A histological evaluation of the human pulp in teeth with varying degrees of periodontal disease

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Careful periodontal documentation and subsequent histological examination of 46 human teeth with varying degrees of periodontal involvement showed that their pulps remained within normal limits regardless of the severity of the periodontal disease. Furthermore, it was observed that very deep caries or extensive coronal restorations were associated with pulpal changes regardless of the degree of periodontal involvement. On the basis of the teeth examined in this study, no correlations could be made between the presence or severity of periodontal disease and pulpal changes.

Both periodontal and endodontic therapy is concerned with maintaining the health of the attachment apparatus. Periodontal therapy deals with many aspects of the supporting structures, including the prevention and repair of lesions of the gingival sulcus. Endodontics deals primarily with diseases of the pulp and periapical tissues, and often, because of the presence of accessory canals, disease processes at some distance from the apex as well. Success of both periodontal and endodontic therapy on a given tooth depends on the elimination of both disease processes, whether they exist separately or as a combined lesion.

The interrelationship of periodontal and endodontic disease has been a subject of speculation for many years. Investigators have expressed an entire spectrum of opinions on this interrelationship, ranging from statements that the pulps of all periodontally involved teeth be removed to redirect the nutritional supply from the pulp to the periodontium, to statements that the histological condition of the pulp is completely independent of periodontal disease and that the amount of periodontal involvement in no way affects the health of the pulp. These hypotheses are obviously incompatible.

REVIEW OF THE LITERATURE

Much has been written concerning the histological condition of the pulp in the presence of periodontal disease. The extraordinary variety of results reported reinforces the need for careful investigations that can lead to reproducible experimental results and clinically useful conclusions.

Many studies in the literature indicate, for example, that combined periodontal and endodontic therapy is essential for successful healing of a periodontal-endodontic lesion. It has been said that either endodontic or periodontic treatment alone would not lead to a satisfactory prognosis if both disease entities were present and that both must be considered together.[1-5] Hiatt and Amen[6] claimed that persistent periodontal disease may clear up only after definitive periodontal therapy is followed by successful endodontic treatment. Most authors agree that both forms of therapy are essential for successful healing of the combined lesion. However, the problem arises as to which lesion came first and which caused or perpetuated the clinical problem.

There is general agreement that pulpal disease could initiate or perpetuate (or both) periodontal disease; the opposite theory is controversial. Seltzer and others[7] demonstrated in dogs and monkeys that, after mechanically inducing pulpal disease, interradicular lesions were

initiated and perpetuated opposite lateral canals by inflamed or necrotic pulps. In humans, Johnston and Orban[8] showed that periodontal disease that remained after unsuccessful endodontic therapy cleared up after successful endodontic therapy. Several authors[1,6,8] have shown remission of severe periodontal bone loss after endodontic therapy alone. Forrest[9] stated that disease originating in the pulp may later affect the periodontium. Simring and Goldberg[10] postulated that endodontic therapy is indicated in the treatment of terminal periodontal disease that does not respond to periodontal therapy. Stahl[11,12] questioned the relationship of pulpal pathosis and periodontal disease, noting that the material in the literature was confused on the subject and further studies were needed before definitive information would be available.

It appears reasonably clear that disease of the pulp plays a significant role in the initiation, perpetuation, and healing of periodontal conditions, and that combined therapy is essential for healing of the combined periodontal-endodontic lesion. How the pulp of a tooth is affected by the periodontal condition, however, is still controversial. There is no agreement on this subject in the literature.

Many early and recent studies suggest that a definite cause-and-effect relationship exists between periodontal disease and pulpal pathosis. Brammer,[13] in a study with neither documentation nor controls, noticed atrophic changes in the pulps of teeth with periodontal disease. Lang and McConnell,[14] in another uncontrolled study, found calcified masses in the pulps of eight of 20 teeth with pyorrhea. Bauchwitz,[15] again without the use of control teeth, concluded that teeth loosened by pyorrhea showed pulps that were "different" from the pulps of normal teeth.

Cahn[16] reported large channels (accessory canals) running from the periodontal ligament to the pulp, indicating a close relationship between the periodontium and the pulp. He concluded that he had not seen a "normal pulp" in a tooth with periodontal disease.

Craney[17] made a serious attempt to classify the amount of periodontal disease by apportioning teeth into three groups to indicate varying amounts of resorption of alveolar bone. His findings indicated a full array of pulpal changes, and normal pulps, in each of his three groups. Paradoxically, in spite of finding normal pulps in periodontally involved teeth, he then concluded that pyorrhea did have some effect on the pulp. He subsequently qualified his conclusion by admitting that this influence was neither constant nor regular.

Rubach and Mitchell,[18] in a well-documented study of periodontal disease, concluded that pulpitis or necrosis, or both, could occur as a result of periodontal inflammation. However, no normal control teeth from the same mouth were used, and no mention was made of previous history of periodontal therapy.

In 1963, Seltzer and others[19] introduced the concept of "retrograde pulpitis." They concluded that, through interference with the nutritional supply, periodontal lesions routinely produced atrophic and degenerative changes in the pulps of involved teeth. The histological observations may have been correct, but conclusions cannot be drawn solely on the basis of such observations. No statement was made regarding previous periodontal therapy, no periodontally normal teeth were examined, and, most important, no control teeth from the same mouths were examined to see if perhaps all pulps from these patients were atrophic, regardless of the extent of the periodontal disease. Such sweeping generalizations on the etiology of observed pulpal changes cannot be made without careful documentation, clinical controls, and history of previous periodontal therapy.

These are the most significant studies currently indicating a direct effect of periodontal disease on pulpal pathosis. None of them was conducted with the strict experimental control and documentation necessary to obtain the base of this problem. On the basis of the previous studies, it has been assumed that periodontal disease directly affects the pulp, an assumption found throughout the literature.

Posteraro[20] said that, in advanced periodontal disease, the pulp becomes infected by the mechanism of lymph drainage or direct extension and, without endodontic intervention, the pulp dies. This was based on his own general information and on one clinical case presentation in which he did not specify the vitality of the pulp, pulpal necrosis, or possible occlusal trauma. Forrest[9] said that disease may initiate in the pulp with later effects in the periodontium, or vice versa, again an unsupported assumption. Serene,[3] on the basis of a superficial review of the literature and on four clinical cases, concluded that destruction of the periodontium had been responsible for the contamination of many teeth. Stallard,[21] at a meeting of the American Institute of Oral Biology, attempted to summarize the existing

evidence and report current investigative findings on the interrelationship of periodontal disease and pulpal pathology. In reviewing the literature, he said that all the hypotheses concerning this relationship cannot be true because they conflict radically. Nevertheless, he concluded that an interrelationship did exist between pulpal and periodontal tissues, with diseases of either adversely affecting the other, despite his previous statements.

However, several studies demonstrate that periodontal disease as such does not have a direct or constant effect on pulpal pathosis and may be independent of it. Fish and MacLean[22] performed an experiment that invalidated previous studies that found bacteria in the pulps of teeth with pyorrhea.[23-26] By cauterizing the gingival sulcus before extraction of the periodontally involved teeth, they found a complete absence of microorganisms. They concluded that the presence of microorganisms in nearly all teeth of these previous studies[23-26] was the result of the pumping action of luxation and extraction, and was unrelated to pulpal infection. Unfortunately, in spite of their findings, the earlier bacterial studies are still quoted as evidence that periodontal disease results in infection of the pulp.

Sauerwein[27] undertook a study to determine whether, and to what degree, a relationship exists between periodontal conditions, both inflammatory and dystrophic, and degenerative changes in the pulp. After reviewing the literature, he said that most authors assumed that periodontal disease caused degenerative alterations of the pulp. He then reported on 104 teeth with periodontal disease, the pulps of which he examined histologically. He found no regressive alterations in 15% of the teeth, and said that changes in the remaining teeth were no different from alterations he observed in a normal dentition. He further stated that these observed changes are not necessarily a sequel of periodontal breakdown but could be caused by obstruction of the nutritional channels by denticles as well as by exogenous irritants.

Mazur[28] and Mazur and Massler[29] reported on a two-part study, involving 106 cariesfree teeth from 26 patients and then 22 teeth from four additional patients, correlating pulpal changes with varying amounts of peridontal disease. The second part of the study was well controlled, and the severity of the periodontal disease was documented carefully. They found no relationship between the amount of periodontally exposed root and pulpal changes. A full array of pulpal conditions was found in each group of periodontal diseases, a finding seen also by Craney,[17] but which he interpreted incorrectly. In the same patient, Mazur and Massler found no correlation between the severity of periodontal disease and pulpal changes. In fact, the pulps of the same patient showed similar appearances regardless of the amount of periodontal involvement. Teeth with normal periodontium showed the same pulpal changes found in the periodontally involved teeth.

Much of the pulpal pathology associated with many previous studies involved so-called degenerative conditions termed "reticular atrophy," "hydropic degeneration," or "fatty degeneration." Langeland,[30-32] Arey,[33] and Zander[34,35] contend that these alterations do not reflect true pathosis but rather the difficulty in obtaining good fixation of the dental pulp. In addition, the question also exists as to whether previous investigators paid sufficient attention to normal histologic variation in diagnosing so-called pathosis in the pulp.

In addition, it was not clear whether these cases of periodontal disease had or had not received previous treatment. In our opinion, extensive periodontal manipulation may at times compromise the well-being of the pulp. We have been unable to decide whether such pulpal pathosis reported in the literature was associated with the periodontal lesions per se or was caused by their treatment.

Further evidence is needed to help resolve this question. Therefore, a study was designed with the following considerations:

—Any case involving previous periodontal therapy was to be excluded.

—All aspects of the periodontal condition would be documented clinically and adequate controls would be maintained.

—The procedures would ensure histologic preparation adequate to differentiate fixation artifact from true pathosis.

In this way, it was hoped to establish the degree to which pulpal pathosis is associated with periodontal disease, as opposed to causation by other complications such as decay, operative procedures, or, most important, periodontal manipulation.

MATERIALS AND METHODS

The study involved the clinical and histological examination of 46 human teeth in patients ranging in age from 14 to 89 years. These teeth were designated for extraction by the

oral diagnosis department of the Boston University School of Graduate Dentistry. Before extraction, each tooth was carefully documented, both periodontally and endodontically. The sample included 38 intact, caries-free teeth, and 12 teeth with evidence of caries or a history of operative manipulation.

In addition to a routine medical history, the data recorded (Fig 1) included the tooth number and reason for extraction. Periodontal evaluation included measurement of pocket depth at six positions around each tooth, mobility patterns, soft tissue recession, occlusal evaluation, radiographic analysis of bone level, and verification that no previous periodontal therapy had been performed. Furcation involvements, if any, were noted, and the teeth were given scores of 0 to 8, according to Russell's Periodontal Index. Each tooth was then placed into one of three general periodontal categories: normal, gingivitis, or periodontitis.

Endodontic evaluation included notation of the condition of the clinical crown; that is, whether the tooth was intact, carious, or had a history of operative manipulation. Both electrical and thermal tests of pulp vitality were performed, and tenderness to percussion was noted. Radiographs were studied for depth of restorations, possible pulp capping, or periapical pathosis, to determine to which of these general endodontic groups the tooth belonged: normal, hyperemic, pulpitic, or necrotic. According to these classifications, 39 teeth were normal, six were necrotic, one was hyperemic, and none was pulpitic. However, of the pulps classified not within normal limits, all but one were necrotic. In the case of each necrotic pulp, the tooth had either extensive caries or massive restorations. The absence of teeth with inflamed vital pulps may be explained by the symptomfree nature of the teeth randomly selected for the study. Inflamed vital pulps do exist often enough under carious lesions or extensive restorations. Figure 2 illustrates a severe pulpal inflammation from an unrelated investigation[36] for purposes of histological comparison only. With use of local anesthesia, the teeth were extracted as atraumatically as possi-

SCORE SHEET

Name: _____
Age: _____
Sex: _____

Tooth Number: _____

Reason for Extraction: _____

Periodontal Evaluation:

Pocket Measurement: buccal mesial ____ lingual mesial ____
buccal ____ lingual ____
distal ____ distal ____

Mobility: ____(0,1,2,3) + or -

Gingival Margin from CEJ (Recession): _____

Occlusal Evaluation: _____

Radiographic Evaluation: _____

Previous Periodontal History or Therapy: _____

Russell's Periodontal Index Score: _____

Furca Involvement: _____

General Periodontal Group: _____

Endodontic Evaluation:

Crown Condition: _____

Vitality Test: _____

Thermal Tests: Cold: _____
Heat: _____

Purcussion: _____
Radiographic Findings: _____

General Endodontic Group: _____

Fig 1—In addition to routine medical history, data recorded for each tooth determined general periodontal group of normal, gingivitis, or periodontitis, and its general endodontic classification of normal, hyperemic, pulpitic, or necrotic.

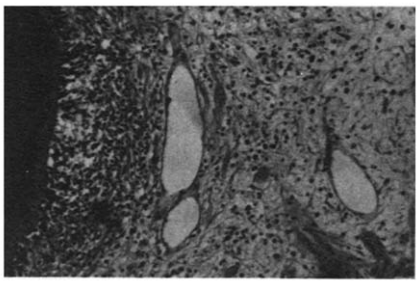

Fig 2—Section from another histopathologic study of dental pulp (courtesy of Dr. M. Piekoff) illustrates severe inflammation of pulp caused by deep caries.

ble to prevent the histological sequelae of traumatic extraction. The histological technique used in this study has been used at Boston University School of Graduate Dentistry since 1969.[36] By far the most crucial step in the histological preparation is prompt and adequate fixation of the pulp. Langeland's studies[30,32] have demonstrated that merely dropping a tooth in a jar of Formalin, even if done immediately after extraction, is inadequate to permit subsequent critical examination of the dental pulp. This has been a serious problem in many previous studies.

Our fixation procedure involved refinements of the basic methods developed by Langeland,[31,32] Zander,[34,35] and others. It represents the most predictable method known to us of achieving adequate pulpal fixation. The teeth were obtained immediately after extraction. The apical ends of the roots were cut off promptly and the sides of the teeth were ground away under an abundant flow of cool water until the pulps were nearly exposed longitudinally. The teeth were placed immediately into 10% neutral buffered Formalin solution and left there for at least 48 hours. The teeth were then decalcified in 5% nitric acid for ten days. After paraffin embedding under vacuum, serial sections were made and stained with hematoxylin and esoin. Each pulp was examined serially. The histopathologic condition was evaluated and recorded according to the protocol illustrated in the Table.

This chart was prepared in accordance with Langeland's criteria[32] for true pathosis as opposed to residual technical artifact or normal range of variation in an asymptomatic, healthy pulp. Cavity formation in the pulp sections both in the odontoblast layer and centrally served to evaluate, on a 0 to 3 basis, the degree of artifact associated with incomplete or delayed fixation of the pulp tissue. Indication of true pathosis included degenerated odontoblasts or aspirated odontoblast nuclei, inflammatory cells either in the odontoblast layer or centrally, dilation of capillaries, extravasation or breakdown of erythrocytes, and calcific changes. The final diagnosis evaluated not only the severity of any one criterion, but also the number of criteria met and the combination in which they occurred. The final histological evaluation of each tooth ranged from within normal limits, through moderate and severe inflammation, to complete necrosis, or autolysis.

RESULTS

The results of our histological evaluations are summarized graphically in Figures 3 and 4. Figure 3 illustrates the distribution of the teeth

Table • Periodontitis group.

Tooth	Cavity formation		Changes in odontoblast layer	Aspirated odontoblast nuclei	Inflammatory cells			Dilation of capillaries	Extravasation of RBS's	Brown pigment	Calcifications		General histological evaluation
	Odontoblast layer	Centrally			Odontoblast layer	Centrally					Pulp stones	Secondary or tertiary dentin	
						In vessels	Outside vessels						
F1	2	2	0	0	0	1	1	1	0	0	0	1	NL*
F2	1	2	0	0	0	0	1	1	1	0	0	0	NL
F3	0	1	0	0	0	0	1	1	0	0	1	0	NL
F4	2	2	0	0	0	0	0	1	0	0	0	1	NL
F5	1	1	0	0	0	0	0	1	1	0	1	0	NL
G1	2	1	2	2	1	1	2	2	2	1	0	2	Severe inflammation
G2	1	2	2	1	0	1	1	1	0	0	0	1	Moderate inflammation
H1	0	0	0	0	0	0	0	1	1	0	0	0	NL
H2	0	1	0	0	0	0	0	1	0	0	1	0	NL
H3	1	2	0	0	0	0	0	1	1	0	1	0	NL
H4	1	2	0	0	0	0	0	1	0	0	3	0	NL

*NL indicates within normal limits.

according to their periodontal condition. Four teeth were clinically normal, eight showed evidence of gingivitis, and 34 showed periodontitis. None of the teeth in the normal periodontal group showed histological evidence of pulpal pathosis. In the gingivitis group, only one tooth contained a pulp that was outside normal limits histologically. In the largest category, the periodontitis group, six teeth showed evidence of pathologic pulpal changes.

Figure 4 further analyzes the data by segregating and comparing the intact caries-free teeth in each periodontal group from the teeth in that group that were carious or had histories of operative manipulation. In the periodontally normal group, all the teeth were intact, and all the pulps appeared within normal limits. In the gingivitis group, six teeth were intact, and all the pulps appeared within normal limits. Of the two teeth in this group that were not intact, one had a pulp within normal limits, and the other had a pulp with moderate inflammatory changes. This last tooth contained a deep mesial occlusal and distal amalgam and also a large buccal amalgam. All 20 intact teeth in the periodontitis group contained pulps that were histologically within normal limits. Fourteen teeth in this group were not intact; of these 14 teeth, eight contained pulps that were histologically within normal limits, and the remaining six showed pathologic changes. Seven of the eight teeth in the group with normal pulps contained shallow restorations or minimal decay; only one tooth had a large restoration. On the other hand, the remaining six teeth in this periodontitis group had extensive restorations or decay; only these six teeth

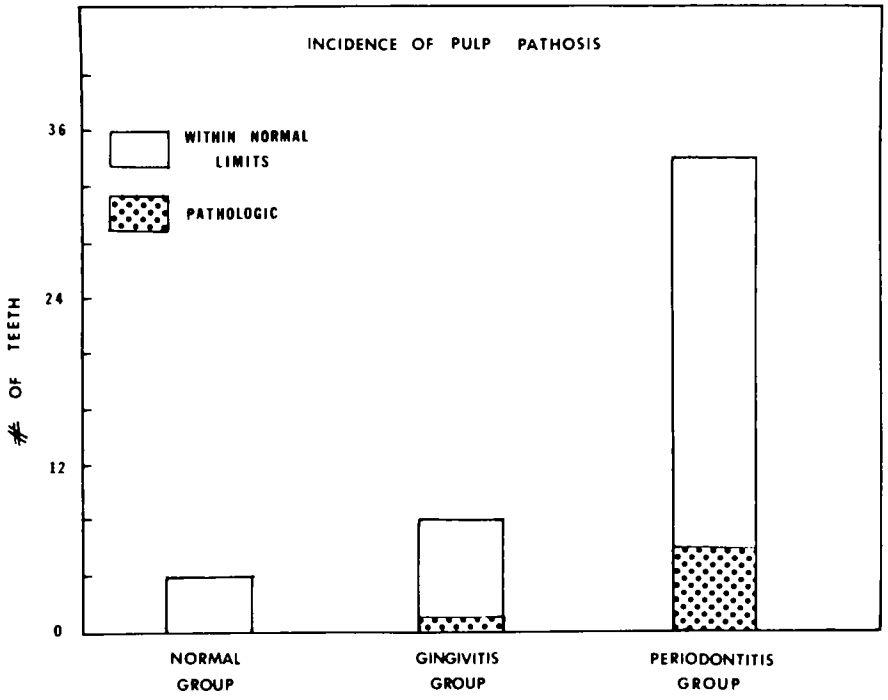

Fig 3—Graph of results. Of four teeth in normal group, all appeared histologically within normal limits. Only one tooth in gingivitis group contained pulp that was not within normal limits. Of 34 teeth in periodontitis group, six teeth contained pulps that were not within normal limits.

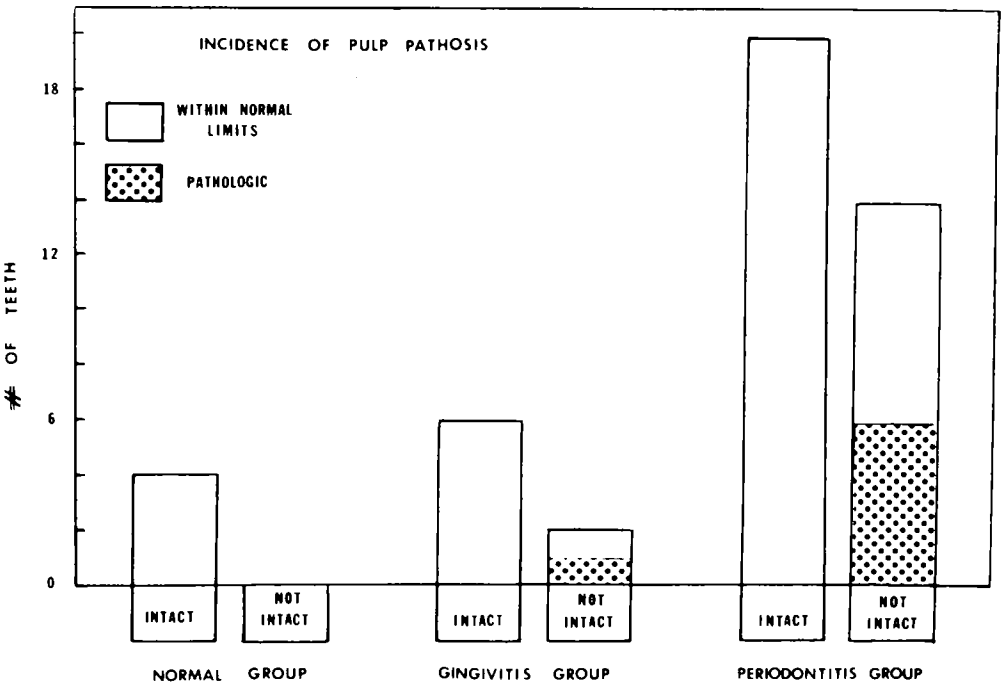

Fig 4—Graph illustrates same data as in Figure 3 but segregates intact teeth in each group.

had pulps that were not histologically within normal limits.

Figures 5 through 18 illustrate representative pulp sections and radiographs of teeth with varying degrees of periodontal involvement and, where significant, compare the radiographic appearance of the attachment apparatus with the histological appearance of the pulp. The legends for each illustration and the following discussion give a detailed description of the periodontal condition, as a combination of many factors contributed to the periodontal evaluation of each tooth.

DISCUSSION

A thorough analysis of the results of this study shows a pattern of noncorrelation between the pulpal appearance and the coincidental periodontal condition of each tooth. In the experimental sample, only teeth with extensive decay or operative manipulation showed pulpal pathosis, regardless of the absence, presence, or severity of associated periodontal disease. Our attempt to identify periodontal disease as a causative agent in pulpal pathosis has been completely unsuccessful. The pulps of all intact teeth, as well as many that were not intact, were diagnosed histologically as within normal limits regardless of the severity of any associated periodontal disease.

Figure 5 illustrates an example of a pulp from a young patient that was interpreted to be within normal limits from the periodontally normal group. Notice the relative absence of morphologic changes and total absence of inflammatory cells, compared with pulp shown in Figure 2.

Figure 6 shows a pulp from the gingivitis group. Notice the intact

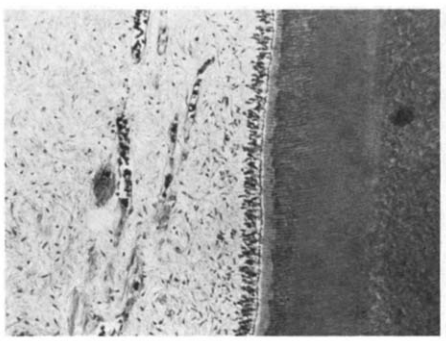

Fig 5—Pulp from intact tooth in normal group, from young person, might be considered ideally normal. Note intact zones and vessels, complete absence of inflammatory changes.

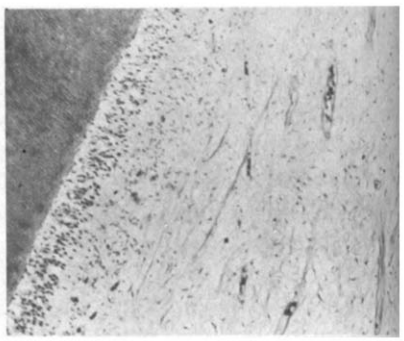

Fig 6—Pulp from gingivitis group shows normal appearance of cell zones, even with imperfect fixation. Diagnosis as within normal limits was made owing to absence of pathologic criteria.

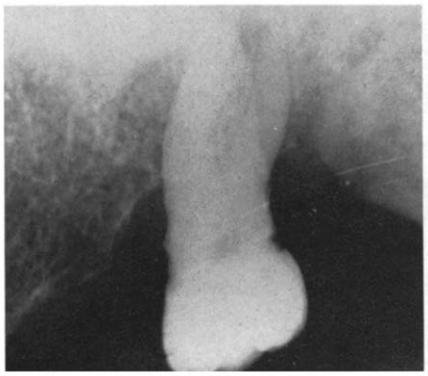

Fig 7—Radiograph of tooth from 89-year-old woman with severe periodontal involvement. Note degree of bone loss. All furcations were exposed, and distobuccal root (histology in Figure 8 and 9) could be probed nearly to apex.

Fig 8—Pulp in distobuccal root of tooth in Figure 8. It is from periodontitis group in this study and shows variation of normal pulp classification because of age. Degree of fibrosis and diffuse calcification is not surprising as pulp came from 89-year-old woman. Same fibrosis and calcification were seen in nine other teeth with vital pulps extracted from patient at same time. Though tooth was associated with severe periodontal disease with bone loss involving almost complete length of root, pulp was diagnosed as within normal limits.

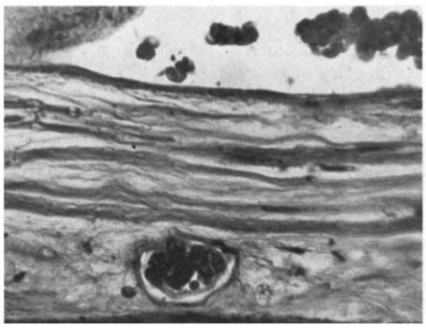

Fig 9—High-power magnification of pulp of distobuccal root of tooth shown in Figures 7 and 8. Note erythrocytes in intact functioning blood vessels (indicative of vital pulp). Even with extensive calcifications and fibrous stroma, pulp was classified as within normal limits because no criteria of pathology, such as inflammatory changes, could be seen.

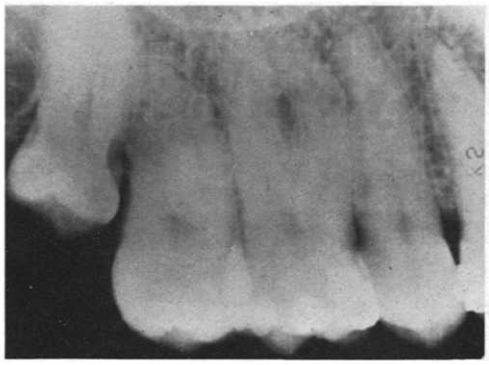

Fig 10—Maxillary second molar showing moderate mobility, trifurcation involvement, deep proximal infrabony pockets, and two small occlusal restorations.

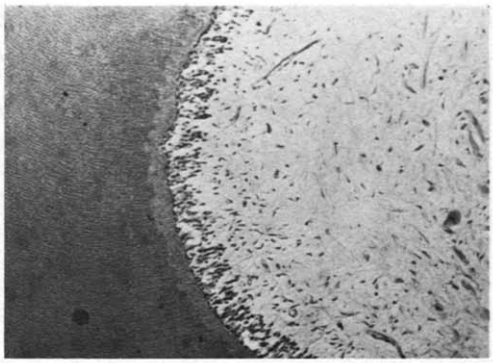

Fig 11—Pulp of second molar in Figure 10 could not be distinguished from our completely healthy controls. Intact cell zones, functioning blood vessels, and fine connective tissue stroma with no inflammatory or degenerative changes can be seen.

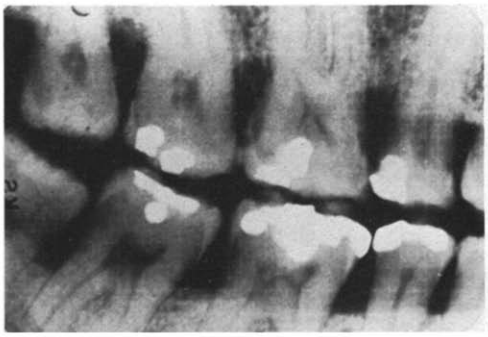

Fig 12—Maxillary second molar shows much horizontal bone loss, slight mobility, and two small occlusal restorations.

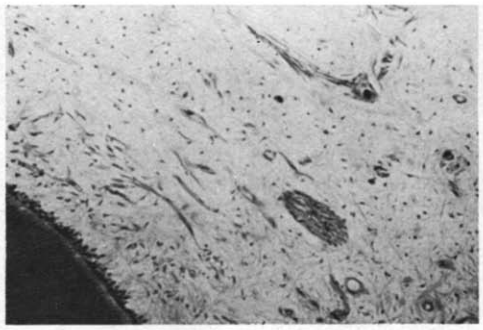

Fig 13—Pulp of second molar in Figure 13 could not be distinguished from healthy controls. Intact cell zones, functioning blood vessels, and fine connective tissue stroma with no inflammatory or degenerative changes can be observed.

cell zones, intact vessels, and the absence of inflammatory changes. The observed cavity formation is an artifact of imperfect fixation. Hence, this pulp was diagnosed as being within normal limits.

Figures 7, 8, and 9 represent a radiograph and histology of a pulp from the periodontitis group, showing a variation of our normal pulp classification because of the patient's age. The degree of fibrosis and calcification seen is not surprising because the pulp is from an 89-year-old woman. The same picture was seen in nine other teeth with vital pulps extracted from this patient at the same time. Thus, even though this tooth was associated with severe periodontal disease with bone loss involving almost the entire length of the root, the pulp was diagnosed as within normal limits.

Figure 7 is a radiograph showing severe breakdown of the attachment apparatus. The distobuccal root could be probed to the level of the apex, and all furcations were exposed periodontally. Figures 8 and 9 show the pulp of the tooth in Figure 7 in both low- and high-power magnification. As explained previously, pulps of this type are considered within normal limits, in spite of the fibrous stroma and generalized calcifications, both because of the

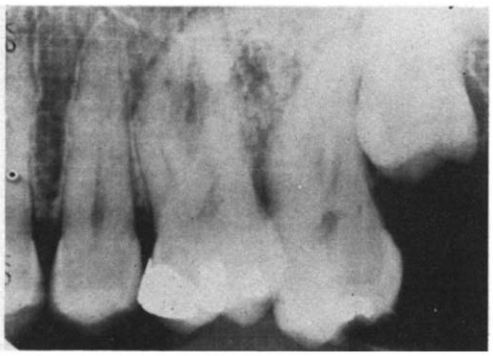

Fig 14—In same patient as shown in Figures 12 and 13, maxillary second molar had severe horizontal and vertical bone loss, especially distally, involving more than half length of root. All furcations were exposed and tooth showed moderate mobility.

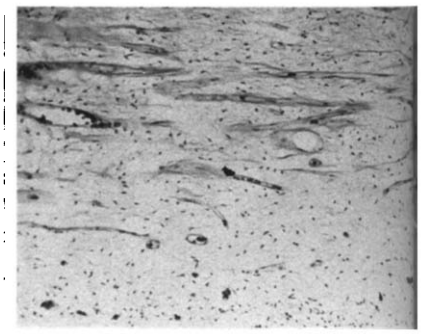

Fig 15—Pulp stroma of second molar in Figure 15 appeared well organized and free of inflammatory changes in spite of severe periodontal involvement of tooth. Normal appearance of pulp was not an isolated finding in this patient, as several other teeth with periodontal involvement were also studied and showed identical appearances.

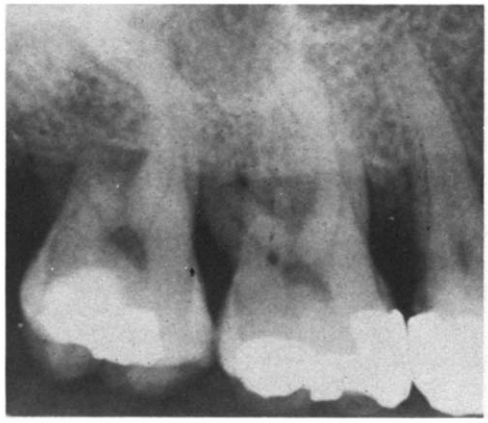

Fig 16—Figures 16 to 18 show that pulp can remain viable and within normal limits, not only in presence of severe periodontal disease, but also with restorations of such depth as to produce significant secondary dentin formation. Radiograph of maxillary first molar shows severe horizontal bone loss, severe trifurcation involvement, and deep coronal restoration. Distobuccal root could be probed almost to apex.

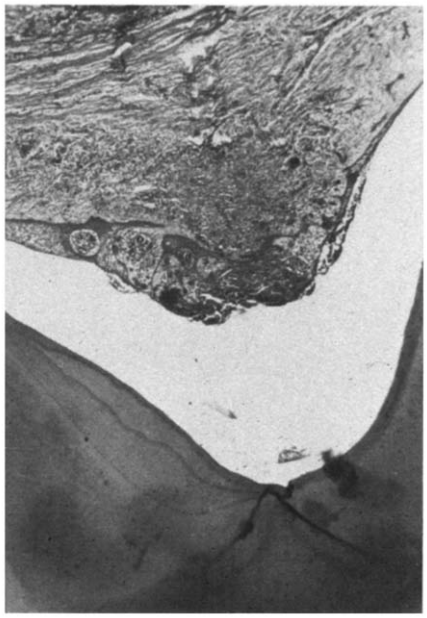

Fig 17—Histological appearance of periodontal lesion in trifurcation of first molar shown in Figure 16. Note inflamed granulation tissue that has completely replaced alveolar bone in region of furcation.

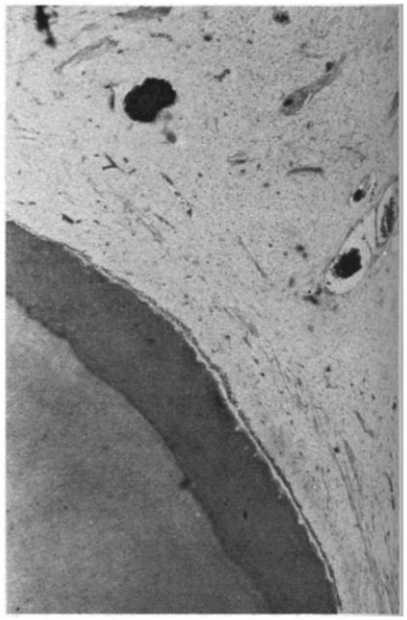

Fig 18—Even in presence of large trifurcation lesion, pulp remained within normal limits. Note buildup of secondary dentin on root of pulp chamber because of extensive operative manipulation. Under this calcification, pulp shows normal structure with intact cell zones, functioning blood vessels, normal connective tissue stroma, and absence of inflammatory changes, and is indistinguishable from any pulp in normal control group.

advanced age of the patient and because of the total absence of vascular or inflammatory changes.

The teeth discussed so far from all three periodontal groups were all intact. The extremes of normal limits in both the very young and the very old patient should be noticed. The next group of illustrations are from middle-aged patients in whom both periodontal disease and coronal restorations were present.

Figures 10 and 11 show a radiograph and pulp section of a maxillary second molar with much horizontal bone loss, deep infrabony pockets on the mesial and distal aspects, involvement of all three furcations, two small occlusal restorations, and moderate mobility. The

pulp of this tooth (Fig 11) could not be distinguished from completely healthy controls. Perfectly intact cell zones, intact vessels, and a fine connective tissue stroma with no inflammatory or degenerative changes were observed.

Illustrated in Figures 12 and 13 is a maxillary second molar from another patient with severe periodontitis and horizontal bone loss. Its pulp stroma appeared well organized and free of inflammatory changes in spite of the severe periodontal involvement of the tooth. The normal appearance of this pulp was not an isolated finding in this patient; several other teeth with distinct periodontal involvement yielded identical appearances. For example, Figures 14 and 15 illustrate another maxillary second molar from the same patient with an even more severe periodontal involvement. There were deeper infrabony pockets, which could be probed almost to the level of the apex on the distal aspect, together with moderate mobility. Again, the pulp appears within normal limits with intact cell layers, vessels, and no inflammatory or degenerative changes.

An apparently dramatic, but actually routine finding in our study is depicted in Figures 16, 17, and 18. In spite of extensive coronal restoration, severe horizontal and vertical bone loss, and trifurcation involvement, the pulp was still viable and was within normal limits histologically (Fig 18). Extensive secondary dentin formation is visible, indicative of the extensive operative manipulation to which this tooth has been subjected.

Figure 17 shows the histological appearance of a periodontal lesion in the furcation of another maxillary molar from the same patient. Notice the inflamed granulation tissue which has completely replaced alveolar bone in this region. Even in the presence of this huge trifurcation lesion, the pulp of the tooth (Fig 18) remained within normal limits. The intact cell layers and vessels and normal-appearing connective tissue stroma make the pulp indistinguishable from any pulps in our periodontally normal group. Calcifications, though not seen in our normal controls, have been described by Langeland,[30,32] among others, even in young healthy teeth.

Thus, the data from this study support the position that the condition of the pulp is independent of the presence or severity, or both, of periodontal disease. There was no evidence in the serial sections of the pulps of these 46 teeth that the presence of periodontal disease in and of itself has any effect on the condition of a normal, vital pulp. Likewise, in none of these 46 teeth was there evidence of even one inflamed vital pulp; although seven necrotic pulps were observed, all were in teeth with extensive restorations or decay.

The assumption in the literature that advancing periodontal disease adversely affects the pulp has been a serious problem for many years. This assumption was reinforced by the difficulty in obtaining controlled and documentable studies to correlate accurately the clinical periodontal condition and the histological condition of the pulp. This does not imply that some investigators did not seriously attempt to do so, but it remains evident that success in this correlation is difficult to achieve.

Starting with Sauerwein[27] in 1961, investigators uncovered histological evidence that questioned the assumption of the automatic adverse effect of periodontal disease on the pulp. Schilder,[1,37] has shown abundant clinical evidence, but limited histological evidence, of pulp vitality in the presence of massive periodontal disease. Mazur and Massler[29] documented further evidence of histologically normal pulps in teeth with severe periodontal breakdown.

An example of how authors have considered that periodontal disease may not affect pulp vitality, but concluded that it did, can be found in Stallard's review.[21] In summarizing much of the evidence up to that time on the interrelationship of periodontal disease and pulpal pathosis, Stallard indicated that much conflicting evidence exists. He correctly pointed to the fact that two opposing and apparently irreconcilable lines of evidence have been developed but concluded that the older assumption was the valid one.

Neither debate nor reviews will resolve the issue; it can be resolved only on the evidence of reproducible controlled investigations. It is strongly urged that a more controlled effort to reproduce the work of Sauerwein, Schilder, and Mazur and Massler be undertaken by other investigators and other institutions to verify the validity of our findings.

One point remains to be clarified. The evidence presented here supports the position that periodontal disease in itself does not affect the pulp. However, severing the apical vessels of major accessory canals during either periodontal therapy or by actual denudation resulting from gingival recession creates an entirely different clinical picture and doubtless a different histological picture as well. The teeth in this study were selected carefully to exclude any that had undergone periodontal therapy. Furthermore, this study does not address itself to regressive pulpal

changes associated with disrupted supply of blood to the pulp. One of the authors' has reported on this type of endodontic-periodontal interrelationship in bifurcation areas and elsewhere along the root. Although we recognize the great importance of this type of interrelationship, it is not the issue under study in this report.

SUMMARY

Forty-six human teeth that had not undergone previous periodontal therapy were examined and were apportioned on the basis of their clinical periodontal condition into three groups according to Russell's Periodontal Index: normal, gingivitis, and periodontitis.

After extraction, the pulps of the teeth were prepared for histological study and each was examined in serial section. Pulp conditions were recorded as normal, hyperemia, pulpitis, and necrosis.

Four teeth were categorized periodontally normal; all of their pulps were normal. Eight teeth were in the gingivitis group. Seven of these teeth had normal pulps; one was minimally inflamed. Thirty-four teeth were in the periodontitis group. Twenty-eight teeth had pulps within normal limits, and six had necrotic pulps. All of the necrotic pulps and the slightly inflamed pulp were found in teeth with caries or large restorations, or both. All teeth with intact clinical crowns in all three periodontal groups had pulps that were within normal limits.

CONCLUSIONS

No correlation could be found between the severity of periodontal disease in itself and the presence or absence of pulpal pathosis. Normal pulps were seen in teeth with advanced periodontal disease.

Pulpal pathosis is often associated with deep decay or extensive restorations. This relationship, when it occurs, is independent of the absence, presence, or severity of periodontal disease.

No conclusions can be drawn from this study as to the effect of periodontal therapy on the condition of the pulp, because the teeth examined had no history of periodontal treatment; this variable was intentionally eliminated from the study.

Many previous studies inadequately considered the multiple variables associated with decay, size and depth of coronal restorations, age, and histological artifacts in examining the relationship of periodontal and pulpal disease.

A parallel study of teeth that have undergone periodontal therapy and root planing should be undertaken to clarify further the concept of the inherent resistance of the pulp to regressive changes in the presence of periodontal disease.

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