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Periodontal disease, bacteria, and pulpal histopathology

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Sixty teeth with various degrees of periodontal disease were extracted and studied histologically in order to determine the effect of periodontal disease on the pulp. Pathologic changes occurred in the pulp tissue when periodontal disease was present, but the pulp did not succumb as long as the main canal—the major pathway of circulation—was not involved. The cumulative effect of periodontal disease on the pulp was manifested by pulpal inflammation, calcifications, apposition of calcified tissue, and resorption. Pulpal inflammation from involved lateral canals or root caries will damage the pulp, but total disintegration apparently occurs only when all main apical foramina are involved by bacterial plaque.

The possibility that periodontal disease might be related to, or cause, pulpal disease was reported by Colyer[1] and Cahn,[2] who described structures which are currently termed *lateral canals.* Cahn stated: "It does not require a wide stretch of the imagination to see how easily an infective process might spread from without inwards, rapidly involving the pulp, especially in teeth having these side canals."

Since then, several researchers have dealt with this subject in clinical and histopathologic investigations.[3-17] The presence of lateral canals has been established beyond doubt. Furthermore, it has been established in a clinical study

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that caries of root surfaces creates a major problem in periodontal disease after the exposure of the root surface.[18] Although the literature indicates that periodontal disease may have an effect upon the pulp tissue of the involved teeth, no direct relationship between periodontal disease, root caries, and pulp response has been clearly demonstrated. In previous histologic investigations, conclusions concerning pulpal disease have been drawn upon the basis of nonvalid criteria, such as "reticular atrophy," "fatty degeneration," "fibrosis," "vacuoles," and "generalized hyperemia," despite the fact that these may be artifacts.[4, 14, 16, 19]

The purpose of this study was to correlate the presence of bacterial plaque over the entrance of either a lateral canal or a main canal with an inflammatory response in the root pulp.

MATERIALS AND METHODS

The materials consisted of sixty teeth with various degrees of periodontal involvement. All teeth were without clinically identified caries. The presence of periodontal disease was determined clinically by visual examination, use of the periodontal probe, palpation for motility, and radiographic analysis. The teeth concerned were extracted under general or local anesthesia. Immediately after extraction, the teeth either were bisected by a diamond wheel, or their selected opposing surfaces were reduced, under water spray. They were then immersed in 10 per cent neutral buffered formalin for 48 hours, or in 0.5 per cent cyanuric chloride-ethanolamine for 24 hours.

After fixation, the teeth were demineralized under gentle agitation in a mixture of 25 per cent formic acid and 10 per cent sodium citrate, or in ion exchange resin at room temperature for a period of 3 to 6 days.[18]

The specimens were histologically processed, embedded in paraffin, and serially sectioned with the use of a microtome set at 5 μm. Four to five sections were mounted on each slide, and every third slide was stained with hematoxylin and eosin. After microscopic evaluation of the first stained sections without knowledge of the clinical data, selected sections were then stained with Masson's trichrome to show the presence of collagen and epithelium, by the Brown & Brenn method to stain bacteria, and by the alcian blue–PAS method to demonstrate both bacteria and inflammatory cells.

In the histologic investigation, the roots were arbitrarily divided into coronal third, middle third, and apical third zones for the purpose of evaluating the presence of bacteria, inflammation, resorption, and apposition of newly formed hard tissue.

OBSERVATIONS

The presence of lateral canals was established in earlier published material.[16] An example of a canal with openings into the periodontal ligament and into the pulp in the same section is demonstrated. In this specimen, there was evidence of previous resorption and recent apposition of tissue in the lateral canal. The soft tissue was vital and uninflamed. Bacteria were not present at the level of the canal orifice in the periodontal space, and there were no inflammatory cells, but numerous calcifications were present in the coronal pulp and in the root canals

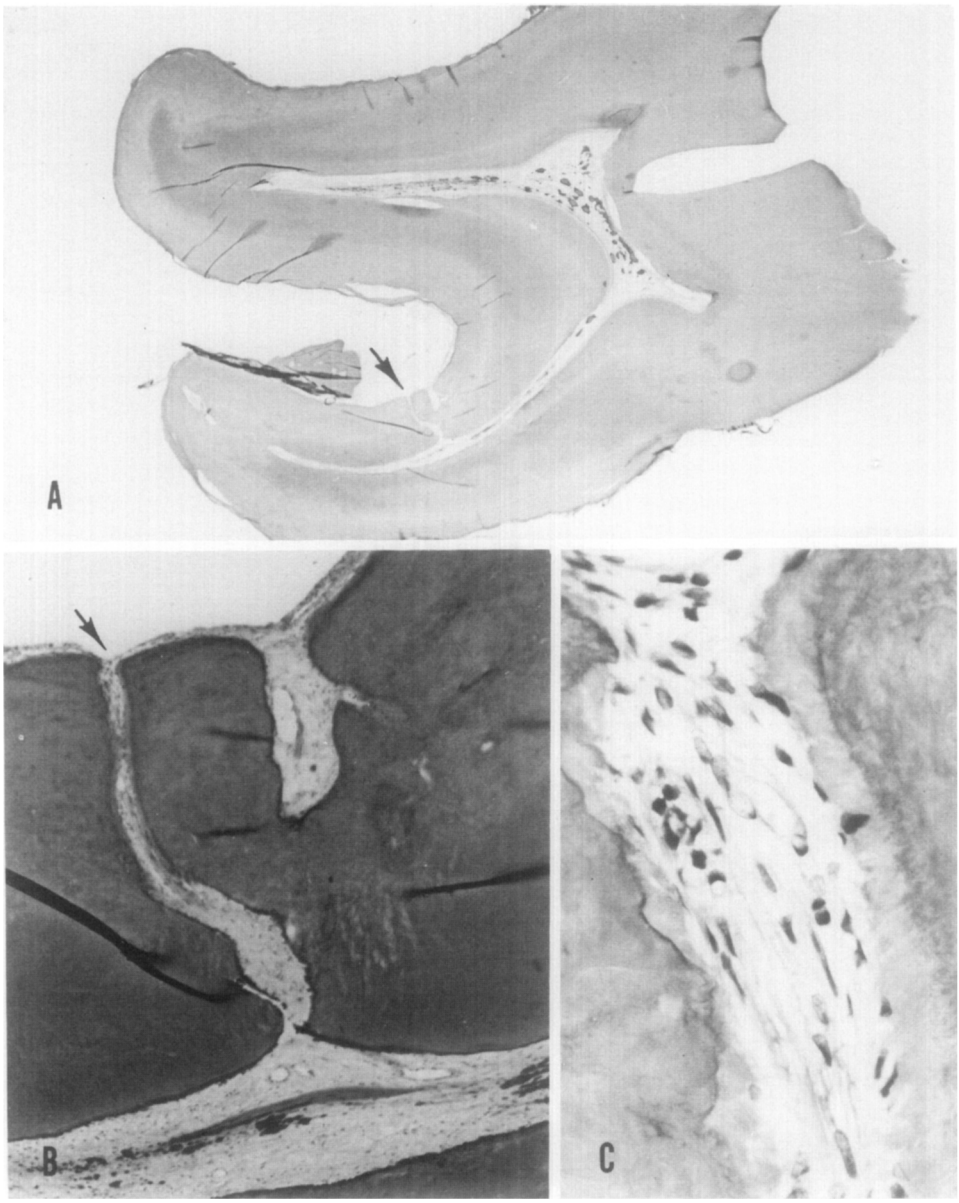

Fig. 1. Clinical data: Mandibular left third molar of 45-year-old man. Pain. Occlusal caries, no periodontal involvement. *Histologic data: A* and *B,* No bacteria present on root surfaces. Medium deep occlusal caries, slightly inflamed pulp with considerable calcifications. Interradicular canal in mesial root. *B* and *C,* Signs of earlier resorption; presently, apposition in the canal near the periodontal ligament. (Magnifications: *A,* ×8.5; *B,* ×700; *C,* ×400.)

(Fig. 1). In other cases in this study, lateral canals or apical ramifications were observed in the serial sections.

Remnants of the periodontal ligament and bacteria at various depths were observed on the root surface in all cases. In thirty-one cases, all types of inflam-

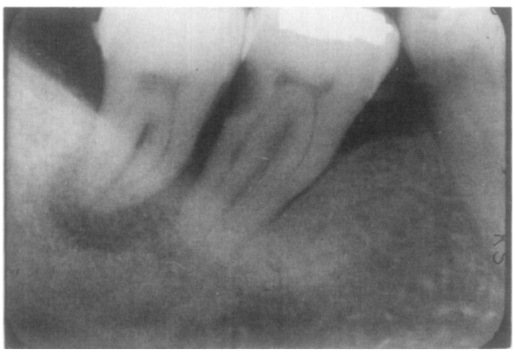

Fig. 2. Clinical data: Mandibular right third molar of 45-year-old woman. Periapical lesion. Localized pain. Caries free, calculus, periodontal disease, and bone loss involving 3/3 of roots.

matory cells were present in the adjacent periodontal ligament. In the remainder of the cases, the amount of periodontal ligament attached to the teeth after their extraction was insufficient for this evaluation to be made.

The occurrence of inflammatory cells in the dental pulp was recorded simultaneously with the occurrence of bacteria at varying depths on the root surface. The presence of inflammatory cells in the pulp occurred more frequently relative to the apical progress of the bacteria on the root surface.

In forty-seven cases, pulpal calcifications were present. On the root surface, active resorption or evidence of earlier resorption was observed in forty cases. There was an increase in frequency as the depth of bacterial penetration increased.

In seven cases, bacteria involved the apical foramen of one or more roots, and, in these cases, varying degrees of pulpal inflammation were found. Two specific examples of pulpal pathosis as a consequence of periodontal disease, in multirooted teeth, will be described in detail. In one, the effect upon the pulp of bacterial plaque involvement of a single root apex is compared with that in the other, in which all the apices are similarly involved.

The first example was a mandibular third molar in which, before extraction, a periodontal probe reached the apical area. A periapical radiolucency that involved both roots was seen on the roentgenogram (Fig. 2). Histologic sections showed bacterial plaque involving the apical foramen of the mesial root (Fig. 5, *C*) but not that of the distal root (Fig. 3, *B*).

In the apical portion of the mesial root canal there were remnants only of pulp tissue (Fig. 5, *A*) and, in this area, the Brown and Brenn staining method showed bacteria (Fig. 5, *D*). None was observed in the upper part of this canal or elsewhere in the pulp.

In sections stained with hematoxylin and eosin, no cellular distinctions could be seen in the apical part of the mesial root canal; but in the middle third of this canal numerous, distinctly stained, inflammatory cells were present (Figs. 3, *C* and 4, *F* and *D*). These cells, although now fewer in number, were also evident in the region of the pulpal orifice of the mesial canal (Figs. 3, *A* and *C*, and 4, *B, D,* and *F*). A few scattered inflammatory cells were observed in the pulpal orifice of the distal canal, and even fewer in the pulp chamber (Figs. 3, *A* and 4, *A*).

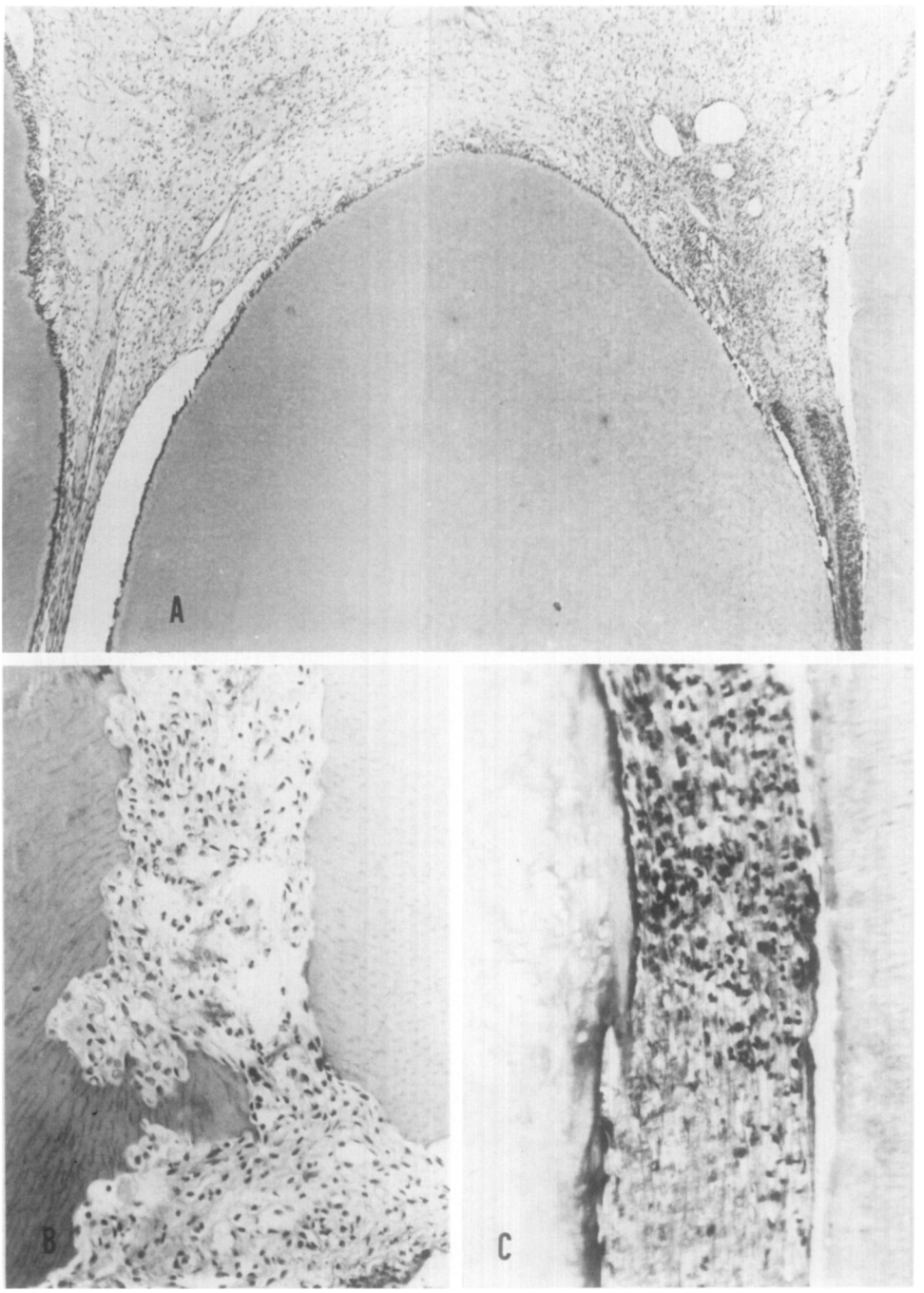

Fig. 3. Clinical data: As in Fig. 2. *Histologic data: A,* Composite picture of crown pulp and orifices to mesial canal *(right)* and distal canal *(left)*. Concentration of cells in orifice and in mesial canal. Scattered inflammatory cells in the pulp chamber; and in orifice to distal canal, few inflammatory cells, *B,* Apical area of distal canal resorption and slightly inflamed vital tissue. (Magnification, ×125.) *C,* Transition zone between area of acute inflammation *(top)* and fully disintegrating pulp tissue in apical part of root canal *(bottom)*. (Magnification, ×125.)

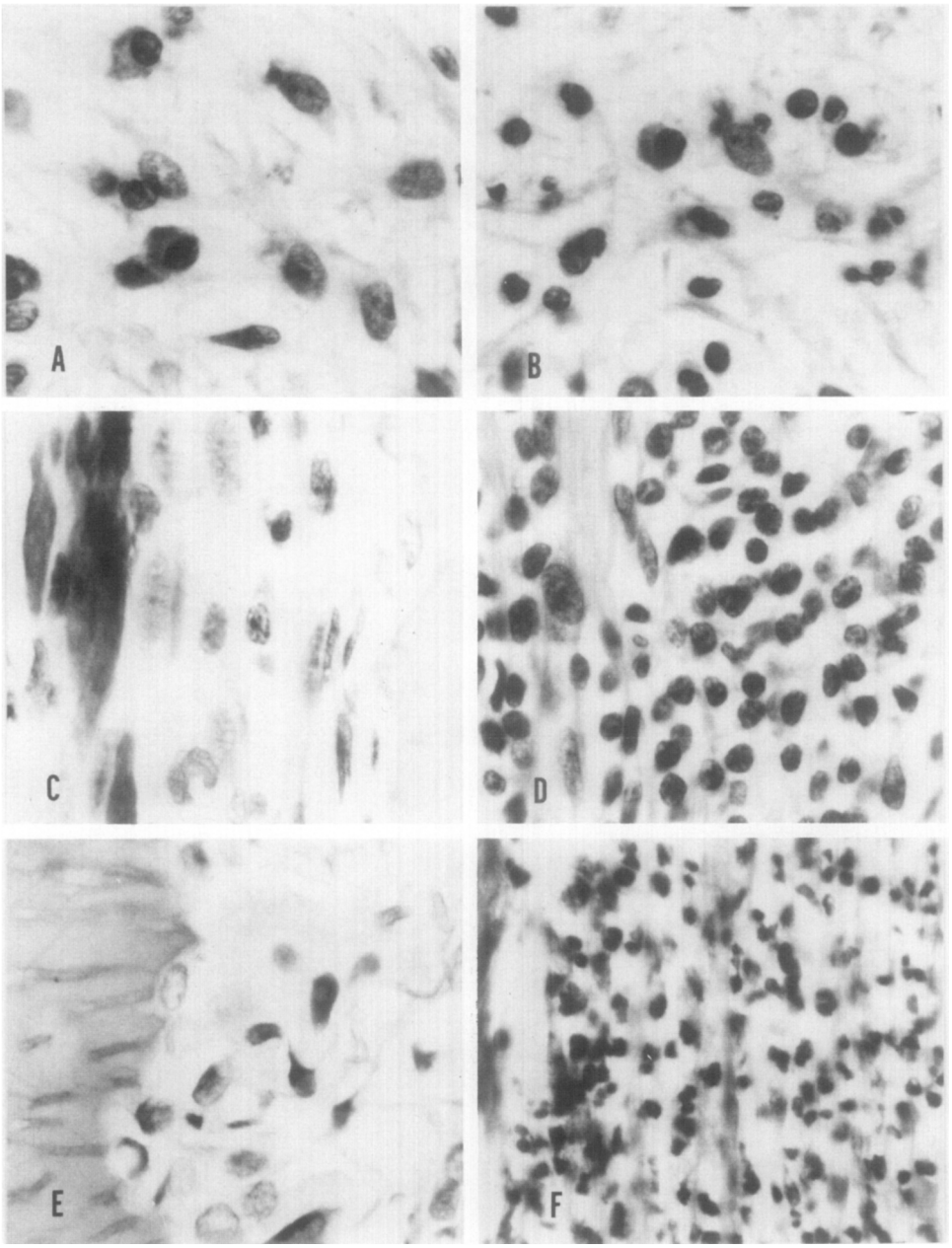

Fig. 4. Clinical data: As in Fig 2. *Histologic data: A, C, E,* Distal root. *B, D, F,* Mesial root. *A,* Few plasma cells, *mostly normal cells in pulp tissue next to orifice. C,* Calcification and normal pulp cells in root canal. *E,* Earlier resorption. Normal pulp cells in apical part of canal. *B,* Lymphocytes, plasma cells, and neutrophilic leukocytes in pulp chamber orifice. *D,* Lymphocytes and macrophages in upper part of root canal. *F,* Neutrophilic leukocytes further *apically.*

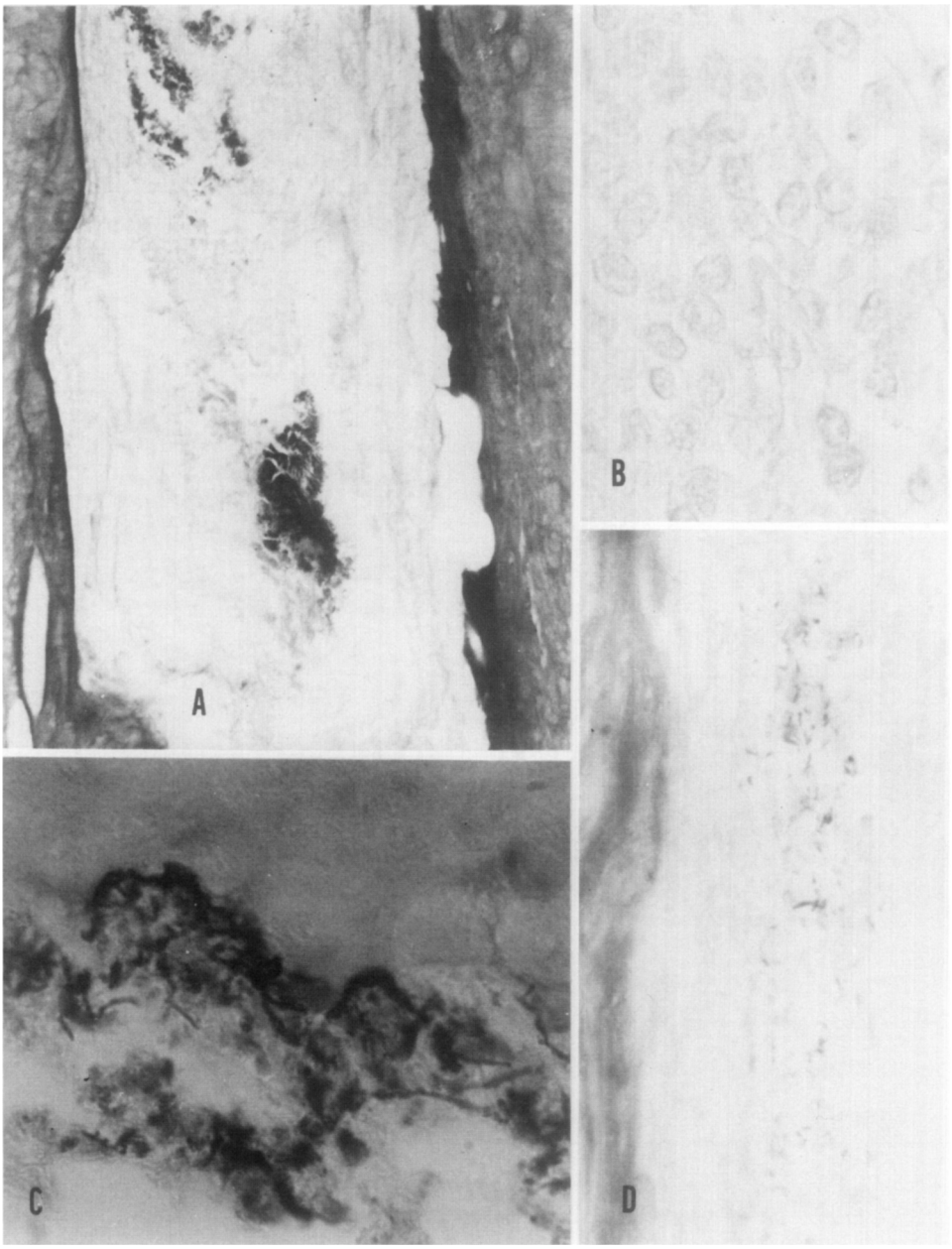

Fig. 5. Clinical data: As in Fig. 2. *Histologic data: A, B, D,* Apical part of mesial root canal. *C,* Apical surface of mesial root. *A,* Hematoxylin-eosin-stained section. Reversal line indicating earlier resorption of canal walls, calcification in fully disintegrating pulp tissue. *B,* No bacteria in coronal part of canal in presence of chronic inflammatory cells. *C,* Brown & Brenn staining. Bacterial colonies on apical root surface. *D,* Brown & Brenn staining. Bacterial colonies in fully disintegrating pulp tissue in apical root canal.

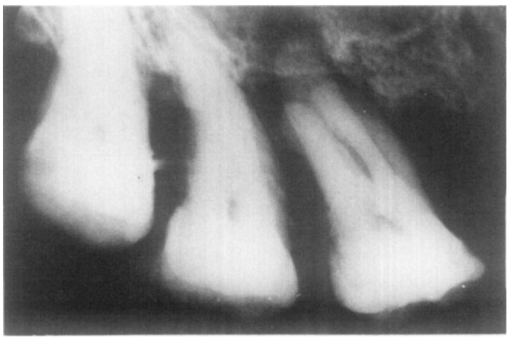

Fig. 6. Clinical data: Maxillary right first molar of 45-year-old woman. No pain. Caries free. Calculus, periodontal disease, and bone loss involving 3/3 of the roots. Periapical lesions involving all of roots.

Toward the apical portion of the distal canal, the pulp tissue appeared to be unaltered, apart from some resorption and apposition on the canal walls, together with some intrapulpal calcifications (Figs. 3, *B* and 4, *C* and *E*).

In the second example, a maxillary molar, the roentgenogram indicated periodontal involvement of all three roots (Fig. 6). The histologic sections showed disintegrating pulp tissue in all roots. There were large and numerous calcifications in the pulp chamber (Figs. 7, *B* and 8, *A*). In some areas, a concentration of neutrophilic leukocytes was observed (Figs. 8, *B* and *C* and 9, *B* and *F*). In the pulp chamber in all canals, including lateral canals, areas of full disintegration were observed (Figs. 8, *A* and *B* and 9, *A* and *C*). In these areas within the canals, colonies of bacteria were present (Fig. 9, *D*), whereas in the same section, bacteria were not present in the granuloma, although they were seen in the bacterial plaque on the apical root surface (Fig. 9, *E*).

Another important observation was made. Although clinically and roentgenographically no caries was observed in these cases, the histologic sections revealed gingival caries in five cases. One of those showed the presence of bacterial plaque in the buccal gingival area (Fig. 10, *A*), associated with bacteria in the adjacent dentinal tubules (Fig. 10, *B* and *D*), and a concentration of inflammatory cells in the pulp in the area in which the involved dentinal tubules terminated in the pulp (Fig. 10, *C*).

DISCUSSION

Pulpal calcifications seem to be more prevalent with increasing periodontal involvement, without any conspicuous inflammation. However, since calcifications replace the vital tissue of the pulp, their presence is therefore considered to be a pathologic sequela of the periodontal involvement. Apposition and resorption of the walls of the pulp chamber and canals of the roots are considered to be in the same category. The demonstration in the same section of the presence of bacteria within the pulp and on the root surfaces, but absence of them in the attached granuloma, corroborates our earlier findings, but contradicts the findings of other investigators.[22]

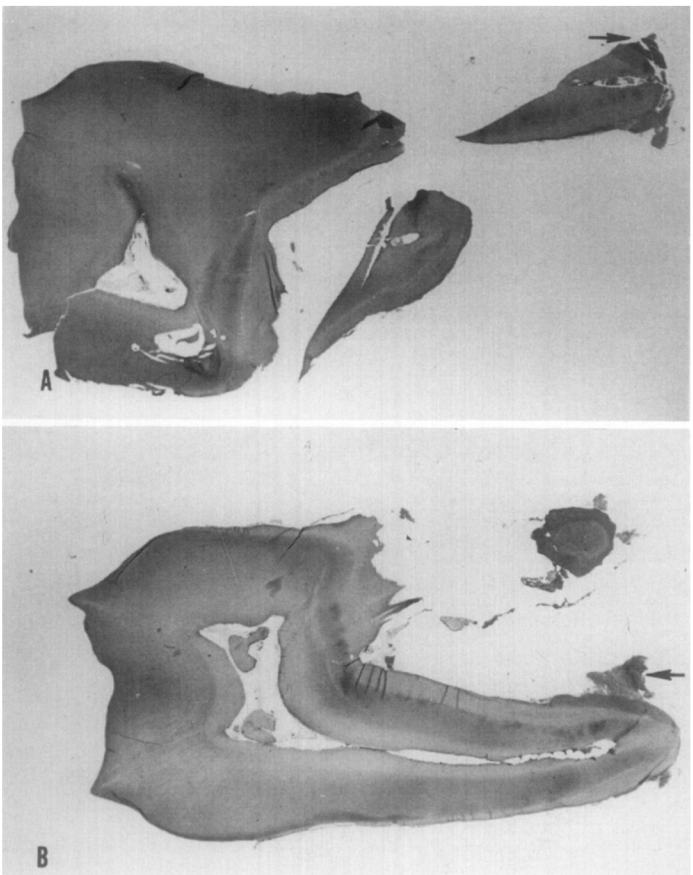

Fig. 7. Clinical data: As in Fig. 6. *Histologic data: A,* Overview of buccal roots. Granuloma *(arrow). B,* Overview of palatal root. Granuloma *(arrow).* (Magnification, ×8.5.)

The presence of lateral canals and the occurrence of root caries form the basis for a discussion of the possibility that the pulp could be influenced by a pathologic condition in the periodontal ligament or vice versa. The existence of this communicating structure (Fig. 1) has been established beyond doubt in a number of papers.[2, 8-10, 13, 15-17] Furthermore, it is common knowledge that disintegration products from the pulp will cause an inflammation in the periodontal ligament wherever a main or lateral canal terminates.[8-10, 15, 16] It has also been demonstrated that root caries causes a pulp inflammation (Fig. 10). Accordingly, the pathway for irritants from one location to the other is established. However, it is by no means certain that the entire pulp will succumb despite the fact that one or more lateral canals, or a number of dentinal tubules, are involved, as long as the main canal—the major pathway of circulation—is not seriously involved.

The reasoning behind this thought is to be found in the three specific examples described in this investigation. It was evident in our material that the pulp

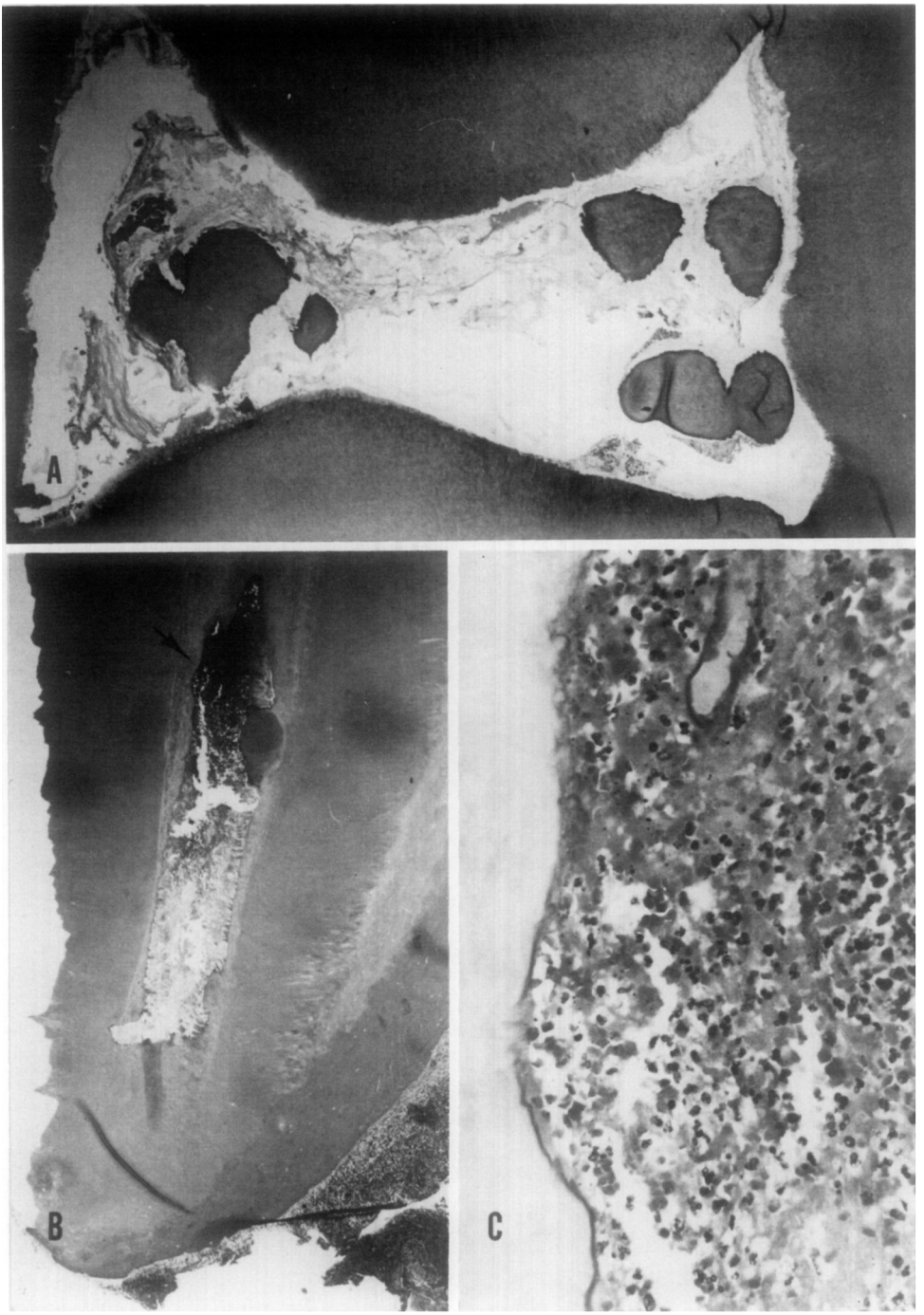

Fig. 8. Clinical data: As in Fig. 6. *A*, Coronal pulp with calcifications in fully disintegrating tissue. (Magnification, ×25.) *B*, Palatal root with concentration of cells *(point of arrow)* and fully disintegrating pulp tissue. *Lower left,* Lateral canal exiting from main canal. (Magnification, ×25.) *C*, Concentration of cells (neutrophilic leukocytes) from point of arrow in *B*. (Magnification, ×400.)

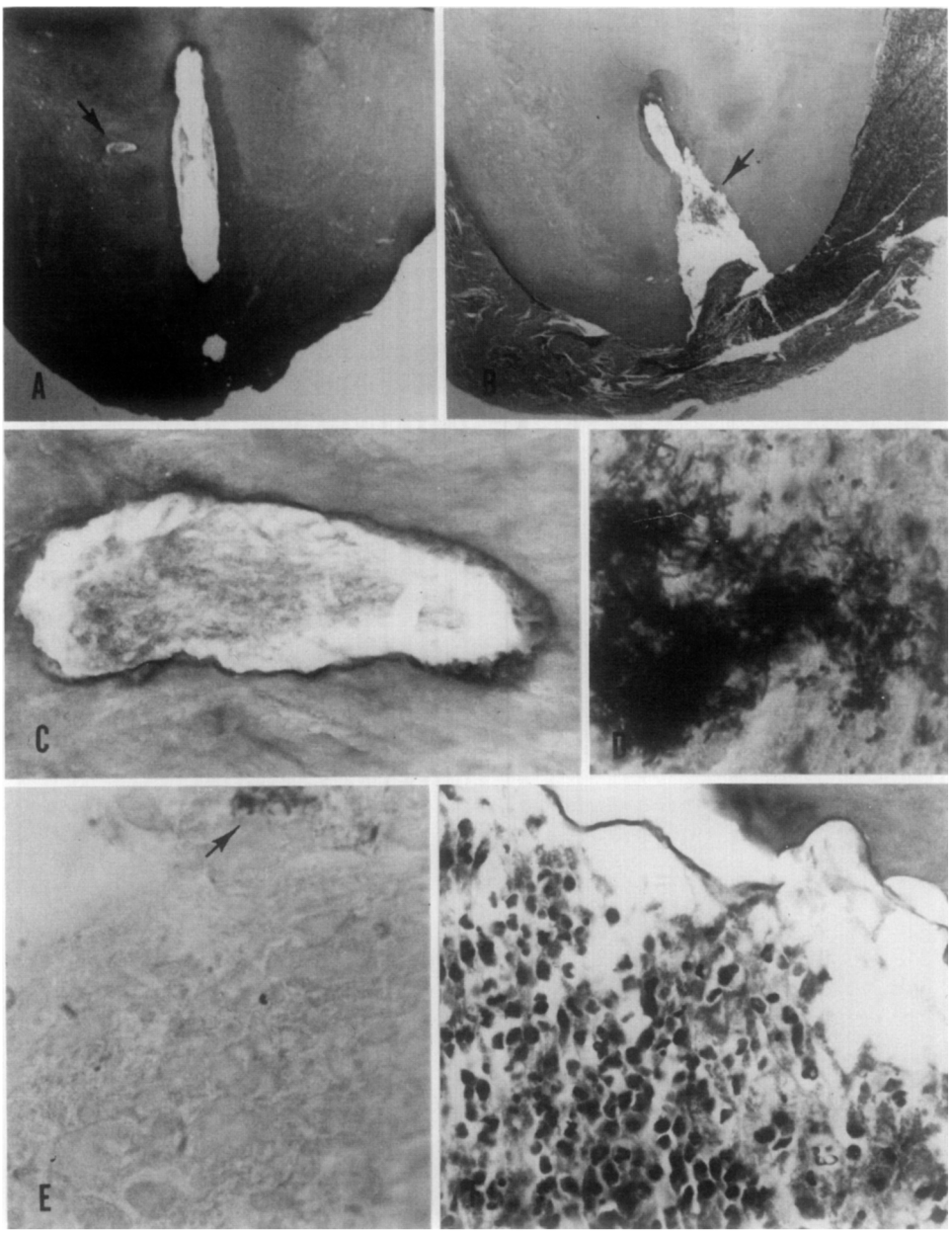

Fig. 9. Clinical data: As in Fig. 6. *A* and *B,* Buccal roots. (Magnification, ×25.) *A,* Remnants of disintegrating tissue. *Point of arrow,* Lateral canal. (Magnification, ×25.) *C,* From point of arrow in *A.* Remnants of pulp tissue in lateral canal, where bactreia are seen in neighboring sections stained by Brown & Brenn method. (Magnification, ×400.) *D,* Bacterial colonies in pulp chambers. (Magnification, ×1,000.) *E,* Brown & Brenn staining of section neighboring that shown in *B.* Granuloma. *Point of arrow,* Bacterial colony on root surface next to granuloma. No bacteria in granuloma proper. (Magnification, ×400.) *F,* From point of arrow in *B,* neutrophilic leukocytes. (Magnification, ×400.)

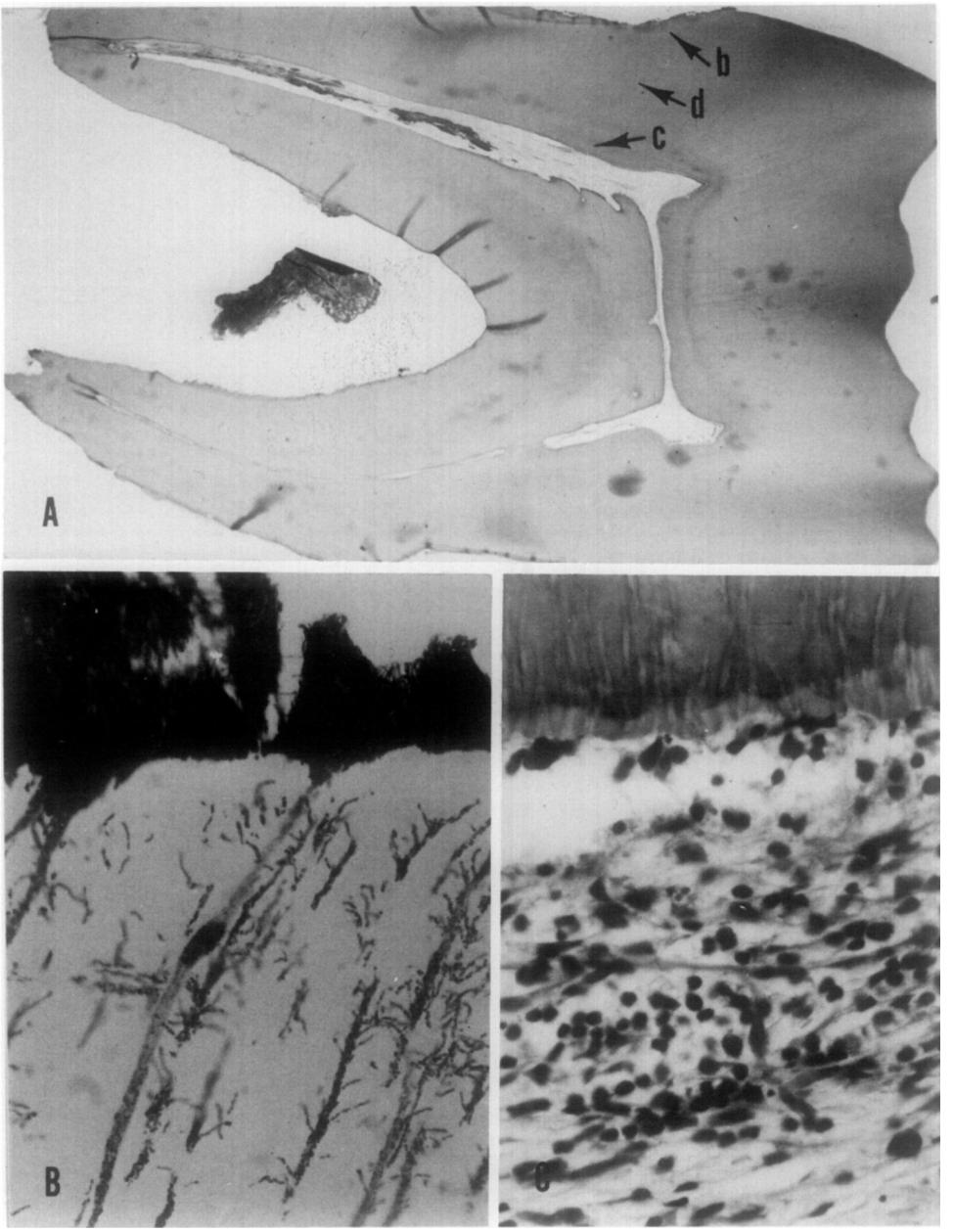

Fig. 10. Clinical data: Mandibular first molar of 49-year-old woman. No pain. Caries free. Calculus, periodontal disease, and bone loss from 2/3 of root. *Histologic data: A,* Observe narrow pulp chamber which is seen throughout all serial sections. (Magnification, ×8.5.) *B,* Bacterial plaque and adjacent dentinal tubules with bacteria *(c* in *A).* (Magnification, ×400.) *C,* Lymphocytes and macrophages where dentinal tubules invaded by bacteria terminate in the pulp *(c* in *A).* (Magnification, ×400.) *D,* Farthest penetration of bacteria in dentinal tubules *(d* in *A).* (Magnification, ×400.)

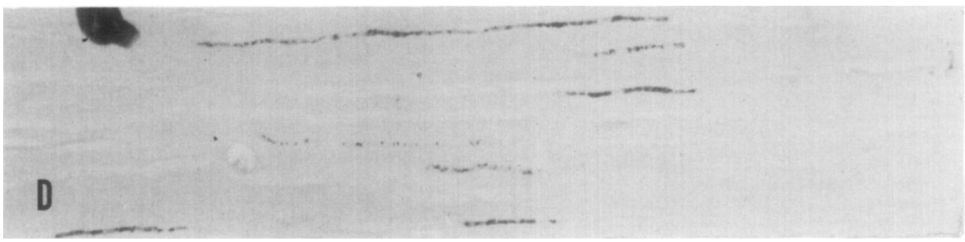

Fig. 10, D. For legend, see opposite page.

remained vital as long as one major canal was not involved (Figs. 2 to 5), whereas full disintegration occurred when the bacterial plaque involved all the canals (Figs. 6 to 9). The pulp also remained vital despite the presence of root caries (Fig. 10). It is interesting to note that a severe inflammatory process can be maintained in one canal, even though all circulation is cut off at the apex, the inflammation obviously being furnished with the necessary metabolites from the other canal. This allows the speculation that, even though the bacterial plaque has reached the orifice of a lateral canal, it may not result in total pulp destruction, although it may cause a localized inflammation where the involved canal terminates in the pulp.[8-10, 13, 14] These thoughts are further supported by the findings in the three-rooted tooth in which the bacterial plaque has reached all apices. In this case, the entire pulp tissue had disintegrated, except for the presence of a number of neutrophilic leukocytes (Figs. 8, *B* and *C* and 9, *B* and *F*). Since it is common knowledge that neutrophilic leukocytes survive, at the most, only a few days after having left the vessels and entered the tissue, a provision for their existence is that there are functioning vessels in this area. A part of a vessel can be observed in Fig. 8, *C*, although it cannot be followed through the root canal to the granuloma. However, functioning vessels and nerves in the root canal have been demonstrated earlier in teeth with well-developed granulomas.[16] Accordingly, the presence of neutrophilic leukocytes in one of the canals of the upper molar (Fig. 6) indicates that the vessel in Fig. 8 is still functioning although we have missed the direct connection to the granuloma despite serial sectioning.

It is well established that initial dentin caries in the crown causes a pulpal inflammation.[19, 21] Therefore, it is no surprise that root caries causes a similar pulp involvement. This type of pulp involvement is directly related to bacterial plaque on the root surface. Whether the cementum is mechanically removed, abraded, eroded, or destroyed by caries is irrelevant, as long as the bacteria obtain access to the adjacent dentinal tubules (Fig. 10).[2, 18, 23, 24]

The cumulative effect of periodontal disease, as indicated by the factors of calcifications, apposition of calcified tissue, resorption, or inflammation from root caries or from involved lateral canals, will be damaged pulp tissue, but total disintegration is a certainty only when all main apical foramina are involved by bacterial plaque.

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