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**HISTOPATHOLOGY OF
THE TEETH**

AND

THEIR SURROUNDING STRUCTURES

BY

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TO
MY WIFE
MARGARET
WHOSE LOVING COMPANIONSHIP
UNENDING PATIENCE AND SYMPATHETIC ENCOURAGEMENT
MADE THIS WORK POSSIBLE



PREFACE.

THE rapid progress in microscopic dental research during the last few years has brought forth so many new and fundamental facts that a description of these recent findings together with the resulting new conceptions of old problems needs no justification.

Previous to the last decade, dental research made use of only three types of specimens for microscopic studies, namely, extracted human teeth, animal jaws, and dried human bone specimens. By means of such material much valuable and fundamental knowledge of a great many subjects in normal and abnormal dental histology was gained. While a study of dental caries and a study of the histology and pathology of enamel, dentin, and pulp could be made satisfactorily from ground or decalcified sections of extracted human teeth, normal or pathological conditions lying beyond the tooth proper presented an entirely different problem. Here the study of extracted teeth could not give the desired information. The older investigators, realizing this limitation, took refuge in the study of animal tissues, and microscopic descriptions of the hard and soft tissues investing the tooth were obtained largely from specimens of sheep, dogs, and other animals. But it remained an open question as to what extent the periodontal tissues of these animals compared with the corresponding structures in man. Thus there was evident the need for a new type of investigation, namely, the study of human teeth in connection with their investing hard and soft tissues under normal and pathological conditions.

Some problems discussed in this book, such as the histology of the epithelial attachment and the gingival crevice or the influence of normal functional conditions upon teeth and surrounding structures, belong to the field of physiology and biology rather than to pathology. However, since the pathological changes in the gingival tissues and in the periodontal membrane cannot be understood without a thorough knowledge of normal conditions, it was indispensable to enlarge upon the latter before entering into the question of the former.

The purpose of this book then may be characterized as follows:

to illustrate by means of human specimens the actual tissue changes that correspond to certain well-defined, clinical conditions. For example, a cavity is prepared and a filling is inserted. What tissue changes can be expected to follow this operation? The radiograph shows an area of bone destruction at the root end of a pulpless tooth. What tissues and cell forms are found in this area? A pulp is removed and the root canal is treated and filled. How will the periapical tissue react to this procedure? A bridge is cemented on abutments, thereby increasing the occlusal stress exerted upon these teeth. What will be the immediate and the later changes in the soft and hard tissues about the roots of the abutments? A tooth is moved orthodontically. What are the bone changes that make possible the movement of the tooth and its retention in the desired position? The author hopes, by introducing the clinical aspect of these problems instead of merely describing microscopic specimens, to facilitate the understanding of many clinical manifestations and practical observations.

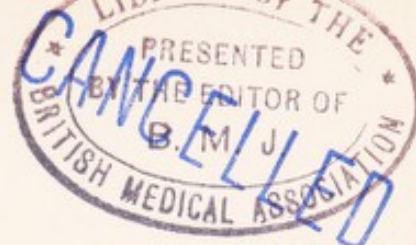
The field of dental and oral pathology is so large that it is impossible to treat any particular subject in detail. Some problems have been well described and illustrated in earlier publications and textbooks. Others are relatively new and unknown to the profession; this latter group will be considered here more in detail.

The majority of the specimens described in this book has been taken from man. Animal tissues were used only to illustrate changes that were produced experimentally. All illustrations, except the diagrams, are photomicrographs, the author believing that, for the sake of objectivity, photographic reproductions are preferable to drawings. Unless otherwise indicated, the specimens from which the photomicrographs were taken belong to the collection of the Research Department of the Chicago College of Dental Surgery, Dental Department of Loyola University.

I wish to thank Dr. W. H. G. Logan for the helpful encouragement he has given this work, and to express my appreciation to Drs. R. W. Bunting, E. D. Coolidge, and E. B. Fink for the many valuable suggestions that I received from them and incorporated into this book. I also wish to express my gratitude to Miss Maurine Willman, who skilfully prepared all the necessary histological specimens and assisted in the preparation of the manuscript.

R. K.

CHICAGO, ILL.



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HISTOPATHOLOGY OF THE TEETH.

CHAPTER I.

HISTOLOGY AND PHYSIOLOGY OF THE DENTAL TISSUES.

NORMAL HISTOLOGY OF PULP AND DENTIN.

THE dental pulp is a connective tissue organ built up of spindle-shaped or star-shaped cells of the fibroblast type with protoplasmic processes running in all directions. The pulp cells and their processes form a fine network, the interspaces of which are filled with a gelatinous ground substance. The cells of the young pulp differ somewhat from the fibroblasts found elsewhere in the body; they are usually described as embryonic cells, as they are of the same structure as the connective tissue cells of a fetus or the cells of the umbilical cord.

Arteries, veins, capillary vessels, lymphatics and myelinated nerves are embedded in the pulp tissue. The vessels and nerves enter the pulp through the apical foramen and connect the pulp with the circulatory and nervous systems of the body.

The outline of the pulp, as a whole, duplicates the general contour of the tooth. The main vessels and nerves usually lie close together and run parallel to the long axis of the tooth, splitting into finer branches as they spread out toward the surface of the pulp (Fig. 1). Next to the dentin the bloodvessels and nerves form a delicate terminal plexus.

The structure of the pulp in the root canal is very similar to that in the pulp chamber. In a cross-section through the apical portion of a root canal, the larger vessels, that carry the blood to and from the pulp, and the main trunks of the pulpal nerves are seen in transverse section embedded in the pulp tissue (Fig. 2).

The bloodvessels of the pulp have very thin walls; this is easily understood if we consider the fact that the entire organ is enclosed in a rigid capsule of hard substance which prevents any strain

or pressure from reaching the soft tissues. They are embedded in a fine, fibroblastic network (Fig. 3).

Histiocytes and undifferentiated mesenchyme cells, both of which play important rôles in the defense reaction of the pulp tissue in

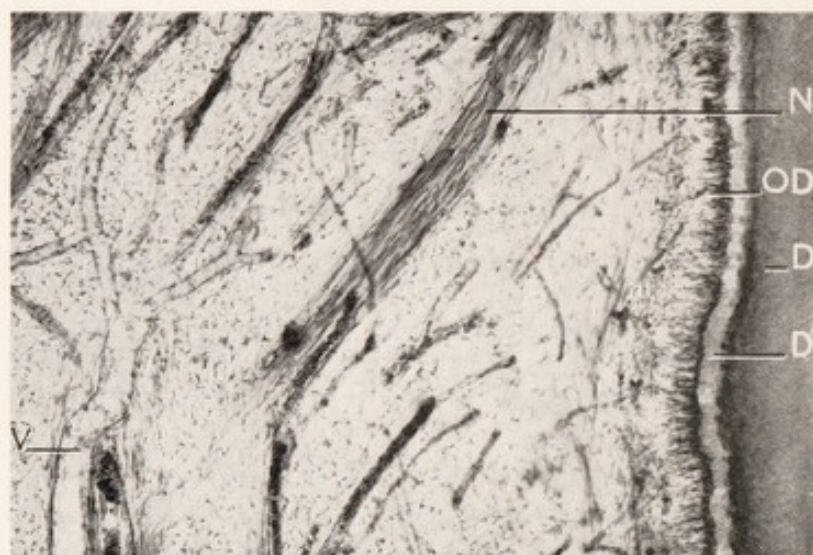


FIG. 1.—Human pulp. Lower molar of a young adult. V, bloodvessel (vein); N, nerve; OD, odontoblasts; D', dentinoid; D, dentin.

case of injury or infection, have recently been studied and demonstrated in the human pulp (Orban). The presence of these cells in normal pulp tissue indicates that the pulp is equipped with the same defense mechanism as the connective tissue in any other part

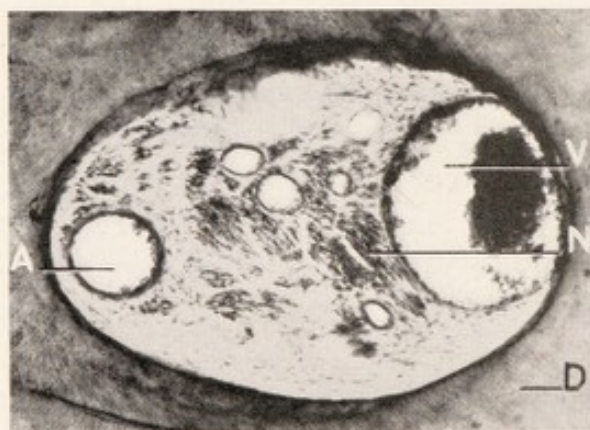


FIG. 2.—Cross-section through the root canal of a human bicuspid near the apex. A, artery; V, vein; N, nerve bundles; D, dentin.

of the body. Fig. 4 shows a small capillary vessel in a normal human pulp. The vessel contains erythrocytes and coagulated serum, and in its delicate wall may be seen the large, flat nuclei of the endothelial cells. Two large cells with elongated nuclei and

granulated protoplasm are found in the fibrillar pulp matrix near the capillary. These cells are the so-called resting wandering cells, or histiocytes; according to Maximow they develop from cells which

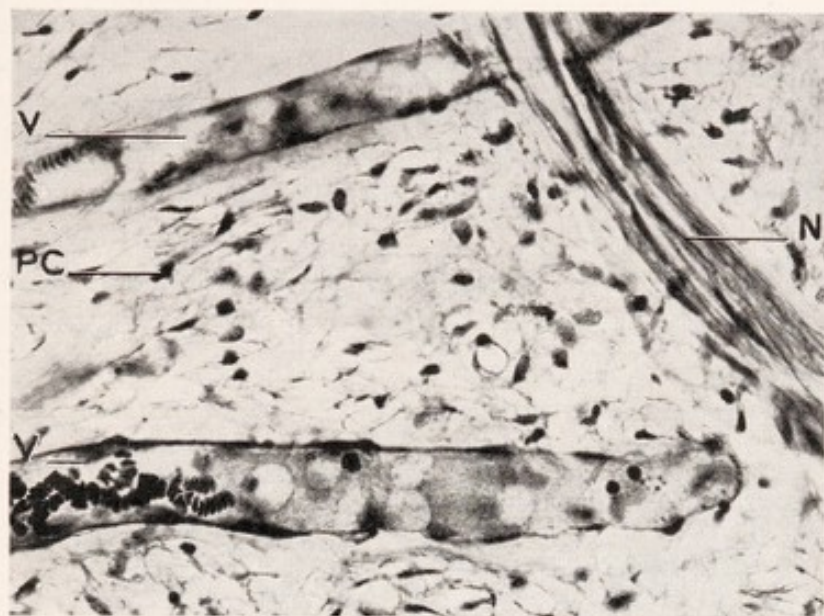


FIG. 3.—Capillaries and a small nerve in the connective tissue of the pulp. V, vessels containing red blood corpuscles; N, nerve; PC, nuclei of pulp cells.



FIG. 4.—High magnification of a capillary vessel and the surrounding tissue in a human pulp. H, resting wandering cells (histiocytes); UM, undifferentiated mesenchyme cell; E, endothelial cell of the capillary; PC, pulp cell. (Orban, Jour. Am. Dent. Assn.)

wander through the loose connective tissue over all the body. The histiocytes serve both as phagocytes and as storage cells. In case of inflammation they develop into amœboid phagocytes, and wander to the place of irritation where they engulf and carry away bacteria

and tissue débris. These cells also have the property of storing foreign bodies that are brought into the organism.

The undifferentiated mesenchyme cells, which are found in close proximity to the bloodvessels, have the properties of embryonic connective tissue cells: under an adequate stimulus they may become transformed into any type of connective tissue cell or into blood cells. They may also become phagocytic wandering cells under the stimulus of inflammation. In Fig. 4 two of these undifferentiated (perivascular) mesenchyme cells can be seen near the capillary wall.

The main function of pulp tissue is to build dentin. The formation and structure of human dentin will be outlined only briefly in this book.¹

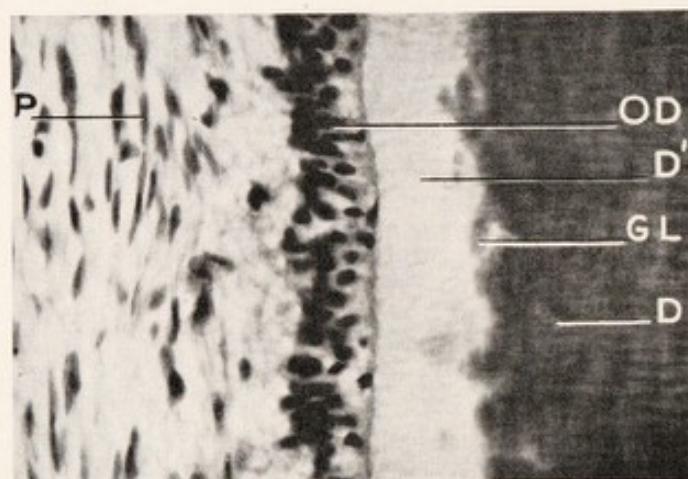


FIG. 5.—Normal young human pulp and dentin. P, pulp tissue; OD, odontoblasts; D', dentinoid (uncalcified dentin matrix); D, dentin with dentinal tubules; GL, globules of calcification at the border between dentin and dentinoid.

In the teeth of young people the entire surface of the pulp is lined by a layer of large, elongated cells. These cells, the odontoblasts, are specialized pulp cells developing from the superficial layers of the dental papilla of the tooth germ. Each odontoblast has a long protoplasmic process, the Tomes' fiber or dentinal fibril which extends through the entire thickness of the dentin to the dento-enamel junction. The Tomes' fibers lie in fine canaliculi in the dentin, the dentinal tubules.

The dentin consists of a soft, fibrillar matrix, the predentin, in which lime salts are deposited. The deposition of inorganic matter into the forming dentin begins at a distance from the pulp surface;

¹ For more detailed information the reader is referred to the publication of Orban (*Jour. Am. Dent. Assn.*, 1929, **16**, 1547), which also contains a complete bibliography on this subject.

the innermost layer of the dentin, therefore, consists of uncalcified matrix and is called dentinoid. In specimens stained with hematoxylin, the dentinoid appears lighter in color than the calcified dentin (Fig. 5). The calcium salts in the matrix are deposited in the form of globules which gradually fuse. They can be observed in sections of dentin in which calcification is not complete. In the periphery of the pulp chamber of a young tooth the relationship between pulp, odontoblasts, dentinoid, and dentin is plainly visible (Fig. 5). *Tomes' fibers* may be seen running through the dentinoid as well as through the dentin.

Near the dentin surface the dentinal tubules ramify and communicate with branches of neighboring tubules (Fig. 6). Thus, the tubules form at the borderline between dentin and enamel the so-called marginal plexus.

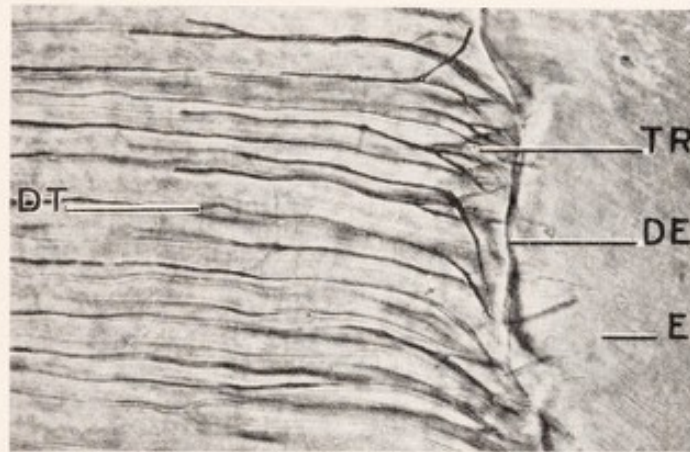


FIG. 6.—Terminal ramification of the dentinal tubules at the dento-enamel junction. Ground section. DT, dentinal tubules; TR, terminal ramifications (marginal plexus); E, enamel; DE, dento-enamel junction.

Recent investigations have shown that *Tomes' fibers* are hollow, pipe-like structures filled with a fluid or semi-fluid protoplasmic content (Wolf, Fig. 7). They are separated from the wall of the dentinal tubules by a capillary space (perifibrillar space, Fish). It has been found by animal experiments that fluids, solutions and minute solid particles may be carried from the pulp into the dentin. This transportation may take place in the capillary space between the tubule and the dentinal wall of the tubule, or perhaps inside of the tubule itself.

Dentin formation starts in the fifth month of fetal life in the deciduous teeth, and is normally continued in the permanent teeth all during life. This means that the pulp chamber steadily decreases in size, a fact of great importance in the practice of dentistry.

The knowledge concerning the mechanism of dentin development was revised and enlarged recently by Orban. He showed that the Korff's fibers, the argyrophil fibers of the pulp (fibers that stain black with silver stains), continue between the odontoblasts into the dentin, where they spread fanwise and interlace, and that there they undergo a structural and chemical transformation into the collagenous fibers (as indicated by the change in staining properties) which form the dentinal matrix. Orban has demonstrated also that throughout life Korff's fibers play the same rôle in the development of the dentin. In the teeth of older people they can be plainly seen in connection with the dentinal matrix. This would indicate that the dentinal matrix is actually built up by the Korff's fibers of the pulp and not by the odontoblasts. The odontoblasts, however, furnish Tomes' fibers and seem to be important for the vital processes (sensitiveness, nutrition, etc.) in the dentin.

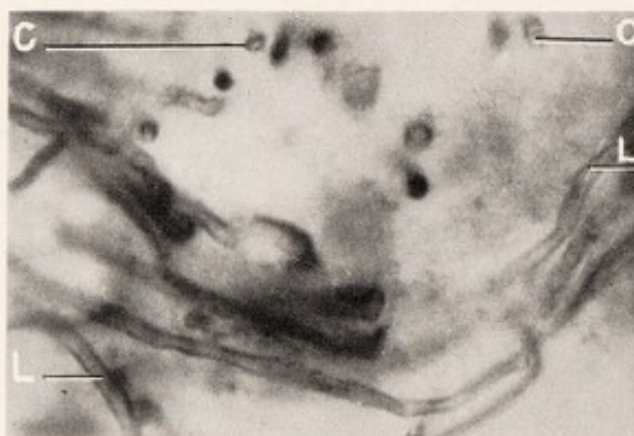


FIG. 7.—Odontoblastic processes (Tomes' fibers) isolated by preparatory over-decalcification of the dentin. Longitudinal (*L*) and cross (*C*) sections through Tomes' fibers. In the cross sections the fibers appear as hollow pipe-like structures. (Wolf, *Ztschr. f. Stom.*, courtesy of Urban & Schwarzenberg, Berlin-Wien.)

Under certain pathological conditions or in case of abrasion, another type of dentin is formed, the secondary or irregular dentin. This dentin develops without the presence of odontoblasts. It consists of calcified dentinal matrix with few and irregular dentinal tubules, and it is built by direct calcification of the pulp fibers (see Chapter II).

THE PROBLEM OF INNERVATION OF THE DENTIN.

The dentin of a young tooth with a vital pulp is highly sensitive to any kind of mechanical, chemical, or thermal irritation. This is best illustrated by the sensitiveness of the exposed dentin at the

bottom of a freshly prepared cavity in a vital tooth. By such a dental operation many thousands of dentinal tubules are cut open, and the odontoblastic processes lying in these tubules are severed and exposed to outer irritation; any stimulus that acts upon the exposed dentin surface is conveyed to the pulp along the 'Tomes' fibers.

The high sensitiveness of the dentin suggested the presence of nerves, and for a long time histologists have been trying to demonstrate the existence of these nerves. This appears to be a simple matter. All that seems necessary is to treat the dentin with stains having a peculiar affinity to nerve tissue and thus prove or disprove the presence of nerves. In reality, however, the staining of dentinal nerves is an exceedingly difficult problem of histological technique. In order to cut dentin into thin sections, it must be decalcified, and the action of the acid greatly lessens the staining qualities of the tissues. The metal salts (silver stains) that impregnate nerve tissue affect connective tissue fibrillæ similarly, so that the latter can hardly be distinguished from unmyelinated nerve fibers. In addition to these difficulties, it must be kept in mind that a dentinal tubule is not wider than 1 or 2 microns; therefore, since most of the tubule is occupied by the 'Tomes' fiber, an accompanying nerve fiber can be only a fraction of a micron thick.

Due to all these difficulties the presence of nerves in the dentin was undecided until very recent times. Some authors had reported the finding of nerves; others had given a different interpretation to the structures found. Under these circumstances it was not surprising that some investigators, especially Walkhoff, strictly denied the existence of nerves in the dentin. Walkhoff believed that the protoplasm of the 'Tomes' fiber itself had the property of transmitting stimuli from the periphery to the pulp, where the irritation was taken over by the nerves of the pulp. Other investigators were of the opinion that certain delicate structures observed inside the dentinal tubules were true nerves (Dependorf, Mummery).

In 1927 Dieck and Toyoda reported investigations by which the presence of nerves in the dentin seemed to be definitely established. The authors used a special histological technique by which, after decalcification, complete removal of the acid from the dentin was possible, thus greatly improving their specimens. By this method, they demonstrated how the myelinated nerves of the pulp, after losing their myelin sheath near the pulp surface, enter the dentinal tubules and extend through the thickness of the dentin to the dento-enamel junction. These unmyelinated nerve fibers are arranged on

the surface of the protoplasmic process and with their fine ramifications embrace the process. Very fine branches run from the tubules into the dentin substance.

THE PROBLEM OF CIRCULATION IN THE DENTAL HARD TISSUES.

Ever since dental structures were made the subject of scientific investigations, the existence of some form of metabolism or circulation in the hard tissues has occupied the minds of scientists. An enormous bibliography has accumulated on this subject, but conclusive evidence still seems to be lacking. It is impossible to outline all the work that has been done within the last half century; therefore, the author will confine himself to a brief description of the possible pathways along which such a supposed circulation or fluid exchange within the dental hard substances could take place and of some findings obtained in animal experimentation.¹

1. Pathways for a Possible Penetration or Exchange of Fluids in Dentin and Enamel.—Dentin is a calcified substance pierced by a large number of fine canaliculi, the dentinal tubules. These tubules contain living protoplasm, namely, the protoplasmic processes of the odontoblasts, and, consequently, the taking up of food materials and the removal of waste products can be expected. In fact, in animals it is possible to show that certain substances brought into the tooth or into the organism follow the preëxisting path of the dentinal tubules through the dentin to the dento-enamel junction.

The enamel offers a decidedly different problem, since no channels are present that compare with the dentinal tubules. The enamel, generally speaking, is completely calcified, and contains only a few organic structures that could be considered pathways for the exchange of fluids. These structures are the spindles, the tufts, the lamellæ, and, within certain limits, the interprismatic substance and the prism sheaths.

The enamel spindles are club-shaped extensions of the dentinal tubules projecting beyond the dento-enamel junction into the enamel (Fig. 8). Their distribution and frequency is rather variable; while they may be very numerous in some teeth, they may be completely missing in other teeth or in different areas of the same tooth. The direct communication of these spindles with the dentinal tubules has been proved in staining experiments on extracted

¹ To anyone intending to make a more detailed study of the subject, the critical review by Chase of 136 papers may be recommended as a starting point (*Jour. Am. Dent. Assn.*, 1931, **18**, 697).

human teeth; dye, brought under pressure into the pulp chamber of such teeth, has been forced through the tubules into the spindles.

The tufts are groups or bunches of poorly calcified enamel rods that extend from the dento-enamel junction through about one-third of the thickness of the enamel. They are always found in sections through human teeth (Fig. 9).

The enamel lamellæ are bands that extend from the outer enamel surface into the enamel. They may extend partly into the enamel, or through its entire thickness to the dento-enamel junction, or past the dento-enamel junction into the dentin. They consist either of poorly calcified enamel substance or of organic tissue rem-

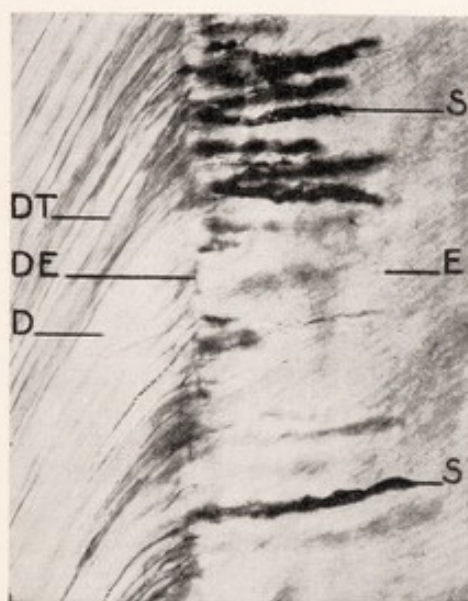


FIG. 8.—Enamel spindles near the tip of an upper incisor. D, dentin; DT, dental tubules; DE, dento-enamel junction; E, enamel; S, enamel spindles.

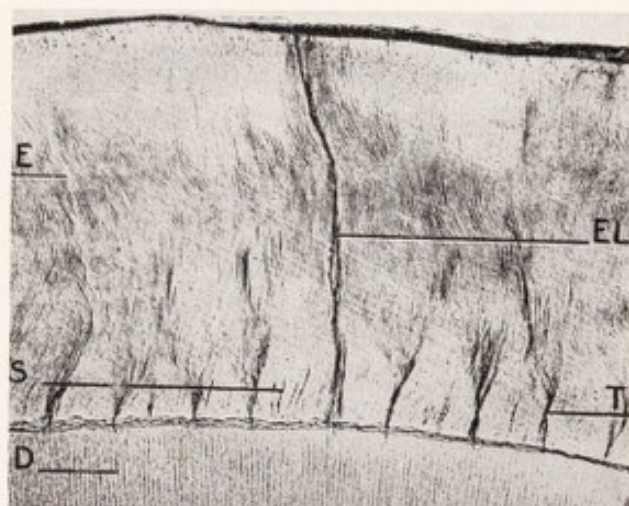


FIG. 9.—Ground section through a human tooth at right angles to the long axis of the tooth. E, enamel; D, dentin; EL, enamel lamella; T, tufts; S, enamel spindles. (Orban, Dental Histology, courtesy of P. Blakiston's Son & Co.)

nants that grew into crevices of the enamel before the eruption of the tooth (Orban). These organic bands run lengthwise through the crown in a plane perpendicular to the dentin surface (Fig. 9).

The interprismatic or cementing substance and the prism sheaths are found between the enamel rods. They are variable and inconstant structures. In human enamel only a small amount of calcified cementing substance is found between the rods. The enamel of dogs' teeth has a well-developed interprismatic substance between the individual prisms. In man the interprismatic substance is calcified, the same as the rods themselves; in young dogs, however, there is an organic prism sheath between rod and interprismatic substance.

2. Demonstration of the Pathways on Extracted Human Teeth by Means of Dyes.—In order to investigate to what extent the above-described organic structures in the enamel are actually accessible to fluids, staining experiments were performed on extracted teeth. Beust reported, in 1912, that an alcoholic fuchsin solution, brought into the pulp chamber of an extracted human tooth, passed through the dentinal tubules to the dento-enamel junction and entered the enamel, and that the enamel tufts and the interprismatic substance were stained. Smreker obtained very similar results with diamond fuchsin; the amount of dye in the enamel decreased with increase in distance from the dentin surface. Recently Beust stained enamel spindles and tufts through the dentin. He stated that while in young teeth the prism substance has an affinity for stain, with advancing age this colorability of the rod disappears; the interprismatic substance continues to conduct stain for a time, but in old teeth the enamel is entirely impermeable to dyes.

The dentin, too, changes its permeability during life. Beust showed that with advancing age, especially under the influence of outer irritation (abrasion), the dentinal tubules become less and less permeable to dyes, apparently by obliteration of their lumen through deposition of calcium salts.

Fish reported that, after placing methyl blue into the pulp chamber of extracted human teeth and keeping them in saline solution for several weeks, the dye could be seen filling the dentinal tubules to the dento-enamel junction, and that some of the prism sheaths next to the enamel tufts were stained blue also, indicating that there is communication between the dentin and the uncalcified portion of the cementing substance.

3. Demonstration of the Pathways by Vital Staining.—The biological way to investigate whether fluids can enter the dental hard tissues is by means of vital staining. A large number of such experiments has been performed. They can be carried out either by introducing some dye into the general circulation (intravenous, subcutaneous, or intraperitoneal injection) or by introducing some dye directly into the pulp tissue.

(a) *Introduction of Dyes Into the General Circulation.*—The first group of experiments may be subdivided into investigations on developing, growing teeth, and on fully developed teeth. Gottlieb fed and injected puppies with madder, a red dye of vegetal origin that has a special affinity for calcified tissues. He demonstrated the presence of red zones in the enamel of the teeth that were being formed at the time of the experiment, each zone corresponding to

one distinct period of injection. Similar results were reported by Gies, Blotevogel, and Proell.

Gies demonstrated on a number of dogs the effect of intraperitoneal injections of trypan blue at different stages of growth and dentition. He found that, if trypan blue were injected before the permanent teeth had begun to form, the permanent teeth were blue over the entire surface of each crown. If, however, trypan blue were injected soon after the permanent teeth had begun to form, the teeth were white except for a wide blue zone in the cervical portion. If the injections were made after the enamel of the permanent teeth had been fully formed, the enamel was white while the dental pulp and adjacent dentin were blue. In all animals, when the injections had been made shortly before death, gums and skin were blue in color; while in a dog in which the injections had been begun soon after the dog's birth but had been discontinued two months prior to death, the enamel of all permanent teeth was deeply and uniformly blue; whereas, practically all the blue color had disappeared from the gums and skin. If such blue teeth were fractured, it was found that the blue pigment was contained in both dentin and enamel. From these findings Gies suggests that substances circulating in the blood during the period of enamel development may be permanently incorporated into and retained by the enamel. The problem of mottled enamel in man may be considered from the same point of view. Here, too, we are dealing with a preëruptive change in the enamel, brought about by the fluorin content of the drinking water (McKay). The resulting pigmentation and disturbed calcification of the enamel is an irreversible process: once the teeth have been affected in childhood by fluorin in the drinking water and have become mottled, they remain so throughout life. It is not possible later to remove the discoloration of the mottled enamel, nor can the condition be produced after all of the enamel has been formed.

These findings are of great importance in connection with the statement occasionally encountered in literature "that calcium may be mobilized from the teeth in case of a general deficiency in the mineral metabolism." This conclusion seems to be drawn by analogy. While it is a well-established fact that under certain conditions (pregnancy) the mineral salts of the bone may be mobilized and used in other parts of the organism to meet an emergency, such a process has never been demonstrated in the teeth, although it is often alluded to as a fact. On the contrary, the permanent retention of dyes in the enamel suggests very strongly that no substance

can be mobilized again once it has become incorporated into the enamel.

Urbantschitsch experimented on bats with fully developed teeth by injecting subcutaneously from 1 to 1.5 cc. of a solution of trypan blue or lithium carmine. After a period of from several hours to three days, ground sections of the teeth were prepared. The dye was found stored in the pulp tissue. From there it had penetrated into the dentin and, in places, also into the enamel; the latter showed a delicate blue or pink hue, depending upon the dye that had been used. The intensity of the coloring of the enamel was

greatest near the dento-enamel junction and decreased toward the enamel surface.

(b) *Introduction of Dyes Directly Into the Pulp Tissue.*— Since any supposed circulation in the hard tissues, dentin and enamel, must necessarily be controlled and regulated by the pulp, the expectation would be that, if such a circulation actually exists, minute particles of dye would be carried by the fluid stream from the pulp into the dentin or enamel. Fish, who during recent years published a number of ex-

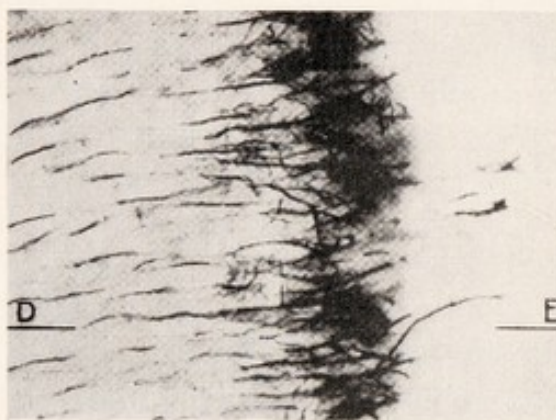


FIG. 10.—Marginal lymph plexus of the dentin of a dog. Solid dye was placed in the pulp tissue of the living tooth and sealed there for twelve hours. The anastomosis of the terminal branches is seen to form a marginal lymph plexus for the exchange of lymph between the tubules. D, dentin; E, enamel. (Fish, *British Dent. Jour.*)

periments on this subject, demonstrated such a vital transport of dyes from the pulp into the dental hard tissues. He used dogs and monkeys for his investigations. Under general anesthesia a hole was drilled into a tooth, carefully exposing the pulp. Solid methyl blue was then placed very gently upon the pulp, and the cavity was sealed without pressure. After twenty-four hours the tooth was extracted and sections were prepared. Fig. 10 shows the result of such an experiment in a dog's tooth. The dentinal tubules are stained all the way to the dento-enamel junction, where they form a network of anastomosing terminal branches, the marginal plexus (see Fig. 6). In some places the dye can be seen entering the enamel. Similar results were obtained in the teeth of a monkey. Here, too, some of the dentinal tubules were stained to the dento-enamel junction; however, the enamel appeared free from stain.

In dogs' teeth, if ground sections at right angles to the course of the enamel prisms are prepared, the prism sheaths appear stained. In a higher magnification of this area, the hexagonal enamel rods, the prism sheaths, and the calcified interprismatic substance can be seen plainly. In three places the dye that had been placed into the pulp stained the prism sheaths; the prism itself as well as the interprismatic substance is free from the dye (Fig. 11).

In cross-sections through stained dentinal tubules of dogs' teeth, Fish found the dye between the wall of the tubules and the Tomes' fiber; the dye appeared as a ring around the dentinal fibril, which would indicate that it is located in the capillary lymph space around the odontoblastic process, in the perifibrillar space.



FIG. 11.—Transverse section of dog's enamel prisms. Methyl blue was placed in the pulp of the dog's tooth twenty-four hours before death. The dye can be seen in some of the prism sheaths. The prisms and the interprismatic substance are free from dye. (Fish, Jour. Am. Dent. Assn.)

An important observation made by Fish in the course of his experiments was that the permeability of dogs' enamel is dependent largely upon the age of the animal. Whenever methyl blue was placed upon a vital pulp, it was always found in the enamel after twenty-four hours had elapsed; however, in young dogs the dye entered the enamel far more freely and deeply than it did in older dogs. In very young animals the dye reached the enamel surface and the cuticle, while in older animals the dye in the enamel was confined to small areas near the dento-enamel junction. In all cases the dye was confined to the prism sheaths.

Fish also found that, by cementing a metal cap containing methyl blue for twenty-four hours upon the tip of an intact dog's tooth, the tooth substance could be stained from without inward. In young animals the dye penetrated the entire thickness of the enamel along the prism sheaths and entered the dentin, while in very old

dogs there usually was no penetration. Identical results were obtained when postmortem material of dogs' teeth was used, although in this case it took much longer, about a week, to obtain the same amount of penetration that in living dogs could be obtained in twenty-four hours.

The experiments of Fish, some of which have been described, suggest the existence of a lymph stream from the dog's pulp into the dentin and occasionally into the enamel. Although the dentin of dead, extracted teeth stains similarly if dye has been placed in the pulp chamber, this staining occurs very much more slowly than if the dye has been placed upon a living pulp. This would indicate that a vital force plays a rôle in the staining process.

It has been shown also that the vital staining process occurs much more extensively in young teeth than in old ones, which fact is in full accordance with the findings of Beust concerning the gradual obliteration of the dentinal tubules in advancing age.

To what extent the experimental findings of Fish and other investigators are applicable to man has not yet been decided. However, clinical as well as experimental evidence points very strongly toward the assumption that, while growing human enamel may be affected by diet and other influences, the fully formed enamel of an adult human tooth is void of any vital processes. Certain clinical manifestations, such as changes in the susceptibility to dental caries, must then be accounted for by environmental influences (bacteria, saliva, etc.) rather than by changes within the enamel itself.

Up to this time we have had no scientific evidence for the assumption that there is a lymph or fluid stream in the fully calcified human enamel. Even though occasionally traces of substances or solution, pigmentation or discoloration may pass from the pulp chamber into the enamel by diffusion, these observations by no means justify the far-reaching conclusions that have been drawn by some of the men who believe in the existence of metabolic processes in the enamel. The theory of an "active defense" of the enamel against bacterial activity is not based upon any clinical or laboratory data. On the contrary, all the evidence is against such a theory. If the enamel really were able to produce a defense reaction, pulpless teeth would be relatively more subject to dental decay than teeth with vital pulps. No such difference has been recorded so far. If a lymph circulation were present in the enamel which could ward off dental caries, such a lymph stream would, of course, depend largely upon the permeability of the dentin. The investigations of

Beust and Fish have definitely established the fact that the permeability of the dentin steadily decreases with advancing age. Consequently there is an abundant lymph supply in the dentin of young teeth and a greatly reduced amount in older ones. Still, we find the highest incidence of dental caries in youth and a steadily decreasing incidence with advancing age, which is just the opposite of what should logically be expected if dentinal lymph circulation had any relation to caries incidence.

BIBLIOGRAPHY.

- AMBERSON, W. R.: The Permeability of Enamel, *Dental Cosmos*, 1927, **69**, 638.
- APPLEBAUM, EDMUND: Lymph Channels in the Dentin and Enamel Stained by Amalgam, *Jour. Dent. Res.*, 1929, **9**, 487.
- Concerning the Permeability of Human Enamel, *Jour. Dent. Res.*, 1931, **11**, 611.
- BEUST, TH. B.: A Contribution to the Study of Immunity to Dental Caries, *Dental Cosmos*, 1912, **54**, 659.
- Morphology and Biology of the Enamel Tufts, with Remarks on Their Relation to Caries, *Jour. Am. Dent. Assn.*, 1932, **19**, 488.
- BLACK, G. V., and MCKAY, F. S.: Mottled Teeth: An Endemic Developmental Imperfection of Teeth Heretofore Unknown in Literature of Dentistry, *Dental Cosmos*, 1916, **58**, 129, 477, 627, 781, 894.
- BLOTEVOGEL, W.: Der vitale Farbstofftransport während der Zahnbildung, *Vrtljschr. f. Zhk.*, 1924, **40**, 185.
- BOEDECKER, C. F.: Permeability of the Enamel in Relation to Stains, *Jour. Am. Dent. Assn.*, 1923, **10**, 60.
- A New Theory of the Cause of Dental Caries, *Dental Cosmos*, 1929, **71**, 586.
- BOEDECKER, C. F., and APPLEBAUM, EDMUND: Metabolism of the Dentin; Its Relation to Dental Caries and to the Treatment of Sensitive Teeth, *Dental Cosmos*, 1931, **73**, 995.
- CAHN, L. R.: Notes on the Histology of the Dental Pulp, *Dental Items Int.*, 1931, **53**, 668.
- CHASE, SAMUEL W.: A Critical Review of the Controversy Concerning Metabolism in the Enamel, *Jour. Am. Dent. Assn.*, 1931, **18**, 697.
- Histogenesis of the Enamel, *Jour. Am. Dent. Assn.*, 1932, **19**, 1275.
- DEPENDORF, TH.: Ergebnisse eigener Untersuchungen über Innervierung des menschlichen Zahnes mit Berücksichtigung der Hartsubstanzen, *D. Mon. f. Zhk.*, 1913, **31**, 377, 570, 689.
- DEWEY, K., and NOYES, F. B.: A Study of the Lymphatic Vessels of the Dental Pulp, *Dental Cosmos*, 1917, **59**, 436.
- DIECK, W.: Über die alte Streitfrage der Existenz oder Nichtexistenz von Nervenfasern im menschlichen Zahnbein und ihre positive Lösung, *Korr. f. Zahnärzte*, 1927, **51**, 138.
- FABER, FRITZ: Das organische Gewebe des menschlichen Zahnschmelzes, *Ztschr. f. Anat. u. Entw.*, 1928, **86**, Nos. 1 and 2.
- Die Frage der Vitalität des menschlichen Zahnschmelzes im Lichte der neueren Forschung, *Ztschr. f. Stom.*, 1929, **27**, 530.
- Histologie, *Fortschr. d. Zhk.*, 1932, vol. **8**, No. 3.
- FISH, E. W.: Circulation of Lymph in the Dentinal Tubuli with Some Observations on the Metabolism of the Dentine, *Proc. Roy. Soc. Med.*, 1926, **19**, 59.

- FISH, E. W.: The Circulation of Lymph in Enamel and Dentin, *Jour. Am. Dent. Assn.*, 1927, **14**, 804.
- The Physiology of the Dentine and Its Reaction to Injury and Disease, *Brit. Dent. Jour.*, 1928, **49**, 593.
- A Physiological Survey of the Nature of Enamel in Carnivora and Primates, *Dental Rec.*, 1928, **48**, 289.
- Age Changes in the Permeability of Dog's Enamel, *Jour. Physiol.*, 1931, No. 3, p. 321.
- GIES, W. J.: Studies of Internal Secretions in Their Relation to the Development and Condition of the Teeth, *Jour. Nat. Dent. Assn.*, 1918, **5**, 527.
- GOTTLIEB, B.: Experimentelle Untersuchungen über den Kalkstoffwechsel in den Zahngeweben mittels vitaler Färbung, *Vrtljschr. f. Zhk.*, 1913, **29**, 470.
- HANAZAWA, KANAE: A Study of the Minute Structure of Dentin, Especially of the Relation Between the Dentinal Tubules and Fibrils, *Dental Cosmos*, 1917, **59**, 125.
- HOPEWELL-SMITH, A.: Concerning Human Enamel. Facts, Explanations and Applications, *Dental Cosmos*, 1926, **68**, 639.
- KLEIN, H., and AMBERSON, W. R.: A Physico-chemical Study of the Structure of Dental Enamel, *Jour. Dent. Res.*, 1929, **9**, 667.
- LEIGH, R. W.: Staining of Human Enamel in Bulk, *Jour. Am. Dent. Assn.*, 1925, **12**, 1415.
- McKAY, FREDERICK, S.: Present Status of Investigation of Cause, and of Geographical Distribution, of Mottled Enamel, Including a Complete Bibliography on Mottled Enamel, *Jour. Dent. Res.*, 1930, **10**, 561.
- Fluorin Content of Certain Waters in Relation to the Production of Mottled Enamel, *Jour. Am. Dent. Assn.*, 1932, **19**, 1715.
- MAXIMOW, A. A.: Morphology of the Mesenchymal Reactions, *Arch. Path. and Lab. Med.*, 1927, **4**, 557.
- Bindegewebe und Blutbildende Gewebe, Möllendorff's Handb. d. mikr. Anat., Berlin, Springer, 1927, **2**, 232.
- MEYER, W.: Über die sogenannten Interglobularräume des Dentins, *Deutsch. Mon. f. Zhk.*, 1925, **43**, 175.
- Die feinere Histologie der Dentinkanälchen, *Deutsch. Mon. f. Zhk.*, 1926, **44**, 649.
- Schmelzlamellen und Schmelzbüschel, *Deutsch. Mon. f. Zhk.*, 1926, **44**, 750.
- MUMMERY, I. H.: The Innervation of Dentin, *Dental Cosmos*, 1916, **58**, 258.
- The Nerve Supply of the Dentin, *Proc. Roy. Soc. Med.*, 1924, **17**, 35.
- NOYES, F. B.: A Review of the Work on the Lymphatics of Dental Origin, *Jour. Am. Dent. Assn.*, 1927, **14**, 714.
- ORBAN, B.: Schmelz- und Zahnoberhäutchen. Schmelzlamellen und Büschel, *Ztschr. f. Stom.*, 1926, **24**, 1.
- Histology of the Enamel Lamellæ and Tufts, *Jour. Am. Dent. Assn.*, 1928, **15**, 305.
- Contribution to the Histology of the Dental Pulp and Periodontal Membrane, with Special Reference to the Cells of "Defence" of These Tissues, *Jour. Am. Dent. Assn.*, 1929, **16**, 965.
- The Development of the Dentin, *Jour. Am. Dent. Assn.*, 1929, **16**, 1547.
- PROELL, F.: Vitalfärbung bei Tieren zum Studium der Verkalkungs- und Stoffwechsel-Vorgänge in Kiefern und Zähnen, *Vrtljschr. f. Zhk.*, 1927, **43**, 467.
- RADOSEVIC, E.: Das Grundprincip des Stoffwechsels im Zahne, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 145.
- SCHOUR, I.: A Review of Maximow's Research on Inflammatory Reaction, *Jour. Am. Dent. Assn.*, 1930, **17**, 1605.

- SMREKER, E.: Über Injektion des Schmelzes durch die Zahnbeinkanälchen, *Ztschr. f. Stom.*, 1926, **24**, 460.
- TOJODA, MINORU: Beiträge zur Kenntnis der Dentinverkalkung, *Korr. f. Zahnärzte*, 1926, **50**, 374.
- URBANTSCHITSCH, E. H.: Ursachen und Bahnen der endogenen Zahnverfärbungen. I. Die Ursachen, *Ztschr. f. Stom.*, 1926, **24**, 1050; II. Die Bahnen, 1927, **25**, 19, 789, 1054.
- WALKHOFF, O.: Die Nervenfrage im Zahnbein, *Deutsch. Zhk.*, 1923, vol. 60.
- WILLIAMS, T. L.: Mottled Enamel and Other Studies of Normal and Pathological Conditions of This Tissue, *Jour. Dent. Res.*, 1923, **3**, 117.
- Can We, by a Change in Food Habits, Change the Structure of Formed Enamel? *Dental Cosmos*, 1927, **69**, 590.
- WOLF, H.: Ein Beitrag zur Histologie der Zahnbeinfaser, *Ztschr. f. Stom.*, 1931, **29**, 584.

CHAPTER II.

REGRESSIVE CHANGES IN PULP AND DENTIN.

BEFORE entering into the pathology of the dental tissues a group of conditions will be discussed for which the term regressive changes will be used. These conditions are: the formation of secondary or irregular dentin, the atrophy of the pulp tissue with advancing age, and the formation of calcified bodies in the pulp tissue.

These deviations from the normal histology of dentin and pulp are, in time, invariably formed in every human tooth, and, therefore, must be considered separately from distinctly pathological conditions such as caries or pulpitis. The regressive changes in dentin and pulp take place without any clinical symptoms, developing gradually and without interference with the life or the function of the tooth. They can, therefore, be considered as an expression of the general senescence of the organism, and also to a certain extent as a defense mechanism against the many outer influences to which every tooth is exposed during life.

SECONDARY (IRREGULAR) DENTIN.

In a young tooth with an intact covering of enamel, dentin formation occurs in a regular, undisturbed manner and results in the formation of dentinal tubules that converge toward the pulp in a uniform, wavy course. This type of dentin is called primary or regular dentin, because it is the dentin that is formed in the early part of the life of every tooth, and because of the regular course of the dentinal tubules.

Sooner or later after the tooth has erupted and has come into function, the enamel is worn through by mastication, and the dentin becomes exposed to the influences of the oral cavity. Exposure of the primary dentin is accompanied by injury to the peripheral ends of the 'Tomes' fibers which transmit impulses to the pulp. The defensive reaction of the pulp to this irritation consists of a hurried deposition of calcified tissue over the central endings of the injured dentinal tubules. This newly formed hard substance is the secondary dentin. The course of the dentinal tubules is much less regular

in the secondary than in the primary dentin, and, therefore, this type is also called the irregular dentin. The secondary dentin contains less organic substance than the primary dentin and is less permeable, in which way it is better equipped to perform its function of protecting the pulp against injury or irritation from without. Due to the difference between the structure of primary and secondary dentin, it is easy to find the borderline between the two kinds in microscopic specimens (Figs. 12 and 13).



FIG. 12.



FIG. 13.

FIGS. 12 and 13.—Diagrams of the distribution of secondary dentin in an upper bicuspid as the result of abrasion. The worn portion of enamel and dentin is indicated by dotted lines. The secondary dentin is indicated by black shading.

FIG. 12.—Beginning abrasion. The dentin is exposed at the tips of both cusps. Corresponding to the exposed area, secondary dentin is formed in the pulp horns.

FIG. 13.—Advanced abrasion. A large portion of the dentin is exposed. The occlusal portion of the pulp chamber is filled by secondary dentin.

From a clinical viewpoint it seems advisable to describe separately the different factors that lead to the formation of secondary dentin, keeping in mind, however, that the secondary dentin produced under these various clinical circumstances is not different under the microscope.

The following factors lead to secondary dentin formation:

1. Abrasion (attrition, occlusal wear).
2. Dental caries.
3. Dental operations that involve the dentin.
4. Fracture of a part of the crown without exposure of the pulp.

1. **Abrasion and Secondary Dentin.**—In a mouth with a full row of upper and lower teeth in normal alignment, the incisal edges and occlusal surfaces of all crowns are exposed to a great amount of wear. This is especially true in healthy people with strong, sound teeth who make good use of their jaws. In such persons, usually between ten and fifteen years after the permanent teeth have come into full occlusion, it will be found that the enamel has been ground

down considerably and that facets have been formed. In the fourth decade of life, as a rule, this process of abrasion reaches the tip of the dentin, first at the incisal edge of the incisor teeth and at the cusps of the first molars. The process of wearing down continues until, on examining, for example, the occlusal surfaces of a man in the fifties with strong, healthy teeth, all cusps have practically disappeared and the dentin has been exposed on almost every tooth, appearing as a yellow or brown area on the occlusal surface, surrounded by a white seam of enamel.

Teeth without cusps must be considered the natural tooth form for older people. This point should be noted especially, as there still are dentists who consider every evidence of abrasion as pathological or as a symptom of injury to the tooth. Although Nature has equipped man with only one set of permanent teeth that is supposed to last for a lifetime, these teeth need not necessarily retain the form and shape that they had at the time of their eruption. The human teeth are hard working tools, and since there is no possibility of replacing that part of the crown that is lost by constant wear, the teeth, like all other tools, will in time become shorter. This process is known as physiological abrasion. If no pathological changes interfere, if there is no loss of teeth by caries or pyorrhea, abrasion will not become excessive before the natural limit of life is reached.

Unfortunately in modern man physiological abrasion is a rare occurrence since abnormal alignment, insufficient use, and dental caries among our civilized races preclude proper occlusal wear. It is almost necessary to go back to the teeth of primitive men to find what we might call the ideal form of abrasion: a perfect set of thirty-two teeth, with uniform wear-down of the cusps, elimination of incisal overbite, and creation of an occlusal plane in which the lower jaw can move and grind in every direction without cusp interference. This type of abrasion occurs so regularly in primitive man that anthropologists use the amount of occlusal wear as an important factor in determining the age of the individual.

These considerations may raise the question as to whether or not abrasion is ever pathological. There is, of course, such a thing as pathological abrasion. Every dentist has seen older people with excessively abraded front teeth, worn down almost to the gingivæ, and sometimes even with exposed pulps. But in these cases the pathological abrasion is merely the secondary result of another preëxisting pathological condition, such as the loss of molars, malalignment or malocclusion of the teeth, or abnormal chewing habits.

Abrasion found under such circumstances cannot be compared with that occurring in a normal set of teeth without disturbances in number or alignment.

The process of abrasion is always accompanied by the formation of secondary dentin as soon as it reaches the dento-enamel junction. The amount of secondary dentin deposited depends upon several factors, namely, the extent of the exposed dentin area, the length of time that has passed since the dentin was exposed, and the individual reaction of the pulp. Some pulps seem to have a greater and others a lesser tendency toward the formation of secondary dentin.

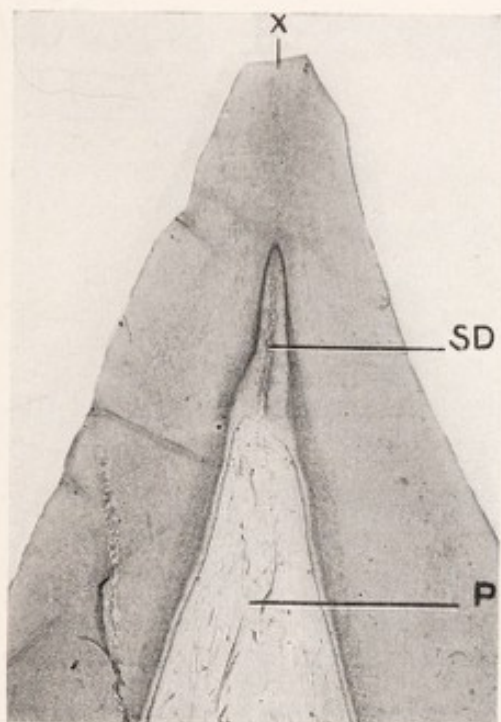


FIG. 14.—Beginning abrasion. Lower cuspid. Decalcified section. X, abraded area of dentin; SD, secondary dentin in the tip of the pulp chamber; P, pulp. (Courtesy of W. Willman.)

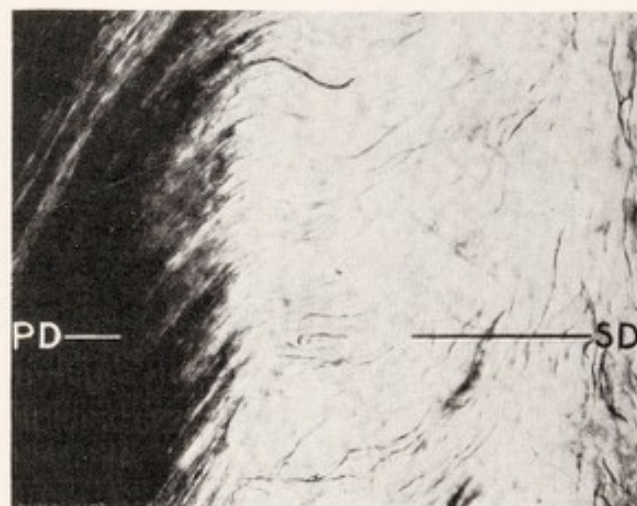


FIG. 15.—Borderline between primary and secondary dentin. Ground section. PD, primary dentin; SD, secondary dentin. Note the small number and irregular course of the dentinal tubules in the secondary dentin. Most of the dentinal tubules of the primary dentin end at the border line between primary and secondary dentin.

The actual distribution of secondary dentin in abraded human teeth will be illustrated by a few typical cases. Fig. 14 shows the tip of the crown of a lower cuspid with slight abrasion, in which the dentin was just exposed. The specimen was decalcified in preparation, and, therefore, the enamel has disappeared; the abraded plane of dentin can be seen running approximately at right angles to the long axis of the tooth. The tip of the original pulp chamber, which has been filled in by secondary dentin, is still indicated by a dark line dividing primary and secondary dentin.

The main difference between primary and secondary dentin lies, as already mentioned, in the different course and number of the

dentinal tubules. This can be seen plainly in Fig. 15, which was taken of a stained ground section. The regular, parallel, dentinal tubules of the primary dentin end abruptly at a certain line; from there on only a very few irregular tubules are present, running through the solid, calcified dentin matrix.

The formation of secondary dentin is especially important when abrasion reaches the level of the original pulp horns (Fig. 16). From this specimen it becomes evident that were it not for the secondary dentin, the pulp would have been exposed. As abrasion advances a considerable portion of the original pulp chamber which has been

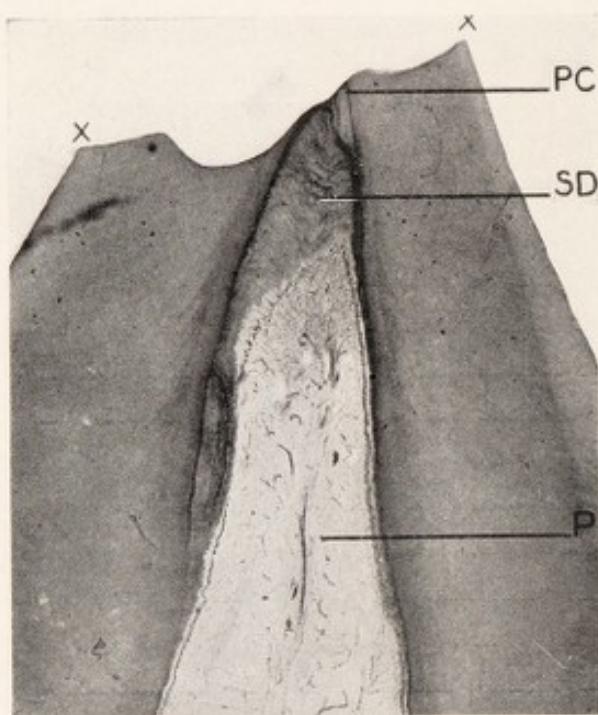


FIG. 16.—Abrasion. Lower cuspid. At PC the dentin is worn down to the level of the original pulp chamber. X-X, abraded dentin surface; PC, outline of the original pulp chamber; SD, secondary dentin; P, pulp. (Courtesy of W. Willman.)

filled with secondary dentin may be worn away (Fig. 17). But as long as this process of wearing away occurs gradually and there is enough time for secondary dentin to form, there is no danger of infection or exposure of the pulp.

In old people sometimes almost the entire pulp chamber may be filled by secondary dentin (Fig. 18). This fact is important because teeth in which the pulp ends apically from the cemento-enamel junction do not necessarily respond to the vitality test; dental operations, such as grinding or drilling, usually painful in teeth with vital pulps, can be performed on such old teeth with little sensation.

The formation of secondary dentin must be considered as a reaction of great efficiency and of highest practical importance, because by this process a sufficient thickness of hard substance is constantly maintained between the tooth surface and the pulp. Furthermore, the newly formed hard substance is denser than primary dentin and contains less organic material, qualities that increase its ability to seal the pulp against outer influences.

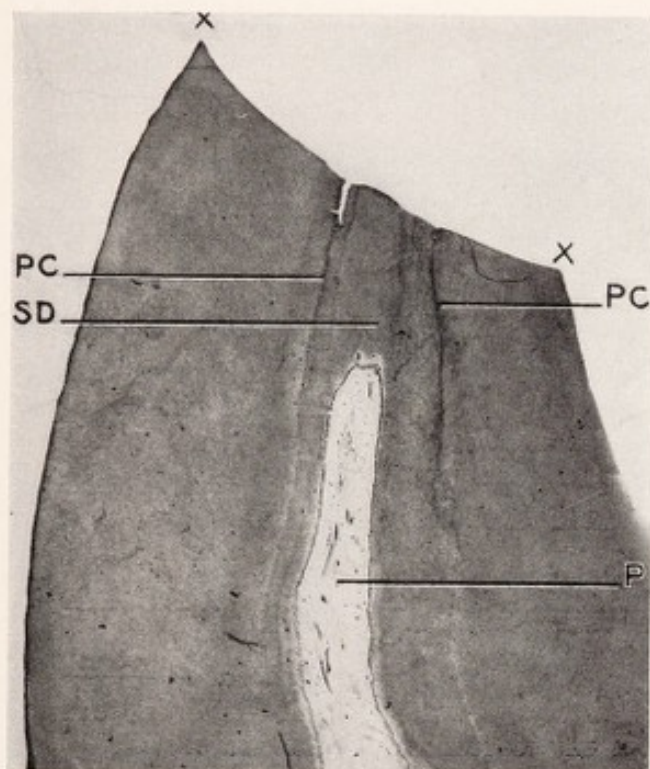


FIG. 17.—Advanced abrasion. Lower cuspid. X-X, abraded dentin surface; PC, original outline of pulp chamber; SD, secondary dentin; P, pulp. (Courtesy of W. Willman.)

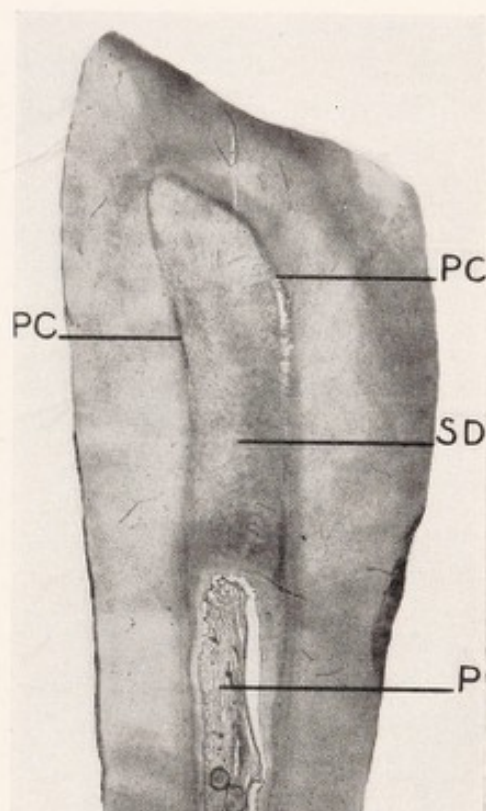


FIG. 18.—Advanced obliteration of the pulp chamber by secondary dentin. Lower incisor of an old individual. PC, original outline of pulp chamber; SD, secondary dentin; P, pulp. (Courtesy of W. Willman.)

The immediate stimulus causing the formation of secondary dentin seems to be the injury to the odontoblastic processes in the dentinal tubules. This opinion is greatly supported by the observation that the secondary dentin is formed only at the central endings of those dentinal tubules of which the periphery has been exposed. Fish regards the deposition of secondary dentin as a specific reaction of the dentinal tubules to injury. This author has called attention to the fact that two phases can sometimes be distinguished in the formation of secondary dentin. Immediately following injury to the protoplasmic process a "barrier" of calcium salts is deposited

over the central opening of every injured tubule; upon this barrier the actual secondary dentin is formed. The tubules in the primary and secondary dentin are thus completely separated by a highly calcified obstruction, and the primary dentin is cut off from the pulp. Clinically this is manifested by a loss of sensitivity in the involved portion of the dentin. Fish calls the tract of dentin between the peripheral lesion and the secondary dentin a "dead tract." He shows that if methyl blue dye is introduced into the pulp chamber of an extracted human tooth, the dye enters the dentinal tubules of the intact primary dentin and penetrates to the dento-enamel junction, but wherever secondary dentin covers the central endings of the tubules, no dye can enter and the dentin remains unstained.^{1,2}

Occlusal abrasion must not be confused with the loss of tooth substance that is sometimes found at the neck of teeth on the labial or buccal side. These defects, which are called erosions, are usually wedge-shaped and cut deep into the substance of the tooth. They are due to the improper use of the tooth-brush together with a sharp, gritty dentifrice. Under such erosions secondary dentin may develop, its presence depending upon the rate of destruction and upon the defensive power of the pulp.

The pulp tissue under secondary dentin shows certain characteristic changes. As a result of the injury to its protoplasmic processes, the entire odontoblastic layer may die and be covered over by secondary dentin, in which event the newly formed hard substance will consist of a structureless, calcified mass without any dentinal tubules. However, some of the odontoblasts generally survive, leaving their protoplasmic extensions tortuously and irregularly embedded in the secondary dentin. The number of odontoblasts is always greatly reduced; they are smaller than usual and their arrangement is irregular (Fig. 19).

Due to the continuous formation of primary dentin and to the gradual development of secondary dentin, the size of the pulp chamber of all human teeth slowly and steadily decreases with advancing age. In the anterior teeth the deposition of secondary dentin leads to a considerable reduction of the length of the pulp chamber; at the same time the root canal becomes narrower. Since

¹ Brit. Dent. Jour., 1932, 53, 351.

² While this book was being printed, the investigations of Fish were published in book form. (Fish, E. W.: *An Experimental Investigation of Enamel, Dentin and the Dental Pulp*, London, John Bale, Sons & Danielsson, 1932.) His book, which represents an outstanding piece of work on the permeability of the dental hard tissues, on the metabolic changes in the dentin and on the reaction of dentin and pulp to peripheral injuries of all kinds, can be highly recommended.

these changes are of great practical importance they will be illustrated by comparing labio-lingual sections through upper central incisors at four different ages (Figs. 20 to 23). Fig. 20 illustrates a section through an incisor shortly after eruption at the age of eight years. The pulp chamber is very large; the root end is wide open. Fig. 21 shows a section through the corresponding tooth of a boy, aged fourteen years. The root end is fully developed. No incisal abrasion has yet taken place, and, therefore, only primary dentin is present. In Fig. 22 the corresponding section of an individual, aged thirty-five years, is reproduced. The process of abrasion has reached the dentin at the incisal edge; secondary

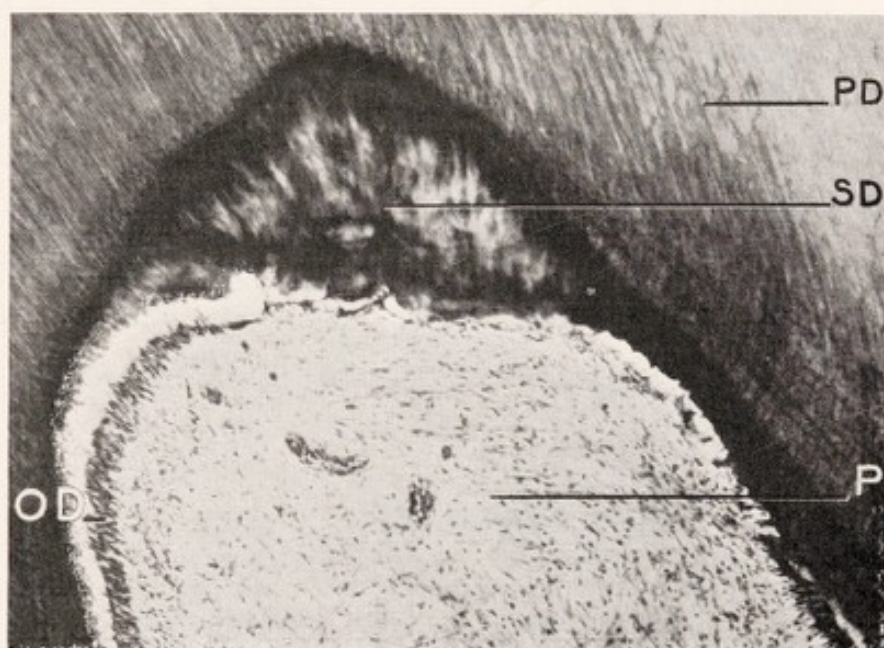


FIG. 19.—Secondary dentin formed in a pulp horn of a lower molar as the result of abrasion. On the surface of the secondary dentin the odontoblasts are small, irregular, and in places entirely missing. PD, primary (regular) dentin; SD, secondary (irregular) dentin; OD, odontoblasts; P, pulp.

dentin has been formed, causing a considerable decrease in the size of both pulp chamber and root canal. In Fig. 23 the same tooth is illustrated in a man, aged about fifty-five years. Notice the advanced abrasion. The crown has been worn flat. The pulp chamber has been reduced to a fine, canal-like space; more than two-thirds of the original length of the pulp chamber has been obliterated by secondary dentin.

2. Dental Caries and Secondary Dentin.—When caries reaches the dento-enamel junction it causes the formation of secondary dentin on the wall of the pulp chamber in the same way as was shown in the case of abrasion. By tracing the course of the dentinal tubules

from the carious lesions to the pulp, it is obvious that the secondary dentin corresponds exactly to the decayed area in extent and localization. The lack of tubules in the secondary dentin may be considered a protective measure against a possible invasion of the dentin by bacteria.



FIG. 20.



FIG. 21.



FIG. 22.



FIG. 23.

FIGS. 20 to 23.—Size of the pulp chamber. Comparison of labio-lingual sections through upper central incisors of different ages.

FIG. 20.—Aged eight years. Open root end; very wide pulp chamber and root canal.

FIG. 21.—Aged fourteen years. Root fully formed. No abrasion; no secondary dentin formation.

FIG. 22.—Aged about thirty-five years. Beginning abrasion; secondary dentin in the incisal portion of the pulp chamber.

FIG. 23.—Aged about fifty-five years. Advanced abrasion. The larger part of the original pulp chamber has become obliterated by secondary dentin. The root canal is very narrow.

In Fig. 24 is shown a mesio-distal section through a lower molar with both occlusal and mesial caries. Two distinct deposits of secondary dentin are present on the wall of the pulp chamber, one on the roof, which corresponds to the occlusal caries, and another one on the mesial wall. Several calcifications (denticles) can be seen in the pulp tissue.

The combined influence of abrasion and caries produces conditions like the one illustrated in Fig. 25. In a lower incisor with mesial and distal caries, formation of secondary dentin has taken place on the mesial and distal walls of the pulp chamber; rootward

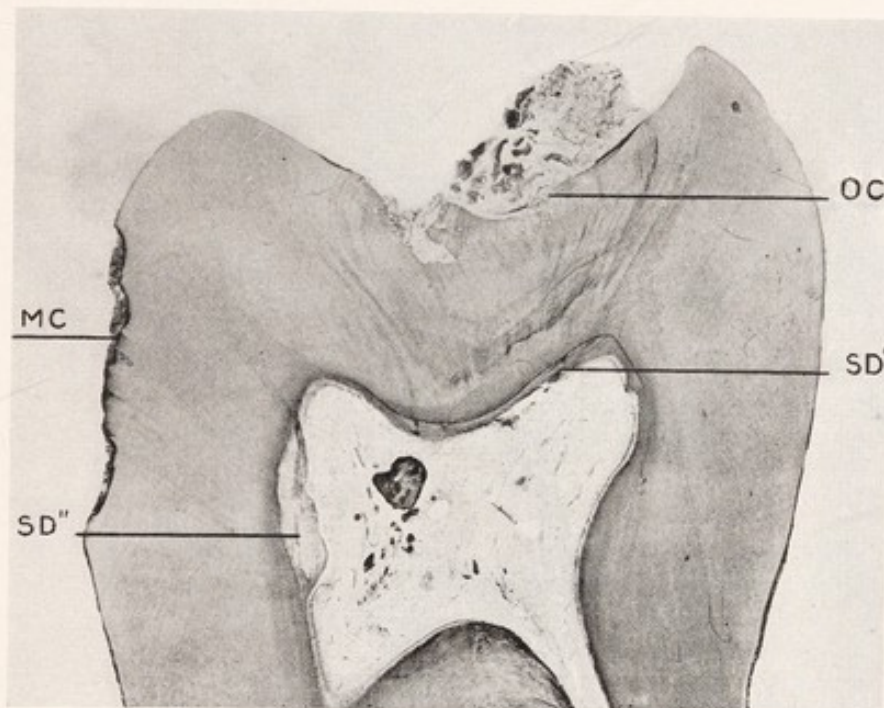


FIG. 24.—Secondary dentin formation under caries. Mesio-distal section through lower molar with occlusal (OC) and mesial (MC) caries. SD', secondary dentin corresponding to the occlusal decay; SD'', secondary dentin corresponding to the mesial decay. The dentinal tubules leading from the mesial decay to the pulp chamber converge toward the pulp; therefore, the area of secondary dentin formation is smaller than the carious area of the dentin surface. (Coolidge, Int. Dent. Congress; Illinois Dent. Jour.)

the secondary dentin ends rather abruptly. In tracing the dentinal tubules from here back to the surface of the dentin, a point is reached exactly at the apical end of the dental caries. In addition to the deposits on the mesial and distal wall of the pulp chamber, secondary dentin is present at the incisal end of the pulp chamber; this is evidently due to the incisal abrasion that occurred in this tooth. The original partition of the incisal end of the pulp chamber into three horns, corresponding to the three small incisal cusps of the erupting tooth, is still visible and marks the borderline between primary and secondary dentin.

It might be appropriate at this point to discuss in general the relationship between caries and secondary dentin formation. In the specimens illustrated here the pulp responded readily to approaching caries with the formation of secondary dentin. This is corroborated clinically by the observation of teeth with advanced caries, in which, after excavating the softened dentin, the pulp has been found intact and well protected by a hard, brown layer of secondary dentin. On the other hand, it is known from clinical experience that caries often reaches the pulp despite secondary dentin forma-

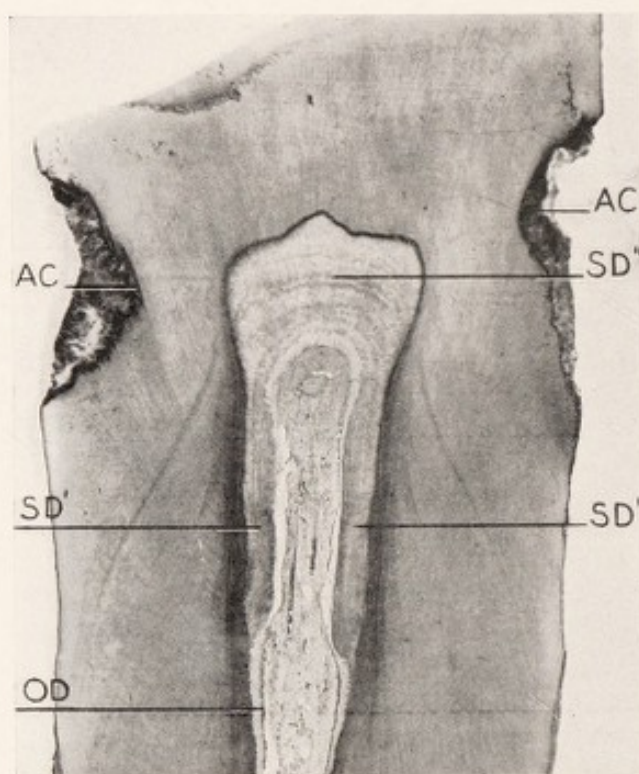


FIG. 25.—Secondary dentin under caries and abrasion. Mesio-distal section through lower incisor. AC, approximal caries; SD', secondary dentin corresponding to the approximal caries; SD'', secondary dentin corresponding to the incisal abrasion; OD, odontoblasts rootward from the secondary dentin. (Coolidge, *Int. Dent. Congress; Illinois Dent. Jour.*)

tion. How can this different clinical outcome of apparently the same process be accounted for? Two factors play an important rôle here, namely, the rate of speed with which caries progresses and the individual reaction of the pulp. As far as the first factor is concerned, it must be kept in mind that it takes anywhere from a few weeks to several months for the formation of secondary dentin of considerable thickness. Therefore, if the decay is very rapid, as, for instance, in case of fissure caries in the first permanent molars in children, the pulp will hardly have time to form sufficient secondary dentin to ward off the rapidly spreading infection and

decomposition of the dentin. If, on the other hand, the progress of caries is slow and superficial, sufficient secondary dentin will be formed to keep up with the advancing decay, and the pulp will not be exposed.

The second factor involved, namely, the individual reaction of the pulp, is not yet understood. Just why one pulp will form a large mass of secondary dentin and, as a result, remain intact, whereas another pulp will produce little defensive reaction of this type and will become infected or exposed, is one of the many problems of the biological sciences that is still awaiting solution. In the meantime, the only thing that can be said is that one pulp has greater resistance than another; but the factors that determine or regulate this individual variation are unknown.



FIG. 26.—Caries on the distal side of a lower bicuspid. Deposition of secondary dentin on the mesial wall of the pulp chamber. CD, carious and decomposed dentin; SD, secondary dentin; P, pulp. The pulp tissue is normal. The line X-X indicates the approximate outline of a cavity preparation that would remove all decayed dentin and leave a layer of dentin of uniform thickness sufficient to protect the pulp.

3. Dental Operations and Secondary Dentin.—Every drilling or grinding of a tooth will lead to the formation of secondary dentin provided this operation involves the dentin. The extent and amount

of the deposition of secondary dentin, under these circumstances, will depend upon the size and the depth of the cavity, upon the age of the individual, upon the time that has elapsed since the operation was performed, and upon the individual reaction of the pulp. The larger the area of dentin that is exposed, the more secondary dentin will be deposited; the younger the individual, the more readily will the pulp react with such a deposition; and, finally, the more responsive the individual pulp to irritating stimuli, the more readily and extensively will secondary dentin develop.



FIG. 27.—Diagram of the distribution of secondary dentin following the preparation of a mesio-occlusal cavity and insertion of a filling in a lower molar. The secondary dentin is indicated by black shading.

In cavity preparation a certain amount of secondary dentin is usually present as the result of the existing caries (Fig. 26); however, since it is necessary to enlarge the cavity to comply with the laws of extension, new areas of dentin with intact dentinal tubules are cut open by bur or stone, and secondary dentin is formed subsequently at the central ends of these freshly exposed tubules. In other cases, if a large cavity is prepared in a formerly intact tooth that is intended to serve as an abutment, no secondary dentin is present at that time.

The distribution of secondary dentin under an approximo-occlusal metal filling in a lower molar is illustrated diagrammatically in Fig. 27. The area of the pulp chamber occupied by the secondary dentin is determined by the extent of the exposed dentin surface; the thickness of the secondary dentin depends upon the length of time that has elapsed since the filling was inserted and upon the individual pulp reaction.

Another dental operation that is a common cause of secondary dentin formation is the grinding of a tooth with a stone. If, for instance, cusps or incisal edges of teeth are ground off, so that the dentin is exposed, the result will be exactly the same as in tooth abrasion. The difference between the two processes is that abraded teeth usually are not sensitive; whereas, ground-down teeth are often very sensitive to touch and to thermal changes. It seems that, during the comparatively slow process of abrasion, the sensitiveness of the exposed tubules is checked by the gradual process of calcification at their proximal ends. After grinding, however, vital tubules are suddenly exposed on the dentin surface, and, as a result, the dentin is very sensitive until, during the

weeks and months following the grinding, secondary dentin seals the central ends of the involved tubules.

Secondary dentin may be produced experimentally by exposing the primary dentin. It has been observed that secondary dentin formation in the teeth of young dogs begins immediately after a cavity has been drilled; after six weeks a considerable amount of secondary dentin has already been formed. In experiments of this kind it apparently does not make any difference whether such an artificial cavity is filled with any kind of filling material or whether it is left open; the mere fact that dentinal tubules in the periphery have been mechanically injured is the deciding factor that stimulates secondary dentin formation.

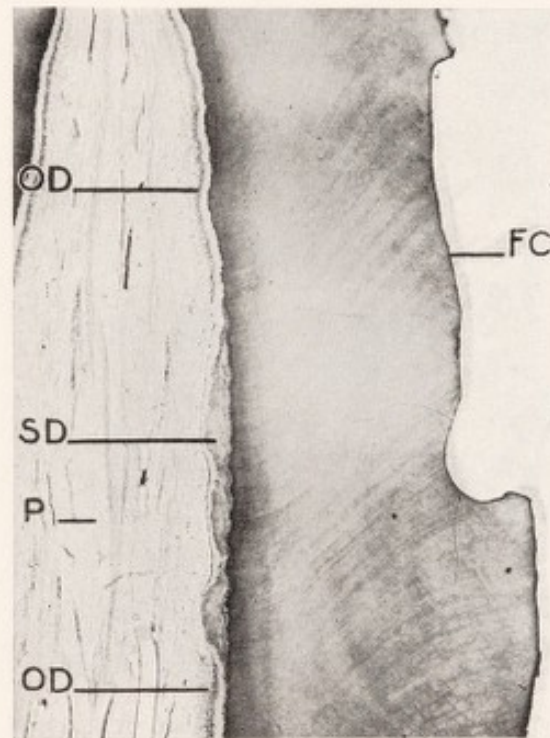


FIG. 28.—Secondary dentin under a shallow metal filling on the labial surface of an upper cuspid. FC, floor of cavity; SD, secondary dentin; P, pulp; OD, odontoblasts. Note the irregularity of the odontoblasts overlying the secondary dentin.

A few specimens of human teeth that carried fillings over a period of several years will be used to illustrate the changes caused by such restorations. Fig. 28 shows a labio-lingual section through an upper cuspid that carried a shallow labial metal filling for one year. Corresponding to the extent of the cavity, secondary dentin has been deposited on the labial wall of the pulp chamber. The odontoblasts overlying the secondary dentin are much smaller and more irregular than the ones on the surface of the primary dentin. The secondary dentin is located slightly rootward from the

floor of the cavity; this is due to the apical deviation of the dentinal tubules in their course from the cavity to the pulp chamber.

Fig. 29 shows a mesio-distal section through a human lower molar. The outline of a mesio-occlusal cavity, in which an amalgam filling had been placed eight years before extraction, can be seen plainly in the specimen. A mesial pulp horn has been completely obliterated by secondary dentin which undoubtedly was already present at the time the cavity was prepared. There is also a deposit of secondary dentin on the roof of the pulp chamber corresponding in form and extent to the occlusal portion of the cavity. On the distal surface of the same tooth caries is beginning at the cemento-enamel junction, causing a circumscribed deposit of secondary dentin on the distal wall of the pulp chamber.

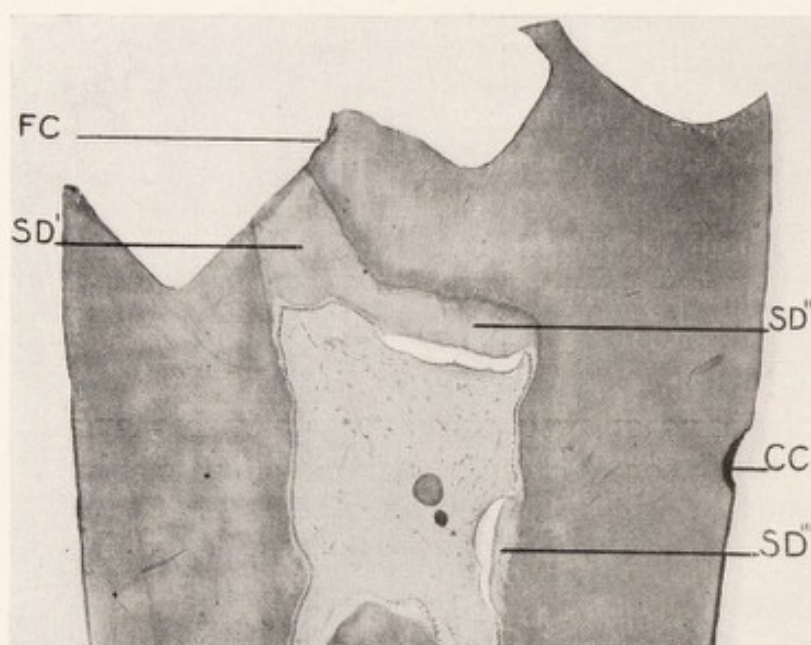


FIG. 29.—Secondary dentin under mesio-occlusal metal filling in lower molar. Filling inserted eight years previous to extraction. FC, floor of cavity; SD', secondary dentin in mesial pulp horn; SD'', secondary dentin corresponding to the occlusal portion of the cavity; CC, cervical caries on the distal surface of the molar; SD''', secondary dentin corresponding to this decayed area. (Coolidge, *Internat. Dent. Congress; Illinois Dent. Jour.*)

In Fig. 30, also a mesio-distal section through a lower molar, the direct causal connection between cavity and secondary dentin is plainly indicated by the extent and arrangement of the latter. The bottom of the cavity almost reaches the outline of the original pulp chamber, and had it not been for the secondary dentin previously formed in this area the pulp undoubtedly would have been exposed.

4. **Tooth Fracture and Secondary Dentin.**—A kick, blow, or fall upon the face may cause a fracture of the edge of an anterior tooth

exposing the dentin. If in such a case the pulp is not exposed and is still protected by sufficient dentin, secondary dentin will be deposited in the pulp chamber corresponding to the position of the line of fracture. The process of secondary dentin formation in a fracture of this type corresponds exactly in etiology to the changes that occur if the dentin is exposed by grinding or drilling. An illustration of secondary dentin formation following tooth fracture is found in Fig. 346.

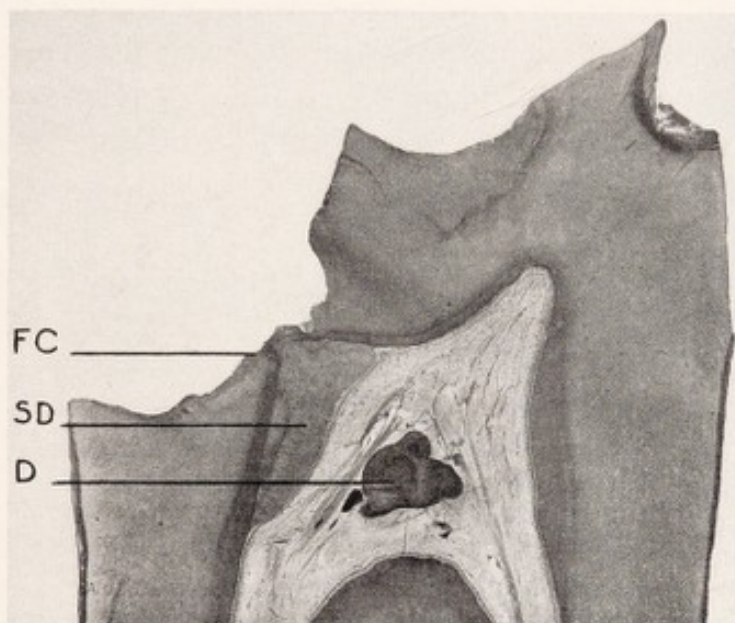


FIG. 30.—Secondary dentin under filling in lower molar. FC, floor of cavity. SD, secondary dentin in the mesio-occlusal corner of the pulp chamber; D, denticle; (Coolidge, Internat. Dent. Congress; Illinois Dent. Jour.)

REGRESSIVE CHANGES IN THE PULP TISSUE.

The term regressive changes is used for certain conditions in the pulp tissue that are usually associated with the formation of secondary dentin and like the latter are a manifestation that stands on the borderline between normal and pathological. Such changes are found invariably in all teeth of older persons; a pulp of the type that is reproduced in Fig. 1 is found in young individuals exclusively and even there only occasionally. Otherwise all human teeth that the author has examined microscopically showed, at least in some places, beginning regressive changes, atrophy of the pulp tissue or degeneration of the odontoblasts.

Regressive changes in the pulp do not cause any trouble or discomfort and can be diagnosed only by the microscope; clinically the tooth may appear perfectly healthy and intact. This is an

important point because here, as in so many problems, the dentist's clinical conception is in opposition to the pathologist's microscopic diagnosis. The dentist would call a tooth normal and healthy if the crown were intact, if the pulp reacted to the electric vitality test, and if there were no evidence of pathological changes in the supporting structures. The pathologist in examining the pulp of the same tooth under the microscope might find marked degenerative changes, vacuolization of the odontoblasts, atrophy and calcification of the pulp tissue, and certainly would not call such a pulp healthy. Still, from the practical point of view the dentist is right in his diagnosis. It seems advisable to bring up these considerations at this point as they prove that close coöperation between practitioner and pathologist is necessary in order to avoid misunderstandings and diagnostic errors.

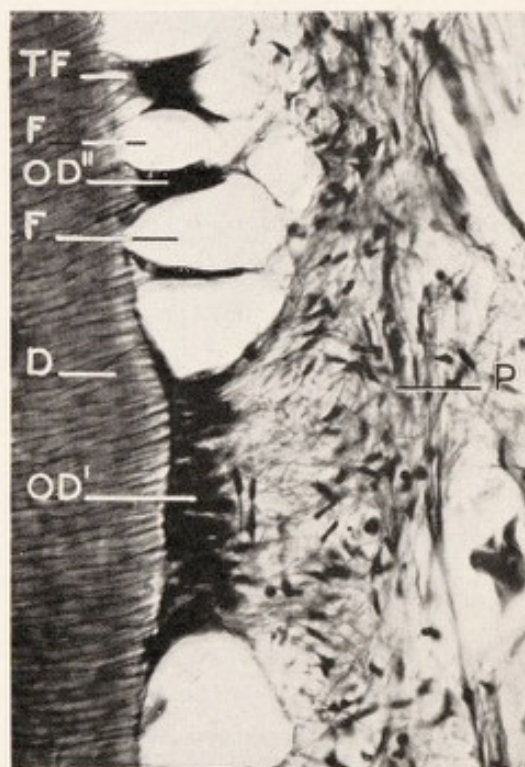


FIG. 31.—Vacuolization of the odontoblastic layer. OD', normal odontoblasts; F, accumulation of fluid between the odontoblasts; OD'', groups of odontoblasts between the vacuoles. The connection between the odontoblasts and the central ends of Tomes' fibers, TF, can be plainly seen; D, dentin; P, pulp. (Courtesy of W. Willman.)

The first symptom of beginning degenerative changes in the pulp is the presence of fat in the form of fine droplets in the pulp tissue. These deposits of fat may be found in the odontoblasts as well as in the nuclei of the pulp cells and in the walls of the pulp capillaries. Further degenerative changes in the pulp are vacuo-

lization of the odontoblastic layer and atrophy of the pulp tissue. Both these processes are characterized by a decrease in the number and size of the pulp cells and by a replacement of the cellular elements by fibers (fibrosis of the pulp).

Fig. 31 shows the process of vacuolization of the odontoblasts. In the center of the field the odontoblasts are still visible as a continuous line of elongated cells; toward the sides, however, the cells are pushed apart by an intercellular accumulation of fluid. The pressure of this fluid has displaced some of the odontoblasts from the dentin surface.

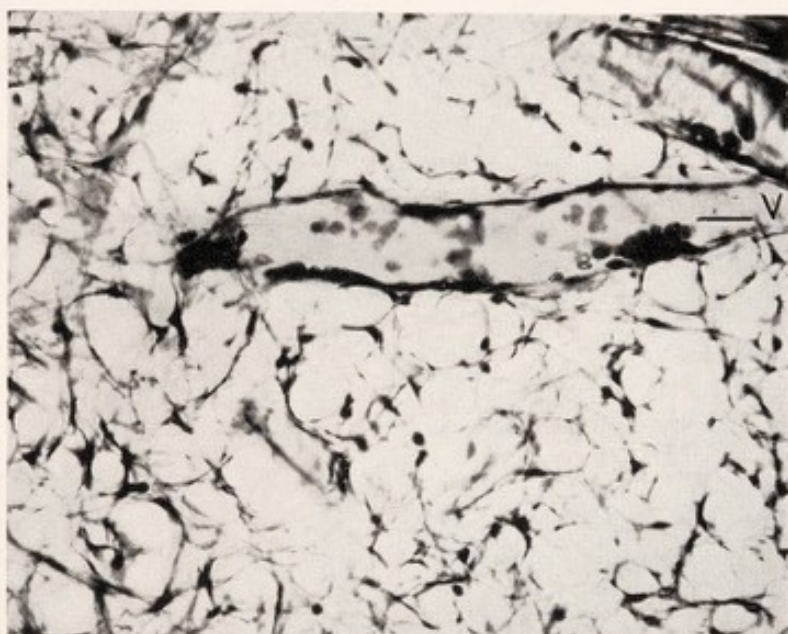


FIG. 32.—Beginning reticular atrophy of the pulp. Reduction in the number of pulp cells. The intercellular accumulation of fluid gives the pulp a net-like appearance. V, bloodvessel. (Courtesy of W. Willman.)

Atrophy of the pulp tissue can be studied by comparing Fig. 3, which was taken of the normal pulp of a young tooth, with Fig. 32. Fig. 3 illustrates the original arrangement of pulp cells and fibrous structures of the pulp. In Fig. 32 the number of cells has decreased; small, round, fluid-containing areas have appeared between the individual cells. As a result of these changes, the pulp tissue assumes the appearance of a net and, therefore, this condition is called reticular atrophy. With advancing reticular atrophy, the spaces or vacuoles become larger, the amount of fibrous tissue is reduced and the number of cells is greatly lessened (Fig. 33). In extreme cases of reticular atrophy few cellular elements are left; the pulp tissue consists of irregular, coarse, fibrous strands stretched across the pulp chamber. Bloodvessels and nerves are very much

reduced both in number and size, which fact accounts for the greatly decreased sensitiveness of such pulps. The odontoblasts have completely disappeared (Fig. 34).

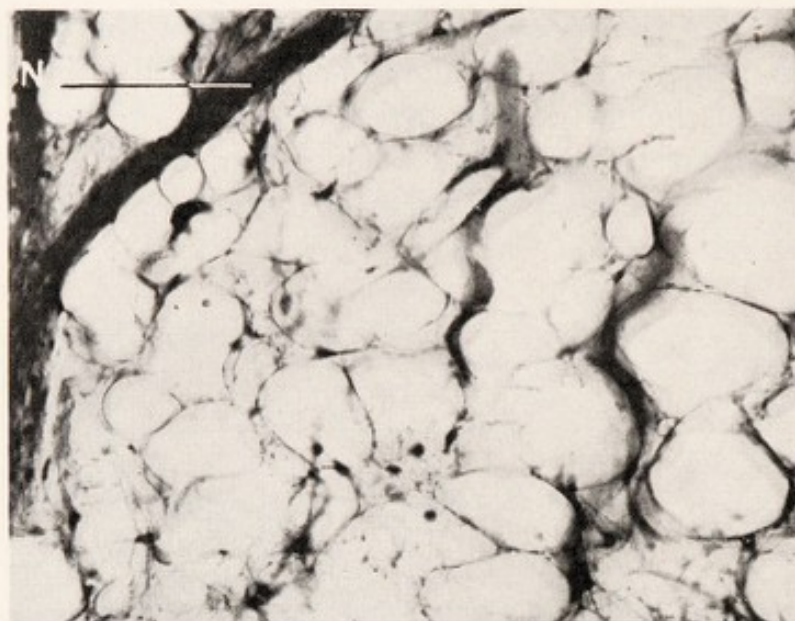


FIG. 33.—Reticular atrophy of the pulp. Formation of large spaces in the fibrous pulp tissue. Greatly reduced number of pulp cells. N, nerve. (Courtesy of W. Willman.)

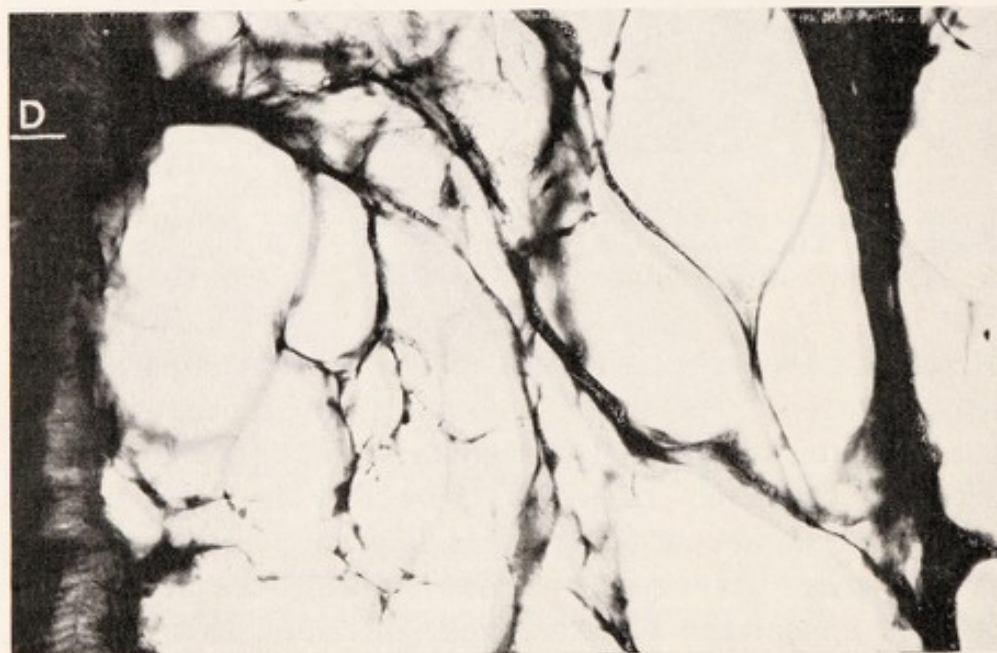


FIG. 34.—Advanced reticular atrophy of the pulp. Only a few coarse strands of fibrous tissue are left. Hardly any cellular elements are present. D, dentin. (Courtesy of W. Willman.)

DENTICLES AND CALCIFICATIONS OF THE PULP.

The human pulp tissue is highly subject to abnormal calcification. Since these calcifications show wide variations, it is necessary to

classify them according to size, structure and localization. Relatively large, well-outlined bodies of hard substance found in the pulp chamber are called denticles. In addition, fine, diffuse calcifications are frequently observed in the pulp tissue; they are spoken of as pulp calcifications or as calcific degeneration of the pulp tissue.

1. **Denticles.**—According to their microscopic structure denticles are classified as true and false.

(a) True denticles are denticles consisting of irregular dentin.

(b) False denticles are degenerative calcifications of the pulp tissue. They usually show, under the microscope, a concentric, lamellated arrangement as the result of a deposition of consecutive layers of calcium salts around a central nucleus.

According to their relation to the walls of the pulp chamber, all denticles may be classified as follows:

Free denticles: denticles lying freely in the soft tissue of the pulp without connection with the walls.

Adherent denticles: denticles attached to the wall of the pulp chamber.

Interstitial denticles: denticles embedded in the dentin. This latter type develops from free or adherent denticles by continued dentin formation by which the denticle finally becomes entirely surrounded by dentin.

Clinically, denticles may sometimes be diagnosed by means of the radiograph. They then appear either as prominences adherent to the wall of the pulp chamber or lying freely in the pulp tissue. Their size in the radiograph varies from hardly visible, dust-like granules to bodies the size of 2 or 3 mm. filling almost the entire pulp chamber. Upon the removal of the roof of the pulp chamber, denticles can sometimes be taken out with a spoon excavator (free denticles), while in other cases they are in solid junction with the walls of the pulp chamber (adherent denticles) and then may cause great difficulties if a treatment of the root canals is intended. Large denticles in the pulp chamber can make the root canals almost inaccessible; those adhering to the walls of the root canals sometimes form an insurpassable obstacle to broaches or reamers.

(a) *True Denticles.*—Denticles with the histological structure of dentin are the result of a localized over-formation of dentin. They develop in a large majority of cases from the wall or from the bottom of the pulp chamber because of excessive dentin-forming activity in a circumscribed area, and thus are closely related to secondary dentin. Fig. 35 shows a photomicrograph of a ground section through an upper molar. A large true denticle has devel-

oped from the floor of the pulp chamber; it grew toward the roof of the pulp chamber, until it came in contact with the latter, thereby dividing the pulp chamber into two small sections. The denticle

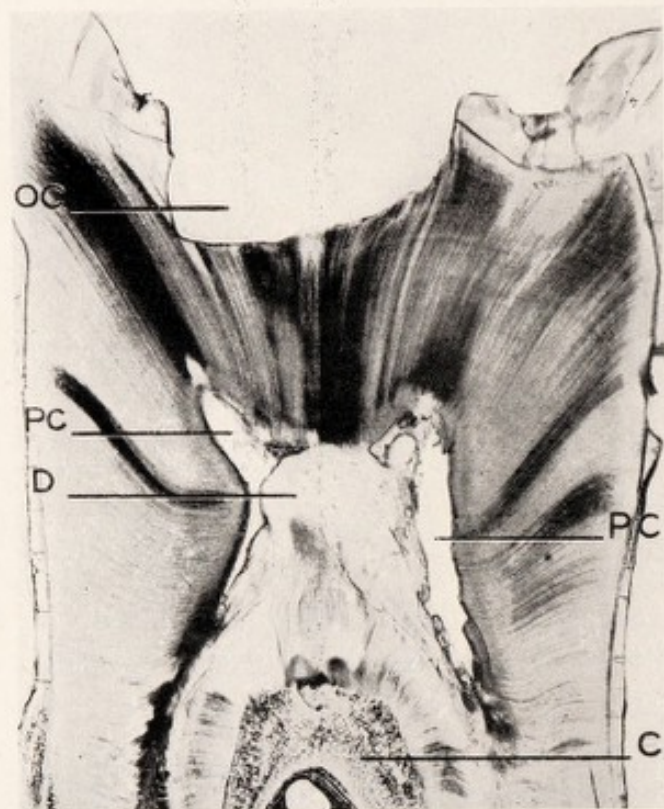


FIG. 35.—Large true denticle in the pulp chamber of lower molar. Ground section. The denticle is attached to the floor and to the roof of the pulp chamber, thus separating the pulp chamber into a mesial and a distal portion. OC, occlusal cavity; D, denticle; PC, pulp chamber; C, cementum in bifurcation.

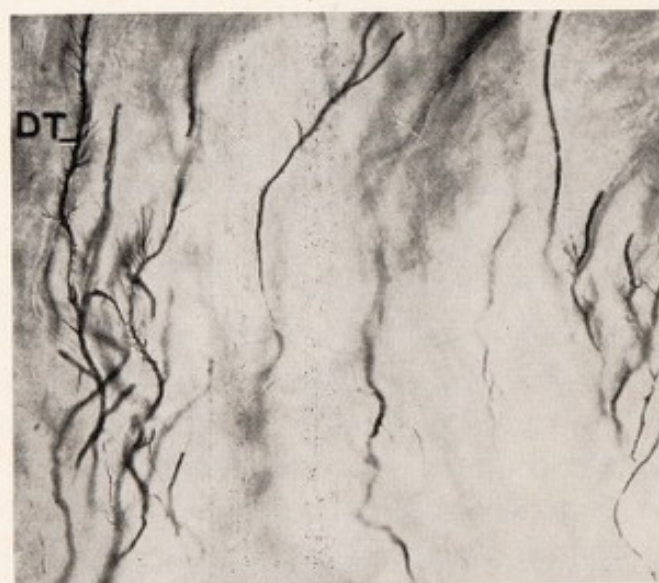


FIG. 36.—High magnification of the denticle in Fig. 35. The denticle consists of secondary dentin with very irregular dentinal tubules, DT.

consists of secondary dentin with scanty, irregular dentinal tubules (Fig. 36).

Sometimes true denticles are also found lying freely in the pulp chamber entirely surrounded by pulp tissue. However, such free true denticles are rare, and most of the free denticles must be classified histologically under the second group, namely, false denticles. For the formation of a free true denticle with dentinal tubules, the presence of odontoblasts in parts of the pulp other than in the periphery is necessary. It seems that the cells in portions of the pulp other than in the periphery may become trans-

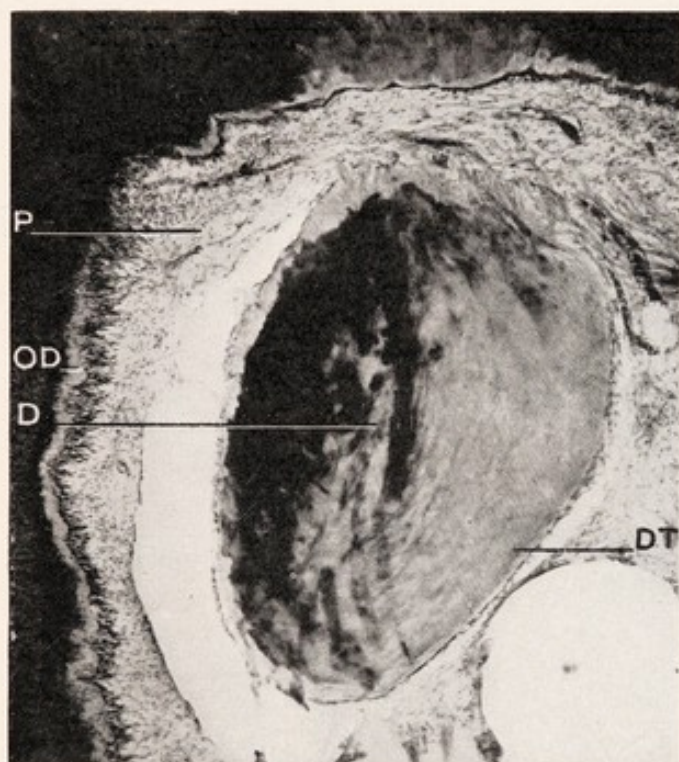


FIG. 37.—Free true denticle. D, denticle; DT, dentinal tubules in the denticle; P, pulp tissue; OD, odontoblasts.

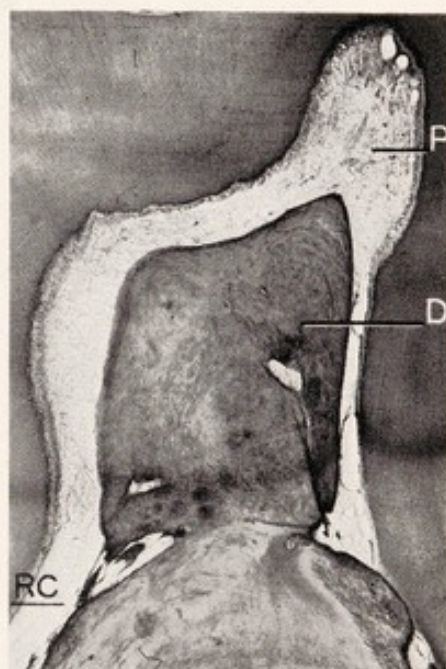


FIG. 38.—Large denticle attached to the floor of the pulp chamber of an upper molar. The pulp chamber is reduced to a narrow slit between denticle and dentin wall. D, denticle; P, pulp; RC, root canal.

formed occasionally into odontoblasts and may take part in the formation of dentin. The presence of epithelial cells in the pulp may have some connection with this type of dentin formation (Orban).

Fig. 37 shows a free true denticle. The inner part of the denticle consists of calcified pulp fibers, the outer part of irregular dentin; the surface is covered by a layer of dentinoid and is surrounded by odontoblasts. Evidently the calcification of pulp fibers took place first, these calcified masses forming the nucleus around which irregular dentin was later deposited.

(b) *False Denticles*.—This group includes the great majority of the denticles found in human teeth. False denticles originate from deposits of calcium salts in the pulp tissue, probably following minor circulatory disturbances in the pulp vessels. These denticles vary



FIG. 39.—High magnification of a small free denticle of concentric lamellated structure (false denticle). D, denticle; OD, odontoblasts and dentinoid on the floor of the pulp chamber. Notice the strand of calcified fibrous tissue in the center of the denticle. This calcified tissue forms the nucleus around which the denticle is built up in concentric layers.



FIG. 40.—Several free and adherent denticles in lower molar. The larger denticles are the result of a coalescence of several smaller denticles of concentric lamellated structure (false denticles). FD, free denticles; AD, adherent denticle on the floor of the pulp chamber.

greatly in size; sometimes they fill almost the entire pulp chamber. Fig. 38 illustrates a large denticle which is attached by a broad base to the floor of the pulp chamber. The pulp tissue is confined to a narrow uniform space all around the denticle between the latter and the wall of the pulp chamber.

Denticles of the size shown in Fig. 38 are rather rare; small denticles, however, are very common and, in the author's experience, are present in at least 80 per cent of all adults' teeth.

False denticles are formed by a concentric deposition of consecutive layers of calcium salts around a central nucleus which usually consists of



FIG. 41. — Small adherent denticle in the root canal of a molar. P, pulp; AD, adherent denticle; SD, secondary dentin; PF, calcifying pulp fibers.

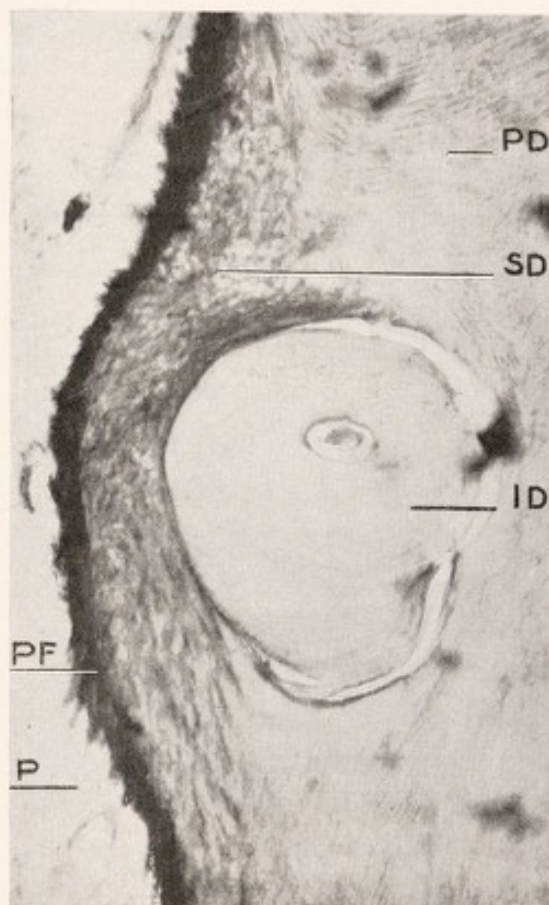


FIG. 42. — Interstitial denticle in the wall of the root canal of a molar. P, pulp; ID, interstitial denticle; PD, primary dentin; SD, secondary dentin; PF, calcifying pulp fibers. The denticle shows a concentric laminated structure around a central nucleus (necrotic pulp tissue?).

some degenerated pulp tissue (Fig. 39). As these round denticles grow they approach each other and may finally become fused. If they are close to the wall of the pulp chamber, they gradually become adherent by a solid union with the dentin (Fig. 40).

Two small, false denticles in the root canal of a lower molar are illustrated in Figs. 41 and 42. In Fig. 41 the denticle is attached to the wall of the root canal. Evidently it was originally formed

freely in the pulp tissue; later on, when the lumen of the canal was gradually reduced by continued formation of dentin, the denticle became adherent. As the deposition of dentin goes on, the denticle finally may become entirely surrounded by dentin, and is then called an interstitial denticle. This condition is shown in Fig. 42: a small, round denticle of concentric, lamellated structure is completely embedded in the wall of the root canal. These denticles are usually small and of little practical importance.

2. Pulp Calcifications (Calcific Degeneration of the Pulp Tissue).—Fine, fibrillar calcifications are abundant



FIG. 43.—Diffuse calcification (DC) of the pulp tissue in an intact lower molar of an adult. D, denticles resulting from fusion of minute calcified areas.

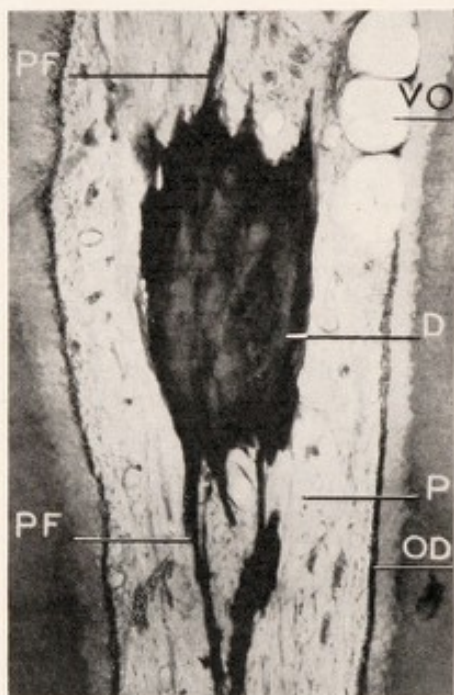


FIG. 44.—Fibrillar calcification of the pulp tissue in a root canal. P, pulp tissue; OD, odontoblasts and dentinoid; PF, calcified pulp fibers; D, denticle formed by coalescence of calcified pulp fibers; VO, vacuolization of the odontoblastic layer.

in some pulps, especially in those of older teeth (Fig. 43). Sometimes such calcifications begin in the wall of a bloodvessel or in the perineural connective tissue of a larger nerve, and then follow the course of the strands of vessels and nerves in the pulp. By the fusion of such calcifications long, thin denticles are formed, the origin of which can be recognized easily under the microscope (Fig. 44).

3. Etiology of Denticles and Pulp Calcifications.—The formation of denticles is often associated with the presence of an irritant of long standing, such as caries, deep fillings, or chronic inflammation. They occur more often in old people than in young ones. Denticles have been found, however, in very young teeth and even in tooth

germs before eruption; therefore, outer irritations cannot be the only cause for the formation of these calcifications. Due to the absence of any collateral circulation, the dental pulp is subject to minor circulatory disturbances more than any other organ. As a result, the connective tissue of the pulp frequently shows small areas of hyalinization, which later may become calcified and form the nuclei for the formation of denticles.

The presence of denticles is sometimes blamed for pulpitis pain or even trifacial neuralgia. In fact, in some of our histological specimens, denticles were found in close proximity to pulpal nerves, and it is not impossible that these nerves may become compressed and irritated by the growing denticle. However, in clinical examinations of such cases it will be necessary to study and analyze all circumstances most carefully and to rule out all other possible causes for the neuralgia before a denticle can be looked upon as the etiological factor.

METAPLASIA OF THE PULP.

The term "metaplasia of the pulp" (Euler) indicates the possibility of a change in the biological character of the pulp. In order to understand this point, it is necessary to recall the biological relationship between connective tissue and pulp tissue. The connective tissue of the jaws is able to produce bone or bone-like cementum, hard substances that consist of a calcified intercellular substance with cells embedded in it. The pulp is able to produce a more highly differentiated form of hard tissue, the dentin, the structure and function of which is different from that of the bone. The term metaplasia means that the pulp tissue has deteriorated from its higher specialization (dentin formation) to the quality of connective tissue (bone or cementum formation); as a result bone or cementum will be formed within the pulp chamber.

A case of metaplasia of the pulp is illustrated in Fig. 45. In examining sections through an intact upper third molar of an adult, it was found that the pulp chamber was lined completely by a layer of cell-containing cementum which was separated by a very definite line from the dentin. Nothing is known about the history of this tooth, and nothing in the specimen indicates any outer interference; all that can be said is that for some unknown reason the pulp changed its function from the formation of typical dentin to the formation of a hard substance having the characteristics of bone.

True metaplasia of the pulp is a rare condition; it must not be confused with the frequent observation of cementum in the apical

portion of root canals following root canal treatment or pulp injuries and also in the apical portion of root canals of intact teeth. These deposits of cementum are the result of an actual ingrowth of periodontal connective tissue through the apical foramen into the root canal and have nothing to do with metaplasia of the pulp.

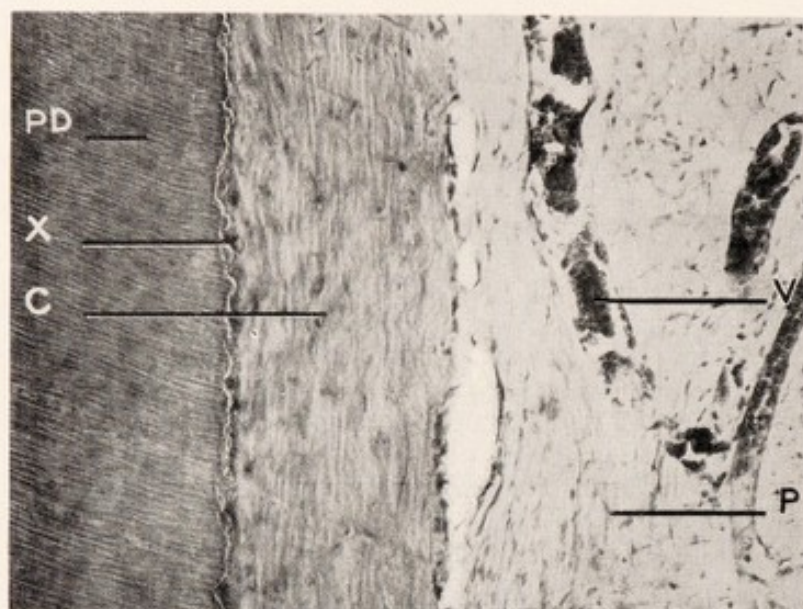


FIG. 45.—Metaplasia of the pulp. Intact upper third molar. Deposition of cementum on the wall of the entire pulp chamber. PD, primary dentin; X, borderline between dentin and cementum; C, cell-containing cementum; P, pulp tissue; V, bloodvessels of the pulp.

BIBLIOGRAPHY.

- BEUST, TH. B.: Reactions of the Dentinal Fibril to External Irritation, *Jour. Am. Dent. Assn.*, 1931, **18**, 1060.
- BOEDECKER, C. F.: Changes in Teeth from Youth to Old Age, *Dental Cosmos*, 1925, **67**, 543.
- BOEDECKER, C. F., and APPLEBAUM, EDMUND: Effect of Filling Materials upon Teeth, *Dental Cosmos*, 1930, **72**, 1001.
- BRAMMER, FRIEDRICH: Über Atrophie pulpæ reticularis bei chronisch-entzündlichen Veränderungen des Parodontiums, Berlin, Otto Elsner, 1927.
- CAHN, L. R.: Calcifications of the Dental Pulp, *Dent. Items Int.*, 1926, **48**, 808.
- COOLIDGE, EDGAR D.: Heterotopic Ossification in the Pulp: A Contribution to Research on the Biology of the Dental Pulp, *Jour. Am. Dent. Assn.*, 1929, **16**, 821.
- The Treatment of Deep Dental Caries, *Ill. Dent. Jour.*, May, 1932.
- DEMOLIS, PAUL: Les denticles pulpaire, *Schweiz. Mon. f. Zhk.*, 1931, **41**, 1180.
- EULER, H.: Metaplasie der Pulpa, *Vrtljschr. f. Zhk.*, 1921, **37**, 303.
- Sekundäre Odontoblastenbildung, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 488.
- Über Zystenbildung in der Pulpa, *Ztschr. f. Stom.*, 1930, **28**, 1019.
- Kalkeinlagerungen und Pulpgefäße, *Paradentium*, 1932, **4**, 102.
- EULER, H., and REBEL, H. H.: Sekundäre Odontoblastenbildung, *Ztschr. f. Stom.*, 1932, **30**, 515, 588.
- FISH, E. W.: The Reaction of the Dental Pulp to Peripheral Injury of the Dentine, *Proc. Roy. Soc. B*, 1931, **108**, 196.
- The Physiology of Dentin and Its Reaction to Injury and Disease, *Brit. Dent. Jour.*, 1928, **49**, 593.

- FISH, E. W.: Lesions of the Dentin and Their Significance in the Production of Dental Caries, *Jour. Am. Dent. Assn.*, 1930, **17**, 992.
- The Pathology of the Dentine and the Dental Pulp, *Brit. Dent. Jour.*, 1932, **53**, 351.
- FRIDRICHOVSKY, JAN: Zur Histologie der Dentikel, *Ztschr. f. Stom.*, 1927, **25**, 124.
- HOPEWELL-SMITH, A.: Some Remarks on the Human Dental Pulp: Its Reactions to Injury, Its Diseases and Their Immediate and Remote Complications and Sequelæ, *Dental Cosmos*, 1924, **66**, 489, 601.
- Adventitious Dentin and Infection of the Dental Pulp, *Dent. Items Int.*, 1925, **47**, 477, 557.
- KOKUBAN, SHIRO: Beiträge zur Lehre der Fettstoffwechselstörungen der Zahnpulpa unter Berücksichtigung der Pulpagesfäße, *Vrtljschr. f. Zhk.*, 1931, **47**, 139.
- MEYER, W.: Ein neuer Fall von Metaplasie der Pulpa, *Paradentium*, 1931, **3**, 48, 50.
- MÜNCH, JOSEF: Die harten Neubildungen der Zahnpulpa, *Deutsch. Zhk.*, 1925, vol. **64**.
- OKINO, SETSUKO: Experimentelle Untersuchungen über die Durchlässigkeit der transparenten Zone bei der Dentinkaries am lebenden Zahn, *Schweiz. Mon. f. Zhk.*, 1930, **40**, 309.
- ORBAN, B.: Epithel in der Pulpa, *Schweiz. Mon. f. Zhk.*, 1927, **37**, 610.
- Epithelial Rests in the Teeth and their Surrounding Structures, *Proc. Fifth Annual Meeting of the Am. Assn. of Dental Schools*, March 1928, p. 121.
- OTTOLENGNI, R., and CAHN, L. R.: The Pathology of the Dental Pulp, and the Practical Significance of Such Knowledge in the Treatment of Vital and Pulpless Teeth, *Dent. Items Int.*, 1926, **48**, 897.
- PALAZZI, S.: Experimentelle Untersuchungen und histopathologische Betrachtungen über die Pulpa der mit sogenannten Jacketkronen überkappten Zähne, *Schweiz. Mon. f. Zhk.*, 1928, **38**, 343.
- REBEL, H. H.: Beitrag zur Inneren Zementbildung durch Echte Metaplasie, *Ztschr. f. Stom.*, 1920, **18**, 335.
- REICH, P.: Das irreguläre Dentin der Gebrauchsperiode, Jena, Fischer, 1907.
- Einiges über das irreguläre Dentin und vorläufige Bemerkungen zur Kritik desselben, *Vrtljschr. f. Zhk.*, 1908, **24**, 83.
- RYWKIND, A.: Über Zementablagerung in den Wurzel-Kanälen und der Pulpa-kammer, *Ztschr. f. Stom.*, 1926, **24**, 923.
- VOGELSANG: Die Reaktion der Pulpa auf plötzlichen Schmelzmangel, *Deutsch. Mon. f. Zhk.*, 1922, **40**, 97.
- WANNENMACHER, E.: Ein Beitrag zur pathologischen Histologie der Pulpa, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 12.
- Die Veränderungen der Pulpa bei Keilförmigen Defekten mit besonderer Berücksichtigung der Reizdentinbildung, *Korr. f. Zahnärzte*, 1927, **51**, 388.
- Schmelzverlust und Pulpa, *Deutsch. Zahnärztl. Wehnschr.*, 1928, **31**, 281.
- WILLNER, HANS: Über die fettige Degeneration der Pulpa, *Deutsch. Zahnärztl. Wehnschr.*, 1926, **29**, 57.

CHAPTER III.

DENTAL CARIES.

DENTAL caries, due to the frequency and to the great medical and social importance of this disease, for a long time has been the subject of intense study. The pioneers in dentistry, men like Black, Miller, Williams, and others, have devoted a great deal of their time and energy to investigations on dental caries, and from their work the pathological manifestations of caries, its relative frequency in various groups of teeth, and the points of most frequent localization on the tooth surface are well known.

CLINICAL CHARACTERISTICS OF DENTAL CARIES.

Although it is not the purpose of this book to give detailed clinical data of oral diseases, still, in consideration of the great importance of dental caries, it might be appropriate to present a brief outline of the clinical aspect of dental caries that has been compiled and published by Dr. R. W. Bunting of Ann Arbor.¹

"1. Dental caries is a destruction of the hard substances of the tooth by a process, the initial stage of which is a decalcification by acids.

"2. The acids active in caries are not generally distributed in the saliva, but are localized and concentrated on certain areas of the tooth surfaces.

"3. Carious lesions occur most frequently in the pits and fissures of the occlusal surfaces and on certain areas of the approximal, buccal, and lingual surfaces of the teeth, at which locations there is opportunity for stagnation and the retention of foreign matter. They do not occur on smooth enamel surfaces which are frequently cleansed.

"4. All initial lesions of caries contain acid-forming bacteria capable of producing and living in acids of sufficient potential to decalcify the enamel.

"5. The hardness or softness of the teeth may affect the rate of progress and extent of caries but do not alone determine its occur-

¹ Dental Cosmos, 1930, **72**, 399.

rence. Caries, as a rule, runs a more rapid and extensive course in poorly formed teeth than in the hard and well-formed varieties, but instances commonly occur in which the poorest formed teeth are wholly free from the disease.

"6. Malhygiene of the mouth frequently favors the inception of dental caries and increases its activity, but alone does not determine its occurrence. Mouths that are habitually unclean are often wholly free from caries and, conversely, mouths that are scrupulously clean may be seriously affected by the disease.

"7. The process of dental caries is related to and often determined by certain constitutional states and conditions of bodily health. The nature of these general influences and the manner in which they affect the course of this dental disease are not clearly understood at this time. The following bodily conditions are perhaps best known as systemic factors which either favor or oppose dental caries:

"(a) *Heredity*.—There are strong evidences that the tendency toward dental caries or an immunity to the disease may be transmitted from parent to child, according to the laws of familial inheritance.

"(b) *Susceptibility to dental caries* is clearly influenced by age. Incidence of the disease is known to be highest during the ages of seven to twenty years. After twenty years the tendency to caries is markedly decreased.

"(c) *Health*.—It is frequently noted that severe onsets of dental caries follow attacks of general disease and disturbance of bodily health. During pregnancy caries may be unusually active. Children who are undernourished or are suffering from a general debility are specially prone to dental caries.

"(d) *Racial Influences*.—Dental caries is more prevalent in certain races than in others. The natives of Africa, South America and the South Sea Islands, the Esquimaux and many other primitive peoples are notably free from the disease, while those who live in the more civilized communities are extremely susceptible to it. There are evidences that the disease often increases in prevalence as the people advance in the scale of civilization. It is also observed that when individuals migrate from a caries-free nation to a country in which it is prevalent they and their progeny may later develop dental disease. There is no indication that dental caries is an endemic affection or that it is induced by any particular climatic conditions. The most constant and important variable between immune and susceptible races appears to be that of diet."

EXPERIMENTAL RESEARCH IN DENTAL CARIES.

The problem of the etiology of dental caries has been approached recently in two ways: (1) In man, by clinical examination of groups of people, especially children who live under known dietary conditions; (2) in animals, by experimental production of caries or caries-like lesions.

1. Influence of Dietary Conditions Upon Dental Caries in Children.—Investigations dealing with the frequency and progress of dental decay in man can be carried out only with large groups of children who live in closed institutions (schools, orphanages) so that it is possible to control the actual intake of food of every child over a period of several years. During this time the children live under a certain dietary régime which, in the main, consists of a well-balanced diet with a high content of vitamins and roughage and a low content of carbohydrates. The total amount of tooth decay present in the teeth of these children is carefully checked and tabulated and compared with figures obtained from the examination of control children who live under different dietary conditions.

At the present time such studies on large groups of children are being made in several parts of this country (R. W. Bunting and associates in Ann Arbor, C. L. Drain and associates in Iowa City, M. T. Hanke in Chicago, H. F. Hawkins in Los Angeles, and Martha R. Jones in Hawaii). Although the different investigators do not entirely agree as to just what constitutes the effective factors in the control of dental caries, they all state that the teeth of a child who is fed on an adequate and well-balanced diet are less subjected to decay than are those of a child who is fed on an average or poor diet. The results obtained by these investigators are highly encouraging, for it seems actually possible to reduce the frequency and the extent of dental caries by an adequate dietary régime.

An example of an experiment of this kind, a few of the actual findings of the "Michigan group" of investigators (R. W. Bunting, Philipp Jay, Dorothy G. Hard) at a county orphanage in Maumee, Ohio, will be discussed. In accompanying Dr. Bunting on his semi-annual dental check-up in the institution, the author had an opportunity to examine the mouths of more than 60 children; the occurrence and extent of dental caries were compared with the very exact charts that had been made of the dental conditions of these children one year before. The unusually healthy state of the mouths of the children and the small amount of caries present was very remarkable. In many instances cavities that had been recorded a

year before had not changed in size, and the bottom of the cavities appeared surprisingly hard when examined with an explorer. The diet of these children consisted of large amounts of milk, vegetables, eggs, and meats; fruit, both raw and stewed, was liberally given. Carbohydrates were eliminated as much as possible. The main carbohydrate foods given were potatoes and dark bread. Sweets and candy were eliminated entirely. Under this diet the children were in excellent health; they did not miss candy, since they did not have it and did not come in contact with outside children who would have stimulated their desire for it.

Bunting bases the reduction of sugar upon the assumption "that the average child consumes too much carbohydrates in the form of candy and highly sweetened foods, as a result of which the appetite for other foods is perverted and a state of nutritional unbalance is produced; furthermore, that candy and sticky confections retained about the teeth afford a most favorable pabulum for acid fermentations in the mouth."¹

The results of this survey in several institutions that were under the dietary control of Bunting and his collaborators were published recently, and some of the actual figures obtained in the orphanage in Maumee are as follows: Of 156 children under control, 125, or 80 per cent, showed no trace of active caries appearing during one year. In only 8 children, 5 per cent, was active caries found; however, the lesion consisted merely of from 1 to 3 small cavities per child on the occlusal or buccal surfaces of the teeth. The remaining 15 per cent has such small defects that their significance as caries was questionable in the opinion of the examiners. Sixty-six of the 125 children who showed no new caries at the end of the year had had from 1 to 10 open cavities in their mouths when the experiment was started. These cavities had been untouched during the year. At the final examination these defects had not increased in size, nor had new caries developed.

Although there is no possibility of obtaining adequate "controls" for experiments of this type, the above-mentioned figures show a percentage of active caries that is far below that which every practitioner or pediatricist is able to observe among average American school children. Therefore, it seems that the favorable condition found under certain food régimes actually can be attributed to the dietary factor.

Boyd and Drain at the University of Iowa observed during routine mouth examinations in a dental clinic that a group of 28 children

¹ Bunting, R. W.: *Am. Jour. Dis. Child.*, 1930, **40**, 536.

showed arrest of dental caries. These children had open, untreated cavities which showed no tendency to increase in size; the walls of the cavities had become hard and dark. Mouth hygiene apparently had no relation to this change since some of the mouths were decidedly unclean. It was found that all these patients with arrested caries were diabetic children who had been on a diabetic diet for six months or more. This diet consisted of meat, eggs, butter, milk, vegetables, fruit, and cod-liver oil.

Boyd and Drain attribute the arrest of caries in these cases to the correction of faulty nutrition and to the intake of an adequate amount of vitamins and mineral salts. In order to determine whether the fact that the children had diabetes and were being treated with insulin had anything to do with the control of dental caries, the investigators put 4 non-diabetic children with active, soft dental caries on a typical diabetic diet for a period of four months. During this time no progress of the carious process in the existing open cavities could be observed. After these 4 children had resumed their ordinary diet, 2 of them developed recurrent, active caries.

From a study of the literature on experimental caries prevention in children, one gets the impression that the investigators still disagree as to the actual mechanism of caries prevention. Bunting and his collaborators emphasize the importance of an acidogenic type of microorganism, the *Bacillus acidophilus*. The factor of diet may produce an environment in the mouth that is favorable or unfavorable for the development and activity of this organism. This infective theory of caries would give the clew to many clinical observations such as, for instance, the fact that some teeth are subject to rapid decay despite extreme cleanliness, whereas others are immune to caries in the presence of extreme neglect of oral hygiene; it also explains adequately the definite localization of caries on certain areas of the teeth. Boyd and Drain believe that improved nutritional conditions bring about a hardening of the tooth and thus an increased resistance against decay. Hawkins stresses the importance of the acid-base balance and the calcium-phosphorus balance in blood and saliva as related to nutrition. As a whole, however, all investigators express the opinion that a nutritionally sound and well-balanced diet is of greatest importance for the prevention and control of dental caries.

2. Experimental Production of Caries in Animals.—The second group of experimental investigation deals with the experimental production of caries in animals. In this field, the work of Bunting,

Grieves, Howe, Klein, Marshall, and Mellanby is most important. These investigators were able to produce structural imperfections and, in some instances, typical caries in the teeth of animals by certain variations in the food, especially by a reduction of the mineral and vitamin content and by an increase in the amount of carbohydrates.

The molars of the albino rat are excellent material for the study of experimental caries because they are in many respects similar to human molars. The rat is an omnivorous animal that is easy to

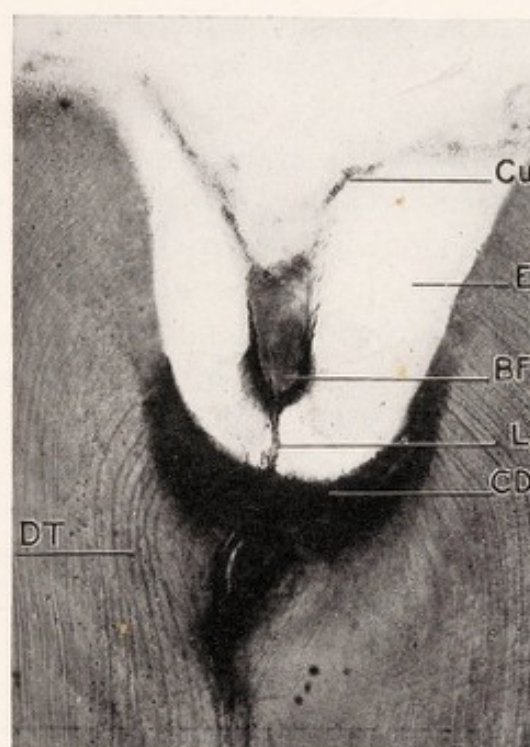


FIG. 46.—Beginning caries in an occlusal fissure of the molar of a white rat. The process of decay has passed from the bottom of the fissure through an enamel lamella to the dento-enamel junction; from there it spread into the dentin, thereby undermining the enamel. Decalcified section. BF, bottom of occlusal fissure; E, enamel; Cu, cuticle; L, enamel lamella extending from the bottom of the fissure through the enamel into the dentin; CD, caries of the dentin; DT, infected dentinal tubules. (Barker, Jour. Am. Dent. Assn.)

keep and that can easily be used in large numbers. Its molar teeth resemble human molars very closely in form and structure. They are subject to occlusal and, although less frequently, to approximal caries with all the histological characteristics that are found under similar conditions in human teeth. The occlusal caries of the molars of rats begins at the bottom of the fissures and follows the course of the lamellæ to the dento-enamel junction (Fig. 46). As the decay spreads into the dentin, secondary dentin formation can be observed in the pulp chamber. Sooner or later the pulp becomes exposed,

pulpitis develops, and the final outcome is the complete breaking down of the tooth with formation of periapical abscesses. Rats fed on a normal and well-balanced diet are affected very little by caries; whereas, animals kept on various kinds of deficient diets show a high incidence of decay.¹

HISTOPATHOLOGY OF TEETH WITH DENTAL CARIES.

1. **Enamel Cuticle and Enamel Lamellæ.**—The destruction of the hard substances by dental caries starts in the crown portion of the tooth, on the surface of the enamel. This is true for all cases except for caries of the cementum which is occasionally observed in old people with exposed roots. Since the enamel is covered by the cuticle, it is evident that this structure must play an important rôle in the development and in the early stages of dental decay. In fact, we may say that it is the cuticle that is first attacked by the process of caries, and not the enamel. Caries of the enamel develops only after the cuticle has been lost or destroyed.

The enamel cuticle is connected with the endings of the lamellæ on the enamel surface. The enamel lamellæ are bands of organic structure running perpendicularly from the enamel surface into the enamel, to the dento-enamel junction, or into the dentin. Thus, the lamellæ present natural lines of weakness in the enamel which are able to transmit caries from the surface into the enamel and to the dentin.

According to Gottlieb the enamel cuticle is composed of two parts, namely, the primary and the secondary enamel cuticle. The primary cuticle, which is formed by the ameloblasts, is a very thin membrane that usually becomes calcified. It is not resistant to decalcification, and under the influence of acids it is readily dissolved; therefore, it is of no significance in connection with dental caries. The secondary cuticle is derived from the epithelial attachment. It is much thicker than the primary cuticle, usually being about 7 microns. The secondary cuticle is built by the cells of the epithelial attachment; since in later periods of life the epithelial attachment is found on the surface of the root, the secondary cuticle

¹ It has been the author's observation that it is very difficult for anybody, and particularly for the undergraduate student, to find his way through the large amount of evidence and controversial opinions brought forth in the literature on caries during the last decade. The author, therefore, recommends the articles by R. W. Bunting (*Jour. Am. Dent. Assn.*, 1931, **18**, 785) and by R. G. Kesel (*Jour. Am. Dent. Assn.*, 1932, **19**, 903) as a very excellent starting point for the study of the recent investigations of dental caries. Both articles also contain an ample bibliography on this subject.

can also be observed on the cementum. In this sense it is generally spoken of as "cuticula dentis."

The most important characteristic of the secondary cuticle is the fact that it consists of a horny substance (keratin). The presence of horn is evident from several observations:

The secondary cuticle is highly resistant to acids and alkalis; this resistance is typical of all horn substances. In decalcified teeth, the secondary cuticle remains after all the enamel has disappeared.

It is possible to stain the secondary cuticle with stains that are specific for horn (Gram-Ernst method of staining horny substances)

The secondary cuticle is formed by that portion of the epithelial attachment that corresponds to the stratum corneum of the oral epithelium. Orban has shown that the cuticula dentis is the result of a process of keratinization of the cells of the epithelial attachment (see Fig. 232). In that way the hornified cuticle may be looked upon as a continuation of the protecting keratinous layer of the oral epithelium over the erupted portion of the tooth.

The hornified secondary cuticle is not always found; its development seems to be subject to individual variations. In mouths in which the gingival epithelium seems to have little tendency toward formation of a horny layer, the cuticula dentis is usually poorly developed or entirely missing, while in other mouths a typical hornified cuticula dentis is present on every tooth. It must also be mentioned that in erupted, functioning teeth the cuticula dentis can be found only in those areas of the tooth surface that are protected from the direct influence of mastication, namely, in the gingival portion of the teeth, on the approximal surfaces, and in the bottom of the grooves and fissures. In all other parts of the tooth surface the cuticle is worn away.

In order to study cuticle and lamellæ, it is essential to use both ground and decalcified sections. In ground sections the enamel is preserved, but the non-calcified structures, especially the cuticle, cannot be well differentiated. In decalcified sections the enamel is lost, but cuticle and lamellæ are left behind; they can be stained and examined under the microscope. Only by combining the evidence gained from these two types of sections can one fully understand the relationship between the organic structures in and around the enamel.

In a ground section made at right angles to the long axis of a tooth, a lamella appears as a dark line running from the tooth surface into the enamel and sometimes extending into the dentin (Fig. 47). If a section in an identical plane is made through a

decalcified tooth that has been embedded in celloidin, an entirely different picture is found. The enamel having disappeared, the lamella, an organic band supported by the surrounding enamel, has lost its support and has formed folds. By using special precautions during decalcification of the enamel and embedding of the specimen, it is possible to hold the cuticle in its original position after the loss of the enamel. Then the relationship between cuticle, lamella, and dentinal part of the lamella is especially clear; it seems not surprising that outer influences can easily reach the dentin along a lamella.

The enamel lamellæ were first described by C. F. Boedecker, who speaks of leaf-like processes of organic substance extending from

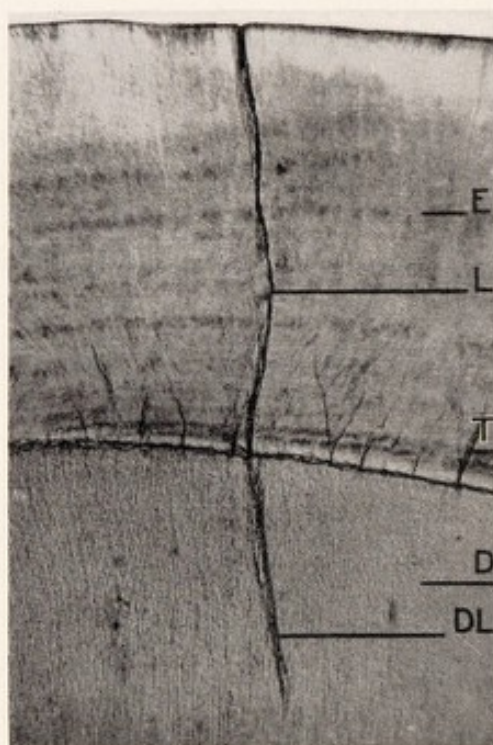


FIG. 47.—Enamel lamella. Ground section, unstained. E, enamel; D, dentin, L, lamella; DL, dentinal part of the lamella; T, tufts.

the dento-enamel junction to the enamel surface. Gottlieb later showed that the lamellæ do not necessarily end at the dento-enamel junction, but that some of them extend into the dentin (Fig. 47). In 1921 Gottlieb called attention to the rôle of the enamel lamellæ in the progress of caries from the tooth surface to the dentin as well as to the importance of the hornified cuticle as a protection against dental caries. Since then the significance of the enamel lamellæ in the caries problem has been studied and emphasized by several authors (Barker, Euler-Meyer, Faber, Orban).

Why is it, then, that bacteria present in the oral cavity do not in every instance invade the organic substance of the enamel

lamellæ and cause decay? Here is where the structure of the cuticle has to be considered. As stated before, the erupted enamel surface is usually covered by a keratinous layer in all areas that are not directly exposed to mastication, especially in its cervical portion and in the pits and fissures. Since keratin (horn substance) is highly resistant to acids, the hornified cuticle is a protection of the enamel surface against the action of acid-producing microorganisms. The hornified cuticle also covers the outer part of the enamel lamellæ where the latter reach the enamel surface. Orban has shown that the hornification may extend into the lamella itself by a transformation of the peripheral portion of the lamella into horn substance (Fig. 48). Under these circumstances the lamella is, by the

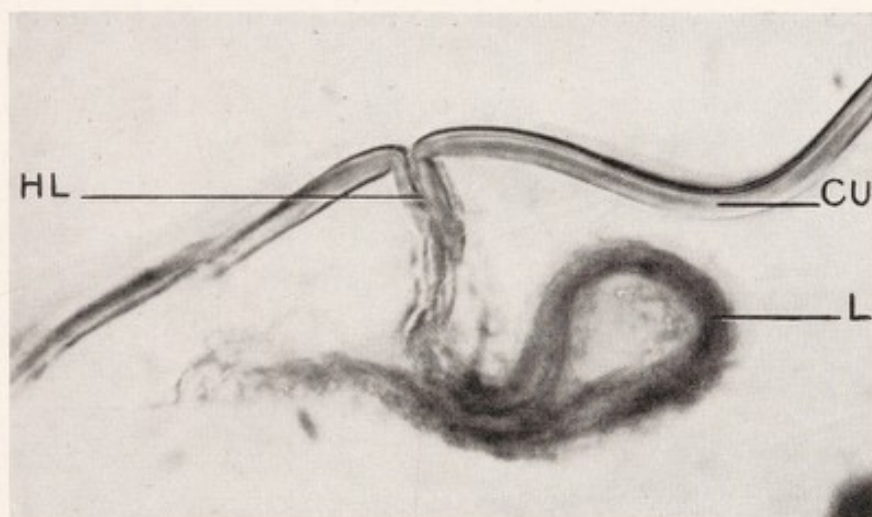


FIG. 48.—Hornified (secondary) cuticle and enamel lamella. The peripheral part of the lamella is formed by a duplicature of the cuticle. Decalcified section. CU, cuticle; HL, hornified outer part of the lamella; L, inner part of the lamella consisting of organic material without hornification. The lamella has been torn loose from the dentin and has folded up after the decalcification of the enamel.

presence of a hornified cuticle, just as much protected against bacterial invasion as is the rest of the tooth surface. On the other hand, teeth are encountered without hornified cuticle and without hornification of the peripheral endings of the lamellæ. Such teeth are, no doubt, less resistant to bacterial activity. Thus, the presence or lack of a well-developed hornified cuticle on the enamel surface seems to be an important contributing factor in the varying susceptibility of different individuals to dental decay.

2. **Caries of the Enamel.**—Many investigators have made a comprehensive study of caries, particularly the clinical side of the disease with reference to restoration and prophylaxis. The author, however, intends to go more into detail on some of the recent micro-

scopic findings in this field and to bring out a few points which are comparatively new in dental literature.

Clinically, we can differentiate between caries of the smooth enamel surfaces (approximal and gingival enamel surfaces) and caries of the grooves and fissures. The decay of the smooth enamel surfaces will be considered first.

(a) *Caries of the Smooth Surfaces of the Enamel.*—A preliminary stage in the development of caries on a smooth plane of enamel is the presence of a so-called plaque on the surface of the tooth. A plaque is an accumulation or film of soft material on the tooth surface; microscopically, it consists of microorganisms (mostly of the leptothrix family), of mucus, and of desquamated and degenerated cells. Under the protection of these masses, different bacteria develop and grow, among them the acid-producing forms (*Bacillus acidophilus*).

The formation and localization of the plaques is largely influenced and controlled by masticatory conditions, by form and arrangement of the teeth, and by the status of oral hygiene. The cusps and incisal edges are normally never the site of plaques, except in the case of hypoplastic pits, as mastication and grinding make any accumulation of material in these areas impossible. The favorite localizations for plaques are the gingival portion of the crown, the approximal surfaces, and the depths of pits and fissures. Here plaques can form without being disturbed by mastication unless they are removed by use of the tooth-brush or other mechanical force.

The presence of a plaque may lead to decalcification and caries of the underlying enamel. Whether or not such decay actually develops depends upon several factors. One of these factors is, as already mentioned, the presence and the condition of the hornified cuticle. A tooth with a well-developed hornified cuticle offers greater resistance to decalcification by acid-producing organisms than a tooth without such protecting cuticle. Another important factor is the time element. Unless the plaque is allowed to remain undisturbed over a certain period of time, no decalcification of the underlying enamel will occur. This can be observed especially in children with poor oral hygiene who show superficial decalcification of the enamel beginning under large plaques. If these children are taught to remove the plaques by using a hard tooth-brush properly and regularly, the process of decay may come to a complete standstill.

Most important is the presence or absence of acid-producing

microorganisms in the plaques. Under plaques containing such microorganisms the enamel will soon become decalcified. Plaques that do not contain acid-forming bacteria may be able to cause gingivitis but no caries.

The relationship between tooth surface, cuticle, and plaque is illustrated in Fig. 49, which was taken from a horizontal section through a central incisor at a level slightly crownward from the bottom of the gingival crevice. The specimen had been decalcified and embedded in celloidin. The same series of sections was used for some of the illustrations in the publication of Gottlieb in which he pointed out the importance of cuticle and lamellæ for the development of dental caries. In these sections the enamel has been lost



FIG. 49.—Horizontal section through gingival portion of an upper central incisor. Decalcified section. D, dentin; E, space formerly occupied by the enamel; BP, bacterial plaque on the enamel surface; CU, hornified cuticle; L' and L'', enamel lamellæ torn loose from the dentin and folded.

by decalcification during the preparation of the specimen. Plaques are present on both the mesial and the distal side of the tooth. These plaques, which in the original specimen are stained deep blue, consist mostly of bacteria. The portion of the enamel surface that is free of plaques is covered by a continuous hornified cuticle. At intervals lamellæ extend from the tooth surface toward the dentin. Some of these lamellæ connect with the hornified cuticle on the enamel surface; others lead into the dark masses of the plaque. In the latter case the peripheral part of the lamellæ can be seen invaded by microorganisms (Fig. 50). This is especially plain in the lamella illustrated in Fig. 51. This lamella has been torn loose from the dentin and has folded up. At the point of the enamel surface where the lamella originates, a distinct accumulation of microorganisms

is found; these organisms have also invaded the peripheral portion of the lamella itself. This condition can be interpreted as the earliest onset of caries; although neither enamel nor dentin is as yet



FIG. 50.—High magnification of a part of the plaque in Fig. 49. P, plaque; L, lamella; DL, dentinal part of lamella; CU, hornified cuticle; P', peripheral portion of the plaque overlying the hornified cuticle.

actually affected, masses of microorganisms are present at the peripheral end of the lamella, ready to invade the enamel. The accumulation of bacteria in this area is apparently made possible

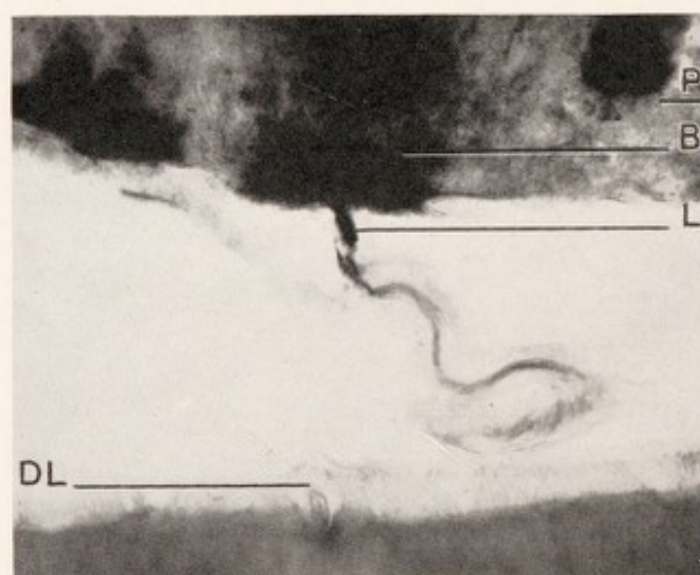


FIG. 51.—High magnification of a lamella under a bacterial plaque (Fig. 49, L"). P, plaque; B, accumulation of bacteria around the peripheral end of the lamella; L, peripheral portion of the lamella showing bacterial invasion; DL, attachment of the lamella to the dentin from which it has been torn loose after the decalcification of the enamel.

or at least facilitated by the presence of the plaque that covers and protects the microorganisms.

In a still higher magnification of a plaque covering an enamel lamella, the composition of the plaque can be recognized (Fig. 52).

Most of it consists of an amorphous mass in which cell outlines are visible in some places. Near the tooth surface grows a colony of microorganisms forming long filaments (leptothrix?); still closer to the enamel we find an accumulation of bacteria (in the actual specimen dark blue) that have entered the peripheral part of the enamel lamella present in this area.

The early stages of caries on the smooth enamel surfaces can be studied best on ground sections that run through the tooth so that they include a small area of discoloration (white or brown spot) or superficial destruction of the enamel. In such sections the earliest lesions of caries appear microscopically as a superficial decalcifica-

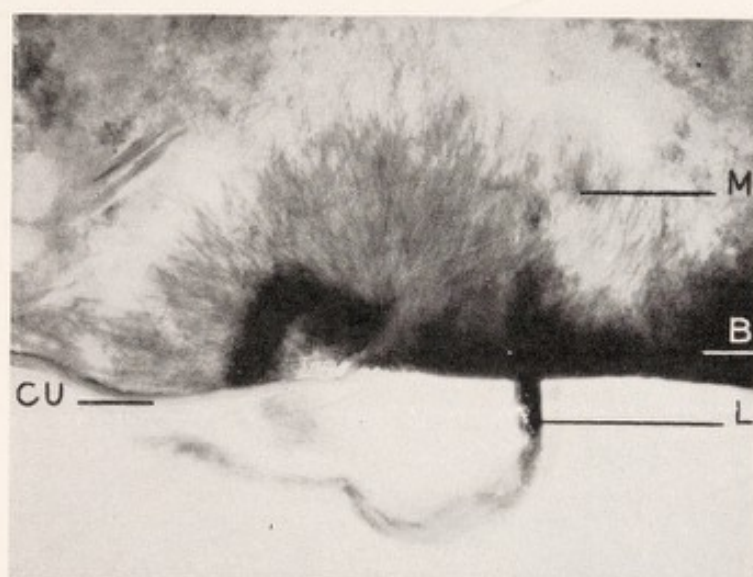


FIG. 52.—High magnification of a lamella under a plaque. M, microorganisms (leptothrix?) forming long filaments on the enamel surface; B, bacteria on the enamel surface; CU, remnants of a hornified cuticle; L, lamella, the peripheral portion of which has been invaded by microorganisms.

tion of the enamel without loss of substance on the enamel surface (Figs. 53 and 54).

In order to understand the phenomenon during pathological decalcification, it is necessary to consider briefly the physiological process of calcification of the enamel. The human enamel consists of prisms and interprismatic substance. The prism itself is formed by the union of numerous calcified globules or droplets (calcospherites) that are deposited successively by the ameloblast into the space bordered by the interprismatic substance or prism sheath. These globules calcify first; next follow the portions of the prism between the globules, and finally the interprismatic substance. In case of decalcification the loss of inorganic salts occurs in the reverse order: that part of the enamel which became calcified last is decalcified

first. Thus the interprismatic substance is decalcified first, which process is immediately followed by the decalcification of that portion

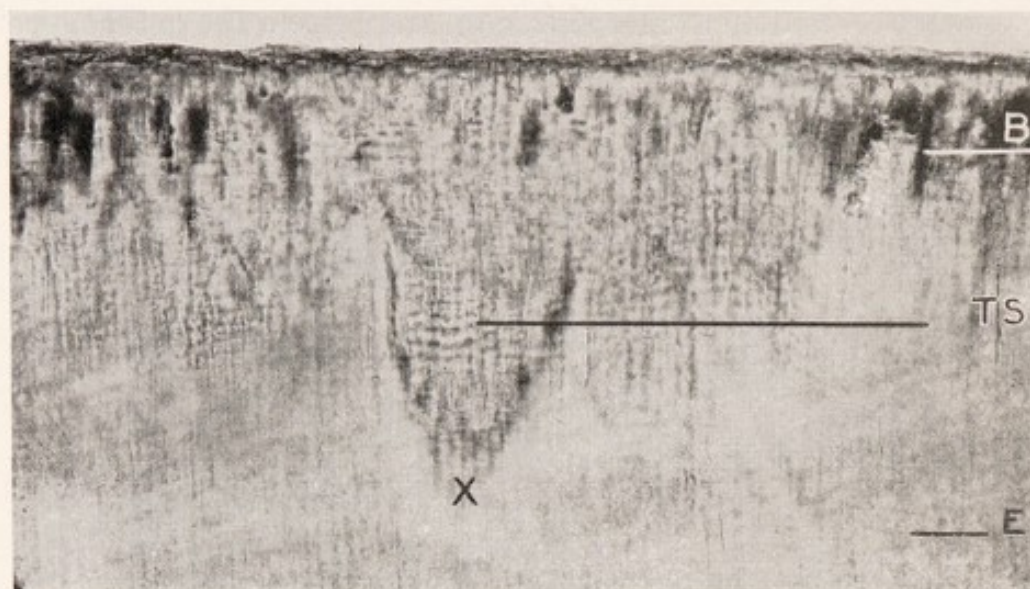


FIG. 53.—Superficial caries of the enamel on the labial surface of a tooth near the gingival margin (clinically a "white spot"). B, bacterial invasion of the enamel; X, deepest point of decalcification of the enamel; TS, transverse striation of the prisms at the borderline between normal and decalcified enamel; E, normal enamel.

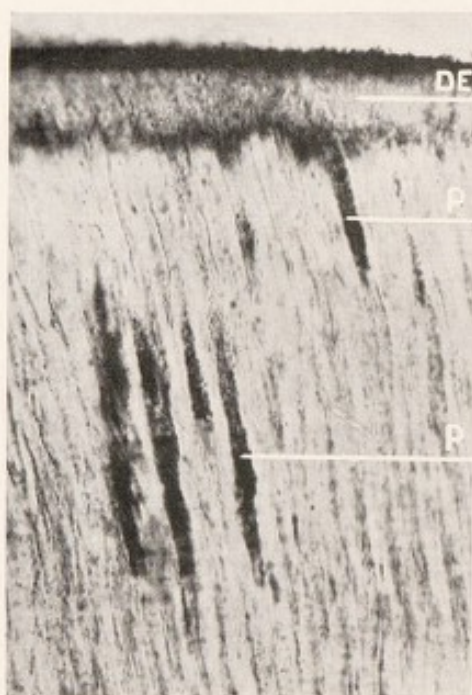


FIG. 54.—High magnification of Fig. 53. DE, superficial decalcification and disintegration of the enamel; P, decalcified and infected enamel prisms.

of the prism that united the individual calcospherites. Thereby these individual segments or divisions of each enamel prism become visible, giving the prism a segmented appearance; in the enamel as

a whole this phenomenon is called transverse striation, because the segmentation of each prism is manifested by dark lines running at right angles to the rods (Fig. 55). The same appearance of transverse striation may be observed when a ground section through normal intact enamel is exposed to the action of a diluted acid under the microscope; as soon as the enamel is reached by the acid, the rods begin to show this segmentation. Therefore, the assumption that this phenomenon is the expression of beginning calcification seems to be well supported.

Another important phase in connection with early caries of the enamel is the distinct appearance of the stripes of Retzius (Fig. 56). The stripes of Retzius are dark lines in the enamel that run more or less parallel to the enamel surface. They are also called develop-



FIG. 55.—Transverse striation of the enamel prisms in the periphery of a carious lesion of the enamel.

mental lines as they mark variations in the calcification of the forming enamel. Periods of good calcification alternate with periods of poor calcification while the rods are forming; thus stripes result that are arranged at an angle to the course of the rods. In poor calcification of the enamel (disturbance during the formative period, rickets), the stripes of Retzius are very pronounced; the dark lines represent the corresponding zones of poor calcification in each prism, the light lines the ones of good calcification. According to the modern ideas of calcification, the precipitation of calcium salts from a solution is not a continuous process, but occurs periodically. Liesegang, who made a study of the precipitation of inorganic salts from solutions, comes to very interesting conclusions. In a paper, "Cycle Formations in Nature," he discusses the fact that most of

the processes in nature occur not continuously but in cycles or periods. He differentiates between "exogenous cycles" and "endogenous cycles." An example of an exogenous cycle is the sedimentation of sand by a river in layers of different colors and thickness. In spring the melting snow increases the amount of water and the amount of erosion, and as a result more and different sediment is deposited than in the dry season. The rings in the wood of trees are caused by alternations of density controlled by the seasonal variations in growths.

As to the "endogenous cycles," Liesegang gives a very instructive example. If an aqueous solution of colorless silver nitrate and an aqueous solution of orange-colored potassium bichromate are mixed, insoluble, amorphous red silver chromate is immediately precipitated. In another experiment the potassium chromate solution is mixed with so much warm gelatin solution that after cooling off it forms a firm gelatin. If silver nitrate solution is poured on top of this gelatin, the solution will gradually enter the gelatin by diffusion, which takes several days. In this case, too, the insoluble red precipitate of silver chromate will form, but not uniformly throughout the gelatin. Rather will it be found that, if proper concentrations of the salt solutions were used, there will be red layers in the gelatin alternating with colorless zones, arranged parallel to the surface of the gelatin or at right angles to the direction of the diffusion. How can this phenomenon be accounted for? There is no doubt that the diffusion of the silver solution is continuous and uniform throughout the gelatin; still, we find distinct layers of precipitation. The following explanation is offered: Silver chromate forms continuously as the silver solution enters the chromium gelatin. However, the silver chromate is not precipitated immediately but remains in solution until the solution is over-saturated. Then at a certain point, the precipitation of all the silver chromate from the oversaturated solution occurs suddenly, causing a zone of red precipitate in the gelatin. After that it again takes a while until enough concentration is attained to cause precipitation, hence the colorless zone in the gelatin. Thus the uniform process of diffusion is transformed into a rhythmic precipitation of the forming salts.

Liesegang uses a very good physical rather than chemical comparison for this phenomenon that is otherwise rather hard to imagine. If a faucet is turned on, the water will run out in a uniform, continuous stream. If, however, the faucet is nearly shut off allowing only drops to pass, the small amount of water will not drop immediately after passing the faucet; it will hang there for a

while until the last milligram of water causes the drop to fall, taking along all the water that was present; then again it takes a while until enough water has accumulated for another drop to fall. Thus, the constant flow of water through the faucet is transformed into rhythmic drops, in the same way in which the constant diffusion of a solution leads to rhythmic precipitation.

In the calcifications that occur in living organisms the expression of this rhythmic precipitation is very commonly found. Gall stones, pearls, denticles show a concentric lamellated structure (see Fig. 39). In all these cases the inorganic salts that originally were in solution in the surrounding medium are not precipitated uniformly but in cycles, thus causing the presence of zones in the calcified material.

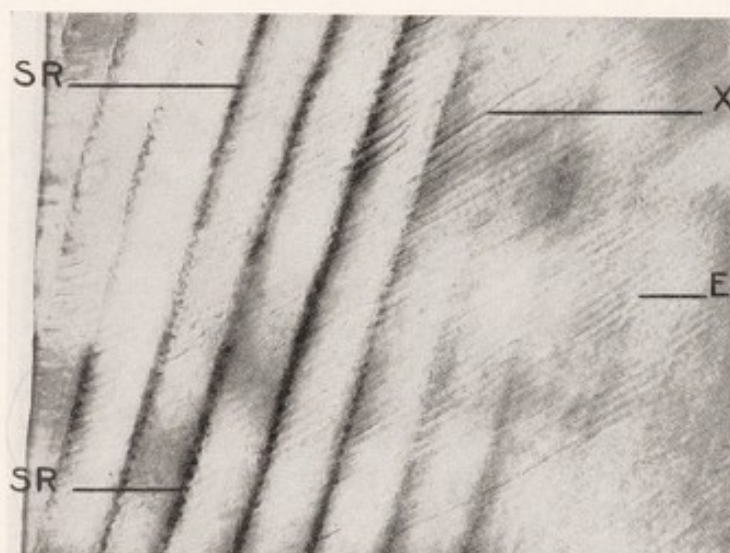


FIG. 56.—Marked stripes of Retzius in the superficial layer of enamel in the periphery of a "white spot." SR, stripes of Retzius; X, beginning decalcification of the interprismatic substance in the enamel between the stripes of Retzius; E, normal enamel.

The same is true of the calcifying enamel. The enamel calcification is a process of precipitation of inorganic salts into a colloidal matrix. If the supply of these salts is abundant, the precipitation will be rather constant, and no stripes of Retzius will be visible. If, however, the supply of mineral salts is limited, zones of abundant precipitation will alternate with zones of poorer calcification; then this rhythmic deposition will find its morphological expression in the stripes of Retzius.

During decalcification of the enamel by caries the stripes of Retzius will become very marked through the loss of inorganic salts; they are then found in the involved strata of enamel only; whereas, they are almost or entirely invisible in the deeper intact layers of enamel.

Fig. 56 shows the appearance of the stripes of Retzius in a very early stage of superficial decalcification of the enamel. This condition can hardly be called caries, although an actual loss of inorganic salts seems to have taken place. From this illustration it can be plainly seen that the stripes of Retzius are the optical expression of the fact that each individual enamel prism, at regular intervals, shows a dark zone of poorer calcification. All these zones together appear as lines. In some places beginning decalcification of the interprismatic substance can be noticed.

In case of active enamel caries on a smooth surface of the tooth, the lesion usually extends into the enamel in the form of a cone (Fig. 57) with the base located on the enamel surface, and the tip pointing toward the dentin. On the enamel surface bacterial inva-

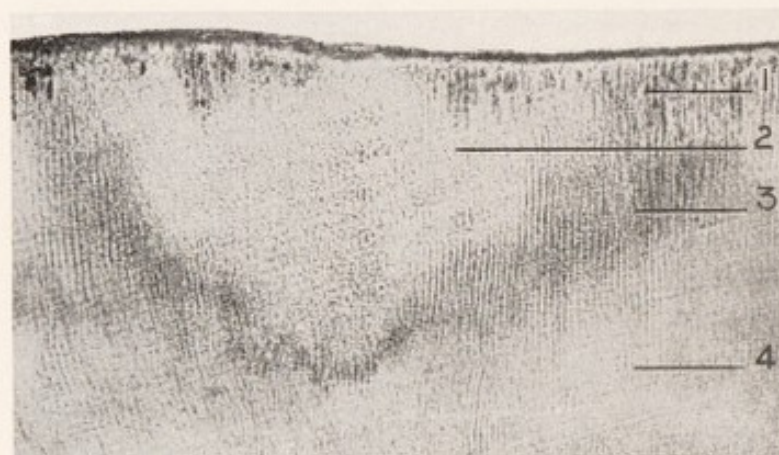


FIG. 57.—Decalcification of the enamel ("white spot") on the approximal surface of a bicuspid. The area of decay in the enamel has the shape of a cone with the base on the enamel surface and the tip pointing toward the dentin. 1, area of bacterial invasion of the enamel; 2, loss of prism structure; 3, transverse striation; 4, normal enamel.

sion and beginning disintegration of the decalcified enamel prisms can be noticed; this is followed by a zone in which the rod structure is partly lost; then, next to the intact enamel, follows a zone of transverse striation as an expression of just beginning calcification.

Sometimes the triangular shape of the decalcified area is due to the presence of an enamel lamella, along which the progress of the decay is faster than in the surrounding enamel.

All the conditions illustrated in Figs. 53 to 57 correspond to what is clinically known as "white spots" or "brown spots." They are conditions of actual enamel decalcification without loss of substance of the enamel surface. Whether such a decalcified area is white or brown depends largely upon the age of the patient and the rapidity of the destructive process. In children, the decalcified spots appear

white, as the involved enamel has lost its normal translucency and has become opaque and chalky. Such areas of superficial decalcification may develop into true carious defects, or they may remain stationary for a long time, even for a lifetime. If the process of caries is slowed down considerably or completely arrested, the white spots gradually become brown by pigmentation. The latter is observed, for instance, when a tooth has been extracted, thereby exposing a caries mark on the adjacent surface of a neighboring tooth. Then the lesion lies in a self-cleansing area, the decay usually stops, and a brown spot develops. Whether such superficially decalcified areas of the enamel can actually become recalcified is still under discussion. Some investigators claim that such a repara-

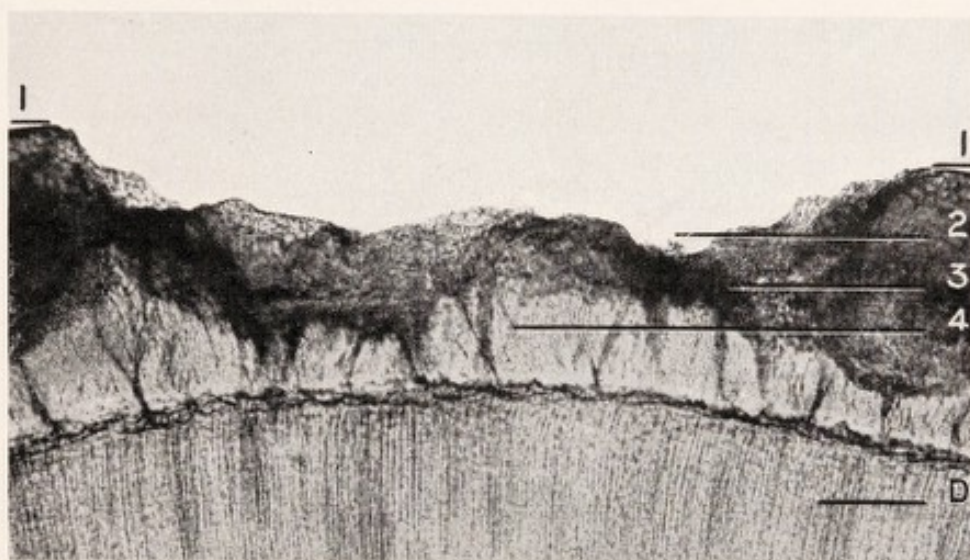


FIG. 58.—Caries of the enamel. Shallow defect in the enamel. 1, original enamel surface; 2, defect in the enamel; 3, dark brown pigmentation of the carious enamel; 4, enamel showing transverse striation; D, dentin.

tive calcification is possible (Pickerill, Head; Andresen's theory of remineralization of the enamel), while others deny this possibility or at least do not consider recalcification of the enamel as proved (Weber). The author does not believe that an actual "remineralization" of such an early carious lesion is possible. The brownish spots that are observed where superficial decay has once started appear under the microscope as a very slow, chronic form of caries; therefore, it would appear more appropriate to speak of "arrested caries" than of "healed caries" or "recalcification."

As the process of caries reaches the dentin the enamel surface is usually already destroyed to such extent that with the tip of an explorer a marked roughness or softening of the enamel will be noticed, due to the loss of the superficial enamel rods following decal-

cification of the interprismatic substance (Fig. 58). If the surface of the decayed and softened enamel is examined under high magnification, it can be seen how the prisms separate, apparently as a result of the decalcification of the interprismatic substance (Fig. 59); finally the peripheral ends of the prisms fade away in the mass of bacteria and detritus overlying the carious enamel.

In summarizing, the histological picture of acute caries of the enamel would usually show the following changes in the enamel rods: near the outer surface of the enamel a complete decalcification

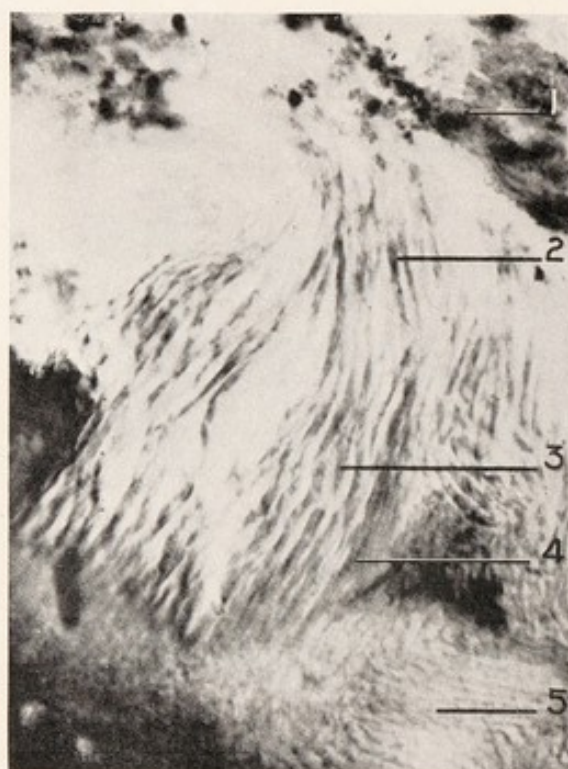


FIG. 59.—Decalcification and disintegration of the enamel prisms. Loss of interprismatic substance. 1, bacteria and detritus; 2, disintegration of the prisms; 3, enamel prisms separated through decalcification of the interprismatic substance; 4, transverse striation of the prisms; 5, intact enamel. (Decalcified section.)

and softening of the prisms, nearer the dentin surface beginning decalcification and loss of prism structure, and nearest the dentin surface transverse striation of the enamel rods (earliest evidence of decalcification). It must be emphasized, however, that these different stages cannot always be found, nor do they always occur in a set order; certain deviations can be observed, depending upon the individual case.

(b) *Caries of the Grooves and Fissures.*—Caries of the grooves and fissures presents itself somewhat differently from caries of the smooth surfaces. The destructive process spreads rapidly from the bottom

of the fissure into the surrounding enamel and soon reaches the dentin, which is usually not far away from the deepest point of the fissure.

The microscopic examination of the occlusal surface of normal human teeth reveals wide variations in the depth of the fissures. Sometimes the fissures are rather shallow, so that a thick layer of enamel separates the bottom of the fissure from the dentin, while in other instances very deep fissures are found; then the bottom

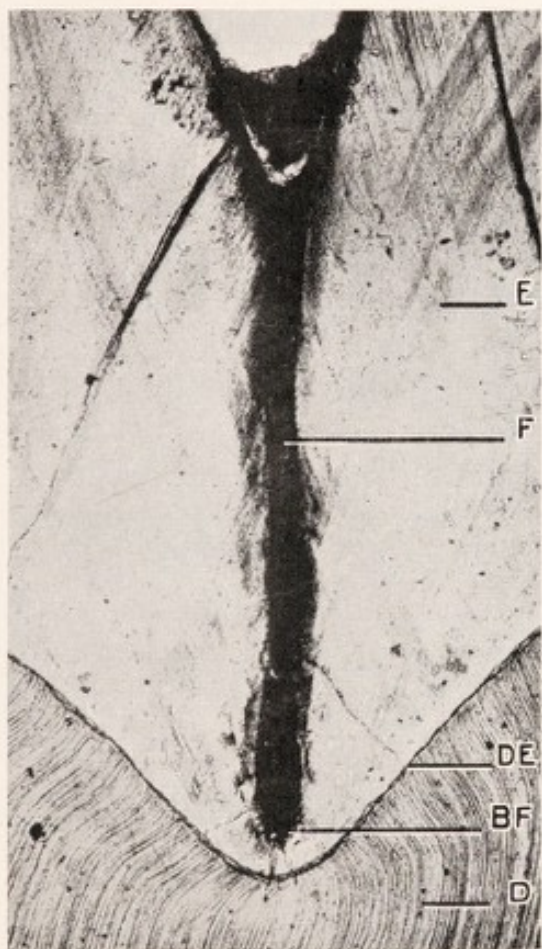


FIG. 60.—Deep intact occlusal fissure in a lower molar. E, enamel; F, fissure filled with detritus; BF, bottom of fissure; DE, dento-enamel junction; D, dentin.

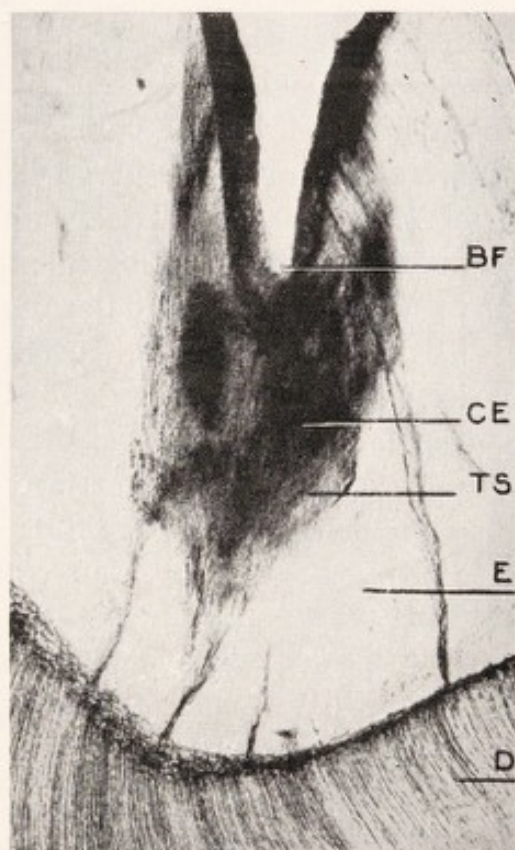


FIG. 61.—Beginning caries of the enamel at the bottom of an occlusal fissure. BF, bottom of fissure; CE, caries of the enamel; TS, transverse striation of the enamel prisms; E, normal enamel; D, dentin.

of the fissure is located almost at the dento-enamel junction. Fig. 60 shows such a deep fissure in a lower first molar. The fissure is filled with detritus, and the surrounding walls of enamel are intact except for a minute pigmentation in some places.

In view of the occurrence of fissures of the type illustrated in Fig. 60, one is again reminded of the extreme difficulty of giving a satisfactory explanation of the problem of dental caries. There is no doubt that such a steep, narrow cleft in the enamel presents an

ideal breeding place for microorganisms. Still in some mouths such fissures or deep grooves stay intact throughout life, while in other individuals all occlusal fissures show extensive decay.¹ It may be appropriate to recall in this connection the complexity of factors that may be involved in the development of dental decay: character of saliva, variations in the types of microorganisms of the oral cavity, condition of the enamel cuticle, condition of the enamel lamellæ, and structure and calcification of the enamel. It seems reasonable to assume that no one of the above-mentioned factors can be held solely responsible, but that susceptibility and immunity to dental caries are the result of the combined action of two or more of these etiological factors. At any rate, from a microscopic examination of fissures, one gains the impression that there is nothing in the structure of the fissures that can be used to explain the presence or absence of decay; all deep fissures present an area of predisposition to decay, and the histological structure does not give an explanation of why some of them do decay and others do not.

As already mentioned, occlusal caries usually originates at the bottom of the fissure (Fig. 46). From here the process of decalcification follows the general direction of the enamel prisms, so that the changes in the prisms are the same as in caries of a smooth enamel surface (see page 82): complete destruction of the prisms at the bottom of the fissure, next a zone of decalcification of the interprismatic substance, then a zone of transverse striation, and finally the normal layer of enamel next to the dento-enamel junction (Fig. 61). Since the enamel prisms diverge from the bottom of the fissure toward the dento-enamel junction, the decayed enamel usually forms an approximate cone. The tip of the cone is located at the bottom of the fissure, its base at the dento-enamel junction (Fig. 62). In addition, the decay spreads from the walls of the fissure so that sooner or later the superficial layers of enamel are undermined and lost, with resulting widening of the fissure and roughening of the enamel margins (Fig. 63). In other cases, however, the enamel in the occlusal portion of the fissure stays intact at first; the decalcification and destruction of the enamel spreads for a considerable distance along the dento-enamel junction, undermining the clinically healthy-appearing enamel around the opening of the fissure. Thus we find cases in which the tip of the explorer after passing an apparently intact or only slightly discolored or chalky fissure encounters extensive softening and decay of the deeper strata.

Because of the rapid spreading of decay along the dento-enamel

junction, the enamel may become decalcified from within (secondary caries of the enamel). This process accelerates the undermining and breaking-in of the non-supported enamel.

Due to the close relationship between the fissures and the underlying dentin, the latter is usually involved very early. Most of the cases of occlusal decay of the enamel show transparency of the dentin, and frequently beginning decalcification and caries of the dentin.

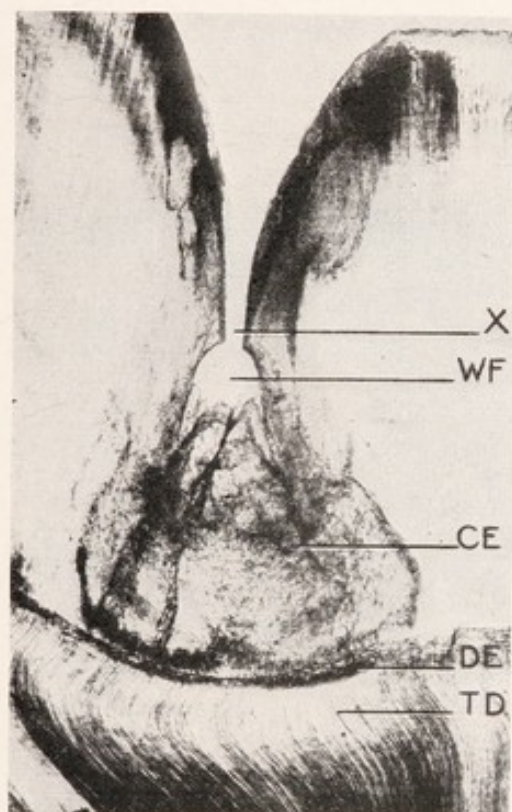


FIG. 62.—Caries at the bottom of an occlusal fissure. X, narrowest point of the fissure; WF, widening of the fissure caused by destruction of the enamel; CE, cariou enamel at the bottom of the fissure; DE, caries reaching the dento-enamel junction; TD, transparent dentin.

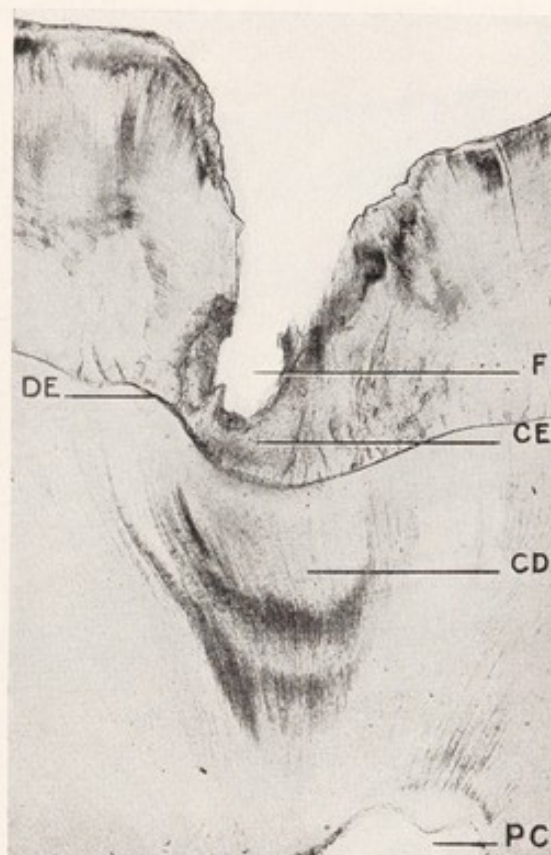


FIG. 63.—Advanced caries of an occlusal fissure of a lower molar. F, fissure widened by destruction of the enamel; CE, cariou enamel of the bottom of the fissure; DE, dento-enamel junction; CD, caries of the dentin; PC, pulp chamber.

3. Caries of the Dentin.—(a) *Significance of the Enamel Lamellæ in the Early Development of Caries of the Dentin.* *Early Stages of Caries of the Dentin.*—Caries of the dentin is unique because of the rôle that the dentinal tubules and their contents play in the progress of the decay. In the enamel a considerable amount of acidity is necessary to decalcify the prisms and to make possible the involvement of deeper strata; in the dentin the pathways for the invasion and the spreading of microorganisms are predetermined,

and it is, therefore, not surprising that acute caries of the dentin shows a much more rapid course than caries of the enamel. On the other hand, the very same structures in the dentin that facilitate the spreading of the decay, namely, the 'Tomes' fibers, are capable of producing a defense reaction consisting of the formation of a calcified barrier. Thus, caries of the dentin of a tooth with a healthy pulp differs basically from caries of the enamel in that the enamel appears morphologically inert and does not show any evidence of a vital reaction to the advancing microorganisms, while the living dentin reacts with a specific protective mechanism.

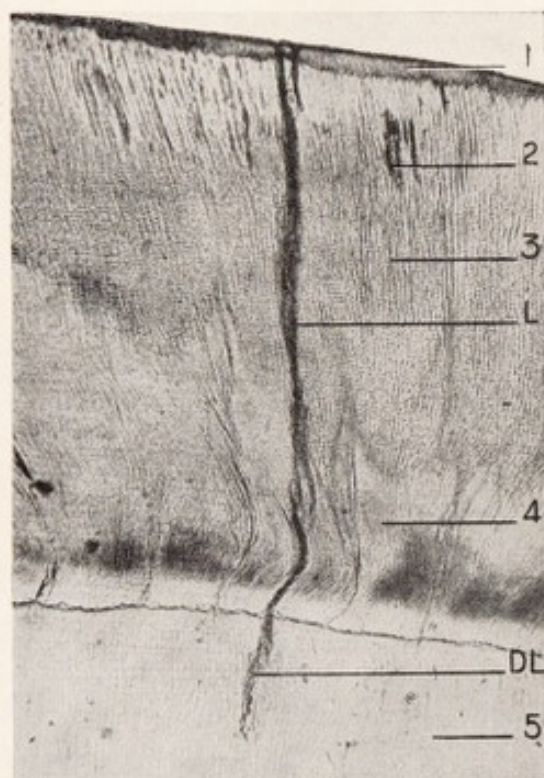


FIG. 64.—Transmission of enamel caries to the dentin by way of a lamella. 1, superficial structureless layer of carious enamel; 2, zone of bacterial invasion; 3, zone of transverse striation; 4, normal enamel; 5, dentin; L, enamel lamella; DL, dentinal part of the lamella.

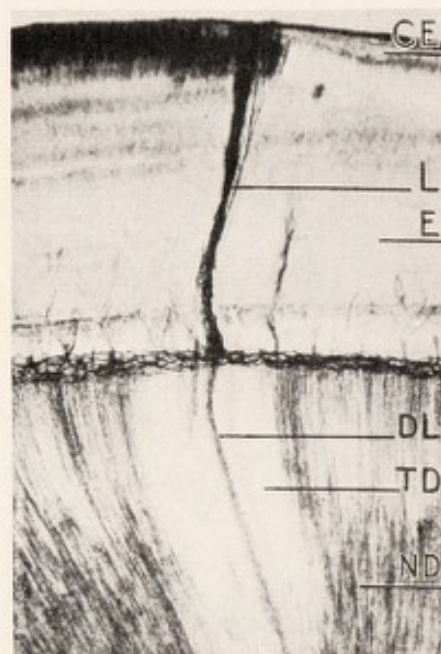


FIG. 65.—Transparent dentin around the dentinal part of an enamel lamella. CE, superficial caries of the enamel; E, normal enamel; L, enamel lamella; DL, dentinal part of the lamella; TD, transparent dentin; ND, normal dentin.

Since the enamel lamellæ are of great importance to the onset of caries of the dentin, a few specimens illustrating lamellæ and dentin caries will be illustrated here, and in this connection the changes in the dentin will be discussed.

In Fig. 64 the relationship between enamel caries, lamella, and dentin is very plain. The enamel shows the various zones that were described as being characteristic of early carious lesions: a struc-

tureless, brown layer of the surface followed by a stratum of enamel that is invaded by microorganisms along the prisms. In the next layer toward the dentin follows a broad zone of transverse striation as an expression of the penetration of acid into this part of the enamel. A lamella runs through the enamel and into the dentin. Around the dentinal portion of this lamella the tubules are slightly discolored, indicating the very beginning of caries of the dentin. It can readily be assumed that microorganisms from the infected superficial layer of enamel can pass into the deeper intact enamel strata and in that way reach the dentin, where they find much less resistance to their decalcifying action.

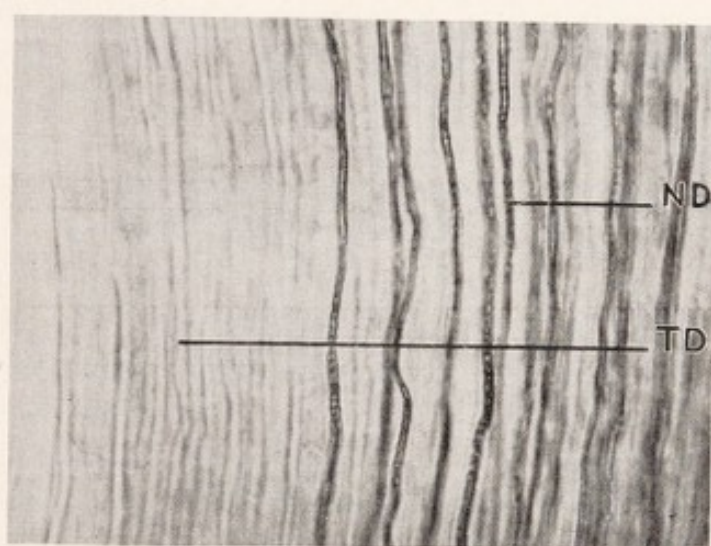


FIG. 66.—High magnification of the border between normal and transparent dentin in Fig. 65. ND, normal dentin; the tubules contain air and appear dark in transmitted light; TD, transparent dentin; the lumen of the tubules is obturated by calcium salts. The tubules appear light in transmitted light.

The reaction of the dentin to the irritation that is conveyed by an enamel lamella is a condition that is called transparency of the dentin (Miller) (Fig. 65). The term transparency originated from the appearance of ground sections of dentin; in such sections the areas of irritation appear lighter than the surrounding dentin. In order to understand this phenomenon it is necessary to discuss briefly the optical conditions present in dentin specimens. After normal dentin has been ground, treated with alcohol and ether, and embedded in balsam, the tubules contain either air, remnants of Tomes' fibers, balsam, or, if the section has been stained, they appear colored. At any rate, the substance contained in the lumen of the tubule will have a different optical refraction from that of the surrounding matrix; hence the tubule will appear as a

dark line in a light matrix (Fig. 66). In case of irritation or injury to the dentin, the 'Tomes' fibers will become calcified; that is, the calcium salts that are normally in solution in the content of the tubules will be precipitated and the tubules will be obliterated by inorganic material. As a result, the difference between the optic refraction of the tubules and of the matrix is greatly decreased and the dentin becomes transparent (Fig. 66, *TD*). Beust reported a difference in the penetration of dyes into the dentin under different conditions. While the dentin of young teeth stained readily from the pulp chamber outward to the dento-enamel junction, he found in older teeth areas which could not be entered by staining solutions. Beust called these transparent areas sclerosed dentin, as they are the result of a precipitation of inorganic salts into the tubules. He found this change under abrasion as well as under caries; in either case the sclerosis is indicative of a defense reaction of the 'Tomes' fiber to external irritation. Beust also called attention to the fact that sclerosis of the dentinal tubules is invariably found, though to a varying extent, in teeth of individuals of advanced age, regardless of outer irritation. Here the areas of transparency are an expression of the gradual decrease of dentin vitality concurrently with the reduction of the size of the pulp chamber and regressive changes in the pulp tissue (see page 40). It seems very reasonable to assume that the slower progress of caries in the dentin of old teeth may have something to do with the increased calcification and decreased organic content of older dentin. Since sclerosis of the dentin always accompanies caries of teeth with vital pulps, Beust comes to the conclusion that "an irritant acting on a fibril commonly causes a reaction within the tubule leading to its complete obliteration. Caries of the dentin, therefore, cannot be regarded as a simple proteolysis of dead tissue." It should be understood that in the dentin of a pulpless tooth caries actually is a simple decalcification and proteolysis; the bacteria enter the dead dentin and cause its dissolution without any defense reaction on the part of the organism.

By treating areas of transparency around the dentinal part of enamel lamellæ with an alcoholic solution of diamond-fuchsin, Orban found that the dye entered all dentinal tubules and their ramifications except for the zones of transparency in which the tubules did not stain at all. This indicated that these tubules were completely obstructed; their staining properties were not influenced by treating the section with ether, which proved that no fatty or lipid substance was present in the tubules. The con-

tent of the tubules in the transparent area had the same optical properties as the dentin between the individual tubules.

A simple comparison may help to make the optical difference between normal and transparent dentin clear. If a glass tube containing air is submerged in water, it is plainly visible due to the difference in the optical properties of water and air. If the air is let out and water enters the glass tube, this difference is greatly decreased; the glass tube is then hardly visible. The air-containing tube corresponds to the normal dentin with the optical difference between non-calcified tubules and calcified matrix. The tube containing water represents transparent dentin; the optical properties are homogeneous throughout.

In caries of the dentin, transparency of the dentin in the periphery of the carious lesion is always found if the tooth under consideration has a vital healthy pulp. The amount of transparency depends upon the individual circumstances: age of the tooth, condition of the pulp, rate of progress of caries. In that respect, transparency of the dentin is rather similar to secondary dentin formation: both occur as a reaction to irritation or injury; both consist of the formation of a calcified barricade; both occur more extensively if the tooth is young and if the progress of the irritation is slow.

Recent investigations concerning the changes in the dentinal tubules have revealed that the precipitation of calcium salts is not the earliest expression of irritation. Rather has it been shown by staining ground sections or frozen sections through transparent dentin with fat stains (Sudan, scarlet red) that the calcification is preceded by the appearance of fat droplets in the tubules. Weber, Euler, and Meyer have published specimens in which in the periphery of sclerosed areas the content of the tubules stained bright red with Sudan, indicating the presence of fat. Thus, in Tomes' fibers in case of irritation, the zones are (in order from the periphery toward the pulp): calcification (sclerosis), deposition of fat, normal portion of the fibers.

Once caries has involved the enamel, it, as a rule, is transmitted to the dentin by way of the enamel lamellæ. In stained sections the invasion of the organic substance of the lamellæ can be plainly seen. Sometimes the enamel surrounding the lamella is slightly decalcified, showing transverse striation or discoloration; in other cases the enamel around the lamella is not altered, and the decay involves only the dentin at the point of attachment of the lamella. Fig. 67 shows an early stage of dentin caries that was transmitted from the superficially decayed enamel by a lamella. On the surface

the enamel is brown and discolored, and in some places small defects are present. Slightly deeper the enamel shows transverse striation indicative of beginning decalcification; near the dento-enamel junction the enamel is normal. At *L* a lamella runs through the enamel; at the point where the lamella reaches the dentin, caries spreads along the dento-enamel junction and into the peripheral portion of the dentinal tubules. The infected tubules are surrounded by an area of transparency in the periphery of which normal tubules are visible.

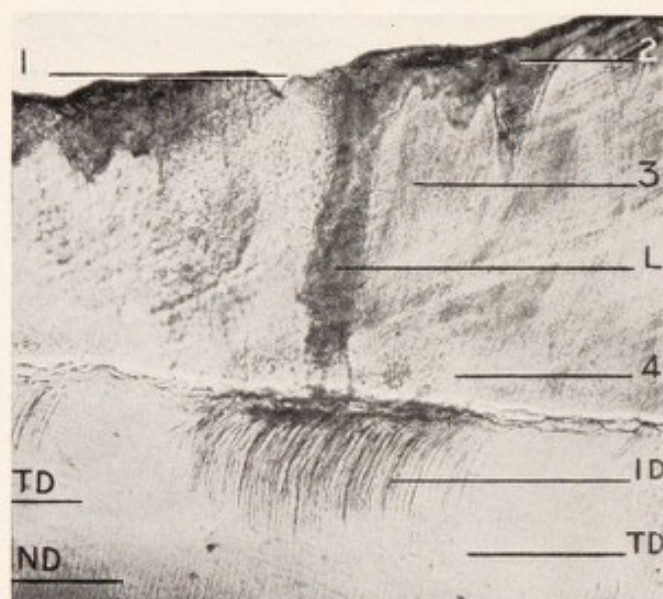


FIG. 67.—Beginning caries of the dentin, spreading from the dentinal end of an enamel lamella. 1, superficial defects in the enamel; 2, decalcification and discoloration of the enamel; 3, transverse striation; 4, normal enamel; L, enamel lamella; ID, infected dentinal tubules; TD, transparent dentin; ND, normal dentin.

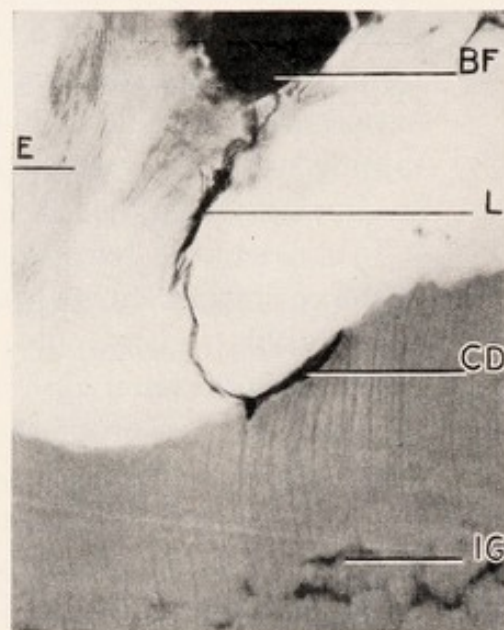


FIG. 68.—Enamel lamella at the bottom of an occlusal fissure of a molar. Caries is transmitted through the lamella from the fissure to the dento-enamel junction. Decalcified stained section. BF, bottom of occlusal fissure filled with detritus; L, enamel lamella; E, enamel; CD, caries of the dentin spreading along the dento-enamel junction; IG, infected interglobular spaces. (Barker, Jour. Am. Dent. Assn.)

In decalcified, embedded sections, the close relationship between lamellæ and dentin caries can be plainly demonstrated. Fig. 68 shows a lamella running from the bottom of an occlusal fissure to the dento-enamel junction. The specimen was prepared from the lower first molar of a child; through the preparatory decalcification of the specimen the enamel was lost, and the lamella appears as a strand of organic tissue. Where this strand reaches the dentin, caries spreads along the dento-enamel junction and into the adjacent dentinal tubules. The interglobular spaces in the dentin are also invaded by microorganisms. Barker has shown that in poorly

calcified dentin the areas of uncalcified matrix between the calcified globules are easily attacked by caries and offer little resistance to the spreading infection of the dentin.

The first symptom of actual caries of the dentin is the invasion of the tubules by microorganisms; next follows the decalcification of the surrounding matrix by the acid produced by these microorganisms. The finer microscopic details of the infection and decalcification of the dentin will be shown in later illustrations; first, however, will be mentioned a phenomenon that is of greatest practical importance, namely, the retraction of the decalcified dentin from the inner surface of the enamel and the formation of spaces between enamel and dentin.

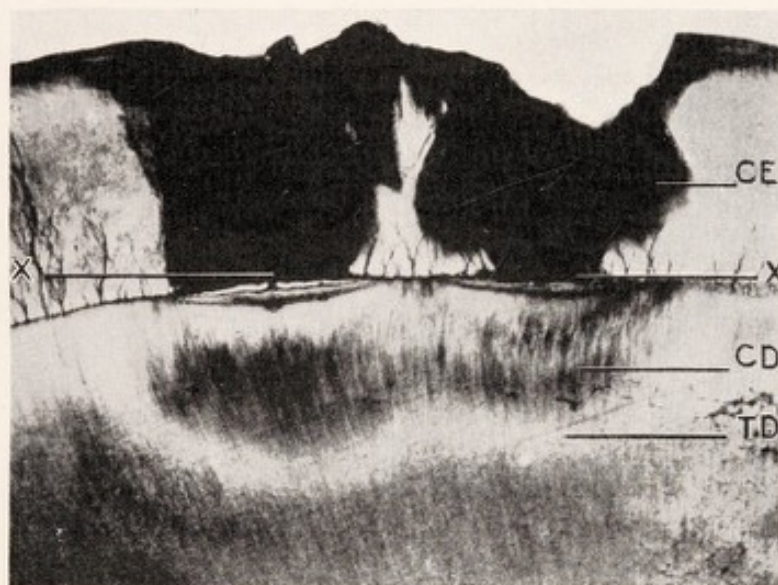


FIG. 69.—Caries of enamel and dentin. Shrinkage of the decalcified dentin; the dentin has retracted from the lower surface of the enamel, thus causing the formation of gaps between enamel and dentin. CE, caries of the enamel; CD, carious dentin; X, gap between enamel and dentin; TD, transparent dentin. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

The decalcification of the dentin matrix causes a reduction of volume and shrinkage of the matrix. The direction of shrinkage is at right angles to the dentin surface, parallel to the course of the tubules. Since the overlying enamel is less affected than the dentin, empty spaces form at the dento-enamel junction which are bordered by biconvex walls (Figs. 69 and 70). The presence of these spaces is of great practical importance since it accounts for the ease with which the enamel overlying decayed dentin breaks under the pressure of a chisel or enamel hatchet. The dentin actually separates and retracts from the enamel, depriving the enamel of its mechanical

support. In studying Fig. 70 it can easily be understood that, although the actual opening through the enamel is small, the entire enamel overlying the decalcified and retracted dentin will break easily under slight pressure.

(b) *Advanced Changes in Carious Dentin.*—As caries penetrates into deeper layers of the dentin, the same order of changes in the tubules can be observed that was originally found near the dentin surface. Fig. 71 shows a portion of a stained ground section through carious dentin. To the left of this illustration, dark masses of decomposed dentin cover the surface of the carious cavity. Next are visible dentinal tubules that appear dark brown because of the presence of microorganisms. The next zone in the direction of the

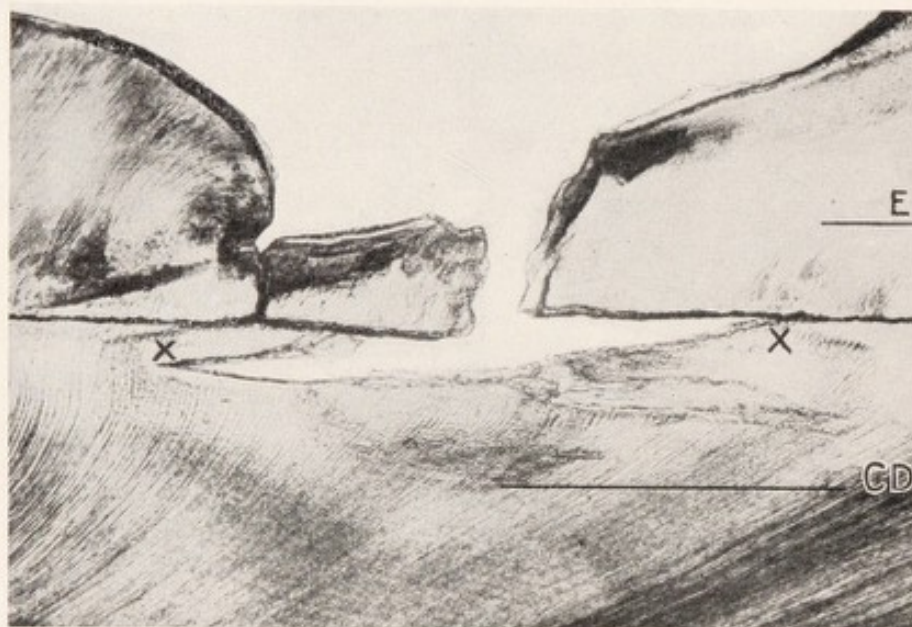


FIG. 70.—Caries of dentin undermining the overlying enamel through retraction and shrinkage of the decalcified dentin matrix. E, enamel; X-X, space between enamel and dentin; CD, carious dentin.

pulp is one of transparency or sclerosis, indicating a disturbance in the metabolism of the Tomes' fibers with a precipitation of calcium salts in the tubules. Finally to the right, normal tubules are visible appearing black, due to the presence of air that entered during grinding. These various zones are especially distinct in the specimen illustrated in Fig. 72, which represents a stained ground section through a deep occlusal carious lesion in a molar tooth. The dentin in the periphery of the decayed area appears normal (Fig. 72, 5), but further toward the surface the dentinal tubules appear darker (Fig. 72, 4); this zone corresponds in its position and arrangement to the zone of fatty degeneration of the Tomes' fibers that has been

described by some investigators. This zone is followed by a very distinct light area of transparency that encircles the entire decayed dentin (Fig. 72, 3). The latter appears dark, since the bacteria and the decomposed content of the tubules assume a dark purple or brownish color when the section is stained (Fig. 72, 2). The cavity itself is lined by decomposed dentin (Fig. 72, 1). The study of sections like that illustrated in Fig. 72, together with decalcified embedded sections through carious dentin, has revealed that at least five distinct zones can be differentiated in the dentinal tubules of carious dentin. They are:

1. A zone of complete decalcification of the walls of the tubules and of the matrix with subsequent decomposition of the decalcified matrix.

2. A zone of beginning decalcification of the content of the tubules by acids produced by

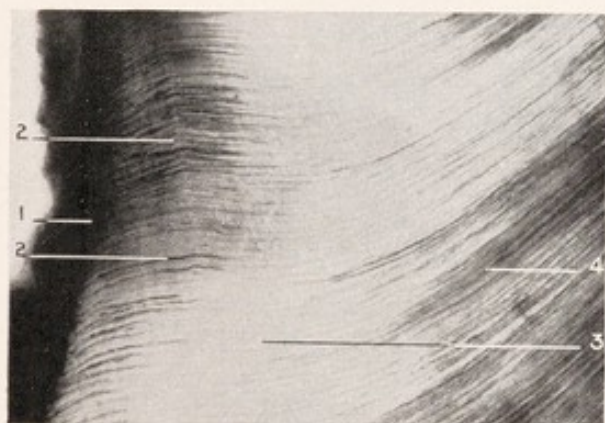


FIG. 71.—Caries of the dentin. Stained ground section. 1, structureless decomposed dentin at the bottom of the carious cavity; 2, dentinal tubules invaded by microorganisms; 3, zone of transparent dentin; 4, normal dentinal tubules.

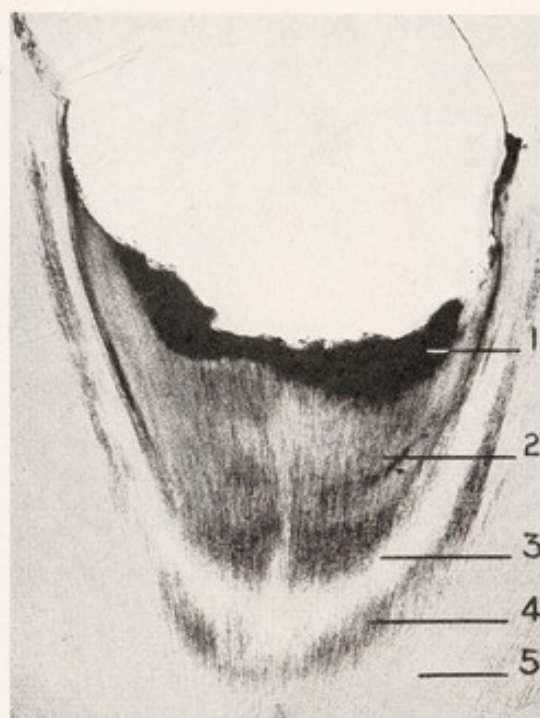


FIG. 72.—Caries of the dentin. Stained ground section showing the various zones in the dentin. 1, decomposed softened dentin at the bottom of the carious cavity; 2, zone of decalcification of the tubules and invasion by microorganisms; 3, zone of transparency; 4, zone of fatty degeneration and beginning calcification of Tomes' fibers; 5, normal dentin.

microorganisms and subsequent invasion of the tubules by these organisms.

3. A zone of complete obliteration of the tubules by calcification (sclerosis) of Tomes' fibers (zone of transparency).

4. A zone of fatty degeneration and beginning precipitation of calcium droplets within the protoplasm of Tomes' fibers.

5. A zone of undisturbed dentinal tubules containing normal Tomes' fibers.

Stages 3 and 4 are the result of the disturbed metabolism of the Tomes' fibers as a reaction to a distant irritation; stages 1 and 2 are the result of the actual bacterial invasion and decalcification of the dentin.

In teeth without vital pulps stages 1 and 2 only can be observed; the microorganisms spread rapidly along the open empty dentinal tubules and are followed by decalcification and destruction of the surrounding matrix.

The minute changes associated with the infection of the dentin can be studied best in decalcified sections through dentin stained with Gram. In such sections those tubules that have been invaded by microorganisms are distinctly different from the surrounding, non-infected tubules. Fig. 73 shows a section through a small,

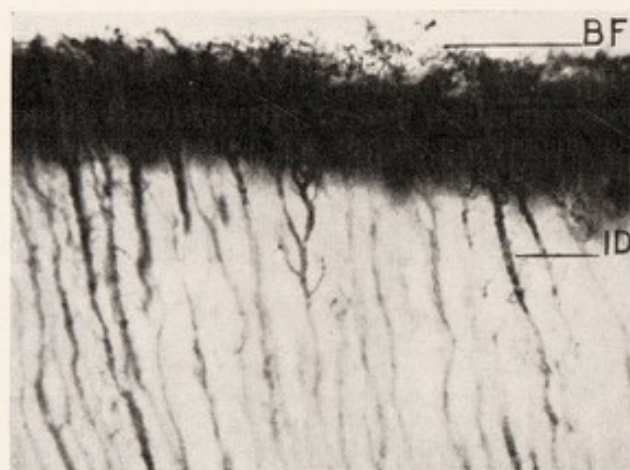


FIG. 73.—Bacteria invading the dentinal tubules at the dento-enamel junction. BF, bacterial film on the dentin surface; ID, infected dentinal tubules and ramifications.

carious lesion at the dento-enamel junction. The enamel has been destroyed; the surface of the dentin is covered by a dark film of microorganisms that have begun to enter the endings of the dentinal tubules. Thus the tubules and their terminal ramifications appear dark against the lighter matrix. In the deeper layers of dentin the bacteria can be seen travelling along some of the tubules, while the surrounding tubules are still free of infection (Fig. 74). Furrer called these first microorganisms "pioneers," which term appears to be well chosen considering the fact that these bacteria actually appear like scouts preceding the large army of dentin-infecting bacteria. Soon more and more tubules become infected; we then see conditions like those in Fig. 75, in which all tubules in an area of dentin are packed with microorganisms.

Observation of the pioneer bacteria in the dentin raises a ques-

tion of great practical importance. In excavating carious dentin from a cavity, the clinical borderline between decayed and healthy dentin is drawn according to hardness: as long as the dentin is so soft that it can be cut with the excavator it should be removed;

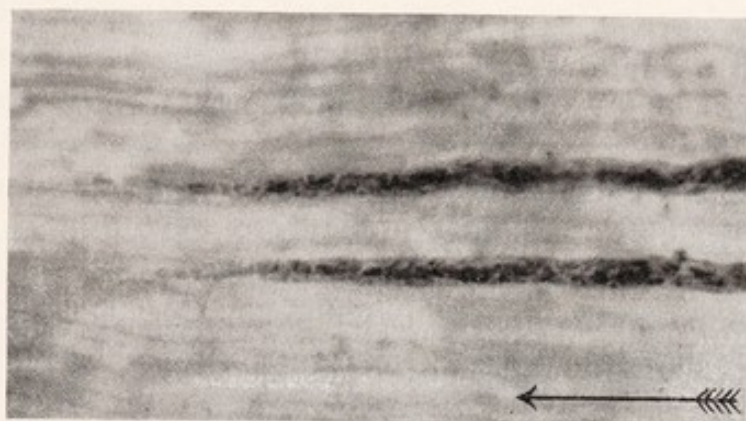


FIG. 74.—Beginning invasion of the dentin by microorganisms. Two dentinal tubules contain "pioneer" microorganisms. The caries progresses in the direction indicated by the arrow.

when the dentin is so hard that the instrument gives a clicking sound and does not cut easily, it is considered healthy. From the microscopic study of carious dentin we know that the zone of bacterial invasion does not coincide with the zone of softening and decalcification of the dentin: the bacteria in the tubules are always

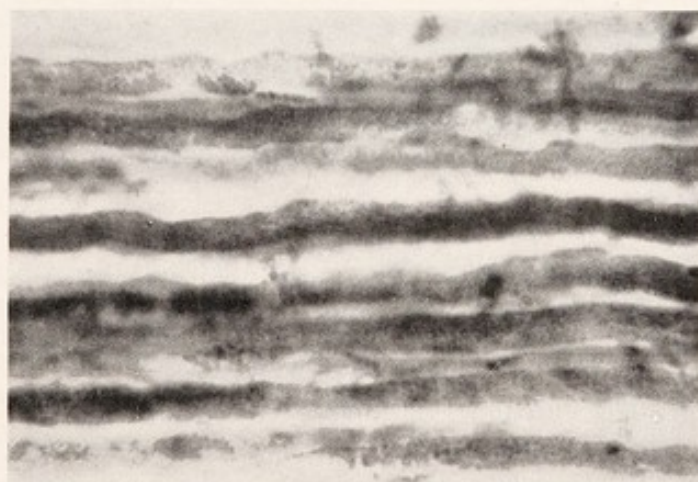


FIG. 75.—Generalized infection of the dentinal tubules. Each tubule is packed with microorganisms.

ahead of the actual decalcification of the matrix. The hard dentin at the bottom of an excavated carious cavity, in all likelihood, is infected. On the other hand, we know from clinical experience that if such a cavity is properly filled, no further decay of the

dentin occurs despite the presence of bacteria in the dentin at the bottom of the cavity. What is it then that stops the progress of the pioneer microorganisms in the tubules below the filling? It must be assumed that lack of moisture, lack of food supply, change in oxygen tension, and discontinuation of the supply of new bacteria from the carious cavity are sufficient to overcome the microorganisms in the deeper tubules. In addition, the pulp throws up a barricade by calcification of the tubules and by secondary dentin formation; thus, the bacteria are enclosed between the filling on one side and the reactive calcification on the other side and cannot spread any further. However, in view of the presence of these "pioneers" in the tubules at the bottom of the cavity, the application of a dentin disinfectant, such as phenol or mercury bichloride, before insertion of the filling appears well justified.



FIG. 76.—Beginning distention of the infected tubules by the growths of bacteria and by the decalcification of the surrounding dentin matrix. Beaded appearance of the infected tubules.

The microorganisms in the dentinal tubules, being an acid-producing type, decalcify the matrix that forms the walls of the tubules; this decalcified matrix is then destroyed, probably by proteolytic ferments produced again by the bacteria. In some of the tubules the bacteria form colonies which, by their growth, distend the tubule at the expense of the surrounding softened matrix (Fig. 76). The tubules assume a beaded appearance. As dentin decalcification and destruction progresses, the matrix between the distended tubules is completely destroyed and transformed into a soft, crumbly mass; oblong cavities form in the dentin parallel to the direction of the tubules. They are the result of the breaking down of the matrix and of the coalescence of a number of infected tubules. In

Fig. 77 this process of cavity formation within the carious dentin is illustrated. On the right side of the figure, many infected tubules can be seen. The tubules coalesce and, by morbid dilatation of the walls, form a larger cavity that is filled with broken-down matrix and bacteria. That such cavities actually grow by expansion rather than by dissolution of the walls is indicated by the condition of the tubules in the surrounding decalcified dentin. The tubules in the periphery of the cavity show a marked deviation from their normal straight course; at the same time their width is reduced by compression. Both deviation and narrowing of the tubules are infallible symptoms that the dentin in this area, although otherwise



FIG. 77.—Cavity in the dentin formed by coalescence of infected dentinal tubules. The cavity contains detritus and bacteria. C, cavity; ID, infected dentinal tubules; X, deviation and compression of the tubules in the decalcified dentin in the periphery of the cavity.

of normal appearance, has been decalcified and has a soft cartilaginous consistency. One would almost be tempted to speak of an abscess of the dentin, were it not for the fact that no cellular elements (pus) are present in the cavity, only necrotic dentinal matrix and bacteria.

Eventually the individual areas of breaking-down dentin coalesce, and the entire dentin is transformed into a soft, structureless mass. In a photograph of decayed infected dentin under high magnification, this transformation can be plainly seen (Fig. 78). On the left-hand side of this illustration, the dentin contains numerous infected tubules. Close to the surface these tubules coalesce, forming large, necrotic cavities; finally, the most superficial dentin is

completely decomposed and structureless. The carious surface is covered by a dense film of microorganisms, chiefly saprophytes, that dispose of the necrotic dentin remnants (Fig. 79).

In many specimens of dentin caries, cavities or clefts can be observed that run at right angles to the course of the dentinal tubules (Fig. 80). This arrangement of the clefts in the dentin results from the course of the fibrils of the matrix. The dentinal matrix is built up of very fine fibrils that run at right angles to the Tomes' fibers, namely, parallel to the dentin surface. In case of decalcification and subsequent shrinkage of the matrix, the layers of fibrils represent lines of cleavage along which gaps may form by

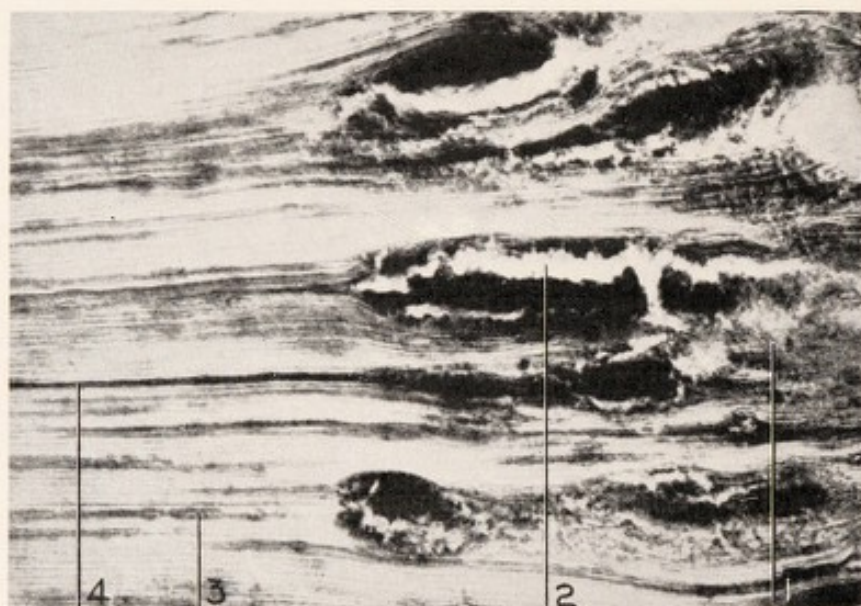


FIG. 78.—Advanced decomposition of the carious dentin. 1, disintegration of the decalcified matrix; 2, formation of cavities by coalescence of the infected tubules; 3, swelling and distention of the infected tubules; softening of the surrounding matrix; 4, infected dentinal tubules.

retraction of the walls. Naturally, these gaps follow the general direction of the fibrils, which is at right angles to the dentinal tubules. If a considerable amount of decayed dentin is present, such as in the case of extensive occlusal decay of a molar, large gaps form in the decalcified dentin parallel to the original dentin surface, while the general progress of caries occurs at right angles to the surface (Fig. 81). Through this arrangement of the clefts in the softened dentin, the removal of the decay by means of excavators is greatly facilitated: the carious dentin peels off in layers or flakes separating easily from the underlying hard dentin.

Finally, it is necessary to consider briefly caries of the secondary dentin. In the preceding chapter the secondary dentin formation



FIG. 79.—Superficial layer of carious and decomposing dentin. C, large necrotic cavity in the softened dentin causing compression and deviation of the surrounding tubules; D, disintegration of the softened dentin matrix through the action of saprophytic micro-organisms.



FIG. 80.—Advanced caries of the dentin. Formation of clefts at right angles to the dentinal tubules. ID, infected dentinal tubules; C, clefts in the dentin containing necrotic masses. At X, the necrotic content of the clefts has fallen out.

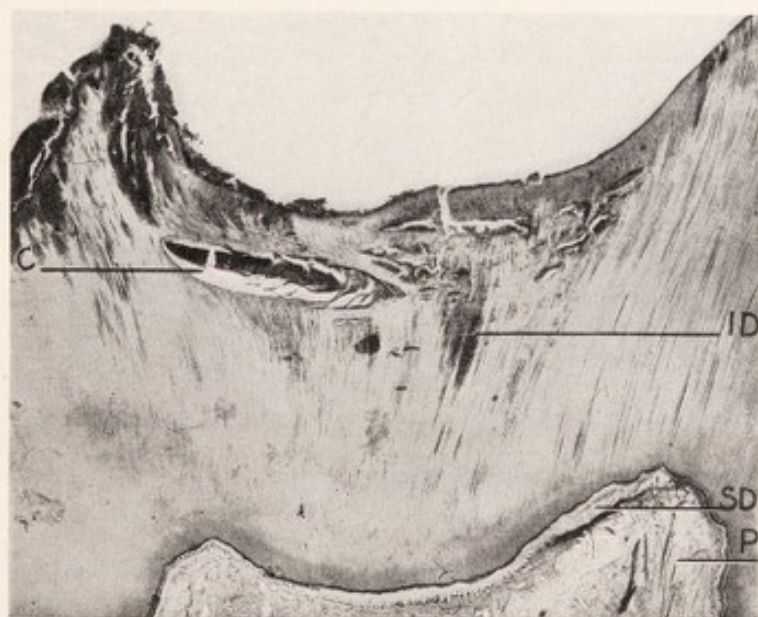


FIG. 81.—Extensive occlusal caries in a lower molar. Formation of clefts in the carious dentin. C, large cleft arranged parallel to the dentin surface; ID, infected dentinal tubules; SD, secondary dentin; P, pulp.

and its significance as a protective measure against caries has been illustrated. As decay progresses through the primary dentin, sooner or later it reaches the border between primary and secondary dentin and involves the latter (Fig. 82). The progress of caries in the

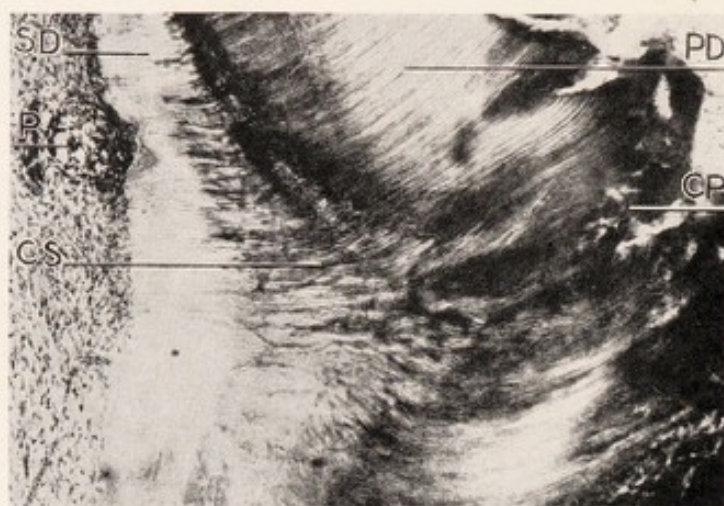


FIG. 82.—Caries of secondary dentin. PD, primary dentin; CP, caries of the primary dentin; SD, secondary dentin; CS, caries of secondary dentin; P, pulp in a state of beginning inflammation (pulpitis).

secondary dentin is usually somewhat slower due to the smaller number of dentinal tubules and better calcification. The micro-

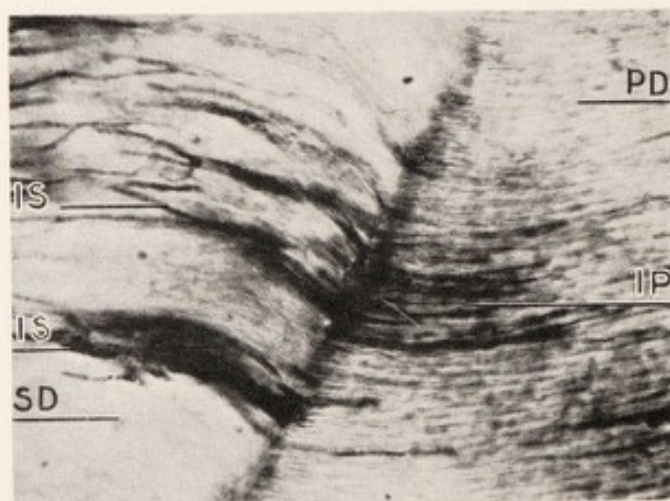


FIG. 83.—Caries at the borderline between primary and secondary dentin. PD, primary dentin; IP, infected tubules of the primary dentin; SD, secondary dentin; IS, infected irregular tubules of the secondary dentin.

scopic appearance of decayed secondary dentin is characterized by the irregularity of the tubules and the resulting irregular distribution of infection (Fig. 83).

In older individuals, in whom part of the root is exposed, caries

of the cementum can be observed. In the primary cementum, the microorganisms follow the course of the fibrils; soon the cementum is decalcified and the underlying dentin is involved (Fig. 84).

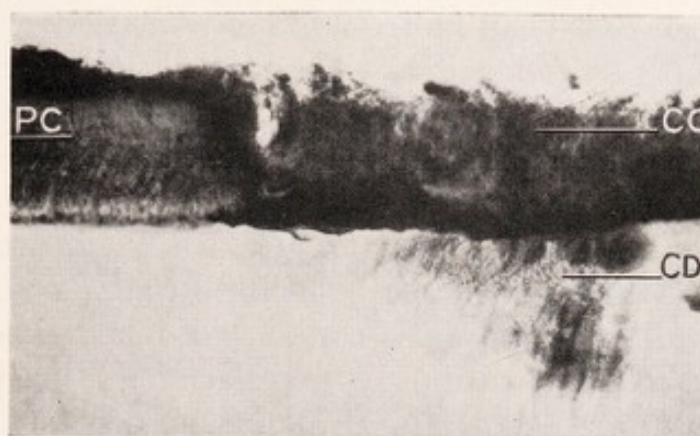


FIG. 84.—Caries of the cementum. PC, primary cementum; CC, caries of the cementum. Disintegration of cementum into its fibrillæ; CD, superficial caries of the dentin.

4. Chronic Caries.—Chronic caries of the dentin is occasionally encountered on the occlusal surfaces of molars. The exposed dentin has a yellow, brown, or black polished appearance, and is very hard. The etiology of this condition is that, in the course of undermining by occlusal caries, the surrounding enamel walls break off. The softened dentin is exposed and worn away by mastication until the hard, intact dentin at the bottom of the cavity is reached. The smooth surface and the impossibility of the lodgment of fermenting substances and microorganisms prevent further decay of the dentin. Under the microscope a limited number of microorganisms is found in the superficial portion of the exposed dentinal tubules; however, the lack of food retention and the absence of acid production on the surface seems to hinder further development of these microorganisms. The deeper layers of dentin are extensively sclerosed; the pulp chamber is usually greatly reduced in size by secondary dentin formation. This latter reaction of the pulp is very essential. Exposure of the pulp can usually be avoided only by the extensive building of secondary dentin under the decayed portion of the crown.

In this connection the problem of so-called "healed dental caries" should be discussed. The term healed caries is used by some investigators to indicate a condition in which no further destruction and dissolution of the dental hard tissues takes place in a carious lesion. It is the author's opinion that the use of the word healed is not justified. Nothing indicates that carious dentin, once it has been

decalcified, ever recalcifies, hardens, or heals. There are no anatomical connections present along which such a reparative process could take place: the decayed surface is separated from the pulp by the zone of transparency, and the decalcification of the matrix is always accompanied by proteolytic processes that make a restoration of the dentin impossible. Therefore, instead of speaking of healed caries, the term "arrested caries" should be used, indicating that the process clinically does not make any progress. A good example of arrested caries is the brown caries mark that is found on the approximal surface of a tooth after a decayed adjacent tooth has been lost. This brown spot, which otherwise probably would have developed into typical soft caries, may remain unchanged for decades after this part of the tooth surface has been transformed into a self-cleansing area. The lack of food retention and plaque formation on the surface prevents further progress of the bacteria in the tubules. A similar explanation must be offered for the cases of arrested caries under the influence of a changed dietary régime (see page 65). Here the progress of the decalcifying microorganisms is slowed down or perhaps is stopped by the changed conditions; the exact mechanism of this phenomenon is still under discussion and is at the present time the subject of extensive investigations. In a case of arrested caries of this type the softened dentin in the cavity apparently is lost and worn away so that finally the discolored hard dentin at the bottom of the cavity is exposed. In the same way chronic caries is not healed, but is arrested as a result of the mechanical transformation of the decayed surface into a self-cleansing area.

BIBLIOGRAPHY.

- ANDRESEN, V.: Über Mineralisation und Remineralisation des Zahnschmelzes, Deutsch. Mon. f. Zhk., 1921, **39**, 97.
———Mineralisation zur Behandlung der Caries incipiens, Deutsch. Mon. f. Zhk., 1922, **40**, 707.
BARKER, F. J.: Importance of Normal and Abnormal Tooth Structures in the Process of Dental Caries, Jour. Am. Dent. Assn., 1931, **18**, 17.
BEUST, TH. B.: Microorganisms and Caries, Jour. Am. Dent. Assn., 1930, **17**, 1536.
———Resistance to Caries, Jour. Dent. Res., 1931, **11**, 619.
———Reactions of the Dentinal Fibril to External Irritation, Jour. Am. Dent. Assn., 1931, **18**, 1060.
———Morphology and Biology of the Enamel Tufts with Remarks on Their Relation to Caries, Jour. Am. Dent. Assn., 1932, **19**, 488.
BLACK, G. V.: Operative Dentistry, Chicago, Medico-Dental Publishing Company, 1920.
———Special Dental Pathology, Chicago, Medico-Dental Pub. Company, 1920.

- BOEDECKER, C. F.: The Cause of the Rapid Decay of Occlusal Enamel Fissures, *Dent. Items, Int.*, 1931, **53**, 21.
- The Lipin Content of Dental Tissues in Relation to Decay, *Jour. Dent. Res.*, 1931, **11**, 277.
- BOEDECKER, C. F., and APPLEBAUM, EDMUND: Metabolism of the Dentin; Its Relation to Dental Caries and to the Treatment of Sensitive Teeth, *Dental Cosmos*, 1931, **73**, 995.
- Experimental Dental Caries in Rats, *Dental Cosmos*, 1932, **74**, 335.
- BOEDECKER, C. F., and BOEDECKER, HENRY, W. C.: The Bacterial Destruction of Dental Enamel, *Jour. Dent. Res.*, 1929, **9**, 37.
- BOYD, J. D., and DRAIN, C. L.: Arrest of Dental Caries in Childhood, *Jour. Am. Med. Assn.*, 1928, **90**, 1867.
- BOYD, J. D., DRAIN, C. L., and NELSON, MARTHA V.: Dietary Control of Dental Caries, *Am. Jour. Dis. Child.*, 1929, **38**, 721.
- BUNTING, R. W.: Experimental Production of Dental Caries in Animals, *Dental Cosmos*, 1925, **67**, 771.
- Certain Considerations in the Problem of Dental Caries, *Dental Cosmos*, 1930, **72**, 399.
- A Review of Recent Researches on Dental Caries, *Jour. Am. Dent. Assn.*, 1931, **18**, 785.
- BUNTING, R. W., HADLEY, F. P., JAY, P., and HARD, D. G.: The Problem of Dental Caries, *Am. Jour. Dis. Child.*, 1930, **40**, 536.
- BUNTING, R. W., JAY, PHILIP, and HARD, DOROTHY G.: A Report of the Successful Control of Dental Caries in Three Public Institutions, *Jour. Am. Dent. Assn.*, 1931, **18**, 672.
- BUNTING, R. W., and PALMERLEE, FAITH: The Rôle of *Bacillus Acidophilus* in Dental Caries, *Jour. Am. Dent. Assn.*, 1925, **12**, 381.
- CAUSH, D. E.: Caries of the Enamel, *Brit. Dent. Jour.*, 1921, **42**, 137.
- CHASE, SAMUEL W.: The Origin, Structure and Duration of Nasmyth's Membrane, *Anat. Rec.*, 1926, **33**, 357.
- DUBS, B.: Neue Beiträge zur Zahnkaries: Welche Rolle spielen die Mikroorganismen bei der Schmelzkaries? *Schweiz. Mon. f. Zhk.*, 1926, **36**, 49.
- EDDY, W. H.: Diet and Dentition, *Dental Cosmos*, 1931, **73**, 346.
- ENRIGHT, J. J., FRIESELL, H. E., and TRESCHER, M. O.: Studies of the Cause and Nature of Dental Caries, *Jour. Dent. Res.*, 1932, **12**, 759.
- ENTIN, D. A.: Zur physikalisch-chemischen Theorie der Karies, *Ztschr. f. Stom.*, 1929, **27**, 239.
- EULER, H.: Der Ablauf der Zementkaries, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 455.
- EULER, H., and MEYER, W.: Pathohistologie der Zähne, München, Bergmann, 1927.
- FABER, FRITZ: Die Caries an der Schmelzdentingrenze, *Deutsch. Mon. f. Zhk.*, 1928, **46**, 695.
- Histologie, *Fortschr. d. Zhk.*, 1932, **8**, 257.
- FEILER, ERICH: Die Transparenz des Zahnbeins, *Ztschr. f. Stom.*, 1923, **21**, 595.
- FISH, E. W.: Pathology of Dental Caries, *Dent. Rec.*, 1929, **49**, 151.
- Lesions of the Dentin and Their Significance in the Production of Dental Caries, *Jour. Am. Dent. Assn.*, 1930, **17**, 992.
- FLEISCHMANN, L.: Zur Pathogenese der Zahnkaries, *Ztschr. f. Stom.*, 1921, **19**, 153.
- FURRER, B.: Die Verkalkungszonen bei der Dentinkaries, *Schweiz. Mon. f. Zhk.*, 1922, **32**, 329.
- GOTTLIEB, B.: Untersuchungen über die organische Substanz im Schmelz menschlicher Zähne, *Vrtljschr. f. Zhk.*, 1915, **31**, 19.
- Ätiologie und Prophylaxe der Zahnkaries, *Ztschr. f. Stom.*, 1921, **19**, 129.

- GRIEVES, C. J.: Preliminary Study of Gross Maxillary and Dental Defects in Three Hundred Rats on Defective and Deficient Diet, *Jour. Am. Dent. Assn.*, 1922, **9**, 467.
- The Effect of Defective Diets on Teeth, *Jour. Am. Dent. Assn.*, 1923, **10**, 573.
- HADLEY, FAITH, P., BUNTING, R. W., and DELVES, EDNA A.: Recognition of *Bacillus Acidophilus* Associated with Dental Caries: A Preliminary Report, *Jour. Am. Dent. Assn.*, 1930, **17**, 2041.
- HANAZAWA, K.: Studien über die Karies des Dentins, *Vrtljschr. f. Zhk.*, 1923, **39**, 289.
- HANKE, M. T.: Relation of Diet to Caries and Other Dental Disorders, *Jour. Am. Dent. Assn.*, 1929, **16**, 2263.
- Relation of Diet to General Health and Particularly to Inflammation of Oral Tissues and Dental Caries, *Jour. Am. Dent. Assn.*, 1930, **17**, 957.
- HATTON, EDWARD H.: Caries: A Résumé of Our Knowledge of Its Action Together with Some of the More Recent Research Work, *Jour. Am. Dent. Assn.*, 1932, **19**, 1398.
- HAWKINS, H. F.: Manipulation of Food in the Control of Dental Caries and Systemic Pyorrhea, *Jour. Am. Dent. Assn.*, 1932, **19**, 963.
- HEAD, J.: Enamel Softening and Rehardening as Factor in Erosion, *Dental Cosmos*, 1910, **52**, 46.
- Saliva and Hardening and Softening of Tooth Enamel, *Dental Rec.*, 1913, **33**, 590.
- HESS, A. F.: Rickets, Including Osteomalacia and Tetany, Lea & Febiger, 1929.
- HIGAKI, RINZO: Beitrag zur Kenntnis des Schmelzoberhäutchens, *Deutsch. Zahnärztl. Wehnschr.*, 1931, **34**, 672, 723.
- HOWE, P. R.: Studies of Dietary Disorders Following Experimental Feeding with Monkeys, *Jour. Am. Dent. Assn.*, 1924, **11**, 1149.
- New Research on Dental Caries, *Dental Cosmos*, 1926, **68**, 1021.
- Practical Nutritional Suggestions for Dentists, *Jour. Am. Dent. Assn.*, 1930, **17**, 2140.
- JONES, MARTHA R., LARSEN, N. P., and PRITCHARD, G. P.: Dental Disease in Hawaii, *Dental Cosmos*, 1930, **72**, 439, 574, 685, 797.
- KESEL, R. G.: What Do We Know About Dental Caries? (A Critical Review of Recent Investigations), *Jour. Am. Dent. Assn.*, 1932, **19**, 903.
- KLEIN, HENRY, and SHELLING, DAVID H.: The Histopathology of Experimental Molar Caries in Rats, *Jour. Dent. Res.*, 1931, **11**, 151.
- LIESEGANG, R. E.: Rhythmenbildung in der Natur. 51. Bericht der Senckenbergischen Naturforschenden Gesellschaft, H. 2, Frankfurt a. M., July, 1921.
- Zur Kalkchemie des Zahnes, *Deutsch. Zahnärztl. Wehnschr.*, 1924, **27**, No. 10.
- Die Transparenz des Zahnbeins, *Ztschr. f. Stom.*, 1924, **22**, 649.
- MALLESON, H. C.: The Histology of Enamel Caries, *Brit. Dent. Jour.*, 1925, **46**, 907.
- MARSHALL, J. A.: Dental Caries and Pulp Sequelæ Resulting from Experimental Diets, *Jour. Am. Dent. Assn.*, 1927, **14**, 3.
- Control of Dental Caries by Means of Diet, *Jour. Am. Dent. Assn.*, 1928, **15**, 295.
- MELLANBY, MAY: The Chief Dietetic and Environmental Factors Responsible for the High Incidence of Dental Caries: Correlation Between Animal and Human Investigations, *Brit. Dent. Jour.*, 1928, **49**, 769.
- Experiments on Dogs, Rabbits and Rats, and Investigations on Man which Indicate the Power of Certain Food Factors to Prevent and Control Dental Diseases, *Jour. Am. Dent. Assn.*, 1930, **17**, 1456.

- MELLANBY, MAY, and PATTISON, C. L.: Some Factors of Diet Influencing the Spread of Caries in Children, *Brit. Dent. Jour.*, 1926, **47**, 1045.
- MILLER, W. D.: The Question of the Transparency of the Dentin, *Dental Cosmos*, 1903, **45**, 253.
- Über die Transparenz des Dentins, *Deutsch. Mon. f. Zhk.*, 1903, **21**, 182.
- MUMMERY, I. H.: Translucent Zones in Enamel, *Brit. Dent. Jour.*, 1926, **47**, 473.
- The Structure of Enamel and Dentin with Reference to the Pathology of the Teeth, *Jour. Am. Dent. Assn.*, 1927, **14**, 204.
- NISHIMURA, TOYOJI: Histologische Untersuchungen über die Anfänge der Zahnkaries, speziell der Karies des Schmelzes, *Schweiz. Mon. f. Zhk.*, 1926, **36**, 49.
- ORBAN, B.: Histology of the Enamel Lamellæ and Tufts, *Jour. Am. Dent. Assn.*, 1928, **15**, 305.
- PICKERILL, H. P.: The Prevention of Dental Caries and Oral Sepsis, London, Baillière, Tindall & Cox, 1923, 3d Edition, p. 122.
- RODRIGUEZ, F. E.: Incidence of Lacto-bacillus Acidophilus, *Jour. Am. Dent. Assn.*, 1930, **17**, 1711.
- ROSEBURY, THEODOR, and KARSHAN, MAXWELL: Studies, in the Rat, of Susceptibility to Dental Caries. 1. Bacteriological and Nutritional Factors, *Jour. Dent. Res.*, 1931, **11**, 121.
- Pathological Changes in the Teeth of Rats Produced by Synthetic Diets, *Jour. Dent. Res.*, 1931, **11**, 137.
- STEIN, T. B., HINCK, C. F., and HOSKINS, M. M.: The Anatomy, Physiology and Chemistry of Nasmyth's Membrane, *Dental Cosmos*, 1928, **70**, 592.
- SWANSON, JOHN H.: Age Incidence of Lines of Retzius in the Enamel of Human Permanent Teeth, *Jour. Am. Dent. Assn.*, 1931, **18**, 819.
- TÜRKHEIM, H.: Karies-Forschung, yearly contribution in *Fortschr. d. Zhk.*, 1925-1932, Leipzig, Thieme, vols. **1** to **8**.
- WEBER, R.: Zur Kenntnis des Auftretens von Fett am Zahn, *Vrtljschr. f. Zhk.*, 1926, **42**, 64.
- Experimentelle Untersuchungen über die Frage der Remineralisation des Zahnschmelzes durch den Speichel, *Ztschr. f. Stom.*, 1929, **27**, 912.
- Neue Untersuchungen über das Auftreten von Fett im Zahn, *Deutsch. Mon. f. Zhk.*, 1930, **48**, 1489.
- WILLIAMS, T. L.: Can We, by a Change in Food Habits, Change the Structure of Formed Enamel? *Dental Cosmos*, 1927, **69**, 590.
- YUMIKURA, SHIGEIE: Eine neue Färbemethode für gingivale Epithelverhornung und für das sog. sekundäre Schmelzoberhäutchen bzw. Cuticula dentis Gottliebs, *Ztschr. f. Stom.*, 1925, **23**, 868.

CHAPTER IV.

INFLAMMATION OF THE PULP (PULPITIS).

THE pulp is an extremely sensitive organ which reacts very readily to any irritation or injury. A severe injury may even cause immediate death of the pulp, while a lesser injury causes a typical inflammation known as pulpitis.

Not every injury to which a tooth is exposed leads to pulp inflammation. Minor injuries, such as abrasion or superficial caries, do not cause an inflammatory reaction of the pulp tissue but produce another form of reaction, namely, the deposition of secondary dentin. The inflammatory reaction seems to be the result of more severe injuries that expose or nearly expose the pulp. In the latter case, when the irritating factors act through a thin layer of intact dentin, the individual resistance of the pulp tissue plays an important rôle. In one instance, the irritation from deep-seated caries or a large filling will stimulate merely the formation of secondary dentin, and the pulp tissue itself will not become inflamed; while in another instance, a similar cavity or filling may cause typical pulpitis. In every tissue reaction the factor of resistance of the tissue cells is of decisive influence, and since the amount of this resistance is unknown, it is impossible to determine how much of a certain irritation will be necessary to produce a certain form of tissue reaction.

The microscopic manifestations of pulpitis are typical of inflammation as observed in other tissues, although there is a basic difference between the final outcome of pulpitis and the outcome of most of the inflammatory processes elsewhere in the body. Nearly all organs can survive a certain amount of inflammation; they may heal either by resolution or by scar formation, but in either case the organ as a whole may survive and continue to function. This is not true, however, of dental pulp. The location of the pulp tissue inside a hard-tissue encasement renders the possibility of an inflamed pulp's healing theoretically very doubtful, and practically nil. Clinical experience demonstrates that inflammation of the dental pulp will lead invariably, either quickly or slowly, to complete destruction of this organ. In case of a mild injury and a resistant pulp, the

process may continue for years and even show upon microscopic examination a marked tendency of the tissue to wall off the progressing destruction. The final outcome, however, will always be necrosis of the entire pulp.

The invariably unfavorable outcome of pulpitis is due to the location of the pulp inside a rigid, hard-tissue capsule. Because of little communication with the bloodvessels of the body, the defensive mechanisms of the vascular system of the pulp are limited. The typical manifestations of inflammation: hyperemia, edema and cellular infiltration, cause an increase in the volume of inflamed tissue; in case of the pulp, in which the soft tissue is encased between unyielding walls, any increase in size will cause increased pressure and disturbed circulation, and the pulp will actually strangle itself. Another basic difference between an exposure of the pulp and a wound elsewhere in the oral cavity lies in the fact that there is no epithelial tissue in the pulp to play a part in the healing process. Every exposure of connective tissue of the oral cavity will finally heal by epithelization. In a pulp wound no such form of healing is possible.

The only way in which an exposed pulp could possibly heal would be by deposition of hard tissue (dentin) in the injured area, forming a solid cover over the exposure. This form of pulp healing is theoretically possible, and the different clinical methods of pulp capping are based upon this possibility. But actual observations in human teeth show that by far the majority of cases of pulp exposure and pulp capping are followed by inflammation of the pulp tissue with subsequent necrosis of the pulp rather than by reparative, hard-tissue formation. These results have led to the general belief that attempts to save an exposed pulp should, as a rule, be discouraged in all but children's teeth, in which a wide foramen offers a better blood supply and in which the higher resistance of the young tissue may present a greater chance for successful pulp capping. The general statement can be made that, excluding this one exception, every exposed pulp must be considered as the seat of actual or future pulpitis, with final complete destruction of the pulp. Hence, an exposed pulp is a lost pulp.

According to etiology, three forms of pulpitis can be differentiated:

1. Pulpitis due to bacterial invasion (infective pulpitis).
2. Pulpitis due to chemical irritation.
3. Pulpitis due to thermal irritation.

The first form, infective pulpitis, is the most frequent type, having its inception in an invasion of the pulp tissue by bacteria

from the bottom of a carious cavity or through an accidental pulp exposure (fracture, exposure during cavity preparation).

Chemical pulpitis is caused by the action of some toxic drug, such as arsenic trioxide, upon the pulp tissue. Some of the silicate cements also may be responsible for chemical pulpitis and death of the pulp, for it is not an uncommon observation that pulps have become inflamed and died under silicate fillings, probably due to diffusion of some cell poison from the filling material through the dentin into the pulp.

The third clinical form, pulpitis due to thermal irritation, occurs under large or deep-seated metal fillings without proper insulation. After the insertion of such fillings thermal stimuli, especially cold, are transmitted readily to the pulp and there register as soreness or pain. The outcome will depend largely upon the individual resistance of the pulp. Some pulps will be able to produce secondary dentin, which reaction will express itself clinically as a gradually decreasing sensitiveness of the pulp. In other teeth, the soreness will steadily increase, and pulpitis will finally develop as the result of the continued thermal shock.

HYPEREMIA OF THE PULP.

Before pulpitis develops, a preliminary stage of hyperemia is frequently observed in the pulp. Clinically, pulp hyperemia is characterized by marked sensitiveness to thermal changes, especially to cold. This sensitiveness is of temporary character, occurring only upon the stimulation of cold water, ice cream, etc., and subsiding as soon as the irritation is removed. Microscopically, a marked dilatation and engorgement of all pulp vessels are found in these cases; the outline of the vessels is slightly irregular, indicating the beginning of injury to the vessel walls (Fig. 85). The mechanism of the development of pulp hyperemia is as follows: The arterial vessels pump an increased amount of blood into the irritated pulp (active hyperemia); at the same time a paralysis of the contractile elements in the walls of the capillaries takes place, causing dilatation of the vessel and delayed flowing-out of the blood through the veins (passive hyperemia). The pulp vessels at the apical foramen pass through a narrow, rigid channel; therefore, if the arteries are dilated, they automatically compress the veins and increase the interference with normal circulation.

Hyperemia of the pulp is a reversible condition. This means that if the irritation is definitely removed, the pulp may return to

normal; the bloodvessels may contract, and normal circulation may be reestablished. In other cases hyperemia develops, without sharp transition, into acute pulpitis, and then the pulp is lost. It is the author's opinion that in clinical practice the diagnosis of hyperemia is often made in teeth that present merely increased sensitiveness of exposed living dentin. In many cases of so-called hyperemia of the pulp under a deep cavity, the dentin itself is extremely sensitive to touch with an explorer point, and this phenomenon suggests hypersensitiveness of the dentin rather than an actual change in the pulp tissue.

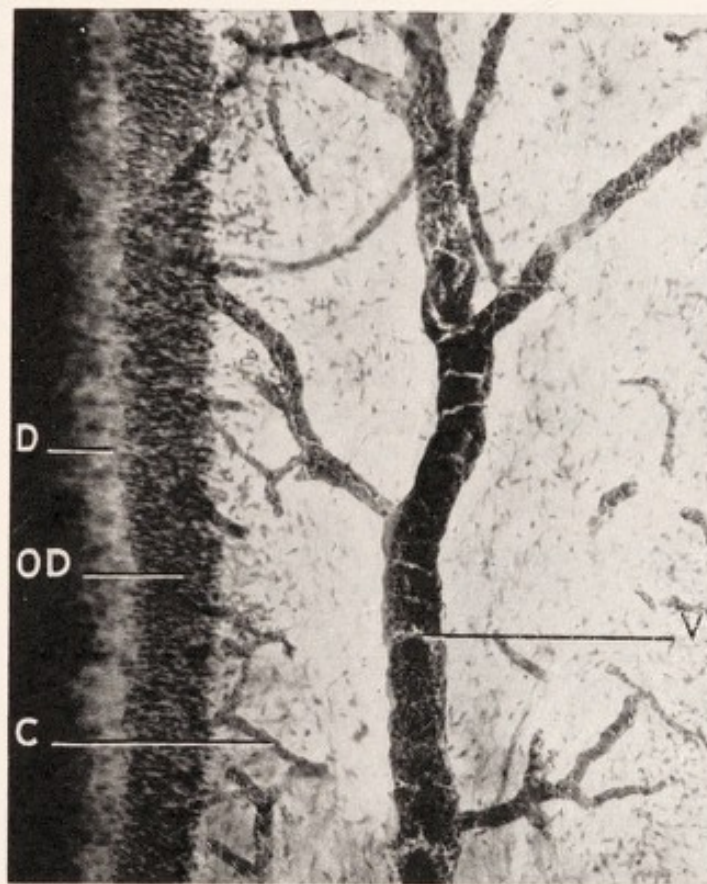


FIG. 85.—Hyperemia of the pulp. Dilatation and hyperemia of the pulp vessels. The pulp tissue between the vessels is free from inflammatory changes. V, hyperemic bloodvessel (vein); C, capillaries near the odontoblastic layer; OD, odontoblasts; D, dentinoid.

ACUTE PULPITIS.

The microscopic changes in pulpitis are rather constant and typical regardless of the etiology of the pulp inflammation; however, as infective pulpitis has by far the greatest clinical importance, principally this type of pulpitis will be described and illustrated.

In infective pulpitis, as in every inflammatory process, we can differentiate between acute and chronic forms. Clinically, the acute

forms are manifested by acute pain, whereas the chronic forms cause little or no pain. Microscopically the acute forms show all the characteristics of an acute inflammation: enlargement of the blood-vessels, slowing up of the circulation, accumulation of white blood cells in the periphery of the blood stream, extensive migration of these cells (leukocytes) through the vessel wall, diffusion of blood serum through the walls of the vessels, and mobilization of the cells of defense (polyblasts) in the pulp tissue. Subsequent breaking down of tissue and formation of pus takes place. The chronic forms of pulpitis, however, present a different microscopic picture, for, in addition to destructive changes that are characteristic of the acute forms, reparative processes can also be observed. A chronic ulcer or granulation tissue is formed on the surface of the inflamed pulp, which condition may exist for years without clinical symptoms.

Depending upon the clinical symptoms and upon the amount of pulp tissue involved, acute pulpitis can be divided into acute partial pulpitis and acute total (suppurative) pulpitis. However, the clinical symptoms do not always occur in the order in which they are described here; neither has every form of pulpitis a certain, well-outlined, clinical counterpart. Sometimes a pulp that has caused the patient unbearable pain presents, upon microscopic examination, only a very small, inflamed area; again, pulps that microscopically show a severe suppurative pulpitis may have a clinical history of only temporary, indefinite pain. The wide variation in the mental reaction of different patients to pain, the difference in the location of the pulpitic area, and the presence or absence of larger pulp nerves in the diseased portion of the pulp may be some of the factors that are responsible for the occasional contradiction between clinical history and microscopic findings.

1. **Acute Partial Pulpitis.**—Acute partial pulpitis is diagnosed clinically by the presence of a characteristic kind of pain in a tooth with a large defect or filling. The pain appears in attacks that are produced by changes in temperature, especially cold fluids or cold air. After the thermal irritation has subsided, the pain continues for a while. Spontaneous attacks of pain also occur. Usually the involved tooth shows extensive caries that extends close to or completely into a pulp horn. The tooth is not sensitive to percussion. Microscopically, two forms of acute partial pulpitis can be distinguished: a serous form and a purulent form.

(a) *Acute Partial Pulpitis, Serous Form.*—This form represents the earliest stage of pulpitis, developing from a hyperemia of the pulp by migration of white blood cells and diffusion of blood serum

through the walls of the pulp capillaries (Fig. 86). These three changes, namely, hyperemia of the neighboring pulp vessels, pres-

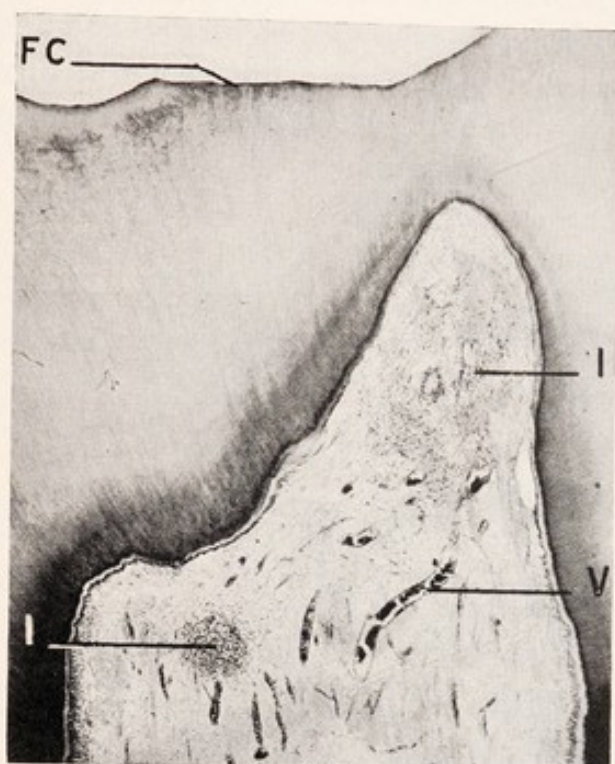


FIG. 86.—Acute partial pulpitis (serous form) under a filling. Upper molar. FC, floor of cavity; I, areas of cellular infiltration of the pulp tissue; V, hyperemic bloodvessels.

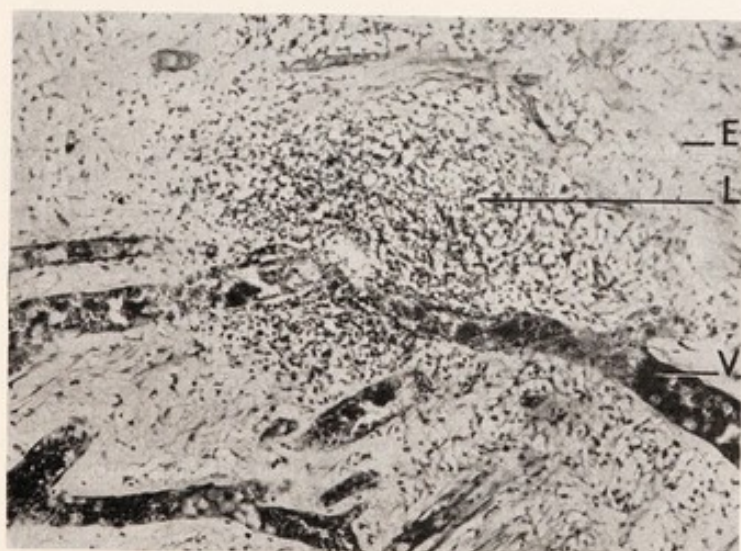


FIG. 87.—Higher magnification of Fig. 86. Accumulation of inflammatory exudate cells around the capillaries of the pulp. L, exudate cells (leukocytes); V, hyperemic bloodvessels; E, edema of the pulp tissue.

ence of polymorphonuclear leukocytes in the perivascular tissue, and serous imbibition of the pulp tissue (edema) are the microscopic characteristics of the earliest form of acute pulpitis. Of the pulp

cells the odontoblasts seem to be the most sensitive to the presence of irritation, as this layer is always discontinued in the inflamed area. In Fig. 87 a microscopic area of pulpitis is illustrated. Inflammatory round cells are accumulated in the pulp tissue around a capillary. All capillaries in the neighborhood of the inflamed area are dilated and hyperemic; the surrounding connective tissue of the pulp shows marked edema.

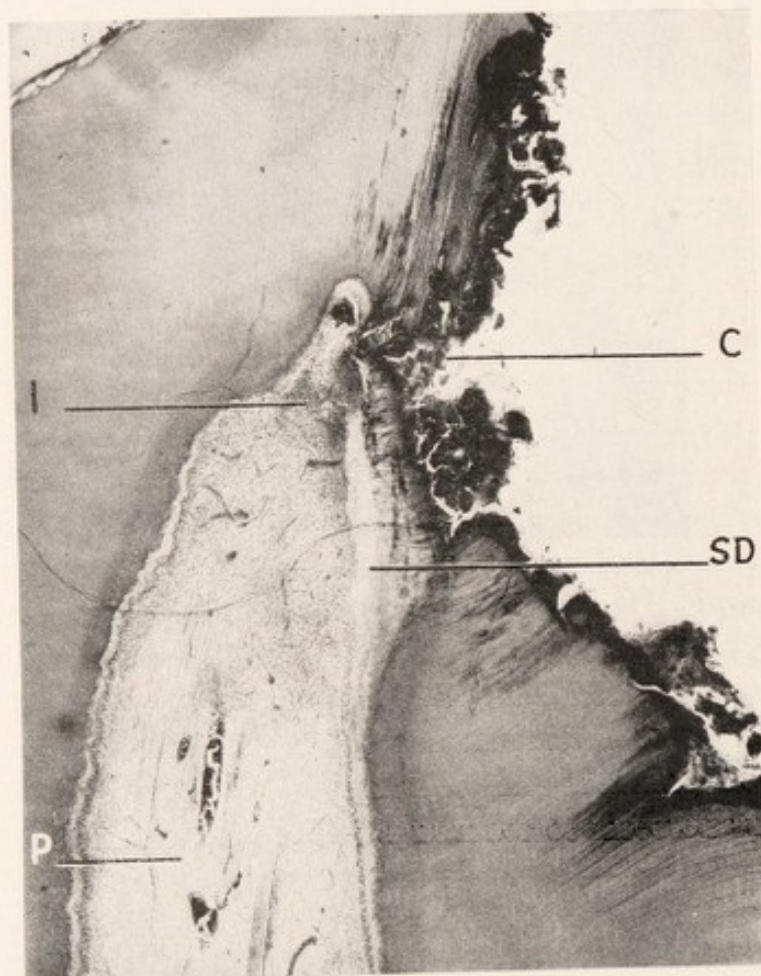


FIG. 88.—Acute partial pulpitis (purulent form) under penetrating caries. Lower bicuspid. C, carious destruction reaching the pulp horn; SD, secondary dentin; I, dense inflammatory infiltration of the pulp tissue with beginning abscess formation; P, normal pulp tissue with slightly dilated bloodvessels.

(b) *Acute Partial Pulpitis, Purulent Form.*—In acute partial pulpitis the inflammation gradually reaches a stage in which the combined action of bacterial toxins and leukocytes causes a breaking down of some of the pulp tissue; the tissue debris, together with dead and dying white blood cells, forms pus which accumulates on the surface of the inflamed area. When pus formation has led to the development of a cavity in the pulp tissue from which the pus is being discharged, we speak of the condition as a pulp abscess.

Fig. 88 shows an early stage of pulp abscess. Deep caries is present on the mesial side of a lower bicuspid. At first secondary dentin had been deposited on the inner wall of the pulp chamber, but caries progressed rapidly and, when the tooth was extracted, had penetrated the thin layer of secondary dentin, and the bacterial invasion had reached the pulp horn. The pulp shows a dense inflammatory infiltration with leukocytes; the pulp tissue adjacent to the carious dentin broke down and has been replaced by a small space containing leukocytes and cell débris. The bloodvessels in the immediate neighborhood of the pulpitic area are hyperemic. Beyond this area the pulp tissue is absolutely normal, which is the reason for calling this condition partial pulpitis.

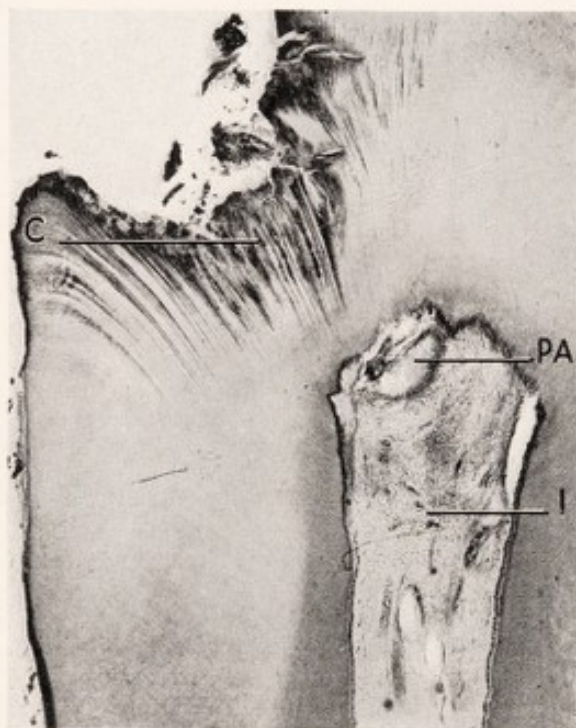


FIG. 89.—Acute partial pulpitis under deep caries. Formation of a pulp abscess, Lower bicuspid. C, caries of the dentin; PA, pulp abscess; I, infiltrated pulp tissue.

A slightly advanced stage of partial pulpitis with formation of a small abscess is seen in Fig. 89 under conditions similar to those shown in the preceding picture. The incisal part of the pulp is densely infiltrated. Toward the carious cavity a small abscess cavity has developed in the pulp tissue in which, under higher magnification, a large number of polymorphonuclear leukocytes can be seen which, together with exudate and cell débris, form pus (Figs. 90 and 91). The pulp tissue nearer to the root is normal except, perhaps, for a slight enlargement of the bloodvessels. In some of the

veins an increased number of polymorphonuclear leukocytes can be observed; the surrounding pulp tissue, however, is normal.

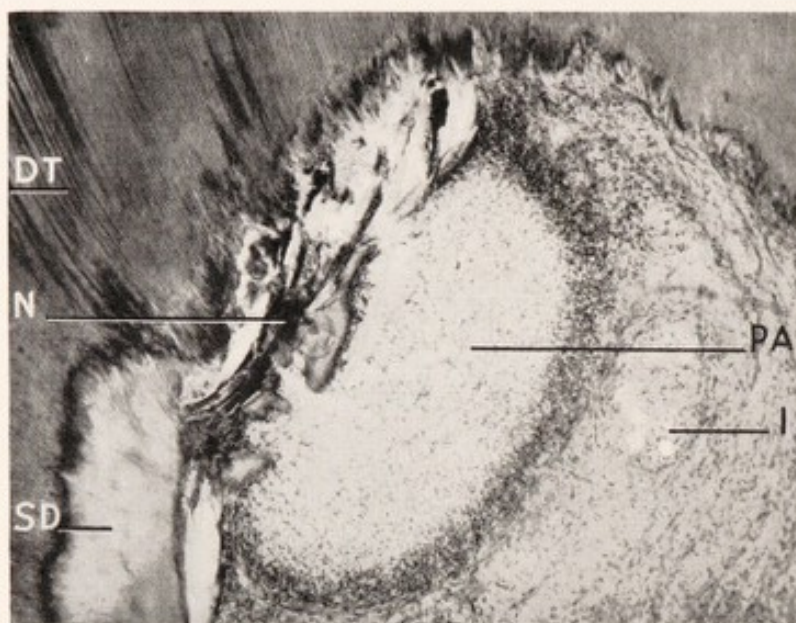


FIG. 90.—Higher magnification of Fig. 89. Pulp abscess. DT, infected dentinal tubules; N, necrosis of dentin and pulp tissue; PA, pulp abscess resulting from breaking down of the pulp tissue next to the infected dentin; I, densely infiltrated pulp tissue; SD, secondary dentin.

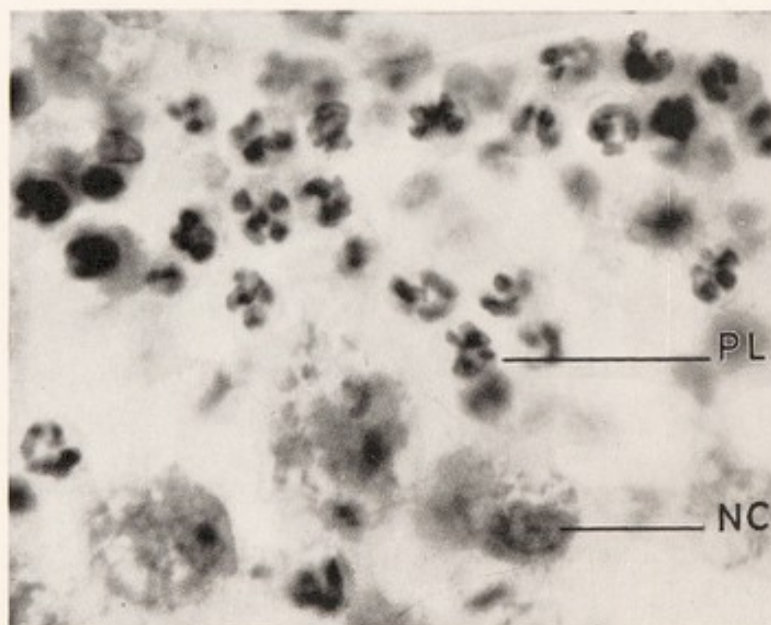


FIG. 91.—Characteristic inflammatory cell forms present in acute purulent pulpitis: polymorphonuclear leukocytes (PL). NC, necrotic cells.

Acute pulpitis, just like any other acute purulent inflammation, is microscopically characterized by the predominance of neutrophil, polymorphonuclear leukocytes in the cellular exudate.¹

¹ For more detailed information about the various cell forms participating in the process of inflammation see page 141.

2. **Acute Total Pulpitis (Suppurative Pulpitis).**—Acute total pulpitis can be defined as a condition in which the major portion of the pulp tissue is involved in an acute inflammation. It develops from acute partial pulpitis by the spreading of the inflammation over the entire pulp. From this definition it is evident that there is no sharp borderline between “partial” and “total” pulpitis, but that many intermediate stages or more or less involved pulps may be encountered upon microscopic examination. The same is true of the consideration of clinical symptoms: the transition from partial pulpitis with temporary attacks of pain to total pulpitis with constant throbbing pain is a gradual one, usually developing over a period of several days. In a typical case of total pulpitis the pain is very severe; the patient is kept awake at night; thermal changes increase the pain, but the pulp is usually more sensitive to the influence of warmth than to the application of cold. This seems to be the reason for the exacerbation of pain at night in bed. The tooth is sometimes slightly sensitive to percussion, indicating a collateral edema of the apical periodontal membrane.

In the microscopic examination of teeth that were extracted under the clinical diagnosis of acute total pulpitis, it is usually found that the closed forms in which the inflamed pulp is still covered by a layer of carious dentin or by a filling have given the greatest pain; this fact is easily explained by the pressure that develops when the forming pus has no outlet. Those cases of total pulpitis that have open communication with the oral cavity through a carious cavity usually cause less severe pain, since there is a free drainage of the forming purulent exudate, and, therefore, no pressure is exerted upon the nerves of the inflamed pulp.

The final outcome of untreated total pulpitis seems to be largely dependent upon this topographic difference between the open and closed forms. In closed pulps in which the exudate has no drainage, infiltration of the entire pulp tissue, abscess formation, and finally necrosis of the pulp will occur rather quickly.

The other possible outcome of total pulpitis, namely, the transition into a chronic form of pulpitis is usually found in cases that have an open communication with the oral cavity. The drainage of exudate prevents pressure and allows the pulp tissue to build up a wall of resistance that may establish some kind of temporary balance between advancing infection and defensive reaction of the pulp tissue. This condition will be described more in detail under the heading of chronic pulpitis.

(a) *Acute Total Pulpitis, Open Form.*—In this condition a large portion of the pulp or the entire pulp is acutely, purulently inflamed,

and there is communication with the oral cavity usually through a carious cavity (Fig. 92). In the upper right corner of the specimen can be seen the carious cavity, which is filled with débris, broken-down dentin, pus cells, and food particles. In one of the former pulp horns, the pulp is exposed; from its surface a purulent exudate is being discharged; part of the pulp tissue has been destroyed. The larger part of the pulp is densely infiltrated with inflammatory exudate cells. The bloodvessels are dilated and hyperemic; the odontoblastic layer has completely disappeared near the area of pulp exposure and shows cell degeneration in the more distant parts of the pulp surface. As a whole this condition differs from acute

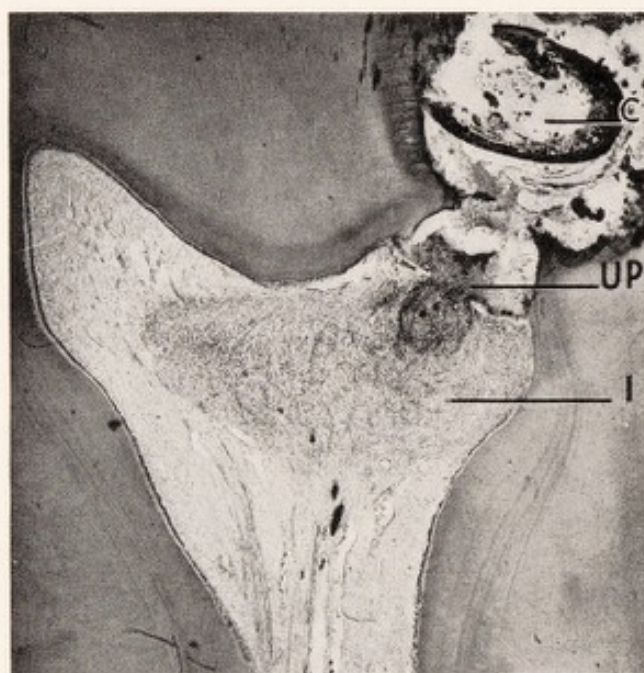


FIG. 92.—Acute total pulpitis (open form). Upper molar. C, carious cavity containing food débris; UP, ulcerated exposed pulp surface at the bottom of the cavity; I, diffuse inflammatory cell infiltration in the crown portion of the pulp.

partial pulpitis (Figs. 88 and 89) only in the degree and the extent of inflammation, for, instead of only one small area of pulp tissue, the entire crown portion of the pulp is involved.

If caries and pulp exposure are found in the cervical portion of a tooth, it can be observed that pulpitis starts in the root canal next to the area of exposure and from there spreads crownward into the pulp chamber and rootward into the canal (ascending pulpitis). Such a condition is seen in Fig. 93, illustrating a specimen of a lower molar with clinical symptoms of total pulpitis. The occlusal portion of the crown is intact; deep caries is present on the distal surface of the crown, extending downward into the root canal.

The microscopic examination shows that the major portion of the distal root canal is filled with pus and that an abscess cavity has developed in the pulp tissue crownward from the carious cavity. The entire pulp is in a stage of diffuse, purulent inflammation, the intensity of which decreases toward the mesial pulp horn.

(b) *Acute Total Pulpitis, Closed Form.*—In the closed form of total pulpitis the formation of abscess cavities in the pulp tissue is the predominant microscopic finding. Due to the impossibility of escape, the purulent exudate accumulates under pressure inside the pulp chamber and causes large areas of tissue destruction in the pulp. When a pulp abscess is exposed by removing the overlying



FIG. 93.—Acute total pulpitis (open form) under penetrating gingival caries. Lower molar. C, carious cavity on the distal surface of the distal root; PA, pulp abscess in the distal root canal extending upward into the crown portion of the pulp; I, diffuse inflammatory infiltration of the crown portion of the pulp.

dentin, a drop of pus is seen to emerge from the opening, seemingly under pressure. Immediately the patient feels great relief from pain, comparable to the relief that follows lancing an abscess, evacuating the pus, and removing the pressure of the retained exudate.

Suppurative pulpitis in a closed pulp chamber usually causes rapid breaking-down and necrosis of the entire pulp. Such a condition is shown in Fig. 94. In a lower molar total pulpitis developed under a large caries which almost reached the pulp; only a thin, but still intact, layer of dentin covered the pulp chamber. Nothing was done to treat the pulpitis, as is frequently the case in patients that are rather insensitive or overcome pain by using anodynes. After a period of from several days to several weeks the pain subsided,

and the pulp showed clinically and microscopically evidence of necrosis. The cells lose their staining properties, but calcifications and dead exudate cells can sometimes still be recognized in the tissue remnants. Farther apically in the root canals there is frequently some living inflamed pulp tissue still present (see Figs. 98 and 99).



FIG. 94.—Necrosis of the pulp. Lower molar. SD, layer of secondary dentin covering the pulp chamber; N, necrotic tissue in the pulp chamber and in the root canals.

CHRONIC PULPITIS.

As stated before, the outcome of total pulpitis may be twofold: either rapid, complete destruction of the entire pulp, or transition into chronic pulpitis. The latter outcome is encountered mostly in young pulps with high tissue resistance, in the presence of an infection of low virulence, and in such cases where drainage of the inflammatory exudate is possible.

Two forms of chronic pulpitis can be differentiated clinically and microscopically: an ulcerative and a hypertrophic form.

1. *Chronic Ulcerative Pulpitis*.—In chronic ulcerative pulpitis an ulcer develops on the surface of the pulp, while in the surrounding pulp tissue a dense wall of round cells borders the less inflamed portions of the pulp. This condition, which offers few if any clinical symptoms, may exist for a long time. Upon clinical examination a cavity is usually found leading into the pulp; the tooth shows little or no reaction to cold or warmth. Upon exploring the pulp chamber, living, bleeding pulp tissue is found, which is much less sensitive to touch than a normal or acutely inflamed pulp, probably because of partial degeneration of the pulpal nerves.

Microscopically there is a tendency toward incapsulation of the inflammatory process and formation of granulation tissue in the pulp. The transition from acute suppurative pulpitis into a chronic form is illustrated in Fig. 95. A large abscess cavity occupies the mesio-occlusal third of the pulp of a lower first molar under occlusal caries. The rest of the pulp tissue has been altered very little; in the distal portion of the pulp, the odontoblastic layer on the wall and on the floor of the pulp chamber is microscopically intact. The pulp tissue is arranged parallel to the wall of the abscess cavity, thus forming a fibrous capsule around the abscess. We may con-

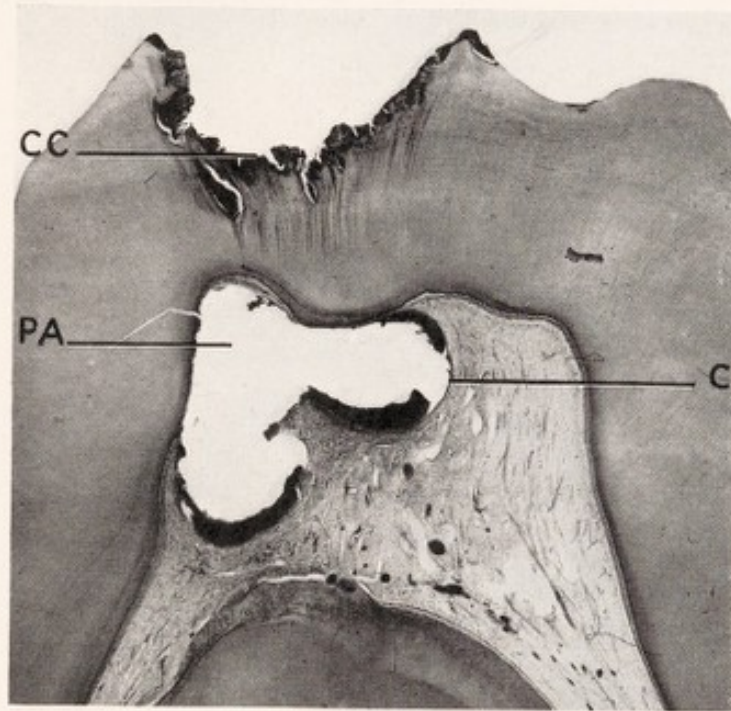


FIG. 95.—Pulp abscess under occlusal caries in an upper molar. The abscess is surrounded by a connective tissue capsule. The rest of the pulp tissue shows only slight inflammatory changes. CC, carious cavity; PA, pulp abscess; C, capsule of pulp tissue surrounding the abscess cavity.

sider this condition as the manifestation of a highly resistant pulp tissue: an attempt is made by the pulp to wall off the spreading inflammation and to form a fibrous capsule around the abscessed area.

A different form of chronic ulcerative pulpitis is seen in Fig. 96. The entire pulp of a lower second deciduous molar is in a state of chronic inflammation; two large abscess cavities are present in the pulp chamber. Caries has destroyed a part of the pulp tissue in the mesial pulp horn; the ulcerative pulp surface, however, is not bare but covered by an irregular calcified mass that extends from one side of the opening to the other, leaving only a small communication

between pulp and cavity (Fig. 97). Such calcific deposits are frequently found in pulps that are chronically inflamed; they develop

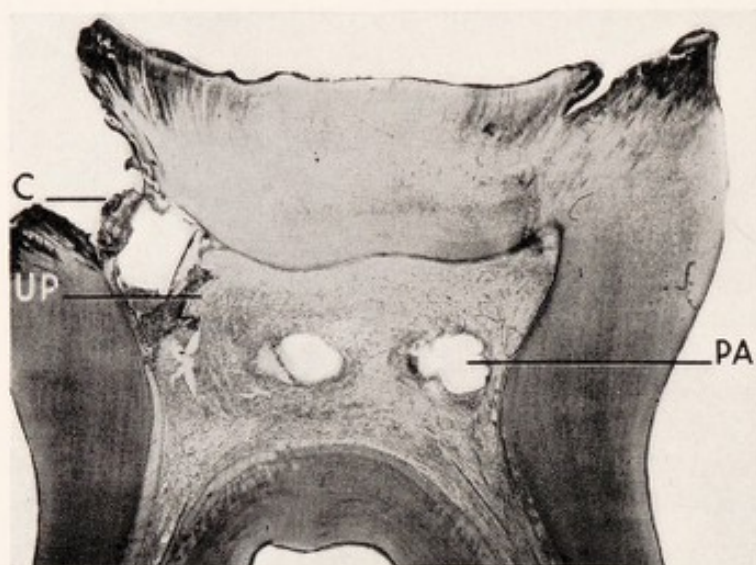


FIG. 96.—Chronic ulcerative pulpitis. Lower deciduous molar. All of the pulp tissue shows extensive diffuse round-cell infiltration. C, carious cavity; UP, ulcerated surface of the pulp; PA, abscess cavities within the pulp tissue.

through calcification of the dying or necrotic tissue. In this particular case we may consider these calcific masses as an attempt to bring about healing of the exposed pulp horn. The presence of such cal-

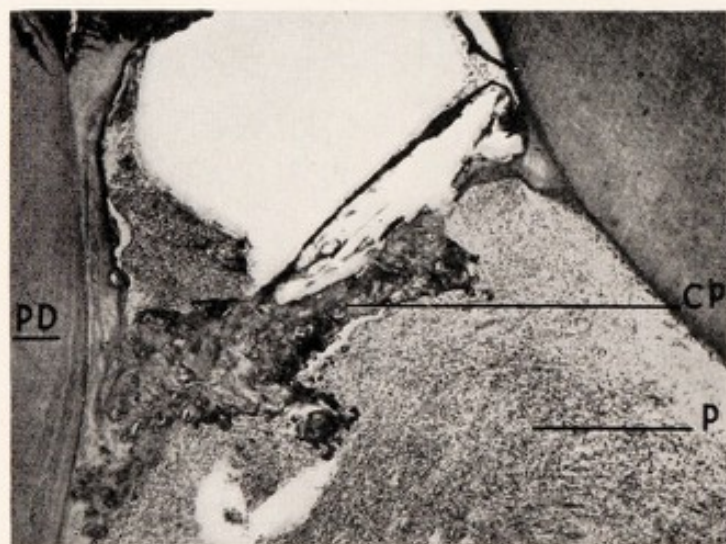


FIG. 97.—Higher magnification of Fig. 96. P, densely infiltrated pulp tissue; CP, irregular calcification of the pulp tissue covering the area of pulp exposure; PD, primary dentin.

cifications is always evidence that the pulp inflammation is of long standing.

Sometimes in the course of acute total pulpitis the crown part of the pulp is completely destroyed; however, the destructive process does not continue through the root canal all the way to the apical foramen. The apical part of the root canal still contains living tissue in a state of chronic inflammation. On the surface of this pulp stump an ulcer discharges exudate and cells into the empty part of the root canal (Fig. 98). Clinically, this condition is frequently found in teeth with deep caries and an exposed pulp chamber containing necrotic pulp tissue; there is no vital reaction in the coronal portion of the root canal, but in the apical part the entering explorer or broach still meets vital, sensitive, bleeding tissue before the apex

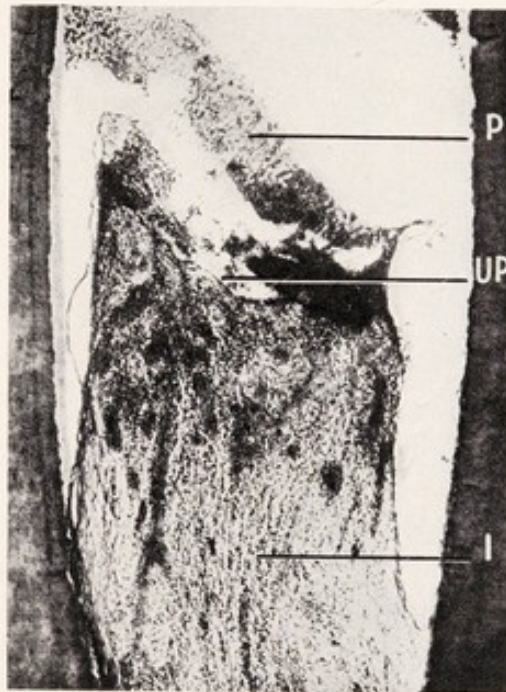


FIG. 98.—Chronic ulcerative pulpitis. Pulp stump in the root canal of a molar after breaking-down of the crown portion of the pulp; I, inflamed pulp tissue; UP, ulcerated surface of pulp stump; P, purulent exudate in the root canal.

is reached. Coolidge described the histological findings in a case of this type. He called attention to the fact that, while the root canals still contained vital, inflamed pulp remnants, the apical periodontal tissues had undergone marked changes. Enlarged capillaries and an accumulation of plasma cells were found in the periodontal membrane. A marked break had developed in the continuity of the bone plate around the root end. Two of Coolidge's illustrations will be reproduced here. In the first illustration (Fig. 99) a lower molar is shown with penetrating mesial caries. The pulp chamber is full of necrotic tissue and debris. The apical portions of the root canals still contain vital, inflamed pulp tissue.

At the apices the pulp strand passes through the foramen; the apical fibers of the periodontal membrane have been destroyed, and the periodontal membrane itself has been widened around the root end

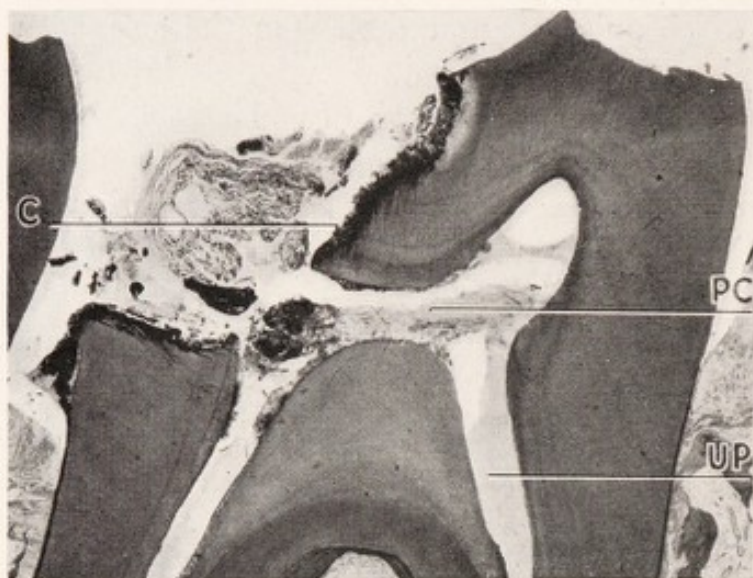


FIG. 99.—Chronic ulcerative pulpitis in the root canals of a lower molar. C, penetrating caries in the mesial portion of the crown; PC, pulp chamber filled with necrotic pulp debris and exudate; UP, ulcerated surface of the pulp stump in distal root canal. (Coolidge, Jour. Am. Dent. Assn.)

(Fig. 100). The soft tissue in this area has lost its normal fibrous structure; irregular fiber bundles are present, between which are

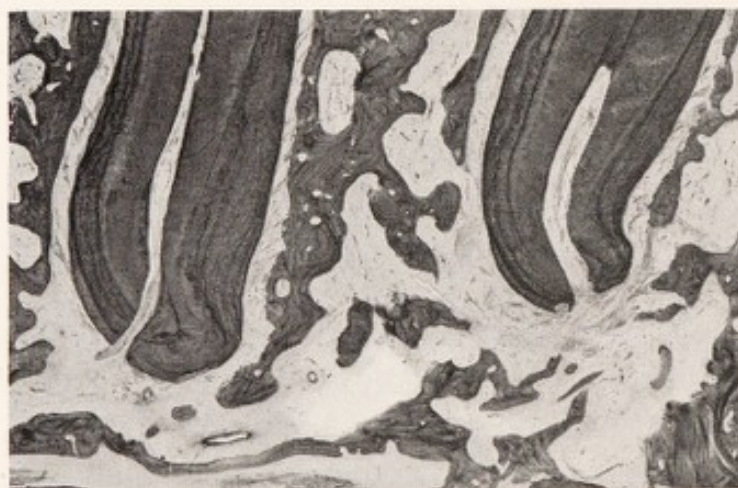


FIG. 100.—Apices of the roots of the molar shown in Fig. 99. Both root canals contain vital pulp tissue. There is a break in the inner plate of the alveolar bone at the apex of each root. The apical fibers of the periodontal membrane are destroyed. (Coolidge, Jour. Am. Dent. Assn.)

wide capillaries and groups of plasma cells (polyblasts). These findings are of great importance because they illustrate the gradual transition from pulpitis to periodontitis. There is no sharp line

between these two conditions: while inflamed, vital pulp tissue is still present in the root canals, the action of the bacteria extends beyond the apex. Inflammatory products are probably carried from the ulcer on the surface of the pulp stump through the blood and lymph channels into the periapical tissue and there initiate periodontitis. Thus it can be understood how a tooth, that still contains vital, inflamed pulp tissue, may show radiographically a widened periodontal space around the apex. (See also Figs. 112 and 113.)

Another chronic ulcer in a lower second molar is illustrated in Fig. 101. A large occlusal cavity caused exposure of the distal pulp



FIG. 101.—Chronic pulpitis following pulp exposure in a lower molar. The pulp is transformed into granulation tissue. GT, plug of granulation tissue proliferating through the opening in the pulp chamber.

horn, but instead of the usual progressive destruction of the pulp tissue, the pulp reacted by a generalized, diffuse inflammation, thereby being transformed into granulation tissue. A higher magnification shows nothing of the original pulp structure; instead a new type of tissue is present, consisting of newly built connective tissue with a large number of inflammatory exudate cells (Fig. 102). Some of these cells are plasma cells, characterized by their large, protoplasmic bodies and the eccentric nuclei; others are small lymphocytes. Plasma cells are typical of chronic inflammations, whereas leukocytes are predominant in acute inflammations (see Fig. 91). At the point where the pulp was exposed, the granulation tissue has proliferated through the small opening in the pulp chamber

and appears in the cavity as a bright red tissue plug of pinhead size. This plug, as well as the rest of the pulp, is only slightly sensitive. No nerve tissue could be found in the entire pulp chamber. The tissue proliferation indicates the transition from the chronic ulcerative type of pulpitis to the chronic hypertrophic form.

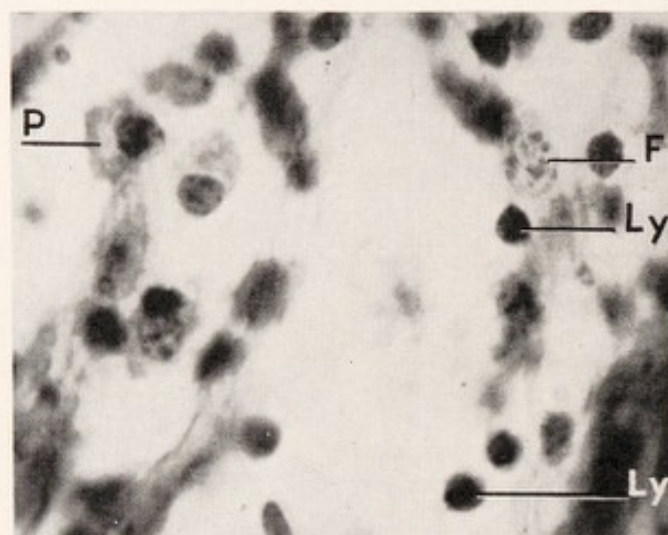


FIG. 102.—Characteristic cell forms present in chronic pulpitis: lymphocytes and plasma cells (polyblasts). P, plasma cell; Ly, lymphocytes; F, fibroblast. (See also Fig. 110.)

2. Chronic Hypertrophic Pulpitis (Pulp Polypus).—In some cases the stimulus of a chronic, low-grade inflammation leads to hypertrophic pulpitis. Two factors are essential for the development of this condition: the pulp must be highly resistant, and a wide communication with the oral cavity must be present. For these two reasons chronic hypertrophic pulpitis is for the most part found in children and adolescents, with their naturally high general tissue resistance, and in broken-down teeth that allow free proliferation of the hypertrophic tissue. Clinically, the exposed pulp appears as a red, rounded growth in the pulp chamber of a badly destroyed tooth. This mass of tissue varies from the size of a pinhead to the size of a pea, and is only slightly more sensitive to touch than normal gingival tissue. Upon examination with an instrument it is found that this red mass of soft tissue connects with a strand of living tissue that extends into the root canal. From the similarity of this condition to a polypus of the mucous membrane, this inflammatory hypertrophy of the pulp tissue is also called pulp polypus. Sometimes the surface of the hypertrophied pulp is raw and bleeds easily during mastication or when touched by an instrument. In other cases the surface is much more resistant, being covered with epi-

thelium and having the appearance of gingival tissue. This epithelium originates from the surrounding gingival epithelium, from which it is transplanted in the form of minute cell grafts upon the raw pulp surface where it develops into a continuous epithelial cover.

The tissue of a hypertrophied pulp is typical granulation tissue (see page 140) containing newly formed bloodvessels, fibroblasts (proliferated pulp cells), and polyblasts (inflammatory exudate cells).

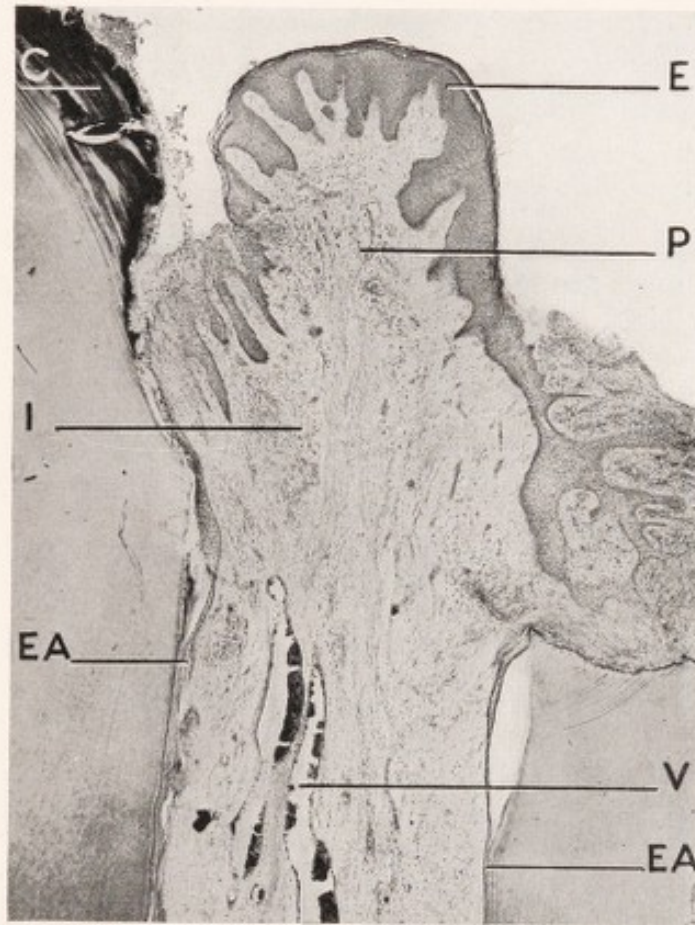


FIG. 103.—Chronic hypertrophic pulpitis (pulp polypus). Lower bicuspid. C, carious dentin on the inner wall of the broken-down crown; E, stratified squamous epithelium with marked keratinized layer; P, hypertrophied pulp tissue; I, inflammatory exudate cells in the pulp tissue; V, enlarged bloodvessels; EA, epithelial attachment to the wall of the root canal. (Boulger, Jour. Dent. Res.)

A case of a hypertrophied pulp with an epithelial lining was recently reported by Boulger (Fig. 103). In this tooth, a lower bicuspid of an adolescent, the pulp polypus was completely covered by a layer of stratified squamous epithelium which formed an epithelial attachment to the wall of the root canal all around the opening into the canal. Underneath the hypertrophied part of the pulp, the walls of the root canal had been greatly narrowed by the deposition of calcified masses and secondary dentin. This condi-

tion may be considered a defensive attempt of the pulp to throw up a barricade of hard tissue against advancing inflammation. In the apical part, the root canal contained normal pulp tissue and in the upper part, fibrous tissue (granulation tissue) with considerable infiltration and dilatation of the bloodvessels.

BIBLIOGRAPHY.

- ADRIEN, W.: Beiträge zur Histologie der Pulpitis chronica granulomatosa, Deutsch. Mon. f. Zhk., 1923, **41**, 47.
- BOULGER, EARL P.: Histologic Study of a Hypertrophied Pulp, Jour. Dent. Res., 1931, **11**, 257.
- CAHN, L. R.: Pathology of the Dental Pulp, Dent. Items Int., 1926, **48**, 1.
- Apical Foci of Infection on Teeth in Which the Pulps are Still Alive, Dent. Items Int., 1926, **48**, 239.
- COOLIDGE, EDGAR D.: Pulp Pathology and Treatment Problems, Jour. Am. Dent. Assn., 1928, **15**, 1623.
- EULER, H.: Beiträge zur Histologie der chronischen Pulpitis, Deutsch. Zahnärztl. Wehnschr., 1925, **28**, 31.
- GOFFUNG, E. M.: Zur Frage über das aktive Mesenchym der Pulpa, Deutsch. Mon. f. Zhk., 1928, **46**, 1260.
- HATTON, E. H.: Pulp Pathology from the Standpoint of the Clinician, Jour. Am. Dent. Assn., 1930, **17**, 2262.
- HESS, WALTER: Nekrose der Pulpa, Handw. d. ges. Zhk., vol. **3**, p. 2274.
- HOPEWELL-SMITH, A.: Adventitious Dentin and Infection of the Dental Pulp, Dent. Items Int., 1925, **47**, 477, 557.
- Some Remarks on the Human Dental Pulp: Its Reactions to Injury, Its Diseases and Their Immediate and Remote Complications and Sequelæ, Dental Cosmos, 1924, **66**, 489, 601.
- KLEIN, ALEXANDER: Zur Histologie des Pulpapolyphen, Ztschr. f. Stom., 1927, **25**, 44.
- MÜLLER, OSCAR: Epithel im Wurzelkanal, Deutsch. Mon. f. Zhk., 1928, **46**, 562.
- OTTOLENGUI, R., and CAHN, L. R.: The Pathology of the Dental Pulp, the Practical Significance of Such Knowledge in the Treatment of Vital and Pulpless Teeth, Dent. Items Int., 1926, **48**, 897.
- PALAZZI, SILVIO: Experimentelle Untersuchungen über das Problem der Heilungsvorgänge in der blossgelegten gesunden Pulpa. Die Veränderungen in der entblösten gesunden Zahnpulpa, Ztschr. f. Stom., 1927, **25**, 91.
- THOMA, KURT H.: A Practical Discussion of Pulp Disease Based on Microscopic Study, Dent. Items Int., 1925, **47**, 637.
- The Infected Vital Dental Pulp an Important Focus of Systemic Disease, Jour. Dent. Res., 1928, **8**, 529.
- A Comparison of Clinical, Roentgen and Microscopic Findings in Fifteen Cases of Infected Vital Pulps, Jour. Dent. Res., 1929, **9**, 447.
- WEBER, R.: Zur Kenntnis des Auftretens von Fett am Zahn, Vrtljschr. f. Zhk., 1926, **42**, 64.

CHAPTER V.

ACUTE INFLAMMATION OF THE APICAL PERIODONTAL TISSUES (ACUTE PERIODONTITIS).

It is the author's opinion that every inflammatory reaction occurring in the soft tissue and bone surrounding the root surface should be given the general name periodontitis, and that this term should be employed in the general description of any inflammatory conditions in this area regardless of the etiology or type of inflammation. Many of the other names that are found in dental literature describing this condition are either derived from older terms (such as "pericementitis" from "pericementum," a term that has been replaced by "periodontium") or are too specific (for example, "periapical abscess": not every inflammation of the periapical tissue causes abscess formation). In the detailed description of the different clinical types of periodontitis, however, other terms will be used for conditions, such as dento-alveolar abscess, granuloma, and cysts, which may occur in later stages of periodontitis.

Since there are great variations in the causes and manifestations of periodontitis, it seems advisable to introduce a classification which will help in understanding and describing the pathology of the condition. According to etiology periodontitis may be classified as follows:

- A. Traumatic periodontitis.
- B. Chemical periodontitis.
- C. Infective periodontitis.

According to the form of inflammation and to the time that has elapsed since the beginning of the pathological changes, periodontitis may be divided into acute periodontitis and chronic periodontitis. This chapter will deal only with the acute condition.

ACUTE TRAUMATIC PERIODONTITIS.

Acute traumatic periodontitis is the reaction of the periodontal tissues to injury caused by a trauma of any kind. The most common causes of acute traumatic periodontitis are: (1) blow or fall upon the mouth and teeth; (2) injuries during dental operations (separation, malleting, root canal operations), and (3) excessive occlusal stress.

1. **Acute Traumatic Periodontitis Following a Blow or Fall.**—Following a blow or fall upon the mouth, one or several of the teeth are often found loosened in their sockets and sore to touch or percussion due to the tearing of the tissues and the extravasation of blood and lymph into the periodontal membrane. In mild cases this soreness usually disappears without any treatment after a few days. Although an acute injury of this type in human tissues has not yet been studied under the microscope, similar conditions have been experimentally created in animals and the tissues have been examined microscopically. In such specimens the force of the trauma has resulted in the tearing of the fibers of the periodontal membrane in many places and in minute hemorrhages from the blood-vessels of the periodontal membrane. Immediately following the acute injury white blood cells accumulate in the damaged area and aid in the reparative changes by a phagocytic removal of tissue debris and blood remnants. At the same time, new fibers are built through the activity of the connective tissue cells, and, after a certain period of time, the periodontal membrane will be completely regenerated and every evidence of the trauma will have disappeared.

2. **Acute Traumatic Periodontitis Following Injuries During Dental Operations.**—In the practice of dentistry slight traumatic periodontitis is sometimes produced by the dental operation itself. One of the most common causes of mild, temporary periodontitis is the improper use of the metal separator. To a certain extent the fibers of the periodontal membrane will yield to the force of tooth separation without injury, but if the separation is continued beyond the limit of tensile strength of the fibers, fiber bundles will be torn, slight hemorrhages will occur, and as a result, the teeth that were separated will be sore for one or several days. This is especially true if the separation is carried out while the teeth are under the influence of an anesthetic, and pain, the natural tissue protection, is not present to warn the dentist. Urban, Beisler and Skillen, in a recent report on forced tooth separation in animals, clearly demonstrated hemorrhages and tearing of the periodontal fiber bundles in their specimens. These tissue injuries cause a reactive inflammation which, as a rule, terminates in the repair of the damaged tissue.

The type of traumatic periodontitis that has the greatest clinical significance is that caused by injuries in root canal operations. Occasionally it happens that a broach or reamer passes through the apical foramen and causes injury to the apical periodontal membrane and even to the bone beyond the foramen. Clinically, such

damage to the periapical tissues is usually indicated by hemorrhage through the root canal and by soreness of the tooth. In these damaged tissues a low-grade inflammation occurs, which either may terminate in healing and scar formation or may develop into chronic periodontitis. This latter development will always take place when the injured, apical tissue is invaded by pathogenic microorganisms during or following the root canal operation.

Even the simple removal of a vital pulp under anesthesia is followed by a low-grade, traumatic periodontitis, provided that the injured area is close enough to the apical foramen. This mild form of periapical irritation will usually heal, and the formation of cementum will take place later on over the root end of such a tooth.

Lateral perforations of the roots by means of rotating instruments (burs) result in a severe traumatic periodontitis that usually leads to the loss of the tooth. Injuries of this type are clinically known as root perforations. If such a perforation occurs in the pulp chamber it can be seen easily, but if the perforation is located in the root canal, it can be diagnosed only by the unusual amount of hemorrhage from the canal and by means of radiographic examination. At any rate more or less extensive destruction of the periodontal membrane and bone is present at the place of perforation; leukocytes accumulate around the perforation, and in most cases a chronic inflammation develops with formation of granulation tissue and progressive destruction of both soft and hard structures. In favorable cases healing and scar formation have been observed after a traumatic perforation of the root of a tooth.

3. Acute Traumatic Periodontitis Following Excessive Occlusal Stress.—An example of this type of injury is the loosening and soreness of a tooth after unexpectedly biting upon a hard object, such as a shot, during mastication of food. Under the microscope the same changes occur in the periodontal membrane after such an injury as are found after a light blow or kick upon a tooth. Another cause of acute traumatic periodontitis is the insertion of a restoration (crown, filling) in such a manner that it interferes with normal occlusion. Then the tooth is subjected to an increased amount of occlusal force and will develop traumatic periodontitis that persists until the obstacle to the normal occlusion has been removed. The tissue changes in this case are the same as described above: slight lacerations and hemorrhages in the periodontal membrane with reactive infiltration that will readily disappear after the cause of the mechanical injury has been removed.

CHEMICAL PERIODONTITIS.

Chemical periodontitis is due to the action of drugs which have been brought into the pulp canal during root canal therapy and which have penetrated beyond the apical foramen. From experimental investigations, it seems that practically every drug in a root canal causes at least a slight periapical inflammation. The amount of inflammation is in direct proportion (1) to the concentration and penetrative power of the drug, (2) to the amount of drug that is used, (3) to the time the drug is allowed to act upon the tissues, and (4) to the width of the apical foramen. It is obvious that drugs with a high penetrating power will, under otherwise identical circumstances, cause more tissue destruction than drugs with coagulating and therefore rather self-limiting properties. It is easily understood also that in a young person the drug will act more rapidly and extensively upon the apical tissues than in an old individual with very narrow or almost obliterated root canals.

Clinically, the best known type of toxic periodontitis is that which follows the incorrect application of arsenic trioxide upon the pulp. It has been found experimentally in animals that a slight amount of periapical irritation and inflammation always accompanies the use of arsenic trioxide; however, in the first twenty-four or forty-eight hours the changes in the periodontal membrane consist merely of hyperemia and infiltration without severe tissue injury. In this initial stage healing occurs after the removal of the drug from the pulp chamber. If, however, the arsenic trioxide has been allowed to act upon the periapical tissues for a longer period of time, the periodontal membrane will be injured beyond repair; the bone becomes involved, and the final outcome will be the formation of a chronic periapical inflammation unless the tooth has been extracted in the earlier stages of periodontitis.

Since it is easy to produce chemical periodontitis experimentally in animals, the development and pathology of this condition has been the subject of a number of experimental studies, particularly on dogs. In 1930 Feldmann published a monograph which is entirely devoted to the subject of chemical periodontitis in dogs. He used practically all known drugs and methods of treatment and studied their influence upon the apical tissues over various lengths of time. Feldmann showed that all drugs sealed in the root canal caused at first a reactive infiltration of the periapical tissues which was followed after a period of several months by various reactions, depending upon the types of drugs and treatments used. Roots

filled with guttapercha or oxyphosphate cement after using a mild drug were found to be surrounded by normal periodontal tissue, and it was observed that cementum had been deposited over the root end. Other teeth, in which powerful and penetrating drugs had been sealed in the root canals, revealed a progressive inflammation and destruction of the apical hard and soft tissues, without evidence of a healing tendency.

Similar experiments were carried out by Coolidge. After removing the pulp from the incisors of dogs, he sealed various drugs in

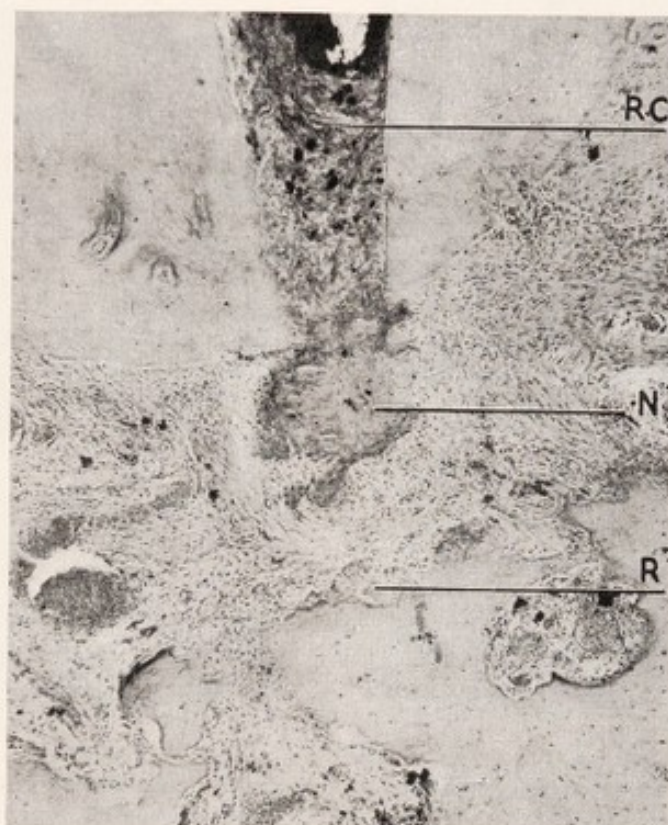


FIG. 104.—Acute chemical periodontitis produced by removing the pulp and sealing phenol into the root canal of a dog for twenty-one days. Circumscribed coagulation necrosis of the periodontal membrane at the apical foramen. RC, root canal; N, necrosis of the periodontal membrane; R, resorption of alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

the root canals for a period of twenty-one days. The histological findings revealed great variation in the extent and type of reaction. A few characteristic specimens will be reproduced here to illustrate the tissue changes in chemical periodontitis.

When phenol was sealed in the root canal of a dog, the drug, due to its strong coagulating power, caused necrosis of the periodontal membrane (Fig. 104). The surrounding periodontal tissue shows but few changes, its structure being well-preserved. In the alveolar bone osteoclastic resorption is taking place. After sealing formo-

cresol in the root canal of a dog, a similar, circumscribed mass of coagulated tissue resulted at the apical foramen; the periodontal membrane in the vicinity of this necrotic tissue presented the symptoms of a low-grade inflammation with marked hyperemia of the bloodvessels.

When drugs are used that have not such a marked power to coagulate albumen, for instance, essential oils or chlorine preparations, a much more extensive and diffuse inflammation of the periapical



FIG. 105.—Beginning formation of an acute periapical abscess produced by sealing eugenol in the root canal of a dog's tooth. RC, root canal containing the drug; A, abscess formation in the periodontal membrane; I, round cell infiltration (leukocytes) in the periphery of the abscess; V, hyperemic bloodvessels of the periodontal membrane. (Courtesy of E. D. Coolidge.)



FIG. 106.—Periapical abscess produced by sealing eucalyptol in a dog's tooth. A, abscess cavity filled with pus; R, extensive resorption of the alveolar bone surrounding the abscess cavity. (Coolidge, Jour. Am. Dent. Assn.)

tissues is the result. A mild case of diffuse chemical periodontitis is illustrated in Fig. 105, in which tooth oil of cloves was sealed for twenty-one days. At the apical foramen there is a dense inflammatory infiltration which is continued without a sharp borderline into the adjacent periodontal membrane. A much more severe form of acute periodontitis resulted from sealing eucalyptol, an essential oil, into a dog's tooth for twenty-one days (Fig. 106). The entire periapical region is densely infiltrated with inflammatory round cells; this inflammation has caused considerable bone resorp-

tion and a widening of the periodontal space. Near the center of inflammation at the apical foramen, the periodontal tissue has broken down completely and an abscess has developed.

Chlorine preparations, due to their power to dissolve albumen, produce a reaction of the periapical tissues similar to Fig. 106. From findings of this kind in dogs, however, no definite conclusions can be drawn as to the advisability of using or discarding certain drugs in the practice of dentistry. The main difference between these animal experiments and the condition in man is that in the dog's tooth an intact, uninfected pulp is removed and the drug is placed upon a fresh wound in heretofore healthy tissue, while, in the practice of root canal therapy in man, pulp tissue and root canal are usually infected; therefore, the use of penetrating drugs with bactericidal properties is necessary to control the existing infection. Although drugs of the latter type may prove harmful to the tissues of dogs, we must not draw the direct conclusion that they should not be used in man, because the same drugs that injure and irritate intact, vital tissue by their penetrating power may be, for the same reason, of great value in fighting bacteria in infected human root canals and periapical tissues.

ACUTE INFECTIVE PERIODONTITIS AND ACUTE DENTO-ALVEOLAR ABSCESS.

1. **Etiology and Pathology of Acute Infective Periodontitis.**—The inflammation of the apical periodontal tissues due to the presence of pathogenic microorganisms is the most common form of periapical inflammation and is the form with which the dentist is most frequently concerned.

From the standpoint of clinical diagnosis, the infections of the periapical tissues are usually divided into acute and chronic forms. Acute infective periodontitis is characterized by one or several of the following clinical symptoms: pain, swelling, heat and redness of the soft tissues overlying the tooth, fever. Chronic inflammation of the periapical region sometimes causes no clinical symptoms at all, or perhaps only a temporary feeling of slight uneasiness or discomfort in the affected tooth.

In studying infection of the periapical tissue under the microscope, it is impossible to draw a sharp line between periodontitis (inflammation of the periodontal membrane) and alveolar osteitis (inflammation of the alveolar bone). Whenever an infective process has reached or passed the apical foramen and involves the apical

periodontal membrane, the microscope reveals an alteration of the alveolar bone long before this bone change would be visible in the radiograph. The bloodvessels in the bone surrounding the inflamed periodontal membrane are hyperemic and contain a large number of polymorphonuclear leukocytes. The alveolar bone in the periphery of the bloodvessels is densely beset with polynuclear osteoclasts (giant cells) (Fig. 107). Thus it is impossible to draw a sharp line between inflammation of the periodontal soft tissues only and inflammation also involving the bone; therefore, these two conditions will be discussed under one heading.

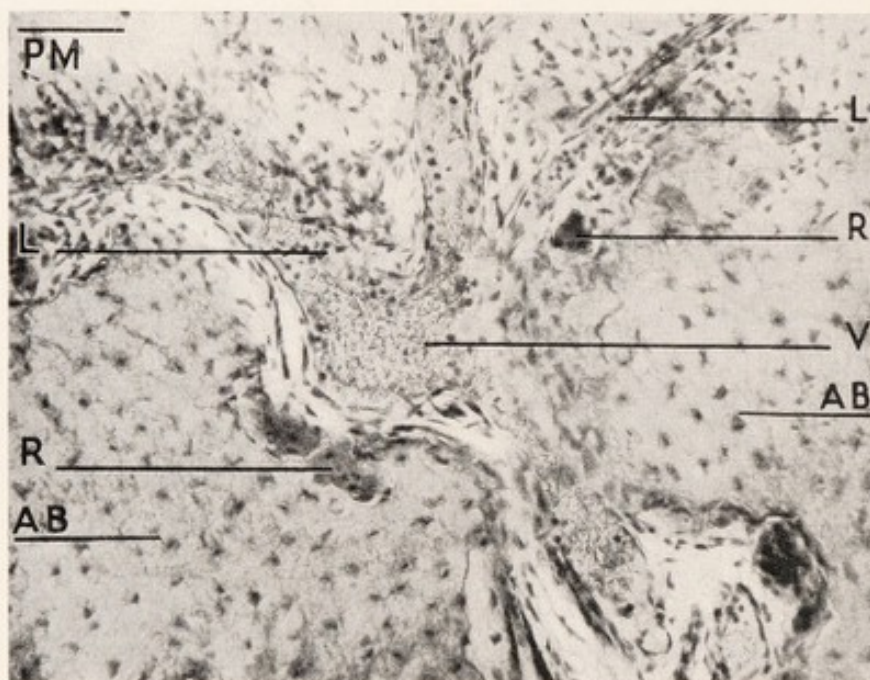


FIG. 107.—Beginning osteitis of the alveolar bone in case of acute infective periodontitis. PM, periodontal membrane; V, hyperemic bloodvessel (vein) leading from the periodontal membrane into the bone; L, polymorphonuclear leukocytes in the vessel; AB, alveolar bone; R, giant cell resorbing the alveolar bone adjacent to the bloodvessel.

Acute infective periodontitis is caused by the action of pathogenic microorganisms that reach the apical foramen through the pulp canal and spread into the periodontal tissues. In the clinical examination of a case of acute periodontitis it is usually easy to trace the way by which the infection reached the periapical region. The crown of the tooth is usually destroyed by caries; the pulp is decomposed; the root canal is empty or filled with débris, or contains a root canal filling. Frequently infective periodontitis develops in connection with root canal therapy. This will, for instance, be the case when, after removal of a pulp, the tooth gradually becomes more and more sensitive and painful until finally typical perio-

dontitis develops with pyogenic microorganisms present in root canal and periapical region. In such a case it can be readily assumed that these bacteria gained access to the periapical tissues through the root canal, usually by a lack of asepsis in the operator's technique.

On the other hand, there is a group of cases in which the etiology of acute periodontitis or of acute alveolar osteitis is not so evident at first glance. A tooth may present symptoms of an acute inflammation of the apical periodontal tissues with pain, soreness to percussion, elongation, loosening and, in addition, swelling and redness of the overlying soft tissues, and still the crown may appear intact, no caries or defect being visible. In such a case the dentist, in making a careful investigation into the history of the patient, may be able to learn of a trauma (blow, kick) that occurred some time previous. A sterile necrosis of the pulp followed the injury. This condition may be present without any clinical symptoms and can be diagnosed only by the vitality test. A severe, acute, purulent inflammation may suddenly develop from such a tooth, and pyogenetic bacteria (staphylococci) will be found in the pus formed during this process. Whether these bacteria are carried by way of the blood stream to the tooth, where they find good soil for development in the disintegrated content of the pulp canal (hematogenous infection), or whether the necrotic pulp tissue becomes infected through minute cracks or defects in the tooth substance or in fillings (stomatogenous infection) is uncertain and difficult to decide.

The first symptoms of the presence of infection in the periodontal membrane are the same as are found elsewhere in the connective tissue of the body, namely, hyperemia and stasis in the vessels, migration of white blood cells (polymorphonuclear leukocytes) from within the bloodvessels into the surrounding connective tissue (diapedesis), and diffusion of fluid through the walls of the vessels (edema). Sooner or later, depending upon the virulence of the bacteria and upon the resistance of the body, the infiltrated tissue breaks down, and pus is formed around the root end, a condition called acute purulent periodontitis. As a rule the alveolar bone is already involved when the process reaches this stage, and osteoclasts are found in great number resorbing the inner lamina of the alveolus and the cancellous bone behind it. In that way the process of pus formation and the breaking down of tissue spread through the bone, and a purulent alveolar osteitis develops.

The acute inflammation of the periodontal membrane causes very characteristic clinical symptoms and, therefore, the study of the histopathology of this condition must be carried through in close

connection with the clinical picture. The first reactions of the periodontal soft tissues against the invasion of bacteria are hyperemia, edema and leukocytic infiltration. These reactions cause an increase in the volume of soft tissues and, thus, the early clinical symptoms are manifestations of increased tissue pressure at the apex. The tooth will appear slightly elongated, protruding from the alveolus, as a result of the increased tissue tension at the root end; the tooth may even loosen slightly as the fibers of the periodontal membrane become edematous. The involved tooth is sore, either spontaneously or on percussion and during mastication, as the nerves in the periodontal membrane are irritated and pressed by the inflammatory process. It must be understood, however, that not all the symptoms of acute apical infection, namely, elongation, loosening and soreness, are necessarily present in every case, although they will be observed in typical cases. If a tooth be extracted in this stage of acute periodontitis, the hyperemia of the periodontal membrane is indicated by the intense redness of the soft tissue that adheres to the apical part of the extracted root.

After the infiltration of the periodontal membrane has reached a certain degree, the soft tissues begin to break down. Pus accumulates in the periodontal space and in the marrow spaces of the bone around the opening of the pulp canal. In this stage the patient suffers severe pain; the tooth is often very sore, and continuous pulsating pain indicates the presence of a purulent process in the depth of the alveolus.

A symptom that must not be overlooked in the clinical examination of cases of this type is the change in the regional lymph nodes. These lymph nodes are located on the lower border of the mandible. The anterior group, the submental nodes, are closely related to the lower anterior teeth; the posterior group, the submaxillary nodes, are connected with the upper and lower bicuspid and molars. During acute purulent periodontitis these nodes are very often swollen and tender when palpated.

If in this stage of the process drainage be established through the root canal by trepanation of the tooth and removing debris and root filling material, thick creamy pus will sometimes be discharged through the root canal, after which the patient feels immediate relief from the throbbing pain. The same relief is obtained if the tooth be extracted; the purulent inflammation then has open drainage through the alveolus and clears up in a short time.

If drainage is not established, the purulent apical inflammation spreads through the bone and soon reaches the outer surface of the

alveolus. Here the process is usually temporarily detained by the rather resistant layer of periosteum that covers the outer surface of the bone. This stage is spoken of as acute dento-alveolar abscess.

2. Acute Dento-alveolar Abscess.—Topography of the Root Ends and Surrounding Structures.—As the inflammation approaches the surface of the bone, a new and very important symptom is observed that is characteristic of acute, purulent, dento-alveolar abscesses, namely, a collateral edema and swelling of the soft tissues of the face. This condition is known as cellulitis. It usually reaches its height when the pus has perforated the outer lamina of the alveolar process and appears under the periosteum as a subperiosteal abscess.

If the inflammatory process starts from an upper tooth, the upper lip, cheek and, in severe cases, the lower and also the upper eyelid are swollen. If a lower anterior tooth is involved, the lower lip and the chin region are the seat of the collateral edema; in case of a lower posterior tooth the swelling may extend over the lower part of the cheek, the angle of the mandible, and sometimes downward into the submaxillary region.

After the purulent process has perforated the outer plate of the alveolar process, it spreads along the bone surface, separating the periosteum and the mucous membrane from the bone. This soft tissue lining is elevated by the pressure of the underlying pus, and fluctuation is found when this elevation is palpated with the finger.

The lymph nodes at the lower border of the mandible are usually involved in this stage. There is a rise in temperature; in addition, headache, constipation and general prostration are not uncommon symptoms that accompany such severe purulent processes in the jaw.

Finally, the pus forces its way through the overlying soft tissues and appears on the surface; or the swelling is incised, the abscess is opened, and the pus is discharged. With the free drainage of the pus, the acute dento-alveolar abscess usually subsides within a short time; pain, swelling and fever disappear, and the patient feels comfortable again. The final outcome of the process is either extraction of the tooth and healing of the bone changes, or transition from the acute into a chronic inflammatory process. By far the largest number of acute dento-alveolar abscesses actually develop as a flaring-up of a preëxisting chronic periapical inflammation; after the acute symptoms have subsided, the old chronic process is left, forming a potential source of new acute attacks (see Fig. 130).

The place where the pus appears on the surface in an acute dento-alveolar abscess depends upon the topographic relation

between root end and surrounding parts. Usually the pus makes its way through the jaw in the direction of least resistance; that means, it will appear on that side of the jaw where there is the least thickness of bone between root end and outer soft tissues. It is, therefore, very important to know how the variations in resistance and the anatomical relationship of the apices to their surroundings determine the way in which the pus reaches the bone surface.

In upper incisors, cuspids, and on the buccal roots of upper bicuspids and molars the pus is most likely to make its way through the thin labial or buccal plate of the alveolar process to appear in the vestibulum. Sometimes in individuals with low alveolar processes and long roots, it may happen that an inflammatory process at the apex of a central incisor perforates into the floor of the nose, producing a condition similar to a furuncle of the nose.

The apex of the upper lateral incisor is sometimes found lingually to the row of the other front teeth, lying close to the lingual plate of the alveolar process. An acute dento-alveolar abscess originating in this tooth may, therefore, erupt toward the palate and form a palatal abscess. An acute dental abscess on the palate frequently exists for some time before perforation takes place, because of the extreme rigidity and resistance of the overlying soft tissue lining; in the meantime the pressure of the subperiosteal abscess may have detached the palate lining to a great extent. This is especially true of palatal abscesses originating from the lingual roots of upper molars, in which case the abscesses often have a tendency to spread under the periosteum back toward the soft palate instead of perforating into the oral cavity. In case of a palatal abscess, usually no edema or cellulitis of the face is present.

The upper cuspid has a long root, the end of which is sometimes located in a much higher level than the vestibulum oris. An inflammatory process originating from the apex of this tooth may extend upward into the canine fossa and then perforate through the skin below the inner angle of the eye. In a case of chronic periodontitis of the cuspid, a sinus may develop in this area which may be confused with a fistula of the naso-lacrimal duct.

In an acute dento-alveolar abscess originating from upper bicuspids and molars, the relationship between these teeth and the maxillary sinus (antrum of Highmore) must be considered. The participation of the sinus in inflammatory processes at the root ends will depend upon the individual topography: if the roots are close to or even rising above the floor of the sinus, the sinus will undoubtedly be affected; if the floor of the sinus is far above the

root ends and the latter are close to the buccal bone surface, the abscess most likely will erupt into the vestibulum (see Fig. 121).

In the mandible, the relation of the root ends to the surrounding structure is mainly dependent upon the length of the roots, the height of the alveolar process, and the location of the attachment of the tissues to the outer and inner plane of the lower jaw. Usually an acute dento-alveolar abscess originating from a lower tooth will erupt into the labial or buccal vestibulum; here it appears as an acute inflammation and swelling of the soft tissues overlying the mandible, accompanied by a cellulitis of lip, chin or cheek. Occasionally a perforation toward the lingual side is observed, in which case the pus appears in the floor of the mouth. This is especially true of the lower second and third molars, the roots of which are separated from the outer surface of the bone by a thick layer of compact bone; therefore, a perforation toward the lingual side is more likely to occur.

A not uncommon complication of an acute, purulent, periapical inflammation of a lower tooth is the perforation of the pus through the skin along the lower margin of the body of the mandible. This is particularly observed in the case of acute dento-alveolar abscesses developing from the lower incisors and the lower first molar in young persons. These teeth frequently have long, strong roots that are embedded deep in the body of the mandible; if the teeth have not fully erupted and if, in addition, the mandible is low, the apices will be much closer to the lower margin of the mandible than to the buccal or lingual surface. Consequently, pus developing at the root ends will be more likely to perforate along the lower margin through the skin than to go the long way into the vestibulum.

BIBLIOGRAPHY.

- COOLIDGE, EDGAR D.: Reaction of Dog Tissue to Drugs Used in Root Canal Treatment, *Jour. Am. Dent. Assn.*, 1932, **19**, 747.
- FELDMANN, GREGORY: Die Apikale Parodontitis im Lichte des Experimentes, Berlin, Meusser, 1931.
- REBEL, H. H.: Arsenstudien, Arsen oder Injektionsanästhesie, *Deutsch. Mon. f. Zhk.*, 1924, **42**, 60, 545.
- ROSENTHAL, ARTHUR: Die Wirkung der arsenigen Säure, insbesondere des Nervarsens, auf das Periodontium des Hundes, *Schweiz. Mon. f. Zhk.*, 1928, **38**, 1.
- SICHER, H., and TANDLER, J.: Anatomie für Zahnärzte, Berlin, Springer, 1928.
- URBAN, L. B., BEISLER, E. H., and SKILLEN, W. G.: Tissue Disturbances Caused by Mechanical Separation of the Teeth of the Dog, *Jour. Am. Dent. Assn.*, 1931, **18**, 1943.

CHAPTER VI.

CHRONIC INFLAMMATION OF THE APICAL PERIODONTAL TISSUES (CHRONIC PERIODONTITIS).

CHRONIC inflammation of the apical periodontal tissue is characterized by the presence of a chronic osteitis with transformation of periodontal membrane and alveolar bone into granulation tissue. Granulation tissue is of great significance in the study of chronic inflammatory processes around the root ends of teeth with infected root canals, since its presence precedes all other possible manifestations and developments. It is, therefore, important to have accurate knowledge of the characteristics of granulation tissue and to understand its rôle in chronic inflammation before proceeding with this study.

GRANULATION TISSUE, ITS STRUCTURE AND SIGNIFICANCE.¹

Granulation tissue may be defined as newly-formed tissue that is the result of a productive inflammation of chronic character. Its formation may be considered as the defensive or reparative reaction of the organism inasmuch as in many instances granulation tissue initiates or precedes the healing of wounds.

A typical instance of the development of granulation tissue is the healing of a large, open wound on the body surface. A few days after the epithelium has been lost, the raw surface is covered by a newly-formed tissue that has an uneven, granulated surface, giving it the appearance from which it derives its name. Soon the entire wound surface is covered by this new tissue; the epithelium from the margins of the wound begins to grow over the granulation tissue, which subsequently becomes transformed into scar tissue.

Histologically, granulation tissue is composed of several tissue elements which are listed in the order of their frequency and significance: Fibroblasts; capillaries; inflammatory exudate cells; giant cells.

¹ In general pathology two kinds of granulation tissue are distinguished: specific and non-specific. Specific granulation tissue is the result of infection with a special microorganism (*Bacillus tuberculosis*, *Spirochæta pallida*). Non-specific granulation tissue may develop as a result of any irritation or inflammation of long standing, and it is not associated with the action of any particular type of microorganism. Only the latter type of granulation tissue will be considered here.

1. **Fibroblasts.**—Fibroblasts (connective tissue cells) constitute the bulk of granulation tissue. They are large cells with pale, oval nuclei containing fine, dust-like chromatin and several nucleoli. The protoplasm of the fibroblasts has spear-shaped processes that extend from one cell to another. The product of the fibroblasts is a fibrous intercellular substance, the connective tissue fibrillæ; these fibrillæ form, together with the cells, a fine network which is the basis of every soft tissue of connective tissue type.

The fibroblasts in granulation tissue develop by mitosis from the preëxisting fibroblasts in the inflamed or injured area; in a skin wound, for instance, the fibroblasts in the granulation tissue are derived from the fibroblasts in the subcutaneous connective tissue around the wound. In a chronic inflammation of the alveolar bone, the fibroblasts in the granulation tissue develop from the periodontal membrane and from the connective tissue that normally covers the bone in the form of periosteum.

The fibroblasts do not, in a strict sense, take part in the inflammatory process; they have rather a reparative or regenerative function, such as incapsulating an inflamed area, replacing lost tissue, and forming scar tissue.

2. **Capillaries.**—Granulation tissue contains a great number of capillaries, whose presence gives this tissue its typical red color. These capillaries develop from the bloodvessels in the surrounding tissue. From these bloodvessels solid sprouts of proliferating endothelial cells invade the forming granulation tissue; later they are transformed into true capillaries by being hollowed from within. (Fig. 108.)

3. **Exudate Cells.**—The so-called inflammatory round cells or exudate cells are the most characteristic constituent of granulation tissue. The usual term "inflammatory round cells" or "round cell infiltration" has only a very general meaning. "Round cells" merely indicates that the cells in consideration do not have an oval nucleus and irregular protoplasm like the fixed connective tissue cells (fibroblasts) but that the cells have a more rounded form. It is necessary to study the cell types under high magnification and to consider their origin in order to understand their significance. Two main types of inflammatory round cells can be differentiated: (1) leukocytes and (2) polyblasts.

(a) *Leukocytes.*—Neutrophilic granular leukocytes with lobulated nuclei (polymorphonuclear leukocytes) are, under normal conditions, always found in the blood stream and there constitute from 65 to 75 per cent of the total amount of white blood cells. Their

nuclei consist of several nuclear fragments that are held together by very delicate chromatin bridges. They develop in the bone-

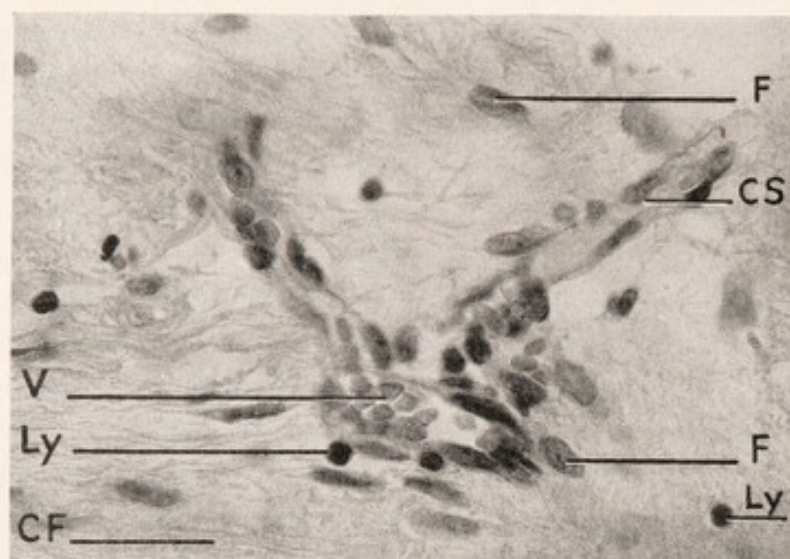


FIG. 108.—Capillaries in granulation tissue in the oral cavity. V, capillary blood-vessel containing erythrocytes; CS, capillary sprout growing from the vessel into the surrounding granulation tissue; F, nuclei of fibroblast; CF, connective tissue fibrillae; Ly, polyblast (lymphocyte).

marrow from the myelocytes. The polymorphonuclear leukocytes are not found in normal connective tissue. However, they accumulate at once in the small bloodvessels of injured or infected areas;

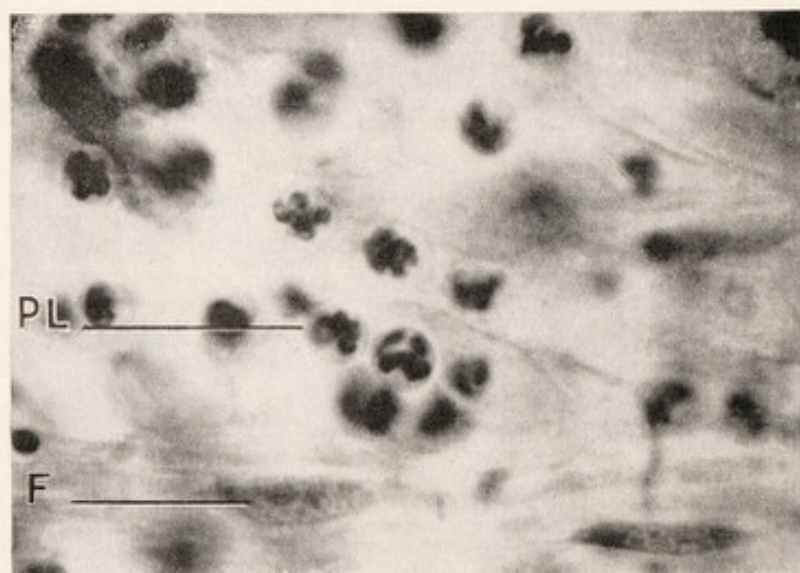


FIG. 109.—Typical cell forms in case of acute purulent inflammation of the periapical tissue. Infiltration of the connective tissue with polymorphonuclear leukocytes, PL, with round protoplasmic bodies and lobular nuclei (see also Fig. 91). F, fibroblast.

then they migrate through the walls of these cells and appear in large numbers in the connective tissue (Fig. 109). Polymorpho-

nuclear leukocytes are especially numerous in acute suppuration; they are the main cellular constituents of pus. In the later stages of inflammation, the leukocytes degenerate and disappear. For this reason they are observed only occasionally in typical granulation tissue, as the latter is the result of chronic inflammations of longer standing.

The function of polymorphonuclear leukocytes is the destruction of the pyogenic microorganisms in the inflamed tissue. This is performed in two ways: (1) by phagocytosis and intracellular destruction of the bacteria, and (2) by a discharge of bactericidal and detoxicating substances. These substances seem to be set free whenever large numbers of leukocytes disintegrate as in the case of acute suppuration.

(b) *Polyblasts*.—Maximow, who made a very thorough study of the development and morphology of the various cell types involved in inflammatory reactions, calls all inflammatory exudate cells, with the exception of the polymorphonuclear leukocytes, "polyblasts," thus indicating their different shape and origin. According to Maximow, these cells have a double origin: they are derived either from local connective tissue cells, histogenous polyblasts, or from non-granular leukocytes, hematogenous polyblasts. The histogenous polyblasts develop from the histiocytes or resting wandering cells of the loose connective tissue (Fig. 4). Under the influence of inflammation, the histiocytes become active and are transformed into large cells with amœboid motility; these cells migrate toward the center of inflammation where they assume phagocytic properties and destroy bacteria and cell debris. The hematogenous polyblasts originate from the lymphocytes and monocytes (mononuclear leukocytes) of the normal blood. In case of inflammation these blood cells, together with the polymorphonuclear leukocytes, migrate through the walls of the vessels and appear in the connective tissue; but whereas the polymorphonuclear leukocytes disappear shortly thereafter, the lymphocytes and monocytes persist in the field of inflammation and soon are transformed into large amœboid phagocytic polyblasts. The hematogenous polyblasts cannot be distinguished from the histogenous polyblasts.

According to Maximow, the endothelial cells of the capillary walls do not contribute to the formation of exudate cells. The endothelial cells of the adult organism are highly differentiated cell elements that cannot be transformed into polyblasts. The latter are derived wholly from the two sources that already have been mentioned, namely, the histiocytes and the white blood cells.

Polyblasts are always found in large numbers in granulation tissue. They appear in different forms and sizes. Some polyblasts have simple, round nuclei similar to those of lymphocytes. Others have nuclei that are oval or kidney-shaped. The so-called plasma cells are also a form of polyblasts. Plasma cells are characteristic of chronic infections and granulation tissue; they can be recognized easily by their large protoplasmic bodies and round nuclei located in an eccentric position in the periphery of the protoplasm. The nucleus of the plasma cell shows a peculiar and characteristic arrangement of the chromatin, the granules being arranged all around the periphery of the nucleus; thus the nucleus of a plasma cell presents a design similar to a wheel with spokes ("cartwheel nucleus," Fig. 110).

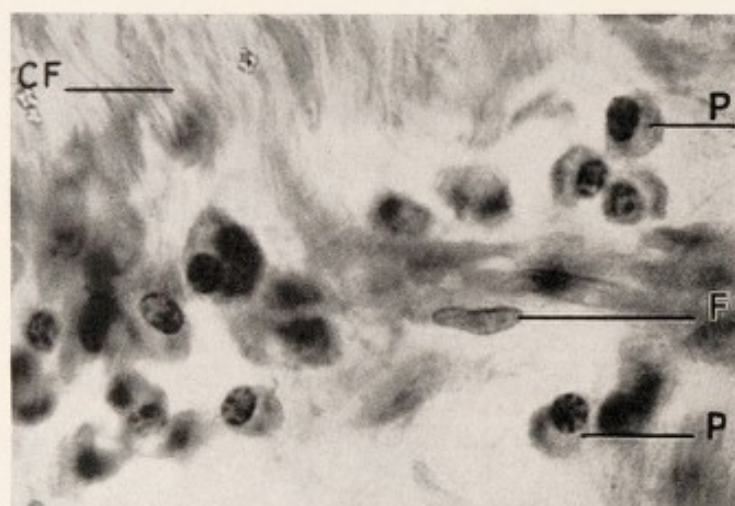


FIG. 110.—Typical cell forms in case of chronic inflammation of the periapical tissue. P, plasma cells (polyblasts) with "cartwheel" nuclei; F, fibroblast; CF, connective tissue fibrillae.

Polyblasts, which are storage and phagocytic cells, have the property of amœboid movement, and migrate actively through the tissues. If they encounter bacteria or remnants of tissue decomposition, such as dead cells and fat granules, they engulf these remnants and store and digest them in their bodies. A typical example of phagocytosis is shown in Fig. 111, which was taken of an area of granulation tissue in the oral cavity. A minute hemorrhage had occurred in this tissue some time previous to the removal of the specimen. Two large oblong cells are found in the connective tissue slightly distant from the center of the hemorrhage; their protoplasmic bodies are full of bright yellowish-brown granules whose color and form indicate that they consist of hemosiderin (blood pigment) which was stored within the cells. The nuclei of these cells appear

only faintly among the yellowish-brown masses. From the form of the cells and their nuclei, it is probable that they originally were polyblasts of the plasma cell type.

If an abscess cavity is present, as is frequently the case in dental granulomas, polyblasts from the surrounding granulation tissue are constantly discharged into it (see Fig. 120). The pus phagocytes in an old abscess are derived partly from the monocytes and lymphocytes of the capillaries and partly from the histiocytes in the surrounding fibrous tissue capsule.

If the chronic inflammation be terminated by resolution and healing, the polyblasts may be transformed back into resting wandering cells (histiocytes) and finally into fibroblasts, thus taking part in the formation of scar tissue.

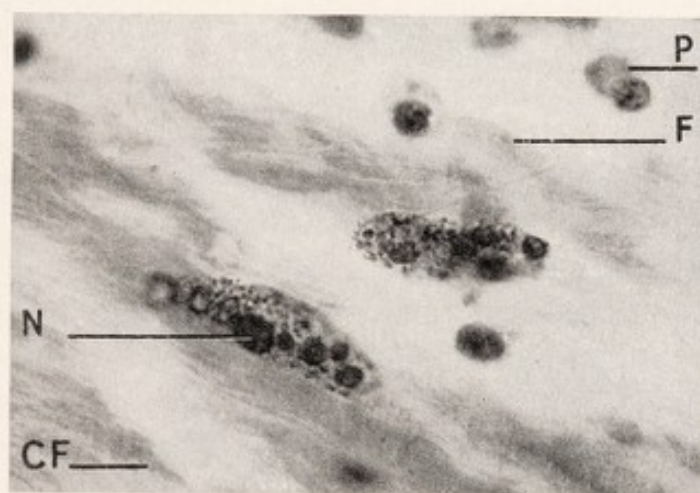


FIG. 111.—Phagocytosis in granulation tissue in the oral cavity. Two large phagocytic polyblasts, the cytoplasm of which contains a large number of granules of blood pigment from a nearby small hemorrhage. N, nucleus of the polyblast; F, fibroblast; P, polyblast (plasma cell); CF, connective tissue fibrillæ.

4. **Giant Cells.**—Giant cells are sometimes found in granulation tissue, although they are not an essential component of it. In an epulis or in tuberculous granulation tissue, for instance, giant cells are abundant; in granulation tissue around infected root ends, they usually are found lining the surface of the surrounding bone as osteoclasts but are not found within the granulation tissue itself. Giant cells are derived most probably from connective tissue and osteogenic cells by continued reproduction of the nuclei without subsequent division of the protoplasm, or they develop by a fusion of polyblasts; thus very large cells result with one protoplasmic body and many nuclei. The function of giant cells is the elimination of material that is rather resistant to dissolution, such as elastic fibers, blood pigment and calcified tissue.

CHANGES IN GRANULATION TISSUE IN CASE OF INCREASE OR DECREASE OF A
CHRONIC INFLAMMATION.

Elements of Typical Granulation Tissue: Fibroblasts, Connective Tissue Fibrillæ, Capillaries, Polyblasts (Lymphocytes, Plasma Cells, Various Phagocytes); occasionally Leukocytes, Giant Cells.

A ↓	B ↓
Acute exacerbation of inflammation:	Decrease of inflammation:
Large increase in number of polymorphonuclear leukocytes.	Reduction in number of polyblasts.
Destruction of connective tissue elements (histiolysis).	Large increase in number of fibroblasts; formation of connective tissue fibrillæ.
Discharge of cell débris, exudate, and leukocytes as pus.	
Termination in abscess formation.	Termination in scar tissue formation.

This table gives a schematic outline of the changes that granulation tissue may undergo under various conditions. Two practical examples may illustrate conditions *A* and *B* of the table.

A, acute exacerbation of a chronic inflammation: An acute dento-alveolar abscess develops from a chronic inflammatory condition (granuloma) with all the clinical symptoms of an acute purulent inflammation. The connective tissue elements in the granulation tissue break down, and an abscess cavity is formed filled with pus (leukocytes, bacteria and cell débris) (Fig. 131).

B, decrease of inflammation: A tooth with an infected root canal and chronic periapical inflammation (granuloma) is extracted; the granulation tissue remains in the socket. Within the next few days following the removal of the source of irritation and infection the fibrous elements in the granulation tissue proliferate, the polyblasts disappear, and a connective tissue scar develops. Subsequently bone is formed in the fibrous tissue of the scar. (Fig. 119.)

The conditions described under *A* and *B* are sometimes found simultaneously in one granuloma. In the periphery farthest from the tooth, scar tissue may be present, while toward the center there will be an increase in inflammatory cells (polyblasts); at the very apex the tissue will be breaking down and pus will be forming. Thus, the tissue is prepared for all clinical possibilities. If the inflammation increases, the small area of pus formation around the apex will spread, and a large abscess cavity will finally occupy the place of the former granulation tissue; on the other hand, if the infection in the root canal is controlled, the proliferation of connective tissue elements and decrease of inflammatory cells will advance from the periphery toward the center until the entire granulation tissue is transformed into scar tissue and ultimately into bone.

HISTOPATHOLOGY OF THE VARIOUS MANIFESTATIONS OF CHRONIC PERIAPICAL INFLAMMATION.

1. **Granuloma.**—The simplest form of chronic periapical inflammation consists of granulation tissue replacing the periodontal membrane and the alveolar bone around the root end. This is called apical granuloma. An apical granuloma may vary from the size of a pin-head to that of a large pea or even larger, depending upon the length of its duration and upon the intensity of the inflammation.

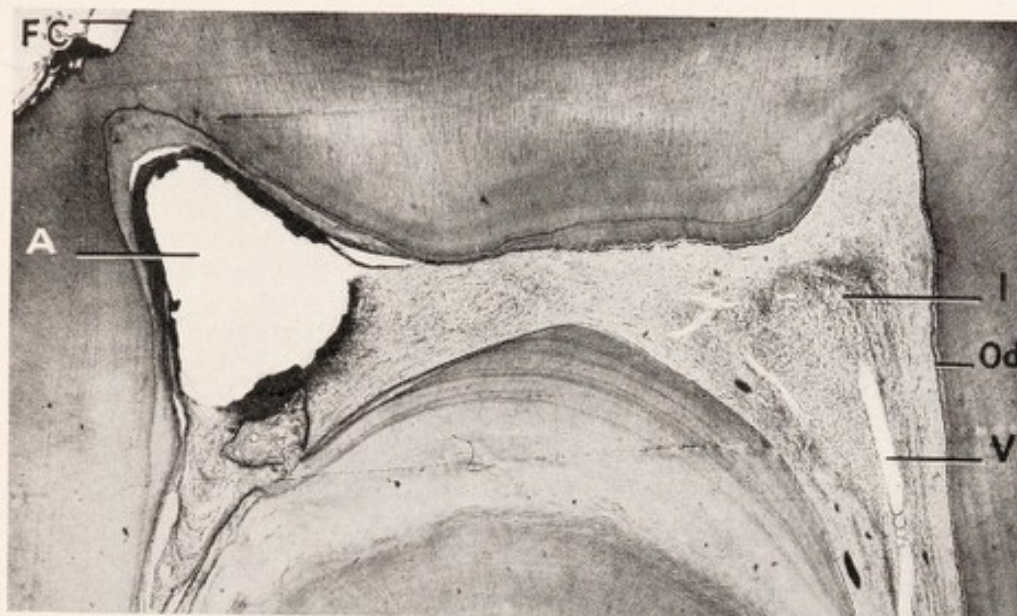


FIG. 112.—Chronic pulpitis with formation of a pulp abscess under a deep cavity in a lower second molar. FC, floor of cavity; A, pulp abscess; I, diffuse inflammatory infiltration of the entire pulp; Od, odontoblasts on the distal wall of the pulp chamber; V, enlarged bloodvessels in the distal root canal.

In a study of chronic periapical inflammation, it is best to commence with the earliest inflammatory changes, which usually take place when the pulp canal still contains some vital pulp tissue in a state of extensive pulpitis. An example may illustrate this point. In the microscopic examination of a lower human molar that had been removed under the diagnosis of total pulpitis, the pulp reveals severe inflammatory changes (Fig. 112). In the mesial pulp horn, a large abscess has developed directly under the floor of a deep cavity; this abscess is separated from the surrounding pulp tissue by a distinct line of demarcation which, as well as an area of calcification around the abscess cavity, indicates a chronic inflammation of long standing. In the distal portion of the pulp chamber, the pulp tissue shows diffuse infiltration and enlargement of the blood-

vessels. On the distal wall of the chamber, odontoblasts are still present. The microscopic diagnosis of this condition would be chronic pulpitis with large pulp abscess. The practical question arises, are the periapical tissues already involved? Clinically, it is known that frequently such teeth are sensitive to percussion, which indicates participation of the periodontal membrane in the inflammatory process. The microscopic examination of the root ends of this molar reveals the presence of a circumscribed area of round-cell infiltration in the periodontal membrane next to the apical foramen (Fig. 113). The inflammatory cells are polyblasts, mostly plasma cells, indicating an inflammation of long standing.

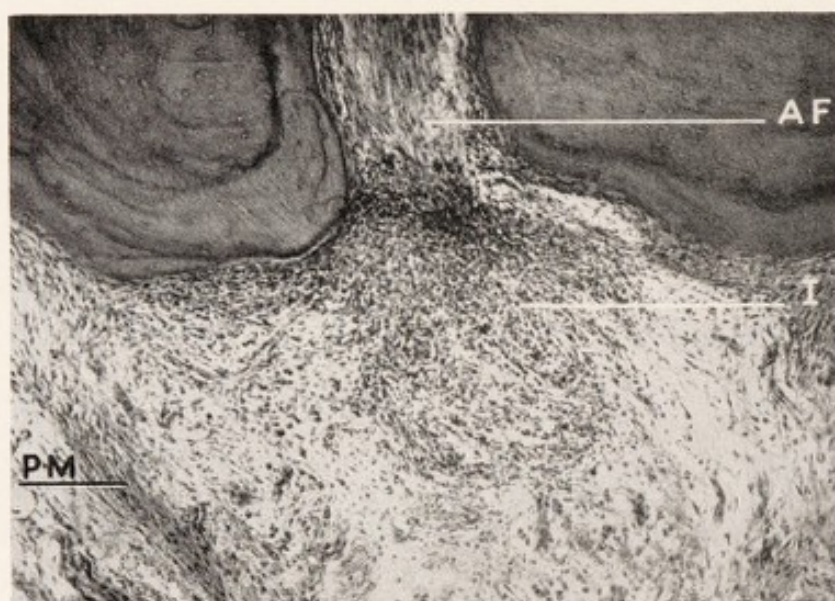


FIG. 113.—Mesial root end of the tooth shown in Fig. 112. Dense accumulation of inflammatory exudate cells (polyblasts) at the apical foramen. AF, apical foramen; I, inflammatory round cells; PM, fibrous connective tissue of the periodontal membrane.

The size of the area at the root end is certainly below clinical visibility; the entire condition is something like a microscopic periodontitis but it is a typical periodontitis and can be considered the first step in the gradual development of a more extensive periapical inflammation. There can be no doubt that, as the inflammation and destruction of the pulp advance rootward, the periapical inflammation will increase; it will gradually extend beyond the periodontal membrane and involve the alveolar bone. This specimen is additional proof for the statement made on page 122 that there is no distinct dividing line between pulpitis and periodontitis, neither clinically nor microscopically.

A few typical cases of apical granuloma of the kind most fre-

quently found in clinical examinations follow. These specimens were obtained by autopsy from human jaws. Since the radiograph is most commonly used in dental practice to diagnose chronic periapical inflammation, a radiograph of the actual jaw will accompany the microscopic picture of the specimen.

(a) *Solid Granuloma*.—Fig. 114 illustrates a mesio-distal section through the root end of an upper lateral incisor. The radiograph

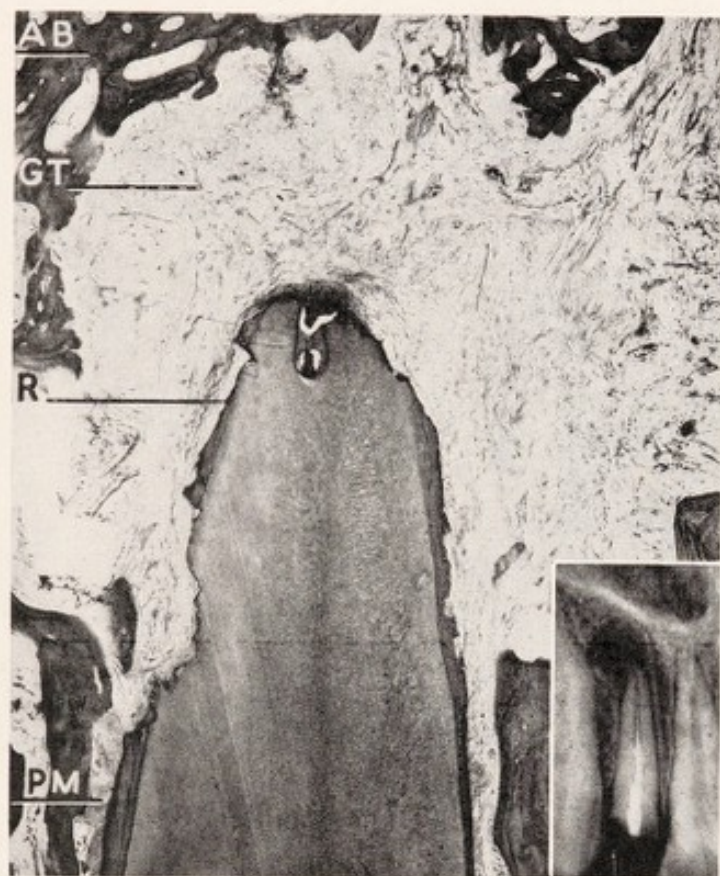


FIG. 114.—Solid granuloma. Mesio-distal section through the root end of an upper lateral incisor with decomposed pulp and imperfect root canal filling. The radiograph of the specimen taken before decalcification is inserted in the lower right corner. Due to a slight apical curvature of the root, the root canal is visible only at the apical foramen. AB, alveolar bone; GT, granulation tissue occupying the space between alveolar bone and root; R, resorption of the root surface; PM, normal periodontal membrane.

shows a poor root canal filling. Around the apex a sharply outlined area of bone destruction extends about 2 mm. crownward from the root tip so that the root end projects freely into the radiolucent area. The rest of the periodontal space around the root appears intact. The space around the root end is occupied by granulation tissue; at the apical foramen a small accumulation of inflammatory exudate cells is present, indicating that the center of irritation and inflammation is located at this very point. That part of the root

surface located within the granuloma shows rather extensive resorption of both dentin and cementum, which is a rather common occurrence in periapical inflammatory processes of long standing (see also Figs. 199-201).

In Fig. 115 a mesio-distal section through a lower first molar with decomposed pulp and empty root canals is reproduced. The radiograph shows a sharply outlined radiolucent area around the mesial root end and a similar smaller and more indefinite area at the apex of the distal root. The root canals appear empty. The



FIG. 115.—Mesio-distal section through a lower first molar with broken-down crown and decomposed pulp. The radiograph of the specimen, taken before decalcification, is inserted in the lower right hand corner. It shows a considerable amount of bone destruction around the distal root end and a small amount of bone destruction around the mesial root end. This radiographic diagnosis is verified by the section which shows a corresponding amount of bone destruction and granulation tissue (GT) at both root ends. PM, normal periodontal membrane in the upper portion of the root.

histological section fully corroborates the radiographic findings. Around both root ends the otherwise regular and normal periodontal space has been widened considerably, the space between root surface and bone being occupied by granulation tissue. In the periphery of the granuloma the tissue is mostly fibrous; further toward the apex are found an increasing number of polyblasts which reach their densest accumulation in the tissue directly opposite the apical foramen (Fig. 116).

Fig. 117 shows a mesio-distal section through the gangrenous roots of an upper first and second bicuspid. In the radiograph large,



FIG. 116.—Higher magnification of the distal root end in Fig. 115. AF, apical foramen; RI, round cell infiltration (polyblasts) near the foramen; CT, fibrous connective tissue of the periphery of the granuloma; AB, alveolar bone in an aplastic stage, showing neither resorption nor new formation.

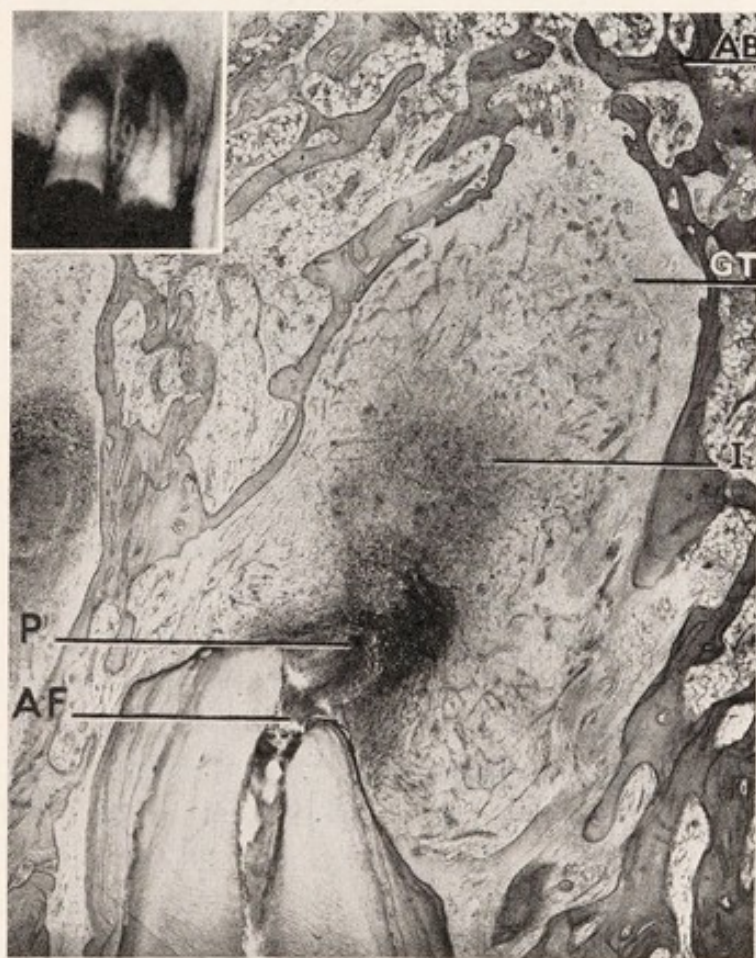


FIG. 117.—Mesio-distal section through the apex of an upper first bicuspid with granuloma. The radiograph of the specimen is inserted in the upper left corner. It shows large areas of bone destruction around the root ends of both upper bicuspids. AF, apical foramen; P, breaking down of tissue and formation of pus at the foramen; I, dense cellular infiltration next to the foramen; GT, granulation tissue; AB, alveolar bone.

diffuse areas of bone destruction can be seen over each root end; in the histological specimen this area is occupied by granulation tissue. In the periphery next to the bone, the soft tissue consists principally of fibroblasts and connective tissue fibers, few inflammatory round cells (polyblasts) being present. The fibrous connective tissue is arranged in the form of a capsule around the entire pathological area, evidently walling off the latter from its surroundings. Toward the center of the process the polyblasts increase in number. Opposite the foramen, the granulation tissue is densely infiltrated and is breaking down, discharging tissue débris and pus into the empty root canal.



FIG. 118.—Mesio-distal section through a lower second and third molar. The third molar has an intact crown and an intact, vital pulp. P, open untreated pulp canals of the second molar; CT, fibrous connective tissue occupying the large area of bone destruction at the root end of the lower second molar. Notice the destruction of bone along the mesial side of the mesial root of the third molar.

A slightly different kind of tissue reaction to chronic periapical infection is seen in Fig. 118, a mesio-distal section through a lower second and third molar. The crown and pulp of the third molar are intact; the second molar carried a large metal filling extending into the pulp chamber; the root canals are empty. The alveolar bone has almost entirely disappeared around the distal root and partly around the mesial root of the second molar; in the bifurcation the process of bone resorption left only the crest of the interdicular septum behind. At the mesial side of the mesial root of the intact third molar the alveolar bone has been destroyed and the space formerly occupied by the bone is now occupied by fibrous

tissue. A higher magnification of the soft tissue occupying the entire area of bone destruction shows dense bundles of fibrous connective tissue of the typical appearance of scar tissue (Fig. 119). The few exudate cells present in this tissue are distributed regularly but with a slight increase in number near the root end. As a whole, the character of the soft tissue gives the impression of a low-grade chronic inflammation with a marked tendency toward formation of scar tissue. In accordance with this finding is the presence of tender trabeculae of newly built bone at the walls of the soft tissue area. The microscopic appearance of the bone surrounding such areas of inflammatory soft tissue enables us to draw conclusions as to the character of the process at the root end. As long as the inflam-



FIG. 119.—Higher magnification of Fig. 118. CT, irregular fibrous connective tissue (scar tissue) without evidence of inflammation; AB, newly formed trabeculae of alveolar bone in the periphery of the scar tissue.

mation is still active, which means that it still has a tendency to spread and involve additional areas of bone, marked symptoms of osteoclastic activity will be observed on the bone lining the cavity. If the process is at a standstill, clinically manifested by slight or no changes in the radiographic appearance over a long period of time, the bone around the involved area will be found in an aplastic state: neither resorption nor new formation of any significance is visible under the microscope. If there is a healing tendency, indicated radiographically by a decrease in the size of the area of bone destruction, new formation of bone will be encountered on the walls of the bone cavity.

The preceding specimens of granulomas have the following characteristics in common:

1. A distinct area of bone destruction around the apex is visible in the radiograph.

2. This area, upon microscopic examination, is found to be occupied by granulation tissue.

3. The granulation tissue presents various conditions corresponding to various degrees of inflammation, namely, the formation of fibrous scar tissue in the periphery, an increasing number of polyblasts toward the root end, and the breaking down of tissue and pus formation at the apical foramen.

4. Either the bone surrounding the granulation tissue is in a stage of inactivity or there is evidence of new formation of bone, indicating that the inflammatory process is of a mild and chronic character and has no active tendency to spread or to involve additional bone.

All these qualities characterize the chronic form of periapical inflammation known as granuloma.

The solid granulomas just described may show clinically as well as microscopically certain variations or conditions that represent further stages in the development of the pathological process. Such conditions are the breaking down of tissue and the formation of cavities in the center of the granuloma, and communication between the granuloma and the outside of the jaw by way of a sinus.

(b) *Granuloma With Central Abscess Cavity*.—Through disintegration of tissue in the center of the granuloma, abscess cavities are formed, the sizes of which vary from one to several millimeters in diameter. These cavities may be distinguished from dental cysts by the absence of an epithelial lining; the wall of the cavity is formed by granulation tissue rich in exudate cells. The inflammatory lining is called a pyogenic membrane. Cells and cell débris are discharged from the walls into the cavity, forming pus.

Fig. 120 illustrates a mesio-distal section through an upper first and second bicuspid. The radiograph shows only a diffuse and indistinct area of bone destruction around the root ends of these two teeth. The microscopic examination reveals that only the first bicuspid has a decomposed pulp; the pulp of the second bicuspid is intact and vital. The root end of the first bicuspid rises into an abscess cavity about 3 mm. in diameter which is partly filled by pus. The lining of this cavity consists of granulation tissue; farther out toward the periphery of the cavity the character of the tissue approaches that of fibrous scar tissue, until next to the bone a plain connective tissue capsule is found. The surrounding bone shows in some places evidence of resorption.

(c) *Granuloma Connected With the Surface of the Alveolar Process by a Sinus.*—Frequently the inflammation does not stay within the limits of the jaw bone, but finds its way through the bone and through the overlying mucoperiosteal lining to the surface of the jaw. The perforation is connected with the granuloma inside of the jaw by a duct lined with granulation tissue, clinically known as a sinus, through which a slight discharge of pus or serum takes place.



FIG. 120.—Granuloma with central abscess cavity. Mesio-distal section through an upper first and second bicuspid. The second bicuspid is intact and vital; the first bicuspid has a decomposed pulp and an infected root canal. A granuloma with central abscess cavity has developed at the root end of this tooth. AB, alveolar bone; CT, connective tissue capsule; P, purulent exudate in the abscess cavity; PM, normal periodontal membrane in the lower portion of the root.

The development of a sinus can follow the perforation of an acute dento-alveolar process. After the acute symptoms have subsided, the perforation fails to heal, and a small, fistulous opening surrounded by granulation tissue remains, from which a yellowish exudate is discharged. In other cases the sinus appears without any preceding acute symptoms, indicating that a chronic inflammatory process around the root end of a pulpless infected tooth has gradually reached and perforated the overlying tissues.

The location of the opening of a sinus depends upon the topog-

raphy of the infected root end and its surrounding tissues (see page 138). Most sinuses open into the labial or buccal vestibulum. Occasionally they are found on the lingual surface of the upper and lower jaw, in the floor of the nose, and in the maxillary sinus. The inflammation may also perforate through the skin, and a sinus will then appear on the face. The latter condition is frequently observed with fistulas originating in upper cuspids (skin fistula in the region of the inner angle of the eye) and in lower incisors (chin fistula). Children's lower first molars with decomposed,

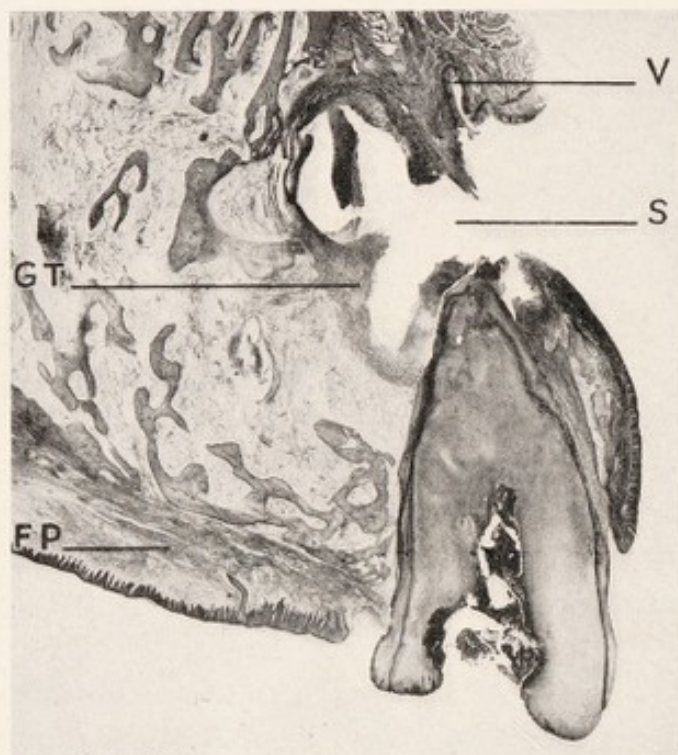


FIG. 121.—Chronic periapical inflammation with formation of a sinus. Bucco-lingual section through a buccal root of a broken-down upper first molar. At the root end an abscess cavity has formed from which a sinus leads into the buccal vestibulum. V, vestibulum (point of reflection of the mucosa of the cheek); S, sinus; GT, granulation tissue forming the wall of the abscess cavity; FP, fibrous tissue of the hard palate.

infected pulps have a tendency to form fistulas that perforate the skin at the lower margin of the mandible and cause depressed, funnel-shaped scars in this region.

Fig. 121 illustrates a bucco-lingual section through the mesio-buccal root of an upper first molar with decomposed pulp. A sinus leads into the vestibulum oris. Inside the alveolar process there is a cavity into which the apex of the molar root projects. Pus in the lumen of the cavity indicates that during life pus was discharged from the abscess cavity into the vestibulum.

It is known from clinical experience that a sinus heals if the inflammation inside of the jaw ceases. After the extraction of a pulpless tooth or after successful treatment of a root canal, a sinus will disappear. The same thing happens if drainage for the discharge has been established through the root canal.

(d) *Significance of Epithelium in the Granuloma.*—Microscopic examinations of serial sections have shown that, in from 40 to 50 per cent of all chronic inflammatory processes of the periapical region, epithelium is found in the granulation tissue. This epithelium at the root end originates from the epithelial rests of the periodontal membrane. Epithelial rests are present in every normal periodontal membrane. They are derived from the sheath of Hertwig, the epithelial structure found at the apical end of every forming tooth or root. The epithelial rests appear as small clusters or strands of squamous epithelial cells lying between the fiber bundles of the periodontal membrane, and normally seem to remain in an aplastic or inactive condition; at least we do not find any noticeable difference between the size or number of the epithelial rests in young and in old teeth.

If chronic inflammation develops in the neighborhood of the epithelial rests, they seem to awake from their resting state. The number of epithelial cells greatly increases by mitosis; the epithelium begins to proliferate and forms epithelial strands or bands in the inflammatory tissue around the infected root end. Usually the epithelium, during its growth, comes into direct contact with the root surface; here its relationship to the dental hard substance (cementum) is identical to that found where the oral mucosa is attached to the tooth surface: an epithelial attachment develops.

Some investigators claim that the epithelium in dental granulomas is derived from the oral epithelium. While it cannot be denied that an ingrowth of oral epithelium into a granuloma is possible by way of a sinus, this form of epithelization must be considered an exception rather than a rule. The author has convinced himself, through careful examination of serial sections through granulomas *in situ*, that epithelium can be found at the apex in cases where absolutely no connection with the surface epithelium was possible. Therefore, it should again be stated that in the vast majority of cases the epithelial rests of the periodontal membrane are the source of the epithelium in dental granulomas and root cysts.

Since epithelial rests are found in every periodontal membrane, the question arises, why do we not find epithelium in every periapical inflammation? Stein, who made a study of the apical tissues

of pulpless teeth in sections through a large number of human jaws, expressed the opinion that this variability depends upon the intensity of the inflammation. In a very severe inflammation, the amount and rate of tissue destruction is so great that the epithelial rests are destroyed; consequently, after the acute inflammation has subsided, no more epithelium is left to be a source of new proliferation.

Dental root cysts develop from the epithelium in granulomas. It is impossible to draw a sharp line between epithelium-containing granulomas and radicular cysts, as in almost every case of granuloma with epithelium one or several areas may be considered as



FIG. 122.—Epithelium-containing granuloma. Section through the root end of an upper molar with decomposed pulp and infected root canal. AB, alveolar bone; CT, connective tissue; E, epithelium arranged in garlands around the apical foramen; I, inflammatory round cell infiltration between the epithelial projections; RC, root canal containing detritus and purulent exudate. (Stein, *Ztschr. f. Stom.*, courtesy of Urban & Schwarzenberg, Berlin-Wien.)

the earliest stages of cyst formation. However, we will describe separately granulomas that contain only such minute cysts, and larger cysts that are clinically well-defined cavities of considerable size.

In Fig. 122, a mesio-distal section through the root end of an upper molar with a decomposed pulp and empty root canal is illustrated. The apical foramen is located on the distal side of the root end. The bone opposite the foramen has been destroyed and replaced by granulation tissue; around the opening of the root canal, epithelium has proliferated into the granulation tissue and is attached to the root surface above and below the foramen. The

epithelium in this section appears to be arranged in garlands, surrounding or including areas of connective tissue with round cell infiltration.

Fig. 123 shows a section through the root end of an upper bicuspid with decomposed pulp. Extensive epithelial proliferations are present at the root end. The epithelium is attached to the root surface and is surrounded by granulation tissue showing a low-grade inflammation. The squamous epithelium has formed a typical hornified cuticle on the surface of the root, which reaction is identical to the formation of hornified (secondary) cuticle on the tooth

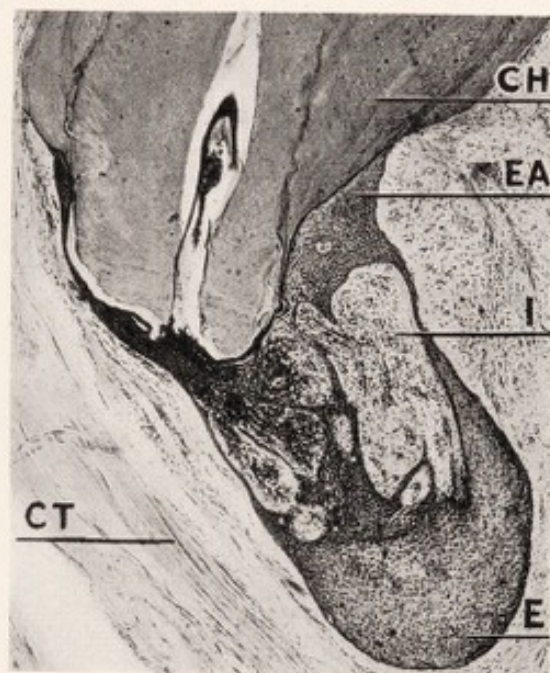


FIG. 123.—Root end of an upper bicuspid with decomposed pulp. Solid mass of epithelium present in the apical granuloma. E, epithelial proliferations at and near the root end; EA, epithelial attachment to the root surface; CH, cementum hyperplasia above the epithelial attachment; I, round cell infiltration in the connective tissue between the epithelial strands; CT, connective tissue capsule. (Gottlieb, Jour. Am. Dent. Assn.)

surface underlying the epithelial attachment. Above the end of the epithelial attachment a cementum hyperplasia has developed (see also Fig. 186).

2. Radicular Cysts (Root Cysts, Dental Cysts).—The squamous epithelium at the root ends of pulpless teeth has a marked tendency toward the formation of cystic cavities. Cysts are pathological cavities lined with epithelium, and when they are found at the root ends of pulpless teeth, we speak of radicular cysts, root cysts, or dental cysts. Thus a dental cyst may be defined as a pathological cavity of inflammatory origin in the alveolar bone, lined by epithelium derived from the epithelial rests.

Radicular cysts can develop in two ways. An abscess cavity may be formed by the inflammatory breaking down of the tissue around the infected apex of the root, following which the squamous epithelium, because of its inherent tendency to cover and line raw surfaces, covers the wall of this cavity. The other possibility is that cystic degeneration takes place within the epithelial strands themselves, and the epithelial masses are hollowed out, enlarged, and transformed into cysts.

The earliest stage of cyst formation, the growth of a central cavity inside of a solid strand of epithelium, is illustrated in Fig. 124. Here a small cystic cavity is forming by the breaking down of the



FIG. 124.—Earliest stage of cyst formation. Cystic degeneration in epithelial masses near the root end of an upper incisor with infected root canal. Cy, cystic degeneration within an epithelial mass; E, small solid mass of epithelial cells; I, round cell infiltration; RE, root end.

epithelial cells in the center of the strand and by subsequent accumulation of fluid.

Fig. 125 shows a mesio-distal section through the bucco-distal root of an upper second molar with decomposed pulp. A granuloma is present at the apex, in the center of which a cystic cavity lined by epithelium (dental cyst) has developed. The inflammation is considerable as indicated by the presence of a large number of polyblasts in the granulation tissue and by an accumulation of free cells and cell debris (pus) around the apex. The epithelium of the cyst cavity is attached to the entire circumference of the root with a typical epithelial attachment. The pus at the apical foramen is the result of a discharge of inflammatory cells, leukocytes

and polyblasts, from the granulation tissue into the cyst cavity. Osteoclasts on the surrounding bone indicate that the cyst has an active tendency to grow.

In large radicular cysts the inflammation is sometimes of a very low grade, the fibrous tissue surrounding the cysts showing few inflammatory changes; the cyst wall is smooth and lined by a thin, even layer of stratified squamous epithelium. The content of the cyst gradually changes from a purulent exudate to a clear, yellowish, serous fluid. This fluid sometimes contains a large number of long, thin, spear-shaped crystals of cholesterol. Cholesterol is normally found in small quantities in the bile and in the



FIG. 125.—Small radicular cyst formed by epithelization of the wall of an abscess cavity at the root end. Upper molar. E, epithelial lining of the cyst cavity; P, purulent exudate within the cyst next to the apical foramen; EA, epithelial attachment of the cyst epithelium to the root surface; CT, connective tissue; AB, alveolar bone; R, resorption of the alveolar bone. The presence of bone resorption indicates that the cyst was growing at the time the individual died.

blood. Under pathological conditions it is abundant in some types of gall stones (cholesterol stones), and it may also be found anywhere in the body where tissue decomposition, especially decomposition of epithelial tissue takes place. Pure cholesterol is a solid, white substance that crystallizes in the form of long, thin needles, it is soft and has a silky, oily feeling. Chemically it is a monatomic alcohol derived from a complicated hydrocarbon.

Cholesterol crystals are frequently observed suspended in the fluid of dental cysts, appearing as fine, glittering flakes. They seem to be the end-product of the continuous desquamation and disintegration of epithelial tissue from the cyst wall. If specimens of

cystic periapical processes are treated with alcohol or ether during the preparation of microscopic slides, the cholesterol itself is dissolved away; however, in the final specimens the spaces originally occupied by the needle-shaped crystals can be recognized easily (cholesterol slits).

Fig. 126 is a section through the root of an extracted upper molar with decomposed pulp. A cyst of about 8 mm. in diameter came along with the root in extraction. The inside of the cyst is filled with a mass of coagulated fibrin that shows a large number of fine, streak-like spaces. In a higher magnification (Fig. 127) these spaces



FIG. 126.—Root end of an extracted upper molar with root cyst attached to it.
Ch, mass of cholesterol and fibrin in the cyst.

appear as impressions of the needle-shaped cholesterol crystals that were originally enclosed in the mass of fibrin. When the specimen was treated with alcohol and ether, the cholesterol was dissolved so that only the outline of the crystals is now visible. The wall of the cyst is lined by several layers of stratified squamous epithelium; the surrounding fibrous connective tissue shows only very little infiltration, indicating a chronic, low-grade inflammation.

Radicular cysts in the jaw occasionally become very large, and may cause a slowly growing prominence on the outer surface of the bone. This prominence is at first covered by a thin plate of bone, which later disappears as a result of the continued pressure of the

cyst. When the bone has disappeared, the cyst wall lies directly beneath the oral mucosa and can be felt by the palpating finger.

A large dental cyst of this type is illustrated in Fig. 128. The patient, a boy aged eight years, had a slowly growing, painless swelling on the right side of the maxilla. The right deciduous central and lateral incisors were still in place; on the left side the permanent central and lateral incisors had already erupted. Both right deciduous incisors appeared clinically intact; the deciduous central incisor, however, was markedly discolored, which, in connection with the history of a trauma (blow), suggested death of the pulp with subsequent periapical infection. The radiographic examination revealed the presence of a large cavity in the bone involving the entire anterior portion of the right maxilla. In the upper part of this cavity the displaced crowns of the permanent



FIG. 127.—High magnification of Fig. 126. Cholesterol slits (CS) in a radicular cyst. The cholesterol forms needle or spear-shaped crystals which leave slits of the same shape after the cholesterol has been dissolved in the preparation of the specimen.

incisors are visible. The root end of the deciduous central incisor is markedly resorbed. These radiographic findings lead to the diagnosis: traumatic death of the pulp followed by infection and inflammation of the periapical tissue and resorption of the root; development of a dental cyst with displacement of the crowns of the permanent teeth by the pressure of the growing cyst sac.

This diagnosis was verified by the operation, which consisted of the removal of the central and lateral deciduous incisors together with the surrounding soft tissues and part of the cyst sac. In the months following the operation the misplaced permanent incisors showed a very marked eruptive movement, and are expected finally to assume their normal place in the jaw.

The specimen removed during the operation was sectioned in labio-lingual direction. Fig. 128 shows the central incisor with its

intact crown and necrotic, decomposed pulp. The upper half of the root has been extensively resorbed; the root end extends into a large cystic cavity, which, through the apical foramen, communicates with the contents of the pulp canal. The wall of the

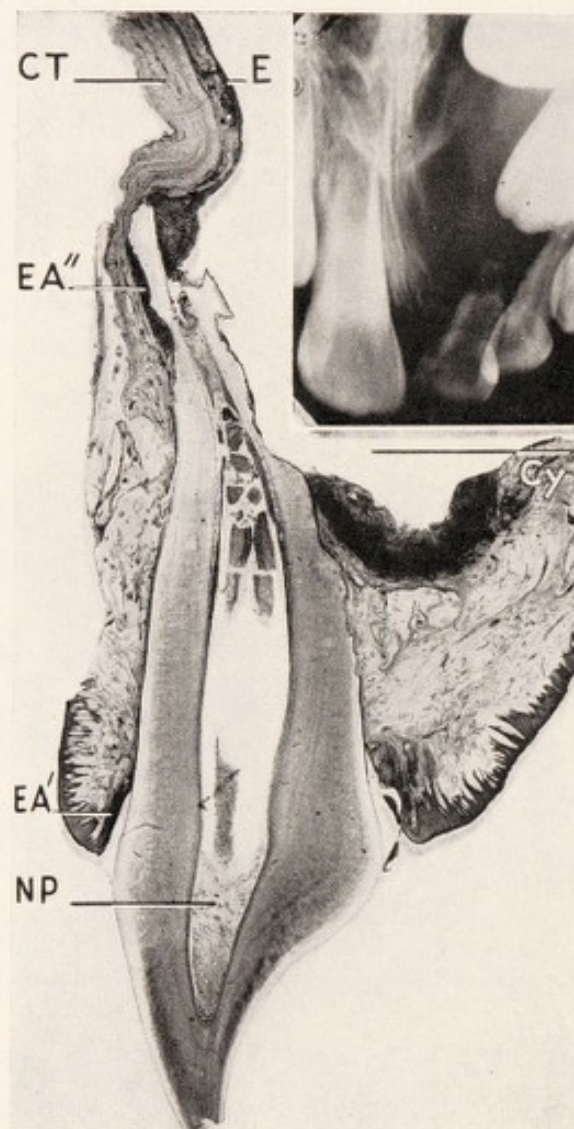


FIG. 128.—Large radicular cyst originating from a deciduous right upper central incisor following traumatic necrosis of the pulp. The radiograph shows the resorbed root end of the deciduous incisor projecting freely into the cyst cavity. This observation is substantiated by the microscopic finding of extensive resorption of the root end. NP, necrotic pulp tissue; Cy, cyst cavity; E, epithelial lining of the cyst; CT, connective tissue capsule of the cyst; EA', epithelial attachment of the oral epithelium to the enamel; EA'', epithelial attachment of the cyst epithelium to the resorbed root surface.

cyst consists of a dense, fibrous connective tissue capsule with an inner lining of stratified squamous epithelium. It is interesting to notice that the tooth shown in Fig. 128 has two epithelial attachments: one in the usual place, where the gingival epithelium is attached to the tooth surface, and the other where the cyst epi-

thelium is attached to the circumference of the root. Under both epithelial attachments a hornified (secondary) cuticle can be found (Fig. 129).

The periodontal membrane surrounds that portion of the root that lies between these two epithelial attachments; the firmness of the tooth depends largely upon the extent of root surface remaining.

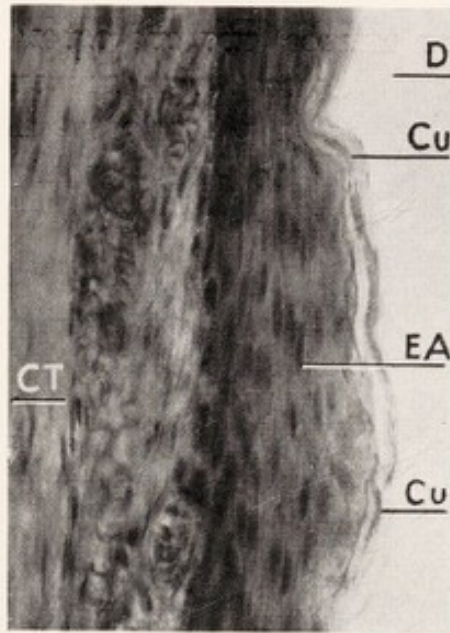


FIG. 129.—Higher magnification of EA" in Fig. 128. D, dentin; Cu, hornified secondary cuticle on the resorbed dentin surface; EA, stratified squamous epithelial cells of the epithelial attachment; CT, connective tissue.

3. Acute Exacerbation of Chronic Periapical Inflammation.—It has been mentioned in the discussion of acute periapical inflammation that the majority of acute dento-alveolar abscesses develops from chronic inflammations such as granulomas or cysts. The reasons for such an acute exacerbation may be manifold. Sometimes a chronic condition may turn into an acute abscess apparently for no reason, unless perhaps because the virulence of the bacteria increases or the general resistance of the organism decreases to a point where the infection breaks down the wall of resistance and defense that has been built up around the chronic process. Frequently, however, some outer interference is responsible, especially a change in drainage conditions. For instance, if a chronic inflammatory process steadily discharges a small amount of fluid through an open root canal into the oral cavity, usually no clinical symptoms will be present. If, however, the root canal is obstructed by coagulated fibrin or debris, the retention of fluid (pus) within the bone may cause an acute exacerbation with pain, swelling and fever, a sudden

increase in the number of leukocytes, and an acute abscess formation. In the same way acute exacerbations following root canal therapy on pulpless teeth may be accounted for. The manipulations in the root canal and at the apex disturb the equilibrium between the bacteria in the periapical process and the defense mechanism of the organism; the infection spreads and causes an acute abscess.

Fig. 130 shows a mesio-distal section through the posterior part of the upper jaw. The crowns of the upper second and third molars



FIG. 130.—Exacerbation of chronic periapical inflammation: formation of an acute abscess. Mesio-distal section through the posterior portion of the maxilla. Second and third molar with decomposed infected pulps. The radiograph in the lower left corner shows the carious destruction of the crowns and the extensive periapical bone destruction. A, abscess cavity extending over the lingual roots of both molars; MS, floor of the maxillary sinus; Hy, hypertrophic gum tissue located in the cavity on the distal side of the second molar (gum polypus).

are broken down and the pulps are decomposed. An acute alveolar abscess around the roots of the teeth has developed from a chronic inflammation. A cavity of irregular outline and of more than 1.5 cm. in diameter is present around the root ends of the upper molars. The abscess cavity is filled with large masses of pus, and the walls are densely infiltrated with inflammatory round cells, which extend into the marrow spaces of the surrounding bone. The presence of epithelium in the granulation tissue around the abscess cavity and

the arrangement of fibrous tissue in the form of a capsule indicate that the inflammation must have been of long standing and that only shortly before the death of this individual an acute exacerbation had developed. A higher magnification of the periphery of the abscess cavity reveals the spreading of the inflammation beyond the fibrous capsule; the neighboring bone is lined by osteoclasts indicating that active bone destruction has taken place (Fig. 131). Almost all of the inflammatory round cells in the tissue around the abscess and in the pus are polymorphonuclear leukocytes.

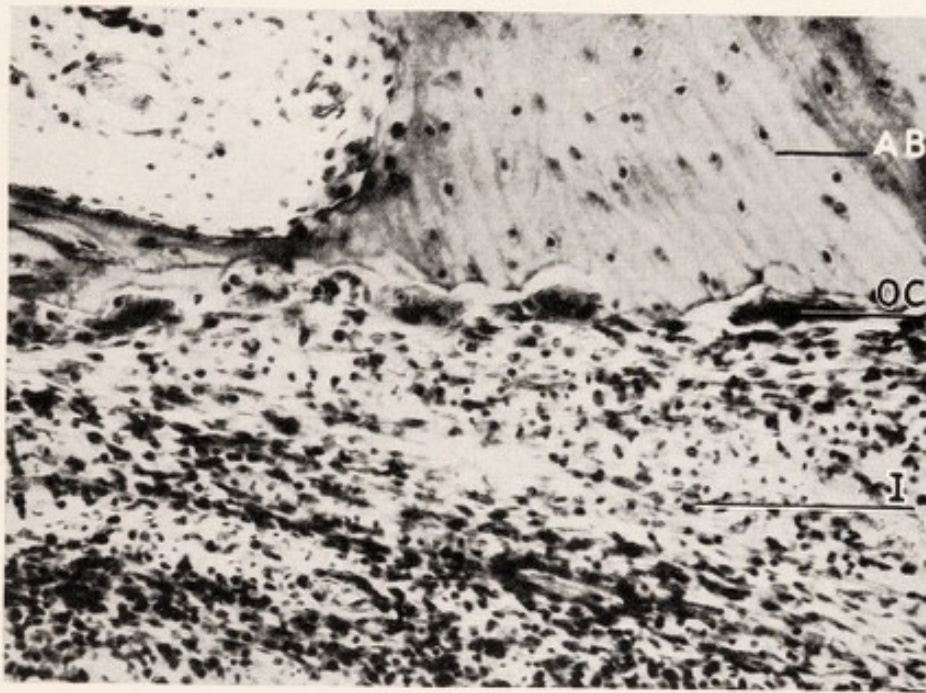


FIG. 131.—Higher magnification of the periphery of the abscess cavity in Fig. 130. AB, alveolar bone; OC, osteoclasts; I, inflammatory exudate cells, mostly polymorphonuclear leukocytes. The presence of these leukocytes and of the large number of osteoclasts indicates an acute inflammatory process with progressive bone destruction.

4. Periapical Inflammation in Its Relationship to the Maxillary Sinus.—The root ends of the upper posterior teeth are often in intimate contact with the maxillary sinus (antrum of Highmore); thus, inflammatory processes originating from these teeth may have a direct influence upon the condition of the sinus.

The maxillary sinus is a cavity lying within the body of the maxilla. It communicates with the nasal cavity through an opening, the maxillary ostium, that leads into the middle meatus. The maxillary sinus has the approximate shape of a pyramid; the base of the pyramid is formed by the lateral wall of the nasal cavity, the tip lying in the zygomatic process. The other boundaries of the maxillary sinus are: the anterior wall, which is formed by the

anterior plate of the maxilla (canine fossa); the lower wall, formed by the alveolar process; the posterior wall, formed by the maxillary tuberosity, and the upper wall, formed by the floor of the orbit. The maxillary sinus is lined by columnar ciliated epithelium with a very thin submucosa.

The size of the antrum is subject to great individual variation. In the antero-posterior dimension the sinus may extend from the cuspid region to the third molar and into the maxillary tuberosity. The other extreme is a small sinus that does not extend over more than two or three posterior teeth.

The height of the maxillary sinus also is extremely variable. A large sinus may extend far down into the alveolar process, diverticles being located between the roots of the upper molars and bicuspid, so that these roots appear as elevations rising above the floor of the sinus. In such cases, only a very thin plate of bone is found between the sinus and the root ends of the teeth. Sometimes even this thin septum of bone is missing, and the apex is covered simply by the mucous membrane of the sinus. In other cases there is a thick layer of bone between the floor of the sinus and the apices of the upper posterior teeth, this distance being 6 or 8 mm. or even more. Consequently, in the first case, a periapical inflammatory process will almost invariably involve the floor of the sinus; whereas, in the other extreme even an extensive inflammation of dental origin is not likely to extend into the antrum. A case of the latter type has been illustrated in Fig. 130, where, in the presence of an extensive inflammation in the maxilla, the sinus appeared not at all affected due to the distance between it and the diseased area.

Fig. 132 illustrates a mesio-distal section through the lingual root of an upper second molar with decomposed pulp. The root is prominent above the floor of the maxillary sinus, its apex lying about 4 mm. higher than the deepest point of the sinus. The root is surrounded by a thin plate of bone, which at the same time represents the wall of the sinus and the alveolar bone of the tooth. Around the root end a granuloma has developed; as a result the distance between the root surface and the bone is greater than the width of the normal periodontal membrane on the sides of the root. Probably the root was originally surrounded by a uniformly wide periodontal membrane and by a normal plate of bone; later, when the inflammation developed, the bone was resorbed on the inside of the alveolus, resulting in compensatory new formation of bone on the upper (outer) side of the alveolar plate. The bone plate gradually became more distant from the root surface until

the present condition resulted. The mucosa of the maxillary sinus covering the prominence caused by the presence of the root is histologically normal. If a chronic periapical inflammation like the one illustrated in Fig. 132 persists over a long period of time, it may be expected that the dividing tissues between the inflammation and the lining of the sinus will finally be destroyed and the latter will become involved. This condition is illustrated in the next case.

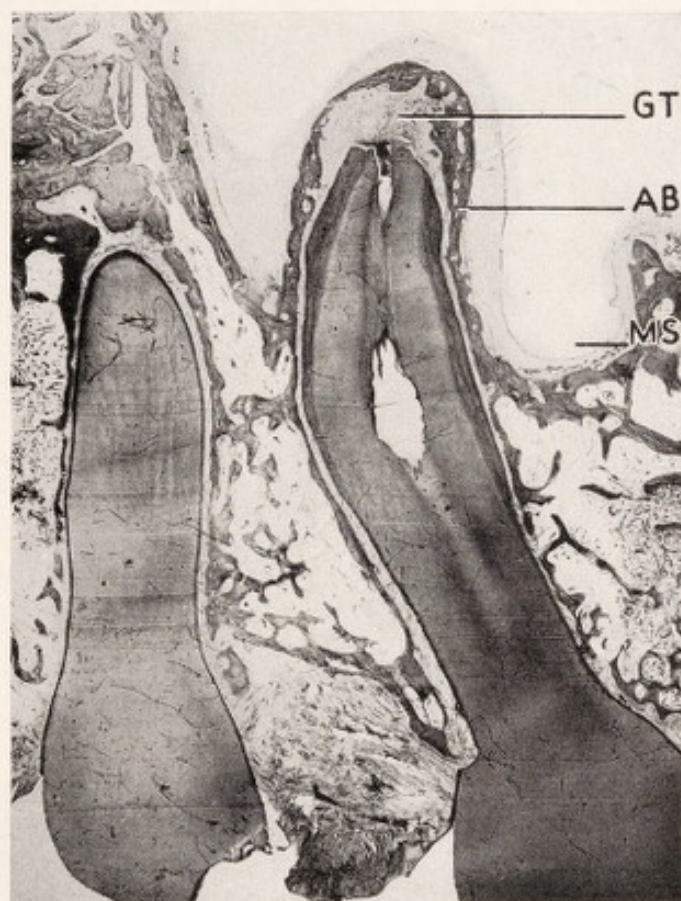


FIG. 132.—Relationship between periapical infection and maxillary sinus. Mesio-distal section through the lingual root of an upper second molar with decomposed infected pulp. The apical foramen of this root is located about 4 mm. above the deepest point of the maxillary sinus, MS. GT, granulation tissue at the root end; AB, alveolar bone prominent over the floor of the maxillary sinus. The mucosa of the maxillary sinus is intact.

Fig. 133, a mesio-distal section through the posterior part of the upper jaw, shows an upper first and second molar with broken-down crowns and decomposed pulps and a third molar with intact pulp. The maxillary sinus is large, extending over the area of the root ends of all three upper molars. At the root end of the second molar, a cyst of about 1 cm. in diameter has developed; the apex of the tooth rises freely through the floor of the cyst. At its highest point

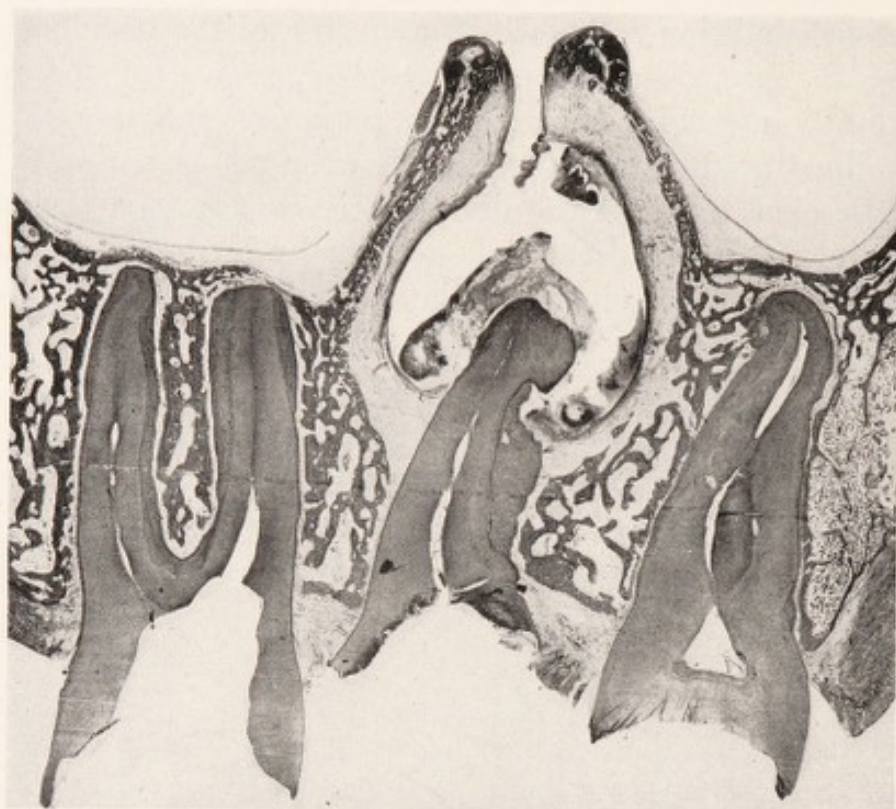


FIG. 133.—Relationship between periapical infection and maxillary sinus. Mesio-distal section through the posterior part of the maxilla with first, second and third molars. All root ends are close to the floor of the maxillary sinus. The pulp of the second molar is decomposed and infected. At the root end a cyst has developed which caused an elevation of bone over the floor of the maxillary sinus and finally perforated into the sinus at the highest point of this elevation. The lower portion of the cyst contains purulent exudate.



FIG. 134.

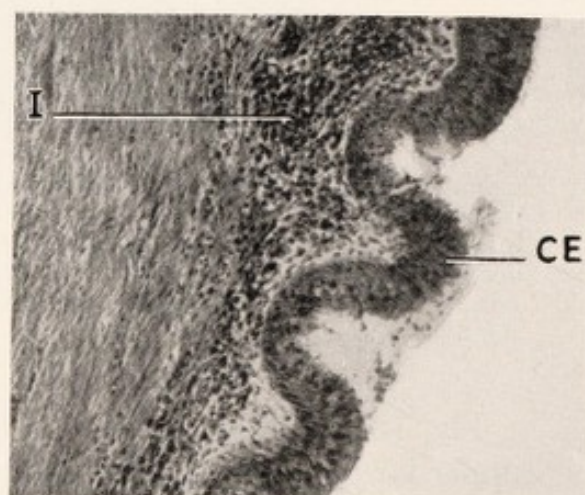


FIG. 135.

FIGS. 134 and 135.—Higher magnification of the cyst wall in Fig. 133.

FIG. 134.—Lower portion of the cyst wall. SE, stratified squamous epithelium; I, subepithelial round-cell infiltration; P, purulent exudate.

FIG. 135.—Upper portion of the cyst wall. CE, columnar epithelium originating from the maxillary sinus; I, subepithelial round-cell infiltration (polyblasts).

the cyst has perforated into the maxillary sinus, and there is an open communication between the cyst cavity and the antrum. The cyst is partly filled with desquamated epithelial cells, polyblasts and coagulated fibrin. This particular case offers several interesting points. In its lower portion, the wall of the cyst is lined by stratified squamous epithelium (Fig. 134) as is usually found in dental cysts. In its upper part, however, the epithelial lining consists of columnar epithelium (Fig. 135), as is characteristic of the accessory sinuses of the nasal cavity. Evidently the lower portion of the cyst still contains the original cyst epithelium; whereas, the upper part has become covered with epithelium originating from the adjacent maxillary sinus. The structure of the bone which forms part of the cyst wall and which rises from the floor of the sinus is decidedly different from the structure of the bone bordering the sinus over the first and third molars. This indicates that the bone around the cyst was formed only recently. The mucosa of the maxillary sinus covering the mesial prominence of the cyst shows rather extensive inflammatory changes, indicating the presence of circumscribed sinusitis. The rest of the mucosa in this area does not show any pathological changes. The extraction of the second molar would, at least temporarily, create a communication between oral cavity and maxillary sinus.

CONDENSING OSTEITIS (BONE SCLEROSIS) DUE TO CHRONIC PERIAPICAL INFLAMMATION.

In the preceding paragraph rarefying osteitis of the jaw-bone was described. But chronic inflammations about the root ends of infected teeth do not always lead to bone destruction. Sometimes a low-grade irritation of long standing, instead of destroying the bone, causes hypertrophy in a circumscribed area. This condition is called bone sclerosis.

Histologically, the sclerosed area may consist of either a mass of bone trabeculae of much denser arrangement than the surrounding bone, in which case the border between normal and abnormal bone might be rather indefinite, or of a mass of very compact bone having few marrow spaces, in which case the compact bone is usually separated from the surrounding marrow spaces and cancellous bone by a distinct line.

The radiograph shows a corresponding difference between these two types of condensing osteitis. In some cases a diffuse area of denser bone is present around a tooth with chronic infection of the

periodontal tissues, while in other cases a sharply outlined, dense mass of bone is located next to the diseased root end. Thus the radiograph offers an effective and the only method of discovering such areas in the bone.

No certain therapy is known for this condition, as it is very difficult, especially in more diffuse cases, to localize and surgically to remove the condensed area of bone. The extraction of the tooth that caused the inflammation in the bone by no means causes the area of bone condensation to disappear; in fact it usually persists. Such remaining areas of dense bone have sometimes been suspected as areas of "residual infection" in the jaws.

This question is, of course, difficult to decide, since material for both bacteriological and histological examination of such bone is hard to procure; however, it is the author's opinion that the potential danger of such areas in the jaw has been greatly overemphasized. After the removal of an infected tooth the chances of the infection's remaining are very small; the healing tendency and resistance of the organism are usually sufficient to overcome any infection that might incidentally be left behind. But it cannot be expected that an area of condensed bone that has developed in the jaw over a period of years should disappear after the irritation has subsided; usually such an area will persist and form a bony scar.

To the author's knowledge, condensing osteitis of the jaws has been demonstrated only radiographically or by bone specimens removed in surgery, but as yet has not been studied in histological sections through the condensed bone in connection with teeth and surrounding jaw. The accompanying photomicrographs were prepared in order to bring out more clearly the relation of the sclerosed bone to the surrounding tissues.

The specimen in Fig. 136 was taken from a human jaw of which neither radiographs nor other data were available. It shows a mesio-distal section through the mandible. A small root end of the lower first molar, the surface of which has been badly destroyed and hollowed out by caries, is attached superficially to the jaw bone. The bone underneath the root end is distinctly different from the bone in the rest of the jaw; the trabeculae are much denser, with only very small marrow spaces between them. In this case the chronic irritation from the retained and infected root tip must be held responsible for the condensing osteitis under the root. It is probable that this bone area would have remained in the jaw after the complete elimination of the root.

In Fig. 137 a radiograph is reproduced that was taken of the right

maxilla of a man aged thirty-eight years. The jaw was edentulous distally from the first bicuspid; the soft tissues on the ridge appeared completely healed. In the radiograph a dense area is seen in the



FIG. 136.—Bone sclerosis due to chronic low-grade inflammation around the distal root of a broken-down first molar. R, root with carious surface; SB, area of sclerosed bone below the root end; M, intact second molar.

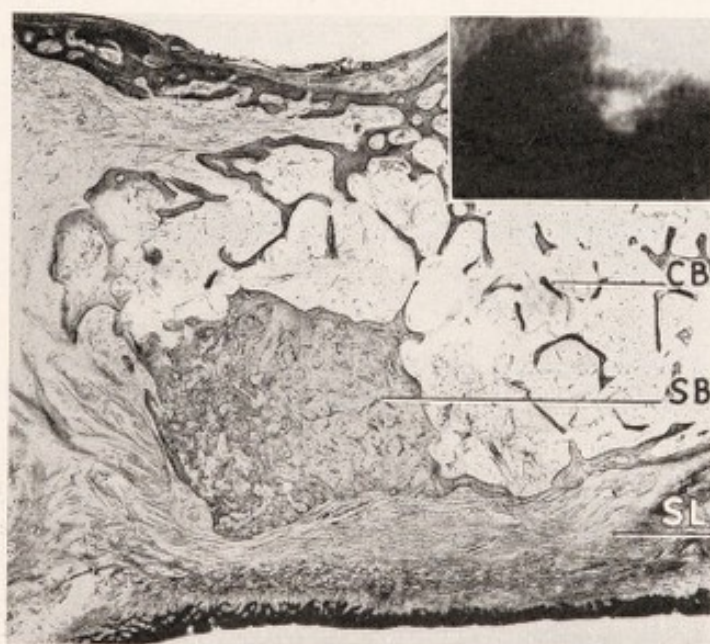


FIG. 137.—Mesio-distal section through an edentulous maxilla. The radiograph in the right upper corner shows a distinctly outlined radiopaque area in the posterior portion of the maxilla. SL, soft tissue lining of the edentulous maxilla; CB, cancellous bone of the maxilla; SB, sharply outlined area of compact bone lying next to the surface of the bone.

alveolar ridge in the region of the molars, which looks very much like a root. However, upon close examination no periodontal space as is usually observed around healed-in roots could be found; there is

a diffuse transition between the dense area and the surrounding cancellous bone. This fact makes the diagnosis of an area of condensed bone most probable. The edentulous part of the jaw was sectioned mesio-distally. These specimens show a sharply outlined area of compact bone in the region in which radiographically there was great resistance to the penetration of the rays (Fig. 137). Neither the bone nor the surrounding fat marrow shows any trace of inflammation, and it is difficult to determine the etiology of the circumscribed bone condensation. Most probably at some previous time an infected molar root caused irritation and subsequent bone condensation, similar to the condition illustrated in Fig. 136. Later on, this root was extracted or fell out and the dense bone remained, now a non-inflamed, bony scar. Of course, it also is possible that originally a rarefying osteitis was present around the infected root and that, after the infection had subsided, the former granuloma was transformed into fibrous scar tissue, which was changed into compact bone.

In view of the microscopic findings in these and several other cases of condensing osteitis caused by infected teeth, surgical interference is contraindicated in areas of compact bone. After the removal of the infected tooth, the remaining bony scar is free from inflammation, and nothing would be gained by its removal; besides, since the transition from condensed bone to normal cancellous bone is frequently very indistinct, a clean surgical removal of all sclerotic bone would be very difficult.

BIBLIOGRAPHY.

- BAUER, W.: Zur Kenntnis der bisher als "Zementcuticula" bezeichneten Epithel-Hyalinmembran im Wurzelgebiete der Zähne, *Ztschr. f. Stom.*, 1926, **24**, 606.
- Über zystische Bildungen im Kiefer, *Ztschr. f. Stom.*, 1927, **25**, 205.
- COOK, THOMAS J.: Dental Granuloma, *Jour. Am. Dent. Assn.*, 1927, **14**, 2231.
- EULER, H.: Periapikale Herde in Ihrer Bedeutung für die fokale Infektion, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 683.
- Apikale Periodontitis, *Handw. d. ges. Zhk.*, vol. **3**, p. 2014.
- FELDMANN, G.: Epitheliale Vegetationen bei chronischer Periodontitis, Apikalen Granulomen und Cysten, *Deutsch. Mon. f. Zhk.*, 1928, **46**, 1251.
- FREEMAN, N.: Histopathological Investigations of the Dental Granuloma, *Jour. Dent. Res.*, 1931, **11**, 175.
- GAYLER, V.: Studien über den histologischen Aufbau der Zahngranulome, *Deutsch. Mon. f. Zhk.*, 1930, **48**, 1451.
- GOTTLIEB, B.: Ätiologie und Prophylaxe der Zahnkaries, *Ztschr. f. Stom.*, 1921, **19**, 129.
- HARNDT, E.: Histo-bakteriologische Studie bei Periodontitis chronica granulomatosa, *Korr. f. Zahnärzte*, 1926, **50**, 330, 365, 399, 426.
- HÄUPL, K., and BAUER, W.: Über die Apikale Parodontitis, *Ztschr. f. Stom.*, 1929, **27**, 275.

- HESS, WALTER: Grangrän, *Handw. d. ges. Zhk.*, vol. 2, p. 768.
———Nekrose der Pulpa, *Handw. d. ges. Zhk.*, vol. 3, p. 2274.
- HILL, THOMAS J.: The Epithelium in Dental Granulomata, *Jour. Dent. Res.*, 1930, 10, 323.
———Experimental Dental Granulomas in Dogs, *Jour. Am. Dent. Assn.*, 1932, 19, 1389.
- JAMES, W. WARWICK and COUNSELL, ARTHUR: A Histological Study of the Epithelium Associated with Chronic Apical Infections of the Teeth, *Brit. Dent. J.*, 1932, 53, 463.
- JOB, THESLE T., and FOUSER, RALPH H.: Relationship of the Teeth to the Mandibular Canal and the Maxillary Sinus, *Jour. Am. Dent. Assn.*, 1927, 14, 1072.
- MACMILLAN, HUGH W.: The Relationship of the Teeth to the Maxillary Sinus: Anatomic Factors Underlying the Diagnosis and Surgery of This Region, *Jour. Am. Dent. Assn.*, 1927, 14, 1635.
- MAXIMOW, ALEXANDER A.: Morphology of the Mesenchymal Reactions, *Arch. Path. and Lab. Med.*, 1927, No. 4, p. 557.
———Bindegewebe und Blutbildende Gewebe, *Möllendorff's Handb. d. mikr. Anat.*, Berlin, Springer, 1927, 2, 232.
- ORBAN, B.: Contribution to the Histology of the Dental Pulp and Periodontal Membrane, with Special Reference to the Cells of "Defence" of These Tissues, *Jour. Am. Dent. Assn.*, 1929, 16, 965.
- SCHOUR, I.: A Review of Maximow's Research on Inflammatory Reaction, *Jour. Am. Dent. Assn.*, 1930, 17, 1605.
- SHEARER, W. L.: The Maxillary Sinus, *Jour. Am. Dent. Assn.*, 1931, 18, 340.
- STEIN, GEORG: Histologische Untersuchungen im Wurzelspitzengebiete Pulpotomierter Zähne, *Ztschr. f. Stom.*, 1929, 27, 108.
- THOMA, K. H.: A Histopathologic Study of the Dental Granuloma and Diseased Root Apex, *Jour. Am. Dent. Assn.*, 1917, 4, 1075.
- THOMA, K. H.: The Histological Pathology of Alveolar Abscesses and Diseased Root-Ends, *Dent. Cosmos*, 1918, 60, 13.
- TSCHISTOWITSCH, TH., and MECHTEIS, J.: Über die Rolle des Epithels in den Wurzelgranulomen, *Ztschr. f. Stom.*, 1930, 28, 1163.
- TSUZUKI, MASAO: Beiträge zur Histologie der Zahnwurzelcysten mit besonderer Berücksichtigung der Wände grosser Cysten, *Deutsch. Mon. f. Zhk.*, 1928, 46, 65.
- WEBER, W.: Periodontitis Granulomatosa, *Deutsch. Mon. f. Zhk.*, 1926, 44, 675.
- YUMIKURA, SHIGEIE: Über einen rätselhaften Fall von Cuticula dentis an einer von Periodontium entblösten Wurzelspitze, *Ztschr. f. Stom.*, 1925, 23, 860.

CHAPTER VII.

TISSUE CHANGES FOLLOWING ROOT CANAL THERAPY.

ANATOMY OF THE HUMAN ROOT CANALS.

KNOWLEDGE of the anatomy of the human root canals is essential for all systematic root canal therapy. The investigations of the last decade have brought about so much new and basic information on the form and arrangement of the root canals that it seems necessary to describe the most typical forms before entering into the discussion of the tissue changes associated with the treatment of these canals.

Through the apical foramen the dental pulp is connected with the blood and nervous systems of the jaw. In a certain percentage of teeth this connection is established by a single strand of tissue that passes through one wide straight apical foramen. Much more frequently, however, the pulp is divided at the root end into several branches that pass through the dentin and cementum by several fine canals.

In case of one simple apical opening a number of variations can be observed, which will be illustrated by means of longitudinal sections through root ends of teeth in position in the jaw. The simplest known form of apex is illustrated in Fig. 138. The apical foramen is simply a continuation of the wide, straight root canal, its opening lying in the most apical portion of the root. The bloodvessels and nerves of the pulp pass through the foramen and connect through the adjacent bone with the main vessels and nerves of the jaw. The apical foramen itself is formed by cementum; the border between cementum and dentin, the dento-cemental junction, has no significance whatsoever in the form or diameter of the apical foramen, nor is there any indication that the foramen is the narrowest point in the entire root canal. While there is in some cases a narrowing of the root canal in its apical part, frequently the root canal is of uniform width throughout, and sometimes it even becomes wider at the apical foramen. The author wishes to emphasize this point as there seems to be a rather widespread opinion among practitioners that the foramen is always the narrowest point in the course of the root canal and, therefore, may be recognized clinically.

The simple, straight form of apical opening illustrated in Fig. 138 is not frequently found. In examining histological sections of various human teeth of different ages this type of foramen was found only in about 22 per cent, or about one-fifth of several hundred cases. More frequently the apical part of the root canal shows a certain degree of curvature; either the entire root end is curved, or



FIG. 138.—Apex of upper central incisor. Straight, wide root canal. Slight constriction of the canal at the apical foramen. (Coolidge, Jour. Am. Dent. Assn.)

the root end is straight and the canal is curved, so that the foramen will not be located at the extreme end of the root. This is illustrated in Figs. 139 and 140. In Fig. 139 the foramen is found at the very tip of the root, but the entire apical portion of the root is curved. In Fig. 140 the root end is straight, but, due to a deviation of the the root canal, the apical foramen is located on the side of the root tip.

In the majority of human root ends the root canal does not run as one single canal all through the dentin, but is separated into two or several canals near the root end. These divisions of the root canal are called apical ramifications. Other root canals have branches that run at right angles from the main canal to the root surface; they are called lateral branches. The frequent occurrence



FIG. 139.—Apex of mesial root of lower second molar. The root end is slightly curved toward the distal side. (Coolidge, Jour. Am. Dent. Assn.)

and clinical importance of these structures have not been recognized until recently. Only through the painstaking investigations of Hess do we have complete knowledge of the anatomical variations in human root canals in different groups of teeth.¹

¹ The results of the work of Hess and his associate, Zürcher, have been published in English under the title, "The Anatomy of the Root Canals of the Teeth of the Permanent Dentition. The Anatomy of the Root Canals of the Teeth of the Deciduous Dentition and of the First Permanent Molars" (London, J. Bale, Sons and Danielsson, 1925). The study of this book is highly recommended to any one interested in the minute anatomy of human root canals.

The following technique was used by Hess for his investigations: A large number of intact human teeth were collected and recorded separately as to kind of tooth and age of patient. The pulp chambers were then opened and the organic content of the pulp canals removed by maceration and rinsing. After the canals were thoroughly cleaned, unvulcanized soft rubber was pressed into



FIG. 140.—Apex of a cuspid. The root end is straight but the root canal is curved. As a result, the apical foramen is not located at the root end but on the side of the root. (Coolidge, Jour. Am. Dent. Assn.)

them from the pulp chamber until the rubber completely filled all spaces inside the tooth. Then the tooth was put in the vulcanizer and vulcanized until the rubber in the canals had become hard. By subjecting these teeth to the action of a strong acid, the entire dentin was corroded, and the remaining vulcanite cast represented an exact reproduction of all hollow spaces inside the tooth. These

vulcanite casts were studied, and the number and distribution of the ramifications recorded. A few of the specimens of Hess will be reproduced here, as they give a clear picture of the inside anatomy of human teeth.

Fig. 141 shows the vulcanite casts of four upper central incisors. Two of the illustrated teeth have simple root canals without branching; whereas, of the other two, one shows several small apical branches, and the other shows a larger canal at right angles to the main canal. Similar conditions are found in upper lateral incisors (Fig. 142).

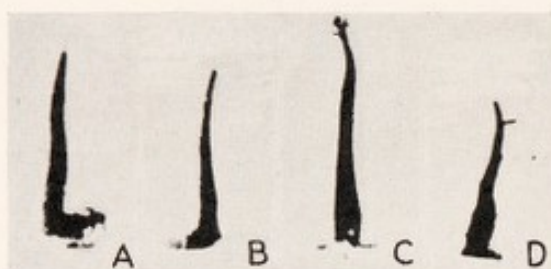


FIG. 141.

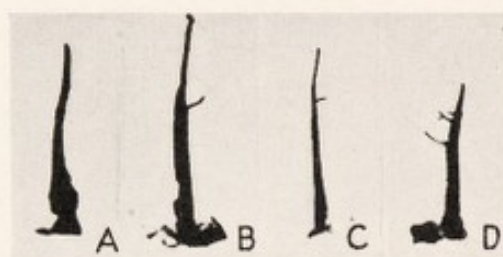


FIG. 142.

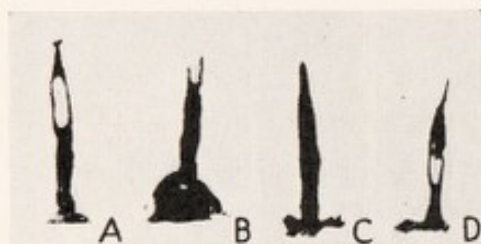


FIG. 143.

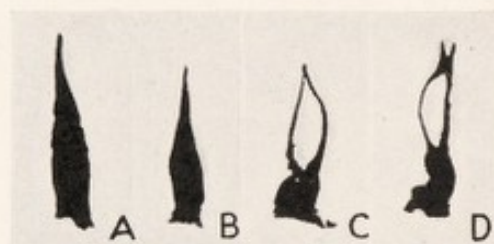


FIG. 144.

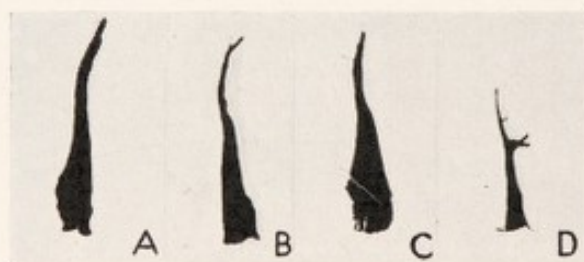


FIG. 145.

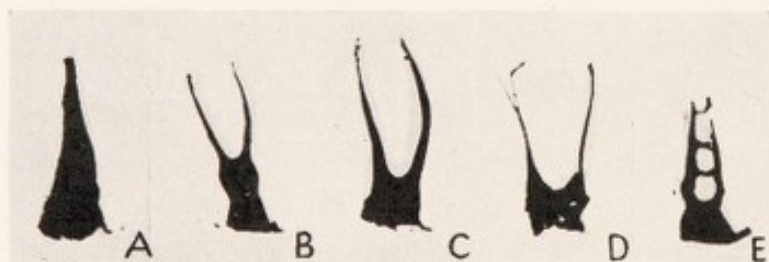


FIG. 146.

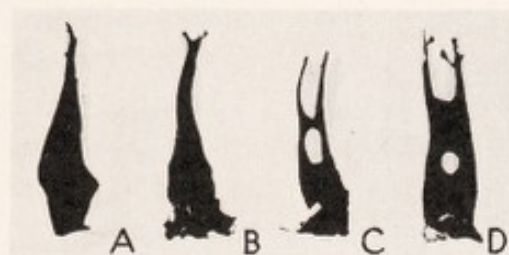


FIG. 147.

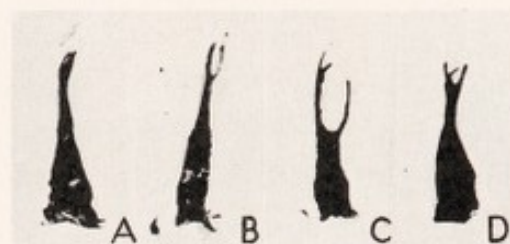


FIG. 148.

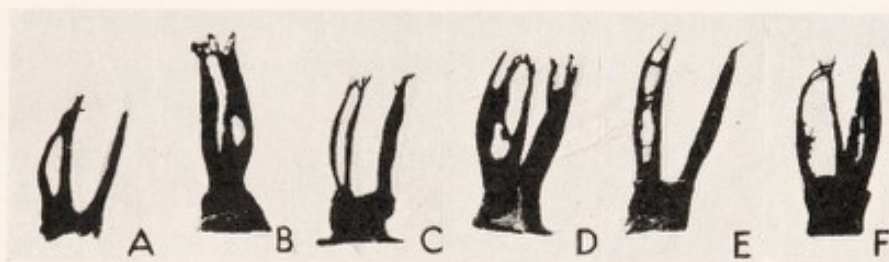


FIG. 149.

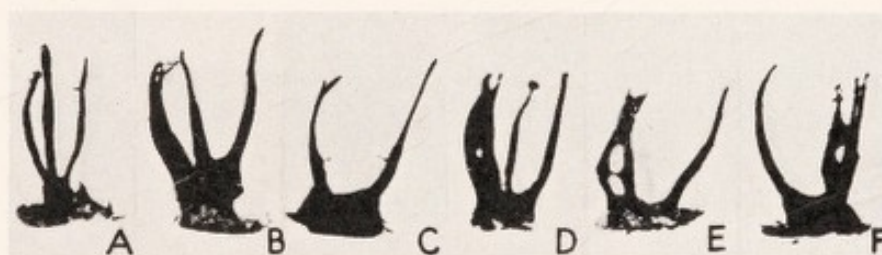


FIG. 150.

FIGS. 141-150.—Anatomy of the root canals of human permanent teeth. (Hess, Schweiz. Vrtljschr. f. Zhk.)

FIG. 141.—Vulcanite casts of the root canals of the upper central incisors. A, B, single straight canal; C, apical ramifications; D, lateral branch.

FIG. 142.—Upper lateral incisors. A, single straight canal; B, C, D, lateral branches.

FIG. 143.—Lower central and lateral incisors. A, B, D, partial division of the main canal; C, single wide canal.

FIG. 144.—Lower cuspid. A, B, single wide canal; C, D, two canals.

FIG. 145.—Upper cuspid. A, single wide canal; B, apical ramification; C, D, lateral branches.

FIG. 146.—Upper first bicuspid. A, single wide canal; B, C, D, different forms of two canals; E, connections between the two canals.

FIG. 147.—Upper second bicuspid. A, single wide canal; B, single canal with apical ramifications; C, two canals; D, partial division of the canal with apical ramifications.

FIG. 148.—Lower first and second bicuspids. A, B, D, apical ramifications; C, partial division of canal.

FIG. 149.—Lower first and second molars. A, C, three canals with apical ramifications; E, connections between the two mesial canals; B, F, wide canals with apical ramifications; D, four canals.

FIG. 150.—Upper first and second molars. A, B, three canals with apical ramifications; C, two canals; D, E, irregularities of the buccal canals; F, four canals.

In the lower incisors and cuspids, the most remarkable anatomical variation is the occurrence of two main canals that may separate either on the level of the pulp chamber or further apically. Hess found such divisions in lower incisors in 37 per cent of all cases, and in 43 per cent of all lower cuspids (Figs. 143 and 144).

The upper cuspid has a straight root canal which is much wider than the one of the upper incisors. In about 25 per cent of all upper cuspids apical ramifications are observed; sometimes a lateral branch leaves the main canal in its apical portion at a right angle (Fig. 145).

In the upper bicuspid the anatomy of the root canals is rather complicated. According to the investigations of Hess, 19.5 per cent, or one-fifth of all upper first bicuspid have one single, wide canal (Fig. 146, A). The remaining four-fifths have two canals with numerous finer branches, some of which connect the two canals with each other (Fig. 146, E), while others represent apical ramifications (Fig. 146, C, D). In the upper second bicuspid the division into two canals is less frequent; 56 per cent, or more than one-half, show one single canal (Fig. 147). Apical ramifications are very common.

Lower bicuspid (Fig. 148) usually have one root canal, a division into two canals being more frequent in the second bicuspid than in the first.

Lower molars vary greatly in form and number of root canals as follows (Hess):

	1 canal.	2 canals.	3 canals.	4 canals.
Lower molars . . .	0.3 per cent	17.7 per cent	78 per cent	4 per cent

Apical ramifications are frequently found on all roots (Fig. 149). Mesial root canals are usually connected to each other by fine branches running horizontally from one main canal to the other (Fig. 149, E). The lower third molar usually has two canals with comparatively few ramifications.

The upper first and second molars usually have three canals; this number may be increased to four by a division of the mesio-buccal canal (Fig. 150, D, F). Apical ramifications were found in more than one-half of all upper molars examined. The root canals of the upper third molar vary more than those of any other tooth; anywhere from one to four or five root canals can be found.

In deciduous teeth similar anatomical conditions were reported. Hess showed that deciduous incisors and cuspids, as a rule, have short, wide, single root canals with occasional ramifications. Deciduous molars, however, have complicated root canals; both upper

and lower molars frequently have four canals and, in addition, many ramifications.

The average frequency of irregularities in the anatomy of the root canals of human permanent teeth was shown by Hess in a table which is reproduced here in abbreviated form. Hess differentiated between apical ramifications, a term he used only for the terminal branches of the main canal, and marrow canals or lateral branches, connections between the main canal and the periodontal membrane at some other place along the root than at the apex. In this table apical ramifications and lateral branches are recorded separately. Several thousand teeth of patients of all ages were used for these investigations; thus, the figures present the average of different ages. It was found that in very young teeth the number of branches and ramifications is small, since the final differentiation and configuration of the root end has not yet been reached. Between the ages of twenty and forty years the number is largest. Later in life some are obliterated by progressing dentin formation, so that in older teeth fewer ramifications are encountered.

THE RELATIVE FREQUENCY OF APICAL RAMIFICATIONS AND LATERAL BRANCHES IN HUMAN PERMANENT TEETH. (AFTER HESS.)

Kind of tooth.	Apical ramifications, per cent.	Lateral branches, per cent.
Upper central incisor	25	21
Upper lateral incisor	31	22
Upper cuspid	25.5	18
Lower central and lateral incisor	21.6	10
Lower cuspid	39	12
Lower first bicuspid	44	17.3
Lower second bicuspid	49	20
Upper first bicuspid	41	18
Upper second bicuspid	50	19
Upper first and second molar	67	16
Upper third molar	80	23
Lower first and second molar	73	13.5
Lower third molar	10	6

In histological specimens these ramifications can easily be studied in serial sections. Figs. 151 to 154 illustrate cross-sections through the apex of a human lower first bicuspid at different levels. In Fig. 151, a cross-section through the root in its lower third, the root canal is single; slightly toward the apex (Fig. 152) the canal changes its shape; here a smaller branch can be seen leaving the main canal. In Fig. 153 the lateral branch has become completely separated from the main canal. The latter splits again as it approaches the root tip (Fig. 154) so that actually three apical foramina are present in this particular apex.

In longitudinal sections through human root ends, lateral canals are found to branch from the main canal at different levels. Apical

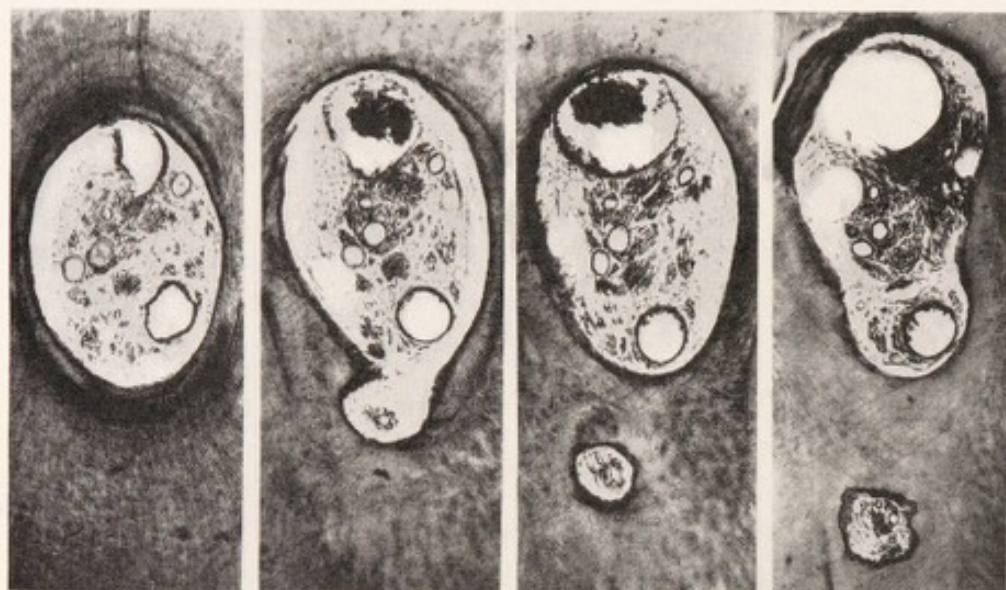


FIG. 151.

FIG. 152.

FIG. 153.

FIG. 154.

FIGS. 151-154.—Successive cross-sections through the apical portion of the root of an intact lower first bicuspid.

FIG. 151.—Single round canal.

FIG. 152.—Branching off of apical ramification.

FIG. 153.—Main canal and apical ramification separated.

FIG. 154.—Branching off of another apical ramification from the main canal.



FIG. 155.—Root end of intact lower second bicuspid of an adult. MC, main canal; AR, apical ramifications; PM, periodontal membrane; AB, alveolar bone; FM, fat marrow of alveolar process.

ramification of the pulp canal near the foramen in a lower bicuspid is illustrated in Fig. 155. Here the pulp branches into three strands

which all pass through the root end (see Fig. 148, D). It should be mentioned that a similar form of apical ramification is always encountered in dogs' teeth (see Fig. 106).

GENERAL CONSIDERATION OF THE CHANGES FOLLOWING REMOVAL OF THE PULP AND TREATMENT OF THE ROOT CANAL.

We shall now consider the tissue changes that take place if a vital pulp is removed from a root canal. A vital pulp may be intact, or inflamed. The main point is that when the pulp is removed, the apical part of the root canal still contains living pulp tissue.

In the following examples the changes at the root end will be described in the order in which they develop. The knowledge of these conditions has been gained partly from human specimens and partly from experimental animals on which identical operations were performed. Pulp removal following exposure of the pulp by caries will be considered first. The dentist, after removing the soft, decayed dentin and finding the pulp exposed, administers a local anesthetic, applies the rubber dam, and, after good access to the pulp chamber has been gained, removes the bulk of the pulp tissue from the root canal by means of a barbed broach. By this operation the pulp strand is torn off somewhere near the apical foramen, creating a wound on the surface of the remaining tissue. The actual location of the wound depends entirely upon local circumstances. In a case like the one illustrated in Fig. 138, the tissues most probably will be severed at the foramen or beyond it, in the apical periodontal tissue. If a condition like the ones illustrated in Figs. 139 and 140 were present, the pulp wound usually will be located at the point where the straight canal becomes curved. The removal of the pulp invariably will be followed by a certain amount of hemorrhage from the torn pulp vessels. After the hemorrhage subsides, a clot of fibrin is formed on the surface of the pulp wound.

The further fate of this wound depends principally upon two factors:

1. Presence or absence of infection.
2. Condition of the soft tissue in the region of the pulp wound.

1. **Infection.**—Every fresh wound presents a good medium for the invasion and development of pathogenic microorganisms. This is especially true of lacerated wounds of the type that results from pulp removal, an uneven wound with a jagged, torn surface and

considerable tissue injury caused by tearing and crushing. Therefore, if pathogenic microorganisms gain access to the pulp wound, the chances are that they will spread rapidly, multiply, and cause a progressive infection of the pulp wound and the underlying tissue. Maintenance of strict asepsis during all manipulation in and about the root canal is essential to prevent infection of the pulp stump. A favorable result of the root canal operation will largely depend upon care in this respect.

2. **Condition of the Remaining Tissue.**—The healing processes after removal of a pulp occur in the tissue immediately adjacent to the point where the pulp was severed. It is, therefore, of great importance to retain the vitality of these tissues in order to make healing possible. Aside from infection, the greatest damage to the tissues at the pulp wound can be done by the use of too powerful and harmful drugs. When root canal therapy was first being developed and practised, strong and powerful drugs were frequently used to enlarge the root canal and to control infection. Concentrated hydrochloric acid and sulphuric acid were among the drugs recommended and used. Since then we have learned to understand and appreciate the importance of the apical tissues for the successful outcome of any root canal operation, and all these strong and tissue-destroying drugs have been ruled out by the dentist. The enlargement of the canals, formerly done by acids, is now performed with sterile, clean-cutting instruments, and the necessity of using strong disinfectants has been overcome by working out a constantly improving system of asepsis. Thus, the living tissues in the depth of the root canal are kept alive and can fulfill their reparative functions.

In this connection it may be of interest to call attention to the parallel development in general surgery and root canal operations. When knowledge about the importance and the prevention of infection was first introduced into the practice of general surgery, strong antiseptic solutions, such as phenol, were generally used on instruments, tissues, and dressings. Since then we have learned about the injurious influence of such drugs upon the living cells; asepsis has taken the place of antiseptics, and instead of fighting bacteria in the field of operation, they are kept out by aseptic measures. No modern surgeon would apply a powerful drug to living tissue that is expected to heal.

In returning from these considerations to the practical case of pulp removal, a sterile, non-irritating dressing is sealed in the root canal. The pulp wound will continue to discharge a serous exudate for a day or two; at the same time, an accumulation of white blood

cells will be found in the tissue next to the wounded surface, a reaction that is found in the neighborhood of every wound of the human body during the early stages of repair. The white blood cells form a protective wall between the wound and the rest of the body; by phagocytosis they dispose of the cell debris that invariably is present wherever injury to the tissue has occurred, and they help overcome the low-grade infection that may be present in the wound.

The exudation and round-cell infiltration following the traumatic injury caused by removal of the pulp are responsible for the slight soreness frequently encountered the day following the operation. Concurrently with the development of the healing process and with the disappearance of both exudation and infiltration, this soreness subsides.

Soon the discharge from the wound stops; a clot of fibrin forms, and under this protective cover fibroblasts begin to proliferate and initiate scar formation. Concurrently with the consolidation of the scar, the number of round cells in the tissues steadily decreases until a plain connective-tissue scar is formed at the point where the pulp was severed.

Quite different changes take place, however, if infection has gained access to the pulp wound, or if a strong, harmful drug has been sealed into the root canal. In the first days after the removal of the pulp, the condition is similar to that present in a sterile case. Soon, however, the amount of round-cell infiltration begins to increase very rapidly, indicating the reaction of the tissue to advancing infection. Instead of the formation of a scar on the surface of the pulp stump, more and more tissue breaks down; sooner or later the periodontal membrane around the foramen is involved, and periodontitis will develop—in clinical terms, the root canal operation has been a failure.

When the reader considers these tissue reactions following pulp removal, he can readily appreciate the advantage of local anesthesia over arsenic trioxide for the removal of living pulp tissue. While the arsenic trioxide may have clinical advantages, it most likely will cause toxic damage to the apical tissues, thereby decreasing the chances for prompt healing of the pulp wound. By using a local anesthetic, the full vitality of the pulp tissues at the wound is preserved, and the chances for prompt healing of the wound and a favorable outcome of the operation are increased.

The portion of the root canal from which the pulp tissue has been removed is usually filled with a root filling material, in most cases guttapercha. The type of material used does not seem to

be of great importance as long as the foreign substance is well tolerated by the tissues and has physical qualities that make its manipulation and practical application possible. That the connective tissue can heal around a non-irritating, sterile, foreign material has been shown for guttapercha as well as for silver or ivory. Because of advantages in manipulation, guttapercha is most commonly used at the present time to fill root canals.

The formation of a connective tissue scar at the root end, after removal of a pulp and filling of the root canal, is almost invariably followed by a deposition of cementum in this area. It is hard to determine just how soon after the removal of a pulp the formation of cementum begins. From findings on human teeth that were extracted a few months following such operations, it seems that the deposition of cementum begins several weeks after the root canal operation; from this time on the amount of the cementum slowly but steadily increases. Cementum formation is particularly important because of the anatomical variations in human root canals (curvature, ramifications) which make a perfect root canal filling technically impossible. It has been found that cementum has a tendency to obliterate all openings that are left after filling a root canal. That such openings may exist is obvious from the study of specimens like the ones shown in Figs. 141 to 155. Only in a root canal of the type that is represented in Fig. 138 is a perfect root filling possible; but even when the filling, upon radiographic examination, may extend to the apical foramen, the root canal will usually be either slightly overfilled or underfilled. In most cases living pulp tissue is left in parts of the main canal or ramifications that are inaccessible to root canal instruments. Following the transformation of the pulp tissue in these canals into connective tissue, a reparative formation of cementum takes place, which finally leads to the obliteration of the lumen, thus forming a far better root canal filling than any foreign material used by the dentist.

The deposition of cementum following root canal therapy was first observed and described in this country by Davis and Grove. In Europe, Swiss investigators lead in this particular field of research (Gysi, Hess, O. Mueller). Later American publications dealing with the same subject have appeared by Blayney, Coolidge, Hatton, Moen, Skillen, and others. All these men report that cementum is deposited upon the root end and in the unfilled part of the canal, with the tendency to cover the root filling material and to obliterate any lumen still present in the apex. They also show that this process occurs only in the absence of infection.

Therefore, this type of cementum formation can actually be considered as the morphological expression of successful root canal therapy.

A few actual findings will be described that were obtained in the study of human teeth whose vital pulps had been removed. In order to interpret the tissue changes found at the apices of such teeth, it might be advisable to compare these periapical changes with the changes following bone fracture, a point recently brought out by Hatton. He said: "In the healing of the broken bone there are found the same stages that are found in the apical region of treated and filled teeth: (1) A stage of destruction or dissolution of the products of the injury, such as blood, damaged soft tissues, and a certain amount of bone; (2) a stage of repair, characterized by the formation, first of a fibrous callus, and later by the production of new bone or the bony callus; and (3) the resting stage."¹ In dentistry the first stage is represented by the reaction of the periodontal tissues to the trauma of pulp removal; during this stage resorptions may occur on both root surface and alveolar bone; hence, the frequent observation of repaired resorptions at the apices of well-healed pulpless teeth (Figs. 156 and 158). After the results of the primary trauma have been overcome, scar formation takes place beginning with the formation of fibrous connective tissue, which later is replaced by bone and cementum. This process of hard tissue formation at the apex is continued until the root end has reached a condition which is most favorable for individual functional conditions; then we find what Hatton terms the "resting stage" during which the pulpless tooth, without any considerable tissue changes, is a useful part of the organism, similar to a bone after a well-healed fracture.

FINDINGS IN HUMAN TEETH THAT WERE REMOVED AFTER ROOT CANAL OPERATIONS HAD BEEN PERFORMED.

The most important source of knowledge of tissue reactions following root canal operations are extracted human teeth with known clinical histories. There are, of course, other lines of approach to this problem, of which the most important one is experimental root canal therapy in animals, but because of the possible difference in tissue reaction between man and animals, the results obtained in animals are of limited value. It must be realized, too, that all animal experiments are of comparatively short duration;

¹ E. H. Hatton, Jour. Am. Dent. Assn., 1931, 18, 1805.

whereas, in man it is possible to examine teeth that were treated ten or fifteen years before the extraction. Since the root end of a devitalized tooth heals slowly, the time factor is of great importance. On the other hand, animal experimentation has the advantage that teeth and jaw together can be removed during autopsy and examined in their original relationship; whereas, in extracted human teeth this relationship is lost. Methods have been suggested by which human root ends and their surrounding tissues can be removed from the jaw by using a trephine, but the resulting injury to the jaw forbids this method as a routine procedure in man. Fortunately it is not necessary to take such drastic steps. We have found that the great majority of extracted human teeth retain enough periodontal tissue on the root surface and at the foramen to allow drawing definite conclusions as to the condition of the root end. The following illustrations are all taken from such specimens, and in every one of these cases the changes associated with the loss of the pulp can be clearly recognized.

1. Tissue Changes in the Main Canal Following Root Canal Filling.—The microscopic examination of a large number of teeth with root canal fillings that upon radiographic examination seemed entirely to fill the root canal revealed that only in a small number of these roots did the filling material end level with the apex. In most cases the roots were slightly underfilled; the length of the unfilled portion of the main canal varied between 0.5 and 2 mm. The unfilled part of the root canal contained fibrous connective tissue, which may have been either a remnant of the original pulp tissue in this part of the canal or periodontal connective tissue that proliferated into the open apical portion of the root canal. The connective tissue had a marked tendency toward the formation of cementum which was deposited in layers on the wall of the pulp canal. Fig. 156 illustrates an early stage of cementum deposition. The gutta-percha cone ends about 1.5 mm. short of the apical foramen; the remaining portion of the root canal is filled by fibrous tissue. The cementum covering the outer surface of the root end continues through the foramen into the canal; its thickness decreases toward the root canal filling. Six months had elapsed between the removal of the living pulp and the extraction of this tooth.

In Fig. 157, a lower bicuspid, a similar condition is illustrated. Here a slightly more advanced cementum deposition has taken place because more time has elapsed since the root canal operation was performed. The guttapercha cone ends about 1 mm. above the apex. The apical portion of the root canal contains well-

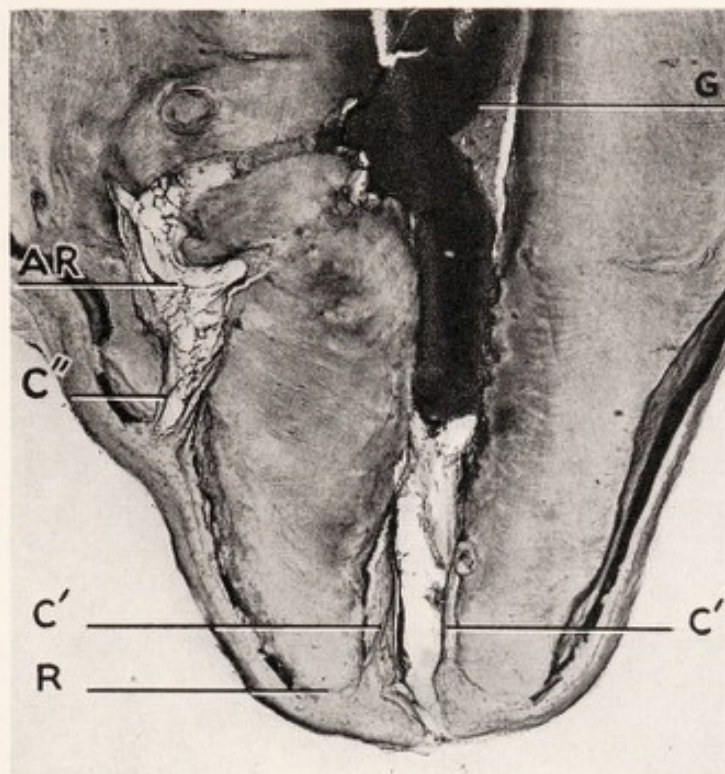


FIG. 156.—Condition of root end six months after pulp removal and root canal filling. Buccal root of upper molar. G, guttapercha filling in main canal; AR, apical ramifications of the main canal; R, areas of resorption in cementum and dentin covered over by new layers of cementum; C', cementum extending upward into the unfilled portion of the main canal; C'', cementum extending into the apical ramifications. (Coolidge, Jour. Am. Dent. Assn.)



FIG. 157.—Cementum deposition in the apical portion of the root canal several years after pulp removal and root canal filling. G, guttapercha; C, cementum covering the wall of the apical portion of the root canal; C', cementoid (newly formed uncalcified cementum) deposited upon the guttapercha; CT, connective tissue in the root canal.

vascularized connective tissue that is entirely free from any evidence of inflammation; cementum has been deposited all over the wall of the root canal. The surface of the guttapercha cone is covered by a layer of uncalcified cementum (cementoid). Attention should be called to the fact that the right wall of the root canal runs in an absolutely straight, smooth line which can be explained only by the action of a cutting instrument, a broach or reamer. During enlargement of the root canal the operator apparently reached the apical foramen; the root canal was filled, but the filling material did not reach the foramen and the apical portion of the canal was subsequently filled in by cementum.

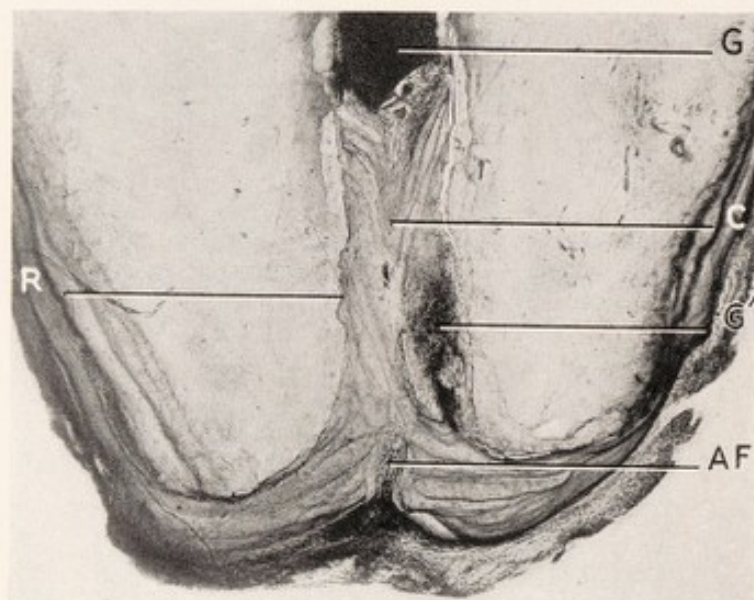


FIG. 158.—Advanced stage of cementum deposition in the apical portion of the root canal. The pulp was removed and the canal filled eight years before the extraction of the tooth. G, guttapercha; C, cementum in the apical portion of the root canal; R, resorptions in the wall of the root canals filled in by cementum; G', small particle of guttapercha completely embedded in cementum; AF, apical foramen. (Courtesy of E. D. Coolidge.)

The condition of the root end eight years after the root canal operation is illustrated in Fig. 158. The obliteration of the unfilled, apical part of the canal is almost complete; the connective tissue in the canal has been reduced to a thin strand of tissue while several layers of cementum cover the dentinal walls. The surface of the dentin forming the wall of the root canal was resorbed before cementum deposition took place; evidently resorption preceded tissue reparation in the canal. A small piece of guttapercha, which became dislodged close to the apical foramen, has been completely embedded in the layers of cementum that surround the remaining thin strand of connective tissue.

If the guttapercha reaches the apical foramen and ends in the level of the root surface, a fibrous capsule is formed bridging the foreign material; later on, new cementum is deposited on the root surface and sometimes also directly upon the guttapercha surface (Fig. 159). The latter observation is of great practical and theoretical importance. It shows that guttapercha as a root filling material is well tolerated by human connective tissue, as this tissue not only grows against the guttapercha without any evidence of irritation, but also a direct deposition of cementum occurs upon the guttapercha surface. Such findings certainly indicate that theoretical objections against guttapercha as a foreign body are

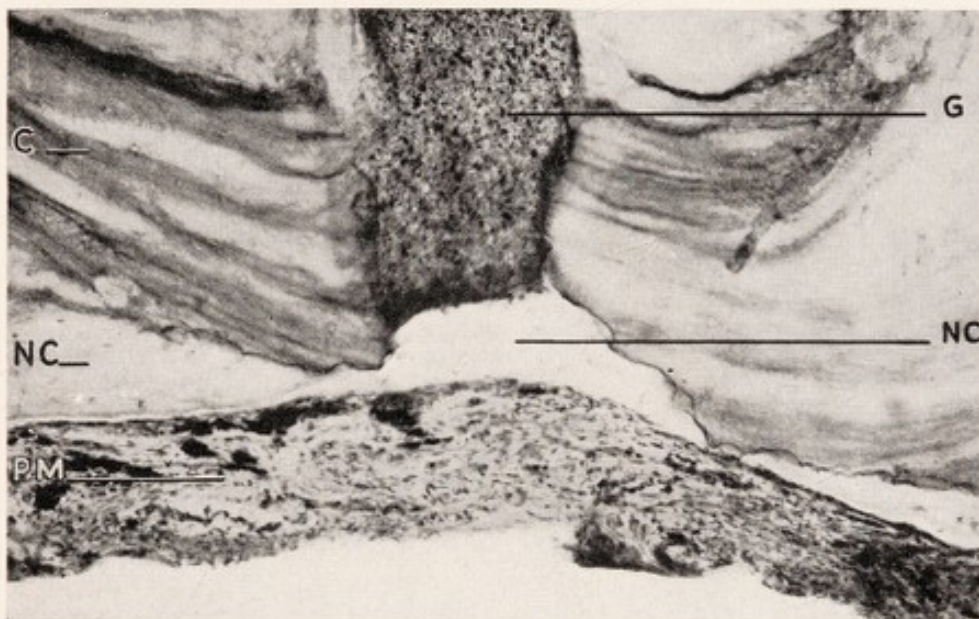


FIG. 159.—Deposition of cementum upon root end and root canal filling. Lingual root, upper molar; treated and filled seven years before extraction. G, guttapercha; C, original cementum on root surface; NC, newly deposited cementum covering root surface and end of guttapercha cone; PM, periodontal membrane.

not supported by the actual reaction of the living human tissue to this material. In an examination of the periodontal tissue covering the root end of the specimen, only fibroblasts and connective tissue fibrillæ are found; no evidence of inflammatory reaction is present.

If the root filling material protrudes slightly beyond the apical foramen it is covered over with a dense, fibrous, connective tissue capsule (Fig. 160), which is attached all around the apical foramen to a newly deposited layer of cementum. In the tissue of the capsule, giant cells are sometimes found which compare to the foreign-body giant cells found in the neighborhood of silk sutures or other foreign materials that are implanted or inserted into the

tissues. No inflammatory cells can be found in the periapical connective tissue of non-infected cases.

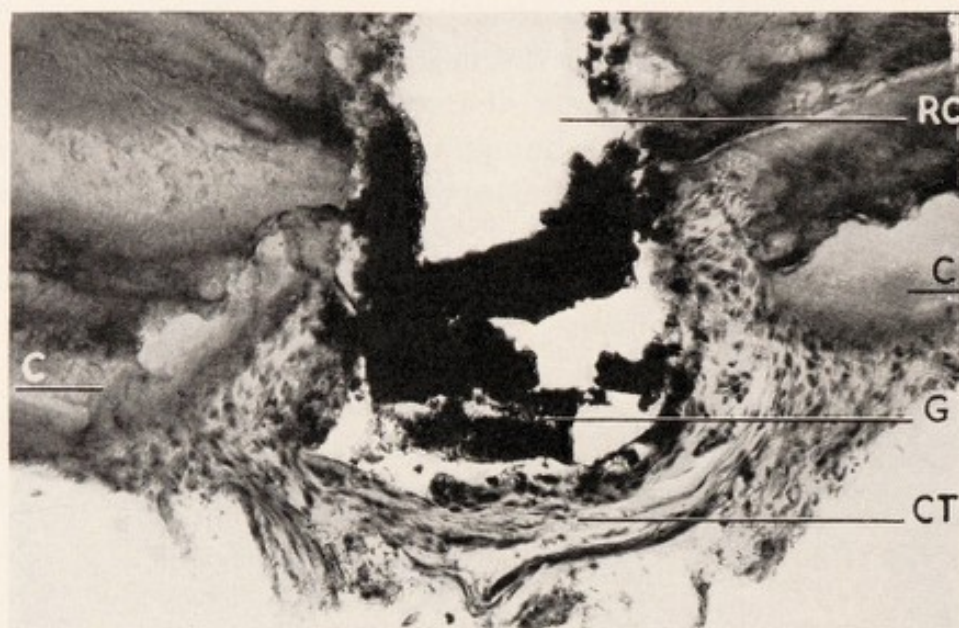


FIG. 160.—Root end with slightly overfilled root canal. Lower bicuspid. RC, root canal; G, guttapercha; CT, connective tissue capsule surrounding the end of the root canal filling; C, deposits of new cementum on either side of the root canal.

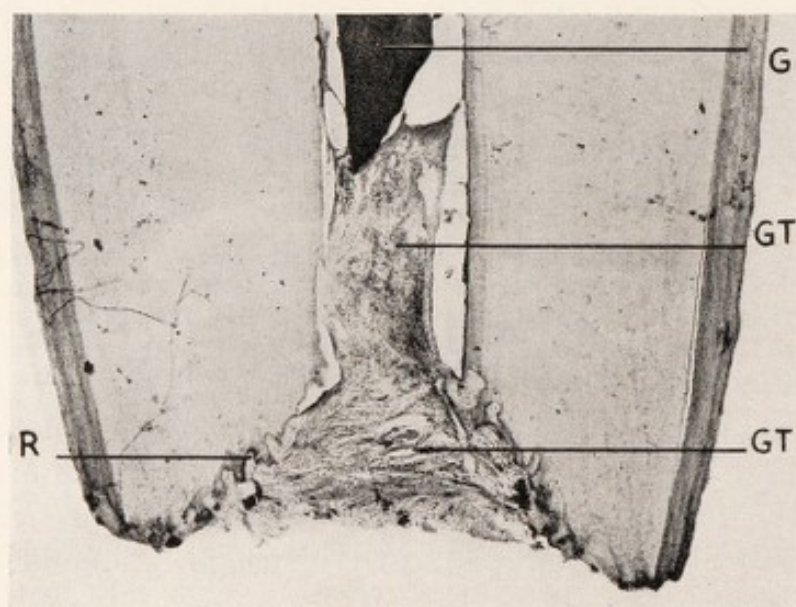


FIG. 161.—Infected root end. Upper central incisor. G, guttapercha; GT, granulation tissue in the root canal and at the apical foramen; R, resorption of root end. Notice the complete absence of cementum formation in the root canal and at the apical foramen.

An entirely different microscopic picture is encountered in root ends of infected pulpless teeth. Here the typical deposition of cementum that leads to the formation of a final hard-tissue scar

is never found; instead, granulation tissue develops at the root end. The width of the root canal is not reduced; on the contrary, the canal may be found widened by resorption without evidence of reparative changes. Such a condition is illustrated in Fig. 161, which was taken of the root end of an upper incisor which, radiographically, showed resorption of both root ends and surrounding bone. The guttapercha root canal filling ends about 2 mm. above the apical foramen; the latter has assumed a funnel shape by resorption of the surrounding dentin. The unfilled portion of the root canal is occupied by granulation tissue with a large number of inflammatory round cells of the chronic type (polyblasts). The

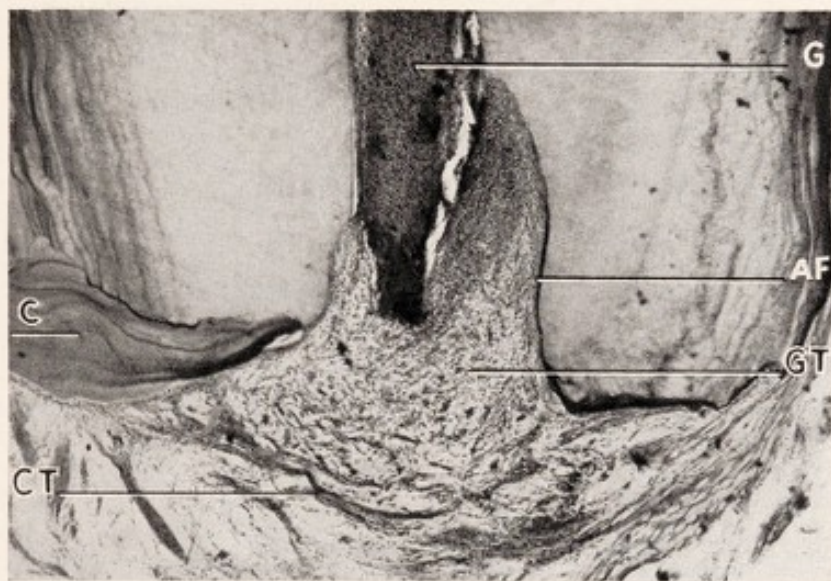


FIG. 162.—Infected root end. Mesial root, lower molar. G, guttapercha; GT, granulation tissue in root canal; AF, apical foramen widened by resorption of the walls; C, deposits of cementum in the periphery of the apical foramen; CT, connective tissue capsule extending across the apical foramen. Notice the absence of cementum at the apical foramen and in the root canal.

granulation tissue has grown against the walls of the canal and against the guttapercha cone without any deposition of cementum. A simple comparison of Fig. 161 with Fig. 158 will reveal the vast difference between tissue changes in the presence and in the absence of infection.

Another case of infection in a tooth with a root canal filling is illustrated in Fig. 162, which was taken of the distal root of a lower molar. The apical foramen has been widened by resorption; densely infiltrated tissue fills the space between the wall of the foramen and the guttapercha root canal filling. Farther distant from the infected root canal, cementum has been deposited upon the root surface; from this new layer of cementum, strands of connective

tissue extend across the root opening forming a kind of capsule over the granulation tissue. Cementum formation is completely absent in the inflamed area inside of the apical foramen. In a higher magnification of the tissue in the root canal, the cell types that are characteristic of chronic inflammation can be recognized: plasma cells, lymphocytes, and large phagocytic cells (polyblasts).

2. Tissue Changes in the Lateral Branches and Apical Ramifications Following Root Canal Filling.—Ever since the existence of the various branches and ramifications of the human root canals became known, these structures were considered of great significance in the problem of root canal therapy. It is evident that no technique



FIG. 163.—Opening of a lateral branch of the same root the apex of which was illustrated in Fig. 160. Ingrowth of cementum from the root surface into the lateral canal. D, dentin; C, cementum of root surface; PM, fibers of periodontal membrane attached to the root surface; C', cementum deposition in the lateral canal; CT, strand of connective tissue and capillaries entering the lateral canal.

could make possible a perfect root filling in a canal of the form illustrated, for example, in Fig. 155. Such irregularities and ramifications of the root canal can never be filled completely. Fortunately, however, a complete filling in the mechanical sense is by no means necessary. Microscopic findings on extracted teeth with clinically well-filled, uninfected main canals prove that nature takes care of the remaining unfilled lateral branches and apical ramifications. All these fine canals contain living tissue which remains vital after the pulp has been removed from the main canal and forms cementum which eventually may completely obliterate the lateral canals.

An early stage of reparative cementum formation in the apical

ramifications of the main canal is illustrated in Fig. 156. The fine branches contain connective tissue; an invagination of the cementum on the root surface into the opening of the ramifications has taken place, reducing the width of these canals. A more advanced-stage of cementum deposition was observed in a fine lateral branch of the same tooth, the apex of which is shown in Fig. 160. The continuation of the cementum into the lateral canal is plainly visible in Fig. 163; the tissue in this canal as well as the surrounding

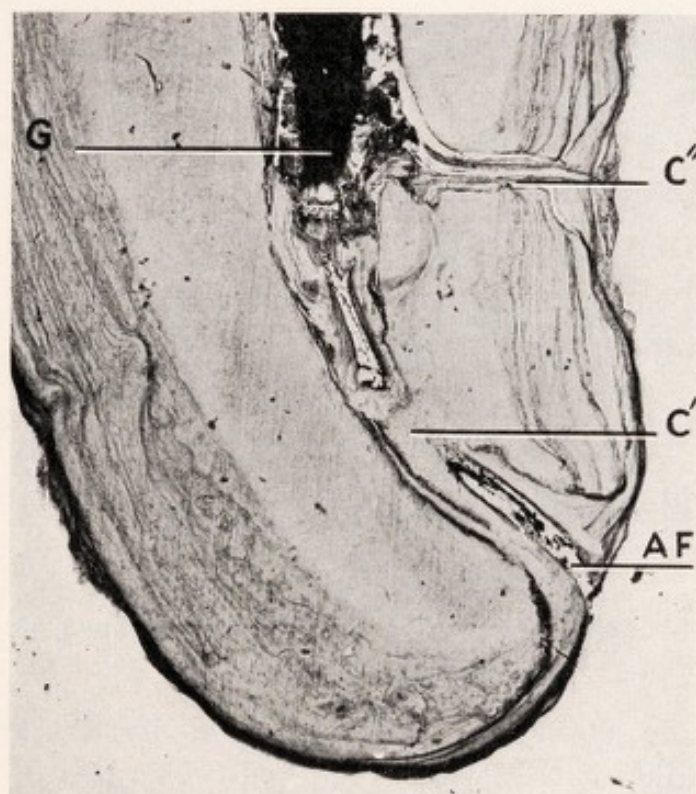


FIG. 164.—Root end of lower molar. Curvature of main canal, lateral branch. G, end of gutta-percha root filling; C', cementum deposition in the curved apical portion of the main canal; C'', cementum deposition in the lateral branch; AF, apical foramen of the main canal greatly reduced in width by newly formed cementum. (Coolidge, Jour. Am. Dent. Assn.)

periodontal connective tissue are entirely free from any inflammatory reaction.

A root end in which the same reparative cementum deposition has occurred in the curved apical portion of the main canal as well as in a lateral branch is illustrated in Fig. 164. The gutta-percha cone ends approximately at the level of the lateral branch; from here to the apex both the main canal and the lateral branch are filled by cementum, leaving only a thin strand of living fibrous tissue in the center of each canal. Eventually the lateral canals may become completely obstructed by cementum (Fig. 165); then

the cementum on the root surface is continuous over the former openings of the ramification.

At the apical end of main canals that are inaccessible to root canal instrumentation on account of large denticles or marked curvature of the root end, the same changes that occur in inaccessible lateral branches can be reasonably expected. The remaining tissue will form cementum, and will eventually lead to obliteration of the apical portion of the canal, provided infection has been kept out (see Fig. 164).



FIG. 165.—Complete obliteration of the opening of a lateral branch. G, gutta-percha in the main canal; some of the gutta-percha has entered the lateral branch; CT, connective tissue remnants in the lateral canal; C, cementum on the root surface covering the opening of the lateral canal in a continuous layer.

3. Changes in the Apical Soft and Hard Tissues Following the Treatment of Infected Pulpless Teeth.—In Chapter VI the histopathology of the apical tissues of teeth with decomposed pulps and infected root canals was described. In such teeth an area of bone destruction around the apex is almost always found upon radiographic examination. Their treatment is a rather difficult problem. The dentist faces the questions: Should an attempt be made to save such teeth? What are the chances for success in such an operation? Microscopic findings justify such an attempt. Not only is it possible to make considerable areas of periapical bone destruction disappear through treatment of the teeth, but also it has been found in the microscopic examination of treated teeth of this kind that the same reparative and healing processes occurred as after the removal of a vital pulp. However, it must always be kept in mind that the treatment of a tooth with a decomposed or infected pulp may or may not be successful, and that there is a decided difference between the condition present in a tooth after

removal of a vital pulp and the one present in a tooth with a decomposed pulp and periapical infection. In case of pulp removal, the chances for undisturbed healing are the same as the chances for success in any other properly performed dental operation. In case of a tooth with decomposed pulp and infected periapical tissues, the treatment is complicated and the prognosis is uncertain.

The difficulty in treating teeth with decomposed pulps seems to be failure to get complete access to all parts of the root canal and to the diseased area beyond the apex. In discussing the tissue changes following removal of a vital pulp, it has been shown that those parts of the pulp canal that are inaccessible to instruments are readily taken care of by nature and are eventually obliterated by formation of cementum (Figs. 163 to 165). In pulpless, infected teeth, such inaccessible parts (ramifications, curved canals) continue to act as a source of infection, making healing of the periapical region impossible. If a tooth with a configuration of the apex of the type illustrated in Fig. 139 or 164 were infected, the curved root end would make access to the periapical region difficult, perhaps impossible; if the root canal had lateral branches, cleaning and filling the main canal would not control the infection in the ramifications. From this viewpoint, the prognosis of periapical infection in pulpless teeth appears rather doubtful. Still every practitioner has seen treated cases in which large areas of bone destruction around the apex disappeared after treatment and the radiograph showed a normal periodontal space. Such teeth, when examined under the microscope, did not show inflammation of the periodontal tissues; cementum had been deposited upon the root end, and the connective tissue surrounding the root was free from inflammation. Such a case is illustrated in Figs. 166 and 167, (Coolidge). Nine years before extraction, this tooth, a lower cuspid, showed an imperfect root canal filling and an area of bone destruction around the apex. At that time the root canal was opened, cleaned, and filled; subsequently the bone around the apex regenerated. The tooth was observed for nine years and then extracted to make room for an artificial restoration. The microscopic examination shows a root filling extending all the way to the apex. A layer of connective tissue remained attached to the root end after extraction; this tissue proved to be free from any round-cell infiltration; it had grown against the root filling material (guttapercha). The dentin surface at the root end shows evidence of previous resorptions which were repaired by deposition of cementum.

Similar changes may take place after a root resection has been performed. Clinically, it is well known that large areas of bone

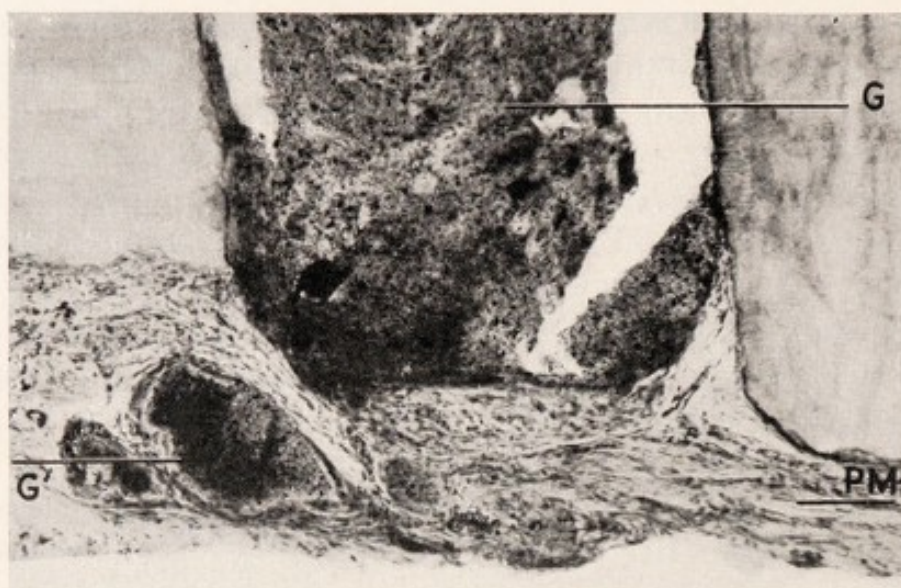


FIG. 166.—Root end of lower cuspid. Chronic periapical infection treated and root canal filled nine years before extraction. G, guttapercha in root canal; G', particles of guttapercha dislodged beyond the apical foramen; PM, periodontal membrane. (Courtesy of E. D. Coolidge.)

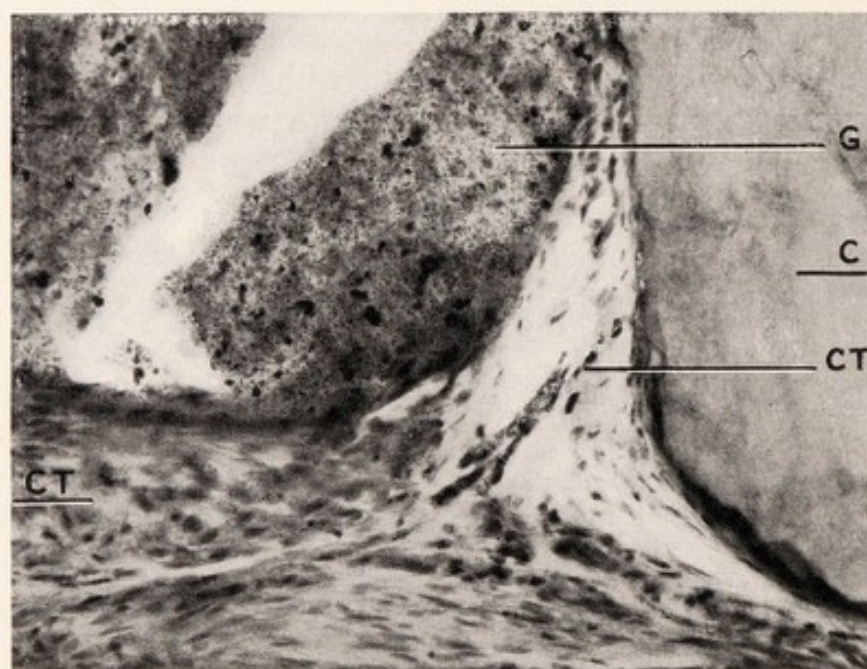


FIG. 167.—High magnification of Fig. 166. G, guttapercha; C, cementum; CT, fibrous connective tissue of periodontal membrane lying in direct contact with guttapercha and wall of root canal. Notice the absence of any inflammatory reaction in the periapical connective tissue. (Courtesy of E. D. Coolidge.)

destruction around the apex of an infected, pulpless tooth may heal completely after the root end has been resected and the pulp

canal filled. Several cases of this type were examined microscopically (Aisenberg, Blayney, Blum, Coolidge, Hill). All these clinically successful cases showed a normal, uninfamed periodontal membrane and deposition of cementum upon the resected dentin surface. The case published by Coolidge will be illustrated here. Fig. 168, A, shows an extensive area of bone destruction around the root end of an upper first bicuspid; this radiograph was taken in

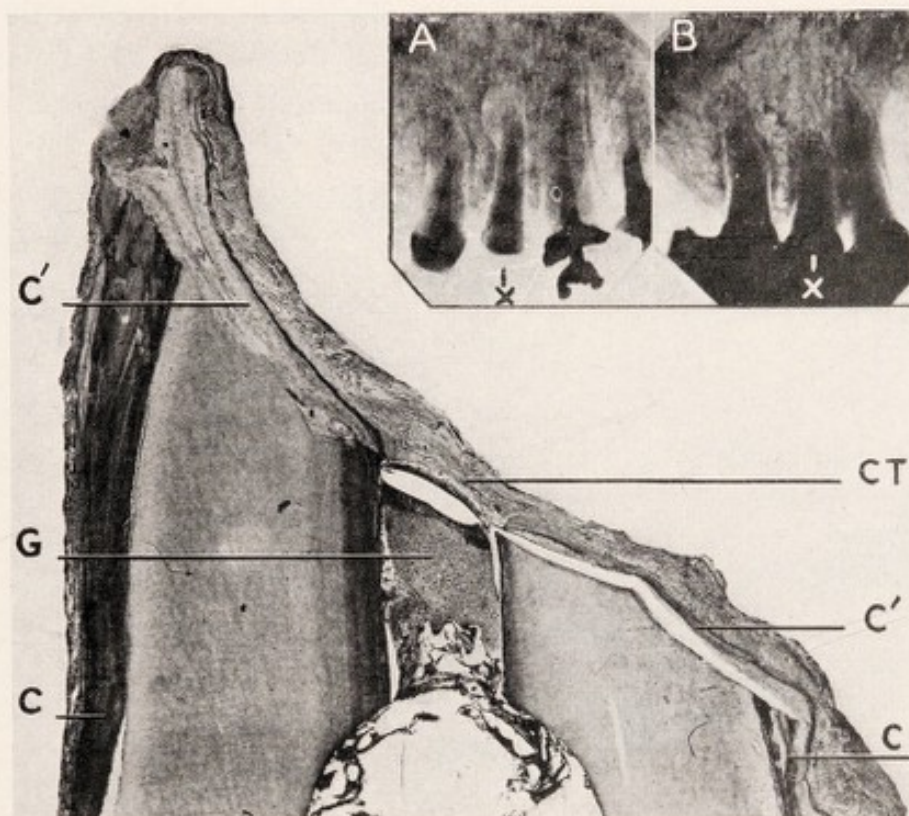


FIG. 168.—Tissue healing following root resection. A, radiograph of an upper second bicuspid with imperfect root canal filling and marked destruction of the periapical bone. This radiograph was taken in 1914, immediately before the root resection was performed. B, same tooth as it appeared in 1928. The area formerly occupied by the apex and the granuloma is entirely filled in by bone. Bucco-lingual section through resected root end. Deposition of cementum and regeneration of a normal periodontal membrane on the resected surface. G, guttapercha root canal filling; C, original cementum on root surface; C', newly deposited cementum on the resected root surface; CT, connective tissue capsule bridging the root filling material. (Coolidge, Jour. Am. Dent. Assn.)

1914, immediately preceding the root resection. Fourteen years later, in 1928, the bone had been completely regenerated (Fig. 168, B). The histological examination of the root end revealed the presence of a continuous layer of newly formed cementum upon the dentin surface (Fig. 168, C); the upper end of the guttapercha cone is covered by fibrous connective tissue without any evidence of inflammation (Fig. 169). The periodontal tissue extending from

the newly formed cementum on the root end to the bone has the typical fibrous structure of a normal periodontal membrane.

The deposition of cementum upon the dentin of a formerly undoubtedly infected root and the regeneration of a normal periodontal membrane are of great practical importance; they show that complete healing in such a case is actually possible. The process of healing after infection of root canal and periapical tissues has recently been studied experimentally by Stein, who used the teeth of dogs for his investigations. The pulps of the teeth were exposed, and the pulp tissue was infected with a known pathogenic strain of microorganisms (streptococci). Then the tooth was sealed

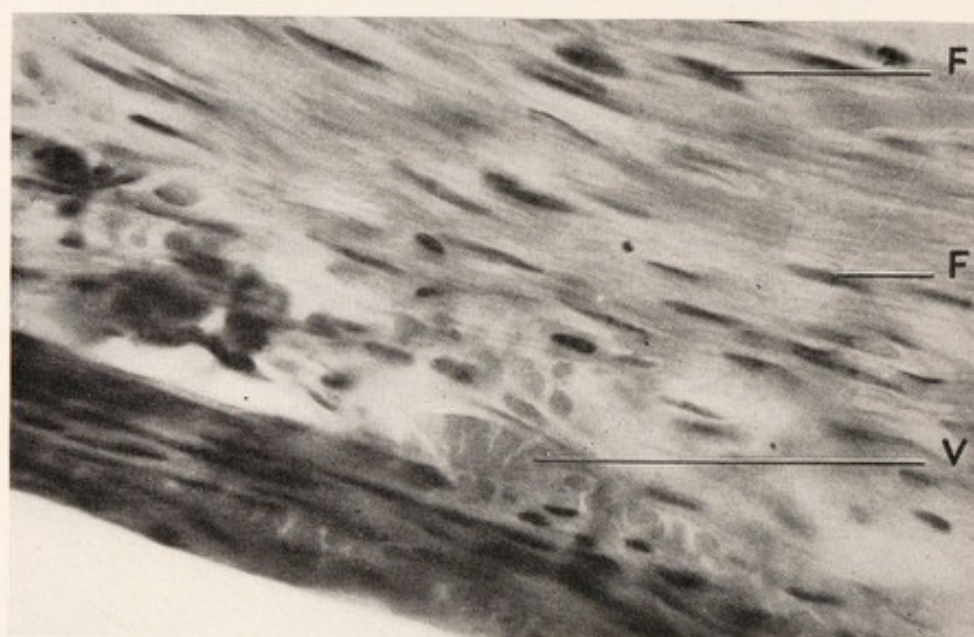


FIG. 169.—High magnification of the connective tissue covering the root canal filling in Fig. 168. F, fibroblasts; V, capillary vessel containing red blood corpuscles. (Coolidge, Jour. Am. Dent. Assn.)

and the animal kept under observation for from five to seven months. During this time, the development of an area of bone destruction around the apex could be observed by means of the radiograph. The infection spread beyond the foramen and involved the bone. After this period, a rubber dam was placed over the infected teeth; the root canals were opened and treated in the same manner as in identical circumstances in men. After root fillings had been inserted into the canals, the animal was kept under observation for another period of several months. During this time, frequent radiographic check-ups revealed that the areas of bone destruction around the apices were decreasing in size and in some instances disappeared entirely. The animals were then killed, and

the treated teeth together with their surrounding jaw tissues were sectioned. In these sections, Stein demonstrated that such areas of chronic inflammation may heal: new bone is built, scar tissue forms on the surface of the root and over the root filling material, and the round-cell infiltration disappears. The histological findings around these infected and treated animal teeth are similar to the conditions found in human teeth with a similar clinical and radiographic history.

In summarizing, it must be said that the outcome of the treatment of pulpless, infected teeth must be considered doubtful. However, in view of the fact that evidence of complete healing and repair following treatment of such teeth has been produced both clinically and microscopically, an attempt to save such teeth seems to be well justified. There is no doubt in the author's mind that a large number of the pulpless teeth treated by present-day routine methods are still in a state of chronic infection; but as the methods and the technique of root canal therapy improve and develop, the number of cases showing complete healing will steadily increase.

BIBLIOGRAPHY.

- AISENBERG, MYRON S.: Root Resection after Four Years: Report of a Case, *Jour. Am. Dent. Assn.*, 1931, **18**, 136.
- BAUER, W.: Histologische Befunde an Zähnen nach Wurzelspitzenamputation, *Ztschr. f. Stom.*, 1922, **20**, 601.
- Mikroskopische Befunde an Zähnen und Parodontien nach experimenteller Wurzelspitzenamputation unter besonderer Berücksichtigung der Bedeutung functioneller Auswirkungen, *Ztschr. f. Stom.*, 1925, **23**, 122.
- BLAYNEY, J. R.: The Biologic Aspect of Root-canal Therapy, *Dent. Items Int.*, 1927, **49**, 681.
- What Teeth Should be Extracted: A Report Based upon Further Studies of Root-canal Therapy, *Jour. Am. Dent. Assn.*, 1928, **15**, 1217.
- Tissue Reactions in the Apical Region to Known Types of Treatment, *Jour. Dent. Res.*, 1929, **9**, 221.
- The Problem of the Pulpless Tooth as Seen by Clinical and Laboratory Investigation, *Jour. Dent. Res.*, 1930, **10**, 425.
- Fundamentals Governing Pulp-canal Therapy, *Dental Cosmos*, 1932, **74**, 635.
- BLAYNEY, J. R., and WACH, EDWARD C.: A Study of Tissue Repair Around a Resected Root End, *Dental Forum*, 1924, p. 58.
- BLUM, THEODOR: Root Amputation: A Study of One Hundred and Fifty-nine Cases, *Jour. Am. Dent. Assn.*, 1930, **17**, 249.
- Additional Notes on Root Amputation, Including a Study of Thirty-eight New Cases, *Jour. Am. Dent. Assn.*, 1932, **19**, 69.
- BUNTING, R. W.: Is a Pulpless Tooth a Dead Tooth? *Jour. Am. Dent. Assn.*, 1921, **8**, 1016.
- COOLIDGE, EDGAR D.: Pulp Pathology and Treatment Problems, *Jour. Am. Dent. Assn.*, 1928, **15**, 1623.
- Studies of Germicides for the Treatment of Root Canals, *Jour. Am. Dent. Assn.*, 1929, **16**, 698.

- COOLIDGE, EDGAR D.: Anatomy of the Root Apex in Relation to Treatment Problems, *Jour. Am. Dent. Assn.*, 1929, **16**, 1456.
- Root Resection as a Cure for Chronic Periapical Infection: Histologic Report of a Case Showing Complete Repair, *Jour. Am. Dent. Assn.*, 1930, **17**, 239.
- The Reaction of Cementum in the Presence of Injury and Infection, *Jour. Am. Dent. Assn.*, 1931, **18**, 499.
- Reaction of Dog Tissue to Drugs Used in Root Canal Treatment, *Jour. Am. Dent. Assn.*, 1932, **19**, 747.
- Pathology, Diagnosis and Treatment of the Pulp and Preparation of Root Canals for Filling, *Jour. Am. Dent. Assn.*, 1932, **19**, 1964.
- DAVIS, W. CLYDE: Histopathology of the Cementum as Related to Pulp canal Surgery, *Dental Cosmos*, 1920, **63**, 766.
- Structural Changes Within the Pulp Canals of Teeth Following Partial Pulp Removal, *Dental Summary*, 1920, p. 311; 1921, pp. 482, 563, 653, 787.
- EULER, H.: Experimentelle Studie über den Heilverlauf nach Wurzelspitzenresektion und über den Einfluss verschiedener Wurzelfüllungsmaterialien auf den Heilverlauf, *Deutsch. Mon. f. Zhk.*, 1923, **41**, 321.
- FELDMANN, G.: Die Apikale Parodontitis im Lichte des Experimentes, Berlin, Meusser, 1931.
- Neue Wege in der Therapie von Zähnen mit entzündeter Pulpa, *Vrtljschr.*, 1932, **48**, 211, 307.
- GOTTLIEB, B., and ORBAN, B.: Veränderungen im Periodontium nach chirurgischer Diathermie, *Ztschr. f. Stom.*, 1930, **28**, 1208.
- GOTTLIEB, B., ORBAN, B., and STEIN, G.: Das Problem der Wurzelbehandlung. Die Kontrolle von Wurzelbehandlungsmethoden durch experimentelle Wurzelbehandlung, *Ztschr. f. Stom.*, 1932, **30**, 187.
- GROVE, C. J.: Nature's Method of Making Perfect Root Fillings Following Pulp Removal, with a Brief Consideration of the Development of Secondary Cementum, *Dental Cosmos*, 1921, **63**, 968.
- HARTZELL, THOMAS B.: The Pulpless Tooth, *Dental Cosmos*, 1930, **72**, 1177.
- HATTON, EDWARD H.: Possibility of Apical Regeneration after Root Canal Filling from the Histopathologic Point of View, *Jour. Am. Dent. Assn.*, 1922, **9**, 192.
- Histopathology of the Periapical Region, *Jour. Am. Dent. Assn.*, 1925, **12**, 49.
- Histologic Studies of Living Tissue Reactions Associated with Pulpless Teeth that May be Taken as Evidence of Satisfactory or Physiologic Healing, *Jour. Am. Dent. Assn.*, 1931, **18**, 1508.
- HATTON, EDWARD H., MARSHALL, JOHN A., RICKERT, U. GARFIELD, BLAYNEY, JAMES R., and HALL, EDOUARD M.: Methods and Fundamentals in the Allied Sciences Essential to Successful Root-canal Surgery, *Dental Cosmos*, 1928, **70**, 249, 380.
- HATTON, EDWARD H., SKILLEN, W. G., and MOEN, O. H.: Histologic Findings in Teeth with Treated and Filled Canals, *Jour. Am. Dent. Assn.*, 1928, **15**, 56.
- HESS, WALTER: Zur Anatomie der Wurzelkanäle des menschlichen Gebisses, *Schweiz. Vrtljschr. f. Zhk.*, 1917, **27**, 1.
- Formation of Root Canals in Human Teeth, *Jour. Am. Dent. Assn.*, 1921, **8**, 704, 790.
- Anatomie des Wurzelkanals, *Handw. d. ges. Zhk.*, vol. 4, p. 3146.
- HESS and ZÜRCHER: The Anatomy of the Root Canals of the Teeth of the Permanent Dentition. The Anatomy of the Root Canals of the Teeth of the Deciduous Dentition and of the First Permanent Molars, London, J. Bale, Sons and Danielsson, 1925.
- HILL, T. J.: Regeneration of Periodontal Membrane after Root Curetment, *Dental Cosmos*, 1931, **73**, 799.

- KRONFELD, RUDOLF: Zur Frage der Wurzelspitzenamputation, *Ztschr. f. Stom.*, 1928, **26**, 1105.
- The Present Status of the Pulpless Tooth, *The Bur*, Chicago College of Dental Surgery, March, 1932.
- MOEN, O. H.: Tissue Changes in Treated Teeth of Known History, *Jour. Am. Dent. Assn.*, 1928, **15**, 2075.
- ORBAN, B.: The Problem of Root Canal Treatment, *Jour. Am. Dent. Assn.*, 1932, **19**, 1384.
- RYWKIND, A.: Über Zementablagerung in den Wurzel-Kanälen und der Pulpa-kammer, *Ztschr. f. Stom.*, 1926, **24**, 923.
- SCHACHTEL, E.: Über die Heilungsvorgänge nach Wurzelspitzenresektion, *Deutsch. Mon. f. Zhk.*, 1929, **47**, 618.
- SKILLEN, W. G.: Hard Tissue Changes Noted Within the Canals of Treated Teeth and Their Possible Significance, *Jour. Am. Dent. Assn.*, 1924, **11**, 350.
- The Status of the Treated Tooth, *Jour. Am. Dent. Assn.*, 1926, **13**, 291.
- Morphology of Root Canals, *Jour. Am. Dent. Assn.*, 1932, **19**, 719.
- STEIN, GEORG: Experimentelle Wurzelbehandlung, *Ztschr. f. Stom.*, 1931, **29**, 744.
- ULLIK, R.: Die histologische Kontrolle von verschiedenen Methoden der Wurzelkanalreinigung, *Ztschr. f. Stom.*, 1929, **27**, 1037.

CHAPTER VIII.

THE BIOLOGY OF CEMENTUM.

DEVELOPMENT AND STRUCTURE OF CEMENTUM.

CEMENTUM is a calcified substance that covers the outer surface of the root. It is a product of the connective tissue of the periodontal membrane, consisting of an organic ground substance (matrix) into which inorganic salts are precipitated. The deposition of cementum begins while the root portion of the tooth germ is developing within the jaw; under physiological conditions it continues throughout life, so that the thickness of the cementum increases steadily as the tooth grows older.

In order to understand the various structural changes found in cementum at different ages, it is necessary first to consider its development. Since the fine details of early cementum formation cannot be seen in hematoxylin-stained specimens, the preparations have to be treated with silver stains; by this method it is possible to see the first delicate layers of cementum and their relationship to the collagenous fibers of the periodontal membrane. The author has studied the consecutive stages of cementum formation in specimens of young human teeth; a few of the characteristic findings will be reproduced here.

Immediately after the dentin of the root surface is formed, it lies in contact with the connective tissue that surrounds the germ; however, it has no vital connection with this connective tissue. As the dentin is formed within the tooth by the pulp, all its vital connections, its metabolism, and its innervation are derived from the pulp tissue; the outside of the dentin apparently presents an inert and biologically inactive surface to the adjacent connective tissue. Gottlieb compared the dentin in this stage of development with a sterile, hard, foreign substance, such as a piece of ivory, implanted into connective tissue, but without organic connection with its surroundings. The organic connection between the dentin and the connective tissue is later established by a layer of cementum that is deposited upon the dentin surface from the outside. During this deposition collagenous fibers of the adjacent connective tissue are embedded into the forming cementum, establishing the

first true connection between dentin and periodontium. Since the other ends of these fibers are attached to the bone, the foundation for later functional unison, cementum—fibers—bone, is laid.

The deposition of the first very delicate layer of cementum and the simultaneous embedding of fibers into this cementum can be visualized by comparing sections of the same specimen that have been stained differently. Fig. 170, *A* and *B*, illustrates an area of a first permanent molar before its eruption through the overlying soft tissue. The dentin surface is surrounded by loose connective tissue, the fibrous elements of which run parallel to the root surface for the most part, since functional stimuli upon the tooth are still absent and no functional orientation of the periodontal membrane

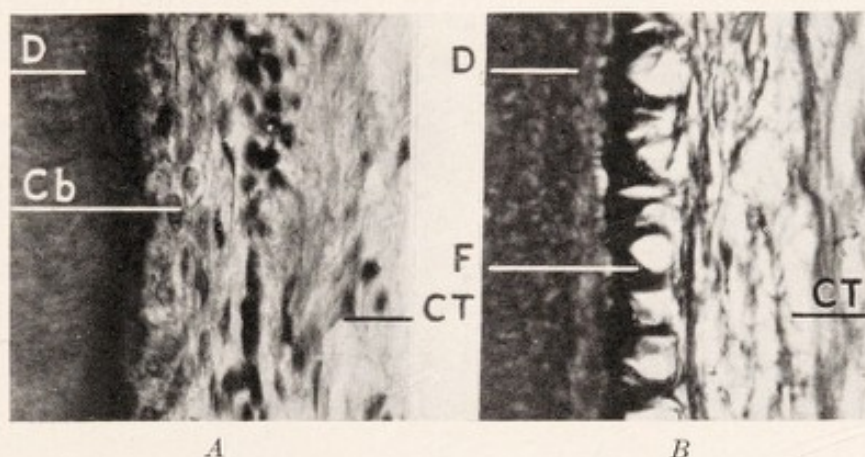


FIG. 170.—Earliest stage of cementum formation on the root of a human tooth before eruption. *A* and *B* illustrate corresponding areas of the root surface; the section *A* is stained with hematoxylin and eosin, the section *B* with Bielschowsky's silver impregnation method. D, dentin; Cb, cementoblasts; F, fibers attached to the dentin surface with club-shaped endings; CT, loose connective tissue surrounding the root. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

has yet taken place. Next to the root surface, the cells are densely arranged, and some of them are grouped in a single row on the root surface, an arrangement which is typical of cementoblasts.

If the same specimen be treated with silver stain (Bielschowsky—method of silver impregnation) the cellular elements in the tissue become invisible; but the fibers lying between these cells stain black (Fig. 170, *B*). Thus, it can be seen that the dentin surface is beset with club-shaped, thickened ends of fibers that appear to be glued or cemented to the dentin surface by a fine black layer of cementum. If the surface of the root of this same tooth is examined farther toward the crown where the cementum is older, it is found that the fibers are attached to the slightly thicker cementum in an identical manner. Each fiber bundle becomes thicker next to the

root surface, and this thickened end is attached to the cementum. The fiber bundles cannot be followed farther into the cementum; the latter shows only a fine, fibrillar structure; the bundles end directly on its surface. The peripheral endings of the fiber bundles radiate into a diffuse fibrous network surrounding the root.

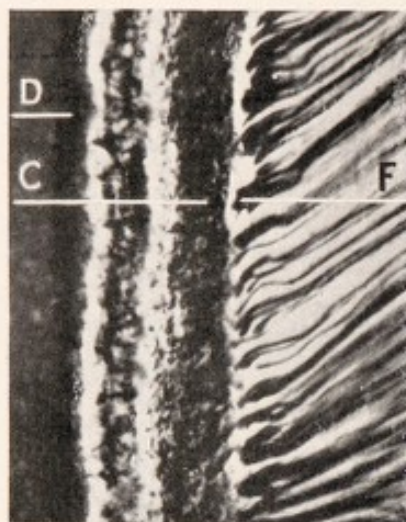


FIG. 171.—Cementum in an erupting molar of a child. Sharpey's fibers are attached only to the superficial layers of the cementum. No continuation of these fibers into deeper layers of cementum is visible (Bielschowsky stain). D, dentin; C, cementum; F, attachment of Sharpey's fibers to the surface of the cementum. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

When the tooth erupts the cementum has already attained a certain thickness (Fig. 171). The fiber bundles still end on the very surface of the cementum, with slightly thickened endings. The course of the fibers, however, is decidedly different from that found in unerupted teeth; the fibers are

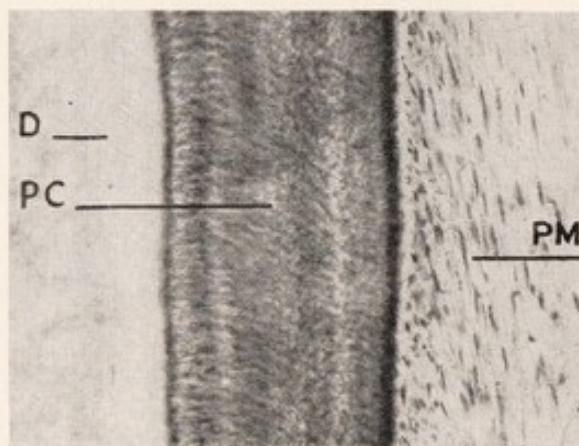


FIG. 172.—Primary cementum. D, dentin; PC, primary cementum showing a fine fibrillar structure; PM, periodontal membrane.

arranged in bundles, as is characteristic of the Sharpey's fibers of functioning teeth, and run at an angle of about 45 degrees from the root surface to the alveolar bone. The cementum has only a fine fibrillar structure, no continuation of the fiber bundles into the cementum being visible.

It is usual to differentiate in the study of the morphology of the cementum between primary cementum and secondary cementum.

Primary cementum contains no cells and shows only a fine fibrillated pattern at right angles to the root surface (Fig. 172). It is usually found in the coronal portion of the root. Primary cementum rarely becomes very thick.

Secondary cementum contains cells embedded in the matrix, which gives it an appearance very much like bone. Secondary

cementum is usually found in the apical portion of the tooth, although it extends crownward to a different degree in different teeth. It should be emphasized, in opposition to statements that are occasionally encountered in dental literature, that, except for their structural differences, primary and secondary cementum are not different in any way. It is not yet known why in some teeth the major portion of the root surface is covered by primary cementum, while in others secondary cementum is predominant, but as far as functional and biological significance are concerned, the two types of cementum are identical. As will be shown later on (Figs. 178 and 179), a resorbed area on the root surface may be repaired just as well by primary cementum as by secondary cementum with apparently no difference in the functional value of the repaired area.



FIG. 173.—Lacunæ and canaliculi in the secondary cementum (stained ground section). The canaliculi radiate from each lacuna in all directions and are connected with canaliculi from the neighboring lacunæ.

Usually secondary or cell-containing cementum is formed at the root end first, from which its deposition progresses crownward with advancing age. Its histological characteristic is the presence of cells, the cement corpuscles or cement cells, that lie in lacunæ in the matrix. These cells, which are derived from the periodontal membrane, are cementoblasts that became embedded into the matrix during its deposition. Each individual lacuna in the secondary cementum is connected with neighboring lacunæ by fine channels, the canaliculi, which contain the protoplasmic processes of the cells in the lacunæ (Fig. 173). Thus, a system of channels is established by which fluids may pass from the periodontal membrane into the superficial layer of cementum and from there may be conveyed to

deeper strata. The presence and arrangement of the cells makes the secondary cementum appear similar to bone; hence the name osteocementum is sometimes used for this hard tissue.

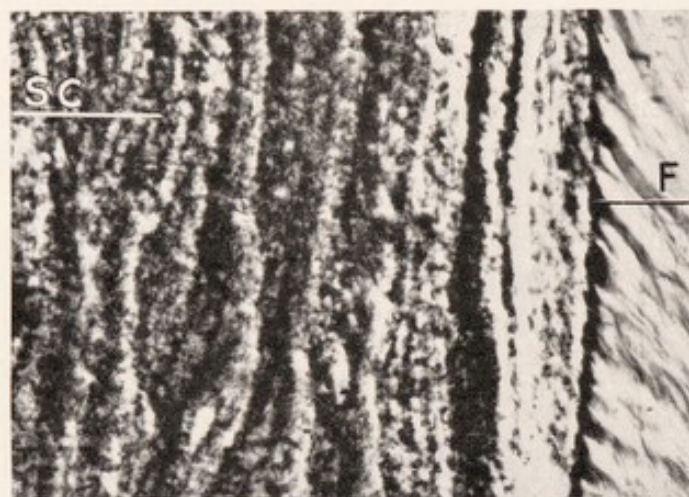


FIG. 174.—Thick layer of secondary cementum on the root of an erupted molar (Bielschowsky stain). SC, secondary cementum showing lamellated structure (lines of deposition); F, Sharpey's fibers attached to the surface of the secondary cementum. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

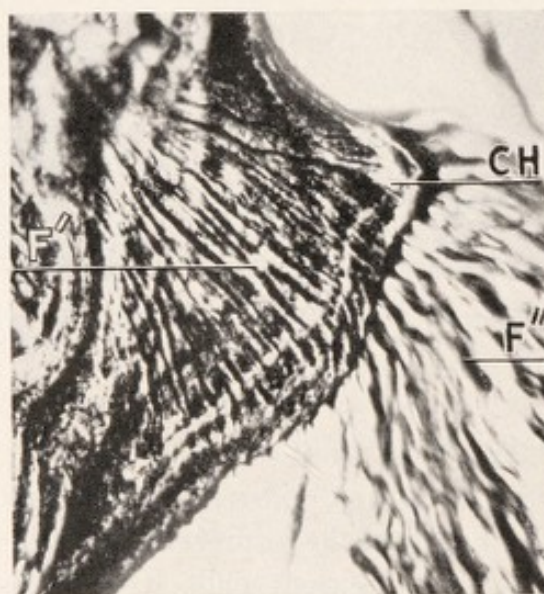


FIG. 175.—Sharpey's fibers embedded into a small cementum hyperplasia. The fibers can be traced into the deeper layers of cementum (Bielschowsky stain). CH, cementum hyperplasia; F', Sharpey's fibers embedded into the cementum; F'', Sharpey's fibers in the periodontal membrane. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

Secondary cementum usually shows a distinctly lamellated structure. The lamellæ run parallel to the root surface, and represent layers of consecutive deposition and calcification. The attachment

of the fiber bundles of the periodontal membrane to the secondary cementum is analogous to their attachment to the primary cementum, the fibers reaching only as far as the most superficial layer of cementum; the deeper layers present only a lamellated pattern (Fig. 174). Only in case of a definite cementum hyperplasia is it sometimes possible to demonstrate the continuation of the fiber bundles into the deeper layers of the secondary cementum (Fig. 175).

It is difficult to give any certain data on the distribution of primary and secondary cementum on the root surface. The author has found during the microscopic examination of human permanent molars shortly before eruption that most of these teeth have very thin layers of primary cementum on the entire outer surfaces of the roots; the sides of the roots, however, that face the interradicular septum and the bifurcation are covered with a thick layer of secondary cementum. If we consider that these teeth are still completely embedded in the child's jaw, it is apparent that neither function nor any other outer influence can be held responsible for the variations in the distribution of cementum on different parts of the root surface.

It may be advisable to compare cementum and bone, and to correlate the physiological changes that take place during life in both these hard tissues.

Morphology.—Bone has a calcified matrix which is arranged in lamellæ that either run parallel to the bone surface or show a circular arrangement around a central lumen. In the matrix, spaces (lacunæ) are found containing bone cells. These cells are connected with each other by fine, protoplasmic extensions lying in the bone canaliculi.

Cementum also has a calcified matrix, identical to bone matrix, which is arranged in lamellæ parallel to the root surface. Primary cementum is free from cells. Secondary cementum has lacunæ containing cementum cells similar to bone cells, which are likewise connected to each other by canaliculi.

Physiology.—In bone a continuous process of elimination by resorption and replacement by newly formed bone takes place (process of molting); the total amount of bone, however, remains unchanged.

Cementum is replaced differently from bone; areas of lowered vitality are not resorbed, but are covered over by a new, highly vital layer of cementum. The total amount of cementum increases steadily throughout life; no resorption occurs under physiological conditions.

FUNCTION OF CEMENTUM.

In the preceding paragraph it was shown that the fiber bundles of the periodontal membrane enter the superficial layers of the cementum, while no fibers are visible in the deeper layers. Gottlieb, who, among dental histologists, was the first to call attention to the biological importance of cementum and to make this hard tissue the subject of intensive study, expressed the following opinion concerning the physiological changes in the cementum: the fiber bundles of the periodontal membrane, like any other tissue in the organism, are subject to involutionary changes: they live and function for a certain period of time, and then they die. Concurrently with this process of aging or involution, new fibers are built that take over the function of the old ones. In the fibers of the periodontal membrane that are embedded in the cementum, these regressive changes are indicated by a precipitation of calcium salts into the fibers. The fibers calcify, and their vitality and functional value is greatly reduced or lost. The complete calcification of all fibers in any circumscribed area of cementum surface would mean the loss of functional attachment in this area. The only way to restore function then would be by the deposition of a new layer of cementum into which a new generation of fibers is embedded. In that way the continuous deposition of cementum throughout life can be regarded as a process by which calcified strata of cementum with low vitality are covered by new layers with living, uncalcified fiber attachments and high vitality.

That this conception of the change in vitality is more than a mere hypothesis can be demonstrated by a study of the cementum cells in the secondary cementum. These cells indicate the vitality of the surrounding cementum. In examining cementum cells in the root of a healthy human tooth, it can be observed that those cells in the superficial layer appear normal and well stained (Fig. 176). In a slightly deeper level the cells show marked degenerative changes; the nuclei are small, the cell bodies are shrunken and irregular. Still deeper in the cementum the lacunæ are empty, the cells having completely degenerated and disappeared. Fig. 177 shows cementum cells from the different layers in Fig. 176 under higher magnification. In Fig. 177, *A*, which was taken of an area of cementum near the surface, two young cells are seen. The cell bodies are slightly shrunken away from the walls of the lacunæ; otherwise, they do not show any pathological changes. In a slightly deeper layer (Fig. 177, *B*), the shrinkage of the cells is much more

pronounced; a large portion of the lacuna appears to be empty. Still further away from the surface the nuclei of the cementum cells

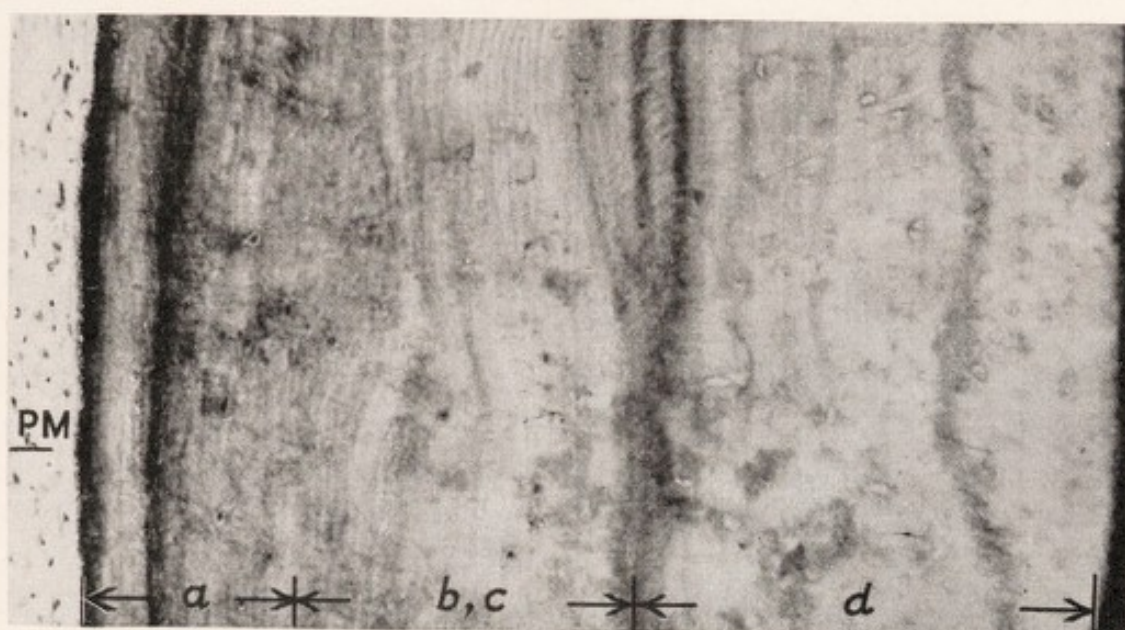


FIG. 176.—Thick secondary cementum on the root of an intact upper bicuspid. PM, periodontal membrane; a, area of normal vital cells. The cementum corpuscles in this area are well stained and appear normal; b, c, area of degenerating cells. The cells stain poorly; some of the lacunæ are empty; d, area of dead cells. All lacunæ of the cementum are empty.

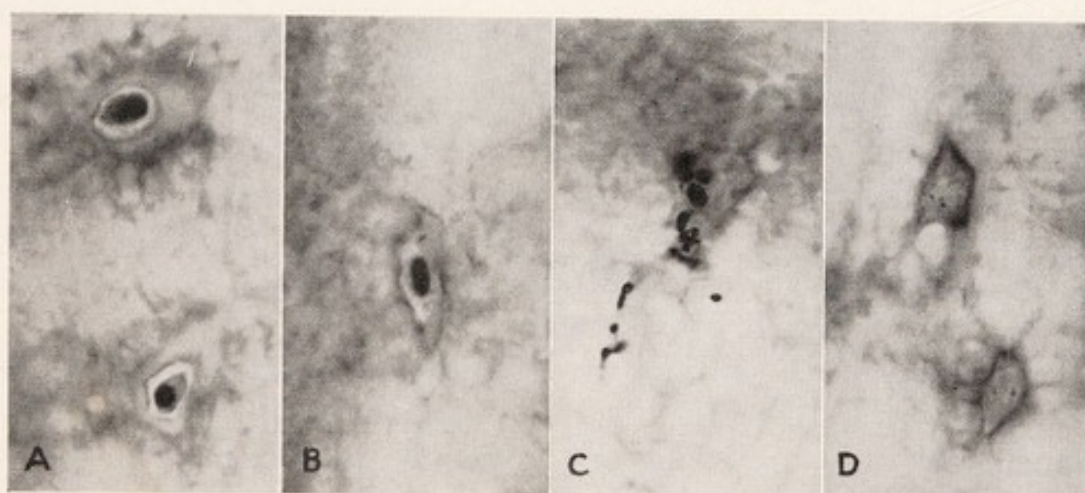


FIG. 177.—High magnification of cementum corpuscles in different layers of Fig. 176. A, near the surface of the cementum (Fig. 176, a): normal cementum cells showing nucleus and cell body; B, slightly deeper (Fig. 176, b): beginning shrinkage of cementum cells; C, still deeper layer (Fig. 176, c): disintegration of cell; droplets of fat and protoplasm in lacuna and canaliculi; D, in the deepest layers of cementum (Fig. 176, d): lacunæ containing cell debris.

have disintegrated; remnants of the nucleus and fat granules are found in the lacuna and in some of the surrounding canaliculi (Fig. 177, C). Finally, in the deepest layer of cementum, the cells

have completely disappeared; the lacunæ are empty except for some fine, dust-like débris (Fig. 177, *D*). Thus the changing condition of the cells in various depths indicates the high vitality of the young, superficial layers of cementum, and the low vitality of the deeper layers. The oldest cementum in the deepest strata has lost all of its vitality; it acts merely as mechanical support.

In primary cementum the vitality changes cannot be demonstrated by changes in the cells; however, here also advancing calcification and concurrent biological deterioration are revealed by the progressive calcification and disappearance of the embedded fibers of the periodontal membrane.

The space necessary for the deposition of cementum on the root surface is created by the minute bone resorptions constantly occurring in the socket of every tooth during function. Slight excess of functional stress exerted upon the crown causes resorption of the alveolar bone; the result is a widening of the periodontal space, which is partly compensated for by subsequent new formation of cementum. Another way in which space is created for cementum deposition is by the gradual occlusal movement of all teeth during life. This movement results in a widening of the periodontal space, especially at the apex and in the bifurcation in case of multirooted teeth; cementum deposition in these areas reduces the periodontal space to its normal width. Cementum formation as compensation for the occlusal movement of a tooth was observed as far back as 1909, when Loos called attention to the relative thickness of the cementum of extruding teeth without antagonists. In such teeth functional stimuli are no doubt much less significant than in teeth in occlusion. Therefore, the only way to account for these cementum deposits is to consider them a reparative or defensive measure against rapid occlusal displacement.

With these considerations we enter into a subject of great practical importance, namely, the rôle of cementum in repairing any damage or injury to which a root may be subjected. Cementum covers injured areas, reestablishes severed function, and reunites parts that were separated by a trauma (fracture).

One of the common forms of injury to the root surface is shallow resorptions due to excessive occlusal stress. Such resorptions are encountered especially in the teeth of individuals who make good use of their teeth; such people grind their teeth, or bite on some hard body with such force as to cause damage to the periodontal membrane and to produce circumscribed resorption of the root surface. These small damages are invariably repaired after the

injured periodontal membrane has recovered. New cementum is deposited upon the original cementum or the dentin in the resorbed area; new Sharpey's fibers are embedded into this cementum, and thus the damaged portion of the root surface is restored to its normal functional properties. The reparative cementum may be either primary or secondary cementum, or both types may take part in the repair of the resorbed area. In resorptions in the apical portion of the root, secondary cementum will most commonly be

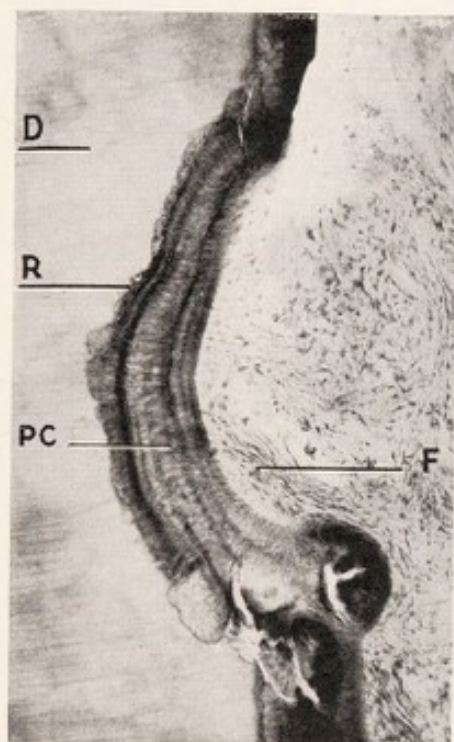


FIG. 178.—Repaired resorption of the root surface. The reparative cementum is primary cementum. D, dentin; R, line of resorption in the dentin; PC, primary cementum in the resorbed area; F, Sharpey's fibers embedded into the regenerated cementum surface.



FIG. 179.—Repaired resorption of the root surface. The reparative cementum is secondary cementum. D, dentin; R, line of resorption in the dentin; PC, primary cementum on the original root surface; SC, secondary cementum in the resorbed area; F, Sharpey's fibers embedded into the secondary cementum.

found; in the coronal portion of the root, either primary or secondary cementum may be deposited. In many instances, primary cementum is deposited directly upon the resorbed root surface (Fig. 178). In other cases, cell-containing secondary cementum is deposited first, and the regenerated fiber bundles of the periodontal membrane become embedded in the reparative cementum (Fig. 179). Again, in other instances, secondary cementum is the first hard tissue to be formed in the resorbed area, a layer of primary cementum being deposited upon the secondary cementum (Fig. 180).

From the viewpoint of restoration of function in the damaged area, either form of repair seems to be satisfactory (see Figs. 210 and 305).

Another very important occasion for reparative cementum formation is injury to the root end during root canal operation. Here, due to the location, cell-containing secondary cementum is most frequently found. The periodontal membrane reacts, in the absence of infection, to the removal of a pulp and to the insertion of a foreign material into the root canal by depositing cementum, which finally covers root filling material, pulp remnants and ramifications as a solid callus and causes healing of the apical wound. One

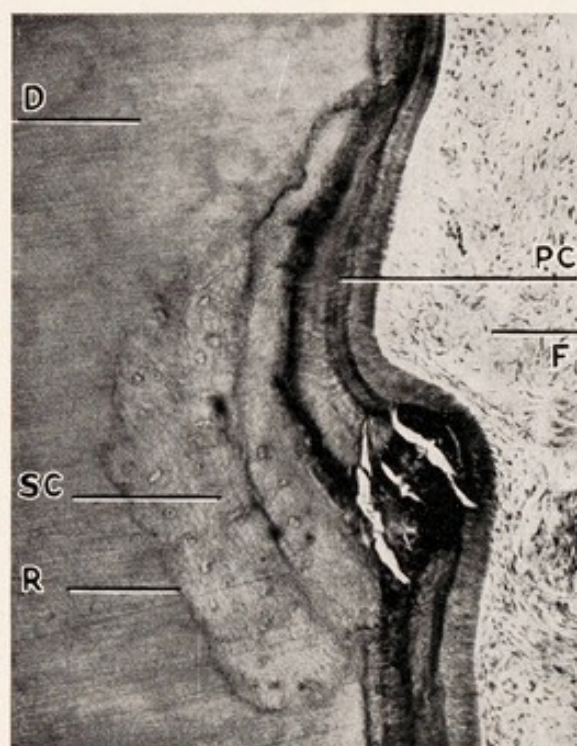


FIG. 180.—Repaired resorption of the root surface. Repair first by secondary, then by primary cementum. D, dentin; R, line of resorption in the dentin; SC, secondary cementum deposited upon the resorbed dentin surface; PC, primary cementum deposited on top of the secondary cementum; F, fiber bundles embedded into the primary cementum.

interesting observation along this line is the possibility of cementum deposition directly upon the surface of a foreign body, such as guttapercha (Figs. 157 and 159); this condition must, in a biological sense, be looked upon as the ideal outcome and as the final goal in every pulp canal operation (see Chapter VII).

The rôle of cementum in tooth fractures will be discussed in detail in Chapter XVI. If the continuity of the root has been severed by a trauma, the periodontal membrane will deposit cementum upon the fractured surfaces, and if the circumstances are favorable a reunion will result.

EXCESSIVE FORMATION OF CEMENTUM.

Abnormal or excessive formation of cementum can be divided into two groups: cementicles and cementum hyperplasias.

1. **Cementicles.**—Cementicles are calcified bodies found in the periodontal membrane of the teeth of adults. They are usually small and seldom measure more than 0.3 to 0.4 mm. In the periodontal membrane of some teeth they are very numerous, in others entirely absent. The development of cementicles occurs close to the epithelial rests in the periodontal membrane, and frequently epithelial cells can be observed near the cementicles (Fig. 181). In these cases cementicles seem to form by the deposition of cemen-

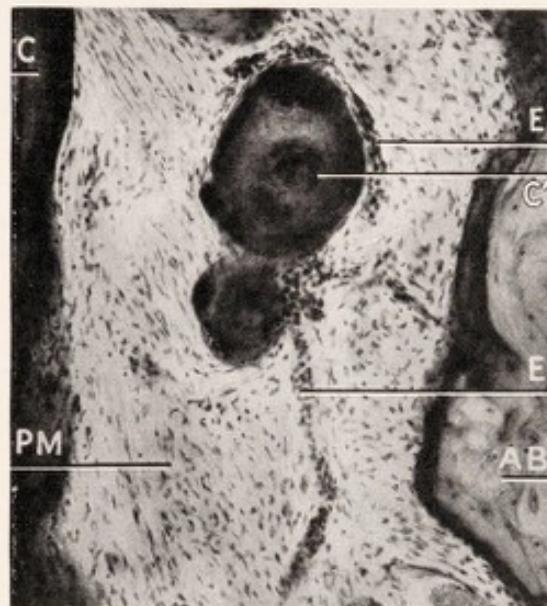


FIG. 181.—Cementicle in the periodontal membrane. C, cementum on the root surface; PM, periodontal membrane; C', cementicle lying free in the periodontal membrane; E, epithelial strands near and around the cementicle; AB, alveolar bone.

tum over degenerating or dead epithelial cells (Gottlieb). The close relationship of cementicles and epithelium is also corroborated by the common occurrence of cementicles in the neighborhood of enamel drops. These atypical enamel formations are covered by a layer of epithelial cells which gradually degenerate. The dying cells form centers of calcification around which cementicles develop (Fig. 182).

If cementicles are very numerous in the periodontal membrane of a tooth and lie close to the root surface, they may become adherent to the latter by the advancing formation of cementum, resulting in a peculiar, ragged appearance of the root surface (Fig. 183). Each one of these small prominences of the cementum (cementum

exostosis) is a cementicle of typical concentric structure that originally lay free in the periodontal membrane and later became united with the root surface.



FIG. 182.—Cementicles in the proximity of an enamel drop in the bifurcation of a lower molar. D, dentin; ED, enamel drop; C, cementum; CT, fibrous connective tissue; C', groups of small cementicles accumulated around degenerating epithelial cells.

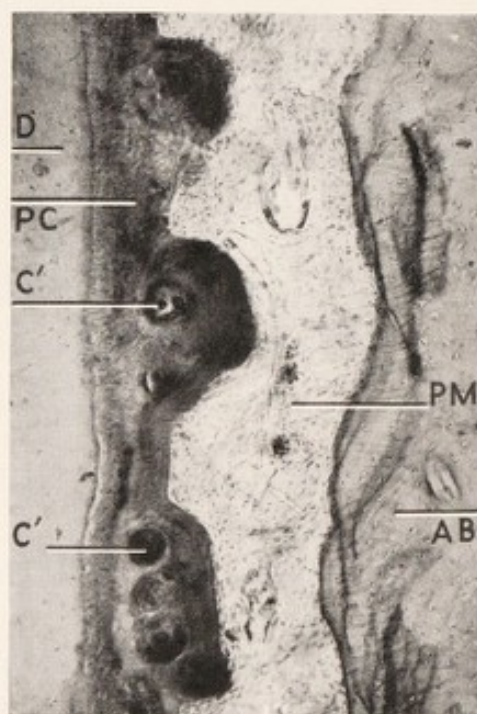


FIG. 183.—Adherent cementicles on the root surface of a lower molar. D, dentin; PC, primary cementum; C', cementicles of concentric lamellated structure embedded in primary cementum; PM, periodontal membrane; AB, alveolar bone.

2. Cementum Hyperplasia (Hypercementosis).—Cementum hyperplasia can be defined as an excess formation of cementum, either in a circumscribed area of the root, or on its entire surface. The first

type, circumscribed cementum hyperplasias, is very small, and is usually the result of calcification of the periodontal fiber bundles near their points of attachment. They are occasionally found on the surfaces of the roots of teeth that are subjected to considerable functional stress; under these circumstances the prominences or spike-like cementum hyperplasias to which the fiber bundles are attached may be compared with the bony projections or exostoses found at the point of attachment of tendons. Gottlieb found this type of cementum formation on the root ends of two lower incisors of an elderly man who used to carry a heavy pipe hanging on these two teeth (Fig. 184). It must be said, however, that such spike-like hyperplasias are by no means a frequent occurrence.



FIG. 184.—Apex of a lower lateral incisor that was subjected to heavy occlusal stress over a long period of time. Spike-like cementum hyperplasias (CH) arranged in the direction of Sharpey's fibers. (Gottlieb, *Jour. Am. Dent. Assn.*)

Much more common is the other form of cementum hyperplasia or hypercementosis, which consists of a diffuse thickening of the cementum over a large portion of the root surface (Fig. 185). The clinical significance of this condition has been the subject of a great deal of consideration, and, as we shall see, of unsubstantiated speculation. It is the author's opinion that hypercementosis is never a harmful or objectionable condition. Clinically, hypercementosis may be found either in all teeth of a mouth or in some teeth, especially in those without vital pulps. In the first case, generalized hypercementosis, evidently a congenital tendency toward abundant deposition of cementum is present. An observation by Zemsky

corroborates this theory. Three members of one family, a mother and two adult daughters, showed radiographically distinct hypertrophy of cementum on the roots of otherwise intact teeth. But to go straight to the point in this question: How are we able to diagnose hypercementosis? The prefix hyper indicates that more than a normal amount of something is present. But what is normal in this case? Are we able to say how thick normal cementum should be so that we may be justified in diagnosing a deviation from this norm? A survey of a number of teeth of different ages and different kinds will reveal wide variations in the thickness of the cementum. What, then, is normal? The author does not believe that any definite figures can be given for the thickness of the cementum. Hence, the diagnosis hypercementosis must be

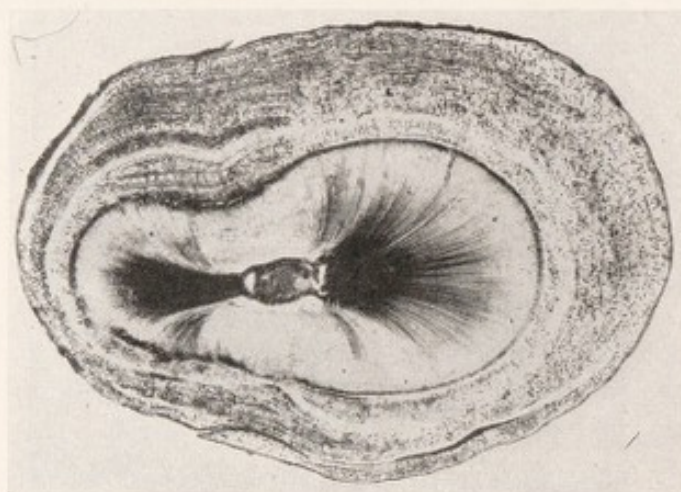


FIG. 185.—Cross section through the root end of an intact lower molar with cementum hyperplasia (ground section). Notice the regular undisturbed arrangement of the successive layers of cementum on the root surface.

made with caution and with the understanding that it is merely relative: if all roots in a jaw appear thin and delicate and only one or two teeth show heavy, bulky roots, we may say that these teeth present hypercementosis; but if all roots in this mouth were thick and bulky, the tooth or teeth under consideration would not differ from the rest of the set. Then all we can say is that this individual has teeth with thicker cementum than the average.

Cementum hyperplasia in pulpless teeth has a distinct clinical significance: it indicates a defense reaction of the organism. If a low-grade irritation, originating from within the pulpless tooth, stimulates and irritates the periodontal membrane over a long period of time, the tissue in some cases tries to ward off this irritation by laying a calcified barricade over the root end. The

result is a relative hypercementosis, a tooth the cementum of which is thicker than that of any of the adjacent teeth with vital pulps. The space for the formation of cementum is in this case created by the inflammatory destruction of the alveolar bone. Such a hypercementosis is not in itself a pathological condition; it is merely a reparative and protective attempt of nature, and, as such, a useful reaction. If a pulpless tooth with hypertrophy of the cementum were to be extracted, it should be understood that the extraction must be indicated by the infection of the root canal and not by the cementum hyperplasia.

A great number of statements made in dental literature refer to possible dangers and consequences of hypercementosis. In reading such data one is led to believe that many of them are simply repetitions from previous sources and have been taken over without due criticism or control. When making such a statement as that blindness, deafness, paralysis, or insanity might be caused by "hypercementosed" teeth and might be relieved by the removal of such teeth, one should expect positive proof of at least one case. Up to this time, the author has never seen or heard of any such proof; all that has been seen are some very general statements that are copied over again and again. Just why an increase in the amount of calcified tissue on the surface of a tooth should be responsible for severe disturbances of the central nervous system is hard to conceive. In any case of callus formation following bone fracture ten times as much new calcified substance is formed as in all hypercementosed teeth together, and yet nobody has seen in such a callus the etiological factor for insanity.

As far as the etiology of hypercementosis is concerned, our knowledge is rather limited. Besides the already mentioned congenital or hereditary factor, there is really only one condition the relationship of which to some forms of hypercementosis is well established: this is chronic, low-grade inflammation at the root ends of infected pulpless teeth with good tissue defense. In such teeth we frequently find a marked hypertrophy of cementum directly above the area of the apex that is included in the inflammatory process (Fig. 186). The inflammation appears to be the stimulus for cementum formation; in a biological sense the resulting increase of root surface may be considered as compensation for the loss of attachment at the apex. The inflammatory process destroys the alveolar bone and the apical fibers and thus weakens the tooth, and the newly built cementum provides a new vital surface for the attachment of additional periodontal fibers.

Functional stimuli do not seem to be of especial importance in the development of cementum hyperplasias. Despite contrary statements found in dental literature, increased function does not cause increased thickness of the cementum; the only exception is the occasional microscopic cementum spikes on functioning teeth. Kellner made comparative measurements of the thickness of cementum on corresponding functioning and non-functioning teeth of the same jaw. He found thicker cementum in non-functioning teeth than in those having antagonists. The most striking com-

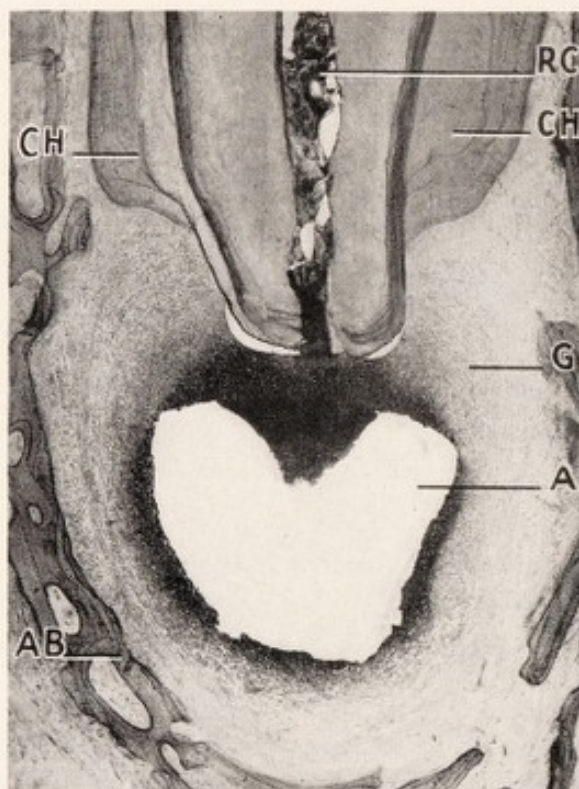


FIG. 186.—Hyperplasia of cementum in the proximity of chronic periapical inflammation. Upper bicuspid. CH, cementum hyperplasia; RC, root canal containing necrotic pulp remnants; G, granuloma; A, abscess cavity containing a purulent exudate; AB, alveolar bone. (Courtesy of Wm. G. Skillen.)

parison of this type was made between a completely impacted lower bicuspid and the corresponding lower bicuspid on the other side which was in normal occlusion: the impacted tooth had thicker cementum than the erupted one. Unerupted and malposed teeth frequently have very thick layers of cementum. These observations indicate that function is not of primary importance in hypercementosis.

The changed concept concerning hypercementosis is best expressed in the recently published clinical study carried out by Boyd S. Gardner and Harold Goldstein on the large amount of material

available at the Mayo Clinic. The authors reported the following results of their investigation: Of 529 teeth with hypercementosis, 428 were vital and 101 non-vital, or, in other words, 4.2 vital teeth were involved for every non-vital tooth. From these findings, Gardner and Goldstein draw the following important conclusions concerning the etiology of hypercementosis: "This apparently limits the force of any reference to the factor of infection playing a part in the etiology of the hyperplasia, although occasionally we encountered, in examination of non-vital teeth, zones of rarefaction associated with apical hypercementosis. But in each case in which this phenomenon was presented, other vital teeth with hypercementosis were found, pointing to the likelihood of an inherent tendency of certain persons rather than to a hyperplastic response to infection only." In other words, cementum hyperplasia is non-pathogenic, and is merely indicative of an individual tendency toward overproduction of cementum in certain teeth. Hence, the authors come to the practical conclusion that "extraction of vital teeth with hypercementosis, in the hope of relieving systemic pathologic conditions, as often practised by dentists, is contraindicated."

BIBLIOGRAPHY.

- BAUER, W.: Über Zementikel und Zementikelähnliche Einlagerungen in den Wurzelhaut, *Vrtljschr. f. Zhk.*, 1929, **45**, 345.
- BOEDECKER, CHARLES F.: A Consideration of Some of the Changes in Teeth from Youth to Old Age, *Dental Cosmos*, 1925, **67**, 543.
- COOLIDGE, EDGAR D.: The Reaction of Cementum in the Presence of Injury and Infection, *Jour. Am. Dent. Assn.*, 1931, **18**, 499.
- DEWEY, K. W.: Normal and Pathological Cementum Formation, *Dental Cosmos*, 1926, **68**, 560.
- EULER, HERMANN: Zementhypertrophie, *Handw. d. ges. Zhk.*, vol. **4**, p. 3309.
- GARDNER, BOYD S., and GOLDSTEIN, HAROLD: The Significance of Hypercementosis, *Dental Cosmos*, 1931, **73**, 1065.
- GOTTLIEB, B.: Histologische Befunde an umgelegten Wurzeln, *Ztschr. f. Stom.*, 1921, **19**, 1.
- Zementexostosen, Schmelztropfen und Epithelnester, *Ztschr. f. Stom.*, 1921, **19**, 565.
- Tissue Changes in Pyorrhea, *Jour. Am. Dent. Assn.*, 1927, **14**, 2178.
- KELLNER, ERNST: Das Verhältnis der Zement- und Periodontalbreiten zur funktionellen Beanspruchung der Zähne, *Ztschr. f. Stom.*, 1931, **29**, 44.
- KRONFELD, RUDOLF: Die Zementhyperplasien an nichtfunktionierenden Zähnen, *Ztschr. f. Stom.*, 1927, **25**, 1218.
- Zement und Sharpey'sche Fasern, *Ztschr. f. Stom.*, 1928, **26**, 714.
- LOOS, O.: Über die Ursachen des sogenannten Längerwerdens der Zähne bei fehlenden Antagonisten, *Strassburg, Heitz*, 1909.
- MEYER, W.: Die Vitalität des Zementes, *Vrtljschr. f. Zhk.*, 1927, **43**, 488.
- Histologie des Zementes, *Handw. d. ges. Zhk.*, vol. **4**, p. 3299.

- RYWKIND, A.: Zur Frage der Zementikelbildung, *Ztschr. f. Stom.*, 1930, **28**, 1178.
- SIPPY, B. O.: Regeneration of Tissues Following Experimental Injury of Tooth Roots, *Dental Cosmos*, 1927, **69**, 771.
- SPRING, K.: Beitrag zur Kenntnis der Exzementosen, *Ztschr. f. Stom.*, 1930, **28**, 972.
- WEBER, RUDOLF: Über die feineren Vorgänge bei der Histogenese des Zementes, *Deutsch. Mon. f. Zhk.*, 1925, **43**, 217.
- ZEMSKY, JAMES L.: Hypercementosis in Relation to Unerupted and Malposed Teeth: A Preliminary Report, *Jour. Dent. Res.*, 1931, **11**, 159.
- Hypercementosis and Heredity: An Introduction and Plan of Investigation, *Dent. Items Int.*, 1931, **53**, 355.

CHAPTER IX.

ROOT RESORPTION.

GENERAL CONSIDERATIONS CONCERNING HARD TISSUE RESORPTION.¹

NORMALLY, hard substances are found in the human body in only two structures: bone and teeth. These calcified tissues are not all of the same origin. Bone, dentin, and cementum originate from the mesoderm, enamel from the ectoderm.

The formation of the mesodermic parts of the teeth (dentin, cementum) and of the bone takes place through the formation of an organic matrix and subsequent deposit of mineral salts into this matrix, giving to the hard substances their definite, unyielding form and structure. The reverse process, however, the elimination of hard substances, takes place in an entirely different way. Formerly it was the belief that a process of decalcification (halisteresis) normally initiates the elimination of hard tissues, which would mean that first the inorganic salts are removed and dissolved away from the matrix, and then the organic matrix itself disappears. The investigations of Koelliker and others, however, have definitely established the fact that the resorptive process on the hard tissues, teeth as well as bone, is always characterized by the presence of special cells, called osteoclasts, that seem to eliminate the calcified tissue at once without any previous decalcification. True decalcification (halisteresis) occurs only in severe bone diseases, such as osteomalacia.

The origin of the osteoclasts is still a much discussed question. There are two possible sources from which they may develop, either from the capillaries in the connective tissue adjacent to the

¹ In modern terminology the term "resorption" is used exclusively for the elimination of hard tissues by cellular activity, instead of the old term "absorption." As indicated by the prefix, *re*, resorption means that some structure or substance that is formed or built by the body is taken back into the organism. This is actually true of teeth and bone which are built by the body, and in case of resorption are dissolved and taken back into the general circulation. Absorption, on the other hand, would mean that some new, external material or substance is sucked up or taken into something. It is, therefore, correct to say that moisture is absorbed by cotton or that a drug is absorbed by the intestinal mucosa. (See also Becks and Marshall, Jour. Am. Dent. Assn., 1932, 19, 1528.)

hard substance, or from the connective tissue itself by the transformation of osteogenic cells or undifferentiated mesenchyme cells into osteoclasts. Circumstances seem to determine which of these two sources shall furnish the osteoclasts. Morphologically, osteoclasts appear in the tissues either as polynuclear or as mononuclear cells. Polynuclear osteoclasts, or giant cells, contain anywhere from two or three to a few dozen nuclei which are usually clustered in the centers of the large protoplasmic cell bodies (Fig. 187). These osteoclasts, by their resorbing activity, cause bay-like excavations in the hard substance, Howship's lacunæ. Mononuclear osteoclasts are small spindle-shaped cells resembling osteoblasts; they are found in shallow indentations along the hard substance surface

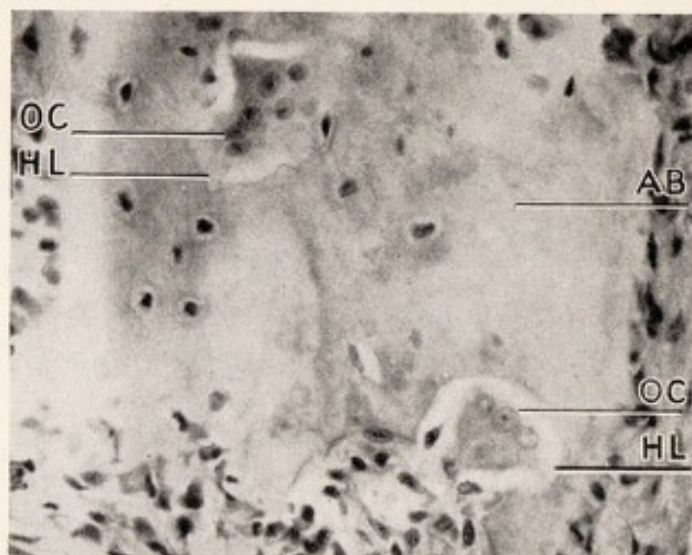


FIG. 187.—Polynuclear osteoclasts (giant cells) resorbing the alveolar bone. OC, osteoclast; AB, alveolar bone; HL, depression in the bone surface caused by the resorbing action of the giant cell (Howship's lacunæ).

(Fig. 188). It is impossible to determine just what the difference is in the action of polynuclear and mononuclear osteoclasts, or why in some instances or areas one type of osteoclasts is encountered and, in others, the other type. It seems, however, that the resorbing action of polynuclear osteoclasts (giant cells) upon hard tissues is faster and more effective than that of the mononuclear cells; at least, giant-cell osteoclasts will be found to be the prevailing type whenever rapid and extensive resorption of hard tissue occurs.

There is apparently no difference between the mechanism of bone, dentin, cementum, and enamel resorption (Fig. 189). There is, however, one basic biological difference between the resorptive processes in bone and teeth: in bone, both new formation and resorption occur as a physiological process throughout life; in teeth,

physiologically, only new formation takes place. All resorption in teeth is the expression of a pathological process or injury, the only exception to this rule being the resorption of the deciduous teeth during shedding. In bone, under physiological conditions, through-

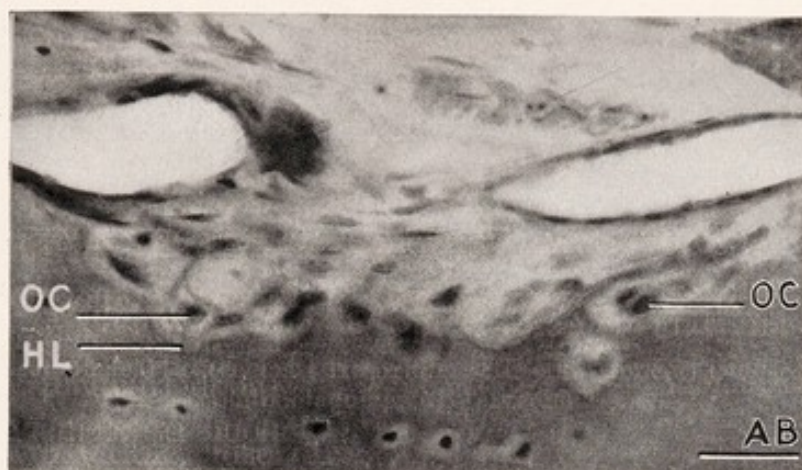


FIG. 188.—Mononuclear osteoclasts resorbing alveolar bone. OC, osteoclasts; HL, Howship's lacunae; AB, alveolar bone.

out life there will always be tearing down in some places and compensatory formation of new bone in others. In the teeth, on the contrary, no resorption takes place normally, but a continuous new formation of hard substances occurs. The root surface is covered by additional new layers of cementum, thus steadily increas-

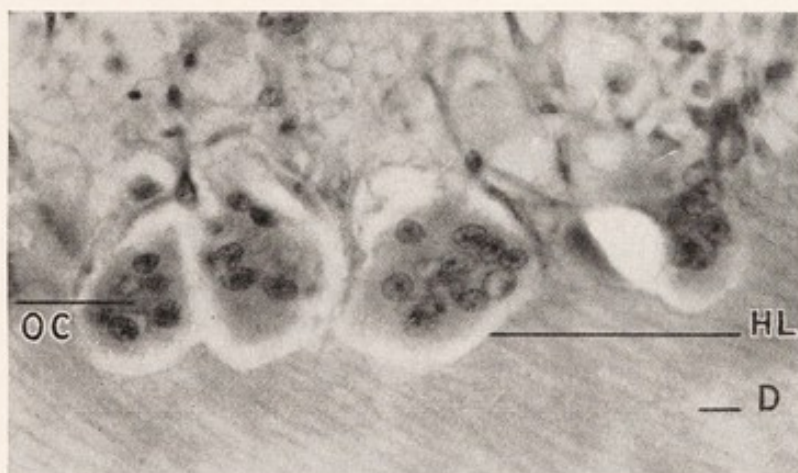


FIG. 189.—Polynuclear osteoclasts (giant cells) resorbing dentin. D, dentin; OC, osteoclasts with numerous nuclei; HL, Howship's lacunae in the dentin.

ing the total amount of hard tissue present. A similar continuous deposition of dentin occurs inside the tooth causing a constant decrease in the size of the pulp chamber.

In conclusion it may be stated that, in examining a specimen of

normal bone taken at random from any part of the body, new formation and resorption will always be found occurring simultaneously. In a healthy permanent tooth, on the other hand, new formation only will be found on the outside of the tooth (cementum) as well as on the inside (dentin). Every microscopic evidence of active tooth resorption indicates the presence of pathological interference; every repaired tooth resorption indicates that a pathological process was present in the past.

PHYSIOLOGICAL RESORPTIVE PROCESS ASSOCIATED WITH THE SHEDDING OF THE DECIDUOUS TEETH.

The roots of the deciduous incisors, as a rule, reach their full development at the age of two years, the roots of the cuspids and molars usually about a year later. At about the age of four years, the developing permanent germs lying above and between the roots of the deciduous teeth begin to cause resorption of these roots.

In saying "the permanent teeth cause resorption of the deciduous roots" it must, of course, be understood that a permanent tooth itself has no resorptive properties. A permanent tooth moving occlusally exerts a slight pressure upon the connective tissue that surrounds its crown; this stimulates resorptive processes in the hard structures (bone, deciduous roots) lying in the path of the growing permanent crown, and thus makes possible the progress of the moving tooth.

It seems advisable to consider the question of why, under these circumstances, only bone and deciduous root are resorbed and not the permanent tooth also. In fact, if the permanent tooth compresses the connective tissue between it and the deciduous root there is no mechanical reason evident why the permanent crown should not be attacked by the osteoclasts just as is the deciduous root. The answer to the question seems to be that the enamel epithelium covering the permanent crown prevents it from being in contact with connective tissue and thus protects it from resorbing forces. This opinion is supported by the observation that the enamel of impacted teeth, after the loss of the covering enamel epithelium, is attacked by resorption just as are the roots of the deciduous teeth (see Figs. 205 and 335).

The relative positions of an upper central deciduous incisor and a permanent incisor in a child, aged two and a half years, may be seen in Fig. 190. The deciduous incisor has erupted and its root is fully developed. The permanent germ, consisting only of enamel

and a thin shell of dentin, is located above and behind the deciduous tooth, just below the floor of the nasal cavity. The enamel, most of which has been lost in the preparation of the microscopic specimen, is covered by the enamel epithelium and is surrounded

by a layer of connective tissue. A thin layer of bone separates this tissue from the deciduous root.

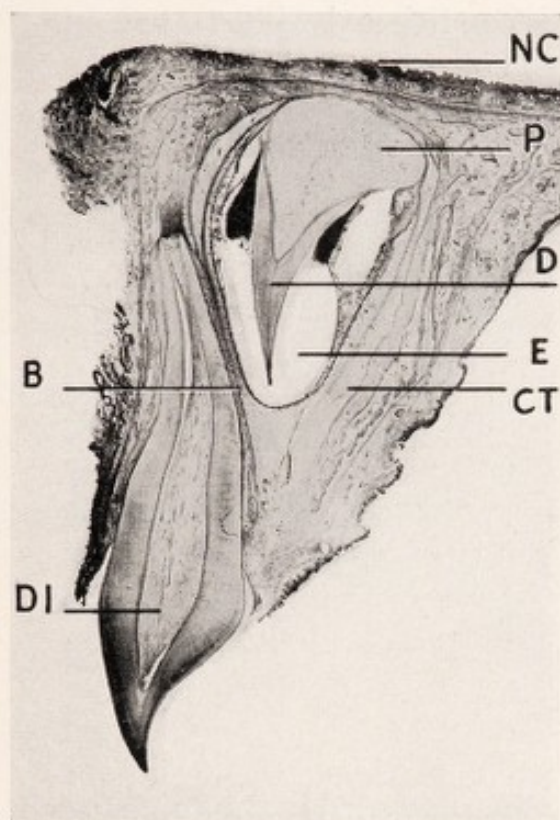


FIG. 190.—Deciduous and permanent upper central incisor. Labio-lingual section. Age, eighteen months. DI, deciduous incisor; P, pulp of permanent incisor; D, dentin of permanent incisor; E, enamel of permanent incisor; CT, connective tissue capsule surrounding the permanent germ; B, thin plate of bone separating deciduous and permanent tooth; NC, floor of nasal cavity.

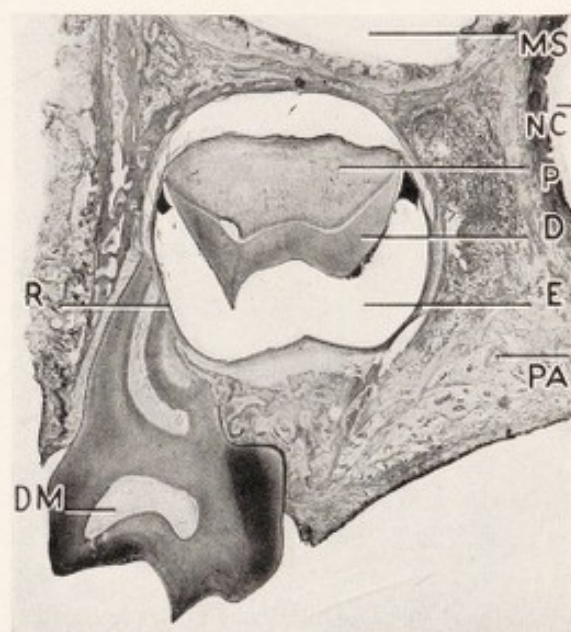


FIG. 191.¹—Resorption of the mesio-buccal root of an upper deciduous second molar. Bucco-lingual section. Human specimen; age, four and one-half years. DM, second deciduous molar; P, pulp of the bicuspid germ; D, dentin of the bicuspid germ; E, enamel of the bicuspid germ; R, resorption of the root end of the deciduous molar; NC, nasal cavity; MS, maxillary sinus; PA, palatine artery.

The resorption of the deciduous root usually begins on the side facing the permanent successor. The resorption of the root of a deciduous tooth is shown in Fig. 191, taken of an upper second deciduous molar and upper second bicuspid germ in a child, aged four and one-half years. The bicuspid germ at this age is found lying between the roots of the deciduous molars and below the floor of the maxillary sinus. The mesio-buccal root that is reproduced here is considerably shortened by resorption; the opening of the root canal is adjacent to the enamel epithelium of the bicuspid crown.

¹ Figs. 191–198 reprinted from Kronfeld, *The Resorption of the Roots of Deciduous Teeth*, Proceedings of the Eighth Int. Dental Congress, August, 1931.

As the permanent tooth elongates and moves toward the occlusal, the deciduous roots gradually become shorter until, finally, the

deciduous crown only is left, superficially attached to the gingivæ. Such a condition is illustrated in Fig. 192, taken of the lower cuspid in a child, aged eight years. A higher magnification shows the lower surface of the crown densely beset with osteoclasts that are resorbing the dentin (Fig. 193). The pulp is intact. The space between the enamel epithelium of the permanent cuspid and the dentin of the deciduous teeth is occupied by a highly vascularized granulation tissue ("absorbent organ" of J. Tomes); after the final elimination of the deciduous crown, this granulation tissue is usually found as a bright red, irregular, easily bleeding tissue in the space formerly occupied by the deciduous tooth.

Fig. 192 shows a rather unusual arrangement of the bone trabeculæ underneath the permanent cuspid. In a higher magnification of these parallel rows of bone, it is apparent that each trabecula is resorbed on the lower surface; whereas, new bone is formed on the upper side, facing the erupting permanent tooth. This distribution of resorption and

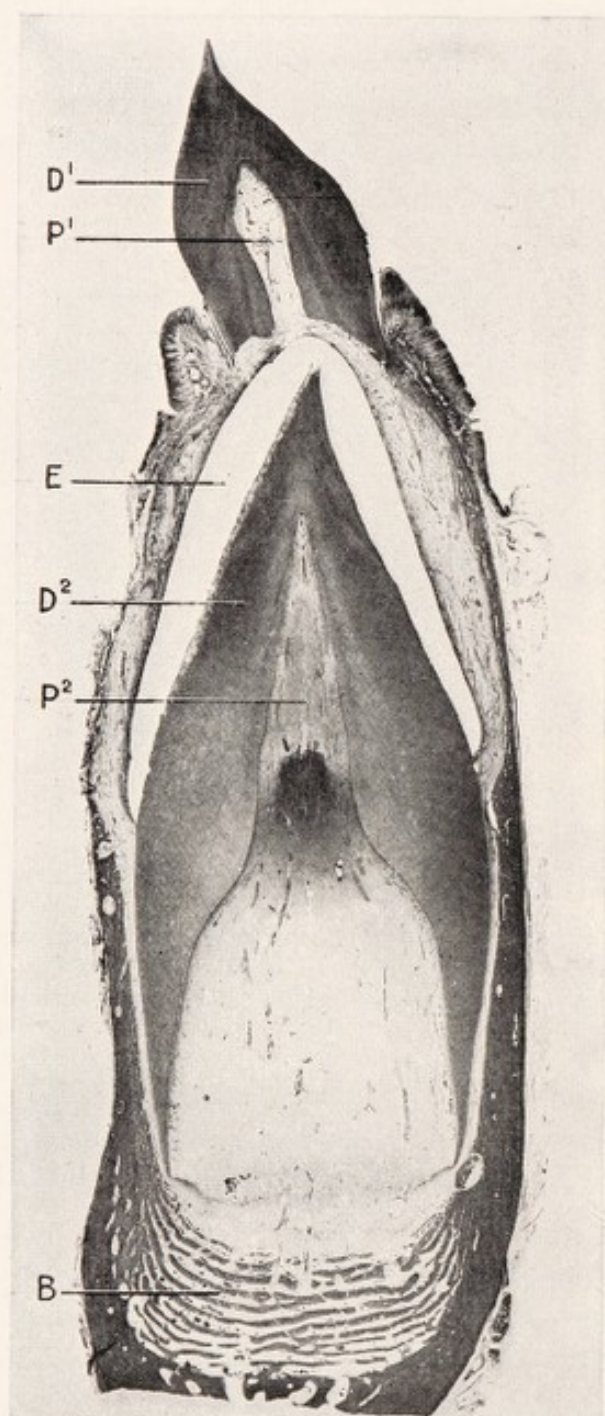


FIG. 192.—Lower deciduous and permanent cuspid. Labio-lingual section. Human, aged eight years. D¹, dentin of the deciduous cuspid; P¹, pulp of the deciduous cuspid; E, enamel of the permanent cuspid; D², dentin of the permanent cuspid; P², pulp of the permanent cuspid; B, rows of bone beneath the apical opening of the permanent cuspid.

new formation indicates that the permanent cuspid moves occlusally, away from the fundus of the alveolus, and that new trabeculae of bone arranged in parallel rows fill the space behind the moving tooth. This finding corroborates the investigations of Orban concerning the occlusal movement of tooth germs during their development; it shows that the old conception of the root's growing into



FIG. 193.—Higher magnification of Fig. 192. E, enamel of the permanent cuspid; D, dentin of the permanent cuspid; P, pulp of the deciduous cuspid; R, resorption on the lower surface of the deciduous tooth; EA, proliferation of the epithelial attachment along the surface of the deciduous root.

the underlying bone is incorrect since the entire tooth, together with the lengthening root, moves occlusally.

There are several points of interest in the resorption of deciduous teeth. One of these is the observation that this process does not proceed continuously from the periphery of the root toward the pulp; that part of the dentin, calcified and uncalcified (dentinoid), immediately surrounding the pulp seems to be more resistant to

resorption than the rest of the dentin. Its higher resistance can sometimes be observed in the radiograph.

J. Tomes made this observation and wrote: "The cementum is usually attacked first, but eventually dentin, and even enamel come to be scooped out and removed by an extension of the process. That part of the dentin, however, which immediately surrounds the pulp appears to have more power of resistance than any other part of the tooth, and thus often persists for a time as a sort of hollow column." In the last few years this observation has been the point of issue for a discussion as to whether or not the condition of a hard tissue itself has something to do with the way in which resorption occurs. Several observations have been published that seem to indicate that the better calcified a substance is the easier it is resorbed; *vice versa*, poorly calcified or still uncalcified hard tissue seem to offer more resistance to resorptive processes. In Fig. 213, the dentin nearest to the pulp was formed last, and is still uncalcified or was only recently calcified. This "young" hard tissue seems to have a higher degree of vitality than the older, more calcified dentin, and this higher vitality has been held responsible for the increased resistance to resorption.

Another point of great practical and clinical importance is the observation that the process of eruption of the permanent teeth is interrupted by periods of inactivity or rest, during which intermissions the resorption of the deciduous teeth is also discontinued; then reparative processes take place on the resorbed deciduous teeth and alveolar bone. Tomes describes this phenomenon as follows: "The process of absorption once commenced does not necessarily proceed without intermission, but may give place for a time to actual deposition of osseous tissue on the very surface eroded. . . ." ". . . These alternations of absorption and deposition . . . often occur during the normal process of the removal of the deciduous teeth, and result in the deposition of a tissue not unlike cementum in excavations of the dentin, or even of the enamel." Oppenheim, who studied this problem on histological material from men and animals, found that during the eruptive phase both alveolar bone and deciduous tooth are resorbed to a larger extent than the actual movement of the permanent tooth would necessitate, and that this excess resorption is made up for by a reparative new formation of bone and cementum in the period of rest. During repair a temporary solid junction between bone and deciduous tooth can take place, which explains the great variations in the firmness of deciduous teeth; during the period of active

forward growth of the permanent teeth, the temporary teeth are rather loose; whereas, in the following period of inactivity, they may become firm again, sometimes even surprisingly firm if a solid bony junction has taken place.

An extensive bony junction between a lower deciduous second molar and the alveolar bone will be illustrated (Fig. 194). The

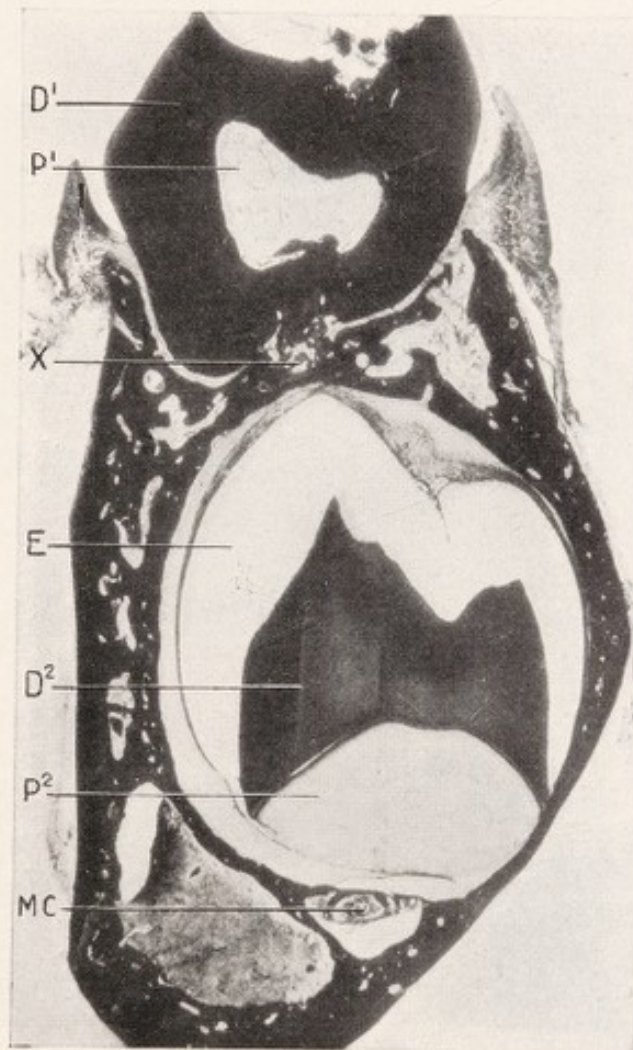


FIG. 194.—Bony union between a lower deciduous second molar and the alveolar bone. Bucco-lingual section. Human, aged eight and one-half years. D', dentin of the deciduous molar; P', pulp of the deciduous molar; E, enamel of the bicuspid germ; D², dentin of the bicuspid germ; P², pulp of the bicuspid germ; X, area of bony union; MC, mandibular canal.

specimen was obtained by autopsy from a boy, aged eight and one-half years. Bony union has taken place in the bifurcation of the roots, the resorption having been repaired and the tooth and bone having grown together; under higher magnification the original line of resorption can still be seen (Fig. 195). Clinically, a deciduous tooth in a condition like that illustrated in Fig. 194 would appear

very firm, perhaps even firmer than the neighboring teeth, since the latter show slight physiological motility; whereas, the deciduous tooth in consideration is actually ankylosed in its socket. If such bony ankylosis of a deciduous tooth develops to a great extent, it may even make normal exfoliation of the deciduous tooth and eruption of the permanent successor impossible.

The occurrence of impacted or submerged deciduous molars can also be explained by resorption and subsequent ankylosis with the



FIG. 195.—Higher magnification of area X in Fig. 194. D, dentin of the deciduous molar; B, newly-formed bone uniting tooth and jaw; AB, alveolar bone.

alveolar bone. Such teeth have ceased to move occlusally and remain below the occlusal plane of the other teeth. Occasionally they may form an obstacle to the eruption of the bicuspid; if so, they must be removed. (Noyes.)

New formation of bone and cementum during the phase of rest in the process of shedding is illustrated under high magnification in Fig. 196. The specimen was taken from a young monkey. In the lower part of the picture the outer enamel epithelium that covers the crown of the permanent tooth (bicuspid) is visible. The irregu-

lar line in the dentin indicates how far resorption of the deciduous tooth has gone. At the time of the animal's death, cell-containing cementum had been deposited on the resorbed dentin surface;

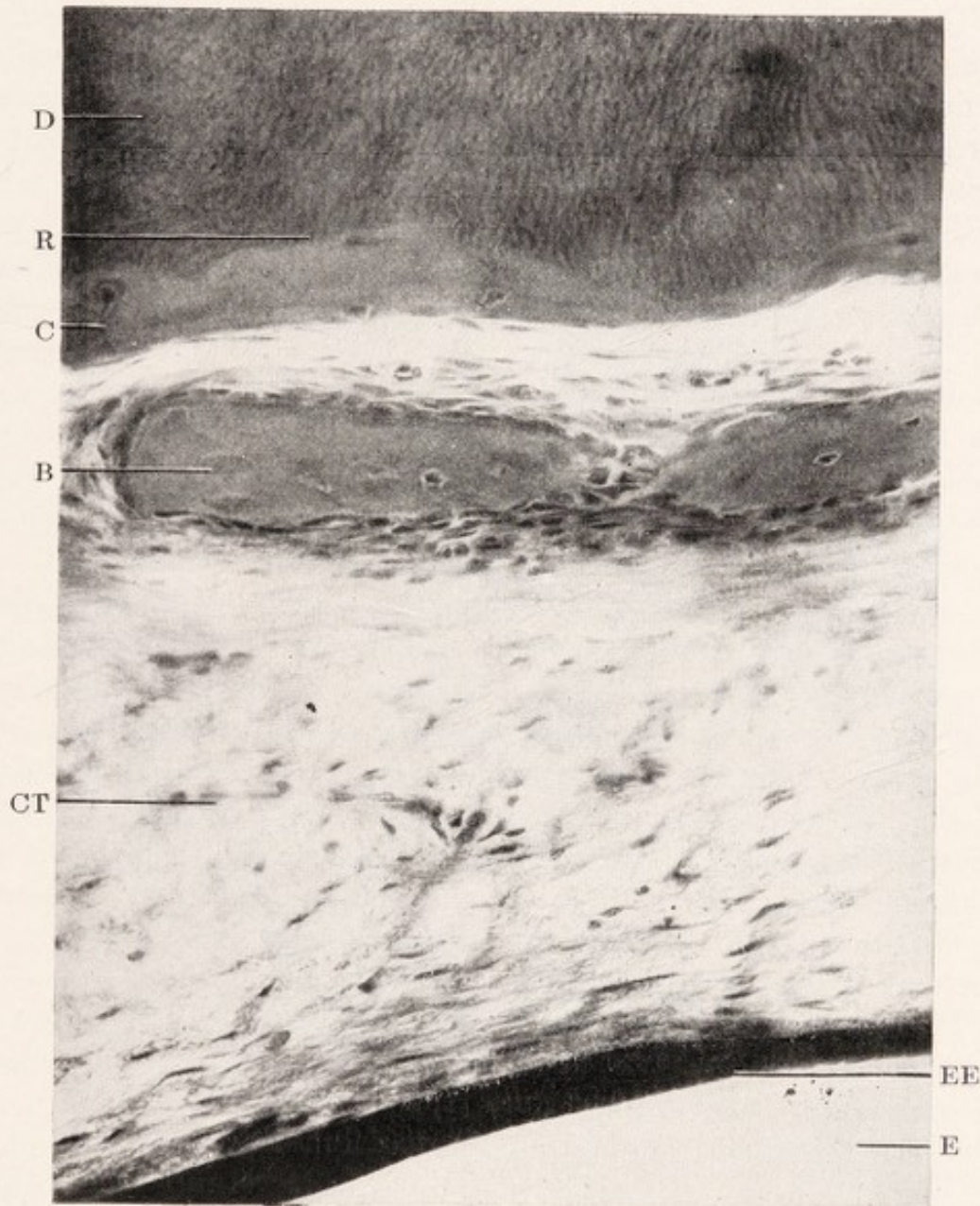


FIG. 196.—New formation of cementum and bone in the reparative phase during the shedding of a deciduous molar. Monkey. E, enamel; EE, enamel epithelium of permanent tooth; D, dentin of deciduous tooth; R, line of resorption of deciduous tooth; C, newly deposited cementum; B, newly formed bone surrounded by osteoblasts; CT, connective tissue separating deciduous and permanent tooth.

tender trabeculae of newly formed bone can be recognized in the connective tissue between deciduous and permanent tooth. It is reasonable to assume that in the next period of eruption of the permanent tooth both newly formed bone and cementum would

have been resorbed as well as a larger area of dentin. This might again have been followed by a period of rest and new formation. Resorption, however, is always more extensive than the following repair, so that finally the tooth is completely loosened and eliminated.

The participation of the pulp in the process of shedding the deciduous teeth is another important question. Several authors in dealing with this subject state that the pulp of the deciduous tooth forms osteoclasts that hollow the crown by resorbing the dentin from the inside of the tooth; if this were so, the pulp would take active part in the resorptive process. These publications were, as a rule, illustrated with specimens from animal jaws, especially dogs and cats. In these animals the pulp in the crown of the deciduous tooth appears, in some instances, completely beset with giant cells lying in Howship's lacunæ on the inside of the dentin, and the crown is actually hollowed from within. This process, however, cannot be observed in human deciduous teeth. In man, the pulp tissue itself does not participate in the resorption of deciduous teeth, but retains its histological characteristics as pulp and not as an "absorbent organ." Only in the last stages of resorption does granulation tissue sometimes proliferate into the deciduous crown and cause resorption on the inside of the pulp chamber. The osteoclasts are found only on the surface of the deciduous tooth that directly faces the permanent crown (see Fig. 193); the inside of the pulp chamber does not show any resorptive changes. The clinical significance of this observation is that deciduous teeth without vital pulps are resorbed and eliminated just as regularly as deciduous teeth with intact pulps, provided no infection is present.

The resorption of deciduous teeth with dead pulps and infected root canals is much slower and more irregular than the shedding of such teeth with vital healthy pulps. However, we do not need the active participation of the living pulp to explain this phenomenon. It is well known that infected necrotic hard tissues are very resistant to osteoclastic resorption; they are instead eliminated *in toto* like a sequestrum. In the same manner, infected deciduous roots are pushed out by the permanent successors instead of being gradually shortened by resorption.

Resorption of Deciduous Teeth Without Permanent Successors.—From the study of findings such as illustrated in Figs. 191 and 192, it is evident that resorption of the roots of deciduous teeth is in the first place controlled by and dependent upon the permanent successors. But this purely mechanical explanation is insufficient

to explain the resorptions of the roots of deciduous teeth without permanent successors. In these cases root resorption takes place without the stimulus of the permanent tooth; hence it must be concluded that not every resorption of a deciduous tooth is necessarily due to the action of the permanent successor.

A great variety of clinical observations has been made of retained deciduous teeth. In cases where the permanent successors are either impacted or not formed, some deciduous teeth, particularly



FIG. 197.—Radiographs of a patient, aged forty years, with retained lower second deciduous molars. No second bicuspid are present. Notice the areas of resorption on the mesial roots of the deciduous molars. (Courtesy of G. E. Morgan.)

cuspid and molars, are retained often for years in good functional condition. Again, such deciduous teeth become loose and are lost for no apparent reason; their roots are extensively resorbed, exactly like the normal manner of shedding deciduous teeth. It seems that the condition of the deciduous tooth itself decides the final outcome. Every root will be retained in its socket only as long as it has a vital cementum surface. As long as the deciduous tooth



FIG. 198.—Radiographs of a child, aged thirteen years, with retained lower deciduous molars. No second bicuspid are present. The roots appear to be considerably shortened by resorption. (Courtesy of G. E. Morgan.)

has such a surface, a good functional connection with the alveolus is assured. If small resorptions do occur in some places, they are readily repaired by new deposits of cementum. How long this favorable condition will last cannot be predicted (Figs. 197 and 198). In many cases of this type the vitality of the deciduous tooth seems to be lost in the second or third decade of life. No new deposits of new cementum take place on the root surface; incidental functional resorptions are no longer repaired, but instead the resorptive

process continues until the deciduous tooth is loosened and finally lost. These changes in the tooth's vitality are an expression of senescence, and as such are largely independent of outer influences.

In summing up the clinical and histological findings on the shedding of human deciduous teeth, the following conclusions can be drawn: In the first place, the resorption of the deciduous teeth is certainly stimulated by the growing and erupting permanent successors; the distribution of the areas of resorption depends upon the form and the position of the permanent crown. In addition, however, resorption of deciduous teeth is also observed in the absence of permanent successors, and, in that event, the biological condition of the deciduous tooth itself seems to decide the outcome of the resorptive process.

RESORPTIVE PROCESSES OCCURRING IN PERMANENT TEETH.

As has been mentioned in the beginning of this chapter, resorptive processes in permanent teeth are always the expression of some pathological interference. Several injurious factors may cause resorption of the teeth of the permanent set. These causes will be enumerated, and each group will be discussed both from the clinical and the histopathological viewpoint. The various groups of teeth occasionally subjected to resorption are:

1. Pulpless teeth.
2. Replanted teeth.
3. Impacted teeth.
4. Teeth in close proximity to tumors and cysts of the jaws.
5. Teeth exposed to excessive occlusal trauma.
6. Root resorption of unknown etiology or of constitutional origin. (Idiopathic root resorption.)

1. **Root Resorption in Pulpless Teeth.**—A rather frequent clinical observation, usually revealed by the radiograph, is that roots may be resorbed following necrosis of the pulp. In all of these cases, chronic periapical infection seems to be the etiological factor in the resorptive process. The resorption can begin either at the root end and involve the apical part of the root, or on the side of the root and progress toward the root canal.

A large percentage of pulpless teeth with chronic periapical infection shows a certain amount of resorption at the apex (Figs. 161 and 162). This type of resorption is, as a rule, not very extensive and often hardly visible in the radiograph. In case of success-

ful root canal treatment these areas of resorption are covered by a reparative deposit of new cementum.

A case of extensive root resorption was observed by Willman in a boy, aged fourteen years, whose upper left central incisor gradually became shorter than the neighboring teeth (Fig. 199). The radiograph revealed such extensive destruction of the root that there was little left besides the guttapercha root canal filling (Fig. 200).

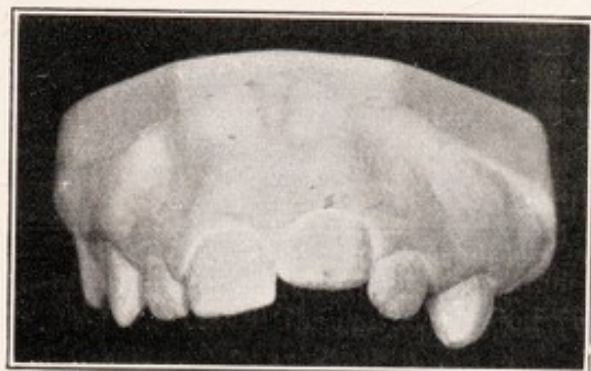


FIG. 199.—Plaster cast of the upper anterior teeth of a boy, aged fourteen years, showing the upper left central incisor approximately 2 mm. shorter than the neighboring teeth.



FIG. 200.—Radiograph of the upper central incisor. The pulp was removed and the canal filled two years before. At the present time most of the root has been destroyed by resorption.

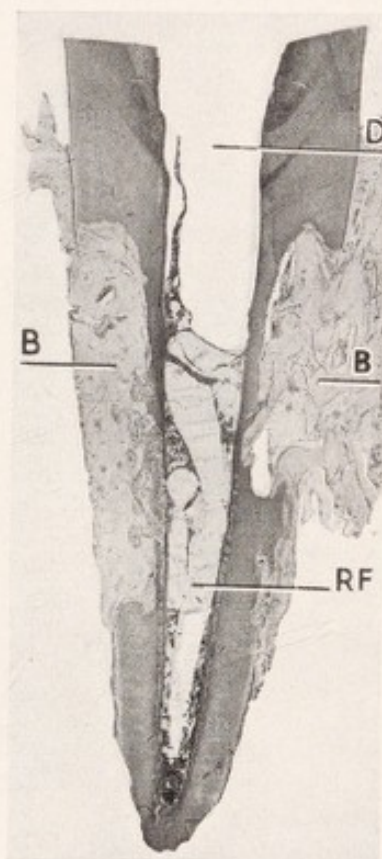


FIG. 201.—Mesio-distal section through the root of the upper left central incisor showing large areas of resorption with ingrowth of bone. B, bone; D, space formerly occupied by the dowel supporting the porcelain crown; RF, root canal filling. (Willman, Jour. Am. Dent. Assn.)

The microscopic examination of the removed specimen showed the amount of resorption of the root and the subsequent ingrowth of alveolar bone (Fig. 201). By the growth of bone into the resorbed area the tooth had become anchored in its socket, and its further eruption had been prevented; the neighboring teeth continued their occlusal movement resulting in the apparent shortening of the left central incisor.

2. **Root Resorption in Replanted Teeth.**—Extracting and replanting a tooth is perhaps the surest way to produce root resorption. Regardless of whether or not the replanted tooth has a root filling or of whether it is a single-rooted or multi-rooted tooth, an examination after a period of several months or a year will almost invariably reveal more or less extensive resorption. Sometimes ingrowing bone replaces the resorbed dentin and holds the replanted tooth solidly in place, in which instance it may give good functional service



FIG. 202.

FIG. 202.—Replanted lower incisor of dog. The tooth was extracted, the root canal filled with guttapercha, and then the tooth was replanted and held in place by wiring. Note the extensive resorption of the root at R. At B, root and bone are grown together. G, guttapercha cone.



FIG. 203.

FIG. 203.—Higher magnification of the bony union. From A to B the original cementum surface is united with the bone; from B to C the bony junction has taken place following root resorption.

(From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

for a long time; in other cases, no such repair takes place; the replanted tooth becomes loose, and when it is finally eliminated there is little if any root left.

Fig. 202 shows a dog's incisor that had been extracted, the pulp removed, and the canal filled with guttapercha. The tooth was then replanted and held in place by wiring. Upon microscopic examination three months after the operation beginning resorption of the root is evident. In some places root surface and alveolar

bone have grown together, providing, at least temporarily, good retention of the tooth in the jaw (Fig. 203).

The high incidence of extensive root resorption in the case of replantation suggests that the extraction and manipulation of the tooth may have interfered with the vitality of the root surface to such an extent that the replanted tooth is biologically no longer different from the surrounding bone; then it becomes subject to resorptive and reparative changes analogous to the processes that take place in a piece of ivory or bone that has been implanted in the jaw.

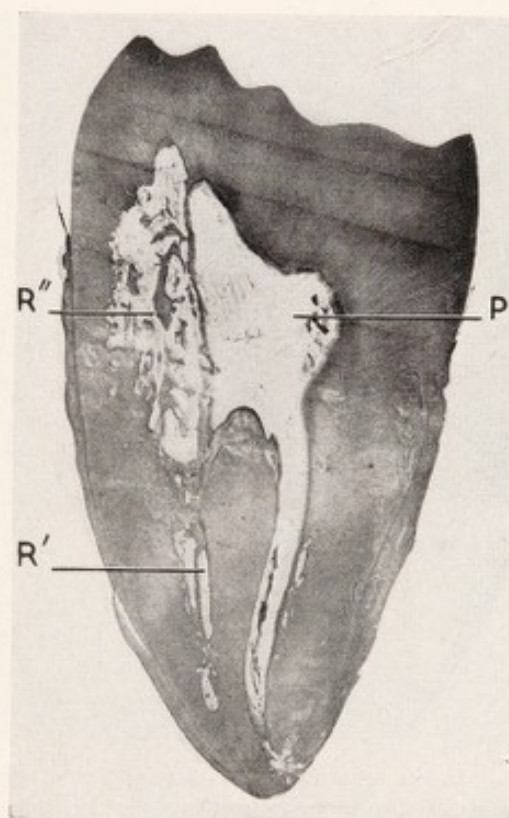


FIG. 204.—Resorption of the dentin of an impacted lower third molar. R', resorption of the root; R'', resorption of the crown with subsequent deposition of bone; P, pulp.

3. Resorptive Processes Associated With Tooth Impaction.—The pathology of tooth impaction will be discussed in a later chapter of this book; in this place only the possibilities of tooth resorption in connection with impaction will be considered. It is necessary to differentiate between two forms of resorption associated with impacted teeth, namely, resorption of the impacted tooth itself, and resorption of neighboring teeth caused by the impacted tooth.

(a) *Resorptive Processes Occurring on Impacted Teeth.*—The examination of impacted teeth occasionally reveals the presence of

resorptive processes that begin either on the crown or on the root of the impacted tooth (Fig. 204). However, it should be emphasized that the resorption of teeth that are completely embedded in the jaw bone is not a frequent occurrence. Much more often such embedded teeth are found to be intact, having a smooth, normal surface even though they have been impacted for decades. Normally the enamel epithelium of the unerupted tooth protects the enamel by isolating it from surrounding connective tissue. In order to make possible resorptive processes on the crown of an impacted tooth, it is necessary that the enamel epithelium covering

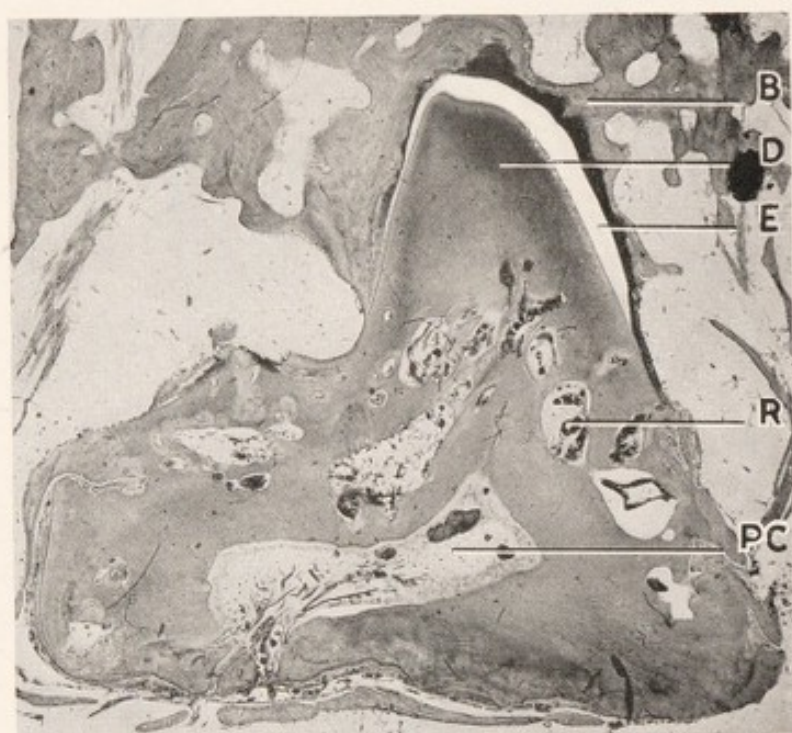


FIG. 205.—Impacted lower premolar of dog showing extensive resorption. E, enamel; B, bone deposited upon the enamel; D, dentin; R, resorption of dentin; PC, pulp chamber. (Worman, Jour. Am. Dent. Assn.)

the crown be destroyed, exposing the enamel to the surrounding connective tissue. Inflammation spreading from the infected root canal of a neighboring erupted tooth may cause destruction of the enamel epithelium.

Another possible explanation of the resorption of an impacted tooth is that the cementum on the root surface gradually loses its vitality and makes possible resorptive processes on the root, similar to those occurring in replanted teeth. Worman who studied the jaws of an old dog containing several unerupted, embedded teeth found evidence of resorption on all of these teeth. Fig. 205 illustrates an impacted, malformed premolar of this dog. The crown

is small, and the root is broad and short, a malformation not uncommon in impacted teeth. Alveolar bone and enamel have grown together; the dentin shows several large areas of resorption extending close to the pulp. A higher magnification of the crown surface shows the deposition of bone upon the irregularly scooped-out enamel surface (Fig. 206). Several findings in these specimens concerning the distribution of enamel epithelium and enamel resorption corroborate the opinion that the loss or destruction of the protecting enamel epithelium occurs first; then, when the enamel is unprotected and exposed to the periodontal connective tissue, resorption usually takes place (see also Fig. 335).

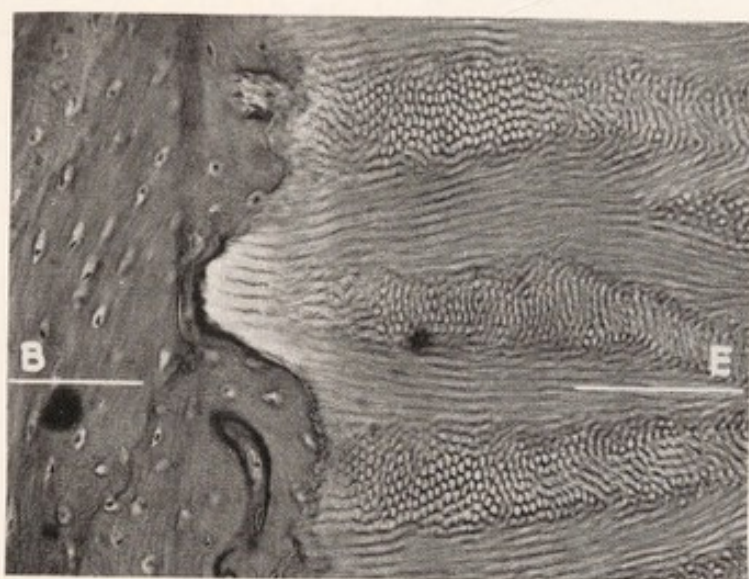


FIG. 206.—Higher magnification of Fig. 205. Deposition of bone, B, on the irregularly resorbed surface of the enamel, E. (Worman, Jour. Am. Dent. Assn.)

From time to time reports of what the observer calls "caries" on impacted teeth appear in dental literature. The author wishes to emphasize that caries on a completely embedded tooth is impossible. These reports are based on a failure to distinguish between caries and resorption. While in the radiograph resorptive defects in the crown of an impacted tooth may look like caries, the microscopic examination of the extracted specimen will invariably reveal the presence of giant cells and Howship's lacunæ in the enamel and dentin of the impacted tooth and thus establish the diagnosis of resorption beyond dispute (see Fig. 335).

(b) *Resorption of the Roots of Neighboring Teeth Caused by Impacted Teeth.*—In their attempt to erupt, impacted teeth often move with considerable force in the direction of eruption. Thus they frequently come in close contact with the roots of neighboring

teeth, causing root resorption. The most common occurrences of this type are resorption of the root of the lateral incisor by the crown of an impacted cuspid, and the resorption of the distal root of the lower second molar by the crown of a lower third molar in a case of horizontal impaction. Clinically, the development of such resorptions is often indicated by soreness, loosening, or displacement of the erupted tooth on which the impacted tooth impinges. The movements of an impacted tooth are not continuous but are interrupted by periods of inactivity and rest; thus, a period mani-

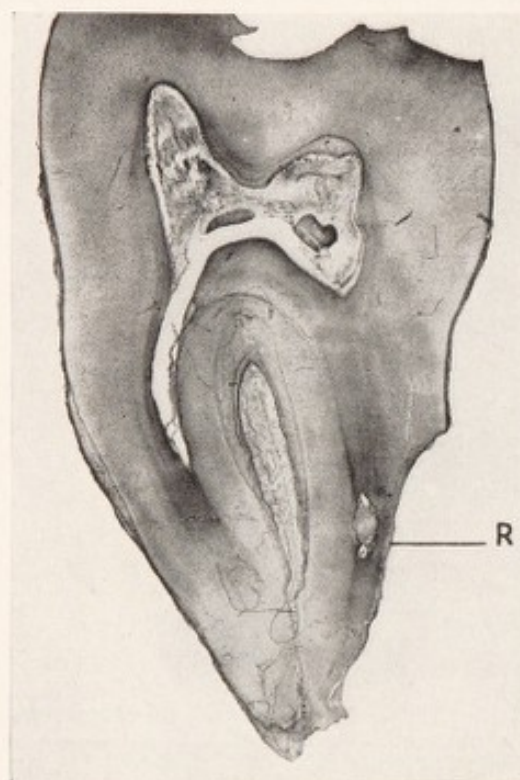


FIG. 207.—Resorption of a lower second molar caused by the crown of an impacted lower third molar. R, resorption on the distal side of the distal root covered by a thin layer of newly deposited cementum.

festated clinically by pressure by the impacted tooth is followed by a symptomless period. During the rest periods the resorbed area may be partially or totally repaired by newly deposited cementum. Such a reparative deposit of cementum also takes place if the impacted tooth is removed surgically, or if the direction of movement changes; in the latter case the resorbed area will be repaired, and new resorption will occur corresponding to the change in position (see Figs. 331, 332, and 333).

Fig. 207 shows a lower second molar against which an impacted third molar was pressing. About one-third of the distal root of the second molar had been resorbed; as the extraction of the tooth was performed during a period of rest,

a reparative deposition of cementum can be seen on the resorbed root surface.

If the impacted tooth has a strong eruptive forward movement, the enamel epithelium of the crown and the connective tissue between the crown and the neighboring root may be destroyed, and the enamel will come in direct contact with the neighboring root. Resorption being impossible, the impacted tooth pushes directly against the root, often with a great amount of force, and may cause displacement of the tooth and considerable pain.

The effect upon the neighboring teeth depends largely upon the extent of the resorption. If the resorbed area is shallow, it will readily become repaired when the impacted tooth has been removed surgically. If the resorption goes deep or exposes the pulp, usually no spontaneous repair will take place, and the involved tooth may eventually be lost.

4. **Tooth Resorption in the Presence of Tumors and Cysts of the Jaws.**

—Every pathological growth that develops in the jaws is able to cause resorption of the teeth in its neighborhood. The amount and rate of resorption depends largely upon the type of growth. A benign growth (dentigerous cyst, dental root cyst, epulis) is more likely to displace teeth or to push them apart than to cause root resorption. A malignant tumor, on the other hand, due to its rapid and destructive growth, usually causes rapid and extensive resorption of the roots that lie in the direction of its expansion.

The mechanism of tooth resorption in such cases is the same as in the case of resorption by a moving impacted tooth. The tumor causes pressure upon the connective tissue between its surface and the root, stimulating the connective tissue to osteoclastic activity and to resorption of the root. This is particularly true of tumors of epithelial origin (adamantinoma, carcinoma). The epithelial tissue of these tumors has no resorptive properties; it merely stimulates, by its growth, the resorptive activity of the connective tissue between the tumor and the root (Euler, Hofer).

In all cases of resorption of teeth by growths in the jaws, resorptive and reparative processes interchange or are found simultaneously in different parts of the same tooth. It seems that either the growth of the tumor is not continuous, making possible temporary reparative action, or temporary or permanent relief from pressure is created by a change in the direction or rate of tumor growth.

The resorption of a lower cuspid by a rapidly growing malignant tumor of mesodermic origin (osteogenic sarcoma) is illustrated in Fig. 208. The patient, a young woman, developed in a period of less than a year a large growth that involved the entire anterior part of the mandible. On removing the lower right cuspid in biopsy it was discovered that the pulp was vital and apparently normal, although more than one-half of the root had been destroyed.

If the destruction involves only a small part of the root, it is possible that a tooth may be restored to function after the damaging growth has been removed surgically. Fig. 209 shows a lower incisor from an area formerly occupied by a cystic adamantinoma.

The tumor had been removed surgically several years before. The resorbed lower surface of the root is covered by reparative deposits of cementum; the periodontal membrane has been regenerated all around the root; strong fiber bundles run from the newly formed apex to the fundus of the alveolus. Cementum has been deposited in the apical portion of the pulp chamber; the pulp tissue is vital.

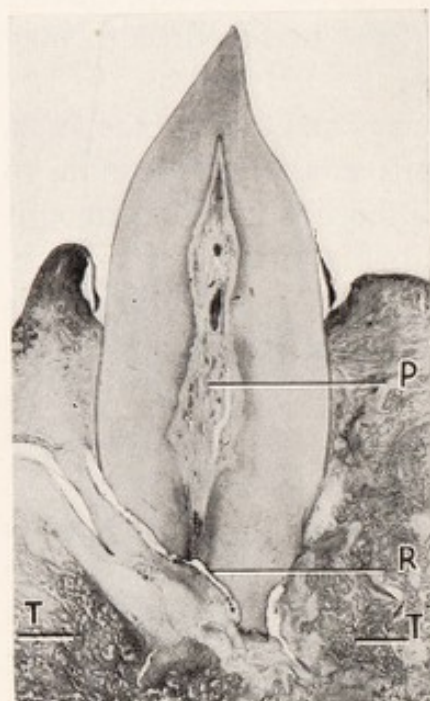


FIG. 208.—Resorption of the root of a lower cuspid by a malignant tumor (osteogenic sarcoma). P, pulp of cuspid; R, resorbed surface of the root; T, tumor consisting of the large irregular masses of bone and cartilage.

5. Root Resorption in Teeth Exposed to Excessive Occlusal Trauma.—The experiments of Gottlieb and Orban on the influence of functional stress upon the teeth of dogs have shown that occlusal trauma of long standing is able to cause root resorption. Similar resorptions were reported by Orban in a human jaw under identical conditions (see Fig. 304). In examining sections of human teeth that were in normal function over a long period of time, evidence of resorption on the surface of the root is frequently found in areas that according to the functional arrangements are areas of pressure. The



FIG. 209.—Repaired resorption of the root of a lower incisor. A cystic tumor had been removed in this area several years previous. P, pulp; C, deposits of cementum on the walls of the root canal; R, resorbed root surface covered by newly formed cementum; AB, alveolar bone; F, fiber bundles running from the newly deposited cementum to the alveolar bone. Through these fiber bundles a functional connection between root and bone has been reestablished.

large majority of these resorptions are repaired by newly formed cementum so that they have to be considered not as actual wounds but rather as scars after completely healed wounds. It must be assumed that at some period in the life of such a tooth an occlusal trauma had occurred that caused this resorption which was subsequently repaired (Fig. 210). It seems that sometimes a minute trauma, such as biting upon a shot or a cherry stone, may be responsible for a slight damage to the periodontal membrane and for subsequent root resorption in the damaged area. Such minor traumatic injuries are probably responsible for the great number of small repaired resorptions that are found in teeth of people who use much force in mastication.

Whenever resorption of the root takes place the periodontal fibers lose their attachment to the involved area of the root surface; the corresponding alveolar bone will also be resorbed due to the loss of functional stimuli. The periodontal membrane thus becomes wider in the damaged area. When repair takes place, new cementum in which new fibers are embedded is deposited on the resorbed root surface; functional stimuli are again transmitted to the bone, which is regenerated until the original width of the functioning periodontal membrane is restored. This process is called functional repair. Often the reparative formation does not reach the level of the original root surface, in which case the bone projects into the depressed area of the root leaving a periodontal space of uniform width between the two hard substances (see Fig. 305).

Excessive and irregular force applied in orthodontic movement of teeth may result in extensive resorption of the roots of those teeth. In extreme cases the teeth become loosened since little of the

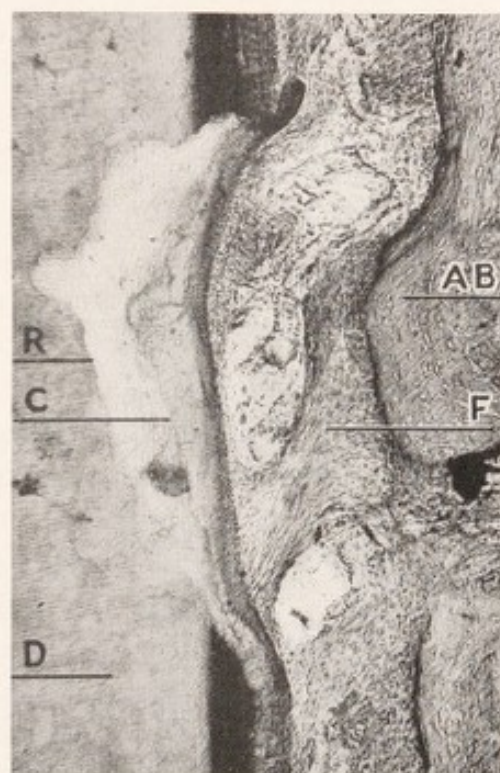


FIG. 210.—Repaired resorption on the root of an upper molar. The alveolar bone and periodontal membrane have been completely regenerated in the resorbed area (see also Figs. 178–180). D, dentin; R, line of resorption in the dentin; C, reparative deposits of cementum; F, fiber bundles of the periodontal membrane; AB, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

roots is left, although the pulps usually remain alive. If the orthodontic force is relieved, the teeth may become firm again by reparative deposits of cementum, provided that the destruction of the root was not too extensive. However, such roots always remain shorter than they were originally (see Fig. 314).

6. Root Resorption of Unknown Etiology or of Systemic Origin.—Under this heading we must put all of the puzzling cases of root resorptions that are from time to time observed in permanent teeth with intact pulps. Sometimes only one tooth in a full set is resorbed; sometimes several teeth are involved. But all cases have one thing in common: namely, it is impossible to explain the process by a local cause. Sometimes the case history can be traced back to some accident (trauma) that might have started the process; it seems possible that a small traumatic resorption, instead of being repaired, became larger and gradually involved the entire root. In other cases, there is no history of a possible cause, several teeth being frequently attacked in different parts of the jaw under quite unrelated outer circumstances. It must then be assumed that some internal disturbance led to these generalized resorptions.

This internal factor is still largely unknown, and consequently many cases of root resorption cannot be explained. We may be able to give an accurate clinical and histopathological description of the case, but we do not know its cause nor how to influence its progress. Only further careful observation and close coöperation with investigators in the field of internal medicine may eventually lead to a solution of this problem.

(a) *Resorption of Individual Permanent Teeth, Etiology Unknown.*—A case of extensive resorption was observed in the upper first bicuspid of a boy, aged fifteen years. The tooth appeared slightly discolored, showing a decidedly pinkish hue. The radiograph disclosed a very indistinct root outline and an abnormal configuration of the pulp chamber. The tooth was in normal occlusion, the crown intact; all other teeth in the mouth were normal. The bicuspid was removed. In a radiograph of the specimen, the dentin of both the root and the crown appears to have been replaced by cancellous bone (Fig. 211), a fact confirmed by the histological section. Almost all of the dentin has disappeared, being replaced by fibrous connective tissue and newly formed bone trabeculae. In some places the process of dentin resorption was still going on. Of the entire crown there was left only a thin outer cap of enamel, which also had been attacked by the resorptive process. The pink discoloration of the tooth was caused by the presence of a highly

vascularized soft tissue inside the crown that shone through the thin, transparent enamel ("pink spots" of Mummery). No cause for the resorption can be given in this case.

Another case of tooth resorption of unknown origin is illustrated in Fig. 212. The two upper central incisors of a man, about fifty years of

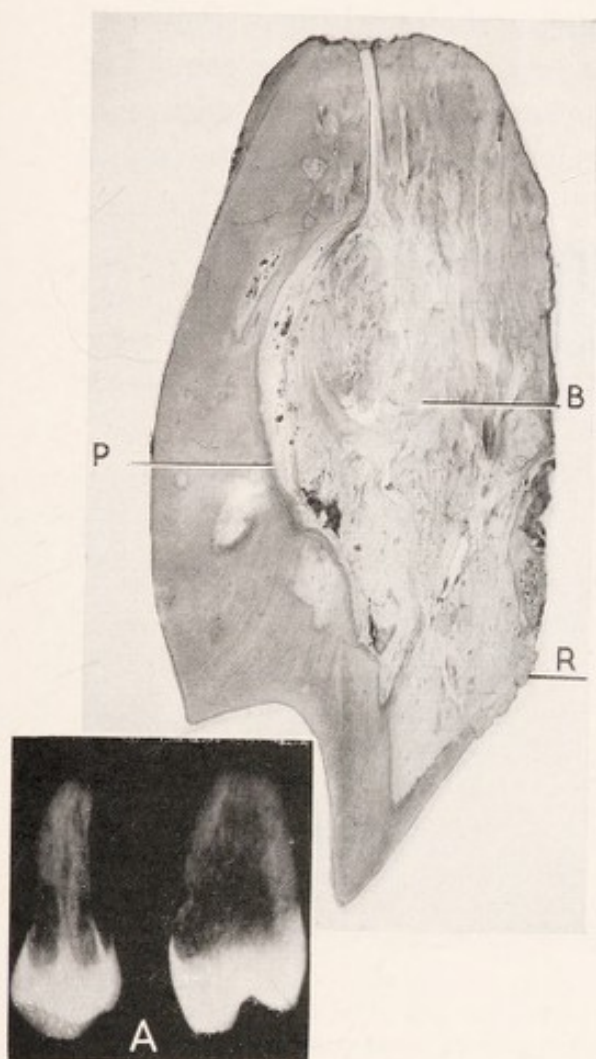


FIG. 211.—Extensive resorption of an upper first bicuspid of a boy, aged fifteen years. Formation of bone in the resorbed area. Etiology unknown. A, radiographs of the extracted tooth. Bucco-lingual section through the specimen. P, original outline of pulp chamber; B, connective tissue and newly formed trabeculae of bone replacing the dentin; R, resorption of enamel. (Courtesy of D. M. Gallie, Jr.)

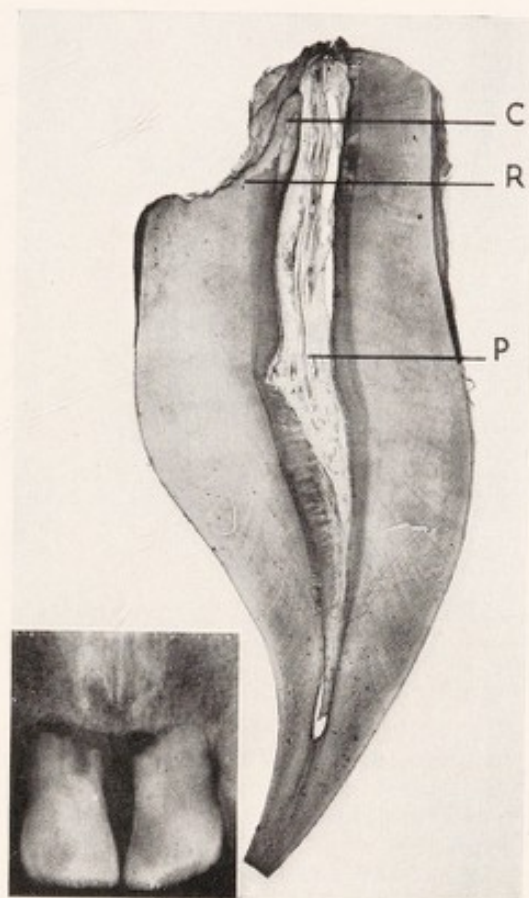


FIG. 212.—Resorption of the roots of the upper central incisors of a man, aged fifty years. The roots appear radiographically very short. Clinically the teeth were moderately loose. P, pulp; R, line of resorption on the root; C, cementum covering the resorbed root surface.

age, appeared very loose upon clinical examination. The other teeth in his mouth were in fairly good condition. The radiograph revealed the surprising fact that both central incisors consisted of practically nothing but crowns attached to the gums. No roots were to be seen. The area normally occupied by the roots had been filled in by bone. The patient could give no information

on this condition; he asserted that the teeth, until a short time before, had been firm and normal. The histological examination showed a normal incisor crown and a normal pulp chamber. The root ended abruptly in its gingival third; the root end had been covered by a layer of cementum to which periodontal fibers were still attached. It seems that, after the largest part of the roots had been eliminated by a resorptive process of unknown nature, a reparative deposit of cementum occurred and a new attachment

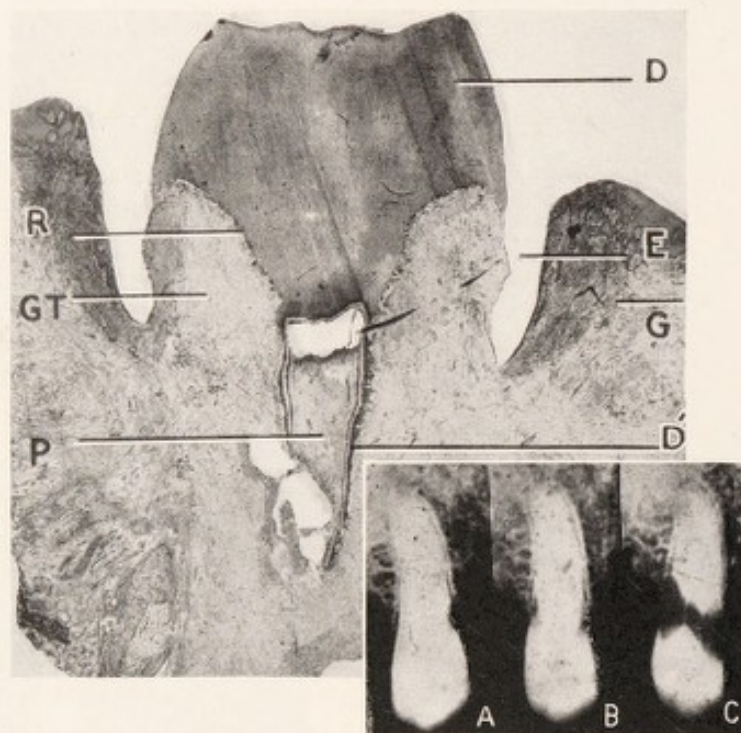


FIG. 213.—Extensive resorption of roots and crown of the upper right second bicuspid in a case of rapid resorption of the permanent set of teeth of a woman, aged thirty-eight years. The three radiographs in the right lower corner show the rapid progress of destruction of this tooth. A, January 19, 1929: shallow indentation on the distal side of the crown. B, March 6, 1929: indentation on both the mesial and distal side of the root. C, May 27, 1929: in less than three months the defects visible in B have become confluent in the middle of the tooth, so that crown and root appear to be almost entirely separated. Mesio-distal section through the crown portion of the bicuspid: D, dentin; E, enamel; G, gingival tissue; R, resorption undermining the enamel; GT, granulation tissue; P, pulp; D', dentinoid and dentin surrounding the pulp. (Mueller, Jour. Am. Dent. Assn.)

was formed, but that the small area of attachment was not big enough to retain the large crown, and the teeth were lost. A similar case has been described by Zemsky.

(b) *Generalized Root Resorption*.—We shall now proceed to the description of two cases of generalized root resorption that were studied and described histologically. In both cases there is at least a certain probability that known internal disturbances were responsible for the destruction of the teeth. In one case, reported

by Mueller, Rony, and Kronfeld, there was present a distinct disturbance in the function of the liver; the teeth were obtained by biopsy. The other specimen, obtained by autopsy from an individual who died from grippe, is the case of diffuse atrophy published by Gottlieb.

The first patient, a woman, aged thirty-eight years, was observed over a period of two years, during which time extensive resorptions developed on the roots of several intact permanent teeth. The radiographs of some of these teeth may give an idea of the rapidity of the destructive process (Fig. 213). In January, 1929, a shallow defect was

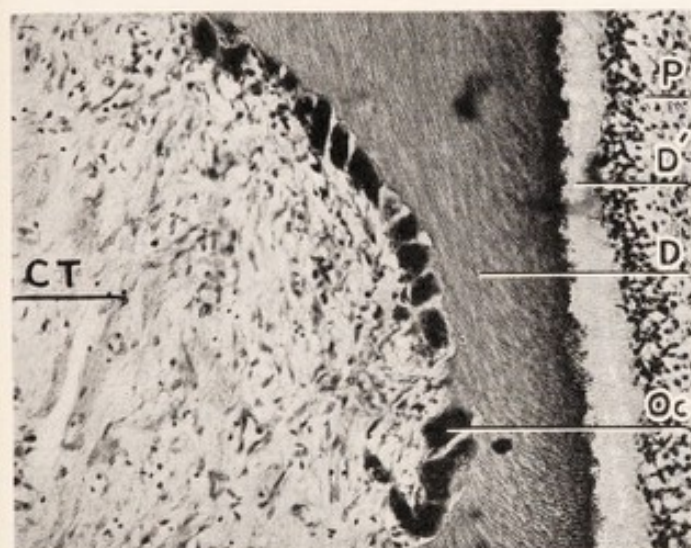


FIG. 214.—Higher magnification of the dentin resorption in Fig. 213. The entire dentin surface is densely beset with polynuclear osteoclasts (giant cells). P, pulp; D', dentinoid; D, dentin; Oc, osteoclasts lying in Howship's lacunæ in the dentin; CT, connective tissue. (Mueller, Jour. Am. Dent. Assn.)

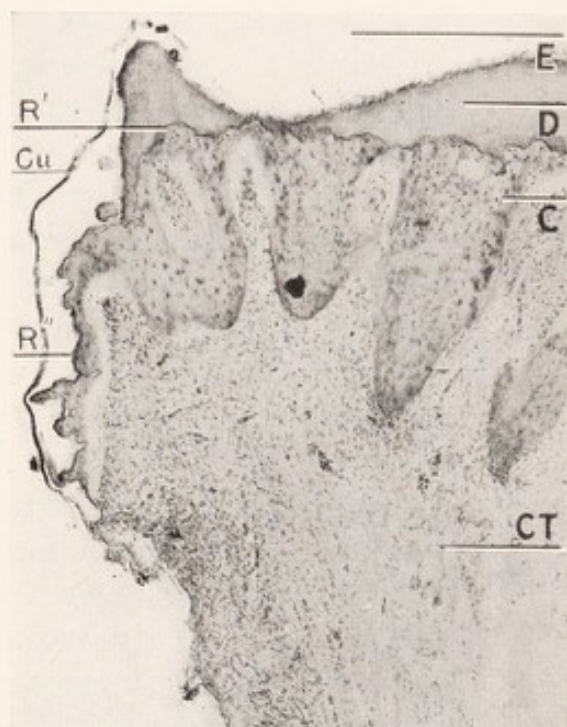


FIG. 215.—Reparative formation of bone on the inside of the resorbed crown of an upper lateral incisor. Same case as shown in Figs. 213 and 214. D, dentin; E, enamel; Cu, cuticle; R', line of resorption in the dentin; R'', enamel resorption; C, deposits of bone on the resorbed surface of enamel and dentin; CT, connective tissue. (Mueller, Jour. Am. Dent. Assn.)

visible near the alveolar margin on the mesial side of an upper second bicuspid. On March 6, a similar defect had appeared on the distal side of this tooth. About three months later, on May 27, these two defects involved the entire thickness of the root, thus separating root and crown. The tooth was extracted together with some of the gum tissue, and studied under the microscope. Fig. 213 shows a mesio-distal section through the crown. The resorptive process had undermined the enamel of the crown. The dentin immediately surrounding the pulp formed a "hollow column" (Tomes, page 232). A higher magnification of the inside

of the crown gives a good illustration of the great number and dense arrangement of the giant cells, which is not surprising considering the unusual rapidity with which the dentin of this tooth was resorbed (Fig. 214).

The lower right cuspid and first bicuspid were observed radiographically over a period of about seven months. They showed the same rapid process of root destruction between January and July 1929. A specimen consisting of these two teeth was removed in July 1929. It showed evidences of beginning repair in places, as well as the extensive resorptive destruction of tooth substance. This is still clearer in the upper right lateral incisor of the same patient. This tooth was intact in January 1929; in July 1929, two deep resorptions penetrating from the mesial and distal sides had almost completely separated the root from the remaining part of the crown. From that time on, however, there was no further destruction; on the contrary, later radiographs showed that the size of the defect had been reduced by newly deposited hard substance. In February 1930, the lateral incisor was removed and examined microscopically. A photograph of a section through the crown revealed the presence of extensive deposits of a bone-like substance (cementum) inside the hollow crown. In some areas where the undermining resorption of the crown had already attacked the enamel from the inside, extensive deposits of cementum had occurred upon the inner enamel surface (Fig. 215).

In the case described above there was a generalized rapid process of tooth resorption going on in almost all the teeth. On making a thorough metabolic and internal examination, a disturbance in the liver was found. In the summer of 1929 treatment of the liver was started. From this time repeated tests showed that the function of the liver improved and that the resorptive process on the teeth came to a standstill; no more teeth were attacked, and the destroyed areas were reduced by new deposits of hard tissue.

Whether the dental condition and the hepatic disorder were related etiologically remains, of course, to be proved; such proof could be established only after the same two conditions were found associated in a series of cases. It is suggested that anyone observing a similar case of resorption should keep in mind the possibility of a hepatic or any other metabolic disorder.

In the case of generalized root resorption the histological findings of which were reported by Gottlieb in 1923, the patient, a man, aged twenty-two years, had died from influenza (grippe). Except for the upper first bicuspid and the lower first molars, all teeth were in

place. A microscopic examination of the teeth showed superficial resorptions of all the roots (see Fig. 268); in some places beginning reparative processes could be noticed. The clinical importance of this type of resorption and its relationship to the alveolar bone (diffuse atrophy of the alveolar bone) will be discussed elsewhere. (See Chapter XI.) It seems most probable that the severe disease to which the patient finally succumbed caused a (toxic?) damage to the periodontal tissues and to the root surface, and as a result the cementum was resorbed.

In summarizing, our knowledge of this last type of tooth resorption is still very limited. Only a very few cases have been studied, and each case offered a different clinical and microscopic appearance. Only further careful study and examination of each case that may be observed can help toward a solution of this problem.

BONY JUNCTION BETWEEN ROOT AND ALVEOLUS.

It seems advisable to give a brief review of the facts known about solid bony junctions between roots and alveolus. Since the condition almost invariably is preceded by root resorption, its discussion is included in this chapter.

In the first place, it is necessary to bring up the question: Why are not root surface and bone found grown together more often? Under the microscope, cell-containing cementum on the root surface and alveolar bone on the other side of the periodontal space look practically alike. There seems to be no apparent reason why these two hard tissues should not grow together, if, for instance, functional stimuli are entirely eliminated. Still we know from clinical and microscopic observations that impacted teeth as well as fractured roots that are left behind after extractions usually do not unite with the surrounding bone, although functional factors are completely absent (see Fig. 359). On the other hand, it is well known that replanted teeth and alveolar bone usually do grow together, although these teeth are in occlusion and under the constant influence of functional stresses. Therefore, it cannot be primarily the functional factor that controls the development of a bony union. In order to find out what determines the bony junction between tooth and bone, the factors that cause such a junction will be reviewed.

Replantation results most frequently in a solid union with the alveolar bone, since almost all replanted teeth are found ankylosed to the bone if they remain long enough in the jaw. Next in fre-

quency are pulpless teeth with periapical inflammation (see Figs. 200 and 201). Here extensive resorptions of the root are sometimes found, and when bone grows into the resorbed areas, it is likely to become united with the root. Another cause for a bony junction

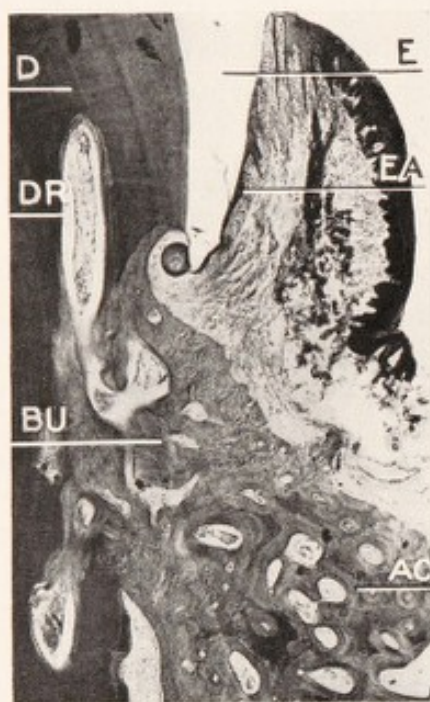


FIG. 216.—Bony union between tooth and alveolus (dog). The union was produced experimentally by placing a metal cap upon the tooth for about one month. Then the cap was removed and the animal kept alive for three additional months. The heavy occlusal stress when the cap was worn caused root resorption; in the subsequent reparative phase, root and bone became united. D, dentin; E, enamel; EA, epithelial attachment; DR, dentin resorption causing duct-like defects in crown and root; BU, bony union between root and alveolus; AC, alveolar crest. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

between root and jaw has been produced experimentally by excessive occlusal trauma and subsequent relief. The trauma caused root resorption. After there was relief, these resorptions were repaired, and bone and tooth became united (Fig. 216). Of similar origin also is the occasional union between deciduous teeth and bone (see Fig. 194).

What is the common factor in all the conditions mentioned above? It can be summarized under two headings:

1. The junction is usually preceded by more or less extensive root resorption.
2. The condition that led to this junction caused primarily an injury to the periodontal tissues and to the root surface.



FIG. 217.—Spontaneous bony union between teeth and alveolus in an old dog. BU, points of bony union. (Köhler, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

The first point is self-explanatory by studying the specimens shown in this chapter. With regard to the second point, it has been stated before that bone and cementum normally have no tendency to unite, although they are similar in origin and structural

appearance. It seems that the periodontal membrane or the cementum or perhaps both have certain qualities, not yet explained, that maintain the tooth as an independent organ in the surrounding bone. Injury to the tooth surface seems able to destroy this independence after which the tooth is just a part of the skeletal bone, and, like the latter, is subject to the usual resorptive and regenerative changes occurring in bone. With this thought in mind we are justified in saying that all of the causes of ankylosis between root and bone, such as replantation, periapical inflammation, and occlusal trauma, are harmful to the root. These conditions reduce the formerly independent and autonomous cementum surface to the lower standard of bone, and then, like bone, the tooth is resorbed and new bone is built instead. With a similar deterioration of the surface of the root, we may also explain the occurrence of the bony junction that has occasionally been observed between root and bone in old individuals. Köhler reported a number of cases of extensive bony ankylosis in the teeth of old dogs (Fig. 217). The experiments of Gottlieb and Orban have shown that the root of an old tooth has a diminished resistance to resorption as compared with that of a young tooth. Therefore, we may assume that the lowered vital qualities of old teeth tend to decrease the biological difference between cementum and bone.

BIBLIOGRAPHY.

- AISENBERG, MYRON S.: Report of a Case of Ankylosis of a Tooth, *Dental Cosmos*, 1932, **74**, 1071.
- AUSTIN, L. T., and STAFNE, EDWARD C.: Retained Deciduous Roots, *Jour. Am. Dent. Assn.*, 1932, **19**, 1320.
- BECKS, HERMANN, and MARSHALL, JOHN A.: Resorption or Absorption? *Jour. Am. Dent. Assn.*, 1932, **19**, 1528.
- CAHN, L. R.: A Case of Bone Metaplasia in the Pulp Canal of a Tooth, *Dent. Items Int.*, 1932, **54**, 518.
- EULER, H.: Über das Verhalten der Zähne bei malignen Kiefertumoren, *Deutsch. Mon. f. Zhk.*, 1925, **43**, 701.
- Resorption der Wurzel, *Handw. d. ges. Zhk.*, vol. **3**, 2460.
- FIGG, WILLIAM A.: Report on a Case of a Persistent Deciduous Canine, *Dent. Items Int.*, 1930, **52**, 606.
- FISCHER, GUIDO: Beiträge zum Durchbruch der bleibenden Zähne und zur Resorption des Milchgebisses nebst Untersuchungen über die Genese der Osteoklasten und Riesenzellen, *Anat. Hefte*, 1909, vol. **38**, No. 116.
- GÖLLNER, LUDWIG: Zahnverlust durch Resorption am Zahnhals. Knochenbildung in der Pulpakammer, *Ztschr. f. Stom.*, 1931, **29**, 613.
- GOTTLIEB, B.: Die diffuse Atrophie des Alveolarknochens, *Ztschr. f. Stom.*, 1923, **21**, 195.
- Formation of the Pocket; Diffuse Atrophy of the Alveolar Bone, *Jour. Am. Dent. Assn.*, 1928, **15**, 462.
- GOTTLIEB, B., and ORBAN, B.: Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne, Leipzig, Thieme, 1931.

- GOTTLIEB, B., and ORBAN, B.: Veränderungen im Periodontium nach chirurgischer Diathermie, *Ztschr. f. Stom.*, 1930, **28**, 1208.
- HAM, ARTHUR W.: The Function of Bone as a Calcium Reservoir with a Consideration of the Cellular Pictures Seen in Resorption with Particular Reference to the Significance of Osteoclasts, *The Angle Orthodontist*, 1932, **2**, 142.
- HESSE, G.: Über persistierende Milchzähne bei unterzähligen Gebissen, zugleich ein Beitrag zur Frage ob die Pulpa Zement bildet, *Deutsch. Mon. f. Zhk.*, 1921, **39**, 161.
- HOFER, O.: Alveolarknochen und Zahn bei bösartigen Geschwülsten der Kiefer, *Ztschr. f. Stom.*, 1925, **23**, 522.
- HOPEWELL-SMITH, A.: The Process of Osteolysis and Odontolysis, or So-called "Absorption" of Calcified Tissues: A New and Original Investigation, *Dental Cosmos*, 1930, **72**, 323, 462, 610, 912, 1036, 1151.
- KÖHLER, J.: Verwachsung zwischen Zahn und Knochen, *Ztschr. f. Stom.*, 1927, **25**, 896.
- KOTANYI, E.: Histologische Befunde an retinierten Zähnen, *Ztschr. f. Stom.*, 1924, **22**, 747.
- Histologische Befunde an Milchzahnresten, *Ztschr. f. Stom.*, 1925, **23**, 516.
- Histologische Befunde an einem retinierten Milchmolaren und einem retinierten Weisheitszahn, *Ztschr. f. Stom.*, 1931, **29**, 764.
- KRONFELD, RUDOLF: Spielt die Qualität der Hartsubstanzen bei der Resorption eine Rolle? *Ztschr. f. Stom.*, 1927, **25**, 1099.
- The Resorption of the Roots of Deciduous Teeth, *Dental Cosmos*, 1932, **74**, 103.
- Die Resorption der Milchzahnwurzeln, *Deutsch. Mon. f. Zhk.*, 1932, **50**, 97.
- KRONFELD, RUDOLF, and MUELLER, E.: Ein Fall von generalisierter Zahnresorption, *Ztschr. f. Stom.*, 1931, **29**, 276.
- LUKOMSKY, L.: Befunde an einem retinierten Zahn, *Deutsch. Mon. f. Zhk.*, 1931, **49**, 321.
- MALEY, CHARLES: The Replantation of Teeth, *Dent. Items Int.*, 1932, **54**, 94.
- MARSHALL, JOHN A.: A Comparison of Resorption of the Roots of Deciduous Teeth with the Absorption of Roots of the Permanent Teeth Occurring as a Result of Infection, *Pacific Dent. Gaz.*, October, 1928, p. 597.
- MUELLER, E.: Histologic Study of a Case of Extensive Root Resorption in a Human Being, *Jour. Am. Dent. Assn.*, 1931, **18**, 684.
- MUELLER, E., and RONY, H. R.: Laboratory Studies of an Unusual Case of Resorption, *Jour. Am. Dent. Assn.*, 1930, **17**, 326.
- MUMMERY, I. H.: The Pathology of "Pink Spots" on Teeth, *Brit. Dent. Jour.*, 1920, **41**, 301.
- Some Further Cases of Chronic Perforating Hyperplasia of the Pulp (the So-called "Pink Spot"), *Brit. Dent. Jour.*, 1926, **47**, 801.
- NOYES, FREDERICK B.: Submerging Deciduous Molars, *The Angle Orthodontist*, 1932, **2**, 77.
- OPPENHEIM, A.: Histologische Befunde beim Zahnwechsel, *Ztschr. f. Stom.*, 1922, **20**, 543.
- ORBAN, B.: Growth and Movement of the Tooth Germs and Teeth, *Jour. Am. Dent. Assn.*, 1928, **15**, 1004.
- Resorption and Repair on the Surface of the Root, *Jour. Am. Dent. Assn.*, 1928, **15**, 1768.
- ORBAN, B., and WEINMANN, J.: Die ursächlichen Bedingungen für den Abbau der Hartsubstanzen, *Virchow's Arch. f. path. Anat.*, 1928, **262**, No. 2, 446.
- PRITCHARD, G. B.: A Specimen Showing "Pink Spot," *Dent. Surg.*, 1931, p. 669.

- SCHÖNBAUER, FRANZ: Knöchern eingeheilte Milchzahnreste bei älteren Individuen, *Ztschr. f. Stom.*, 1931, **29**, 892.
- SCHWEITZER, G.: Interne Granulome der Zahnpulpa und ihre resorbierende Wirkung im Innern des Zahnkörpers, die "Rosaflecken" (Pink Spots) Krankheit der Zähne, *Deutsch. Zahnärztl. Wehnschr.*, 1931, **35**, 175, 245.
- SICHER, HARRY: Die sogenannten verkürzten Zähne, *Ztschr. f. Stom.*, 1928, **26**, 396.
- TOMES, CHARLES S.: *A Manual of Dental Anatomy*, London, 1876.
- TOMES, JOHN: *Dental Surgery*, Lindsay & Blackiston, London, 1859.
- TYLMAN, STANLEY D.: The Dento-enamel Junction, *Jour. Dent. Res.*, 1928, **8**, 615.
- URBAN, LLOYD: Findings in Relation to Resorption of Deciduous Teeth in the Dog, *Jour. Dent. Res.*, 1931, **11**, 711.
- WEBER, R.: Demonstration über die Resorption intakter Zähne, *Paradentium*, 1929, **1**, 29.
- Zur Kasuistik der histologischen Veränderungen an retinierten Zähnen, *Deutsch. Mon. f. Zhk.*, 1925, **43**, 813.
- WILLMAN, WARREN: An Apparent Shortening of an Upper Incisor: Its Significance, *Jour. Am. Dent. Assn.*, 1930, **17**, 444.
- WORMAN, HAROLD G.: A Histopathologic Study of Impacted Teeth, *Jour. Am. Dent. Assn.*, 1929, **16**, 1885.
- ZEMSKY, J. L.: Root Resorption and Its Clinical Significance, *Jour. Am. Dent. Assn.*, 1929, **16**, 520.
- ZILKENS, K.: Einiges über die Resorption der Zähne, *Deutsch. Zahnärztl. Wehnschr.*, 1924, **27**, No. 10.

CHAPTER X.

EPITHELIAL ATTACHMENT AND GINGIVAL CREVICE.

BEFORE considering periodontal diseases, it is necessary to describe the normal conditions and physiological changes found in gingival tissues. Investigations in this field during the last decade have resulted in many and important new findings which necessitate a revision of our previous conceptions of the gingival tissues under normal and abnormal conditions.

EPITHELIAL ATTACHMENT.

The former conception of the relationship between tooth and gingivæ was that the gingivæ are attached to the tooth in its entire circumference at the cemento-enamel junction. The soft tissues crownward from the cemento-enamel junction were called free gingivæ. Recent investigations have proved that the condition just described never existed and, consequently, all the theoretical and practical considerations that were based on this conception have had to be changed. Therefore, the actual relationship between tooth surface and investing soft tissues is described as it was found through the histological study of a large number of specimens from human jaws of different ages.

1. **Definition and Terminology.**—In 1921, Gottlieb demonstrated that in a young tooth the bottom of the gingival crevice is not located, as was formerly believed, at the cemento-enamel junction, but upon the surface of the crown, and that the cervical part of the enamel and the enamel epithelium are still in organic junction. Gottlieb called the band of epithelium that is in organic connection with the tooth surface the “epithelial attachment.”

The difference between the old and the new conception is illustrated by a diagram (Fig. 218).

Old Conception.—The epithelium is attached to the tooth in a line around the cemento-enamel junction (Fig. 218, left side at *c*).

New Conception.—The epithelium is not attached in a line but in a band. This band of epithelium (Fig. 218, right side *A-C*) surrounding the tooth is united with the tooth surface.

The presence of the epithelial attachment is evident to anyone who studies well-prepared sections of jaw specimens from men and animals. The location of the epithelial attachment on the tooth surface varies greatly in different teeth, in different individuals, and at different ages. But in every erupted tooth the oral epithelium is united with the tooth surface by an epithelial attachment.

It has been the author's observation that the nomenclature in this particular field is rather vague and, therefore, the terms that are at the present time generally accepted will be enumerated here.

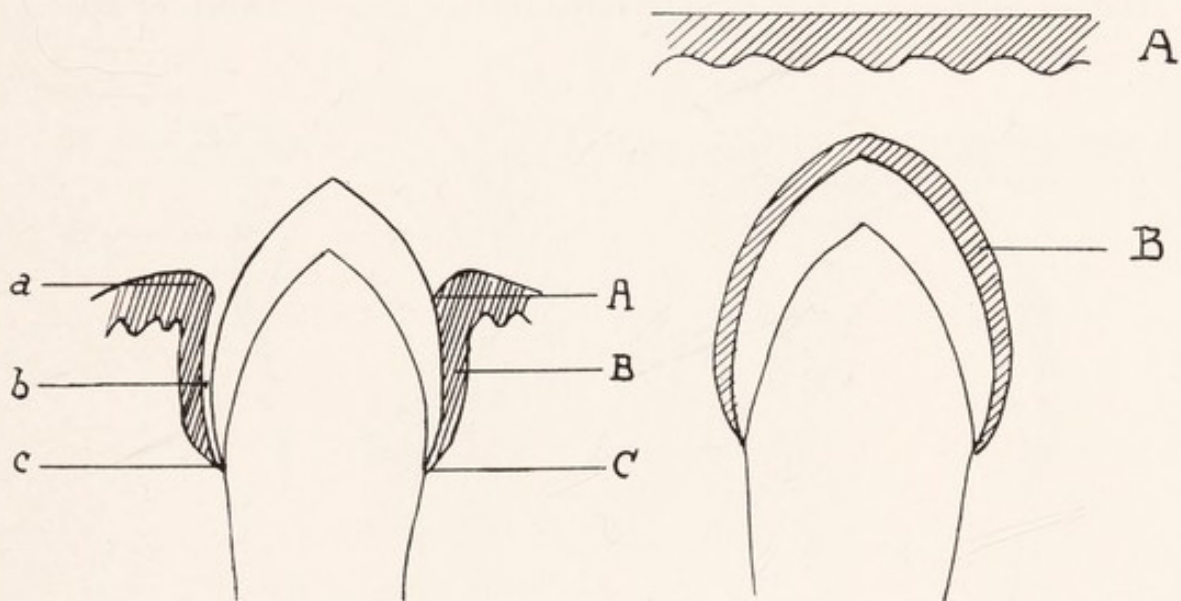


FIG. 218.

FIG. 219.

FIG. 218.—Diagram illustrating the difference between the old and new conceptions of the attachment of the gingival tissues to the tooth. The small letters are used to illustrate the old conception; the capitals, the new conception. *a*, free gingiva; *b*, subgingival space; *c*, gingival line; *A*, bottom of the gingival crevice; *B*, epithelial attachment; *C*, cemento-enamel junction (deepest point of the epithelial attachment).

FIG. 219.—Diagram illustrating a tooth before eruption. *A*, mouth epithelium; *B*, enamel epithelium in organic connection with the enamel. (Orban and Mueller, Jour. Am. Dent. Assn.)

The term gingival margin is used to describe that extreme crest of the gingiva from which the crevice epithelium begins its descent into the crevice. The crevice epithelium extends from the gingival margin to the line of attachment of epithelium to the tooth surface, which line is obviously the bottom of the gingival crevice. The epithelial attachment is that portion of the epithelium which is in organic connection with the tooth surface and is, consequently, found rootwise from the bottom of the crevice. The gingival crevice is the space that is bordered on the inner side by the tooth surface and on the outer side by the crevice epithelium.

2. **Development of Epithelial Attachment and Gingival Crevice.**—Epithelial attachment and gingival crevice can be fully understood only when studied in connection with tissue changes occurring during the eruption of the tooth. Therefore, these changes will be briefly reviewed.

The enamel of the tooth is formed by the enamel organ, which originally consists of four layers. The ameloblasts or ganoblasts, cylindrical cells whose function is to form the enamel, constitute the innermost layer of the enamel organ. The stratum intermedium, composed of several rows of cells, is adjacent to the ganoblasts. Still farther outward is the stellate reticulum, a loose network of epi-

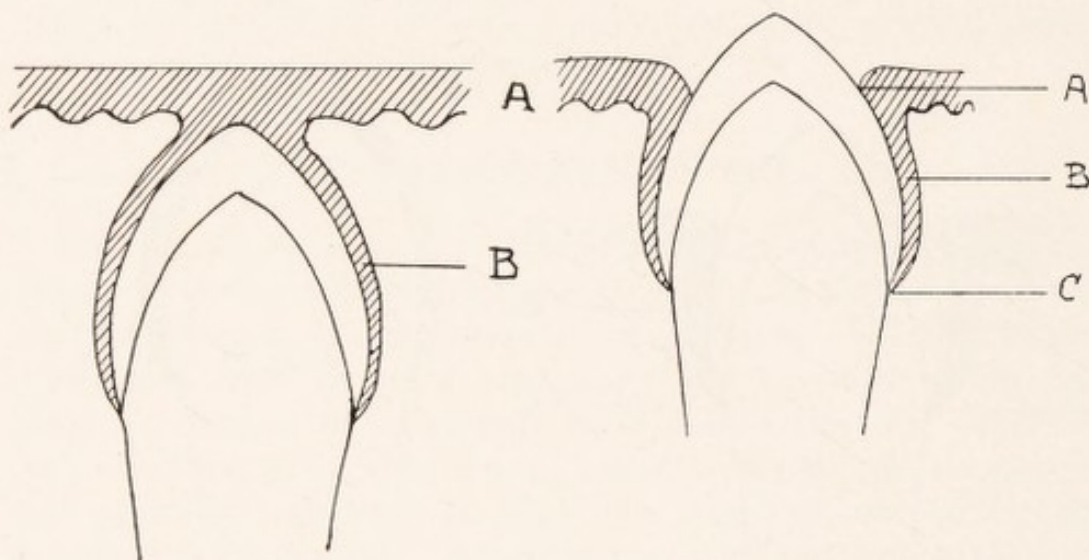


FIG. 220.

FIG. 221.

FIG. 220.—The tooth is a little further advanced in eruption than in Fig. 219. A, mouth epithelium; B, enamel epithelium. The epithelial tissues are united.

FIG. 221.—The tip of the enamel is erupted. A, bottom of the gingival crevice. The tooth is erupted to this point; B, epithelial attachment in organic connection with the enamel; C, cemento-enamel junction. (Orban and Mueller, Jour. Am. Dent. Assn.)

thelial cells that form the main part of the enamel organ. Finally there is the outer enamel epithelium which covers the surface of the enamel organ and consists of several layers of cuboidal epithelial cells. After the enamel formation has been finished, the ameloblasts disappear; the remaining layers of the enamel organ are fused and finally form several rows of stratified squamous epithelial cells, the reduced enamel epithelium (Orban). This layer of epithelial cells is in organic connection with the surface of the enamel, separating the crown from the surrounding connective tissue of the jaw (Fig. 219). During the eruptive movement of the tooth, the crown moves closer to the oral epithelium, and on reaching it, the enamel epi-

thelium on the crown surface unites with the oral epithelium (Fig. 220). Thus, when the tip of the enamel appears in the oral cavity, the oral epithelium is continuous with the enamel epithelium all around the crown (Fig. 221). Inasmuch as the enamel epithelium is in organic connection with the enamel surface, it is obvious that the cervical portion of the enamel is not exposed but is still united with the gum tissue.

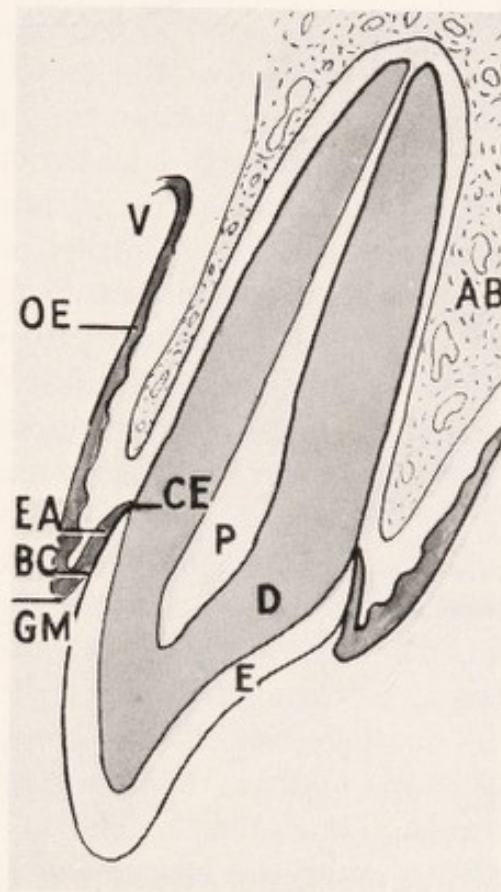


FIG. 222.—Diagram of an upper central incisor of a child at the age of ten years. Labio-lingual section. This diagram has been drawn from specimens, such as illustrated in Figs. 223 and 224. P, pulp; D, dentin; E, enamel; CE, cemento-enamel junction; OE, oral epithelium; V, labial vestibulum; GM, gingival margin; BC, bottom of the gingival crevice on the enamel surface; EA, epithelial attachment to the enamel; AB, alveolar bone. The free gingivæ extend from BC to GM.

A drawing made from a specimen of an upper central incisor in a child, aged ten years, illustrates the actual condition at this period of life (Fig. 222). The crown has reached the occlusal plane and articulates with its antagonists. Approximately three-fourths of the enamel of the crown is exposed and rises into the oral cavity; the gingival fourth of the enamel is covered by the soft tissue which was formerly believed to be the "free gingivæ." But under normal conditions the free gingivæ constitute only a small portion of the tissues that cover the gingival part of the enamel; a large portion

of the gingival tissues which overlay the enamel is not free but is in organic connection with the enamel surface through the epithelial attachment.

After it had been found that the bottom of the gingival crevice at the time the tooth reaches the occlusal plane is not at the cemento-enamel junction, it seemed advisable to introduce new terms for the erupted and the unerupted parts of the tooth surface. In every human tooth we can differentiate between the crown, that is covered by enamel, and the root, that is covered by cementum. Gottlieb called the enamel covered portion of the tooth the "anatomical crown" and the cementum covered portion the "anatomical root," the term anatomical referring to the fact that the borderline between crown and root, the cemento-enamel junction, can always be recognized on an extracted tooth. Gottlieb called that part of the tooth which projects above the bottom of the gingival crevice the "clinical crown," and that part which extends rootward from the bottom of the gingival crevice the "clinical root." From this definition it is evident that clinical crown and clinical root can be distinguished only as long as the tooth is in connection with the surrounding soft tissues and, furthermore, that the ratio between clinical crown and clinical root depends upon the location of the bottom of the gingival crevice on the tooth surface (see Fig. 235).

The space drawn in the diagram between the free gingivæ (*BC* to *GM*) on one side and the enamel surface on the other side does not exist in life. The small seam of the free gingivæ is in close contact with the surface of the tooth so that the gingival crevice forms a capillary cleft between *GM* and *BC*. The space in the diagram has been drawn only to make the location of the bottom of the gingival crevice plainer.

3. Discovery of Epithelial Attachment.—There are several reasons why the existence of an epithelial attachment to the enamel was not discovered until rather recently and why some men engaged in dental research have not yet been able to convince themselves of its existence. These reasons are:

It is difficult to prepare histological sections of both the tooth and the surrounding soft tissues and bone. In ground sections, most of the histological details of the soft tissues are destroyed. In frozen sections and in sections of jaw specimens that have been embedded in paraffin, misleading tears and distortions are likely to occur; thus, the epithelial attachment is often torn away from its original position, making the correct interpretation of the specimens difficult and sometimes impossible.

It is difficult in this country to procure human jaw specimens for histological investigations because of the prevailing legislation concerning autopsy. This fact is perhaps the main reason why knowledge concerning the histology of the gingival crevice has been gained earlier in Europe than in the United States. It is a fact that there are still men engaged in dental histological research in the United States who are unable to obtain freshly fixed human jaw specimens of different ages for their studies. Without such specimens, however, a full understanding of the changes occurring in the gingival tissues during life is impossible. Some investigators used animal tissues for their studies. From the histological findings in animal specimens, conclusions concerning conditions in man cannot be drawn except with wide reservations. In the teeth of sheep, for instance, which were in the past frequently used for microscopic studies, the entire enamel surface is covered by a layer of cementum; this creates a condition basically different from that which is found on the surface of human teeth (see also p. 281).

Furthermore, it is necessary to decalcify teeth and bone in order to be able to section them on the microtome and to prepare microscopic specimens. In the process of decalcification the enamel is dissolved. Thus, it becomes difficult to visualize the anatomical relationships present before the loss of the enamel. In a decalcified specimen, no enamel is visible; the empty space crownward from the cemento-enamel junction between dentin and gum tissue has often been erroneously called the "gingival space," and, consequently, the bordering epithelium was named the "pocket epithelium." These two terms were frequently misinterpretations: the empty space was not the gingival space, but the space formerly occupied by the enamel; consequently, at least a part of the pocket epithelium was in reality the epithelial attachment.

It is difficult to keep pathological and normal conditions apart. To some extent this distinction is still unsettled and subject to discussion. However, there is no doubt that many specimens heretofore used in text-books and publications to illustrate the relationship between gingival tissues and tooth surface showed distinctly pathological changes, particularly extensive destruction of the epithelial attachment by inflammatory processes. The type of human material that is available for histological investigations is very likely to cause a preponderance of pathological conditions in our specimens. For social reasons we hardly ever have access to jaw specimens from people with clean teeth and good oral health.

For these reasons the earlier investigators did not know about

an epithelial attachment. Gottlieb gave the first description of it in 1921. In an article by Hermann Prinz (*Dental Cosmos*, 1926), Gottlieb's findings were first mentioned in American literature. Skillen and Mueller were the first ones in the United States to give the right conception of gingival crevice and epithelial attachment, using their own histological specimens (1927). Prinz and Orban, in 1928, gave a correct description of the epithelial attachment in their text-books. In 1930, in the *Text-book of Histology*, by Maximow and Bloom, a correct illustration of the epithelial attachment in a young human tooth is reproduced; here, for the first time in a book on general histology, the description of the gingivæ is modified according to recent findings.

MICROSCOPIC APPEARANCE OF THE GINGIVAL CREVICE.

How is it possible, in studying decalcified specimens of human teeth and jaws, to determine where the bottom of the gingival crevice was located during life? Several landmarks help us properly to interpret specimens of this kind. These landmarks are:

1. Location of the enamel cuticle.
2. Location of the subepithelial infiltration.
3. Difference in appearance of epithelial attachment and crevice epithelium.
4. Location of calculus in the crevice.

1. **Location and Significance of the Enamel Cuticle.**—The enamel cuticle is a thin layer of organic tissue that is attached to the surface of the enamel. In young individuals where only a part of the enamel is erupted, the cuticle covers this exposed portion of the enamel. After the enamel is dissolved in the preparation of the specimen, the part of the cuticle crownward from the bottom of the gingival crevice will be left behind and will be visible in the specimen as a fine thread extending from the bottom of the gingival crevice in the direction of the former surface of the enamel (Figs. 224, 230).

Fig. 223 shows one of the sections that was used in drawing the diagram, Fig. 222. It represents the labial side of an upper incisor of a child with clinically normal, healthy gums. The enamel has been lost in the preparation of the specimen; the outline of the cervical portion of the enamel is shown by the epithelial attachment, *EA*. Toward the gingival margin the epithelium of the epithelial attachment becomes thicker; in a higher magnification of the gingival margin (Fig. 224) it can be seen that the epithelium changes its character: the cells become lighter and larger and show

signs of beginning hornification. At *BC*, the cuticle is detached from the inner surface of the epithelial attachment indicating the location of the bottom of the gingival crevice. The gingival crevice extends from *BC* to *GM*; its depth is about 0.03 mm., or practically

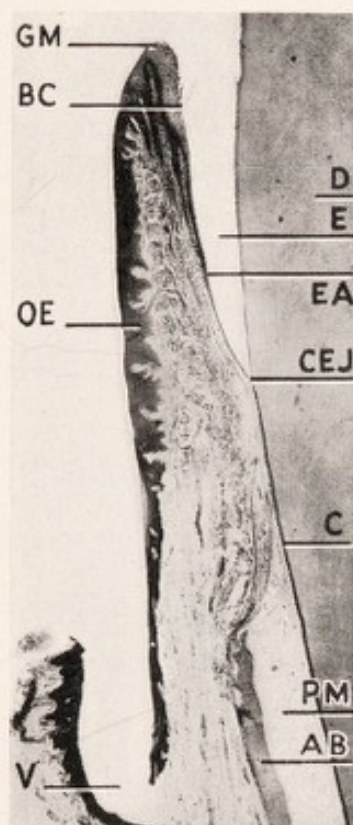


FIG. 223.—Photomicrograph of one of the specimens that were used in drawing the diagram, Fig. 222. Labial side of an incisor of a child, aged ten years. D, dentin; E, enamel¹; CEJ, cemento-enamel junction; EA, epithelial attachment; BC, bottom of gingival crevice; GM, gingival margin; GM-BC, gingival crevice; OE, oral epithelium; V, labial vestibulum; C, cementum; AB, alveolar bone; PM, periodontal membrane.

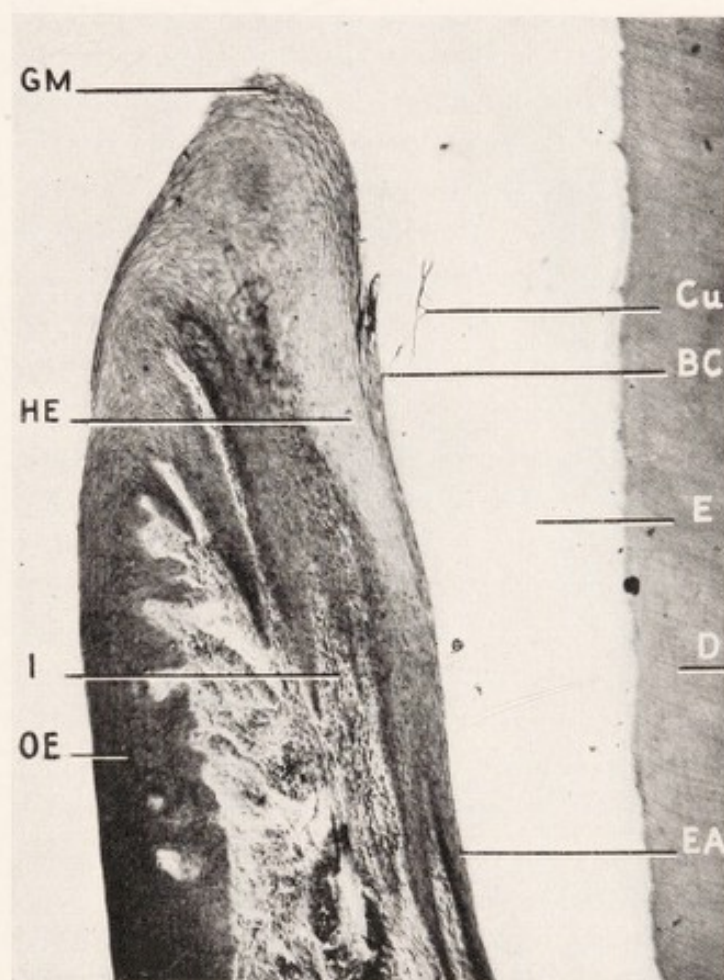


FIG. 224.—Higher magnification of the gingival margin in Fig. 223. D, dentin; E, enamel; EA, epithelial attachment to the enamel; Cu, enamel cuticle; BC, bottom of the gingival crevice; HE, beginning hornification of the cells of the epithelial attachment initiating the process of detachment of the attachment from the enamel surface; GM, gingival margin; OE, oral epithelium; I, subepithelial round-cell infiltration.

zero. The cuticle that followed the outline of the enamel surface before decalcification lost its support when the enamel dissolved, and became folded.

¹ In this, as well as in all similar specimens illustrated in Chapters X and XI, the enamel was lost during the preparation of the sections. Therefore, it must be understood that E in these illustrations indicates the space formerly occupied by the enamel.

It is appropriate to recall in this connection the new conception concerning the etiology and structure of the dental cuticle. According to Gottlieb, we have to differentiate between two types of cuticle: the primary cuticle and the secondary cuticle. The primary cuticle, the last product of the ameloblasts, is formed on the enamel surface before the ameloblasts degenerate and disappear. It is about 1 micron thick and usually becomes calcified. In the event of decalcification, it disappears with the enamel. The secondary cuticle is a horny structure formed by the squamous cells of the epithelial attachment. It is homogeneous. It appears bright pink when stained with eosin, and is about 7 microns thick. The secondary cuticle can be found on the enamel as well as on the root surface and, therefore, has been called the "cuticula dentis." As horn is very resistant to alkalis and acids, the secondary cuticle withstands decalcification during the preparation of histological sections, and is frequently found in decalcified sections. The bottom of the gingival crevice is determined by the point of separation of the cuticle from the crevice epithelium (see also p. 68).

2. Location of the Subepithelial Infiltration.—Another important indicator for localization of the bottom of the gingival crevice in the specimens is the position of the subepithelial infiltration. The great majority of microscopic specimens of gingival crevices in man as well as in animals shows a certain amount of round-cell infiltration in the tissue immediately underlying the bottom of the gingival crevice, probably as the result of a slight amount of irritation existing in every gingival crevice. The location of this round-cell infiltration aids in determining where the bottom of the crevice is located.

In Fig. 224 we find the round-cell infiltration at *I*, corresponding to the location of the bottom of the gingival crevice at *BC*. In a high-power photograph of the cemento-enamel junction of this specimen, no trace of inflammatory infiltration is found (Fig. 225).

3. Difference in Appearance of Epithelial Attachment and Crevice Epithelium.—The epithelial attachment consists of several layers of stratified squamous epithelial cells. The side of the epithelial attachment that faces the enamel and is united with it is a replica of the enamel surface at that place.

The crevice epithelium is usually thicker than the epithelial attachment and is characterized by high papillæ projecting into the subepithelial connective tissue. The surface of the crevice epithelium borders the gingival crevice, and its outline depends entirely upon conditions present in the crevice; therefore, it never

appears as even and uniform as the epithelial attachment. This can be best explained by an actual case. On the distal side of a cuspid, the condition illustrated in Fig. 226 was found. The distance from the gingival margin to the cemento-enamel junction was measured and found to be 3.5 mm. The question arises, how much of this distance is actually the depth of the gingival crevice? A higher magnification of the area around the cemento-enamel junction shows that the epithelium from *CEJ* to *BC* follows in a smooth curve the original outline of the enamel surface; at *BC* we find the enamel cuticle, indicating

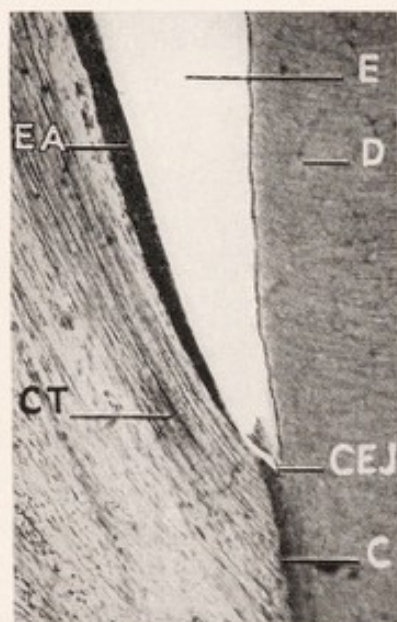


FIG. 225.—High magnification of the cemento-enamel junction in Fig. 223. D, dentin; E, enamel; CEJ, cemento-enamel junction; EA, epithelial attachment to the enamel; CT, connective tissue; C, cementum. Notice the complete absence of round-cell infiltration in the subepithelial connective tissue.

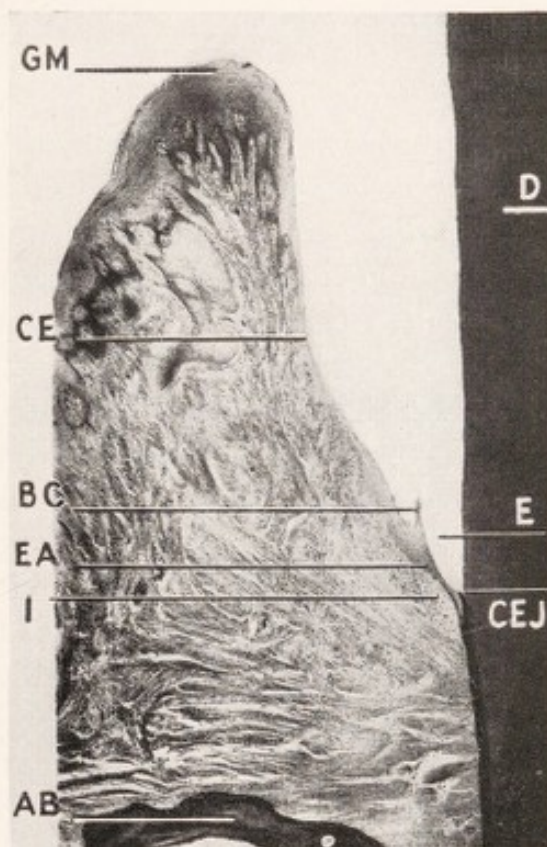


FIG. 226.—Gingival crevice on the distal side of a lower cuspid. D, dentin; E, enamel; CEJ, cemento-enamel junction; EA, epithelial attachment to the enamel; BC, bottom of the gingival crevice; CE, crevice epithelium; I, subepithelial round-cell infiltration; GM, gingival margin; AB, alveolar bone. The gingival crevice extends from GM to BC; its depth is 3 mm.; the length of the epithelial attachment to the enamel (BC to CEJ) is 0.45 mm.

that *BC* is the bottom of the gingival crevice and that, consequently, the epithelium between this point and the cemento-enamel junction is the epithelial attachment. This location of the bottom of the crevice is corroborated by the location of the subepithelial infiltration. The gingival crevice in this case extends from *BC* to *GM*.

4. **Location of Calculus in the Crevice.**—Calculus is present in a large number of the gingival crevices of which decalcified histological sections were studied. Since calculus is a product of the saliva, it can be deposited only on that part of the tooth surface that is accessible to the saliva. The most apical extension of the mass of calculus found in any specimen of gingival crevice indicates the location of the bottom of this crevice (see Fig. 238).¹

MEANING OF THE TERM EPITHELIAL ATTACHMENT.

The existence of an epithelial attachment is still doubted by some men. However, as a gradually increasing number of investigators succeed in preparing good histological slides of both animal and human specimens, they will undoubtedly be convinced of the existence of such an organic connection between tooth surface and epithelium. At present we are not able to give exact information as to the way in which this attachment of the epithelial cells to the enamel or to the cuticle on the tooth surface takes place. Further investigations in this field will be necessary to reveal the histological characteristics of this union. But there is such a union, and we have to take this existing organic connection between tooth surface and epithelial attachment into consideration in a clinical as well as in a microscopic study of the gingival tissues.

A method for determining whether the epithelial attachment is "attached" to the enamel or is merely in "close contact" with it is the study of the histological findings made on extracted teeth. During extraction the tissues are, as a rule, torn from the body along the line of least resistance. In examining extracted teeth from young individuals, the epithelial attachment can frequently be found still in connection with the enamel surface, indicating that the connection between tooth surface and epithelium was stronger than the connection between epithelium and underlying tissue.

Figs. 227 and 228 show such a case. The photomicrograph was taken from the mesial side of a lower bicuspid which was extracted because of a deep cavity and pulpitis. The extracting forceps held the tooth in its bucco-lingual diameter; since the area reproduced in Figs. 227 and 228 was on the mesial side, it could not be touched by the forceps. Any soft tissue found in this location must, consequently, have been attached to the tooth surface. The mesial side of the tooth surface near the cemento-enamel junction is covered by a thin layer of soft tissue extending over the enamel as well as over the cementum. In a higher magnification of the cemento-

enamel junction, this soft tissue adherent to the tooth can be recognized as squamous epithelium. This proves that the mechanical attachment of the epithelium to enamel and cementum was strong enough to withstand the gross laceration of the tissues during extraction.

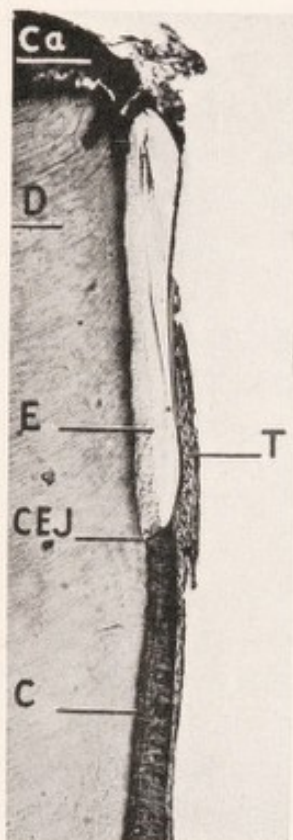


FIG. 227.

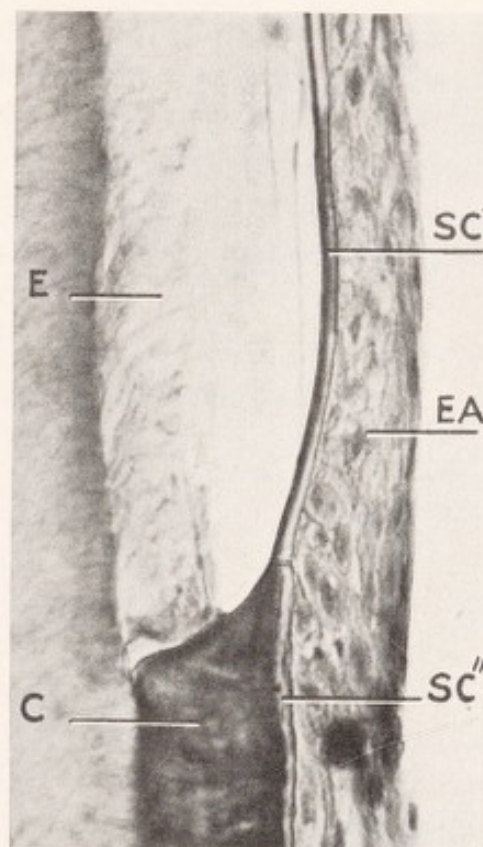


FIG. 228.

FIGS. 227 and 228.—The attachment of the epithelium to the enamel and to the cementum.

FIG. 227.—Cemento-enamel junction of a human lower bicuspid. The tooth was extracted, decalcified, and embedded in celloidin. D, dentin; E, enamel; C, cementum; CEJ, cemento-enamel junction; Ca, caries; T, soft tissue attached to the enamel and to the cementum.

FIG. 228.—Higher magnification of the cemento-enamel junction. E, enamel; C, cementum; SC', hornified secondary cuticle on the enamel; SC'', hornified secondary cuticle on the cementum; EA, stratified squamous epithelium of the epithelial attachment. This epithelium was torn off from the subepithelial connective tissue during extraction of the tooth and remained attached to the tooth surface. (Kronfeld, Jour. Am. Dent. Assn.)

In this connection, the point is frequently brought up by practitioners that it is possible to pass an explorer between the enamel and the gingival tissues of young patients. But this is no argument against the existence of an epithelial attachment to the enamel, since it is possible to separate by means of force structures that originally were united.

In summarizing, the following description of the relationship between gingival soft tissues and tooth surface in young persons can be given: At the time when the tooth reaches the occlusal plane, the erupted part of the enamel, the "clinical crown," includes two-thirds to three-fourths of the length of the crown; the cervical portion of the enamel is still in organic connection with the epithelial attachment. The bottom of the gingival crevice is located on the enamel surface at the borderline between crevice epithelium and epithelial attachment. The gingival crevice extends from this point crownward to the free gum margin; the walls of the gingival crevice are formed by the crevice epithelium on the outer side and by the enamel or the enamel cuticle on the inner side.

CLINICAL SIGNIFICANCE OF THE EXISTENCE OF AN EPITHELIAL ATTACHMENT TO THE ENAMEL.

Since in young people the cervical part of the enamel is in organic junction with the epithelial attachment, this part of the crown is not exposed to the influences of the oral cavity and, therefore, cannot decay. This observation was comprehensively discussed by G. V. Black. He showed specimens of extensive approximal decay in the teeth of young patients in which the gingival portion of the enamel was entirely free from decay. Black did not know about the existence of an epithelial attachment to the enamel; he thought that the bottom of the gingival crevice was at the cemento-enamel junction. Consequently, he drew the only logical conclusion, namely, that the gingival crevice is a self-cleansing area and that the enamel is protected from caries by the free margin of the gum. From the findings described in the foregoing paragraphs, it is clear that the presence of smooth, intact enamel in the gingival portion of young, badly decayed teeth must be explained differently. The cervical portion of the enamel is not protected by a gingival crevice, but it is still in organic connection with the gingiva by the epithelial attachment; therefore, it is inaccessible to bacteria and other oral influences and cannot decay. In a tooth like the one shown in Figs. 222 and 223, only the portion of the enamel crownward from *BC* (clinical crown) is accessible to caries. The enamel from *BC* to *CEJ* has not yet erupted into the oral cavity. If the tooth were extracted, the cervical portion of the enamel would appear white and intact even in the event of extensive gingival or approximal decay (see Fig. 242).

Another clinical observation of importance is the relationship

between epithelial attachment and deposits of calculus. For obvious reasons, calculus can be deposited only on the surface of the clinical crown. Since the bottom of the gingival crevice in young people is located on the enamel surface, no calculus can be deposited on the portion of the enamel rootward from the bottom of the crevice. On extracted teeth of young individuals, serumal calculus is frequently found deposited in a more or less complete ring around the cervical portion of the enamel. Between the deposit and the cemento-enamel junction there is a circular area of clean, white enamel without any calculus on it. In correlating this clinical observation with the microscopic findings in young teeth, it can be easily understood that the white area of enamel rootward from the deposit corresponds to the area between *BC* and *CEJ* which was not yet erupted (see Fig. 254).

LOCATION OF GINGIVAL CREVICE AND EPITHELIAL ATTACHMENT ON THE TOOTH SURFACE AT DIFFERENT PERIODS OF LIFE.

When a tooth reaches the occlusal plane, about one-third or one-fourth of the enamel near the cemento-enamel junction is in organic connection with the epithelial attachment. Rootward, the epithelial attachment ends at the cemento-enamel junction; however, this condition is not permanent. Gradually, more and more of the enamel is exposed by a separation of the epithelial attachment from the tooth surface. At the same time, the tissue of the free gingivæ is reduced by atrophy so that an increasing amount of enamel is exposed. The eruption of the tooth is continued although at a very much slower rate than in the first years of eruption. Due to this continued detachment, the bottom of the gingival crevice gradually approaches the cemento-enamel junction.

1. **Proliferation of the Epithelial Attachment Along the Cementum.**—Before the bottom of the gingival crevice reaches the cemento-enamel junction, another process invariably takes place: the deepest point of the epithelial attachment at the cemento-enamel junction begins to proliferate and grows downward along the cementum of the root (Fig. 229). Thus, at the time when the bottom of the gingival crevice reaches the cemento-enamel junction, the deepest point of the epithelial attachment is always found on the cementum. This rule has no exception.

Fig. 230 illustrates this condition. The bottom of the gingival crevice is located at *BC*, on the enamel surface. The deepest point

of the epithelial attachment has passed the cemento-enamel junction and is found on the cementum. The epithelial attachment, therefore, can be divided into an epithelial attachment to the enamel (*BC* to *CEJ*) and into an epithelial attachment to the cementum (*EA''*). The subepithelial connective tissue is infiltrated with inflammatory exudate cells (polyblasts). The depth of the crevice is zero.

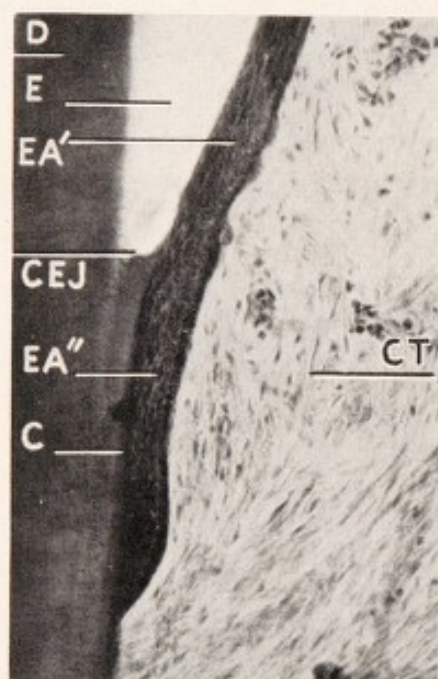


FIG. 229.—Proliferation of the epithelial attachment along the cementum. D, dentin; E, enamel; C, cementum; CEJ, cemento-enamel junction; EA', epithelial attachment to the enamel; EA'', epithelial attachment to the cementum; CT, subepithelial connective tissue, free of inflammation.

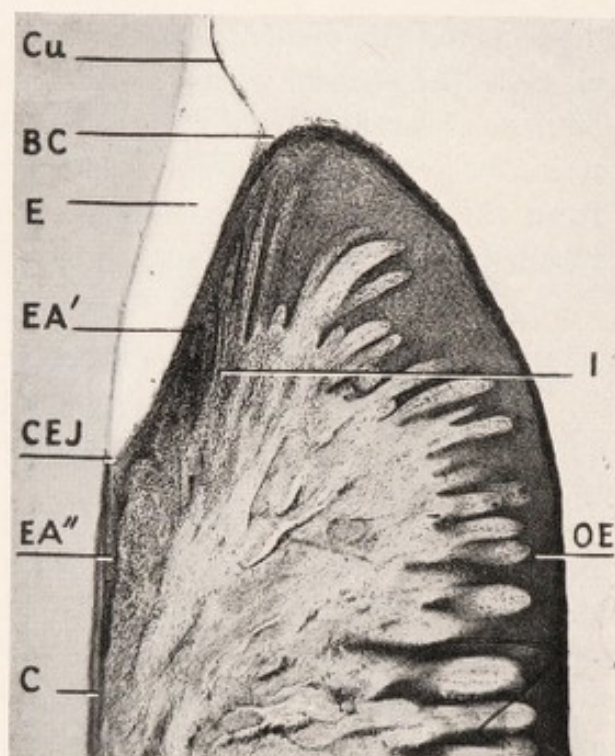


FIG. 230.—The deepest point of the epithelial attachment has passed the cemento-enamel junction and is found on the cementum. The bottom of the gingival crevice is still located on the enamel. E, enamel; C, cementum; CEJ, cemento-enamel junction; EA'', epithelial attachment to the cementum; EA', epithelial attachment to the enamel; BC, bottom of the gingival crevice; I, round-cell infiltration; Cu, cuticle; OE, oral epithelium.

As epithelium and enamel surface continue to separate, the bottom of the gingival crevice finally reaches the cemento-enamel junction (Fig. 231). In this specimen, the bottom of the gingival crevice and the cemento-enamel junction coincide. The epithelial attachment extends from the cemento-enamel junction rootward along the surface of the cementum. The epithelial lining of the gingiva is intact; the crevice has no depth.

2. **Microscopic Structure of the Epithelial Attachment.**—The epithelial attachment consists of the typical stratified squamous epithe-

lial cells that build up the oral epithelium. We can differentiate between two sides of the epithelial attachment: the inner side, connected with the subepithelial connective tissue, and the outer side, facing the tooth and attached to the tooth surface. The terms inner and outer side have been taken over from the gingival epithelium, the inner side of the gingival epithelium being the germinative or basal layer where new cells are formed, and the outer side being the hornified surface toward the oral cavity. Since

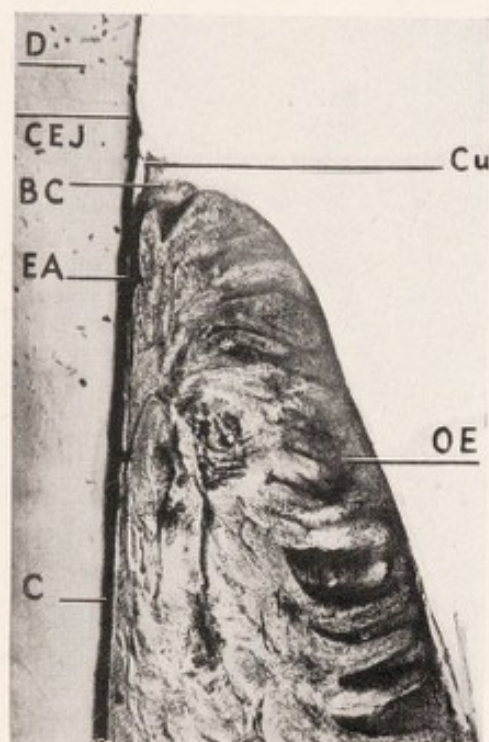


FIG. 231.—Bottom of the gingival crevice at the cemento-enamel junction. D, dentin; C, cementum; CEJ, cemento-enamel junction; BC, bottom of gingival crevice; Cu, cuticle; EA, epithelial attachment to the cementum; OE, oral epithelium. The depth of the crevice is zero.

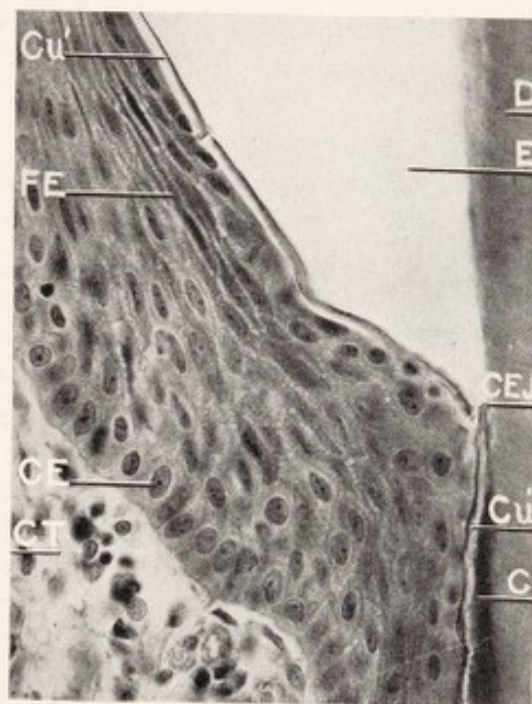


FIG. 232.—High magnification of the epithelial attachment at the cemento-enamel junction. D, dentin; C, cementum; E, enamel; CEJ, cemento-enamel junction; Cu', hornified (secondary) cuticle on the enamel; Cu'', hornified cuticle on the cementum; FE, flat epithelial cells with intercellular bridges; CE, cuboidal epithelial cells in the basal layer of the epithelial attachment; CT, subepithelial connective tissue. (Orban, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

the epithelial attachment is nothing but a duplicature or invagination of the oral epithelium around the tooth, the hornified cuticle on the tooth surface corresponds to the hornified layer on the surface of the gingival epithelium.

In a recent publication Orban described the form and the distribution of the cells of the epithelial attachment. He showed that the cells forming the epithelial attachment to the cementum are not different from the cells that constitute the epithelial attachment

to the enamel (Fig. 232). In both cases the inner surface of the epithelial attachment is formed by a layer of regular, cuboidal cells with round, dark nuclei. These cells are the basal cells of the epithelium. Toward the tooth surface these basal cells are followed by several layers of flat, squamous cells that are connected with each other by intercellular bridges. Next to the cementum, the nuclei are rather irregular and light; directly upon the cementum, a hornified cuticle is found. Toward the cemento-enamel junction, the cuticle becomes gradually thicker;

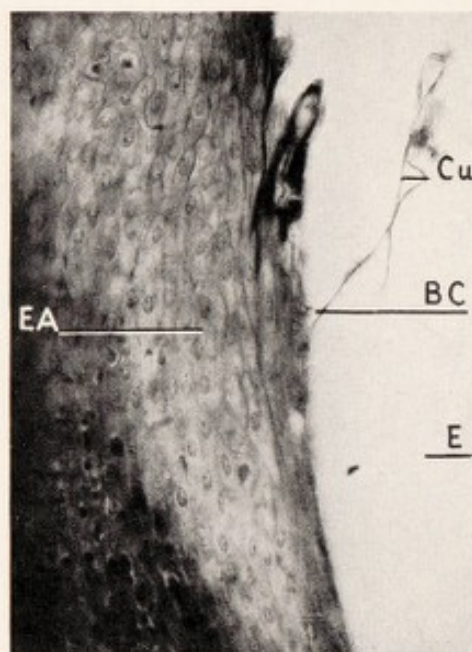


FIG. 233.—High magnification of the bottom of the gingival crevice. Degeneration of the superficial cells of the epithelial attachment and detachment of the cuticle. E, enamel; BC, bottom of gingival crevice; Cu, enamel cuticle; EA, large, light, hornifying cells of the epithelial attachment next to the bottom of the crevice.

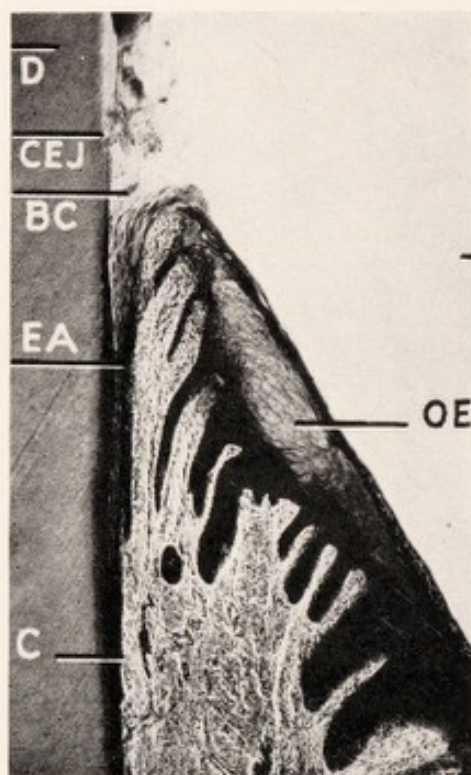


FIG. 234.—Bottom of the gingival crevice on the surface of the cementum. Labial surface, upper first bicuspid. D, dentin; C, cementum; CEJ, cemento-enamel junction; BC, bottom of the gingival crevice; EA, epithelial attachment to the cementum; OE, oral epithelium. The depth of the crevice is zero. (Kronfeld, Jour. Am. Dent. Assn.)

at the cemento-enamel junction the cuticle is continuous from the cementum to the enamel. The arrangement of the cells in the epithelial attachment to the enamel is the same as on the surface of the cementum: a regular, basal cell layer next to the subepithelial connective tissue, flat cells arranged parallel to the tooth surface, and a layer of cells with small, irregular nuclei next to the cuticle.

Further crownward on the enamel surface, close to the bottom of the gingival crevice, the epithelial attachment shows certain

characteristic changes. The cells begin to degenerate, thereby preparing the way for the separation of the epithelial attachment from the cuticle (Fig. 233). The epithelial cells next to the bottom of the gingival crevice are pale and irregular; their nuclei are shrunk and frequently disappear entirely. From these findings it is evident that the gingival crevice is actually formed by a degeneration of those cells of the epithelial attachment that are lying next to the tooth surface, and by a detachment of these cells from the cuticle.

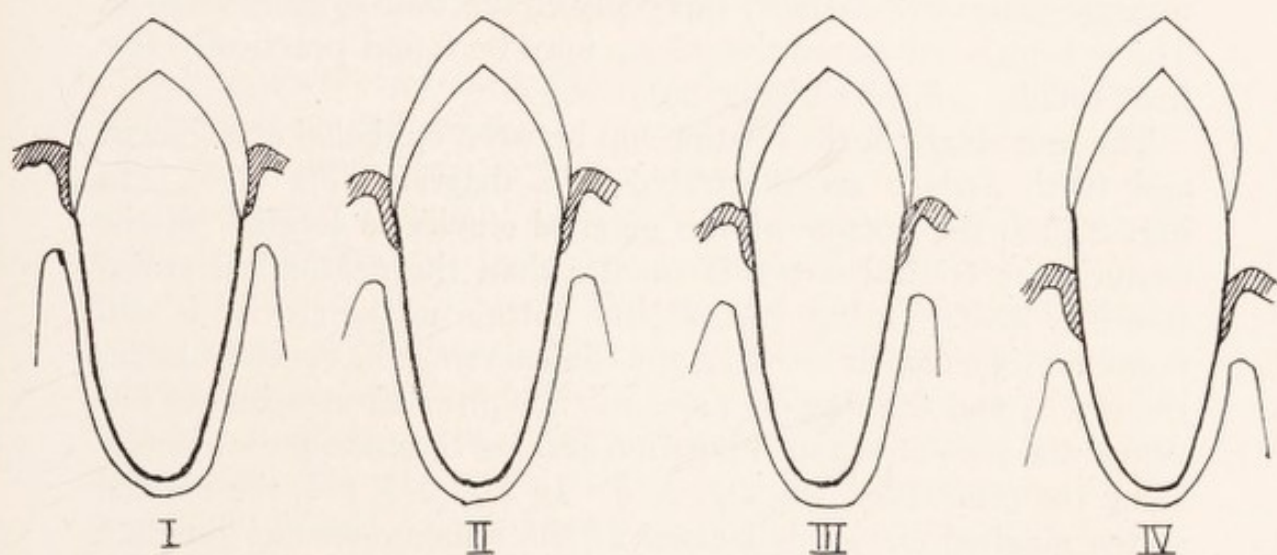


FIG. 235.—Diagram illustrating four different stages of the relationship existing between the tooth and the surrounding tissues, especially the epithelial attachment. I, bottom of the crevice on the enamel, deepest point of the epithelial attachment at the cemento-enamel junction; II, bottom of the crevice still on the enamel, but the deepest point of the attachment is already on the cementum; III, bottom of the crevice just at the cemento-enamel junction; the epithelial attachment on the cementum; IV, bottom of the crevice on the cementum; the deepest point of the epithelial attachment shifted further apically. (Orban and Mueller, Jour. Am. Dent. Assn.)

3. Gingival Crevice and Cemento-enamel Junction.—The cemento-enamel junction has no significance whatsoever in the detachment of the epithelial attachment from the tooth surface. The location of the bottom of the crevice at this point is nothing but a transitory stage in the course of the gradual separation of the soft tissues from the tooth surface. In the microscopic examination of a large number of human teeth of different ages, the bottom of the gingival crevice is only very seldom found at the cemento-enamel junction; but even in the few cases where it has been observed right at this point, the neighboring sections of the same specimen showed the location of the bottom of the crevice either crownward or rootward from the cemento-enamel junction.

Recently Becks made the assertion that under physiological conditions the detachment of the gingival tissues from the tooth

surface comes to a standstill once the bottom of the gingival crevice has reached the cemento-enamel junction and that any further apical shifting of the bottom of the crevice would have to be considered as the expression of a pathological process. These statements are not substantiated by clinical observations, nor has Becks produced actual histological proof for his assertions.

In later periods of life, the bottom of the gingival crevice is normally located on the surface of the cementum. The crevice may be extremely shallow, especially if the oral hygiene is good. The subepithelial connective tissue may be found practically free from cellular infiltration (Fig. 234).

The four stages in the relationship between epithelial attachment and tooth surface are illustrated in a diagram (Fig. 235). In Fig. 235, *I*, the bottom of the gingival crevice is located on the enamel; the clinical crown is smaller than the anatomical crown (see Fig. 223). In Fig. 235, *II*, the bottom of the crevice is still found on the enamel; however, the clinical crown has become larger than in *I*, and the deepest point of the epithelial attachment has passed the cemento-enamel junction and has begun to grow apically along the cementum (see Fig. 230). In Fig. 235, *III*, the bottom of the gingival crevice is located at the cemento-enamel junction (see Fig. 231). In Fig. 235, *IV*, finally, the bottom of the crevice is found on the surface of the cementum; the clinical crown is greater than the anatomical crown (see Fig. 234). It must be understood, however, that these diagrams do not mean that the same condition must necessarily be found all around the tooth. On the contrary, in the histological examination of human jaw material, different conditions are usually encountered on different sides of a tooth, and again different conditions are encountered on various teeth in the same mouth. Frequently the bottom of the gingival crevice is located on the surface of the enamel on one side of a tooth, and on the surface of the cementum on the opposite side of the same tooth. Only in very young individuals is the condition illustrated in Fig. 235, *I*, usually found all around the teeth, and the same is sometimes true in old persons for the condition in Fig. 235, *IV*.

It has been the author's observation, in teaching the subject of the gingival crevice, that the student easily makes the mistake of confusing "downward growth of the epithelial attachment" with "deepening of the crevice." These two terms are by no means synonymous. The shifting of the bottom of the gingival crevice apically may continue all during life without ever being accompanied by a deepening of the crevice. Especially will this be true of mouths

in good, hygienic condition. In such mouths, crevices with no depths at all can frequently be observed; in youth, they are located on the surface of the enamel, in advanced age, on the surface of the cementum. In some of the microscopic specimens reproduced in this book, attention has been called to the presence of such extremely shallow crevices in different locations on the tooth surface.

SIGNIFICANCE OF THE GRADUAL DETACHMENT OF THE GINGIVAL SOFT TISSUES FROM THE TOOTH SURFACE.

The separation of epithelial attachment from the root surface proceeds at no definite rate of speed. It is rapid in the first years after the tooth appears in the mouth; it slows up later. The rate of detachment, clinically known as recession, is subject to great individual variations. In some individuals, the cervical portion of the root may be found exposed in almost all teeth at a relatively early period of life; in others, the bottom of the crevice is located on the enamel until late in life. This observation has led to a discussion of whether the continued detachment of the epithelial attachment from the tooth surface and the final exposure of the cementum should be considered a pathological or a physiological process. Ever since Gottlieb's first publication on the epithelial attachment, there has been a diversity of opinion concerning this particular point, and at the present time the question is by no means settled. Gottlieb, Orban, E. Mueller, the author, and several other investigators consider the continued detachment of the epithelial attachment and the proliferation of the epithelium along the root surface a physiological process, namely, a regressive change associated with the aging of the organism. Another group of investigators, the best known of whom are Bauer, Häupl, and Lang, look at the proliferation of the epithelium along the root surface as a strictly pathological process, namely, as a result of mechanical injuries and of subepithelial inflammation. The latter group of authors agree that an epithelial attachment to the enamel exists when the tooth reaches the occlusal line. But they consider only the condition present at that time as normal or physiological; any further detachment and especially the proliferation of the epithelium along the cementum is looked upon as a pathological manifestation.

Gottlieb speaks of the various successive stages as they are illustrated in Fig. 235 as "stages of eruption," the meaning of the word eruption being that the tooth under normal conditions continues to move occlusally although at a much slower rate than

in childhood. During this occlusal movement the gingival soft tissues are separated from the tooth at the bottom of the crevice. The crown is continuously worn off by abrasion; at the same time cementum is deposited on the surface of the root. As long as all these processes occur slowly and uniformly, they are expressions of the natural process of senescence. Through detachment of the gingival tissues at the bottom of the crevice, a fairly uniform length of the clinical crown is maintained despite abrasion. Through formation of cementum the root is elongated accordingly, keeping the clinical root an adequate length.

There is no doubt that the downward growth of the epithelium may also be the expression of a pathological process; this is especially true when it occurs abnormally fast, exposing large areas of the root at an early period of life. The pathological conditions resulting from deviations from the normal relationship between tooth and investing tissues will be discussed in Chapter XI.

The rôle of subepithelial round-cell infiltration deserves special consideration as this infiltration is the main issue in the discussion on the proliferation of the epithelial attachment along the tooth surface. In by far the greatest number of human specimens, inflammatory round cells (polyblasts) are found in the tissue bordering the crevice, and it does not seem very far-fetched to hold this inflammation responsible for the changes in the epithelium. However, it is the author's opinion, gained from the study of a large number of human specimens of all ages and in all possible gradations between normal and badly diseased, that a direct causal connection between inflammation and downward growth of the epithelium along the root surface cannot be demonstrated. Proliferation of the epithelial attachment along the cementum can be observed in specimens where the bottom of the crevice and the subepithelial infiltration were far away from the epithelial attachment; on the other hand, specimens are found with severe inflammation but with no changes in the epithelium. Gottlieb's opinion is that a gradual decrease in vitality of the tooth surface is the factor that stimulates the epithelium to proliferate rootward; it is difficult to find any other working hypothesis or explanation that would be more helpful in the understanding of the various phases of this problem. But the author is aware that it is impossible to settle this question definitely until human jaw specimens of individuals with excellent oral hygiene and perfectly healthy, normal gums will be available for histological examination. In such specimens the inflammatory component can be excluded entirely from the

investigations, and then it will be possible to decide how the epithelial attachment acts in complete absence of inflammation. Up to this time, such specimens have not been reported.

EPITHELIAL ATTACHMENT AND GINGIVAL CREVICE IN ANIMALS.

Since we learned about the changes that occur in the epithelial attachment of human teeth, it has been interesting to compare these changes with what could be found in animals under similar conditions. For this purpose the author, in collaboration with Ullik, has examined the teeth of small, wild mammals that have not been subject to the influences of domestication, among them the hedgehog (*Erinaceus europeus*). This animal fulfilled all postulates necessary for such a comparative investigation. It is omnivorous; its teeth show histologically a great resemblance to human teeth; it grinds down its teeth during life, like man; it can be examined easily direct from wild life without any possible interference of domestication and captivity. In examining hedgehogs of different ages, all stages indicated in Fig. 235 could be found. In young hedgehogs, the gingival tissues are united with the enamel by a typical epithelial attachment. The bottom of the gingival crevice is located on the enamel surface. The epithelial attachment ends at the cemento-enamel junction. Moreover, in the young animals, high cusps and thin cementum are found, analogous to the conditions in man. In older hedgehogs, the epithelial attachment grows apically along the surface of the cementum (Fig. 236). In this illustration the bottom of the gingival crevice on the enamel is very clearly indicated by the point of detachment of the enamel cuticle. In some teeth a slight subepithelial infiltration is found; others are without any inflammation.

In old hedgehogs, the same regressive changes were observed that are found in man. The cusps are worn flat by occlusal wear; the size of the pulp chamber is greatly reduced by secondary dentin formation. Heavy layers of cementum are present on all root ends and in the bifurcation of the molars. The bottom of the gingival crevice has passed the cemento-enamel junction and is found on the root surface, so that the clinical crown includes part of the cementum surface. The depths of the gingival crevices in the old animals are about the same as in the young ones. These findings in wild living animals suggest that the identical changes that are found with advancing age in human teeth are characteristic of all mammals with teeth similar to human teeth.

It may be of interest in this connection to consider the forms of epithelial attachment found in mammals with teeth having a structure basically different from that of human teeth. These animals can be divided into two major groups: rodents and herbivorous animals.

The incisors of all rodents are continuously growing teeth: that means that the constant loss of tooth substance by abrasion at the

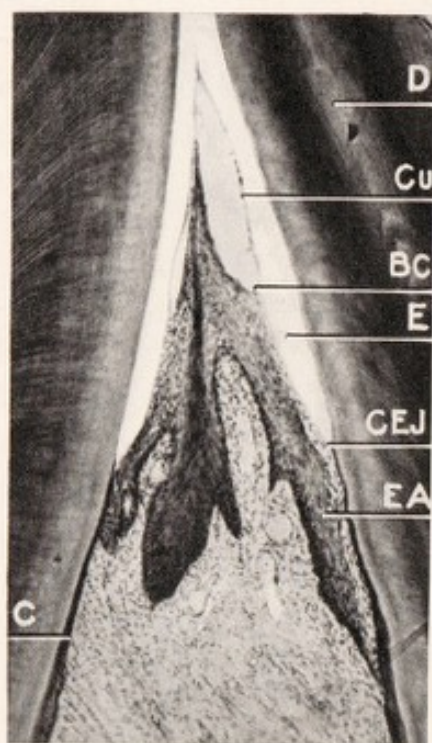


FIG. 236.—Interproximal space between two incisors of adult hedgehog. Proliferation of the epithelial attachment along the cementum on both teeth. D, dentin; E, enamel; C, cementum; CEJ, cemento-enamel junction; BC, bottom of the gingival crevice on the enamel surface; Cu, enamel cuticle; EA, epithelial attachment to the cementum. A slight sub-epithelial round-cell infiltration is present. (Gottlieb, *Fortschr. d. Zhk.*, courtesy of Georg Thieme, Leipzig.)

occlusal surface is replaced by the equally constant formation of new material at the apical end of the tooth. Consequently, teeth of rodents develop no fixed apical foramen as do the teeth of men. The distribution of enamel and cementum in rats' teeth is as follows: The labial (outer) surface of the tooth is covered with enamel over its entire length; the entire lingual surface is covered with cementum. Consequently, the cemento-enamel junction is represented by two lines running lengthwise on the mesial and distal surface along the entire tooth. Since the oral epithelium is attached to the entire circumference of the tooth at the bottom of the gingival crevice, we find, on the labial surface, an epithelial attachment on the enamel, and on the lingual side, an epithelial attachment on the cementum. Around the entire tooth, the deepest point of the epithelial attachment is continuously migrating toward the apical end as the tooth erupts. At the bottom of the gingival crevice, the epithelium is continuously being separated from the enamel on the

labial side and from the cementum on the lingual side. Thus, a uniform length of clinical root and clinical crown is maintained despite the continuous growth. Almost identical conditions are found on the molars of some rodents, such as the rabbit and the guinea-pig. The molars of these animals are growing continuously; they have a wide open apical foramen. New tooth

substance is constantly being built at the apical end, while a corresponding amount of tooth structure is constantly worn off at the occlusal end. Consequently, the epithelial attachment shifts apically at the same rate of speed at which the tooth moves occlusally. These conditions, however, are not found in the molars of all rodents. The rat's molars, for instance, are built very much like human molars.

The teeth of herbivorous animals, represented by sheep, goats, and cattle, have long, enamel-covered crowns and relatively short roots. The most important peculiarity of these teeth is that the entire enamel is covered with cementum, the enamel being exposed only on the worn occlusal surface. The epithelial attachment, therefore, is always located on the cementum. It grows apically along the cementum surface and becomes detached from it at the bottom of the gingival crevice.

DEPTH OF THE GINGIVAL CREVICE.

The depth of the gingival crevice is subject to wide individual variation. Orban and Köhler measured 356 crevices on specimens of human teeth. Only such crevices as had an intact epithelial lining were measured. In 15 crevices the depth was zero. In 17 crevices the depth varied from 0 to 0.1 mm. One hundred and twenty-nine crevices measured between 0.1 and 0.5 mm., 102 between 0.5 and 1 mm., and 17 between 1 and 1.5 mm., 21 between 1.5 and 2 mm.; 13 crevices ranged between 2 and 3 mm., and 2 ranged between 4 and 6 mm. This gives an average depth of about 0.8 mm. However, it must be remembered that the specimens were obtained from individuals who habitually neglected oral hygiene and dental care; in people with better oral conditions, shallower crevices must be expected.

The age of the individual has no influence upon the depth of the crevices; under good hygienic conditions, shallow crevices are found both clinically and microscopically in adults as well as in children.

The findings with regard to the depth of the gingival crevice can be summarized as follows:

The depth of the gingival crevice varies from zero to several millimeters; the majority of the crevices in human teeth measure between 0.1 and 1 mm.

The depth of the crevice varies inversely to the health of the mouth; that is, the better the oral hygiene, the shallower are the crevices.

The location of the crevice on the tooth surface has no influence upon its depth; shallow crevices may be found on the enamel surface in young individuals and on the root surface in older persons.

The shallower the crevice, the fewer are the chances for a possible development of pathological conditions in the crevice.

Since the shallowest crevices that were actually found have no depth at all, a depth of 0 mm. must be considered as the ideal, and our therapeutic measures must tend to create and to maintain crevices that approach as nearly as possible this ideal condition.

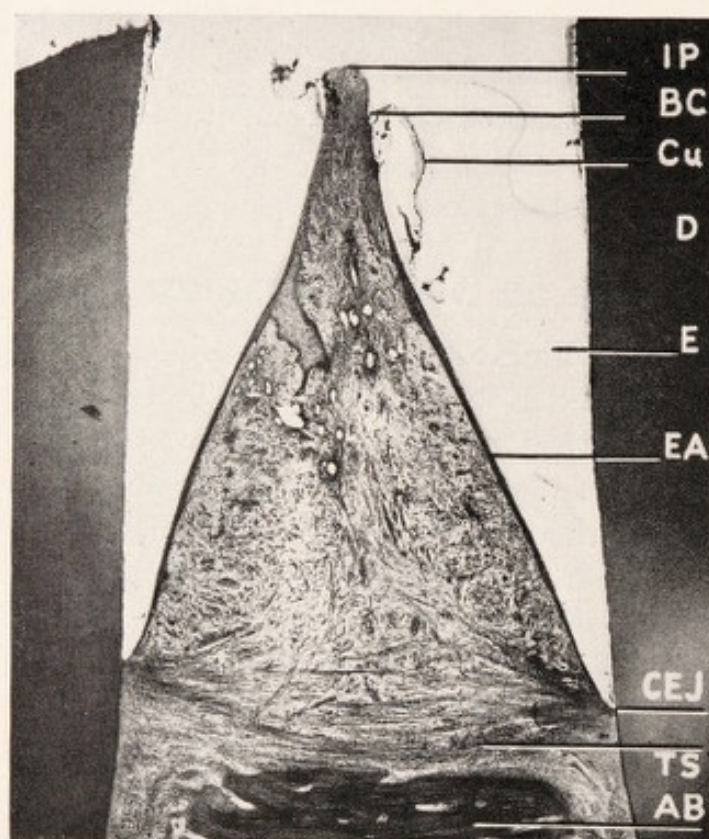


FIG. 237.—Interproximal space between the lower first and second bicuspid in a boy, aged eight years. The teeth have just erupted into the oral cavity. The epithelial attachment extends from the cemento-enamel junction crownward almost to the level of the contact point. IP, tip of interdental papilla; BC, bottom of the gingival crevice; Cu, cuticle; EA, epithelial attachment to the enamel; E, enamel; D, dentin; CEJ, cemento-enamel junction; TS, transeptal fibers; AB, alveolar bone.

HISTOLOGY OF THE INTERDENTAL TISSUES.

All the specimens of human gingival crevices illustrated were taken from the labial or lingual side of the teeth. We shall now consider the conditions and changes that are found in the area between the teeth. Fig. 237 shows the soft tissues between the crowns of the lower first and second bicuspid in a boy, aged eight years. Only about one-half of the crowns had erupted into the

oral cavity. The bottom of the gingival crevice is indicated by the presence of the enamel cuticle, *Cu*, and by the location of a slight round-cell infiltration. From *BC* to the cemento-enamel junction, *CEJ*, the epithelium is still in organic connection with the enamel. The gingival crevice in this case measures about 0.05 mm., or practically zero.

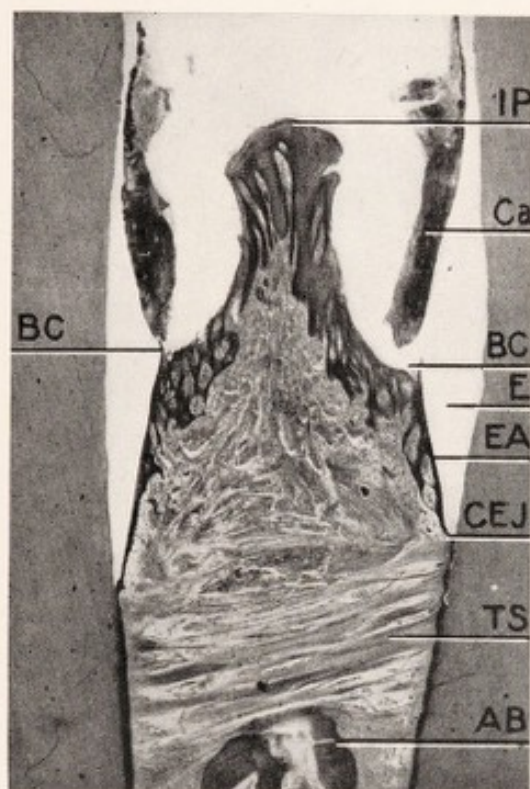


FIG. 238.—Interproximal space between upper central and lateral incisors of a boy, aged fourteen years. The bottom of the gingival crevice is located on the enamel; the epithelial attachment ends at the cemento-enamel junction. IP, tip of interdental papilla; BC, bottom of the gingival crevice; Ca, calculus in the gingival crevice; EA, epithelial attachment to the enamel; E, enamel; CEJ, cemento-enamel junction; TS, transeptal fibers; AB, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

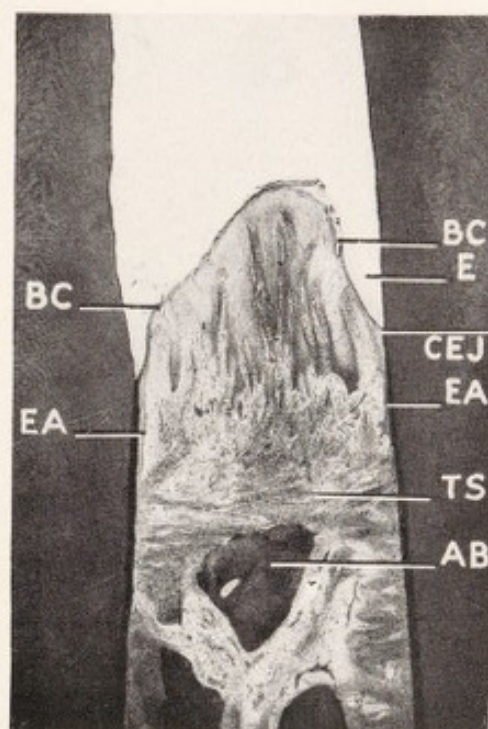


FIG. 239.—Interproximal space between lower lateral incisor and cuspid in a young adult. The bottom of the gingival crevice is located on the enamel, close to the cemento-enamel junction; the epithelial attachment extends down upon the cementum. BC, bottom of the gingival crevice; E, enamel; CEJ, cemento-enamel junction; EA, epithelial attachment to the cementum; TS, transeptal fibers; AB, alveolar bone.

A slightly different condition is found between the upper central and lateral incisor of a boy, aged fourteen years (Fig. 238). The bottom of the crevice is still located on the enamel surface; however, the part of the enamel that is still in connection with the epithelial attachment is greatly reduced compared with the condition present at the age of eight years (Fig. 237). The portion of the enamel between the bottom of the gingival crevice, *BC*, and the

cemento-enamel junction is only about one-fifth of the length of the anatomical crown. The epithelial attachment ends on both teeth at the cemento-enamel junction, corresponding to the diagram, Fig. 235, *I*. The depths of the crevices in this case are about 1.5 mm. on either tooth, measured from the tip of the interdental papilla to the bottom of the crevice at *BC*.

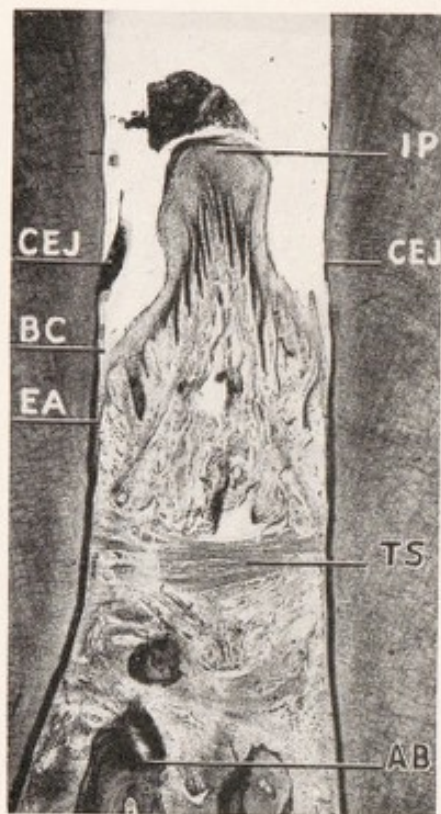


FIG. 240.—Interproximal space between two upper incisors in an adult. The bottom of the crevice has passed the cemento-enamel junction and is found just beyond the cemento-enamel junction on the root surface. IP, tip of interdental papilla; BC, bottom of gingival crevice; EA, epithelial attachment on the cementum; CEJ, cemento-enamel junction; TS, transeptal fibers; AB, alveolar bone.

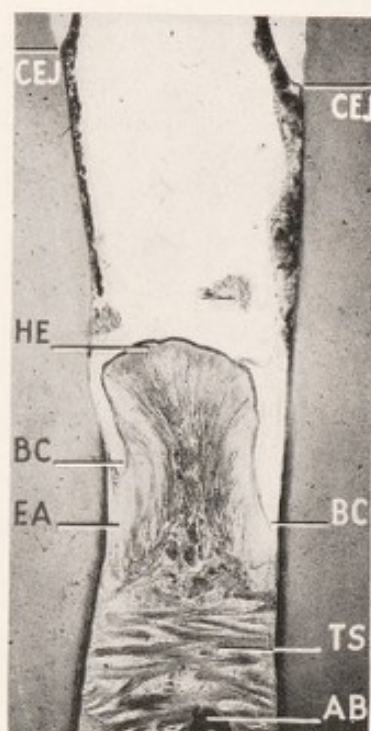


FIG. 241.—Interproximal space between two upper incisors in an adult. The bottom of the gingival crevice is located about 4 mm. rootward from the cemento-enamel junction. HE, hornified epithelium covering the interdental papilla; BC, bottom of the gingival crevice; EA, epithelial attachment on the cementum; CEJ, cemento-enamel junction; TS, transeptal fibers; AB, alveolar bone.

As the bottom of the crevice approaches the cemento-enamel junction, the epithelial attachment begins to proliferate along the surface of the cementum. This is illustrated in Fig. 239, taken from the field between a lower lateral incisor and cuspid in a young adult. The bottom of the gingival crevice is located at *BC*; only a small cervical portion of the enamel, from *BC* to *CEJ*, is still in connection with the epithelial attachment. The depth of the

gingival crevice on the left side of the papilla is about 1 mm.; on the right side there is practically no crevice present.

In the further process of detachment, the bottom of the crevice passes the cemento-enamel junction and is then found on the root surface. This condition is illustrated in Figs. 240 and 241. In Fig. 240 the bottom of the crevice is located on either side on the cementum just apically of the cemento-enamel junction; in Fig. 241 a considerable portion of the root surface is exposed between

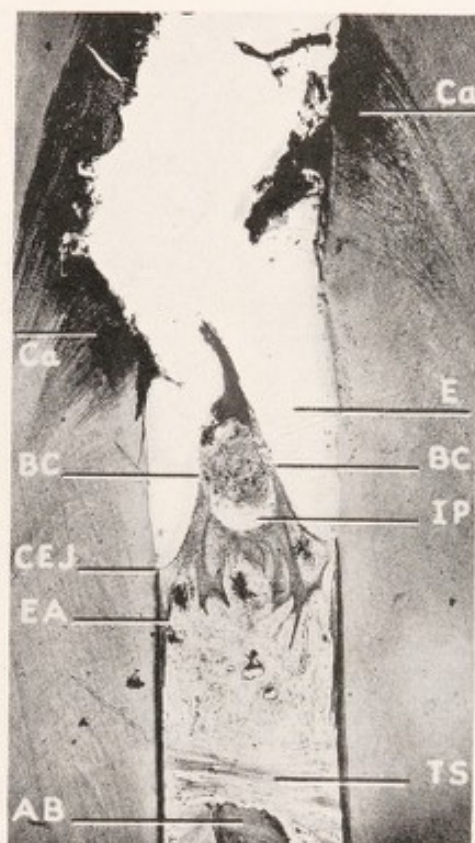


FIG. 242.—Caries in the interproximal space between the lateral incisor and cuspid in a man, aged twenty-four years. Ca, caries; IP, interdental papilla deformed by impinging débris; BC, bottom of gingival crevice on the enamel; E, enamel; CEJ, cemento-enamel junction; EA, epithelial attachment to the cementum; TS, transeptal fibers; AB, alveolar bone.

the cemento-enamel junction and the bottom of the crevice, *BC*. In both Figs. 240 and 241 the epithelial covering of the papilla is intact and shows a well-developed keratinized layer.

The transeptal fibers, that in Figs. 237 and 238 are located directly below the cemento-enamel junction, are found further apically in Figs. 239, 240, and 241. The interdental alveolar bone is gradually being shortened by resorption.

G. V. Black and others have taught that approximal caries begins in a field immediately rootward from the contact point. This

condition is illustrated in Fig. 242, showing caries of enamel and dentin on the neighboring surfaces of an upper lateral incisor and cuspid in a man, aged twenty-four years. The enamel rootward from the contact point has been destroyed by caries which also has begun to involve the underlying dentin. The most cervical part of the enamel of both teeth between the bottom of the crevice at *BC* and the cemento-enamel junction is intact and healthy, as it is still united with the epithelial attachment. The epithelial attachment has begun to proliferate apically along the surface of the cementum.

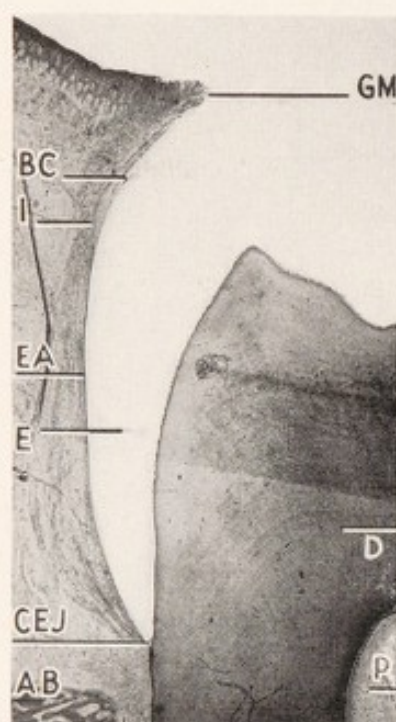


FIG. 243.—Distal surface of the crown of a lower first molar in a child aged eight years. The distal cusps had just erupted. GM, gingival margin; BC, bottom of gingival crevice; I, subepithelial infiltration; EA, epithelial attachment to the enamel; E, enamel; CEJ, cemento-enamel junction; AB, alveolar bone; D, dentin; P, pulp.

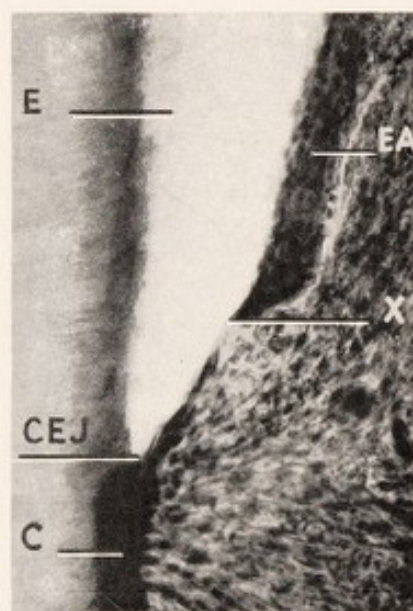


FIG. 244.—High magnification of cemento-enamel junction in Fig. 243. E, enamel; C, cementum; CEJ, cemento-enamel junction; EA, epithelial attachment to the enamel; X, deepest point of the epithelial attachment to the enamel. From X to CEJ, the enamel is in contact with the connective tissue.

ANOMALIES OF THE EPITHELIAL ATTACHMENT AT THE CEMENTO-ENAMEL JUNCTION.

In examining specimens of teeth of young individuals, it can be observed rather frequently that the epithelial attachment on the enamel does not extend all the way to the cemento-enamel junction, but that it ends on the enamel surface crownward from

the cemento-enamel junction. Thus, a small area of the enamel next to the cemento-enamel junction is not covered by epithelium, but is in immediate contact with the surrounding connective tissue. This condition was first observed by Meyer; it is illustrated in Fig. 243, which was taken of a lower first molar of a child, aged eight years. The molar had just erupted; only about one-fourth of the length of the crown was visible in the mouth. The illustration shows the epithelial attachment extending over almost three-fourths of the distal side of the enamel. The bottom of the gingival crevice is located at *BC*; its position is indicated by the thickening of the epithelium, by the presence of the cuticle, and by the location

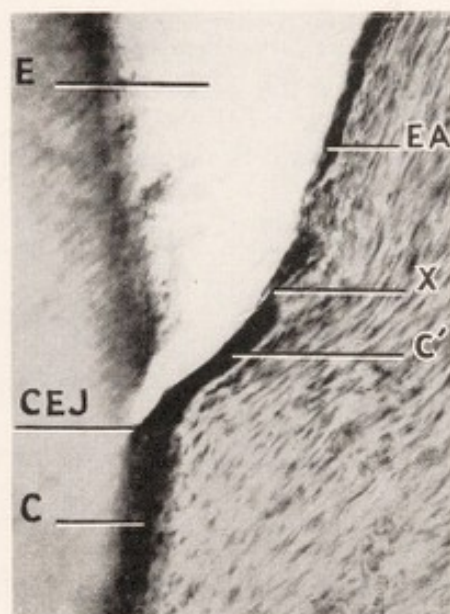


FIG. 245.—Deposition of cementum upon the enamel next to the cemento-enamel junction. E, enamel; C, cementum; CEJ, cemento-enamel junction; EA, epithelial attachment; X, deepest point of the epithelial attachment to the enamel; C', cementum deposited upon the enamel surface (cementum spur).

of the subepithelial infiltration. Toward the cemento-enamel junction, the epithelial attachment ends abruptly on the enamel surface above the deepest point of the enamel (Fig. 244). The significance of the lack of epithelium near the cemento-enamel junction has been discussed recently by Orban. He called attention to the fact that in tooth germs the inner and outer enamel epithelium frequently separate slightly crownward from the cemento-enamel junction. The inner enamel epithelium can be followed to the cemento-enamel junction; the outer enamel epithelium is broken up into the epithelial rests of the periodontal membrane. After the ameloblasts on the cervical portion of the crown have degenerated, the enamel next to the cemento-enamel junction is left without epithelial covering; as

a result, cementum will be deposited occasionally upon the enamel in this region, causing an overlapping of the cementum over the margin of the enamel (Fig. 245). Such extensions of the cementum upon the enamel are called cementum spurs.

Cementum deposits on the enamel surface and spurs of cementum at the cemento-enamel junction are frequently found in impacted teeth (see Fig. 334). In these cases the conditions leading to cementum deposition upon the enamel are the same as in Figs. 244 and 245: loss of epithelial covering of the enamel, contact between enamel and connective tissue, and subsequent cementum deposition upon the enamel.

BIBLIOGRAPHY.

- ADRIEN, W.: Vergleiche histologischer Untersuchungen über das Verhalten des Epithels am Zahnhals, *Deutsch. Mon. f. Zhk.*, 1926, **44**, 22.
- ANDERSON, B. G.: The Fate of the Ameloblastic Cells of the Enamel Organ, *Jour. Dent. Res.*, 1929, **9**, 689.
- BECKS, H.: Normal and Pathologic Pocket Formation, *Jour. Am. Dent. Assn.*, 1929, **16**, 2167.
- Zur Frage der Taschenbildung, *Paradentium*, 1930, **2**, 137.
- Mund und Schmelzepithel in ihrem beiderseitigen Verhalten zur Zahnoberfläche, *Paradentium*, 1931, **3**, 52.
- BLACK, G. V.: *Operative Dentistry*, Chicago, Medico-Dental Publishing Company, 1920.
- CHASE, SAMUEL W.: The Origin, Structure and Duration of Nasmyth's Membrane, *Anat. Rec.*, 1926, **33**, 357.
- CHURCHILL, HERMAN R.: New Ideas on the Gingival Crevice, *Dental Roster*, 1931, **4**, Nos. 17, 18.
- *Human Odontography and Histology*, Philadelphia, Lea & Febiger, 1932.
- EULER, H.: Der "Epithelansatz" in neuerer Beleuchtung, *Vrtljschr. f. Zhk.*, 1923, **39**, 103.
- GOTTLIEB, B.: Der Epithelansatz am Zahne, *Deutsch. Mon. f. Zhk.*, 1921, **39**, 142.
- Schmutzpyorrhöe, Parodontalpyorrhöe und Alveolaratrophie, Wien, Urban & Schwarzenberg, 1925.
- What is a Normal Pocket? *Jour. Am. Dent. Assn.*, 1926, **13**, 1747.
- Tissue Changes in Pyorrhea, *Jour. Am. Dent. Assn.*, 1927, **14**, 2178.
- HÄUPL, K., and LANG, F. J.: *Marginale Parodontitis*, Berlin, Meusser, 1927.
- HIGAKI, RINSO: Beitrag zur Kenntnis des Schmelzoberhäutchens, *Deutsch. Zahnärztl. Wehnschr.*, 1931, **34**, 672, 723.
- KRONFELD, RUDOLF: The Epithelial Attachment and So-called Nasmyth's Membrane, *Jour. Am. Dent. Assn.*, 1930, **17**, 1889.
- KRONFELD, RUDOLF, and ULLIK, R.: Brechen auch bei wilden Tieren die Zähne kontinuierlich durch? *Ztschr. f. Stom.*, 1928, **26**, 84.
- MAXIMOW, ALEXANDER A., and BLOOM, WILLIAM: *A Text-book of Histology*, Philadelphia, W. B. Saunders Company, 1930.
- MEYER, W.: Neue Befunde am Epithelansatz, *Paradentium*, 1929, **1**, 21.
- Neue Befunde am Epithelansatz, *Ztschr. f. Stom.*, 1930, **28**, 775.
- NEUWIRTH, FR.: Die Schmelzmembran und der Epithelansatz am Zahne, *Ztschr. f. Stom.*, 1925, **23**, 318.
- ORBAN, B.: Das normale Paradentium der Hunde, *Ztschr. f. Stom.*, 1924, **22**, 847.

- ORBAN, B.: Dental Histology and Embryology, Chicago, Rogers Printing Company, 1st ed., 1928, Philadelphia, P. Blakiston's Son & Co., 2d ed., 1929.
- Zahnfleischtasche und Epithelansatz, *Ztschr. f. Stom.*, 1931, **29**, 858, 1005, 1360.
- ORBAN, B., and KÖHLER, J.: Die physiologische Zahnfleischtasche, Epithelansatz und Epitheltiefenwucherung, *Ztschr. f. Stom.*, 1924, **22**, 353.
- ORBAN, B., and MUELLER, EMIL: The Gingival Crevice, *Jour. Am. Dent. Assn.*, 1929, **16**, 1206.
- PRINZ, H.: The Etiology of Pyorrhea Alveolaris, *Dental Cosmos*, 1926, **68**, 1.
- Diseases of the Soft Structures of the Teeth and Their Treatment, Lea & Febiger, 1928, pp. 387-390.
- SCHNITER, M.: Beitrag zur Frage des Epithelansatzes, *Deutsch. Mon. f. Zhk.*, 1930, **48**, 705.
- SKILLEN, W. G.: The Morphology of the Gingivæ of the Rat Molar, *Jour. Am. Dent. Assn.*, 1930, **17**, 645.
- Normal Characteristics of the Gingivæ and Their Relation to Pathology, *Jour. Am. Dent. Assn.*, 1930, **17**, 1088.
- Normal Anatomic and Physiologic Gingiva and Its Relation to Pathologic Processes, *Jour. Am. Dent. Assn.*, 1931, **18**, 600.
- A Contribution to the Anatomy and Pathology of the Human Gingiva, *Jour. Dent. Res.*, 1931, **11**, 727.
- SKILLEN, W. G., and MUELLER, EMIL: Epithelium and the Physiologic Pocket, *Jour. Am. Dent. Assn.*, 1927, **14**, 1149.
- Findings in Studies of Tooth Development, *Jour. Am. Dent. Assn.*, 1929, **16**, 98.
- WESKI, O.: Röntgenologisch-anatomische Studien aus dem Gebiete der Kieferpathologie, *Vrtljschr. f. Zhk.*, 1921, **37**, 1; 1922, **38**, 1.
- YUMIKURA, SHIGEIE: Eine neue Färbemethode für gingivale Epithelverhornung und für das sog. sekundäre Schmelzoberhäutchen bzw. Cuticula dentis Gottliebs, *Ztschr. f. Stom.*, 1925, **23**, 868.

CHAPTER XI.

DISEASES OF THE PERIODONTAL TISSUES (GINGIVITIS, PYORRHEA).

NOMENCLATURE AND CLINICAL CONSIDERATIONS.¹

FEW subjects in dental pathology have caused more discussion and more diversity of opinion than the so-called "pyorrhea alveolaris." This existing confusion is perhaps best expressed by the numerous attempts to introduce new names and terms and to create new clinical and histopathological classifications. Pyorrhea alveolaris, parodontitis, periodontoclasia, and parodontosis are some of the more common terms; variations of these terms, such as paradontitis, parodontoclasia, and paradontosis, do not help very much in clarifying the situation. Therefore, there seems to be no reason for adding any new terms or names to the existing variety; rather is it the author's intention to give in this chapter a simple description and illustration of the various forms of periodontal diseases as they are found by the clinician and as they appear under the microscope. Special emphasis will be given to the differentiation between those pathological conditions that are produced by local causes and those conditions that have to be considered local manifestations of systemic or metabolic disturbances. The author is convinced that any nomenclature is useless without a clear understanding of the pathological tissue changes associated with the various clinical forms of pyorrhea. This lack of understanding of the basic principles of periodontal diseases makes it also impossible to discuss intelligently symptoms and therapy since identical terms are applied at random by different authors to etiologically widely different conditions. Once the etiological factors involved in the pathology of the periodontal tissues are clear in the minds of both laboratory investigators and clinicians, it will be very easy to find

¹ During the Eighth International Dental Congress held in Paris, August 2 to 8, 1931, the Committee on Terminology of the International Dental Federation (F.D.I.) recommended a nomenclature for periodontal diseases (see Transactions of the Eighth International Dental Congress, Section IV). The author feels that although this nomenclature is not yet definitely approved and may still be subject to modifications, the terms suggested by the F.D.I. should be given in this book. Therefore, these terms will be put in brackets after the usual terms, with the letters, F.D.I.

simple terms that can serve as a means of correct diagnosis and mutual understanding.

When the author at this time uses two or three clinical terms that were introduced by Gottlieb during the last decade, he does this because these terms have been widely used in Europe since then and have also become rather popular in this country. However, the use of this nomenclature does not indicate that other terms may not be just as acceptable and suitable, provided that they actually indicate a certain well-defined clinical and pathological picture and thus can serve as a means of communication for diagnosis and therapy.

Gottlieb gave a clinical classification of periodontal diseases in which he drew a definite line between the conditions developing as the result of an inflammation of the gingival tissues and those developing as the result of systemic disturbances. In the following table, the author follows, on the whole, Gottlieb's classification with only minor modifications that may help to correlate the clinical picture and the pathological manifestations.

From this table, which is the result of both clinical and laboratory observations, it is evident that a clear distinction must be made between two main forms of periodontal disease: a "local" type beginning with a marginal gingivitis, and a "systemic" type beginning with bone atrophy.

The first form (A in table, page 292) is the result of neglect with resulting inflammation. Gottlieb called this condition "Schmutzpyorrhoe" which term may be translated "pyorrhea due to uncleanness." As a result of uncleanness, irritating debris and calculus accumulate in the gingival crevices. The gingival tissues respond with hyperemia and engorgement, and, finally, breaks (ulcers) develop in the epithelial covering through which a purulent exudate is discharged. Gradually the inflammation progresses in the direction of the margin of the alveolus, and inflammatory bone resorption takes place at the alveolar crest next to the superficial inflammation (marginal atrophy); however, the major portion of the alveolar bone and the periodontal membrane remains intact. Hence there is the clinical observation that the teeth are firm and remain so except in extreme cases of long standing.

The second form has definitely a systemic cause (C II in table, page 293). Its early clinical symptoms are the manifestations of primary bone lesions, resorptions of root surface and bone, with resultant widening of the periodontal membrane and loosening of the affected teeth. Gottlieb called this condition "diffuse atrophy of

CLASSIFICATION OF PERIODONTAL DISEASES (PYORRHEA).

	Clinical manifestations.	Etiology.	Pathology.	Therapy.
A—Gingivitis "Schmutzpyorrhoe" (Gottlieb)—"Pyor- rhea due to unclean- ness" [Gingivitis margin- alis suppurativa, (F. D. I.)]	Hyperemia. Redness, or livid discolora- tion, and swelling of the gin- gival tissue, especially of the interdental papillae. Bleeding of gingiva upon insignificant mechanical in- juries. Formation of ulcerations of the crevice epithelium; occa- sionally discharge of pus. Pockets comparatively shallow. In more advanced cases, re- sorption of the bone at the alveolar crest. Teeth remain firm; loosen- ing only in the last stages. No pain present.	Local irritation of any kind. In most cases, soft and hard deposits (calculus) on the teeth. Neglect of oral hygiene. Gingival caries. Lack of contact point. Poor dental work, such as overhanging fillings, ill-fit- ting crowns, and other dental restorations that irritate the gingival soft tissues.	In early stages, increased amount of subepithelial round cell infiltration in the gingival tissues. In more advanced stages, formation of breaks in the epithelial covering (ulcera- tion) and discharge of pus. Inflammatory infiltration of the deeper gingival tissues. Resorption of the bone of the alveolar crest (marginal atrophy); otherwise perio- dontal membrane is intact and of uniform width.	Removal of etiologic factors. Scaling. Proper use of tooth-brush. Improvement of oral health and hygiene is usually fol- lowed by prompt improve- ment in the inflammation and by healing of the gum tissue.
B—Paradental pyor- rhea (Gottlieb) [Paradentitis pro- funda (suppurativa) simplex, (F. D. I.)]	Individual deep pockets. Irregular distribution of pockets. Sometimes only one paradental pocket is present in the entire mouth. In other cases, many pockets are found. Depth of pockets varies from several millimeters to entire length of root. Discharge of pus from pocket. Moderate amount of calcu- lus usually present in pocket. No pain except in case of pus retention and acute in- fection (lateral abscess).	Accelerated detachment of the epithelial attachment from the root surface with- out corresponding involution and recession of the soft tis- sues. Causes: (a) local causes: Habitual lack of oral hy- giene, especially in the inter- proximal areas. Mechanical deepening of pocket (toothpick, food pack, etc.). Lateral stress in one direc- tion. (b) Systemic causes (see C II, diffuse atrophy of the alveolar bone).	Rapid apical proliferation of the epithelial attachment, and extensive detachment of the periodontal tissues from one side of the root; other- wise the periodontal mem- brane is healthy. Wall of the pocket is formed by inflamed connec- tive tissue with epithelial covering on the outer side and ulcerated surface facing the root. Inflammatory destruction of alveolar bone around the pocket.	Removal of etiologic fac- tors. Scaling. Treatment of pocket: re- moval of outer wall of pocket (gingival flap) by packing or excision of flap.

<p>C—Alveolar atrophy</p> <p>I. Alveolar atrophy as result of excessive occlusal trauma</p>	<p>Resorption of the alveolar bone as a result of excessive occlusal trauma, as in case of a single tooth exposed to excessive lateral stress after loss of adjacent teeth.</p> <p>Loosening.</p> <p>Sometimes formation of pocket on the side toward which tooth is tipped. (Paradental pyorrhea, see B.)</p>	<p>Mutilation of mouth by multiple extractions.</p> <p>Shifting and tipping of remaining teeth with resulting lateral stress.</p> <p>Dental restorations, such as fillings or crowns, that interfere with normal occlusion and cause excessive occlusal stress.</p> <p>Failure of the tissue to repair repeated minor traumatic injuries to the periodontal membrane and to the alveolar bone.</p>	<p>Resorption of alveolar bone on the side towards which the tooth is pressed.</p> <p>Failure to compensate for loss of bone by reparative new-formation.</p> <p>Tooth embedded in fibrous connective tissue with varying degree of (secondary) inflammation.</p>	<p>Unless bone destruction is too far advanced, relief from stress by means of grinding or by an artificial restoration may bring about regeneration of bone.</p>
<p>II. Diffuse atrophy of the alveolar bone (Gottlieb)</p> <p>[Dystrophia diffusa and paradentitis dystrophicans complicata, (F. D. I.)]</p>	<p>Loosening, pathologic wandering of the teeth (elongation, drifting, rotation), and formation of diastemas between the anterior teeth are the earliest symptoms.</p> <p>The radiograph shows irregular and extensive loss of alveolar bone.</p> <p>Pocket formation and supuration appear only in a later stage as secondary symptoms.</p> <p>In pathologic wandering, the pocket appears on the side from which the tooth is drifting.</p> <p>Many of the cases of diffuse atrophy are observed in clean mouths with good oral hygiene; usually there is a remarkable immunity to dental caries.</p>	<p>Systemic or metabolic disturbances.</p> <p>In some cases, etiology is glandular or dietary insufficiency; in others, etiology is unknown.</p> <p>It is advisable to insist on a thorough physical examination and careful analysis in every case to discover, if possible, any systemic factor involved.</p>	<p>Presence of areas of resorption on the root surface.</p> <p>Resorption of corresponding alveolar bone.</p> <p>Widening of periodontal space with transformation of fibrous periodontal membrane into loose vascular connective tissue.</p> <p>These changes may occur anywhere on the root surface.</p> <p>If the process involves areas of the root surface near the alveolar margin, rapid proliferation of the epithelial attachment along the root surface with subsequent detachment and formation of deep pockets takes place; secondary infection and supuration from these pockets usually develop.</p>	<p>(a) Systemic treatment:</p> <p>Improvement of general health.</p> <p>Improvement of dietary conditions.</p> <p>If glandular deficiency is suspected, replacement therapy.</p> <p>(b) Local treatment:</p> <p>Grinding of affected teeth so that they are out of occlusion.</p> <p>Balancing of occlusion.</p> <p>Prophylactic checking of overbite and of lateral stress.</p> <p>Symptomatic treatment of pockets.</p> <p>Mechanical fixation in early and favorable cases.</p>

the alveolar bone" in order to stress the difference between these lesions, which may occur anywhere on the root surface, and marginal atrophy, resulting from marginal inflammation (gingivitis). The resorptive processes on the root surface and alveolar bone and the resulting destruction of the periodontal membrane cause a displacement of the tooth that is clinically known as pathological wandering; this wandering may occur in a vertical direction (extrusion) or in horizontal direction (drifting, diastema formation). In diffuse atrophy, pocket formation and suppuration are distinctly late and secondary symptoms. If a tooth shows definite pathological wandering, a pocket will develop on the side of the root from which the tooth is moving; for instance, in case of labial drifting of an incisor, a pocket is usually found on the lingual side.

The clinical diagnosis differentiating between these two main types of pyorrhea is of greatest practical importance. The primarily local and inflammatory forms are readily influenced and usually completely checked by removing the sources of irritation and maintaining rigid oral hygiene and cleanliness. Diffuse atrophy is a much more serious condition; due to the uncertainty of the etiology, therapeutic procedures are usually restricted to treating the symptoms, checking the occlusion, excising the pockets, and early fixation of the loose teeth.

The clinical course of diffuse atrophy is subject to individual variations. In some cases, loosening and shifting come to a standstill after having persisted for a period of several months; the teeth become firm in their new position and remain firm unless another attack of loosening and wandering takes place. In these cases it must be assumed that a temporary systemic condition (pregnancy, metabolic disturbance, temporary glandular insufficiency) was responsible for the transitional lesion of the periodontal membrane and alveolar bone. In other cases of diffuse atrophy, the destructive process spreads, involving large areas of the tooth surface; one tooth after the other is lost until most of the teeth have been exfoliated. Secondary infection of the pockets and secondary occlusal overstress, induced by the extrusion of the affected teeth from their sockets, hasten the loosening.

In the final stages of pyorrhea, when the tooth has lost the greatest portion of its attachment and is very loose, pyorrhea due to uncleanness and diffuse atrophy usually cannot be distinguished. It is then impossible to determine whether marginal inflammation or primary bone atrophy caused the destruction of the periodontal attachment. However, in such an advanced stage the condition is hopeless, and the differential diagnosis is of no special significance.

Paradental pyorrhea (B in table, page 292) is a connecting link between marginal inflammation and diffuse atrophy, since it may accompany either one of these conditions. Clinically, paradental pyorrhea may be defined as a condition in which discharge of pus from the pockets persists despite removal of all possible local irritations (Gottlieb). In other words, paradental pyorrhea exists if such deep pockets are present that purulent inflammation is perpetuated in their depths despite the usual procedures, scaling, brushing or massaging. Such deep pockets may be the result either of local conditions, habitual lack of cleanliness and chronic inflammation, especially in the interproximal spaces, or of mechanical injuries to the bottom of the gingival crevice, such as intrusion of a toothpick or a bristle of a tooth-brush, or of excessive lateral stress and pounding in one direction with resulting destruction of the periodontal attachment and pocket formation on the side toward which the tooth is pressed.

In other cases, paradental pyorrhea is a late symptom accompanying diffuse atrophy: the loss of bone and periodontal attachment is followed by rapid detachment of the soft tissues alongside the root and subsequent formation of a deep pocket in the affected area.

The division of pyorrhea into a local and systemic form is generally accepted by modern investigators. Gottlieb gave a very definite clinical description of the two forms and contributed an illustration of the normal and pathological tissue changes connected with the pyorrhea problem. Since then practically all investigators have accepted the same division: (1) marginal inflammatory conditions of local origin, and (2) primary bone changes of systemic etiology with suppuration as a late symptom. These two principal forms can easily be recognized in Box's simplex and complex periodontitis and in Becks' marginal paradentitis (primary paradentitis due to deposits) and genuine paradentosis (primary bone disturbance).¹ Häupl and Lang take a special stand in this problem. They, too, differentiate between a superficial form (paradentitis marginalis superficialis) and a deep form (paradentitis marginalis profunda) of periodontal lesion. The clinical description of these two forms exactly follows Gottlieb's definition of marginal pyorrhea due to uncleanness (*Schmutzpyorrhoe*) and diffuse atrophy:

¹ The suffix "-itis" denoting "inflammation," "paradentitis" means "inflammation of the parodontium (periodontium)." The term "-osis" denotes "abnormal or diseased condition." Therefore, "paradentosis" would simply mean "disease of the parodontium." However, by analogy to other medical terms such as nephrosis, "paradentosis" would indicate "non-inflammatory degenerative changes in the parodontium."

paradentitis marginalis superficialis is characterized clinically by marginal gingivitis and firm teeth, paradentitis marginalis profunda by loosening and pathological wandering. Häupl and Lang, however, see in the deep lesions only the continuation and the outgrowth of the superficial inflammation, and do not recognize a systemic component or a primary deep lesion in the sense of Gottlieb's diffuse atrophy.

As to the relative frequency of the two main forms of pyorrhea, all investigators agree that the local form is far more frequent than true diffuse atrophy. The enormous frequency of marginal inflammation was vividly expressed by G. V. Black. He stated that in 500 examinations only 5 per cent of the mouths were entirely free from gingivitis; of the remaining 95 per cent, Black attributed about 45 per cent of the cases to calculus, 37 per cent to imperfect dental operations (bad margins of fillings and inlays, impinging margins of crowns, bridge pontics, and removable appliances), and 18 per cent to malformations and malpositions of teeth. Gottlieb gave a similar figure when he said that as high as 90 per cent of all so-called pyorrhea cases are "Schmutzpyorrhoe" or marginal inflammation due to neglect and lack of cleanliness. Hence Gottlieb insists that a pyorrhea diagnosis should be made only in a clean mouth. The first thing to be done in the mouth of every patient is to establish good oral hygiene by scaling, systematic use of the tooth-brush and careful elimination of all local irritations. By eliminating the inflammatory component it will be possible, in most cases, to bring about normal healthy gingivæ; only the few cases still showing pathological conditions after the superimposed inflammation has been controlled will need special treatment.

A few words must be said in this connection about the bacteriological side of the pyorrhea problem. Despite contrary opinions, pyorrhea is not a specific infection. The various forms of microorganisms and protozoa (amœbæ) that are found in the pus of pyorrhea pockets are in part saprophytes, representatives of the normal bacterial growth of the oral cavity, and in part pathogenic microorganisms that have found an excellent place for development in the depths of the pockets and in the inflamed tissues under calcareous deposits. Perhaps the most important argument against a theory of specific infection is the simple clinical observation that every case of pyorrhea, even the most severe and obstinate, invariably heals within a few days after the extraction of the affected tooth or teeth. If we were really dealing with a deep-seated infection of the soft tissue or bone with specific pathogenic microorganisms, it

would be hard to understand how such an infection could be so promptly and invariably influenced by the extraction of an adjacent tooth. The close etiological relationship between suppuration and the presence of a pocket and the immediate subsiding of any suppuration after the removal of this pocket indicates instead that pocket formation is the primary factor and infection a secondary complication; as soon as the excellent culture medium that is presented by any deep pocket is eliminated, the bacteria disappear promptly.

In the literature on this subject, we find reports concerning the successful treatment of pyorrhea with vaccines and arsphenamine preparations. A critical survey of these reports offers two explanations of the therapeutic success. The first explanation is presented by the suggestion found in all of these papers that calculus should, "of course," be removed during the treatment and that good oral hygiene should be established. Had the author established a clean mouth first and then started the specific therapy, he might have found that the latter had become entirely unnecessary. The second explanation is that no distinction has been made between non-specific gingivitis and specific gingivitis (Vincent's infection).

Vincent's stomatitis (stomatitis ulcerosa, trench mouth) is a specific infection; it is caused by definite and well-known types of microorganisms, namely, fusiform bacilli and spirochetæ, and will respond to any treatment directed against these microorganisms. Acute ulcerations extending beyond the gingiva proper are its clinical symptoms. The gingival tissue shows a marked tendency to necrosis (sloughing), a tendency that is never observed in pyorrhetic conditions. Furthermore, Vincent's infection is decidedly painful; whereas, pyorrhea is never accompanied by pain except in case of the rare lateral abscesses.

ALVEOLAR ATROPHY.

The problem of alveolar atrophy is so complex that it can be understood only by careful clinical and microscopic analysis. Alveolar atrophy may be defined as the result of resorption of the alveolar bone without or with insufficient subsequent new formation of bone. The causes for the resorptive process leading to bone atrophy may be manifold; for the sake of differential diagnosis they will be classified as follows:

1. Physiological atrophy of the alveolar margin as part of the general involution with advancing age.

2. Alveolar atrophy due to inflammatory processes in the gingival soft tissues.

3. Alveolar atrophy due to excessive occlusal stress.

4. Alveolar atrophy as the expression of a systemic disturbance (diffuse atrophy).

1. **Physiological Atrophy of the Alveolar Margin as Part of the General Involution With Advancing Age** (*Atrophia Senilis*, F.D.I.).—In order to clarify the significance of the physiological alveolar atrophy that takes place throughout life, it is necessary to recall briefly the general regressive changes to which every normal healthy tooth is subject. These changes have already been described in part in Chapters II, VIII and X.

In the tooth proper the following tissue changes take place with advancing age:

(a) Enamel: mechanical abrasion by use and wear.

(b) Dentin: continual deposition of dentin and gradual sclerosis of the dentinal tubules; abrasion and formation of secondary dentin wherever the dentin surface has been exposed.

(c) Pulp: decrease in the size of the pulp chamber; regressive changes in the pulp tissue, manifested by fibrosis and calcifications of the pulp and atrophy of the cellular and vascular elements.

(d) Cementum: continual deposition of new layers of cementum upon the old ones, resulting in a thickening of the cementum, especially at the apices of all teeth and in the bifurcations of multi-rooted teeth.

In the investing tissues the following involutionary changes occur during life:

(e) Epithelial attachment and gingival crevice: the deepest point of the epithelial attachment constantly shifts rootward. At the same time, the epithelial attachment becomes detached at the bottom of the crevice, and is slowly but steadily displaced apically. As a result, with advancing age, the bottom of the crevice passes the cemento-enamel junction, and then the root surface at the neck of the tooth is exposed.

(f) Topographic relationship between tooth and socket: all teeth move occlusally throughout life. In youth, during the eruptive period, this movement is comparatively fast. Later in life it is much slower, but it can be demonstrated beyond doubt by clinical and histological observations. In addition to this vertical movement, a very slow mesial wandering of all teeth can be observed. This form of movement finds its histological expression in the distribution of lamellated bone and bundle bone on the inner wall of

the alveolus (Stein and Weinmann). Both occlusal and mesial displacements of the teeth occur at the rate at which tooth structure is lost at the occlusal surface and at the contact points by occlusal and interproximal wear respectively.

(g) Alveolar bone: accompanying the downward growth of the epithelial attachment and the extrusion of the tooth from its socket, the alveolar margin is gradually resorbed. This bone atrophy finally leads to what is known clinically as senile alveolar atrophy.

Together with all the other regressive changes, the form of alveolar atrophy described under (g) is part of the normal life cycle to which every tooth is subject between the time of its eruption and the natural death of the individual. The degree and rate of development of this marginal atrophy is, like all other regressive changes, subject to wide individual variations; the slower it takes place, the better it is for the maintenance of undisturbed function. Physiological marginal alveolar atrophy is recognized clinically by the gradual and uniform shortening and rounding of the tips of the interdental bone septa which can be noticed by comparing radiographs of young and old persons with firm teeth and healthy gingival tissues.

Under pathological conditions, alveolar atrophy may occur rather rapidly, thus producing in relatively young individuals a clinical picture that is normally observed only in much more advanced age. The etiology of this premature atrophy of the alveolar process is unknown; the term "*atrophia præcox*" has been proposed for this condition (F.D.I.).

2. Alveolar Atrophy Due to Inflammatory Processes in the Gingival Soft Tissues.—All gingivitis that involves more than just the superficial gingival tissues is bound to cause resorption of the bone at the alveolar crest. The mechanism of bone resorption in this case is the same as in every inflammatory process occurring in the immediate neighborhood of bone: the inflammation causes hyperemia, increased circulation, and increased tension in the soft tissues, resulting in osteoclastic activity and resorption of the adjacent bone. Never is the bone directly involved with a true osteitis or osteomyelitis, and the rather common reference to "necrotic bone" in pyorrhea is distinctly erroneous. Necrosis of the alveolar bone under inflamed gingivæ or under pyorrhea pockets has never been demonstrated. The bone is resorbed by osteoclasts, but it is not exposed and does not become necrotic as in caries or osteomyelitis of the jaws.

The main characteristic of inflammatory bone resorption is that

it is primarily confined to the bone of the alveolar crest; the deeper bone and the periodontal membrane in the deeper portions of the root are intact. Clinically this is indicated by the firmness of the affected teeth. Only if the superficial inflammation is of long standing will the marginal bone destruction reduce the height of the socket to such a degree that the tooth becomes loose. However, it usually takes decades of bad neglect and resulting extensive marginal inflammation to loosen a tooth in that way.

The prognosis of marginal atrophy is decidedly favorable in its early stages. Removal of the inflammation stops the process of bone resorption, and although the periodontal attachment does not regenerate in the areas in which the bone has been destroyed, the remaining portion of the periodontal membrane is strong enough to insure good function.

3. Alveolar Atrophy Due to Excessive Occlusal Stress.—Excessive occlusal stress, particularly in a lateral direction, may cause resorption of the alveolar bone on the side of pressure. If the force exerted upon the tooth is not too great and of too long duration, the loss of bone can be compensated for by new deposition of bone and cementum, and the attachment of the tooth on the side of pressure will be regenerated. If, however, the force is great, especially if there is a marked horizontal (lateral) component involved, no such repair will take place. More and more bone is resorbed until the periodontal space becomes very wide; the fibrous periodontal membrane is replaced by loose connective tissue, and the tooth is markedly loosened in its socket (see Fig. 266).

The prognosis in case of alveolar atrophy resulting from excessive occlusal trauma depends largely upon the extent of bone destruction and the reparative power of the organism. In early cases, in which the loosening of the tooth is not yet excessive, complete relief from occlusal trauma will frequently be sufficient to bring about bone regeneration and tightening of the tooth. In advanced cases of bone atrophy, the tooth is usually lost.

4. Alveolar Atrophy as the Expression of a Systemic Disturbance.—Extensive alveolar atrophy occasionally occurs without any local factors that might explain the condition. These cases have to be explained as the oral manifestation of some systemic disturbance. Radiographs of the teeth of a typical case of this type are reproduced in Fig. 246. The patient, a woman, aged twenty-eight years, had always been in good health. Her teeth were in excellent condition: no tooth was missing, all teeth were well formed, in normal occlusion, and entirely free from caries and deposits.

When the radiographs were taken the patient was in the eighth month of pregnancy; she complained that since she had become pregnant her teeth became loose. The clinical examination revealed that all teeth were markedly loose and that diastemas had developed between some of the anterior teeth. On some teeth there were pockets of considerable depth; however, no suppuration



FIG. 246.—Radiographs of a case of diffuse atrophy of the alveolar bone. The extensive bone destruction and loosening of the teeth developed during pregnancy in a woman aged twenty-eight years. No calcareous deposits are present on the teeth, and there is marked immunity to dental caries. Notice the presence of diastemas, especially between the upper left anterior teeth.

was present, and had it not been for the general loosening and the radiographic evidence of bone resorption, clinically the teeth would have looked normal. In this case there can be no doubt that the loss of alveolar bone (diffuse atrophy) had some etiological connection with pregnancy; the exact manner in which one condition influences the other is not known.

In other cases of primary bone atrophy only one or several teeth in the whole mouth are affected. Sometimes one of the anterior teeth, especially the upper ones, shows loosening, elongation, and drifting (pathological wandering); the change in the position is often first noticed clinically by the formation of a diastema of increasing width between the affected tooth and its neighbors. If a posterior tooth wanders pathologically, it almost invariably assumes a position in which it is exposed to excessive occlusal stress, which will, in turn, hasten its loosening and final loss.

The exact etiology of diffuse atrophy is not yet known. Metabolic disturbances, faulty nutrition, endocrine dysfunction, pregnancy, and diabetes are some of the conditions frequently associated with diffuse atrophy of the alveolar bone. As far as diet in relation to diffuse atrophy is concerned, the author wishes to warn most emphatically against unsubstantiated statements and conclusions on this subject. Lesions in the teeth and alveolar bone of animals produced experimentally by dietary measures (Jones and Simonton, Becks and Weber, and others) resemble only very slightly the typical diffuse atrophy in man. The nutrition of these animals (dogs) is utterly unlike any human diet; besides, it must be emphasized that in these animals the pathological bone changes were not confined to the jaw bone but were found all over the skeleton. No counterpart for this latter observation has as yet been demonstrated in man, that is, in no case of diffuse atrophy of the alveolar bone have identical or even similar changes been found elsewhere in the body. Therefore, the author thinks that it may be better for the time being to confess our ignorance of the etiology of diffuse atrophy than to jump at hasty conclusions and then later have to retract. The dentist is certainly not justified in concluding that because a patient with diffuse atrophy has not, in his own or in the dentist's opinion, had enough yeast, orange juice, or cod-liver oil, the oral lesion must necessarily have a dietary etiology and can be influenced by dietary measures.

Very remarkable is the relative frequency with which diffuse atrophy is observed in the mouths of individuals with large, well-built teeth, very little calculus, and pronounced immunity to dental caries (see Fig. 246).

Because of the uncertainty concerning the true etiology of diffuse atrophy, the therapy is rather indefinite and variable. If the case history reveals a distinct dietary or metabolic deficiency, such deficiency must be checked. The resulting systemic improvement is usually beneficial to the dental lesions. Local treatment consists

of balancing the occlusion and grinding elongated teeth in order to eliminate additional damage through secondary occlusal trauma. In many instances the teeth will show marked improvement under such treatment. Other cases, however, show a progressive tendency that leads to the loss of the affected tooth or teeth.

HISTOPATHOLOGY OF GINGIVAL INFLAMMATION DUE TO LOCAL IRRITATION. (Gingivitis Marginalis Suppurativa, F.D.I.)

1. **Clinical Considerations Concerning Calculus.**—Dental calculus, the main source of gingival irritation, is the result of the calcification of organic matter deposited upon the clinical crowns of teeth. This deposit consists of the following components: desquamated epithelial cells of the oral mucosa, food débris, mucin of the saliva, and various microorganisms of the oral cavity. It has been proved beyond doubt that the primary step in the formation of calculus is the deposition of a delicate, soft film of these substances, that at first can easily be wiped off in mastication or with a tooth-brush, and that the secondary step is the calcification and hardening of the film by the deposition of calcium salts from the salivary fluids. If the forming organic film is carefully removed with a tooth-brush every day, the formation of calculus can be greatly decreased or inhibited.

The chemical analysis of calculus shows considerable variability in the composition. The following figures represent an average:

Calcium phosphate	70 per cent
Calcium carbonate, calcium fluoride and magnesium phosphate	10 per cent
Water and organic matter	20 per cent

Clinically very hard calculus contains a higher percentage of inorganic salts; clinically soft and chalky calculus contains a higher percentage of organic matter and water.

The tendency of calculus to form varies greatly with different individuals. Some mouths are practically free from it; in others calculus forms very rapidly despite frequent removal and good oral care. The reasons for this individual difference are not fully understood.

Calculus is most commonly found on the lingual surfaces of the lower anterior teeth opposite the openings of the submaxillary and sublingual glands and on the buccal surfaces of the upper posterior teeth opposite the opening of the parotid duct. But calculus may be found also on any tooth where there is a possibility of undis-

turbed accumulation of organic matter and subsequent incrustation with calcium salts.

For the sake of clinical description, calculus may be divided into supragingival or salivary calculus and subgingival or serumal calculus. There is no difference in the etiology of these two types of calculus: both develop by an incrustation of soft, organic deposits with calcium salts of the saliva. There is, however, a distinct difference in localization. Salivary or supragingival calculus is most commonly found near the openings of the salivary glands of the oral cavity, namely, on the lingual surface of the lower anterior teeth and on the buccal surface of the upper molars. Its color is usually light yellow unless smoking has caused it to turn brown. Salivary calculus is attached to the tooth surface crownward from the gingival margin (see Fig. 253) and, therefore, is easily accessible to clinical oral inspection. Serumal or subgingival calculus, on the other hand, may be found anywhere in the mouth; its color is dark brown or greenish, its consistency is hard and brittle. This form of calculus is deposited on that part of the tooth surface that is located inside of the gingival crevice between the free margin of the gingiva and the bottom of the crevice; it is, therefore, partly or entirely hidden to direct inspection and can be investigated only by entering the crevice with an explorer.

To avoid misunderstanding, it should be emphasized that calculus can be deposited only on the surface of the clinical crown, namely, on that portion of the tooth lying crownward from the bottom of the gingival crevice and accessible to oral fluids. The bottom of the gingival crevice is the distinct line of demarcation that indicates how far rootward calculus may extend; further apically than this line no deposits can be formed (see Figs. 238, 254).

This difference in the location of salivary and serumal calculus is probably the cause for the difference in the physical qualities of the two forms of calculus: salivary calculus, being deposited in immediate proximity to the sources of the flow of saliva, forms rather rapidly. Due to its superficial location, it is constantly bathed and rinsed in saliva; thus, all pigmentations that may, for instance, result from minute gingival hemorrhages are readily washed out and the calculus retains its typical light color. Serumal calculus, on the other hand, forms much more slowly; under the protecting cover of the gingival crevice, it becomes denser and harder than salivary calculus. Small hemorrhages from the ulcerated soft tissues contribute blood pigment, causing a permanent dark brown or grayish color.

For more detailed information concerning calculus, we refer to the text-books of Black, Bunting, Marshall and Prinz; in this book it is intended to describe and illustrate the actual tissue changes in the presence of calculus as they are found in the microscopic examination of human jaw tissues.

2. Marginal Gingivitis Due to Local Irritation (Lack of Oral Hygiene).

—In the microscopic study of specimens of human gingivæ and teeth, it is difficult to draw a line between what may still be called normal and what may be called early pathological manifestations. As has been pointed out in the discussion of epithelial attachment and gingival crevices, microscopic research along this line is considerably handicapped by the restrictions in obtaining autopsy material. Without exception our specimens come from a class of people who practices very little, if any, oral care and cleanliness. In addition, it must be remembered that many of the individuals from whom the jaws were obtained during autopsy died after long and severe illnesses in some charitable public hospital. Consequently, more soft and hard deposits are present in most of our specimens than in the mouths of patients in a good private practice.

Although as a rule the mouths of children are comparatively clean and free from deposits, still the early stages of marginal gingivitis and calculus formation can frequently be observed in early ages. Since in young teeth the bottom of the gingival crevice is located on the enamel surface, the deposition of calculus occurs on the surface of the enamel, and the resulting gingivitis is found at and behind the epithelial attachment to the enamel.

The first two specimens to be illustrated here represent the labial side of a lower cuspid and the lingual side of a lower central incisor of a boy, aged fourteen years. The mouth was in good condition; no teeth were missing, and only small occlusal carious lesions were present in the first molars. The clinical diagnosis as well as the microscopic appearance of the specimen, which showed a considerable amount of soft deposits on some of the teeth, suggested that during the last few weeks of life oral health had been badly neglected. The condition of the gingival tissues varied according to the degree to which the various tooth surfaces were self-cleansing. Hence the difference between the microscopic appearance of Figs. 247 and 248. On the labial surface of the lower cuspid, an area that is habitually rather clean, the hornification of the gingival epithelium extends to the bottom of the extremely shallow crevice. Only a very small amount of subepithelial round-cell infiltration

is present. The epithelium is still attached to the gingival one-fourth of the enamel. As a whole, the condition illustrated in Fig. 247 can be considered normal and typical for that particular age and stage of eruption (see also Fig. 223).

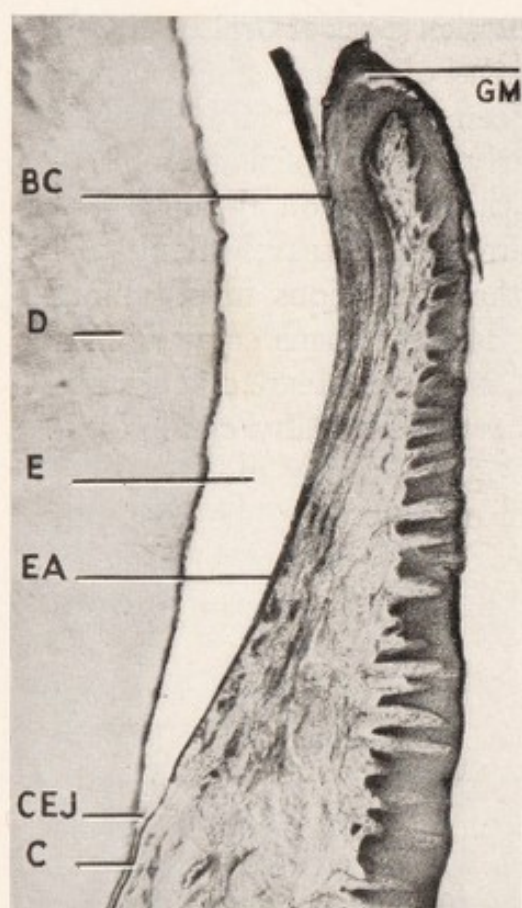


FIG. 247.—Normal gingival crevice on the labial surface of a lower cuspid of a boy, aged fourteen years. The depth of the crevice is practically zero. The hornification of the gingival epithelium extends to the bottom of the crevice. Very small amount of subepithelial infiltration. D, dentin; E, enamel; BC, bottom of gingival crevice; EA, epithelial attachment to the enamel; CEJ, cemento-enamel junction; C, cementum; GM, hornification of the epithelium at the gingival margin.

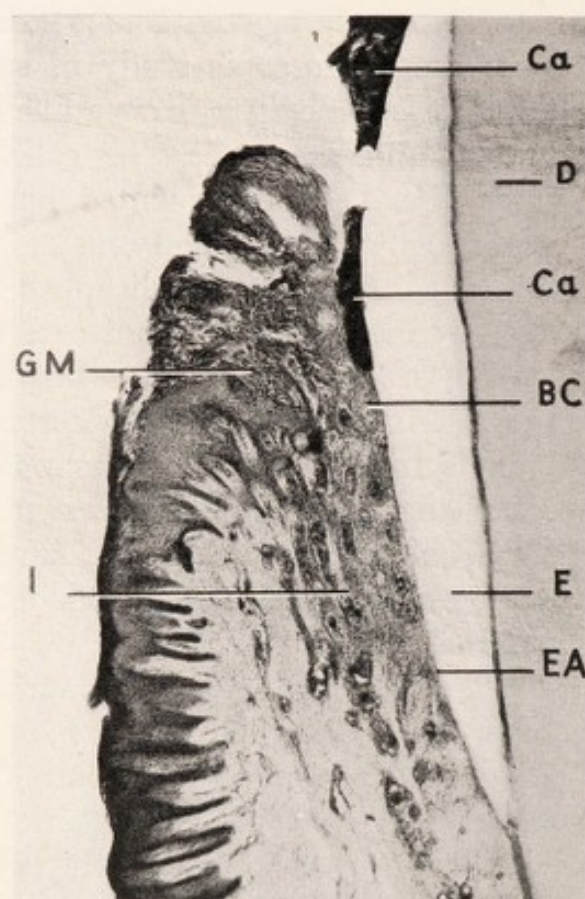


FIG. 248.—Early stage of gingivitis due to lack of oral hygiene. Lingual surface of lower central incisor of the same individual from which Fig. 247 was taken. D, dentin; E, enamel; BC, bottom of gingival crevice; EA, epithelial attachment to the enamel; Ca, calculus on the enamel surface; GM, ulcerated gingival margin covered by masses of desquamated epithelial cells and by soft deposits; I, subepithelial round-cell infiltration and capillary hyperemia.

The presence of a small number of round cells in the subepithelial tissues might be considered an indication of gingivitis; but, in doing so, one would have to consider practically every human gingival crevice as pathological. This, however, would have no practical value and would lead to considerable confusion. It must be kept in mind that the oral mucosa shows normally in many places an

accumulation of lymphatic tissue in the submucosa; therefore, von Ebner considered the subepithelial infiltration of the gingival tissues as part of the lymphatic system and described it as physiological. The author will follow the practice of calling gingivæ and gingival crevices with intact, well-hornified coverings of epithelium, such as the one illustrated in Fig. 247, "normal gingival crevices," despite the presence of a small amount of subepithelial infiltration; on the other hand, the terms "gingivitis" and "pathological crevices" will be used wherever the specimens show distinct breaks and defects in the epithelial covering and marked inflammatory reactions in the subepithelial connective tissue.

It should be stated again that the location of the crevice on the tooth surface has no significance whatsoever in deciding whether the crevice is normal or pathological. The bottom of a pocket may be located on the surface of the enamel and yet the pocket may be distinctly pathological if the crevice epithelium is ulcerated and the subepithelial tissue is badly inflamed; on the other hand, in an older person, the bottom of a pocket may be found on the cementum surface, but the intact epithelial covering, the lack of inflammatory symptoms, and the clinically healthy appearance justify calling such a pocket normal (see Fig. 234).

The lingual surface of the lower central incisor of the same individual, aged fourteen years, shows marked symptoms of early gingivitis (Fig. 248). The crest of the gingiva is covered by a mass of desquamated epithelial cells. The outline of the mass still resembles the outline of the gingival margin. The crevice epithelium and subepithelial tissue present marked inflammatory changes. Dense round-cell infiltration extends to the cemento-enamel junction. The capillaries are enlarged and hyperemic. At the bottom of the crevice the epithelium has been lost, and subepithelial inflammatory cells are being discharged into the crevice. The alveolar bone is not affected; apparently the inflammatory changes were still confined to the gingival margin and had not yet involved the deeper tissues.

This condition can be readily understood if we consider that keratinized dead cells are constantly being desquamated from the squamous epithelial covering of the gingiva. In a clean, well-kept mouth this cell débris is removed daily, by means of the tooth-brush, in all those areas where it is not worn off during mastication. If not removed, the dead cell masses accumulate; they become calcified, and then form calculus.

In specimens of adult persons the bottom of the gingival crevice

in the presence of calculus and inflammation is, as a rule, found on the surface of the root. It is, in these cases, difficult to evaluate the causative connection between inflammation and advanced detachment of the epithelial attachment from the tooth surface. With advancing age, the epithelium invariably proliferates along the root surface (see page 271); it is also known that marginal inflammation destroys the attachment of the periodontal tissues to the cementum and acts as an incentive for proliferation of the epithelium. In analyzing a specimen of an adult tooth with fairly advanced detachment of the gingiva and marginal inflammation, it is hard to decide whether the inflammation was superimposed upon the recession, or the inflammation caused the recession, or both inflammation and recession existed independently of each other. This difficulty has recently been clearly pointed out by Skillen who, after a comparative study of the depths of the pocket, inflammation, and epithelial proliferation, came to the conclusion that "It is impossible, or at least difficult, to correlate the conditions in and about the gingiva with the epithelial proliferation, although these conditions seem to play some rôle in the so-called recession of the gingiva."¹

Figs. 249 and 250 show the lingual side of an upper cuspid in an adult. The bottom of the gingival crevice is located slightly rootward from the cemento-enamel junction. A piece of calculus lies in the pocket, extending into the pocket epithelium and causing a break in the epithelial continuity between gingiva and epithelial attachment. The subepithelial connective tissue shows dense, inflammatory round-cell infiltration which extends along the root surface apically until close to the alveolar crest. The latter presents evidence of resorption, no doubt as a result of the approaching gingival inflammation. It can be noticed that in this specimen the pocket is very shallow, its depth being practically zero.

As will be seen in subsequent illustrations, the relationship between marginal inflammation, calculus, and depth of pocket is subject to wide individual variations. It has been observed in chronic inflammation of the mucosa and chronic gingivitis that some individuals show a marked tendency toward the development of a productive or proliferative inflammation with new tissue formation, while others are more likely to develop an ulcerative form of inflammation with tissue destruction. Thus, it can be understood why, in some cases of extensive chronic gingivitis, the pockets are quite deep, while in others they remain extremely shallow. In

¹ W. G. Skillen, *Jour. Dent. Res.*, 1931, **11**, 727.

the first case, the free gingivæ have become hypertrophic. They are swollen and enlarged. As a result the bottom of the crevice moves apically, and the distance between the bottom of the crevice and the gingival margin becomes larger, causing a deepening of the pocket. In the second case, inflammation and ulceration actually have destroyed the gingival margin, and although the detachment

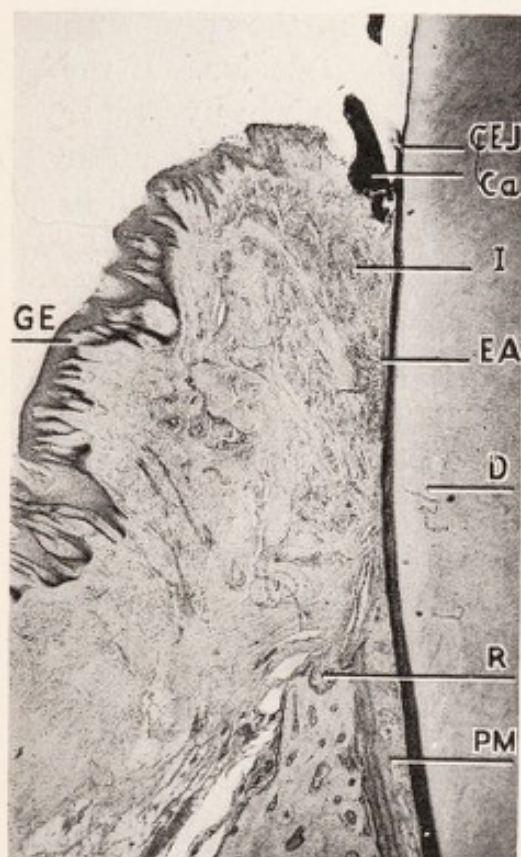


FIG. 249.

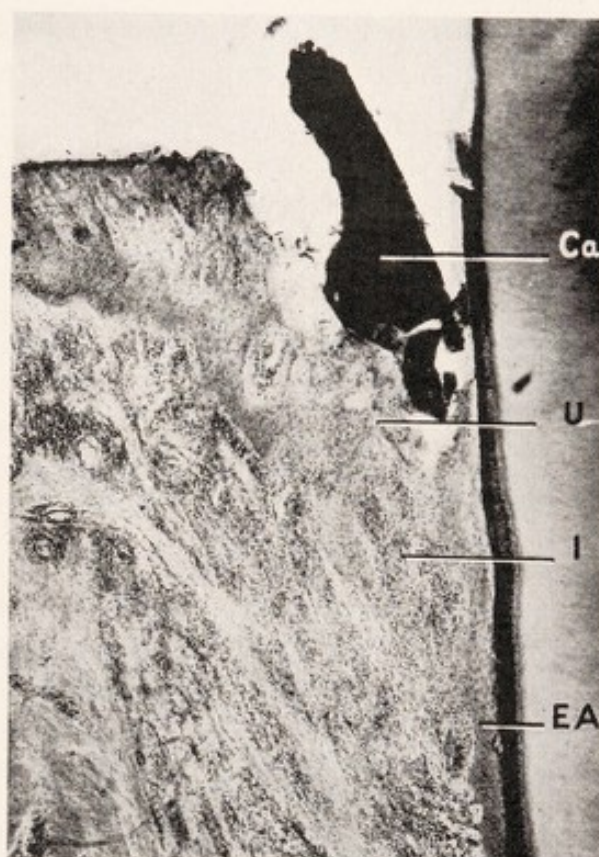


FIG. 250.

FIGS. 249 and 250.—The effect of calculus upon the gingival tissues. Ulceration of the gingiva. Lingual surface of an upper cuspid in an adult.

FIG. 249.—D, Dentin; CEJ, cemento-enamel junction; Ca, calculus at the bottom of the crevice; I, dense inflammatory infiltration of the tissues beneath the calculus; GE, gingival epithelium; EA, epithelial attachment to the cementum; R, resorption of the alveolar crest; PM, intact periodontal membrane.

FIG. 250.—Higher magnification of Fig. 249. Ca, calculus; U, ulceration below the calcific deposit; I, inflammatory exudate cells; EA, epithelial attachment to the cementum.

of the tissues from the tooth surface progresses apically, the pocket does not deepen.

The prognosis in either form of marginal gingivitis is favorable; they both respond readily to removal of the irritation and proper care. As a rule, healing of the ulcerative form is more rapid when the pockets are shallow than when the pockets are deep and present a greater possibility for retention and recurrent inflammation.

The healing of the gingival tissues can readily be understood from a higher magnification of Fig. 249. This figure shows a fragment of calculus in the gingival tissues at the bottom of the crevice. To the left of the calculus, the oral mucosa is intact; to the right, the epithelial attachment covers the cementum surface. Directly beneath the piece of calculus an ulcer has developed; the subepithelial tissue shows extensive inflammatory infiltration with round cells, some of which pass through breaks in the epithelium and form a purulent discharge from beneath the calculus. If the calculus were removed and the gingivæ were kept clean by proper brushing and care, the subepithelial inflammation would disappear within a few days; at the same time, epithelium would begin to proliferate from the mucosa of the gingiva as well as from the epithelial attachment; the newly formed epithelial cells will grow toward each other from these two places, until the entire surface of the former ulcer is covered by a new intact epithelial layer.

It appears appropriate here to give a brief description of the normal histology of the human gingiva. The oral mucosa is usually described as being not hornified, except for a part of the tongue surface. Thus, the oral mucosa differs from the epidermis: while the latter is normally covered by a well-developed keratinized layer, keratinization of the oral mucosa is considered pathological. Orban, who recently studied the hornification of the gingival tissue, found that the above statement is not altogether correct. He showed that, while it is true that the mucosa of the cheek or of the vestibulum does not hornify, the "gingiva propria," that part of the mucous membrane surrounding the teeth and forming the gums and the interdental papillæ, has under normal circumstances a keratinized surface. Hornification of the gingival mucosa is an essential factor in the maintenance of healthy normal gingivæ; in its absence the gingivæ look abnormal, red, and swollen, and bleed easily at the slightest mechanical injury.

This condition was plainly demonstrated by Orban in a comparison of the clinical status of the gingivæ with the microscopic examination of small pieces of gingival tissue removed by biopsy. Fig. 251 illustrates part of the surface of the interdental papilla of an individual, aged twenty-four years. Six months previous to the removal of the specimen, this patient had livid, swollen gums that bled at the pockets; much calculus was present. The teeth were cleaned, and the patient was instructed in the proper use of the tooth-brush. When the piece of gingival tissue was removed, the gingiva looked normal and healthy. It was pale pink and sur-

H

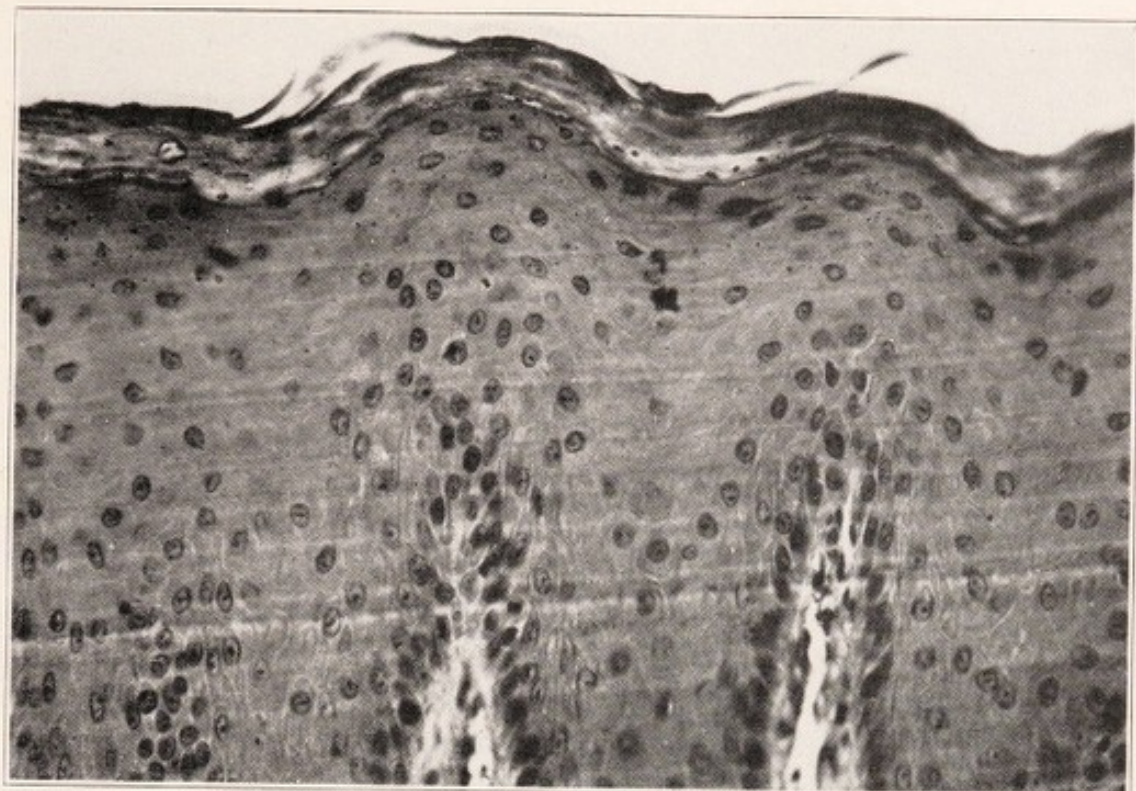


FIG. 251.—Epithelium covering an interdental papilla of an individual, aged twenty-four years. Clinically healthy, pale pink gingiva of firm texture. Good oral hygiene. The wavy surface of the mucosa is covered by a heavy horny layer, H. (Orban, Jour. Am. Dent. Assn.)

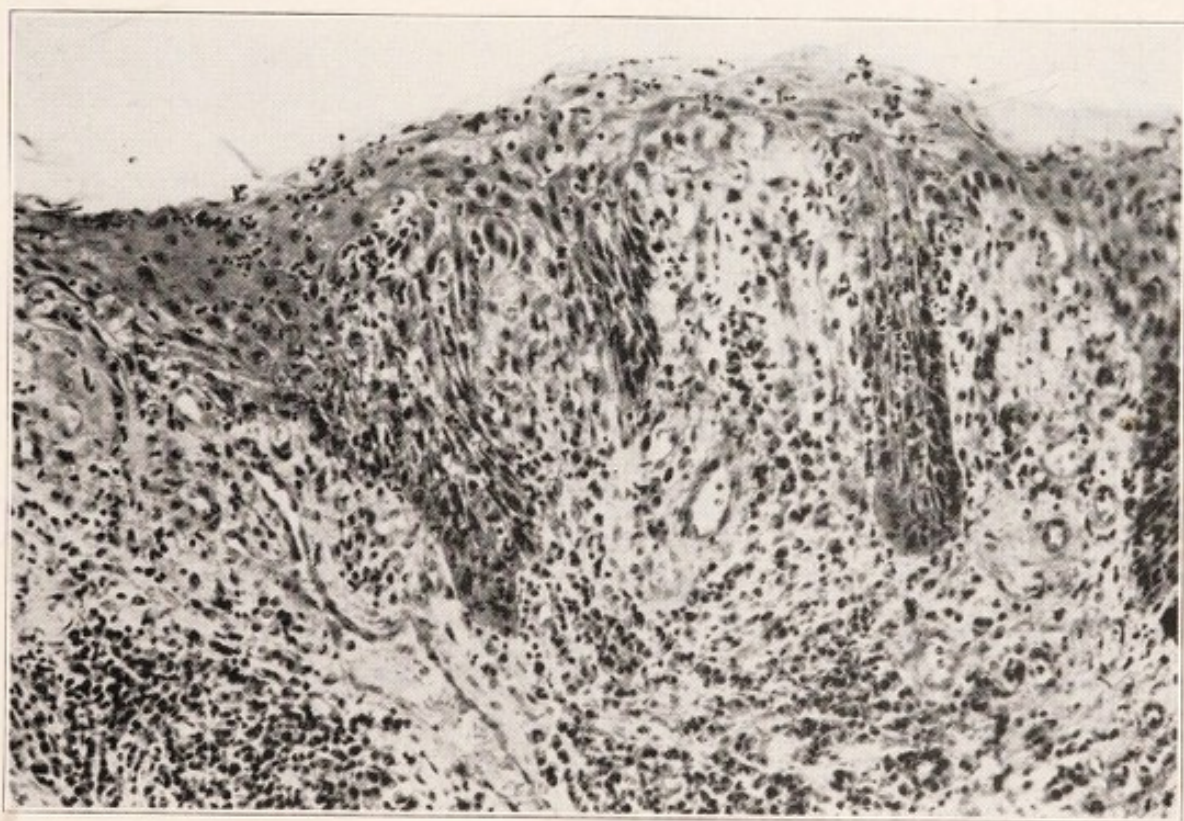


FIG. 252.—Surface of an interdental papilla of an individual, aged fifty-six years. Clinically inflamed, reddish blue, swollen and bleeding gingiva. Large masses of inflammatory cells in the subepithelial connective tissue, and leukocytes migrating through the epithelium to the surface. Absence of a horny layer. (Orban, Jour. Am. Dent. Assn.)

rounded the teeth firmly. Histologically the mucosa shows a normal arrangement of the squamous epithelial cells with no evidence of inflammation. The wavy surface is covered by a heavy horny layer. The superficial layers of horn have been cast off or shed.

An entirely different microscopic picture was found when clinically unhealthy looking, inflamed gingival tissue was examined microscopically. A typical specimen of this kind is reproduced in Fig. 252, taken from the gingival tissue of an individual, aged fifty-six years, with swollen, reddish blue, and easily bleeding gingivæ. The specimen shows degenerative changes in the epithelium, complete absence of a hornified layer, and extensive subepithelial inflammation, indicated by the presence of a large number of leukocytes, polyblasts, and plasma cells. The epithelium has been extensively invaded by leukocytes, which migrate through the epithelial covering and are discharged into the oral cavity.

These two examples may illustrate the difference between normal and abnormal gingival tissues. It should be the aim of the dental profession to produce as many healthy, well-hornified gingivæ as possible of the type shown in Fig. 251. This can be done simply by combining proper dental care with scrupulous cleanliness and rigid oral hygiene. Thus, gingival tissue as seen in Fig. 252 may be transformed into the type illustrated in Fig. 251, a fact that has been demonstrated by Orban through repeated histological examination of the same patient. *Vice versa*, well-hornified, firm, healthy gingivæ may turn into inflamed, swollen, and tender gingivæ if they are neglected and deposits are allowed to accumulate.

As stated before, a large amount of calculus may accumulate and the gingival tissue may be destroyed rather extensively without the formation of a pocket. In such a case the calculus sits upon a flat, raw, ulcerated gingival surface; pus is discharged from the capillary space between the base of the deposit and the underlying gingival tissue. Fig. 253 illustrates a condition of this type. The specimen under consideration shows the buccal side of an upper second molar without antagonists; the entire buccal surface of the crown had been covered with a flat, chalky, yellow deposit of salivary calculus, the thickness of which increased toward the gingiva. In bucco-lingual section, the calculus appears as a wedge-shaped black mass; the entire surface of the underlying tissue has been changed into a flat ulcer. The subepithelial connective tissue is densely infiltrated and inflamed, and the buccal plate of the alveolar bone ends in an uneven resorbed surface. Here, just as in Figs. 249 and 250, there is an epithelial attachment on the surface of the cementum.

This epithelium, as well as the epithelium at the gingival margin and the islands of epithelial tissue within the ulcer, is the source from which, after removal of the calculus, a new epithelial covering develops and within a few days covers the gingival tissue, thus reestablishing normal conditions.

Up to this point all specimens that have been shown concerned shallow crevices and what clinically might be termed salivary



FIG. 253.—Deposits of salivary calculus on the buccal surface of an upper molar. The calculus lies flat upon the ulcerated surface of the gingiva. Ca, calculus; U, ulcer, I, inflammatory infiltration; GE, gingival epithelium; AB, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

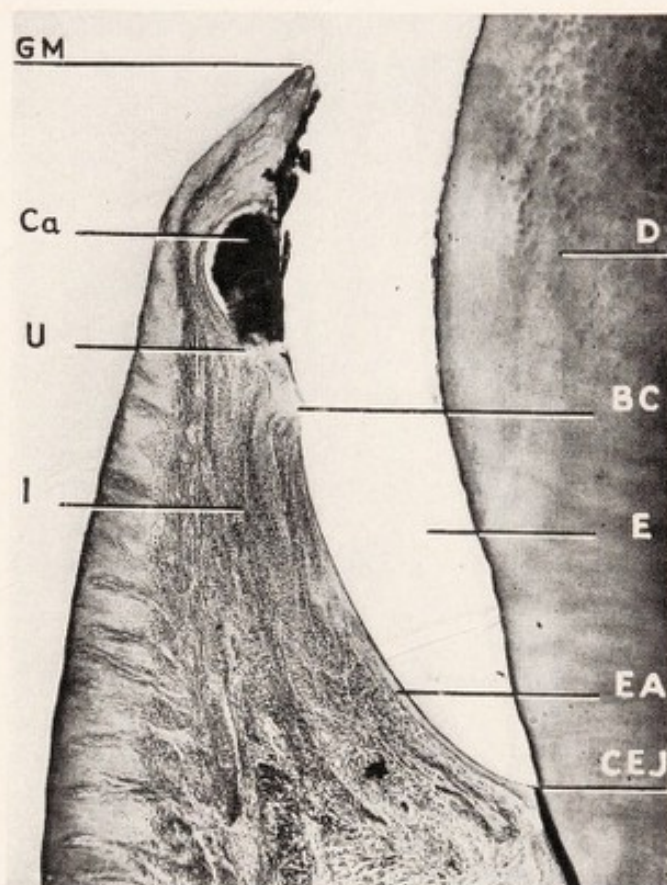


FIG. 254.—Subgingival calculus on the surface of the enamel. Lingual surface of a lower deciduous molar. D, dentin; E, enamel; BC, bottom of gingival crevice; Ca, calculus; U, ulceration of the wall of the crevice overlying the deposit; GM, gingival margin; I, subepithelial infiltration; EA, epithelial attachment to the enamel; CEJ, cemento-enamel junction.

calculus. The following group of specimens deals with the histopathology of the gingival tissues in the presence of serumal calculus in pockets of considerable depth. In the study of such specimens it is impossible to decide definitely the sequence of changes that led to the pathological condition present at the time of death. It is possible that the deepening of the pocket was the primary factor which was followed by deposition of serumal calculus within

the pocket; or it may be that calculus was deposited first in a very shallow pocket, causing inflammatory hypertrophy of the adjacent gingival tissue and deepening of the pocket. Most likely these processes occur simultaneously and lead to the formation of subgingival calculus that is either partly or completely located beneath the free gingivæ.

If subgingival calculus develops in childhood, it is found on the surface of the enamel. Fig. 254 shows such a case taken from the lingual side of a second deciduous molar of a child, aged eight

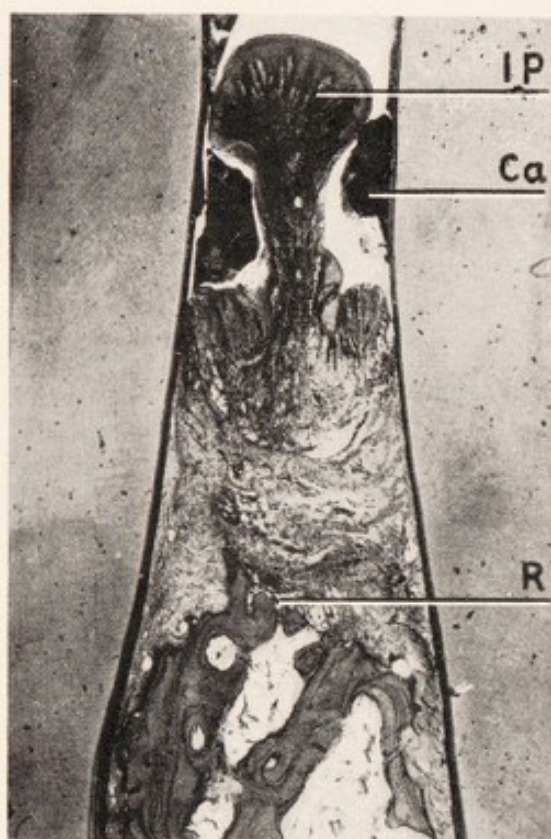


FIG. 255.—Subgingival calculus in the interproximal space between two incisors. Marked swelling and hypertrophy of the interdental papilla. Inflammatory resorption of the crest of the interdental bone septum. IP, interdental papilla; Ca, calculus; R, bone resorption. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)



FIG. 256.—Ulceration and necrosis of the crevice epithelium. Lingual surface of an upper bicuspid. GM, gingival margin; BC, bottom of gingival crevice; U, ulcerated wall of the pocket. Through the breaks in the epithelium, leukocytes migrate from the subepithelial tissues into the crevice (pus formation); EA, epithelial attachment to the cementum.

years. The bottom of the crevice is located on the enamel; between it and the gingival margin, subgingival or serusal calculus extends as a horizontal ridge along the lingual side of the deciduous molar. The subepithelial tissue shows dense inflammatory infiltration; next to the calculus, a small ulceration has developed in the pocket epithelium through which pus is being discharged into the pocket.

In the interproximal spaces, subgingival calculus is frequently

associated with marked swelling and hypertrophy of the interdental papillæ (Fig. 255). If, in a case like the one illustrated in Fig. 255, the interdental areas are subject to a superficial inspection only, the calculus is not discovered since it is hidden by the swollen papillæ; the deposits can then be discovered only by means of an explorer. The pronounced subepithelial inflammation in this case has led to considerable resorption of the crest of the interdental alveolar septum.

The crevice epithelium opposite the subgingival calculus is usually the site of extensive ulceration (Fig. 256). Sometimes it is completely destroyed and the entire surface of the pocket wall appears raw and bleeds easily. After the deposits have been removed by scaling, the wall of the pocket usually becomes epithelized, and the discharge of pus stops. Only if the pocket is extremely deep will suppuration persist after scaling, since the bottom of the crevice fails to heal in such extreme cases (paradental pyorrhea, page 320).

The destruction of the alveolar bone by gingival inflammation under calculus was discussed in the early part of this chapter. The limited extent of marginal bone resorption, the slow progress of the resorptive process, and the absence of any inflammatory changes other than in the immediate vicinity of the alveolar crest indicate that calculus is only of secondary importance as a cause of loosening teeth. The primary etiological factor in producing loosening is the diffuse and extensive destruction of the alveolar bone, which occurs as a disease *sui generis*. Calculus and marginal inflammation are only superimposed secondary complications. As stated before, it takes an unusual degree of gross neglect to let calculus accumulate in such masses and over so long a period of time that the resulting marginal bone destruction actually endangers the firmness of the tooth; whereas, typical diffuse atrophy may actually wreck a perfect, clean set of teeth within a short period of time.

3. Marginal Gingivitis Due to Loss of Contact Point and to Extensive Destruction of the Tooth.—Besides calculus, lack of proper contact, gingival caries, overhanging or defective fillings, and sharp edges of broken-down teeth are the most common causes for chronic marginal gingivitis with more or less marked resorption of the alveolar crest underlying the area of inflammation. For the sake of a clinical and histopathological description, a differentiation will be made between an ulcerative form of gingivitis with destruction of the gingival tissues, especially of the interdental papillæ, and a hypertrophic form with inflammatory tissue proliferation. Which of these two forms will develop in a given case depends upon

the type of inflammation, on the localization of the process, and on the individual tissue reaction. In Figs. 258 and 259 two interdental areas from the same individual are reproduced, illustrating the presence of both forms of interdental gingivitis in the same mouth.

Fig. 257 shows the typical result of the loss of contact point by approximal caries on two molars. Normally, it is the function of the contact point to divert the food toward the labial and lingual

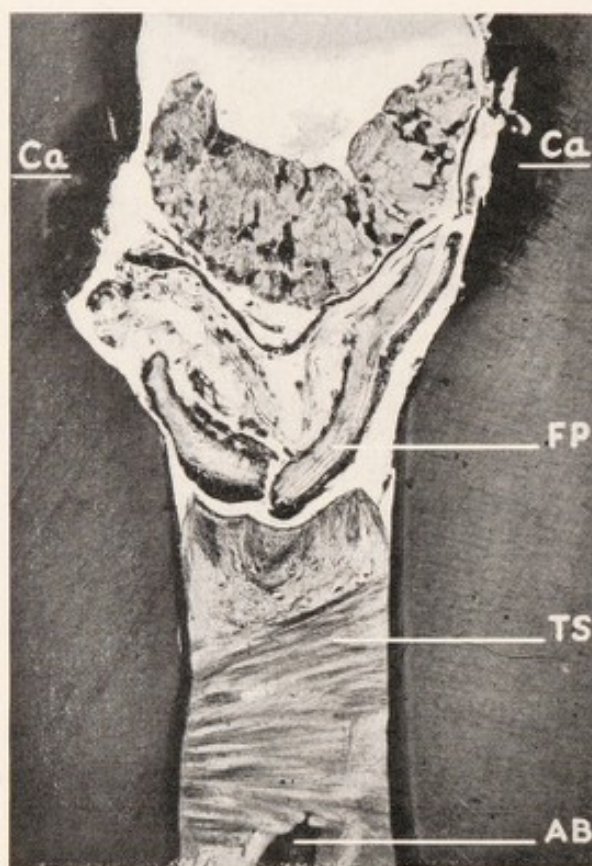


FIG. 257.—Interproximal tissues between two lower molars. Loss of contact point through approximal caries, and destruction of the interdental papilla through food pack. Ca, caries; FP, food particles packed between the teeth and against the papilla; TS, transeptal fibers; AB, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

surface of the teeth, and thus to protect the interdental papilla from injury during mastication. If the contact point is lost, food is packed against the papilla causing destruction and inflammation of the underlying soft tissue, and gradual resorption of the interdental septum of alveolar bone. In Fig. 257 the interdental papilla is concave instead of convex; this shape has been produced by the pressure of food particles of vegetable origin. If such a condition is allowed to persist over a long period of time, both soft and hard tissues between the teeth may eventually be destroyed. If the

condition is remedied by a restoration or filling with proper contact point that prevents further food packing, the interdental tissues will heal, and a new papilla will be established in the level that had been attained when the contact was restored. It must, however, be clearly understood that here, as well as in any other case of healing of marginal inflammatory conditions, there is no possibility of repair in the sense that the tissues grow back to the root surface from which they became detached. The term healing in this connection

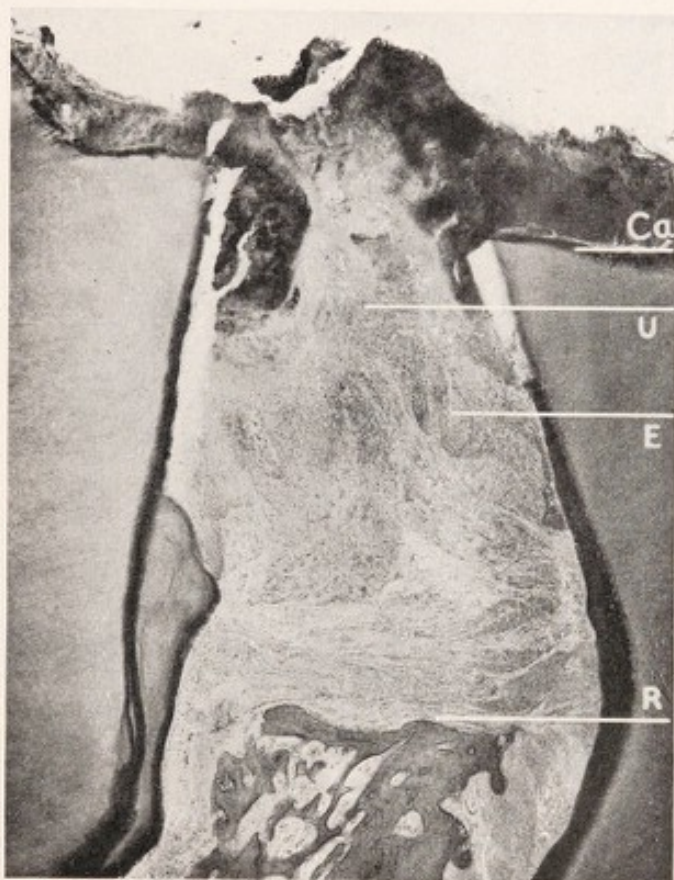


FIG. 258.—Extensive destruction of the interdental soft tissues between two badly broken-down upper molars. Ca, caries; U, ulcerated and necrotic surface of the interdental papilla; E, remnants of the gingival epithelium; R, resorption of the crest of the alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

means only that the destructive process is arrested at a given level; it does not mean regeneration to the *status quo ante*.

Fig. 258 illustrates another case of extensive inflammation and destruction of the interdental papilla between two badly broken-down upper molars. Both crowns have been destroyed almost to the gingival level. The interdental papilla has been transformed into a mass of necrotic tissue, exudate, and inflammatory cells; the flat surface of the interdental gingival tissues consists of degenerating, desquamating epithelium with extensive inflammation extend-

ing far into the subepithelial connective tissue. The crest of the interdental bone has been flattened and shows evidence of active resorption on its surface. Even here, in the presence of extensive necrotic inflammation of the gingiva, the deeper portions of the periodontal membrane are absolutely normal; the inflammation is restricted to the marginal area, and nothing in the actual findings in these specimens justifies the assumption of a deeper or distant injury to periodontal membrane or alveolar bone induced by the superficial inflammatory tissue changes.

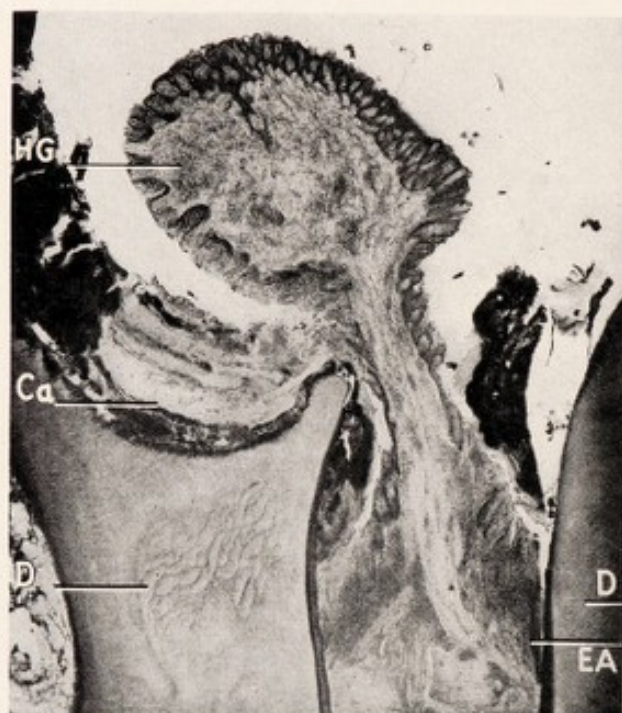


FIG. 259.—Hypertrophic gingivitis (gum polypus) between the roots of two broken-down upper molars. Ca, carious cavity; D, dentin of the roots; EA, epithelial attachment; HG, hypertrophied gingival tissue showing diffuse infiltration with polyblasts. The surface is covered with squamous epithelium.

As stated before, the gingival tissue may also react to chronic irritation by inflammatory proliferation and hypertrophy. Sometimes this hypertrophic gingivitis is general throughout the mouth. At first the condition usually presents itself as a true inflammatory hypertrophy, hyperemia, and swelling of the gingiva. In this stage the tissue appears microscopically as typical granulation tissue. It contains a varying amount of polymorphonuclear leukocytes, depending upon how acute the condition is. If hypertrophic gingivitis persists over a long period of time, a marked fibrosis of the tissues usually takes place; the fibroblasts multiply rapidly, connective tissue fibrillæ are formed, the inflammatory cells disappear, and finally the tissue resembles microscopically a fibroma.

Clinically this transition is characterized by decreasing hyperemia and advancing induration of the hypertrophic gum tissue, until eventually the tissue may resume the color of normal gum tissue and attain a firm, tough consistency.

Localized hypertrophic gingivitis leads to the formation of a so-called gum polypus, a small pedunculated growth that proliferates from the interdental papilla, rarely from any other part of the gingiva, and next to the source of constant irritation and infection. Frequently the mass of hypertrophic gingival tissue is found in a deep carious cavity (Fig. 259). Histologically, the gum polypus



FIG. 260.—Hypertrophic gingivitis (gum polypus) between the mesial and distal roots of a broken-down lower first molar of a child. HG, hypertrophied epithelized soft tissue (granulation tissue) proliferating from the interradicular septum; RC, root canals of the mesial and distal root of the molar.

appears as granulation tissue covered by stratified squamous epithelium; the latter is attached to both neighboring teeth by an epithelial attachment. The amount of vascularization and infiltration in the hypertrophied gingival tissue depends upon the degree of inflammation and infection: if considerable infection or traumatic injury through mastication is present, the epithelial covering is found ulcerated, and subepithelial tissue is hyperemic and densely infiltrated; if the condition is chronic, the covering epithelium is intact or even hornified, and the subepithelial tissue is fibrous and poorly vascularized. Fig. 260 shows a case of hypertrophic gingivitis in a typical location, namely, between the two roots of a completely broken-down lower first permanent molar of a child. The gingival tissue formed a mushroom-shaped growth, overlapping the carious

root surfaces, connected with the tissues of the interradicular septum by a fibrous attachment and with the roots by an epithelial attachment.

PARADENTAL PYORRHEA. (Paradentitis Profunda [Suppurativa] Simplex, F.D.I.)

Paradental pyorrhea has been defined by Gottlieb as a condition in which suppuration from deep pockets is present and persists in spite of routine procedures, such as scaling and brushing. This definition requires some additional explanation. The evidence of suppuration alone does not justify the clinical diagnosis of paradental pyorrhea, since suppuration may occur without the presence of pockets (see Fig. 253); however, in the latter case the discharge is promptly arrested by the removal of the irritation. The presence of rather deep pockets alone does not substantiate the diagnosis either, since these deep pockets may be lined by intact epithelium and then may not present suppuration. Only if such deep pockets have ulcerated walls discharging pus, and if the breaks in the epithelium fail to heal because the pocket forms a deep recess and a habitually unclean area, is the diagnosis paradental pyorrhea justified.

The etiology of paradental pyorrhea is twofold: local and systemic. Local causes of the development of paradental pyorrhea are all circumstances that bring about a relative acceleration of the detachment of the gingival tissues from the tooth surface without simultaneous atrophy of the detached soft tissues, thus resulting in excessive deepening of the pocket. Such causes are: direct mechanical injuries to the soft tissues at the bottom of the pocket by toothpicks, loose bristles of the tooth-brush, food packs, etc.; occlusal trauma with marked lateral stress resulting in injury to the periodontal tissues on the side of pressure (see Figs. 261 and 262); unequal rate of detachment of the soft tissues in the interproximal area, resulting in a great difference between the levels of attachment (see Fig. 265).

A systemic cause of the development of paradental pyorrhea is diffuse atrophy of the alveolar bone. In this disease, deep pockets may develop rather rapidly, accompanying the loosening or pathological wandering of the affected teeth. The sequence of changes, in this case, is that the destruction of bone occurs first; this is followed by rapid apical proliferation of the epithelial attachment and detachment from the root surface with resulting deep pockets. These pockets, being habitually unclean, become infected and sup-

puration starts. In pronounced pathological wandering in a horizontal direction, the paradental pocket develops on the side from which the wandering tooth moved; for instance, in the labial drifting of an upper incisor, the pocket develops on the lingual side of the root, in diastema formation, between the two teeth, etc.

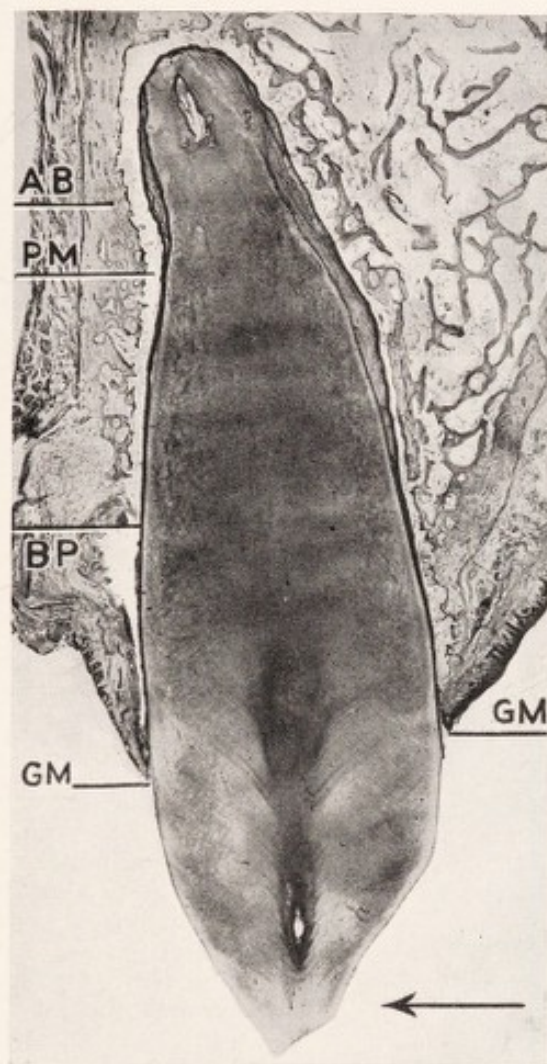


FIG. 261.

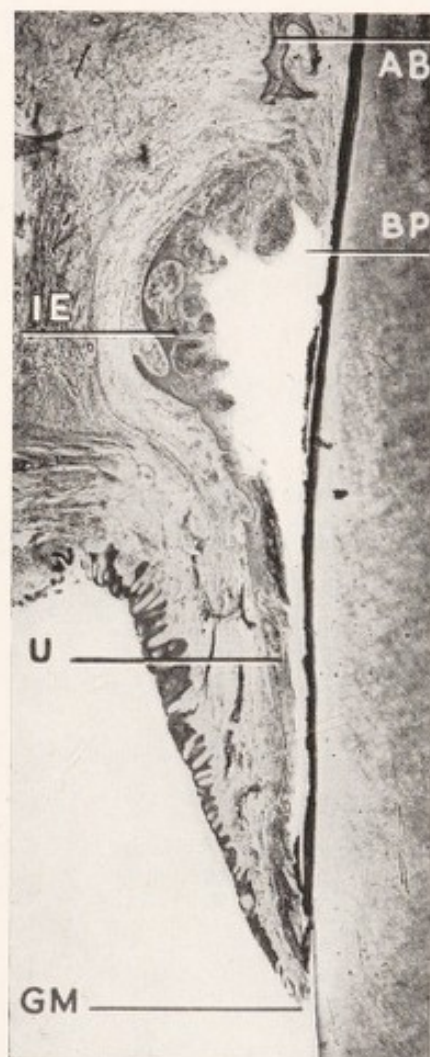


FIG. 262.

FIGS. 261 and 262.—Paradental pyorrhea on the labial side of an upper cuspid. The arrow indicates the direction of the occlusal stress. On the labial surface there is a pocket of about 7 mm. in depth; on the lingual surface the depth of the pocket is zero. (See Fig. 264.)

FIG. 261.—General view of the cuspid. Labio-lingual section. GM, gingival margin; BP, bottom of paradental pocket on the labial side; PM, periodontal membrane; AB, alveolar bone.

FIG. 262.—Higher magnification of the labial surface of the tooth shown in Fig. 261. GM, gingival margin; U, ulcerated wall of the pocket; BP, bottom of the pocket; IE, inflammatory proliferation of epithelium; AB, alveolar bone.

Figs. 261 and 262 illustrates a typical case of paradental pyorrhea due to lateral occlusal stress. The specimen is an upper cuspid of a man, aged thirty-eight years. The mouth had been badly

mutilated by extractions. The bite was very deep, and the lower cuspid was almost completely covered by the overbite of the upper antagonist, so that the latter was exposed to considerable lateral stress in a labial direction as indicated by the arrow in Fig. 261. The bottom of the gingival crevice had passed the cemento-enamel junction in its entire circumference. On the lingual and partly on the mesial and distal sides, the bottom of the crevice is located just rootward from the cemento-enamel junction; the crevice in this area is very shallow and healthy. On the labial side, the side of pressure, however, the tissues have become detached from the cervical third of the root surface; the gingival margin remained in the same level as on the lingual side, forming a pocket 7 mm. in depth. The opening of the pocket is very narrow; toward the bottom the



FIG. 263.—High magnification of the wall of the paradental pocket in Fig. 262. I, subepithelial accumulation of inflammatory exudate cells; E, remnants of the crevice epithelium; X, breaks in the epithelium through which leukocytes migrate into the pocket.

pocket becomes wider and attains its greatest width in an ampulla-shaped dilatation near the bottom. The epithelial tissue shows extensive inflammatory infiltration, especially near the bottom of the pocket. About one-third of the lingual plate of the alveolar bone has been lost by resorption. Along the mesial and distal sides of the root the bottom of the pocket must necessarily extend on a slant from the more apical attachment on the labial side to the cervical attachment on the lingual side.

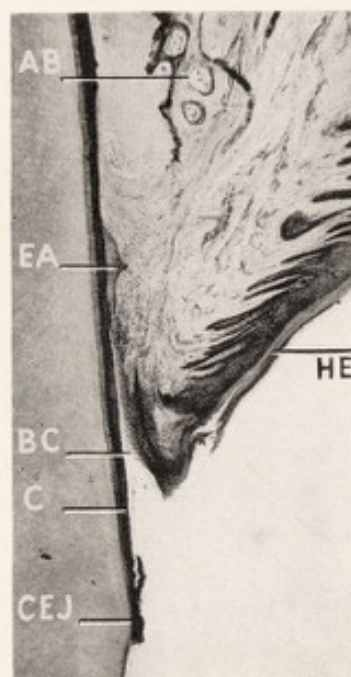


FIG. 264.—Higher magnification of the gingival margin on the lingual side of the cuspid in Fig. 261. Normal gingival crevice. The depth of the crevice is practically zero. CEJ, cemento-enamel junction; C, cementum; BC, bottom of gingival crevice located on the cementum; EA, epithelial attachment to the cementum; HE, hornification of the gingival epithelium; AB, alveolar bone.

The specimen illustrated in Figs. 261 and 262 brings up the question of the therapy in case of such a deep paradental pocket. There is no doubt that the cuspid was firm at the time of the death of the individual from whom the specimen was obtained, since intact attachment of the periodontal membrane on the entire lingual and on the greater part of the mesial, distal, and labial root surfaces is sufficient to hold the tooth firmly in place. But there also can be no doubt that if such a condition is allowed to continue, more and more of the root surface will gradually lose its attachment to the gingiva and will become denuded. In such a deep pocket a vicious circle develops: the existing inflammation and suppuration destroy the tissue attachment and cause deepening of the pocket, which in turn is followed by an increase in inflammation and discharge of pus.

The therapy in a case like the one illustrated in Figs. 261 and 262 consists in the removal of the entire outer wall to the very bottom of the pocket. All the tissue between *GM* and *BP* would have to be removed in order to reestablish clean conditions and to prevent progressive destruction. The removal of the flap that overlies the pocket can be done by surgical excision, by cauterization, by packing, or by a combination of any of these methods. If properly carried out, such therapy will result in the establishment of a crevice of zero depth on the level of *BP*. The root surface between *BP* and *GM* will, of course, be exposed which is esthetically undesirable; however, this is the only safe way to eliminate inflammation and suppuration and to reestablish hygienic conditions. Any etiological factors involved, such as occlusal conditions, will, of course, have to be remedied at once to prevent recurrence of the pocket.

1. **The Problem of Reattachment.**—The clinical observation of individual deep pockets has led quite naturally to attempts to bring about a reattachment of the detached gingival tissues to the denuded root surface. In studying Figs. 261 and 262 it is easy to understand the anatomical basis for this procedure: reattachment would mean that the soft tissues between *GM* and *BP* would be reunited with the root surface and the pocket would disappear. A number of clinicians and pathologists report reattachment of the gum tissue to a denuded root surface in a deep pocket following treatment and medication. However, the author is of the opinion that such reports have to be considered with a scepticism based upon practical as well as theoretical considerations.

The difficulty in diagnosing reattachment after treatment of a deep pocket lies in the fact that the wall of the pocket invariably

has a tendency to shrink; as a result, a pocket may appear shallower after treatment than before, but the reduced depth may be due to contraction of the pocket wall and not to reattachment. The same process of shrinkage can also bring about a close adaptation of the formerly loose, flabby pocket to the root surface, thus making exploration of the pocket difficult and producing the clinical appearance of an obliteration of the pocket by reattachment. In order to prove beyond doubt that the tissue has actually become reattached to the root surface, it would be necessary to measure the exact distance to the bottom of the crevice, before and after the treatment, from a fixed point on the tooth surface, as, for instance, from the gingival margin of a filling; reattachment may be claimed only if such measurements show that the distance from the bottom of the pocket to the margin of the filling has actually become smaller. To the author's knowledge such measurements have not yet been reported.

The second reason for the author's scepticism about the possibility of reattachment is the consideration that the root surface, once it has become denuded and exposed over a long period of time, is in a biological sense a dead surface. This fact can perhaps be expressed more clearly by comparing it to the replantation of a tooth (see page 240). If a tooth be extracted and replanted immediately thereafter, it becomes firm through reattachment of the periodontal fibers to the living cementum surface. This reattachment after replantation is performed by deposition upon the root surface of new layers of cementum in which new periodontal fibers have been embedded. If, however, the extracted tooth is macerated, boiled, or dried so that the vitality of the root surface is lost and the tooth transformed into a dead piece of ivory, it will not heal in, since deposition of living cementum upon a necrotic surface is not likely to occur. In the problem of reattachment, the denuded root surface inside the pocket represents such a necrotic, macerated and infected hard tissue surface; under these circumstances, deposition of new layers of cementum, without which a functional reattachment is impossible, is unlikely and up to this time has not been demonstrated. The question of whether cementum or dentin is exposed in the crevice is without significance. It is known that new cementum may be deposited indiscriminately upon both cementum and dentin, for instance, following root fracture or root resorption (see Figs. 352, 353, and 354). However, in these cases, the surface of dentin and cementum had never been denuded; whereas, in case of a paradental pocket, the dental hard

substances have been exposed for months or years to infection and maceration.

Another complication is the presence of the epithelial attachment to the root surface. As long as the wall of the pocket is covered by epithelium, reattachment of connective tissue is impossible. But even if it were possible to remove all epithelium to the very bottom of the pocket, there is still present beyond this point the epithelial attachment from which new epithelium can proliferate and interfere with cementum formation and connective tissue reattachment (see Fig. 256).

All these factors contribute to make the conditions for reattachment relatively unfavorable. Therefore, the author believes that even though there might be theoretically a certain possibility of reattachment, the practical value of the therapeutic methods based upon this possibility is rather questionable. But the author wishes also to emphasize his willingness to revise his opinion on this subject if clinical proof of reattachment could be established by exact measurements and if deposition of cementum and attachment of functioning periodontal fibers could be demonstrated on a portion of the root surface that previously was denuded and exposed in the pocket.

2. Paradental Pyorrhea on the Approximal Surfaces of Teeth.—One of the causes of the development of paradental pockets that was mentioned at the beginning of this discussion is the different degree of detachment of the soft tissues from the surfaces of two neighboring teeth. If the detachment from one root is rather far advanced,



FIG. 265.—Paradental pyorrhea in the interproximal space between two lower incisors. Deep pocket caused by a different level of attachment of the interdental papilla on the two teeth. IP, interdental papilla; BC', bottom of the crevice on the right tooth; shallow crevice, intact crevice epithelium. BC'', bottom of the crevice on the left tooth; deep crevice with ulcerated crevice epithelium, UE; I, subepithelial inflammation; R, resorption of the alveolar bone at the crest of the interdental septum.

and on the tooth next to it the attachment is still near the cemento-enamel junction, shrinkage and atrophy of the papilla cannot keep pace with the detachment at the bottom of the crevice. The result is the development of a paradental pocket. Fig. 265 illustrates such a condition in the interdental space between two lower incisors. On the right side the pocket is shallow; the papilla is normal and covered with a keratinized layer. On the left side the bottom of the pocket is located at a considerably deeper level; on this side the wall of the papilla is ulcerated, discharging pus into the paradental pocket.

In deep paradental pockets with narrow openings, acute purulent inflammation is occasionally observed; this condition which is caused by accumulation and retention of infective material in the pocket is called paradental abscess or lateral abscess; it must not be confused with periapical abscesses from infected pulpless teeth.

ALVEOLAR ATROPHY AS A RESULT OF EXCESSIVE OCCLUSAL STRESS.

The loss of alveolar bone as a result of excessive occlusal stress is most commonly observed in mouths that have been mutilated by extractions and for which no restorations have been made to replace the lost teeth. In such mouths the remaining teeth have to bear an unusually heavy load of functional stress since a small number of teeth are now subject to the same amount of mastication and muscular force that is normally distributed over all thirty-two teeth. Another condition that is likely to produce excessive occlusal stress is, for example, the forward tipping of a lower second molar following extraction of the first molar. Such a tipped tooth is exposed to considerable horizontal (lateral) stress which, for reasons that will be discussed in detail in Chapters XII and XIII, leads much more easily to injury of the periodontal tissues than vertical (axial) stress does.

Under the above-mentioned circumstances teeth may become loosened through pressure atrophy of the alveolar bone. Whether they become loose depends upon the individual tissue reaction and tissue resistance. In some mouths one tooth after the other will be loosened and finally lost by occlusal overstress. Other patients, with only a few teeth in place and these often in the most unfavorable occlusal conditions, will be able to carry on for decades without any damage to these "overworked" teeth. The microscopic examination of such teeth reveals a strengthening of the periodontal

membrane and reinforcement of the supporting bone indicating favorable resistance and adaptation to the increased functional requirements rather than tissue injury (see pages 344, 350).

A typical example of the loosening of a tooth under excessive stress is illustrated in Fig. 266. The specimen under consideration is a lower second bicuspid. The incisors and the first bicuspid were missing, as well as all lower molars. In the upper jaw all teeth were present. As a result of the closing of the bite as well as the

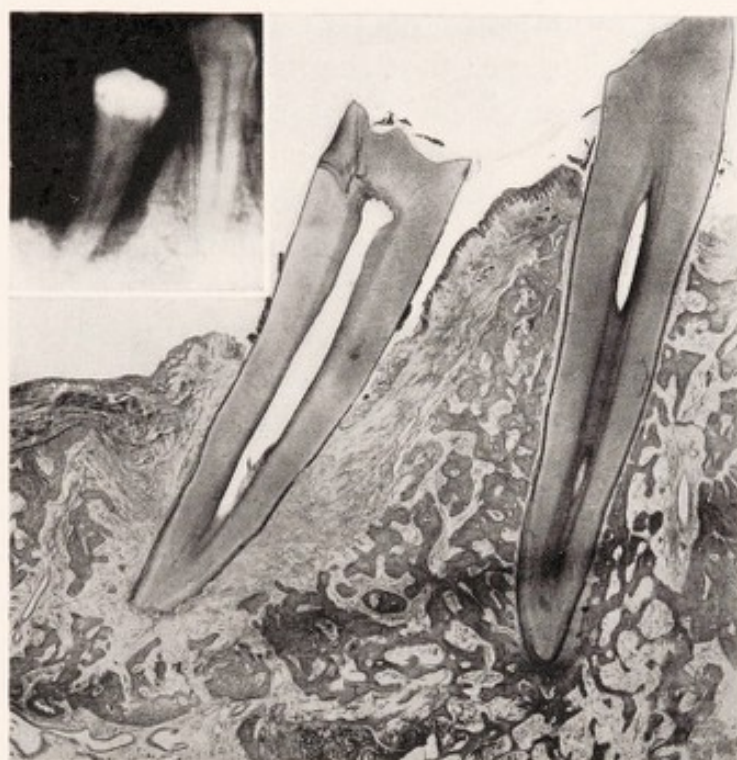


FIG. 266.—Mesial tipping of a lower right second bicuspid after the loss of the neighboring teeth. Extensive destruction of the alveolar bone. The radiograph of the specimen in the upper left corner shows the amount of bone destruction. In a mesio-distal section through the jaw, the bicuspid appears embedded in fibrous connective tissue. Notice the location of the bottom of the gingival crevice on the root surface of the bicuspid. Despite the extensive loss of alveolar bone, about two-thirds of the surface of the anatomical root are still in organic connection with the surrounding tissues. The periodontal tissues of the lower cuspid are intact.

loss of the first bicuspid, the second bicuspid was tipped forward, until at the time of the individual's death, the long axis of the tooth had been tipped about 30 degrees from its normal vertical position. The radiograph shows extensive loss of the alveolar bone except for a small area at the apex. The microscopic examination reveals even a more extensive absence of bone than could be seen in the radiograph: the radiographic appearance of bone at the apex apparently is the result of an optical projection of the bone on the buccal and lingual side of the mandible over the apex; in a central section

through the tooth, no alveolar bone whatsoever is left around the apex. The bicuspid is embedded in fibrous connective tissue that is attached to the apical two-thirds of the root surface, a fact which is of great practical significance. It illustrates very plainly that there is by no means a definite relationship between location of the bottom of the pocket and position of the alveolar crest. The bone has been resorbed as a result of occlusal pressure; however, the detachment of the remaining periodontal soft tissues did not follow the recession of the bone.

The practical importance of this observation can hardly be overemphasized as it shows that the usual procedure of diagnosing "pockets" from a radiograph is not correct. A pocket is determined by the attachment of the soft tissue, not of the bone; a radiograph can demonstrate a pocket only after a radiopaque material (for instance, a guttapercha point) has been introduced into the pocket and is radiographed with the tooth. The only thing that can actually be seen in the routine radiograph is the amount of bone present; the supposition that loss of bone and formation of a pocket must necessarily go hand in hand is not substantiated by the actual examination of the tissues involved.

In the discussion of the clinical prognosis of a tooth that is loosened by excessive occlusal stress, the attachment of the soft tissues will again have to be considered first and the condition of the bone second. To explain this with a concrete example: From the radiograph, the tooth illustrated in Fig. 266 certainly does not look very promising. However, if the pockets were explored, it would be found that a considerable amount of soft tissue attachment was still present and that not more than about one-third of the anatomical root was exposed above the bottom of the crevice. Therefore, there was a definite possibility that the alveolar bone might regenerate around the lower two-thirds of the root, provided that the injurious stress were relieved. If, on the other hand, exploration of the pockets revealed that the detachment of the soft tissues kept pace with the bone destruction so that the greatest portion of the root surface was denuded, the prognosis would be hopeless; reattachment could not be expected under such circumstances and without connective tissue attachment bone regeneration is impossible.

The author has discussed these and other clinical points in great detail because he feels that there is too much loose diagnosis in the field of periodontal diseases, as well as in many other fields of dental pathology, without clear and positive knowledge of the tissue changes

involved; as a result the various clinicians and investigators come to theoretical and practical conclusions that are not substantiated by the actual findings in microscopic examinations of human specimens.

DIFFUSE ATROPHY OF THE ALVEOLAR BONE. (Dystrophia Diffusa; in Later Stages: Parodontitis Dystrophicans Complicata, F.D.I.)

The term diffuse atrophy for a primary lesion of the alveolar bone with clinical symptoms of loosening and wandering was first used more than ten years ago by Gottlieb in connection with his investigations concerning the etiology of pyorrhea. Since that time several investigators studying the same clinical condition have introduced new terms, which to some extent indicate the independence of this condition from purely local conditions. The clinical aspect of diffuse atrophy has been described (see page 300); the microscopic findings will be discussed here.

Typical diffuse atrophy of the alveolar bone is by no means a frequent disease. It is, therefore, not surprising that histological material for its study is extremely hard to obtain. The best known case is one, the microscopic findings of which were published by Gottlieb in 1923. The patient, a man, aged twenty-two years, had died of pneumonia following influenza (grippe) and from general exhaustion. All teeth that were examined microscopically showed resorption of both root surface and alveolar bone with marked widening of the periodontal space and transformation of the fibrous periodontal membrane into loose, highly vascular connective tissue without functional orientation (Figs. 268 and 269). The resorptive processes and the changes in the structure of the periodontal membrane are confined to the apical two-thirds of the roots; the gingival third of the root, as well as the interdental tissues, is intact. The individual being relatively young, the gingival crevice is still found on the enamel; very little subepithelial inflammation is present in the gingival tissue. The bone at the alveolar crest is intact.

Resorption of the alveolar bone and widening of the periodontal membrane are expressed clinically by loosening of the teeth. The destruction of the periodontal fibers and their transformation into loose connective tissue explains the displacement of the teeth known as pathological wandering. If the destructive process takes place in the apical portion of a tooth, the loss of the fibers and the tension of the newly formed connective tissue in this area cause elongation (extrusion, pathological wandering in vertical direction).



FIG. 267.—Diffuse atrophy of the alveolar bone. Man, aged twenty-two years. Evidence of resorption on the roots, especially in the apical portion. Loss of the normal fibrous structure of the periodontal membrane, and resorption of the adjacent alveolar bone. The general view of the upper central and part of the lateral incisor shows the location of the resorptions, R, at the apices of both teeth. The remaining portion of the periodontal membrane appears normal; the gingival tissues are intact; no deposits are present. The bottom of the crevices is located on the enamel. (Gottlieb, Jour. Am. Dent. Assn.)

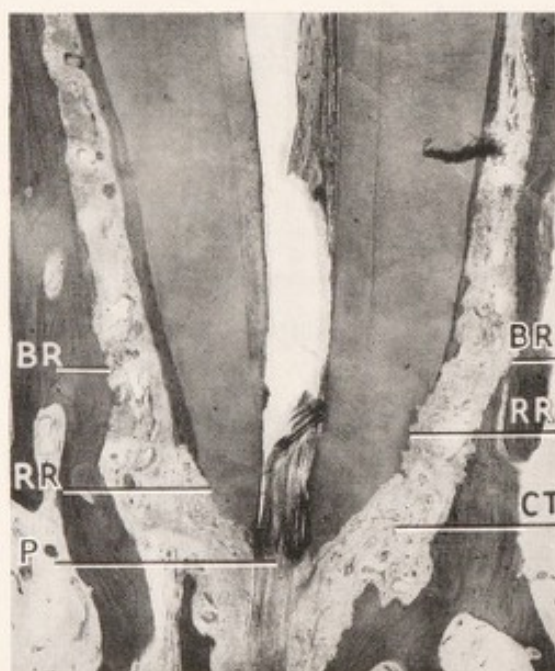


FIG. 268.—Higher magnification of the apex of the central incisor. RR, resorption of the root surface; CT, loose vascular connective tissue replacing the fiber bundles of the periodontal membrane; BR, bone resorption; P, pulp strand passing through apical foramen. Notice the irregular widening of the periodontal space around the entire apical third of the root.

If the changes take place on one side of the root, the tooth will be displaced toward the opposite side (pathological wandering in horizontal direction, drifting); a paradental pocket usually develops later on the damaged side of the root (see page 320).

Gottlieb offers the following explanation for the unusual observation of root and bone resorption in all teeth of the same jaw: the existence of the alveolar bone depends upon the presence of the teeth, and unless sufficient and regular stimuli are transmitted from the root surface *via* Sharpey's fibers to the bone, the latter becomes atrophic. In case of diffuse atrophy, it must be assumed that the

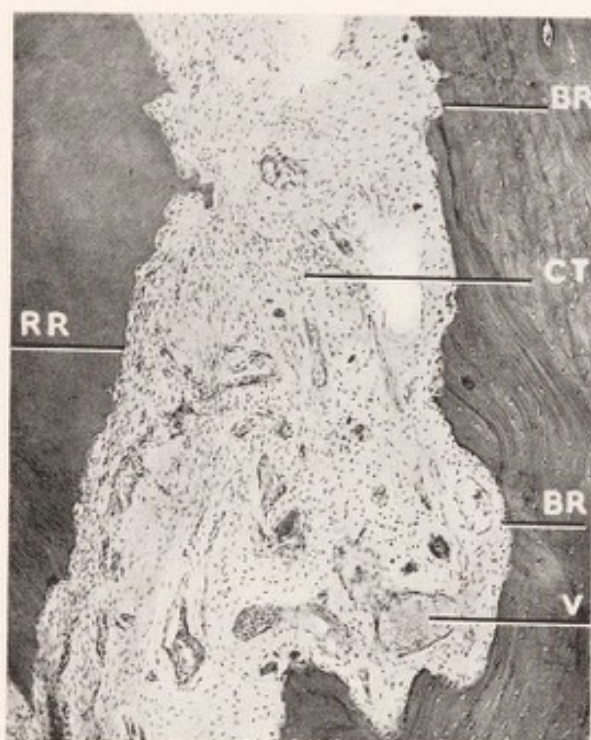


FIG. 269.—High magnification of a portion of the root surface near the apex in Fig. 268. RR, resorbed dentin surface; CT, loose connective tissue; V, bloodvessels; BR, bone resorption.

root surface has been injured in some way, resulting primarily in lowered vitality of the cementum and loss of attachment of the fibers to the root surface. Subsequently the bone, receiving no more stimuli in the damaged area, is resorbed without sufficient repair and becomes atrophic. In the case illustrated here, influenza might have been the cause for the loss of vitality of the root surface; in other cases pregnancy (Fig. 246) or some systemic or metabolic disturbance may play a rôle in the etiology. In all these instances, however, it seems that a systemic disturbance produces a primary lesion in the periodontal membrane and on the root surface resulting in the failure of the periodontal membrane to deposit

new layers of cementum. Thus the functional unison of cementum, periodontal membrane and bone is disturbed; the bone receives no more functional stimuli and is resorbed.

Occlusal trauma is of only secondary importance in the development of diffuse atrophy. Once a tooth has begun to change its position as the result of pathological wandering, it will sooner or later be subject to excessive occlusal stress which will, in turn, accelerate its loosening. Therefore, the occlusion must be kept very carefully under observation, and whenever a tooth shows a tendency to shift or elongate it must be ground out of occlusion to prevent secondary occlusal overstress and to give the periodontal tissues a chance to recover.

The prognosis of diffuse atrophy is doubtful. In some cases the widening of the periodontal membrane and the loss of alveolar bone continues until the tooth has been eliminated. In other instances, the pathological process may come to a temporary or permanent standstill; this finds its histological expression in new formation of cementum on the root surface with establishment of new fibers and regeneration of the alveolar bone due to the renewed functional stimuli. No such repair is possible where deep pockets have already formed, because the presence of the pockets precludes the deposition of new cementum on the root surfaces.

BIBLIOGRAPHY.

- BAUER, W., and LANG, F. J.: Über das Wandern der Zähne, *Vrtljschr. f. Zhk.*, 1928, **44**, 321.
- BECKS, HERMANN: Normal and Pathologic Pocket Formation, *Jour. Am. Dent. Assn.*, 1929, **16**, 2167.
- What Factors Determine the Early Stage of Parodontosis (Pyorrhea)? *Jour. Am. Dent. Assn.*, 1931, **18**, 922.
- BECKS, HERMANN, and WEBER, MORITZ: The Influence of Diet on the Bone System with Special Reference to the Alveolar Process and the Labyrinthine Capsule, *Jour. Am. Dent. Assn.*, 1931, **18**, 197.
- BLACK, G. V.: *Special Dental Pathology*, Chicago, Medico-Dental Publishing Company, 1920.
- BOX, H. K.: *Treatment of the Periodontal Pocket*, Univ. Toronto Press, 1928.
- Necrotic Gingivitis (Trench Mouth)*, Univ. Toronto Press, 1930.
- BUNTING, R. W.: *A Text-book of Oral Pathology*, Philadelphia, Lea & Febiger, 1929.
- COOLIDGE, EDGAR D.: Inflammatory Changes in the Gingival Tissue Due to Local Irritation, *Jour. Am. Dent. Assn.*, 1931, **18**, 2255.
- GOTTLIEB, B.: Die Parodontalpyorrhoe der Rattenmolaren, *Vrtljschr. f. Zhk.*, 1922, **38**, 274.
- Die diffuse Atrophie des Alveolarknochens, *Ztschr. f. Stom.*, 1923, **21**, 195.
- Schmutzpyorrhoe, Parodontalpyorrhoe und Alveolaratrophie, Wien, Urban & Schwarzenberg, 1925.

- GOTTLIEB, B.: Paradentalpyorrhöe und Alveolaratrophie, yearly contribution in *Fortschr. d. Zhk.*, Leipzig, Thieme, 1925-1932, vols. 1-8.
- Tissue Changes in Pyorrhea, *Jour. Am. Dent. Assn.*, 1927, **14**, 2178.
- Formation of the Pocket; Diffuse Atrophy of the Alveolar Bone, *Jour. Am. Dent. Assn.*, 1928, **15**, 462.
- HÄUPL, K., and LANG, F. J.: *Marginale Parodontitis*, Berlin, Meusser, 1927.
- HIRSCHFELD, ISADOR: Food Impaction, *Jour. Am. Dent. Assn.*, 1930, **17**, 1504.
- JONES, MARTHA R., and SIMONTON, F. V.: Mineral Metabolism in Relation to Alveolar Atrophy in Dogs, *Jour. Am. Dent. Assn.*, 1928, **15**, 881.
- McCALL, JOHN OPPIE: An Improved Method of Inducing Reattachment of the Gingival Tissues in Periodontoclasia, *Dent. Items Int.*, 1926, **48**, 342.
- MARSHALL, JOHN ALBERT: *Diseases of the Teeth. Their Diagnosis and Treatment*, Philadelphia, Lea & Febiger, 1926.
- ORBAN, B.: Ist das "Paradentium" eine "organische Einheit?" *Ztschr. f. Stom.*, 1926, **24**, 515.
- Hornification of the Gums, *Jour. Am. Dent. Assn.*, 1930, **17**, 1977.
- PRINZ, HERMANN: *Diseases of the Soft Structures of the Teeth and Their Treatment*, Philadelphia, Lea & Febiger, 1928.
- REPPETO, W. M.: Histopathologic Studies of the Superficial Periodontal Tissues, *Jour. Am. Dent. Assn.*, 1932, **19**, 817.
- SKILLEN, W. G.: Normal Anatomic and Physiologic Gingiva and Its Relation to Pathologic Processes, *Jour. Am. Dent. Assn.*, 1931, **18**, 600.
- A Contribution to the Anatomy and Pathology of the Human Gingiva, *Jour. Dent. Res.*, 1931, **11**, 727.
- SORRIN, SIDNEY, and MILLER, S. C.: *Practice of Periodontia*, New York, The Macmillan Company, 1928.
- STEIN, G., and WEINMANN, J.: Die physiologische Wanderung der Zähne, *Ztschr. f. Stom.*, 1925, **23**, 733.
- STILLMAN, P. R., and McCALL, J. O.: *A Text-book of Clinical Periodontia*, New York, The Macmillan Company, 1922.
- WEINMANN, J.: Das Knochenbild bei Störungen der physiologischen Wanderung der Zähne, *Ztschr. f. Stom.*, 1926, **24**, 397.
- Stoffwechselbefunde bei der diffusen Atrophie des Alveolarknochens, *Ztschr. f. Stom.*, 1927, **25**, 822.
- Stoffwechseluntersuchungen bei der diffusen Atrophie des Alveolarfortsatzes, *Ztschr. f. Stom.*, 1930, **28**, 1154.

CHAPTER XII.

INFLUENCE OF FUNCTION UPON TEETH AND SURROUNDING STRUCTURES.

ARRANGEMENT AND FUNCTION OF THE FIBERS OF THE PERIODONTAL MEMBRANE.

THE root of a tooth is attached to its socket by groups of fiber bundles, the Sharpey's fibers, which constitute the periodontal membrane or dento-alveolar ligament. These fibers can be divided into five groups according to their course and distribution (G. V. Black): (1) transeptal fibers, (2) alveolar crest fibers, (3) horizontal dento-alveolar fibers, (4) oblique dento-alveolar fibers, and (5) apical fibers (Figs. 270 and 271). An additional sixth group of fibers, the free gingival fibers, run from the alveolar margin to the gingiva and therefore do not contribute to the mechanical retention of the root in the socket.

1. **Transeptal Fibers.**—These fibers are confined to the area between two adjacent teeth; they run from the cementum at the neck of one tooth in a more or less horizontal direction across the crest of the interdental septum to the cementum of the next tooth (Fig. 272). The arrangement of the transeptal fibers indicates that their function consists in maintaining the mesio-distal relationship between neighboring teeth and in stabilizing the teeth against separating forces.

2. **Alveolar Crest Fibers.**—The alveolar crest fibers are a rather variable group of fibers. In some teeth they are well developed, in others entirely missing. They run from the crest of the alveolus to that area of the cementum which is bordered crownward by the deepest point of the epithelial attachment or by the transeptal fibers, and is bordered rootward by the crest of the alveolar bone (Fig. 273). Their function is identical with that of the horizontal dento-alveolar fibers described under 3.

3. **Horizontal Dento-alveolar Fibers.**—Horizontal dento-alveolar fibers are found around the entire tooth running horizontally from the alveolar margin to the cementum (Fig. 273). These fibers were originally called the "circular ligament of the tooth." Their function is to stabilize the root against lateral stress.

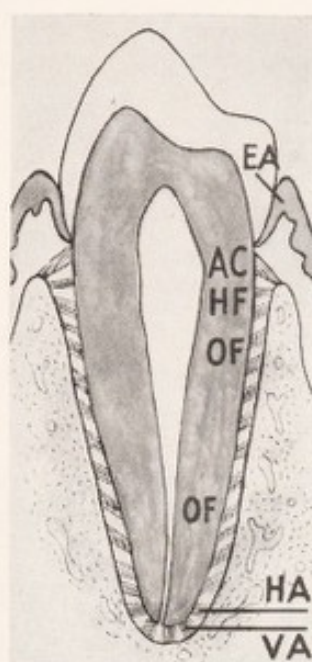


FIG. 270.

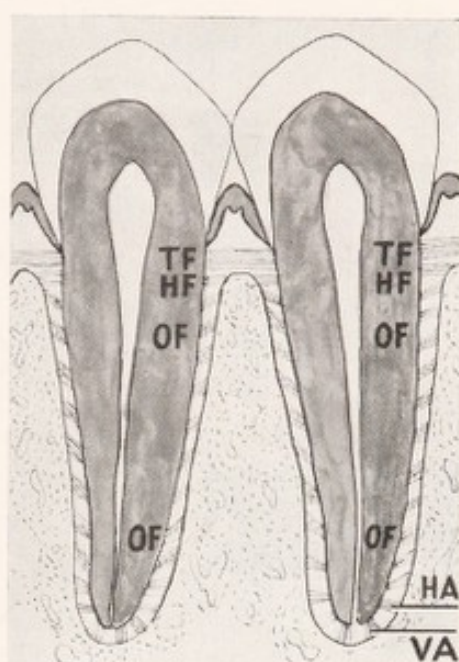


FIG. 271.

FIGS. 270 and 271.—Fiber groups of the periodontal membrane.

FIG. 270.—Bucco-lingual section through lower first bicuspid. EA, epithelial attachment; AC, alveolar crest fibers; HF, horizontal dento-alveolar fibers; OF, oblique fibers; HA, apical fibers, horizontal group; VA, apical fibers, vertical group.

FIG. 271.—Mesio-distal section through lower first and second bicuspid. TF, transeptal fibers; HF, horizontal dento-alveolar fibers; OF, oblique fibers; HA, apical fibers, horizontal group; VA, apical fibers, vertical group.



FIG. 272.—Transeptal fibers between upper central and lateral incisors. TS, transeptal fibers; AB, alveolar bone; OF, oblique fibers; CEJ, cemento-enamel junction; E, enamel; EA, epithelial attachment to the enamel; BC, bottom of gingival crevice; IP, interdental papilla.

The location of transeptal fibers, alveolar crest fibers, and horizontal dento-alveolar fibers on the tooth surface is subject to variations, depending upon age. In young individuals all these fibers are found immediately below the cemento-enamel junction. With advancing age, together with the downward growth of epithelium along the root surface and with the involution of the alveolar crest, the various groups are located further rootward. A typical example of this change in location has been illustrated in Fig. 241, which shows the transeptal fibers in their characteristic relation-

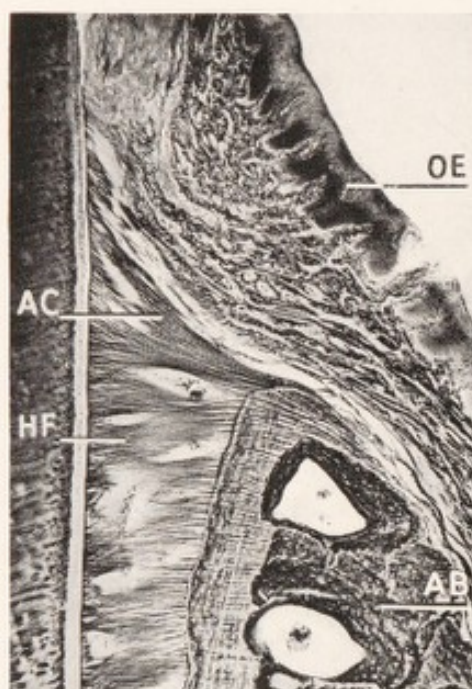


FIG. 273.—Alveolar crest fibers and horizontal dento-alveolar fibers on the lingual side of an upper second bicuspid. Silver staining. AB, alveolar bone; AC, alveolar crest fibers; HF, horizontal dento-alveolar fibers; OE, oral epithelium.

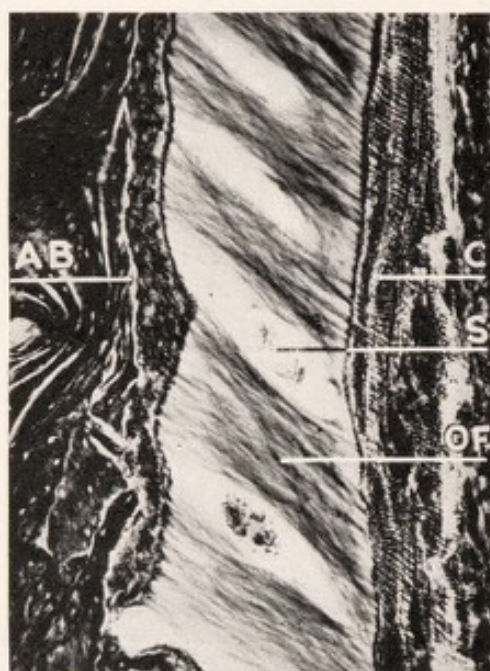


FIG. 274.—Oblique dento-alveolar fibers. Longitudinal section through the root. Silver staining. C, cementum; OF, oblique fibers; S, spaces between the fiber bundles containing the bloodvessels and nerves of the periodontal membrane; AB, alveolar bone.

ship to teeth and bone in a case of considerable exposure of the root surface.

4. Oblique Dento-alveolar Fibers.—This group comprises the bulk of the fibers that form the periodontal membrane. Their arrangement can be understood best by comparing longitudinal and horizontal sections. In a longitudinal section through the root, the oblique fibers run apically from the inner plate of the alveolus to the root surface at an angle of about 45 degrees (Fig. 274). The fibers are arranged in strong, dense bundles with narrow spaces for the periodontal bloodvessels and nerves between them. In a

horizontal section through the root, the oblique fibers are arranged radially; from prominent points or single trabeculae of bone on the inside of the alveolus, their bundles spread fan-wise and are attached to the opposite surface of the cementum.

The function of the oblique fibers is to attach the root to its socket and to counteract occlusal stress. They suspend the root in the alveolus, thus transforming occlusal pressure into a pulling action upon the alveolar bone. It is well known that bone is much better able to withstand pull than pressure. Increased pull leads to functional hypertrophy of bone; increased pressure causes bone resorption. The arrangement of the oblique fibers appears an effective mechanism to transmit the pressure of mastication to the bone in the best form, namely, as pull.

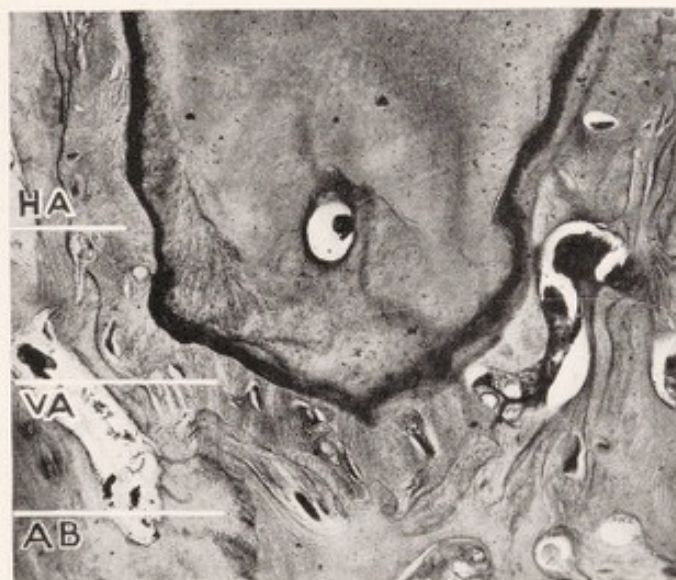


FIG. 275.—Apical fibers at the apex of a lower cuspid. VA, vertical group of apical fibers; HA, horizontal group of apical fibers; AB, alveolar bone.

5. Apical Fibers.—The fibers around the apex can be subdivided into two minor groups: horizontal and vertical apical fibers. Both types frequently show a more or less rudimentary development, and in many specimens are entirely missing. The horizontal apical fibers run in a horizontal direction from the apex of the tooth to the bone; they form a counterpart to the horizontal dento-alveolar fibers, and their functional significance is the same as of this group. The vertical apical fiber bundles run vertically from the root end to the bottom of the alveolus, thus helping to counteract lateral displacement of the apical region of the tooth. These fibers are found only in teeth with fully developed root ends (Fig. 275).

All fiber bundles of the periodontal membrane consist of col-

lagenous fibers without elastic elements. In a state of complete relaxation, the bundles assume a slightly wavy course; minute movements of the tooth are possible by stretching the S-shaped fibers into straight lines.

MASTICATORY FORCES EXERTED UPON HUMAN TEETH.

The masticatory forces may be subdivided into two main groups: (1) vertical (axial) stress and (2) horizontal (lateral) stress.

In mastication the teeth are exposed to a force which is the physical resultant of vertical and horizontal stress; whether the vertical or the horizontal component prevails depends entirely upon the condition present in the individual mouth and upon different masticatory movements. Some teeth are subject to an almost pure vertical or pure horizontal stress.

1. Vertical (Axial) Stress.—Pure vertical stress is exerted only upon teeth with completely flat-worn, occlusal surfaces when the mouth is closed directly in centric relation.

In this case the tooth is pressed into the socket parallel to the long axis. The full amount of force is taken over by the oblique fibers. All of these fibers, as well as almost all of the other fiber groups, are stretched at the same time. Inasmuch as a great deal of resistance is offered to this type of stress, the tooth will not yield except to great pressure and then only to a small extent (Fig. 276). The only fibers in a state of relaxation are the vertical apical fibers and perhaps some of the alveolar crest fibers.

FIG. 276.—Distribution of force over the different groups of fibers in case of vertical (axial) occlusal stress. Lower bicuspid, with considerable occlusal wear. The arrow indicates the direction of force. All the oblique fibers are under uniform strain; only the alveolar crest fibers and some of the apical fibers are relaxed.

2. Horizontal (Lateral) Stress.—Practically every stress exerted upon a tooth results in at least a certain amount of horizontal force and causes a tipping of the tooth. In all young intact teeth, the interlocking of the cusps in occlusion will result in a "sliding-in" along an oblique plane, and create a horizontal force. The same is true for every lateral (grinding) movement of the mandible

Pure horizontal stress exerted upon the crown of a tooth is seldom found. A certain, although perhaps very slight, vertical force is always present. The closest approach to pure horizontal stress can be found in cases of deep overbite in which the anterior teeth overlap entirely, or occasionally during orthodontic movement.

ANALYSIS OF THE TIPPING MOVEMENT OF TEETH. POSITION OF THE FULCRUM.

In order to understand the participation of the various groups of periodontal fibers in case of horizontal stress, it will be necessary to discuss first the mechanics of tipping movement. To simplify the explanation, a single-rooted tooth with a straight, slightly tapering root will be considered. Case, who studied the question from the orthodontic viewpoint, compared a tooth, the crown of which is exposed to lateral (horizontal) stress, with a post in soft homogeneous soil. He describes the following experiment (Fig. 277).

"If you should drive a 4-foot post one-half its length into clayey soil of uniform quality, and then take hold of the top of the post and move it back and forth with a view of subsequently pulling it out of the ground, you would be working a lever which combines the qualities of the first and second kinds, or one like the oar in which the so-called areas of fulcrum and weight act as fulcrums to the other. After pulling the post out of the ground, if it were possible for you to make a transverse section of the soil for the purpose of examining the shape of the hole you had made, you would find it somewhat the shape of an hour-glass; the upper portion of the opening being about twice as large as the lower." From Case's illustration it becomes evident that the upper part of the "root" of the post moves in the same direction as the "crown" part of the post; whereas, the lower portion moves in the opposite direction. The fulcrum being located below the middle of the "root," the excursion of the "apex" is less extensive than the excursion of the upper part of the root. Case states that a tooth moves in the same manner.

Schwarz studied the question of the fulcrum experimentally and confirmed Case's assertion. He showed that under the influence of horizontal stress upon a dog's incisor, the following position was occupied by the tooth: The tooth came close to the alveolar bone at the margin of the alveolus. The apex moved in a direction opposite to that of the crown and also opposite to that of the upper

part of the root; however, the excursion of the apex was less than the excursion of the upper part of the root (Fig. 278). Therefore, Schwarz concluded that the fulcrum was located at X, at the border between the middle and apical third of the intra-alveolar part of the root. Further proofs, gained from animal experiments as well as from human jaw material, confirmed the opinion that this location of

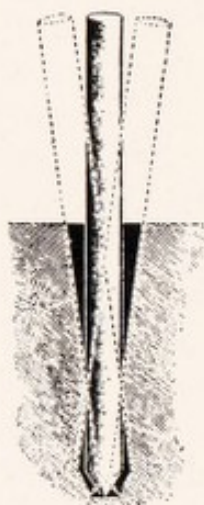


FIG. 277.

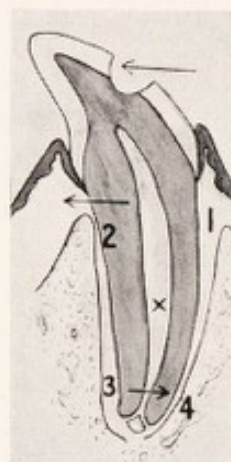


FIG. 278.

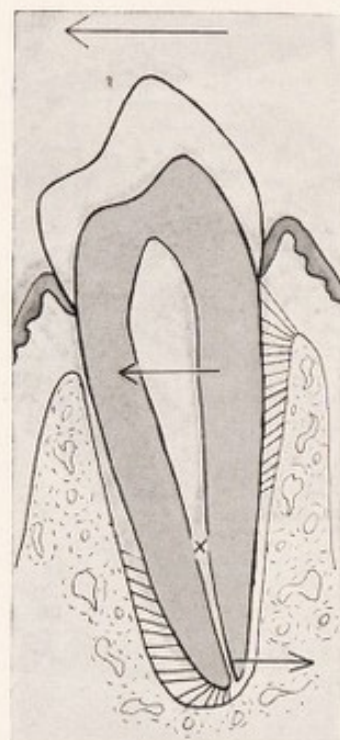


FIG. 279.

FIG. 277.—Case's post lever experiment: If a post sticking in a homogeneous clayey soil is moved back and forth, the fulcrum is located below the middle of the embedded part of the post. The opening in the ground, therefore, assumes the shape of an hour-glass. (Case, Dental Orthopedia.)

FIG. 278.—Schematic drawing of a labio-lingual section through a lower incisor of a dog. Shortly before death the tooth had been ligated to an arch wire pulling the crown toward the lingual side. The arrow indicates the direction of this force. A notch in the tooth was used to hold the ligature in place on the crown surface. Notice the distribution of the width of the periodontal space around the tooth: 1, widening of the periodontal space at the alveolar margin on the labial side (side of pull); 2, narrow periodontal space at the alveolar margin on the lingual side (side of pressure); 3, widening of the periodontal space at the apex on the lingual side (side of pull); 4, narrowing of the periodontal space at the apex on the labial side (side of pressure). As the apex moves in a direction opposite to the upper part of the root, the fulcrum must be located near the middle of the root, at X. (Drawn after Schwarz, *Ztschr. f. Stomatol.*)

FIG. 279.—Distribution of force over the different groups of fibers in case of horizontal (lateral) occlusal stress. Bucco-lingual section through a lower bicuspid. The arrow indicates the direction of force. The crown is displaced toward the lingual (left) side, whereas the apex moves in the opposite direction. The fulcrum of the tipped tooth is located about in the middle of the root at X. The stretched fiber groups only are indicated by heavy lines; the relaxed fibers are not included in the diagram. About one-half of the oblique fibers on either surface of the root is exposed to a heavy strain, the other half is relaxed or compressed. (Compare with Fig. 276.)

the fulcrum was the rule in a tipping movement. In teeth with more than one root, the fulcrum will be located between the roots in the bone of the interradicular septum (see Fig. 288).

One reason for the minor excursion of the apex compared with the excursion of the root at the margin of the alveolus seems to be the presence of the apical fibers that stabilize the apex against lateral displacement. Johnson, Appleton, and Rittershofer showed that in very young teeth, with not yet fully developed apices, the excursions of the root ends were very noticeable as no fibers were present to hinder apical displacement; in these teeth the fulcrum evidently was further crownward, at about the middle of the root. On the other hand, Oppenheim found that in case of delicate lateral stress as applied in careful orthodontic measures on teeth with fully developed root ends, the apex showed hardly any displacement; the amount of excursion increased steadily from the apex crownward. Only the coronal part of the root was displaced, whereby the fulcrum practically coincided with the apex due to the presence of well-developed apical fibers (see Chapter XIV).

Another form of tipping mechanism, the existence of which has been claimed by some authors (Häupl, Lang), is a movement by which the fulcrum would be at the alveolar margin, the amount of excursion steadily increasing toward the apex. This type of tipping movement, however, has never been found to exist in any case examined up to this time.

The various groups of periodontal fibers participate in a tipping movement as follows: About one-half of all the fibers is stretched, the other half is relaxed. The areas of tension and of relaxation are located diagonally opposite each other (Fig. 279). In comparing this figure with Fig. 276 (vertical stress), we can readily understand the clinical observation that teeth stand vertical stress better than horizontal stress: in the case of vertical stress the force is distributed evenly over almost all fibers; in case of horizontal stress, one-half of the fibers is stretched to the limit, the other half is completely relaxed or even compressed.

WIDTH OF THE PERIODONTAL MEMBRANE.

The functional condition of the teeth has a decided influence upon the width of the periodontal membrane. The following observation has been made: the less function, the narrower the periodontal membrane; the more function, the wider the periodontal membrane. Measurements of the width of the periodontal membrane were reported by Klein, Kellner, and Kronfeld. The smallest measurements are obtained from impacted teeth; slightly larger figures, from erupted teeth without antagonists. Considerably larger

measurements were obtained from teeth standing in line with other teeth and having antagonists. The widest periodontal membranes were observed about single teeth that carried a great functional load. The following table gives some of the figures obtained by measuring the width of the periodontal membrane of human permanent teeth under various functional conditions.

WIDTH OF PERIODONTAL MEMBRANE. (In mm.)

Kind of tooth.	Width of periodontal membrane at margin of alveolus.	Width of periodontal membrane at middle of alveolus.	Width of periodontal membrane at fundus of alveolus.	Average width of periodontal membrane.
Impacted lower 2d bicuspid	0.09	0.05	0.05	0.06
Upper 3d molar without antagonist	0.10	0.06	0.06	0.09
Upper cuspid without antagonist	0.18	0.13	0.16	0.16
Upper lateral incisor in normal occlusion	0.28	0.19	0.24	0.27
Upper 2d bicuspid; heavy functional stress	0.35	0.28	0.30	0.31

From this table it is evident that the width of the periodontal membrane is subject to great variations; it is from three to four times wider in functioning teeth than in impacted ones. It seems appropriate, therefore, to differentiate between "biological width of the periodontal space" and "physiological width of the periodontal space" (Gottlieb). The biological periodontal width is found in unerupted and impacted teeth; it represents the minimum distance existing between alveolar bone and cementum surface which has not been increased by function. In figures, the biological width varies from 0.06 to 0.10 mm.; this width is rather uniform all around the root.

The physiological periodontal width is found in teeth that are functioning. As a rule, the physiological width is from two to three times the biological width. This depends, of course, upon the type and amount of functional stress exerted upon the tooth. As an average, a physiological width of between 0.20 and 0.25 mm. is found. Whereas, in unerupted and impacted teeth, the periodontal membrane is uniformly wide around and along the entire root, in functioning teeth the membrane exhibits different widths at different levels and may be thicker on one side of the root than on the other.

As indicated in the table, the greatest width of the periodontal

membrane in a functioning tooth is observed at the margin of the alveolus, the smallest width at about the middle of the root or slightly apically, and a somewhat larger width at the apex. This distribution of the periodontal width again corroborates the statement about the location of the fulcrum in a tipping tooth. Since the fulcrum is located slightly rootward from the middle of the root, the root makes its greatest excursions at the alveolar margin. At the middle of the root, near the fulcrum, an area of relative rest is found; this area makes the least excursions and has the smallest periodontal space. Toward the apex the excursions become wider again; however, as the position of the fulcrum is nearer the apex, the excursions are less extensive, and the width of the periodontal soft tissue is smaller than at the margin of the alveolus.

By comparing teeth that have been subject to different functional conditions it has been possible to demonstrate that the distribution of the periodontal membrane around a root is the result of function. The larger the vertical component of stress, the more uniform will be the periodontal width from neck to apex of the root; the larger the horizontal component, the more will the figures obtained at the margin and at the fundus of the alveolus differ from the figures obtained at the middle of the alveolus. For example, the following figures were obtained from two teeth of the same jaw by measuring the periodontal width (Kronfeld):

I: Right lower central incisor. Direction of force, almost vertical (end-to-end occlusion):

Width of periodontal
membrane at margin
of alveolus.
0.18

Width of periodontal
membrane at middle
of alveolus.
0.16

Width of periodontal
membrane at fundus
of alveolus.
0.17

II: Left lower central incisor. Direction of force, almost horizontal (deep overbite):

Width of periodontal
membrane at margin
of alveolus.
0.26

Width of periodontal
membrane at middle
of alveolus.
0.10

Width of periodontal
membrane at fundus
of alveolus.
0.24

From this comparison the relation between direction of force, excursion of the root, and width of the periodontal membrane are obvious. Measurements of this type can be made only on central sections, that is, on sections that run through the root canal parallel to the long axis of the tooth. If other than central sections are used, the measurements are worthless and misleading.

Age influences the width of the periodontal membrane. In older individuals the measurements are slightly wider than in young persons. Klein found an average periodontal width of 0.23 mm. in individuals between twenty and twenty-five years of age, and an average width of 0.25 mm. in individuals between forty and fifty years of age.

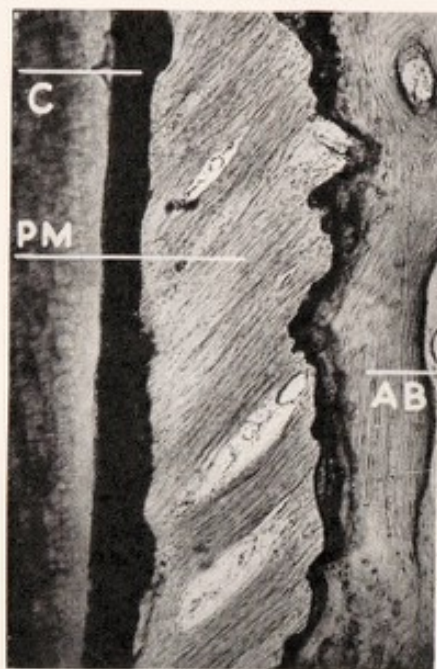


FIG. 280.

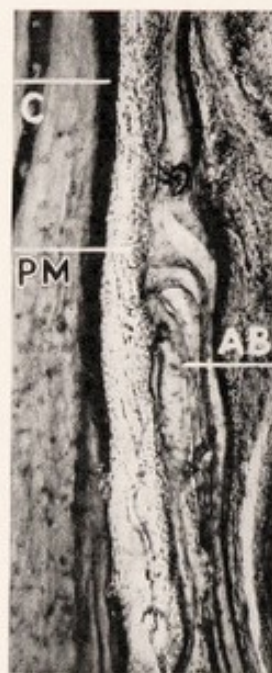


FIG. 281.

FIGS. 280 and 281.—Comparison between periodontal membrane in functioning and non-functioning tooth in the same human jaw.

FIG. 280.—Upper second bicuspid exposed to intensive occlusal stress in a vertical direction. C, cementum; PM, periodontal membrane with strong fiber bundles (oblique fibers); AB, alveolar bone. Width of periodontal membrane 0.30 to 0.36 mm.

FIG. 281.—Upper second molar without antagonists. C, cementum; PM, periodontal membrane consisting of loose fibers without definite arrangement; AB, alveolar bone. Width of periodontal membrane: 0.09 to 0.12 mm.

COMPARISON OF THE STRUCTURE OF THE PERIODONTAL MEMBRANE IN FUNCTIONING AND NON-FUNCTIONING TEETH.

The periodontal membrane of a tooth subjected to normal occlusal stress is characterized by the presence of strong, well-developed fiber bundles. Especially the oblique fibers show a very dense, regular arrangement parallel to each other, leaving only narrow spaces for the vessels and nerves between them (Fig. 280). In a non-functioning tooth the fibers are so scarce that few sharply defined bundles can be found; only a few strands run through the periodontal space in different directions, sometimes parallel to the root surface (Fig. 281).

In impacted teeth the periodontal membrane is sometimes replaced by fat tissue (see Fig. 330). The same change is also occasionally found in erupted teeth that have been out of occlusion for a long time. Fig. 282 shows part of the root surface of an upper second molar from an old man whose mandible had been edentulous for several decades. In the lower portion of this area fibrous periodontal tissue is still present; the arrangement of the fibers is identical to that seen in Fig. 281. The periodontal space is very narrow. At *FT*, the space between bone and cementum is occupied by fat tissue; the distance between bone and root is wide and irregular. Evidently in this part the periodontal membrane has entirely lost its biological and structural qualities; there is no definite relation between tooth and bone; the tooth surface has simply been included in the system of bone and marrow spaces, and the former alveolar bone runs through the fat marrow in the same manner as the trabeculae of cancellous bone run through the jaw.

COMPARISON OF CEMENTUM IN FUNCTIONING AND NON-FUNCTIONING TEETH.

In connection with periodontal width measurements, it is interesting to compare the thickness of the cementum of corresponding functioning and non-functioning teeth in the same jaw. Such investigations were published by Kellner who found that although the thickness of the cementum is subject to much greater individual variation than the width of the periodontal membrane, a constant ratio was present. The thickness of the cementum, if measured in corresponding teeth of the same jaw, was greater in non-functioning teeth than in functioning ones; the ratio was about 3 to 2. Thus the formation of cementum appears to be in reverse ratio to the amount of functional stress; heavy function seems to a certain extent to inhibit cementum deposition; lack of function seems to stimulate its formation.

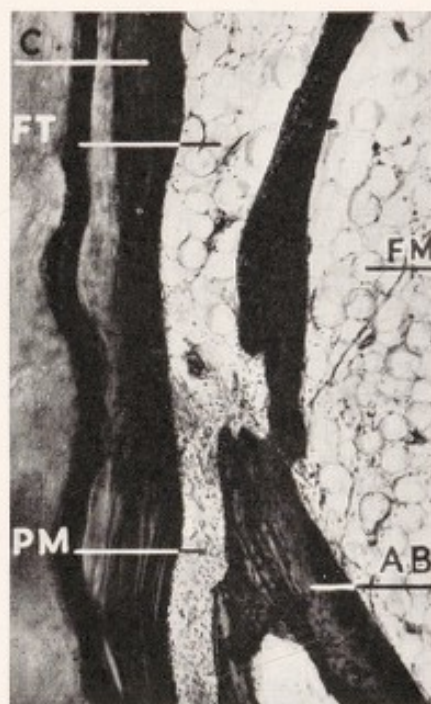


FIG. 282.—Fat tissue in the periodontal space. Upper molar without antagonist. The transformation of the fibrous periodontal membrane into fat tissue must be considered as an expression of inactivity. C, cementum; FT, fat tissue between alveolar bone and root surface; PM, narrow periodontal membrane with poorly developed fibers; AB, alveolar bone; FM, fat marrow. Width of periodontal membrane: 0.09 mm.

The fact that non-functioning teeth (teeth without antagonists and impacted teeth) have thicker cementum than functioning teeth of the same jaw has been known for a long time. The author has called attention to the presence of heavy layers of cementum in the bifurcations of first molars before eruption as well as on the roots of impacted cuspids. In both cases no outer stimuli could have been responsible for the deposition of cementum. Teeth without antagonists, impacted and erupting teeth, seem to have an

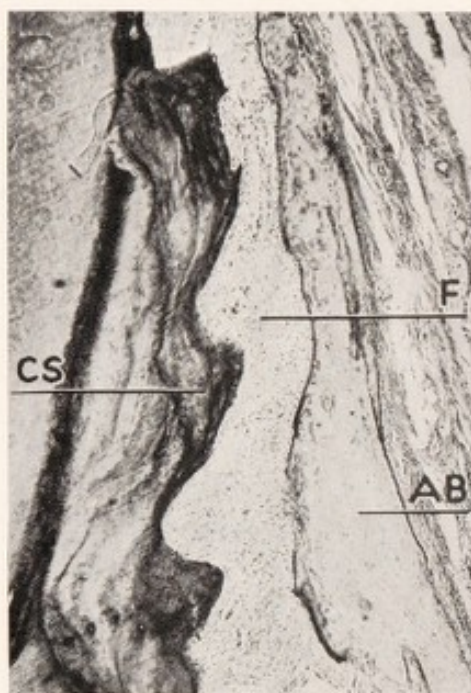


FIG. 283.—Spicules of cementum, CS, at the apex of a functioning tooth. F, fiber bundles of the periodontal membrane; AB, alveolar bone.

inherent tendency to move occlusally. Occlusal displacement causes widening of the periodontal space, especially around the apices and in the bifurcations of multirooted teeth; the increased distance between root surfaces and bone is then reduced by compensatory deposits of cementum at the root ends and in the bifurcations.

The arrangement and distribution of the cementum varies on functioning and non-functioning teeth. On impacted teeth the cementum is frequently arranged evenly all around the root surface, thereby transforming the root into a roundish, smooth body of cementum. On functioning teeth, however, a definite functional arrangement of the cementum is sometimes

observed: spicules and circumscribed cementum hyperplasias are formed as the result of calcification of the point of attachment of Sharpey's fibers (Fig. 283). Such formations are most often found in the apical part of the root; they may be compared with the calcified attachments of tendons to bones in other parts of the body (see Fig. 184).

INFLUENCE OF FUNCTION UPON THE ALVEOLAR BONE.

The alveolar bone is subject, under physiological conditions, to a constant tearing-down and building-up (resorption and new formation). Resorption is stimulated by the presence of old por-

tions of bone that have reached the limit of their life. This process of tissue aging is called senescence; it is found, with few exceptions, all over the organism (elimination of old cells and replacement by new cells). The eliminated portions of bone are normally replaced by newly built bone. New formation is largely controlled by functional factors. Under constant functional conditions the total mass of bone will remain unchanged, the amount of bone that is resorbed being immediately replaced by new bone, the formation of which is necessitated by the prevailing functional demand; thus, tearing down and building up are balanced.

This balance can be disturbed in two ways, either by an increase or a decrease in function. Increased function means increased mechanical stimulus and increased activity of the osteogenic cells; consequently, the physiological resorptive loss will be overcompensated by a surplus of new formation. We might say that for each resorbed unit of bone two new units are rebuilt; the result will be an increase in the total mass of bone. This increase is called functional hypertrophy. Functional hypertrophy is frequently observed in bones under certain functional conditions. A typical example is the increase in weight and thickness of the bones induced by continued heavy physical labor.

If, on the other hand, function is decreased, the resorption will go on as usual; but the resorbed portions of bone will be no longer replaced by an equal amount of new bone since adequate functional demand is lacking. We might say that for each two units of bone resorbed, only one unit is newly built. This, of course, leads to a reduction in the total mass of bone, which is called bone atrophy (disuse atrophy, atrophy of inactivity).

Such changes are found, for example, in the bones of the extremities if a limb has been paralyzed or otherwise put out of function for a long time: resorption continues, but there is no functional stimulus to cause new formation of bone and the result is disuse atrophy of the bone.

In studying functional changes in the alveolar bone it is necessary to distinguish between "alveolar bone" in the strict sense, and "supporting bone" (Orban). Alveolar bone consists of the outer and inner plate of the alveolus (outer surface of alveolus and wall of socket). The space between these two plates is occupied by the supporting bone, which consists of a network of trabeculae connecting the plates of alveolar bone (Fig. 284). It has been found that under changing functional conditions the alveolar bone remains

rather invariable, whereas the supporting bone changes considerably. The greater the functional stress the stronger and denser will be the supporting bone. In case of lack of function the supporting bone will be almost entirely missing; only the thin outer and inner alveolar plates will be left (Figs. 285 and 286).

In a broad sense the entire mandible and maxilla belong to the supporting bone. Both jaws will undergo a considerable amount of change if certain functional conditions exist over a long period of time (see Figs. 291 and 292).

The difference between the structure and density of the alveolar bone around teeth under different functional conditions can some-

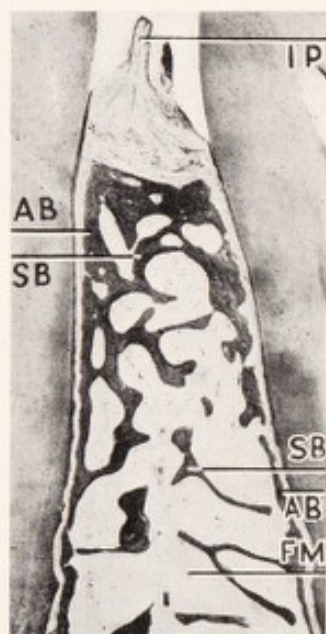


FIG. 284.

FIG. 284.—Alveolar bone, AB, and supporting bone, SB, between two lower molars. FM, fat marrow; IP, interdental papilla.



FIG. 285.

FIGS. 285 and 286.—Comparison of the alveolar bone of upper molars under different functional conditions.

FIG. 285.—Upper second and third molars in occlusion. Well-developed alveolar bone and supporting bone.



FIG. 286

FIG. 286.—Upper third molar without antagonist. Thin alveolar bone. The supporting bone has almost entirely disappeared and is replaced by fat marrow.

times be visualized by comparing radiographs of such teeth. In jaws where a number of teeth have been lost by extractions, the radiograph will sometimes reveal a thickening and condensation of bone around the teeth that are still in occlusion (functional hypertrophy of bone). Around the teeth that have lost their antagonists, the bone will appear much thinner; the structure of the supporting bone will sometimes be barely visible (MacMillan).

In jaws where all teeth are present and in normal occlusion, the same density of bone is usually found around all the teeth.

EXPERIMENTAL PRODUCTION OF VARIOUS FUNCTIONAL CONDITIONS IN THE TEETH OF ANIMALS.

In animal experiments it is possible to create certain abnormal functional conditions in the teeth. This can be done either by decreasing or increasing the normal functional stress. By grinding or eliminating one of a pair of antagonistic teeth, the function can be decreased; by cementing crowns or caps that interfere with normal occlusion the occlusal stress can be greatly increased.

By extracting the molars of young rats on one side in either the lower or the upper jaw, Preissecker was able to compare functioning and non-functioning teeth in the same animal. He found that the width of the periodontal membrane on teeth without antagonists is about one-half of that on teeth in occlusion, and thus he corroborated the measurements made on human teeth under similar functional conditions (see page 342).

The largest experiment of this type was made by Gottlieb, Orban and the author, who used dogs for their studies. The final results were published by Gottlieb and Orban in the form of a monograph. The technique was as follows: Metal caps were cemented upon upper and lower right first molars of dogs. Some caps had oblique occlusal planes, causing a horizontal (lateral) component of force and resulting in a tipping movement of the teeth. Other caps had horizontal occlusal planes, creating a vertical force and causing intrusion of the teeth into their sockets. In the majority of cases, due to the excessive force applied, traumatic injuries to the periodontal tissues were produced. These will be discussed in the next chapter; here merely those findings will be described that are of importance for the mechanism of tooth movement.

An upper incisor was ligated to a labial arch wire. After forty-eight hours the crown had been displaced labially, the apex lingually; the alternating distribution of periodontal width at the alveolar margin and at the apex can be plainly seen (Fig. 287).

A bucco-lingual section through an upper first molar illustrates the tipping movement of a multirooted tooth (Fig. 288). As a result of lateral stress of thirty-six hours' duration, the crown moved labially and the apices of the mesio-buccal and the mesio-lingual roots were displaced toward the lingual side. The fulcrum was located in the interradicular bone septum.

If great stress is applied over a long period of time, the supporting bone and, finally, the configuration of the entire jaw is changed.

Figs. 289 and 290 show corresponding sections through the right and left upper first molars of a dog; a metal cap was worn on the right upper first molar for thirteen months, the left side being used as a control. The greater number and increased thickness of the trabeculae of the supporting maxillary bone on the right side can

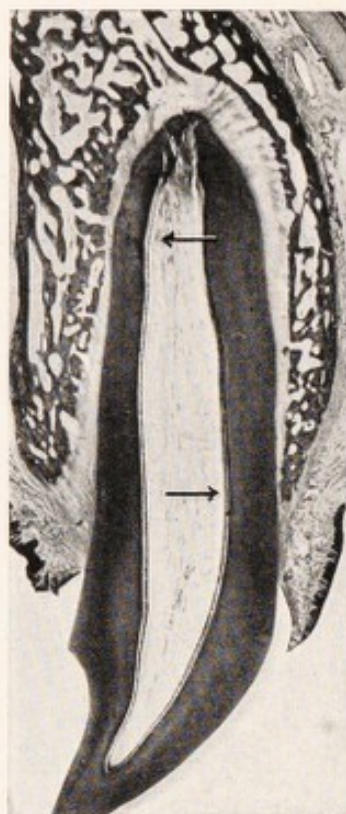


FIG. 287.

FIG. 287.—Upper incisor of dog, ligated for forty-eight hours toward the labial side. The lower arrow indicates the direction of force exerted upon the crown. The apex shows a deviation in the opposite direction. Notice the widening of the periodontal membrane in the areas of pull and the narrowing in the areas of pressure. The fulcrum is located slightly apically from the middle of the root. See also Fig. 278. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)



FIG. 288.

FIG. 288.—Upper molar of dog that carried metal caps with oblique planes for thirty-six hours. The lower arrow indicates the direction of movement of the crown. The apices have moved in the opposite direction. The width of the periodontal membrane is distributed accordingly. The fulcrum is located in the interradicular bone septum. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

be plainly seen. A similar change took place in the mandible of a young dog who wore caps with oblique planes on the right first molars over a period of two months and ten days (Figs. 291 and 292). The section runs in bucco-lingual direction through the interradicular septum; as a result no roots are visible. The architecture of the entire mandible was changed so that the body of

the mandible is considerably lower and thicker than on the control side. The right molar is depressed. It looks as if the right side of the lower jaw had been flattened out by a heavy weight. This change in the configuration of the body of the mandible is the result of a gradual process of rearrangement and reconstruction of the mandible. Under the influence of increased occlusal pressure the bone at the bottom of the alveolus was resorbed, and the tooth was lowered; at the same time compensatory deposition of new

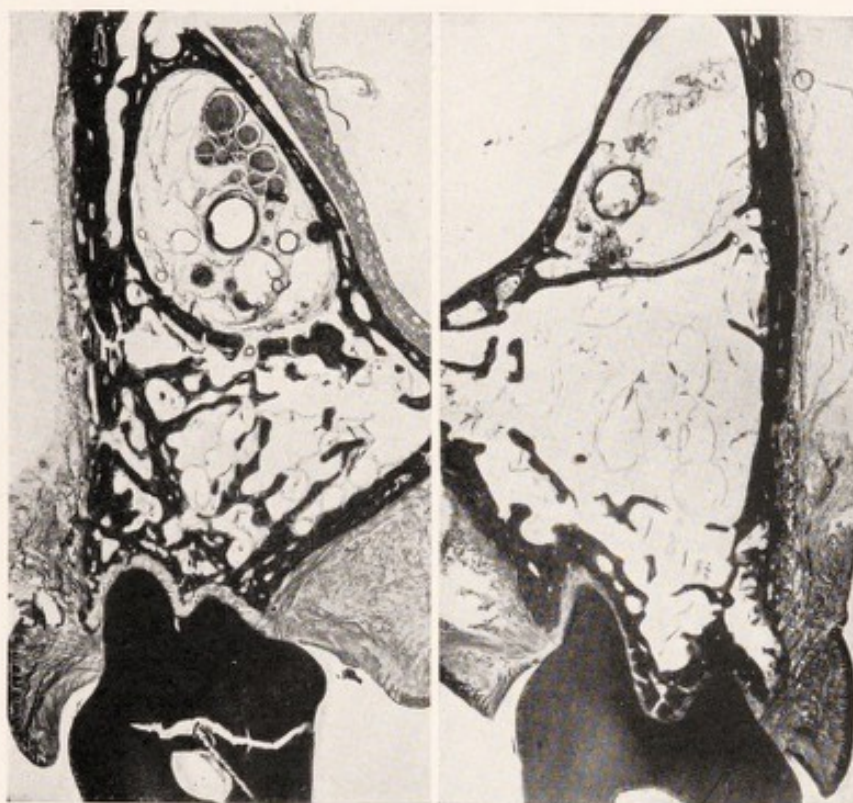


FIG. 289.

FIG. 290.

FIGS. 289 and 290.—Comparison of functioning and non-functioning molars of the same dog. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

FIG. 289.—Right upper molar that carried a metal cap for thirteen months. Section through the interradiacal septum. Notice the well-developed supporting bone.

FIG. 290.—Left upper molar. Poorly developed supporting bone; large areas of fat marrow.

bone took place on the outer surface of the mandible, finally bringing about the condition seen in Figs. 291 and 292.

From the histological study of both human and animal specimens, the following conception can be gained concerning the changes in the periodontal membrane in the different phases of life: before eruption the periodontal space around the root is narrow (biological width); few fiber bundles are present and the surrounding bone does not show the dense arrangement of supporting bone. This

condition may persist for a lifetime if the tooth fails to erupt and remains impacted in the jaw. As the tooth erupts and reaches its normal occlusion, the periodontal space becomes wider (physiological width); well-defined fiber bundles develop between root and bone; the supporting bone becomes reinforced so as to withstand functional stresses. Widening of the periodontal space, (transformation of biological width into physiological width) takes place as a result of bone resorption on the inner plate of the alveolus under the

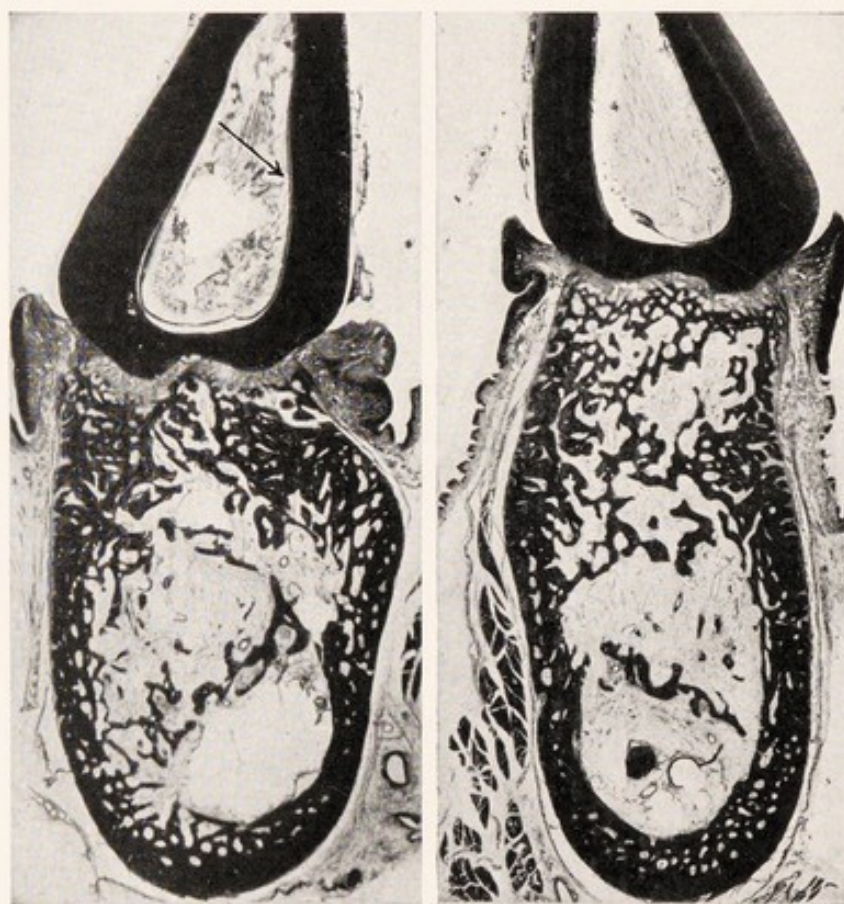


FIG. 291.

FIG. 292.

FIGS. 291 and 292.—Comparison of the right and left side of the mandible of a dog. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

FIG. 291.—Right side. Section through the interradicular septum of first molar which wore a metal cap for two months and ten days.

FIG. 292.—Left side, corresponding section. The mandible is lower and thicker on the side of increased function. The supporting bone has become reinforced.

influence of functional stimuli. Thus, the space between root and bone is gradually widened until the physiological width is reached.

If, on the other hand, a formerly functioning tooth is put out of occlusion, either by destruction of its own crown or by loss of antagonistic teeth, the process is reversed. The periodontal membrane gradually loses its strong, fibrous structure. Its width is reduced

by new formation of both cementum and bone until it may finally reach the biological width present before the tooth erupted.

The physiological width seems to be sufficient to allow the tooth all lateral excursions within physiological range; for instance, by using a metal separator it is possible to separate two healthy upper central incisors approximately 0.5 mm. (twice the thickness of the periodontal membrane on the distal side of each tooth) without pain to the patient. If the separation is continued beyond this distance, the procedure becomes painful, indicating that the biological limit of the tensile strength and compressibility of the periodontal fibers has been reached.

BIBLIOGRAPHY.

- BOEDECKER, C. F.: Critical Review of Gottlieb and Orban's "Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne," *Internat. Jour. Ortho. Oral Surg. and Radiogr.*, 1932, **18**, 895.
- CASE, C. S.: *Dental Orthopedia*, Chicago, C. S. Case, 1921, p. 99.
- GOTTLIEB, B.: Paradentalpyorrhoe und Alveolaratrophie, *Fortachr. d. Zahnheilk.*, 1929, **5**, 351.
- GOTTLIEB, B., and ORBAN, B.: Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne, Leipzig, Thieme, 1931.
- Die Gewebsveränderungen bei Überbelastungen mit besonderer Berücksichtigung von Alter und Konstitution, *Ztschr. f. Stom.*, 1931, **29**, 370.
- HÄUPL, C.: Zur Pathogenese der Paradentitis Profunda, *Vrtljschr. f. Zhk.*, 1926, **42**, 397, 544.
- HÄUPL, C., and LANG, F. J.: *Marginale Paradentitis*, Berlin, Meusser, 1927.
- HÄUPL, K.: Gewebsveränderungen in Paradentien von Brückenpfeilern, *Vrtljschr. f. Zhk.*, 1929, **45**, 145.
- KELLNER, E.: Histologische Befunde an antagonistischen Zähnen, *Ztschr. f. Stom.*, 1928, **26**, 271.
- Das Verhältnis der Zement- und Periodontalbreiten zur funktionellen Beanspruchung der Zähne, *Ztschr. f. Stom.*, 1931, **29**, 44.
- KLEIN, A.: Systematische Untersuchungen über die Periodontalbreite, *Ztschr. f. Stom.*, 1928, **26**, 418.
- KRONFELD, RUDOLF: Histologic Study of the Influence of Function on the Human Periodontal Membrane, *Jour. Am. Dent. Assn.*, 1931, **18**, 1242.
- MACMILLAN, HUGH W.: The Clinical Significance of Disuse Atrophy of the Alveolar Process, *Jour. Am. Dent. Assn.*, 1927, **14**, 697.
- Radiographic and Histologic Evidence of the Functional Adaptation of the Alveolar Process, *Jour. Am. Dent. Assn.*, 1928, **15**, 316.
- Nonuse in the Development and Resistance of the Alveolar Process, *Jour. Am. Dent. Assn.*, 1928, **15**, 511.
- MEYER, W.: *Periodontium*, *Handw. d. ges. Zhk.*, vol. **3**, p. 2047.
- ORBAN, B.: *Dental Histology and Embryology*, Philadelphia, P. Blakiston's Son & Co., 1929, 2d ed.
- PREISSECKER, OTTO: Beeinflussung des Periodontiums durch experimentelle Entlastung, *Ztschr. f. Stom.*, 1931, **29**, 442.
- SCHWARZ, A. M.: Über die Bewegung belasteter Zähne, *Ztschr. f. Stom.*, 1928, **26**, 40.
- Ein weiterer Beitrag zur Frage der Bewegung belasteter Zähne, *Ztschr. f. Stom.*, 1929, **27**, 499.
- Movement of Teeth under Traumatic Stress, *Dent. Items Int.*, 1930, **52**, 96.

CHAPTER XIII.

PATHOLOGICAL CHANGES DUE TO EXCESSIVE FUNCTIONAL STRESS.

CLINICAL CONSIDERATIONS CONCERNING INCREASED OCCLUSAL STRESS.

For a long time the attention of dental practitioners and investigators has been directed upon the possibility of injuries due to occlusal stress. Karoly, of Vienna, was an early observer who expressed the opinion that increased function may cause loosening and loss of teeth.

In this country Stillman and McCall discussed this problem. They created the term "traumatic occlusion," which they defined as abnormal occlusal stress capable of producing or having produced an injury to the periodontium. These authors point out that abnormal stress does not invariably cause injury. When one or several teeth are subject to excessive stress, one of two things will result: Either the supporting structures will become reinforced, thereby making the teeth able to withstand the increased functional demand, or the supporting tissues will be damaged and will break down. Stillman and McCall emphasize the importance of the individual tissue reaction. A tooth subject to "plus-occlusion" may become strengthened or weakened, depending upon the ability of the supporting tissues to adjust themselves to altered occlusal conditions.

A clinical illustration may help to clarify this point. Frequently patients are observed who have lost a great number of teeth by destruction through caries. If no artificial restorations have been made in such mouths, we frequently find single teeth bearing almost the entire stress of the masticatory muscles; yet these teeth are clinically firm and solid; the health and strength of their periodontal tissues also are revealed by microscopic examination. If traumatic occlusion were a primary cause for loosening of teeth, the teeth mentioned above should be the first to become loose.

Another important clinical question in this connection is the problem of fixed bridge construction. To construct a bridge means to charge the roots of the abutments with the occlusal stress that was formerly exerted upon all the teeth that are now missing. It is assumed in practice that two strong, healthy teeth or roots

are able to carry the functional load of four or five teeth (their own load plus that of two or three pontics). That such bridges are carried successfully for long periods of time proves that a tooth is well able to adjust itself (by reinforcing periodontal membrane and bone) to a considerable increase in stress.

The importance of tissue reaction is evident in cases of bridges that, after ten or twenty years of good service, became useless because the abutments had loosened. Frequently no changes in the occlusion could be held responsible for the loosening. Thus it is evident that for ten or twenty years the tissues around the roots of the abutments were able to adjust themselves to the increased function; but as the patient became older, the resistance of the oral tissues diminished concurrently with the aging of all other tissues of the body, and, finally, since the roots of the abutments became unable to stand the same amount of stress that they stood earlier in life, the bridge became loose.

It has been impossible to loosen teeth in experimental animals by increasing the stress. Although considerable damage was done to the periodontal tissues in many of these cases, no loosening or loss of teeth was brought about. The pathological changes found in these animals were similar to the changes found in human jaw material under similar occlusal conditions.

In the study of the periodontal membrane it is very difficult to draw a line between physiological and pathological conditions. Although it is usually apparent from a microscopic specimen whether the amount of function to which the tooth was subjected was within the physiological range, this same diagnosis is very hard to make in a clinical examination of the mouth. No doubt a great many of the minor traumatic injuries to which the periodontal membrane is subject during life are never registered clinically. Thus, all we can do is to describe the changes that we actually find in the tissues of human and animal jaws when teeth had been exposed to an amount of stress evidently beyond the physiological limit; but we are unable to define what the clinical significance of such changes would be or how they could be avoided in the mouths of patients.

From the arrangement of the dento-alveolar fibers described in the preceding chapter, it is evident that every pressure upon the crown will be taken up by various groups of fiber bundles; some fibers will be stretched, others will relax. A physiological force may be defined as one that does not stretch or compress the fibers beyond their physiological resistance. If the force is so great that the physiological resistance of the fibers has been surpassed, the

root surface will come close to or will touch the alveolar bone, and the periodontal membrane between root and bone will be damaged, the degree of damage varying from small hemorrhages to complete crushing. This pathological change is the result of a force greater than the natural resistance of the periodontal fibers, causing their compression and crushing between root and bone.

From this definition it is evident that we shall never be able to express in terms of pounds the limit of physiological masticatory force. The same amount of pressure that will be physiological and proper for one individual or one tooth will be pathological and cause damage to another.

However, several facts have been established from clinical as well as from experimental observation which help us to approach the question of pathological stress. One of these facts is that the teeth of young people, under otherwise the same outer conditions, will stand more stress than the teeth of older people. This fact is based upon clinical observations; it has its root in the biological law that the resistance of tissues is highest in youth and is gradually lowered with advancing age, and that slight injuries that are readily repaired in youth cause permanent damage in old age.

Another fact is that a tooth, under otherwise the same conditions, will stand vertical stress better than the same amount of lateral stress. The reason is, as described on page 338, that in vertical (axial) stress there is a more even distribution of force over all fibers than in horizontal stress.

The increased possibility of damage from lateral stress has been recognized for a long time from clinical observation. Karoly and many others advocated the grinding-off of the occlusal cusps as a prophylactic measure against loosening of the teeth. There has been a general tendency, too, in restorative dentistry not to construct high interlocking cusps which tend to create lateral stress in mastication. After all, the normal involution of teeth points in this direction. In well-used, normal jaws of people in advanced age, the cusps have practically disappeared and the occlusal surfaces are smooth, even planes in end-to-end occlusion. Automatically a transition takes place from the highly resistant young teeth with high cusps to the less resistant older teeth to which lateral stress might be injurious.

From this viewpoint the tendency of dentists to eliminate high cusps and lateral interlocking in older patients must be encouraged. In doing so the dentist performs what nature often fails to do among modern races, namely, to provide adequate occlusal wear.

TISSUE CHANGES UNDER THE INFLUENCE OF EXCESSIVE OCCLUSAL STRESS.

1. **Excessive Occlusal Stress Produced Experimentally in Animals.**— It has been stated that a force that exceeds the physiological resis-

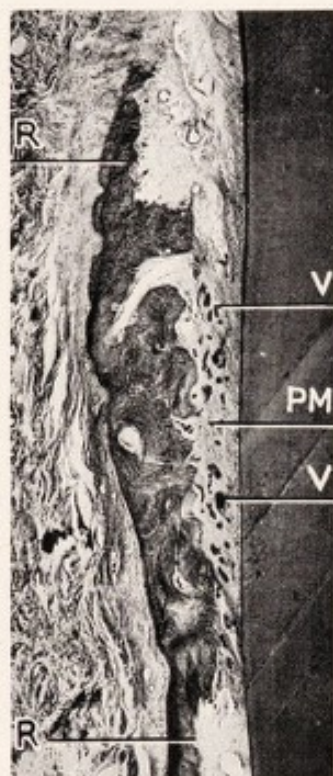


FIG. 293.

FIG. 293.—Compression of the periodontal membrane and thrombosis of the bloodvessels near the alveolar margin. Dog, upper molar. PM, compressed periodontal membrane; V, thrombosed periodontal bloodvessels; R, bone resorption due to pressure. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

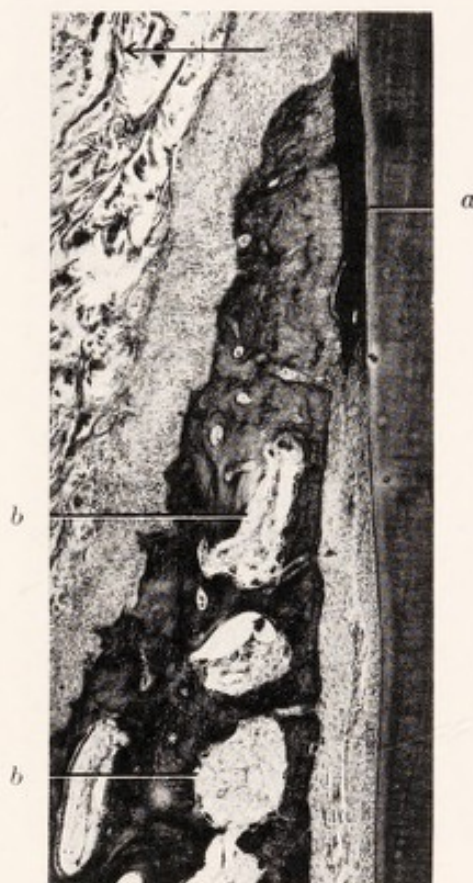


FIG. 294.

FIG. 294.—Crushing of the periodontal membrane at the alveolar margin of a dog's incisor. A strong orthodontic force had been applied by means of silk ligatures for forty-eight hours. The arrow indicates the direction in which the tooth was being moved. *a*, crushed and blood-stained periodontal membrane; *b*, osteoclasts causing bone resorption in the marrow spaces. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

tance of the periodontal fibers will cause injury to the periodontal soft tissues. This injury is followed, as a rule, by resorption of the bone and sometimes of the tooth also.

The following illustrations were taken from a publication by Gottlieb and Orban. They cemented metal caps on molars of dogs for varying lengths of time; then teeth and surrounding bone were

sectioned in the direction of the force. Due to the fact that the bite was opened by the caps, the entire masticatory force was loaded upon these teeth, and consequently rather extensive pathological changes were caused by the heavy stress applied.

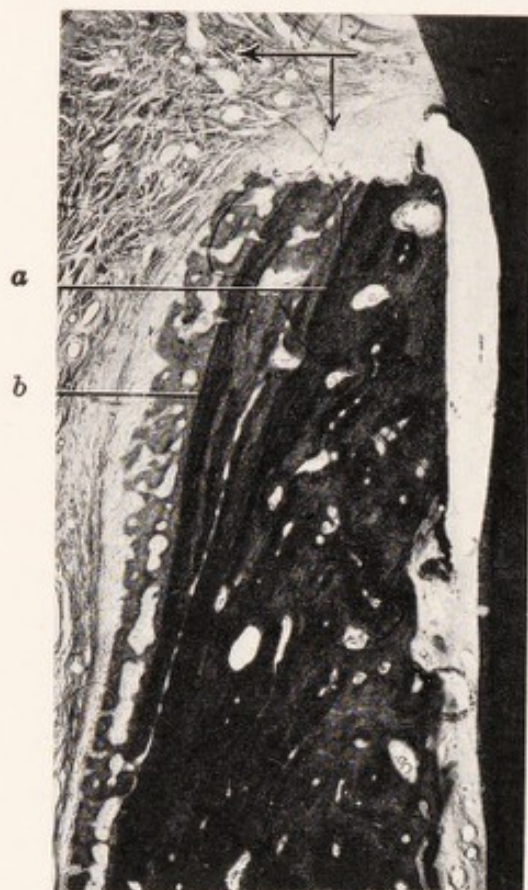


FIG. 295.

FIG. 295.—Destruction of the periodontal membrane of a lower molar near the alveolar margin. A metal cap had been worn on this tooth for five weeks. The arrows indicate that a vertical as well as a horizontal component of force had been active. Notice the bone resorption of the alveolar margin. Due to the lateral force a necrotic cleft resulting from the complete destruction of the periodontal soft tissues has developed between tooth and bone. *a*, *b*, compensatory formation of new bone on the outer surface of the alveolus. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

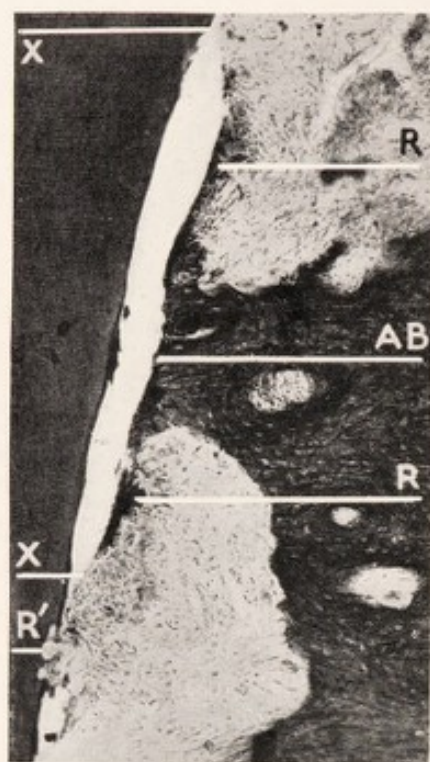


FIG. 296.

FIG. 296.—“Undermining” resorption of the bone that is in contact with the tooth surface. Upper molar, bifurcation. Caps worn for fifteen days. X-X, original extent of destruction of periodontal membrane; AB, alveolar bone still in contact with the tooth; R, areas in which undermining resorption has removed the bone; R', shallow resorption on the root surface. (From the experiment of Gottlieb, Orban and Kronfeld.)

On the side of the tooth sustaining lateral pressure, the periodontal membrane was injured to varying degrees. In many cases thrombosis of the periodontal bloodvessels was the first microscopic symptom that the limit of resistance of the fiber bundles had been exceeded (Fig. 293). The periodontal membrane around these

vessels shows a homogeneous structure; there is no staining of nuclei and other cell elements, which indicates that the vitality of the tissues had been lost. As a result of this tissue injury, no resorptive processes could occur in the damaged area. In order to resorb bone or cementum the periodontal soft tissue must be vital. If the soft tissue is badly damaged, no osteoclasts can be produced, and then the two hard substances, cementum and bone, are found opposite each other without any changes. This is even more true in those cases in which, due to a high amount of force, the periodontal soft tissue has been completely crushed and changed into a structureless, blood-stained mass (Fig. 294). If this condition is allowed to persist for a while, the dead tissue between cementum and bone is worn away entirely, leaving a joint-like capillary space between the two hard substances that grind on each other at every movement of the tooth (Fig. 295). It is easily understood that, under such circumstances, no further displacement of the tooth in the direction of the pressure is possible; the tooth simply leans against the alveolar bone, and no soft tissues are present that could start bone resorption.

After a necrotic space between bone and tooth has once developed, bone resorption starts further distant from the center of tissue injury. Osteoclasts are found in the marrow spaces of the alveolar process and in that part of the periodontal membrane in which normal cell structure is still preserved. From these points, undermining resorption toward the bone adjacent to the necrotic periodontal membrane occurs until this bone has been completely undermined and gives way for further movement of the tooth. Fig. 296 illustrates this process. After the cap was placed on the tooth, the periodontal membrane was crushed and destroyed from X to X; to this extent the bone must have been in immediate contact with the tooth. In the following two weeks two-thirds of the alveolar bone underneath the necrotic area were undermined and eliminated by resorption, leaving a contact between root and bone in only a small area. As resorption continues, the entire alveolar bone in this area is eliminated, allowing the tooth further movement in the direction of the force.

In all teeth illustrated in Figs. 293 to 296 new formation of bone was found on the side of pull, the bone being arranged in spicules parallel to the stretched fiber bundles of the periodontal membrane.

2. Changes in Human Periodontal Tissue Under the Influence of Excessive Occlusal Stress.—Although a large number of animal experiments has established certain changes as typical under given

conditions, there might be some doubt as to the applicability of these findings to man but for the fact that histological investigations on human jaws with teeth that were exposed to an undue amount of stress showed exactly the same type of pathological changes. Such findings were reported by Bauer, Häupl, Orban, and Grohs. Fig. 297 is taken from Orban's publication. It shows an area of necrotic, blood-stained periodontal tissue between the inner alveolar plate of bone and the mesial side of the root of a lower second



FIG. 297.—Compressed blood-stained periodontal membrane on the mesial root surface of a human lower molar that was tipped mesially. D, dentin; C, cementum; PM, compressed periodontal membrane; AB, alveolar bone; NB, compensatory new formation of bone on the side of the bone away from the tooth. (Orban, Jour. Am. Dent. Assn.)

molar. The tooth had been tipped mesially following the loss of the first molar. The excessive tipping force resulted here in a crushing of the periodontal membrane on the side of pressure. A similar but more extensive destruction of the periodontal soft tissues is seen in Fig. 298, which was taken of the bifurcation of another lower molar in the same jaw. A necrotic space was formed between bone and root surface, which was accompanied by resorption of both bone and tooth and by a reparative new formation of bone in

areas more distant from the point of destruction. The dark masses in the periphery of the necrotic space are remnants of old hemorrhages. The bone adjacent to these areas was undermined by resorption (Fig. 299); finally the entire bone that originally bordered the crushed periodontal membrane is eliminated, and the tooth is again able to move in the direction of force. In Fig. 300 the final outcome of undermining resorption is illustrated: the periodontal space is very wide and is occupied by tissue debris and old coagulated blood; a large number of giant cells are working on the final elimination of these tissue rests. At the same time resorption is being continued on the bone lining the newly established periodontal space.

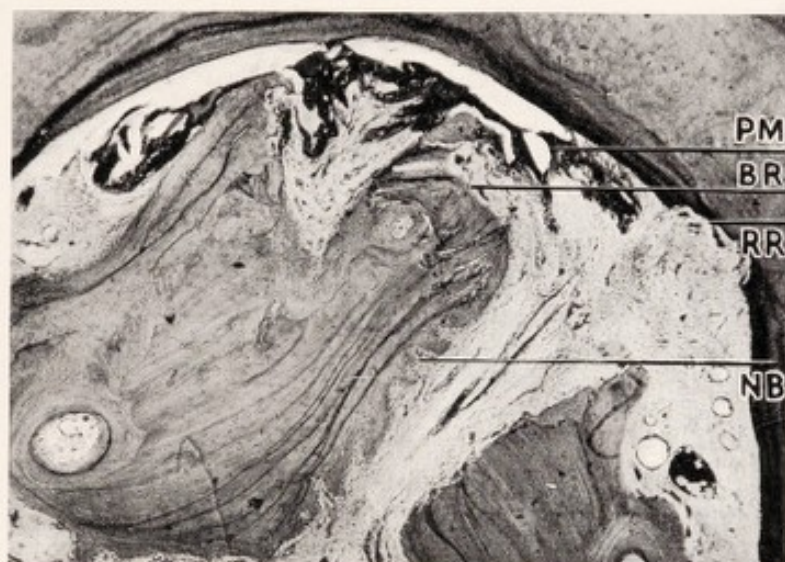


FIG. 298.—Crushing of periodontal membrane between root surface and bone. Human lower molar, bifurcation. PM, crushed and blood-stained periodontal membrane; BR, undermining bone resorption; RR, root resorption; NB, new formation of bone on the side of the bone away from the tooth. (Orban, Jour. Am. Dent. Assn.)

The great number and extent of the necrotic areas in the periodontal membrane in this particular case make it probable that the poor general health and weakened resistance of this patient, who died of tuberculosis after a long illness, may have had something to do with this condition. It is very probable that the same occlusal forces which formerly did not exceed the tensile resistance of the periodontal fibers may have become traumatic and caused injuries to the tissues after the general resistance of the body was lowered by the severe illness.

A similar case was reported by Grohs. In the jaws of a man, aged forty-eight years, a great number of teeth had been lost by extraction, leaving an excessive amount of occlusal stress on the

remaining teeth. In a lower lateral incisor the periodontal tissue had been crushed between the root and the bone near the alveolar margin by an excessive tipping force exerted upon the crown (Fig. 301). The bloodvessels of the periodontal membrane became thrombosed, and the surrounding tissue was transformed into a homogeneous, structureless mass. (See the identical findings in animal experiments in Fig. 293.) Bone resorption took place in the marrow space behind the necrotic periodontal membrane, eventually completely undermining and eliminating the necrotic area.



FIG. 299.—Undermining resorption in case of traumatic necrosis of the periodontal membrane. C, cementum; S, necrotic space between root and bone; R, bone resorption undermining the necrotic area; NB, new formation of bone compensating for the resorption on the inside of the socket. (Orban, Jour. Am. Dent. Assn.)

In case of excessive stress exerted upon a tooth in a vertical (axial) direction, the periodontal membrane may be crushed at the fundus of the alveolus, causing a direct contact between root end and bone at this point. Such a condition is illustrated in Figs. 302 and 303. Fig. 302 shows the root tip of a human upper molar that was exposed principally to vertical stress. At the apex the periodontal membrane has been destroyed and the cementum surface is in direct contact with the bone. A necrotic space has formed between tooth and bone; on either side of this space the periodontal membrane is compressed and poorly stained. Fig. 303 shows an identical condition which was produced experimentally in the lower molar of a dog; the tooth wore a cap causing a force in a vertical direction. The periodontal membrane has been almost entirely

worn away at the apex; an empty space exists between tooth and bone. In the periphery undermining resorption of the alveolar bone is taking place.

These findings establish the similarity of the changes experimentally produced in animals to those occurring in human teeth under similar conditions. However, we must not forget that changes like the ones illustrated here will give few clinical symp-

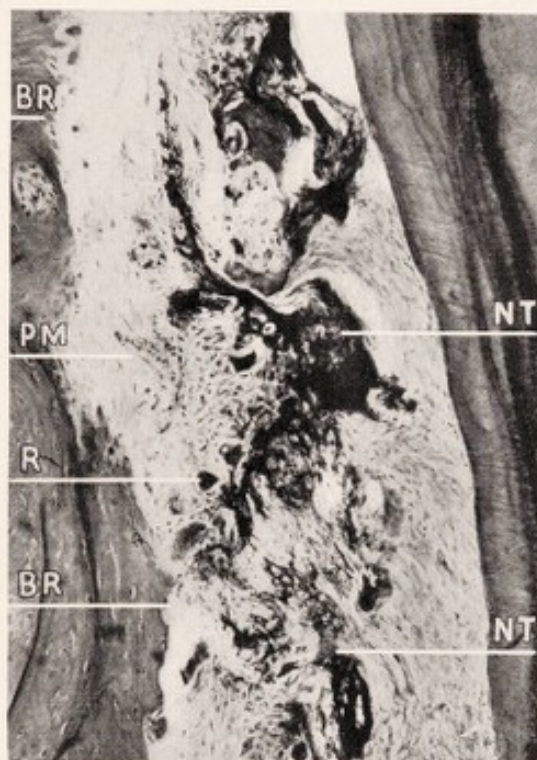


FIG. 300.—Final outcome of undermining resorption: resorption of the necrotic periodontal membrane (human specimen). NT, compressed necrotic connective tissue of the periodontal membrane; R, giant cells resorbing the tissue debris; PM, partly regenerated periodontal membrane; BR, resorption of alveolar bone. (Orban, Jour. Am. Dent. Assn.)

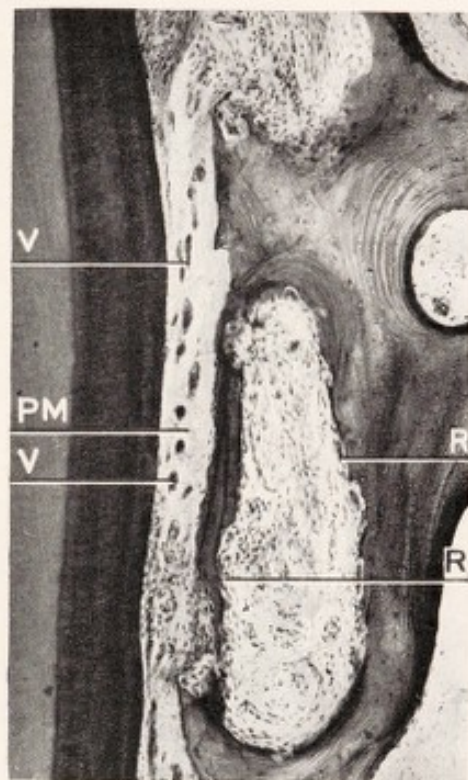


FIG. 301.—Thrombosis of the vessels of the periodontal membrane caused by occlusal trauma. Human lower incisor. PM, compressed periodontal membrane; loss of cell structure; V, thrombosed vessels; R, undermining resorption in the marrow spaces. Compare with Fig. 293. (Grohs, Ztschr. f. Stomatol., courtesy of Urban and Schwarzenberg, Berlin-Wien.)

toms except perhaps a slight soreness of the tooth. It is the author's opinion that such changes occur rather frequently; in fact, almost all human jaws that were examined under the microscope showed some minor traumatic injuries. However, these injuries usually have no clinical significance and will not interfere with the retention of the tooth unless the individual has an unusually low degree of resistance.

The etiology of such microscopic injuries due to occlusal trauma

may be illustrated by a practical example: A dentist sets an inlay that is slightly too high and dismisses the patient without checking the occlusion. The tooth with the inlay will, at least in some positions of the jaws, support the entire muscular force of the masticatory muscles, not unlike the capped teeth of dogs. A few days later the tooth is usually sensitive to pressure, and sometimes is



FIG. 302.



FIG. 303.

FIGS. 302 and 303.—Comparison between the traumatic injuries found in human periodontal tissues and the ones produced experimentally in animals.

FIG. 302.—Crushing of periodontal membrane and contact between root and bone at the bottom of the alveolus in case of vertical occlusal stress. Human upper molar. PM, destruction of periodontal membrane; formation of necrotic cleft between root and bone; AB, bone in contact with root surface; R, undermining bone resorption.

FIG. 303.—Crushing of periodontal membrane and contact between root and bone produced experimentally by cementing a metal cap upon the crown of the tooth. Lower molar of dog. PM, destruction of periodontal membrane; R, undermining bone resorption. (From the experiment of Gottlieb, Orban and Kronfeld.)

slightly loose. If the stress upon the tooth is relieved by grinding the inlay to normal occlusion, these symptoms disappear in a day or two.

From the knowledge gained from animal experiments as well as from human jaw material, we know that the following changes are to be expected in such a case: Immediately following the setting

of the inlay (or other restoration or filling which interferes with the occlusion) the periodontal membrane will be compressed in some places, depending upon the direction of force and the shape of the root. Minute hemorrhages in the periodontal membrane will take place, the tissue will be destroyed in some areas, and the root will come in contact with the bone (Figs. 298 and 301). These changes are manifested by a slight soreness of the tooth.

If at this stage the force exerted upon the tooth is relieved by grinding, the tooth reassumes its former normal position in the socket; the necrotic tissues are resorbed, and a normal periodontal membrane is regenerated.

If such relief is not given, the necrotic areas will be undermined by bone resorption from the periphery until the root is able to move more and more in the direction of the pressure and until the tooth adjusts itself to the newly created occlusal conditions and assumes a position in which the occlusal force is neutralized. Then reparative changes will take place, reestablishing normal function in the new position. If, however, tissue resistance and vitality are low, these traumatic changes will not be repaired; on the contrary, larger areas of the periodontal membrane will be involved until its extensive destruction and resorption of the alveolar bone lead to the loss of the tooth.

From this point of view our tissue studies corroborate what Stillman and McCall call "potential" and "actual" traumatic occlusion. Every increased force involves the potentiality of traumatic occlusion as indicated by microscopic injuries that do not show up clinically; but only if the reparative and adjusting forces of the organism fail, does actual traumatic occlusion develop and the tooth become damaged permanently.

It must, of course, be understood that merely a moderately increased occlusal stress will not cause necrotic changes in the periodontal membrane. If a filling is only slightly too high, perhaps not more than just enough to stretch some fiber groups in a certain direction, the vitality of the periodontal membrane will not be damaged; bone resorption will readily take place in the areas of pressure, relieving the periodontal soft tissues, and the force will be neutralized by only a very minute change in the position of the tooth.

In this connection it seems appropriate to mention an experience encountered in the practice of orthodontia. It was found, when applying an undue amount of force to a tooth in orthodontic movement, that the tooth moved rapidly at first, but then came to a

complete standstill. It was necessary to relieve the tooth of all force for a few weeks, and then carefully to start the movement again. What happened in this case? The excessive force applied stretched the fibers beyond their tensile resistance and caused crushing of the periodontal membrane between root and socket on the side of the pressure (see Fig. 294). Due to this injury the periodontal membrane could not produce osteoclasts, so that any further orthodontic movement was made impossible for the time being. After the tooth had been released, the necrotic membrane was gradually undermined, resorbed, and replaced by normal tissue; and not until the periodontal tissue had been completely repaired and was able to produce osteoclasts was further orthodontic movement possible.

3. Functional Stress and Root Resorption.—The findings in experimental occlusal trauma have shown that the bone is much more easily resorbed than is the tooth. If a tooth is pressed against its socket the bone, as a rule, will be eliminated; whereas, the tooth remains completely intact or is only slightly resorbed. Resorptions due to occlusal trauma found in microscopic examination of human teeth are usually very small and have little clinical significance (Fig. 304). Besides, such shallow resorptions of the root surface are repaired as soon as the trauma subsides. In dogs in which root resorptions had been produced by experimental occlusal trauma, reparative changes take place if the caps are removed and the animal is kept alive without caps for a sufficient length of time to allow cementum deposition on the resorbed dentin surface (Fig. 305).

As a whole, it must be stated that root resorptions due to occlusal trauma are insignificant as compared to idiopathic root resorptions of systemic origin (see Fig. 213). In the latter case, very extensive resorptive destruction of teeth can be observed without any interference of occlusal forces. In the entire problem of root resorption the constitutional factor seems to be of far greater importance than outer influences upon the teeth (trauma, inflammation). In individuals with high resistance, even a great amount of occlusal trauma will be unable to produce resorption of the roots. On the other hand, in patients with a disposition to root resorption very gentle forces, such as normal mastication or careful orthodontic treatment, may occasionally produce progressive root resorption, leading finally to complete destruction of the tooth.

Ketcham published a large number of radiographs of teeth, the roots of which had been extensively resorbed during orthodontic treatment. Although we are not yet in a position to give an exact

explanation of this incident, it seems, according to the observations of Oppenheim and others, that intermittent application of great force ("jerking movements") is responsible for the occurrence of such resorptions; whereas, they are not found or are invisible radiographically in continuous steady movement (see Chapter XIV).

4. Functional Stress and Gingival Crevice.—Functional conditions do not seem to have a great deal of influence upon the attachment of the gingival tissues. In animal experi-

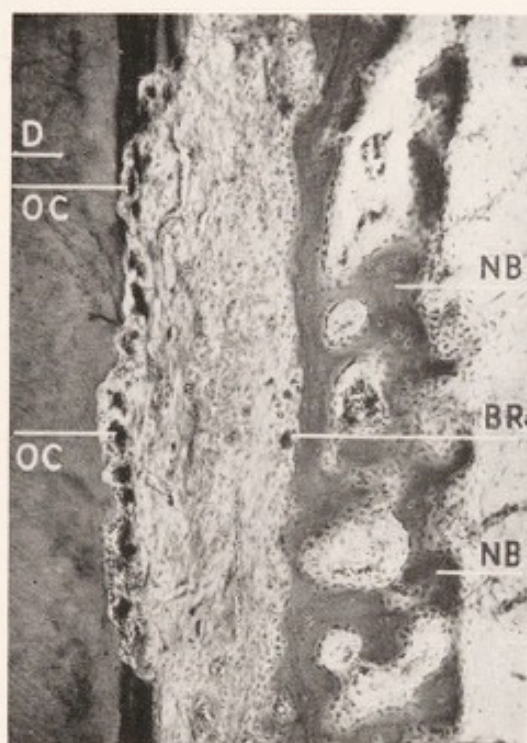


FIG. 304.—Root resorption due to excessive occlusal stress. Human tooth. D, dentin; OC, osteoclasts resorbing cementum and dentin; BR, bone resorption; NB, new formation of bone on the outer side of the alveolus. (Orban, Jour. Am. Dent. Assn.)

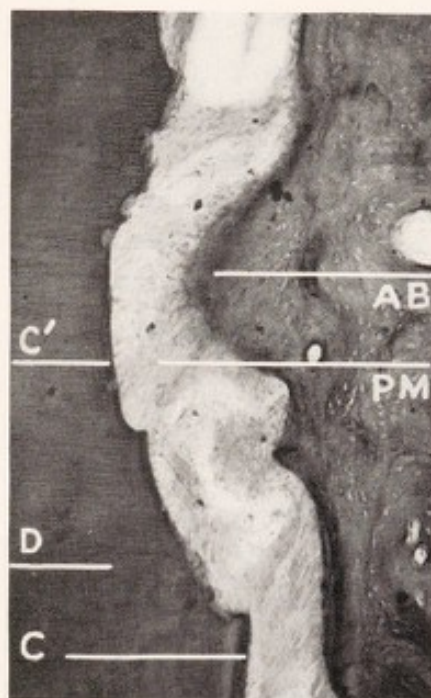


FIG. 305.—Repaired root resorption on a dog's molar. The resorption was produced experimentally by placing a metal cap on the tooth for nearly six months; then the cap was removed and the animal kept alive for an additional six months. D, dentin; C, original cementum surface; C', cementum deposited upon resorbed dentin surface after removal of caps; PM, fiber bundles of periodontal membrane regenerated in the resorbed area; AB, alveolar bone projecting into the resorbed area (functional repair, see Fig. 210). (From the experiment of Gottlieb, Orban and Kronfeld.)

ments it was impossible to produce gingival changes even by applying a great amount of occlusal force. The same observation was made when studying tissue sections of human teeth that had been exposed to great functional stress. There is, up to this time, no positive evidence that teeth exposed to great functional stress present any more advanced detachment of the epithelial attachment from the tooth surface than other teeth. On the contrary, it seems that strong masticatory activity, because of its self-cleansing action and because

of its stimulation of the blood supply, helps to maintain shallow crevices and hygienic gingival conditions. Lack of use and function, on the other hand, seems to favor the accumulation of deposits around the teeth and thus leads to gingival irritation.

The only instance where occlusal stress and gingival changes may have some direct relation is in the case of horizontal force. There the injury to the alveolar margin on the side of pressure may sometimes be the cause of the detachment of the gingival soft tissues and of the formation of a deep pocket in this area. Such a condition has been illustrated in Figs. 261 and 262. However, this tooth was the only one of a large number of teeth with similar occlusal conditions that showed a pocket on the side of pressure; therefore, this condition seems to be an exception rather than a rule.

5. Functional Stress and Pulp Changes.—The dental pulp has only one relation to the masticatory function, namely, that proper use of the teeth causes normal occlusal wear and normal involution of the pulp chamber by secondary dentin formation (see page 35). Denticles (pulp stones) are frequently found in the abraded teeth of older people; they have no significance other than that they are the expression of the tendency toward pulp calcification and reduction of the size of the pulp chamber.

It appears improbable that a pulp will die merely because of excessive functional stress. The bundle of bloodvessels at the apical foramen seems to have enough tensile strength and elasticity to adjust itself to the excursions of the apex and to keep the pulp vital. Death of the pulp occurred in none of the dogs' teeth used for the production of experimental occlusal trauma even if the teeth were markedly dislocated.

Once a tooth is considerably loosened, the vitality of the pulp is usually greatly reduced. Atrophic changes are then found in the pulp tissue, and in advanced stages of loosening the pulp may become necrotic. However, the death of the pulp in these cases is only indirectly related to occlusal stress because, as stated before, loosening of teeth may occur quite independently of occlusal forces.

BIBLIOGRAPHY.

- BAUER, W.: Über traumatische Schädigungen des Zementmantels der Zähne mit einem Beitrag zur Biologie des Zementes, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 769.
- BOEDECKER, C. F.: Critical Review of Gottlieb and Orban's "Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne," *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1932, **18**, 895.
- BOX, HAROLD K.: Traumatic Occlusion and Traumagenic Occlusion, *Oral Health*, 1930, **20**, 642.

- GOTTLIEB, B.: Traumatic Occlusion, Jour. Am. Dent. Assn., 1927, **14**, 1276.
- GOTTLIEB, B., and ORBAN, B.: Die Gewebsveränderungen bei Überbelastungen mit besonderer Berücksichtigung von Alter und Konstitution, Ztschr. f. Stom., 1931, **29**, 370.
- Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne, Leipzig, Thieme, 1931.
- GROHS, RICHARD: Veränderungen im Periodontium bei überlasteten menschlichen Zähnen, Ztschr. f. Stom., 1931, **29**, 386.
- HÄUPL, C.: Über traumatisch verursachte Gewebsveränderungen im Parodontium, Ztschr. f. Stom., 1927, **25**, 307.
- KAROLY, M.: Betrachtungen über Pyorrhea alveolaris und Caries dentium, Vrtljschr. f. Zhk., 1902, **18**, 520.
- KETCHAM, ALBERT H.: A Progress Report of Apical Root Resorption of Vital Permanent Teeth, Int. Jour. Ortho. Oral Surg. and Radiogr., 1930, **16**, 1, 1035.
- McCALL, JOHN OPPIE: Principles of Traumatic Occlusion and Their Application in the Field of Operative Dentistry, Jour. Am. Dent. Assn., 1930, **17**, 609.
- ORBAN, B.: Tissue Changes in Traumatic Occlusion, Jour. Am. Dent. Assn., 1928, **15**, 2090.
- STILLMAN, P. R.: Occlusion, Jour. Am. Dent. Assn., 1930, **17**, 811.
- STILLMAN, P. R., and McCALL, J. O.: A Textbook of Clinical Periodontia, New York, The Macmillan Company, 1922.

CHAPTER XIV.

TISSUE CHANGES IN ORTHODONTIA.

ORTHODONTIC therapy proposes to bring about changes in the position of the teeth and in the form of the jaws and face. By applying forces of different kinds to the teeth and jaws, the position of the teeth, the structure and arrangement of the surrounding bone and the relative position of the upper and lower arches are influenced and altered.

It is evident that the tissue changes in orthodontia must be of unusual interest both to the practitioner and to the scientific investigator. A tooth is normally anchored firmly in its alveolus and yields only fractions of a millimeter even if a heavy force is applied to it for a short period of time. Still, it is known from orthodontic practice that very delicate forces, if applied over a period of months or years, are able to cause very marked changes in the position of teeth and in the bone. This observation aroused the interest of investigators, and two theories were offered to explain the bone changes in orthodontic tooth movement.

Kingsley and Walkhoff¹ explained the orthodontic movements of the teeth as the result of compression and extension (elasticity) of the alveolar bone. If a tooth were forced by an appliance to move in a certain direction, they assumed that the bone was compressed on the side toward which the tooth was moving and extended or stretched on the opposite side of the root.

The other theory, offered originally by Schwalbe and Flouren,² was based on the assumption that the alveolar bone is resorbed and eliminated by osteoclastic activity on the side of pressure, while new bone is built on the side opposite the pull.

OPPENHEIM'S FINDINGS IN ANIMAL EXPERIMENTS.

In order to demonstrate what bone changes do actually take place when teeth are subject to orthodontic forces, Oppenheim, of Vienna, undertook a number of experiments, the results of which were published in 1911. He worked on the incisor teeth of a young

¹ Cited by Oppenheim.

² Ibid.

baboon, using simple orthodontic appliances (arch wires anchored to the molars). The incisors were attached to the arch wire by ligatures, and simple forms of tooth movement such as tipping, elongation, and depression (intrusion) were performed. After wearing appliances for forty days, the animal was killed, and the teeth and jaws were sectioned and studied under the microscope.

By means of these specimens Oppenheim demonstrated that the position of the teeth in the jaw can be changed only by means of resorption and new formation of the surrounding bone. Wherever pull is exerted, new spicules of bone are formed, arranged in the direction of the pulling force, and wherever pressure is exerted upon the alveolus, the bone is resorbed, thus creating space for the intended movement of the tooth. Oppenheim's findings, therefore, definitely disprove the theory of elasticity of bone.

Besides the resorption and apposition of the bone surface directly facing the root, there is also a very distinct, general rearrangement of the alveolar bone around the moved tooth. Under the influence of the moving root, the formerly solid and even bone plate of the alveolus is transformed into spicules of bone that are arranged parallel to the direction of force; each spicule shows bone resorption on one end and new formation of bone on the other end. The tearing-down and building-up of bone on each spicule is continuous as long as constant orthodontic force is applied to the tooth. If orthodontic movement is discontinued and the tooth is retained in its new position, this new arrangement of the alveolar bone gradually changes until the original solid bone plate again surrounds the tooth.

A few of Oppenheim's illustrations will serve to illustrate the fundamental bone changes obtained by orthodontic treatment in monkeys.

1. Tipping of a Tooth.—In a labio-lingual section through an incisor that has been tipped labially or lingually, we must differentiate between two opposite sides of the root surface: a side of pressure, toward which the tooth moves, and a side of pull, where pull is exerted upon the alveolar bone. In labial movement, for instance, the lingual surface of the alveolus is the side of pull. On that side new spicules of bone are built, following in their arrangement the direction of the stretched periodontal fibers (Fig. 306). The end of each spicule that faces the tooth is densely beset with osteoblasts; whereas, the end that is farthest from the tooth shows osteoclastic resorption. In lingual movement, a similar arrangement of the bone is found on the labial side, the side of pull. From these findings it is evident that in orthodontic movement there is

a tendency for new bone to fill in behind the moving tooth on the side of pull. When the orthodontic force is first applied, the periodontal membrane fibers on the side of pull are stretched and the periodontal space is widened. As a result, bone formation is stimulated on the inner wall of the alveolus and tends to reduce the periodontal space to its normal width. This process continues as long as force is exerted upon the crown.

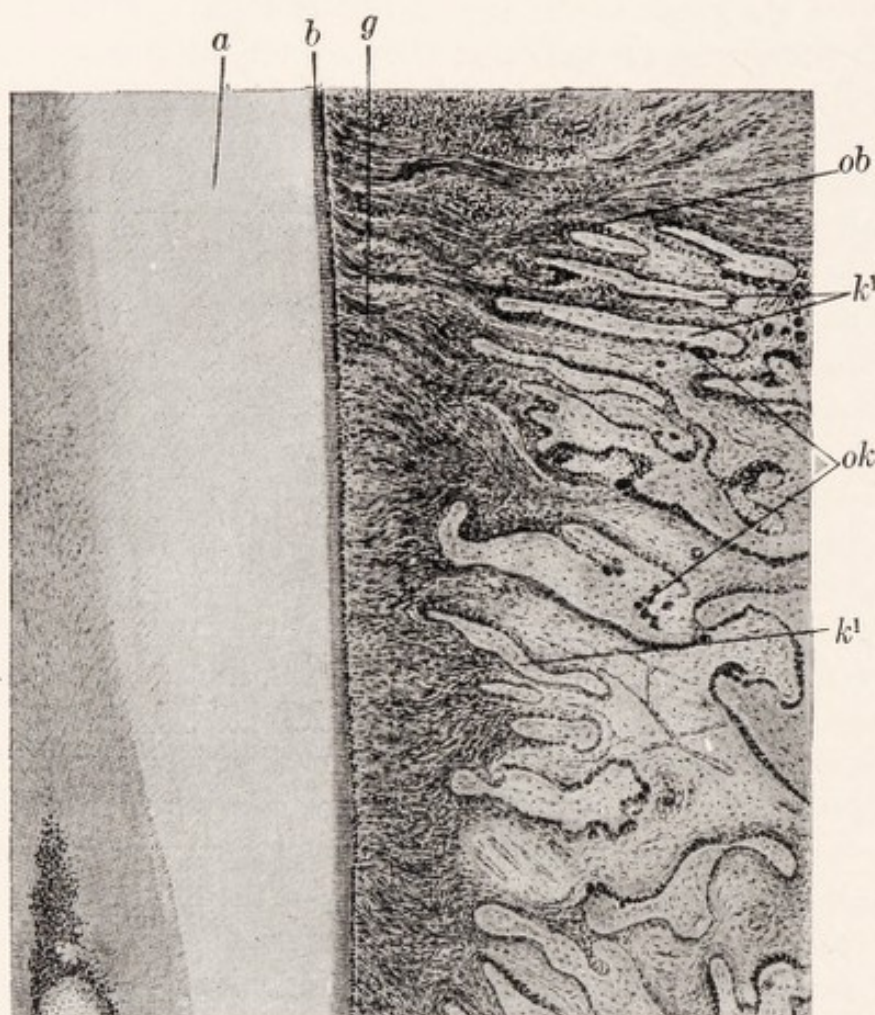


FIG. 306.—Labial movement, lingual side. Incisor of young monkey moved orthodontically for a period of forty days. Near the alveolar margin the bone is arranged in long spicules, k^1 , oriented vertically to the long axis of the tooth. Each spicule is densely beset with osteoblasts, ob , on the end facing the tooth; at the opposite end, the bone is being resorbed by osteoclasts, ok . a , dentin; b , cementum; g , periodontal membrane. (Oppenheim.)

While the bone-building activity on the inner end of the spicules is easy to understand, the resorptive process on the outer ends in case of pull is somewhat more difficult to explain. Perhaps the resorption that is always concurrent with new-formation is the expression of a biological tendency to remove any surplus bone that may form. New formation of bone on the side of pull would

necessarily cause a thickening of the bony plate on that side, but as there is no functional need for such a thickening, the excess bone is removed by resorption.

On the side of pressure, bone changes occur in a somewhat different manner. Here, too, the bone is transformed into spicules arranged

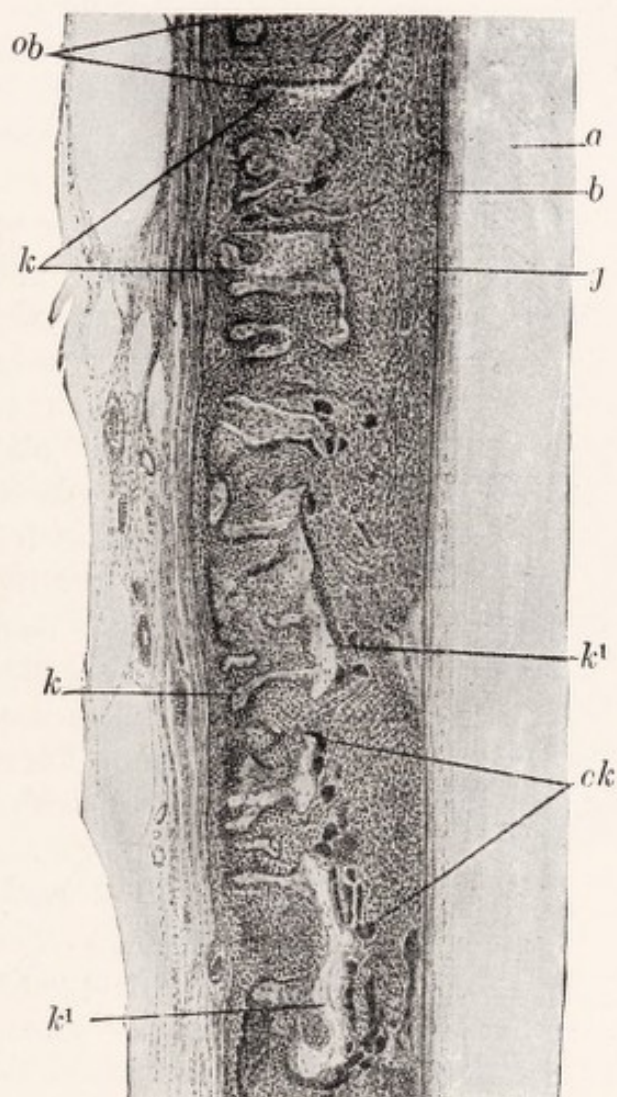


FIG. 307.—Labial movement, labial side. Incisor of monkey. Near the alveolar margin the compact bone has disappeared and is replaced by spongy bone, *k*, with orientation of the spicules vertically to the long axis of the tooth. At the end which faces the tooth, each spicule is beset with osteoclasts, *ok*, at the opposite end with osteoblasts, *ob*. *k*¹, remains of the original old bone; *a*, dentin; *b*, cementum; *g*, periodontal membrane. (Oppenheim.)

at right angles to the direction of force; but the distribution of resorption and new formation on each spicule is exactly opposite to that found on the side of pull. Each spicule shows resorption on the extremity next to the tooth surface and new-formation of bone on the end away from the moving tooth (Fig. 307).

The biological significance of these changes is plain. The labial

and buccal plate of bone covering the roots is very thin; therefore, in labial or buccal movement, the resorption of the bony plate on the side of pressure would rapidly lead to complete destruction of the labial or buccal bone were it not for simultaneous new-formation

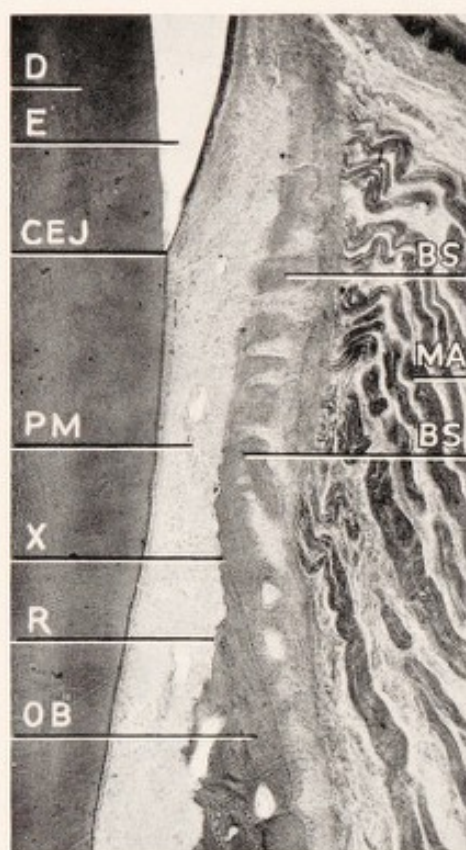


FIG. 308.—Labial movement. Alveolar crest on the labial side. Incisor, monkey. D, dentin; E, enamel; CEJ, cemento-enamel junction; PM, periodontal membrane; OB, old bone; X, upper end of the old bone; R, resorption on the inner surface of the old bone; BS, newly formed bone in spicules arranged vertically to the long axis of the tooth; MA, labial muscle attachment. Each spicule shows evidence of resorption (osteoclasts) on the end facing the tooth and new-formation of bone (osteoblasts) on the opposite end. (Breitner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

space between tooth and bone. When the appliances are put in place and the force is applied for the first time, the tooth is forced against one side of the socket, and pressure is exerted upon the intervening soft tissues of the periodontal membrane. As a result,

of bone. By new-formation an outer plate of bone is constantly maintained, which gradually moves more and more toward the buccal or labial side as the tooth advances in that direction.

Rearrangement of the alveolar bone under the influence of pressure will be illustrated by a specimen from an experiment of Breitner (Fig. 308). An upper incisor of a young monkey had been moved labially by an orthodontic appliance. The orthodontic force resulted in resorption on the inner surface and compensatory bone development on the outer surface of the labial plate. The bone spicules are arranged in the direction of force; on the spicule end near the tooth osteoclasts are present, whereas, on the outer (labial) end of each spicule osteoblasts are active. The X in Fig. 308 indicates the upper end of the original bone; above this point the tipped tooth has passed the plane of the original labial plate and is entirely surrounded by new bone. Had it not been for constant new-formation, no bone would be left on the labial side above X.

Bone resorption on the inner side of the alveolus on the side of pressure is the result of a decrease in the

the bone is resorbed by osteoclastic activity and the pressure upon the soft tissues is relieved. Bone resorption enables the tooth to move in the direction indicated by the orthodontic force; this movement is continuous until the force has expired.

Oppenheim's specimens showed that in tipping movement the bone changes are greatest near the alveolar margin and from there steadily decrease toward the apex. This would indicate that the fulcrum of the tipping tooth is located at the apex. In the discussion of the influence of function upon the teeth, it was stated that the fulcrum of a tipping tooth is located in the apical third of the root, so that the apex moves in a direction opposite to the rest of the root (see page 339). Both of these statements although apparently in discord may hold true, although under different conditions: Powerful tipping forces will cause the apex to move opposite to the direction of the crown, while gentle tipping forces will have so little effect that excursions of the apex may be disregarded for all practical purposes. Hence with delicate forces, as are applied in modern orthodontia, the fulcrum is located at or near the apex; whereas with strong forces the fulcrum is located at the border between the middle and the apical third of the root.

In this country, Johnson, Appleton, and Rittershofer carried out experiments similar to those of Oppenheim and published similar results. They worked on upper central incisors of young monkeys. In their specimens the apex shows a marked deviation toward the side opposite the direction of movement of the crown. Schwarz, in his study of the mechanism of tipping of teeth, offered a possible explanation for the difference between the findings of Oppenheim (apex not displaced) and the American authors (apex moved opposite to the direction of the crown). In Oppenheim's case the teeth had fully formed apices and apical fibers; whereas the monkeys used by Johnson, Appleton, and Rittershofer still had wide open apical foramina and, therefore, no apical fibers. In the first case, the apical fibers anchored the root tip to the fundus of the alveolus; in the second case, such fibers had not yet developed, and therefore the apical portion of the root was more susceptible to orthodontic influence.

There is another important point in connection with orthodontic tooth movement. It is taken for granted that if orthodontic force be properly applied alveolar bone will be resorbed to make way for the tooth and that the tooth will remain intact. This assumption is fully corroborated by clinical observations.

Why is it that when gentle pressure is exerted upon the peri-

odontal membrane, resorptive processes are found only on the bone and not on the tooth surface as well? Physically, the amount of pressure exerted upon the tooth surface must be identical to the amount of pressure exerted upon the bone surface (law of action and reaction). There must, therefore, be a biological difference between tooth and bone surface, and we know from observations



FIG. 309.—Elongation. Apex of the tooth. Incisor of monkey. Long bone spicules, k^1 , arranged in the direction of pull. The upper end of each spicule is surrounded by osteoblasts, *ob*, the lower end by osteoclasts, *ok*. *a*, apex. (Oppenheim.)

in other fields of dental pathology that such a difference exists. Cementum is, under otherwise identical conditions, much more resistant to resorption than bone, and upon this difference the entire practice of orthodontia is based. Were this not so, were cementum as equally subject to resorption as bone, there would be no reason for the tooth not being resorbed just as much as the sur-

rounding bone, which would make orthodontic therapy impossible. Thus it is seen that cementum and bone, although very closely related in origin and structure, are quite different in their biological reaction to pressure stimuli, a fact which has a great practical bearing upon an important branch of dentistry.

2. **Elongation of a Tooth.**—The elongation of a tooth, as it was carried out by Oppenheim in his experiments, results in the formation of new bone at the fundus of the alveolus and at the alveolar margin. At the fundus of the alveolus, bone spicules are found

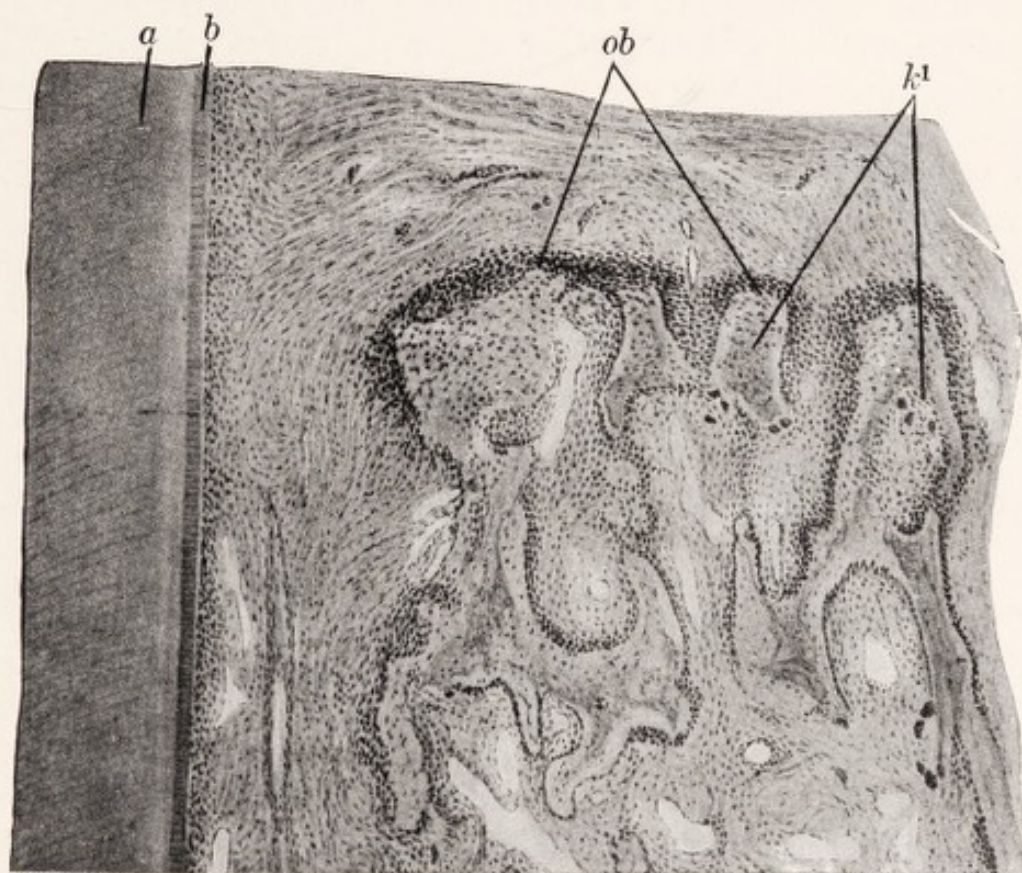


FIG. 310.—Elongation. Lingual alveolar margin. Newly formed spicules, k^1 , of spongy bone arranged in the direction of the pull. The spicules show osteoblasts, ob , at the upper end and osteoclasts at the lower end. a , dentin; b , cementum. (Oppenheim.)

arranged in the direction of the existing pull, out of the socket, parallel to the long axis of the tooth (Fig. 309). Osteoblasts are present on each spicule at the end toward the tooth, and osteoclasts are present on the opposite ends. At the alveolar margin, formation of new bone can be found along the entire circumference of the elongated tooth (Fig. 310), by which development the alveolus evidently adjusts itself to the new raised position of the tooth.

3. **Depression of a Tooth.**—In orthodontic depression (intrusion) of a tooth, the bone changes are exactly opposite to those described

in elongation. There is resorption of bone over the entire inner surface of the socket, but especially at the fundus of the alveolus and at the alveolar margin. These changes indicate that the alveolus tends to assume a new shape adequate to the artificially created, lowered position of the tooth.

4. **Tissue Changes During the Period of Retention.**—Oppenheim showed that after the active orthodontic force has been discontinued and an appliance has been put in place to retain the tooth in its new position, characteristic changes take place in the alveolar bone. During the time of active treatment the bone is arranged in delicate spicules parallel to the direction of force. During the period of retention these spicules are rearranged and are gradually transformed back into the solid alveolar bone plates as they were before the treatment was started. Typical Haversian systems develop and the new bone assumes the character of lamellated bone. The final arrangement of the bone and the form of the alveolus will depend largely upon the prevailing anatomical conditions and on the functional requirements which the tooth has to meet in its new position.

BONE CHANGES AROUND A HUMAN TOOTH FOLLOWING ORTHODONTIC MOVEMENT.

Oppenheim's findings, although largely recognized by the profession as the main source of information about orthodontic bone changes, were applicable to man only with reservations as long as parallel findings in human specimens were not available. Due to technical difficulties, it had been impossible to secure such material from man. It is, therefore, of interest to observe the histological findings in a specimen of a human bicuspid (Herzberg). This tooth had been moved lingually for seventy days, and was then removed together with a small piece of the buccal alveolar bone.

The microscopic examination revealed changes very similar to those produced by Oppenheim in monkeys under identical conditions. Fig. 311 shows the buccal alveolar margin (side of pull) of this specimen. The bone spicules are arranged in the direction of pull at right angles to the surface of the tooth. On the outer side of the alveolar process, resorption can be seen. Fig. 312 shows a high magnification of a small area of alveolar bone on the side of pull, in about the middle of the root. Each small spicule of bone is lined by osteoblasts and osteoid on the side facing the tooth; whereas bone resorption is going on on the side away from the

tooth. By comparing these findings with those shown in Fig. 306 one can readily assume that there really is justification for applying Oppenheim's findings in animal experiments to similar conditions in man.



FIG. 311.—Lingual movement, buccal side. Alveolar margin of human bicuspid that was moved orthodontically over a period of seventy days. D, dentin; C, cementum; PM, periodontal membrane; BS, alveolar bone, arranged in bony spicules orientated parallel to the direction of pull; Ob, osteoblasts and seam of osteoid (newly formed uncalcified bone) bordering the periodontal membrane; R, lacunar resorption of alveolar bone on opposite side of the spicules. (Herzberg, Jour. Am. Dent. Assn.)

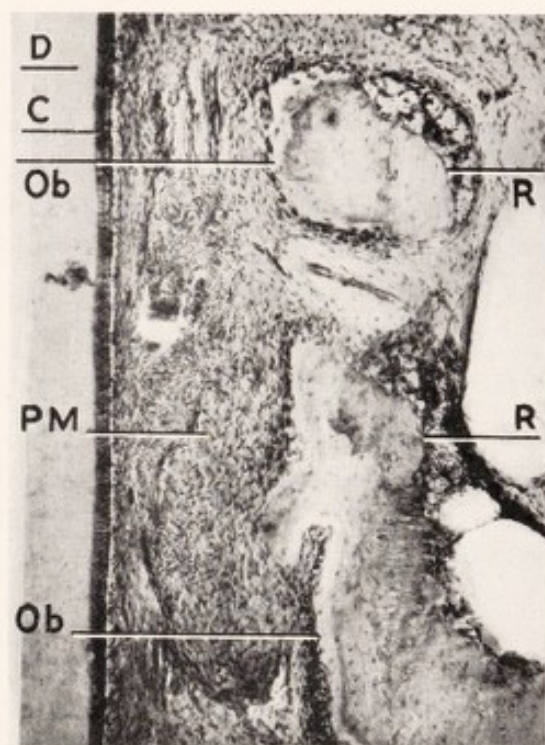


FIG. 312.—Higher magnification of Fig. 311. Distribution of bone resorption and new-formation on the individual spicules. Human bicuspid. D, dentin; C, cementum; PM, periodontal membrane; Ob, osteoblasts and seam of osteoid (newly formed uncalcified bone) bordering the periodontal membrane; R, lacunar resorption of alveolar bone on opposite side of the spicules. (Herzberg, Jour. Am. Dent. Assn.)

QUANTITATIVE EVALUATION OF ORTHODONTIC FORCES.

The laboratory investigator in the field of orthodontia is frequently asked by the practitioner what the term excessive force actually means? Is there any way to determine just how great an orthodontic force must be in order to produce a certain well-defined tissue reaction?

In a series of experiments performed on dogs, Schwarz recently approached this very important problem of determining the relationship between an orthodontic force of known intensity and the

tissue changes produced by this force. He used an appliance that consisted of a strong arch wire anchored to the molars and canines. This arch wire lay on the lingual side of the premolar teeth. Auxiliary springs were soldered to it in such a way that the premolars were moved buccally. The force exerted by the auxiliary springs had been previously measured so that each premolar was subjected to a known amount of force.

The appliances were left in place for a period of several weeks; then the animal was killed, and the teeth together with the surrounding jaw bone were sectioned. The microscopic examination showed a definite relationship between the amount of the force and the type of tissue reaction. Only where the force applied had not exceeded approximately 20 gm. per square centimeter of root surface was the desired ideal biological tissue reaction observed, namely, uniform bone resorption on the side of pressure and corresponding uniform apposition of new bone on the side of pull, without any evidence of injury to the periodontal tissues or of root resorption. When a force of approximately 60 gm. had been applied for several weeks, the periodontal membrane was compressed and crushed between root surface and bone, so that no typical orthodontic bone resorption could take place. Instead, undermining bone resorption was found at the borders of the damaged areas (see Fig. 296). There was evidence of root resorption in addition to the traumatic injury to the periodontal membrane.

Schwarz drew some very important conclusions from these findings: If a constant force is to be exerted upon a tooth without causing damage to the periodontal soft tissues, this force must not exceed the capillary blood-pressure in these tissues. Since the capillary blood-pressure is known to be from 20 to 26 gm. per square centimeter, the orthodontic force should not exceed approximately 20 gm. per square centimeter of root surface. For larger or multi-rooted teeth, the absolute force can, of course, be correspondingly greater. If force in excess of the capillary blood-pressure is applied, it will result in anemia of the compressed periodontal soft tissue with subsequent asphyxia and death of this tissue. Later the death of the periodontal tissue is followed by extensive and irregular undermining bone resorption and frequently also by root resorption.

Schwarz divided the orthodontic forces into four groups according to their biological reaction:

1. *First Degree of Biological Reaction.*—The force is of such short duration or so gentle that it causes no definite reaction in the periodontal tissues.

2. *Second Degree of Biological Reaction.*—The force is gentle in a biological sense: it does not exceed the capillary blood-pressure, which is 20 to 26 gm. per square centimeter, and maintains a continuous resorptive process on the side of pressure. The result is the desired orthodontic movement without any damage to the tissues.

3. *Third Degree of Biological Reaction.*—The force is fairly strong: it is slightly in excess of the capillary blood-pressure in the periodontal membrane on the side of pressure, causing anemia and injury of the soft tissues. No bone resorption can occur in the damaged area, but subsequently resorption takes place at the borders. Usually the outcome will be a regeneration of the tissues after the damaged areas have been removed by undermining resorption; however, this kind of force is likely to cause root resorption.

4. *Fourth Degree of Biological Reaction.*—The force is strong. It crushes the periodontal membrane on the side of pressure, bringing about a direct contact between root surface and bone. There is even a possibility of the pulp dying because of the tearing and hemorrhage at the apex. Later, extensive bone resorption takes place with the probability of root resorption.

The varying individual susceptibility to root resorption will, of course, always have to be considered. The teeth of some individuals will stand a great amount of force without being resorbed, while in others, comparatively small amounts of force will cause extensive root resorption.

In summarizing the knowledge gained from the experiments of Oppenheim, Johnson, Appleton and Rittershofer, and Schwarz, we come to the following conclusions: The only desirable and biological form of tooth movement is that by which regular and uniform bone resorption takes place on the side of pressure and regular and uniform formation of new bone on the side of pull. These changes are brought about by very gentle, uniform and continuous orthodontic forces. The term biological is used here because a gentle orthodontic force brings about exactly the same bone changes as are observed when a tooth moves through the bone during its eruptive movement. Any tissue changes other than those found in an erupting tooth indicate that an excessive or improper force has been applied.

RESULTS OF EXCESSIVE FORCES IN ORTHODONTIC PROCEDURES.

1. *Injury to the Periodontal Membrane.*—The periodontal membrane is a soft tissue organ embedded between the cementum and

bone. If an excessive force is applied to the tooth, this soft tissue will be crushed and injured between the two hard substances, a fact which Oppenheim showed in one of his experiments (Fig. 313, labial movement of an incisor by heavy force over a period of thirty days). The periodontal width on the labial side (side of pressure) appears greatly reduced. The bloodvessels of the periodontal membrane are filled with dark masses of coagulated blood

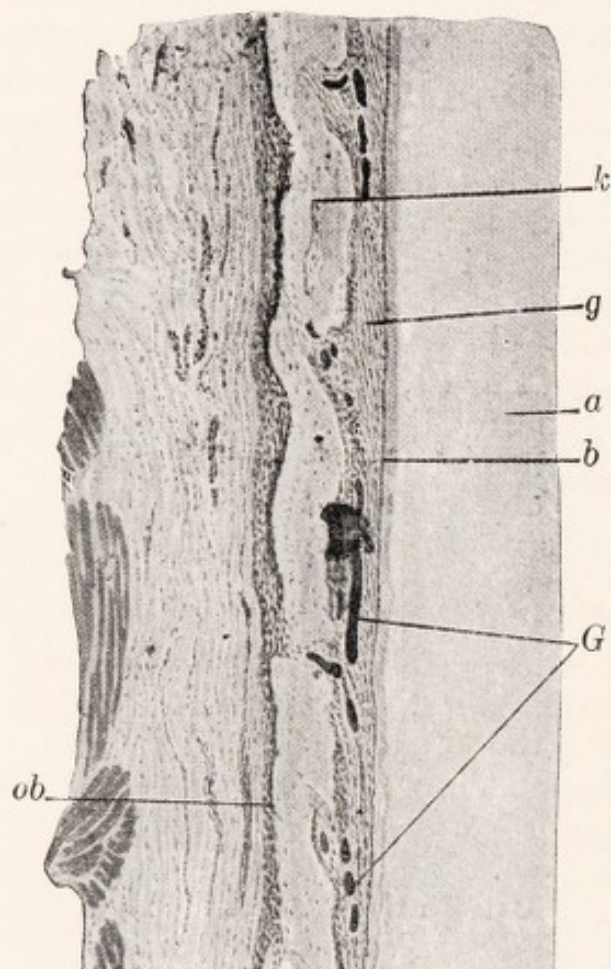


FIG. 313.—Labial movement, labial side; application of intense force. Incisor of monkey. The compact bony plate, *k*, somewhat attenuated throughout, has no change in its architecture. All vessels, *G*, of the periodontal membrane are filled with a homogeneous mass (thrombosis); there is no evidence of bone resorption at the inner wall of the alveolus. *a*, dentin; *b*, cementum, *g*, periodontal membrane; *ob*, osteoblasts. (Oppenheim.)

(thrombosis). This thrombosis is characteristic of all the cases in which the periodontal membrane has been damaged by excessive force exerted upon the tooth (see also Figs. 293 and 301).

When excessive force is applied no changes are visible at first in the bone next to the periodontal membrane containing the thrombosed vessels (Fig. 313). This is an observation of great practical importance. It explains why the use of intense force in ortho-

dontia will sometimes lead to the complete standstill of a tooth rather than to the expected movement. An intact periodontal membrane is essential to the occurrence of bone resorption on the inside of the alveolus. If the periodontal membrane is damaged to such extent that no osteoclasts can be produced, bone resorption and, consequently, orthodontic movement are impossible.

Thus Oppenheim's work for the first time introduced biological thinking into the practice of orthodontia. By means of clear deductions, he showed that the use of an intense force will not lead to any better or faster results than the use of gentle force. It is necessary to give the tissues time to adjust themselves to the changed condition: the bone must be resorbed and rearranged and new bone must be built to make up for the loss by resorption. If too much force is applied, the periodontal soft tissues are damaged, and no coördinated orthodontic movement is possible until these tissues have completely recovered. Oppenheim also showed microscopically with monkeys, as well as clinically with man, that teeth can be moved over great distances without root resorption. Only excessive or irregular forces are likely to cause resorption of the roots.



FIG. 314.—Root resorption on a lower molar in case of intense orthodontic force. Molar, monkey. RR, resorption of root end; FB, fiber bundles of periodontal membrane; BR, resorption of the inner lamina of the alveolar bone. (Breitner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

2. Root Resorption Associated With Orthodontic Treatment.—

Another frequent result of inadequate or excessive force is root resorption during orthodontic treatment. Oppenheim who used very gentle forces on the teeth of monkeys did not observe root resorption in his specimens. Breitner who intentionally put excessive occlusal stress upon some teeth ("jumping the bite") found extensive root resorption (Fig. 314).

But not only the intensity, but also the kind of force plays an important rôle in the problem of root resorption in orthodontia. Intermittent or "jerking" forces seem to be more likely to produce root resorption than constant forces. Gottlieb and Orban offer the following explanation for the higher incidence of root resorption from intermittent forces: In the first place, it must be remembered that the tooth surface is much more resistant to resorption than the surface of the surrounding bone. Therefore, as long as the tooth is moved by gentle or even moderately strong forces, the bone only will be resorbed and the tooth will remain intact. If the application of force is discontinued, a certain amount of reparative new-formation of bone will take place on the side where pressure had previously been exerted. This newly formed bone is more resistant to resorption than the old bone of this area, this higher resistance being the result of the higher vitality of newly formed hard tissue (see also page 232 and Fig. 213). When the orthodontic force is again applied the root meets a bone surface possessing greater resistance to resorption, and consequently there is more likelihood of the tooth being resorbed than before. Again the author wishes to emphasize that resistance, in this connection, must not be interpreted in a mechanical sense. The newly formed bone is not any harder than the old bone; on the contrary, it is in all probability softer. Still this new bone possesses a higher degree of vitality. Therefore, it has a greater biological resistance to resorption and is less easily attacked by osteoclasts than the older bone or perhaps even the root surface.

Little is known at present about the systemic factors involved in orthodontic root resorption. It is known from clinical observation that the susceptibility of teeth to resorption increases with advancing age; hence the increased danger of root resorption in orthodontia in adults. Gottlieb and Orban report a much higher incidence of root resorption due to occlusal overstress in old dogs than in young ones. Marshall produced extensive root resorption by orthodontic appliances on the incisors of a monkey that was fed a diet deficient in vitamin A. However, since this investigator worked with forces that were strong enough to cause an actual fracture of the alveolar process, the part played by the diet in the production of the lesions is hard to determine. The intensity of the forces that he used seems also to have been responsible for the great amount of inflammatory reaction present around the resorbed root, a phenomenon which has not been observed by any other experimenter in this field and which certainly indicates an unusually

violent form of tissue destruction. Marshall's experiments are valuable as a warning against the use of excessive forces, especially if his work is compared with Oppenheim's experimental findings. Oppenheim worked on deciduous teeth which, as is generally known, are *per se* more susceptible to root resorption. Still, by using gentle forces, Oppenheim was able to produce typical orthodontic bone changes with practically no root resorption. Marshall, on the other hand, worked on permanent teeth that are much more resistant to resorption than deciduous teeth; yet, through the combined action of strong orthodontic forces and dietary deficiency with resulting lowered resistance, he produced extensive root resorptions.

The final outcome of root resorption in orthodontia depends largely upon the extent of the resorptive process. In extreme cases a large portion of the root may be destroyed with the result that the tooth is loosened and eventually lost. Small resorptions are usually repaired by deposition of cementum after the stress has been relieved. Sometimes resorption may cause only a shortened root end, indicating that the apex alone had been involved in the resorptive process. Small root resorptions occur perhaps more frequently during orthodontic treatment than is usually assumed; however, since only small areas of the root are involved, repair takes place immediately after the treatment is finished. It must be kept in mind that only a fairly extensive loss of tooth substance is visible in the radiograph, and then only if the involved area is located on the mesial or distal side of the root or at the apex.

Histological investigations of root resorption during orthodontic treatment were recently reported by Grubrich and Gubler. Both authors examined sections through human teeth that had been moved orthodontically over a period of several months and then had been extracted. A large percentage of them showed shallow root resorption on the side of pressure near the alveolar margin. These areas of resorption were so small that they could not be seen in the radiograph. In many instances they had been repaired by depositions of cementum. These findings strongly suggest that root resorptions during orthodontic treatment are much more frequent than is usually believed. However, since most of these resorptions are very small, they cannot be recognized clinically. The author is convinced that if the radiographic diagnosis of root resorption in orthodontia could be replaced by histological examination of the moving teeth, a much higher incidence of root resorption would be found.

3. Changes in the Shape of the Root Following Orthodontic Treatment.—It is frequently observed in radiographs that teeth subject to orthodontic treatment in early life show blunted, shortened roots, different from other roots in the same mouth. The history of these teeth suggests that some influence may have been exerted upon the forming root end. Johnson, Appleton, and Rittershofer found that Hertwig's sheath at the open foramen of a young, moved tooth showed a distinct deformity which was not present in a control tooth

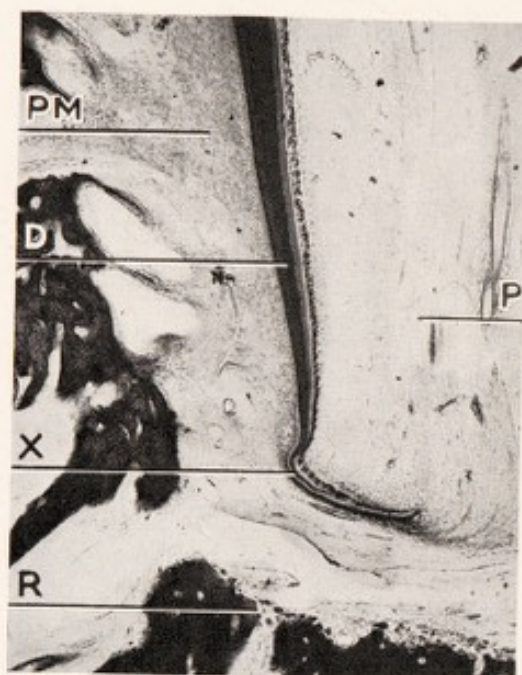


FIG. 315.—Deformity of the end of a lower cuspid in a young dog caused by ligating the tooth to a labial arch wire for seventy-two hours. D, dentin. At X, the young uncalcified dentin and Hertwig's sheath are folded and compressed; P, pulp; PM, periodontal membrane; R, resorption of alveolar bone. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

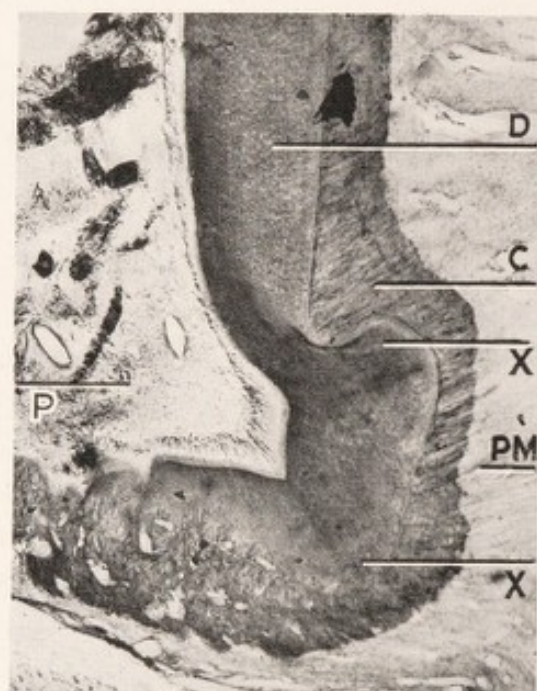


FIG. 316.—Deformity of the root end due to heavy occlusal stress at the time of root development. Apex shortened and blunted. Molar, dog. C, cementum; D, dentin showing at X folding and compression of the root surface (see Fig. 315); P, pulp; PM, periodontal membrane. (From the experiment of Gottlieb, Orban and Kronfeld.)

of the same animal. Since Hertwig's sheath represents the pattern of the root during the developmental period, a deformity of the root can be expected in teeth subjected to orthodontic movement. In the course of the experiments with occlusal trauma published by Gottlieb and Orban, root deformities were produced by applying orthodontic force to very young teeth. In these teeth Hertwig's sheath appeared deformed and folded (Fig. 315). If the animal were then kept alive long enough to allow the apex to form com-

pletely, a corresponding deformity was present in the completely formed root end. The entire root appeared shorter and blunter than the control teeth of the same animal (Fig. 316).

These findings in experimental animals have not yet been corroborated by similar microscopic findings in human teeth; still they suggest such possibilities for human teeth. We may summarize our present knowledge of changes in the form and outline of the root ends of orthodontically moved teeth as follows: Blunted or shortened root ends in teeth following orthodontic movement may be due either to resorption at the apex or to a deformity and distortion of Hertwig's sheath; the latter can be suspected if the tooth in question were moved in early life before root formation was completed. Only a microscopic examination can decide which one of the two processes took place in any particular case.

CHANGES IN THE MESIO-DISTAL RELATIONS OF THE JAWS.

In orthodontic practice, it is possible to change the mesio-distal relationship between upper and lower jaw by the use of intermaxillary rubber bands, inclined planes, and head caps. In 1930 and 1931 Breitner published a series of articles dealing with experimentally produced changes in the relative position of the jaws of monkeys. He used *en bloc* anchorage, covering several posterior teeth in each jaw with caps to which intermaxillary rubber bands were fastened by means of hooks. In one case he moved the whole lower jaw forward, as it is done for the correction of a Class II occlusion (Angle) in man, but since the animal had normal occlusion to begin with, he caused a Class III (Angle) occlusion. In another animal Breitner reversed the appliances and moved the mandible from its normal position (Class I) into a Class II relation. A third experiment, "jumping the bite," was carried out by cementing metal caps with oblique planes on the posterior teeth.

The histological changes found when a mandible was moved forward or backward by means of intermaxillary rubber ligatures can be divided into three groups:

1. Movement of all teeth that were used for anchorage and upon which the force acted.
2. Changes in the form of the mandible (lengthening or shortening) due to rearrangement of the bone of the ramus, the angle and the condyloid process.
3. Displacement of the mandible at the base of the skull by changes in the mandibular fossa.

1. **Mesial Movement of the Mandible.**—After the mandible had been moved forward over a period of eighty-two days by means of orthodontic rubber bands, the animal, which by this time showed a marked Class III occlusion, was killed. The jaws and the joints were sectioned in mesio-distal direction. Only the most significant of the histological findings will be described.

In the mandibular fossa new formation of bone was found on the distal wall; the bone was arranged in horizontal spicules, indicating the presence of a forward pulling force at the condyle (Fig. 317). On the mesial wall of the mandibular fossa, resorption of the bone took place, indicating a beginning mesial displacement of the joint and of the entire mandible.

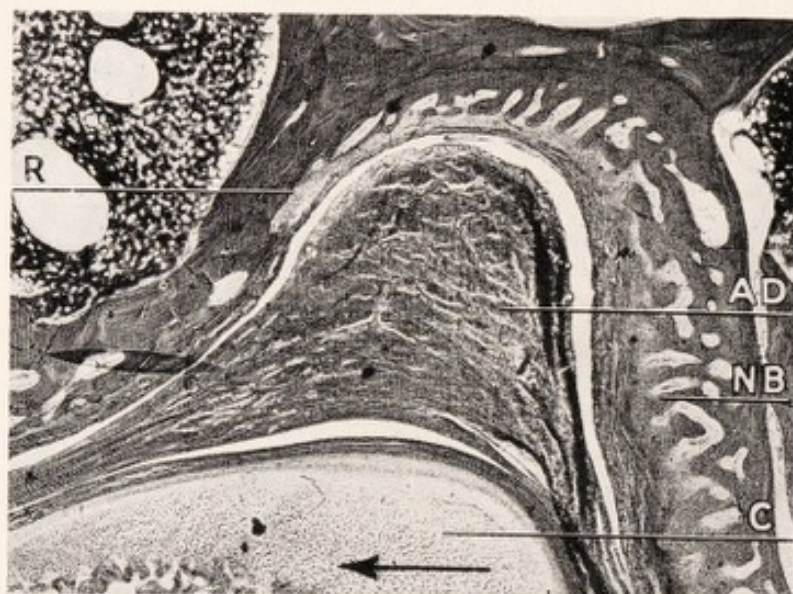


FIG. 317.—Orthodontic mesial movement of the mandible. Changes in the temporo-mandibular articulation. Monkey. C, condyle of mandible; the arrow indicates the direction of movement of the mandible; AD, articular disk; NB, new-formation of bone on the posterior wall of the mandibular fossa; the spicules are arranged in the direction of pull; R, bone resorption on the anterior wall of the mandibular fossa. (Breitner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

On the distal side of the ramus and the condyle, formation of new bone was found (Fig. 318). At the angle of the mandible resorption had taken place.

These findings indicate that in time the form of the mandible would change: the mandible would become longer (addition of bone at the posterior side of the ramus) and the angle more obtuse (resorption at the angle).

The anchor teeth in both the lower and upper jaw were moved, the upper ones distally, the lower ones mesially.

2. **Distal Movement of the Mandible.**—The appliance used was the same as in 1, except that the hooks were arranged in the opposite

direction and pulled the mandible backward. The duration of the experiment was seventy-two days. The arrangement of the bone changes in the mandibular fossa was found to be exactly opposite to that found in the case of mesial movement; there was resorption on the distal wall of the fossa and new-formation of bone on the mesial wall. Thus, the mandibular fossa seems to adjust itself to the intended distal displacement of the condyle.



FIG. 318.—Higher magnification of the distal side of the condyloid process in Fig. 317. C, cartilage; NB', newly formed bone on the distal surface of the neck of the condyle; NB'', newly formed bone of similar arrangement on the posterior wall of the mandibular fossa. (Breitner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

The ramus and the condyloid process showed resorption on the distal surface, while new bone was being formed at the angle of the mandible. By this distribution of the bone changes, the mandible tends to become shorter, and the angle between the ramus and body sharper.

The teeth in the lower jaw were moved distally, the ones in the upper jaw mesially. Fig. 319 shows the crest of the interdental septum between two upper posterior teeth, the arrow indicating the direction of movement of the teeth. On the mesial side of the septum there is new-formation of bone arranged in the direction of pull; on the distal side, the side of pressure, the bone is densely beset with osteoclasts.

3. **"Jumping the Bite."**—Caps with oblique planes were cemented on the posterior teeth and left there for forty-six days; the oblique

planes caused a forward sliding of the mandible during the closing movement of the jaws. In the joint, changes similar to those described under 1 were found, corresponding to a forward displacement of the condyle. All teeth that wore caps showed rather extensive crushing and necrosis of the periodontal membrane, due to the heavy occlusal force exerted upon them. Besides, extensive root resorption was found (see Fig. 314). These histological findings indicate that "jumping the bite" has in this case caused undue stress upon the supporting teeth and produced traumatic destruction of the tissues.



FIG. 319.—Crest of interdental septum in case of orthodontic mesial movement of the lower posterior teeth. The arrow indicates the direction of movement. Monkey. OC, osteoclasts on the distal side of the interdental septum; NF, new-formation of bone on the mesial side of the interdental septum. New spicules arranged in the direction of pull. (Breitner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

The experiments of Breitner offer valuable suggestions. They for the first time have actually demonstrated that intermaxillary forces are able to produce changes in the mandibular fossa and in the architecture of the mandible. To what extent these findings are applicable to man is, of course, doubtful; still, clinical observations in case of mesial or distal movement of the mandible indicate that the entire mandible may change its topographic relation to the temporo-mandibular articulation, and that rearrangement and reconstruction of the bone structure of the mandible may eventually take place under the influence of orthodontic forces.

BIBLIOGRAPHY.

- BOEDECKER, C. F.: Critical Review of Gottlieb and Orban's "Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne," *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1932, **18**, 895.
- BREITNER, CARL: Experimentelle Veränderung der Mesiodistalen Beziehungen der oberen und unteren Zahnreihen, *Ztschr. f. Stom.*, 1930, **28**, 134, 620; 1931, **29**, 343.
- CASE, C. S.: *Dental Orthopedia*, Chicago, C. S. Case, 1908, pp. 116-174.
- GOTTLIEB, B., and ORBAN, B.: *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Leipzig, Thieme, 1931.
- GRUBRICH, W.: Veränderungen an orthodontisch bewegten Zähnen, *Korr. f. Zahnärzte*, 1930, **54**, 153.
- GUBLER, WALTER: Zur Frage der orthodontisch verursachten Wurzelresorption, *Schweiz. Mon. f. Zhk.*, 1931, **41**, 1011.
- HERZBERG, B. L.: Bone Changes Incident to Orthodontic Tooth Movement in Man, *Jour. Am. Dent. Assn.*, 1932, **19**, 1777.
- JOHNSON, A. LEROY, APPLETON, J. L., JR., and RITTERSHOFER, L. S.: Tissue Changes Involved in Tooth Movement, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1926, **12**, 889.
- KETCHAM, ALBERT H.: A Progress Report of Apical Root Resorption of Vital Permanent Teeth, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1929, **15**, 310.
- MCCOY, J. D.: *Applied Orthodontia*, Philadelphia, Lea & Febiger, 1931, pp. 152-173.
- MARSHALL, JOHN A.: Studies on Apical Absorption of Permanent Teeth, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1930, **16**, 1, 1035.
- Root Absorption of Permanent Teeth. II. A Study of Bone and Tooth Changes Incident to Experimental Tooth Movement, *Jour. Am. Dent. Assn.*, 1930, **17**, 1221.
- The Relation of Malnutrition to Dental Pathology, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1931, **17**, 527.
- OPPENHEIM, A.: Veränderungen der Gewebe, insbesondere des Knochens bei der Verschiebung der Zähne, *Vrtljschr. f. Zhk.*, 1911, **27**, 302.
- Tissue Changes Particularly of the Bone Incident to Tooth Movement, *Am. Orthodont.*, October, 1911, vol. **3**, No. 2; January, 1912, vol. **3**, No. 3.
- Die Veränderungen der Gewebe während der Retention, *Verh. d. Europ. Ges. f. Orthod.*, 1912.
- Über Wurzelresorptionen bei orthodontischen Massnahmen, *Ztschr. f. Stom.*, 1929, **27**, 605.
- Bone Changes During Tooth Movement, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1930, **16**, 535.
- OPPENHEIM, A., and GRÜNBERG, J.: *Orthodontische Therapie*, *Fortschr. d. Zhk.*, 1928, **4**, 827.
- SCHWARZ, A. M.: Über die Bewegung belasteter Zähne, *Ztschr. f. Stom.*, 1928, **26**, 40.
- Ein weiterer Beitrag zur Frage der Bewegung belasteter Zähne, *Ztschr. f. Stom.*, 1929, **27**, 499.
- Movement of Teeth under Traumatic Stress, *Dent. Items Int.*, 1930, **52**, 96.
- Die Gewebsveränderungen bei orthodontischen Massnahmen, *Fortschr. der Orthodontik*, 1931, No. 3, 381; No. 4, 539; 1932, No. 1, 11.
- Tissue Changes Incidental to Orthodontic Tooth Movement, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1932, **18**, 331.
- Die biologischen Grundlagen der orthodontischen Therapie nebst Folgerungen für die Praxis, *Ztschr. f. Stom.*, 1932, **30**, 1041.
- WALKHOFF, O.: Beiträge zur Theorie und Praxis der orthodontischen Massnahmen und deren Beziehung zur Entwicklungsmechanik der Kiefer, *Deutsche Zhk.*, 1927, vol. **69**.

CHAPTER XV.

IMPACTED TEETH—DENTIGEROUS CYSTS—MEDIAN ANTERIOR MAXILLARY CYSTS.

IMPACTED TEETH.

IMPACTED teeth are teeth that have failed to erupt because their position within the jaw made eruption impossible, or because some mechanical or systemic condition interfered with their normal eruption.

1. **Clinical Considerations Concerning Impaction.**—For the purpose of clinical diagnosis, it seems advisable to differentiate between impacted teeth that are completely submerged and separated from the surface of the jaw by an unbroken, healthy layer of soft tissue and bone, and impacted teeth, the crowns of which are in communication with the oral cavity. The latter form of impaction, with partially erupted crowns, frequently produces a chronic inflammation of the surrounding tissues; this is especially true for lower third molars, which almost invariably show evidence of infection and inflammation of the investing soft tissues if a part of the crown is exposed to the oral cavity.

Completely embedded teeth present a different condition and are nothing but a continuation of what is normally found in the jaws of children. The teeth are formed in the jaw as usual, but instead of erupting and taking their places in the arch, they remain within their bony bed. The tissues overlying such completely impacted teeth are as normal and healthy as are the tissues overlying the unerupted teeth in a child's jaw.

The lower third molars are most often impacted; next in frequency are the upper cuspids, then the bicuspid, and the supernumerary teeth. Impactions have been observed in practically every tooth of the permanent dentition, but the above-mentioned teeth contribute by far the majority of complete and partial impactions. In some cases, symmetrical impaction is found on both sides of the same jaw; both upper cuspids or even both upper and both lower cuspids may be embedded in the jaw. Another rather common occurrence is the partial or complete impaction of both lower third molars of the same patient.

The causes for impaction, although manifold, may be divided into local factors and general or systemic conditions. Among the local causes, lack of space in crowded arches is of main importance. Certain teeth are more likely to be affected by lack of space than others. The upper cuspid, for example, develops in a much higher level than the other teeth of the upper jaw, its germ lying above the root ends of the incisors and bicuspid, high up in the angle between nasal cavity, maxillary sinus, and anterior plane of the maxilla. A minute rotation or displacement of the cuspid germ by the forces in the growing jaws may be sufficient to deviate the tip of the cuspid forward instead of downward. Then the regular descent of the cuspid crown between lateral incisor and first bicuspid is impossible, and, as a result, the cuspid assumes a more or less

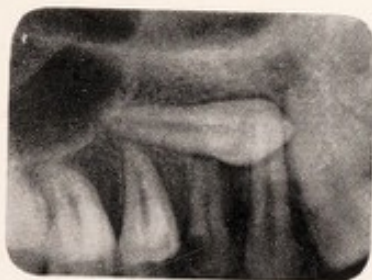


FIG. 320.



FIG. 321.



FIG. 322.

FIG. 320.—Radiograph of an impacted upper cuspid. The tooth is lying horizontally in the maxilla, the tip of the crown is near the root of the central incisor, the apex near the roots of the bicuspids. The deciduous cuspid is still in place. (Ennis.)

FIG. 321.—Radiograph of an impacted lower third molar. The third molar lies in a horizontal position, its crown impinging upon the distal surface of the second molar, its roots extending into the ramus. (Ennis.)

FIG. 322.—Radiograph of an impacted lower bicuspid. (Ennis.)

horizontal position in the maxilla, its crown being located above or behind the roots of the incisors, its root above the bicuspids (Fig. 320).

Another common cause for impaction of the upper cuspid is the premature loss of the deciduous cuspid. In this case, the lateral incisor and the first bicuspid will invariably shift toward each other, reducing the space for the permanent cuspid, and the latter will have no chance to assume its place; it will either remain in the jaw or erupt in an abnormal position.

Topographical reasons also seem to be responsible in a majority of cases for the impaction of the lower third molar. There seems to be a tendency in the human race toward reduction in the size and length of the mandible. In studying the mandibles of primitive races, one finds, as a rule, that the lower third molar stands in line

with the other molars and that there is still some space left between the distal surface of this tooth and the anterior margin of the ramus. Impaction of the third molar is extremely rare in the jaws of primitive people. In mandibles of modern civilized races, on the contrary, very often there is insufficient space between the mandibular second molar and the ramus to allow a third molar to erupt; frequently, the distal part of the third molar is embedded in the ramus or its roots are embedded in the angle of the mandible or in the ramus, resulting in a forward-tipped or even horizontal position of the crown. Lower molars are normally tipped mesially before eruption, a fact which can be plainly seen in any radiograph of the permanent molars in the mandible of a child. Only if the mandible grows sufficiently in the mesio-distal dimension to allow the third molar to straighten and erupt in line with the other molars, are normal conditions possible; otherwise, the third molar will be crowded behind the second molar, its mesial cusps being locked against the second molar. As the eruptive movement of the third molar continues, its forward inclination will be increased instead of being reduced, and, finally, the entire crown of the third molar will impinge upon the second molar (Fig. 321).¹

The lower bicuspid are another group of teeth likely to be impacted. Their germs develop in rather spacious cavities in the body of the mandible, and under normal circumstances they are not infrequently slightly tipped, the cusps pointing toward the mesial side. It is not hard to conceive that some form of outer interference, such as displacement, extraction, or disease of the overlying deciduous molars, may cause displacement of the bicuspid germ and subsequent development in a horizontal position (Fig. 322). Another frequent cause for impaction of lower bicuspid is the premature loss of a deciduous molar. If nothing is done to retain the space formerly occupied by the crown of the deciduous molar, the two teeth that border the space will migrate toward each other until they come almost or entirely in contact. As a result, the corresponding bicuspid cannot erupt although it has a normal vertical position in the jaw; its crown remains wedged between the two neighboring teeth.

Heredity apparently plays a certain rôle in the occurrence of impacted teeth: the same type of impaction can sometimes be

¹ The various forms of impaction of the lower third molar and the operative procedures used for the surgical removal of this tooth have been described by George B. Winter in his book, *Principles of Exodontia as Applied to the Impacted Mandibular Third Molar*. Winter illustrates by means of a large collection of radiographs and drawings all the possible anomalies in the position of the impacted lower third molar.

observed in parents and children, or among several children of the same family.

Besides these more common forms of impaction, unusual cases of displacement or impaction are frequently recorded in dental literature. Upper teeth have been found partially erupted into the nasal cavity or into the maxillary sinus. Impacted lower third molars have been found in the ramus or in the condyloid process of the mandible; impacted lower molars or bicuspid have been reported lying in the jaw with their long axes pointing bucco-lingually, with occasional eruption of the crown into the vestibulum or toward the floor of the oral cavity. All these conditions apparently are due to a displacement or rotation of the tooth germ, followed by development in the wrong direction. Mechanical causes besides the ones already mentioned that may produce impaction are: tumor (odontoma, adamantinoma), overlying crown, cyst formation around the crown (see Fig. 338), supernumerary teeth that interfere with normal eruption (Fig. 324), and inflammatory processes near the germ.

Impacted teeth usually have a more or less pronounced tendency to erupt and assume their natural position in the arch, a tendency which is indicated by the presence of an eruptive force in the direction of the long axis of the tooth. Due to this eruptive tendency, impacted teeth may cause periodic pain and resorption of the roots of other teeth that lie in the path of the crown (see Figs. 207 and 331). If by some dental operation normal eruption is made possible, the impacted tooth will usually take advantage of the new conditions and will erupt. For instance, a lower second bicuspid that has become impacted due to lack of space between first bicuspid and first molar will frequently erupt and assume its normal position in the arch after sufficient space has been created by means of orthodontic appliances. In many cases it is possible to cause completely impacted teeth to erupt and to come into occlusion by means of an orthodontic appliance that exerts a gentle but constant force in the desired direction upon the impacted tooth. For this purpose, the crown of the embedded tooth is exposed surgically; a small hole is drilled into the enamel and a hook is cemented into this hole. Then the tissues are allowed to heal over the tooth. By the delicate but constant force of an orthodontic rubber band or a spring anchored to a neighboring tooth or to a tooth in the opposite jaw, the impacted tooth is gradually raised from its bed, and finally assumes a normal position in the jaw. It is interesting to consider the tissue changes occurring during such treatment, the amount of bone that has to be resorbed and rebuilt

to make such movement possible, the rearrangement of the blood-vessels and nerves at the apex, the transformation of an atrophic narrow periodontal membrane into a wide, fibrous one (see page 344), and the development of a "functioning" alveolus (see page 346).

One of the causes that may stimulate an impacted tooth to erupt, even in advanced age, is the irritation and pressure exerted by a denture. Many a patient, who supposedly had had his last tooth extracted many years before, has observed, to his great surprise, a tooth erupt under his denture. Usually this tooth is a cuspid or bicuspid that had been impacted and had been lying in the jaw for many decades until the pressure of the denture and the atrophy of the overlying alveolar ridges exposed its crown. The common belief among the laity in a "third dentition" apparently is based upon observations of this kind.

Certain systemic conditions are frequently accompanied by multiple impactions of teeth. Diseases of the endocrine glands, especially of the thyroid gland (cretinism, myxedema), cause considerable delay in eruption and sometimes almost all of the teeth remain within the jaw. Another condition that belongs in this group is the so-called cleidocranial dysostosis (Hesse, Payne, Zilkens). This is a hereditary, congenital disturbance of bone development of unknown etiology, characterized by underdevelopment or absence of the clavicles and by complete or almost complete failure of the teeth to erupt. Radiographs reveal the presence of the teeth in the jaws; they are usually well-developed and in an upright position, but the stimulus for eruption is missing. Therefore, it is evident that multiple impactions due to systemic conditions are decidedly different from retentions due to mechanical obstacles, for in the latter eruptive tendencies are present and will bring about eruption after the obstacles have been removed, while in the former the teeth will remain impacted although no mechanical obstructions interfere with eruption.

Supernumerary teeth are frequently impacted. In the region of the upper central incisors, the radiographic examination sometimes reveals the presence of small peg-shaped, impacted, supernumerary teeth that may cause a diastema of the upper central incisors (Fig. 323). Supernumerary teeth may be observed in any part of the dental arch. In some instances, these teeth seem to be just small peg-shaped structures which are apparently the result of an atypical overactivity of the dental lamina. In other cases, it seems that their development might be some form of atavism. It must be kept in mind that the two incisors and two premolars

of modern man are the result of a reduction from the three incisors and three premolars in each half of the jaw of the early ancestors of present-day mammals. Bolk called the peg-shaped supernumerary tooth that is found in the upper jaw in or near the median line "mesiodens." In some jaws one mesiodens is present, in others, two; they may remain impacted and be visible only in the radiograph, or they may erupt between or behind the upper incisors. Their presence must be considered a true atavism, namely, a recurrence of the third incisor that has been eliminated early in the development of the human race.



FIG. 323.



FIG. 324.



FIG. 325.

FIGS. 323, 324 and 325.—Radiographs of impacted supernumerary teeth.

FIG. 323.—Peg-shaped supernumerary tooth between the upper central incisors ("mesiodens" of Bolk). The axis of supernumerary tooth lies in a labio-lingual direction. The surrounding tissues are intact.

FIG. 324.—Supernumerary tooth in the cuspid region interfering with the eruption of the permanent cuspid.

FIG. 325.—Supernumerary tooth (third bicuspid) in the region of the lower second bicuspid. The bicuspid are erupted and in normal occlusion. Of the supernumerary tooth, the crown only is formed.

Fig. 325 shows a radiograph of the mandible of a normally developed girl, aged nineteen years, who, in addition to the thirty-two teeth of the second dentition, had the following supernumerary teeth: two lower third bicuspid, both located below and between the second bicuspid and the first molar, two supernumerary upper incisors, one of which had erupted, both lying between the central incisors, and two upper fourth molars, both lying occlusally to the still unerupted upper third molars.

2. Microscopic Examination of Impacted Teeth With Their Surrounding Tissues.—The only way to obtain positive information about the relationship between impacted teeth and their surrounding structures is by sectioning human jaws that contain such teeth. A few specimens will be described here.

Fig. 326 shows a mesio-distal section through the right maxilla of a young adult. The first bicuspid has erupted in normal alignment, but the right upper cuspid is completely impacted and lies

in the maxilla at an angle of about 45 degrees, with the crown pointing downward against the lateral incisor and the apex in a bony ridge on the floor of the maxillary sinus. The crown of the cuspid is surrounded by a layer of fibrous connective tissue. The alveolar ridge over the impacted cuspid is covered by bone and by normal mucosa; the deciduous cuspid has been lost. Special attention

should be called to the presence of a rather wide space filled with connective tissue between enamel and alveolar bone, a space which appears in the radiograph as a dark area surrounding the crown of the impacted tooth. The distance between root surface and bone, however, is very small. In Figs. 327 and 328 different areas from the surface of this impacted cuspid are reproduced under higher magnification. Fig. 327 corresponds to area I in Fig. 326. The periodontal membrane is thin and atrophic, characteristic of non-functioning teeth (see page 344). Its actual width is about 0.06 to 0.08 mm. Fig. 328 illustrates area II of Fig. 326, on the surface of the crown. The enamel is in organic connection with the enamel epithelium, a condition always found in teeth before eruption (see page 260). The distance between enamel surface and alveolar bone is about ten times the distance between cementum and bone in Fig. 327. Dense, fibrous connective tissue, which is arranged

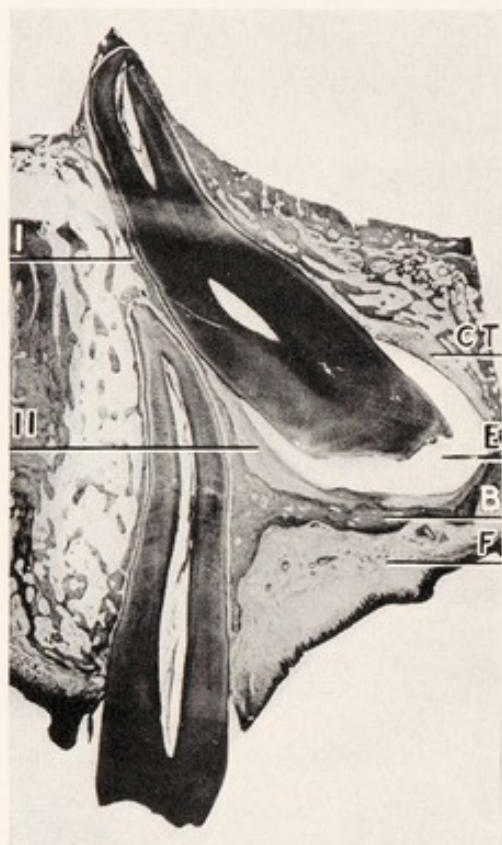


FIG. 326.—Mesio-distal section through an impacted upper cuspid and an erupted first bicuspid. E, enamel of impacted cuspid; CT, connective tissue capsule surrounding the crown of the cuspid; B, bone covering the crown; F, fibrous tissue of the edentulous ridge overlying the impacted cuspid. Higher magnifications of areas I and II will be shown in Figs. 327 and 328. (Gottlieb, Fortschr. d. Zhk., courtesy of Georg Thieme, Leipzig.)

parallel to the surface of the crown, does not show any evidence of inflammation.

The presence of the dark space found in the radiograph around the crown of impacted teeth has in the past often led to diagnostic misinterpretations. This dark area has been spoken of as an "area of infection" (inflammatory bone destruction) or as a cystic

formation. This incorrect diagnosis was due mainly to the lack of proper material for a scientific investigation of impacted teeth. We know now that an impacted tooth, as long as it is completely surrounded by bone and does not come in contact with the oral cavity, is not infected nor any more dangerous or alarming than any unerupted tooth germ in a child. Its enamel surface is united with the enamel epithelium, and the latter is united with the surrounding connective tissue capsule and the bone; in other words, normal tissue is present all around the impacted tooth. Only if the impacted tooth reaches the oral mucosa and is partially erupted can niches for the retention of débris and bacteria develop and

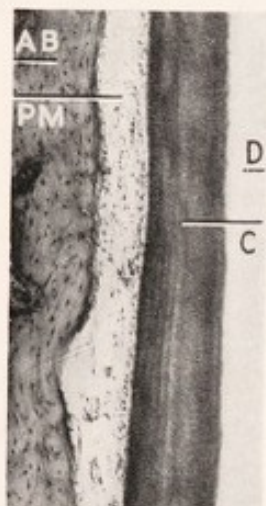


FIG. 327.

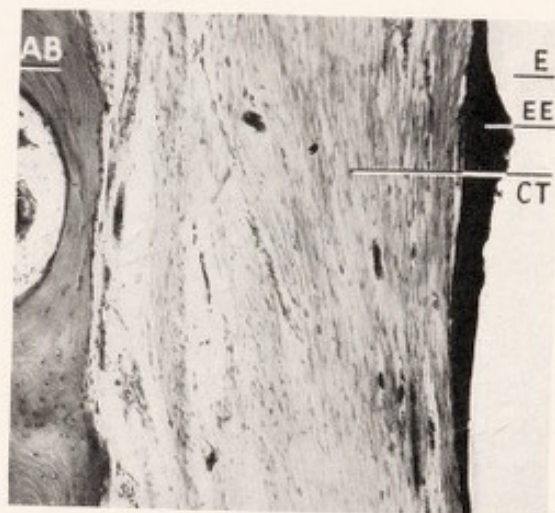


FIG. 328.

FIG. 327.—High magnification of area I in Fig. 326. AB, alveolar bone; PM, periodontal membrane; C, cementum; D, dentin. The periodontal membrane consists of loose connective tissue without definite arrangement (lack of functional stimulus, see Fig. 281). Average width of periodontal membrane, 0.06 mm.

FIG. 328.—High magnification of area II in Fig. 326. AB, alveolar bone; CT, fibrous connective tissue encapsulating the crown; EE, enamel epithelium; E, enamel. Distance between enamel and alveolar bone, 0.7 mm.

cause trouble, such as the well-known inflammations around partially erupted lower third molars. But the completely embedded tooth is not accessible to infection. There may be a possibility of a completely impacted tooth causing pain by impinging upon another tooth or upon a nerve during its attempts to erupt. These symptoms, however, are due merely to mechanical pressure and have nothing to do with infection.

A complete horizontal impaction of a lower bicuspid in the edentulous mandible of an old person is illustrated in Fig. 329. The well-developed bicuspid lies almost horizontally in the body of the mandible, the crown being located slightly higher than the

root but still separated from the surface of the jaw by a bony plate. The enamel is surrounded by the typical layer of connective tissue,

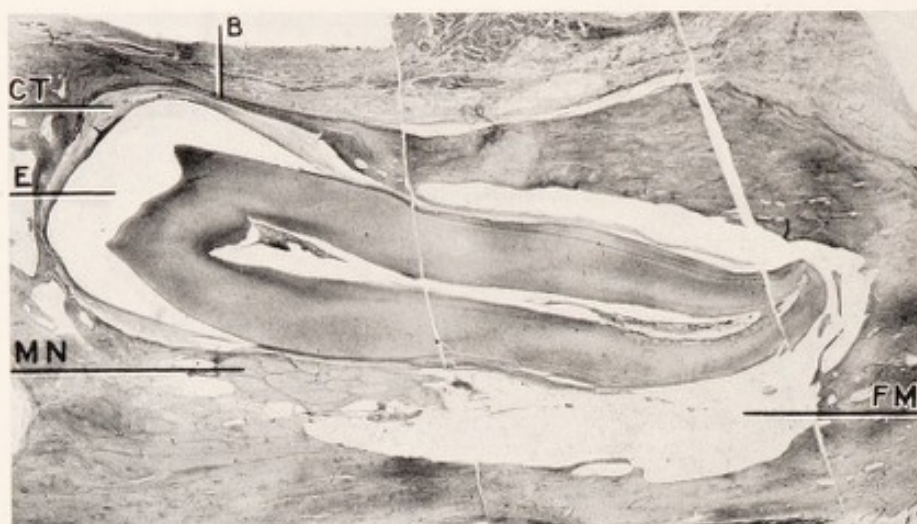


FIG. 329.—Impacted lower bicuspid lying horizontally in an edentulous mandible. E, enamel of bicuspid; CT, connective tissue surrounding the crown; B, bone covering the crown; MN, mandibular nerve located below the impacted tooth; FM, fat marrow. (Kellner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

underneath which the mandibular nerve can be seen. The root is surrounded by a very delicate lamella of bone which in some areas is completely missing. In these areas the root surface of the im-

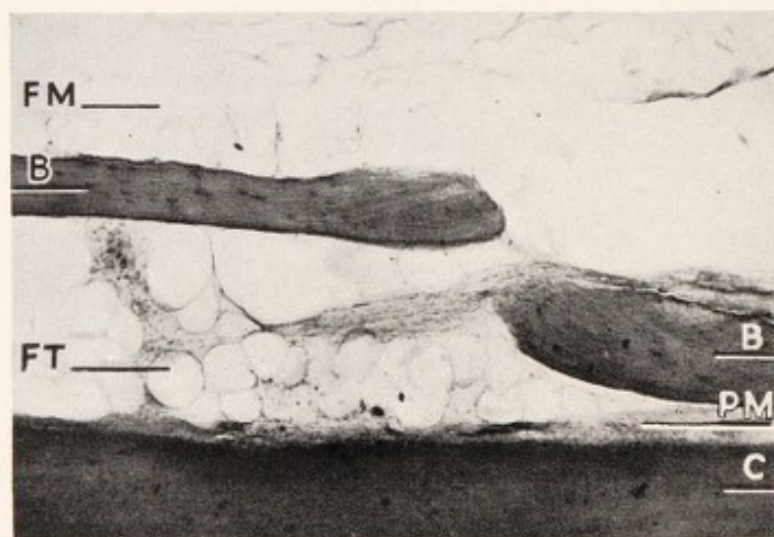


FIG. 330.—Higher magnification of the root surface of the impacted lower bicuspid in Fig. 329. C, cementum; PM, thin atrophic periodontal membrane; FT, fat tissue adjacent to the root surface; B, bone; FM, fat marrow of the mandible. (See Fig. 282.)

pacted tooth is in direct contact with the fat marrow that fills the body of the mandible (Fig. 330). The root is covered by a rather

heavy layer of cementum, indicating the relative independence of cementum formation from functional stimuli (see pages 222 and 346).

A third case of complete impaction concerns an upper cuspid (Fig. 331). This tooth lies in an almost vertical position. Its crown impinges upon the distal surface of the root of the lateral incisor. The microscopic examination of this incisor gives interesting information about the eruptive movement of the impacted cuspid. A higher magnification of area I, the area of contact

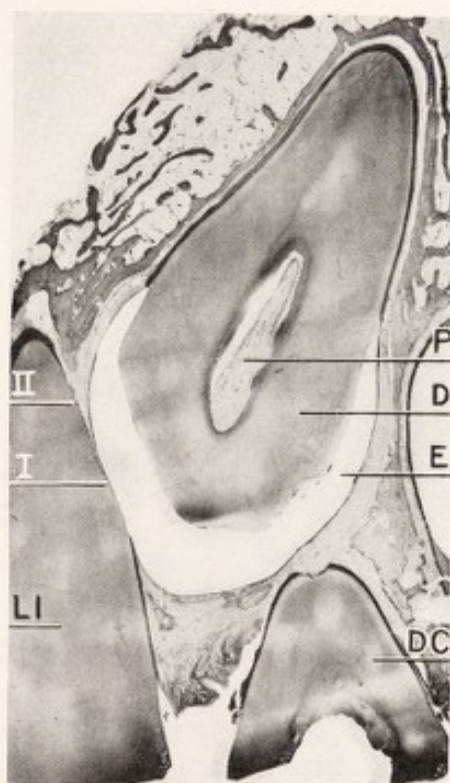


FIG. 331.—Impacted upper cuspid in contact with the root of the lateral incisor. E, enamel of impacted cuspid; D, dentin of impacted cuspid; P, pulp of impacted cuspid; LI, root of lateral incisor; DC, deciduous cuspid. Higher magnifications of the areas I and II are shown in Figs. 332 and 333. (Kotanyi, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

between incisor and cuspid, reveals the presence of active resorption of the root of the incisor (Fig. 332). Further apically, in area II, similar resorption of the dentin can be seen (Fig. 333); however, this resorption is being repaired by deposition of cementum, indicating that at the time of the individual's death there was no more pressure in this field. From this distribution of active and repaired resorption, it can be concluded that the cuspid moved from II to I, sliding down along the distal surface of the root of the lateral incisor in the direction of the surface of the jaw (Kotanyi). The deciduous

cuspid is beginning to show evidence of resorption by the approaching permanent successor.

Two kinds of changes must still be discussed in connection with the histology of impaction, namely, the deposition of cementum upon the enamel, and the resorptive processes on both crown and root of impacted teeth.

The deposition of cementum is the result of the loss of the enamel epithelium that normally covers the crown of every unerupted tooth. As long as the enamel epithelium around the crown of the impacted tooth is intact, the enamel does not come in direct contact

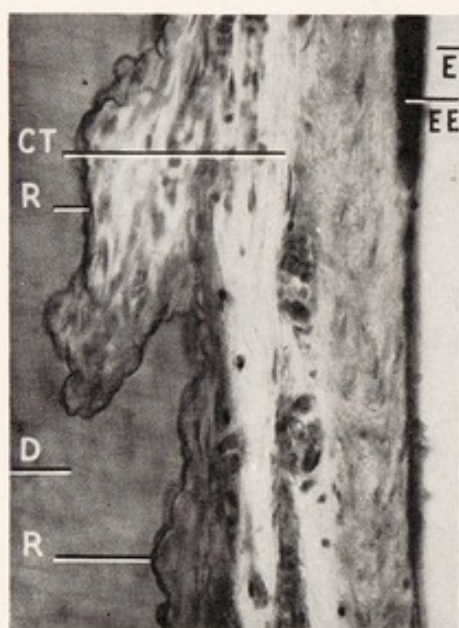


FIG. 332.

FIG. 332.—High magnification of area I in Fig. 331. E, enamel of the impacted cuspid; EE, enamel epithelium of the impacted cuspid; CT, connective tissue capsule surrounding the crown of the cuspid; R, resorption of the root of the lateral incisor; D, dentin of lateral incisor.

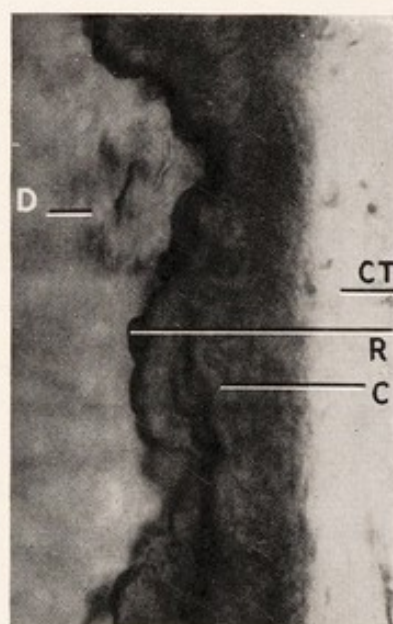


FIG. 333.

FIG. 333.—High magnification of area II in Fig. 331. CT, connective tissue; R, line of resorption in the dentin, D; C, reparative deposit of cementum upon the resorbed dentin surface.

with the surrounding connective tissue. If, however, the impaction persists over a long period of time, the enamel epithelium may gradually degenerate and disappear. Then the enamel surface comes in contact with the connective tissue, and a deposition of cementum can occur directly upon the enamel. The latter condition is shown in Fig. 334, which was taken of an impacted third molar. Starting at the cemento-enamel junction, a layer of cementum extends upon the enamel. A (primary) enamel cuticle is visible on the inner surface of the cementum.

Another possible change that may occur in impacted teeth is the

resorption of the dental hard tissues. If such resorptive processes take place, they give the tooth a peculiar characteristic appearance in the radiograph: instead of having a clear, dense outline, the impacted tooth appears uneven and sometimes large portions of root or crown may be entirely missing and may be replaced by bone. When such a tooth is removed and the specimen is examined microscopically, it has a rough surface and has defects that may look like caries. However, inasmuch as caries is possible only where the fluids of the oral cavity have access, such defects in completely impacted teeth can never be the result of caries but of resorption; this also is invariably confirmed by subsequent microscopic examination (see also page 243).



FIG. 334.—Deposition of cementum upon the enamel of an impacted lower third molar. D, dentin; E, enamel; CEJ, cemento-enamel junction; Cu, enamel cuticle; C, cementum on the surface of the enamel; CT, connective tissue.

In the chapter on Resorption, an impacted tooth of a dog that was penetrated by numerous duct-like resorptions was illustrated. On the resorbed surface of the crown, bone had been deposited, causing a solid junction with the alveolus (Fig. 205). Fig. 335 is a specimen of an impacted human upper cuspid, the crown of which has been rather extensively resorbed.¹ As is usual in these

¹ The term "decalcification," which is frequently used in the clinical description of resorptive processes on impacted and supernumerary teeth, is decidedly incorrect. The only known process of true decalcification is the chemical process that occurs in caries, or when a tooth is submerged in acid until all calcium salts are dissolved and only organic matrix is left. Then the tooth is really "decalcified." In a tooth that is embedded in the jaw, no such change is possible. The only way such a tooth can change its configuration and appearance is through the activity of osteoclasts; this resorption of the hard tissues is usually accompanied by an ingrowth of granulation tissue or spongy bone.

The above statement holds true also for the resorptive processes on the roots of deciduous teeth. Deciduous teeth are not "decalcified," as erroneously stated by some authors, but are resorbed.

cases, resorption and new formation occur in close sequence; bone has been deposited on the resorbed surface of the dentin, and the spaces inside of the crown are occupied by fat marrow. It is very interesting to note that the pulp has reacted to the advancing resorption in the same way in which it reacts to abrasion or caries, namely, by extensive formation of secondary dentin.

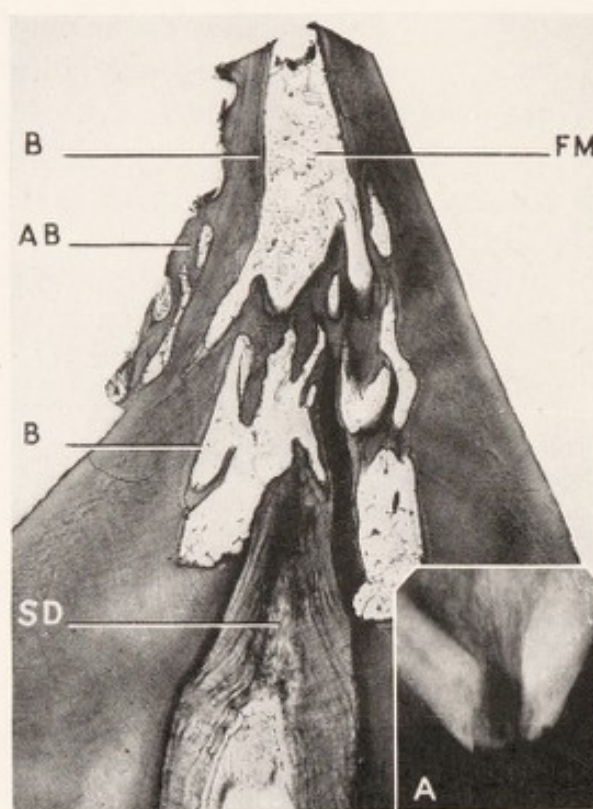


FIG. 335.—Resorption of the crown of an impacted upper cuspid. A: Radiograph of two impacted cusps in an edentulous maxilla. Both cusps show evidence of resorption of the crown. The histologic specimen is the left cuspid. FM, fat marrow occupying the spaces created by dentin resorption; B, deposits of bone upon the resorbed dentin; AB, alveolar bone united with the crown; SD, secondary dentin in the tip of the pulp chamber.

DENTIGEROUS CYSTS.

Dentigerous cysts are cystic cavities within the jaws that contain the crowns of one or several teeth. These cysts develop by an accumulation of fluid between the enamel surface and the surrounding soft tissues. Normally the enamel surface is in organic junction with the enamel epithelium, but if this junction is severed by some interference, fluid will be discharged in the resulting space around the crown; subsequently the surrounding tissues will be distended and form a cystic sac. As the soft tissue investing the unerupted tooth is called the tooth follicle, dentigerous cysts are often spoken

of as follicular cysts. The crown rises freely into the cyst sac that is attached to the tooth at the border between crown and root. The cyst is lined by stratified squamous epithelium derived from the original enamel epithelium; it is filled with a clear, serous, yellowish

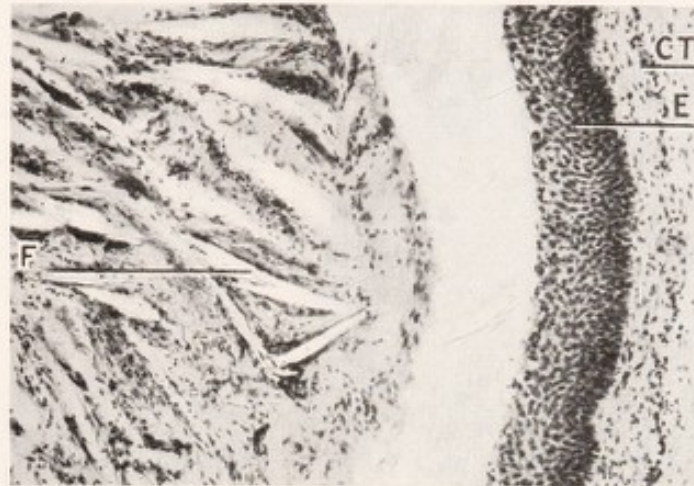


FIG. 336.—Part of the wall of a dentigerous cyst. E, cyst epithelium; CT, connective tissue of the cyst wall; F, coagulated fibrin, wandering cells and cholesterol crystals in the cyst cavity.

fluid frequently containing crystals of cholesterol (Fig. 336). The tooth around which the cyst formed may present various stages of development: if the cyst develops early in life the crown only may be present, while in other cases a part of the root or the entire root may have developed.

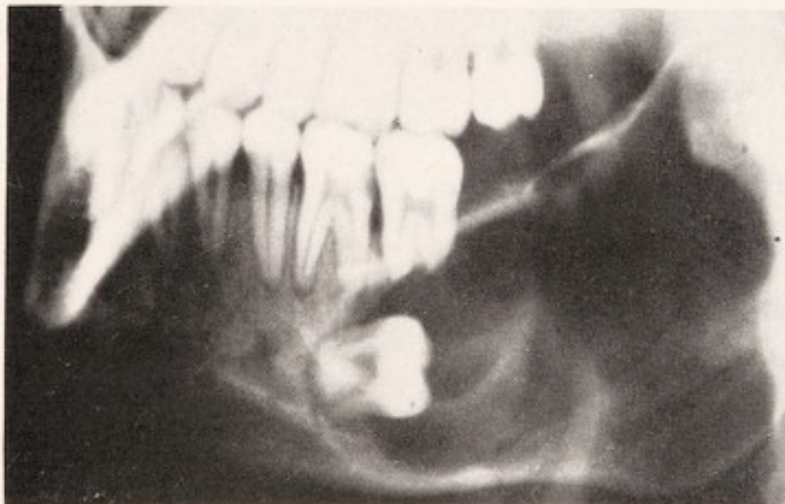


FIG. 337.—Radiograph of a large dentigerous cyst involving angle and ramus of the mandible. The cyst developed from the lower third molar which is displaced below the roots of the first and second molar.

The bone around a dentigerous cyst is resorbed under the slow but steady pressure of the cyst sac; thus large areas of bone destruction may result (Figs. 337 and 338). A typical dentigerous cyst

grows slowly without causing any pain or discomfort; its presence is usually discovered either by the absence of a tooth or by a swelling on the outer surface of the jaw.

As to the etiology of dentigerous cysts, there is by no means a uniformity of opinion among the investigators. Inflammation plays

a rôle in a number of cases, especially in those teeth (bicus-pids) in which the corresponding deciduous teeth had decomposed, infected pulps. Here irritation of the tissue overlying the permanent crown appears

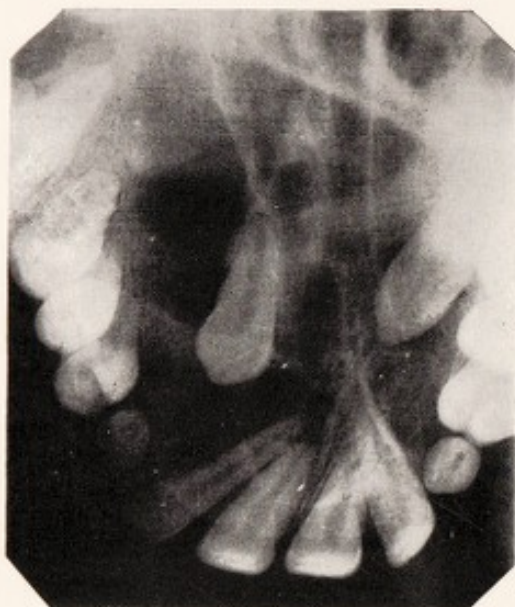


FIG. 338.—Radiograph of a large dentigerous cyst which developed around the crown of an impacted upper left cuspid. (Ennis.)



FIG. 339.—Radiograph of a dentigerous cyst which developed around the crown of an impacted lower bicuspid. (Ennis.)

to be the cause of cyst formation. In other instances, however, as in the case illustrated in Fig. 337, it is difficult to believe in an inflammatory etiology of the dentigerous cyst. It, therefore, must be assumed that the cystic degeneration of the follicle is caused by a congenital malformation. Dentigerous cysts are more frequently observed in children and adolescents than in adults.

The treatment of dentigerous cysts consists of opening the cyst and keeping it open so as to relieve the pressure permanently and to allow the bone to regenerate from below. The removal of the unerupted tooth is sometimes indicated (Fig. 337); in other cases, in which there is a possibility of the tooth's assuming its normal position in the arch, the tooth is left untouched at the bottom of the cyst; it usually erupts after the pressure of the cyst fluid has been removed.

MEDIAN ANTERIOR MAXILLARY CYSTS.

The maxilla is sometimes the site of cysts located in the median line in the anterior portion of the incisive canal. These cysts appear

clinically as smooth, round sacs with a thin epithelial lining and contain a clear, sterile fluid. Their size varies from that of a small pea to that of a cherry or even larger. The clinical diagnosis of this condition is made by the radiograph; since maxillary cysts rarely cause any definite symptoms, their discovery is usually made during routine radiographic examinations of the central incisor region. Only seldom do median cysts reach such a size that they cause bulging of the soft tissues in the median line behind the central incisors.

From the viewpoint of diagnosis, the dentist should be familiar with the existence of such cysts because this knowledge may eventually prevent embarrassing diagnostic errors. In a radiograph of the anterior part of the maxilla, the shadow of a median cyst can easily overlap the root end of a central incisor and look like a root cyst at the apex of the incisor, although in reality the cystic formation has no connection whatsoever with the teeth (Figs. 340 and 341).



FIG. 340.



FIG. 341.

FIGS. 340 and 341.—Radiographs of median anterior maxillary cysts.

FIG. 340.—Large median cyst with very definite outline of the area of bone destruction.

FIG. 341.—Large cystic cavity in the median line causing bulging of the tissues of the incisive papilla. Considerable destruction of the interdental bone between the central incisors. Except for the right central incisor in Fig. 340, all central incisors have intact vital pulps.

The etiology of median anterior maxillary cysts can be understood only in connection with the early development of the palate. In a fetus of less than about 24 mm. total length, the oral cavity is in open communication with the nasal cavity, no palate having yet been formed. Subsequently the palate develops by a junction of three different parts: the median primary palate and two lateral secondary palatal plates. The primary palate grows downward from the median nasal process corresponding to the later premaxilla; the secondary palatal plates develop in horizontal direction from both sides toward the median line, forming what later becomes the

hard and the soft palate. When these three parts unite, they form a suture the shape of a Y, the two forks of the Y pointing forward and upward from the median line of the mouth into the nose, and the lower part of the Y being represented by the median suture of the hard and soft palate back of the incisive foramen. The junction point of the forks of this Y corresponds, in the adult, to the oral opening of the incisive canal.

The junction of the different parts of the palate occurs in embryonic life when the total length of the fetus is between 23 and 28 mm. If this junction fails to take place, either partly or completely, various degrees of cleft palate and harelip may result. When the parts of the forming palate come in contact with one another, the epithelial covering of

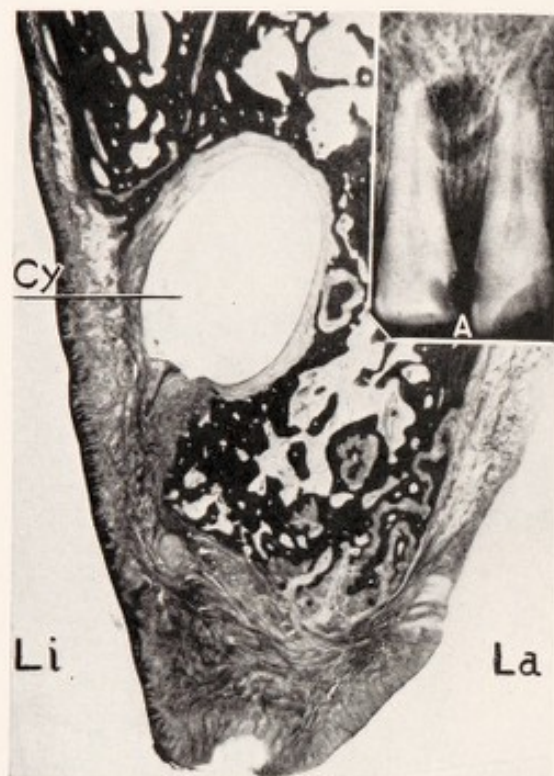


FIG. 342.—Median anterior maxillary cyst in a jaw specimen. A: Radiograph of the specimen showing a round, sharply outlined bone defect between the central incisors. Labio-lingual section through the anterior portion of the maxilla in the median line. La, labial side; Li, lingual side; Cy, cyst of about 6 to 8 mm. in diameter lying near the lower opening of the incisive canal. (Kronfeld, *Korr. f. Zahnärzte.*)

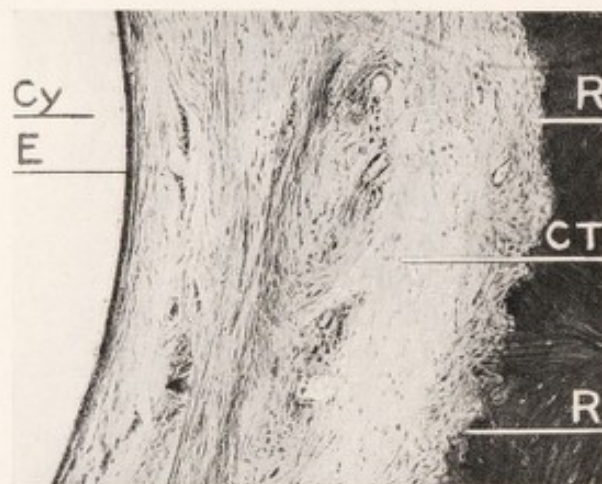


FIG. 343.—Higher magnification of the cyst wall in Fig. 342. Cy, cyst cavity; E, epithelial lining; CT, connective tissue surrounding the cyst; R, resorption of the bone. (Kronfeld, *Korr. f. Zahnärzte.*)

these parts must disappear to make a solid junction possible. Sometimes during this process small islands of epithelium are enclosed between the joining parts. Hence these epithelial remnants are frequently found in children and even in adults along the original fetal Y-shaped line of closure, namely, along the ductus nasopalatini in the incisive canal. Such epithelial islands may sometimes show cystic degeneration, and if they do, they give rise to median anterior maxillary cysts.

The following is a typical case of a median maxillary cyst that was published by the author in 1928. The specimen under consideration was the maxilla of a man, aged thirty years, who died of tuberculosis. A radiograph of the central incisors, both of which had intact vital pulps, showed a round, sharply outlined area of bone destruction in the median line between the central incisors. The bone defect was about 8 mm. in diameter and had a punched-out appearance (Fig. 342). The specimen was sectioned in labio-lingual direction. In the area immediately behind the central incisors there was an oblong cyst about 8 mm. in length and 6 mm. in width, occupying the oral portion of the incisive canal and replac-

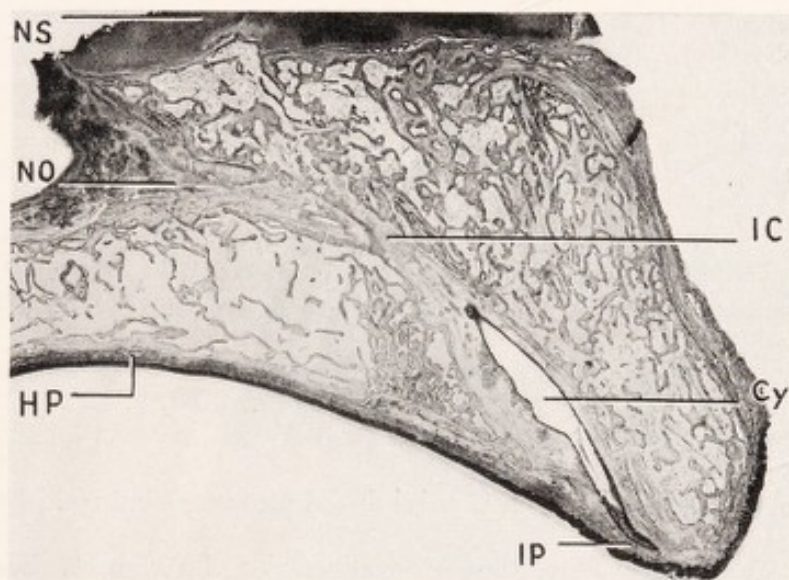


FIG. 344.—Labio-lingual section through the anterior portion of an edentulous mandible and through the incisive canal. IP, incisive papilla; HP, hard palate; IC, incisive canal; Cy, oblong cyst lying close to the oral opening of the incisive canal; NO, nasal opening of the incisive canal; NS, nasal septum (cartilage).

ing the bone in this region. The wall of the cyst consists of several layers of stratified squamous epithelium (Fig. 343); a stratum of loose connective tissue of uniform thickness occupies the space between cyst and surrounding bone. The bone itself in many places shows evidence of resorption. The cyst apparently was growing at the time of the death of the patient, and the resulting increase in pressure caused bone resorption. At the lower pole of the cyst, the epithelial lining is continuous with a solid mass or strand of epithelium that extends downward in the direction of the incisive papilla. The cyst in this case had evidently developed from this solid epithelial remnant. No evidence of inflammation could be found in the specimen.

This observation caused the author to search for similar cystic formations in the region of the incisive canal, and he has since been successful in finding another case in an edentulous human maxilla. In this case a large mass of stratified squamous epithelium lay in the incisive canal of an edentulous jaw specimen (Fig. 344). The epithelium was solid except for the central part where cystic degeneration of the epithelial masses had taken place. Although it cannot be predicted whether actual cyst formation with dilatation of the incisive canal would have occurred later in this case, it seems very probable that, in time, a cyst similar to the one in the first case would have developed. Thus, this latter finding may be considered as a stage preliminary to that illustrated in Fig. 342.

BIBLIOGRAPHY.

Impacted and Supernumerary Teeth.

- BAUER, W.: Zur Kenntnis der Zementauflagerung am Schmelz retinierter Zähne, *Ztschr. f. Stom.*, 1926, **24**, 229.
- BLOCH-JORGENSEN, K.: Notes on the Retention of the Cuspid of the Upper Jaw, *Dent. Items Int.*, 1931, **53**, 241.
- Observation on the Retention of the Upper Cuspid, *Dent. Items Int.*, 1932, **54**, 692.
- BLUM, THEODOR: Malposed Teeth: Their Classification, Pathology and Treatment, *Int. Jour. Ortho. Oral Surg. and Radiogr.*, 1923, **9**, 122.
- BOLK, L.: Supernumerary Teeth in the Molar Region in Man, *Dental Cosmos*, 1914, **56**, 154.
- Die überzähligen oberen Inzisivi des Menschen, *Deutsch. Mon. f. Zhk.*, 1917, **35**, 185.
- FRIDRICHOVSKY, JAN: Über die Aberration der unteren Weisheitszähne, *Vrtljschr. f. Zhk.*, 1932, **48**, 289.
- HARDGROVE, T. A.: The Impacted Tooth, *Jour. Am. Dent. Assn.*, 1931, **18**, 1287.
- HESSE, G.: Dysostosis cleidocranialis unter besonderer Berücksichtigung des Gebisses, *Vrtljschr. f. Zhk.*, 1925, **41**, 162.
- Weitere Befunde am Zahnsystem dysostotischer Individuen, *Ztschr. f. Stom.*, 1926, **24**, 205.
- KOTANYI, E.: Histologische Befunde an retinierten Zähnen, *Ztschr. f. Stom.*, 1924, **22**, 747.
- Histologische Befunde an einem retinierten Milchmolaren und einem retinierten Weisheitszahn, *Ztschr. f. Stom.*, 1931, **29**, 764.
- LONG, CHARLES J.: Supernumerary Tooth in the Nose, *Dental Cosmos*, 1924, **66**, 360.
- LUKOMSKY, L.: Befunde an einem retinierten Zahn, *Deutsch. Mon. f. Zhk.*, 1931, **49**, 321.
- PAYNE, J. LEWIN: Some Cases of Delayed Eruption of the Teeth, *Dental Record*, 1929, **49**, 464.
- PUTERBAUGH, P. G.: Impacted Teeth, *Bull. Chicago Dent. Soc.*, 1932, vol. **12**, No. 24.
- STAFNE, EDWARD C.: Supernumerary Upper Central Incisors, *Dental Cosmos*, 1931, **73**, 976.
- Supernumerary Teeth, *Dental Cosmos*, 1932, **74**, 653.

- WEBER, RUDOLF: Zur Kasuistik der histologischen Veränderungen an retinieren Zähnen, *Deutsch. Mon. f. Zhk.*, 1925, **43**, 813.
- WINTER, GEORGE B.: Principles of Exodontia as Applied to the Impacted Mandibular Third Molar, Am. Medical Book Company, St. Louis, 1926.
- WORMAN, HAROLD G.: A Histopathologic Study of Impacted Teeth, *Jour. Am. Dent. Assn.*, 1929, **16**, 1885.
- ZILKENS, K.: Zahnbefunde bei zwei Fällen von Dysostosis cleidocranialis, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 477.

Follicular Cysts.

- BAUER, W.: Über cystische Bildungen im Kiefer, *Ztschr. f. Stom.*, 1927, **25**, 205.
- Die Entstehung der Folliculärzysten, *Ztschr. f. Stom.*, 1929, **27**, 1071.
- BLOCH-JORGENSEN, K.: Follicular Cysts, *Dental Cosmos*, 1928, **70**, 708.
- Notes on the So-called Follicular Dental Cysts, *Dent. Items Int.*, 1930, **52**, 192.
- Beobachtungen bezüglich der sogenannten follikulären Zahnzysten, *Ztschr. f. Stom.*, 1930, **28**, 245.
- LARTSCHNEIDER, JOSEF: Die Pathogenese, pathologische Anatomie, Prognose und Therapie der follikulären Zahnzysten, *Ztschr. f. Stom.*, 1929, **27**, 546.
- PUTERBAUGH, P. G., and PIKE, G. C.: Conservative Treatment of Cysts. A Case Report: The Bur, Chicago College of Dental Surgery, 1932, No. 1.
- RYGGE, J.: Über die Pathogenese der follikulären Kieferzysten, *Ztschr. f. Stom.*, 1932, **30**, 811.
- SPRAWSON, E.: Case of Multiple Dentigerous Cysts in the Mandible, and Some Remarks on the Pathology of such Cysts, *Proc. Roy. Soc. Med., Odont. Sect.*, 1922, **15**, 56.

Median Anterior Maxillary Cysts.

- BAUER, W.: Über Zysten im Weichgewebe des Ductus Nasopalatinus, *Ztschr. f. Stom.*, 1930, **28**, 481.
- KRONFELD, R.: Röntgenologisch-histologische Untersuchung einer medianen Oberkieferzyste, *Korr. f. Zahnärzte*, 1928, **52**, 360.
- MEYER, A. W.: Hitherto Unrecognized Median Maxillary Cysts, *Pacific Dent. Gaz.*, April, 1931.
- Median Anterior Maxillary Cysts, *Jour. Am. Dent. Assn.*, 1931, **18**, 1851.
- NEUWIRT, FR., and PARMA, C.: Erkrankungen im Gebiete des Ductus nasopalatinus, *Ztschr. f. Stom.*, 1932, **30**, 1195.
- SCHROFF, JOSEPH: Cyst of Naso-palatine Duct. Cyst of Facial Cleft Area (Fissural Cleft), *Dent. Items Int.*, 1929, **51**, 107.
- Cysts in the Incisor Canal, *Jour. Dent. Res.*, 1930, **10**, 739.
- WERMUTH, HANS: Beitrag zur Histologie der Gegend seitlich von der Papilla palatina, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 203.

CHAPTER XVI.

FRACTURES OF TEETH.

A TOOTH fracture is a break in the continuity of the dental hard tissues, caused by a sudden force. Depending upon its etiology, a fracture may be either spontaneous or traumatic: a spontaneous fracture occurs during mastication; a traumatic fracture is caused by an outer force (blow, kick, or fall on the face).

SPONTANEOUS FRACTURES.

Spontaneous (pathological) fractures usually occur in teeth that have been weakened in some way. A frequent cause of spontaneous fractures is large fillings with insufficient protection of the cusps or incisive edges. The cusps, together with splinters of dentin, chip off rootward to varying extents. A typical fracture of this type is illustrated in Black's *Operative Dentistry* (Vol. II, Fig. 245); in an upper bicuspid with a two- or three-surface filling, a longitudinal fracture split the tooth lengthwise. Similarly, a spontaneous cross-fracture of the crown during mastication is sometimes observed in teeth with deep cervical cavities or fillings. Occasionally spontaneous fractures of parts of the crown occur in intact teeth of old patients with a hard bite and brittle dentin.

The clinical prognosis in this type of fracture depends upon the amount of root involved. If the fracture involves the crown only, or just a small splinter of the root, it is usually possible to restore the tooth by means of an artificial crown, sometimes by inserting a dowel into the root canal. If, however, the fracture extends farther into the root, an artificial restoration is impossible.

TRAUMATIC FRACTURES.

Traumatic fractures of the teeth are common, particularly in young people, because of their great interest in sports and athletics. Various games, boxing, and falls on the mouth are among the common causes for traumatic fractures, especially of the upper and lower incisors.

From the viewpoint of diagnosis and prognosis, it seems advisable

to classify tooth fractures as follows, according to their location and to the extent of tooth structure involved (Gottlieb):

1. Fractures located entirely outside (crownward) of the epithelial attachment (fractures of the clinical crown only).
2. Fractures located partly outside, partly inside the epithelial attachment (combined fractures of crown and root).
3. Fractures located entirely inside (rootward) of the epithelial attachment (fractures of the clinical root).

The first two forms of fractures have to be subdivided into fractures that do not expose the pulp and fractures that do expose the pulp.



FIG. 345.

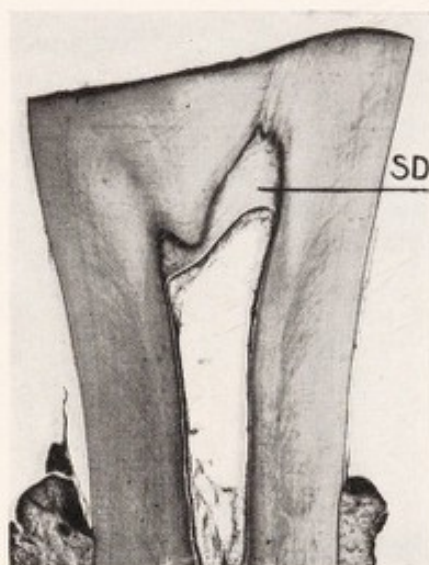


FIG. 346.



FIG. 347.

FIG. 345.—Diagram of a traumatic fracture of an upper incisor. The pulp is not exposed by this fracture.

FIG. 346.—Traumatic fracture on a dog's incisor produced experimentally eight weeks previous to the death of the animal. The pulp was not exposed by this fracture. SD, secondary dentin formation, corresponding to the extent and location of the fracture. (Courtesy of E. D. Coolidge.)

FIG. 347.—Diagram of a traumatic fracture of an upper incisor with exposure of the pulp.

1. Fracture of Clinical Crown Not Exposing Pulp.—A traumatic fracture of the crown not exposing the pulp is illustrated in the diagram, Fig. 345. Subsequent developments in such a case depend entirely upon the reaction of the pulp. If the pulp is not too close to the exposed dentin surface, secondary dentin will form in the incisal part of the pulp chamber; thus a dentin barricade of increasing thickness is built up between pulp and fractured surface and the pulp will stay alive and healthy. Later on it will be possible to replace the lost part of the tooth by an artificial restoration (inlay).

The formation of secondary dentin under a fracture or part of the crown can easily be studied experimentally. Fig. 346 shows a lower incisor of a dog, the tip of which was snipped off eight weeks previous to the removal of the specimen. A considerable amount of secondary dentin has been formed in the pulp chamber.

If, after a fracture of this type, the pulp is very close to the exposed dentin surface, irritation and infection usually reach the pulp through the thin layer of dentin before the pulp tissue can protect itself by the formation of secondary dentin. Pulpitis develops, and the pulp is lost. Then the case has to be considered and treated similarly to those described under 2.

In every instance of tooth fracture without pulp exposure, it is possible that the blow that fractured the crown may also have torn off the pulp vessels at the apical foramen, thus causing necrosis of the pulp. It is, therefore, always advisable to test the vitality of the pulp at frequent intervals following the fracture, in order to make sure that the pulp stays vital.

2. Fracture of Clinical Crown Exposing Pulp.—If a large piece of the crown is broken off by a blow or fall, the pulp, as a rule, will be exposed (Fig. 347). Then care of the pulp is the main object of therapy. The pulp must be removed and the root canal filled; an artificial restoration of the lost part of the crown can be made, using the root canal for anchorage.

The possibility that a traumatic pulp exposure may heal by a deposit of hard substance (dentin) which would seal the opening is merely of theoretical interest. In practice, the success of all attempts at capping or saving an exposed pulp has to be considered highly doubtful; in the majority of cases where such a procedure has been attempted, the pulp died sooner or later and had to be removed.

3. Fracture Located Partly Outside, Partly Inside Epithelial Attachment (Combined Fracture of Crown and Root).—The majority of combined fractures of crown and root is represented by the spontaneous fractures of posterior teeth with large fillings. The pulp is involved in fractures of this type (Fig. 348); however, it is possible, in older people with greatly reduced pulp chambers, that a cusp together with a splinter of crown and root may be broken off without exposing the pulp.

4. Fracture Located Entirely Inside Epithelial Attachment (Fracture of Clinical Root).—If the crown of a tooth is hit by a kick, blow, or fall, the very resistant enamel of the crown frequently remains intact, whereas the more delicate root is fractured; the thinner

apical portion of the root seems to be especially liable to be broken in such a case. For practical reasons complete cross-fractures and incomplete fractures of the root, including cracks in the root surface, will be differentiated. The first type usually is visible in the radiograph; the latter is found only when the tooth is examined microscopically.

(a) *Complete Cross-fracture of Root.*—In a complete cross-fracture of the root the tooth is usually sore and loose; the absolute diagnosis of fracture can be made only by means of the radiograph. The line of fracture runs across or slightly obliquely through the root (Fig. 349). If the fracture occurs near the neck of the tooth, the crown will be very loose; if the apical third or quarter of the root is severed, the loosening will sometimes be insignificant (Fig. 350).

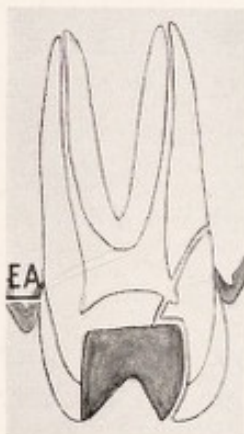


FIG. 348.

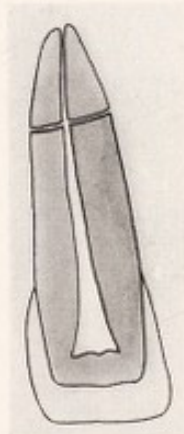


FIG. 349.

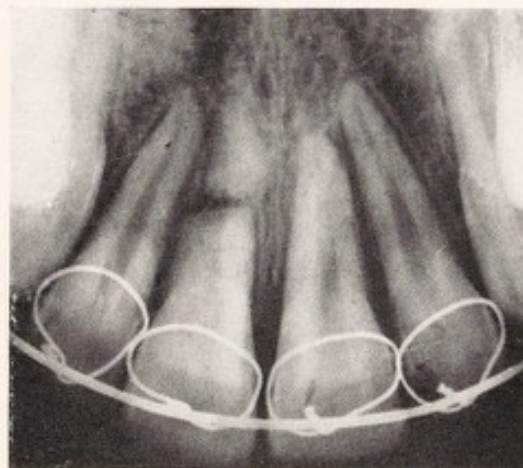


FIG. 350.

FIG. 348.—Diagram of spontaneous fracture of an upper bicuspid carrying a large filling. Exposure of the pulp. EA, position of the epithelial attachment on the tooth surface. The fracture is located partly outside (crownward) of the epithelial attachment, partly inside (rootward) of the epithelial attachment.

FIG. 349.—Diagram of a traumatic cross-fracture of the root of an upper incisor located in the apical third of the root.

FIG. 350.—Radiograph of a cross-fracture of the root of an upper incisor. Considerable displacement of the fragments. Fixation by wiring.

When examining a traumatic tooth fracture clinically one must keep in mind the possibility of a multiple fracture. If, for instance, in an accident part of the crown of an incisor is broken off, there is always the possibility of a cross-fracture of the root in addition to the fracture of the crown; therefore, a radiographic examination is necessary in every case.

Fracture of the root is the only type of tooth fracture in which healing in a biological sense is possible. In fractures that involve only the clinical crown (1 and 2), no reparative processes whatsoever are possible on the fractured surface. In fractures that are

located partly outside and partly inside of the epithelial attachment (3), healing of the fractured portion inside of the epithelial attachment could be possible, but the extension of the fracture into the clinical crown invariably leads to infection of the deeper tissues which prevents healing. Only when the entire line of fracture is well below the epithelial attachment (4) does infection have no access, and only then is healing possible. The final outcome of such a root fracture is governed by the same basic laws that regulate the healing of simple bone fractures. The end-result of any bone fracture can be: either solid healing by newly formed hard substance (callus); or a fibrous juncture (pseudarthrosis) without bony connection; or infection and suppuration which make healing difficult or impossible.

From a clinical viewpoint the first of these three possibilities, the solid bony healing, is obviously the most desirable outcome for every fracture. Four conditions are essential for such a solid bony juncture of a fracture. They are: (1) the fragments must be in a position of close adaptation, (2) the fragments must be immobilized in this position, (3) infection must be absent, and (4) the patient's general health must be such as to make reparative and regenerative processes possible. The following application of these conditions can be made to the problem of root fractures:

1. In a typical bone fracture, a union is possible only if the fragments are in direct contact or at least very close together. The healing of a fracture is initiated by a proliferation of the osteogenic cells of the periosteum and endosteum; these cells differentiate into osteoblasts which lay down bone matrix. By subsequent calcification of this matrix a solid bony union (callus) is formed between the fragments. If the distance between the fractured extremities is too great, the callus cannot bridge the gap; instead, each end will be covered over separately by new bone, and the ends will be united merely by fibrous tissue (pseudarthrosis).

The same is true of a cross-fracture of the root. Shortly after the accident, the hemorrhage that invariably takes place within the line of fracture will be resorbed or organized by ingrowing fibroblasts and cementoblasts. First, a fibrous bridge will be formed between the fragments; later on cementum will be deposited along the line of fracture. If the root fragments are in close adaptation, they will be "cemented together" by the cementum and the continuity of the root thus restored. If the fractured extremities are farther apart, each will be covered over by cementum. Fibrous tissue will remain between them, and no union will take place (see

Fig. 352). Healing of this type may be entirely satisfactory for the maintenance of function, providing that the coronal fragment is long enough to insure sufficient periodontal attachment.

2. In a bone fracture a solid union can be expected only if the fractured ends are in a condition of more or less complete immobilization, hence the general use of splints and plaster casts for the treatment of bone fractures. The same is true of a root fracture. If the coronal fragment is loosened by the trauma and in addition is constantly subject to stress during mastication, a solid union can hardly be expected. Therefore, care should be taken to give a fractured root a chance to heal by immobilizing the coronal fragment as much as possible. This can be done in several ways, depending upon the individual case and its possibilities: Construction of a splint either by using an orthodontic arch wire to which the fractured tooth is ligated, or by making bands for the fractured tooth and its neighbors and soldering them into one fixed appliance which is cemented in place for a period of one to two months. Sometimes, in addition to the construction of a splint, it is necessary to shorten by grinding the fractured tooth or its antagonists to relieve the occlusal stress. In favorable cases the process of repair can be followed in radiographs taken at intervals; it can be seen how the line of fracture becomes more and more indistinct until finally it disappears entirely, indicating that a calcified tissue (cementum) has united the fractured ends.

3. It is a well-established fact that the healing of a bone fracture is greatly delayed and sometimes entirely checked by infection and inflammation. This is especially true of a root fracture. For this reason the condition of the root canal has to be considered very carefully.

If at the time of fracture the tooth was pulpless and had an infected, poorly treated or untreated root canal, the chances for a satisfactory retention of the tooth are practically *nil*. The infection will invariably spread from the root canal or from the infected periapical region into the traumatized tissue near the line of fracture; this will make repair impossible and probably even lead to additional bone destruction around the root. If, however, the fractured pulpless tooth had a satisfactory root canal filling and no periapical infection, the chances of its healing are probably not very different from those of a tooth with a vital pulp.

4. In certain patients bone fractures never heal satisfactorily despite all surgical precautions. It must be assumed that in such people the general reparative power of the organism is below *par*;

this may be due to old age, poor health, incorrect nutrition or to an unknown cause. The same holds true of dental fractures; in some patients, especially in young, strong and healthy individuals, satisfactory tissue repair can readily be expected; in older or in poorly nourished patients, the chances are correspondingly poorer.

Austin, in a recent report from the Mayo Clinic, found that among forty single-rooted anterior teeth with cross-fractures of the roots thirty-one responded to the vitality test. This high percentage of cases in which the pulp remained vital following fracture is indicative of the relatively high resistance of the dental pulp to traumatic injuries providing infection has not gained access.

The reparative and regenerative changes following root fracture have been studied in several cases reported in dental literature. These studies give us a better understanding of the healing process.

Gottlieb described a case in which the apical half of the root of an upper cuspid of a man, who died at the age of twenty-two years, was literally broken into splinters. The crown was intact; nothing was known about the date of the accident or the type of trauma that had caused this fracture. The pulp of the tooth remained vital, although the root was broken into several large and small pieces; deposits of bone and cementum had occurred all over the area of fracture, covering the exposed dentin and partly uniting the fragments. Fig. 351 shows the fractured root end and the dentin splinters. Deposits of bone and cementum are found in the pulp canal and around several larger and smaller root fragments. It should be emphasized that in this case, although it certainly took a great force to cause such splintering of the root, the pulp tissue remained vital, a good example of the surprisingly high resistance and reparative power of the pulp in the absence of infection.

A ground section through a cuspid that suffered a cross-fracture of the root six months before the extraction of the tooth was published by Howe. In this case a complete solid union of the fragments had taken place; the newly formed hard substance between the fragments had been contributed by both periodontal membrane and pulp as it consisted of both cementum and irregular dentin.

Boulger reported a case of cross-fracture of the roots of the lower central incisors. The etiology was known to be an accident (fall on the face) thirty-three years previous to the time of removal of the specimen. Immediately following the accident the teeth were sore and loose, but they became firm again without any treatment. Neither apex became reunited with the upper part of the root, but remained embedded in the jaw; the distance between the frag-

ments was considerable, probably due to a displacement of the upper fragment occlusally during the thirty-three years following the accident. Fig. 352 shows the roots of the central incisors and the apex of the left incisor. In a higher magnification of the root ends of the right incisor, it can be seen that the fractured surface is completely covered by cementum (Fig. 353); the pulp is vital in the upper as well as in the apical fragments. The apical fragments are completely surrounded by cementum; they contain a



FIG. 351.—Root of upper cuspid. Due to a trauma of unknown kind, the apical third of the root was broken into numerous splinters of various sizes. The pulp remained vital. B, ingrowth of bone into the root canal; CR, cracks in the root; DS, splinters of dentin embedded in connective tissue. (Gottlieb, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

thin strand of living tissue that is in connection with the periodontal membrane at the upper (fractured) surface and at the lower end (apex) of the fragments (Fig. 354).

These two cases show that cementum is deposited on fractured root ends. In the case described by Boulger a periodontal membrane was reestablished around the new cementum surface. If the fragments are close enough, this cementum may lead to a solid union of the ends.

In studying the reparative processes following root fracture, it is apparent that deposits of cementum upon the fractured surface are often preceded by some resorption of the exposed dentin surface (see Fig. 353). It seems that resorption usually follows the fracture; later reparation takes place. This preliminary resorption is probably the expression of some tissue injury caused by the trauma: the injured tissues, soft as well as hard tissue (dentin), must be eliminated before repair can take place.



FIG. 352.—Fracture of the root ends of the lower central incisors. Thirty-three years previous to the removal of the specimen the patient, a woman aged forty-five years, fell on her face and fractured the central incisors. The roots of both central incisors radiographically show cross-fractures. The left apex lies close to the upper fragment; the right apex is located at a considerably deeper level. The pulps of both teeth remained vital. The histologic specimen shows the apical fragment of the left incisor separated from the upper portion of the root by connective tissue. CT, connective tissue between the fragments of the left incisor; C, deposits of cementum upon the fractured dentin surfaces; PM, periodontal membrane surrounding the fragments. (Boulger, Jour. Am. Dent. Assn.)

(b) *Incomplete Fracture of Root.*—Fractures of small portions of the root are observed in the form of cracks or tears in the cementum. They have no clinical significance and cannot be recognized by means of the radiograph; however, such small traumatic defects on the root surface are frequently found in the microscopic examination of teeth and are of value as a contribution to our knowledge of the reparative power of cementum and periodontal membrane.

The author described a specimen of a lower incisor in which part of the cementum had been torn away from the root (Fig. 355). The

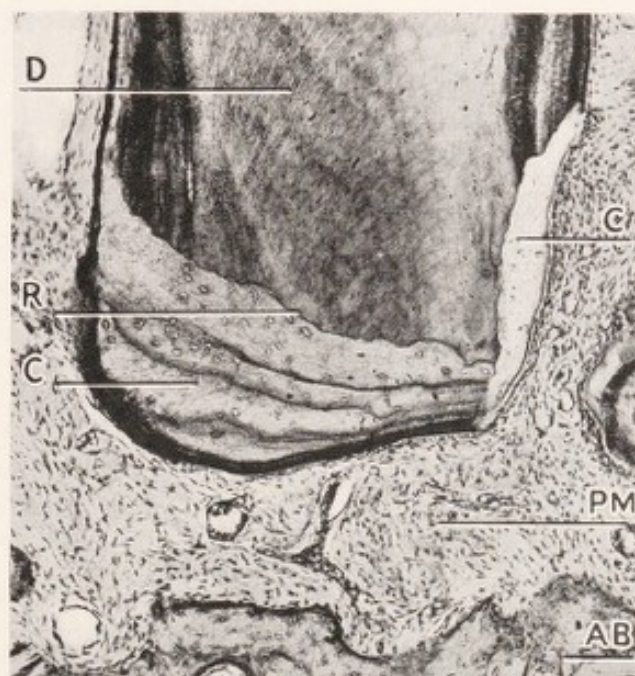


FIG. 353.—High magnification of the root end of the right central incisor of Fig. 352. D, dentin; R, line of resorption on the dentin surface; C, deposits of cementum on the resorbed dentin surface; PM, periodontal membrane; AB, alveolar bone. (Boulger, Jour. Am. Dent. Assn.)

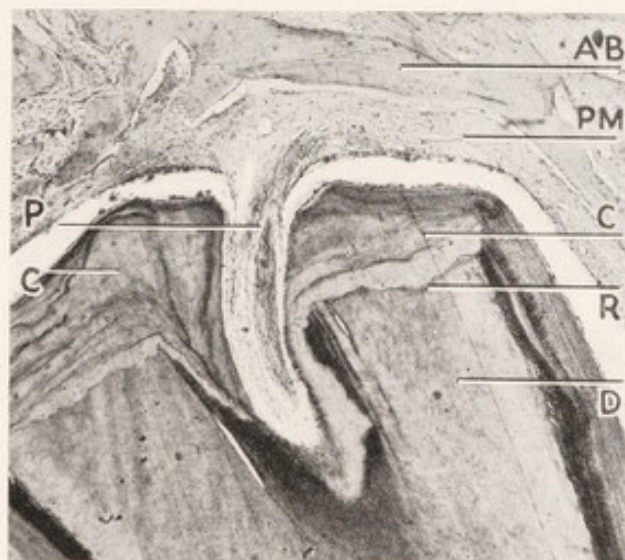


FIG. 354.—High magnification of the upper end of the apical fragment of the right incisor. D, dentin; R, line of resorption on the dentin surface; C, deposits of cementum on the resorbed dentin surface; P, pulp strand entering the root canal on the fractured surface; PM, periodontal membrane; AB, alveolar bone. (Boulger, Jour. Am. Dent. Assn.)

etiology of this kind of fracture is as follows (Euler): when a tooth is hit by a sudden blow, a small area of cementum, whose connection

with the fiber bundles of the periodontal membrane is stronger than its juncture with the dentin, may be torn from the root surface. The cementum splinter lodges in the periodontal membrane. This type of injury undoubtedly occurred shortly before death in the case illustrated here. Fig. 355 shows the tear extending over more than one-half of the clinical root. The upper part of the crack is filled with ingrowing connective tissue from the periodontal membrane. The apical part contains tissue fluid (lymph) and a fine network of cells which, under higher magnification, prove to be

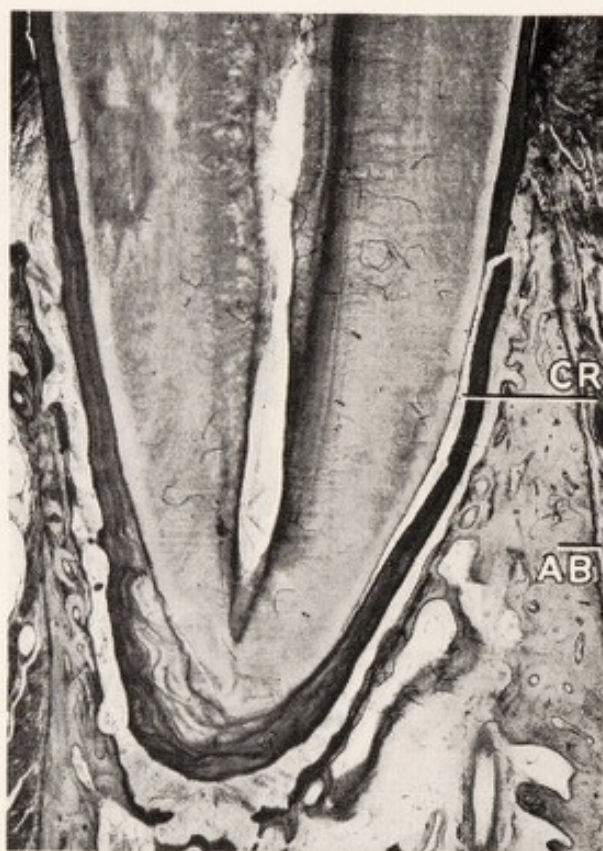


FIG. 355.—Traumatic separation of cementum from the dentin surface. Labio-lingual section through lower central incisor root. CR, crack between cementum and dentin on lingual side of root; AB, alveolar bone. (Kronfeld, Jour. Dent. Res.)

fibroblasts and wandering cells. These cells represent the first step toward the organization of the fluid that entered the crack immediately following the trauma; when the fibroblasts grow and develop they form connective tissue on the walls of the crack and finally cementum, thus reuniting the fractured parts (Fig. 356).

Fig. 357 represents a similar fracture of longer standing. Here a splinter of cementum and dentin had been torn loose from the root near the alveolar margin; this fragment, as well as the corresponding defect on the root surface, has been completely covered by deposits of cementum.

From these findings, the order of tissue changes after a tooth fracture is as follows: Immediately after the fracture, blood and lymph fill the space between the fragments. Soon, from the surrounding periodontal and pulp tissue, fibroblasts and wandering cells proliferate into the crack, just as they would proliferate into any wound or space created within the body; the fibroblasts form connective tissue. Resorption may or may not take place on the fractured ends; if resorption does take place, cementum will be deposited later upon an uneven, eroded surface; if resorption does not take place, cementum will be deposited upon the original

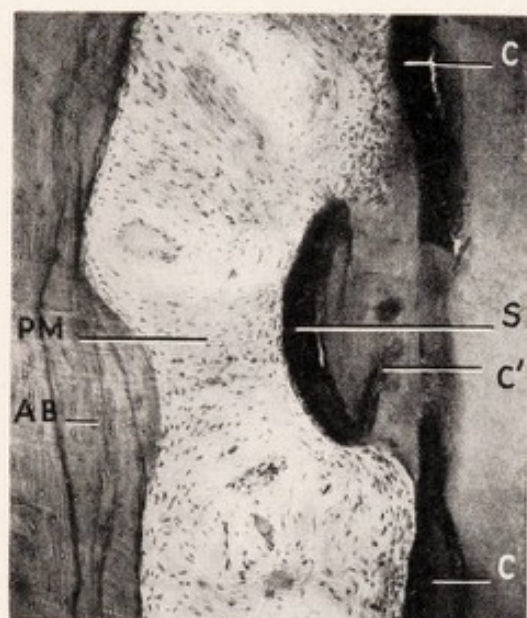


FIG. 356.—Traumatic separation of cementum from the dentin surface. The dislodged splinter of cementum has been reunited with the root by deposits of new cementum. C, original cementum on the root surface; S, splinter of cementum torn away from the root surface; C', cementum uniting splinter and root; PM, periodontal membrane; AB, alveolar bone.



FIG. 357.—Splinter of cementum (S) torn loose from the root surface and embedded in the periodontal connective tissue. Incisor, monkey. X, area from which the splinter of cementum was torn away; C', newly formed cementum covering the root surface; PM, periodontal membrane; AB, alveolar margin. (Figg, Jour. Dent. Res.)

surfaces of the fracture. If the fragments are adjacent, cementum will tie them firmly together; if they are further apart, each fragment will be covered separately, and a periodontal space will develop between the fragments.

The pulp often remains alive, especially in young teeth with wide pulp canals and highly vital tissue. In these cases a collateral blood supply to the pulp develops from the periodontal vessels (Fig. 352).

In other cases the pulp dies and becomes decomposed following a fracture. Clinically, the fate of the pulp in a tooth with a fractured root can be decided only by close observation of the case and repeated vitality tests in the weeks and months following the accident.

PARTIAL FRACTURES OF TEETH NOT FULLY DEVELOPED (DILACERATION).

In this connection mention must be made of a deformity that is sometimes found in erupted teeth and that presents itself as a bend or crease at the junction between crown and root. The microscopic examination reveals that some force or blow against the deciduous tooth had been transmitted to the permanent germ and that, as a result, the forming crown was partly detached or removed from the papilla. Development then continued, but the crown is deformed and the course and direction of the dentinal tubules are markedly changed (Fig. 358).

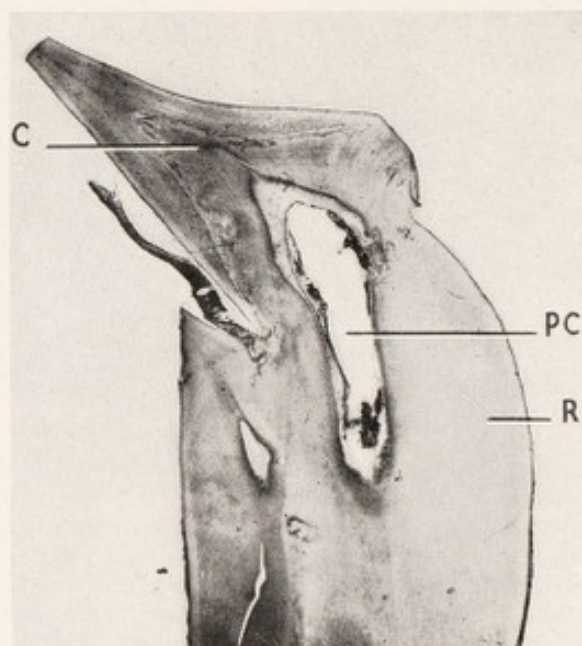


FIG. 358.—Dilaceration (fracture in the developmental period) of a lower incisor. R, root portion of tooth; C, crown portion of the tooth; PC, pulp chamber. The axis of the crown portion of the tooth shows a marked deviation from the axis of the root.

ROOT ENDS REMAINING IN JAW AFTER INCOMPLETE EXTRACTION OF TEETH.

If a piece of a root be fractured during extraction and left in the jaw, the outcome will vary according to the condition of the fragment at the time of the operation. If the root end were chronically

infected, three things are possible: (1) The root fragment may be eliminated gradually by suppuration, coming closer and closer to the surface, finally appearing on the gingivæ, and then being lost. (2) The root fragment may remain in the jaw, the tissues healing over it and forming a sinus leading from the fragment to the surface through which a purulent exudate is discharged. (3) The jaw tissues may heal completely over the fragment, the latter remaining in the jaw without any clinical symptoms as so-called residual or inclosed infection. A low-grade inflammation may flare up eventually causing an acute abscess, or a sinus may be established between the fragment and the outside.

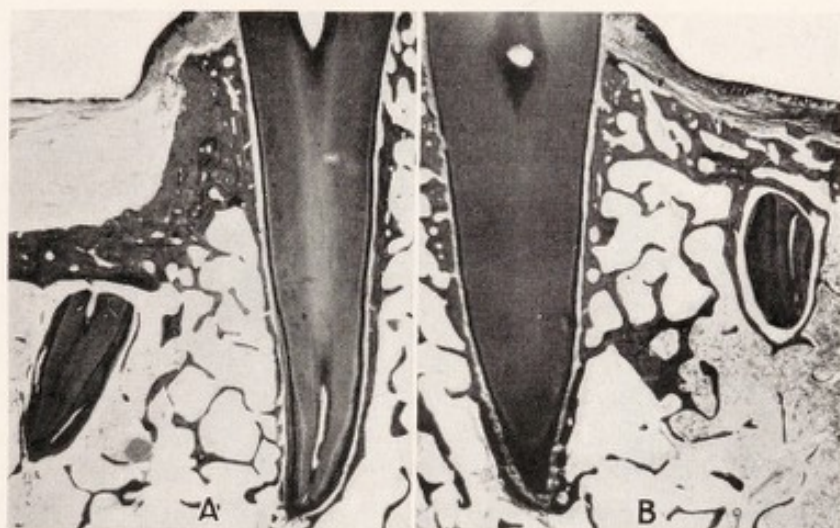


FIG. 359.—Right and left side of the mandible of a woman, aged thirty-eight years. Mesio-distal sections through the region of the second bicuspid and first molar. The mandible was edentulous on both sides back of the second bicuspid. Histological examination revealed on either side the fractured apex of the mesial root of the first molar which had remained in the jaw. Both these fragments are embedded in the surrounding tissues without inflammatory reaction. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

If the root end were not infected, that is, if the extracted tooth had an intact or only superficially inflamed pulp, a fractured root end might heal without any inflammatory reaction, the alveolar bone over the fragment, bone, and soft tissue being completely regenerated, the pulp stump in the fragment remaining alive, and the fractured surface being covered by cementum. The author described the tissue changes around the fractured ends of the mesial roots of the lower first molars on both sides of the same jaw (Fig. 359); both fragments were completely surrounded by bone and did not show any inflammatory changes (Fig. 360). N. G. Thomas reported deposits of cementum on the fractured surfaces of several roots left in the jaw after extractions.

Although favorable conditions are occasionally found around embedded root ends, one must not, however, draw the conclusion that any carelessness in removing fractured root apices is justified. If the field of operation is infected or if infection gains access to the fragment during the extraction, the remaining root end may become harmful to the patient. The old rule still stands: no extraction is completed until the entire tooth is removed.

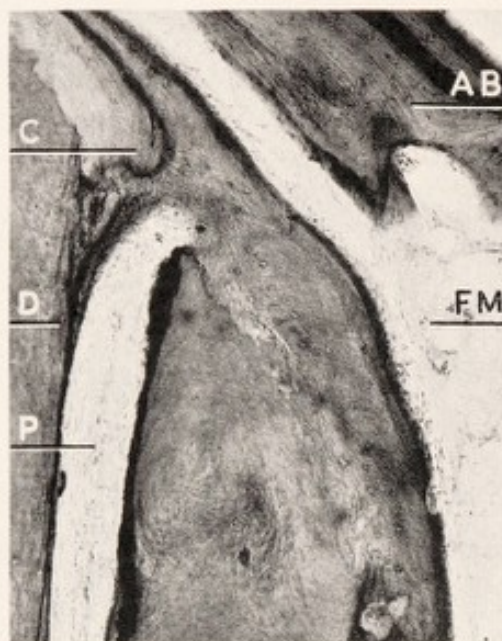


FIG. 360.—High magnification of the surface of the fragment in Fig. 359, B. D, dentin; P, pulp tissue in the root canal; C, cementum deposited upon the fractured dentin surface; FM, fat marrow surrounding the fragment; AB, alveolar bone. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

LUXATIONS OF TEETH.

In a luxation a tooth is either partially or completely severed from its socket by a trauma. In a partial luxation the tooth is sore, loose, and often protrudes from its socket. The therapy is the same as in a fracture: reposition and immobilization (splint) until the tooth becomes firm. A radiograph should always be taken in order to exclude root fracture which is often associated with luxation.

After a tooth has healed following luxation, it is necessary to test the vitality of its pulp, as this organ often suffers apical hemorrhage and lacerations by the injury and is likely to become necrotic. If the pulp does not respond to the electrical and thermal vitality tests, the tooth should be opened, the pulp removed, and the root canal filled. In a complete luxation the therapy consists of replanting the knocked-out tooth. Sometimes such replanted teeth

become embedded in the jaw and function well for a while; but usually they are quickly resorbed and are lost in six months' to several years' time (see page 240). Before replantation it is, of course, absolutely necessary to remove the pulp tissue and to fill the canal, as otherwise the pulp would invariably become decomposed and cause severe inflammation around the replanted tooth.

Traumatic fractures and luxations of teeth are often accompanied by fractures of small parts of the alveolar process surrounding these teeth. Bone fractures of this type usually heal readily if the teeth are held in place properly by a splint.

BIBLIOGRAPHY.

- ADRION, W.: Gewebsveränderungen in der Zugzone des Parodontiums unter traumatischem Einfluss, *Parodontium*, 1932, **4**, 135.
- AISENBERG, M. S.: A Tear in the Cementum, *Jour. Dent. Res.*, 1930, **10**, 599.
- Repair of a Fractured Tooth, *Dental Cosmos*, 1932, **74**, 382.
- AUSTIN, L. T.: A Review of Forty Cases of Retained Fractured Roots of Anterior Teeth, *Jour. Am. Dent. Assn.*, 1930, **17**, 1930.
- BAUER, W.: Über traumatische Schädigungen des Zementmantels der Zähne mit einem Beitrag zur Biologie des Zementes, *Deutsch. Mon. f. Zhk.*, 1927, **45**, 769.
- BLACK, G. V.: *Operative Dentistry*; Vol. II, The Technical Procedures in Filling Teeth, Chicago, Medico-Dental Publishing Company, 1920.
- BOULGER, EARL P.: Histologic Studies of a Specimen of Fractured Roots, *Jour. Am. Dent. Assn.*, 1928, **15**, 1778.
- EULER, H.: Über das Verhalten frakturerter, im Kiefer zurückgebliebener Wurzeln, *Deutsch. Zahnärztl. Wehnschr.*, 1924, **27**, 199.
- Die Rissfraktur am Wurzelzement, *Ztschr. f. Stom.*, 1927, **25**, 801.
- FIGG, W. A.: A Tear in the Cementum, *Jour. Dent. Res.*, 1928, **8**, 623.
- GOTTLIEB, B.: Histologische Untersuchung einer geheilten Zahnfraktur, *Ztschr. f. Stom.*, 1922, **20**, 286.
- Histological Examination of a United Tooth Fracture, *Dent. Items Int.*, 1926, **48**, 877.
- Frakturen und Luxationen der Zähne, *Scheff's Handb. d. Zhk.*, Wien, Hölder, 1927, **3**, 1.
- HOWE, PERCY R.: A New Research on Dental Caries, *Dental Cosmos*, 1926, **68**, 1021. Figs. 3-6.
- KRONFELD, RUDOLF: Zur Frage der Wurzelspitzenamputation, *Ztschr. f. Stom.*, 1928, **26**, 1105.
- Über den Ausgang traumatischer Pulpenschädigung, *Ztschr. f. Stom.*, 1929, **27**, 846.
- The Process of Repair Following Tooth Fracture, *Jour. Dent. Res.*, 1931, **11**, 247.
- Beitrag zur Kenntnis der Heilungsvorgänge nach Zahnfrakturen, *Ztschr. f. Stom.*, 1931, **11**, 418.
- Classification, Diagnosis, and Prognosis of Tooth Fracture, *The Bur, Chicago College of Dental Surgery*, 1932, No. 3.
- MEYER, W.: Ein Beitrag zur traumatischen Schädigung von Zahnkeimen, *Deutsch. Mon. f. Zhk.*, 1924, **42**, 497.
- MÜNCH, JOSEF: Pathologische Histologie der Zähne, *Fortschr. d. Zhk.*, 1929, **5**, 261.

- OTTOLENGUI, R.: A Consideration of the Possible Results of the Fracture of the Root of a Tooth Which Contains a Living Pulp, *Dent. Items Int.*, 1926, **48**, 717.
- Further Consideration of the Possible Results of the Fracture of the Root of a Tooth Which Contains a Living Pulp, *Dent. Items Int.*, 1927, **49**, 79.
- The Practical Application of the Knowledge Gained by the Study of the Possible Results of the Fracture of the Root of a Tooth Which Contains a Vital Pulp, *Dent. Items Int.*, 1930, **52**, 911.
- SCHIER, M. B. A.: Histological Study of a Tooth Retained in Its Socket for More Than Ten Years after Complete Fracture of Its Root, with Evidences of Continued Vitality of the Pulp and Environmental Tissues, *Dent. Items Int.*, 1926, **48**, 81.
- SZABO, J.: Wundheilung bei in der Alveole zurückgebliebenen Wurzelresten, *Ztschr. f. Stom.*, 1928, **26**, 609.
- THOMAS, N. G.: Studies in Protective Cementum Development, *Dental Cosmos*, 1922, **64**, 385.

CHAPTER XVII.

ENAMEL HYPOPLASIA.

CLINICAL MANIFESTATIONS AND ETIOLOGY OF ENAMEL HYPOPLASIA.

AN enamel hypoplasia is a congenital enamel defect in the teeth of the permanent dentition. The clinical appearance of these defects varies widely; in mild cases of hypoplasia, the enamel is intact except for several shallow depressions or grooves on otherwise smooth enamel surfaces; in more extensive cases, the grooves or pits of the enamel are arranged in one or several horizontal rows all around the crown extending into the enamel as far as the dento-enamel junction. In pronounced cases of hypoplasia, the crown may be so involved that the enamel on the incisal edge of the incisors and cuspids will be entirely lacking, and the dentin on the occlusal surface of the first permanent molar will be exposed. The small amount of enamel that is present in cases of severe hypoplasia is distributed irregularly on the labial and lingual surfaces of the teeth.

The term enamel hypoplasia for such defects was first used by Zsigmondy. This term is preferable to the old term atrophy because the condition is characterized by an underdevelopment of the enamel, while the word atrophy indicates a decrease in the size of a fully developed tissue or organ.

Enamel hypoplasia is the expression of a temporary, circumscribed disturbance of the enamel formation. The enamel prisms in the involved area remain permanently in the state of development that they had attained when the disturbance occurred. After the interference subsides, normal enamel may again be formed. If the disturbance is repeated, a zone of missing or underdeveloped enamel will again result; hence, an alternating distribution of the hypoplastic areas on the surface of the crown is observed (Fig. 361).

Since the periods of calcification and enamel formation of the permanent teeth are known, it is possible to estimate from the distribution and localization of the hypoplastic areas the time of their development, that is, the age of the child at the time of the disturbance.

The crowns of the deciduous teeth begin to calcify before birth, and enamel formation is completed between the ages of four and six months. Therefore, enamel hypoplasia of the deciduous teeth is rare, is never very extensive, and has no practical significance. Calcification of the permanent teeth begins shortly after birth with the first permanent molars; these teeth are soon followed by the upper and lower permanent central incisors and by the permanent cuspids. At the age of six months, a small cap of enamel and dentin is formed on the incisal edges of the upper and lower per-

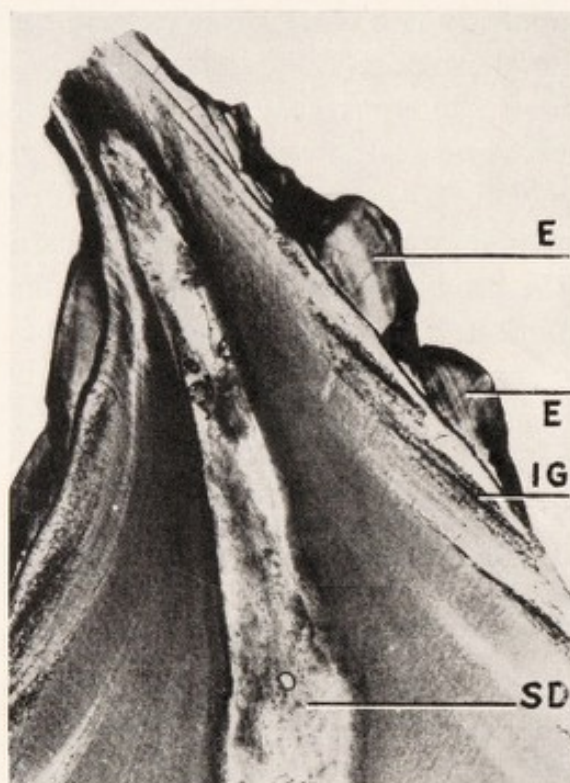


FIG. 361.—Labio-lingual ground section through an incisor with severe enamel hypoplasia. On the labial surface of the crown the enamel is arranged in several horizontal ridges, E, between which the dentin is exposed. IG, interglobular spaces in the dentin arranged in rows corresponding to the distribution of the enamel hypoplasias; SD, secondary dentin in the pulp chamber. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

manent central incisors, the lower permanent lateral incisors and the permanent cuspids. On the first permanent molars, four small individual caps of enamel and dentin are present corresponding to the tips of the four cusps.

Next in the order of calcification are the upper permanent lateral incisors the calcification of which begins considerably later than that of the central incisors and the cuspids, at about the age of one year. Enamel formation on the incisors and first permanent molars is completed in the fourth year of life; the cuspids take somewhat

longer, their crowns being fully formed and calcified in the sixth year.

In the upper and lower bicuspid and second permanent molars, hard substance formation begins during the second and third years, and is complete about the age of seven years. The third molars develop still later, between eight and twelve years of age.

Data concerning the time of calcification of the human teeth were obtained by systematic histological examination of the jaws of 25 children whose ages varied from new-born to fifteen years (Logan and Kronfeld). Certain deviations within a range of one or two months can usually be observed and may logically be expected, depending upon the development and health of the individual child.

Using the above data concerning enamel formation as a guide, it is possible to predetermine the result that a disturbance of the enamel formation at any given period of life may produce; on the other hand, from a hypoplastic area on an erupted tooth one can approximately determine when the causative damage occurred.

If the interference with enamel formation takes place in the first year of life, the central incisors, the lower lateral incisors, and the first permanent molars will most likely show evidence of enamel hypoplasia. The cuspids are usually involved to a slighter degree than the central incisors. If the disturbance occurs in the second year, the upper lateral incisors also will be affected. It may seem strange that the cuspids, which erupt at the age of eleven or twelve years, should begin to form earlier than the upper lateral incisors, which erupt at the age of seven or eight years, but microscopic findings have beyond doubt established the fact that the cuspids begin to calcify at least six months before the upper lateral incisors. This is clearly borne out in some cases in which the central incisors and the cuspids show enamel hypoplasia, whereas the upper lateral incisors are free from defects. The bicuspid and second permanent molars will be affected only if the disturbance occurs or persists during the third and fourth years of life or still later. The third molar is seldom involved.

RICKETS, TETANY, AND ENAMEL HYPOPLASIA.

To quote the various opinions that have been expressed on the etiology of enamel hypoplasia would take too long. Toward the end of the past century, several authors believed rickets to be a definite cause for the development of enamel hypoplasia. Fleischmann, in 1909, subjected these theories to a critical study and came

to the conclusion that rickets cannot be considered as an etiological factor in enamel hypoplasia. He argued that rickets is by far more common than enamel hypoplasia and is, furthermore, a chronic disease while the distribution of areas of enamel hypoplasia points toward a disease with an intermittent course. Fleischmann believed tetany to be the cause of enamel hypoplasia, and he reported an analysis of 10 cases of hypoplasia, all of which gave a history of tetany (convulsions) at the time when the affected teeth were forming.

The investigations of Erdheim and Toyofuku cast new light upon the difficult problem of disturbed enamel calcification. Erdheim reported, in a classic study, that the removal of the parathyroid glands in rats caused severe disturbances in the enamel formation of the incisors, the enamel of the erupted portion of the incisors being discontinued in many places. The enamel epithelium showed extensive proliferations and became detached from the enamel surface, forming irregular, poorly calcified enamel masses. From the similarity of the changes in parathyroidectomized rats to the findings in human jaw specimens Fleischmann came to the conclusion that these experimentally produced enamel defects corresponded to enamel hypoplasia in human teeth, which suggests that human enamel hypoplasia is the result of a disturbance of the parathyroid glands at the time of enamel formation. As mentioned before, Fleischmann corroborated this idea with 10 case histories in which there was a coincidence of tetany and enamel hypoplasia of those teeth that were calcifying at the time of the tetany attack.

It may perhaps be advisable to give a few brief data concerning the pathology and symptoms of tetany. The signs of manifest tetany are mainly spasms of some muscles or convulsions of the body. Certain groups of extremity muscles are most frequently affected, resulting in flexion of the upper and lower extremities, contraction of the hands, bending of the fingers, and downward bending of the toes. The muscles of the face are contracted, giving the face a stiff and rigid appearance; very frequently the muscles of the larynx are involved, resulting in laryngospasm (spasm of glottis) with difficulty in the respiration and hence loud, drawn breathing.

Much more frequently, however, than as manifest tetany the disease occurs in a latent state, as latent tetany. This condition is characterized by hyperirritability of the peripheral nerves when tested by means of galvanic electricity or by other tests that consist of a mechanical irritation (tapping) of certain peripheral nerve

trunks. In latent tetany, contractions of the muscles do not occur spontaneously but can be brought about only by the above-mentioned tests. Fleischmann suggested latent tetany as an explanation of those cases of enamel hypoplasia in which no history of manifest symptoms of tetany can be obtained.

Thus far nothing has been said that could give a clew to the relationship between muscle spasms and enamel hypoplasia. This clew, however, is offered by one of the constant phenomena that accompanies tetany, namely, the lowered concentration of the blood calcium. In all infants suffering from tetany either of the manifest or of the latent type, the calcium content of the serum is distinctly low. This decrease in the blood calcium automatically causes a preponderance of potassium and sodium ions and, as a result, increased irritability of the muscles. It has been well established clinically and experimentally that the parathyroid glands have a distinct controlling and regulating influence upon the calcium metabolism of the body, for the surgical removal of these glands is followed by a decrease in blood calcium and by tetany; this form of tetany may be inhibited and the calcium level raised to normal by administration of the gland extract. Thus the clinical and laboratory evidence concerning tetany, parathyroid glands, and enamel formation seems to be rather definite: the disturbed function of the parathyroid glands causes a decrease in blood calcium resulting in hyperirritability of the muscles, which presents itself clinically as tetany, either of the latent or of the manifest type. The same disturbance in the calcium metabolism also interferes with the normal calcification of the forming enamel, causing defective enamel or enamel hypoplasia. In other words, convulsions do not cause enamel hypoplasia, but both convulsions and hypoplasia are the result of the lowered level of blood calcium. The relationship between parathyroid gland and enamel hypoplasia has been demonstrated by Erdheim and Toyofuku in their experiments on rats.

What, then, is the rôle that rickets plays in this connection? Hess states that without doubt an intimate relationship exists between infantile tetany and infantile rickets. He says that "from a clinical point of view it may be considered that practically all infants with signs of tetany have rickets to some degree." However, the difficult problem of the relationship between rickets and tetany is still under investigation and has not yet been cleared. In many instances where the case history reports rickets only, manifest or latent tetany may have been present at some period. Clinically

rickets is characterized by the inability of the organism to retain the calcium that is taken in with the food, and, as a result, calcium is constantly being lost from the body. The basic difference between rickets and tetany is that in rickets the calcium retention is insufficient; whereas, in tetany the calcium content of the blood is subnormal. Both conditions, however, are severe disturbances of the calcium metabolism.

Gottlieb, Siegmund, and Weber stressed the importance of rickets as an etiological factor in enamel hypoplasia with full recognition, however, of the rôle that tetany plays in this connection. It may be that, finally, the rickets theory and the tetany theory will come to a common solution. Meanwhile it may be fairly safe to say that enamel hypoplasia alone, without severe disturbance of dentin calcification, points toward tetany as causative factor, whereas, poor calcification of the dentin, underdevelopment (shortness) of the roots, and delayed eruption of the teeth are more characteristic of rickets.

HISTOPATHOLOGY OF TEETH WITH ENAMEL HYPOPLASIA.

Few publications have for their subjects the microscopic study of enamel hypoplasia. The earlier reports dealt with the examination of ground sections through fully formed human teeth with hypoplastic enamel; however, such specimens cannot give any definite information as to the early stages and the development of the condition. This information can be obtained only by studying decalcified sections through jaws of young children in which the forming germs show evidence of enamel hypoplasia. An extensive report of the findings in specimens of this type was published by Gottlieb in 1920; his material included the jaws of 5 children who had reached the ages of from two months to one and one-half years. In all these jaws disturbances of varying degrees could be found in the enamel of the permanent tooth germs.

A typical finding in one of these germs is reproduced here (Fig. 362); it shows the tip of a permanent tooth germ. The enamel, which stained dark purple with hematoxylin, is completely absent in one place, exposing the dentin in this area. On a corresponding level on the other side of this germ, the enamel is very much thinner than in the periphery, forming a pit or groove in the enamel surface. The condition as a whole corresponds to the typical arrangement of the enamel in a tooth with severe hypoplasia; in some places enamel is entirely lacking, while in other parts deep

grooves or minor irregularities of the enamel are found. The enamel epithelium surrounding the crown shows distinct pathological changes, the inner enamel epithelium having become detached from the enamel surface and the intervening space having become filled with cells and cell detritus. The ameloblasts have disappeared; the stratum intermedium and the outer enamel epithelium show extensive and irregular proliferation. It is evident that under these circumstances no further enamel formation could take place, and had the child, from whom this specimen was obtained, lived and the tooth erupted, it would have had an appearance similar to that of the tooth illustrated in Fig. 361. The dentin of this tooth germ shows evidence of very poor calcification, wide dentinoid, and numerous large interglobular spaces (see page 441).

From his findings in these specimens, especially of the earliest stages of hypoplasia in the youngest of children, Gottlieb explained the formation of enamel hypoplasia in the following manner: Enamel, like any other hard tissue, is originally formed as an uncalcified protoplasmic substance that, analogous to osteoid, might be called "adamantoid." Under normal conditions, this protoplasmic enamel substance is replaced by lime salts very soon after its formation, leaving only about 3 per cent organic substance.

Von Ebner demonstrated that the transformation from protoplasmic into calcified enamel is accompanied by a change in optical properties as revealed by the examination of forming enamel under the polarization microscope. This change in the enamel from the protoplasmic to the calcified state is also responsible for the well-known observation, made in the study of decalcified sections through developing teeth, that very young enamel is not dissolved by the acid and stains purple with hematoxylin. As the enamel grows older it reaches a stage in which it is almost completely dissolved by acid; only very fine, shadow-like remnants, the above-mentioned 3 per cent organic matter in normal calcified enamel, are found after decalcification.

In cases of disturbed calcification, the formation of the protoplasmic uncalcified enamel occurs normally at first. Very soon, however, the protoplasm disappears and only a very delicate organic scaffold, that would correspond to finished enamel minus lime salts, is left. This fine network of organic material is too weak to retain its form and outline; unless it is supported and reënforced by calcification immediately after its formation, it collapses causing a dip or, in more severe cases, a discontinuity of the enamel. The area in which the uncalcified enamel substance has collapsed pre-

sents itself in the erupted tooth as hypoplasia. During this breaking of the organic enamel tissue, younger portions of the enamel come to lie on top of older ones, which accounts for the lines of injury (*Unterbrechungslinien*, Zsigmondy) (Fig. 363). These lines usually run from the bottoms of shallow groove-like areas of hypoplastic enamel into the underlying enamel, thereby forming a continuation of the tooth surface on one side of the hypoplasia. In

Fig. 363 the first formed enamel can be observed on the left side of the picture; the enamel to the right appears superimposed upon

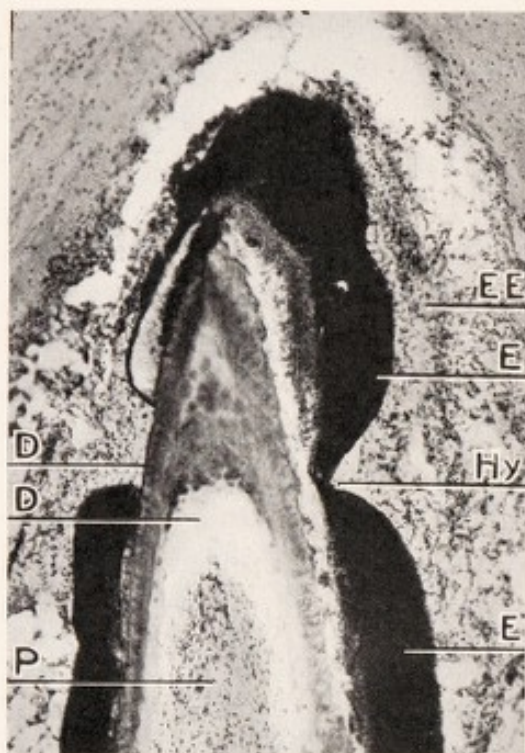


FIG. 362.—Tip of a permanent tooth germ of a child, aged one and one-half years, with severe enamel hypoplasia. The enamel epithelium, EE, is detached from the enamel and shows extensive cell proliferation. E, poorly calcified enamel; Hy, enamel hypoplasia in a corresponding level on the labial and lingual side of the germ; D, exposed dentin at the bottom of the hypoplasia; D', wide layer of dentinoid; P, pulp. (Gottlieb, courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

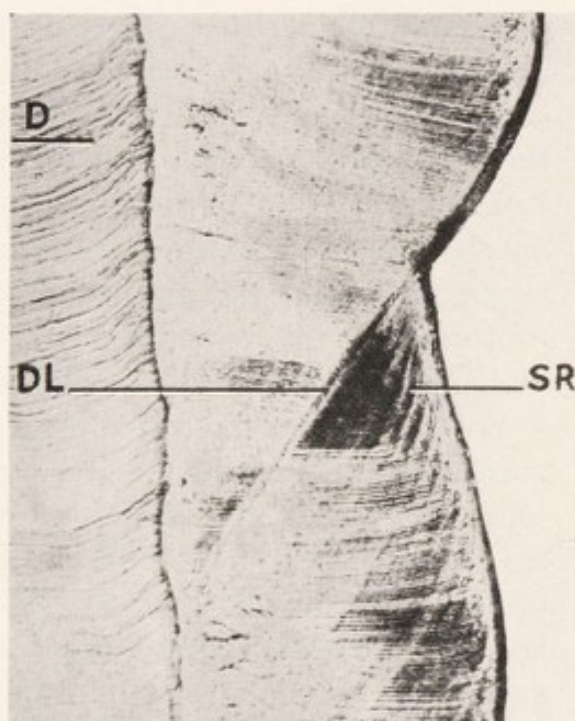


FIG. 363.—Shallow enamel hypoplasia on a human incisor (ground section). From the bottom of the hypoplasia a line of injury, DL, extends into the underlying enamel. The enamel to the right of the line appears superimposed on that to the left. SR, stripes of Retzius converging toward the bottom of the hypoplasia; D, dentin.

that on the left side and separated from it by the line of injury. According to Gottlieb's interpretation, the enamel to the right of the line of injury collapsed during its formation due to defective and delayed calcification. This becomes very evident in studying the minute structure of the enamel at the bottom of the hypoplasia. The enamel below the line of injury appears normal except for poor calcification of the interprismatic substance in some places. The

enamel overlying the line of injury is poorly calcified as indicated by the presence of numerous stripes of Retzius (page 77). The stripes are not arranged parallel to each other but converge toward the bottom of the hypoplasia, indicating that an actual displacement (collapse) of the forming enamel took place. At the line of injury the prisms have been completely discontinued and their course has been changed.

Another form of enamel hypoplasia is illustrated in Fig. 364. The specimen under consideration is the tip of a permanent incisor of a child who died with symptoms of severe rickets (case observed by J. R. Blayney). Near the incisal edge of this tooth two layers

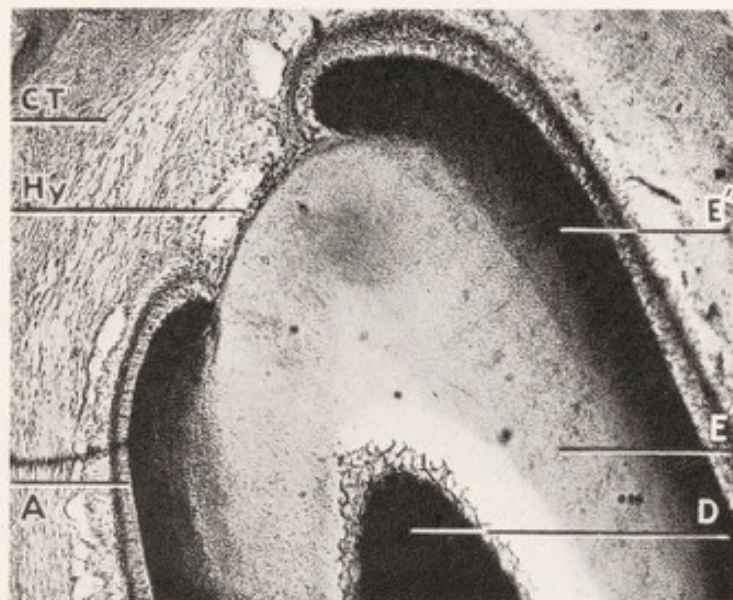


FIG. 364.—Enamel hypoplasia near the incisal edge of a permanent tooth germ of a rachitic child. D, dentin; E', inner zone of enamel; E'', outer zone of enamel; A, ameloblasts; Hy, enamel hypoplasia; CT, connective tissue surrounding the germ. (Courtesy of J. R. Blayney.)

of enamel can be distinguished: next to the dentin a thick layer of enamel that does not show any irregularities, and a thinner superficial layer that stained a much deeper shade of purple than the inner layer of enamel. Near the incisal edge the outer zone of enamel is completely missing, exposing the deeper enamel; likewise, the enamel epithelium over the exposed area has been reduced to a few scattered cells while the normal, outer zone has a regular and continuous epithelial covering.

The rôle of the enamel epithelium in case of hypoplasia is very interesting, and its study is of great importance to the full understanding of the condition. At first one might be tempted to consider enamel hypoplasia simply as the result of a degeneration of

the enamel-forming cells. However, the investigations of Gottlieb on many human specimens have shown that the changes in the enamel epithelium are only of secondary importance; the primary disturbance consists of delay or lack of calcification of the young enamel. The sequence of the changes is as follows: Because of the defective calcification of the enamel, the breaking down of portions of the enamel, and the disturbed formation of dentin, the enamel epithelium separates from the underlying enamel and forms folds. The ameloblasts are not disturbed at first, and for a time

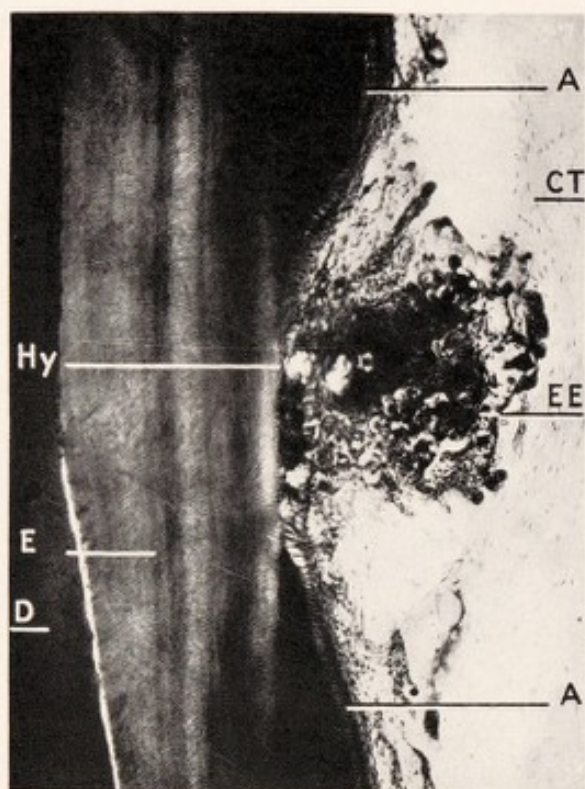


FIG. 365.—Enamel hypoplasia in the first permanent molar of a young monkey. D, dentin; E, enamel; A, ameloblasts; Hy, bottom of hypoplastic area of enamel; EE, proliferated enamel epithelium surrounding irregular amorphous globules of enamel; CT, connective tissue.

may continue to form enamel. The resulting space between enamel epithelium and tooth surface is filled with fluid, cells, and cell debris, and occasionally with droplets of enamel formed by the detached ameloblastic layer. This formation of folds in the detached enamel epithelium explains findings like the one illustrated in Fig. 366 in which droplets of enamel lie free in the tissue around the germ opposite a hypoplasia. Such free droplets of enamel are, of course, found only in sections through hypoplastic teeth before eruption; once the tooth erupts they are lost.

The property of the ameloblasts to form enamel is not continued

indefinitely in their new, atypical position; sooner or later they degenerate and disappear. At the same time, a proliferation of the stratum intermedium takes place, the cells of which multiply rapidly and extend into the connective tissue around the germ.

These changes will be illustrated using sections through the germ of the unerupted first permanent molar of a monkey. Fig. 365 shows a rather shallow enamel hypoplasia, on each side of which the enamel is lined by a regular row of ameloblasts. Toward the hypoplasia the ameloblasts become more irregular, and they disappear completely at the bottom of the groove in the enamel, in which is found an extensive proliferation of the epithelial cells of the stratum intermedium around a dark purple mass, apparently poorly formed enamel.

Free droplets of enamel, formed after detachment of the enamel epithelium, were found by Gottlieb in human tooth germs with hypoplastic enamel. These findings are strong evidence against the assumption of a primary injury to the ameloblasts for, as has been observed, the ameloblasts have by no means lost their enamel-forming properties; on the contrary, even after the enamel epithelium has become detached from its original location, they continue to form enamel in their new position. The primary cause is delayed calcification of the enamel and subsequent detachment of the enamel epithelium; the degenerative changes in the ameloblasts are of secondary nature.

From the description of the minute changes in a forming hypoplasia, the general appearance of the hypoplastic enamel of a tooth germ can be well understood. The inner layer of enamel of the first permanent molar illustrated in Fig. 366 is apparently normally formed and calcified, but the superficial layer of enamel shows evidence of hypoplasia in many places. Corresponding to some of these areas, free droplets of enamel can be seen in the tissue surrounding the germ. From what has been said before, it is evident that this condition developed as a result of the defective calcification of the last-formed enamel. The prisms that were not supported by sufficient lime salts broke down, causing the enamel epithelium to become detached and to form loops, in the center of which the ameloblasts continued to function and formed enamel droplets.

Detachment of the enamel epithelium from a large portion of the crown surface or even from the entire enamel surface means complete interruption of enamel formation; the final enamel will have only the thickness that it had when the connection with the ameloblasts was severed.

It may be appropriate to discuss briefly the very common occurrence of dental caries in teeth with enamel hypoplasia. The hypoplastic defects form areas of retention and in a susceptible mouth it is almost impossible to save such teeth from destruction. Once decay has reached the dento-enamel junction, it usually encounters very little resistance and spreads rapidly through the poorly calcified dentin. As a result, such teeth sometimes are found completely destroyed shortly after eruption.

In summarizing the histological findings in hypoplastic enamel, it may be said that such a formation is caused by insufficient or delayed calcification of the enamel. The uncalcified or poorly calcified matrix is not sufficiently strong to keep its form, and it



FIG. 366.—Enamel hypoplasia in the germ of the lower first permanent molar. Monkey. P, pulp; D, dentin; E, enamel; Hy, enamel hypoplasia. In several places enamel droplets are found overlying the enamel defects. The empty space between dentin and enamel is due to preparatory shrinkage.

collapses, causing interruptions or breaks in the continuity of the forming enamel. The enamel epithelium overlying the damaged area is separated from the young enamel and is folded. For a while the ameloblasts continue to produce enamel in an atypical location; sooner or later, however, the enamel epithelium degenerates and disappears leaving defects and irregularities on the enamel surface.

Other forms of poorly calcified enamel can be considered as preliminary or abortive stages of hypoplastic enamel. An example, for instance, is the congenital white spots that are sometimes observed on the labial surfaces of the incisors. Here defective enamel calcification causes a lack of transparency and a white, opaque enamel. However, the prisms are sufficiently reinforced by calci-

fication to prevent their collapse and the formation of hypoplasia; therefore, such areas of "chalky" enamel are smooth and level with the surrounding surface of the crown.

DISTURBED CALCIFICATION OF THE DENTIN.

Dentin formation normally occurs in the following way: shortly after the dentin matrix has been formed by the pulp tissue, calcium salts are deposited into the matrix (see Fig. 5). Ordinarily, the precipitation of these salts takes place to within a short distance of the pulp; thus the pulp chamber is lined only by a very thin layer of uncalcified matrix (dentinoid). The lime salts are deposited in small, round globules which grow by concentric deposition and finally unite to form the fully calcified dentin.

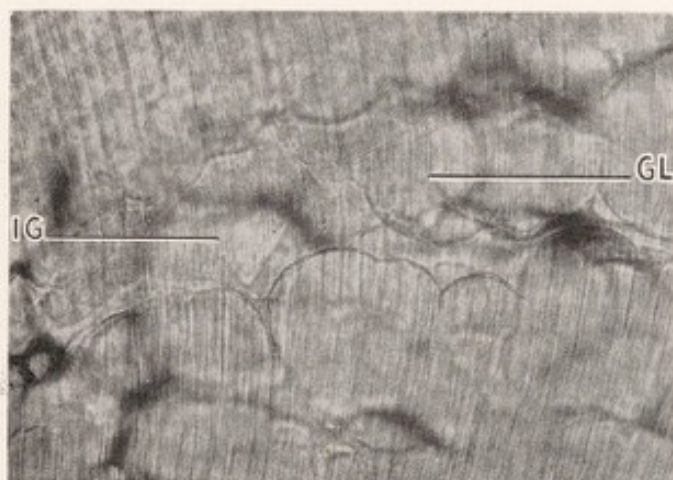


FIG. 367.—Interglobular spaces in the dentin (ground section). GL, globules of calcification; IG, interglobular spaces.

If the calcification of the dentin be slightly disturbed, the union of the globules of calcification is not complete; some uncalcified matrix is left, bordered by convex calcified globules. These areas of matrix are called interglobular spaces; the name is rather misleading since we are not dealing with true spaces but with the uncalcified dentin matrix between the globules (Fig. 367). Therefore, it would be better to use the term interglobular dentin. If dentin calcification be severely disturbed, the number of interglobular spaces is very much increased; in extreme cases of rickets, the dentin may be entirely uncalcified except for some small globules. The latter condition can easily be produced experimentally and studied in the incisors of white rats, which react promptly to a lack of vitamin D with the development of a rachitic condition. Due to the severe

disturbance of the calcium metabolism the dentin is very poorly calcified. Fig. 368 shows a portion of the labial surface of an upper incisor of a rat that had been kept on a rickets-producing diet for eight weeks. Only the layer of dentin that is lying directly beneath the enamel is calcified. Toward the pulp, this zone of calcified dentin is followed by a layer of dentinoid (matrix) with irregularly scattered globules of calcification. The innermost layer of dentin, most recently deposited, does not show any evidence of calcification; it consists entirely of dentinoid. In order to make these changes

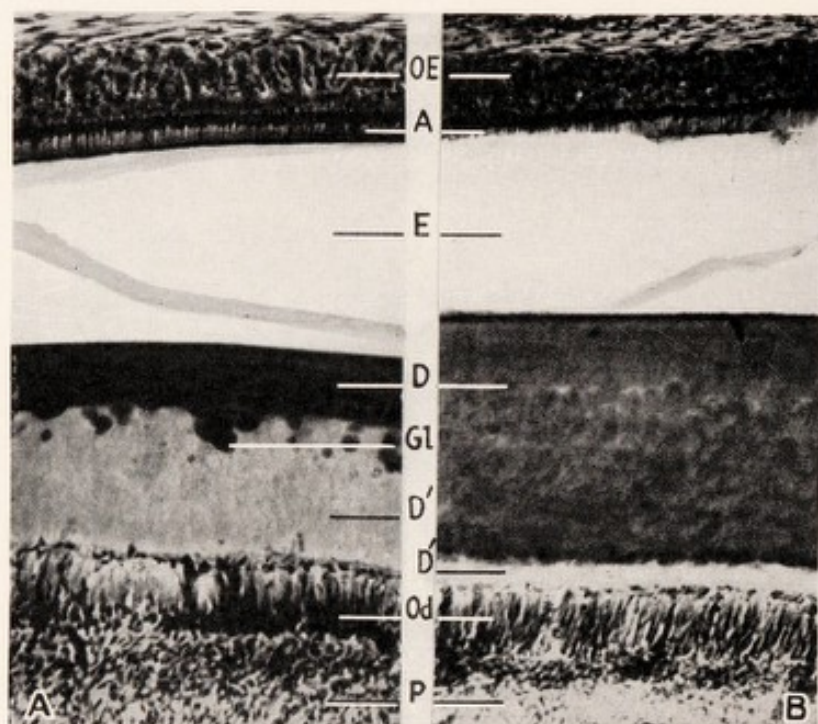


FIG. 368.—A. Incisor of a rat fed for eight weeks on a diet deficient in vitamin D. Poor calcification of the dentin; wide layer of dentinoid. B. Corresponding area of an incisor of a litter mate control animal, fed on a balanced standard diet. Normal calcification of the dentin. OE, outer enamel epithelium; A, ameloblasts; E, enamel; D, calcified dentin; Gl, globules of calcification; D', dentinoid (uncalcified dentin matrix); Od, odontoblasts; P, pulp. (Kronfeld and Barker, *Jour. Am. Dent. Assn.*)

plainer a section through the corresponding area of the incisor of a litter mate control of this animal is reproduced in Fig. 368. The latter rat was kept on a normal sufficient diet. It can be seen that the dentin of the control animal is well calcified throughout; only next to the pulp chamber is there a narrow layer of dentinoid present.

Changes very similar to those that can be produced experimentally in animals are found in the dentin of human teeth in case of rickets. A very instructive case of this type is illustrated in Fig. 369, taken of a human first permanent molar. The dentin shows

bands or zones of very poor calcification alternating with zones of somewhat better calcification. In some places matrix without any calcium globules is present. Next to the pulp chamber a wide

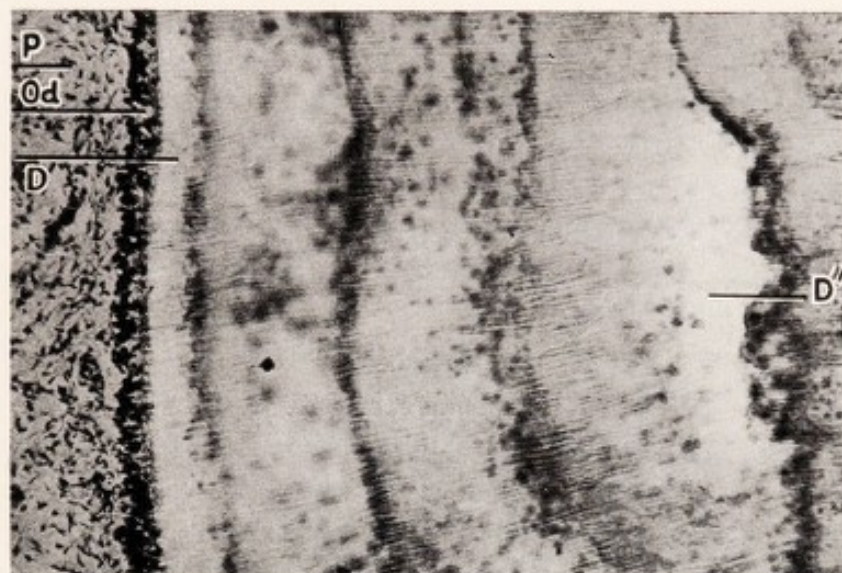


FIG. 369.—Rachitic dentin in a permanent molar of a child. Zones of very poor dentin calcification alternate with better calcified layers. P, pulp; Od, odontoblasts; D', dentinoid next to the pulp; D'', matrix without globules. The dark zones indicate the presence of a larger number of globules of calcification. The light zones consist of uncalcified matrix.

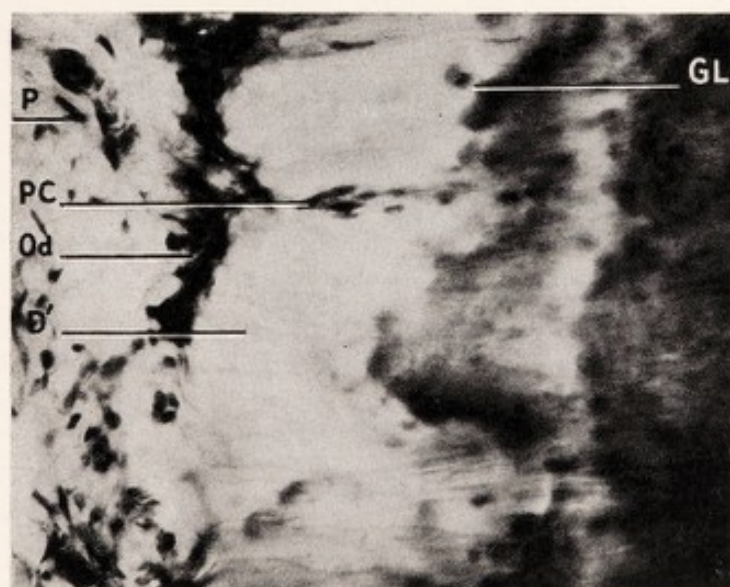


FIG. 370.—High magnification of odontoblasts and dentinoid in Fig. 369. P, pulp; Od, irregular odontoblastic layer; D', dentinoid; PC, pulp cells enclosed in the dentinoid; GL, irregularly distributed globules of calcification.

zone of dentinoid borders the dentin. A higher magnification of this area shows the great irregularity of the border between calcified and uncalcified dentin (Fig. 370). In one place the odontoblasts

have been destroyed by a capillary hemorrhage. At *PC* a group of pulp cells has been embedded into the dentinoid. The latter phenomenon, namely, the enclosure of cells or capillaries into the matrix, is frequently observed in cases of disturbed dentin calcification.

In case of rickets, the increased amount of dentinoid in the teeth is analogous to the broad seams of osteoid found in the bones of the skeleton. This uncalcified matrix is partly the result of delayed calcification of the normally formed matrix, and partly an actual hypertrophy of the matrix comparable to the excess osteoid in rachitic bone.

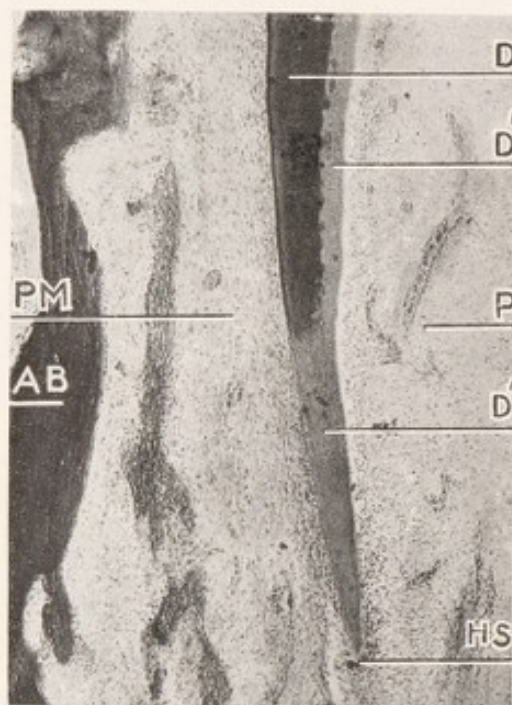


FIG. 371.—Root end of a deciduous molar of a rachitic child. Poor calcification of the dentin. The apex consists mostly of dentinoid. D, calcified dentin; D', dentinoid; HS, Hertwig's epithelial sheath; P, pulp; PM, periodontal membrane; AB, alveolar bone. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

The general distribution of the interglobular spaces in poorly calcified dentin is not always the same. In some rachitic teeth the interglobular spaces are arranged in rows under each area of hypoplastic enamel (Fig. 361); in others, especially in germs, the interglobular spaces are found all over the dentin, apparently without relationship to the overlying enamel.

In young teeth with developing roots the calcification of the root ends is considerably retarded if the individual suffers from rickets. Fig. 371 shows the open root end of a deciduous tooth of a rachitic child. A considerable portion of the root from Hertwig's sheath crownward is uncalcified. The beginning of calcification is indicated by the darker staining of the dentin compared with the dentinoid.

There is one basic difference between hypoplastic enamel and poorly calcified dentin: hypoplastic enamel is irreparable, but poor calcification of the dentin can be improved, at least to a certain degree, by additional precipitation of lime salts in the dentin. This can be shown beyond doubt in animal experiments and most likely occurs also in man. A condition necessary for such reparative additional calcification of rachitic dentin is that the pulp tissue be healthy and vital. Another very important point is that the formation of dentin matrix without subsequent calcification does not go on indefinitely. At first, matrix is formed as usual, but if no calcification takes place, matrix formation will gradually slow down and finally cease entirely. As a result, the teeth in severe rickets cases are considerably shorter than average normal teeth; in both crown and root portions dentin formation ends prematurely.

A very important clinical symptom in rickets is the delayed eruption of the teeth. This phenomenon can easily be demonstrated in animal experiments: the incisors of rachitic rats grow much slower than the incisors of normal control rats. Orban found that practically any kind of dietary deficiency retards the rate of eruption of the incisors. Bauer reported that in a rachitic puppy eruption of the permanent teeth was considerably delayed compared with a litter mate control. Hess made statistics concerning the time of appearance of the first tooth and found a marked delay in rachitic children; even in mild cases of rickets, the dentition is markedly delayed.

To a certain extent delayed eruption can be explained by delayed dentin formation and calcification at the open root end. In addition, however, there must be still another factor involved, since we know that all teeth have an inherent tendency to erupt and to move occlusally even after the roots are fully formed. Gottlieb offers the following explanation: the rate of eruption is to a certain extent influenced by the degree of calcification. The better calcified a tooth is the faster will it erupt; on the other hand, the poorer the calcification, the slower will be the eruption.

On the whole, we still know very little about the forces that play a rôle in the normal eruptive process, hence the great difficulty of interpreting abnormal manifestations under pathological conditions. From this viewpoint the recent experiments of Schour and Van Dyke are of great interest.

These authors succeeded in removing surgically the hypophysis of white rats; some of the animals operated upon were kept alive and observed for more than one year. It was found that the removal of the hypophysis was promptly followed by a considerable

retardation in the rate of eruption of the incisors; in some animals the eruptive movement came to a complete standstill. Since Hertwig's sheath at the root end continued to form dentin, the apical portion of the root formed multiple foldings of the dentin. Schour and Van Dyke were also able to speed up the eruption of the incisors in these hypophysectomized rats by the injection of the growth-promoting hormone of the pituitary body. When the same hormone preparation was injected into normal rats, it did not appreciably alter the eruption rate of the incisors. These experiments are of great value since they contribute to our as yet rather limited knowledge of the factors that control tooth eruption. We know that rickets delays the eruption of the human teeth as well as that of the rat's incisors. We also know that in certain disturbances of the glands of internal secretion, such as in myxedema, eruption is greatly delayed. The experiments of Schour and Van Dyke reveal a heretofore unknown relationship between function of an endocrine gland and tooth eruption; they show that removal of the hypophysis is associated with retarded eruptive movement and that administration of the hormone is followed by an increase in the rate of eruption.

BIBLIOGRAPHY.

- BAUER, W.: Die Veränderungen der Zähne und Kiefer bei experimenteller Hunderachitis, *Ztschr. f. Stom.*, 1925, **23**, 407.
——— Zur Entstehung der rachitischen Schmelzhypoplasien, *Vrtljschr. f. Zhk.*, 1929, **45**, 62.
BECKS, HERMANN: Histologic Study of Tooth Structure in Osteogenesis Imperfecta, *Dental Cosmos*, 1931, **73**, 437.
BECKS, HERMANN and RYDER, W. B.: Experimental Rickets and Calcification of Dentin, *Am. Jour. of Physical Therapy*, 1931, **8**, 358.
BLACK, G. V.: *Operative Dentistry*, Vol. I, Chicago, Medico-Dental Publishing Company, 1920, pp. 6-38.
V. EBNER, V.: Über die histologischen Veränderungen des Zahnschmelzes während der Erhärtung, *Arch. f. mikr. Anat.*, 1906, **67**, 18.
ERDHEIM, J.: Rhachitis und Epithelkörperchen, 90. Bd. d. Denkschriften d. math.-naturw. Klasse d. kais. Akad. d. Wissensch., Wien, 1914.
FLEISCHMANN, L.: Die Ursache der Schmelzhypoplasien, *Vrtljschr. f. Zhk.*, 1909, **25**, 868.
——— Rachitische Veränderungen des Dentins, *Vrtljschr. f. Zhk.*, 1910, **26**, 11.
GOTTLIEB, B.: Schmelzhypoplasie und Rhachitis, *Vrtljschr. f. Zhk.*, 1920, **36**, 71.
——— Rachitis and Enamel Hypoplasia, *Dental Cosmos*, 1920, **62**, 1209, 1316.
GYÖRGY, P.: Die Behandlung und Verhütung der Rachitis und Tetanie, nebst Bemerkungen zu ihrer Pathogenese und Ätiologie, Berlin, Springer, 1929.
HESS, A. F.: Rickets, Including Osteomalacia and Tetany, Philadelphia, Lea & Febiger, 1929.
HESS, A. F., and ABRAMSON, HAROLD: The Etiology of Dental Caries, *Dental Cosmos*, 1931, **73**, 849.

- HESS, ALFRED F., *et al.*: A Radiographic Study of Calcification of the Teeth from Birth to Adolescence, *Dent. Cosmos*, 1932, **74**, 1053.
- HOWE, P. R.: Dental Disorders Following Experimental Feeding with Monkeys, *Jour. Am. Dent. Assn.*, 1924, **11**, 1149.
- KLEIN, HENRY: Etiology of Enamel Hypoplasia in Rickets as Determined by Studies on Rats and Swine, *Jour. Am. Dent. Assn.*, 1932, **19**, 866.
- KLEIN, HENRY, *et al.*: Relation of Diet to Skeletal Development of Swine, Including Development of Teeth, *Jour. Am. Dent. Assn.*, 1930, **17**, 782.
- KOTANYI, E.: Besondere Befunde an Rachitischen Keimen, *Vrtljschr. f. Zhk.*, 1928, **44**, 87.
- KRONFELD, RUDOLF, and BARKER, F. J.: Experimental Study of the Influence of Vitamin D on the Hard Tissues of White Rats, *Jour. Am. Dent. Assn.*, 1932, **19**, 105.
- LIESEGANG, R. E.: Zur Kalkchemie des Zahnes, *Deutsch. Zahnärztl. Wehnschr.*, 1924, **27**, No. 10.
- LOGAN, W. H. G., and KRONFELD, RUDOLF: Development of the Human Jaws and Surrounding Structures from Birth to the Age of Fifteen Years, *Jour. Am. Dent. Assn.*, 1933, vol. **20**, March.
- MCCOLLUM, E. V.: *Newer Knowledge of Nutrition*, New York, The Macmillan Company, 1929.
- MELLANBY, MAY: Experiments on Dogs, Rabbits, and Rats, and Investigations on Man Which Indicate the Power of Certain Food Factors to Prevent and Control Dental Diseases, *Jour. Am. Dent. Assn.*, 1930, **17**, 1456.
- ORBAN, B.: Nutrition and Teeth, *Jour. Am. Dent. Assn.*, 1927, **14**, 1619.
- PRICE, W. A.: Calcium, Activation, Utilization and Metabolism, *Jour. Am. Dent. Assn.*, 1928, **15**, 729.
- Calcium Metabolism Studies on Nature and Rôle of Activators, *Jour. Am. Dent. Assn.*, 1929, **16**, 265.
- SCHÖNBAUER, FRANZ: Histologische Befunde an zwei kindlichen Kiefern nach Vigantoltherapie, *Arch. f. klin. Chir.*, 1930, vol. **160**.
- SCHOUR, I., and VAN DYKE, H. B.: Histologic Changes in the Rat Incisor Following Hypophysectomy, *Jour. Dent. Res.*, 1931, **11**, 873.
- Effect of Hypophysectomy on the Incisor of the Rat, *Jour. Dent. Res.*, 1931, **11**, 934.
- Effect of Replacement Therapy on Eruption of the Incisor of the Hypophysectomized Rat, *Proc. Soc. Exper. Biol. Med.*, 1932, **29**, 378.
- Changes in the Teeth Following Hypophysectomy. I. Changes in the Incisor of the White Rat, *Am. Jour. Anat.*, 1932, **50**, 397.
- SIEGMUND, H., and WEBER, R.: *Pathologische Histologie der Mundhöhle*, Leipzig, Hirzel, 1926.
- STEENBOCK, *et al.*: Cereals and Rickets, *Jour. Am. Med. Assn.*, 1929, **93**, 1868.
- TOJODA, MINORU: Beiträge zur Kenntnis der Dentin-Verkalkung, *Korr. f. Zahnärzte*, 1926, **50**, 374.
- TOVERUD, GUTTORM: *Experimental Studies on Physiological and Pathological Chemistry of the Teeth*, Oslo, 1926.
- TOYOFUKU, TOMAKI: Über die parathyreooprive Veränderung des Rattennagezahnes, *Frankf. Ztschr. f. path. Anat.*, 1911, **7**, 249.
- WILSON, GEORGE W., and STEINBRECHER, M.: Hereditary Hypoplasia of the Dentin, *Jour. Am. Dent. Assn.*, 1929, **16**, 866.
- WIRTH: Der Bau des Dentins bei hypoplastischen Zähnen, *Diss. Frankf. a. M.*, 1922.
- WOLBACH, BERT, and HOWE, P. R.: Effect of Deficiency of Antiscorbutic Factor in Guinea-pigs and of Fat-soluble A in Rats, *Jour. Am. Dent. Assn.*, 1926, **13**, 1592.
- ZSIGMONDY, OTTO: Beiträge zur Kenntnis der Entstehungsursache der Hypoplastischen Emaildefecte (Congenital Defects of Enamel), *Transactions of the World's Columbian Dental Congress*, Vol. I, Chicago, 1894, p. 48.

CHAPTER XVIII.

HISTOLOGY OF EDENTULOUS JAWS.

THE description of the histology of the human jaws after the loss of the natural teeth has not heretofore been considered material for a text-book on dental histopathology, but since it is the purpose of this book to give the microscopic counterpart of as many clinical manifestations and operations as possible, the author considers it advisable to include in this volume some of the more recent investigations on the minute structure of the edentulous jaws. These investigations were carried out in the Research Laboratory of the Chicago College of Dental Surgery by E. C. Pendleton.¹ While the study of this subject has by no means been concluded, a few of the specimens will be described and their significance to practical problems will be pointed out.

UPPER JAW.

1. **Clinical Description of Edentulous Maxilla.**—The clinical examination of an edentulous maxilla reveals several easily differentiated zones of varying tissue resistance that are important from the standpoint of the retention of artificial dentures. Using the alveolar ridge as the starting point for the clinical examination, a firm, resistant tissue of rather uniform thickness is found covering the crest of the ridge. This tissue extends from the maxillary tuberosity of the one side over the anterior part of the maxilla to the tuberosity of the other side and is from 4 to 8 mm. wide. In the diagram (Fig. 381) this area is marked *PS*. The loose and movable tissue of the vestibulum is attached to the buccal and labial periphery of the ridge and is subject to continual change during the movements of the lips and cheeks. *V* in the diagram shows the usual distribution of this type of tissue.

The tissue covering the anterior part of the hard palate (lingual to the crest of the edentulous ridge) is very dense and resistant.

¹ Figs. 372-381 reproduced from E. C. Pendleton's article on "The Anatomy of the Maxilla from the Point of View of Full Denture Prosthesis," Jour. Am. Dent. Assn., 1932, 19, 543.

Numerous transverse ridges radiating from the median raphé are usually found in this area. They are called *plicæ palatinae* or *rugæ palatinae*.

In the posterior portion of the hard palate the mucosa is smooth and elastic. The cushion-like submucosa in this area contains the palatine vessels and nerves and numerous glands; the presence of the latter can easily be recognized by the constant presence of moisture on the surface of the palatine mucosa.

In the median line of the hard palate, a more or less prominent ridge of great resistance and sometimes almost bony hardness is frequently found, indicating that here the lower surface of the hard palate is covered only by a very thin lining of soft tissue. This area, which is called the median raphé, palatine suture, or palatine torus, has an outline somewhat like the kernel of an almond with the tapered end pointing anteriorly. The antero-posterior length and width of this hard area are subject to great individual variations. Its average extension is marked in the diagram (Fig. 381, *R*).

The last area that we have to include in our clinical description is the posterior border of the prospective denture area. There the soft palate or velum is attached to the posterior margin of the hard palate. The line of attachment is plainly indicated by a difference in the appearance and motility of the tissues. The hard palate is grayish in color, while the soft palate is bright pink; the soft palate moves in deglutition and phonation, but the hard palate does not. The line between hard and soft palate runs on both sides in a slight curve from the distal surface of the maxillary tuberosity to the median line (region of the posterior nasal spine), the concavity of the curve pointing backward toward the soft palate (Fig. 381).

2. Histology of Edentulous Maxilla.—The general arrangement of the tissues of the palate in the median line is best illustrated by a sagittal section through an edentulous maxilla (Fig. 372). In the anterior part of the specimen a dense layer of fibrous connective tissue covers the edentulous maxillary ridge. Farther back toward the hard palate, the soft tissue lining of the palate becomes less fibrous, and fat tissue appears in the submucosa. At the height of the vault, the soft tissue covering the lower surface of the palatine bones is very thin, but from there back, the soft tissue rapidly becomes thicker and drops down, forming the transition to the soft palate. The bone extends backward in a horizontal plane from the height of the vault to the posterior nasal spine.

A higher magnification of the edentulous ridge taken of another jaw in the median line shows the attachment of the labial muscles

to the anterior surface of the alveolar process and the loose movable tissue on this surface (Fig. 373). The ridge itself is covered by fibrous connective tissue, but farther lingually fat tissue is present in the submucosa of the hard palate; the irregular outline of the surface is caused by cross-sections through the rugæ of the palate. In a labio-lingual section through the same jaw in the region formerly occupied by the cuspid, the rugæ, which consist histologically of ledges of densely interwoven fibrous connective tissue covered by typical stratified squamous epithelium, appear still higher and more definitely outlined (Fig. 374). The ridge itself is fibrous in structure; on the labial side the muscle attachment can be plainly

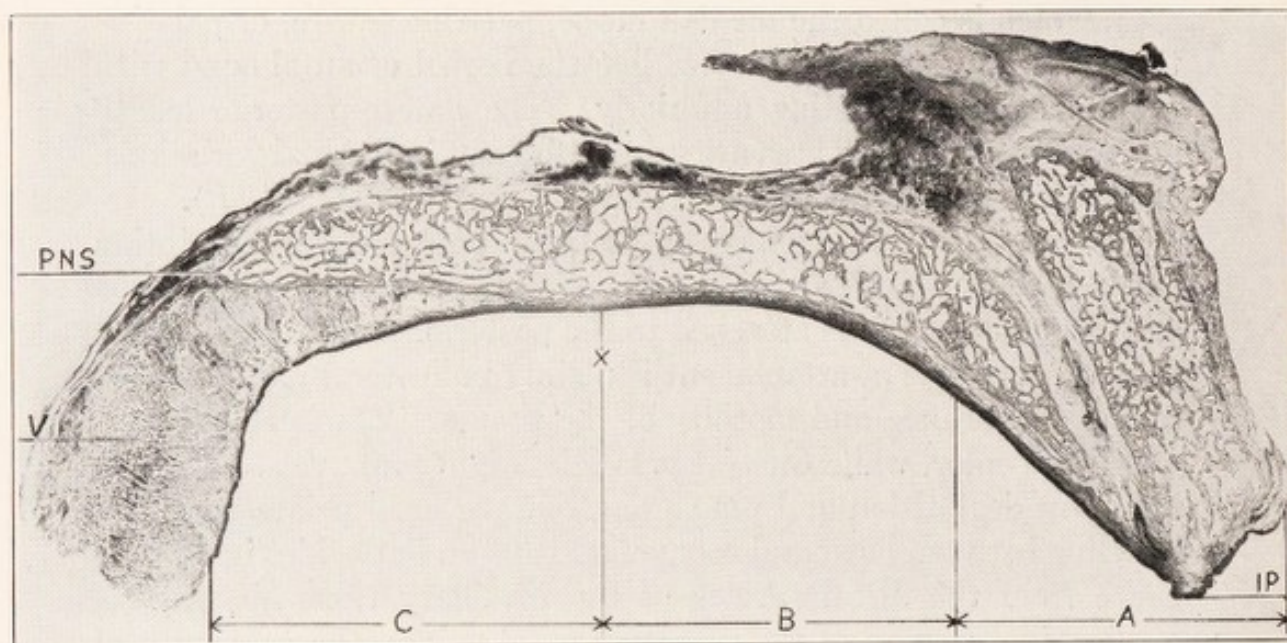


FIG. 372.—Sagittal section through the median line of an edentulous maxilla. A, area of maxillary ridge and incisive foramen; B, hard palate; C, soft palate; IP, incisive papilla; X, crest of ridge of maxillary suture; PNS, posterior nasal spine; V, velum.

seen. The deepest point of attachment of the labial muscles is found at a lower level than the height of the vestibulum, a fact of great clinical significance.

In a frontal section through the entire width of the edentulous maxilla, in the area of the upper first molar, the general distribution of the soft tissues over the bony skeleton of the palate can be studied (Fig. 375). On both sides, the buccinator muscle is attached to the outer plane of the maxilla. The crest of the bony ridge is covered by fibrous connective tissue. Lingually to the crest the bone recedes, forming an angle between the lingual surface of the alveolar ridge and the roof of the palate, in which angle the bloodvessels

and nerves of the palate are embedded. The mucosa is stretched from the crest of the ridge toward the vault, forming a triangular space on either side of the median line which is occupied by fat tissue and some mucous glands.

In the median line below the nasal septum, the soft tissue lining becomes rather thin, the submucosa consisting of fibrous elements



FIG. 373.—Labio-lingual section through the edentulous ridge near the median line in the area formerly occupied by the upper central incisors. ANS, anterior nasal spine; CT, loose connective tissue on labial side of ridge; V, vestibulum; MA, labial muscle attachment; MR, maxillary ridge; R, rugae; FT, fat tissue; IC, incisive canal; NS, nasal septum.

instead of fat tissue or glands. The number of glands increases considerably posterior to the first molar region. In a frontal section through the third molar area of the same jaw (Fig. 376), large masses of glands occupy the entire space between the median line and the alveolar ridges which are covered with fibrous connective tissue of considerable thickness. The bone on the buccal

side of the ridges is rather prominent, forming a distinct undercut on the buccal outline of this area. In the vestibulum a muscle attachment identical to that in the preceding illustrations is present.

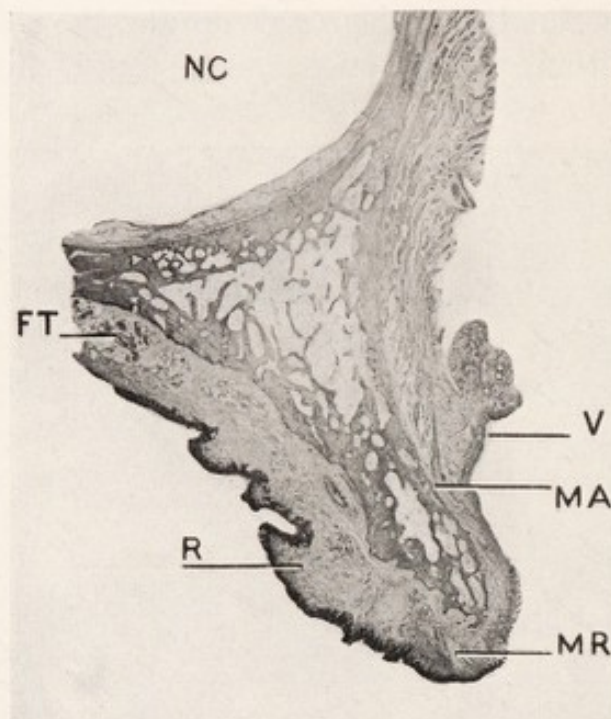


FIG. 374.—Labio-lingual section through edentulous ridge in cuspid region. MA, labial muscle attachment; MR, maxillary ridge covered with fibrous connective tissue; R, rugæ; FT, fat tissue; NC, nasal cavity; V, vestibulum.

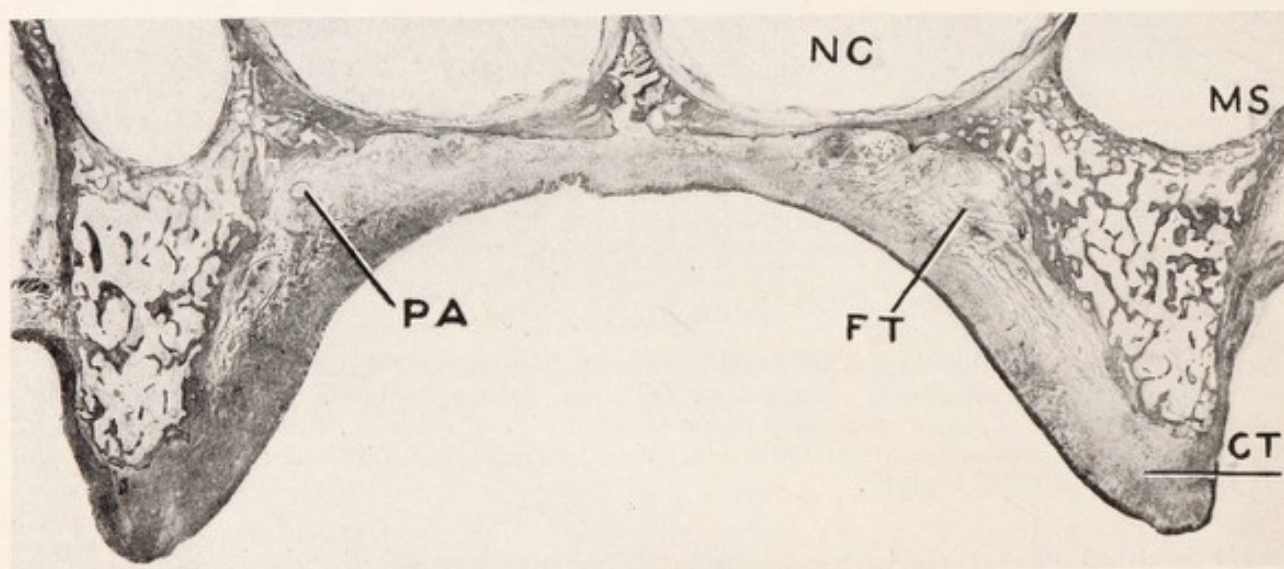


FIG. 375.—Bucco-lingual section through an edentulous maxilla in the region of the upper first molar. NC, nasal cavity; MS, maxillary sinus; CT, fibrous connective tissue on crest of ridge; FT, fat tissue of palate; PA, palatine artery.

In a frontal (bucco-lingual) section through the region of the second bicuspid in another jaw, the arrangement of the various

types of tissue is plainly visible (Fig. 377). On the buccal side of the ridge is the vestibulum and the attachment of the buccal muscles. The ridge is covered by fibrous connective tissue. Lingual

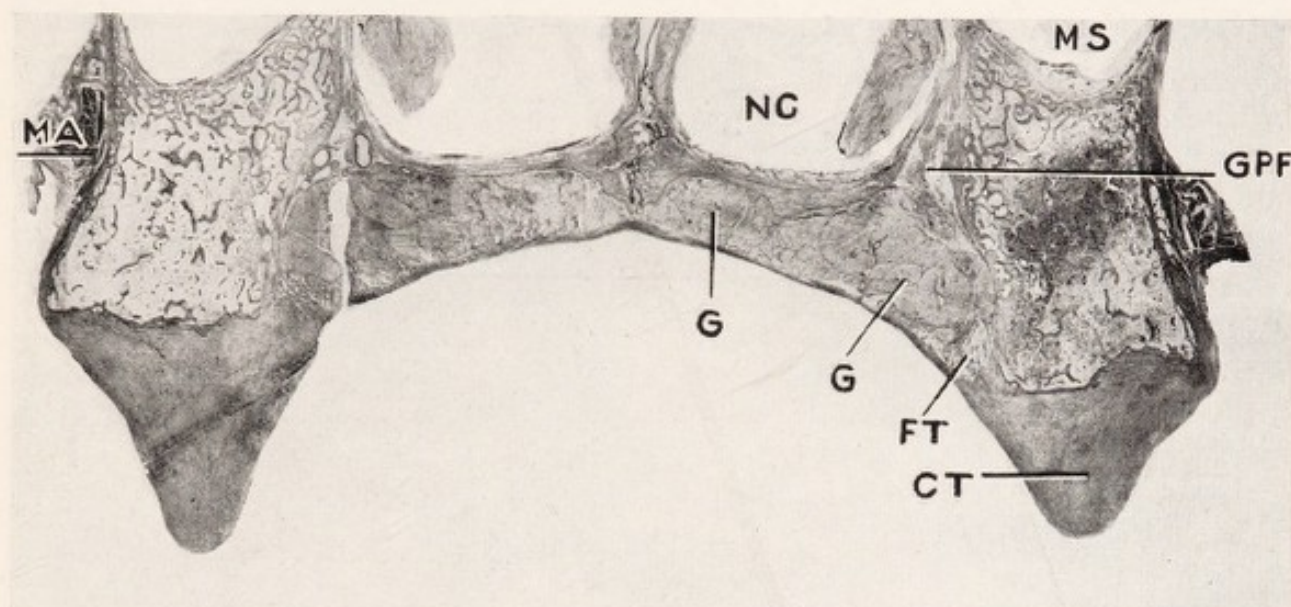


FIG. 376.—Bucco-lingual section through an edentulous maxilla in the region of the upper third molar. NC, nasal cavity; MS, maxillary sinus; GPF, greater palatine foramen; CT, fibrous connective tissue on crest of ridge; FT, fat tissue; G, glands of palate; MA, buccal muscle attachment (buccinator muscle).

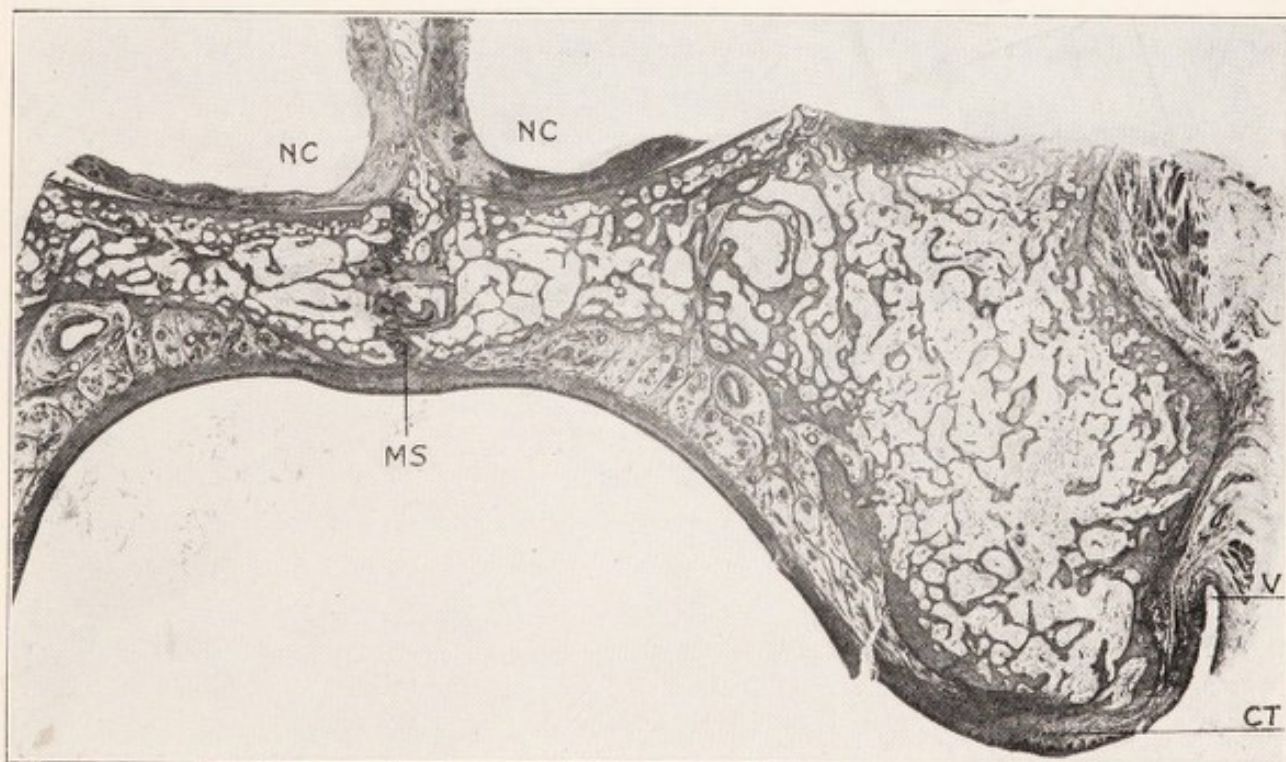


FIG. 377.—Frontal section through an edentulous maxilla in the region of the second bicuspid. NC, nasal cavity; MS, maxillary sinus; V, vestibulum; CT, connective tissue covering residual alveolar ridge.

to the ridge there is a triangular fat tissue area between the mucosa and the bone. From the submucosa, strands of fibrous tissue extend

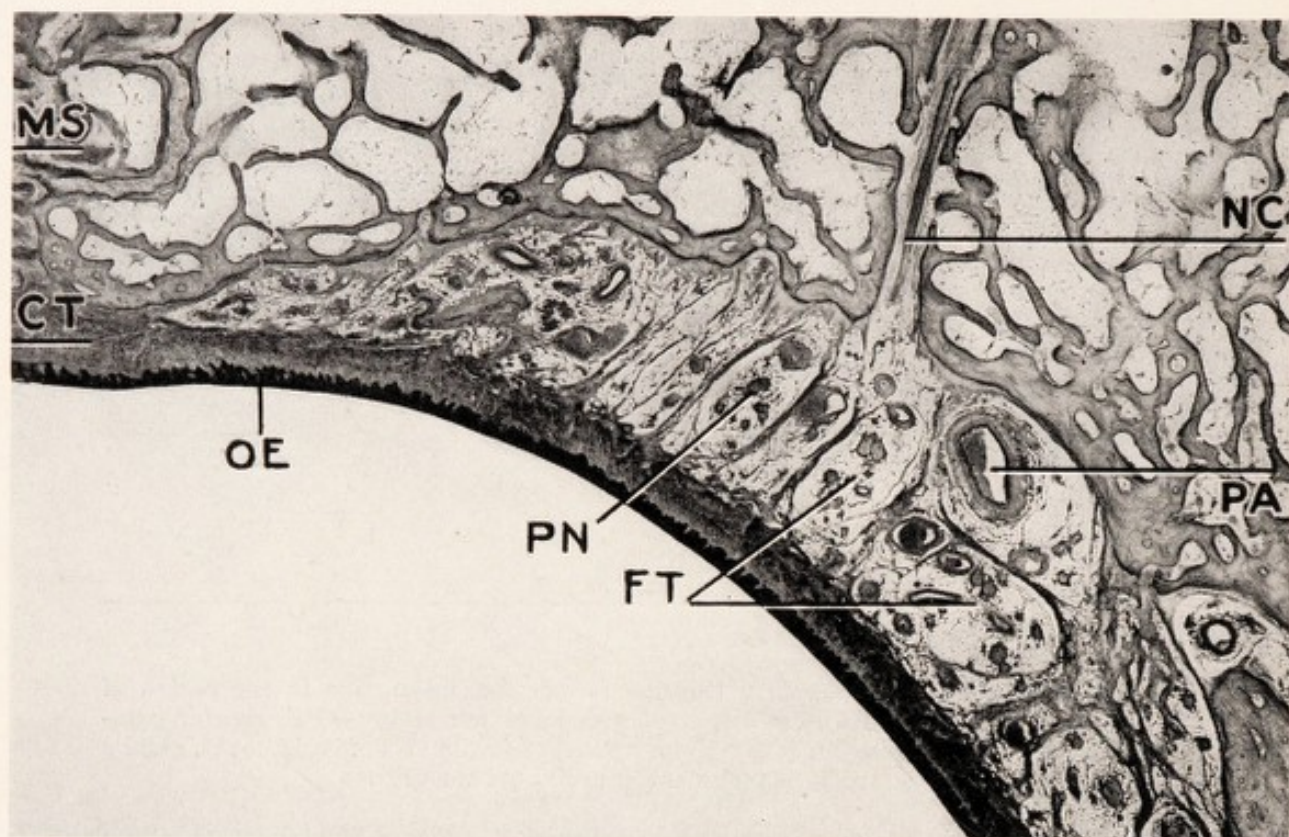


FIG. 378.—Higher magnification of Fig. 377. Area between maxillary ridge and maxillary suture. PA, palatine artery; PN, palatine nerves; FT, fat tissue; CT, fibrous connective tissue overlying the maxillary suture; MS, maxillary suture; NC, nutritional canal in the bone; OE, oral epithelium.

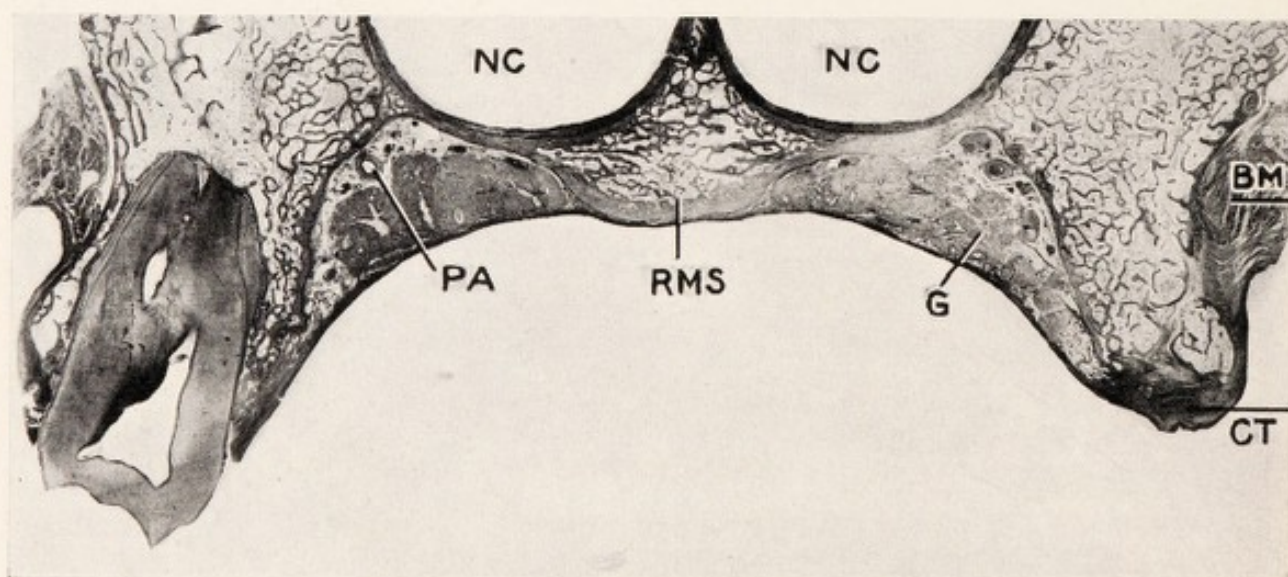


FIG. 379.—Frontal section through an upper jaw in the third molar region. The upper left third molar is still in place; the right side of the maxilla is edentulous. NC, nasal cavity; BM, buccinator muscle; CT, fibrous connective tissue; G, mucous glands of palate; RMS, bony ridge of maxillary suture; PA, palatine artery.

upward to the periosteum on the inferior surface of the palatine bones, dividing the fat tissue into several sections or areas. Embedded in each of these areas, the larger and smaller vessels and nerve bundles of the palate extend parallel to the bone surface (Fig. 378). Toward the median line the distance between oral mucosa and bone becomes smaller, only fibrous tissue being found there. No glands are present.

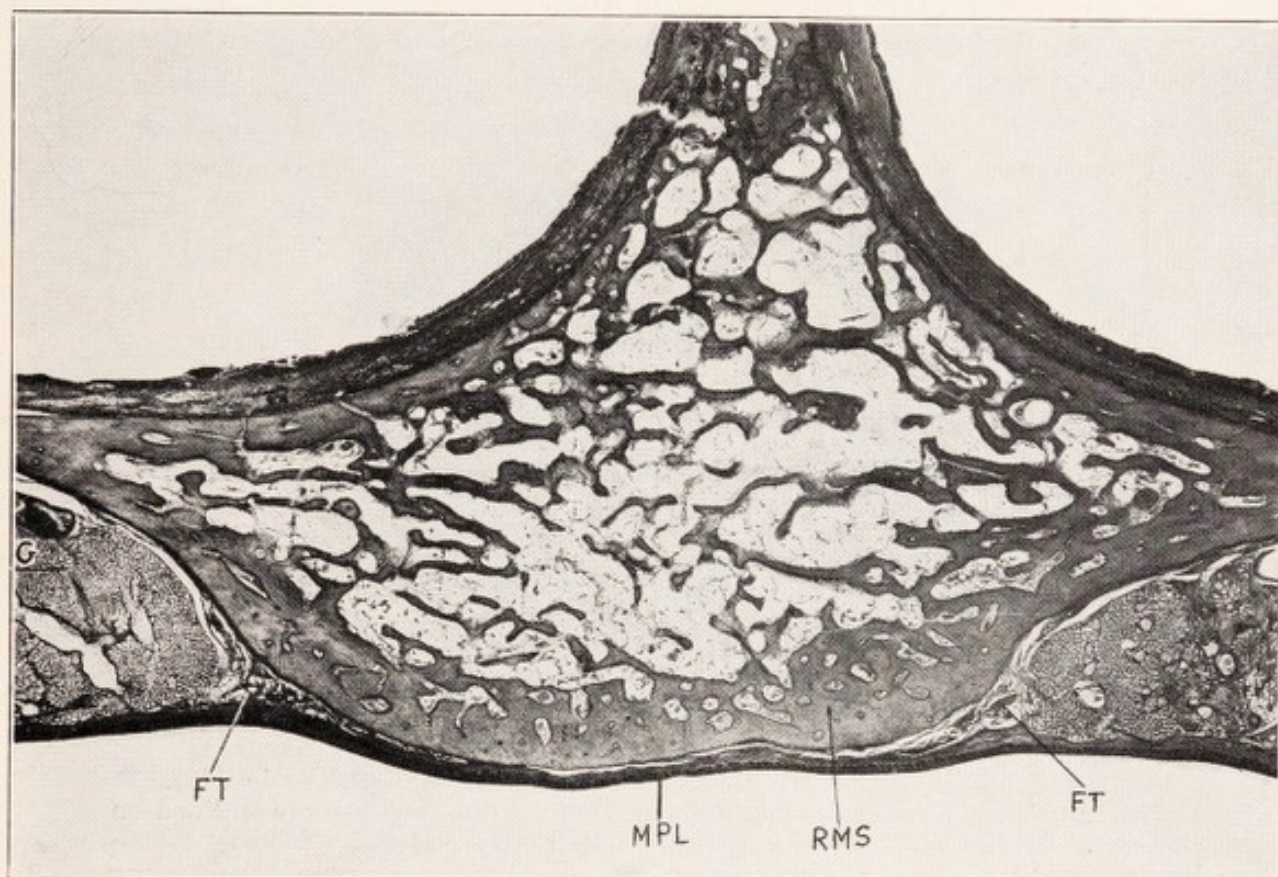


FIG. 380.—Higher magnification of the central portion of Fig. 379. Marked bony prominence in the median line of the hard palate. G, mucous glands of palate; FT, fat tissue; RMS, bony ridge of maxillary suture; MPL, thin mucoperiosteal lining covering the bone in the median line.

A frontal section through the third molar region of the same jaw shows the tooth still in place; the opposite side is edentulous (Fig. 379). In this field the most remarkable structures are the large masses of glands on both sides between ridge and median line and the very marked prominent bony ledge or raphé in the center of the hard palate. The depth of the soft tissue overlying the palatine artery on both sides is approximately 7 mm.; whereas, in the median line the soft tissues covering the bone are only about 0.3 mm. thick. The layer of glands is found on either side of the median raphé (Fig. 380).

3. **Practical Conclusions From Histological Findings in Edentulous Maxilla.**—The various clinical areas described at the beginning of this chapter are utilized in impression taking and denture construction in accordance with their physical properties. In clinical terminology the edentulous maxilla has been divided into four areas (Fig. 381):

- (a) Primary stress-bearing area.
- (b) Secondary stress-bearing area.
- (c) Valve-producing area.
- (d) Relief area.

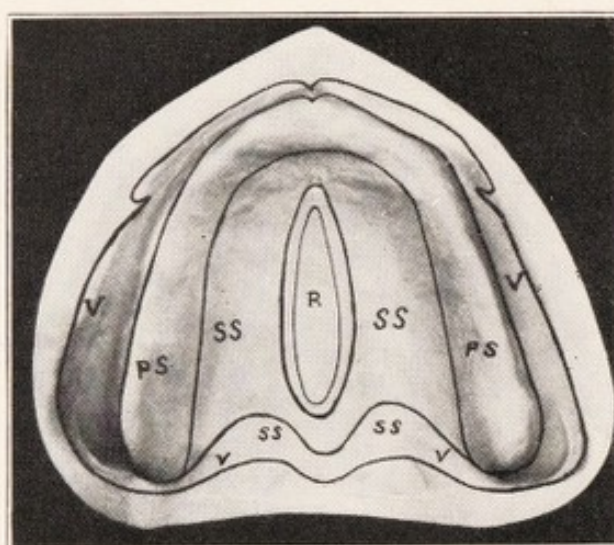


FIG. 381.—Plaster cast of the specimen from which the sections in Figs. 373 to 376 and Fig. 385 were prepared. The areas of denture retention are outlined on the cast. PS, primary stress-bearing area; R, relief area; SS, secondary stress-bearing area; V, valve-producing area.

(a) The primary stress-bearing area, which includes the entire maxillary ridge (Fig. 381, *PS*), carries the main load of the denture under masticatory stress. Histologically this area is characterized by the presence of dense fibrous connective tissue which is very firm yet resilient enough to stand considerable pressure without injury (see Fig. 377).

(b) The secondary stress-bearing area lies between the maxillary ridge and the median raphe (Fig. 381, *SS*); histologically it is represented by rugae in the anterior portion and by fat tissue and glands in the posterior portion of the maxilla. Due to variations in the degree of compressibility and to the vulnerability of the glands compared with the rather insensitive fibrous tissue on the ridges,

these areas are of secondary importance in overtaking the stress of the denture; however, the denture may rest upon them to a certain degree (see Fig. 378).

(c) The valve-producing area (Fig. 381, *V*) extends all around the maxillary ridge on the periphery of the primary stress-bearing area and along the posterior border of the hard palate. In histological specimens of edentulous maxillæ, the valve-producing areas on the labial and buccal sides of the alveolar processes and at the posterior borders of the hard palates are characterized by the presence of loose, movable tissues and muscle attachments. Loose connective tissue is unable to bear stress, but it adapts itself closely to the rounded margins of a denture and thus produces the airtight seal that is indispensable for good denture retention. The danger of overextension of the labial and buccal rims of a denture is easily understood from the arrangement of the tissues shown in Fig. 374. If the denture rim extends higher into the vestibulum than the deepest point of the loose muscle attachment, it will impinge upon the muscles and be displaced by their contractions.

(d) The relief area (Fig. 381, *R*) is the median area of the maxilla over the posterior part of the maxillary suture. From Fig. 380 it is apparent that the layer of soft tissue over the bone is often extremely thin in this area, and cannot, therefore, stand any amount of stress or pressure. Besides, if the denture rested firmly upon the unyielding ridge in the middle of the palate, it would rock over this area because all the surrounding tissues have a much greater compressibility. Therefore, care must be taken to relieve all pressure in the median line by special methods of impression taking.

It seems appropriate to say a few words about the practical significance of the fat tissue that is found lingually to the maxillary ridge and at the border between the hard and soft palate. The amount of this fat tissue varies according to the general amount of fat in the body. This explains the complaint of some patients that, after they have lost considerably in body weight, their formerly well-fitting dentures no longer fit. The reduction of fat tissue in the structures of the palate may easily account for this change in denture retention.

LOWER JAW.

The histological structure of the edentulous lower jaw is much less complicated than is that of the upper jaw. The even outline of bone in the average edentulous mandible and the absence of any

significant amount of fat tissue or glands in the denture area proper, make the description of the tissues of the mandible simple.

Clinically, only two distinct areas can be differentiated: the primary stress-bearing area, including the entire ridge, and the valve-producing area, represented by the tissue in the entire periphery of the primary stress-bearing area. Histologically the primary stress-bearing area consists of fibrous connective tissue covering the crest of the edentulous mandible. The bucco-lingual width of this

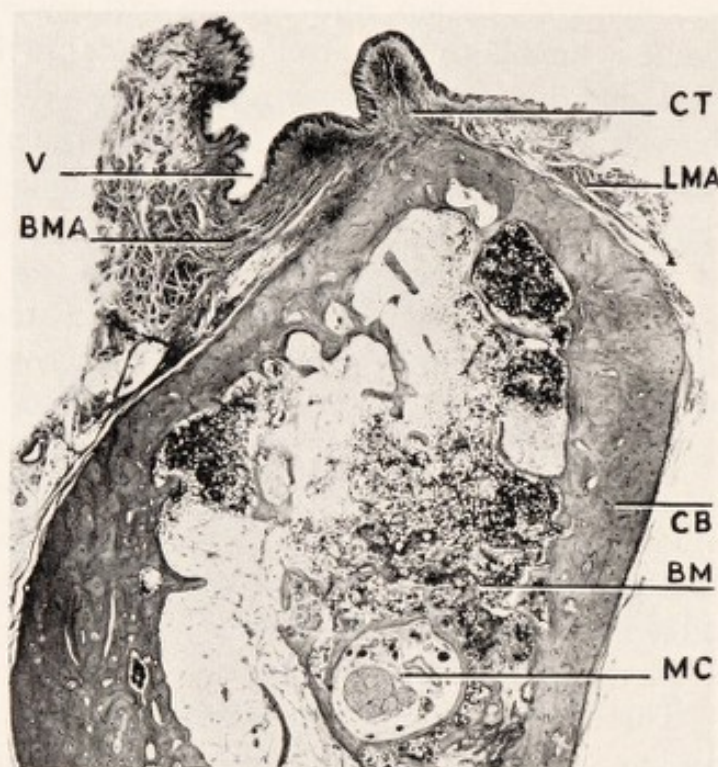


FIG. 382.—Bucco-lingual section through an edentulous mandible in the molar region. V, buccal vestibulum; BMA, buccal muscle attachment; CT, connective tissue of residual ridge; LMA, lingual muscle attachment; CB, compact bone on the surface of the mandible; BM, bone marrow; MC, mandibular canal containing blood-vessels and nerves. (Courtesy of E. C. Pendleton.)

area varies from 4 to 8 mm. in different jaws. On the labial as well as the lingual side, muscles are attached close to the ridge, and these movable muscle attachments together with overlying, loose connective and some fat tissue form the valve-producing area for the lower denture (Fig. 382).

At the present time the histology of the edentulous mandible and its relation to the problem of full denture retention is the subject of extensive investigations. The results of these investigations will be published at a later time.

BIBLIOGRAPHY.

- BRINCH, OVE: Histologische Untersuchungen über pathologisch-anatomische Veränderungen im Gaumengewebe prothesentragender Patienten, *Vrtljschr. f. Zhk.*, 1932, **48**, 120.
- GROHS, R.: Anatomisch-histologische Grundlagen zum Aufbau einer totalen Prothese mit besonderer Berücksichtigung des Abdruckverfahrens, *Ztschr. f. Stom.*, 1931, **29**, 675.
- MENTZ, E.: Histologische Untersuchungen über den Bau der Mundschleimhaut und ihre Beziehungen zum Zahnersatz, *Deutsch. Mon. f. Zhk.*, 1931, **49**, 66.
- PENDLETON, E. C.: Histologic Study of Soft and Hard Tissues of Edentulous Maxillæ, and Their Relation to the Problem of Full Denture Retention, *Jour. Dent. Res.*, 1931, **11**, 490.
- The Anatomy of the Maxilla and Its Relation to the Problem of Full Denture Construction, *Illinois Dent. Jour.*, September, 1931.
- The Anatomy of the Maxilla from the Point of View of Full Denture Prosthesis, *Jour. Am. Dent. Assn.*, 1932, **19**, 543.

CHAPTER XIX.

REMARKS CONCERNING HISTOLOGICAL TECHNIQUE.

IN the extensive field of normal and pathological histology of the human body, the dental structures occupy a unique position. In no other part of the organism does there exist a similar close relationship between soft tissues and calcified structures; in no other organ except in the tooth is the histologist faced with the necessity of preparing sections containing both highly calcified hard tissue and the enclosed and surrounding soft tissues. Due to the great technical difficulties associated with the preparation of such sections, many of the important findings and discoveries in the field of dental histology and pathology have been made very recently. Only within the last few years has it been possible to prepare perfect undistorted serial sections through human teeth and jaws. The histological technique necessary to obtain such sections will be outlined. First, however, some data concerning the principles of histological tissue fixation will be given.

POSTMORTEM CHANGES IN TISSUES. TISSUE FIXATION.

The cells of every tissue undergo degenerative changes following the death of the organism or the removal of the tissue from the organism. These postmortem changes at first consist of a coagulation of the protein substances and of the protein-containing body fluids (blood clot formation). In this stage, the arrangement and structure of the cells is but little altered; examination under the microscope gives very good information about their condition during life.

Later fermentation occurs in the tissue. The cells undergo decomposition, losing more and more of their original structure and appearance. Finally, the tissue is subjected to putrefaction by the action of saprophytic microorganisms. Fermentation and putrefaction destroy all cell details of the tissue.

The rapidity with which these changes occur depends upon several circumstances. Temperature plays a very important rôle. If the

dead tissue is kept at a temperature close to freezing, fermentation and decomposition of the cells can be considerably delayed. The presence of pyogenic microorganisms is another important factor; septic tissue is decomposed much faster than normal tissues. A more detailed description of postmortem cell changes may be found in any text-book on general pathology.

In order to prevent postmortem cell changes, it is necessary to preserve the tissues for histological examination as soon as possible after death, thereby making fermentation and bacterial activity impossible. This is done by treating and impregnating the tissues with a fixing solution, of which there are several different kinds in use. Although these solutions vary widely in their chemical composition, they all have in common the property of coagulating or hardening tissues, as well as distinct bactericidal properties. A few of the most commonly used fixing fluids are: formalin (aqueous solution of formaldehyde), alcohol, acids (glacial acetic acid, osmic acid), solutions of metal salts (mercury bichlorate, potassium bichromate). These solutions may be used either singly (as, for instance, formalin) or they may be mixed and combined so that the most desirable properties of each fixing fluid can be utilized. By the process of fixation the cells are hardened and the cell details, especially the nuclei, are preserved in their original size and structure. It must be understood, however, that every tissue studied under the microscope has undergone some changes in fixation and staining; these changes have been demonstrated by comparing living cells in tissue cultures with the same cells after they had been subjected to the usual fixation and staining methods.

TYPES OF DENTAL TISSUES USED FOR HISTOLOGICAL SPECIMENS—FIXATION AND DECALCIFICATION.

The tissues used for the study of both normal and pathological dental conditions may be classified in three groups: (1) Extracted teeth, decalcified or ground sections; (2) teeth and surrounding soft and hard tissues of the jaws, preserved and sectioned in their original arrangement and relationship; (3) isolated soft tissue removed from the teeth (pulp) or from the jaw (gingival tissue).

As to the applicability of these various forms of specimens to the different problems of dental research, the following brief data may serve: ground sections are used for the study of the enamel, the dentin, and the cementum, occasionally also for the study of the bone. G. V. Black, a master in the preparation of ground sec-

tions, has fully shown the possibilities presented by these sections in the investigation of dental caries. However, for the examination of the dental soft tissues, such as pulp, periodontal membrane, and gingiva, ground sections are of little value. Although it is possible to preserve and recognize some of the soft structures in carefully prepared ground sections, still the results obtained from microtome sections are so far superior that the use of ground sections for the study of soft tissues has been practically abandoned.

For the study of normal and pathological dental conditions, decalcified sections through both hard and soft tissues in their undisturbed relationship are indispensable. Only by the use of such specimens is it possible to understand the changes in the pulp, periodontal membrane, and gingival tissues. Only such sections offer the opportunity to study tooth and periodontium as a whole, as a structural and physiological unit. Figs. 133 and 266 are sufficient proof of this statement.

The third group, soft tissues isolated from the hard structures, are of minor importance. A pulp, when removed from the pulp canal, or a piece of gingival tissue may be of a certain diagnostic value. But as these tissues are usually badly torn, they cannot convey a clear idea of their original relationship to the tooth. Therefore, the use of such specimens should be limited to exploratory excisions of soft tissue when a small piece is needed for an immediate diagnosis.

In this book, ground sections were used only to illustrate some of the structures and conditions of the enamel and dentin (Chapters I and III). These ground sections were prepared in the usual way and embedded in Canada balsam. Some of them are unstained, others stained. The majority of the specimens reproduced in this book, however, were obtained by decalcifying and sectioning parts of jaws containing teeth and surrounding hard and soft structures.

For the study of pulp conditions (normal pulp, secondary dentin, denticles, pulpitis) and of caries of the dentin, extracted human teeth were used. Immediately following extraction, the apical portion of the root was cut into or broken off in order to facilitate the penetration of the fixing fluid, and the tooth was dropped into a bottle containing an 8 or 10 per cent solution of formaldehyde. This concentration of formaldehyde is somewhat stronger than is generally used for the fixation of soft tissues; however, the presence of a layer of hard tissue (dentin) around the pulp requires a more penetrative solution than is necessary for the fixation of other soft tissues. For special staining methods, fixation with Zenker-formol

was used. Figures 85 to 93, for instance, show that pulps of teeth treated in this way fill the entire pulp chamber without preparatory shrinkage or retraction from the walls, thus giving a correct picture of the relationship between pulp and dentin. All cell details are preserved.

These illustrations also prove that specimens of pulps prepared by sectioning the entire tooth with the pulp *in situ* are far superior to specimens obtained by the antiquated method of cracking open the tooth in a vise and removing the pulp for sectioning. This latter method dates back to the time when celloidin embedding was unknown; at that time sectioning an entire calcified tooth including the pulp presented a problem so difficult that most histologists avoided it whenever possible.

For the microscopic examination of normal and pathological conditions of the tissues surrounding the tooth, specimens consisting of pieces of jaw with teeth are indispensable. Such specimens are obtained almost exclusively by autopsy. Only occasionally is it possible to remove by surgery one or several teeth with the surrounding bone and soft tissue (see Fig. 128). By far the majority of the specimens illustrated in this book were removed from human bodies shortly after death; specimens from more than forty different jaws are represented. The usual procedure is that, from six to twenty-four hours after death, both the upper and the lower jaw are removed with saw and chisel and immediately submerged in 10 per cent formalin. After having been kept there for several weeks, the specimens are transferred into 95 per cent alcohol in which they may remain any length of time. This form of fixing entire jaws gives very good results; in many instances, even the finest details of the cell structure can be recognized. In other specimens, minor postmortem changes have taken place, which, however, do not interfere with the diagnosis and the general arrangement of the tissues.

The ages and oral conditions of the individuals from whom the jaws were removed vary greatly. The youngest jaws from which specimens were used in this book were aged two and one-half and four and one-half years (resorption of deciduous teeth, Figs. 190 and 191). The oldest jaws, aged forty-five and fifty-eight years respectively, were used for the study of the histology of edentulous jaws (Chapter XVIII). The other jaws, which were obtained from individuals of all ages, contain a great variety of normal and pathological dental conditions in the same proportion as these conditions are usually encountered in the clinical examination of human mouths.

Radiographs are taken of the jaw specimens and later are compared with the actual microscopic findings. Pulpless teeth with infected root canals are a very common finding, making possible an extensive study of the histopathology of periapical inflammation and its relation to the surrounding bone (see Figs. 114 to 125). The occlusal conditions were recorded by means of photographs and plaster casts so that the tissue reactions caused by the various occlusal forces could be evaluated (see Chapters XII and XIII). The gingival tissues can be seen in their varying relationship to the tooth surface in youth and age, under normal and under pathological conditions. Practically every known manifestation in the field of dental pathology is occasionally encountered in human jaw specimens, and it is possible to study these conditions in their original position. As an example, attention may be called to the occurrence of root fractures (Figs. 351 and 355) and of impacted teeth (Figs. 326 to 333) in our specimens.

In order to section jaw bone and teeth with the microtome, it is necessary first to decalcify these hard tissues, for which purpose a 5 per cent aqueous solution of nitric acid is used. The time of decalcification in this acid is from three to twelve days, depending upon the degree of calcification and the size of the specimen. Acid stronger than 5 per cent decalcifies faster, but has a destructive effect upon the soft tissues. When decalcification is complete, the enamel of fully developed teeth has been entirely dissolved; dentin, cementum, and bone, however, due to their comparatively high content of organic matrix, retain their original form.

EMBEDDING, SECTIONING, MOUNTING, AND STAINING SPECIMENS.

After decalcification the specimen is transferred into a 5 per cent aqueous solution of sodium sulphate to prevent swelling of the connective tissue. Then it is rinsed in water. The hard tissues are now in condition to be cut by the microtome. However, in order to offer sufficient and uniform resistance to the knife, it is necessary to increase the consistency of the specimen to a cartilage-like firmness. This can be done in two ways: by freezing or by embedding.

Freezing specimens by means of compressed carbon dioxide is practised largely in general pathology for the purpose of quickly obtaining diagnostic sections; for dental tissues it is not practical because of the impossibility of obtaining large sections and of keeping serial sections. Therefore embedded specimens are used

almost exclusively for the microscopic study of dental structures. As embedding materials, paraffin or celloidin (collodion) are used. For large sections through tissues varying in firmness (dentin, bone, connective tissue, fat, muscle, and epithelium are frequently present in the same section through a piece of jaw) paraffin is rather difficult to use and is likely to produce tears and distortions; therefore, celloidin is the chosen embedding material for the routine procedure of making serial sections through large pieces of human jaw material.

The author has used in his work the method of embedding, sectioning, and staining that was worked out by Professor Gottlieb and his associates in the Histological Laboratory of the Dental Institute of the University of Vienna. With the exception of some insignificant modifications, this method has not been changed during the last eight or ten years because its results have been far superior to the ones obtained with any other technique. The procedure will be outlined briefly here.

Celloidin is a solution of a special kind of collodion in a mixture of equal parts of absolute alcohol and ether. The specimen must be completely impregnated so that all cells and tissue spaces are filled with celloidin, giving the specimen a uniform firmness throughout. Since water and celloidin are physically incompatible, the specimen must first be entirely free of water. This is done by slowly carrying the specimen through alcohols of rising concentration (50, 75, and 95 per cent, and absolute alcohol). After the highest concentration of alcohol has been reached, the specimen is transferred into a mixture of equal parts of absolute alcohol and ether. It is now entirely free of water and ready to be permeated by celloidin. For this purpose the specimen is transferred into solutions of celloidin of rising concentration. The first solution is 0.5 per cent, that is, it contains one-half part dry celloidin to each 100 parts absolute alcohol-ether. The next concentration is 1 per cent, then 2, 4, and finally 8 per cent. The 8 per cent solution of celloidin has the consistence of thick honey. Absolute alcohol, alcohol-ether, and all solutions of celloidin must be kept over anhydrous cupric sulphate to remove any trace of water that might still remain in the specimen.

The time necessary for dehydrating and embedding a specimen varies from two weeks to three months, depending upon its size. The longer the specimen is kept in each concentration of celloidin, the better it will be impregnated and the better will be the result. Too great haste will prevent sufficient penetration, resulting in difficulties and tearing during sectioning.

After the specimen has gone through the 8 per cent solution of celloidin, it is mounted on a fiber block, hardened in the desiccator under chloroform vapor, and sectioned with the microtome. The microtome consists of a heavy base carrying the holder for the specimen and a horizontal sliding track for the knife. After each cutting, the specimen block is raised automatically to the thickness required, and is then in readiness for the next cutting movement. The thickness of the individual sections containing teeth and bone varies between 15 and 30 microns ($1 \text{ micron} = \frac{1}{1000} \text{ mm.}$), depending upon the size of the specimen and upon the fixation. The smaller the specimen and the better the fixation, the thinner the sections that can be obtained. All sections are taken from the knife as they are cut and are immediately mounted on glass plates

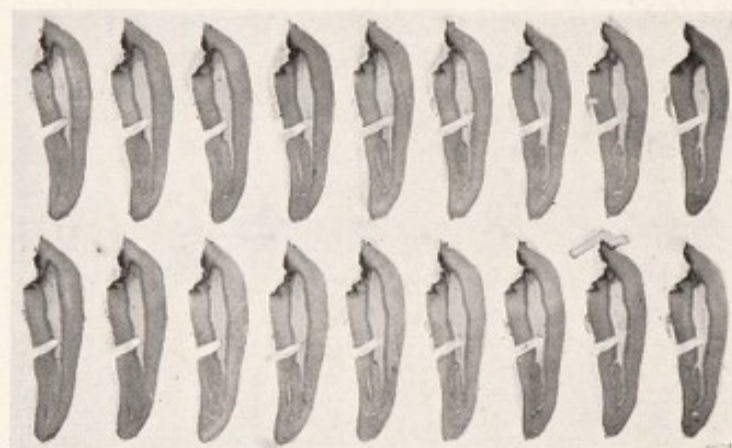


FIG. 383.—Histological slide of 18 serial sections through a lower bicuspid with deep caries and partial pulpitis. Original size of specimens. The cut in the root was made immediately after the extraction of the tooth to make a rapid penetration of the fixing fluid into the pulp tissue possible. A higher magnification of one of these sections is reproduced in Fig. 88.

in the order in which they were sectioned. Such sections are called serial sections; if all the sections of a series were laid upon one another, the original specimen could be reconstructed. The use of serial sections makes it possible to gain a tridimensional insight into the specimen that has been sectioned; by studying the sections in the order in which they were cut, one can get a bodily picture of the organ or structure.

After the sections are fastened on the glass slides with a thin collodion solution, they are stained. Most of the sections illustrated in this book were stained with hematoxylin or hemalum and eosin; some specimens were stained with silver stains (Bielschowsky's silver stain) to demonstrate the arrangement of the fibrous connective tissue (Fig. 273). After staining, the slides are differentiated

in alcohol and cleared in terpeneol. Cover-slips are then mounted on the slides with a heavy solution of dammar resin in xylol.

A few histological slides carrying serial sections of human specimens will be reproduced here in original size. Some of these sections have been shown in higher magnifications in different chapters of this book. Fig. 383 is a photograph of a series of sections through an extracted lower bicuspid with caries and beginning pulpitis. For a higher magnification of the inflamed pulp see Fig. 88. In the



FIG. 384.—Histological slide of 9 mesio-distal serial sections through the upper second bicuspid and the first and second molars. Original size of specimens. Due to the divergence of the roots, the lingual root of the second molar is the only one that appears in central sections in this particular slide. The buccal roots of the molars and the root of the bicuspid are visible in other slides of the same series. The crowns of both molars are extensively decayed; a large abscess cavity has developed in the bone around the root ends. A higher magnification of one of these sections is reproduced in Figs. 130 and 131.

root is a cut that was made with a lightning disk immediately after extraction to facilitate the access of the fixing fluid to the pulp.

The necessity of keeping serial sections cannot be overemphasized. Suppose we were to decide whether in a specimen of a pulpitic tooth, like the one illustrated in Fig. 383, the pulp is exposed. If the area of pulp exposure were large, this question would be easy to decide. If, however, the pulp were exposed in only one minute spot, any number of histological sections that show the pulp covered by

dentin might be obtained, and only very few sections would really show the area of exposure. If only one or two sections were prepared, the chances are that the point of exposure of the pulp would not be included in the sections. But if a complete series of sections is preserved, it will always be possible to recognize, beyond doubt, the actual condition of the pulp.

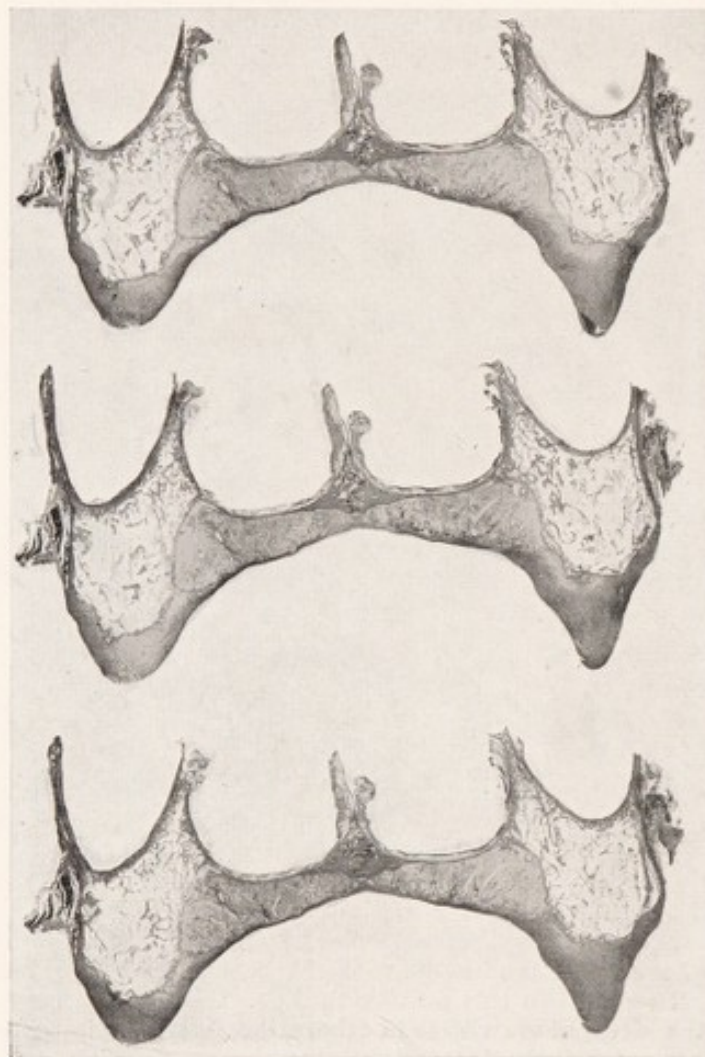


FIG. 385.—Histological slide of 3 frontal serial sections through the entire width of an edentulous maxilla in the region of the third molar. Original size of specimens. Higher magnifications of sections similar to these are reproduced in Figs. 375 and 376.

Fig. 384 is a photograph of a slide with nine mesio-distal sections through a portion of the maxilla containing the second bicuspid and the first and second molars. Both molars have decomposed infected pulps, and around the root ends a large abscess has destroyed the surrounding bone. This abscess cavity can be recognized even in this low magnification. For higher magnification of this specimen, see Figs. 130 and 131.

Finally a slide with three frontal sections through an edentulous maxilla is reproduced. Each section is about 6.5 cm. long and 3 cm. wide, and includes the palate, the edentulous ridges, and the floor of the nasal cavity and of the maxillary sinus. Sections of this type were used for the study of the histology of edentulous jaws (see Chapter XVIII).

These illustrations may give an idea of the progress that has been made in the technique of sectioning dental tissues. Only a few years ago good sections through more than one tooth were practically unknown. Nowadays there is practically no limit as to the type and size of human jaw tissue that can be sectioned in flawless serial sections, preserving all microscopic cell details. With the aid of such specimens, work is now going on toward the solution of a great number of problems in dental and oral pathology.

BIBLIOGRAPHY.

- BILLIG, AMALIE: Erfahrungen bei der Herstellung von Celloidinserien besonders grosser Objekte, *Ztschr. f. Stom.*, 1925, **23**, 136.



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