

## HISTOLOGICAL AND PATHOLOGICAL STUDIES OF THE PERIDONTAL MEMBRANE, CEMENTUM, AND DENTIN WITH REFERENCE TO INFECTIONS

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(Read before the National Dental Association, Boston, Massachusetts, August 23-27, 1920)

EXCLUDING unusual traumatic lesions, it is generally accepted that there are two types of inflammation and suppuration occurring in the peridental membrane. One has its beginning, apparently, at the gingival line of the tooth following a gingivitis, the other, at some point within the membrane following the death or removal of the pulp.

The latter are usually considered apical in location and are so named. In a great many cases, and insofar as subsequent tissue involvement occurs about the apex in others, this usage may not be incongruous. Foramina are, primarily, the pathway by which infection from the pulp reaches the investing tissues. These are not limited to the apical region only and may well be the source of inflammation and suppuration in other parts of the membrane, rendering the term "apical" inadequate.

In presenting the subject announced, it will not be my purpose to enter into a detailed description of all the changes that take place during the course of these lesions. I shall limit myself chiefly to a consideration of the structure and physiology of the teeth and the investing tissues, which appear to be related to the extension and treatment of these diseases.

In the light of recent work alone, which has been done on the tissues of

the tooth, indicating their permeability not only to nutritional fluids but to infective products, and its consequent bearing on the treatment of suppurative conditions about the roots, it is to be hoped that this discussion will be of interest and profit.

### THE INVESTING TISSUES

In the longitudinal section thru tooth and investing tissues, we note the dense layer of epithelium of the exposed surfaces of the gum and gingivae. Here the cells are compact, with the surface layers more or less hardened, thereby better resisting friction and affording protection to all of the underlying structures. Lining the inner surface of the gingivae, i.e., the surface which rests against the enamel or in the gingival space, is an epithelium that is less compact, exhibiting no stratum corneum and offering less resistance to infection. Deeper down the solid mass of fibers of the peridental membrane, attached to the cementum, pass out into the gingivae or to the bone of the alveolar process. Lying upon the surface of the cementum, between the ends of the fibers, may be found cementoblasts which are the formative cells of the cementum, as on the surface of the bone of the alveolar process may be found osteoblasts with a like function. Coursing thru the membrane, about midway between the ce-

mentum and the alveolar process, are groups of blood vessels and nerves. For the most part these run parallel to the long axis of the tooth. To the outer walls of these blood vessels and nerves are attached the minute lymphatic spaces which drain the gingivae and peridental membrane. (15, 17).

The general principles upon which the lymphatics functionate should be remembered. Serum is delivered to all of the connective tissues from the minute capillaries of the blood system to serve as their nutritive fluid. This tissue fluid of lymph, added to by the products of metabolism of the tissues, is gradually drawn into, chiefly, lymph spaces and channels, to be carried finally thru the lymphatic system. In conjunction with the blood system, the lymphatics afford a drainage for the tissues. According to one writer, the latter are purely scavenger (11, 18, 19).

We may say that no connective tissue is without its drainage system, but not until Schweitzer (17), substantiated by Noyes and Dewey (15), demonstrated the presence of these vessels in the peridental membrane and pulp could we say positively that those structures were drained by lymphatics. As these accompany the blood vessels we must note the distribution of the latter.

The blood supply of the peridental membrane is threefold. Vessels pass thru the gum tissue on the outside of the bone of the alveolar process, arching over the crest of the process, to pass down thru the membrane toward the apex of the root. A second source of supply is received from one or more vessels which enter the membrane about the apex, and splitting up into six or more branches, they enter the pulp or pass thru the membrane toward the crown of the tooth. The third group consists of vessels which pass thru the bone of the alveolar process to the membrane on the inner surface. (13, 8).

The lymphatic channels accompany these vessels, the flow of the lymph be-

ing toward the apex. Those which have their beginning under the epithelium lining the gingival space and which drain this part of the gingivae and the tissues about the neck of the tooth, pass down thru the membrane toward the apex (15). Those which drain the gum tissue and the outer surface of the gingivae pass down on the outer surface of the alveolar process toward the apex (16).

#### PATHOLOGY

If the course of the lymphatics and the clinical picture be compared, it is interesting to note the correlation between them. The fact that most of these vessels run parallel to the long axis of the root explains the symmetry of the pus pockets. If the majority of the vessels encircled the tooth, the infection would be carried around the root more rapidly than toward the apex, and the pockets would be broad and shallow rather than deep and narrow.

In those infections caused by deposits of salivary calculus which are located in the gum (14, 1), the inflammatory reaction appears to follow the lymphatics attached to those vessels which course thru the gum on the outside of the alveolar process, supporting the evidence that this form of deposit does not cause the formation of pus pockets along the side of the root.

In the earlier appearance of the inflammatory process arising from salivary calculus irritation, the outer border appears to be the portion of the alveolar process primarily affected; while in those irritations affecting the tissues of the gingival space, and which travel down thru the membrane, the inner or membrane surface of the process is first attacked (14).

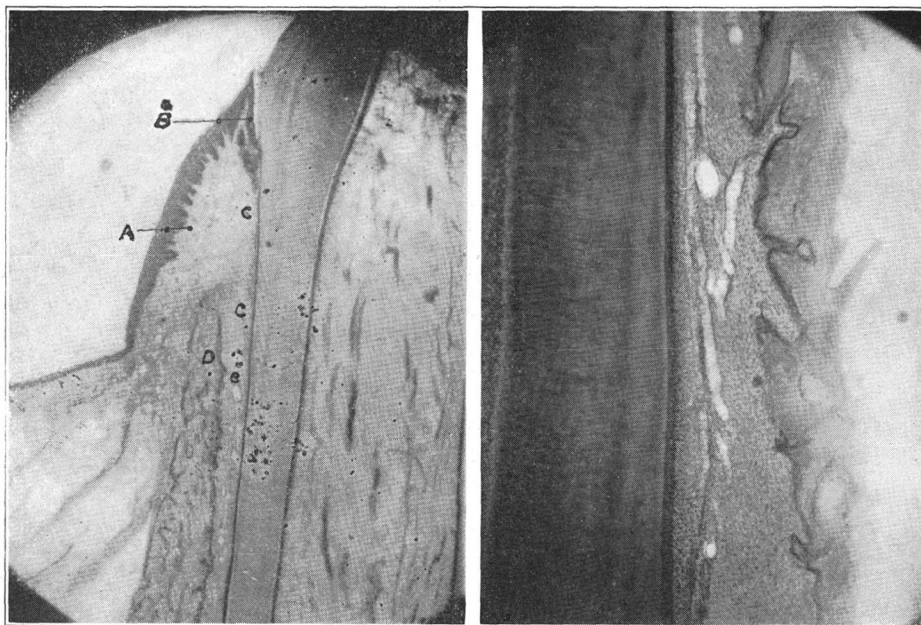
The view that these vessels are controlling influences in the spread of inflammation about the teeth, as in other parts of the body, is further substantiated. In diseases occurring at the apex, tissue destruction does not take place

gingivally to the infected area. The vessels, as they leave the tooth apex, pass down and out thru the bone of the jaw—the lymph flow is apically. Wider destruction of tissue arising from infection in the pulp may be accounted for otherwise, as was indicated in the opening paragraph.

The primary inflammatory infiltra-

amount and takes on an inflammatory character. Inflammatory reaction is evidenced in the blood vessels by an accumulation of round cells and a thickening of their walls.

The subsequent changes that take place appear to be the result of the inflammation following the course of the vessels. The principal fibers, encroached



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Figs. 1, 2.—Fig. 1, the investing tissues: *a*, gum, *b*, gingivae, *c*, peridental membrane, *d*, alveolar process, *e*, vessels; Fig. 2 shows the general direction of vessels in a longitudinal section of peridental membrane.

tion, as pointed out by Hatton (6), is located about the vessels, always in advance of tissue destruction. The first changes occur in the interfiber or interstitial tissue (14), that is, in the slight amount of connective tissue that normally fills the spaces between the principal fibers which pass from the cementum to the bone and gingivae, and that also surrounds the blood vessels and nerves. This tissue is increased in

upon more and more by the thickening of the interstitial tissue, are pushed aside and gradually degenerate. The inflammation following the vessels into the alveolar process causes its removal. Here two processes may be involved, depending upon the location of the inflammation, a pathological one and a physiological one. It must be remembered that the function of the alveolar process is one affording attachment to

the fibers of the peridental membrane. This is removed when these fibers are cut off by the inflammatory process, and it is natural to expect its removal by physiological absorption in the way it occurs following the extraction of a tooth. When bone involvement occurs at the apex of a tooth, the bone absorption would probably be purely patho-

cementum is left denuded and exposed to the action of the infective products.

#### CEMENTUM AND DENTIN

To what extent the cementum or the dentin or both tissues are involved by suppurative processes occurring in the pulp or the investing tissues is of course a problem the answer to which depends upon several factors, i.e., their permea-



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Figs. 3, 4.—Fig. 3 shows vessels passing thru bone at apex (*a*) of tooth; Fig. 4 shows tubule termination in gingival cementum. Decalcified section.

logical, as this is not a part of the alveolar process. Eventually, union of infected areas takes place, resulting in a large area of inflammatory issue. Fibers cut off from their circulation and deprived of nutrition by occlusion of the vessel walls die, and suppuration follows with the formation of a pocket or abscess cavity. Into the pocket so formed, the epithelium encroaches, keeping pace apparently with the further deepening of the pocket until it appears to completely line it (6). The

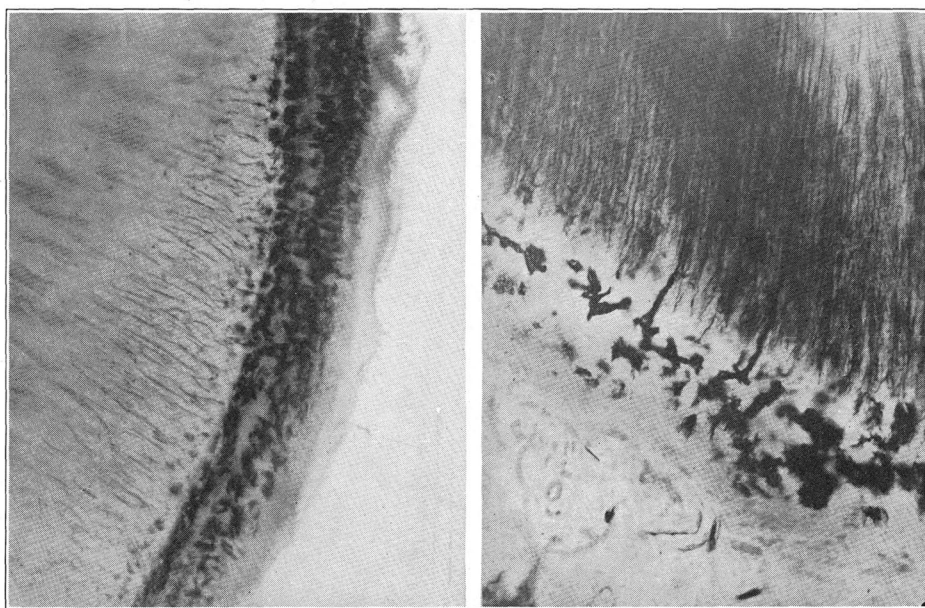
bility, the relation in which they stand to one another in the matter of structural continuity, and the length of time they are exposed to penetrating substances.

Recent work on these tissues which discloses more clearly and emphatically the structure of the granular layer and its relation to dentin and cementum, indicated a closer continuity between cementum and dentin than has heretofore been realized (21, 22).

In the gingival region the cementum

is thin, averaging about sixteen microns or less—a fraction of a millimeter (8, 10, 13). It is usually free of lacunae, which do not appear until the middle or the apical portion of the root is reached. They are quite frequently found in the cementum in the bifurcation of multi-rooted teeth. Innumerable imbedded fibers of the peridental membrane are,

It is composed, apparently, of spaces with which the dentinal tubules anastomose, or, partly, of the tufted endings of the tubules themselves. In the apical region it becomes complicated by the appearance in it of canal and lakelike spaces, anastomosing, on the one hand, with the lacunae and canaliculi of the cementum and, on the other, with the



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Figs. 5, 6.—Fig. 5, gingival cementum. Tubules and imbedded fibers intermingled. Decalcified section; Fig. 6 shows canal-like anastomosing terminations of tubules. Decalcified section.

however, noticeable. These form a series of one or more bands in which the fibers are frequently so closely packed that very little of the intervening cementum matrix may be seen. Frequently these bands are in close contact with the granular layer, which lies internal to them, or with the dentinal tubules, which penetrate to them. The granular layer is sometimes quite extensive, but more often thin in the gingival region, widening out as the apex is approached.

tubules of the dentin. This is seen so often that it is safe to consider it the rule rather than the exception.

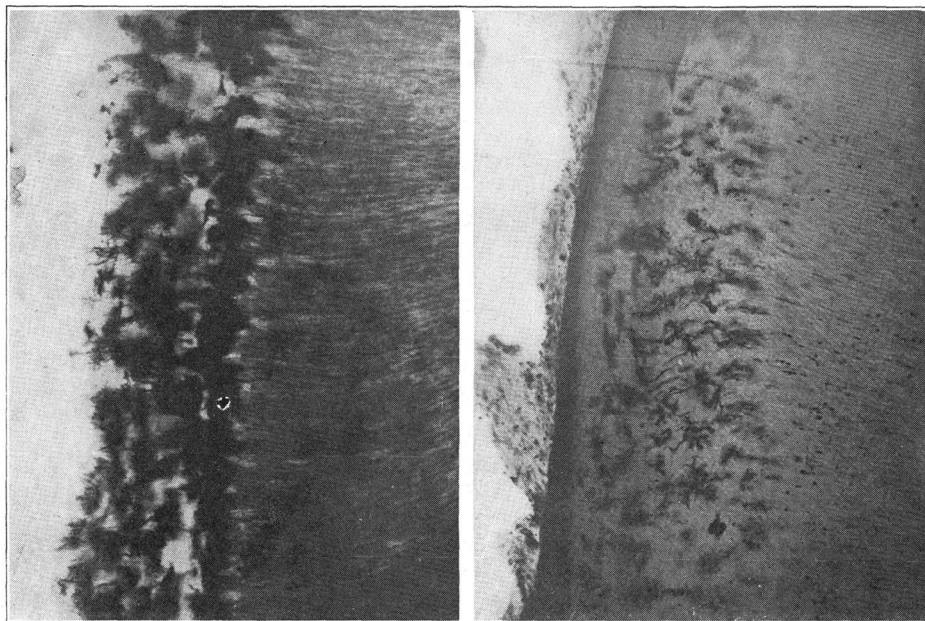
The manner in which the staining of the tissues has occurred would indicate their permeability to surrounding fluids, slowly to be sure, as the process of staining is a long one (22). Isotonic fluids would probably penetrate faster. Permeability to the stain would indicate an ability on the part of nutritional or other fluids from the surrounding tis-

sues to penetrate these tissues to the same extent.

A study of these tissues so stained arouses interesting speculation. Doubt as to their penetrability by nutritional fluids alone, especially the cementum, has been indicated by at least one writer (10). It is conceded that there is a lymph flow thru bone, which to a

not what occurs in the staining of these tissues is due to the presence of lymph in the tissues cannot be stated at this time. There has appeared, during the course of this work, some slight evidence that an alteration in the normal content of the tissues, whatever that may be, affects the staining of them.

The permeability of the tissues alone,



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Figs. 7, 8.—Fig. 7, Tissue complex, near bifurcation. Decalcified section; Fig. 8 shows more regular arrangement. Granular layer and clear layer absent. Decalcified section.

certain extent is similar in composition to cementum, the anastomosis of the canaliculi permitting its penetration. In these specimens, all the lacunae and canaliculi of the bone have been stained and brought into strong relief. Furthermore, areas similar in location to those which Noyes and Dewey indicate as the position of the lymphatics, in the periodontal membrane, the pulp, and the mandibular canal have become impregnated by the staining fluid. Whether or

however, is important, for the apparent pathway which exists between dentin and cementum may not be sufficient in some instances to permit of the passage of infective organisms themselves, the ability of the lymph to penetrate would enable their toxins to pass to the tissues and either maintain a septic area or set up an inflammation.

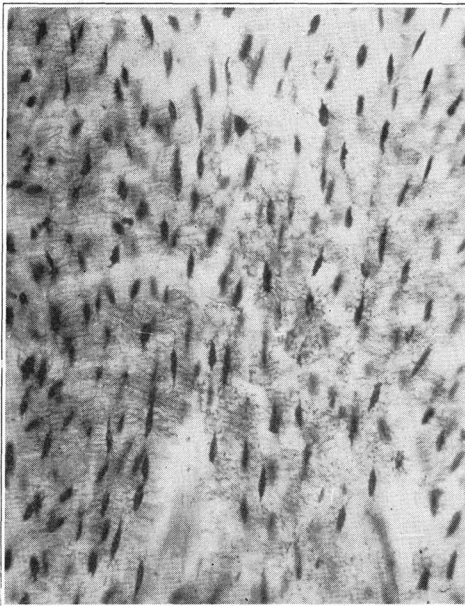
Others have apparently demonstrated the permeability of these tissues (2, 3, 8). Damlos in his work (4) obtained



cultures of streptococcus from both dentin and cementum, to a culture of which they had previously been subjected for three or four days. Marshall (12), in his experiments on rats with vital stains, altho unable to obtain a passage of the stain between the dentin and cementum, has noted a diffused staining of each, attributing this to the passage

anything about it, follow as a rule, suppurations occurring at the gingival.

A word might be said regarding multiple foramina, for, tho these have been shown by various workers in different ways and substantiated by serial sections of teeth to be present very frequently in the roots, note of them is seldom made in connection with trans-



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Figs. 9, 10.—Fig. 9 shows effect of stain on bone of the alveolar process. Decalcified section; Fig. 10 shows effects of stain on *a*, the tooth, *b*, the peridental membrane, *c*, bone of alveolar process. Decalcified section.

of fluids from the vessels of the peridental membrane and the pulp into their respective tissues.

Clinically there is evidence that would bear out Marshall in his assertion that there is no exchange of fluids between the dentin and cementum (12), barring accessory foramina. The loss of the pulp, even by suppuration, does not always cause an involvement of the tissues of attachment; neither does pulp infection, at least as far as we know

ference of infection or in treatment. These canals are characteristically more numerous in the apical region of the root but may be found quite frequently at the bifurcation of multirooted teeth or in other parts of the root. One specimen in our collection, shows a rather large canal at the middle of the root passing at right angles to the length of the tooth, straight from pulp to membrane. Large numbers of these canals contain blood vessels as well as lym-

phatics, as Noyes and Dewey have demonstrated (15).

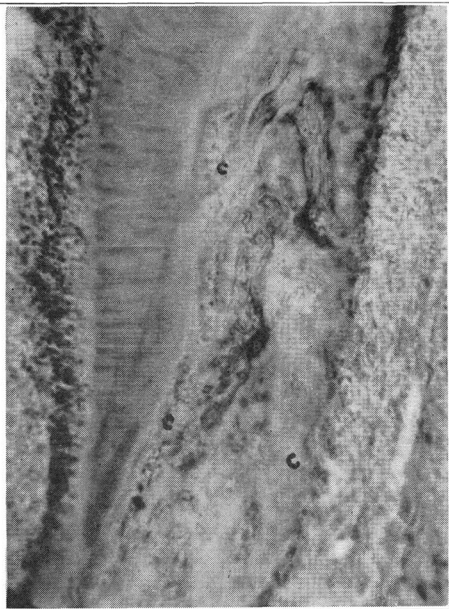
Undoubtedly these canals readily enable infection to travel thru the tissues and probably account for those wider destructions of tissue about the root than would occur if a single canal were the general characteristic (20). As heretofore pointed out, infection does

#### REMARKS

From these considerations we may draw the conclusion that beyond a doubt infective materials may penetrate the cementum from the peridental membrane, or the dentin from the pulp, but, barring accessory foramina, the question of the passage of infection from one



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Figs. 11, 12.—Fig. 11 shows effects of stain on the tissues; *a*, granular layer, *b*, vessels in peridental membrane. Gingival region. Decalcified section; Fig. 12, structure of cementum (*c*) on developing root of a dog's tooth. Decalcified section, hematoxylin and eosin.

not travel gingivally—the flow of lymph is apically.

W. Clyde Davis (5), in noting that these canals are, apparently, obliterated by deposits of cementum after pulp removal, but before inflammation sets in, may have accounted for a large number of the seemingly successful treatments of root-canals. The average method of treatment is not such as would eradicate all these focal areas.

tissue to the other, thru dentin and cementum, must still be held in abeyance.

Given perfection in the form of treatment, there is no justification at the present time either for libeling the pulpless tooth “dead” or for its wholesale extraction.

Let me emphasize that this work is the only one, as far as I know, which reveals a sufficiently recurrent and definite pathway by which, at least, infec-



tive toxins may pass from one tissue to the other. Realistic as it is, I do not believe that the evidence is sufficient. If Dr. Davis is right, in that canals may be occluded by deposits of cementum, and treatment is such that it will thoroly render aseptic and occlude the pulp chamber, canals, and tubules, extraction is not indicated in every case of pulp involvement. Especially is this so when the pulp is not affected by supuration.

It is evident that the cementum receives its nourishment from the peridental membrane; therefore pulp removal does not devitalize this tissue whose function is one of attachment. Furthermore, it may well be that nutritional fluids penetrate the dentin from the cementum and thus maintain a certain vitality in this tissue after pulp removal, expunging one more argument that the tooth becomes a foreign body on the removal of the pulp. Conversely, of course, a proof of this would indicate that infective products could pass from the dentin to the peridental membrane. Treatment, then, would have to be such as to make infective products invalid or the only recourse would be extraction. Results apparently hinge almost entirely on treatment.

On the other hand, however, when suppuration does occur within the peridental membrane and the cementum has been exposed for a time to its toxic products, there is sufficient evidence that they penetrate the cementum—the degree unknown—making it incompatible with the overlying tissue. The possibility or reattachment thus being precluded suggests the desirability of eradicating these foci of infection by the removal of the root end, as has been the *modus operandi* in these cases, and, in the others, the removal of the overlying tissue as recommended by Black (1).

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### DISCUSSION

*Russell W. Bunting, Ann Arbor, Michigan:*  
In his paper your essayist has outlined the basic histologic characteristics of the borderland which lies between the tooth and the surrounding tissues. Inasmuch as the solution of many of the problems with which we are concerned today lies within this field the presentation which has been made is very a propos and worthy of careful consideration. As we consider the subject of infection both within the tooth and immediately without we are repeatedly brought back in our analysis to the question, What is the function of the cementum, does it protect the tooth from the entrance of infectious material from without, does it protect the surrounding tissues from infectious process in the pulp?

As your essayist says, there is no doubt but that infectious organisms or their products may pass in either direction thru the accessory foramina unless they be closed by thrombosis on the pulpal side or by hypercementosis on the outside. But these foramina are for the most part confined to the apical third of the root, while in the remaining two-thirds of the root the integrity of its surface is dependent on the specific qualities of the cementum with which it is covered, barring the occasional presence of an accessory foramina. If then the cementum were perfectly pervious, like the dentin, and an open communication between it and the dentin existed we might expect that infectious processes in the pulp would quickly spread to all parts of the periodontal membrane and conversely that all periodontal infections would readily invade the dentin and pulp.

Clinically we see that this does not usually occur. In many instances highly putrescent pulps produce no apparent effect on the surrounding periodontal tissues. Their infectious organisms are hermetically sealed within the pulp canal by the closure of the foramina and the imperviousness of the cementum. So also do we see the great prevalence of gingival and periodontal infections, which if allowed to enter the tooth would result in wholesale death of pulps. Were this true the troubles of a dental practitioner would be greatly magnified over those that he now has.

Experimentally and from a laboratory standpoint numberless attempts have been made to force stains thru the dentin and cementum from the pulp canal, but with the exception of the escape of fluids at the opening of canals they have failed. In the recent experimentation with AgNo<sub>3</sub> in root-canals it has been noted that the silver salt will completely permeate the bulk of the dentin but that on the periphery and just beneath the cementum there appears to be a sclerotic area which is impenetrable, the black stain escaping to the surface only at the exit of a foramen.

In our work on the permeability of tooth structure we found that sections of dentin and cementum taken from the middle third of the root could not be penetrated by fluids under osmotic pressure, altho they could be made to pass thru the enamel and dentin quite readily. From this we infer that the cementum on the middle and gingival thirds of the root, exclusive of foramina, is normally impervious to fluids and to bacteria or their toxins and is therefore highly protective to the dental and periodontal tissues. It is not quite clear as to whether this impenetrable tissue is an inner and dense layer of the cementum or is a homogeneous layer of dentin matrix just beneath the cementum. The verity of its presence is, however, indubitable.

In the apical third of the root the conditions are quite different. Here the cementodental communication is seemingly quite patent, as shown by the recent work of Harold Box, of Toronto. It is in this region that pulpal infections tend to spread to the surrounding periodontal tissues and, as shown by the essayist, the direction of the lymph flow tends to keep them from migrating gingivally. The recent discoveries of Hartzell and Henrici who found streptococci in the pulps of healthy teeth, indicate that bacteria gain entrance to the pulp tissue more frequently than we had supposed, but the avenues by which they enter are, in all probability, confined to the foramina and other spaces in the apical third of the root, except in rare instances.

We heartily agree with the essayist when he says that the pulpless tooth is not necessarily a dead tooth. The mere removal of the pulp from a tooth in no wise disturbs the vitality or function of the cementum or periodontal membrane unless they be injured by traumatic, chemical, or bacterial agencies. Moreover the relationship of the tooth to the surrounding tissues has not been changed in any way save the possible deflection of the blood, which formerly entered the pulp to the periodontal tissues. A pulpless tooth is therefore not a foreign body for the enamel and dentin alone are dead while the cementum and peri-

dental membrane, those tissues upon which the relationship of the tooth to the surrounding tissues depends, remain alive and maintain their normal function.

We concur with the essayist in the opinion that all serious periapical infections must be treated surgically. However, we do not believe that simple excision of the root apex and curettage are sufficient. The amputated stump of the root presents hundreds of cut dentinal tubuli which are patent and unprotected. In them infectious organisms may invade beyond the reach of the body fluids and their immunizing properties, and later these organisms may be a source of secondary infection of the healing tissues. It is to this fact that many failures of root resection may be attributed. Such operation should always be followed by the treatment of the root stump with silver or some other suitable material which will hermetically seal the dentinal tubuli against bacterial invasion.

We, however, emphatically disagree with the essayist when he says that similar surgical measures are necessary for the treatment of pyorrheal lesions on the lateral surfaces of the root. Nor can we concur with the statement that the peridental membrane cannot be induced to reattach to a "pus-soaked cementum." These lateral and subgingival lesions are quite different from those in the periapical tissues. They have an opening to the exterior at the gingival crevice thru which by proper instrumentation the cemental covering of the root may be freed of excrescences, pits and depressions obliterated, and the surface made smooth and burnished. If in this procedure care be taken not to cut thru the surface of the cementum the root will still be protected by an impervious lamina which will protect the underlying dentin. This smoothed cementum is also perfectly compatible with the surrounding peridental tissues which in a large number of cases under proper treatment may be made to heal from the bottom of the lesion, reattach themselves to the cementum, and obliterate the pocket. It is not easy to present histological slides in proof of this statement for patients for whom this

has been done are loath to part with such teeth for microscopic study. But to the large number of pyorrhea workers who are daily treating this disease by rational methods and who see the lesions day by day become less and less deep under their treatment, until at last the tissues are attached about the teeth so firmly that not even the finest nerve broach may be pushed between the hard and soft tissues—to those practitioners there is no doubt of the possibility of reattachment of tissues to cementum which has been diseased. We admit that this method of treatment requires considerable technical skill and that its use is not always attended with success, but the great mass of clinical evidence which has been presented by the pyorrhea workers and the high degree of success which they have attained by sub-gingival surgery leads us firmly to believe that the cure of pyorrheal lesions is possible by the employment of measures which are far less radical than those suggested by the essayist.

We wish to congratulate the essayist on the clear, interesting, and instructive manner in which he has handled this important subject. The paper should serve to clarify the vision of many who are interested in these problems and should lead them to a better understanding of some of the more debatable questions of modern dentistry.

*Dr. Skillen (closing):* As far as reattachment is concerned, I concur with Dr. Bunting that in some cases this may take place, but I do not believe that a restoration of normal tissue will occur. If the cementum is permeable and there is a connection between it and the dentin, as the evidence seems to indicate, we do not know how far infective substances have permeated these tissues so that scaling down to healthy tissues is a matter of chance. Of course, we know nothing about that possibility as yet. I believe it would be a sorry day that would prove the dentin permeable from the cementum or vice versa, because that would mean wholesale extractions and loss of one of the most valuable organs of the body. I do believe, however, that all treatment should be such as to meet this exigency.