benign character is doubtless due to the bony capsule which surrounds them (Heath), their somewhat scanty vascular supply, and the especially marked tendency possessed by the epithelial cells lining the cysts to undergo colloid degeneration.

The opposite is true of those cysts which are characterized by a sarcomatous or carcinomatous degeneration of their connective or epithelial tissues. Under such conditions there is a distinct tendency to the involvement of the neighboring lymphatics and surrounding tissues, and to the formation of metastatic tumors.

Treatment.—The plan of treatment usually followed in cases of multilocular cysts of the jaws is to make the operation through the mouth, in order that unsightly scars may be avoided. It requires a higher degree of skill and a larger endowment of patience upon the part of the surgeon to operate successfully through the mouth in these cases than it does to lay open the tissues of the face and operate through an external incision.

When operating through the mouth, the jaws should be separated as widely as possible with a suitable mouth-gag or prop. The mucous membrane over the cyst is then freely divided, and dissected from the

external plate of the bone. With gouge and bone-forceps the external plate of bone is cut away, exposing the character of the cyst.

The bony and fibrous septi are now broken down and removed, and the inner surface of the cavity thus formed thoroughly curetted, any sharp projections of bone being smoothed down. After irrigation, the cavity is packed with antiseptic gauze. Granulations soon spring up and fill the cavity, and gradually the expansion of the bone and the fullness of the cheek disappear, and no scar is left to mar the symmetry of the face.

When the character of the cyst is of a nature that requires the exsection of a portion of the body of the jaw, the incisions should be made in such locations as will cause the least deformity by reason of the cicatrix. This may be accomplished by a vertical incision through the lower lip upon the median line, carried under the point of the chin to intercept another incision made from the angle of the jaw, following the inner border, to the median line. The soft tissues are then dissected from the bone and laid back upon the face. This exposes the jaw from angle to symphysis. A tooth is extracted in front of and behind the tumor. The bone is then cut through with the Hey's saw, the chain saw, or a circular saw revolved by the surgical engine, and removed. The vessels are then secured, the flap stitched into position, and antiseptic dressings applied.

By these lines of incision the greater part of the cicatrix is hidden from view, which is a matter of considerable importance to the unfortunate patient.

Sometimes, by cutting away the diseased bone from above, it may be possible to leave a rim of healthy bone at the base of the jaw; this would be much better than to make a complete exsection, as an unsightly deformity often results from the latter operation.

REVIEW.

CHAPTER XLVI.

Is cystoma of the bone a common or a rare affection?

With what bones are the multilocular cysts most frequently associated?

In which maxilla is it most often seen?

What other terms are used to designate this disease?

Is the disease of frequent or rare occurrence?

What are the conditions which determine the application of the term *multilocular cyst?*

What are the causes which may establish the development of this form of cyst?

Is the etiology of the disease well understood?

State the opinion of Eve as to the origin of the disease.

What term did he use to designate the affection?

State the opinion of Senn upon this point.

What explanation is given that may account for the greater frequency of the disease in the lower jaw?

State Sutton's opinion upon the origin of the disease.

What process may take place in the enamel cords to form supernumerary teeth, denticles, and multilocular cysts?

What other term is used to designate the multilocular cysts?

What feature in their development would make such a term applicable?

How may the cancellated structure of the bone favor the growth of these cysts?

What new facts have the researches of Cryer established in relation to the inferior dental canal, the cancellated structure of the bone, etc.?

Is inflammation productive of the formation of tumors?

How may it cause the development of tumors?

What is the stated cause of multilocular cysts of the jaws?

How may the causes be divided?

What is the essential or predisposing cause?

What are the active causes?

At what period of life is the disease most common?

What is the character of the growth and size of multilocular cysts?

In what region of the jaw are they most frequently located?

When located in the upper jaw, what sinus may they involve?

What is the nature of a pure multilocular cyst?

What changes are liable to occur?

Are the malignant cysts of this character benign or malignant at the beginning?

Describe the histologic character of multilocular cysts.

Describe their clinical appearance.

What is the character of the contents?

What is the appearance of the growing portions?

How may they be diagnosed?

Give the differential diagnosis.

What are the symptoms?

Do ulceration and infiltration of the gum and external tissues take place?

What is the prognosis?

How is their comparatively benign character explained?

What is the prognosis for those characterized by malignant degenerations?

What is the tendency in these conditions?

What is the usual location selected for the operation?

What is the value of operating through the mouth?

Describe this operation.

Describe the operation for exsection of the jaw.

Describe the operation by which the conformation of the jaw may be preserved in certain cases.

CHAPTER XLVII.

CYSTOMATA (Continued).

Dentigerous Cysts.

The term "Dentigerous Cyst" is applied by most writers to that form of cystic tumor associated with some aberration in the development of the teeth, and which prevents the normal process of their eruption. Dentigerous cysts are most often found in connection with the permanent teeth, occasionally with supernumerary teeth, but very rarely with the deciduous teeth. The disease may occur in either jaw, but most frequently in the upper.

The most frequent aberrations in the development of the teeth are malpositions and malformations, and these conditions seem to be those which most often act as causative factors in the production of the disease. A careful review of the literature upon the subject, supplemented by personal observation in an extended hospital practice, confirms the opinion that in nearly every case the aberrant tooth or teeth, including the supernumerary teeth, found in a dentigerous cyst were so malposed or malformed as to render it impossible for them to emerge from their crypts and assume a position in the dental arch.

It is not to be supposed, however, that every malposed or malformed tooth which remains impacted in the jaws results in the formation of a cystic tumor, or that even a majority of them so result. Individual teeth frequently fail to make their appearance in the dental arch, and many have been found in abnormal positions, or abnormally developed, but comparatively few ever give rise to serious trouble. The disease may therefore be considered as a rare affection.

Sutton applies the term "Follicular Odontomes" to the tooth-bearing or dentigerous cysts. He describes them as follows: "Follicular odontomes arise commonly in connection with teeth of the permanent set, and especially with the molars. Sometimes these tumors attain large dimensions and produce great deformity. The tumor consists of a wall of varying thickness, which represents an expanded tooth-follicle. In some cases it is thin and crepitant; in others it may be one centimeter thick. The cavity of the cyst usually contains viscid fluid and a crown or root of an imperfectly developed tooth. Occa-

sionally the tooth is loose in the follicle, sometimes inverted, and often its root is truncated; exceptionally the tooth is absent. The walls of the cyst always contain calcific or osseous matter; the amount varies considerably. Follicular odontomes rarely suppurate."

Aberrant teeth which pierce the gum are never productive of cystic tumors, though it occasionally occurs that an accumulation of serous fluid will be found surrounding the crown of an advancing tooth, but this is immediately discharged upon the tooth piercing the gum, and does not again reaccumulate. Cysts of considerable size sometimes develop in this way when the temporary teeth, particularly the molars, are retained beyond the proper time for them to be exuviated. Tomes suggests that this condition may occur in teeth which are deeply imbedded in the jaws, the fluid collecting between the "enamel and the tooth-capsule, and as the fluid increases in amount the bone next to the tooth is resorbed, while new bone is formed upon the outside of the jaw."

Salter says, "When a tooth is impacted in the jaw, its fang is enclosed in a bony socket, lined by periosteum, as in ordinary circumstances, while the crown of the tooth is free in a little bony loculus lined with that which was the so-called 'enamel-pulp.' This structure is clothed with a sort of epithelium, which is apt to assume the function of secreting fluid."

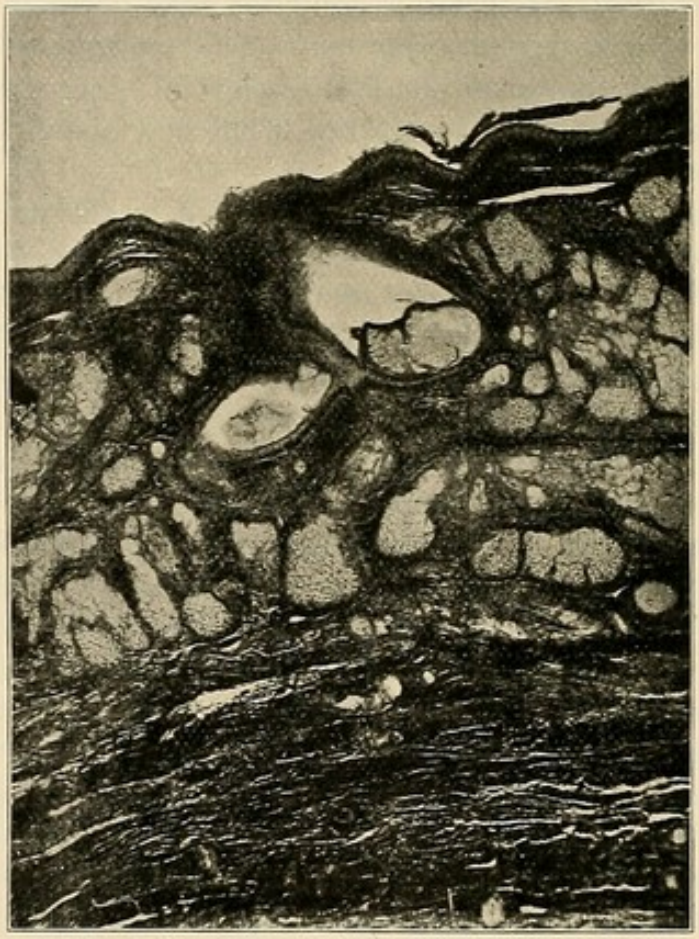
Causes.—All serous exudations are the result of some form of irritation, usually of a mild chronic type, but it is not always an easy matter to determine the exact character of the irritation. In cysts connected with the roots of devitalized teeth, the irritant is easily determined, but in exudations not so associated it is a more difficult matter. It would seem, however, that in the formation of dentigerous cysts the irritation was simply mechanical, identical with that which accompanies the advancing tooth in the normal process of eruption; but on account of the malposition or the malformation of the offending tooth, the advancement of the crown is impeded. As a consequence of this impediment to its progress, irritation of the surrounding tissues is produced, resulting in a low inflammatory process, with serous exudation, gradual accumulation of the fluids, expansion and resorption of the bone, accompanied by more or less swelling and deformity.

The changes which take place in the contents of dentigerous cysts are in no way different from those which are found in connection with serous cysts in other portions of the body.

The dentigerous or tooth-bearing cysts are not always confined to the jaw, but are found in the ovaries of the human female, as expressions of a modified form of reproduction; while those which occur in either sex, and remote from the location of the reproductive organs, must be classed as dermoid cysts.

Dermoid Cysts are formed as a result of an infolding of the epiblast or hypoblast, which is thereby displaced and surrounded by the connective tissue. These cells thus buried in the connective tissue continue to develop, and result in the formation of an epidermal cyst (Fig. 199), which may contain any or all of those tissues which are developed from the epiblast or hypoblast, namely: the skin, hair, nails, mucous membrane, the teeth, and occasionally muscle, bone, and cartilage; but under the latter circumstances portions of the mesoblast must have been included with the misplaced portions of the epiblast and hypoblast.

FIG. 199.

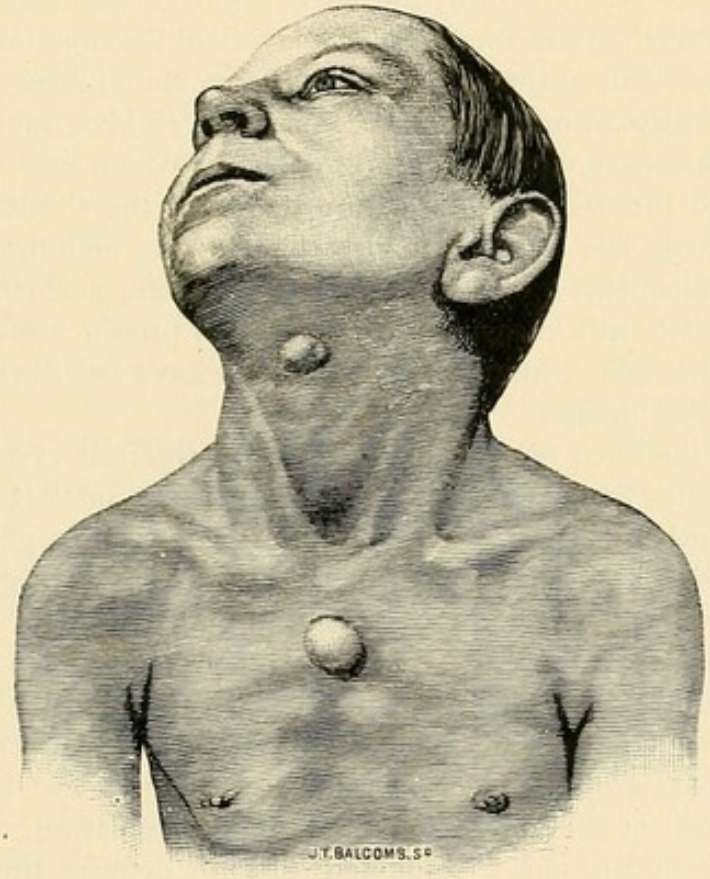


DERMOID CYST—TRANSVERSE SECTION. × 40.

The most common location of dermoid cysts is in the generative organs, especially the ovaries. They are occasionally found in other parts of the body, like the peritoneum, the neck, the sternum (Fig. 200), the region of the orbit, the cheek near the angle of the mouth, in the median line of the chin, on the side of the nose, and in the median line of the palate. In rare cases, fragments of bone, flat or irregular, or of cartilage, and even of teeth, are found beneath the cutaneous layer. The teeth are occasionally free within the cyst. Teeth are

sometimes found in connection with the branchial clefts of sheep, oxen, and horses. Sutton mentions such a case occurring in a sheep with persistent fistula of the second branchial cleft and surmounted by a prominent cervical auricle, covered upon its posterior surface by a number of processes resembling the buccal papillæ of sheep. From this auricle grew an ill-formed incisor tooth, mounted upon a projection of bone and surrounded by mucous membrane. Professor Sayer, of the Chicago Veterinary College, recently opened a dermoid cyst

FIG. 200.



DERMOIDS OVER THE STERNUM AND THE LEFT CORNER OF THE HYOID BONE, IN BOY AGED NINETEEN. (Bramann, after Sutton.)

situated in the temporal region of a horse which had been discharging an offensive secretion for several months, and found lying upon the bottom of the cyst a well-developed incisor tooth.

Dermoid cysts are classed among the congenital tumors. They are found most commonly in young persons, though they have been discovered in the aged. Their growth is usually very slow and entirely painless.

The simple forms are those found in detached or sequestered portions of the surface epithelium, where during embryonic life union takes place between skin-covered surfaces; as, for instance, upon the median line of the body, and along the lines of coalescence of the vari-

ous embryonic fissures of the face and neck. The more complicated forms are those occurring in the ovaries and in connection with mucous surfaces. Sutton is authority for the statement that teeth are never found in those dermoids which arise in connection with the surface epithelium. They are, however, of fairly frequent occurrence in ovarian dermoids, and also in rectal and post-rectal tumors of this character. The writer is of the opinion that teeth are never found in dermoids other than those associated with the generative organs or the mucous membrane. The former class result from an abortive effort of the function of reproduction; the latter from an accident of development. The teeth are formed from the layers of the mucous membrane, the hypoblast, and not, as often stated, from the epiblast. The same accidents of displacement which occur in the epiblast may, under the same circumstances, happen to the hypoblast, which may result in the formation of tumors containing any or all of the tissues which are developed from the hypoblast, viz: the mucous membrane, with its glandular appendages, etc., and the teeth. These accidents of development explain, in a measure, the occasional presence of teeth in the cervical region of the human subject, which have been usually classed as erratic third molars.

One of the most remarkable cases of dentigerous cysts ever noted is that described by F. E. Glaswald, of Pomerania, in 1844, and quoted at some length by Salter. The history of the case, as gathered from Salter, is briefly as follows: The patient was a healthy girl of about eight years of age when the disease first showed itself. The earlier symptoms were frequent attacks of violent pulsating pain in the right superior maxilla, involving the alveoli and the teeth, which were relieved by fomentations and general treatment. A year later the symptoms recurred, accompanied by redness, swelling, and fever. After the subsidence of the active symptoms of inflammation, a permanent enlargement of the cheek remained, but no definite tumor. These attacks were frequently repeated during the following two years, with a constantly increasing fullness of the cheek, so that at the age of ten years the right side of the face presented a large, tumor-like prominence. An unusually violent attack occurred at this time, accompanied by inflammation of the antrum and elevation of temperature, which was supposed to have been the result of exposure to cold. At the end of five days the sinus opened spontaneously in the zygomatic region, with profuse discharge of pus. The case was then treated in hospital by Dr. Warnekros, who extracted the molar teeth, dilated the external aperture, and established a counter-opening in the canine fossa. This resulted in diminishing the size of the tumor. Five years later the left cheek was attacked in precisely the same manner, and continued until the patient was nineteen years of age. The right side

also, at the same time, began slowly to enlarge. A second operation was performed at this time by Dr. Kneip by opening the left antrum, which was followed by the discharge of a large quantity of fetid pus. No necrosed bone could be discovered. The patient had now become very much disfigured, the antra remaining permanently dilated and the cheeks very prominent. A third operation was made by Professor Baum, who opened both antra and removed a portion of the external walls. Upon the left side an opening was made just below the canine fossa, which was followed by the discharge of about an ounce of clear yellow, fetid serum, and upon exploring the antrum with the finger the crown of a molar tooth was discovered firmly attached to the bone; it was extracted with difficulty. The right antrum was opened in about the same location, and a cuspid tooth was found loosely attached to the wall and easily removed. This sinus contained fetid pus. After several months, there was little change in the size of the tumor.

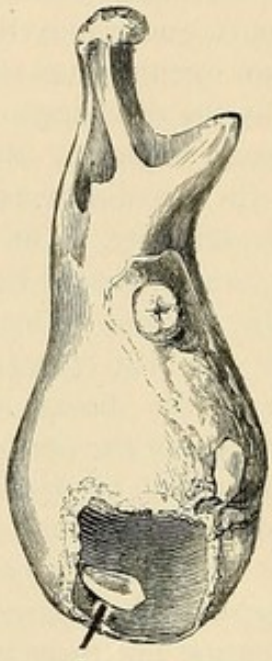
Another notable case of the disease occurring in the inferior maxilla published by Mr. Fearn, of the Derby Infirmary, in the *British Medical Journal,* 1864, and quoted by Heath, is also of especial interest. The patient, a girl thirteen years of age, had a large, hard tumor, which occupied the whole of the horizontal ramus of the left side, and which had been growing for six months. The surface of the tumor showed a fetid discharge, but there was no discoverable opening. The right side of the maxilla was also somewhat enlarged, and the teeth irregular. The teeth of the right side had been extracted with the exception of a temporary molar and the second permanent molar. The left half of the jaw was removed from the symphysis to the articulation, under the mistaken diagnosis of a solid tumor. The tumor proved to be a bone-cyst, formed by the expansion of the two plates of the maxilla, and which extended for some distance to the right of the symphysis. The cyst was lined with a thick vascular membrane, and at the bottom a cuspid tooth was found projecting from the wall, and which was evidently the cause of the disease. (Figs. 201, 202.)

Another somewhat similar case was published by Dr. Forget. The patient was a woman thirty years of age, having a tumor of the right side of the inferior maxilla the size of a hen's egg, which extended from the lateral incisor to the base of the coronoid process, and had been slowly growing for ten years. M. Lisfranc removed the right half of the jaw, and upon examination of the tumor it was found to be a dentigerous cyst, with the third molar in an inverted position, the roots located in the base of the coronoid process, and the crown projecting downward and into the cyst. (Fig. 203.)

The following cases from the practice of the writer are of sufficient interest in this connection to warrant their appearance in these pages:

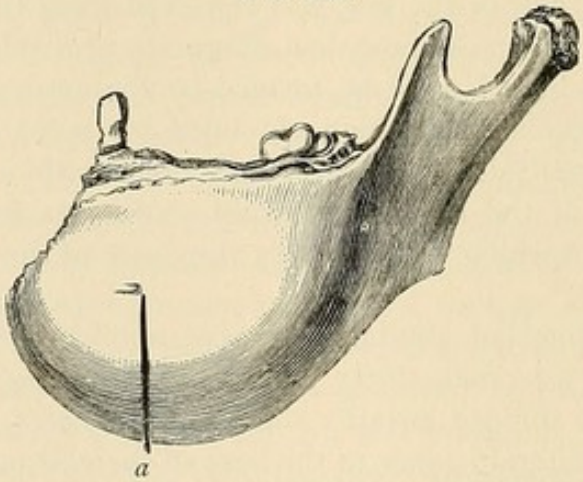
Miss Anna T., aged sixteen years, light complexion, well nourished, and in general good health, was referred to the writer by Professor John Van Duyn, of Syracuse University, November 21, 1881, for a tumor of the left inferior maxilla the size of a hen's egg in the

FIG. 201.



DENTIGEROUS CYST. (After Heath.)
a, unerupted cuspid tooth.

FIG. 202.



DENTIGEROUS CYST—LATERAL VIEW. (After Heath.)
a, mental foramen.

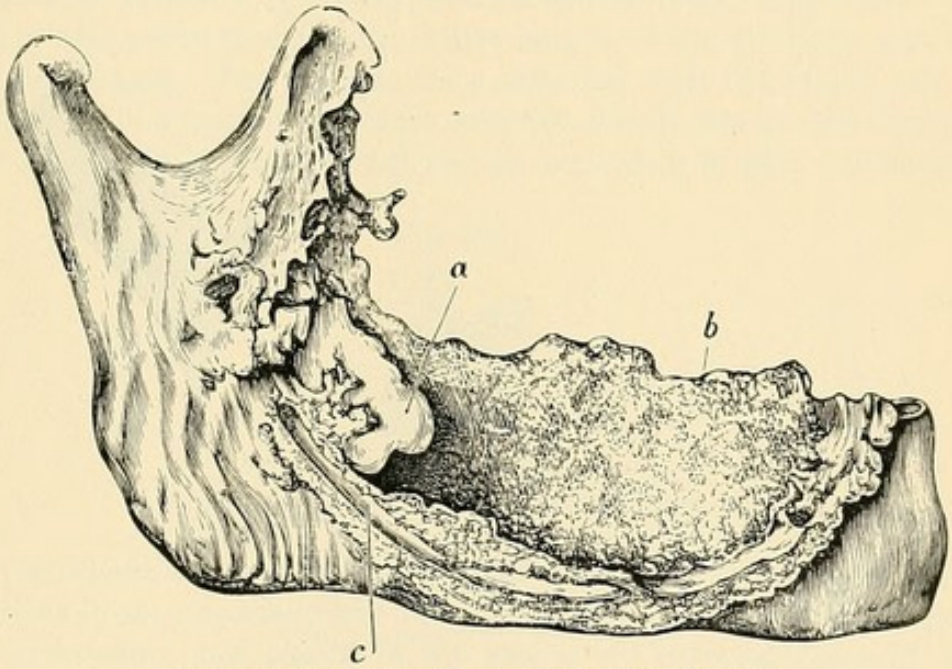
region of the molar teeth, and apparently involving the entire structure of the left half of the jaw, the swelling extending upward along the ramus toward the maxillary articulation.

The first molar tooth had been extracted about three months previously, under the mistaken diagnosis of an alveolar abscess. The

second molar was in place, but very loose; the third molar had not yet made its appearance. The swelling of the jaw, which was first noticed fifteen months before, had been of slow growth and painless, except on taking cold, when the pain was only slight and of short duration. Pressure over the tumor produced indentation of the tissues, with parchment-like crepitation, but there was no discoloration of the external integument. There was slight tenderness of the parts, and some difficulty in opening and closing the mouth, though mastication could still be performed upon the right side.

With the tumor of the jaw there existed a goitre of small size.

FIG. 203.



DENTIGEROUS CYST OF LOWER JAW. (After Forget.)
a, third molar inverted; *b*, internal wall; *c*, inferior dental canal.

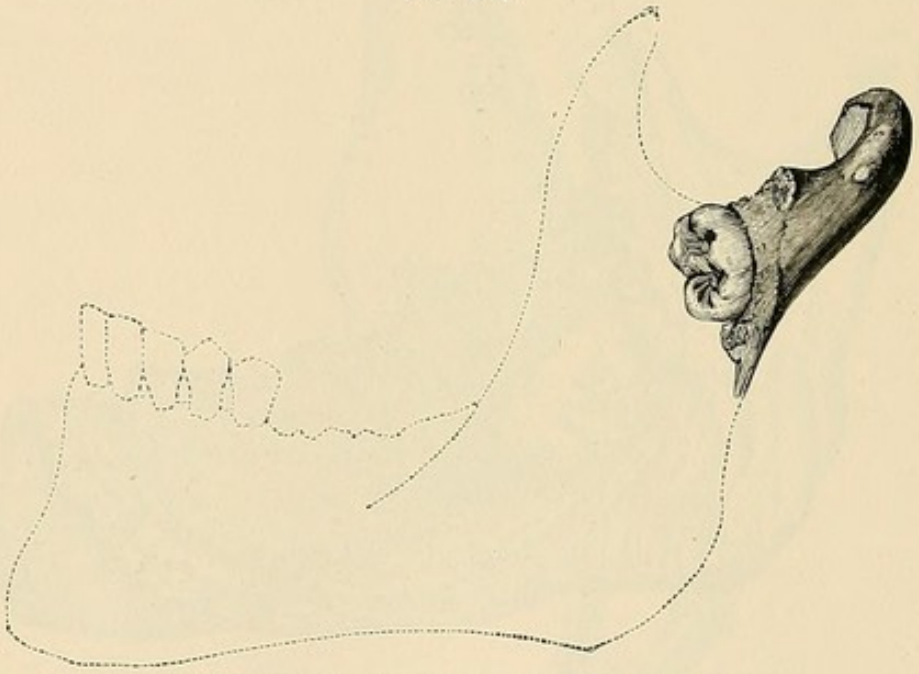
The contents of the tumor were found to be a straw-colored serum. This, with the fact of the location of the tumor, led to the diagnosis of dentigerous cyst, dependent, in all probability, upon the unerupted third molar.

The patient afterward sought other advice, and the second molar was extracted, "followed by the escape of a thick, tenacious fluid resembling the albumen of an egg." The patient returned a month later for operation. In the mean time the cyst had increased to about double its former size. The tumor was laid open within the mouth, along the alveolar border, from the angle to the second bicuspid tooth, and the fluid contents of the cyst removed, which consisted of a thick, dark-yellow serum, slightly mixed with pus, in quantity at least four ounces. On exploration with the finger introduced into the opening, several sharp spiculæ of bone could be felt upon the sides and bottom

of the cyst cavity,—probably the remains of the alveoli of the extracted teeth,—while in the posterior part of the cavity, well up toward the sigmoid notch and at the base of the condyloid process, the crown of the third molar could be distinctly outlined. This was easily dislodged with an elevator, and extracted by the aid of the bullet forceps.

On further examination it was found that the condyloid process and the posterior part of the ramus were separated from the coronoid process and the anterior portion, and detached from the surrounding tissues, the periosteum having been entirely separated from this portion of the bone; it was therefore removed through the cyst cavity and incision within the mouth.

FIG. 204.



DENTIGEROUS CYST, WITH INVERTED THIRD MOLAR.

The extracted condyle showed evidences of necrosis, and upon further examination it was discovered that the cyst had extended so far backward as nearly to sever the condyle from the coronoid process and body of the jaw, while the force applied to dislodge the tooth, though very moderate, no doubt completed the separation. The condyle was also separated from its fibro-cartilage. On placing the tooth in the crypt in which it was developed on the portion of the jaw removed (Fig. 204), it was found to have occupied an inverted position, the grinding surface of the crown directed downward, forward, and outward. The tooth was incomplete in development, the crown only being formed.

The patient made a good recovery, with reformation of lost bone and perfect mobility of the joint.

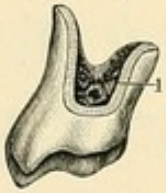
Mrs. Hulda A., aged twenty-nine years, farmer's wife, came for consultation on March 8, 1882, for a tumor of the right side of the

superior maxilla situated in the region of the bicuspid and first molar teeth, occupying the alveolar ridge, and about the size of a pigeon's egg, firm and unyielding to the touch.

Six months previously she had the second bicuspid of the right side extracted, which was badly decayed. The first bicuspid and first molar had been lost for some years; had not noticed the enlargement of the jaw until some weeks after the second bicuspid had been extracted. The formation of the tumor had been slow and painless, and she only sought advice on account of its becoming troublesome from its size and the disfigurement of the face.

The tumor was punctured with a heavy exploring needle, which revealed the presence of a clear, straw-colored, thick, ropy fluid. An opening was made through the entire length of the swelling, and the fluid discharged. The sac was then explored with the finger, and at the upper part a jagged substance was felt, which was at first thought to be a piece of denuded bone; but on removing it with the forceps,

FIG. 205.



FIRST TEMPORARY MOLAR (enlarged).
1, pulp-chamber opened and enlarged by resorption.

after considerable exertion, it was found to be a first deciduous molar of perfect form, except for the loss of a portion of the root, evidently from resorption, and placed in the jaw in an inverted position, the roots pointing downward. Resorption had been most active upon the distal aspect of the roots, and had penetrated the pulp-chamber, which was exceedingly large. The tooth was somewhat discolored, and had the appearance of having been bathed in a yellowish-brown staining fluid for a considerable period. (Fig. 205.)

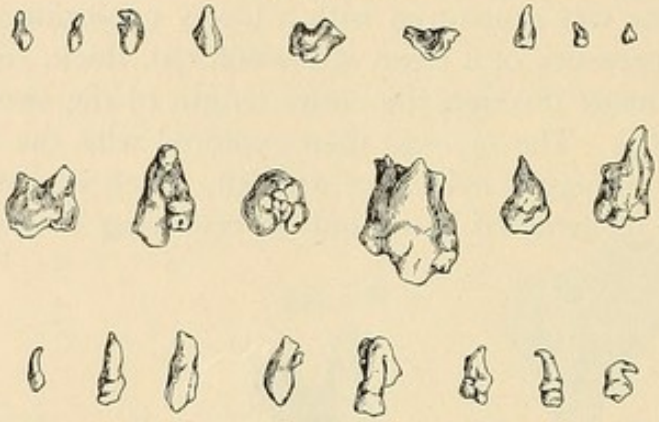
Mr. B., aged twenty-four years, a medical student, consulted the writer, during the winter of 1886, for a tumor in the region of the maxillary tuberosity of the right side, behind the third molar, but which was only partially erupted.

The gum-tissue was considerably swollen, and the jaw much enlarged and broadened at this point. There was a slight fullness under the malar bone, but no crepitation or fluctuation could be detected, though severe pain was at times experienced, and the tissues were painful to touch. A diagnosis of impacted or difficult eruption of the third molar being made, the tooth was cut down upon and removed, the removal being followed by a discharge of a watery fluid mixed

with pus, and of offensive odor. On probing the alveolus from which the tooth had just been extracted, enamel could be felt, and upon enlarging the alveolus with the bone-forceps five small supernumerary teeth were removed, of imperfect form, resembling the canines of a kitten.

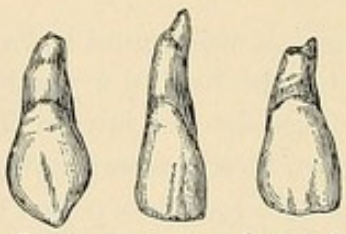
Several cases of this character are to be found on record. Fig. 206 shows a group of dwarfed and malformed teeth removed from a cyst by Tomes, and Fig. 207 shows three teeth removed from a cyst by Cryer.

FIG. 206.



DWARFED AND MALFORMED TEETH FROM A DENTIGEROUS CYST OF THE RIGHT SIDE OF THE UPPER JAW. (After John and Charles S. Tomes.)

FIG. 207.



RIGHT UPPER INCISORS AND CUSPID FOUND IN A CYSTIC CAVITY. (After M. H. Cryer.)

Diagnosis and Symptoms.—The question of age is one that should be considered in making the diagnosis. The disease has occurred as late as the sixtieth year, but the great majority of cases are under thirty years. The disease is essentially one of early life.

The symptoms of a dentigerous cyst are principally local in their manifestations, and consist of expansion of the maxillary bone in some definite locality, with a corresponding deformity of the features, a sense of weight and tension, sometimes pain and general constitutional disturbance.

Pressure over the tumor produces a peculiar parchment-like crepitation, and when the bone is sufficiently thinned, fluctuation may be detected.

The most important diagnostic sign is the absence of one or more

teeth which should have made their appearance in the mouth, and which have not been extracted. The teeth corresponding with the age of the patient, however, may all be in position or accounted for, and yet the fact that a supernumerary tooth may be the cause of the disease must not be overlooked. Impacted temporary teeth are very rarely met with, and the formation of a dentigerous cyst from this cause is even more rare.

Fluid may be detected by the exploring needle, the trocar, or by aspiration. This should never be neglected in a doubtful case, in order that a needlessly severe operation may be avoided. The presence of a serous fluid should be additional evidence in favor of the diagnosis of a tooth-bearing cyst. The discovery of a tooth in the cyst may often be made by the introduction of a probe passed through an incision made in the cyst-wall.

The character of the fluid in dentigerous cysts is usually a clear, yellowish, albuminous, viscid, ropy serum; occasionally it becomes puriform when the cyst has been the seat of a suppurative inflammation or other changes.

The subsequent changes which may take place in dentigerous cysts are the same as occur in cysts in other locations of the body, and may lead to a mistaken diagnosis. The character of their contents is subject to considerable deviation. A trauma may induce a hemorrhage into the cavity, and the contents of the cyst be so mixed with blood as to be mistaken for an extravasation cyst; or colloid degeneration may convert the fluid to a honey-like liquid; or calcification may occur, in floating particles of coagulated fibrin or blood-clots, while the cyst membrane may be so filled with calcareous deposits as to lead to the supposition that the tumor was an osseous growth. Heath describes a case of this character. The cyst was located in the right antrum, but had no attachment to the walls of the sinus except to its floor. Complete calcification had taken place, and upon opening the cyst a supernumerary tooth was found loose in the cavity, but which evidently had been attached originally to its base.

Differential Diagnosis.—Cysts of the maxillary bones may be confounded with other affections of this locality in which swelling or enlargement of the bone is a prominent symptom.

Dentigerous cysts are commonly located in the body of the bone, and usually attain a considerable size, while cysts associated with the roots of devitalized teeth are usually confined to the alveolar process, are of much smaller size, and do not cause expansion of both plates of the bone.

The fibromata, chondromata, sarcomata, myxomata, abscesses, and empyema of the antrum all cause swelling and enlargement of the parts, and consequently errors have been made in diagnosis which

have occasionally led to serious consequences. An abscess, however, can be distinguished by its rapid course and constitutional symptoms, and an empyema usually by the more symmetrical enlargement of the facial surface of the jaw. Tumors occupying the maxillary sinus sometimes produce the same even enlargement, but an exploring needle passed into the tumor will usually demonstrate its character in a general way. If the contents are fluid, the needle will meet with little resistance, while the liquid will escape from the puncture made by it. On the other hand, a solid tumor will offer considerable resistance to the passage of the needle; the more dense the tumor, the greater will be the resistance.

Prognosis.—A dentigerous cyst is usually a curable disease, and the deformity occasioned by the expansion of the bone is one which gradually passes away after the removal of the exciting cause. Occasionally, however, the deformity may persist, even after the disease has been cured. Cysts of the jaws are not themselves liable to cause serious results, but secondary complications may arise, like suppuration followed by septic fever, in which there would be more or less danger of a fatal termination.

When properly treated, the issue of these cases is almost universally satisfactory. They are very rarely associated with other bone-disease.

Treatment.—The requirements of any treatment, to be effective, must comprise not only the evacuation of the cyst, but the removal of the exciting cause of the disease. This may require, in some cases, an extended operation, but with skill it may be confined to the mouth. The writer cannot conceive of a condition of cyst of the jaws that may not be better and more successfully treated through the mouth than by incisions through the external tissues of the face, except where exsection of the body of the jaw is required.

The operation usually practiced is to open the cyst freely, and then for cosmotic reasons to cut away, with bone-forceps, gouge, or burs, the external plate of the expanded bone. Some operators prefer to crush in the external wall instead of cutting it away; but in many cases this is not necessary, as sooner or later resorption will restore the normal outline of the jaw. After the fluid has been evacuated, a careful search must be made for the offending tooth, but such effort does not always prove successful. It is certain, however, that it is somewhere in communication with the cyst, either covered by the cyst membrane or imbedded in a crypt and communicating with the cyst through a small opening.

In large cysts with thin external wall the expanded bone may be crushed down, and afterward the cyst cavity packed with gauze and permitted to close by granulation.

In some instances there will be a persistent reaccumulation of serous fluid. Under such circumstances it may become necessary either to inject the cyst with astringent and stimulating fluids, or to reopen it, and, by means of a curette, thoroughly destroy the investing membrane.

REVIEW.

CHAPTER XLVII.

To what form of tumor is the term dentigerous cyst applied?

With what class of teeth are they usually associated?

In which jaw do they most frequently occur?

What forms of aberration in the development of the teeth most often produce the disease?

Are aberrations of this character likely to cause cystic tumors?

Are aberrant teeth which pierce the gums productive of cystic tumors?

Under what conditions are cysts formed in connection with the temporary teeth?

What is the explanation of their formation?

What form of irritation is productive of serous exudations?

What form of irritation seems to be the prominent factor in the production of dentigerous cysts?

What changes take place in the contents of these cysts?

In what other locations beside the jaws are tooth-bearing cysts found?

How are these classed?

How are dermoid cysts developed?

What is the most common location of dermoid cysts?

At what period of life are they most common?

What is the character of their growth, etc.?

Where are the simple forms most often found?

With what form of tissue are tooth-bearing cysts always associated?

What is the explanation of their formation in these localities?

From what embryonic tissue are the teeth formed?

How is the presence of teeth in the cervical region explained?

What question should be especially considered in making a diagnosis of dentigerous cysts?

At what period of life are they most common?

What are the symptoms?

What is the most important diagnostic sign?

What relation do the temporary teeth bear to the formation of dentigerous cysts?

How may the fluid be detected?

What would the presence of serous fluid indicate?

How may the presence of a tooth sometimes be demonstrated?

What is the character and appearance of the fluid found in dentigerous cysts?

What are the subsequent changes that may take place in the fluid?

What conditions are to be noticed in the differential diagnosis?

What other diseases may be mistaken for dentigerous cysts?

How may abscess and empyema be differentiated from dentigerous cyst?

How may tumors be differentiated?

What is the prognosis of dentigerous cyst?

Is the deformity always corrected by the cure of the cyst?

What are the complications which might cause danger of a fatal termination?

What is the usual issue in these cases after operation?

What are the requirements of any treatment that would be effectual?

When should these cysts be opened?

What condition would make it necessary to operate through the external tissues of the face?

Describe the operation of treating a dentigerous cyst.

If the offending tooth is not readily found, where is it likely to be?

What should be done with the walls of the cyst?

What should be the treatment when there is a reaccumulation of the serous fluid?

CHAPTER XLVIII.

CARCINOMATA.

Definition.—Carcinoma (from the Greek καρκινος, a crab, and ωμα, a tumor).

(Carcinoma is a malignant neoplasm of the epiblastic group of tumors, and is formed by an active proliferation of epithelial cells from a matrix of embryonic cells, usually of congenital origin.)

Carcinoma is of all diseases the one upon which the surgeon looks with the greatest dread and apprehension. It is a disease which for centuries has baffled the most earnest search for its cause, and rendered the highest degree of surgical skill of little benefit as a curative measure, though some little progress has been made during the last two or three decades in certain lines of investigation which have had for their object the tracing of the disease to its histogenetic origin. More recently—during the last decade—great interest has been awakened in the etiology of carcinoma, from the investigations of the bacteriologists who have endeavored to prove the bacterial origin of the disease. Up to the present time, however, the true cause of carcinoma has not been discovered, but there would seem to be reason to hope that eventually such cause will be found, and in all probability through the researches of the bacteriologist.

Investigations looking to the discovery of therapeutic measures for the constitutional treatment of carcinoma have likewise engaged the attention of many of the best minds in the profession, but so far no remedy has been found which has the slightest permanent value as a curative agent.

It is to be hoped, however, that with the discovery of the real cause of carcinoma will come the therapeutic remedy which will not only successfully combat its ravages, but will also furnish the means of prevention.

Origin.—Carcinoma belongs to the epithelial group of tumors. The generally accepted teaching in relation to the origin of all forms of *carcinoma* is the theory of Cohnheim, viz: *That the disease arises from a misplaced matrix of embryonic epithelial cells of congenital origin.*

Senn and others believe that such tumors may also arise from a

proliferation of embryonic cells of post-natal origin, as the result of various forms of injury or disease which may produce a displacement of the epithelial cells into tissues where they do not normally belong, thus forming a tumor-matrix from which a carcinoma may ultimately develop.

Carcinoma may be described as an *atypical* proliferation of epithelial cells (Waldeyer). (The term atypical means irregular, not conformable to the type; the opposite of typical.) Primary carcinomatous growths are usually found associated with tissues like the skin, mucous membrane, or glandular structures, and particularly in those glands having ducts which communicate with the external surfaces of the body, or with canals having such communications.

A typical epithelial tumor is one in which the epithelial elements remain within their normal boundaries; do not break through the *membrana propria* and encroach upon the connective tissue. The basement membrane, or *membrana propria,* forms the dividing line or boundary between the epithelial cells and the connective tissue. A true adenoma is an epithelial tumor of this type. An atypical epithelial tumor is one in which the new epithelial cells break through the physiologic boundaries and extend into the connective tissue. Carcinoma is an epithelial tumor of this type. In other words, a typical epithelial tumor is formed by a proliferation of epithelial cells within epithelial tissue and in a normal location, while an atypical epithelial tumor is formed by the proliferation of epithelial cells within a tissue of a different type, and in a location where they do not properly belong.

In the consideration of primary carcinoma in unusual locations, the possibility of a post-natal origin from traumatisms or disease which may cause a displacement of embryonic epithelial cells must not be overlooked.

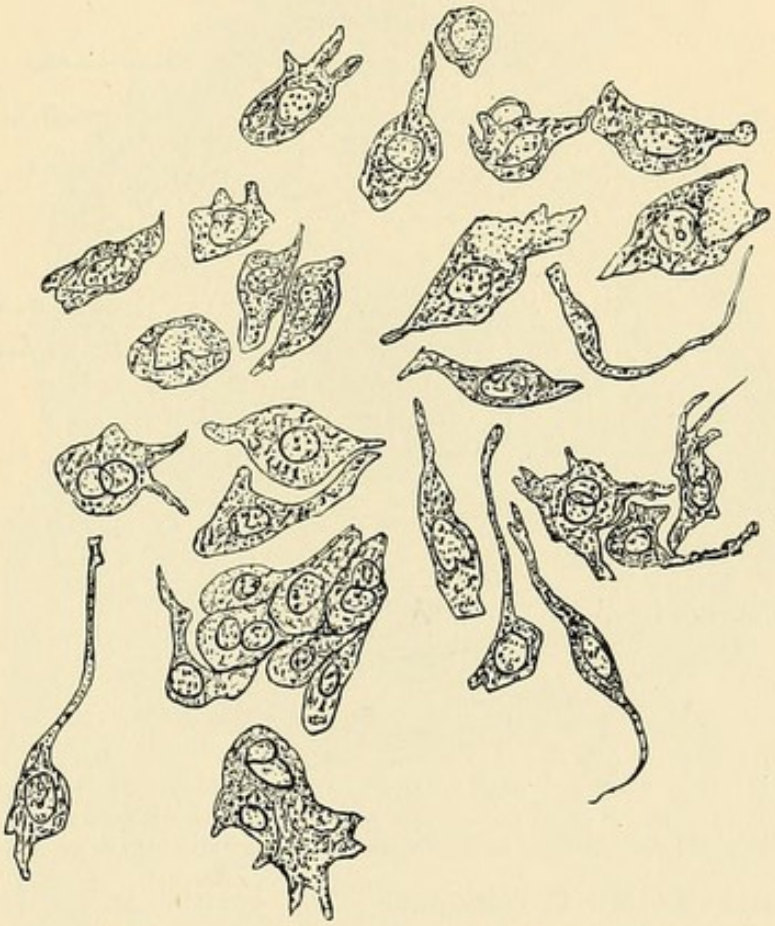
Varieties and Structure.—Histologically, all carcinomatous tumors, of whatever form, are composed of epithelial cells, grouped and arranged in a characteristic order, in an alveolated connective-tissue stroma. The epithelial cells have their origin in a pre-existing tumor-matrix, while the stroma is derived from the connective tissue in which the essential matrix has been implanted, or into which the proliferating epithelial cells afterward migrate. The proliferation or multiplication of the epithelial cells (carcinoma-cells) of these growths is by karyokinesis (Filbry). The embryonic character of the epithelial cells is maintained throughout their entire development, which condition of the cells marks the difference between the benign and the malignant types of epithelial tumors.

The chief differences in structure which exist between the various forms of carcinoma—epithelioma, scirrhus, encephaloid, colloid, and

glandular carcinoma—arise from their location, the type of epithelial cells of which they are composed, or the form and degree of the degenerative changes which take place in them. Histologically, carcinoma may be divided into three forms: the squamous-celled, the cylindrical-celled, and the glandular.

Epithelioma is a term which has been used to designate carcinoma of the skin and mucous membrane, regardless of whether it originated in the rete Malpighii or the glandular appendages of these tissues.

FIG. 208.



CELLS FROM AN EPITHELIAL CARCINOMA OF THE BLADDER. × 250. (After Perls.)

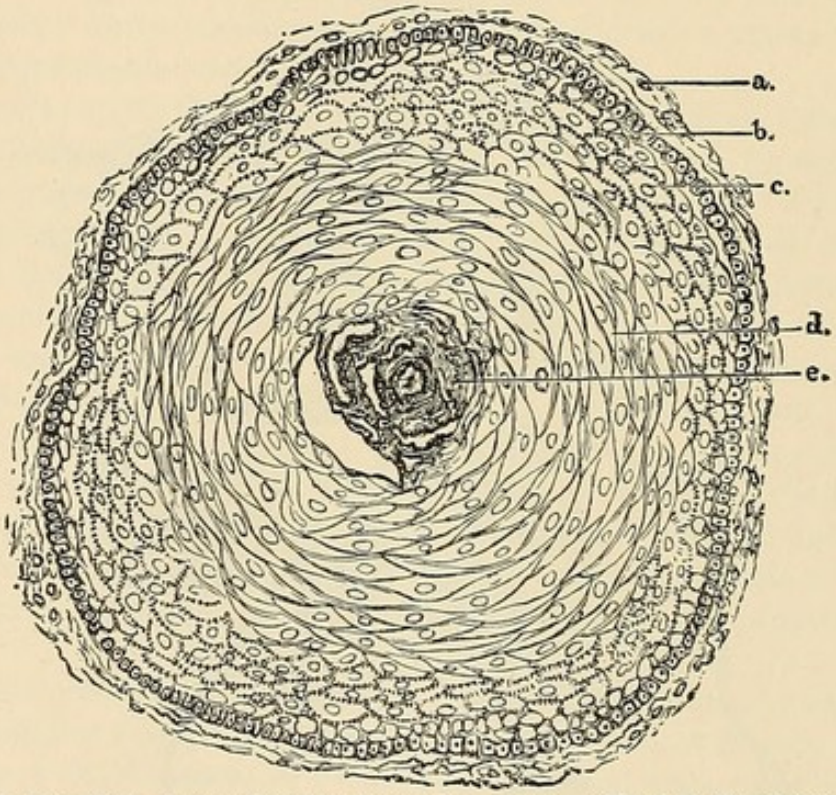
The carcinoma-cells indicate their epithelial origin by their peculiar form. (Fig. 208.) These cells are large, of varying sizes and shapes, containing one or more round or oval nuclei with large, glistening nucleoli (Warren).

The stroma is composed of fibrous tissue, more or less infiltrated with small, round cells, and traversed by blood-vessels. The stroma may be abundant or scanty. In the slow-growing, hard forms of carcinoma the stroma is abundant, while in the rapid-growing, soft variety it is scanty.

The blood-vessels and lymphatics are located in the stroma,

through which they ramify in all directions. The blood-vessels, unlike those in sarcoma, are normal in thickness and construction, and are confined in their ramifications to the stroma, while in sarcoma they pass among the cells. The blood-supply is always increased in the development of carcinoma. The general plan of the histologic structure of all forms of carcinoma is the same, but each variety has some peculiar characteristic in the form and arrangement of its cells which distinguishes it from the others. These may be briefly described as follows:

FIG. 209.



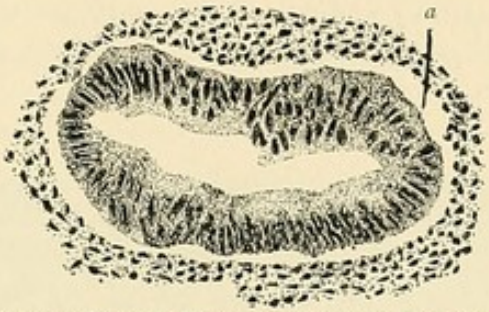
A CELL-NEST FROM A CANCER OF THE LIP. × 300. (After J. D. Hamilton.)

a, stroma of the alveolus in which the cell-nest is contained; *b*, small germinal cells of the periphery; *c*, prickle-cells; *d*, compressed squamous cells; *e*, degenerated cells in the center.

Squamous-Celled Carcinoma.—This variety is located in the skin, the *squamous* epithelial cells of which are arranged in concentric layers within the alveoli, forming what are known as *"cell-nests,"* "cancer-nests," or "epithelial pearls." The oldest cells are found in the center of the nest, the young cells at the periphery. (Fig. 209.) Carcinoma grows or extends by the migration of the cancer-cells—which possess an amœboid movement—into the connective-tissue spaces. Each cell possesses the power of multiplication, and thus new colonies are formed. With the growth of the colony there is a separation of the connective-tissue fibers, resulting in the formation of an alveolus. This process goes on indefinitely, with greater or less rapidity, according to the resistive power of the tissues. This form of the disease is usually described under the term "epithelioma."

Cylindrical-Celled Carcinoma.—This form of the disease is found in the mucous membrane, the columnar or cylindrical cells of which are arranged in the form of tubules, simulating the structure of tubular

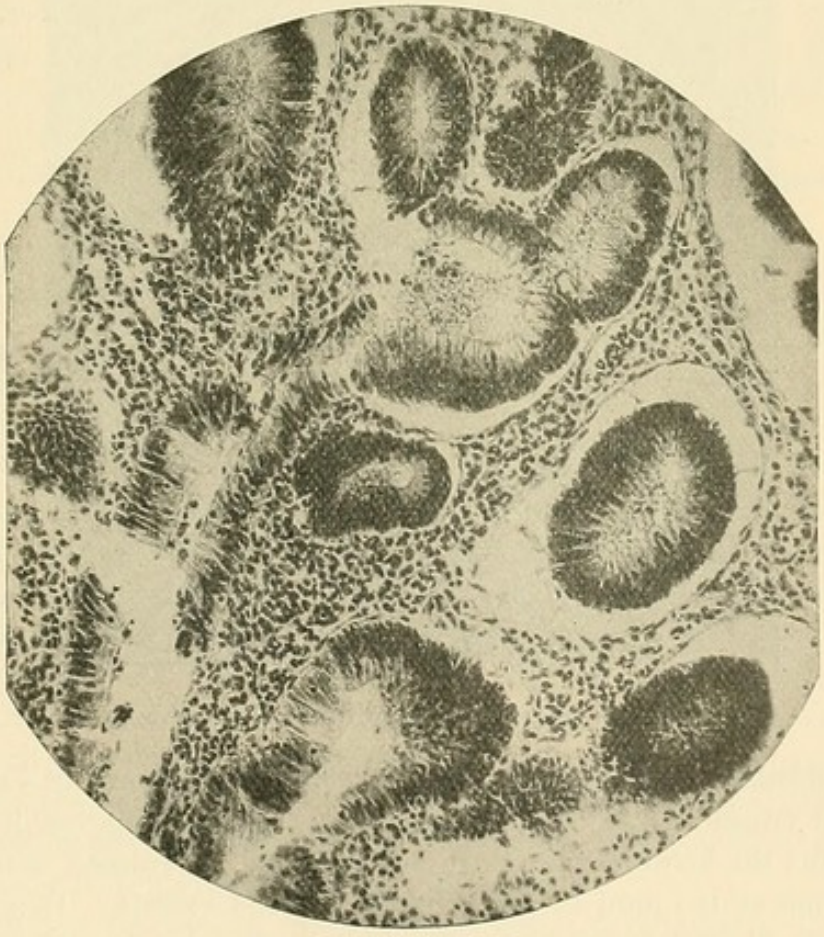
FIG. 210.



CARCINOMA OF THE RECTUM—A SINGLE TUBULE—SHOWING MULTIPLICATION OF CELLS IN ITS LINING. × 170. (After Senn.)

a, space due to shrinkage in hardening.

FIG. 211.



EPITHELIOMA—CYLINDRICAL—OF STOMACH. × 200.

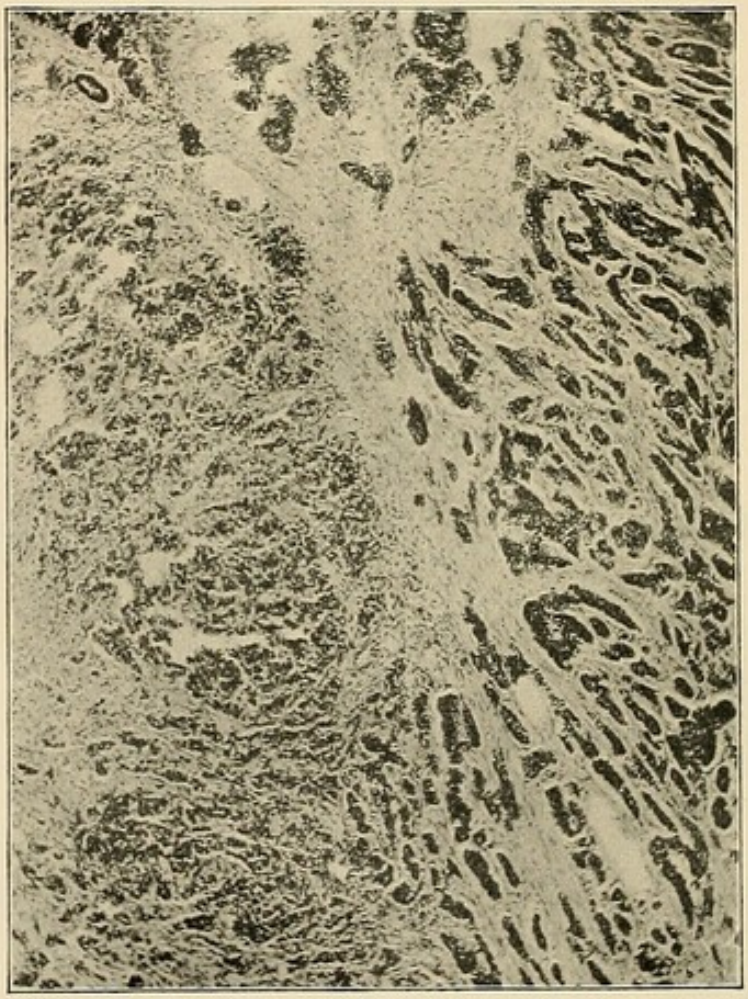
glands. This tubular structure corresponds to the cell-nests of the squamous-celled variety of the disease. The arrangement of the columnar cells does not follow the typical form of a tubular gland, but

forms an irregular lining of the crypts. In this respect it differs from adenoma, in which the typical form of the tubules is present. (Figs. 210, 211.)

Infiltration of the stroma with leucocytes and young carcinoma-cells is a common condition. This indicates its tendency to rapid growth and malignancy. Mucoid and colloid degeneration of the cylindrical cells and stroma frequently attends this form of carcinoma.

FIG. 212.

More degenerate growth and medullary in character.



Typical stroma and cells. Scirrhous type.

CARCINOMA—SCIRRHUS—FROM TONGUE OF COW. × 40.

Glandular Carcinoma.—The location of this form of carcinoma is in the conglomerate glands, like the mammæ, kidneys, liver, testicles, etc., the acini of which present the same alveolated structure of the stroma as is found in the squamous-celled variety. In glandular carcinoma the acini of the gland constitute the alveoli, while the connective tissue between them forms the stroma. The size of the alveoli and the amount of the stroma are governed by the character of the growth and the degree of malignancy.

In the hard, slow-growing varieties the alveoli are small and the

stroma abundant, while in the rapid-growing, soft varieties the stroma is scanty and the alveoli large. It may therefore be stated as a well-established fact that in proportion as the alveoli are large and the amount of connective tissue which makes up the stroma or reticulum is small, so will be the degree of malignancy of the disease.

The term *scirrhus* cancer has been applied to the hard, slow-growing variety of carcinoma. This form of the disease is found most frequently in the mammary glands; it is also occasionally found in the stomach, testis, ovary, pancreas, and kidney. (Fig. 212.)

To the soft, rapidly-growing variety the term *encephaloid* cancer has been applied, from its resemblance to brain-tissue. It is most frequently found in the mucous membrane, the liver, testis, bladder, kidney, ovary, fundus oculi, and occasionally in the breast.

The epithelial cells in both these varieties are spheroidal. This is due to the fact that the epithelium in which the neoplasm originates is spheroidal rather than columnar.

Retrograde changes in cancerous growths frequently take place in the cells and stroma at a very early period in their history.

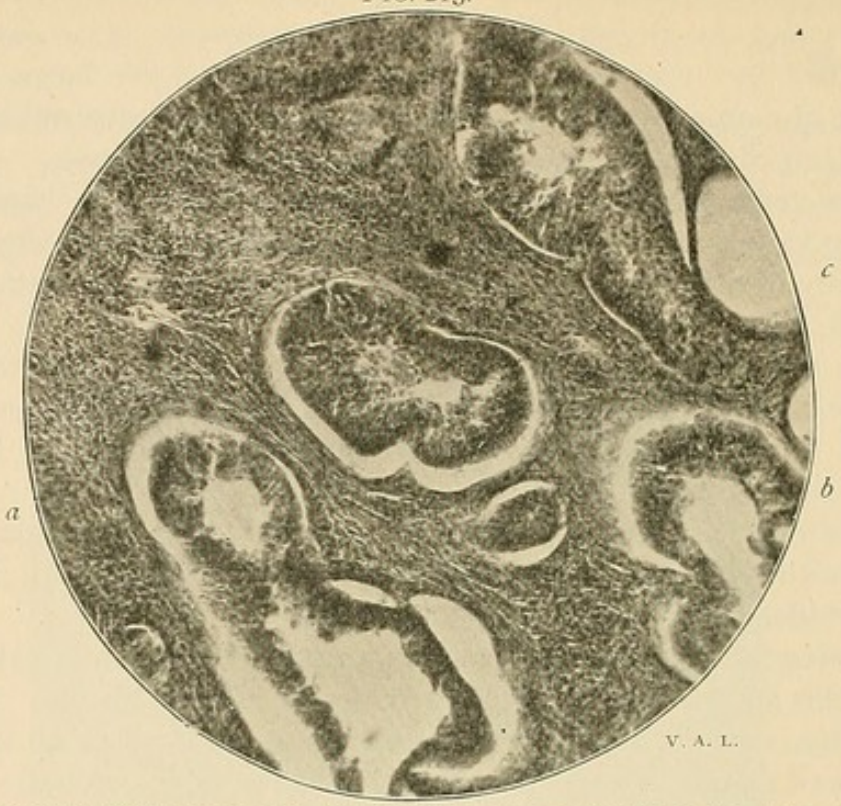
Fatty, mucoid, and colloid degenerations are prone to occur in the cells of glandular carcinoma. Calcification is occasionally seen in cancers of feeble growth. To these degenerative changes is due the confusion which has arisen in the classification of carcinomatous growths.

A very rare and peculiar form of carcinoma is a variety known as *Cylindroma carcinomatodes*. This variety of the disease is a species of colloid degeneration occurring primarily in glandular structures. Ziegler states that he had observed but one case, and this was associated with the lachrymal gland. Histologically it is "characterized by the formation of homogeneous spherules within the cell-nests. These spherules are generally regarded as masses of colloid substance which press asunder the other cells of the group. If a considerable number of these spherules form within the same loculus, the cells may be compressed into slender trabeculæ, and so come to form a kind of anastomosing net-work." (Ziegler.) Fig. 213 shows a primary growth of this character which was located in the stomach, most of which was in a state of colloid degeneration, and Figs. 214 and 215 metastatic growths in the liver and the brain from the same case.

Infection and Dissemination.—All true carcinomatous growths are clinically of a highly malignant character.

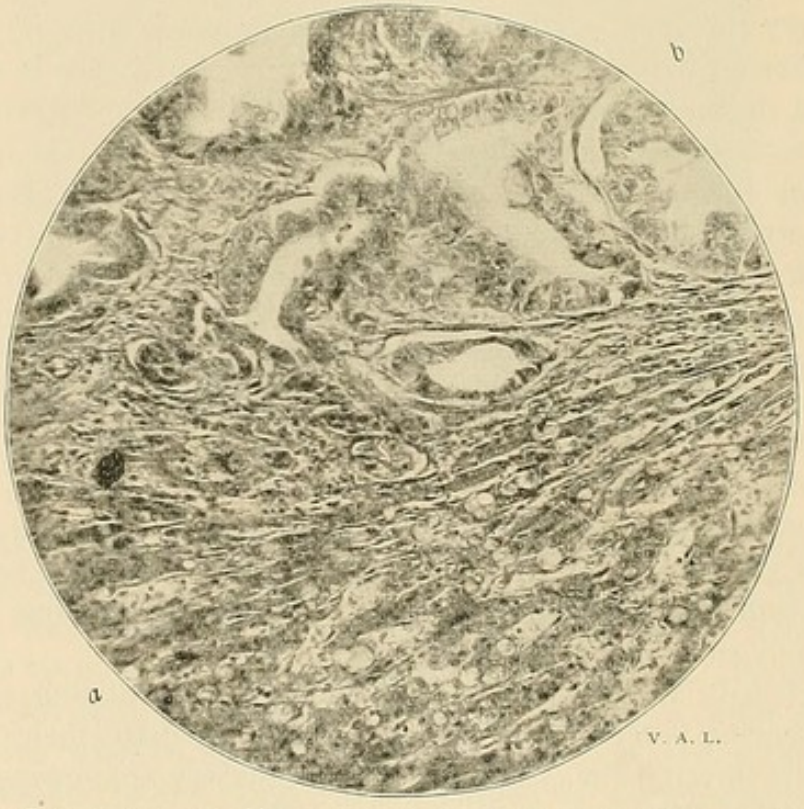
Progressive infection of the immediately surrounding tissue, of the neighboring anatomic structures, and general dissemination of the cancer-cells through the lymphatic and circulatory apparatuses mark the malignant character of carcinoma. These conditions are made manifest in the progressive growth of the tumor, in the involvement of

FIG. 213.



CYLINDROMA CARCINOMATODES. PRIMARY GROWTH OF THE STOMACH. × 75. (A.)
a, stroma with round-celled infiltration; *b*, carcinoma cells; *c*, alveoli or acini.

FIG. 214.



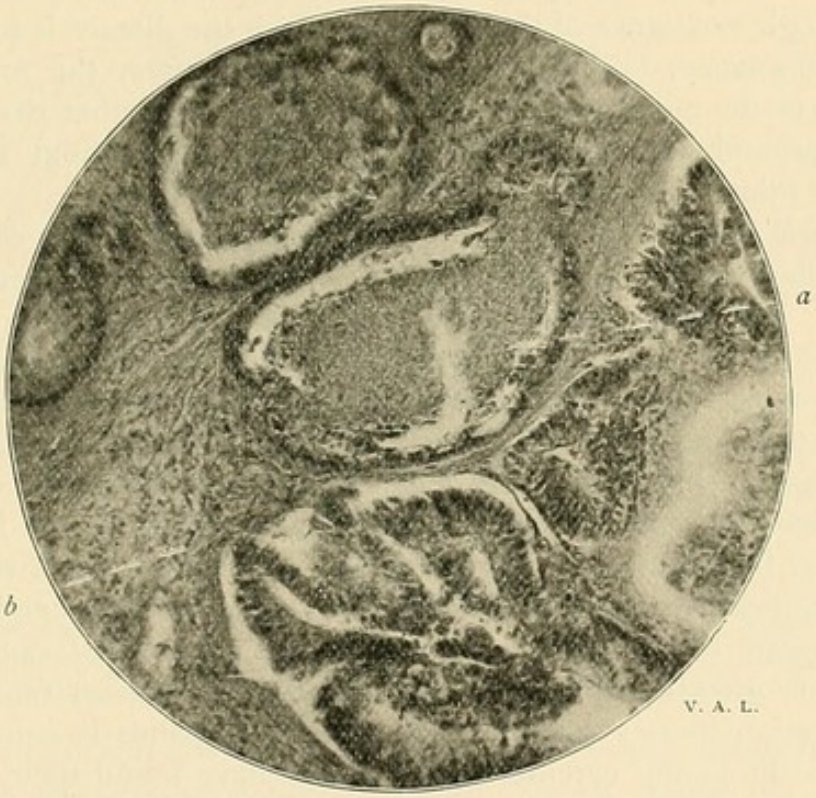
CYLINDROMA CARCINOMATODES. METASTATIC OF THE LIVER, FROM A PRIMARY GROWTH IN THE STOMACH. × 75. (B.)
a, fatty degeneration of liver-cells; *b*, carcinoma.

the neighboring healthy tissues, regardless of the character of these structures, and in the formation of secondary carcinoma in distant organs and tissues which have no anatomic relationship to the tissues which are the seat of the primary disease.

The local extension of carcinoma to all tissues and organs, irrespective of their structure, is the most marked feature in the pathology and the clinical history of the disease, and may be considered almost a pathognomonic sign.

The use of the term *infection,* as applied to tumors of malignant type, rests upon the power possessed by the "cancer-cells" to leave the

FIG. 215.



CYLINDROMA CARCINOMATODES. METASTATIC OF THE BRAIN, FROM A PRIMARY GROWTH IN THE STOMACH. (C.)

a, degeneration (colloid); *b*, brain stroma.

primary tumor and to wander into the surrounding healthy tissue, thus establishing new centers of growth; or by being transplanted through the lymphatic channels or by the blood-current, to reproduce the disease in contiguous regions or in distant parts of the body (Senn).

This power to migrate and multiply in the mesoblastic tissues which is possessed by the cancer-cells does not explain the malignancy of such growths.

Waldeyer and Thiersch both observed and described the power of epithelial cells to penetrate into tissues of apparently healthy character, but "normal epithelial cells do not possess the same power of

multiplication in the mesoblastic tissues as do the epithelial cells of carcinoma" (Senn). It may therefore be stated with some degree of certainty that some change, at present not understood, takes place in the epithelial cells, which increases to a marked degree their power of multiplication, while at the same time the resistive powers of the tissues in which they are implanted are more or less lowered. These changes may be due to the influence of a specific micro-organism which has gained access to the tissues, but proof upon this point has not been demonstrated. If this is a correct view of the conditions, it may be readily understood how the growth and progress of carcinoma on the one hand may be rapid, while upon the other it is slow, these conditions depending upon the rapidity of cell-proliferation and the physiologic resistance of the tissues in which the disease is found.

Malignancy, however, depends not only upon the progressive growth of the neoplasm, but upon the infection of other tissues in its neighborhood and its general dissemination throughout the body, causing numerous other centers of cancerous growth.

Local infection takes place by the migration of young carcinomatous cells from the periphery of the tumor into the connective-tissue spaces. The progressive extension of the growth is always in the direction of the pre-existing connective-tissue spaces, consequently it spreads most rapidly and attains its largest dimensions when located in regions supplying an abundance of loose connective tissue.

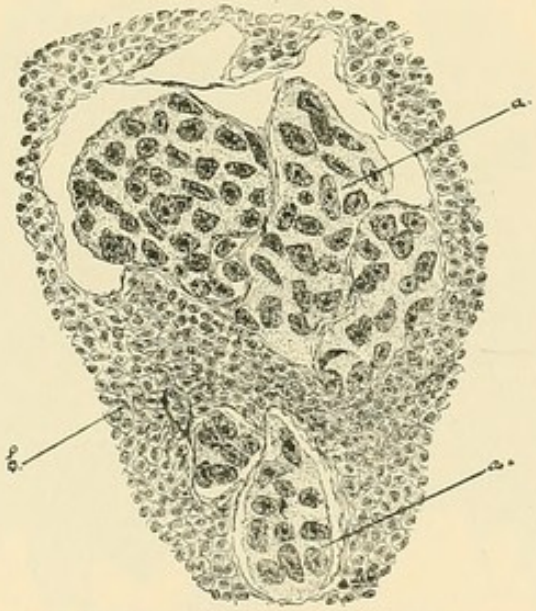
Regional infection is the result of the transplantation of pathogenic material from the seat of the primary tumor to the lymphatic glands of the region, through the lymph-channels. It is a well-established clinical fact that the lymphatics in the immediate neighborhood of a primary carcinoma sooner or later become affected, and that the secondary growth is in all respects similar to the primary tumor. The pathogenic material carried to the lymphatic glands is generally believed to be young carcinoma-cells which have found their way into the lymph-channels and have been carried by the lymph stream and deposited in the glands. The glands, acting as filters, arrest the further progress of the cancer-cells, and new foci are established for the development of the disease in these structures. (Fig. 216.)

Paget was of the opinion that even minute portions of the protoplasm of the carcinoma-cells were as effective in reproducing the disease as the whole cells. Gussenbauer believed secondary carcinoma to be due to the transplantation of minute infective corpuscular elements from the primary tumor to the lymphatic glands through the lymph channels.

General infection is expressed in the development of carcinomatous tumors in tissues and organs in distant locations of the body which have no anatomic relationship with that portion of the body in

which the primary tumor is located. The dissemination of the infective material is brought about through its entrance into the general circulation. This may be accomplished either by direct entrance to the blood-current through the perforation or injury of a vein-wall, or by indirect entrance through the lymphatic system. Such material is capable of passing through the pulmonary circulation, and of being carried to remote portions of the body, where it may become arrested in the capillaries, forming an embolus from which a carcinomatous growth may develop. This process of dissemination is termed metastasis, and the tumors developed from such process metastatic tumors.

FIG. 216.



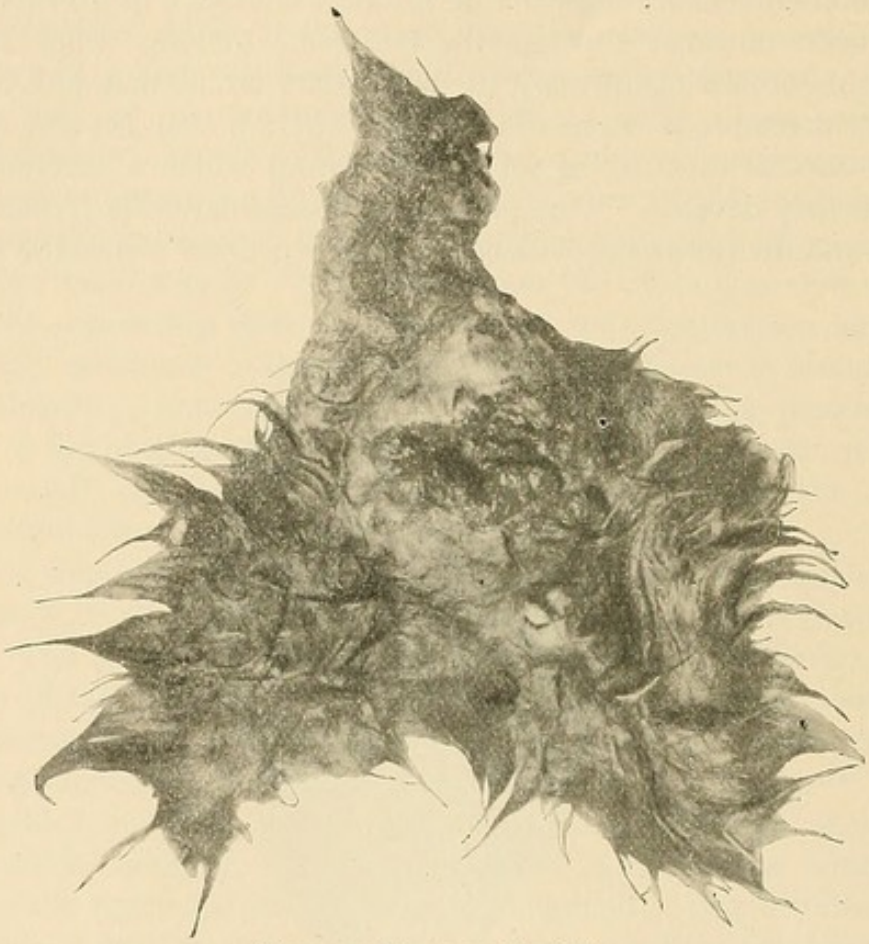
SECONDARY CARCINOMA OF LYMPHATIC GLAND. × 480, reduced one-third. (After Senn.)
a,*a*, groups of carcinoma-cells; *b*, lymphoid corpuscles and reticulum. Each one of the epithelial nests is the product of tissue-proliferation of a single carcinoma cell.

The size of the carcinomatous emboli will in large measure determine the location of the metastatic tumor (Senn). Large emboli may become lodged in the pulmonary artery, while the minute ones pass the pulmonary capillaries and enter the arterial circulation, where they may become attached to the walls of the vessels or pass on until they form emboli in vessels too small to admit them to pass. In this way only the presence of metastatic carcinoma in locations which have no anatomic connections with the seat of the primary disease can be explained.

The organs which are most frequently the seat of metastatic carcinoma are the lungs and the liver. The bone is occasionally the seat of secondary carcinoma, and Wagner has collected fifteen cases of this form of the affection in the choroid. Metastatic carcinoma of the lungs sometimes becomes a supply station from which the entire body may become infected with "miliary carcinosis." This form of the dis-

ease closely resembles miliary tuberculosis in its appearance, and is a rapidly fatal form of the disease. Figs. 217, 218, 219, 220, 221, show

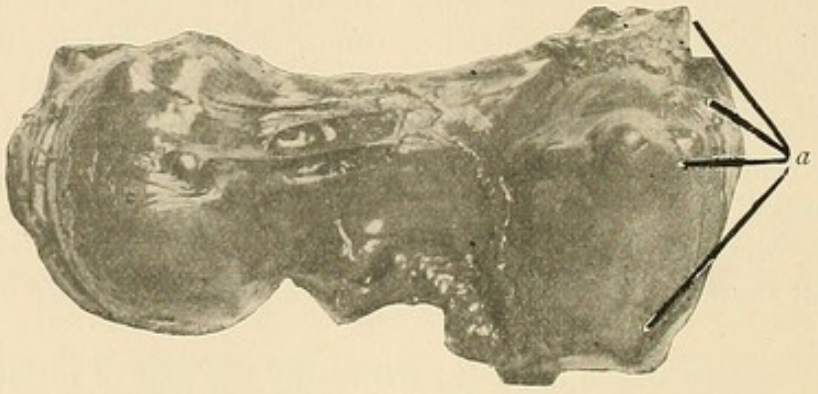
FIG. 217.



MAMMARY GLAND OF CAT (A).

Carcinoma—Scirrhus—Primary growth. Metastatic nodules appeared in the kidneys, intestines, lungs, heart, and spleen.

FIG. 218.



KIDNEYS OF CAT (B).

a, metastatic carcinoma nodules.

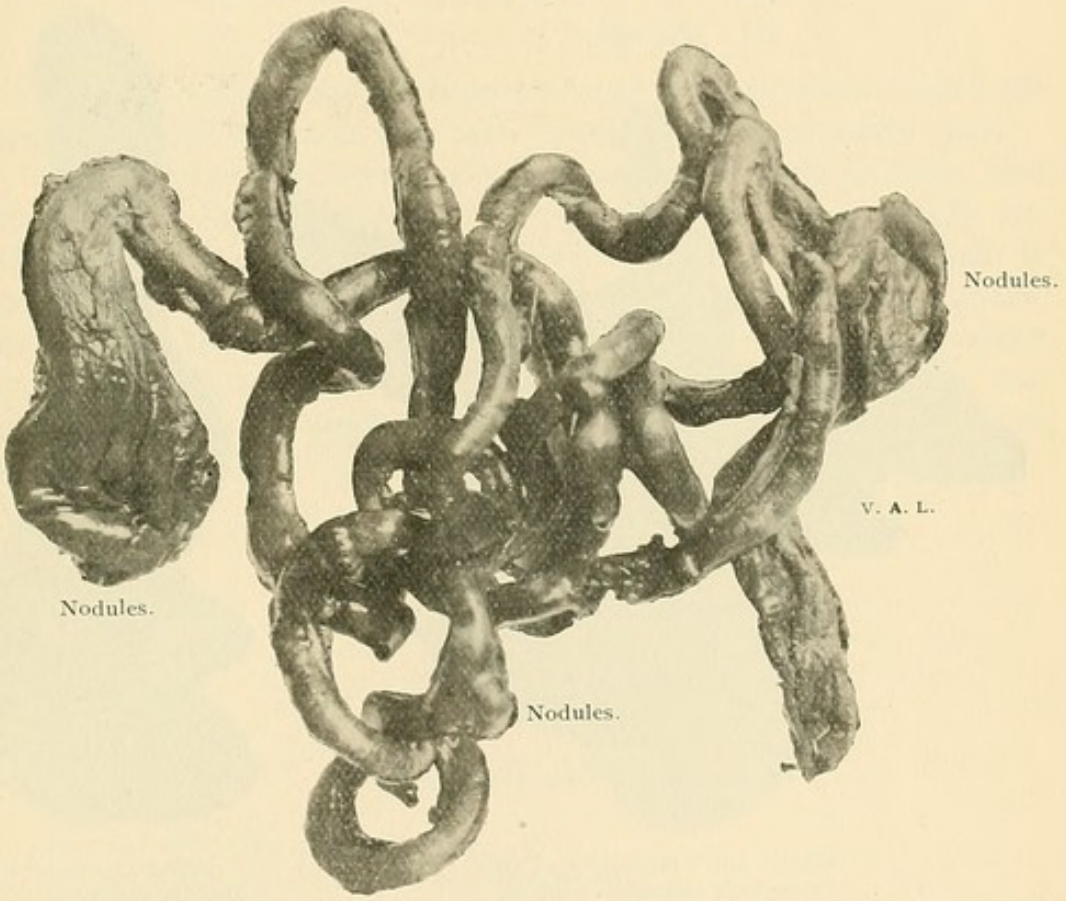
primary scirrhous carcinoma of the mammary gland of a cat and metastatic infection ("miliary carcinosis") of the lungs, the spleen, the kidneys, heart, stomach, ovaries, uterus, and the intestines.

Prevalence.—Climate and habit of life, sex and age, all seem to exert a more or less marked influence over the prevalence of cancerous disease.

Carcinoma is said to be less prevalent in the torrid than in the temperate zones, and most frequent in damp, low-lying districts. Negroes are thought to be less liable to the disease than the white race.

The aboriginal races are singularly exempt from the disease. Few cases have been reported among the North American Indians.

FIG. 219.



STOMACH AND INTESTINES OF CAT (C).
Metastatic carcinoma nodules.

The disease seems to be peculiarly an affection of the higher civilization, and influenced to a considerable degree by climatic conditions. Some idea of the prevalence of the disease may be gained from the statement that there are about thirty thousand persons at all times in England suffering from cancer. The census of 1880 of the United States gives the deaths during that year from cancer as 13,068, of which 4875 were males and 8193 were females.

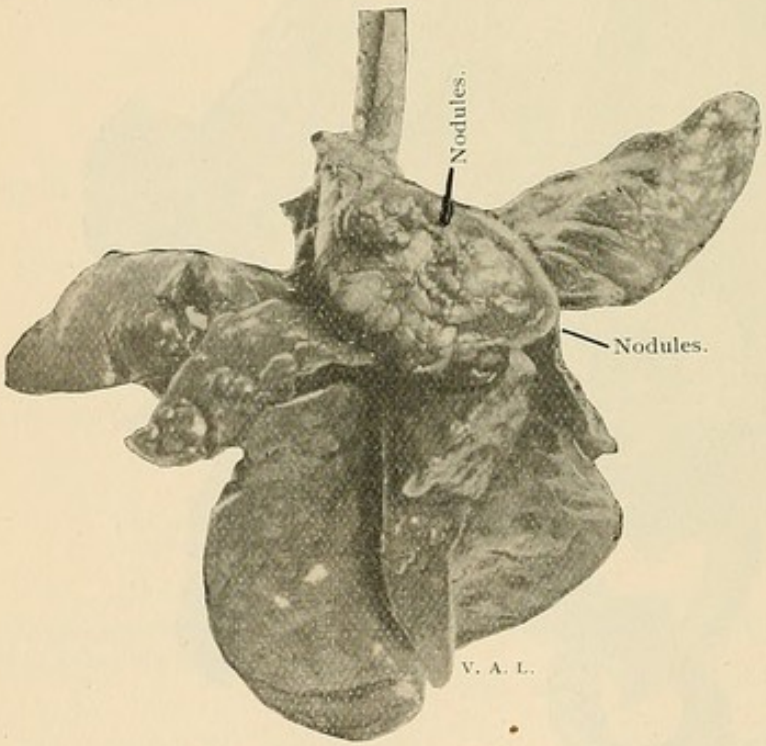
Billings states that cancer is especially prevalent in the New England States and on the Southern Pacific coast; that it is prevalent in New York, Pennsylvania, Ohio, and in the interior of Michigan and

the southern part of Wisconsin. It is least prevalent in the Mississippi Valley and in the South, while the proportions are generally lower in the coast regions than in the interior.

Park claims that the cancer mortality is greater in Western New York and the adjoining region than in any other part of the country except a limited section of California.

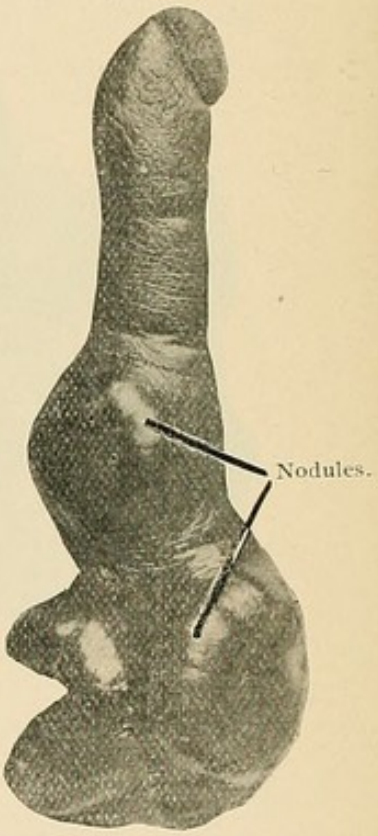
Sex.—Carcinoma in general is much more common in women than in men, and sex seems to exert a strong influence in determining the location of the disease. Carcinoma of the lip and of the pyloric

FIG. 220.



HEART AND LUNGS OF CAT (D).
Metastatic carcinoma nodules.

FIG. 221.



SPLEEN OF CAT (E).
Metastatic carcinoma nodules.

orifice of the stomach are common in men, but rare in women, while, on the other hand, carcinoma of the breast and of the genitals are frequently seen in women and very seldom in men.

Age.—Carcinomatous growths are most prevalent in persons who have reached or passed middle life. They are extremely rare in early life and very common in advanced age. Senile tissue changes seem to be particularly favorable to the development of the disease, while the consequent lowering of the powers of vital resistance and recuperation incident to advanced age places the tissues in a condition most favorable for the rapid growth of a tumor-matrix and the dissemination of the carcinoma-cells through the system.

Paget has published an interesting table showing the influence of age and the general increasing mortality of cancer with each succeeding decade from birth to eighty years of age:

PAGET'S TABLE.

Under 10 years	5	per cent.
Between 10 and 20 years	6.9	"
" 20 " 30 "	21	"
" 30 " 40 "	48.5	"
" 40 " 50 "	100	"
" 50 " 60 "	113	"
" 60 " 70 "	107	"
" 70 " 80 "	126	"

Walshe has also shown from statistics that the death rate from cancer "steadily increases with each decade until the eightieth year."

When the disease occurs in young persons it is an evidence that the epithelial cells composing the tumor-matrix are unusually endowed with the power of cell-proliferation, or that the individual is peculiarly susceptible to cancer formation; or that the tissues containing the misplaced epithelial cells have sustained an injury of some form which has lowered their vitality or physiologic resistance, and placed them in a condition to favor the growth and multiplication of carcinomatous cells.

REVIEW.

CHAPTER XLVIII.

Give the definition of carcinoma.

Has the cause of carcinoma been discovered?

At the present time what line of investigation seems to give the greatest hope of finally discovering the cause?

What results have been obtained in searching for a curative agent for the disease?

What is the generally accepted teaching as to the origin of the disease?

How has this theory been modified?

How may carcinoma be described?

Give the meaning of the term atypical.

With what tissues is primary carcinoma usually associated?

Describe the typical epithelial tumor.

Describe an atypical epithelial tumor.

What possibility should not be overlooked in considering primary carcinoma in unusual locations?

What are the histologic elements of carcinomatous tumors?

From what are the epithelial cells derived?

From what is the stroma derived?

How do the "cancer-cells" multiply?

What does the persistent characteristic of the epithelial cells make manifest?

What gives rise to the differences in structure of the various forms of carcinoma?

How may the carcinomata be divided histologically?

To what forms has the term epithelioma been applied?

By what peculiarity do the carcinoma cells indicate their epithelial origin?

What are the characteristics of the cells of this variety?

What are the characteristics of the stroma?

In what forms of carcinoma is the stroma abundant?

In what form of carcinoma is it scanty?

Where are the blood-vessels and lymphatics located?

How does this condition differ from sarcoma?

How is the blood-supply affected in the development of carcinoma?

Is there any difference in the general plan of the histologic structure of the forms of carcinoma?

How are the varieties distinguished?

What is the location of the squamous-celled carcinoma?

Describe its histologic structure.

How does carcinoma extend its boundaries?

How are the alveoli formed?

What is this form of carcinoma usually termed?

Where is the cylindrical-celled carcinoma usually located?

Describe the structure of this form of the disease.

How does this form differ from adenoma?

What other term is applied to this form of carcinoma?

What is a common condition of the stroma?

What does this indicate?

What forms of degeneration are common in this form of carcinoma?

Where is the glandular carcinoma usually found?

Describe its histologic structure.

What governs the size of the alveoli and the amount of the stroma?

In what variety of glandular carcinoma are the alveoli small and the stroma abundant?

In what variety are the alveoli large and the stroma scanty?

What term has been applied to this slow-growing form of carcinoma?

In what organs is this variety of the disease usually found?

What term has been applied to the soft, rapid-growing glandular carcinoma?

In what organs is this variety most frequently seen?

What is the form of the cells in both of these varieties?

When do the retrograde changes frequently take place?

What form of degeneration is prone to take place in glandular carcinoma?

What has been the effect of these degenerative changes upon the classification of tumors?

What is the clinical character of all true carcinomatous growths?

What marks the malignant character?

How are these conditions made manifest?

Which is the most marked feature in the clinical history?

May this be considered as pathognomonic?

What is understood by the term infection as applied to malignant growths?

Does this power explain the malignancy of such growths?

What indications are thought to be responsible for the malignant character of these tumors?

What other conditions are marks of malignancy?

How does local infection take place?

In what directions does it spread?

How does regional infection take place?

What is the character of the secondary growths found in the lymphatics?

What is the form of pathogenic material carried by the lymphatics?

How do the lymphatic glands arrest the progress of these cells?

What is Paget's view as to the material which would reproduce the disease?

What is the opinion of Gussenbauer upon this subject?

How is general infection expressed?

How is this condition brought about?

Through what channels does it enter the circulation?

How does it reach the capillaries of distant parts of the body?

What is this process of dissemination termed?

What are the tumors called which are formed by this process?

What in large measure determines the location of the metastatic tumor?

What organs are most frequently the seat of metastatic tumors?

From what form of metastatic tumor may general miliary carcinosis take place?

What disease does miliary carcinosis resemble?

What are the conditions which influence the prevalence of carcinoma?

In what climate is it most prevalent?

How are the negroes and whites affected by the disease?

How does it affect the aboriginal races?

In what form of civilization is it most prevalent?

How many persons are said to be affected at all times in England?

How many deaths occurred in the United States from the disease in 1880?

In what sections of the United States was it most prevalent?

Where was it least prevalent?

What is Park's opinion upon the sections in which it is most prevalent?

In which sex is carcinoma most common?

Does sex seem to influence the location of the disease?

In what locations of the body is the disease common in men and rare in women?

In what locations is the disease most common in women?

In what individuals is carcinoma most prevalent?

At what period is it most rare, and most common?

What conditions of the system are most favorable to its rapid growth and dissemination?

How does age affect the mortality from the disease?

What is the explanation of the appearance of the disease in young persons?

CHAPTER XLIX.

CARCINOMATA (Continued).

Causes.—Until some better theory is advanced to account for the origin of carcinomatous growths, we must hold fast to the modified theory of Cohnheim, that all such neoplasms arise from the presence in the mesoblastic tissues of a matrix of embryonic epithelial cells of prenatal or post-natal origin. The essential factor in the production of all forms of epithelial neoplasms, benign and malignant, is the presence of misplaced epithelial cells in tissues of mesoblastic origin, but the influences or agencies which impart innocency or malignancy to growths which in their incipiency are histologically identical,—for all carcinomas have a benign stage,—have not yet been revealed.

The etiology of the disease, therefore, presupposes the existence of an essential cause with a predisposition or tendency to the development of carcinomatous growths; and active or exciting agencies which stimulate the essential cause into active cell-proliferation. The essential cause, if not excited to activity, may remain in a dormant condition for years, or never give evidence of its presence within the tissues.

The essential cause of all carcinomatous growths is the presence within the mesoblastic tissues of embryonic epithelial cells of congenital or post-natal origin. "In the absence of such an essential histologic basis, no exciting cause or combination of exciting causes will result in the production of a carcinoma." (Senn.)

The congenital origin of the tumor-matrix is by far the most frequent. The tumors which arise from post-natal influences, if the investigations of Cohnheim, Billroth, and others, upon the cause of carcinoma, are correct, amounts to about 20 per cent. Boll's statistics place it at 14 per cent. Wolff's yielded only 12 per cent. The post-natal influences which give origin to the formation of carcinoma are traumatisms which bury fragments of embryonic epithelial tissues in structures of mesoblastic origin; or portions of these tissues which are buried in the process of healing of wounds, or the repair of lesions of inflammatory origin. Such a tumor-matrix acts as a foreign substance, inducing vascular excitement in its immediate neighborhood with the proliferation of embryonic epithelial cells, which, failing to reach maturity, become carcinomatous tissue.

Cohnheim, Leopold, and Zahn found by experiments that mature tissue, transplanted into the anterior chamber of the eye and the peritoneal cavity of rabbits, was invariably removed by absorption in a very short time; while, on the other hand, embryonic tissue which had been taken from animals before they were born, and transplanted into living animals, not only retained its vitality, but continued to grow to an extent which was very remarkable. Fetal cartilage grafted in this manner increased in bulk from two to three hundred times its original size.

The most prominent predisposing cause of carcinomatous growths is diminished vital or physiologic resistance (Thiersch), either of the entire organism or of the particular location surrounding the tumor-matrix. The agencies which produce these conditions have not been demonstrated, though there is good reason to believe that many times the predisposition is an inherited one; while in others it is the result of traumatisms and of local pathologic lesions.

Heredity.—It is generally believed that certain individuals inherit a predisposition or tendency to the development of carcinomatous tumors; and that the disease may seemingly be transmitted in an unbroken chain for several generations, or may show itself only occasionally, skipping one or two generations to reappear in a succeeding one.

There is no more reason to doubt the possibility of the hereditary transmission of a peculiar condition of the tissues which predisposes them to the formation of carcinomatous tumors than there is to doubt the possibility of certain congenital deformities, peculiarities of physiognomy, or mental proclivities being directly transmitted from parent to child, or suppressed for a time to reappear again several generations later, or in a distant branch of the family, for proofs of their occurrence are plentiful.

Broca has placed on record the most interesting instance of inherited predisposition to carcinoma that can be found in medical literature.

Madame Z. died of cancer of the breast, in 1788, at the age of sixty. Of four married daughters,—

A died of cancer of the liver in 1820, at the age of sixty-two; B in 1805, at the age of forty-three; C of cancer of the breast in 1814, at the age of fifty-one; D in 1827, at the age of fifty-four.

Of five daughters and two sons born to Madame B, first son died in infancy; second son died of cancer of the stomach, at the age of sixty-four; first daughter died of cancer of the breast, at the age of thirty-five; second, third, and fourth daughters died of cancer of the breast, at the ages of thirty-five to forty. The fifth daughter escaped the affection.

Madame C. gave birth to five daughters and two sons. Both sons remained free from the disease. The first daughter died of cancer of the breast in 1837, at the age of thirty-seven. This woman had

five children,—one, a daughter, died in 1854, of cancer of the breast, at the age of forty-nine; the second daughter died of cancer of the breast in 1822, aged forty; the third daughter died of cancer of the uterus in 1837, aged forty-seven; the fourth daughter died of cancer of the breast in 1848, aged fifty-five; the fifth daughter died of cancer of the liver in 1856, aged sixty-one.

Paget has found carcinoma of the uterus in three successive generations,—grandmother, mother, and daughter. Sibley has seen it in two generations. Warren has related an instance of cancer of the lip in the father and one son, and cancer of the breast in two daughters.

Senn has observed cancer of the breast in two successive generations.

Such family tendencies, however, though proving a hereditary predisposition in certain individuals to the development of cancerous growths, have not been observed in sufficient numbers to establish the law of hereditary transmission of the disease. Lebert, out of 102 cases, could find but ten who had ancestors that had suffered from the disease. Leroy d'Etoilles found but one out of 278 cases giving such a history. Gross analyzed 1164 cases, and found but 4.72 per cent. which could give a history of this character.

Statistics therefore prove that hereditary transmission of the disease is rare.

Bacteria.—During the past few years considerable interest has been awakened in the question of the microbic origin of carcinomatous growths. The disease presents many features in its clinical history which point to an infective origin, but the proofs necessary to establish the fact have not been, up to this date, presented. It would not be wise, however, to say that such proofs can never be furnished, for the writer remembers that such statements were made in reference to tuberculosis, a disease which was thought by many to be infectious in character long before the bacteriologist demonstrated the *Bacillus tuberculosis* and its infectious nature. The difficulties which stood in the way of demonstrating the presence of the tubercle bacillus were largely those of discovering suitable staining reagents. Difficulties now confront the bacteriologist in his efforts to demonstrate a cancer bacillus, but they are no greater than those overcome in relation to tuberculosis. It is possible, therefore, that a micro-organism may yet be discovered in carcinoma, which will fulfill all the requirements of Koch's law.

The discovery of such a micro-organism would work a complete revolution in the generally-accepted teaching of the origin of carcinomatous tumors.

Exciting Causes.—The presence of a tumor-matrix of epithelial cells in mesoblastic tissues is not always productive of the develop-

ment of a carcinomatous tumor. Such a matrix may remain dormant to the end of life, unless stimulated to activity by some agency outside of itself.

Traumatisms, inflammatory lesions, particularly the chronic forms, continued local irritation, and senile tissue-changes are potent factors in exciting an existing tumor-matrix to active cell-proliferation. This may be explained by the fact that tissues which have suffered damage of any sort have not the same resistive power against disease that was possessed by the same tissues in a normal state; while the increased quantity of blood supplied to the part suffering from injury stimulates the misplaced epithelial tissue to active cell-proliferation.

According to Paget, about one-fifth of those who are the victims of carcinoma ascribe the disease to some form of injury. In some cases, the disease follows immediately after the injury; in others it shows itself at a more remote period; while in another and larger class, repeated injuries are necessary to produce such a result. Senn believes that "no amount or kind of injury will produce a carcinoma without the presence of the essential tumor-matrix."

The senile tissue-changes cause the development of such neoplasms by diminishing the physiologic resistance of the tissues. These changes are different from those which take place as a result of debilitation from disease or insufficient nourishment, as is evidenced by the fact that individuals suffering from marasmus caused by debilitating disease or starvation are no more liable to be affected by carcinoma than persons of the same age who are in other respects in perfect health. (Senn.)

Thiersch has observed that in the lips of old persons the fibrous tissue wastes away, while the glandular structures increase in size, thus predisposing to the development of carcinomatous tissue.

Inflammatory lesions of chronic type are not infrequently the exciting cause of carcinoma, and both acute and chronic inflammation, as well as wounds in the process of healing, may become the starting-points of carcinoma by the inclusion of embryonic epithelial cells within the granulation or cicatricial tissue, thus furnishing not only the exciting cause of the disease, but the post-natal essential cause as well.

Insignificant injuries, like punctures, abrasions, and slight wounds, have occasionally been the only recognized exciting cause of the disease.

Irritation of a continued and prolonged character is the most common exciting cause of carcinoma, as is evidenced by the fact that carcinoma is frequently seen in those localities which are subjected to repeated and prolonged irritation. These localities are situated at the entrances of the body, viz: the lips, the nose, the rectum, the labia, and the cervix uteri.

Prolonged irritation of the tongue or cheeks from the sharp edges of carious teeth, or from accumulations of salivary calculus, are often exciting causes of cancerous growths.

Warts and moles which have been the subjects of irritation or inflammation, may also become the starting-points of carcinoma of the skin.

Tubercular lesions of the skin have been thought to be productive of carcinoma from the inflammatory irritation, and also to furnish the essential cause by the inclusion of epithelial elements in the process of healing.

Diagnosis and Symptoms.—The correct and early diagnosis of carcinoma is a matter of the greatest importance to the afflicted individual, for upon it rests the grave question of life or death. The diagnosis of this disease is sometimes an exceedingly difficult task, and the very best diagnosticians do not always succeed.

The difficulties which surround the diagnosis of the disease depend upon the stage of development and the location of the tumor. In carcinoma of the external parts, when the disease is in an advanced stage of development, the diagnosis presents no difficulties; but when located in some internal organ diagnosis becomes many times an impossibility, and the true nature of the disease is not discovered until after death. The life of the patient depends upon an early and positive diagnosis, and prompt radical measures in the surgical treatment.

To reach an early diagnosis requires a careful consideration of the clinical history, of the family history, of the signs and symptoms presented, and a painstaking examination of the tumor itself. The microscope only will reveal the epithelial character of the tumor, and such examination should always be made when possible. The presence of embryonic proliferating epithelial cells in mesoblastic tissues is the most reliable evidence of the carcinomatous nature of the tumor.

When doubt exists as to the true nature of a suspicious tumor, inoculation experiments may be necessary to differentiate between carcinoma and an infective swelling. In the diagnosis of doubtful tumors the age of the patient becomes an important and interesting element, for statistics show that the disease is most prevalent in persons of middle or past middle life, the ratio increasing with the age. All unexplainable tumors, therefore, occurring in individuals past middle life, should be diagnosed as suspicious growths, and treated accordingly.

The location of a carcinoma is largely influenced by the sex of the patient. Carcinoma of the pyloric orifice of the stomach and of the lip are most common in men, while cancer of the breasts and of the genitals are most common in women.

Another important element in the diagnosis is the rapidity of the growth of the tumor. As compared with the benign tumors, carci-

noma grows much more rapidly, and on this account might be mistaken for an inflammatory swelling. In differentiating between a carcinoma and an inflammatory swelling, it should be remembered that, as a rule, an inflammatory swelling increases in size much more rapidly than a carcinomatous growth. The inflammatory swellings for which carcinomas may be mistaken are tuberculosis, gumma, chronic suppuration, and actinomycosis.

Infection of neighboring lymphatic glands is an important symptom, and one of common occurrence in carcinoma; but this is not a pathognomonic sign, as it may occur in connection with certain infective swellings and tumors of different structure having malignant tendencies.

Dilation of the superficial veins is usually present, due to increased vascularity or to obstruction in deep-seated veins, but this condition is quite as common in infective swellings as in carcinoma.

Edema is also present in carcinoma in those cases where regional infection has caused obstruction of the lymphatic or venous circulation, or when the neoplasm has become infected with pus-producing bacteria.

Tenderness and pain are not characteristic symptoms of carcinoma, as is generally supposed by the laity. These symptoms are much more prominent, as a rule, in infective swellings than in carcinoma, although in the advanced stages of the disease they may become more or less marked. Carcinoma of the pyloric orifice of the stomach is comparatively a painless disease, the suffering accompanying it being due more to the obstruction of the orifice from the growth of the tumor than from the tumor itself. Carcinoma of the rectum is productive of little pain except during the passage of feces, and for this reason it often occurs that the patient has been affected with carcinoma for some time before seeking advice, thinking all the time that the case was one of hemorrhoids. Carcinoma of the lips, cheek, and tongue is not usually attended with much pain. The sharp, lancinating pain commonly described as characteristic of carcinoma is by no means constantly present, but is frequently intermittent in type. Tenderness is usually absent in carcinoma, except in its later stages. Redness also is rarely present except when the skin is involved and about to break down with ulceration.

One of the most characteristic diagnostic symptoms of carcinoma is the fixation of the tumor. Benign tumors are usually encapsulated, consequently are not, as a rule, fixed to surrounding tissues by adhesions, but are freely movable in all directions. In carcinoma the opposite conditions obtain, for this is an infiltrating tumor, causing immobility, and having well-defined margins and a nodulated surface.

Ulcerating tumors present the greatest difficulties in the diagnosis

of carcinoma. Syphilis, tuberculosis (lupus), and chronic ulcers of the leg are most frequently mistaken for carcinoma.

In cancer of the breast, as soon as the skin breaks, the ulcer shows a marked tendency to spread, and presents the typical appearance of a cancerous ulcer, viz: raised and rampart-like edges, surrounding an irregular depression, the floor of which is covered with firm granulations, and discharges a foul, ichorous, or blood-stained fluid. (Sutton.)

On section of a carcinoma a juice exudes from the cut surfaces, which is very characteristic of the disease.

The constitutional effect produced by carcinoma is known as the *cancerous cachexia,* and consists in rapid emaciation, anemia, and loss of strength (Warren), but the means by which these conditions are brought about are not understood.

Prognosis.—The prognosis of carcinoma will depend very largely upon its location, its histologic character, and the stage of its development when presenting for treatment.

Carcinoma of the skin is usually the squamous-celled variety. This is much more chronic in its course than either the cylindrical or the glandular-celled variety, and, being located usually upon an exposed surface of the body, attention is called to its existence, and relief is sought at a much earlier period than when the disease is located in an internal organ, or in the mucous membrane in locations which cannot be inspected by the patient. Under the latter circumstances the disease is often so far advanced before surgical advice is sought, that radical treatment would give no hope of eradicating the disease.

A favorable prognosis could only be given in such cases as had not infected the nearest lymphatic glands, nor infiltrated the surrounding tissues to any considerable degree, and in such a location as to permit of its complete removal by a radical operation. When infiltration is extensive, or a chain of glands has been infected, there is always considerable doubt existing as to whether all infected tissue has been removed. In a large majority of cases the inference is that it has not been, as recurrence after a few months is the usual outcome of operations performed at this late period.

If the disease has reached the stage of the formation of a metastatic tumor, or of miliary carcinosis, there is no hope of saving or prolonging the life of the patient, either by operation or other measures. The average duration of life in carcinoma, if unmolested, is from two to three years. (Senn.) Some authors place it at eighteen months to two years. The duration of life largely depends upon the malignancy of the tumor, its location, and the vitality of the patient. Life is finally extinguished as the result of metastasis, septic infection, exhaustion, or of encroachment of the primary or secondary tumor upon vital organs.

Treatment.—All modern writers are agreed, that if operative

treatment of carcinoma is to be successful, it is of the utmost importance that it be undertaken early in the history of the disease. Carcinoma in its earliest stages has a benign period, and if removed at this time has no more tendency to recur than other epithelial tumors which are of benign character. This is exemplified in the *early* excision of carcinoma of the lip, which if thoroughly removed is seldom followed by recurrence. Cylindrical and glandular carcinoma may also be as successfully treated by excision, provided the operation can be made under the same favorable conditions. Senn believes that carcinoma of the breast and of the uterus yield as satisfactory results if operated upon at an early period,—while the disease is still local in its manifestations,—by the removal of the entire organ, as does excision of the carcinoma of the lip. The first question to be decided in every case of carcinoma is, Can it be cured by a radical operation, or has it progressed in its course so far as to admit only of palliative treatment? If operation is decided upon, the knife should be used with a bold and fearless hand, and the entire organ affected should be excised, when practicable, and as much of the surrounding healthy tissue removed as can be done with safety.

Operation is sometimes recommended as a palliative measure, for the relief of pain, and for the purpose of removing necrotic tissue and a disgustingly-foul ulcerating sore.

A radical operation in those cases in which it is impossible to remove all the locally-infected tissue, or in which the lymphatic glands are involved or secondary carcinoma has developed, can be of no curative value, but it may prolong the life of the sufferer for a few months. and this for various business and family reasons is sometimes exceedingly desired by the patient.

Senn gives the following conditions under which a radical operation is contra-indicated: "First, extreme senile marasmus; second, extensive local infection; third, regional infection beyond the reach of complete removal of all the infected tissues; fourth, general infection; fifth, the co-existence of another disease which in itself will prove fatal in a short time."

The great majority of all carcinomata presented to the surgeon for treatment have passed the stage when a radical operation would accomplish anything more than temporary relief. It is a sad commentary upon the art of the surgeon that poor suffering humanity so dreads the knife of the operator that, if the disease is seen in time to save life by an operation and this is advised as the only safe course, the great majority will decline, or procrastinate until it is too late to be of benefit. But, when they realize this fact, they are then not only willing to submit to an operation, but sometimes demand that it be done, even at the risk of their lives while upon the operating-table.

The wound following an operation for the removal of carcinoma should be immediately covered with integument. If this cannot be accomplished by drawing the edges of the wound together by sutures, a flap of skin should be raised in the immediate neighborhood, sufficient to cover it; or, if this is not practicable, skin-grafting after the method of Thiersch may be resorted to. Healing of the wound by first intention is exceedingly desirable, and every effort should be made to secure it.

The application of caustics, arsenic, chlorid of zinc, etc., as a radical method of treatment, is not to be recommended, except in those cases in which the patient will not submit to excision; their use, however, even under such circumstances, should be restricted to small-sized carcinomata of the skin. The removal of cancerous growths by the aid of caustics is much more painful than by excision with the knife, while the patient is under the influence of local or general anesthetics. The operation by caustics also requires much more time both for the removal of the tumor and for the healing of the wound, while the cosmetic effect is not so good, as it invariably leaves a large scar.

Palliative treatment in the non-ulcerative form consists of opium to relieve pain, and local applications of lead-water and tincture of opium, or other soothing remedies. In ulcerative carcinoma the surface may be dusted with iodoform and morphia; cocain solution painted or sprayed upon the surface will also relieve the pain. The fetor of the discharges may be lessened by the use of carbolic acid solutions, phénol sodique, cinnamon water, or a solution of chloral. The employment of dry boric acid dressings is also valuable for the same purpose, as they favor rapid drying of the discharges, and prevent putrefaction.

REVIEW.

CHAPTER XLIX.

What is the essential factor in the production of all forms of epithelial neoplasms?

Have the influences or agencies which impart innocency and malignancy to these growths been discovered?

How may the causes be divided?

What is the essential cause of the disease?

Which is the most frequent origin of the essential cause?

About what per cent. have their origin in post-natal influences?

What are these influences?

How may such a tumor-matrix produce carcinoma?

What is the result of transplanting mature and embryonic tissue?

What is the most prominent predisposing cause of carcinoma?

What are the agencies which produce these conditions?

How is heredity manifested?

What reason is there for believing that such tendencies may be transmitted in certain families?

In how many generations did Broca trace the disease?

Does the evidence of Broca and other writers prove the hereditary transmission of the disease? If not, why?

What do statistics prove in reference to hereditary transmission of carcinoma?

What features in the clinical history of carcinoma point to micro-organisms as a possible cause of the disease?

What were the chief difficulties in the way of demonstrating the tubercle bacillus?

Has a cancer bacillus been demonstrated up to the present date?

What would be the effect of such a discovery upon the teaching of to-day as to the origin of carcinoma?

Is the presence of an epithelial tumor-matrix always productive of a carcinomatous growth?

What are the exciting causes of carcinomatous growths?

How do local lesions produce the disease?

About what per cent. of cases are ascribed by patients to injury?

How soon does the disease follow the injury?

Can injury alone produce the disease?

Are extensive injuries necessary to excite the tumor-matrix to active growth?

How may inflammatory lesions and wounds become the starting point of carcinoma?

How do senile tissue-changes cause the development of such neoplasms?

What tissue-changes were observed by Thiersch in the lips of old persons?

What is the most common exciting cause of the disease?

What is the evidence of this fact?

Where are these localities?

How may decayed teeth and salivary calculus become exciting causes of cancerous growths?

How may warts and moles become the starting-points of carcinoma of the skin?

How may tubercular lesions of the skin cause the disease?

Why is a correct and early diagnosis in carcinoma imperative?

Is such a diagnosis readily made?

What are the difficulties which surround the diagnosis?

Under what circumstances does the diagnosis become impossible?

What are the points which must be considered in the diagnosis?

What is the most reliable evidence of the carcinomatous nature of a tumor?

How may an infectious swelling be differentiated from a malignant tumor?

How does the age of the patient assist in making a diagnosis in suspicious tumors?

In what locations are carcinomata more liable to occur in men than in women?

In what locations do they more often occur in women?

Of what diagnostic value is the slow or rapid growth of the tumor?

What point should be remembered in the differential diagnosis between carcinoma and an infective swelling?

What form of infective swellings may be mistaken for carcinoma?
Is the infection of the lymphatics an important symptom?
What other diseases produce this symptom?
What is the cause of the dilation of the superficial veins?
What is the cause of the edema?
Are tenderness and pain characteristic symptoms?
In what stage of the disease do they become prominent?
What is one of the most characteristic symptoms of carcinoma?
What forms of ulcerating tumors are frequently mistaken for carcinoma?
What is the typical appearance of an ulcerating carcinoma?
What characteristic sign appears upon section of the tumor?
What is the constitutional effect produced by carcinoma?
Upon what does the prognosis of carcinoma depend?
What is the prognosis of carcinoma of the skin?
Under what circumstances could a favorable prognosis be given?
In what class of cases does recurrence usually take place?
At what stage of the disease is there no hope of saving or prolonging the life of the patient?
What is the average duration of the disease?
How is death finally brought about?
Upon what point in the treatment of carcinoma are all modern writers agreed?
How is the fact demonstrated that carcinoma has a benign period?
What is the first question to be decided in every case of carcinoma presenting for treatment?
What should be the character of operative treatment?
Under what conditions may an operation be resorted to as a palliative measure?
What value has a radical operation when lymphatic infection has taken place, etc.?
Name the five contra-indications for operation named by Senn.
How should the wound be treated after excision of the tumor?
By what means may this be accomplished?
Under what circumstances may the use of caustics be recommended?
Which method is the most painful, and which gives the best results?
What is the palliative treatment in the non-ulcerative stage?
How may the ulcerating stage be treated?
How may the fetor of the discharges be overcome?

CHAPTER L.

CARCINOMATA (Continued).

THE varieties of carcinoma which are of the greatest interest to the student of oral surgery are those which appear upon the integument of the face, upon the lips, the oral mucous membrane, the tongue, and the salivary glands.

FIG. 222.



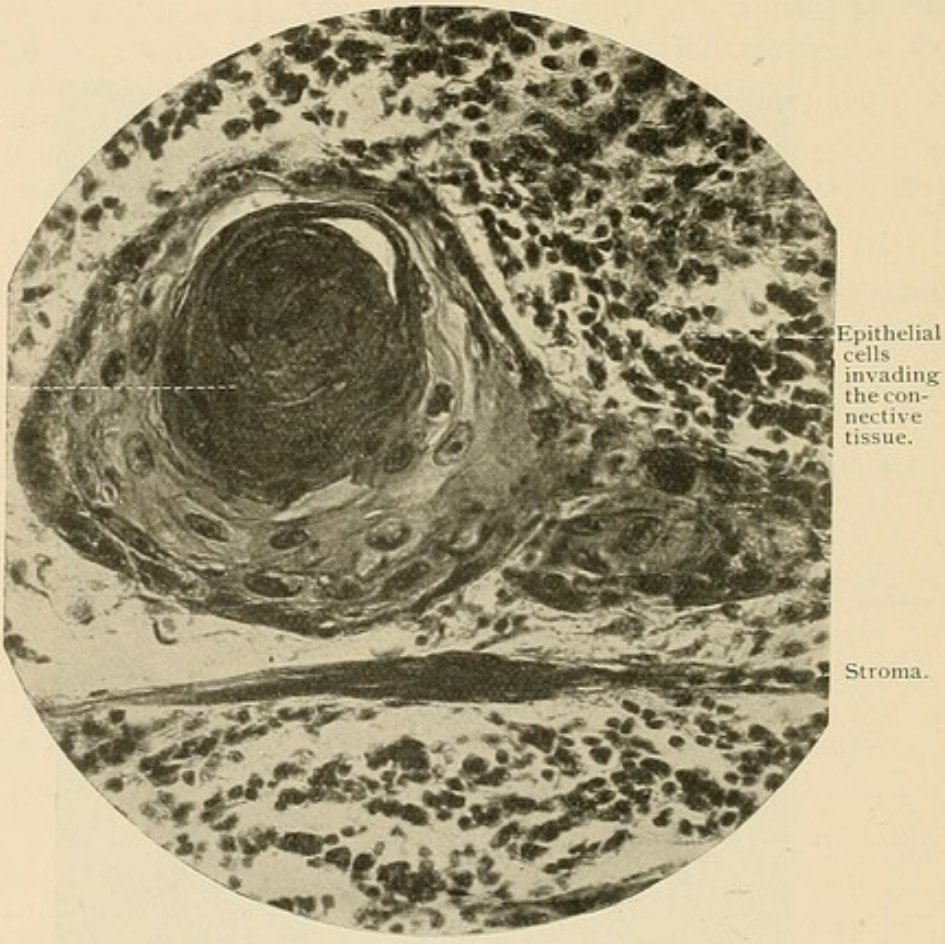
CARCINOMA—EPITHELIOMA—GENERAL TYPE OF THIS FORM OF GROWTH. ×50.

The varieties of carcinoma which are found affecting the superficial surfaces of the skin, the mucous membrane, and the tongue, as well as those which attack the glandular structures of these tissues, are commonly classed under the head of *epithelioma*. (Fig. 222.) This

term came into use before it was discovered that all forms of carcinoma are epitheliomatous (epithelial) growths, and was originally applied to a form of cancer located in the epithelial tissue of the skin. Fig. 223 is from an epithelioma of the skin of the abdomen. Fig. 224 illustrates the microscopic appearance of chimney-sweep's cancer of the scrotum.

Carcinoma of the Skin.—Squamous-celled carcinoma (epithelioma of the skin) may develop in any portion of the body where a squamous or stratified epithelium may be found, but it is most frequently located in those portions where the skin and mucous mem-

FIG. 223.



EPITHELIOMA OF SKIN (ABDOMEN). × 500.

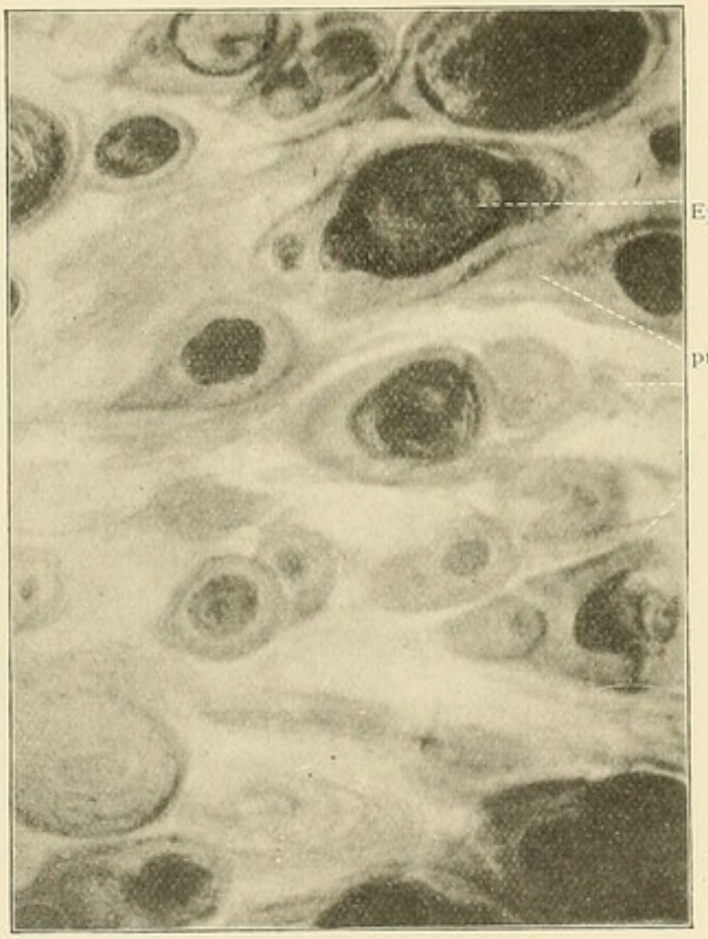
brane come into relation with each other, and at which point there is a transition from the stratified to the columnar form of epithelium.

Carcinoma of the skin differs histologically from the benign forms of epithelial tumor (papillomata) in the fact that it is not confined to the epithelial tissues of the skin, but passes the boundaries of the basement membrane, and involves the surrounding connective tissue. Accompanying this invasion of the connective tissue there is developed a

peculiar arrangement of the epithelial cells known as "cell-nests," or "epithelial nests."

These tumors usually make their appearance upon the surface of the skin, either as warty growths, as slight cracks or fissures covered by a scab or crust, or as flattened tubercles. The base is hard, the margins indurated, and the tumor sometimes slightly elevated above the surrounding skin. In all these forms, ulceration sets in early. Sometimes the ulcer will possess raised, rampart-like edges; in another form,

FIG. 224.



CHIMNEY-SWEEP'S CANCER OF SCROTUM. (T. Charters White.) × 100.

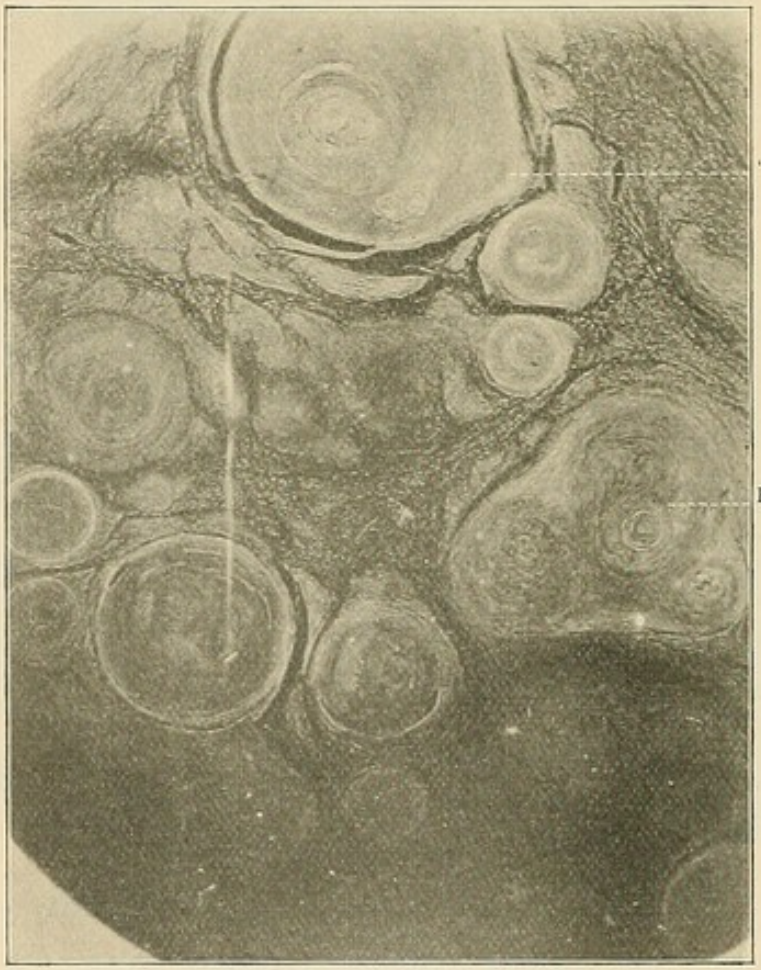
instead of raised edges, the margins will be sharply defined; while occasionally, in still another, the edges will be undermined. The ulceration which takes place is due to a defective blood-supply of the cells forming the central or oldest portion of the initial tumor, which results in their death.

The tendency of primary carcinoma of the skin in the ulcerative stage, when left to itself, is to spread, and to involve extensive areas of tissue, or to form fungous masses, or by gigantic granulations to form large cauliflower-like excrescences. Accompanying these conditions

is a foul, fetid discharge, in which are found small masses of dead tissue, cell débris, and blood. In its most malignant form, the surrounding tissues, either skin, muscle, or mucous membrane, are quickly invaded and destroyed, while the bone even is attacked and rapidly destroyed by erosion and necrosis. Cartilage alone resists the invasion of the disease.

Fatty degeneration is the most common retrograde change that takes place in carcinoma of the skin. Colloid degeneration is more

FIG. 225.



CARCINOMA—EPITHELIOMA—SHOWING PEARL OR COLLOID BODIES. × 75.

rare. (Figs. 225 and 226.) In the warty form the processes which project from the skin are sometimes quite horny in their hardness.

Histologically all of these varieties of carcinoma of the skin are identical in their structure. Sections of these neoplasms so cut as to include the edge of the ulcer and the adjoining tissue also, will show the surface epithelium dipping down into the connective tissue beneath, in the form of long projections, "columns," or "plugs. The tissues about these epithelial projections are infiltrated with epithelial

cells (Sutton), while scattered among these projections and within them in various locations, are found those "peculiar concentric cellular bodies" designated as epithelial nests. A peculiarity of the epithelial projections is that they have no limiting membrane, and that many of the larger projections branch and unite with neighboring columns, forming a sort of net-work or reticulum. "Cell-nests" are a peculiarity of all forms of skin carcinoma, but though the size of the

FIG. 226.



CARCINOMA—SHOWING COLLOID DEGENERATION. × 70

epithelial projections and the number of the cell-nests may vary in the different forms of the disease, the general plan of the extension of the tumor is the same in all. (Fig. 227.)

The locations in which this form of carcinoma is found are the face, the lips, the eyelids, the buccal aspect of the cheeks, the esophagus, the edges of old scars, and various other portions of the body which are not of especial interest in this connection.

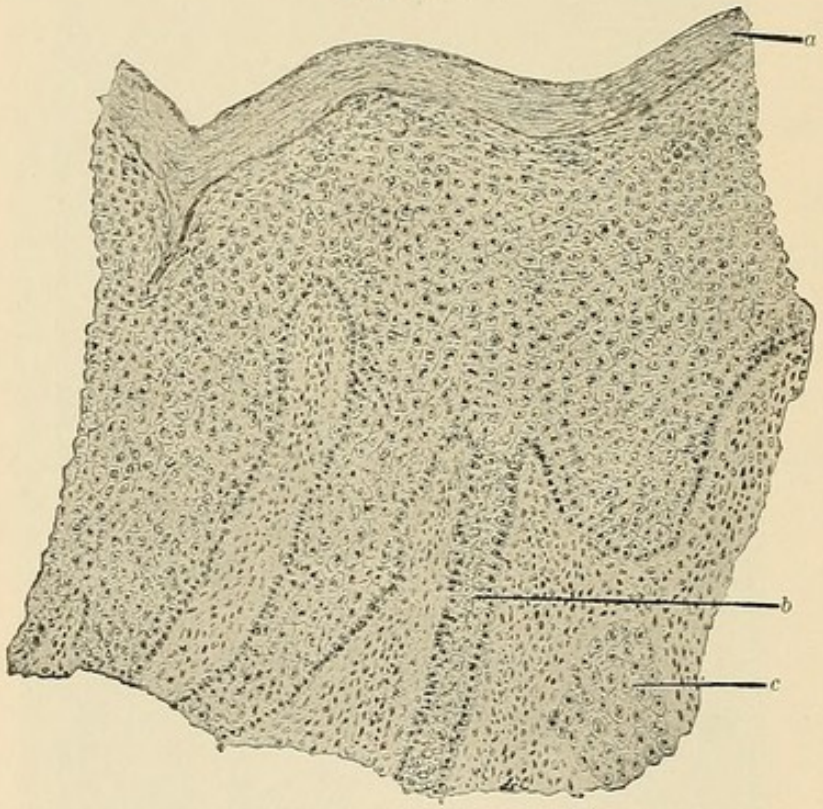
Carcinoma of the Face.—The face is the most frequent location of carcinoma of the skin. Out of 740 cases of tumors of all kinds, sub-

jected to operative treatment, O. Weber found 133 cases of carcinoma of the face, or 17.97 per cent.

It occurs more frequently in men than in women. V. Ziemssen collected 948 cases of carcinoma of the skin; of these 739 (77.95 per cent.) were in men, and 209 (22.05 per cent.) were in women, showing the disease to be about three and one-half times more frequent in men than in women.

The period of life in which carcinoma of the skin is most common is between forty and eighty years of age. The age at which it has been most frequently seen is between fifty-five and sixty.

FIG. 227.



CARCINOMA OF SKIN OF NATES. × 110. Reduced one-third. (After Senn.)

a, hypertrophied stratum corneum; *b*, growth of epithelial cells into subcutaneous tissue; *c*, epithelial nest in vascular connective tissue.

Carcinoma of the skin, according to Thiersch, appears in two forms,—one a superficial ulceration (rodent ulcer), and the other penetrates deeply into the tissues, and successively involves the different structures. This is called the deep-seated, penetrating, or polymorphous-cell cancer.

The superficial variety of carcinoma, "rodent ulcer," is almost always found upon the face, and is frequently preceded or accompanied by *keratosis*, a thickening of the epidermis, and the presence of such callosities as warts, horns, etc.

Superficial carcinoma is characterized by the formation of scabs

or crusts, which may exist for a long time before any malignant tendency is manifested. They appear first as scales, slightly elevated above and somewhat darker than the surrounding skin. The surface is sometimes smooth and shining, and at others covered with slightly adherent scabs. The elevated spots are not sensitive, and when the crust is removed leave an exposed superficial excoriation. This variety of carcinoma does not usually appear until after middle life, is extremely slow in its development, is painless, and on this account does not cause any anxiety as to its nature, until it has assumed considerable dimensions.

Histologically, if the tumor is composed of squamous cells it appears from the beginning as an infiltration with small epithelial cells, which, before ulceration occurs, fill the alveoli of the stroma. If it arises from the glandular appendages of the skin, the columnar epithelial cells will be found arranged in groups resembling the tubular glands. (Senn.) In the early stage of ulceration the ulcer is not deep; its surface is quite flat and bounded by a pearl-colored rim, the shape often resembling a horn waistcoat button. (Warren.) The ulcerative process from this time progresses by an unequal extension in different directions, so that the surface of the ulcer presents a very irregular outline.

In this form of the disease glandular infection usually comes late in the life-history of the disease; for this reason operative procedures are more liable to prove curative than in other forms of carcinoma.

In the superficial form the ulceration takes place rapidly, but it is confined to the skin; while in the deep-seated or penetrating form, besides spreading in various directions, it also extends downward, involving the tissues beneath its base regardless of their structure, be it connective tissue, muscle, or bone.

Carcinoma of the face most often attacks the upper portions; the frontal and malar regions, the eyelids, and the nose. Frightful deformities are sometimes produced by the extension of the disease. Warren describes a case in which the disease originated in the scar of a gunshot wound received in the civil war, and had destroyed the side of the nose, the eye, the ear, and the cheek, including the corresponding half of the upper and lower lips.

Diagnosis.—In the diagnosis of carcinomatous growths of the face it must be borne in mind that there are certain diseases which may be located in the face, such as tuberculosis, syphilis, suppurative conditions of benign tumors, and retention cysts (Senn), which may mislead even careful observers. Tuberculosis of the skin of the face may so nearly resemble carcinoma as to make it impossible to differentiate them without the microscope and inoculative experiments. Tertiary syphilitic affections of the skin may usually be differentiated from car-

cinoma by the presence of scars from syphilitic lesions which have healed. Doubt as to the nature of the disease may be cleared up by a resort to anti-syphilitic treatment for a few weeks, when improvement will soon be manifest if the disease is syphilis.

Prognosis.—The superficial variety of this disease in its early stage gives all the evidence of a benign growth, but there is a period in its history when for some unexplainable reason it suddenly takes on a malignant form, and the superficial variety of the disease is changed to the penetrating or deep variety. The slight malignancy of these ulcerating forms of the disease is thought to be due to the feeble reproductive power of the small epithelial cells, but Warren "thinks it more probable that there are other factors to be considered, such as the anatomical seat of the disease, and, possibly, the nature of the parasite,—if there be one,—which caused it."

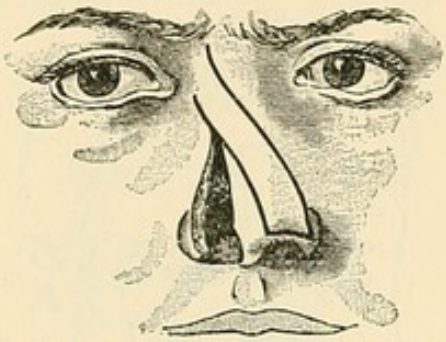
The prognosis is much more unfavorable in the deep-seated variety of the disease than in the superficial form, as the ulceration spreads rapidly, and results in extensive destruction of the various tissues surrounding it in a remarkably short space of time. Warren looks upon the region of the nasal process of the superior maxilla as one of the most dangerous and "important strategic points" in cancer of the face, for the reason that carcinoma originating here, or encroaching upon the inner margin of the orbit, may suddenly involve the lymphatics leading to the base of the skull. He looks upon the disease as incurable after it has once passed the margin of the orbit. Recurrence is the rule, though occasionally a permanent cure takes place. If the disease does not return in five years, the cure may be considered as permanent.

Treatment.—The operative treatment of carcinoma of the face, to be successful, must be undertaken at a period prior to its infection of the lymphatics of its neighborhood. The tendency of the disease to extend toward the orbit, and involve the eye, makes it imperative that operative interference should take place before the orbit is reached. The eyelids and the side of the nose are quite frequently the seat of the disease, and sometimes extensive operations are required for its removal. The defects left by the removal of the cancerous growths are to be remedied by plastic operations and skin-grafting. When the lower eyelid has to be removed, the defect may be most satisfactorily remedied by Dieffenbach's method.

Carcinoma affecting a portion only of one ala of the nose may be removed by cutting out a wedge-shaped piece of the entire thickness of the ala, and closing the defect by taking a flap from the opposite side of the nose, after the method of Langenbeck (Figs. 228, 229), or from the face, after the manner of Esmarch (Fig. 230). Thiersch grafts should be used to cover the wound left by the removed flap. When

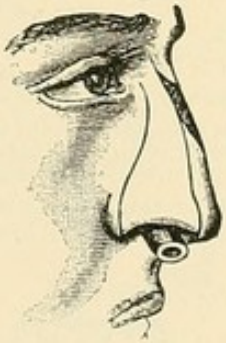
the tumor involves the bridge of the nose and the bone has become infected, an extensive operation becomes necessary, requiring the removal of the bony framework, and sometimes of considerable portions

FIG. 228.



PARTIAL RHINOPLASTY BY TAKING A FLAP FROM THE OPPOSITE SIDE OF THE NOSE. (After Langenbeck.)

FIG. 229.



PARTIAL RHINOPLASTY COMPLETED. (After Langenbeck.)

FIG. 230.



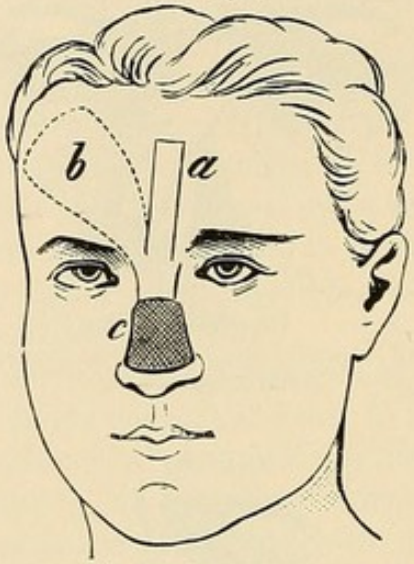
PARTIAL RHINOPLASTY BY TAKING A PEDUNCULATED FLAP FROM THE FACE ALONG THE BASE OF THE NOSE. (After Esmarch.)

of the nasal mucous membrane, as well as the integument. In those cases where the tip of the nose and the nasal apertures have not been involved in the operation, König's flap operation will correct the defect in an admirable manner. (Figs. 231, 232, 233.) When all of the nose

has been sacrificed, Thiersch's operation for restoring the lost organ may be resorted to. (Fig. 234.)

The excision of carcinomatous growths in other portions of the face should be performed with the same desire for thoroughness. In

FIG. 231.



KÖNIG'S RHINOPLASTY.

a, *b*, flap for building nose, including skin, periosteum, and a thin slice of bone; *b*, flap used to cover flap *a*, and to furnish integument for the entire defect; *c*, defect caused by excision of tumor.

FIG. 232.



KÖNIG'S RHINOPLASTY.

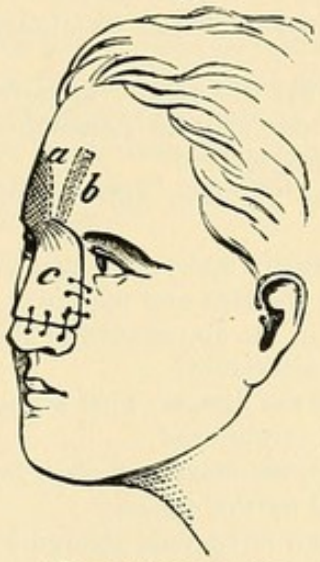
a, flap turned downward; *b*, lower end fastened in place with catgut sutures. The skin at the top of the nose at *b* is left free, and to it flap *b* is sutured.

removing these tumors, it is better to sacrifice a little healthy tissue, and gain a successful issue of the operation, than to insure a speedy recurrence by carrying the incision so close to the growth that some portion of the infected tissue is left behind. The defects caused by the operation should be remedied by bringing the edges of the wound to-

gether with sutures, or by a flap operation, and when this is not possible, by Thiersch skin-grafts.

Recurrence is to be looked for, and repeated operations may be required to eradicate the disease; but so long as lymphatic infection has not taken place and there is no evidence of other than local disease, there is hope of a final cure.

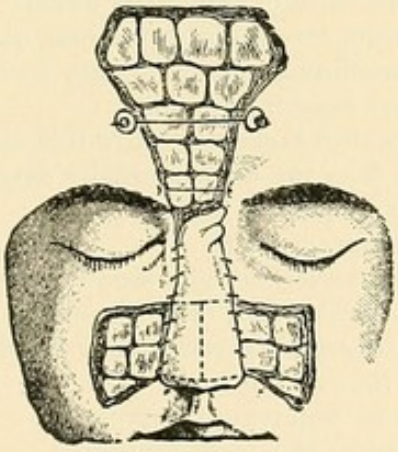
FIG. 233.



KÖNIG'S RHINOPLASTY.

a, *b*, defects over the frontal bone; *c*, flap *b* which covers the bony surface of flap *a*, and furnishes the cutaneous covering for the entire defect, sutured in place.

FIG. 234.



RHINOPLASTY. (After Thiersch.)

Warren mentions a case of a gentleman who allowed a cancer to grow upon the left side of the nose until it involved the skin of that side, and a portion of the right side. The disease returned three times. Finally the left half of the nose and the nasal process of the superior maxilla were excised, and the cavity thus left was covered by a flap taken from the forehead. The disease did not return after this, the fourth operation.

REVIEW.

CHAPTER L.

What varieties of carcinoma are of the greatest interest to the student of oral surgery?

What term is usually applied to those varieties of carcinoma which affect the superficial structures of the skin, mucous membrane, and glandular structures of the mouth?

To what form of cancer was this term originally applied?

In what portions of the body may the squamous-celled carcinoma be found?

Where is it most frequently located?

How does carcinoma of the skin differ histologically from the benign epithelial tumors?

How do these tumors usually make their appearance?

What is the condition of the base and margins?

What are the variations in the appearance of the ulcerated surface?

What is the cause of the ulceration?

In the ulcerative stage of the disease, what are its tendencies?

What accompanies these conditions?

Describe the conditions in the most malignant form of the disease.

How is cartilage affected by the disease?

What is the most common retrograde change in this form of carcinoma?

What sometimes characterizes the warty form of carcinoma?

Describe the histologic structure of carcinoma of the skin.

What is a peculiarity of the epithelial projections?

What is a peculiarity of all skin carcinomas?

In what locations is this form of carcinoma found?

What is the most frequent location of carcinoma of the skin?

What percentage of carcinomatous tumors are found in the face?

In which sex is it most frequently found?

In what proportion are men more subject to this disease than women?

At what period of life does the disease usually develop?

At what age is it most frequently seen?

In what forms does it appear?

Where is the superficial variety usually found?

By what condition of the skin is it often preceded?

By what condition is superficial carcinoma of the skin characterized?

How do these crusts first appear?

What is the condition on removing the crust?

At what period of life does this form appear?

What is the character of its growth, etc.?

What is the histologic character of the tumor when composed of squamous cells?

What, when arising from the glandular appendages?

How does the disease appear in the early stage of ulceration?

What is the character of its progress?

When does glandular infection take place in this form of the disease?

What are the differences in the ulcerative process in the superficial and deep-seated varieties?

In what location of the face is carcinoma most frequently seen?

What disease may be mistaken for carcinoma of the face?

How may doubts in reference to tuberculosis and syphilis be cleared up?

What is the prognosis in the superficial variety?

What is it in the deep-seated variety?

In what portion of the face is carcinoma considered most dangerous?

Is the disease liable to recur?

At what period should operative treatment be made?

By what means are the defects left by the removal of the growths to be remedied?

By what method may the lower eyelid be restored?

By what method may portions of the ala of the nose be restored?

By what means may the bridge of the nose be restored?

By what means may the entire nose be restored?

What should be the character of all operations for carcinoma of the face?

How is recurrence to be treated?

CHAPTER LI.

CARCINOMATA (Continued).

Carcinoma of the Lip.—Carcinoma situated in the lip, like carcinoma in general, rarely develops until after middle life. It is most frequently seen between the fortieth and sixtieth years.

The deep-seated, penetrating, or polymorphous-cell carcinoma is typically represented in carcinoma of the lip. The disease usually commences at the border of the lip, at the junction of the mucous membrane with the skin (Fig. 235), and its first appearance is either in the

FIG. 235.



CARCINOMA OF THE LOWER LIP. (After Senn.)

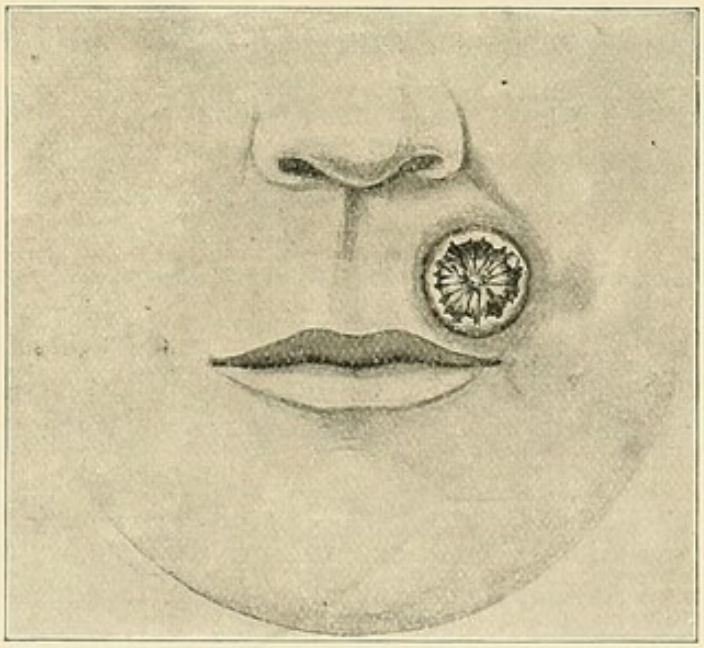
form of a small papule, or as a flat crust (Warren) which frequently scales off only to be re-formed again. It rarely occurs at the angles of the mouth, or upon the upper lip. (Fig. 236.) The general appearance of the disease in its early stage is that of a superficial infiltration of the vermilion border of the lip, having a well-defined indurated outline. Later ulceration of the indurated area takes place in the center, from which may be squeezed the atheromatous contents of the exposed epithelial cell-nests. It then presents a shallow, ulcerated surface with circular outline and elevated overhanging edges. (Fig. 237.) With

FIG. 236.



EPITHELIOMA OF THE UPPER LIP—EARLY STAGE. (After Sutton.)

FIG. 237.



CARCINOMA OF THE UPPER LIP. (After Senn.)

few exceptions, the disease if left to itself involves the greater portion of the lip or destroys it altogether, and the chin, cheek, and lower jaw are successively implicated. The submaxillary lymphatic glands on the affected side at this time are enlarged, and become adherent to the maxillary bone, which gives the appearance of malignant disease arising from the periosteum of the jaw.

The disease sometimes appears as a warty excrescence, which may involve the entire lip. (Fig. 238.)

FIG. 238.



EPITHELIOMA OF THE LOWER LIP—"WARTY" VARIETY. (After Sutton.)

Occasionally it may occur that the lip will be affected upon one side, and the lymphatic glands of the submaxillary region upon the other. No explanation of this peculiarity is yet forthcoming. (Sutton.)

Sometimes the glands are affected upon both sides, but this is usually an expression of rapid extension of the primary disease to the opposite side of the lip, with implication of the mental and submental tissues, of the most serious character, and suggests an early termination of the life of the patient. In the later stages of the disease, the sub-

maxillary triangles and the neck become greatly enlarged, food is taken with difficulty, and the patient slowly dies from marasmus, sepsis, or a general infection and dissemination of the disease, or from hemorrhage. Metastatic nodules are sometimes found in the internal organs. The disease usually terminates fatally in from three to five years, though occasionally it runs a much shorter course.

Carcinoma is much more frequent in the lower than in the upper lip. It is common in men, but exceedingly rare in women. König's statistics place the proportion of males to females as 20 to 1. Warren's reported cases make the proportion 19.25 to 1, while those of Lortets' show the proportion to be 7.6 to 1.

W. Roger Williams, in a recent contribution to the *British Medical Journal* regarding primary neoplasms of the lip, states that of 13,824 primary neoplasms of all kinds, consecutively under treatment at St. Bartholomew's University College, Middlesex, and St. Thomas Hospitals, during the last sixteen to twenty-one years, 352, or 2.5 per cent., originated in the lips. These included 7297 cancers, of which 332 grew from the lips, or 4.5 per cent. Of the 352 lip-neoplasms, 340 sprang from the lower lip, thus: epithelioma, 329 (males 326, females 3); papilloma, 7 (males 4, females 3); angeioma, 3 (male 1, females 2); cystoma, 1 (male). Only 12 originated in the upper lip, thus: epithelioma, 3 (male 1, females 2); sarcoma, 4 (males 2, females 2); angeioma, 3 (male 1, females 2); papilloma, 1 (female); fibroma, 1 (male).

Carcinoma is oftener seen upon the left side of the lip than upon the right. It may also occur upon the median line. (Warren.) The cause of the disease is often ascribed to some form of chronic irritation.

Irritation from the constant use of tobacco, and particularly of the pipe, has been thought by many to be a prolific cause of carcinoma of the lip.

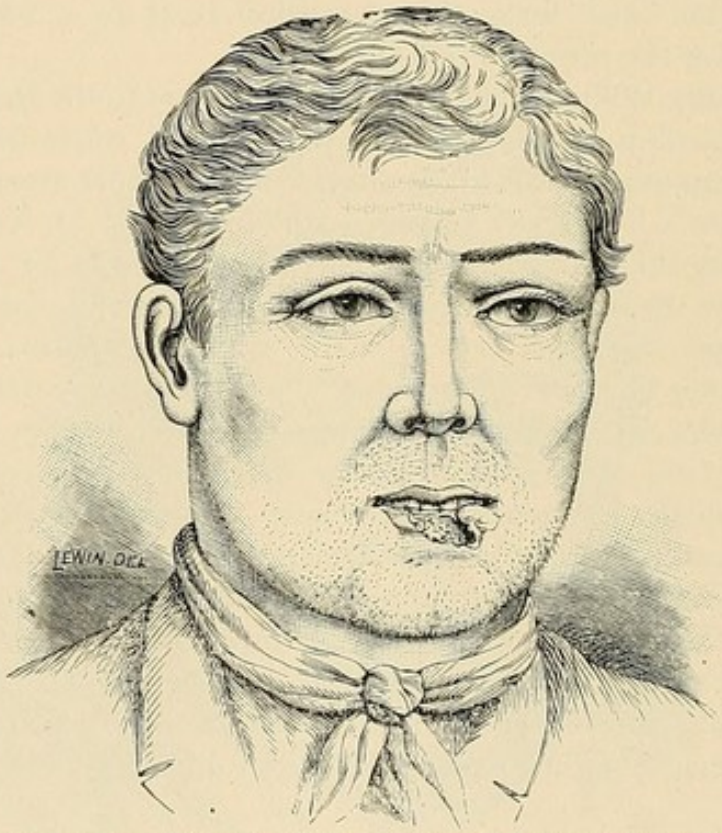
Mason Warren ascertained that out of 77 cases of carcinoma of the lower lip, all but seven were in the habit of smoking; out of this number four were women, and three of them were in the habit of using a pipe.

It is a significant fact in the observation of the writer, that the side of the lip most often the seat of the disease is the side of the mouth upon which the pipe has been habitually carried. It has also been observed that smokers who are subjects of carcinoma of the lower lip have frequently been in the habit of using clay pipes, the stems of which have not been prepared to prevent the irritation of the lip so common in using new ones. J. C. Warren mentions a case of carcinoma of the lip occurring in a woman,—the only one coming under his observation,—and she was in the habit of smoking. The carcinoma in this case was in the upper lip. The writer has seen one case of the disease in an Irish woman, seventy years old, an habitual smoker, and who

always used a clay pipe. In this case the neoplasm was in the lower lip upon the left side. The pipe was carried between the teeth upon the left side, the stem of which had worn for itself a notch in the upper and lower teeth, in which it rested.

The writer has also seen several cases of carcinoma of the lip in men where the evidence was unmistakable that the disease had started in an abrasion or excoriation caused by the stem of the pipe resting upon the lip.

FIG. 239.



EPITHELIOMA OF THE LOWER LIP, BEGINNING IN A FISSURE. (After Sutton.)

The formation of the neoplasm is frequently preceded by a crack or a fissure (Fig. 239), or an eczematous patch in the margin of the lip (Senn), or by an injury. Out of 145 patients suffering from carcinoma of the lip, Koch attributed 15 to traumatisms. (Senn.)

Diagnosis.—In the diagnosis of carcinoma of the lip, there is danger of confounding eczematous conditions with the superficial spreading form of carcinoma in its early state. The exposure of the deeper layers of the skin, the papillomatous appearance, the raw surface and serous transudation all tend to render the diagnosis somewhat difficult. The presence of an indurated condition of the deeper structures of the skin or of the mucous membrane, associated with these symptoms, marks the carcinomatous character of the growth, while the absence of the induration marks its eczematous character. A primary syphil-

itic sore might mislead in the diagnosis, were it not remembered that it develops very rapidly, and is associated at a very early period in its history with glandular infection. The opposite of these conditions marks even the most rapid growth of carcinoma. A secondary syphilitic lesion of the lip usually originates in the mucous membrane of the mouth, and infects the lip by extension. (Senn.) Primary tuberculosis of the lip is a very rare affection. It may be distinguished from

FIG. 240.



CARCINOMA—EPITHELIOMA OF LIP. (A.) × 50.

carcinoma by its more diffuse character from the beginning, and the absence of the induration so characteristic of carcinomatous growths. Doubts as to the character of the tumor can be cleared up by the microscopic character of the tissues, and by inoculation experiments. Figs. 240, 241 show the histologic structure of epithelioma of the lip.

Prognosis.—The prognosis is usually favorable in those cases in which an operation is made in the early stage of the disease—while it is still superficial. But an opinion upon the matter should be guarded even here. Complication of the lymphatic glands is usually consid-

ered as an exceedingly unfavorable symptom, and the utility of an operation for their removal, if much enlarged, is open to serious question. When of small size and freely movable, they should be extirpated, together with as much of the surrounding tissue as may be safely removed.

Occasionally the operation for even the superficial form of the disease is quickly followed by recurrence. Warren relates the case of a "physician who applied for operation about three months after the first

FIG. 241.



CARCINOMA—EPITHELIOMA OF LIP. (B.) × 100.

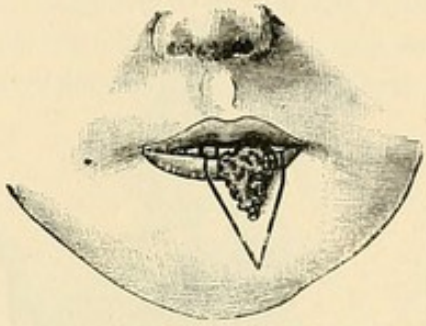
appearance of the disease. There was no return in the lip, but a gland under the jaw began to enlarge six months later, and the patient succumbed eighteen months after the first appearance of the disease." Fortunately such cases are exceptional, but there are few surgeons of many years' practice who have not had like unfortunate experiences.

In those cases in which the glandular structures are involved to any considerable extent, and the jaw has become infected, operations are of very little value.

Treatment.—Carcinoma of the lip in its early stages may be readily

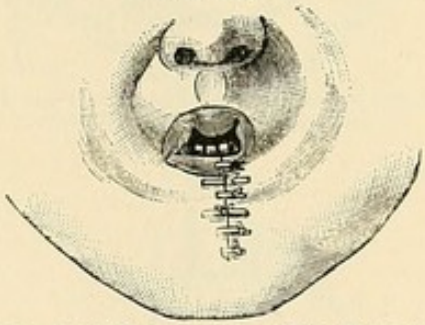
removed by a V-shaped excision of the lip. (Fig. 242.) When the loss of tissue has been comparatively small, not more than half the width of the lip, the defect may be remedied by simply bringing the edges of the wound together and suturing them in that position. This leaves at first a puckered condition of the mouth (Fig. 243), but the lower lip gradually elongates, and after a time a fairly good lip results.

FIG. 242.



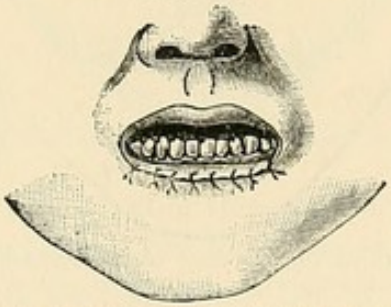
V-SHAPED EXCISION OF THE LOWER LIP FOR CARCINOMA. (After Esmarch.)

FIG. 243.



OPERATION COMPLETED. (After Esmarch.)

FIG. 244.



SUTURING AFTER EXCISION OF THE ENTIRE MARGIN OF THE LOWER LIP FOR CARCINOMA. (After Esmarch.)

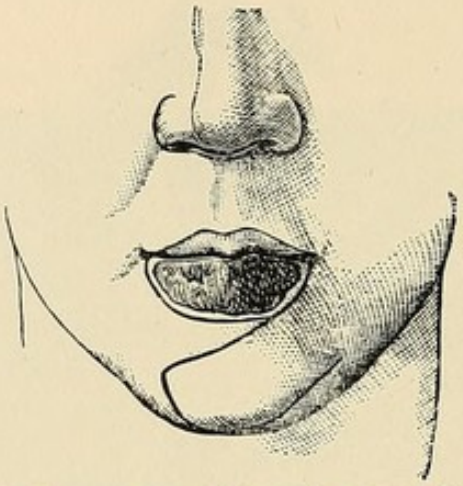
Another method is that of Celsus. After removing the tumor by the V-shaped incision, horizontal incisions are carried out from the base (apex of the V) for a sufficient distance to enable the wound to approximate more easily.

In cases of superficial carcinoma of the border of the lip involving only the mucous and submucous tissues, an incision may be made at one angle of the mouth, and carried through the lip to the opposite

oral angle, removing the entire margin of the lip. The mucous membrane is then reflected over the surface of the wound, and sutured to the margin of the skin. (Fig. 244.)

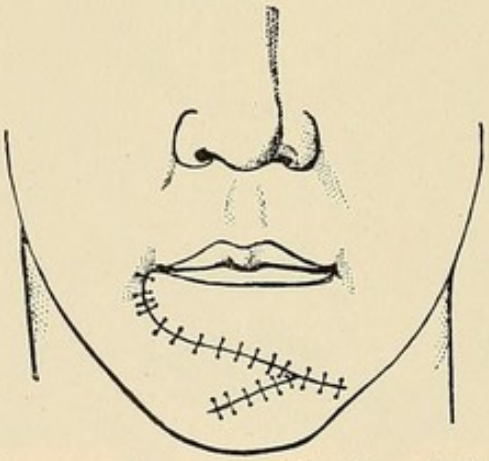
In cases requiring the removal of more than half of the width of the lip, it should be done by a curved incision, the convexity being downward. The mucous membrane may then be lifted and sutured

FIG. 245.



LANGENBECK'S METHOD OF RESTORING THE LOWER LIP AFTER EXCISION FOR CARCINOMA. (After Langenbeck.)

FIG. 246.



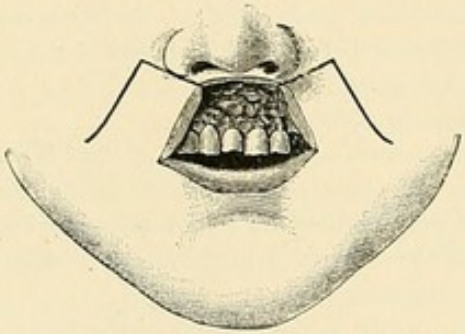
OPERATION COMPLETED. (After Langenbeck.)

over the surface of the wound to the margin of the skin. This leaves a semilunar defect in the border of the lip, but in the course of time it gradually diminishes. When the entire lip has to be excised, a new lip must be made by a plastic operation. Langenbeck has devised a method of accomplishing this. (Fig. 245.) The lower horizontal margin of the defect is prolonged on either side by incisions, which pass along the remainder of the lower lip, around the angles of the mouth and into the upper lip; each portion of the lip is mobilized and drawn together by sutures. Fig. 246 shows the operation completed.

The objection to Langenbeck's second method is the fact that the border of the new lip cannot be covered with mucous membrane, and that cicatricial contraction takes place to a certain extent during the healing of the wound.

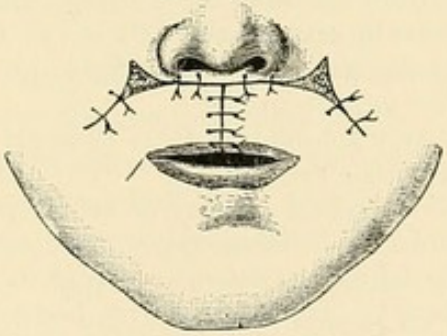
Hueter's operation for the restoration of the lower lip is also a valuable method, and is described as follows: The margins of the defect in the lip are brought together by sutures, and an incision is made in the cheek horizontally outward from the angle of the mouth, involving the entire thickness of the cheek; the mucous membrane on each side of the new wound is reflected a little and then united to the skin by sutures.

FIG. 247.



CHEILOPLASTY. (After Bruns.)

FIG. 248.



OPERATION COMPLETED. (After Bruns.)

Operations for the removal of tumors located in the upper lip do not differ for partial excision from those practiced upon the lower lip. In those cases requiring the excision of the entire upper lip, the defect may be cured by the method devised by Bruns, which consists in fashioning two quadrilateral flaps out of the entire thickness of the cheek and upper lip, on each side of the mouth, and turning them down so that their upper borders can be sutured together in the median line. (Fig. 247.) Finally the edges of the wounds left upon each side are to be brought together with sutures, and their size diminished as much as possible. (Fig. 248.)

In all operations upon the face the utmost care should be exercised to reduce to a minimum the deformities and scars left by surgical

operations and accidents. Plastic surgery has been brought to such a high degree of perfection that there are few defects in connection with the human face which cannot be restored with fairly good cosmetic and functional results.

REVIEW.

CHAPTER LI.

At what period of life does carcinoma of the lip develop?
What form of this disease is found in the lip?
Where does it usually begin?
Where are its first manifestations?
Describe its general appearance in the earlier and later stages of the disease.
At what period does infection of the glands take place?
What peculiarity of gland infection is sometimes observed?
What conditions indicate an early termination of the life of the patient?
How is life finally terminated?
What is the duration of life in carcinoma of the lip?
In which lip is carcinoma most frequent?
In which sex is the disease most common?
In about what proportion are males more affected than females?
Upon which side of the lip is it most often seen?
To what condition is the disease often ascribed?
What would statistics seem to prove?
Does clinical observation substantiate these figures?
By what is the disease often preceded?
What diseases occurring on the lip may be mistaken for carcinoma?
What conditions of the surrounding tissues mark carcinoma?
How may carcinoma be differentiated from syphilis?
How may tuberculosis be differentiated from carcinoma?
What is the prognosis of carcinoma of the lip?
Are operations of value in cases of extreme involvement of the glandular structures and of the jaw?
What form of operation is recommended for small carcinomata of the lip?
What was the method of Celsus?
Describe Langenbeck's operations for restoring the lower lip.
Describe Heuter's method.
How may the defect caused by the removal of the entire upper lip be cured?
Describe Bruns's operation.

CHAPTER LII.

CARCINOMATA (Continued).

Carcinoma of the Buccal Mucous Membrane and Jaws.—The buccal mucous membrane is covered by pavement epithelium several layers in thickness. The superficial layers represent the squamous or flat epithelium, and the deeper layers the cylindric or columnar variety. Carcinoma of the mouth has its origin, as in the skin, either in the stratified epithelium or in the cylindric epithelial cells of its glandular appendages.

Two forms of carcinoma are found in the mucous membrane as in the skin, viz: the *superficial* and the *deep-seated* varieties. The only differences between these forms are those due to peculiarities in the structural arrangement of the epithelial cells, and to the tissues being constantly bathed with moisture.

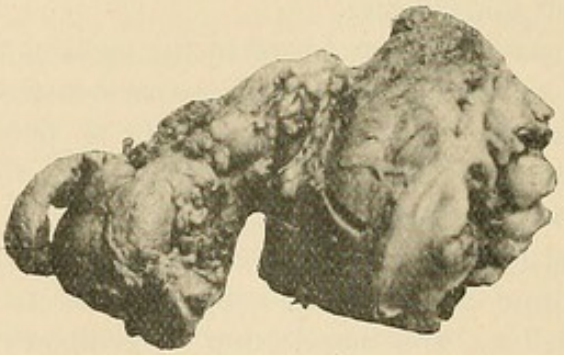
The superficial form of the disease follows the clinical history of the affection as found in the skin, being confined to the superficial layers of the epithelium, and manifesting no tendency to involve the glandular structures of the mucous membrane.

The first manifestation of the deep-seated variety of the disease is usually the formation of a small, hard nodule within a tubular gland of the mucous membrane. The epithelial cells forming the center of the nodule undergo fatty degeneration, and finally, when ulceration takes place, the atheromatous mass can sometimes be squeezed out, leaving a deep central depression upon the surface of the ulcer. The base of the ulcer is always indurated, while the edges are raised above the surrounding surface. The ulcer shows no tendency to heal, but, on the contrary, spreads in all directions. Microscopically the tumor presents a tubular structure, the tubules being lined with columnar epithelial cells. This form of the disease is most frequently located in the mucous membrane of the cheek, but it may originate in the gums, the soft palate, the tonsils, and the pharynx. The disease often starts near the angle of the mouth, and extends backward upon the cheek; or it begins in the gingivo-buccal fold, and occasionally in the center of the cheek upon a line indicated by the occlusion of the upper and lower teeth. The disease is rarely seen at the angle of the mouth or in the upper lip, which is explained by the fact that the mucous glands are less

numerous in these locations than in other portions of the oral mucous membrane.

Carcinoma of the oral mucous membrane has been thought by some observers to be due to the irritating effects of tobacco, as produced by smoking, but as there are no statistics presented to corroborate the statement, it is presented merely as an opinion. Carcinoma of the mucous membrane of the cheek is sometimes preceded by the appearance of a patch of "leucoma" or leucoplakia,—a chronic superficial inflammation of the mucous membrane or the tongue, characterized by the presence of pearly-white or bluish-white patches upon the surface of the gums, mucous membrane, or tongue. The affection is rare in women or in individuals under twenty years of age. It is due to some chronic form of irritation, such as the smoking of a pipe, or the wearing of artificial teeth. Wallenberg believes it is caused most fre-

FIG. 249.

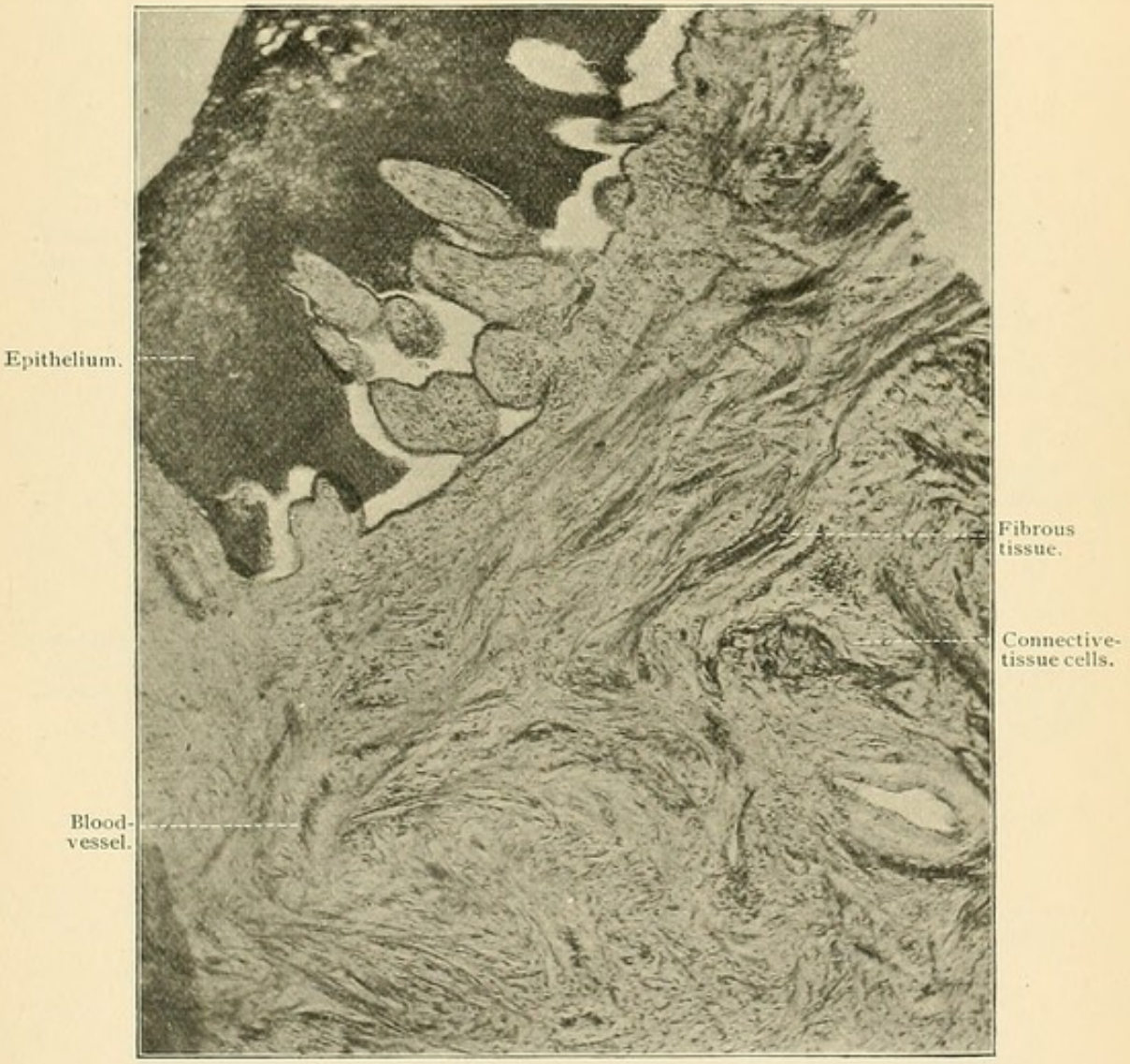


MIXED CARCINOMA OF THE INFERIOR MAXILLA. (A.)

quently by the irritation produced by the volatile and empyreumatic oils of tobacco. (Warren.) The writer has seen but three cases of leucoplakia, all in men. In one the affection was upon the dorsum of the tongue near the center, the patch being the size of a quarter-dollar piece. This gentleman was forty years old, and a great smoker of cigars. It had caused no inconvenience, although it had been there for several years. In another case the disease was located in the left gingivobuccal fold, but principally upon the gum, in a man past fifty years of age. The disease had been noticed for several years, and for more than a year past had been very troublesome, causing pain and smarting when acids, salt, or anything of pungent flavor came in contact with it. Operation was advised, but declined. Soon after this it took on malignant symptoms, extending to the superior maxillary bone and the whole side of the cheek. Three years afterward he died from extension of the disease to the lymphatic glands of the neck. The third case was similar to the second in its location. This patient was a physician forty-five years of age, and appreciated the danger of procrastination in an affection of this character. The presence of the disease was dis-

covered by accident during an examination of his teeth. It was evidently in its early stage of development, as its presence had never been recognized by the patient. He demanded an immediate operation, and the diseased tissue was removed down to the bone. Eight years afterward there had been no recurrence.

FIG. 250.



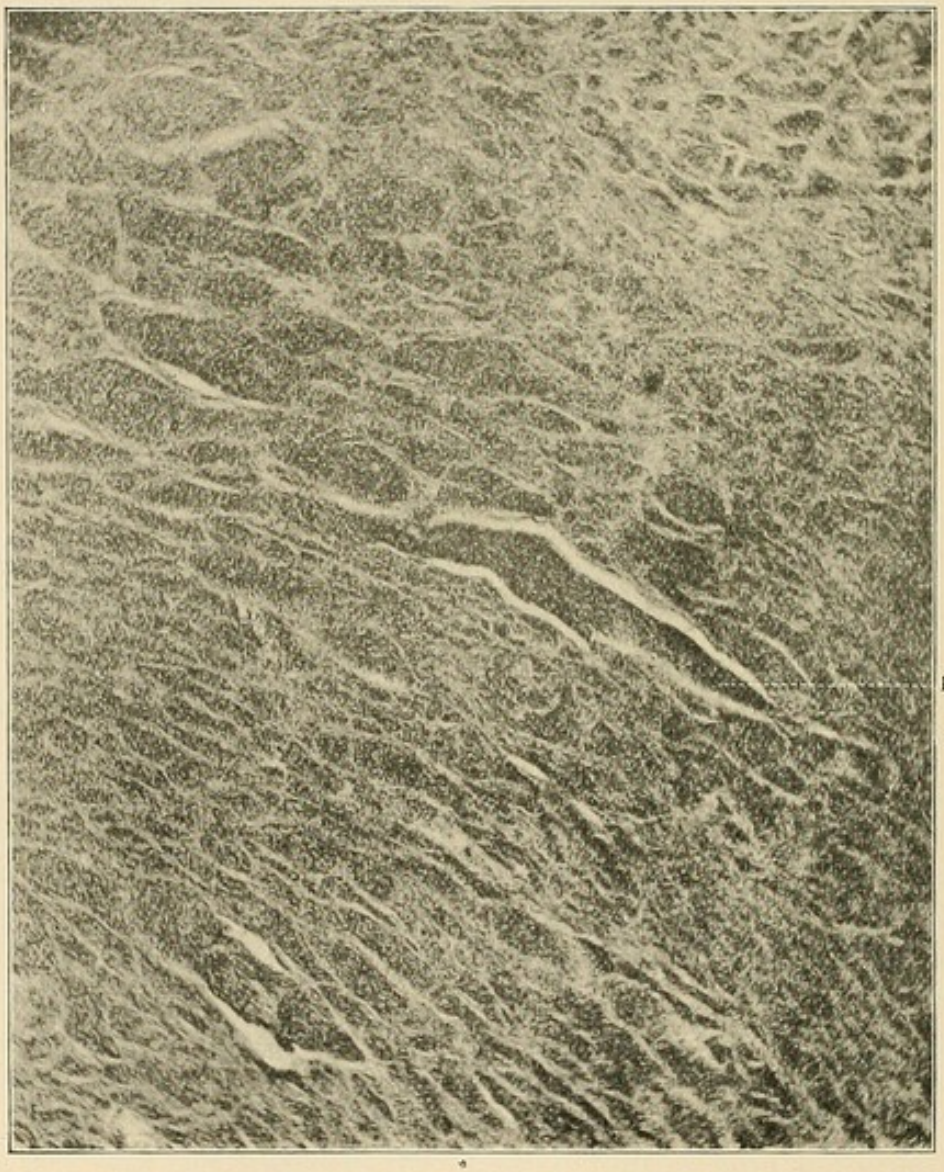
CARCINOMA—MIXED—OF INFERIOR MAXILLA. (B.) FIBROUS PORTION. × 70.

The fact that carcinoma of the cheek frequently appears along the line of the occlusal surfaces of the superior and inferior molars, opposite decayed or roughened buccal surfaces of the teeth, or upon the tongue or gums, irritated by the same means, leaves no doubt that these agencies are prolific exciting causes of the disease.

Carcinoma may appear in the superior or inferior gum, but it is most frequently seen in the mucous membrane covering the lower

alveolar process. The disease usually arises in connection with a carious tooth in which the cavity of decay has extended beneath the margin of the gum, leaving a sharp or jagged edge; or in relation with a devitalized and carious tooth, the crown of which has been lost, leaving the root with rough margins which irritate the gum.

FIG. 251.



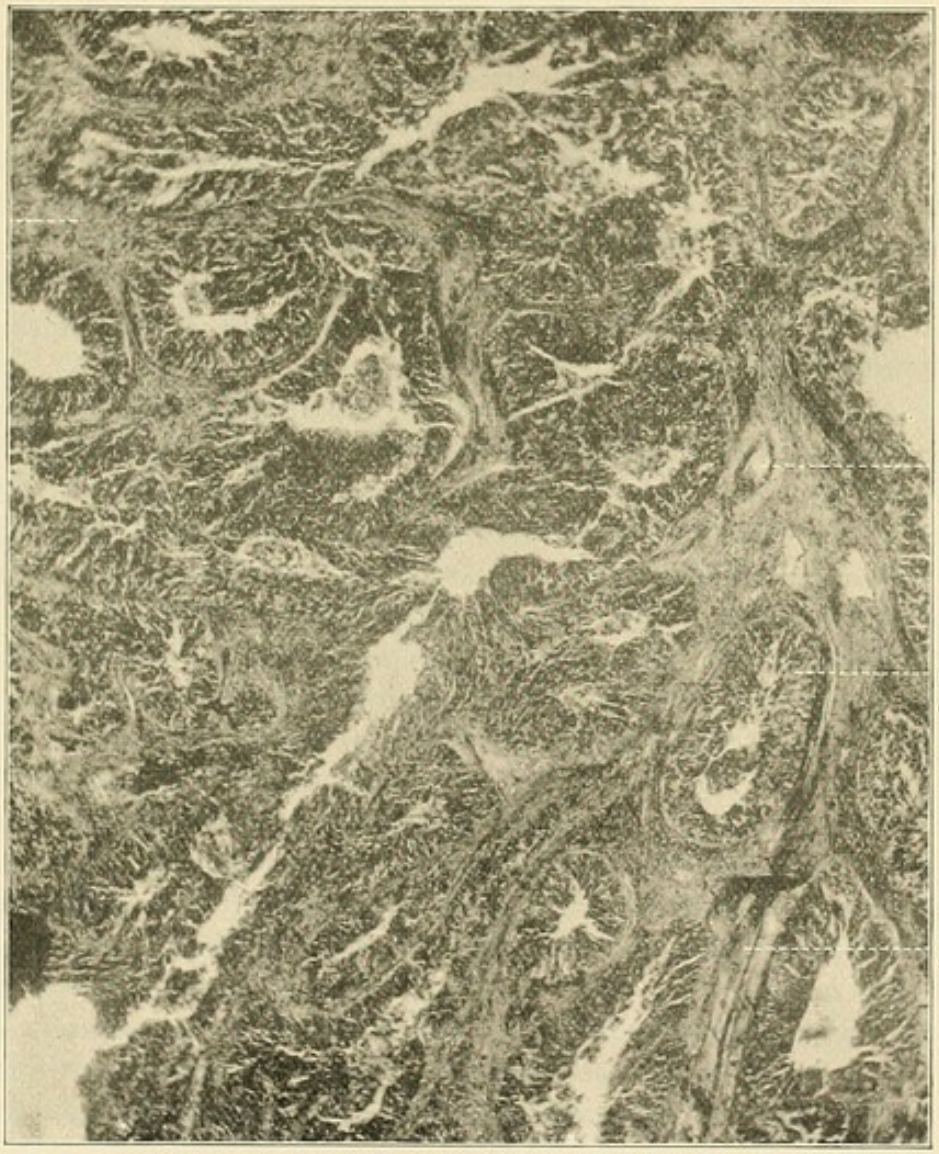
CARCINOMA—MIXED—OF INFERIOR MAXILLA. (C.) × 70.

The affection in this location is characterized by an exceedingly rapid progression. The bone is infected in a very short space of time, and this condition is often mistaken for the primary affection. The disease as it progresses in the lower jaw has a tendency to spread in one direction toward the cheek, and in the other toward the tongue. Figs. 249, 250, 251, 252 are photographs of a mixed carcinoma of the inferior maxilla and its histologic structure. When located in the su-

perior maxilla the alveolar process is quickly eroded, and the antrum is invaded, leaving a foul ulcerating cavity, which discharges an offensive pus. The erosion of the bone as it takes place in either maxilla causes loosening and exfoliation of the teeth in the infected region.

Carcinoma of the Antrum.—Some of the most distressing cases of carcinoma coming under the observation of the writer have been

FIG. 252.



CARCINOMA—MIXED—OF INFERIOR MAXILLA. (D.)

those which involved the antrum of Highmore, either by extension from the mouth, or in which the disease was primarily located in this sinus. The latter form of the disease occurs in individuals past middle life. The first symptom complained of is pain in the jaw, for which no adequate cause can be assigned. (Sutton.) Pain is sometimes entirely absent. Later there is observed a slight fullness in the

infra-orbital region, with perhaps edema of the lower eyelid, occasionally a mild degree of exophthalmos; the skin becomes discolored, and later a carcinomatous ulcer breaks through the skin of the cheek near the inner canthus of the eye, or into the mouth through the alveolus of a recently extracted tooth. Inspection of the mouth will also reveal extensive erosion of the palate process, and bulging of the roof of the mouth upon the affected side. If the case is operated upon at this period, it will be found, upon reflecting the cheek, that the disease has usually made such extended inroads upon the surrounding tissues as to have involved not only the palate process, but all of the bony walls of the antrum, penetrated to the muscles of the cheek, involving the skin, and implicating the contents of the orbit, making it necessary to remove the entire maxillary bone of the affected side, together with the infected tissue of the cheek and the contents of the orbit, leaving a yawning chasm which cannot be closed by plastic operation. The disease is very fatal, and life is rarely prolonged beyond a few months at most, but during that time the patient is spared much suffering and discomfort as a consequence of the operation.

Regional infection of the lymphatic glands is an early and conspicuous feature of the disease when it involves the maxillary bones. Sutton calls attention to the fact that the infected lymph glands of the neck may attain an extraordinary size, while the ulcer upon the gum may be very small, not exceeding one cm. in diameter, thus tending to mislead the surgeon in his diagnosis. He further suggests that rapid-growing glandular enlargements located in the neck in persons past middle life should call for a careful examination by the surgeon "of the mouth and fauces, for small inconspicuous epitheliomatous ulcers, and with every care they sometimes escape detection during life."

Carcinoma of the mouth affecting the mucous membrane of the cheeks and gum, and involving the superior maxillary bones, is an exceedingly rapid and fatal form of the disease, the average duration of life being about eighteen months. As a rule, these growths resent surgical interference. The insidious character of the tumor is such that it has usually passed the time when radical measures are available before its true nature has been discovered. Excision of the growth has almost always been followed by recurrence, for the reasons just mentioned. There would seem, however, to be hope of a permanent cure of the disease if it could be removed before regional infection had taken place.

Treatment.—Surgical operations for carcinoma of the mouth, to be of real value, must be bold, and at the same time most painstaking. There is perhaps no affection with which these qualities of an operation are more imperatively demanded. Timidity or carelessness upon the part of the operator will result not only in a damaged reputation, but

in what is of infinitely more serious consequence to the patient,—a quick recurrence of the disease. Radical operations for carcinoma of the mouth, particularly in those cases affecting the maxillary bones, will require external incisions. Operations conducted through the mouth for so grave an affection are of little practical benefit, as it is impossible to thoroughly trace the ramifications of the disease, or by any possibility remove infected submaxillary glands through this cavity.

The writer had one unfortunate experience in his early practice in attempting to remove such a growth through the mouth. Thorough removal of the diseased tissue was impossible, and the patient's condition three months after the operation was worse than before. The surgical interference, on account of the imperfect removal of the carcinomatous tissue, and the irritation incident to the operation, seemed to stimulate the growth to most active cell-proliferation. Four months after the operation the patient died.

The incisions should always be made with reference to the least possible amount of disfigurement. When operating upon the upper maxilla, the incisions should follow the natural lines of the face as far as possible; in operations upon the lower jaw the lines of incision can be hidden in large part beneath the jaw. The extent and character of the particular operation in each individual case will be indicated by the location of the neoplasm and the extent of the infection. Partial excisions of the superior maxillary bone may be done by the Nélaton operation or that of Guerin. Partial excision of the inferior maxilla may be accomplished by incisions passing through the median line of the lip and chin, and carried backward to the angle of the jaw beneath the lower border of the bone.

Carcinoma of the Tongue.—According to Butlin, carcinoma of the tongue is confined to the squamous-celled variety, and he refutes the statement that the tongue is sometimes the seat of hard and soft carcinoma. Fig. 253 shows a vertical section of a circumvallate papilla of the human tongue.

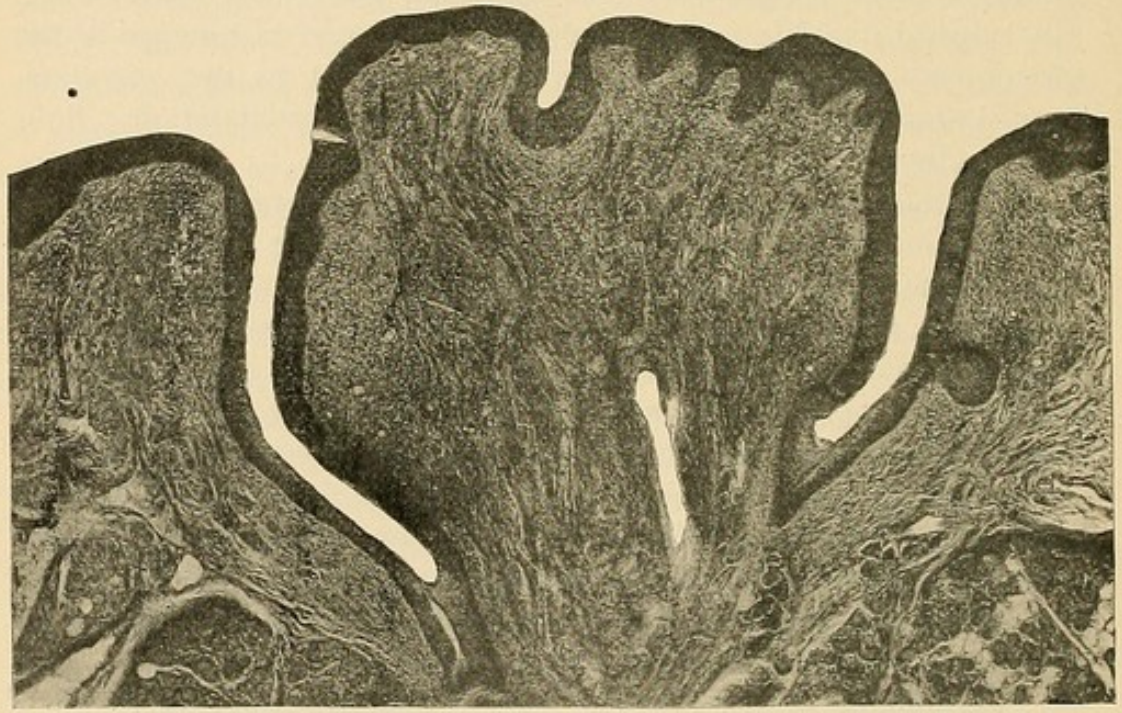
Carcinoma of the tongue is usually located upon the side or the dorsum, usually near the tip. The anterior half is much more frequently the seat of the disease than the posterior half, while it may be stated generally that the disease is usually located in some spot in its anterior two-thirds, the edges being more subject to the disease than either the dorsum or the under side. (Butlin.) The disease is always, even when upon the dorsum, distinctly located either upon one side or the other of the median line, but so far as statistics go, they do not show any distinct preference of the disease for one side over the other.

The disease may make its first appearance either as a blister, an excoriation, an ulcer, a fissure, a pimple or tiny tubercle, a warty

growth, or a nodule (Butlin) upon the surface or within the substance of the tongue. In the early stages of the disease the microscope shows that the tumor retains the papillary structure of the tongue upon its surface. But later, after ulceration becomes more extensive, infiltration from the surface results in the production of epithelial prolongations and the formation of epithelial "cell-nests" within the vascular connective-tissue stroma in typical concentric layers. (Fig. 254.)

Causes.—The most frequent exciting causes of the disease are chronic irritation, from mechanical agencies,—rough or carious teeth, ill-fitting dental plates,—and the irritating influences of smoking

FIG. 253.



VERTICAL SECTION OF CIRCUMVALLATE PAPILLA OF HUMAN TONGUE. (T. Charters White.) × 100.

tobacco. Besides these forms of irritation may be mentioned certain chronic inflammatory affections of the mucous membrane which precede the formation of cancerous growths in the tongue, viz: leucoplakia, ichthyosis, psoriasis, and syphilis.

Symptoms and Diagnosis.—The disease usually runs a rapid course, the lymphatic glands of the neck soon become infiltrated, and death supervenes in from one to two years.

Pain in carcinoma of the tongue is often quite sharp and severe, of stinging character, and sometimes reflected to the ear. As the disease advances, speech and deglutition become more and more embarrassed. Salivation is also a prominent symptom. The surface of the ulcer is sometimes covered with papillary growths, sometimes with sloughing, gangrenous shreds of connective tissue. The indurated character of

the cancerous growths of both base and margins remains throughout the course of the disease.

The disease is more common in men than in women. Sutton places the proportion as 3 to 1; Senn, 7 to 1; Butlin nearly 6 to 1, and Barker, 6.35 to 1. The disease is pre-eminently one of past middle life, being most frequent after the fortieth year. It has been recognized in patients as young as twenty-five, and as old as seventy-five years; the largest number affected being between the ages of forty and sixty, the largest proportion beween forty-five and fifty. (Sutton.)

FIG. 254.



EPITHELIOMA OF THE TONGUE. (T. Charters White.) × 100.

The diseases which are most likely to be confounded with carcinoma are syphilitic indurations and sores, tubercular nodules and ulcerations, warty tumors, traumatic ulcers, fissures, and actinomycosis. Syphilitic indurations—gummata—of the tongue may be differentiated by the presence of other syphilitic lesions of the tongue or other portions of the oral cavity or of the body. Tubercular disease of the tongue is rarely seen in other persons than those suffering from pulmonary tuberculosis. A tubercular ulcer does not possess the in-

durated base and margins characteristic of carcinoma, and its surface is covered with fungous granulations. Actinomycosis is an exceedingly rare disease in man. The microscope is the only means of positive diagnosis.

Prognosis.—The prognosis of carcinoma of the tongue is always unfavorable. When the disease is left to itself, it usually terminates the life of the patient in from one to two years.

Operation in the cases which are unmistakably carcinoma tends to relieve pain and distress, and prolongs life for a short time; but the end is nearly always recurrence in the remaining portion of the tongue, or secondary disease in the lymphatic glands of the neck. The prolongation of life by an operation has been variously estimated at from five to eight months. The operation itself is always a dangerous one; hemorrhage and septic pulmonary conditions being the chief causes of fatalities from the operation.

Treatment.—Radical operations for the treatment of carcinoma of the tongue are to be preferred to treatment by caustics. The application of caustics, as a rule, seems only to stimulate the proliferation of the "cancer-cells," and renders the growth of the tumor more rapid and malignant.

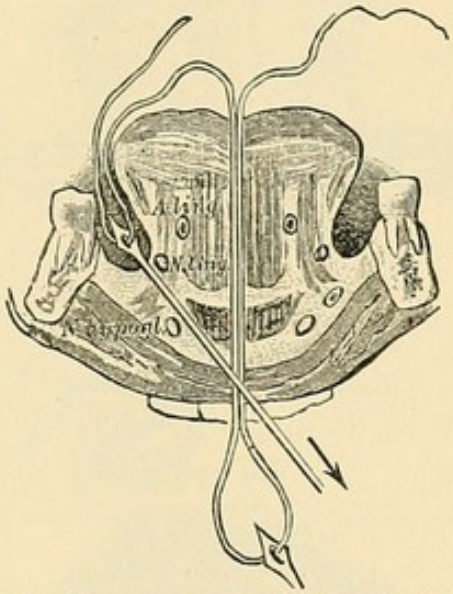
As a preliminary to all operations upon the tongue, Billroth recommended a careful disinfection of the entire cavity of the mouth. This process to be of real value will require a painstaking cleansing of the teeth by the removal of all salivary deposits and alimentary débris, followed by the free use of antiseptic solutions.

To draw the tongue forward, and to hold it in position during the operation, a stout ligature should be passed through the tip at the median line, and the ends tied.

Hemorrhage is often very troublesome in operations requiring partial or complete excision of the tongue; to guard against accidents from this cause many surgeons are in the habit of ligating one or both lingual arteries, or a temporary ligature may be so placed upon the tongue as to entirely prevent the bleeding during excision of any portion of the anterior half, or even two-thirds of the organ. This is accomplished by passing a well-curved needle armed with a strong double silk ligature, through the middle of the tongue, as near its base as possible. The ligature is then cut close to the eye of the needle, and each thread tied separately, one upon each side if the whole tongue is to be rendered bloodless. (Figs. 255 and 256.) If but one half is to be excised, the ligature need be tied only upon the side to be removed. The ligatures may be left long enough to extend outside the mouth, and the ends tied, as they are useful in drawing forward the root of the tongue and giving a good view of that portion of the organ, which is so necessary when the disease is located near its base.

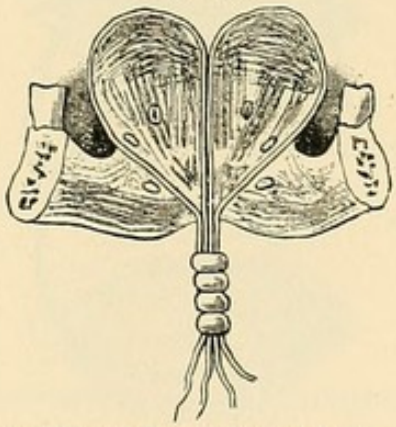
Senn advises the use of an elastic ligature in the form of a piece of small drainage tube, about twelve inches long, doubled and passed through an opening in the base of the tongue at the median line, made by tunneling the tissues with a pair of hemostatic forceps. The tube is to be cut in the center, and each piece tied at opposite sides of the tongue sufficiently tight to arrest all hemorrhage. The jaws must also be opened to their fullest extent by means of a suitable mouth-gag.

FIG. 255.



TEMPORARY CONSTRICTION OF ONE-HALF OF THE TONGUE. (After Esmarch and Kowalzig.)

FIG. 256.

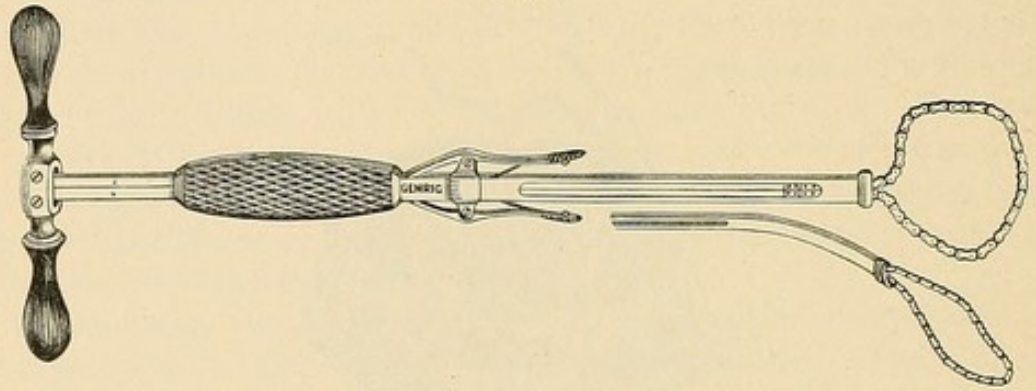


TEMPORARY CONSTRICTION OF THE WHOLE TONGUE AT ITS BASE. (After Esmarch and Kowalzig.)

The operation for partial or complete excision of the tongue may be done by several methods. On account of the hemorrhage which so often follows the use of the knife or scissors, the écraseur (Fig. 257) or the galvano-cautery wire were formerly substituted by many surgeons for this operation, but of late the great majority have gone back to the use of the knife or scissors, as the other means did not always prevent hemorrhage when the excision took place near the base of the tongue.

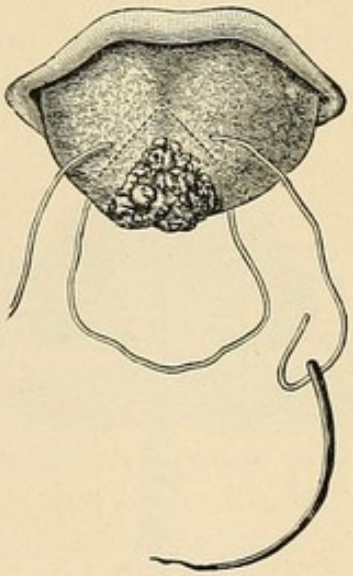
Small carcinomatous tumors may be readily removed through the mouth by a V-shaped incision made by a blunt-pointed bistoury or scissors, the portion to be removed having been previously seized with double-pronged forceps, and the tongue drawn well forward. All bleeding vessels are then secured, and the edges of the wound brought together with sutures. (Figs. 258, 259, 260, 261.)

FIG. 257.



CHASSAIGNAC'S CHAIN ÉCRASEUR.

FIG. 258.



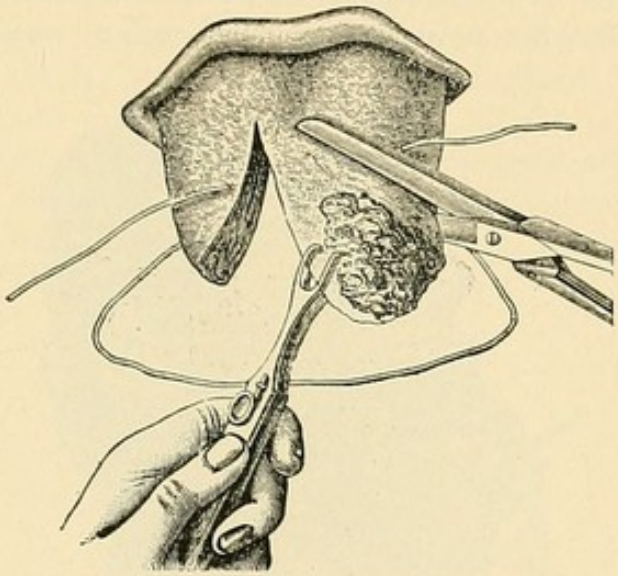
REMOVAL OF TUMOR OF THE TONGUE—INSERTION OF TRACTION-SUTURE. (After Esmarch.)

Operation through the mouth is applicable for the removal of the anterior third or half of the tongue. When a lateral half is to be removed it may be accomplished in the following manner: Two stout ligatures are passed through the tip of the tongue, one on each side of the median line, which are to be used to draw the organ forward; the tip is then raised, the frenum cut with scissors, and the tongue dissected from the floor of the mouth as far back as is necessary. The tongue is then split upon the median line, from before backward, freed

from the underlying parts by tearing with the finger, and the posterior section made with the knife or scissors.

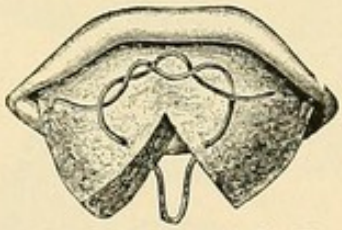
Whitehead removes the entire tongue through the mouth, but does not practice preliminary ligation of the lingual arteries; these vessels he secures as they are divided.

FIG. 259.



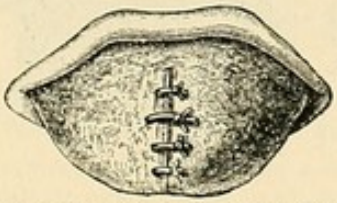
EXCISION OF TUMOR. (After Esmarch.)

FIG. 260.



TYING OF FIRST SUTURE. (After Esmarch.)

FIG. 261.



OPERATION COMPLETED. (After Esmarch.)

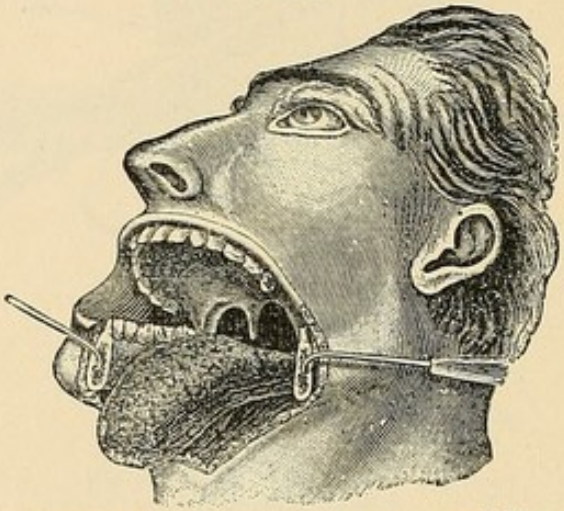
Langenbeck secures access to the base of the tongue by an incision made at the angle of the mouth and carried downward to the thyroid cartilage; through this incision any infected lymphatic glands, or the submaxillary, may be removed. Division of the digastric and hypoglossus muscles is then made, and the lingual artery tied. The jaw is next divided at the line of the incision, and the segments separated, after which the mucous membrane is severed from the inner sur-

face of the posterior segment as far back as the anterior pillar of the fauces. Through this opening it is not only possible to reach the base of the tongue, and amputate it at this point, but also to reach the tonsil and the soft palate, and remove them when necessary. (Fig. 262.)

Billroth modified this operation by dividing the soft tissues and the jaw at both angles of the mouth, and turning down the central segment.

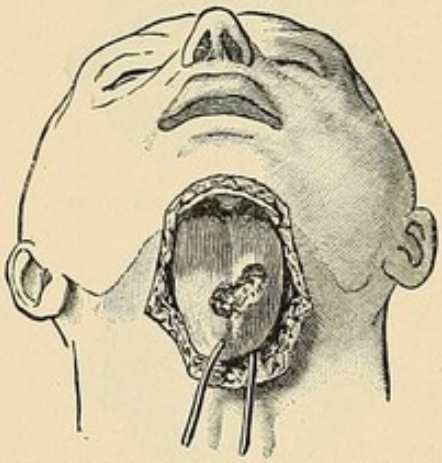
Regnoli devised a method in 1838, later modified by Billroth, which provides free access to the base of the tongue without division of

FIG. 262.



AMPUTATION OF THE TONGUE BY LANGENBECK'S METHOD.

FIG. 263.



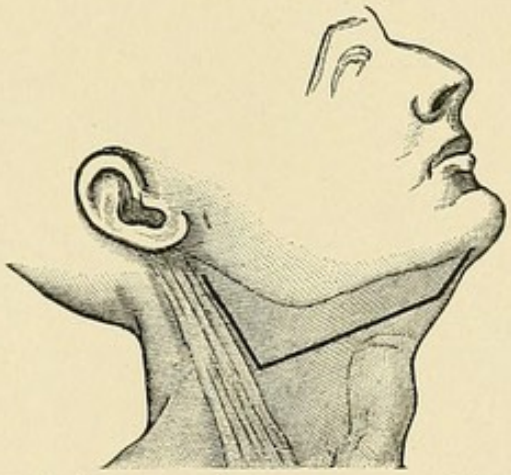
AMPUTATION OF THE TONGUE ACCORDING TO REGNOLI-BILLROTH.

the lower jaw. An incision is made along the inner surface of the lower border of the jaw from angle to angle, and the cavity of the mouth opened from beneath. The tongue is then seized and drawn down and forward, until its base is within easy reach. (Fig. 263.) This operation has the advantage of affording free access to infected glands, the establishment of adequate drainage, and of allowing the best antiseptic treatment of the wound.

Kocher's method is to make an incision "from the under border of

the lower jaw near the symphysis, in the direction of the anterior belly of the digastric muscle to the hyoid bone; thence backward to the anterior border of the sterno-cleido-mastoid muscle; then upward along it to or above the angle of the jaw." The flap is then raised as far as the lower border of the jaw, and an incision made through the floor of the mouth as close to the bone as possible, and through this opening the tongue can be drawn down and amputated. (Fig. 264.) Kocher's operation requires preliminary tracheotomy. The advantages of this operation are the facility with which infected glands may be reached and removed and the lingual artery tied. It also permits the packing of the fauces with sponges or gauze, thus preventing the entrance of blood into the trachea, and facilitates antiseptic treatment of the mouth. The tracheotomy tube should remain for several days, the patient to be fed through an œsophageal tube.

FIG. 264.



KOCHER'S INCISION IN AMPUTATION OF THE TONGUE.

Senn recommends the passing of two traction sutures through the base of the tongue, which are then brought out of the mouth, in cases of amputation of the entire organ, and are used afterward as a means of fixing the stump for a few days in its proper position. He thinks this is an exceedingly important precaution. The after-treatment consists of establishing drainage and as thorough antiseptic conditions as the nature of the case will permit. A saturated boric acid solution, or the Thiersch solution, are the most valuable antiseptics for use in these cases.

Carcinoma of the Salivary Glands.—Primary carcinoma of the salivary glands is not a disease of very common occurrence, but it is exceedingly malignant. As a primary affection it is more common in the parotid than in the other salivary glands.

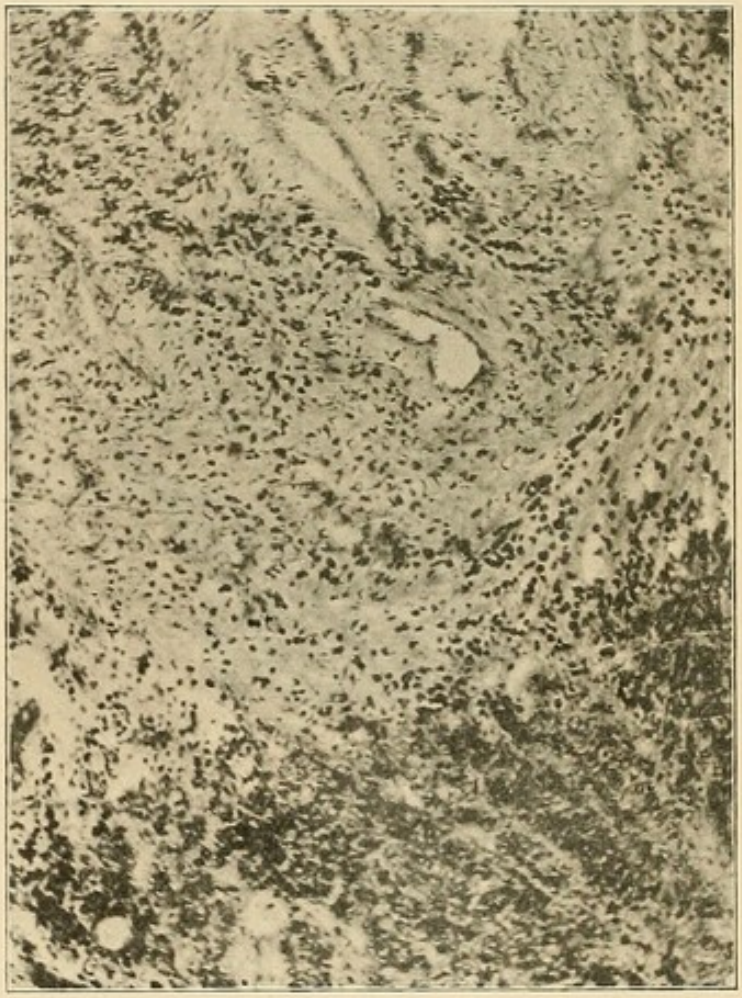
Secondary carcinoma of the submaxillary glands associated with carcinoma of the lower lip, the oral mucous membrane, the gums and

the tongue, are of common occurrence. The primary form of the disease is rarely seen in these or the sublingual glands.

Carcinoma of the parotid gland may be of either the acinous or the tubular variety.

In the acinous form the disease begins as a proliferation of columnar epithelial cells in an individual acinus or lobule of the gland. Its malignant character is marked by the scantiness of the stroma, its rapid growth, and the early infection of the lymph glands.

FIG. 265.



MIXED TUMOR OF PAROTID GLAND. (A.)
(Heterogeneous structure.) × 50.

The tubular variety begins in the tract of the salivary duct, and appears in the form of epithelial pearls or "cell-nests" of columnar epithelia, which arrange themselves in a manner similar to tubular glands. These tubules multiply and extend into the substance of the gland.

Carcinoma of the parotid gland does not appear until middle life. Its general clinical history is that of carcinoma of the face. In persons

of fifty years of age or more, a rapid-growing tumor of the parotid gland which infiltrates the skin, and finally ulcerates, is with few exceptions a carcinoma. Neoplasms of heterogeneous structures are more frequently seen in the parotid gland than tumors of a pure type. Figs. 265, 266, 267 are made from sections taken from three different locations in the same tumor, and nicely illustrate this point.

Treatment.—Carcinoma of the parotid gland calls for early and complete extirpation, with all surrounding infected tissues. The na-

FIG. 266.



MIXED TUMOR OF THE PAROTID GLAND. (B.)
(Heterogeneous structure.) × 50.

ture of the operation is somewhat serious, and requires, on account of the important vessels in the neighborhood, most careful dissection. The extirpation of the gland results in permanent paralysis of the face, and this result should be made perfectly clear to the patient before an operation is undertaken.

Senn suggests that when a large area of skin is infected, the diseased portion should be included within two elliptical incisions, the lower angle of which should be so placed that it will be directly over

the point at which the external carotid artery must be ligated. The temporal artery should be ligated upon the distal side of the tumor, and secured by compression forceps upon the proximal side. Careful dissection of the entire mass is required, and must be carried down to the styloid process. As soon as the external carotid artery is exposed it should be grasped with the hemostatic forceps, the tumor then removed and the artery tied afterward. The wound made by such an

FIG. 267.



MIXED TUMOR OF THE PAROTID GLAND. (C.)
(Heterogeneous structure.) × 50.

operation necessarily leaves a large opening which cannot be closed except by a plastic operation. This may be accomplished by taking a flap from the forehead or the scalp, leaving it attached at its base by a pedicle until union has taken place in its new position. The wound left by removing the flap should be at once covered by skin grafts after Thiersch's method. In those cases in which the skin can be preserved Senn exposes the gland by a curved incision, the convexity looking downward, and extending from the mastoid process to near the malar eminence. The tumor is then removed by the method just described.

REVIEW.

CHAPTER LII.

What is the character of the epithelium of the mucous membrane?
In what portions of the mucous membrane may carcinoma be located?
How many forms of carcinoma are found in the mucous membrane?
How do they differ from carcinoma of the skin?
What is the clinical history of the superficial variety?
Describe the cause of the deep-seated variety.
Where is this form of the disease most frequently located?
In what other locations is it found?
In what locations is it rarely seen?
By what conditions is carcinoma of the cheek sometimes preceded?
What is leucoplakia?
What is thought to be the cause of leucoplakia?
How may it terminate?
What are the active causes of carcinoma?
In which jaw is carcinoma of the mouth most common?
How may carious teeth and roots cause carcinoma?
How is the disease characterized in this location?
How does it progress?
What sinus may be involved when the disease is located in the upper jaw?
How are the teeth affected by the disease?
At what period of life does the disease occur?
What are the symptoms of carcinoma of the antrum?
Describe the clinical course of the disease.
When does regional infection take place?
What relation does the size of the ulcer bear to lymphatic infection?
What is the character of carcinoma of the cheek and gums involving the maxillary bones?
What is the average duration of life in this form of the disease?
Are they amenable to surgical treatment?
At what period in the history of the disease does surgical treatment give the best results?
What should characterize the surgical treatment?
Can the disease when affecting the maxillary bone be successfully removed through the mouth?
What rule should be followed in making the external incisions for removal of the disease in the upper jaw?
How may partial excision of the superior maxilla be made?
What lines of incision should be adopted in partial excision of the inferior maxilla?
What form of carcinoma is found in the tongue?
Where is it usually located?
Which part of the tongue is most frequently affected?
In what forms may the disease first make its appearance?
What is the microscopic character?
What are the exciting causes?
What is the character of the course of the disease?

How long does the patient usually live after the establishment of the disease?

What are the symptoms of the disease?

In which sex is the disease most common?

What is the proportion?

At what period of life does it appear?

What are the limits of age at which the disease has been noticed?

Between what ages does the largest number occur?

What diseases may be mistaken for carcinoma of the tongue?

How may syphilitic indurations be differentiated?

How may tuberculosis be differentiated?

How may actinomycosis be differentiated?

What is the prognosis of carcinoma of the tongue?

What is the value of operation in these cases?

How long is life prolonged by an operation?

What are the causes of fatalities following operation?

How does the application of caustics affect the disease?

What preliminary steps should be taken in all operations upon the tongue?

How guard against severe hemorrhage?

Describe the method of temporary ligation of the lingual arteries.

What is Senn's method?

What methods of operation are introduced to prevent hemorrhage?

Are these methods still practiced?

Describe the operation for removing small tumors.

When is operation through the mouth applicable?

What method may be employed to remove a lateral half of the tongue?

By whose method may the entire tongue be removed through the mouth?

How does Langenbeck secure access to the base of the tongue?

How does Billroth modify this operation?

Describe the Regnoli operation as modified by Billroth.

Describe Kocher's method.

What preliminary operation is required?

What recommendation does Senn make in reference to the stump of the tongue after amputation?

Is primary carcinoma of the salivary glands common?

What is its character?

Which of the salivary glands are most often affected with primary carcinoma?

Under what conditions is carcinoma of the salivary glands common?

What varieties of carcinoma are found in the parotid glands?

How does the acinous form of the disease begin?

What marks its malignant character?

Where does the tubular variety of the disease begin?

How are the epithelial cells arranged?

At what period of life does the disease occur?

What is its clinical history?

What should be the character of the operation in carcinoma of the parotid?

Is this a serious operation; and if so, why?

What condition of the face results from extirpation of the parotid gland?

How does Senn operate for extirpation of the gland when a large area of skin is involved?

How should the wound be closed?

What method does Senn employ when the skin can be preserved?

CHAPTER LIII.

MESOBLASTIC TUMORS.

Fibromata.

In the connective-tissue group of tumors are included all those neoplasms which arise from tissues developed from the *mesoblastic layer* of the germinal disk of Pander. The tumors which compose this group have their genesis in a matrix of misplaced connective-tissue cells, of embryonic type, of either pre-natal or post-natal origin. Tumors of this class may be, as with the neoplasms of the epithelial group, either benign or malignant in their character; innocency and malignancy depending upon the stage of differentiation reached by the tumor-cells. Cells of high differentiation will produce innocent tumors; cells of low differentiation will result in the development of malignant tumors. Under the head of benign mesoblastic tumors may be placed fibroma, lipoma, myxoma, chondroma, osteoma, angioma, neuroma, and lymphangioma. Some of these, however, have a tendency under favoring conditions to undergo sarcomatous transformation.

Under the head of malignant tumors of mesoblastic origin are placed the various forms of sarcoma.

The tumors of the mesoblastic type which are of most frequent occurrence in connection with the face, mouth, and jaws, are the fibromata, chondromata, osteomata, angiomata, and sarcomata. The other forms are so rare in these locations that their presentation may be omitted.

Definition.—*Fibroma* (Lat. *fibra*, a fiber, and Gr. ὄμα, a tumor).

"A Fibroma is a benign tumor, composed of mature fibrous tissue produced from a matrix of fibroblasts."

Fibromata are the most representative of the mesoblastic tumors, and are the most common. They are to be found in all parts of the body where connective tissue and blood-vessels form a part of the structure.

The chief locations of fibrous tumors are the periosteum, especially of the jaws, the skin, the uterus, the ovaries, the neurilemma of the nerves, the terminal or peripheral ends of nerves,—where they form

painful tubercles within the subcutaneous tissue,—the rectum, and the naso-pharynx,—where they form polypi.

Origin.—Fibromata have their origin in a matrix of congenital fibroblasts, which for some reason have been arrested in the process of differentiation during the development of the embryo, and have remained in a more or less embryonic condition in the connective tissue until some influence, either local or general, has stimulated their dormant energies and powers of cell proliferation into activity. It is thought by some authorities that the matrix may sometimes be of post-natal origin, as it frequently occurs that such tumors develop in the

FIG. 268.



FIBROMA—KELOID—IN THE LOBE OF THE PINNA, ASSOCIATED WITH AN EAR-RING PUNCTURE. (After Sutton.)

cicatrices following wounds and traumatic injuries of any form (Fig. 268), and in the regenerative process following suppurative inflammation.

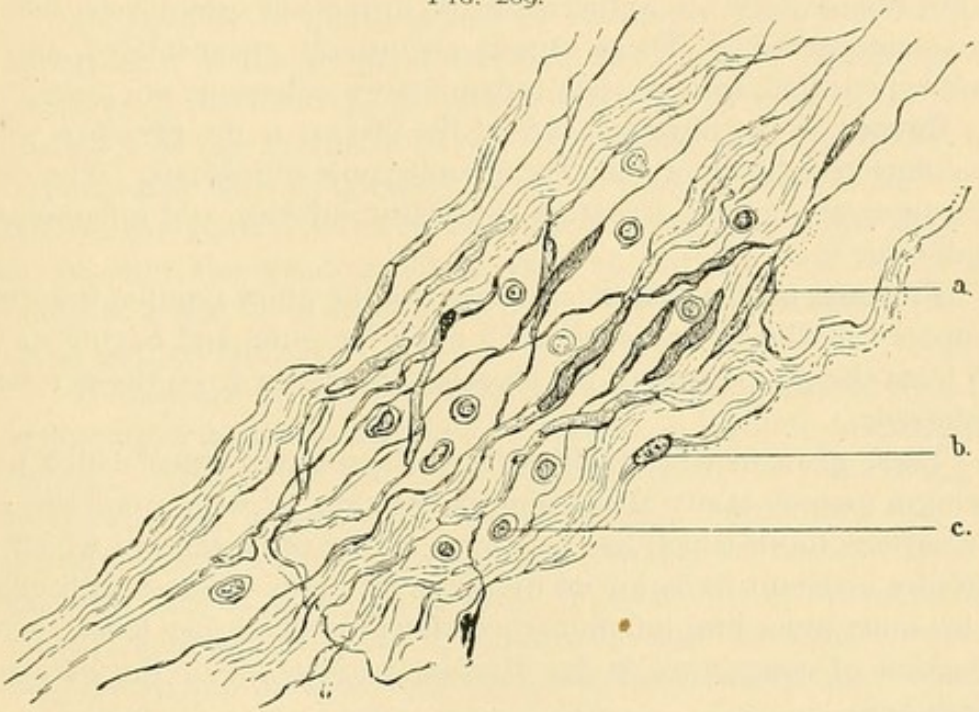
Ziegler says, "The fibromata are developed from proliferous connective-tissue cells."

Histologically, a fibroma is composed of interlacing bands or bundles of connective tissue, often showing upon the cut surface a concentric arrangement of the connective-tissue fibers in various parts of the tumor. These whorls are arranged around blood-vessels. The bundles are composed of long, slender, fusiform or spindle cells, closely packed together (see Fig. 269). They usually possess a distinct capsule, which renders it an easy matter to enucleate them.

Fibromata may arise from either the periosteum or the endosteum. Fibrous epulides are usually periosteal or peridontal in their origin, while antral fibromata are endosteal growths arising from the muco-periosteum lining the maxillary sinus.

Varieties.—Fibrous tumors are sometimes dense and firm as a tendon,—known as *hard fibroma;* at others soft and spongy, and desig-

FIG. 269.



FIBROUS TUMOR FROM THE ANTRUM OF HIGHMORE. × 450. (After D. J. Hamilton.)
a, fusiform nucleus; *b*, younger nucleus of an oval shape; *c*, isolated fibroblast.

FIG. 270.



HARD FIBROMA FROM FASCIA OF RIB. (After Lücke.)

nated as *soft fibroma.* In the former variety, the intercellular tissue is very scanty, and the fibrillæ are arranged in compact wavy bundles, or in whorls. (Fig. 270.) On section they are smooth, glistening, dense, and of grayish-white color. The latter variety, known as *soft fibroma,* consists of a more or less loose, spongy, fibrous tissue, abundantly supplied with blood-vessels. Upon the character of the fibrous tissue which enters into the formation of these growths will depend the general appearance of the tumor, which may be more or less yellowish,

glistening, semi-transparent, or gelatinous. The ordinary nasal polypus is a typical illustration of this form of fibroma. Degenerative changes frequently take place in these tumors, viz: myxomatous, calcareous, colloid, ulcerative, and sarcomatous. Occasionally changes of a higher degree take place. In fibrous tumors arising from the periosteum or the endosteum, ossification is most frequently seen. Whether this is really the result of changes produced by tendencies of the tumor itself, or is the result of misplaced osteoblasts, has not been demonstrated. These tumors are usually encapsulated, and are easily enucleated, except when inflammatory adhesions are present.

Causes.—The *essential cause* of the disease is the presence within the connective tissue of a matrix of embryonic fibroblasts. The exciting causes are chronic irritation, traumatic injuries, and inflammatory conditions.

Fibroma of the Gums.—Fibroma of the gums (epulis) is a tumor composed of fibrous tissue, situated upon the gum, and having its origin from the periosteum of the alveolar process or from the peridental membrane.

These growths will usually be found associated either with a tooth having a carious cavity at the gingival margin, or with a retained root of a carious tooth which has been covered by the gum, but which has caused a constant irritation of the gingival tissue by its rough edges; or by some preceding inflammatory affection which has left a chronic condition of congestion in the alveolar periosteum or the peridental membrane.

Tumors of this character are of slow growth, painless, and usually composed of firm fibrous tissue, covered with the gingival mucous membrane. (See Fig. 250.) In form they may be sessile or pedunculated, usually the latter. In size they may vary from a walnut to a man's fist. Sessile tumors of large size exert great pressure upon the alveolar arches, changing the form of the maxillæ, and crowding the teeth out of position. The lips are sometimes protruded, the cheek distorted, and when located in the region of the posterior molars, the palate may be encroached upon. In the pedunculated form the tumor may attain such a size as to preclude the possibility of closing the mouth.

The writer once assisted in the removal of such a tumor, which was located between the first and second inferior left molars, upon the buccal aspect of the gum, in a girl thirteen years of age. The tumor was as large as a Messina orange, somewhat pear-shaped in form, and attached at the small end by a narrow pedicle.

Fibromata associated with the gums and the teeth are of frequent occurrence. The simple fibroma is the most common, and it rarely reaches a size larger than a walnut. Pure fibroma is occasionally seen

in this location, but in the experience of the writer, fibro-angioma, fibro-chondroma, and fibro-osteoma are much more common. Fibrous tumors in this location are prone to ossification. Degenerative changes also frequently occur in them, ulceration and sarcomatous transformation being the most common.

The disease is one which occurs in early life, and is rarely seen after the thirty-fifth year.

Diagnosis.—In the diagnosis of fibroma of the gum, the fact that hyperplasia of the gingival festoons and fungoid conditions of the dental pulp (hernia) may so closely resemble fibroma as to be readily mistaken for that tumor, must not be overlooked. Hyperplasia of gum festoons may be distinguished by its very broad pedicle, and by its retaining the general form of the festoon. Fungoid pulps (hyperplasia of the pulp, the result of hernia) may be distinguished by the growth being attached by a constricted pedicle, which arises from the central canal of the tooth.

Fibroma of the gums is usually a hard tumor covered by a healthy appearing mucous membrane; its surface is smooth, sometimes glistening. When large they may become injured by the closing of the teeth, and the injury result in ulceration. Sometimes they have a purplish color, showing an abnormal supply of blood-vessels, when they are classed as fibro-angioma. Tumors of this character are frequently erectile. The fibro-chondromata and the fibro-osteomata are very dense, and the latter contain numerous small spiculæ of bone.

Prognosis.—The prognosis of fibroma of the gum is favorable. The character of the tumor is benign. But in a tumor of long standing there is a possibility that a sarcomatous degeneration may have set in, consequently the opinion in such cases should be guarded until a positive diagnosis can be made by the microscope. In simple fibroma, recurrence does not take place after excision.

Treatment.—Local remedies for the cure of fibroma, which stimulate the absorbents, are worse than useless, as they only cause irritation; and irritation may result in sarcomatous degeneration. Radical cure of a fibroma of the gums can only be effected by excision of the alveolar process of the jaw. To accomplish this operation it is first necessary to extract a tooth upon each side of the tumor, and then with a small Hey's saw (Fig. 271), or small circular saw, revolved by the surgical engine, divide the bone to the base of the alveolar process upon each side of the tumor; then by a horizontal incision of the bone with the circular saw or a metacarpal saw uniting the perpendicular incisions, the section is removed; the same end may be accomplished with chisel and mallet.

Resection of the jaw is only admissible when the character of the tumor is undoubtedly malignant.

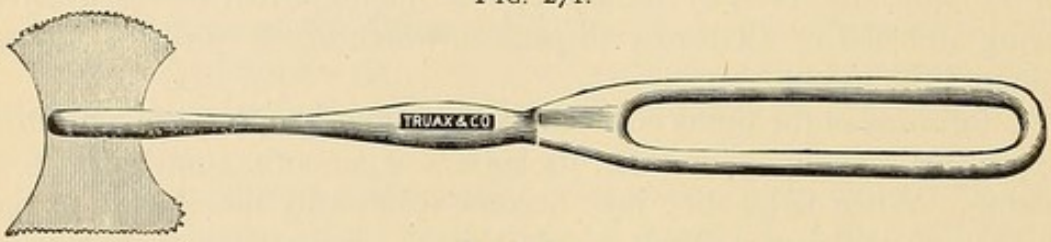
Antiseptic mouth lotions, used with persistence, is the only treatment of the wound required.

Hemorrhage may sometimes be troublesome, but this is usually controlled by packing the wound.

Fibroma of the Jaws.—Aside from fibroma located upon the gum, fibromata of the jaws are rare. The maxillary periosteum and bone are the most frequent location of fibroma. Fig. 272 is made from a plaster cast of a tumor of this character located in the lower jaw.

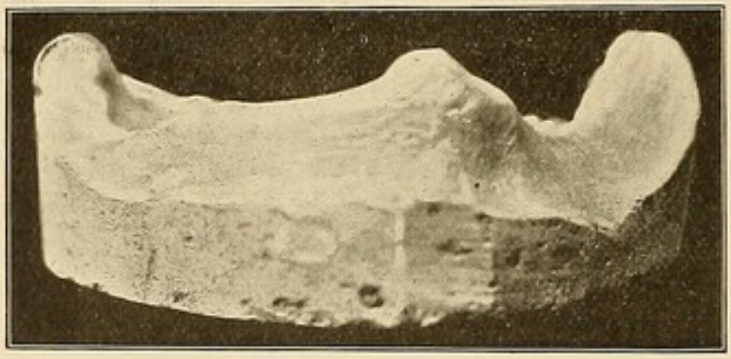
These tumors usually arise from one or the other of two situations, either the maxillary sinus or the alveolar processes of either jaw. Tumors of this class located within the antrum are capable of causing great destruction of surrounding bony tissue by pressure-atrophy.

FIG. 271.



HEY'S SAW.

FIG. 272.



FIBROMA—SESSILE—OF THE LOWER JAW.

Fibromata in this location are usually very slow in their growth, and their presence is not recognized until the neoplasm has filled the antrum, and has caused expansion and thinning of the walls of the sinus. They may also encroach upon the nasal passages, and completely fill them. Senn reports such a case, which is illustrated by Figs. 273, 274, 275.

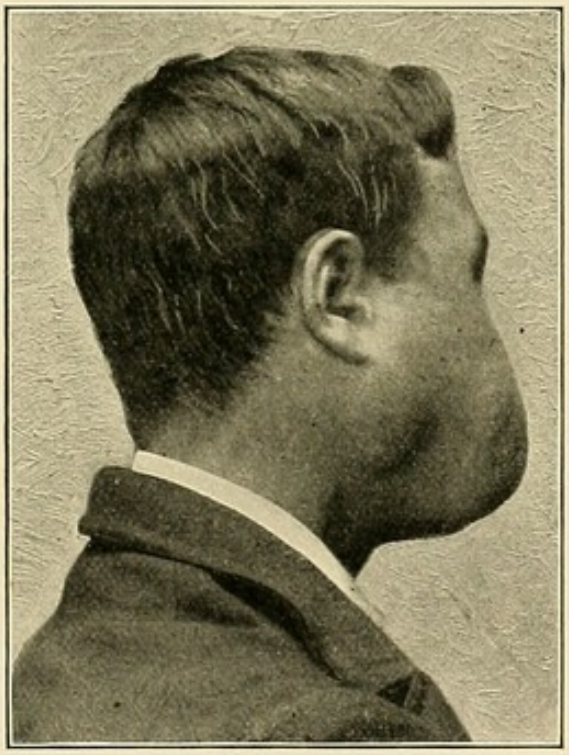
Heath describes a case in which the tumor "projected upward into the orbit, destroying the floor of that cavity, and protruding from its inner margin forward into the cheek. It had also destroyed the anterior wall of the antrum, and displaced the malar bone forward and outward; inward it projected into the nose beneath the middle

turbinated bone, and downward it made its appearance on the surface of the alveolar process in the form of a rounded mass, destroying the floor of the antrum in the neighborhood of the anterior molar teeth. Behind, the tumor appeared in the zygomatic fossa by the absorption of the outer surface of the tuberosity of the superior maxillary bone."

Liston and Paget both record cases of similar character.

Fibrous tumors arising from the alveolar process of the upper maxilla sometimes attain such size as to encroach upon the facial and palatal surfaces, causing pressure-atrophy, and crushing in the walls of the antrum, though not actually involving the sinus. On the other hand, although the tumor is located upon the alveolar process, it may secondarily involve the antrum, destroying its walls, enter the nasal fossa and penetrate the palatal process, projecting into the mouth.

FIG. 273.



ENORMOUS FIBROMA OF THE SUPERIOR MAXILLA. (A.) (After Senn.)

Tumors of this character arising from the alveolar process of the lower jaw may attain a very considerable size, causing loss of bone-tissue by pressure-atrophy, and great deformity of the face. A growth of this character located in the anterior portion of the lower jaw, which came under the observation of the writer several years ago, had so displaced the six anterior teeth and the bicuspids of the right side that they projected at a right angle to their normal position outward and beyond the lower lip. The tumor seemed to grow from the lingual surface and base of the alveolar border, and in its growth the entire

alveolar process in that location seemed to be lifted up and rolled outward. The privilege of taking a photograph or a cast of the mouth was declined by the patient.

Fibromata of the jaws associated with the alveolar process are usually sessile, generally lobulated and round or oval in form, covered by the gum-tissue of the part, and are slow of growth and painless unless irritated. Tumors of this class located in the jaws do not as a rule cause local infection nor metastatic deposits.

Diagnosis.—In fibroma of the antrum it is by no means always possible to make a correct diagnosis until after the removal of the tumor. Pain and tenderness are usually absent except when the tu-

FIG. 274.



DISTORTION OF THE DENTAL ARCH CAUSED BY ENORMOUS FIBROMA. (B.) (After Senn.)

mor is intimately associated with the trunk of a nerve of sensation, upon which it causes pressure, or when it is the subject of infection and inflammation. The great difficulty in the diagnosis arises from the similarity in the symptoms of fibroma, chondroma, and slow-growing sarcoma.

Prognosis.—The prognosis is favorable even though extensive

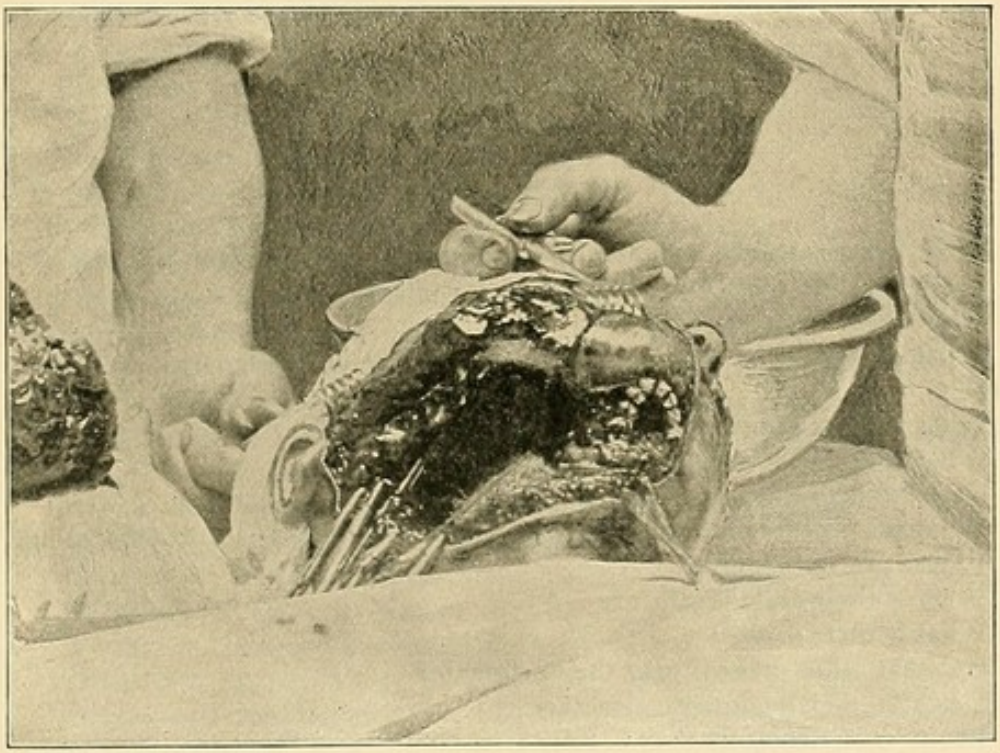
loss of bone-tissue may have resulted. Recurrence does not take place after enucleation has been performed.

Treatment.—Surgical treatment consists in the entire removal of the growth. This may be accomplished by enucleation.

Enucleation of a fibroma of the superior maxilla is best accomplished by external incision, which must be governed as to location and extent entirely by the size of the tumor. In operations upon the inferior maxilla for the removal of a large tumor, care should be exercised to leave at least a narrow rim of bone at the base of the jaw rather than to make a complete exsection of the bone. The advantages of this will be readily understood.

Small tumors may be removed through the mouth.

FIG. 275.



ENORMOUS FIBROMA OF THE UPPER MAXILLA, SHOWING CONDITION OF PARTS IMMEDIATELY AFTER EXCISION OF TUMOR. (C.) (After Senn.)

Fibroma of the Skin.—Fibrous tumors frequently appear upon the face, neck, and trunk of the body. Their growth is very slow and painless, and they rarely exceed the size of a filbert nut. They appear first as enlargements in the connective tissue of the skin; as they grow the skin is elevated by the tumor, which projects more and more until the skin at its base becomes constricted. The weight of the tumor causes elongation, which results in the formation of a pedicle. The tumor contains in its center the principal artery, which sometimes, in consequence of an injury or of textural change (Senn), becomes stran-

gulated by a thrombus, when gangrene results, and the tumor is cured spontaneously.

Fibrous tumors of the face are most often sessile, and are easily enucleated by an incision over the surface or at the base.

A mole is a flat fibroma of the skin, of congenital origin. These tumors vary in size from a pin-head to growths three to four inches in diameter. The increase in size progresses until puberty, when they usually become stationary. It has long been recognized that these growths are very prone to take on carcinomatous and sarcomatous degenerations. As a consequence of this tendency, their early removal should be advised. If the area of skin is large that must be sacrificed in their removal, the defect can be remedied by a skin flap or Thiersch skin-grafts.

REVIEW.

CHAPTER LIII.

What neoplasms are included in the connective-tissue group of tumors?
From what do these tumors have their origin?
How are they classed?
What condition of structure gives innocency or malignancy to these tumors?
What tumors are classed as benign?
What tumors are classed as malignant?
Give the definition of fibroma.
What form of tumor is the most representative of the mesoblastic group?
Where are they found?
What is their chief location?
What is their origin?
By what other process may they originate?
Describe their histologic structure.
From what tissues do they usually arise?
From what tissues do fibrous epulides arise?
What is the origin of antral fibroma?
How are the fibromata classed?
Describe the hard fibroma.
Describe the soft fibroma.
What degenerative changes may take place in these neoplasms?
In which variety is ossification most likely to occur?
What is the essential cause?
What is the exciting cause?
From what tissues do fibromata of the gum arise?
With what conditions of the teeth are they usually associated?
What is the character of their growth?
Of what tissue are they composed?
What is said of their form and size?

Are such tumors common or rare?
What variety is the most common?
What degenerative change is apt to occur in these growths?
At what period of life do they most frequently occur?
How may these growths be differentiated from hyperplasia of the gum festoons?
How differentiated from fungous growth of the pulp?
What is the appearance of fibroma of the gums?
What other varieties are often seen?
Describe the appearance of fibro-angioma.
What is the character of a fibro-osteoma?
What is the prognosis?
Is this tumor benign or malignant?
What form of degeneration may occur?
Does recurrence take place after operation?
Is local treatment of value?
How may a radical cure be effected?
Describe the operation.
Under what conditions is resection of the jaw admissible?
How may hemorrhage be controlled?
In what tissues do fibromata of the jaws originate?
In what particular parts are they usually found?
What is the character of their growth?
What sometimes results from fibroma of the antrum?
What may be the extent of the deformity?
What may be the extent of the growth and deformity when the tumor arises from the alveolar processes of the upper jaw?
What is the result of tumors of this variety located in the lower jaw?
Is the diagnosis of fibroma of the antrum difficult to make?
What is the difference between the growth of fibromata and malignant tumors?
Under what circumstances are fibromata painful and tender?
What causes the difficulty in the diagnosis?
What is the form of fibrous tumors associated with the alveolar processes?
What is the character of their growth, etc.?
Do fibromata cause glandular infection?
What is the prognosis of fibroma of the antrum?
Is the prognosis equally favorable when the neoplasm is located in the alveolar processes?
What should be the surgical treatment of these forms of fibroma?
What should govern the location and extent of the external incision?
In removing large growths from the lower jaw, what method is advised?
In what locations are fibromata of the skin most common?
What is the character of their growth?
Describe their appearance.
How may spontaneous cure take place?
What is the shape of fibroma of the face?
What term is applied to the common form of fibromata of the face?
What is the variation in their size?
When is their growth most active?
What forms of degeneration occur in these growths?
When should they be removed?
How should large wounds be covered?

CHAPTER LIV.

CHONDROMATA.

Definition.—Chondroma (χονδρος, cartilage, ομα, tumor).

Chondroma is a cartilaginous tumor,—a tumor consisting of cartilage.

Chondromata are tumors composed histologically of hyaline cartilage. They occur in locations, principally, where cartilage is normally found, viz: associated with the bones and within the cartilaginous structures of the respiratory organs. They are also occasionally found in locations where cartilage has no normal existence. In the former case the growth may be due, as pointed out by Virchow, to the presence of embryonic or untransformed portions of cartilage remaining in the bones, and which later take on active cell-proliferation, thus becoming the starting-point of a tumor. The latter condition is explained by the modified theory of Cohnheim, of a misplaced matrix of embryonic chondroblasts in tissues where they have no legitimate presence, as, for instance, in the parotid gland, ovary, etc.

Typical chondromata are found in the long bones, usually in relation with the epiphyseal cartilages; consequently they occur most frequently in growing children and in young adults. The long bones of the hands and feet are especially liable to the affection. Fig. 276 illustrates a remarkable case of this character published by Stendil.

Chondromata may be described as slow-growing, painless tumors, firm to the touch, and always encapsulated. During their growth they displace the soft tissues, and cause absorption of the bone from which they spring, fashioning for themselves large cavities in which they rest.

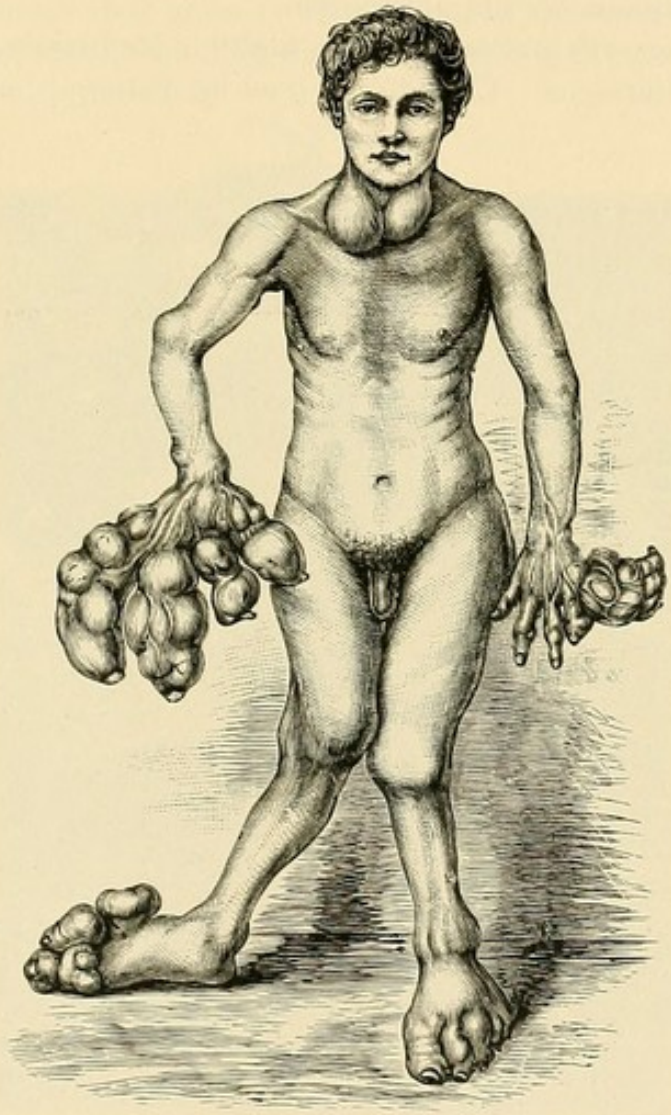
Calcareous, mucoid, and myxomatous degenerations frequently occur in the cartilaginous tumors. They are prone to ossification and to sarcomatous transformation. For the latter reason these tumors have always been looked upon with more or less suspicion. Fig. 277 illustrates the histologic structure of an ossifying chondroma.

Injuries of a traumatic nature seem to be most prolific in causing the disease. Rachitis is a frequent exciting cause. Sutton observes that "it is a curious circumstance that the tissue of a chondroma re-

sembles, histologically, the bluish, translucent epiphyseal cartilage characteristic of progressive rickets."

A chondroma consists of cartilage and connective tissue; the connective tissue, however, is found in limited quantity. The tumor is composed of numerous lobes of varying size, which are separated from one another by the connective tissue. Occasionally the fibrous tissue

FIG. 276.



CHONDROMATA—MULTIPLE—IN LAD TWENTY YEARS OF AGE. (After Stendil.)

is largely in excess of the cartilage. Such tumors are termed fibrochondromata. In size, form, and numbers the cartilage cells vary greatly in different tumors, and also in different locations in the same tumor. (Warren.) The tumor grows by additions to its external surface. When ossification takes place, it usually begins at the center of the tumor. These growths are sometimes inclosed within a real bony layer or capsule derived from the bone-tissue in which they are formed.

Diagnosis.—A chondroma may be recognized by its lobulated form, which increases with the growth of the tumor, by its density, except in locations where mucoid or colloid degenerations have taken place, when fluctuation may be present; by the early age at which they appear, and the tendency which they exhibit to become stationary at the age of puberty, and by the slow growth of the tumor and the absence of pain and tenderness.

The disease in the long bones is very often multiple, though individual chondromata are not uncommon.

Its slow growth and tendency to multiple formations differentiate it from osteo-sarcoma. Chondroma may be distinguished from oste-

FIG. 277.



CHONDROMA—OSSIFYING. × 50.

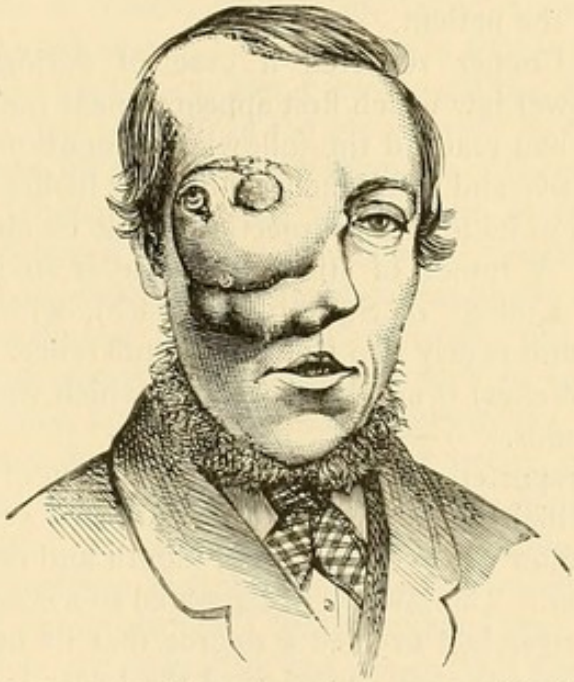
oma by the use of the exploring needle; chondromata will permit the passage of the needle, while osteomata will not.

Prognosis.—Pure chondromata are benign tumors, and but for the tendency of these growths to take on sarcomatous transformation the prognosis would be favorable. Recurrence sometimes takes place after the removal of tumors located in the maxillæ, which casts a suspicion that they may be sarcomatous.

Chondroma of the Jaws.—Chondroma of the bones of the face is a somewhat rare affection, but when it does occur it is most often associated with the superior maxillary bones. Chondroma of the inferior maxilla is exceedingly rare. The disease when associated with the superior maxillæ may arise from the surface of the bone, or from within the antrum. In the former case it has its origin in the *periosteum* of the jaw; in the latter it is derived from the *endosteum,* which lines the cavity of the antrum.

Heath describes four cases of chondroma of the upper jaw, and four post-mortem specimens. In three out of the four cases operated

FIG. 278.



CHONDROMA OF THE UPPER JAW.—RECURRENT. (After Heath.)

upon, the disease originated in the antrum of Highmore; and of the four post-mortem specimens two had their origin in this sinus.

Chondroma of the antrum has a tendency to extend into the accessory sinuses, sometimes completely filling the nasal cavity, the frontal sinus, and encroaching upon the orbit, dislodging the eye, with loss of sight. Fig. 278 is a case of this character reported by Heath. In two out of the eight examples mentioned, the tumor penetrated the cranium.

Tumors of this class may reach an immense size, as in the case of Shaughnessy (Heath), who removed a cartilaginous tumor, together with the superior maxillary bone, from a Hindoo patient, which weighed four pounds.

Chondroma of the inferior maxilla is observed in two forms, the *endosteal* and the *periosteal.* In the endosteal variety the tumor occu-

pies the cancellated portion of the bone, being covered by the external plate or compact tissue of the jaw. The periosteal form is located upon the external surface of the bone, and is covered by the gum-tissue, and for these reasons it becomes exceedingly difficult to diagnose it from the hard variety of fibroma. In the endosteal variety it is also difficult to arrive at a diagnosis on account of the similarity in the clinical history of osteo-sarcoma of the same location. The periosteal variety may attain very large dimensions, and even cause death by so filling the mouth and crowding the tongue into the fauces as to interfere with respiration and deglutition.

The endosteal form of the disease does not reach such large dimensions, but it frequently causes such enlargement of the jaw as to greatly disfigure the patient.

Sir Astley Cooper reported a case of periosteal chondroma (Heath) of the lower jaw which first appeared near the chin, and which in a year's time had reached the following dimensions: From side to side it measured five and a half inches, and four inches from the incisor teeth to the limit of its forward projection, while its circumference was sixteen inches. A tumor of still larger size is to be found in the museum of the College of Surgeons (Heath), which measures six inches in depth and nearly two feet in circumference. In the Heidelberg Museum (Weber) is another specimen which weighs about three and one-half pounds.

Briggs has reported a case of osteo-chondroma of the lower jaw of immense size, which had a history of twenty-two years' growth. A portion of the tumor projected from the mouth and measured fully six inches in diameter. The jaws were separated to a considerable extent, and the mouth stretched to such a degree that its original form was completely lost, and the only portion of the lower face that could be seen was the upper surface of the everted lower lip. The tumor was very dense, irregularly round in shape, and occupied almost the entire cavity of the mouth. The tongue was doubled up and forced backward. The teeth, with the exception of two or three roots, were lost. After removal of the growth it was found to be irregularly pear-shaped, with an average diameter of seven and a half inches, and a circumference of about twenty-three inches in its largest part.

The clinical history of chondroma of the jaws is so similar to that of fibroma in this location that it is exceedingly difficult from the general appearance and the progress of the disease, to differentiate between them. Two points in the history of chondroma may assist in the diagnosis: It grows more rapidly than fibroma, and shows a greater tendency to recurrence.

Heath records a case from the practice of Lawson in which ten operations were made during eighteen years for recurrence of a chon-

droma in the lower maxilla of a woman fifty-seven years old at the last operation. The tumor weighed eighteen ounces.

Virchow reports a similar case in which the tumor was removed seven times at varying intervals of six months to two years.

Treatment.—The treatment of chondroma of the jaws differs little from the treatment of fibroma, except that on account of its tendency in some cases to recur, and its liability to sarcomatous transformation, greater care should be exercised in the operation for its removal. Better include a portion of healthy tissue surrounding it and secure immunity, than to invite recurrence by a timid operation.

Occasionally it may be necessary to perform partial or complete exsection of the maxillæ in order to thoroughly extirpate the tumor. In the majority of chondromata of the jaws, enucleation can be practiced; but to guard against recurrence, it is well to curette the surface, particularly all existing cavities communicating with the seat of the tumor, for fear that portions of the growth may be hidden within them.

Chondroma of the Salivary Glands.—Chondromata of the soft tissues differ from those having their origin in connection with the bones and epiphyseal cartilages, in that the tissue of which they are composed is fibro-cartilage, although occasionally hyaline cartilage is found in these tissues. The parotid and the submaxillary glands are among the most common locations in which these tumors are found. The parotid gland, however, is more frequently the seat of the affection than the submaxillary. Bryant observed twelve cases of chondroma in the soft tissues. Of these nine occurred in the parotid gland, two in the submaxillary, and one in the leg. Senn claims that chondroma is found more frequently in connection with the salivary glands than any other form of benign tumor. Sutton classes the chondromata of the parotid gland as sarcomata, for the reason that the spindle-celled sarcomata found in this location are very apt to contain cartilage. These neoplasms, according to Ziegler, are very prone to exhibit a mixed type of structure, and cartilaginous, mucoid, sarcomatous, and fibrous elements may all occur within the same tumor. (Figs. 265, 266, 267.)

Their origin has already been suggested as dependent upon a matrix of misplaced embryonic chondroblasts. Some authorities are of the opinion that these tumors arise from the connective tissue. Sutton says, "It appears to be an extremely easy task for connective tissue to form hyaline cartilage." Lücke and König believe (Senn) that the tumor may spring from the capsule of the gland, or from the surrounding connective tissue.

These neoplasms are of slow growth, and they may vary in size from a pea to a walnut. They are encapsulated, and hence movable. In form they are lobulated, and in their growth do not involve the surrounding tissues, but push them aside.

A pure chondroma of the salivary glands is rare. Cartilaginous tumors of the parotid are prone to sarcomatous transformation.

Treatment.—The treatment of chondroma of the salivary glands is enucleation. Especial care should be exercised in the operation for the removal of a parotid tumor, to avoid injury to the facial nerve, or cutting Stenson's duct, as paralysis might result from the former, and salivary fistula from the latter. Complete removal of the tumor is necessary to avoid recurrence of the growth, and the possible danger that recurrence may be accompanied by sarcomatous degeneration.

REVIEW.

CHAPTER LIV.

Give the definition of chondroma.
Of what are chondromata composed?
In what locations do they occur?
How does Virchow explain their origin?
By what theory is the presence of these growths explained?
Which occur in locations where cartilage has no normal existence?
Where are typical chondromata usually found?
In what class of individuals do they most frequently occur?
What particular bones are most liable to the disease?
How may chondromata be described?
What forms of degeneration most commonly occur in cartilaginous tumors?
What other changes often take place in this form of tumor?
What are the most prolific causes of the disease?
What disease is frequently an exciting cause?
What is the histologic structure of chondroma?
How do these tumors grow?
At what point does ossification usually begin?
How may a chondroma be recognized?
What differentiates chondroma from osteo-sarcoma?
How may chondroma be differentiated from osteoma?
What is the prognosis in chondroma?
If the tumor recurs after its removal, what suspicion would be aroused as to its nature?
Are chondromata of the bones of the face rare or common affections?
With which jaw are they most often associated?
In what directions are chondromata of the superior maxillæ most likely to occur?
From what tissue does the former arise?
From what tissue does the latter arise?
In which of these locations have chondromata most frequently been found?
What tendency does chondroma of the antrum exhibit in its growth?
What is the tendency as to size?
In what forms is chondroma observed in the inferior maxilla?

What portion of the bone does the endosteal variety occupy?

What is the location of the periosteal variety?

What are the difficulties in the diagnosis of these forms?

How may the periosteal variety become a menace to life?

Does the endosteal variety ever attain such dimensions as to disfigure the patient?

What is the clinical history of chondroma of the jaw?

What two points in the history of chondroma may assist in the diagnosis?

Does chondroma exhibit a tendency to recurrence?

What is the treatment of chondroma of the jaws?

What tendency is exhibited by chondroma which requires great care in its removal?

By what operation may it usually be removed?

How should the cavity be treated after the enucleation of the tumor?

How do chondromata of the soft tissues differ from those arising from bone and cartilage?

What are the most common locations of chondroma of the soft tissues?

Which of the salivary glands is most frequently the seat of this form of tumor?

What are Bryant's statistics in relation to the location of chondroma of the soft tissues?

How does Sutton class chondroma of the parotid gland?

What types of mixed structure are these neoplasms prone to exhibit?

What do Sutton and others say of their origin?

What is the character of the growth, etc., of these tumors?

What form of degeneration is prone to occur in chondroma of the parotid gland?

What is the treatment of chondroma of the salivary glands?

What especial care must be exercised in operating upon the parotid gland?

What should be the character of the operation as to thoroughness, etc.?

CHAPTER LV.

OSTEOMATA.

Definition.—Osteomata (Gr. ὀστέον, bone; ὄμα, tumor).

An osteoma is an osseous tumor; a tumor consisting of osseous tissue.

Senn defines an osteoma as "a tumor which possesses a structure resembling that of cancellated or compact bone, produced from a congenital or post-natal matrix of osteoblasts." Sutton defines them as "ossifying chondromata."

The osteomata include all tumors composed of bone-tissue. These formations have received different names, according to their location and the character of their structure. *Hyperostosis* is a diffused and extensive outgrowth in a bone. An *osteophyte* is a small local new-formation growing from pre-existing bone-tissue; when of larger size and assuming a more tumor-like form, it is termed an *exostosis*. A bony growth occurring within the interior of bones is designated as *endostosis*. "Bony growths which are not rigidly connected with the bone are divided into *mobile periosteal exostoses*, which are seated on the periosteum, though separate from the bone; *parosteal osteomata*, placed near to the bone, but not connected with it; *independent osteomata*, remote from the bone and seated in tendon or muscle; and, finally, the strictly *heteroplastic osteomata*, which may be seated in the lungs, cerebral membranes, diaphragm, skin (rarely), parotid gland" (Ziegler), testicle, ovary, and eyeball.

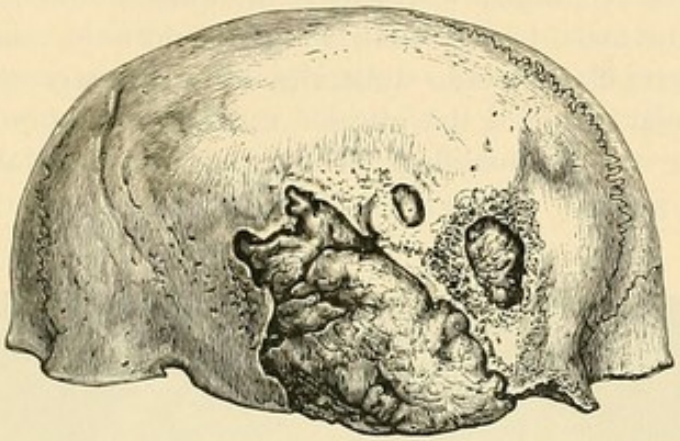
Sutton divides the osteomata into three classes:

1. Compact osteomata.
2. Cancellated osteomata.
3. Exostoses.

Compact Osteomata.—These tumors are histologically identical with the compact tissue of the shaft of long bones. This variety of tumor may occur in any part of the skeleton, but it is most frequently found in the bones of the skull, frontal sinus (Figs. 279, 280), the external auditory meatus, and the mastoid process, and it is occasionally seen upon the nasal process of the superior maxilla, the malar bone, and the body of the inferior maxilla. Tumors of this class are frequently designated as *eburnated osteomata*, or *ivory exostoses*. They

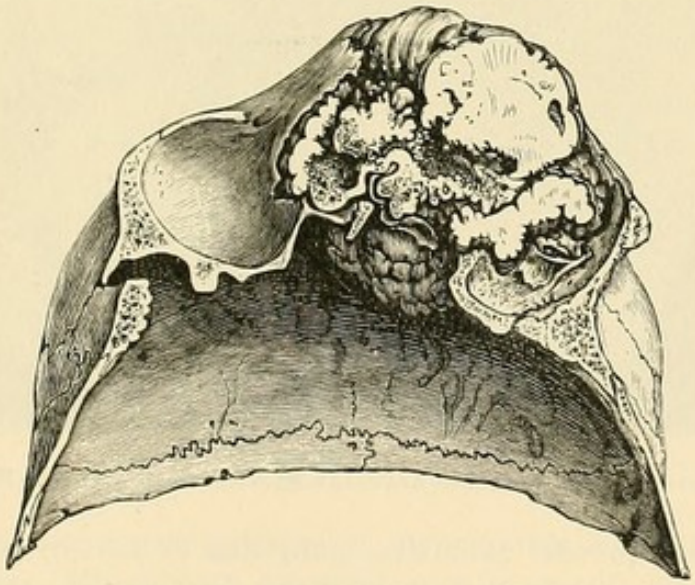
consist, histologically, of thick, ivory-like osseous tissue, having a concentric lamellar arrangement; the bone-corpuscles are so arranged in the lamellæ that their prolongations are directed toward the periphery

FIG. 279.



OSTEOMA OF THE LEFT FRONTAL SINUS—ANTERIOR VIEW. (After Sutton.)

FIG. 280.



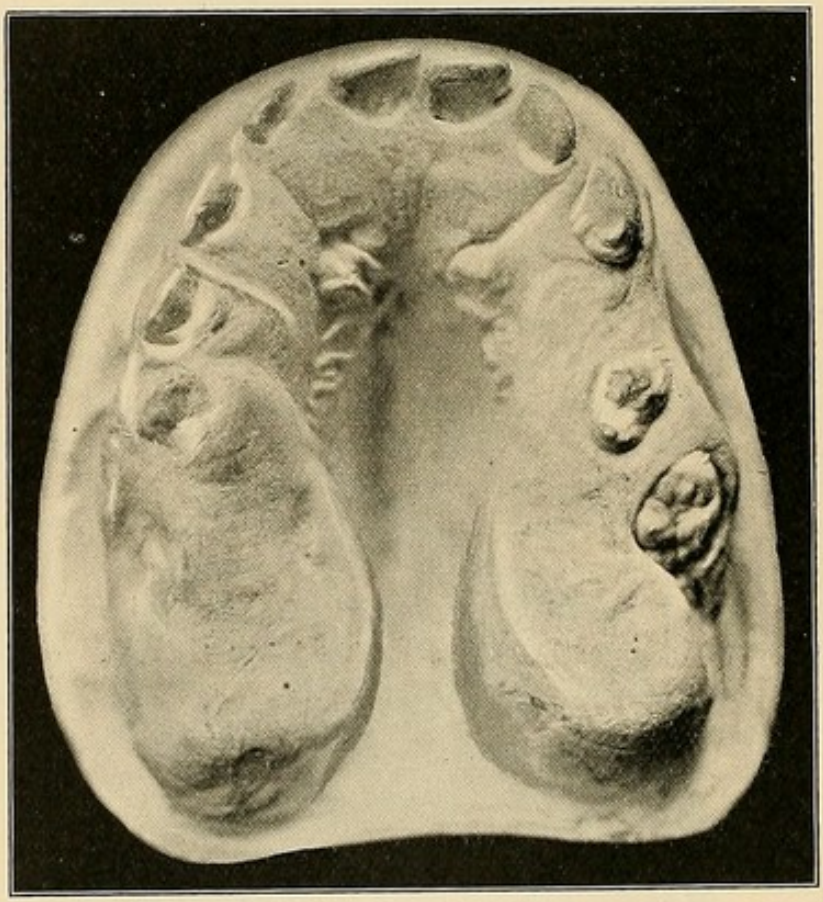
OSTEOMA OF LEFT FRONTAL SINUS, SEEN FROM BELOW. (After Sutton.)

of the tumor. (Warren.) They are sparingly supplied with blood-vessels. The surface of the growth is nodular and covered with periosteum. In the early clinical history of their growth they are frequently separated from the bone from which they spring by a thin layer of

fibrous tissue; later they become solidly united to the bone. These tumors are usually located upon the surface of bone, and are in all probability developed from the periosteal osteoblasts, or they may have their origin in chondromata (Senn) which later have become ossified.

Periosteal or compact osteoma is the most common variety associated with the facial bones. The simplest and most common form is the small, irregular osseous tubercles so frequently seen upon the lingual alveolar plate of the inferior maxilla and upon the external alveolar plate of both maxillæ. More rarely the alveolar processes of

FIG. 281.



HYPERTROPHY OF THE JAWS AND ALVEOLAR PROCESSES—UPPER JAW.—NATURAL SIZE.

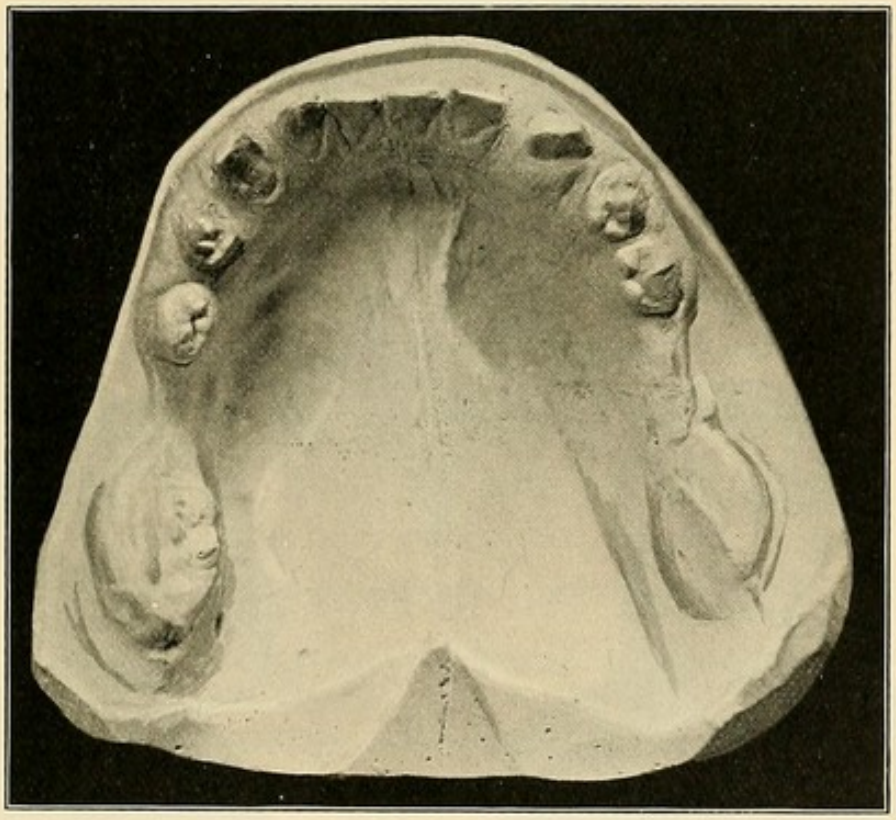
both jaws will present extensive, somewhat evenly distributed, diffuse enlargement,—*hyperostosis*,—having the character of *osteoma durum*, or ivory exostosis. Figs. 281, 282 illustrate this form of growth.

These illustrations are made from casts in the possession of the writer, and are exact copies of impressions taken by him of the jaws of a German girl, twenty-four years old, kindly referred to him by Dr. Fütterer, of Chicago.

Diffuse hyperostosis of the maxillary bones and upper bones of

the face and cranium occasionally occurs. (Fig. 283.) This disease has been designated by Virchow *leontiasis ossea*. It is characterized by a hyperostosis that is usually bilateral and symmetrical. It begins ordinarily in the superior maxillary bones, most frequently in the antra; enlargement takes place in the form of prominences upon the inner walls of the antra, under the skin of the face, and projections into the nasal passages. It exhibits a marked tendency to involve the bones of the face and the cranium. The disease is one of youth; its course is exceedingly slow, but progressive, and tends to destroy life by virtue of its progressive development. It may begin in the lower jaw and hyoid bone, and extend upward, as in a case recently reported by Brown. (Figs. 284, 285.) The cause of the disease is unknown.

FIG. 282.



HYPERTROPHY OF THE JAWS AND ALVEOLAR PROCESSES—LOWER JAW.—NATURAL SIZE.

Osteoma of the nasal process of the superior maxillary bones is quite common. Hutchinson describes such a case in which the outgrowths were bilateral and symmetrical. (Fig. 286.)

Hilton, in Guy's Hospital Reports, describes a case of similar location of the tumor, but of greater interest, from the fact that after a period of years it spontaneously separated from its attachments and was exfoliated. "The patient was a man aged thirty-six years, who had noticed twenty-three years before a pimple below the left eye, close

to the nose, which he irritated, and from that spot the tumor appears to have originated. The tumor in its growth displaced the eyeball, giving rise to excruciating pain, which subsided on the bursting of the ball. The tumor began to loosen by a process of ulceration around its margin six years before it fell out, which event was unattended by either bleeding or pain. The tumor weighed fourteen and

FIG. 283.



HYPEROSTOSIS—LEONTIASIS OSSEA OF THE SUPERIOR BONES OF THE FACE. (After Howship.)

FIG. 284.



LEONTIASIS OSSEA. (After Brown.)

FIG. 285.



LEONTIASIS OSSEA. (After Brown.)

three-quarter ounces. It was tuberculated externally, and an irregular cavity existed at the posterior part. A section of the tumor presented a very hard, polished surface, resembling ivory, and exhibited lines of concentric curves, enlarging as they were traced from the posterior part. The huge cavity left by the tumor was bounded below by the floor of the nose and antrum, above by the septum nasi, and externally

by the orbit, which had been considerably encroached upon by the tumor."

In this case it seems quite evident that the tumor had never become solidly united to the bone from which it sprang, but that it was separated from it by a layer of connective tissue, which was probably destroyed by the suppurative process which was going on for so many years about the tumor, and, finally extending to all the surrounding soft tissues, loosened it from its attachments.

Heath reports a similar case of a young man, twenty-one years of age, who was under the care of Sir William Fergusson. In this case the tumor occupied the left side of the face, and caused displacement of the eye. The tumor had been growing for twelve years, but during that

FIG. 286.



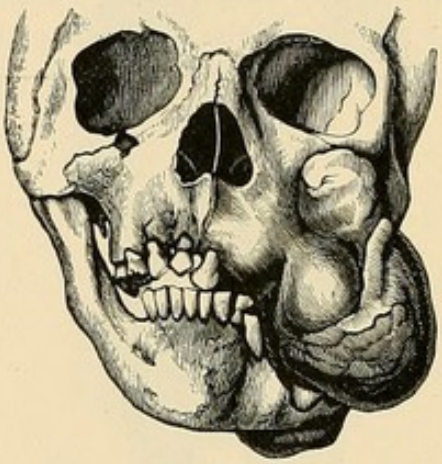
SYMMETRICAL EXOSTOSIS OF THE NASAL PROCESSES OF THE MAXILLÆ. (After Hutchinson.)

time the patient had suffered no headache nor pain in the tumor, and his sight had been unaffected. The tumor, when removed, weighed ten and one-half ounces, and consisted, in its anterior part, of nodulated bone as hard as ivory, and posteriorly of very dense ordinary bone, mixed with a small amount of cartilage. The tumor invaded the antrum, orbit, and nasal fossa. An interesting point in this case is the fact that it seemingly illustrates the transitional steps in the conversion of a cartilaginous tumor into an osseous tumor.

Cancellous Osteomata.—These tumors histologically resemble the cancellated structure of bone, and are consequently much softer than the "compact osteomata." Examples of this variety of osteoma located in the maxillary bones are very rare. These growths are of spongy texture, lobulated in form, and appear to be formed by the ossification of chondromata.

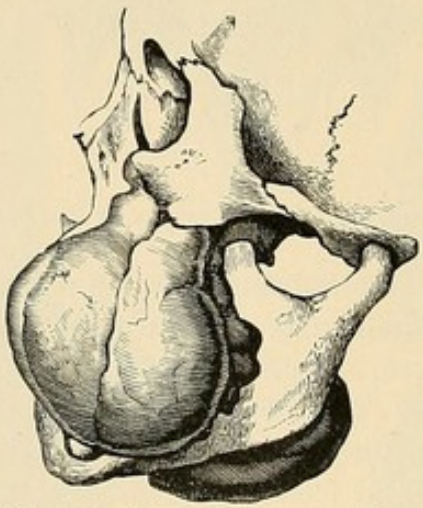
Vidal describes a very remarkable case of this kind, the specimen of which is preserved in the Musée Dupuytren, Paris, shown in Figs. 287 and 288. "The tumor is connected with the left superior maxilla, being limited internally by the intermaxillary suture, behind by the pterygoid process, above and externally by the malar bone. The tumor encroaches considerably upon the cavity of the mouth, and reaches back as far as the front of the spine. Its form is bilobed, and

FIG. 287.



OSSEOUS TUMOR OF THE LEFT SUPERIOR MAXILLA. (Dupuytren.) (After Vidal de Cassis.)

FIG. 288.



OSSEOUS TUMOR OF THE LEFT SUPERIOR MAXILLA—LATERAL VIEW. (Dupuytren.) (After Vidal de Cassis.)

in the deep sulcus between the lobes can be seen a molar tooth. All of the other teeth have disappeared, and there is no trace of their alveoli. The left orbit and nasal fossa are not sensibly diminished in size, but the cavity of the mouth is almost entirely occupied by the posterior lobe of the tumor. The lower jaw has in this case undergone several remarkable alterations. It must at first have pressed

upon the growth and produced the deep sulcus between the lobes, but in its turn the tumor has reacted upon the lower jaw, with the following effect: It has caused a double luxation of the jaw, the left condyle resting against the base of the zygoma and the glenoid cavity being filled with soft material. The teeth of the left side of the lower jaw have disappeared, and absorption of part of the coronoid process and the whole of the alveolus has taken place, so that only the base of this part of the bone is left. The outer surface of the tumor is smooth, and presents numerous vascular grooves of good size; at many points it is perforated with holes. The vascularity of the other bones of the face does not appear augmented."

Heath records three other specimens of this variety of osteoma, neither of which, however, was so large as the one just mentioned.

The student is referred to this author's "Injuries and Diseases of the Jaws," third edition, for a detailed account of these cases.

Osteoma of the lower maxilla is an affection of rather rare occurrence, if a judgment may be based upon the limited number of cases recorded in surgical literature. The disease appears in the same forms as in the superior maxilla, viz, the *compact* and the *cancellous* varieties, the compact or ivory exostoses being the most common.

Tumors of this form have a predilection for the angle of the jaw. Out of five cases of this variety situated in the lower jaw, four were located at the angle and one upon the lingual plate of the alveolar process on either side of the symphysis, in positions corresponding to the bicuspid and first molar teeth. (Heath.) They vary in size from two-thirds or three-fourths of an inch in diameter to three or four inches. Their form is lobulated, and they have smooth surfaces. The growth seems to form around the angle, so that this portion of the jaw rests in a deep groove in the surface of the tumor.

Cancellous osteomata appear in the same locations of the maxillary bones as the compact osteomata, and they may convert the entire thickness of the jaw into a lobulated mass of spongy bone-tissue, as described in one case by Heath.

It may also form a distinct tumor within this cancellated tissue of the body of the jaw,—*endostosis*. Sir James Paget describes such a case in which the tumor was located in the interior of the angle of the jaw, and composed of hard, finely cancellous bone.

The presence of osteoma in the parotid gland and other locations where bone-tissue does not normally belong is explained in two ways: either by metaplasia of the connective tissue, or by the presence of a matrix of misplaced embryonic osteoblasts. The latter explanation seems the more rational, and more in accord with physiologic laws.

Osteoma in the parotid gland is much less frequent than chondroma.

Sarcomatous transformation occasionally takes place in the osteomata. Fig. 289 shows a commencing sarcomatous degeneration taking place in a tumor of this character.

Treatment.—Unless the tumor interferes with speech or mastication, or presses upon nerves, or causes an unsightly deformity, surgical interference is not indicated, for these growths are not painful, and do not cause inconvenience except in the ways indicated.

FIG. 289.



OSTEOMA WITH COMMENCING SARCOMA. × 60.

Accessible tumors located upon the surface of bones may be removed with the surgical engine and bone burs, a fine saw, chisel and mallet, or with strong cutting forceps. Osseous tumors which involve the antrum and accessory sinuses may sometimes require exsection of the jaw for their removal.

If the tumor is imbedded between the plates of the bone, it should be enucleated, when located within the jaw, if possible, without external incision.

REVIEW.

CHAPTER LV.

Give the definition of osteoma.
What is Senn's definition?
What tumors do the osteomata include?
What is hyperostosis?
What is an osteophyte?
What is an exostosis?
What is an endostosis?
What is the condition known as mobile periosteal exostosis?
What is the condition known as parosteal osteomata?
What are independent osteomata?
What are heteroplastic osteomata?
Into how many classes does Sutton divide osteomata?
Describe compact osteomata.
Where may this variety of tumor occur?
Where is it most frequently found?
What other terms are applied to this class of osteomata?
What is their histologic structure?
What is the clinical history of these growths?
Where are these tumors usually located?
What form of osteoma is most commonly associated with the facial bones?
What is the simplest form of compact osteoma?
Does this form of the disease ever become diffusive in the alveolar processes?
What term is then applied to it?
Does it ever extend beyond the maxillary bones?
What term is applied to it when the upper bones of the face and the cranium are involved?
How is this form of the disease characterized?
At what period of life does it occur?
What is the character of its course?
Does it ever begin in the lower jaw?
In what other processes of the maxillary bone does it commonly occur?
Are these tumors ever spontaneously exfoliated?
What may sometimes be the extent of these growths?
What form of bone-tissue do cancellous osteomata resemble?
Are they common or rare in the maxillary bones?
What is the texture and form of these growths?
Do they ever attain a large size?
Are osteomata found in the lower maxilla?
In what forms do they appear?
Which variety is the most common?
For what portion of the jaw do these tumors seem to have a predilection?
What is the variation in the size?
What is the character of their form and surfaces?
In what locations do the cancellous osteomata occur?
Do they ever involve the entire thickness of the bone?

What are they termed when located in the cancellous structure of the bone?

How is the presence of osteoma in the parotid gland and other locations where bone-tissue does not normally belong explained?

Are osteomata or chondromata most frequent in the parotid gland?

Under what circumstances is surgical interference justifiable in osteomata?

How may accessible tumors upon the jaws be removed?

What operation may be necessary when the disease involves the antrum and accessible sinuses?

If the tumor is located between the plates of the bone, how should it be removed?

CHAPTER LVI.

ANGIOMATA.

Definition.—Angioma (Gr. *αγγειον*, a vessel; *ομα*, a tumor). A tumor formed of blood-vessels.

An angioma is a tumor composed of an abnormal development of blood-vessels. They may be congenital, may develop during the first few weeks after birth, or not until late in life.

Angiomata appear in three forms, viz: *Simple nevi, cavernous nevi,* and *plexiform angiomata.*

Origin.—According to Senn, these tumors are derived from a matrix of angioblasts.

Angioblasts are modified fibroblasts, and their function is to form new blood-vessels. In the growth of normal blood-vessels the angioblasts furnish the essential tissue-elements of blood-vessels; after the blood-vessels reach their requisite normal size the process becomes stationary. The cells from which an angioma is developed observe no such limitations of function, but continue cell-proliferation, with the result of producing atypical blood-vessels, which are not required by the tissues in which they are developed, and which constitutes the tumor-tissue.

Angiomata are generally described as tumors mainly composed of blood-vessels, the principal portion of which are new-formed; others may be composed of pre-existing blood-vessels more or less changed. These changes consist chiefly in dilatation, or thickening of the walls.

The majority of angiomata are congenital, and they possess a tendency to spread or increase in size. Occasionally, however, the opposite condition takes place, and they gradually disappear. Angiomata frequently appear as complications in other forms of tumors, and impart to them most serious clinical features. They are often combined with lipoma, fibroma, adenoma, carcinoma, and sarcoma. Sarcomatous transformation of an angioma is not a rare feature in the clinical history of these growths; hyaline and colloid change also occasionally takes place.

A simple nevus (capillary angioma), or "birth-mark," is the most common form of angioma. It is an incipient form of vascular tumor, and in its typical form it is most frequently found upon the skin of the

face and the orbit. More rarely it appears upon the lip, tongue, buccal mucous membrane, and conjunctiva, varying in color from a bright pink to a deep blue. The color, according to Billroth, depends upon the depth at which they are located in the tissues. The most superficial ones, known as "port-wine stain," are red, while those situated deeper are blue. Sutton thinks the nevus is "bright pink" when the tumor is composed of arterioles, and of a "bluish tint" when composed mainly of venules. Such nevi may be no larger than a pin-head, or they may cover a large portion of the face. They consist of an abnormal development of arterioles or venules within the skin or the subcutaneous tissue.

Histologically they are composed of minute but abnormally developed blood-vessels imbedded in adipose tissue, the vessels separated by a greater or less quantity of connective tissue; communicating with the nevus there are usually two or more larger vessels, which are derived from an adjacent artery or vein. The arterioles or venules are often abnormally enlarged or sacculated. (Fig. 290.)

Diagnosis.—The tumor may usually be emptied by compression upon it, when the color of the overlying skin will become normal, but sometimes this cannot be accomplished.

As soon as the compression is removed, the blood immediately returns to the tumor and its color is restored.

In the simplest form of nevus there is no elevation of the growth above the surrounding surface; it merely looks like a patch of skin of a different color which has been substituted for the normal tissue. In the more complicated forms of this variety, the surface of the growth is slightly elevated above the surrounding skin.

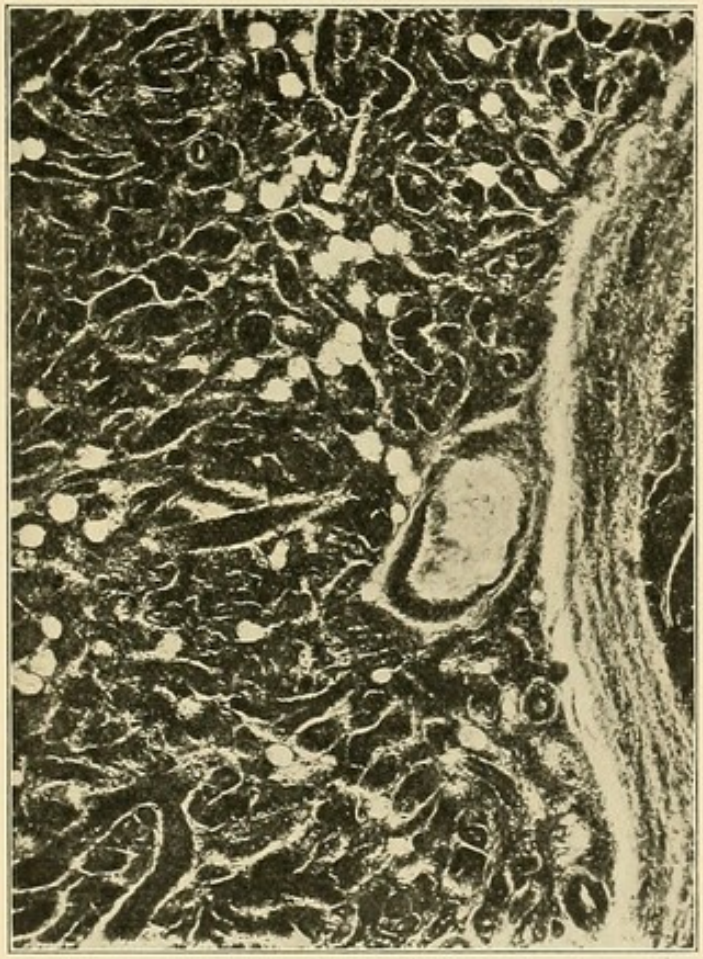
Simple angiomata are quite frequently seen upon the vermilion border of the lower lip, upon its inner aspect, and upon the mucous surface of the cheeks at the angle of the mouth. They rarely in these locations assume a size larger than a split pea, though they may attain very large dimensions. Warren mentions a case in a child which involved the entire lower lip and extended to the neck. Simple angiomata are also found with considerable frequency in the tongue. They may be simple or multiple. The venous variety is usually congenital; the arterial form generally develops later in life. They rarely reach a size larger than a nut, are quite painless, and usually give no inconvenience except from their size. Accident which ruptures the surface is liable to be followed by profuse and repeated hemorrhage. (Butlin.)

Capillary angiomata sometimes develop into the cavernous variety, and become formidable tumors; on the other hand, they occasionally disappear spontaneously.

A cavernous nevus (cavernous angioma) may be distinguished from

the simple form of nevus by the difference in the form of the blood-vessels. The tubular form is characteristic of simple nevus, while in the cavernous variety the blood-vessels are developed into a series of variously-shaped sacs or cavities, and separated from one another only by fibrous septi, lined with endothelium. This form of angioma is rarely congenital, and usually appears rather late in life. It is of slow growth, but often attains to a large size. Many of them are exceed-

FIG. 290.



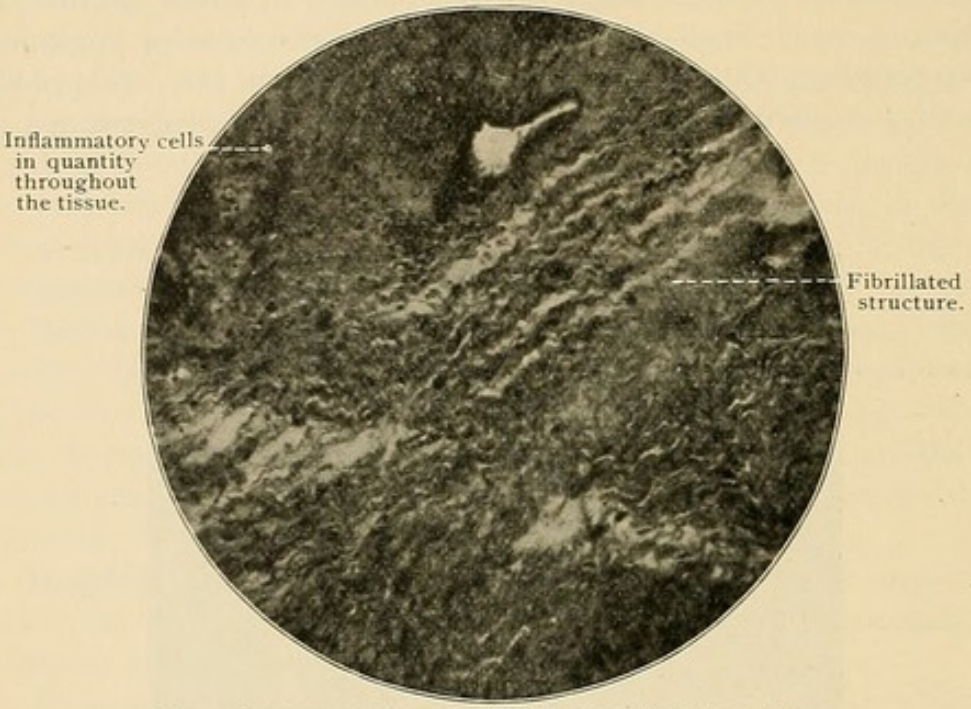
NEVOID GROWTH OF CHEEK. ×40.

ingly sensitive, and on account of their liability to hemorrhage are often dangerous. Angioma is occasionally found in the antrum, usually in the form of a fibro-angioma.

The writer has seen one case of this variety of tumor. It was located in the antrum of Highmore, and caused pressure-atrophy of the walls of the cavity and bulging of the roof of the mouth. The case was diagnosed as probably sarcoma of the antrum. The patient was a young Greek, about twenty years of age, in other respects in good health. Upon opening the antrum a gush of blood followed, which

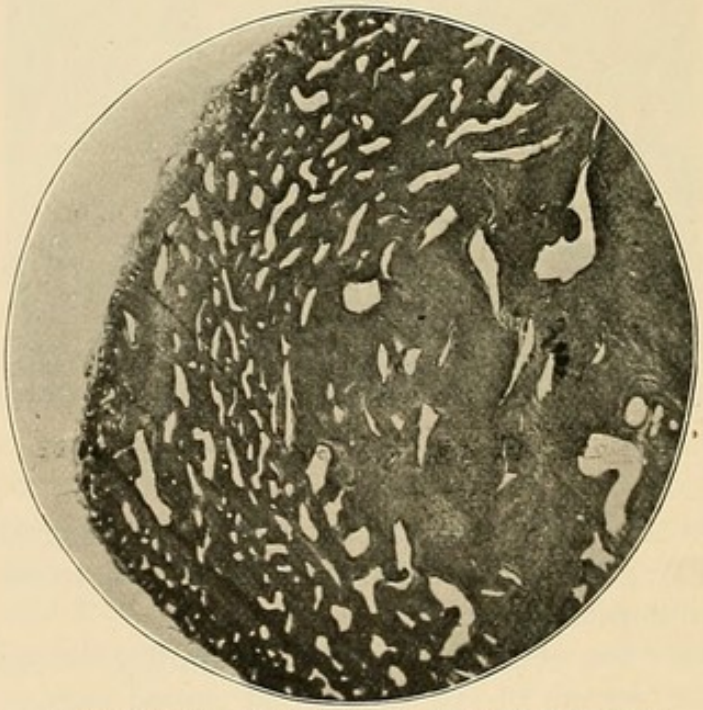
was very alarming, threatening to choke the patient before the hemorrhage could be checked. By rapid work the antrum was cleared of

FIG. 291.



FIBRO-ANGIOMA OF ANTRUM (A)—FIBROUS PORTION. × 50.

FIG. 292.



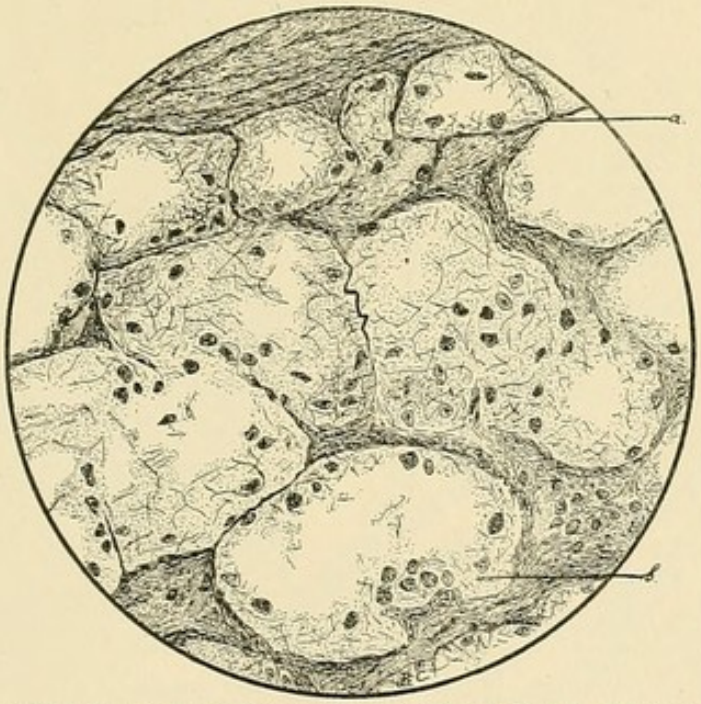
FIBRO-ANGIOMA OF ANTRUM (B)—CAVERNOUS PORTION.

the growth by spoon curettes, and then tightly packed with gauze. The patient made a good recovery. Figs. 291, 292 show the character of the growth.

Cavernous angiomata are rarely seen in the tongue. Bryant records a case in which the tumor occupied the lip and right side of the anterior half of the tongue. The parts were congested, swollen, and covered with large veins, and great tortuous arteries could be felt running up from the base of the tongue to supply the tumor. Compression emptied the tumor, which again instantly refilled on removing the pressure. Fig. 293 illustrates the formation of an angioma as seen in the tongue. Fig. 294 shows the structure of a tumor of this character in the lip.

Cystic and warty degeneration sometimes takes place in these growths. Glandular infection does not take place, but occasionally

FIG. 293.



ANGIOMA OF THE TONGUE, SHOWING NEWLY-FORMED BLOOD-SPACES NOT YET IN CONNECTION WITH PRE-EXISTING VESSELS. × 330. (After Senn.)

a, angioblast; *b*, newly-formed spaces filled with delicate fibrous net-work and amorphous material.

the lymphatic vessels become dilated in the floor of the mouth and the neck. (Butlin.) These enlargements are painless, and if punctured discharge a thin, watery, albuminous fluid.

A plexiform angioma (cirsoid aneurism, or aneurism by anastomosis) consists of a number of abnormally developed, tortuous blood-vessels, moderate in size, and arranged in a generally parallel direction to one another, making a tumor sometimes of considerable size. These tumors consist in some instances entirely of arteries; in others, of veins only, or of arteries and veins in about equal proportions. Angiomata of this variety are exceedingly rare in any portion of the body, but are found most frequently about the face and scalp. Bruns de-

scribes a remarkable case involving the face, forehead, and temporal region. (See Fig. 295.) The writer has recorded a case of this form of angioma (anastomosing aneurism) in a young man about twenty-six years old, in whom the angioma developed as the result of injury in the extraction of the upper molar teeth. It involved the hard palate and the antrum of Highmore, and seemed to be connected with the posterior palatine artery of the right side. The growth gave distinct

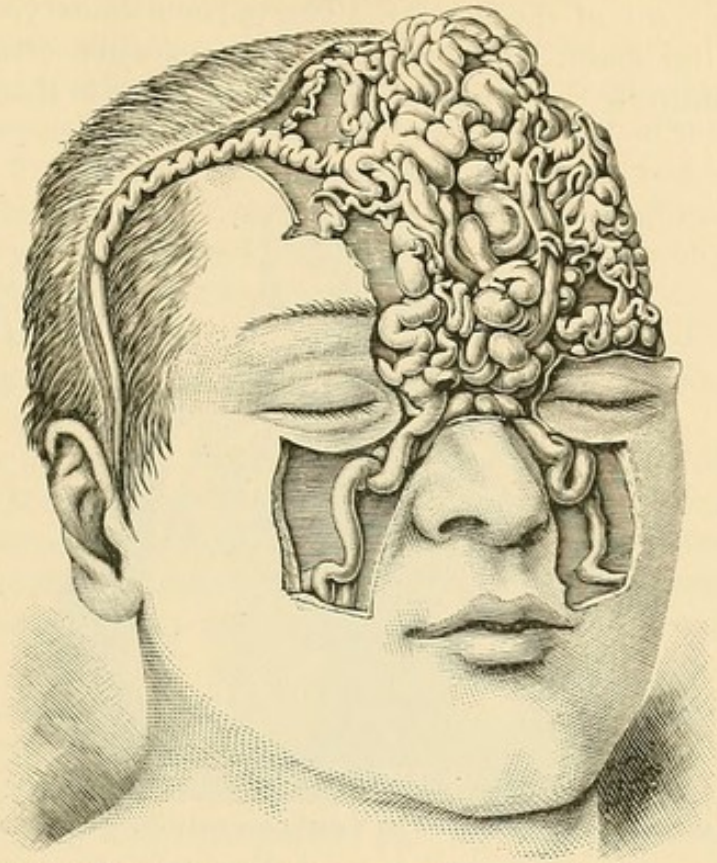
FIG. 294.



CAVERNOUS ANGIOMA OF LIP. × 50.

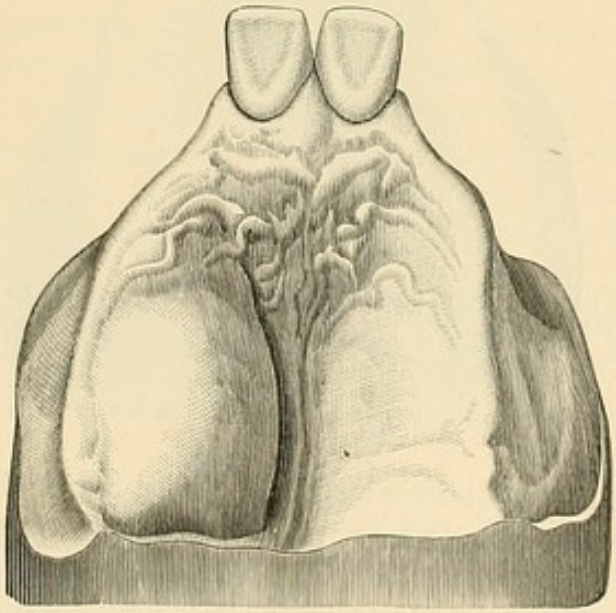
pulsations, which could be felt, and which were perceptible to the patient. The tumor was about an inch and one-half in length by about one inch in width. This case was treated by injection of perchlorid of iron, with complete cure of the disease, which was demonstrated by its non-recurrence two years after the operation. Fig. 296 represents the tumor one month before the operation. Fig. 297 shows the increase in growth from that time to the date of operation, and Fig. 298 the result of the operation.

FIG. 295.



DISSECTION OF A PLEXIFORM ANGIOMA OF THE FOREHEAD. (Müller, after Sutton.)

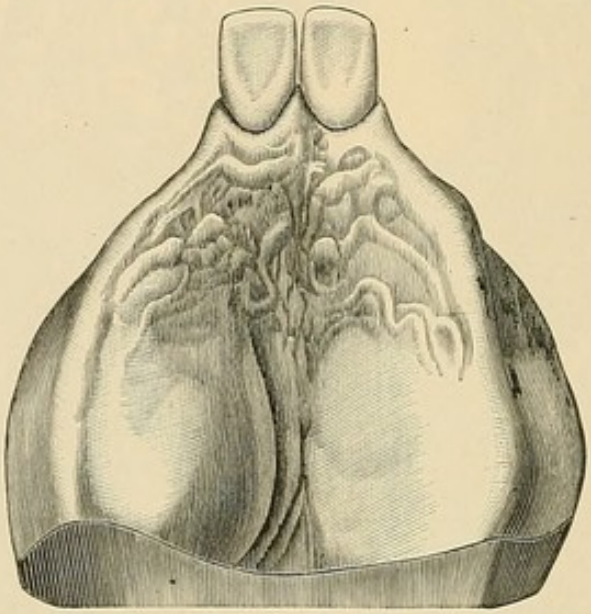
FIG. 296.



CIRSOID ANEURISM OF THE ANTRUM OF HIGHMORE.

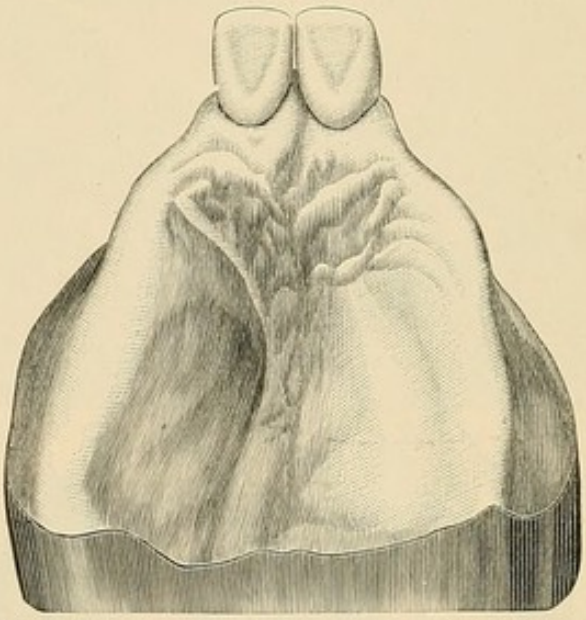
Treatment.—The treatment of angioma will vary with the character and extent of the disease. The galvano-cautery, coagulating injections, the ligature, ignipuncture, the application of caustics, and excision constitute the principal surgical resources in the treatment.

FIG. 297.



CIRSOID ANEURISM OF THE ANTRUM OF HIGHMORE, SHOWING INCREASE IN GROWTH.

FIG. 298.



CIRSOID ANEURISM OF THE ANTRUM OF HIGHMORE, SHOWING RESULT OF OPERATION.

Simple angioma, "port-wine stain," if not larger than a silver half-dollar piece, may be successfully treated by the electro-thermal cautery, but when of greater extent than this, treatment for its removal is inadmissible. Treatment by electrolysis should be conducted with great

care. Only a small part of the discolored surface should be treated at each sitting, and repeated at intervals of three or four days.

Nevi appearing in young children should be carefully watched, and if they give evidence of growth they should receive prompt treatment. If of small size, no treatment is so successful as excision.

Sutton prefers the knife to electrolysis, nitric acid, ethylate of sodium, or the ligature. Growing nevi, if left to themselves, may soon pass the limits of justifiable surgery. It is therefore of the utmost importance that operation be undertaken early.

Butlin favors the galvano-cautery for the removal of all such growths located in the tongue, on account of the hemorrhage which is so likely to attend an operation with the knife or scissors.

There is less hemorrhage attending the use of the galvano-cautery heated to a dull red than when a white heat is used. No definite line of procedure can be laid down for the treatment of cavernous angiomata or cirsoid aneurisms. The surgeon must be governed by the conditions surrounding the case, and the size and location of the growth.

Fayrer successfully treated a case of cirsoid aneurism of the tongue by injections of a strong solution of tannic acid. The writer succeeded in his case of cavernous angioma, which was located in the antrum, by breaking it up with curettes and packing with gauze, while in the cirsoid aneurism located in the hard palate and involving the antrum success attended the injection of a ten per cent. solution of the tincture perchlorid of iron.

The dangers which attend the treatment of such growths by the injection of coagulating remedies are those of embolism, from the floating away of small clots in the blood-current, which may become lodged in the vessels of remoter regions. Ulceration and suppuration have been frequent sequelæ of the injection treatment, and instances are on record in which they produced instant death.

The writer is of the opinion that if the injection method is used the remedy employed should be of such a character and strength as to produce instantaneous coagulation of the whole mass of blood contained in the growth, which would reduce the dangers from embolism to the minimum.

In those cases requiring the removal of any considerable amount of skin, preventing coaptation of the wound, the surface should be covered by a Wolfe graft or by Thiersch grafts.

REVIEW.

CHAPTER LVI.

Give the definition of angioma.

In how many forms do angiomata appear?

Name these forms.

What is the origin of these tumors?

What are angioblasts?

What is their function?

Do the cells from which the angioma is developed observe the same limitations of function?

What is the result of their cell-proliferation?

How are angiomata generally described?

What is the character of the change in pre-existing blood-vessels?

Are the majority of angiomata congenital or post-natal in their origin?

What tendency do they possess?

Does angioma complicate other forms of tumor?

What retrogressive changes are liable to take place in these tumors?

What other term is applied to simple nevi?

Describe this form of angioma.

Where is it most commonly located?

What is its color?

What governs this difference in color?

What is the color of the most superficial variety?

How does Sutton explain the differences in color?

What may be the variations in size?

What is the histologic structure of angioma?

How may compression become a diagnostic sign in angioma?

What is the appearance of the simplest form of nevus?

What is the appearance of the more complicated forms?

In what locations besides the skin may simple angioma most frequently be seen?

What is their usual size in these locations?

Are they ever found upon the tongue?

When do the venous and arterial forms develop?

What size may they attain?

What follows rupture of their surface?

Into what more serious forms do the simple varieties sometimes develop?

Does a spontaneous cure ever take place?

How may the cavernous nevus be distinguished from a simple nevus?

What characteristics of the blood-vessels distinguish between these two forms of angioma?

At what period of life does the cavernous angioma appear?

What is the character of its growth?

Is it sensitive?

What particular liability makes them dangerous growths?

What sinus do they sometimes occupy?

Are they ever found upon the tongue?

What forms of degeneration sometimes take place in these growths?

What is the character of a plexiform angioma?

What is the character of the blood-vessels that make up these tumors?

Are these tumors common or rare?

Upon what does the treatment of angioma depend?

What are the surgical resources in angioma?

How may small angiomata be treated?

How should the treatment by electrolysis be conducted?

How should nevi in young children be cared for?

What is the best treatment of these cases?

What is the tendency of growing nevi?

What methods of treatment are advised for nevi of the tongue?

What degree of heat is most valuable for removing the nevi by the galvano-cautery?

What line of treatment is followed in cavernous angioma?

How did Fayrer succeed in curing a cirsoid aneurism?

How did the writer succeed in his case?

What are the dangers attending the injection method of treatment?

What should be the character of the remedies used for injection?

How should the wound be treated in those cases where large sections of skin have been sacrificed?

CHAPTER LVII.

SARCOMATA.

Definition.—Sarcoma (Gr. σάρξ, flesh, and ωμα, tumor). A sarcoma is a connective-tissue tumor, composed of connective-tissue cells and a fibrous stroma.

Sarcomata hold the same relationship to the mesoblastic group of tumors that carcinoma occupies in relation to the epithelial group; namely, that of malignancy.

Sarcoma is composed of connective-tissue cells in a more or less embryonic state of development, and these cells so predominate in number and size as to render the intercellular substance a secondary element in the structure of the tumor.

Origin.—Sarcoma may be defined, adopting the modified theory of Cohnheim, as "an atypical proliferation of connective-tissue cells from a matrix of fibroblasts of congenital or post-natal origin." (Senn.) Sarcoma is found invariably to originate in tissues which belong to the mesoblastic group, viz: in formed and unformed fibrous tissue, in cartilage, in bone, and in mucoid, lymphoid, neurogliar, and adipose tissue, and, according to Ziegler, the transformation of these tissues into tumor-tissue is effected by the growth and multiplication of the constituent cells.

Virchow maintained that sarcoma-cells may arise by proliferous multiplication from connective-tissue cells of perfectly normal appearance. Ziegler coincides with this view, but says "it may vary to this extent, that the development begins in tissue which is already morbidly altered. This new-formed cartilage may pass into sarcoma by over-intense proliferous growth of the cartilage cells and disappearance of the matrix substance. *But it is of great interest to note that cells which form part of what we might call congenital heteroplastic foci may often serve as the starting-point of a sarcoma.*"

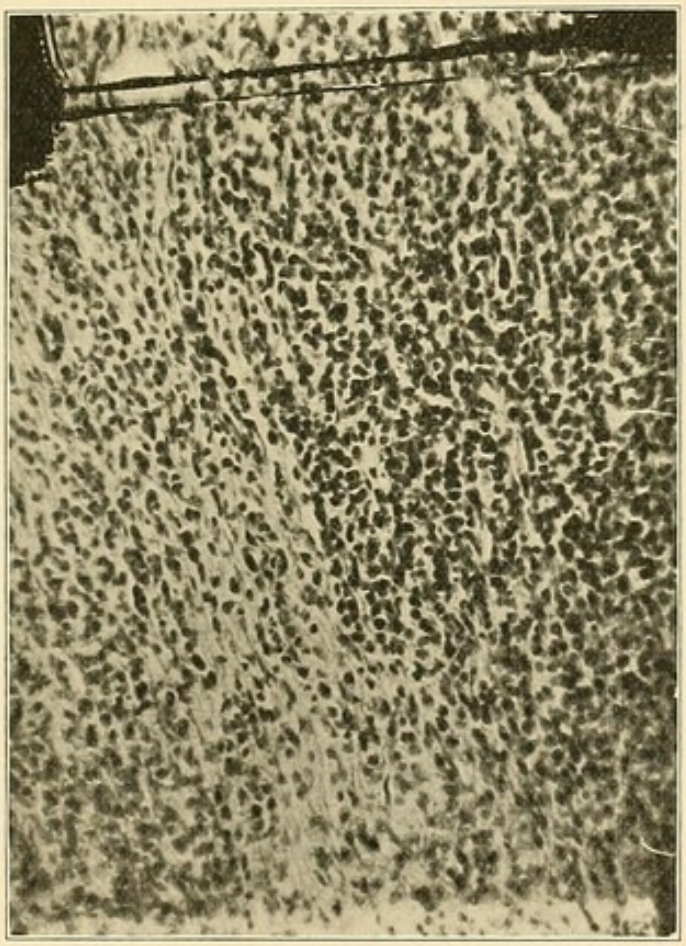
In the growth of sarcoma the tumor-cells never reach a mature type, but are always more or less embryonic in character. This feature, coupled with the marked tendency to infiltrate and involve surrounding tissue, to recur, and to disseminate and form metastatic tumors, indicates the malignant character of the neoplasm.

Varieties and Structure.—The sarcomata are generally classified

according to their histologic structure, and with especial reference to the size, shape, and disposition of the cells and the character and amount of the intercellular substance:

1. Round-celled sarcoma. { Small round-celled. / Large round-celled. / Lympho-sarcoma.
2. Spindle-celled sarcoma.
3. Myeloid sarcoma (giant-celled).
4. Alveolar sarcoma.
5. Melano-sarcoma.

FIG. 299.



SMALL ROUND-CELLED SARCOMA. × 150.

Round-celled Sarcoma.—This form of sarcoma may be divided into three varieties: Small round-celled, large round-celled, and lympho-sarcoma.

Small round-celled sarcoma is of a very elementary character in its construction, consisting of small round cells, blood-vessels, and a very limited quantity of intercellular substance, which is granular or slightly fibrillated. The cells contain a large, round, vesicular nucleus and a very limited amount of protoplasm. (Fig. 299.) This is particularly

noticed in young sarcomata, in which the existence of the large nucleus almost obscures the protoplasm. In stained sections the nuclei are always conspicuous objects. (Sutton.) The vascular supply is usually abundant; the blood-vessels are large, and have thin walls; they penetrate between the cells, and often give rise to a distinct pulsation in the tumor. The degree of vascularity of the sarcomata will indicate the rapidity of their growth. The spindle-celled sarcomata are not so vascular as the round-celled variety, and consequently do not grow so rapidly.

There are no lymphatics in any of the sarcomata, therefore dissemination takes place through the venous circulation. It is characteristic of the small round-celled variety of sarcoma that it grows rapidly, is soft, always infiltrates the surrounding tissues, recurs speedily after extirpation, and sooner or later causes metastatic deposits. It occurs chiefly in the connective tissues of muscles and bones, and also in the skin, testes, ovary, and lymphatic glands. (Ziegler.) These tumors have usually a milky-white appearance on section, and occasionally contain soft, cheesy patches. On scraping the cut surface, a milky juice is obtained.

Large round-celled sarcoma differs from the variety just described in that the cells are considerably larger, often uniformly so, and have an abundant protoplasm and large, oval, vesicular nucleus. Many of the cells are binuclear, and a few multinuclear. (Ziegler.) These cells are so large that they often look like epithelium. The intercellular substance is somewhat more abundant than in the small round-celled variety, and is arranged in a sort of net-work, interspersed with fusiform and ramiform cells, which together form a kind of alveolar reticulum, in the spaces of which are imbedded the large round epithelium-like cells. The vascular supply is not so abundant as in the preceding variety, and the vessel-walls are usually thin. This form of sarcoma occurs in the same locations as the small round-celled variety, but it is generally less malignant, does not grow so rapidly nor infect the surrounding tissues so quickly, but it may eventually form metastatic growths.

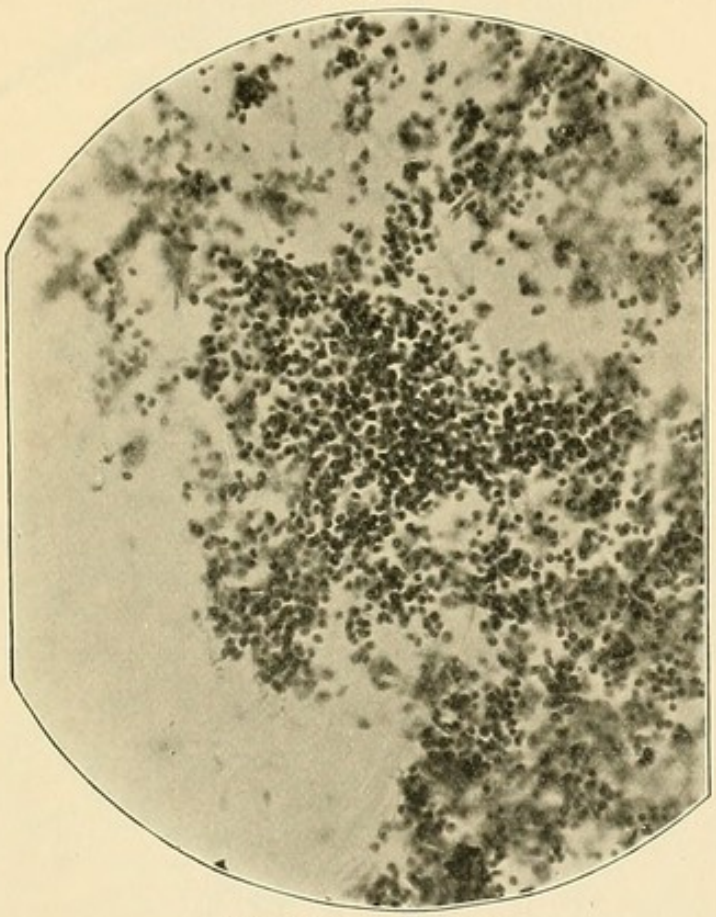
Lympho-sarcoma is a variety of small round-celled sarcoma, but whose structure somewhat resembles the structural arrangement of a lymphatic gland; hence the name lympho-sarcoma. It possesses a delicate reticular stroma, composed in part of intercellular substance and in part of ramiform cells whose processes anastomose. Within the meshes of this reticulum a multitude of small round cells are imbedded. (Fig. 300.) The tumor, in general appearance and clinical history, is not different from the ordinary form of small round-celled sarcoma. Both are exceedingly malignant, both grow rapidly, and both give rise to metastatic deposits which may affect the entire organ-

ism. (Ziegler.) The location of lympho-sarcoma is most commonly in the lymphatic glands and the mucous membrane. It is not, however, confined to these structures, but may occur in other tissues.

Lympho-sarcoma may be differentiated from hyperplastic lymphoma by the greater rapidity of its growth, by the tendency to extend beyond the limits of the gland, and by the formation of metastatic tumors.

Spindle-celled Sarcoma.—This variety of sarcoma is the most common and usually the least malignant of all the tumors of this class.

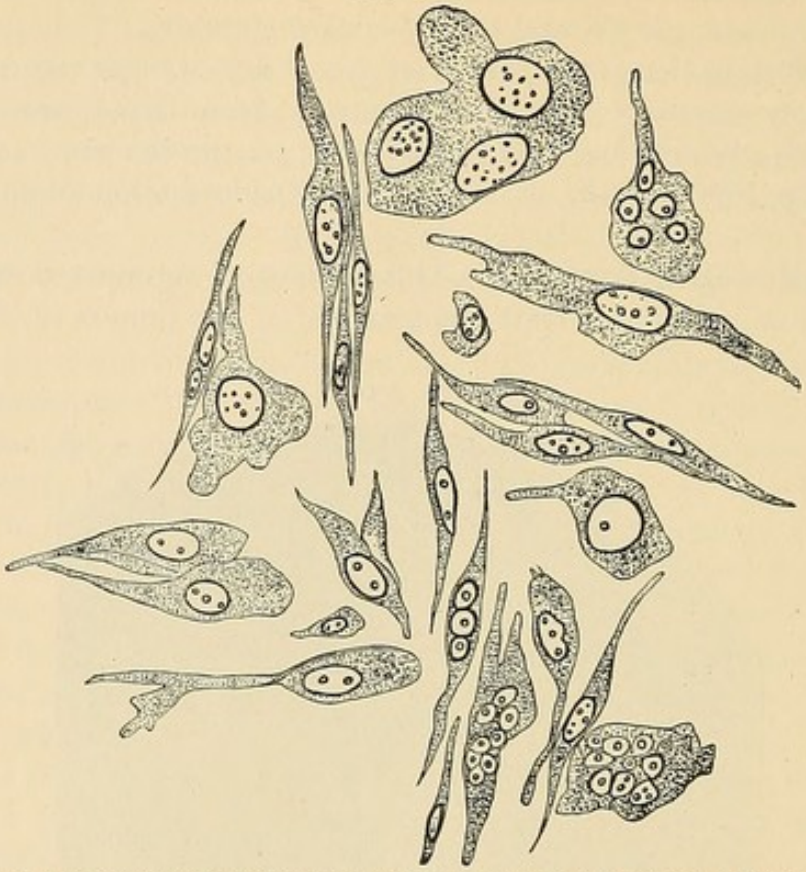
FIG. 300.



LYMPHO-SARCOMA. × 200.

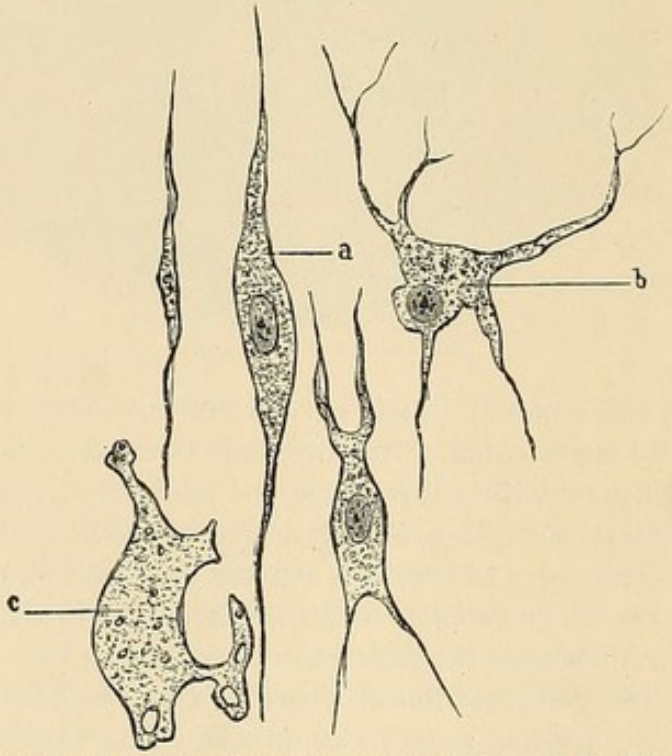
The degree of malignancy, however, will depend upon the abundance or scantiness of the stroma. An abundant stroma indicates a slow-growing hard tumor with a low degree of malignancy, while a scanty stroma will indicate a rapid-growing soft tumor with a high degree of malignancy. This form of tumor is usually divided into two varieties, the *small* and the *large spindle-celled sarcoma.* Senn thinks this subdivision is superfluous, as the difference between the varieties is simply one of size of the cells, as the structure of these tumors is the same. These tumors are located most frequently in dense fibrous tissues like the periosteum, the sheaths of muscles, and the skin. They are com-

FIG. 301.



CELLS FROM A SPINDLE-CELLED SARCOMA, TREATED FRESH IN A SOLUTION OF SODIC CHLORID. × 250. (After Perls.)

FIG. 302.

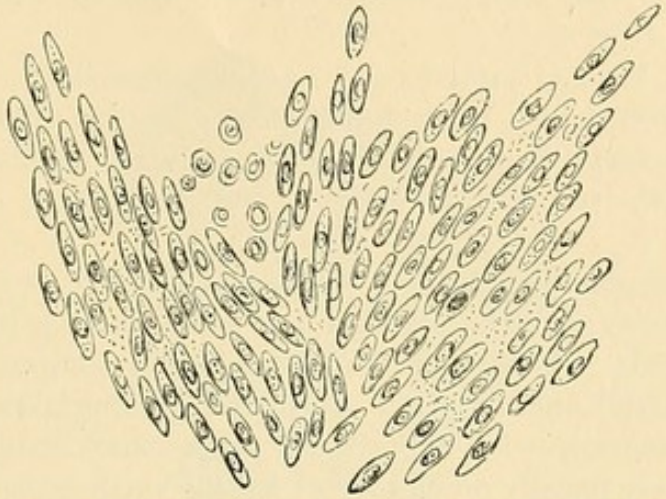


LARGE SPINDLE-CELLED SARCOMA. × 400. (After D. J. Hamilton.)

a, ordinary spindle cell; *b*, branched flat cell; *c*, flat endothelium-like cell.

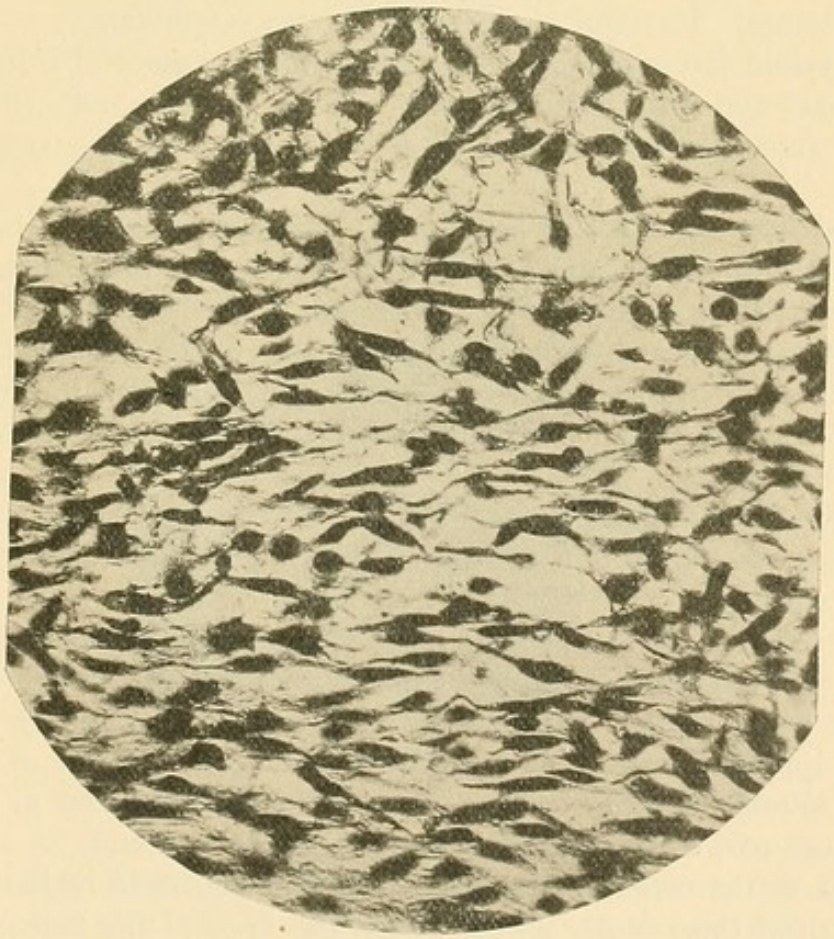
posed of long spindle-cells of varying size, closely packed together, lying side by side, and grouped into bundles, running in various direc-

FIG. 303.



OAT-SEED-LIKE SPINDLE-CELLED SARCOMA. × 300. (After D. J. Hamilton.)

FIG. 304.



LARGE SPINDLE-CELLED SARCOMA (ORBIT.) × 500.

tions. (Fig. 301.) The cells lie with their broad sides toward each other. The difference in the size of the cells is very considerable, the

large spindle-cells being from three to four times as large as the small cells. (Senn.) On isolating the cells by tearing out fragments of the tumor, they will be found to vary very greatly in shape; some will be fusiform, with long prolongations; others ramiform, with several prolongations. (Fig. 302.)

Another form of sarcoma-cell has been described and figured by D. J. Hamilton, which he designates oat-seed-like spindle-celled sarcoma. The chief difference between the ordinary spindle-cell and this form is that the terminations of the spindles are more obtuse. (Fig. 303.)

The intercellular substance of spindle-celled sarcoma is usually small in quantity, and the blood-vessels are located in the axes of the bundles of cells. (Warren.) The reticulum is composed of delicate connective-tissue fibers and the fibrillated prolongations of the fusiform and ramiform cells. (Fig. 304.) Sarcomata made up of large spindle-cells are usually much softer than the small-celled variety.

Myeloid Sarcoma (giant-celled sarcoma).—This variety of sarcoma is composed of tissue resembling the medullary substance of young bones. Its name is derived from this resemblance.

Myeloid sarcoma is characterized by the presence of cells of various shapes and sizes, but principally of large multinuclear cells,—giant cells,—which resemble osteoclasts,—the bone-destroying cells. (Fig. 305.)

This form of sarcoma arises from bone, principally in the medullary canal, but tumors of a similar character are found in the periosteal and other tissues. Kölliker is of the opinion that the giant cells of this form of sarcoma act the part of osteoclasts by destroying the bone. Sections of such tumors show the giant cells imbedded in a matrix of spindle or round cells. Stellate and club-shaped cells may also be present. (Fig. 306.)

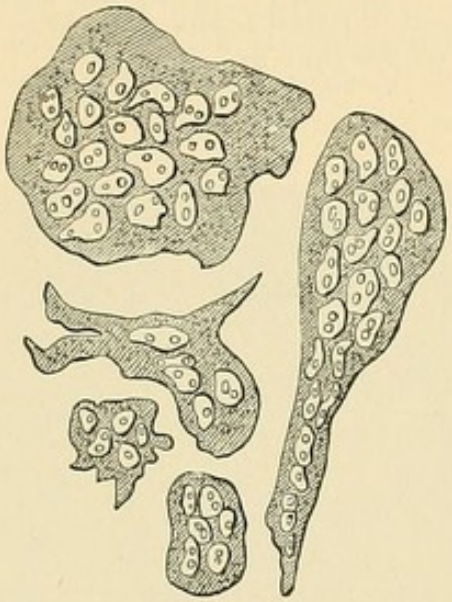
Myeloid sarcoma occurs principally in the long bones, is usually of a deep red or maroon color, and when fresh looks like a piece of liver. (Sutton.)

The periosteal form of myeloid sarcoma is most frequently associated with the alveolar processes of the jaws, usually arising from the sockets of the teeth. Sutton thinks the term *myeloid sarcoma* should not be applied even to tumors occupying the central canal of the long bones, unless the giant cells are present in such quantity as to make up a large part of the tumor. Fig. 307 is from a section of a myeloid sarcoma of the superior maxilla, which had its origin in the alveolus of the right third molar and involved the floor of the antrum to the cuspid tooth. Fig. 308 is a gland of Serres found in the normal gum-tissue covering the tumor.

In the central or myelogenous form of sarcoma the arteries are

so large and so numerous, and their walls so thin, that they frequently impart pulsation to the tumor. (Senn.) In this condition lies the danger of mistaking them for aneurisms of bone.

FIG. 305.



GIANT CELLS FROM SARCOMA. (After Lücke.)

FIG. 306.



GIANT-CELLED SARCOMA FROM UPPER JAW. × 230. (After Perls.)

Alveolar Sarcoma.—Alveolar sarcoma is a peculiar type of tumor in which the cells and the stroma, contrary to the general rule, are arranged after the type of glandular tumors or epithelial neoplasms. (Fig. 309.) This appearance in the structure of these tumors is due in part to the epithelial-like form of the cells, but chiefly to their aggre-

gated arrangement in groups and the separation of the groups by fibrous septa. (Ziegler.)

Such an arrangement of cells and stroma closely simulates that found in carcinoma, and it is sometimes exceedingly difficult to distinguish between them. The cells exactly resemble epithelial cells; they are, like them, grouped in masses, and sharply distinguished from the fibrous reticulum in which they are imbedded. (Ziegler.) The retic-

FIG. 307.



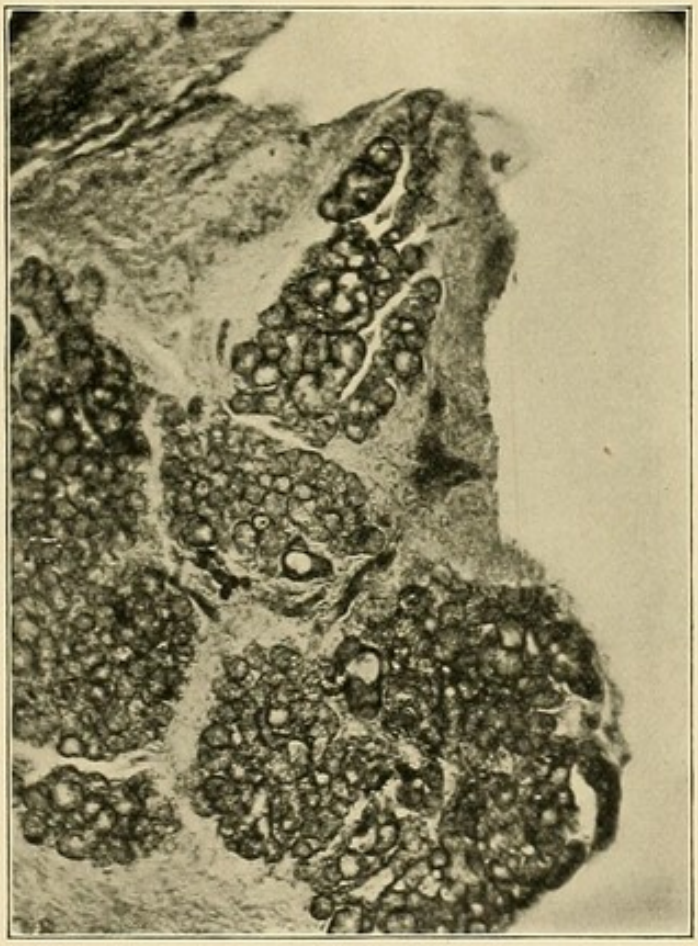
MYELOID SARCOMA OF SUPERIOR MAXILLA—REGION OF THIRD MOLAR AND FLOOR OF ANTRUM. (A.) × 50.

ulum contains the blood-vessels, and no vessels penetrate the cell groups, which is another point in the similarity of tumors of the epithelial group. Sutton says it is possible in carefully prepared sections "to distinguish a delicate reticulum between the individual cells, a condition never found in carcinoma," thus establishing the diagnosis. Sarcoma of this variety is not very common, and is found principally in the skin, muscle, and bone. When located in the skin, it usually originates in connection with warts and the hairy and pigmented moles.

Melano-sarcoma.—This variety of sarcoma may be composed structurally of round or spindle cells; sometimes the stroma presents an alveolar arrangement. (Fig. 310.) The particular feature which distinguishes this tumor from other sarcomatous growths is the presence within the cells, the intercellular substance, and the vessel-walls of a variable amount of black or dark-brown pigment. (Fig. 311.)

Melano-sarcoma is the most malignant of all the forms of sarcoma. Its most common seat is the skin, the eye, and pigmented warts

FIG. 308.



GLAND OF SERRES—FROM THE GUM-TISSUE COVERING THE MYELOID SARCOMA OF SUPERIOR MAXILLA. (B.) × 50.

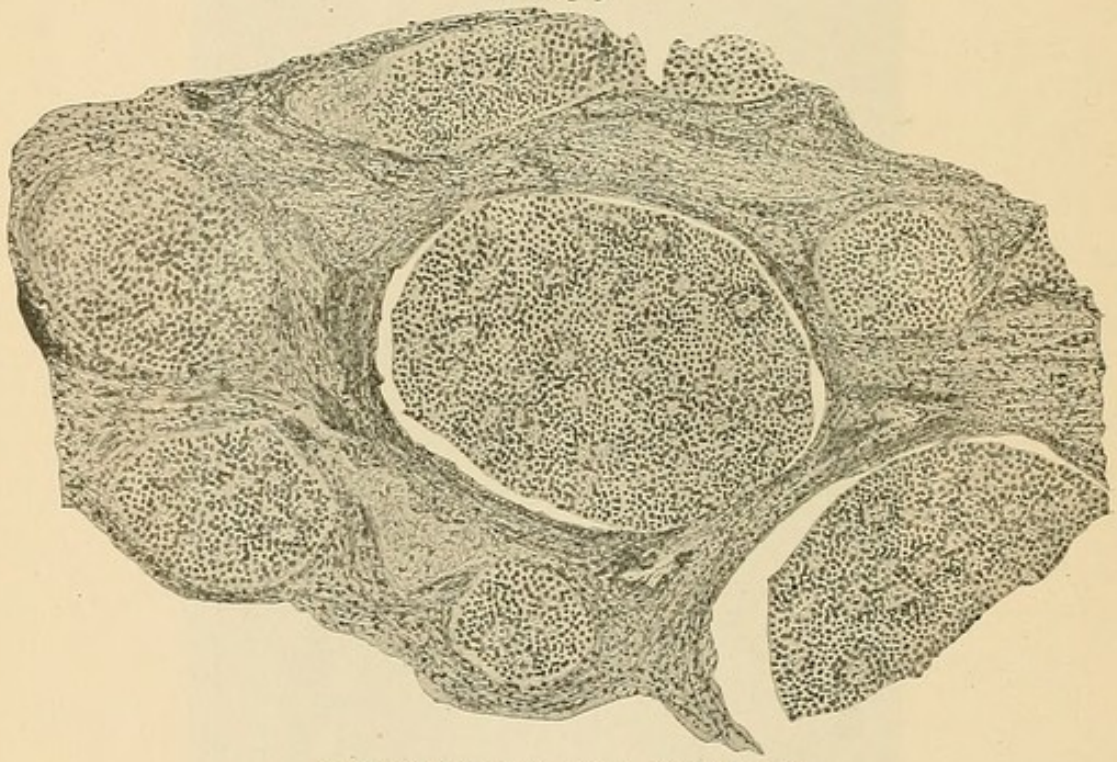
and moles. These growths are characterized by early local and regional infection and metastatic deposits. Primary melanotic sarcoma sometimes occurs in tissues in which pigment material is not present in a normal condition of the tissues. The origin of the pigment material has not yet been satisfactorily explained. Virchow was of the opinion that the pigment was formed within the cells.

It has been customary to explain the presence of the pigment material as derived from the *hematin* of the blood. Neuski found sulfur

in the pigment material, Dressler found iron, and Rindfleisch was quite positive that the *melanin*—the term applied to the coloring material found in melano-sarcoma—was derived from the hematin of the red blood-corpuscles. Senn is of the opinion that the coloring material is derived from the "presence of pigmented cells deposited in the tissues by errors of development,—that is, the existence of a matrix of pigmented cells."

The coloring material, according to Ziegler, occurs chiefly in the form of amorphous granules, but usually there are a number of cells as well, which are diffusely stained.

FIG. 309.



ALVEOLAR SARCOMA. × 100. (After Senn.)

The color of the tumor on section will vary with the amount of pigment material present. When not extreme, it will appear brownish-gray, or show brown or black patches, while in the more marked cases it will be uniformly black.

The pigment-granules are found in the body of the cell, but never in the nucleus. According to Warren, "the pigment is arranged in many cells, so as to distend them and alter their shape, the pigment-granules appearing as large, dark globular masses, the clear nucleus being crowded into one corner of the cell." The younger portions of the tumor have no pigment material at all.

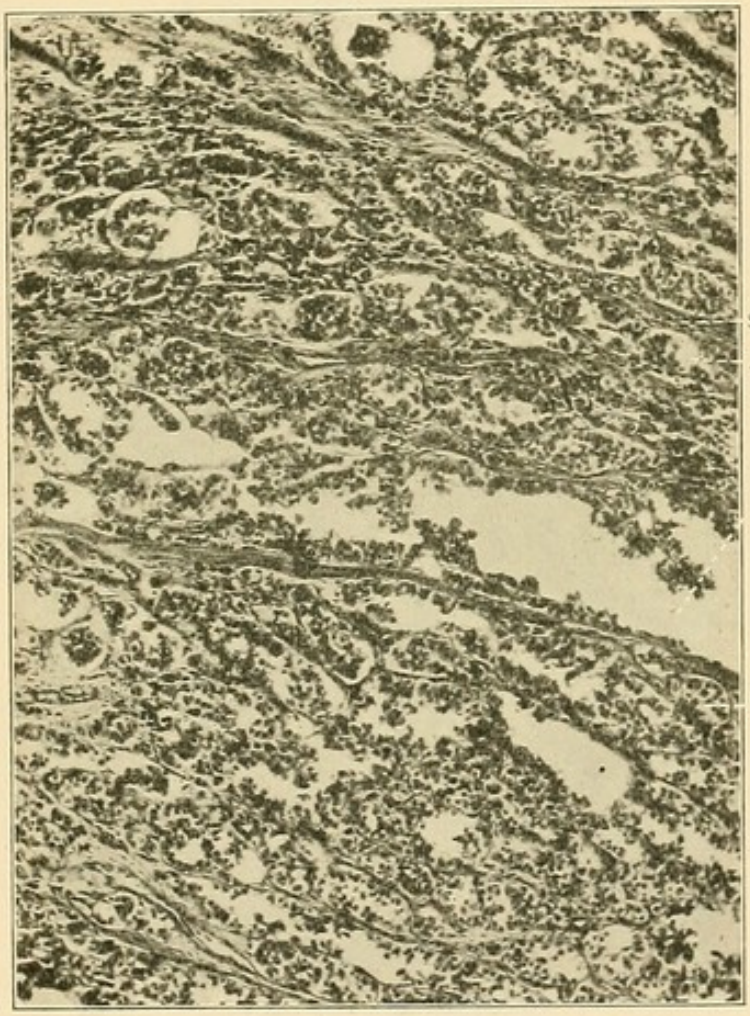
Melano-sarcoma should not be confounded with sarcomata which have absorbed coloring matter from a blood-clot, the result of hemorrhage within their structure. The metastatic tumors of this variety of

sarcoma closely resemble the primary growth, so far as the pigmentation is concerned. (Senn.) (Fig. 312.)

Mixed-cell Sarcoma.—These tumors, as their name indicates, are composed of cells of different forms and structure; there is a mingling of the round, the spindle, and the giant cells in varying proportions.

All tumors, in a certain sense, are mixed tumors, for none of them consist of a single tissue; all possess fibrous tissue, vessels, and char-

FIG. 310.



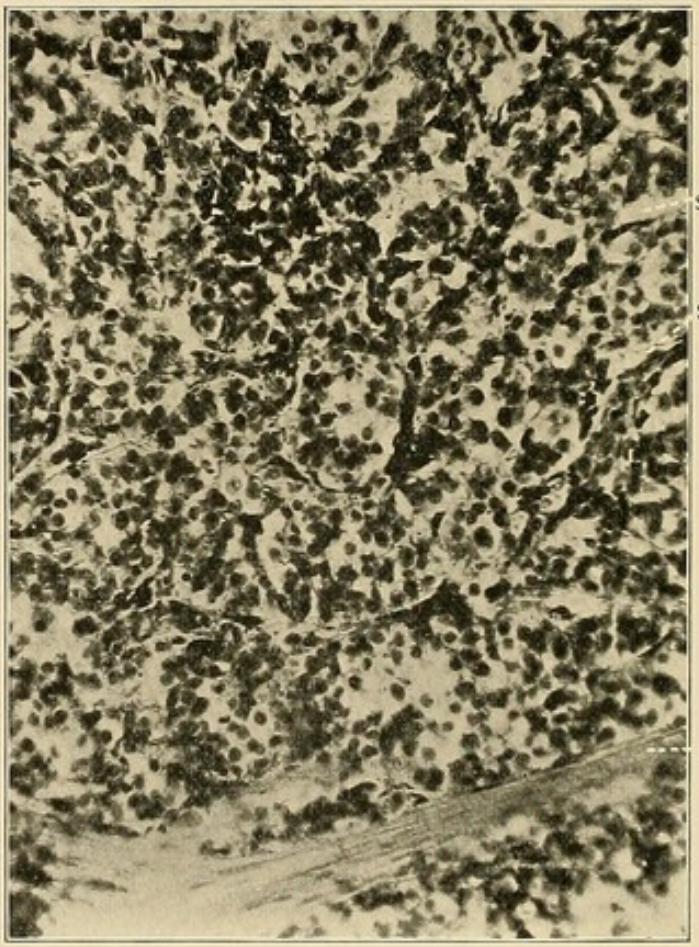
SARCOMA—ALVEOLAR—MELANOTIC. × 60.

acteristic cells; but certain other elements are found in the mixed tumors in combination with the true tumor-tissue, such as cartilage, bone, fibrous and myxomatous tissue (Fig. 314), which changes the character of the neoplasm and designates it as a mixed tumor. For instance, if the fibrous element is in excess in a sarcoma, it is called a fibro-sarcoma; if cartilage is present, it is termed a chondro-sarcoma; if bone enters into its composition, it is designated an osteo-sarcoma, and so on. (Figs. 314, 316, 317, 320.) Ziegler says, "It is not rare for three or four kinds of neoplastic tissue to be found within the same tumor."

Retrogressive Changes.—Sarcoma is prone to undergo retrograde changes early in its clinical history. This is due to the absence of a well-developed reticulum, the great activity in cell-multiplication, and the abnormal vascular supply, both as to the amount of blood and the character of the vessels, which are large and thin-walled, often appearing as mere channels between the cells.

Fatty degeneration is the most common retrogressive change which takes place in sarcoma. This is eminently true of those forms

FIG. 311.



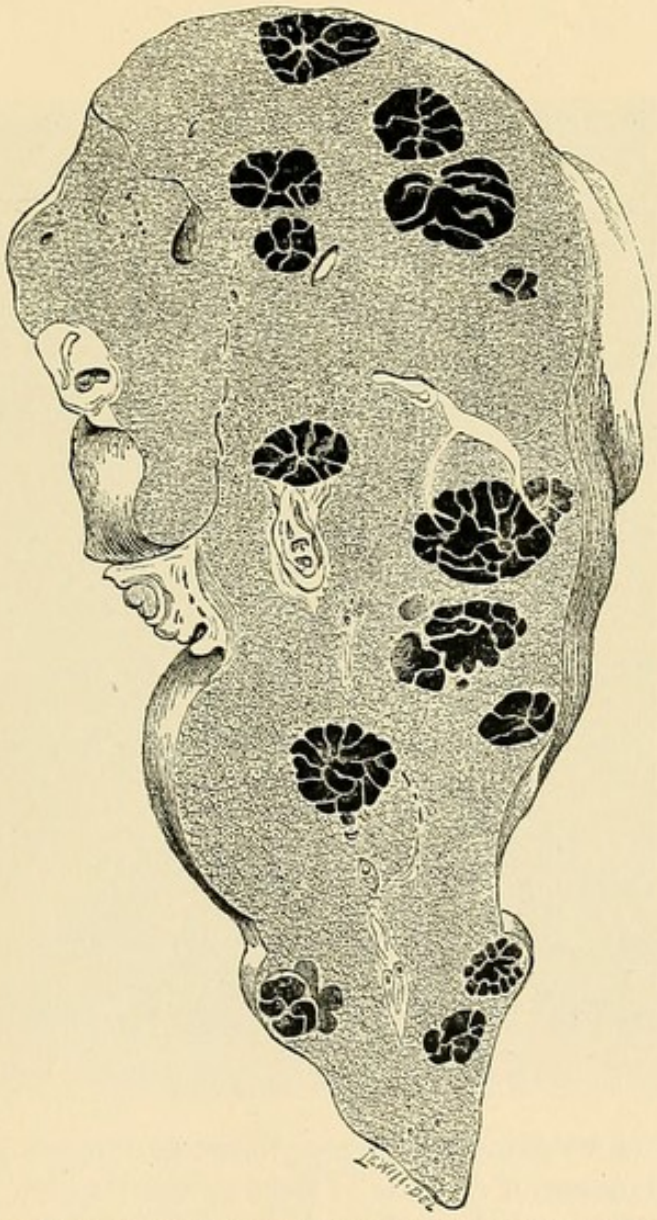
MELANOTIC SARCOMA. × 150.

which are most cellular in structure and most active in growth. Hyaline degeneration is not so common in sarcoma as in carcinoma. Myxomatous degeneration is the most frequent form of retrogressive change in sarcomatous growths. (Figs. 313, 314.) Caseation is occasionally observed, but this, in all probability, is the result of infection with the tubercle bacillus, as it is an open question whether local anemia alone can produce it.

Ulceration and sloughing is a common occurrence in sarcoma, and takes place as soon as the tumor by invasion or pressure pene-

trates the overlying tissues and reaches a free surface. Portions of the tumor often break down before this growth penetrates the overlying tissues, as the result of the rupture of the softened walls of the blood-vessels, giving rise to extravasations of blood, which may result in spurious blood-cysts, or, by leaving the coloring matter behind, lead to a mistaken diagnosis of melano-sarcoma.

FIG. 312.



SECONDARY NODULES OF MELANO-SARCOMA IN THE LIVER. (After Sutton.)

Sarcoma frequently takes on transformations of its tissue into higher physiologic types. These transformations most frequently take place in connection with periosteal sarcoma and sarcomatous epulides. (Fig. 316.) Chondrification and ossification are the two principal types of transformation which take place in these tumors.

Infection and Dissemination.—Local infection takes place by cell-migration. Sarcoma cells possess to a higher degree than carcinoma cells the power to migrate from the primary tumor into the surrounding connective-tissue spaces. These spaces serve the purpose of a stroma, in which the recently migrated cells become located and establish independent foci of neoplastic growth. Later the pre-existing connective tissue is absorbed, and in its place is formed the characteristic reticulum produced by the growth of the fibrillated sarcoma cells.

FIG. 313.



MYXO-SARCOMA. × 100.

Sarcoma in its growth displaces tissue to a much greater extent than does carcinoma; it extends in the directions which offer the least resistance, but no tissue is sufficiently dense to offer a permanent barrier to its extension, for eventually it attacks and destroys adjacent tissue, regardless of its anatomical structure. (Senn.) Cartilage of all the tissues is the last to be invaded and destroyed by sarcoma.

Regional infection takes place by the disease following the sheaths of blood-vessels and veins, but it is rarely disseminated through the lymphatics. This is explained by the fact that sarcomata have no

lymphatic vessels. Regional lymphatic infection, therefore, when it does take place through the lymphatics, must be by migration of cells to vessels of this character lying in the immediate neighborhood. As a rule, regional infection does not take place so early in sarcoma as in carcinoma.

General dissemination takes place through the circulation, and it occurs at an earlier period in the life-history of sarcoma than it does in carcinoma. This may be explained by the intimate and extensive

FIG. 314.



SARCOMA—MYELOID AND OSTEOID, WITH MYXOMATOUS DEGENERATION—OF INFERIOR MAXILLA. × 60.

relationship which exists between the blood-vessels and the tissue of the tumor. In this respect sarcoma may be said to possess greater powers of malignancy than carcinoma. General dissemination takes place much more frequently in the small round-celled sarcoma than in the spindle or giant-celled variety. (Senn.) Metastatic deposits are most frequently found in the lungs, and next in order of frequency are the spleen, the kidneys, and the liver.

Causes.—"*The essential cause* of sarcoma is the presence of a tumor-matrix of embryonic fibroblasts, of either congenital or post-natal origin, which produces an atypical proliferation of connective-tissue cells," as a result of irritation and especial environment.

That certain individuals inherit a tendency or predisposition to sarcoma is generally recognized. Sarcoma as a congenital tumor is very rare. Age does not play so important a part in the predisposition to sarcoma as it does in carcinoma. No age, from infancy to extreme old age, is exempt from the disease. It is most frequently seen in childhood and early adult life. Youth predisposes to sarcoma of the bone; old age to sarcoma of the glandular organs. Chronic irritation and chronic inflammatory conditions predispose the tissues to sarcomatous growths.

The exciting causes of sarcoma are traumatic injuries and various forms of irritation. The effects of traumatic injuries are more decided in the production of sarcoma than in carcinoma. The development of a sarcoma at the seat of a bruise, a contusion, or of a fractured bone has been frequently observed. The influence of chronic irritation and of trauma in the production of sarcoma has been conclusively shown in connection with the origin of sarcoma in warts and pigmented moles. Either of these skin defects may remain entirely harmless throughout a lifetime, but under the provocation of chronic irritation or injury they may take on sarcomatous transformation and develop into a melano-sarcoma. Upon the location occupied by a sarcoma and the character of its tissue will depend the clinical significance.

Diagnosis and Symptoms.—The diagnosis of sarcoma is often a difficult matter, and involves a careful study of the clinical history and a minute examination, supplemented by an exploratory puncture and a microscopic examination of sections made from fragments of the tumor removed for that purpose.

Two questions will claim answers in each case presenting for examination: First, Is it an inflammatory swelling? Second, Is it a malignant growth? and if so, is it a sarcoma or a carcinoma?

The clinical history will in a majority of cases decide the first question; if not, it may sometimes be settled by an exploratory puncture, while in a case in which a diagnosis between sarcoma and gumma is not clear the matter can be cleared of doubt by placing the patient upon a vigorous anti-syphilitic treatment for a few weeks.

The second question may be answered by the clinical history, so far as concerns the class of malignant growths to which the tumor belongs,—except when the primary growth is located in the tonsil, the testicle, or the lymphatic glands,—by the early infection of the lymphatic glands in carcinoma and their comparative immunity in sarcoma; by the frequent presence of cysts in sarcoma; by the greater

mobility of the sarcomatous tumor; by the freedom from early involvement of the skin; by the enlargement of the superficial veins, and by the greater rapidity of growth in sarcoma. There are, however, exceptions to this latter rule. Malignant epulides grow slowly.

The shape of the tumor is also an aid in the diagnosis. A sarcomatous tumor is usually smooth, globular, oblong, flat, or spindle-shaped (Senn), the form being governed by the particular location of the tumor and the character of the tissues in which it is imbedded.

Pain is not a marked or constant symptom. In neuro-sarcoma, or where a nerve is involved by pressure, pain is sometimes severe. Central sarcoma of bone does not always cause severe pain. The general health of the patient is rarely affected by the disease until ulceration or general dissemination has taken place.

Prognosis.—The prognosis in sarcoma is always grave, but its gravity is modified by the location and accessibility of the tumor. In tumors located in tissues of great physiologic importance or in close proximity to vital organs, the prognosis is most grave. The most malignant forms of sarcoma are the soft, small-celled varieties. These grow rapidly and produce early regional infection and general dissemination. The degree of malignancy may be increased by the rapidity of the growth. Melano-sarcoma is the most malignant of all the varieties, and is the least amenable to treatment. A fatal termination takes place earlier in sarcoma than in carcinoma. Recurrence takes place more frequently and more rapidly in sarcoma than in carcinoma. Errors in diagnosis are more frequently made in the most malignant varieties of sarcoma on account of the close resemblance between these forms of sarcoma and inflammatory swellings.

The spindle-celled and giant-celled sarcoma are the most amenable to treatment, and therefore offer the most favorable prognosis. The forms of sarcoma which are most favorable for operation are the sarcomatous epulides and myeloid sarcoma of bone. Recurrence in the subcutaneous or muscular fascia is to be treated by removal as often as it may appear, provided the growths are accessible and the general system has remained free from dissemination. Permanent cures have sometimes resulted from repeated operations for the removal of recurrent growths.

Treatment.—The character of the treatment will depend upon the variety of sarcoma which has to be dealt with, and the tissue or organ in which it is located. Operative treatment, to be of any real value, must be undertaken before regional and general infection has taken place. It has been urged in the treatment of carcinoma that early operation was imperative if a successful issue was to be looked for. In the treatment of sarcoma this advice should be doubly emphasized, on account of the fact that local, regional, and general dissem-

ination of the disease takes place at a much earlier stage in the history of sarcoma than in carcinoma, and that for these reasons the disease sooner passes beyond the limits of successful surgical procedure. (Senn.) Radical operations only hold out any hope of a cure of the disease, and recurrences should be treated in the same manner.

The removal of the central myeloid sarcoma by scraping and the enucleation of myeloid sarcoma of the inferior maxilla have in a few cases proved successful, but it would be safer to remove at the same time as much of the surrounding tissue as might be done without danger to the patient. In sarcoma of the long bones and of the soft tissues of the extremities, especially if the tumor is of the round-celled type, high amputation is often advised. Sarcoma of the superior maxilla calls for removal of the entire half of the affected side. When the disease is located in the lymph glands or the tonsils, operative treatment is of little avail, and except as a palliative measure it should be rarely undertaken. Removal of sarcoma by excision should include a zone of apparently healthy tissue from half an inch to an inch in width, according to the size of the growth and its location. The overlying skin or mucous membrane of a sarcoma should invariably be removed with the neoplasm.

From time to time various drugs have been lauded as curative in sarcoma, but after ample experience in practical demonstration it is the consensus of opinion of the most eminent surgeons all over the civilized world that no drug which has so far been presented to the profession has any real curative effect upon the disease. It has been known for a long time that occasionally an intercurrent attack of erysipelas has proved curative in sarcoma.

Busch made the discovery of the apparent curative power of erysipelas in sarcoma, and reported two cases in which accidental inoculation of sarcomatous tumors with erysipelas greatly reduced the size of the growths, and in one of these cases which died from facial erysipelas ten days after the attack the autopsy showed extensive fatty degeneration of the sarcoma cells. Fehleisen made the attempt some years ago to cure sarcoma and carcinoma by inoculation with the coccus of erysipelas, but his hopes were not realized. Lately this treatment has been revived by hypodermic injections of the toxins of erysipelas instead of the streptococcus, for the cure of inoperable sarcoma. The toxin of the *Bacillus prodigiosus*, combined with that of the *Streptococcus erysipelatus*, increases its reaction. So far this treatment has not proved as successful as it was hoped it would. More recently serum from immunized sheep inoculated with erysipelas toxins has been tried in the same class of cases with apparent success.

REVIEW.

CHAPTER LVII.

Give the definition of sarcoma.
What relationship does sarcoma hold to the mesoblastic group of tumors?
Of what is sarcoma composed?
What is the origin of sarcoma?
In what class of tissues does it invariably originate?
Name some of these tissues.
What was Virchow's opinion as to the origin of sarcoma cells?
What does Ziegler teach upon this point?
What is always the character of the tumor-cells found in sarcoma?
What marks the malignant character of sarcoma?
How are sarcomata usually classified?
Name the varieties.
How are the round-celled sarcomata divided?
Of what do the round-celled sarcomata consist?
What is the character of the cells?
What is the particular character of the blood-vessels?
Do sarcomata contain lymphatics?
Where are the round-celled chiefly found?
What is the appearance of these tumors on section, etc.?
How does the large round-celled sarcoma differ from the small variety?
What is the arrangement of the intercellular substance in these growths?
What is the character of the blood-supply?
Where is it most commonly found?
What is its degree of malignancy?
How does lympho-sarcoma differ from the preceding varieties?
How does it differ clinically?
What is the degree of its malignancy?
In what structures is it most commonly found?
How may it be differentiated from hyperplastic lymphoma?
What form of sarcoma is the most common?
Upon what condition does the degree of malignancy depend?
How is this form of sarcoma usually divided?
Where are these tumors most frequently located?
What is their histologic composition?
What are the forms of the cells?
What is the form of the cell described by Hamilton?
Is the stroma scanty or abundant?
What forms the delicate reticulum?
What substance does myeloid sarcoma resemble?
From what does it derive its name?
What form of cells are characteristic of myeloid sarcoma?
From what tissue does this form of sarcoma usually arise?
What forms of cells are found in this class of tumors?
In what location is the periosteal form of myeloid sarcoma usually found?
What objection does Sutton raise to the use of the term myeloid sarcoma?
What are the characters of the blood-vessels in central myeloid sarcoma?

For what other condition may these tumors be mistaken?

What is an alveolar sarcoma?

What is the cause of the alveolar arrangement?

What other form of tumor does it closely simulate?

Where are the blood-vessels found?

What peculiarity in its form distinguishes it from carcinoma?

Where are alveolar sarcomata most commonly found?

What growths are they associated with when found in the skin?

What is the composition of melano-sarcoma?

What particular feature of these growths distinguishes them from other forms of sarcoma?

What is the degree of its malignancy?

What is the most common seat?

By what conditions are these growths early characterized?

What is the origin of the pigment material?

What condition may appear in certain forms of sarcoma which would give them the appearance of melano-sarcoma?

Are the secondary growths pigmented?

What are the mixed-cell sarcomata?

What are the various tissues found in these growths?

By what terms are they designated?

What are the causes of the retrogressive changes in sarcomata?

What is the most common retrogressive change in sarcomata?

In what class of tumors is hyaline degeneration most common?

Is myxomatous degeneration common or rare?

Does caseation ever occur?

What is said of ulceration?

What higher types of tissue-transformation may take place in these tumors?

How does local infection take place?

What purpose do the tissue-spaces serve in the migration of the sarcomatous cells?

How is the reticulum produced?

Do sarcomata displace tissue or absorb it in their growth?

What tissue of the body is the last to be invaded and destroyed by sarcoma?

How does regional infection take place?

Through what avenue does general dissemination take place?

In what form of sarcoma is general infection most frequent?

In what organs are metastatic deposits most frequently found?

What is the essential cause of sarcoma?

Is the tendency to the formation of carcinoma inherited?

What influence does age possess in predisposing to sarcoma?

At what period of life is sarcoma most frequent?

What local conditions predispose to sarcoma?

What are the exciting causes of sarcoma?

How has the influence of chronic irritation and trauma been demonstrated as the exciting cause of sarcoma?

Upon what does the clinical significance of sarcoma depend?

What are the means of diagnosis? What two questions will claim attention in every case presented for examination?

What will usually decide the first question?

How may a doubtful diagnosis between sarcoma and gumma be cleared up?

How is the second question answered?

What are the symptoms of sarcoma?

Is the general health affected?

What is the prognosis of sarcoma?

What modifies the gravity of the prognosis?

Which are the most malignant forms?

How may the degree of malignancy be measured?

Which form is least amenable to treatment?

In what varieties of sarcoma are errors of diagnosis most liable to be made?

Is operative treatment ever curative?

How are recurrences to be treated?

Upon what will the character of the treatment of sarcoma depend?

At what period must operative treatment be taken to be of value?

What sort of operations only give hope of a cure?

What methods sometimes prove successful in myeloid sarcoma?

What kind of an operation is imperative in sarcoma of the superior maxilla?

What line of treatment should be adopted in sarcoma of the lymph-glands and tonsils?

How is removal by excision to be practiced?

Has systemic medication any value?

Is the erysipelas toxin curative?

CHAPTER LVIII.

SARCOMATA (Continued).

Sarcoma of the Jaws.

Of all the tumors which affect the maxillary bones, the sarcomata are by far the most frequent. The varieties which are found in this location are the round-celled, the spindle-celled, and the giant-celled sarcoma. In addition to the primary growths which may appear in the maxillary bones and their appendages, they are liable on account of their surroundings to be invaded by sarcomatous growths arising in the orbit, the nose, and the naso-pharynx. Sarcomata of the jaws in a large majority of cases belong to the mixed-cell varieties.

Sarcoma of the jaws arises from three sources, the *periosteum*, and the *muco-periosteum* lining the antrum of Highmore and the nasal fossa, and the *cancellated structure* of the bone. Sarcoma arising from the first two of these sources usually belongs to either the round or the spindle-celled variety. The round-celled variety is the most malignant, the giant-celled the most benign; while in the mixed-celled varieties the malignancy is governed by the number of round cells present in the growth. The giant-celled variety is most often found in the cancellated structure of the body of the lower jaw.

The consideration of sarcoma of the jaws may be divided, for convenience, according to the location of the tumor, into four heads, viz:

Periosteal Sarcoma;

Muco-periosteal Sarcoma;

Endosteal Sarcoma (Myeloid);

Odonto-sarcoma.

Periosteal Sarcoma.—This variety, as already stated, may be composed of either the round or spindle-celled form of sarcoma. Periosteal sarcoma may arise from any surface of the maxillary bone. The disease is most frequently associated with the alveolar processes and the gum; less frequently with the facial surface, and very rarely with the palatal process. Periosteal sarcoma rarely makes its appearance before the fifteenth year, though it has occasionally been seen in very young children. The period of life in which it most commonly develops is between the twentieth and the sixtieth years of age.

These tumors are more frequently associated with the upper than

with the lower jaw, and they sometimes grow to very large dimensions, and cause great deformity. Fig. 315, described by Heath, is peculiar in the fact that the disease involved both halves of the superior maxilla, appearing first upon the left and later upon the right side. When growing from the alveolar processes and gum, it appears in its early stages as a small, rounded tubercle, generally of bright red color, and located between two teeth, being attached apparently to the gum, sometimes by only a slender pedicle. It is, however, periosteal or peridonteal in its origin, for it is quite common to find it growing from the alveolus of the tooth, and in intimate relation with the peridental membrane. As the tumor grows, it often pushes the tooth aside and

FIG. 315.



SARCOMA—ROUND-CELLED—OF THE SUPERIOR MAXILLARY BONES, INVOLVING BOTH SIDES OF THE FACE. (After Heath.)

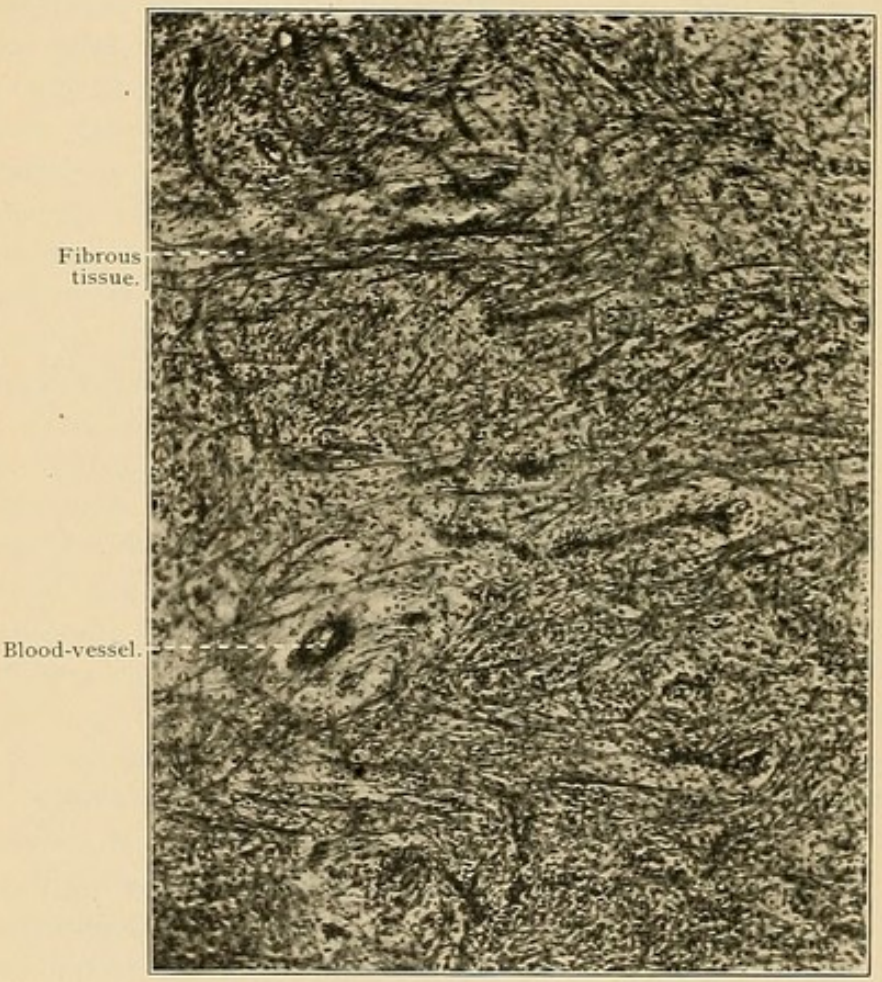
loosens it. Later the bone becomes softened and eroded, and the whole structure of the jaw may become involved. The small-celled varieties of sarcoma are the most destructive. After the tumor has reached a size that causes it to project beyond the crowns of the teeth, it is subject to various injuries from mastication. Inflammation is induced, and ulceration sets in, which aggravates the local condition, and increases its malignant tendency.

This form of tumor is known as *epulis*. The term epulis is given to growths of all forms which appear from the gum, but its use is generally restricted to the periosteal form of sarcoma of the jaw. An epulis may contain round cells, but it is more commonly composed of spindle-cells. Giant cells are also found in these growths in connection with the round or spindle-cells. (Fig. 316.)

Virchow recognized two forms of epulis, the *hard* and the *soft*.

The hard variety contains a large quantity of fibrous tissue, a few small cells, and giant cells are seen scattered between the fibers. The soft variety is quite vascular, and hemorrhage frequently occurs within its substance, which after absorption of the blood leaves the pigment-granules behind, and gives the tumor a brownish hue. These tumors have been designated *pigment epulis*. Periosteal sarcoma springing from the outer surface of the ramus might readily be mistaken for a parotid tumor.

FIG. 316.



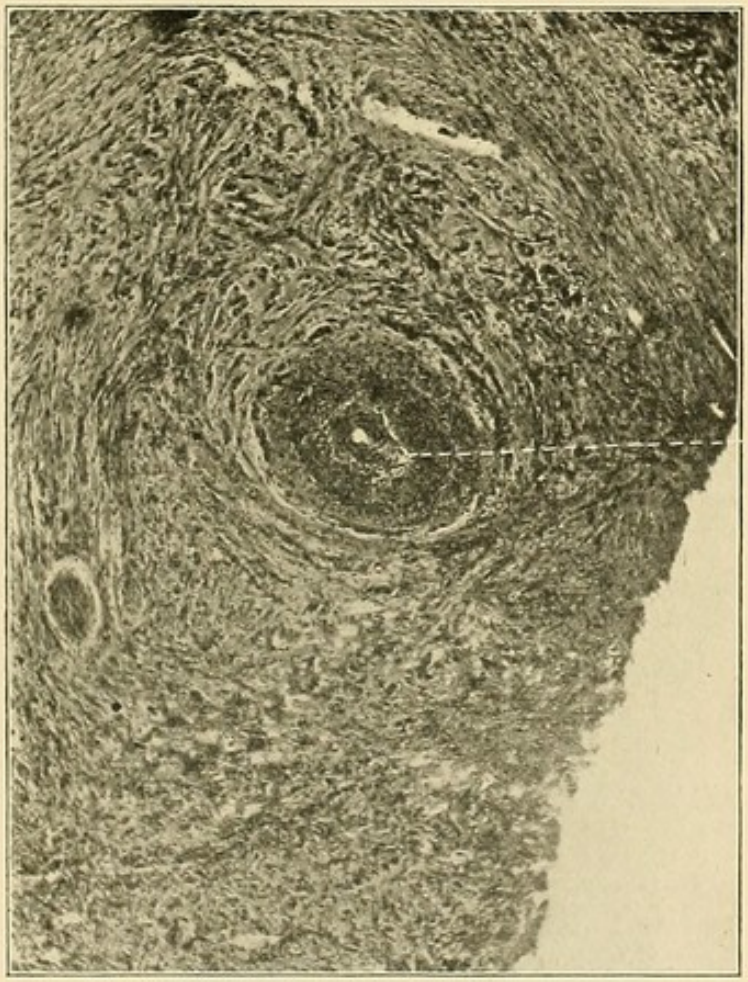
EPULIS WITH SARCOMATOUS CELLS. SARCOMATOUS TRANSFORMATION. × 50.

A common form of mixed-sarcoma is the fibro-sarcoma; tumors in which the fibrous element predominates over that of the sarcoma cells. These are most frequently seen in relation with the maxillary bones and alveolar processes. Fig. 317 shows the structure of a tumor of this character which was located in the malar bone.

Myxomatous degeneration frequently occurs in this variety of sarcoma. Fig. 320 shows the process taking place in sarcoma of the malar bone. Cartilaginous transformation sometimes takes place in the periosteal sarcoma (chondro-sarcoma), or ossification represented

by delicate spiculæ of bone may take place (osteo-sarcoma). Figs. 318 and 319 represent an osteo-sarcoma of the left side of the inferior maxilla of a negro boy. Fig. 318 was made from plaster casts of the case before the operation. Fig. 319 shows the tumor after its removal. The more dense the tumor the slower is the growth, and consequently there is a diminished degree of malignancy, as a result of the decreased liability to regional and general infection. The disease if left to run its course, sooner or later causes extensive softening and erosion of the

FIG. 317.



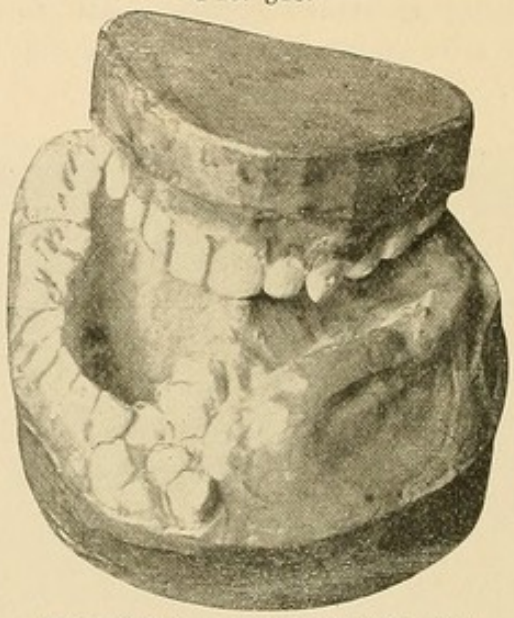
FIBRO-SARCOMA OF THE MALAR, SHOWING PERIARTERITIS. × 60.

bone from which it sprang, and it may involve the entire thickness of the jaw. Infection of the lymphatics takes place, and if the patient holds out long enough, the disease may be disseminated through the system, producing metastasis in the lungs, liver, and kidneys.

Muco-periosteal Sarcoma.—The most common location of sarcoma arising from the muco-periosteum is the antrum of Highmore. These are often of the giant-celled or myeloid variety. Figs. 321 and 322 show the pathologic histology of one of these growths. Sarcoma

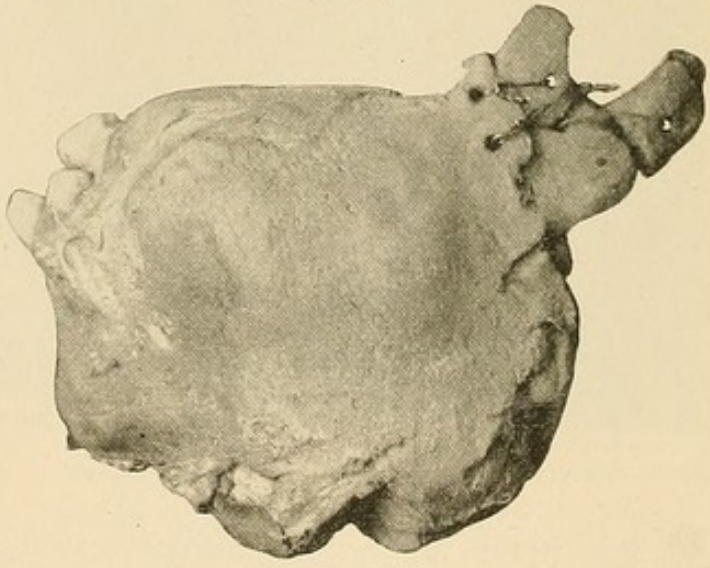
growing in this location soon fills the cavity and produces thinning and expansion of its walls. As the bone is expanded it encroaches upon the nasal fossa, obstructing the air-passages and interfering with nasal breathing. Absorption of the orbital plate may also take place to such an extent as to permit of bulging of the floor of the

FIG. 318.



OSTEO-SARCOMA OF LOWER JAW. (A.)

FIG. 319.

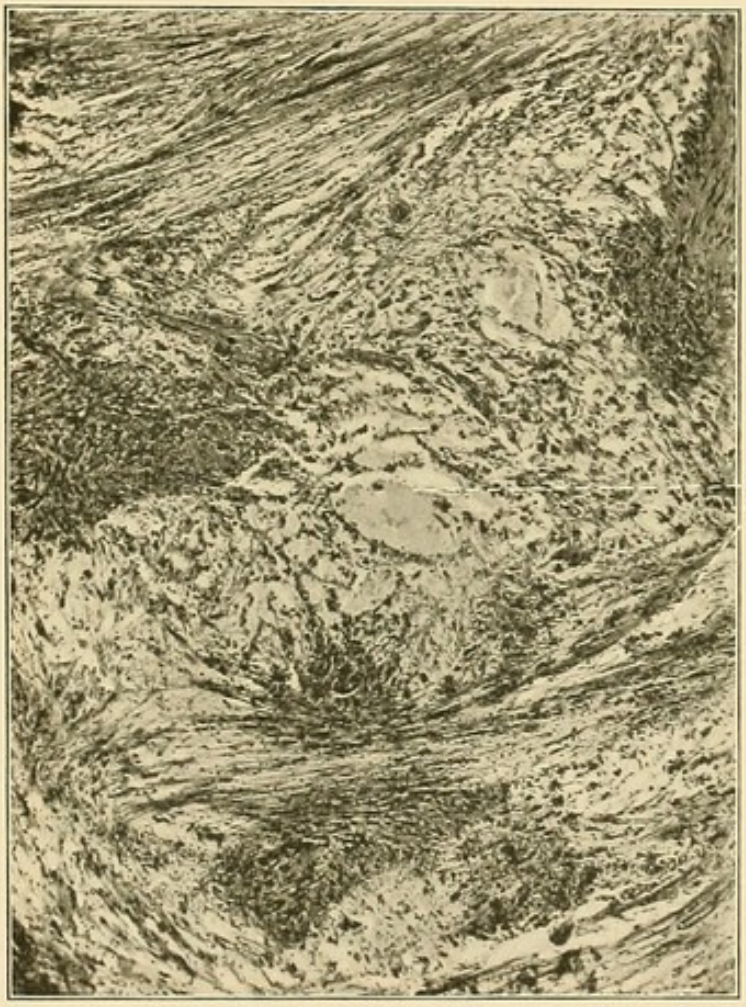


OSTEO-SARCOMA OF LOWER JAW. (B.)

orbit and displacement of the eyeball. In the more severe cases the alveolar and palate processes may be depressed; sometimes the palate process upon the affected side is decidedly convex. The nasal duct is often implicated, and sometimes complete stenosis takes place. By slow degrees the processes of the tumor finally penetrate the thin walls,

and implicate the skin of the cheek, or make their way through the floor of the antrum, and involve the mucous membrane of the palate, or following the alveolus of a recently-extracted tooth—which has become loosened by the destruction of the alveolar process—appear upon the gum; or it may project into the nasal fossa, where ulceration may take place and give rise to frequent and sometimes alarming recurring attacks of epistaxis. The tumor sometimes penetrates the posterior

FIG. 320.



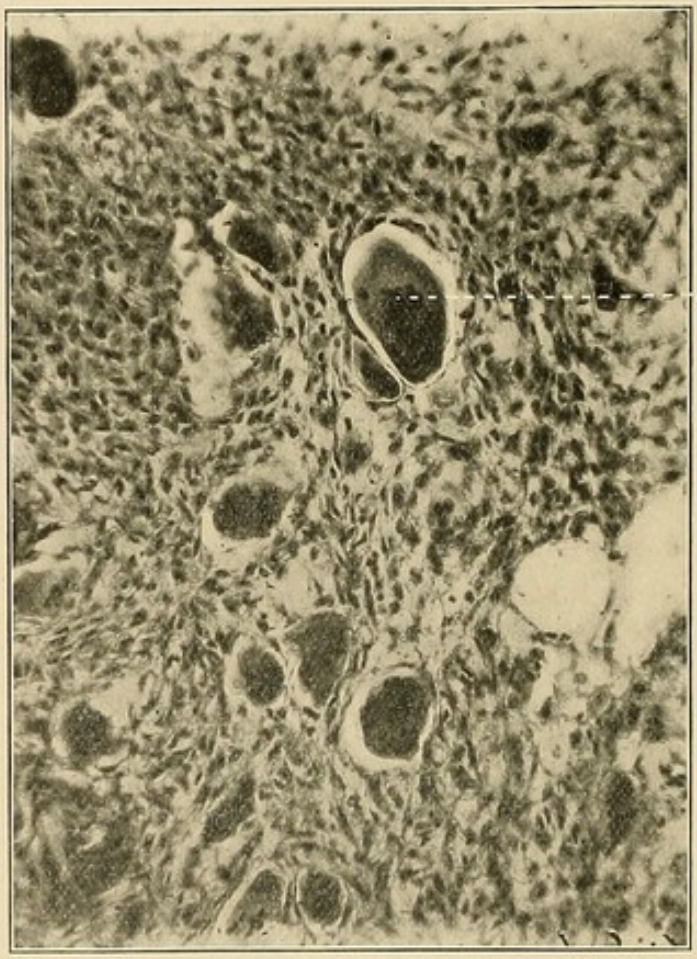
FIBRO-SARCOMA OF MALAR BONE, SHOWING MYXOMATOUS DEGENERATION. × 60.

wall of the antrum, and enters the zygomatic and spheno-maxillary fossa,—when it may be mistaken for a tumor of the parotid gland,—from which by slow degrees it creeps upward to the temporal fossa; or it may follow the spheno-maxillary fissure and involve the tissues of the orbit; or it may creep through the sphenoidal fissure or penetrate the foramen rotundum, and gain access to the middle fossa of the cranium. This is the most distressing form of sarcoma connected with the maxillary bones. It is usually very malignant, and the least amenable to

surgical treatment. Occasionally a permanent cure is obtained by complete excision of the affected half of the maxilla, but this to be successful must be made while the tumor is still confined to the limits of the antrum; in other words, during its benign stage. After the walls of the antrum have been penetrated, operation is of no permanent value. As a palliative measure it is sometimes of benefit.

The nasal passages and the antra sometimes become involved by sarcomata springing from adjacent parts, a fact which should not be

FIG. 321.



MYELOID SARCOMA OF SUPERIOR MAXILLA ARISING FROM THE ANTRUM. × 150.

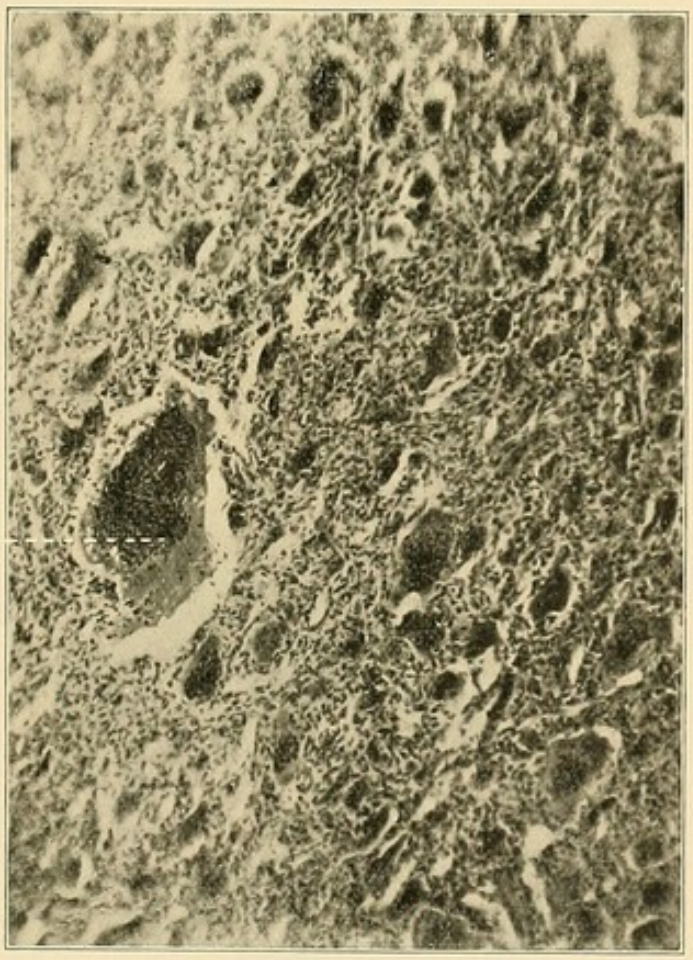
lost sight of in the study of the clinical history of these growths. The naso-pharynx and the nasal fossa are the most common locations from which such involvement may arise.

The under surface of the body of the sphenoid bone forms the vault of the naso-pharynx, which is covered with the pharyngeal mucous membrane. From the muco-periosteum of this region there occasionally develop spindle-celled sarcomata. "It is not uncommon for such tumors to extend into and plug one or both nasal fossæ, processes of the tumor appearing at the nostril; or they may extend downward

into the pharynx and impede deglutition." (Sutton.) Tumors of this character are usually attended with severe and agonizing frontal headache. Profuse, recurring epistaxis is also a common symptom.

Sarcoma originating in the nasal fossa and penetrating the maxillary sinuses is not a very frequent occurrence. The disease is rarely seen except between the ages of fifteen and twenty years. A most remarkable case of this character has been reported by Moore.

FIG. 322.

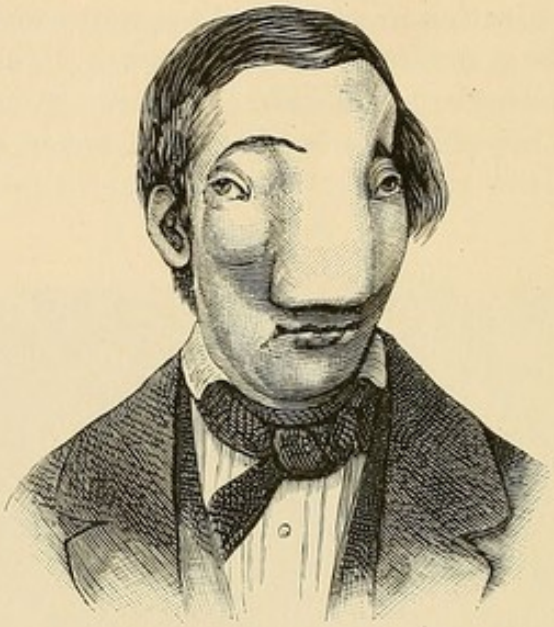


MYELOID SARCOMA OF SUPERIOR MAXILLA ARISING FROM THE ANTRUM, SHOWING GIANT CELLS. × 300.

The tumor was a mixed-cell sarcoma, having its origin in the nasal septum and extending laterally, filling both antra. As the tumor grew the face widened and projected forward, causing a most dreadful disfigurement. (Fig. 323.) Pain was absent, the sense of smell was lost, and the sight of the right eye impaired. Operation for the removal of the growth was undertaken by Moore, but the patient died while undergoing the operation, in consequence of some interference with respiration. Fig. 324 is a sagittal section of the facial region of

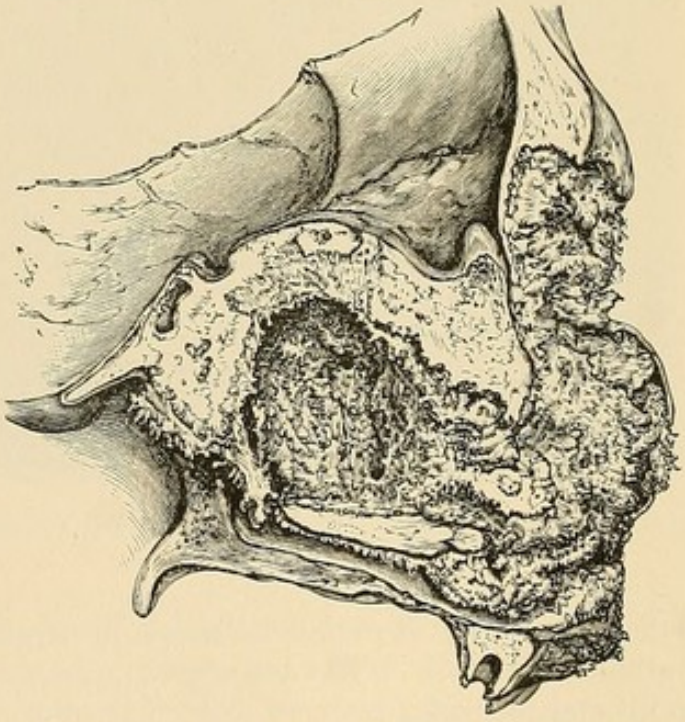
the skull of this case, showing that the sarcoma was confined to the nasal septum.

FIG. 323.



DEFORMITY PRODUCED BY A SARCOMA OF THE NASAL SEPTUM. (Moore, after Sutton.)

FIG. 324.



FACIAL REGION OF THE SKULL SHOWN IN FIG. 323, SEEN IN SAGITTAL SECTION. THE SARCOMA IS RESTRICTED TO THE NASAL SEPTUM. (Moore, after Sutton.)

Endosteal Sarcoma.—This form of sarcoma is generally located in the lower jaw, and is usually designated as *central sarcoma,* because it arises in the cancellated structure of the bone. When the disease is

located in the upper jaw, it as a rule arises in the nasal processes. They are slow-growing tumors, but they nevertheless often attain a very large size. These tumors are usually classed as giant-cell or myeloid sarcoma. They are composed of cells of a great variety of shapes and sizes, but the characteristic cells of the tumor are the giant cells. The growth is a soft, pulpy tumor, having little intercellular substance, abundant blood-vessels, and in color a brownish hue. Contrary to the usual order, though soft in structure, it is the least malignant of the sarcomata found in the jaws. Endosteal sarcoma by its growth produces expansion of the compact plates of the body of the jaw, and develops a rounded or oval enlargement of the bone. The growth of the tumor, however, is such as to cause a thinning of the bone,—in the long bones such a condition sometimes causes fracture upon very slight exertion,—which may finally be absorbed and permit the tumor to pass the boundaries of the bone. Under such circumstances, on account of the vascularity of the growth rhythmical pulsations may sometimes be felt in the tumor, simulating aneurism. The size rarely exceeds that of an orange or the fist.

Sutton is of the opinion that myeloid sarcoma of the jaws is not a disease of such frequency as the text-books would indicate, and he thinks this error is due to the fact that sufficient attention has not been devoted to a study of sarcomata arising in connection with developing teeth. Tumors of various benign forms, such as the fibrous odontomes and cementomes, frequently occur in connection with the developing teeth. The fibrous odontomes often contain a few multinucleated cells. It can therefore be readily understood how such a tumor as a fibrous odontome might, if surrounded by favoring circumstances, take on a sarcomatous transformation of the giant-celled variety.

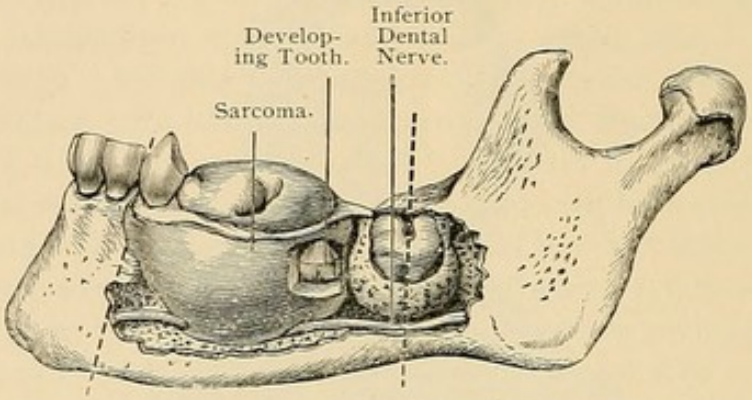
Odonto-sarcoma.—The odonto-sarcomata arise from the follicles of developing teeth. These tumors belong to the mixed-cell varieties of sarcoma, and are composed of round and spindle-cells with giant cells interspersed through the substance of the tumor-tissue. "Sarcoma of a tooth-follicle only occurs in children, and is particularly apt to involve the germ of the first permanent molar." (Sutton.) (Fig. 325.)

In the early history of these growths—like the fibrous odontome and cementome—they are distinctly encapsulated.

The reason why the first permanent molar should be particularly prone to the disease is not forthcoming. The clinical history of these tumors is very similar to that of myeloid sarcoma. Growth of the tumor causes expansion of the plates of the alveolar processes; penetration of the tumor through its bony envelope generally occurs upon the gum, which ulcerates and gives rise to hemorrhage. Ulceration of the

gum is usually followed by infection of the neighboring lymphatic glands, and general dissemination may take place, but in this regard they are no more liable than the myeloid sarcoma.

FIG. 325.



SARCOMA ARISING IN THE FOLLICLE OF A DEVELOPING TOOTH. THE DOTTED LINES INDICATE THE AMOUNT OF THE JAWS REMOVED AT THE OPERATION. (After Sutton.)

Sarcoma of the skin is an exceedingly rare affection. It is occasionally seen, however, in connection with pigmented warts and moles, when it assumes the form of melano-sarcoma.

Sarcoma of the Salivary Glands.—Primary sarcoma is not infrequently observed in the salivary glands. The parotid is more often the seat of the disease than the submaxillary or the sublingual gland.

According to Billroth, about three-fourths of the tumors found in the parotid gland are sarcomatous. The period of life at which these tumors are most liable to develop is between fifteen and twenty-five years of age. These tumors are usually of the mixed type, chondro-sarcoma being the most common form.

Primary sarcoma of the parotid gland may be described as a smooth, lobulated, oval, elastic, rapid-growing tumor, located directly in front of or behind the angle of the jaw. (Fig. 326.) By its rapid growth it soon involves the entire gland. As the tumors increase in size they sometimes become tuberculous and implicate the tragus of the ear. When allowed to progress uninterruptedly "they burrow deeply within the tissues of the neck, dip beneath the sterno-mastoid, and acquire attachments to the carotid sheath; sometimes they creep upward and adhere to the under surface of the petrosal, and pushing toward the median line, so bulge the pharyngeal wall inward as to impede deglutition." (Sutton.) The rapid-growing tumors are prone to early perforation of the capsule, and a tendency to involve the skin and to ulcerate. In the growth of the tumor the facial nerve is often implicated by the extension of the tumor behind the ramus of the jaw, and occasionally paralysis follows. Senn mentions two cases in which paralysis of the facial nerve was a complication.

In structure these tumors exhibit a great variety of forms; some may be pure sarcoma, but a majority of them will be fibro-, myxo-, or chondro-sarcoma. The pure sarcoma is composed of round or spindle cells, and is inclosed within a capsule from the beginning.

The fibro-sarcomata are spindle-celled tumors. Myxo-sarcomata are composed of round and spindle cells, and myxoma cells; these tumors may attain the size of a child's head. They are round and soft, and the tissue of which they are composed is yellowish or reddish in color. The chondro-sarcomata are nodulated upon the surface, and consist almost entirely of hyaline cartilage arranged in lobules bound

FIG. 326.



PAROTID SARCOMA IMPLICATING THE PINNA IN A WOMAN THIRTY-FIVE YEARS OF AGE. (After Sutton.)

together by loose connective tissue. (Sutton.) The cartilage cells rarely possess capsules, and often present the stellate form of immature cartilage. The chondro-sarcomata are prone to mucoid degeneration, while the fibro-sarcomata are more liable to undergo a myxomatous change.

On account of the great variety of tissues found in a sarcoma of the parotid gland, it often becomes exceedingly difficult to make a positive diagnosis as to the character of the tumor. Sutton says, "It is not unusual in sections from a parotid sarcoma to meet with spindle cells, cartilage, myxomatous tissue, glandular acini, and fibrous tissue in an area two centimeters square." As a rule, the most complex

tumors grow the most rapidly. Some of them infect the lymphatic glands in their neighborhood, and cause metastatic deposits in the lungs. Operation to be successful must occur while the tumors are in their benign state. After infiltration of the tissues takes place in the rapid-growing varieties, complete removal often becomes impossible.

A fatal issue of the disease may be from dysphasia induced by pressure upon the pharynx; hemorrhage from ulcerative changes in the large vessels of the neck or from secondary deposits in the lungs, inducing broncho-pneumonia. (Sutton.)

Sarcomata of the submaxillary glands are most commonly of the cartilaginous type, and are usually encapsulated and readily enucleated. When they are the seat of the rapid-growing varieties, the whole gland should be extirpated, together with the surrounding connective tissue, at least to an extent that will insure the removal of all diseased tissue.

REVIEW.

CHAPTER LVIII.

Which are the most frequent tumors found in the jaws?
What varieties of sarcoma are most commonly found in this location?
From what three sources are sarcomata of the jaw derived?
To what class of tumors do the first two belong?
Which variety is the most malignant and which the most benign?
What governs the malignancy of the mixed-cell variety?
Where is the giant-cell variety usually found?
Into how many varieties may sarcomata of the jaws be divided?
Name these varieties.
What is the composition of the periosteal sarcoma?
Where is it most frequently located?
At what period of life is it most frequent?
With which maxilla is it most commonly associated?
What is its clinical appearance when growing from the alveolar processes and gums?
From what particular tissue does it originate?
How are the teeth and bone affected by its growth?
What are some of the causes of ulceration of these growths?
What other term is applied to tumors of this class?
What form of cells are most commonly found in this class of tumors?
How many forms of epulis are recognized?
What is the structure of the hard variety?
How does the soft variety differ from this?
What forms of transformation may take place in these growths?
What favorable change does this transformation produce?
How does the disease terminate when left to itself?
What is the most common location of muco-periosteal sarcoma?
What are the progressive stages of the disease when located in the antrum?

What is the degree of malignancy of these growths?

Are these cases curable after the disease has penetrated the wall of the antrum?

From what source may the maxillary bones become secondarily involved?

In what portion of the naso-pharynx do sarcomata sometimes arise?

What is the appearance of such growths?

What are the symptoms?

Is sarcoma of the nasal fossa common?

At what period of life does it appear?

Where are endosteal sarcomata usually located?

What other term is applied to this form of sarcoma?

When the disease is in the upper jaw, in what portion is it usually located?

What is the character of its growth?

What is its histologic structure?

What is the degree of its malignancy?

What changes are wrought upon the bone in its growth?

What vascular tumor does it sometimes simulate?

Is this form of disease common in the jaw?

What form of tooth-tumors sometimes develops into myeloid sarcoma?

From what do the odonto-sarcomata arise?

To what class of sarcoma do these tumors belong?

At what period of life do they occur?

With what particular tooth are they most often associated?

In what respect do they resemble the fibrous odontomes?

What is the clinical history of these tumors?

What condition usually follows ulceration of the gums in this class of tumors?

Is sarcoma of the skin a common or a rare affection?

With what skin defects is it commonly associated?

Are sarcomata ever associated with the salivary glands?

Which of these glands is most often the seat of the disease?

What percentage of the tumors of the parotid glands are sarcomatous?

At what period of life do sarcomata of the parotid glands generally develop?

To what form of sarcoma do these tumors usually belong?

Describe the appearance and clinical course of primary sarcoma of the parotid gland.

What is the tendency of the rapid-growing tumors of this location?

What important nerve is often implicated in these tumors?

What may be the result of this implication?

What other forms of sarcoma are found in this location besides the chondro-sarcoma?

What kind of cells are found in the myxo-sarcomata?

What size may these tumors sometimes attain?

What is the appearance and formation of the chondro-sarcomata?

What is the most common degenerative change in the fibro-sarcomata?

Why is the diagnosis of sarcomata in this location difficult?

What form of mixed tumor grows the most rapidly?

At what period should the operation be made in order to be successful?

By what means does the disease cause a fatal termination?

Which is the most common type of sarcoma of the submaxillary glands?

What should be the character of the operation in the rapid-growing sarcomata of the parotid gland?

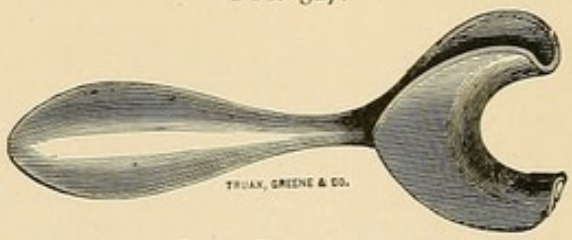
CHAPTER LIX.

TREATMENT OF SARCOMA OF THE JAWS.

It is important in all operations upon the lower jaw, even for the removal of new growths, to preserve the continuity of the bone if possible, on account of the disfigurement which so frequently takes place as a result of section of the body of the bone. The conservation of the outlines of the face must, however, give way before the necessity of a complete extirpation of the local disease.

In periosteal sarcoma of the alveolar process and the gum, and in endosteal myeloid sarcoma of limited growth, the continuity of the body of the bone may in many cases be preserved. The operation for the removal of sarcomatous growths confined to the gum and alveolar process, consists in first extracting the teeth upon either side of the

Fig. 327.



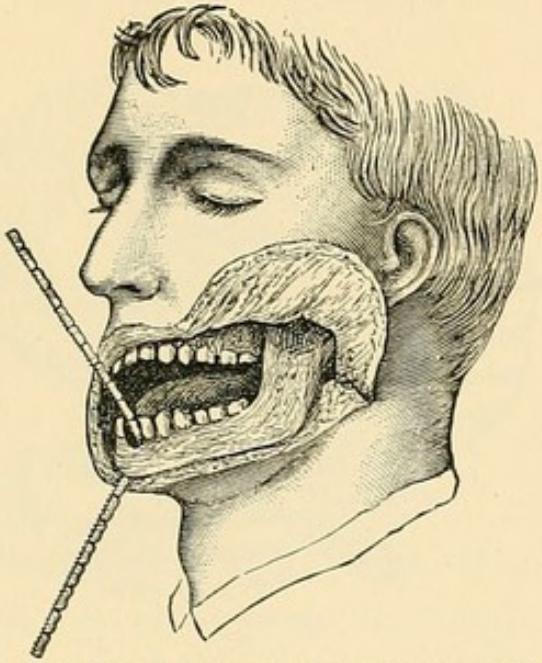
Cheek-Retractor.

growth, when with scalpel, chisel, and mallet a sufficient amount of the gum and bone may be removed to include not only the growth itself, but enough healthy-appearing tissue to insure a complete encompassment of all the prolongations of the tumor. Many times this operation may be successfully accomplished without external incision. Under such circumstances the cheek-retractor (Fig. 327) will be of great service in giving a good view of the parts to be operated upon. Occasionally it will be found necessary to operate through external incisions, especially when the growth is located in the posterior part of the jaws. The incision should be made along the lower border of the jaw, and of sufficient extent to give free access to the parts to be operated upon.

In endosteal sarcoma, even of limited extent, an external incision will give the most satisfactory opportunity for a thorough operation. This operation consists of first cutting through the compact layer of the bone at a sufficient distance from the tumor to include a portion

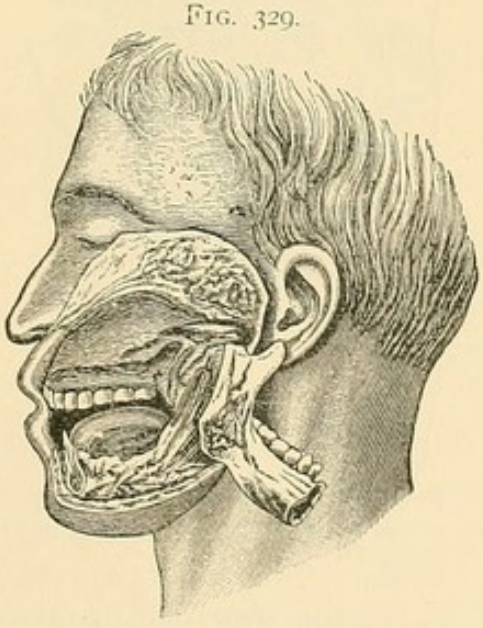
of healthy bone, and afterward, to insure its complete extirpation, curette the cancellated structure in all directions, being sure to follow up any point of suspicion. The odonto-sarcoma may be treated in the same manner.

FIG. 328.



EXCISION OF ONE-HALF OF THE LOWER JAW; EXTERNAL INCISION. (After Esmarch.)

FIG. 329.



DISARTICULATION OF ONE-HALF OF THE LOWER JAW BY TWISTING. (After Esmarch.)

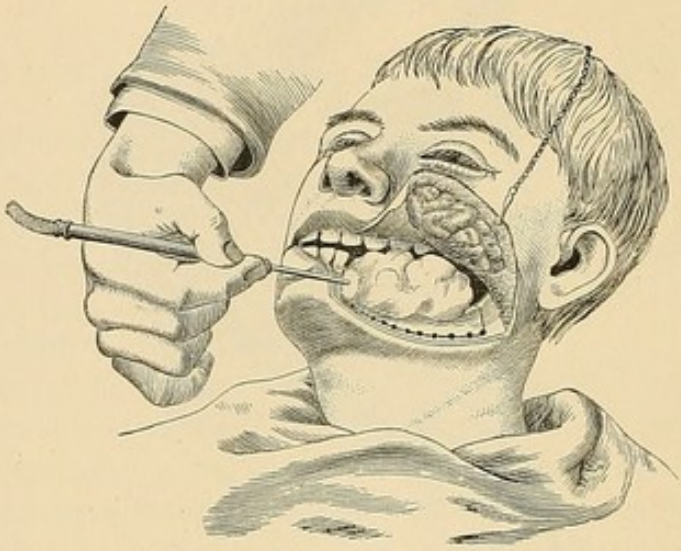
In periosteal and endosteal sarcomata which have involved the entire depth of the jaw, exsection of the diseased half often becomes necessary. Senn recommends the removal of the entire half from symphysis to articulation, as he considers the proximal fragment is

rather detrimental to the patient than useful, and the severity of the operation is not increased by such a procedure.

In any case of carcinomatous or sarcomatous disease of the jaws, no attempt should be made to conserve the periosteum.

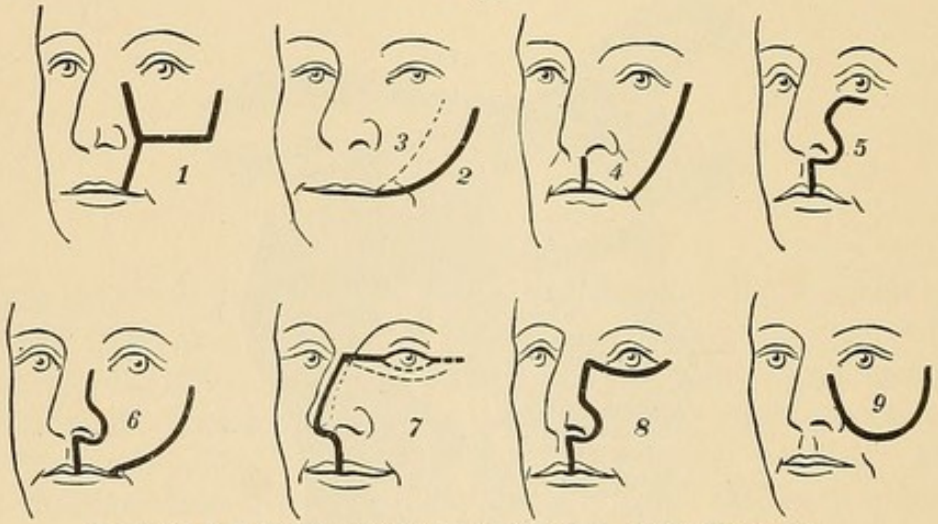
Resection of the ramus and half of the body of the lower jaw for

FIG. 330.



GARRETSON'S OPERATION FOR PARTIAL EXSECTION OF THE LOWER JAW. (After Garretson.)

FIG. 331.



INCISIONS FOR RESECTION OF THE UPPER JAW. (After Esmarch.)

1, Gensoul's; 2, Velpeau's; 3, Syme's; 4, Malgaigne's; 5, Nélaton's; 6, Fergusson's; 7, Dieffenbach's; 8, Weber's; 9, Von Langenbeck's.

sarcoma may be accomplished, after the manner of Esmarch, by an incision begun close to the posterior border of the ramus, on a level with the lobe of the ear; thence to the angle of the jaw, and forward along the lower border to the symphysis, where it may intersect a vertical incision at the median line of the lip. This flap is dissected from the bone as far as may be without opening into the cavity of the

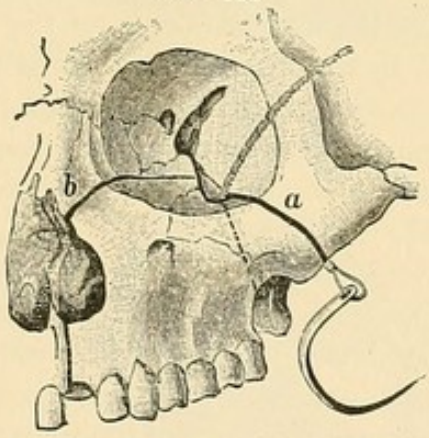
mouth, and the facial artery ligated. A central incisor tooth is then extracted. The tissues are now tunneled behind the symphysis, and the bone divided with a chain saw passed through the tunnel, the division of the bone being made from within outward. (Fig. 328.) If the surgical engine is used, the bone is divided from without inward. The jaw is then freed from its attachments to the soft tissues

FIG. 332.



EXCISION OF THE UPPER JAW.—WEBER'S INCISION. (After Weber.)

FIG. 333.



BONE-SECTION IN EXCISION OF THE UPPER JAW. (After Esmarch.)

of the floor of the mouth, and to a point near the temporo-maxillary articulation. The bone is disarticulated by forcibly twisting it backward. (Fig. 329.) The object of wrenching the bone from its joint rather than by severing its attachments with cutting instruments, is to prevent injury to the internal maxillary artery. The wound should be closed by uniting the mucous membrane of the mouth with a separate line of sutures to exclude the fluids of the mouth from the wound. The external wound may be treated in the usual manner. Drainage should be established for a few days. For partial exsection of the lower jaw in which a portion of the body of the bone can be conserved, Garretson's operation made with the drill and circular saw, driven by

the surgical engine, is *par excellence*. Reference to Fig. 330 will indicate the technique of the operation.

In operation for removal of the entire inferior maxillary bone the incisions are the same as for the removal of one half, with the exception that the lip must not be divided at the median line, and the horizontal incision is carried from the lobe of one ear down to the posterior border of the ramus and beneath the jaw to the same point upon the opposite side. The jaw is divided at the symphysis, and each half removed separately.

For the removal of the body of the bone between the angles, the same incisions may be made, and the jaw divided with the saws of the surgical engine, or with the chain saw at the angles.

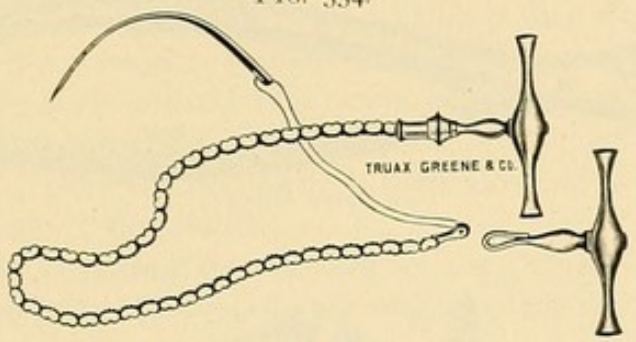
The treatment of localized sarcomatous growths—epulis and endosteal tumors—of the upper jaw does not differ from the treatment of the same affections in the lower jaw. In periosteal sarcoma and in sarcoma involving the antrum of Highmore, excision of the affected half or of the entire jaw is imperatively demanded.

The various incisions which have been recommended for exposing the superior maxillary bone preparatory to its excision, are shown in the accompanying illustrations. (Fig. 331.)

Weber's incision is the one most commonly practiced, for the double reason that it gives the best exposure of the bone, and leaves the least deformity, as it follows the natural lines of the face. (Fig. 332.) In this operation the upper lip is divided through the median line to the base of the cartilaginous septum of the nose; thence around the ala of the nose and upward along the side of the nose to a point a little below the inner canthus of the eye; then outward along the lower border of the orbit to the outer angle of the orbit. (8, in Fig. 331.) The flap is then dissected from the bone and turned outward. The orbital contents are next carefully detached from the orbital plate of the maxilla. The malar bone is divided with the chain saw, passed through the orbital fissure, along the posterior surface of the malar bone, and out at the malar fossa (Fig. 333), by means of a curved needle armed with a heavy silk ligature. (Fig. 334.) The nasal process may be divided with heavy cutting forceps (Fig. 335), and the maxillary bones divided at the median line by the chain saw. The saw is passed by first puncturing the vault of the mouth through the nasal fossa, at the junction of the hard with the soft palate on the median line, with a drainage trocar, and the saw is drawn through the mouth and out at the nostril upon the affected side. (Fig. 336.) The soft palate is then separated from the posterior border of the palate by a transverse incision. The bone may now be loosened from its other attachments by inserting an elevator into the division made through the malar bone, and then seized with Fergusson's lion-jaw forceps (Fig. 337), and the bone twisted

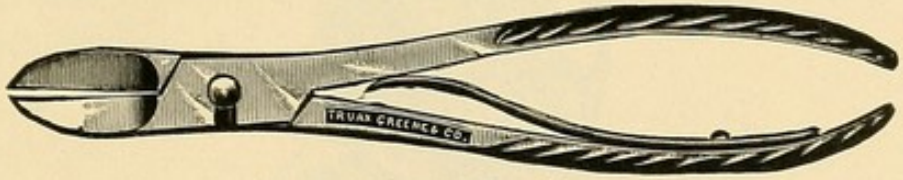
from its bed. (Fig. 338.) The internal maxillary artery is next secured and tied, but this is not always necessary, for frequently it gives no trouble from hemorrhage. Other bleeding points may be treated by torsion; capillary oozing may be controlled with sponges wrung out of hot water, or by packing. As soon as hemorrhage has ceased, the wound cavity (Fig. 339) should be packed with iodoform gauze or

FIG. 334.



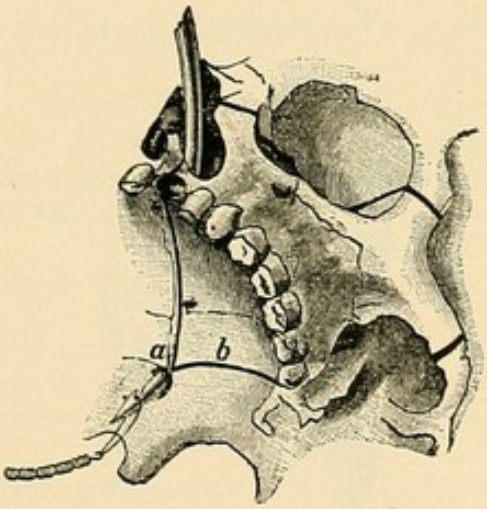
CHAIN SAW.

FIG. 335.



STRAIGHT BONE-CUTTING FORCEPS.

FIG. 336.



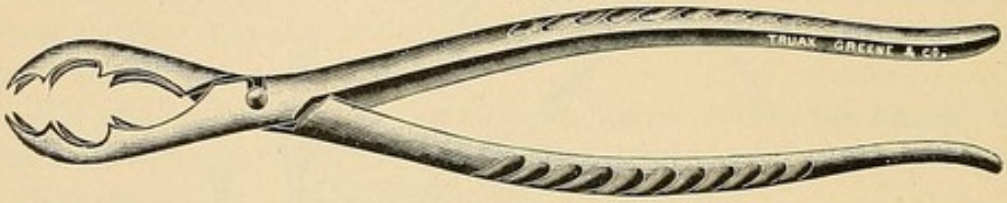
SHOWING LINE OF MEDIAN BONE SECTION AND METHOD OF APPLYING CHAIN SAW.
(After Esmarch.)

boric acid gauze, and the external wound sutured, and dressings applied. The after-treatment consists of frequent irrigation of the mouth with antiseptic washes, and careful feeding. The packing should be removed on the third or fourth day, or sooner, if it seems to cause irritation or there are signs of suppuration, which would be indicated by a

rise in the body temperature. The tampon should be replaced by a smaller one, and changed thereafter every day, the wound being most thoroughly cleansed each time with antiseptic solutions.

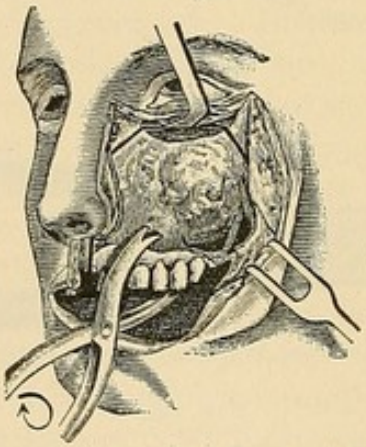
The writer prefers the surgical engine armed with suitable saws for all sections of bone that can be reached by them, to the chain saw or

FIG. 337.



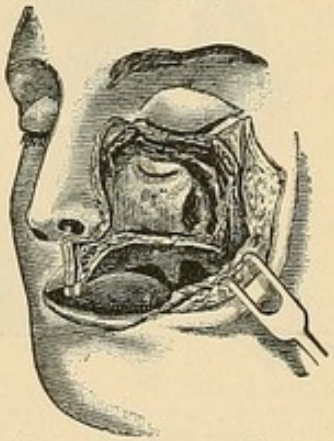
LION-JAWED FORCEPS.

FIG. 338.



REMOVAL OF THE BONE WITH FERGUSSON'S LION-JAWED FORCEPS. (After Esmarch.)

FIG. 339.



WOUND CAVITY AFTER RESECTION OF THE UPPER JAW. (After Esmarch.)

the chisel and mallet, on account of the greater neatness and the saving of time in the operation.

In simultaneous excision of both halves of the superior maxilla the same incisions are made upon both sides as just described for the removal of one; or Dieffenbach's median incision may be practiced.

(7, in Fig. 331.) This consists of dividing the upper lip and nose upon the median line, and then carrying a transverse incision to a point just below the inner canthus of each eye and outward, following the margin of the orbit to its outer angle.

For the removal of portions of the superior maxillary bones below the infraorbital foramen, Nélaton's (5, in Fig. 331) or Weber's incisions may be employed, and the soft tissues raised from the bone. After extracting an incisor tooth, the maxilla is then divided upon the median line with a narrow saw passed through the nostril, the horizontal section being made with the saw or chisel through the malar process and the maxillary tuberosity. The soft palate is detached by a transverse incision. The detached piece is then loosened with an elevator and wrenched out of its position.

Operations requiring the removal of only small sections of the alveolar process can with safety to the success of the operation be made through the mouth.

The operations for the extirpation of the parotid and submaxillary glands have already been described in the section on carcinoma of the salivary glands.

REVIEW.

CHAPTER LIX.

Why should the continuity of the bone of the lower jaw be preserved if possible in operations for the removal of malignant growths?

In what form of the disease may the continuity be preserved?

Describe the operation for sarcomatous growths confined to the gum and alveolar processes.

Under what circumstances may these operations be made without making an external incision?

Where should the external incision be made for the removal of growths in the posterior portion of the jaw?

Describe the operation for the removal of a limited sarcoma of the lower jaw.

How should the odonto-sarcomata be treated?

What should be the character of the operation in sarcoma which has involved the entire depth of the jaw?

Should the periosteum be considered in any of these operations?

Describe the operation for resection of the lateral half of the lower jaw from symphysis to articulation.

Describe the operation for the removal of the jaw from symphysis to angle.

How may it be removed from angle to angle?

What is the treatment of localized sarcomatous growths of the upper jaw?

What should be the character of the operation where the antrum is involved?

Whose incision for the removal of the upper jaw leaves the least deformity?

Describe the operation for removing the upper jaw.

How is the wound treated after the removal of the upper jaw?

How long may the packing be allowed to remain?

What is to be the daily treatment of the wound?

Describe Dieffenbach's operation for simultaneously removing both halves of the superior maxilla.

Describe the operation for the removal of portions of the superior maxillary bones below the infra-orbital foramen.

Can small tumors be safely removed through the mouth?

CHAPTER LX.

ODONTOMATA.

Definition.—Odontome (from the Greek *οδόντος*, tooth, and *ομα*, tumor).

An odontome is a tooth-tumor; a tumor composed of dental tissues in varying degrees of development and in various proportions.

Tumors arising from tooth-germs are more common in the lower animals than in man. The animals in which they are most frequently found are the goat, sheep, bear, lion, marmot, agouti, porcupine, kangaroo, horse, and elephant.

The odontomata are growths belonging to the composite group of tumors. They are composed of tissues which arise from abnormal conditions of the enamel-organs from the capsule, from the papilla, or from the entire germ. Fig. 340 shows the structures of a developing tooth-germ. According to Sutton, the tumors which arise from the tooth-germ in part or in whole are,—

1st. Those which develop from the enamel-organ: Epithelial odontomes.

2d. Those which develop from the capsule: Fibrous odontomes, cementomes, compound follicular odontomes.

3d. Those which develop from the papilla: Radicular odontomes.

4th. Those which are formed from the entire germ: Composite odontomes.

From the foregoing classification it will be noticed that Sutton classifies the odontomata according to the *particular region from which these tumors have their origin.*

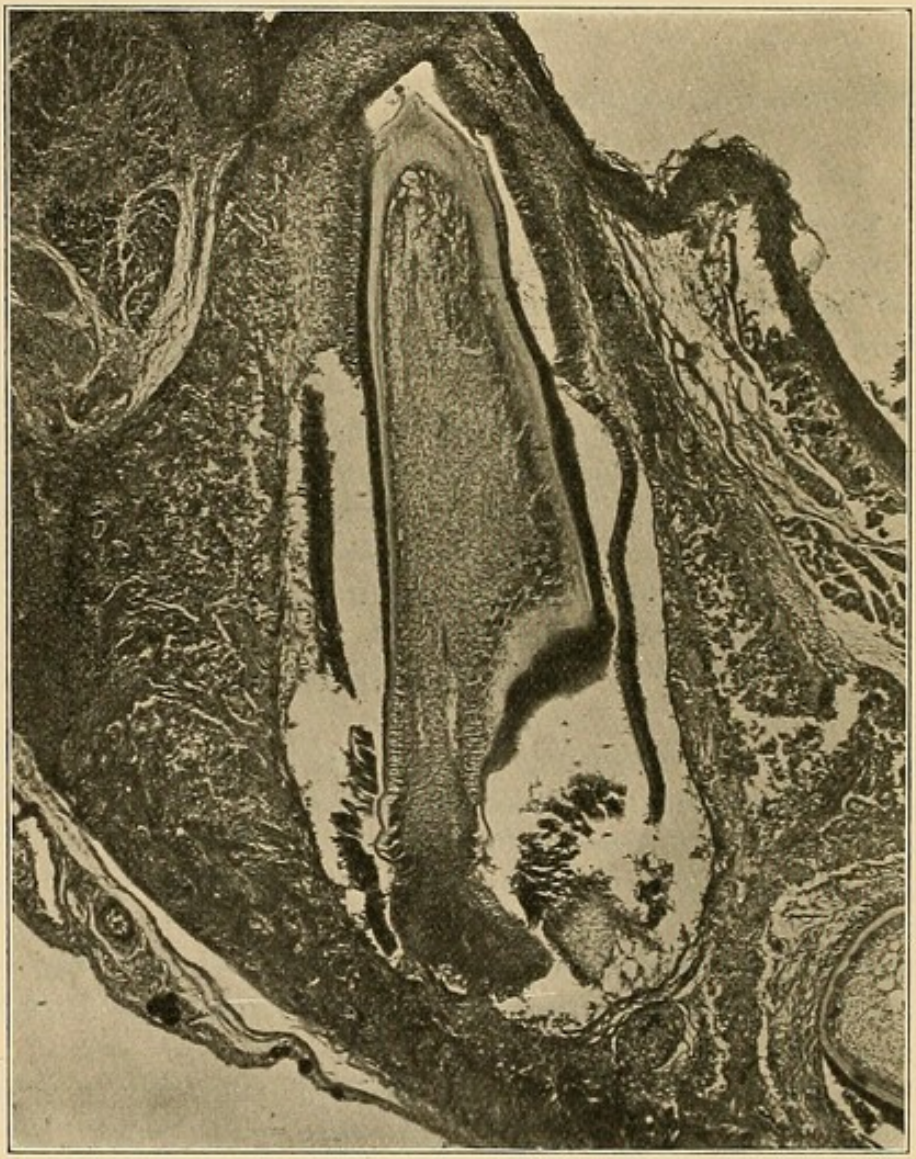
Broca, in his "Treatise on Tumors," classified the odontomata according to the *period of their origin,* and divided them into four classes, viz: Odontomes embryoplastiques, odontomes odontoplastiques, odontomes coronaires, odontomes radiculaires.

So far as the origin, development, and pathology of the odontomata are concerned, there is no important difference in the views held by Broca and Sutton. The teaching of Broca upon this topic may be briefly stated as follows:

1st. *Odontomes embryoplastiques.* These are tumors which are supposed to arise from the *membrana eboris* before the development of

the odontoblastic layer of the dentin papilla. Their development takes place at a period when there has not yet occurred any distinctive differentiation of cells peculiar to tooth-structure. They are derived from embryonic tissue, and contain no elements which would mark them as being of dental origin. Their structure is fibrous or fibroplastic, and does not differ from tumors of this character found in other locations of the body.

FIG. 340.



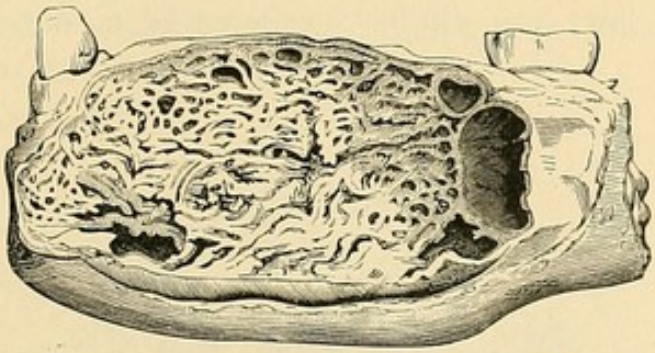
DEVELOPING TOOTH. × 75.

2d. *Odontomes odontoplastiques.* This form of odontome arises from the layer of odontoblastic cells before the dentin has begun to develop. The papilla takes on an irregular growth, and the tissue so formed, by reason of the presence of the odontoblasts, finally becomes calcified, presenting characteristics of structure more or less like true dentin; or it may present certain elements of enamel commingled with

them; or it may assume the form of a mass of heterogeneous dental tissues in no way resembling a tooth in external outline. Sometimes, as a result of the destruction of the odontoblasts, the tissue may resemble an imperfect bone formation.

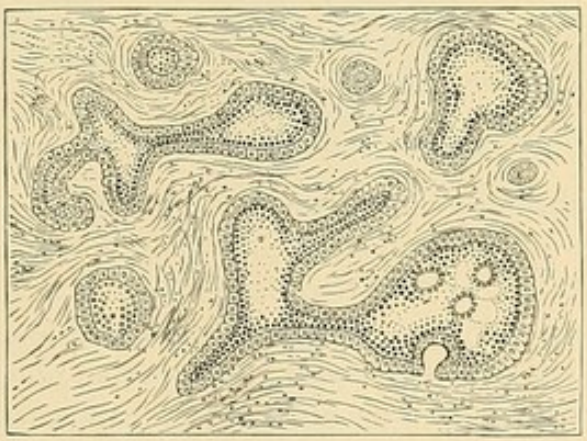
3d. *Odontomes coronaires.* This variety of odontome develops during the formation of the tooth, after a cap of dentin has been formed over the dentinal papilla, and is the result of a localized hyperplasia. The tumor is confined to the crown of the tooth, and no matter how

FIG. 341.



EPITHELIAL ODONTOME.—NATURAL SIZE. (After Sutton.)

FIG. 342.



MICROSCOPICAL CHARACTER OF AN EPITHELIAL ODONTOME. (After Sutton.)

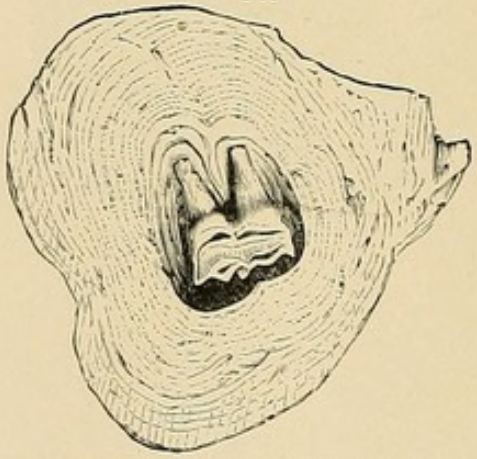
much the tumor may be changed in form by subsequent hyperplasia, the mass still bears a clear resemblance to normal tooth-structure.

4th. *Odontomes radiculaires.* The odontomes of this class are developed during the formation of the root of the tooth, after the crown has been completed, and are the result of a general hyperplasia of the dentinal papilla occurring at this period. These tumors are confined to the roots of the affected tooth, which they may envelop. The character of the tissue in the cases which have been examined is dentin and cementum.

This classification of Broca has been almost universally adopted

by writers upon this subject ever since its publication; but the recent classification of Sutton is, in the opinion of the writer, the more accurate, and gives a better foundation for a correct understanding of the origin, development, and pathology of these growths, and will therefore be followed in this work. The writer, however, takes the liberty of placing the *epithelial odontomes* (Fig. 341) and the *follicular odontomes*, on account of their character, under the head of cysts and of discussing the former with the multilocular cysts (Fig. 342), and the latter in a separate chapter under the head of Dentigerous Cysts. The other forms of odontomes, as classified by Sutton, are productive of *solid tumors* of the jaws, and will be considered in the order arranged by this author.

FIG. 343.



FIBROUS ODONTOME FROM A GOAT.—NATURAL SIZE. (After Sutton.)

Fibrous Odontomes.—Odontomes of this form are the result of excessive growth of the fibrous capsule which invests the tooth-germ. This capsule, or sac, is derived from the connective tissue at the base of the dentin papilla, and invests the crown of the developing tooth. The capsule proper consists of two layers: An external one, consisting of a dense fibrous structure, and an internal one composed of a soft, gelatinous substance, rich in connective-tissue corpuscles, and containing some true fasciculi of connective tissue. (Kölliker.)

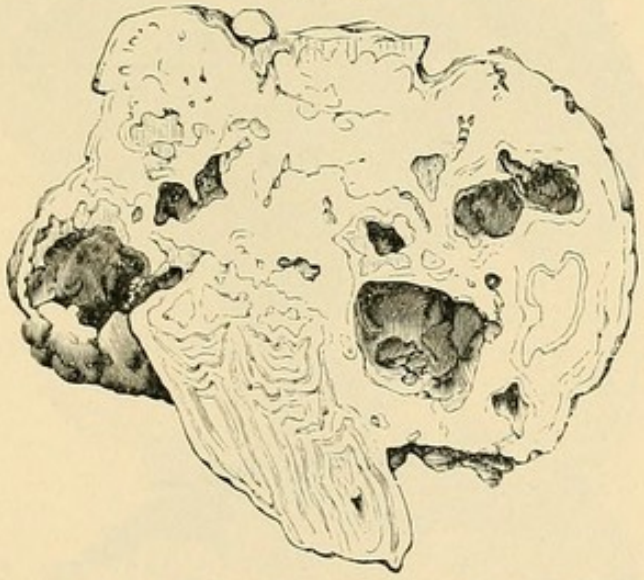
The tissues composing the capsule or dental sac, under certain conditions, become very much thickened and consolidated, and constitute, with the inclosed tooth, a tumor designated as a *fibrous odontome*.

The conditions which bring about this thickening and consolidation of the dental capsule are not very well understood, but Sutton is of the opinion that rickets plays an important part in the causation of the disease, from the fact that in rickety children those membranes are most affected which are engaged in the production of bone. Tumors of this character are frequently mistaken for fibromata. The error is

most likely to occur in those cases in which the tooth is small or imperfectly developed. Microscopically the fibrous elements are found to be arranged in laminæ, with strata of calcareous material. (Sutton.) (Fig. 343.) These tumors are not confined to the human species, but are quite common in certain of the lower animals, principally in the mammalia. The ruminants are most frequently affected with the disease, particularly goats.

Heath removed two tumors of this character from a boy seven and one-half years old, which developed symmetrically in the angles of the lower jaw. These tumors were covered by a thin layer of compact bone-tissue. The boy had suffered from rickets, but at the time of the operation was well nourished.

FIG. 344.

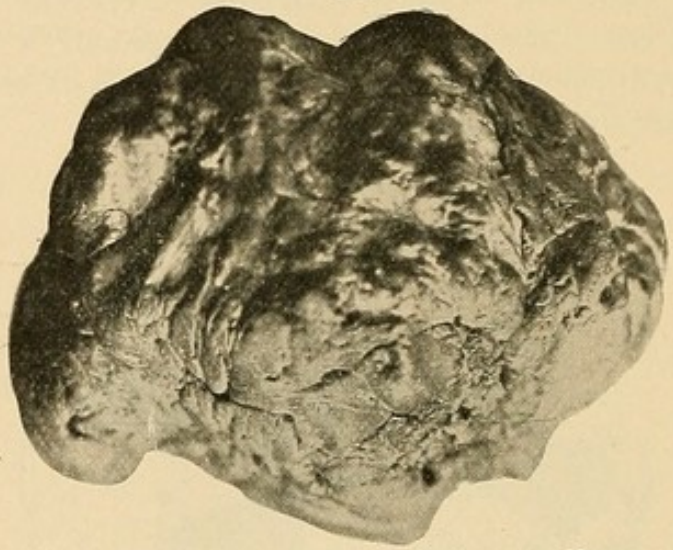


CEMENTOME FROM A HORSE.—HALF NATURAL SIZE. (After Sutton.)

Cementomes.—Cementomes are the result of calcification of the thickened and enlarged tooth-capsule, which eventually leaves the tooth imbedded in a mass of ossific matter. Odontomes of this character are most frequently seen in the horse. Occasionally they are seen in the human subject. The tissue of which the tumor is composed is apparently a modified cementum. It is arranged in laminæ similar to the laminated structure of the fibrous odontome. They sometimes attain a very large size. Tomes described an odontome of this character removed from the jaw of a horse which weighed ten ounces. Sutton has given an account of another which weighed twenty-five ounces. In this mass of tissue there could be observed the outlines of three teeth imbedded in the cementum. (Fig. 344.) The largest cementome ever recorded up to the present time weighed seventy ounces; this specimen is preserved in the Royal Veterinary College, London.

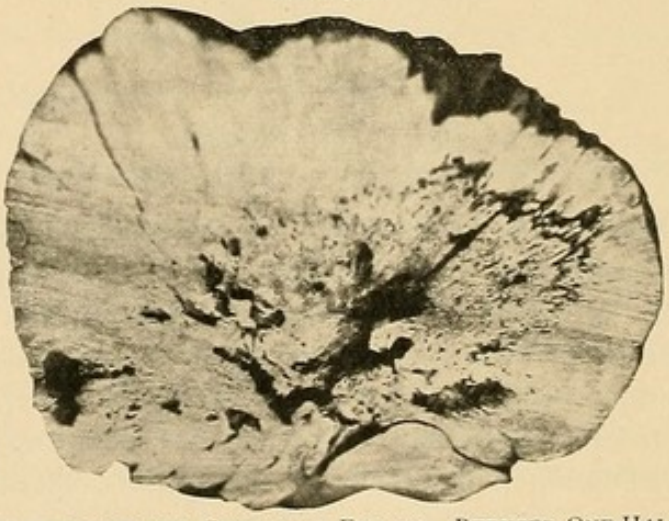
Through the kindness of Professor Sayer, of the Chicago Veterinary College, the writer has been permitted to examine and photograph an enormous cementome taken from a female colt two years of age, *post-mortem,* which weighed in the moist state ninety-six ounces, and in its present dried state fifty-nine ounces. It measures through its longest diameter five and three-fourths inches; through its shortest diameter, four and one-fourth inches; its largest circumference is six-

FIG. 345.



CEMENTOME FROM YOUNG HORSE.—REDUCED ONE-HALF NATURAL SIZE.

FIG. 346.



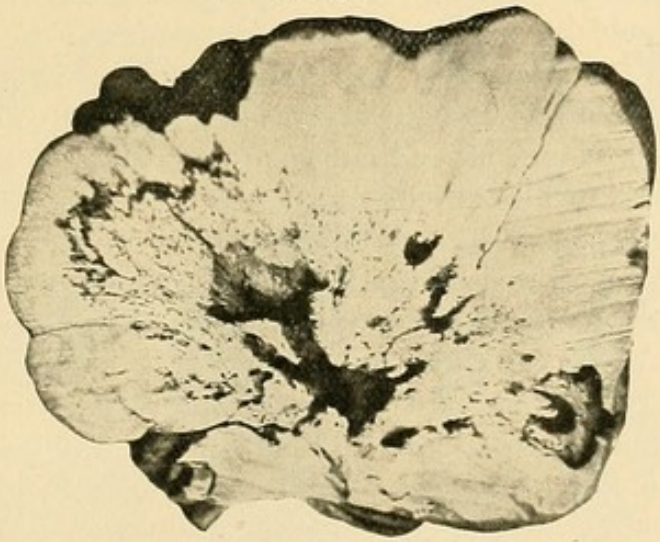
VERTICAL SECTION THROUGH THE CENTER OF FIG. 345. REDUCED ONE-HALF NATURAL SIZE.

teen and one-fourth inches, and its smallest circumference fourteen and three-fourths inches. The tumor was located in the right superior maxilla, and was first discovered when the animal was about five months old. It grew very rapidly, producing extensive inflammation of the jaw, with abscess, which constantly discharged through several sinuses. Death resulted from inability to take food.

The growth upon its external surface is lobulated, the base being

made up of numerous irregular lobes. (Fig. 345.) On section through its longest diameter, which intersected its base, it appeared to be a homogeneous mass of cementum, containing an irregular cavity in its center, surrounded by an area of spongy-looking tissue, similar to the cancellated tissue of bone. (Fig. 346.) This cavity has the same appearance as the pus-cavities sometimes found in the tusks of elephants. On closer inspection a distinct laminated structure can be seen at the periphery of the tumor, which appears to be as dense as ivory. After the most critical inspection it was not possible to discover any evidence of a tooth or even a suggestion of tooth-formation. At the base of the tumor there is a large depression or excavation, which has the appearance of having been the resting-place of a molar tooth. (Fig. 347.)

FIG. 347.



VERTICAL SECTION THROUGH THE CENTER OF FIG. 345, OPPOSITE HALF.—REDUCED ONE-HALF NATURAL SIZE.

Compound Follicular Odontomes.—This form of odontome is the result, according to Sutton, of a "sporadic calcification of the thickened and enlarged tooth-capsule," while in the formation of cementomes the process of calcification takes place *en masse*. By this sporadic calcification of the capsule there is developed within the tumor "a number of small teeth or denticles, consisting of cementum or dentin, or even ill-shaped teeth composed of the three dental elements —cementum, dentin, and enamel." (Sutton.)

The writer is inclined to the opinion that the explanation of the presence of *nests* of teeth or denticles in such tumors is that the epithelial elements confined within the tooth-capsule break up, as is the case with the epithelial cord, into minute masses, which under favorable conditions assume shapes similar to the normal teeth, but smaller in size, or ill-shaped teeth, or masses of tissue of heterogeneous structure which have no definite outline.

These tumors have been observed in the human subject, and in goats, horses, and other mammalia. In man they usually develop in early life, during the period of second dentition. Among the records of cases of this character, the youngest was a boy ten years old and the eldest a woman of twenty-seven years.

The number of denticles found in tumors of this character varies greatly. From three or four to as many hundreds have been removed from the jaws of a single patient.

Among the recorded cases of special interest which will be briefly mentioned are the following:

Tellander reported a case of tumor of the right side of the superior maxilla in a woman twenty-seven years of age, which had been growing since she was twelve years old. The character of this growth was a hard, painless swelling. It occupied the space in which should have been located the first molar, the bicuspids, and the cuspid of the permanent set. These teeth had not erupted. Upon opening the tumor it was found to contain several minute teeth; nine individual teeth with conical crowns and conical roots, each complete in itself, the crown being furnished with enamel; and six masses of dental tissues having the appearance of being formed by a union of several single teeth, and all presenting the characteristics of supernumerary teeth. Another tooth made its appearance about a year later in the location from which the tumor was removed.

Another case, recorded by Windle and Humphreys, which occurred in the practice of Sims, of Birmingham, England, was found in the mouth of a boy ten years of age. The lateral incisor and cuspid of the permanent teeth had not made their appearance, and this space was occupied by a tumor with hard, unyielding walls, from which were taken forty small, irregularly-formed denticles.

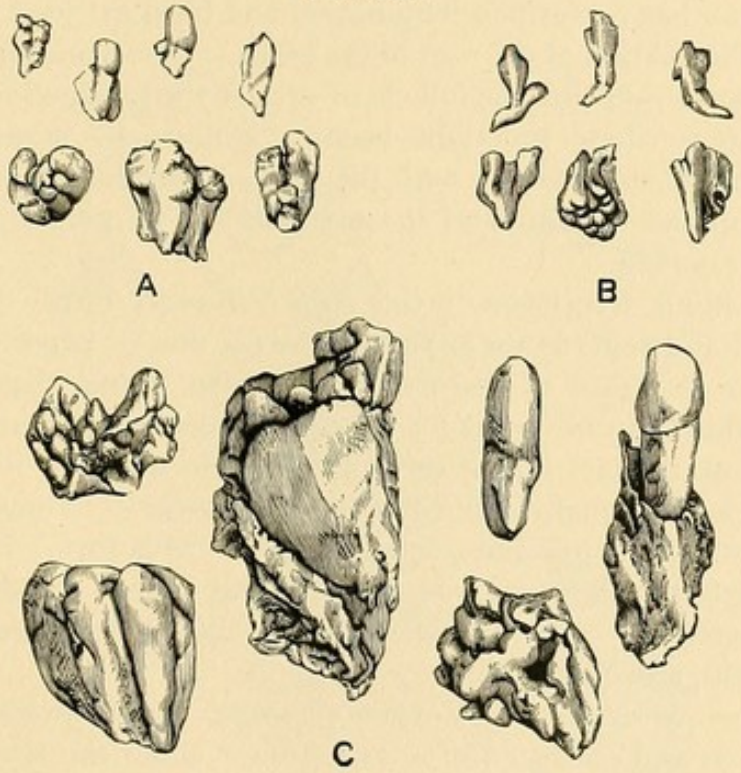
Tomes has recorded a case in a Hindoo, aged twenty, having a tumor of this character located in the front of the mouth, and for whom Mathias removed fifteen masses of ill-formed and united supernumerary teeth and bone. The deformity of the jaw rapidly disappeared after the removal of the supernumerary dental tissues. The only peculiarity that could be noticed later was the absence of the central and lateral incisors. (Fig. 348.)

The writer has recorded a case in the chapter on Dentigerous Cysts from which five small teeth or denticles were removed.

The most remarkable case of "abnormal development of the teeth forming tumors of the jaw" has been reported by O. Hildebrand, of Göttingen. "A child twelve years of age had suffered from enlargement and tumefaction of the jaws, resulting from an excessive development of teeth, she having submitted, in 1889, to several operations, and been relieved of between one hundred and fifty and two hundred

teeth of various sizes and forms. In July, 1891, the patient presented again for operation, the lower jaw being much thickened upon both sides, and also the right upper jaw. Seventeen teeth were found, part of them normally developed, others in an imperfectly developed condition and very irregular in position. From the upper and lower jaws several masses of teeth were again removed, and represented about one hundred and fifty teeth in conformation like those previously removed, making in all between three hundred and fifty and four hundred. Two round, glassy bodies were also found, each about the size of two peas, which were composed of tooth-structure, as revealed by

FIG. 348.



A, Denticles from Tellander's Case. Total number, twenty-eight.
B, Denticles from Sims's Case. Total number, forty.
C, Denticles from Mathias's Case. Total number, fifteen.
(After Sutton.)

the microscope. The writer of the report was of the opinion that tooth-formation would continue, in all probability, until the epithelial tissues of the mucous membrane covering the jaws had reached their final development." (Matas.)

Sutton describes a case of this character occurring in a Himalayan goat which had a tumor in each upper jaw. "The interior of each tumor was occupied with teeth, denticles, and fragments of cementum, varying in size, numbering in all three hundred. Many were firmly imbedded in the fibrous walls of the tumor, whilst those which were free in the sac had become loosened by suppuration."

Logan has recorded a similar case occurring in the maxilla of a

horse, which contained four hundred small, ill-formed teeth or denticles.

Radicular Odontomes.—These tumors are located upon the roots of the teeth, and are due to excessive growth of the formative pulp after the development of the crown has been completed, and while the root is in process of formation. They appear in the form of enlargements upon the root, which they may completely imbed, and sometimes reach a size many times larger than the tooth itself. The growth causes little change in the character of the tooth or of its roots.

The tissues which make up the tumor are dentin and cementum; enamel does not enter into its formation, for the reason that the enamel-organ has performed its function and been calcified some time prior to the formation of the root of the tooth. The principal functions of the formative pulp and the follicle or sac are the production of dentin and cementum; these functions become abnormally stimulated, for some reason not understood, with the result of producing an enlarged or hypertrophied condition of these tissues. The pulp is large and sometimes calcified.

Salter, in his description of this class of tumors, terms them *hernia of the fang*, and regards them as outgrowths due to hypertrophy and dilatation of the root of the tooth. He also thinks that the term applied to this form of tumor by Broca, *odontome radiculaire*, is objectionable, from the fact that it carries with it no meaning beyond that of location, and is equally applicable to other tumors. They have been found in both the upper and the lower jaws. This form of odontome is exceedingly rare in the human subject, only a very few ever having been removed, the principal ones being those of Forget, Tomes, Heider, Wedl, and Salter.

The one described by Forget occurred in the practice of M. Maisonneuve, and consisted of a large tumor about the size of a bantam's egg, which was attached to the dental surface of a left lower molar tooth, involving the neck and a large part of the root. The tumor caused considerable disfigurement of the face. In an effort to extract the tooth the tumor came away attached to it. The patient was a man forty years of age.

The second example was described by Tomes, the specimen, which is the largest ever seen in the human subject, having been presented by Hare. It was removed from the upper jaw of a man forty-one years of age. The tumor had been growing for several years, and finally caused suppuration, with perforation of the cheek. The growth is a large, lobulated mass connected with the roots of an upper molar tooth, and four or five times as large as the tooth itself. (Fig. 349.)

Heider and Wedl briefly mentioned a case in many respects like the one just described. In this case the second molar tooth of the

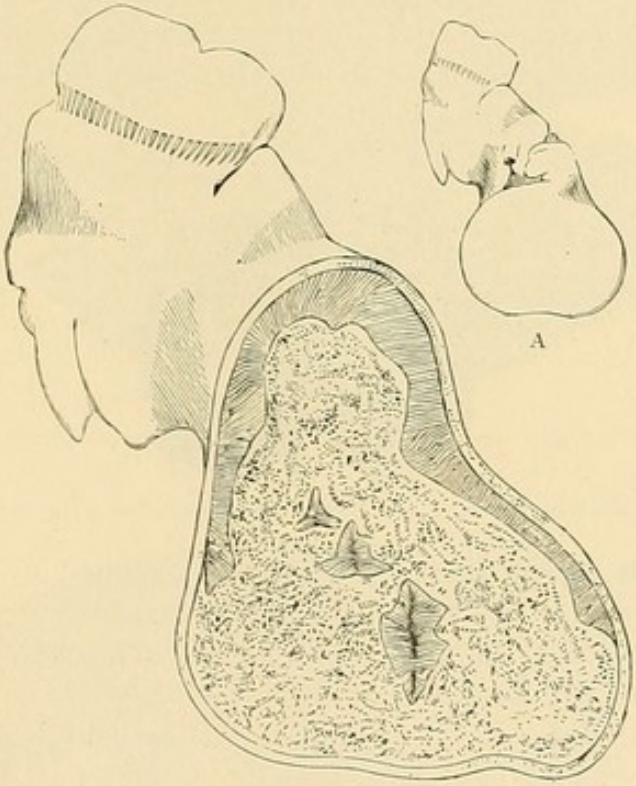
right side had developed into a large, irregular mass, which held down the third molar. The fourth example occurred in the practice of

FIG. 349.



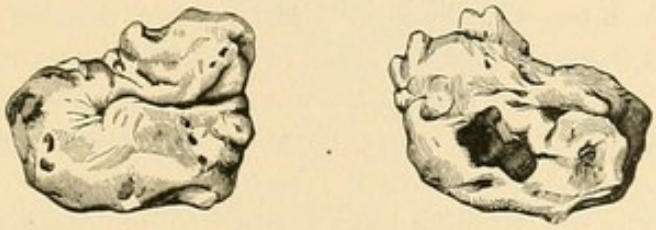
RADICULAR ODONTOME.—NATURAL SIZE. (After John Tomes.)

FIG. 350.



RADICULAR ODONTOME FROM HUMAN SUBJECT.—A, NATURAL SIZE OF THE SPECIMEN. (After Salter)

FIG. 351.



RADICULAR CEMENTOME FROM MAN AGED TWENTY-FIVE YEARS. (After Sutton.)

Heath, and formed the subject of a classic paper by Salter. This specimen consisted of a rather small lower molar tooth, to the distal root

of which is attached a large, lobulated tumor more than twice the size of the tooth. (Fig. 350.) The outer layer of this mass is composed of cementum; within this is a stratum of dentin, and the center is occupied by a calcified tooth-pulp, the latter being formed of a "confused mass of bone-structure and dentin-structure surrounding a vascular net-work of the same character of the dentinal pulp." (Heath.)

Windle and Humphreys have described a case obtained from a man twenty-five years of age. The odontome was situated in the lower jaw, in the region of the right second molar tooth. (Fig. 351.)

These tumors are much more frequent in the lower animals than in man. In the other mammalia they are often multiple. Rodents are especially liable to them, on account, no doubt, of the fact that their teeth grow from persistent pulps. (Sutton.) They have been found in marmots, porcupines, and agoutis. The largest odontomes have been found associated with the roots of the tusks of elephants.

FIG. 352.



COMPOSITE ODONTOME FROM YOUNG STEER —REDUCED ONE-HALF NATURAL SIZE.

Composite Odontomes.—This term is applied by Sutton to all those hard tooth-tumors which lack the form of teeth, but are found in the jaws, and consist of a heterogeneous mass of enamel, dentin, and cementum. This form of odontome is the result of an abnormal growth of all the elements which compose the dental germ,—enamel-organ, formative pulp, and sac or follicle.

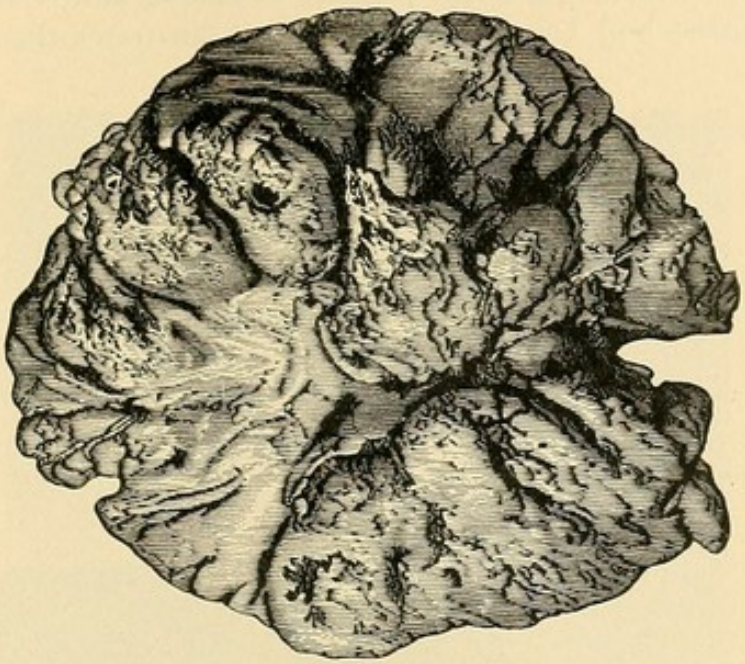
Salter applies the term *"warty teeth"* to this form of odontome. Broca designates them as *odontomes coronaires.*

These tumors are often found to be composed of two or more tooth-germs fused together in an indiscriminate manner. They can be differentiated from the cementomes in which two or more teeth are united together by the fact that in the composite odontome the various tissues form a heterogeneous mass, while in the cementomes the form of each tooth can be readily defined. (Sutton.)

The composite odontomata have seemed to be confined to man, as no cases up to the present time have been recorded as occurring in the lower animals. Fig. 352 is a composite odontome removed from the jaw of a young steer which had died from extensive suppuration and

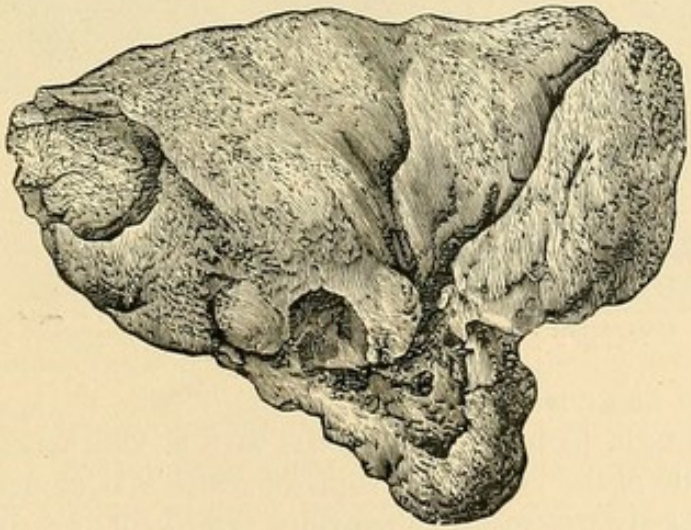
necrosis of the lower jaw. The disease was found to have been caused by the presence of this growth, which appears to be a fusion of two molar teeth making an irregular mass of dental tissues. They are found in the human subject in both the upper and lower maxillæ; the majority of the reported cases, however, have been located in the lower

FIG. 353.



ODONTOME FROM THE SUPERIOR MAXILLA.—NATURAL SIZE. (Michon, after Sutton.)

FIG. 354.



COMPOSITE ODONTOME FROM THE SUPERIOR MAXILLA.—NATURAL SIZE. (Duka, after Sutton.)

jaw. Sutton is of the opinion that many cases of reported exostosis of the antrum were really composite odontomes, and cites the case of M. Michon, who removed a large tumor from the antrum of a young man aged nineteen years, which was described as an exostosis, but which in reality was a composite odontome, as proved by the report which

accompanied it of its microscopical structure. (Fig. 353.) This consisted of "tissue presenting many parallel tubules having the appearance of exaggerated dentinal tubes." Duka reported a similar case occurring in a Mahomedan woman twenty-six years of age, in which the tumor was located in the right antrum. (Fig. 354.)

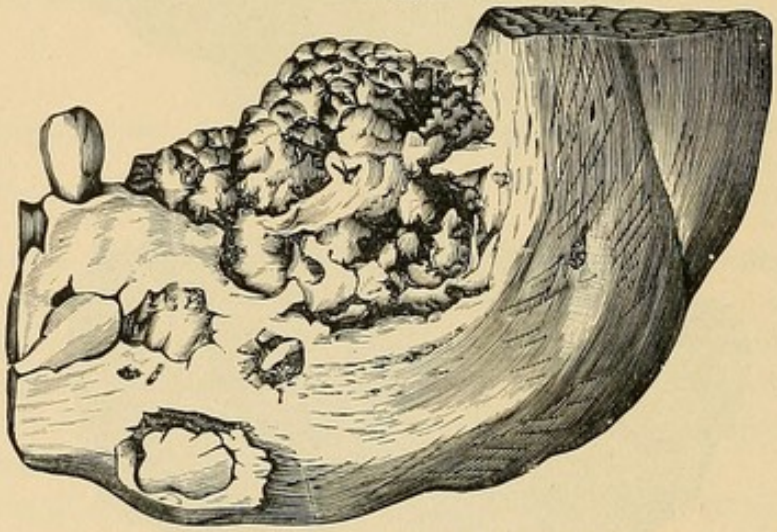
The diagnosis of these tumors is often a difficult matter. The following case, occurring in the practice of Heath, and related by him in his "Injuries and Diseases of the Jaws," illustrates the difficulties

FIG. 355.



COMPOSITE ODONTOME FROM YOUNG LADY AGED EIGHTEEN. (After Heath.)

FIG. 356.



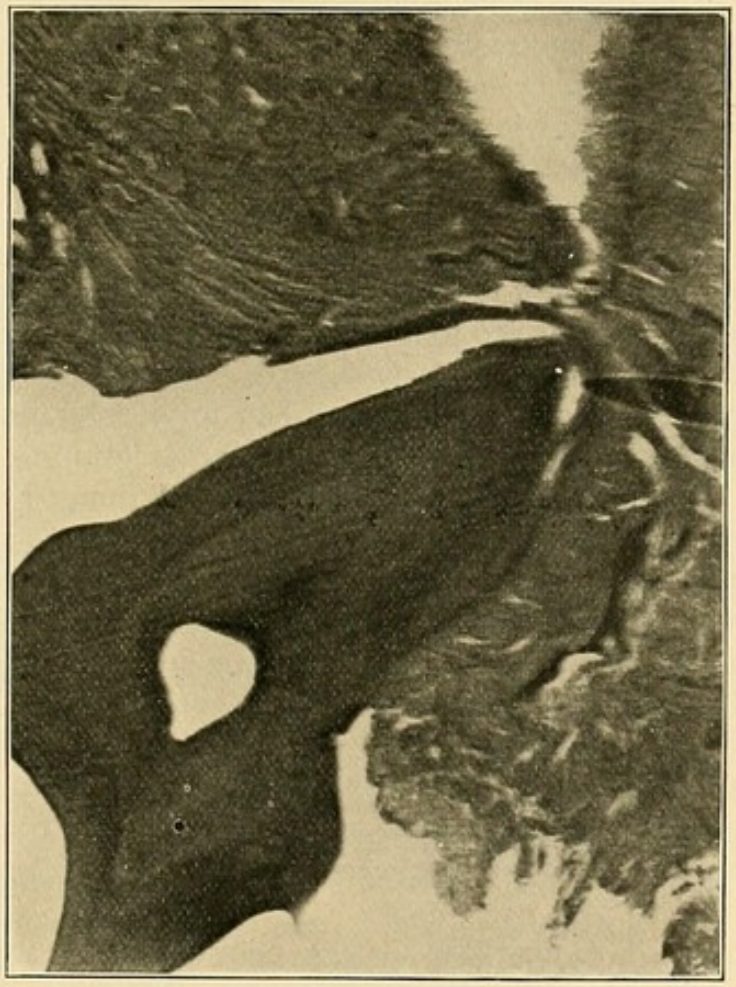
COMPOSITE ODONTOME.—NATURAL SIZE. (After Forget.)

experienced in diagnosing the odontomata. "Miss C., aged eighteen years, was brought to me in July, 1881, with a considerable swelling of the right side of the lower jaw, some of which was evidently inflammatory, and partly the result of previous treatment; but there was, I thought, sufficient evidence of expansion of the jaw to warrant the opinion that a tumor was present, and I therefore recommended the removal of a portion of the jaw. Suppuration was then present, and with the finger a rough surface of apparently exposed bone could be felt, but this I regarded as the result of inflammatory action excited by the injudicious irritation of a periosteal growth, since partial ne-

crosis of a jaw involved by cartilaginous or malignant growths, which have been irritated by exploratory measures, is in my experience by no means common. The patient had the advantage of the opinion of Sir James Paget, who was not perfectly satisfied as to the existence of a tumor, and expressed a hope that the case might prove to be one of necrosis. Under these circumstances the operation was postponed.

"On my return to town in September I found the patient improved

FIG. 357.



COMPOSITE ODOMTONE—TRANSVERSE SECTION—FROM A FULL-TERM HUMAN FETUS.—MAGNIFIED.

in health, and the swelling diminished by the subsidence of the inflammation, but a considerable enlargement of the lower jaw was still present, with a sinus opening externally. From the mouth a white mass was visible, which, appearing among granulations, looked like necrosis, and I agreed that an attempt should be made to remove this, although I could not think it accounted for the expansion of the jaw. Under chloroform I proceeded to examine the mouth with my finger. I soon found that the white mass was not bone, but tooth, and yet was unable to make out its outline. I was unable to make any impression

with a chisel or gouge, but at last with an elevator succeeded in lifting out of its bed a mass of dental structures, which measured one and one-half inches antero-posteriorly, one inch transversely, and one and one-fourth inches from above downwards. It weighed three hundred and fifteen grains." (Fig. 355.) The specimen was placed in the hands of C. Tomes for microscopic and histologic examination, and was found to be composed of a conglomerate mass of enamel, dentin, and osteo-dentin.

The most remarkable example of a composite odontome is the classic case of Forget. This occurred in the person of a young man aged twenty years, who presented himself, in 1855, with disease of the lower jaw, from which he had suffered since he was five years of age. The tumor consisted of a large, round, smooth, hard, unyielding swelling, which occupied nearly the whole of the left side of the lower jaw. The teeth posterior to the first bicuspid were absent. The tumor was removed by dividing the jaw in front of the first bicuspid and through the ascending ramus on a line with the inferior dental foramen. The tumor when removed proved to be a hard oval mass the size of an egg, having an uneven surface, in places covered with small tubules, which were coated with enamel. The tumor had formed for itself a large cavity in the jaw, which extended from the first bicuspid to the ramus. (Fig. 356.) The microscopic examination of the tumor showed it to be composed mainly of dentin, in places covered with enamel which dipped down in the crevices, while in the bottom of the crevices cementum was found. The origin of the tumor was the fusion and hypertrophy of the last two molars. Heath gives a description of six other cases of this form of odontome which he has found recorded. Fig. 357 is a microscopic section of an odontome found in the jaws of a full-term human fetus which was otherwise malformed. The case is interesting from the fact that it must be an exceedingly rare condition in a child at birth, as the writer has been unable to find a like case upon record.

Diagnosis.—The diagnosis of the odontomata is sometimes a matter of considerable difficulty, for the reason that the symptoms are usually very obscure, and those which are prominent are often leading symptoms in other affections, as, for instance, in the *fibrous odontomes*, which may be readily mistaken for myeloid sarcoma; or the cementomes or composite odontomes may be taken for exostosis, and in others, when suppuration is present, for necrosed bone.

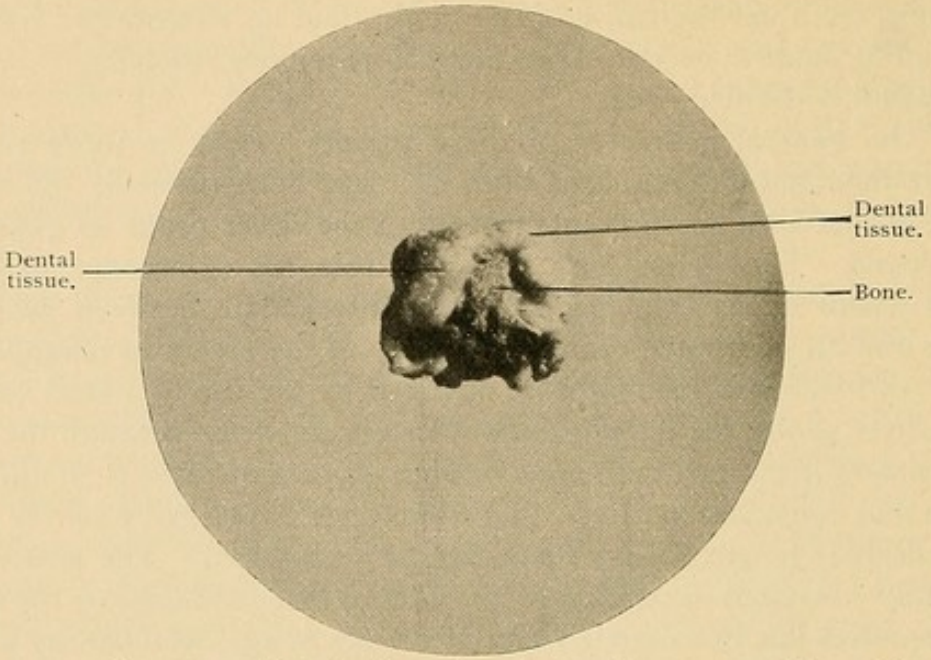
Sutton says, "It is a curious fact that up to this date there is no instance on record in which an odontome, other than a follicular cyst, has been diagnosed before operation." In all doubtful cases of tumors of the jaws, occurring in youths and young adults, the absence in the location of the tumor of teeth, which are commonly erupted before the

age at which the tumor began to grow, should excite suspicion that the missing teeth are the cause of the tumor, and an exploratory incision should be made to ascertain this fact before a more extended or serious operation is decided upon.

The general character of these tumors might be more readily ascertained and differentiated from necrosed bone-tissue by the use of steel probes with sharp points instead of the silver probe so generally employed. The writer feels positive, from his experience in other lines where it has been necessary to differentiate between necrosed bone and an unerupted tooth, that many of the errors in diagnosis in cases of this character might be obviated by the use of a steel instead of a silver probe, for the sensation which is conveyed through the steel instrument in contact with bone or with tooth-structures is so different from that conveyed by silver, that when once experienced by the careful observer it will always thereafter be recognized. The writer was recently consulted in relation to an obscure case of disease of the angle of the lower jaw in a married lady fifty years of age, who had up to one year ago enjoyed uninterrupted good health. The disease began by a painful swelling and enlargement of the left side of the lower jaw in the region of the third molar tooth. This tooth, which was sound, became very sore and loose, and was extracted by the family dentist upon the supposition that the disease was alveolar abscess. On removing the tooth a considerable discharge of pus took place. The swelling and enlargement of the jaw steadily progressed, from this time, with pain and a constant discharge of pus. The second molar had sometime afterward been removed and the cavity curetted, and this was followed by the exfoliation of several small pieces of necrosed bone. She next consulted an eminent surgeon, who thought it a case of necrosis, and operated twice by cutting into the jaw through an external incision and removing considerable portions of the cancellated structure of the bone. Six weeks afterward the discharge had not abated.

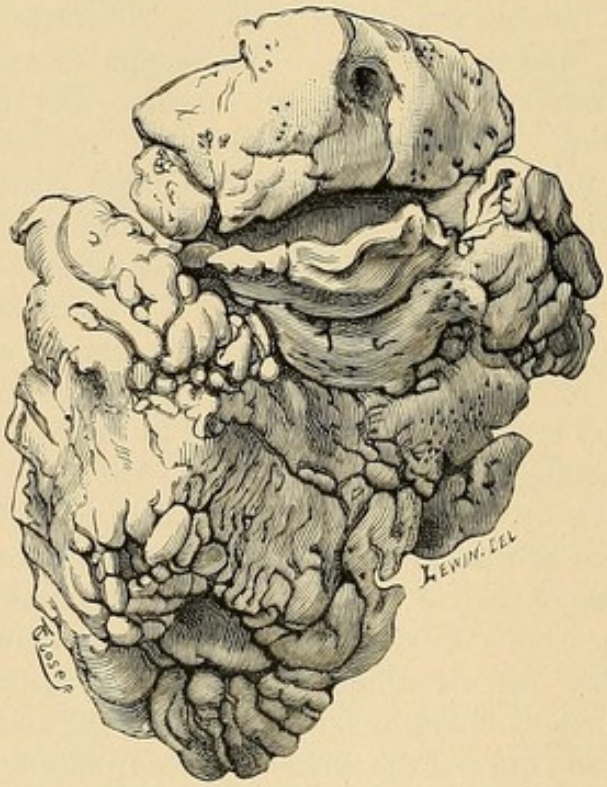
The conditions at the time of the consultation were as follows: An opening one and one-half inches in length existed in the soft tissues and the jaw, just in front of the angle and at the lower border of the bone. This opening went entirely through the bone and communicated with the cavity of the mouth at the alveolar border, which was greatly thickened at this location. Careful exploration of the wound cavity in the body of the jaw, with a sharp-pointed steel probe, revealed the presence of a body more dense than the surrounding bone, and located well under the ascending ramus. Upon further examination certain dense surfaces could be felt which gave the assurance of being dental tissue. So sure did the writer feel that there could be no mistake as to the presence of such a body in this location, that he made an unqualified diagnosis of an odontome and advised an immediate opera-

FIG. 358.



COMPOSITE ODONTOME.—NATURAL SIZE.—FROM LOWER MAXILLA OF A WOMAN FIFTY YEARS OF AGE.

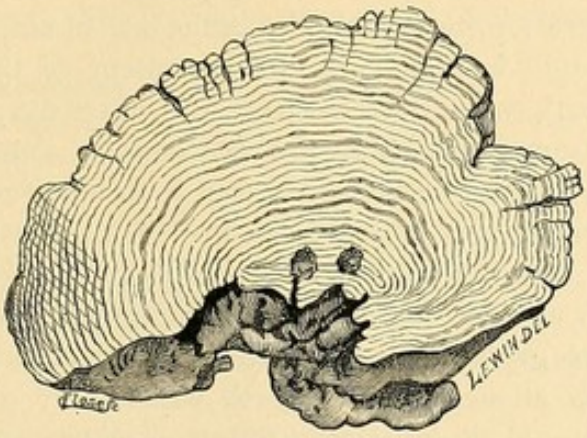
FIG. 359.



LARGE ODONTOME SPONTANEOUSLY SHED FROM THE ANTRUM; WEIGHT NEARLY FIFTEEN OUNCES. (Hilton, after Sutton.)

tion. The operation consisted of cutting away the bone from around the tumor with gouge, chisel, and mallet—this was done through the mouth—and the tumor then lifted from its bed in the jaw by means of an elevator and the external wound closed. Fig. 358 is a photograph of the odontome, natural size. The character of the growth would class it as a composite odontome. Rapid improvement took place after the operation, and at the end of six weeks the patient was discharged cured.

FIG. 360.



SECTION OF THE ODONTOME FIG. 359, SHOWING THE CONCENTRIC LAMINATION. (Hilton, after Sutton.)

FIG. 361.



ODONTOME.—NATURAL SIZE. (Parker, after Sutton.)

Prognosis.—The prognosis of odontomata is favorable. Operation is always required for their removal, though Heath mentions a case recorded by Harrison in which the odontome was spontaneously expelled. Sutton mentions two cases; the first is recorded by Hilton in Guy's Hospital Reports. This was the largest odontome that has been known to form in the human antrum. It weighed nearly fifteen ounces. (Fig. 359.) The patient was thirty-six years old. The disease was first noticed thirteen years before: the cheek swelled, the eye was displaced from the orbit and finally burst. The cheek sloughed away later, leaving the growth exposed. Suppuration was profuse, accompanied by necrosis of the bone. The tumor finally dropped out,

leaving an enormous hole in his face. Section of the odontome showed it to be a laminated structure. (Fig. 360.)

The second case is recorded by Parker as occurring in a young lady aged nineteen years. The growth was attached to the left second molar. In an effort to extract the tooth it broke, leaving the tumor behind. Subsequent attempts were made to extract it, but without success. Two years later it was spontaneously expelled from the jaw. (Fig. 361.)

Treatment.—The removal of the various forms of odontomata as a rule does not require an extensive operation. If they can be diagnosed before operation, the matter becomes one of simple enucleation. Every effort should be made, in the treatment of these tumors, to extract the growth without removing any extensive portion of the bone. This in some cases may not be possible, and a considerable portion of the bone must be sacrificed; but it would seem, from a study of the reported cases, that in every one in which a portion of the body of the bone had been removed with the tumor, this serious operation might have been avoided but for the difficulties of diagnosis. Heath says, "Where the growth is presumably connected with a tooth, the rule of removing all neighboring teeth which may possibly be connected with it should be invariably followed before any more serious operation is undertaken."

After the enucleation of the tumor, the cavity may be packed for a few days with boric acid gauze, and allowed to close by granulation.

REVIEW.

CHAPTER LX.

What is an odontome?
In what class of animals are these tumors common?
In which of the lower animals are they most frequently found?
To which group of tumors do they belong?
Of what tissue are they composed?
Give Sutton's classification.
Upon what does Sutton base his classification?
How does Broca classify these tumors?
Give the classification of Broca.
Describe the odontomes embryoplastiques.
Describe the odontomes odontoplastiques.
Describe the odontomes coronaires.
Describe the odontomes radiculaires.
What advantage has Sutton's classification over that of Broca?

How are the fibrous odontomes formed?
From what is the follicle or sac derived?
Describe the follicle and its layers.
What is the condition of the tissue of the sac in the fibrous odontomes?
What are the conditions which bring about these changes in the capsule?
For what are these tumors sometimes mistaken?
What is the microscopic appearance of these tumors?
In what other animals than man are these tumors frequently seen?
How are the cementomes developed?
In what animals are these tumors most frequently found?
What is the character of the tissue of which they are formed?
Describe the cementome examined by the writer.
What is Sutton's view as to the formation of the compound follicular odontomes?
What other explanation of their origin is offered?
In what animals have these tumors been observed?
At what period of life in man are they most frequently seen?
What were the limits of age as gathered from the reported cases?
What variations in the number of denticles have been found in these cases?
What is the location of the radicular odontomes?
From what are they formed?
How do these tumors appear?
What, if any, changes are caused in the tooth or roots by the presence of these growths?
What kinds of tissue make up the tumor?
How is the absence of enamel accounted for?
What term does Sutton apply to this form of tumor?
Where are they found?
Are they rare or common in the human subject?
In what animals are they sometimes multiple?
What animals are especially liable to them?
In what animals are the largest specimens found?
To what form of tumors is the term composite odontome applied?
From what are these tumors formed?
What terms do Sutton and Broca apply to these growths?
Of what are these odontomes sometimes formed?
How may they be differentiated from cementomes in which two or more teeth are united?
Are the composite odontomes found in the lower animals?
In which jaw are they most commonly found?
What is Salter's opinion as to the character of the supposed exostosis of the maxillary sinus?
Are the odontomata difficult to diagnose?
What is the character of the symptoms?
What are the fibrous odontomes sometimes mistaken for?
What are the cementomes and composite odontomes sometimes mistaken for?
How may doubtful cases of tumors of the jaws be diagnosed?
How may the diagnosis be assured?
What form of probe is useful in diagnosing this form of tumor?
What is the prognosis?
Are these tumors ever spontaneously expelled?
What form of operation is usually required for their removal?

What should be the particular care of the surgeon in removing these growths?

Can all cases be treated in this manner?

What does a study of the operations performed in the reported cases teach?

What valuable suggestion does Heath make in regard to the extraction of neighboring teeth?

What is the after-treatment for enucleation of these growths?

CHAPTER LXI.

ODONTOMATA (Continued).

In the preceding chapter upon the Odontomata, those forms of odontomes were described which had their origin in some form of *aberration in development*. The maxillary bones, however, are occasionally the seat of tumors which arise from *aberration in the position* of the teeth. These growths are not included in the classification of either Broca or Sutton.

Irregularities in the position of the germs of the teeth are sometimes of such a nature as to prevent their eruption, and thereby cause the production of tumors.

Tumors arising from this cause have seldom been observed in connection with the deciduous teeth, for the reason that irregularities in either development or position are very rarely associated with these teeth. The period covered by second dentition is the age at which these tumors are most likely to be developed, the most active stage of this function being the time when they most frequently appear. But it is not at all uncommon for individuals past middle life to be the subjects of such growths.

These tumors are associated with teeth which by reason of their malposition in the jaws are unable to emerge from the crypts in which they have been developed. As a result, there is in certain cases a tendency, due to the irritation produced by the malposition of the tooth, to the formation of a tumor, sometimes of a cystic nature, as in dentigerous cysts, or a dense, osseous growth, which appears to be a thickening or hyperplasia of the overlying bone-tissue, resembling an osteoma with a tooth in its center. The teeth found in these tumors are in various stages of development.

The solid tumors of the jaws resulting from aberrations in the position or the development of the teeth are many times exceedingly difficult to correctly diagnose, their character often remaining concealed until after an operation has been made for their removal. Some of the very best surgeons have been misled in their diagnosis of these tumors, and have made extensive and needlessly severe operations under the belief that the growth was an osteoma or an osteo-sarcoma.

Sir William Fergusson made a mistake of this character in 1856,

in removing a large, dense tumor of the superior maxilla of a girl thirteen years of age. On dividing the tumor after its removal, it was found to contain a tooth imbedded in its center, and which was undoubtedly the cause of the growth.

The deviations in the position of the unerupted teeth which may cause solid tumors of this character may be classed under two heads: 1st, those contained within the alveolar process; 2d, those situated in other and sometimes remote portions of the maxillary bones.

Causes.—The teeth which are most liable to occupy malpositions in the jaws and cause the formation of tumors are the superior and inferior third molars, and the superior cuspids. Occasionally the superior first and second molars, the superior central and lateral incisors, and the bicuspids are misplaced in the jaws. The absence of a certain tooth from its position in the affected side of the jaw, and in which there is no history of its having been removed, should indicate the possibility or probability that the tumor was caused by the missing dental organ. The importance of this fact should not be overlooked in the diagnosis, for by its careful observance many otherwise obscure cases may be correctly diagnosed, and the disease cured by a simple operation.

Aberrations in Development and Position.—Aberrations in the form and position of the teeth are of interest, from the surgical standpoint, when by reason of their non-eruption they are productive of tumors of the maxillary bones. A few facts and suggestions upon some of the causes of the irregularities in the position of the teeth may not, however, be out of place in this connection.

Irregularities of the teeth are very common among civilized nations, much more so, seemingly, than existed a few generations ago, or than now exist among the aborigines, and nations which by their customs or isolated position geographically prevent immigration or the intermingling of the blood of other nations with their own.

In those races and nations of the highest civilization, as well as those which have received the greatest and most varied admixture of foreign blood—and in the present day these conditions may be considered almost the equivalent of each other—irregularities of the teeth are most frequently met with. The Anglo-Saxon race, and particularly the American branch of it, is a well-known illustration of this fact, as is generally recognized by the most eminent dental pathologists and by specialists in the department of orthodontia.

The effect of the intermarriage of distinct races and nations operates at first to lower the vitality and physical endurance, to break up the racial types in the conformation of the bones of the face, and the harmonious relationship which naturally exists between the size of the jaws and the teeth. Children often inherit the physical peculiari-

ties of one parent and the mental capabilities of the other, while in a general way it may be stated that the boys favor the mother and the girls the father.

A child may also resemble one parent in the size of the bony frame-work and the other in the dermal skeleton, as is frequently evidenced by the size of the body on the one hand, and upon the other in the color of the hair and eyes, the complexion, the size and characteristics of the teeth. Among animals the sire most frequently determines the size and general conformation of the body, but the color seems to follow no definite rule. Sometimes, when the colors of the parents are not the same, both colors are reproduced in the offspring, particularly in horses, cattle, and dogs, while in other instances the color of the paternal or maternal ancestor alone may be transmitted.

These conditions may be accounted for by the laws of hereditary transmission, operating through the differences in the embryonic origin and the morphological peculiarities of the various tissues. The skin and its appendages are developed from the external layer of the germinal disk of Pander, the *epiblast;* the teeth, mucous membrane, and its appendages from the internal layer, the *hypoblast;* while the bones, muscles, connective tissue, vital organs,—in fact, the great bulk of the body,—are developed from the middle layer, the *mesoblast*. The parent possessing the greatest vitality at the time of conception usually exerts a dominating influence over the development of the embryo, as well as the vital and physical peculiarities of the offspring. The same proposition holds true in relation to the mental endowment, while the color of the hair, eyes, complexion, and the character of the teeth are usually transmitted by the parent of lowest vital powers.

On the other hand, it is frequently noticed that the male offspring seem to possess an inherent tendency to reproduce the physical and mental peculiarities of the mother, while the female children inherit the same tendency in relation to the father. This tendency is often demonstrated in the peculiarities in the form, position, and suppression of the teeth. A somewhat remarkable illustration of an inherited suppression of the superior first bicuspids came under the notice of the writer in which the best of evidence was presented that this peculiarity had existed in one family for four generations.

The deformity was first noticed in a young lady nineteen years of age, the eldest of four children,—two girls and two boys. Neither of the brothers nor the younger sister had the deformity. The father of these children had never erupted these teeth, neither had his mother nor her father. The father of the young lady had one sister, but no brothers. The sister had a complete set of teeth. The father's mother was the only member of her family who had the deformity, and although it was commonly known in the family that her own father (the

great-grandfather of the young lady) had never erupted these teeth, she did not know whether any of her father's brothers or sisters had been so afflicted.

The various races of mankind are distinguished from one another principally by the color of the skin, the character of the hair, and the conformation of the features (this includes the conformation of the eyes, nose, lips, and jaws).

Nations are also, in a modified sense, recognized in the same way. The features, for instance, of the typical Irish, German, or English peasant are so marked as rarely to be mistaken one for the other. The conformation of the maxillary arches is also indicative, in a general way, of civilization and culture of races and nations. The more nearly the conformation of the typical maxillary arch of a race or a nation approaches the outline of the half of an ellipse cut through its short diameter, the higher the civilization.

The American nation is made up principally of the three nations just mentioned. The conformation of the jaws and the characteristics of the teeth in each are so different as to be readily distinguished. The intermarriage of these peoples has destroyed the characteristic national types of features, the conformation of the jaws, and the regularity of the teeth, but eventually there will be evolved from this mixture of nations, as in the composite photograph, a new type of features which will be distinctively American. When this time comes there will be more perfect harmony between the size of the maxillary arches and the teeth than now exists, with the result that irregularities in their position will be less frequent.

In children born of parents with wide racial differences in the size of the osseous skeleton, the form of the maxillary bones, and the characteristics of the teeth, there is a greater liability to irregularities in the position of the teeth than when those differences are not so strongly marked. As a result of this liability, teeth which are out of all harmony to the size of the jaws are not uncommon sights to the observing surgeon and dentist. This lack of harmony may be a combination of small teeth in a large jaw, or large teeth in a small jaw. In the former the teeth will stand widely apart; in the latter they will be crowded out of position. Both are deformities, but the latter only is amenable to correction by the methods of treatment adopted by the orthodontist.

Other important factors in the causation of irregularities in the position of the teeth are, arrested development in one or both jaws, but most often observed in the superior maxilla, and the hereditary tendency in certain families to malformations of the maxillary bones. The etiology of the irregularities of the teeth is a topic of considerable magnitude, but it has little bearing upon the subject of the formation of tumors of the jaws, except in a general way, for it is only when

these irregularities in position and development are such as to make it impossible for certain teeth to erupt that they become interesting from a pathologic and a surgical standpoint.

Malpositions of the unerupted teeth which are contained within the alveolar processes and remote portions of the maxillary bones are not always productive of the formation of tumors. An examination of the skulls in the dissecting-rooms, and of those found in the museums, abundantly proves this statement, as such teeth have remained imbedded in the jaws for a lifetime without giving any evidence of their abnormal position.

The forms of malposition of the teeth which are most common in the alveolar processes are those in which the teeth lie horizontally in the jaws, or take an obliquely upward or downward direction, or are inverted.

The horizontal and oblique malpositions are usually associated with the third molars and the cuspid teeth, and the writer, judging from his own experience, is of the opinion that the inferior third molars and the superior cuspids are more frequently retained in the jaws from horizontal malpositions than any of the other teeth, or from any other cause.

The following cases are of interest as illustrating these points:

Tumor of the left superior maxilla in a woman fifty-two years of age. Six months previously all of the teeth had been extracted. The tumor had been growing for three months, and was situated upon the external surface and at the base of the alveolar process, extending from the canine prominence backward for more than an inch. Operation by drilling into the tumor revealed a tooth, which was removed with some difficulty, and proved to be a well-developed cuspid, one inch in length. Its direction in the jaw was nearly horizontal, having an oblique inclination downward and forward. After the removal of the tooth the tumor rapidly disappeared.

Another case in a woman of forty-two years; the tumor upon the right side of the vault of the mouth. Teeth were all lost, and she had worn an artificial set of teeth for three years; for the last year could not wear the plate on account of the growth of the tumor. The patient was sure that the right superior cuspid had never erupted. The deciduous cuspid remained until the other teeth were extracted. This made the diagnosis of impacted tooth quite plain. The tumor extended nearly the entire length of the palate process of the jaw, and from the alveolar process to the median line. A thick plate of bone had to be cut away in both cases before the tooth was reached. This also proved to be a well-developed cuspid tooth. The enlargement of the jaw rapidly disappeared after the operation, and did not recur.

A third case in a woman forty-five years of age; tumors or swell-

ings upon both sides of the superior maxilla. The teeth had all been extracted from this jaw fifteen months before, and for twelve months an artificial denture had been worn. During the last half of that time the denture had not fitted well, on account of tumors or swellings located upon the right and left sides of the maxilla in the alveolar ridge, and extending from about half an inch from the median line back to the region of the first molar. The enlargement of the maxilla seemed to be confined to the alveolar ridge. Later the crowns of two cuspid teeth made their appearance at the anterior part of the tumors. The direction of the crowns showed the teeth to be lying in a horizontal position in the jaw. They were extracted with little difficulty. These teeth are well-formed cuspids, evidently not supernumerary teeth, as the patient gives a clear history of retained deciduous cuspids which were not lost until a short time before the jaw was cleared of teeth for the purpose of inserting the artificial denture.

The third molars, in the experience of the writer, are rarely productive of solid tumors of the jaws. Malpositions of these teeth, producing impaction, are much more likely to cause acute symptoms of inflammation, the formation of cysts, and neuralgic affections, both local and reflex, than to result in the development of osseous tumors. Occasionally there will be, as a result of the irritation produced by the impacted tooth, more or less hyperplasia of the overlying bone-tissue, but rarely a condition which could be termed a distinct solid tumor.

Inversion is usually confined to the third molars, but occasionally other teeth may have this malposition. The deciduous teeth are rarely the subjects of retention in the jaws by reason of malpositions, while inversion of these teeth is still more rare.

One such case occurring in a first superior deciduous molar which was inverted has already been referred to in another chapter of this work (page 497).

The most common malpositions occurring in other portions of the jaws—outside of the alveolar processes—are those in which the tooth lies in the palate process of the superior maxilla, or projects into the palate bone. Forget mentions a case of this character in a woman. The tumor was located upon the left side, and extended from the region of the cuspid tooth to the soft palate, and reached beyond the median line. Blandin, in operating for its removal, discovered its cause to be two dwarfed and abnormally-placed molar teeth which had taken a direction toward the median line and had penetrated the palatal wall of the alveolar process. Tomes records a similar case of tumor of the palate caused from a misplaced molar tooth.

Exceptional malpositions of the teeth of the inferior maxilla are the projection of the third molar into the sigmoid notch, or its location in other portions of the ramus. These forms of malposition, however,

would be more likely to induce acute inflammatory symptoms with suppuration, or cystic tumor, than the formation of a solid growth.

Rare malpositions of the third molar teeth of the superior maxilla, like the penetration of the cheek or the floor of the antrum of Highmore, are occasionally recorded. Tomes describes a case in which the superior third molar projected through the cheek, and the writer has referred to a case in another part of this work in which the superior third molar penetrated the antrum of Highmore, and was eventually expelled from the nose through the posterior nares.

Diagnosis and Symptoms.—The diagnosis, as already suggested, is sometimes exceedingly difficult to reach. The age of the patient should be first ascertained, and the progress of second dentition noted, in those passing through this period, to see if the teeth proper to a certain age have made their appearance; or if the individual is an adult, and the tooth is missing in the location of the tumor which cannot be accounted for, this should indicate with considerable certainty that the tumor is caused by a malposed unerupted tooth.

Supernumerary teeth which are impacted in the jaws may also be the cause of tumors of this character. It is therefore possible that such a tumor may occur in a jaw in which the full complement of teeth are in position, by the presence of a supernumerary tooth imbedded within the alveolar process or other portions of the maxillary bone.

The symptoms are simply a slow, painless, progressive enlargement of a certain portion of the maxillary bone, which may be so small or so located as not to cause any inconvenience or deformity; while, upon the other hand, its position and size may be such as to cause great inconvenience or deformity, or both.

Prognosis.—The prognosis in this form of tumor is always good. The removal of the cause is all that is necessary to insure a complete cure.

Treatment.—The treatment in all cases of solid growths of the jaws is to first make an exploratory puncture with needle, trocar, or drill. If the tumor contains a tooth, the exploring instrument will at once indicate this by the greater density of this portion of the tumor as compared with the overlying tissue and the peculiar sensation transmitted through the probe by contact with the tooth itself.

This fact being established, it only remains for the surgeon to cut down upon the tooth with gouge or bur and remove it, after which the enlargement of the bone will gradually disappear, and eventually resume its normal size.

REVIEW.

CHAPTER LXI.

What aberration of the teeth besides that of malformation may cause tumors of the jaws?

What are the causes of such tumors?

Are tumors arising from this cause ever associated with the deciduous teeth?

At what period of life are these tumors most likely to develop?

Is their development confined to this period?

With what condition of the teeth are they associated?

What forms of tumor may arise from the irritation produced by these malpositions?

Which is the most common?

What is the character of these tumors?

What is the stage of development of the teeth found in these tumors?

Is there difficulty in diagnosing the solid tumors of the jaws arising from this cause?

How may the deviations in the position of the unerupted teeth which cause solid tumors of this character be classed?

Which teeth are most liable to occupy malpositions in the jaws?

What would the absence of a certain tooth in the position of the tumor indicate?

Of what value is this fact in the diagnosis?

Are irregularities of the teeth more common among civilized nations to-day than formerly existed?

Are these aberrations more common among such nations than among the aborigines, etc.?

What seems to be an important factor in the production of these aberrations?

In what race are these aberrations in the position of the teeth most common?

What is the effect of the intermarriage of distinct races and nations?

How does heredity affect the transmission of the physical peculiarities and mental capabilities of parents to offspring?

What peculiarities are sometimes noticed in reference to the inheritance of the bony skeleton, and the dermal skeleton?

How is this illustrated in animals?

How may these conditions be accounted for?

From which layer of the germinal disk are the skin and its appendages developed?

From which layer are the teeth, mucous membrane and its appendages developed?

What tissues are developed from the mesoblast?

Which parent usually exerts the dominating influence over the embryo?

In what other respects does this hold true?

What is the converse of this law?

What tendency is frequently inherited by the male offspring?

Do the female offspring inherit the same tendency toward the peculiarities of the father?

How is this tendency often demonstrated?

By what peculiarities are the various races of mankind distinguished from one another?

How are nations distinguished from one another?

Is the conformation of the maxillary arches indicative of the civilization and culture of nations?

What form of arch is indicative of the highest civilization?

What is the result of the intermarriage of so many races and nations?

What form of feature may we hope to see evolved finally from this mixture of nations?

What will be the effect upon the teeth?

What is the effect upon the offspring of parents possessing wide racial differences in the size of the osseous skeleton, the form of the maxillary bones, and the characteristics of the teeth?

How is this fact illustrated?

What are some of the other factors in the causation of irregularities of the teeth?

Are malposed unerupted teeth always productive of tumor formation?

What forms of malposition of the unerupted teeth confined to the alveolar process are most common?

What teeth are usually associated with the horizontal and oblique positions?

Are the third molars often productive of solid tumors?

What conditions are more likely to develop as a result of their impaction?

Which teeth are most likely to be inverted in their position?

Are the deciduous teeth subject to retention and inversion?

What malpositions are most common outside the alveolar processes?

Name exceptional malpositions of the inferior third molar.

What conditions would such malpositions be likely to cause?

What are exceptional malpositions of the superior third molar?

What are the principal points to be observed in making a diagnosis?

Are supernumerary teeth productive of such tumors?

Describe the symptoms.

What is the prognosis?

What preliminary operation should be made in doubtful cases?

How is the offending tooth to be removed?

What is the effect upon the enlargement of the jaw by removing the tooth?

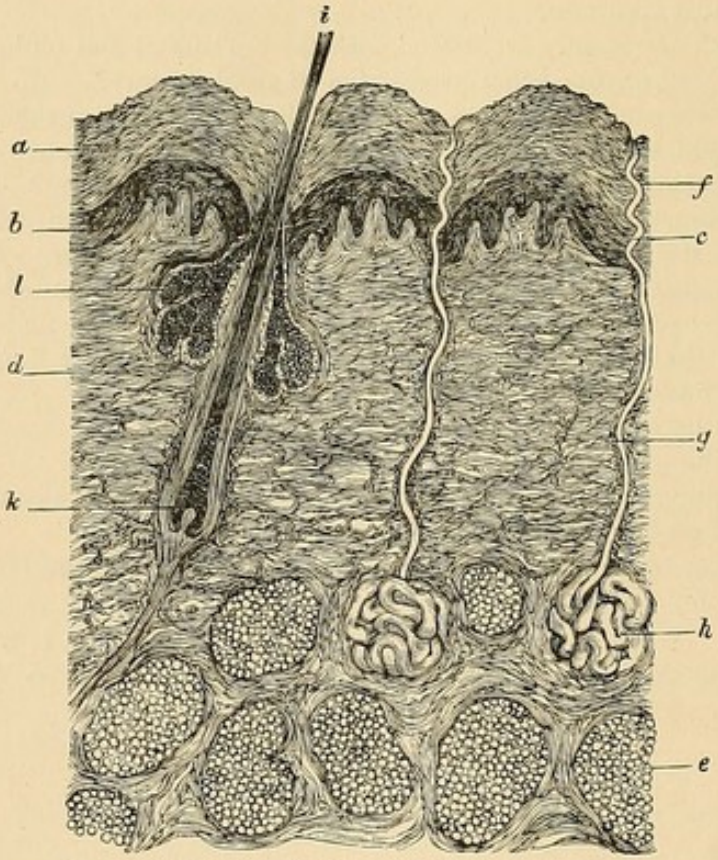
CHAPTER LXII.

RETENTION CYSTS.

Definition.—"A retention cyst is a swelling caused by the accumulation of a physiologic secretion or excretion in a gland or its duct, by obstruction of its natural outlet."

The retention cysts which are located in the region of the face and mouth are associated either with the glandular structures of the skin,

FIG. 362.



VERTICAL SECTION THROUGH THE HEALTHY SKIN.

a, Epidermis; *b*, Rete Malpighii; *c*, Papillary layer; *d*, Derma; *e*, Adipose tissue; *f*, *g*, *h*, Sweat-gland and duct; *i*, Hair; *k*, Hair-follicle and papilla; *l*, Sebaceous gland.

the mucous membrane, or the salivary gland. These cysts may be grouped as follows:

Sebaceous Cysts, Sudoriparous Cysts,	Skin.
Muciparous Cysts,	Mucous Membrane.
Ranula,	Salivary Glands.

Cysts of the Skin.

The glandular structures of the skin are the sebaceous or oil glands, and the sudoriparous or sweat glands. The sebaceous glands belong to that class of glandular structures known as racemose, while the sudoriparous glands belong to the coil form of glandular structures (Fig. 362).

Under normal conditions the epithelial structures of the sebaceous glands secrete an oily liquid known as *sebum,* which becomes condensed in the gland or its duct, into a yellowish semi-fluid, greasy material, of peculiar odor, and containing disintegrated epithelial cells. The accumulation and retention of sebum in the glands or ducts, as a result of obstruction of the outlet, is productive of various disorders of the skin. Among them may be mentioned Comedo, Milium, and Wens, which are forms of cysts.

Comedo, vulgarly known as "black heads," is a disease in which the condensed secretion of the sebaceous glands is retained in the excretory ducts, and becomes visible upon the surface of the skin in yellowish-white or brownish-black points (Hyde). This material is composed of sebum and horny epidermal cells, which occasionally contain minute hairs and the parasite *Demodex folliculorum* (Ziegler). Pressure, if applied, will express the material in the form of a small plug, which is popularly called the "worm."

Comedones are most frequently located upon the face; the nose, forehead, cheeks, and chin being the most favored sites of the disease. It is most frequently observed at the age of puberty, and both sexes are equally liable to the affection. The disease, however, is not confined to this period of life, for it is occasionally seen at all ages.

Causes.—The etiology of the disease is somewhat obscure. It is sometimes associated with an improper care of the skin, but neglect is not always followed by the disease, for certain of the trades, like coal heavers, chimney sweeps, machinists, masons, etc., whose faces are constantly begrimed, are very rarely affected with the disease. On the other hand, it is frequently seen upon the faces of young men and women of the better social classes, whose habits of cleanliness and forms of recreation are of the most healthful character (Hyde). The disease is generally considered as one peculiarly liable to appear during the age of puberty, and in some way associated with the development of the sexual functions. Dyspepsia, constipation, scrofulosis, chlorosis, and menstrual irregularities, are unquestionably associated in the causation of the disease.

Treatment.—The treatment should be first directed to the correction of the constitutional conditions just mentioned, by the use of internal medication, such as the bitter tonics, iron, cod-liver oil, cathartics, and malt preparations, as indicated by the special needs of the

patient. Out-door exercise and hygienic rules in general are to be carried out. The local treatment consists of frequent bathing with hot water, followed by thorough drying, and anointing the face at night with one of the following preparations:

Ointment.

℞—Sulf. precip.,
Vaselin, ℥j. M.

Or the following lotion may be applied:

Lotion.

℞—Sulf. precip.,
Alcohol,
Tinct. lavend. comp.,
Glycerol,
Aquæ camph., *aa* ℥j. M.
(Hyde.)

Unna recommends the following paste for the removal of the comedones, to be applied morning and night:

℞—Acetic acid, ℥ij;
Glycerol, ℥iij;
Kaolin, ℥iv.

The plugs may be dislodged by pressure with the finger-nail, a spatula, or with a watch-key applied over the spots.

Milium or *Grutum* appear as minute, roundish, white or yellowish elevations of the skin (Ziegler). They are millet-seed to pin-head sized, globular masses, rarely attaining the size of a coffee bean, and having the appearance of kernels of rice, lying beneath the translucent layer of the skin (Hyde). They are due to the accumulation of sebum and worn-out epithelial cells, in the sebaceous glands whose ducts have been obliterated. When the nodules are opened, the contents are often found to be concreted into a firm and sometimes calcareous mass. Occasionally they project from the surface of the skin in such a manner as to resemble small vesicles (Hyde), containing a creamy-white substance. Locations in which they are most commonly seen are the eyelids, the cheeks, and the temples, though they are sometimes seen in other portions of the body. They are often of congenital origin, and are frequently seen upon the eyelids and temples of new-born infants. The disease is sometimes developed in middle life. Its progress at such times is slow, and it may persist for an indefinite period, sometimes for several years.

Causes.—The etiology of milium, like that of comedo, is obscure. The disease is sometimes produced by accidental or surgical injury to the sebaceous gland, whereby one or more of the acini are cut off from the main body of the gland, and its duct is obliterated. The contraction of cicatricial bands may also obliterate the duct, and cause a

similar result. In general terms it may be stated that the disease is caused by any condition which prevents the normal transformation of the lining epithelial cells of the glands into sebum, and the excretion of this substance upon the surface of the skin.

Treatment.—Milium is a disease which rarely requires treatment. Milia are relatively few in number, cause no pain, no deformity, or other unpleasant symptom. When treatment is required, each elevation may be opened, the cheesy matter turned out, and the interior of the cyst touched with the tincture of iodin or with a 25 per cent. solution of the nitrate of silver. Hyde recommends a 50 per cent. solution of chromic acid to destroy the gland and prevent a recurrence. Electrolysis is also used for the same purpose.

Sebaceous Cysts or Wens.—The development of sebaceous cysts does not materially differ from that of milia. They originate from occlusion of the natural outlet of the gland, which soon becomes distended by the accumulation of a more or less fluid secretion, resulting in the formation of a cyst. The gland, its duct, and the hair-follicle, are all involved in the cyst, which may be as large as a pea, or attain the size of a large walnut, or exceptionally even greater dimensions. They are usually of slow growth, and are devoid of painful sensations. They appear as single or multiple tumors, most frequently situated upon the scalp, and they are sometimes found upon the back of the neck, and upon the face, but less often upon the trunk and the limbs. Their situation may be upon, within, or beneath the skin. They are usually adherent to the deeper or subcutaneous tissue, and are covered by integument, which is usually devoid of hair. The overlying skin may be normal in color, or abnormally pale from pressure, or reddened, shiny, and greasy in appearance, especially upon the bald scalp of certain fleshy men of middle life (Hyde). The contents of a sebaceous cyst may be soft and pulpy, or firm and friable. Occasionally they are fluid and creamy, or fluid and purulent. They consist of fatty detritus, sebum, epidermal cells, undeveloped hair, and sometimes crystals of cholesterin (Ziegler), inclosed in a capsule composed of layers of epithelial cells and fibrous tissue.

Papillary growths, covered with epidermoid cells, sometimes arise from the inner surface of the walls of a sebaceous cyst, and may increase to such an extent as to completely fill the cyst (Ziegler). The internal layer of cells may become dry and horny (Chiari). In time it may become calcified (Förster).

Causes.—The causes which produce sebaceous cysts are injuries of a traumatic or surgical nature, and inflammatory conditions of the skin, which bring about a permanent occlusion or obstruction of the secretory duct.

Prognosis.—Sebaceous cysts, if injured, are liable to inflame and

ulcerate. Their removal, particularly when located in the scalp, has been followed by erysipelas. With ordinary care as to surgical cleanliness such a result would hardly follow. In the aged these cysts sometimes take on a carcinomatous degeneration.

Treatment.—Excision or enucleation should be performed, either by cutting through the cysts and tearing out each half of the cyst-walls with a pair of forceps, or by cutting through the overlying skin and carefully dissecting out or enucleating the cyst.

Sudoriparous Cysts.—Cysts of the sweat-glands are of very rare occurrence, and little is known about them. They have their origin in the obstruction or occlusion of the duct of the gland, and instead of the gland becoming inflamed and suppurating, the fluid collects and distends the duct. (Tilbury Fox.) The occasional appearance of moisture upon the surface of the swelling, the result of leakage in a partially obstructed duct, has been considered to be a pathognomonic symptom. This form of cyst is sometimes seen upon the face, as the result of the cicatrices of acne, which have obliterated the ducts of the sweat-glands. In strumous subjects it is sometimes quite difficult to cure.

Treatment.—The treatment consists of puncturing the cysts and allowing the contents to escape, followed by repeated application of flexible collodium, or excision of the glands may be practiced. The latter method is most reliable.

Cysts of the Mucous Membrane.

The mucous membrane and the skin are, from the anatomic standpoint, closely related to each other, the difference being that in the skin the epithelium is composed of squamous cells arranged in stratified layers, while in the mucous membrane the epithelium, with few exceptions, is composed of columnar cells arranged in a single layer.

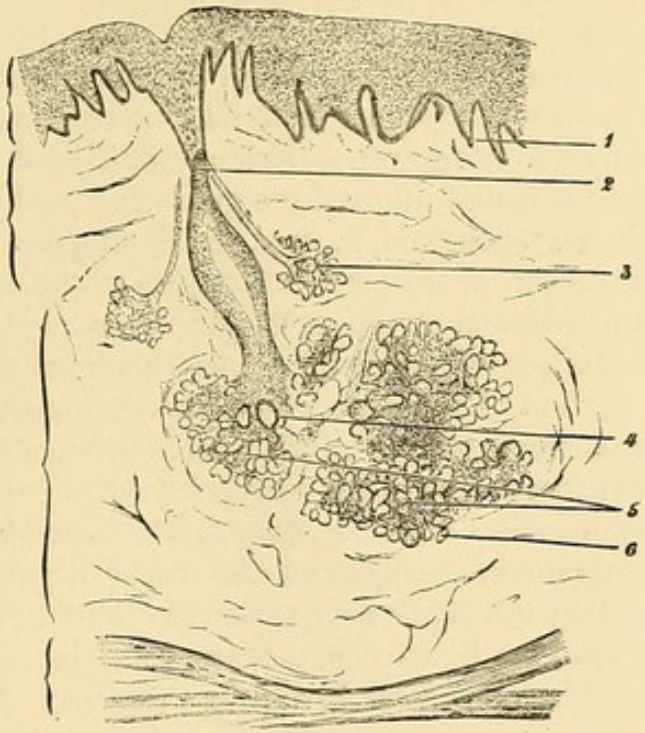
The mucous membrane is also more highly endowed with glandular structures. The mucous glands have their counterpart in the sebaceous glands of the skin, and the retention of their secretions results in the formation of cysts, which have their representatives in the retention cysts of the skin (Fig. 363).

The mucous glands of the mouth (the labial, buccal, lingual, and palatal) are for the most part tubular and racemose glands. The normal secretion of these glands is a grayish, viscid fluid, containing a number of leucocytes and desquamated epithelial cells. The mucus secreted by these glands is of great service to the membrane in both normal and abnormal conditions, as it forms a protective covering, and assists in preventing injury to the membrane from the effects of harmful influences or substances which might come in contact with it.

Muciparous Cysts.—Muciparous cysts of the oral mucous membrane are quite common, and they are found located upon the lips,

cheeks, palate, and tongue. They are small in size, varying from pin-head points to that of a filbert nut or almond. The walls of the cysts are exceedingly delicate; the mucous membrane covering them is very thin, being sometimes almost transparent, and easily ruptured. They contain a viscid fluid, but if the cyst is one of long standing, the fluid may undergo change and become serous in character. These cysts often rupture spontaneously, and leave a deep circular ulcer, which is sometimes slow to heal, if left to itself.

FIG. 363.



VERTICAL SECTION THROUGH THE MUCOUS MEMBRANE OF LIP OF ADULT MAN. × 30.

1, Papilla; 2, Excretory duct, the lumen is cut open at one point only; 3, Accessory gland; 4, Branch of the excretory duct in transverse section; 5, Gland follicles grouped into lobules by connective tissue; 6, A gland tubule in transverse section.

The lips are the most common seat of these cysts, the lower more than the upper. The frequency with which they are found in the other locations may be indicated in the following order: the cheeks, the tongue, and the palate. These cysts are usually single, and several may appear at the same time in the same neighborhood.

Multiple mucous cysts of the lips sometimes occur, and cause such extensive thickening of the lips as to give them the appearance of being double. Excision of the cysts restores the lips to their normal size and form. The origin and appearance of mucous cysts of the cheeks differ in no way from those found upon the lips.

The muciparous cysts of the soft palate, and especially of that portion lying in the vicinity of the tonsils, are of frequent occurrence. They are recognized by their form, which is usually globular or ovoid,

and by the sense of fluctuation (Cohen) when palpation is employed. They are usually small in size, rarely exceeding the dimensions of a buckshot, though they occasionally reach the size of a pigeon's egg.

The tongue is rarely the seat of retention cysts, but when they do occur they are located upon the dorsum or the borders, and more frequently in the posterior portion where the mucous glands are most numerous. In size they rarely exceed that of an almond. In shape they are globular or ovoid; the mucous membrane covering them is smooth (Butlin), and their outlines are well defined. Fluctuation can be detected in the larger cysts, and when near the surface the membrane covering them is translucent. Mucous cysts of the tongue are observed most frequently in adults, though they may occur at any age.

Causes.—Muciparous cysts are caused by obstruction or occlusion of the ducts; or are due to an over-secretion of fluid by the glands. In either case the result is distention of the gland and its duct by the accumulation of the secretion, and, in time, the development of a definite swelling or enlargement of the part. Occlusion of the duct, however, is not an absolute essential to the formation of retention cysts. Obstruction of the duct by a narrowing of its orifice, or by a stricture located in any portion of the duct which caused a partial occlusion, would under these circumstances be effectual in producing a cyst, as it would prevent the free escape of the secreted fluid.

Diagnosis and Symptoms.—The diagnosis of muciparous cysts of the mucous membrane of the lips and the cheeks is generally a very simple matter, as they usually lie near the surface, and are distinguished by their limited size, the translucency of the overlying mucous membrane, their slow development and painless character. Mucous cysts of the palate and tongue are sometimes more difficult to diagnose, especially when they are small and rather deep-seated. The difficulty lies in being able to differentiate them from solid tumors like the fibroma and lipoma, or from a chronic abscess. The only means of positive diagnosis is by an exploratory puncture.

There are no subjective symptoms, and the cyst may not be recognized until it has attained a sufficient size to cause inconvenience, or ruptures spontaneously and is followed by an ulcer.

Treatment.—The treatment of mucous cysts to be curative requires the excision of the cyst or the destruction of its walls. Excision may be employed in the cysts of small size. In those of large size the contents are evacuated by puncture or incision, and afterward the walls of the cyst are destroyed by cauterization with stick nitrate of silver or chromic acid, or by the injection of irritating substances, like carbolic acid, or the tincture of iodin, to excite inflammation and obliteration of the gland. When the cyst is very large it may be packed with antiseptic gauze to promote healing by granulation.

Mucous Cysts of the Antrum of Highmore.—Cysts of the antrum, arising from the mucous follicles of its lining membrane, have been found of such dimensions as to fill the entire cavity, and to cause disfigurement by the distention of its bony walls. (Giraldes.) Cases of this character are likely to be mistaken for mucous engorgement of the antrum resulting from hypersecretion due to catarrhal conditions of the membrane. In fact, there seems to be no way of reaching an absolute diagnosis in these cases except by making a temporary resection of the anterior wall of the antrum. Senn makes this operation "by detaching from within the mouth with a small chisel, a quadrangular muco-osseous flap on three sides, and fracturing its fourth or upper side; by raising this flap the antrum is thoroughly exposed and every part accessible to treatment."

This is an admirable method of gaining access to the antrum in all cases where it is necessary to secure an ocular examination of this cavity to settle a doubtful diagnosis. The flap can be replaced and stitched in position by a few catgut sutures. Union takes place in a few days and the integrity of the antrum is restored, which certainly is better in these cases than to leave a large opening by the sacrifice of tissue, and which may fail to close by granulation.

Treatment.—The treatment, after the antrum has been opened, consists of thoroughly removing the walls of the cyst and curetting the inner surface of the sinus at the point of origin of the cyst. Drainage should be secured for a few days at the lowest point of the flap. Senn recommends the establishment of free drainage through the nose.

Cysts of the Salivary Glands.

Ranula is a term applied to retention cysts of the ducts of the submaxillary and sublingual glands (Wharton's and Rivini's ducts).

Cysts of the duct of the parotid glands (Stenson's duct) also occasionally occur, but in comparison with the prevalence of cysts in the ducts of the salivary glands of the floor of the mouth, they are very rare. A ranula is a collection of salivary fluid in the excretory duct of the salivary gland. Its most common location is in the floor of the mouth, beneath the tongue, upon either side of the median line. It varies in size from that of a pea to a pigeon's egg, or it may be so large as to crowd the tongue back into the fauces and threaten suffocation. Its walls may be thick or thin, and the swelling hard and firm, or soft and fluctuating.

A thick-walled hard swelling indicates a cyst of long standing and inflammatory thickening, with contents greatly changed from the normal secretion, while a thin-walled, soft, fluctuating swelling is evidence of a recently-formed cyst, with a fluid more nearly approximating a normal secretion. The contents of the cyst will vary in character ac-

cording to its period of existence. In cysts of recent formation, the contents are a clear, watery fluid, an unchanged secretion from a normal gland. In cysts of longer standing the fluid may become thick and ropy or gelatinous. Ordinarily the contents are a yellowish, tenacious, ropy, albumen-like substance, too thick to flow from the cyst without pressure upon its walls after an incision has been made for its evacuation. Occasionally the cyst will rupture spontaneously and the tumor disappear as if by magic. One such case the writer had under observation for over ten years, in which this process was repeated several times.

All tumors situated beneath the tongue are not necessarily ranula. Cystic tumors of other varieties are not infrequently found in the floor of the mouth, such as muciparous cysts of the Blandin-Nuhn gland, and dermoid cysts and cystic hygroma.

Ranula is a common affection of men, women, and children, but is more frequently seen in adults than in children. Congenital ranula has also been described by Lannelongue. Butlin thinks that the so-called congenital ranula is probably "congenital cystic hygroma, or one of the cysts belonging to such a tumor."

Causes.—The causes of ranula are contraction or stenosis of the excretory duct, or cicatricial obliteration due to traumatic injuries or inflammatory processes, or to the presence of foreign substances within the duct. Richet in one case discovered the cause of the obstruction to be a fragment of a blade of grass. The writer in one case found a barley beard lodged in the duct of Wharton as a cause of the obstruction; in another the bristle of a tooth-brush, both causing inflammation and stenosis, and in a third a small salivary calculus plugged the orifice of the duct. The most common causes are stenosis and cicatricial obliteration due to injuries and inflammation.

Occlusion or obliteration of the excretory duct are not absolutely necessary, according to Bernard, Weber, Baker, and Senn, for the production of ranula. All of these observers have seen cases of cysts in Wharton's duct in which the orifice was patulous, and a probe could be passed into the cyst. Senn, by pressure, could evacuate the contents through the orifice of the duct. Ranula sometimes follows amputation of the tongue from injury or implication of the salivary ducts in the cicatrix of the wound. It often results from superficial ulcers of the mucous membrane located in the immediate neighborhood of the orifices of the ducts.

Sonnenburg and Von Recklinghausen believed these cysts were due to the dilation of the Blandin-Nuhn gland, which is situated in the floor of the mouth beneath the *frenum linguæ*. This is a racemose mucous gland.

Diagnosis and Symptoms.—The diagnostic signs of ranula are a

progressively-increasing swelling located in the floor of the mouth beneath the tongue, upon either side, accompanied by a sense of fullness, and of painless character. In appearance it is a smooth, bulging tumor of bluish tint and sometimes translucent aspect. When of large size, the tongue is lifted up and there is fullness in the submaxillary triangle; the swelling is not painful to the touch, or only slightly so; it may be hard or fluctuating, of long or recent existence. When the former, it is hard and firm; when the latter, it is soft and fluctuating. A positive diagnosis may be made by the introduction of an exploring needle.

Prognosis.—Recurrence is the rule unless a permanent orifice to the duct can be established, or a radical operation is made for the removal of the gland.

Treatment.—The main object in the treatment of ranula is to secure the establishment of an artificial orifice of the excretory duct, which shall answer the purpose of a normal apparatus. This can only be secured by a surgical procedure.

Various methods of surgical treatment have been suggested and practiced with this end in view:

1st. The evacuation of the cyst by free incision of its superior wall is a common method; but this affords only temporary relief, as it is usually but a short time before the wound closes and the fluid reaccumulates.

2d. Excision of a portion of the superior wall of the cyst at its anterior extremity, followed by cauterization of the cyst walls, gives better success, and yet even this treatment does not in some instances give the desired result. This is explained by the fact that after the contents of the cyst have been evacuated, the edges of the wound fall together, and they are eventually united, and the object of the operation is defeated.

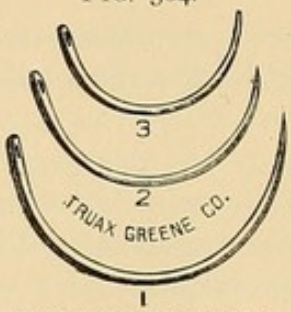
3d. Another method is to introduce a seton through the walls of a cyst, and allow it to remain until permanent openings are secured at the points of entrance and exit of the seton. The operation consists of threading a large, full, curved needle (Fig. 364), with heavy braided silk previously waxed, or with silver wire, and passing the needle through the walls of the cyst from side to side, near the anterior extremity, cutting the suture of proper length, and uniting the ends by tying the silk and twisting the wire. The seton should remain from one to two weeks, or until such time as the punctures in the cyst wall have healed around the seton. After the seton has been secured the cyst should be opened by an incision, and its contents evacuated. This method gives good results.

4th. Another method of securing a permanent opening in the wall of the duct is to make a V-shaped incision in the superior wall of

the cyst, turn back the triangular flap thus made, and stitch it in its new position. This leaves a somewhat large opening, which will in some cases remain patulous and give free exit to the secreted fluid.

5th. A more heroic method, based upon the supposition that the disease is due to the dilatation of the acini of the Blandin-Nuhn gland, is the extirpation of the entire cyst and the remains of the gland. This is a much more difficult operation than the others, and in some instances may be necessary. When thoroughly done it proves successful.

FIG. 364.



FULL-CURVED NEEDLES.

Efforts have been made to destroy the cyst by injections of the tincture of iodin, but this is unsafe practice on account of the great swelling likely to occur, and the dangers of causing suffocation by forcing the tongue into the fauces. The late Professor Garretson mentions such a case as occurring in his own practice which nearly proved fatal.

The use of stimulating applications to the inner walls of the cyst is to be deprecated for the same reasons, and also because they are unnecessary, as nature very soon reduces the expanded duct to its proper caliber after the establishment of patulous openings in the walls of the cyst.

REVIEW.

CHAPTER LXII.

Give the definition of the term *retention cyst*.

With what structures are retention cysts of the face and mouth associated?

What forms of cysts are located in the skin?

What term is applied to the retention cysts of the mucous membrane?

By what term are the retention cysts of the salivary glands designated?

To what classes of glandular structures do the sebaceous and the sudoriparous glands belong?

What is the name and the character of the material secreted by the sebaceous glands?

What forms of cysts of the skin are caused by the obstruction of the outlet of these glands?

What are the cysts known as comedo?

Of what is the retained material composed?

Where are comedones most frequently seen?

What portions of the face are favored locations?

At what age is the disease most prevalent?

What are its causes?

What general conditions are often associated with it?

Describe the treatment.

Give the formula for the sulfur ointment.

Give the formula for the sulfur lotion.

Give the formula for the acetic acid paste.

How may the contents of the cysts be dislodged?

Describe the disease known as milium.

What are the contents of these cysts?

Where are they most frequently located?

At what periods of life are they generally seen?

What is the character of their development when occurring in middle life?

What are the causes of the disease?

Is treatment required?

When required, how should the cysts be treated?

Describe the development of a sebaceous cyst.

What is its size and rapidity of growth?

Where are they most frequently located?

What is their situation in the skin?

What is the appearance of the overlying integument?

What is the character of their contents?

By what structure is this material surrounded?

What changes sometimes take place within the cyst?

What are the causes?

State the prognosis.

Describe the treatment.

Describe the development of a sudoriparous cyst.

Where is it sometimes located?

What are its causes?

Describe the treatment.

What is the chief anatomic difference between the skin and the mucous membrane?

In what respects are they very similar?

To what class of glandular structures do the mucous glands belong?

What is the character and appearance of their normal secretion?

What is the function of the mucus?

Are muciparous cysts common?

Where are they found located?

What is their size?

What is the character of the cyst walls?

Of what do the contents consist?

What follows spontaneous rupture of the cysts?

State the most frequent location of these cysts.

In what other locations are they found?

Do they appear singly or in groups?
How do multiple cysts affect the lips?
What, if any, difference exists in the origin and appearance of cysts of the lips and of the cheeks?
In what portions of the tongue are these cysts sometimes observed?
How are they recognized?
How large do they grow?
In what portions of the tongue are these cysts sometimes observed?
What size may they attain?
What is their form?
Is the covering membrane smooth or rough?
Are the outlines indistinct or well defined?
Under what circumstances is fluctuation detected?
At what age are they most frequent?
What are the causes of muciparous cysts?
Is occlusion of the duct necessary to cause a cyst?
What other conditions may be effectual?
Give the diagnosis of mucous cysts.
Are cysts of the palate and tongue more difficult to diagnose, and if so, why?
By what means may a positive diagnosis be reached?
What are the symptoms?
What conditions are necessary to obtain a cure?
Which method is applied to the small cysts?
Describe the methods of treating a large cyst.
What is the character and size of mucous cysts of the antrum?
For what affection might they be mistaken?
How may a positive diagnosis be established?
Describe Senn's operation for opening the antrum.
What is the advantage of the operation?
What is the treatment after the sinus has been opened?
What is the definition of the term *ranula?*
Is ranula ever associated with the duct of Stenson?
What is a ranula?
Where are they usually located?
What size may they attain?
What is the character of the walls?
What do these conditions indicate?
What is the character of the contents of these cysts?
What do these conditions indicate?
Describe the appearance of the fluid ordinarily found in a ranula.
Does spontaneous rupture effect a cure?
What other cysts are found beneath the tongue?
Is ranula common?
At what period of life is it most frequent?
What are the causes?
What forms of foreign bodies have been known to obstruct the duct?
What are the most common causes?
Is occlusion or obliteration of the duct necessary to produce ranula?
What do authorities say upon this point?
Name other causes of ranula.
What other gland may become the seat of a retention cyst and simulate ranula?

Give the diagnostic signs and symptoms of ranula.
How may a positive diagnosis be made?
What is the prognosis?
What is the main object in the treatment of ranula?
By what methods may this be accomplished?
Describe the first method.
Is this usually successful? if not, why?
Describe the second method.
Does it give better results?
When it fails, what is the cause of the failure?
Describe the third method.
How long should the seton remain?
What are the results?
Describe the fourth method.
Does this succeed?
Describe the fifth method.
Upon what is this method based?
What remedies are used in the injection method?
What are the objects of this method?
State the complications and dangers which arise from the adoption of this method.
Why are stimulating applications also to be deprecated?

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