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A Comparison of Periapical Healing After Induced Apical Periodontitis

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A COMPARISON OF PERIAPICAL HEALING AFTER INDUCED APICAL PERIODONTITIS

by

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DEDICATION

To my father, Theodore C. Neach, who passed away on November 9, 1981. He was a man who taught me to love and respect life and family. He willingly sacrificed many of his own wants so that I might complete my education. I will always be grateful and forever miss him.

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LIFE

Charles R. Neach, the son of Theodore C. Neach and Virginia (De Witt) Neach, was born in Washington, D.C. on January 3, 1946.

Following elementary and junior high school education in the Langdon Elementary (Washington, D.C.), Twinbrook Elementary (Rockville, Md.), and Broome Jr. High School (Rockville, Md.), he graduated from the Richard Montgomery High School (Rockville, Md.) in June of 1963. He attended the University of Maryland where he was awarded the Bachelor of Science Degree with Honors in August of 1967. He attended the University of Maryland School of Dentistry where he was awarded the Doctor of Dental Surgery degree in June of 1970.

Upon graduation from dental school he was chosen by the U.S. Public Health Service for a rotating dental internship program at the U.S. Public Health Service Hospital in Seattle, Washington. After completing the dental internship in June of 1971, he was assigned to be a staff member responsible for teaching dental interns at the U.S. Public Health Service Hospital in Galveston, Texas. In July of 1973, he resigned his Commission in the U.S. Public Health Service to enter Loyola University School of Dentistry and to begin graduate study toward the degree of Master of Science in Oral Biology and clinical specialty training in Endodontics. He is currently a clinical associate professor in the department of Endodontics at Loyola University School of Dentistry and maintains a private office for the practice of Endodontics in Chicago.

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INTRODUCTION

Ninety percent of patients with dental pain have inflammation of the pulp and/or periapical tissues and are thus potential endodontic patients. This statistic was first reported by Mitchell and Tarplee[1] and then corroborated in an exhaustive study of over 1,600 dental patients in pain evaluated by Hasler and Mitchell.[2] One of the most important functions of any dentist is to give efficient and efficacous treatment so that his patients are relieved of pain. Unfortunately, the correct emergency endodontic procedure to be utilized for each of the different painful conditions is open to considerable controversy.

It is a common endodontic procedure to open a tooth that is tender to percussion to establish drainage and then to allow the tooth to be left open to the oral fluids after this initial appointment. The rationale for this type of therapy is to reduce pressure build-ups inside the tooth, thus decreasing the chances for apical exacerbations and reducing inter-treatment emergency visits for relief of recurrent painful episodes. However, many endodontic clinicians think that these same teeth should not be left open, but rather should always be sealed from the oral fluids during endodontic therapy. The rationale for this view is that the area of apical inflammation will not be invaded by massive numbers of microorganisms from the oral cavity. This could lead to more complex problems such as difficulty in keeping the tooth closed once it has been sealed in later appointments. In addition, there will be fewer total appointments necessary.

Because there are no scientific studies with histologic evidence concerning this controversy, the rationales for some leaving teeth open or closed are not based on evidence from scientific research but rather on clinical impression. One can see that a controversy over methods of pain relief treatments exists.

It is the objective of this study to compare the periapical healing in teeth with induced apical periodontitis after thorough biomechanical and chemical canal cleansing where access cavities for endodontic therapy have been left open to the oral fluids and where endodontic access cavities have been sealed from the oral fluids.

LITERATURE REVIEW

Diseases of the dental pulp and periapical tissues have afflicted men from earliest prehistoric times. The earliest evidence is found in remains from the Paleolithic period (25,000 to 40,000 years ago).[3] Through the years many varied and unusual cures have been described.

The teachings of Hippocrates (460-370 B.C.) marked the dissociation of medicine and dentistry from religion. Hippocrates recommended that a painful tooth which is firm and not decayed be dessicated by cauterizing the pulp.[3]

Archigenes of Syria (98-117 A.D.) found a tooth which ached violently could be relieved by opening into the central chamber through the carious lesion with a trephine.[3] Galen (131-201 A.D.) also recognized the need to gain access to the pulp canal.[3]

In the middle ages there was a strong belief that tooth decay was caused by the presence of tooth worms. Abulcasis (1050-1122) cauterized the pulp with a red-hot needle introduced through a tube in order to protect surrounding areas of the mouth.[4] Guy de Chauliac, a medieval surgeon, used a mixture of camphor, sulphur, myrrh and asafetida as a filling material to cure toothache caused by worms.[4]

In the early Renaissance period, when alchemy was respectable, the materia medica for odontalgias was often just as bizarre. The mechanical aspects of treatment were dependent upon an appreciation of anatomy and so they lagged behind the pharmacological approach until the end of the

eighteenth century when dental anatomy was scientifically recorded by Hunter (1771).[3] Johann Stephan Strabelbergen (1630) used oil of vitriol or a concoction made of a frog cooked in vinegar to kill worms in teeth.[4] Lazarre Rivierre was the first to call for a remedy that is still being used for toothaches today--placing a small piece of cotton moistened with oil of cloves into the cavity.[4] The first dental textbook in English, written by Charles Allen (1687), describes purely a pharmacological approach to odontalgias.[3] Pierre Fauchard, the founder of modern dentistry, assured his readers that rinsing the mouth every morning and evening with a spoonful of one's own urine immediately after it has been emitted produces great relief for those suffering from toothache.[4] Fauchard also illustrated instruments used for cautery and for enlarging the root canal to fit a pivot tooth.[3]

L.B. Lenter, a German, wrote a pamphlet in 1756 recommending electricity as a means for curing toothache.[4] Others have recommended the use of a magnet as an alternative. Bourdet (1757) removed the pulp with an instrument bearing a three-sided point. He also illustrated the first cauteries specifically for use in the root canal.[3] Hunter (1771) states that it is necessary to cauterize "to the point of the fang" and then suggests the use of oil of vitriol in the tooth or when gravity worked against it, the use of caustic alkali which was solid.

In 1794 Raniere Gerbi describes an insect living habitually in the flowers of the Cardisus Spinosimus that can be used to cure a violent toothache. The larvae or fully developed insects are crushed between

the thumb and forefinger until the matter is entirely absorbed. The two fingers are then held upon the decayed and aching tooth.[4]

Snell (1832) describes the use of acetate of morphine and actual cautery for the destruction of inflamed and sensitive pulps. He devised a steel instrument with a bulb at the end from which projected a platinum wire. Heat was retained in the steel bulb for such a time as to allow the platinum wire to destroy the contents of the root canal.[4]

Until the early 1800's, there seemed to be no distinction in treatment of toothaches caused by teeth with a vital inflamed pulp, pulp necrosis, or periapical involvement. Pioneers in endodontic therapy during this period attempted to relieve pain and yet preserve the vitality of the dental pulp by various methods. D.L. Koecker (1826) had patients pay close attention to their diets. He cauterized the exposed pulp and stimulated externally with myrrh, camphor and opium to reduce inflammation of the gums.[5] Fitch (1829) used astringents, alum, borax and aleppo gall which he applied every ten to fifteen days for several weeks or months as necessary.[5] Bell (1837) used stimulants such as alcohol and spirits of camphor for treatment of pulp exposures.[5] Harbert (1847) soldered a gold tube to the cap in order to allow any discharge from the pulp to pass through the filling.[5] Robinson of England (1849) used collodion with morphia over pulp exposures and then filled the cavity with asbestos saturated with collodion.[5] Tomes (1859) used mat gold where the opening was small and a softened, shaved piece of quill where a great amount of decomposed dentine existed around the opening to the pulp.[5] Rogers (1858) used a plate of gold over the exposure site and filled the rest of the

cavity with amalgam.[5] Harris (1858) used gold filling without a cap; however, he built up arching over the pulp.[5] Foster (1858) used Hill's vegetable stopping (gutta percha) to line the inside of the cap.[5] Codman of Boston used cotton and refilled daily from eight to fifteen months.[5] Allport of Chicago, excised a portion of the pulp and brought the edges together into close apposition and thus obtained healing by first intention.[5] Richardson (1861) suggested that the exposed pulp be excised and the cavity be filled with a preparation of artificial dentine in immediate contact with the exposed nerve.[6] Elliot (1868), where there was an inflamed pulp and pain, made a slight puncture in the pulp to relieve the engorged vessels within the tooth and then set leeches upon the gum.[7] The use of leeches had great popularity before the years of "wonder drugs".[4] Page (1871) used chloral hydrate at the exposure site to deaden and allay the pain.[8] Cutler (1872) used a covering of gutta percha dissolved in chloroform to cover the pulp because it settled down over the pulp exposure site without pressure and the chloroform rapidly evaporated.[9] Stevens (1872) noted the nerves were dulled when capped with osteo-plastics.[10] He also placed little confidence in the efficacy of drugs. To cap the pulp, he used lead foil carefully packed over the cavity making sure not to compress the nerve. Jack (1873) noted failures with the use of oxychloride of zinc preparations.[11] He applied aconitum to the exposed pulp tissue, then dilute carbolic acid covered by a gutta percha varnish and followed by a tin or lead covering to protect the pulp from compression by the temporary filling.[11] He waited three years before placing the permanent filling material.[11] C.E. Francis (1873) used

sufficient carbolic acid at the exposure site to allay the pain, then placed a paper cap with a solution of balsam of fir and chloroform covered by a paste of oxychloride of zinc.[12]

During the 1870's doubts over the success of pulp capping began to be expressed. Pease (1861) states, "I do not know that I have plugged over a single exposed nerve that is now alive. Bear in mind, I do not say none of them are. Some of them have given no trouble: others I know are dead."[13] Truman (1870) states, "The treatment of exposed pulps, their destruction or preservation, is an open question with our profession which the experiments and experience of many long years have failed to decide."[14] Welchins (1870) states that pulp capping is controversial and at many times unreliable.[15] Gaine (1872) states that efforts to preserve the vitality of the nerve after capping procedures are futile and that root canal therapy should be performed.[16] These doubts about the successful outcome of pulp capping procedures and the discovery of a less painful method to extirpate the pulp lead to more definitive root canal treatment procedures including debridement and obturation of the root canal space.

The first use of arsenious acid for pulp devitalization was recommended by Dr. S. Spooner in 1836.[4] He recommended a fortieth or fiftieth part of arsenious acid to be mixed with an equal quantity of sulphate of morphia and then applied to the exposed dental pulp.[4] The pulp's vitality would be destroyed in three to seven hours but not without pain. The dental literature of this period, before the discovery of local anesthetics, is filled with articles on the use of arsenious acid to devitalize troublesome pulps.[17,18,19,20,21,22,23,24,25,26] Many practitioners used

arsenious acid in combination with another agent or in a different manner to achieve pulp devitalization. Latimer (1867) allowed the arsenical paste to stay in teeth from one to three days before removing it. He made no attempt to extirpate the pulp tissue until ten days later.[27] If this method of treatment failed, Latimer would then use a form of general anesthesia (nitrous oxide) in order to extirpate the pulp.[28] Chase (1867) used an arsenic preparation also. However, if there was pain on extirpation of the pulp he would prescribe a preparation of "mercurius vivus".[29] Neeland (1870) noted that patients experienced pain after the application of the arsenical paste.[30] He used a small quantity of carbolic acid on the exposed pulp for ten to fifteen minutes before applying the arsenical paste and noted little or no pain when the pulp tissue was removed. Palmer (1871) used a very involved method to obtain pulp devitalization.[31] He would apply the arsenical paste to the exposure site and allow it to remain for two to three days. He then removed the arsenical paste and applied only creosote allowing it to remain for eight to ten days. He would then remove the remaining pulpal tissue. Shadoan (1872) would obtund any sensitivity by using chloroform or creosote with a tincture of aconite before using the arsenious acid and carbolic acid with the sulfate or acetate of morphia.[32] M'Quillan (1871) stated the importance of arsenical applications when he said, "The employment of arsenious acid, some forty years ago, for the purpose of devitalizing the exposed pulps of teeth may be justly regarded as one of the most important steps taken by the profession in the preservation of teeth; for prior to that time aching teeth were invariably extracted."[33] The main problems associated

with arsenic applications were that it was painful to apply and that it would leak out of the pulp cavity at times thus destroying the periodontal ligament and alveolar bone.

In 1884 Koller discovered the anesthetic effect of cocaine.[4] Burge (1888) used cocaine in a hypodermic syringe inserting into the pulp cavity and injecting the cocaine directly into the pulp.[34] Myers (1904) developed a high-pressure syringe for injecting cocaine under considerable pressure directly into the pulp.[34] Einhorn (1905) synthesized Novocaine, a more effective and less toxic anesthetic than cocaine.[34] Vaughn (1906) recommended infiltration anesthesia. However, it was not commonly used for pulp extirpation until the early 1920's when the breech-loading syringe and carpule were introduced.[34] It was not until this time that dentistry, and endodontics in particular, entered its "painless" era.

Treatment of the nonvital pulp or pulpless tooth, particularly one with symptoms of an alveolar abscess, has been extremely varied over the years.[35] Atkinson (1863) describes two forms of alveolar abscess. The benign form required local treatment consisting of evacuation of pus and the malignant form required local and constitutional treatment. Constitutional treatment consisted of antidoting the patient by administration of preparations of codeine, mercury and potash. Atkinson (1862) also suggested the use of cold, warm, or hot applications (whichever comfortable) to the tumified face to reduce swelling.[36] This was continued until the abscess parted; at which time it was opened, the pus evacuated and a dressing of creosote and iodine placed. Abbott (1872) also suggested that treatment of alveolar abscesses consists of local and constitutional

measures.[37] Local treatment included an abortive phase of opening into the pulp chamber, a palliative phase of relieving pain by means of a hypodermic injection of morphia, and a curative phase of removing the necrotic pulp tissues and introducing antiseptic solutions (permanganate of potash, carbolized oil) into the pulp canals and abscess cavity. Constitutional treatment included allaying undue febrile action by means of arterial sedatives and insuring a proper supply of nutritious food.

Specific treatment regimens for alveolar abscesses during the late 1800's depended on the whim of the operator. Most recognized the need to establish drainage and to evacuate any pus and necrotic contents from the pulp canals. Their methods to accomplish this aim varied greatly. They included the use of strong antiseptic preparations within the tooth, incision and drainage through the soft tissue of the oral cavity, drainage through the tooth itself by keeping it open to the oral fluids for various lengths of time, and apical surgery.

Of the antiseptic preparations, creosote was one of the most popular. Taylor (1856) suggested that for a tooth with suppuration access to the nerve cavity be made by drilling under the free margin of the gum.[18] All foreign material should be removed from the canal and injections of tepid water and chlorine preparations be made. The nerve cavity was filled with cotton or thread moistened with creosote. If a discharge continued, injections of a nitrate of silver solution were made every two or three days. Farrar (1863) treated the alveolar abscess by making an artificial fistula if none existed.[38] Creosote was used to cauterize the inside of the abscess sac. He also suggested cauterization of the

abscess sac by passing a silver wire dipped in nitric acid into it. He also treated the alveolar abscess systemically using common antiphlogistic remedies such as opium or anodynes. Fitch (1864) used creosote that was carried to the very apex.[39] Chase (1866) and Palmer (1871) also used creosote which was forced through the roots.[40,31] Chase thought that for treatment to be successful there must be an outlet through the gums. Latimer (1867) suggested the use of iodine together with creosote inclosed in teeth.[27] Teeth with sinus tracts were treated through the canal and sinus tract. Latimer (1866) was one of the first practitioners to allude to the pulpless tooth that was quiet for months or even years that became very troublesome after the commencement of treatment.[41] He also stated that the time employed in treatment of alveolar abscesses was greater than in treatment of other conditions.[41] Dean (1882), one of the first to employ a rubber dam during treatment procedures, passed a broach through the apex gently to remove any obstruction to liquids and then injected creosote through the apical foramen to the abscessed alveolar bone.[42] As soon as the fistula closed, he would finish the root canal therapy. In treatment of a blind abscess (one with no fistula), Dean recommended that the canal be cleansed gently to avoid debris through the apex. A broach was passed through the apex to stimulate drainage and the canal washed with eucalyptus oil or phenol sodique. A dressing of creosote would then be sealed in the canal. Watson (1883) also differentiated between treatment of the abscessed tooth with a fistula and one without a fistulous opening.[43] For teeth with a fistula, he cleansed the canals using a weak solution of carbolic acid making sure to work the solution

into the abscessed area. He used a dressing of eucalyptus oil and iodoform. For teeth without a fistula, he changed the antiseptic dressings every two to three days but also administered a brisk purgative and either sulphate of quinine or calcium sulphide in order to absorb the inflammatory exudate.[44] Kulp (1885) was also a proponent of the use of creosote. He suggested that the canal be opened carefully and without forcing air or an instrument into the canal, cotton saturated with creosote and tannin be placed in the root for twenty-four hours. This was to be continued until no pus was seen emanating from the pulp canal. Niles (1888) suggested that in treatment of abscessed and septic teeth a long cleansing process was needed.[23] The teeth were kept closed and a strong solution of bichloride of mercury (5%) was used. Overholzer (1890) used such disinfectants as bichloride of mercury, peroxide of hydrogen, oil of eucalyptus, and phenol of camphol phenique to clean the pulp canal.[45]

One of the few who warned against the use of creosote or other caustics was Sace (1872).[46] He thought that these agents may find their way into the antrum and produce inflammation of the lining membrane. He especially advised care in the treatment of maxillary bicuspids and molars.

White (1856) and Fitch (1864) were two of the early proponents of incision and drainage.[47,39] If there was swelling with fluctuance, Fitch incised the area and opened it to the apex. If pus was present, a splinter of wood with cotton dipped in a solution of resublimed iodine and creosote was carried to the depth of the wound and renewed every day.[39] M'Quillan (1871) passed a lancet into the area of fluctuation to stimulate pus drainage.[48] Flagg (1872) was also a proponent of incision and

drainage.[49] He first applied heat such as hot baths, heated cloths or warm poultices to produce an area that was pointing before any incision and drainage. He used general stimulant ointments and mixtures such as various salves of rosin, beeswax, etc., and employed anodynes such as tincture of aconite, carbolic acid, and chloroform to relieve pain during the progress to the stage where there was sufficient suppuration. Others using incision and drainage or actual fistulation were Watson (1883), Bate (1883), Rambo (1885) and Balding (1890).[43,50,51,52]

In 1863 one of the earliest descriptions of a tooth opened and left open for drainage was written.[57] The patient's symptoms were described as the tooth being raised in the alveolus, the tooth felt longer, and there was pain to percussion. When the pulp canal was opened the pulp was found to be nonvital. The pulp canal was washed out and left open until the next day or when the symptoms had disappeared. Palmer (1871) left the tooth open for drainage when there was no fistulous opening.[31] He made access to the pulp canals, placed iodine and creosote in all dressed parts and then left the tooth open for several days. He then closed the tooth, but not very firmly, and would fill the tooth several weeks later. Townsend (1882) would merely open into the pulp chamber to allow an exit for gases.[24] He waited one or two days for the tenderness to subside before continuing treatment. Howe (1888) only stopped the access cavity loosely with cotton without any medication for a pulpless tooth with drainage.[54] If there were no purulent discharge, he placed a loose dressing of iodoform covered only by cotton. For treatment of a dormant abscess, Harlan (1888) packed a dressing of essential oil in the

canal and covered it with cotton and gutta percha which was perforated to allow air or gas to escape.[55] Harlan suggested that successful treatment was due to thorough removal of all pulp tissue and other foreign matter and filling the roots with a substance not porous or corruptible by the fluids of the mouth or products of microbes.

Howe (1888) was an early advocate of apical surgery for pulpless teeth.[54] He advised amputating a portion of the root for the tooth where there was continued suppuration. Gaffe (1888), after canal enlargement through the apical foramen, pumped hydroxyl and perchloride of mercury through the foramen until there was no further discharge of pus. He then advised immediate root canal filling and opening into the abscess to remove it through the alveolus.[56] Hartzell (1909) expressed his belief that abscessed teeth are cause for great distress in the average dental practice to both the dentist and the patient.[57] He felt the safest rule in pus infections was to evacuate the pus early and provide free drainage. The root canal space was cleansed, made as sterile as possible, and then filled. With a trephine, he took out a button of soft tissue and then with a series of pear-shaped burs penetrated the bone to the root end, taking off sufficient root to obliterate the evidence of infection. Buckley (1911) advocated surgical intervention in cases where the tissues in the apical area had been affected so long that no cure can be effected by means of drugs.[58]

During the early 1900's, Hunter expounded his theory of focal infection accusing the dental profession of creating "...a veritable

mausoleum of gold over a mass of sepsis." This led to the extraction of many pulpless teeth. With the advent of the dental X-ray, advances in the fields of dental microbiology and therapeutics, and the determination of a small group of endodontic pioneers, the theory of focal infection was repudiated and needless extractions of pulpless teeth ceased. Although advances in dental research during the twentieth century have found the answers for many dental problems, the treatment for the complex problem of the acute alveolar abscess remains based on the dental operator's clinical impression and not on scientific evidence.

Coolidge (1950) recommended that in treatment of the acute dentoalveolar abscess the tooth should be left open for twenty-four hours only if active drainage occurs from the mouth of the canals.[59] He felt that the tooth should be closed if there was no active drainage because of the danger of further infection entering the canal and periapical tissues. Waterston (1960) initiated drainage from the periapical area through the root canal by widely opening the apical foramen.[60] He left the tooth open for four to seven days until the symptoms subsided and then used a polyantibiotic mixture to fill the canal and periapical area. He did not place a permanent root canal filling until after a second mixture of the polyantibiotic was used. The endodontic section of the Detroit Dental Clinic Club suggested that treatment of the acute apical abscess include opening the tooth for drainage and prescriptions for an antibiotic and analgesic.[61] Sommer, Ostrander and Crowley (1962) recommended that for a mild case of an acute alveolar abscess the tooth be opened and allowed to

remain open until all symptoms have subsided before instituting further endodontic treatment.[62] In the severe case (intraoral swelling, cellulitis, temperature), they recommended incision and drainage in any area of fluctuation; opening into the pulp chamber to establish drainage through the canal, and prescribing antibiotic therapy. Zeldow and Ingle (1962) warned of a large incidence of penicillin- and tetracycline-resistant staphylococcus and streptococcus organisms and recommended culturing to obtain antibiotic sensitivity reactions in order to avoid trial and error in the use of antibiotic therapy.[63] Norris (1963) recommended opening and draining through the canal in the management of the acute alveolar abscess.[64] The tooth was allowed to stay open from two days to a week before it was closed with a germicidal dressing. An abscess that had localized in the oral vestibule was also incised and drained through the soft tissues. If good drainage could not be obtained through the tooth or soft tissues, Norris recommended the use of antibiotic therapy using either a systemic penicillin or sulfonamide. Abramson and Norris (1966) recommend the same therapy as Norris (1963) for the treatment of the acutely infected pulpless tooth.[65] Grossman (1965) stated that for the management of the acute alveolar abscess "treatment consists in establishing drainage at once."[66] Drainage could be established through the root canal or by incision through the soft oral tissues that are fluctuant. The tooth was allowed to remain open for a few days. Supportive treatment consisted of "prescribing an anodyne where much pain is present, a mild mouth wash, a saline cathartic to assist in

elimination, a liquid diet or one of light nourishing foods, and ordering much-needed sleep or rest." In severe cases Grossman recommended the use of antibiotic therapy also opting for either a systemic penicillin or or tetracycline. Frank, et al (1968) suggested that with the use of sulfathiazole it was possible to initiate endodontic therapy immediately and to reduce pain while rarely leaving a tooth open to the oral fluids.[67] He also alluded to the fact that many dentists have found it difficult to start treatment again on teeth that had not been sealed between appointments. Auslander (1970) states that the acute apical abscess must be allowed to drain before closing the tooth.[68] He obtained drainage within a few minutes by passing a file through the apex and then observed the flow of the drainage for a particular sequence. Pus is the first material to start flowing, then blood and lastly serum. Once the serum was seen, Auslander believed that further irrigation and canal enlargement might continue and the tooth should be sealed to prevent the acute case from becoming a chronic one. Weine (1976) suggested a definite sequence in the treatment of an acute alveolar abscess.[69] He believed that drainage should be established through the tooth and the tooth left open to allow further exudate to drain. At the next appointment, within three to seven days, the canals are thoroughly irrigated to remove debris, dried, and without any instrumentation medicated with either a sulfonamide or sulfathiazole and sealed from the oral fluids. Antibiotic therapy is suggested when the patient is febrile and minimal drainage has occurred.

Even today, in this age of specialization, there is a lack of

agreement among diplomates of the American Board of Endodontics whether a tooth should be allowed to stay open to drain or sealed from the oral fluids.[70] In a recent survey questioning treatment modalities for endodontic emergency conditions ranging from pain with a vital pulp to a tooth with a necrotic pulp having diffuse swelling with no drainage through the canal, diplomates varied greatly in their methods of treatment. Some allowed teeth in each of the emergency states to stay open for drainage, some allowed only some emergency conditions to stay open and some allowed no emergency conditions to remain open. Clearly, there is no agreement on which method of treatment provides for the surest healing.

Because of the lack of scientific evidence based on sound research, dental clinicians throughout the ages have had to rely on their own clinical intuition in treatment of these problems.

MATERIALS AND METHODS

Rhesus monkeys were chosen as the experimental animal because of the similarities of their dental anatomies to humans. Four adult Rhesus monkeys weighing from 5.5 Kg to 6.8 Kg were obtained from India via an importer (Primate Imports, New York) and were housed in restraining cages at the Animal Research Facility of Loyola Hospital, under the care of Charles Larson, D.V.M., M.S., and his staff throughout the experimental period. The dental status was that of a mature adult monkey, demonstrating that all permanent teeth were erupted and that some occlusal attrition had occurred. A full set of maxillary and mandibular preoperative radiographs were taken of each animal before any treatment was rendered. The maxillary and mandibular lateral incisors and second premolars were chosen to be used during the experiment.

To prepare the animals for intraoral procedures, an IM injection of phenylcyclidine hydrochloride[a] 20 mg/ml was given in dosages of .1 ml/kg as well as an IM injection of .1 cc atropine.[b]

The experimental teeth were isolated under a rubber dam, the occlusal surfaces were swabbed with Bactine[c] and endodontic access

a) Sernylan (for veterinary use only), Bio-Centric Laboratories, Inc., St. Joseph, Missouri
b) Atropine Sulfate Injection U.S.P., Med-Tech Inc., Elwood, Kansas
c) Bactine, Miles Laboratories, Elkhart, Indiana

cavities were cut into each tooth using a slow-speed portable belt-driven electric engine equipped with a contra-angle handpiece with diamond and carbide burs. A known pure inoculum of S. faecalis[a] was then injected into the pulpal tissues of each tooth and forced into the pulp canals by use of a small K-type endodontic file. The teeth were then sealed from the oral environment by means of a cotton pledget saturated with the S. faecalis placed over the canal orifi and IRM cement[b] placed in the access cavity opening. The experimental teeth were radiographed at monthly intervals to detect periapical changes. Once periapical changes were noted, the experimental teeth of each monkey were again isolated as before and the IRM temporary cement sealing the access cavities removed. A culture of each canal was taken with a sterile paper point premeasured to reach the apical portion of the canal using a thioglycollate broth[c] and incubated at 37°C for 72 hours. After culturing, the working measurement of each canal was determined by routine radiographic techniques after the insertion of a #08 or #10 file. RC-Prep[d] was used to negotiate the full working length of those canals that proved to be calcified and presented

a) Streptomycin resistant Streptococcus faecalis
b) Intermediate Restorative Material, The L.D. Caulk Company, Milford, Delaware
c) Fluid Thioglycollate Medium, Difco Laboratories, Detroit, Michigan
d) RC-Prep, Premier Dental Products Co., Philadelphia, Pennsylvania

problems in negotiation to the apex. Once the full working length was reached, RC-Prep was no longer used. The canals were then irrigated heavily with Glyoxide[a] during the instrumentation with smaller files (#'s 08, 10, and 15) and with 5% NaOCL with larger files. Because of the extremely narrow diameter and curvature of the canals, complete debridement was obtained using filing action, incremental instrumentation and flare preparations as described by Weine.[69] Upon complete debridement, one-half of the experimental teeth of each monkey (one maxillary and mandibular lateral incisor and one maxillary and mandibular second premolar) were sealed from the oral fluids by means of a sterile cotton pledget placed into the pulp chamber, IRM placed immediately over the cotton and an amalgam alloy placed over the IRM. The contralateral half of the experimental teeth were left unsealed and thus open to the oral environment. Eight teeth having fourteen canals were prepared in each animal. Tooth location was selected so that each operated tooth had at least one tooth on its mesial and distal side which was left undisturbed.

The animals were sacrificed at four different postoperative intervals: eight, fifteen, thirty-six and forty-four days, using a lethal dose of sodium pentobarbitol. The maxillary and mandibular experimental quadrants were dissected away with a scalpel and striker bone saw. The specimens were placed immediately in a 10% formalin solution. The specimens were radiographed and all soft tissue was removed. The excess

a) Glyoxide, International Pharmaceutical Corporation, Kansas City, Missouri

cortical alveolar bone was reduced with a high speed dental bur and water coolant to aid in further fixation and decalcification. Decalcification was accomplished in a solution of 50% formic acid and 20% sodium citrate. The specimens were embedded in paraffin, serial sections were cut and alternate sections stained with hematoxylin and eosin, Giemsa and Brown-Brenn stains.

RESULTS

All four monkeys were in apparent good physical health throughout the experimental period. No undue stress or irritation to the animals was noted before, during or after treatment procedures. The oral health of each animal was that of an intact adult dentition, with the exception of the 44 day animal who was missing a mandibular left lateral incisor. The occlusal surfaces of each animal demonstrated some attrition and mild gingival inflammation was noted also. Preoperative radiographic surveys of each animal revealed no periapical pathosis and demonstrated a variety of pulp canal morphology ranging from somewhat large and straight canals to very narrow, calcified and curved ones. After inoculation of the pulpal tissues with the S. faecalis, radiographic changes in the periapical tissues were noted after approximately two months. These changes ranged from no change at all, to thickened periodontal ligaments, small periapical radiolucencies and large well-defined periapical radiolucencies. Table 1 tabulates the results of the radiographic surveys for postinoculation with the S. faecalis and postintracanal treatment procedures at the time of sacrifice.

The postinoculation radiograph showed one tooth with no periapical change, fifteen teeth with a thickened periodontal ligament space; thirteen teeth with a small periapical radiolucency; and three teeth with a large periapical radiolucency. Radiographs at the time of sacrifice showed six teeth with normal periapical anatomy; thirteen teeth with a

thickened periodontal ligament space; eight teeth with a small periapical radiolucency; and five teeth with a large periapical radiolucency.

Fourteen teeth demonstrated no differences between the postinoculation and time of sacrifice radiographs. Of these, four had been sealed from the oral fluids and ten had been left open. Eleven teeth demonstrated an improved radiographic appearance between the postinoculation and time of sacrifice radiographs. Of these, ten had been sealed from the oral fluids and one had been left open. Seven teeth demonstrated a further decline in their radiographic appearance between the postinoculation and time of sacrifice radiographs. Of these, two had been sealed from the oral fluids and five had been left open.

All cultures were positive for bacterial growth after one week of incubation with the exception of the maxillary left lateral incisor of the eight day animal and the mandibular right lateral incisor of the fifteen day animal.

The cleansing and shaping procedures proved to be very difficult, especially in the premolar teeth, because of the narrowness and the extreme curvature of these canals. All maxillary second premolars demonstrated three roots and three canals while the mandibular second premolars demonstrated two roots and two canals. The mandibular right second premolar of the fifteen day animal was perforated during operative procedures. All amalgam seals were intact at the time of sacrifice.

Tables 2 and 3 tabulate the results of histologic studies. Table 2 tabulates results of the hematoxylin and eosin stain, while Table 3

tabulates results of the Brown-Brenn stain. Serial sections of the hematoxylin and eosin demonstrated nine teeth with a rating of 1, five teeth with a rating of 2, eight teeth with a rating of 3, and ten teeth with a rating of 4. Of the nine teeth with a rating of 1, five had been kept closed and four had been left open to the oral fluids. Of the five teeth with a rating of 2, four had been kept closed and one had been left open to the oral fluids. Of the six teeth with a rating of 3, three had been kept closed and three had been left open to the oral fluids. Of the twelve teeth with a rating of 4, four had been kept closed and eight had been left open to the oral fluids. Fifteen teeth demonstrated marked osteoblastic activity. Of these, eight had been kept closed and seven had been left open to the oral fluids. Five teeth demonstrated marked osteoclastic activity. Of these, all five had been left open to the oral fluids.

Serial sections of the Brown-Brenn stains revealed the presence of bacteria in the prepared canals of twenty-six out of twenty-eight experimental teeth. In four teeth, the prepared canals were not evident enough to make an accurate judgment regarding the presence of bacterial plaques. The two teeth with an absence of bacterial plaques in the prepared canals had both been sealed from the oral fluids. Only five teeth demonstrated the presence of bacterial plaques in their periapical tissues. Two of these had been left open.

The results of the Giemsa stain have been exluded because of technical difficulties in producing quality stained sections. A yeast

organism has become a part of the staining material and at the time of this writing, the technicians have not been able to remove this organism from the Giemsa stain. As a consequence, accurate reading of the serial sections is impossible.

Table 4 compiles the results of both the radiographic and histologic findings. It compares the results between the paired, open or closed, experimental teeth.

Table 1

	Postinoculation	Open/closed	Postintracanal treatment at time of sacrifice
Eight day animal			
MxRP	3	C	4
MxRL	2	C	2
MxLL	2	0	4
MxLP	3	0	4
MnLP	3	0	3
MnLL	2	0	2
MnRL	2	C	1
MnRP	3	C	3
Fifteen day animal			
MxRP	3	0	3
MxRL	2	0	2
MxLL	2	C	1
MxLP	2	C	2
MnLP	2	C	2
MnLL	2	C	1
MnRL	2	0	2
MnRP	2	0	3

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar
MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left central incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

0--Left open
C--Sealed closed with IRM and amalgam

1--No changes noted 2--Thickened periodontal ligament 3--Small periapical radiolucency
4--Large periapical radiolucency

Table 1 (continued)

	Postinoculation	Open/Closed	Postintracanal treatment at time of sacrifice
Thirty-six day animal			
MxRP	3	0	4
MxRL	3	0	3
MxLL	3	C	1
MxLP	2	C	1
MnLP	2	C	1
MnLL	2	C	3
MnRL	3	0	3
MnRP	3	0	3
Forty-four day animal			
MxRP	3	C	2
MxRL	4	C	2
MxLL	4	0	2
MxLP	4	0	4
MnLP	1	0	2
MnLC	2	0	2
MnRL	3	C	2
MnRP	3	C	2

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar
MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left cenral incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

0--Left open
C--Sealed closed with IRM and amalgam

1--No changes noted 2--Thickened periodontal ligament 3--Small periapical radiolucency
4--Large periapical radiolucency

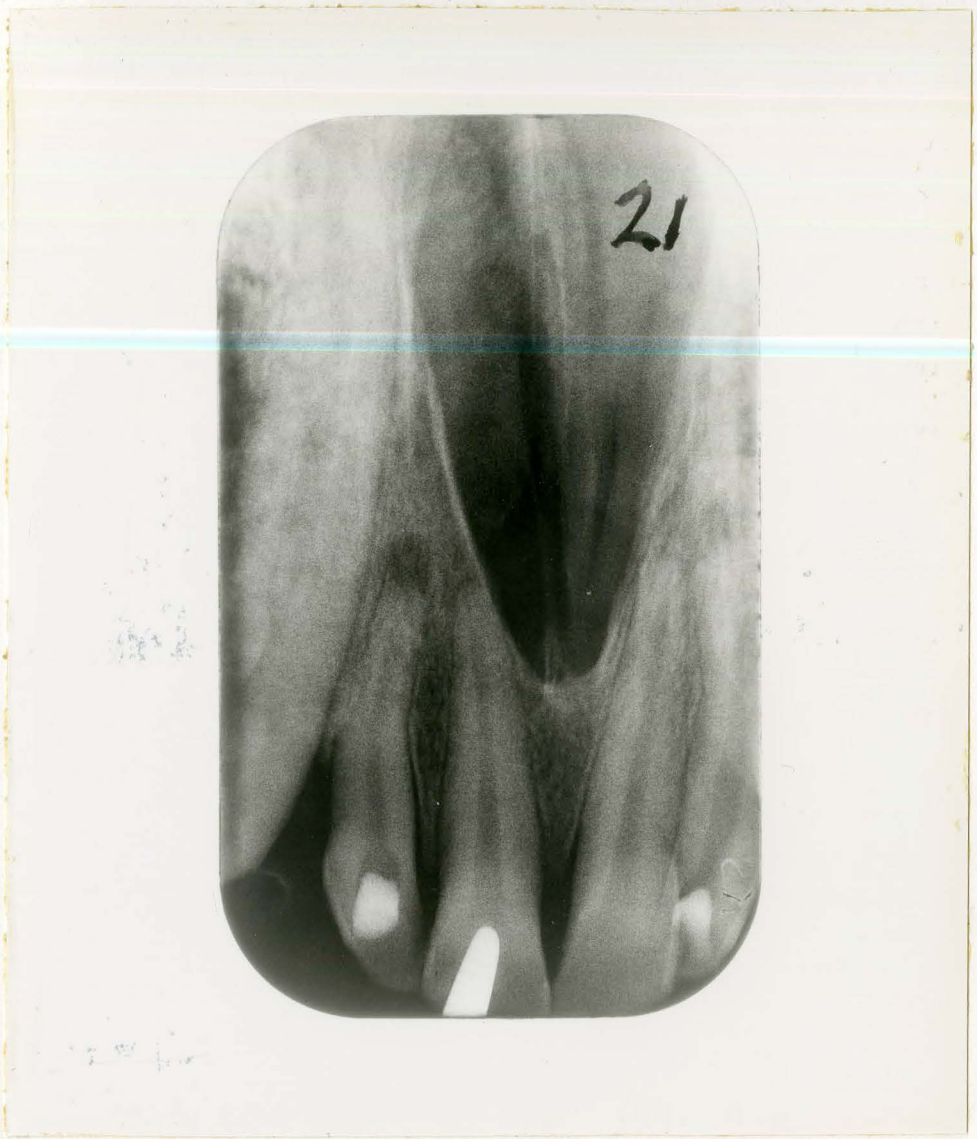

Fig. 1 Demonstrates a large well-defined periapical radiolucency in the forty-four day animal (MxRL). It is an example of the type of periapical radiolucency given the designation 4 in Table 1. This is the postinoculation radiograph taken before the biomechanical cleansing procedures were started.

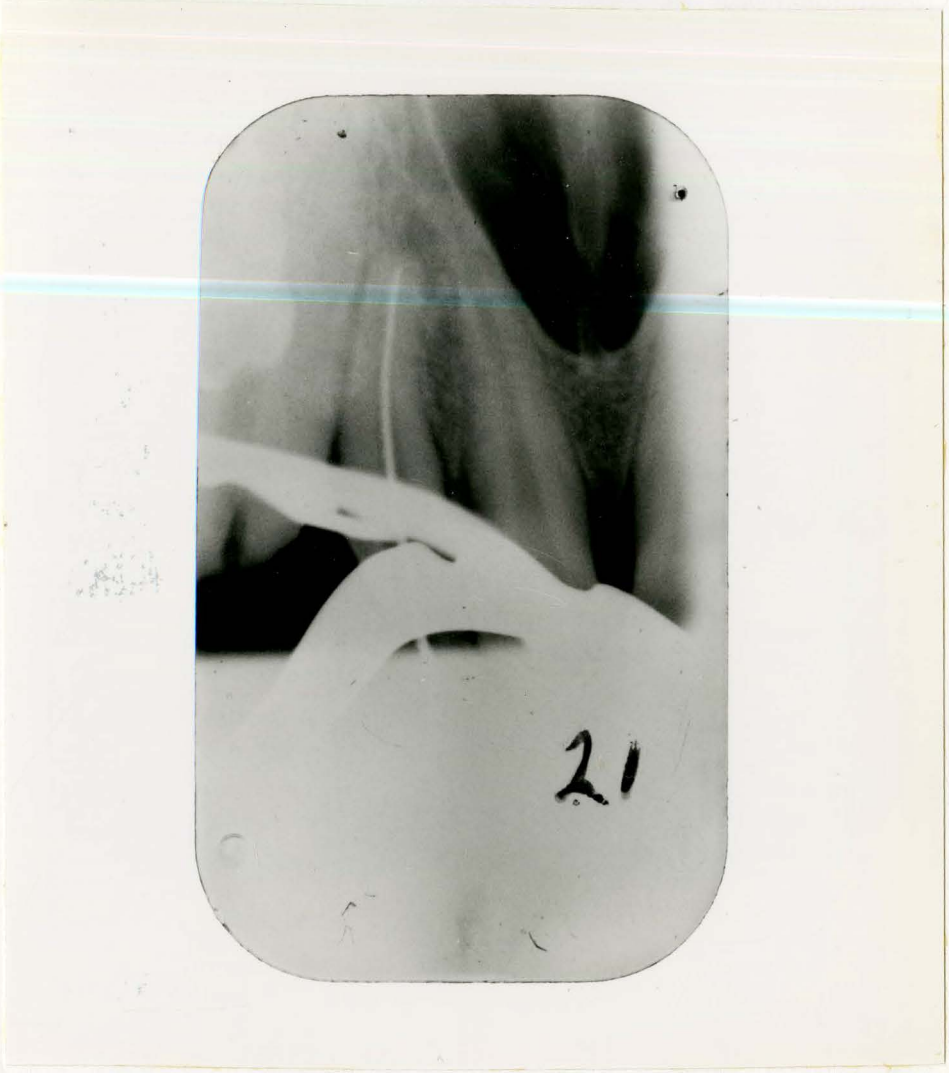

Fig. 2 Demonstrates the isolation and working radiograph taken during the biomechanical cleansing procedures in the forty-four day animal (MxRL).

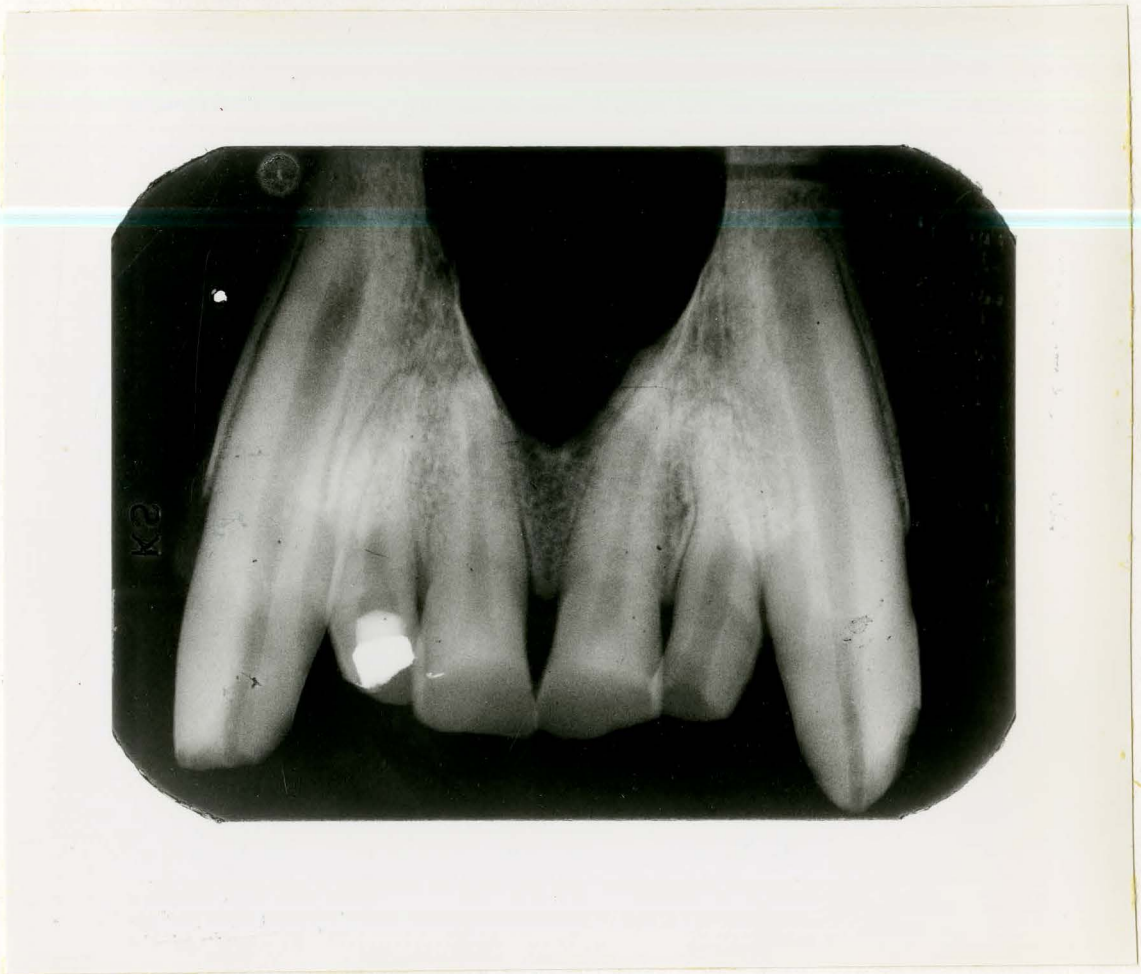

Fig. 3 Demonstrates the postintracanal treatment radiograph of the block section taken at the time of sacrifice of the forty-four day animal. In this radiograph both the tooth kept closed (MxRL) as well as the contralateral tooth left open (MxLL) can be seen. The periodontal ligament space around each apex appears to be thickened and each is an example of the designation 2 in Table 1.

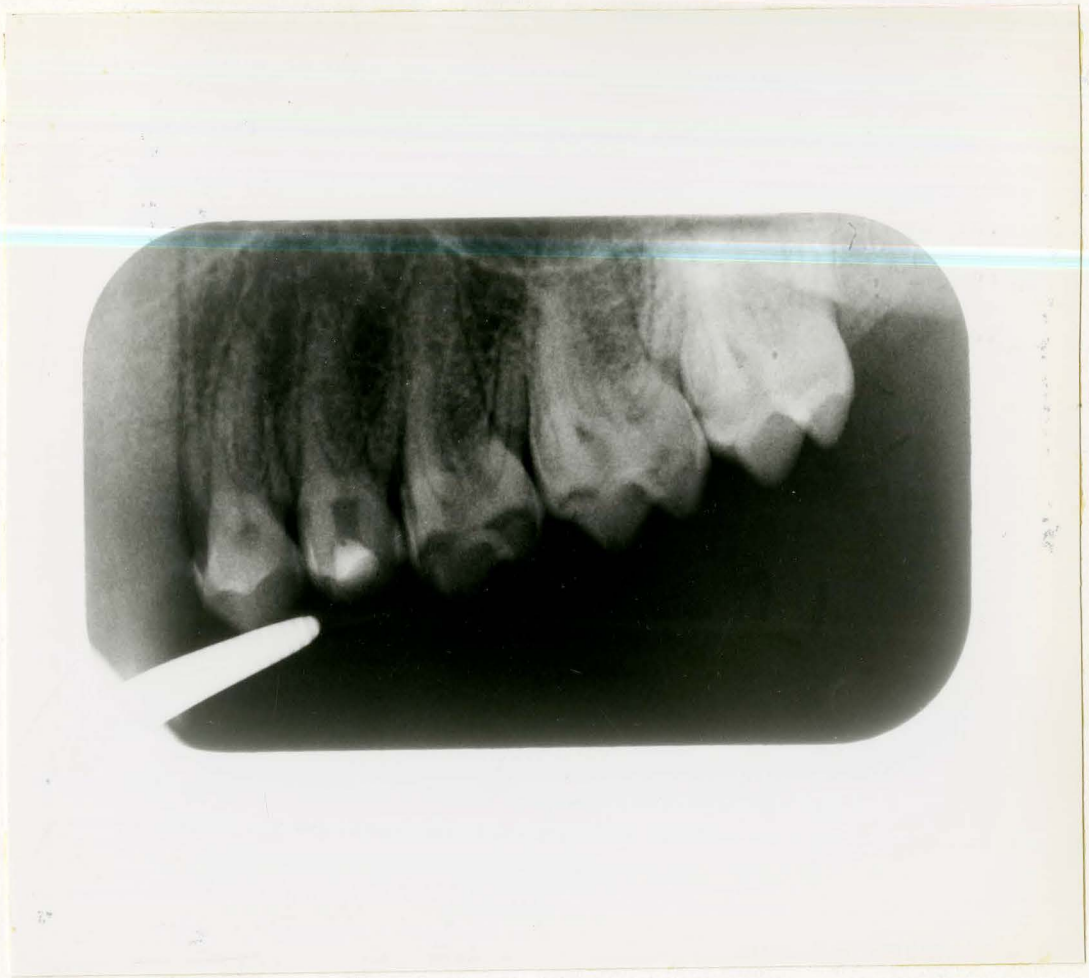

Fig. 4 Demonstrates a large well-defined periapical radiolucency of the palatal root in the forty-four day animal (MxLP). It is an example of the type of periapical radiolucency given the designation 4 in Table 1. This is the postinoculation radiograph taken before the biomechanical cleansing procedures were started. This radiograph also demonstrates the 3 roots of the maxillary premolars and the difficulty in seeing each apex clearly.

Fig. 5 Demonstrates the isolation and working radiograph taken during the biomechanical cleansing procedures of each root in the forty-four day animal (MxLP). It vividly shows the difficulty of determining the correct working length of each root.

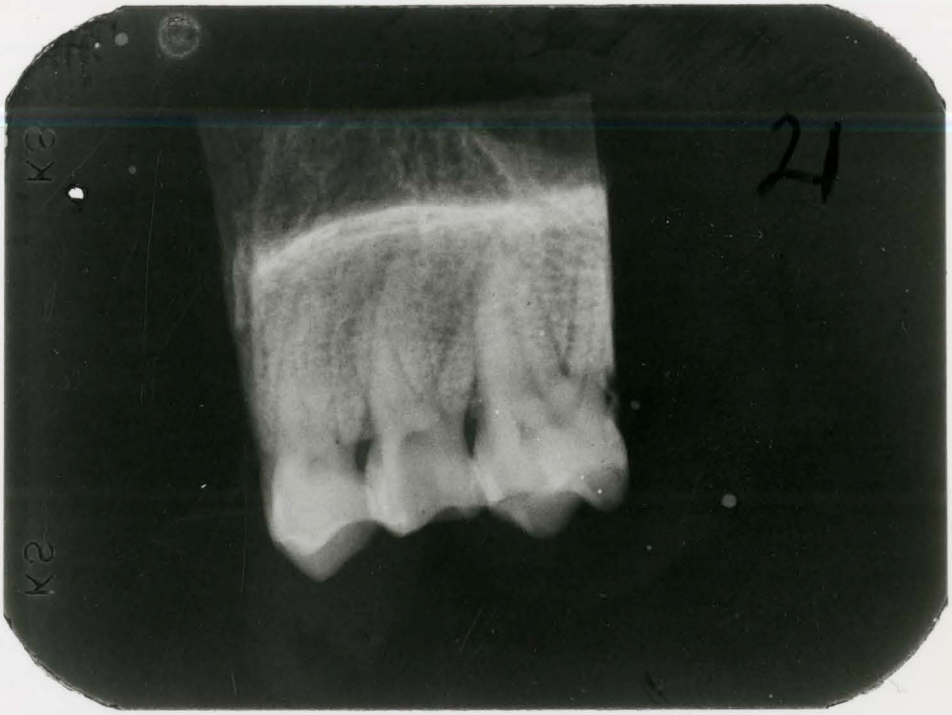

Fig. 6 Demonstrates the postintracanal treatment radiograph of the block section taken at the time of sacrifice of the forty-four day animal (MxLP). In this radiograph, the periapical radiolucency of the palatal root has remained the same size. It was given the designation 4 in Table 1. This radiograph also shows the formation of a smaller periapical radiolucency around the apex of the mesiobuccal root. This tooth had been allowed to remain open to the oral environment after intracanal cleansing procedures.

Table 2 (H & E)

	H & E result	Open/closed
Eight day animal		
MxRP	2	C
MxRL	3 Ob	C
MxLL	4 Ob	O
MxLP	4 Oc	O
MnLP	4 Oc	O
MnLL	3 Ob	O
MnRL	2 Ob	C
MnRP	3	C
Fifteen day animal		
MxRP	4 Ob	O
MxRL	1	O
MxLL	1	C
MxLP	2	C
MnLP	3 Ob	C
MnLL	1	C
MnRL	2 Ob	O
MnRP	4 Oc	O
Thirty-six day animal		
MxRP	1	O
MxRL	3 Ob	O
MxLL	4 Ob	C
MxLP	4 Ob	C
MnLP	1	C
MnLL	4 Ob	C
MnRL	4 Ob	O
MnRP	3 Ob	O

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar
MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left central incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

Ob--Osteoblastic activity
Oc--Osteoclastic activity

1--Normal periodontal ligament, root apex and bone
2--Fibrous capsule surrounding slight chronic inflammatory infiltrate
3--Fibrous capsule surrounding inflammatory infiltrate of plasma cells and lymphocytes
4--Fibrous capsule surrounding inflammatory infiltrate of focal accumulation polymorphonuclear leukocytes as well as plasma cells and lymphocytes

Table 2 (continued)

	H & E result	Open/closed
Forty-four day animal		
MxRP	1	C
MxRL	2 Ob	C
MxLL	1	O
MxLP	4 Oc	O
MnLP	1	O
MnLC	4 Oc	O
MnRL	4 Ob	C
MnRP	1	C

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar
MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left central incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

Ob--Osteoblastic activity
Oc--Osteoclastic activity

1--Normal periodontal ligament, root apex and bone
2--Fibrous capsule surrounding slight chronic inflammatory infiltrate
3--Fibrous capsule surrounding inflammatory infiltrate of plasma cells and lymphocytes
4--Fibrous capsule surrounding inflammatory infiltrate of focal accumulation polymorphonuclear leukocytes as well as plasma cells and lymphocytes

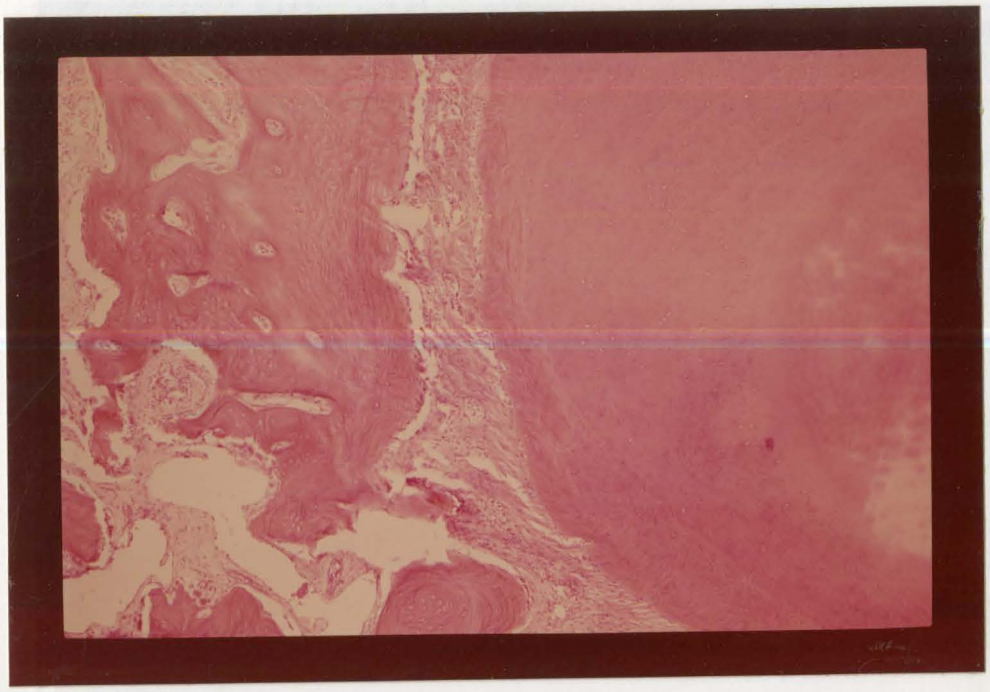

Fig. 7 Demonstrates normal periodontal ligament tissue, alveolar bone and root apex as found in the forty-four day animal's maxillary left lateral incisor. No inflammatory cells can be seen and it was given the designation 1 in Table 2. This tooth had been left open to the oral environment. (40X)

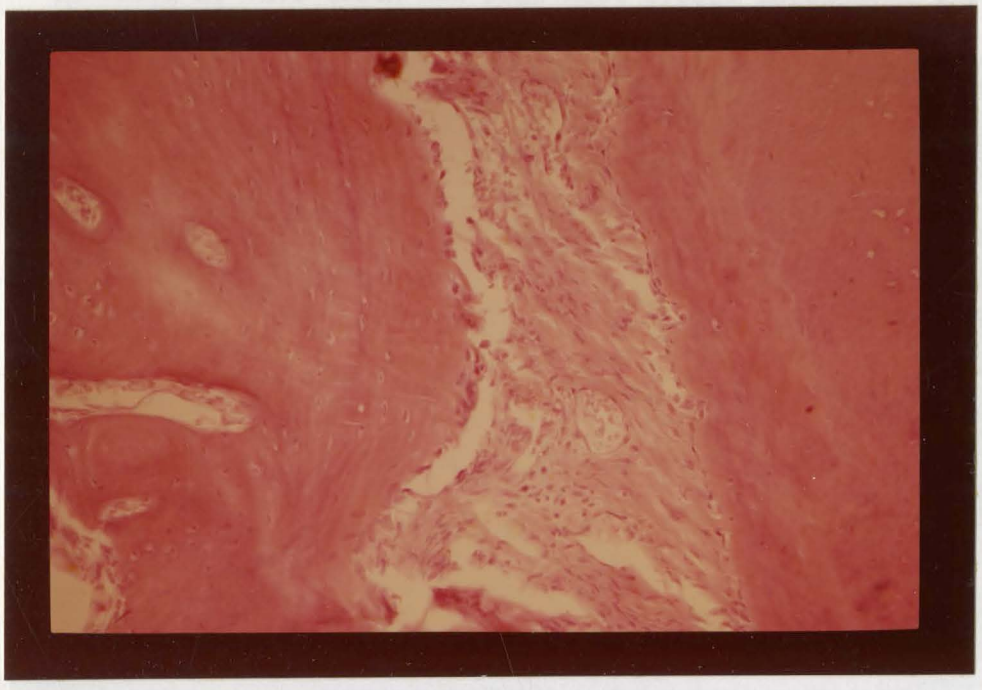

Fig. 8 Demonstrates the same field as Fig. 7 but under higher magnification (100X). No indication of any inflammatory response can be seen.

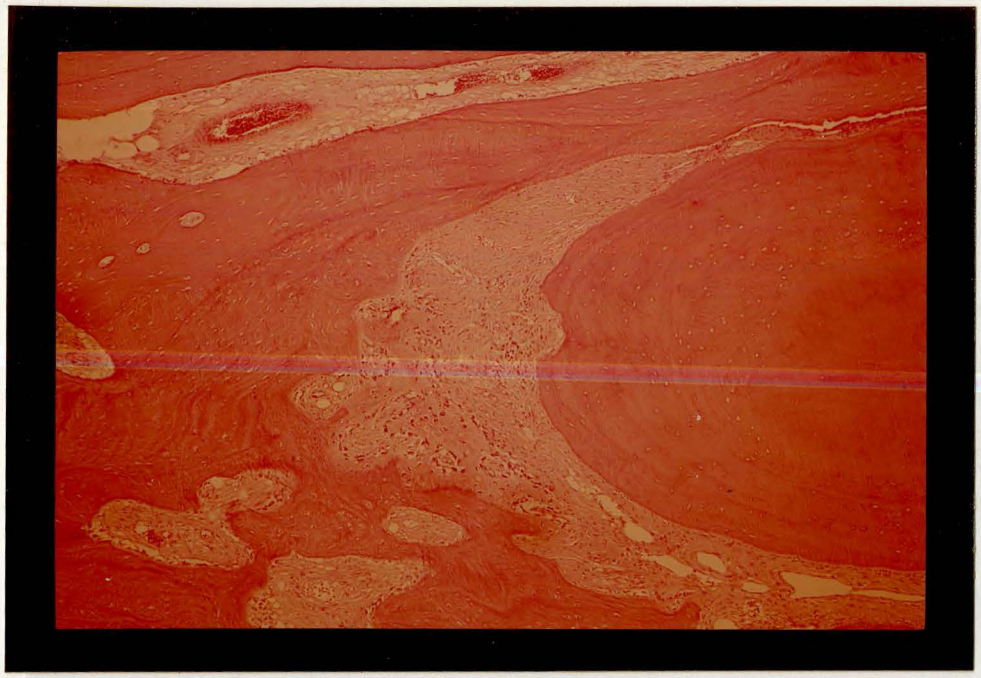

Fig. 9 Demonstrates a slight chronic cell inflammatory infiltrate in the periapical tissue of the eight day animal's mandibular right lateral incisor. It is surrounded by a fibrous capsule that limits the extent of the lesion. Many osteoblasts are seen indicating new bone formation. It was given the designation 2 Ob in Table 2. This tooth had been kept closed throughout treatment. (40X)

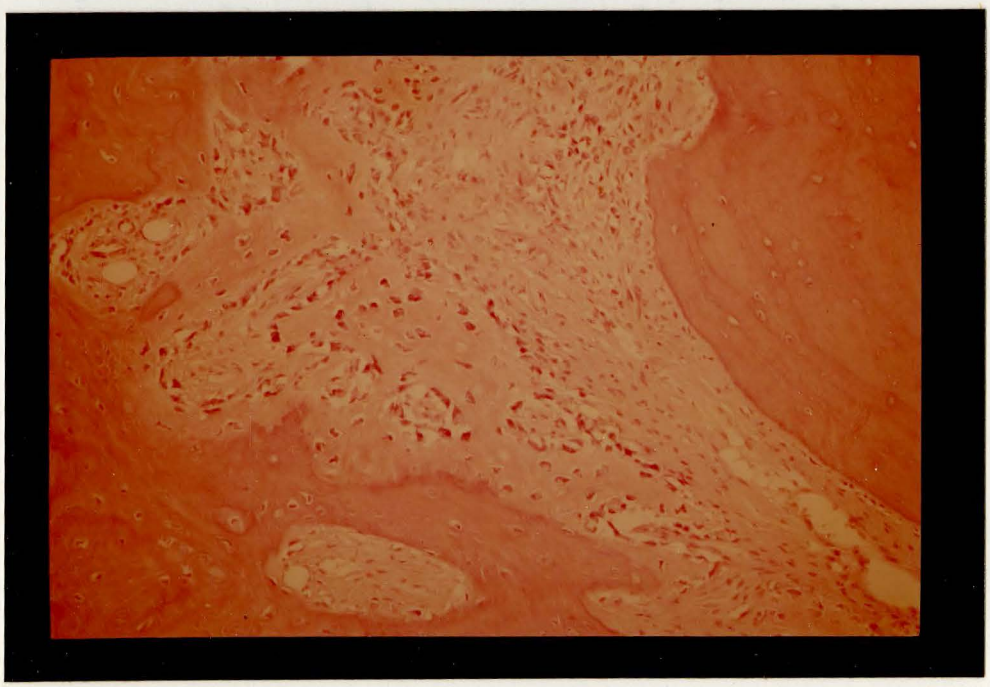

Fig. 10 Demonstrates the same field as Fig. 9 but under higher magnification (100X). The many osteoblasts can be clearly seen.

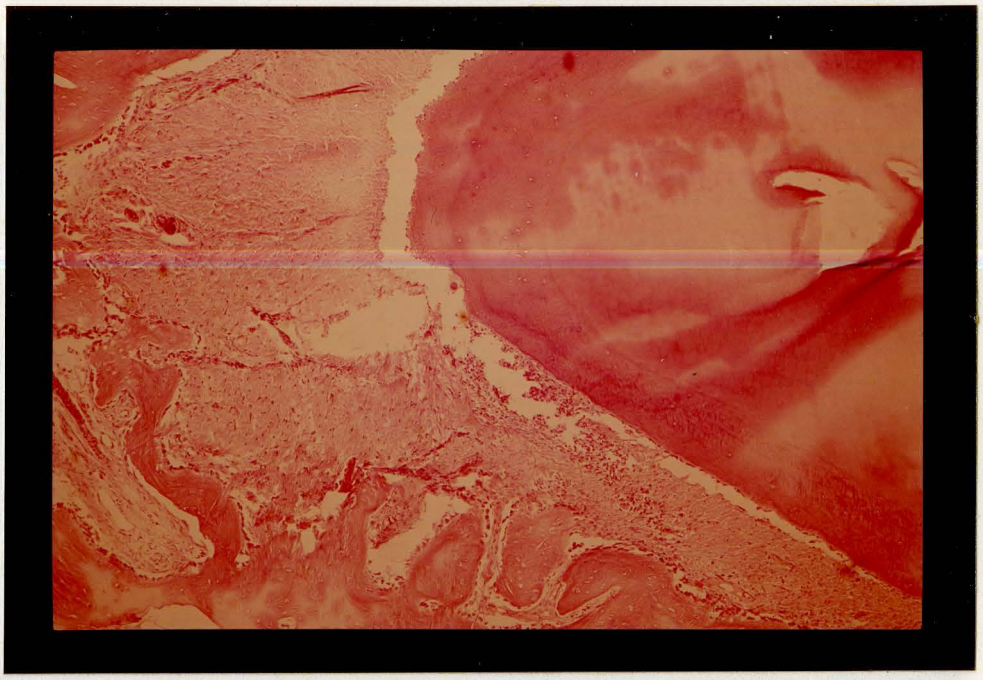

Fig. 11 Demonstrates a moderate chronic cell inflammatory infiltrate in the periapical tissue of the thirty-six day animal's maxillary right lateral incisor. Some scattered polymorphonuclear leukocytes and plasma cells are seen as well as osteoblasts making new bone. It was given the designation of 3 Ob in Table 2. This tooth had been left open to the oral enivronment. (40X)

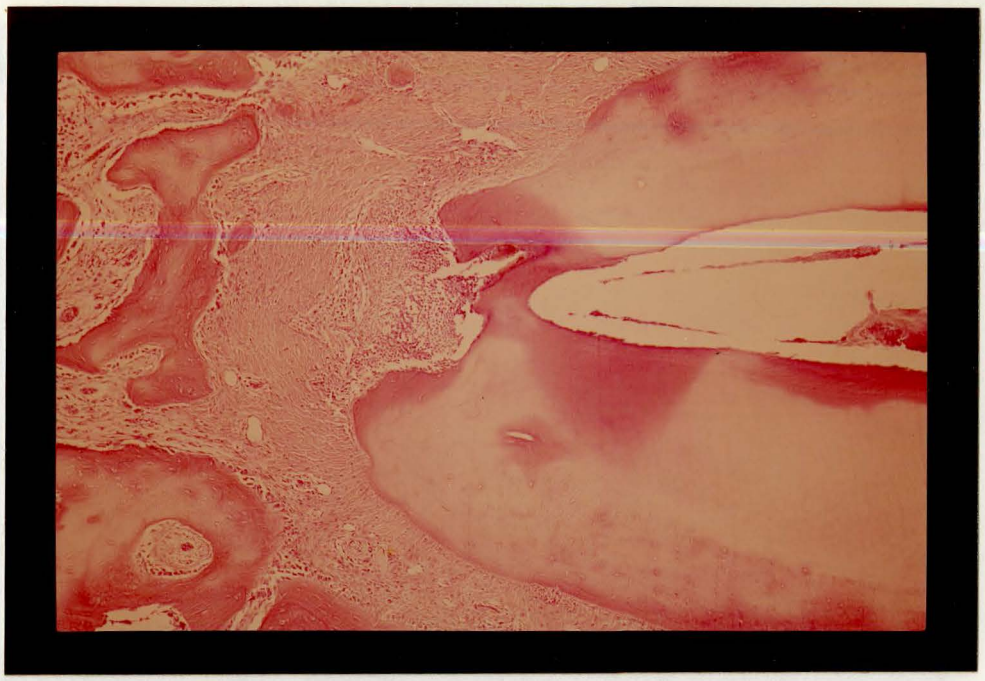

Fig. 12 Demonstrates a focal accumulation of polymorphonuclear leukocytes in the periapical tissue of the thirty-six day animal's maxillary left lateral incisor. This area of acute cells is found in close approximation of the root end directly adjacent to where the canal exits into the periapical tissue and is surrounded by a fibrous capsule limiting its extent as well as osteoblasts making new bone. It was given a designation of 4 Ob in Table 2. This tooth had been kept closed throughout treatment. (40X)

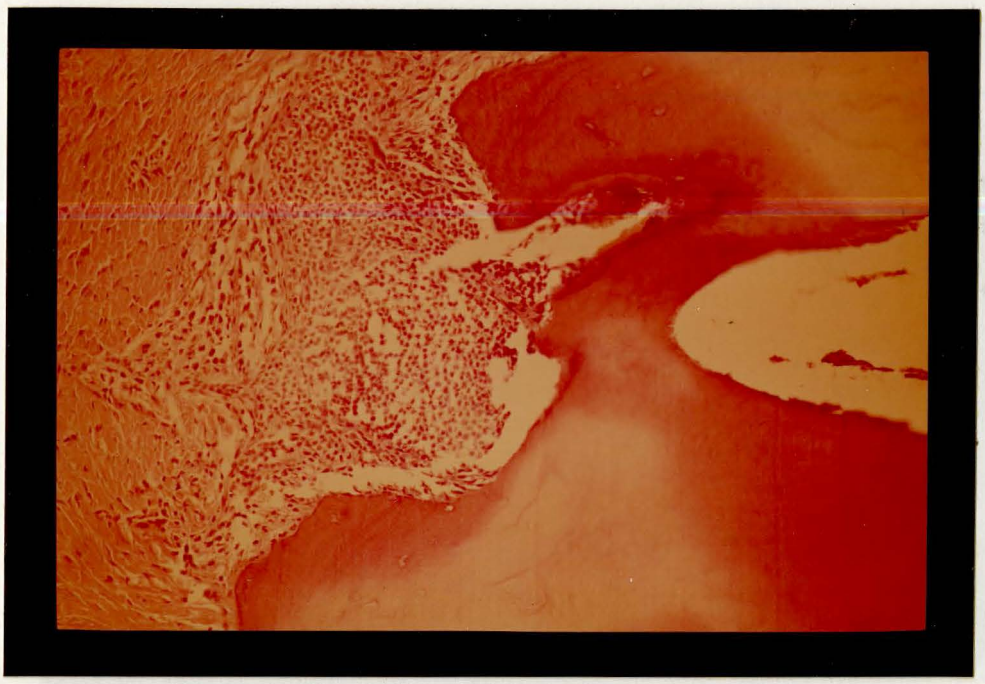

Fig. 13 Demonstrates the same field as Fig. 12 but under higher magnification (100X). The heavy focal accumulation of polymorphonuclear leukocytes can be clearly seen directly adjacent to the root end and the prepared canal. Some debris can be seen in the canal space also.

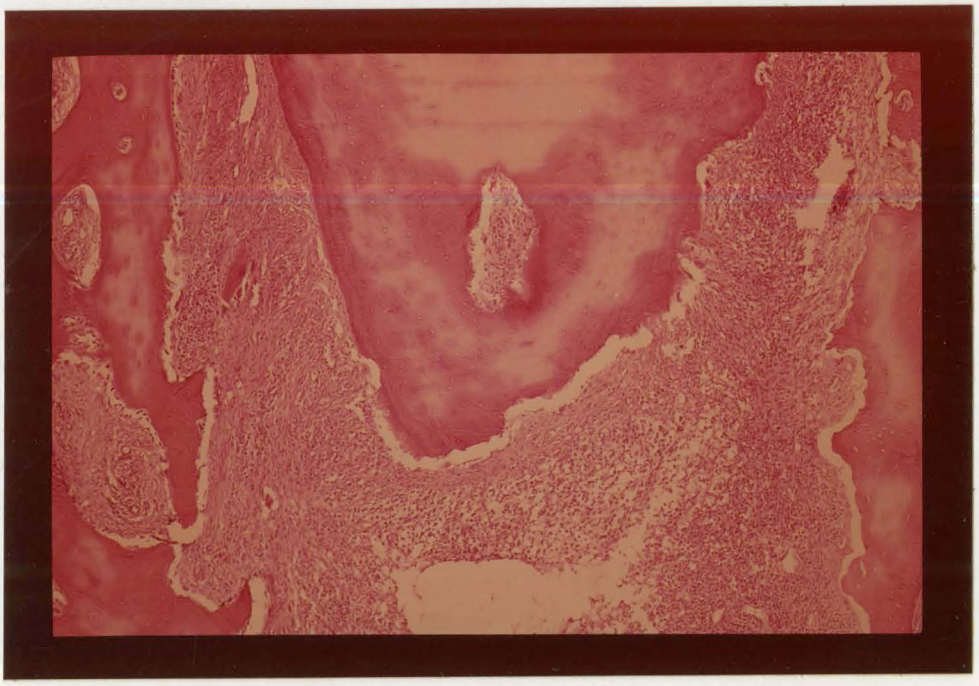

Fig. 14 Demonstrates a massive inflammatory cell infiltrate of both acute and chronic cells in the periapical tissue of the forty-four day animal's mandibular left central incisor. Osteoclastic activity in the bone as well as areas of root resorption can be seen also. It was given a designation of 4 Oc in Table 2. This tooth had been left open to the oral environment. (40X)

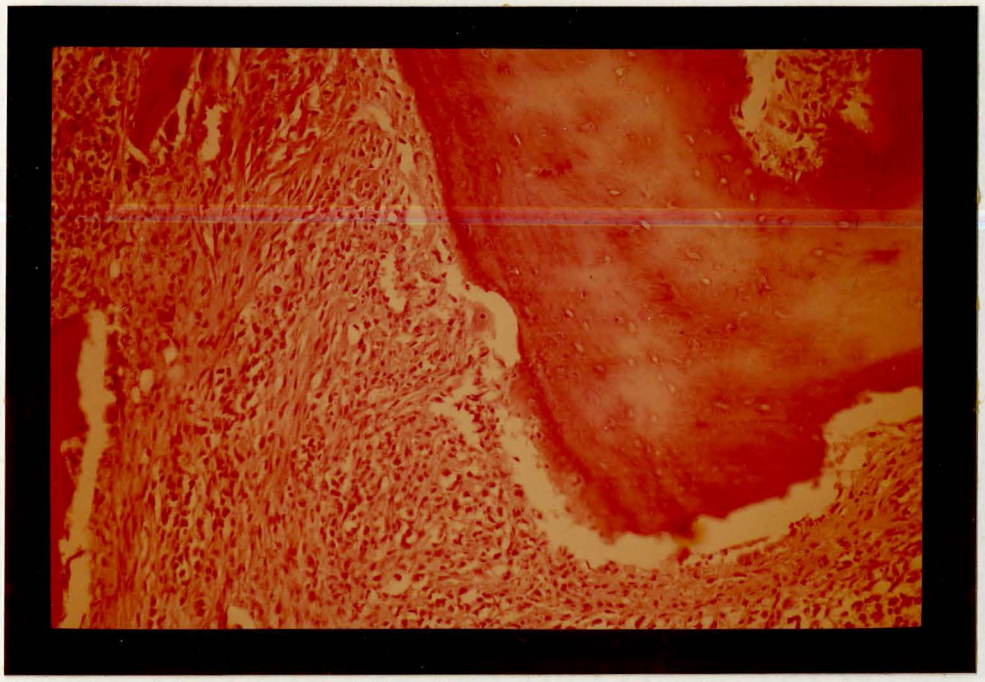

Fig. 15 Demonstrates the same field as Fig. 14 but under higher magnification (100X). The areas of root resorption can be clearly seen as well as the denseness of the inflammatory cell infiltrate.

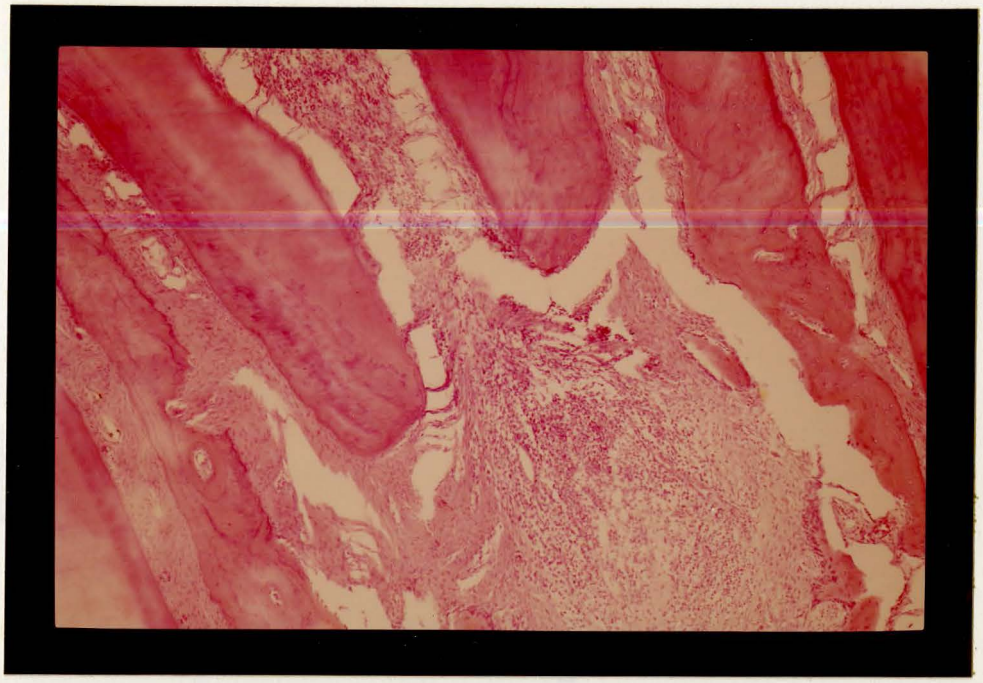

Fig. 16 Demonstrates a massive inflammatory cell infiltrate of both acute and chronic cells in the periapical tissue and prepared canal space of the forty-four day animal's mandibular right lateral incisor. It can be seen that apical foramen was violated during the intracanal cleansing procedures. It is unknown what effect the overinstrumentation had on the outcome of the lesion. It was given the designation of 4 Ob in Table 2 because some osteoblastic activity can be seen. This tooth had been kept closed throughout treatment. (40X)

Table 3 (Brown-Brenn)

	Bacteria in canal	Bacteria in periapical tissue	Open/closed
Eight day animal			
MxRP	+	-	C
MxRL	+	-	C
MxLL	+	+	O
MxLP	+	-	O
MnLP	+	+	O
MnLL	+	+	O
MnRL	+	-	C
MnRP	+	-	C
Fifteen day animal			
MxRP	+	-	O
MxRL	+	-	O
MxLL	+	-	C
MxLP	+	-	C
MnLP	slides of canal are unreadable	-	C
MnLL	+	-	C
MnRL	+	-	O
MnRP	+	-	O

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar

MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left central incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

O--Left open
C--Sealed closed with IRM and amalgam

+ --Bacteria present
- --Bacteria absent

Table 3 (continued)

	Bacteria in canal	Bacteria in periapical tissue	Open/closed
Thirty-six day animal			
MxRP	+	-	O
MxRL	+	-	O
MxLL	+	-	C
MxLP	+	+	C
MnLP	slides of canal are unreadable	-	C
MnLL	+	+	C
MnRL	+	-	O
MnRP	+	-	O
Forty-four day animal			
MxRP	slides of canal are unreadable	-	C
MxRL	-	-	C
MxLL	+	-	O
MxLP	+	-	O
MnLP	slides of canal are unreadable	-	O
MnLC	+	-	O
MnRL	-	-	C
MnRP	+	-	C

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar
MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left central incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

O--Left open
C--Sealed closed with IRM and amalgam

+ --Bacteria present
- --Bacteria absent

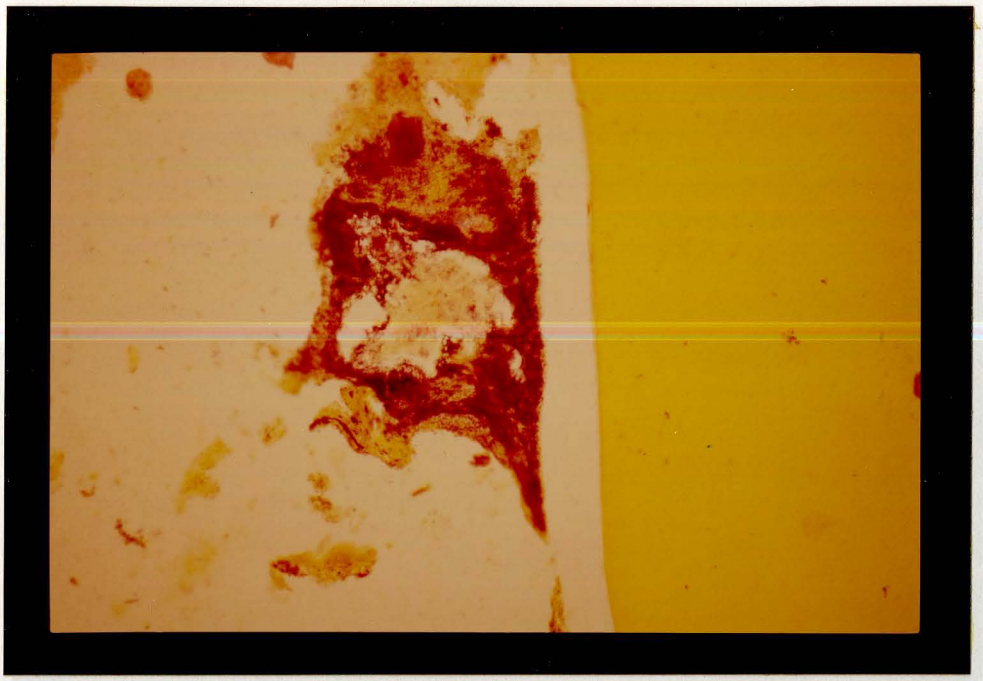

Fig. 17 Demonstrates a heavy plaque of bacteria and debris inside the prepared canal space of the forty-four day animal's maxillary left lateral incisor. This tooth had been left open to the oral environment. (100X)

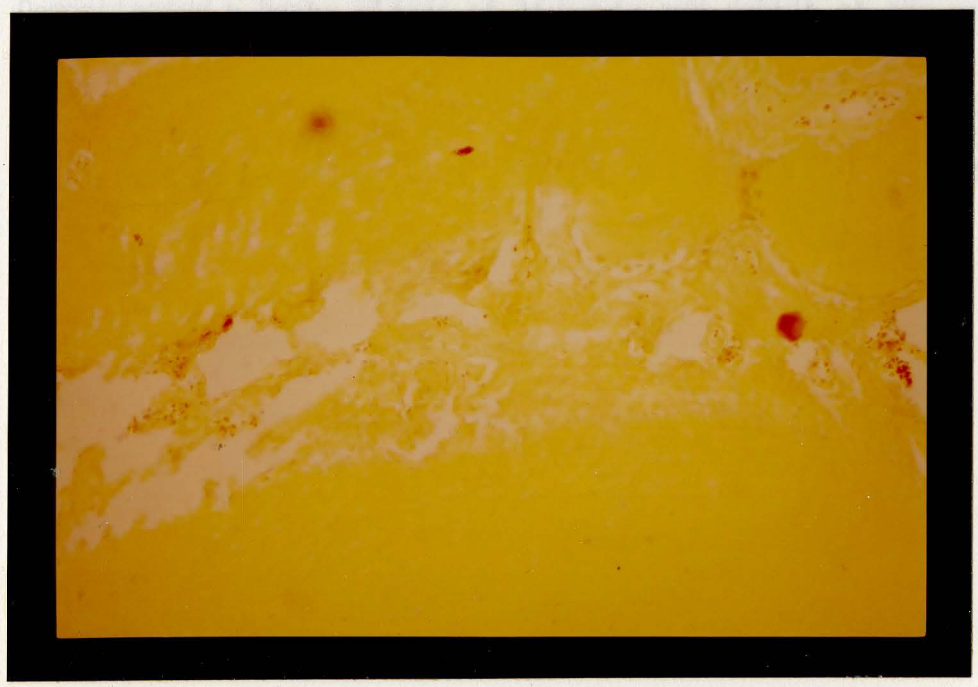

Fig. 18 Demonstrates the bacteria-free periapical tissue of the forty-four day animal's maxillary left lateral incisor. This tooth had been left open to the oral environment. (100X)

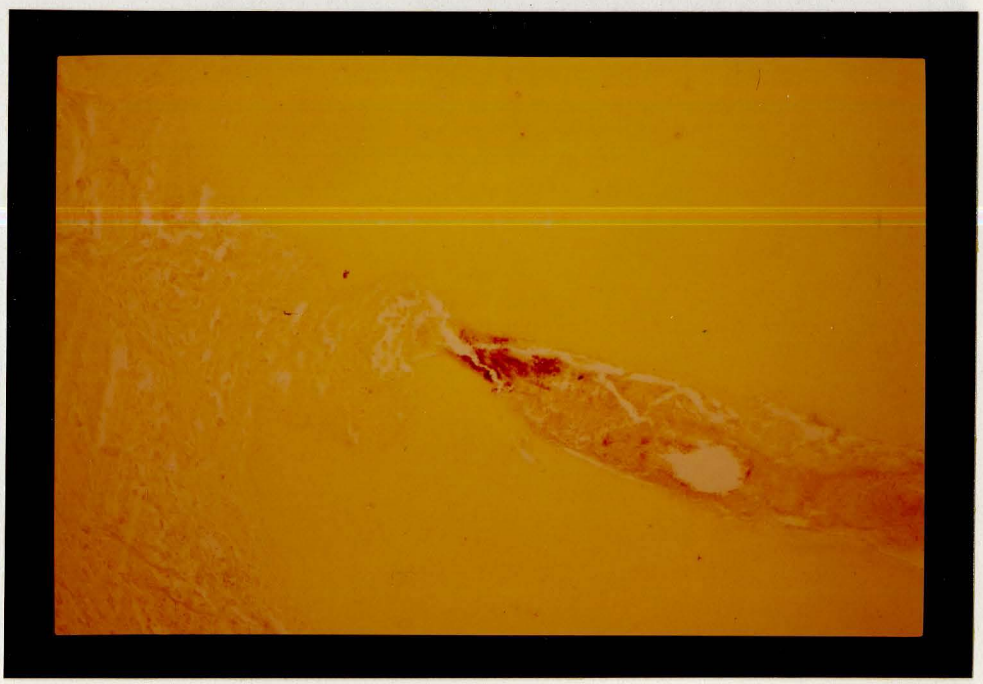

Fig. 19 Demonstrates a heavy plaque of bacteria and debris at the very tip of the prepared canal space of the thirty-six day animal's maxillary left lateral incisor. However, as heavy as the bacterial plaque is and as close to the periapical tissue it is, there are no bacterial plaques evident in the periapical tissue. This tooth had been kept closed throughout treatment. (100X)

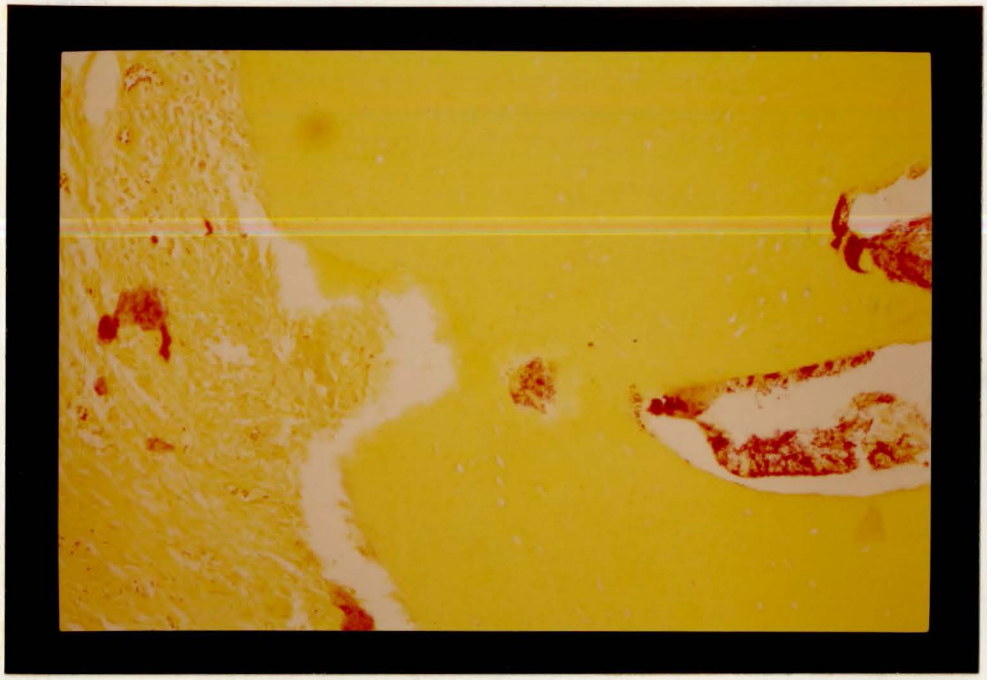

Fig. 20 Demonstrates heavy plaques of bacteria and debris in the prepared canal space and at the very tip of the prepared canal space of the eight day animal's maxillary left lateral incisor. It also demonstrates a few scattered plaques of bacteria in the periapical tissues. This tooth had been left open to the oral environment. (100X)

Table 4

Eight day animal

	Open		Closed	
MxLL		Radiographic findings		MxRL
	2	Postinoculation	2	
	4	Postintracanal treatment at time of sacrifice	2	
		Histologic findings		
	4 Ob	H & E stain	3 Ob	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	+	Bacteria in periapical tissue	-	
MxLP		Radiographic findings		MxRP
	3	Postinoculation	3	
	4	Postintracanal treatment at time of sacrifice	4	
		Histologic findings		
	4 Oc	H & E stain	2	
	-	Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	-	
MnLL		Radiographic findings		MnRL
	2	Postinoculation	2	
	2	Postintracanal treatment at time of sacrifice	1	
		Histologic findings		
	3 Ob	H & E stain	2 Ob	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	+	Bacteria in periapical tissue	-	
MnLP		Radiographic findings		MnRP
	3	Postinoculation	3	
	3	Postintracanal treatment at time of sacrifice	3	
		Histologic findings		
	4 Oc	H & E stain	3	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	+	Bacteria in periapical tissue	-	

Table 4 (continued)

Fifteen day animal

	Open		Closed	
MxRL		Radiographic findings		MxLL
	2	Postinoculation	2	
	2	Postintracanal treatment at time of sacrifice	1	
		Histologic findings		
	1	H & E stain	1	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	-	
MxRP		Radiographic findings		MxLP
	3	Postinoculation	2	
	3	Postintracanal treatment at time of sacrifice	2	
		Histologic findings		
	4 Ob	H & E stain	2	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	-	
MnRL		Radiographic findings		MnLL
	2	Postinoculation	2	
	2	Postintracanal treatment at time of sacrifice	1	
		Histologic findings		
	2 Ob	H & E stain	1	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	-	
MnRP		Radiographic findings		MnLP
	2	Postinoculation	2	
	3	Postintracanal treatment at time of sacrifice	2	
		Histologic findings		
	4 Oc	H & E stain	3 Ob	
		Brown-Brenn stain		
	+	Bacteria in canal	slides are unreadable	
	-	Bacteria in periapical tissue	-	

Table 4 (continued)

Thirty-six day animal

	Open		Closed	
MxRL		Radiographic findings		MxLL
	3	Postinoculation	3	
	3	Postintracanal treatment at time of sacrifice	1	
		Histologic findings		
	3 Ob	H & E stain	4 Ob	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	-	
MxRP		Radiographic findings		MxLP
	3	Postinoculation	2	
	4	Postintracanal treatment at time of sacrifice	1	
		Histologic findings		
	1	H & E stain	4 Ob	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	+	
MnRL		Radiographic findings		MnLL
	3	Postinoculation	2	
	3	Postintracanal treatment at time of sacrifice	3	
		Histologic findings		
	4 Ob	H & E stain	4 Ob	
		Brown-Brenn stain		
	+	Bacteria in canal	+	
	-	Bacteria in periapical tissue	+	
MnRP		Radiographic Findings		MnLP
	3	Postinoculation	2	
	3	Postintracanal treatment at time of sacrifice	1	
		Histologic findings		
	3 Ob	H & E stain	1	
		Brown-Brenn stain		
	+	Bacteria in canal	slides are unreadable	
	-	Bacteria in periapical tissue	-	

Table 4 (continued)

Forty-four day animal

Open		Closed	
MxLL	Radiographic findings		MxRL
4	Postinoculation	4	
2	Postintracanal treatment at time of sacrifice	2	
	Histologic findings		
1	H & E stain	2 Ob	
	Brown-Brenn stain		
+	Bacteria in canal	-	
-	Bacteria in periapical tissue	-	
MxLP	Radiographic findings		MxRP
4	Postinoculation	3	
4	Postintracanal treatment at time of sacrifice	2	
	Histologic findings		
4 Oc	H & E stain	1	
	Brown-Brenn stain		
+	Bacteria in canal	slides are unreadable	
-	Bacteria in periapical tissue	-	
MnLC	Radiographic findings		MnRL
2	Postinoculation	3	
2	Postintracanal treatment at time of sacrifice	2	
	Histologic findings		
3 Oc	H & E stain	3 Ob	
	Brown-Brenn stain		
+	Bacteria in canal	-	
-	Bacteria in periapical tissue	-	
MnLP	Radiographic findings		MnRP
1	Postinoculation	3	
2	Postintracanal treatment at time of sacrifice	2	
	Histologic findings		
1	H & E stain	1	
	Brown-Brenn stain		
slides are unreadable	Bacteria in canal	+	
-	Bacteria in periapical tissue	-	

Table 4 (continued)

Legend: MxRP--Maxillary right second premolar
MxRL--Maxillary right lateral incisor
MxLL--Maxillary left lateral incisor
MxLP--Maxillary left second premolar
MnLP--Mandibular left second premolar
MnLL--Mandibular left lateral incisor
MnLC--Mandibular left central incisor
MnRL--Mandibular right lateral incisor
MnRP--Mandibular right second premolar

Radiographic findings:
1--No changes noted
2--Thickened periodontal liagment
3--Small periapical radiolucency
4--Large periapical radiolucency

Histologic findings:
H&E stain
1--Normal periodontal ligament, root apex and bone
2--Fibrous capsule surrounding slight chronic inflammatory infiltrate
3--Fibrous capsule surrounding inflammatory infiltrate of plasma cells and lymphocytes
4--Fibrous capsule surrounding inflammatory infiltrate of focal accumulation polymorphonuclear leukocytes as well as plasma cells and lymphocytes

Ob--Osteoblastic activity
Oc--Osteoclastic activity

Brown-Brenn stain
+ --Bacteria present
- --Bacteria absent

DISCUSSION

It is apparent from the results of this study that periapical radiographic changes can be produced in a majority of teeth of the Rhesus monkey by means of inoculating the pulpal tissues with a known inoculum of S. faecalis. These changes will be seen as early as two months after inoculation and can vary from no periapical change, to a thickening of the periodontal ligament space, and to a well-defined periapical radiolucency. The reasons for varied radiographic appearances are most likely due to the amount of cortical bone involved by the lesion. Seltzer and Bender have shown that "...areas of rarefaction manifest themselves only if there is erosion of the cortex from the inner or outer surface or if there is frank perforation."[71] A lesion cannot be visualized on a radiograph if it is confined to the cancellous bone. It would seem that with such a short time limit to develop periapical changes, those teeth with their apices in closest proximity to the cortical bone would demonstrate the greatest and most severe periapical changes on a periapical radiograph. Teeth with their apices further from cortical bone would demonstrate the least amount of periapical change because a lesser degree of cortical bone has had time to become involved. It would even be possible for a tooth to have periapical pathology present but not demonstrate a change on a periapical radiograph because the cortical bone has not had time to become involved. Other factors which may account for the variety of

radiographic appearances might include the size and shape of the canal, the difficulty of taking an accurate radiograph particularly in the second premolar area, and the variability of host resistance. The smaller, more calcified and curved canals might not allow a sufficient number of bacteria or bacterial degradation products to reach the periapex to overcome the host's defenses and involve the cortical bone. Taking an accurate radiograph in the second premolar region proved to be a very difficult task. The palatal vault and floor of the mouth were very shallow in some of the animals making a radiograph using a paralleling technique impossible to take. Because the maxillary second premolars were three-rooted teeth and the mandibular second premolars were two-rooted, radiographs visualizing each root clearly were difficult to obtain. To attempt to correlate differences between the postinoculation and time of sacrifice radiographs would prove to be inaccurate because the exact film placement as well as the vertical and horizontal components of the X-ray beam could not be duplicated. Thus, minor distortions of the lesion would occur. The final factor which may account for the variety of radiographic appearances is host resistance. The stronger, healthier animals with a more competent defense system would be able to resist the influx of microorganisms with less tissue damage. With a lesser amount of tissue damage, the amount of cortical bone involved would be diminished. Thus, the radiographic appearance would demonstrate either no change if the pathology had not involved the cortical bone or a mild thickening of the periodontal ligament space if only a small portion of the cortical bone were involved. Conversely, an animal with a less

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canal space. Torneck (1969) has also stated that "there are several etiological factors which can delay or prevent healing in the periapical tissues following treatment of the root canal."[72] One of these factors is the presence of microorganisms. However, they must co-exist with some other factor that would prevent repair. Not all microorganisms or their by-products can exert this influence and even among those that can, a critical number must be present as well as a time factor to manifest this effect.

In this study, periapical disease was created from the inoculation of the pulpal tissues with S. faecalis. Approximately two months after inoculation, biomechanical cleansing and shaping with heavy use of lubricants and irrigants of the infected pulp canals was begun. One can only speculate whether the periapical disease at that time reflected periapical inflammation or infection. However, the vast majority of pulp canals (thirty out of thirty-two teeth) demonstrated infection as shown in the results of the cultures. The cleansing and shaping of the infected pulp canals proved to be exceedingly difficult because of the narrowness and curvature of the canals. Even with the use of various lubricants, irrigants, incremental instrumentation, and flare preparations the bacteria in the canals were not entirely eliminated. In only two teeth were there no bacteria present in the microscopic sections of the prepared pulp canals. This finding is in agreement with Matsumiya and Kitamura who have stated "by methods of sterilization of the root canal which are now generally employed, traces of bacteria can always be found not only in the apical ramifications, the cementum lacunae and the dentinal tubules,

but they were also frequently observed in the principal root canals immediately after sterilization."[73] However, even with the apparent incompleteness of the elimination of bacteria from the infected pulp canals by means of cleansing and shaping them and the presence of an additional factor of allowing salivary contamination in half of the teeth, bacteria were found in the periapical tissues in only five teeth. The bacterial plaques that were found seemed to be very few and scattered in number. Of these, three were subjected to the additional factor of salivary contamination. It is evident that in this study the additional factor of salivary contamination of the prepared pulp canals did not change the charac ter of the periapical disease. The periapical pathology seen was not one of the periapical infection, but rather one of periapical inflammation. Of the twenty-three teeth with periapical inflammation (nine teeth had normal appearing periapical tissues), all demonstrated the presence of a fibrous capsule. The fibrous capsule is a response of the periapical tissues to wall off the periapical lesion and to promote healing. Thus, each experimental tooth with periapical disease (inflammation) was actively engaged at the time of sacrifice in healing the periapical lesion. Whether or not the experimental teeth demonstrating the more severe category of periapical inflammation as witnessed by foci of polymorphonuclea leukocytes would continue the healing process until returning to a normal state of periapical tissue is a matter of speculation. All that can be stated from the results of this study is that in each experimental tooth the first step of healing, limiting the progress of pathology, was taken.

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The controversy over whether to keep a tooth closed or leave a tooth open for drainage exists because patients demand relief from pain. The dentist who leaves a tooth open for drainage believes that by doing this his patient will be relieved of the pain in the most efficient manner and shortest time. The dentist who attempts to keep a tooth closed during treatment believes that his patient will have the most efficient treatment with the fewest exacerbations. The common parameter in each type of dentist's thinking is to relieve his patient's pain and to keep him comfortable. Because this was an animal study an accurate assessment of pain differences between the two procedures could not be accomplished. A possible solution to part of this problem would entail a future study of the histochemicals involved in both the cell-mediated and humoral immunologic phenomena of the inflammatory response. During an acute inflammatory response in the pulp or periapical tissue, chemical mediators such as histamine, 5-hydroxytryptamine, kinins and various other polypeptides are released. In addition, prostaglandins and cyclic AMP (adenosine 3',5' monophosphate) probably also play roles in acute inflammation.[74] Complement must also be present to mediate the response in the later stages of acute inflammation. An intense infiltration of polymorphonuclear leukocytes may elicit severe reactions from the release of lysosomal contents such as hydrolytic enzymes. The release of the enzymes produces damage in nearby cells which may result in severe pain. In chronic inflammation of the pulp or periapical tissue the presence of

macrophages and lymphocytes indicates that both cell-mediated and humoral immune reactions are involved thus producing immunoglobulins, complement fixation, and plasma cell infiltration.[74] A qualitative study demonstrating the presence or absence of these histochemicals as well as a quantitative one demonstrating the amounts involved may be helpful in determining if there might be less pain involved in one type of treatment such as keeping a tooth closed as opposed to the other such as opening and allowing a tooth to remain open for drainage.

Although it would appear from the results of the histologic survey that the teeth left open to the oral fluids demonstrated a slightly more severe inflammatory reaction in their periapical tissues than the teeth kept sealed, other variable factors prohibit drawing this conclusion. All experimental teeth demonstrated that initial efforts in the form of a fibrous capsule had been made to defend against and limit the progress of the periapical pathology. Actual differences in the severity of the periapical inflammation might be attributed to an incomplete cleansing of the pulp space and/or possible overinstrumentation during the cleansing and shaping procedures. As shown by the results in Table 3, the majority of prepared pulp canals had bacteria remaining in the canal space after the cleansing and shaping procedures were completed. Although no conclusions could be made concerning the amount of bacteria remaining in the prepared canals, it may be assumed that some canals had more remaining bacteria and pulpal debris than others, thus possibly accounting for a difference in the severity of periapical inflammation. Because many of the pulp

canals were extremely curved and calcified and because of the difficulty in obtaining accurate working length radiographs particularly in the multirooted premolar teeth, some pulp canals were overinstrumented unintentionally. This additional factor could also be responsible for a difference in the severity of periapical inflammation among the experimental teeth.

In order to obtain results from which valid conclusions might be drawn some changes might be suggested for future studies. Methods might be developed to eliminate the variables involved with the cleansing and shaping of the pulp canals to ensure that accurate length determination might be made and that thorough cleansing and shaping of the canals occurs. Different time periods should be studied to determine if differences exist over the immediate or long term. A sacrifice time between one and three days could possibly demonstrate a significant difference in the acute inflammatory responses between teeth left open to the oral fluids and those kept sealed. It is within this time interval where the majority of clinical practitioners find most of their problems. Sacrifice times between three months and one year would help to demonstrate if any differences exist over long periods of time and if continued healing and a return to normal periapical tissue follow the initial reaction of fibrous encapsulation. As mentioned earlier, the histochemicals involved in the cell-mediated and humoral immunologic phenomena of the inflammatory response should be studied to possibly help understand the pain involved in these procedures. Lastly, more animals and experimental teeth

SUMMARY AND CONCLUSION

The pulp tissues of thirty-two teeth in Rhesus monkeys were inoculated with S. faecalis in order to induce periapical pathosis. The infected pulp canals were cleansed and shaped by chemical and biomechanical means. Half of the prepared canals were sealed with IRM and amalgam alloys while the contralateral half were left open to the oral fluids. The post-operative sacrifice times were at eight, fifteen, thirty-six, and forty-four days.

The radiographic results demonstrated that changes in the periapical tissues on a periapical radiograph could be noted. These changes ranged from no change at all, to thickened periodontal ligaments, small periapical radiolucencies, and large well-defined periapical radiolucencies.

The histologic observations revealed that despite careful chemical and biomechanical cleansing and shaping, the majority of pulp canals had bacteria and debris in the prepared pulp canal spaces. The periapical tissues of the majority of teeth demonstrated an inflammatory reaction and were bacteria free. Of the teeth that demonstrated bacteria in their periapical tissues, the bacteria seemed to be very few and scattered. The bacteria were found in both the teeth where the canals had been left open and those that had been sealed with IRM and amalgam alloys.

A difference in periapical healing between the teeth that were left open and those that were sealed from the oral fluids could not be seen.

The periapical tissues of all teeth, although showing a difference in the severity of the inflammatory response, demonstrated the presence of a fibrous capsule surrounding and limiting the lesion. All experimental teeth were making an attempt to resolve the periapical pathology.

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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 Committee with reference to content and form.

The thesis is therefore accepted in partial fulfillment of the requirements for the degree of master of Science in Oral Biology.

April 8 1982 Patrick D. Toto D.D.S. M.S.

Date Director's Signature