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THE INTERRELATIONSHIP OF PULP AND PERIODONTAL DISEASE

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I N A previous study¹ attempts were made to correlate clinical signs, tests, and symptoms in teeth to be extracted with histologic observations after extraction. Among these teeth were some that were periodontally involved. Some of the latter had been the source of previous episodes of pain and other subjective symptoms. Some showed evidence of dental caries, restorations, or other operative manipulation, together with the characteristic inflammatory and reparative responses to such stimuli. A comparison of the findings in teeth with and without periodontal involvement gave the impression that the pulps of periodontally involved teeth had been more drastically affected, showing severe inflammatory and degenerative changes. The present study was carried out in order to make a more accurate assessment of the status of pulps from teeth with periodontal lesions.

PROCEDURE

The general procedure was as described in the previous report.¹ All of the teeth with evidence of periodontal disease, such as deep pockets, internadicular bone resorptions, lateral root resorptions, and mobility, were re-evaluated. The presence of periodontal involvement was confirmed, in most instances, by histologic examination of the periodontal membranes attached to the teeth following extraction. The presence of chronic inflammatory exudate, together with down-

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ward epithelial proliferations, crestal bone resorptions (when bone was also removed), and x-ray evidence, confirmed the diagnosis of periodontal disease.

Altogether, eighty-five periodontally involved teeth were examined. Of these, fifty-three had carious lesions, restorations, or both, and the remaining



Fig. 1.—Composite photomicrograph of mesiodistal section of lower right first molar. Sections are shown of a lateral canal (LC) which extends from the bifurcation region (Bi) through the dentine (D) to pulp (P). (Magnification, approximately $\times 92$; reduced $\frac{1}{4}$.)

thirty-two teeth were free of cavities or signs of previous operative interference. These two groups of teeth were evaluated separately in order to isolate the effects of periodontal lesions from the effects of caries or operative manipulation.

FINDINGS

Lateral and Accessory Canals.—Lateral canals (canals which are perpendicular to the main canal) were found in profusion in the roots of posterior teeth and occasionally in anterior teeth (Figs. 1, 6, and 9). Accessory canals



Fig. 2.—Composite photomicrograph of buccolingual section of upper right second premolar. There are multiple apical ramifications (AR) of the pulp (P) in the apical portion of the root canal (RO). There is also a lateral canal (LC) in the upper portion of the apical third of the root. D, Dentine. (Magnification, approximately $\times 92$; reduced $\frac{1}{4}$.)



Fig. 3.—Composite photomicrograph of mesiodistal section of lower left first molar. A lateral canal (LC) is present in the root close to the bifurcation region (Bi). D, Dentine; P, pulp. (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)

and foramina in the apical third of the roots were also frequently seen (Fig. 2). In molars there was a multitude of accessory canals, especially within the cementum "web" fusing the molar roots (Fig. 6). In some instances these canals were present not only in the apical third of the root but also toward the coronal portion of the tooth. Lateral canals in the bifurcation or trifurcation regions of molars were profusely evident (Fig. 3). In some instances they could be seen at different levels, coursing from the interradicular region of the tooth into the coronal portion of the pulp. In other instances canals in the bifurcation region were seen to traverse the root and enter the root canal (Fig. 9). The canals were filled with capillaries, pulp cells, ground substance, and fibers, and this tissue was confluent with the pulp tissue. In many teeth, however, the width of the accessory foramina or lateral canals was exceedingly small, permitting the presence of only small-caliber vessels and their supporting stroma. At some levels the canals appeared to be obliterated, and at other levels some remnants of pulp

TABLE I. PERIODONTAL INVOLVEMENT

HISTOLOGIC DIAGNOSIS OF PULP	PERIODONTAL INVOLVEMENT		AGE (YEARS)			
	TOTAL	WITH PAIN;	0 то 30	31 то 40	41 то 50	51+
Intact-uninflamed	5 (6)	0 (0)	2	1	2	()
Atrophic	23(27)	7(30)	3	8	10	2
Inflamed¶	42(49)	22(52)	9	8	14	11
Totally necrotic	15(18)	9 (60)	5	2	0	8
Totals	85 (100)	38 (44)	19 (22)†	19 (22)	26 (30)	21 (25)

*Percentages are shown in parentheses.

[†]Percentage in this column based on total number of cases of periodontal involvement (eighty-[‡]Percentage in this column based on total number of cases having same histologic diagnosis [§]Percentage in this column based on total number of cases having same clinical condition as [¶]Percentage in this column based on total number of cases having same histologic diagnosis [¶]Inflamed teeth included those with transitional conditions (Tr), chronic partial pulpitis (CPP) tial liquefaction and partial coagulation necrosis.

tissue were discernible. When the nutrition of the pulp was interfered with through involvement of these foramina by periodontal disease, small regions of necrosis or infarction occurred within the pulp, causing pulp-tissue breakdown, fatty degeneration, and calcification.

Especially in the distal roots of lower molars and in the palatal roots of upper molars, many anomalies in the size and shape of the root canals were found. Frequently, in these teeth, the canals fanned out toward the apex of the tooth in a "canoe-shaped" arrangement. In some instances, chronic inflammation was found in one portion of the "canoe" and not in the remainder.

Effect of Periodontal Lesions on the Pulp (Correlation With Histologic Diagnosis).—

Atrophy: Eighty-five teeth showing evidence of periodontal disease were studied. Intact, uninflamed, and seemingly unaffected pulps were found in only five teeth (Table I). Atrophic pulps were discovered in twenty-three teeth, the largest number in any specific diagnostic category. The atrophic pulps invariably had fewer than the normal number of cells in the coronal and radicular portions. Abundant dystrophic calcifications were discovered throughout the pulp tissue, often almost completely obliterating the coronal portions of the pulp (Figs. 4 and 10) and heavily infiltrating the fibrous tissue in the roots. In addition, the root canals were excessively narrowed by the deposition of large quantities of reparative dentine along the dentinal walls (Fig. 9). This dentine was highly irregular, having little or no tubular appearance. In some places the canals appeared to be obliterated, but at other levels some remnants of pulp tissue were seen. In those instances the canals appeared to be completely calcified, as judged by x-ray examination. However, completely calcified canals were never discovered histologically.

These observations seem to indicate that periodontal lesions produce a degenerative effect on the dental pulps of the involved teeth. Further confirmation of the atrophy-inducing influence of periodontal lesions on dental pulps was obtained from an examination of the thirty-two teeth in which there was no evidence of dental caries or restorations (Table I). Among this group, twelve teeth (37 per cent) had pulps that exhibited various degrees of atrophy.

TEETH WITH CARIES, RESTORATIONS, OR BOTH		TEETH WITH NO CARIES OR RESTORATIONS		NONPERIODONTALLY INVOLVED TEETH WITH CARIES, RESTORATIONS, OR BOTH	
TOTALŞ	WITH PAIN	TOTALŞ	WITH PAIN	TOTALŞ	WITH PAIN
0 (0) 11 (21) 30 (57) 12 (23)	0 (0) 2 (18) 18 (60) 9 (75)	5 (16) 12 (37) 12 (37) 3 (9)	$\begin{array}{c} 0 & (0) \\ 3 & (25) \\ 4 & (33) \\ 0 & (0) \end{array}$	$\begin{array}{c} 15 \ (22) \\ 7 \ (10) \\ 37 \ (54) \\ 9 \ (13) \end{array}$	$\begin{array}{c} 2 \ (13) \\ 2 \ (29) \\ 17 \ (46) \\ 4 \ (44) \end{array}$
53 (100)	29 (55)	32 (100)	7 (22)	68 (100)	25 (37)

(NUMBER OF TEETH AND PERCENTAGES*)

five cases).

as shown in preceding column.

shown at bottom of same column.

and same clinical condition as shown in column immediately to the left.

with partial liquefaction or partial coagulation necrosis, and chronic total pulpitis (CTP) with par-

Even where caries, restorations, or both were present, there were proportionately twice as many periodontally involved teeth as nonperiodontally involved teeth with atrophic pulps. However, the difference was not statistically significant (P > 0.10).

Inflammation: Inflammatory lesions of varying intensities were discovered in forty-two periodontally involved teeth. Necrotic pulps were found in fifteen teeth. However, since caries or restorations might have been factors in producing inflammations and necrosis of the pulps, the effects of periodontal lesions could better be studied by considering the thirty-two teeth devoid of caries or restorations. Among this group, twelve teeth had inflammatory pulp lesions and three teeth had completely necrotic pulps.

Deep periodontal lesions were frequently found exposing the lateral canals along the sides of the roots, thereby interfering with the nutritional supply of the pulps. In the more advanced lesions, necrotic pulp tissue was discovered in the larger lateral canals which were exposed (Figs. 7 and 12).

In one instance, where a pericoronitis was present around a partially erupted third molar, external root resorption was found on the distal surface of the tooth in the region of the cementoenamel junction. In the pulp of this tooth, a small area of chronic inflammation was discovered adjacent to the resorbed area. The remainder of the pulp was atrophic. There were profuse scattered regions of dystrophic calcifications in the root canal, as well as large pulp stones within the coronal portion of the pulp.

Interrelationship of caries, operative procedures, and periodontal lesions: The effects of caries or operative procedures on the pulps of periodontally involved teeth and, conversely, the effects of periodontal lesions on the pulps of operatively treated teeth were compared.

Teeth subjected to a combination of pulp and periodontal irritants had a greater incidence of inflammatory reaction than those subjected to operative procedures alone; 79 per cent of the teeth with both periodontal lesions and caries or restorations showed some degree of inflammation or necrosis, as compared with 61 per cent of the teeth with only periodontal lesions or only caries or restorations. This difference is statistically significant (P < 0.05).



Fig. 4.—Photomicrograph of mesiodistal section of upper first premolar. The tooth had a deep periodontal pocket. The pulp (P) is atrophic. There are abundant dystrophic calcifications (DC) throughout the coronal portion. (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)

(DC) throughout the coronal portion. (Magnification, approximately $\times 92$; reduced $4_{4,1}$ Fig. 5.—Photomicrograph of mesiodistal section of lower right first molar of 17-year-old girl. An extensive granuloma, resulting from pulpal involvement, is present. The inflammation (Inf) has spread to the bifurcation region (Bi), causing crestal bone resorption (R). D. Den-tine; C, cementum. (Magnification, approximately $\times 92$; reduced $4_{4,1}$) Fig. 6.—Photomicrograph of buccolingual section of upper left second molar of 51-year-old woman. Near the trifurcation region (Tri) is a series of canals, depicted by solid line. C, Cementum; D, dentine. (Magnification, approximately $\times 92$; reduced $4_{4,1}$)

Fig. 7.—Fbotomicrograph of mesiodistal section of lower right second molar of 54-year-old woman. The tooth was extremely mobile, and there were swelling and a fistula on the lingual side. A continuous, persistent pain was present. A large lateral canal containing necrotic pulp tissue (N) was found near the bifurcation region (Bi). D, Dentine. (Magnification, approximately $\times 92$; reduced $\frac{1}{4}$.)



Fig. 8.—Composite photomicrograph of mesiodistal section of lower right first molar of 25-year-old man. The patient complained of pain and swelling for 3 days. The pain was dull, diffused, and continuous. A carious pulp exposure was present. The apical granuloma (G) below the apical foramen (AF) extended into the bifurcation region (Bi), causing alveolar crest resorption. Arrows indicate regions of resorption of the cementum (C) and dentine (D) along the sides of the roots. P, Pulp. (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)



Fig. 9.—Composite photomicrograph of mesiodistal section of lower right third molar of 41-year-old man. Pain was present and was increased by cold and mastication. Resorption (R) of the root canal is present. Solid lines indicate the connection between the bifurcation region (Bi) and the dental pulp through lateral canals (LC). (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)

The effect of periodontal lesions on the pulp presumably comes about through interference with the nutritional supply of the pulp, inducing atrophic and other degenerative changes, such as reduction in number of pulp cells, dystrophic calcifications, fibrosis, reparative dentine formation, inflammation, and resorptions. Because of the impaired nutrition, which is gradual and takes place over long periods of time, some pulp cells do not come into equilibrium with the available blood supply and therefore die. However, death of the cells is so gradual that morphologic evidence sometimes appears to be lacking. The



Fig. 10.—Composite photomicrograph of lower right canine of 66-year-old man. The tooth was periodontally involved but had no caries or restorations. The dentine (D) and cementum (C) of the apical portion of the root are resorbed (R). Granulation tissue (Inf) is present at the apical foramen. The pulp above this is necrotic. The necrotic pulp (P) in the coronal portion of the tooth is heavily inflitrated with dystrophic calcifications (DC). D, Dentine. (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)



Fig. 11.—Photomicrograph of mesiodistal section of lower right first molar of 17-year-old man. A dull, throbbing pain was present, and the pulp was exposed by caries. A granuloma (G) is present in the bifurcation region next to the comentum (C) in the right-hand view. In the view on the left connection between the pulp (P) and this granuloma is shown through numerous lateral canals (LC). Some resorption (R) is present within the root canal. (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)

pulps of periodontally involved teeth had smaller than normal cells, with greater than normal collagen deposition. Pressure atrophy may also have occurred because of the mobility of those teeth. The increased pressure from the movement of the teeth affects the blood vessels and reduces the vascular supply to the tissues, resulting in vascular atrophy.

Resorptions: Resorptions of the sides of the roots were frequently found subjacent to the granulation tissue overlying the roots (Fig. 8). Where the periodontal lesions were deep, resorptions were found also within the root canals, often opposite lateral canals (Figs. 9 and 11), and at the apical foramina (Figs. 10 and 13).

The mechanism for the production of atrophy within the pulps was thereby discovered. There is interference with the nutritional supply through the lateral canals, both within the bifurcation or trifurcation regions and along the sides of the roots. The blood vessels supplying a small area of the pulp are involved by the periodontal lesion. Loss of the blood supply to a small region of the pulp tissue leads to death of the pulp cells supplied by the affected capillaries. Inasmuch as immediate adequate collateral circulation is not available, there is insufficient nutriment and oxygen to satisfy the metabolic needs of the cells, and the cells die. In other words, a small area of infarction takes place, with resultant coagulation necrosis (Fig. 11). The death of the cells and their subsequent calcification are a natural sequence of the deprivation of nourishment.

Inflammatory lesions in the pulp could also be responses to toxic products entering through canal openings normally covered with bone and periodontal Volume 16 Number 12

membrane but now exposed to the oral fluids (Fig. 12). In severe periodontal lesions, not only were apical granulomas and root resorptions produced through extension of the granulation tissue from the pocket, but also inflammatory cells were actually detected infiltrating the apical pulp tissues, thereby causing an apical pulpitis (Fig. 13).

Inflamed or necrotic pulps, produced from periodontal lesions, were then instrumental in perpetuating the periodontal lesion by elaborating toxic products into the periodontal tissues through the same lateral canals or other means of ingress. Thus, a vicious circle was established. In terms of treatment, it would be difficult to visualize an effective cure without the concurrent elimination of both the pulpal and periodontal lesions.

Local medication is another possible cause of injury and necrosis of pulp cells. The use of drugs for desensitization of the necks of teeth, especially when root surfaces have been exposed by loss of bone and an epithelial downward proliferation, is potentially damaging. In those circumstances, irritating chemicals may enter the pulp tissue through accessory or lateral foramina, thereby causing injury to the pulp cells as well as the vessels which supply them with



Fig. 12.—Composite photomicrograph of lower right first molar of 30-year-old woman. No symptoms were present. There was a deep periodontal pocket in the bifurcation region (Bi). The downward epithelial proliferation (EP) is depicted by the arrow. A lateral canal has been exposed, and the pulp within it has become necrotic (N). C, Cementum; D, dentine. (Magnification, approximately \times 92; reduced $\frac{1}{4}$.)

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Fig. 13.—Composite photomicrograph of mesiodistal section of upper left first premolar of 42-year-old man. A deep periodontal pocket was present, but there were no caries or restorations. In the lower view, the apical portion of the root is resorbed (R) by granulation tissue (G) near the apical foramen (AF). D, Dentine. (Magnification, approximately $\times 92$; reduced $\frac{1}{4}$.) In the upper view the pulp (P) in the coronal portion of the tooth is shown; it is infiltrated with chronic inflammatory cells (Inf). (Magnification, approximately $\times 230$; reduced $\frac{1}{4}$.)

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nutrients. For example, formalin, in relatively low concentrations, exerts a lethal effect on cells. Other substances may cause destruction of cells by derangement of their osmotic equilibrium.

The microorganisms present in periodontal lesions may also be capable of producing necrosis of cells through the action of their metabolic products, destructive enzymes, or other mechanisms.

Effect of Pulpal Lesions on Periodontal Lesions.—

Microscopic findings: Granulation tissue was occasionally found attached to, and obviously emanating from, inflamed pulp tissue in lateral canals and accessory foramina. This tissue was an extension of the chronic pulp inflammation caused by caries or operative procedures. In molars, especially, abundant lateral and accessory foramina were found, particularly in the cementum "web" between the roots and in the bifurcation and trifurcation regions. Inflammation of the periodontal membrane from severely inflamed pulp lesions and necrotic pulps was readily spread through these channels.

In molars containing enamel "pearls," many openings were found through which pulps could be affected (Fig. 6).

Another mechanism through which the periodontal structures became involved was discovered in some teeth where the interradicular bone remained attached to the roots after extraction. When the apical granulomas resulting from necrotic pulps were extensive, the granulation tissue was present all along the lateral aspects of the roots, causing extensive resorptions (Fig. 8). In addition, the crest of the alveolar ridge was also resorbed. This occurred in some instances without the apparent presence of bifurcation canals, although the latter may have been present but undiscovered (Fig. 5).

Thus, extensive pulp lesions cause periodontal changes through lateral canals and accessory foramina and also through the crestal extension of the granulomatous lesions. In those instances, periodontal treatment alone could not be effective in eliminating the lesion. Only effective endodontic treatment could result in its eradication.

Correlation of Pain With Periodontal Involvement.—Pain was present in periodontally involved teeth which had no caries and/or restorations, but to a lesser degree (22 per cent) than in periodontally involved teeth with caries and/or restorations (55 per cent) (significant, P < 0.01). Atrophy or inflammation of the pulp was responsible for the greatest incidence of pain in periodontally involved teeth.

The incidence of pain in teeth with inflamed or atrophic pulps was approximately 45 per cent if caries or restorations were present but periodontal lesions were absent. The superimposition of periodontal lesions appeared to increase the incidence of pain (75 per cent in totally necrotic pulps and 60 per cent in the inflamed pulps). These data were not sufficient to establish statistical significance (P > 0.1).

Correlations With Age.—Periodontally involved teeth were found in all age categories. However, only two teeth in patients from the 1- to 20-year category were periodontally involved. The number of extracted teeth which had been periodontally involved increased with age, as would be expected; seventeen

	PATIENTS' COMPLAINTS ABOUT INCREASE OF PAIN					
HISTOLOGIC DIAGNOSIS OF PULP	TOTAL TEETH CONSIDERED	INCREASED BY HEAT	INCREASED BY COLD	INCREASED BY BOTH HEAT AND COLD		
Intact—uninflamed	0	0	0	()		
Transitional	0	()	()	0		
Atrophic	16	5	6	$\tilde{2}$		
Chronic partial pulpitis	9	õ	3	I		
Chronic total pulpitis	13	5	4	4		
Totally necrotic	0	0	0	0		
Totals	38	15	13	10		

TABLE II. THERMAL RESPONSES IN TEETII

*Includes teeth with or without caries and/or restorations.

†Although thirteen teeth in this category were studied, thermal tests were made on only seven.

teeth were obtained from patients 20 to 30 years old, nineteen teeth from persons 31 to 40 years of age, and twenty-six teeth from persons aged 41 to 50 years. There was a drop in the number of periodontally involved teeth obtained from patients older than 50 years (Table I).

These findings are not necessarily indicative of the incidence of periodontal involvement per age group in the general population.

Correlation of Thermal Responses to Periodontal Lesions .---

Reaction to thermal tests: Among all periodontally involved teeth (including those with and without caries and restorations), normal responses to applied heat and cold were obtained from four out of five teeth with intact, uninflamed pulps. Eleven of twenty-two teeth (50 per cent) with atrophic pulps reacted normally to heat and cold tests. Of the remaining eleven teeth, ten reacted abnormally to heat or cold or both, and one tooth did not respond (Table II). The number of abnormal reactions to thermal tests increased sharply in all the inflammatory states (statistically significant, P < 0.05), many reacting abnormally to both heat and cold. There appeared to be no correlation between the type of pulp inflammation and the response to a specific thermal test.

In teeth without caries or restorations, fifteen (50 per cent) responded normally to applied heat and cold (Table III). Three teeth did not respond. These teeth had either necrotic pulps or totally inflamed pulps. Twelve teeth responded abnormally to thermal tests. The greatest number of abnormal responses occurred in teeth with atrophic pulps (seven of eleven teeth). Teeth

TABLE III. THERMAL RESPONSES	IN	TEETH	WITHOUT	CARIES
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	PATIENTS' COMPLAINTS ABOUT INCREASE OF PAIN					
HISTOLOGIC DIAGNOSIS OF PULP	TOTAL TEETH CONSIDERED	INCREASED BY HEAT ONLY	INCREASED BY COLD ONLY	INCREASED BY BOTH HEAT AND COLD		
Intact—uninflamed	0	0	0	0		
Transitional	0	0	0	0		
Atrophic	10	3	4	3		
Chronic partial pulpitis	6	2	2	2		
Chronic total pulpitis	1	1	0	0		
Totally necrotic	0	0	0	0		
Totals	17	6	6	5		

*Although thirty-two teeth were studied, thermal tests were not made on two teeth.

	RESPONSE TO THERMAL TESTS							
TOTAL TEETH CONSIDERED	ABNORMAL TO HEAT	ABNORMAL TO COLD	NO RESPONSE	ABNORMAL TO BOTH HEAT AND COLD	NORMAL RESPONSE			
5	0	1	0	0	4			
10	1	0	1	2	6			
22	2	2	1	6	11			
18	1	3	0	9	5			
13†	0	1	2	$\overline{2}$	2			
15‡	0	1	6	0	2			
830	4	8	10	19	30			

WITH PERIODONTAL INVOLVEMENT*

‡Although fifteen teeth in this category were studied, thermal tests were made on only nine. §A total of eighty-three teeth were studied, but thermal tests were made on seventy-one.

with chronic partial pulpitis also responded abnormally to heat and cold (three out of four).

Patients' complaints: Among the patients complaining of pain (Table II), the largest number reporting increased pain from cold were those with atrophic pulps (six cases). In most instances patients complained of pain on application of both heat and cold. Patients whose pulps were intact and uninflamed, mildly inflamed, or necrotic (that is, those whose pulps were not atrophied or severely inflamed) did not report increased pain to thermal stimuli. Similar findings were made in those periodontally involved teeth that were free of caries or restorations (Table III).

Thus, patients' complaints relating to pain on thermal stimuli cannot be used as indicators of the pathologic state of the pulp in periodontally involved teeth.

SUMMARY

In order to ascertain whether or not a relationship exists between periodontal and pulpal lesions, eighty-five teeth with periodontal lesions were examined histologically. Prior to extraction, subjective symptoms and the results of clinical tests were recorded. In addition, a medical and dental history was elicited.

In many teeth a profusion of lateral and accessory canals and foramina

RESPONSE TO THERMAL TESTS							
TOTAL TEETH CONSIDERED	ABNORMAL TO HEAT ONLY	ABNORMAL TO COLD ONLY	NO RESPONSE	ABNORMAL TO BOTH HEAT AND COLD	NORMAL RESPONSE		
5	0	1	0	0	4		
4	0	0	0	1	$\overline{3}$		
11	1	2	0	4	4		
4	1	0	0	$\overline{2}$	1		
3	0	0	1	ō	$\overline{\overline{2}}$		
3	0	0	$\overline{2}$	Ŏ	1		
30*	2	3	3	7	15		

OR RESTORATIONS BUT WITH PERIODONTAL INVOLVEMENT

were found, especially in the bifurcation and trifurcation regions of molars. Where the roots were fused with cementum, accessory canals were frequently seen. The pulps of only five teeth (6 per cent) were found to be uninvolved. Atrophic pulps were found in twenty-three teeth (27 per cent). The pulps were inflamed in forty-two teeth (49 per cent) and totally necrotic in fifteen teeth (18 per cent). These observations appeared to indicate that periodontal lesions produced a degenerative effect on the pulps of the involved teeth.

In order to separate the effects of caries or operative procedures in the pulps of those teeth with periodontal involvements, we examined thirty-two periodontally involved teeth in which there was no evidence of caries or restorations. Among this group, twelve teeth (37 per cent) had pulps exhibiting various degrees of atrophy and twelve teeth (37 per cent) had inflammatory pulp lesions. In three teeth the pulps were completely necrotic.

Further analyses indicated that pulps subjected to a combination of pulp and periodontal irritants showed a greater incidence of inflammatory reactions than those subjected to operative procedures alone.

Pulp lesions were found to have an effect on the severity of the periodontal lesion. Inflammation of the periodontal membranes from inflamed and necrotic pulps was readily spread through lateral canals and accessory foramina, especially in molars. Also, extensive apical granulomas caused resorption of the crest of the interradicular alveolar ridge. Thus, retention of these teeth could be accomplished only through combined endodontic and periodontal therapy.

Pain in periodontally involved teeth was also investigated. Atrophy or inflammation of the pulp was responsible for the greatest incidence of pain. The pain incidence appeared to increase when caries or restorations were present.

Thermal responses in teeth with periodontal lesions increased significantly when the pulps were found to be inflamed, but there was no correlation between the type of pulp inflammation and a specific thermal test.

The patients' complaints relating to pain on thermal stimuli were not found to be reliable indicators of the state of the pulp in periodontally involved teeth.

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