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# A histologic evaluation of periodontal tissues adjacent to root perforations filled with Cavit

Ronald C. K. Jew, D.D.S., M.S.,\* Franklin S. Weine, D.D.S., M.S.D.,\*\*  
Joseph J. Keene, Jr., D.D.S., M.S.,\*\*\* and Marshall H. Smulson, D.D.S.,\*\*\*\*  
Maywood, Ill.

LOYOLA UNIVERSITY SCHOOL OF DENTISTRY

Root perforations were created in dog premolars; thirty-one were filled with Cavit, whereas seventeen were left unfilled. The animals were killed after 1, 15, 30, 60, 120, and 180 days, and hematoxylin and eosin sections were prepared. Perforations near the gingival sulcus usually led to severe destruction, whereas those surrounded by ample bone tended to gain a favorable response, even when left unfilled.

An endodontic perforation may be defined as an artificial opening in a tooth or its root, created by boring, piercing, cutting, or pathologic resorption, which results in a communication between the pulp canal and the periodontal tissues. As a consequence, the retention of an involved tooth may be compromised. In an analytic study of endodontic failures in 1961, Ingle<sup>1</sup> reported that root perforations were the second greatest cause of failure, accounting for 9.62 percent of all unsuccessful cases, and that incomplete filling of the canal space was the leading cause, with 58.65 percent of the failures. Seltzer and associates<sup>2</sup> attributed 3.52 percent of endodontic failures to perforations and added that such accidents often occurred without the knowledge of the operator.

Perforations of the root or pulp chamber are among the most common problems associated with endodontic procedures. Attempting to locate canals hidden beneath a large restoration may frustrate even the most experienced operator. Greater life expectancy and superior dental procedures have allowed for increased preservation of teeth, but many

of these retained members of the arch have decreased canal size, making location difficult. Procedural accidents may occur in the confined area of the treated tooth, even when one has knowledge of dental anatomy and topographic relationships.<sup>3-6</sup> Inadequate access preparation may result in misdirection of a bur while one is gaining entrance to the pulp canals and a perforation may be inadvertently created in seconds.

Perforations may also occur near the apex of a tooth, causing a "false" canal to leave the main canal. This usually is found in a tooth where the canal has an apical curve or dilaceration and the "false" canal comes off tangentially to the main canal (Fig. 1, A).<sup>7,8</sup> Another cause of perforation may be the misuse of rotary instruments in preparing room for a post or dowel (Fig. 1, B). Still another is the "stripping" of a root as a result of excessive enlargement of the middle third of the canal. This generally occurs in teeth that have a figure-of-eight shape in cross section, such as the mesial root of a mandibular molar (Fig. 1, C) or the mesiobuccal root of a maxillary molar.<sup>7</sup>

Numerous investigators have commented that the most favorable prognosis results when a perforation is sealed immediately after it is encountered.<sup>3,9-17</sup> Failure to seal the defect immediately permitted rapid periodontal breakdown which greatly complicated later attempts at repair because of the decreased likelihood of regeneration of the periodontal fibers and the alveolar bone. Many authors have

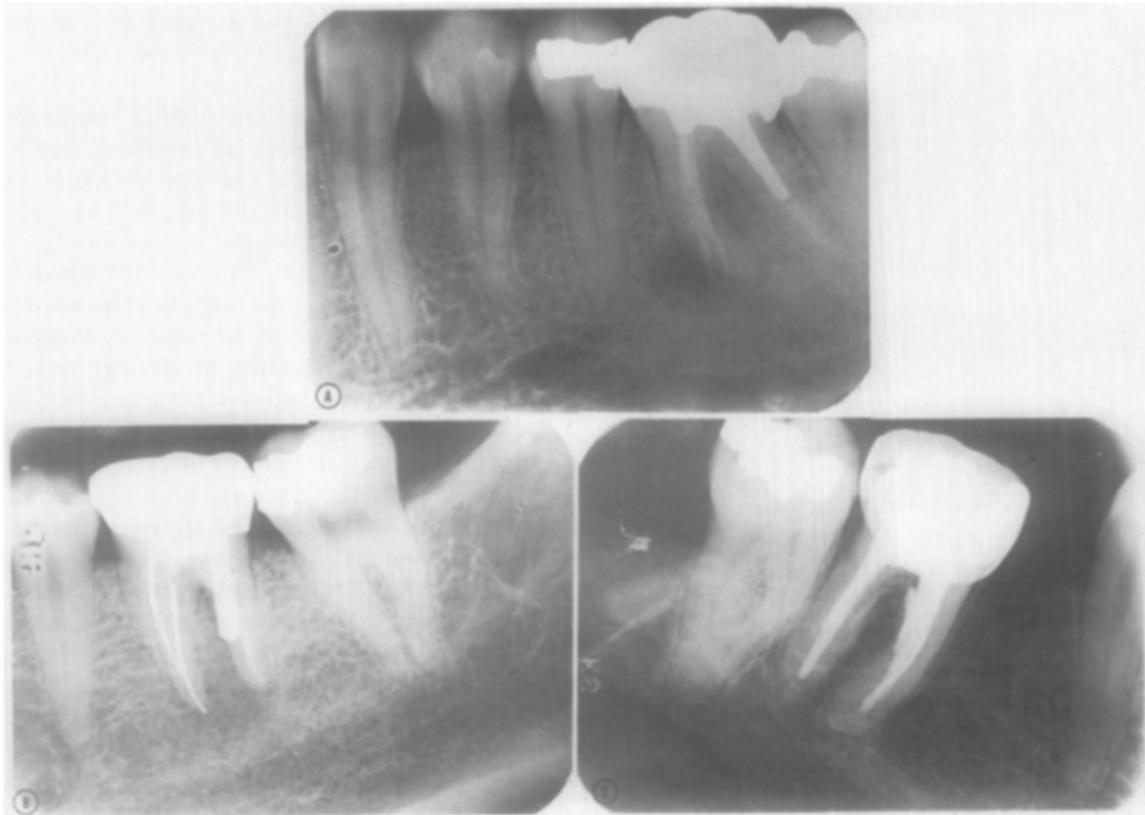
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\*Formerly Graduate Student; now in private practice, San Francisco, Calif.

\*\*Professor and Director Graduate Endodontics.

\*\*\*Associate Professor of Periodontics.

\*\*\*\*Professor and Chairman, Department of Endodontics.



**Fig. 1.** A, Perforation near the apex on the mesial root of the mandibular first molar caused by improper canal preparation. B, A rotary instrument was used to make post room in the distal root of the mandibular first molar, with a resultant perforation. C, A "strip" perforation on the mesial root of the mandibular first molar caused by excessive flaring of the middle third of the root which, in cross section, has a figure-of-eight shape.

reported that the location of the perforation site relative to the gingival sulcus has a definite bearing on the healing potential.<sup>11-18</sup> They found that perforations located in the furcation or coronal third of the root have a doubtful prognosis because of the proximity to the gingival sulcus.

Treatment of perforations may fall within the province of the endodontist, the periodontist, or the oral surgeon. Removal of the entire root or a portion of the root apical to the perforation is a common treatment in all three areas of specialization. Repair of the perforation from within the tooth with some solid or semisolid material is the method usually employed by endodontists. Surgical repair of the defect may be attempted by the raising of a flap to the site of perforation and enlarging the area with a small preparation to accommodate a temporary or permanent type of filling material.<sup>7</sup>

From a historical standpoint, as early as 1901, Guilford suggested that pulp chamber perforations be repaired with moistened plaster of Paris fixed in

place with a zinc phosphate cement.<sup>19</sup> In 1903 Peeso recommended copper and amalgam, and in 1904 Head described the use of gutta-percha, platinum sheet, and lead discs.<sup>19</sup>

Much more recently, Nicholls<sup>20</sup> and Taatz and Stiefel<sup>21</sup> suggested that small perforations of the pulp chamber be treated with a calcium hydroxide cement and then covered with amalgam or a well-condensed gutta-percha filling. Harris<sup>22, 23</sup> reported a clinical and radiographic evaluation of 159 patients in whom perforations were filled with Cavit; he claimed a success rate approaching 90 percent in a long-term follow-up.

Cavit was manufactured to be a temporary filling material and contains zinc oxide, calcium sulfate, glycol acetate, polyvinyl acetate, polyvinyl chloride-acetate, triethanolamine, and red pigment but no eugenol. The setting reaction is hygroscopic and is initiated in part by saliva, with the reaction with water, calcium sulfate, and zinc oxide-zinc sulfate producing the set.<sup>24</sup>



**Fig. 2.** A, radiograph of treated area after 60 days. Note separation of Cavit and thin radiolucent line around excess filling material (arrow). B, Radiograph of treated area after 180 days.

The purpose of this study was to investigate the histologic response of the periodontium to nonsurgical repair of endodontic perforations sealed with Cavit.

#### MATERIALS AND METHODS

Six adult beagle dogs weighing between 9.5 and 13.5 kilograms each were used as experimental subjects. They were kept on a diet of standard laboratory meal and water ad libitum and maintained at the Loyola University Medical Center Animal Research Facility. A total of thirty-six mandibular premolar teeth in twelve quadrants were operated on and examined in this study, allowing for examination of thirty-one perforations sealed with Cavit and seventeen control perforations.

Each animal was premedicated with an intramuscular injection of 1 c.c. of Inovar Vet\* per 7 to 9 kilograms of body weight for sedation and subcuta-

neous injection of 1 c.c. atropine sulfate injection USP\* (0.5 mg./ml.) to decrease salivary secretions.

Routine operating room sterility was employed, and general anesthesia of surgical depth was obtained via intravenous administration of sodium pentobarbital injection† (65 mg./ml.) by venipuncture on the dorsal surface of the foreleg in the anterior superficial vein. This provided approximately 2 hours of working time, and maintenance doses of 1 c.c. were administered as required. Preoperative radiographs for evaluation of alveolar crest level, indication of apical pathosis extending incisally, as well as patency of the root canal spaces were taken prior to any operative procedure.

The canals were located and prepared according to the method of Weine,<sup>7</sup> with sterile saline solution used as irrigant. Final instrument sizes ranged from No. 30 to No. 70 files, consistent with canal morphology and the age of the animal. The canals were dried with sterile absorbent paper points. Wach's Paste‡ was mixed and placed into the canals on a sterile file, and the canal space was obliterated with laterally condensed gutta-percha. The coronal portion of the gutta-percha filling was removed from the canals with heated endodontic pluggers and large files. Apical gutta-percha, 3 to 4 mm., was left in the canal to maintain an apical seal.

Engine-driven reamers, sizes 80 and 90, were used to create perforations through the side of the root, through the periodontal ligament, and into the adjacent periodontium. The lateral perforations were made toward the proximal aspect to allow for better visualization on radiographic and histologic examination, with only one perforation created in any one particular interproximal space to forego the possibility of confusing the histologic pictures should two perforations be made in the same immediate area from adjacent teeth. Each perforation was mechanically cleansed by irrigation with sterile saline solution, and bleeding was controlled by the use of sterile paper points. The experimental perforations were sealed with Cavit§ packed with endodontic pluggers. No attempt was made to limit the material strictly to the tooth, as overfilling with Cavit was judged to simulate actual clinical conditions and allow examination of the tissue response to the extruded material.

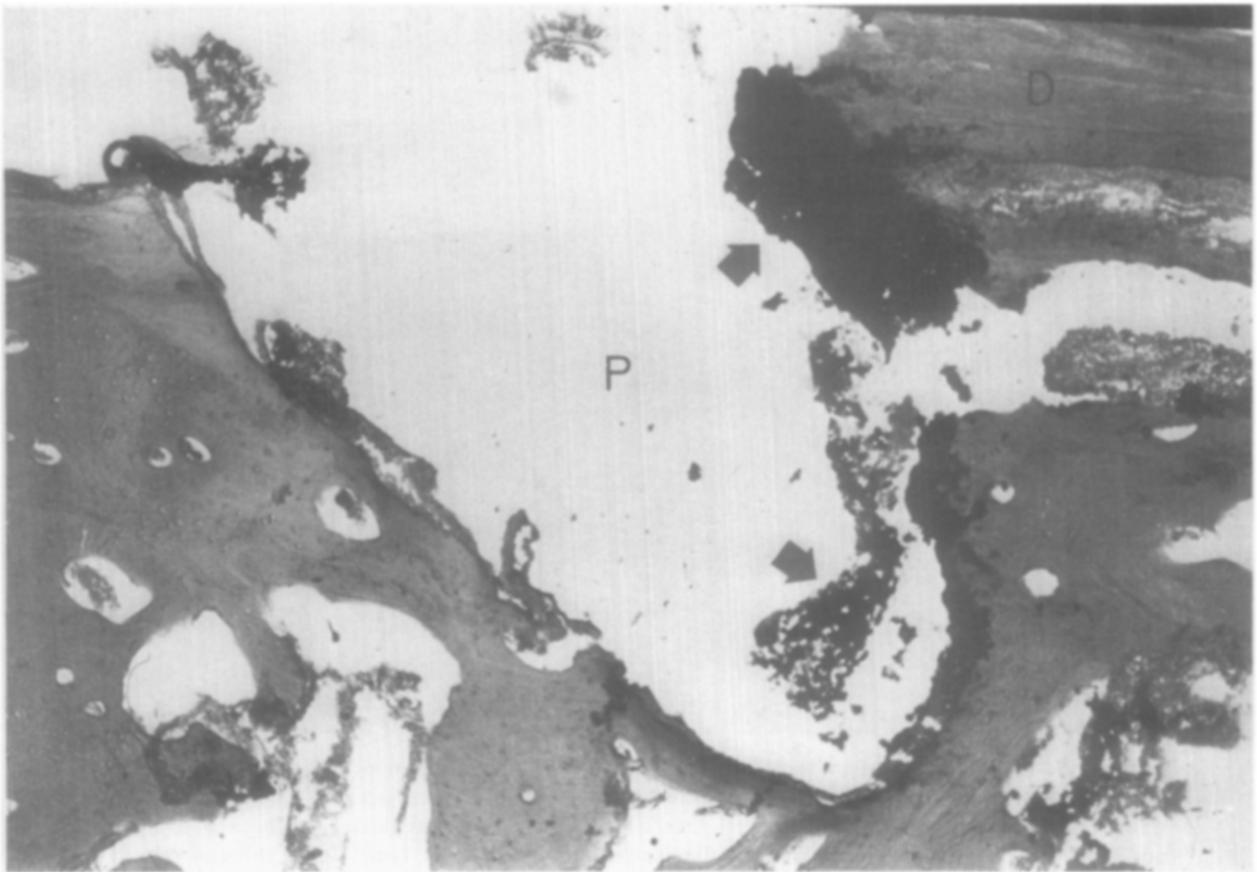
\*Med. Tech., Inc., Elwood, Kan.

†W.A. Butler Company, Columbus, Ohio.

‡King's Specialty, Ft. Wayne, Ind.

§Premier Dental Products Company, Philadelphia, Pa.

\*Pitman-Moore, Inc., Washington Crossing, N. J.



**Fig. 3.** Unfilled perforation after 1 day. Osteocytes are absent from Howship's lacunae. There are considerable signs of inflammation and osseous activity. The perforation (*P*) is lined with hemosiderin (arrows) and dentin (*D*). (Hematoxylin and eosin stain. Original magnification,  $\times 40$ .)

The control teeth were similarly perforated and bleeding was controlled, but no attempt was made to seal the perforation other than through the placement of a cotton pellet adjacent to the perforation within the tooth. In all teeth the coronal accesses were sealed with temporary stopping and amalgam, and postoperative radiographs were taken.

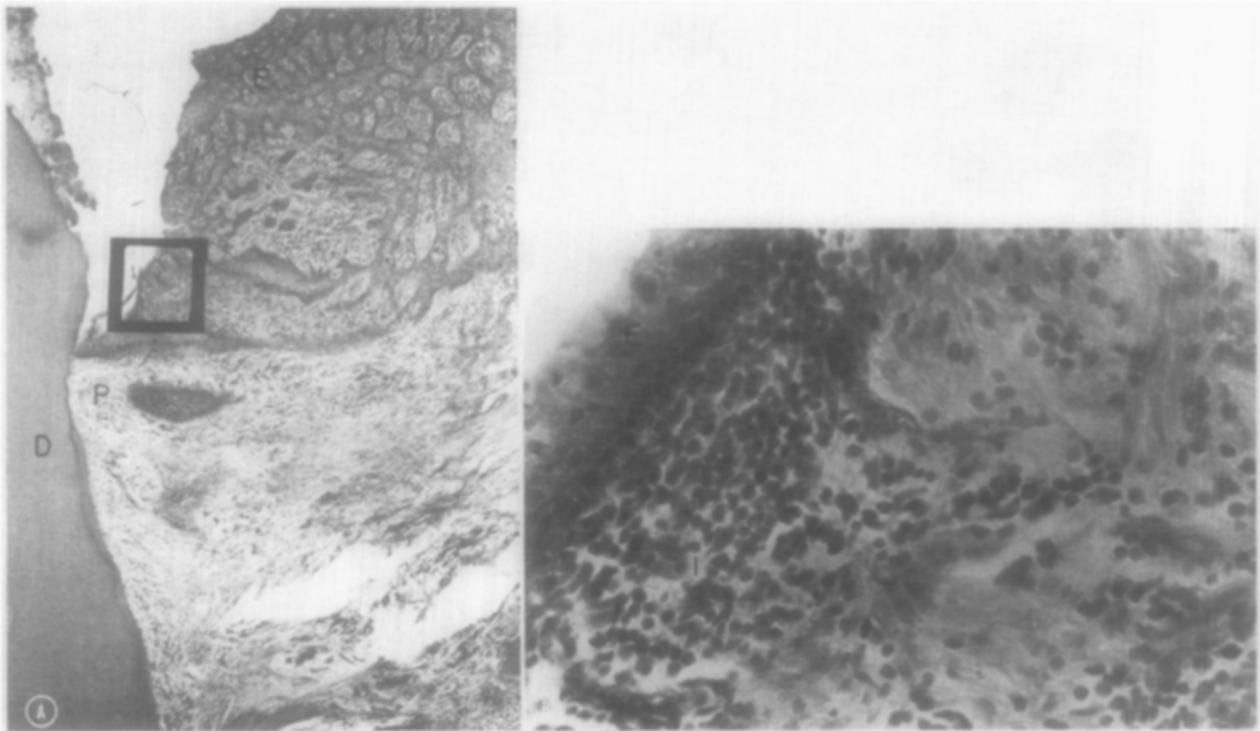
The six dogs were killed at postoperative intervals of 1, 15, 30, 60, 120, and 180 days. At that time the animals were anesthetized, radiographed, and probed periodontally. Tooth mobility, draining sinus tracts, or any obvious soft- or hard-tissue pathoses were noted and recorded. Each animal was killed by injection of 8 to 10 c.c. of Beuthanasia-D.\* The mandibles were isolated, fixed in 10 percent neutral buffered formalin, radiographed, and decalcified. Sections 6 microns thick were cut and stained with hematoxylin and eosin by routine methods.

\*Burns-Biotec Laboratories Division, Chromally Pharmaceutical, Inc., Oakland, Calif.

## RESULTS

Preoperatively, all dogs were examined and found to have intact and caries-free experimental teeth. In all dogs, periodontal examination revealed a chronic gingivitis as evidenced by red and inflamed gingival tissues, gingival bleeding to periodontal probing, and the presence of plaque and calculus, indicative of an incipient periodontitis which was confirmed by later histologic examination. Several of the experimental teeth demonstrated increased pocket depth when examined at the time of death; these were found to correspond histologically to periodontal pockets.

Radiographs taken at the time of death were quite interesting. It was apparent that the technical aspect of sealing perforations with Cavit was difficult in that gross overfillings generally occurred despite the best efforts of the operator. At the 60-day interval and beyond (Fig. 2, *A* and *B*), the formation of a thin radiolucent line was noted around the excess filling material and was interpreted as capsule formation around the Cavit. In a number of specimens, both



**Fig. 4.** A, Unfilled perforation in 180-day animal. Epithelial proliferation and degeneration (*E*) are evident, as well as destruction of periodontal ligament (*P*), with dentin (*D*) adjacent. (Hematoxylin and eosin stain. Original magnification,  $\times 40$ .) B, Higher magnification of rectangle in A. There is a layer of proliferated epithelium (*E*) and chronic inflammatory cells (*I*), mostly lymphocytes, plasma cells, and macrophages. (Hematoxylin and eosin stain. Original magnification,  $\times 250$ .)

filled and unfilled, progressive destruction of the alveolar bone as well as small areas of root resorption could be visualized. In several instances fracture of the Cavit had occurred just beyond the root, leaving a segment of the material embedded in the periodontal tissues.

#### UNFILLED PERFORATIONS

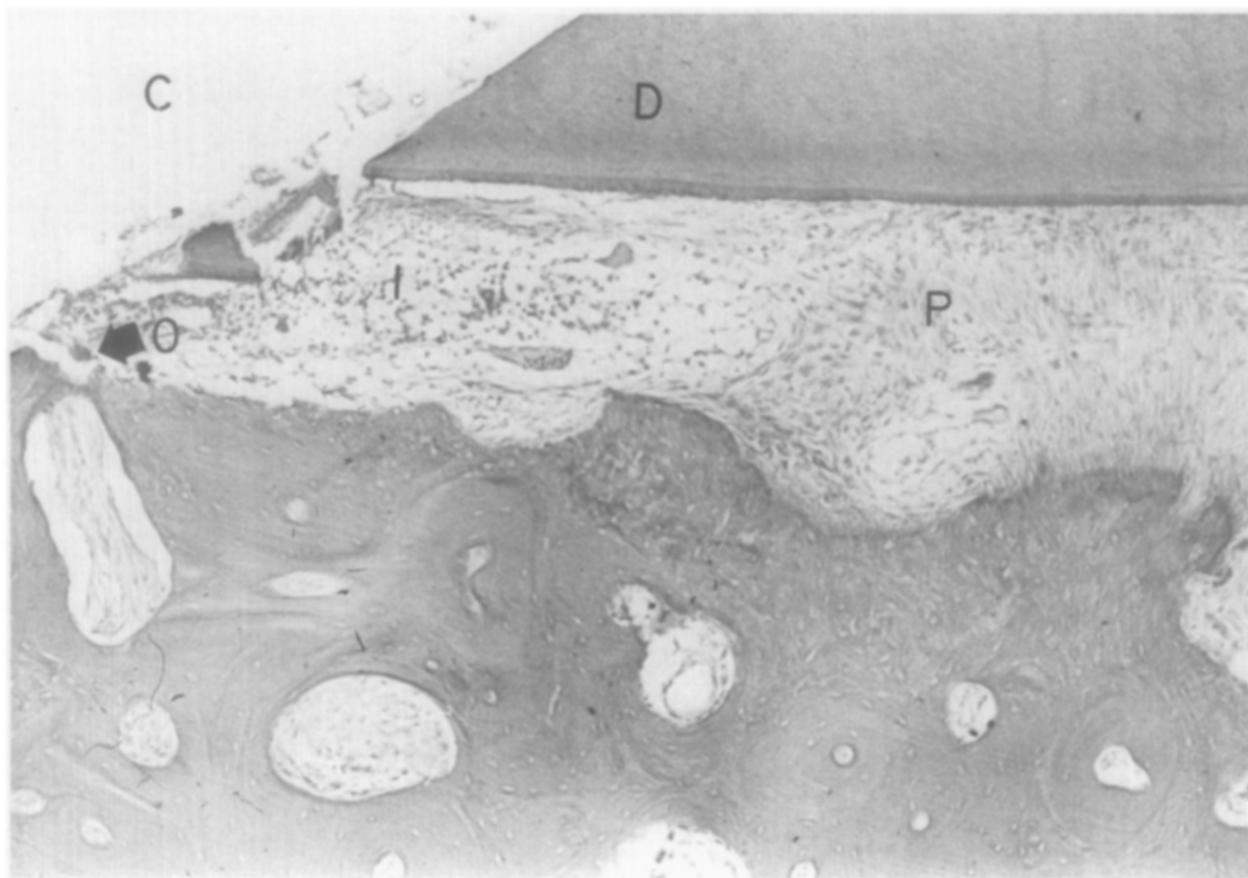
The 1-day animal demonstrated typical acute inflammation, including vasodilation, margination of neutrophilic white blood cells, and histiocytic changes within the adjacent bone marrow spaces. At the site of injury, osteocytes were not seen within the adjacent alveolar bone, and clumps of debris, consisting of hemosiderin, extravasated red blood cells, dentin, and bone particles, were found in the perforating canal. The periodontal ligament was severely disrupted, probably as a result of mechanical trauma from the procedure (Fig. 3).

In the 15-day animal, unfilled perforations located in midroot revealed a dense infiltration of polymorphonuclear leukocytes, macrophages, and some plasma cells with concomitant vasodilation and extravasation of red blood cells when viewed under higher-

power magnification. Particularly noteworthy was the spread of inflammation incisally and apically along the periodontal ligament, which contained a dense collection of acute inflammatory cells. The adjacent alveolar bone stained basophilic, and loss of osteocytes and osteoclastic activity were seen.

In the 30-day animal, unfilled perforations were found to have been placed in the coronal third of the root. An acute inflammatory reaction was seen along with vascular engorgement of blood vessels in the adjacent connective tissue and bone marrow spaces. Incisally, the periodontal ligament appeared to be in various stages of degeneration, whereas disorganization and fibroblastic activity were seen apically. The alveolar bone immediately adjacent to the perforation had resorbed, resulting in vertical bone loss. Root resorption was seen at different points along the root surface, with few signs of repair.

In the 60-day animal, the unfilled perforations were situated in the coronal third of the roots. Inflammation was marked with predominance of polymorphonuclear leukocytes and macrophages adjacent to the defect, with chronic inflammatory cells observed peripherally. There was evidence of



**Fig. 5.** Filled perforation in 15-day animal with advanced inflammatory reaction. Note inflammation (*I*) spreading along the periodontal ligament (*P*), resulting in degenerative changes. Osteoclastic cell (*O*) is seen with dentin (*D*) on opposite side and site of Cavit was (*C*). (Hematoxylin and eosin stain. Original magnification,  $\times 125$ .)

communication between inflammatory processes associated with the gingiva and the perforation with loss of the epithelial attachment. The surrounding alveolar bone had been resorbed and replaced by an irregular loose connective tissue stroma.

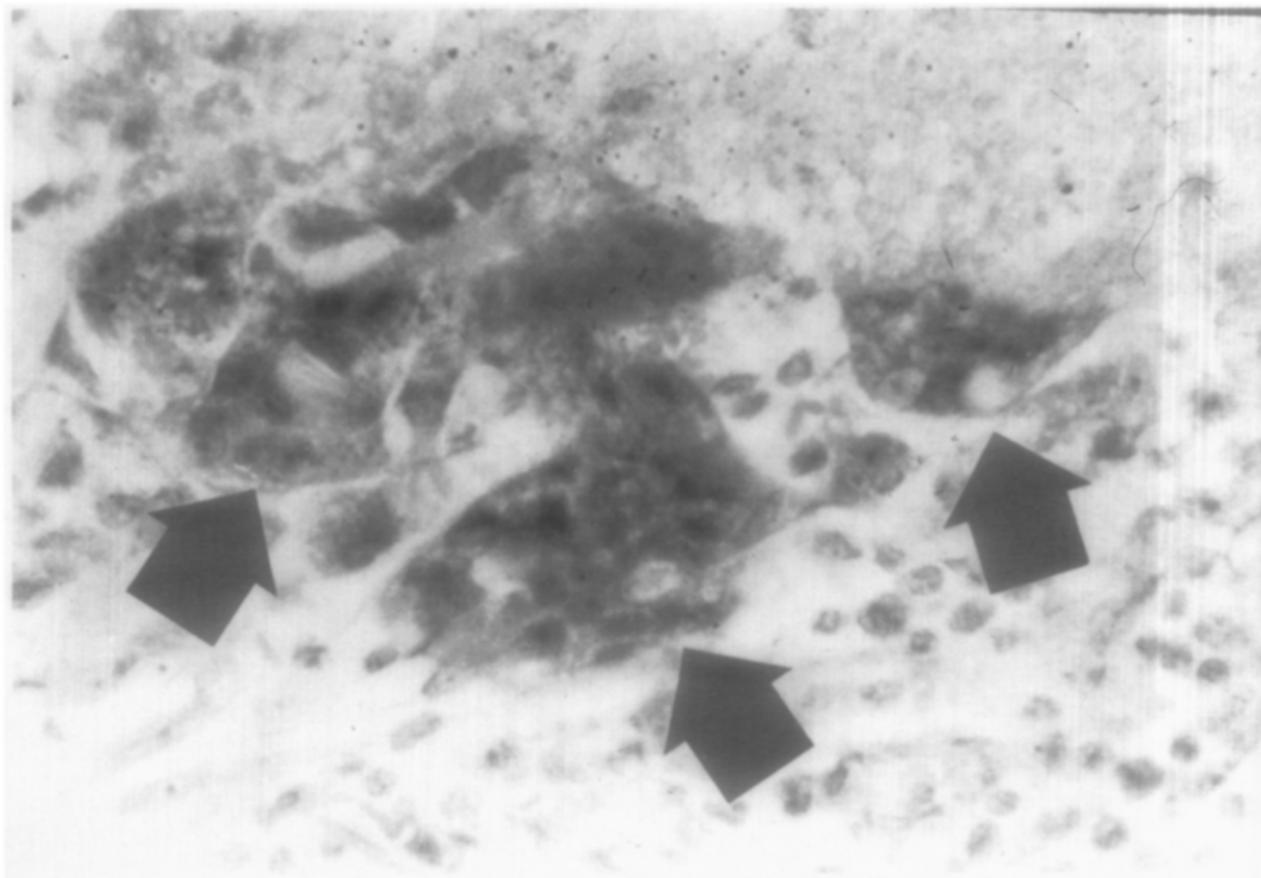
In the 120-day animals, the three unfilled perforations were placed in the coronal third of the roots. A loose connective tissue stroma had replaced the resorbed alveolar bone and had extended into the perforated defects in the roots. Incisally and apically, the periodontal ligament appeared disorganized and evidenced collagen fibers oriented parallel to the root surface.

In the 180-day unfilled perforations, a pattern of severe destruction was seen. Epithelium had proliferated almost to the tooth apex, forming a deep periodontal pocket. Beneath the epithelium was seen an adjacent dense infiltrate of chronic inflammatory cells. A large bony defect was evident, while active bone remodeling was noted apically, and root and cementum resorption continued with little repair

seen. Over-all, the histologic picture was compatible with that of a periodontal abscess (Fig. 4, *A* and *B*).

#### PERFORATIONS FILLED WITH CAVIT

In the 1-day animal, the histologic picture of the filled perforations paralleled that of the unfilled perforations. In the 15-day animal with filled perforations, two somewhat different histologic pictures were discernible. Three well-filled perforations at midroot revealed an inflammatory response consisting of polymorphonuclear leukocytes, macrophages and a few multinucleated giant cells, and a scattering of chronic inflammatory cells peripheral to the immediate perforation site. The periodontal ligament was intact near the Cavit but demonstrated evidence of disorganization and depolymerization of its collagen fibers. Osteocytes were absent from the lacunae of the adjacent bone, which stained a more basophilic color, whereas some osteoclasts were noted along the periodontal ligament.



**Fig. 6.** Filled perforation in 30-day animal. Many multinucleated giant cells (*arrows*) are found adjacent to the Cavit. (Hematoxylin and eosin stain. Original magnification,  $\times 400$ .)

Three additional perforations (two in the coronal third of the root and one in midroot) revealed a more intense inflammatory response. These specimens demonstrated a severe disruption of the periodontal ligament with loss of organization, vasodilation, and hyalinization. The supracrestal gingival fibers appeared intact but manifested early signs of degeneration (Fig. 5). In all instances the oral sulcular epithelium appeared normal, and neither root nor cementum resorption was evident.

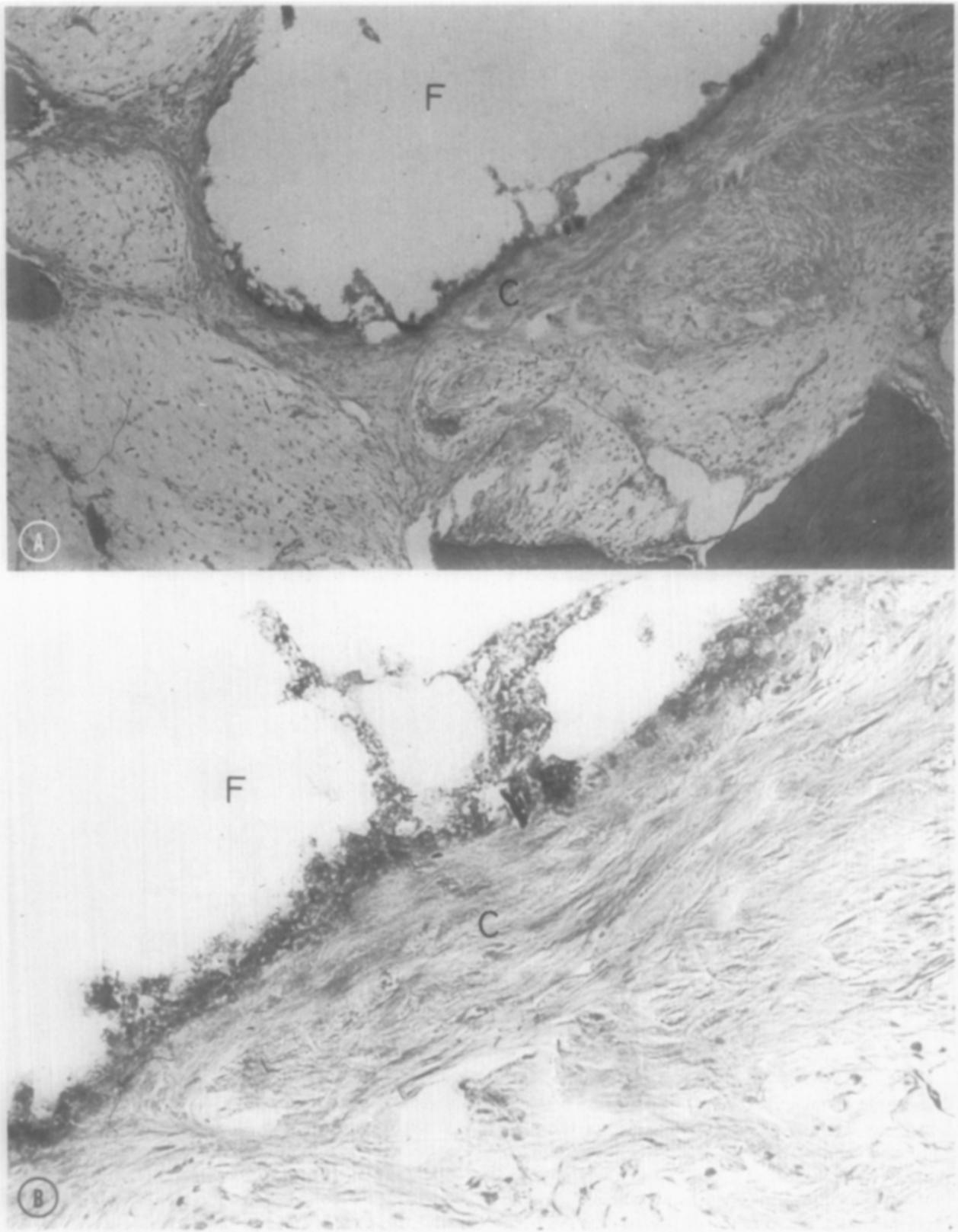
At the 30-day interval, the filled perforations presented a chronic mild to moderate inflammatory response, consisting primarily of plasma cells and macrophages. The alveolar bone surrounding the Cavit had undergone osteoclastic resorption and was replaced by an irregular connective tissue stroma displaying fibroblastic activity and incipient formation of a fibrous capsule. A few multinucleated giant cells were also noted in the capsule surrounding the Cavit (Fig. 6).

The 60-day specimen revealed two histologic patterns. Two filled perforations situated high in the coronal third of the root displayed epithelial prolifer-

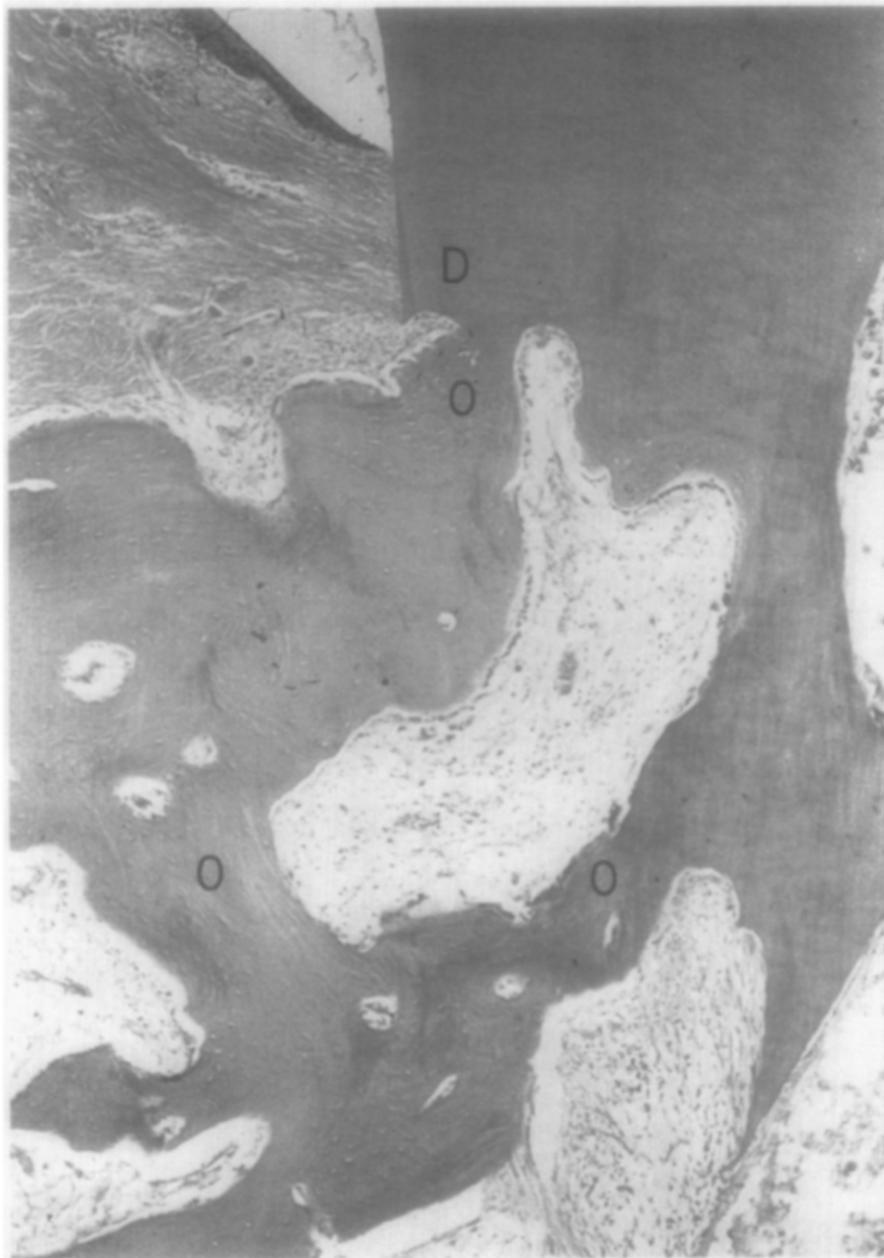
ation beneath the level of the defect, resulting in loss of the alveolar crestal bone and supracrestal gingival fibers. A dense accumulation of chronic inflammatory cells was evident beneath the epithelium, whereas a decreased inflammatory reaction was visible in the nearby loose connective tissue. Although resorption of the surrounding alveolar bone had taken place, osteoblastic activity was now present.

Three filled perforations at this same time interval of 60 days displayed neither epithelial proliferation nor degeneration. Inflammation was chronic, though mild in intensity, with multinucleated giant cells seen as well as a small number of polymorphonuclear leukocytes. Surrounding the Cavit was a loose stroma of collagen fibers and capillaries which exhibited marked fibroblastic activity and a definite fibrous encapsulation of the filling material (Fig. 7, *A* and *B*). Reversal lines indicated new osteoid formation, and osteocytes could be distinguished in the newly formed bone. Regeneration of the periodontal ligament had taken place and collagen fibers were noted to be oriented parallel to the root surface.

The 120-day animals were found to display three



**Fig. 7.** Filled perforation in 60-day animal. **A**, Low-power view of collagen capsule (*C*) surrounding Cavit filling material (*F*). (Hematoxylin and eosin stain. Original magnification,  $\times 40$ .) **B**, Higher-power view of capsule. Note orientation of collagen fibers (*C*) parallel to the Cavit filling material (*F*). Inflammation is minimal. (Hematoxylin and eosin stain. Original magnification,  $\times 160$ .)



**Fig. 8.** Root surface incised to filled perforation in 180-day animal. There is repair of resorbed dentin (*D*) by osteoid deposition (*O*), resulting in areas of ankylosis. Chronic inflammatory tissue is found nearby. (Hematoxylin and eosin stain. Original magnification,  $\times 40$ .)

discernible histologic pictures. Three filled perforations were situated in the coronal third of the root. Epithelial proliferation had occurred and surrounded the Cavit filling material, resulting in a communication between the oral sulcus environment and the experimental site. Another perforation at midroot had been only partially filled with Cavit. The epithelial attachment was intact and showed apical proliferation with a histologic picture of gingivitis. The level of alveolar bone was unchanged, but a very

extensive bone remodeling was evident near the site of perforation. A well-filled perforation at midroot exhibited a low-grade inflammatory response. Enveloping the Cavit was a thick encapsulation of horizontal bands of collagen fibers which were continuous with the periodontal ligament.

In the 180-day animal, one of the filled perforations was found high in the occlusal third of the root, and apical proliferation of the epithelial attachment resulted in a deep periodontal pocket. Fairly intense

chronic inflammation was present in the connective tissue beneath the attachment epithelium, and collagen fibers were seen to surround the zone of inflammation. The four remaining filled perforations revealed repair by fibrous encapsulation, with no changes within the epithelial attachment or oral sulcular epithelium. In the tissue around the perforated region, mild to moderate chronic inflammation was seen and the presence of a few multinucleated giant cells was noted. A dense fibrous encapsulation had occurred around the Cavit, and in some areas the continuity of the collagen capsule was disrupted by an apparent disintegration of the filling material. Root and cementum resorption had stopped, and there was evidence that repair by cementoblastic activity in several areas had resulted in ankylosis (Fig. 8).

## DISCUSSION

Any material that is used in direct contact with living tissue must meet certain standards of tissue compatibility in addition to providing therapeutic or mechanical benefit. Harris reported clinically successful treatment with Cavit but acknowledged the absence of histologic evidence. The present study was undertaken to investigate the histopathologic tissue response to perforations filled with Cavit.

A nonsurgical approach was used in the experimental model and, because of their unfavorable prognosis, perforations were not created in the furcation area. Since most investigators recommend immediate sealing of endodontic perforations, all perforations were repaired at the same appointment in which they were created.

In attempting to pack these defects, it was soon apparent that extrusion of the material was difficult to avoid when condensing to gain an adequate seal. Thus, large overfillings resulted, just as they might when one is attempting to seal a clinical case. Obviously, the physical effect of the overfilling added to the over-all reaction of the material itself. Thus, although it was surprising that some of the unfilled perforations gave the best healing seen in the study, careful analysis showed the logic of that result.

When unfilled perforations were located in the coronal third of the root near the oral sulcular epithelium, progressive destruction of the surrounding periodontal structures resulted. In these cases, the most striking observation was the spread of inflammation from the perforation incisally along the periodontal ligament space which resulted in destruction of the ligament, alveolar bone, and supracrestal gingival fibers. This inflammation was seen to have an adverse effect on the overlying oral

sulcular epithelium, as evidenced by apical migration of the epithelial attachment with alveolar bone loss. There was an attempt by the underlying connective tissue to accomplish encapsulation, but the final result was the formation of an advanced periodontal defect with widespread inflammation and abscess development.

When the unfilled perforations were located in midroot, however, a definite healing response was noted. In these cases, the overlying bone and periodontal ligament may have presented an adequate barrier to the destructive inflammatory reaction following the perforation, so that the integrity of the periodontium was apparently maintained while the inflammation subsided, allowing for repair attempt by fibrosis. Whether or not repair would be permanent is uncertain and may be an area for future investigations. Clinically, the findings do suggest a more favorable prognosis for those perforations situated apical to the gingival sulcus and well within alveolar bone.

The filled perforations were observed to heal by fibrous connective tissue encapsulation which could be characterized as scar tissue formation. Ultimately, a stroma organized to form a fibrous capsule characterized by connective tissue organized parallel to the surface of the filling material. After severe disruption and disorganization, the periodontal ligament was observed within 30 days to regenerate with the formation of collagenous fibers oriented parallel to the root surface and continuous with the collagen fibers of the capsule surrounding the Cavit. Root and cementum resorptions were observed to be repaired by secondary cementum deposition and, in some cases, ankylosis. Ankylosis is observed frequently following severe irritation or injury to the periodontal ligament, such as that occurring with a root perforation or tooth replantation. Such damage may effectively inhibit regeneration of the periodontal ligament and allow for repair by fusion of the tooth root with alveolar bone. Such healing is not considered to be highly desirable.

At the 120- and 180-day examinations, five of the filled perforations in the coronal third of the root were surrounded by an epithelial capsule extending from the oral sulcular epithelium to below the defect. The close proximities of the perforations to the gingival sulcus appear to be a major factor in the lack of repair and the formation of a periodontal lesion. This contention was supported by the finding that a more severe tissue response was observed following both filled and unfilled perforations in the coronal third of the root. From an endodontic viewpoint, these findings indicate a more promising prognosis for those perforations located in the middle

and apical thirds of the root. It would appear that an adequate amount of connective and osseous tissue incisal to the defect may well allow the preservation of the gingival fibers and an opportunity for regeneration of the damaged periodontium.

Radiographically, at the 60-day interval and beyond, instances in which the Cavit overfilling had fractured just outside the perforation canal in the tooth were seen. This resulted in segments of the material being implanted in the periodontium. It appears that the brittle nature of the material after setting may have allowed for fracture during the masticatory movements. In addition, since occlusal reduction was performed, passive eruption of the teeth may have further contributed to the fractures observed, but the actual effect is somewhat uncertain. The physical presence of the foreign body in the periodontal tissues could act as a continuous source of irritation and inflammation. Clinically, however, such occurrences are fairly common, particularly in the cases of canal overfillings with gutta-percha which are not found to affect adversely the prognosis of the treated tooth as long as the apical portion of the canal is sealed. From purely an endodontic viewpoint, the separation of Cavit may be regarded as less than optimal but unlikely to affect the repair process negatively as long as a seal of the perforation is maintained.

In the present study, there were considerable variations as to size, depth, and proximity of the perforations to the oral sulcular epithelium. Also, many of the perforations were not consistent with those encountered in clinical cases. Although the histologic findings were informative, future studies should consider methods by which consistent perforation defects similar in size, depth, and location to those encountered clinically could be created.

Criticism may be directed toward the fact that a comparison was not made with another filling material, most notably gutta-percha. Numerous previous studies, however, have examined extensively the tissue compatibility and inflammatory potential of gutta-percha. Furthermore, the classic studies of Lantz and Persson,<sup>12-14</sup> discussed at length the tissue reactions to perforations in dogs' teeth filled with gutta-percha. Nevertheless, it is acknowledged that the present investigation might have been more valid had a comparison of the tissue reactions to both Cavit and gutta-percha been made.

#### SUMMARY AND CONCLUSIONS

A study was undertaken to investigate the periodontal tissue reaction to perforations in dogs' teeth filled with Cavit. Thirty-six lower premolar teeth in

six beagles were treated endodontically and canals were filled with gutta-percha and sealer. Immediately thereafter, thirty-one experimental perforations were created and filled with Cavit. An additional seventeen perforations were created and left unfilled to serve as controls. The animals were killed at intervals of 1, 15, 30, 60, 120, and 180 days. The jaw sections were removed at necropsy and histologic sections were prepared with hematoxylin and eosin stain. The sections were studied microscopically and the observations were recorded.

Within the scope of this study, the following conclusions were made:

1. Progressive destruction of the periodontal tissues took place in those perforations located near the oral sulcus.
2. When Cavit was used immediately to seal the noncoronal perforations, fibrous encapsulation occurred.
3. Any perforation, filled or unfilled, when placed close to the gingival sulcus, could result in proliferation of oral sulcular epithelium and negate any possibility for repair to occur.
4. Perforations located away from the oral sulcular epithelium displayed the most favorable healing response, even when left unfilled.
5. Root and cementum resorption always occurred with perforations and was repaired only in successfully filled perforations.

Within the scope of this study, the following impressions were gained:

1. The use of a filling material with a low inflammatory potential is definitely indicated in light of the frequently observed overfilling during the attempt to repair perforations.
2. However, Cavit appears to possess a mild to moderate inflammatory potential. Further studies are required to clarify this property as well as to determine solubility of the material under conditions similar to those encountered in the periodontium.
3. Varied experimental conditions similar to those encountered clinically should also be evaluated. These might include perforations contaminated by the oral environment, time lapse between the perforation and the attempt at repair, and small accessible perforations of the furcation area.

#### REFERENCES

1. Ingle, J. I.: A Standardized Endodontic Technique Utilizing Newly Designed Instruments and Filling Materials, *ORAL SURG* **14**: 83, 1961.
2. Seltzer, S., Bender, I. B., Smith, J., Freedman, I., and Nazimov, H.: Endodontic Failures; an Analysis Based on Clinical, Roentgenographic, and Histologic Findings, *ORAL SURG* **23**: 500, 1967.

3. Grossman, L. I.: The Management of Accidents Encountered in Endodontic Practice, *Dent. Clin. North Am.* **11**: 903, 1957.
4. Lange, V. G.: Artifielle perforation des Wurzelkanales und deren behandlung, *Dtsch. Zahnärztl. Z.* **6**: 299, 1958.
5. Weisman, M. I.: Treatment of an Unusual Perforation of an Anterior Tooth, *ORAL SURG.* **12**: 732, 1959.
6. Guilford, S. H.: Root Perforation, *Dent. Office & Laboratory* **20**: 122, 1901.
7. Weine, F. S.: Endodontic Therapy, ed. 2, St. Louis, 1976, The C.V. Mosby Company, chap. 7.
8. Bence, R.: Handbook of Clinical Endodontics, St. Louis, 1976, The C.V. Mosby Company.
9. Sinai, I.: Endodontic Perforations: Their Prognosis and Treatment, *J. Am. Dent. Assoc.* **95**: 90, 1977.
10. Stromberg, T., Hasselgren, G., and Bergstedt, H.: Endodontic Treatment of Traumatic Root Perforations in Man, *Sven. Tandlak. Tidskr.* **65**: 457, 1972.
11. Frank, A. L.: Resorption, Perforations and Fractures, *Dent. Clin. North Am.* **18**: 465, 1974.
12. Lantz, B., and Persson, P.: Experimental Root Perforations in Dogs' Teeth; a Roentgen Study, *Odontol. Revy* **16**: 238, 1965.
13. Lantz, B., and Persson, P.: Periodontal Tissue Reactions After Root Perforations in Dogs' Teeth, *Odontol T* **75**: 209, 1967.
14. Lantz, B., and Persson, P.: Periodontal Tissue Reactions After Surgical Treatment of Root Perforations in Dogs' Teeth: A histologic Study, *Odontol Revy* **21**: 51, 1970.
15. Schwartz, S. F.: Treated Perforations of the Pulp Chamber Floor: Histopathologic and Technical Study, Master's Thesis, The University of Texas Dental Branch, Houston, June, 1970.
16. Seltzer, S., Sinai, I., and August, D.: Periodontal Effects of Root Perforations Before and During Endodontic Procedures, *J. Dent. Res.* **49**: 332, 1970.
17. Bhaskar, S. N., and Rappaport, H. M.: Histologic Evaluation of Endodontic Procedures in Dogs, *ORAL SURG.* **31**: 526, 1971.
18. Schilder, H.: Canal Debridement and Disinfection. *In* Cohen, S., and Burns, R.C. (editors): Pathways of the Pulp, 1976, The St. Louis, C.V. Mosby Company, p. 132.
19. Grossman, L.I.: Endodontic Practice, ed. 7, Philadelphia, 1974, Lea & Febiger, pp. 205-207.
20. Nicholls, E.: Treatment of Traumatic Perforations of the Pulp Cavity, *ORAL SURG.* **15**: 603, 1962.
21. Taatz, A., and Stiefel, A.: Treatment of Perforations of Teeth, *Zahnärztl. Welt.* **66**: 814, 1965.
22. Harris, W. E., and Davis, J. E.: Repair of a Perforation in the Bifurcation of a Mandibular Molar, *J. Georgia Dent. Assoc.* **49**: 2, 1975.
23. Harris, W. E.: A simplified Method of Treatment for Endodontic Perforations, *J. Endod.* **2**: 126, 1976.
24. Marosky, J. E., Patterson, S. S., and Swartz, M.: Marginal Leakage of Temporary Sealing Materials Used Between Endodontic Appointments and Assessed by Calcium-45—An In Vitro Study, *J. Endod* **3**: 110, 1977.

*Reprint requests to:*

Dr. Franklin S. Weine  
Loyola University School of Dentistry  
2160 South First Ave.  
Maywood, Ill. 60153