

endodontics

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The transmission of pathologic changes between the pulp and the periodontal structures

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The possibility that pathologic conditions in the dental pulp or periodontal ligament cause changes in the other structure was explored. This was undertaken by creating pulpal and periodontal lesions in different areas in white rats and examining the adjacent pulp or periodontium after varying intervals of time. The data seem to substantiate the interrelationship of these two organs.

PART I. EFFECT OF PERIODONTAL LESIONS UPON A NORMAL PULP

There has been much speculation in regard to the potential or actual interrelationship of the pulp and the periodontal ligament. It has been postulated by many¹⁻⁸ that pathologic changes may be transmitted between the pulp and the periodontal structures, whereas other investigators⁹ have contended that no changes in the pulp could be ascribed to this cause.

The purpose of this study was to determine whether pathologic changes in the pulp can produce alterations in the periodontal ligament, and, furthermore, to consider the possibility of alteration in the pulp due to pathologic conditions present in the periodontal structures. Part I of this article will consider the premise that pathologic conditions in the periodontal structures can stimulate pulpal alterations, and Part II will consider the effect of pulpal pathologic conditions on the periodontal structures.

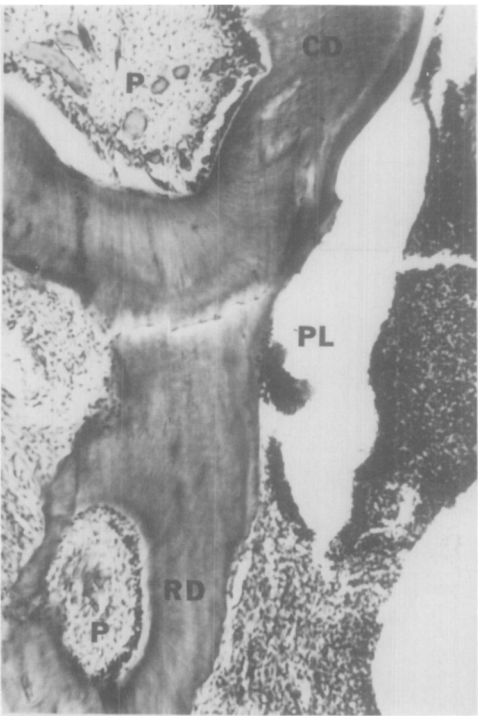


Fig. 1. Normal pulp (P) in a rat molar adjacent to a periodontal lesion (PL). Dentin of crown (CD) and dentin of root (RD).

Table I. Periodontally induced pulpal changes

Time (wk.)	Number of specimens	Response normal	Definite change	Possible change (interstitial hemorrhage)
1	6	6	0	0
1½	7	3	1	3
2	8	5	1	2
3	1	0	0	1
4	11	6	4	1
7	2	1	1	0
8	9	3	2	4
Total	44	24	9	11

Methods and materials

Seventy-five white rats were used for this experiment. General anesthesia was obtained by the use of either ether or ether and intraperitoneal Nembutal. Periodontal lesions were produced by opening the contact between the maxillary left first and second molars with a bur cut produced at the expense of the distal surface of the maxillary left first molar, with care being taken to avoid any injury to the maxillary left second molar. In this manner, the response of the pulp of the maxillary left second molar to the periodontal lesion on its mesial surface could be evaluated. No effort was made to evaluate the severity

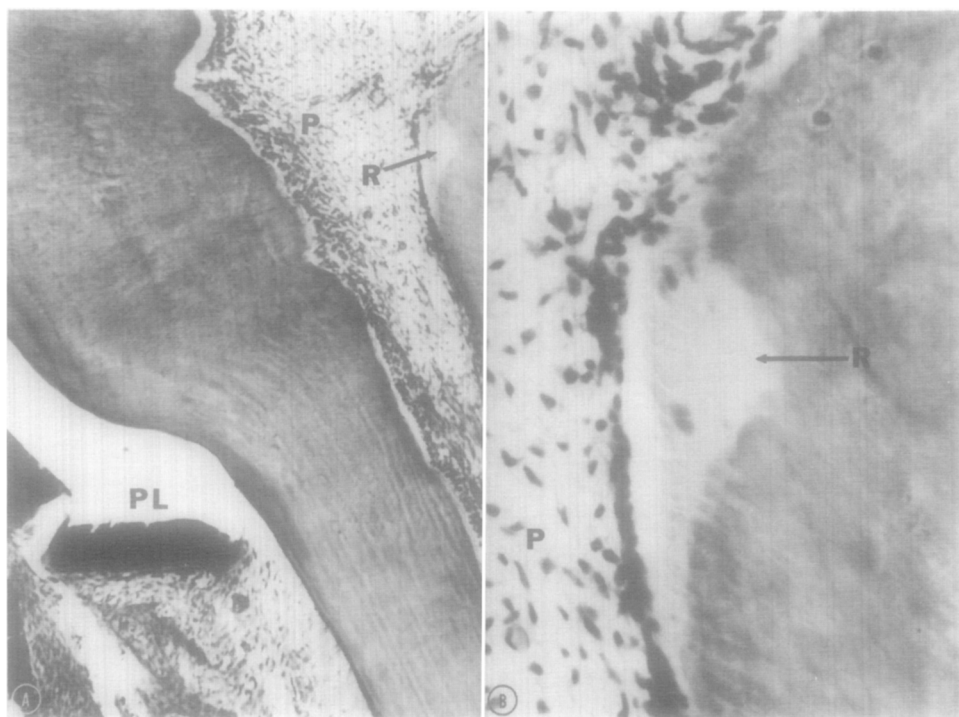


Fig. 2. A, Resorption and redeposition of dentin in the chamber. P, Pulp. R, Resorbed and repaired area. PL, Periodontal lesion. B, Enlargement of area of resorption (R) and redeposition of dentin. P, Pulp.

of the periodontal lesion, which ranged from marginal gingival damage to lesions involving most of the mesial root of the second molar.

The rats were killed at intervals of from 1 week to 8 weeks after the operation. The maxilla was immediately dissected and placed in 10 per cent formalin. The second molars were sliced mesiodistally and stained with hematoxylin and eosin.

Results

Because of technical and operative difficulties, only forty-four of the seventy-five animals were usable in this phase of the study. Of the forty-four specimens obtained for evaluation of the pulpal response to the presence of a periodontal lesion (Table I and Fig. 1), twenty-four showed no pulpal changes. Twenty specimens were of 4-week duration or less, whereas only four specimens of longer than 4-week duration were normal.

Nine teeth showed definitive pulpal changes. Of these, seven were of a duration of 4 weeks or longer, whereas one was 1½ weeks, and one was a 2-week specimen. No pulpal changes were found in the 1-week specimen. The changes found included internal resorption and redeposition of dentin (Fig. 2), rapid deposition of dentin with cellular inclusions (Fig. 3), root resorption apparently producing extensive predentin deposition (Fig. 4), and partial pulpal necrosis (Fig. 5).

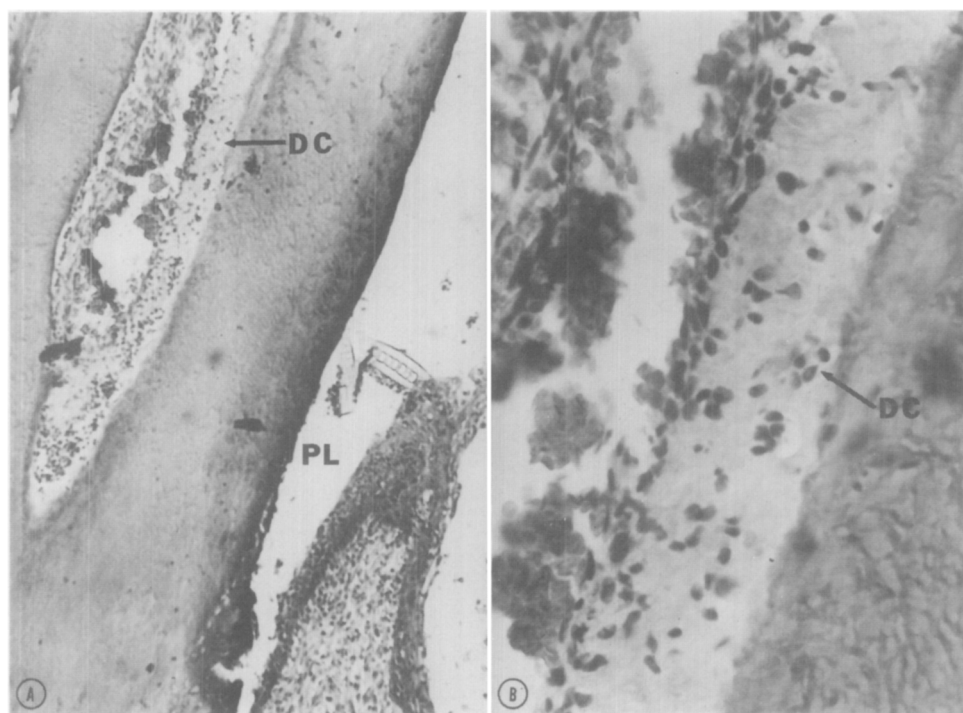


Fig. 3. A, Rapid deposition of dentin with cellular inclusions (DC) in pulp canal on side of periodontal lesion (PL). B, Enlargement of area of rapid deposition of dentin and cellular inclusion (DC).

Table II. Pulpally induced periodontal changes

Time (wk.)	Number of specimens	Response normal	Inflammatory changes	Other (ankylosis)
1	3	3		
1½	5	1	4	
2	5	1	3	1
8	3		3	
Total	16	5	10	1

In the category of possible changes the only characteristic noted was that of interstitial hemorrhaging (Fig. 6). The distribution shows that six of the eleven instances of interstitial hemorrhage occurred during the first 4 weeks, with no changes evident in the 1-week specimen. No effort was made to determine the severity of the periodontal lesion or to relate the severity of the periodontal lesion to the nature of the response. Discussion of this phase of the experiment will be undertaken at the end of Part II of this article.

PART II. EFFECT OF PULPAL INVOLVEMENT UPON THE PERIODONTAL STRUCTURES

Methods and materials

The methods and materials were given in Part I. The modification for this part of the experiment consisted of exposing the pulp of the maxillary right first

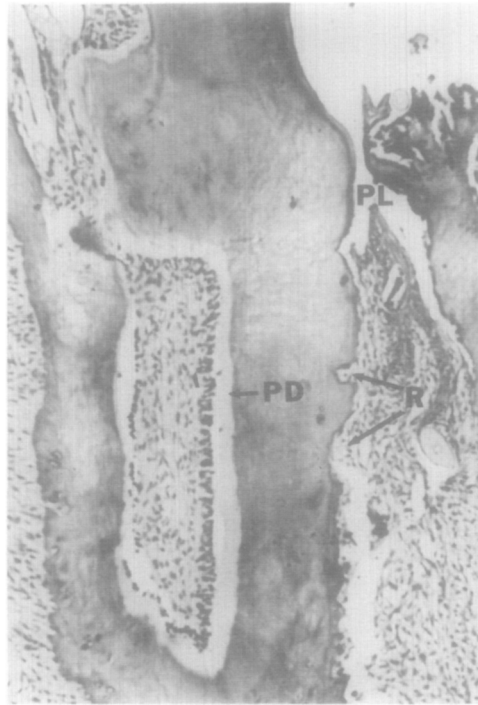


Fig. 4. External root resorption (*R*) present in an area of a periodontal lesion (*PL*), with extensive predentin deposition (*PD*) on the pulpal side of the root wall.

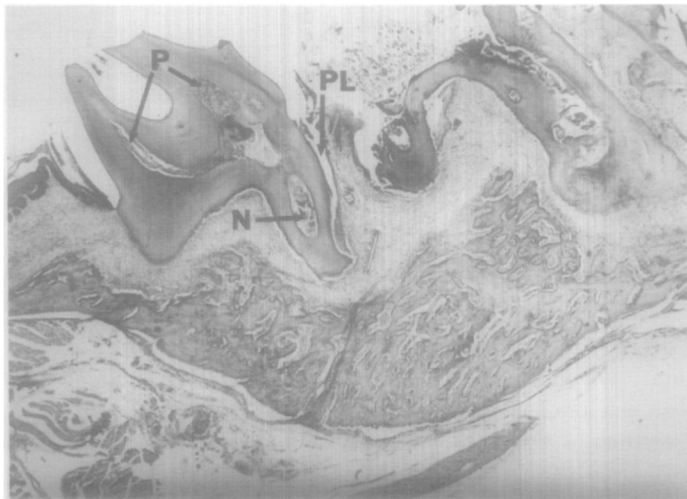


Fig. 5. *A*, Extensive periodontal damage (*PL*) along the side of the root, with necrosis (*N*) of the pulp in the canal of that root and a transition to vital pulp (*P*) in the horn of the same side and in the chamber above the other root. *B*, Enlargement of the areas of necrosis (*N*) adjacent to the periodontal lesion (*PL*). *C*, Transition from necrosis (*N*) through inflammation (*I*) to vital pulp (*P*) in the tooth on the side of the periodontal lesion. *D*, The vital pulp (*P*) horn on the side of the pulp chamber opposite from the periodontal lesion (*PL*).

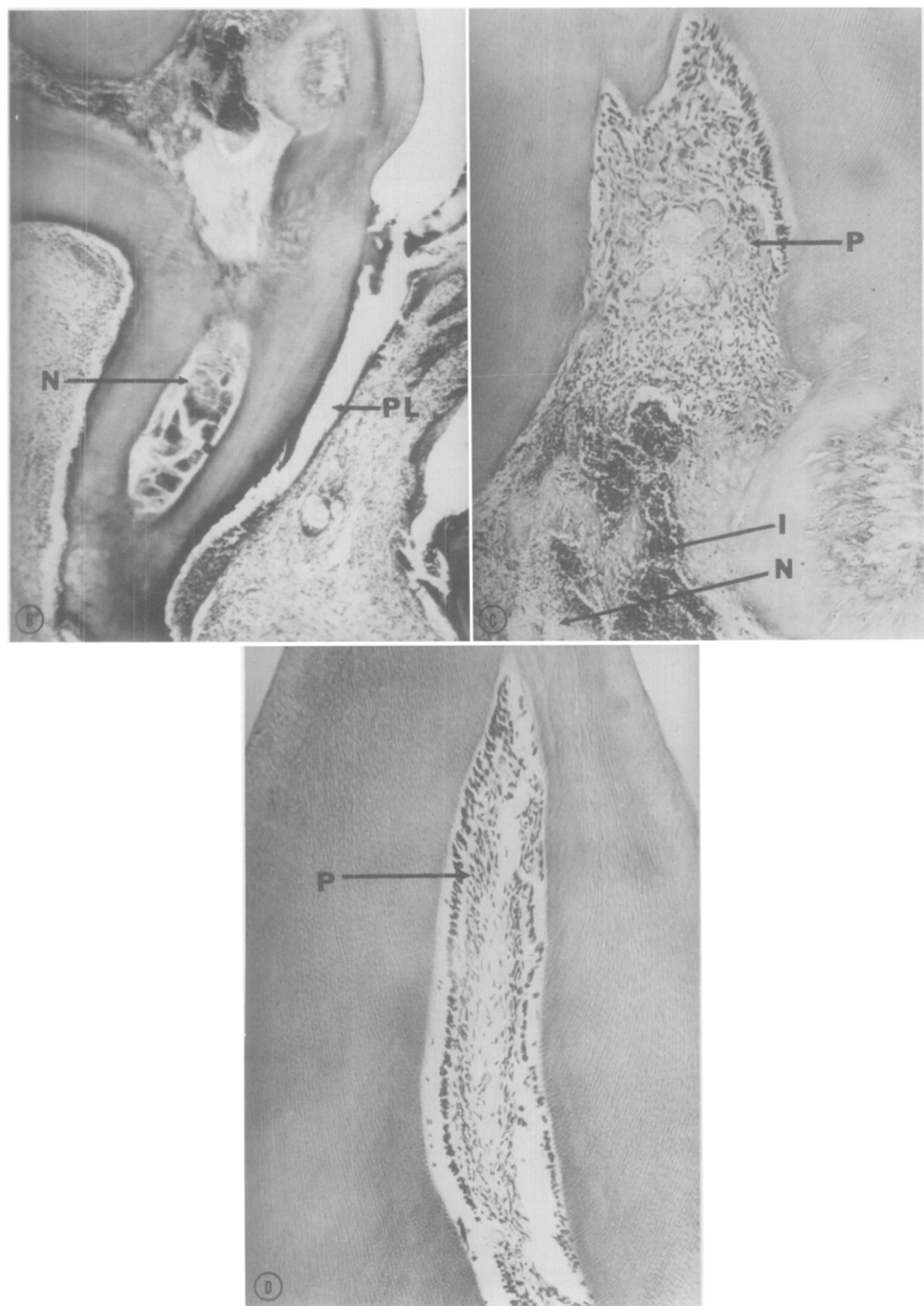


Fig. 5, B-D. For legend, see opposite page.

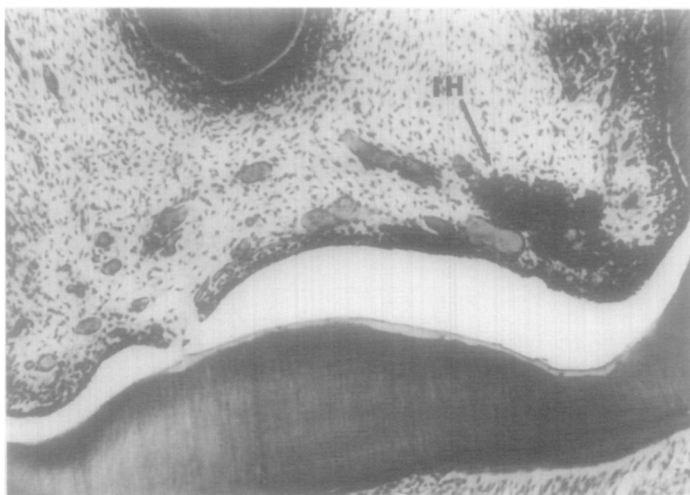


Fig. 6. Interstitial hemorrhage (IH) seen in the pulp near the floor of the chamber in a rat molar with an adjacent periodontal lesion.

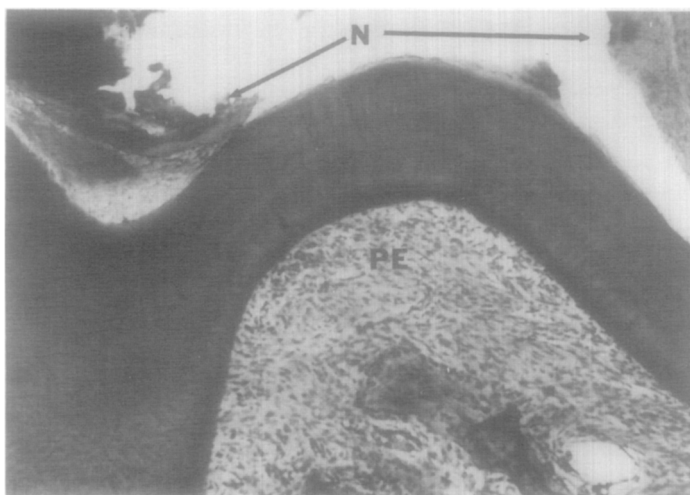


Fig. 7. Normal periodontal ligament (PE) in the furcation beneath the pulp chamber containing remnants of necrotic pulp (N).

molar through the occlusal surface with a No. $\frac{1}{2}$, round, high-speed bur. Those teeth that were perforated were discarded, which accounts for the difference in the number of specimens in each part.

Results

Sixteen specimens were obtained for evaluation of the response of the periodontal tissue to pulpal damage and pulpal necrosis (Table II). Of the sixteen specimens, five showed no change in the periodontal ligament (Fig. 7). Ten specimens showed inflammatory changes in the periodontal ligament (Fig. 8). One specimen, listed as *Other* in the table, exhibited ankylosis in the furcation

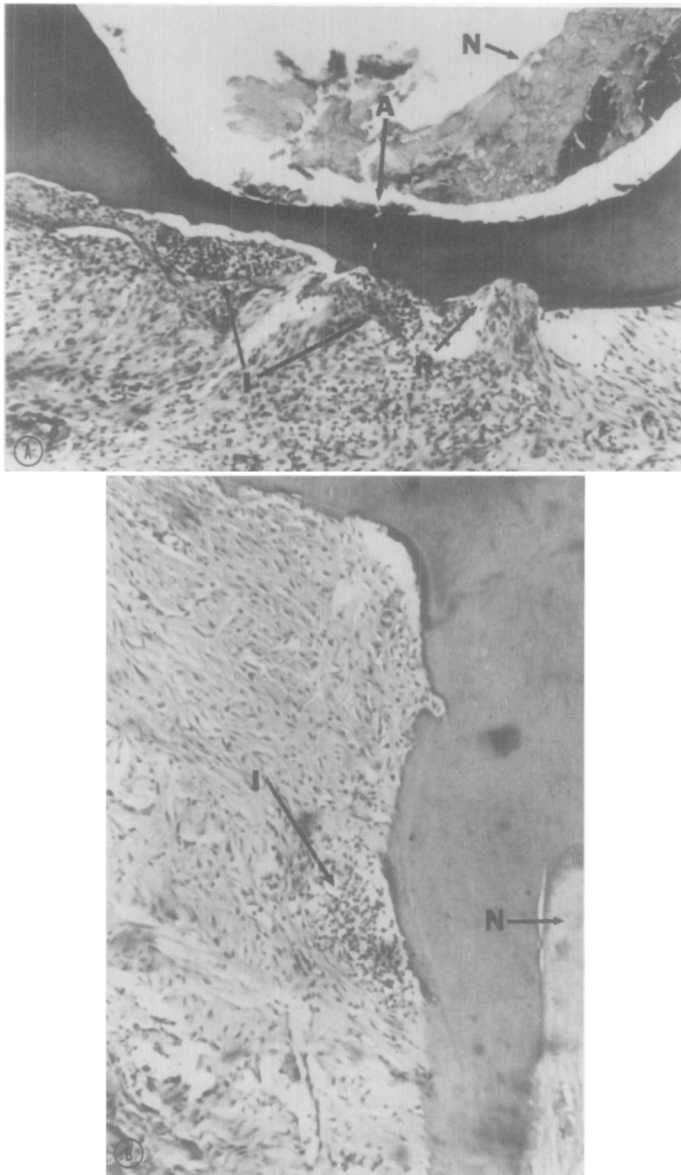


Fig. 8. A, Inflammatory (I) changes in the periodontal ligament and roof (R) of the furcation beneath the pulp chamber containing necrotic pulp (N). A, Accessory canal. B, Inflammatory (I) changes in the periodontal ligament adjacent to a root with necrotic pulp (N) in the root canal.

(Fig. 9). Of all the teeth examined in both phases of the study, this was the only tooth to exhibit ankylosis.

Discussion

Pathologic changes apparently may be induced across the barrier of the untreated tooth surface. In the phase of the study in which the pulp was exposed,

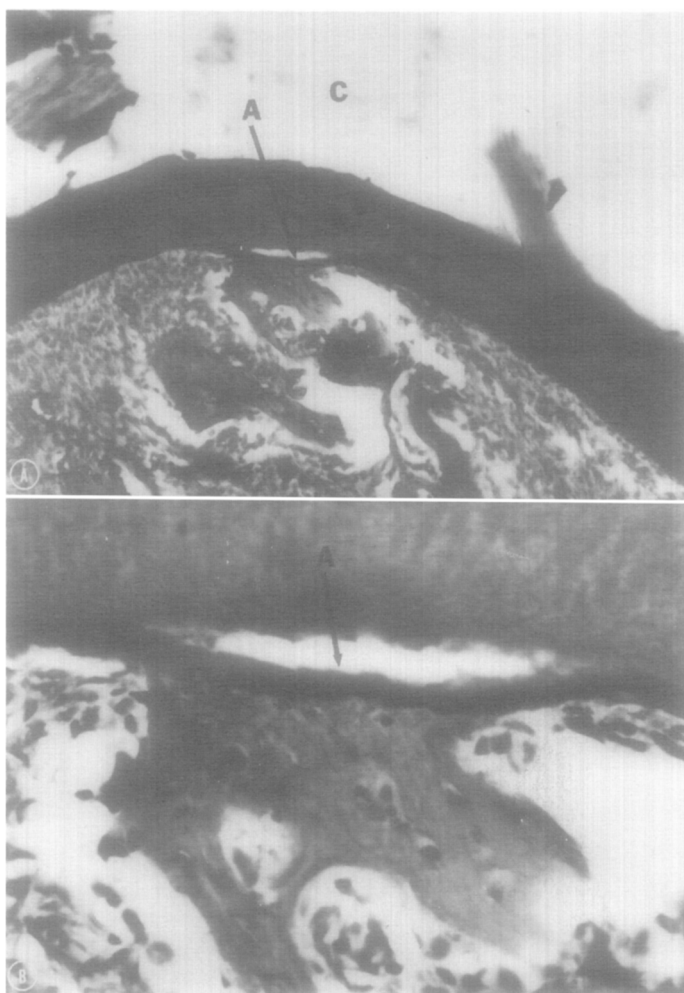


Fig. 9. A, Ankylosis (A) of a bone spicule to cementum in the roof of the furcation beneath an empty pulp chamber (C). B, Enlargement of ankylosed area (A).

eleven of sixteen specimens demonstrated definite changes in the periodontal ligament, as compared to alterations produced in the pulp of only nine of forty-four specimens when periodontal lesions were created. Further comparison of the two studies indicated that, although changes in the periodontal ligament occurred as early as 11½ weeks after pulpal exposure, changes in the pulp due to periodontal lesions occurred not only less frequently but also later in the study. Only two changes in the pulp occurred as early as 2 weeks, with the remainder occurring after 4 weeks.

The nature of the changes in each area was also of interest. The change in the periodontal structure was invariably inflammatory (Fig. 8), except for the one instance of ankylosis. However, the changes that occurred in the pulps because of the periodontal lesions were either resorptive or proliferative in

nature, except in one instance in which the pulp in one root became necrotic, with resultant adjacent inflammation (Figs. 2 to 5).

The category of *Possible change* in Table I consisted of small hemorrhagic areas (Fig. 6). Although the manner of sacrifice and fixation may have caused these hemorrhagic areas, they were noted primarily in those teeth with periodontally involved roots.

In consideration of the foregoing, it seems evident that a relationship exists between pathologic conditions in the pulp or periodontium and changes in the other structure in the maxillary molar of rats. The mechanism of the transmission of pathologic change from the one to the other can be postulated in some instances to be the presence of accessory canals in the floor of the chamber (Fig. 8, A). In other instances, the permeability of the chamber floor or root surface may be adequate to produce the changes found.^{1, 7, 8, 10, 11, 12}

The numerous instances of occurrence of inflammatory changes in the periodontal structures subsequent to pulpal injury would seem to lend credence to the concept of the transmission of irritants across the barrier of tooth structure.

In periodontally induced changes in the pulp the occurrence appeared to be less frequent and more gradual, requiring, perhaps, greater intervals of time. The findings, however, are consistent with those of other studies.^{2, 4, 7} Of considerable interest, though of indeterminate significance, is the fact that the periodontal response was *always* of an inflammatory nature; whereas, except in one instance, the pulpal response was never inflammatory, but, rather, reparative, resulting in deposition of secondary dentin, or resorptive, followed by a reparative response. In no instance was the accumulation of inflammatory cells noted in the pulp adjacent to a periodontal lesion, except in the one instance mentioned. In this one instance, the periodontal lesion was to the apex of the root and apparently caused the necrosis of the pulp in that root, with an attendant inflammatory response in the adjacent portion of the pulp (Fig. 5). The mechanism to allow periodontal lesions to induce pulpal changes would also seem to call for the permeability of the root surface to the influences of the inflammatory changes occurring in the periodontal ligament. Communication of the inflammatory process in the periodontal ligament with the apical foramen was noted in some instances and may also have contributed to the production of change in the pulp.

SUMMARY

1. Periodontal and pulpal lesions were created in the molars of rats.
2. Definite changes were noted in the periodontal structures of the rat molars that had pulpal exposure in eleven of sixteen specimens.
3. Reparative or resorptive and reparative changes were noted in the pulp in only eight of forty-four specimens that had periodontal lesions, with one instance of partial pulpal necrosis and inflammation.
4. Inflammatory changes in the periodontal ligament and pulpal necrosis apparently produce changes across the barrier of the intact tooth surface by undetermined mechanisms.

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