0099-2399-84/1002-0048/$02.00/0
JOURNAL OF ENDODONTICS

Printed in U.S.A.
VOL. 10, No. 2, FEBRUARY 1984

The Histopathogenesis of Vertical Root Fractures

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Fractured roots were examined histologically, using different stains, to study the morphological fracture patterns and to identify potential irritants. Fractures were usually, but not always, complete and extended from a surface to include the root canal. The potential irritants identified in both the fracture space and communicating canal were bacteria, necrotic tissue, food debris, and unidentifiable amorphous substances. Soft tissues on the root surface adjacent to the fracture were inflamed with occasional ingrowth into the fracture space. The nature of the fractures and irritants demonstrated why vertical root fractures often cause marked tissue destruction.

A vertical root fracture is a devastating *occurrence.* Once diagnosed, the fractured root or tooth usually has an unfavorable prognosis (1-3) and requires removal.

The published information on vertical root fractures is a series of case reports (4-6) and a clinical study (7). Meister et al. (7) described the signs, symptoms, and radiographic findings present in a series of clinical cases. Although there were reported variations in diagnostic findings, they concluded that the problem was usually severe because of significant damage to the periodontium. The outcome of the majority of the cases was extraction of the affected tooth or removal of the fractured root in molars.

The clinician and investigator alike are impressed with tthe profound effect that the vertical root fracture has on the periodontium adjacent to the tooth. Bone loss is often rapid and soft tissues may manifest swelling or suppuration. Probing usually reveals a deep periodontal defect (4, 7-9).

Several causes of vertical root fractures have been proposed; such as wedging of endodontic posts (6), expansion of posts or pins from corrosion (10, 11), or excessive force during lateral condensation (7). However, the precise etiology or etiologies of the accompanying tissue destruction have not been clarified. There are no published reports pertaining to histological or bacterial findings which might clarify the source and location of the irritants which lead to the associated pathogenesis.

OBJECTIVES

The purpose of this study was to histologically examine roots and adherent tissues that were removed after the diagnosis of vertical fracture. Specifically, the specimens were studied to ascertain the pattern of the fractures and the nature and location of potential irritants. The resultant findings might then be used to explain the marked tissue response that occurs adjacent to the fracture line.

MATERIALS AND METHODS

The specimens utilized in the study were fractured roots obtained following the diagnosis and determination that their prognosis was hopeless because of significant tissue damage. A total of 36 roots were available for subsequent sectioning, staining, and microscopic analysis.

Immediately upon extraction, the roots or teeth were immersed in 10% neutral buffered formalin. The specimens were then decalcified in 10% formic acid and infiltrated and embedded in paraffin. Step serial sections were cut with the blocks oriented two ways. The majority of the roots were cut in cross-section at the cervical, middle, and apical thirds while the remaining roots were oriented so that longitudinal sections could be cut in order to study the fracture from the cervix to the apex.

From each area, adjacent sections were subjected to three different types of stains: (a) hematoxylin and eosin, to study general soft and hard tissue morphology and changes; (b) bacterial stains (Brown and Brenn or McCallum's), to identify bacteria; and (c) Wilder's reticulum, to characterize granulation (reparative) tissue.

All sections were examined, and photomicrographs were made with a Zeiss II photomicroscope. The specimens were examined for the following: extent and location of fracture lines; location, concentration, and nature of bacteria in canals, fracture spaces, and adjacent periodontal tissues; nature of the contents of the canal adjacent to the fracture; evidence of and identification of foreign debris in the fracture space; and presence of obturating materials in the fracture space and adjacent periodontal tissues

For this report, no attempt was made to correlate the associated clinical and radiographic findings with the histopathology.

RESULTS

All of the specimens demonstrated marked changes in both the hard and soft tissues. Furthermore, most histological sections demonstrated irritants in the fracture spaces, which could potentially have contributed to the inflammatory response observed clinically and histologically in the adjacent periodontium.

Fracture Characteristics

The fractures were readily visible on all sections. The majority (90%) of the fractures observed in the roots cut in cross-section were complete; meaning that the fracture passed completely through the root to include opposite surfaces (Fig. 1). The remaining 10% of the fractures were incomplete; the fracture extended only to one surface of the root (Fig. 2). Of the longitudinally sectioned roots, the majority showed complete fractures that extended from the cervix to the apex. When the fracture was confined to the root or appeared to have initiated apically, the fracture was in a facial-lingual direction. In contrast, the fracture was mesial-distal when molar and premolar teeth had crown-root fractures which appeared to have initiated from the crown.

Regardless of whether the fractures were complete or incomplete, all extended into the root canal. When the root contained two canals, the fracture included both canals (Fig. 1). In addition to the major fracture (or primary fracture) lines that extended from the surface of the root to the canal, there frequently were secondary fractures. These did not extend to the root surface but emanated from the canal or primary fracture to form a blind-ending within the dentin.

Bacterial Characteristics

A frequent finding in the fracture spaces was bacteria (Figs. 3 and 4). Brown and Brenn, and McCallum stained sections showed both gram-positive and gram-negative bacteria, with a predominance of gram-positive, within the canals of 32 of the 36 specimens studied. Frequently, the bacteria were seen in defects, secondary fractures, or dentinal tubules that communicated with the fracture (Figs. 5 and 6). Generally, the bacteria were in low concentrations. However, there were occasionally dense accumulations of bacteria in a pattern resembling plaque.

Canal Contents

Frequently, necrotic tissue, bacteria, and amorphous debris were observed in the canal space adjacent to the communicating fracture line or lines (Fig. 7). Occasionally, these would be seen to extend into the primary and secondary fracture spaces.

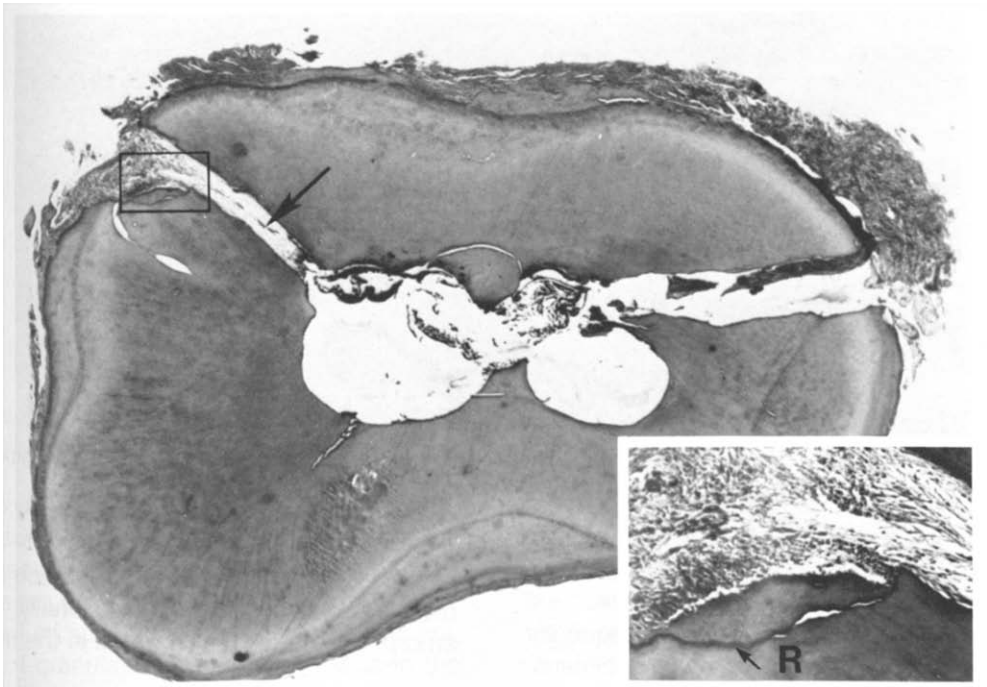


FIG 1. Complete fracture seen in cross-section of the middle one-third of the root. It extends from facial to lingual and includes both canals. Note tissue ingrowth *(arrow)* (hematoxylin and eosin; original magnification x25). *Insert:* higher magnification of area indicated by the *box.* Note the reversal line (R), which indicates earlier resorption on the fracture surface followed by apposition of cementum-like tissue (original magnification ×100).

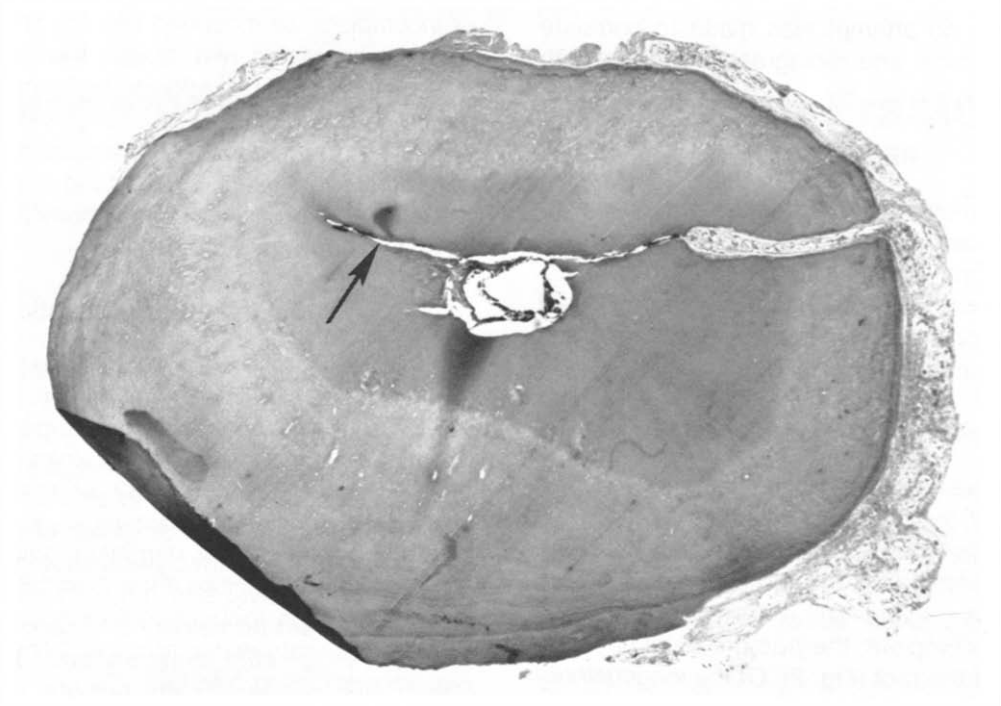


FIG 2. Incomplete fracture includes one surface and canal. Note that the fracture extends beyond the canal *(arrow)* but not to opposite root surface (hematoxylin and eosin; original magnification x25).

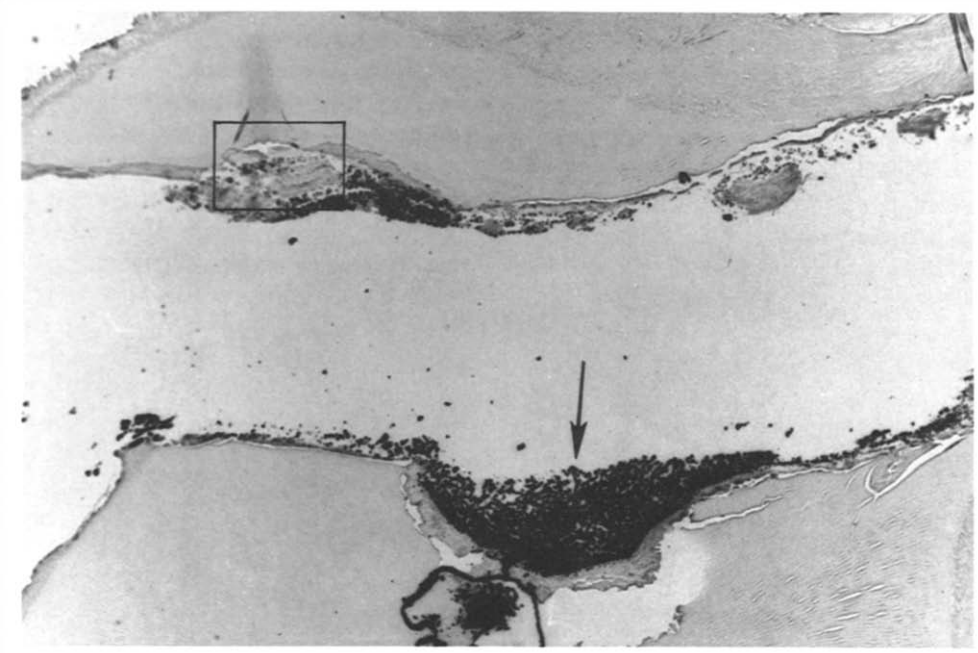


FIG 3. Fracture extending through canal which contains gutta-percha and sealer *(arrow).* The wide separation of the root segments is preparation artifact (Brown and Brenn gram stain; original magnification ×110).

Fracture Contents

Foreign materials of various types were frequently observed in the fracture spaces. In two specimens, which showed fractures extending into the crown, the spaces were filled with food debris mixed with bacteria (Figs. 8 and 9). Gutta-percha and sealer (as identified by polarized light) often appeared in the primary and secondary fracture spaces and in communicating tubules (Figs. 10 and 11). Occasionally, an unidentified amorphous material was found in the fracture spaces.

Tissue Reaction

In many specimens, tissue was adherent to the root surface adjacent to the fracture (Fig. 12). Examination

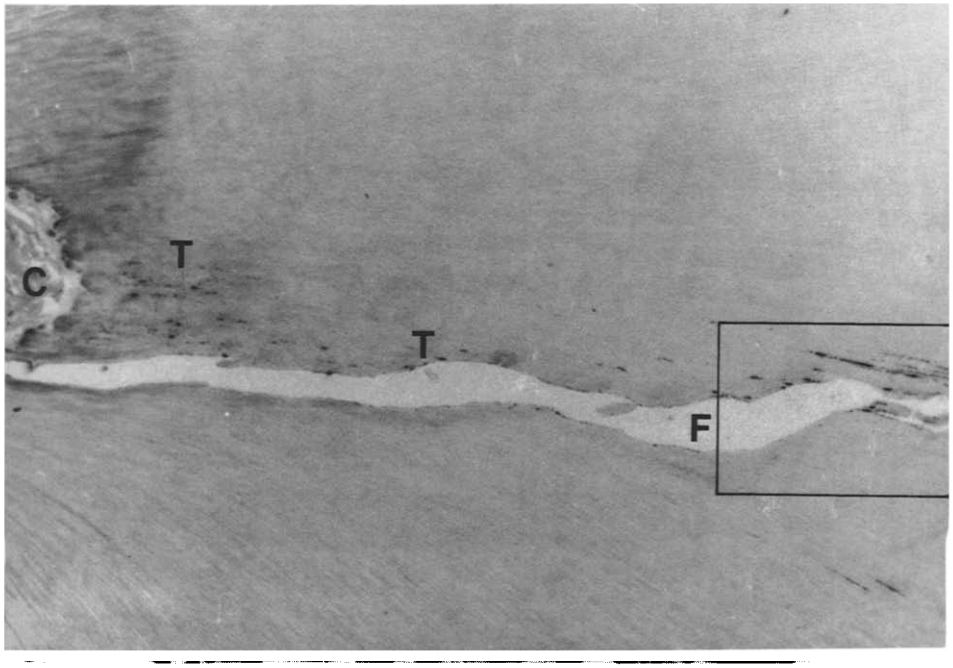


FIG 4. Higher magnification of *boxed area* indicated in Fig. 3. Dense accumulations of bacterial colonies *(arrows)* line the walls. Erythrocytes (E) are a frequent finding (Brown and Brenn stain; original magnification x520).

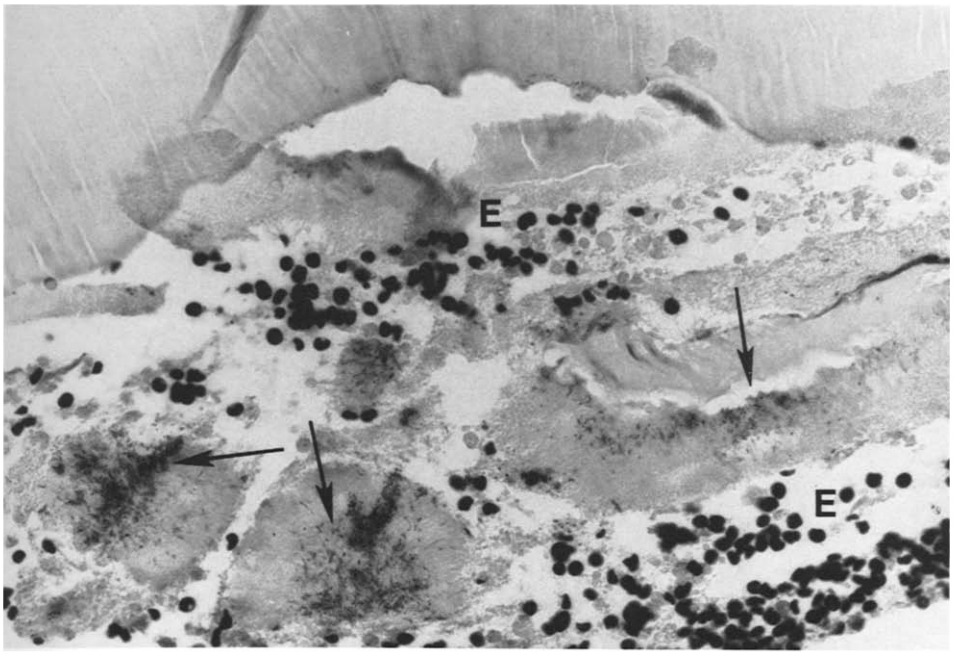


FIG 5. Bacteria inhabit the fracture space and dentinal tubules (T). The tubules communicate with both the canal (C) and fracture (F) (McCallum's stain; original magnification x160).

of the hematoxylin and eosin sections showed that, invariably, this soft tissue was inflamed. Principally, the inflammation was chronic and this granulomatous tissue was often seen extending into the fracture space.

Wilder's reticulum stain, specific for reticulum fibers, showed areas of granulation (reparative) tissue in the fracture space. This ingrowth would occasionally extend through the fracture into the canal or canals.

Scattered areas of resorption were often visible along the fracture line. Many of these areas showed reversal lines overlayed by cementum-like tissue which apparently formed after the resorption (Fig. 1). The reticulum stain also showed fibers inserted into this cementum-like tissue that had formed on the raw dentin surface within the fracture (Fig. 13).

DISCUSSION

This study identified several probable and potential irritants at the fracture site. However, the *precise* **etiol-**

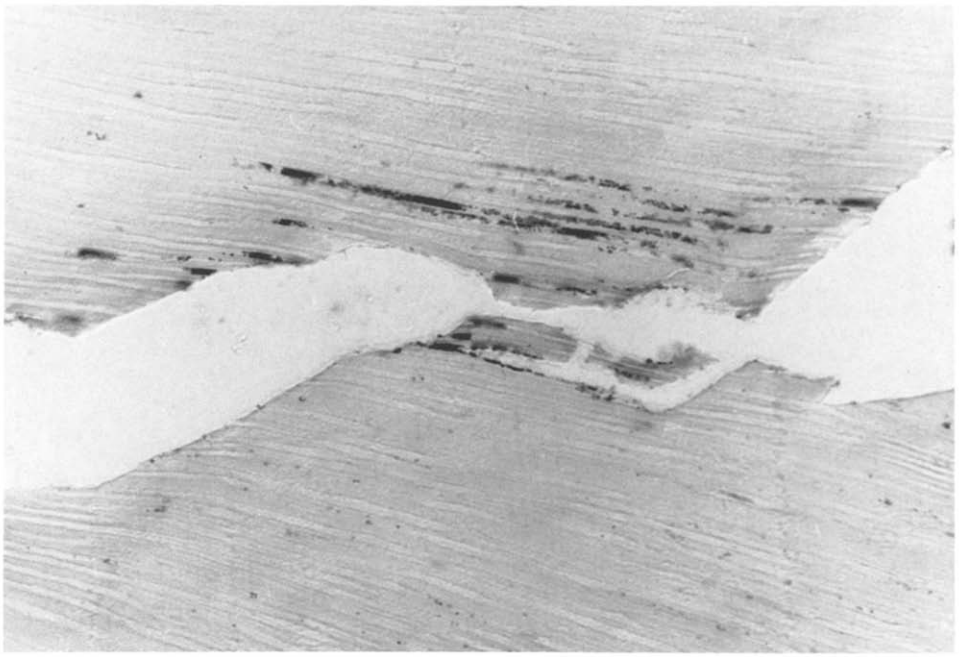


FIG 6. Higher magnification of *boxed area* in Fig. 5. Bacteria occupy tubules that open into the fracture space (McCallum's stain; original magnification x410).

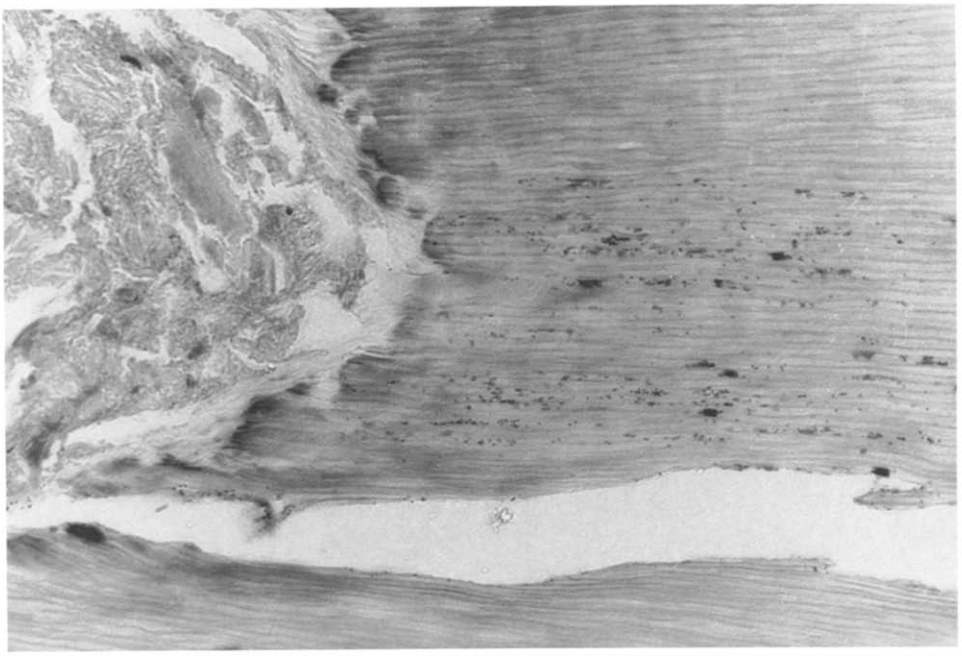


FIG 7. Region indicated by C in Fig. 5 shows bacteria in canal debris and in adjacent tubules. The debris is in a region that was unprepared by endodontic instruments (McCallum's stain; original magnification x410).

ogical agent or agents that cause the periodontal inflammation was not definitely determined. The agents which were associated with the fracture and which, in other studies, have demonstrated irritating properties were bacteria and their metabolites (12, 13), necrotic pulpal tissue (14), sealer components (15, 16), and food debris forced into the fracture during mastication. Another possibly significant irritant is the disintegration products of percolating tissue fluids (17).

The number of bacteria in the fracture were small, but this may not represent the true picture. Histological stains are nonspecific and are not quantitative. Generally, numbers of bacteria are many times greater than those shown by the actual staining.

There are several potential sources for the bacteria seen in the fracture. One is anachoresis. However, this is unlikely, as bacteria could not be introduced into empty pulp spaces (18), or even into pulp canals con-

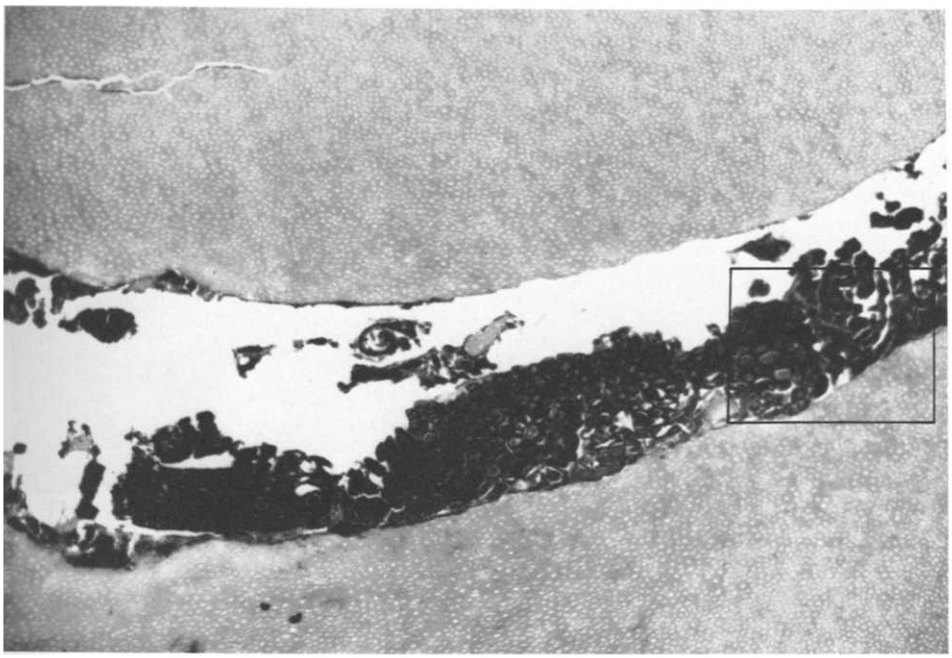


FIG 8. Vegetable matter and other debris in a fracture space (hematoxylin and eosin; original magnification x160).

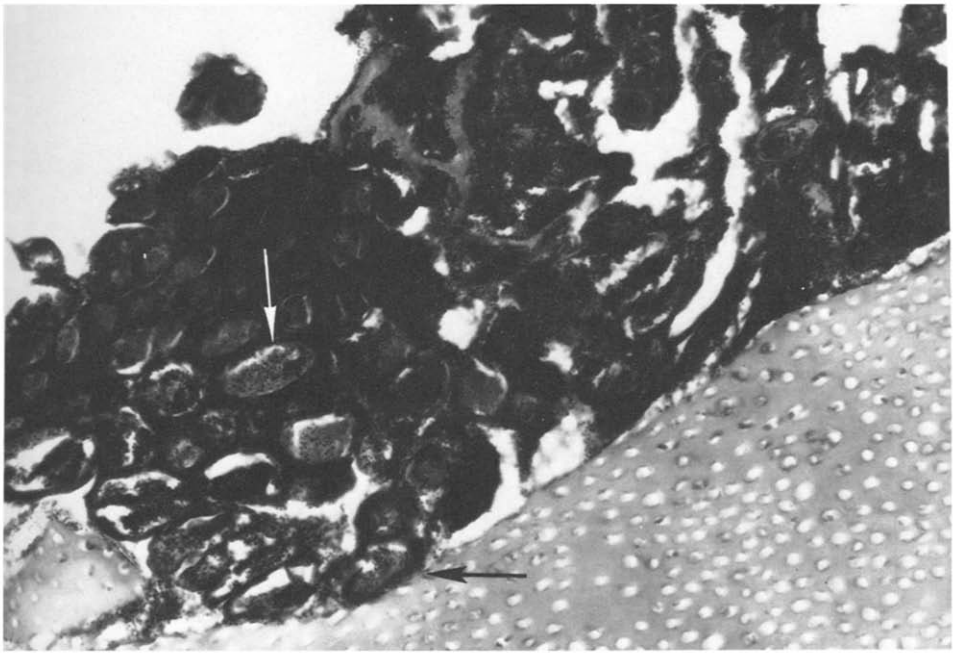


FIG 9. Higher magnification of *boxed area* in Fig. 8. Compartmentalized areas *(arrows)* appear to be "skeletons" of vegetable cell walls filled with bacteria (hematoxylin and eosin; original magnification x520).

taining necrotic tissue (13). Another source may be direclty from the oral cavity when the fracture communicates with the gingival sulcus. The most interesting Possibility is from the canal space itself. Often bacteria are not totally removed during canal preparation (19, 20). Following obturation, some of these microorganisms may survive in an inactive state. However, with a subsequent fracture, substrates may enter the pulp canal, allowing the microorganisms to proliferate and to produce virulence factors. It is readily accepted that bacteria are an important etiological factor for periapical inflammation (21). The same principle should apply to the fracture, with the exception that a greater area of exposure is provided by the fracture.

Significantly, these bacteria were never observed in vital tissue. This demonstrated either their low pathogenicity or high host resistance.

It was significant that necrotic debris was frequently

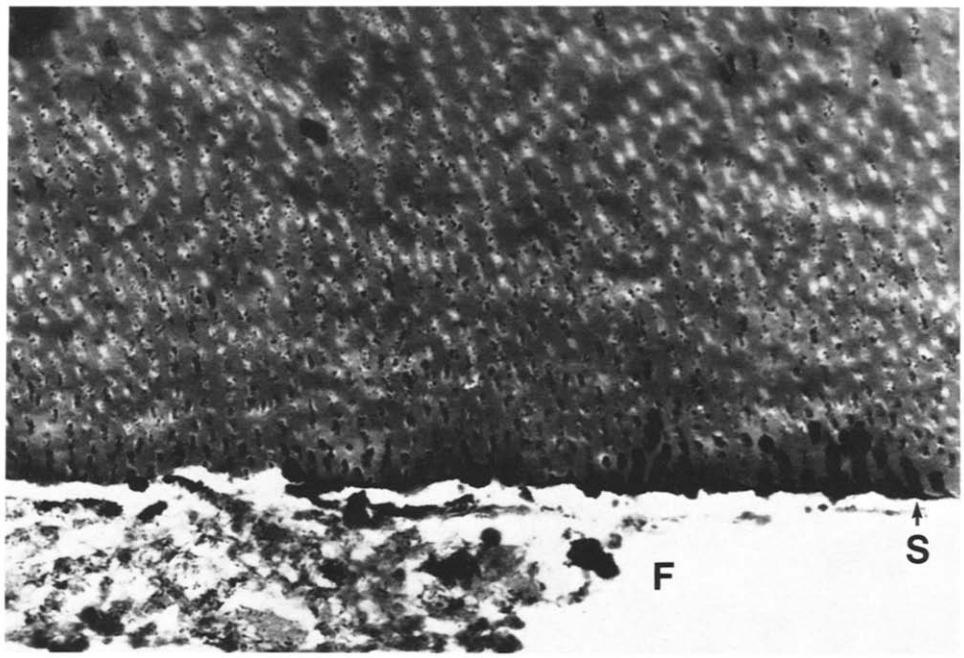
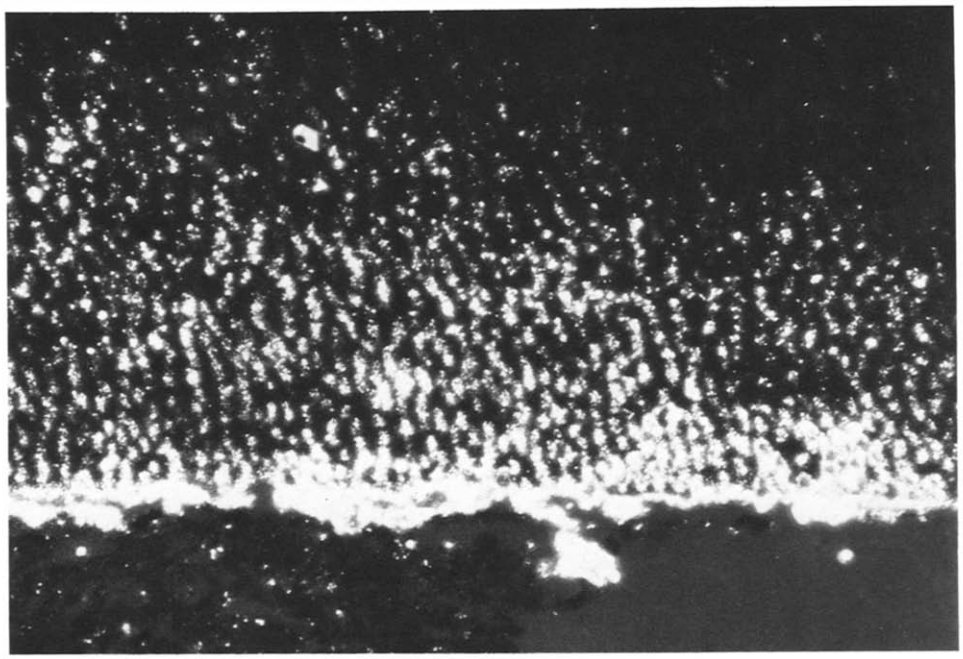


FIG 10. Fracture space (F) and adjacent tubules contain a dense substance (s) (hematoxylin and eosin; original magnification x410).



F~G11. Polarizing microscopy of the same region shown in Fig. 10 proves this dense substance to be sealer, which is birefringent. The sealer was probably forced into the fracture which was created during condensation (hematoxylin and eosin; original magnification x410).

found in the canal space adjacent to the fracture. It could not be ascertained whether this debris was originally in the canal, or had seeped in via the fracture from the oral cavity or surrounding periodontium. It is likely, however, that this necrotic tissue represented remnants which are frequently found in the canal system after debridement (22-25). Normally, this debris would be sealed in the canal and inaccessible to the periodontal tissues. A fracture, however, would proivde easy access to the periodontium.

Sealers are subject to the same principles. Their irritating components are largely confined to the canal space after obturation. A fracture line would provide extensive contact between the sealer and the periodontium, resulting in a source of continual irritation.

Food debris, saliva, and their myriad components may certainly be important irritants. These substances from the oral cavity may also supply the substrates for plaque formation when forced by mastication into a communicating fracture. This may result in the periodontal breakdown and the deep probing defects usually associated with vertical root fractures (7).

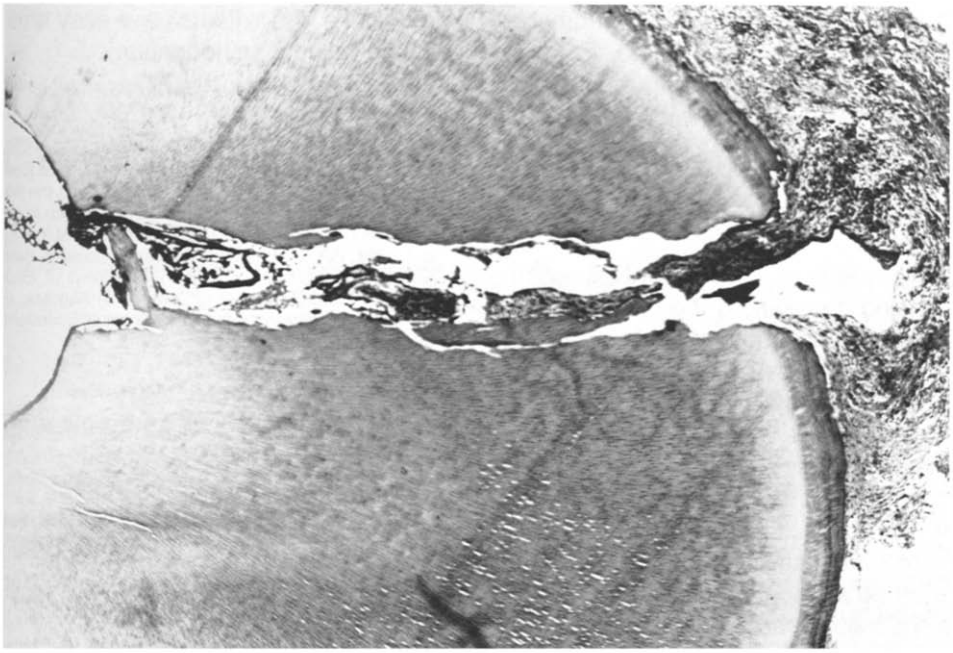


FIG 12. Tissue adherent to root surface adjacent to fracture has a dense infiltrate of chronic inflammatory cells. A finger of this inflammatory tissue extends into the fracture space (hematoxylin and eosin; original magnification xS0).

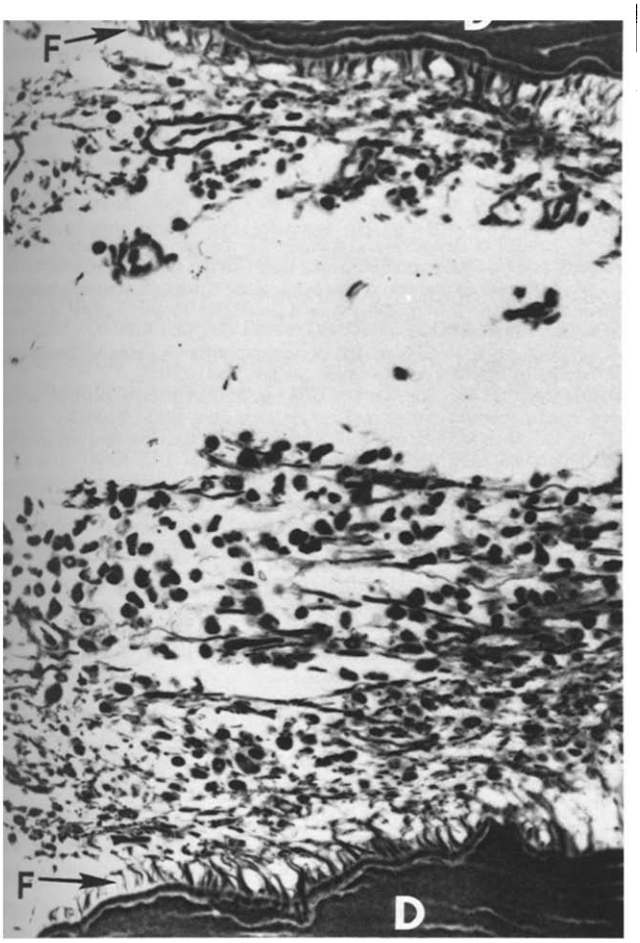


FIG 13. Special stain shows fiber patterns in the fracture space. Small fibers (F) insert directly into a thin layer of cementum-like tissue that overlays dentin (D) (Wilder's reticulum stain; original magnification ×410).

Percolation and breakdown of tissue fluids within the fracture may be an important irritant. Although the "hollow tube theory" (17) is disputed (26, 27), the fracture represents a considerable area of exposure to the canal space for substances to enter, deteriorate, and then exit as irritants. Again, the critical factor is that the tissue area exposed to a vertical fracture is much larger than that afforded by an apical foramen.

The fracture patterns and their contents gave an indication of the time of the occurrence of the fracture. If sealer was not present in the fracture space in the histological section, the fracture may have occurred at some time following obturation or post placement. The possibility of a delayed fracture is plausible. Meister et al. (7) presumed that many of the root fractures occurred at varying periods of time after obturation or post placement. Stresses created in the root at the time of treatment may manifest themselves as strains (fractures) at a later date.

The presence of sealer in the fracture space, however, would not guarantee that the fracture occurred at the time of obturation. Being that substances do migrate (28), it is possible that sealer moved into the space after the actual fracture. Another consideration is that the sealer present in the fracture site is an artifact resulting from its solubilization during histological processing. However, when sealer was found in the tubules communicating with the fracture (Figs. 10 and 11) or in the small secondary fractures, it could be assumed that this was the result of condensation forces.

The incomplete fracture is interesting both from a morphological and from a diagnostic standpoint. This phenomenon demonstrates that the dentin has suffi-

cient elasticity to permit separation of root segments without a through-and-through, or complete, fracture. This could present difficulties in visualizing the fracture clinically. If the fracture is only on the lingual and does not extend to the facial, it could not be observed during a surgical procedure if only a facial flap is reflected. This obviously would complicate the diagnosis of a vertical root fracture.

The inflammation present in the adjacent periodontium was not surprising considering the variety of potential irritants already discussed. What *was* surprising was how far the inflamed tissue extended into the fracture site from the root surface. In some specimens, tissue filled the entire fracture space. Why this tissue demonstrated considerably less inflammation than that on the adjoining root surface cannot be explained.

Also significant was the capability of this tissue to supply cells to form hard tissue on the walls of the fracture. However, in no section was there an attempt to "bridge" or heal the fracture as may occur in horizontal root fractures (29). This hard tissue formation presumably was only a reactive phenomenon which is induced when connective tissues contact dentin.

SUMMARY AND CONCLUSIONS

Extracted roots with vertical root fractures were examined histologically. Hematoxylin and eosin, bacterial stains, and reticulum Stain were used to study morphological patterns of the fracture, the status of associated soft tissues, and to identify potential irritants. The sample included 36 roots. The following observations and conclusions were made:

1. The majority of fractures were complete in that they extended from a peripheral surface to the opposite surface and included at least a canal or canals. A few specimens showed incomplete fractures.
2. Bacteria were demonstrated in most fracture spaces, occasionally in communicating canals, and often in secondary fractures and/or tubules.
3. Canals that were continuous with the fractures often contained potential irritants such as food debris, sealer, necrotic tissue, bacteria, and unidentifiable amorphous substances.
4. Soft tissue on the root surface adjacent to the fracture always demonstrated inflammation. Tissue ingrowth was frequently observed in the fracture space and would often be seen along its entire length. Exposed dentin in the fracture spaces was occasionally overlayed by cementum-like hard tissue.

The fracture line appears to form an open pathway for irritants from the oral cavity and from the canal space to adjacent tissues. Because the fracture is extensive, the irritants have easy and extensive access to the reactive periodontium.

We wish to thank Mrs. Dame, Taylor for her patience and care in preparing this manuscript.

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