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A Histologic Evaluation of Periodontal Tissues Adjacent to Root Perforations Filled With Cavities

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A HISTOLOGIC EVALUATION OF PERIODONTAL TISSUES ADJACENT
TO ROOT PERFORATIONS FILLED WITH CAVIT

By

Ronald C.K. Jew, B.A., D.D.S.

A Thesis submitted to the Faculty of the Graduate School of
Loyola University of Chicago in Partial Fulfillment
of the Requirements for the
Degree of Master of Science

[May 1979]

DEDICATION

To my parents, James and May-Lin, whose unending faith and many sacrifices have made possible so many dreams in my life.

To my wife, Karen, whose love and support have allowed me to reach beyond that only wished for by other men.

ACKNOWLEDGMENTS

To Doctor Franklin Weine whose dedication and contribution to endodontics is unsurpassed by none and to whom I pledge my deepest respect and unbounded gratitude.

Doctor Marshall Smulson, your inspiration and unselfishness in the field of education is without equal and to whom I express my most sincere appreciation.

To Doctor Joseph Keene, my friend and advisor, I thank you for your help and long hours in the evaluation of the results of this paper.

Special thanks to Doctor James E. McCormick and Doctor Richard Munaretto for their gracious assistance and valuable insight which have made this study a reality.

VITA

The author, Ronald C.K. Jew, is the son of James and May-Lin Jew. He was born September 28, 1949 in Washington, District of Columbia.

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In September, 1971, he entered the University of California School of Dentistry at San Francisco where he graduated with a Bachelor of Arts degree in Dental Science and the degree of Doctor of Dental Surgery in June of 1975.

After receiving a D.D.S. degree, he was in private practice in Stockton, California, from August, 1975 to June, 1977.

In September of 1977 he entered the Loyola University School of Dentistry and pursued the degree of Master of Science in Oral Biology and a certificate of Specialty Training in Endodontics.

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CHAPTER I

INTRODUCTION

The art and science of endodontics has shown remarkable advancement since the days of the theory of focal infection when the pulpless tooth was unjustly condemned for its role in a variety of systemic diseases. Increased understanding of the pathological and repair processes of the pulp and periapical tissues have disposed the misconceptions of the past and provided endodontics with a biologically sound foundation. Numerous anatomical studies have added significantly to our perception of the morphology of the root canal system. Standardization combined with metallurgically superior materials has provided improved instruments for canal debridement and preparation.

Endodontic therapy now enjoys the highest rate of success of any single phase of dentistry. It has gained almost universal acceptance by patients and dentists alike as a preferable and predictable alternative to extraction.

Yet this rise in popularity has also been accompanied by an increase in the degree and complexity of procedures encountered in endodontics. For example, the diagnosis of a combined endodontic and periodontal lesion is frequently difficult and may necessitate alterations in the prognosis and treatment modality. The retreat-

ment of failing endodontic cases often involves a very formidable and laborious task. The location of canals hidden beneath a large restoration may frustrate even the most experienced operator. In addition, greater life expectancy and better dental procedures have allowed preservation of increasing numbers of teeth in our population. Many of these retained members of the arch, however, demonstrate decreased canal size, making location and/or enlargement difficult.

For these reasons, perforations of the root or pulp chamber continue to be among the most common problems associated with endodontic procedures. There is, however, uncertainty as to the optimal treatment modality for perforations. Surgical approaches have been suggested, including reverse filling of the defect, apicoectomy and root resection. Nonsurgical approaches have also been introduced such as the sealing of the defect with gutta-percha, amalgam or zinc oxide-eugenol.

The purpose of this study is to investigate the histologic response of the periodontium to non-surgical repair of endodontic perforations sealed with Cavit, a temporary filling material.

CHAPTER II

REVIEW OF THE LITERATURE

THE PERIODONTIUM

The understanding and interpretation of the pathologic processes resulting from root perforations require a review of the histology, functions and repair capacity within the clinically healthy periodontium. Emphasis is directed primarily towards those histologic structures at or adjacent to the site of injury within this study, namely the periodontal ligament, alveolar process, cementum, and gingival connective tissues.

The functions of the periodontal ligament are: 1) secure the tooth in its socket, 2) provide a source of undifferentiated cells in order to support growth and repair of the alveolar bone, periodontal ligament and cementum, and 3) supply sensory proprioception and, 4) nutrition². The periodontal ligament consists primarily of collagen fibers which have been classified histologically as principal and secondary fiber bundles. The principal fiber bundles traverse the periodontal space inserting obliquely in cementum and bone where they are termed Sharpey's fibers. Secondary fiber bundles are comprised of randomly oriented collagen fibers located between the principal fiber bundles³. The collagen fibers of the periodontal ligament are characterized by an elevated rate of metabolic activity as well as a rapid turnover. The latter is in contrast

to the collagen of other tissues⁴. Kraw and Enlow⁵ stated that a continuous and rapid synthesis of collagen fibers occur to accommodate fiber reorientation in order that the tooth may adapt to the demands of eruption, growth, occlusal stress, and other functional factors.

The collagen fiber bundles are contained within a mucopolysaccharide ground substance in which are also found fibroblasts, osteoblasts, osteoclasts, cementoblasts and cementoclasts. These cells are involved in the formation and destruction of the periodontal ligament, cementum and alveolar bone. Defense cells of the periodontal ligament include histiocytes, undifferentiated mesenchymal cells and lymphoid cells. Also found are epithelial rests of Malassez which may proliferate under the stimulus of chronic inflammation⁶.

The alveolar process supports the tooth socket and consists of two parts, the alveolar bone proper and supporting alveolar bone. The alveolar bone proper is a thin, hard, compact lamella of bone to which Sharpey's fibers of the periodontal ligament are inserted. This bone is often of a coarse, fibrous nature and has been referred to as bundle bone. In radiographs, the alveolar bone proper is described by the term lamina dura⁷. Microscopically, the alveolar bone proper is perforated by numerous minute foramina (Volkman's canals) through which pass blood vessels, lymphatics and nerves. Accordingly, through these lateral perforations, the periodontal ligament receives its major blood supply from interalveolar vessels in the supporting bone which are derived from the interdental artery⁸.

The supporting alveolar bone surrounds the alveolar bone proper and consists of two portions: the compact cortical plates of the vestibular and oral surfaces, and the trabecular or spongy bone between the cortical plates and the alveolar bone proper⁹.

The most essential characteristic of alveolar bone is its capacity to undergo continuous remodeling in response to functional demands. The osteoblasts and progenitor cells of the periosteum, endosteum and periodontal membrane serve to allow continuous modification of the alveolar bone due to physiologic or pathologic changes¹⁰. However, during the process of inflammation, bone resorption is commonly found to be accentuated in contrast to bone deposition. Endotoxins, prostaglandins and other factors such as generated by activation of the immune system and complement cascade, have been postulated to account for enhancement of bone resorption during inflammation³. Seltzer² noted that inflammation produces local tissue acidity which interferes with the critical concentration of calcium and phosphorus needed for precipitation into the organic matrix of bone.

Cementum is a highly specialized form of calcified tissue that resembles bone structurally, but lacks innervation, a direct blood supply or lymph drainage¹¹. It is deposited first as primary cementum which is acellular and continues its formation by the deposition of successive layers of secondary cementum which may be acellular or cellular depending on the presence of cementocytes¹². Cementum functions primarily through the attachment of the periodon-

tal ligament fibers whose ends become embedded at right angles to the root surface as Sharpey's fibers. The most dynamic feature of the cementum is its continuous deposition throughout life, thereby compensating for tooth movement and permitting the repair of root resorptions resulting from either physiologic or disease processes⁹. Continuous cementum deposition may further protect the calcified cementum from resorption by the adjacent connective tissue and provide continuous new surfaces for aging cementum which would otherwise become nonvital².

Kerr¹³, and Erausquin and Muruzabal¹⁴ showed that resorption of cementum and dentin frequently occurred with necrosis of the periodontal ligament caused by trauma or inflammation. Where periodontal ligament repair took place, secondary cementum deposition was found to fill in the resorbed dentin and cementum defects.

The connective tissue of the gingiva coronal to the alveolar crest consists primarily of cells, fibers, blood vessels, and sensory nerves embedded in an amorphous mucopolysaccharide ground substance¹⁵. The collagen fiber bundles of the gingiva, have been described on the basis of their location, origin, and insertion as the dentogingival, dentoperiosteal, alveologingival, circular, and transseptal fiber bundles³. Hurt¹² noted that the transseptal and dentoperiosteal fiber bundles were particularly important in preventing the progression of gingival inflammation apically.

Stone and Kalis¹⁶ stated that virtually all clinically normal gingival connective tissue contains cells of the plasma and lympho-

cytic series as well as small numbers of polymorphonuclear leukocytes. These inflammatory cells were found to represent a constant chronic inflammatory response to the ever present irritation of bacteria and bacterial products within the gingival sulcus.

PERFORATIONS

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 of this perforation, the retention of an involved tooth may be compromised. In an analytical study of endodontic failures in 1961, Ingle¹⁷ found that root perforations were the second greatest cause for failure and accounted for 9.62% of the unsuccessful cases, while improper obturation of the root canal space was the leading cause attributing for 58.65% of the endodontic failures. Seltzer, et al.¹⁸, in 1967 attributed 3.52% of endodontic failures to perforations and added that such accidents often occurred without the knowledge of the operator. (Figure 1)

Grossman¹⁹ in 1957 ascribed iatrogenic perforations of the pulp chamber to the introduction of high speed cutting instruments coupled with the individual variations of pulp chamber size and tooth alignment. Numerous authors have stressed that since endodontic procedures are carried out in a very limited and confined area, the lack of a thorough knowledge of dental anatomy and topographical relationships may lead to procedural accidents^{20,21,22,23}. Inadequate access preparation may also result in the misdirection of a bur while gaining

entrance to the pulp chamber or canal orifices which could inadvertently result in a perforation in a matter of seconds^{21,23,24,25,26}. To consider the occlusal relationship and neglect the axial inclination of the tooth was observed to be another frequent cause of perforations^{1, 27}. Additional complications arise when the crown-root relation has been altered by an extracoronary restoration which may mask the orientation of the roots and radiographically hide the location and size of the pulp chamber^{1,23,28}. Furthermore, poor quality or improperly developed radiographs often hinder the operator's ability and judgment to visualize the correct root canal morphology^{1,21,23,26}.

Bence²⁹ and Weine¹ explained that perforations near the apex of a root are most often the result of improper root canal instrumentation. Bence listed the causes for problems as: 1) Use of a reaming action in a curved canal; 2) Attempting to enlarge the apical portion of a narrow or curved canal beyond a flexible file size; 3) Failure to precurve files; 4) Failure to flare the canal; 5) Overuse of a chelating agent; 6) Use of engine driven reamers in the canal; and 7) Failure to use files in sequential order of size. Weine, Kelly and Bray³⁰ in 1976 further stated that the length, curvature, and small diameter of certain canals can make the procedure of canal debridement quite tedious and time consuming. They cautioned that hasty treatment in such difficult cases may lend themselves to procedural accidents such as iatrogenic perforations.

Nicholls^{31,32} and Luks²³ stated that the use of excessive pressure with a hand instrument may result in a perforation by forcing the instrument through the side of the root. Frank³³ noted that "stripping" of a root may occur as a result of overfiling and over-instrumentation. In this manner the lateral wall of the root canal is mechanically removed which results in a perforation. Luebke and Dow³⁴ in 1964 warned that attempts to operate through a small and inadequate occlusal opening may lead to root perforation particularly if the root canal is curved and large instruments are used with force. He further added that an obstruction or an accessory canal may intercept the end of a root canal instrument and guide it into the periodontal tissues. Root perforations have also been reported to occur during the removal of root filling material in preparation of post room (Figure 2) or in retreatment of an inadequate root canal filling 25,28,31,34,35,36,37.

The misuse of engine driven instruments for partial or total canal instrumentation has also been indicted by a number of authors as a cause for root perforations^{20,26,30,31,32}. This was found to occur most frequently in curved canals where inflexibility of the instrument often resulted in a teardrop shaped perforation of the root apex or in a penetration through the lateral wall of the root. Weine³⁸ in 1976 showed that the use of the fistulator as recommended by Sargenti³⁹ for the trephination of the cortical bone, often resulted in perforation of the root or roots of the involved tooth as

well as adjacent teeth. Poor directional control of an instrument, inadequate knowledge of root canal morphology, and failure to utilize straight and angled radiographs have been discussed as additional factors conducive to the creation of root perforations^{1,23,36}.

Tooth resorption, when allowed to continue for an adequate length of time, can produce a communication between the pulp canal and the periodontal structures⁴⁰. Internal resorption has been described as a physiologic or pathologic process originating in the pulpal cavity which results in loss of dentin of the tooth root⁴¹. Although trauma^{36,42,43,44}, calcium hydroxide pulpotomy⁴⁵, and pulpal infections from carious exposure⁴¹ have been suggested as possible causes, the etiology of internal resorption is uncertain. Chivian⁴⁶ noted that this process is usually asymptomatic and may swiftly destroy the tooth in untreated cases. External root resorption leading to perforation has most often been attributed to trauma whereby crushing of the periodontal ligament and possibly pulpal death induce the inflammatory reactions culminating in odontoclastic activity^{41,44}. Inflammatory tissue in periodontal pockets has also been suggested as another mechanism of external resorption⁴⁷.

Perforations may also occur as a result of carious invasion of the floor of the pulp chamber and extension through to the furcation⁴⁰.

CLASSIFICATION

Stromberg, Hasselgren, and Bergstedt⁴⁸ in 1972 presented a

followup study of 24 cases of traumatic perforations. They attempted to determine if there was a relationship between the location of the perforation and the final diagnosis. They subsequently divided perforations into four groups dependent on the different locations of the defect. These were: 1) the coronal portion of the root under the marginal bone level; 2) the furcation area and 2mm apically; 3) the middle portion of the roots; and 4) the apical portion of the root (Figure 3).

CLINICAL AND DIAGNOSTIC MANIFESTATIONS

The most common sign that a perforation has occurred will be profuse bleeding as a result of damage to the periodontal structures^{21,42,49,50,51,52}. This was noted to be particularly true when obvious bleeding occurs from a pulpless tooth²⁸. Pronounced pain may also be present^{35,37,53}, but the differential diagnosis between perforation and pulpal remnants may at times be difficult²⁰.

Morse²⁸ has stated that persistent pain, the presence of a sinus tract or the appearance of localized problems such as pocket formation or furcation involvement following apparently adequate endodontic therapy may indicate the existence of a perforation. Another clue of a possible perforation occurs when a file or reamer is placed into an opening and the instrument appears to be loose rather than snug as would be expected in a true canal. Leeb⁵² noted that when a perforation has been left untreated by the operator, the continued presence of a serohemorrhagic exudate will often be seen. Bence²⁹

observed that the involved tooth was frequently tender to percussion while Taatz and Stiefel⁵³ added that chronic inflammation of the gingiva will occur if the perforation penetrates the alveolar bone.

Definitive diagnosis of a perforation in the pulp chamber or root canal is best determined by radiographic examination with root canal instruments or contrast media placed in the suspected defect and the canals of the tooth^{31,34} (Figure 4). The deviation of an instrument from the long axis of a canal was noted to provide definite proof of a perforation (Figure 5). Furthermore, the use of radiographs taken from different angles was suggested as an aid to demonstrate the actual location of the instrument in a tooth²⁹ (Figure 6).

PROGNOSIS

The prognosis for a tooth with a perforation has been found to be dependent on the time lapse between occurrence and repair of the perforation, location of the perforation, adequacy of the perforation seal, sterility of the perforation, accessibility of the main canal, and its size.

It has been stated by numerous investigators^{20,40,48,54,55,56,57,58,59,60,61,62} that upon first encountering a perforation, one will obtain the most favorable prognosis by sealing the defect immediately. They observed that failure to immediately seal the defect allowed rapid periodontal breakdown which greatly complicated repair when attempted at a later date due to the decreased likeli-

hood of regeneration of the periodontal fibers and alveolar bone. Furthermore, one may have lost the matrix against which a filling material may be condensed^{31,35,36}.

Many authors^{27,31,48,50,53,56,57,58,59,60,61,62} have reported that the location of the perforation site relative to the gingival sulcus has a definite bearing on the healing potential. They found that perforations located in the furcation or coronal one-third of the root have a doubtful prognosis due to proximity to the gingival sulcus. Sinai⁴⁰ and Frank³³ explained that perforations in these areas often present as serious problems in that the ensuing inflammation may rapidly produce a communication with the oral environment and result in noncorrectable periodontal lesions. Lantz and Persson^{57,58} observed that perforations in the apical and middle one-third of the root offered a more favorable prognosis provided an immediate seal was obtained. Frank⁵⁶ further added that perforations located entirely within alveolar bone are more predictable, since an adequate bridge of bone may preclude the possibility of communication of the repair process with the contaminating influence of the oral environment.

Most investigators have stated that unless a permanent seal of the perforation is obtained, the final outcome will involve extensive destruction of the root and the surrounding periodontium which will necessitate extraction or root resection^{28,31,32,33,40,48,53,56,57,58,59,61,62,63,64}. Schwartz⁶⁰ in 1970 indicated that the

manner of sealing the perforation is unimportant provided a seal is obtained and the filling material is compatible with and allows for periodontal healing. When an adequate seal cannot be obtained because of extensive size or inaccessibility of the defect, the prognosis becomes guarded⁶⁵.

Several reports^{20,53, 54,57,58,62} have stated that the prognosis is considerably worsened for perforation sites which have become infected. These studies have indicated that the time period a perforation has been open to contamination is a major factor contributing to the degree of inflammatory change and periodontal breakdown. Seltzer, Sinai, and August⁶¹ in 1970 and Bhaskar⁶² in 1971 observed that leaving a perforation exposed to saliva and microorganisms for long periods of time effectively destroyed the periodontium adjacent the defect by stimulating the apical migration of the epithelium attachment with subsequent epithelial proliferation.

Sinai⁴⁰, Nicholls³², and Frank³³ indicated that accessibility to the main canal is another factor affecting the prognosis of a perforation. They observed that endodontic perforations frequently occur while attempting to locate a canal orifice or during the instrumentation and negotiation of a curved or constricted canal. Following immediate sealing of the perforation, the true canal must be found, instrumented, and filled. However, in a number of cases the true canal may be impossible to locate and therefore not accessible to endodontic treatment. They cautioned that failure at this stage will decrease the chances of success for healing of the perio-

dontium and the involved root.

It has been stated by several authors^{31,32,33,60} that small perforation sites are more favorably disposed to direct and immediate sealing with a decreased likelihood of periodontal breakdown. They observed that as the size of the defect increases, so does the potential for an overfilling and inadequate seal.

MANAGEMENT OF PERFORATIONS

The literature reveals that the concern and interest in the management of perforations is not of current vintage. As early as 1901, Guilford²¹ suggested that perforations of the pulp chamber be filled with a piece of moistened plaster of Paris which was locked in place with a zinc-phosphate cement. He added that perforations occurring beyond midroot were best treated by root resection at the level of the defect, while a root perforation of a multirooted tooth could be considered a candidate for hemisection or root amputation. Peeso⁶⁶ in 1903 recommended the use of copper amalgam where the perforation was accessible. Perforations of the pulp chamber were first covered with a piece of thin, soft platinum or tin sheet which was sealed in place with the root canal filling material.

A report by Head⁶⁷ in 1904 described the methods of root perforation repair used at that time. Gutta-percha, platinum sheet, and lead discs utilized with various cements were observed as being feasible sealing materials. There was agreement among the practitioners that the removal of granulation tissue, hemorrhage control,

and avoidance of overfilling were necessary for the most favorable prognosis. Brown⁶⁸ in 1905 stressed that the mechanical handling and treatment of the perforation were important to success rather than the materials or medicinal agents used. He noted that perforations through or near the apex were to be treated in the same manner utilized to fill the root canal in hope that the perforation would also be sealed. For accessible perforations he advised the use of gutta-percha sealed in place with chloro-percha. Brown was also one of the earliest authors to suggest the surgical exposure of inaccessible perforations and their repair with either amalgam or gutta-percha.

Hirschfield⁶⁹ in 1912, observing a favorable tissue response to porcelain, proposed that perforations of the pulpal floor be filled and sealed with an appropriate size plug of porcelain. He also emphasized the use of a rubber dam to maintain asepsis during the repair of a perforation. Hicks⁷⁰ in 1913 and Spaulding⁷¹ in 1914 advocated the use of lead in the form of points or wheels as a material for the repair of perforations. Burchard and Inglis⁷² in 1915 described a treatment for perforations of the pulp chamber whereby the exposed tissues were first treated with an antiseptic varnish and the perforation sealed with copper amalgam.

The literature review will now concern itself with a discussion of the management of perforations more currently in use. A classification scheme as suggested by Sinai⁴⁰ will be utilized with two additional categories added.

1. Perforations Sealed During Routine Endodontic Treatment.

Numerous investigators have suggested that when possible, the perforation may be sealed conservatively by a conventional endodontic technique^{19,20,26,28,31,48,63,73,74}. Frank³³ advised that strip perforations occurring during root canal instrumentation must be filled immediately together with the true canal using a root canal filling material, preferably gutta-percha and sealer. Clark⁷⁵ and Nicholls³² observed that perforations in the apical one-third of the root may be handled as an accessory canal and filled with an excess amount of sealer in an effort to fill the perforation. Weine¹ advised the use of heavy condensation or solvent techniques with gutta-percha as more conducive toward sealing the true canal as well as the perforation. Sinai⁴⁰ further suggested that routine root canal filling may be utilized in cases of internal resorption when a small perforation has occurred.

2. Perforations Sealed as an Additional Canal.

Several authors^{26,33,40} have noted that perforations in the coronal two-thirds of the root will often extend diagonally through the root wall for a distance creating a canal-like effect. Burns⁷⁶ observed this to be particularly true in perforations occurring during post root preparation and advised that the defect be treated as a large canal and obturated accordingly. Nicholls³² showed that when perforations occurred

on the mesial or distal aspect of the root, fairly accurate length determinations could be made by the use of radiographic examination with an instrument placed in the defect. This would then allow debridement and filling of the perforation as a separate canal.

3. Perforations Sealed Through the Chamber.

Frank³³ and Burns⁷⁶ held the view that treatment of a perforation via an intracoronal access was preferable in light of the possibility of a periodontal defect occurring if a surgical approach was taken. Consequently, many methods have been suggested for the management of perforations in the furcation area and coronal one-third of the root when access is available through the pulp chamber. Several authors^{21,28,35,49,63,64} have stated that perforations in the floor of the pulp chamber should be treated by the creation of a retentive preparation in the area of the defect. Amalgam may then be condensed into the perforation with care such as to not extrude excessive filling material. Nicholls³¹ and Taatz and Stiefel⁵³ have 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. Harty²⁶, Bouchon⁵¹, and Lange²⁰ advocated that the accessible perforation should be covered with a sheet of gold, tin, or platinum, which is then sealed in place with gutta-percha, amalgam or a suitable cement. Auslander and Weinberg⁷⁷ in 1968

reported the use of indium foil, a pure, soft, ductile metal, as a matrix for perforation repair. They indicated that the indium foil and amalgam will coalesce into a single solid phase with no detectable junction and provide a satisfactory seal.

Diamashkieh⁷⁸ in 1975 described a procedure whereby sterile oxidized cellulose was first packed into the bony defect of a perforation to form a base for the condensation of amalgam in order to seal the perforation. Harris⁷⁹ in 1976 reported a clinical study where perforations were successfully sealed with Cavit through the intracoronal approach. Weissman⁸⁰ in 1970 presented a case where a furcation perforation had taken place. A wedge was placed between the roots of the mandibular molar to allow condensation of amalgam via the pulp chamber.

Frank⁵⁶ in 1974 stated that perforations coronal to the supporting structures may be sealed with an acceptable filling material, preferably amalgam. Where esthetics became a factor, he recommended the use of a more esthetic filling material such as a composite or silicate restoration.

4. Perforations Sealed via a Surgical Approach

Numerous clinicians^{21,24,26,34,50,53,59,63,65,73,74,81,82,83,84,85,86} have discussed the employment of a surgical approach for labially placed perforations to expose the defect and effect its repair with amalgam, gutta-percha, or zinc-oxide and eugenol cement. Nicholls³¹ summarized the possible indications for sur-

gical correction of a perforation. They were: perforations due to resorption, perforations involving a large area of the root surface, perforations which could not be adequately filled by a conventional endodontic procedure, and when healing has failed to take place after an attempt to seal the perforation nonsurgically. He also noted that perforations at or near the alveolar crest often resulted in the formation of a periodontal pocket due to apical proliferation of epithelium. Surgical exposure in such cases was recommended to recontour the alveolar crest and to allow either filling of the perforation or to facilitate its inclusion within a larger coronal restoration. It was further added that surgical repair may also be necessary in cases of large overfillings of the defect⁴⁸, posts cemented in place⁴⁰, or where large extensive coronal restorations are present³⁵.

5. Apicoectomy, Root Amputation or Hemisection.

Many authors^{21,35,36,40,63,74,75} have observed that perforations occurring near the apex of the tooth frequently make instrumentation and filling of the true canal impossible. Where such misadventures had occurred, it was suggested that an apicoectomy be performed provided a satisfactory crown-root ratio remain after surgery. Luebke, Glick, and Ingle⁸² in 1964 advised that the root canal filling be completed to the point at which the instrument deviated from the canal and the apical root tip be resected down to sound root structure with an adequate root

filling. Weine¹ further commented that a reverse fill may be placed if the canal filling appears deficient.

A more complex situation is noted to exist when the perforation occurs in a surgically inaccessible area such as the distal surface of the mesial root of a mandibular molar or the lingual surface of the mesiobuccal root of a maxillary molar. In such cases, root amputation or hemisection have been advocated as necessary to salvage at least a portion of the tooth^{1,19,40,82,84}. Several authors^{28,31,48,56} have also suggested similar procedures for perforations of the furcation due to their less favorable prognosis.

6. Perforations Repaired by Stimulation of Calcium Hydroxide.

Frank⁸⁷ in 1966 demonstrated the potential of continued apical development in the pulpless undeveloped tooth with a wide open apex through the use of a temporary seal of calcium hydroxide. Frank and Weine⁸⁸ in 1973, based on the above study, presented a technique whereby calcium hydroxide was utilized to effect periodontal repair in perforation defects resulting from internal resorption. After hemorrhage control and biomechanical enlargement, they suggested that the canal be filled with a paste of calcium hydroxide and camphorated parachlorophenol. When repair had taken place, the temporary seal of calcium hydroxide was removed and replaced with a permanent root canal filling. The healed periodontal tissues then served as a

limiting factor for the condensation of the filling material. Other investigators^{31,40,53,56,89} have also recognized the potential of calcium hydroxide as an inducer of hard tissue formation external to the tooth. They have thus indicated the use of calcium hydroxide in iatrogenic perforations too extensive to be sealed by routine filling methods.

7. Intentional Replantation.

Grossman⁹⁰ in 1966, Chivian⁹¹ in 1967, and Messing⁹² in 1970, in discussions on intentional replantations, listed as possible indications such complications as perforating internal or external resorptions and iatrogenic perforations which could not be sealed off or were inaccessible to a surgical approach. A number of other authors^{21,28,81,74,91,93} have also suggested the use of intentional replantation as an alternate method of treating perforations.

The procedure involves the intentional removal of the tooth and its reinsertion into its socket after endodontic manipulation or obturation of the canals or both. After extraction, the perforated region is prepared with a sterile bur to produce a retentive preparation and the defect sealed with amalgam. Luebke, Glick, and Ingle⁸², however, expressed the view that intentional replantation is a rarely utilized surgical technique which is applied only as a last resort.

8. Orthodontic Treatment of Perforations.

Heithersay⁹⁴ in 1973 and more recently Simon and colleagues⁹⁵ in 1978 reported on a combined endodontic-orthodontic technique which would facilitate treatment of root perforations in the coronal one-third of the root. The described procedure involved the use of orthodontic extrusion to elevate the root perforation above the level of the alveolar crest. In such a position, the perforated region would be eliminated and would allow restoration of the tooth without extensive surgery.

HISTOLOGIC STUDIES

Although numerous articles have been written concerning the management of perforations, relatively few histologic studies have dealt with the nature of the tissue reaction and subsequent attempts at repair of perforations.

Euler⁹⁶ in 1925 was one of the first to study the histological tissue response to perforations. Experimental perforations were made in the coronal one-third of the root of the canines of dogs. He sought to compare tissue reactions following perforations made under aseptic conditions with those following salivary contamination. The perforated canals were untreated while the entrances were sealed with phosphate cement. The early periodontal tissue reactions associated with the aseptic perforations revealed a differentiation of fibrous connective tissue, followed after 14 days by osteoblastic activity and apical proliferation of the gingival crevicular epithe-

lium. The end result, after ninety days, was an epithelialized polyp extending from the gingival margin into the perforation canal. The early tissue reactions following contaminated perforations were marked inflammation and irregular formation of fibrous connective tissue. The final result was proliferation of crevicular epithelium and ultimate abscess formation surrounded by necrotic connective tissue and osteoclasts.

Kubler⁵⁴ in 1941 studied the tissue reactions following infected and noninfected perforations in humans and dogs. The perforations were divided into two groups based on whether a solid or resorbable filling material was used to seal the defect. The histological pictures were, in the beginning, very similar for both infected and noninfected perforations. Following noninfected perforations filled with a resorbable material, fibrous connective tissue proliferated into the perforation canal, provided a rapid apical migration of gingival epithelium did not occupy it first. Some regeneration of previously resorbed bone and cementum did occur, although repair was observed to be minimal overall. Abscess formation occurred where the perforations were infected. Kubler concluded that only those perforations treated under aseptic conditions offered a favorable prognosis.

Ruchenstein⁹⁷ in 1944 reported on a study of the tissue responses to perforations in humans and dogs after sealing the defect with calxyl, a mixture of calcium hydroxide and Ringer's solution. Histologic sections taken five months after the initial operative

procedures in dogs revealed apical proliferation of the crevicular epithelium and inflammatory changes in the surrounding connective tissue in perforations located in the coronal one-third of the root. Granulation tissue and fibrous connective tissue encapsulation surrounded the orifices of the more apically located perforations. Although some repair of bone and cementum was noted, resorption of bone, cementum, and dentin was evident after nine to ten months in most human specimens. In two cases, granulomas had formed at the perforation exits which were presumed to be a result of contamination during the procedures.

Kaufmann⁵⁵ in 1944 conducted an experimental investigation of root perforations on humans. Solid materials such as gold, porcelain and amalgam were used to seal perforation canals situated coronal to the gingival crevice, while iodoform paste was used to seal the more apically positioned perforations. The histologic examination revealed formation of granulation tissue around the perforation exits and, in some cases, obturation of the exits by apposition of cementum. The evidence of repair was less noticeable in the more coronally situated perforations. Kaufmann compared the perforations adjacent to the gingival crevice with a marginal periodontitis which caused exposure of the perforation exits.

Taatz and Stiefel⁵³ in 1965 reported the histological tissue reactions to an untreated perforation 18 months after the operative procedure. The site of the perforation showed irregular resorption and some proliferation of cellular hard tissue which appeared to be

of osteodentin type. They stated that granulation tissue covered the area of the perforation and extruded into the perforation canal from the periodontal membrane. Although the root pulp apical to the perforation was vital, retrogressive changes and calcific deposition was seen separating the pulp from the perforation.

In 1965 and 1967, Lantz and Persson^{57,58} reported an extensive study of the periodontal tissue reactions following root perforations in dogs. This study was conducted in two parts, the first one in which changes in the periodontal bone was studied by means of serial "identical" roentgenograms, and the other concerned with histological examination of the same experimental material. In the roentgenogram study⁵⁷ they observed that perforation canals filled immediately with gutta-percha and chloroform-rosin healed with no destruction of the periodontal bone. On the other hand, rapid bone destruction and resulting periodontal defects were observed in perforations left open or sealed with phosphate cement.

These radiographic findings were confirmed by a follow-up histologic examination⁵⁸ of the same experimental material. A favorable healing response was observed with immediate filling of the perforations. Generally, the initial acute inflammation became chronic in nature after seven days with differentiating fibrous connective tissue forming a limiting capsule around the perforation site. At observation intervals from 83 up to 230 days, inflammation became less noticeable, and osteoblastic activity predominated particularly on

on the bone surface bordering the excess gutta-percha filling. Only scattered osteoclasts were observed in resorption lacunae and the formation of new cementum close to the perforation exit was noted after 178 days. Healing was found to consist of a fibrous encapsulation at the site of the perforation which the authors considered to be a physiologic and functional repair. If the perforation site was not immediately sealed, chronic inflammation and progressive bone destruction ensued until the perforation was sealed whereupon healing was observed to occur, but at a slower rate. When the canal was left with no seal, the perforation area underwent osseous resorption, granulation tissue formation, epithelial proliferation, and periodontal pocket formation. The perforation canals became infected foci with resultant widespread inflammation and abscess formation.

In a review of accidental perforations of the pulp chambers from previous experiments on primates, Seltzer, Sinai and August⁶¹ in 1970 found that the damage to the periodontium ranged from mild to severe, depending in part on how quickly the perforation was closed. The most severe reactions occurred when the perforations were not closed immediately. Exposure to salivary contamination stimulated proliferation of epithelium and resorption of bone and dentin which resulted in a decreased capacity for the regeneration of periodontal fibers and bone. In every instance, chronic inflammation, often accompanied by acute inflammatory cells, was present which indicated the continuation of the destructive process. However, when the perforation was sealed immediately, there appeared to be potential for

periodontal tissue regeneration except over the perforated region provided that the periodentium incisal to the defect remain intact. Their findings indicated that perforations of the floor of the pulp chamber result in a doubtful prognosis. However, the prognosis for furcation perforations was improved when the defeat was sealed immediately.

Bhaskar⁶² in 1971 reported that perforations of the furcation area of dogs' teeth which were left unfilled demonstrated osteoclastic resorption of the interradicular bony septum, formation of granulation tissue, and epithelial proliferation. The establishment of furcation involvements occurred as well as destruction of the periodontal fibers of the area. The presence of chronic inflammation led to proliferation of the epithelial attachment over the furcation area where the periodontal ligament had been destroyed. Epithelial islands and sheets were observed in the granulation tissue and deep in the interradicular bone. A number of teeth showed resorption of cementum and all unsealed perforations demonstrated the histologic features of a periodontal pocket. Bhaskar observed that furcation perforations that were sealed showed less severe interradicular damage than those left unsealed.

Schwartz⁶⁰ in 1970 examined the histologic response of the periodontal tissues to treated mechanical perforations of the floor of the pulp chamber in six Rhesus monkeys. Due to variation in size of the perforations produced, an unexpected division of the experimental material resulted. He observed that in the sections examined

in the 24 hour to 8 week interval, the perforation was relatively small and well delineated. Extremely large perforations were found in the 12 week and the 24 week specimens. The small sealed perforations that were removed from the crevicular epithelium demonstrated successive diminution of the inflammatory reaction and progressive repair. The larger defects exhibited a similar initial acute inflammatory response which became progressively chronic in nature. Although there was an attempt by the underlying connective tissue to organize a protective barrier, the overall tissue response demonstrated a chronic cellular inflammation with epithelial proliferation.

Lantz and Persson⁵⁹ in 1970 endeavored to find a clinically superior method of treatment for root perforations. Their previous investigations^{57,58} had found that a major problem in sealing perforations was the extrusion of excess filling material into the periodontal tissues during the nonsurgical repair of perforations. Utilizing various techniques and examining the results histologically, they found that an immediate filling of the perforation with either amalgam or gutta-percha through a surgical approach provided an optimal prognosis. They observed that gutta-percha was better tolerated by the tissues than amalgam, although both materials resulted in a persistent, mild chronic inflammatory reaction.

CAVIT

In general, endodontic procedures usually require multiple appointments, thus necessitating the use of a temporary filling material to seal the access opening of the treated tooth between appointments. This prevents contamination of the root canal by the oral environment and protects the canal orifices from blockage by food particles and debris.

For many years, the universal temporary filling material was gutta-percha, the coagulated milky precipitate of the sap of the Malaysian rubber tree. Additives such as zinc oxide and white wax were incorporated with this rubber-like material to render it usable as a temporary restorative or root canal filling. The gutta-percha was softened by heat and inserted into the cavity where it hardened as it cooled. However, though commonly used in the past, it soon became apparent that gutta-percha was not a satisfactory temporary filling material because of its excessive marginal leakage and lack of strength.

Among the other available materials, the zinc oxide-eugenol cements were found to be superior in demonstrating minimal marginal leakage. However, the low strength, poor abrasion resistance and high flow of conventional zinc oxide-eugenol cements had limited its usefulness as a temporary restorative material. A widely used method to compensate for these deficiencies was to fabricate the restoration of gutta-percha and cement it in place with a zinc oxide-eugenol cement.

The zinc phosphate cements were introduced and found to be superior to gutta-percha and zinc oxide-eugenol in terms of compressive strength and abrasion resistance. However, in areas subjected to masticatory stress, it was observed that these cements still lacked the necessary strength and resistance to abrasion. Furthermore, it became obvious that the zinc phosphate cements were ineffective in producing an adequate seal when utilized as a temporary restoration. Similar observations were noted for the silicophosphate cements⁹⁸.

Although strength and resistance to abrasion were observed to be important factors, the primary consideration for an endodontic temporary filling material must be its ability to seal the access opening to prevent seepage in either direction⁹⁹. Numerous studies have been made concerning the sealing ability of various temporary filling materials. Fraser¹⁰⁰ in 1929 concluded that a gutta-percha temporary restoration was "most ineffecient" in sealing a cavity to the passage of bacteria. Grossman¹⁰¹ in 1939, tested the sealing abilities of various materials in capillary tubes. Only zinc oxide-eugenol cement was found to seal effectively, while zinc oxyphosphate was least effective and gutta-percha intermediate in sealing ability. Massler and Ostrovsky¹⁰² in 1954 utilizing glass cavities under controlled conditions, showed that marginal leakage occurred within twenty-four hours with all materials except zinc oxide-eugenol cement and amalgam. Similar results were obtained by Moller¹⁰³ in 1966 who reported that zinc oxide-eugenol cements yielded excellent

results in producing a bacteria-tight seal. In 1963, Kakar and Subramanian¹⁰⁴ and Norman, Swartz and Phillips¹⁰⁵ both concluded that zinc oxide-eugenol cements provided the most effective sealing ability of temporary filling materials tested.

In contrast to the previously discussed studies, Fischer¹⁰⁶ in 1949 and Nelson, Wolcott, and Paffenbarger¹⁰⁷ in 1952 demonstrated that under the conditions of their investigations, all zinc oxide-eugenol cement formulations showed some degree of fluid penetration at the margins.

Noting the above two studies and the fact that no material has met all the requirements of an effective temporary restoration as listed by Grossman²¹, various manufacturers have endeavored to produce a superior temporary filling material. An example of these products would include Cavit*, a premixed polyvinyl paste¹⁰⁸.

The literature review will now concern itself with a discussion of those studies relating to the biological and physical properties of Cavit, and its uses in clinical applications.

Cavit is manufactured in West-Germany and contains zinc oxide, calcium sulfate, zinc 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: the reaction of water with calcium sulfate and zinc oxide-zinc sulfate produces the set¹⁰⁹.

* Premier Dental Products Company, Philadelphia, Pennsylvania.

Widerman, Eames, and Serene¹¹⁰ in 1971 reported on the physical and biological properties of Cavit as compared to those of zinc oxide-eugenol. They found that the compressive strength of Cavit was approximately one-half of the value reported for zinc oxide-eugenol (1,973 psi vs. 4,000 psi). The coefficient of linear expansion was almost double that reported for zinc oxide-eugenol and this was postulated as being responsible for the sealing effectiveness of Cavit. With penetration tests utilizing methylene blue dye, the dye was visible throughout the depth of the material rather than only at its interface with the walls of the test cavity. This indicated to the investigators that instead of actual marginal leakage, the dye was absorbed by the Cavit. The solubility and disintegration values obtained for Cavit appeared to be thirty times greater than those values observed with zinc oxide-eugenol. In addition, the degree of water absorption of Cavit was found to be approximately six times that observed with the control.

Parris and Kapsimalis¹¹¹ investigated the sealing ability of various temporary filling materials in vitro. The apices of extracted single rooted teeth were resected and the root end filled with amalgam. Lingual access cavities were then made, cotton fibers placed inside and sealed with one of the test materials. The teeth were next subjected to cycles of temperature changes from 4°C to 60°C and then immersed into two percent aqueous solutions of aniline dye. After seventy-two hours, the access cavities were opened and

the cotton fibers examined for evidence of dye penetration. Cavit and amalgam were the only materials to effectively seal all experimental cavities at both room temperature and after repeated temperature changes. Gutta-percha, zinc oxide-eugenol, and zinc phosphate cement all demonstrated leakage to some extent.

Parris, Kapsimalis, Cobe, and Evans¹¹² in 1964 then investigated the sealing ability of various temporary filling materials to both bacterial and dye penetration. Similar methodology was utilized as in the earlier study by Parris and Kapsimalis¹¹¹ except that in addition to thermal cycling, the teeth were also immersed in twenty-four hour growth solutions of Serratia marcescens and Sarcina lutea alternatively to test bacterial penetration at the margins of the tested materials. Under sterile conditions, the access cavities were reopened and the cotton fibers recovered and cultured for the presence of the test bacteria. Cavit and amalgam were again found to be superior to gutta-percha, zinc phosphate cements and zinc oxide-eugenol.

Marosky, Patterson and Swartz¹⁰⁹ in 1977 compared the micro-leakage of six commercially available products used as temporary restorations. Their sealing effectiveness was based on their ability to prevent penetration of an aqueous solution of calcium chloride Ca^{45} at the tooth-restoration interface. The materials were subjected to thermal cyclings between 10°C and 50°C and tested for leakage at three and ten days after placement of the

temporary filling material through the use of autoradiograph examination. Cavit was shown to have superior sealing ability when compared to zinc oxide-eugenol cement, zinc phosphate cement, Intermediate Restorative Material (IRM) and polycarboxylate cements, but not as well as Temp-Seal. However, it should be noted that the Union Broach Company had recalled all Temp-Seal temporary filling material because of recent Federal Drug Administration findings of its lead and cadmium content.

Weine¹ discussed the results of an autoradiographic study of the sealing capabilities of Cavit and Cavit G. Central incisor access cavities closed with Cavit demonstrated considerable leakage when submitted to autoradiogram examination. He acknowledged, however, that a sizable percentage of practitioners do obtain negative cultures with Cavit, indicating that isotopes may penetrate while microorganisms may not.

In another autoradiogram study, Bramante and Bernardinelli¹¹³ in 1977, sealed extracted premolars with various temporary filling materials. After immersion in a solution of NaI with active I¹³¹ for twenty-four hours, the teeth were sectioned and prepared for autoradiographic examination. They found that IRM sealed best with zinc oxide-eugenol and Cavit less effective in sealing ability. However, the differences were not quantitated and all three materials appeared similar in sealing ability.

Gilles, Huget, and Stone¹¹⁴ in 1975 investigated the effects

of thermal cycling on the dimensional stability of temporary restorative materials. They noted that materials such as Cavit absorb water readily and thus may expand markedly in the aqueous environment of the oral cavity. Yet these dimensional changes may be augmented or counteracted by temperature fluctuations. In their experiment, Cavit was found to show less dimensional changes than gutta-percha temporary stopping and two cements containing zinc oxide-eugenol. However, since the testing was performed under dry conditions, correlation to actual in vivo conditions may be limited.

Krakow, deStoppelaar, and Gron⁹⁹ in 1977 performed the only reported in vivo study of temporary filling materials used specifically in endodontics. The access cavities of previously filled root canals of anterior teeth in patients were utilized as the test model with all materials being tested in the same tooth for a minimum of one week. Seepage was determined bacteriologically by culturing a cotton pellet which had been sealed in the access cavity. Cavit was observed to seal with no leakage or minor leakage and was approximately equal to zinc oxide-eugenol in sealing ability. Antimicrobial action was also tested and their results indicated Cavit to be less antibacterial than zinc oxide-eugenol both in degree of bacterial inhibition and in types of colonies inhibited.

The preceding studies revealed that Cavit could produce a reliable leakproof seal. A number of clinicians have then tested Cavit as a candidate for use as a root canal filling material and

for reverse filling procedures.

Wallentin¹¹⁵ in 1973 reported an in vitro study where thirty extracted teeth were filled with Cavit or gutta-percha and Grossman's sealer. After immersion for 72 hours in an aqueous aniline dye solution, the teeth were sectioned and examined through the light and electron-scanning microscope. The results revealed a better marginal adaptation and less penetration of dye in those teeth filled with Cavit when compared to those teeth filled with gutta-percha and Grossman's sealer. Wallentin expressed the belief that the expansion of Cavit due to its water absorption was responsible for its superior adaptation. In a later report in 1974, Wallentin¹¹⁶ noted clinically successful results with the use of Cavit as a root canal filling material in an unspecified number of cases treated in his private practice.

Fogel¹¹⁷ in 1977 examined the suitability of utilizing various filling materials for endodontic obturation with a pressure syringe. Extracted teeth were used and after filling the canals with a test material, they were subjected to immersion in methylene blue dye for intervals of one, seven and thirty days. He found that Cavit and gutta-percha with Rickert's formula did not differ significantly from each other in terms of marginal penetration and permeability. Fogel felt that while gutta-percha with sealer is known to be clinically successful, on the basis of his study, Cavit may also provide equally good results. However, Cavit was shown to be the

most porous of the materials tested which indicated that the slight marginal leakage exhibited may be due to its porosity and not to a lack of marginal adaptation.

It would be well to note here that Frank³³ in 1978, under whose direction the study by Wallentin¹¹⁵ was conducted, stated that the most recent clinical impressions have been that Cavit has not proven to be as successful as conventional endodontics utilizing gutta-percha and sealer. The consensus of both Frank and Wallentin was that after a number of years, there appeared to be a deterioration of the seal afforded by Cavit due to a probable dissolution of the material. This resulted in an underfilled root canal which, in a significant number of cases, have led to failure necessitating retreatment. Frank concluded that the use of Cavit as a root canal filling material was not indicated due to lack of reliable and substantiated long term results.

Cavit has also been advocated by a number of authors for use as a root-end filling material. McGivern in 1974¹¹⁸ reported clinical success in fifty cases of reverse filling with Cavit observed for up to two years after the initial procedure. Radiographically, he observed that no foreign body reaction occurred and concluded that Cavit was superior to amalgam as a root-end filling material due to its ease of placement and "tighter bacteriostatic" apical seal.

Nord¹¹⁹ in 1970 reported a study of 354 teeth reverse filled with Cavit which were examined from six months to six and one-half

years after the surgical procedure. His interpretation clinically and roentgenographically was that healing of the bone, with re-establishment of the periodontal ligament space, occurred in 61%, incomplete healing in 17%, and no healing in 22%. He noted that resorption of the reverse Cavit filling was observed in eight cases, but of these five were considered to be successful. Nord further stated that he found Cavit to be unaffected by moisture or hemorrhage during manipulation, and easy to handle.

In a similar study, Persson, Lennartsson and Lundstrom¹²⁰ compared amalgam with Cavit as a reverse filling material. Based on roentgenographic interpretation, they found a significantly better result with amalgam. It was postulated by the investigators that Cavit provided an inferior obliteration of the canal when compared to amalgam.

A three year follow-up review by Finne, Nord, Persson and Lennartsson¹²¹ in 1977, was conducted on those patients previously observed in the study by Persson, Lennartsson and Lundstrom¹²⁰. It was again reported that amalgam produced significantly better results than Cavit when used as a reverse filling material. They found that a high percentage of the failures associated with Cavit occurred in those teeth with incomplete orthograde root canal fillings. Moreover, a dissolution of the Cavit filling was observed in several cases in the unsuccessful group. They interpreted this as a lack of marginal seal by Cavit to the egress of toxic root

canal products due either to inherent physical characteristics such as through and through absorption of fluids, or to dissolution of the material in tissue fluids. They concluded that Cavit was unable to provide either a complete or permanent obliteration as a reverse filling material.

Pisano¹²² in 1976 utilized C¹⁴-labeled glucose in a quantitative radioautographic investigation of microleakage in endodontic reverse fillings. Seventy-two extracted central incisors were reverse filled with amalgam, Cavit and warm gutta-percha to investigate the marginal sealing capabilities of these materials. His study concluded that Cavit was inferior to silver amalgam and warm gutta-percha with sealer.

Delivanis and Tabibi¹²³ in 1978 reported the results of an in vivo study designed to compare the sealing properties of Cavit and zinc polycarboxylate cement with amalgam when used as reverse filling materials. The test materials were surgically placed in the apices of dogs' teeth and left for a six month period. Upon extraction, the amount of marginal leakage was evaluated by the use of C¹⁴-labeled urea. A second part of the study included an in vitro evaluation of the same materials to compare and evaluate the effects of aging on the seal afforded by the various materials. In the in vivo study, it was revealed that the Cavit reverse fillings had degraded in a number of cases. The adaptation of Cavit to the walls of the root end preparation was poor and the surface rough with

defects. The poor results were assumed to be due to dissolution and disintegration of the material in tissue fluids. Resorption of Cavit by the body's defense mechanism was postulated as being an additional factor. The seal provided by amalgam was noted to improve with time, while that provided by Cavit deteriorated to a significant extent. Under the conditions of the in vitro study, amalgam was observed to provide a better seal than Cavit after six months.

As mentioned previously, Harris⁷⁹ presented a nonsurgical approach for the repair of perforations with Cavit. A clinical and radiographic evaluation of 159 patients treated in this manner revealed a success rate approaching 90 percent.

Studies concerned with the tissue tolerance of Cavit have been quite limited as the majority of research has been involved with the physical characteristics and sealing ability of the material. Any evaluation of a material to be used in direct contact with living tissue demands an insight into past findings dealing with the biological acceptance of that material.

Wilderman, Eames and Serene¹¹⁰ found that Cavit placed in dried Class V cavity preparations resulted in aspiration of odontoblastic nuclei in all teeth tested. This was observed to subside after ten days with no permanent pathosis other than a rapid deposition of reparative dentin. Provant and Adrian¹²⁴ in 1978 evaluated the use of Cavit as an insulating base and temporary restorative

material in teeth with vital pulps. The displacement of odontoblastic nuclei was used as a criteria of pulpal injury and graded (1) minimal, (2) mild, (3) moderate, or (4) severe. Statistical analysis of the average intensity values of odontoblastic-nuclei displacement at different time periods showed no significant difference between Cavit and zinc oxide-eugenol in pulpal response.

Flanders¹²⁵ in 1975 utilized the connective and osseous tissues of the rat to investigate the tissue tolerance of Cavit in comparison to zinc-free amalgam. The experiment involved direct implantation subcutaneously and adjacent to bone. Histopathologic criteria were: relative thickness of the connective tissue capsule surrounding the implanted material, cellularity and vascularity of the capsule, relative numbers and types of inflammatory cells, and response of the adjacent tissue, particularly bone. Subcutaneously, the connective tissue capsule surrounding the Cavit implant was observed to be four times thicker than the capsule around the amalgam implant. The inflammatory cell infiltrate consisted primarily of macrophages, lymphocytes, plasma cells and a few multinucleated giant cells. Numerous blood vessels were observed in the capsule and a zone of necrotic tissue mixed with Cavit debris was found immediately adjacent the implant. Around the capsule was noted an irregular, immature collagenous connective tissue stroma with a large number of fibroblasts. The paramandibular implants of Cavit resulted in a foreign body response in bone. Cellular death was indicated by the

absence of osteocytes in Howship's lacunae. The response to Cavit appeared to be well localized, but overall produced a more severe foreign body response than amalgam at every time interval.

Antrium¹²⁶ in 1976 evaluated the cytotoxicity of Grossman's sealer, N2, Rickert's sealer and Cavit. After incubation at 37°C for periods ranging from twenty-four hours to seven months, the experimental materials were exposed to radioactively labeled human tissue culture cells. This resulted in a cell-material interaction which allowed for quantitation of tissue cell cytotoxicity. The results indicated that all materials possessed lasting tissue toxicity. In consideration of relative toxicity, Grossman's sealer and N2 were considered highly toxic while Rickert's sealer displayed moderate toxicity. Cavit was observed to be mild to moderate in degree of toxicity.

CHAPTER III

MATERIALS AND METHODS

Six adult beagle dogs, three females and three males were used as experimental subjects. They were procured, treated and maintained at the Loyola University Medical Center Animal Research Facility and weighed between 9.5 and 13.5 kilograms with an average weight of 11.1 kilograms. Each dog was numbered by collar tags for consistent identification and all procedures recorded on an appropriate work sheet. The animals were maintained on a diet of standard laboratory meal and water ad libitum.

A total of thirty-six mandibular premolar teeth in twelve quadrants were operated upon and examined in this study. This allowed for examination of thirty-one perforations filled with Cavit and seventeen control perforations.

Each animal was first premedicated in its cage with an intramuscular injection of one c.c. of Inovar Vet* per seven to nine kilograms of body weight for sedation to allow its safe removal and handling. A time of twenty to thirty minutes was found to be sufficient for the tranquilizer to act. At the same time, the dog received a subcutaneous injection of one c.c. of Atropine Sulfate

* Pitman-Moore, Inc., Washington Crossing, New Jersey.

Injection U.S.P.* (0.5 mg./ml.) to decrease salivary secretions.

Utilizing routine operating room sterility, general anesthesia of surgical depth was obtained by administration intravenously of Sodium Pentobarbital Injection** (65 mg./ml.). Venipuncture was made on the dorsal surface of the foreleg in the anterior superficial vein. The barbituate was slowly administered until adequate anesthesia was obtained as determined by loss of pedal and corneal reflex and the presence of deep, regular breathing. This provided one and a half to two hours of working time and maintenance dosages of one c.c. were administered as required due to the length of the procedure.

Preoperative radiographs were taken prior to any operative procedure for evaluation of alveolar crest level, indication of apical pathosis extending incisally as well as patency of the root canal spaces. A portable hand-held x-ray unit*** utilized at a setting of sixty Kvp. at twenty milliamperes for 0.2 second provided acceptable radiographs. Occlusal ultra-speed dental x-ray film**** was used, and developed and fixed for twenty and forty seconds respectively in rapid processing solutions***** in a portable table-top developing box.

The maxillary and mandibular jaws were retracted and held open

* Med. Tech., Inc., Elwood, Kansas.

** W.A. Butler Company, Columbus, Ohio.

*** General Electric - 15 ma portable unit.

**** Kodak DF-46, Eastman Kodak Company, Rochester, New York.

***** Insta-Neg/Instra-Fix, Microcopy, Culver City, California.

by the use of a spring loaded prop that attached to the cuspid incisal tips of the side opposite that being instrumented. The teeth to be operated upon were evaluated periodontally using a periodontal probe and the readings recorded for comparison at the time of sacrifice. A #557 carbide bur with water in a high speed handpiece was used to grind down the crowns of the teeth until near exposure of the pulpal tissues. Coronal access was gained and mechanical undercuts created for retention of the restorative material used at the end of the procedure.

The root canals were located and prepared according to Weine¹, including incremental instrumentation and flaring. Sterile saline was used as the irrigant. Final instrument sizes ranged from number thirty to seventy files, consistent with canal morphology and the age of the animal.

The root canals were dried with sterile absorbent paper points and examined carefully for any signs of hemorrhage or organic debris. Wach's Paste* was mixed and placed into the canals on a sterile file. The root canal space was then obliterated by the lateral condensation technique using a standardized gutta-percha master cone** and conventional sized accessory gutta-percha cones***. The coronal portion of the gutta-percha filling was removed from the root canals with heated endodontic pluggers and large files. Three to four millimeters

* King's Specialty, Fort Wayne, Indiana.

** Premier Dental Products Company, Norristown, Pennsylvania.

*** Mynol, Inc., Broomall, Pennsylvania.

of apical gutta-percha was left in the canal to maintain a seal at the apex.

Engine driven reamers* sized eighty and ninety were used to create perforations through the side of the root. The extent of the perforations were through the periodontal ligament and into the adjacent periodontium. The lateral perforations were made toward the proximal such as to allow better visualization on radiographic and histologic examination. Only one perforation was created in any one particular interproximal space. This was to forego the possibility of confusing the histologic pictures should two perforations be made in the same immediate area from two adjacent teeth. The perforation was mechanically cleansed by irrigation with sterile saline and bleeding controlled by use of sterile paper points. The experimental perforations were sealed with Cavit placed with endodontic pluggers. No attempt was made to limit the material strictly within the tooth as overfilling with Cavit was judged to stimulate actual clinical conditions and allow examination of the tissue response to the extruded material.

The control teeth were likewise perforated and bleeding controlled in a similar manner. However, no attempt was made to seal the perforation other than the placement of a cotton pellet into the perforation entrance. In both experimental and control teeth, the coronal accesses were sealed with temporary stopping and amalgam (Figure 8 and Table 1).

* Union Broach Corp., Long Island City, New York.

Postoperative radiographs were taken and the animals returned to their cages.

The six dogs were sacrificed at intervals of one, fifteen, 30, 60, 120 and 180 days. The animals were anesthetized, radiographed and probed periodontally. Tooth mobility, draining sinus tracts or any obvious soft or hard tissue pathosis were noted and recorded. Each animal was sacrificed by an injection of eight to ten c.c. of Beuthanasia-D*.

The mandibles were exposed by dissection of the surrounding soft tissues. The jaw sections containing the experimental and control teeth were removed with a reciprocating surgical saw and immediately placed in a solution of ten percent neutral buffered formalin for fixation. One hour later, unnecessary hard tissue and the amalgam restorations were removed with high speed burs to facilitate tissue fixation. Final radiographs of the jaw sections were taken for correlation with later histologic examination.

Approximately fifteen days later, the sections were removed from the formalin solution and rinsed for twenty-four hours under running water. The specimens were decalcified for four to six weeks in a solution of equal parts of twenty percent sodium citrate and fifty percent formic acid until they were of a rubber-like consistency. They were then trimmed with a razor blade to eliminate extraneous tissue and sectioned longitudinally to expose the perforation sites.

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

After decalcification for several additional days to allow better penetration, the specimens were dehydrated, embedded in paraffin, and six micron sections cut and mounted on glass slides. The sections were deparaffinized, hydrated and stained with hematoxylin and eosin. The stained histologic sections were examined using a light microscope and evaluated according to accepted histologic techniques. All data was recorded.

CHAPTER IV

RESULTS

The dogs were reexamined at varying intervals from the time of operation to the time of sacrifice and appeared to suffer no ill effects from either the experimental procedures or the anesthesia.

The dentition of beagles contains four two-rooted premolars in each lower quadrant of which the second, third, and fourth premolars were utilized. As noted previously, only one perforation per interproximal space was created. In the discussion and tables to follow, the teeth and their respective perforation sites will be designated as in the following samples: lower left third premolar, distal root = LL3-distal; lower right second premolar, mesial root = LR2-mesial; and so forth.

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. These findings were indicative of an incipient periodontitis which was confirmed by later histologic examination. Periodontal pocket depth readings were obtained both preoperatively and at the time of sacrifice. The data is summarized in Table II. Increased pocket depths at sacrifice were found to

correspond histologically to the formation of a periodontal pocket.

Radiographs of the jaw sections taken after sacrifice were quite revealing and are shown in Figures 9-14. Though the findings of this portion of the study are tentative, a number of significant observations were made. It was first apparent that the technical aspect of sealing perforations with Cavit was difficult in that gross overfilling generally occurred despite the best efforts of the operator. At the 60 day interval and beyond, the formation of a thin radiolucent line was noted around the excess filling material and was clinically interpreted as capsule formation around the Cavit. In a number of specimens, both filled and unfilled, progressive destruction of the alveolar bone could be visualized as well as small areas of root resorption. Finally, it was found in several instances, that fracture of the Cavit had occurred just beyond the root leaving a segment of the material embedded in the periodontal tissues.

Histologic examination of the normal periodontium revealed a para-keratinized oral sulcular epithelium with a mild infiltration of plasma cells within the underlying connective tissue (Figure 15). The supra-crestal gingival fibers were intact and a small number of plasma cells and lymphocytes were seen interspersed among the fibers (Figure 16). The alveolar bone appeared normal with a number of areas showing evidence of osseous remodeling. The trabecular spaces were generally occupied with a normal fatty bone marrow and blood vessels. The periodontal ligament was free of periodontal disease with collagen fibers

oriented obliquely and perpendicularly to the root and alveolar bone proper (Figure 17). Root and cementum resorption was not found, though secondary cementum deposition was observed near the apical regions.

UNFILLED PERFORATIONS

In the one day animal inflammation consisted of vasodilation, margination of white blood cells and histiocytic enlargement within the adjacent bone marrow spaces (Figure 18). At the site of injury, osteocytes were missing from the adjacent alveolar bone and clumps of debris, consisting of hemosiderin, extravasated red blood cells, dentin and bone particles were found in the perforation canal. The periodontal ligament was severely disrupted probably as a result of mechanical trauma from the procedure (Figure 19).

In the fifteenth day animal (Figure 20), two unfilled perforations located in mid-root revealed under higher magnification, an acute inflammatory response with a dense infiltration of polymorphonuclear leukocytes, macrophages and some plasma cells. Concomitant vasodilation and extravasation of red blood cells was also noted. Hyalinization of the adjacent periodontal ligament indicated degenerative changes. 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 was seen.

Multinucleated giant cells were evident in lacunae along the adjacent root surface.

In the thirty day animal, two of the three unfilled perforations (LL3-mesial and LL4-distal) were found to have been placed in the coronal one-third of the root. A pronounced acute inflammatory reaction was seen along with vascular engorgement of blood vessels in the adjacent connective tissue and bone marrow spaces. Surrounding the defect was a loose connective tissue stroma of fibroblasts, undifferentiated mesenchymal cells, single collagen fibers and inflammatory cells. Incisally, the periodontal ligament appeared to be in various stages of degeneration while apically disorganization with fibrotic activity was seen. The supracrestal gingival fibers were intact, but appeared to be degenerating due to the coronal extension of inflammation from the perforation defect. The alveolar bone immediately adjacent to the perforation had resorbed, resulting in a vertical periodontal defect. Osseous remodeling was now evident with pronounced osteoblastic activity. Root resorption was seen at different points along the root surface with few signs of repair (Figure 21).

The third defect (LR2-mesial) in this time interval was located just at the level of the alveolar crest and revealed a mixed inflammatory infiltrate of polymorphonuclear leukocytes, macrophages, plasma cells and lymphocytes. The epithelial attachment was lost and proliferation of epithelium towards the defect was observed

(Figure 22). This produced a communication of the inflammatory process from the perforation with that of the gingiva. Medullary space fibrosis was noted as well as marked osteoclastic and dentinoclastic activity. Evidence of repair was minimal.

In the sixty day animal, the three unfilled perforations were situated in the coronal one-third of the roots. Inflammation was marked with a predominance of polymorphonuclear leukocytes and macrophages near the defect with chronic inflammatory cells observed nearby. Vasodilation and a few multinucleated giant cells were also noted. Incisally, the periodontal ligament had undergone progressive degeneration and hyalinization of the supracrestal fibers were seen. There was evidence of communication of the inflammatory processes associated with the gingiva and the perforation. The epithelial attachment was lost and rete pegs of the epithelium were noted to be extending apically. The surrounding alveolar bone had been resorbed and replaced by an irregular loose connective tissue stroma (Figure 23). In one specimen, chronic inflammatory tissue had grown into the perforation canal in the root (Figure 24). Evidence of bony remodeling with signs of osteoid formation and medullary space fibrosis was noted. Active root and cementum resorption was seen with little indication of any repair process (Figures 25 and 26).

In the 120 day animals, the three unfilled perforations were placed in the coronal one-third of the roots. A loose connective

tissue stroma with moderate fibroblastic activity had replaced the resorbed alveolar bone and had extended into the perforation canals in the roots (Figure 27). A low grade chronic inflammation was present, consisting of plasma cells, lymphocytes and macrophages. Active bone reformation was observed beyond the defect. Incisally and apically, the periodontal ligament appeared disorganized and displayed collagen fibers oriented parallel to the tooth surface. The oral sulcular epithelium was found to be intact with no signs of invasion apically. In two more apically located defects, the gingival fibers revealed little change. The third more coronally placed defect, however, showed some degeneration apparently associated with the spread of inflammation incisally from the perforation defect. Root and cementum resorption had essentially halted and some repair by secondary cementum deposition was seen.

In two of the 180 day unfilled perforations (LL2-mesial and LR3-mesial), a pattern of severe destruction was revealed. Epithelium had proliferated almost to the apex, forming a deep periodontal pocket. Beneath the epithelium was chronic inflammatory tissue with an adjacent dense infiltrate of inflammatory cells. A sparse, irregular stroma of connective tissue was present. A large bony defect was evident, while apically active bone remodeling and elaboration was noted. Root and cementum resorption continued with little repair seen. Overall, the histologic picture was compatible with a diagnosis of a periodontal abscess (Figures 28, 29, and 30).

In a third unfilled perforation (LL4-distal) at the 180 day interval, inflammation had subsided to a mild infiltration of plasma cells, lymphocytes and a few monocytes. The oral sulcular epithelium was intact and revealed no observable dystrophic changes. At the perforation site, the resorbed alveolar bone had been replaced by moderately dense connective tissue which demonstrated encapsulation around the defect. A strong osteoblastic response was observed in the surrounding alveolar bone. The collagen fibers of the periodontal ligament were disoriented, but intact. Root and cementum resorption was still evident though the prevalent observation was repair by cementoid deposition. This particular specimen was located in midroot.

PERFORATIONS FILLED WITH CAVIT

In the one day animal, the histologic pictures of the filled perforations paralleled that of the unfilled perforations. Polymorphonuclear leukocytes and vasodilation of blood vessels were observed in the nearby marrow spaces. Disruption of the periodontal ligament had occurred and osteocytes were lost from the lacunae of the adjacent bone. Necrotic and amorphous non-cellular debris lining the Cavit was evident. In total, the initial stages of an acute inflammatory response had begun to appear.

In the fifteen day filled perforations, two somewhat different histologic pictures were discernible. Three well-filled perforations at mid-root (LL2-mesial, LL3-distal, and LR3-mesial) revealed an

inflammatory response consisting of polymorphonuclear leukocytes, macrophages and a few multinucleated giant cells. A scattering of chronic inflammatory cells was visible beyond 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 slightly more basophilic color. Minimal osseous activity was observed, though some osteoclasts were noted along the periodontal ligament (Figure 31).

Three additional filled perforations, two in the coronal one-third of the root and one in mid-root (LR3-distal, LR4-distal and LL4-distal) revealed a more intense inflammatory response. A moderate infiltration of acute inflammatory cells was observed in the immediate area of the perforation. However, in the adjacent connective tissue and periodontal ligament space, an intense chronic inflammatory reaction consisting of plasma cells and lymphocytes was noted. The alveolar bone bordering the Cavit stained more basophilic, but displayed little response other than for a few scattered areas of osteoclastic activity. These specimens revealed 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 (Figure 32). In all cases the oral sulcular epithelium appeared normal and neither root nor cementum resorption was evident.

At the thirty day interval, the five filled perforations presented very similar histologic findings (Figure 33). Generally, a chronic mild to moderate inflammation was present, consisting primarily of plasma cells, macrophages and monocytes. A few multinucleated giant cells were also noted in the capsule surrounding the Cavit (Figure 34). 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. Intense osteoclastic activity was now seen on the bone surfaces bordering the Cavit, while bone remodeling and reformation were noted beyond the immediate area. In one specimen, loss of the alveolar crest had occurred, yet evidence of new osteoid formation could be distinguished in the connective tissue incisal to the perforation. Active root and cementum resorption was not discernible. In the immediate vicinity were areas of root repair by cementoid deposition and osteoid formation which was indicative of ankylosis (Figure 35). In all cases, the epithelium was normal though the gingival fibers appeared to have undergone various degrees of hyalinization and disorganization.

The sixty day specimens revealed two histologic patterns. Two filled perforations (LR3-mesial, and LL2-mesial) situated high in the coronal one-third of the root displayed epithelial proliferation beneath the level of the defect, resulting in the loss of the alveolar crest and supracrestal gingival fibers. A dense collection

of chronic inflammatory cells was evident beneath the epithelium while a moderate inflammatory reaction was visible in the nearby loose connective tissue. Resorption of the surrounding alveolar bone had taken place. Osteoblastic activity was now present in an effort to limit the perforation wound. Extensive fibroblastic activity was also present which displayed an attempt at fibrotic encapsulation below the epithelium. The periodontal ligament had undergone considerable disruption for some distance apically and now exhibited orientation of collagen fibers parallel to the tooth surface. Previous extensive root and cementum resorption now showed evidence of repair by secondary cementum deposition.

Three filled perforations (LR4-distal, LR3-distal, and LL4-distal) at this same time interval of sixty days, displayed neither epithelial proliferation nor degeneration. Inflammation was chronic, though mild in intensity. Multinucleated giant cells were 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 fibrotic encapsulation of the filling material (Figures 36 and 37). Reversal lines indicated new osteoid formation and osteocytes could be distinguished in the newly elaborated bone. Regeneration of the periodontal ligament had taken place, and collagen fibers were noted to be oriented parallel to the root surface. Active cementum and root resorption was still conspicuous, but concomitant repair by cementoid and osteoid deposition was also observed.

The 120 day animals were found to display three discernible histologic pictures in regard to filled perforations.

Three filled perforations (LL2-mesial, LR2-mesial, and LR3-mesial) were situated in the coronal one-third of the root. Epithelial proliferation had occurred, effectively surrounding the Cavit and exposing the defect and filling material to the oral environment. Chronic inflammatory tissue was observed beneath the partially degenerated epithelium as well as evidence of fibroblastic activity (Figure 38).

Another perforation (LR4-distal) at mid-root had only been partially filled with Cavit. The epithelium was intact, but extension of rete pegs apically and degenerative changes in the oral sulcular epithelium were noted. Inflammation was chronic, but mild in nature. An ingrowth of chronic inflammatory tissue and a deposition of secondary cementum were also seen along the inner dentinal walls of the perforation canal. The level of the alveolar crest was unchanged, but very extensive bone remodeling was evident near the site of perforation (Figure 39).

A well-filled perforation of mid-root (LL3-distal) exhibited a very low grade inflammatory response. Enveloping the Cavit was a thick encapsulation by horizontal bands of collagen fibers which were continuous with the periodontal ligament. Active osseous remodeling was noted as was secondary cementum deposition in areas of root resorption (Figures 40 and 41).

In the 180 day animal, one of the filled perforations (LL2-mesial)

was found located high in the coronal one-third of the root and revealed apical proliferation of epithelium resulting in a deep periodontal pocket. Fairly intense chronic inflammation was present in the connective tissue beneath the epithelium and collagen fibers were seen to surround the zone of inflammation. Apically, bone remodeling was observed and the periodontal ligament appeared normal.

The four remaining filled perforations revealed repair via fibrotic encapsulation (Figures 42 and 43). The epithelium was normal and exhibited no overt signs of degeneration. In the tissue around the perforated region, a mild to moderate chronic inflammation was visible and the presence of a few multinucleated giant cells was noted (Figure 44). The bone bordering the Cavit exhibited evidence of extensive remodeling with a prevalence of osteoid formation. A heavy fibrous connective tissue encapsulation had occurred around the Cavit. However, in some areas the continuity of the collagen capsule was disturbed by an apparent disintegration of the material. The tissue around the capsule and between the newly formed bone contained many capillaries, fibroblasts and a relatively loose arrangement of collagen fibers (Figure 45). Regeneration of the periodontal ligament had resulted, though some disorientation of the collagen fibers was still present. Root and cementum resorption had halted and evidence of repair by cementoblastic and osteoblastic activity in several areas resulted in ankylosis (Figure 46).

CHAPTER V

DISCUSSION

It is imperative that any material which is used in direct contact with living tissue meet certain standards of tissue compatibility in addition to any therapeutic or mechanical benefit which it may provide. In this regard, Harris⁷⁹ reported clinically successful results with the use of Cavit, but acknowledged that there existed no histologic evidence in direct support of his findings. Therefore, this study was undertaken to investigate the histopathologic tissue response to endodontic perforations filled with Cavit.

Since perforation sites are often inaccessible and/or may lead to serious periodontal defects if repaired surgically, a non-surgical approach was utilized in the experimental model. Due to their unfavorable prognosis, perforations were not created in the furcation area. Because the most favorable response to perforation sealing occurs when they are sealed immediately, all were repaired at the same appointment.

While a deliberate attempt was made to confine the Cavit to just beyond the perforation defect, it was soon apparent that extrusion of the material was difficult to avoid since condensation was required in the attempt to gain an adequate seal. Clinically, this usually caused a large overfilling, the effects of which would depend, to a great extent, on the toxicity of the material. Consequently, no

attempt was made to limit the extrusion of the Cavit in hopes of more fully delineating the tissue reactions to the material.

The significance of the present study may be best appreciated by a comparison of the histologic pictures between the experimental and control groups.

When unfilled perforations were located in the coronal one-third of the root near the crevicular 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 and supracrestal gingival fibers. This inflammation was seen to affect adversely the overlying oral sulcular epithelium by stimulating its apical proliferation and effectively causing resorption of the adjacent alveolar bone. There was an attempt by the underlying connective tissue to encapsulate the inflammatory infiltrate, but the overall tissue response seemed to be one of submission to the extensive trauma. The final result was the formation of an advanced periodontal defect with communication to the oral cavity, widespread inflammation and abscess formation in some cases.

A variation in response was noted in the 120 and 180 day animals where three unfilled perforations located in midroot, exhibited a definite healing response. In speculation, it appeared that the overlying bone and periodontal ligament may have represented an

adequate barrier to the destructive inflammatory reaction following the perforation. The integrity of the periodontium was apparently maintained while the inflammation subsided and allowed for a repair attempt by fibrosis. Whether or not repair would be permanent is uncertain and may be an area of future investigations. Clinically, the findings do suggest a more favorable prognosis for those perforations situated away from 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 formation. At the fifteen day interval, the adjacent bone became necrotic and was replaced by a loose connective tissue stroma. At later intervals, this 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 by 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. Following the initial acute response seen at fifteen days, by 30 days a persistent chronic, but low grade inflammation was noted. Accompanying the alteration of inflammation, root and cementum resorptions were observed to be repaired by secondary cementum deposition and, in some cases, by ankylosis. Ankylosis is observed frequently following severe irritation or injury to the

periodontal ligament, such as that occurring with a root perforation, and thus is the probable etiology. Such damage may effectively inhibit regeneration of the periodontal ligament and allow repair by fusion of tooth root with alveolar bone. Persisting ankylosis was noted in the 180 day specimens as well.

At the 120 and 180 day examination periods, five of the filled perforations in the coronal one-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 formation of a periodontal lesion. This contention was supported by the findings that a more severe tissue response was observed following both filled and unfilled perforations in the coronal one-third of the root. The observations revealed that inflammation following coronally placed perforations often resulted in acute inflammation which progressed along the periodontal ligament causing destruction of the gingival fibers and apical migration of the oral sulcular epithelium. It appears that if the epithelial attachment and supracrestal gingival fibers remain intact, there is a change for connective tissue repair and bone reformation adjacent the perforation provided a breakdown of the periodontium does not result from the inflammation following perforation. From an endodontic standpoint, these findings indicate a more promising result for those perforations located in the middle and apical one-third of the root. It would appear then 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. In addition, the size of the perforation, inadvertent infection and/or toxicity of the filling material may have further contributed to the inflammatory response. Since a nonreversible damaged periodontium resulted preventing any regeneration, it is possible that some combination of these conditions was also responsible.

The origin of the proliferating epithelium causing the periodontal lesions in both experimental and control perforations which had failed to heal is also a matter of some controversy. Scott and Symons⁷ stated that oral sulcular epithelium is characterized by a considerable proliferative ability in its capacity for repair and renewal. However, Kakehashi, Stanley and Fitzgerald¹²⁷ inadvertently produced perforations in germfree rats and conjectured that the ensuing inflammation initiated the proliferation of the epithelial rests of Malassez. These proliferating areas of epithelium coalesced to line any debris embedded in the defect, thus forming periodontal pockets and lesions. In the present study, degeneration of the overlying oral sulcular epithelium and downgrowths of epithelial rete pegs indicated that the proliferating epithelium originated from the oral sulcular epithelium. It might then be proposed that the origin of the epithelium may occur from proliferation of the epithelial rests of Malassez, apical migration of oral sulcular epithelium, or a combination of both. Whichever is the case, epithelial proliferation in the present study correspond with a total lack of repair and no regeneration of periodontal fibers or

alveolar bone over the perforated region. Clinically, the formation of a periodontal pocket in association with a perforation would indicate failure necessitating either extraction or root resection.

Radiographically, at the sixty day interval and beyond, instances where the Cavit overfilling had fractured just outside the perforation canal in the tooth were seen. This resulted in segments of the material implanted in the periodontium. Dissolution or attack by the animals' defense mechanisms could have been responsible for these observations. Yet were this the case, disruptions or degradation would have been evident along the entire periphery of the material. Rather it appears that the brittle nature of the material after setting may have allowed for fracture during masticatory movements. In addition, since occlusal reduction was performed, passive eruption of the teeth may have further contributed to the fractures observed. The actual effect of this is somewhat uncertain. The physical presence of a 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 cases of root 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 an endodontic viewpoint, the separation of Cavit may be regarded as less than optimal, but unlikely to affect negatively the repair process as long as a seal of the perforation is maintained.

It is clear that the use of a material with a low inflammatory potential is of particular importance in root perforations where the

contact area between the periodontal tissues and filling material often will be quite large. Since irritating materials place an increased burden on defense mechanisms, the progress of healing and repair could be suppressed. In the present study, Cavit was observed to be encapsulated by a relatively thick fibrous connective tissue capsule. The capsule was surrounded by a low grade, chronic inflammatory response which apparently did not completely impair connective tissue regeneration. Generally, capsule thickness is considered to be correlated to the degree of inflammation induced by a material. On that basis, Cavit would appear to possess a mild to moderate component of inflammatory potential. Apparently, this inflammation is insufficient to cause diffuse necrosis, but did stimulate the proliferation of connective tissue in an effort to wall off and contain the material. The persistent inflammation was often characterized by the presence of multinucleated giant cells in the capsule surrounding the Cavit. This verifies the irritational component caused by Cavit placed in contact with living connective tissue.

Toxicity of a material is dependent not only on its toxic effects per se, but also upon such physical properties as resorbability, solubility in tissue fluids or fragmentation which could influence the inflammatory reactions observed with a material in vivo^{128,129}. In the present investigation, actual disintegration or dissolution of Cavit could not be visualized as a result of its loss in histologic preparation. However, certain observations were evident that may have indicated deterioration of the material. For one, it was apparent in a number of specimens that an ingrowth of chronic inflammatory tissue

was present in the interface between the Cavit and dentinal walls of the perforation canal indicating marginal breakdown of the filling material. Also seen were discontinuities in the collagen capsule surrounding the Cavit which may have represented disruptions in the surface of the filling material. It is then conceivable that in tissue fluids, Cavit may be susceptible to significant degrees of deterioration and/or resorption by the body's defense mechanisms. This phenomenon in part may explain the lasting irritational and inflammatory potential associated with Cavit as noted in the final observation period of this study. Therefore, additional long term investigations are indicated in order to more fully clarify the relative and lasting toxicity of Cavit with respect to its physical properties in living tissue.

As noted previously, the fibrotic repair associated with Cavit is characteristic of scar formation which is composed of collagen bundles, fibrocytes with a few spindle-shaped fibroblasts as viewed histologically. It may be stated that in areas of perforations filled with Cavit, complete return of the tissues to their normal state does not occur. Rather, repair takes place by the formation of fibrotic scar tissue around the filling material. Although radiographically this may appear as a radiolucency around the material, clinically the defect may be considered acceptably healed.

It is of interest to note that Lantz and Perrson⁵⁷ cultured enterococci and coliform rods from unfilled perforations which had

failed to heal. In the present study, the possible role of bacterial contamination was not investigated. However, the presence of extensive inflammation and abscess formation in a number of filled and unfilled perforations strongly alluded to the possibility of infection. It might be postulated that an additional factor in the periodontal destruction observed with unsuccessful filled perforations may be due to secondary infection via the gingival pocket or anachoretic spread. If one assumes that infection can play a role in inhibiting repair, an area of further investigation may be whether or not the use of a systemic antibiotic would improve the prognosis of a filled perforation.

Considerable variations existed in the present study as to the size, depth and proximity to the oral sulcular epithelium. It is recognized that many of these perforations were not consistent with those observed in clinical cases. Although the histologic findings were quite informative, future studies would seem to warrant the development of a method which would produce consistent perforation defects as to size, depth and location similar to those encountered clinically.

In the 120 day animal, epithelial proliferation and subsequent periodontal breakdown occurred in four of the five filled perforations. However, all three unfilled perforations in the same animal revealed evidence of healing. Whether or not these findings are attributable to individual variations with respect to systemic conditions and powers of repair is a matter of conjecture. In any case, a clear deficiency in this study was the limitation of only one observation period per

animal. This may have been handled more effectively by creating experimental and control perforations at different time intervals in each particular animal. This would have reduced the variable of individual dissimilarities and allowed for a survey of the progression of the tissue reaction to the experimental lesions.

Criticism may be directed towards 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 by Lantz and Persson^{57,58,59} discussed at length the tissue reactions to perforations in dogs' teeth filled with gutta-percha. Nevertheless, it is acknowledged that increased validity for the present investigation may have been attained had a comparison of the tissue reactions to both Cavit and gutta-percha been performed.

Because of the limitations on the use of human subjects, animal experimentation for basic research is necessary. In this regard, the dog has been used extensively by investigators as an experimental animal. Barker and Lockett¹³⁰ in 1971 evaluated the mandibular premolars of dogs for endodontic research. They reported that the tissue responses were essentially similar in range to that observed in humans, although canine tissue appeared more sensitive to injury with a decreased capacity for repair. Earlier studies by Matsumiya and Kitamura¹³¹, Synder, Seltzer and Moodnik¹³² and Seltzer et al.¹³³

also revealed similar healing patterns in humans and dogs.

Based on the above studies, it was felt that the dog would serve as an acceptable experimental model for the present investigation. Yet the application of the results of any animal study to man must be performed with a degree of caution. Clafin¹³⁴ and Boyne¹³⁵ observed that healing and tissue regeneration progressed more rapidly in dogs than in humans. Gads¹³⁶, however, noted that periodontal disease progressed at a much faster rate in dogs than in humans. Whether or not there are similar differences with respect to perforations is unknown.

Perforations do occur in endodontics and cause clinical problems. It is imperative to understand the pathogenesis of such misadventures and evaluate methodology for treatment. It is hoped that this paper will expand the endodontic community's knowledge on the subject and delineate areas for further study.

CHAPTER VI

SUMMARY AND CONCLUSIONS

This investigation deals with the study of periodontal tissue reactions to perforations in dogs' teeth filled with Cavit. Thirty-six lower premolar teeth in six beagle hounds were treated endodontically and 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 sacrificed at intervals of one, fifteen, 30, 60, 120 and 180 days. The experimental jaw sections were removed at necropsy and histologic sections prepared using hematoxylin and eosin stain. The sections were studied microscopically and observations recorded.

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

- a) Progressive destruction of the periodontal tissues took place in those perforations left unfilled particularly when located near the oral sulcular epithelium.
- b) When Cavit was used to seal the perforation immediately, fibrous encapsulation occurred.
- c) 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.

- d) Perforations located away from the oral sulcur epithelium displayed the most favorable healing response even when left unfilled.
- e) Root and cementum resorption always occurred with perforations and were repaired only in successfully filled perforations.
- f) Variations in healing ability can modify expected results.

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

- a) The use of a filling material with a low inflammatory potential is definitely indicated in light of the frequently observed overfilling during the repair of perforations.
- b) Cavit appears to possess a mild to moderate inflammatory potential. Further studies are required to clarify this property as well as solubility of the material under conditions similar to those encountered in the periodontium.
- c) Varied experimental conditions similar to those encountered clinically should also be evaluated. These might include: perforations contaminated by the oral environment, where some time interval has elapsed before an attempt to repair the defect, and small accessible perforations of the furcation area.

CHAPTER VII

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TABLE VIII

TABLES AND FIGURES

ABBREVIATIONS FOR TABLES

CT	-	Connective Tissue
PDL	-	Periodontal Ligament
ECP	-	Encapsulation
PMN	-	Polymorphonuclear Leukocytes
VSD	-	Vasodilation
OCA	-	Osteoclastic Activity
OBA	-	Osteoblastic Activity
FBA	-	Fibroblastic Activity
SGF	-	Supracrestal Gingival Fibers
ETP	-	Epithelial Proliferation
GT	-	Granulation Tissue

TABLE I

EXPERIMENTAL DESIGN

	Mandibular Right Side						Mandibular Left Side					
	Third Premolar		Second Premolar		First Premolar		First Premolar		Second Premolar		Third Premolar	
	D	M	D	M	D	M	M	D	M	D	M	D
1 Day Animal	C	U	E	E	U	C	E	U	E	E	U	E
15 Day Animal	E	U	E	E	U	C	E	U	C	E	U	E
30 Day Animal	E	U	E	E	U	C	E	U	C	E	U	C
60 Day Animal	E	U	E	E	U	C	E	U	C	C	U	E
120 Day Animal	E	U	C	E	U	E	E	U	C	E	U	E
180 Day Animal	E	U	E	C	U	C	C	U	E	E	U	C

LEGEND

D - Distal Root Side
 M - Mesial Root Side
 E - Experimental Perforation Filled with Cavit
 C - Perforation Left Unfilled
 U - Untreated

TABLE II(a)

COMPARISON OF PERIODONTAL POCKET DEPTHS
FOR FILLED AND UNFILLED PERFORATIONS SITES
INITIALLY AND AT SACRIFICE

Animal	Experimental Site	Treatment	Initial	At Sacrifice
1 Day Dog	LR4 - distal	C	3	3
	LR3 - distal	F	2	2
	LR3 - distal	F	2	2
	LR2 - mesial	C	2	2
	LL2 - mesial	F	2	2
	LL3 - mesial	F	2	2
	LL3 - distal	F	3	3
	LL4 - distal	C	3	3
15 Day Dog	LR4 - distal	F	3	3
	LR3 - distal	F	2	2
	LR3 - mesial	F	2	2
	LR2 - mesial	C	2	6
	LL2 - mesial	F	2	3
	LL3 - mesial	C	2	3
	LL3 - distal	F	3	3
	LL4 - distal	F	2	3
30 Day Dog	LR4 - distal	F	3	3
	LR3 - distal	F	3	3
	LR3 - mesial	F	2	2
	LR2 - mesial	C	2	4
	LL2 - mesial	F	2	3
	LL3 - mesial	C	3	4
	LL3 - distal	F	3	3
	LL4 - distal	C	3	3

TABLE II(b)

COMPARISON OF PERIODONTAL POCKET DEPTHS
FOR FILLED AND UNFILLED PERFORATIONS SITES
INITIALLY AND AT SACRIFICE

Animal	Experimental Site	Treatment	Initial	At Sacrifice
60 Day Dog	LR4 - distal	F	3	3
	LR3 - distal	F	2	2
	LR3 - mesial	F	3	6
	LR2 - mesial	C	3	5
	LL2 - mesial	F	3	4
	LL3 - mesial	C	2	6
	LL3 - distal	C	3	7
	LL4 - distal	F	3	4
120 Day Dog	LR4 - distal	F	3	6
	LR3 - distal	C	3	4
	LR3 - mesial	F	2	4
	LR2 - mesial	F	2	4
	LL2 - mesial	F	2	3
	LL2 - mesial	C	3	6
	LL3 - distal	F	3	3
	LL4 - distal	C	3	4
180 Day Dog	LR4 - distal	F	3	4
	LR3 - distal	F	2	2
	LR3 - mesial	C	2	6
	LR2 - mesial	F	2	5
	LL2 - mesial	C	2	7
	LL3 - mesial	F	2	3
	LL3 - distal	F	2	3
	LL4 - distal	C	3	3

LEGEND: F - Filled Perforation
C - Perforation Left Unfilled

LR4 - distal - lower right fourth premolar, distal root
LR3 - distal - lower right third premolar, distal root
LR3 - mesial - lower right third premolar, mesial root
LR2 - mesial - lower right second premolar, mesial root
LL2 - mesial - lower left second premolar, mesial root
LL3 - mesial - lower left third premolar, mesial root
LL3 - distal - lower left third premolar, distal root
LL4 - distal - lower left fourth premolar, distal root

TABLE III
1 DAY SPECIMENS

EXPERIMENTAL SITE TREATMENT LOCATION	INFLAMMATORY RESPONSE	BONE RESPONSE	CONNECTIVE TISSUE, PDL REACTION	ROOT AND CEMENTUM RESORPTION	EPITHELIAL REACTION
LR4-Distal Unfilled Coronal	Margination of PMN's, some VSD	Osteocytes lost in ad- jacent bone	Disruption of PDL	None	None
LR3-Distal Filled Midroot	Margination of PMN's, some VSD	Osteocytes lost in ad- jacent bone	Marked dis- ruption of PDL	None	None
LR3-Mesial Filled Midroot	Margination of PMN's, some VSD	Osteocytes lost in ad- jacent bone	Disruption of PDL	None	None
LR2-Mesial Unfilled Coronal	Margination of PMN's, some VSD	Osteocytes lost; no activity	Disruption of PDL	None	None
LL2-Mesial Filled Coronal	Margination of PMN's	Osteocytes lost in ad- jacent bone	Disruption of PDL	None	None
LL3-Mesial Filled Coronal	Margination of white blood cells	Osteocytes lost in ad- jacent bone	Disruption of PDL, lots of VSD	None	None
LL3-Distal Filled Mid-root	Margination of PMN's, some VSD	Osteocytes lost in ad- jacent bone	Marked dis- ruption of PDL	None	None
LL4-Distal Filled Coronal	Margination of PMN's	Osteocytes lost in ad- jacent bone	Disruption of PDL	None	None

TABLE IV
15 DAY SPECIMENS

EXPERIMENTAL SITE TREATMENT LOCATION	INFLAMMATORY RESPONSE	BONE RESPONSE	CONNECTIVE TISSUE, PDL REACTION	ROOT AND CEMENTUM RESORPTION	EPITHELIAL REACTION
LR4-Distal Filled Midroot	Acute in bone; chronic in PDL	Osteocytes lost; mini- mal activity	Disruption of PDL	None	None
LR3-Distal Filled Coronal	Acute - mild	Osteocytes lost; mini- mal activity	Disruption of PDL	None	None
LR3-Mesial Filled Midroot	Acute - very mild	Osteocytes lost; some remodeling	Disruption of PDL, hyalinization	None	None
LR2-Mesial Unfilled Midroot	Acute - very severe response	Marked OCA	PDL and SGF degeneration	Active with no repair	None
LL2-Mesial Filled Midroot	Acute - low grade response	Basophilic staining; some OCA	Disruption of PDL, hyalinization	None	None
LL3-Mesial Unfilled Midroot	Acute - very severe response	Osteocytes lost, mini- mal activity	Severe degeneration of PDL	Active with no repair	Acute inflam- ed, ETP noted
LL3-Distal Filled Midroot	Acute - very mild	Osteocytes lost; mini- mal activity	Disruption of PDL, hyalinization	None	None
LL4-Distal Filled Coronal	Mixed acute and chronic response	Basophilic staining, some OCA	PDL degene- ration, very inflamed	None	None

TABLE V
30 DAY SPECIMENS

EXPERIMENTAL SITE TREATMENT LOCATION	INFLAMMATORY RESPONSE	BONE RESPONSE	CONNECTIVE TISSUE, PDL REACTION	ROOT AND CEMENTUM RESORPTION	EPITHELIAL REACTION
LR4-Distal Filled Coronal	Chronic- moderate	Remodeling	Marked FBA adjacent Cavit	Active, some repair seen	None
LR3-Distal Filled Midroot	Chronic- moderate	Remodeling	Disorgani- zation of PDL, ECP	Active, some repair seen	None
LR3-Mesial Filled Midroot	Chronic- very mild	OCA with remodeling externally	Widened PDL Loose CT stroma	Active, signs of repair seen	None
LR2-Mesial Unfilled Coronal	Mixed acute and chronic	OCA with remodeling externally	FBA to limit defect	Active, no repair seen	ETP and degeneration
LL2-Mesial Filled Coronal	Chronic- mild	Remodeling	Widened PDL Loose CT stroma	Active, no repair seen	None
LL3-Mesial Unfilled Coronal	Acute and intense; VSD	Active bone reformation	PDL lost, SGF degene- ration	Active, no repair	None
LL3-Distal Filled Apical	Chronic - mild	OCA near Cavit	PDL disor- ganization, FBA	Active, some repair noted	None
LL4-Distal Unfilled Coronal	Acute and intense	Remodeling	PDL lost, loose CT stroma	Active, no repair seen	None

TABLE VI
60 DAY SPECIMENS

EXPERIMENTAL SITE TREATMENT LOCATION	INFLAMMATORY RESPONSE	BONE RESPONSE	CONNECTIVE TISSUE, PDL REACTION	ROOT AND CEMENTUM RESORPTION	EPITHELIAL REACTION
LR4-Distal Filled Midroot	Chronic - very mild	Remodeling	PDL regene- ration, defi- nite ECP	Repair by cementoid deposition	None
LR3-Distal Filled Coronal	Chronic - mild with VSD	Remodeling	PDL regene- ration, defi- nite ECP	Active with some signs of repair	None
LR3-Mesial Filled Coronal	Chronic - mild lots of VSD	Resorption of adjacent bone	ECP attempt PDL lost in- cisally	Repair by cementoid deposition	Beginning signs of ETP
LR2-Mesial Unfilled Coronal	Chronic - very severe	Resorption of adjacent bone	PDL lost, replaced by loose CT	Active, no repair seen	ETP and degeneration
LL2-Mesial Filled Coronal	Chronic - moderate	Bone reformation	FBA, ECP	Active, no repair seen	Intact with signs of degeneration
LL3-Mesial Unfilled Midroot	Severe - mixed acute and chronic	Some bone reformation	Degeneration of PDL	Active, no repair seen	Apical ETP
LL3-Distal Unfilled Midroot	Severe - mixed acute and chronic	Some bone reformation	PDL lost, weak ECP attempt	Active, no repair seen	Apical ETP
LL4-Distal Filled Midroot	Chronic - mild	Lots of OBA	PDL regeneration, ECP	Repair by cementoid deposition	None

TABLE VII
120 DAY SPECIMENS

EXPERIMENTAL SITE TREATMENT LOCATION	INFLAMMATORY RESPONSE	BONE RESPONSE	CONNECTIVE TISSUE, PDL REACTION	ROOT AND CEMENTUM RESORPTION	EPITHELIAL REACTION
LR4-Distal Filled Coronal	Chronic - mild with VSD and plasma cells	Active bone reformation	Poor ECP at- tempt; PDL disoriented	Repair by cementoid deposition	Downgrowth of rete pegs
LR3-Distal Unfilled Midroot	Chronic - mild	Active bone reformation	CT ingrowth in perfora- tion canal	Repair by cementoid deposition	Intact
LR3-Mesial Filled Coronal	Chronic - mild	Bone remodeling	ECP attempt PDL disor- ganized	No repair, oral commui- cation	Proliferation around Cavit
LR2-Mesial Filled Coronal	Chronic - mild	Bone reformation	PDL dis- oriented, FBA	No repair, oral commui- cation	Proliferation around Cavit
LL2-Mesial Filled Coronal	Chronic - mild	Bone remodeling	ECP Attempt PDL disorien- ted	Little repair seen	Proliferation around Cavit
LL3-Mesial Unfilled Coronal	Chronic - moderate	Bone remodeling	Weak ECP attempt	Osteoid and cementum repair	Intact
LL3-Distal Filled Midroot	Chronic - mild	Bone remodeling	Heavy ECP, PDL disorien- ted	Osteoid and cementum repair	Intact
LL4-Distal Unfilled Coronal	Chronic - moderate	Bone remodeling	PDL and SGF degeneration	Repair by cementoid deposition	Degeneration changes noted

TABLE VIII
180 DAY SPECIMENS

EXPERIMENTAL SITE TREATMENT LOCATION	INFLAMMATORY RESPONSE	BONE RESPONSE	CONNECTIVE TISSUE, PDL REACTION	ROOT AND CEMENTUM RESORPTION	EPITHELIAL REACTION
LR4-Distal Filled Apical	Chronic - moderate	Bone reformation	ECP, PDL disoriented	Repair by cementoid disposition	None
LR3-Distal Filled Midroot	Chronic - mild	Bone remodeling	ECP, PDL disoriented	Repair by cementoid disposition	None
LR3-Mesial Unfilled Coronal	Chronic - intense	Apical bone remodeling	PDL and SGF lost	Active with little repair	ETP to below defect
LR2-Mesial Filled Coronal	Chronic - intense	Apical bone remodeling	PDL lost, attempt at ECP	Repair apically	ETP and degeneration
LL2-Mesial Unfilled Midroot	Chronic - intense, VSD	Continued OCA	PDL lost, replaced by GT	Active with little repair	Apical ETP
LL3-Mesial Filled Midroot	Chronic - moderate	Bone remodeling	ECP, PDL disoriented	Repair by cementoid deposition	None
LL3-Distal Filled Midroot	Chronic - moderate	Bone remodeling	ECP, PDL disoriented	Repair by cementoid deposition	None
LL4-Distal Unfilled Coronal	Chronic - moderate	Predominate OBA	Attempt at ECP	Active, some repair noted	None

TABLE IX

SUMMARY OF HISTOLOGIC FINDINGS

ANIMAL	PERFORATIONS FILLED WITH CAVIT	PERFORATIONS LEFT UNFILLED
1 Day Dog	Identical tissue responses at this interval. Minimal inflammatory reactions; vasodilation and margination of neutrophilic leukocytes in blood vessels. Osteocytes absent from Howship's lacunae with severe disruption of the periodontal ligament. Extravasation of red blood cells and hemosiderin seen in the unfilled perforations. Necrotic and amorphous non-cellular debris observed lining the Cavit in the filled perforation.	
15 Day Dog	Inflammation ranged from mild acute to a mixed acute and chronic reaction. The alveolar around the Cavit stained basophilic with scattered areas of osteoclastic activity. The supracrestal gingival fibers and periodontal ligament demonstrated signs of disorganization due to spread of inflammation incisally. No root resorption seen.	Inflammation was acute and of an intense nature; vasodilation and extravasated red blood cells seen. Degenerative changes in the periodontal ligament due to extension of inflammation incisally. Osteoclastic activity now evident as well as root and cementum resorption.
30 Day Dog	A mild to moderate chronic inflammation existed. Alveolar bone around the Cavit had been resorbed with osteoclastic activity still present. Bone remodeling noted peripherally. Loose connective tissue with fibroblastic activity had replaced the resorbed bone. Root resorption was seen with areas of cementoid and osteoid deposition.	Chronic inflammation of periodontal ligament with beginning degeneration of gingival fibers. Resorption of adjacent bone with continued osteoclastic activity. One specimen revealed mixed acute and chronic inflammation with epithelial proliferation. Fibrotic attempt at capsule formation and active root resorption.

TABLE IX (Continued)

ANIMAL	PERFORATIONS FILLED WITH CAVIT	PERFORATIONS LEFT UNFILLED
60 Day Dog	Two specimens revealed epithelial proliferation with loss of the supracrestal fibers and alveolar crestal bone. Three specimens exhibited fibrotic encapsulation of the Cavit with bone reformation and regeneration of the periodontal ligament. Root and cementum resorption still active, but with signs of repair noted.	Marked inflammation; acute near defect, chronic more peripherally. Progressive degeneration of the periodontal ligament, spread of inflammation incisally and proliferative changes of the epithelium. Some degeneration of gingival fibers. Resorption of adjacent bone replaced by loose connective tissue. Active root and cementum resorption with no repair.
120 Day Dog	Three cases revealed epithelial proliferation around the Cavit. A partially filled specimen displayed an ingrowth of granulation tissue and cementoid deposition along the perforation canal. Slight inflammation was seen with degenerative changes in the epithelium. A well-filled perforation exhibited a heavy connective tissue encapsulation; minimal inflammation, bone reformation.	Low grade inflammatory response with active bone reformation and fibroblastic activity. Neither epithelial proliferation or extensive bone destruction had occurred. A loose connective tissue stroma had replaced the resorbed bone and displayed evidence of fibrotic repair. Repair of root resorption was also noted.
180 Day Dog	Proliferation of epithelium occurred in one case resulting in a periodontal pocket. Four remaining specimens showed repair by fibrotic tissue encapsulation with no signs of epithelial proliferation or degeneration. Inflammation was chronic though mild. Repair of root resorption by cementoid deposition was also evident.	Inflammation was mild and chronic in nature. Epithelium had proliferated almost to the apices of the involved teeth forming deep periodontal pockets and abscess formation. One defect revealed some repair attempt by fibrosis. Bone remodeling was evident peripherally with repair of root resorption.

- Figure 1: A. Mandibular molar treated eight months previously. Patient presented with pain and swelling.
- B. Cause of failure revealed by instrument in perforation in distal aspect of mesial root.

Figure 2: Perforation created with an engine driven bur during removal of gutta-percha in preparation of post room.



C. The middle portion of the root.



Figure 3: Illustration of a method for classification of root perforation areas. The different areas are as given.

- A. The coronal portion of the root under the marginal bone level.
- B. The furcation area and 2mm apically.
- C. The middle portion of the root.
- D. The apical portion of the root.

(Stromberg, T., Hasselgren, G. and Bergstedt, H.: Endodontic treatment of traumatic root perforations in man. Swed. Dent. J. 65, 457-466, 1972.)

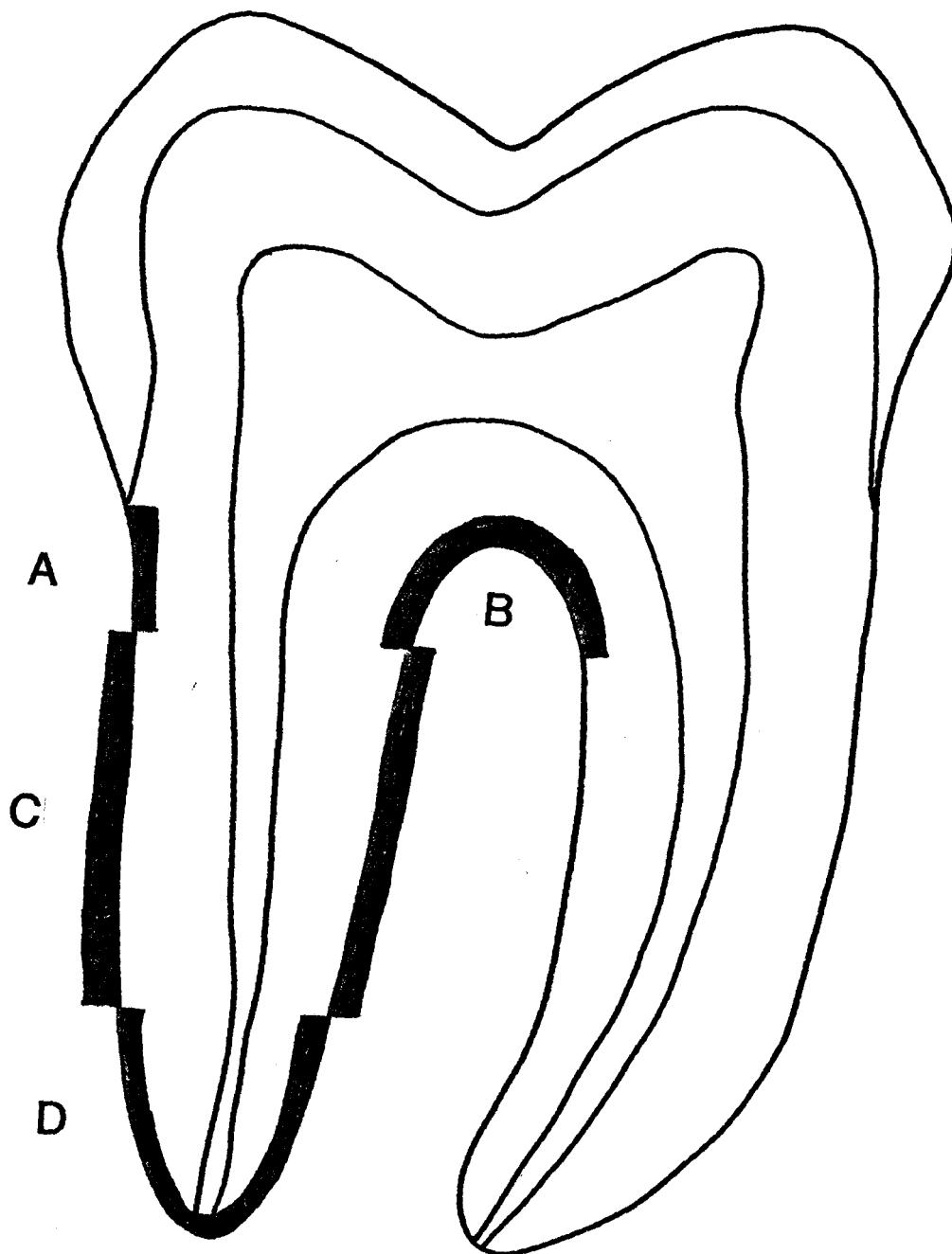
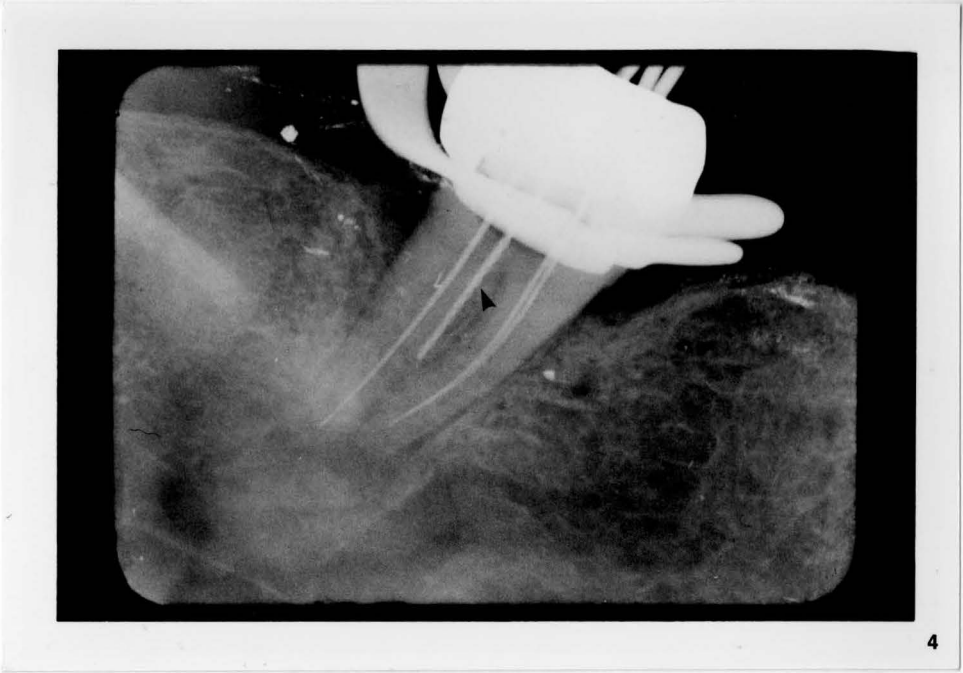


Figure 4: Radiograph of a mandibular molar with instruments placed in the root canals and into a perforation (ARROW) of the floor of the pulp chamber.

Figure 5: Radiograph of an upper bicuspid with an instrument in a perforation and demonstrating deviation of the instrument from the long axis of the tooth.



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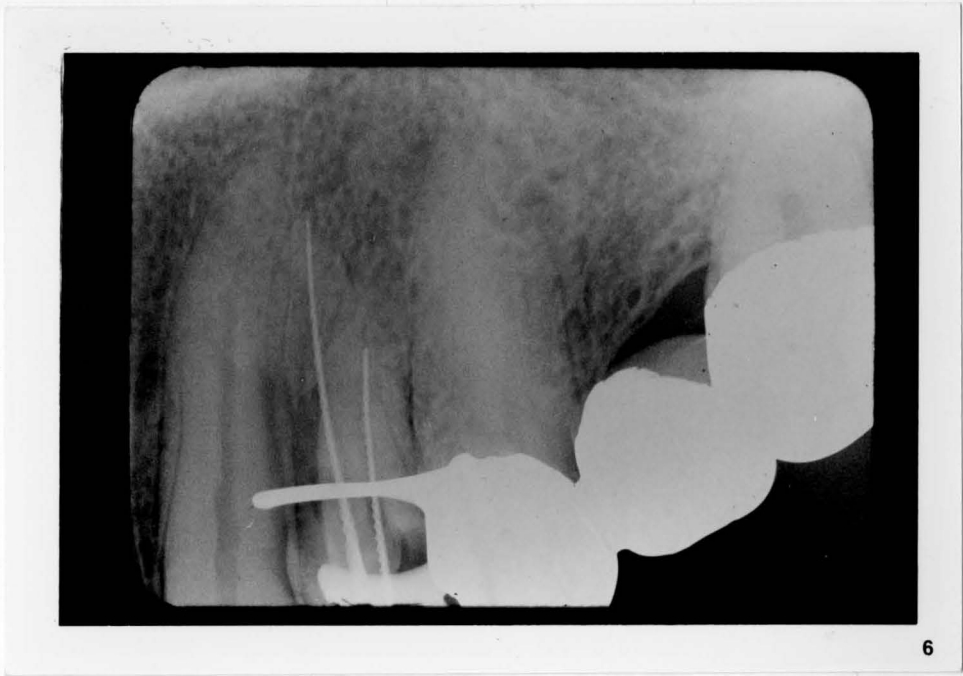


Figure 71

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Figure 6: Angled radiograph of a buccal perforation of an upper bicuspid. The defect could not be visualized on a straight-on view.

Figure 7: Typical preoperative radiographic survey. Note the level of the crest of the interdental septum. Second premolar (2), third premolar (3), fourth premolar (4).



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Figure 6. Diagrammatic illustration of experimental method.



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Figure 8: Diagrammatic illustration of experimental method.

LEGEND

- AMALGAM
- TEMPORARY STOPPINGS
- COTTON
- GUTTA-PERCHA
- CAVIT

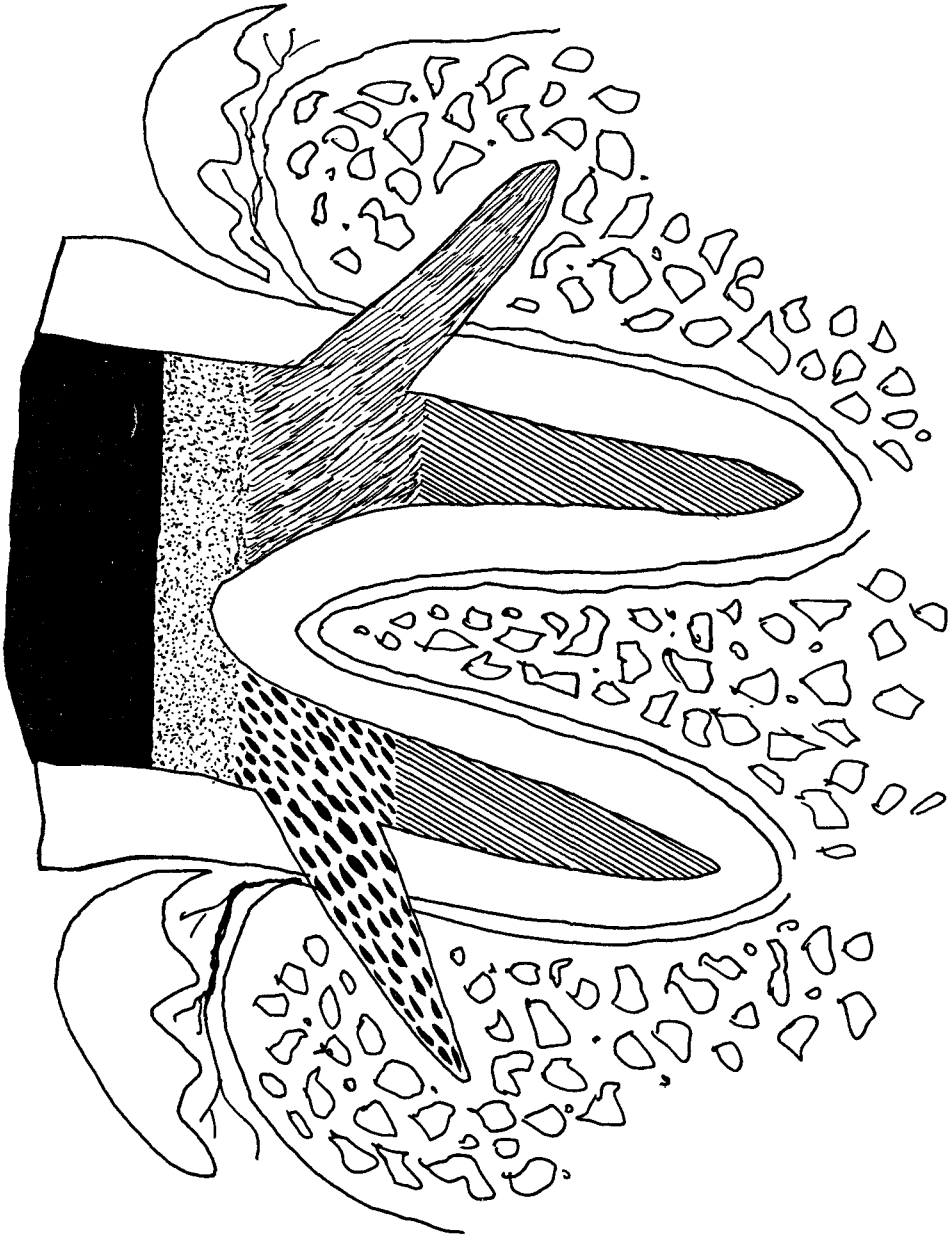


Figure 9: Radiographic survey of one day animal at sacrifice. Note overfillings of perforations with Cavit (ARROWS).

Figure 10: Radiographic survey of fifteen day animal at sacrifice.

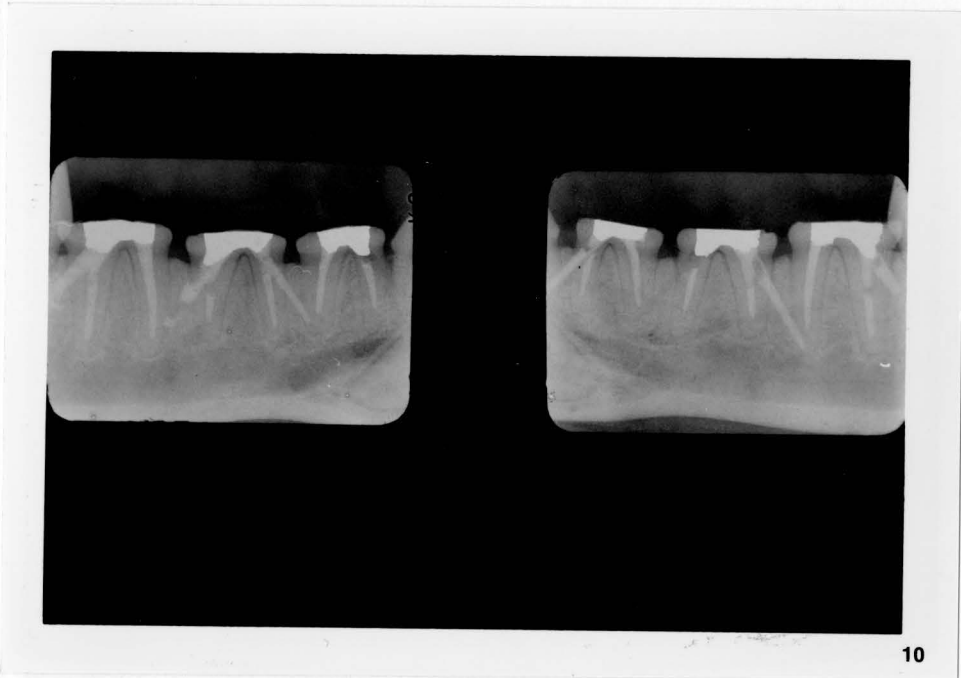
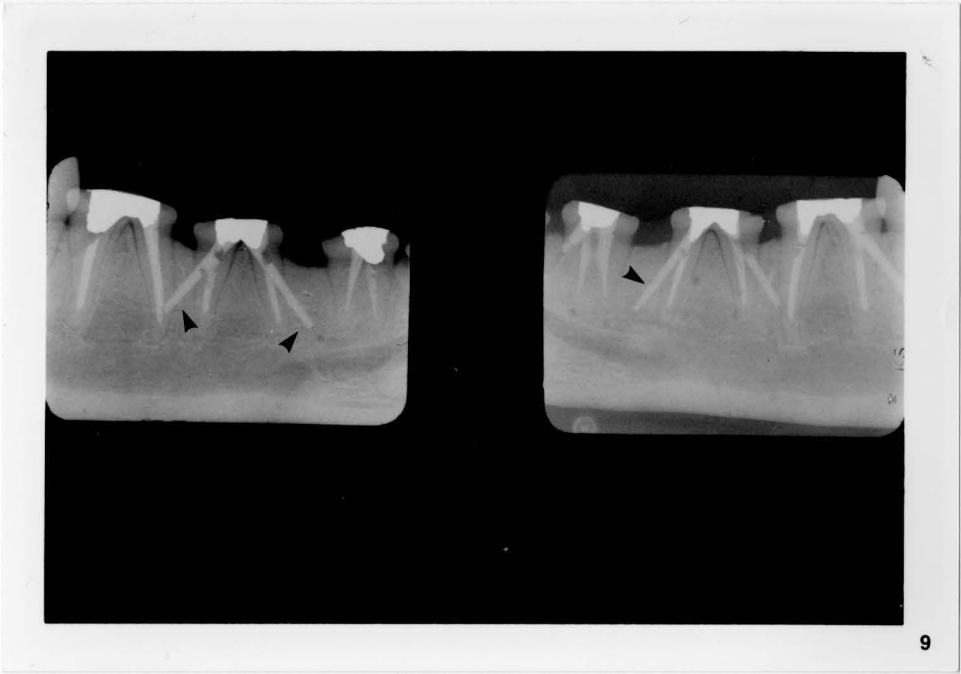
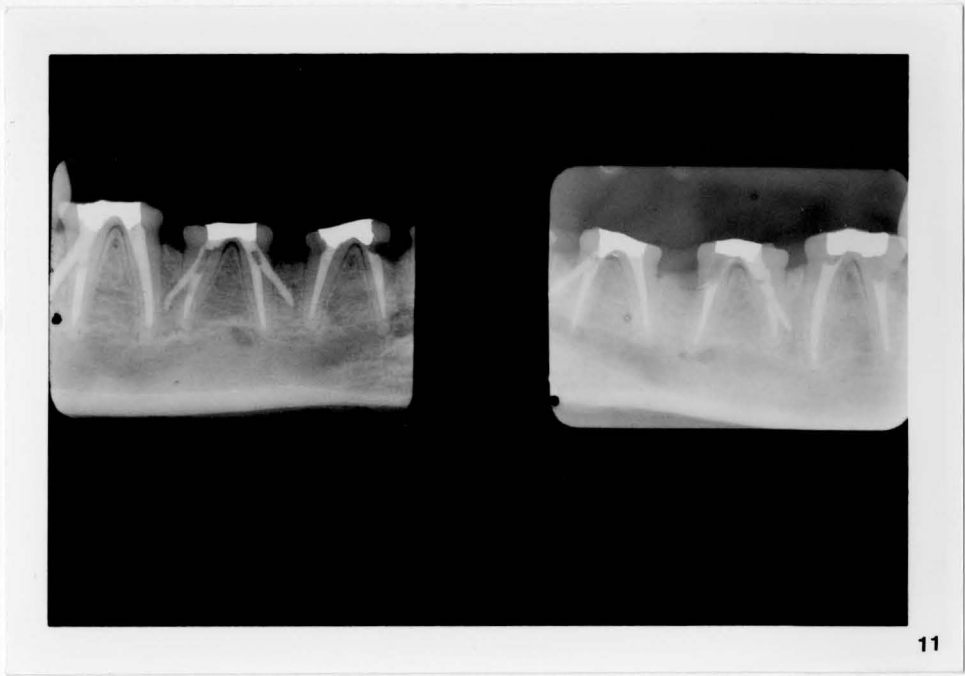
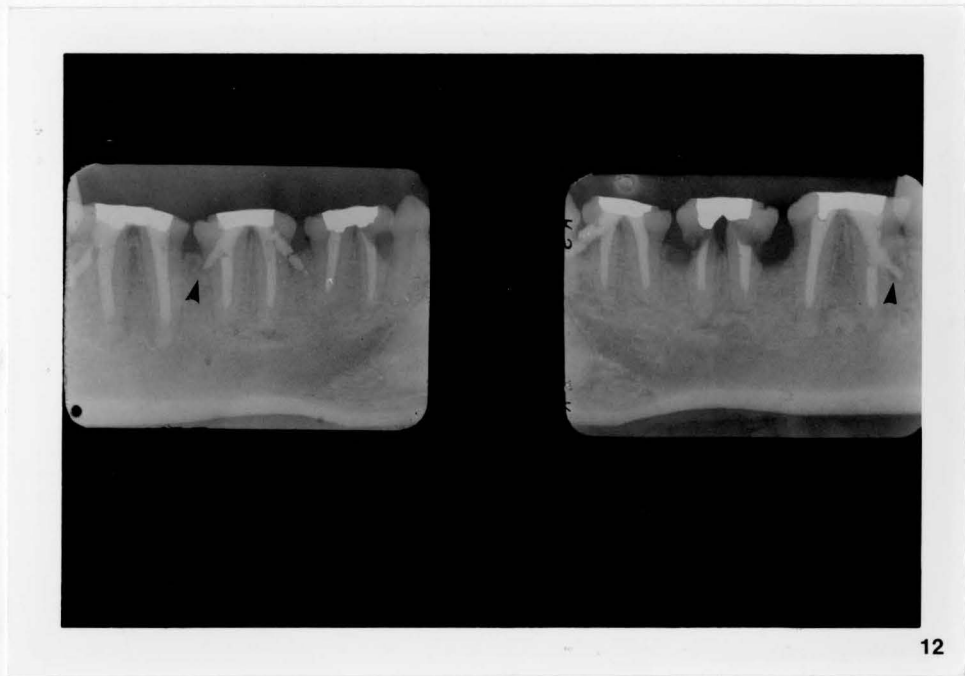


Figure 11: Radiographic survey of thirty day animal at sacrifice.

Figure 12: Radiographic survey of 60 day animal at sacrifice. Note separation of Cavit and thin radiolucent line around excess filling material (ARROWS).



11



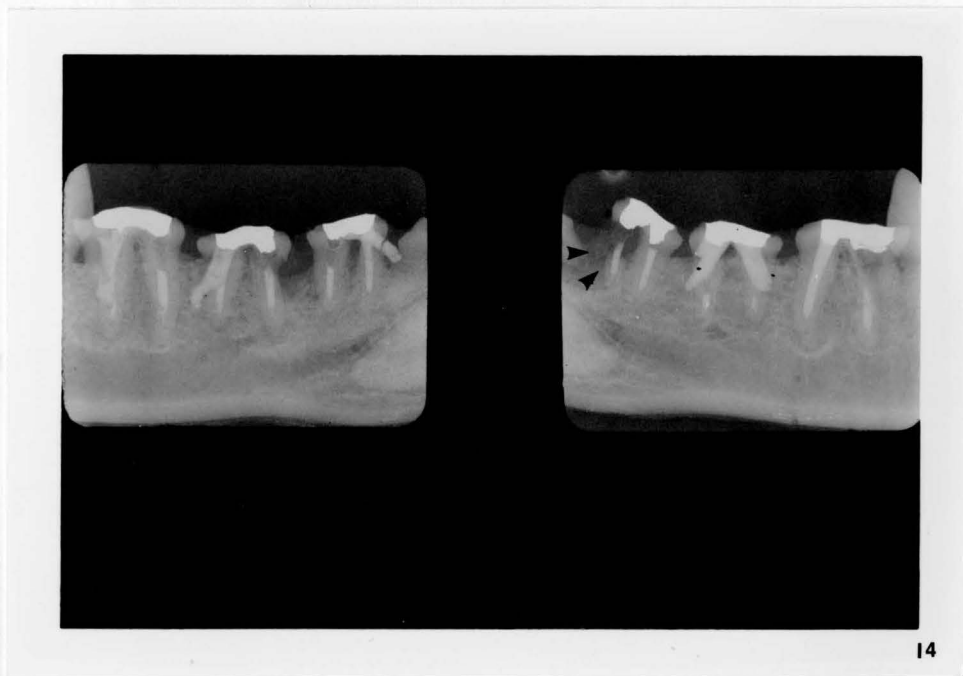
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Figure 13: Radiographic survey of 120 day animal at sacrifice.

Figure 14: Radiographic survey of 180 day animal. Note severe loss of alveolar bone and near exfoliation of tooth with unfilled perforation (ARROWS).



13



14

Figure 15: Normal stratified squamous epithelium (E).
Note slight infiltration of plasma cells,
(Hematoxylin and eosin stain, original
magnification X40.)

Figure 16: Epithelial attachment (E), supracrestal
gingival fibers (G) and alveolar bone (B)
in an area unrelated to a perforation.
Dentin (D). (Hematoxylin and eosin stain,
original magnification X40.)

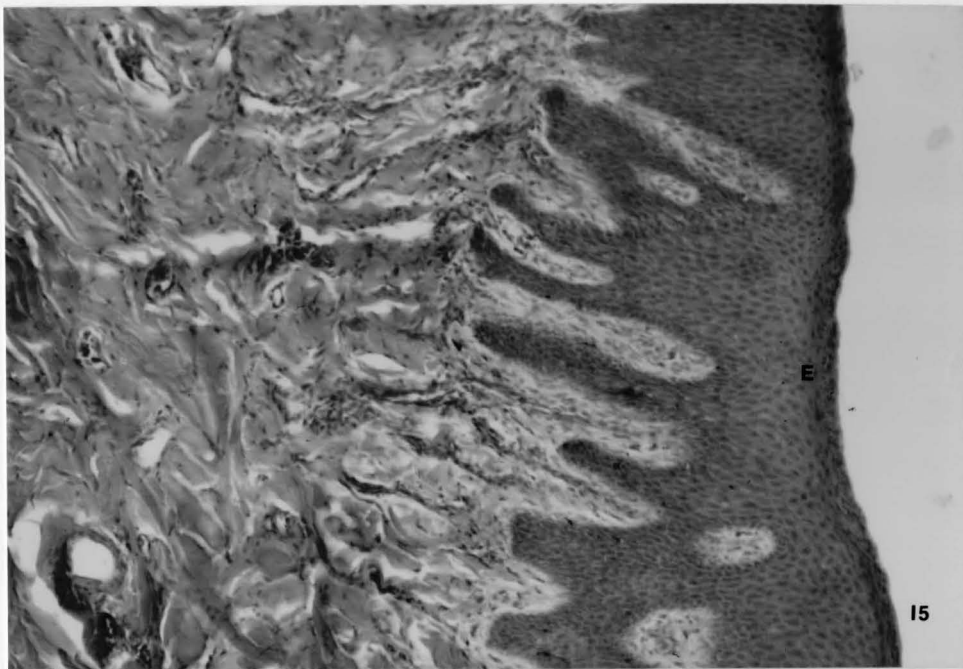


Figure 18:

Figure 17: Normal periodontal ligament (P) with collagen fibers running obliquely and perpendicularly to cementum (C) and alveolar bone (A). Note presence of bundle bone (B). (Hematoxylin and eosin stain, original magnification X40.)

Figure 18: Alveolar bone adjacent to unfilled perforation at one day. Note increased vascularity of blood vessels (B) and margination of white blood cells (ARROWS). (Hematoxylin and eosin stain, original magnification X100.)

Figure

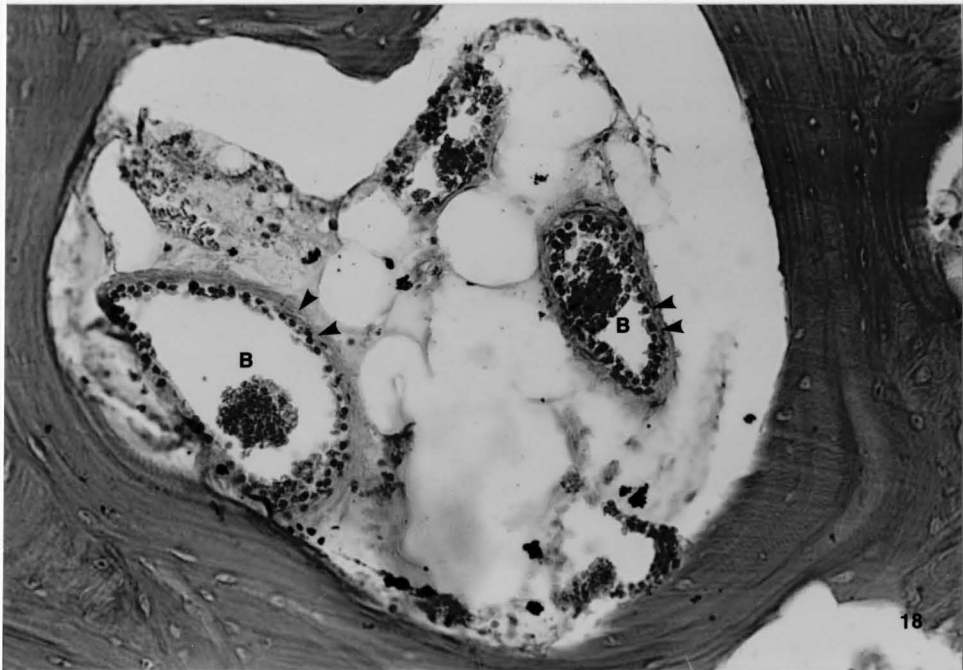
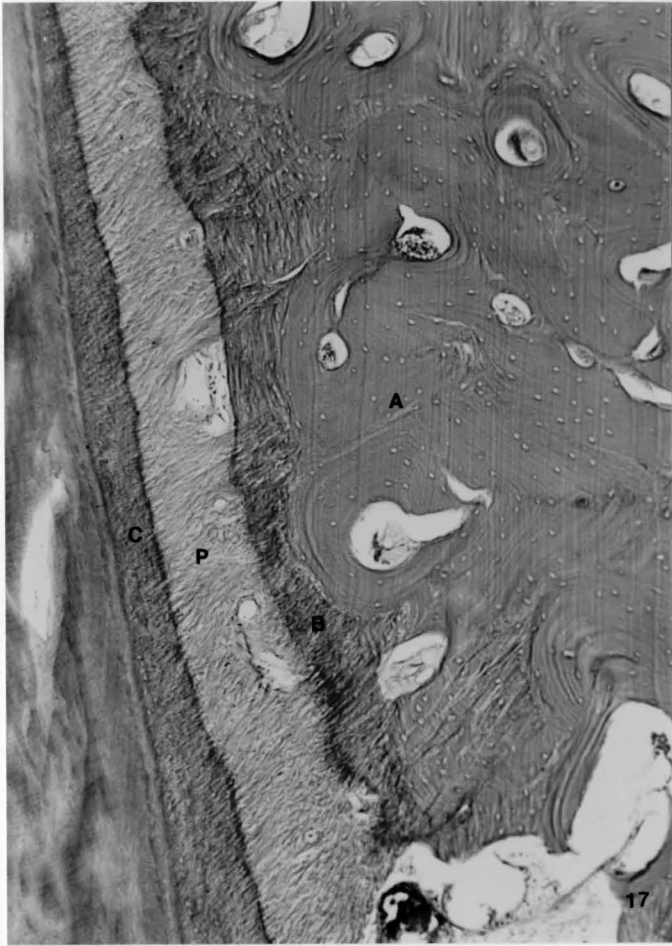


Figure 19: Unfilled perforation at one day. Osteocytes are absent from Howship's lacunae. Signs of inflammation and osseous activity are minimal perforation (P), dentin (D), hemosiderin (ARROWS). (Hematoxylin and eosin stain, original magnification X40.)

Figure 20: Unfilled perforation in fifteen day animal. Disruption of the periodontal ligament (P) by spread of inflammatory infiltrate (INF) is evident. Necrotic debris and inflammatory cells are noted in the perforation canal (C). Alveolar bone (B), dentin (D). (Hematoxylin and eosin stain, original magnification X40.)

Figure

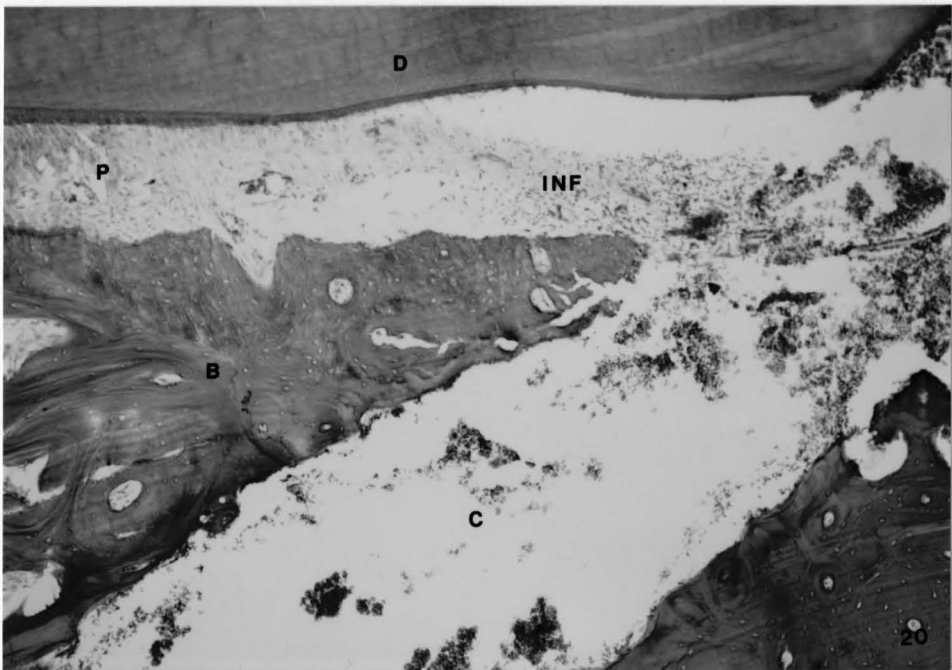
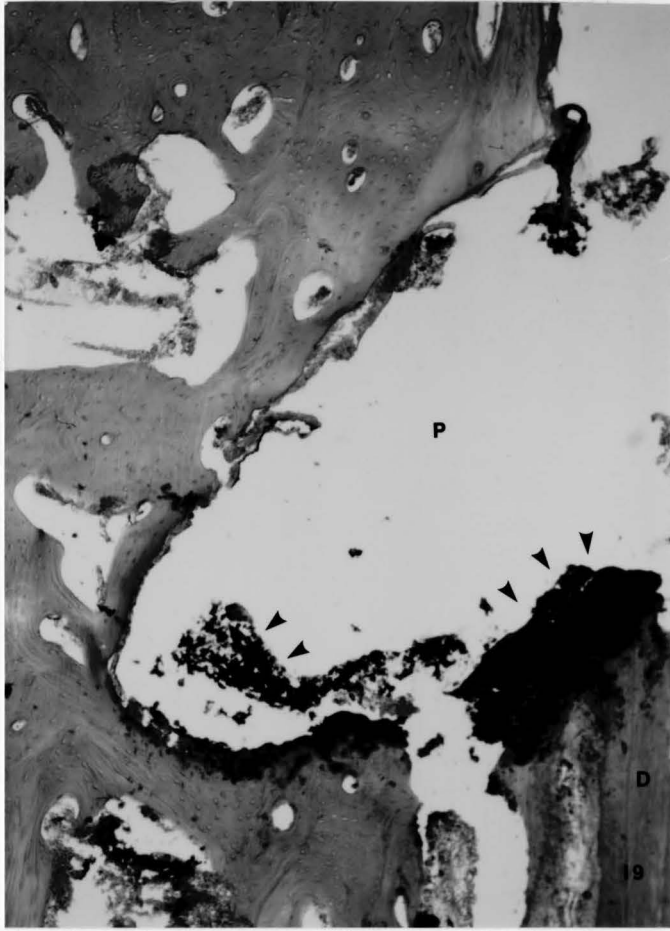


Figure 21: Unfilled perforation in 30 day animal. The adjacent bone has been resorbed and replaced by an acute inflammatory infiltrate (I). Dentin (D), perforation (P), hemosiderin (ARROWS). (Hematoxylin and eosin stain, original magnification X40.)

Figure 22: Unfilled perforation at 30 days revealing proliferation and degeneration of epithelium (E) and destruction of the periodontal ligament (P) and the supracrestal gingival fibers (S). Note spread of inflammatory cells (INF) along periodontal membrane space. (Hematoxylin and eosin stain, original magnification X40.)

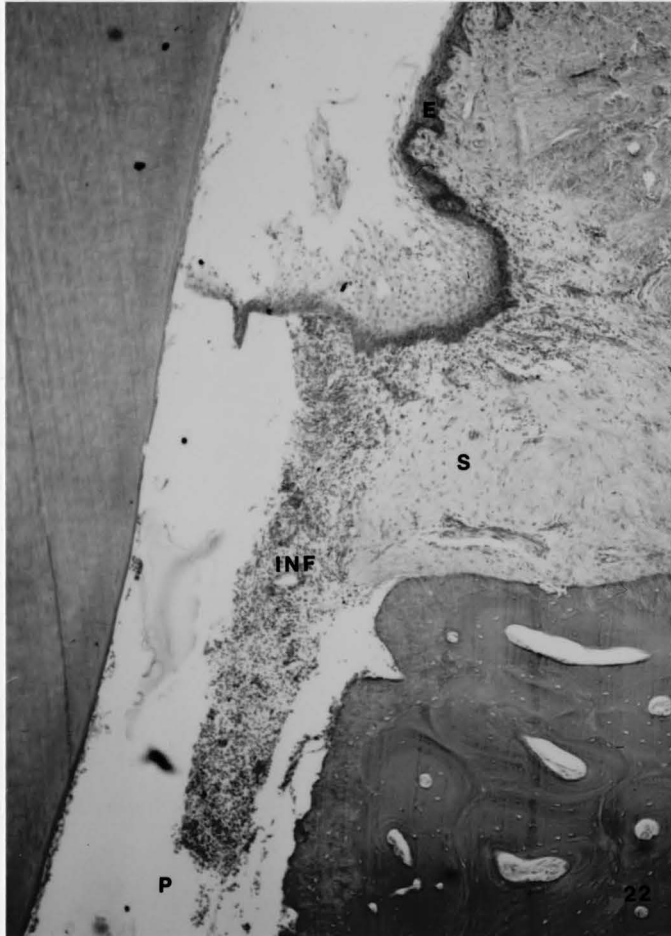
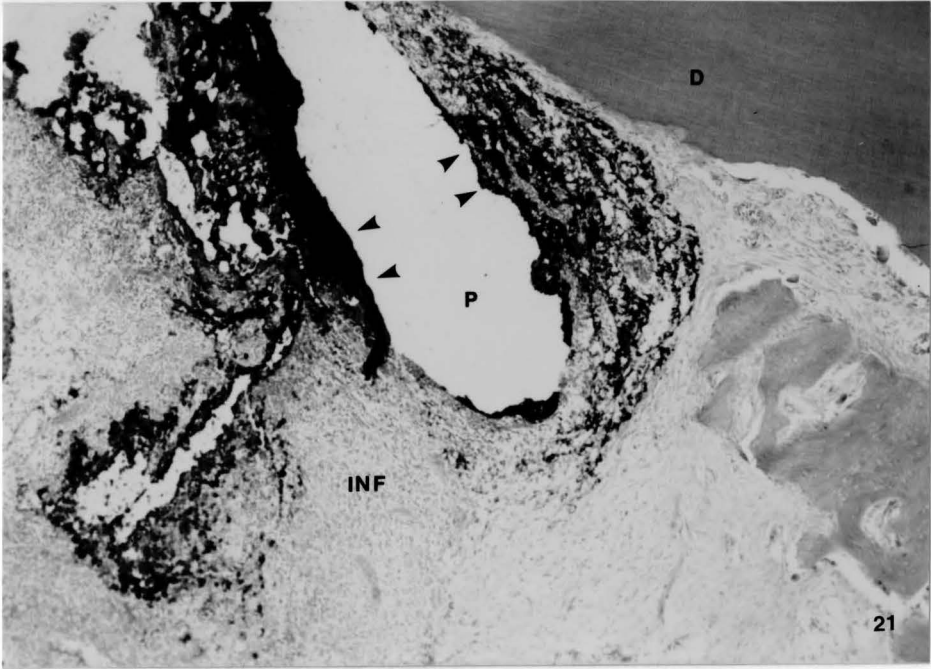


Figure 21

Figure 23: Unfilled perforation in 60 day animal. Observe a dense inflammatory infiltrate (INF) adjacent the defect (D) while chronic inflammatory tissue (C) has replaced the resorbed alveolar bone. The periodontal ligament (P) shows continued degeneration and the alveolar bone evidence of remodeling (ARROWS). (Hematoxylin and eosin stain, original magnification X64.)

Figure 24: Ingrowth of granulation tissue (G) in unfilled perforation canal (C) in root of the 60 day animal. Dentin (D). (Hematoxylin and eosin stain, original magnification X40.)

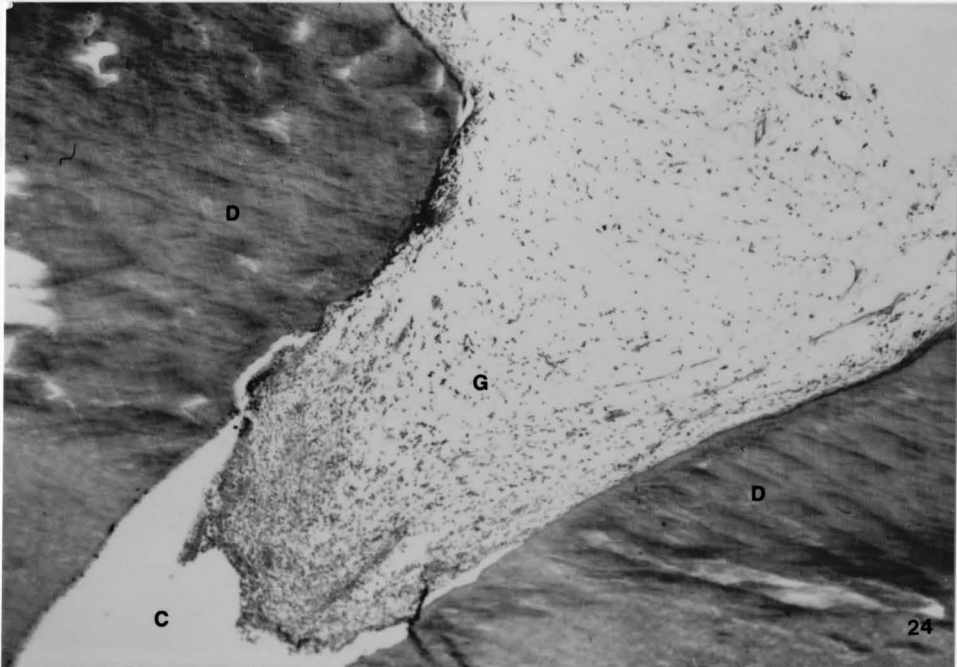
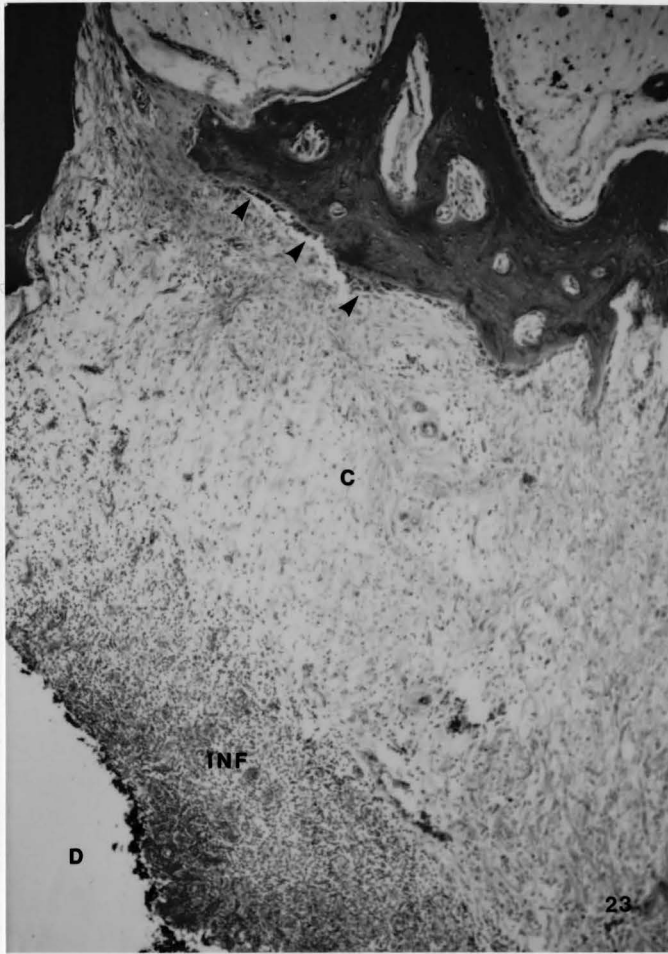


Figure 25: Areas of dentin (D) and cementum (C) resorption apical to an unfilled perforation in the sixty day animal. The spread of chronic inflammatory cells (INF) along the periodontal ligament is also evident. (Hematoxylin and eosin stain, original magnification X160).

Figure 26: Surface root resorption (ARROWS) in the sixty day animal adjacent an unfilled perforation. Cementoclast (CC), dentin (D), cementum (C). Note partial repair by cementoblasts (CB) and regeneration of connective tissue by fibroblasts (F). (Hematoxylin and eosin stain, original magnification x425).

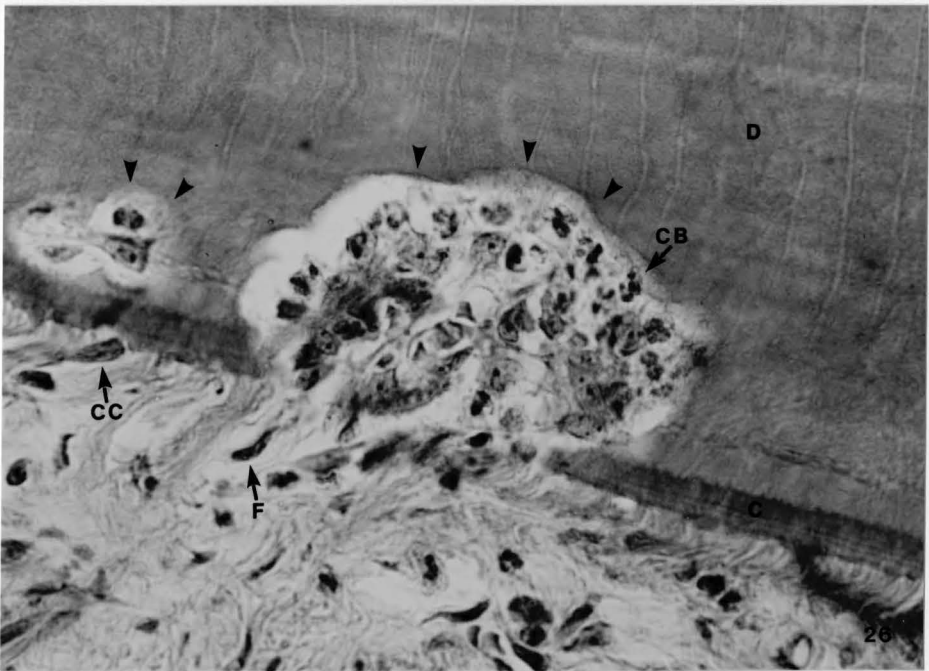
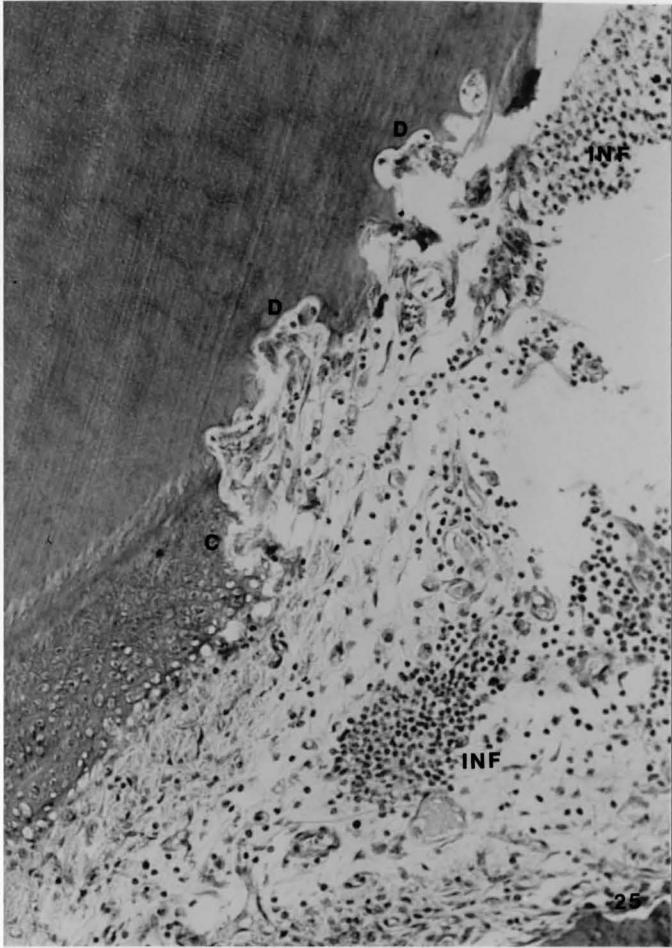


Figure 27: Unfilled perforation in the 120 day animal. Note destruction of the supracrestal gingival fibers (S) and the ingrowth of a loose connective tissue stroma (C) into the defect. Dentin (D). (Hematoxylin and eosin stain, original magnification X40.)

Figure 28: Unfilled perforation in 180 day animal. Note the degeneration and proliferation of epithelium (E) to below the level of the defect. A dense infiltrate of inflammatory cells (INF) is seen as well as an ingrowth of chronic inflammatory tissue (C) into the perforation defect. Dentin (D), abscess (A). (Hematoxylin and eosin stain, original magnification X40.)

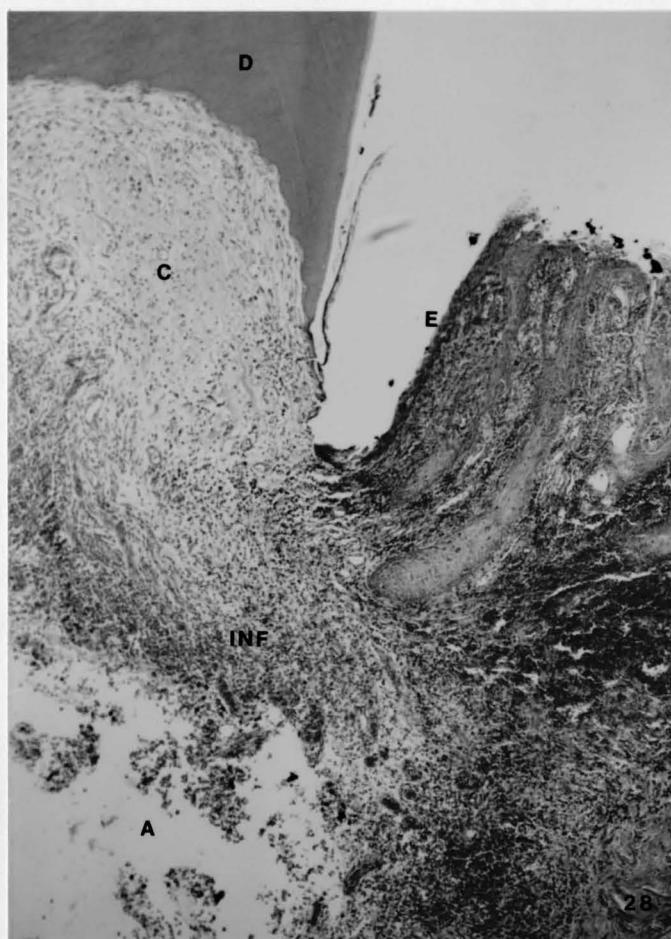
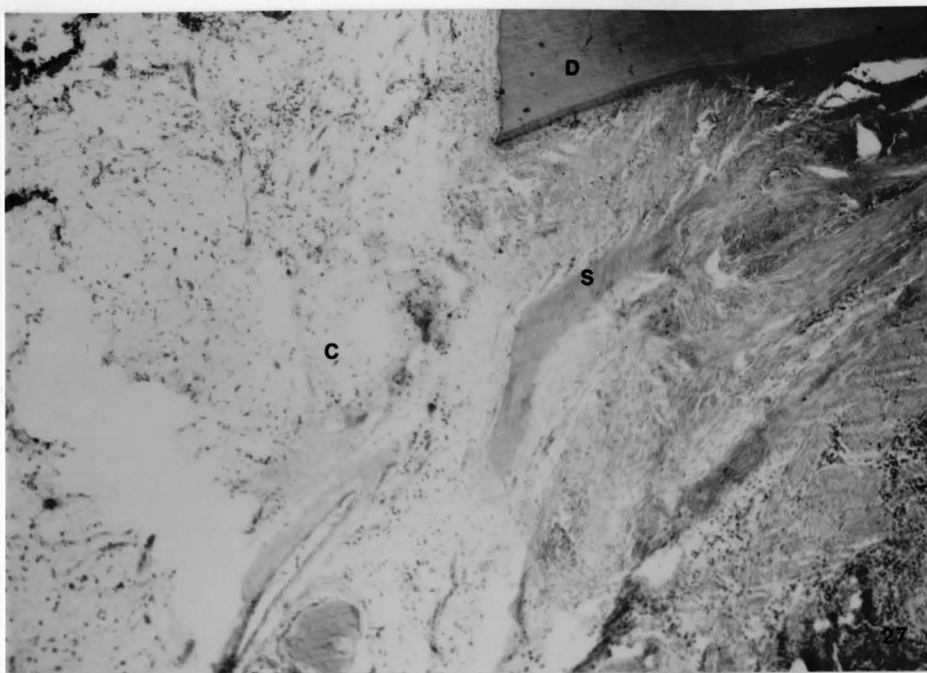


Figure 30:

Figure 29: Unfilled perforation in 180 day animal. Epithelial proliferation and degeneration (E) is evident as well as destruction of the periodontal ligament (P). Dentin (D). (Hematoxylin and eosin stain, original magnification X40.)

Figure 30: Higher magnification of area outlined by rectangle in Figure 29. Note the layer of proliferated epithelium (E) and chronic inflammatory cells (I) present. The cells consist of plasma cells, lymphocytes and macrophages. (Hematoxylin and eosin stain, original magnification X250.)

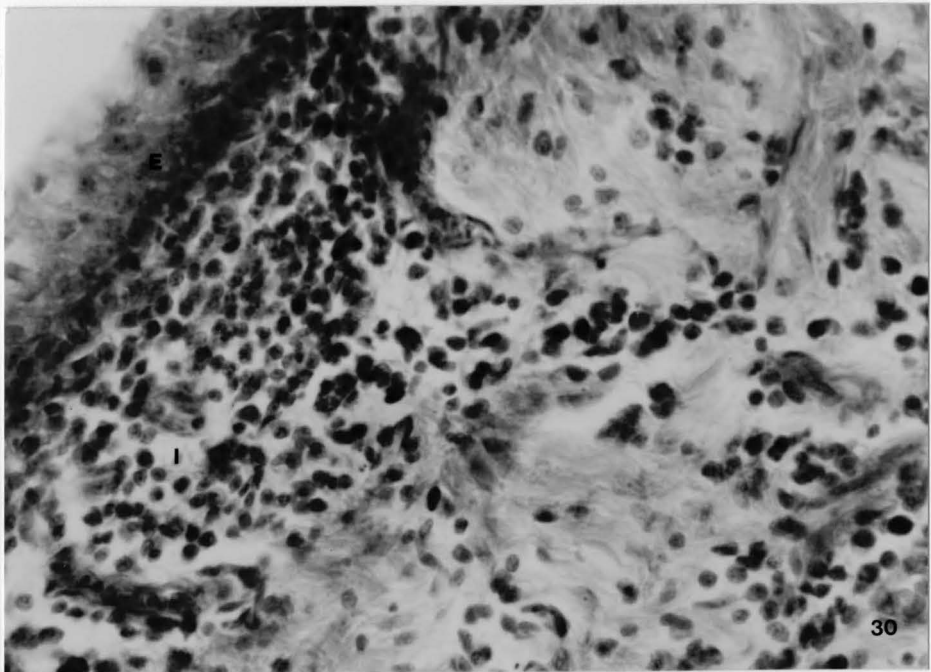


Figure 31: Filled perforation (LR4-distal) in fifteen day animal. Note basophilic staining of the adjacent alveolar bone (ARROWS). Inflammation and osseous activity is minimal. Dentin (D). (Hematoxylin and eosin stain, original magnification X40.)

Figure 32: Filled perforation (LL3-distal) in fifteen day animal showing an advanced inflammatory reaction. Note spread of inflammation (INF) along the periodontal ligament (P) resulting in degenerative changes. Osteoclast (OC), dentin (D), Cavit filling material (C). (Hematoxylin and eosin stain, original magnification X125.)

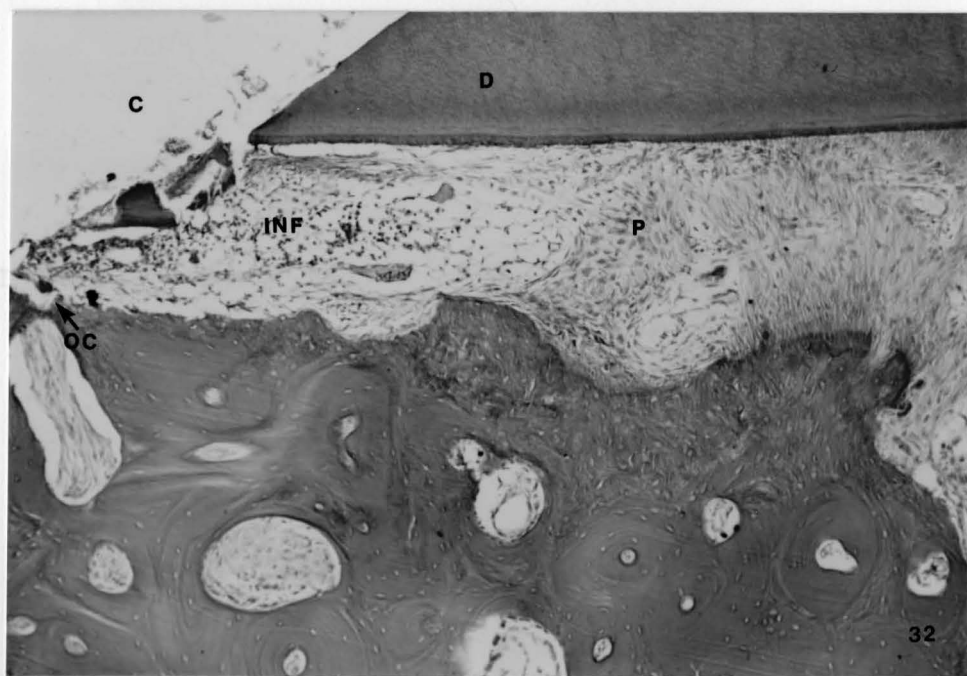
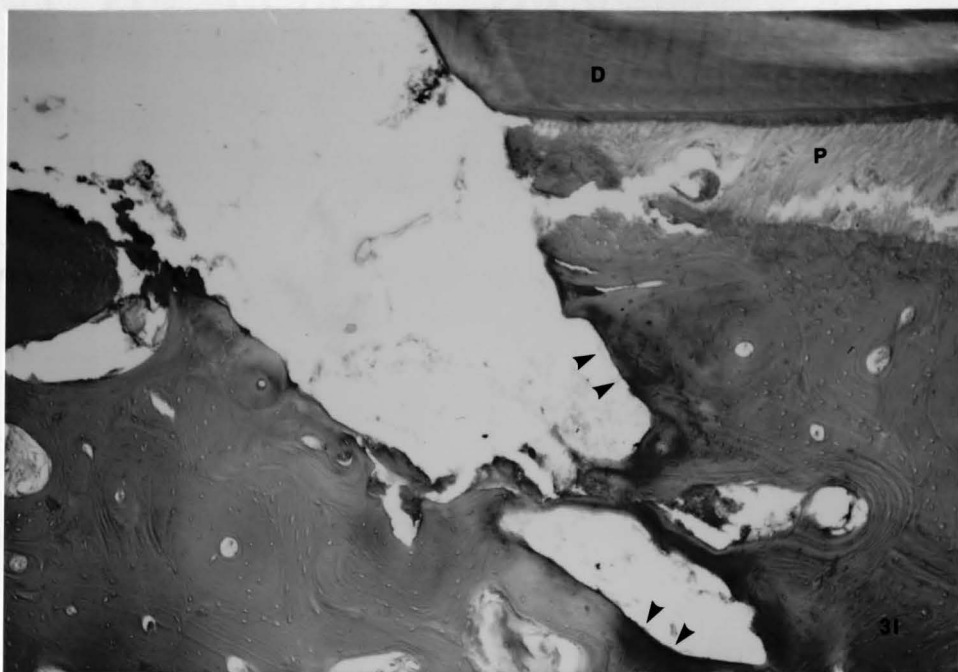


Figure 33: Filled perforation in thirty day animal. Note fibrotic activity (F) adjacent Cavit (C) filling material. Dentin (D), alveolar bone (B). (Hematoxylin and eosin stain, original magnification X40.)

Figure 34: Higher magnification of area outlined by rectangle in Figure 33. Observe the presence of multinucleated giant cells (ARROWS) and inflammatory cells in the loose connective tissue stroma (S) adjacent the Cavit (C) filling material. (Hematoxylin and eosin stain, original magnification X400.)

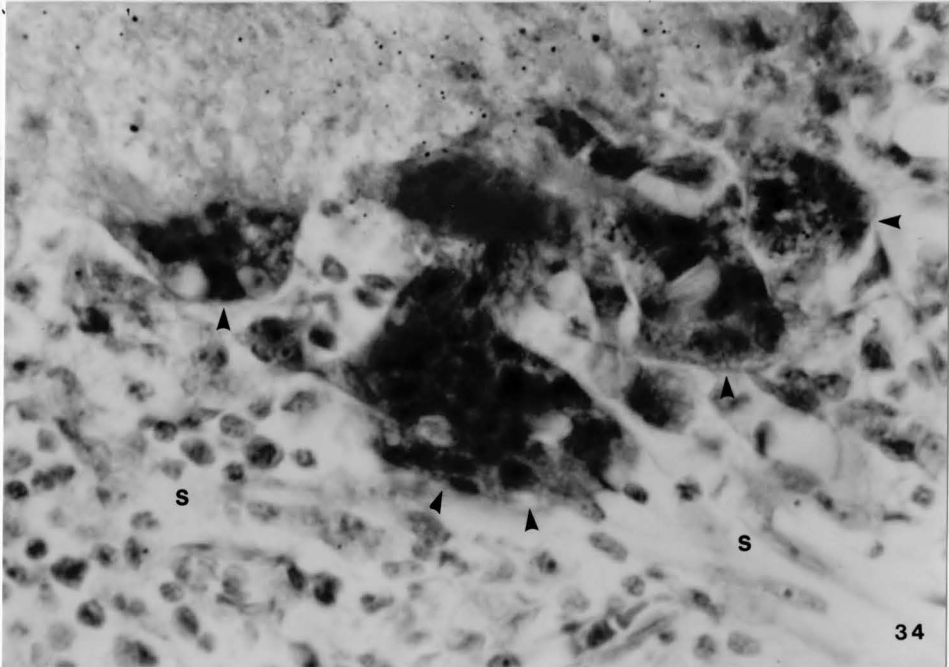
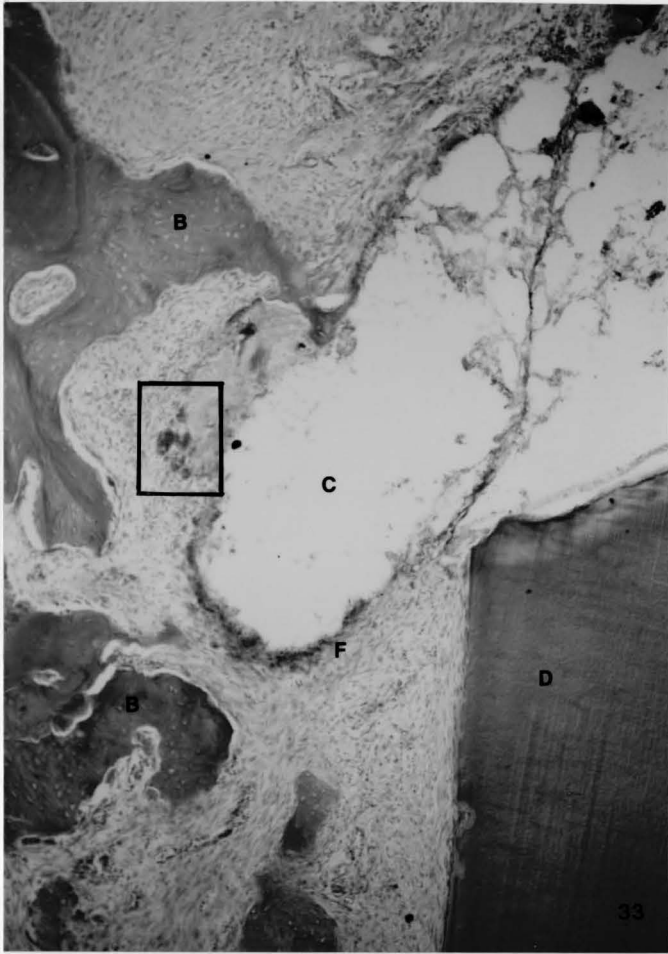


Figure 35: Area just incisal to filled perforation (P) in thirty day animal. Note areas of root and cementum resorption (ARROWS) and repair by osteoid formation (O) resulting in ankylosis. (Hematoxylin and eosin stain, original magnification X40.)

Figure 36: Collagen capsule (C) surrounding Cavit filling material (F) in a filled perforation in 60 day animal. (Hematoxylin and eosin stain, original magnification X40.)

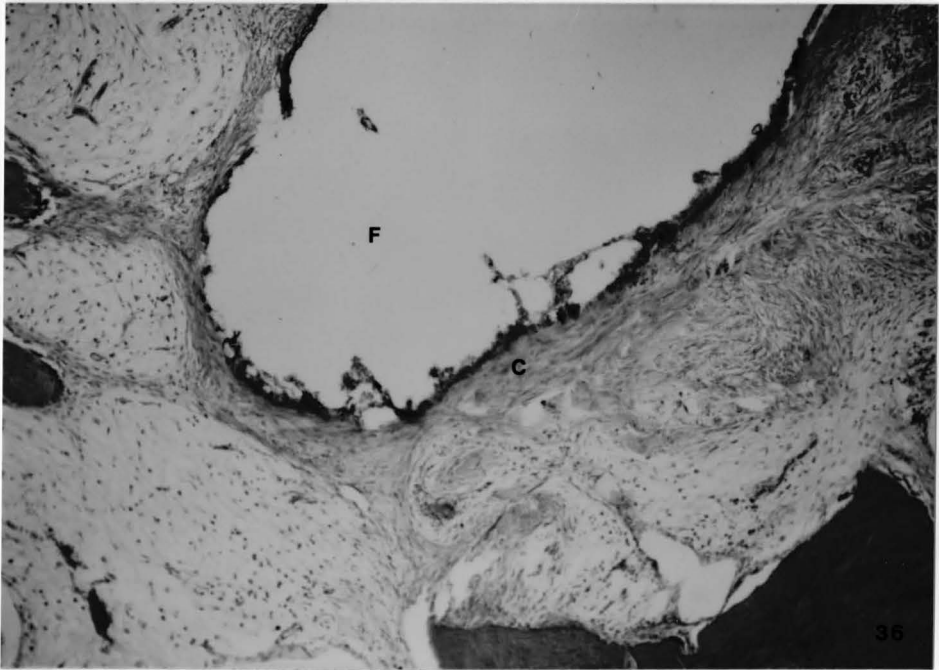
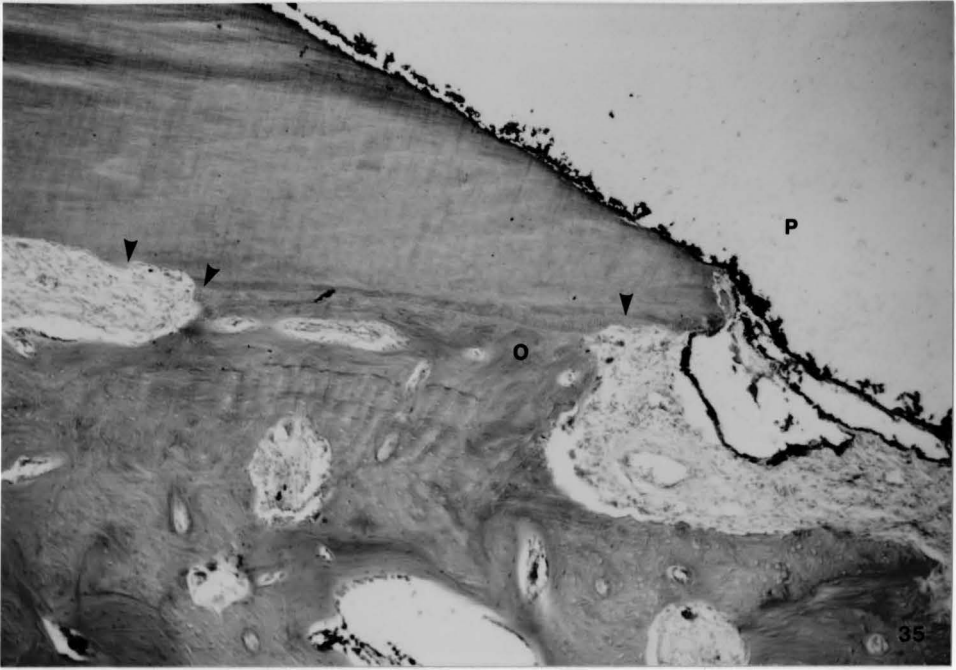


Figure 37: Higher power of collagen capsule in Figure 37. Note orientation of collagen fibers (F) parallel to the Cavit (C) filling material. Inflammation is also noted to be minimal. (Hematoxylin and eosin stain, original magnification X160.)

Figure 38: Filled perforation in 120 day animal showing proliferation of epithelium (E) around Cavit filling material (C). Note the minimal inflammation and the fibrotic activity (F) attempting to wall off the defect. Dentin (D), alveolar bone (B). (Hematoxylin and eosin stain, original magnification X40.)

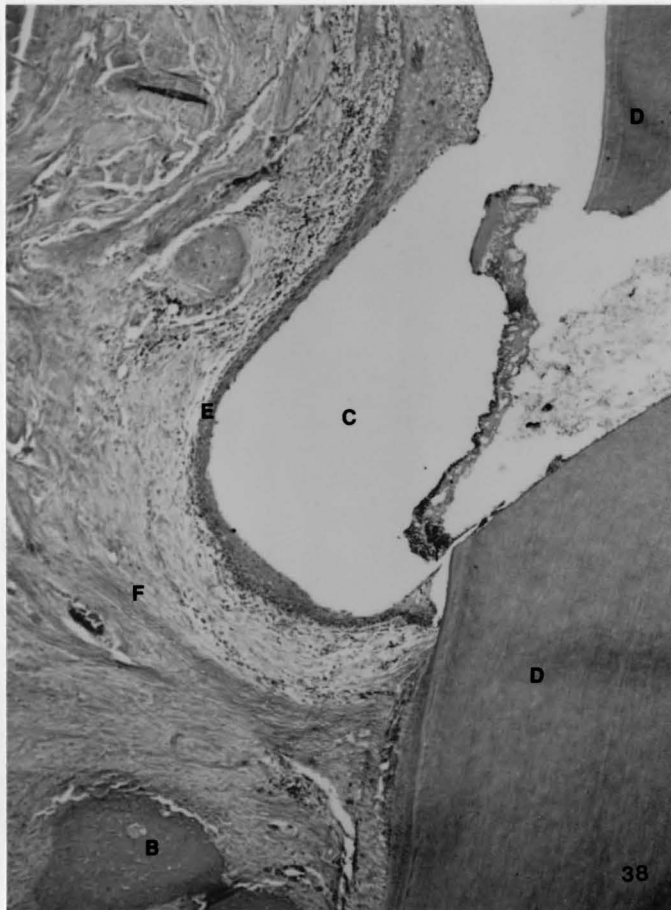
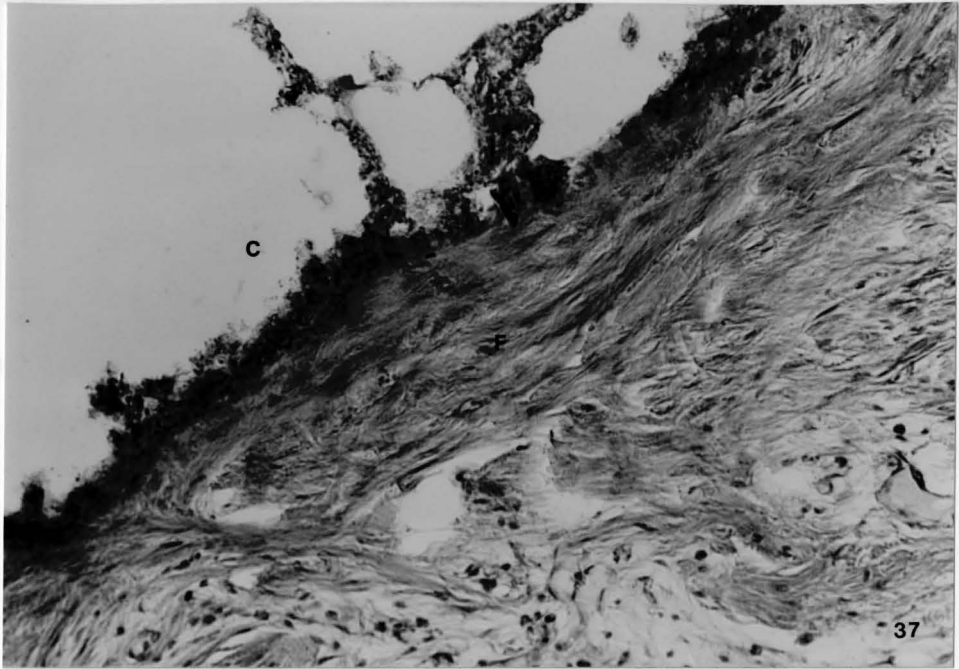


Figure 39: A partially filled perforation in the 120 day animal. An ingrowth of moderately inflamed connective tissue (I) is found adjacent the filling material (C) as well as a deposition of secondary cementum (S) on the inner dentinal walls of the perforation canal. Dentin (D). (Hematoxylin and eosin stain, original magnification X40.)

Figure 40: Filled perforation in the 120 day animal revealing encapsulation by fibrous connective tissue (F) and a slight chronic inflammatory response (I). Cavit (C). (Hematoxylin and eosin stain, original magnification X64.)

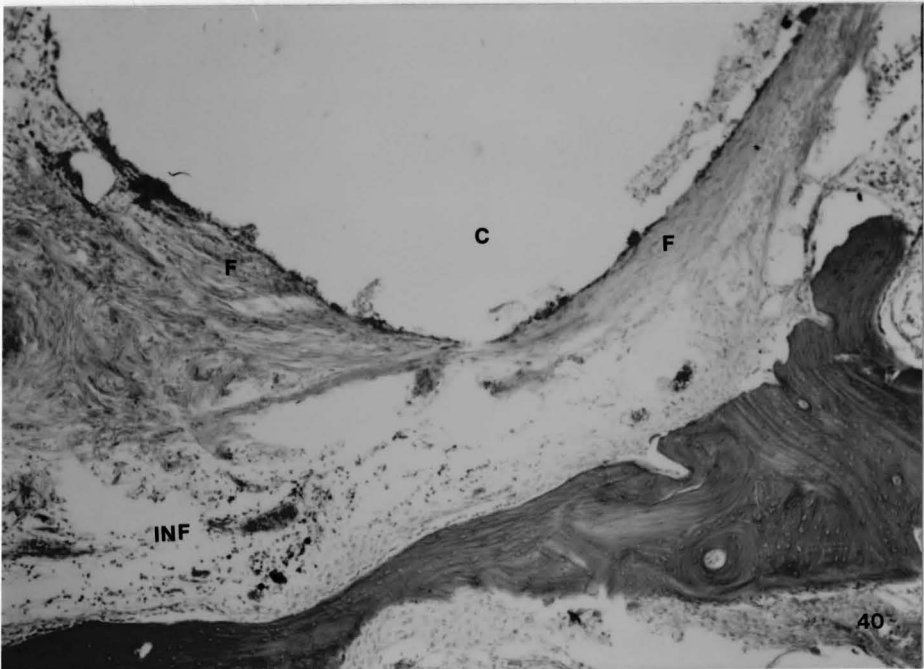
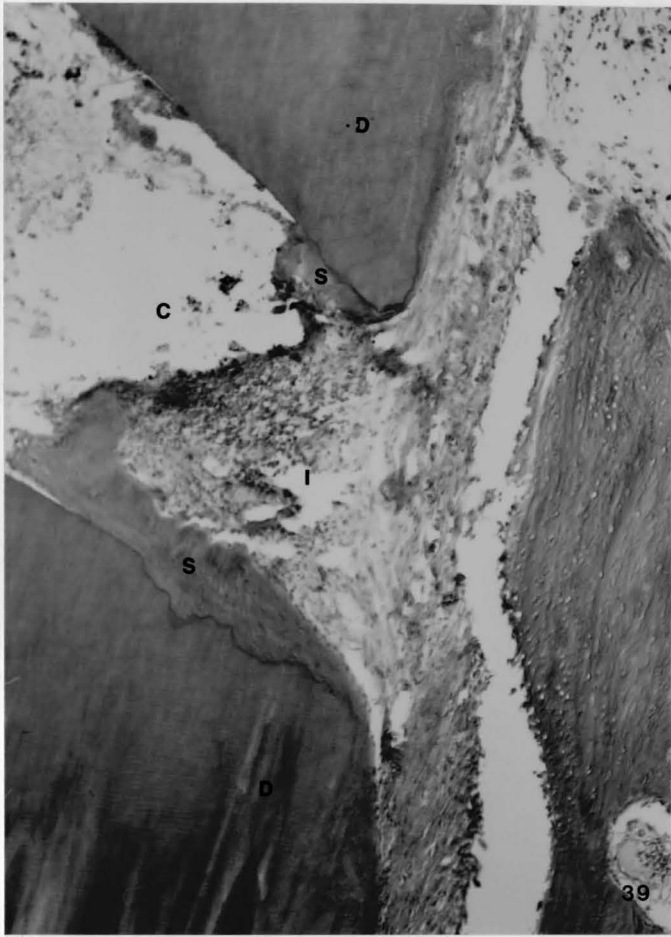


Figure 41: High magnification of collagen capsule in 120 day animal. Note dense connective tissue (F) surrounding Cavit (C). Also present are osteoclasts (OC) and multinucleated foreign body giant cells (GC). (Hematoxylin and eosin stain, original magnification X250.)

Figure 42: Filled perforation in 180 day animal. Note heavy fibrous tissue encapsulation (F) around Cavit (C). Inflammation is chronic, though mild in intensity (INF). (Hematoxylin and eosin stain, original magnification X40.)

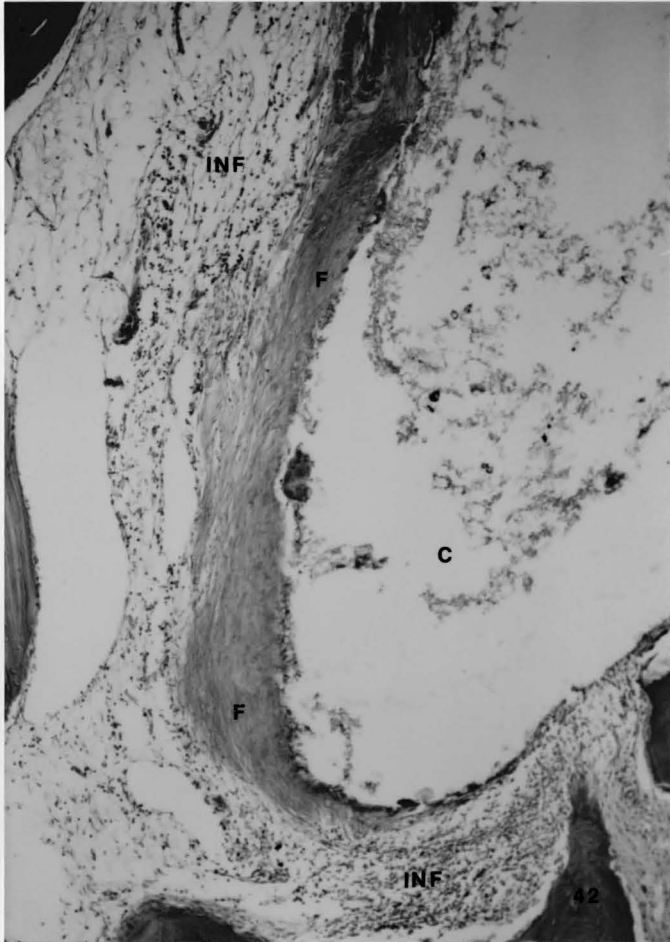
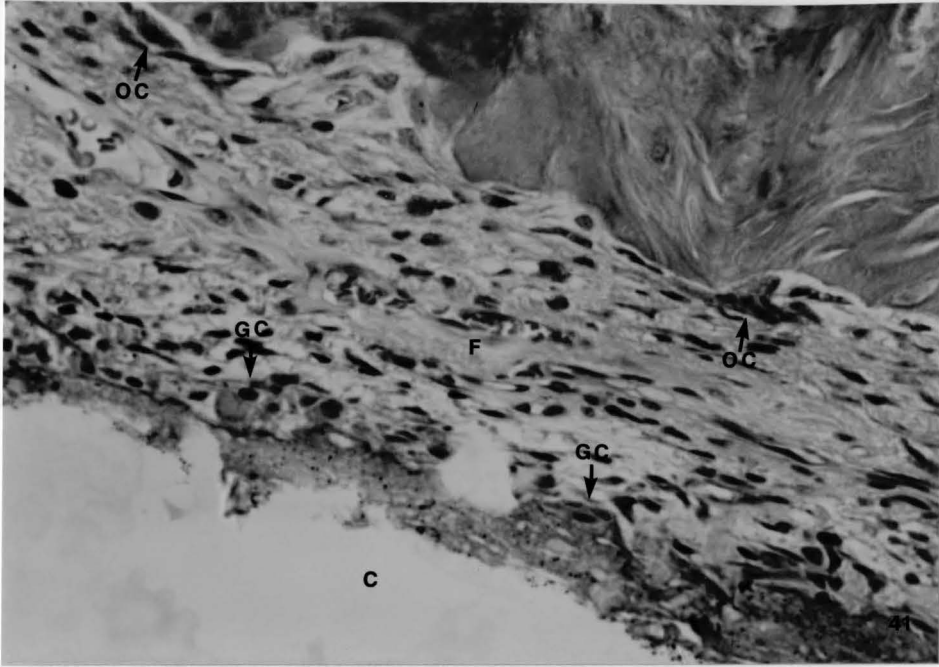


Figure 43: Higher magnification of area outlined by rectangle in Figure 42 of 180 day animal. Note density of fibrous connective tissue capsule (F) adjacent the Cavit (C) filling material. (Hematoxylin and eosin stain, original magnification X125.)

Figure 44: Higher magnification of Figure 43 showing loose connective tissue stroma external to the capsule around Cavit. Note the mild infiltration of plasma cells (ARROWS) and continued fibroblastic activity. (Hematoxylin and eosin stain, original magnification X425.)

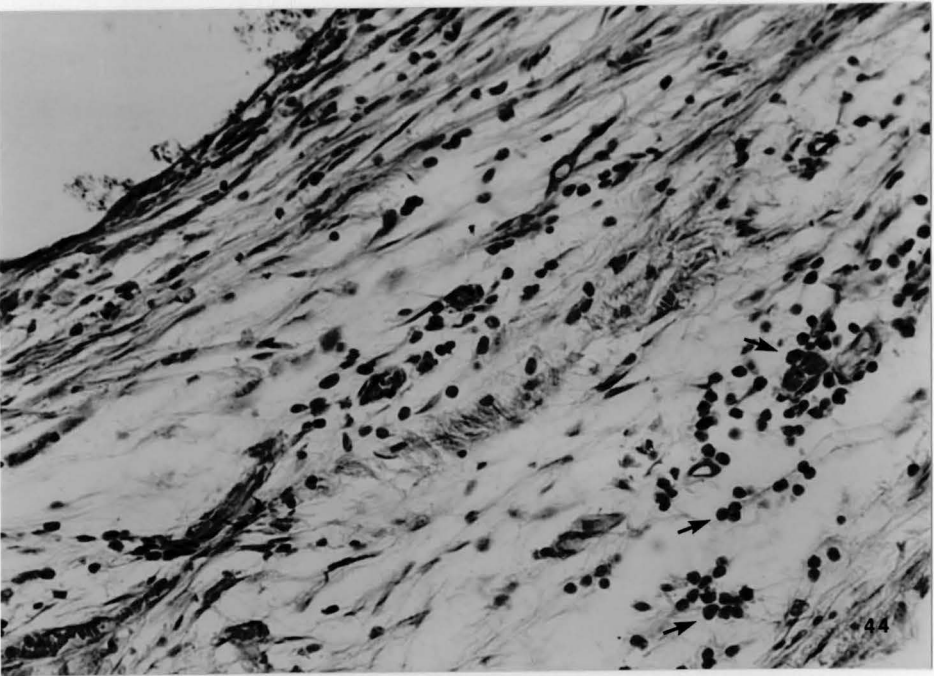
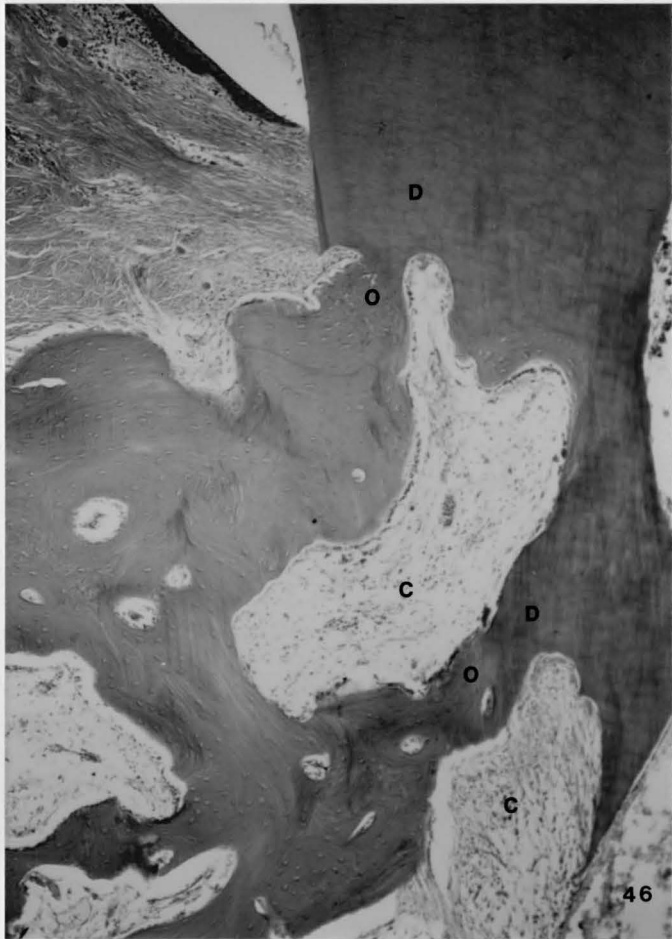
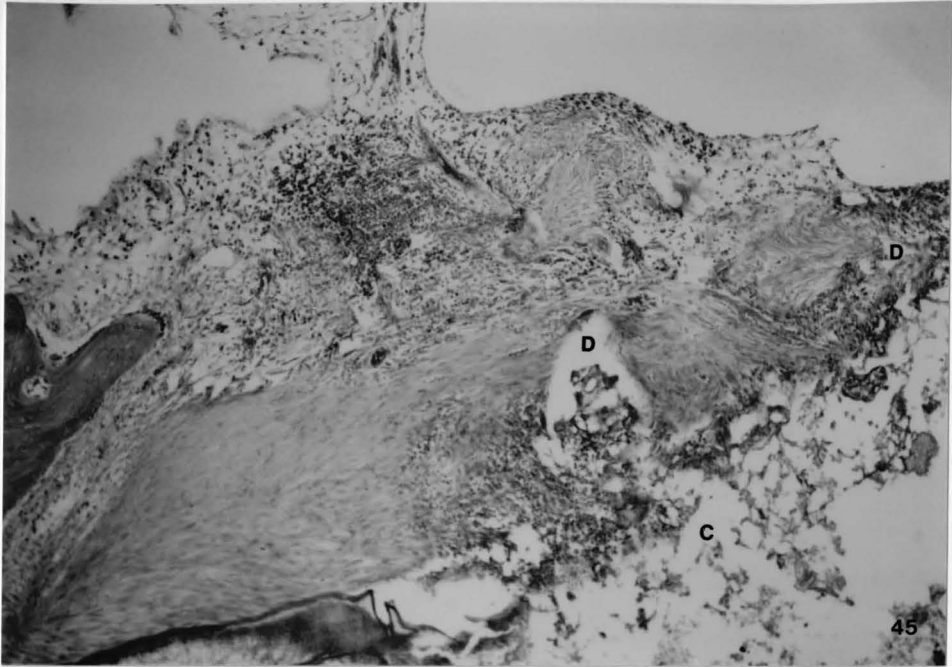


Figure 45: Filled perforation in 180 day animal. Note disruptions (D) in the collagen capsule due to apparent disintegration of the Cavit (C). (Hematoxylin and eosin stain, original magnification X100.)

Figure 46: Root surface incisal to filled perforation in 180 day animal. Note repair of resorbed dentin (D) by osteoid deposition (O) resulting in areas of ankylosis. Also shown are areas of chronic inflammatory tissue (C) and new bone apposition (ARROWS). (Hematoxylin and eosin stain, original magnification X40.)



APPROVAL SHEET

This thesis submitted by Ronald C.K. Jew, D.D.S., has been read and approved by the following committee:

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The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis is now given final approval by the above committee with reference to content, form, and mechanical accuracy.

The thesis is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science.

Franklin S. Weine, D.D.S., M.S.D.

5-29-79
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